ASSESSING THE RISK FACTORS ASSOCIATED WITH LOW BIRTH WEIGHT (LBW) AND MEAN ACTUAL BIRTH WEIGHT OF NEONATES: A CASE STUDY OF

ST. MARTIN'S HOSPITAL, AGROYESUM.

By

ANTWI – BOASIAKO ISHMAEL

(BSC. COMPUTER SCIENCE)

A Thesis submitted to the Department of Mathematics, Kwame Nkrumah University of Science and Technology, Kumasi

in partial fulfilment of the requirement for the degree of

Master of Science

Industrial Mathematics

INSTITUTE OF DISTANCE LEARNING

October, 2011

DECLARATION

I, hereby declare that this submission is my own work towards the award of MSc and that, to the best of my knowledge, it contains no materials previously published by another person nor material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text.

Antwi-Boasiako Ishmael (PG3009009)	……	……
Student's Name and ID	Signature	Date

Certified by:

Nana Kena Frempong	……	……
Supervisor's Name	Signature	Date
K. F. Darkwah	……..	……
Head of Department's Name	Signature	Date
Prof. I. K. Dontwi	…….	……
(Dean of Institute of Distance Learning's Name)	Signature	Date

DEDICATION

This work is dedicated to the Almighty God who is my source of wisdom, knowledge and power.

ABSTRACT

A study on some selected socio-biological and demographic determinants of mean actual birth weight and Low Birth Weight (LBW) was conducted in St Martins Hospital, Agroyesum, Ghana. Records of 221 single live births over a period of one year (1st January to 31st December 2010) were analysed.

The study was aimed at gaining understanding on the determinants of LBW and mean Actual Birth weight in the study area. The data were analyzed using SAS package. Predictors of LBW were assessed through logistic regression analysis and probit regression analysis whilst that of mean actual birth weight was also assessed through multiple linear regression analysis. The factors which were significant for LBW were gestation age, age of the mother and weight gain during pregnancy and that of mean actual birth weight was gestation age whilst occupation, sex of the infant and hypertension were not significant for LBW.

The results from the three models indicate that gestation age was the predictor that has an influence on birth outcome. We also conclude from the probit and logit models that gestation age, mother's age and weight of the mother at last menstrual period(MWT) are risk factors of low birth weight.

ACKNOWLEDGEMENT

I am grateful to the Almighty God for He is my source of inspiration.

I wish to express my profound gratitude to Nana Kena Frempong, my supervisor, whose contributions towards the completion of this work are immeasurable.

Finally, I am grateful to my parents Mad. Adjei Rose and Mr. R. K. Otchere, my Uncle, Owusu Adjei, Clara Ahene-Affoh and Rebecca Manu for the encouragement and assistance given to me when this project was being carried out.

TABLE OF CONTENTS

CONTENT **PAGE**

CHAPTER TWO:REVIEW OF LITERATURE

CHAPTER THREE: METHODOLOGY

LIST OF TABLES

LIST OF FIGURES

LIST OF ACRONYMS/ABBREVIATIONS

ART……..Artificial Reproductive Technology

ACC/SCN…Administrative Committee on Coordination/Subcommittee on Nutrition

ALRI ……...Acute Lower Respiratory Infections

ANOVA…..Analysis of Variance

BPD…….Brochopulmonary Dysplasis

BMI ……Body Mass Index

CHD……Coronary Heart Disease

CI……Confidence Interval

ELBW…….Extremely Low Birth Weight

EUS…….Endoscopic ultrasound

EVS……..EUROPEAN VOLUNTARY SERVICE

IGT……..Impaired Glucose Tolerance

HIV/AIDS.. Human_immunodeficiency_virus /Acquired immune deficiency syndrome

IMR…….Infant Mortality Rates

IUGR……...Intra-Uterine Growth Restriction

IVR……..In Vitro Fertilization

LBW……Low Birth Weight

LMP…….Last Menstrual Period

MAR……Medical Assisted Reproduction

MDG……Millennium Development Goal

MLE…….Maximum Likelihood Estimation

MRC…….Medical Research Council

NICU……Neonatal Intensive Care Unit

NIDDM…….Non-insulin Dependent Diabetes Mellitus

OR……Odd Ratio

PID…….. .Pelvic Inflammatory Disease

PTB……...Preterm Birth

SGA……. .Small for Gestational Age

SMA……. …..Society of African Missions

SAS…… ….Statistical Package for Social Sciences

UNICEF…..**..**United Nations Children's Fund

VLBW……Very Low Birth Weight

WHO……..World Health Organization

CHAPTER ONE

1.0 INTRODUCTION TO THE STUDY

The issue concerning post maternal outcomes is a concern to the World Health Organisation (WHO). One of the issues of birth outcomes is low birth weight (LBW). This is defined as weight at birth less than 2500g (5.5pounds). Low birth weight (LBW) is one of the major predictors of neonatal and perinatal morbidity and mortality both in developed and developing countries. It is believed that the better a population socioeconomic development, the better its health indicators, including LBW. That is LBW is a major factor contributing towards high infant mortality in developing countries. The proportion of infants with low birth weight reflects the socio-economic development of any region or country (Murthy, 1991).

This is based on epidemiological observations that infants weighing less than 2,500 g are approximately 20 times more likely to die than heavier infants (UNICEF- WHO, 2004). LBW is more common in developing than developed countries; a birth weight below 2,500 g contributes to a range of poor health outcomes. However, lower LBW rate of populations with lower socioeconomic levels has been observed compared to populations with better indicators. This is the so-called epidemiological paradox of LBW. LBW is mostly common in developing countries, where the burden of malnutrition and infectious diseases is heavy, and the incidence is estimated to be more than twice that of developed countries (Dreyfuss *et al.,* 2001). In sub-Saharan Africa, estimated rate of LBW is 14 per 100 live birth based on statistics derived from health facilities, which constitute about 35% of all live births occurring in the region. Similarly, based on health facility statistics of 1999, which included only 45% of infants weighed at birth, incidence rate of LBW in Tanzania was 13 per 100 live births (UNICEF- WHO, 2004).

1.1 BACKGROUND OF THE STUDY

The cohort of LBW (birthweight <2.5 kg) infants is likely to reflect two effects, namely a short gestational age (preterm births) and small for gestational age (SGA). Small for gestational age usually results from intra-uterine growth restriction (IUGR). However, if the mother is small, it may be normal for her to have a small fetus. In the current study gestational age is included as an explanatory variable; therefore the study is focused on the identification of risk factors for the complementary effect which is SGA.

In a literature survey, de Onis et al (1998) found that IUGR infants are at increased risk of perinatal mortality and morbidity, i.e. sudden infant death syndrome, poor cognitive development and neurologic impairment, cardiovascular disease, high blood pressure, obstructive lung disease, diabetes, high cholesterol concentrations and renal damage in adulthood. Such infants remain a burden on government expense in developed countries and a permanent problem for their families in developing countries.

The incidence of LBW (<2.5 kg irrespective of gestational age) is estimated to be 16% worldwide, 19% in the least developed and developing countries and 7% in the developed countries. The incidence of LBW is 31% in South Asia followed by Middle East and North Africa (15%), Sub-Saharan Africa (14%) and East Asia and Pacific 7%. Of the total estimated IUGR infants (<2.5 kg and = >37 weeks), Asia accounts for 75%, and with 20% and 5% born in Africa and Latin America, respectively. UNICEF estimates show that in Ghana, the percentage of low birth weight infants is 9, adjusting for both mother's assessment and heaping on 2500g, yields higher estimates of the incidence of low birth weight (UNICEF- WHO, 2004). The IUGR accounts for 11% of the total

infants in developing countries ranging from 2% to 21%, that is 6 times higher compared to developed countries.

The diversity in area-status (Tribal/Settled), and differential in overall geo-demographics factors suggest a need to investigate LBW and mean birth weight in the area. The current study derives an explanatory multivariate regression model for both LBW and actual birth weight that includes gestational age as an predictor covariate. The remaining selected covariates are thus interpreted as explanatory of incidence of LBW and actual birth weight under the category of SGA. Therefore, this prospective public hospital-based study in Amansie West focuses on LBW and Actual birth weight to investigate associated explanatory factors. These factors contribute towards the explanation of the observed births that are SGA.

1.2 STATEMENT OF THE PROBLEM

In Ghana, the issue of birthweight and factors influencing it has not received the much needed attention. This should not be the case because birthweight is a strong predictor of an individual baby's survival and a person's personality. The high rate of low birthweight (LBW), defined as weight at birth less than 2.5kg is a major problem in developing countries. From 1998 to 2004, Ghana recorded higher LBW cases of 16% compared to the average of 14% for sub-Saharan Africa (UNICEF,2004). Cases of low birthweight are more common in the developing countries than in the developed countries.

Prior to this study, Amansie West District was challenged with pregnancy outcomes as data at St Martin's Hospital over the years recorded a high number of infants born with LBW.

It is therefore imperative to identify risk factors for LBW in district in order to come up with feasible intervention strategies to minimize the problem.

1.3 OBJECTIVES

The main objectives of this study are:

1. To identify risk factors associated with probability of giving birth to a low birth weight using logistic and Probit regression models.

2. To model the effects on the mean actual birth weight.

1.4 METHODOLOGY:

The study was carried out from October1, 2010 to December 2010. The inclusion criteria were all infants with actual birth weight measured at the Neonatal Intensive Care Unit (NICU) of St Martin's Hospital from January 2010 to December 2010.

Data for the study were sourced from the birth registers available in the hospital. For this purpose, the cases of births over a year i.e. from 1st January 2010 to 31st December 2010 were taken into consideration. Since the data were recorded primarily for the use of the hospital, these data may be regarded as secondary data. The data consist of 221 observations. Incomplete records and multiple births were excluded.

The data included variables such as prenatal demographic details, maternal pregnancy history and antenatal care, details of the delivery, the infant's status at delivery and outcome at discharge. Maternal attributes included maternal age, maternal hypertension status and the maternal weight. Labor employment history, delivery, and newborn characteristics were: gestational age (determined according to at least 2 of the following parameters: last menstrual period) and birth weight.

The data was analysed using the Statistical Analysis System (SAS). Chi-square test was used to test independence of the categorical variables. Linear regression analysis model was used to test the effects of risk factors associated with mean Actual Birth weight and

also Multiple Logistic regression analysis and Probit regression analysis were used to determine the risk factors associated with Low Birth Weight. *P*-values of less than 0.05 and 0.1 were considered statistically significant because of the nature of the data.

Regression methods are essential to any data analysis which attempts to describe the relationship between a response variable and any number of predictor variables. Frequently, situations involving discrete variables arise. Logistic regression analysis and probit regression analysis extend the techniques of multiple linear regression analysis to research situations in which the outcome variable is categorical, that is, taking on two or more possible values.

1.5 JUSTIFICATION OF THE STUDY

Birth weight is a strong predictor of infant growth and survival. Infants born with low birth weights begin life immediately disadvantaged and face extremely poor survival rates. In most developing countries it was approximated that every ten seconds an infant dies from a disease or infection that can be attributed to low birth weight.

The reduction of the incidence of low birth weight also forms an important component of the Millennium Development Goals (MDGs) on child health. Activities towards the achievement of the MDGs will need to ensure a healthy start in life by making certain that women commence pregnancy healthy and well nourished, and go through pregnancy and childbirth safely. Low birth weight is, therefore, an important indicator for monitoring progress towards these internationally agreed-upon goals.

Earlier works stated the birth weight of infants in Ghana ranged from 2.00 to 3.00 kg.

It is in this light that this research seeks to investigate the effect of some of the factors that affect birth weight in the Amansie West District, one of the poorest districts in Ghana.

It is hoped that the results of this study would help to inform the health authorities about the local risk factors for LBW and actual birth weight in order to introduce programmes to reduce the prevalence of low birth weight.

1.6 LIMITATIONS

As the research was completed in a relatively short period of time other factors and variables such as multiple birth, parity etc. were not considered. This might have an impact on the results of the study.

In spite of these limitations, the findings of the study constitute a strong basis for generalisation.

1.7 ORGANIZATION OF THE STUDY

The study has been organised into five chapters. Chapter one which is the introduction deals with the background to the study, statement of the problem, purpose of the study, justification of the study, limitations of the study and organisation of the study.

Chapter two discusses the literature related to the study. The review involves theoretical and empirical studies related to the problem under study.

The third chapter describes the methodology used in the study. Specifically, the research design, the research instrument, the procedure for data collection and the data analysis are discussed.

In chapter four, the main focus is the analysis of the data and the presentation of the results.

Finally, Discussion of the results, conclusions and recommendations are presented in chapter five.

CHAPTER TWO

REVIEW OF LITERATURE

2.0 INTRODUCTION

The review, which involves both theoretical and empirical approaches, presents a broad overview about the literature related to the study.

2.1 DEFINITION OF LOW BIRTH WEIGHT

Birth weight is the first weight of the foetus or newborn obtained after birth. For live births, birth weight should preferably be measured within the first hour of life before significant postnatal weight loss has occurred.

Low birth weight (LBW) is defined as the weight of liveborn infants less than 2,500 g, for a given time period. LBW is closely associated with fetal and neonatal mortality and morbidity, inhibited growth and cognitive development, and chronic diseases later in life. At the population level, the proportion of infants with a LBW is an indicator of a multifaceted public-health problem that includes long-term maternal malnutrition, ill health, hard work and poor health care in pregnancy. On an individual basis, LBW is an important predictor of newborn health and survival and is associated with higher risk of infant and childhood mortality (Stevens-Simon and Orleans, 1999).

The WHO 2,500 grams criterion for the definition of low birth weight mentioned at the beginning of the paper is quite arbitrary. There exists a significant relationship between the mean height of mothers in a population and the mean birth weight of children in that

same population. Therefore, if the standard WHO criterion is applied, populations with shorter women will almost by definition have a higher proportion of low birth weight. In those populations, the 'optimal' birth weight, defined as the birth weight at which mortality is lowest, is different from the European one. This is noticeable when one compares for instance countries like Portugal, Italy, Spain and Great Britain, where the average maternal height is between 1.61 and 1.63 meters, with countries like Denmark, the Netherlands and Germany, where the average maternal height is between 1.67 and 1.69 meters (Lack et al. 2003). The relationship is also noticeable when comparing Europe as a whole with non-European populations. The study of Vangen et al. (2002), which compared the birth weight and perinatal mortality of Vietnamese, Pakistani, Norwegian and North African infants, found that mean birth weights were lower for Vietnamese and Pakistani children and higher for Norwegian and North African mothers.

