Exercício, Obesidade e Síndrome Metabólica

Profª. Drª. Ana Paula Trussardi Fayh

Tratamento da Obesidade

- O tratamento dietético é mais bem-sucedido quando aliado a aumento no gasto energético e a um programa de modificação comportamental(D).
- O sucesso de qualquer dieta depende de um balanço energético negativo(D).
- Para o sucesso do tratamento dietético, deve-se manter mudanças na alimentação por toda a vida(D). Dietas muito restritivas, artificiais e rígidas não são sustentáveis(B). Um planejamento alimentar mais flexível, que objetive reeducação, geralmente obtém mais sucesso(B).

Tratamento da Obesidade

- O método, a velocidade de perda de peso, o ajuste fisiológico e a habilidade de manter as mudanças comportamentais de dieta e atividade física é que determinarão o sucesso, em longo prazo, de qualquer programa de emagrecimento(D).

- Qualquer dieta prescrita para reduzir peso tem de considerar, além da quantidade de calorias, as preferências alimentares do paciente, o aspecto financeiro, o estilo de vida e o requerimento energético para a manutenção da saúde.

FAZENDO DIETA...

Números *Mágicos* no Tratamento da Obesidade

10% de redução no peso inicial

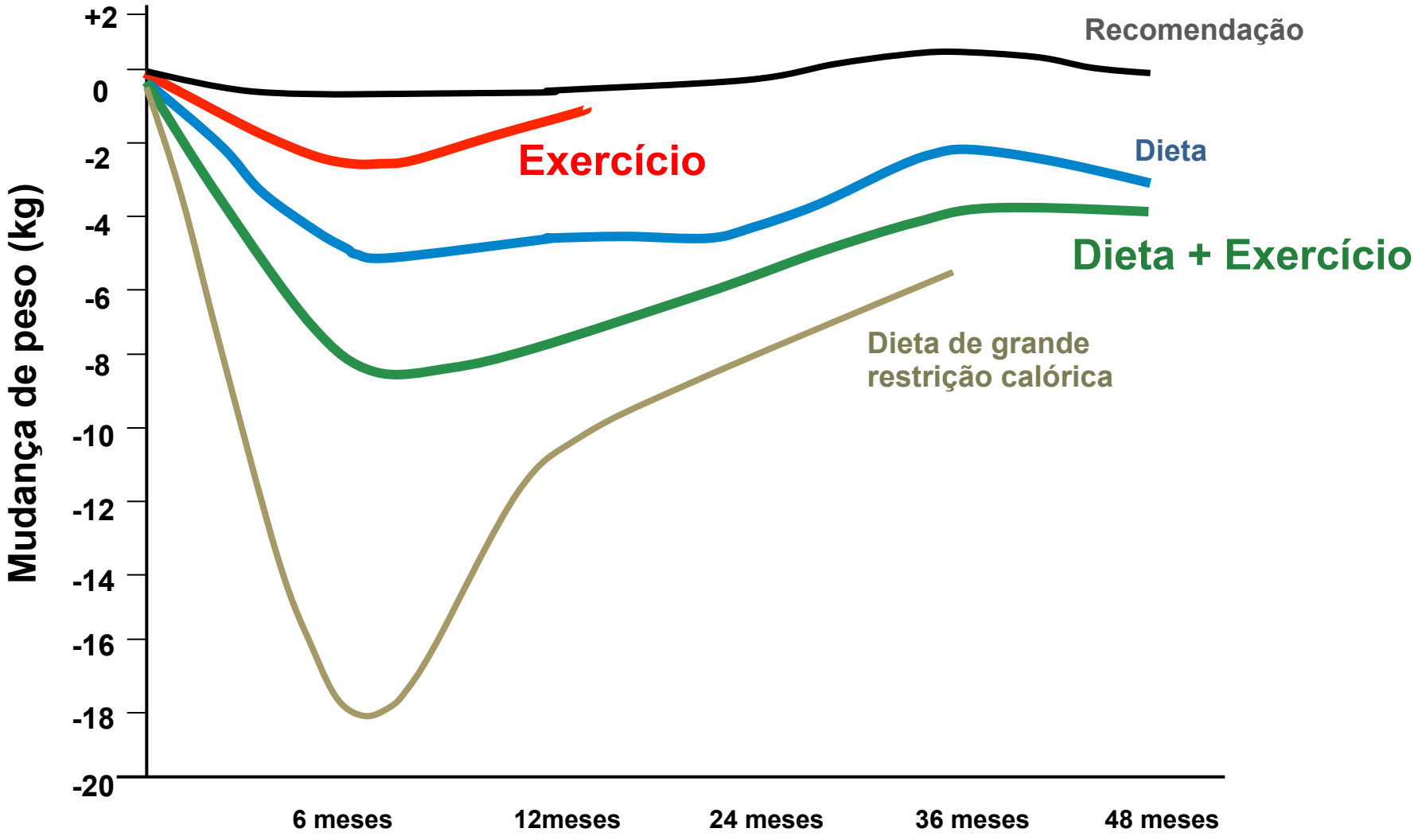
30% dos pacientes recuperam >5% do peso inicial

3-5 anos: peso corporal em valores iniciais

Wing RR, Hill JO. Annu Ver Nutr 2001, 21: 323-41.

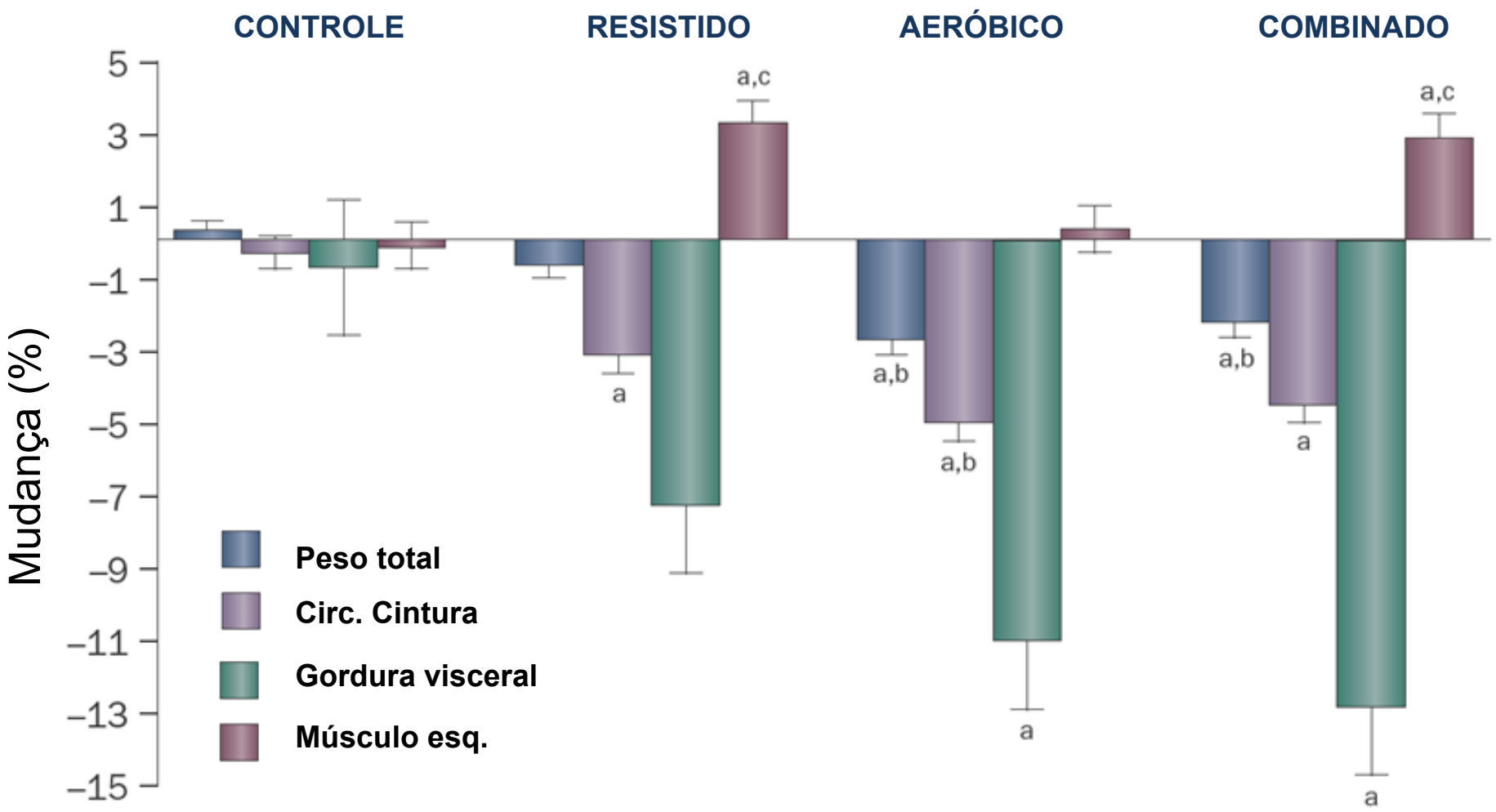
Weiss et al. Am J Prev Med 2007, 33: 34-40.

Exercício ou Dieta para Redução de Peso



Franz et al. J Am Diet Assoc 2007; 1755-1767

Tipos de Treinamento nas Variáveis de Interesse

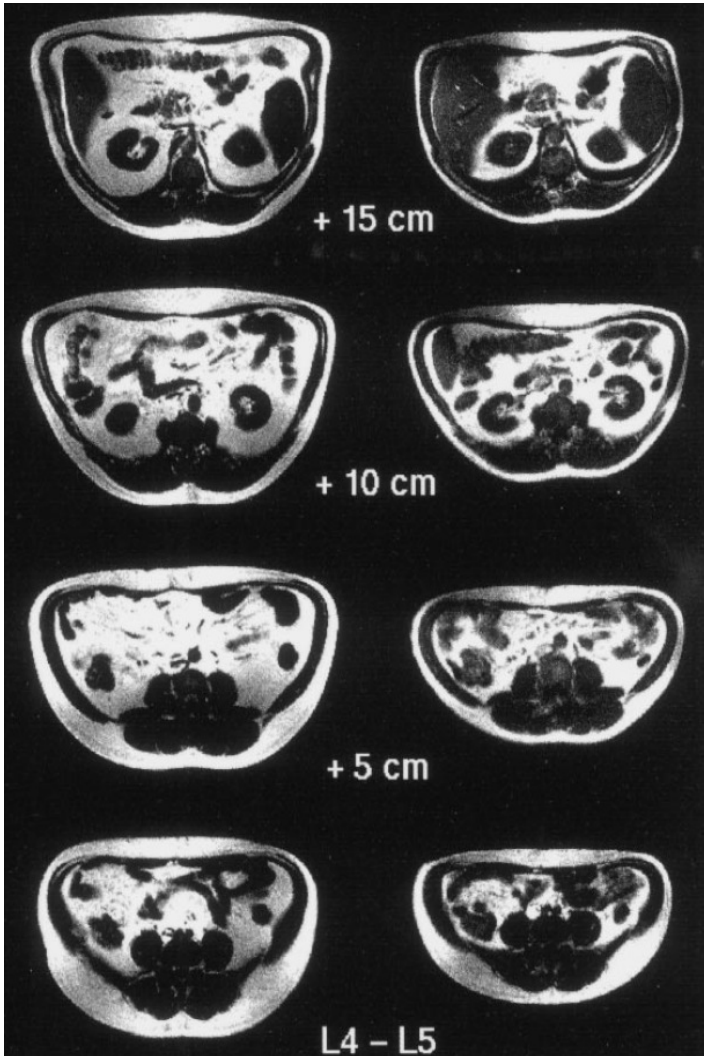


Davidson et al. JAMA Int Medicine 2009, 169(2): 122-131

Exercício de Força, Aeróbico ou Dieta?

