

# Hormonal assessment and body composition in young males with metabolic syndrome

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## ABSTRACT

**Aim:** To determine the relationship between body composition and hormonal levels in young men with metabolic syndrome.

**Materials and Methods:** 123 males with a mean age of  $24.1 \pm 4.3$  years (33 with metabolic syndrome (MS group) and 90 healthy physically active men (control group) were recruited at the study of body composition and hormone status. The total testosterone, cortisol, and insulin in blood serum by ELISA, the body weight (kg), lean body mass (kg) and fat mass (kg, %) by bioimpedance analysis method were investigated.

**Results:** It was established the significant difference the mean value of body composition (body weight, lean body weight, fat body mass (kg, %), testosterone, cortisol insulin, and glucose concentration between MS group and control group.

**Conclusions:** A present study established the significant correlation of testosterone, insulin, and glucose concentration with fat body mass in all participants (MS and control groups). The negative effect of overweight (BMI > 25; FBM > 18 %) and obesity (BMI > 30; FBM > 25 %) for testosterone concentration was determined due to an increase of FBM > 20 % and insulin increasing > 9,0  $\mu$ U/l.

**KEY WORDS:** metabolic syndrome, testosterone, insulin, fat body mass, body mass index

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## INTRODUCTION

### METABOLIC SYNDROME IN YOUNG MEN

Metabolic syndrome is a group of conditions, which together increase the risk of developing atherosclerotic cardiovascular disease, insulin resistance and diabetes mellitus, and vascular and neurological diseases. Metabolic syndrome is associated with high all-cause mortality as well as mortality due to cardiovascular disease [1]. The main causes of metabolic syndrome are abdominal obesity, high blood pressure, diabetes, and dyslipidemia. With an increase in the obese population worldwide, the prevalence of metabolic syndrome is increasing [2, 3]. The global prevalence of metabolic syndrome varies slightly depending on the definition of each component and ranges from 24.3% to 45.5% [4, 5].

Since metabolic syndrome is a cluster of factors, it is difficult to manage and treat this condition compared to other diseases. The pathogenesis of metabolic syndrome can be described by a complex mechanism. Overweight and obesity are central to the development of metabolic syndrome, predisposing to hypertension, insulin resistance, and dyslipidemia, all of which are risk factors of metabolic syndrome. Physical inactivity and fatty food intake are major causes of obesity [6, 7]. There is substantial heterogeneity by sex and ethnicity

in the prevalence of the metabolic syndrome [7, 8]. The syndrome is generally more common amongst young men compared with women and the prevalence tends to increase with age [9, 10].

### THE SIGNIFICANCE OF FAT BODY MASS IN YOUNG MEN WITH METABOLIC SYNDROME

The fat body mass or adiposity in young men with metabolic syndrome is an important factor to consider because it is strongly associated with the development and progression of metabolic disorders. Several studies have shown that increased adiposity in young men is a significant risk factor for the development of metabolic syndrome. Excess body fat, particularly abdominal fat, is associated with insulin resistance, which can lead to high blood sugar levels and eventually type 2 diabetes. Additionally, excessive fat deposition in the liver and other organs can contribute to the development of non-alcoholic fatty liver disease (NAFLD), which is closely linked to metabolic syndrome [11-13].

Furthermore, increased adiposity is associated with chronic low-grade inflammation, which can contribute to the development of atherosclerosis and other cardiovascular diseases. This chronic inflammation can

also impair the function of the endothelium, which is the inner lining of blood vessels, leading to endothelial dysfunction and increased risk of heart attacks and strokes [14].

In conclusion, the fat body mass in young men with metabolic syndrome is a significant factor to consider as it is closely associated with the development and progression of metabolic disorders, including insulin resistance, type 2 diabetes, NAFLD, chronic inflammation, and cardiovascular diseases. Effective strategies to manage adiposity through lifestyle modifications, such as dietary changes and physical activity, can improve metabolic health outcomes and reduce the risk of cardiovascular disease in young men with metabolic syndrome [13 - 29].

### INSULIN AND ITS INTERRELATIONS WITH CORTISOL AND TESTOSTERONE IN YOUNG MEN WITH METABOLIC SYNDROME

In young men with metabolic syndrome, the relationships between insulin, testosterone, and cortisol are complex and can have significant effects on their metabolic health.

