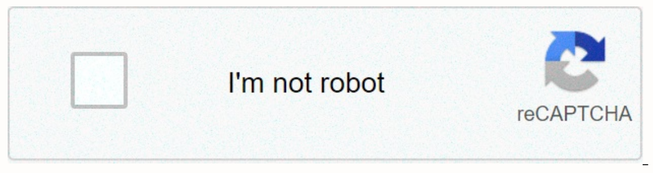

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Metabolic syndrome criteria 2017 pdf

Metabolic syndrome refers to several known cardiovascular risk factors including insulin resistance, obesity, atherogenic dyslipidus and hypertension. These conditions are linked and share mediators, mechanisms and underlying pathways. There has been a recent controversy about its definition and its usefulness. In this article, I review current definitions for metabolic syndrome and why the concept is important. It identifies a subgroup of patients with common pathopsology who are at high risk of developing cardiovascular disease and type 2 diabetes. Considering the central characteristics of metabolic syndrome and how they are associated, we may better understand the underlying and pathogenic pathopology. A comprehensive definition for metabolic syndrome and its key characteristics facilitates research on its causes and hopefully leads to new insights into pharmacological treatment approaches and lifestyles. Table 1 summarizes four of the most commonly used definitions of metabolic syndrome. The World Health Organization first developed its definition in 1998 (Alberti & Zimet, 1998). Because insulin resistance was felt to be central to the pathopsology of metabolic syndrome, evidence for insulin resistance is an absolute requirement in the WHO definition. This can be fasting sugar disorder [IFG, defined as a higher fasting glucose level than predetermined discontinuation, usually 100 mg per deciliter (mg/dl)] or impaired glucose tolerance (IGT, defined as a higher glucose level than a predetermined discontinuation, usually 140 mg/dl, for 120 minutes after swallowing 75 grams of glucose load during a oral glucose tolerance test). Alternatively, other measures could act as evidence of insulin resistance, such as evaluating a high homostatic model of insulin resistance value (HOMA-IR) that fits the fasting insulin product and fasting glucose levels. Finally, studies of euglismic hyperensolinmic clamps can be used as evidence of insulin resistance. In addition to the absolute need for insulin resistance, two additional criteria must be met. Obesity, dyslipidosis, high blood pressure and microalbuminuria are among these. Definitions of metabolic syndrome defined who was the first to tie together key components of insulin resistance, obesity, dyslipidosis and high blood pressure. The definition decrees that insulin resistance exists; without it, even if all other criteria were met, the patient would not have metabolic syndrome. The WHO definition also allows patients with T2D to be diagnosed with metabolic syndrome if other criteria meet other criteria. Because some measurements are not typically performed, for example, yoglisimi clamp studies, this definition does not apply easily clinically and does not lend itself to large epidemiological studies, where quick and simple assessments are important. In 1999 the European Group for the Study of Insulin (EGIR) proposed an amendment to the WHO definition (Balkau and Charles, 1999). Like who, EGIR felt that insulin resistance is central to the pathophilology of metabolic syndrome, so it also needs to be defined. In this case, insulin resistance is defined by the amount of fasting plasma insulin, which is greater than the 75th percention. The use of high fasting insulin alone simplified the definition as a reflection of insulin resistance, but it also means that patients with T2D cannot be diagnosed as having metabolic syndrome, as fasting insulin may be a useful measure of insulin resistance in such patients. Similar to the WHO definition, the definition of EGIR requires two additional criteria that can be chosen from obesity, high blood pressure and dyslipidosis. Obesity criteria were simplified around the waist, while the WHO definition used a choice of waist-to-hip ratio or body mass index. Microalbuminuria was removed as a diagnostic criterion. In 2001, the National Cholesterol Education Program (NCEP) developed the Adult Treatment Panel III (ATP III) for Metabolic Syndrome (National Cholesterol Education Program, 2002), updated by the American Heart Association and the National Institute of Lung and Heart Blood in 2005 (Grundy et al., 2005). According to the NCEP ATP III definition, there is metabolic syndrome if three or more of the following five criteria are met: waist circumference of more than 40 inches (men) or 35 inches (women), blood pressure over 130/85 mmHg, fasting triglyceride (TG) levels of more than 150 mg The definition of NCEP ATP III is one of the most widely used criteria for metabolic syndrome. This includes key characteristics of hyperglycemia/insulin resistance, visceral obesity, atherogenic dyslipidemia and high blood pressure. It uses laboratory measurements and results that are readily available to physicians and facilitate its clinical and epidemiological application. It's also simple and easy to remember. It is important that it does not require that a specific criterion be met, only that at least three of the five criteria are met. Therefore, the definition does not make a default on the underlying cause of metabolic syndrome, whether insulin resistance or obesity. In 