Another comment relates to the fact that, for quite a long time, low birth weight and premature birth (defined as a gestational age of less than 37 weeks - Kramer, 1987) were used as synonyms. It has now been recognised, however, that not all premature births result in low birth weight and that not all low birth weights result from premature births. In the latter case, the terms intrauterine growth retardation (IUGR) or small-for-gestational age (SGA) are used - defined as "birth weight less than 10th (or 5th) percentile for gestational age; birth weight less than 2,500g and gestational age greater than or equal 37 weeks; and birth weight less than 2 standard deviations below the mean value for gestational age" (Kramer, 1987). The fact that a distinction is made between SGA and other low birth weight infants already points to differences in health consequences for the two groups.

The reason why it has become possible to make this distinction more precisely is that the measurement of gestational age, while still not perfect, has greatly improved recently,

with an increased use of both the last menstrual period approach (LMP - self-reported) and ultra-sound techniques (Foix-L'Helias and Blondel, 2000). North African mothers have fewer children with low birth weight when they migrate. This is considered to be an epidemiological paradox.

Yang et al. (2002) note that gestational ages measured through an Endoscopic Ultrasound Scan (EUS-scan) are lower on average than those measured via the LMP-method, resulting in a higher rate of preterm birth.

Low birth weight can be caused either by premature delivery (short gestation) or by foetal growth retardation. Known factors for pre-term delivery and foetal growth retardation which are associated with LBW include low maternal food intake, hard physical work during pregnancy, and illness, especially infections (Klingenberg *et al.* 2003).

Low birthweight (LBW) relates to intra-uterine growth retardation (IUGR) and preterm birth (PTB) which are two distinct processes with differing etiology and consequences. LBW is defined by WHO as a birthweight less than 2500g. IUGR has no generally accepted standard definition, but the following are commonly used: birthweight less than 10th percentile for gestational age; birthweight less than 2500g and gestational age greater than 37 weeks; and birthweight less than 2 standard deviations below the mean value for gestational age. PTB is defined as gestational age less than 37 weeks at delivery. It is important to bear in mind that only a proportion of infants with IUGR or PTB will be classified as having LBW as shown in Figure 1.1.

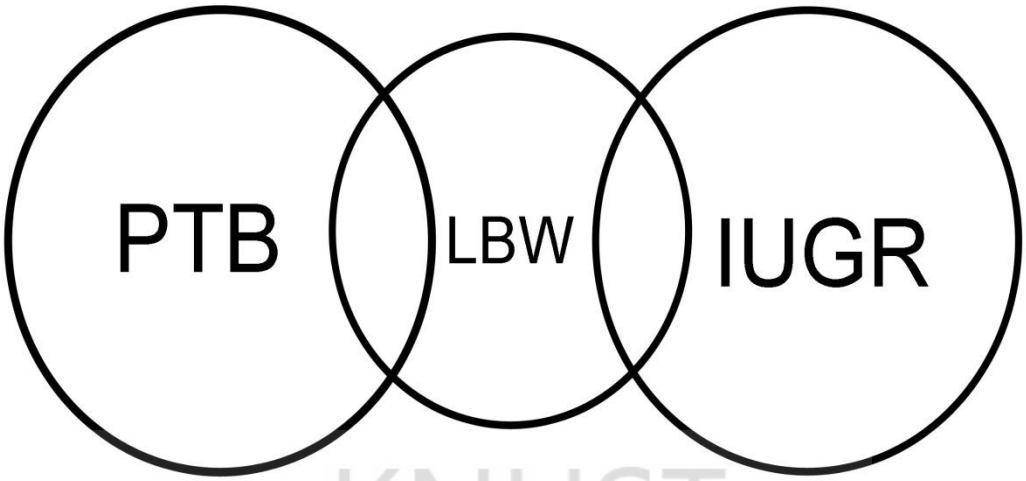

Figure 1.1 Relationships between PTB, LBW AND IUGR

Subtypes of IUGR are described according to whether restriction in growth relates more to weight or length of the fetus. Infants who are less than 10th percentile for both weight and ponderal index are classified as 'asymmetric' IUGR. Infants who are lessthan10th percentile for weight but not ponderal index (PI) are classified as'symmetric' IUGR.

The existence of subtypes of IUGR has led to inferences about the timing of growth restriction, based on Tanner's schematic curves of fetal and neonatal length growth which suggested that the velocity of growth in length was greater during early pregnancy. This had led to the view that symmetric IUGR is related to earlier and more persistent impairment of growth. However real data on fetal length growth suggest that growth in length during pregnancy is linear. A more accurate interpretation of the different subtypes of IUGR may therefore be that 'asymmetric'IUGR reflects more severe degrees of growth restriction.

PTB and IUGR are associated with early mortality and morbidity and with adverse long-term outcomes. The long-term sequelae of PTB include: Neurological complications such as periventricular leucomalacia, Cerebral Palsy, Seizures, delayed development, and learning difficulties: Pulmonary outcomes such as bronchopulmonary dysplasia, recurrent wheezing with respiratory infection: Opthalmologic complications such as retinopathy

and blindness. The long-term sequelae of IUGR include small permanent deficits in weight and length, mild neurocognitive deficits and increased risk of hypertension, coronary heart disease and diabetes in adult life.

2.2.1 LEVELS, PATTERNS AND DETERMINANTS OF LBW IN DEVELOPING COUNTRIES

Table 1.1 Incidence of low birth weight in selected African Countries

Country	% of low Birth weight infants	Year
Ghana	**9**	**2006**
Gabon	**14**	**2000**
Botswana	**10**	**2000**
Nigeria	**14**	**2003**
Tanzania	**10**	**2004-2005**
Angola	**12**	**2000**
Rwanda	**6**	**2005**
Mali	**23**	**2001**

SOURCE: UNICEF 2008

At least 20 million infants are born every year with LBW, representing about 16% of all newborns in developing countries. Nearly 80% of all affected newborns with LBW *at term* are born in Asia (mainly south-central Asia, with Bangladesh having the highest incident rate in the world (Arifeen , 1997); about 15% and 11% are born LBW *at term* in middle and western Africa respectively, and approximately 7% in the Latin American and Caribbean region(ACC/SCN, 2000). The geographical incidence of LBW *at term* in selected Asian and African countries (Figures 2.1 and 2.2 respectively) and table 1.1 confirm that many developing countries exceed the internationally recommended cut-off

levels which should trigger public health action. Incident rates of >15% for LBW and >20% for IUGR indicate that LBW *at term* is a major public health problem. Population-wide interventions aimed at preventing LBW *at term* are therefore urgently required (de Onis et al. 1998).

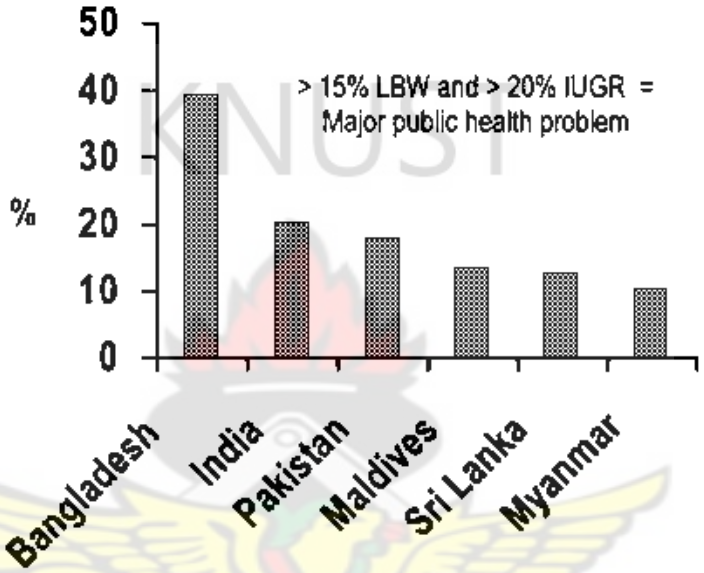

Figure 2.1 Incidence of LBW *at term* in selected Asian countries

Source: de Onis et al. (1998) *Eur J Cl Nutr* 52(S1):S5.

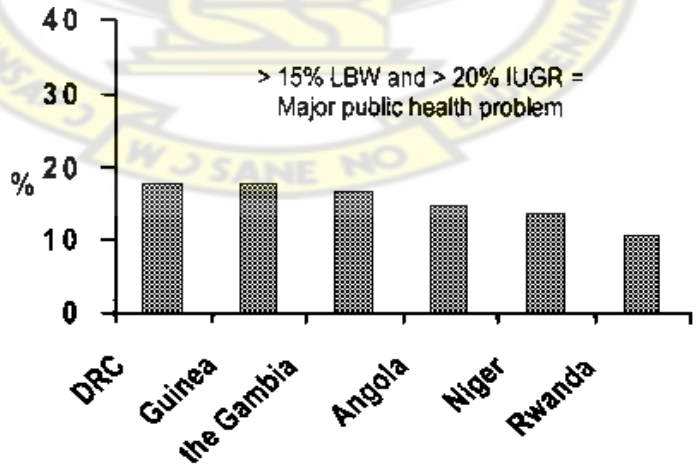

Figure 2.2. Incidence of LBW *at term* in selected African countries

Source: de Onis et al. (1998) *Eur J Cl Nutr* 52(S1):S5.

Historically, because valid assessment of gestational age is often not available in developing countries, evidence of LBW has often been used as a proxy to quantify the magnitude of IUGR. The incident rates for LBW *at term* conservatively estimate IUGR because when all infants below the 10^{th} percentile of the birthweight-for-gestational-age reference are considered, approximately 24% or 30 million newborns in developing countries would be affected each year. Major constraints to deriving this estimate include both the quantitative and qualitative limitations of the available birthweight data (de Onis et al. 1998). Most of the data available from different parts of the world are from clinic or hospital deliveries, whereas, in some regions of Africa and south-east Asia most infants are born at home and are not measured. There is a need to determine whether data from hospital-born infants in developing countries are representative of the large population born at home.

Prematurity and IUGR are the two main causes of LBW. The majority of LBW in developing countries is due to IUGR, while most LBW in industrialized countries is due to preterm birth (Villar and Belizán , 1982).

In many cases, the causes of prematurity are unknown; they may include high maternal blood pressure, acute infections, hard physical work, multiple births, stress, anxiety, and other psychological factors. Causes of IUGR are complex and multiple, but center on the foetus, the placenta, the mother, and combinations of all three. For instance, growth will be retarded *in utero* if the placenta is abnormally small or blocked causing insufficient nutrients to reach the foetus. The maternal environment is the most important determinant of birthweight, and factors that prevent normal circulation across the placenta cause poor nutrient and oxygen supply to the foetus, restricting growth. These factors may include maternal undernutrition, malaria (where it is endemic), anaemia, and acute and chronic

infections (such as sexually transmitted diseases and urinary tract infections) (Cameron and Hofvander, 1983). Also associated with IUGR are primiparity; multiple gestation; foetal, genetic or chromosomal anomalies; as well as maternal disorders such as renal diseases and hypertension (Prada and Tsang, 1998). Cigarette smoking and pre-eclampsia cause the highest relative risks for IUGR in industrialized countries, while alcohol and drug use may also restrict foetal growth (Kramer ,1998).

Major determinants for LBW in developing countries, however, are poor maternal nutritional status at conception, low gestational weight gain due to inadequate dietary intake, and short maternal stature due to the mother's own childhood undernutrition and/or infection (Kramer, 1987). Because maternal undernutrition is a major determinant of LBW in developing countries, high rates of LBW should be interpreted not merely as an indicator of undernutrition, morbidity and mortality for the newborn, but as an urgent public health warning that women of childbearing age are undernourished as well. Countries with higher percentages of LBW infants generally have a higher percentage of women with low body mass index (BMI) and a higher percentage of underweight children (WHO,1997)

To address these issues successfully, the underlying and basic causes of LBW in developing countries such as household food security, maternal and child care, access to and quality of antenatal and other health services, sanitation and hygiene, education, gender discrimination and poverty must be included in any long-term strategies for prevention.

Based on a meta-analysis and critical assessment of the English and French language medical literature published over the period 1970-84, (Kramer, 1987) presents a synthesis comprising 43 potential maternal risk factors for low birth weight grouped into 7

categories: (1) genetic and constitutional factors; (2) demographic and psychosocial factors; (3) obstetric factors; (4) nutritional factors; (5) maternal morbidity during pregnancy; (6) toxic exposures; and (7) antenatal care.

The Kallan (1993) framework comprises 11 'intervening variables' (maternal characteristics) grouped into 4 categories: (1) socio-demographic (age; education; marital status); (2) health-related (parity; prior history of foetal loss or low birth weight; hypertension; diabetes; pelvic infectious disease); (3) attitudinal (wantedness of the pregnancy); and (4) behavioural (smoking; prenatal care).

Both frameworks deserve a careful assessment - even though Kramer, for instance, admits that nearly 75% of the causal factors for prematurity cannot be identified (Kramer, 1987). Both frameworks provide a comprehensive overview of risk factors and group them into suitable categories, while also acknowledging that the causes for prematurity and IUGR respectively are different. However, the direct and indirect effects of certain factors (e.g. the direct biological effect of smoking and smoking as a marker of social disadvantage) are not well explained.

Furthermore, neither framework distinguishes between risk factors by birth weight category (low vs. very low vs. extremely low birth weight). Little account is also taken of combined maternal characteristics, such as low parity at high age. Paternal characteristics or the timing of the onset of menarche are also disregarded (Forssas et al. 1999). Both authors have been able to assess the relative importance of each risk factor. According to Kramer (1987) the ranking is as follows for IUGR: *cigarette smoking; low caloric intake or weight gain; low prepregnancy weight; primiparity; female sex of the child; short stature; nonwhite race; maternal LBW and prior LBW history; general morbidity; other (together accounting for an almost complete explanation*). And the ranking is as follows for prematurity: *cigarette smoking; prior prematurity and spontaneous abortion; low pre-*

pregnancy weight; and in utero exposure to diethylstilbestrol; unknown (together accounting for a 1/3 explanation)

According to Kallan (1993) the most important intervening variables between race and preterm birth are: *parity; prior LBW; hypertension; and pelvic inflammatory disease (PID).* The most important intervening variables between race and IUGR are: *marital status; prior LBW; pregnancy wantedness; and smoking.*

In short, the risk factors for IUGR are fairly well understood, while that is less the case for prematurity. There exists more clarity when it comes to the role of behavioural and health factors, while the role of sociodemographic factors is still part of the big unknown.

