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## REVIEW

### METABOLIC SYNDROME – A COMMON CONDITION IN MODERN SOCIETIES

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**Abstract.** *The presence of multiple unknowns and uncertainty factors in medical research has made it extremely challenging to distinguish the preclinical phase of a chronic disease, such as the metabolic syndrome (MS), from the initial condition of health. MS is a complex and multifactorial medical condition characterized by the presence of at least three of the following conditions: high insulin levels (normal HOMA qualitative index < 2), elevated serum glucose level > 126 mg/dl, patients with abdominal obesity, a body mass index (BMI)  $\geq 30$  kg/m<sup>2</sup>, a lipid panel showing a triglyceride level  $\geq 150$  mg/dl, and HDL-CO < 35 mg/dl in men and < 45 mg/dl in women. In all developed countries, the number of obese people diagnosed with insulin resistance (IR) has rapidly increased to > 40% in recent years. This condition precedes the development of MS. The likelihood of having MS rises with advancing age. Additionally, new research has identified other influential hormones produced by adipose tissue that significantly impact metabolism, such as lipid cytokines, leptin, adiponectin, and resistin. Researchers believe that central obesity and the chronic inflammatory process play a key role in the development of metabolic syndrome and focus on mitochondrial dysfunction, changes in the gut microbiome, and pancreatic beta cell dysfunction. The primary determinant in averting the development of MS is humanity's struggle against its own bioenergetic entropy, which may induce catabolic processes that abbreviate life expectancy while promoting negentropy until the fulfillment of the life program predetermined in the genetic code.*

**Keywords:** *metabolic syndrome, qualitative index HOMA, body mass index.*

**DOI** <https://doi.org/10.56082/annalsarscimed.2023.2.6>

**Abbreviations:** **MS** - Metabolic Syndrome; **BMI** - Body mass index; **IR** - insulin resistance; **IW** - ideal weight; **ACSM** - American College of Sports Medicine; **METs** - metabolic equivalents; **TSM** - total body muscle mass; **THR** - target heart rate; **HR** - heart rate.

### **Introduction**

Numerous unknowns and numerous uncertainty factors encountered over the years of medical research have made it almost impossible to delineate the preclinical phase of a chronic disease from the initial state of health. The detection of biochemical changes in the body's apparatuses and systems before their clinical expression was quite unreliable through the investigations carried out with the usual paraclinical means.

### **Pathogenesis of metabolic disorders**

Modern methods of clinical and paraclinical investigation, by introducing new means of exploring biological reactions, try to solve the problems involved in fundamental research, with the following aspects: the existence of persistent or transient disturbances in the molecular plane of the biological phenomenon that inevitably leads to pathological alteration of the relationship between cellular structures and functions, the precession of the biochemical and enzymatic phenomenon compared to the morphological one, as well as the differentiation of the epiphenomena induced by the damaged structural support and the determination of the oscillating states of cellular bioenergy in healthy and diseased cells.

### **Anabolic and catabolic reactions, components of metabolism**

The term "metabolism" encompasses all biochemical and energetic changes that occur within the tissues of an organism. Metabolic processes are intricate, involving energy and matter exchanges and incorporating two (simultaneous) opposing processes:

- *catabolism* refers to the complete set of chemical processes in the body that break down substances by breaking the bonds between carbon atoms in different molecules, resulting in the release of energy (exothermic reaction).

- anabolism, refers to the chemical processes involved in the production of the components that constitute living matter (endothermic reaction).

Anabolic reactions involve an increase in the biological structure, and according to the second law of thermodynamics, there is an increase in the entropy of the system as energy is needed. In contrast, catabolic reactions involve a decrease in entropy and a release of energy. Catabolic pathways generate metabolites (amino acids, proteins, fatty acids, and glucose) and large, intermediate energies used in anabolism.

Glucose is anaerobically degraded during an intense muscular effort, with pyruvic acid being converted to lactic acid and lactic acid accumulating in proportion to the effort's intensity and duration. Pathological modifications in lipid metabolism exhibit a correlation with metabolic changes in carbohydrates and proteolytic compounds, and are contingent upon the functional condition of the principal organs implicated in the metabolism of said substances. Glucose is aerobically degraded during moderate muscular effort by oxidizing pyruvic acid produced by glycolysis via tricarboxylic acids.

### **Risk factors for the metabolic syndrome**

Metabolic syndrome, also known as syndrome X, is a complex and multifactorial medical condition characterized by the presence of at least three of the following conditions: high insulin levels (normal HOMA qualitative index < 2), elevated serum glucose level > 126 mg/dL, patients with abdominal obesity, a body mass index (BMI)  $\geq 30 \text{ kg/m}^2$ , a lipid panel showing a triglyceride level  $\geq 150 \text{ mg/dL}$ , and HDL-CO

< 35 mg/dL in men and < 45 mg/dL in women [1].

