

## AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke: 2002 Update

### Consensus Panel Guide to Comprehensive Risk Reduction for Adult Patients Without Coronary or Other Atherosclerotic Vascular Diseases

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The initial Guide to the Primary Prevention of Cardiovascular Diseases was published in 1997 as an aid to healthcare professionals and their patients without established coronary artery disease or other atherosclerotic diseases.<sup>1</sup> It was intended to complement the American Heart Association (AHA)/American College of Cardiology (ACC) Guidelines for Preventing Heart Attack and Death in Patients with Atherosclerotic Cardiovascular Disease (updated<sup>2</sup>) and to provide the healthcare professional with a comprehensive approach to patients across a wide spectrum of risk. The imperative to prevent the first episode of coronary disease or stroke or the development of aortic aneurysm and peripheral arterial disease remains as strong as ever because of the still-high rate of first events that are fatal or disabling or require expensive intensive medical care. The evidence that most cardiovascular disease is preventable continues to grow. Results of long-term prospective studies consistently identify persons with low levels of risk factors as having lifelong low levels of heart disease and stroke.<sup>3,4</sup> Moreover, these low levels of risk factors are related to healthy lifestyles. Data from the Nurses Health Study,<sup>5</sup> for example, suggest that in women, maintaining a desirable body weight, eating a healthy diet, exercising regularly, not smoking, and consuming a moderate amount of alcohol could account for an 84% reduction in risk, yet only 3% of the women studied were in that category. Clearly, the majority of the causes of cardiovascular disease are known and modifiable.

This 2002 update of the Guide acknowledges a number of advances in the field of primary prevention since 1997. Research continues to refine the recommendations on detec-

tion and management of established risk factors, including evidence against the safety and efficacy of interventions once thought promising (eg, antioxidant vitamins).<sup>6</sup> This, in turn, has stimulated a large number of additional guidelines for specific demographic groups (eg, women), on individual risk factors (eg, diabetes, smoking), and for the primary prevention of stroke. In all of these guidelines, there is an increasing emphasis on further stratifying patients by level of risk and matching the intensity of interventions to the hazard for cardiovascular disease events.<sup>7</sup>

Therefore, this 2002 update of the Primary Prevention Guide serves to integrate other guidelines and consensus statements developed since the initial Guide's approval. This Guide might be viewed as the entry point to the more specific and detailed recommendations and the rationale behind them. The recommendations, as presented in the accompanying tables, are therefore consistent with the following recommendations: Agency for Healthcare Policy and Research Guidelines on Treating Tobacco Use and Dependence<sup>8</sup>; the Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI)<sup>9</sup>; the AHA Dietary Guidelines, Revision 2000<sup>10</sup>; the AHA Statement on Alcohol and Heart Disease<sup>11</sup>; the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults<sup>12</sup>; American Heart Association Scientific Statements and Advisories on Physical Activity<sup>13,14</sup> and the American College of Sports Medicine Guidelines<sup>15</sup>; the Clinical Guidelines for the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults from the

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**TABLE 1. Guide to Primary Prevention of Cardiovascular Disease and Stroke: Risk Assessment**

Risk Assessment	Recommendations
<b>Risk factor screening</b> Goal: Adults should know the levels and significance of risk factors as routinely assessed by their primary care provider.	Risk factor assessment in adults should begin at age 20 y. Family history of CHD should be regularly updated. Smoking status, diet, alcohol intake, and physical activity should be assessed at every routine evaluation. Blood pressure, body mass index, waist circumference, and pulse (to screen for atrial fibrillation) should be recorded at each visit (at least every 2 y). Fasting serum lipoprotein profile (or total and HDL cholesterol if fasting is unavailable) and fasting blood glucose should be measured according to patient's risk for hyperlipidemia and diabetes, respectively (at least every 5 y; if risk factors are present, every 2 y).
<b>Global risk estimation</b> All adults $\geq 40$ y of age should know their absolute risk of developing CHD. Goal: As low risk as possible.	Every 5 y (or more frequently if risk factors change), adults, especially those $\geq 40$ y of age or those with $\geq 2$ risk factors, should have their 10-y risk of CHD assessed with a multiple risk score. Risk factors used in global risk assessment include age, sex, smoking status, systolic (and sometimes diastolic) blood pressure, total (and sometimes LDL) cholesterol, HDL cholesterol, <sup>12,28</sup> and in some risk scores, diabetes. <sup>29,30</sup> Persons with diabetes or 10-y risk $>20\%$ can be considered at a level of risk similar to a patient with established cardiovascular disease (CHD risk equivalent). Equations for calculation of 10-y stroke risk are also available.