Insulin resistance is a key feature of metabolic syndrome and can contribute to the development of low testosterone levels in young men. Insulin resistance leads to an increase in insulin levels, which can inhibit the production of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in the pituitary gland. These hormones are necessary for the production of testosterone by the testes. Therefore, insulin resistance can contribute to a decrease in testosterone levels in young men with metabolic syndrome [15, 23].

On the other hand, low testosterone levels can also contribute to the development of metabolic syndrome. Testosterone has been shown to improve insulin sensitivity by increasing glucose uptake by the muscles and improving insulin signaling pathways. Therefore, low testosterone levels in young men with metabolic syndrome may lead to insulin resistance and the development of metabolic disorders [16-18].

Cortisol levels can also play a role in the relationship between insulin and testosterone in young men with metabolic syndrome. High cortisol levels have been associated with a decrease in testosterone levels in men with metabolic syndrome, as cortisol can inhibit the production of LH. Additionally, high cortisol levels can contribute to insulin resistance, as cortisol promotes the breakdown of glucose for energy, leading to an increase in blood sugar levels and a decrease in insulin sensitivity [19, 20].

It is known that metabolic syndrome at men is correlated with testosterone level. The studies (Tsai *et al.* 2000) have shown the possible association of low testosterone levels with obesity, insulin resistance and an adverse lipid profile in men. Conversely in men with metabolic syndrome and type 2 diabetes have a high prevalence of hypogonadism. Metabolic syndrome and low testosterone status are both independently associated with increased all-cause and cardiovascular mortality [21]. Men are at increased risk of developing coronary artery disease earlier in life compared with women. The reason for this bias toward males is still poorly understood. Several longitudinal studies have shown that low testosterone is an independent risk factor for the development of diabetes and metabolic syndrome [13, 22]. Baseline testosterone levels correlate inversely with the accumulation of central fat but not other fat deposits in a cohort of 110 men (Tsai *et al.* 2000). Three other studies, the Massachusetts Male Aging Study (MMAS) (Stellato *et al.* 2000), the Multiple Risk Factor Intervention Trial (MRFIT) (Haffner *et al.* 1996) and Rancho Bernardo (Oh *et al.* 2002), showed an inverse correlation between baseline testosterone and the future development of diabetes. A Finnish study showed that low baseline testosterone and SHBG predicted metabolic syndrome and diabetes after a 11-year follow up (Laaksonen *et al.* 2004). The mechanisms linking testosterone with insulin resistance and type 2 diabetes are still not fully understood. Although testosterone deficiency leads to increased fat deposition and this would result in increasing insulin resistance, it may not explain the total action on insulin sensitivity. For example, one study which assessed insulin resistance by hyperinsulinaemic/euglycaemic clamps in 60 men with a range of glucose tolerance from normal to diabetic levels (Pitteloud *et al.* 2005). Their findings confirmed an inverse relation between total testosterone and insulin resistance. From muscle biopsies they showed that low testosterone impairs mitochondrial oxidative phosphorylation. As up to 70% of the body's insulin sensitivity can be accounted for by muscle this tissue may develop reduced insulin sensitivity in the hypogonadal state sufficient to contribute in part to the overall state of insulin resistance [22, 24].

Furthermore, chronic stress, which can lead to an increase in cortisol levels, is common in young men with metabolic syndrome. Chronic stress can also lead to an increase in appetite and food intake, particularly for high-calorie, high-fat foods, leading to weight gain and further exacerbating the relationship between insulin resistance, testosterone levels, and cortisol levels [19].

In conclusion, insulin, testosterone, and cortisol levels are interrelated and can influence each other in

**Table 1.** Instrumental and laboratory characteristics of metabolic syndrome (MS) group and control ( $\bar{X} \pm \bar{S}$ )

Indicators		MS, n=36	Control, n=91	P
	TBW, kg	105,42±16,93	78,15±15,54	0,05
FBM	%	28,40±5,55	10,92 ± 4,12	0,05
	kg	29,93 ± 7,37	8,93 ± 5,38	0,05
	BMI, kg/m <sup>2</sup>	32,85± 4,42	23,75 ±3,18	0,05
	LBW, kg	77,02±17,93	68,89±11,80	0,05
	Glucose, mmol/l	5,31±0,92	4,54 ±0,43	0,05
	HOMA-IR index	3,18±1,59	0,99 ±0,79	0,05

Note: TBW - total body weight; FBM - fat body mass, BMI – body mass index; LBW – lean body weight; HOMA-IR - homeostatic model assessment of insulin resistance.  $p < 0.05$  – statistically significant differences.

