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Original / *Síndrome metabólico*

Effect of a long-term physical exercise program and/or diet on metabolic syndrome in obese boys

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Abstract

Introduction: **There have been just a few studies examining the influence of detraining on obese boys. They conclude that any gains regress to the untrained control values during the detraining period.**

Objective: **The objective of the present study was thus to evaluate the effects of detraining (6 months) on metabolic syndrome after two types of intervention (both 31 months), one of an exercise program alone and the other of a diet-plus-exercise program, in obese boys.**

Methods: **The participants were 18 sedentary boys (8-11 years old) with a body mass index equal or greater than the 97th percentile for the age and sex (male) of the subject, without any dysfunction or metabolic problem. The participants were divided into two groups – the E group (physical exercise program) and the E+D group (physical exercise program plus a low calorie diet). Metabolic parameters were evaluated (TC, HDL, LDL, TG, glucose, insulin, Systolic Blood Pressure, and Diastolic Blood Pressure), allowing the metabolic syndrome index to be calculated.**

Results: **Changes were observed in LDL-C (effect sizes = -3.19 and -2.28) and in the LDL-C/HDL-C ratio (effect sizes = -3.02 and -1.16) in the E and E+D groups, respectively. The prevalence of metabolic syndrome and obesity was completely removed only in the E group (100% no-risk and non-obese subjects – < 90th percentile).**

Conclusions: **Detraining from a long-term exercise program (with or without diet) seems not to negatively affect the cardiovascular profile, suggesting that the program provides benefits and fosters healthy habits that can be maintained over time, preventing the development of metabolic syndrome.**

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Key words: *Obese boys. Detraining. Physical exervise. Longitudinal intervention. Metabolic syndrome.*

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EFECTOS DE UN PROGRAMA DE EJERCICIO FÍSICO Y/O DIETA A LARGO PLAZO SOBRE EL SÍNDROME METABÓLICO EN NINOS OBESOS

Resumen

Introducción: **Existen pocos estudios que examinen la influencia del desentrenamiento en niños obesos. Estos estudios concluyen que tras el desentrenamiento se regresa a los valores iniciales antes de la intervención.**

Objetivo: **El objetivo del presente estudio fue examinar los efectos del desentrenamiento (6 meses) sobre e l síndrome metabólico después de dos tipos de intervención (31 meses), una de las intervenciones consistió en un programa de ejercicio físico aislado y la otra además del ejercicio físico incluyó la dieta.**

Métodos: **Participaron 18 niños sedentarios (8-11 años) con un índice de masa corporal igual o superior al percentil 97 en función de la edad y sexo (varones) sin ninguna disfunción o problema metabólico. Los participantes fueron divididos en dos grupos: Grupo E (realizó el programa de ejercicio físico) y grupo E+D (realizó el programa de ejercicio físico más una dieta hipocalórica). Se evaluaron los parámetros metabólicos (CT, HDL-C, LDL-C, TG, glucosa, insulina y presión sistólica y diastólica) que permitieron calcular el síndrome metabólico.**

Resultados: **Se observaron cambios en el LDL-C (TE = -3.19 y -2.28) y el índice LDL-C/HDL-C (TE = -3.02 y -1.16) en el grupo E y E+D respectivamente. La prevalencia del síndrome metabólico y obesidad fue eliminada por completo en el grupo E (100% sin riesgo en niños no obesos – < percentil 90).**

Conclusiones: **El desentrenamiento tras un programa de ejercicio a largo plazo (con y sin dieta) parece no tener efectos negativos en el perfil cardiovascular, sugiriendo que el programa aporta beneficio sobre la salud (síndrome metabólico) que se mantienen en el tiempo.**

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Palabras clave: *Niños obesos. Desentrenamiento. Ejercicio físico. Intervención longitudinal. Síndrome metabólico.*

Abbreviations

BMI: Body mass index.
DBP: Diastolic Blood Pressure.
ES: Effect size.
MetS: Metabolic syndrome index.
PA: Physical activity.
SBP: Systolic Blood Pressure.

Introduction

The worldwide prevalence of childhood obesity has increased dramatically over the past three decades, and has come to be considered a pandemic of the 21st century[1]. The mechanisms responsible for the increasing prevalence of childhood obesity are not fully understood, although lifestyle changes characterized by increased caloric intake and decreased energy expenditure play a key role[2,3]. Various instruments are available to assess or estimate physical activity (PA) prior to intervention aimed at increasing it: questionnaires such as the IPAQ (International Physical Activity Questionnaire), pedometers, or accelerometers. These last have been widely used in children[4].

Governments worldwide are working to implement strategies to prevent obesity, encouraging the pursuit of healthier lifestyles[5]. Prevention, especially in young people, is universally regarded as the best method to reverse the rising global prevalence of obesity[5]. It is known that changes in lifestyle habits can reduce the risk of cardiovascular disease in adulthood[1]. In this sense, the latest Cochrane Library review suggests the need for longitudinal studies, because they would provide invaluable information on the sustainability of the beneficial effects of these interventions in overweight and obese children[5]. Many studies have examined how programs of physical exercise and/or diet plus physical exercise affect metabolic parameters in obese children. Few, however, have assessed how the subsequent detraining might influence those parameters in this obese population[6-8].