2005, the International Diabetes Foundation published new criteria for metabolic syndrome (Zimmet et al., 2005). Although the same general criteria include other definitions, it requires that obesity, but necessarily insulin resistance, exist. The need for obesity is met with population-specific cut-out points. This constitutes the fact that different populations, ethnicities and nationalities have different distributions of norms for body weight and waist circumference. It also recognizes the relationship between these values and risk T2D or CVD varies in different populations. South Asian populations, for example, have an increasing risk for T2D and CVD at a smaller waist circumference that is not intended to meet the criteria in a Western population. Although visceral obesity is now considered an important factor, the IDF's definition has been criticized in pathopsology for its emphasis on obesity rather than insulin resistance (Reaven, 2006). The concept of metabolic syndrome has several practical applications. One of the most important uses in everyday clinical evaluation of patients is identifying patients at higher risk of T2D or CVD. However, metabolic syndrome should not be considered as just a way to identify patients at increased risk, as other established risk assessment methods address other important factors (Migs, 2004). For example, none of the definitions of metabolic syndrome takes into account the family history of diabetes, which is one of the strongest known risk factors for T2D. Therefore, determining metabolic syndrome will be more effective in using a specific risk assessment method such as the diabetes prediction model that takes into account family history. Similarly, definitions of metabolic syndrome of age do not take gender into account (although some cut-off points are gender-specific), smoking, low-density lipoprotein (LDL) or total cholesterol levels, all of which are known to be important CVD risk factors. In this way, metabolic syndrome will be more meta-modeled to predict cvd risk than a risk assessment tool such as a risk score. The major use of metabolic syndrome is not much in identifying patients at general risk of CVD and T2D, but also identifies a specific subtype of patients with common pathophilology. Therefore, the term acts as short-handed for physicians for common underlying biological processes. The definition of NCEP ATP III is easily applied in clinical environment. Doctors can easily score patients (and indeed, motivated patients can score themselves) in five criteria using easily measured end points and come up with a 'yes' or 'no' answer as to whether there is metabolic syndrome. This is different from some of the more complex risk calculation methods, which may require complex algorithms or calculations to get an answer. Although it has not been proven, the hope is that the realization of a diagnosis of metabolic syndrome will motivate individuals and their doctors to take appropriate steps to reduce their risk of CVD and T2D. This may include lifestyle changes such as improving food choices and increasing physical activity, and pharmacological management suitable for component criteria. Metabolic syndrome links insulin resistance, visceral fat, dyslipidosis and high blood pressure, which are known to be related. By doing so, the concept may help us better understand common pathopsological processes; to develop animal models useful for disruption; And by invention. Test new treatments. Metabolic syndrome has its own ICD-9 diagnostic code: 277.7. Despite this, there has been ongoing controversy about whether metabolic syndrome is a homogeneous disorder or disease, and whether it merits recognition as a syndrome (Meigs, 2004; Grundy et al., 2005; Kahn et al., 2005; Reaven, 2006; Grundy, 2007). When considering pathophilology, it is important to recognize that people who have isolated components but do not fit the definition of metabolic syndrome are not as high a risk for T2D or CVD. For example, people with isolated hypertension or isolated hyperlipidemia are at risk of CVD, but less so than people with multiple criteria. People with isolated obesity are at risk of T2D, but less so than those with metabolic syndrome. Although diabetes is considered equivalent to CVD by NCEP ATP III, additional risk factors leading to the diagnosis of metabolic syndrome further increase the risk of CVD in these patients. It has been argued that hypothetical patients with some, but not other, characteristics may be categorized by one or the other misdemeers (Reaven, 2006). However, as discussed below, the structure of definitions makes this unlikely, and patients who truly reflect common pathophilological processes that monitor metabolic syndrome should actually be captured by most definitions. There have been many epidemiological studies on metabolic syndrome, focusing on the prevalence of metabolic syndrome in different populations and the magnitude of risks for T2D, CVD and other related medical problems including fatty liver, gall cholesterol stones, polycystic ovary syndrome, obstructive sleep apnea and moaning. Such epidemiological studies require a simple and readily applied definition. These studies may add not only to our understanding