**Table 2.** Hormonal status of metabolic syndrome (MS) group and control ( $\bar{X} \pm \bar{S}$ )

Indicators	MS, n=36	Control, n=91	P
Testosterone, nmol/l	15,29±4,83	25,13±11,13	0,05
Cortisol, nmol/l	303,24±76,41	638,41±317,99	0,05
Insulin, $\mu$ U/l	13,27±6,26	5,52±3,77	0,05

Note: the average value  $\pm$  SD is given for the case of the normal distribution law.  $p < 0.05$  – statistically significant differences.

complex ways in young men with metabolic syndrome. Lifestyle modifications, such as weight loss, regular exercise, acute influence of physical loads for hormone content [25-26] and a healthy diet, can help improve insulin sensitivity and testosterone levels while reducing cortisol levels and overall stress.

## AIM

The aim of this study was to determine the relationship between body composition and hormonal levels in young men with metabolic syndrome.

## MATERIALS AND METHODS

In line with the study's objectives, we enrolled 123 male participants with an average age of  $24.1 \pm 4.3$  years. This cohort consisted of 33 men with both metabolic syndrome and a sedentary lifestyle (referred to as the «MS group» with a mean age of  $23.3 \pm 4.8$  years) and 90 healthy, physically active men (referred to as the «control group» with a mean age of  $24.4 \pm 4.6$  years). The individuals in the control group engaged in an average of 4 hours of physical activity per day, six days a week, resulting in an average of  $24.87 \pm 2.1$  hours of weekly physical activity. Our study included patients who were examined at the consultative and diagnostic departments of the "V.P. Komisarenko Institute of Endocrinology and Metabolism of the NAMS of Ukraine" and the Scientific Research Institute of Physical Culture and Sport. Before undergoing examination, all patients were informed about the research protocol, which adhered

to the ethical principles outlined in the Helsinki Declaration of the World Medical Association, the Council of Europe's Convention on human rights and biomedicine, and the legislation of Ukraine. They provided their informed consent for participation in the study and did not receive any medications.

To identify signs of metabolic syndrome in the participants, we utilized the IDF criteria from 2005. In our controlled clinical study of metabolic syndrome patients, we employed elements of typological sampling (stratification randomization). Inclusion criteria: male gender, age 18-45 years old, absence of any earlier determined diseases and prescribed medications. All patients underwent standard clinical and laboratory assessments, including hormonal blood parameters (total testosterone, cortisol, and insulin). Total body weight (TBW, kg), fat body mass (FBM, kg, %), lean body weight (LBW, kg) were determined using bioelectric impedance analysis with InBody 770 from the Republic of Korea. The HOMA IR indicator was calculated using the formula:  $\text{HOMA IR} = \text{fasting blood glucose (mmol/l)} \times \text{fasting blood insulin } (\mu\text{U/l}) / 22.5$  [Matthew D. R., 1985]. We assessed the degree of general obesity using BMI indicators in accordance with the recommendations of the WHO (1997) and the International Diabetes Federation (2005). A BMI within the normal range was defined as less than  $24 \text{ kg/m}^2$ , and waist circumference (WC) was considered normal if it was less than 94 cm in men. The HOMA IR indicator, which should not exceed 2.77, was also used for evaluation. To determine the concentration of testosterone, cortisol, and insulin in blood serum, we