33 homens obesos

Somente Dieta	Dieta + Aeróbico	Dieta + Força
↓ 1000 kcal/dia	5x/sem	3x/sem
	37' / sessão	9 exercícios
	77% $FC_{máx}$	8-12 RMs

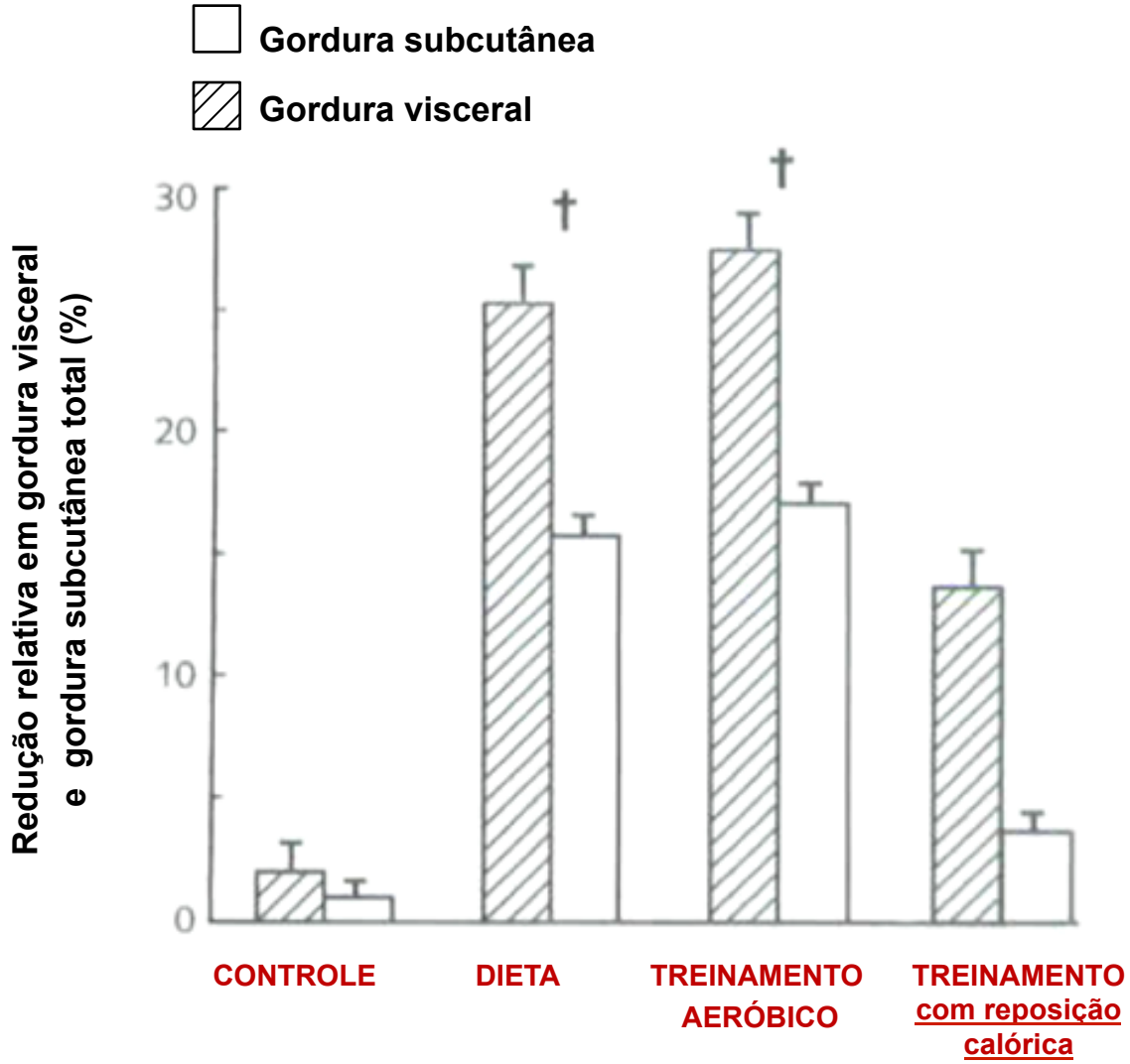


Ross R et al. J Appl Physiol 1996, 81: 2445-55

Sem diferenças entre os grupos

↓ Absoluta de gordura	~ 11,5 kg
↓ Gordura subcutânea	~ 24 %
↓ Gordura visceral	~ 40 %

Treinamento Aeróbico e Redução de Gordura



Ross R et al. Ann Intern Med 2000, 133: 92-103

TABLE 1
Descriptive characteristics and inflammatory markers at baseline and changes (Δ) after 6 and 18 mo by treatment group[1]

	Dietary weight-loss group	Exercise group	Dietary weight loss + exercise group	Control group
n				
Baseline	71	67	64	70
6 mo	63	58	58	63
18 mo	53	53	53	60
Age (y)	68 ± 5[2]	69 ± 6	68 ± 7	69 ± 6
Women (%)	74	74	74	65
Nonwhite (%)	27	20	21	19
Body weight (kg)				
Baseline	95.6 ± 15.2	92.4 ± 14.6	91.8 ± 17.4	95.7 ± 18.8
Δ 6 mo	−10.6 ± 14.0	−3.0 ± 14.6	−10.2 ± 10.5	−0.15 ± 6.7
Δ18 mo	−12.8 ± 19.2	−4.1 ± 11.1	−8.2 ± 13.3	−2.3 ± 11.6
BMI (kg/m^2)				
Baseline	34.4 ± 4.9	34.6 ± 5.8	33.9 ± 5.6	34.5 ± 5.3
Δ 6 mo	−1.78 ± 3.0	−1.16 ± 3.4	−1.62 ± 1.75	−0.52 ± 2.5
Δ 18 mo	−2.0 ± 3.1	−1.2 ± 3.1	−1.48 ± 2.2	−0.96 ± 3.7
CRP (μg/mL)				
Baseline	6.0 ± 6.5	6.8 ± 7.8	6.5 ± 7.9	5.9 ± 6.0
Δ 6 mo	−0.11 ± 0.72	−0.07 ± 0.42	−0.11 ± 0.52	0.08 ± 0.63
Δ 18 mo	−0.13 ± 0.53	−0.02 ± 0.47	−0.18 ± 0.54	0.35 ± 1.9
IL-6 (pg/mL)				
Baseline	4.7 ± 3.4	4.4 ± 3.1	4.9 ± 3.0	4.7 ± 3.2
Δ 6 mo	−0.51 ± 2.1	0.15 ± 1.8	−0.35 ± 2.15	0.19 ± 2.8
Δ 18 mo	−0.71 ± 2.4	0.02 ± 2.4	−0.35 ± 1.8	0.27 ± 2.8
IL-6sR (pg/mL)				
Baseline	35 197 ± 8911	34 581 ± 9596	35 156 ± 11,075	38 040 ± 10,764
Δ 6 mo	−838 ± 3645	−856 ± 5135	−759 ± 3235	−1247 ± 5159
Δ 18 mo	−1522 ± 8344	94 ± 4399	−746 ± 4166	−739 ± 4910
TNF-α (pg/mL)				
Baseline	2.5 ± 1.8	3.4 ± 4.8	3.4 ± 6.4	3.8 ± 7.5
Δ 6 mo	−0.23 ± 1.8	−0.69 ± 5.8	−0.46 ± 3.7	−0.74 ± 3.7
Δ 18 mo	0.64 ± 5.9	0.28 ± 6.3	−0.72 ± 4.6	−0.77 ± 3.7
sTNFR1 (pg/mL)				
Baseline	1409 ± 470	1433 ± 404	1395 ± 397	1464 ± 421
Δ 6 mo	−92 ± 290	−38 ± 224	−80 ± 226	−10 ± 291
Δ 18 mo	−34 ± 362	25 ± 252	−3 ± 241	62 ± 312
sTNFR2 (pg/mL)				
Baseline	2674 ± 842	2760 ± 807	2656 ± 792	2850 ± 1127
Δ 6 mo	−53 ± 547	−148 ± 409	−78 ± 427	−59 ± 915
Δ 18 mo	38 ± 665	24 ± 514	−61 ± 460	38 ± 561

[1] CRP, C-reactive protein; IL-6, interleukin 6; IL-6sR, soluble IL-6 receptor; TNF-α, tumor necrosis factor α; sTNFR1 and sTNFR2, soluble TNF-α receptors 1 and 2, respectively. There were no significant differences between treatment groups at baseline for any variable.

[2] $\bar{x}$ ± SD (all such values).

Sobrepeso
Obeso
18 meses

Am J Clin Nutr 2004;79:544–51.

Déficit Calórico pelo Exercício Físico

52 homens obesos | IMC: 31,3 ± 2 kg/m², Circunferência cintura: 110 ± 6 cm

- **Grupo controle (C)**
- **Grupo Dieta (DWL)**
- **Grupo Exercício sem compensação (EWL)**
- **Grupo Exercício com compensação (EWW)**

- **Dieta: - 700 kcal/dia (55 a 60% CHO, 15 a 20% PRO e 20 a 25% LIP).**
- **Exercício: caminhada ou corrida em esteira, diária, com duração suficiente para um gasto de 700 kcal. Duração: 12 semanas.**

Ross R et al. Ann Intern Med 2000, 133: 92-103.

Treinamento Aeróbico e SUPORTE calórico

Efeitos no peso corporal:

- Grupo controle (C): **+ 0,1 kg**
- Grupo Dieta (DWL): **- 7,4 kg**
- Grupo Exercício sem Compensação (EWL): **- 7,5 kg**
- Grupo Exercício com Compensação (EWW): **- 0,5 kg**

Ross R et al. Ann Intern Med 2000, 133: 92-103.

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Effect of Diet With and Without Exercise Training on Markers of Inflammation and Fat Distribution in Overweight Women

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Conclusão: Parece que a perda de peso é o mais importante para promover efeitos benéficos do que o exercício.

Table 2

Body composition and markers of inflammation with weight loss by intervention group

	Diet (*n* = 29)		Aerobic (*n* = 43)		Resistance (*n* = 54)		G	T	G × T
	Baseline	**Weight reduced**	**Baseline**	**Weight reduced**	**Baseline**	**Weight reduced**	***P***	***P***	***P***
Body weight (kg)	78 ± 8^{a}	66 ± 7^{b}	77 ± 7^{a}	65 ± 6^{b}	78 ± 8^{a}	66 ± 7^{b}	0.628	0.001	0.314
BMI ($kg.m^2$)	28 ± 1^{a}	24 ± 1^{b}	28 ± 1^{a}	24 ± 1^{b}	28 ± 1^{a}	24 ± 1^{b}	0.853	**0.001**	0.272
Fat mass (kg)	33 ± 6^{a}	22 ± 5^{b}	34 ± 5^{a}	22 ± 5^{b}	33 ± 5^{a}	22 ± 4^{b}	0.967	**0.001**	0.312
Body fat (%)	44 ± 4^{a}	35 ± 5^{b}	46 ± 4^{a}	35 ± 5^{b}	45 ± 4^{a}	34 ± 4^{b}	0.416	**0.001**	0.062
Lean mass (kg)	41 ± 4^{a}	41 ± 4^{a}	40 ± 4^{a}	40 ± 4^{a}	41 ± 4^{a}	42 ± 4^{b}	0.079	0.568	**0.001**
IAAT (cm^2)	93 ± 35^{a}	58 ± 26^{b}	79 ± 31^{a}	48 ± 21^{b}	77 ± 31^{a}	53 ± 31^{b}	0.227	**0.001**	0.434
SSAAT (cm^2)	183 ± 49^{a}	127 ± 54^{b}	205 ± 55^{a}	140 ± 55^{b}	189 ± 45^{a}	140 ± 40^{b}	0.348	**0.001**	0.220
DSAAT (cm^2)	137 ± 55^{a}	86 ± 38^{b}	149 ± 52^{a}	77 ± 33^{b}	135 ± 52^{a}	93 ± 38^{b}	0.975	**0.001**	0.104
TNF-α (pg/ml)	1.15 ± 1.71^{a}	0.81 ± 0.50^{a}	1.11 ± 1.00^{a}	0.94 ± 0.74^{a}	0.98 ± 0.73^{a}	0.97 ± 0.80^{a}	0.785	**0.001**	0.226
sTNF-R1 (ng/ml)	1.86 ± 0.34^{a}	1.83 ± 0.26^{a}	1.80 ± 0.38^{a}	1.73 ± 0.35^{a}	1.79 ± 0.40^{a}	1.73 ± 0.39^{a}	0.404	**0.021**	0.605
sTNF-R2 (ng/ml)	3.94 ± 0.77^{a}	3.83 ± 0.66^{a}	4.15 ± 1.11^{a}	3.82 ± 0.91^{a}	3.95 ± 1.21^{a}	3.74 ± 1.06^{a}	0.644	**0.003**	0.505
IL-6 (pg/ml)	$1.71 \pm 1.17^{a,b}$	$1.27 \pm 0.65^{a,b}$	$1.82 \pm 1.27^{a,b}$	$1.38 \pm 0.59^{a,b}$	1.85 ± 1.46^{a}	1.34 ± 0.86^{b}	0.724	**0.001**	0.539
CRP (mg/l)	$1.93 \pm 1.64^{a,b}$	1.13 ± 1.23^{c}	$1.91 \pm 1.97^{a,b,c}$	$1.59 \pm 1.67^{a,b,c}$	2.20 ± 2.05^{a}	$1.61 \pm 1.94^{b,c}$	0.653	**0.001**	0.407

All data are presented as means ± s.d. Far-right columns indicate results of repeated-measures ANOVA for main effects of group (G), time (T), and the group-by-time interaction (G × T). Within a row, results of *post-hoc* analyses among the three groups are provided with superscripts; means with different superscripts are significantly different ($P < 0.05$). The boldface values are indicative of G, T, or G × T effects that were statistically significant.

