

## 1. CALORIC PROTEIC MALNUTRITION TODAY

*M. Moya.*

The term ‘Caloric proteic malnutrition’ (Jelliffe, 1920) has been gradually substituted by ‘Subnutrition’ because it better reflects the minor nutrient and energy intake to live and grow. The incidence of subnutrition according to WHO has decreased worldwide since 1960, when it was estimated that 300 million children were affected compared to 146 million in 2000. The prevalence of pediatric subnutrition in developed countries is clearly less than in elderly people. Evaluation of subnutrition is complex. The most usual procedure is to consider it when weight is under the third percentile for its age and gender. That would indicate a chronic or recent subnutrition. Height under the P3 indicates a chronic evolution of subnutrition (stunting) and in our environment it is necessary to know target height for its correct interpretation. Weight decreased in relation to height ( $\text{kg/m}^2$  o BMIr) should clearly indicate an acute subnutrition. The availability of computer programming support has allowed a more precise evaluation of the somatometric data and so it is named subnutrition when BMIr is lower than 85% or BMI ZS is bigger than -2 SD, and it is called ‘Underweight’ if the figures are between 90 to 85% or -1 a -2 SD respectively. The aim of this study is to analyze subnutrition in our environment (developed countries) where the epidemiologic criteria ‘under five year of age’ is less important.

During the last 2 years and over a total of 500 patients seen in a general out patient clinic of this Service, ten cases of subnutrition were detected (IMC ZS > -2.0 SD) and 31 of underweight (IMC ZS -1.0 – 1.9). All of them were between 2-18 year of age. The most representative somatometric data are (table):

### SUBNUTRITON/UNDERWEIGHT. CHARACTERISTICS

	SUBNUTRITION (ZS > -2 SD)		UNDERWEIGHT (ZS -1-2 SD)
n	10	p =	31
Birth Weight (g)	2941 ± 637	0.07	3217 ± 522
Visit Age (a)	10.2 ± 5.6	0.16	8.7 ± 3.7
Visit Height (ZS)	-1.98 ± 0.43	0.001	-0.79 ± 1.1
Target Height (cm)	166.6 ± 7.1	0.3	165.4 ± 8.4
Weight ZS	-2.38 ± 0.27	0.001	-1.35 ± 0.41
BMI ZS	-1.87 ± 0.36	0.003	-1.28 ± 0.48

In this sample we excluded preterm babies, restricted intrauterine growth and those suffering from organic diseases which could affect nutrition.

Two things are worth mentioning after the study of the sample; first the most efficient method to make the diagnosis of subnutrition (or underweight) has been the calculation of ZS of ideal weight for age and gender. Second the most important cause of underweight has been the exogenic component (28/31). Undernutrition in the two first years of life is related to nutritional aspects pre and early postnatals.

## 2. NUTRITIONAL RICKETS IN CHILDHOOD.

*D. Yeste y A. Carrascosa. Servicio de Endocrinología Pediátrica. Hospital Vall d'Hebron. Barcelona. Universidad Autónoma de Barcelona.*

Rickets is a skeletal disease in the growing child caused by defective mineralization of bone tissue with excess of bone matrix or non-mineralized osteoid tissue due primarily to vitamin D deficiency. Vitamin D is produced mainly in the skin following exposure to ultraviolet rays, although a small proportion derives from the diet. Levels vary according to diet and climate characteristics. Vitamin D is an essential prohormone for normal intestinal calcium absorption and promotes bone tissue mineralization.

Nutritional rickets constituted one of most common diseases in childhood until the beginning of the 20<sup>th</sup> century. Once vitamin D had been identified and simple methods of nutritional supplementation had been developed, nutritional rickets practically disappear in industrialized countries. However, recent decades have seen a re-emergence of the disease in infancy. In a recent multicenter study, we analyzed the clinical and epidemiologic features of 64 infants and children diagnosed of nutritional rickets at the Pediatric Departments of Catalonia (Spain). Mean age (SDS) at diagnosis was  $9.9 \pm 7$  months (range: 3-36); 35.5% were under 6 months of age, 38.7% between 6 months and 1 year and the remaining 25.8% were children over the age of one. Race distribution: black: 61%, dark-skinned: 36%; Caucasian: 3%. Country of origin: Sub-Saharan Africa 59.7%, Morocco 33.9%, Catalonia 3.2%, Pakistan 1.6%. At diagnosis, 72% were following an exclusively milk diet (48% mother's milk alone) not supplemented with vitamin D. The most frequent form of clinical presentation in infants under 6 months of age was: hypocalcemic tetany/seizures; in children from 6-12 months: failure to thrive; and in children over the age of one: skeletal deformities. Weight and height expressed as z-score value at diagnosis were -0.67 and -0.91, respectively.

In summary, nutritional rickets is an emerging disease in Catalonia, affecting mainly black or dark-skinned immigrant infants and children, fed with maternal milk alone, without vitamin D supplementation and with little exposure to the sun. Systematic, preventive supplementation with vitamin D is essential in these populations.

### 3. INTEGRATED CLASIFICACION OF METABOLIC SYNDROME

*Leis R<sup>1</sup>, Gil-Campos M<sup>2</sup>, Olza J<sup>3</sup>, Bueno G<sup>4</sup>, Aguilera CM<sup>3</sup>, Valle M<sup>5</sup>, Cañete R<sup>2</sup>, Tojo R<sup>1</sup>, Moreno LA<sup>4</sup>, Gil A<sup>3</sup>.*

<sup>1</sup> Unit of Investigation in Nutrition, Growth and Human Development of Galicia, Department of Pediatrics, Clinic University Hospital of Santiago, University of Santiago de Compostela, Spain (R.L. R.T.); <sup>2</sup> Unit of Pediatric Endocrinology, Reina Sofia University Hospital, Córdoba, Spain (M.G-C., R.C.); <sup>3</sup> Department of Biochemistry and Molecular Biology II, Institute of Nutrition and Food Technology, Center for Biomedical Research, University of Granada, Granada, Spain (J.O., C.M.A., A.G.); <sup>4</sup> Pediatric Department, Lozano Blesa University Clinical Hospital, University of Zaragoza, Spain (G.B.); School of Health Science, University of Zaragoza, Spain (L.M.); <sup>5</sup> Unit of clinical analyses, Valle de los Pedroches Hospital, Cordoba, Spain (M.V.).

Obesity in childhood is increasing Worldwide being associated to a high prevalence of cardiovascular and metabolic disease, such as type 2 diabetes, dyslipidemia, hipertensión, atherosclerosis and non-alcoholic fatty liver disease. These comorbidities are rising and are explained by the earlier onset and duration of obesity.

Cardiovascular disease risk factors namely hypertension, hypertriglyceridemia, low HDL cholesterol and impaired glucose metabolism tend to cluster in obese children. The association of these factors is often recognized as metabolic syndrome (MS). Despite the difficulty inherent in defining the key elements of MS, a condition modulated by so many genetic and environmental factors, strong evidence supports obesity as the main correlate of cardiometabolic risk, especially when adiposity is centrally distributed.

The prevalence of MS in children varies widely depending on obesity definition and MS features analyzed. So, in pediatric literature, MS definitions have been adapted for children and adolescents by different authors, who used diverse criteria. Table I. Although one single definition, albeit with gender and ethnicity specific cut off points, is suitable for use in the at-risk adult population, transposing it to children and adolescents is troublesome. Recently, the International Diabetes Federation (IDF) has published a consensus definition for the MS in children and adolescents, inspired in part by the IDF worldwide definition of MS in adults, trying to unify the existing multiple definitions to have a single diagnostic tool to use in clinical practice. IDF has suggested that MS should not be diagnosed in children younger than 10 years, excepting those with family history of MS and their associated comorbidities. This definition has to be evaluated scientifically and IDF recognizes that the definition criteria should be reassessed in the future based on new evidence and emerging data. Furthermore, little is known concerning the factors influencing the frequency of the MS in children and adolescents and particularly those related to pubertal status.