2.2.2 BEHAVIOURAL RISK FACTORS

2.2.2.1 Smoking:

As already pointed out, smoking is an important risk factor. Jaakkola and Gissler (2004), in their attempt to assess whether there exists a causal relation between maternal smoking and asthma at age 7, studied the 1987 Finnish cohort of singleton births, and found that the risks for low birth weight, SGA, and preterm delivery were strongly related to maternal smoking during pregnancy. Zarén et al. (1997), examining the effect of maternal smoking on the relationship between maternal haemoglobin levels and pregnancy outcome, analysed data on Swedish and Norwegian women coming to antenatal care from before 17 to after 37 weeks between January 1986 and March 1988. They found that maternal smoking negatively affects foetal growth. In another article, based on the same population and study design, Zarén et al. (2000), found that maternal smoking affects male and female foetuses differently. Male and female foetuses of smoking mothers

2.2.2.2. Alcohol

(Larroque et al. 1995), in their article on moderate prenatal alcohol exposure and psychomotor development at preschool age in France, refer to a number of studies finding associations between moderate alcohol consumption and decreases in birth weight.

2.2.2.3. Stress

(Copper et al. 1996), in a psychosocial assessment study at 25 to 29 weeks of gestation, and based on an analysis of data from the Maternal-Fetal Medicine Units Network of the National Institute of Child Health and Human Development between October 1992 and July 1994, found that after adjusting for maternal demographic and behavioural characteristics, stress was associated with spontaneous preterm birth and low birth weight.

2.2.2.4. Coffee

(Vik et al. 2003), in their study in Norway and Sweden of enrolments that took place between 1 January 1986 and 31 March 1988 of women at either parity 1 or 2, of Caucasian origin, singleton pregnancy and registered before the 20th week of gestation, found that a high caffeine intake in the third trimester of pregnancy was associated with an increased risk of SGA. The increased risk was only found for male foetuses, not for female foetuses. The association held even after controlling for smoking.

2.2.3 HEALTH-RELATED RISK FACTORS

2.2.3.1 Abortion

A distinction has to be made between spontaneous and induced abortion. Based on an analysis of the Danish population registry over the period 1980-1992,(Basso et al.1998) found that a prior spontaneous abortion constitutes a risk factor for both very preterm and preterm delivery, even though spontaneous abortion and preterm delivery themselves also appear to share some risk factors. Another study by Henriet and Kaminski (2001), on the basis of the 1995 French national perinatal survey, found that women who had experienced induced abortion had a higher risk of preterm delivery.

2.2.3.2 Sub-fecundity and fertility treatment:

A clear link has been established between fertility treatments, also called Artificial Reproductive Technology (ART), and multiple births. In countries such as Canada, France and the US, it has been found that "estimates based solely or partly on data from surveys or registers showed that in the late 1980s and in the 1990s, between 20% and 40% of triplet deliveries followed ART, and all in all, that about three quarters of triplet deliveries occurred after procedures for sub-fertility"(Blondel et al. 2002). Similar evidence that fertility treatments increase the likelihood of multiple gestation (twins, triplets), or of sole surviving singletons from an initially larger group of foetuses, was found (Schieve et al. 2002). Also Macfarlane et al. (1990) found in their study on the UK in the years 1980 and 1982-85 that there exists a strong association between infertility treatment and the occurrence of multiple births: 55 percent of mothers of quadruplets and above and 31 percent of the mothers of triplets had undergone some form of

gynaecological investigation for infertility compared to only seven percent of mothers of twins and under three percent of mothers of singletons.

Multiple gestation increases the likelihood of low birth weight. This is the case for twins. For example, "the risks of very preterm delivery and very low birth weight attributable to twins were higher than the risks of overall preterm delivery or low birth weight" (Blondel et al. 2002). But this is also the case for triplets, quadruplets and higher order multiples.

Schieve et al. (2002) found that the proportion of low birth weight among those who underwent assisted reproductive technologies in the US 1996-97 was 13.2 percent among singletons and nearly 100 (%) percent among quadruplets or higher order multiples. Singletons were more likely to be low birth weight when originally more than 2 foetuses were found by ultrasonography.

2.2.3.3 Hypertension:

Hypertension is also considered to be a risk factor mainly for IUGR, although more study is required to verify this. Studying French data for the years 1991 and 1993, Haelterman et al. (1997) found that chronic hypertension has an impact on having a small child. In their analysis, the mean birth weight decreased by 161g for women with chronic hypertension compared to other women.

2.2.4 SOCIO-DEMOGRAPHIC RISK FACTORS

Finally, attention has to be paid to socio-demographic factors such as age, marital status and education. These are highly complex. They have their own predictor effect. They also interact among themselves, however (e.g. age, education and class interact with each other). Furthermore, they interact with behavioural and health related factors.

2.2.4.1 Age:

Maternal age constitutes an important risk factor. Foix-L'Helias and Blondel (2000) found that in France a maternal age above 34 years old constituted an important risk factor in both 1981 and 1995 while a maternal age below 20 years constituted an important risk factor in 1981 but not in 1995. Similar results were obtained by Dičkute et al. (2004), who in their study of Lithuania in 2001-2 identified a U-shaped relationship between maternal age and LBW risk (younger than 20 years and 35 years and older), although other factors also clearly played a role.

The importance of a young maternal age appears to be decreasing in European countries. In countries where the proportion of young mothers is small (below 3 percent), examples being France, Finland and Sweden in the 1990s, there is no (or only a weak) association between young maternal age and pre-term birth (Foix-L'Helias and Blondel, 2000, referring to Hemminki and Gissler, 1996).

On the other hand, as Foix-L'Helias and Blondel and others have demonstrated, a higher maternal age (e.g. above 34 years old) remains an important risk factor. Other authors have also found a strong relation between a higher maternal age and the risk for premature birth. An Italian study (Astolfi and Zonta, 2002) has found that the 35 plus age group always has a significantly higher risk of still or preterm birth, or low birth weight at term, even when parity and education variables are controlled for. A French study (Bréart, 1997) has similarly found negative consequences of delayed childbearing (35 plus) in the format of higher featal death rates.

Similar findings were also reported for Alberta, Canada, where Tough et al. (2002) reported that "when only those age 35 and older of first parity were considered, the contribution to the population increase in LBW and preterm delivery was less than half of the total contribution for this age group (36%). The negative effect of a higher maternal

age can be weakened by education and parity, but only to a rather small extent (Astolfi and Zonta, 2002, referring also to the Danish case-study of Basso et al, 1997).

2.2.4.2 Education:

Another socio-demographic factor is education. Most studies conclude that higher levels of maternal educational attainment lead to better pregnancy outcomes.

Raum et al. (2001) examined the influence of maternal education on IUGR in two different political and social systems, West and East Germany in 1987/88 and 1990/91 respectively. They found that mothers among the lowest category of education in both West and East Germany had an unadjusted relative risk of 2.5 for delivering an SGA child compared to high education.

In France, two methods are used for legal abortions: surgical abortion (vacuum aspiration followed by curettage) and prescription of abortion pills mifepristone (RU-486) and prostaglandins for < 7 weeks gestational age. Intervention at high gestational age is low (4% after 11 weeks) in France (Henriet and Kaminski, 2001) Those of the highest education category, This indicates that the education of the mother is a proxy for the socio-economic and lifestyle status, including smoking behaviour, frequency of prenatal care visits (health seeking behaviour), etc.

2.2.4.3 Marital status:

Another socio-demographic factor is marital status. Foix-L'Helias and Blondel (2000) found that risk factors included being single in 1981 but not in 1995. So at the country level, the importance of being single as a risk factor appears to decrease over time in France. In countries with a high proportion of non-married mothers there is no association

between marital status and pre-term birth. Examples of such countries are Finland, Sweden and France in 1995 (Foix-L'Helias and Blondel, 2000).

2.2.4.4 Social class:

The socio-demographic risk factor of class interacts with behavioural risk factors such as smoking. This is demonstrated by inter alia Kramer et al. (2000) Many times, the socio-demographic risk factor of class is treated together with education and marital status. Dičkute and his colleagues (2004), based on a study of Lithuania in 2001-2, found that there exist a U-shaped relationship between maternal age and LBW risk (younger than 20 years and 35 years and older) but clearly say that low birth weight is associated with other factors: low levels of education, unstable marital status, and low income. However, the role of socio-economic status is very complex.

A good example is its interaction with selection and screening processes. On the one hand, Carlson et al. (1999) and Carlson and Hoem (1999), by focusing on trajectories of foetal loss in the Czech republic, identify pregnancy outcomes that are more positive for low than for high socioeconomic status women, in the format of the 'survival paradox'. On the other hand, Khoshnood et al. (2004) report in their study based on the French National Perinatal Survey of 1998 on the better access of high socioeconomic status women to maternal serum screening for Down syndrome, and thus, to more informed decision-making as compared to low socioeconomic status women.

The above clearly shows that most studies of low birth weight and SGA have focused on the relationship between perinatal factors and cognitive and neurological development. Relatively little is known about other factors which are important to child health development, such as the social and home/family environment (Andersson et al. 1997).

Nevertheless it has been suggested that the environment, especially the early home environment, plays a central role in child health and (cognitive) development. Andersson et al. (1997), for instance, have noted how the quality and amount of stimulation received by children in different environments varies greatly, care-taking practices of lower- and middle-class parents differ substantially, and children from lower-class families seem to develop more slowly. Angelsen et al. (2001) have highlighted the importance of environmental factors - such as adequate nutrition and the parental ability to create a good and stimulating home environment – and have focused on the positive correlation between breastfeeding and cognitive development in children. Further, in the study by Smith et al. (2003), where the effect of breastfeeding (which was believed to be associated with confounding socio-economic factors) on childhood cognitive development among VLBW children was examined, it was found that both expressed milk feedings and direct breastfeedings were associated with improved visualmotor function. Breastfeeding mothers were more likely to be older and married, and to smoke less.

So far, however, the role of the environment in shaping low birth weight children's health outcomes had not been studied sufficiently, or incorrectly. Andersson et al. (1997) have noted that "the relationship between cognitive development and social conditions among infants born SGA had been sparsely studied". On the other hand, Saigal et al. (2003) have noted that "socio-economic factors, racial and ethnic differences, the nature of funding of health care … may further contribute to differences in the reported outcomes" (but that quite often these are not sufficiently controlled for."

Another problem can be a strong selection effect generating an overrepresentation of lower socio-economic groups. In one study on the *20* year outcome of infants weighing 600 grams or less at birth (Sweet et al. 2003), for instance, all mothers were of low socio-

economic origin, whether measured by race, marital status, the receipt of social assistance, or tobacco use. This selection effect may stem from the fact that higher socio-economic groups are more likely to engage less in behaviour possibly giving rise to low birth weight (smoking, drinking, drugs, etc.). Furthermore, even if a low birth weight child is born to women in these groups, they may prefer not to pursue aggressive health treatments since they are better able to assess the possible negative impacts of low birth weight on later health and on their own lives. Those findings that are available thus far on the role of the socioeconomic environment tend to contradict each other. At least one study found that the socio-economic background does not matter much for child health outcomes (Hack et al. 2002). On the other hand, Resnick et al. (1999) concluded that the impact of socio-demographic factors on adverse educational outcomes is greater than that of perinatal factors. Singer et al. (1997) noted that mental outcomes at age 3 were affected by socio-economic factors such as minority status, race and lower social class. The relation with Bronchopulmonary Dysplasia (BPD) and higher rates of learning disabilities at school age in VLBW cohorts was not clear, however.

2.3 MORBIDITY AND MORTALITY CONSEQUENCES OF LBW IN NEONATES

LBW is generally associated with increased morbidity and mortality, impaired immune function, and poor cognitive development for neonates (newborns 1-28 days of age) and infants. Infants born LBW are at risk to develop acute diarrhoea or to be hospitalized for diarrhoeal episodes at a rate almost two to four times greater than their normal birthweight counterparts (Bukenya et al. 1991)

Infants who are LBW risk contracting pneumonia or acute lower respiratory infections (ALRI) at a rate almost twice that of infants with normal birthweight; and more than three

times greater if their weight is less than 2000 g (Cerqueiro et al. 1990)). LBW is also implicated as a contributor to impaired immune function which may be sustained throughout childhood.

The risk of neonatal death for infants who are LBW weighing 2000-2499 g at birth is estimated to be four times higher than for infants weighing 2500-2999 g, and ten times higher than for infants weighing 3000-3499 g (Ashworth,1998)). In Brazil, 67% of all infants dying during their first week of life are LBW infants; in Indonesia the rate is 40%; and in the Sudan the rate is 35%. Infant mortality (less than one year of age) due to LBW was slightly lower: 47% in Brazil and 19% in Indonesia (Barros, et al. (1992). LBW infants during the post-neonatal period (>28 days of age) also have high mortality rates - and in some cases their risk may be greater than those for LBW infants during the neonatal period (Ashworth A and Feachem RG,1985). LBW accounted for 69% of the ALRI deaths in India, and it is estimated that in Bangladesh, almost half of the infant deaths from pneumonia or ALRI and diarrhoea could be prevented if LBW were eliminated (Datta et al. 1987).

Over the past few decades, the chances of survival for low birth weight children have improved significantly. This is reflected in the decrease in overall infant mortality rates, which is mainly due to decreasing mortality for the lowest birth weights. Data from the WHO 'Health for All' database show that foetal, neonatal and infant mortality rates have been decreasing over the past few decades. However, while neonatal and infant mortality rates are clearly converging, this does not yet appear to be the case for foetal mortality rates.25 In the 10 new EU Member States too all three mortality rates are broadly decreasing, but at higher levels than the EU-15, and with as yet much less convergence.

