

Central European Journal of Medicine

The weight change impact on metabolic syndrome: a 17-year follow-up study

Research Article

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Received 17 February 2011; Accepted 17 August 2011

Abstract: Introduction: Data on long-term patterns of weight change in relation to the development of metabolic syndrome (MetS) are scarce. The aim of the study was to evaluate the impact of weight change on the risk of MetS in men. Material and Methods: Prospective longitudinal observation (17.9 ± 8.1 years) of apparently healthy 324 men aged 18-64 years. Metabolic risk was assessed in weight gain (≥ 2.5 kg), stable weight (> -2.5 kg and < 2.5 kg) and weight loss (≤ -2.5 kg) groups. Adjusted relative risk (RR) of MetS was analyzed using multivariate logistic regression. Results: The prevalence of MetS over follow-up was 22.5%. There was a strong relationship between weight gain and worsening of MetS components among baseline overweight men. Long-term increase in weight was most strongly related with the risk of abdominal obesity (RR=7.26; 95% CI 2.98-18.98), regardless of baseline body mass index (BMI). Weight loss was protective against most metabolic disorders. Leisure-time physical activity (LTPA) with energy expenditure > 2000 metabolic equivalent/min/week was associated with a significantly lower risk of MetS. Conclusions: Reducing weight among overweight and maintaining stable weight among normal-weight men lower the risk of MetS. High LTPA level may additionally decrease the metabolic risk regardless of BMI.

Keywords: Weight change • Metabolic syndrome • Physical activity • Men • Prospective

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1. Introduction

Metabolic syndrome (MetS) as a cluster of abdominal obesity, dyslipidemia, hypertension, and hyperglycemia is a strong predictor of cardiovascular diseases (CVD) morbidity and mortality [1-3]. Although the increase in risk begins with the presence of even one of the above metabolic abnormalities, abdominal obesity and disturbed insulin are among crucial risk factors [3-5]. According to the national representative data prevalence of obesity, abdominal obesity and Mets among Polish adults aged 20-74 years reached about 20.3%, 33.1%

and 21%, respectively, with substantial differences within sex and age groups. Prevalence of MetS in adults aged 20-60 years is significantly higher in men than in women (20% and 14%, respectively) [6,7].

Excessive weight and body mass index contribute substantially to the metabolic risk, but data on long-term patterns of weight change in relation to the development of MetS are scarce [8,9]. Several previous studies were based partly on self-reported weight recall, limited to young adults, or with a relatively short time of observation [10-13]. Recently published observation of Bot et al. (2010) covered a period of 16 years, but did not determine the relationship between weight change and

MetS due to lack of triglycerides [14]. Moreover, although weight gain is strongly associated with MetS or its components, the influence of stable weight or moderate weight loss on the metabolic is less well known [15,16]. In the available literature we have not found any research that has prospectively evaluated the effect of objectively measured weight change on MetS parameters in Central and Eastern Europe. Our previous report on the relationship between weight change and CVD risk has not concerned the risk of MetS and its components [17].

Hence, the purpose of this study was to evaluate prospectively the relationship between average weight change (both gain and loss) on the risk of MetS among metabolically healthy normal weight and overweight/ obese men. Considering an important role of lifestyle habits in the development of the MetS and its components, the secondary aim was to examine the impact of physical activity level on the metabolic risk in the context of weight change.

2. Materials and methods

Of the initial 977 adult male volunteers, who attended the Healthy Men Clinic and the Department of Preventive Medicine of the Medical University of Lodz (Poland) in the period 1980-1988, data of 324 men aged 18-64 years (mean 40.1 ± 9.3 years at baseline) were used in the present analysis. Patients were eligible if they participated in at least two examinations (baseline and final), had body mass index (BMI) > 18.5 kg/m^2 , were free from chronic diseases and treatment, abdominal obesity and MetS at baseline. Follow-up visits were planned once a year between the baseline (1980-1988) and final (2003-2005) examination. The mean observation time was 17.9 years with the mean age of the subjects 40.1 ± 9.3 and 57.8 ± 8.6 years at baseline and final visit, respectively.