The aims of our study is to compare the prevalence of MS according to different definitions in children and adolescents and analyze the impact of each component of MS in relation to age, sex and pubertal status, so to study new possible factors associated with the MS in children to propose an integrated MS definition.

Table I.

METABOLIC SYNDROME DEFINITION						
	Age	Waist circumference	Triglycerides (mg/dl)	HDL (mg/dl)	Baseline glycemia (mg/dl)	Systolic or Diastolic Blood Pressure (mm Hg)
Cook <sup>1</sup> MS $\geq$ 3 criteria		$\geq$ p 90*	$\geq$ 110	< 40	$\geq$ 110	$\geq$ p 90
De Ferranti <sup>2</sup> MS $\geq$ 3 criteria		$\geq$ p 75*	$\geq$ 100	<45: 15-19y <50: < 15y	$\geq$ 110	$\geq$ p 90
Weiss <sup>3</sup> MS $\geq$ 3 criteria		BMI>p97*	$\geq$ p 95	< p 5	140-199mg/dl (After 2 hours of oral glucose tolerance test )	$\geq$ p 95
Internacional Diabetes Federation (IDF) <sup>4</sup>	6-10y	$\geq$ p 90*	Metabolic syndrome cannot be diagnosed, but further measurements should be made if family history of metabolic syndrome, type 2 diabetes mellitas, dyslipidaemia, cardiovascular disease, hipertensi3n, or obesity.			
	10-16y	$\geq$ p 90	$\geq$ 150	<40	$\geq$ 100	$\geq$ 130 SBP $\geq$ 85 DBP
MS = Central Obesity + 2 or more criteria	> 16 y	$\geq$ 94cm Male $\geq$ 80cm Female	$\geq$ 150	<40 Male <50 Female	$\geq$ 100	$\geq$ 130 SBP $\geq$ 85 DBP

References:

- 1) Cook S, Auinger P, Li C Ford ES. Metabolic Syndrome Rates in United States Adolescents, from the National Health and Nutrition Examination Survey, 1999-2002. J Pediatric 2008;152:165-70.
- 2) De Ferranti S, Gauvreau K, Ludwig DS, Neufeld EJ, Newburger JW and Rifai N. Prevalence of the Metabolic Syndrome in American Adolescents: Findings from the Third National Health and Nutrition Examination Surevey. Circulation 2004;110:2494-2497.
- 3) International Diabetes Federation 2007. The IDF consensus definition of the Metabolic Syndrome in children and adolescents. International Diabetes Federation 2007. ISBN 2-930229-49-7.

- 4) Tojo R, Leis R y grupo de trabajo Estrategia NAOS. 2007. La obesidad en la infancia y adolescencia. En: Nutrición, actividad física y prevención de la obesidad. Estrategia NAOS. Moreno, B. y Charro, A. (Coordinadores). 69-112. Ed Médica Panamericana. Ministerio de Sanidad y Consumo. Madrid
- 5) Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW et al. Obesity and the Metabolic Syndrome in children and adolescents. N Engl J Med 2004;350-2362-74.

#### 4. THICKENING INFANT FORMULA, RHEOLOGICAL STUDY OF THE IN VITRO PROPERTIES

*Infante Pina, Dámaso. Unidad de Gastroenterología, Hepatología y Nutrición. Hospital Materno Infantil Vall d'Hebron. Barcelona; Lara-Villoslada, Federico. Departamento de Nutrición y Seguridad Alimentaria. Puleva Food. Granada*

Thickened infant formula, specially formulated to increase the viscosity, are commonly used in the treatment of regurgitation, a clinical symptom of gastroesophageal reflux. From this date up to nowadays, the use of thickening agents, such as starch, guar gum or carob bean gum has widely spread in the production of the so called antireflux or antiregurgitation infant formula. The objective of the present work was to analyze viscosity and the rheological behaviour of different thickened formula from the Spanish market compared to an standard formula with or without the addition of 10g/100mL of gluten-free cereals. Viscosity of the samples was evaluated in a Bohlim CS-10 controlled-stress rheometer and was performed at basal conditions (25°C and pH 7) and at simulated gastrointestinal conditions (37°C, pH 4 and 10g/100mL of pepsin) at time 0 and after 60 minutes of incubation. Results showed that at basal conditions, those formula containing bean gum (2.9g/100mL) and bean flour (2.9g/100mL) showed the highest viscosity, which was similar to that obtained for the cereal formula. In contrast, the addition of bean flour at the concentration 3.5 g/100mL or corn starch at 2.2 g/100mL do not appear to be effective in the basal increment of viscosity. At gastrointestinal simulated conditions, all the formula experienced an increase in viscosity although the behavior after 60 minutes of incubation was heterogeneous, since formula with the same thickener showed different behaviors. As a conclusion, thickening agents are effective in the increment of viscosity but the rheological behavior depends on the type and concentration of thickening agent. It remains to be elucidated the ideal viscosity to be reached and the role of other components of the infant formula in the viscosity and rheological behavior.

#### 5. NUTRITION AND ADHD.

*Durá T, Yoldi ME, Gallinas F, Molins T, Aguilera S. Hospital Materno-infantil Virgen del Camino. Pamplona*

**OBJECTIVE:** Evolution study of nutritional status of a group of patients diagnosed with attention deficit and hyperactivity disorder (ADHD) being treated with methylphenidate.

**MATERIAL AND METHODS:** Clinical records from 187 patients diagnosed with ADHD being treated with extended-release methylphenidate have been reviewed. The sample consisted of 129 males (69%) and 58 females (31.0%). Weight, height and percent body mass index (%BMI) as well as methylphenidate doses after 6, 12, 18, 24 and 30 months of evolution were recorded.

**RESULTS:** Mean age at diagnosis was 8.3 years (4.8-14.3).

Evolution (months)	Methylphenidate (mg/kg/d)	Weight score	(Z)Height score	(Z)%BMI score	(Z)Malnutrition (%)
0	---	-0,144	-0,111	-0,143	30,7
6	0,79	-0,242	+0,060	-0,341*	33,7
12	0,80	-0,448*	-0,241	-0,438*	43,9
18	0,88	-0,496*	-0,199	-0,529*	48,4
24	0,92*	-0,342	-0,299*	-0,271	36,8
30	0,95*	-0,313	-0,379*	-0,209	43,1

(\*) P<0,005 with regard to the time at diagnosis.

#### CONCLUSIONS.

- By the time of diagnosis of ADHD, 31% of patients (one out of three) are in a malnutrition status that tends toward deterioration after treatment (methylphenidate).
- Treatment with methylphenidate has a negative influence over height that could be reduced by improving the nutritional status of the patients.
- It is possible that symptoms could get slightly better after improving the nutritional status of the patients with ADHD (nutritional intervention).

## 6. CURVES OF GROWTH: REFERENCES AND STANDARDS.

*Cidrás M<sup>1</sup>, Saavedra P<sup>2</sup>.*

<sup>1</sup> *Sección de Neonatología del Hospital Universitario Virgen de la Arrixaca, Murcia*

<sup>2</sup> *Departamento de Matemáticas. Universidad de Las Palmas de Gran Canaria*

Growth curves are a reference that allows comparing an individual with a population. The concept of "reference" is done with descriptive sense, as opposed to the concept of "standard" that establishes an ideal of optimum growth. Weight is considered an anthropometric measurement with "natural" skewness. Current postnatal weight curves show greater skewness towards higher percentiles than classic curves, with possible subestimation of the real obesity.

The purpose of this paper was to question the skewness of weight and, consequently, to draw standard weight charts with symmetrical curves. A preliminary study was done with fetal growth curves.