CRP, C-reactive protein; DSAAT, deep subcutaneous abdominal adipose tissue; G × T, group × time interaction; G, intervention group; IAAT, intra-abdominal adipose tissue; IL-6, interleukin-6; SSAAT, superficial subcutaneous abdominal adipose tissue; sTNF-R1, tumor necrosis factor receptor I; sTNF-R2, tumor necrosis factor receptor 2; T, time; TNF-α, tumor necrosis factor-α.

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DOI 10.1007/s00394-012-0450-1

ORIGINAL CONTRIBUTION

Effects of 5 % weight loss through diet or diet plus exercise on cardiovascular parameters of obese: a randomized clinical trial

Ana Paula Trussardi Fayh · André Luiz Lopes · Antônio Marcos Vargas da Silva · Álvaro Reischak-Oliveira · Rogério Friedman

Fig. 1 Flow diagram of patient recruitment and randomization

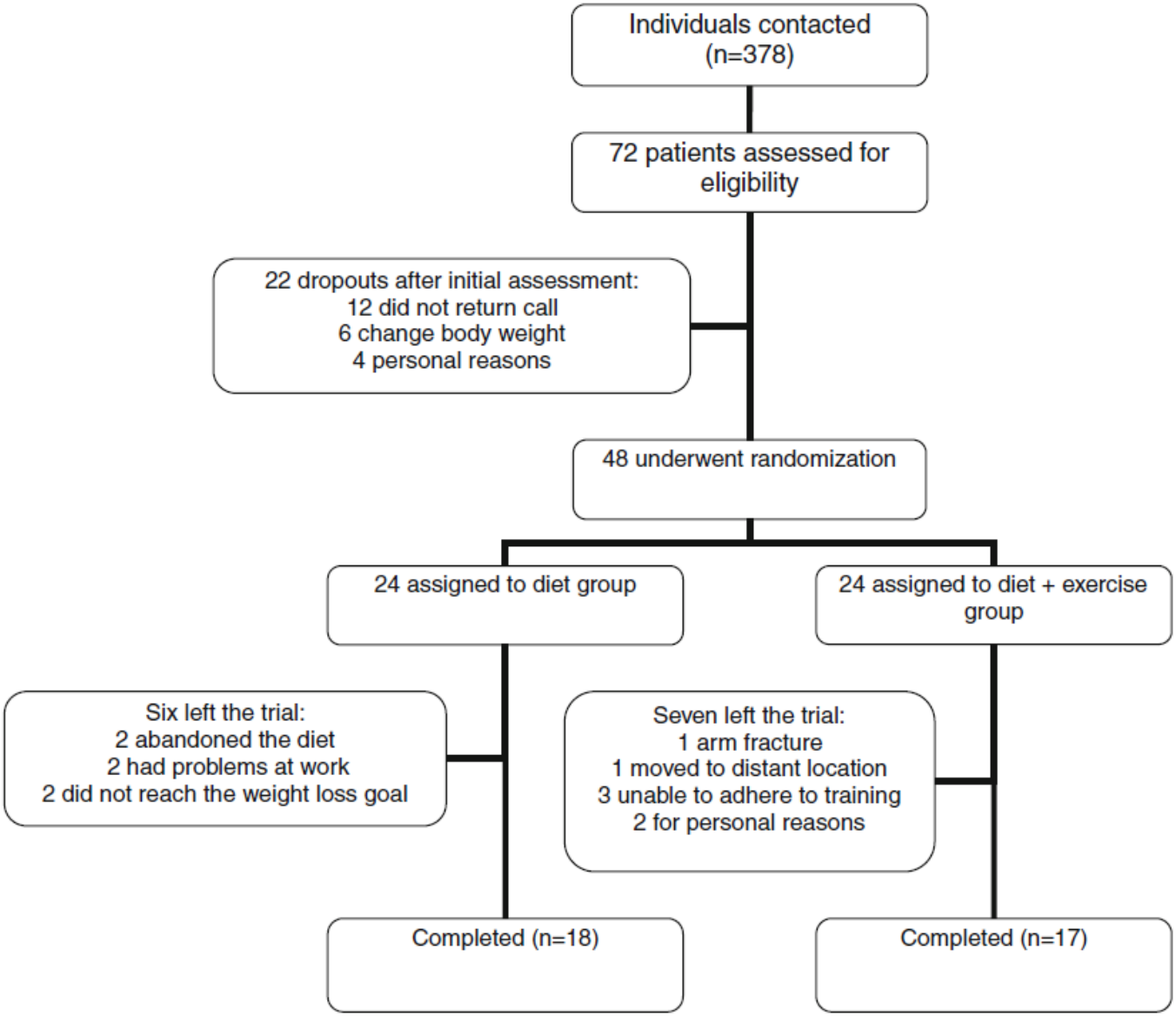

Table 1 Baseline clinical and laboratory characteristics of study group after randomization

Variables	DI ($n = 24$)	DI + EXE ($n = 24$)
Male (*n*/%)	8 (33.3)	8 (33.3)
Age (years)	31.4 ± 5.6	32.3 ± 6.4
Body weight (kg)	95.4 ± 12.1	99.1 ± 12.0
Height (m)	1.65 ± 0.09	1.69 ± 0.07
Body mass index (kg/m^2)	34.8 ± 2.4	34.7 ± 2.2
Obesity grade I (*n*/%)	14 (58.3)	13 (54.17)
Waist circumference (cm)	111.7 ± 7.7	110.8 ± 6.6
Hip circumference (cm)	118.9 ± 8.6	120.9 ± 6.0
Waist-to-hip ratio	0.84 ± 0.09	0.85 ± 0.08
Total cholesterol (mg/dL)	192.4 ± 35.5	182.2 ± 30.3
HDL cholesterol (mg/dL)	47.7 ± 9.8	48.0 ± 12.5
LDL cholesterol (mg/dL)	114.3 ± 28.4	106.4 ± 27.4
Triglycerides (mg/dL)	119 (93–203)	127 (69.5–186)
High-sensitive C-reactive protein (g/dL)	3.8 (2.6–5.8)	4.1 (1.4–7.1)
von Willebrand factor (%)	117.4 ± 34.4	124.6 ± 41.6
Fibrinogen (mg/dL)	388.0 ± 96.3	376.3 ± 91.5
Basal diameter of vessel (mm)	3.23 ± 0.48	3.48 ± 0.53
FMD (%)	10.47 ± 4.90	8.20 ± 5.05
EID-NTG (%)	18.37 ± 6.18	16.16 ± 5.71

Table 2 Anthropometric and biochemical changes with interventions

	DI ($n = 18$)			DI + EXE ($n = 17$)			P^a	P^b	P^c
	Before	After	Change	Before	After	Change			
Body weight (kg)	95.8 ± 13.7	91.5 ± 14.2	−4.31 ± 0.5	98.7 ± 13.0	94.0 ± 13.0	−4.66 ± 0.52	0.00	0.64	0.63
Body mass index (kg/m^2)	34.7 ± 2.4	33.1 ± 2.6	−1.58 ± 0.17	34.7 ± 2.4	33.1 ± 2.1	−1.62 ± 0.17	0.00	0.79	0.79
Waist circumference (cm)	112.0 ± 8.7	108.3 ± 8.7	−3.42 ± 0.44	110.9 ± 7.4	107.0 ± 7.8	−3.92 ± 0.45	0.00	0.76	0.76
Hip circumference (cm)	120.4 ± 8.8	117.1 ± 8.6	−3.31 ± 1.18	120.2 ± 5.1	117.0 ± 4.7	−3.18 ± 2.2	0.00	0.83	0.84
Waist-to-hip ratio	0.83 ± 0.09	0.83 ± 0.09	0.00 ± 0.00	0.86 ± 0.08	0.85 ± 0.07	0.01 ± 0.00	0.06	0.05	0.09
Total cholesterol (mg/dL)	191.4 ± 31.5	175.4 ± 37.1	−15.83 ± 4.75	185.5 ± 31.2	175.3 ± 32.6	−10.47 ± 4.89	0.00	0.40	0.44
HDL cholesterol (mg/dL)	45.5 ± 7.6	42.1 ± 9.3	−3.55 ± 1.60	47.2 ± 11.6	44.6 ± 11.2	−2.47 ± 1.65	0.02	0.75	0.64
LDL cholesterol (mg/dL)	115.4 ± 27.2	109.5 ± 29.1	−5.23 ± 4.13	108.8 ± 29.0	107.8 ± 27.3	−1.67 ± 4.25	0.27	0.43	0.55
Triglycerides (mg/dL)	122 (94–206)	94 (65–177)	−33.8 ± 10.0	142 (83–202)	104 (67–158)	−39.4 ± 10.3	0.00	0.81	0.70
hs-CRP (mg/L)	3.3 (2.4–6.4)	2.8 (1.5–4.8)	−1.35 ± 0.41	3.5 (1.5–5.8)	3.0 (1.1–5.9)	−0.45 ± 0.43	0.01	0.13	0.14

Data are presented in mean ± SD or median (interquartile range)

hs-CRP high-sensitivity C-reactive protein

P^a for intervention with repeated measures general linear model

P^b for intervention x group with repeated measures general linear model

P^c with analysis of covariance adjusted for baseline measures

Table 3 Changes in vascular parameters with interventions

	DI ($n = 18$)			DI + EXE ($n = 17$)			P^a	P^b	P^c
	Before	After	Change	Before	After	Change			
Fibrinogen (mg/dL)	386.2 ± 104.8	380.0 ± 110.2	−5.59 ± 13.23	377.5 ± 90.0	372.4 ± 85.3	−5.79 ± 13.61	0.56	0.95	0.99
Von Willebrand factor (%)	120.8 ± 37.0	103.5 ± 29.2	−17.63 ± 4.77	124.1 ± 39.4	119.4 ± 41.0	−4.27 ± 4.91	0.01	0.10	0.06
Basal diameter of artery (mm)	3.21 ± 0.46	3.15 ± 0.42	−0.07 ± 0.05	3.51 ± 0.63	3.34 ± 0.55	−0.74 ± 0.06	0.07	0.64	0.88
FMD (%)	9.9 ± 3.4	10.1 ± 5.8	0.73 ± 1.11	8.1 ± 3.6	10.7 ± 3.6	2.08 ± 1.15	0.10	0.17	0.41
EID-NTG (%)	18.2 ± 5.5	19.4 ± 6.7	1.7 ± 1.21	15.9 ± 3.9	17.6 ± 4.2	1.06 ± 1.25	0.14	0.83	0.72

Data are presented in mean ± SD

FMD flow-mediated dilation measured by vascular ultrasound

EID-NTG endothelium-independent dilation after the administration of sublingual nitroglycerin

P^a for intervention with repeated measures general linear model

P^b for intervention x group with repeated measures general linear model

P^c with analyses of covariance adjusted for baseline measures

In conclusion, our findings indicate that, in obese adults clinically free from cardiovascular disease, a 5 % reduction in body weight is associated with beneficial changes on total cholesterol, triglycerides and hs-CRP. Biochemical parameters of endothelial function (vWf) also improve after weight loss, but this is not reflected in a change of FMD. Based on these findings, we could confirm that the non-pharmacological treatment of obesity (lifestyle change and diet) is effective in reducing inflammation and blood coagulation parameters and improves some parameters of lipid profile in these patients. At least during the first very few months of treatment, weight loss seems to be the key variable, and physical training added little or no beneficial effect.

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Impact of weight loss with or without exercise on abdominal fat and insulin resistance in obese individuals: a randomised clinical trial

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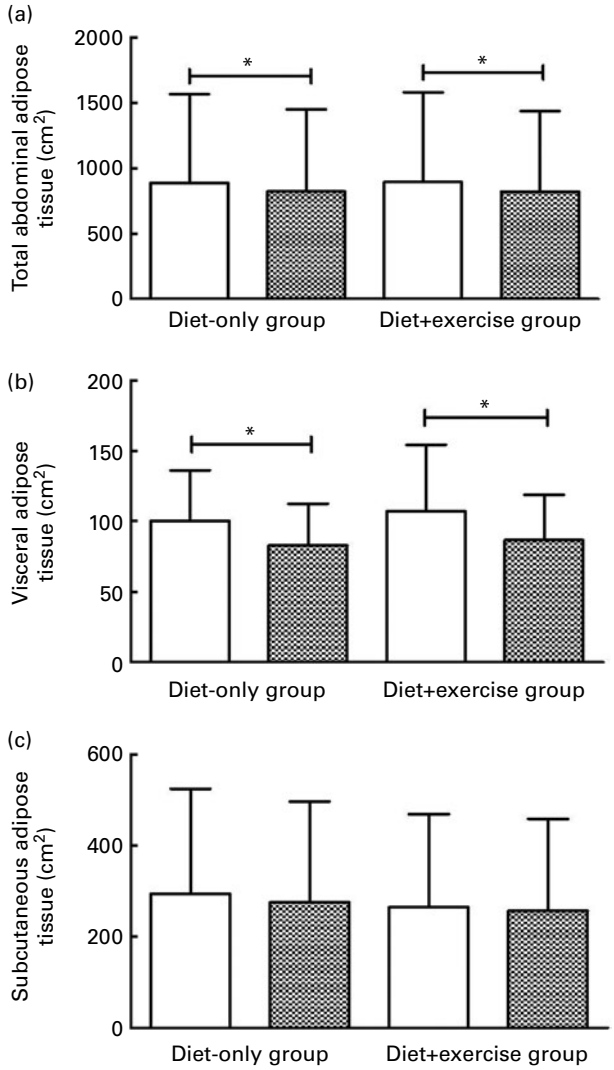

Fig. 2. Effect of interventions on (a) total abdominal adipose tissue, (b) visceral adipose tissue and (c) subcutaneous adipose tissue evaluated by computed tomography. Values are means and standard deviations represented by vertical bars. * $P < 0{\cdot}05$ for intervention with general linear model for repeated measurements (before (□) *v.* after (▩)).