of status pathopsology, but also its genetic basis, using genome-wide communication approaches. They may also lead to the development of therapeutic approaches that target composite physiological abnormalities, rather than individual component criteria. The expression, 'It's as simple as possible, but not easier' is attributed to Albert Einstein. Following this principle, current definitions of metabolic syndrome may be extended to four central characteristics: insulin resistance, visceral obesity, atherogenic dyslipidosis and endothelial dysfunction. Of these, it seems that the first two are absolutely needed for metabolic syndrome. In patients with metabolic syndrome, weight loss can lead to improvements in multiple characteristics at the same time, so a certain degree of adiposity seems to be needed to reveal abnormal pathophilology. Conversely, there are patients who are obese but do not reveal any of the other components of metabolic syndrome, so both metabolic susceptibce to insulin resistance and obesity seem necessary to express the phenotype of metabolic syndrome. Atherogenic Following insulin resistance and visceral obesity, and can be captured in definition including separate criteria for high serum TG levels and low HDL levels. Ondothial dysfunction is also followed by insulin resistance and from adipokines and free fatty acids (FFAs) that are released from visceral adipose tissue. Endothelial dysfunction is captured by the need for high blood pressure in definition. Both atherogenic dyslipidosis and onothelial dysfunction contribute to the development of atherosclerosis and CVD.Thus, four central characteristics – insulin resistance, visceral fat, atherogenic dyslipidus and anodyal dysfunction – the simplest comprehensive definition for metabolic syndrome, which cannot be easier. Even if other related findings such as systemic inflammation, hypercoagulability or microalbuminuria are important for pathophykology, they were not necessary as part of the definition because these findings were not needed independently. We will discuss each of these central features in the following section. Their symings are shown in the accompanying poster. Insulin is produced by the pancreas in response to hyperglycemia and stimulates different use of glucose in different tissues. The tissues that remove glucose from circulation and impact glucose make the most of are skeletal muscle, liver and adipose tissue. In skeletal muscle and adipose tissue, insulin stimulates glucose uptake by transferring GLUT4 glucose to the cell surface. In skeletal muscle and liver, insulin stimulates glycogen synthesis from glucose and inhibits glycogenolase. In the liver, insulin also reduces liver gluconogenesis and prevents more glucose infestation into the bloodstream. In adipose tissue, insulin inhibits fat breakdown, or lipolysis, and stimulates glucose uptake. The net effect of all these changes is to increase glucose uptake, reduce circulating glucose levels and increase glucose conversion into storage, glycogen or fat molecules (Kim et al., 2006). In insulin resistance, fat, muscle and liver cells do not respond properly to insulin, and circulating glucose levels remain high, leading to pathology. This is exacerbated by deregulation of feedback mechanisms. Insulin-mediated glucose excretion rates vary more than six times in the population. Some of this variation is due to adiposis and fitness, and some are the result of genetic origin. Insulin resistance occurs when there is a decrease in the responsiveness of peripheral tissues (skeletal muscle, fat and liver) to the effects of insulin. Insulin resistance is a powerful predictor of T2D, and hyperinsolinemia is a surrogate marker for insulin resistance. Physiological insulin signaling occurs after insulin binds to the insulin receptor, an active tyrosine kinase of the serend. Insulin binding leads to Tyrosine From downstream substrates and activation of two parallel pathways: phosphoinositaid 3-kinase (PI3K) pathway and mytogen-activated protein kinase pathway (MAP). Tyrosine activates the phosphorylation of insulin receptor substrates (IRS) PI3K, leading to the activation of phosinocytide-dependent protein kinase 3-phosphoinosite 1 (PDK1) kinase and Akt kinase. The PI3K-Akt pathway is responsible for many of the downstream metabolic effects of insulin. In vascular endothelial cells, aetkinase is phosphorylates and the activation of endothelial nitric oxide centase (eNOS). In skeletal muscle and adipose tissue, Akt kinase stimulates glut4 insulin-responsive glucose transfer to the cell surface, leading to increased glucose uptake. In parallel, phosphorylation of Tyrosine activates the SHC protein of GTP Sos exchange agent. This leads to activation of map kinase pathway including ross, rough, MAP kinase kinase (MEK) and extracellular regulated kinase (ERK). Map kinase pathway mediates the production of endothelin-1 (ET-1) that leads to vessels; In insulin resistance, the PI3K-Akt pathway is affected, while the MAP pathway is not kinase. This leads to a change in the balance between these two parallel paths. Inhibition of the PI3K-Akt pathway leads to a decrease in endothelial nitric oxide (NO) production, leading to endothelial dysfunction