*Review*

# Metabolic syndrome: What are the risks for humans?

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**Summary** **Metabolic syndrome (MetS) is a widely prevalent and multi-factorial disorder. The syndrome has been given several names such as insulin resistance (IR) syndrome, plurimetabolic syndrome, Reaven's syndrome, Syndrome X, and the deadly quartet. The formulation of National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP) guidelines has led to some uniformity and standardization of the definition of MetS and has been helpful epidemiologically. The clinical relevance of MetS is related to its role in the development of cardiovascular disease. Weight reduction is one of the mainstays of treatment. This article provides a comprehensive discussion of metabolic risk factors, the history of MetS, and its diagnosis, epidemiology, etiology, pathophysiology, and treatment. There is a need to comprehensively review this particular syndrome in view of the ever increasing-incidence of this condition.**

***Keywords:*** Metabolic syndrome (MetS), human, health

## 1. Introduction

India is a major contributor to the global increase in cardiovascular disease through the increased mortality and prevalence of metabolic syndrome (MetS). MetS is a constellation of physiological and biochemical abnormalities characterized by diabetes or high fasting glucose, central obesity, abnormal cholesterol and triglyceride (TG) levels, and hypertension (*1*,*2*). This clustering of abnormalities is frequently seen and attributed to people's dietary habits. One in approximately every 4 or 5 adults has developed MetS depending on the environmental conditions and daily lifestyle habits of the country where he or she resides. The incidence of this syndrome has been estimated to increase with age for individuals over 50 years of age. MetS affects 27% of the population in India, nearly 30% in Europe (*2*), and more than 40% in the US (*3*). MetS has been accepted worldwide as a clinical marker for earlier detection of cardiovascular disease and type 2 diabetes (*4*,*5*). People with MetS are estimated to have twice the risk of developing cardiovascular disease compared to healthy individuals and a five-fold increased risk of type 2 diabetes (*1*,*5*). However, the underlying pathophysiological processes leading to its development are unclear and there is confusion over its conceptual definitions and criteria, allowing the medical controversy over MetS to continue. An increase in total body fatness and preferential upper body accumulation of fat is independently related to insulin resistance (IR). Obese women with a greater proportion of upper body fat tend to be more insulin-resistant, hyperinsulinemic, glucose-intolerant, and dyslipidemic than obese women with a greater proportion of lower body fat. Therefore, the distribution of body fat is an important correlate of MetS. The term "metabolic" refers to the biochemical processes involved in the body's normal functioning. Risk factors are behaviors or conditions that increase a disease.

## 2. History

The term "MetS" dates back to at least the late 1950s but came into common usage in the late 1970s to describe various risk factors associated with diabetes, something that had been noted as early as the 1920s (*6*,*7*).

*i*) In 1947, the Marseilles physician Dr. Jean Vague made the interesting observation that upper body obesity appeared to predispose one to diabetes,

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atherosclerosis, gout, and calculi (*8*).

*ii*) Avogaro, Crepaldi and their co-workers described six moderately obese patients with diabetes, hypercholesterolemia, and marked hypertriglyceridemia, all of which improved when the patients were put on a hypocaloric, low carbohydrate diet (*9*).

*iii*) In 1977, Haller used the term "MetS" for the association between obesity, diabetes mellitus, hyperlipoproteinemia, hyperuricemia, and steatosis hepatis when describing the additive effects of risk factors on atherosclerosis (*10*).

*iv*) The same year, Singer used the term for the association of obesity, gout, diabetes mellitus, and hypertension with hyperlipoproteinemia (*11*).

*v*) In 1977 and 1978, Gerald B. Phillips developed the concept that risk factors for myocardial infarction coincide to form a "constellation of abnormalities" (*i.e.*, glucose intolerance, hyperinsulinemia, hyperlipidemia [hypercholesterolemia and hypertriglyceridemia] and hypertension) that is associated not only with heart disease but also with aging, obesity and other clinical states. He suggested there must be an underlying linking factor, the identification of which could lead to the prevention of cardiovascular disease; he hypothesized that this factor was sex hormones (*12,13*).