Detraining has been defined as the partial or complete loss of training-induced anatomical, physiological, and performance adaptations as a consequence of training reduction or cessation[9]. In this regard, a recent study has confirmed that there has been a marked lack of work following up the detraining after programs designed to treat pædiatric cases of obesity[8]. The few studies that exist on detraining indicate that, following short-term (3-4 months)[7,8,10] and medium-term (9 months)[6] physical exercise programs alone[8,9,11] or in combination with diet[7], there is usually a negative effect on various metabolic parameters[7,8,10]. In particular, the results show negative changes in triglycerides[6-8,10], insulin[6,10], glucose,[10] and HOMA-IR[6] following the detraining period. These studies show that, while supervised exercise can slow the progress of obesity, and improve insulin sensitivity and metabolic risk factors, once the supervised exercise has stopped, the health benefits weaken or vanish[6]. There have as yet been no studies examining the influence of long intervention programs (of at least one year) on obese boys' detraining. Long-term intervention might generate more consistent and lasting benefits[9]. Given this context, the objective of the present study was to evaluate the effects of detraining (6 months) on metabolic syndrome after an intervention (31 months –including holiday period) of an exercise program and of a diet-plus-exercise program in obese boys.

Material and methods

Study design

The study was of a quasi-experimental design, with the intervention being conducted from november 2007 to june 2011.

Subjects

A total of 105 boys were invited to participate through the collaboration of various schools in the town of Cáceres (Spain). The inclusion criteria were: (i) a body mass index (BMI) equal to or greater than the 97th percentile for the age and sex (male) of the subject, and (ii) age between 8 and 11 years as defined by Spanish population curves[11]. Subjects were excluded if they were: (i) regularly practising physical activity (PA), or following an exercise program or some other therapy (n = 65); (ii) involved in any weight control program (n = 18); (iii) were taking any medication (n = 8); (iv) had any type of dysfunction limiting their PA (n = 2); other reasons (metabolic problems, unable attend the exercise program, etc.) (n = 24). The final sample consisted of 18 Caucasian boys (10.7±0.9 years). They were divided into two groups the exercise group (E group) who followed a multi-sports exercise program (n = 8, 10.9 ± 1.0 years), and the physical exercise program plus a low calorie diet (E+D group) who followed a combination of two programs –the exercise program and a low calorie diet (n = 10, 10.5 ± 0.85 years). Several subjects ate at their school's refectory (were included in the E group), making it impossible to randomly assign membership to one or the other group. All the children's parents completed a prior informed consent form. The subjects were referred to the Servicio de Pediatría del Hospital San Pedro de Alcántara of Cáceres (Spain) for evaluation. The study was approved by the Bioethics and Biosafety Committee of the University of Extremadura (Spain) and respected the principles of the Declaration of Helsinki.

Interventions

Exercise program

The exercise program was based in a multi-sports hall, supervised by two MSc's in Sports Sciences (AGH & AMD), and under the overall supervision of two PhD's in Sports Sciences (JMS & YE). The program design was based on previous studies[6,7] and on the more than 15 years experience in implementing this type of health-related exercise program of two of the authors (JMS & YE). The program was of three weekly 90-min sessions. It comprised a warm-up (15-20 min), a main part consisting of pre-sports and multi-sports games with a moderate to vigorous intensity aerobic component (60-65 min), and a cool-down (5-10 min). A progression was established to steadily ramp the subjects up to 60-65 minutes of moderate to vigorous intensity (table I). The intensity of the session was monitored by accelerometry to ensure that all the subjects performed the activities with the same intensity. A Caltrac accelerometer (Hemokinetics, Madison, WI, USA) was used to this end, programmed to function as a PA monitor[12]. This uniaxial accelerometer contains a piezoelectric bender element which assesses the intensity of movement in the vertical plane. Its validity has been demonstrated as a method for estimating energy expenditure in children[13], and it has been used in other studies[12,14]. Although it does not record such activities as rowing or swimming, no activity of this type was used either in the exercise program or in the subjects' daily PA for the duration of the study.

Compliance was assessed as percentage of exercise sessions attended, and was found to be good, with the children attending more than 78% (81% and 77% in E and E+D group, respectively) of the total exercise sessions (230 sessions). Quantifying the intensities of 13 of the sessions/year selected at random showed no significant differences between the E and the E+D groups in any session, with a mean of 79 and 81 motion counts per session. In the measurement sessions, each child wore an accelerometer. The values taken were the mean of the exercise performed by the group. Not all the sessions were quantified since the programming and placement of the accelerometers meant taking time away from the physical exercise program. The use of accelerometers allows one to objectively quantify the subjects' PA, ensuring that the intensity was similar in the two groups. In developing treatment strategies for obesity, one requires quantitative information on PA to provide more effective goals[15].

Table I
Main exercise intervention program

Time (months)	*Exercise intensity**	*Exercise duration (min)*
0-6	Moderate	60-65
6-12	Moderate	40
	Moderate to Vigorous	20-25
12-18	Moderate	30
	Moderate to Vigorous	30-35
18-24	Moderate	15
	Moderate to Vigorous	45-50
24-31	Moderate to Vigorous	60-65

* All exercise training sessions started with 15-20 minutes warm-up exercises and ended with 5-10 minutes cool-down exercises.

