The Metabolic Syndrome: Definition, Pathophysiology, and Scope of the Problem

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Goal. The goal of this lesson is to define the metabolic syndrome, described by some as the “new epidemic.” Pathophysiology and scope of the problem will be emphasized.

Objectives. At the completion of this activity, the participant will be able to:
1. demonstrate knowledge of the metabolic syndrome including definitions and prevalence;
2. identify underlying pathophysiology of the components of the metabolic syndrome, and recognize how each component relates to the others;
3. select normal and/or cutoff points for each component of the metabolic syndrome; and
4. exhibit knowledge of lifestyle interventions to convey to patients and their caregivers to manage the syndrome.

The metabolic syndrome (MetS), which affects an estimated one-fourth of the adult population of the United States, has been identified as a central player in the growing epidemic of cardiovascular disease (CVD) and diabetes throughout the Western world. At present, coronary heart disease (CHD) is the leading cause of death in the United States, accounting for an estimated 680,000 deaths annually, or about one of every five deaths from all causes. Adults with diabetes have heart disease death rates about two to four times higher than adults without diabetes. CHD is also the leading contender in causing premature, permanent disability in the U.S. labor force. Persons with MetS have a two- to three-fold increased risk for CHD, a similar risk for future ischemic stroke, and a much greater risk for developing future type 2 diabetes mellitus (T2DM).

Over the past several decades, evidence has emerged that suggests there is a close link between T2DM and CHD. It has long been recognized that patients with diabetes are at significantly increased risk of developing CHD. Indeed, evidence suggests that diabetic patients without previous myocardial infarction (MI) have as high a risk of MI as non-diabetic patients with previous MI. Furthermore, evidence strongly supports the contention that diabetes and prediabetic conditions are common in patients with CHD. The more components of MetS a patient exhibits, the greater the risk, which is made much worse by concomitant low-density lipoprotein cholesterol (LDL-C) elevation. Although the U.S. National Cholesterol Education Program’s Adult Treatment Panel III (NCEP:ATP III) identified CHD as the primary clinical outcome of MetS, most individuals with this syndrome have insulin resistance, which confers increased risk for T2DM. When diabetes becomes clinically apparent, CHD risk rises sharply. Beyond CHD and T2DM, individuals with MetS are also susceptible to other conditions, notably polycystic ovary syndrome, fatty liver, cholesterol gallstones, asthma, sleep disturbances, and some forms of cancer.

Characteristics of the Metabolic Syndrome

Defining the Syndrome. The concept of MetS was identified in the 1920s. The constellation of metabolic abnormalities that defines MetS includes glucose intolerance (T2DM, impaired glucose tolerance or impaired fasting glycemia), insulin resistance, central obesity (i.e., the amount of fat in the waist area), dyslipidemia, and hypertension, all well documented risk factors for CHD. These conditions co-occur in an individual more often than would be expected by chance. Grouped together, these abnormalities are associated with increased risk of CHD above what would be expected for each individually. Intensive investigation continued to
explain the constellation of cardiovascular risk factors of Syndrome X, also referred to as the “deadly quartet,” DROP (Dyslipidemia, insulin Resistance, Obesity and blood Pressure), and the “insulin resistance syndrome.” It would not be until later in 1998, however, that there was an initiative to develop an internationally recognized definition (Table 1). In an attempt to achieve some agreement on its definition and also to provide a useful tool for clinicians and researchers, the World Health Organization (WHO) proposed a set of criteria. This was followed by attempts by other professional groups, including the European Group for the Study of Insulin Resistance (EGSIR), to formulate their own specific definitions. Subsequently, the NCEP:ATP III formulated its own definition, and published it in 2001. The definitions agree in principle on the essential components: glucose intolerance, central obesity, hypertension and dyslipidemia, but differ in detail and significance for each criterion. The WHO definition and that of other groups agree, in that they include either glucose intolerance or insulin resistance as an essential component. For the NCEP:ATP III definition, however, this criterion is not included as essential. Additionally, cut-off points for each component of the cluster of symptoms and the way they are combined to define the MetS differ between the various definitions.

