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.