Diet program

The low-calorie diet consisted of five balanced meals spread throughout the day, with an energy intake of 1,500 kcal/day. It was designed by two endocrinologists (JA & PB) of the Pædiatric Service of the San Pedro de Alcántara Hospital in Cáceres (Spain). In this sense, there have been studies that recommend diets of between 1,500 and 1,800 kcal/day in obese children who are still growing, since in this way their growth and development are not compromised. Thus the diet prescribed was of 1,500 kcal/day, similar to that of other studies[16]. The diet consisted of 57% carbohydrates, 17% proteins, and 26% fats. Foods were selected according to the subject's dietary habits. A series of general recommendations were established focused on basic healthy lifestyle eating: consume ≥ 5 servings of fruits and vegetables every day; minimize sugar-sweetened beverages such as soft drinks, sports drinks, and sugar-added fruit juices; have more meals prepared at home rather than purchasing take-away restaurant food; etc. Regular meetings were held with the children's parents for the control and monitoring of the diet.

Measurements

Each subject was evaluated for the following parameters: eating habits, daily PA, pubertal status, and metabolic parameters. The evaluations were made at the start (baseline), and at 31 (3rd-year) and 37 (detraining) months into the program.

Eating habits

Nutrition was assessed with a self-reported 3-day food record (2 weekdays and 1 weekend day in succession – Thursday, Friday, and Saturday) filled in by the parents. The weight of the food was estimated from the parents' records. A computerized database NutrIber was used to calculate the daily intake[17]. Thus the program recorded the average of the three days (kcal/day).

Daily physical activity

Daily PA was measured before the intervention, during the follow-up, and in detraining with a validated

uniaxial accelerometer (Caltrac) covering a 3-day period (thursday, friday, and saturday), except during bathing and swimming. All participants were instructed to record the amount of time spent cycling or swimming during the evaluation period. At the beginning and the end of the day, the subjects recorded the number of "motion counts" of the accelerometer, following previously published protocols[12]. The final Caltrac score was recorded, as also was the average of the three days (motion counts per day). Figure 1 shows the evolution of the physical activity carried out over the three years and the detraining period.

Pubertal status and kinanthropometry

Pubertal stage was evaluated by a trained pædiatrician according to pubic hair development using the Tanner classification criteria[18]. This evaluation was applied to ensure that changes throughout the exercise program were not due to differences in the evolution of pubertal status, which otherwise could influence the results. The kinanthropometric measurements followed the International Standards for Anthropometric Assessment (ISAK) protocol[19]: body height, body weight, and waist circumference. Height and weight were measured using standard procedures. BMI was calculated as weight divided by height squared (kg/m^2), and the obese prevalence were determined[11]. Waist circumference was measured three times with a flexible tape at the midpoint between the last rib and the iliac crest at the end of a normal expiration. Standard equipment was used: a scale-mounted stadiometer (Seca, Berlin, Germany), a weight scale (Seca, Berlin, Germany), and a non-extensible measuring tape (Holtain, Crymych, Great Britain).

Metabolic parameters

A venous blood sample was collected after at least 10 h overnight fasting. Aliquots of 5 mL were then assayed for the concentrations of plasma total cholesterol (Chod-Pad assay, automatic analyser), HDL (HDL-C plus assay, automatic analyser), triglyceridæmia (Chod-Pad assay, automatic analyser), blood glucose (glucose HK assay, automatic analyser), and insulinæmia (human insulin RIA kit, Linco Research, Missouri, USA). The brachial resting BP was measured 3 times at 2-min intervals after 10 min of rest in supine position with the back supported, using a validated automated device (OMRON RX, Matsusaka, Japan). The homeostasis model assessment of insulin resistance (HOMA-IR) was used to measure the changes in insulin sensitivity among the subjects, and was calculated using the following formula[20]: fasting plasma insulin (μU/mL) × fasting plasma glucose (in mg/dL) ÷ 22.5 × 18.182. The following atherogenic indices were also calculated: LDL/HDL ratio and TC/HDL ratio. The mean arterial pressure was then calculated using the following formula: DBP + (0.333 × (SBP-DBP)).

Metabolic Syndrome Index

The Metabolic Syndrome (MetS) index was calculated as the sum of the age-sex standardized scores of waist circumference, TG-to-HDL-C ratio, mean arterial pressure, and fasting insulin. The validity of this MetS index has been previously tested using confirmatory factor analysis[21]. Metabolic syndrome was considered to be present when a boy had a MetS index above 4.2.