The metabolic syndrome arises from a combination of various factors, including genetic, environmental (such as a sedentary lifestyle and high-calorie diet), hormonal, and metabolic factors. These variables interact with each other and contribute to the development of the specific clinical characteristics that define this syndrome.

The prevalence of insulin resistance (IR) among obese adults has significantly risen to above 40% in recent years, observed in both developed and certain developing countries. IR is a pathophysiological condition characterized by a subnormal physiological response to insulin concentrations. This condition precedes the development of metabolic syndrome (MS). Insulin resistance is often considered a pre-diabetic condition [2].

A blood glucose level exceeding 120 mg/dl is indicative of insulin resistance in non-diabetic patients who are obese. Additionally, a qualitative HOMA index greater than 2 signifies the presence of liver fat, which may be assessed using CT/PET tomography [3].

The risk of developing metabolic syndrome and its constituents rises with advancing age. In recent years, it has become clear that lipid cytokines secreted by adipose tissue, such as leptin, adiponectin, and resistin, play additional roles in metabolism [4].

The development of a metabolic syndrome involves multiple intricate pathways that are now being studied and discussed within the scientific community. Some researchers believe that central obesity and the chronic inflammatory process play a key role in the development of metabolic syndrome, while others focus on mitochondrial dysfunction, changes in the gut microbiome, and pancreatic beta cell dysfunction (Figure 1) [5].

The progression of pre-diabetes to diabetes, however, can be prevented through a

combination of weight loss and increased physical activity, according to studies [6]. Metabolic syndrome can be avoided through lifestyle optimization, which is applicable to both at-risk individuals and the general population. In essence, it pertains to the reduction of cardiovascular risk factors through the consistent maintenance of a healthy body weight and adhering to at least 30 minutes of physical activity per day [7].

### **Ideal weight condition**

The ideal weight (IW) is recommended to be calculated according to height in centimeters (H) and age in years (A). Using the EMBED method, for men,  $IW = 50 + 0.75(H - 150) + [(A - 20) / 4]$ , and for women,  $IW \times 0.9$ . For individuals who are overweight or have degrees I–IV obesity, it is necessary to combine a physical exercise program with a daily decrease in energy intake from their diet. A prior study involving sedentary individuals demonstrated that engaging in self-paced walking (10,000 steps per day, 6 km) for 3 days per week outdoors, while expending 400 kcal, resulted in enhanced lipoprotein profiles and an upregulation of genes associated with lipid reverse transport (leptins).

The energy consumption per minute for jogging can be calculated as  $E \text{ (kcal)} = 0.8 \times v + 0.5$ , where  $v$  is the jogging speed (3.5–6.5 km/h) [8]. Metabolic equivalents (METs) are beneficial in situations where clinicians (American College of Sports Medicine) suggest walking as a form of exercise. A MET is defined as the energy or oxygen level utilized while at rest, with 1 MET equivalent to 3.5 VO<sub>2</sub> mL/kg/min. Recent research suggests that individuals with coronary artery disease have a mean resting MET level that is 23–36% lower than the usual norm of 3.5 ml/kg/min (Table 1) [9].

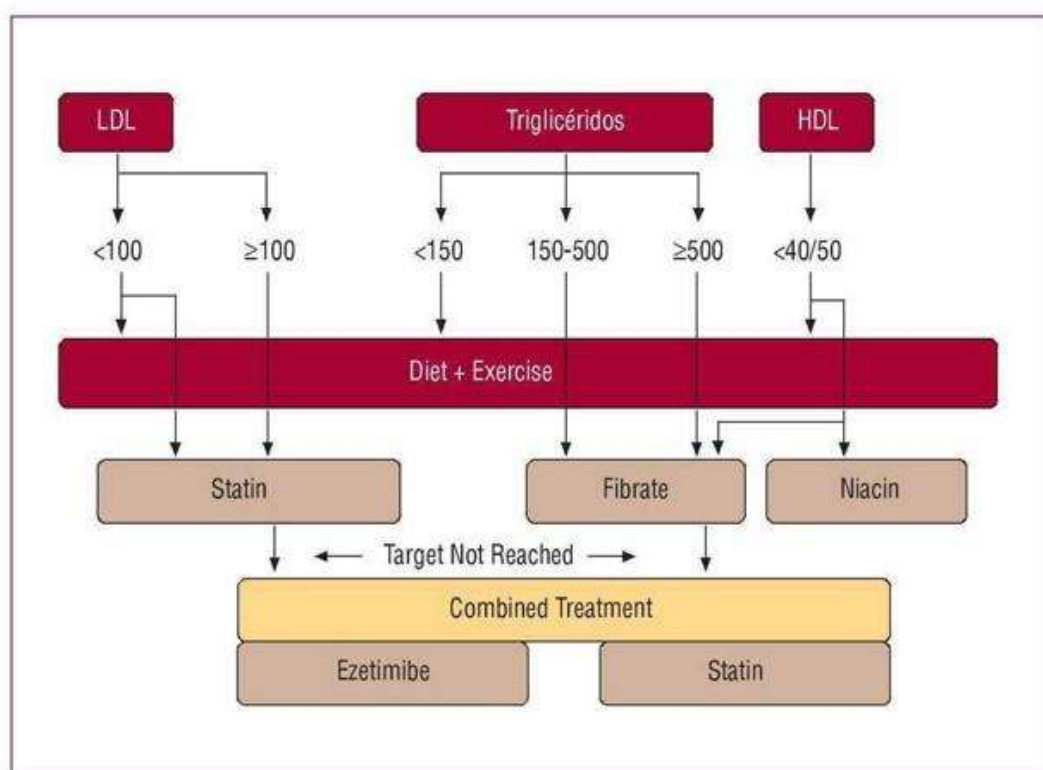
	Target heart rate	Heart rate (beats/min)	V <sub>O2</sub> (mL/min)	Energy consumption (kcal/min/kg)
Walking at 2 km/hour = 2 METs	Efficient/recovery zone – 60% to 70%	Heart rate at rest = 70-80 BTS	1000	7
Walking at 3 km/hour = 4 METs	Aerobic zone – 70% to 80%	Aerobic zone = 120-160 beats	2000	14

