The Effect of Maternal Anthropometry, Demography and Health Conditions on Newborn Anthropometry in Federal Teaching Hospital Gombe, Gombe State, Nigeria.

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THE EFFECT OF MATERNAL ANTHROPOMETRY, DEMOGRAPHY AND HEALTH CONDITIONS ON NEWBORNANTHROPOMETRY IN FEDERAL TEACHING HOSPITAL GOMBE, GOMBE STATE, NIGERIA

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DECLARATION

I hereby declare that the work in this dissertation titled “**The Effect of Maternal Anthropometry, Demography and Health Conditionson Newborn Anthropometry in Federal Teaching Hospital Gombe, Gombe State, Nigeria.**” was performed by me in the department of Human Anatomy, Faculty of Medicine, Ahmadu Bello University, Zaria, under the supervision of Dr. S.B. Danborno and Dr. W.O. Hamman.

The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this work has been presented for another degree at any institution or elsewhere for the award of any certificate.

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Ahmed HassanDate

CERTIFICATION

The dissertation titled **The Effect of Maternal Anthropometry, Demography and Health Conditionson Newborn Anthropometryin Federal Teaching Hospital Gombe, Gombe State, Nigeriaby Ahmed HASSAN** meets the regulations governing the award of degree of Master of Science in Ahmadu Bello University, Zaria and is approved for its contribution to knowledge and literacy presentation.

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DEDICATION

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ABSTRACT

Maternal characteristics have been reported previously to have impact on the progress and outcome of pregnancy, especially those related to birth weight (BW)and perinatal mortality.

Birth weight plays an important role in infant survival, child development, and adult metabolic diseases. The present study aimed at investigating the influence of maternal anthropometry, demography and health conditions on neonatal or newborn anthropometry in Gombe, Gombe state, Nigeria. A data set of 10,586 singleton live births from 2001-2014 was retrieved from maternity registers at the labour ward, Department of Obstetrics and Gynecology, Federal Teaching Hospital Gombe, Gombe State, Nigeria. Male infants (n = 5660; 53.5 %) while female infants (n = 4926; 46.5 %). The information extracted were:maternal age at the time of delivery, parity, mode of delivery, infant birth weight (BW), placental weight (PW), head circumference (HC), chest circumference (CC), birth length (BL), sex of the infant, Apgar score (AS) of the infant at 1^{ST} and 5^{TH} of life. For mother and infant pair, only two thousand, two hundred and seventy six (2276) infants had information on their mother's health conditions during pregnancy, demography and anthropometric measurementsretrieved from hospital records at the department of Biostatistics and medical records due to lack of accessibility. Maternal health conditions extracted included: diabetes mellitus, HIV infection, malaria infection, blood pressure; maternal anthropometric measurements retrieved included: height, weight, gestational weightgain and body mass index (BMI) and maternal and paternal socio-demographic information retrieved were: place of residence, ethnicity, marital status, occupation, and level of education. Results from this study show that low birth weight birth (LBW) was more frequent among female infants compared to male infants, ($P = 0.001$). Mothers age less than 20 years (<20 years) and greater than or equal to 40 (≥ 40) had more incidence of low birth weight (LBW) delivery than mothers age 20 to 30 years and 31 to 39 years, ($P= 0.001$). Low birth weight was more prevalent in infants born preterm (<37 completed weeks) compared to those born at term (37 completed weeks), ($P = 0.001$). Low birth weight delivery was more prevalent in mothers with no education and least among mothers with tertiary education ($P = 0.001$). It was found that the prevalence of LBW was least among infants that had fathers with tertiary education and more in infants that had fathers with no education, ($P = 0.001$). The mean BW of male and female babies were respectively 3.15 ± 0.53 and 3.01 ± 0.52 ($t = -14.128$, $p < 0.001$). Also, there was significant difference in other anthropometric parameters (BL, HC, CC and PW) between male and female babies with male babies having higher values, ($p < 0.001$). Regarding mode of delivery (MOD), the mean BW and PW of newborns delivered via vacuum delivery were significantly higher than those delivered via caesarean section (CS), forceps and spontaneous vaginal delivery (SVD),

(p<0.001). The BW and PW of newborns delivered to mothers with tertiary education were significantly higher than those delivered to mothers with secondary, primary and no formal education (p<0.001). Also BW and PW of those delivered to married mothers were significantly higher than those delivered to single or unmarried mothers (p < 0.001). Birth weight (BW) and placental weight (PW) of infants delivered to diabetic mothers were higher than those delivered to non-diabetic mothers (p < 0.001). Also, BW and PW of infantsdelivered to mothers who never had an episode of malarial attacked during pregnancy were significantly higher than those inmothers who had at least an episode of malarial attacked (p < 0.001). Also, the BW and PW of infants in mothers that were normotensive and hypertensive mothers were significantly higher than those in hypotensive mothers (P<0.001). Birth weight of infants correlated positively with all maternal anthropometric variables at (P<0.001). Linear regression models for estimating birth weight (BW) of newborns from maternal and newborn anthropometric and demographic variables were generated. In conclusion, male babies or newborns had significantly higher birth weight, placental weight, birth length, head and chest circumferences relative to female babies. Also maternal ill health during pregnancynegatively influenced newborn anthropometry.

CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND OF STUDY

Birth weight (BW) plays an important role in infant survival, child development, and the onset adult metabolic diseases (Kramer, 1987; Barker, 1992; National Population Commission, 2009; Opinion Research Corporation Macro, 2009).Maternal characteristics have been variously shown to impact on the progress and outcome of pregnancy, especially those related to BW and perinatal mortality (Macleod and Kiely, 1988; Voigt *et al.,* 2004; Kazaura *et al.,* 2006; Mohanty *et al.*, 2006; Singh *et al.,* 2009; Amosu *et al.,* 2014). Such maternal factors like age, ethnic group, weight (WT),gestational weight gain (GWG), height, BMI, socio-economic, genetic endowment, and medical conditions (e.g. hypertension, diabetes mellitus, malarial infection, HIV infection, urinary tract infections, malnutrition and anaemia) are strongly associated with foetal complications especially low birth weight (LBW), prematurity and birth asphyxia all of which act individually or in concert with each other to increase neonatal and infant mortality(Voigt *et al.,* 2004; Mohanty *et al.,* 2006; Danborno and Afegbu, 2006; Kazaura *et al.,* 2006; Danborno *et al.,* 2008; Padilhal *et al.,* 2009).In Nigeria, most births are unattended to by a trained birth attendant occurring at home or in settings outside the hospital. Even the few that come to the hospital book very late with little or no window for effective intervention (National Population Commission, 2009).

The World Health Organization had defined low birth weight (LBW) as abirth weight of less than 2,500 grams (WHO, 1992). This practical cutoff point forinternational comparison is based on epidemiological observations that infants weighing less than 2,500 g or 2.5 kg are approximately 25 to 30 times more likely to die than infant with BW exceeding this cutoff, and it increases sharply as BW decreases (Chang*et al.,* 2003). More than 20 million infants worldwide, representing 15.5 percent of all births are born with LBW – 95.6 percent of them in developing countries making low birth weight (LBW) an important infant health problem in many populations (Kazaura, *et al.,* 2006). The 2008 Nigerian Demographic Health Survey estimates the incidence of low birth weight in Nigeria to be 14 percent (140 per 1,000), which however varies considerably across social and geographic areas (NPC, 2009; ORC Macro, 2009).

Maternal height is significantly correlated with gestational age and, birth order is one of the strongest determinants of BW compared to other maternal characteristics **(**Eltahir and Gerd, 2008).It is reported thatmaternal height below 155 cm increases the risk of neonatal death (Baqui*et al.*,1994). This confirms the value of maternal height as a predictor of childhood morbidity and mortality. It is also suggested by Zhang *et al.,*2007 that the slower fetal growth due to short maternal stature appears to be physiologic. It was also pointed out by Veena*et al.*,2004 that the size of the infant at birth is influenced by paternal rather than maternal height while also (Voigt *et al.,*2004) found that the influence of paternal characteristics on infant size at birth is negligible. A study have shown that birth weight increases with birth order(Hirve*et al.*,1994) in India found a 1.3 higher relative risk for LBW in primipara and in Africa (Lawoyin, 2007) found that first born babies had a 3.1 fold higher mortality risk.

The magnitude of the contribution of low birth weight to infant mortality is higher in developing countries given that the survival of such infants is dependent on environmental sanitation, effective post-natal nutrition and rehabilitation, and the availability of medical care (Mondal, 2000). Low birth weight (LBW) remains a public health problem in many parts of the world and is associated with a range of health problems, lasting disabilities and even deaths (Najmi, 2000; Wasunna and Mohammed, 2002; NPC 2004; ORC, 2009). One-half of LBW infants in industrialized countries are born preterm (<37wk gestation), however, in the developing countries these children are born at term but are affected by intrauterine growth retardation that begins early in pregnancy (Ramakrishnan, 2004).

Maternal age and all maternal anthropometric measurements are positively correlated with birth weight (Grimmer *et al.*, 2002). It was also reported by (Karim and Mascie-Taylor, 1997) that birth weight increases with higher maternal education, while in Germany women with the lowest education had significantly elevated risk for small for gestational age newborns (Grimmer *et al.*, 2002). The main non-genetic factors affecting birth weight are: gestation length, smoking, pre-natal health care, maternal nutrition (including alcohol and coffee consumption) and maternal stress. Maternal education can potentially affect all these inputs, and a correlation between BW and maternal education is a robust finding (Behrman and Wolfe, 1989; World Bank, 1993). Maternal education affects BW by improving the probability and /or productivity of health investment. Additionally, maternal education improves the financial resources available to the child directly and indirectly through the choice of partner, timing of fertility, and number of offspring (Chevalier and O'Sullivan, 2007).Socioeconomic status was associated with lower mean birth weights with maternal education and paternal occupation and education exerting the higher influences on newborn anthropometrics (Njokanma, 2013).

At birth, fetal weight is accepted as a parameter that is directly related to the health and nutrition of the mother as well as an important determinant of the chances of the infant to survive and experience healthy growth and development (Nwokocha, 2004).Birth weight also shows a reverse social gradient such that increasing disadvantage is associated with decreasing birth weight (Wilcox, 1992; Berney*et al.*,2000). The prenatal period is one of the most vulnerable in the human life cycle (Koupilova*et al.*,2000). During this period, the mother serves as a gatekeeper and child health is dependent on whether she admits into her own system those elements that are essential to a healthy pregnancy (Kramer *et al.*,2000). These include adequate nutrition,timely medical care and sufficient education to make informed choices on behalf of her unborn child. It is also likely to play a key role in the production of social group's differences in infant survival because it is one of the strongest predictors of infant mortality risk (Kramer, 1987; Podja and Kelly, 2000).

The ability of the fetus to grow and thrive *in utero* depends on the placental function and the average weight of the placenta at term (37 completed weeks) is 508 g or 0.508 kg (Gichangi*et al.*, 1994; Williams *et al.*, 1997). High placental weight (PW) was associated with a poor perinatal outcome, a low Apgar score (AS), respiratory distress syndrome and perinatal death; whereas a low PW was associated with medical complications in the mother (Williams *et al.*, 1997). It was also reported by Barker *et al.*, 1990that altered growth of the placenta was a predictor of maternal medical diseases including cardiovascular disease, hypertension and diabetes mellitus. Other factors such as race and socioeconomic status also affect the PW.Clinical associations with placental weights (PWs) andfetal/placental (F/P) ratio have been documented, for example, small placentas may be associated with trisomies, whereas large placentas may be associated with maternal diabetes (Williams *et al.,* 2000). Disproportionately large placentas (low F/P ratio) could reflect acute placental injury resulting in villous edema or a chronic process leading to placental overgrowth, such as maternal anemia or malnutrition (Godfrey et *al.,* 1991). Disproportionately small placentas (high F/P ratio) may be seen in maternal hypertension, and may result in fetal distress or low AS (Barker *et al.,* 1990). There is a positive correlation between the placental and infant's weight and the ratio of the placenta and foetal weights at term decreases with advancing gestational age (Abubakar*et al.,*2012). Thus, prolongation of pregnancy at term may adversely affect the fetus.

Infant's sex differences, birth to conception interval, gestational age, and Apgar score (AS) are associated with infant BW (Hobcraft *et al.,*1983; Ferraz *et al.,*1988; Finster and Wood, 2005). Boys grow faster than girls from an early stage of gestation, even from before implantation (Pedersen, 1980). A study in Indonesia reported that mean birth weight for male babies were greater than girls at birth (Jane *et al.,* 1989). It was found that birth to conception interval of six months or less was associated with an increased risk of intrauterine growth retardation (Miller, 1989).It was also reported by Macleod and Kiely, (1988)that there is a strong association between birth weight and duration of pregnancy.

Apgar score (AS) is a simple and repeatable method to quickly and summarily assess the health of newborn infants immediately after birth (Apgar, 1953). The five-minute AS is positively correlated with birth weight and is higher in small for gestational age (SGA) infants compared with their appropriately grown counterparts (Stark *et al.,* 1990).

Pregnancy risk factors are all the aspects of pregnancy that endanger the life of the mother and the baby (Mondal, 2000). These factors may include poor nutrition of the woman, child spacing, maternal age (under 15 years and over 35 years), inadequate prenatal and perinatal care, lifestyle behaviors (e.g. smoking, alcohol consumption, drug abuse and unsafe sex), overweight, obesity, AS and poverty (Wardlaw andKessel, 2002). A study by Kazaura*et al.* (2006) reported that several risk factors influence neonatal mortality; these include parity, maternal age, race, marital status, smoking, BW, gestation age, labour complications, antenatal care, previousunfavorableoutcomes (e.g. stillbirth, neonatal deaths), maternal morbidity (e.g. malaria and HIV infection) and poor socio-economic conditions. Poor nutritional status during pregnancy has been associated with irreversible damage to the infant brain and central nervous system leading to poor brain development and intelligence (Kotelchuck, 1994; Koupilova, *et al.*,2000).There is ample evidence that obesity and non-communicable diseases, for instance, cardiovascular diseases start early in childhood (Wardlaw andKessel, 2002).

A unifying framework in research findings is the large maternal and socioeconomic disparities in the birth weight of infants; in line with this, many authors have highlighted the importance of considering social and class factorin addition to biological ones to explain LBW. In particular, significant associations have been found between low socioeconomic status and LBW (Rodríguez *et al.*, 1995; LekeaKaranika*et al.*, 1999). These socioeconomic differences have been found in many countries, even in those where access to prenatal care is universal (Kramer *et al.*, 2001).

While it is important to describe the independent effects of different behavioral and socioeconomic risk factors, we must bear in mind that thesefactors are not isolated events in women's lives, but are a part of many interrelated and complex behavior and environmental risks. (Karim and Mascie-Taylor, 1997; Celik and Younis, 2007). Many of the known determinants of a baby's birth weight are not within a woman's immediate control (Kazaura*et al.*, 2006). Clearly, the relationship between lifestyle risk factors and birth weight is complex and is affected by psychosocial, socioeconomic, and biological factors; it is also clear that birth weight outcomes are socially stratified (Baqui*et al.*, 1994; Grimmer*et al.*, 2002). For many women in the developing world, economic, social and cultural factors make it difficult for them to obtain the necessary food and health care, which are closely interrelated (Klufio*et al.*, 2001). Some researchers consider that health, therefore, may be an important determinant of opportunities in life and this process termed 'selection by health', and suggest that health 'selects' people in different social strata (Jane*et al.*,1989; Godfrey *et al.*,1991;Gichangi*et al.*,,1993; Wadworth, 1999).

Some of the major determinants of birth weight in developing countries include maternal nutritional status at conception, gestational weight gain in accordance with dietary intake, parental socioeconomic status, malaria, anaemia, and chronic infections during pregnancy (Podja and Kelly, 2000; Koupilova*et al.*,2000; Kramer *et al.*,2001). Social demographers (Singh and Yu, 1996) have long emphasized the importance of "nonmedical" barriers –behavioral, social,environment, and economic to good or adverse birth outcomes. Likewise, people’s health occurs within cultural systems that are concerned with broader issues of well-being than addressed by the physician’s concerns with disease and injury (Podja and Kelly, 2000). Other environmental factors that have been identified include socio-cultural traditions and customs concerning pregnancy, access to good quality prenatal care, culturally embedded demands for unlimited numbers of children (Nwokocha, 2004).

This present study is therefore designed to investigate the effect of maternal anthropometry, socio-demography and health conditions during pregnancy on newborn anthropometry in Gombe, Gombe State, Nigeria.A studyof this nature isimportant in view of the persistent high infant and child mortality and morbidity in Nigeria and the need to identify critical variables that are amenable to appropriate intervention to enhance the survival and life chances of children.

1.2 STATEMENT OF RESEARCH PROBLEM

Maternal characteristics such as anthropometry, medical conditions and socio-demography have been reported severally to be associated with infant anthropometry especially low or high birth weight in the case of poor maternal characteristic (Ever *et al.,* 2002; Guyyatt and Robert, 2004; Mohanty *et al.,* 2006; Padilhal *et al.,* 2009; Kumar *et al*., 2011; Amosu *et al.,* 2014). However, studies on the association between maternal characteristics and newborn anthropometry in Gombe State, Nigeria are non-existent.

1.3 JUSTIFICATION OF STUDY

Maternal factors such as age, ethnic group, weight, height, BMI, socio-economic, genetic endowment, socio-cultural, demographic, and medical conditions (e.g. hypertension, malaria, diabetes mellitus, urinary tract infections, HIV infection, malnutrition and anaemia) are strongly associated with foetal complications especially low birth weight, high birth weight (macrosomia) prematurity and birth asphyxia all of which act individually or in concert with each other to increase neonatal and infant mortality (Ever *et al.,* 2002; Guyyatt and Robert, 2004;Padilhal*et al.,*2009; Kehinde *et al.,* 2013).

The burden of low birth weight (LBW)in developing countries like Nigeria is heavy.The 2008 Nigerian Demographic Health Survey estimates the prevalence of low birth weight in Nigeria to be 14 percent (140 per 1,000).Thus, investigating maternal characteristics that critically affect infant birth weight and other infant anthropometric parameters is of great importance, as this will provide caregivers and health planners the necessary data for appropriate intervention to enhance the survival of infants.

1.4 AIM AND OBJECTIVES OF STUDY

1.4.1 Aim of Study

This retrospective study was aimed at investigating the effect of maternal anthropometry, socio-demography and health conditions during pregnancyon thenewborn anthropometry in Gombe, Gombe State, Nigeria.

1.4.2 Objectives of Study

The objectives of the study were to investigate

i. the association between maternal characteristics and birth weight.

ii. sexual dimorphism in anthropometric parameters of the newborn infants.

iii. To see the trend of newborn anthropometric parameters (birth weight, birth length, head and chest circumferences and placental weight) over the course of study period.

iv. the association between maternal and paternal demographic factors (level of education, marital status) on newborn anthropometric parameters.

v. the effect of maternal medical conditions (diabetes mellitus, malaria infection, HIV infection and blood pressure) on newborn anthropometric parameters.

vi. the association between newborn and maternal anthropometric parameters and to

vii. generate predictive equations for the estimation of birth weight.

1.5 RESEARCH HYPOTHESIS

This research is driven by the following hypotheses:

Maternal characteristics (anthropometry, socio-demography and health conditions) would significantly influenced newborn anthropometric (birth weight, birth length, head and chest circumferences).

CHAPTER TWO

2.0 LITERATURE REVIEW

Birth weight (BW)is the first weight of an infant recorded at birth (Barker, 1992). A low birth weight (LBW) infant is defined as an infant whose BW is less than 2.5 kg (< 2.5 kg). Normal birth weight (NBW) is weight at birth ranging from 2.5 kg – < 4.0 kg, while High birth weight (HBW) is a weight of greater than or equals to 4 kg (≥ 4.0 kg) at birth (Campbell *et al.,* 1996). On the other hand infants with birth weight of < 2.5 kg can be classified into Small for gestational age (SGA)and preterm infants. Low birth weight infants can be further classified intoVery low birth weight (VLBW)< 1.5 kg and Extremely low birth weight (ELBW)< 1.0 kg (Padilhal *et al.,* 2009).Infant birth weight can also be classified into weight for a specific gestational age:Small for gestational age (SGA)Weight<10th percentile, Appropriate for gestational age (AGA) Weight 10-90th percentile, Large for gestational age(LGA) or macrosomia Weight>90th percentile (Osullivan *et al.,*1965;Kloosterman, 1954;Kramer, 1987; Evers *et al.,* 2002;Rhee *et al.,*2015).

A baby's low weight at birth is either the result of preterm birth (before 37 completed weeks of gestation) or of restricted fetal (intrauterine) growth (Barker, 1992). Intrauterine growth restriction (IUGR), also known as intrauterine growth retardation, refers to poor growth of a fetus while in the mother's womb during pregnancy (Vandenbosche *et al.,* 1998; White and Cynthia, 2014). The causes can be many, but most often involve poor maternal nutrition or lack of adequate oxygen supply to the fetus (Lawn *et al.,* 2005). At least 60% of the 4 million neonatal deaths that occur worldwide every year are associated with low birth weight (LBW), caused by intrauterine growth restriction (IUGR), preterm delivery, and genetic/chromosomal abnormalities, demonstrating that under-nutrition is already a leading health problem at birth (Lawn *et al.,* 2005). Intrauterine growth restriction can result in a baby being Small for gestational age (SGA), which is most commonly defined as a weight below the 10th percentile for the gestational age (Barker, 1992).At the end of pregnancy, it can result in a low birth weight.Other common factors associated with intra-uterine growth retardation in developing countries, other thanlow energy intake or weight gain during pregnancy and low pre-pregnant weight, include ethnicity,short maternal stature, and malaria infection (Kramer, 1987).

It is well documented that BW increases with parity in man and in other mammals (Osullivan *et al.*,1965). Also, it had been found that parity have stronger association than maternal age on birth weight (Kloosterman, 1954).Among mothers who had full-term births (birth after the 37th gestational week), shorter maternal height was associated with low birth weight(LBW) (Inoue *et al.,* 2016).

On the basis of data from animal studies and cross country comparisons, poor maternal nutrition has been implicated as one of the key" adverse environmental influences in utero", which could lead to compromised fetal and placental growth and adverse long term consequences (Barker, 1992). The observational studies of British women by Campbell *et al.* (1996), Godfrey *et al.* (1996) also suggest that maternal diet is an important determinant of infant and placental size. A LBW is known to be a risk factor for fetal compromise, being a typical finding in cases of chronic placental insufficiency (Golan *et al.*, 1994).

Apgar score (AS) which has been used as the best established index of immediate postnatal health (Heggi *et al.*,1998; Casey *et al.*,2001) may also reflect a complication of pregnancy. A low AS may be related to prematurity, congenital malformations, perinatal infections (Casey *et* *al.*,2001). The AS continues to provide convenient shorthand for reporting the status of the infant and the response to resuscitation. An AS that remains 0 beyond 10 minutes of age may be useful in determining whether additional resuscitative efforts are indicated (Jain *et al.*,1991). Previously, an AS of 3 or less at 5 minutes was considered an essential requirement for diagnosis of perinatal asphyxia. Low AS is associated with increased neonatal morbidity in preterm newborns (Weinberger *et al.*,2000) and increased neonatal mortality among preterm and term infants (Casey *et al.*,2001; Pollack *et al.*,2000). A depressed five-minute AS reflects a host of intrauterine and perinatal insults, some of which are also known or suspected risk factors for neurologic morbidity: hypoxic and mechanical brain trauma, birth defects, non-optimal BW or gestational breech presentation, delivery complications, maternal age and smoking, as well as the newborn poor response to resuscitation prompted by a low one-minute AS (Kliegman, 1996; Viera and Castillo, 2005).

Infants who are smaller for gestational age are at risk of death during the prenatal period (Abdulkarim, 1981) with prenatal mortality rates of four to eight times higher than those of infants whose weights are appropriate for gestational age (Seed, 1984). In addition such infants have been associated with other diseases even as adults.Risk factors for the occurrence of SGA births have not been completely clarified. Some risk factors known to be associated with occurrence of low birth weight (LBW) have not been studied specially for their association with SGA births, even though a short inter-pregnancy interval has been identified to be a risk factor for the occurrence of LBW infants (Eisner *et al.*, 1979).

One of the most consistent risk factors for delivery of a LBW infant is a history of LBW in previous pregnancy (Bakketeic, 1970). The survival of very LBW infants (less than 1500 gm) in developed countries has improved markedly in recent years (Stewart *et al.*, 1981). And the factors responsible for this improvement are related to advances made in the clinical care for the mother and their infants such as increased use of fetal monitoring and regionalized care for mothers and infants who are at high risk (Harris *et al.,* 1981). Infants of LBW are often handicapped later in addition, if they are growth retarded (Gross *et al.,* 1978).Weight gain during pregnancy is associated with fetus, development of maternal reproductive tissue increased blood volume and extra-cellular fluid and increased in maternal energy stores (Morgan, 1997). Consequently, maternal weight gain is often monitored carefully and is encouraged during prenatal care in order to improve the chances of favourable pregnancy outcome. Current standards for adult gravidas recommend total gains of between 9kg and 14kg to minimize the risk of LBW and infant morbidity (Hytten, 1980), although it has been suggested that larger total weight gains may be appropriate for pregnant adolescents (Horon and Strobino**,** 1983).

Maternal anaemia in pregnancy increase the risk of LBW babies (Ahmad *et al.*,2011).High caffeine intake during pregnancy was associated with a significant increase in the risk of LBW, and this risk appears to increase linearly as caffeine intake increases (Rhee *et al.*,2015).

2.1 PHYSIOLOGY OF FETAL GROWTH AND DEVELOPMENT

Fetal growth and development is dependent on adequate transfer of nutrient and oxygen across the placenta, by itself is dependent on adequate maternal nutrition and placental perfusion(Golan *et al.*, 1994).Fetal hormones have an important role in fetal development they affect the metabolic rate, growth of tissues and maturation of individual organs (Barker, 2002; White and Cynthia, 2014). For example: Insulin –like growth factors (IGFs) co-ordinate a precise and orderly increase in growth through late gestation. Insulin and thyroxin are required through late gestation to ensure appropriate growth in normal and adverse nutritional circumstances (fetal hyperinsulinemia as in diabetes mellitus (DM) result in macrosomia due excessive fat deposition, while in growth restricted fetuses fetal insulin levels are low) (Kulkarni *et al.*, 2009).

Lack in thyroid hormone produces deficiency in skeletal and cerebral maturation (cretinism), also there is delayed surfactant production (Barker, 2007). Cortisol has a limited role in stimulating growth, but it is essential in: (1). lung compliance and surfactant release, which ensure that spontaneous breathing can occur at birth, (2). In the fetal liver, it induces beta receptor and glycogen deposition to maintain a glucose supply to the neonate after delivery and (3). In the gut it is responsible for villus proliferation and induction of digestive enzymes, which enable the neonate to switch to enteral feeding after birth (Chang *et al.*, 1984).

The average birth weight is about 3.5 kg at the end of normal pregnancy (De Grauw *et al.*, 1986). One third (1/3) of the eventual birth weight is reached by 28 completed weeks, half (½) by 31 completed weeks, two third (2/3) by 34 completed weeks.Each baby has its own optimal growth potential, which is predictable from physiological characteristics known at the beginning of pregnancy; those factors are:Pre-pregnancy weight and maternal booking weight (increasing with maternal weight), Maternal height(increasing with maternal height), Maternal age and parity increased with mother > para2, Fetal sex (male >female), Paternal height (Boulet *et al.*, 2003).

Maternal smoking affect the birth weight significantly adversely, it is consistent and dose dependent (Wardlaw andKessel, 2002).Growth restricted fetuses are those who have failed to achieve their growth potential,they have significantly higher perinatal mortality and morbidity rate. Growth restricted fetuses/infants are more likely suffer from:intrauterine hypoxia/asphyxia, stillborn, hypoxic, ischaemic encephalopathy (sezuire), multi organ failure, neonatal hypothermia, hypoglycaemia, infection and necrotizing enterocolitis, cerebral palsy, in adulthood they are at greater risk of hypertension and type 2 diabetes mellitus (Barker, 1992; Barker, 2002).

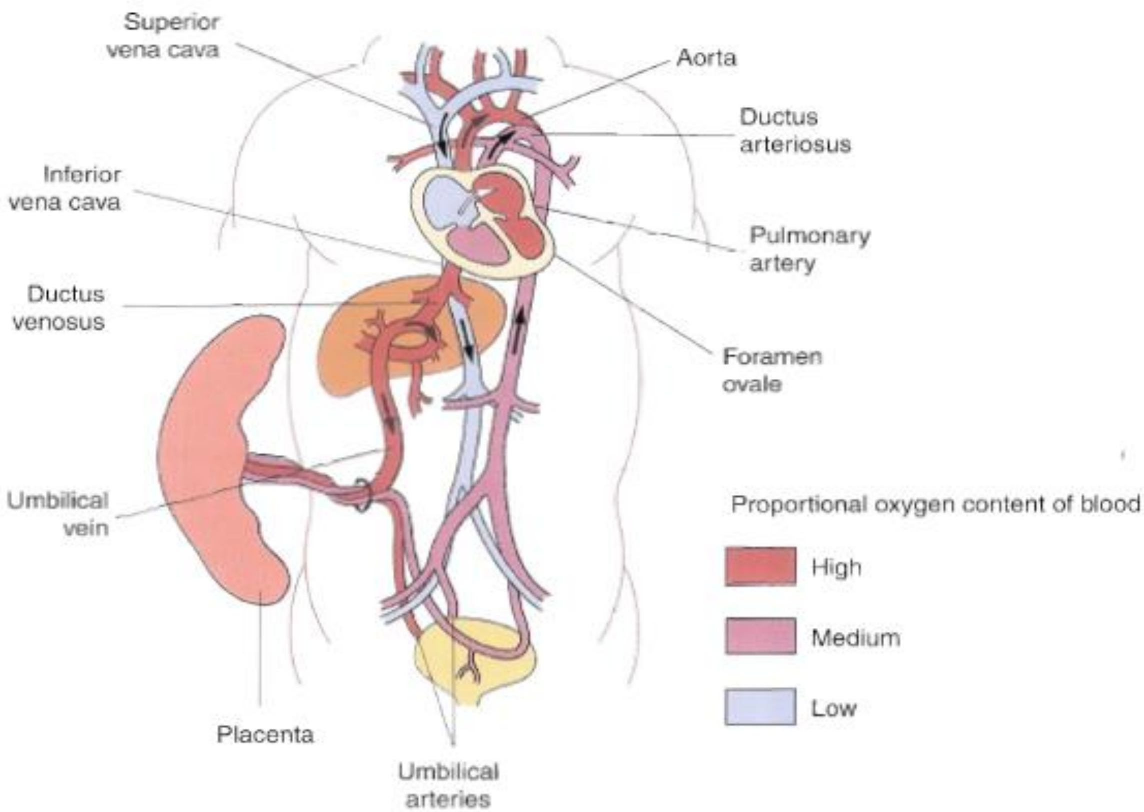

Figure 2.1: fetal circulation system

Source: American Heart Association

Fetal circulation is quite different from that of the adult as follows:oxygenation occurs in the placenta not in the lung, the right and left ventricles work in parallel rather than in series, the heart,brain and the upper body receive blood from the left ventricle, while the placenta and lower body receive blood from both right and left ventricles.

There are modifications in fetal vascularity that ensure that the best, oxygenated blood from the placenta is delivered to the fetal brain, these are:the ductus venosus that shunts blood away from the liver, the foramen ovale, shunts blood from right to left atrium, the ductus arteriosus that shunts blood from the pulmonary artery to the aorta. Oxygenated blood from the placenta returns to the fetus through the umbilical vein, which is divided into two main branches:One supply the portal vein in the liver, another narrow vessel called ductus venosus which joins the inferior vena cava as it enters the right atrium. Fifty percent (50%) of oxygenated blood will to the portal system and 50% will pass to the ductus venosus.The ductus is a narrow vessel and a high blood velocities are generated within it.The streaming of ductus venosus blood, together with a membranous valve in the right atrium (the crista dividens), prevents mixing of the well-oxygenated blood from the ductus venosus with the desaturated blood of the inferior vena cava. The ductus venosus stream passes across the right atrium through a physiological defect in the atrial septum (foramen ovale) to the left atrium, then the blood will pass to the left ventricle through the mitral valve and hence to the aorta.

Fifty percent (50%) of the left ventricle blood goes to the head and upper extremities, the remainder passes down to the aorta to mix with blood of reduced oxygen saturation from right ventricle.Blood from inferior vena cava and superior vena cava is directed across the tricuspid valve to the right venricle.Only a small portion of right ventricle blood passes to the lungs as they are not functional.Most of the right ventricle blood is directed through a narrow vessel (ductus arteiosus) into the descending aorta below the origin of head and neck vessels from the aortic arch.The desaturated blood from the right ventricle passes down the aorta to enter the umbilical arterial circulation and hence to the placenta.Prior to birth, the ductus remains patent due to production of the prostaglandin E2 and prostacyclin which act as local vasodilator, so administration of cyclo-oxygenase inhibitor will lead to premature closure of the ductus.At birth,the cessation of umbilical blood flow causes cessation of flow in the ductus venosus, a fall in the right atrium pressure and closure of the foramen ovale.Ventilation of the lungs opens the pulmonary circulation, with rapid fall in pulmonary vascular resistance.The ductus arteriosus closes functionally within a few days of birth.

2.2**REPORTED WORKS BY OTHER INVESTIGATORS**

Recent reports suggest that advanced maternal age may be associated with increased risk of low birth weight among singletons, but this effect has been described only among African American women (Collins and David, 1990; Star field *et al.*, 1991; Geronimus, 1996) with Geronimus using the term "weathering" for the deterioration in reproductive health status over the childbearing years among African American women (Geronimus, 1992). Although these findings suggest that maternal age operates differently for African American and White women, studies of birth outcomes generally treat this factor as a covariate or confounder, rather than exploring the joint impact of maternal age, race, and other risk factor. The best period to have a baby of high BW is 21-30 years (Singh *et al*, 2009). Pregnancy outcomes are affected by some maternal factors as well as nutrient intake (Laraia *et al.,* 2006). These factors are age, occupation, family, pregnancy experiences and morning sickness (Freisling *et al.*, 2006; Laraia *et al.*, 2006). It has been shown that BW of babies' tallies with the BW and adult size of both its parents especially with the weight of the mother, it has also been seen that the paternal birth length has a sharper effect than the maternal birth length on the babies (Veena *et al*, 2004).

Heredity plays a large role in infant size, ordinarily, parent descended from lineage of big people breed infants of large size, and those with small parents and grandparents produce babies of less than average birth weight (Haste *et al.*, 1990; Anahita *et al.*, 1998).

Coad *et al.* (2005) found significantly lower energy and fat intakes in women who suffered from morning sickness, and women who experienced morning sickness during pregnancy had significantly lower intakes of protein, carbohydrates, fiber, vitamin E and D, B vitamins (excluding folic acid), iron and zinc. They suggest, therefore, that interventions are needed to alleviate the symptoms in pregnant women who suffer from morning sickness.

Polednak found that infant mortality rates even among African Americans differed by degree of racial segregation, possibly because highly segregated areas are characterized by extremely concentrated poverty, inadequate health care, substandard housing, crime, and other stressors (Polednak, 1997). Consistent with this finding, is a report that rate of LBW (specifically, intrauterine growth retardation) among low-income African Americans are higher for women who reside in more violent, as compared with less violent, communities (Collins and David, 1997). Finally, a number of studies have shown that African American women born in the United States have higher rates of LBW infants than African American women born outside the United States, suggesting the importance of nativity in addition to community of current residence (Caral *et al.*, 1990; David and Collins, 1997).

Rauh *et al.*(2001) reported a study that assessed the contribution of age and other risk factors to racial disparities in rates of moderately low birth weight (MLBW; 1500-2499 g) and very low birth weight (VLBW; <1500 g). Logistic regression models were developed to determine the effects on MLBW and VLBW of maternal age, race, and poverty, adjusting for birth order, smoking, substance abuse, marital status, and educational level. The sample consisted of 158, 174 singleton births to US-born African American and White women in New York City between 1987 and 1993. The effects of maternal age on MLBW varied by race and poverty, with the most extreme effects among poor African American women. The effects of maternal age on VLBW also varied by race, but these effects were not moderated by poverty. Community poverty had a significant effect on MLBW among African American women, but no effect on VLBW. The adverse effect of older maternal age on MLBW and VLBW did not vary with community poverty. Older maternal age is associated with reduced BW among infants born to African American women, and the age effect is exacerbated by individual poverty.

Chauhan *et al.*(2001) sought to describe the peripartum outcome of women weighing >300 pounds (135kg) who were candidates for trial of labor after a prior cesarean delivery. All pregnant women who weighed in excess of 300 pounds and had a prior cesarean delivery were included in this prospective investigation. Student t test, 2 analysis, or Fisher exact tests were used. Odds ratios and 95% confidence intervals were calculated. P<0.05 was considered significant. During a 2-year period, 69 patients met the inclusion criteria; 39 (57%) underwent an elective repeat cesarean delivery. The demographics of age, race, gravidity, maternal weight, and preexisting medical conditions are similar for the two groups. Vaginal birth after prior cesarean delivery occurred in 13% (4/30). Reasons or failure included a labor arrest disorder in 46%, fetal distress in 38%, and failed induction in 15%. The rates of endometritis and wound breakdown were higher in the women undergoing trial of labor (30% and 23% respectively) than in those undergoing repeat elective cesarean delivery (20% and 8%). The combined infectious morbidity rate was significantly higher for women attempting trial of labor (53%) than those undergoing elective repeat cesarean delivery (28%; odds ratio 1.78, 95% confidence intervals 1.05, 3.02). The success rate for a vaginal delivery in the morbidly obese woman with a prior cesarean delivery is less than 15%, and more than half of the patients undergoing a trial of labor have infectious morbidity.

Recent reports indicate that altered growth in utero is a predictor of adult-onset diseases that include hypertension (Barker *et al.*,1990; Curhan *et al.*,1996; Whincup *et al.*,1999), coronary heart disease (Leon *et al.*,1998; Forsen *et al.*,1999; Barker *et al.*,1999) and diabetes (Plante, 1998; Rich-Edwards *et al.*,1999).

Roth*et al.*(2003) observed that there was a decline in placental-weight ratio (i.e. the ratio of PW to infant weight at birth times 100%) with increasing gestational age in the study of placentas of 1,621 singleton births of at least 28 weeks gestation in a Ukranian city during 1993-1994 and it is similar to that described by many others (Sinclair, 1948; Molteni *et al.*,1984;Leyland and Pritchard, 1992; Dombrowski *et al.*,1994). The placenta and its relationship to the size of the infant at birth have been investigated for more than 100 years (Roth *et al.*, 2003).

Recent reports also suggest that malaria commonly occurs in mothers in Malawi and is likely to have restricted placental function (Brabin *et al.,* 2004). The combined effect of maternal malaria and HIV infection would synergistically act to influence fetal growth especially late in gestation (Kalanda *et al.*,2005). Seasonality of mean BW variation has been associated with ecological cycles linked to subsistence, for example, among rural Gambian agriculturalists, seasonal variation of BW has been associated with maternal under nutrition during the last trimester of pregnancy and this usually takes place during the wet season, a time of heavy workload and low food availability (Prentice *et al.*,1987). Ventura (1996) found that there is ethnic variation in the rates of LBW. The prevalence of LBW among African-Americans is 13.1% and doubled that of Whites which is 6.4%. (Ventura,1996). Strobino *et al.*(1995) studied three hypothesized explanations for reduced BW of infants born to United State adolescent mothers; social disadvantage, biological immaturity and unhealthy behaviours during pregnancy. Smith *et al.*(2000) from his study on teenage mothers in the United State found that there was a strong relationship between young maternal age and a high infant mortality and also the relationship between young maternal age and high prevalence of LBW.

The causes of LBW are multifactorial (Kamalados *et al.*,1992): it is associated with sex of baby (Pakrasi *et al.*,1985; Oni, 1986; Kramer, 1987), maternal hemoglobin level during pregnancy, hard manual labour (Ghosh *et al.*,1977), maternal nutrition (Fredrick and Adelstein, 1978), economic condition (Pakrasi 1985; Dhall and Bagga, 1995), maternal height, antenatal care (Rehan, 1982; Kamalados *et al.*,1992), parents education (Makhija and Murthy, 1990), maternal weight (Mavalankar *et al.*,1994), tobacco consumption (Varma, 1983), place of residence (Makhija and Murthy,1989), season of the year, ethnicity (Bantje, 1983) and most importantly mother's age and parity (Kramer, 1995).

Samiran *et al.* (2006) shown that young mothers (age < 19 years) delivered a high rate of LBW baby than those mothers age 19 years onwards and the rate of LBW decreased with the increasing age of the mothers after 18 years. The rate of LBW also decreases with increasing parity. It is now universally acknowledged that maternal age is an important factor influencing the incidence of LBW (Samiran *et al.*,2006). Fetuses who suffer from growth retardation have higher perinatal morbidity and mortality (Williams *et al.*,1982; Villar *et al.*,1984; Tylor *et al.*,1989).

An important and somewhat underinvestigated area of inquiry is women's mental health during pregnancy and its impact on perinatal outcomes. There are animal studies supporting that exposure to stressful environment during pregnancy is associated with adverse perinatal outcomes (Istvan, 1986; Paarlberg, 1995). Anxiety may also cause pregnant mothers to engage in unhealthy behaviours during pregnancy such as smoking, drinking alcohol, or not attending prenatal visits, either in an attempt to reduce symptoms; these unhealthy behaviours then increase the risk of experiencing adverse pregnancy outcomes like LBW, preterm delivery, low 5-minute Apgar (Istvan, 1986; Paarlberg, 1995). Another possibility is that experiencing high levels of anxiety may lead to an increase in stress hormones which can lead to decreased uterine blood flow, as well as induction of preterm labour (Johnson, 2003).

Recent report by Premkumar *et al.*suggest that smoothedpercentile curves for birth weight by gestational age increased progressively till 38 weeks of gestation and levels off thereafter and also, Compared with dichorionic twins, monochorionic twins had lower birth weight for gestational age from after27 weeks of gestation (Premkumar *et al.*,2016).

A Statistics in Medicine paper by Bollen *et al.* (2016) examined favorable fetal growth conditions (FFGC) as a latent variable. Their study of Filipino children from Cebu provided evidence consistent with treating FFGC as a latent variable that largely mediates the effects of mother's characteristics on BW, BL, and gestational age (GA).A current study by Camerota and Bollen, in North Carolina and Pennsylvania confirms and replicates the existence of a latent variable representing favorablefetal growth conditions which underlies the relationship between maternal characteristics andchild birth outcomes (Camerota and Bollen, 2016).

In a prospective study of women in the USA, Song *et al.*,reportedthat LBW was significantly associated with increased type 2diabetes risk later in life. They also found that insulin resistancemediated a considerable amount of the total effect on type 2diabetes risk due to LBW, and this effect was further mediated bylow sex hormone binding globulin (SHBG) concentration, elevated blood E-selectin level andincreased systolic blood pressure (Song *et al.*,2014).

A baby's BW is related to BW of both parents and more strongly through the maternal line (deStavola *et al*., 2015); Women born with LBW have a higher risk of also having LBW babies. Whenthese femalebabies enter motherhood they are at risk of having hypertension, diabetes and delivering babieswith LBW (deStavola *et al*., 2015). Khan *et al.* (2016) suggested that LBW is associated with a group of factors which may be regarded as high risk factors, these include: low socio economic status, anaemia, primiparity, short maternal height and less than average weight (<50kg).Only three parental factors were needed to substantially predict offspring birth weight, these include mid-parental (paternal + maternal/2) weight which was the most explanatory variable, followed by parity, and then maternal weight (Taiwo and Akinde, 2012).

A recent study conducted by Lu *et al.* (2016)amongst Chinese pregnant women found that comparedto a traditional Cantonese diet high in cereals, eggs, and Cantonese soups, a diet high in fruits, nuts,and Cantonese desserts might be associated with a higher BW, while a varied diet might beassociated with a greater BW and also a decreased risk of having a small for gestational age (SGA) baby. Pregnant women are recommended to consume different kinds offood, and increase the proportion of vegetables and fruits in their diet, especially for those who areyounger and have low socio-economic status (Lu *et al.*, 2016).

The BW of neonates was apparently related directly to their nutritional status (Kensara and Azzeh, 2016). The correlation was clear as the minimum BW was combined with the worst nutritional conditions as was obvious in terms of low anthropometric measures and the abnormal biochemical results (Kensara and Azzeh, 2016). No correlation was found between low birth weight of the neonates and complete blood count (CBC) parameters (Cunningham *et al.,* 2010; Kensara and Azzeh, 2016).

A study by Amosu *et al*. (2014) in Nigeria indicated that women at risk of delivering low birth weight babies include;primiparous mothers, illiterate or poorly educated mothers, women engaged in menial jobs and petty trading, low maternal monthly income, maternal age (20 years and below), mothers with past history of previous LBW deliveries, abortions and perinatal death. The fact that better educated pregnant mothers delivered lesser number of LBW newborns maybe due to their increased awareness as regards available medical services which influenced their health seeking-behaviour and nutritional status (Amosu *et al.,*2014).

Independent of medically relevant gestational diseases or malformations, women at risk of poverty had increased chances ofhaving an LBW baby (Altenhoner *et al.,* 2016).Women with lower secondary school education or less were more likely to give birth to a child with a birth weight of 2500g or less, and this was independent of relevant illnesses, malformations or damaging health behaviours (Koller *et al.,* 2009; Wiist, 2015; Altenhoner *et al.,* 2016).

Martinez *et al.*(2015)reported that the average weight of neonates of both sexes rose from week 30 to week 42 and the coefficient of variation between weeks 34 and 42 was 11.6%–19% in girls and 12.1%–21.3% in boys. Also, the 10th percentile value at 36 weeks of gestation was 2140 g for girls and 2200 g for boys. For girls, cutoff points for the 10th percentile (small for gestational age infant) were higher at 34–42 weeks and for boys at 36–42 weeks (Martinez *et al.,* 2015).

Dahlui *et al.* (2016) reported that paternal employment, geopolitical zone, parity, number ofpregnancies, and maternal weight were the significant factorsfor low birth weight (LBW) in Nigeria. Even though LBW is influenced by a multiplicity of factors, the incidence of LBW could bereversed if maternal risk factors are detected earlier andappropriate prevention strategies are delivered to the high-risk group (Dahlui *et al.,* 2016).

A UK Biobank study conducted by Day *et al.* (2015)reported that BW, age at menarche and height, but not BMI, werehighly significantly associated with season of birth. Individuals born insummer (June–July–August) had higher mean BW,later pubertal development and taller adult heightcompared to those born in all other seasons (Day *et al.*,2015) which is similar to the reports of (Chodick *et al.*,2007; Strand *et al.*,2011). Concordantly,those born in winter (December–January–February) showed directionallyopposite differences in these outcomes. A secondary comparison of theextreme differences between months revealed higher odds ratios [95%confidence intervals (CI)] for LBW in February vs. September, for early puberty in September vs. Julyand for short stature in December vs. June (Day *et al.,*2015). The above associations were also seen with total hours of sunshine during the second trimester, but not during the first three months after birth. Additional associations were observed witheducational attainment; individuals born in autumn vs. summer were morelikely to continue in education post age 16 years orattaina degree-level qualification (Day *et al.*, 2015).