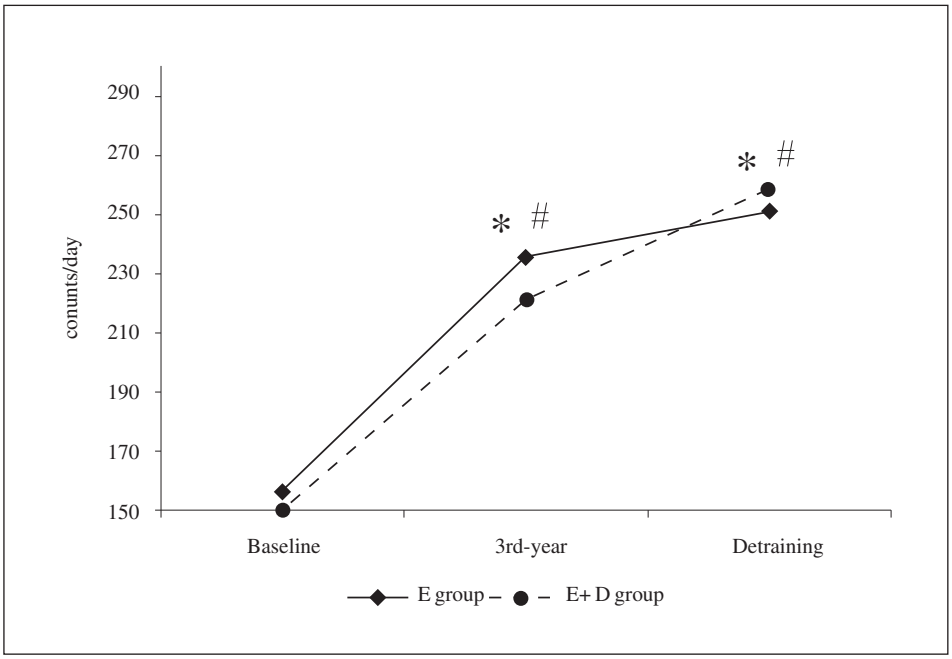

*Fig. 1.—Changes in daily PA at the baseline, third year, and detraining evaluations in obese boys; *p < 0.05 in the E group; #p < 0.05 in the E+D group.*

Detraining

At the end of the 3-year intervention programs, the participants were encouraged to join sports activities independently. Thus, 72% of the E group subjects and 75% of the E+D group subjects performed regular physical exercise during detraining, thereby increasing their daily PA compared to the baseline. Following detraining, the subjects were invited back to undertake a re-evaluation of their metabolic parameters. The same methods described previously were used in the re-evaluation.

Statistics

All the variables satisfied the tests of homoskedasticity (Levene test) and normality (Kolmogorov-Smirnov test) of their distributions. We nonetheless used a non-parametric test as is recommended in cases of small samples[22]. The basic descriptive statistics (means and standard deviations) were calculated. The Kruskal-Wallis test was applied to reveal overall inter-group differences (between the E and E+D groups), and measurements showing significant differences were further analysed for differences between individual groups by the Mann-Whitney U test (baseline *vs* detraining, and 3rd year *vs* detraining). Cohen's categories were used for the magnitudes of the effect size (ES): small if $0 \leq |d| \leq 0.2$; medium if $0.2 < |d| \leq 0.5$; and large if $|d| > 0.5$[23]. Metabolic syndrome and obese prevalence were compared using Pearson's χ^2. The level of significance for all statistical tests was set at $p \leq 0.05$. All calculations were performed using SPSS (version 16.0).

Results

The variables satisfied the tests of normality (Kolmogorov-Smirnov: $0.351 \geq z \leq 1.142$, $p > 0.05$) and homoscedasticity (Levene: $0.008 \geq F \leq 3.784$, $p > 0.05$). Also, there were no inter-group differences in eating habits, daily PA, ponderal status, or metabolic parameters at the start of the program. Table II lists the characteristics of the groups.

Intra-group differences

Table III shows the changes and treatment effects at the different evaluations (baseline [a], 3rd-year [b], and detraining [c]) in the kinanthropometric and metabolic parameters. Changes were observed neither in daily PA nor in pubertal status until the detraining (table II). This indicates that the changes brought about by the intervention cannot be attributed to these variables. However, there were changes in obese prevalence and both groups in detraining ($p < 0.05$) (fig. 2a). For the E group, there were differences between different moments of evaluation in TC (baseline *vs* detraining, $p = 0.014$, ES = -3.75; b>c, $p = 0.025$, ES = -1.40), LDL-C (baseline *vs* detraining, $p = 0.014$, ES = -3.19), LDL-C/HDL-C ratio (baseline *vs* detraining, $p = 0.014$, ES = -3.02), the TC/HDL-C ratio (baseline *vs* detraining, $p = 0.014$; ES = -0.46), and glycæmia (3rd-year *vs* detraining, $p = 0.024$, ES = 3.44). For the E+D group, there were differences between evaluations in LDL-C (baseline *vs* detraining, $p = 0.004$, ES = -2.28), the LDL-C/HDL-C ratio (baseline *vs* detraining, $p = 0.017$, ES = 1.62), insulinæmia (baseline *vs* detraining, $p = 0.034$, ES = 1.10), and HOMA-IR (baseline *vs* detraining, $p = 0.047$, ES = 1.03).

With respect to obesity, in the E group its prevalence was reduced to 0% (all boys below 90th percentile). In the E+D group, 20% of boys did not cease to be obese (fig. 2a). For its parts, regarding to metabolic syndrome, in the E group its prevalence was reduced to an index below the threshold established as representing metabolic risk in 100% of the subjects ($p = 0.001$). In the E+D group, the equivalent proportion with reduced prevalence was 50% of the subjects ($p > 0.05$) (fig. 2b).

Inter-group differences

Table IV shows the inter-group differences in metabolic parameters. There were no differences between the two groups ($p > 0.05$).

Discussion

The present study analysed the effects of detraining (six months) from a longitudinal intervention program

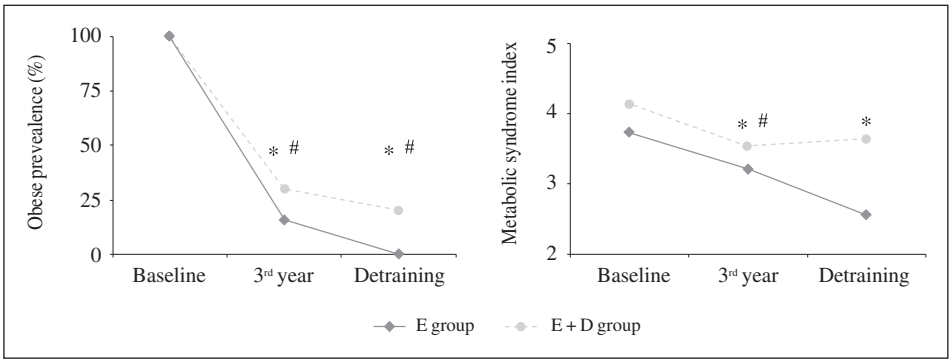

*Fig. 2.—Changes in the Metabolic Syndrome Index (a) and ponderal status (b) at the baseline, third year, and detraining evaluations in obese boys. Prevalence of metabolic syndrome and obesity: *p < 0.05 in the E group; #p < 0.05 in the E + D group.*