The WHO report stated that the definition would be modified as new information became available concerning the components. The WHO definition turns out to be better suited for use as a research tool, whereas the NCEP:ATP III definition is more appropriate for clinical practice. Clinicians prefer simplified means with which to assess patients and improve their healthcare management. The NCEP:ATP III requires only assessment of fasting blood glucose, whereas the WHO definition can require an oral glucose test. Moreover, an accurate assessment of insulin resistance to meet the WHO definition requires a more complicated test (e.g., the

<table>
<thead>
<tr>
<th>Criteria</th>
<th>WHO</th>
<th>NCEP:ATP III</th>
<th>IDF</th>
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<tbody>
<tr>
<td>Essential</td>
<td>Diabetes, IFG, IGT, or insulin resistance (assessed by clamp studies) and at least two of the following:</td>
<td>Three or more of the following risk factors:</td>
<td>Central obesity plus any two of the following:</td>
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<tr>
<td>Central obesity</td>
<td>Waist-to-hip ratio &gt;0.90 in men and &gt;0.85 in women or BMI &gt;30 kg/m²</td>
<td>Waist circumference &gt;102 cm (40 in) in men and &gt;88 cm (35 in) in women</td>
<td>Waist circumference ≥94 cm (37 in) in men and ≥80 cm (31 in) in women</td>
</tr>
<tr>
<td>Insulin resistance</td>
<td>Diabetes, IFG, IGT, or insulin resistance by clamp studies</td>
<td>FPG ≥100 mg/dL (5.6 mM)</td>
<td>FPG ≥100 mg/dL (5.6 mM) or previously diagnosed type II diabetes</td>
</tr>
<tr>
<td>Lipid profile</td>
<td>Serum triglycerides ≥150 mg/dL (1.7 mM) and/or HDL-C &lt;0.9 mmol/L (35 mg/dL) in men and &lt;1.0 mmol/L (39 mg/dL) in women</td>
<td>Triglycerides ≥150 mg/dL (1.7 mM). HDL-C &lt;40 mg/dL (1.03 mmol/L) in men and &lt;50 mg/dL (1.29 mmol/L) in women</td>
<td>Triglycerides ≥150 mg/dL (1.7 mM) or specific treatment for this lipid abnormality. HDL-C &lt;40 mg/dL (1.03 mmol/L) in males and &lt;50 mg/dL (1.29 mmol/L) in females or specific treatment for this lipid abnormality.</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Blood pressure ≥ 140/90 mmHg</td>
<td>Systolic BP ≥130 or diastolic BP ≥85 mmHg</td>
<td>Systolic BP ≥130 or diastolic BP ≥85 mmHg for previously diagnosed hypertension</td>
</tr>
<tr>
<td>Others</td>
<td>Urinary albumin excretion rate &gt;20 µg/min or albumin to creatinine ratio ≥30 mg/g</td>
<td></td>
<td>Additional metabolic criteria supportive of but not essential for the diagnosis</td>
</tr>
</tbody>
</table>

BMI = body mass index; BP = blood pressure; FPG = fasting plasma glucose; HDL-C = high-density lipoprotein cholesterol; IDF = International Diabetes Federation; IFG = impaired fasting glucose; IGT = impaired glucose tolerance; NCEP:ATP III = National Cholesterol Education Program Adult Treatment Panel III; WHO = World Health Organization.
hyperinsulinemic euglycemic clamp technique).

Because several definitions of the syndrome are in use by various investigators around the world, and indeed the number of definitions is much greater than what is expressed herein, it is difficult to compare prevalence and impact between countries. Fortunately, there is now a more rational approach. In May 2004, the International Diabetes Federation (IDF) recognized the need for a simple, easily applicable tool for diagnosis of the syndrome that could be used universally. IDF proposed its own unified definition for MetS, and highlighted areas where more research into the syndrome is needed. Interestingly, the IDF emphasized abdominal obesity as the initiating factor in MetS.

A similar process has been initiated jointly by the National Heart, Lung and Blood Institute (NHLBI) and the American Heart Association (AHA). Additional modifications of the NCEP-ATP III definition are expected to follow.

Ultimately, the combined efforts of the IDF and NHLBI/AHA definitions will no doubt result in a new definition(s) of MetS that will be suitable for use in clinical practice worldwide. Meanwhile, it will be wise for healthcare providers to consider all criteria in MetS in their totality, rather than to single out individual criteria as being more or less important for characterizing the syndrome. All criteria are important and all are relevant when considering the pathology involved in the syndrome and attempting to treat it.

Prevalence. Comparisons of published reports describing prevalence for different populations are difficult to interpret despite attempts to reach consensus on the definition of the syndrome. Many studies attempt to compare prevalence based on different criteria, akin to comparing apples to oranges. Perhaps their main achievement is to again reinforce the need for a standardized international definition.