A study conducted by Racape *et al.* (2016) in Belgium indicated that, compared to Belgians, all migrant groups were observed to have an increased risk of perinatal mortality, despite lower rates ofLBW in some nationalities. Immigrant mothers with the Belgiannationality had similar rates of perinatal mortality to women of Belgian origin and maintained their protectionagainst LBW (Racape *et al.*,2016).

Jeschke *et al.*(2016)showed that an overall 180 days survival rate of preterm infants with a BW<1,500 g admitted to neonatal intensive care units in Germany 2008–2012 of 89.1%, with 14.1% of deaths occurring between 30 and 180 days of life.Rates of survival strongly declined alongside decreasing birth weight, from 97.9% in 1,250–1,499 g BW infants to 38% in infants below 500 g BW, while the rates of surgically treated major morbidities in survivors increased from 1.7 to 32%; (Jeschke *et al.,* 2016) which is consistent with the report of Stichtenoth *et al.* (2012).

Holle Greil in 2006 studied the pattern of sexual dimorphism from birth to senescence in Germany. He reported that Girls are shorter at birth, but they increase in length at higher rates thanboys and even temporarily overgrow the boys up to age 12. Thereafter, males show an obvious growth advantage leadingto some 6 to 9% more length in adult males. In contrast, female circumferences are always smaller, from birth to senescence. Though, the differences between the sexes are low in circumferences, up to age 13, sexual dimorphism increases to17% in the thoracic circumference at adulthood. Sexual dimorphism in weight and BMI is comparably with that inlength measurements while subcutaneous fat and total body fat content are always higher in females. There is sexual difference in anthropometric parameters at birth between male and female neonates (Martinez *et al.,* 2015).Boys grow faster than girls from an early stage of gestation, even from before implantation (Pedersen, 1980). Study in Indonesia, it was seen that mean BW, BL, HC and CC for male babies were greater than girls at birth (Jane *et al.,* 1989).

2.3BIRTH WEIGHT AND FACTORS ASSOCIATED WITH IT

2.3.1Cardiovascular Diseases and Birth Weight

Epidemiologicalstudies of populations' in England and Wales have shown an association between small body size at birth and increased risk of cardiovascular disease in adulthood (Barker *et al.,* 1989; Barker, 1998). These findings have been replicated in the United States (Rich Edwards *et al.,* 1997), Sweden (Leon *et al.,* 1997), (Finland Eriksson *et al.,* 1999), The Netherlands (Law and Shiel, 1996), Croatia (Kolacek *et al.*, 1993), India (Stein *et al.*, 1996), and Japan (Law and Shiel, 1996). Studies of BWs of relatives (Penrose, 1954; Morton, 1955), together with evidence from animal crossbreeding experiments (Walton and Hammond, 1938; Wilson *et al.,* 1998; Giussani *et al.*, 1999), have led to the conclusion that the predominant influence on fetal growth is the intrauterine environment rather than the fetal genotype. These observations have led to "the fetal origins hypothesis," which proposes that cardiovascular disease and type II diabetes originate through adaptations that the fetus makes when it is under nourished. These adaptations, which include slowing of growth, permanently change the structure and function of the body (Barker, 1995).

Coronary heart disease remains the leading cause of mortality and morbidity in the world (WHO, 1997).Landmark epidemiological studies have identified key factors in adult life that increase the risk of coronary events, (Gordon and Kannel, 1971; Dawber, 1980)and yet, these still only account for a proportion of the disease burden (Rose, 1985).Recently, evidence has accumulated that cardiovascular disease is also predicted by antenatal events (Barker, 1992; Barker *et al.*,1993; Barker *et al.*,1993).LBW has been related to mortality from coronary disease and to the development of hypertension and diabetes (Barker *et al.*,1989; Hales and Barker, 1992; Martyn *et al.,*1995).These associations may be due to an effect of prenatal growth directly on the pathogenesis of early atherosclerosis, by "programming" the development of risk factors (Barker, 1992), or due to altered "stability" of established atherosclerotic lesions. Vascular endothelial dysfunction is a key event early in atherosclerosis and is important in the development of all the cardiovascular diseases associated with early growth(Harris and Macleod, 1988; Panza *et al.,* 1990; Ross, 1993).LBW is associated with reduced flow-mediated dilation (coefficient=0.18 kg^{-1}, 95% CI 0.004 to 0.35, P=0.04) but not with endothelium-independent dilation (Leeson *et al.,* 2008).Also LBW is associated with endothelial dysfunction in young adults. This is most marked in individuals with lower risk factor profiles and may be relevant to the pathogenesis of atherosclerosis in later life (Leeson *et al.,* 2008).BW has also be reported to be associated with subsequent growth, and it has been suggested that the association with cardiovascular disease may be due to factors that influence postnatal growth (Barker *et al.,* 1989; Taylor *et al.,* 1997; Lucas *et al.,* 1999).Genetic factors and in utero growth are of particular interest because of their consistent associations with coronary heart disease mortality and risk factors (Barker *et al.,*1989; Barker, 1992;Hales and Barker, 1992; Barker *et al.,* 1993; Barker *et al.,*,1993;; and Martyn *et al.,* 1995).

The "Developmental origin of coronary heart diseases" proposes that under nutrition in utero permanently changes body functions and metabolism leading to an increased risk of coronary artery diseases (CAD) in adult life (Barker, 2007). The association between LBW and risk factors for CAD, hypertension and type 2 diabetes reported in various longitudinal studies (Frankel *et al.,* 1996 and Leon *et al.,* 1998) is in line with the developmental theory. Some studies, however, suggest that birth weight may not be a major risk factor for development of hypertension and cardiovascular disease (Vestbo *et al.,* 1996 and Rabia *et al.,* 1999).Gender differences in the association between BW and risk factors for CAD have been reported in some studies (Kolacek *et al.,* 1993 and Davis *et al.,* 2004), but not in others (Lawler *et al.,* 2002). Wilkins and Murphy, (2006)have suggested that gender-specific genes affecting insulin sensitivity are responsible for the gender difference in birth weight: females would be genetically less responsive to the trophic effect of insulin resulting in a lower birth weight. Insulin resistance would be a risk factor for CAD.Hormonal differences between sexes influence intrauterine development and may interact with genetic and environmental factors resulting in a different pattern of susceptibility in adult life to coronary artery diseases (Wilkins and Murphy, 2006).

Birth weight has been reported to be inversely associated with cardiovascular disease risk factors such as raised blood pressure, dyslipidemia, and glucose intolerance (Mckeigne *et al.,* 1998 and Huxley *et al.,*2000). However, only a small number of studies have looked at the association between BW and coronary heart disease (CHD), and even fewer have looked at this association with stroke (Lawlor *et al.,* 2004). Most,although not all, studies to date have demonstrated an inverse association between birth size and cardiovascular disease outcomes (Barker *et al.,* 1989;Eriksson *et al.,* 1994; Gunnarsdottir *et al.,* 2002; Lawlor *et al.,* 2004).Studies have been unable to assess the potential confounding or mediating effects of maternal and pregnancy characteristics, which affect both BW and cardiovascular disease risk (Sattar and Greer, 2002). Furthermore, with the exception of 1 study conducted in India between 1934 and 1951(Stein *et al.,* 1996), all studies have examined the association among populations born before the 1940s in Europe or the United States, with most being in populations born before the 1930s. Environmental factors, which might be relevant to the association between BW and cardiovascular disease, have improved between the early 1900s and the 1950s(Charlton, 1997).

For example, between 1901 and 1905, infant mortality was 130 per 1000 in Scotland; by 1941 to 1945, it had decreased to 80, and a marked decline over the subsequent decade led to a rate of 37 deaths per 1000 by 1951 to 1955 (Charlton, 1997).

A Brazilian study by Alves *et al.* (2016) reported that LBW did not increase cardiovascular disease risk factors in young adults in the second decade oflife and states that the absence of an association between LBW and poorhealth outcomes among adolescents in a low averagesocioeconomic status population from a capital in theBrazilian northeast corroborates previousfindings in other countries with low average socioeconomic status.

2.3.2**Birth Weight and Intelligent Quotient**

Many studies have shown that children born with LBW (<2500 g) have shown deficits in average intelligence test scores at school age (Breslau, 1995). Within the LBW range, children who are smaller at birth have larger deficits relative to those closer to normal BW (McCormick *et al.*, 1992; Breslau *et al.*, 1994). The effect seems to be similar for both performance and verbal IQ (Breslau, 1995). Recent studies, as well as some earlier reports, have suggested that the direct relation of BW to measured intelligence continues well into the normal range of BW (Record and Mickeown, 1969; Hardy and Mellitis, 1977; Breslau *et al.*, 1996; Sorensen *et al.*, 1997; Richards *et al.*, 2001), However, these studies have not fully controlled for potential confounders**,** the most important of which is family social environment.

Record and Mckeownfound an association between birth weight and verbal reasoning scores in a birth cohort overall, but not within siblings, suggesting that the association was confounded by family environment (Record and McKeown, 1969). A study of the Dutch famine though showed that maternal starvation during pregnancy lowered birth weight but not IQ (Stein *et al.*, 1975).

It is less clear, however, whether birth-weight variations within the normal range are associated with differences in intelligence (Shenkin *et al.,* 2004). Most investigators have focused on infants born premature or small for gestational age or have included these risk groups in the sample without presenting specific analyses of birth-weight variations within the normal range (Shenkin *et al.,* 2004). Thus, the findings of those studies may not be fully representative of the great majority of infants, who are born at term at a weight appropriate for their gestational age (Shenkin *et al.,* 2004; Newcombe *et al.,* 2007; Broekman *et al.,* 2009). In 6 studies of children born at term at a weight higher than 2,500 g, reviewed in 2004, there was a small, positive correlation between BW and childhood cognitive ability (Shenkin *et al.,* 2004). Later studies of the relation between birth-weight variations within the normal range and childhood intelligence gave mixed results (Tong *et al.,* 2006; Lawlor *et al.,* 2006; Newcombe *et al.,* 2007; Yang *et al.,* 2008; Broekman *et al.,* 2009). Among the studies that examined effects of birth-weight variations within the normal range on childhood intelligence,3 studies were identified in which comparisons between siblings were made (Matte *et al.,* 2001; Lawlor *et al.,* 2006; Yang *et al.,* 2008). Only 1 of these showed a significant within-family association (Matte *et al.,* 2006), and in that case, the association was seen only in the male sibling pairs (Matte *et al.,* 2001). 5 studies were identified in which the relation between birth-weight variations within the normal range and adult intelligence was analyzed (Martyn *et al.,* 1996; Sorensen *et al.,* 1997; Eide *et al.,* 2007; Shenkin *et al.,* 2009).

Comparing siblings brought up in the same family represents a powerful method of controlling for familial and parental characteristics, since the relation between differences in BW and intelligence within sibships (the within-family effect) cannot be confounded by factors shared by the siblings (Carlin *et al.,* 2005). In addition, studies of siblings may provide information about between-family effects (Begg and Parides, 2003).

Newborns with extremely low birth weight (ELBW, <1000 g) experience significant health problems such as delayed growth, recurrent infections, frequent hospitalizations, and neurological alterations (Hirata *et al.,* 1983; Hack and Fanaroff, 1986; Lipper *et al.,* 1990; McCornick *et al.,* 1992; Stevenson *et al.,* 1998). Neurological disorders such as cerebral palsy, epilepsy, mental retardation, deafness, and blindness are frequently found in follow-up (Vohr *et al.,* 2000; Hack *et al.,* 2000; Hoekstra *et al.,* 2004). However, evaluation of upper cortical functions such as language, reading, writing, and numerical calculations require a longer follow-up period in carefully selected samples of children without neurological disability (Klein *et al.*, 1985; Klein *et al.,* 1989; Ross *et al.*, 1991; Teplin *et al.*, 1991; Hack *et al.*, 1994; Breslau *et al.*, 1996; Johnson and Breslau, 2000).

It is estimated that approximately 25% of survivors in neonatal intensive care units (NICUs) have more than one intelligence quotient (IQ) mean and standard deviation scores below the general population (Saigal *et al.*, 1990; Saigal *et al.,* 1991; Kitchen *et al.,* 1992; Johnson *et al.*, 1993; Breslau *et al.,* 1994; Sorensen *et al.,* 1997). Some studies have reported that extremely low birth weight (ELBW) infants have lower mean IQ scores than newborns with higher birth weights; as IQ scores increase, limitations in performing daily life activities decrease with higher birth weights infants (Sorensen *et al.,* 1997). Other studies reported that only 27% of ELBW children are competitive in the academic field. However, other studies do not support this observation (Moolhom-hansen *et al.,* 2002), and this remains an open question.

2.3.3Low Birth Weight and the Risk of Type II Diabetes

According to epidemiological studies in the U.K and other countries, individuals with LBWs often develop insulin resistance-based disorders (Hales *et al.*, 1991; Barker *et al.*, 1993; Valdez*et al.*, 1994; Lithell *et al.*, 1996; Rich-Edwards *et al.*, 1999) in a Research carried out among Pima Indians and Taiwanese school children, a higher prevalence of type II diabetes was observed in individuals with both low and high birth weights (U-shaped relationship of birth weight and diabetes) (McCane *et al.*, 1994; Wei *et al.*, 2003).

Anazawa *et al.* (2003) reported a study they conducted among the Japanese to check for the relationship between low birth and type II diabetes. They found that the prevalence of LBW in the 301 diabetic subjects, including those in the occupational cohort and the 1,823 non diabetic subjects in the occupational cohort, was 18.6% (56 of 301) and 9.8% (178 of 1,823), respectively (P<0.001).

LBW has also been associatedwith the development of type 2 diabetes in Japanese subjects and that not only genetic influences but also higher BMI in adulthood seemed to be important in the development of type 2 diabetes in individuals with LBW (Sonoko *et al.*, 2003). The significantly higher incidence of hypertension in diabetic subjects with low birth weight compared with diabetic subjects with normal birth-weight suggests that insulin resistance might be stronger in the former (Sonoko *et al*., 2003).

2.3.4 Placental Weight and Birth Weight

The ability of the fetus to grow and thrive *in utero* depends on the placental function and the average weight of the placenta at term is 0.508 kg (Cunningham *et al.*, 2005). The ratio between PW and BW of the newborn is 1:6 (Cunningham *et al.*, 2005).However, methods of measurement vary widely particularly due to differences in placental preparations (Thomson *et al.,* 1969). PW and its relationship to infant size at birth have been studied for more than a century (Adair and Thelander, 1925).Past studies indicated that PW was associated with pregnancy outcome (Eriksson *et al.*, 2000). High PW was associated with a poor perinatal outcome, a low AS, respiratory distress syndrome and perinatal death; whereas a low PW was associated with medical complications in the mother (Naeye, 1987).Barker*et al.* (1990) reported that altered growth of the placenta was a predictor of maternal medical diseases including cardiovascular disease, hypertension and diabetes mellitus (Barker *et al.,* 1990).Other factors such as race and socioeconomic status also affect the PW (Perry *et al.,* 1995).

Careful examination of the placenta can provide insight regarding the *in utero* environment of the fetus before delivery (Perry *et al.,* 1995). Two standard references are endorsed by the College of American Pathologists: absolute PW and fetal/placental weight (F/P) ratio (Molteni *et al.,* 1978; Naeye, 1987; Langston *et al.,* 1997).Clinical associations with PWs and F/P ratio have been documented. For example, small placentas may be associated with trisomies, whereas large placentas may be associated with maternal diabetes (Langston *et al.,* 1997). Disproportionately large placentas (low F/P ratio) could reflect acute placental injury resulting in villous edema or a chronic process requiring placental overgrowth, such as maternal anemia or malnutrition (Naeye, 1987; Fox *et al.,* 2002). Disproportionately small placentas (high F/P ratio) may be seen in maternal hypertension, and may result in fetal distress or low AS (Molteni *et al.,* 1978; Naeye, 1987; Fox *et al.,* 1991; Eriksson *et al.,* 2000; Robertson *et al.,* 2002). Recent BW tables' show fetal BWs at term have increased over time (Robertson *et al.,* 2002).There is a positive correlation between fetal weight and placental weights (Naeye, 1987).The standard method of weighing the placenta, after trimming the placental disk of membranes and umbilical cord, may also merit simplification (Leary *et al.,* 2003). Leary *et al.* (2003) suggested that the fetal weight/placental weight correlation does not change when placentas are weighed trimmed compared to when they are weighed untrimmed.The placenta can be weighed with membranes and cord attached, but the standard approach since its proposal by Benirschke in the early 1960s is to weigh the placenta after the extra placental membranes and the umbilical cord are trimmed from the disk (Benirschke, 1961).This limits the measurement to the weight of the placental disk, the actual nutrient exchange part of the placenta (Benirschke, 1961). However, Leary *et al.*(2003) suggested that trimmed and untrimmed placental weights are exchangeable, based on their high correlation. PW and BW of the neonate are widely available measures (Cunningham *et al.,* 2005). The ratio of these two variables is a useful marker of foetal nutrition and utero-placental function (Cunningham *et al.,* 2005).The variations in the mean weight of the placenta may be due to variations in the methodology of preparing and weighing the placenta together with cord clamping time (Yao *et al.,* 1969).Kloosterman reported that the placentae and babies from multiparous women from 32 weeks onwards are heavier than those from primiparous women (Kloostermann, 1970).

A study by Abubakar *et al.* (2012) in Sokoto, Nigeria observed a positive correlation between PW and BW of neonates. However, the ratio of the placental and neonatal birth weights at term decreases with advancing gestational age. Thus, prolongation of pregnancy at term may adversely affect the fetus (Abubakar *et al.,* 2012). Madkar *et al.* reported that high placental weight/birth weight (PW/BW) ratio was associated with increased rates of admission to the neonate’s intensive care unit (NICU) and Apgar scores < 7 at 5 minutes as compared to normal PW/BW ratio, and the low PW/BW ratio group showed decreased rates of NICU admission. A high PW/BW ratio is significantly correlated with short-term adverse perinatal outcomes(Madkar *et al.,* 2015).There is Sex difference in placental weight of neonates (Barker *et al.*, 1990; Perry *et al.,* 1995; Abubakar *et al.,* 2012; Madkar *et al.,* 2015), with male neonates having higher mean placental weights than female neonates.

2.3.5Birth Weight and Immune System

Several studies have reported that infants with LBW or intra-uterine growth retardation or who were small for gestational age have a lower percentage of Tor B lymphocytes and lower vaccine-specific IgG responses (Chafrath *et al.*,1997; Das *et al.*, 1998; Golebiowska *et al.*,, 1999) than do newborn infants with normal birth weight (Moscatelli *et al.*,1976; Singh *et al.*,1978; Kiss *et al.*, 1984; Belloni *et al.*,1998; Khalak *et al.*,1998; Arinola *et al.*, 2003).

In one study, school children born preterm had a significantly lower percentage of CD4+T cells and lower CD4:CD8 ratios than did children born at term (Pelkonen *et al.*, 1999). Morbidity and mortality of LBW infants are known to be high due to infectious diseases (Valero *et al.*,2004) which have lead researchers to explore the effects of LBW on immune function.

2.3.6 Apgar Score and Cognitive Outcome

Odd *et al.*(2000) have shown an association between poor conditions at birth and long term cognitive functioning in term infants without apparent neonatal encephalopathy. Infants with prolonged or even brief low AS but without neurological signs seemed to have a higher risk of poor IQ (intelligent quotient) scores at age 18 years. The long term outcome of infants with low AS who do not develop encephalopathy is considered to be normal with respect to cerebral palsy as shown by Nelson and Ellenberg, (1981), although infants with moderate neonatal encephalopathy who do not develop encephalopathy may develop some degree of cognitive impairment as teenagers (Lindstrom *et al.*,2006; Gonzalez and Miller, 2006).

Lan *et al.* (1991) collected data on the BWs and 1 min and 5 min AS of new born infants between 1982 and 1987 at a teaching hospital in Central Taiwan. They reported that Compared to babies with normal AS, infants with low ASs were found to be born with low and very low BWs. They also reported that, in the 1 min of life test, the relative risks of LBWs among infants with AS of 0 to 3 and 4 to 6 were 115.0 and 5.9 times higher than those of normal infants, respectively. In the VLBW category, the relative risks of the above score were 252.5 and 51.1, in this order. In the 5 min of life test, the relative risks of the above scores were 16.2 and 12.1 in the LBW category, respectively. However, among babies of VLBW, the relative risks of the same scores were 121.2 and 84.9, in this order. They conclude that, the 5 min AS might be a useful prognostic index for the relationship between health and BW of new born infants.

2.4GESTATIONAL WEIGHT GAIN, BODY MASS INDEX AND BIRTH WEIGHT

Gestational weight gain (GWG) is a modifiable factor that can be controlled through nutritional counseling in order to gain adequate weight gain during pregnancy (Sajjad and Khan, 2010). GWG is one of the risk factors of health consequences and it is very important factor to be controlled (Cedergren, 2006; Tsukamoto *et al.*,2007; McDonald *et al.*, 2010).

Recommendations for weight gain during pregnancy differed from a single target for all women to different targets, depending on maternal Body Mass Index (BMI) (Islam and Ullah, 2005). During pregnancy, pregnant women have been advised to gain certain weights that have been recommended as to have a safe pregnancy and healthy infants without any complication (Islam and Ullah, 2005; Ochsenbein-Kolble *et al.*,2007; Borazjani *et al.,* 2011).

Women who gained less GWG were associated with small-for-gestational age (SGA) infants or LBW, while large for-gestational age (LGA) infants or macrosomia infant were associated with those having GWG outside the recommendation (Devader *et al.,* 2007). Excessive weight gain might contribute to an increased risk of preterm delivery, cesarean delivery and macrosomia, while inadequate weight gain during pregnancy will lead to preterm delivery and LBW (Kabali and Werler, 2007; Nohr *et al.*,2008; Briese *et al.,* 2009; Oken *et al.,* 2009; Rodrigues *et al.,* 2010). Gaining high and low weight have greater risk of LBW and high birth weight (macrosomia) and might related to obesity in later life (Johansson *et al.,* 2007). Supported by Kiel *et al.* (2007), women that gained less weight had significantly lower risk of large for gestational age (LGA) birth but high significant in small for gestational age(SGA) birth. Abrams *et al.* (2000) explained that, caesarean delivery and maternal weight gains was significant when weight gain exceed 16 kg. Underweight and average height women with low weight gain tend to be expose to preterm delivery, while overweight and obese women that gained low GWG were having moderate risk of preterm delivery (Schieve *et al.*, 2000; Mohsen and Wafay, 2007; Wang *et al.,* 2010).When obese women were compared to normal-weight women, the newborns of obese women had more than double the risk of macrosomia compared to those of women with normal weight (Yogev and Langer, 2008).

Pre-pregnancy BMI and GWG influence infant BW and play significant roles in adverse pregnancy outcomes including LBW (Ferland and O'Brien, 2003; Mattews *et al.,* 2004; Rogers *et al.,* 2004; Friis *et al.,* 2004) and macrosomia (Krammer *et al.,* 2002; Bergmann *et al.,* 2003;

Boulet *et al.,* 2003).In 1990, the Institute of Medicine (IOM) established guidelines for gestational weight gain based on pre-pregnancy BMI, aimed at achieving optimal pregnancy outcomes (Institute of Medicine, 1990). In a study of Icelandic women of normal pre-pregnancy BMI (BMI 19.5-25.5 kg/m^2) investigators noted that those who gained weight within the IOM guideline of 11.5-16.0 kg experienced less delivery complications than those who gained >20 kg (Thorsdottir *et al.,* 2002).Merchant *et al.* (1999)reported lower mean BW of newborns for women with pre-pregnancy BMI < 19 kg/m^2 who gained <12.5 kg compared to those who gained >12.5 kg among Pakistani women (Merchant *et al.,* 1999).Previous study results are inconsistent, partly due to differences among study populations (Merchant *et al.,* 1999; Thorsdottir *et al.,* 2002),in time and measurement of weight gain (Merchant *et al.,* 1999; Thorsdottir *et al.,* 2002; Neufeld *et al.,* 2004),in methods for controlling confounding, and relatively small sample sizes (Merchant *et al.,* 1999; Thorsdottir *et al.*, 2002; Brown *et al.*, 2002; Thame *et al.,* 2004).Literature is sparse on the independent effect of pre-pregnancy BMI on infant BW because many studies focused exclusively on GWG (Thorsdottir *et al.*, 2002; Brown *et al.,* 2002; Thame *et al.*, 2002; Neufeld *et al.,* 2004).In addition, there is need to validate IOM GWG guidelines in different populations (Merchant *et al.*, 1999).

Appropriate nutrient intake and weight gain during pregnancy are considered two of the most important modifiable behaviours for improved maternal and infant outcomes (Institute of Medicine, 1990). However, inappropriate dietary intake has been observed as a common practice among the adolescent population (Moran, 2007), and this could interact with their growth status and have an impact on the pregnancy outcome (Nielsen *et al.*, 2006). The burden of LBW has been reported to be higher in the developing countries, with 95.6% of all LBW births occurring in low and middle-income countries (Wardlaw, 2008).Maternal weight gain during pregnancy has been consistently associated with infant BW and pregnancy outcome (Caton and Hess, 2010) and low maternal weight gain is considered as a preventable risk factor for LBW (Abrams *et al.*, 1995). LBW and preterm deliveries in adolescent pregnancies are twice as common as in adult pregnancies and neonatal mortality in adolescent pregnancies are three times higher in frequency than for adult pregnancies (King, 2003). In Ilorin, Nigeria, Taiwo *et al.* (2014) reported that, there is a strong association between maternal weight gain and infant BW ($r= 0.816$; $p<0.01$).Adolescent mothers are at higher risk of delivering infants with LBW compared to the adult mothers, in spite of the higher gestational weight gain (GWG) (Taiwo *et al.*, 2014).

A study conducted by John *et al.*in Jos, Nigeria amongst 262 mother- child pairs reported thus: the Prevalence of maternal underweight, overweight and obesity was 4.2% (11/262),29.4% (77/262) and 25.9% (68/262), respectively. Child overweight/obesity was 5.4% (14/262), severe under-nutrition 5.7%(15/262).MeanmaternalBMIwashigherintheolder,moreeducatedandhighersocioeconomicstatus(SES).ChildmeanBW,weightforageZscoreandBMIforageZscore(BAZ)werehigheramongmotherswithBMI≥25kg/m.Alllargeforagebabieswereinmotherswithmaternal BMI≥25kg/m.Childhoodover-nutritionwasmorecommoninmaternalBMIof≥25kg/m.Overall,BMIforageZscore (BAZ)wasdirectlyrelatedwithmaternalBMI,maternalageandBW,althoughitwasinverselyrelatedwithmaternalBMI≥25kg/m (John *et al.*,2015). There were no differences in duration of any Breastfeeding (BF) or exclusive breastfeeding (EBF) according to pre-pregnancy BMI or GWGcategories (Castillo *et al.*, 2016). There was an increased predicted probability for weaning before the age of 3 months among infants from obesewomen, compared with those from mothers with normal pre-pregnancy BMI, with margins adjusted predictions of 0.36 (95%) confidence interval (CI) 0.31–0.41) and 0.23 (95% CI 0.21–0.25), respectively (Castillo *et al.*, 2016).

2.5MATERNAL DEMOGRAPHY AND BIRTH WEIGHT

2.5.1 Marital Status and Birth Weight

Marital status has been shown to correlate with intendedness of birth, economic status of mother, and social support of the mother and factors that may influence the health of the mother and infant (Chomitz *et al.*, 1995). The objective of this study was to determine if marital status is a predictor of LBW while adjusting for other associated risk factors for LBW (Wood, 1997). The risk factors were: minority race (Center for Disease Control, 1991,Ventura, 1995); maternal age under 18 (Ventura, 1995); maternal age greater than 34 (Ventura, 1995); maternal education less than high school (Parker *et al.*, 1994); late (after 1st trimester) or no prenatal care (Ventura, 1995); maternal smoking (Wilcox, 1993); and father's name not listed on the birth certificate (Shiono and Behrman, 1995).The absence of the father’s name on the birth certificate suggests a lack of paternal support (Guadino, 1993).Married mothers without the father’s name on the birth certificate have a higher rate of LBWs than unmarried mothers without the father’s name on the birth certificate. This appears to be a high risk group who may have special needs (Guadino, 1993; Ventura, 1995). The lower risk of LBW with the father’s name recorded on the birth certificate for both married and unmarried mothers could be attributed to the beneficial impact of the father’s involvement with the family (Guadino, 1993).Without the support of the father, the mothercould suffer emotionally as well as financially. Un-married women in the United States are typicallyof lower economic status, many living below thepoverty level (Ventura, 1995).

Being unmarried is also associatedwith other risks factors such as inadequate maternal weight gain and inadequate diet and nutritionintake (Chomitz *et al.*, 1995).

Births to unmarried women are sometimes considered illegitimate because the legal family structure of marriage in which to raise the child is not in place at the time of birth (Holt *et al.*,1997). LBW is a problem in all population groups. Reducing LBW will help to lower infant mortality, adverse health effects, and developmental problems (Shiono and Behrman, 1995). In North Carolina (among African Americans and other minorities), Over 12 percent of the births to unmarried women were LBW in 1994-95, compared to 7.0 percent for married women (Ventura, 1995). Unmarried women and their babies continue to be at higher risk.

Maternal age >18 is not a risk for unmarried mothers, yet it is formarried mothers (Parker *et al.*,, 1994; Ventura, 1995). Bennett *et al.* (1994) compared unmarried and married teenage mothers in a study on infant mortality. They reported thatunmarried teens are more likely not to live with theirfamilies, which can decrease social and economicsupport, are less likely to remain in school, and aremore likely to have another child before age 20 (Bennett *et al.*, 1994). Bennett states that marital status is often used as asubstitute for socioeconomic status, yet its association with poor birth outcomes has many other dimensions as well (Bennett, 1992). The effect of change of marital status on risk ofLBW was studied by Holt, *et al.* (1997).Thisstudy determined the relative risk of having a LBW infant for the 2nd birth by women whowere either married at the first birth and single atthe second, married at both births, or single at thefirst birth and married at the second: A change frommarried to single status was associated with a 30to 40 percent increase in risk of LBWrelative to remaining married. A change from singleto married status was associated with a 20 to 30percent reduction in risk of LBW relative to remaining single.

An infant born of LBW is at higher risk for health complications later in life (Paneth, 1995). Studies suggest that unmarried status is associated with LBW (Gaudino, 1993; Ventura, 1995; Holt *et al.,* 1997). Holt *et al.* (1997) advise that public health policy and programs which are directed at high risk mothers and infants should be aware of the increased risks and needs of the unmarried child-bearing women. Pregnancy provides an opportunity for increased contact with the health care system. The awareness and knowledge needed to improve health behavior may be increased by programs directed towards educating unmarried woman on smoking,nutrition and diet, elimination of drug use, stress control, early prenatal care, social support, and financial management (Holt *et al.,* 1997).

2.5.2 Birth Weight and Race

The black-white disparity in LBW in the United States is glaring and persistent (Case *et al.*, 2002; Cande and Annath, 2003). In 2000, 13 percent of babies born to black mothers were LBW, compared to 6.5 percent of those born to white mothers (Yollanda and Padilla, 2002).By contrast, rates of LBW for the other racial groups reported by the National Center for Health Statistics were close to that of whites: 6.8 percent among American Indians and 7.3 percent among Asians and Pacific Islanders (Wanda, 2004). The two-to-one disparity between blacks and whites has persisted for more than forty years, exists at most maternal age ranges, cannot be explained by differences in rates of multiple births, and cannot be explained by socioeconomic status alone (Howard and Cabral, 1996). Even infants born to college-educated black women are at much greater risk than infants born to college-educated white women of being low birth weight (Kenneth and Schoendorf, 1992).Black mothers were 63 percent more likely to have preterm deliveries than white mothers (17.3 percent as against 10.6 percent) in 2000 (Kenneth and Schoendorf, 1992; Case *et al.,* 2002; Yolande and Padilla, 2002).The rates of small-forgestational-age births among infants born at term in 1998 were 17.4 percent among blacks and 9.0 percent among whites (Cande and Ananth, 2003).

Michael *et al.*propose a 12-point plan to reduce Black-White disparities in birth outcomes using alife-course approach. The first four points (increase access to interconception care, preconception care, quality prenatal care, and healthcare throughout the life course) address the needs of African American women for quality healthcare across the lifespan. The next four points (strengthen father involvement, systems integration, reproductive social capital, and community building) go beyond individual-level interventions to address enhancing family and community systems that may influence the health of pregnant women, families, and communities.The last four points (close the education gap, reduce poverty, support working mothers, and undo racism) move beyond the biomedical model to address the social and economic inequities that underlie much of health disparities. Closing the Black-White gap in birth outcomes requires a life course approach which addresses both early life disadvantages and cumulative allostatic load over the life course (Michael *et al.,* 2010).

2.6 MATERNAL MEDICAL CONDITIONS AND BIRTH WEIGHT

2.6.1Birth Weight and Maternal Diabetes Mellitus

Pregnancy in women with pre-gestational diabetes is still associated with adverse perinatal outcomes, with nearly half of the infants born large for gestational age (Evers *et al.,* 2004; Jessen*et al.,* 2004; Persson *et al.,* 2009; Glinianaia *et al.,*2012; Klemetti *et al.,* 2012). This is mainly attributed to the placental transfer of maternal glucose, leading to fetal hyperinsulinemia and accelerated fetal growth (Pedersen, 1967), and strict maternal glycemic control is therefore a cornerstone in the clinical management of these women (Evers *et al.*, 2002; Ekbom *et al.*, 2012). Poor maternal glycemic control before and during pregnancy has been associated with both restrictive (Glinianaia *et al.*, 2012) as well as excessive fetal growth (Glinianaia *et al.*, 2012; Klemetti *et al.*, 2012), and elevated third-trimester HbA_{1c} predicts both increased offspring birth weight (Glinianaia *et al.*, 2012)and the prevalence of macrosomia (Klemetti *et al.*, 2012). The recommendation of frequently performed plasma glucose measurements and the use of insulin analogs, insulin pumps, and continuous glucose monitoring are recent attempts striving for maternal normoglycemia (Mathiesen *et al.*, 2012; Secher *et al.*, 2013; Mathiesen *et al.*, 2014). However, other nutritional substrates than glucose, such as lipids, probably also contribute to fetal overgrowth in pregnancies of women with diabetes (Schaeter *et al.*, 2008; Kulkari *et al.*, 2013).

Gestational diabetes mellitus (GDM) is defined as glucose intolerance of variable degrees with an onset, or first recognized, during pregnancy (Barker *et al.*, 2002; Buchanan and Xiang, 2005; Moore, 2010). About 15-45% of babies born to diabetic mothers can have macrosomia, which is a 3-fold higher rate when compared to normoglycemic controls (Barker *et al.*, 2002). Macrosomia is typically defined as a BW above the 90th percentile for gestational age or >4,000 g (Buchanan and Xiang, 2005; Chu *et al.*, 2007).

Unlike maternal hyperglycemia, maternal obesity has a strong and independent effect on fetal macrosomia (Ehrenberg *et al.*, 2004). Gestational age at delivery, maternal pre-pregnancy body mass index (BMI), pregnancy weight gain, maternal height, hypertension and cigarette smoking also have a significant impact (Hunter *et al.*, 1993; Athukorala *et al.*, 2007;Yogev and Langer, 2008).

Data from the Diabetes in Early Pregnancy Study indicate that fetal birth weight correlates best with second- and third-trimester postprandial blood sugar levels and not with fasting or mean glucose levels (Jovanovic-Peterson *et al.,* 1991).When postprandial glucose values average 120 mg/dl or less, approximately 20% of infants can be expected to be macrosomic, and if the glucose values are as high as 160 mg/dl, the rate of macrosomia can reach up to 35% (Gilman, 2005; Haji-Ebrahim *et al.,*2007; Crume *et al.,*2011). Macrosomic fetuses in diabetic pregnancies develop a unique pattern of overgrowth, involving the central deposition of subcutaneous fat in the abdominal and interscapular areas (Mcfarland *et al.,* 1998). They have larger shoulder and extremity circumferences, a decreased head-to-shoulder ratio, significantly higher body fat and thicker upper-extremity skinfolds (Hunter *et al.,* 1998; Buchanan and Xiang, 2005; Athukorala *et al.,* 2007). Because fetal head size is not increased, but shoulder and abdominal girth can be markedly augmented, the risk of Erb's palsy, shoulder dystocia and brachial plexus trauma is more common (Mcfarland *et al.,* 1986; Yogev and Langer, 2008). However, skeletal growth is largely unaffected. Macrosomia is associated with excessive rates of neonatal morbidity (Barker *et al.,* 2007). Macrosomic neonates have 5-fold higher rates of severe hypoglycemia and a doubled increase in neonatal jaundice in comparison with the infants of mothers without diabetes (Hunter *et al.,* 1993).In addition, there appears to be a role for excessive fetal insulin levels in causing accelerated fetal growth (Jovanovic-Ebrahim-Tehram *et al.,* 2007; Athukorala *et al.,* 2007). In a study which compared umbilical cord sera in infants of diabetic mothers and controls, the heavier, fatter babies from diabetic pregnancies were also hyperinsulinemic (Simmons, 1995).

2.6.2 Low Birth Weightand Maternal HIV Infection

Although new infections of human immunodeficiency virus (HIV) show a descending trend in recent years, the number of people living with HIV has been rising year by year. More than 16 million (95 % CI: 14.8 million, 17.4 million) female adults had been infected with HIV by the end of 2012 (UNAIDS, 2014). There is a risk for mother-to-child transmission of HIV. Among HIV infected women who took the highly active antiretroviral therapy (HAART), studies reported that the mother-to-child transmission (MTCT) rate ranged from 1 to 5 % (Townsend, 2008), and it was around 10 % among women who did not (Townsend *et al.,* 2007; Vander *et al.*, 2011). There is also a possibility that maternal HIV infection has severe impacts on pregnancy outcomes. It has been reported that HIV infected women are more likely to encounter adverse pregnancy outcomes, such as low birth weight (LBW) and preterm delivery (PTD) (Brocklehurst and Frend, 1998). And it is suggested that LBW and PTD are important risk factors for post-neonatal mortality and morbidity and other adverse events including neurodevelopmental problems (McCormick, 1985; Le Doare *et al.,* 2012).

Studies have provided inconsistent results for the association between maternal HIV infection and LBW/PTD. Some studies suggested that maternal HIV infection could increase the risk of low birth weight (LBW) and pre-term delivery (PTD) (Temmerman *et al.,* 1994; Ellis *et al.*, 2002; Schulte *et al.,* 2007), but others reported no significant association between them (Bucceri *et al.,* 1997; Patil *et al.,* 2011;Awoleke, 2012). Brocklehurst and Frend, summarized the study results published between 1983 and 1996. They found the association of maternal HIV infection with adverse pregnancy outcomes such as LBW and PTD, but failed to assess the effect of antiretroviral drugs on it.

It has been previously reported that compared with unexposed children, children who encountered intrauterine HIV exposure are more vulnerable to stunting, underweight and wasting, and their BW, HC and CC are generally lower than their unexposed counterparts (McGrath *et al.*, 2012). However, it was controversial for the effect of maternal HIV infection on both LBW and PTD. Brocklehurst and Frend reported that the increasing risks of LBW and PTD were associated with maternal HIV infection. And they found that women in developing countries had higher risks of both LBW and PTD than those in developed countries.

The HIV associated Low birth weight (LBW) and pre-term delivery(PTD) might be related to the damaged human immune system, especially the reduced CD4+ T cells and immunosuppression. Previous studies documented that women with CD4 cell counts <350 cells/mm^3 had an increased risk of having LBW infants (RR = 1.57; 95 % CI: 1.16, 2.12) compared to women with higher CD4 cell counts (Vander *et al.,* 2011; Kim *et al.,* 2012). There is a possibility that women are immunocompromised during pregnancy (Dong *et al.*, 2001), and if accompanied with HIV infection, disease progress might be accelerated. Simultaneously, reproductive tract infections, which are contributed to the incidence of adverse pregnancy outcomes, would occur more frequently due to the immunosuppression (Tanton *et al.*, 2001). Some studies have reported that HIV-1 can replicate in the placenta (Kumar *et al.,* 2011), and it has also been shown that HIV-1 infection may alter the cytokine profile in the placenta (Moussa *et al.,* 2001; Faye *et al.,* 2007). This may affect the function of placenta during pregnancy, and then restrict the development of fetal, which might be another incentive of LBW and PTD.

After stratified by antiretroviral drugs use before or during pregnancy, Xiao*et al.* (2015) found that HAART or other regimens of antiretroviral therapy (ART) had no obvious effect on the associations between maternal HIV infection and LBW/PTD. It is suggested that intrauterine ARVs exposure did not decrease or increase the risk of LBW or PTD in HIV infected women (Townsend *et al.,* 2007; Vander *et al.,* 2011).However, Papp *et al*. (2015) suggested that Protease

Inhibitor (PI)-based ART could increase the risk of adverse pregnancy outcomes mainly due to lower level of progesterone, which was significantly associated with fetal weight. Sibiude *et al.*(2012) also found that ARVs and, particularly, with the initiation of ritonavir-boosted PI therapy during pregnancy were correlated with PTD in HIV infected women.

2.6.3Low Birth Weight and Maternal Malaria Infection

Over 30 million women become pregnant yearly in malarious regions of Africa, and most of these women reside in areas of stable malaria transmission (WHO/UNICEF, 2003). Guyatt and Robert, in 2004, in Nairobi observed that vast majority of women with placental malaria during pregnancy areasymptomatic. Probable reason for this could be thefact that most of these women now register for antenatal care (ANC), and are placed on sulfadoxine and pyrimethamine as routine intermittent preventive therapy for malaria(IPT) (Steketee *et al.*, 2001; Scott *et al.,* 2005). Placental malaria increases the risk of women delivering LBW neonates and this is an important risk factor for infant morbidity and mortality (Guyatt and Robert, 2004; Scott *et al.*, 2005). Placentasthat are infected with malaria parasites tend to recruit antibodies, cytokines and macrophages indicating immune response to these parasites (Ismail *et al.,* 2000). The LBW related to placental malaria may be related to impaired nutrient transport to the foetus. High densities of malaria parasites in the placenta coupled with immune response may result in consumption of nutrient that would have been delivered to the foetus, leading to LBW (Guyatt and Robert, 2004).

Ismail *et al*.in 2000, found that malaria infected placentas have thickening of placental membranes. This may again interfere with nutrient and oxygen transport across the placenta to the foetus. Multiparous mothers were initially thought to have low risk of placental malaria relative to primigravidae and secundigravidae (Brabin, 1991). Multiparous mothers now are known to beat risk of placental malaria like the primigravidaeand secundigravidae (Brabin, 1991; Ter Kuile *et al.,* 2003). This could bedue to loss of previously acquired immunity in these mothers making them particularly susceptible to placental malaria. In Kenya, Ter Kuile *et al.,* in 2003, had further argued that multiparous mothers could have high risk of placental malaria, particularly if they are living in stable malaria areas. Many authors in Nigeria and other sub-saharan countries of the world on placental malaria looked at its prevalence (Steketee *et al.*, 2001; Scott *et al*., 2005; Erhabor *et al.*, 2012; Rachas *et al.*, 2012). Few of them examined the relationship between placental malaria and other febrile illnesses (Rachas *et al.*, 2012), another few of them described the prevalence and impact of placental malaria during pregnancy with an attempt to quantify this burden on neonatal BW (Steketee *et al.*, 2001; Scott *et al*., 2005; Ezebialu *et al.,* 2012; Amal *et al.,* 2013; Okolie *et al.,* 2014). Ahmadu *et al.* (2014) in Maiduguri, northeast Nigeria reported that although the relationship between placental malaria and neonatal LBW was not significant, most neonates with low birth weight LBWhad their mothers diagnosed with placental malaria.

2.6.4 Blood Pressure and Birth Weight

Hypertension causes blood vessel stenosis in pregnant women, reduces plasma volume, reduces the supply of nutrients to the foetus thus affecting foetal growth and results in LBW (Nahum and Stanislaw, 2004; Stoll and Chapman, 2008; Takai *et al.,* 2014).

During the past decade, epidemiologists have focused on the influence of events that occurred during fetal life on the cardiovascular risk in later life (Barker *et al.*, 1989). Most of the studies have reported a negative association between BW and systolic blood pressure (BP), (Law and Shiel, 1996; Huxley *et al.*, 2000; Lurbe *et al.*, 1996) as well as an increase in the risk of developing hypertension among subjects with the lowest BWs. Studies that have reported results for both systolic and diastolic BP found either an inverse relation with BW for systolic but not diastolic BP, or an inverse relation with BW that was greater for systolic than for diastolic BP (Law and Shiel, 1996;Lurbe*et al.*, 1996; Huxley *et al.*, 2000). The results of a recent study found a relationship between BW and pulse pressure (PP) measured in the arm over 24 hours, a surrogate measure of arterialstiffness (Lurbe *et al.*,2001).In numerous global studies, adult risk of hypertension and other chronic disease is associated with LBW (Tian *et al.*, 2006; Barker *et al.*, 2006). In an emerging economy, LBW babies who show catch-up growth may be at particular risk of developing such disease in midlife (Bhargava *et al.*, 2004). These observations subsequently led to the 'developmental origins' hypothesis.In newborns, their blood pressure (BP) correlates with BW (Hulman *et al.*, 1991; Zubrow *et al.*, 1995; Sadoh and Ibhanesebhor, 2009). There may be differences in the relationship between BW and BP in preterm babies small for gestational age (SGA) and appropriate for gestational age (AGA) in the first week of life (Smal *et al.*, 2009). AGA babies showed the expected positive correlation between BW and BP while SGA babies did not(Smal *et al.*, 2009).

Size at birth was not related to systolic pressure in Sagamu (Law *et al.*, 2000). Whereas studies of children have generally shown an inverse relation between birthweight and blood pressure after adjustment for current weight (Murray and Lopez, 1997), this inverse relation is less consistent in studies of black populations. A significant inverse relation between BW and systolic pressure, after adjustment for current weight, has been found in Jamaican children aged

10–16 years, (Forrester *et al.,* 1999)in 5- and 6-year-old children in southern Africa, (Woelk *et al.,* 1998 and Levitt *et al.,* 1999) and in 7–11-year-old African American boys but not girls (Donker *et al.,* 1997). However, a study of 10–12-year-olds in Jamaica showed a weak positive relation, (Godfrey *et al.,* 1994)and there was no relationship in 1–9-year-old Gambian children (Margetts *et al.,* 1991).In a small study of young African American adults, BW was weakly positively related to blood pressure (Falkner *et al.,* 1997). An inverse relation between ponderal index at birth and systolic pressure has been found in other studies of children and adults in Europe (Law *et al.,* 1991 and Barker *et al.*, 1992).It has not, however, been found consistently. Published findings show that in developed countries raised blood pressure may be related to either LBW, or thinness, or short length at birth (Murray and Lopez, 1997).