Reductions in low birth weight mortality have greatly contributed to the decrease in overall mortality. Hack et al. (1994) note that "during the past decade, advances in perinatal care have resulted in increases in the survival of extremely small and immature infants. Whereas few infants with birth weights below 750g were actively treated before the 1980s, treatment is now accepted practice for most infants born in North America with birth weights of at least 500g, those born at 24 or more weeks' gestation, or both". Forssas et al. (1998) note that the largest decline in stillbirth mortality occurred among those weighing below 1,000g, while for early neonatal deaths the group most affected weighed 1,000-1,499g at birth.

One has to be careful when discussing death rates, however. There is still substantial underreporting of stillbirths and early neonatal deaths. This was convincingly demonstrated though the recent PERISTAT project a lower rate of post-term birth. The usual explanation for this systematic difference includes inaccurate reporting of the last normal menstrual period and the more frequent occurrence of delayed rather than early ovulation. While its conclusions are not completely definite, this study indicates that "EUS-scan-based estimates are more valid on average than LNMP-based estimates. According to Lack et al. (2003) the use of ultrasound decreases the proportion of reported post-term pregnancies and increases the proportion of preterm infants, implying the underestimation of gestational age.

2.4 GROWTH IN LBW CHILDREN

Do LBW infants grow normally? What are the consequences of LBW on body size, composition, strength and cognitive development? Attaining full growth potential is

especially important for women and girls in order to break the intergenerational cycle of LBW and have fewer delivery complications. Maternal height is not only a reflection of genetic make-up, but also reflects her dietary history. From societal, community and individual standpoints, adolescents and adults born with LBW generally have less strength and lower lean body mass resulting in decreased work capacity and lost productivity, which may cost nations billions of dollars (Martorell et al. 1998)

When growth restriction *in utero* occurs early in pregnancy, infants exhibit symmetrical (or proportional) growth with length, weight, head and abdominal circumference all below the 10^{th} percentile reference for a given gestational age (stunting). When growth restriction *in utero* occurs late in pregnancy, the infant exhibits asymmetrical (or disproportionate) growth with a normal length and head circumference, but low weight due mainly to a lower proportion of visceral and fat tissue (wasting) (Bakketeig ,1998). Neonatal mortality rates are reported to be higher among asymmetrical IUGR infants, but if they survive, they have a better prognoses for long-term growth and development than that for symmetrical IUGR infants. IUGR infants catch-up partially in growth relative to their appropriate birthweight counterparts during their first one or two years of life. Thereafter, IUGR children maintain their place in the distribution and neither catch-up nor fall further behind. They remain about 5 cm shorter and 5 kg lighter as adults. Premature infants (who are usually asymmetric LBW), who survive their first year, have a much better prognosis in terms of future growth than IUGR infants. Despite their earlier disadvantage, preterm children gradually catch-up with their appropriate birthweight, term counterparts. Premature infants and IUGR infants should be studied as separate groups because they show different patterns of growth, morbidity and mortality. From a programmatic viewpoint these differences have enormous implications for intervention

strategies and limitations of the approach of nutritional recovery of IUGR infants in early childhood.(Hass, et al.1987)

Neurological dysfunction is often associated with attention deficit disorders, hyperactivity, clumsiness, and poor school performance. Neurologic dysfunction, when present, seems to affect IUGR boys more than girls, and children of lower socioeconomic circumstances. If IUGR infants are symmetrical and head growth is affected, there seems to be more of an impact on neurological function and it is not clear whether interventions directed toward these infants will improve their outcome. For asymmetric IUGR infants, preventing asphyxia should reduce the prevalence of major and minor handicaps, especially cerebral palsy and mental impairment frequently seen in these infants. IUGR is a much larger public health problem in developing countries than in industrialized countries and the outcomes are more likely to be aggravated by obstetric complications and perinatal problems, and later by poor health and nutrition as well as psycho-social deprivation (Goldenberg et al.1998).

In developing countries children are exposed to poor nutrition, high levels of infections, and other conditions of poverty, thus, their long term development is dependent to a large extent on the quality of their environment. It is difficult to isolate the effects of IUGR from these factors in relation to cognitive development. Cognitive deficits appear to change over time. For instance, when IUGR infants were examined, no differences were found during the first year of life, but differences emerged during two and three years of age; and then differences disappeared at four to five years. Deficits have been found in children with very low birthweights, the smallest size, or with early IUGR (growth restriction prior to 26 weeks gestation). Since LBW occurs more often in deprived environments, it can serve as a marker for the associated poor outcomes throughout life.

A length deficit at an early age (stunting) would be the best predictor of motor and mental development deficits (Grantham-McGregor, 1998)

CHAPTER THREE

METHODOLOGY

3.0 INTRODUCTION

This chapter deals with the study area, method and formula of Multiple logistic regression model, concept of Multiple logistic regression models, preliminary test, linear regression model and probit regression model.

3.1 STUDY AREA

St Martin's hospital is a nonprofit health facility which was founded in 1957. It was elevated to the status of a district hospital in 1990. The hospital is the property of the Obuasi Catholic Diocese, under the general supervision of his Lordship Rt. Rev. Justice Yaw Anokye, the Bishop. It is the only hospital facility in the Amansie West District of the Ashanti and serves a population of 144,924 people within 160 communities.

The hospital's humble beginning is traceable to the untiring efforts of Rev. Fr. L Bekema (SMA)**,** Dutch who first settled at Agroyesum in 1953 as a missionary. He was later joined by Rev. Fr. Dr. Fevers (both missionary and medical practitioners) and the two of them were able to convince the traditional authorities of the need to establish a health facility in the Manso area to cater for the health needs of the rural dwellers. Having been given the mandate, the hospital was founded in October, 1957, first as Maternity Home in the small Mission House of St Anthony's Parish-Agroyesum. In 1960, Dr De-groot another Dutch medical practitioner took over the running of the new facility as the first Medical Officer. He later acquired the land for the hospital's permanent site, and through communal efforts, together with support from the Dutch Missionaries, the first permanent

structures consisting of a ward with small attachment for surgical procedure was built in 1965. The hospital grew steadily afterwards, amidst grave difficulties. It was elevated to the status of a District Hospital in 1990, and has since remained a focal point in the health care delivery system of the district. The hospital is well acclaimed both locally and internationally for its painstaking devotion to the treatment of Buruli Ulcer, mycobacterium ulcerous infections, endemic in the catchment area of the hospital, and other parts of the country.

3.1.2 RANGE OF SERVICE OF THE HOSPITAL

The Hospital is a general Hospital and provides a wide range of diagnostic, curative and preventive services befitting its status as the District Referral facility. These include; 24-hour Out and In-patient Care, Laboratory Services, X-Ray, Ultra Sound, Surgical Services, (both emergency and elective), Ophthalmic Service (Eye Clinic) Child Welfare and Primary Health care facility, Physiotherapy, Reproductive Health Care and Save Motherhood, as well as Specialized Clinic for Diabetic, HIV/AIDS, TB, and other patients with chronic diseases or conditions.

Though not well equipped for the treatment and management of Buruli Ulcer cases yet the Hospital is renowned, both locally and internationally for services it offers to the people with this ailment. The Hospital treats Buruli Ulcer referral cases from across the length and breadth of the country.

The hospital has a bed compliment of 116 distribute over the four admitting wards as follows; males- 41, females-26; maternity -16 and children's wards - 33.

3.2 MEASUREMENT AND DATA ANALYSIS

This was a health facility based retrospective review of data of pregnant mothers who delivered in the health facility in Amansie West district. Records of women who

delivered a singleton in the hospital in 2010 were extracted from the registers and those met were asked mothers to show the health card on which the birth weight was recorded. Birth weights were recorded in grams to the nearest 10g. Low birth weight, one of the response variables in our analysis, was defined as a weight at birth of less than 2,500g. The biological data collected include age of mother at the time of delivery, birth weight and sex of the child. Birth weights of newborns were measured without clothes within 15-30 minutes of birth to the nearest 10g, using an infant beam balance.

The socio-economic data available from hospital case cards related to the occupation of the mother were also taken. Data on socio-economic and biological determinants of birth weight in Ghana is generally incomplete.

We used multiple logistic regression analysis to assess the strength of association of each explanatory variable with the likelihood of a child having low birth weight, after adjusting for other covariates. Results of the regression analyses are presented in the form of odds ratios (OR), with 95% confidence intervals (CI) and *p*-values. We used Hosmer-Lemeshow and Pearson's goodness-of-fit tests to check the fit of the logistic regression models.

The multiple regression analyses presented are based on a final sample size of 212 neonates for low birth weight. A level of $p < 0.05$ and 0.1 were considered statistically significant, and all statistical analyses were conducted using SAS.

3.2.1 COVARIATES

Biological attributes at birth: The biological attributes at birth are those that cause poor developmental outcomes for the fetus. These include maternal attributes as well as the child's. In the analysis, child-level covariates include birth weight (<2,500g, ≥2,500g) and

sex (boy, girl). Maternal covariates include mother's age at each child's birth, Gestation age and mother's weight and Hypertension status.

3.3 CHI-SQUARE TEST FOR INDEPENDENCE

A chi-square test for independence was used to determine whether there was a significant association between occupation, hypertension and sex that were categorical in the data and the LBW.

TABLE 3.1: CONTINGENCY TABLE

		J				
		1	2	3 ...	j	
I	1	0_{11}	0_{12}	0_{13} ...	0_{1j}	0_{1+}
	2	0_{21}	0_{22}	0_{23} ...	0_{2j}	0_{2+}
	.	.	.	.	.	.
	.	.	.	.	.	.
	.	.	.	.	.	.
	i	0_{i1}	0_{i2}	0_{i3} ...	0_{ij}	0_{i+}
		0_{+1}	0_{+2}	0_{+3} ...	0_{+j}	N

The test statistic is a chi-square random variable (χ^2) defined by the following equation.

$$\chi^2 = \sum_i^I \sum_j^J \frac{(O_{ij} - E_{ij})^2}{E_{ij}} \sim \chi^2_{(I-1)(J-1)} \quad (3.1)$$

Where the expected frequency E_{ij} is defined as

$$E_{ij} = \frac{O_{i+} \times O_{+j}}{N} \quad (3.2)$$

The O_{ij} is the observed frequency count at level O_{i+} of Variable j and level O_{+j} of Variable *i* and *(i-1)(j-1)* is the degrees of freedom.

3. 4 MULTIPLE LINEAR REGRESSION MODEL

Multiple regression is used to account for (predict) the variance in an interval dependent, based on linear combinations of interval, dichotomous, or dummy predictor variables. Power terms can be added as predictor variables to explore curvilinear effects. Cross-product terms can be added as predictor variables to explore interaction effects. Using hierarchical regression, one can see how much variance in the dependent can be explained by one or a set of new predictor variables, over and above that explained by an earlier set. The estimates (β coefficients and constant) can be used to construct a prediction equation and generate predicted scores on a variable for further analysis.

We have *k* variables that we control, or know in advance of outcome, that are used to predict *Y*, the response (dependent variable). The *k* predictor variables are labeled X_1, X_2,...,X_k. The levels of these variables for the i^{th} case are labeled X_{1i} ,..., X_{ki} . Note that simple linear regression is a special case where *k*=1, thus the methods used are just basic extensions of what we have previously done.

$$E(Y \mid X) = \beta_o + \beta_1 x_1 + \beta_2 x_2 + ... + \beta_K x_K + \varepsilon \qquad (3.3)$$

where β_j is the change in mean for *Y* when variable X_j increases by 1 unit, while holding the *k*-1 remaining predictor variables constant (partial regression coefficient). This is also referred to as the slope of *Y* with variable X_j holding the other predictors constant.

i. *Y* is the continuous and random variable we wish to estimate or predict $x_1, x_{2,...,}x_k$.

ii. $x_1, x_{2,...,}x_k$ are the predictor variables that are measured without error.

iii.$\beta_0, \beta_1, \beta_{2,...,} \beta_k$ are the parameters to be determined.,

iv.ε is the random error for any given set values of $x_1, x_{2,\ldots,}x_k$.

3.4.1 Assumptions

i. $E(\varepsilon) = 0$ for $i = 1,2,\ldots,n$ that is a given set values $x_1, x_{2,\ldots,}x_k$.

$$E(y_i) = \beta_0 + \beta_1 x_1 + \beta_2 x_{i2} + \ldots + \beta_k x_{ik} = 0$$

ii. $Var(\varepsilon_i) = \sigma^2$ which is constant for all the values of x. and $Var(y_i) = \sigma^2$, a constant for each recorded value of y_i.

iii. The random error ε_i is independent, that is their covariance is equal to zero.

$$\text{cov}(\varepsilon_i, \varepsilon_k) = 0 = E(\varepsilon_i, \varepsilon_k) = 0, i \neq k$$

iv. The random error $\varepsilon_i \sim N(0, \delta^2)$, that is

$$y \sim N(\beta_0 + \beta_1 x_{i1} + \beta_2 x_{i2} + \ldots + \beta_k x_{ik}, \delta^2)$$

3 .4.2 Inferential Analysis

Analysis of Variance (ANOVA) is an inferential method that is used to test the equality of three or more population means. The application of ANOVA in regression analysis is based on the partitioning of the total variation and its degree of freedom into components.

We have:

$$(y_i - \hat{y}) = (y_i - \hat{y}_i) + (\hat{y}_i - \bar{y})$$

Where

$(y_i - \bar{y}) =$ total variation

$(y_i - \hat{y}_i) =$ unexplained variation

$(\hat{y}_i - \bar{y}_i) =$ explained variation

$$\sum_{i=1}^{n}(y_i-\bar{y})^2=\sum_{i=1}^{n}\left[(y_i-\hat{y}_i+(\hat{y}_1-y_i)\right]^2 \tag{3.4}$$

$$\sum_{i=1}^{n}(y_i-\bar{y})^2=\sum_{i=1}^{n}(y_i-\hat{y}_i)^2+\sum_{i=1}^{n}(\hat{y}_i-\bar{y})^2$$

Where:

$$\sum_{i=1}^{n}(y_i-\bar{y})^2 = \text{total variation } (SS_{yy})$$

$$\sum_{i=1}^{n}(y_i-\hat{y}_i)^2 = \text{unexplained variation } (SSE)$$

$$\sum_{i=1}^{n}(\hat{y}_1-\bar{y})^2 = \text{explained variation } (SSR)$$

The three quantities are measures of dispersion. SS_{yy} is a measure of dispersion of the total variation in the observed values, y_i. SSR measures of amount of total variation in the observed values of y_i that is accounted for by the linear relationship between the observed values of x and y. The unexplained sum of squares (SSE) is a measure of dispersion of the observed y_i values about the regression line which is sometimes called the error or residual sum of squares.