*vi*) In his Banting lecture in 1988, Gerald M. Reaven proposed IR as an underlying factor and named the constellation of abnormalities Syndrome X. Reaven did not include abdominal obesity, which has also been hypothesized to be an underlying factor, as part of the condition (*14*).

## 3. Risk factors

The following factors increase chances of developing MetS.

### 3.1. *Age*

The risk of MetS increases with increasing age, affecting less than 10% of people in their 20s and 40% of people in their 60s. However, some research shows that about one in eight school children has three or more components of MetS. Other research has also identified an association between childhood MetS and adult cardiovascular disease decades later.

### 3.2. *Race*

About 47 million adults in the United States (almost 25%) have MetS. The condition is more common in African American women and Mexican American women than in men of the same racial groups. MetS affects Caucasian women and men roughly equally. Some racial and ethnic groups in the United States are at higher risk of MetS than others. Mexican Americans have the highest rate of MetS, followed by Caucasians and African Americans. In addition, certain ethnic groups, such as Hispanics and South Asians, are at increased risk for MetS.

### 3.3. *Obesity*

A body mass index (BMI), a measure of percentage of body fat based on height and weight, greater than 25 increases the risk of MetS. Excess fat in the abdominal area is a greater risk factor for heart disease than excess fat in other parts of the body, such as on the hips. Therefore, so does abdominal obesity, *i.e.*, having an apple shape rather than a pear shape.

### 3.4. *History of diabetes*

There is a greater likelihood of MetS if a family history of type 2 diabetes or a history of diabetes during pregnancy (gestational diabetes) is present.

### 3.5. *Other diseases*

A diagnosis of fatty liver, gallstones, breathing problems during sleep, cardiovascular disease, or polycystic ovary syndrome (such metabolic problems affect a woman's hormones and reproductive system) also increases the risk of MetS.

## 4. How is MetS diagnosed?

The risk factors seen in MetS include: IR, obesity (especially abdominal obesity), high blood pressure, high fasting glucose or hyperglycemia, and lipid abnormalities. There must be at least three of the following five metabolic risk factors for an individual to be diagnosed with MetS:

a) A higher TG level (≥ 150 mg/dL than normal). TGs are a kind of fat and hang out in fat cells but they also circulate in the blood.

b) A lower high density lipoprotein (HDL) cholesterol level (< 50 mg/dL for women and < 40 mg/dL for men than normal). HDL is sometimes called "good" cholesterol because it helps in removing cholesterol from arteries. It cleans out the low density lipoproteins (LDLs) or "bad" cholesterol from blood. When there are not enough HDLs, the LDLs can run rampantly, causing plaque to build up in artery walls and put a strain on the heart and circulatory system. A low HDL cholesterol level increases the risk of coronary heart disease.

c) Higher blood pressure (≥ 130/85 mmHg than normal). Blood pressure pushes the blood against the arterial walls as the heart pumps out blood. If this pressure rises and remains high, it can damage the heart and lead to plaque buildup.

d) Higher fasting blood glucose level (more than 100 mg/dL). Fasting blood glucose between 100 and 110 mg/dL is a sign of MetS. A mildly high blood sugar (between 100 and 125 mg/dL) may be an early predictor of diabetes. About 85% of people who have type 2 diabetes, the most common type of diabetes, also have MetS. These people have a much higher risk of heart disease than the 15% of people who have type 2 diabetes but not MetS.
e) Large waist circumference (≥ 35 inches for women and ≥ 40 inches for men). Having a large waist circumference or apple-shaped figure means that there is excess weight around the waist (abdominal obesity), representing an increased risk of heart disease and other health problems.

