



## Effect of Birth Weight and Mode of Feeding During Early Infancy on Clinical Indicators of Obesity and Lipid Profile in Adulthood

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### Authors' contributions

This work was carried out in collaboration between all authors. Authors OJ and APKN designed the study, wrote the protocol, and wrote the first draft of the manuscript. Authors JLN and JTN managed the literature searches and author OJ managed the analyses of the study. Authors DK and GBAK performed the statistical analysis. All authors read and approved the final manuscript.

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### ABSTRACT

**Aims:** To study effect of birth weight and mode of feeding during early infancy on clinical indicators of obesity and lipid profile in adulthood.

**Place and Duration of Study:** The study was conducted on 260 adults aged 21-31 years censured in Yaoundé between February and March 2012.

**Methodology:** The study was transversal and retrospective. Data such as birth weight, type of breastfeeding and duration of breastfeeding was collected. Participants also followed a food diary for a week.

Anthropometric measurements and cardiovascular risk factors such as total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides and blood pressure were assessed.

**Results:** There were no significant difference between LBW group and NBW. In HBW group weight, BMI and lean mass were significantly ( $p < .05$ ) higher than in NBW group. Body fat was significantly ( $p < .05$ ) high among NBW compare to HBW. These results

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were confirmed by binary regression for body fat .918 (.852-.988) and lean mass 1.100 (1.039-1.164). Relation between birth weight and body fat showed an inverse and significant ( $P < .05$ ) correlation. Participants with low birth weight (LBW) had a concentration of total plasma cholesterol significantly higher than participants with normal birth weight (NBW) ( $p < .05$ ) or High birth weight (HBW) ( $p < .05$ ). Furthermore, a significant inverse correlation was noted between the total plasma cholesterol ( $P < .01$ ), LDL cholesterol ( $P < .05$ ) and the birth weight. The plasmatic total cholesterol ( $p < .05$ ) and LDL cholesterol ( $p < .05$ ) of participants who had mixed feeding was also higher compared to those who have been under exclusive breastfeeding for 6 months. **Conclusion:** birth weight and mode of feeding during early infancy have effects on clinical indicators of obesity and lipid profile in adulthood.

*Keywords: Birth weight; obesity; adulthood; lipid profile; breastfeeding; clinical indicators.*

## ABBREVIATIONS

*LBW: low birth weight; NBW: normal birth weight; HBW: high birth weight; BMI: body mass index; LDL: Low density lipoprotein cholesterol; HDL: high density lipoprotein cholesterol; CT: total cholesterol; TG: triglycerides; CVD: cardiovascular disease.*

## 1. INTRODUCTION

Chronic degenerative diseases are largely regarded as diseases of affluence. Chronic diseases are associated with sedentary lifestyle and high calorie that characterize many industrialized countries. However, some believe that these chronic diseases could also be due to poverty, particularly poverty early in life and during fetal development. This hypothesis is particularly interesting given that ischemic heart disease are likely to be the leading cause of death and disability worldwide by 2020 [1].

For some years, the 'fetal origins hypothesis' on early causes of later diseases has become one of the most promising theoretical frameworks in medicine. Especially, birth weight has been suggested and used as basic indicator to establish these highly influential concepts [2]. A high birth weight has been suggested to program an increased risk of later obesity, as measured by body mass index (BMI; in kg/m<sup>2</sup>) [3]. The association between birth weight and BMI, however, contradicts considerable evidence that a high birth weight programs less susceptibility rather than greater susceptibility to cardiovascular disease (CVD) risk factors [4]. One hypothesis that could partially explain this paradox is that low birth weight is associated with programming of greater abdominal fat mass [5]. It seems that there is also a possibility of metabolic programming due to the mode of growth and nutritional intake early in life. Thus, the "catch up growth by accelerating the velocity of growth in weight and size after birth increases the metabolic risk [3]. The influence of early nutrition on long-term adiposity has focused on the possible protective role of breastfeeding. However, while there is little doubt that breast milk is the best source of nutrition for the newborn, whether breastfeeding has long-term health benefits remains controversial. A case-control study by Kramer [6] was one of the first studies to suggest breastfeeding protected against later obesity. Since then, as summarized recently in four systematic reviews [3], many population-based studies have confirmed an association between breastfeeding and lower risk of later adiposity.