CHD indicates coronary heart disease.

National Heart, Lung, and Blood Institute Expert Panel<sup>16</sup> and an accompanying statement from the AHA Nutrition Committee<sup>17</sup>; the American Diabetes Association Standards of Medical Care for Patients with Diabetes<sup>18,19</sup> and the AHA Statement on Diabetes and Cardiovascular Disease<sup>20</sup>; the AHA Guidelines on the Primary Prevention of Stroke<sup>21</sup>; AHA Guidelines for Prevention of Cardiovascular Disease in Women<sup>22</sup>; ACC/AHA/European Society of Cardiology (ESC) Guidelines for the Management of Patients With Atrial Fibrillation<sup>23</sup>; the AHA Scientific Statement on Hormone Replacement Therapy and Cardiovascular Disease<sup>24</sup>; and the US Preventive Services Task Force evidence for use of aspirin in primary prevention.<sup>25</sup> The aspirin guidelines recommended here agree with the Task Force Report in the use of aspirin in persons at high coronary and stroke risk but use a  $\geq 10\%$  risk per 10 years rather than  $>6\%$  risk over 10 years. This improves the likelihood of a positive balance of coronary risk reduction over bleeding and hemorrhagic stroke caused by aspirin.<sup>26,27</sup>

Although this Guide largely applies to adults, it does identify high-risk patients for whom screening and intervention in first-degree relatives (including children) would be an important aspect of primary prevention. However, this Guide will not provide specific recommendations for the reduction of cardiovascular risk in children and adolescents. This important issue will be the subject of a separate guide. However, a family-centered approach to primary prevention should be emphasized, inasmuch as it recognizes both the genetic and behavioral causes of the well-established familial aggregation of heart disease and stroke.

This Guide is intended to assist primary care providers in their assessment, management, and follow-up of patients who may be at risk for but who have not yet manifested cardiovascular disease. The continuing message is that adoption of healthy life habits remains the cornerstone of primary prevention, including the avoidance of tobacco (including secondhand smoke), healthy dietary patterns, weight control, and regular, appropriate exercise. An important role of healthcare providers is to support and reinforce these public health recommendations for all patients.

Table 1 is presented to guide the identification and assess-

ment of modifiable risk. The assessment of absolute cardiac risk is increasingly advocated by international organizations and by individual risk factor guidelines in the United States.<sup>12,25,28</sup> The Framingham database has been widely used, though we acknowledge that the multiple risk score may not apply equally to all sex, race, and ethnic groups.<sup>29,30</sup> The use of more sophisticated technologies than a risk factor inventory and global risk score has been addressed,<sup>31</sup> and we conclude that most screening tests for occult atherosclerosis remain in the research arena, with the exception of the ankle-brachial blood pressure index. Similarly, those recommended interventions involving "nutriceutical" and pharmaceutical interventions in Table 2 have support from randomized clinical trials establishing their efficacy and safety. More controversial interventions, such as very low-fat diets,<sup>32</sup> dietary supplements,<sup>6,33</sup> and potentially cardioprotective drugs other than aspirin require additional investigation in well-designed clinical trials in persons without established cardiovascular disease.

The gap between which evidence-based interventions are recommended and what is actualized is large.<sup>34,35</sup> Guidelines, even when based on the best available evidence from randomized, controlled trials, cannot be successfully implemented without acceptance by the entire healthcare team, including physicians, nurses, nutritionists, and other healthcare professionals. A physician-patient partnership must be forged, on the physician's part by assessing and communicating risk and by codeveloping with the patient a plan of preventive action. New tools for providers are available to foster this partnership, such as the AHA's Get With the Guidelines.<sup>36</sup> Information for the public on cardiovascular and stroke risk factors is available on the AHA web site.<sup>37</sup>