Throughout the years, several definitions of MetS have been proposed, emphasizing IR or abdominal/visceral obesity. However, the 4 main definitions are from the World Health Organization (WHO) definition 1999 (*15*), the Adult Treatment Panel III (ATPIII) Report 2001 (*16*), the European Group for the Study of IR (EGIR) 1999 (*17*), and the International Diabetes Federation (IDF) consensus (*18*) on MetS (Table 1). The definition of MetS according to the National Cholesterol Education Program (NCEP) was slightly updated by the American Heart Association (AHA) and National Heart, Lung, and Blood Institute (NHLBI) in 2005 (*19*,*20*) and the same year IDF proposed a new definition (*21*) based on clinical criteria. The two are very similar and should presumably identify many of the same individuals as having MetS. This modification of the IDF and ATP III definitions increased the emphasis on abdominal obesity as the core feature of MetS. The two differences are that the IDF definition excludes any subject lacking an increased waist circumference while the NCEP definition diagnoses MetS based on other criteria. Secondly, the IDF definition uses physical feature-specific cut-off points for waist circumference, while the NCEP definition uses only one set of cut-off points for waist circumference regardless of physical features. Abdominal obesity measured by waist circumference is an essential requirement for the diagnosis, while other variables featured in the ATP III definition have changed slightly (Table 1).

4.1. *WHO criteria* (*15*)

According to WHO criteria (1999), the presence of MetS requires the presence of diabetes mellitus, impaired glucose tolerance, impaired fasting glucose or IR, and at least two of the above factors.

4.2. *NCEP-ATP III criteria* (*16*)

The NCEP-ATP III definition (2001) requires at least three of the risk factors.

4.3. *EGIR criteria* (*17*)

The EGIR definition (1999) requires IR in the top 25% of the fasting insulin values among non-diabetic individuals and two or more risk factors.

4.4. *AHA/NHLBI/Updated NCEP criteria* (*19*,*20*)

There is confusion as to whether AHA/NHLBI intended to create another set of guidelines or simply update the NCEP-ATP III definition. According to Scott Grundy, University of Texas Southwestern Medical School, Dallas, Texas, the intent was just to update the NCEP-ATP III definition and not create a new definition.

**5. Insulin resistance – A key aspect of MetS?**

A key aspect of MetS is IR. In the body's attempt to counterbalance IR, extra insulin is produced, leading to increased insulin levels. The increased insulin levels can directly or indirectly lead to the characteristic metabolic abnormalities seen in patients. Frequently, the IR will progress to overt type 2 diabetes, further increasing the risk of cardiovascular complications.

**6. Who is more susceptible to MetS?**

MetS tends to run in families, along with the propensity for type 2 diabetes. MetS will occur in susceptible people who become overweight and sedentary. Therefore, MetS (like type 2 diabetes) can most often be prevented by exercise and maintaining a healthy body weight.

**7. Current global status of MetS and cardiovascular disease**

MetS is a cluster of metabolic risk factors that is strongly associated with the potential development of atherosclerotic cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM). The prevalence of MetS depends on age, ethnic background, and gender. It increases linearly from the age of 20 until age 50, when it plateaus. Global statistics show that approximately a quarter of the adult population suffers from this clinical condition. Various studies from around the world, including those of the general population, indicate that individuals age 20-25 and up have a prevalence of 24% (India), 28% (USA), 30.1% (Tehran), 33.4% (Turkey), and 39.3% (Saudi Arabia). A high prevalence of MetS has been reported in sub-Saharan Africa and the Middle East; South Africa, Morocco, Oman, and Iran have reported prevalence rates of 33.5%, 16.3%, 21%, and 33.7%, respectively. Prevalence rates are also high in Venezuela (31.2%) and urban Brazil (25.4%) (*22*,*23*). The situation appears to be similar in South Asian countries. The recent data show that one-