Table II
Eating habits, daily PA, pubertal status, kinanthropometric, ponderal status, and metabolic syndrome index at baseline, third year, and detraining evaluations in obese boys

	Baseline			*Third year*			*Detraining period*		
Variables	*E group Mean ± SD (n = 8)*	*E+D group Mean ± SD (n = 10)*	*Intergroup differences p*	*E group Mean ± SD (n = 8)*	*E+D group Mean ± SD (n = 10)*	*Intergoup differences p*	*E groups Mean ± SD (n = 8)*	*E+D group Mean ± SD (n = 10)*	*Intergroup differences p*
Eating habits									
Energy intake (kcal/day)	1952.4 ± 202.8	1928.6 ± 257.4	0.673	1752.4 ± 202.8	1528.6 ± 227.1	0.077	1833.4 ± 302.8	1928.6 ± 257.4	0.332
Daily PA									
3-day physical activity (counts/day)	156.2 ± 36.7	149.9 ± 36.3	0.914	235.3 ± 45.1	221.0 ± 67.1	0.427	250.9 ± 39.1	257.9 ± 45.2	0.923
Pubertal status									
Tanner stage (pubic hair)	1.62 ± 0.52	1.80 ± 0.63	0.937	3.00 ± 0.21	2.57 ± 0.46	0.635	3.33 ± 0.22	3.50 ± 0.14	0.783
Kinanthropometric									
Height (m)	1.49 ± 0.07	1.47 ± 0.09	0.235	1.62 ± 0.07	1.60 ± 0.13	0.653	1.66 ± 0.08	1.63 ± 0.12	0.493
Weight (kg)	62.4 ± 11.1	60.5 ± 11.8	0.815	76.6 ± 14.0	71.9 ± 18.4	0.598	76.8 ± 16.0	75.5 ± 21.0	0.903
Waist circumference (cm)	94.0 ± 9.38	92.0 ± 8.06	0.538	88.4 ± 8.45	87.5 ± 11.9	0.874	90.5 ± 16.6	94.3 ± 12.5	0.687
Ponderal status									
Obese (%) #*	100	100	1.000	15.5	30.0	0.768	0.0	20.0	0.648
Metabolic Syndrome Index									
At risk (%)*	62.5	50.0	0.894	0.0	25.0	0.231	0.0	43.0	0.052

* chi-squared test; # ≥ 97th percentile.

Table III
Mean and standard deviation of metabolic parameters at the baseline, third year, and detraining evaluations in obese boys

		Intervention time		*Detraining time*						
		Baseline (a)	*3-year (b)*	*6 months (c)*	*Kruskal-Wallis*		*Differences with detraining evaluations*			
	Group	*Mean ± SD*	*Mean ± SD*	*Mean ± SD*	*H*	*p*	*a vs c*		*b vs c*	
TC (mg/dl)	E	171.9 ± 16.7	150.4 ± 23.2	127.3 ± 2.08	9.509	0.009	**0.014**	-3.75	**0.025**	-1.40
	E+D	161.7 ± 24.5	153.1 ± 24.3	142.8 ± 23.6	2.826	0.243	0.143	-0.79	0.283	-0.43
HDL-C (mg/dl)	E	53.2 ± 6.84	55.8 ± 6.91	54.7 ± 7.23	0.547	0.761	0.759	0.21	0.365	-0.16
	E+D	44.2 ± 11.6	53.6 ± 20.2	55.8 ± 21.8	1.257	0.533	0.416	0.66	0.775	0.10
LDL-C (mg/dl)	E	106.1 ± 17.8	82.6 ± 17.7	59.3 ± 10.7	8.459	0.015	**0.014**	-3.19	0.101	-1.59
	E+D	101.3 ± 16.4	81.0 ± 18.2	67.8 ± 12.8	11.742	0.003	**0.004**	-2.28	0.100	-0.84
TG (mg/dl)	E	62.9 ± 30.9	59.6 ± 16.1	67.0 ± 34.8	0.041	0.980	0.683	0.12	0.881	0.27
	E+D	81.1 ± 57.2	92.4 ± 50.6	95.5 ± 78.3	0.527	0.768	0.914	0.21	0.721	0.05
LDL-C/HDL-C	E	2.00 ± 0.29	1.48 ± 0.23	1.11 ± 0.30	11.411	0.003	**0.014**	-3.02	0.101	-1.38
	E+D	2.42 ± 0.66	1.73 ± 0.82	1.38 ± 0.62	6.835	0.033	**0.017**	-1.62	0.352	-0.48
TC/HDL-C	E	3.25 ± 0.28	2.69 ± 0.17	2.36 ± 0.33	10.953	0.004	**0.014**	-0.46	0.297	-1.26
	E+D	3.83 ± 0.97	3.16 ± 1.17	2.86 ± 1.12	4.595	0.101	0.065	-0.93	0.391	-0.26
Insulin (μU/ml)	E	12.7 ± 8.51	13.8 ± 3.70	13.6 ± 4.20	1.926	0.382	0.307	0.28	0.655	0.20
	E+D	9.73 ± 2.41	17.8 ± 6.76	19.9 ± 12.9	7.318	0.026	**0.034**	1.10	1.000	0.20
Glucose (mmol/l)	E	91.4 ± 5.95	78.8 ± 3.35	88.7 ± 2.31	10.057	0.007	0.469	-0.60	**0.024**	3.44
	E+D	87.6 ± 5.50	88.4 ± 8.06	89.7 ± 5.46	0.795	0.672	0.435	0.38	0.424	0.19
HOMA-IR	E	2.90 ± 2.17	2.68 ± 0.78	3.17 ± 0.97	1.434	0.488	0.307	0.16	0.456	0.56
	E+D	2.08 ± 0.57	3.91 ± 1.64	4.51 ± 3.28	5.969	0.050	**0.047**	1.03	1.000	0.23
SBP (mm Hg)	E	110.0 ± 7.86	110.5 ± 10.9	110.7 ± 3.05	0.054	0.973	0.838	0.11	1.000	0.02
	E+D	117.2 ± 14.4	113.0 ± 9.63	113.5 ± 10.0	0.257	0.879	0.786	-0.29	0.838	0.05
DBP (mm Hg)	E	64.1 ± 6.45	63.5 ± 6.66	63.3 ± 4.93	0.053	0.974	0.836	-0.13	0.858	-0.03
	E+D	67.9 ± 8.48	60.1 ± 4.88	67.0 ± 3.26	4.543	0.103	0.828	-0.13	0.836	0.59