Methods. From the weight of 10697 newborns we estimate through the method of Cole and Green (Cole TJ, Green PJ. Smoothing reference centile curves: the LMS method and penalized likelihood. *Stat Med* 1992;11(10):1305-19) the curves L, M and S from which the a-percentile curves are drawn:  $C_a = M(1 + z_a \cdot S \cdot L)^{1/L}$ . These curves estimate the real curves of weights of the newborns. The problem of deciding which would be the ideal pattern is considered now. We set off of the idea that what is atypical breaks the normality of the distribution or to be more precise, the symmetry of the distribution. Fortunately, the median is resistant to the outliers, therefore we can admit that this is not affected markedly by the extreme observations. In the case of the weight of the newborns it does not seem reasonable to think that extreme observations are produced in the higher percentiles, however, if it is expectable in the lower percentiles due to intrauterine growth retardation. Under these suppositions we consider the following method of obtaining of ideal curves of growth:

1. In an ideal scenario, the data do not present skewness. That means admitting that the curve of powers of ideal Box-Cox is constantly equal to the unity; this is:  $L=1$ .
2. The ideal median is not affected by the extreme observations what means that it coincides with the real one.
3. A higher percentile, as the  $C_{0.90}$ , is not affected either by the extreme cases.
4. The previous assumptions determine the ideal curve of coefficients of variation in the form:  $S = (C_{0.90} - M) / (z_{0.90} \cdot M)$ .

Finally we define the curves of ideal percentiles:  $C_a = M(1 + z_a \cdot S)$ . It is noticed that eliminating the curve L means that the ideal curves correspond with the hypothesis of normality.

Our theoretical results coincide with some ones obtained in real observations (1.- Burkhardt T et al. Newborn weight charts underestimate the incidence of low birthweight in preterm infants. *Am J Obstet Gynecol* 2008;199:139.e1-139.e6. 2.- Ferdynus et al. Can birth weight standards based on healthy populations improve the identification of small-for-gestational-age newborns at risk of adverse neonatal outcomes? *Pediatrics* 2009; 123: 723-730).

In conclusion, with the same approach, using only the median and a percentile of ideal reference (in this case the  $C_{0.10}$ ), postnatal standard weight curves would be drawn.

## 7. FOCUS ON FAMILY OBESITY MATTERS

*M. Carmen Ochoa, Amelia Martí, J. Alfredo Martínez*

*Departamento de Ciencias de la Alimentación, Fisiología y Toxicología, Universidad de Navarra (Pamplona).*

Childhood obesity is a multifactorial disease, involving both genetic and environmental causes as well as lifestyle factors, in which family members substantially share the genome and the environment

Research conducted on twins, siblings and families have shown that obesity and overweight risk increases when parents are obese. Moreover, the trend of childhood obesity to obesity in adulthood is also greater in subjects who have one parent (or both) obese. The influence of parental obesity in childhood obesity is especially strong between 2 and 10 years. Also studies with adopted children indicate that there is a greater correlation between the degree of obesity of children with biological parents than adoptive parents, which confirms the role of genetics in the risk of obesity.

The influence of heredity on obesity has been studied from three different viewpoints. On the one hand, complex congenital syndromes in which obesity is only part of the clinical anomalies and manifestations. On the other hand, there are monogenic causes of obesity, in which a single gene defect (LEP, LEPR, POMC, MC4R, etc...) produces dysfunctions in critical regulatory elements concerning weight control. Finally, polygenic obesity, in which the

genetic determinants of variation among individuals in the body fat are probably multiple and interact among them. So far there are more than 120 candidate genes associated with obesity phenotypes.

The family is also a major agent of the environmental causes of obesity, by establishing energy intake and physical activity patterns or lack thereof (sedentary). The fetal nutrition and post natal maternal feeding as well as the epigenetic programming indicate that the mother's nutrition during pregnancy can significantly influence disease in the adult period, and even that this genetic perinatal coding may be transferred between generations.

Families of obese children have a decisive bearing on their dietary habits and physical exercise. Therefore, it is very important to involve parents in the treatment of childhood obesity. In those studies that implicate families in the weight loss intervention, long-term outcomes are much more satisfactory. Moreover, the weight maintenance is better in the case of parents with overweight that begin a program to lose weight with their children. Therefore, it is sometimes necessary to raise awareness among parents of the problem of obesity in their children before starting the intervention. Family involvement is less important as the child grows, so that in the case of adolescents it is important to maintain sessions with the patient separately from their parents. Moreover, the psychological support of children during treatment is important for success. Therefore, obese children should not be isolated and should participate in family meals and eating in a manner similar to other family members.

Camió J, Milagro FI, Martínez JA. Individuality and epigenetics in obesity. *Obes Rev.* 2009; (en prensa)

Marti A, Moreno-Aliaga MJ, Hebebrand J, Martinez JA. Genes, lifestyles and obesity. *Int J Obes Relat Metab Disord.* 2004; 3:S29-36.

Moreno-Aliaga MJ, Santos JL, Marti A, Martinez JA: Does weight loss prognosis depend on genetic make-up?. *Obes Rev.* 2005; 6:155-68.

Moreno LA, Ochoa MC, Wärnberg J, Marti A, Martínez JA, Marcos A. Treatment of obesity in children and adolescents. How nutrition can work?. *Int J Pediatr Obes.* 2008;3 Suppl 1:72-7.

Ochoa MC, Marti A, Martinez JA. Estudios de obesidad en genes candidatos. *Med Clin (Barc).* 2004;122:542-51.

Ochoa MC, Marti A, Azcona C, Chueca M, Oyarzábal M, Pelach R, Patiño A, Moreno-Aliaga MJ, Martínez-González MA, Martínez JA; Gene-gene interaction between PPAR gamma 2 and ADR beta 3 increases obesity risk in children and adolescents. *Int J Obes Relat Metab Disord.* 2004 ;28 Suppl 3:S37-41.

Ochoa MC, Moreno-Aliaga MJ, Martínez-González MA, Martínez JA, Marti A; TV watching modifies obesity risk linked to the 27Glu polymorphism of the ADRB2 gene in girls. *Int J Pediatr Obes.* 2006;1(2):83-8.

Ochoa MC, Santos JL, Azcona C, Moreno-Aliaga MJ, Martínez-González MA, Martínez JA, Marti A; GENOI Members. Association between obesity and insulin resistance with UCP2-UCP3 gene variants in Spanish children and adolescents. *Mol Genet Metab.* 2007 (4):351-8..

Ochoa MC, Azcona C, Biebermann H, Brumm H, Razquin C, Wermter AK, Martínez JA, Hebebrand J, Hinney A, Moreno-Aliaga MJ, Marti A, Patiño A, Chueca M, Oyarzabal M, Pelach R; A novel mutation Thr162Arg of the melanocortin 4 receptor gene in a Spanish children and adolescent population. *Clin Endocrinol (Oxf).* 2007 66.:652-8.

Ochoa MC, Moreno-Aliaga MJ, Martínez-González MA, Martínez JA, Marti A. Predictor factors for childhood obesity in a Spanish case-control study. *Nutrition.* 2007 May;23(5):379-84.

Steffen LM, Dai S, Fulton JE, Labarthe DR. Overweight in children and adolescents associated with TV viewing and parental weight: Project HearBeat! *Am J Prev Med.* 2009;37:S50-5.

Yang W, Kelly T, He J. Genetic epidemiology of obesity. *Epidemiol Rev.* 2007;29:49-61.

## **8. OBESE CHILD AND ADOLESCENTS EMOTIONAL DISORDERS.**

*Gonzalo Morandé*

*Child Jesus Hospital. Madrid*

**IX SEINAP Conference 2009-08-27**

**Obesity Symposium**

**Communication summary**

Anxiety, specially the one linked to negative emotional conditions, is present with overweight and obesity **pathogenesis**.(ov/ob).