Ethnic origin can modify the components of the MetS. An interesting example is to compare the prevalence in the U.S., with lower prevalence rates in non-Hispanic Caucasians compared with Mexican Americans, and in African-American men compared with non-Hispanic Caucasians and Mexican-American men.

A consistent finding also is that the prevalence is highly age-dependent. This pattern is clear in that, where the prevalence is less than 10 percent for both men and women in the 20- to 29-year age-group, it rises to 38 percent and 67 percent respectively, in the 60- to 69-year age-group. Similarly, in a French population, prevalence increases from 5.6 percent in the 30- to 39-year age-group to 17.5 percent in the 60- to 64-year age-group. Moreover, its prevalence in the U.S. has increased from 7 percent in participants aged 20 to 29 years, to 44 percent for those aged 60 to 69 years. When all factors are considered, estimates from the National Health and Nutrition Examination Survey III are that approximately 24 percent of all Americans meet three or more of the five MetS criteria, meaning they qualify for positive diagnosis of the syndrome.

Until recently, MetS was considered to be a disease of adults. However, there has been a renewed interest in MetS for children in association with increasing childhood obesity. The origins of MetS in adults can often be traced back to childhood. With increasing rates of overweight and obesity in young people, however, it is clear that MetS can onset at different ages in all ethnic groups.

Estimates of prevalence in younger age groups differ from those of adults because of the problem of not having an appropriate definition of the syndrome in this younger population. In the U.S., one group of medical researchers reported that the prevalence of MetS increased with severity of obesity and reached 50 percent in severely obese youngsters. Each half-unit increase in the body mass index (BMI; BMI = weight [kg] divided by height [m²]) was associated with increased risk of MetS in overweight and obese individuals, as was each unit of increase in insulin resistance. The prevalence of MetS increased significantly with increasing insulin resistance after adjustment for ethnic group and extent of obesity. The rate in children varied widely depending on a number of factors such as ethnicity, weight, and gender. C-reactive protein (CRP) – a substance produced by expanded adipose tissue mass – concentrations increased and adiponectin (an anti-inflammatory substance produced exclusively by adipocytes) concentrations decreased with increasing obesity. The investigators concluded that prevalence of MetS is high in obese children and adolescents, and increases with worsening obesity. Biomarkers (CRP, etc.) of increased risk for future cardiovascular events are already present in these youngsters.

Pathophysiology of Underlying Criteria of MetS

Insulin Resistance. The most widely accepted and unifying hypothesis to describe pathophysiology of MetS is that insulin resistance is its underlying cause. Insulin resistance can be defined as a state where there is a reduced biologic response for any given concentration of insulin. Insulin resistance and resulting hyperinsulinemia have been implicated in development of glucose intolerance (and progression to T2DM). Conversely, treatment and consequent improvement of insulin resistance have been shown to result in better outcomes in virtually all of these conditions. Insulin resistance has traditionally been defined with a gluco-centric view – that is, a defect in insulin action results in fasting hyperinsulinemia to maintain euglycemia (normal glucose concentration). Yet, even before fasting hyperinsulinemia develops, post-prandial hyperinsulinemia exists.

A major contributor to
development of insulin resistance is an overabundance of circulating fatty acids. Plasma albumin-bound free fatty acids are attained primarily from adipose tissue triglyceride stores that are released through the action of the cyclic-AMP-dependent enzyme lipase. Fatty acids are also derived via lipolysis of triglyceride-rich lipoproteins in tissues by the action of lipoprotein lipase.

Insulin has a key role to play in inhibition of lipolysis and in lipogenesis. When insulin resistance develops, there is an increase in lipolysis in adipose tissue and a decline in lipogenesis, resulting in excessive amounts of free fatty acids. Although free fatty acids can stimulate insulin secretion, prolonged exposure to excessive concentration of fatty acids can result in declining insulin secretion.

**Glucose Intolerance.** Defects in insulin action on glucose metabolism include deficiencies in its ability to suppress glucose production by the liver and kidney, and to mediate glucose uptake and metabolism in insulin sensitive tissues (i.e., muscle and adipose tissue). The relation between impaired fasting glucose or impaired glucose tolerance and insulin resistance is well supported by studies in humans and other primates, and in rodents. To compensate for defects in insulin action, insulin secretion and/or clearance must be modified to sustain euglycemia. If this compensation fails, defects in insulin secretion predominate.