3.4.3 Analysis of Variance Table (Based on *k* Predictor Variables)

The decomposition of the total sum of squares and its degrees of freedom (*df)* are displayed in the table below:

Total Sum of Squares and df: $SST = \sum_{i=1}^{n}(Y_i - \bar{Y})^2 \qquad df_T = n-1$

Regression Sum of Squares: $SSR = \sum_{i=1}^{n}(\hat{Y}_i - \bar{Y})^2 \qquad df_R = k$

Error Sum of Squares: $SSE = \sum_{i=1}^{n} (Y_i - \hat{Y}_i)^2$ $\quad df_E = n - k - 1$

TABLE 3.2 Analysis of variance (based on k predictor variables)

Source	*df*	*SS*	*MS*	*F*
Regression	df_R	*SSR*	*MSR=SSR/k*	F_{obs}=*MSR/MSE*
Error	df_E	*SSE*	*MSE=SSE/(n-k-1)*	---
Total	df_T	*SST*	---	---

3.4.4 *F*-TEST FOR OVERALL MODEL

F-test is used to test whether any of the predictor variables are linearly associated with *Y*.

H_0: $\beta_1 = ... = \beta_k = 0$ (*Y* is not linearly associated with any of the predictor variables)

H_A: Not all $\beta_j = 0$ (At least one of the predictor variables is associated with *Y*)

TS: $F_{obs} = \dfrac{MSR}{MSE}$

RR: $F_{obs} \geq F_{\alpha,k,n-k-1}$ that is reject H_0: $\beta_1 = ... = \beta_k = 0$

P-value: Area in the *F*-distribution to the right of F_{obs}

3.4.5 MULTIPLE COEFFICIENT OF DETERMINATION

Proportion of variation in *Y* "explained" by the regression on the *k* predictor variables is called multiple coefficient of determination denoted by R^2.

If $R^2 = 1$, the fitted model passes through all the data point and also

$\hat{\varepsilon}_i = 0$ **for all** $i = 1,2,3,...,n$. If $R^2 = 0$, $\hat{\beta}_0 = \bar{y}$ **and** $\beta_1 = \beta_2 = ...\hat{\beta}_k = 0$

In this case the predictor variables $x_{i1}, x_{i2}, x_{i3}, ..., x_{ik}$ have no imfluence on the response

variable $\hat{y}_i$. If more predictor variables are introduced in the model R^2 tend increase it value.

The coefficient of determination R^2 is given as

$$R^2 = r^2_{Y\bullet 1,\ldots,k} = \frac{\sum_{i=1}^{n}\left(\hat{Y}_i - \bar{Y}\right)^2}{\sum_{i=1}^{n}\left(Y_i - \bar{Y}\right)^2} = \frac{SSR}{SST} = 1 - \frac{SSE}{SST}$$

Since R^2 is often by including larger number of predictor variables, it is sometime suggested to modified R^2 to account for the number of independent variables in the model,

The adjusted multiple coefficient of determination, denoted, R^2 is defined by

$$R_a^2 = 1 - \left(\frac{n-1}{n-\rho}\right)\frac{SSE}{SST} = 1\frac{(1-R^2)(n-1)}{n-k-1} \qquad (3.5)$$

3.4.6 Confidence Interval for Estimated Coefficients in Multiple Regressions

If the regression assumptions on the residuals are satisfied, including the normality assumption, then the sampling distribution of an estimated regression coefficient is normal with a variance proportional to the residual mean square (MSE). The variance of the estimator also depends on the variances and covariances of the predictors. For more than one predictor, the confidence intervals for regression can be computed similarly, but the equation is more complicated. The equation for the variances and covariances of estimated coefficients is expressed in matrix terms by

$$\text{Var}(\hat{\beta}) = S_e^2(X^TX)^{-1} = \text{MSE}(X^TX)^{-1} \qquad (3.6)$$

Where S_e^2 is the residual mean square and X is the time series matrix of predictors. Equation (3.7) returns a matrix, with the variances of the parameters along the diagonal, and the covariances as the off-diagonal elements. The appropriate degrees of freedom of the *t* distribution is *df = n-k-1* , where *K* is the number of predictors in the model, and *n* is the sample size.

The *95% Confidence Interval for β* provides the lower and upper bounds for the unstandardized regression coefficients, based on the previously computed standard error.

3.5 THE MULTIPLE LOGISTIC REGRESSION MODEL

Logistic regression analysis extends the techniques of multiple regression analysis to research situations in which the outcome variable is categorical, that is, taking on two possible values. In this study, the risk factors for the low birth weight are identified using logistic regression analysis.

Logistic regression is a useful way of describing the relationship between one or more predictor variables (e.g., age, sex, etc.) and a binary response variable, expressed as a probability, that has only two values, such as having low birth weight ("has low birth weight" or "doesn't have low birth weight")

In setting up the logistic regression model, we must first establish the fundamental model for any multiple regression analysis.

To explain the popularity of logistic regression, we show here the logistic function**,** *f(z)* which describes the mathematical form on which the logistic model is based and Consider a collection of *k* predictor variables denoted by the vector $x' = (x_1, x_2 \dots x_p)$. It is assumed that the outcome variable z is a linear combination of a set of predictors.

$$Z = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \dots + \beta_K x_K \qquad (3.7)$$

and β_0 and the β_i are constant terms representing unknown parameters. This function, called $\boldsymbol{f(z)}$, is given by

$$f(z) = \frac{1}{1+e^{-(\beta_0+\sum_{i=1}^{k}\beta_i x_i)}} \tag{3.8}$$

The logistic regression model indirectly models the response variable based on probabilities associated with the values of *Y*. We will use π(x) to represent the probability that Y =1, which is the presence of low birth weight similarly, we will define 1- $\pi(x)$ to be the probability that Y =0, which is absence of low birth weight. in which case the logistic regression model is:

The general formular for the logistic regression model with single variable is;

$$\pi(x) = \frac{e^{\beta_o+\sum_{i=1}^{k}\beta_i x_i}}{1+e^{\beta_o+\sum_{i=1}^{k}\beta_i x_i}} \tag{3.9}$$

From equation (3.9), k = the number of independent variables and P(Y=1/**x**) = π **(x) =** the conditional probability that the outcome is present. The equation(3.9) above can be put as:

$$\mathrm{P(Y=1}|x_1, x_2, ..., x_k) = \frac{1}{e^{-(\beta_0+\sum_{i=1}^{k}\beta_1 x_i)}} \tag{3.10}$$

The logits, natural logs of the odds, of the unknown binomial probabilities are modeled as a linear function of the X_K.

$$\text{Logit } (\pi(\mathrm{x})) = \ln[\frac{\pi(\mathrm{x})}{1-\pi(\mathrm{x})}] = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + ... + \beta_k x_k \tag{3.11}$$

Note that a particular element of X_K can be set to 1 for all K to yield an intercept in the model. The unknown parameters β_K are usually estimated by maximum likelihood using a method common to all generalized linear models.

The interpretation of the β_K parameter estimates is as the additive effect on the log of the odds for a unit change in the Kth explanatory variable. In the case of a dichotomous explanatory variable, for instance gender, e^{β} is the estimate of the odds of having the outcome for, say, males compared with females.

3.5.2 FITTING THE MULTIPLE LOGISTIC REGRESSION MODEL

Assume that we have a sample of n predictor observations $(x_i y_i), i = 1,2, \dots, n$. As in the univariate case, fitting the model requires that we obtain estimates of the vector $\beta' = (\beta_\circ, \beta_{1,\dots,}\beta_k)$. The method of estimation used in the univariate situation will be employed in the multivariate case – maximum likelihood. There will be *k+1* likelihood equations that are obtained by differentiating the log likelihood function with respect to the *k+1* coefficients. The likelihood equations that result may be expressed as follows:

$$\sum_{i=1}^{n} [y_i - \pi(x_i)] = 0$$

and

$$\sum_{i=1}^{n} x_{ij} [y_i - \pi(x_i)] = 0 \; for \; j = 1,2, \dots, k$$

As in the univariate model, the solution of the likelihood equations requires special software that is available in most, if not all, statistical packages. Let β denote the solution to these equations. Thus, the fitted values for the multiple logistic regression model are $\hat{\pi}(x_i)$, the value of the expression in equation (3.11) computed using β and x_i.

In the previous section only a brief mention was made of the method for estimating the standard errors of the estimated coefficients. Now that the logistic regression model has been generalized both in concept and notation to the multivariate case, we consider estimation of the standard errors in more detail.

The estimators are obtained from the matrix of second partial derivatives of the log likelihood function. These partial derivative have the following general form

$$\frac{\partial^2 L(\beta)}{\partial \beta_j^2} = -\sum_{i=1}^{n} x_{ij}^2 \pi_i (1-\pi_i) \qquad (3.12)$$

and

$$\frac{\partial^2 L(\beta)}{\partial \beta_j^2} = -\sum_{i=1}^{n} x_{ij}\, x_{il} \pi_i (1-\pi_i) \qquad (3.13)$$

$for\ j, l = 0,1,2, \dots, k$ where π_i denotes $\pi(x_i)$. Let the $(k+1) \times (k+1)$ matrix containing the negative of the terms given in equations (3.12) and (3.13) be denoted as $I(\beta)$. This matrix is called the observed information matrix. The variances and covariances of the estimated coefficients are obtained from the inverse of this matrix which we denote as $Var(\beta) = \mathrm{I}^{-1}(\beta)$. Except in very special cases it is not possible to write down an explicit expression for the elements in this matrix. Hence, we will use the notation $Var(\beta_j)$ to denote the j^{th} diagonal element of this matrix, which is the variance of $\hat{\beta}_j$, and covariance $Cov(\beta_j, \beta_l)$ to denote an arbitrary off-diagonal element, which is the covariance of $\hat{\beta}_{\mathrm{j\ and}}\ \hat{\beta}_{\mathrm{l}}$. The estimators of the variances and covariances, which will be denoted by $\hat{Var}(\hat{\beta})$ are obtained by evaluating $Var(\beta)\ at\ \hat{\beta}$. We will use $\hat{Var}(\hat{\beta}_j) and\ Cov(\hat{\beta}_j, \hat{\beta}_l), j, l = 0,1,2, \dots, k$ to denote the values in this matrix.

For the most part, we will have occasion to use only the estimated standard errors of the standard coefficients, which we will denote as

$$S\hat{E}(\hat{\beta}_j) = \sqrt{[V\hat{a}r(\hat{\beta}_j)]} \tag{3.14}$$

for $j = 0,1,2,\ldots,k$. We will use this notation in developing methods for coefficient testing and confidence interval estimation.

A formulation of the information matrix which will be useful when discussing model fitting and assessment of fit is $\hat{I}(\hat{\beta}) = X'VX$ where X is an $n\ by\ k+1$ matrix containing the data for each subject, and V is an n by n diagonal matrix with general element $\hat{\pi}_i(1-\hat{\pi}_i)$. That is, the matrix X is

$$X = \begin{bmatrix} 1 & x_{11} & \cdots & x_{1k} \\ 1 & x_{21} & \cdots & x_{2k} \\ \vdots & \vdots & \cdots & \vdots \\ 1 & x_{n1} & \cdots & x_{nk} \end{bmatrix}$$

and the matrix V is

$$V = \begin{bmatrix} \hat{\pi}_1(1-\hat{\pi}_1) & 0 & \cdots & 0 \\ 0 & \hat{\pi}_2(1-\hat{\pi}_2) & \cdots & 0 \\ \vdots & 0 & \ddots & \vdots \\ 0 & \cdots & 0 & \hat{\pi}_n(1-\hat{\pi}_n) \end{bmatrix}$$

3.5.3 Testing for the Significance of the Model

Before concluding that any or all of the coefficients are nonzero, we may wish to look at the univariate Wald test statistics,

$$W_j = \frac{\hat{\beta}_j}{\widehat{SE}(\hat{\beta}_j)} \tag{3.15}$$

The multivariable analog of the Wald test is obtained from the following vector-matrix calculation:

$$W = \hat{\beta}'\left[\hat{\text{Var}}(\hat{\beta})\right]^{-1}\hat{\beta}$$

$$W = \hat{\beta}'(X'VX)\hat{\beta}, \qquad (3.16)$$

which will be distributed as chi-square with $k+1$ degrees of freedom under the hypothesis that each of the $k+1$ coefficients is equal to zero.

Then multivariable analog of the Score test for the significance of the model is based on the distribution of the p derivatives of $L(\beta)$ with respect to β. The computation of this test is of the same order of complication as the Wald test.

3.5.3 HOSMER-LEMESHOW STATISTIC

A summary measure verifies the fit of a model by first computing the difference between the observed and predicted scores throughout the entire collection of observations, then examining the individual contribution of the difference from each pair relative to the error structure of the whole model, and, finally, determining the suitability and efficiency of the model by a single statistic. Empirical consistency, which refers to the observed outcome behaving in accordance with the model prediction, is revealed by a chi-square statistic that is computed with a contingency table that collapses the observations into a fixed number of groups.

Hosmer and Lemeshow (1980) proposed a grouping strategy that is based on the value of estimated probabilities. Supposing that there are *n* observations in the probability model, Group 1 would consist of the *n*/*g* observations with the lowest predicted probabilities, Group 2 would consist of the *n*/*g* observations with the next-lowest predicted probabilities, and so on. Once all of the groups are created, a Pearson chi-square statistic is estimated based on the observed and expected number of observations in the interest

category of every group. When the Pearson chi-square statistic is significant, the null hypothesis that the proposed model sufficiently describes the empirical association is rejected.