**A.-As a predisposing condition:** in those children who were born or became anxious to eat or look insatiable. It's no wonder that someone in the family has a similar profile.

**B.-As a precipitating condition:** or in response to a psychotraumatic environmental situation. The girl begins to gain weight and start an obesogenic condition that goes unnoticed after parents' separation and leave home dad.

**C.-As a maintenance condition:** Maintenance conditions are the most important ones and the first to be confronted in clinical. In ov/ow occur: negative reinforcement, defence mechanism they use to not collapse, the development of pathologic habits, family incompetence and self perception of the problem.

**C.1.- Negative reinforcement:** ov/ow boys or girls are not well regarded. Children even siblings tease them or just don't bear them in mind on their games. Neither do adults, teachers, paediatricians and even parents, they also do not appreciate them.

They can be victims of attacks and can't respond or victimize themselves. A bad answer since it is difficult to solve. Treatment should ignore this response and seek resources to solve complicated situations, asking for help among others.

**2.-Defence mechanism:** They normally use resources they have to not get depressed and so can save some aspects of their self-esteem. Thereby they offset a bad image of themselves in body shape, with and maintaining or improved self concept on familiar, social, emotional and even school plane. Every compensation has its cost, more eating and obesogenic behaviour. It's not surprising that some of them tolerate social reproval, achieve acceptance for his good humour, friendliness and apparent happiness.

**3.- Bad habits perpetuate the problem:** Overweight can be understood as habits pathology. Together with the eating disorder, sleep habits, physical activity, study, evacuation, and social life are altered. Every treatment includes the establishment of healthy living habits.

**4.-Family incompetence:** There is almost always some family complicity, for suffering identical disorder, negligence and also incompetence. (medical incompetence?). There are mothers and also fathers who see their daughter's ov/ob as a personal failure even those who bring them visit. Every treatment begins with the parents.

**5.-Body dissatisfaction a maintenance factor and a risk factor of first order.** The child does not feel comfortable with his/her appearance or physical efficiency. Feeling ill at ease with himself does not facilitate behaviour change, but quite the opposite.

As a risk factor is non-specific, since not only predisposes to Anorexia and Bulimia but also Depressive Disorders, Substance Abuse and other adolescents behaviours including sexual promiscuity and pregnancy. All maintenance factors are risk factors.

**D.-There are differences in the emotional response to obesity among boys and girls:** Girls have a higher body dissatisfaction, which is accentuated when they are depressed, they have more drive for thinness. Since they are nine they compared with other girls, some of them in a compulsive way and with the menarche they become emotionally vulnerable. It increases the likelihood of suffering an Eating Disorder.

**E.-A high percentage of obese children and teenagers have school problems.** There are hypotheses linked to ADD and EDD. An intense obesogenic process in a developing brain could have psycho-neurological consequences. Probably none of them is sufficient.

**F.- Prevalence and emotional risk:** The high prevalence of ov/ob among children as its impact on their mental balance and development makes them one of the main risk groups of emotional disorders. To this is joined the late detection of symptoms and thus the constraints for fast therapeutic intervention.

## 9. EFFECT OF CLA ON ADIPOSITY IN PEDIATRIC OBESITY

*M.Moya, M.Juste, F.Carratalá, J.Caturla*

Body mass index (Kg/m<sup>2</sup>) in its different ways of use is reasonably related to adiposity, but it is definitely the deposit of fat, mainly the perivisceral one which is associated to obesity complications. So, if we are able to measure the fat exactly we could minimize the prognostic variability that body mass index implies. The antiadipogenic effects of conjugated linoleic acids (CLA) have been well tested in experimental research, but its use in humans brings some controversial results and there is very little information about its use in the paediatric population.

The aim of the study is to evaluate the evolution the two groups of obese patients receiving the same treatment over a year (diet and physical activity) and one of two groups receiving CLA.

Three groups of patients were studied, a reference group with 17 subjects with a normal BMI and two subgroups of obese patients 14 of them receiving CLA and 8 control group only with the standard treatment. All accomplished treatment for one year

In addition to the general somatometric measures and the usual blood test in the unit, a Dexa was performed at the beginning and at the end of the study period to evaluate changes in the total body fat and the central adiposity one by the study of percentage of total body fat. The most discriminative data changes in the group of patients receiving CLA are in the next table:

**Results:**

	C.L.A (N=14)			CONTROL (N=8)		
	INITIAL	FINAL	P (Z wilcoxon)	INITIAL	FINAL	P (Z-wilcoxon)
Age	11.109±3.6	12.58±3.5	<b>0.005</b>	9.6±3.1	10.0±2.2	0.5
IMC ZS	4.1±1.9	2.88±0.8	<b>0.016</b>	4.31±1.6	3.35±2.3	0.225
% Central Fat	47.39±4.6	44.33±7.7	0.229	42.8±4.9	39.96±9.7	0.5
% Total Fat	48.86±5.6	45.72±7.7	0.221	45.4±4.1	43.38±9.2	0.686

In the CLA Group (n=14, 7 males); a significant increase of age was demonstrated (according to the study design) and a high significant decrease in the rBMI ZS figures was observed. Percentages of central fat and total body fat showed no significant reductions. The control Group results have the same distribution as those in the CLA group but its differences were never significant. Perhaps it is justified by the small number of the sample.

When study about decrease of fatness (total or perivisceral) is started, as in this case, to separate patients by sex is mandatory because of the different fat behaviour in girls which is higher. Teenage girls have a BF percentage around 30 whereas in boys they have almost half of this. Generally the younger teenagers can be united because the increase of BF % is not excessive.

According to the preliminary results of this study, where only 20 of the 80 patients studied finished the year of the observation period it can be concluded that the weight loss and the decrease of the % BF were similar in both groups.

**BIBLIOGRAFY**

1.- Lasa A, Churrua I, Simón E, Fernández-Quintela A, Rodríguez VM, Portillo MP. [Trans-10, cis-12-conjugated linoleic acid does not increase body fat loss induced by energy restriction](#). Br J Nutr. 2008 Dec;100(6):1245-50.

2.- Larsen T, Toubro S, Gudmunson O, Astrup A. Conjugated linoleic acid supplementation for 1 does not prevent weight or body fat regain. Am J Clin Nutr 2006;83: 606-12.

**10. FAMILIAL HYPERCHOLESTEROLAEMIA, TRACKING AND ITS APPROACH TO CARE**

*Rafael Tojo, Rosaura Leis. Unit of Investigation in Nutrition, Growth and Human Development of Galicia, Department of Pediatrics, Clinic University Hospital of Santiago, University of Santiago de Compostela, Spain*

The recent and growing epidemic of obesity favors not only the high prevalence of abnormal lipid metabolism in children and adolescents, but also the risk of type 2 diabetes, hypertension and cardiovascular disease, increasingly younger ages of life. In this context, pediatricians should prioritize the onset of primary prevention in early childhood, paying particular attention to improving lipid and lipoprotein concentrations and to protect the heart. In this line fits the Clinical Report: Lipid Screening and Cardiovascular Health in Childhood, the American Academy of Pediatrics (1), which replaces previous recommendations.

Several aspects deserve the attention of pediatricians: the low nutritional quality of diet, food predominantly high in energy, saturated fat and trans fats, sugar and salt, the lack of physical activity in all its forms and increased sedentary lifestyle, inactivity, increasingly linked to time spent on the screens, especially television and computer, in addition to this teenagers, smoking and oral contraceptive use. Another point to mention here the epidemiological importance of lipid and lipoprotein tracking in time, as demonstrated in children and adolescents in the Muscatine Study and the

Bogalusa Heart Study. A key element of risk identification in the child is family history of hyperlipidemia and early cardiovascular disease.