In Cameroon, the rising trends in non communicable diseases have been documented for hypertension and diabetes, with a 2-5 and a 10-fold increase in their respective prevalence between 1994 and 2003. Magnitudes are much higher in urban settings, where increasing prevalence of overweight/obesity (by 54-82%) was observed over the same period. Thus, national-level prevention and control programs for chronic diseases (mainly diabetes and hypertension) have been established for more effective national-level tobacco control measures and food policies, as well as campaigns to promote healthy diets, physical activity and tobacco cessation [7]. Nothing have been done concerning early risk factors during infancy on obesity in adulthood why this study investigated the influence of birth weight and post natal mode of feeding during early infancy on clinical indicators of obesity and lipid profile in adulthood.

## **2. MATERIALS AND METHODS**

### **2.1 Protocol**

The study was cross-sectional study focusing on young adults between February and March 2012 in Yaounde, Cameroon.

The evaluation study was designed to include Cameroonian university students aged between 21-31 years at the Yaounde 1 university, campus of Ngoa Ekellé, Yaounde. The study was approved by the National Ethics Committee of Cameroon under the number 139/CNE/SE/09. All participants were informed about the purpose of the study and data collection procedures. Participation was on a volunteer basis. All participants signed the informed consent form. Exclusion criteria were pregnancy, weight loss of more than 5 kg during the last 6 months prior to the study and participation in other research projects and not physically malformed or suffered from any infirmity that might not enable the collection of anthropometric data. After recruitment, information on the student (birth weight, type of feeding practice received at birth, duration of maternal breastfeeding, the weaning age obtained over the form filled by their mothers. Among the 540 participants, 260 mothers of the participants included in the study provided complete informations of their birth weight and infant feeding practice.

### **2.2 The 7-days Energy Intake and Nutrients Evaluation**

The participants got written and oral instructions how to weigh and record all foods and beverages consumed in a food diary for 7 days, divided into 2 periods of 4 and 3 consecutive days, one week apart, including all days of a week. Each participant was provided with a food diary. Food dairies were analyzed using food table and the daily energy, proteins, carbohydrate, lipids intake were determined.

### **2.3 Physical Activity**

Physical activity was assessed with three non-consecutives 24-hour recalls [8]. The participants were asked about all the activities of the previous day, including time spent in bed, in various modes of transportation, for main (and secondary) occupations, for house chores, and for leisure activities. The different activities were classified in three groups: main occupation, transportation and leisure. In each group, the activities were then categorized according to intensity level according to the metabolic equivalents (METs), based on the compendium of physical activities [9]: light ( $< 3.0$  METs); moderate ( $3.0 \leq \text{METs} \leq 6.0$

METs), and vigorous (> 6.0 METs). Time devoted to each type of activity was also expressed as mean number of hours per day for each level of energy expenditure. The total daily hours of intense, moderate and light activity were computed.

## **2.4 Anthropometry and Body Composition**

Anthropometric measures were all taken by the first author. Weight was measured on a portable mechanical scale with a maximum capacity of 150 kg (Seca Model 761 Mechanical Personal Scale, Germany) to the nearest 0.1 kg and height was measured to the nearest of 0.5 cm with a stadiometer (Seca 214 Portable Height Rod, Germany). BMI (kg/m<sup>2</sup>) was computed (weight divided by height squared). The BMI cut-offs for overweight and general obesity was 25 and 30, respectively; underweight was defined as BMI < 18.5 [10]. While subjects were standing and breathing normally, waist circumference (WC) was measured to the nearest of 0.1 cm with a flexible nonstretch steel tape at the midpoint between the lower rib margin and the iliac crest. The average of two separate measures of WC was used in the analyses. Classification of participants according to birth weight was : < 2500 grs = low birth weight (LBW), [2500, 4000]= normal birth weight (NBW), > 4000grs= high birth weight (HBW). LBW and HBW were compared to NBW.

## **2.5 Blood Pressure**

Blood pressure was evaluated by auscultatory method using a stethoscope and a sphygmomanometer. The JNC 7 [11] criteria were used for blood pressure classification. Prehypertension is defined for systolic blood pressure/diastolic blood pressure range 120/80 and 139/89. Hypertension for systolic blood pressure/diastolic blood pressure higher than 140/90.