The subjects of the study were Caucasians, predominantly (beyond 85%) white collar workers with university or secondary education level, whose occupational activity low. All participants provided details of their sociodemographic status, lifestyle behaviours (smoking status, diet, physical activity level) and medical history during a detailed questionnaire interview.

Data on leisure-time physical activity (LTPA) were collected during the interview prior to physical examination. The participants were asked about their habitual type of activity, average frequency as well as time per week spent performing such activity. A metabolic equivalents (MET) value was assigned to each sports activity based on the compendium of physical activities [18]. Time spent per week performing each activity was

multiplied by the MET value of the activity to obtain total MET-minutes per week. A combined MET-minutes/week score was calculated by multiplying the weekly frequency, duration, and intensity of the activity.

Measurement of biological factors was performed by trained personnel and comprised height, weight, waist circumference, seated blood pressure, fasting serum glucose and lipid concentrations, and exercise test during each visit to the clinic. Body weight was measured to the nearest 100 g on calibrated scales (in light indoor clothes and without shoes). Height was measured with a stadiometer (without shoes) to the nearest 0.5 cm. Waist circumference was measured with a tape measure at the middle of the distance between the lowest rib and the iliac crest (in underwear, standing position) to the nearest 0.5 cm. BMI was calculated as weight (kilograms) divided by square of height (meters). Obesity was defined as BMI ≥ 30 kg/m². Abdominal obesity was defined as waist circumference ≥ 102 cm. Systolic and diastolic blood pressure at rest (at least 5 min) was measured in a supine position on the left arm using a mercury sphygmomanometer. The mean value of two measurements taken at 5 min intervals was used for the analysis.

Venous blood samples were collected after a 12 h fast. Fasting plasma glucose (FPG) was assessed by standardized enzymatic method using an enzymatic colorimetric method with glucose oxidize. HDL-C was measured by enzymatic method after precipitation and triglycerides (TG) were assayed using an enzymatic colorimetric tests with glycerol phosphate oxodize. All the measurements were performed in the laboratory of the Department of Preventive Medicine of the Medical University of Lodz (Poland). Serum triglycerides were not measured during the baseline visit.

According to the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP Adult Treatment Panel III) criteria the metabolic syndrome was diagnosed in men with at least 3 of the following factors: abdominal obesity (waist circumference > 102 cm), elevated blood pressure (BP)(≥ 130/80 mmHg), elevated TG (≥ 1.7 mmol/L), low HDL-C (< 1.03 mmol/L), elevated FPG (≥ 5.6 mmol/L) [1].

The graded submaximal exercise test was carried out on a Monark type 818E (Stockholm, Sweden) bicycle ergometer with 30 W increments every 3 min to achieve at least 85% of maximal age-predicted heart rate (220-age).

Weight change was defined as the absolute change between the baseline and final examination. Based on the distribution of weight change over the whole observation period we divided weight change patterns into 3 categories: weight gain (≥ 2.5 kg), stable weight (> -2.5 kg)

and < 2.5 kg), and weight loss (≤ -2.5 kg). We evaluated absolute rather than percentage weight change in order to make clinical interpretation more clear and useful.

2.1 Statistical analysis

All statistical analysis were performed with STATIS-TICA Windows XP version 8.0. Descriptive data are expressed as mean values (± SD) or frequency (percentage). Differences between the baseline and the final means and percents were assessed by using paired t-test and McNemar test, respectively. Differences in the mean values of metabolic risk factors according to the weight change pattern were tested by Kruskal- Wallis and Mann-Whitney tests. Crude and adjusted relative risks (RR) and 95% confidence intervals (CI) of metabolic disorders corresponding to the weight change category were calculated by the logistic regression analysis; the stable weight group was used as referent. When comparing differences between studied groups, adjustments for age, smoking, education, alcohol consumption, LTPA and VO_{2max} were included. A p value <0.05 was considered statistically significant.