Table IV
Differences between groups for the changes in metabolic parameters

	E *Mean ± SD*	*E+D* *Mean ± SD*	*Mann-Whitney U*		
			U	*p*	*ES*
ΔTC (mg/dl)					
Baseline - detraining	-46.0 ± 17.1	-22.3 ± 13.3	2.00	0.058	-1.57
Year 3 - detraining	-11.3 ± 7.57	-11.5 ± 9.63	8.00	0.796	0.02
ΔHDL-C (mg/dl)					
Baseline - detraining	3.00 ± 7.21	10.0 ± 11.6	5.50	0.361	-0.71
Year 3 - detraining	2.33 ± 6.81	2.33 ± 4.63	9.00	1.000	0.00
ΔLDL-C (mg/dl)					
Baseline - detraining	-47.3 ± 15.6	-35.8 ± 12.6	5.50	0.361	-0.82
Year 3 - detraining	-14.7 ± 4.51	-13.8 ± 11.7	7.50	0.697	-0.10
ΔTG (mg/dl)					
Baseline - detraining	-8.00 ± 16.8	16.8 ± 29.9	5.00	0.302	-0.99
Year 3 - detraining	6.00 ± 23.8	-0.17 ± 35.8	8.00	0.796	0.20
ΔLDL-C/HDL-C					
Baseline - detraining	-0.95 ± 0.25	-1.02 ± 0.39	7.50	0.697	0.21
Year 3 - detraining	-0.31 ± 0.17	-0.39 ± 0.33	9.00	1.000	0.29
ΔTC/HDL					
Baseline - detraining	-0.99 ± 0.31	-0.92 ± 0.47	6.00	0.439	-0.17
Year 3 - detraining	-0.30 ± 0.29	-0.38 ± 0.33	9.00	1.000	0.26
ΔInsulin (μU/ml)					
Baseline - detraining	3.81 ± 5.55	6.08 ± 5.41	5.00	0.456	-0.41
Year 3 - detraining	-0.57 ± 1.92	2.41 ± 7.01	7.00	0.606	-0.55
ΔGlucose (mmol/l)					
Baseline - detraining	-4.00 ± 3.60	5.17 ± 4.49	2.00	0.060	-2.22
Year 3 - detraining	8.67 ± 2.52	4.00 ± 3.03	1.50	0.051	1.66
ΔHOMA-IR					
Baseline - detraining	0.71 ± 1.34	1.44 ± 1.30	5.00	0.456	-0.82
Year 3 - detraining	0.79 ± 1.87	0.71 ± 1.34	8.00	0.796	0.05
ΔSBP					
Baseline - detraining	-0.33 ± 6.81	-1.23 ± 4.60	7.50	0.697	0.15
Year 3 - detraining	-0.11 ± 5.36	-0.23 ± 5.60	8.00	0.796	0.02
ΔDBP					
Baseline - detraining	-0.21 ± 5.81	-0.23 ± 5.60	9.00	1.000	0.00
Year 3 - detraining	-0.10 ± 5.36	0.43 ± 6.23	8.00	0.796	-0,09

based on exercise and/or diet on metabolic parameters in obese boys. To the best of our knowledge, the present work is the first of this type to study the detraining that follows a longitudinal intervention program in obese boys. The results indicated that the changes achieved in the metabolic parameters after both longitudinal interventions were maintained following the detraining, and even improved, especially in the lipid profile. Similarly, there was a reduction in the prevalence of obese and metabolic syndrome in the subjects after the intervention period, and this also was maintained after detraining in the E group. The results thus suggest that long-term exercise (with or without diet) generates lasting, healthy habits that might prevent and alleviate metabolic syndrome. However, due to the number of subjects (n = 18), the present study it can be considered only a preliminary investigation.