**Table 1. Diagnostic criteria for metabolic syndrome**

| S. No. | Criteria for metabolic syndrome | Obesity | | Dyslipidemia | | Blood pressure (Systolic and Diastolic) | Glucose | Insulin resistance | Other |
|---|---|---|---|---|---|---|---|---|---|
| | | Male | Female | Male | Female | | | | |
| 1. | WHO (5th or 6th + ≥ 2 criteria), 1999 | WHR > 0.90 and/or BMI > 30 kg/m$^2$ | WHR > 0.85 and/or BMI > 30 kg/m$^2$ | TG ≥ 150 mg/dL (≥ 1.7 mM); HDL-C < 35 mg/dL (< 0.9 mM) | TG ≥ 150 mg/dL (≥ 1.7 mM); HDL-C < 39 mg/dL (<1.0 mM) | ≥ 140/90 mmHg | T2DM, impaired glucose tolerance, impaired fasting glucose | IR measured under hyperinsulinemiceu glycemic conditions | Urinary albumin excretion rate ≥ 20 μg/min or albumin: creatinine ratio ≥ 30 mg/g |
| 2. | EGIR (5th + ≥ 2 criteria), 1999 | WC ≥ 94 cm | WC ≥ 80 cm | TG ≥ 177 mg/dL (≥ 2.0 mM); HDL-C < 39 mg/dL (< 1.0 mM) | | ≥ 140/90 mmHg or on medication | Fasting glucose > 110 mg/dL (≥ 6.1 mM) | IR | – |
| 3. | NCEP-ATP III (≥ 3 criteria), 2001 | Abdominal obesity WC ≥ 102 cm or 40 inches | Abdominal obesity WC > 88 cm or 36 inches | TG ≥ 150 mg/dL; HDL-C < 40 mg/dL or on therapy | TG ≥ 150 mg/dL; HDL-C < 50 mg/dL or on therapy | ≥ 130/85 mmHg or on therapy | Fasting glucose > 110 mg/dL (≥ 6.1 mM) | – | – |
| 4. | AHA/NHLBI or Updated NCEP criteria, 2005 | WC ≥ 102 cm (Asian ≥ 90) or 40 inches | WC > 88 cm (Asian ≥ 80) or 36 inches | TG ≥ 150 mg/dL (≥ 1.7 mM); HDL-C < 40 mg/dL (1.0 mM) or on therapy | TG ≥ 150 mg/dL (≥ 1.7 mM); HDL-C < 50 mg/dL (1.0 mM) or on therapy | ≥ 130/85 mmHg or on medication | Fasting glucose > 100 mg/dL (5.6 mM) | – | – |
| 5. | IDF (1st + ≥ 2 other criteria), 2005 | Ethnicity-specific WC (≥ 90 cm for men and ≥ 80 cm for women) for Indian subjects (If BMI is > 30 kg/m$^2$, central obesity can be assumed and WC does not need to be measured) | | TG ≥ 150 mg/dL (≥ 1.7 mM); HDL-C < 40 mg/dL (1.03 mM) or on therapy | TG ≥ 150 mg/dL (≥ 1.7 mM); HDL-C < 50 mg/dL (1.3 mM) or on therapy | ≥ 130/85 mmHg or on therapy | Fasting glucose > 100 mg/dL (5.6 mM) or DM | – | – |

Abbreviation: Waist circumference (WC).

fourth to one-third of the urban population of India has MetS (*24*). MetS is highly prevalent among urban populations of Indians (35.2% *vs.* 20.6%) compared to rural populations. Its prevalence increases with age and is 1.5-2 times higher in women than in men (*24,25*). Interestingly, certain communities in India (*e.g.* the Punjabi Bhatia community in northern India) tend to have an inordinately high incidence of obesity, T2DM, and MetS (*26*). Two Indian studies described vastly different rates of MetS prevalence in India. Both had different definitions of obesity: one used obesity criteria tailored to Indians while the other (*27*) used the standard ATP III definition of obesity. Both studies used population-based samples within the same age range but reported prevalence of 13% in Jaipur (*27*) and 41% in Chennai (*28*). The studies had far larger differences in terms of the prevalence of elevated TG (46% *vs.* 30%), hypertension (55% *vs.* 39%), and elevated fasting plasma glucose (27% *vs.* 5%) although both reported having used the same cut-off points. Interestingly, a third Indian study (*29*), also of Chennai, reported a MetS prevalence of 11.2% (using EGIR criteria), which was much closer to the prevalence rate reported for Jaipur than that reported for Chennai. Therefore, there appear to be significant differences in the prevalence of both individual factors that constitute MetS and MetS itself even within the same ethnic population. Hispanics and African-Americans have the greatest risk for developing MetS, followed by Caucasians. Asians have the lowest risk, at least in the United States. The prevalence of MetS, when based on the ATP III criteria, varies as described earlier among ethnic groups like Finnish and Native American men. Studies of these two groups involved subjects with comparable age ranges (42-60 and 44-49 years, respectively), and yet the Finnish study found a prevalence of only 14% compared with a prevalence of 43.6% in the study of Native Americans. Prevalence rates vary from a low of 13.9% in black men to a high of 27.2% in Mexican American women (*30*). The literature indicates that MetS is currently more prevalent and a danger to a large number of people worldwide.