Let n_k be the number of observations in the *k*th group and y_i be the response of the *i*th observation. Then, the number of observed responses in interest for the *k*th group would be

$$o_k = \sum_{i=1}^{n_k} y_i \,. \tag{3.17}$$

In contrast, if $\hat{\pi}_i$ denotes the predicted probability for the *i*th observation, then the average estimated probability for this group would be

$$\overline{\pi}_k = \sum_{i=1}^{n_k} \frac{\hat{\pi}_i}{n_k} \,. \tag{3.18}$$

Eventually, the Pearson chi-square statistic is determined by

$$\hat{C} = \sum_{k=1}^{g} \frac{\left(o_k - n_k \overline{\pi}_k\right)^2}{n_k \overline{\pi}_k \left(1 - \overline{\pi}_k\right)} \,, \tag{3.19}$$

with a (*g*-2) degree of freedom.

The Hosmer-Lemeshow statistic is usually computed using $g = 10$ groups. However, because the restriction of $g > p + 1$, where p is the number of covariates in the proposed model, arose in the simulation process that was demonstrated by Lemeshow and Hosmer (1982), $g = 50$ is used in the current study, as more than 40 covariates are considered in the proposed logistic regression model.

3.5.4 CONFIDENCE INTERVAL ESTIMATION

The confidence interval estimator for the logit is a bit more complicated for the multiple variable mode. The general expression for the estimator of the logit for a model containing k covariates is $\hat{g}(x) = x'\hat{\beta}$ (3.20)

where the vector$\beta' = (\hat{\beta}_{\circ}, \hat{\beta}_{1,\ldots,}\hat{\beta}_k)$ denotes the estimator of the $k+1$ coefficients and the vector $x' = x_0, x_1, x_{2,\ldots,}x_k$ represents the constant and a set of values of the k – covariates in the model, where $x_0 = 1$.The expression of the variance of the estimator of the logit in (3.21) is

$$\hat{var}[\hat{z}(x)] = \sum_{j=0}^{k} x_j^2 \, \hat{var}(\hat{\beta}_j) + \sum_{j=0}^{k} \sum_{k=j+1}^{k} 2x_j x_p \, \hat{cov}(\hat{\beta}_j \hat{\beta}_p) \qquad (3.21)$$

We can express this result much more concisely by using the matrix expression for the estimator of the variance of the estimator of the coefficients. From the expression for the observed information matrix, we have that

$$\hat{var}(\hat{\beta}) = (\mathrm{X}'\mathrm{VX})^{-1} \qquad (3.22)$$

The equivalent expression for the estimator in (3.21) is

$$\hat{var}[(\hat{z}(x))] = x' \hat{var}(\hat{\beta}) x \qquad (3.23)$$

3.5.5 ODDS RATIO

In order to interpret this result we need to introduce and discuss measure of association termed the odds ratio.

The possible values of the logistic probabilities may be conveniently displayed in a 2×2 as shown in Table 3.3.

Table 3.3 Values of the Logistic Regression Model When the Predictor Variable Is Dichotomous

	Predictor Variable (X)	
Outcome Variable (Y)	$x = 1$	$x = 0$
$y = 1$	$\pi(1) = \frac{e^{\beta_0+\beta_1}}{1+e^{\beta_0+\beta_1}}$	$\pi(0) = \frac{e^{\beta_0}}{1+e^{\beta_0}}$
$y = 0$	$1-\pi(1) = \frac{1}{1+e^{\beta_0+\beta_1}}$	$1-\pi(0) = \frac{1}{1+e^{\beta_0}}$
Total	1.0	1.0

The odds of the outcome being present among individuals with $x = 1$ is defined as $\pi(1)/[1-\pi(1)]$. Similarly, the odds of the outcome being present among individuals with x = 0 is defined as $\pi(0)/[1-\pi(0)]$. The odds ratio, denoted *OR*, is defined as the ratio of the odds for $x = 1$ to the odds for $x = 0$, and is given by the equation

$$OR = \frac{\pi(1)/[1-\pi(1)]}{\pi(0)/[1-\pi(0)]} \tag{3.24}$$

Substituting the expression for the logistic regression model shown in Table 3.3 into (3.24) we obtain:

$$OR = \frac{\left(\frac{e^{\beta_0+\beta_1}}{1+e^{\beta_0+\beta_1}}\right)\Big/\left(\frac{1}{1+e^{\beta_0+\beta_1}}\right)}{\left(\frac{e^{\beta_0}}{1+e^{\beta_0}}\right)\Big/\left(\frac{1}{1+e^{\beta_0}}\right)}$$

$$= \frac{e^{\beta_0+\beta_1}}{e^{\beta_0}}$$

$$= e^{(\beta_0+\beta_1)-\beta_0}$$

$$= e^{(\beta_1)}$$

Hence, for logistic regression with dichotomous predictor variable coded 1 and 0, the relationship between the odds ratio and the regression coefficient is

$$OR = e^{\beta_1} \tag{3.25}$$

Nevertheless, if the coding scheme is different from the (0, 1) then the odds ratio formula needs to be modified, but for the purpose of this study all the dichotomous variables will be coded using the (0, 1) coding scheme.

The simple relationship between the coefficient and the odds ratio is the fundamental reason why logistic regression has proven to be such a powerful analytic research tool. The odds ratio is a measure of association which has found a wide use, especially in epidemiology, as it approximates how much more likely (or unlikely) it is for the outcome to be present among those with $x = 1$ than among those with $x = 0$.

The interpretation given for the odds ratio is based on the fact that in many instances it approximates a quantity called the relative risk. This parameter is equal to the ratio $\frac{\pi(1)}{\pi(0)}$. It follows from (3.25) that the odds ratio approximates the relative risk if $[1 - \pi(0)]/[1 - \pi(1)] \approx 1$. This holds when $\pi(x)$ is small for both $x = 1$ and 0.

A $100(1 - \alpha)\%$ confidence interval (CI) estimate for the odds ratio is obtained by first calculating the endpoint of a confidence interval for the coefficient, β_1, and then exponentiating these values. In general, the endpoints are given by the expression

$$exp\left[\hat{\beta}_1 \pm Z_{1-\frac{\alpha}{2}} \times S\hat{E}(\hat{\beta}_1)\right]$$

Because of the importance of the odd ratio as a measure of association, many software packages automatically provide point and confidence interval estimates based on the

exponentiation of each coefficient in a fitted logistic regression model. These quantities provide estimates of odds ratios of interest in only few special cases (e.g. a dichotomous variable coded zero or one that is not involved in any interactions with other variables).

3.6 PROBIT REGRESSION MODEL

Probit regression, also called a probit model, is used to model dichotomous or binary outcome variables. Probit models simply use the cumulative Gaussian normal distribution rather than the logistic function for calculating the probability of being in one category or not. In the probit model, the inverse standard normal distribution of the probability is modeled as a linear combination of the predictors. The probit link is another function suitable for fitting regression and ANOVA models to binomial data.

Probit regression models the probability that Y=1 using the cumulative standard normal distribution function, evaluated at

$$Z = \beta_0 + \beta_1 x_1 + \ldots + \beta_k x_k$$

Thus, the probit linear regression model can be written as

$$\Pr(Y = 1 \mid X) = \Phi(\beta_0 + \beta_1 x_1 + \ldots + \beta_k x_k) \qquad (3.26)$$

Equation (3.26) can be transformed to

$$= \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{\beta_0 + \beta_1 x_1 + \ldots + \beta_k x_k} e^{-\frac{1}{2} z^2} dz$$

Where Φ is the cumulative normal distribution function.

- $z = \beta_0 + \beta_1 x_1 + \ldots + \beta_k x_k$ is the "z-value" or "z-index" of the probit model.

where $\beta_{0;}\, \beta_{1,\ldots}\, \beta_k$ are the parameters to be determined and the symbol Φ is simply the cumulative standard distribution, while the lower case symbol, φ, as before, represents the standard normal density function. We maximize the same log-likelihood function.

Note that this gives the model in the form of the inverse link. You can write the probit model in terms of the link function as

$$\text{Probit}(\pi) = \Phi^{-1}(\pi) = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \ldots + \beta_k X_k, \qquad (3.27)$$

where $\Phi^{-1}(\pi)$ means the *Z* value such that the area under the curve less than Z is π.

Interpretation of the probit coefficients is, in some senses, rather easier than the logit. The regression coefficients of the probit model are effects on a cumulative normal function of the probabilities that *Y = 1* (i.e. the probability that a neonate is a having LBW).

CHAPTER FOUR

4.0 RESULTS AND DISCUSSION

Logistic regression and probit models were used to identify the risk factors associated with LBW using *SAS*. Frequency, mean and standard deviation were obtained for continuous variables while the categorical variable was assessed by computing frequencies. Odds ratio (OR) and 95% confidence interval (CI) for each variable of interest were calculated. *P*-values were calculated by likelihood ratio test for the significance of the beta coefficients; *P-values* of 0.05 and 0.1 were considered significant for all the predictor variables in the model. Linear regression was also computed to identify factors associated with mean birth weight. Data gathered from the study were analysed according to WHO definition of LBW (< 2.5 kg).

4.1 DESCRIPTIVE ANALYSIS

The descriptive analysis examines the incidence of birth weight and low birth weight on selected variables. A total of 221 infants were eligible and all satisfied the inclusion criteria and so were recruited from January 2010 to December, 2010.

From Table 4.1, the Mean birth weight of the infants was 2.812 kg. Mean gestation age of the sample is 36.39 weeks and mean weight of women whose infants were studied was 57.633kg. Majority of the LBW infants 45 (78.9%) were preterm gestational age infants and 12 (21.1%) were at-term gestational age. The mean age of women whose infants were studied is 24.57 years ranging from 14 years to 44 years.

From Table 4.2, there were 95 male infants which represent about 43% and 126 female infants which also represent about 57% of the study population The birth weight were categorised into high and low based on the WHO standards. There were a total of 57 LBW infants which represents about 26%, with males accounting for 23 representing

about 10% and 34 Females representing 15% and 164 infants representing about 74% were normal birth weight as shown from Table 4.2.

Most of the women whose infants were studied were in the age group 20-25 years (39%) followed by women aged <20 years (23.2%),with 20.1% aged between 26-30 years and above 30 years (17.7%) (*Appendix A.9*).

Out of the total number of low birth weight infants seen, 31 (54.4%) of them were delivered by mothers in the age group 20 -30 years, 11(19.3%) of LBW infants were delivered by women in age group less than 20 years and about 15(26.3%)LBW infants were delivered by women of age above 30 years .

Many of the by mothers whose occupation was farming had the highest LBW infants (17.65%), followed by mothers who were students (5.43%). Petty traders were in the minority (2.71%). *(Appendix A.2)*

TABLE 4.1 *Summary statistics of selected variables*

Variable	Mean	Minimum	Maximum	Std Dev	Lower 95% CL for Mean	Upper 95% CL for Mean
AGE(years)	24.57	14.000	44.000	6.237	22.929	26.210
BWT	2.812	1.000	4.500	0.642	2.727	2.897
GAGE	36.398	26.000	48.000	3.131	35.983	36.813
MWT	57.633	42.000	75.000	5.539	56.899	58.368

TABLE 4.2 ***Test for independence (sex and low birth weight)***

SEX	LOW		Total
	>=2500g	<2500g	
Male	72	23	95
%	32.58	10.41	42.99
Female	92	34	126
%	41.63	15.38	57.01
Total	164	57	221
%	74.21	25.79	100.00

The chi-square test for independence was performed to assess for the association between the outcome of Low and some variables such as Sex, Occupation and Hypertension. In other words, it is seen whether the proportion of low birth weight is the same in each category. From Table 4.2, Chi-square test indicates that the outcome of LBW and Sex is predictor (P=0.6408)

Also from the contingency tables in appendix A, the usual Chisquare tests were performed to see if the outcome of low birth weights is associated with hypertension and occupation. In both cases there were no significant results between low birth weight and the predictors.

TABLE 4.3 *Parameter estimates and Standard errors with associated p-values for Multiple regression model*

Variable	DF	Parameter Estimate	Standard Error	t Value	Pr > |t|
Intercept	**1**	-0.46648	0.61467	-0.76	0.4487
AGE	**1**	-0.03027	0.03414	-0.89	0.3763
SEX	**1**	-0.03862	0.08034	-0.48	0.6312
GAGE	**1**	0.08362	0.01311	6.38	<.0001
FARMING	**1**	-0.09163	0.13730	-0.67	0.5053
STUDENT	**1**	-0.34321	0.17011	-2.02	0.1149
MWT	**1**	0.00771	0.00725	1.06	0.2889
HT	**1**	0.10105	0.20324	0.50	0.6196

4.2.1 MULTIPLE LINEAR REGRESSION RESULTS

Tables 4.3 show the result of linear regression analysis investigating the factors affecting mean Birth weight among neonates in St Martins Hospital. The data set contains six predictor variables: Mothers Age(Age), categories of Occupation(farming and student), Hypertension(HT), Sex, Mothers weight(MWT) and gestation age(GAGE). The response variable was the mean actual birth weight(BWT).

The R^2 value of 0.20 shows that about 20% of the variability in birth weight data is accounted for (appendix A.4). This reveals that the regression model may not be appropriate for prediction purpose. This may mean that some important variables were not considered.

The result of the regression analysis as shown in appendix A.4 the global test for model significant is statistically significant (F= 7.29, P=<0.0001). The variable that showed significant with mean birth weight is Gestation Age (p value of <0.0001) as shown in Table 4.1. However, Age of the mother, sex of the baby, mother`s age ,weight of the mother at last menstrual period as well as the history of hypertension were not seen as

having an effect on the mean actual Birth weight in this model as shown in table 4.3 above. The fitted multiple regression model is:

$\hat{BWT}$ = -0.46648 - 0.03027*AGE - 0.03862*SEX + 0.08362*GAGE - 0.09163 *FARMING - 0.34321* STUDENT + 0.00771* MWT + 0.10105 *HT

TABLE 4.4 *Parameter estimates and Standard errors with associated p-values and odd ratios for Multiple Logistic regression model*

Parameter		Estimate	Standard Error	Wald 95% Confidence Limits		Pr > Chi Sq	Exp(Est)
Intercept		17.5752	4.8173	8.1334	27.0170	0.0003	60771156
AGE		0.4119	0.2526	-0.0832	0.9069	0.1030	1.510
SEX		0.1460	0.5344	-0.9014	1.1934	0.7847	1.157
GAGE		-0.2589	0.1080	-0.4706	-0.0472	0.0165	0.772
OCCU	**FARMIG**	0.1331	1.2605	-2.3374	2.6035	0.9159	0.807
OCCU	**STUDENT**	0.9094	1.4053	-1.8450	3.6638	0.5176	1.754
OCCU	**TRADER**	0.0000	0.0000	0.0000	0.0000	.	-
MWT		0.0691	0.0505	-0.0298	0.1681	0.1707	1.072
HT		0.9862	1.2934	-1.5487	3.5212	0.4457	2.681

4.2.2 MULTIPLE LOGISTIC REGRESSION RESULTS

Logistic regression was performed to investigate the factors that contribute to the risk of Low Birth Weight among neonates. The model contained six predictor variables, however only gestation age was statistically significant at 5% level of significance with p value of 0.0165 as depicted from table 4.4 above. Age of the mother is however slightly significant at 10% level of significance (p =0.103, OR= 1.510). From table 4.4 above, the non-significant variables are: sex of the child, Mother's weight, occupation of the mother and Hypertension status of the mother.