**Obesity and Increased Waist Circumference.** The worldwide obesity epidemic has been the most important driving force in recognition of MetS. Despite the importance of obesity in describing the syndrome, it should be noted that patients of normal weight can also be insulin resistant and also experience hypertension or dyslipidemia.

Several definitions of MetS include waist circumference as a criterion. Mechanistically, a distinction between a large waist size due to increases in subcutaneous adipose tissue versus abdominal fat continues to be debated. Several decades ago, European investigators recognized that abdominal adiposity correlated with a higher risk of cardiovascular events. MetS can best be explained by viewing abdominal adipose tissue as an endocrine organ that releases into the circulation excess harmful free fatty acids, angiotensin II, adiponectin and plasminogen activator inhibitor (PAI-1). When there is increased abdominal adipose tissue, there is a higher rate of flux of adipose tissue-derived free fatty acids to the liver. Increased abdominal subcutaneous fat releases lipolysis products into the systemic circulation, and avoids more direct effects on hepatic metabolism (i.e., glucose production, lipid synthesis and PAI-1). Despite these potential differences in mechanisms related to excessive abdominal adipose tissue distribution, clinical diagnosis of MetS does not distinguish between increases in subcutaneous and abdominal fat. On the other hand, perhaps by a mechanism related to free fatty acid flux and metabolism, the relative predominance of visceral rather than subcutaneous adipose tissue with increasing waist circumference in Asians and Asian Indians renders the relative prevalence of the syndrome higher than in African-American men in whom subcutaneous fat predominates. Moreover, there is evidence that the elevated postprandial free fatty acid release in upper-body obese women originates from non-splanchnic upper body fat, rather than from the visceral depot. These findings suggest that visceral fat might be a marker for, but not the source of, excess postprandial free fatty acids in obesity.

Traditional means for determining adiposity, such as referring to relative weight tables provided by insurance companies, are important determinants of a variety of cardiovascular risk factors, especially T2DM, hypertension and dyslipidemia. BMI has become the measure commonly used to assess total adiposity. BMI is believed to play a secondary role, following abdominal adiposity, in development of several metabolic risk processes. Investigators who have studied the effects of total obesity as determined by BMI values versus abdominal obesity (waist or waist/hip) measurements on outcomes of MetS usually report that both measures of obesity are associated with increased risk of heart disease and T2DM.

During clinical assessment, three measurements should be assessed on all patients: height, weight, and maximal abdominal girth. The BMI should be calculated and the patient informed of his or her category: normal (BMI 18.5 to 25), overweight (BMI 25 to 30), or obese (BMI >30). According to NCEP:ATP III criteria, increased waist girth (≥35 in [88 cm] for women, or ≥40 in [102 cm] for men) defines excess abdominal adiposity.

**Dyslipidemia.** With increased free fatty acid flux to the liver, there is increased production of apolipoprotein B (apo B)-containing, triglyceride-rich, very low-density lipoproteins (VLDL). The effect of insulin on this process is somewhat complex. With insulin resistance, there is increased flux of free fatty acids to the liver to increase hepatic triglyceride synthesis. Under physiological conditions, however, insulin inhibits rather than increases secretion of VLDL into the systemic circulation. This response, in part, is an effect of insulin on the degradation of apo B. Yet insulin is also lipogenic, increasing the transcription and enzyme activity of many genes that modulate triglyceride biosynthesis. Whether or not this pathway remains operational during systemic insulin resistance has not been completely addressed. Moreover, insulin resistance could also reduce the concentrations of lipoprotein lipase in peripheral tissues (i.e., in adipose tissue more than muscle). This alteration in lipoprotein lipase, however, seems to contribute less to the hypertriglyceridemia than does overproduction of VLDL. Nevertheless, hypertriglyceridemia
is an excellent reflection of the insulin resistant condition and is one of the important criteria for diagnosis of MetS.

The other major lipoprotein disturbance in MetS is a reduction in high density lipoprotein cholesterol (HDL-C; “good cholesterol”). This reduction is a consequence of changes in HDL composition and metabolism. In the presence of hypertriglyceridemia, a decrease in the cholesterol content of HDL results from decreases in the cholesteryl ester content of the lipoprotein core with variable increases in triglyceride, making the particle small and dense, a function, in part, of cholesteryl ester transfer protein. This change in lipoprotein composition also results in increased clearance of HDL from the circulation. The association of these changes in HDL to insulin resistance is probably indirect, arising in concert with the changes in triglyceride-rich lipoprotein metabolism. Analysis of four prospective U.S. studies showed that each 1-mg/dL decrement in HDL-C was associated independently with a 2 to 3 percent increase in CHD risk.