Little is known about the association between birthweight and blood pressure in developing countries, where intra-uterine growth retardation is common (United Nation Children's Fund, 1997).An inverse relation between birthweight and blood pressure, after adjustment for current weight, has been shown in studies of children in Zimbabwe (Woelk *et al.,* 1998),South Africaand Jamaica (Forrester *et al.,* 1999).No relation was found in Gambian children (Margetts *et al.*, 1991)or in a separate study of Jamaican children (Godfrey *et al.*, 1994).

A report by Nwokoye *et al.* (2015) showed a weak correlation between BW and SBP at birth. Mode of delivery was not significantly associated with BP (Earley *et al.*, 1980; Nascimento *et al.,* 2002; Nwokoye *et al.,* 2015). However, Holland and Young (1956) found that SBP was lower among CS delivered infants compared with those by SVD. It is also earlier reported that the more trauma the head of a newborn passes through (e.g. during normal delivery and also during forceps delivery) the higher the BP (Holland and Young, 1956).

CHAPTER THREE

3.0 MATERIAL AND METHODS

3.1STUDY LOCATION

The study was conducted in Federal Teaching Hospital Gombe, Gombe State, Nigeria (fig 3.1).The hospital is a tertiary healthcare institution with a 300 beds capacity establish in the year 2000.

Gombe is a city in Northeastern Nigeria (and a Local Government Area). It is the capital city of Gombe State and has an estimated population of 261,536 (2006 census). The city is the headquarters of the Gombe Emirate, a traditional State that covers most of Gombe State. It is located on latitude 10”17’N 11”10’E and longitude 10.283”N 11.16”E.

Gombe State is one of the 36 state of the Federal Republic of Nigeria, located in the Northeast region of the country on latitude 9”30’ and 12”30’N, longitude 8”5’ and 11”45’E. It is bordering Bauchi, Adamawa, Yobe, Taraba and Borno States, with a land area of 20,265 square kilometers. The state climate is generally warm,with temperatures not exceeding 40°C during the months of March-May considered to be the hottest months. Average annual rainfall of 850mm. GombeState is generally a guinea savannah grassland with concentration of woodlands in the southeast and south west.

3.2 DEMOGRAPHICS

Gombe State is mainly populated by Fulani, constituting more than half of the state population and other ethnic groups like Hausa, Bolewa, Kanuri, Tera, Tangale, Waja, Tula, Cham, Lunguda, Dadiya, Kamo/Awak, Pero/Shonge, Kumo, Banbuka, Jukun and others. Hausa is the commercial language amidst the people.

The people of Gombe State are primarily farmers producing food and cash crops, which include maize, sorghum, rice, wheat, groundnuts, soya beans, tomatoes, onions, sugarcane, gum Arabic to mention a few.

3.3 INCLUSION AND EXCLUSION CRITERIA

3.3.1Inclusive Criteria

The following were included for the study:

i. Live singleton births
ii. Maternity or birth records from January 1, 2001 to December 31, 2014
iii. Maternity or birth records within this hospital
iv. Mothers who attended antenatal (prenatal) care in this hospital

3.3.2 Exclusion Criteria

The following were excluded from the study:

i. Birth records outside of this hospital
ii. Still births
iii. Infants with visible congenital anomalies at birth
iv. Multiple births

3.4CHARACTERISTICS OF THESAMPLE POPULATION

A data set of 10,586 singleton live births from 2001-2014 was retrieved from maternity registers at the labour ward, Department of Obstetrics and Gynecology, Federal Teaching Hospital Gombe, Gombe State, Nigeria. Male infants (n = 5660; 53.5 %) while female infants (n = 4926; 46.5 %). For mother and infant pair, only two thousand, two hundred and seventy six (2276) infants had information on their mother's health conditions during pregnancy, Socio-demography and anthropometric measurements retrieved from hospital records at the department of Biostatistics and medical records due to lack of accessibility of the other records.

3.5MINIMUM SAMPLE SIZE

The sample size for this study was obtained using the formula:

[n= z2pq/d2] (Naing *et al.*,2006)

Where:

n= the desired sample size

z= the standard normal deviation, usually set at 1.96 (approx. 2.0)

p= the proportion in the target population having the particular trait (when no estimate 50% is used; i.e. 0.5)

q= 1.0-p

d= degree of accuracy desired, usually set at 0.05

Therefore, n= (1.96)2(0.5) (0.50)/(0.05)2=384

For the sake of accuracy and reliability of the study a total of 10,586 data was obtained from Federal Teaching Hospital Gombe.

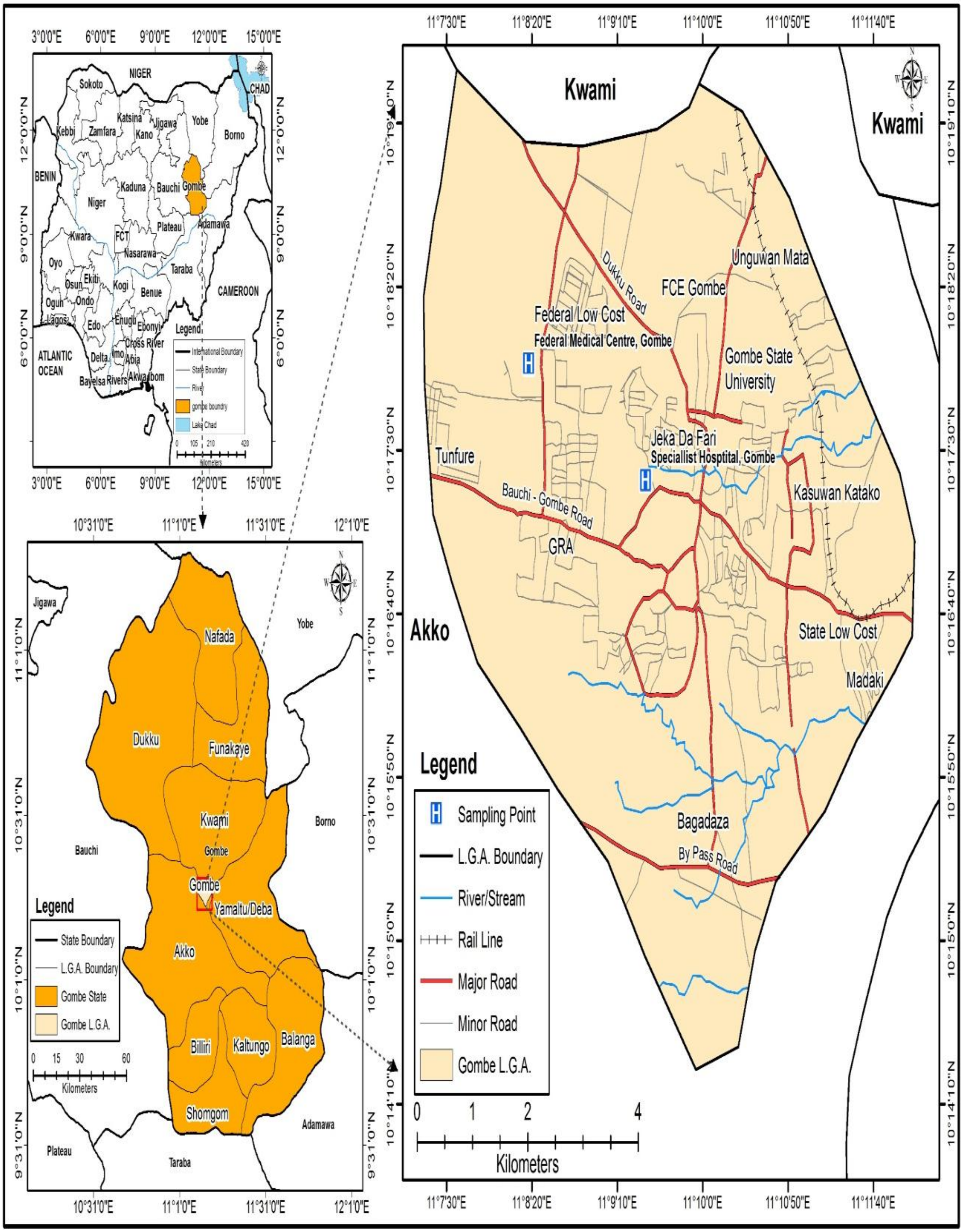

Figure 3.1: Map of Gombe L.G.A. Showing Sampling Points

Source: Adapted from the Administrative Map Gombe State

3.6 METHODOLOGY

A data set of 10,586 singleton live births from 2001-2014 was retrieved from maternity registers at the labour ward, Department of Obstetrics and Gynecology, Federal Teaching Hospital Gombe, Gombe State, Nigeria. Male infants (n = 5660; 53.5 %) while female infants (n = 4926; 46.5 %). The information extracted were: maternal age at the time of delivery, parity, mode of delivery, infant birth weight (BW), placental weight (PW), head circumference (HC), chest circumference (CC), birth length (BL), sex of the infant, Apgar score (AS) of the infant at 1ST and 5TH of life. For mother and infant pair, only two thousand, two hundred and seventy six (2276) infants had information on their mother's health conditions during pregnancy, Socio-demography and anthropometric measurements retrieved from hospital records at the department of Biostatistics and medical records due to lack of accessibility of the other records. Maternal health conditions extracted included: diabetes mellitus, HIV infection, malaria infection, blood pressure; maternal anthropometric measurements retrieved included: height, weight, gestational weight gain and body mass index (BMI) and maternal and paternal socio-demographic information retrieved were: place of residence, ethnicity, marital status, occupation, and level of education.

i. **Gestational Weight Gain** was obtained from subtracting weight of mother at her first visit at the antenatal (prenatal) clinic from weight of mother at her last visit (appointment) before delivery. Mothers started attending antennal clinic during first trimester and first month of second trimester of pregnancy.

ii. Body Mass Index (BMI): The BMI of mothers was calculated as follows: weight (kg)/ [height x height (m^2)].

iii. **Maternal blood pressure for each mother**was obtained by taking the average of systolic blood pressure (SBP) and diastolic blood pressure (DBP) of all maternal antenatal visits.

- Normotensive = SBP (90 – 140 mmHg) and DBP (60 – 90 mmHg).
- Hypertensive = SBP (> 140 mmHg) and DBP (> 90 mmHg)
- Hypotensive = SBP (< 90 mmHg) and DBP (< 60 mmHg)

iv. Diabetes mellitus diagnosis: Maternal fasting blood sugarof greaterthan 7.0 mmol/L (>7.0mmol/L)andrandom blood sugarof greater than 11.1mmol/L (>11.1mmol/L). These includes mothers with pre-pregnancy diabetes and gestational diabetes. Efforts were made to control blood sugar level in all the diabetic mothers during the course of the gestation.

v. Malaria infection diagnosis: Woman who had an episode of malarial attacked at any point during the course of gestation and was diagnosed using microscopic blood film or antigen-based rapid diagnostic test (RDTs).

3.7ETHICAL APPROVAL

Ethical approval was obtained from the Health Research Ethics Committee, Faculty of Medicine, Ahmadu Bello University Zaria. Ethical approval was also obtained from the Research Ethics Committee of Federal Teaching Hospital, Gombe, Gombe state, Nigeria.

3.8 STATISTICAL ANALYSES

Data wereexpressed as mean ± standard deviation (SD). Student's t-test was used to test for significant difference in the means of two groups of variables. Chi- square was used to test for significant association between birth weight and maternal characteristics (anthropometry, health conditions and demographic factors).One way analysis of variance (ANOVA) was used to test for significant difference in the means of three or more groups of variables. Pearson's correlation coefficient was used to test for significant association between newborn and maternal anthropometric and demographic variables.Linear regression was used to generate predictive equations of birth weight.

$P<0.05$ was deemed statistically significant. SigmaStat version 3.5 (Systat Software Inc., San Jose, California)and SPSS version 20 (SPSS IBM Inc., Chicago, IL) were used for the statistical analyses.

CHAPTER FOUR

4.0 RESULT

4.1ANALYSES OF STUDY POPULATION

Maternities from 2001 - 2014 (n = 10586) were collected and analyze, five thousand six hundred and sixty were male (n = 5660;53.5 %) while four thousand nine hundred and twenty six (n = 4926;46.5 %) were female. However, for mother and infant pair, only two thousand, two hundred and seventy six (2276) infants had information on their mother's health conditions during pregnancy, Socio-demography and anthropometric measurements retrieved from hospital records at the department of Biostatistics and medical records due to lack of accessibility of the other records. The study was conducted in Federal Teaching Hospital Gombe. The prevalence of low birth (< 2.5 kg), normal birth weight (2.5 kg – 3.99 kg) and high birth weight or macrosomia (≥ 4) were 10.2%, 86.0% and 3.8% respectively (Table 4.1). The age range of mothers in the study sample was 15-49 years, while the mean birth weight of newborns was3.09 ± 0.53 kg and ranged from 1.00 – 6.50 kg (Table 4.3).

4.2 DESCRIPTIVE STATISTICS

Table 4.1 shows the frequency and percentage of birth weight (BW) and mode of delivery (MOD) categories. Out of the entire sample population of neonates from 2001-2014 (n = 10586), 1078 (10.2%) had low birth weight (LBW), 9103 (86.0%) had normal birth weight (NBW) and 402 (3.8%) had high birth weight (HBW) or macrosomia. Also, from table 4.1, 8082 (76.3%) were delivered via spontaneous vaginal delivery (SVD), 2138 (20.2%) were delivered via caesarean section (C/S), 319 (3.0%) were delivered via vacuum assisted delivery and 47 (0.4%) using forceps assisted delivery.

Table 4.2 shows the relationship between maternal anthropometry, demography, health conditions and birth weight (BW) of neonates. From the table the prevalence of low birth weight (LBW) (< 2.5 kg) amongst male and female babies were 8% and 12.7% respectively. This shows that there was high incidence of LBW among female neonatescompare to male neonates which was statistically significant, (χ^2 = 65.25, p = 0.001). Also, the incidence of LBW deliveries among maternal age categories, less than 20 years of age, 20 – 30 years, 31 – 39 years and 40 – 49 years of age were 17.6%, 9.5%, 9.1% and 12.6% respectively. This indicate that LBW delivery was more common among teenage mothers and mothers age forty and above which was statistically significant, (χ^2 = 64.04, p = 0.001). The table also showed the incidence of LBW among neonates born preterm (< 37 weeks) and at term (37 completed weeks) to be respectively 25.4% and 4.04%which indicatesthat LBW was more common among neonates born preterm compared to those born at term, which also showed to be statistically significant, (χ^2 = 1081.52, p = 0.001). The table showed that mothers with no formal education have the highest prevalence of LBW deliveries (49.3%) compared to mothers with primary level of education (26.6%), mothers with secondary education (11.0%) and the least LBW prevalence was observed among mothers with tertiary education (8.9%), which showedto be statistically significant, (χ^2 = 148.98, p = 0.001). Prevalence of LBW among neonates born to fathers with no formal education, primary level of education, secondary level of education and tertiary level of education were respectively 25.8%, 17.5%, 15.8% and 7.9%. This showed that birth weight (BW) of neonates increases as paternal education advances, which also proved to be statistically significant, (χ^2 = 67.32, p = 0.001). The prevalence of LBW delivery among mothers giving birth for the first time (primipara) was (11.8%), while among mothers delivering for the second time (Parity 1), third time (Parity 2), fourth time (Parity 3) and fifth and above times (Parity ≥ 4) were 9.3%, 9.9%, 8.3% and 10.7% respectively. This indicates there was higher prevalence of LBWamong primiparous mothers compared to mothers who had delivered once or more times, witha statistical significant difference, (χ^2 = 14.89, p = 0.005). From the table, the prevalence of LBW deliveries among mothers who gained less than 5.0 kg of weight was 28.6%, among those who gained 5.1 – 11.9 kg of weight, the incidence of LBW was 3.4% and LBW delivery did not occur among mothers who had gained 12 kg and above. This indicates that LBW delivery was more common in mothers who gained less than 5 kg relative to mothers who gained greater than 5kg during the gestational period with a statistical significant difference, (χ^2 = 328.08, p = 0.001). The prevalence of LBW delivery among married mothers was 11.8% while among single or unmarried mothers was 37.5%. This showed that LBW was more associatedwith single mothers compared to married mothers which showed statistical significant difference, (χ^2 = 34.817, p = 0.001). From the table, the prevalence of LBW delivery among mothers who had at least an episode of malarial attacked was 23.6% while the prevalence of LBW delivery among mothers who never had an episode of malarial attacked was 10.7%. This showed that high prevalence of LBW delivery was associated with at least an episode of malarial attached, which showed statistical significant difference, (χ^2 = 52.39, p = 0.001). The prevalence of LBW delivery among HIV negative and HIV positive mothers were 11.7% and 22.9% respectively. This showed that LBW delivery are more associated with HIV infection which showed statistical significant difference, (χ^2 = 24.73, p = 0.001). Also from the table, the prevalence of LBW delivery among diabetic and non- diabetic mothers were 14.1% and 12.4%. This also showed that LBW wasassociated more with diabetes mellitus which proved to be statistically significant, (χ^2 = 18.03, p = 0.001). Lastly, the incidence of LBW delivery among normotensive, hypertensive and hypotensive mothers are 10.3%, 13.9% and 23.7% respectively. This indicates that LBWdelivery was higher among hypotensive mothers compared to normotensive and hypertensive mothers, which was statistically significant, (χ^2 = 76.56, p = 0.001).

Table 4.3 shows the descriptive statistics of the entire sample population of newborns or neonates from 2001- 2014 (n =10586, males n = 5660 and females n =4926). From Table 4.3,the mean birth weight (BW) of the entire sample population was (3.09 ± 0.53) kg. Also from the Table, the minimum BW of newborns in the study was1.00 kgwhile the maximumBW of newborns in the study was6.50 kg. The mean BW of male and female newborns were respectively 3.15 ± 0.53 and 3.01 ± 0.52 kg indicating that male newborns weigh significantly higher compared to female newborns, (t = -14.13, p <0.001). Also, BWs of male newborns ranged from (1.00 -6.50) kg while that of their female counterparts ranged from (1.01-5.40) kg.The results also revealed that there were significant difference in mean gestational age (GA), birth length (BL), head circumference (HC), chest circumference (CC), placental weight (PW) between the sexes. The male newborns had higher values compared to their female counterparts with a statistical significant difference at (p < 0.001) except for Apgar score (AS). The result showed that there were significant difference in mean maternal height, maternal weight and maternal gestational weight gainbetween the mothers of the two sexes. The mothers of male newborns had higher values compared to the mothers of female newborns with a statistical significant difference at (p < 0.05) except for maternal age, parity, Body mass index (BMI) and packed cell volume (PCV).

Table 4.1: Frequency and percentage of birth weight and modes of delivery of newborns

	Frequency	**Percent**
Birth weight (kg)	(n = 10586)	100
LBW (< 2.5 Kg)	1078	10.2
NBW (2.5 – 3.99 Kg)	9103	86.0
HBW (≥ 4 Kg)	402	3.8
Mode of delivery	(n = 10586)	100
C/S	2138	20.2
Forceps	47	0.4
SVD	8082	76.3
Vacuum	319	3.0

LBW = Low birth weight, NBW = Normal birth weight, HBW = High birth weight
C/S = Caesarean section, SVD = Spontaneous vaginal delivery.

Table 4.2: Relationship between maternal anthropometry, demography, health conditions and birth weight of neonates

	N	LBW	NLBW	df	χ^2	*p- value*
Sex of newborns	10586					
M	5660	451 (8%)	5209 (92%)	1	65.250	0.001
F	4926	627 (12.7%)	4299 (87.3%)			
Maternal age (years)	10586					
<20	933	164 (17.6%)	769 (82.4%)	3	64.040	0.001
20 – 30	7131	677 (9.5%)	6454 (90.5%)			
31 – 39	2292	208 (9.1%)	2084 (90.8%)			
40 – 49	230	29 (12.6%)	201 (87.4%)			
Gestational age (weeks)	10586					
< 37 (Preterm)	3043	773 (25.4%)	2270 (74.6%)	1	1081.521	0.001
≥ 37 (term)	7543	305 (4.04%)	7238 (95.95%)			
Maternal level of education	2276					
Nil	67	33 (49.3%)	34 (50.7%)	4	148.976	0.001
Primary	109	29 (26.6%)	80 (73.4%)			
Secondary	1280	141 (11.0%)	1139 (88.9)			
Tertiary	820	73 (8.9%)	747 (91.1%)			
Paternal level of education	2244					
Nil	89	23 (25.8%)	66 (74.2%)	4	67.317	0.001
Primary	171	30 (17.5%)	141 (82.5%)			
Secondary	688	109 (15.8%)	579 (84.2%)			
Tertiary	1296	102 (7.9%)	1194 (92.1%)			
Parity	10586					
0	2686	316 (11.8%)	2370 (88.2%)	4	14.899	0.005
1	2666	247 (9.3%)	2419 (90.7%)			
2	2162	213 (9.9%)	1949 (90.1%)			
3	1150	96 (8.3%)	1054 (91.7%)			
≥ 4	1922	206 (10.7%)	1716 (89.3%)			

LBW = low birth weight   NLBW = Not-low birth weight

Table 4.2: Relationship between maternal anthropometry, demography, health conditions and birth weight of neonates

	N	LBW	NLBW	df	χ^2	*p- value*
Gestational weight gain (kg)	2276					
< 5 kg	817	234 (28.6%)	583 (71.4%)	2	328.083	0.001
5.1 – 11.9 kg	1252	42 (3.4%)	1210 (96.6%)			
≥ 12 kg	207	0 (0%)	207 (100%)			
Marital status	2276					
Married	2244	264 (11.8%)	1980 (88.2%)	2	34.817	0.001
Single	32	12 (37.5%)	20 (62.5%)			
Malaria infection	2276					
Uninfected	2026	217 (10.7%)	1809 (89.3%)	2	52.393	0.001
Infected	250	59 (23.6%)	191 (76.4%)			
HIV infection	2276					
Negative	2180	254 (11.7%)	1926 (88.3%)	2	24.729	0.001
Positive	96	22 (22.9%)	74 (77.1%)			
Diabetes mellitus	2276					
Normal	2198	273 (12.4%)	1925 (87.6%)	2	18.026	0.001
Diabetic	78	11 (14.1%)	67 (85.9%)			
Maternal blood pressure (mmHg)	2276					
Normotensive	1791	184 (10.3%)	1607 (89.7%)	3	76.560	0.001
Hypertensive	244	34 (13.9%)	210 (86.1%)			
Hypotensive	241	57 (23.7%)	184 (76.3%)			

LBW = low birth weight NLBW = Not-low birth weight

Table 4.3: Descriptive statistics of neonatal and maternal characteristics for the entire sample population.

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 10586)		(n = 5660)		(n = 4926)			
Maternal age (years)	26.79 ± 5.69	15.00 -49.00	26.79 ± 5.76	15.00 – 49.00	26.78 ± 5.62	15.00 -46.00	-0.0591	0.953
Parity	1.97 ± 2.01	0.00 – 13.00	1.99 ± 2.04	0.00 – 13.00	1.94 ± 1.97	0.00 – 13.00	-1.320	0.187
Gestational age (weeks)	37.47 ± 2.55	28.00 – 58.00	37.65 ± 2.47	28.00 – 58.00	37.27 ± 2.63	28.00 – 53.00	-7.560	<0.001
Apgar Score (1minute)	7.62 ± 1.37	1.00 – 10.00	7.62 ± 1.37	1.00 – 10.00	7.62 ± 1.36	1.00 – 10.00	0.0407	0.968
Apgar Score (5minutes)	9.18 ± 1.02	2.00 – 10.00	9.19 ± 1.01	2.00 – 10.00	9.16 ± 1.04	3.00 – 10.00	-1.131	0.258
Birth weight (kg)	3.09 ± 0.53	1.00 -6.50	3.15 ± 0.53	1.00 -6.50	3.01 ± 0.52	1.01-5.40	-14.128	<0.001
Birth length (cm)	50.12 ± 3.68	27.00 -64.00	50.54 ± 3.52	28.00 – 64.00	49.65 ± 3.79	27.00 – 61.00	-11.397	<0.001
Head circumference (cm)	34.27 ± 2.22	15.00 – 43.00	34.46 ± 2.18	15.00 – 43.00	34.05 ± 2.24	15.00 – 40.00	-8.847	<0.001
Chest circumference (cm)	33.09 ± 2.30	15.00 – 43.00	33.32 ± 2.23	18.00 – 43.00	32.85 ± 2.36	15.00 – 42.00	-9.659	<0.001
Placental weight (cm)	0.62 ± 0.15	0.05-1.90	0.63 ± 0.16	0.05-1.90	0.61 ± 0.14	0.18 - 1.40	-6.873	<0.001
N	(n = 2276)		(n = 1169)		(n = 1107)			
Maternal height (cm)	159.38 ± 6.04	142.00 – 182.00	159.85 ± 6.14	145.00 – 182.00	158.88 ± 5.89	142.00 – 181.00	-3.876	<0.001
Maternal weight (kg)	67.58 ± 11.79	36.00 – 129.00	68.18 ± 11.97	40.00 – 129.00	66.94 ± 11.58	36.00-110.00	-2.518	0.012
Gestational weight gain (kg)	6.92 ± 3.06	1.00 – 19.00	7.28 ± 3.15	1.00 -19.00	6.55 ± 2.91	2.00 -18.50	-5.693	<0.001
BMI	26.51 ± 3.79	15.38-47.96	26.59 ± 3.88	17.19-47.96	26.42 ± 3.69	15.38-43.30	-1.092	0.275
PCV	0.33 ± 0.05	0.18-0.62	0.33 ± 0.05	0.18-0.62	0.32±0.05	0.18-0.54	-1.881	0.060

Table 4.4 shows descriptive statistics of the sample population of newborns delivered in 2001 (n = 309, males n = 181 and females n = 128). The mean birth weight (BW) of the entire sample population was (3.12 ± 0.5) kg. Also from the Table, the minimum BW of newborns was1.50 kgwhile the maximum BW of newborns was5.20 kg. The mean BW of male and female newborns were respectively 3.14 ± 0.56 and 3.07 ± 0.49 kg showing no significant statistical difference between the sexes, ($t = 1.101$, $p = 0.272$). Birth weight of male newborns ranged from (1.50 -5.20) kg while that of their female counterparts ranged from (1.75-4.30) kg .The result also shows no statistical significant difference between the sexes with all the variables compared (maternal age, parity, gestational age and Apgar score) with $p > 0.05$.

Table 4.5shows descriptive statistics of the sample population of newborns delivered in 2002 (n = 319, males n = 159 and females n = 160). The mean birth weight (BW) of the entire sample population was (3.08 ± 0.47) kg. Also from the Table, the minimum BW of newborns was1.50 kgwhile the maximum BW of newborns was4.40 kg. The mean BW of male and female newborns were respectively 3.17 ± 0.46 and 2.99 ± 0.48 kg showing that male newborns weigh significantly higher compared to female newborns, ($t = -3.252$, $p = 0.001$). Also, birth weight of male newborns ranged from (1.60 -4.40) kg while that of their female counterparts ranges from (1.50 -3.80) kg. There was no statistical significant difference between the sexes with respect to other variables compared (maternal age, parity, gestational age and Apgar score) with $p > 0.05$.

Table 4.4: Descriptive statistics of neonates delivered in 2001

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 309)		(n = 181)		(n = 128)			
Maternal age (years.)	25.9 ± 6.1	15.00 -45.00	25.6 ± 5.8	16.00 – 45.00	26.5 ± 6.5	15.00 – 42.00	1.38	0.17
Parity	2.2 ± 2.5	0.00 – 13.00	2.1 ± 2.23	0.00 – 13.00	2.4 ± 2.74	0.00 – 13.00	1.075	0.283
Gestational age (weeks)	37.2 ± 2.9	28.00 – 57.00	37.2 ± 2.99	28.00 – 57.00	37.3 ± 2.69	28.00 – 52.00	0.12	0.906
Birth weight (kg)	3.12 ± 0.5	1.50 -5.20	3.14 ± 0.56	1.50 -5.20	3.07 ± 0.49	1.75-4.30	1.101	0.272
Apgar Score (1minute)	6.72 ± 1.53	2.00 -9.00	6.77 ± 1.5	2.00 – 9.00	6.64 ± 1.11	2.00 – 9.00	-0.72	0.47
Apgar Score (5minute)	8.8 ± 1.3	4.00 -10.00	8.8 ± 1.27	4.00 – 10.00	8.7 ± 1.4	4.00 – 10.00	-0.44	0.66

Table 4.5: Descriptive statistics of neonates delivered in 2002

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 319)		(n = 159)		(n = 160)			
Maternal age (years)	25.6 ± 5.88	15.00 -45.00	25.5 ± 5.66	15.00 – 40.00	25.7 ± 6.12	16.00 – 45.00	0.25	0.802
Parity	2.04 ± 2.2	0.00 – 13.00	2.04 ± 2.01	0.00 – 10.00	2.03 ± 2.41	0.00 – 13.00	-0.051	0.96
Gestational age (weeks)	37.03 ± 2.63	28.00 – 55.00	37.14 ± 2.65	28.00 – 55.00	36.9 ± 2.6	29.00 – 47.00	-0.766	0.444
Birth weight (kg)	3.08 ± 0.47	1.50 -4.40	3.17 ± 0.46	1.60 -4.40	2.99 ± 0.48	1.50 -3.80	-3.252	0.001
Apgar Score (1minute)	7.01 ± 1.6	1.00 – 10.00	6.96 ± 1.7	2.00 – 10.00	7.07 ± 1.52	2.00 – 10.00	0.625	0.532
Apgar Score (5minute)	8.79 ± 1.4	2.00 – 10.00	8.72 ± 1.41	3.00 – 10.00	8.88 ± 1.31	4.00 – 10.00	0.995	0.320

Table 4.6shows descriptive statistics of the sample population of newborns delivered in 2003 (n = 602, males n = 325 and females n = 277). The mean birth weight (BW) of the entire sample population was (3.09 ± 0.54) kg. Also from the Table, the minimum BW of newborns was1.15 kgwhile the maximum BW of newborns was5.15 kg. The table shows the mean BW of male and female newborns to be 3.16 ± 0.48 and 3.09 ± 0.55 kg respectively, showing no statistical significant difference the sexes, (t = -1.575, p = 0.116). Also, birth weights of male newborns ranged from (1.50 -5.00) kg while that of their female counterparts ranged from (1.15-5.15) kg. The also revealed no statistical significant difference between the males and females with respect to variables tested (maternal age, parity, gestational age and Apgar score) with p > 0.05.

Table 4.7 shows descriptive statistics of the sample population of newborns delivered in 2004 (n = 561, males n = 307 and females n = 254). The mean birth weight (BW) of the entire sample population is (3.13 ± 0.51) kg. From the table, the minimum BW of all the sample population was1.40 kgwhile the maximum BW of newborns was4.85 kg. The table shows the mean BW of male and female newborns to be 3.17 ± 0.53 and 3.0 ± 0.54 kg respectively, showing that male newborns weigh significantly higher compared to female newborns, (t = -3.577, p <0.001). Also, birth weight of male newborns ranged from (1.50 -4.85) kg while that of their female counterparts ranged from (1.40 -4.60) kg. The result shows no statistical significant difference between the sexes with respect to other variables tested (maternal age, parity, gestational age and Apgar score).

Table 4.6: Descriptive statistics of neonates delivered in 2003

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 602)		(n = 325)		(n = 277)			
Maternal age (years)	26.5 ± 5.77	15.00 – 45.00	26.49 ± 6.04	15.00 – 43.00	26.57 ± 5.44	15.00 – 45.00	0.158	0.875
Parity	2.53 ± 2.39	0.00 – 11.00	2.59 ± 2.53	0.00 – 11.00	2.46 ± 2.21	0.00 – 10.00	-0.646	0.518
Gestational age (week)	37.1 ± 2.73	28.00 – 51.00	37.3 ± 2.57	28.00 – 50.00	36.9 ± 2.89	28.00 - 51.00	-1.850	0.065
Birth weight (kg)	3.13 ± 0.51	1.15-5.15	3.16 ± 0.48	1.50 -5.00	3.09 ± 0.55	1.15-5.15	-1.575	0.116
Apgar Score (1minute)	7.29 ± 1.51	2 .00 – 10.00	7.31 ± 1.56	2.00 – 10.00	7.28 ± 1.45	2.00 – 10.00	-0.187	0.852
Apgar Score (5minute)	9.05 ± 1.157	3.00 – 10.00	9.09 ± 1.19	4.00 – 10.00	9.01 ± 1.12	3.00 – 10.00	-0.790	0.430

Table 4.7: Descriptive statistics of neonates delivered in 2004

	Combined population		Male		Female			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 561)		(n = 307)		(n = 254)			
Maternal age (years)	26.5 ± 5.82	15.00 – 45.00	26.6 ± 5.66	15.00 – 41.00	26.41 ± 6.0	15 .00 – 45.00	-0.439	0.661
Parity	2.49 ± 2.28	0.00 – 13.00	2.58±2.36	0.00 – 11.00	2.38±2.18	0.00 – 13.00	-1.025	0.306
Gestational age (week)	37.19 ± 2.78	28.00 – 48.00	37.39±2.54	28.00 – 48.00	36.9±3.03	28.00 – 48.00	-1.960	0.050
Birth weight (kg)	3.09 ± 0.54	1.40 -4.85	3.17 ± 0.53	1.50 -4.85	3.0 ± 0.54	1.40 -4.60	-3.577	<0.001
Apgar Score (1minute)	7.4 ± 1.53	1.00 – 10.00	7.45 ± 1.48	2.00 – 10.00	7.43 ± 1.59	1.00 – 9.00	-1.101	0.919
Apgar Score (5minute)	9.03 ± 1.26	3.00 – 10.00	9.04 ± 1.16	4.00 – 10.00	9.02 ± 1.38	3.00 – 10.00	-0.144	0.885

Table 4.8shows descriptive statistics of the sample population of neonates delivered in 2005 (n = 385, males n = 194 and females n = 191). The mean birth weight (BW) of the entire sample population was (3.18±0.57) kg. From the Table, the minimum BW of all the sample population was1.20 kilogramwhile the maximum BW of neonates was5.00 kg. The Table shows the mean BW of male and female neonates to be 3.24 ± 0.56 and 3.11 ± 0.58 kg respectively, showing that male neonates weigh significantly higher compared to female neonates, ($t = -2.274$, $p = 0.024$). Also, birth weight of male neonates ranged from (1.50 -4.60) kg while that of their female counterparts ranged from (1.20 -5.00) kg. The result shows no statistical significant difference between the sexes with respect to other variables tested (maternal age, parity, gestational age and Apgar score, birth length, head circumference, placental weight) with $p > 0.05$ except chest circumference at $p = 0.038$.

Table 4.9shows descriptive statistics of the sample population of neonates delivered in 2006 (n = 770, males n = 394 and females n = 376). The mean birth weight (BW) of the entire sample population was (3.10 ± 0.55) kg. From the table, the minimum BW of all the sample population was1.20 kgwhile the maximum BW of neonates was5.10 kg. The table shows the mean BW of male and female neonates to be 3.15±0.56 and 3.05±0.54 kg respectively, indicating that male neonates weigh significantly higher compared to female neonates in this sample population, ($t = -2.618$, $p = 0.009$). Also, birth weight of male neonates ranged from (1.20 -5.10) kg while that of their female counterparts ranged from (1.40 -4.65) kg. The result revealed that there was statistical significant difference in birth length, head circumference, chest circumference and placental weight between the sexes with ($p < 0.001$, $p = 0.005$, $p = 0.003$ and $p = 0.009$)

respectively except for maternal age, parity, gestational age and AS which are not statistically significant.

Table 4.8: Descriptive statistics of neonates delivered in 2005

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 385)		(n = 194)		(n = 191)			
Maternal age (years)	27.3 ± 5.90	15.00 -42.00	27.3 ± 6.10	15.00 – 42.00	27.26 ± 5.74	15.00 – 41.00	-0.122	0.903
Parity	2.05 ± 2.05	0.00 – 13.00	2.03 ± 1.99	0.00 – 8.00	2.07 ± 2.11	0.00 – 13.00	0.227	0.820
Gestational age (weeks)	37.4±2.60	28.00 – 47.00	37.46 ± 2.49	28.00 – 45.00	37.29 ± 2.73	28.00 -47.00	-0.660	0.509
Birth weight (kg)	3.18±0.57	1.20 – 5.00	3.24 ± 0.56	1.50 -4.60	3.11 ± 0.58	1.20 -5.00	-2.274	0.024
Placental weight (kg)	0.64 ± 0.153	0.30 -1.40	0.65 ± 0.161	0.30 -1.30	0.63 ± 0.143	0.33-1.40	-1.273	0.204
Apgar score (1minute)	7.4±1.44	2.00 – 10.00	7.39 ± 1.45	2.00 – 10.00	7.45 ± 1.43	3.00 – 10.00	0.363	0.717
Apgar score (5minute)	9.04±1.09	4.00 – 10.00	9.02±1.12	4.00 – 10.00	9.06 ± 1.07	5.00 – 10.00	0.331	0.741
Birth length (cm)	51.2 ± 3.92	37.00 – 64.00	51.49±3.89	39.00 – 64.00	50.94±3.95	37.00 – 58.00	-1.370	0.172
Head circumference (cm)	34.68 ± 2.18	27.00 – 40.00	34.8±2.26	28.00 – 40.00	34.5±2.1	27.00 – 38.00	-1.471	0.142
Chest circumference (cm)	33.78 ± 2.55	24.00 – 39.00	34.05±2.81	24.00 – 39.00	33.5±2.25	26.00 – 38.00	-2.077	0.038

Table 4.9: Descriptive statistics of neonates delivered in 2006

	Combined population		Males		Females			
	Mean ± SD (n = 770)	Min-Max	Mean ± SD (n = 394)	Min-Max	Mean ± SD (n = 376)	Min-Max	t	*P*-value
Maternal age (years)	27.24 ± 5.6	15.00 – 46.00	26.96 ± 5.57	15.00 – 45.00	27.54 ± 5.63	15.00 – 46.00	1.451	0.147
Parity	1.73 ± 1.51	0.00 – 10.00	1.72 ± 1.49	0.00 – 10.00	1.73 ± 1.53	0.00 – 8.00	0.0249	0.980
Gestational age (weeks)	37.45 ± 2.53	28.00 – 51.00	37.46 ± 2.42	28.00 – 44.00	37.44 ± 2.65	28.00 – 51.00	-0.0974	0.922
Birth weight (kg)	3.10 ± 0.55	1.20 -5.10	3.15±0.56	1.20 -5.10	3.05±0.54	1.40 -4.65	-2.618	0.009
Placental weight (kg)	0.64 ± 0.18	0.18-1.18	0.66 ± 0.17	0.18-1.12	0.62 ± 0.18	0.18-1.18	-2.618	0.009
Apgar score (1m)	7.65 ± 1.48	1.00 – 9.00	7.69±1.51	1.00 – 9.00	7.61±1.45	2.00 – 9.00	-0.714	0.476
Apgar score (5m)	9.22 ± 1.162	3.00 – 10.00	9.22±1.23	3.00 – 10.00	9.23±1.08	3.00 – 10.00	0.187	0.852
Birth length (cm)	49.9 ± 3.25	28.00 – 60.00	50.3±3.15	28.00 – 57.00	49.5±3.31	38.00 – 60.00	-3.431	<0.001
Head circumference (cm)	34.66 ± 2.23	20.00 – 40.00	34.88±2.24	20.00 – 40.00	34.43 ± 2.19	22.00 – 39.00	-2.829	0.005
Chest circumference (cm)	33.12 ± 2.32	18.00 – 38.00	33.35 ± 2.37	18.00 – 38.00	32.85 ± 2.25	20.00 – 36.00	-3.004	0.003

Table 4.10shows descriptive statistics of the sample population of neonates delivered in 2007 (n = 980, males n = 544 and females n = 436). The mean birth weight (BW) of the entire sample population was (3.09 ± 0.48) kg. Also from the Table, the minimum BW of neonates in the entire sample was1.80 kgwhile the maximum BW of neonates was4.75 kg. The table shows the mean BW of male and female neonates to be 3.15 ± 0.47 and 3.02 ± 0.48 kg respectively, indicating that male neonates weigh significantly higher compared to female neonates in this sample population, ($t = -4.285$, $p <0.001$). Also, birth weight of male neonates ranged from (1.80 -4.75) kg while that of their female counterparts ranged from (1.80 -4.30) kg. The result also revealed statistical significant difference in all the variable tested between the sexes with $p < 0.05$.

Table 4.11shows descriptive statistics of the sample population of neonates delivered in 2008 (n = 1225, males n = 663, and females n = 562). The mean birth weight (BW) of the entire sample population was (3.08 ± 0.53) kg. From the table, the minimum BW of all the sample population was1.01 kgwhile the maximum BW of neonates was6.10 kg. The table shows the mean BW of male and female neonates to be 3.15 ± 0.54 and 2.99 ± 0.52 kg respectively, indicating that male neonates weigh significantly higher compared to female neonates in this sample population, ($t = -5.304$, $p <0.001$). Also from the table, birth weight of male neonates ranged from (1.09-6.10) kg while that of their female counterparts ranged from (1.01-5.40) kg. The result also shows there was statistical difference in birth length, head circumference, chest circumference and placental weight between the sexes with ($p <0.001$, $p <0.001$, $p <0.001$ and $p = 0.03$) respectively except for maternal age, parity, gestational age and Apgar score at $p > 0.05$.

Table 4.12shows descriptive statistics of the sample population of neonates delivered in 2009 (n = 805, males n = 418, and females n = 387). The mean birth weight (BW) of the entire sample population was (3.06 ±0.53) kg. From the table, the minimum BW of the sample population was1.10 kgwhile the maximum BW of neonates was4.77 kg. The table shows the mean BW of male and female neonates to be 3.13 ±0.54 and 2.98 ±0.51 kg respectively, indicating that male neonates weigh significantly higher compared to female neonates in this sample population, (t = -4.142, p <0.001). Also from the table, birth weight of male neonates ranged from (1.10 – 4.77) kg while that of their female counterparts ranged from (1.13 – 4.60) kg. The result indicate statistical significant difference in gestational age, birth length, and head circumference between the sexes with (p <0.001, p <0.001 and p = 0.002) respectively, but no statistical significant difference was seen in other variable tested.

Table 4.10: Descriptive statistics of neonates delivered in 2007

	Combined population		Males		Females			
	Mean ± SD (n=980)	Min-Max	Mean ± SD (n=544)	Min-Max	Mean ± SD (n=436)	Min-Max	t	*P*-value
Maternal age (years)	26.01 ± 5.35	15.00 -45.00	25.68 ± 5.59	17.00 – 45.00	26.42 ± 5.01	15.00 – 36.00	2.172	0.030
Parity	1.49 ± 1.29	0.00 – 10.00	1.41 ± 1.25	0.00 – 10.00	1.60 ± 1.35	0.00 – 7.00	2.296	0.022
Gestational age (weeks)	37.46 ± 2.24	30.00 – 44.00	37.77 ± 1.88	31.00 – 43.00	37.07 ± 2.57	30.00 – 44.00	-4.914	<0.001
Birth Weight (kg)	3.09 ± 0.48	1.80 -4.75	3.15 ± 0.47	1.80 -4.75	3.02 ± 0.48	1.80 -4.30	-4.285	<0.001
Placental weight (kg)	0.603 ± 0.16	0.20 -1.40	0.62 ± 0.17	0.30 -1.40	0.59 ± 0.14	0.20 -1.30	-2.883	0.004
Apgar Score (1minute)	7.63 ± 1.27	2.00 – 10.00	7.73 ± 1.17	3.00 – 9.00	7.49 ± 1.37	2.00 – 10.00	-2.864	0.004
Apgar Score (5minute)	9.38 ± 0.90	5.00 – 10.00	9.48 ± 0.79	5.00 – 10.00	9.26 ± 1.01	5.00 – 10.00	-3.858	<0.001
Birth Length (cm)	49.05 ± 3.64	40.00 – 57.00	49.41 ± 3.47	40.00 – 57.00	48.60 ± 3.79	40.00 – 56.00	-3.456	<0.001
Head circumference (cm)	33.48 ± 2.17	28.00 – 40.00	33.72 ± 2.07	28.00 – 40.00	33.18 ± 2.25	28.00 – 38.00	-3.878	<0.001
Chest circumference (cm)	33.22 ± 2.15	26.00 – 39.00	33.44 ± 2.15	26.00 – 39.00	32.94 ± 2.12	28.00 – 36.00	-3.623	<0.001

Table 4.11: Descriptive statistics of neonates delivered in 2008

	Combined population		Males		Females			
	Mean ± SD (n = 1225)	Min-Max	Mean ± SD (n = 663)	Min-Max	Mean ± SD (n = 562)	Min-Max	t	*P*-value
Maternal age (years)	26.82 ± 5.69	15.00 – 45.00	26.86 ± 5.78	16.00 – 45.00	26.77 ± 5.59	15.00 – 44.00	-0.274	0.784
Parity	1.84 ± 1.88	0.00 – 13.00	1.87 ± 1.94	0.00 – 13.00	1.81 ± 1.81	0.00 – 10.00	-0.548	0.584
Gestational age (weeks)	37.37 ± 2.63	28.00 – 50.00	37.48 ± 2.43	28.00 – 50.00	37.24 ± 2.84	28.00 – 50.00	-1.587	0.113
Birth Weight (kg)	3.08 ± 0.53	1.01-6.10	3.15 ± 0.54	1.09-6.10	2.99 ± 0.52	1.01-5.40	-5.304	<0.001
Placental weight (kg)	0.62 ± 0.15	0.20 -1.55	0.63±0.16	0.30 -1.55	0.61±0.14	0.20 -1.12	-2.136	0.033
Apgar score (1minutes)	7.86 ± 1.28	2.00 – 10.00	7.85±1.26	3.00 – 10.00	7.87±1.29	2.00 – 10.00	0.372	0.710
Apgar score (5minutes)	9.42 ± 0.90	3.00 – 10.00	9.43±0.87	5.00 – 10.00	9.39±0.94	3.00 – 10.00	-0.668	0.504
Birth length (cm)	50.37 ± 3.9	29.00 – 62.00	50.75±3.55	35.00 – 62.00	49.92±4.24	29.00 – 61.00	-3.738	<0.001
Head circumference (cm)	33.99 ± 2.38	20.00 – 41.00	34.22±2.24	20.00 – 41.00	33.71±2.49	20.00 – 39.00	-3.761	<0.001
Chest circumference (cm)	33.05 ± 2.36	20.00 – 43.00	33.38±2.19	22.00 – 43.00	32.67±2.49	20.00 – 38.00	-5.315	<0.001

Table 4.12: Descriptive statistics of neonates delivered in 2009

	Combined population		Males		Females			
	Mean ± SD (n = 805)	Min-Max	Mean ± SD (n = 418)	Min-Max	Mean ± SD (n = 387)	Min-Max	t	*P*-value
Maternal age (years)	26.55 ±5.71	16.00 – 46.00	26.89 ±5.84	17.00 – 46.00	26.18 ±5.54	16.00 – 42.00	-1.770	0.077
Parity	1.99 ±2.10	0.00 – 12.00	2.07 ±2.19	0.00 – 12.00	1.91 ±1.99	0.00 – 10.00	-1.077	0.282
Gestational age (weeks)	37.62 ±2.57	28.00 – 55.00	37.91 ±2.68	28.00 – 55.00	37.31 ±2.42	28.00 – 53.00	-3.320	<0.001
Birth Weight (kg)	3.06 ±0.53	1.10 -4.77	3.13 ±0.54	2.00 – 9.00	2.98 ±0.51	3.00 – 9.00	-4.142	<0.001
Placental weight (kg)	0.62 ± 0.13	0.16-1.30	0.63 ± 0.13	0.16-1.30	0.62 ± 0.12	0.20 -0.99	-1.394	0.164
Apgar Score (1minutes)	7.61 ±1.28	2.00 – 9.00	7.58 ±1.27	5.00 – 10.00	7.64 ± 1.29	3.00 – 10.00	0.683	0.495
Apgar Score (5minutes)	9.03 ±0.92	3.00 – 10.00	9.01 ± 0.89	1.10 -4.77	9.05 ± 0.96	1.13-4.60	0.567	0.571
Birth Length (cm)	49.23 ±4.38	27.00 – 59.00	49.72 ± 4.24	32.00 – 59.00	48.69 ± 4.48	27.00 – 59.00	-3.353	<0.001
Head circumference (cm)	34.42 ±2.20	15.00 – 40.00	34.65 ± 2.09	25.00 – 40.00	34.17 ± 2.29	15.00 – 39.00	-3.095	0.002
Chest circumference (cm)	32.96 ±2.44	15.00 – 43.00	33.07 ± 2.22	24.00 – 39.00	32.85 ± 2.67	15.00 – 43.00	-1.303	0.193

Table 4.13shows descriptive statistics of the sample population of neonates delivered in 2010 (n = 1230, males n = 655, and females n = 575). The mean birth weight (BW) of the entire sample population was (3.08 ± 0.55) kg. From the table, the minimum BW of the sample population was1.00 kgwhile the maximum BW of neonates was5.60 kg. The table shows the mean BW of male and female neonates to be 3.15±0.53 and 2.99±0.56 kg respectively, indicating that male neonates weigh significantly higher compared to female neonates in this sample population, (t = -5.059, p <0.001). Also from the table, birth weight of male neonates ranged from (1.00 -5.60) kg while that of their female counterparts ranged from (1.15-5.06) kg. The result from the table shows significant difference in gestational age, birth length, head circumference, chest circumference, placental weight, maternal height, maternal weight and gestational weight gainbetween male and female neonates. The males having higher means compared to the female neonates, with statistically significant difference (p = 0.001, p < 0.001, p = 0.003, p < 0.001, p = 0.004, p = 0.002, p = 0.002 and p < 0.001) respectively except for maternal age, parity, Apgar scor, BMI and PCV with, (p > 0.05).