Conclusion

A reduction of 5 % of the initial body weight resulted in significant decreases in VAT and total abdominal adipose tissue in obese individuals, the primary outcome of the present study. Additionally, this weight loss decreased HOMA-IR and hs-CRP. Exercise did not add any measurable benefit in as far as the variables in the present study are considered.


BOM... EU ACHO QUE VOCÊ ESTÁ NO PESO IDEAL. SÓ PRECISA CRESCER UNS TRÊS METROS AGORA.
LEE331

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Eight-Year Weight Losses with an Intensive Lifestyle Intervention: The Look AHEAD Study

The Look AHEAD Research Group

Abstract

Objective—To evaluate 8-year weight losses achieved with intensive lifestyle intervention (ILI) in the Look AHEAD (Action for Health in Diabetes) study.

Design and Methods—Look AHEAD assessed the effects of intentional weight loss on cardiovascular morbidity and mortality in 5,145 overweight/obese adults with type 2 diabetes, randomly assigned to ILI or usual care (i.e., diabetes support and education [DSE]). The ILI provided comprehensive behavioral weight loss counseling over 8 years; DSE participants received periodic group education only.

Results—All participants had the opportunity to complete 8 years of intervention before Look AHEAD was halted in September 2012; ≥88% of both groups completed the 8-year outcomes assessment. ILI and DSE participants lost (mean±SE) 4.7±0.2% and 2.1±0.2% of initial weight, respectively ($p<0.001$) at year 8; 50.3% and 35.7%, respectively, lost ≥5% ($p<0.001$), and 26.9% and 17.2%, respectively, lost ≥10% ($p<0.001$). Across the 8 years ILI participants, compared with DSE, reported greater practice of several key weight-control behaviors. These behaviors also distinguished ILI participants who lost ≥10% and kept it off from those who lost but regained.

Conclusions—Look AHEAD's ILI produced clinically meaningful weight loss (≥5%) at year 8 in 50% of patients with type 2 diabetes and can be used to manage other obesity-related co-morbid conditions.

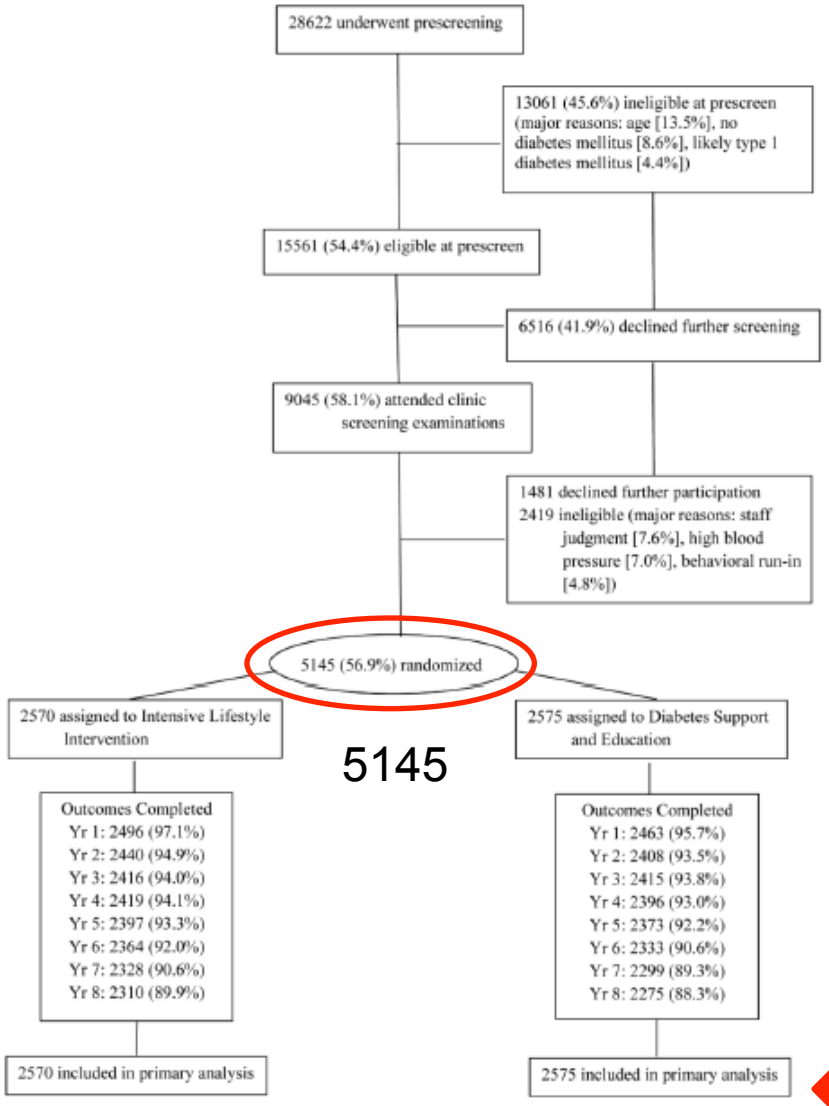


Figure 1.
Flowchart for screening, randomization, and follow-up of participants. Participants who did not complete outcome assessments in years 1 through 8 had either died, withdrawn from the study, or missed the assessment.

AMOSTRA

- Ambos os sexos
- 16 Centros dos USA
-DM2 e sobrepeso
-Aptos para exercício

Estudo de efetividade

Intervenção no Estilo de Vida: Redução > 7% PI em 12 meses

Dieta

- Dieta de restrição calórica:
  - 1200 a 1800 kcal/dia, segundo recomendações gerais.
- 1-4 mês:
  - Refeições estruturadas e fornecidas: shakes e barras
  - Substituir 2 refeições e 1 lanche
- 5-12 mês:
  - Substituir 1 refeição e 1 lanche

Treinamento Físico

- 1-6 mês:
  - 175 minutos/semana;
  - Nas primeiras três semanas do mês: grupo (60-75min);
  - Na última semana do mês: individual (20-30min).
- 7-12 mês:
  - Aumentar para 200 minutos/ semana;
  - -Redução dos encontros do grupo (2x mês) e manutenção dos encontros individuais.

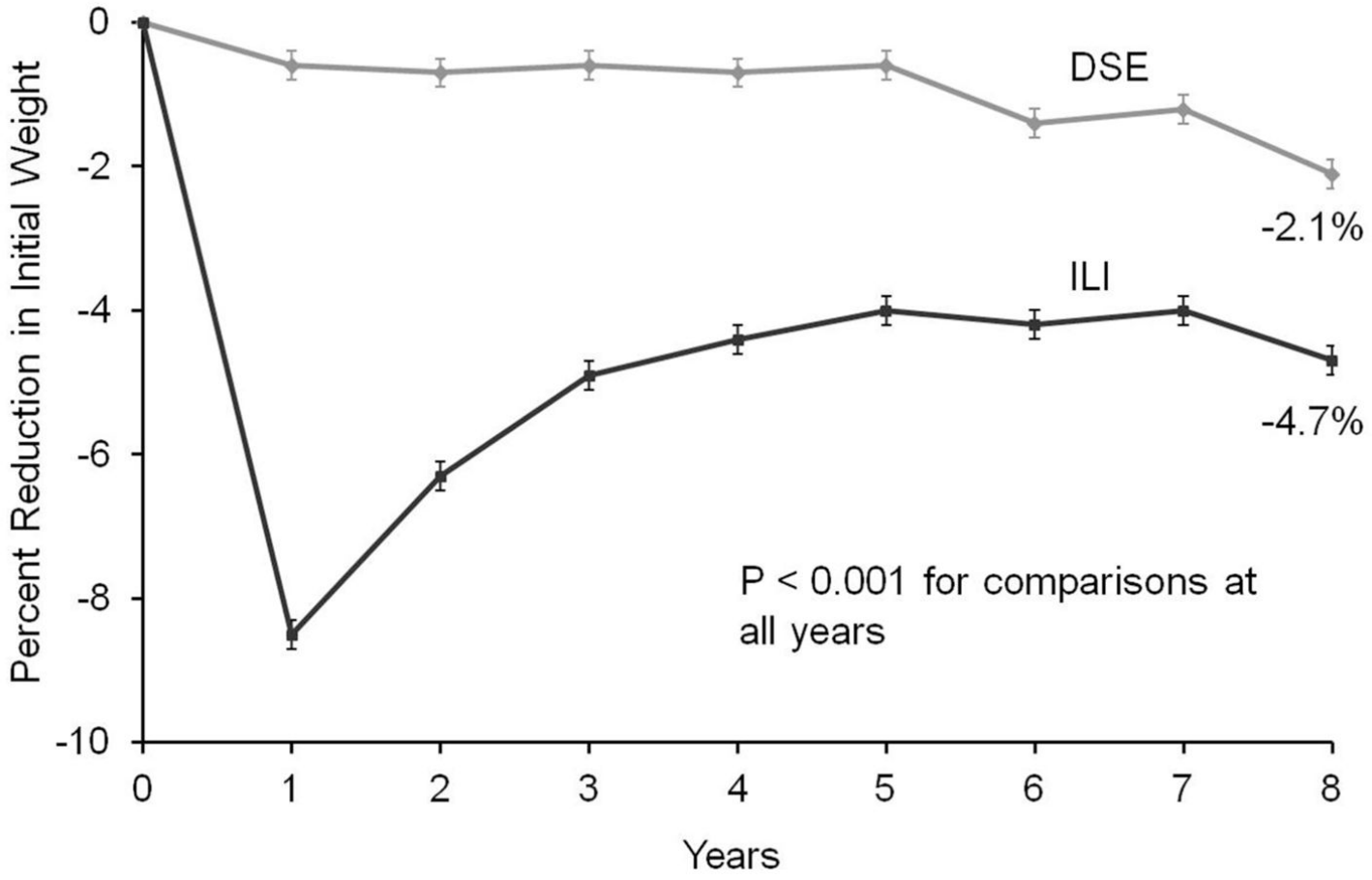


Figure 2.
Figure shows mean (±SE) weight losses over 8 years for participants randomly assigned to an intensive lifestyle intervention (ILI) or diabetes support and education (DSE; usual care group). Differences between groups were significant ($p<0.001$) at all years.