## **2.6 Laboratory Methods**

Blood samples were collected after a 12 h overnight fast into heparinized tubes. The concentrations of total cholesterol, triglycerides, HDL-cholesterol in plasma were measured using a commercial diagnostic kit (Cholesterol infinity, triglycerides Int, EZ HDL™ cholesterol, respectively) from SIGMA Diagnostics. LDL-cholesterol was calculated using the equation of Friedewald [12]. Abnormal values were TG > 2g/l, CT < 2g/l, LDL >1.3 g/l, HDL < 0.35 g/l.

## **2.7 Statistical Analysis**

Results were expressed as mean ± SEM. ANOVA test was carried out on the LBW, NBW and HBW patients and correlation were carried out using spearman correlation. LBW and HBW were comparing to NBW separately. Binary regression analysis of proportion was also used to compare LBW and HBW to NBW.

The software used for analysis was SPSS version 10.1 for windows.

### 3. RESULTS

#### 3.1 Characteristics of Studying Population

Our studying population was 260 students aged 21 to 31 years old with 128 wemens and 132 mens, 32 actually underweight, 176 normal and 62 overweight and obese. According to birth weight, 42 low birth weight were recorded, 182 normal birth weight and 36 high birth weight. Considering blood pressure 24 participants were suffering from systolic hypertension, 110 from systolic prehypertension, 10 from diastolic hypertension and 38 from diastolic prehypertension. Abnormal value of total cholesterol was encountered to 40 participants, HDL to 60 and LDL to 22 participants.

#### 3.2 Influence of Birth Weight on some Indicators of Obesity

##### 3.2.1 Influence of birth weight on some indicators of obesity in LBW group compare to NBW

We observed no significant difference in LBW group compare to NBW (Table 1).

**Table 1. Influence of birth weight on anthropometric parameter, body fat and blood pressure between LBW and NBW group**

	<b>LBW(42)</b>	<b>NBW(182)</b>
Age (years old)	24.23±1.39	25.42±7.40
weight (Kg)	60.98±10.41	63.30±9.77
height(cm)	158.70±2.15	164.01±18.84
BMI (Kg/m <sup>2</sup> )	22.67±3.01	22.87±2.82
Body fat (%)	25.12±12.18	23.43±7.28
Fat mass (Kg)	15.88± 9.85	14.79± 5.39
Lean mass(Kg)	45.10±8.70	48.41± 8.89
Waist (cm)	82.82±13.45	82.45±13.85
Hip (cm)	86.82±13.7	88±11.05
Waist to hip ratio	1.082±0.27	1.11±0.244
Bicipital skin folk thickness (mm)	10.05±3.24	11.06±6.60
Tricipital skin folk thickness (mm)	15.23±6.45	15.74±6.28
Suscapulary skin folk thickness (mm)	15.82±5.89	15.46±5.09
Supra iliac skin folk thickness (mm)	14.35±5.57	14.94±5.97

##### 3.2.2 Influence of birth weight on some indicators of obesity in HBW compare to NBW

Participants with HBW have weight, BMI and lean mass significantly (p<.05) high compare to NBW . Body fat was significantly (p<.05) higher in NBW group than HBW (Table 2).

**Table 2. Influence of birth weight on anthropometric parameter, body fat and blood pressure between NBW and HBW groups**

	<b>NBW(182)</b>	<b>HBW(42)</b>
Age (years old)	25.42±7.40	24.50±2.11
weight (Kg)	63.30±9.77	69.67±9.42*
height(cm)	164.01±18.84	170.13±7.79
BMI (Kg/m <sup>2</sup> )	22.87±2.82	25.77± 9.31*
Body fat (%)	23.43±7.28	19.30±6.91*
Fat mass (Kg)	14.79± 5.39	13.47±5.37
Lean mass(Kg)	48.41± 8.89	56.19± 8.83*
Waist (cm)	82.45±13.85	82.90±10.56
Hip (cm)	88±11.05	89.72±11.46
Waist to hip ratio	1.11±0.244	1.104±0.217
Bicipital skin folk thickness (mm)	11.06±6.60	9.68±2.60
Tricipital skin folk thickness (mm)	15.74±6.28	13.36±4.21
Suscapulary skin folk thickness (mm)	15.46±5.09	14.22±3.66
Supra iliac skin folk thickness (mm)	14.94±5.97	12.92±4.98

\*P<0.05; compare to NBW group

### **3.3 Influence of Birth Weight on Anthropometric Parameters According to BMI**

#### **3.3.1 Influence of birth weight on anthropometric parameters in actually under weight participants**

No significant differences were observed among individuals actually underweight.