CVD causes numerous deaths worldwide and in India. People with MetS are at higher risk of morbidity and mortality from CVD (*31*). The main reason for this is that the combination of MetS risk factors interacts synergistically to start or accelerate the progression of atherosclerosis (*31*). At the same time, early diagnosis, prevention, and management of MetS are considered the key approaches in reducing the risk of progression of atherosclerosis and development of CVD (*32*). One report suggested that MetS could be responsible for approx 7% of total mortality, regardless of the cause, and up to 17% of CVD (*33*). Similarly, a report from the Framingham Heart Offspring Study showed that the contribution of MetS to the risk of CVD was 34% in men and 16% in women (*34*). In that analysis, the components of the syndrome that contributed most to the CVD outcomes were high blood pressure (33%) and low HDL-cholesterol (25%). A meta-analysis of 37 longitudinal studies found a 78% increased risk for CVD events and death in people with MetS (*35*). The ability of CVD to predict MetS may vary by ethnicity, gender, and the presence or absence of hyperglycemia. Moreover, few studies have investigated the mortality rate, lifestyle behaviors, and nutritional factors that are known to be risk factors for MetS and CVD in India (*36*). Major differences in CVD mortality rates in different Indian states were reported, varying from 75-100 per 100,000 people in sub-Himalayan states of Nagaland, Meghalaya, Himachal Pradesh, and Sikkim to a high of 360-430 in Andhra Pradesh, Tamil Nadu, Punjab, and Goa. The pressing issues with regard to MetS and CVD in India is that the prevalence of the two conditions has increased dramatically and that there is no clear information on the risk factors for MetS and CVD among the population of Northern India. MetS therefore may become a major contributor to accelerated aging and functional decline and could represent a major public health problem, eclipsing CVD and type 2 diabetes in the near future.

**8. Risk of developing CVD in individuals with MetS**

The relative risk of developing CVD associated with MetS as defined by NCEP-ATP III or by other organizations has increased 2- to 5-fold in both men and women and in various populations (*31,37,38*). Data from a Quebec cardiovascular study of individuals with several risk factors associated with MS were characterized by a tremendous increase in the relative risk of CVD compared to individuals who had only one or none of the risk factors. For example, non-diabetic men who had hyperapoB, small dense LDL, and hyperinsulinemia simultaneously had a 20-fold increase in the risk of CVD over 5 years compared to men who had none of these metabolic perturbations (*39*).

Another study showed that the risk associated with hypertriglyceridemia was modulated to a significant extent by the presence or absence of other components of MetS. For example, men with marginally increased plasma TG levels (above 1.6 mM) but with no other features of IRS had a 3-fold increase in the risk of CVD compared to men with normal plasma TG levels (*40*). The risk of CVD increased 13-fold for subjects with moderate hypertriglyceridemia who also had hyperapoB, reduced HDL-C levels, and increased insulin concentrations (*40*). These data clearly indicate that MetS, irrespective of its definition, may be associated with a significantly increased risk of CVD. Therefore, components of MetS may significantly contribute to this increased risk of CVD.

**9. Etiology**

The cause of MetS is unknown. Its pathophysiology is extremely complex and has been only partially elucidated. Most patients are older, obese, sedentary, and have a degree of IR. The most important factors are, in order: *i*) aging, *ii*) genetic makeup, and *iii*) daily lifestyle and habits (*e.g.* low physical activity and excess caloric intake).