Table 4.14shows descriptive statistics of the sample population of neonates delivered in 2011 (n = 841, males n = 425, and females n = 416). The mean birth weight (BW) of the entire sample population was (3.07 ± 0.53) kg. From the table, the minimum BW of the sample population was1.10 kgwhile the maximum BW of neonates was4.82 kg. The table shows the mean BW of male and female neonates to be 3.13 ± 0.54 and 2.99 ± 0.51 kg respectively, indicating that at birth male neonates weigh significantly higher compared to female neonates in this sample population, (t = -3.779, p <0.001).Also from the table, birth weight of male neonates ranged from (1.10 -4.66) kg while that of their female counterparts ranged from (1.13-4.82) kilogram. Results from the table shows there are significant difference between the sexes in gestational age, birth length, head circumference and gestational weight gain with the males having higher values at birth, with statistically significant difference ($p = 0.004$, $p < 0.001$, $p = 0.001$ and $p = 0.005$) respectively. However, there was no statistical significance in maternal age (MA), parity, Apgar score, chest circumference, placental, maternal (height, weight, gestational weight gain, BMI), and PCV with $p > 0.05$.

Table 4.15shows descriptive statistics of the sample population of neonates delivered in 2012 (n = 910, males n = 490, and females n = 420). The mean birth weight (BW) of the entire sample population was (3.07±0.56) kg. From the table, the minimum BW of the sample population was1.01 kgwhile the maximum BW of neonates was6.20 kg. The result in the table shows the mean BW of male and female neonates to be 3.16±0.58 and 2.97±0.51 kg respectively, indicating that at birth male neonates weigh significantly heavier compared to female neonates in this sample population, ($t = -5.107$, $p <0.001$). Also from the table, birth weight of male neonates ranged from (1.25-6.20) kg while that of their female counterparts ranged from (1.01-4.57) kg. t – test also reveals there were statistical significance difference between male and female neonates in BL, HC, CC, PW, GWG ($p < 0.001$, $p = 0.014$, $p = 0.002$, $p = 0.008$ and 0.03) respectively. However, there is no statistical significant in maternal age, parity, gestational age. AS, maternal (height, weight, BMI) and PCV.

Table 4.13: Descriptive statistics of neonates delivered in 2010 and maternal characteristics

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n = 1230)		(n = 655)		(n = 575)			
Maternal age (years)	26.52 ± 5.77	15.00 – 49.00	26.62 ± 5.75	15.00 – 49.00	26.41 ± 5.79	15.00 – 45.00	-0.646	0.518
Parity	1.92 ± 2.09	0.00 – 12.00	1.89 ± 2.06	0.00 – 11.00	1.94 ± 2.13	0.00 – 12.00	0.319	0.750
Gestational age (weeks)	37.75 ± 2.27	28.00 – 57.00	37.94±2.25	28.00 – 57.00	37.52±2.27	29.00 – 48.00	-3.239	0.001
Apgar Score (1minute)	7.57 ± 1.24	1.00 – 9.00	7.52±1.27	2.00 – 9.00	7.61±1.20	1.00 – 9.00	1.254	0.210
Apgar Score (5minutes)	9.03 ± 0.95	1.00 – 10.00	9.01±0.96	3.00 – 10.00	9.04±0.94	1.00 – 10.00	0.603	0.546
Birth weight (kg)	3.08 ± 0.55	1.00 -5.60	3.15±0.53	1.00 -5.60	2.99±0.56	1.15-5.06	-5.059	<0.001
Placental weight (kg)	0.63 ± 0.14	0.18-1.25	0.64±0.14	0.19-1.25	0.61±0.14	0.18-1.25	-2.850	0.004
Birth Length (cm)	50.02 ± 3.12	33.00 – 60.00	50.41±2.97	38.00 – 60.00	49.57±3.23	33.00 – 60.00	-4.773	<0.001
Head circumference (cm)	34.42 ± 2.20	15.00 – 40.00	34.59±2.28	15.00 – 40.00	34.22±2.09	24.00 – 40.00	-2.952	0.003
Chest circumference (cm)	32.98 ± 2.29	18.00 – 42.00	33.19±2.21	18.00 – 42.00	32.74±2.37	22.00 – 42.00	-3.386	<0.001
	(n = 875)		**(n = 448)**		**(n = 427)**			
Maternal height (cm)	159.31 ± 6.19	145.00 – 182.00	159.95±6.37	145.00 – 182.00	158.63±5.95	145.00 – 178.00	-3.162	0.002
Maternal weight (kg)	66.23 ± 11.37	39.00 -108.10	66.71±11.49	41.00 – 107.00	65.72±11.23	39.00 -108.10	-1.287	0.002
Gestational weight gain (kg)	8.03 ± 3.18	2.00 – 19.00	8.56 ± 3.15	2.00 – 19.00	7.49 ± 3.13	2.00 -18.50	-5.044	<0.001
BMI	25.99 ± 3.60	17.23 – 43.30	25.98 ± 3.62	17.36 – 37.91	26.02 ± 3.59	17.23 – 43.30	0.179	0.858
PCV	0.32 ± 0.04	0.18-0.62	0.33 ± 0.05	0.18-0.62	0.32 ± 0.04	0.18-0.54	-1.264	0.206

Table 4.14: Descriptive statistics of neonates delivered in 2011 and maternal characteristics

	Combined population		Males		Females			
	Mean ± SD (n = 841)	Min-Max	Mean ± SD (n = 425)	Min-Max	Mean ± SD (n = 416)	Min-Max	t	*P*-value
Maternal age (years)	26.58 ± 5.68	16.00 – 46.00	26.69 ± 5.81	17.00 – 46.00	26.46 ± 5.55	16.00 – 42.00	-0.572	0.568
Parity	2.11 ± 2.109	0.00 – 12.00	2.23 ± 2.22	0.00 – 12.00	1.98 ± 1.97	0.00 – 10.00	-1.719	0.086
Gestational age (weeks)	37.65 ± 2.64	28.00 – 58.00	37.92 ± 2.78	28.00 – 58.00	37.39 ± 2.46	28.00 – 49.00	-2.926	0.004
Apgar Score (1minute)	7.70 ± 1.26	1.00 – 9.00	7.69 ± 1.24	1.00 – 9.00	7.70 ± 1.29	2.00 – 9.00	0.0273	0.978
Apgar Score (5minute)	9.15 ± 0.93	4.00 – 10.00	9.14 ± 0.91	5.00 – 10.00	9.15 ± 0.95	4.00 – 10.00	0.128	0.898
Birth weight (kg)	3.07 ± 0.53	1.10 -4.82	3.13 ± 0.54	1.10 -4.66	2.99 ± 0.51	1.13-4.82	-3.779	<0.001
Placental weight (kg)	0.63 ± 0.13	0.20 -1.30	0.63 ± 0.14	0.20 -1.30	0.62 ± 0.12	0.20 -1.12	-0.981	0.327
Birth Length (cm)	50.41 ± 3.42	35.00 – 61.00	50.81 ± 3.43	35.00 – 60.00	50.01 ± 3.37	38.00 – 61.00	-3.386	<0.001
Head circumference (cm)	34.44 ± 2.04	24.00 – 40.00	36.66 ± 2.09	24.00 – 40.00	34.22 ± 1.96	25.00 – 39.00	-3.195	0.001
Chest circumference (cm)	33.05 ± 2.25	23.00 – 41.00	33.17 ± 2.26	24.00 – 38.00	32.93 ± 2.23	23.00 – 41.00	-1.529	0.127
Maternal height (cm)	158.86 ± 6.01	144.00 – 181.00	159.18 ± 5.99	147.00 -180.50	158.54 ± 6.0	144.00 – 181.00	-1.525	0.128
Maternal weight (kg)	68.54 ± 13.08	36.00 – 129.00	69.32 ± 13.45	40.00 – 129.00	67.73 ± 12.66	36.00 – 110.00	-1.765	0.078
Gestational weight gain (kg)	6.36 ± 2.81	1.00 – 16.00	6.62 ± 2.97	1.00 – 16.00	6.08 ± 2.60	2.00 – 16.00	-2.808	0.005
BMI	27.06 ± 4.39	15.38 – 47.96	27.28 ± 4.62	17.19 – 47.96	26.85 ± 4.15	15.38 – 42.59	-1.417	0.157
PCV	0.33 ± 0.05	0.18-0.61	0.33 ± 0.05	0.18-0.61	0.33 ± 0.045	0.18-0.53	-0.936	0.349

Table 4.15: Descriptive statistics of neonates delivered in 2012 and maternal characteristics

	Combined population		Males		Females			
	Mean ± SD	Min-Max	Mean ± SD	Min-Max	Mean ± SD	Min-Max	t	*P*-value
	(n= 910)		(n = 490)		(n = 420)			
Maternal age (years)	27.54±5.69	15.00 – 46.00	27.74±5.69	16.00 – 46.00	27.32±5.69	15.00 – 44.00	-1.092	0.275
Parity	1.99±1.98	0.00 – 13.00	2.08±2.07	0.00 – 13.00	1.89±1.87	0.00 – 8.00	-1.388	0.165
Gestational age (weeks)	37.67±2.57	28.00 – 52.00	37.82±2.48	28.00 – 52.00	37.50±2.67	28.00 – 50.00	-1.852	0.064
Apgar Score (1minute)	7.90±1.29	2.00 – 10.00	7.91±1.32	2.00 – 10.00	7.89±1.27	2.00 – 9.00	-0.280	0.780
Apgar Score (5minute)	9.29±0.95	3.00 – 10.00	9.31±0.93	3.00 – 10.00	9.27±0.97	3.00 – 10.00	-0.692	0.489
Birth weight (kg)	3.07±0.56	1.01-6.20	3.16±0.58	1.25-6.20	2.97±0.51	1.01-4.57	-5.107	<0.001
Placental weight (kg)	0.63±0.16	0.05-1.90	0.64±0.17	0.05-1.90	0.61±0.13	0.20 -1.30	-2.657	0.008
Birth Length (cm)	50.59±3.65	32.00 – 62.00	51.04±3.59	37.00 – 62.00	50.08±3.65	32.00 – 59.00	-3.974	<0.001
Head circumference (cm)	34.37±2.19	20.00 – 43.00	34.54±2.30	20.00 – 43.00	34.18±2.05	24.00 – 39.00	-2.461	0.014
Chest circumference (cm)	32.98±2.31	22.00 – 42.00	33.20±2.27	22.00 – 42.00	32.73±2.33	23.00 – 38.00	-3.098	0.002
	(n=560)		**(n=296)**		**(n=264)**			
Maternal height (cm)	160.25±5.76	142.00 – 180.00	160.68±5.88	145.00 – 180.00	159.77±5.58	142.00 – 178.00	-1.885	0.060
Maternal weight (kg)	68.22±10.11	45.00 – 105.00	68.77±10.09	45.00 – 105.00	67.59±10.11	45.00 – 98.00	-1.373	0.170
Gestational weight gain (kg)	6.04±2.69	1.00 – 16.00	6.28±2.74	1.00 – 16.00	5.78±2.61	2.00 -15.30	-2.176	0.030
BMI	26.46 ± 2.88	19.47 – 37.20	26.54 ± 2.82	19.81 – 37.20	26.38 ± 2.95	19.47 – 36.79	-0.632	0.527
PCV	0.32±0.05	0.18-0.52	0.32±0.05	0.20 -0.52	0.32±0.049	0.18-0.48	-1.216	0.225

Table 4.16shows descriptive statistics of the sample population of neonates delivered in 2013 (n = 910, males n = 473, and females n = 437). The mean birth weight (BW) of the entire sample population is (3.06±0.53) kg. Also from the table, the minimum BW of the sample population was1.09 kgwhile the maximum BW of neonates was4.82 kg. The result on the Table 4.16 shows the mean BW of male and female neonates to be 3.15±0.54 and 2.97±0.50 kg respectively, indicating that at birth male neonates weigh significantly heavier compared to female neonates in this sample population, ($t = -5.160$, $p <0.001$). Also from Table 4.16, birth weight of male neonates ranged from (1.09-4.71) kg while that of their female counterparts ranged from (1.13-4.82) kg. The result also shows statistical significance between the sexes in gestational age, birth length, head circumference, chest circumference and placental weight. The male neonates having higher scores in the aforementioned parameters, ($p = 0.014$, $P < 0.001$, $P < 0.001$, $P < 0.001$, $P < 0.001$) respectively. However, there were no statistical significance in maternal age, parity and Apgar score, $p > 0.05$.

Table 4.17shows Descriptive statistics of the sample population of neonates delivered in 2014 (n = 740, males n = 432, and females n = 308). The mean birth weight (BW) of the entire sample population was (3.09±0.55) kg. Also from the table, the minimum BW of the sample population was1.15 kgwhile the maximum BW of neonates was6.50 kg. The result on Table 4.17 shows the mean BW of male and female neonates to be 3.16±0.544 and 3.01±0.541 kg respectively, indicating that at birth male neonates weigh significantly heavier compared to female neonates in this sample population, ($t = -3.806$, $p <0.001$). Also from Table 4.17, birth weight of male neonates ranged from (1.40 -6.50) kg while that of their female counterparts ranged from (1.15-

5.06) kg. The result also shows statistical significant between male and female neonates in gestational weight, birth length, head circumference and chest circumference, with males having higher values, ($p = 0.03$, $p < 0.001$, $p = 0.036$ and $p = 0.004$) respectively. However, there is no statistical significance in maternal age, parity, Apgar score and placental weight.

Table 4.16: Descriptive statistics of neonates delivered in 2013

	Combined population		Males		Females			
	Mean ± SD (n = 910)	Min-Max	Mean ± SD (n = 473)	Min-Max	Mean ± SD (n = 437)	Min-Max	t	*P*-value
Maternal age (years)	27.83±5.49	15.00 – 46.00	27.77 ± 5.55	15.00 – 44.00	27.90 ± 5.43	16.00 – 46.00	0.374	0.708
Parity	2.08±2.11	0.00 – 13.00	2.07 ± 2.16	0.00 – 13.00	2.09 ± 2.05	0.00 – 11.00	0.0927	0.926
Gestational age (weeks)	37.51±2.49	28.00 – 55.00	37.71 ± 2.47	28.00 – 55.00	37.30 ± 2.49	28.00 – 53.00	-2.450	0.014
Apgar Score (1minute)	7.72±1.33	1.00 – 10.00	7.67±1.36	1.00 – 10.00	7.77±1.29	3.00 – 10.00	1.167	0.243
Apgar Score (5minute)	9.18±0.99	3.00 – 10.00	9.17±0.96	5.00 – 10.00	9.18±1.05	3.00 – 10.00	0.144	0.886
Birth weight (kg)	3.06±0.53	1.09-4.82	3.15±0.54	1.09-4.71	2.97±0.50	1.13-4.82	-5.160	<0.001
Placental weight (kg)	0.63 ± 0.14	0.20 -1.55	0.64±0.14	0.30 -1.55	0.61±0.13	0.20 -1.12	-3.351	<0.001
Birth Length (cm)	50.56 ± 3.53	30.00 – 61.00	51.13±3.29	34.00 – 60.00	49.95±3.67	30.00 – 61.00	-5.078	<0.001
Head circumference (cm)	34.44 ± 2.28	15.00 – 42.00	34.72±2.24	23.00 – 42.00	34.14±2.29	15.00 – 40.00	-3.854	<0.001
Chest circumference (cm)	33.15 ± 2.41	15.00 – 43.00	33.42±2.26	23.00 – 43.00	32.86±2.54	15.00 – 38.00	-3.562	<0.001

Table 4.17: Descriptive statistics of neonates delivered in 2014

	Combined population		Males		Females			
	Mean ± SD (n = 740)	Min-Max	Mean ± SD (n = 432)	Min-Max	Mean ± SD (n = 308)	Min-Max	t	*P-value*
Maternal age (years)	27.09±5.52	15.00 – 48.00	27.21±5.67	15.00 – 48.00	26.94±5.30	16.00 – 43.00	-0.642	0.521
Parity	1.73±1.81	0.00 – 10.00	1.83±1.89	0.00 – 10.00	1.58±1.68	0.00 – 10.00	-1.876	0.061
Gestational age (weeks)	37.40±2.57	28.00 – 50.00	37.58±2.40	28.00 – 50.00	37.16±2.76	28.00 – 50.00	-2.200	0.028
Apgar Score (1minute)	7.81±1.30	2.00 – 10.00	7.79±1.29	3.00 – 10.00	7.85±1.32	2.00 – 10.00	0.620	0.535
Apgar Score (5minute)	9.31±0.95	3.00 – 10.00	9.33±0.90	5.00 – 10.00	9.29±1.00	3.00 – 10.00	-0.563	0.573
Birth weight (kg)	3.09±0.55	1.15-6.50	3.16±0.544	1.40 -6.50	3.01±0.541	1.15-5.06	-3.806	<0.001
Placental weight (kg)	0.63±0.16	0.20 -1.70	0.63 ± 0.16	0.27 – 1.1.70	0.62 ± 0.15	0.20 -1.30	-1.532	0.126
Birth Length (cm)	50.44±3.67	32.00 – 64.00	50.89±3.48	38.00 – 64.00	49.79±3.83	32.00 – 59.00	-4.048	<0.001
Head circumference (cm)	34.29±2.12	20.00 – 43.00	34.42±2.04	20.00 – 43.00	34.09±2.23	24.00 – 39.00	-2.106	0.036
Chest circumference (cm)	33.16±2.19	22.00 – 42.00	33.36±2.03	22.00 – 41.00	32.89±2.36	23.00 – 42.00	-2.926	0.004

Figure 4.1 shows the trends in mean birth weight (MBW) of male and female babies from 2001 to 2014. In male babies the MBW showed an upward trend from 2001 until it reached its peak in 2005 and then decline sharply in 2006 (- 0.09 kg), from 2007 down to 2014 MBW continued to show an inconsistent upward and downward trends. In female babies the trends inMBW showed a sharp decline 2002 and 2004 then reached its peak in 2005. From 2006 its started to show a downward trends til 2014. The wide gap seen between the line graph of male and female babies over the years is indicating the wide disparity in MBW between them.

Figure 4.2 shows the trends in mean placental weight(MPW) of male and female babies from 2005 to 2014. For male babies the MPW rose in 2006 which is the peak and then decline in 2007 (-0.04 kg), from 2008 MPW continued to show inconsistent upward and downward trends til 2014. In female babies MPW peaks in 2005 and then declined from 2006 (-0.01 kg) to 2007 (-0.04 kg) from 2008 to 2009 it showed an upward trend. Subsequent years showed inconsistent upward and downward trends til 2014. Also, the wide gap observed between the male and female babies graph indicate wide disparity in MPW values over the years.

Figure 4.3 shows the trends in mean birth length(MBL) of male and female babies from 2005 to 2014. In male babies MBL reached its peak in 2005 and showed a downward trend in 2006 (-1.19cm) and 2007 (-2.08cm) then rose in 2008 and declined in 2009. From 2010 it showed an upward trend til 2014. In female babies MBL peaked in 2005 and then began to show a downward trend from 2006 (-1.44cm) to 2007 (-2.34cm) and then rose in 2008 and declined in 2009 and subsequently from 2010 shows an upward trend til 2014.

Figure 4.4 shows the trends in mean head circumference (MHC) of male and female babies from 2005 to 2014. In male babies the MHC showed an inconsistent upward and downward trends from 2005 to 2010 until it peaked around 2011 and then showed a downward trend form 2012 (-2.12cm), 2013 (-1.94cm) and 2014 (-2.24cm). In female babies the MHC reached its peaked in 2005, then showed a downward trend in 2006 (-0.07cm), 2007 (-1.32cm) and 2008 (-0.79cm) and then from 2009 it rose and showed steady trends til 2014.

Figure 4.5 shows the trends in mean chest circumference(MCC) of male and female babies from 2005 to 2014. In male babies the MCCreached its peak in 2005 and then declined in 2006 (-0.7cm) and then subsequently from 2007 continued to show almost a steady trend down to 2014. In female babies the MCC reached itspeak in 2005 then declined in 2006 (-0.65cm) and then subsequently from 2007 continued to show almost a steady trend down to 2014. The wide gap seen between the line graphs of male and female babies indicates a wide disparity in MCC values between them over the years.

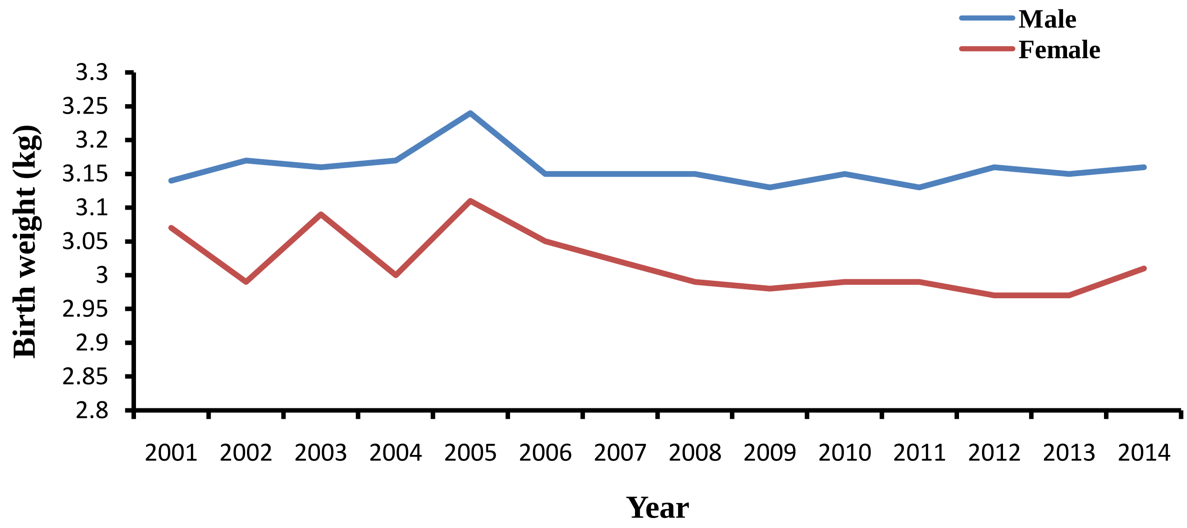

Figure 4.1: Fourteen year trend of mean birth weight in Gombe metropolis, Nigeria

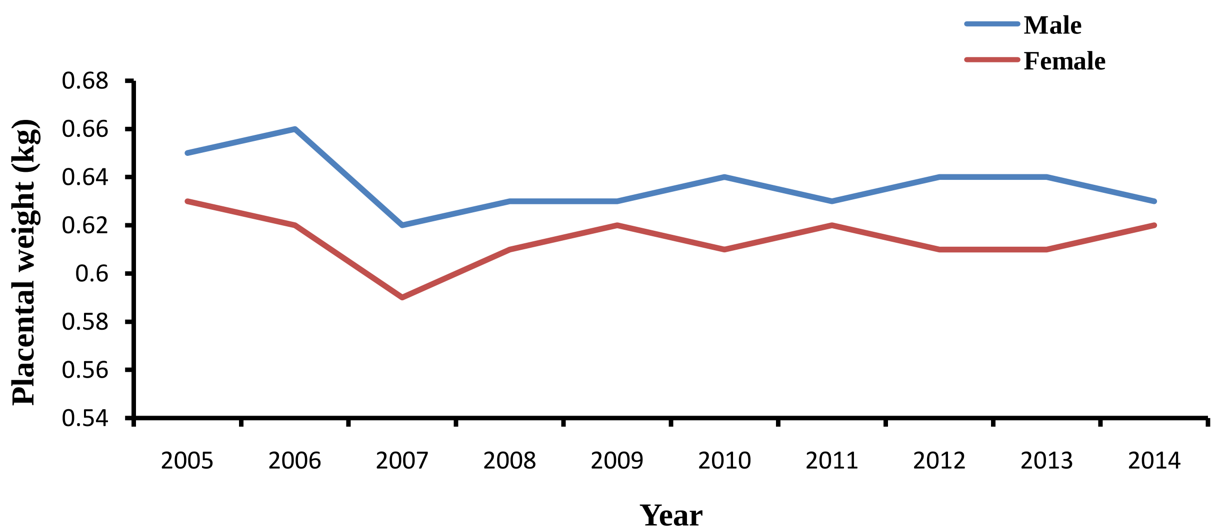

Figure 4.2: One-decade trend of mean placental weight in Gombe metropolis, Nigeria

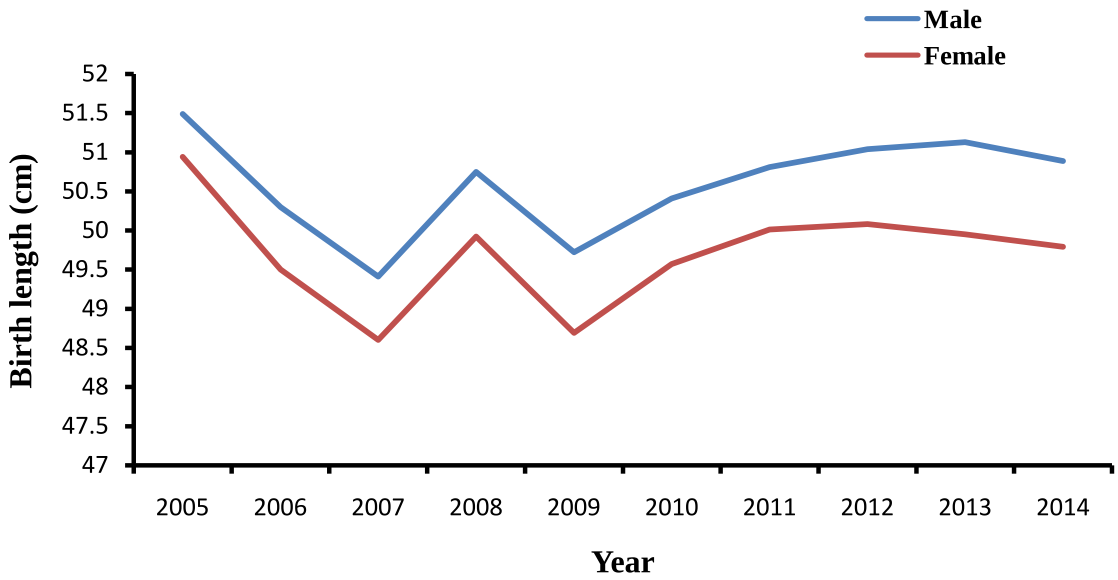

Figure 4.3: One-decade trend of mean birth length in Gombe metropolis, Nigeria

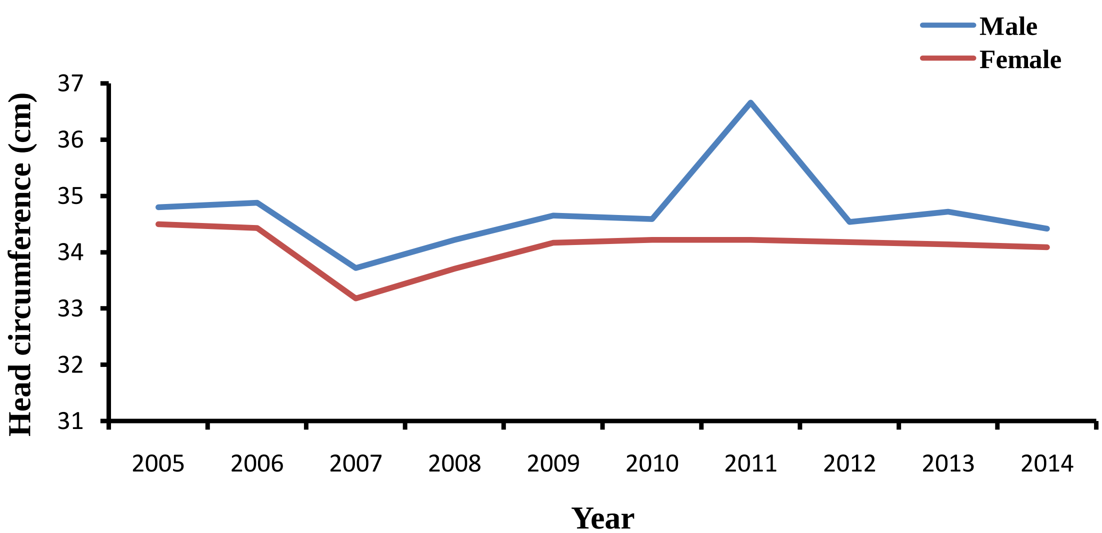

Figure 4.4: One-decade trend of mean head circumference in Gombe metropolis, Nigeria

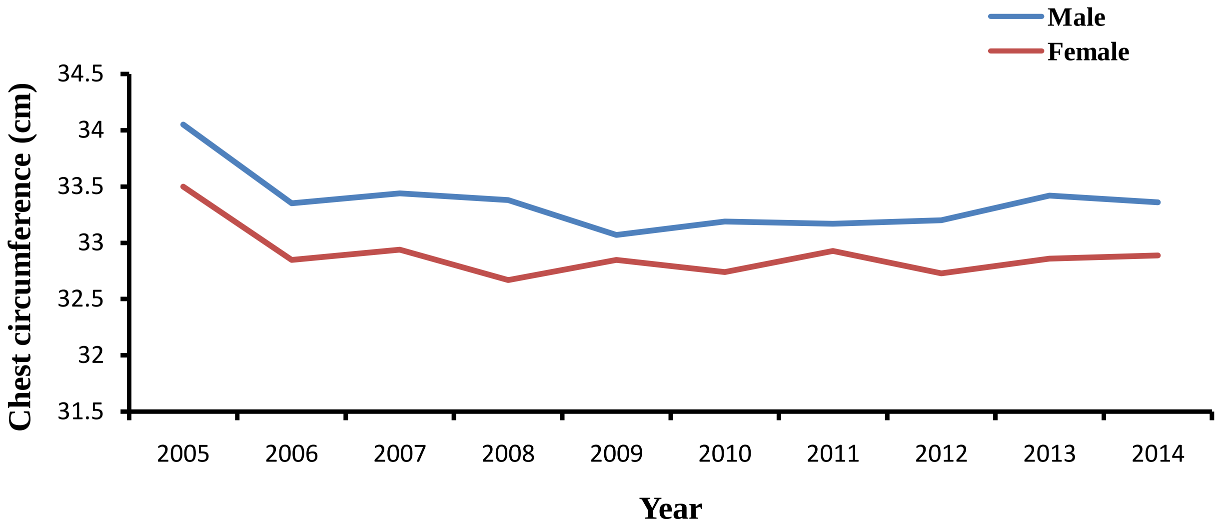

Figure 4.5: One-decade trend of mean chest circumference in Gombe metropolis, Nigeria

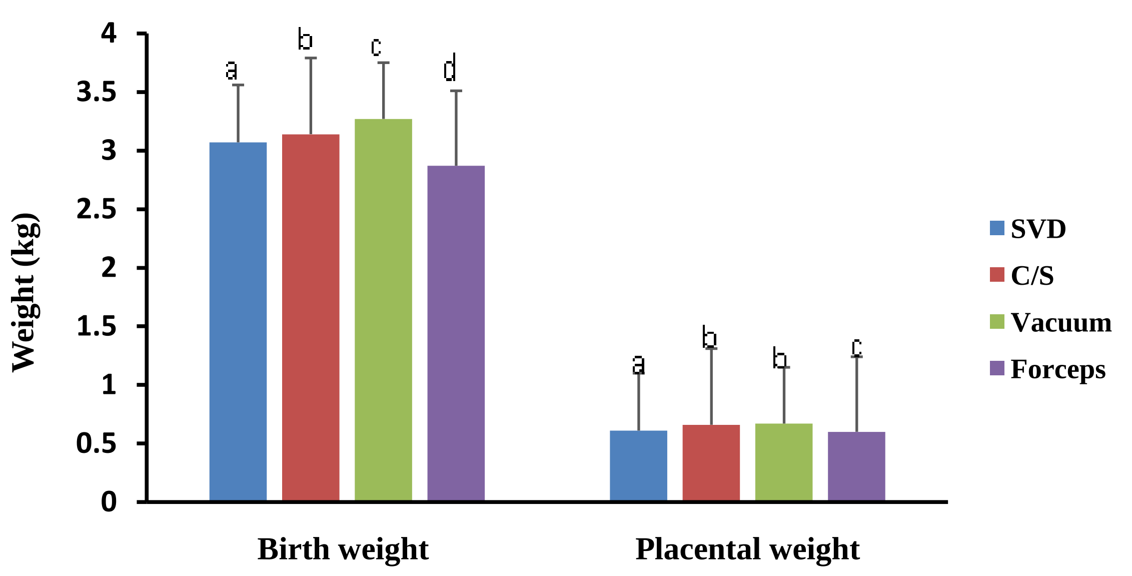

Figure 4.6: Birth and placental weights male and female newborns according to mode of delivery. One way analysis of variance indicates significant difference in birth and placental weights based on mode of delivery, with F = 24.34 and F = 47.27, for birth weight and placental weight respectively. The birth weight of newborns in vacuum delivery are significantly higher than SVD, C/S and forceps deliveries (a vs b vs c vs d $P< 0.001$). Also, the placental weight of newborns in caesarian and vacuum deliveries are significantly higher than SVD and forceps deliveries (a vs b vs c $p<0.001$). Mode of delivery with different superscript are significantly different with $p< 0.001$.

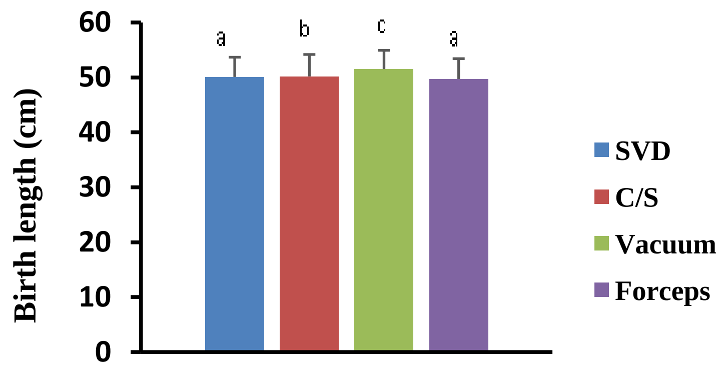

Figure 4.7: Birth length of male and female newborns according to mode of delivery. One way analysis of variance indicates significant difference in birth length based on mode of delivery, with F = 11.73. The birth length of newborns in vacuum delivery are significantly higher than SVD, C/S and forceps deliveries (a vs b vs c $P< 0.001$). Mode of delivery with different superscript are significantly different with $p< 0.001$.

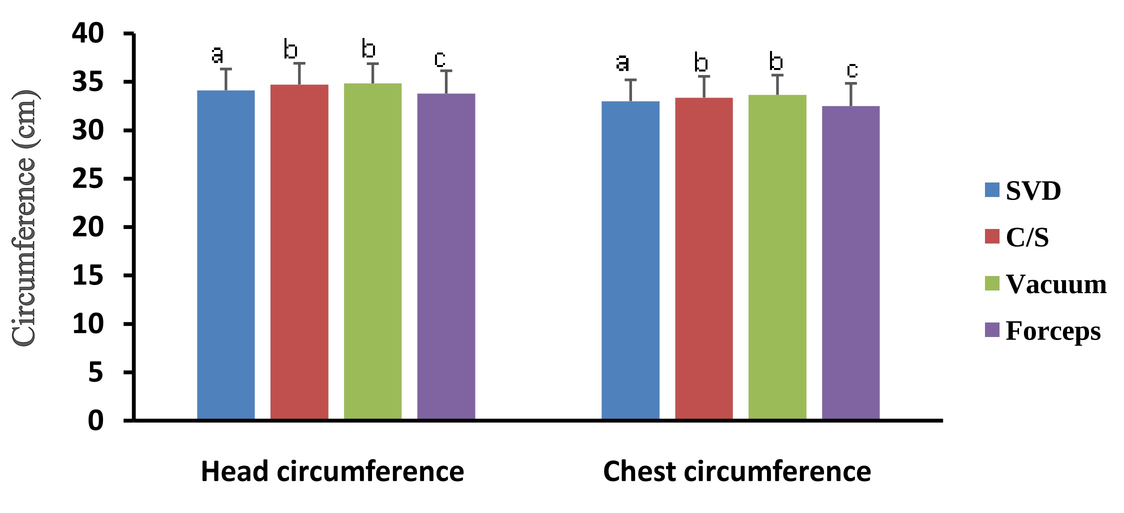

Figure 4.8: Head and chest circumferences of male and female newborns according to mode of delivery. One way analysis of variance indicates significant difference in head and chest circumferences based on mode of delivery, with F = 43.12 and F = 18.63, for head and chest circumferences, respectively. The head and chest circumference of newborns in vacuum and caesarian deliveries are significantly higher than SVD and forceps deliveries (a vs b vs c $P< 0.001$). Mode of delivery with different superscript are significantly different with $p< 0.001$.

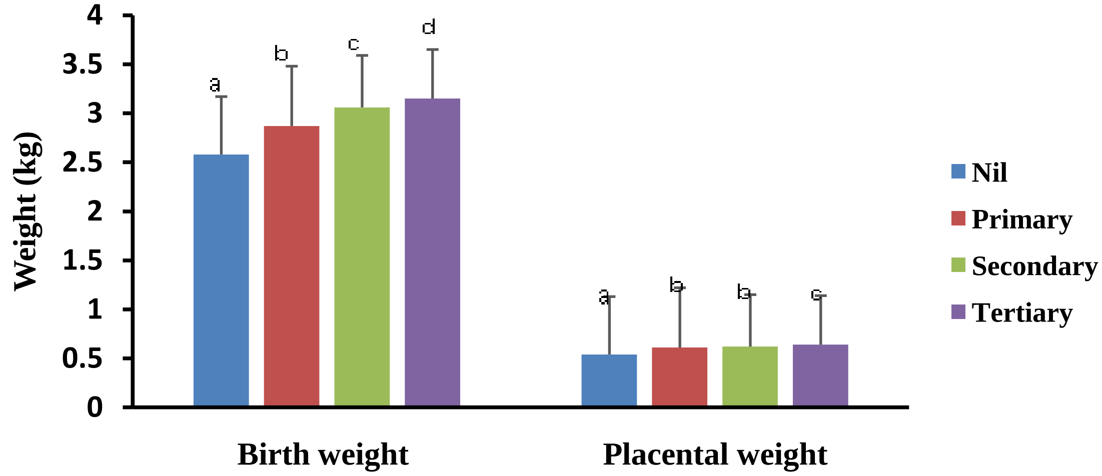

Figure 4.9: Birth and Placental weights of male and female newborns according to maternal level of education. One way analysis of variance indicates significant difference in birth and placental weight based on maternal level of education, with F = 31.13, and F = 14.50 for birth and placental weights respectively. The birth and placental weight of newborns in mothers with tertiary education are significantly higher than secondary, primary and nil (a vs b vs c vs d $P< 0.001$). Maternal level of education with different superscript are significantly different with $p< 0.001$.

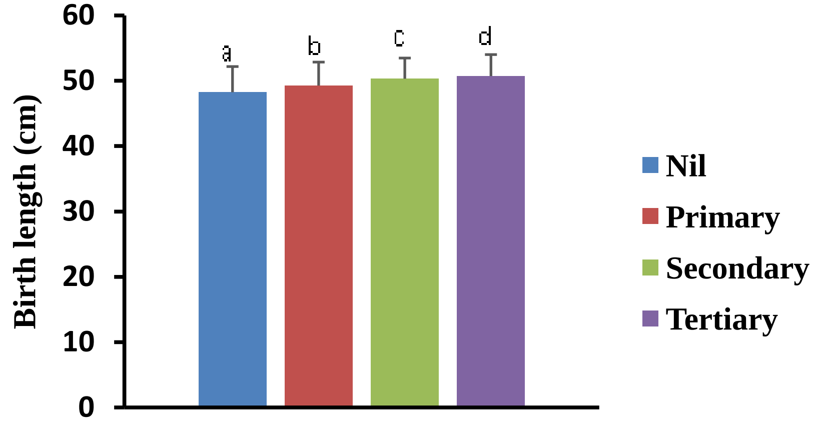

Figure 4.10: Birth length of male and female newborns according to maternal level of education. One way analysis of variance indicates significant difference in birth length based on maternal level of education, with F = 16.85. The birth length of newborns in mothers with tertiary education are significantly higher than mothers with secondary, primary and nil (a vs b vs c vs d $P < 0.001$). Maternal level of education with different superscript are significantly different with $p < 0.001$.

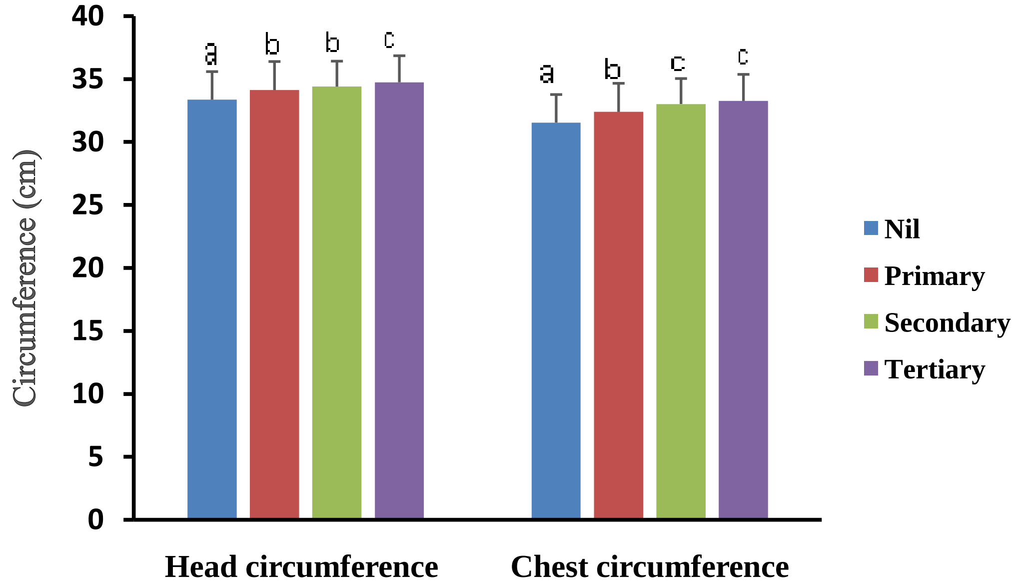

Figure 4.11: Head and chest circumferences of male and female newborns according to maternal level of education. One way analysis of variance indicates significant difference in head and chest circumference based on maternal level of education, with F = 12.42 and 15.66 respectively . The head circumference of newborns in mothers with tertiary education is significantly higher than mothers in secondary, primary, nil (a vs b vs c $p < 0.001$). Also, the chest circumference of newborns in mothers with tertiary and secondary education are significantly higher than mothers in primary and nil (a vs b vs c $p < 0.001$). Maternal level of education with different superscript are significantly different with $p < 0.001$.

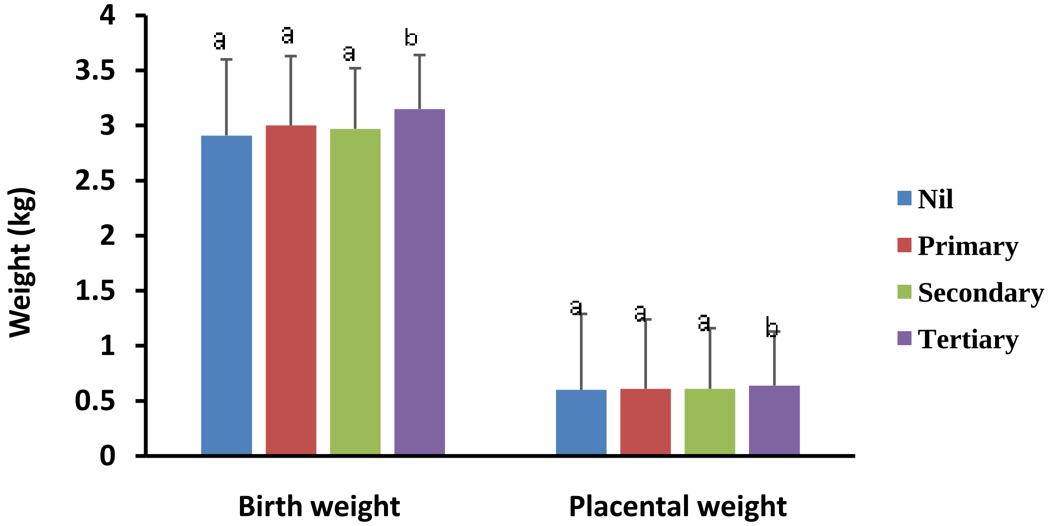

Figure 4.12: Birth and Placental weights of male and female newborns according to paternal level of education. One way analysis of variance indicates significant difference in birth and placental weight based on paternal level of education, with F = 23.71, and F = 12.89 for birth and placental weights respectively. The birth and placental weight of newborns in fathers with tertiary education are significantly higher than secondary, primary and nil (a vs b $P < 0.001$). Paternal level of education with different superscript are significantly different with $p < 0.001$.