Of all the alterations of lipid metabolism, characterized by its often polygenic hypercholesterolemia, and its severity, familial hypercholesterolemia (FH), with a presence in heterozygotes of 1:500. The traditional study of abnormal cholesterol concentrations are based on total cholesterol (TC) and LDL-c (NCEP-AAP), but recently the American Heart Association recommends incorporating levels of triglycerides and HDL, because of its importance in the expression risk factors and comorbidities, including the Metabolic Syndrome. Currently, two guides provide recommendations for diagnosis and monitoring. One of the UK National Institute for Health and Clinical Excellence (NICE) (2) and other U.S. American Academy of Pediatrics (AAP) (1). In NICE, the diagnosis should be made according to criteria of Simon Broome (family history, clinical signs, lipid profile and test DNA) and statin use after age 10 (2). The AAP recommends screening from 2 years and not later than 10. The use of statins can be recommended from age 8 (pravastatin) or 10 years (atorvastatin) (1). The AHA Scientific Statement on high-risk lipid abnormalities in children and adolescents (3) highlights the increasing use at younger ages of drugs that have proven their efficacy and safety in adults. In several studies, short-term statin use reveals a significant improvement in lipid profile, vascular endothelial function and regression of intima media thickness (IMT) carotid.

Pediatricians should be sensitive to the possible causal relationship between hyperlipidemia in children and adult cardiovascular events, and therefore the dispute between the benefits and risks of a universal cholesterol screening and increased use of drugs early and aggressive in children, whose long-term effects, benefits and risks are not currently known.

Despite all this, we must bear in mind the importance of screening children and adolescents at risk and therefore the need for third-level units that respond to the comprehensive care of this condition and comorbidities. Moreover, both as prevention and / or intervention in hypercholesterolemia in childhood, promoting healthy lifestyles, especially diet, activity and inactivity play an important role, even as an adjunct to drug therapy.

#### Reference

1. Daniels SR, Creer FR, and the Committee on Nutrition. Lipid Screening and Cardiovascular Health in Childhood. *Pediatrics* 2008;112:198-208.
2. DeMott K, Nherera L, Shaw EJ, Minhas R, Humphries SE, et al. Clinical Guidelines and Evidence Review for Familial hypercholesterolemia: the identification and management of adults and children with familial hypercholesterolemia. 2008. London: National. Collaborating Centre for Primary Care and Royal College of General Practitioners.
3. McCrindle BW, Urbina EM, Denninson BA, Jacobson MS, Steinberger J, et al. A Scientific Statement From the American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth committee, Council of Cardiovascular disease in the Young, With the Council on Cardiovascular Nursing. Drug Therapy of High-Risk Lipids Abnormalities in Children and Adolescents. *Circulation* 2007;115:1948-1967.

## 11. POLIMORFISMOS GÉNICOS ASOCIADOS A LA OBESIDAD

*CM Aguilera García<sup>1</sup>; J Olza Meneses<sup>1</sup>; M Gil-Campos<sup>2</sup>; R Leis Trabazo<sup>3</sup>, M ValleJiménez<sup>4</sup>, R Tojo Sierra<sup>3</sup>, R Cañete Estrada<sup>2</sup>, A Gil Hernández<sup>1</sup>.*

*<sup>1</sup>Departamento de Bioquímica y Biología Molecular II, Instituto de Nutrición y Tecnología de Alimentos, Centro de Investigaciones Biomédicas, Universidad de Granada; <sup>2</sup>Unidad de Pediatría Endocrinológica, Hospital Universitario Reina Sofía, Córdoba; <sup>3</sup>Unidad de Investigación en Nutrición, Crecimiento y Desarrollo Humano de Galicia, Departamento de Pediatría, Hospital Clínico Universitario de Santiago, Universidad de Santiago de Compostela; <sup>4</sup>Unidad de Análisis Clínicos, Hospital Valle de los Pedroches, Córdoba.*

#### ABSTRACT

The prevalence of obesity among adults and children is increasing at an alarming rate. Although the impact of environmental factors is likely to be significant, it is clear that obesity has a large underlying genetic component. Around 5% of obesity cases are due to single-gene mutations (monogenic obesity), and to a number of Mendelian syndromes rarely encountered in the general population. However, for the most part obesity can be attributed to the interaction of certain gene polymorphisms with the environment.

Around 130 genes are currently linked to obesity, and that number continues to grow. In the last years, the genome-wide association (GWA) studies have identified a whole range of genes involved in the etiology of obesity. Among those there are genes encoding hunger- and satiety-signaling peptides, genes regulating adipocyte growth and differentiation, metabolic genes and genes involved in the control of energy output.

The most relevant gene related to obesity is the *fat mass and obesity associated (FTO)*. This gene is involved in the regulation of appetite. To confirm the results from GWA studies, alternative study designs and additional obesity-related phenotype data are considered mandatory. In our research group a total of 1536 SNPs have been genotyped in 777 children, 246 obese (BMI $\geq$ 95<sup>th</sup> percentile) and 309 normal-weight control subjects (25<sup>th</sup>  $\geq$ BMI  $\leq$ 95<sup>th</sup> percentile). In this study we have confirmed the association of genetic variants of *FTO* with obesity in Spanish children.

## 12. INTESTINAL PERMEABILITY

*C. Sierra, J. Blasco, V. Navas, A. Barco, J. Serrano*

*Unidad de Gastroenterología, Hepatología y Nutrición Infantil. Hospital Materno-Infantil. Málaga.*

The intestinal permeability (PI) can be defined like the flow of a solute through a membrane area unit in a time given. It is not synonym of absorption, in which the flow of a solute in a time given occurs, without relation with the area of membrane. The most appropriate concept corresponds to the property that presents the intestinal mucous membrane to modify the penetration of a solute through the intestinal wall.

Lactulose to mannitol (L/M) ratio measurement is a proven, effective index of intestinal permeability. Both lactulose and mannitol are absorbed as whole molecules by the human small intestine. Mannitol is small with a molecular radius of 0.4 nm and easily diffuses through the small intestine cell membrane. Lactulose with a radius of 0.52 nm is too large for intracellular diffusion and therefore relies on paracellular diffusion for intestinal absorption.

The intestinal capacity for absorption of lactulose is a direct measure of the tightness of junctional complexes and thereby a measure of intestinal barrier. The absorption of mannitol is less dependent on intestinal integrity. Therefore, ingestion of lactulose and mannitol simultaneously controls for fluctuations in gastric emptying, intestinal fluid volume, and intestinal transit time, allowing direct measurement of the paracellular pathway. Attaining these molecules for measurement is easily performed with collection of urine. Both lactulose and mannitol have similar volumes of distribution within the body and similar renal clearance with excretion in the urine over a 5-hour period after oral administration.

Lower IP has been showed with term breastfed infants compared to formula-fed infants at postnatal day 7 but not at postnatal day 1 or 30. A study of term infants over the first 6 postnatal days showed decrease in permeability to lactulose with initiation of human milk feedings but not formula feedings. A physiological state of increased IP is described during the immediate neonatal period, probably to permit the transport of macromolecules and to facilitate the development of immune tolerance. The end of this process has been called gut closure and is determined by hormonal and dietary factors.

The integrity of the intestinal barrier can be altered in various pathological situations with intestinal mucosa damage, as feeding allergy, celiac disease, Crohn's disease, enteritis by chemotherapy or radiation therapy, prolonged parenteral nutrition and cystic fibrosis.