There is debate regarding whether obesity or IR is the cause of MetS or if obesity and IR are consequences of more far-reaching metabolic dysfunction. A number of markers of systemic inflammation, including C-reactive protein (CRP), often increase, as do interleukin 6 (IL-6), tumor necrosis factor-alpha (TNF-α), resistin, leptin, and adiponectin.

**10. Pathophysiology**

Obesity and metabolic abnormalities were known to be associated with poor cerebrovascular outcomes when the concept of MetS first appeared. In 1995, Dr. G Reaven noticed that those outcomes were found in people who had hyperinsulinemia, high TG, low HDL cholesterol, and hypertension, all of which were considered factors for the development of CVD. Many earlier studies measured only serum total cholesterol regardless of LDL-cholesterol levels, although most total cholesterol consists of LDLs. Thus, the robust relationship between total cholesterol and coronary heart disease found in epidemiological studies strongly implies that an elevated LDL is a highly prevalent and powerful risk factor. Epidemiological investigations of human populations point to high levels of LDL cholesterol as being atherogenic lipoprotein.

Previous research demonstrated that adipose tissue plays an important role in energy regulation *via* endocrine, paracrine, and autocrine signals (*41*) and various factors known as 'adipokines' released by adipose cells cause IR. These adipokines are defined as insulin antagonists (TNF-α, IL-6, and resistin) and insulin sensitizers (leptin and adiponectin) (*42*). These adipokines markedly affect peripheral functions and influence the pathogenesis of obesity-related disease, particularly diabetes and cardiovascular disorders.

Visceral fat accumulation is found to be specifically associated with metabolic alteration of obesity in both men and women. Increasing accumulation of visceral fat leads to the overproduction of some adipokines such as IL-6, TNF-α and resistin, decreasing insulin action in muscles and/or the liver, while some adipokines like leptin and adiponectin have a beneficial effect on energy balance, insulin action, and vasculature. Leptin regulates energy balance and has an insulin-sensitizing effect. These beneficial effects are reduced in obesity due to leptin resistance. Adiponectin increases insulin action in muscles and the liver and has an anti-atherogenic effect. Conversely, excessive production of other adipokines is deleterious. The increased levels of circulating adipokines associated with visceral obesity may be attributed to production by ectopic adipose tissue. Adiponectin is the only known adipokine with circulating levels that decrease as a result of visceral obesity while levels of other adipokines increase. This dysregulation of adipokine production may promote obesity-linked metabolic disorders and CVD.

The accumulation of visceral fat hastens the release of non-esterified fatty acids (NEFAs), resulting in greater lipolytic activity in obese individuals and increasing NEFA levels in systemic circulation. This increased release of NEFAs into the portal circulation stimulates hepatic glucose production and reduces hepatic insulin clearance, ultimately resulting in insulin resistance, hyperinsulinemia, and hyperglycemia (*43*). When obesity develops, abnormal production of these adipokines by more visceral fat contributes to a proinflammatory state. This state of inflammation is likely to contribute to the health problems associated with obesity such as dyslipidemia, insulin resistance, and atherosclerosis. In contrast to these adipokines, levels of the insulin sensitizing and anti-inflammatory adipokine adiponectin are reduced in visceral obesity (*44*), and this may further exacerbate the state of low-grade systemic inflammation. In the liver, adiponectin increases insulin sensitivity by lowering NEFA uptake, increasing fatty acid oxidation, and reducing hepatic gluconeogenesis and very low density lipoprotein (VLDL) production. In muscle, adiponectin stimulates glucose uptake and fatty acid oxidation (*45*). Therefore, the altered expression of adipokines associated with visceral obesity induces a state of low-level systemic inflammation and dyslipidemia, eventually leading to atherosclerosis. In visceral obesity, dysregulated production of specific adipokines may contribute to hypertension and CVD. Hypertension is a common manifestation of the metabolic disturbances associated with insulin resistance and hyperinsulinemia, a key factor for MetS. Studies using the hyperinsulinemic-euglycemic clamp technique have demonstrated that hyperinsulinemia occurs in hypertension as a compensatory response to reduced insulin-stimulated glucose uptake by skeletal muscle (*46*,*47*). Adipocytes synthesize and release several factors that have been linked to blood pressure control, including adiponectin, leptin, and resistin. Increasing evidence suggests that aberrant production and release of such adipokines as adiponectin, leptin, and resistin by adipocytes may contribute to the high prevalence of hypertension in visceral obesity. Therefore, adipokines are potential causes of insulin resistance, endothelial dysfunction, and hypertension and reflect the role visceral obesity plays as a causal factor in metabolic and vascular disease.