**Table 3.** Body composition and biochemical parameters at men with different range of BMI ( $X \pm \sigma$ )

Indicators	MS, n=33		Control, n=91		
	OB, n=23	OW, n=10	OB, n=5	OW, n=22	NW, n=65
TBW, kg	109,50±18,30	96,00±7,50*	120,20±15,54 <sup>a</sup>	94,40±10,00 <sup>#q</sup>	70,80±8,30
FBM, %	28,20±5,60	28,90 ± 5,80	19,80 ± 7,80 <sup>a</sup>	13,40 ± 3,60 <sup>#q</sup>	9,60 ± 2,80
FBM, kg	30,90 ± 8,00	27,70 ± 5,50	24,10 ± 10,00 <sup>a</sup>	12,80 ± 3,50 <sup>#q</sup>	6,80 ± 2,40
BMI, kg/m <sup>2</sup>	34,60± 4,20	28,80 ± 1,20*	33,30 ± 2,20 <sup>a</sup>	27,20 ± 1,50 <sup>#q</sup>	22,20 ± 2,40
LBW,kg	81,40±9,00	67,10±10,00 *	96,20±8,20 <sup>a</sup>	80,90±7,40 <sup>#q</sup>	63,60±7,40
Glucose, mmol/l	5,26±0,99	5,42 ± 0,78	4,82 ± 0,41	4,63 ± 0,48	4,49 ± 0,41
HOMA-IR index	3,10±1,71	3,50±1,29	1,80 ± 0,97 <sup>a</sup>	1,10 ± 0,62 <sup>#</sup>	0,90 ± 0,78
Testosterone, nmol/l	16,20±5,10	13,30±3,60	21,60±5,80	23,60±11,20	25,70±11,30
Cortisol, nmol/l	301,20±83,20	308,00 ± 61,60	541,30 ± 192,70	716,70 ± 318,20	621,30 ± 322,90
Insulin, μIU/l	12,80 ± 6,70	14,40 ± 5,20	8,60 ± 4,40 <sup>a</sup>	6,70 ± 3,90 <sup>q</sup>	5,00 ± 3,60

Note: the average value ± SD is given for the case of the normal distribution law;

\* - statistically significant differences ( $p < 0.05$ ) between obese and overweight group in men with metabolic syndrome;

a - statistically significant differences ( $p < 0.05$ ) between obese and normal weight men in control group;

# - statistically significant differences ( $p < 0.05$ ) between obese and overweight men in control group;

q - statistically significant differences ( $p < 0.05$ ) between overweight and normal weight men in control group.

employed ChemWell enzyme-linked immunosorbent assay equipment from Awareness Technology (USA) with the use of AccuBind ELISA test systems from Monobind Inc. (USA). Blood sampling and subsequent processing followed the provided instructions. Our study excluded individuals with stable heart rhythm disorders (such as atrial fibrillation or frequent ventricular extrasystoles), clinically diagnosed heart failure, severe kidney or liver dysfunction, drug or alcohol addiction, and those who had experienced acute inflammatory diseases within the previous month.

All statistical analyses were performed using «STATISTICA 12» with a significance level set at 0.05 unless stated to the contrary. One-way ANOVA, correlation analysis (Spearman correlation) was used for determine the significant of the hormone status for BMI and percent body fat in young men with metabolic syndrome and physically active healthy. The Duncan method (post hoc analysis) was used for differences estimation between control and MS groups. The correspondence of the sample to the normal distribution was checked for asymmetries and excesses, which indicated the proximity of the distribution to the normal curve.

## RESULTS

Firstly, patients were divided into 2 groups: 1 – young men with metabolic syndrome and 2- control one (physically active and healthy individuals). Then we compared the patients' main instrumental and lab-

oratory characteristics (Table 1). The differences in hormonal indicators are demonstrated in table 2.

We have established statistically significant differences in all instrumental and laboratory parameters of the studied groups. So, total body weight (TBW, kg) of control group was 25.71% less; fat body mass (FBM, %) was 61.65% less and insulin levels were 58.40% lower than in men with metabolic syndrome, while cortisol and testosterone levels were 52.51% and 40.00% higher.

Thus, we have found significant differences in all characteristics of two groups. Secondly, to consider detailed differences between groups, we decided to divide each group into three sub-groups depending on their BMI into three main groups: normal weight men with BMI = 18,5-24,9 kg/m<sup>2</sup> (NW), overweight ones with BMI= 25,0 – 29,9 kg/m<sup>2</sup> (OW) and obesity ones with BMI above 30,0 kg/m<sup>2</sup> (OB) (Table 3).

The following results were obtained. Firstly, in the group with metabolic syndrome, there was a statistically significant difference only in terms of TBW and LBW, and in the obese group this indicator was 17.28% higher than in the overweight group. The FBM and hormones level in metabolic syndrome OW and OB groups don't differ (Table 3).

Secondly, in the control group, statistically significant differences in body composition assessment indicators were established both between groups with obesity and overweight, and when comparing these groups with normal body weight.