#### **3.3.2 Influence of birth weight on anthropometric parameters in normal weight**

No significant differences were observed among individuals actually normal.

#### **3.3.3 Influence of birth weight on anthropometric parameter, body fat and blood pressure Between LBW and NBW in actually overweight and obese group**

The fat mass was significantly ( $p < 0.05$ ) higher among low birth weight compared to those with normal at birth (Table 3).

**Table 3. Influence of birth weight on anthropometric parameter, body fat and blood pressure Between LBW and NBW groups blood pressure in actually overweight and obese group**

	LBW (10)	NBW(40)
Age (years old)	25.66±2.08	24.4±3.29
weight (Kg)	78.53±9.15	72.85±8.33
height(cm)	135.33±48.01	155.78±36.97
BMI (Kg/m <sup>2</sup> )	27.86±0.68	27.08±2.094
Body fat (%)	31.12±12.18	29.94±6.55
Fat mass	31.52±8.61*	21.77±2.19
Lean mass	47.00±12.02	51.02±1.76
Waist (cm)	97.33±14.50	82.45±13.85
Hip (cm)	101.0±14.93	88±11.05
Waist to hip ratio	1.067±0.301	1.11±0.244
Diastolic blood pressure (mmHg)	72.00±1.73	72.75±8.50
Systolic blood pressure (mmHg)	120.66±10.50	121.47±16.05
Bicipital skin folk thickness (mm)	15.66±2.88	11.06±6.60
Tricipital skin folk thickness (mm)	24.0±6.92	15.74±6.28
Suscapulary skin folk thickness (mm)	26.66±6.11	15.46±5.09
Supra iliac skin folk thickness (mm)	22±2	14.94±5.97
Waist to hip ratio	1.067±0.301	1.11±0.244

\*p&lt;0.05 compare to NBW group

**3.3.4 Influence of birth weight on anthropometric parameter, body fat and blood pressure Between NBW and HBW groups blood pressure in actually overweight and obese group**

Hip was significantly (p &lt; 0.05) higher among HBW compared to those with NBW (Table 4).

**Table 4. Influence of birth weight on anthropometric parameter, body fat and blood pressure Between NBW and HBW groups blood pressure in actually overweight and obese group**

	NBW(40)	HBW(12)
Age (years old)	24.4±3.29	24.50±2.11
weight (Kg)	72.85±8.33	69.67±9.42
height(cm)	155.78±36.97	170.13±7.79
BMI (Kg/m <sup>2</sup> )	27.08±2.094	25.77±9.31
Body fat (%)	29.94±6.55	22.66±8.93
Fat mass	21.77±2.19	17.22±3.10
Lean mass	51.02±1.76	58.44±3.61
Waist (cm)	82.45±13.85	81.83±8.06
Hip (cm)	88±11.05	100.33±5.81*
Waist to hip ratio	1.11±0.244	1.23±0.076
Diastolic blood pressure (mmHg)	72.75±8.50	67.0±6.89
Systolic blood pressure (mmHg)	121.47±16.05	125.00±7.89
Bicipital skin folk thickness (mm)	11.06±6.60	11.1±4.49
Tricipital skin folk thickness (mm)	15.74±6.28	16.5±4.37
Suscapulary skin folk thickness (mm)	15.46±5.09	17.66±5.11
Supra iliac skin folk thickness (mm)	14.94±5.97	17.16±7.16
Waist to hip ratio	1.11±0.244	1.23±0.076

\* P &lt;0.05 compared to NBW.