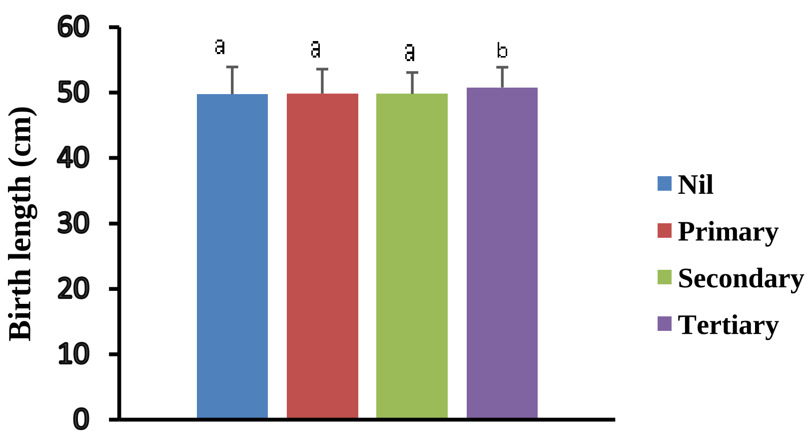

Figure 4.13: Birth length of male and female newborns according to paternal level of education. One way analysis of variance indicates significant difference in birth length based on paternal level of education, with F = 14.85. The birth length of newborns in fathers with tertiary education are significantly higher than fathers with secondary, primary and nil (a vs b $P < 0.001$). Paternal level of education with different superscript are significantly different with $p < 0.001$.

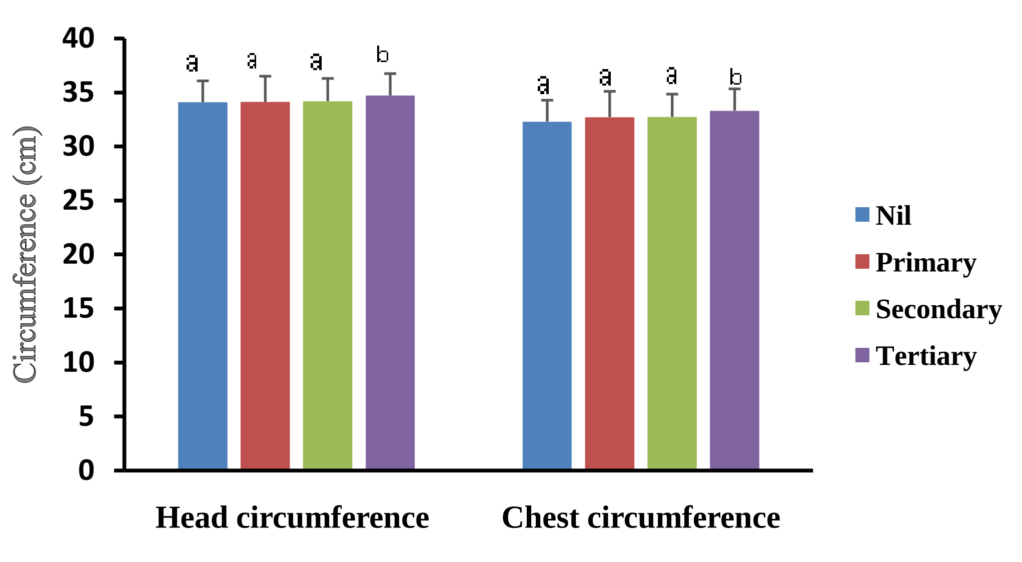

Figure 4.14: Head and chest circumferences of male and female newborns according to paternal level of education. One way analysis of variance indicates significant difference in head and chest circumference based on paternal level of education, with F = 10.49 and 14.10 for head and chest circumferences respectively. The head and chest circumference of newborns in fathers with tertiary education is significantly higher than mothers in secondary, primary, nil (a vs b p < 0.001). Paternal level of education with different superscript are significantly different with *p*< 0.001.

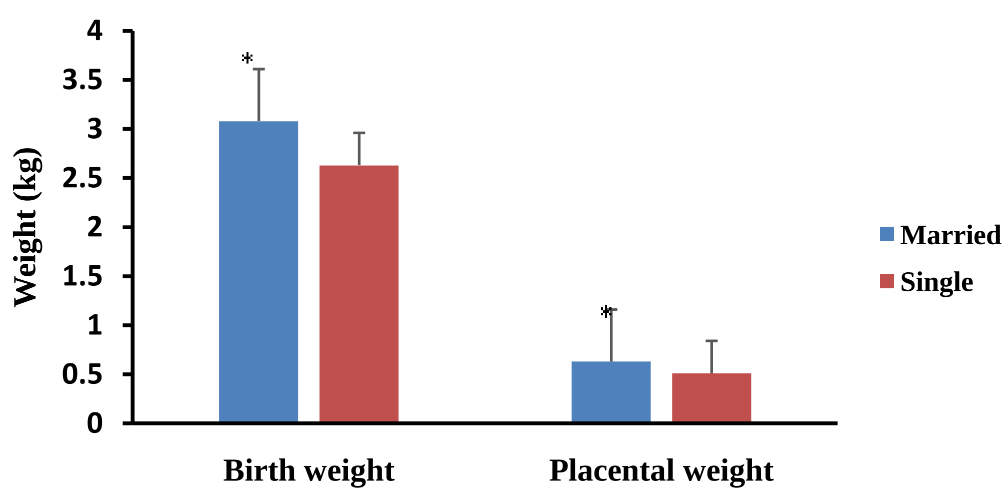

Figure 4.15: Birth and placental weightsof male and female newborns according to maternal marital status. Birth and placental weight of newborns in married mothers are higher than single mothers. Student t-test indicates significant difference in birth and placental weight of newborns of married and single mothers, (t = 4.68 and t = 4.79, p < 0.001).

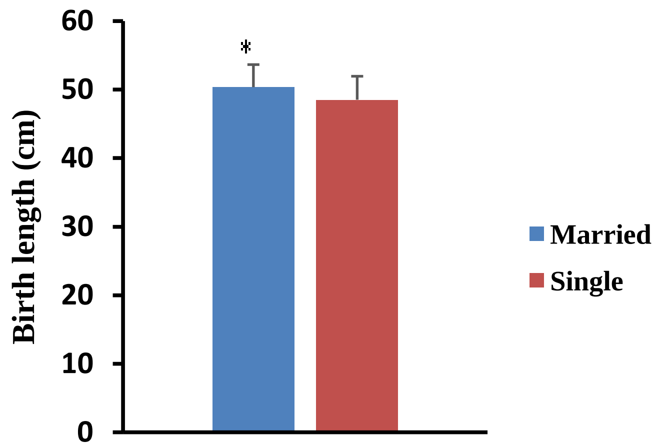

Figure 4.16: Birth length of male and female newborns according to maternal marital status. Birth length of newborns in married mothers are higher than single mothers. Student t-test indicates significant difference between the birth length of newborns in married and single mothers, ($t = 3.13$, $p < 0.01$).

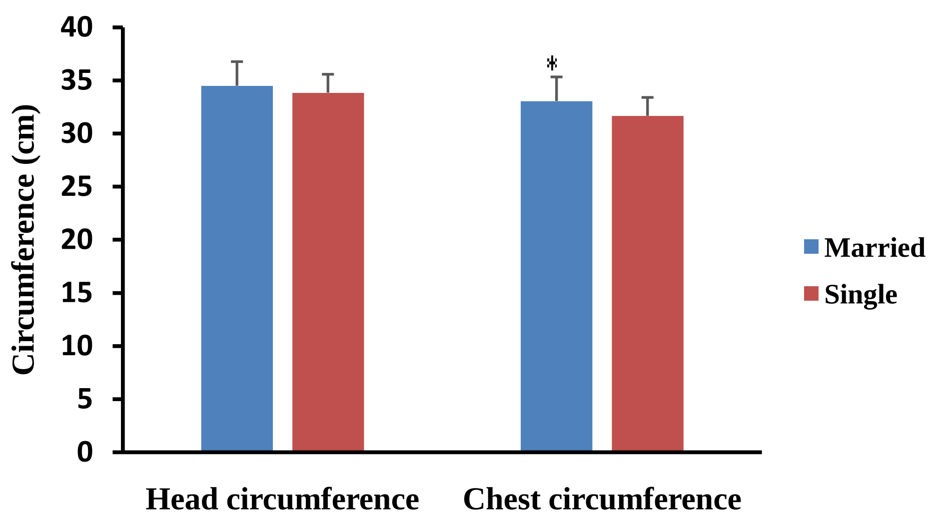

Figure 4.17: Head and chest circumference of male and female newborns according to maternal marital status. Student t-test indicates no significant difference between the head circumference of newborns in married and single mothers, ($t = 1.73$, $p > 0.05$). However, the chest circumference of newborns in married mothers are significantly higher than single mothers, ($t = 3.46$, $p < 0.001$).

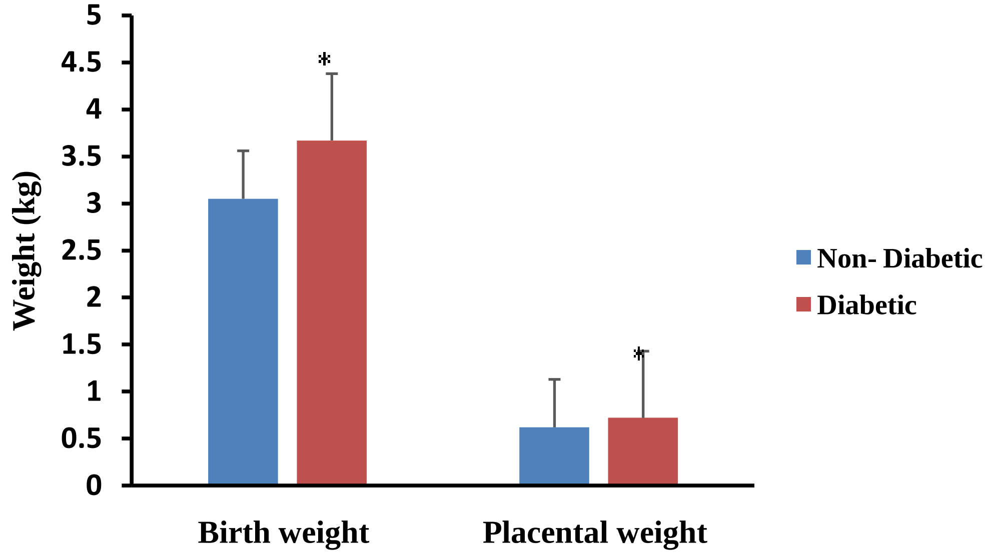

Figure 4.18: Birth and placental weights of male and female newborns according to maternal diabetes status. Birth and placental weight of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference in birth and placental weight of newborns of diabetic and non-diabetic mothers, ($t = -10.37$ and $t = -6.22$, $p < 0.001$).

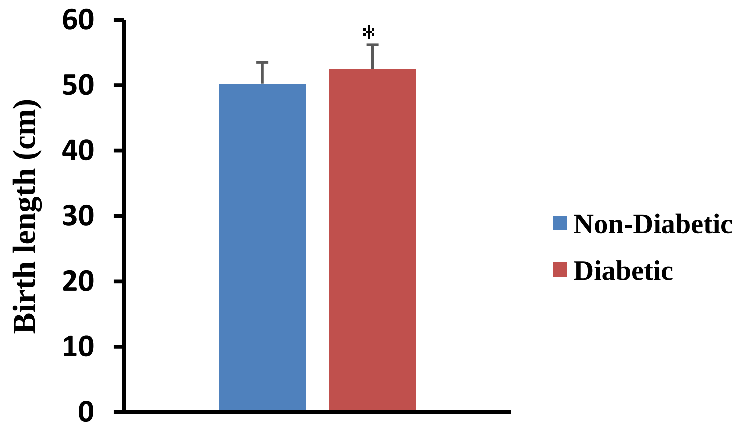

Figure 4.19: Birth length of male and female newborns according to maternal diabetes status. Birth length of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference between the birth length of newborns in diabetic and non-diabetic mothers, ($t = -5.98$, $p < 0.001$).

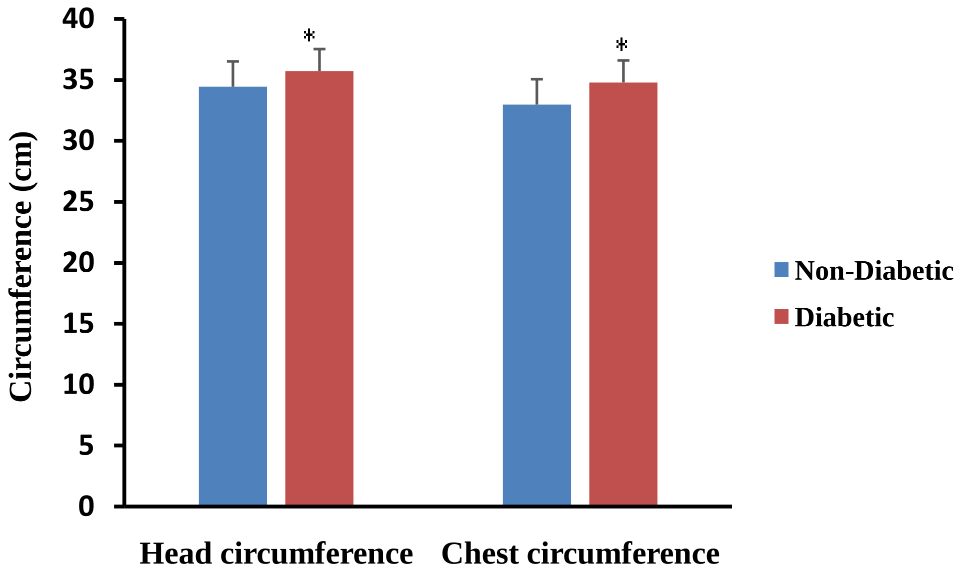

Figure 4.20: Head and chest circumferences of male and female newborns according to maternal diabetes status. Head and chest circumference of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in diabetic and non-diabetic mothers, ($t = -5.37$ and -7.04, $p < 0.001$).

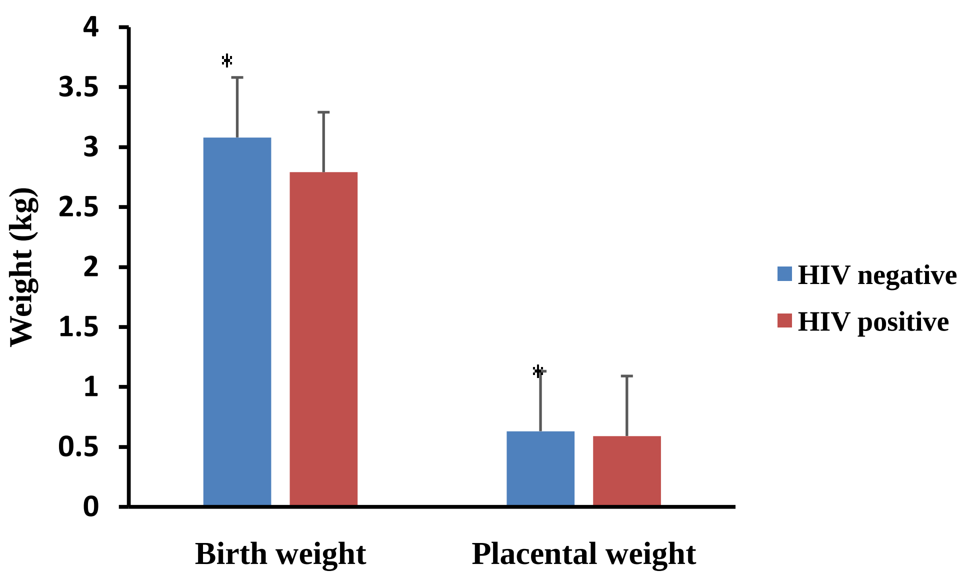

Figure 4.21: Birth and placental weights of male and female newborns according to maternal HIV status. Birth and placental weight of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between birth and placental weight of newborns in HIV negative and HIV positive mothers, ($t = 5.10$ and 3.06, $p < 0.001$).

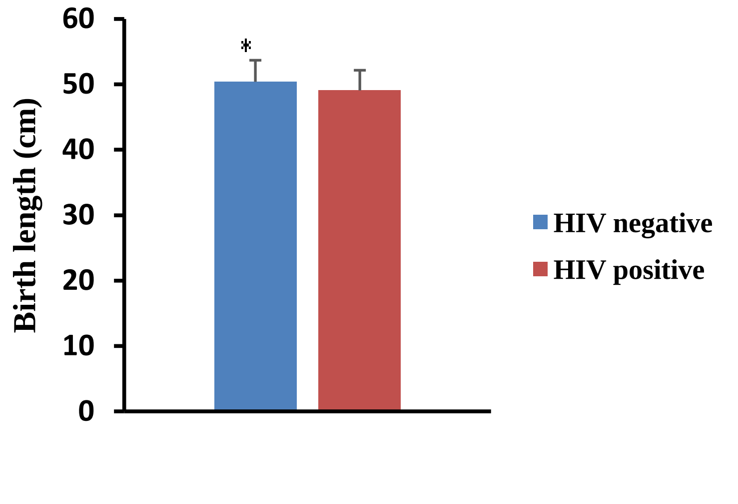

Figure 4.22: Birth length of male and female newborns according to maternal HIV status. Birth length of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between the birth length of newborns in HIV negative and HIV positive mothers, ($t = 3.86$, $p < 0.001$).

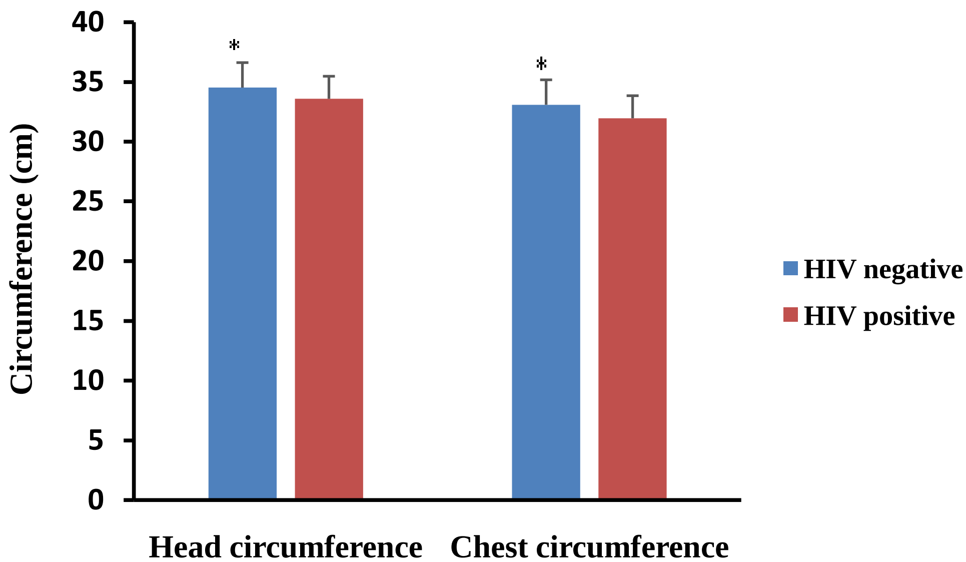

Figure 4.23: Head and chest circumferences of male and female newborns according to maternal HIV status. Head and chest circumference of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in HIV negative and HIV positive mothers, ($t = 4.31$ and 4.81, $p < 0.001$).

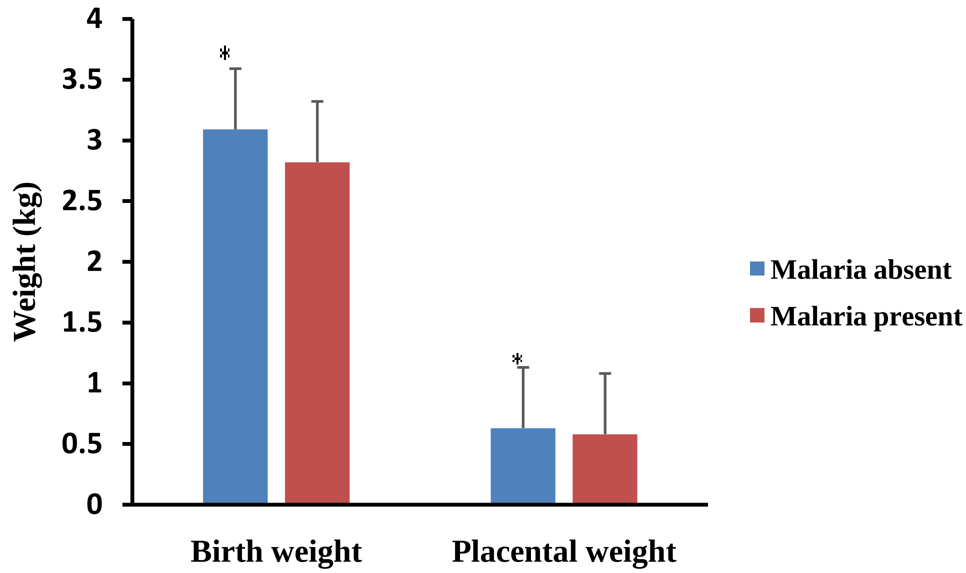

Figure 4.24: Birth and placental weights of male and female newborns according to maternal malarial episode. Birth and placental weight of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between birth and placental weight of newborns in malaria absent and malaria present mothers, ($t = 7.89$ and 5.75, $p < 0.001$).

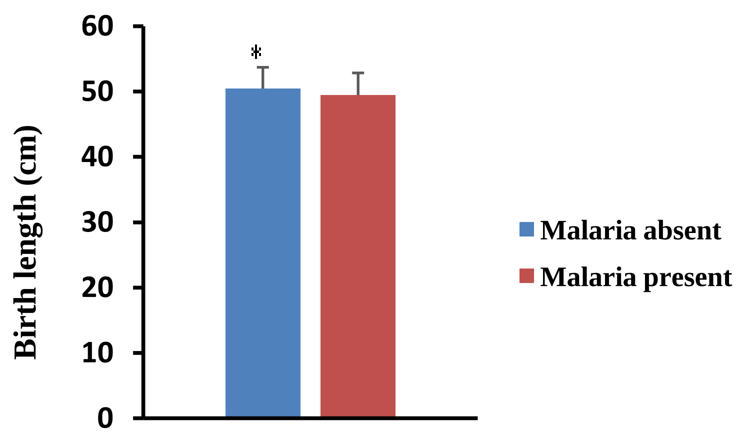

Figure 4.25: Birth length of male and female newborns according to maternal malarial episode. Birth length of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between the birth length of newborns in malaria absent and malaria present mothers, ($t = 4.48$, $p < 0.001$).

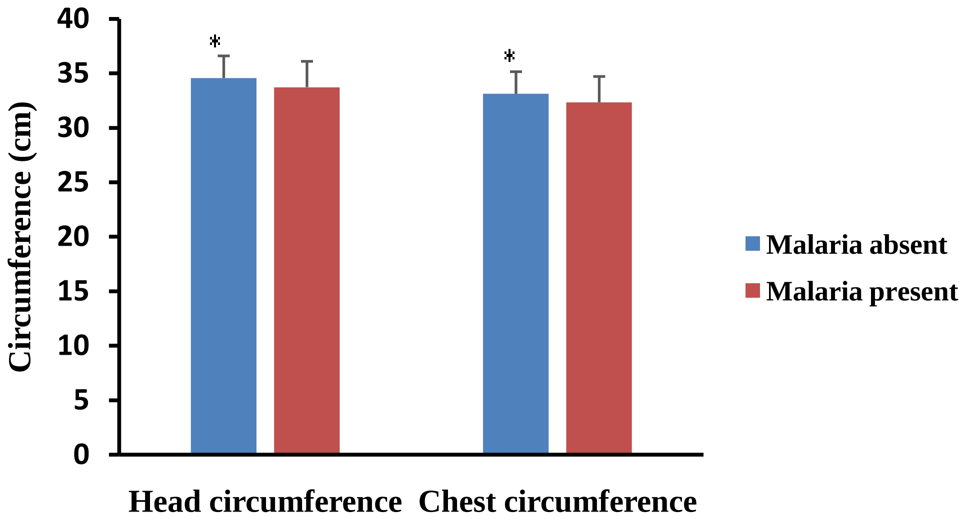

Figure 4.26: Head and chest circumferences of male and female newborns according to maternal malaria episode. Head and chest circumference of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in malaria absent and malaria present mothers, ($t = 6.07$ and 5.14, $p < 0.001$).

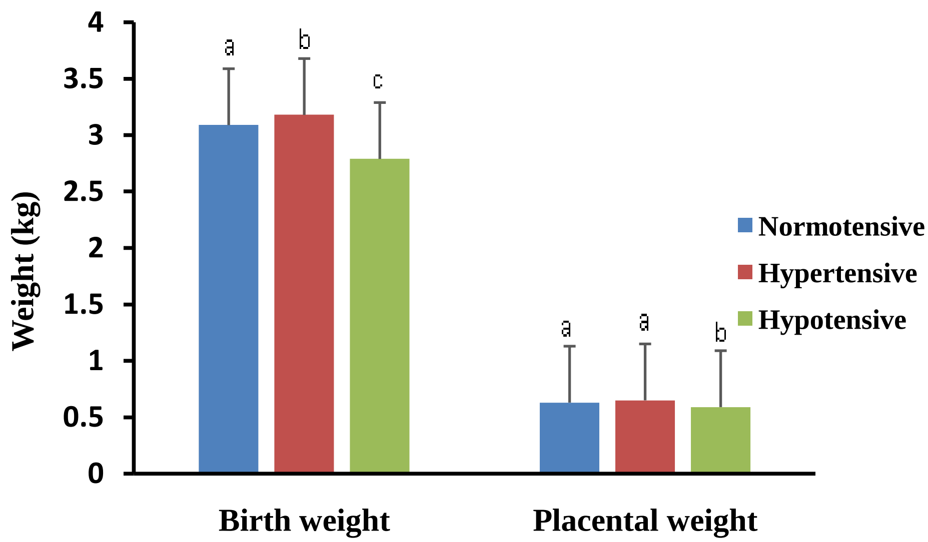

Figure 4.27: Birth and placental weights of male and female newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in birth and placental weight based on maternal blood pressure, with $F = 39.99$, and $F = 14.36$ for birth and placental weights respectively. The birth and placental weight of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b vs c $P< 0.001$). Maternal blood pressure with different superscript are significantly different with $p< 0.001$.

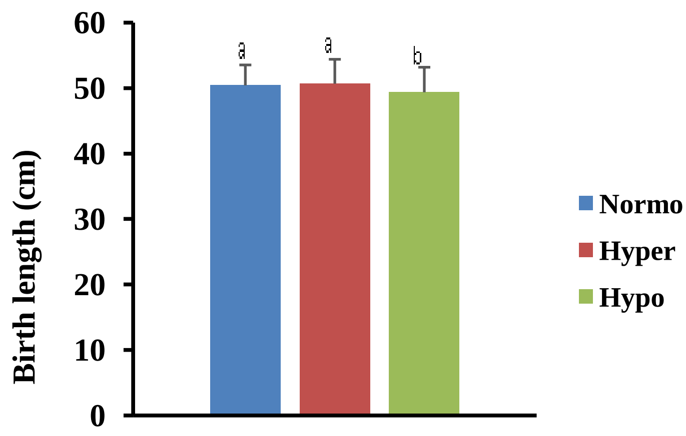

Figure 4.28: Birth length of male and female newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in birth length based on maternal blood pressure, with F = 12.53. The birth length of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b p<0.001). Maternal blood pressure with different superscript are significantly different with *p*< 0.001.

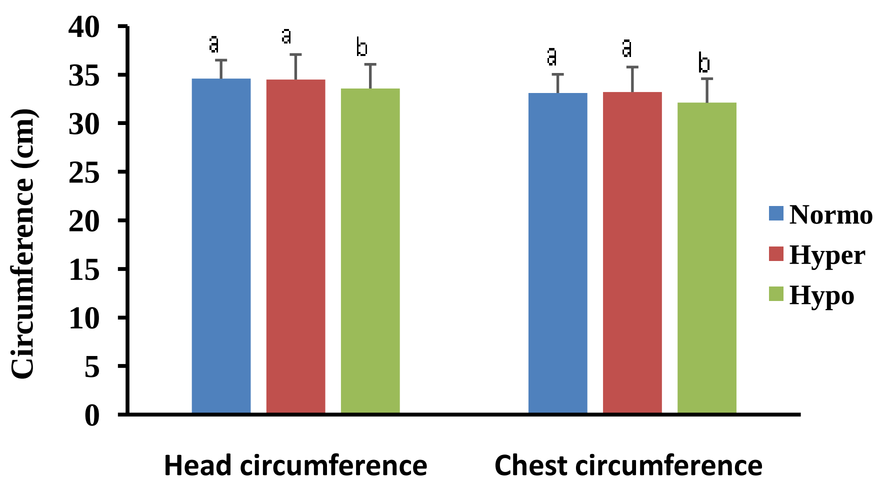

Figure 4.29: Head and chest circumferences of male and female newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in head and chest circumference based on maternal blood pressure, with F = 24.91 and 22.95, for head and chest circumference respectively. The head and chest circumference of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b p<0.001). Maternal blood pressure with different superscript are significantly different with *p*< 0.001.

Male newborns only

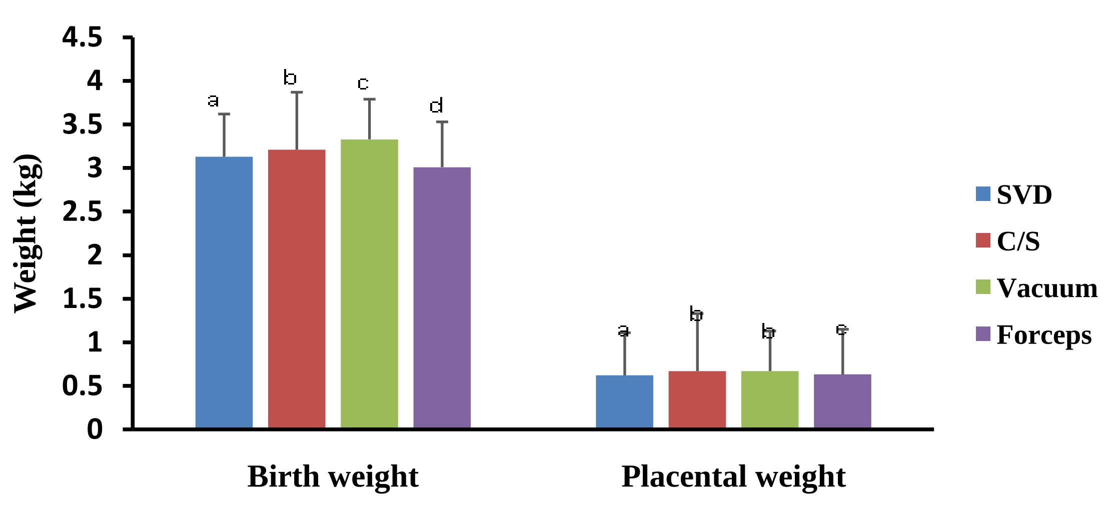

Figure 4.30: Birth and placental weights of male newborns according to mode of delivery. One way analysis of variance indicates significant difference in birth and placental weight based on mode of delivery, with F = 15.73, and F = 28.19 for birth and placental weights respectively. The birth weight of newborns in vacuum delivery are significantly higher than SVD, C/S and forceps deliveries (a vs b vs c vs d *P*< 0.001). Also, the placental weight of newborns in caesarian and vacuum deliveries are significantly higher than SVD and forceps deliveries (a vs b vs c p<0.001). Mode of delivery with different superscript are significantly different with *p*< 0.001.

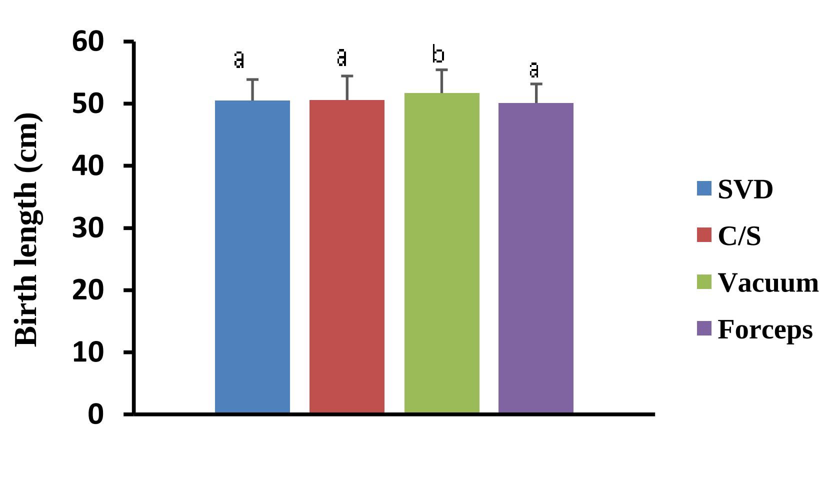

Figure 4.31: Birth length of male newborns according to mode of delivery. One way analysis of variance indicates significant difference in birth length based on mode of delivery, with F = 5.46. The birth length of newborns in vacuum delivery are significantly higher than SVD, C/S and forceps deliveries (a vs b *P*< 0.001). Mode of delivery with different superscript are significantly different with *p*< 0.001.

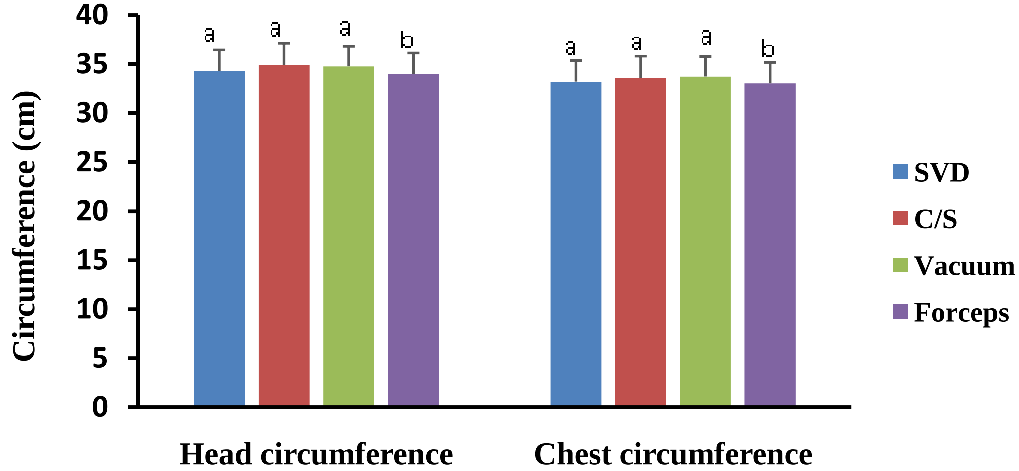

Figure 4.32: Head and chest circumferences of male newborns according to mode of delivery. One way analysis of variance indicates significant difference in head and chest circumference based on mode of delivery, with F = 21.81 and 9.73, head and chest circumference respectively. The head and chest circumference of newborns in SVD, C/S and vacuum deliveries are significantly higher than forceps deliveries (a vs b $P<0.001$). Mode of delivery with different superscript are significantly different with $p< 0.001$.

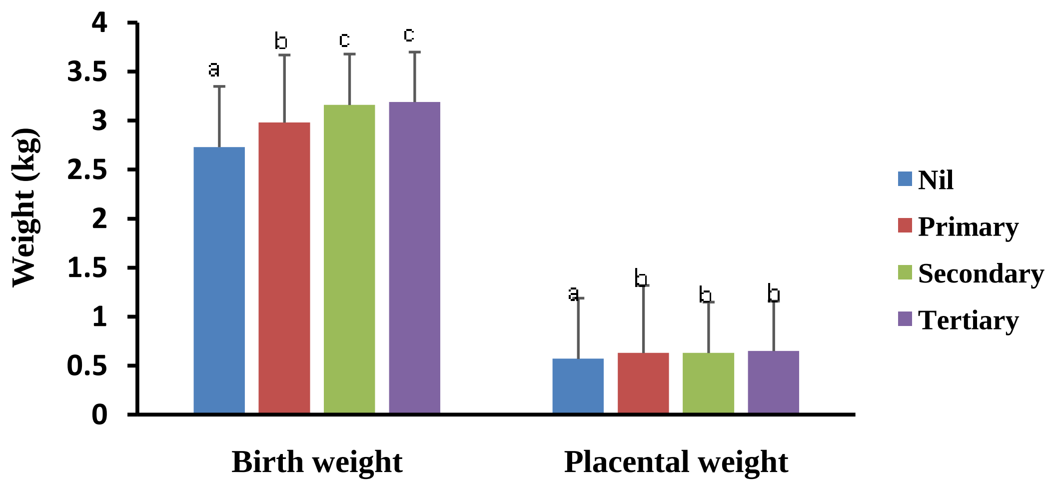

Figure 4.33: Birth and placental weights of male newborns according to maternal level of education. One way analysis of variance indicates significant difference in birth and placental weight based on maternal level of education, with F = 11.47, and F = 5.34, for birth and placental weights respectively. The birth weight of newborns in mothers with secondary and tertiary education are significantly higher than mothers with primary and nil (a vs b vs c $P< 0.001$). Also, the placental weight of newborns in mothers with primary, secondary and tertiary are significantly higher than mothers with no formal education (a vs b $p<0.01$). Maternal level of education with different superscript are significantly different with $p< 0.01$.

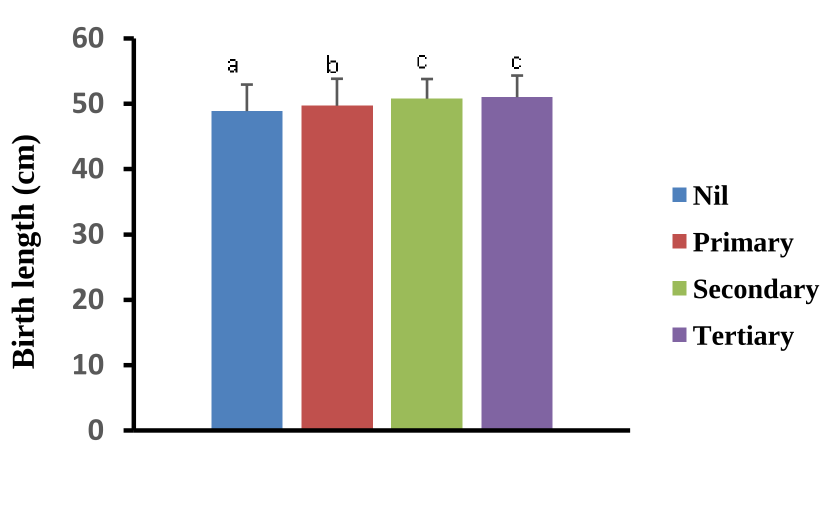

Figure 4.34: Birth length of male newborns according to maternal level of education. One way analysis of variance indicates significant difference in birth length based on maternal level of education, with F = 7.66. The birth length of newborns in mothers with secondary and tertiary education are significantly higher than mothers with primary and nil (a vs b vs c *P*< 0.001). Maternal level of education with different superscript are significantly different with *p*< 0.001.

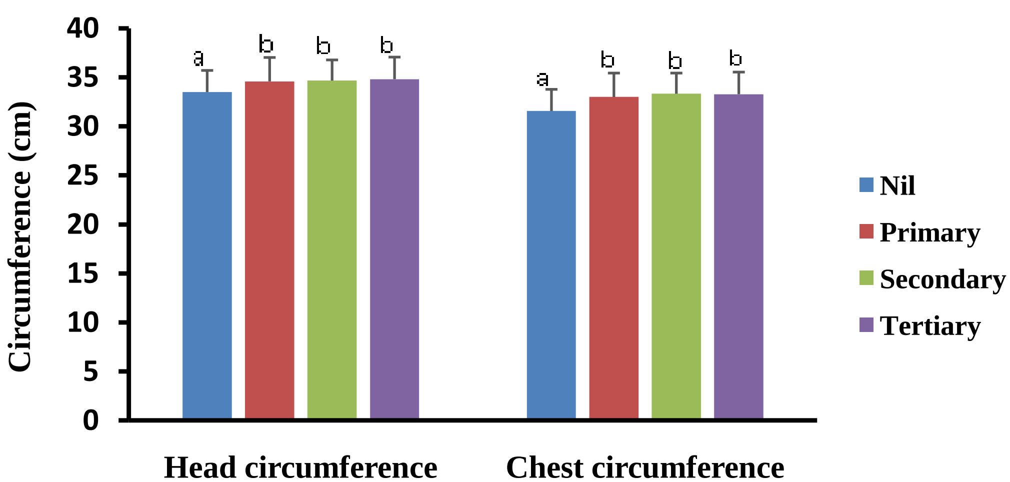

Figure 4.35: Head and chest circumferences of male newborns according to maternal level of education. One way analysis of variance indicates significant difference in head and chest circumference based on maternal level of education, with F = 4.64, and F = 8.32, for head and chest circumference respectively. The head and chest circumference of newborns in mothers with primary, secondary and tertiary education are significantly higher than mothers with no formal education (a vs b *P*< 0.001). Maternal level of education with different superscript are significantly different with *p*< 0.001.

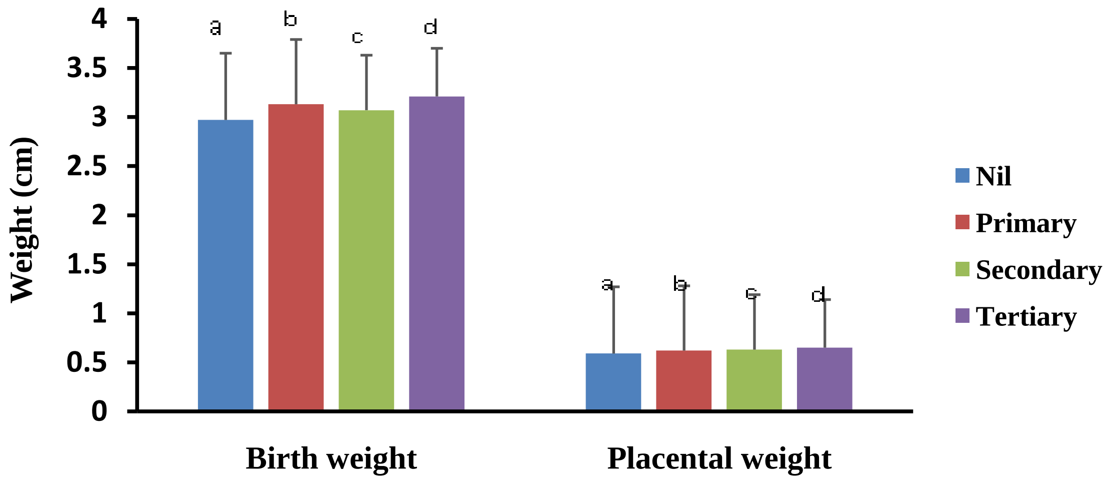

Figure 4.36: Birth and placental weights of male newborns according to paternal level of education. One way analysis of variance indicates significant difference in birth and placental weight based on paternal level of education, with F = 8.31, and F = 6.09, for birth and placental weights respectively. The birth and placental weight of newborns in fathers with tertiary education are significantly higher than fathers with secondary, primary and nil (a vs b vs c vs d $P< 0.001$). Paternal level of education with different superscript are significantly different with $p< 0.001$.

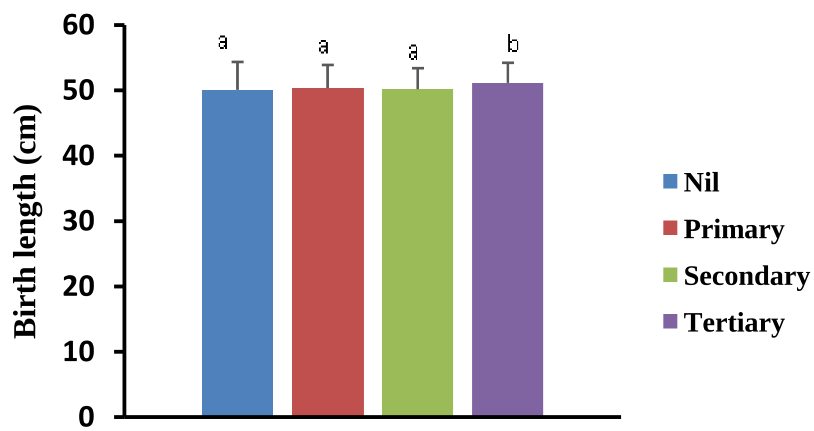

Figure 4.37: Birth length of male newborns according to paternal level of education. . One way analysis of variance indicates significant difference in birth length based on paternal level of education, with F = 7.58. The birth length of newborns in fathers with tertiary education are significantly higher than fathers with secondary primary and nil (a vs b $P< 0.001$). Paternal level of education with different superscript are significantly different with $p< 0.001$.

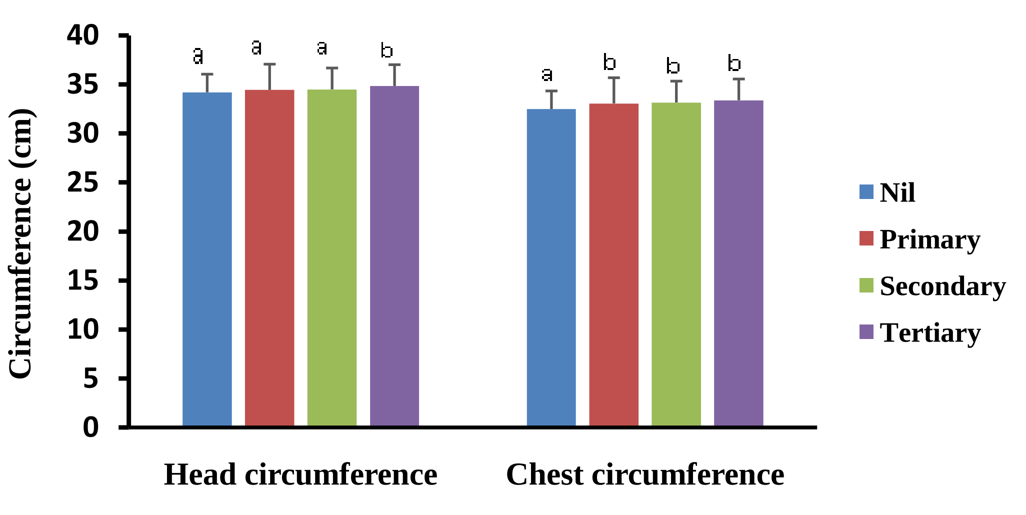

Figure 4.38: Head and chest circumferences of male newborns according to paternal level of education. One way analysis of variance indicates significant difference in head and chest circumference based on paternal level of education, with F = 2.97, and F = 4.48, for head and chest circumference respectively. The head circumference of newborns in fathers with tertiary education are significantly higher than fathers with secondary, primary and nil (a vs b $P < 0.05$). Also, the chest circumference of newborns in fathers with primary, secondary and tertiary education are significantly higher than in fathers with no formal education (a vs b $p<0.01$). Paternal level of education with different superscript are significantly different with $p < 0.05$.