This means that a mother with gestation age less than 37 weeks has 1.3 times chance giving birth to an infant with low birth weight than at-term (gestation age greater than or equal to 37 weeks gestation age).

The fitted multiple logistic regression model is:

Logit(π) = 17.5752 + 0.4119*AGE + 0.1460*SEX – 0.2589*GAGE +0.1331 *FARMING + 0.9094* STUDENT + 0.0691* MWT + 0.9862 *HT

TABLE 4.5 *Parameter estimates and Standard errors with associated p-values for Multiple Probit regression model*

Parameter		Estimate	Standard Error	Wald 95% Confidence Limits		Chi-Square	Pr > ChiSq
Intercept		7.7412	2.1975	3.4342	12.0482	12.41	0.0004
AGE		0.2630	0.1289	0.0103	0.5157	4.16	0.0414
SEX		0.2141	0.2843	-0.3432	0.7714	0.57	0.4514
GAGE		-0.1492	0.0514	-0.2499	-0.0485	8.44	0.0037
OCCU	**FARMING**	0.3718	0.5967	-0.7977	1.5413	0.39	0.5332
OCCU	**STUDENT**	0.8129	0.6843	-0.5282	2.1541	1.41	0.2348
OCCU	**TRADER**	0.0000	0.0000	0.0000	0.0000	.	.
MWT		0.0457	0.0268	-0.0068	0.0982	2.91	0.0883
HT		0.6358	0.7311	-0.7971	2.0686	0.76	0.3845

4.2.3 MULTIPLE PROBIT REGRESSION RESULTS

The multiple probit model was also used to examine the relationship between the likelihood of low birth weight and other variables. The results of the probit model were similar to the logit model except that this model gave more predictor variables to be significant at 5% and 10% levels of significance.

Gestation age and Age of the mother were statistically significant at 5% as depicted from table 4.5 above. The weight of the mother gained after last menstrual period (MWT) in table 4.5, although was not significant at 5% would be significant at 10%. From table 4.5, it can also be seen that the non-significant predictor variables at both 5% and 10% level of significance are: sex of the child, levels of occupation of the mothers and Hypertension status of the mother.

The fitted probit model is

Probit(π) = $\phi^{-1}(\pi) = 7.7412 + 0.2630$*AGE + 0.2141*SEX – 0.1492*GAGE +0.3718 *FARMING + 0.8129* STUDENT + 0.0457* MWT + 0.6358 *HT

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

5.1 SUMMARY

The study results highlight some factors that are significantly associated with mean actual birth weight and LBW. The data used for this study consist of eight predictor variables; age, occupation, hypertension, actual birth weight, low birth weight, sex, mothers weight and gestation age.

The preliminary results showed the results from our independent test of LBW with some predictor variables (hypertension, sex and occupation). It was reported that the predictors hypertension, sex and occupation were not statistically significant to our outcome variable, LBW.

Findings from this study show that the modal age group of the women who gave birth at the St Martin's Hospital was 20 to 25 years age (39%). Although this is the recommended reproductive age group, was responsible for the about 45.6% of low birth weight infants followed by Mothers aged < 20 years with 19.3% (*appendix A.9.).* This indicates that LBW incidence is largely a young adult phenomenon.

The study also indicates that about 60% of LBW cases were found among female infants. The mean actual birth weight of 2812 grams observed in this study. In the study it was found out that more than three quarters (78.9%) of all low birth infants to be pre-term

(gestation age < 37 weeks) and 21.1 % of low birth weight deliveries were at-term (gestation age >= 37 weeks).

The findings also suggest that 68.4% of low birth weight infants observed among mothers who were farmers.

From the Multiple linear Regression model, Gestation age was significant with the mean actual birth weight. The regression coefficient of gestation age shows that there is a positive relationship between gestation age and mean birth weight. The regression coefficient estimate of gestation age (0.084) means that for one unit increase in gestation age, the mean actual birth weight would increase by 0.084 units, when other predictor variables remains identical or unchanged.

The other predictor variables age, categories of occupation, sex, mothers weight and hypertension were not statistically significant. This may mean that the mean actual birth weight do not depend lineally on these predictor variables.

From the Logistic regression model, Gestation age was significant with the Low birth weight. The regression coefficient of gestation age shows that there is a negative relationship between gestation age and Low birth weight indicating that the higher the gestation age the lesser the chance giving birth to an infant with low birth weight. This may mean that preterm infants were 1.3 times more likely to have low birth weights than full term infants.

Maternal age also showed positive association with low birth weight in the logistic model at 10% level of significance. The findings suggest that as maternal age increase, the risk of LBW also increase. The other predictor variables categories of occupation, sex, mothers' weight and hypertension were not statistically significant. This may mean that the Low birth weight do not depend on these predictor variables.

According to the probit model results of this study, gestation age also shows a significant negative relationship with LBW. Age of the mother as expected shows a significant positive relationship with LBW implying that the infants of younger mothers have lesser chance to get LBW than that of older mothers. Low birth weight and maternal weight also showed relationship in the probit model. The other predictor variables categories of occupation, sex and hypertension were not statistically significant. This may mean that the Low birth weight do not depend on these predictor variables.

5.2 CONCLUSION

From the whole study, the following conclusions are drawn from both our preliminary results and the results from our model in achieving our objectives;

The independent test of LBW versus some predictor variables showed that occupational status of the mother, sex of the infant and hypertension status were not associated with an infant's risk of being LBW.

The results from the three models indicate that gestation age was the predictor that has an influence on birth outcome. This may mean that the mean actual birth weight depends lineally on gestation age.

We also conclude from the probit and logit models that gestation age, mother's age and weight of the mother at last menstrual period are risk factors of low birth weight (LBW).

It was observed that predictors like sex, occupation and hypertension status of the mother were not significant to the study.

5.3 RECOMMENDATIONS

Based on the findings from this study, the following recommendations are made to reduce the risk of an infant getting low birth weight.

It is recommended that Adolescent nutrition and reproductive health behaviours, maternal malnutrition for improved pregnancy outcomes and reversal of negative implications of LBW incidence in Amansie west district and for that matter Ghana, must be effectively addressed.

In addition, there is a need to explore the extent to which the incidence of low birth weight could be reduced by giving to all prospective mothers' access to antenatal care but keeping constant their other socio-economic characteristics.

There is also the need for further research on first, an in-depth study on the impact of several factors like multiple births, parity, number of ante-natal visits, mother's educational levels qualitative studies on mothers' cultural and behavioural factors determining on birth size is necessary.

REFERENCES

1. Adelusi B, Ladipo O.A (1976). Preterm and other Infants with low birth weight in Ibadan. Trop. Geogr. Med., 28: 316-322.

2. ACC/SCN (1999) Prevention of Foetal and Infant Malnutrition. Geneva: UN ACC/SCN Working Group.

3. ACC/SCN (2000) *Fourth Report on the World Nutrition Situation*. Geneva: ACC/SCN in collaboration with IFPRI.

4. Andersson, H. W., S. J. Gotlieb and K. G. Nelson (1997) Home environment and cognitive abilities in infants born small-for-gestational-age. In: *Acta Obstetrica et Gynecologica Scandinavia Supplement.* 165, 76,82-6.

5. Angelsen, N. K., T. Vik, G. Jacobsen and L. S. Bakketeig (2001) Breast feeding and cognitive development at age 1 and 5 years. In. *Archives of Disease in Childhood.* 85,183-88.

6. Arifeen S.E. (1997) Birth weight, intrauterine growth retardation and prematurity: a prospective study of infant growth and survival in the slums of Dhaka, Bangladesh Doctor of Public Health dissertation, Johns Hopkins University, Baltimore MD.

7. Ashworth A, Feachem R.G. (1985) Interventions for the control of diarrhoeal diseases among young children: prevention of low birth weight. *Bulletin of the World Health Organization* 63:165-184.

8. Ashworth A. (1998) Effects of intrauterine growth retardation on mortality and morbidity in infants and young children. *European Journal of Clinical Nutrition* 52(Supplement 1):S34-S42.

9. Astolfi, P. and L. A. Zonta (2002) Delayed maternity and risk at delivery. In: *Paediatric and Perinatal Epidemiology*. 16, 67-72.

10. Bakketeig L.S. (1998) Current growth standards, definitions, diagnosis and classification of fetal growth retardation. *European Journal of Clinical Nutrition* 52(Supplement 1):S1-S4.

11. Barker D.J.P, Osmond C. (1986) Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet* I:1077-1081.

12. Barker D.J.P. (1998) *Mothers, Infants and Health in Adult Life*. Edinburgh: Churchill Livingstone.

13. Barros F.C., Huttly S.R.A., Victoria C.G., Kirkwood B.R., Vaughan J.P. (1992) Comparison of the causes and consequences of prematurity and intrauterine growth retardation: a longitudinal study in southern Brazil. *Pediatrics* 90:238-244.

14. Basso, O., J. Olsen, A. M. T. Johansen and K. Christensen (1997) Change in social status and risk of low birth weight in Denmark: population based cohort study. In: *British Medical Journal*. 315: 1498-1502.

15. Basso, O., J. Olsen and K. Christensen (1998) Risk of preterm delivery, low birth weight and growth retardation following spontaneous abortion: a registry-based study in Denmark. In: *International Journal of Epidemiology.* 27, 642-46.

16. Blondel, B., M. D. Kogan, G. R. Alexander, N. Dattani, M. S. Kramer, A. Macfarlane and S. W. Wen (2002) The impact of the increasing number of multiple births on the rates of preterm birth and low birth weight: an international study. In: *American Journal of Public Health*. 92, 8, 1323-30.

17. Bréart, G. (1997) Delayed childbearing. In: *European Journal of Obstetrics and Genecology and Reproductive Biology*. 75, 71-73.

18. Bukenya G., Barnes T., Nwokolo N. (1991) Low birthweight and acute childhood diarrhoea: evidence of their association in an urban settlement of Papua New Guinea. *Annals of Tropical Paediatrics* 11(4):357-362.

19. Cameron M., Hofvander Y. (1983) *Manual on Feeding Infants and Young Children*, ed. Series, Third ed. Oxford: Oxford University Press.

20. Carlson, E., J. M. Hoem and J. Rychtarikova (1999) Trajectories of fetal loss in the Czech Republic. In: *Demography*. 36, 3, 327-37.

21. Carlson, E. and J. M. Hoem (1999) Low-weight neonatal survival paradox in the Czech Republic. In: *American Journal of Epidemiology*. 149, 5, 447-53.

22. Cerqueiro M., Murtagh P., Halac A., Avila M., Weissenbacher M (1990) Epidemiologic risk factors for children with acute lower respiratory tract infections in Buenos Aires, Argentina: a matched case-control study. *Reviews of Infectious Diseases* 12(S8):S1021-1028.

23. Copper, R. L., R. L. Goldenberg, A. Das, N. Elder, M. Swain, G. Norman, R. Ramsey, P. Cotroneo, B. A. Collins, F. Johnson, P. Jones, A. Meir and the National Institute of Child Health and Human Development Maternal-Fetal Medicine Units

Network (1996) The preterm prediction study: maternal stress is associated with spontaneous preterm birth at less than thirty five weeks gestation. In: *American Journal of Obstetrics and Gynecology.* 175, 5, 1286-92.

24. Datta N, Kumar V, Kumar L, Singhi S (1987) Application of case management to the control of acute respiratory infections in low-birth-weight infants: a feasibility study. *Bulletin of the World Health Organization* 65:77-82.

25. Dičkutė, J., Ž. Padaiga, V. Grabauskas, R. J. Nadišauskienė, V. Basys and A. Gaižaukienė (2004) Maternal socio-economic factors and the risk of low birth weight in Lithuania. In: *Medicina (Kaunas).* 40, 5, 475-82.

26. de Onis M., Blössner M. (1997) *WHO Global Database on Child Growth and Malnutrition*. Geneva: WHO.

27. de Onis M, Blössner M, Villar J (1998) Levels and patterns of intrauterine growth retardation in developing countries. *European Journal of Clinical Nutrition* 52(Suppl. 1):S5-S15.

28. Dreyfuss, M.L., Msamanga, G.I., Spiegelman, D., Hunter, D.J., Urassa, E.J.N., Hertzmark, E. & Fawzi, W.W. (2001) Determinants of low birth weight among HIV-infected pregnant women in Tanzania. *American Journal of Clinical Nutrition* 74, 814–826.

29. Foix-L'Hélias and B. Blondel (2000) Changes in risk factors of preterm delivery in France between 1981 and 1995. In: *Paediatric and Perinatal Epidemiology.* 14, 314 23.

30. Fonseca W, Kirkwood BR, Victoria CG, Fuchs SR, Flores JA, Misago C (1996) Risk factors for childhood pneumonia among the urban poor in Fortaleza, Brazil: a case-control study. *Bulletin of the World Health Organization* 74:199-208.

31. Forsdahl A. (1977) Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? *British Journal of Preventive Social Medicine* 31:91-95..

32. Forssas, E., M. Gissler, M. Sihvonen and E. Hemminki (1999) Maternal predictors of perinatal mortality: the role of birth weight. In: *International Journal of Epidemiology.* 28, 475-78.

33. Goldenberg R., Hoffman H., Cliver S. (1998) Neurodevelopmental outcome of small-for-gestational age infants. *European Journal of Clinical Nutrition* 52(S1):S54-S58.