### 3.4 Influence of Birth Weight on Energy Intake, Protein Intake, Carbohydrates Intake, Lipids Intake and Energy Expenditure

There were no significant difference between LBW and NBW (result not shown), only the daily protein intake was significantly high among NBW ( $P < 0.05$ ) compared to HBW (Table 5).

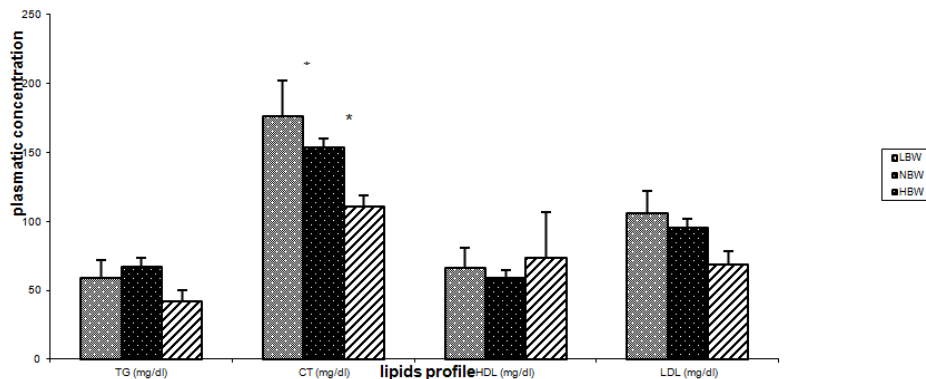
**Table 5. Influence of birth weight on energy intake, protein, carbohydrates, lipids and energy expenditure**

	NBW (n=182)	HBW (n=42)
Daily energy intake (Kcal)	2084.39 ±54.81	2021.16±89.55
Daily protein intake (g)	81.66 ±4.76 *	60.64 ±7.26
Daily carbohydrate intake (g)	261.60 ±10.00	282.10 ± 7.26
Daily fat intake (g) (g)	78.30 ±6.17	67.69 ±16.48
Daily energy expenditure (Kcal)	1438.56 ±29.39	1337.53 ±51.44

\*  $P < 0.05$  compared to overweight at birth.

### 3.5 Influence of Birth Weight on Lipid Profile

The total cholesterol level was significantly higher ( $p < 0.05$ ) among individuals with a low birth weight compared to normal and overweight at birth (Fig. 1).



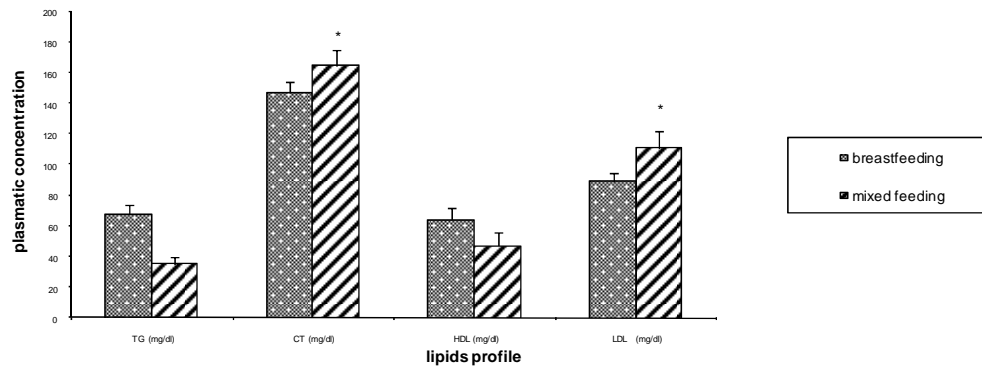
\*  $p < 0.05$  compare to HBW

Fig. 1 Influence of birth weight on lipids profile

### 3.6 Influence of Type of Feeding on the Lipid Profile

The plasma levels of total cholesterol and LDL cholesterol were significantly higher ( $p < 0.05$ ) among individuals who were mixed feeding compared with those exclusively breastfeeding (Fig. 2).