3. Results

Table 1 presents baseline and final characteristics of the study population. The current analysis included 324 men aged 18-64 years (33.9% aged 18-34 years) with a mean age of 40.1 ± 9.3 years. At baseline 46.9% of all participants were normal-weight (BMI 18.5-24.9 kg/m²),

51.9% were overweight (BMI 25-29.9 kg/m²), and 1.2% were obese (BMI ≥ 30 kg/m²). All the participants were free of abdominal obesity and the metabolic syndrome according to the NCEP ATP III 2005 criteria at baseline.

After a mean of 17.9 ± 8.1 years of follow-up, the average BMI of the cohort increased to 27.9 ± 6.5 kg/m2 and the average weight increased by 6.1%. Prevalence of normal weight decreased to 42.3%, overweight decreased to 36.6%, and obesity increased to 21.1%, respectively. The increase in the prevalence of excessive weight was more evident among baseline overweight subjects. Furthermore, 5.2% of initially overweight men lost weight enough to be classified as normal weight at final examination; while 15.8% of initially normal weight subjects became finally overweight (12.5%) or obese (3.2%). As presented in Table 1 most of the analyzed metabolic parameters worsened over follow-up, except for HDL-C. Due to lack of serum TG the precise change in the number of MetS components over time was not possible to assess. The most common MetS parameter was elevated BP (38.5%), followed by high TG (24.8%), abdominal obesity (22.8%), elevated FPG (21.9%), and low HDL-C (12.8%).

Table 2 reveals that increasing weight gain correlates with worsening of the analyzed MetS parameters independently of the baseline BMI. However, the observed relationships were less evident among baseline normal-weight subjects, especially in systolic BP (p = 0.049) and FPG (p = 0.051). Over almost 18 years of follow-up, 73 cases of MetS (22.5%) were noted, predominantly among men who gained weight (15.6%)

Table 1. Characteristics of the study participants at baseline and 17 year follow-up examination, by baseline body mass index (BMI) status.

	I	BMI < 25 kg/m²			$BMI \geq 25 \ kg/m^2$		
	n = 152			n = 172			
	At baseline	At follow-up	р	At baseline	At follow-up	р	
Age (year)	39.9 ± 10.4	57.6 ± 11.1	< 0.001	41.9 ± 8.1	57.9 ± 9.6	< 0.001	
Weight (kg)	70.2 ± 6.6	74.7 ± 7.8	< 0.05	83.6 ± 9.3	89.1 ± 10.6	< 0.01	
BMI (kg/m²)	23.0 ± 1.4	25.8 ± 4.4	< 0.01	27.2 ± 2.2	28.9 ± 5.6	< 0.01	
WC (cm)	85.2 ± 9.2	89.7 ± 8.7	< 0.01	88.5 ± 7.7	98 ± 10.0	< 0.001	
FPG (mmol/l)	4.51 ± 0.8	5.29 ± 0.6	< 0.05	4.69 ± 1.4	5.72 ± 1.2	< 0.05	
TG (mmol/l)	NA	1.24 ± 0.5	-	NA	1.57 ± 0.87	-	
HDL-C (mmol/l)	1.32 ± 0.5	1.49 ± 0.2	< 0.01	1.29 ± 0.9	1.39 ± 0.82	< 0.05	
SBP (mmHg)	126.5 ± 15.3	129.5 ± 18.1	NS	128.0 ± 13.6	135.6 ± 18.6	< 0.05	
DBP (mmHg)	83.7 ± 9.9	84.6 ± 9.8	NS	84.5 ± 9.8	85.9 ± 10.3	NS	
Current smokers	37.3 %	19.3 %	< 0.001	33.7 %	14.5 %	< 0.001	
Alcohol units/weeek	3.1 ± 3.6	5.6 ± 12.2	< 0.001	3.3 ± 4.1	6.5 ± 13.1	< 0.001	
LTPA	1929.6 ± 2080.7	1897.3 ± 2160.7		1502.9 ± 2760.7	1490.1 ± 1874.7		
(MET/min/week)			NS			NS	
VO _{2max} (ml/kg/min)	40.8 ± 10.9	34.1 ± 8.8	< 0.01	35.4 ± 9.3	27.8 ± 7.1	< 0.001	
MetS	-	10.2 %	-	-	33.1%	-	

Abbreviations: BMI – body mass index; WC – waist circumference; FPG – fasting plasma glucose; TG – triglycerides; HDL-C – high density lipoprotein cholesterol; SBP – systolic blood pressure; DBP – diastolic blood pressure; MetS – metabolic syndrome; LTPA – leisure-time physical activity; VO2max - maximal oxygen uptake; MET – metabolic equivalent.