Intra-group differences

In the lipid profile of the obese subjects, various indicators improved after detraining in both interventions. There stand out the reductions relative to the baseline in LDL-C in the E group (ES = -3.19) and in the E+D group (ES = -2.28), and in the LDL-C/HDL-C ratio in the E group (ES = -3.02) and in the E+D group (ES = -1.62). This shows the importance of aerobic exercise[24] and combined aerobic-plus-diet[25] programs in generating benefits in LDL-C in an obese pædiatric population. In this sense, a study by Shalitin et al.[7] confirmed that even short-term exercise and exercise-plus-diet programs led to decreases in LDL-C levels that were preserved at the end of 1 year, despite weight gain. The indication is thus that both short- and long-term exercise interventions may provide benefits in the prevention of part of obesity-related morbidity. The only parameter that was reduced from the three-year period to detraining was TC, particularly marked in the E group (ES = -1.40). Contrary to these findings, other studies have reported increases in TG after physical exercise and/or diet programs (detraining) with TC remaining unchanged (3-10 months)[6,7]. The levels of the other lipid parameters remained close to the initial values, especially in the case of the combined program of diet plus physical exercise, as has also been reported in other studies in the literature[7]. These results demonstrate that short/medium-term interventions are insufficiently effective at maintaining beneficial effects in the long term (after termination of the programs). Therefore, intervention programs for obese boys probably require either ongoing support or longer durations for the benefits to be maintained over time. Such an approach would preserve the beneficial effect of the program and prevent the morbidity associated with obesity by fostering the generation of healthy habits in later life.

Regarding the insulin resistance markers, the detraining appeared to have a negative influence on these in both interventions. Thus, there was a significant increase in glucose levels from the three-year period to detraining with a large ES (ES = 3.44). Confirming this result, another study observed an increase in this parameter after three months detraining (ES = 0.23), and especially emphasized the importance of exercise in controlling this parameter[6]. Similarly, after the combined (E+D group) intervention there was an increase in insulin in the detraining (ES = 1.10), and consequently also in HOMA-IR (ES = 1.03). This could reflect the importance of diet in the control of these parameters, as indicated by the results of previous studies[7].

Finally, with respect to the metabolic syndrome, in the E group the prevalence in obese children decreased ($\chi^2 = 6.571$, $p < 0.001$). In this same line, another study has documented dramatic reductions in the prevalence of metabolic syndrome in obese children in an 8-week period of exercise training targeted at the fat (max) zone[26]. There have been no other studies that analyse detraining and metabolic syndrome, and the present study has shown that, after detraining, no subject surpassed the index established as representing metabolic risk (≥ 4.2)[21]. It therefore seems that physical exercise is consolidated as a key factor in the prevention of cardiovascular disease risk in obese children, and that this is so even after controlled termination of the program[27,28]. However, it is likely that boys included in the study were those with a high level of motivation, so they might continue following the program (exercise and diet) after finishing the intervention.

Inter-group differences

No differences were observed in any of the parameters evaluated after the detraining period, suggesting that the two interventions generate similar long-term changes in these parameters. These results appear to indicate that the long-term benefits of the two interventions are equivalent, as also they were during the detraining. Thus, it would seem that in the long term there is no requirement for any comprehensive medical control (dietician and/or endocrinologist) of the obese subject's diet, although healthy habits together with moderate-vigorous physical exercise could generate benefits in this regard.

Conclusions

In conclusion, the detraining from a long-term program of physical exercise alone or in combination with a low calorie diet seemed to have no negative effects on metabolic parameters (parameters not worsen), thus helping resolve metabolic syndrome or prevent its emergence. Indeed, the subjects in the E group classified with metabolic syndrome ceased to be so after the physical exercise program, and this status

was maintained after detraining. These results suggest that long-term programs of this kind promote benefits and healthy habits in obese boys that are maintained over time. In particular, such non-invasive intervention could be applied for a variety of motives, for instance, when subject are unwilling or unable to undergo clinical treatment or intervention. The results also highlight the importance of exercise itself in maintaining the benefits that have been obtained in the metabolic parameters of obese boys.

Limitations

A number of limitations of this study need to be borne in mind. First, there was a lack of initial randomization of the groups. Nonetheless, the homogeneity of the groups was supported by the absence of initial differences in any of the variables (table I). Second, while the absence of a no-intervention control group was clearly far from ideal, it was unavoidable since it proved impossible to find a group of obese children who were not partaking in any type of intervention to improve their condition. Third, the number of subjects in the study was small (n = 18), although the study's longitudinal character could make this limitation of only relative importance. And fourth, we did not record the type of activity or sport performed by each subject during the course of detraining. Such information could have been useful in interpreting the results.