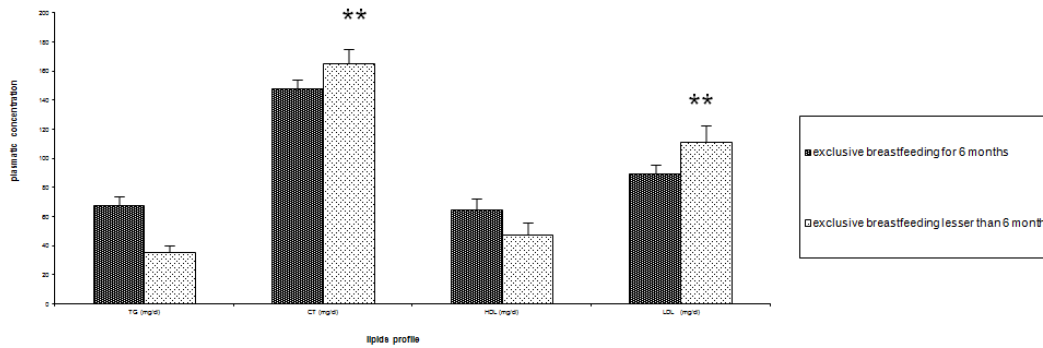


\*  $p < 0.05$  compare to exclusive breastfeeding group

**Fig. 2. Effect of feeding pattern**

### 3.7 Influence of the Duration of Exclusive Breastfeeding on the Lipid Profile

The plasma levels of total cholesterol and LDL cholesterol were significantly higher ( $p < 0.01$ ) among participants who were exclusively breastfed for less than six months compared to those exclusively breast fed for more than six months (Fig. 3).

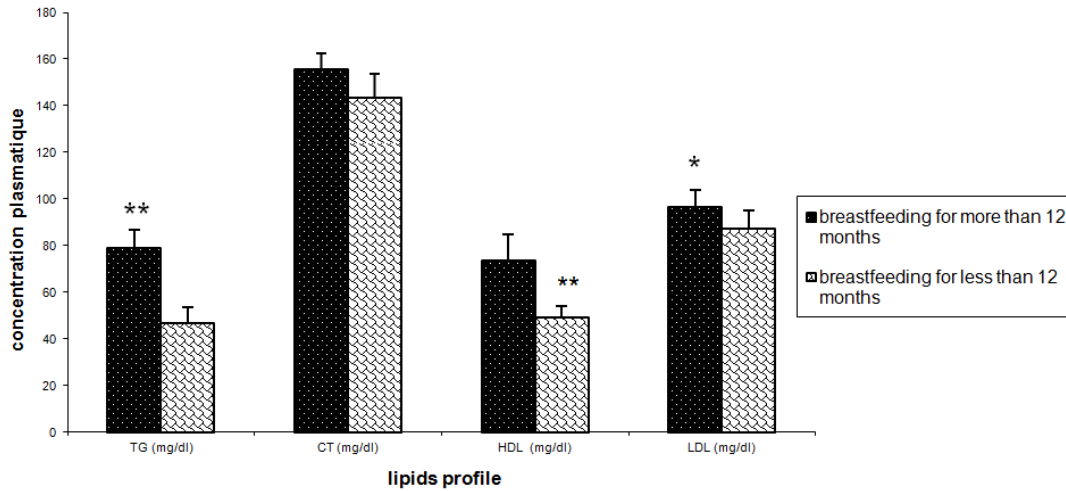


\*\* significant difference at  $p < 0.01$  compared with those having been breast fed exclusively for six months.

**Fig. 3. Influence of the duration of exclusive breastfeeding on the lipid profile**

### 3.8 Influence of Duration of Breast Feeding on the Lipid Profile

The plasma levels of triglycerides and LDL cholesterol were significantly higher ( $p < 0.01$ ) and ( $p < 0.05$ ) respectively among those breastfed for 12 months compared to those breastfed for more than 12 months (Fig. 4).



\*\* significant difference at  $p < 0.01$  compared with those having been breast fed for more than 12 months.  
 \* significant difference at  $p < 0.05$  compared with those having been breast fed for more than six months.

**Fig. 4. Influence of the duration of breastfeeding on the lipid profile**

### 3.9 Correlation between Lipids Profile, Birth Weight and Duration of Breastfeeding

The birth weight was significantly and negatively correlated to plasma levels of total cholesterol ( $p < 0.01$ ) and LDL ( $p < 0.05$ ). The duration of breastfeeding was inversely and significantly ( $p < 0.01$ ) correlated with plasma triglycerides (Table 6).