and decreased GLUT4 transmission, leading to a decrease in skeletal muscle absorption and fat glucose. In contrast, the MAP kinase pathway is unaffected, so there is continued production of ET-1, expression of molecules glued by vascular cells and pathogenic stimuli to smooth vascular muscle cells. In these ways, insulin resistance leads to vascular abnormalities that are susceptible to atherosclerosis. Insulin increases local blood flow in tissues through eNOS activation, leading to two detachable effects (Kim et al., 2006; Jonk et al., 2007). Capsule absorption occurs within minutes, while the diffusion of larger resistance vessels increases overall perfosis between 30 minutes and 2 hours. Both of these effects contribute to vasodilation and increase the delivery of glucose and insulin to tissues. Vascular effects of glucose hemostasis pair insulin with blood flow and contribute to glucose metabolism in physiological concentrations of insulin. Pharmacological inhibition reduces glucose excretion by 40%. In this way, insulin signaling harmoniously affects the use of peripheral glucose, vascular tone and blood flow. Common mechanisms that contribute to insulin resistance can therefore also affect vascular function, including hyperglycemia, advanced glycation products, toxicity caused by FFAs, obesity, dyslipidemia and other inflammatory conditions. Visceral obesity reduces mediated insulin Absorption, and clearly related to insulin resistance. Mechanisms of this are likely to include adipokines, which are made by adipose tissue, which modulate the cross between metabolism and vascular function (Kershaw & Flypast, 2004). Among them are tumor α necrosis agent (TNFα) and interleukin-6 (IL-6), which are pro-influenzaamateur and contribute to insulin resistance and vascular dysfunction. Renin's angiotensin system is also activated in adipose tissue, leading to high blood pressure and insulin resistance. In contrast, adiponectin is a protective adipokine that pairs insulin sensitivity with energy metabolism. Adiponectin levels decrease in obesity, T2D and metabolic syndrome. In addition to these adipokines, FFAs that are released from visceral fat and bioactive fat intermediaries act together to disrupt the PI3K-Akt pathway and increase oxidative stress. Key characteristics of atherogenic dyslipidemia are high plasma TG levels, low HDL cholesterol levels and small dense LDL increases. Insulin resistance and visceral obesity are associated with atherogenic dyslipidemia (Samankovic, 2006). Insulin resistance leads to atherogenic dyslipidosis in several ways. First, insulin routinely suppresses lipolysis in adipocytes, thus impairing insulin signaling increases lipolysis, resulting in increased FFA levels. In the liver, FFAs act as a substrate for the synthesis of TGs. FFAs also stabilize the production of apoB, the major lipoprotein of very low-density lipoprotein particles (VLDL), resulting in more VLDL production. Second, insulin typically degrades apoB through PI3K-dependent pathways, thus directly increasing insulin resistance to VLDL production. Third, insulin regulates lipoprotein lipase activity, rate limiter and major VLDL clearance mediator. Therefore, hypertri glyceridemia in insulin resistance is the result of both increased VLDL production and decreased VLDL clearance. VLDL is metabolized into residual lipoproteins and small dense LDL, both of which can promote the formation of atheroma. TGs in VLDL are transmitted to HDL by protein transporting cholesterol ester (CETP) in exchange for esters, resulting in HDL enriched with TG and VLDL particles enriched with ester cholesterol. TG-enriched HDL is a better platform for hepatic lipase, so it quickly clears out of circulation and fewer HDL particles remain in place to participate in the reverse transport of cholesterol from the vascular. Andothelial dysfunction is the latest common pathway between many cardiovascular risk factors and the development of atherosclerosis (Gimbrone et al., 2000; Huang, 2005; Kim et al., 2006). Endothelial cells line the inner surface of blood vessels and serve important mechanical, as well as biological, functions. Andothelium senses and responds to physiological and pathological stimuli, producing vasoactive substances including NO, prostaskilin and andotlins. Anesothelial expression of cellular adhesive molecules on interactions with Leukocytes and monocytes affect effective inflammation, with circulating platelets, homeostasis and thrombosis. Endothelium also modulates the response of the vascular smooth muscle layer, which may help inthymal formation during the development of atherosclerotic plaques. Normal endothelial function protects against these processes, and central endothelial dysfunction is used to pathogen the development of atherosclerotic damage. Widely defined endothelial dysfunction occurs when endothelium fails to serve its natural physiological and protective mechanisms. This may be because endothelium is damaged or missing, as in the case of danode endothelium in the coronary arteries that have underwent angioplasty. It may occur when normal andothelium responses are affected, for example by oxidative stress, hyperglycemia, advanced