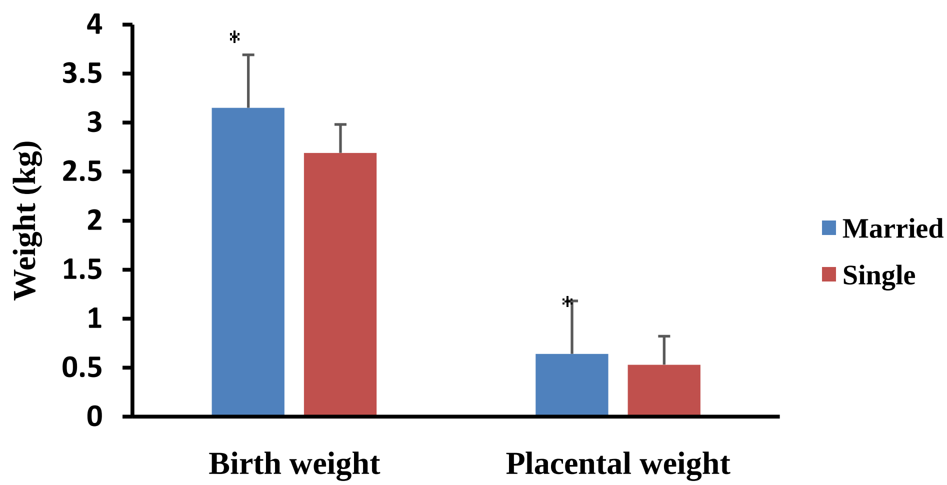

Figure 4.39: Birth and placental weights of male newborns according to maternal marital status. Birth and placental weight of newborns in married mothers are higher than single mothers. Student t-test indicates significant difference between birth and placental weight of newborns in married and single mothers, (t =3.63 and 3.36, $p < 0.001$).

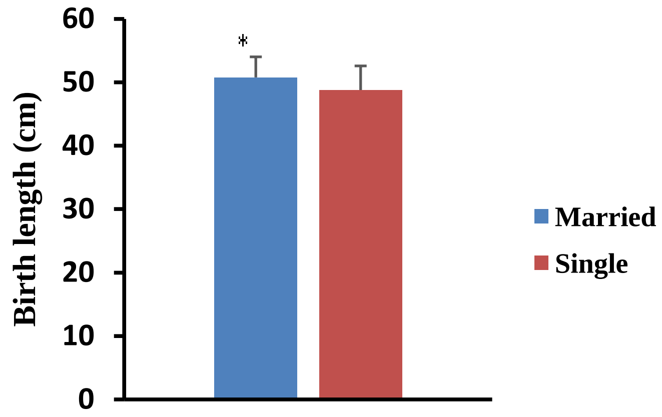

Figure 4.40: Birth length of male newborns according to maternal marital status. Birth length of newborns in married mothers are higher than single mothers. Student t-test indicates significant difference between the birth length of newborns in married and single mothers, ($t = 2.58$, $p < 0.05$).

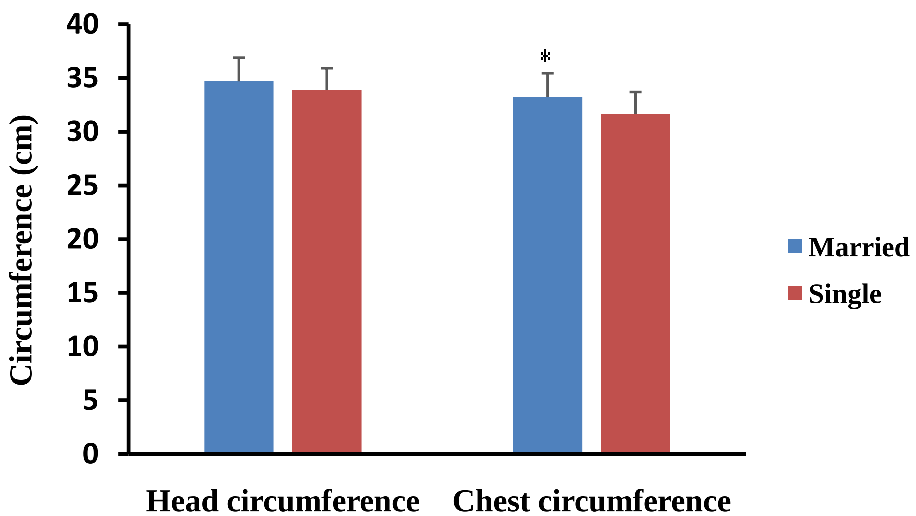

Figure 4.41: Head and chest circumferences of male newborns according to maternal marital status. Student t-test indicates no significant difference between the head circumference of newborns in married and single mothers, ($t = 1.54$, $p = 0.125$). However, the chest circumference of newborns in married mothers is significantly higher than in single mothers, ($t = 2.96$, $p < 0.01$).

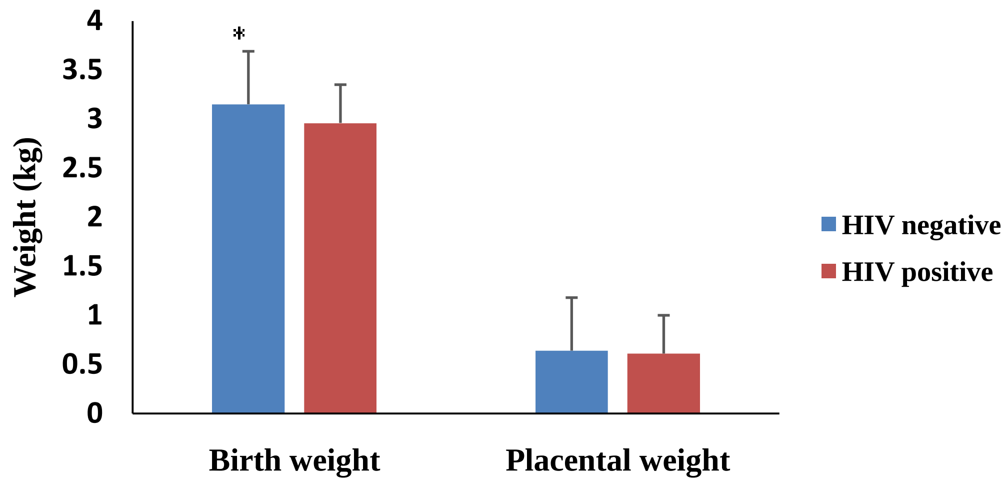

Figure 4.42: Birth and placental weights of male newborns according to maternal HIV status. Birth weight of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between birth weight of newborns in HIV negative and HIV positive mothers, ($t = 2.13$ $p < 0.05$). However, there is no significant difference in placental weight of newborns of HIV negative and HIV positive mothers ($t = 1.03$ $p = 0.30$).

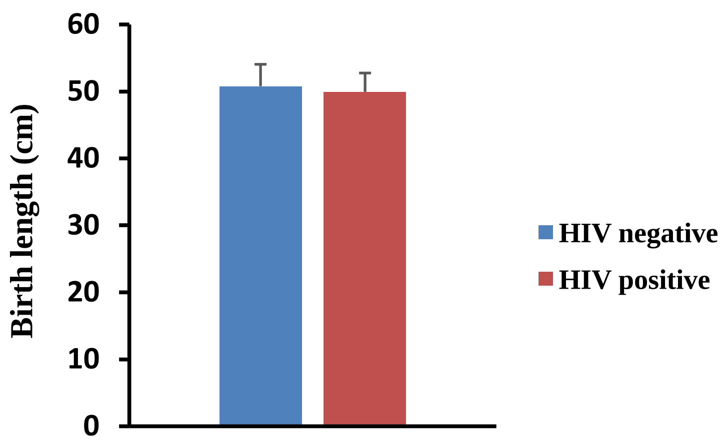

Figure 4.43: Birth length of male newborns according to maternal HIV status. Student t-test indicates no significant difference between the birth length of newborns in HIV negative and HIV positive mothers, ($t = 1.55$, $p = 0.12$).

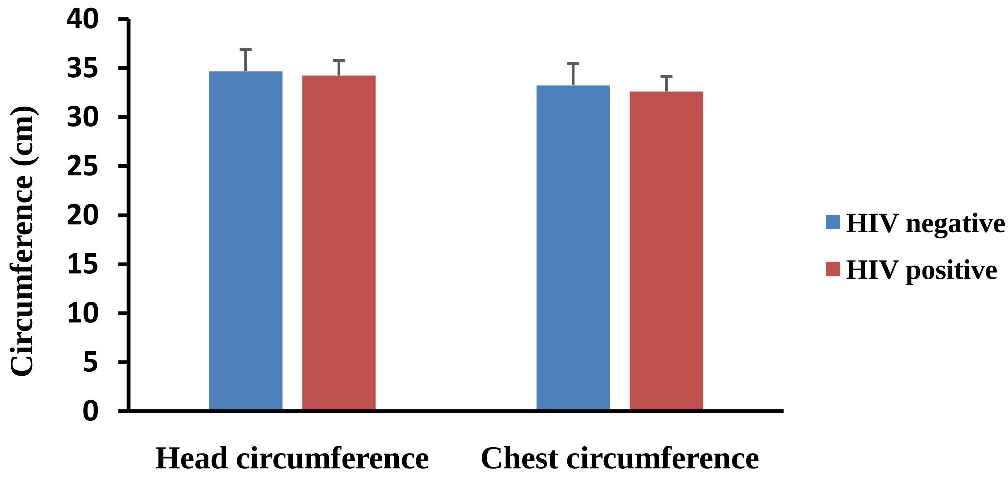

Figure 4.44: Head and chest circumferences of male newborns according to maternal HIV status. Student t-test indicates no significant difference between the head and chest circumferences of newborns in HIV negative and HIV positive mothers, ($t = 1.22$ and 1.66, $p > 0.05$).

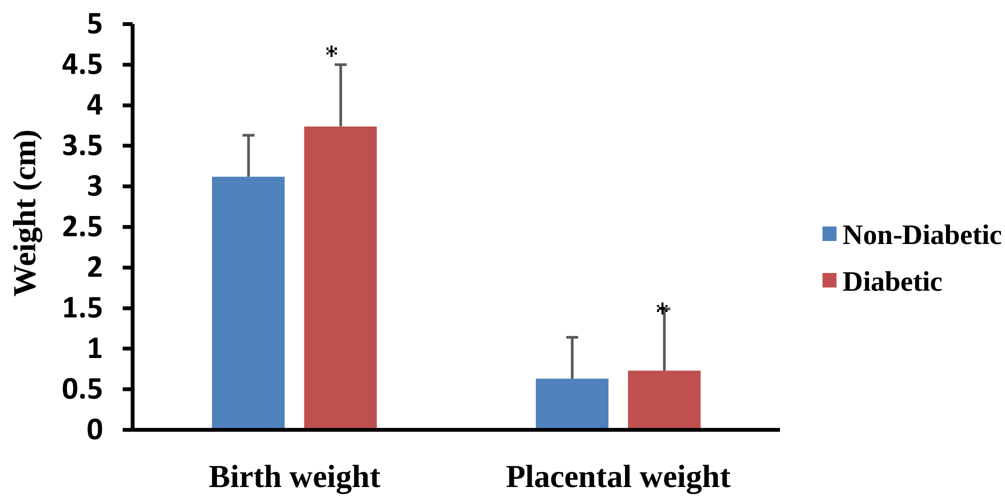

Figure 4.45: Birth and placental weights of male newborns according to maternal diabetes status. Birth and placental weight of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference in birth and placental weight of newborns of diabetic and non-diabetic mothers, ($t = -7.89$ and $t = -4.91$, $p < 0.001$).

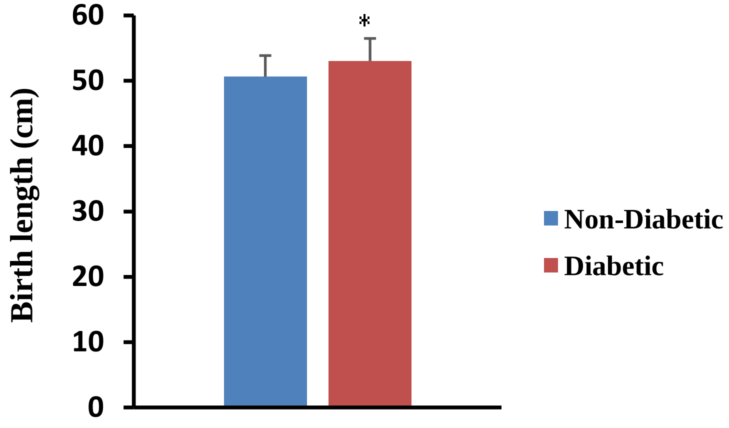

Figure 4.46: Birth length of male newborns according to maternal diabetes status. Birth length of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference between the birth length of newborns in diabetic and non-diabetic mothers, ($t = -4.91$, $p < 0.001$).

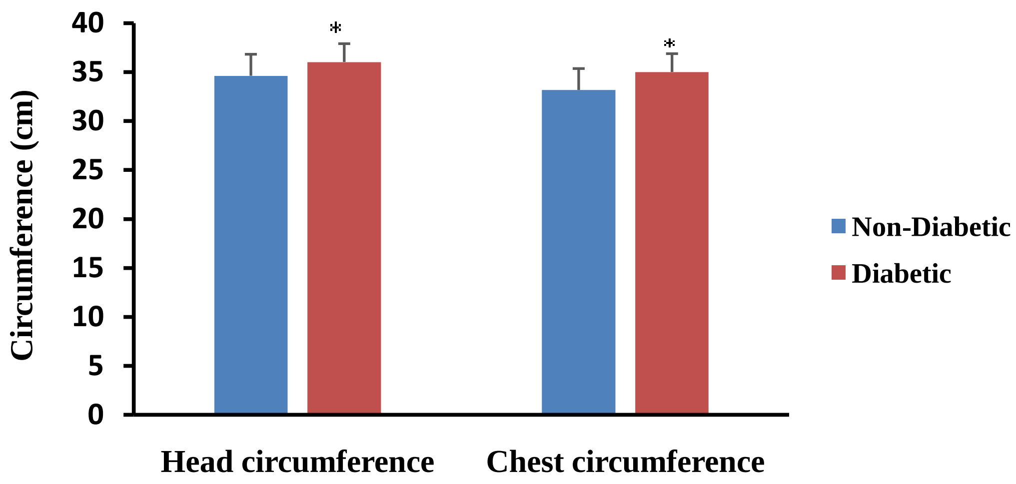

Figure 4.47: Head and chest circumferences of male newborns according to maternal diabetes status. Head and chest circumference of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in diabetic and non-diabetic mothers, ($t = -4.26$ and -5.49, $p < 0.001$).

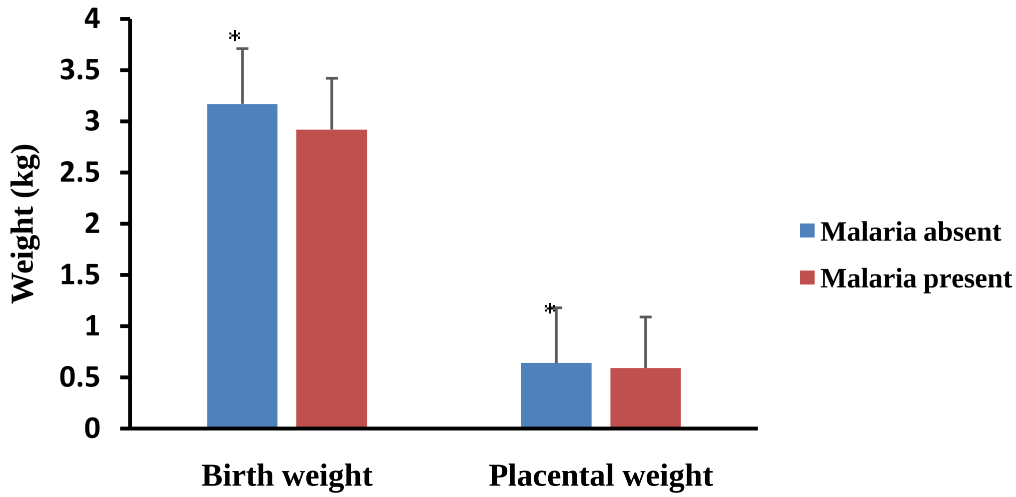

Figure 4.48: Birth and placental weights of male newborns according to maternal malarial episode. Birth and placental weight of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between birth and placental weight of newborns in malaria absent and malaria present mothers, ($t = 5.23$ and 3.95, $p < 0.001$).

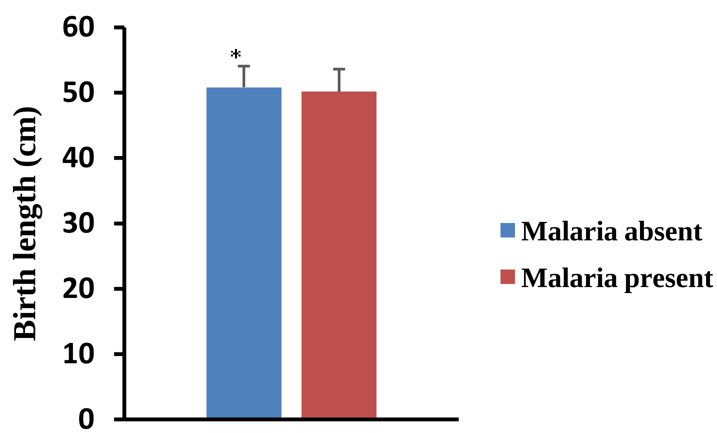

Figure 4.49: Birth length of male newborns according to maternal malarial episode. Birth length of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between the birth length of newborns in malaria absent and malaria present mothers, ($t = 2.17$, $p < 0.05$).

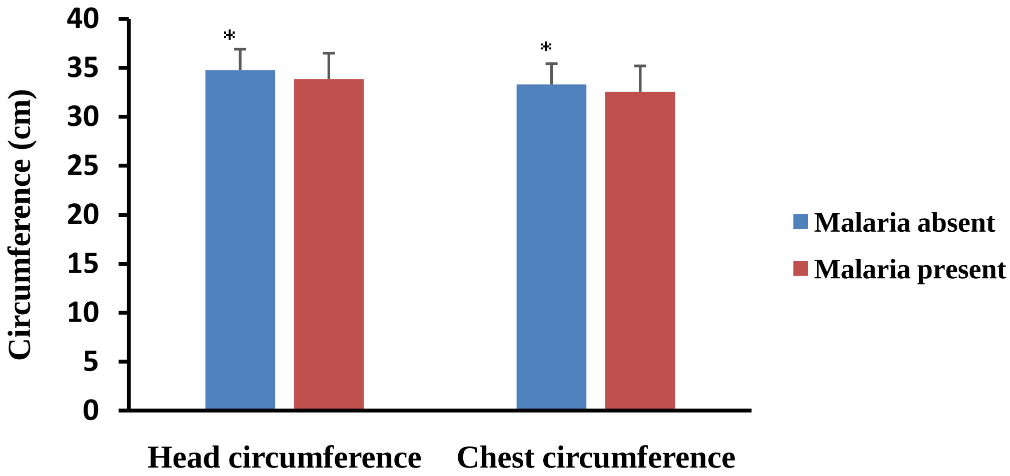

Figure 4.50: Head and chest circumferences of male newborns according to maternal malarial episode. Head and chest circumference of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in malaria absent and malaria present mothers, ($t = 4.62$ and 3.63, $p < 0.001$).

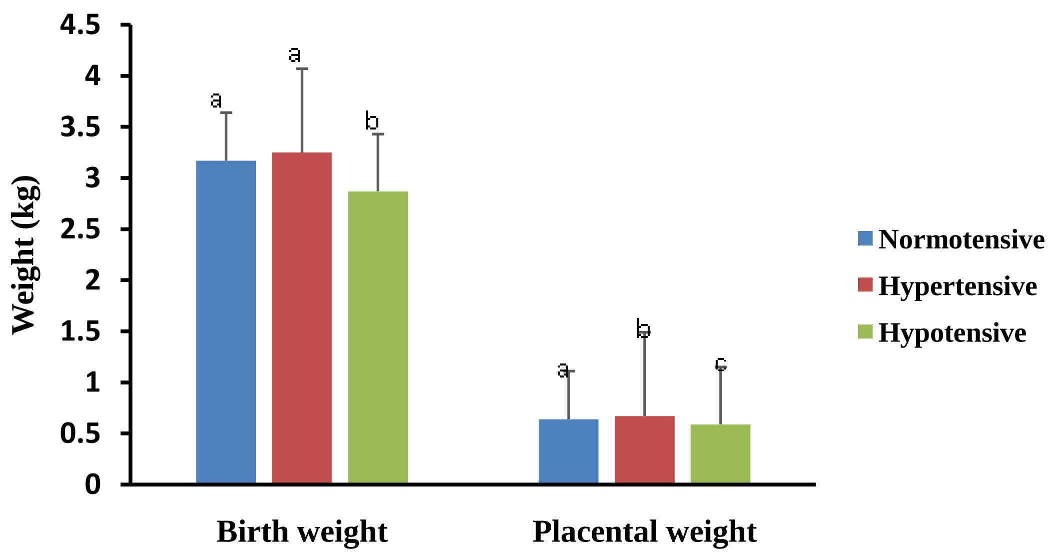

Figure 4.51: Birth and placental weights of male newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in birth and placental weight based on maternal blood pressure, with $F = 19.71$, and $F = 9.14$ for birth and placental weights respectively. The birth weight of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b $P < 0.001$). Also, the placental weight of newborns in hypertensive mothers are significantly higher than normal and hypotensive mothers (a vs b vs c $p<0.001$). Maternal blood pressure with different superscript are significantly different with $p < 0.001$.

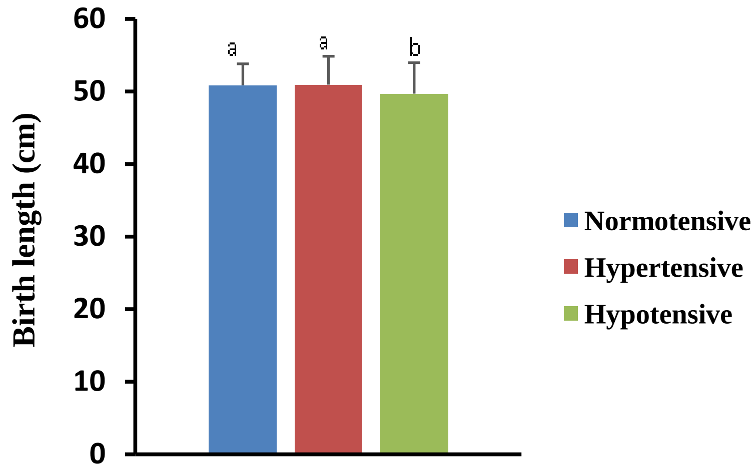

Figure 4.52: Birth length of male newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in birth length based on maternal blood pressure, with F = 6.98. The birth length of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b $p<0.001$). Maternal blood pressure with different superscript are significantly different with $p< 0.001$.

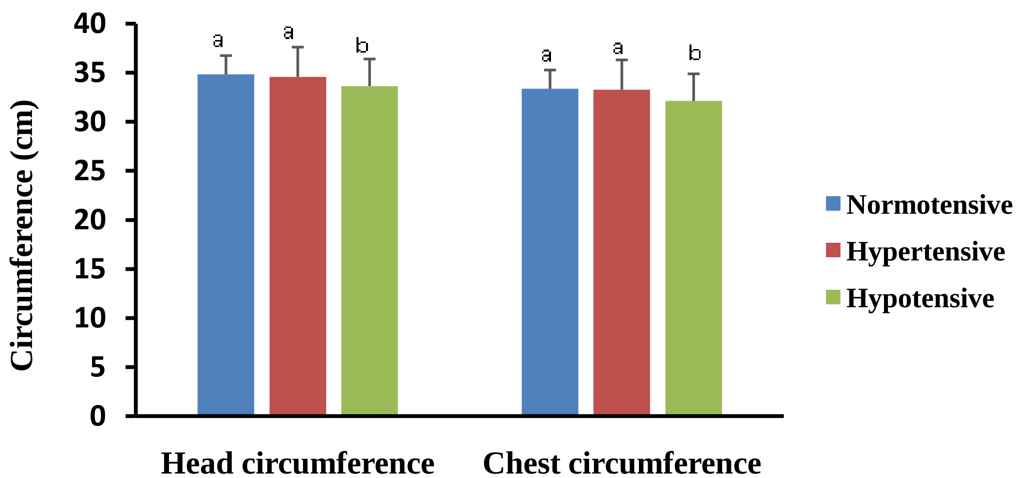

Figure 4.53: Head and chest circumferences of male newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in head and chest circumference based on maternal blood pressure, with F = 15.96 and 15.99, for head and chest circumference respectively. The head and chest circumference of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b $p<0.001$). Maternal blood pressure with different superscript are significantly different with $p< 0.001$

Female newborns only

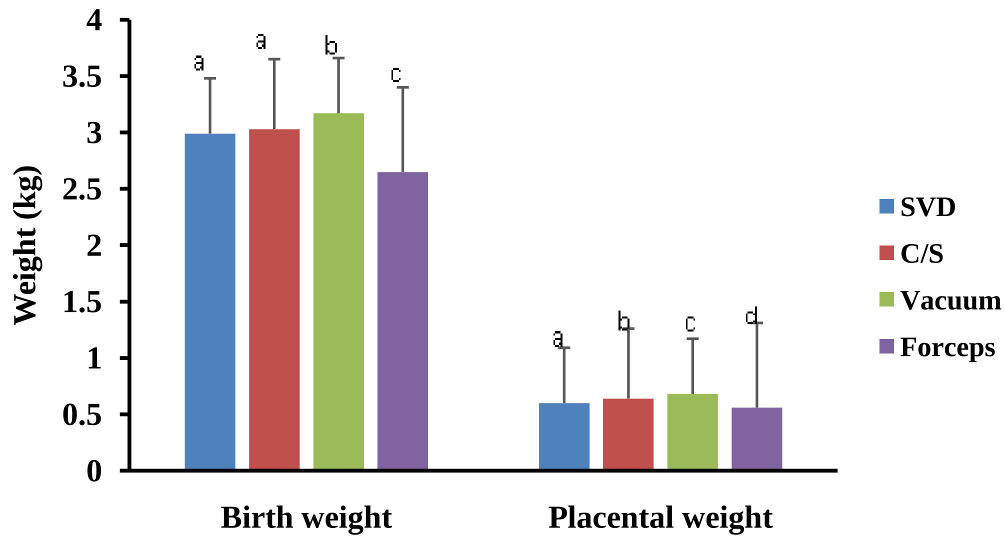

Figure 4.54: Birth and placental weights of female newborns according to mode of delivery. One way analysis of variance indicates significant difference in birth and placental weight based on mode of delivery, with F = 8.33, and F = 19.05 for birth and placental weights respectively. The birth and placental weight of newborns in vacuum delivery are significantly higher than SVD, C/S and forceps deliveries (a vs b vs c vs d $P< 0.001$). Mode of delivery with different superscript are significantly different with $p< 0.001$.

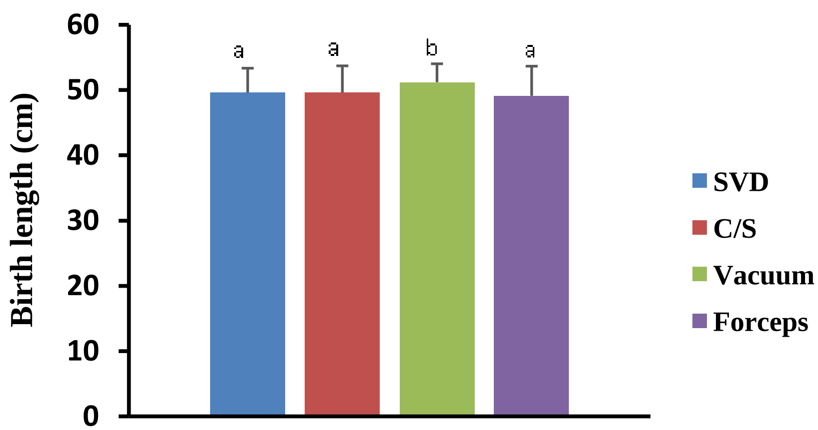

Figure 4.55: Birth length of female newborns according to mode of delivery. One way analysis of variance indicates significant difference in birth length based on mode of delivery, with F = 5.51. The birth length of newborns in vacuum delivery are significantly higher than SVD, C/S and forceps deliveries (a vs b $P< 0.001$). Mode of delivery with different superscript are significantly different with $p< 0.001$.

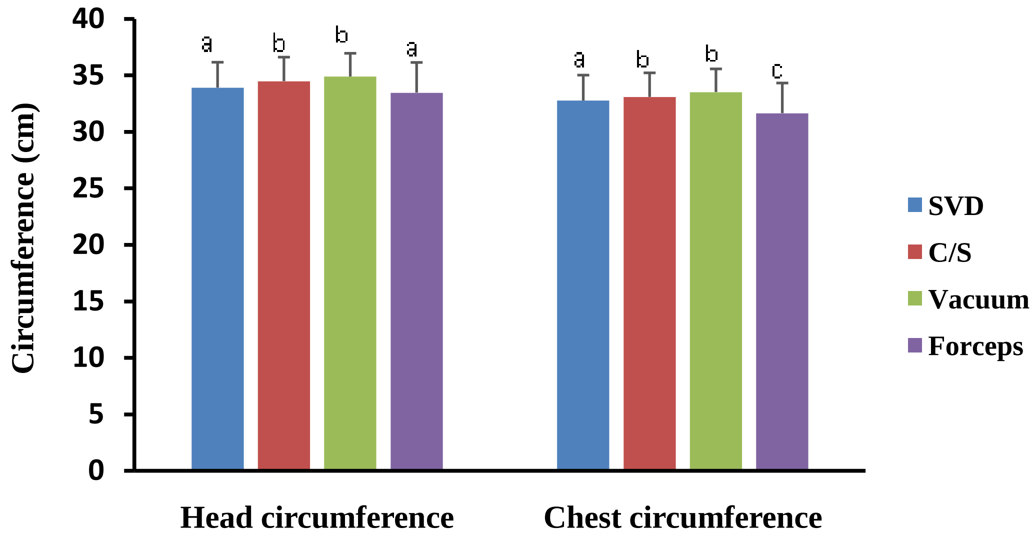

Figure 4.56: Head and chest circumferences of female newborns according to mode of delivery. One way analysis of variance indicates significant difference in head and chest circumference based on mode of delivery, with F = 19.89 and 8.02, head and chest circumference respectively. The head and chest circumference of newborns in vacuum and caesarian deliveries are significantly higher than SVD and forceps deliveries (a vs b vs c $P < 0.001$). Mode of delivery with different superscript are significantly different with $p < 0.001$.

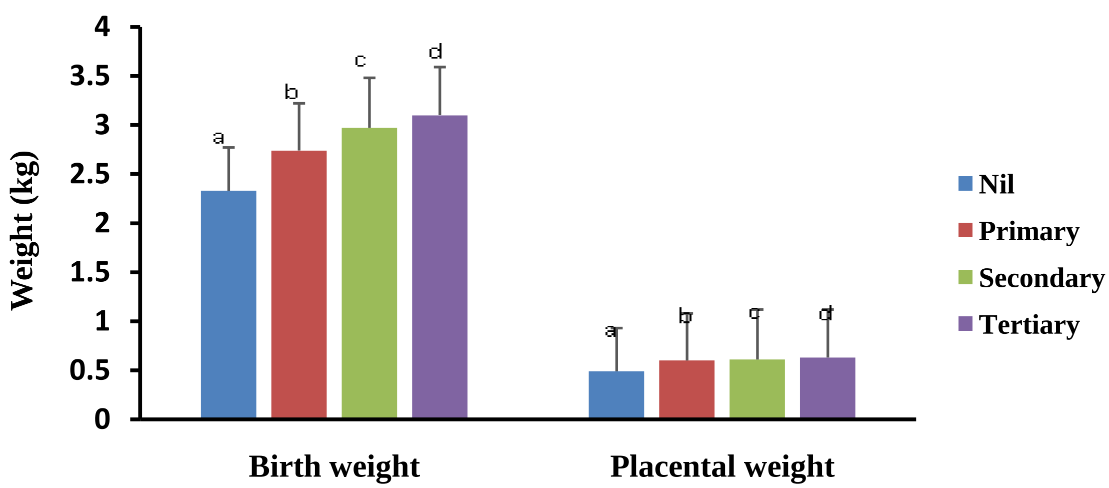

Figure 4.57: Birth and placental weights of female newborns according to maternal level of education. One way analysis of variance indicates significant difference in birth and placental weight based on maternal level of education, with F = 25.12, and F = 10.98, for birth and placental weights respectively. The birth and placental weight of newborns in mothers with tertiary education are significantly higher than secondary, primary and nil (a vs b vs c vs d $P < 0.001$). Maternal level of education with different superscript are significantly different with $p < 0.001$.

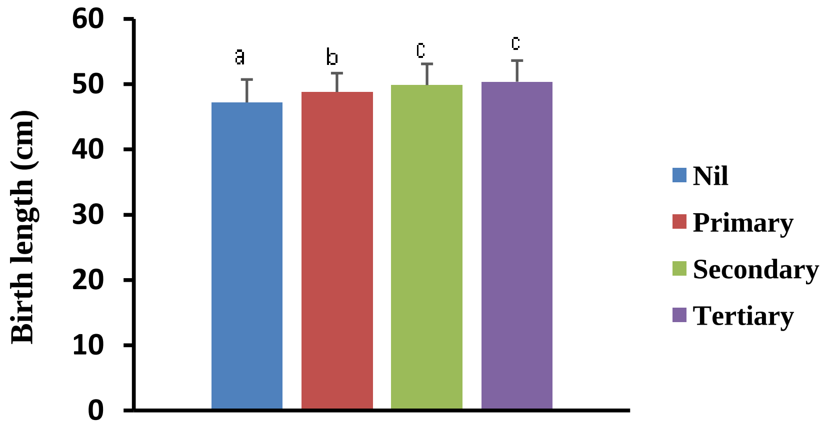

Figure 4.58: Birth length of female newborns according to maternal level of education. One way analysis of variance indicates significant difference in birth length based on maternal level of education, with F = 10.59. The birth length of newborns in mothers with secondary and tertiary education are significantly higher than mothers with primary and nil (a vs b vs c *P*< 0.001). Maternal level of education with different superscript are significantly different with *p*< 0.001.

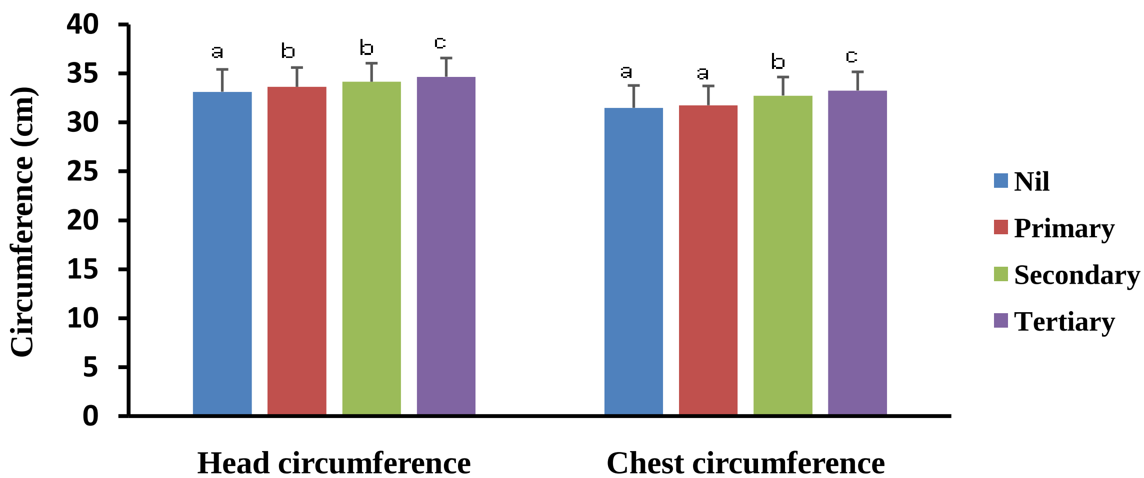

Figure 4.59: Head and chest circumferences of female newborns according to maternal level of education. One way analysis of variance indicates significant difference in head and chest circumference based on maternal level of education, with F = 10.84, and F = 11.99, for head and chest circumference respectively. The head and chest circumference of newborns in mothers with tertiary education are significantly higher than secondary, primary and nil (a vs b vs c *P*< 0.001). Maternal level of education with different superscript are significantly different with *p*< 0.001.

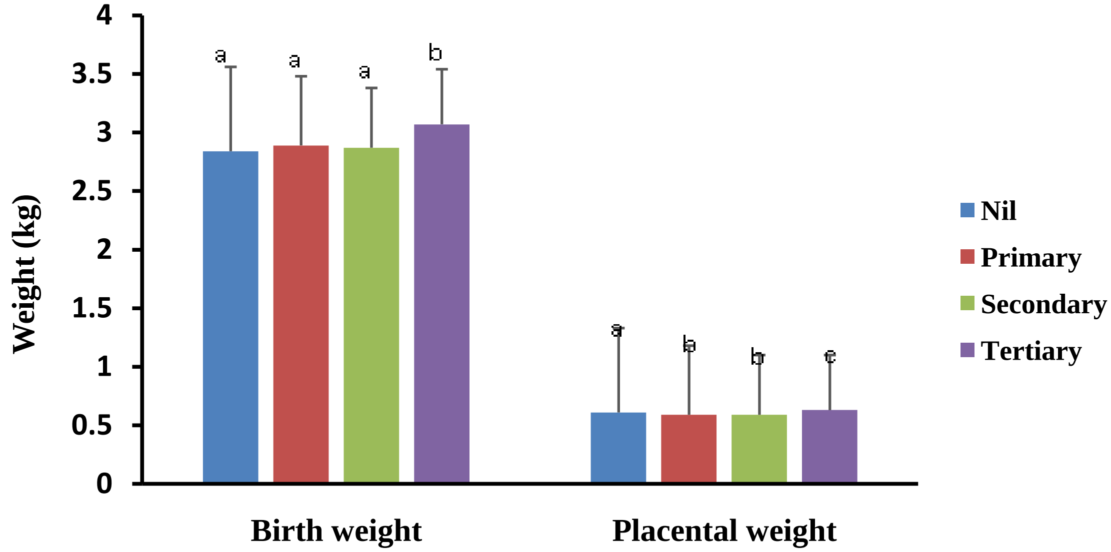

Figure 4.60: Birth and placental weights of female newborns according to paternal level of education. One way analysis of variance indicates significant difference in birth and placental weight based on paternal level of education, with F = 16.13, and F = 7.21, for birth and placental weights respectively. The birth and placental weight of newborns in fathers with tertiary education are significantly higher than fathers with secondary, primary and nil (a vs b vs c*P*< 0.001). Paternal level of education with different superscript are significantly different with *p*< 0.001.

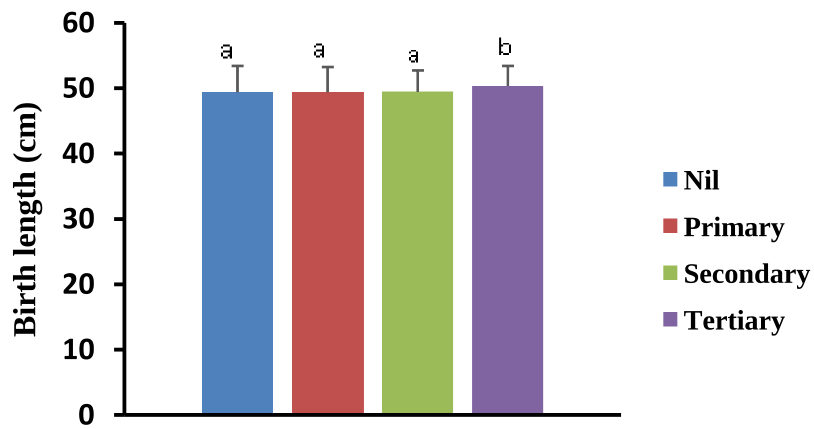

Figure 4.61: Birth length of female newborns according to paternal level of education. One way analysis of variance indicates significant difference in birth length based on paternal level of education, with F = 6.44. The birth length of newborns in fathers with tertiary education are significantly higher than fathers with secondary primary and nil (a vs b *P*< 0.001). Paternal level of education with different superscript are significantly different with *p*< 0.001.

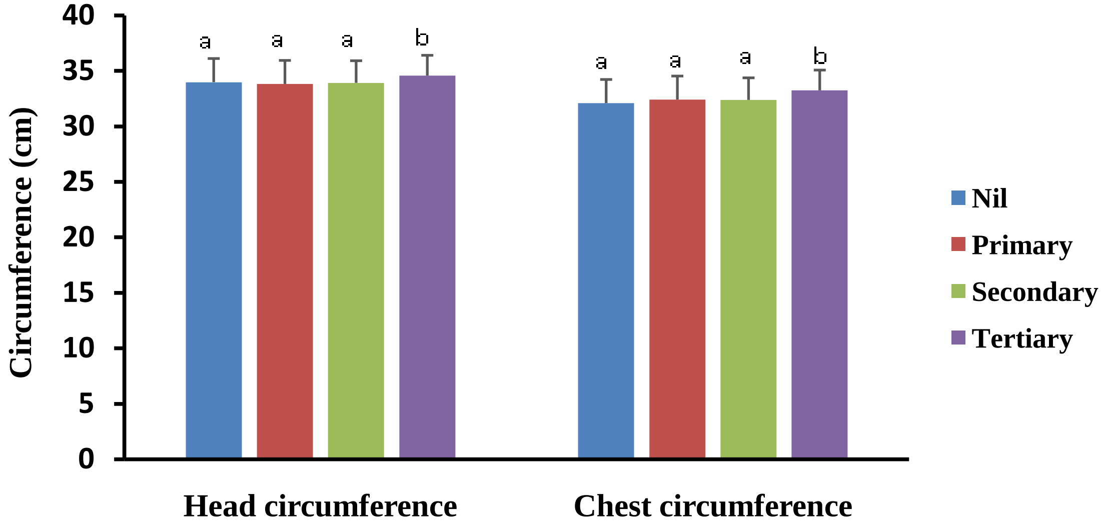

Figure 4.62: Head and chest circumferences of female newborns according to paternal level of education. One way analysis of variance indicates significant difference in head and chest circumference based on paternal level of education, with F = 8.45, and F = 11.66, for head and chest circumference respectively. The head and chest circumference of newborns in fathers with tertiary education are significantly higher than fathers with secondary, primary and nil (a vs b $P < 0.001$). Paternal level of education with different superscript are significantly different with $p < 0.001$.

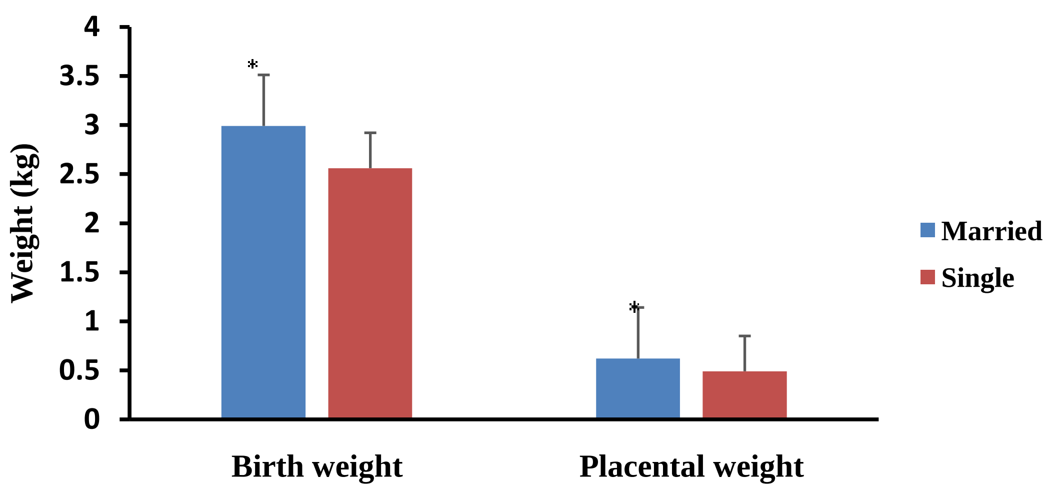

Figure 4.63: Birth and placental weights of female newborns according to maternal marital status. Birth and placental weight of newborns in married mothers are higher than single mothers. Student t-test indicates significant difference between birth and placental weight of newborns in married and single mothers, (t =3.16 and 3.52, $p < 0.01$).

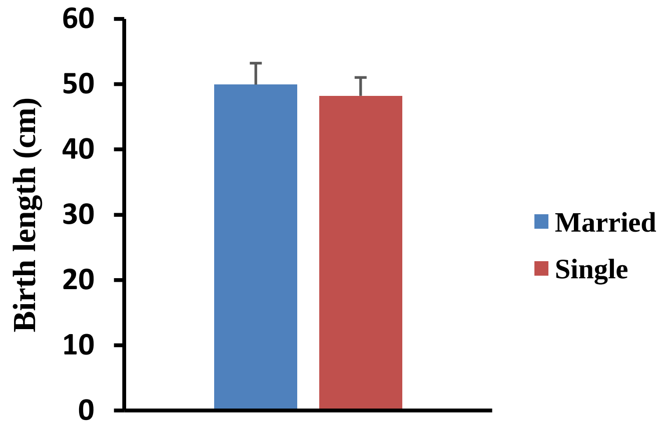

Figure 4.64: Birth length of female newborns according to maternal marital status. Student t-test indicates no significant difference between the birth length of newborns in married and single mothers, ($t = 1.95$, $p = 0.05$).

