were enrolled at the outpatient pediatric clinic of our Institution from January 2008 to December 2009. All subjects had physical examination with measurement of body weight, waist and hip circumference, and blood pressure. Serum levels of glucose, insulin, HDL-cholesterol, tryglicerides and C-reactive protein were assessed. MetS was diagnosed according to modified ATP III criteria.

Results: MetS was demonstrated in 19.3% of cases. 76.9% of them presented BMI >95th percentile, 64.1% HDL-cholesterol < 5th percentile, 76.9% tryglicerides >95th percentile, and 51.3% systolic blood pressure >95th percentile. MetS prevalence was greater, although not statistically significant, in males than in females (24.5% vs 15%, respectively). Waist/Hip ratio and basal glicemia were significantly higher in males ($p=0.02$ and $p=0.05$, respectively). HDL-cholesterol levels were significantly lower in females than in males ($p=0.04$). Children with MetS showed significantly higher insulin levels, but HOMA index values were not significantly greater than in subjects without MetS. Linear regression analysis demonstrated that predisposing factors for MetS were HDL-cholesterol values < 5th percentile ($p=0.001$), tryglicerides values >95th percentile ($p=0.03$) and systolic blood pressure values >95th percentile ($p=0.045$).

Conclusions: Our data confirm that a correct diet, through tryglicerides decrease, and physical activity, through HDL-cholesterol increase, are the basis for the treatment of pediatric obesity.

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NEWBORN BODY COMPOSITION ON DAY TWO OF LIFE MEASURED USING AIR-DISPLACEMENT BODY PLETHYSMOGRAPHY

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Background and aims: In addition to the immediate neonatal risks, fetal growth restriction has been increasingly recognised as a risk for long term metabolic disease. Most studies have relied on standardised birth weight centiles to categorise fetal growth. We wished to examine the true rate of growth abnormal fetal growth and compare this to the use of indirect methods.

Methods: Infant were recruited antenatally as part of a prospective birth cohort study, the BASELINE study. Body fat and fat free mass were measured on day 2 of life in infants using air displacement body plethysmography . Gender, gestation, birth weight(BW), weight, length, mid arm circumference (MAC), Ponderal Index (PI) were recorded .

Results: To date 200 infants have been recruited, with mean (SD) gestational age of 39.7 (1.2)weeks and mean (SD) BW = 3.520 (0.472) kg. The mean (SD) fat mass was 0.389 kg (0.272), giving a mean (SD) percentage body fat of 11(4)%. In the 22 infants with a body fat mass < 10th centile birth weight was 3.12(2.7-3.88)kg. Only one baby had a BW < 10th centile using standard centiles. MAC correlated with total body mass ($R=0.705$, $p<0.001$) and showed a negative correlation with lean body mass $R=-0.263$, $p<0.001$).

Conclusion: We have provided normal ranges for infant fat mass on day 2 of life. Standard birth centiles do not accurately detect low body fat or fetal growth restriction.

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CORTISOL CONCENTRATIONS IN PREGNANCY AND ARTERIAL ELASTICITY IN CHILDHOOD

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Prenatal overexposure to glucocorticoids may programme a range of tissue-specific effects in the foetus. The objective of this study was to assess the relationship between cortisol concentrations in the last trimester of pregnancy and arterial elasticity in childhood. The study is part of a cohort involving 130 Brazilian pregnant women and their children, ages 5 to 7 years. Maternal cortisol was determined in saliva by an enzyme immunoassay utilizing the mean concentration of 9 samples of saliva (3 in each different day), collected at the same time, early in the morning. The large arterial elasticity index (LAEI) and the small arterial elasticity index (SAEI) were assessed by the HDI/PulseWave CR-2000 Cardiovascular Profiling System®. Socioeconomic and demographic characteristics and life style factors were determined by a questionnaire. The nutritional status of the women and children was assessed by

the body mass index-BMI. The association between maternal cortisol and LAEI and SAEI in childhood was calculated by multivariate linear regression analysis. There was a statistically significant association between maternal cortisol in pregnancy and LAEI in childhood, controlling for birth weight, age, BMI and gender of the children. The data suggest that exposure to excess glucocorticoid in the prenatal period is associated to a reduction of the arterial elasticity in childhood, predisposing to cardiovascular diseases in later life.

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ABNORMAL LIPID COMPOSITION OF HDL SUBCLASSES IN CHILDREN WITH METABOLIC SYNDROME IS INDEPENDENT OF SRBI (+1050T/C) AND ADIPONECTIN (+45T/G, +246G/T) POLYMORPHISMS.

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Background: SRBI and adiponectin regulate HDL metabolism but little is known about their role on the abnormal HDL size distribution in children with metabolic syndrome (MS).

Aims: to determine lipids of HDL subclasses and establish whether SRBI (+1050T/C) and adiponectin (+45T/G, +246G/T) polymorphisms contribute to HDL lipidic composition.

Methods: Fifty MS children (IDF criteria), mean age=11.3±3.2y and 50 controls matched by age and gender were included. New enzymatic methods were developed to stain the lipids of HDL subclasses on gel surface.

Results:

	MS n= 50 (F: 25, M:25)			CONTROL n= 50 (F:24, M:26)		
HDL Sub-classes	TC (mg/dL)	Tg (mg/dL)	Ph (mg/dL)	CT (mg/dL)	Tg (mg/dL)	Ph (mg/dL)
HDL 2b	9.6 ± 3.5*	5.7 [4.6-8.5]	27.4 ± 14.32*	13.0 ± 4.3	7.2 [4.39-10.1]	35.3 ± 18.2
HDL 2a	4.3 [3.8-5.6]*	3.3 [2.1-4.2]	13.2 ± 5.4	5.3 [4.8-6.9]	3.6 [2.0-4.7]	15.4 ± 6.3
HDL 3a	10.2 ± 2.5*	7.5 [5.0-9.6]	30.5 ± 11.2	11.9 ± 3.2	6.6 [2.0-4.7]	30.4 ± 11.6
HDL 3b	6.0 ± 1.5*	4.9 [3.0-6.3]	19.0 [14.6-24.8]	7.0 ± 2.2	3.9 [2.5-6.0]	17.3 [14.0-20.7]
HDL 3c	6.6 ± 2.4	5.5 [3.9-6.3]*	24.2 ± 11.1*	7.3 ± 2.8	4.1 [2.5-6.0]	19.1 ± 9.9
MS: Metabolic Syndrome, F: Female, M: Male, TC: Total Cholesterol, Tg: Triglycerides, Ph: Phospholipids, *p: < 0.05						

[Plasma Lipid Concentration of HDL Subclasses]

The HDL subclasses cholesterol-to-phospholipid ratio was lower in patients, (p< 0.001). +1050T carriers of the SR-BI gene polymorphism were more frequent in MS group (64% vs 33%, p=0.004). However, SR-BI polymorphisms did not correlate with HDL lipids. Adiponectin gene polymorphisms were similar between MS and control subjects.

Conclusion: MS in children is associated with low plasma concentration of HDL subclasses cholesterol, independently of SRBI and adiponectin polymorphisms. Our data strongly suggest an abnormal HDL structure in MS that may be related with and impaired function of these lipoproteins.