References

1. Han JC, Lawlor DA, Kimm S. Childhood obesity. *Lancet* 2010; 375 (9727): 1737-48.
2. Eisenmann JC, Wickel EE, Welk GJ, Blair SN. Relationship between adolescent fitness and fatness and cardiovascular disease risk factors in adulthood: the Aerobics Center Longitudinal Study (ACLS). *Am Heart J* 2005; 149 (1): 46-53.
3. Ratner R, Durán S, Garrido MJ, Balmaceda S. Impacto de una intervención en alimentación y actividad física sobre la prevalencia de obesidad en escolares. *Nutr Hosp* 2013; 28 (5): 1508-14.
4. Rowlands AV. Accelerometer assessment of physical activity in children: an update. *Pediatr Exerc Sci* 2007; 19 (3): 252-66.
5. Waters E, de Silva-Sanigorski A, Hall BJ, Brown T, Campbell KJ, Gao Y et al. Interventions for preventing obesity in children. *Cochrane Database Syst Rev* 2011 (12): CD001871.
6. Chang C, Liu W, Zhao X, Li S, Yu C. Effect of supervised exercise intervention on metabolic risk factors and physical fitness in Chinese obese children in early puberty. *Obes Rev* 2008; 9 (Supl. 1): 135-41.
7. Shalitin S, Ashkenazi-Hoffnung L, Yackobovitch-Gavan M, Nagelberg N, Karni Y, Hershkovitz E et al. Effects of a twelve-week randomized intervention of exercise and/or diet on weight loss and weight maintenance, and other metabolic parameters in obese preadolescent children. *Horm Res Paediatr* 2009; 72 (5): 287-301.
8. Woo J, Shin KO, Yoo JH, Park S, Kang S. The effects of detraining on blood adipokines and antioxidant enzyme in Korean overweight children. *Eur J Pediatr* 2012; 171 (2): 235-43.
9. Mujika I, Padilla S. Detraining: loss of training-induced physiological and performance adaptations. Part I: short term insufficient training stimulus. *Sports Med* 2000; 30 (2): 79-87.
10. Ferguson M, Gutin B, Le N, Karp W, Litaker M, Humphries M et al. Effects of exercise training and its cessation on components of the insulin resistance syndrome in obese children. *Int J Pediatr Obes* 1999; 23 (8): 889-95.
11. Hernández M, Castellet J, Narvaiza JL, Rincon JM, Ruiz I, Sánchez E et al. Curvas y tablas de crecimiento: Garsi; 1988.
12. Sallis JF, Buono MJ, Roby JJ, Carlson D, Nelson JA. The Caltrac accelerometer as a physical activity monitor for school-age children. *Med Sci Sports Exerc* 1990; 22 (5): 698-703.
13. Maliszewski AF, Freedson PS, Ebbeling CJ, Crussemeyer J, Kastango KB. Validity of the Caltrac accelerometer in estimating energy expenditure and activity in children and adults. *Pediatr Exerc Sci* 1991; 3: 141-51.
14. Raudsepp L, Päll P. The relationship between fundamental motor skills and outside-school physical activity of elementary school children. *Pediatr Exerc Sci* 2006; 18 (4): 426-35.
15. Kumahara H, Schutz Y, Ayabe M, Yoshioka M, Yoshitake Y, Shindo M et al. The use of uniaxial accelerometry for the assessment of physical-activity-related energy expenditure: a validation study against whole-body indirect calorimetry. *Br J Nutr* 2004; 91 (2): 235-43.
16. Golan M, Crow S. Targeting Parents Exclusively in the Treatment of Childhood Obesity: Long-Term Results. *Obesity* 2004; 12 (2): 357-61.
17. Mataix, J, Collado F, Dominguez I, Sumalla S, Sierra J, Battino M et al. NutrIber. Nutrición Iberoamericana. Barcelona (España): Fundación Universitaria Iberoamericana; 2006.
18. Tanner JM, Whitehouse RH, Takaishi M. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. I. *Arch Dis Child* 1966; 41 (219): 454-71.
19. Norton K, Whittingham N, Carter L, Kerr D, Gore C, Marfell-Jones M. Measurement techniques in anthropometry. Anthropometrica 1996; 1: 25-75.
20. Matthews D, Hosker J, Rudenski A, Naylor B, Treacher D, Turner R. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; 28 (7): 412-9.
21. Martínez-Vizcaíno V, Martínez MS, Aguilar FS, Martínez SS, Gutiérrez RF, López MS et al. Validity of a Single-Factor Model Underlying the Metabolic Syndrome in Children A confirmatory factor analysis. *Diabetes Care* 2010 ;33 (6): 1370-2.
22. Nevill AM, Atkinson G, Hughes MD, Cooper SM. Statistical methods for analysing discrete and categorical data recorded in performance analysis. *J Sports Sci* 2002; 20 (10): 829-44.
23. Cohen J. Statistical power analysis for the behavioral sciences: Lawrence Erlbaum; 1988.
24. Escalante Y, Saavedra JM, García-Hermoso A, Domínguez AM. Improvement of the lipid profile with exercise in obese children: A systematic review. *Prev Med* 2012; 54 (5): 293-301.
25. Watts K, Jones TW, Davis EA, Green D. Exercise training in obese children and adolescents: current concepts. *Sports Med* 2005; 35 (5): 375-92.
26. Ben Ounis O, Elloumi M, Makni E, Zouhal H, Amri M, Tabka Z et al. Exercise improves the ApoB/ApoA I ratio, a marker of the metabolic syndrome in obese children. *Acta Paediatr* 2010; 99 (11): 1679-85.
27. Guinhouya BC, Samouda H, Zitouni D, Vilhelm C, Hubert H. Evidence of the influence of physical activity on the metabolic syndrome and/or on insulin resistance in pediatric populations: a systematic review. *Int J Pediatr Obes* 2011; 6 (5 6): 361-88.
28. Vásquez F, Díaz E, Lera L, Meza J, Salas I, Rojas P et al. Efecto residual del ejercicio de fuerza muscular en la prevención secundaria de la obesidad infantil. *Nutr Hosp* 2013; 28 (2): 333-9.