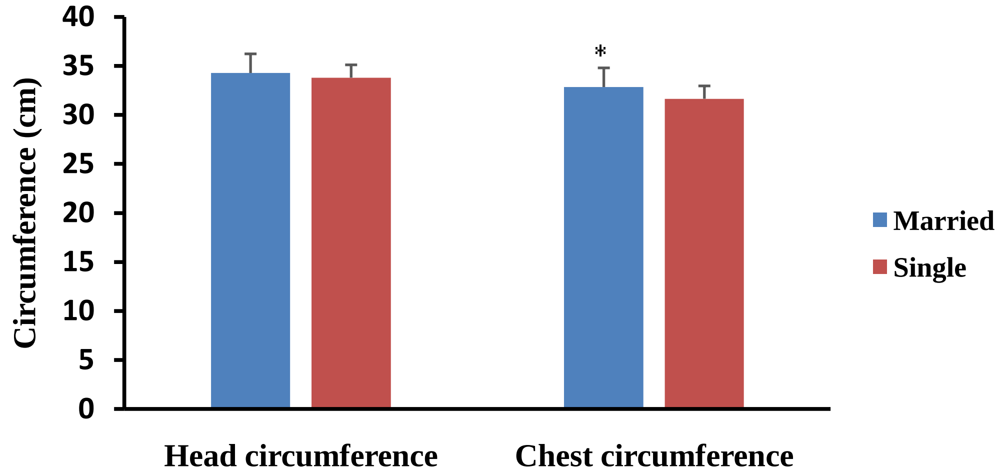

Figure 4.65: Head and chest circumferences of female newborns according to maternal marital status. Student t-test indicates no significant difference between the head circumference of newborns in married and single mothers, ($t = 0.93$, $p = 0.351$). However, the chest circumference of newborns in married mothers are significantly higher than single mothers, ($t = 1.97$, $p < 0.05$).

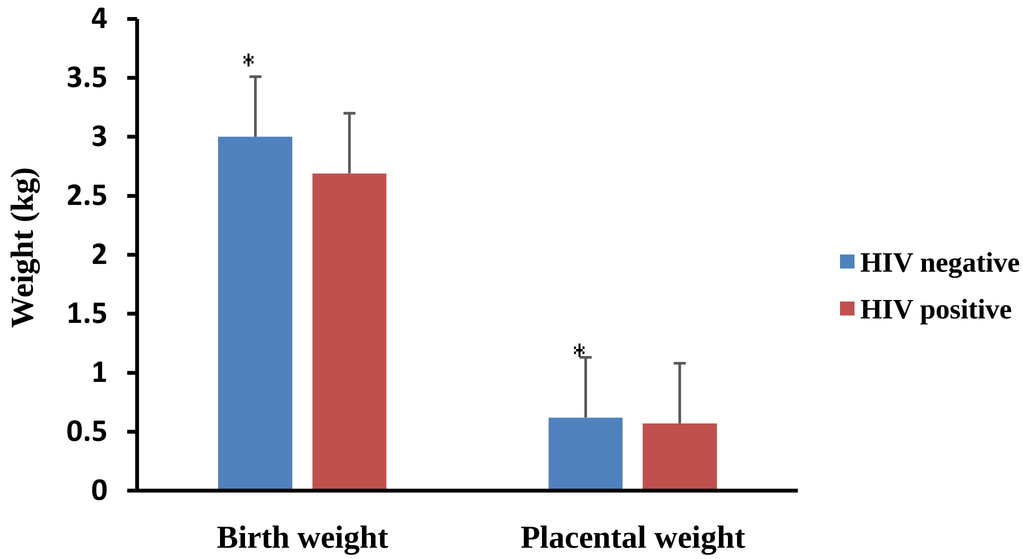

Figure 4.66: Birth and placental weights of female newborns according to maternal HIV status. Birth and placental weight of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between birth and placental weight of newborns in HIV negative and HIV positive mothers, ($t = 4.48$ and 2.97, $p < 0.001$).

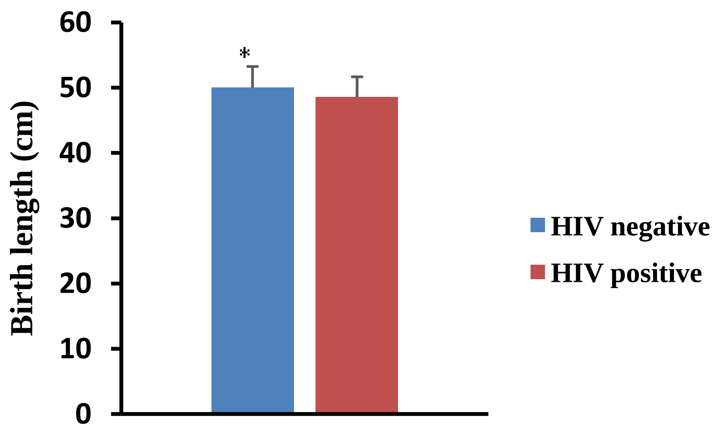

Figure 4.67: Birth length of female newborns according to maternal HIV status. Birth length of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between the birth length of newborns in HIV negative and HIV positive mothers, ($t = 3.32$, $p < 0.001$).

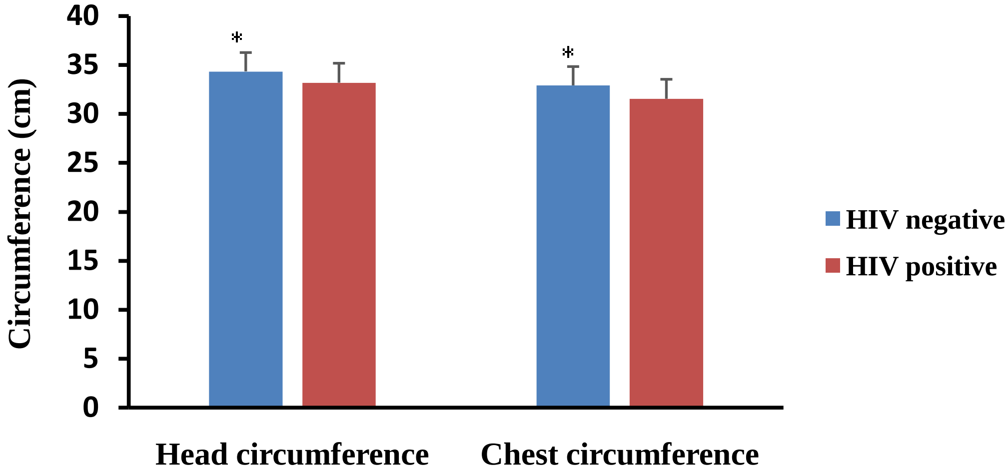

Figure 4.68: Head and chest circumferences of female newborns according to maternal HIV status. Head and chest circumference of newborns in HIV negative mothers are higher than HIV positive mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in HIV negative and HIV positive mothers, ($t = 4.51$ and 4.58, $p < 0.001$).

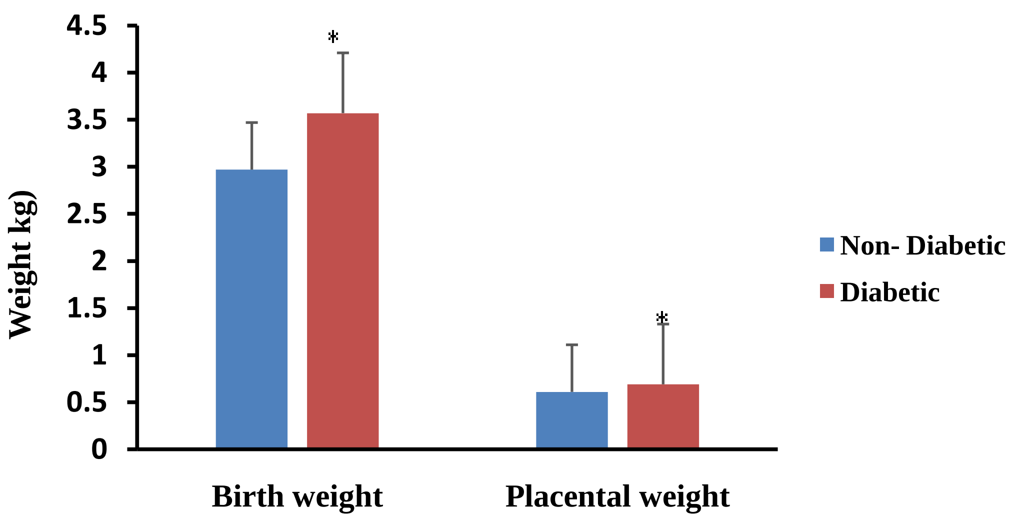

Figure 4.69: Birth and placental weights of female newborns according to maternal diabetes status. Birth and placental weight of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference in birth and placental weight of newborns of diabetic and non-diabetic mothers, ($t = -6.53$ and $t = -3.60$, $p < 0.001$).

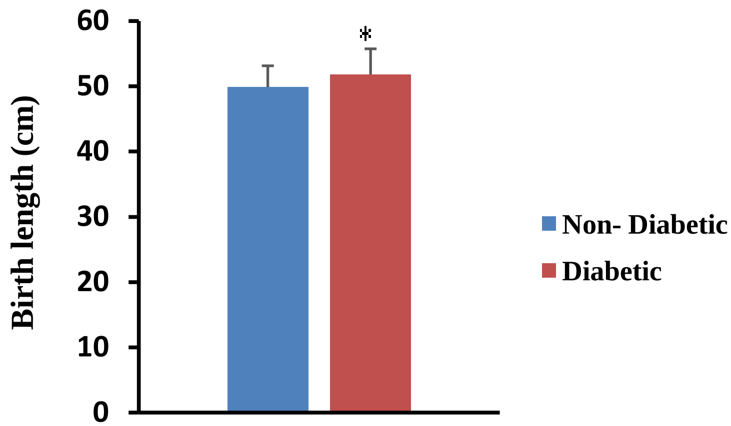

Figure 4.70: Birth length of female newborns according to maternal diabetes status. Birth length of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference between the birth length of newborns in diabetic and non-diabetic mothers, ($t = -3.28$, $p < 0.01$).

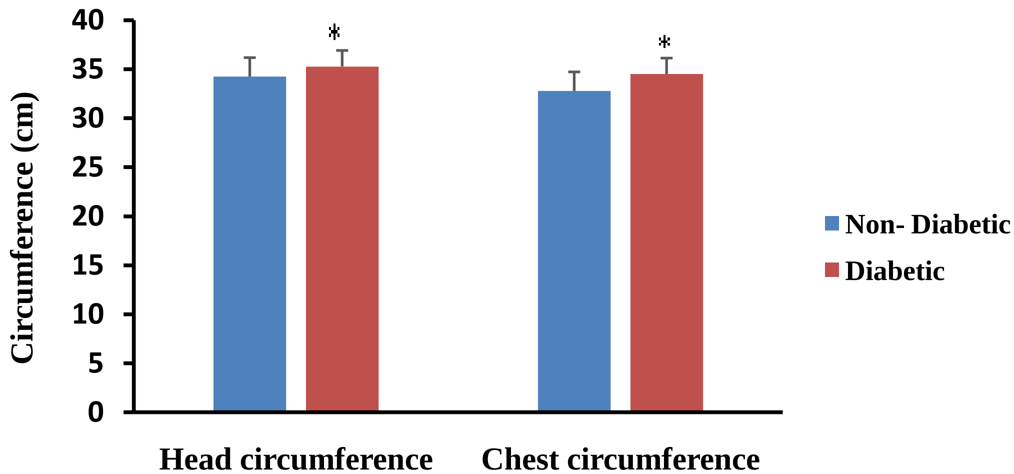

Figure 4.71: Head and chest circumferences of female newborns according to maternal diabetes status. Head and chest circumference of newborns in diabetic mothers are higher than non-diabetic mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in diabetic and non-diabetic mothers, ($t = -3.02$ and -4.27, $p < 0.01$).

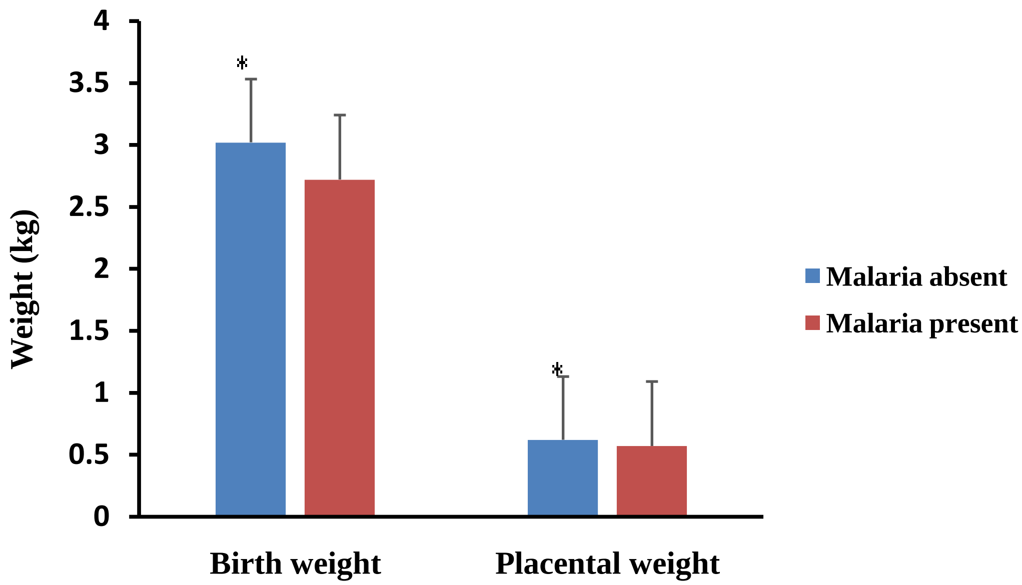

Figure 4.72: Birth and placental weights of female newborns according to maternal malarial episode. Birth and placental weight of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between birth and placental weight of newborns in malaria absent and malaria present mothers, ($t = 6.15$ and 4.26, $p < 0.001$).

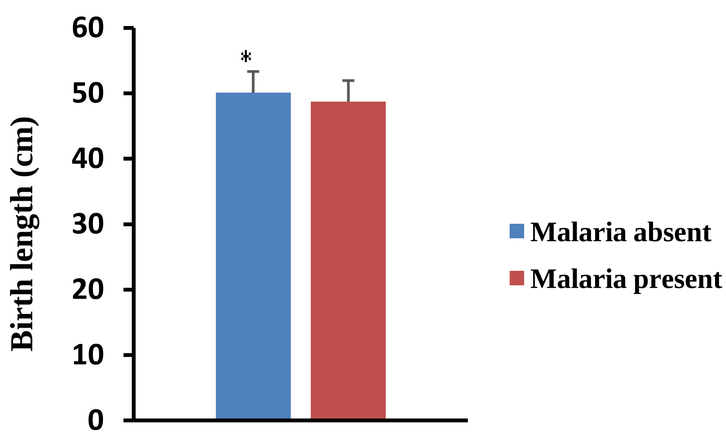

Figure 4.73: Birth length of female newborns according maternal malarial episode. Birth length of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between the birth length of newborns in malaria absent and malaria present mothers, ($t = 4.30$, $p < 0.001$).

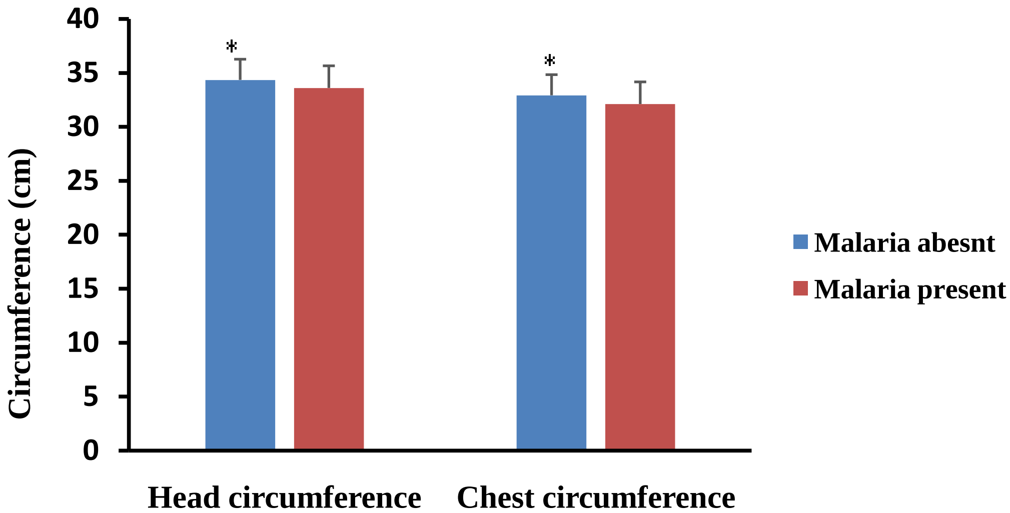

Figure 4.74: Head and chest circumferences of female newborns according to maternal malarial episode. Head and chest circumference of newborns in malaria absent mothers are higher than malaria present mothers. Student t-test indicates significant difference between the head and chest circumference of newborns in malaria absent and malaria present mothers, ($t = 4.02$ and 3.70, $p < 0.001$).

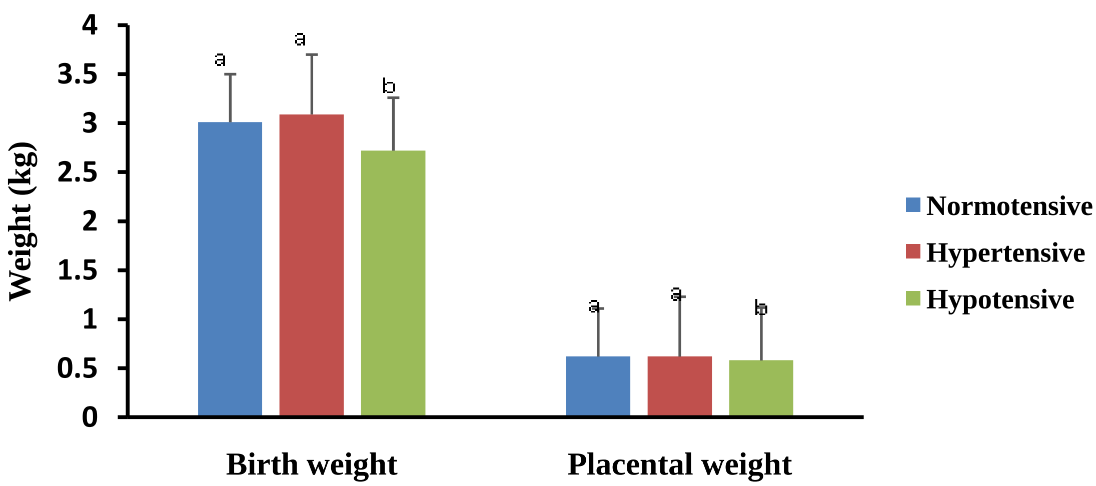

Figure 4.75: Birth and placental weights of female newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in birth and placental weight based on maternal blood pressure, with $F = 19.81$, and $F = 6.40$ for birth and placental weights respectively. The birth and placental weight of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b $P < 0.01$). Maternal blood pressure with different superscript are significantly different with $p < 0.01$.

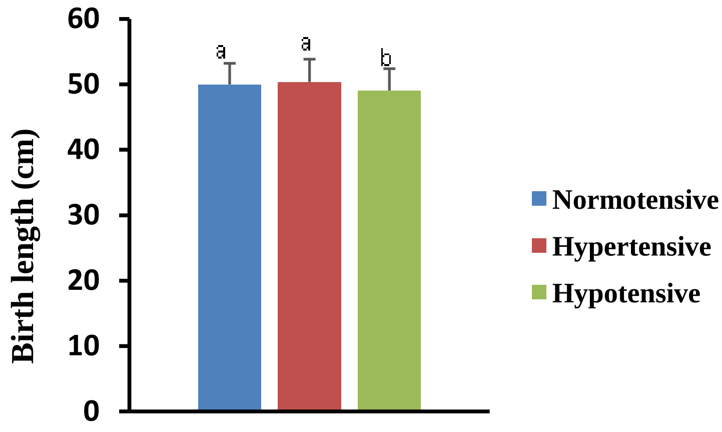

Figure 4.76: Birth length of female newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in birth length based on maternal blood pressure, with F = 5.46. The birth length of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b $p<0.01$). Maternal blood pressure with different superscript are significantly different with $p< 0.01$.

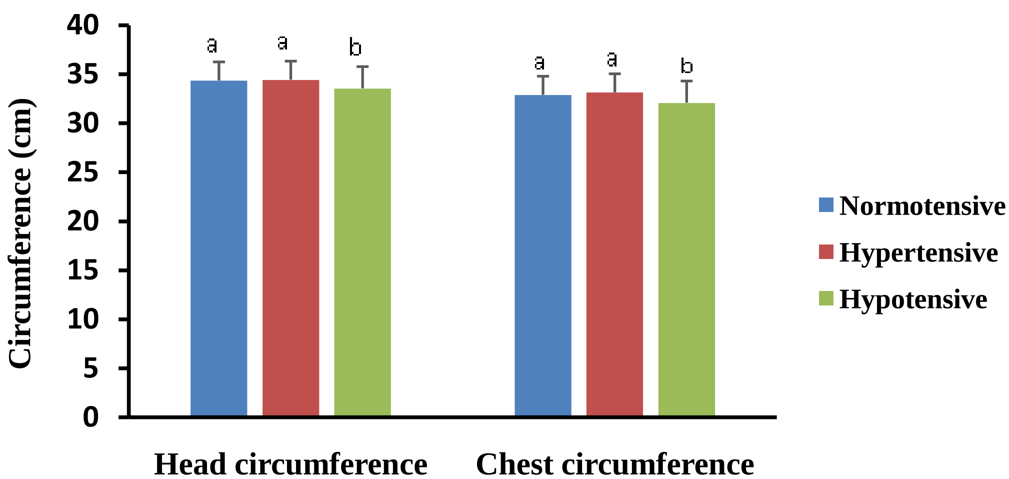

Figure 4.77: Head and chest circumferences of female newborns according to maternal blood pressure. One way analysis of variance indicates significant difference in head and chest circumference based on maternal blood pressure, with F = 9.61 and 8.08, for head and chest circumference respectively. The head and chest circumference of newborns in normotensive and hypertensive mothers are significantly higher than hypotensive mothers (a vs b $p<0.001$). Maternal blood pressure with different superscript are significantly different with $p< 0.001$

4.3. CORRELATION BETWEENNEONATAL AND MATERNALDEMOGRAPHIC AND ANTHROPOMETRIC VARIABLES

Table 4.18 showed the overall correlation matrix of neonatal and maternal anthropometric and demographic characteristics. Pearson's correlation coefficient was used to correlate the birth weight (BW) of newborns to the other parameters. Ninety four percent (94%) of the parameters correlated at $p < 0.001$, $p < 0.01$ and $p < 0.05$. BW showed a positive correlation with maternal age, parity, gestational age, Apgar score (AS) (1 and 5 min), birth length, head circumference, chest circumference, placental weight, maternal height, maternal weight, gestational weight gain, BMI, and PCV. So, in essence BW showed positive correlation with all the variables. Placental weight correlated positively with all the variables except PCV. Gestational age of the newborns correlated positively with all the variables. Birth length, head and chest circumference showed positive correlation with all the variables. AS (1min) correlated with all the variables except PCV. AS (5min) correlated with most of the variables except parity, BMI and PCV. Maternal height and weight showed positive correlation with all the variables. Gestational weight gain showed positive correlation with most of the variable and negative correlation with parity with no correlation with maternal age and maternal BMI. Maternal BMI showed positive correlation with all the variables except Apgar score (5min) and maternal gestational weight. PCV showed positive correlation with most of the variables except AS (1 and 5 min) and placental weight.

Table 4.19presents the correlation matrix of male neonatal and maternal anthropometric and demographic variables. Pearson's correlation coefficient was used to reveal the birth weight of male newborns and other parameters. Ninety three percent (93%) of the parameters correlated at $p < 0.001$, $p < 0.01$ and $p < 0.05$. BW of male newborns showed a positive correlation with maternal age, parity, gestational age, AS (1 and 5 min), birth length, head circumference, chest circumference, placental weight, maternal height, maternal weight, gestational weight gain, BMI, and PCV. So, in essence BW of male newborns showed positive correlation with all the variables. Placental weight correlated with all the variables except with maternal gestational weight gain. Gestational age of the male newborns correlated positively with all the variables. Birth length, head and chest circumference showed positive correlation with all the variables. AS (1min) correlated with most of the variables except maternal gestational weight gain and PCV. AS (5min) correlated with most of the variables except parity, maternal BMI and PCV. Maternal height showed positive correlation with all the variables. Maternal weight showed positive correlation with all the variables. Gestational weight gain showed positive correlation with some of the variables and negative correlation with parity with no correlation with maternal age, AS (1min), placental weight and maternal BMI. Maternal BMI showed positive correlation with all the variables except AS (5min) and maternal gestational weight gain. PCV showed positive correlation with most of the variables except AS (1 and 5 min).

Table 4.20 presents the correlation matrix of female neonatal and maternal anthropometric and demographic variables. Pearson's correlation coefficient was used to correlate the BW of female newborns to the other parameters. Ninety one percent (91%) of the parameters correlated at $p < 0.001$, $p < 0.01$ and $p < 0.05$. BW of female newborns showed a positive correlation with maternal age, parity, gestational age, AS (1 and 5 min), birth length, head circumference, chest circumference, placental weight, maternal height, maternal weight, gestational weight gain, BMI, and PCV. So, in essence BW of female newborns showed positive correlation with all the variables. Placental weight correlated with all the variables. Gestational age of the female newborns correlated positively with all the variables. Birth length, head and chest circumference showed positive correlation with all the variables. AS (1min) correlated with most of the variables except maternal weight, maternal BMI and PCV. AS (5min) correlated with most of the variables except maternal weight, maternal BMI and PCV. Maternal height showed positive correlation with all the variables. Maternal weight showed positive correlation with most of the variables except AS (1 and 5 min). Gestational weight gain showed positive correlation with most of the variable and negative correlation with parity with no correlation with maternal age and maternal BMI. Maternal BMI showed positive correlation with all the variables except AS (1 and 5min) and maternal gestational weight gain. PCV showed positive correlation with most of the variables except maternal age and AS (1 and 5 min).

Table 4.18: Correlation matrix of neonatal and maternal anthropometric and demographic variables for the combined population (male + female newborns).

	MA	P	GA	AS (1m)	AS (5m)	BW	BL	HC	CC	PW	MH	MW	GWG	BMI	PCV
MA	-	0.59[c]*	0.14[c]*	0.06[c]*	0.06[c]*	0.15[c]*	0.08[c#]	0.09[c#]	0.11[c#]	0.10[c#]	0.48[c†]	0.72[c†]	-0.02[†]	0.64[c†]	0.25[c†]
P	-	-	0.07[c]*	0.04[c]*	0.02*	0.08[c]*	0.04[c#]	0.04[c#]	0.06[c#]	0.07[c#]	0.27[c†]	0.49[c†]	-0.13[c†]	0.46[c†]	0.13[c†]
GA	-	-	-	0.14[c]*	0.17[c]*	0.56[c]*	0.42[c#]	0.48[c#]	0.49[c#]	0.43[c#]	0.17[c†]	0.22[c†]	0.34[c†]	0.17[c†]	0.11[c†]
AS (1m)	-	-	-	-	0.82[c]*	0.17[c]*	0.17[c#]	0.14[c#]	0.16[c#]	0.09[c#]	0.09[c†]	0.07[c†]	0.06[b†]	0.04[a†]	0.03[†]
AS (5m)	-	-	-	-	-	0.22[c]*	0.19[c#]	0.17[c#]	0.20[c#]	012[c#]	0.08[c†]	0.06[b†]	0.07[c†]	0.03[†]	0.02[†]
BW	-	-	-	-	-	-	0.67[c#]	0.67[c#]	0.76[c#]	0.71[c#]	0.29[c†]	0.31[c†]	0.62[c†]	0.23[c†]	0.15[c†]
BL	-	-	-	-	-	-	-	0.52[c#]	0.54[c#]	0.52[c#]	0.16[c†]	0.18[c†]	0.42[c†]	0.14[c†]	0.08[c†]
HC	-	-	-	-	-	-	-	-	0.74[c#]	0.55[c#]	0.19[c†]	0.19[c†]	0.39[c†]	0.13[c†]	0.09[c†]
CC	-	-	-	-	-	-	-	-	-	0.58[c#]	0.23[c†]	0.22[c†]	0.47[c†]	0.15[c†]	0.11[c†]
PW	-	-	-	-	-	-	-	-	-	-	0.22[c†]	0.25[c†]	0.41[c†]	0.19[c†]	0.11[†]
MH	-	-	-	-	-	-	-	-	-	-	-	0.60[c†]	0.21[c†]	0.21[c†]	0.24[c†]
MW	-	-	-	-	-	-	-	-	-	-	-	-	0.09[c†]	0.91[c†]	0.30[c†]
GWG	-	-	-	-	-	-	-	-	-	-	-	-	-	-0.00[†]	0.14[c†]
BMI	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.24[c†]
PCV															

[*]n=10586 [#]n=8795 [†]n=2276 a = P<0.05 b = P<0.01 c = P<0.001

MA= Maternal age, P= Parity, GA= Gestational age (in weeks), AS (1m)= Apgar Score (1 minute), AS (5m)= Apgar Score (5 minute), BW= Birth weight (kg), BL= Birth length (cm), HC= Head circumference (cm), CC= Chest circumference (cm), PW= Placental weight (kg), MH= Maternal height (cm), MW= Maternal weight (kg), GWG= Gestational weight gain (kg), BMI= Body mass index (kg/m^2), and PCV= Packed cell volume.

Table 4.19: Correlation matrix of male neonatal and maternal anthropometric and demographic variables

	M. Age	Parity	G.A	A.S (1m)	A.S (5m)	BW	BL	HC	CC	PW	M.H	M.W	GWG	BMI	PCV
M. Age	-	0.57^{c}*	0.13^{c}*	0.06^{c}*	0.05^{c}*	0.14^{c}*	$0.08^{\#}$	$0.09^{c\#}$	$0.11^{c\#}$	$0.10^{c\#}$	$0.47^{c\dagger}$	$0.68^{c\dagger}$	$-0.04^{\dagger}$	$0.58^{c\dagger}$	$0.22^{c\dagger}$
Parity	-	-	0.07^{c}*	0.04^{b}*	0.00*	0.08^{c}*	$0.03^{a\#}$	$0.04^{a\#}$	$0.04^{b\#}$	$0.08^{c\#}$	$0.24^{c\dagger}$	$0.47^{c\dagger}$	$-0.14^{c\dagger}$	$0.44^{c\dagger}$	$0.12^{c\dagger}$
G.A	-	-	-	0.10^{c}*	0.13^{c}*	0.53^{c}*	$0.36^{c\#}$	$0.44^{c\#}$	$0.44^{c\#}$	$0.39^{c\#}$	$0.17^{c\dagger}$	$0.20^{c\dagger}$	$0.32^{c\dagger}$	$0.16^{c\dagger}$	$0.09^{c\dagger}$
A.S (1m)	-	-	-	-	0.83^{c}*	0.17^{c}*	$0.14^{c\#}$	$0.14^{c\#}$	$0.16^{c\#}$	$0.08^{c\#}$	$0.09^{b\dagger}$	$0.10^{c\dagger}$	$0.04^{\dagger}$	$0.08^{b\dagger}$	$0.01^{\dagger}$
A.S (5m)	-	-	-	-	-	0.19^{c}*	$0.16^{c\#}$	$0.15^{c\#}$	$0.19^{c\#}$	$0.09^{c\#}$	$0.08^{b\dagger}$	$0.08^{b\dagger}$	$0.06^{a\dagger}$	$0.05^{\dagger}$	$-0.01^{\dagger}$
BW	-	-	-	-	-	-	$0.67^{c\#}$	$0.67^{c\#}$	$0.75^{c\#}$	$0.71^{c\#}$	$0.29^{c\dagger}$	$0.29^{c\dagger}$	$0.59^{c\dagger}$	$0.19^{c\dagger}$	$0.13^{c\dagger}$
BL	-	-	-	-	-	-	-	$0.49^{c\#}$	$0.52^{c\#}$	$0.50^{c\#}$	$0.13^{c\dagger}$	$0.15^{c\dagger}$	$0.40^{c\dagger}$	$0.11^{c\dagger}$	$0.07^{a\dagger}$
HC	-	-	-	-	-	-	-	-	$0.71^{c\#}$	$0.54^{c\#}$	$0.18^{c\dagger}$	$0.16^{c\dagger}$	$0.37^{c\dagger}$	$0.10^{c\dagger}$	$0.06^{c\dagger}$
CC	-	-	-	-	-	-	-	-	-	$0.57^{c\#}$	$0.22^{c\dagger}$	$0.19^{c\dagger}$	$0.43^{c\dagger}$	$0.11^{c\dagger}$	$0.09^{c\dagger}$
PW	-	-	-	-	-	-	-	-	-	-	$0.22^{c\dagger}$	$0.25^{c\dagger}$	$0.39^{\dagger}$	$0.18^{c\dagger}$	$0.08^{b\dagger}$
M.H	-	-	-	-	-	-	-	-	-	-	-	$0.58^{c\dagger}$	$0.22^{c\dagger}$	$0.18^{c\dagger}$	$0.23^{c\dagger}$
M.W	-	-	-	-	-	-	-	-	-	-	-	-	$0.07^{a\dagger}$	$0.90^{c\dagger}$	$0.29^{c\dagger}$
GWG	-	-	-	-	-	-	-	-	-	-	-	-	-	$-0.04^{\dagger}$	$0.13^{c\dagger}$
BMI	-	-	-	-	-	-	-	-	-	-	-	-	-	-	$0.24^{c\dagger}$
PCV															

*n=5660 #n=4688 †n=1169 a = $P<0.05$ b = $P<0.01$ c = $P<0.001$

MA= Maternal age, P= Parity, GA= Gestational age (in weeks), AS (1m)= Apgar Score (1 minute), AS (5m)= Apgar Score (5 minute), BW= Birth weight (kg), BL= Birth length (cm), HC= Head circumference (cm), CC= Chest circumference (cm), PW= Placental weight (kg), MH= Maternal height (cm), MW= Maternal weight (kg), GWG= Gestational weight gain (kg), BMI= Body mass index (kg/m^2), and PCV= Packed cell volume.

Table 4.20: Correlation matrix of female neonatal and maternal anthropometric and demographic variables

	MA	P	GA	AS (1m)	AS (5m)	BW	BL	HC	CC	PW	MH	MW	GWG	BMI	PCV
MA	-	0.58^{c*}	0.15^{c*}	0.07^{c*}	0.07^{c*}	0.17^{c*}	$0.09^{c\#}$	$0.09^{c\#}$	$0.11^{c\#}$	$0.10^{c\#}$	$0.50^{c\dagger}$	$0.74^{c\dagger}$	$-0.01^{\dagger}$	$0.65^{c\dagger}$	$0.28^{\dagger}$
P	-	-	0.08^{c*}	0.04^{b*}	0.03^{a*}	0.09^{c*}	$0.05^{c\#}$	$0.05^{b\#}$	$0.07^{c\#}$	$0.06^{c\#}$	$0.30^{c\dagger}$	$0.53^{c\dagger}$	$-0.12^{c\dagger}$	$0.49^{c\dagger}$	$0.14^{c\dagger}$
GA	-	-	-	0.17c*	0.21^{c*}	0.58^{c*}	$0.47^{c\#}$	$0.50^{c\#}$	$0.52^{c\#}$	$0.46^{c\#}$	$0.16^{c\dagger}$	$0.22^{c\dagger}$	$0.35^{c\dagger}$	$0.19^{c\dagger}$	$0.12^{c\dagger}$
AS (1m)	-	-	-	-	0.80^{c*}	0.18^{c*}	$0.20^{c\#}$	$0.14^{c\#}$	$0.16^{c\#}$	$0.12^{c\#}$	$0.08^{b\dagger}$	$0.04^{\dagger}$	$0.08^{b\dagger}$	$0.01^{\dagger}$	$0.05^{\dagger}$
AS (5m)	-	-	-	-	-	0.24^{c*}	$0.24^{c\#}$	$0.18^{c\#}$	$0.21^{c\#}$	$0.15^{c\#}$	$0.07^{a\dagger}$	$0.04^{\dagger}$	$0.09^{b\dagger}$	$0.01^{\dagger}$	$0.05^{\dagger}$
BW	-	-	-	-	-	-	$0.67^{c\#}$	$0.66^{c\#}$	$0.77^{c\#}$	$0.70^{c\#}$	$0.28^{c\dagger}$	$0.33^{c\dagger}$	$0.63^{c\dagger}$	$0.27^{c\dagger}$	$0.18^{c\dagger}$
BL	-	-	-	-	-	-	-	$0.53^{c\#}$	$0.56^{c\#}$	$0.53^{c\#}$	$0.16^{c\dagger}$	$0.20^{c\dagger}$	$0.42^{c\dagger}$	$0.17^{c\dagger}$	$0.09^{b\dagger}$
HC	-	-	-	-	-	-	-	-	$0.76^{c\#}$	$0.56^{c\#}$	$0.18^{c\dagger}$	$0.21^{c\dagger}$	$0.41^{c\dagger}$	$0.17^{c\dagger}$	$0.12^{c\dagger}$
CC	-	-	-	-	-	-	-	-	-	$0.59^{c\#}$	$0.23^{c\dagger}$	$0.24^{c\dagger}$	$0.49^{c\dagger}$	$0.18^{c\dagger}$	$0.12^{c\dagger}$
PW	-	-	-	-	-	-	-	-	-	-	$0.21^{c\dagger}$	$0.24^{c\dagger}$	$0.43^{c\dagger}$	$0.19^{c\dagger}$	$0.14^{c\dagger}$
MH	-	-	-	-	-	-	-	-	-	-	-	$0.62^{c\dagger}$	$0.19^{c\dagger}$	$0.24^{c\dagger}$	$0.24^{c\dagger}$
MW	-	-	-	-	-	-	-	-	-	-	-	-	$0.11^{c\dagger}$	$0.91^{c\dagger}$	$0.30^{c\dagger}$
GWG	-	-	-	-	-	-	-	-	-	-	-	-	-	$0.03^{\dagger}$	$0.14^{c\dagger}$
BMI	-	-	-	-	-	-	-	-	-	-	-	-	-	-	$0.25^{c\dagger}$
PCV															

*n=4926 $^{\#}$n=4107 †n=1107 a = P<0.05 b = P<0.01 c = P<0.001

MA= Maternal age, P= Parity, GA= Gestational age (in weeks), AS (1m)= Apgar Score (1 minute), AS (5m)= Apgar Score (5 minute), BW= Birth weight (kg), BL= Birth length (cm), HC= Head circumference (cm), CC= Chest circumference (cm), PW= Placental weight (kg), MH= Maternal height (cm), MW= Maternal weight (kg), GWG= Gestational weight gain (kg), BMI= Body mass index (kg/m^2), and PCV= Packed cell volume.

Tables (4.21 – 4.23) showed linear regression equations of birth weight (BW) of both male and female neonates from neonatal and maternal anthropometric and demographic variables Table 4.21. From the table, chest circumference (CC) was a stronger predictor of BW of neonates than the other variables. The value of R^2 (coefficient of determination) for predicting BW of both male and female neonates using CCwas0.58, $p < 0.001$. Also, placental weight (PW), birth length (BL), head circumference (HC), gestational weight gain (GWG), and gestational age (GA) were better predictors of BW of both sexes relative to other variables. The values of R^2 (coefficient of determination) for predicting BW of both sexes using the aforementioned variableswere respectively 0.50, 0.46, 0.45, 0.38 and 0.31, $p < 0.001$. Table 4.22 present linear regression equations of BW of male neonates from neonatal and maternal anthropometric and demographic variables. Also from the table, CC was a stronger predictor of BW of male neonate than the other variables. The value of R^2 (coefficient of determination) for predicting BW of male neonates using CC was0.56, $p < 0.001$. Also, PW, BL, HC, GWG, and gestational age (GA) were better predictors of BW of male neonates relative to other variables. The values of R^2 (coefficient of determination) for predicting BW of male neonates using the aforementioned variables were respectively 0.50, 0.45, 0.45, 0.36 and 0.29, $p < 0.001$. Table 4.23 showed linear regression equations of BW of female neonates from neonatal and maternal anthropometric and demographic variables. Also from the table, chest circumference (CC) was a stronger possible predictor of birth weight of female neonates relative to other variables. The value of R^2 (coefficient of determination) for predicting birth weight of female neonates using CC was0.59, $p < 0.001$. Also, PW, BL, HC, GWG, and GA were better predictors of BW of female neonates relative to other variables. The values of R^2 (coefficient of determination) for predicting BW of female neonate using the aforementioned variables were respectively 0.50, 0.45, 0.43, 0.40 and 0.34, $p < 0.001$.

Table 4.21: Linear regression table showing predictive equations of the birth weight of both male and female neonates from neonatal and maternal anthropometric and demographic variables

Parameters	N	Predictive Equation	r	r^2	SEE	B	T	F	P
Maternal Age	10586	BW= 2.707 + (0.014* MA)	0.151	0.023	0.526	2.707	110.114	247.589	<0.001
Parity	10586	BW= 3.043 + (0.022* P)	0.083	0.007	0.530	3.043	421.448	73.492	<0.001
Gestational age	10586	BW= -1.284 + (0.117* GA)	0.559	0.313	0.441	-1.284	-20.339	4814.395	<0.001
Apgar Score (1m)	10586	BW= 2.576 + (0.0667* AS)	0.172	0.029	0.524	2.576	89.229	321.249	<0.001
Apgar Score (5m)	10586	BW= 2.048 + (0.113* AS)	0.218	0.047	0.519	2.048	44.982	526.179	<0.001
Birth length (cm)	8795	BW= -1.835 + (0.098* BL)	0.674	0.455	0.395	-1.835	-31.874	7336.254	<0.001
Head circumference (cm)	8795	BW= -2.443 + (0.161* H.C)	0.667	0.445	0.399	-2.443	-37.056	7053.980	<0.001
Chest circumference (cm)	8795	BW= -2.753 + (0.176* CC)	0.759	0.576	0.349	-2.753	-51.396	11925.525	<0.001
Placental weight (kg)	8795	BW= 1.491 + (2.550* PW)	0.708	0.502	0.378	1.491	85.820	8852.747	<0.001
Maternal height (cm)	2276	BW= -1.033 + (0.026* MH)	0.291	0.085	0.511	-1.033	-3.655	210.811	<0.001
Maternal weight (kg)	2276	BW= 2.115 + (0.0141* MW)	0.312	0.097	0.507	2.115	34.188	244.959	<0.001
Gestational weight gain (kg)	2276	BW= 2.320 + (0.108* GWG)	0.620	0.384	0.419	2.320	106.724	1417.568	<0.001
BMI	2276	BW= 2.210 + (0.0324* BMI)	0.230	0.053	0.520	2.210	28.695	126.923	<0.001

MA= Maternal age, P= Parity, GA= Gestational age (in weeks), AS (1m)= Apgar Score (1 minute), AS (5m)= Apgar Score (5 minute), BW= Birth weight (kg), BL= Birth length (cm), HC= Head circumference (cm), CC= Chest circumference (cm), PW= Placental weight (kg), , MH= Maternal height (cm), MW= Maternal weight (kg), GWG= Gestational weight gain (kg) and BMI= Body mass index (kg/m^2).