## 13. PREVENCIÓN DE LA OSTEOPOROSIS EN NIÑOS QUE NO TOMAN LECHE

*M. Alonso Franch<sup>1</sup>, P. Redondo del Río<sup>2</sup>, C Alonso Vicente<sup>1</sup>*

*D<sup>o</sup> de Pediatría, Obstetricia-Ginecología, Nutrición-Bromatología. Areas de <sup>1</sup>Pediatría y de <sup>2</sup>Nutrición. Facultad de Medicina. Universidad de Valladolid*

Milk is a nourishment having elevated nutritional quality given its low energy density and high nutrient content. Within these, it is necessary to stress that milk is the only dietary source of lactose and a of the principal ones of calcium and phosphorus. During the first months of live, it can cover all of the nutritional needs of the infant, but its importance within the diet progressively decreases with age. One of the reasons that adolescents stop drinking milk is precisely its identification as a food for children. On others occasions, it is an aversion and intolerance to different types that make it necessary to substitute the milk (and sometime the lactic products) with other nutrients.

Bone mineralization shows a positive balance during childhood, until it reaches its maximum (bone mass peak) a few years after growth ends. There are at least 3 critical periods for calcium and phosphorus apposition: the last three months of pregnancy, the first three years of live and puberty. Intake recommendations are adjusted to the physiological changes in bone mineralization although they are type AI (adequate intake), since sufficient data is still lacking to scientifically assure (RDA) the requirements at each age.

Key words: low calcium diet; adolescence; osteopenia; osteoporosis; adequate calcium/phosphorus intake

## 14. DIET AND CONSTIPATION IN CHILDREN. ROLE OF DIETARY FIBER.

*Gutiérrez Junquera C. Unidad de Gastroenterología Pediátrica. Hospital Universitario Puerta de Hierro-Majadahonda. Madrid.*

**Introduction:** Although constipation is less frequent among societies with high intake of dietary fiber the pathogenic role of dietary fibre in constipation in children is still controversial. There are conflicting reports showing that constipated children have a lower, equivalent or higher intake of dietary fiber compared to children without constipation.

**Objective:** To determine dietary fiber intake and its relation with colonic transit time in severe constipated children and in a group of children with normal bowel habits. **Subjects and methods.** Thirty children with normal bowel habits and 38 children with chronic functional constipation, aged 2 to 14 years, were studied. The patient group had a history of chronic constipation for more than 6 months, with or without secondary encopresis, that was refractory to conventional treatment of deimpaction, reeducation of defecatory habits, measures to increase dietary fiber content, and administration of mineral oil or osmotic-type laxatives. The total and segmental colonic transit times were estimated by administering multiple radiopaque markers for 6 days and performing a single abdominal radiograph on day 7. Children in the study ate their normal diets, and the daily caloric and fiber intake was estimated using a Food Consumption Frequency Questionnaire, previously validated for application in current study.

**Results:** The observed upper reference values for colonic transit time were 19.02 hours for the right colon, 19 hours for the left colon, 32 hours for the rectosigmoid colon, and 45.7 hours for the total colon. Fifty percent of the children with chronic functional constipation had colonic transit times within reference values, whereas 37% had left colonic and rectosigmoid delays and 13% had global delay in all colonic segments (colonic inertia). Mean daily fiber intake was  $10,68 \pm 6,62$  g/day. Mean daily dietary fiber intake was lower in the group of children with normal bowel habits compared to constipated children ( $8.8 \pm 5.4$  versus  $12.5 \pm 7.3$  g/day, respectively; although the difference was not statistically significant. Fiber consumption below that recommended ("age + 5"), was found in 50% of constipated children and 52.6% of controls. No significant correlation between fiber intake and colonic transit time was found. Mean colonic transit time was similar in the three groups established considering the dietary fiber intake tertiles (< 8g/day, 8-16 g/day and >16 g/day). Daily caloric and fat intake were directly correlated with colonic transit time.

**Discussion:** We observed that our group of children with severe refractory constipation tended to have higher mean daily fiber intake than the control group, reflecting relative compliance with dietary changes advised in management of constipation. In spite of these measures only 50% of constipated children reached the current recommendation for fiber consumption in children ("age + 5"). No significant correlation between fiber intake and colonic transit time was shown. These results suggest that while a balanced diet including fruits, vegetables and fiber may be useful in preventing and treating mild constipation, the role of fiber in the treatment of severe chronic functional constipation in children is less clear.

#### References

1. Gutiérrez C, Marco A, Nogales A, Tebar R. [Total and segmental colonic transit time and anorectal manometry in children with chronic idiopathic constipation.](#) J Pediatr Gastroenterol Nutr. 2002 Jul;35(1):31-8.
2. Roma E, Adamidis D, Nikolara R, Constantopoulos A, Messaritakis J. [Diet and chronic constipation in children: the role of fiber.](#) J Pediatr Gastroenterol Nutr. 1999 Feb;28(2):169-74.
3. de Carvalho EB, Vitolo MR, Gama CM, Lopez FA, Taddei JA, de Moraes MB. [Fiber intake, constipation, and overweight among adolescents living in Sao Paulo City.](#) Nutrition. 2006 Jul-Aug;22(7-8):744-9.

## 15. IRON INTAKE IN 1ST AND 2ND YEAR OF LIFE

*Henedina Antunes*

*Hospital*

*Gastroenterology, Hepatology and Nutrition Unit, Pediatrics Department, S. Marcos Hospital, Apartado 2242 4701-965 Braga, Portugal*

*Faculdade*

*Life and Health Sciences Research Institute (ICVS), School of Health Sciences, University of Minho, Campus de Gualtar, 4709-057 Braga, Portugal*

Iron deficiency anemia (IDA) is associated with development delay.

Long-term prospective studies show that delay is not completely reversible when IDA is already installed. And these consequences, even after treatment, can persist until adolescence.

Currently, iron deficiency (ID) without anemia, can also be associated with development delay.

Iron in childhood is, therefore, essential to a normal development.

#### Iron intake in 1st year

The infants more than 6 months have greater iron needs (1mg/Kg/day) than in any other period of life.

It is consensual, and compelling in European Community, that all formulas for infants are supplemented with iron.

Since 2003, World Health Organization has recommended the use of iron, *per os*, 1 mg/kg/day, in all infants exclusively breast-feed until 6 months. This supplementation should be use until 12 months.

In premature and intrauterine growth restriction (IUGR) infants, the recommendations are clear and 1 mg/Kg/day should be initiated when they double the birth weight or at 2 months of life (IUGR).

IDA risk is low in infants that are feed with formula fortified with iron, particularly if girls.

In infants, male gender is at higher risk of IDA. This notion is recent and reason is unknown. This predominance might be explained by having more lean mass, even in infant, or a bigger weight increase and therefore more iron needs. Multivariate analysis in my PhD shows that gender is a factor independent of the number of breast-feeding months and weight increase.

A simple risk score to ID may be applied, at 6 months of life, including the number of months of breast-feeding, weight increase and gender. Iron supplementation should not be started before 6 months without blood ID screening.

#### **Iron intake the 2nd year of life**

The discussion to give iron supplements in 1st year of life, in high risk groups, is valid. But, in 2nd year of life healthy children don't need.

Milk fortified with iron is a good way to give iron in the 2nd year of life. However, is expensive and the sweat taste can difficult the posterior introduction of cow milk. Cereals should also be iron supplemented. Actually, it is really important to learn how to eat.

The excess of calcium or its use with the lunch and diner meals can be the most important reason to ID in 2nd year of life. The nutritional advices need to include fish or meat daily.

The higher IDA risk that is present at 2nd year of life can be prevented with a correct nutrition.

## **16. CAROTID INTIMA-MEDIA THICKNESS IN HEALTHY CHILDREN WITHOUT CARDIOVASCULAR RISK FACTORS.**

*\*Federico Argüelles Martín.*

*\*\*Ana M<sup>a</sup> Argüelles Arias*

*\*Libia Quero Acosta.*

*\* Section of Paediatric Gastroenterology*

*\*\*Department of Radiology*

*Hospital Universitario Virgen Macarena. Sevilla.*

The consequences of atherosclerosis, such as coronary heart disease (CHD), rank among the most important public health issues. On the other hand, the development of non-invasive methods, such as high-resolution ultrasonography, now permits clinical assessment of abnormalities in vascular structure and function.