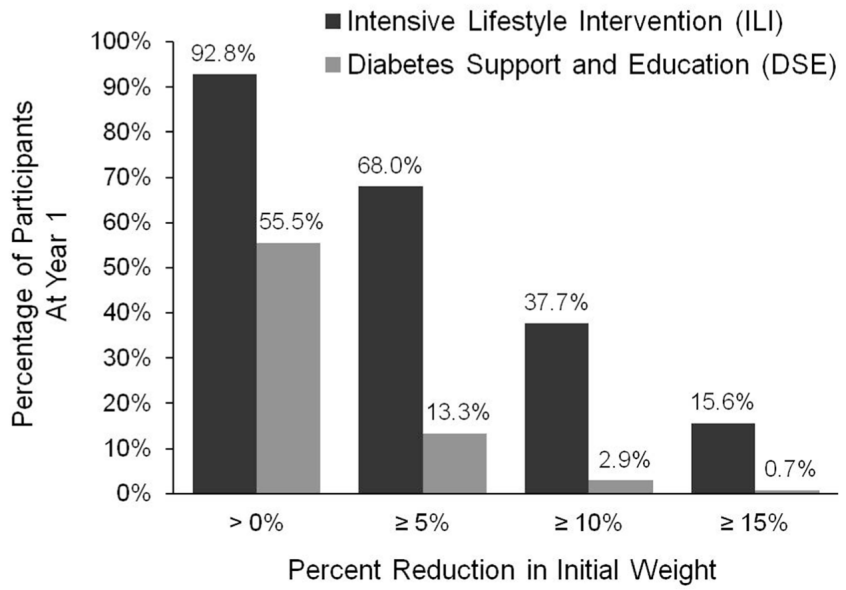
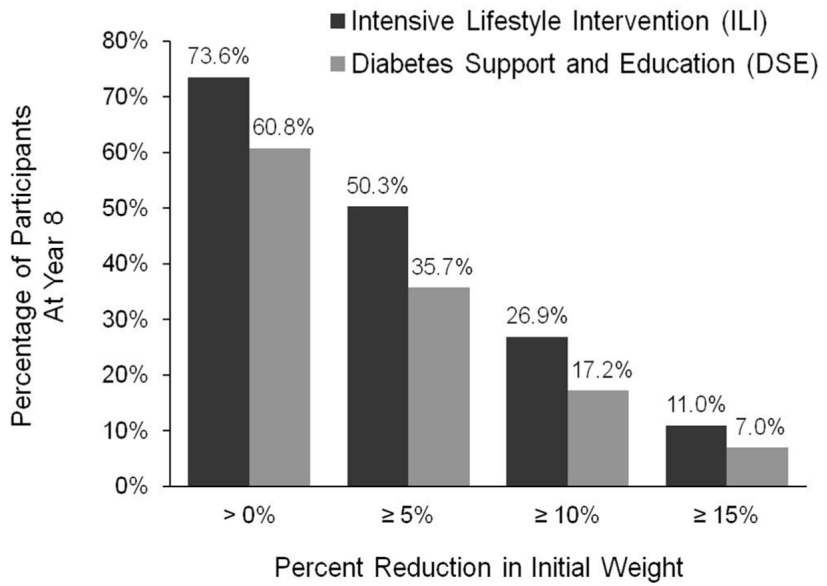


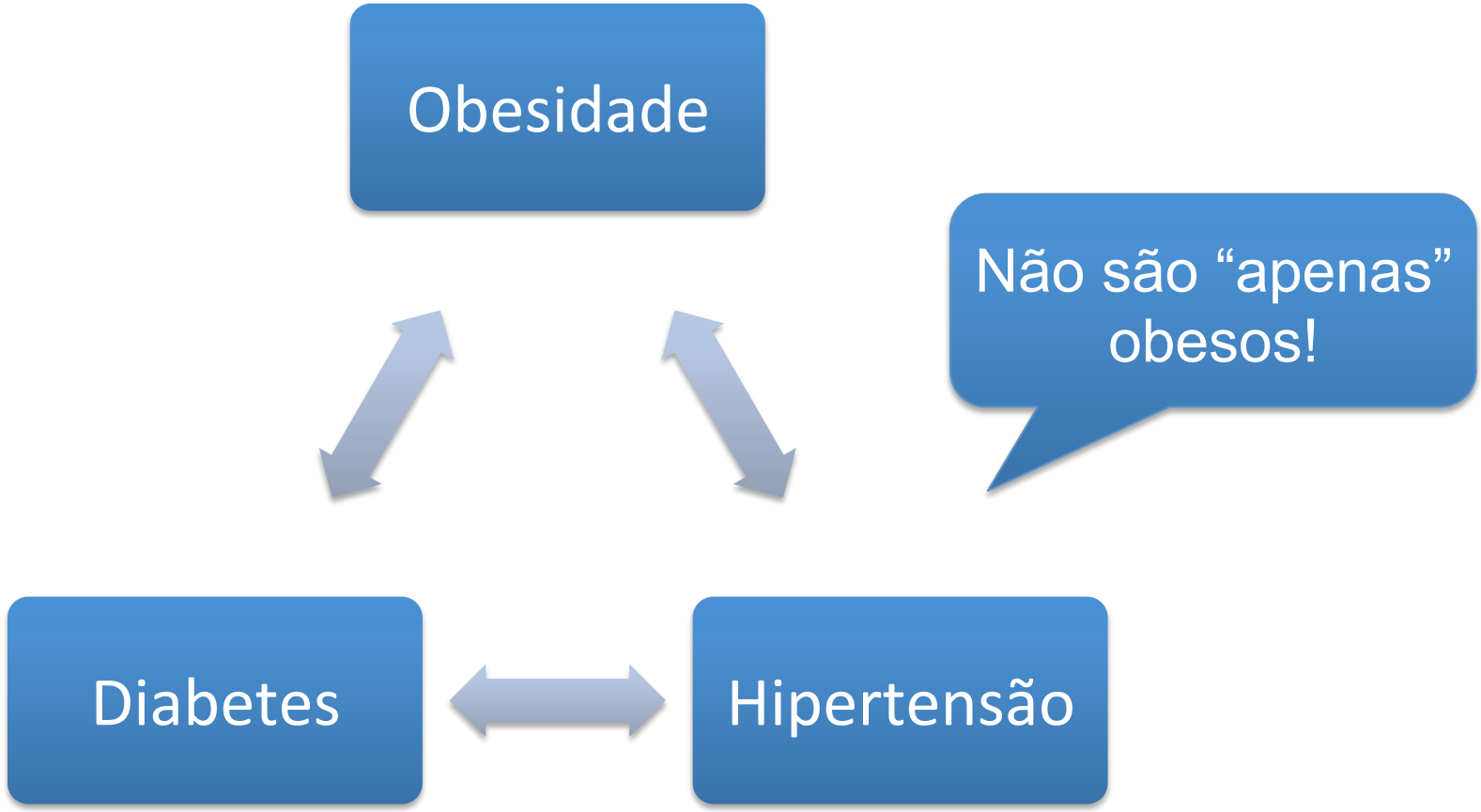
Figure 3b



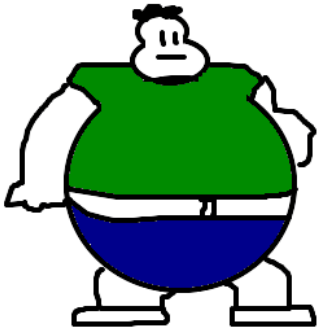
* Em 8 anos, 50% dos pacientes do grupo intervenção intensa perdeu mais de 5% do PI.
* Com o passar dos anos, a educação passa a ter uma contribuição importante para a perda de peso.


ESTÁ TÃO DIFÍCIL EMAGRECER
Frozen Indelicada
QUE ESTOU PENSANDO EM TOMAR DETERGENTE YPÊ, QUE REMOVE ATÉ AS GORDURAS MAIS DIFÍCEIS...

A grande dificuldade na interpretação dos estudos sobre obesidade:



Apesar das evidências...



Não é uma condição homogênea

Lemieux I et al. Hypertriglyceridemic waist: A useful screening phenotype in preventive cardiology? Can J Cardiol 2007;23(Suppl B):23B-31B.

Curr Atheroscler Rep (2014) 16:441
DOI 10.1007/s11883-014-0441-1

NUTRITION (JP FOREYT, SECTION EDITOR)

Metabolically Healthy Obesity—Does it Exist?

Patchaya Boonchaya-anant · Caroline M. Apovian

Published online: 5 August 2014

Abstract The prevalence of obesity has been increasing worldwide over the past 30 years and is a major public health concern. Obesity is known to be associated with metabolic disturbances including insulin resistance and inflammation; however, there is a subset of obese subjects who have normal metabolic profiles, and they have been identified as the metabolically healthy obese (MHO). Several studies have described MHO as obese individuals who have high levels of insulin sensitivity and the absence of diabetes, dyslipidemia, or hypertension. The prevalence of MHO varies from 20 to 30 % among obese individuals. This review will discuss the MHO phenotype; the differences between MHO and metabolically unhealthy obese (MUO) individuals; and the possible underlying mechanisms including adipocyte differentiation, immune regulation, and cellular energy metabolism.

Keywords Metabolically healthy obese · Insulin resistance · Adipose tissue inflammation

This article is part of the Topical Collection on *Nutrition*

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Introduction

The prevalence of obesity has been increasing worldwide over the past 30 years. A recent report has shown that from 1980 to 2013, the prevalence of obesity continued to rise in both developed and developing countries, especially in children and adolescents. In addition, the percentages of adults with a body mass index (BMI) of ≥25 kg/m^2 were reported to be 36.9 % in men and 38 % in women [1•]. The increase in the prevalence of obesity is also parallel with the increase in the prevalence of diabetes. Obesity is well described as a major risk factor for metabolic complications such as insulin resistance, type 2 diabetes, nonalcoholic fatty liver disease, dyslipidemia, hypertension, and sleep apnea and can lead to many other morbidities including atherosclerosis, cardiovascular disease, and cancer as well as increased mortality [2].

Obesity was declared as a disease itself by the American Medical Association in 2013 and continues to be considered a major public health issue. In 2008, it was estimated that the US healthcare costs related to obesity were $147 billion per year, and the annual healthcare costs for an obese individual were 42 % higher compared to a normal-weight individual [3].

Fat, Inflammation, and Adipose Tissue Dysfunction

Adipose tissue is now considered an endocrine organ due to its ability to secrete adipokines, which are bioactive molecules that play an important role in glucose/lipid metabolism, insulin sensitivity, regulation of appetite/satiety, immune cell attraction, and endothelial function [4]. With the development of obesity, adipose tissue becomes dysfunctional and an altered adipokine secretion pattern has been demonstrated. In obese individuals, lower adiponectin levels [5], increased retinol-binding protein 4 (RBP4) levels [6], and increased resistin levels [7] in the circulation were associated with insulin resistance and systemic inflammation. Adipose tissue

The Myth of Healthy Obesity

In this issue, Kramer and colleagues' meta-analysis provides strong evidence that "healthy obesity" is a myth (1). This evidence fuels the debate about the existence of a subset of obese persons who are unlikely to have long-term, negative health effects and should not be targeted for treatment (2). The review identified 8 studies that included a total of 61 386 persons followed long enough to investigate the associations of body mass index (BMI) and metabolic status with total mortality and cardiovascular events.

Not surprisingly, the evidence showed that metabolically healthy nonobese persons (the reference group) had the lowest risk for these outcomes and that being metabolically unhealthy, regardless of BMI, increased risk. The most interesting finding was that the metabolically healthy obese group was also at increased risk. However, this risk was observed only in studies with more than 10 years of follow-up. Metabolically healthy overweight persons had a risk similar to that of the reference group.

Kramer and colleagues conclude that being metabolically unhealthy at any weight confers health risks and that normal weight does not indicate cardiometabolic health. These findings cast doubt on the existence of metabolically healthy obesity. The authors speculate that persons who are metabolically healthy but obese probably have subclinical levels of risk factors that worsen over time. If so, the question is whether this change in risk is an inevitable consequence of obesity or is due to subsequent weight gain or behaviors. For obese persons to be truly healthy, must they have and maintain a healthy lifestyle?

Also of interest is that the metabolically unhealthy overweight group had an increased risk for total mortality and cardiovascular events over time, whereas the metabolically healthy overweight group did not. Controversy exists over the effect of overweight on total mortality, with some reports suggesting that overweight may be protective (3). It is essential to consider metabolic risk factors when examining the effect of overweight on mortality.

The meta-analysis has limitations. Most studies had inadequate information on participants' health behaviors, did not present data about weight gain, focused only on total mortality and cardiovascular events, and did not include older participants. By uncovering the limitations of the current evidence, this review will hopefully stimulate research to more thoroughly understand the interactions among weight status, metabolic status, and health outcomes. The results are consistent with the notion that obesity is a disease. In light of these findings, we consider common misperceptions about obesity.

First, the review casts doubt that any obese persons have no long-term risk for cardiometabolic disease. Obesity affects almost all aspects of human function and physiology. Although Kramer and colleagues focused on total mortality and cardiovascular events, obesity also increases risk for type 2 diabetes, kidney disease, and some types of cancer (4). It is linked to orthopedic problems, reproductive problems, depression, asthma, sleep apnea, renal disease, back pain, skin infections, and cognitive decline (4). Obesity produces social stigma and overall reduced quality of life (5). It would be a mistake to label obese persons as healthy on the basis of only the presence or absence of risk factors for cardiometabolic disease.

A second common misperception is that we cannot afford to treat everyone with obesity, so we have to prioritize those with cardiometabolic risk. However, doing so would deny treatment to those who may later develop cardiometabolic disease. Although many health care providers argue that avoiding diabetes and cardiovascular disease is the most important reason to tackle obesity, many patients would probably prioritize other outcomes. We believe that there are many good reasons to lose weight. If we assume that we cannot afford to treat all obesity, denying treatment on the basis of cardiometabolic risk will be extremely difficult to justify.

A third misperception is that effective treatment for obesity is unavailable. Although we lack a simple algorithm or medication to eradicate this condition, clinically significant weight loss can be achieved with behavioral treatment, pharmacologic agents, and bariatric surgery (6). However, treatment of obesity brings real challenges.

Obesity is not cured even when the excessive body fat is successfully reduced. Patient adherence and long-term sustainability are just as challenging in treatment of obesity as they are in long-term treatment of any disease. Losing weight and maintaining a reduced body weight are different physiologic processes and therefore require different treatment strategies for maximum success (7). Just as in treatment of other chronic conditions, treatment of obesity needs to be evaluated and adjusted over time to maximize success. Health care providers may not have eagerly stepped up to tackle obesity partly because many practicing physicians today have had no formal training in treating this condition and do not feel confident that they have the tools, skills, and time needed to be successful.