In the men of control group with BMI > 30 (OB subgroup), FBM (%) was 51.52% and 32.32% higher than

**Table 4.** Body composition and biochemical parameters at men with different range of BMI and FBM

Indicators	MS, n=33		Control, n=91		
	OB, n=12	OW, n=21	OB, n=2	OW, n=9	NW, n=80
TBW, kg	102,80±13,40	106,90±18,80	124,10±15,54 <sup>a</sup>	100,90±13,70 <sup>#q</sup>	75,20±12,50
FBM	%	34,00±3,80	25,20 ± 3,40 <sup>*</sup>	17,90 ± 2,00 <sup>#q</sup>	9,80 ± 2,60
	kg	34,90 ± 5,70	27,10 ± 6,70 <sup>*</sup>	33,60 ± 2,00 <sup>a</sup>	18,10 ± 3,50 <sup>#q</sup>
BMI, kg/m <sup>2</sup>	31,70± 3,70	33,50 ±4,80	35,50 ±0,20 <sup>a</sup>	28,30 ± 2,40 <sup>#q</sup>	23,10 ± 2,40
LBW, kg	68,80±14,30	81,70±14,63 <sup>*</sup>	90,50±11,80 <sup>a</sup>	82,80±10,90 <sup>q</sup>	67,00±10,50
Glucose, mmol/l	5,70±1,00	5,10 ±0,80	4,60 ±0,20	4,80 ±0,40 <sup>q</sup>	4,50 ±0,40
HOMA-IR index	3,90±1,60	2,80±1,40 <sup>*</sup>	1,50 ±1,30	1,50 ±0,90 <sup>q</sup>	0,90 ±0,70
Testosterone, nmol/l	13,00±2,70	16,60±5,30 <sup>*</sup>	21,10±15,54	24,50±12,30	25,20±11,10
Cortisol, nmol/l	288,70±61,20	311,60 ± 84,20	495,60 ± 200,00	703,50 ± 307,30	636,60 ± 321,40
Insulin, µU/l	15,50 ± 6,70	12,00 ± 5,80	7,20 ± 6,20	6,50 ± 4,00	5,40 ± 3,70

Note: the average value ± SD is given for the case of the normal distribution law.

\* - statistically significant differences (p< 0.05) between obese and overweight group in men with metabolic syndrome;

a - statistically significant differences (p< 0.05) between obese and normal weight men in control group;

# - statistically significant differences (p< 0.05) between obese and overweight men in control group;

q - statistically significant differences (p< 0.05) between overweight and normal weight men in control group.

that of men with normal and overweight, and LBW - by 33.89% and 15.90%, respectively.

It should be noted that the HOMA-IR index in the group of men with metabolic syndrome exceeded normal values (normal up to 2.7), while in the control group it was within the normal range. However, a statistically significant difference was obtained in the HOMA-IR index between control subgroups with obesity compared to overweight and normal body weight by 38.89% and 50.00%, respectively, against the background of a statistically significant difference in insulin levels established in these subgroups - by 22.09% and 41.86% respectively.

Thirdly, we decided to divide each group into three sub-groups depending on their BMI + FBM into three main groups: normal weight men with BMI= 18,5-24,9 kg/m<sup>2</sup> and FBM ≤ 18 %, overweight ones with BMI= 25,0 – 29,9 kg/m<sup>2</sup>, FBM 18,1 - 25 % and obesity ones with BMI above 30,0 kg/m<sup>2</sup>, FBM above 25 % (Table 4).

Based on the analysis, we found that there was no statistically significant difference in the levels of insulin and cortisol in both groups. Testosterone rates in men with metabolic syndrome were statistically significantly higher at 21.69% in the overweight group than in obese men.

To determine the relationship between body composition and hormonal levels, a correlation analysis was carried out in each group separately and in the total sample. Thus, in the group of men with metabolic syndrome, BMI had a direct correlation only with testosterone concentration (r=0.46), while FBM has an inverse correlation with testosterone (r=-0.36) and a direct correlation with insulin (r=0,47). In the control

group low correlation was found between FBM and hormone levels, in BMI it had a direct correlation with insulin (r=0.22).

## DISCUSSION

First and foremost, our study draws attention to the difference in hormone levels (insulin, cortisol, testosterone) and BMI and FBM in two comparative groups of young men. This once again underscores the contribution of regular physical activity to hormonal health in individuals, even at a young age.