Table 2. Odds ratios (OR) and 95% confidence intervals (CI) of gastric cancer according to tertile of intake of selected minerals.

	Weight loss	Stable weight	Weight gain	
	≤ -2.5 kg	> -2.5 and < 2.5 kg	> 2.5 kg	р
BMI $< 25 \text{ kg/m}^2$				
n	17	39	96	
WC (cm)	85.2 ± 8.5	87.4 ± 9.4	93.2 ± 7.9	p < 0.05
FPG (mmol/l)	5.23 ± 0.5	5.27 ± 1.4	5.30 ± 1.3	p= 0.051
TG (mmol/l)	1.19 ± 0.8	1.23 ± 0.4	1.26 ± 0.9	p< 0.05
HDL-C (mmol/l)	1.53 ± 0.1	1.46 ± 0.2	1.47 ± 0.1	P < 0.05
SBP (mmHg)	127.9 ± 17.5	128.7 ± 19.5	133.2 ± 21.6	p = 0.049
DBP (mmHg)	81.1 ± 9.4	84.2 ± 9.5	85.3 ± 12.1	p > 0.05
LTPA (MET/min/week)	2301.3 ± 2200.7	1987.3 ± 1150.9	1727.3 ± 1960.2	p < 0.01
BMI ≥ 25 kg/m²				
n	32	43	97	
WC (cm)	92.4 ± 9.5	97.3 ± 6.3	101.5± 11.1	p < 0.001
FPG (mmol/l)	5.39 ± 0.7	5.61 ± 1.5	6.26 ± 1.6	p < 0.01
TG (mmol/l)	1.39 ± 0.5	1.51 ± 0.3	1.6± 1.5	p < 0.01
HDL-C (mmol/l)	1.44 ± 0.6	1.39 ± 0.7	1.29± 0.2	p < 0.001
SBP (mmHg)	133.8 ± 19.5	133.2 ± 22.7	139.0 ± 19.7	p < 0.05
DBP (mmHg)	83.7 ± 10.5	85.0 ± 8.5	86.2 ± 9.5	p < 0.05
LTPA (MET/min/week)	2103.6 ± 2002.7	1810.3 ± 1970.1	1280.6 ± 2100.6	p < 0.001

Abbreviations: BMI – body mass index; WC – waist circumference; FPG – fasting plasma glucose; TG – triglycerides; HDL-C – high density lipoprotein cholesterol; SBP – systolic blood pressure; DBP – diastolic blood pressure; LTPA – leisure-time physical activity; MET – metabolic equivalent

among normal weight and 45.4% among overweight at baseline). Importantly, only one case of MetS developed in normal weight men who lost or maintained weight.

Table 3 shows the adjusted RR (95% CI) of the MetS and its components in relation to weight change patterns and BMI status at baseline. Increase in weight was the strongest predictor of abdominal obesity in both groups as well high TG and FPG among overweight at baseline. Certain trend in increasing the risk of elevated BP in relation to weight gain was noted, but statistical significance was not reached (p = 0.057 among over-

weight). Weight loss had a protective effect on the MetS components (except for elevated BP) in both studied cohorts.

Further analysis was conducted in order to examine the relationship between LTPA and the metabolic risk. Among all subjects with baseline BMI \geq 25 kg/m², energy expenditure (EE) > 2000 MET/min/week correlated with significantly lower risk of low HDL-C (RR 0.41; p<0.01), high TG (RR 0.39; p<0.01) and MetS (RR 0.36; p<0.01) referring to LTPA with EE < 1000 MET/min/week. The associations were even stronger

Table 3. Association of weight change over 17 year follow-up with the MetS and its components, by baseline BMI status.