The challenge for healthcare professionals is to engage greater numbers of patients, at an earlier stage of their disease, in comprehensive cardiovascular risk reduction with the use of interventions that are designed to circumvent or alleviate barriers to participation and adherence, so that many more individuals may realize the benefits that primary prevention can provide. The healthcare professional should create an environment supportive of risk factor change, including long-term reinforcement of adherence to lifestyle

**TABLE 2. Guide to Primary Prevention of Cardiovascular Disease and Stroke: Risk Intervention**

Risk Intervention and Goals	Recommendations
<b>Smoking</b> Goal: Complete cessation. No exposure to environmental tobacco smoke.	Ask about tobacco use status at every visit. In a clear, strong, and personalized manner, advise every tobacco user to quit. Assess the tobacco user's willingness to quit. Assist by counseling and developing a plan for quitting. Arrange follow-up, referral to special programs, or pharmacotherapy. Urge avoidance of exposure to secondhand smoke at work or home.
<b>BP control</b> Goal: <140/90 mm Hg; <130/85 mm Hg if renal insufficiency or heart failure is present; or <130/80 mm Hg if diabetes is present.	Promote healthy lifestyle modification. Advocate weight reduction; reduction of sodium intake; consumption of fruits, vegetables, and low-fat dairy products; moderation of alcohol intake; and physical activity in persons with BP of $\geq 130$ mm Hg systolic or 80 mm Hg diastolic. For persons with renal insufficiency or heart failure, initiate drug therapy if BP is $\geq 130$ mm Hg systolic or 85 mm Hg diastolic ( $\geq 80$ mm Hg diastolic for patients with diabetes). Initiate drug therapy for those with BP $\geq 140/90$ mm Hg if 6 to 12 months of lifestyle modification is not effective, depending on the number of risk factors present. Add BP medications, individualized to other patient requirements and characteristics (eg, age, race, need for drugs with specific benefits).
<b>Dietary intake</b> Goal: An overall healthy eating pattern.	Advocate consumption of a variety of fruits, vegetables, grains, low-fat or nonfat dairy products, fish, legumes, poultry, and lean meats. Match energy intake with energy needs and make appropriate changes to achieve weight loss when indicated. Modify food choices to reduce saturated fats (<10% of calories), cholesterol (<300 mg/d), and <i>trans</i> -fatty acids by substituting grains and unsaturated fatty acids from fish, vegetables, legumes, and nuts. Limit salt intake to <6 g/d. Limit alcohol intake ( $\leq 2$ drinks/d in men, $\leq 1$ drink/d in women) among those who drink.
<b>Aspirin</b> Goal: Low-dose aspirin in persons at higher CHD risk (especially those with 10-y risk of CHD $\geq 10\%$ ).	Do not recommend for patients with aspirin intolerance. Low-dose aspirin increases risk for gastrointestinal bleeding and hemorrhagic stroke. Do not use in persons at increased risk for these diseases. Benefits of cardiovascular risk reduction outweigh these risks in most patients at higher coronary risk. <sup>25–27</sup> Doses of 75–160 mg/d are as effective as higher doses. Therefore, consider 75–160 mg aspirin per day for persons at higher risk (especially those with 10-y risk of CHD of $\geq 10\%$ ).
<b>Blood lipid management</b> Primary goal: LDL-C <160 mg/dL if $\leq 1$ risk factor is present; LDL-C <130 mg/dL if $\geq 2$ risk factors are present and 10-y CHD risk is <20%; or LDL-C <100 mg/dL if $\geq 2$ risk factors are present and 10-y CHD risk is $\geq 20\%$ or if patient has diabetes. Secondary goals (if LDL-C is at goal range): If triglycerides are >200 mg/dL, then use non-HDL-C as a secondary goal: non-HDL-C <190 mg/dL for $\leq 1$ risk factor; non-HDL-C <160 mg/dL for $\geq 2$ risk factors and 10-y CHD risk $\leq 20\%$ ; non-HDL-C <130 mg/dL for diabetics or for $\geq 2$ risk factors and 10-y CHD risk >20%. Other targets for therapy: triglycerides >150 mg/dL; HDL-C <40 mg/dL in men and <50 mg/dL in women.	