The arterial wall contains three layers: intima, media and adventitia. Atherosclerosis is a disease affecting the intima leading to intimal thickening. The intima-media thickness may be measured with ultrasound so an increase in intima-media thickness in atherosclerotic prone areas is used as an indicator of intimal thickening.

Although the clinical end-point of atherosclerosis mainly occurs in middle age or later in life, observations in pathology studies revealed that atherosclerotic lesions begin during childhood. In addition, some of the traditional cardiovascular disease (CVD) risk factors, which include hypercholesterolemia, hypertension, cigarette smoking, diabetes and obesity, may have an association with the development of atherosclerosis in adolescents or young adults. Recent studies have consistently shown that CVD risk factors identified in childhood predict the carotid intima-media thickness (CIMT) in adulthood. These observations suggest that early detection of modifiable CVD risk factors in this population may have an impact on health in later life.

Leeson et al. [1997] reported a relationship between **flow-mediated dilatation** (FMD) and birth weight in children between 9 and 11 years of age. They suggested that endothelial function, a key factor in atherogenesis, is "programmed" in early life and thereby influences the long-term risk of developing significant cardiovascular disease. Vascular endothelium is a dynamic endocrine organ that regulates vascular tone, local homeostasis, and the fibroinflammatory proliferative process. Vascular endothelial injury is subclinical in patients with cardiovascular risk factors or coronary artery diseases.

Increased CIMT and enlarged left ventricular mass (LVM) are reported to occur in an earlier phase of the atherosclerotic process. Pathologic studies of children and young adults suggest that the process depends on both the number and extent of risk factors identified in childhood that are predictive of adulthood risk for coronary artery disease.

Findings have shown that the risk factors for subclinical atherosclerosis are similar to the risk factors for clinical CVD. Augmented carotid CIMT has been taken as evidence of subclinical atherosclerosis (endothelial dysfunction) and as a strong predictor of subsequent arteriographically documented vascular lesions, myocardial infarction, and stroke. Age, family history of CVD and obesity has been associated with increased CIMT in children. On the other hand, the longitudinal impact of these associations is not fully understood, and there are few reports regarding intima-media complex in healthy children.

Our study was realized in Macarena Hospital in Seville, Spain. It was cross-sectional study enrolled 160 asymptomatic children (100 males and 60 females) without cardiovascular risk factors. Age at examination ranged

from 6 to 14 years (mean  $9 \pm 2.7$ ). The study protocol was approved by the Hospital Ethics Committee, and a written consent was obtained from each subject's parents or guardian before enrolment.

Body composition was measured in all participants, using anthropometry (weight, height, skin fold) and electric bioimpedance (Tanitas) to obtain percentage body fat. The World Health Organization (WHO) standard definitions for overweight and obesity were followed for classification of the research subjects. We excluded everybody with BMI  $> 95$ . Data from a nationally representative cross-sectional Spain's growth study—Obergozo Foundation—were used in computing and for age and sex correction of individual BMI, which were used in statistical analyses. Blood pressure determinations followed the established criteria for children. Exclusion criteria included a medical history or evidence with physical examination of cardiovascular disease, diabetes mellitus, hypertension, endocrine disorders, or medication that could affect cardiovascular function or metabolism. A 5-ml venous blood sample was collected from each subject for biochemical determinations after a 12-h fasting period, were measure Hematologic study, glucose, total cholesterol (T-CHOL), high-density lipoprotein (HDL), and triglycerides (TGL) and Low-density lipoprotein (LDL) cholesterol.

The internal carotid artery, carotid bulb, and common carotid artery (3 measurements were realized in each side) were examined on both sides with a 7.5 MHz linear transducer and ultrasound equipment (TOSHIBA Aplio XV) using the technique described by Wikstrand(2007). All the ultrasound scans and CIMT measurements were performed by a single investigator.

To ascertain whether the subjects had a family history of CVD and sporting activity, we utilize one standardized questionnaire.

Only perform sport, % body fat and age, were related to CIMT in a multiple linear (forward) regression model that included the variables of age, gender, family history of CVD, triglycerides, BMI.

The value of CIMT average (of the 6 measures conducted in each patient) of the group of study had one mean of 0,49 mm with a standard deviation (SD)  $\pm 0,098$  mm

There are few reports regarding intima-media complex in healthy children, but the relationship between age intima-media complex in previous studies is positive. In our study group, CIMT was significantly correlated with age. **CIMT (males) =  $0.15 + 0.37 \times \text{age (years)}$  and CIMT (females) =  $0.17 + 0.034 \times \text{Age (years)}$ .**

Internal carotid artery plaques, which were seen in the arteriosclerotic artery, were not detected in any of our patients. Furthermore, there was no significant difference in intima-media complex between genders.

## 17. OBESITY IN CHILDREN AND SUBCLINIC CARDIOVASCULAR DISTURBANCES

*C. Ruipérez, C. Castaño, M. Juste, M. Moya*  
*Hospital Clínico Universitario de San Juan Alicante.*

**Introduction**— The effect of weight in the cardiac structure and function in obese children is not well established. Obesity is associated with hypertension and myocardial dysfunction, independent of comorbidities. We sought whether body mass index (BMI) with subclinical myocardial disturbances.

**Methods and Results**— Systolic and diastolic blood pressure were determined. Transthoracic echocardiography, Using pulsed-wave Doppler, mitral inflow velocities, peak early diastolic velocity (E), peak late diastolic velocity (A), E/A ratio, and isovolumetric relaxation time (IVRT) were measure. Were obtained in 102 overweight or obese subjects ( $r\text{BMI} > 121\%$ ) and 37 controls ( $r\text{BMI} < 111\%$ ). BMI correlated with left ventricular (LV) mass and wall thickness ( $P < 0.05$ ). LV wall thickness, diameters, volumenes and LV mass indexed to height increased with increasing BMI. In the analysis were significantly different ( $p < 0.05$ ), SIVTD, DTDVI, FA, FE, TRIV. These morphological and functional measures, were significantly different in obese groups as compared with the referents. In obese groups the LV ejection fraction remained normal. The systolic and diastolic blood pressure were bigger in obese subjects (112.6/66.2mmHg) than the control group (97.2/53.5mmHg), were significantly different ( $p < 0.05$ ). The arterial pressure were  $< P90$  in obese groups.

**Conclusions**— Overweight and obese children have subclinical changes of left ventricular structure. Ejection fraction is significantly different, were mayor in the referents group, but in two groups were normal. The systolic and diastolic blood pressure were mayor in obese group the controls.

## 18. HELENA PROJECT. ACCELEROMETERS

*Luis A. Moreno*  
*E. U. Ciencias de la Salud, Universidad de Zaragoza, [lmoreno@unizar.es](mailto:lmoreno@unizar.es)*

Physical activity is defined as “any body movement produced by skeletal muscle that results in energy expenditure”. Low levels of physical activity are associated with an increased risk for developing several chronic diseases, including obesity, cardiovascular diseases, diabetes and colon cancer. Physical activity is a complex behaviour that is difficult to measure in free living conditions over extended periods of time. To describe levels and patterns of physical activity, information regarding the duration, frequency and intensity needs to be assessed. Accurate assessment of physical activity in children is necessary to identify current levels of activity and to assess the effectiveness of intervention programmes designed to increase physical activity.

The most common way to measure activity are self report questions, which provide simple and acceptable methods. Because limitations of self reported physical activity, it has been an increasing interest in objective physical activity measurement. The simplest and cheapest device is the pedometer, but pedometers can not be used to describe the intensity of the performed physical activity. Another option, is heart rate monitoring, but heart rate monitoring needs individual calibration; furthermore, the heart rate is highly influenced by other factors than physical activity, such as stress.