	Weight loss ≤ -2.5 kg		Stable weight	Weight gain > 2.5 kg	
			> -2.5 and < 2.5 kg		
	RR	95% CI	RR 95% CI	RR	95% CI
BMI < 25 kg/m ²					
Central obesity	0.49	0.11-1.48*	1.0	4.71	2.91-7.38***
High FPG	0.59	0.22-0.83*	1.0	1.37	0.52-3.78*
High TG	0.57	0.17-0.55**	1.0	2.24	1.2-5.85**
Low HDL-C	0.51	0.15-0.66*	1.0	1.51	0.85-2.24
High BP	0.76	0.47-1.23	1.0	1.26	0.47-4.23
MetS		-	1.0	2.55	0.45-1.05*
BMI ≥ 25 kg/m ²					
Central obesity	0.29	0.08-1.28**	1.0	9.51	3.65-18.75***
High FPG	0.48	0.25-0.93*	1.0	2.78	1.25-6.65**
High TG	0.31	0.28-0.77**	1.0	4.31	1.58-6.22**
Low HDL-C	0.62	0.33-1.3*	1.0	1.92	0.99-3.4*
High BP	0.73	0.32-1.02	1.0	1.73	1.42-5.02
MetS	0.55	0.45-1.05*	1.0	3.68	1.17-8.09**

^a Relative risk (RR) and 95% confidence intervals (95% CI) adjusted for age, education, smoking, physical activity level and VO_{2max} *p<0.05 ** p<0.01 *** p<0.001

among baseline overweight who lost weight in the risk of abdominal obesity (RR 0.22; p<0.01); RR of low HDL-C (RR 0.34; p<0.001), high TG (RR 0.27; p<0.001) and MetS (RR 0.21; p<0.001). In baseline lean men group high LTPA was protective for the risk of low HDL-C (RR 0.46; p<0.05), elevated FPG (RR 0.48; p<0.05) and the MetS (RR 0.27; p<0.05). Mean EE 1000-2000 MET/min/ week was significantly correlated only with the lower risk of low HDL-C in baseline overweight men who increased weight over time.

4. Discussion

This prospective long-term study showed that weight change over a 17-year period was a significant predictor of metabolic disorders in adult men without abdominal obesity and MetS at baseline. Weight gain (≥ 2.5 kg) was strongly associated with worsening of MetS parameters in comparison to those who remained relatively stable in weight. Furthermore, the risk of development of the MetS was about 3-fold higher in the weight gain group after adjustment for age, education, smoking and LTPA. Except for elevated BP, weight loss was protective against MetS disorders. However, several discrepancies in modifying the risk were observed according to baseline body mass index as well as weekly energy expenditure.

In our study, the MetS was incident in 22.7% after a follow-up, with a markedly higher incidence among baseline overweight who remained stable or increased their weight. MetS was accompanied by 3.08% incident of new type 2 diabetes (data not shown). Similar incidence rate of MetS (23%) was presented by Onat et al. in a representative Turkish sample followed for 5.9 years [19]. Lower prevalence of MetS was noted in a 3-year observation of Iranian men (18.4%) and a 4-year cohort of Taiwanese men (17.5%) [13,19]. The incidence of MetS ranged between 1.9% (fluctuating weight) and 20.5% (increased weight) among young adult men in the CARDIA study [11]. Hypertensive participants of the European Lacidipine Study on Atherosclerosis MetS developed MetS in 21.4% over a 3.7 years follow-up [21]. The present analysis demonstrates that metabolic impact of weight change was different among baseline normal-weight and overweight subjects. Weight gain was positively related with all MetS components and negatively with HDL-C. The obtained correlations are comparable to several previous long-term observations with a follow-up period of 2-16 years. Strong linear relationship with weight gain and worsening of all MetS parameters was found by Hilier et al. [12]. Bot et al. (2010) showed negative correlation with HDL-C and unfavorable changes in WC, systolic and diastolic BP, and FPG level related to weight gain (≥ 2.5 kg) over a 16-year follow-up [14].

Most, but not all, studies demonstrated the positive association between weight gain and FPG. In our study no significant differences were noted in the mean values of FPG across weight change categories among normal-weight subjects. However, a strong relationship between weight gain and FPG was found in overweight men.