glycation products, FFAs, inflammatory cytokines or adipokines. One of the common features of endothelial dysfunction is the reduction of NO bioavailability in vessels. There are several mechanisms for andothelial dysfunction (Huang, 2005). The most important of them is the reduction of eNOS phosphorylation in S1177 (Dimmeler et al., 1999; Fulton et al., 1999) and the rapid reaction of NO with superoxide to form peroxynitrite anion (Beckman and Koppenol, 1996). In addition, asymmetric dimethyl larginin (ADMA) may compete with arginine to reduce no endothelial production. eNOS requires enzymatic cofactors, including flavin adenine di naclotide (FAD), flavon mono leothide (FMN), NADPH and tetrahydrobiopterin (BH4). In the absence of BH4, electron transport via eNOS can become 'uncoupled', resulting in the production of superoxide by eNOS. Superoxide, whether composed by NADPH oxidase or uncoupled by eNOS, reacts with NO in a very rapid, limited release to the formation of peroxidinate anion, which has its own toxic effects.eNOS phosphorylation in S1177 seems to be a crucial regulator of its enzymatic activity. S1177 phosphorylation leads to an increase in electron flux through the Redux domain and a decrease in calmodulin dissociation. As a result, eNOS becomes more active and produces more NO, even at resting levels of intracellular calcium. Phosphorylation of eNOS decreased in diabetes, hypercholesterolemia and atherosclerosis. Physiological insulin signaling increases eNOS phosphorylation via pi3K-Akt pathway. Estrogens, statins, VEGF and leptin all increase eNOS phosphorylation by Akt kinase. Adiponectin, protective adipokine, increases eNOS phosphorylation by AMP kinase. The fact that diverse signaling pathways affect multiple kinases that converge by phosphorylation to modulate eNOS activity suggests that this is a common integration point that underlines endothelial dysfunction from various causes. Therefore, eNOS phosphorylation in S1177 seems to be a very important step in regulating eNOS activity and an important goal for intervention for endothelial treatment (Huang, 2005; Atochin et al., 2007). Insulin resistance reduces endothelial dysfunction by reducing Akt kinase activity, resulting in decreased phosphorylation and eNOS activity. Since eNOS phosphorylation in S1177 is required for insulin hemodynamic actions, this leads to a decrease in blood flow to skeletal muscle, creating a male cycle where endothelial dysfunction then worsens insulin resistance. In addition, the expression of ET-1 mediated by insulin and the detrimental effects of vascular smooth muscle is not affected by insulin resistance, further contributes to endothelial dysfunction. Visceral adiposity causes andothelial dysfunction through the effects of rystin, IL-6 and TNFα on eNOS phosphorylation. In addition to blocking IRS-1 activation, TNFα directly activates NADPH oxidase and increases superoxide production; TNFα also stimulates lipolysis, leading to the release of the FFA. In contrast, adiponectin, which stimulates eNOS phosphorylation, decreases in metabolic syndrome. In visceral fat, leptin resistance increases the production of reactive oxygen species. FFAs contribute to endothelial dysfunction by combining reducing PI3K-Akt signaling, increasing reactive oxygen species and increasing ET-1 production. In summary, the central characteristics of metabolic syndrome include insulin resistance, visceral adiposity, atherogenic dyslipidus and anodhelial dysfunction. These conditions are related and have common mediators, pathways and pathosological mechanisms. A comprehensive definition of metabolic syndrome, which is simply expressed as much as possible, will only include these characteristics. The need for multiple criteria ensures the exclusion of individuals with individual components (such as isolated hypertension or isolated hyperlipidemia), versus the compound pathophilology discussed above. The inclusion of both TG and HDL measures increases the atherogenic dyslipidemia feature, and the inclusion of the blood pressure criterion ensures that physiological hesitations are severe enough to lead to endothelial dysfunction. From different definitions for metabolic syndrome, the definition of NCEP ATP III is easiest for clinical and epidemiological practices because it uses the right criteria that are easily measured. Despite the ongoing controversy over whether or not the concept of metabolic syndrome is beneficial, it clearly defines certain pathopsological mechanisms that link central characteristics. Considering metabolic syndrome as a specific entity allows for genetically based research for sensitivity to this syndrome, a better understanding of its underlying pathophilology and the development of treatment approaches. Alberti K.G., Zimmet P.Z. (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of the Interim Report on Diabetes Mellitus from the Advice of the World Organization II. Diabet Med. 15, 539–553 [PubMed] [Google Scholar]Atochin Wang A., Liu V.W., Critchlow J.D., Dantas A.P., Looft-Wilson R., Murata T., Salomone S., Shin H.K., Ayata C., et al. (2007). ENOS phosphorylation mode modulates vascular resorbability and brain ischemia result in vivo. J. Klein. 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