If LDL-C is above goal range, initiate additional therapeutic lifestyle changes consisting of dietary modifications to lower LDL-C: <7% of calories from saturated fat, cholesterol <200 mg/d, and, if further LDL-C lowering is required, dietary options (plant stanols/sterols not to exceed 2 g/d and/or increased viscous [soluble] fiber [10–25 g/d]), and additional emphasis on weight reduction and physical activity. If LDL-C is above goal range, rule out secondary causes (liver function test, thyroid-stimulating hormone level, urinalysis). After 12 weeks of therapeutic lifestyle change, consider LDL-lowering drug therapy if: $\geq 2$ risk factors are present, 10-y risk is >10%, and LDL-C is $\geq 130$ mg/dL; $\geq 2$ risk factors are present, 10-y risk is <10%, and LDL-C is $\geq 160$ mg/dL; or $\leq 1$ risk factor is present and LDL-C is $\geq 190$ mg/dL. Start drugs and advance dose to bring LDL-C to goal range, usually a statin but also consider bile acid-binding resin or niacin. If LDL-C goal not achieved, consider combination therapy (statin+resin, statin+niacin). After LDL-C goal has been reached, consider triglyceride level: If 150–199 mg/dL, treat with therapeutic lifestyle changes. If 200–499 mg/dL, treat elevated non-HDL-C with therapeutic lifestyle changes and, if necessary, consider higher doses of statin or adding niacin or fibrate. If >500 mg/dL, treat with fibrate or niacin to reduce risk of pancreatitis. If HDL-C is <40 mg/dL in men and <50 mg/dL in women, initiate or intensify therapeutic lifestyle changes. For higher-risk patients, consider drugs that raise HDL-C (eg, niacin, fibrates, statins).
<b>Physical activity</b> Goal: At least 30 min of moderate-intensity physical activity on most (and preferably all) days of the week.	If cardiovascular, respiratory, metabolic, orthopedic, or neurological disorders are suspected, or if patient is middle-aged or older and is sedentary, consult physician before initiating vigorous exercise program. Moderate-intensity activities (40% to 60% of maximum capacity) are equivalent to a brisk walk (15–20 min per mile). Additional benefits are gained from vigorous-intensity activity (>60% of maximum capacity) for 20–40 min on 3–5 d/wk. Recommend resistance training with 8–10 different exercises, 1–2 sets per exercise, and 10–15 repetitions at moderate intensity $\geq 2$ d/wk. Flexibility training and an increase in daily lifestyle activities should complement this regimen.
<b>Weight management</b> Goal: Achieve and maintain desirable weight (body mass index 18.5–24.9 kg/m <sup>2</sup> ). When body mass index is $\geq 25$ kg/m <sup>2</sup> , waist circumference at iliac crest level $\leq 40$ inches in men, $\leq 35$ inches in women.	Initiate weight-management program through caloric restriction and increased caloric expenditure as appropriate. For overweight/obese persons, reduce body weight by 10% in first year of therapy.
<b>Diabetes management</b> Goals: Normal fasting plasma glucose (<110 mg/dL) and near normal HbA1c (<7%).	Initiate appropriate hypoglycemic therapy to achieve near-normal fasting plasma glucose or as indicated by near-normal HbA1c. First step is diet and exercise. Second-step therapy is usually oral hypoglycemic drugs: sulfonylureas and/or metformin with ancillary use of acarbose and thiazolidinediones. Third-step therapy is insulin. Treat other risk factors more aggressively (eg, change BP goal to <130/80 mm Hg and LDL-C goal to <100 mg/dL).
<b>Chronic atrial fibrillation</b> Goals: Normal sinus rhythm or, if chronic atrial fibrillation is present, anticoagulation with INR 2.0–3.0 (target 2.5).	Irregular pulse should be verified by an electrocardiogram. Conversion of appropriate individuals to normal sinus rhythm. For patients in chronic or intermittent atrial fibrillation, use warfarin anticoagulants to INR 2.0–3.0 (target 2.5). Aspirin (325 mg/d) can be used as an alternative in those with certain contraindications to oral anticoagulation. Patients <65 y of age without high risk may be treated with aspirin.

BP indicates blood pressure; CHD, coronary heart disease; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; and INR, international normalized ratio.

and drug interventions. Practice-based systems for risk factor monitoring, reminders, and support services need to be established, reimbursed, and otherwise supported by managed care organizations and third-party payers. Primary prevention, by its very nature, requires a lifetime of interactions that virtually define successful provider-patient relationships.

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