A fourth misperception is that weight loss and reducing cardiometabolic risk are the highest-priority goals in obesity treatment. Yet, is a person who has lost enough weight to achieve normal metabolic measures but who has sleep problems, orthopedic issues, or difficulty managing stress really "healthy"? Perhaps we need a more comprehensive measurement of well-being to measure success. For example, decades of work from Blair and associates (8) has consistently shown that cardiorespiratory fitness is a very strong predictor of total and cardiovascular mortality independent of BMI. We must develop a means of assessing success in obesity treatment that considers overall well-

European Journal of Clinical Nutrition (2010) 64, 1043–1051

0954-3007/10
www.nature.com/ejcn

ORIGINAL ARTICLE

Metabolically healthy obesity: different prevalences using different criteria

S Velho[1], F Paccaud[2], G Waeber[3], P Vollenweider[3] and P Marques-Vidal[2,4]

Table 1 Criteria used for the definition of MHO

	Wildman et al. (2008)	*Karelis et al. (2004)*	*Lynch et al. (2009)*	*Meigs et al. (2006)*[a]	*Meigs et al. (2006)*[b]	*Aguilar-Salinas et al. (2008)*
Blood pressure	SBP ⩾130 or DBP ⩾85 or treatment	—	SBP⩽130 and DBP⩽85 and no treatment	SBP ⩾130 or DBP ⩾85 or treatment	—	SBP <140 and DBP <90 and no treatment
Triglycerides	⩾1.70 mmol/l	<1.70 mmol/l	—	⩾1.70 mmol/l	—	—
HDL-cholesterol	<1.04 mmol/l (M) <1.30 mmol/l (W) or treatment	⩾1.30 mmol/l and no treatment	—	<1.04 mmol/l (M) <1.30 mmol/l (W)	—	⩾1.00 mmol/l
LDL-cholesterol	—	<2.60 mmol/l and no treatment	—	—	—	—
Total cholesterol	—	<5.20 mmol/l	—	—	—	—
TG/HDL ratio	—	—	⩽1.65 (M) ⩽1.32 (W) and no treatment	—	—	—
Fasting glucose	⩾5.55 mmol/l or treatment	—	⩽5.60 mmol/l and no treatment	⩾5.60 mmol/l or treatment	—	<7.00 mmol/l and no treatment
HOMA	⩾90th percentile	<1.95	—	—	<75th percentile[c]	—
Other	CRP⩾90th percentile	—	No history of cardiovascular, respiratory or metabolic diseases	Waist ⩾102 cm (M) Waist ⩾88 cm (W)	—	—
Criteria for MHO	<2 of the above	⩾4 of the above	All	<3 of the above	All	All

Eur J Nutr (2013) 52:1443–1450
DOI 10.1007/s00394-012-0450-1

ORIGINAL CONTRIBUTION

Effects of 5 % weight loss through diet or diet plus exercise on cardiovascular parameters of obese: a randomized clinical trial

Ana Paula Trussardi Fayh · André Luiz Lopes · Antônio Marcos Vargas da Silva · Álvaro Reischak-Oliveira · Rogério Friedman

Table 2 Anthropometric and biochemical changes with interventions

	DI ($n = 18$)			DI + EXE ($n = 17$)			P^a	P^b	P^c
	Before	After	Change	Before	After	Change			
Body weight (kg)	95.8 ± 13.7	91.5 ± 14.2	−4.31 ± 0.5	98.7 ± 13.0	94.0 ± 13.0	−4.66 ± 0.52	0.00	0.64	0.63
Body mass index (kg/m^2)	34.7 ± 2.4	33.1 ± 2.6	−1.58 ± 0.17	34.7 ± 2.4	33.1 ± 2.1	−1.62 ± 0.17	0.00	0.79	0.79
Waist circumference (cm)	112.0 ± 8.7	108.3 ± 8.7	−3.42 ± 0.44	110.9 ± 7.4	107.0 ± 7.8	−3.92 ± 0.45	0.00	0.76	0.76
Hip circumference (cm)	120.4 ± 8.8	117.1 ± 8.6	−3.31 ± 1.18	120.2 ± 5.1	117.0 ± 4.7	−3.18 ± 2.2	0.00	0.83	0.84
Waist-to-hip ratio	0.83 ± 0.09	0.83 ± 0.09	0.00 ± 0.00	0.86 ± 0.08	0.85 ± 0.07	0.01 ± 0.00	0.06	0.05	0.09
Total cholesterol (mg/dL)	191.4 ± 31.5	175.4 ± 37.1	−15.83 ± 4.75	185.5 ± 31.2	175.3 ± 32.6	−10.47 ± 4.89	0.00	0.40	0.44
HDL cholesterol (mg/dL)	45.5 ± 7.6	42.1 ± 9.3	−3.55 ± 1.60	47.2 ± 11.6	44.6 ± 11.2	−2.47 ± 1.65	0.02	0.75	0.64
LDL cholesterol (mg/dL)	115.4 ± 27.2	109.5 ± 29.1	−5.23 ± 4.13	108.8 ± 29.0	107.8 ± 27.3	−1.67 ± 4.25	0.27	0.43	0.55
Triglycerides (mg/dL)	122 (94–206)	94 (65–177)	−33.8 ± 10.0	142 (83–202)	104 (67–158)	−39.4 ± 10.3	0.00	0.81	0.70
hs-CRP (mg/L)	3.3 (2.4–6.4)	2.8 (1.5–4.8)	−1.35 ± 0.41	3.5 (1.5–5.8)	3.0 (1.1–5.9)	−0.45 ± 0.43	0.01	0.13	0.14

Data are presented in mean ± SD or median (interquartile range)

hs-CRP high-sensitivity C-reactive protein

P^a for intervention with repeated measures general linear model

P^b for intervention x group with repeated measures general linear model

P^c with analysis of covariance adjusted for baseline measures

Table 1. Anthropometric and biochemical changes with different interventions

(Mean values and standard deviations; medians and interquartile ranges (IQR))

	DI (*n* 18)				DI + EXE (*n* 17)								
	Before		After		Before		After			Mean difference between groups	95 % CI		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P**		Lower	Upper	*P*†
Age (years)	30·1	5·5	–	–	32·4	7·0	–	–	–	–	–	–	–
Body weight (kg)	95·8	13·7	91·5	14·2	98·7	13·0	94·0	13·0	0·00	0·35	−1·13	1·83	0·63
Vo_{2max} (ml/kg per min)	24·5	4·3	25·2	4·4	25·1	4·9	27·6	5·2	0·04	1·8	0·35	2·8	0·02
BMI (kg/m^2)	34·7	2·4	33·1	2·6	34·7	2·4	33·1	2·7	0·00	0·01	−0·02	0·02	0·79
Waist circumference (cm)	112·0	8·7	108·3	8·7	110·9	7·4	107·0	7·8	0·00	0·20	−1·08	1·47	0·76
Hip circumference (cm)	120·4	8·8	117·1	8·6	120·2	5·1	117·0	4·7	0·00	−0·12	−1·29	1·06	0·84
Waist:hip ratio	0·83	0·09	0·83	0·09	0·86	0·08	0·85	0·07	0·06	0·01	−0·01	0·02	0·09
Glycaemia (mg/l)	924	92	897	92	904	97	904	68	0·42	−27·2	−60·4	5·9	0·59
Insulin (μIU/ml)‡									0·01	−0·03	−0·15	0·08	0·56
Median	17·3		15·4		15·5		13·4						
IQR	14·7–21·6		7·8–19·6		11·4–18·7		8·8–19·8						
HOMA									0·01	−0·04	−0·17	0·08	0·53
Median	4·1		3·3		3·1		3·2						
IQR	3·1–5·6		1·7–4·3		2·5–4·7		2·0–4·4						
Uric acid (mg/l)	5·4	1·3	5·3	1·1	5·4	1·6	5·4	1·1	0·65	−0·01	−0·39	0·36	0·95
hs-CRP (mg/l)									0·01	−0·06	−0·19	0·08	0·39
Median	3·3		2·8		3·5		3·0						
IQR	2·4–6·4		1·5–4·8		1·5–5·8		1·1–5·9						

DI, diet-only group; DI + EXE, diet and exercise group; HOMA, homeostasis model assessment; hs-CRP, high-sensitivity C-reactive protein.

* *P* for intervention with general linear model for repeated measurements (before *v.* after).

† *P* with ANCOVA adjusted for baseline measures.

‡ 1 μIU/ml = 6.945 pmol/L.

O grupo que treinou não diminui glicemia de jejum e resistência à insulina!

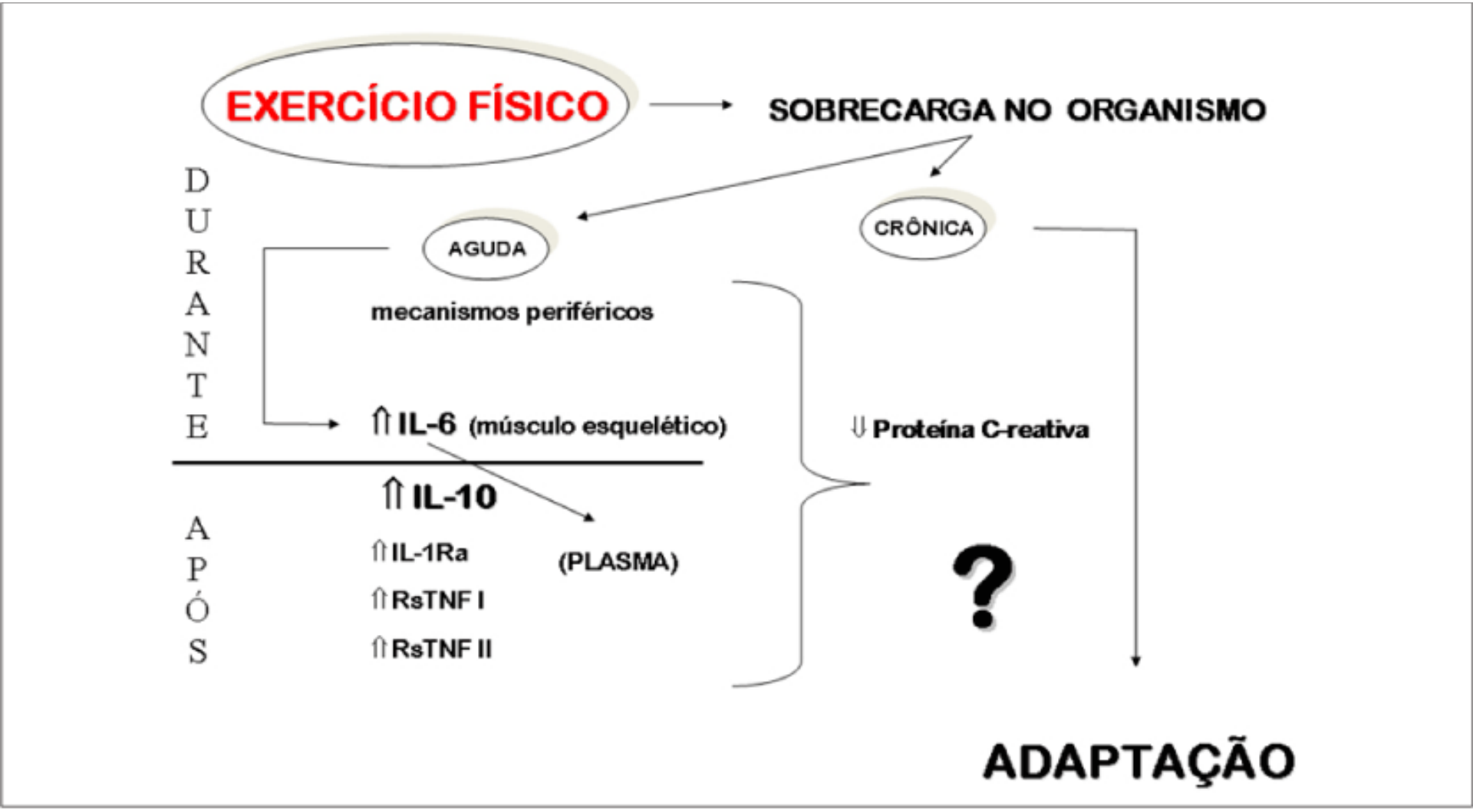


Fig. 2 - Efeitos agudo e crônico (treinamento) do exercício nos níveis plasmático de citocinas antiinflamatórias, mediadas pela interleucina 6 (IL-6), secretada pelo músculo esquelético (agudamente). O treinamento físico induz à redução nos níveis de proteína C reativa (PCR), condição mais evidente em quadros patológicos que apresentam níveis elevados dessa proteína de fase aguda (cronicamente). Além disso, supõe-se que a somatória dos efeitos agudos tendem a direcionar ao predomínio das citocinas antiinflamatórias, principalmente a interleucina 10 (IL-10). IL-1ra: receptor antagonista para IL-1; RsTNF-I: receptores de TNF-α tipo I; RsTNF-II: receptores de TNF-α tipo II.