**Table 6. Relationship between lipid profile, weight at birth and duration of breastfeeding**

	TG	Total cholesterol	HDL cholesterol	LDL cholesterol	Birth weight
TG	1				
Total cholesterol	0.308**	1			
HDL cholesterol	0.279**	0.233**	1		
LDL cholesterol	0.028	0.808**	-0.229*	1	
Birth weight	-0.40	-0.254**	-0.236	-0.236*	1
Duration of breastfeeding	-0.241**	0.033	-0.164	0.091	

\*\* Correlation is significant at  $p < 0.01$

\* Correlation is significant at  $p < 0.05$

## 4. DISCUSSION

The objective of this study was to examine effect of birth weight and mode of feeding during early infancy on clinical indicators of obesity and lipid profile in adulthood. HBW was associated with increased risk of lean mass (OR=1.100; 95% CI 1.039-1.164), and decreased risk of body fat (OR=0.918; 95% CI 0.852-0.988) and high total cholesterol (OR =

0.982; 95% CI 0.962-0.994). A high birth weight, usually related to greater BMI later in life, has also been associated with less body fatness assessed by skinfold-thickness measurement [13].

The idea that factors act during early critical windows such as intrauterine or early postnatal life to influence or “program” long term health is now a major public health concern [14]. For instance, a high birth weight has been suggested to program an increased risk of later obesity, as measured by body mass index (BMI; in kg/m<sup>2</sup>) [3]. The association between birth weight and BMI, however, contradicts considerable evidence that a high birth weight programs less susceptibility rather than greater susceptibility to cardiovascular disease (CVD) risk factors [4]. One hypothesis that could partially explain this paradox is that low birth weight is associated with programming of greater abdominal or truncal fat mass [5], which would increase the metabolic risk of CVD.

According to actually overweight and obese participants, the fat mass was significantly ( $p < 0.05$ ) higher among low birth weight compared to those with normal weight at birth. Weight and lean mass was significantly higher ( $p < 0.05$ ) among overweight individuals at birth weight compared to normal weight at birth. Barker hypothesis could then partly explain observations of greater percentage body fat in adults of low birth weight. Our observations suggest that poor fetal growth, as measured by low birth weight, programs a higher proportion of fat mass later in life. Earlier studies have shown inconsistent associations between birth weight and later body composition. Furthermore, a study in twins discordant for birth weight showed interpair differences in height, but not BMI, which contradicts an influence of fetal growth on later body fatness [15]. In a good-quality environment, the mother is predicted to maximize her own reproductive success by investing in offspring lean mass. In a poor-quality environment, however, the long-term benefits of lean mass are offset by the more immediate benefits of fatness for survival in the weaning period [16]. Therefore, in poorer environments, with scarce resources for investment, lean mass deposition is reduced, and fat mass deposition is enhanced, which leads to the “fat-thin” phenotype of the growth-retarded infant [17]. Thus, our findings are likely to have several implications for the early origins of obesity.

Blood pressure was not significantly different probably due to the fact that our study population was young.

Only the daily protein intake was significantly higher among normal weight at birth ( $P < 0.05$ ) compared to overweight at birth. The total energy intake and energy expenditure was not different in birth weight group showing that results obtained are not due to food intake and energy expenditure.

The total plasma cholesterol level was significantly higher ( $p < 0.05$ ) in individuals having a low weight at birth compared to normal weight and overweight at birth. The LDL-cholesterol was higher among low birth weight with a non-significant difference. Similarly, the plasma level of total cholesterol and LDL cholesterol were inverse and significantly associated to birth weight. High cholesterol is a major risk factor for the occurrence of cardiovascular disease [18] and can be programmed from the first moments of life [19]. According to the Barker hypothesis, intrauterine growth retardation lead to changes in lipid metabolism and would be one explanation for the inverse association between birth weight and the occurrence of cardiovascular disease in adulthood [20]. These results are similar to those obtained by [21] among obese men.