Hypertension. The mechanism leading to hypertension in MetS is multifactorial and may be related, among other factors, to obesity and dietary intake. Insulin-related alteration in renal sodium handling and salt sensitivity, central activation of the sympathetic nervous system, along with angiotensin II and endothelin 1-mediated vasoconstriction might also contribute.

Correlation between insulin resistance and hypertension is well documented and relates to several different mechanisms. First, it is important to note that insulin is a vasodilator when given intravenously to individuals of normal weight, with additional effects on sodium reabsorption in the kidney. Evidence indicates that sodium reabsorption is increased in Caucasians with MetS, but not in persons of African or Asian descent. With insulin resistance, the vasodilatory effect of insulin can be lost, but the renal effect on sodium reabsorption continues. Fatty acids themselves can mediate vasoconstriction. Insulin also increases the activity of the sympathetic nervous system, an effect that might also be preserved with insulin resistance. However, insulin resistance contributes only modestly to the increased prevalence of hypertension in MetS.

Thrombogenicity. MetS predisposes to a prothrombotic state as a result of elevated fibrinogen levels along with decreased fibrinolytic activity due to increased PAI-1. Platelet function is also disturbed, leading to increased aggregation and thrombin generation.

Inflammation and Endothelial Dysfunction. MetS is a proinflammatory state. Insulin resistance and atherogenic dyslipidemia stimulate up-regulation of inflammatory substances including tumor necrosis factor-α, interleukin 6, and CRP, and a decrease in adiponectin. Overexpression of the inflammatory proteins further interferes with insulin signaling pathways, enhances lipid peroxidation and increases free fatty acid flux. Some of the risk factors listed for MetS are not included in Framingham risk scoring. These include abdominal obesity, a proinflammatory state (high levels of CRP, impaired fasting glucose or impaired glucose tolerance), and a prothrombotic state (high concentrations of fibrinogen). NCEP-ATP III guidelines do not recommend routine measurement of these risk factors, but list them as optional measures. If shown to be abnormal in patients with characteristics of MetS, the clinician has the option to adjust risk higher than estimated from Framingham risk scoring. An extremely useful and easy-to-use program for self-use (by all healthcare professionals and patients) to calculate the Framingham 10-year risk percentage for CHD as well as to provide treatment guidelines based on the latest clinical data is available at www.globalrph.com/atp_calc.htm.

Management of Patients with the MetS

Lifestyle Measures. Non-pharmacologic measures (lifestyle intervention), including diet to reduce body weight, is a mainstay in management of MetS. The most significant lifestyle characteristics associated with MetS include a diet that is high in calories along with a high content of saturated fats, and a lack of physical exercise. These are both behavioral patterns that occur frequently in the general population and are generally associated with above-average weight or obesity.

Regular exercise is the first step in treating MetS because it increases glucose metabolism by muscle to reduce hyperglycemia and insulin resistance, and helps in weight reduction. Dietary therapy has two components. The first is weight reduction, which is essentially a matter of achieving a sustained negative caloric balance. Available data indicate that even moderate weight loss (e.g., 5 percent of body weight) benefits patients with MetS by improving insulin action, thus lowering glucose and hemoglobin A1C (HbA1c) levels and often reducing doses of or the need for antidiabetic medications. Additionally, weight loss improves the cardiovascular risk profile by reducing blood pressure, triglycerides and cholesterol, as well as coagulation and antifibrinolytic and inflammatory markers. A review of studies conducted between 1996 and 2001 indicated a 25 percent reduction in mortality among obese patients with T2DM who intentionally lost weight, with a weight loss of 9 to 13 kg (20 to 28.5 lbs) being most protective. Lifestyle changes are, however, notoriously difficult to achieve and maintain. Furthermore, there is some evidence that weight loss is more difficult to achieve in obese patients with diabetes than in obese counterparts without diabetes.