The obtained results from comparing various body composition indicators based on BMI emphasize once again the need for a more extensive and thorough analysis of these indicators (FBM, TBW, LBW) in patients with varying body weights. In alignment with many other contemporary studies, our research demonstrates the lack of universality of a metric such as BMI in assessing metabolic health [1, 4 - 6].

The absence of an increase in the HOMA-IR index in the group of young men with metabolic syndrome and the presence of a statistically significant difference in the HOMA-IR index between control groups with obesity compared to overweight and normal body weight by 38.89% and 50.00% is very interesting and discussable question. Several factors can potentially explain these findings: 1. The control group men in the study might have significantly improved insulin sensitivity due to regular exercise, which can counteract the effects of obesity or overweight to some extent. Regular physical activity can enhance glucose uptake by muscles and



improve insulin sensitivity. 2. The control group may follow a specific diet that promotes better insulin sensitivity. Their dietary habits could be a contributing factor to maintaining lower HOMA-IR values despite obesity or overweight. 3. Genetic factors can also play a role. Some individuals may have genetic predispositions that make them less susceptible to insulin resistance, even in the presence of excess body weight. 4. The control groups' hormonal profile, including factors such as testosterone and adiponectin, could influence insulin sensitivity. Their hormonal balance may be more favorable compared to the group with metabolic syndrome. 5. The composition of the weight (lean muscle mass vs. fat mass) can be different between the groups. Control group men may have a higher proportion of lean muscle mass, which can improve insulin sensitivity.

In common, metabolic syndrome is a complex condition with varying degrees of severity and individual factors. It's possible that the metabolic syndrome group in this study represents a milder form of the condition. Understanding the exact reasons for these findings would likely require further investigation, possibly through additional studies and more comprehensive assessments of the participants' lifestyles, genetics, and metabolic health. The study's sample size and how individuals were selected for each group can also impact the results.

FBM and testosterone levels are closely linked, and there is a bidirectional relationship between them. Testosterone is an anabolic hormone that plays a crucial role in regulating body composition, including fat mass and lean mass. Studies have shown that increased FBM is associated with lower testosterone levels, and vice versa [21]. Adipose tissue, particularly visceral fat, contains an enzyme called aromatase, which converts testosterone to estrogen, leading to a decrease in testosterone levels. Furthermore, the relationship between FBM and testosterone levels is particularly relevant in men with metabolic syndrome. Metabolic syndrome is associated with an increased risk of low testosterone levels, which can exacerbate metabolic dysfunction,

including insulin resistance and dyslipidemia. In our study we also found direct correlation of BMI and inverse one of FBM with testosterone concentration ( $r=0.46$  and  $r=-0.36$  respectively) in metabolic syndrome young men. Our results are consistent with data of other authors [21-24], but unlike other studies, our research exclusively involved young men (under 44 years old), emphasizing both the early onset of testosterone level reduction and the independence of this issue from age.

FBM and insulin are also closely linked, and excess adiposity is a major risk factor for insulin resistance and type 2 diabetes: Excess adiposity, particularly visceral fat, leads to the release of pro-inflammatory cytokines, which can impair insulin signaling and contribute to insulin resistance [28, 30, 31]. In our study we found the strong correlation between FBM and insulin level ( $r=0.47$ ) only in metabolic syndrome group. This further underscores the strong association between excess weight and visceral obesity with the development of type 2 diabetes, as well as the significance of regular physical activity even in young individuals. What makes our study unique is that we did not find correlations between the BMI and insulin levels, only with FBM, whereas in most other studies, authors examined such associations primarily with the BMI [7, 9, 15, 27].

## CONCLUSIONS

A present study established the significant correlation of testosterone, insulin, and glucose concentration with FBM in all participants (MS and control groups). The negative effect of overweight (BMI > 25; FBM > 18 %) and obesity (BMI > 30; FBM > 25 %) for testosterone concentration was determined due to an increase of FBM > 20 % and insulin increasing > 9,0  $\mu$ U/l. Low testosterone and high insulin levels are associated with an increased risk of cardiovascular disease. Early effective strategies to manage adiposity and improve metabolic health outcomes especially in young men, including dietary changes and physical activity, can help improve testosterone and insulin levels and reduce the risk of cardiovascular diseases.

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### CONFLICT OF INTEREST

The Authors declare no conflict of interest

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