4.22: Linear regression table showing predictive equations of the birth weight of male neonate from neonatal and maternal anthropometric and demographic variables

Parameters	n	Predictive Equation	r	r^2	SEE	B	T	F	P
Maternal Age	5660	BW= 2.812 + (0.013* MA)	0.138	0.019	0.527	2.812	84.433	109.810	<0.001
Parity	5660	BW= 3.114 + (0.019* P)	0.075	0.006	0.530	3.114	316.136	32.006	<0.001
Gestational age	5660	BW= -1.173 + (0.115* G.A)	0.534	0.285	0.450	-1.173	-12.849	2255.473	<0.001
Apgar Score (1m)	5660	BW= 2.659 + (0.065*AS)	0.167	0.028	0.525	2.659	67.352	161.599	<0.001
Apgar Score (5m)	5660	BW= 2.212 + (0.102* AS)	0.195	0.038	0.522	2.212	34.985	224.438	<0.001
Birth length (cm)	4688	BW= -1.996 + (0.102* BL)	0.668	0.447	0.399	-1.996	-23.794	3785.331	<0.001
Head circumference (cm)	4688	BW= -2.557 + (0.166* HC)	0.671	0.450	0.398	-2.557	-27.701	3841.655	<0.001
Chest circumference (cm)	4688	BW= -2.866 + (0.181*CC)	0.749	0.561	0.356	-2.866	-36.760	5986.864	<0.001
Placental weight (kg)	4688	BW= 1.597 + (2.455* PW)	0.710	0.504	0.378	1.597	68.764	4760.617	<0.001
Maternal height (cm)	1169	BW= -0.841 + (0.025* MH)	0.285	0.081	0.516	-0.841	-2.139	102.952	<0.001
Maternal weight (kg)	1169	BW= 2.269 + (0.0129* MW)	0.286	0.082	0.515	2.269	26.024	104.287	<0.001
Gestational weight gain (kg)	1169	BW= 2.402 + (0.102* GWG)	0.599	0.359	0.431	2.402	75.764	653.435	<0.001
BMI	1169	BW= 2.426 + (0.027* BMI)	0.195	0.038	0.527	2.426	22.700	46.269	<0.001

MA= Maternal age, P= Parity, GA= Gestational age (in weeks), AS (1m)= Apgar Score (1 minute), AS (5m)= Apgar Score (5 minute), BW= Birth weight (kg), BL= Birth length (cm), HC= Head circumference (cm), CC= Chest circumference (cm), PW= Placental weight (kg),MH= Maternal height (cm), MW= Maternal weight (kg), GWG= Gestational weight gain (kg) and BMI= Body mass index (kg/m^2)

Table 4.23: Linear regression table showing predictive equations of the birth weight of female neonate from neonatal and maternal anthropometric and demographic variables

Parameters	n	Predictive Equation	R	r^2	SEE	B	T	F	P
Maternal Age	4926	BW= 2.585 + (0.016* MA)	0.170	0.029	0.514	2.585	72.420	146.545	<0.001
Parity	4926	BW= 2.962+ (0.024* P)	0.091	0.008	0.520	2.962	284.681	40.806	<0.001
Gestational age	4926	BW= -1.292 + (0.115* G.A)	0.581	0.338	0.425	-1.292	-15.013	2508.680	<0.001
Apgar Score (1m)	4926	BW= 2.481+ (0.069* AS)	0.181	0.033	0.513	2.481	59.830	167.012	<0.001
Apgar Score (5m)	4926	BW= 1.879 + (0.123* AS)	0.244	0.059	0.506	1.879	29.255	312.933	<0.001
Birth length (cm)	4107	BW= -1.580 + (0.092* BL)	0.671	0.451	0.387	-1.580	-19.950	3367.499	<0.001
Head circumference (cm)	4107	BW= -2.198 + (0.153* HC)	0.655	0.429	0.394	-2.198	-23.440	3088.461	<0.001
Chest circumference (cm)	4107	BW= -2.544 + (0.160* CC)	0.765	0.585	0.336	-2.544	-34.797	5782.794	<0.001
Placental weight (kg)	4107	BW= 1.393 + (2.628* PW)	0.704	0.496	0.370	1.393	53.652	4038.584	<0.001
Maternal height (cm)	1107	BW= -0.942 + (0.025* MH)	0.282	0.079	0.497	-0.942	-2.338	95.272	<0.001
Maternal weight (kg)	1107	BW= 1.993 + (0.015* MW)	0.332	0.110	0.489	1.993	23.110	137.034	<0.001
Gestational weight gain (kg)	1107	BW= 2.254 + (0.112* GWG)	0.630	0.397	0.402	2.254	75.744	727.531	<0.001
BMI	1107	BW= 1.993 + (0.038* BMI)	0.268	0.072	0.499	1.993	18.357	85.612	<0.001

MA= Maternal age, P= Parity, GA= Gestational age (in weeks), AS (1m)= Apgar Score (1 minute), AS (5m)= Apgar Score (5 minute), BW= Birth weight (kg), BL= Birth length (cm), HC= Head circumference (cm), CC= Chest circumference (cm), PW= Placental weight (kg),MH= Maternal height (cm), MW= Maternal weight (kg), GWG= Gestational weight gain (kg) and BMI= Body mass index (kg/m^2)

CHAPTER FIVE

5.0 DISCUSSION

The prevalence of low birth weight (LBW) neonates in this study of 10.2% is lower than 11.4% (Olowonyo *et al.*, 2006) observed in Ogun State, south west Nigeria and 26.8% (Mumbare *et al.*, 2012) reported in India. Also, the prevalence of high birth weight (HBW) or macrosomia of 3.8% is lower than 5.6% (Padilhal *et al.*, 2009) observed in Brazil and 10.6% (Njokanma, 2013) observed in South Eastern Nigeria. In this survey the prevalence of LBW was significantly higher in female newborns compared to male newborns (P = 0.001). This finding is not consistent with the report of Idris *et al.* (2014) in Maiduguri but is consistent with reports of Finster and Wood, 2005 and Kramer, 1987. Also, LBW deliveries was more prevalent in teenage mothers (17.6%) and mothers aged forty (40) and above (12.6%), compared to the prevalence of LBW deliveries among mothers aged 20 to 30 (9.5%) and 31 to 39 (9.1%) which showed statistical significant difference, (P = 0.001). This could be due to lack of maternal biological maturity among teenage mothers and reproductive tissue deterioration and decrease energy storesin older mothers. This is in keeping with the reports of Voight *et al.* (2004) in Germany, Smith *et al*. (2000) in the United States, Amosu *et al*. (2014) and Taiwo *et al*. (2014) in Nigeria.This survey also found the prevalence of LBW deliveries to be more common in babies delivered preterm (25.4%) compared to term babies (4.04%) which showed statistical significance,(P = 0.001). This was also reported by Macleod and Kiely, 1988, Voight *et al.* (2004), Kazaura *et al.*(2006).The current survey found that maternal education has effect on birth weight (BW) of babies with mothers with no education having higher prevalence of LBW deliveries (49.3%) compared to mothers with primary (26.6%), secondary (11.0%) and the least among mothers with tertiary (8.9%) levels of education whichwas statistically significant,(P =

0.001). Education could guide a woman to make appropriate dietary intake and informed choices on behalf of her unborn child. Also in most cases the higher the educational attainment of the mother the better her socioeconomic status. This was earlier reported (Chevalier and O'Sullivan, 2007; Padilhal *et al.,* 2009; Njokanma, 2013; Amosu *et al.*, 2014; Wiist, 2015; Altenhoner *et al.,* 2016). The prevalence of LBWdeliveries was observed to be significantly higher (P = 0.001) among babies that had fathers with no education (25.8%) relative to babies born to fathers with primary education (17.5%), secondary level of education (15.8%) and fathers with tertiary education (7.95%).This is in keeping with earlier reports(Makhija and Murthy, 1990; Njokanma, 2013; Dahlui *et al.*,2016). In this survey, the prevalence of LBW was significantly higher (P = 0.005) among primiparous mothers (11.8%) compared to mothers giving birth for the second (9.3%), third (9.9%), fourth (8.3%) and fifth and above times (10.7%). This was earlier reported (Hirve *et al.,* 1994;Lawoyin, 2007; Amosu *et al.,* 2014; Dahlui*et al.,*2016). The prevalence of LBW deliveries was observed to be significantly higher (P = 0.001) among mother who gained less than 5 kg (28.6%) compared to those mothers who gained from 5 to 11.9 kg (3.4 %)) and none of the mothers who gained 12 kg and above delivered LBW baby. This could be that gestational weight gain results in the development of maternal reproductive tissue which in turn increase blood volume and extracellular fluid and also increases maternal energy store. The finding in this study is consistent with what was reported byMorgan, 1997 in United States, Mohanty *et al.* (2006) in India, Padilhal *et al.* (2009) in Brazil.It was observed that the prevalence of LBWwas significantly higher (P = 0.001)among single mothers (37.5%) compared to married mothers (11.8%).This is in keeping with earlier reports (Guadino, 1993; Ventura, 1995; Chomitz *et al.,* 1995; Kazaura *et al.,* 2006). Mothers who had an episode of malarial attacked during pregnancy had significantly higher (P = 0.001) prevalence of LBW babies

(23.6%) compared to mothers who didn't have an episode of malarial attacked (10.7%). This isbecause malaria parasite (Plasmodium) can cause placental thickening and subsequent deposition of fibrins, which impaired nutrient and oxygen transport across the placenta. This is in keeping with earlier reports(Ismail *et al.,* 2000; Guyatt and Robert, 2004; Brabin *et al.,* 2004; Rachas *et al.,* 2012; Amal *etal.*, 2013; Ahmadu *et al.*, 2014). The prevalence of LBW deliveries was significantly higher (P = 0.001) in HIV infected mothers (22.9%) relative to mothers who were uninfected (11.7%). This agrees with earlier reports (Brocklehurst and Frend, 1998;Patil *et al.*, 2011; vander *et al.,* 2011; LeDoare *et al.*, 2012; Papp *et al*., 2015). Also, it was observed that LBW deliveries was significantly higher (P = 0.001) among diabetic mothers (14.1%) compared to non-diabetic mothers (12.4%). Diabetes mellitus can cause restricted fetal growth if it triggers pregnancy complications.This agrees with earlier reports (Persson *et al.*, 2009; Glinianaia*et al.*, 2012; klemetti *et al.*, 2012).The prevalence of LBW babies was observed to be significantly higher (P = 0.001) among hypotensive mothers (23.7%) compared to hypertensive mothers (13.9) and normotensive mothers (10.3%). This was observed by Yilgwan *et al.* (2012) in Jos, Nigeria.

The meanbirth weight (BW) of newborns in this study of3.09 kg for both sexes is close to what was observed in Makurdi, Nigeria of 3.08 kg(Swende, 2011). It was much higher than 2.89 kg reported in Nnewi, in Anambra State, Nigeria (Azubike, 1982). It was however lower than 3.16 kg (Lawoyin, 1991) in Ilorin, Nigeria, 3.43 kg (Barker *et al.*, 1990), 3.38 kg (Perry *et al.*, 1995) 3.24 kg (Premkumar *et al.*, 2016) and 3.40kg (Camerota and Bollen, 2016) reported in Ukraine, Western Europe, India and United States of America respectively.These differences in mean birth weight may be due to maternal genetics, altitude, nutrition, socioeconomic status and maternal diseases (Njokanma and Sule-Odu, 1998). In this study sex difference in birth weight (BW) showed statistical significant difference ($P < 0.001$), with male newborns having higher mean birth weight (3.15 kg) compared to female newborns (3.01 kg).This might be because boys grow faster than girls from an early stage of gestation, even from before implantation (Pedersen, 1980). Wilkins and Murphy, (2006)have suggested that gender-specific genes affecting insulin sensitivity are responsible for the gender difference in BW: females would be genetically less responsive to the trophic effect of insulin resulting in a LBW. Sex differencein BW was reported in a study in Indonesia (Jane *et al.,* 1989), in Central Nigeria (Lawoyin, 1991), Germany (Greil, 2006) Cuba (Martinez *et al.*, 2015),India(Premkumar *et al.*, 2016), and in the US (Camerota and Bollen, 2016).

The mean Placental weight observed in this study of 0.62 kg is higher than 0.59 kg (Abubakar *et al.,* 2012) observed in Sokoto, Nigeria, 0.58 kg (Perry *et al.,* 1995) and 0.47 kg (Little *et al.,* 2013) observed in Asia and Ukraine, respectively. It is however lower than 0.64 kg (Barker *et al.,* 1990) and 0.63 kg (Adinma and Agbai, 1995) reported in Western Europe and eastern Nigeria, respectively.However, the variations in the mean weight of the placenta may be due to variations in the methodology of preparing and weighing the placenta together with cord clamping time (Yao *et al.*, 1999). It has also been reported that ethnicity, birth weight and some unknown factors may affect the placental weight (Leary *et al.*, 2013).In this study sex difference in Placental weight (PW) of neonates wasstatistically significant ($P < 0.001$), male neonates (0.63 kg) had higher mean placental weight than female neonates (0.61 kg). This is in line with earlier reports (Barker *et al*., 1990; Perry *et al.,*1995; Fox *et al.*, 2002; Cunningham *et al.*, 2005; Abubakar *et al.*, 2012; Madkar *et al.*, 2015).

The mean maternal age in this study of26.79 years is lower than 27.58 years (Danborno *et al.*, 2008)observed in Zaria, Nigeria but higher than 25.5 years (Idris *et al.*, 2014) observed in Maiduguri, North East Nigeria. The mean gestational age in the study of 37.47 weeks was lower than 39.7 weeks (Burkhardt *et al.*, 2006), 39.9 weeks (Adinma and Agbai, 1995) and 38.8 weeks (Abubakar *et al.*, 2012) reported in western Europe, eastern Nigeria, and northern Nigeria respectively. The result in this study showed significant difference in gestational age of male and female newborns (P < 0.001), with male newborns having higher mean gestational age than female newborns. This is in line with previous reports (Adinma and Agbai, 1995; Burkhardt *et al.*, 2006; Martinez *et al.*, 2015). This study also observed statistical significant difference in other neonatal anthropometric parameters namely, birth length (BL), head circumference (HC) and chest circumference (CC) between the sexes. Male neonates had higher mean values relative to female neonates. This was also observed in Germany (Greil, 2006), Northern Nigeria (Abubakar *et al.*, 2012) and in Cuba (Martinez *et al.*, 2015).

Maternal level of education is shown to have significant effect on birth weight(BW) and placental weight (PW) of the neonates. The mean BW and PW of neonates in mothers with tertiary education were significantly higher than those in mothers with secondary, primary and no formal education (< 0.001) in the combined sample (male +female) neonates. Maternal education affects BW by improving the probability and /or productivity of health investment. Additionally, maternal education improves the financial resources available to the child directly and indirectly through the choice of partner, timing of fertility, and number of offspring (Chevalier and O’Sullivan, 2007). This finding is consistent with the report in Bangladesh(Karim and Mascie-Taylor, 1997), in Germany (Grimmer *et al.*, 2002), in England (Chevalier and

O'Sullivan, 2007) and Western Nigeria (Njokanma, 2013). In the female neonates group the same result was observed as with the combined sample of male and female neonates. While in the male neonates group, the mean birth weight of male neonates born to mothers with secondary and tertiary education was significantly higher compared to neonates born to mothers with primary or no formal education, (P < 0.01). The mean placental of male neonates born to mothers with primary, secondary and tertiary level of education was significantly higher than themean placental weight of neonates born mothers with no formal education, (P < 0.01). The mean birth length of babies born to mothers with tertiary education was significantly higher than the mean birth length of babies born to mothers with secondary, primary or no formal education, (P < 0.001) in the combined sample (male + female) babies. However in the separate groups (male babies only or female babies only), mean BL of male or female babies born to mothers with secondary and tertiary level of education was significantly higher than those born to mothers with primary or no formal education, (P < 0.001). This also agrees with earlier reports(Njokanma, 2013 and Chevalier and O'Sullivan, 2007). In this study it is also shown that the means of head and chest circumferences of babies born to mother with tertiary education is significantly higher compared to babies born to mothers with secondary, primary and no formal education, (P < 0.001) in the combined sample and in the female babies; while in the male babies, the mean of head and chest circumference of male babies born to mothers with primary, secondary and tertiary level of education was significantly higher than those born to mothers with no formal education, (P < 0.001). This is also reported by (Mumbare *et al.,* 2012).

This study showed that paternal level of education also have significant effect on neonatal anthropometry: birth weight (BW), placental weight (PW), birth length (BL), head circumference(HC) and chest circumference (CC). In this study, the mean birth and placental weight of babies with fathers with tertiary level of education were significantly higher compared to babies born to fathers with secondary, primary and no formal education, ($P < 0.001$) in the combine sample (male + female) and the same result was observed in the male babies and female babiessample.Disadvantaged socioeconomic status was associated with lower mean birth weight and other neonatal anthropometry measurements with maternal and paternal education and occupation exerting the higher influences. This was reported in the US (Chevalier and O'Sullivan, 2007) and in western Nigeria (Kehinde *et al.,* 2013). This study also showed that the mean BL, HC and CC of babies born to fathers with tertiary level of education was significantly higher than those in babies born to fathers with secondary, primary and no formal education, ($P < 0.001$) in the combined sample population (male + female) and separate groups (male babies only or female babies only). This agrees with earlier reports (kehinde *et al.*, 2013; Dahlui *et al.*, 2016), who reported that the mean of infant anthropometric parameters advances with parental education with parent with no formal education or least education giving birth to LBW babies.

In this survey the marital status was significantly associated neonatal anthropometry. The mean birth and placental weight of babies born to married mothers were significantly higher relative to those born to single mothers, ($P < 0.01$) in the combined sample (male + female) and in the separate groups (male babies only and female babies only).Marital status has been shown to correlate with intendedness of birth, economic status of mother, and social support of the mother and factors that may influence the health of the mother and infant (Chomitz *et al.*, 1995). The finding in this study agrees with previous reports (Shiono and Behrman, 1995; Guadino, 1993; Holt *et al.*, 1997; Bennett, 1992). The mean BL of babies born to married mothers was significantly higher than the mean BL of babies born to unmarried mothers in the combine sample (male + female) and in male babies, (P < 0.05) but no significant difference was observed between babies born to married and unmarried mothers in mean BL in the female babies group (P > 0.05).This study also observed no significant difference between the mean head circumferences of babies born to married and unmarried mothers in both the combined sample (male + female) and in the separate groups (male or female babies) with, P > 0.05. The mean chest circumference was observed to be significantly higher in babies born to married mothers compared to babies born to unmarried or single mother in both the combined sample (male and female) babies, male babies only and female babies only with, (P < 0.05). This report on differences in the neonatal anthropometric measurement between babies born to married mothers and single mothers has been reported by Chomitz *et al.* (1995) and Holt *et al.* (1997).

In this study the effect of diabetes mellitus during gestation was observed to be significantly associated with neonatal anthropometric parameters. The mean birth and placental weight of babies born to mothers with diabetes mellitus were significantly higher than the mean birth and placental weight of babies born to non-diabetic mothers in both the combined sample (male + female), male babies only and female babies only with (P < 0.001). This could mainly be attributed to high prevalence of macrosomic babies in diabetic pregnancies resulting from placental transfer of maternal glucose, leading to fetal hyperinsulinemia and accelerated fetal growth. However, despite the regulation of glucose in diabetic mothers, other nutritional substrates than glucose, such as lipids, probably also contribute to fetal overgrowth in pregnancies of women with diabetes (Kulkari *et al.*, 2013). This finding is consistent with earlier reports (Evers *et al.*, 2004; Jessen *et al.*, 2004; Persson *et al.*,2009; Kulkari *et al.*,2013).

The mean BL, HC and CC of babies born to mothers with diabetes mellitus were significantly higher than the mean BL, HC and CC of babies born to non-diabetic mothers in both the combined sample (male + female), and the separate groups (male or female babies) with statistical significant difference at, ($P < 0.001$). This was also reported byPersson *et al.* (2009) and Kulkari *et al.* (2013).

The effect of HIV infection in this survey was also observed to have significant influenced on neonatal weight and other neonatal anthropometric parameters. The mean birth and placental weight of babies born to HIV negative mothers were significantly higher than the mean birth and placental weight of babies born to HIV positive mothers in both the combined sample (male + female) and female babies ($P < 0.001$). However, in male babies, the mean birth weight of babies born to HIV negative mother was significantly higher than those born to HIV positive mothers ($P < 0.05$), but no significant difference was observed between their mean placental weight, ($P = 0.30$). The mean birth length, head and chest circumference of babies born to HIV negative mothers were significantly higher than the mean birth length, head and chest circumference of babies born to HIV positive mothers in the combined sample and among female babies ($P < 0.001$). However, there were no significant difference observed between the mean birth length, head and chest circumference of babies born to HIV negative and HIV positive mothers in the male babies, ($P > 0.05$).These differences observed between babies born HIV infected mothers and babies to uninfected mothers might be related to the damaged human immune system, especially the reduced CD4+ T cells and immunosuppression amongst HIV infected mothers which result in development of opportunistic infection in the reproductive tract.Some studies have reported that HIV-1 can replicate in the placenta resulting in placental insufficiency and restricted fetal growth (Kumar *et al.*, 2011).This might be another incentive of

LBW and preterm delivery. The finding in this study is consistent with previous reports (Moussa *et al.*,2001; Faye *et al.,* 2007; Kumar *et al.,* 2011; McGrath *et al.,* 2012;Xiao*et al.*,2015).

At least an episode of malarial attacked during pregnancy was observed to have significant impact on neonatal anthropometry. This current study observed the mean birth and placental weight of babies born to mothers who never had an episode of malarial attacked during pregnancy were significantly higher than in those babies born to mothers who had at least an episode in the combined sample (male + female babies), male babies and female babies ($P < 0.001$).This could be because low birth weight (LBW) related to placental malaria may be related to impaired nutrient transport to the foetus. High densities of malaria parasites in the placenta coupled with immune response may result in consumption of nutrient that would have been delivered to the foetus, leading to LBW. Also, the mean birth length, head and chest circumferenceof babies born to mothers who never had an episode of malarial attacked during pregnancy were significantly higher than those born to mothers who had at least an episode in the combined sample, male babies and females babies ($P < 0.001$). This is consistent with the reports of many researchers (Ismail *et al*., 2000; Steketee *et al.*, 2001; Guyatt and Robert, 2004; Scott *et al*., 2005; Ezebialu *et al.*, 2012; Amal *et al.*, 2013; Okolie *et al.*, 2014).

This current survey observed the effect of blood pressure on neonatal anthropometry with babies born to hypotensive mothers having significantly lower mean values compared to babies born to either normotensiveand hypertensive mothers in the combined sample (male + female babies) and in the separate groups, male babies and female babies, ($P <0.001$). This findings is consistent with report of Smal *et al.* (2009) and Yilgwan *et al.*, (2012).

Concerning the correlation matrix of the study population of both sexes, the results showed that birth weight (BW) of neonates or newborns showed strong positive correlation with maternal age (MA), parity, gestational age (GA), Apgar score 1 minute (AS 1min.), Apgar score 5 minute (AS 5min.), birth length (BL), head circumference (HC), chest circumference (CC), placental weight (PW), maternal height (MH), maternal weight (MW), gestational weight gain (GWG), maternal body mass index (BMI) and maternal packed cell volume (PCV). This strong positive correlation between BW and the aforementioned variables was also observed in both the male neonate correlation matrix and female neonate correlation matrix. This was previously reported (Hobcraft *et al.*, 1983;Ferraz *et al.*, 1988; Stark *et al.*, 1990; Grimmer *et al.*, 2002; Finster and Wood, 2005; Zhang *et al.*, 2007;Eltahir and Gerd, 2008;Abubakar *et al.*, 2012; Madkar *et al.*, 2015).

Concerning linear regression for both sexes, birth length, head circumference, chest circumference, gestational weight gain and gestational age appeared to be the best predictors of the birth weight of neonates with much higher coefficient of determination (R^2).

CHAPTER SIX

6.0 CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

In conclusion prevalence of low birth weight (LBW) and high birth weight (HBW) or macrosomic babies was 10.2 and 3.8 percent respectively. Spontaneous vaginal deliveries was more prevalent compare to caesarean section, vacuum assisted delivery and forceps assisted delivery. Low birth weight was significantly higher in female neonates compare to male neonate ($P = 0.001$). Low birth weight was more associated with teenage Mothers and older mothers ($\geq$ 40) compared to mothers in other age groups ($P = 0.001$). Low birth weight was more common among preterm neonates (< 37 completed weeks) compared to those born at term (37 completed weeks). Low birth weight neonates were significantly associated with Parents with no formal education compare to parents with primary, secondary and tertiary education at($P = 0.001$). Primiparous mothers had the highest incidence of LBW delivery compared to mothers with 1, 2, 3, 4 and above parity, ($P = 0.005$). Mothers who gained 12 kg and above during gestation delivered no LBW neonate and low birth weight neonates was more associated with mothers who gained less than 5 kg. Single or unmarried mothers delivered significantly higher LBW neonates compared to married mothers, mothers who had at least an episode of malarial attacked during the gestational period had significantly higher LBW babies relative to mothers who had no malaria infection, HIV infected mothers delivered significantly higher LBW babies compared to HIV negative mothers. Also, diabetic mother had significantly more LBW babies relative to non-diabetic mothers, and hypotensive mothers had significantly higher LBW babies compared to hypertensive mothers and normotensive mothers with , ($P = 0.001$).

Male neonates hadsignificantly higher anthropometric measurements (Birth weight, birth length, head circumference, chest circumference and placental weight) and gestational age than female newborns at ($p < 0.001$). This study also showed that parental education and marital status has significant effect on neonatal anthropometric parameters with those born to parent with tertiary education and to married mothershaving higher means. Maternal ill health hadnegative impact on neonatal anthropometric measurements.

Also, birth weight of neonates was observed to have strong positive correlation with maternal age, parity, gestational age, Apgar score (1min.), Apgar score (5min.), birth length, head circumference, chest circumference, placental weight, maternal height, maternal weight, maternal gestational weight gain, maternal BMI and maternal PCV (packed cell volume) at ($p< 0.001$) and also, neonatal chest circumference appeared to be the best predictor of birth weight.

6.2 RECOMMENDATIONS

Based on the findings of the present study, the following recommendations weremade:

i. appropriate gestational weight gainshould be encourage among pregnant mothers so as to reduce the risks of low birth weight deliveries.

ii. prophylactic measures should be encourage in pregnant mothers and maternal ill health should be closely monitored and treated adequately to reduce the risk of unfavourable birth outcomes .

iii. teenage pregnancy should be discourage.

iv. efforts should be put in place by Federal and State governments to ensure that girls are educated since maternal level of education is strongly associated with good birth outcomes.

6.3 CONTRIBUTIONS TO KNOWLEDGE

i. Predictive equation of birth weight using chest circumference is the best predictor of birth weight $BW = -2.753 + (0.176 * CC)$ $P<0.001$.

ii. Paternal level of education has stronger effect on the birth weight of newborns than maternal level of education (3.21 ± 0.49; 3.15 ± 0.50, $P<0.001$)

iii. Mothers of males newborns are significantly taller than mothers of female newborns (159.85 ± 6.14; 158.88 ± 5.89; $t= -3.876$, $P<0.001$).

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APPENDIX I

FEDERAL TEACHING HOSPITAL, GOMBE

Ashaka Road, Gombe
P.M.B. 0037

Tel: 072-223418,
223064
Fax:072-223909

Our Ref: NHREC/25/10/2013

Date: 17 September, 2015

Ahmed Hassan
Department of Human Anatomy,
Faculty of Medicine,
Ahmadu Bello University,
Zaria,
Kaduna State

Sir,

Ethical Clearance

I am directed to inform you that your application and proposal Titled: "**RETROSPECTIVE STUDY OF THE RELATIONSHIP BETWEEN INFANT BIRTH WEIGHT, APGAR SCORE, MATERNAL ANTHROPOMETRIC CHARACTERISTICS IN THE TWO TERTIARY HOSPITALS IN GOMBE (1995 – 2014).**" Submitted to the Hospital Research and Ethics Committee, have been duely reviewed and approved.

On behalf of the committee, I wish a successful execution.

Thank you.

B.A. Sambo Mrs. (Jp, CLN, ADL)
Secretary R&EC.

Chief Medical Director: Dr. Abubakar Sa'idu

HEALTH RESEARCH ETHICS COMMITTEE

AHMADU BELLO UNIVERSITY TEACHING HOSPITAL

SHIKA - ZARIA, NIGERIA.

E-mail: [illegible] website: [illegible]

Chairman of Board: Chief [illegible]
Chief Medical Director: Prof. Lawal Khalid, [illegible]
Chairman, Medical Advisory Committee: Prof. Abdullahi Mohammed, [illegible]
Director of Administration: Barr. [illegible]

ABUTH/HREC/TRG/36 — 18[th] April, 2016

Our Ref: ______ Date: ______

Your Ref: ______

ABUTH HREC FULL ETHICAL CLEARANCE CERTIFICATE

RE: *"Retrospective Study of the Relationship between Infant Birth Weight, Apgar score, and Maternal Anthropometric Characteristics in the Two Tertiary Hospitals in Gombe (2010-2014)"*

ABUTH Ethics Committee assigned number:	-	**ABUTH/HREC/ P 36 /2015**
Name of the principal investigator:	-	**Mr. Ahmed Hassan**
Address of the Principal investigator:	-	**Department of Human Anatomy**
		A.B.U-Zaria
Date of receipt of valid application:	-	**5/5/15**
Date of meeting when final determination		
On ethical approval was made:	-	**18/4/16**

This is to inform you that the research described in the submitted protocol, the consent forms, and other participant information materials have been reviewed and ***given full approval by the ABUTH Ethics Committee.***

Please note: this approval dates from ***18[th] April, 2016 to18th April, 2017.*** No participant recruitment into this research may be conducted outside these dates.

All informed consent forms in this study must carry the ABUTH HREC number assigned to this research and the duration of ABUTH HREC approval of the study.

This HREC expects that you submit your application as well as an annual report for ethical clearance renewal 3 months prior to expiration of study dates. This is to enable you obtain renewal of your approval and avoid interruption of your research.

If there is delay in starting the research, please inform the ABUTH HREC so that starting dates can be adjusted accordingly.

No changes are permitted in the research without prior approval by ABUTH HREC, except in circumstances outlined in national code for Health Research Ethics: http://www.nhrec.net.

ABUTH HREC reserves the right to conduct compliance assessment visits to your research site without prior notification.

Prof. Aisha I. Mamman, MBBS, FMCpath.
Chairman HREC.

APPENDIX II

Study Questionnaire

MATERNAL DEMOGRAPHICS

1. Age at the time of delivery______ (yrs)

2. Ethnic group__________________

3. State of origin_________________

4. Place of residence______________

5. Number of children (parity) _______

MATERNAL ANTHROPOMETRIC MEASUREMENTS

6. Gestational weight gain__________ (kg)

7. Weight at delivery_______________ (kg)

8. Height________________ (cm)

9. BMI________________ (kg/m^2)

10. Obesity______________ (kg)

HEALTH CONDITIONS

11. Cardiovascular disease (Hypertension) ____________

a. Yes [ ] b. No [ ]

12. HIV infection ______________

a. Yes [ ] b. No [ ]

13. Malaria ___________

a. Yes [ ] b. No [ ]

14. Gestational diabetes ____________

a. Yes [ ] b. No [ ]

15. STD(s) __________________

a. Yes [ ]   b. No [ ]

16. Haemoglobin concentration_____________ g/dl

MATERNAL AND PATERNAL SOCIOECONOMIC STATUS

17. Mother's occupation_________________________

18. Mother' level of education_____________________

19. Fathers occupation___________________________

20. Father's level of education_____________________

INFANT DEMOGRAPHICS

21. Gestational age_____________ (wks/months)

22. Sex__________

23. Apgar Score_____________

24. Mode of delivery____________

25. Number of siblings:

a. Brothers_________(from mother only)

b. Sisters__________ (from mother only)

26. What is infant birth order?

a. Firstborn [ ] b. Second born [ ] c. Third born [ ] d. Later born [ ]

ETHNIC BACKGROUND

27. Mother's Ethnic Group________

28. Father's Ethnic Group_________

INFANT ANTHROPOMETRIC PARAMETERS

29. Birth Weight________ (kg)

30. Birth Length_______ (cm)

31. BMI_________ (kg/m^2)

32. Head circumference______ (cm)

33. Chest circumference______ (cm)

APPPENDIX III

Tables for Males + Females newborns

Mode of delivery

	SVD	C/S	Vacuum	Forceps	F	P
Birth weight (kg)	3.07 ± 0.49^{a}	3.14 ± 0.65^{b}	3.27 ± 0.48^{c}	2.87 ± 0.64^{d}	24.338	<0.001
Birth length (cm)	50.06 ±3.59^{a}	50.16 ± 3.99^{b}	51.49 ± 3.41^{c}	49.69 ± 3.70^{a}	11.725	<0.001
Head circumference (cm)	34.12 ±2.20^{a}	34.72 ± 2.19^{b}	34.83 ± 2.04^{b}	33.79 ± 2.35^{c}	43.108	<0.001
Chest circumference (cm)	33.00 ±2.25^{a}	33.37 ±2.49^{b}	33.65 ± 1.84^{b}	32.49 ± 2.69^{c}	18.629	<0.001
Placental weight (kg)	0.61 ± 0.14^{a}	0.66 ± 0.18^{b}	0.67 ± 0.15^{b}	0.60 ± 0.19^{c}	47.270	<0.001

Maternal level of education

	Nil	Primary	Secondary	Tertiary	F	P
Birth weight (kg)	2.58 ± 0.59^{a}	2.87 ± 0.61^{b}	3.06 ± 0.53^{c}	3.15 ± 0.50^{d}	31.128	<0.001
Birth length	48.24 ± 3.93^{a}	49.26 ± 3.59^{b}	50.30 ± 3.17^{c}	50.71 ± 3.29^{d}	16.853	<0.001
Head circumference (cm)	33.36 ± 2.23^{a}	34.12 ± 2.27^{b}	34.40 ± 2.02^{b}	34.74 ± 2.11^{c}	12.416	<0.001
Chest circumference (cm)	31.54 ± 2.51^{a}	32.39 ± 2.69^{b}	33.02 ± 2.24^{c}	33.26 ± 2.17^{c}	15.661	<0.001
Placental weight (kg)	0.54 ± 0.12^{a}	0.61± 0.13^{b}	0.62 ± 0.13^{b}	0.64 ± 0.13^{c}	14.504	<0.001

Paternal level of education

	Nil	Primary	Secondary	Tertiary	F	P
Birth weight (kg)	2.91 ± 0.69^{a}	3.00 ± 0.63^{a}	2.97 ± 0.55^{a}	3.15 ± 0.49^{b}	23.711	<0.001
Birth length (cm)	49.76 ± 4.15^{a}	49.84 ± 3.74^{a}	49.79 ± 3.27^{a}	50.78 ± 3.09^{b}	14.849	<0.001
Head circumference (cm)	34.09 ± 1.98^{a}	34.12 ± 2.38^{a}	34.19 ± 2.10^{a}	34.72 ± 2.02^{b}	10.495	<0.001
Chest circumference (cm)	32.30 ± 2.87^{a}	32.72 ± 2.52^{a}	32.74 ± 2.36^{a}	33.31 ± 2.10^{b}	14.103	<0.001
Placental weight (kg)	0.60 ± 0.15^{a}	0.61 ± 0.14^{a}	0.61 ± 0.13^{a}	0.64 ± 0.13^{b}	12.887	<0.001

Marital status

	Married (n=2244)	Single (n=32)	t	P
Birth weight (kg)	3.08 ± 0.53	2.63 ± 0.33	4.68	< 0.001
Birth length (cm)	50.36 ± 3.29	48.53 ± 3.42	3.13	0.002
Head circumference (cm)	34.49 ± 2.27	33.84 ± 1.73	1.73	0.083
Chest circumference (cm)	33.05 ± 2.27	31.66 ± 1.96	3.46	< 0.001
Placental weight (kg)	0.63 ± 0.13	0.51 ± 0.08	4.79	< 0.001

Blood sugar

	Non-Diabetic	Diabetic	T	P
Birth weight (kg)	3.05 ± 0.51	3.67 ± 0.71	-10.370	<0.001
Birth length (cm)	50.26 ± 3.25	52.51 ± 3.69	-5.984	<0.001
Head circumference (cm)	34.43 ± 2.08	35.72 ± 1.80	-5.374	<0.001
Chest circumference (cm)	32.97 ± 2.25	34.79 ± 2.26	-7.042	<0.001
Placental weight (kg)	0.62 ± 0.13	0.72 ± 0.18	-6.221	<0.001

HIV infection

	HIV negative	HIV positive	T	P
Birth weight (kg)	3.08 ± 0.53	2.79 ± 0.49	5.103	<0.001
Birth length (cm)	50.39 ± 3.29	49.07 ± 3.08	3.855	<0.001
Head circumference (cm)	34.52 ± 2.09	33.58 ± 1.89	4.310	<0.001
Chest circumference (cm)	33.08 ± 2.26	31.95 ± 2.39	4.806	<0.001
Placental weight (kg)	0.63 ± 0.13	0.59 ± 0.12	3.059	0.002

Malarial episode

	Malaria absent	Malaria present	T	P
Birth weight (kg)	3.09 ± 0.53	2.82 ± 0.52	7.894	<0.001
Birth length (cm)	50.45 ± 3.26	49.46 ± 3.39	4.482	<0.001
Head circumference (cm)	34.57 ± 2.03	33.73 ± 2.37	6.073	<0.001
Chest circumference (cm)	33.12 ± 2.25	32.34 ± 2.34	5.143	<0.001
Placental weight (kg)	0.63 ± 0.14	0.58 ± 0.12	5.748	<0.001

Blood pressure

	Normotensive	Hypertensive	Hypotensive	F	P
Birth weight (kg)	3.09 ± 0.49[a]	3.18 ± 0.74[b]	2.79 ± 0.55[c]	39.997	<0.001
Birth length (cm)	50.42 ± 3.13[a]	50.68 ± 3.73[a]	49.37 ± 3.83[b]	12.531	<0.001
Head circumference (cm)	34.59 ± 1.92[a]	34.51 ± 2.58[a]	33.59 ± 2.49[b]	24.910	<0.001
Chest circumference (cm)	33.13 ± 2.12[a]	33.22 ± 2.89[a]	32.11 ± 2.41[b]	22.946	<0.001
Placental weight (kg)	0.63 ± 0.13[a]	0.65 ± 0.16[a]	0.59 ± 0.13[b]	14.361	<0.001

Tables for male newborns only

Mode of delivery

	SVD	C/S	Vacuum	Forceps	F	P
Birth weight (kg)	3.13 ± 0.49[a]	3.21 ± 0.66[b]	3.33 ± 0.46[c]	3.01 ± 0.52[d]	15.727	<0.001
Birth length (cm)	50.48 ± 3.39[a]	50.56 ± 3.88[a]	51.69 ± 3.75[b]	50.08 ± 3.08[a]	5.458	<0.001
Head circumference (cm)	34.31 ± 2.14[a]	34.90 ± 2.23[a]	34.78 ± 2.04[a]	34.00 ± 2.14[b]	21.806	<0.001
Chest circumference (cm)	33.22 ± 2.16[a]	33.59 ± 2.46[a]	33.74 ± 1.74[a]	33.04 ± 2.18[b]	9.733	<0.001
Placental weight (kg)	0.62 ± 0.14[a]	0.67 ± 0.19[b]	0.67 ± 0.15[b]	0.63 ± 0.21[c]	28.196	<0.001

Maternal level of education

	Nil	Primary	Secondary	Tertiary	F	P
Birth weight (kg)	2.73 ± 0.62[a]	2.98 ± 0.69[b]	3.16 ± 0.52[c]	3.19 ± 0.51[c]	11.467	<0.001
Birth length	48.86 ± 4.07[a]	49.69 ± 4.13[b]	50.77 ± 3.01[c]	50.99 ± 3.32[c]	7.659	<0.001
Head circumference (cm)	33.50 ± 2.21[a]	34.59 ± 2.44[b]	34.67 ± 2.11[b]	34.81 ± 2.26[b]	4.644	0.003
Chest circumference (cm)	31.57 ± 2.52[a]	33.00 ± 3.02[b]	33.33 ± 2.18[b]	33.28 ± 2.18[b]	8.322	<0.001
Placental weight (kg)	0.57 ± 0.11[a]	0.63 ± 0.16[b]	0.63 ± 0.14[b]	0.65 ± 0.14[b]	5.339	0.001

Paternal level of education

	Nil	Primary	Secondary	Tertiary	F	P
Birth weight (kg)	2.97 ± 0.68[a]	3.13 ± 0.66[b]	3.07 ± 0.56[c]	3.21 ± 0.49[d]	8.311	<0.001
Birth length (cm)	50.08 ± 4.27[a]	50.35 ± 3.54[a]	50.18 ± 3.21[a]	51.14 ± 3.08[b]	7.576	<0.001
Head circumference (cm)	34.19 ± 1.85[a]	34.44 ± 2.62[a]	34.49 ± 2.18[a]	34.84 ± 2.17[b]	2.968	0.019
Chest circumference (cm)	32.48 ± 2.90[a]	33.06 ± 2.61[b]	33.15 ± 2.32[b]	33.38 ± 2.11[b]	4.483	0.001
Placental weight (kg)	0.59 ± 0.15[a]	0.62 ± 0.15[b]	0.63 ± 0.13[c]	0.65 ± 0.14[d]	6.095	<0.001

Marital status

	Married (n=1151)	Single (n=18)	T	P
Birth weight (kg)	3.15 ± 0.54	2.69 ± 0.29	3.63	< 0.001
Birth length (cm)	50.77 ± 3.24	48.78 ± 3.80	2.58	0.010
Head circumference (cm)	34.69 ± 2.20	33.89 ± 2.03	1.54	0.125
Chest circumference (cm)	33.25 ± 2.25	31.67 ± 2.25	2.96	0.003
Placental weight (kg)	0.64 ± 0.14	0.53 ± 0.07	3.36	< 0.001

HIV infection

	HIV negative	HIV positive	T	P
Birth weight (kg)	3.15 ± 0.54	2.96 ± 0.39	2.131	0.033
Birth length (cm)	50.76 ± 3.27	49.92 ± 2.81	1.553	0.121
Head circumference (cm)	34.69 ± 2.22	34.24 ± 1.54	1.224	0.221
Chest circumference (cm)	33.25 ± 2.27	32.62 ± 1.79	1.661	0.097
Placental weight (kg)	0.64 ± 0.14	0.61 ± 0.09	1.032	0.302

Blood sugar

	Non-Diabetic	Diabetic	T	P
Birth weight (kg)	3.12 ± 0.51	3.74 ± 0.76	-7.899	<0.001
Birth length (cm)	50.64 ± 3.21	53.02 ± 3.45	-4.906	<0.001
Head circumference (cm)	34.62 ± 2.19	36.02 ± 1.87	-4.259	<0.001
Chest circumference (cm)	33.16 ± 2.22	35.00 ± 2.54	-5.491	<0.001
Placental weight (kg)	0.63 ± 0.14	0.73 ± 0.18	-4.914	<0.001

Malarial episode

	Malaria absent	Malaria present	T	P
Birth weight (kg)	3.17 ± 0.54	2.92 ± 0.50	5.233	<0.001
Birth length (cm)	50.81 ± 3.23	50.15 ± 3.43	2.168	0.030
Head circumference (cm)	34.78 ± 2.12	33.85 ± 2.64	4.618	<0.001
Chest circumference (cm)	33.31 ± 2.24	32.55 ± 2.34	3.628	<0.001
Placental weight (kg)	0.64 ± 0.14	0.59 ± 0.11	3.949	<0.001

Blood pressure

	Normotensive	Hypertensive	Hypotensive	F	P
Birth weight (kg)	3.17 ± 0.47^{a}	3.25 ± 0.82^{a}	2.87 ± 0.56^{b}	19.710	<0.001
Birth length (cm)	50.84 ± 2.96^{a}	50.93 ± 3.91^{a}	49.69 ± 4.27^{b}	6.975	<0.001
Head circumference (cm)	34.83 ± 1.92^{a}	34.58 ± 3.03^{a}	33.64 ± 2.76^{b}	15.964	<0.001
Chest circumference (cm)	33.36 ± 1.98^{a}	33.28 ± 3.26^{a}	32.13 ± 2.57^{b}	15.997	<0.001
Placental weight (kg)	0.64 ± 0.13^{a}	0.67 ± 0.18^{b}	0.59 ± 0.15^{c}	9.140	<0.001

Tables For female newborns only

Mode of delivery

	SVD	C/S	Vacuum	Forceps	F	P
Birth weight (kg)	2.99 ± 0.49[a]	3.03 ± 0.62[a]	3.17 ± 0.49[b]	2.65 ± 0.75[c]	8.327	<0.001
Birth length (cm)	49.61 ± 3.73[a]	49.63 ± 4.07[a]	51.20 ± 2.82[b]	49.12 ± 4.53[a]	5.508	<0.001
Head circumference (cm)	33.91 ± 2.25[a]	34.48 ± 2.13[b]	34.90 ± 2.05[b]	33.47 ± 2.67[a]	19.890	<0.001
Chest circumference (cm)	32.77 ± 2.32[a]	33.09 ± 2.53[b]	33.52 ± 1.97[b]	31.65 ± 3.20[c]	8.016	<0.001
Placental weight (kg)	0.60 ± 0.13[a]	0.64 ± 0.16[b]	0.68 ± 0.15[c]	0.56 ± 0.16[d]	19.095	<0.001

Maternal level of education

	Nil	Primary	Secondary	Tertiary	F	P
Birth weight (kg)	2.33 ± 0.44[a]	2.74 ± 0.48[b]	2.97 ± 0.51[c]	3.10 ± 0.49[d]	25.118	<0.001
Birth length (cm)	47.20 ± 3.51[a]	48.79 ± 2.89[b]	49.85 ± 3.25[c]	50.37 ± 3.24[c]	10.596	<0.001
Head circumference (cm)	33.12 ± 2.28[a]	33.62 ± 1.97[b]	34.14 ± 1.89[b]	34.65 ± 1.91[c]	10.839	<0.001
Chest circumference (cm)	31.48 ± 2.55[a]	31.74 ± 2.13[a]	32.73 ± 2.26[b]	33.24 ± 2.18[c]	11.992	<0.001
Placental weight (kg)	0.49 ± 0.12[a]	0.60 ± 0.09[b]	0.61 ± 0.13[c]	0.63 ± 0.12[d]	10.982	<0.001

Paternal level of education

	Nil	Primary	Secondary	Tertiary	F	P
Birth weight (kg)	2.84 ± 0.72[a]	2.89 ± 0.59[a]	2.87 ± 0.51[a]	3.09 ± 0.47[b]	16.127	<0.001
Birth length (cm)	49.39 ± 4.04[a]	49.38 ± 3.88[a]	49.45 ± 3.29[a]	50.36 ± 3.07[b]	6.435	<0.001
Head circumference (cm)	33.98 ± 2.13[a]	33.82 ± 2.12[a]	33.92 ± 1.99[a]	34.57 ± 1.83[b]	8.453	<0.001
Chest circumference(cm)	32.09 ± 2.85[a]	32.41 ± 2.40[a]	32.38 ± 2.34[a]	33.24 ± 2.09[b]	11.655	<0.001
Placental weight (kg)	0.61 ± 0.15[a]	0.59 ± 0.12[b]	0.59 ± 0.13[b]	0.63 ± 0.12[c]	7.212	<0.001

Marital status

	Married (n=1093)	Single (n=14)	T	P
Birth weight (kg)	2.99 ± 0.52	2.56 ± 0.36	3.16	0.002
Birth length (cm)	49.93 ± 3.28	48.21 ± 2.81	1.95	0.051
Head circumference (cm)	34.27 ± 1.95	33.79 ± 1.31	0.93	0.351
Chest circumference (cm)	32.84 ± 2.27	31.64 ± 1.59	1.97	0.049
Placental weight (kg)	0.62 ± 0.13	0.49 ± 0.09	3.52	< 0.001

HIV infection

	HIV negative	HIV positive	T	P
Birth weight (kg)	3.00 ± 0.51	2.69 ± 0.51	4.479	<0.001
Birth length (cm)	49.99 ± 3.27	48.54 ± 3.14	3.318	<0.001
Head circumference (cm)	34.33 ± 1.92	33.17 ± 1.99	4.505	<0.001
Chest circumference (cm)	32.90 ± 2.22	31.53 ± 2.62	4.575	<0.001
Placental weight (kg)	0.62 ± 0.13	0.57 ± 0.14	2.973	0.003

Blood sugar

	Non-Diabetic	Diabetic	T	P
Birth weight (kg)	2.97 ± 0.50	3.57 ± 0.64	-6.531	<0.001
Birth length (cm)	49.86 ± 3.25	51.78 ± 3.93	-3.279	0.001
Head circumference (cm)	34.24 ± 1.94	35.28 ± 1.63	-3.015	0.003
Chest circumference (cm)	32.78 ± 2.26	34.50 ± 1.78	-4.268	<0.001
Placental weight (kg)	0.61 ± 0.12	0.69 ± 0.18	-3.601	<0.001

Malarial episode

	Malaria absent	Malaria present	T	P
Birth weight (kg)	3.02 ± 0.51	2.72 ± 0.52	6.145	<0.001
Birth length (cm)	50.06 ± 3.26	48.71 ± 3.22	4.300	<0.001
Head circumference (cm)	34.35 ± 1.91	33.60 ± 2.05	4.015	<0.001
Chest circumference (cm)	32.92 ± 2.25	32.11 ± 2.33	3.701	<0.001
Placental weight (kg)	0.62 ± 0.13	0.57 ± 0.13	4.261	<0.001

Blood pressure

	Normotensive	Hypertensive	Hypotensive	F	P
Birth weight (kg)	3.01 ± 0.49[a]	3.09 ± 0.61[a]	2.72 ± 0.54[b]	19.814	<0.001
Birth length (cm)	49.98 ± 3.23[a]	50.36 ± 3.48[a]	49.05 ± 3.34[b]	5.464	0.004
Head circumference (cm)	34.35 ± 1.89[a]	34.43 ± 1.89[a]	33.55 ± 2.21[b]	9.609	<0.001
Chest circumference (cm)	32.89 ± 2.24[a]	33.14 ± 2.38[a]	32.08 ± 2.26[b]	8.082	<0.001
Placental weight (kg)	0.62 ± 0.13[a]	0.62 ± 0.14[a]	0.58 ± 0.12[b]	6.404	0.002

APPENDIX IV

BW = Birth weight

PW = Placental weight

AS = Apgar score

BL = Birth length

CC = chest circumference

MS = Marital status

MLE = Maternal level of education

PLE = Paternal level of education

MH = Maternal height

MW = Maternal weight

GWG = Gestational weight gain

BMI = Body mass index

MA = Maternal age

P = Parity

GA = Gestational age

HI = HIV infection

DM = Diabetes mellitus

MI = Malaria infection

BP = Blood pressure

SBP = Systolic blood pressure

DBP= Diastolic blood pressure

PCV = Packed cell volume