The second is adherence to a dietary plan, which includes foods that are especially high in olive oil and nuts. One such plan termed
The Mediterranean diet reduces blood pressure, as well as fasting glucose and insulin secretion. In one study that lasted three months, it modestly increased HDL-C while decreasing triglyceride levels when compared with a low-fat diet. Applied specifically to patients with MetS over two years, the Mediterranean diet decreased body weight and inflammatory markers. In one study, subjects who adhered closely to the Mediterranean style of diet experienced a 20 percent lower risk of MetS. Healthy food choices together with regular exercise and not smoking reduce the risk of CHD, in part, through anti-inflammatory mechanisms. The Mediterranean diet, therefore, seems to be a good choice, usually very acceptable to patients. Its major disadvantage is that it does not aid in weight reduction, which requires exercise and a decreased caloric intake.

**Summary and Conclusions**

Metabolic syndrome is a rapidly expanding global epidemic. The syndrome is a combination of cardiovascular risk factors, such as obesity, hypertension, dyslipidemia and insulin resistance, that are associated with increased risk of morbidity and mortality. Insulin resistance and central obesity are increasingly recognized as primary constituents in the pathogenesis of MetS. Management of insulin resistance to achieve glycemic control commonly improves the outcome.

Aggressive lifestyle intervention can delay or even prevent the development of serious health problems in patients with MetS or one of the MetS components. Diets with decreased calories and saturated fat content to decrease body weight are critical to managing patients with MetS. The Mediterranean diet, which includes foods that are especially high in olive oil and nuts, is one such diet. Regular exercise is also critical in treating MetS.

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**Table 2**

**Key components of the Mediterranean diet**

<table>
<thead>
<tr>
<th>Component</th>
<th>Details</th>
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<tbody>
<tr>
<td>Getting plenty of exercise</td>
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<tr>
<td>Eating primarily plant-based foods, such as</td>
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<tr>
<td>fruits and vegetables, whole grains, legumes</td>
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<tr>
<td>and nuts</td>
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<tr>
<td>Replacing butter with healthy fats</td>
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<tr>
<td>such as olive oil and canola oil</td>
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<tr>
<td>Using herbs and spices instead of</td>
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<tr>
<td>salt to flavor foods</td>
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<tr>
<td>Limiting red meat to no more than</td>
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<tr>
<td>a few times a week</td>
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<tr>
<td>Eating fish and poultry at least</td>
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<tr>
<td>twice a week</td>
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<tr>
<td>Drinking red wine in moderation</td>
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<td>(optional)</td>
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</table>

The diet also recognizes the importance of enjoying meals with family and friends.

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This lesson is a knowledge-based CE activity and is targeted to pharmacists in all practice settings.

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The Metabolic Syndrome: Definition, Pathophysiology, and Scope of the Problem

1. At present, the leading cause of death in the U.S. is:
   a. coronary heart disease.
   b. diabetes mellitus.
   c. lung cancer.
   d. obstructive pulmonary disease.

2. The constellation of metabolic abnormalities that defines the metabolic syndrome (MetS) include all of the following EXCEPT:
   a. central obesity.    c. insulin resistance.
   b. glucose intolerance.  d. elevated HDL-C.

3. The syndrome referred to as the “deadly quartet,” DROP is Syndrome:
   a. D.   c. Y.
   b. X.   d. Z.

4. All of the following risk factors are included in the NCEP-ATP III MetS definition EXCEPT:
   a. FPG>100 mg/dL.
   b. systolic BP ≥130 mmHg.
   c. waist-to-hip ratio >0.90.
   d. triglycerides ≥150 mg/dL.

5. When comparing WHO, IDF and NCEP:ATP III criteria for defining MetS, which one is best suited for use as a research tool?
   a. WHO      c. NCEP:ATP III
   b. IDF

6. Which definition emphasized abdominal obesity as the initiating factor in MetS?
   a. WHO      c. NCEP:ATP III
   b. IDF

Completely fill in the lettered box corresponding to your answer.

1. [a] [b] [c] [d]  2. [a] [b] [c]  3. [a] [b] [c] [d]  4. [a] [b] [c] [d]  5. [a] [b] [c] [d]
   6. [a] [b] [c]  7. [a] [b] [c] [d]  8. [a] [b] [c] [d]  9. [a] [b] [c] [d]  10. [a] [b] [c] [d]
   11. [a] [b] [c] [d]  12. [a] [b] [c] [d]  13. [a] [b] [c] [d]  14. [a] [b] [c] [d]  15. [a] [b] [c] [d]

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