**11. Treatments and drugs for MetS**

Tackling one of the risk factors for MetS is tough – taking them all on might seem overwhelming. That says, healthy or aggressive lifestyle changes and, in some cases, medication can improve every component of MetS. Lifestyle changes include losing weight, getting more regular physical activity, following a heart healthy diet, quitting smoking, reducing one's blood pressure, and improving one's cholesterol and blood sugar levels. The main focus of treating MetS is managing the risk factors that are under control, such as being overweight or obese, having an inactive lifestyle, or consuming an unhealthy diet. These changes are the key factors in reducing metabolic risk.

11.1. *Exercise*

More activity means more of a benefit. The four main types of physical activity are aerobic, muscle strengthening, bone strengthening, and stretching. Physical activity can be light, moderate, or vigorous. Doctors recommend 30 to 60 min of moderate-intensity exercise, such as brisk walking, every day.

11.2. *Losing weight*

In general, people who have MetS and are overweight or obese and who then lose as little as 7-10% of their body weight can reduce their insulin levels and blood pressure and decrease their risk of diabetes.

11.3. *Lipid abnormalities*

While the lipid abnormalities seen with MetS (low HDL, high LDL, and high TGs) respond nicely to weight loss and exercise, drug therapy is often required. Treatment should be aimed primarily at reducing LDL levels according to specific recommendations. Once reduced LDL targets are reached, efforts should be made to reduce TG levels and raise HDL levels.

11.4. *Clotting disorders*

People with MetS can have several coagulation disorders that facilitate the forming of blood clots within blood vessels. These blood clots are often a precipitating factor for a heart attack. Excessive blood clotting is a condition that often occurs with MetS.

11.5. *Eating healthy*

A heart healthy diet is an important part of a healthy lifestyle. The Dietary Approaches to Stop Hypertension (DASH) diet and the Mediterranean Diet, like many healthy-eating plans, limit unhealthy fats and emphasize fruits, vegetables, fish, and whole grains. Both of these dietary approaches have been found to offer important health benefits – in addition to weight loss – for people who have components of MetS. A doctor's guidance is needed before starting a new eating plan.

11.6. *Stopping smoking*

Smoking can increase the risk of heart disease and heart attacks and worsen other heart disease risk factors. Smoking cigarettes increases IR and worsens the health consequences of MetS. A doctor should be consulted for help in quitting cigarettes. The doctor can help the individual to monitor weight and blood glucose, cholesterol, and blood pressure levels in order to ensure that lifestyle modifications are working.

11.7. *Medication*

If goals cannot be achieved with lifestyle changes, a doctor may also prescribe medications to lower blood pressure with diuretics or angiotensin-converting enzyme (ACE) inhibitors, reduce unhealthy cholesterol levels with statins, fibrates, or nicotinic acid, or provide help in losing weight. High blood sugar is treated with oral medicines such as metformin, insulin injections, or both. Low-dose aspirin can help reduce the risk of blood clots, especially for people at high risk of heart disease.

**12. Conclusion**

Both an increased waist:hip ratio and IR are emerging risk factors for MetS, and Indians are considered to be more genetically predisposed to both. Larger numbers of people with MetS are linked to a rise in obesity rates among adults. In the future, MetS may overtake smoking as the leading risk factor for heart disease. The key to preventing MetS, however, remains diet and exercise. Any person with a strong family history of MetS or type 2 diabetes should be especially careful to maintain a healthy lifestyle. A healthy lifestyle is a lifelong commitment. Successfully controlling MetS takes a long-term effort and teamwork with health care providers.

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