The most realistic option is the use of accelerometers. Accelerometers are sophisticated electronic devices that measure accelerations produced by body movement. When applied to the measurement of physical activity, an accelerometer can assess the magnitude and total volume of movement as a function of time. Most accelerometers used in physical activity assessment operate using piezoelectricity derived from microscopic crystalline structures. In such designs, force created from acceleration causes crystals to become stressed/compressed, which in turn generates an electric charge proportional to the magnitude of the acceleration force. The generated electric charge is filtered and converted by the accelerometer in samples taken multiple times every second. These samples are summed over a user-specified cycling period, known as an epoch (e.g. 1s, 15 or 60 s), and are recorded to the accelerometer’s internal memory. After recording the magnitude of the accelerations over a given epoch in activity “counts”, the numerical integrator is reset and the process is repeated. As vertical ambulatory movements or movements of the trunk generate the most physical activity-related energy expenditure, all accelerometers measure movement in the longitudinal axis (up and down movement). Some accelerometers are also designed to measure in two (biaxial) or three (triaxial) axis.

Accelerometers were used in HELENA, together with traditional questionnaires. The HELENA Study used the MTI Actigraph (accelerometer) model GT1M (ActiGraph, Fort Walton Beach, FL, USA.) which is the most commonly used accelerometer. In European adolescents, the objective assessment of physical activity showed that girls spent more time inactive than boys. Compared to girls, boys were more active at moderate and vigorous physical activity.

## **19. MATERNAL OBESITY DURING PREGNANCY: PRESENT AND FUTURE MECHANISMS AND CONSEQUENCES FOR THE OFFSPRING.**

*Juan A. Molina Font<sup>1</sup>, M. C. Collado<sup>2</sup>, L. García-Valdés<sup>1</sup>, M. T. Segura<sup>1</sup>, T. Anjos<sup>1</sup>, J. Martino<sup>1</sup>, M. Martí-Romero<sup>3</sup>, R. M. Lopez<sup>3</sup>, Yolanda Sanz<sup>2</sup>, Cristina Campoy<sup>1</sup>*

<sup>1</sup>*Department of Paediatrics. School of Medicine. University of Granada. Spain.*

<sup>2</sup>*Institute of Agrochemistry and Food Technology (IATA), Spanish National Research Council (CSIC), Valencia, Spain.*

<sup>3</sup>*Department of Obstetrics and Gynecology. University Clinical Hospital San Cecilio de Granada.*

Recently, the study in 150000 Swedish pregnant women has reported that obesity during pregnancy will clearly increase the risk of the pregnancy outcome<sup>1</sup>, but it will be also source of health risk for the offspring, and actually the health and wellbeing of the future generations. The early programming of obesity could take place due a permanent modification of one or more of the relevant pathways during the early development. Mother’s genetics, diet during pregnancy and lactation and infant feeding during first step of life may have long term effects on the infant health, and could predispose the baby to suffer illnesses such obesity. Experimental studies in animals show that the fetus and the newborn could be receptors of different hormonal and dietetic damage effects, which could converge in a common phenotype of hyperphagia, obesity, altered adipocyte function and low physical activity. Different mechanisms during critical windows of development could be involved in the early programming of adult obesity: 1) Adipose tissue formation and leptin synthesis and secretion regulation before birth; 2) Related genes with the obesity development: FTO, INSIG 2, MC4R, PPAR $\gamma$ 2 Pro12Ala y Ala12Ala, LEP, POMC C8246T y C1032G Polymorphisms and the epigenetic changes of fetal genome; 3) Prenatal nutrition, birth weight, placenta/fetal size ratio and postnatal growth rate; 4) Programming of the Hypothalamic neuroendocrine circuitry and “adipose tissue-brain axis”. In the obese mother, with glucose intolerance, and especially in the diabetic ones, the mother and fetus plasma levels of glucose are higher, and this determines a higher birth weight in their offspring, which is related also with higher body mass index (BMI) in adult life with a high risk for obesity and glucose intolerance development. On the other hand, recent studies have shown that deviations of gut microbiota composition and low systemic inflammation level predispose to an excessive energy store and so to obesity. Gut microbiota is an important factor which could influence the human health through the impact with gut barrier, the immune system development and the utilization of nutrients. The animal studies suggest that the nature and composition of gut microbiota are altered in obesity. The first published results in humans shown that lean individuals have more *Bacteroides*, while obese ones have more *Firmicutes*, including *Clostridium* clusters, in their intestinal microbiota. It has been proposed that this gut microbiota composition determines a higher energy extraction from food intake and so a higher store in the adipose tissue.

Although mother obesity is associated to complications during pregnancy and the risk of obesity and other illnesses in the offspring, the relationship with the changes in gut microbiota composition is not clear. The aim of the present study is to analyse the gut microbiota composition in obese pregnant women to compare with normal weight mothers and to establish potential relationships between changes in gut microbiota, weight gain and biochemical parameters during pregnancy.

**Methods: Subjects:** A total 50 pregnant women were recruited at the 12 weeks of pregnancy and followed-up until delivery; 16 were overweight and 34 had an adequate weight per height just before pregnancy, following the body mass index (BMI). **Microbiological and Biochemical Analysis:** The gut microbiota composition was analysed using quantitative PCR (qRT-PCR). The biochemical parameters glucose, cholesterol, urea, total proteins, amilase, ferritin,... between others were analysed in each trimester of the pregnancy. **Statistical Analysis:** Statistical analyses were done using the SPSS 15.0 software (SPSS Inc, Chicago, IL, USA). The microbiological and biochemical data have been expressed as median and interquartils due they did not follow a normal distribution. The Mann-Whitney U-test as well as Chi-square test were used. A correlation Spearman and Pearson analysis were performed. The minimum level of significance was  $p < 0.05$ .

**Results:** Significant higher levels of *Bifidobacterium* and *Bacteroides* were found in the normo-weight pregnant women, however, the overweight ones showed a different trend in the gut microbiota, with significantly higher levels of *Staphylococcus*, *Enterobacteriaceae* y *E. coli*. Moreover, the *Enterobacteriaceae*, *E. coli* and *Bacteroides* levels were significantly higher in those women with an excess of weight gain during pregnancy in front of those with a normal weight gain. On the contrary, the higher levels of *Bifidobacterium* group and *Akkermansia* were significantly correlated with the normal weight gain during pregnancy. The cholesterol and triglycerides levels were significantly higher and cholesterol-HDL lower during pregnancy in the overweight mothers respect to mothers with normal weight. These parameters were also correlated with the weight and the BMI. Higher levels of *Staphylococcus* observed at the 2<sup>o</sup> trimester of pregnancy were positively correlated with the higher concentrations of cholesterol ( $r=0.68$ ,  $p=0.003$ ). The *Enterobacteriaceae* and *E. coli* concentrations were correlated with high levels of ferritin and the transferrin saturation ratio and with low levels of transferrin. However, higher levels of *Bifidobacterium* were related to low levels of ferritin and transferring ratio, as well as with higher levels of transferrin and folate. The *Bacteroides* genus concentrations were correlated with high levels of cholesterol-HDL and folic acid and with low levels of triglycerides.

**Conclusion:** Gut microbiota composition, mother weight and weight gain during pregnancy are related to an “obesogenic” change of gut microbiota. More studies are needed to understand the effect of these changes of gut microbiota on the offspring gut “imprinting” and the long-term consequences for the offspring health.

**\*\* This work was supported by grants P06-CTS-02341 (Excellence Project PREOBE) from Consejería de Innovación, Ciencia y Empresa de la Junta de Andalucía, (Spain), AGL2008-01440/ALI, Consolider Fun-C-Food CSD2007-00063 from the Spanish Ministry of Science and Innovation, AP-124/09 from Conselleria de Sanitat (Valencia, Spain).**