34. Grantham-McGregor S.M. (1998) Small for gestational age, term infants, in the first six years of life. *European Journal of Clinical Nutrition* 52(Supplement 1):S59-S64.

35. Hack, M., D. J. Flannery, M. Schluchter, L. Cartar, E. Borawski and N. Klein (2002) Outcomes in young adulthood for very low birth weight infants. In: *New England Journal of Medicine.* 346, 3, 149-57.

36. Haelterman, E., G. Bréart, J. Paris-Llado, M. Dramaix and C. Tchobroutsky (1997) Effect of uncomplicated chronic hypertension on the risk of Small-for-Gestational Age Birth. In: *American Journal of Epidemiology.* 689-95.s

37. Hales C.N., Barker D.J.P., Clark P.M.S., Cox L.J., Fall G., Osmond C. et al. (1991) Fetal and infant growth and impaired glucose tolerance at age 64. *British Medical Journal* 303:1019-1022.

38. Hass J, Balcazar H, Caulfield L (1987) Variations in early neonatal mortality for different types of fetal growth retardation. *American Journal of Physical Anthropology* 76:467-473

39. Henriet, L. and M. Kaminski (2001) Impact of induced abortions on subsequent pregnancy outcome: the 1995 French national perinatal survey. In: *British Journal of Obstetrics and Gynaecology.* 108, 1036-42.

40.Hosmer, D.W., and Lemershow, S. (1980). A goodness-of-fit test for the multiple logistic regression model. *Communications in Statistics,* A10, 1043-1069.

41. Jaakkola, J. J. K. and M. Gissler (2004) Maternal smoking in pregnancy, fetal development and childhood asthma. In: *American Journal of Public Health.* 94, 136-40.

42. Jurjus, A.R. (1995) Low birth wight in Lebanon: morphological parameter and a health status indicator. *Eastern Mediterranean Health Journal* 1, 194–200.

43. Kallan, J. E. (1993) Race, intervening variables and two components of low birth weight. In: *Demography.* 30, 3, 489-506.

44. Kelly, Y. J., J. Y. Nazroo, A. McMunn, R. Boreham and M. Marmot (2001) Birth weight and behavioural problems in children: a modifiable effect? In: *International Journal of Epidemiology*. 30, 88-94.

45. Khoshnood, B., B. Blondel, C. de Vigan and G. Bréart (2004) Socioeconomic barriers to informed decision making: regarding maternal serum screening for down syndrome: results of the French National Perinatal Survey of 1998. In: *American Journal of Public Health.* 94, 3, 484-91.

46. Klingenberg, C., Olomi, R., Oneko, M., Sam, N. & Langeland, N. (2003) Neonatal morbidity and mortality in a Tanzanian tertiary care referral hospital. *Annals Paediatrics* 23, 293-299.

47. Kramer, M. S. (1987) Determinants of low birth weight: methodological assessment and meta-analysis. In: *Bulletin of the World Health Organizations.* 65, 5, 663-737.

48. Kramer M. S. (1998) Socioeconomic determinants of intrauterine growth retardation. *European Journal of Clinical Nutrition* 52(S1):S29-S33.

49. Lack, N., J. Zeitlin, L. Krebs, W. Kűnzel and S. Alexander (2003) Methodological difficulties in the comparison of indicators of perinatal health across Europe. In: *European Journal of Obstetrics and Genecology and Reproductive Biology*. 111, S33-44.

50. Larroque, B., M. Kaminski, P. Dehaene, D. Subtil, M. J. Delfosse and D. Querleu (1995) Moderate prenatal alcohol exposure and Psychomotor Development at Preschool Age. In: *American Journal of Public Health.* 85, 12, 1654-61.

51. Lawoyin T.O, Oyediran A.B.O. (1992). A Prospective Study on some Factors which Influence the Delivery of Low Birth Weight Infants in a Developing Country. Afr. J. Med. Sci., 21(1): 33-39.

52. Lemeshow, S., Teres, D., Avrunin, J. S., and Pastides, H. (1988). Predicting the outcome of intensive care uniy patients. *Journal of the American Statistical Association,* 83, 348-356.

53. Macfarlane, A. J., F. V. Price and E. G. Daw (1990) Infertility drugs and procedures. In: B. J. Botting, A. J. Macfarlane and F. V. Price, eds, *Three, four and more - A study of triplet and higher order births.* 46-58.

54. Martorell R, Ramakrishnan U, Schroeder DG, Melgar P, Neufeld L (1998) Intrauterine growth retardation, body size, body composition and physical performance in adolescence. *European Journal of Clinical Nutrition* 52(Supplement 1):S43-S53.

55. Murthy H.V.V. (1991). Birth weight and the associated factors-a review. *Ind J Soc*

Work L11, 151-156.

56. Prada J., Tsang R. (1998) Biological mechanisms of environmentally induced causes in IUGR. *European Journal of Clinical Nutrition* 52(S1):S21-S28

57. Raum, E., B. Arabin, M. Schlaud, U. Walter and F. W. Schwartz (2001) The impact of maternal education on intrauterine growth: a comparison of former West and East Germany. In: *International Journal of Epidemiology.* 30, 81-87.

58. Resnick, M. B., R. V. Gueorguieva, R. L. Carter, M. Ariet, Y. Sun, J. Roth, R. L. Bucciarelli, J. S. Curran and C. S. Mahan (1999) The impact of low birth weight, perinatal conditions and socio demographic factors on educational outcome in kindergarten. In: *Paediatrics.* 104, 6, e74.

59. Saigal, S., L. den Ouden, D. Wolke, L. Hoult, N. Paneth, D. L. Streiner, A. Whitaker and J. Pinto-Martin (2003) School-age outcomes in children who were extremely low birth weight from four international population-based cohorts. In: *Pediatrics.* 112, 4, 943-50.

60. Schieve, L., A., S. F. Meikle, C. Ferre, H. B. Peterson, G. Jeng and L. S. Wilcox (2002) Low and very low birth weight in infants conceived with use of assisted reproductive technology. In: *New England Journal of Medicine.* 346, 10, 731-37.

61. Singer, L., T. Yamashita, L. Lilien, M. Collin and J. Baley (1997) A longitudinal study of developmental outcome of infants with Bronchopulmonary Dysplasia and very low birth weight. In: *Pediatrics*. 100, 6, 987-93.

62. Smith, M. M., M. Durkin, V. J. Hinton, D. Bellinger and L. Kuhn (2003) Influence of breastfeeding on cognitive outcomes at age 6-8 years: follow-up of very low birth weight infants. In: *American Journal of Epidemiology.* 158, 11, 1075-82.

63. Stevens-Simon, C. and Orleans, M. (1999). Low birthweight prevention programs: the enigma of failure. Birth 26: 184-91.

64. Sweet, M. P., J. E. Hodgman, I. Pena, L. Barton, Z. Pavlova and R. Ramanathan (2003) Two year-outcome of infants weighing 600 grams or less at birth and born 1994 through 1998. In: *Obstetrics and Gynaecology*. 101, 1, 18-23.

65. Tough, S. C., C. Newburn-Cook, D. W. Johnston, L. W. Svenson, S. Rose and J. Belik. (2002) Delayed childbearing and its impact on population rate changes in lower birth weight, multiple birth, andpreterm delivery. In: *Paediatrics*. 100, 3.

66. UNICEF-WHO (2004) United Nations Children's Fund and World Health Organisation, (2004) *Low Birth Weight; Country and Global Estimates*. UNICEF, New York

67. Vangen, S., C. Stoltenberg, R. Skjaerven, P. Magnus, J. R. Harris and B. Stray-Pedersen (2002) The heavier the better? Birth weight and perinatal mortality in different ethnic groups. In: *International Journal of Epidemiology*. 31, 654-660.

68. Vik, T., L. S. Bakketeig, K. U. Trygg, K. Lund-Larsen and G. Jacobsen (2003) High caffeine consumption in the third trimester of pregnancy: gender-specific effects on fetal growth. In: *Paediatric and Perinatal Epidemiology*. 17, 324-31.

69. Villar J, Belizán J.M (1982). The relative contribution of prematurity and fetal growth retardation to low birth weight in developing and developed societies. *American Journal of Obstetrics and Gynecology* 143:793-798.

70. Westwood, M., M. S. Kramer, D. Munz, J. M. Lovett, and G. V. Watters (1983) Growth and development of full-term nonasphyxiated smallfor- gestational-age newborns: Follow-up through adolescence. In: *Pediatrics*. 71, 376-82.

71. WHO (1997) National reports on the third evaluation of the implementation of "Health for All" strategies. New Delhi: WHO Global Database.

72. Yang, H., M. S. Kramer, R. W. Platt, B. Blondel, G. Bréart, I. Morin, R. Wilkins and R. Usher (2002) How does early ultrasound scan estimation of gestational age lead to higher rates of preterm birth?. In: *American Journal of Obstetrics and Gynaecology.* 1, 186, 3, 433- 7.

73. Zarén, B., G. Lindmark and P. Bergsjø (1997) Hemoconcentration in smoking mothers is associated with impaired fetal growth. In: *Acta*n *Obstetricia et Gynecologica Scandinavica.* 76, 933-41.

74. Zarén, B., G. Lindmark and L. Bakketeig (2000) Maternal smoking affects fetal growth more in the male fetus. In: *Paediatric and Perinatal Epidemiology.* 14, 118-26.

75. Zeitlin, J. A., M. J. Saurel-Cubizolles, P. Y. Ancel and the EUROPOP Group (2002) Msarital status, cohabitation, and the risk of preterm birth in Europe: where births outside marriage are common and uncommon. In: *Paediatric and Perinatal Epidemiology.* 16, 124-130.

APPENDICES

APPENDIX A

Table A.1 ***Test for independence (hypertension vs low birth weight)***

HT	LOW		Total
	NO	YES	
NO	157	55	212
%	71.04	24.89	95.93
YES	7	2	9
%	3.17	0.90	4.07
Total	164	57	221
	74.21	25.79	100.00

Statistics for Table of HT by LOW

Statistic	DF	Value	Prob
Chi-Square	1	0.0625	0.8026
Likelihood Ratio Chi-Square	1	0.0644	0.7996

Table A.2 T*est for independence (occupation vs low birth weight)*

OCCU	LOW		Total
	no	yes	
FARMING	131	39	170
%	59.28	17.65	76.92
STUDENT	18	12	30
%	8.14	5.43	13.57
TRADER	15	6	21
%	6.79	2.71	9.50
Total	164	57	221
%	74.21	25.79	100.00

Statistics for Table of OCCU by LOW

Statistic	DF	Value	Prob
Chi-Square	2	3.9707	0.1373
Likelihood Ratio Chi-Square	2	3.7039	0.1569

Table A.3 Statistics for Table of SEX by LOW

Statistic	DF	Value	Prob
Chi-Square	1	0.2177	0.6408
Likelihood Ratio Chi-Square	1	0.2185	0.6402

TABLE A.4 Analysis of variance table for regression model

Analysis of Variance					
Source	**DF**	**Sum of Squares**	**Mean Square**	**F Value**	**Pr > F**
Model	7	17.53962	2.50566	7.29	<.0001
Error	213	73.22825	0.34379		
Corrected Total	220	90.76787			

R-SQUARE=0.20

Table A.5: ***Model fit Statistics for Logistic regression***

Model Fit Statistics		
Criterion	**Intercept Only**	**Intercept and Covariates**
AIC	254.324	115.204
SC	257.722	145.788
-2 Log L	252.324	97.204

Testing Global Null Hypothesis: BETA=0			
Test	**Chi-Square**	**DF**	**Pr > ChiSq**
Likelihood Ratio	155.1194	8	<.0001
Score	110.5747	8	<.0001
Wald	43.4891	8	<.0001

TABLE A.6: ***Odd ratio estimates of logistic regression model***

Odds Ratio Estimates			
Effect	**Point Estimate**	**95% Wald Confidence Limits**	
AGE	1.510	0.920	2.477
SEX	1.157	0.406	3.298
BWT	0.003	<0.001	0.020
GAGE	0.772	0.625	0.954
OCCU FARMING vs TRADER	1.142	0.097	13.511
OCCU STUDENT vs TRADER	2.483	0.158	39.008
MWT	1.072	0.971	1.183
HT	2.681	0.213	33.826

TABLE A.7 ***criteria for assessing goodness of fit for logistic regression***

Criterion	**DF**	**Value**	**Value/DF**
Deviance	212	97.2045	0.4585
Scaled Deviance	212	97.2045	0.4585
Pearson Chi-Square	212	2065.5977	9.7434
Scaled Pearson X2	212	2065.5977	9.7434
Log Likelihood		-48.6022	

TABLE A.8 criteria for assessing goodness of fit for probit (normit) regression

Criterion	**DF**	**Value**	**Value/DF**
Deviance	212	104.7543	0.4941
Scaled Deviance	212	104.7543	0.4941
Pearson Chi-Square	212	1836.6472	8.6634
Scaled Pearson X2	212	1836.6472	8.6634
Log Likelihood		-52.3772	

Table A.9. Summary of dataset

Variable	Birth weight		Total
	Birth weight>=2.5kg	**`Birth weight** < 2.5 kg	
Sex of the baby Male	72(32.6%)	23(10.4%)	95(43.0%)
Female	92(41.6%)	34(15.4%)	126(57.0%)
Gestation Age < 37 wks	81(36.7%)	45(20.4%)	126(57.0%)
>= 37wks	83(37.6%)	12(5.4%)	95(43.0%)
Hypertension No	157(71.0%)	55(2.5%)	212(95.9%)
Yes	7(3.2%)	2(1.0%)	9(4.1%)
Occupation *farmer*	131(59.3%)	39(17.6%)	170(76.9%)
Trader	15(6.8%)	6(2.7%)	21(9.5%)
Student	18(8.1%)	12(5.4%)	30(13.6%)
Age of mother < 20 yrs	38(17.2%)	11(5.0%)	49(22.2%)
20-25	64(30.0%)	26(11.8%)	90(40.7%)
26-30	33(14.9%)	5(2.3%)	38(17.2%)
31-34	12(5.4%)	8(3.6%)	20(9.0%)
> =35	17(7.7%)	7(3.2%)	24(10.9%)