Paradoxo da Obesidade

Epidemiologia Reversa { **Obesidade = Proteção Mortalidade**

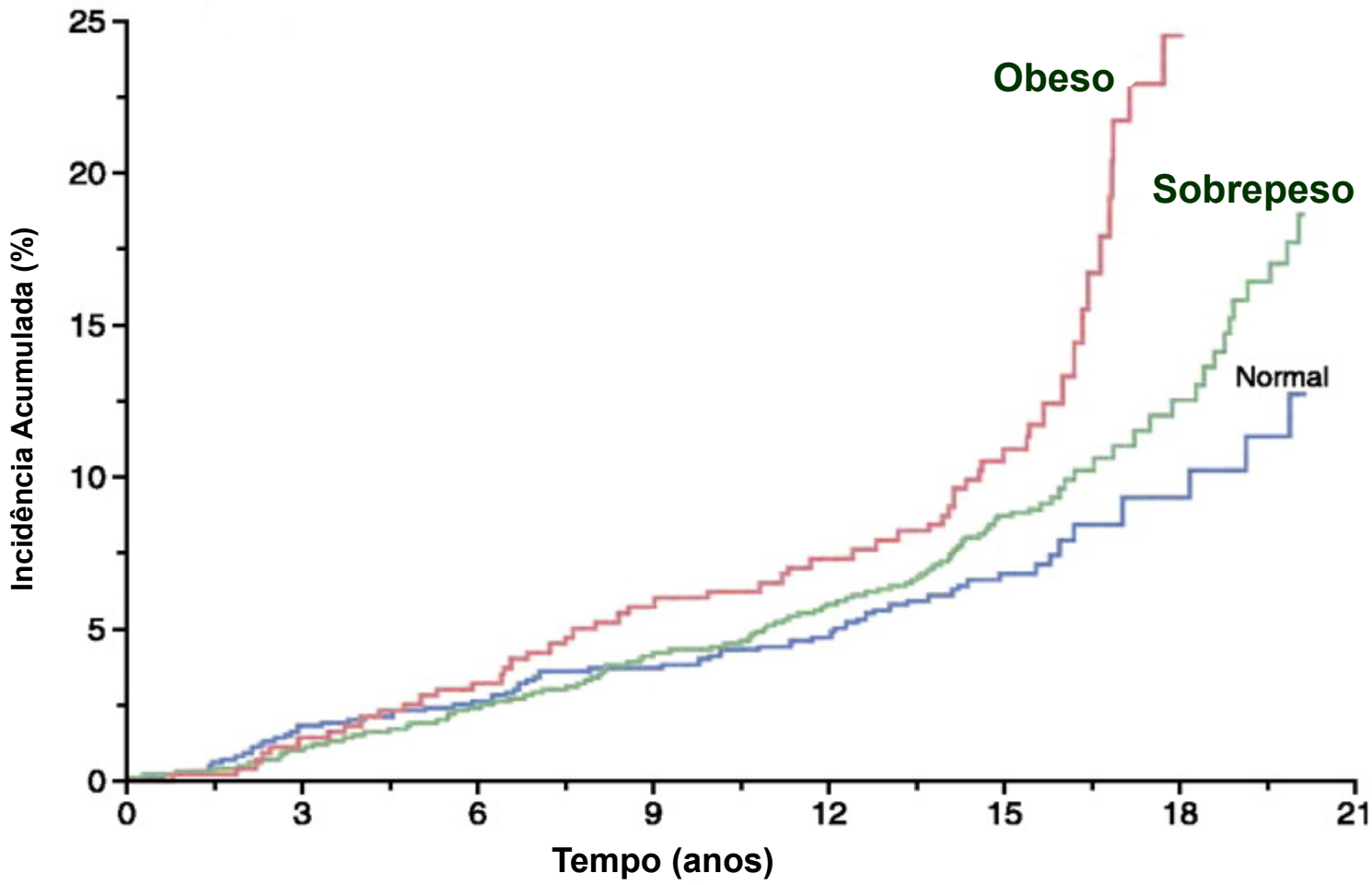
Insuficiência Renal Crônica

Insuficiência Cardíaca

Transição Nutricional

Paradoxo da Obesidade

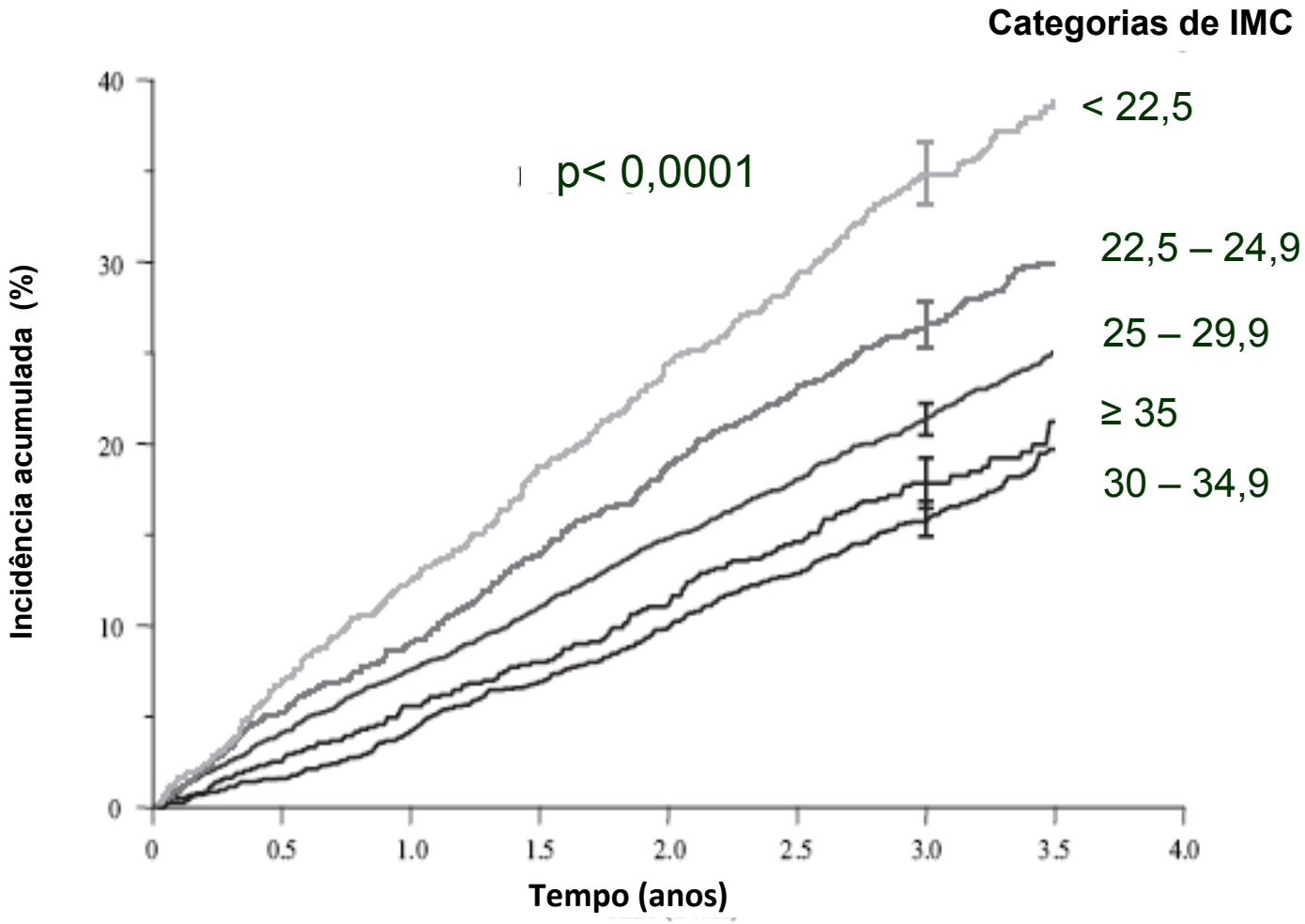
IMC e desenvolvimento de IC



Abel et al. Physiol Rev 2006;88: 389–419

Paradoxo da Obesidade

IMC e Mortalidade na IC



Kenchaiah S et al. Circulation 2007;116(6):627-36

Paradoxo da Obesidade

Outras condições clínicas

Hipertensão

Doença arterial coronariana

Revascularização percutânea

Cirurgia cardíaca

Doença arterial periférica

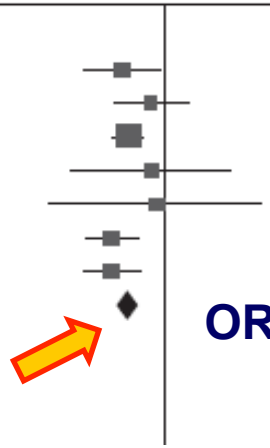
Achados eletrocardiográficos específicos

Lavie et al. J Am Coll Cardiol 2009;53:1925–32

Paradoxo da Obesidade

Metanálise – mortalidade pós CRM

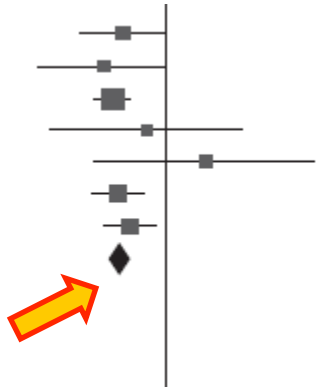
Study or sub-category	Overweight/obesity n/N	Normal BMI n/N	OR (random) 95% CI
01 Normal BMI vs. Overweight in PCI			
Christakis	68/3389	54/1825	
Kim	65/3320	52/2344	
Potapov	385/9050	591/10131	
Reeves	18/2170	11/1166	
Orhan	11/632	6/320	
Jin	129/6683	108/3475	
Pan	125/4257	103/2184	
Subtotal (95% CI)	29501	21445	
Total events: 801 (Overweight/Obesity), 925 (Normal BMI)			
Test for heterogeneity: $Chi^2 = 4.30$, df = 6 ($P = 0.64$), $I^2 = 0\%$			
Test for overall effect: Z = 7.23 ($P < 0.00001$)			



Sobrepeso

OR 0,70 (IC 95% 0,63 – 0,77)

02 Normal BMI vs. Obese		
Christakis	38/1811	54/1685
Kim	11/924	52/2344
Potapov	158/4518	591/10131
Reeves	7/903	11/1166
Orhan	7/254	6/320
Jin	116/5970	108/3475
Pan	115/3421	103/2184
Subtotal (95% CI)	17801	21305
Total events: 452 (Overweightflbesity), 925 (Normal BMI)		
Test for heterogeneity: $Chi^2 = 4.19$, df = 6 ($P = 0.65$), $I^2 = 0\%$		
Test for overall effect: Z = 7.60 ($P < 0.00001$)		



Obesidade

OR 0,63 (IC 95% 0,56 – 0,71)

Oreopoulos et al.Obesity 2008;16:442–50

Paradoxo da Obesidade

Mecanismos

- Reservas energéticas aumentadas – caquexia
- ↑ produção repectores TNF-α – tecido adiposo

  IC isquêmica - ↑ produção TNF-α
- Detoxicação LPS (lipopolissacarídeos bacterianos tóxicos) – colesterol e triglicerídeos

Viés de Seleção ?

Arena R et al. Future Cardiol 2010; 6:1–6

The Obesity Paradox: Fact or Fiction?

Amit Habbu, MBBS, MPH[a], Nasser M. Lakkis, MD[b], and Hisham Dokainish, MD[b,*]

Although the adverse health consequences of obesity in the general population have been well documented, recent evidence suggests that obesity is associated with better outcomes in patients with heart failure (HF). Studies of patients with HF that specifically examined the impact of body mass index (BMI) on outcomes have suggested the existence of an "obesity paradox." However, closer examination of these studies raises important questions on the validity of the paradox. First, the diagnosis of HF in obese patients, particularly when made using clinical variables, may not be accurate; the obese patients in these studies may actually be "healthier" than their nonobese comparators. Second, the deleterious effects of cachexia, rather than the salutary ones of obesity, are likely the main reason for the inverse correlation between BMI and HF outcome, especially once the underlying biologic mechanisms behind cachexia and obesity in patients with HF are considered. Furthermore, few studies have specifically examined the more severely obese population (BMI >35 kg/m^2) when assessing outcomes, and those that have suggest that severely obese patients may have worse outcomes than patients with normal weights or those who are mildly obese. Therefore, a "U-shaped" outcome curve according to BMI for patients with HF may actually exist, in which mortality is greatest in cachectic patients; lower in normal, overweight, and mildly obese patients; but higher again in more severely obese patients. Further prospective studies assessing the impact of more marked degrees of obesity on outcomes in patients with HF are needed to more conclusively determine whether the obesity paradox truly exists. (Am J Cardiol 2006; 98:944–948)


E SE A GENTE TENTASSE COM HOMEOPATIA ?
É DE COMER ?
CLAÚDIA& BIRA


GIASBERGEN
Dentro de mim, há uma pessoa elegante, lutando para sair. Felizmente, assim, ela está queimando um monte de calorias.