Contrary to other authors' findings, we did not find statistically significant association between the weight gain and the risk of elevated BP according to the NCEP ATP III definition. However, additional analysis of the relative risk of hypertension (defined as BP \geq 140/90 mmHg) revealed significant relationship among men who increased weight (data not shown).

Long-term increase in weight was most strongly related to waist circumference and the risk of abdominal obesity which is in line with the findings of other authors [12,14].

Most of the previous studies demonstrated that a modification of metabolic risk resulting from weight change patterns is independent of baseline BMI. In the CARDIA study, incidence of MetS in a 15-year prospective observation was similar in normal weight and overweight young adults [11]. Although no significant relationships were found in the above study, baseline BMI was not unimportant and initially overweight young adults were more likely to continue increase their BMI. In order to compare our results with the findings of Lloyd-Jones *et al.*, we conducted an analysis among men aged 18-30 in our cohort (n=101). The obtained results revealed less evident differences between normal weight and overweight subjects, but the direction and significance of correlations remained.

A number of studies have reported that regular physical activity may substantially reduce the prevalence of MetS or its components [22-26]. Our previous report demonstrated that energy expenditure was a strong predictor of long-term metabolic risk. However, those data were not correlated with baseline BMI and metabolic status [26]. Energy expenditure was most strongly related with the metabolic risk among baseline overweight men who lost weight. Evident associations were found for lowering the risk of dyslipidaemia and MetS in men with the highest physical activity level (EE above 2000 MET/min/week; p<0.001). Our observations confirm findings of other authors that regular physical exercises favorably affect individual metabolic components as well as MetS itself. However, only EE > 2000 MET/min/week may result in reducing metabolic risk.

Reducing weight or maintaining BMI in a healthy range, although hard to achieve, seems to be crucial

in the context of future cardiovascular events. Weight loss has been shown to have beneficial influence on cardiovascular and metabolic risk in various populations [13,14, 27-29]. The results of our study are in line with findings of other authors indicating the protective effect of weight loss, especially among overweight individuals. In contrast to previous studies, recently published findings of Ärnlöv *et al.* demonstrate that overweight and obese subjects without MetS also had an increased risk of CVD events and deaths in a 30-year observation [3]. Therefore, so-called "metabolically healthy obese" do not represent benign conditions and shall be also among public health priorities.

Several limitations of the study should be acknowledged. Firstly, due to not have a large sample size, our study cohort does represent all Polish young and middle-aged men. Participants of this study declared much higher level of PA in comparison to national statistics [29]. However, the whole studied group was quite uniform according to ethnicity, educational level, income, dietary patterns and alcohol consumption. Also baseline metabolic profile is comparable between the normalweight and overweight men (except for anthropometrics). Important limitation is also a lack of triglycerides at baseline examination enabling to assess full metabolic profile of the study participants. Well known limitation is also the possibility of recall bias due to self-reported data on physical activity. No specific repeatability or validity tests for our questionnaire were performed, but similar methods have been already used in a large number of studies in Poland [29,30]. The precise comparison of the present data with other studies is difficult due to important differences in population characteristics, length of follow-up and the way of expression of weight change. As no standard classifications of weight change have been elaborated we decided to use the cut-off value of weight change on 2.5 kg. Similar method has already been used by Bot *et al.* [14].

Despite the above limitations, these data indicate that metabolic syndrome is significantly associated with weight change in adult men. Important strengths of this study are long period of observation (17.9 years) and objective measurements of weight made by trained personnel. Furthermore, including to the analysis only subjects without abdominal obesity and MetS at baseline enabled to determine the incidence of these disorders in longitudinal observation.

Concluding, there is a positive association between weight gain and metabolic risk in adult men. Weight loss among overweight or obese and maintenance of stable weight among normal-weight individuals have beneficial effect on the metabolic disorders and MetS itself. High LTPA level with energy expenditure above 2000 MET/min/week is a strong protective factor against development of MetS and its components. Reducing weight in overweight and obese as well as maintaining stable weight in lean subjects shall be a priority in the context of health promotion and prevention of future cardiovascular events and deaths.

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