**Table 1.** Energy values of different types of exercise in terms of heart rate and respiratory equivalent.

The total energy consumed, Q (kcal), can be calculated by the theoretical equation  $Q$  (kcal) = oxygen consumption (VO<sub>2</sub>) x isocaloric coefficient, (4.83 kcal) = [(5.8 x W) + (151 + 10.1 x P) x 4.83], where W represents the patient's weight (kg) and P is the power to pedal on a treadmill (Watt/sec) [10]. Lipid energy consumption, QL (kcal), can be estimated using the equation  $Q_1 = VO_2 - [17.35 \times 4.83 \times \text{total body muscle mass (TSM)}]$ .

Lipid energy consumption must surpass 7 kcal/min for optimal physical exercise. The

target heart rate (THR), used to evaluate exercise intensity, was typically determined by the resting heart rate (HR) and the heart rate during physical exertion (HRP). The suggested range for HRP was 60–80% of the values associated with aerobic metabolism. For a value of 60% of aerobic metabolism, the THR is 136 bpm. Periodically order the control of risk factors (Figure 2).



**Scheme 2.** Control of metabolic syndrome risk factors

### Conclusions

The most important factor in preventing the installation of metabolic syndrome is man's fight with himself, with his bioenergetic entropy that could cause the catabolic processes of shortening his life, with the promotion of negentropy until the end of the life program written in the genetic code.

**Author Contributions:** *A.U and M.C. conceived the original draft preparation. A.U and M.C. were responsible for conception and design of the review. A.U was responsible for the data acquisition and for the collection and assembly of the articles/published data, and their inclusion and interpretation in this review. All authors contributed to the critical revision of the manuscript for valuable intellectual content. All authors have read and agreed with the final version of the manuscript.*

### Compliance with Ethics Requirements:

*“The authors declare no conflict of interest regarding this article”.*

**Acknowledgments:** *None.*

### References

1. Timar O, Sestier F, Levy E. The metabolic syndrome: an emerging risk state for cardiovascular disease. *Vasc Med* 2004;(9)1:55-68.
2. Kelly GS. Insulin resistance: lifestyle and nutritional interventions. *Altern Med Rev* 2000; (2):109-132.
3. Ma X, Holalkere SN, Kambadakone RA, Kenudson MN. Imaging-based quantification of hepatic fat: Methods and clinical applications. *Radiographics* 2009;29:1253-1277.

4. Man AM, Petra DN, Șulea AP, Varga A. Metabolic syndrome. Editorial Group: MEDICHUB MEDIA, DOI: 10.26416/Med 2021;(143)5: 5539.
5. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L et al. ESC Scientific Document Group. 2019 ESC/EAS Guidelines for the management of dyslipidemias: lipid modification to reduce cardiovascular risk. *Eur Heart J* 2020;41(1):111-188.
6. Twigg MS, Kamp CM, Davis MT, Neylon KE, Flack RJ. Prediabetes: A positionMetabolic Syndrome, a common condition at all ages statement from the Australian Diabetes Society and Australian Diabetes Educators Association. *Med J Aust* 2007;186 (9):461-465.
7. Donna KA, Roger SB, Michelle AA, et al. Guideline on the Primary Prevention of Cardiovascular Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guideline. *Circulation* 2019; 140:563-595.
8. Udristioiu A. Fitness bike and health, vol 1, Bucharest, Medical Ed. 2007: 5-45.
9. McAuley P, Meyers NJ, Abella PJ, Tan YS. Exercise capacity and body mass as predictors of mortality among male veterans with type 2 diabetes. *Diabetes Care* 2007;30(6):1539-1543.
10. Udristioiu A, Cojocaru M. From the Metabolic Syndrome to a Normal Status. *Int. Res. J. Basic Clin. Stud* 2016;4(2):017-022.