The NEW ENGLAND JOURNAL of MEDICINE

SPECIAL ARTICLE

Myths, Presumptions, and Facts about Obesity

Krista Casazza, Ph.D., R.D., Kevin R. Fontaine, Ph.D., Arne Astrup, M.D., Ph.D., Leann L. Birch, Ph.D., Andrew W. Brown, Ph.D., Michelle M. Bohan Brown, Ph.D., Nefertiti Durant, M.D., M.P.H., Gareth Dutton, Ph.D., E. Michael Foster, Ph.D., Steven B. Heymsfield, M.D., Kerry McIver, M.S., Tapan Mehta, M.S., Nir Menachemi, Ph.D., P.K. Newby, Sc.D., M.P.H., Russell Pate, Ph.D., Barbara J. Rolls, Ph.D., Bisakha Sen, Ph.D., Daniel L. Smith, Jr., Ph.D., Diana M. Thomas, Ph.D., and David B. Allison, Ph.D.

N Engl J Med 2013;368:446-54.
DOI: 10.1056/NEJMsa1208051

MITO 1

MITO

- Pequenas mudanças sustentáveis no consumo ou gasto energético produzirão grandes alterações de peso a longo prazo.

BASE DA CONJECTURA

- Diretrizes nacionais de saúde e sites respeitáveis anunciam que pequenas e sustentadas modificações no estilo de vida podem proporcionar

  grandes alterações no peso (ex: caminhar por 20 minutos/dia ou comer duas batatas fritas adicionais).

MITO 2

MITO

- Definir metas realistas no tratamento da obesidade é importante, caso contrário os pacientes ficam frustrados e perdem menos peso.

BASE DA CONJECTURA

- Metas inatingíveis prejudicam o desempenho no tratamento da obesidade;
- Incongruência entre a perda de peso desejada e real pode levar à interrupção de comportamentos necessários para perda de peso.

MITO 3

MITO

- Perda de peso rápida e em grande quantidade prejudica a manutenção do peso em longo prazo, quando comparada com a perda de peso lenta e gradual.

BASE DA CONJECTURA

- Provavelmente surgiu em reação aos efeitos adversos da dietas de muito baixa caloria (<800 kcal por dia) na década de 1960;
- A crença persistiu, foi repetida nos livros didáticos e recomendações das autoridades de saúde, e tem sido oferecida como regra por nutricionistas.

MITO 4

MITO

- Avaliar o estágio de mudança do comportamento alimentar ou prontidão à dieta é importante para pacientes que procuram tratamento para perda de peso.

BASE DA CONJECTURA

- Muitos acreditam que os pacientes que se sentem prontos para perder peso são mais propensos a fazer as mudanças de estilo de vida necessários.

Prontidão ≠ Adesão ≠ Perda de Peso

MITO 5

MITO

- Aulas de educação física em seu formato atual desempenham um importante papel na prevenção ou redução da obesidade infantil.

BASE DA CONJECTURA

- As aulas possuem duração suficiente, freqüência,

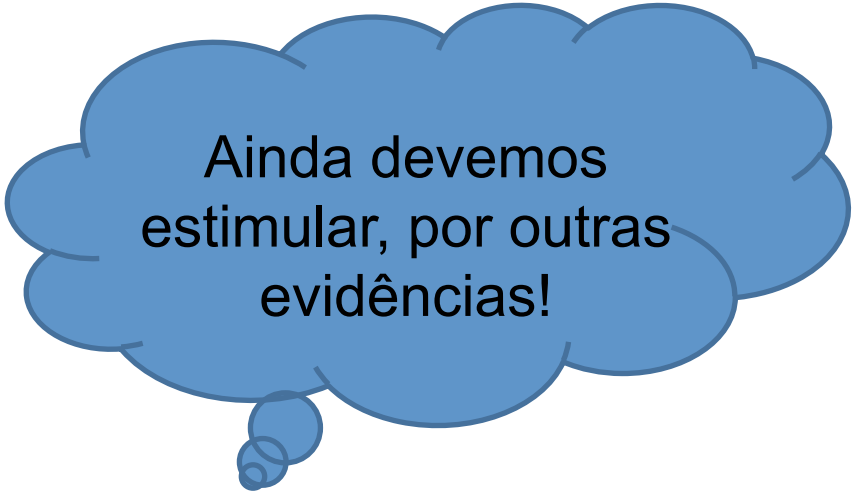
  e intensidade para promover adaptações benéficas para a saúde e redução da adiposidade.

Ensaios clínicos e Metanálises

MITO 6

MITO

- O aleitamento materno protege contra a obesidade.



BASE DA CONJECTURA

- A crença de que as crianças amamentadas têm menos probabilidade de se tornar obeso tem persistido por mais de um século e é apaixonadamente defendida.

Casazza K, Fernandez JR, Allison DB. Nutr Today 2012;47:33-8.
Kramer MS, Matush L, Vanilovich I, et al. Am J Clin Nutr 007;86:1717-21.
Gillman MW. Int J Epidemiol 2011;40:681-4.

MITO 7

MITO

- Uma atividade sexual vigorosa pode queimar 100 a 300 kcal por pessoa envolvida.

BASE DA CONJECTURA

- Muitas fontes afirmam que o gasto energético da atividade sexual de dois adultos é substancial.

Tempo de duração da atividade sexual :
60min ou 6 min (21 kcal)?

PRESUNÇÃO 1

PRESUNÇÃO

- Realizar regularmente comer (vs. pular) o desjejum é uma atitude protetora contra a obesidade.

BASE PARA A CONJECTURA

- Não realizar o desjejum supostamente leva a comer demais no final do dia.

Qualidade e quantidade do desjejum

PRESUNÇÃO 2

PRESUNÇÃO

- A primeira infância é o período durante o qual devemos aprender que hábitos como a realização de exercícios e alimentação saudável influenciam o nosso peso ao longo da vida.

BASE PARA A CONJECTURA

- Índices peso-para-altura, comportamentos alimentares e preferências que são presente na primeira infância persistem ao longo da vida.

Hábitos x Genética?

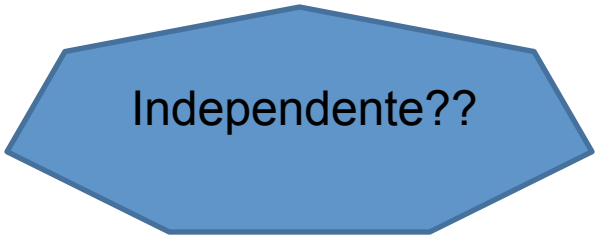
PRESUNÇÃO 3

PRESUNÇÃO

- Comer mais frutas e verduras resulta em perda de peso ou menos ganho de peso, independentemente de alterações intencionais comportamentais ou ambientais.

BASE PARA A CONJECTURA

- Ao comer mais frutas e verduras, uma pessoa presumivelmente e/ou espontaneamente come menos alimentos, e consequentemente, reduz sua ingestão calórica pela baixa densidade calórica destes alimentos.



PRESUNÇÃO 4

PRESUNÇÃO

- O efeito sanfona está associado com o aumento da mortalidade.

BASE PARA A CONJECTURA

- Em estudos observacionais, as taxas de mortalidade foram menores entre pessoas com peso estável do que entre aqueles com peso instável.

Possível viés de confusão, pois os achados não são reproduzidos em modelo animal.

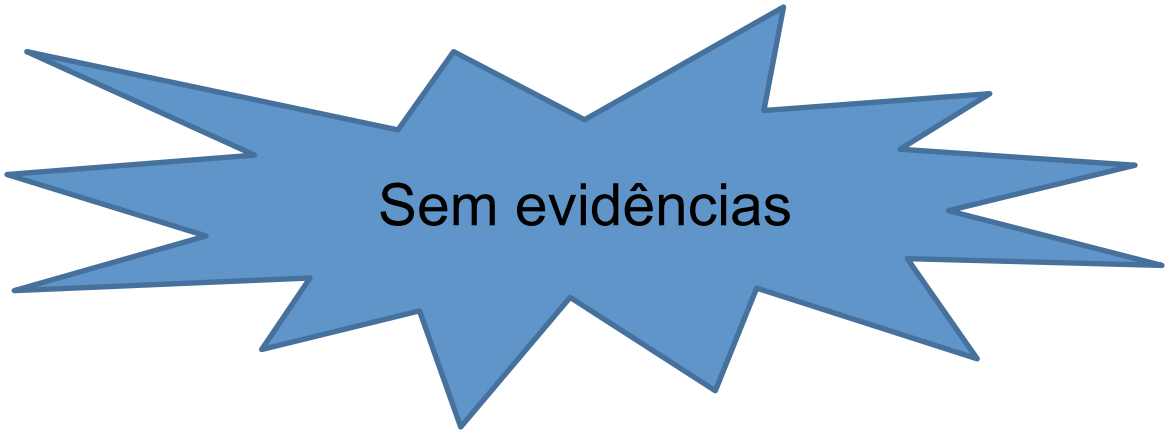
PRESUNÇÃO 5

PRESUNÇÃO

- Salgadinhos contribuem para o ganho de peso e obesidade.

BASE PARA A CONJECTURA

- Presume-se que o consumo de salgadinhos fica incompletamente compensado pelas refeições subseqüentes, levando ao ganho de peso.



PRESUNÇÃO 6

PRESUNÇÃO

- As construções ambientais, como calçadas e parque, influenciam na incidência ou prevalência da obesidade.

BASE PARA A CONJECTURA

- Recursos ambientais podem promover ou inibir física atividade, afetando a incidência ou prevalência da obesidade.

FATO 1

FATO

- Embora os fatores genéticos desempenham um grande papel, a hereditariedade não é destino; cálculos mostram que moderadas mudanças ambientais podem promover a perda de peso tanto quando o mais eficaz agente farmacêutico disponível.

IMPLICAÇÃO

- Se pudermos identificar os fatores ambientais fundamentais e influenciá-los com sucesso, podemos alcançar reduções clinicamente significativas na obesidade.

FATO 2

FATO

- Dietas (ou seja, redução do consumo de energia) são eficazes na redução do peso, mas geralmente as orientações não funcionam bem em longo prazo.

Eficácia x Efetividade

IMPLICAÇÃO

- Reconhecer esta distinção ajuda a entender que a redução energética é necessária; orientações como comer mais vegetais ou tomar café da manhã diariamente podem ajudar apenas se forem acompanhados por uma redução no consumo diário de energia.

FATO 3

FATO

- Independentemente do peso corporal ou perda de peso, um aumento do nível de exercício melhora a saúde.

IMPLICAÇÃO

- Exercício oferece uma maneira de atenuar os efeitos prejudiciais à saúde da obesidade, mesmo sem perda de peso.

FATO 4

FATO

- Atividade física ou exercício, em quantidade suficiente, auxilia na manutenção do peso em longo prazo.

IMPLICAÇÃO

- Programas de atividade física, são importantes, especialmente para as crianças, mas para promover alterações no peso, esta atividade deve possuir quantidade suficiente de movimento, e não a mera participação.

FATO 5

FATO

- A continuidade das condições que levaram à perda de peso promove a manutenção do baixo peso.

IMPLICAÇÃO

- A obesidade é melhor conceituada como uma condição crônica, necessitando de acompanhamento contínuo para manter a perda de peso a longo prazo.

FATO 6

FATO

- Para as crianças com excesso de peso, programas que envolvem os pais e o ambiente doméstico promovem uma maior perda ou manutenção de peso.

IMPLICAÇÃO

- Programas fornecidos apenas em escolas ou outros ambientes não domésticos podem ser convenientes, mas os programas que incluem intervenções que envolvem os pais e são fornecidos em casa tendem a produzir melhores resultados.

FATO 7

FATO

- O fornecimento de refeições e uso de produtos que substituem refeições promovem maior perda de peso.

IMPLICAÇÃO

- Estrutura e planejamento em relação as refeições estão associados com maior peso perda, em comparação com programas “holísticos” que são aparentemente embasados em conceitos de equilíbrio, variedade e moderação.

FATO 8

FATO

- Alguns fármacos podem ajudar os pacientes a atingir uma perda de peso clinicamente significativa, e a manter a redução enquanto estes ainda foram utilizados.

IMPLICAÇÃO

- Enquanto aprendemos a alterar o ambiente e os comportamentos individuais para prevenir a obesidade, podemos oferecer tratamento moderadamente eficaz para pessoas obesas.

FATO 9

FATO

- Em pacientes clinicamente indicados, a cirurgia bariátrica resulta em uma perda de peso a longo prazo e redução na taxa de incidência de diabetes e mortalidade.

IMPLICAÇÃO

- Para as pessoas com obesidade grave, a cirurgia bariátrica pode oferecer uma mudança de vida, e, em alguns casos graves, o tratamento.

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