The plasma levels of total cholesterol and LDL cholesterol were significantly higher ( $p < 0.05$ ) among individuals who were mixed breastfed compared to those exclusively breastfed. The concept of fetal programming is that, during critical periods of prenatal life, the expression of the genotype being impeded by changes in hormonal and nutritional environment of the fetus, leaving permanent sequelae in several structures and physiological functions [22]. Fetal growth adjust its metabolism to the intrauterine environment, but this adaptation has a price in a more positive future, that of an increased risk of chronic diseases [23]. Unlike other permanent effects of foetal malnutrition, such as neural tube defects resulting from a deficiency of folate, the foetal programming of chronic diseases such as metabolic syndrome or coronary artery disease is difficult to verify in humans, partly because of the time lag between the achievement and foetal effects and the many factors that may modulate this association throughout life. Work on animal models tend to support the hypothesis of fetal programming and identify plausible mechanisms. Dietary manipulations in rats have shown, for example, that under-nutrition in early gestation had little effect on birth weight, but that they easily become obese rats when exposed to a liberal ration, with exaggerated hyperinsulinemia, high blood pressure and hyperleptinemia [24].

Several mechanisms have been proposed to explain the foetal origins of chronic disease. It has been suggested that programming of the cardiovascular system induced by maternal malnutrition is a phenomenon dependent on steroids. The chronic increase in blood pressure is accompanied, for example, by a decrease of enzyme dehydrogenation of steroids (11-beta-OHSD) and thus expression [25]. Excessive exposure of the foetus to glucocorticoids due to hyperactivity of the hypophyso- hypothalamus-adrenal axis response to stress of malnutrition might be involved in the programming of chronic diseases [26]. Overexposure can also result to a deficiency of the enzyme 11-beta-OHSD which is normally used to protect the foetus of maternal glucocorticoids by inactivation. A high level of glucocorticoids provides short-term benefits to the foetus, since it promotes the availability of glucose and other energy substrates but the long term effects on the cardiovascular system may be deleterious, for example altered sensitivity of the axis hypophyso- hypothalamus-adrenal axis to feedback hormones [27].

The deficit in foetal growth can increase the risk in different ways either by increasing the tendency to obesity, by increasing the risk normally associated with obesity, or through the remedial potential of growth after birth. It was noted that individuals with low birth weight have a propensity to obesity and adiposity [28].

The plasma levels of total cholesterol and LDL cholesterol were significantly higher ( $p < 0.05$ ) among individuals who were mixed breastfed compared with those who receive exclusive breastfeeding. The plasma levels of total cholesterol and LDL cholesterol were significantly higher ( $p < 0.01$ ) among those exclusively breastfed for less than six months compared to those exclusively breast fed for more than six months. The plasma levels of triglycerides and LDL cholesterol were significantly higher ( $p < 0.01$ ) and ( $p < 0.05$ ) respectively among those breastfed for up to 12 months compared to those in breast-feeding for more than 12 months.

The programming of adiposity by breastfeeding is because of a number of bioactive nutrients in human milk that are absent from some formulas (e.g. long-chain polyunsaturated fatty acids [29]. Differences in early protein intake (up to 70% greater in formula-fed than breastfed infants [30] could also affect later adiposity [31]. A higher protein intake in infancy has been suggested to promote later obesity by stimulation of insulin release and programming of higher long-term insulin concentrations [32]. Finally and most recently, we have suggested that the benefits of breastfeeding for long-term obesity may be due to a

slower pattern of growth in breastfed compared with formula-fed infants, the growth acceleration hypothesis. This proposes that faster post-natal growth programmes several components of the metabolic syndrome, including insulin resistance, higher low-density lipoprotein cholesterol concentration, higher blood pressure and obesity. Similarly, for cholesterol concentration, slower neonatal weight gain was associated with 20% lower cholesterol concentration compared with 10% lowering of cholesterol concentration associated with breastfeeding rather than formula feeding [5].

## **5. CONCLUSION**

Low weight at birth promotes high body fat and high total plasma cholesterol in adulthood. High weight at birth is associated with a higher lean mass, lower fat mass and total plasma cholesterol in adulthood.

Breastfeeding protects against the development of obesity and bad lipids profile in adulthood.

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## **COMPETING INTEREST**

All authors declared that there are no financial and personal relationships with other people or organizations that could inappropriately influence (bias) their work.

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