

2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American Pharmacists Association, American Society for Nutrition, American Society for Preventive Cardiology, American Society of Hypertension, Association of Black Cardiologists, National Lipid Association, Preventive Cardiovascular Nurses Association, and WomenHeart: The National Coalition for Women With Heart Disease

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Preamble and Transition to ACC/AHA Guidelines to Reduce Cardiovascular Risk

The goals of the American College of Cardiology (ACC) and the American Heart Association (AHA) are to prevent cardiovascular diseases (CVDs); improve the management of people who have these diseases through professional education and research; and develop guidelines, standards, and policies that promote optimal patient care and cardiovascular health. Toward these objectives, the ACC and AHA have collaborated with the National Heart, Lung, and Blood Institute (NHLBI) and stakeholder and professional organizations to develop clinical practice guidelines for assessment of cardiovascular risk, lifestyle modifications to reduce cardiovascular risk, management of blood cholesterol in adults, and management of overweight and obesity in adults.

In 2008, the NHLBI initiated these guidelines by sponsoring rigorous systematic evidence reviews for each topic by expert panels convened to develop critical questions (CQs), interpret the evidence, and craft recommendations. In response to the 2011 report from the Institute of Medicine on the development of trustworthy clinical guidelines,¹ the NHLBI Advisory Council recommended that the NHLBI focus specifically on reviewing the highest-quality evidence and partner with other organizations to develop recommendations.^{2,3} Accordingly, in June 2013 the NHLBI initiated collaboration with the ACC and AHA to work with other organizations to complete and publish the 4 guidelines noted above and make them available to the widest possible constituency. Recognizing that the Expert Panels/Work Groups did not consider evidence beyond 2011 (except as specified in the methodology), the ACC, AHA, and collaborating societies plan to begin updating these guidelines starting in 2014.

The joint ACC/AHA Task Force on Practice Guidelines (Task Force) appointed a subcommittee to shepherd this transition, communicate the rationale and expectations to the writing panels and partnering organizations, and expeditiously publish the documents. The ACC/AHA and partner organizations recruited a limited number of expert reviewers for fiduciary examination of content, recognizing that each document had undergone extensive peer review by representatives of the NHLBI Advisory Council, key federal agencies, and scientific experts. Each writing panel responded to comments from these reviewers. Clarifications were incorporated where appropriate, but there were no substantive changes because the bulk of the content was undisputed.

Although the Task Force led the final development of these prevention guidelines, they differ from other ACC/AHA guidelines. First, as opposed to an extensive compendium of clinical information, these documents are significantly more limited in scope and focus on selected CQs on each topic, based on the highest-quality evidence available. Recommendations were derived from randomized trials, meta-analyses, and observational studies evaluated for quality and were not formulated when sufficient evidence was not available. Second, the text accompanying each recommendation is succinct, summarizing the evidence for each question. The Full Panel/Work Group Reports include more detailed information about the evidence statements (ESs) that serve as the basis for recommendations. Third, the format of the recommendations differs

from other ACC/AHA guidelines. Each recommendation has been mapped from the NHLBI grading format to the ACC/AHA Classification of Recommendation/Level of Evidence (COR/LOE) construct (Table 1) and is expressed in both formats. Because of the inherent differences in grading systems and the clinical questions driving the recommendations, alignment between the NHLBI and ACC/AHA formats is in some cases imperfect. Explanations of these variations are noted in the recommendation tables, where applicable.

In consultation with NHLBI, the policies adopted by the writing panels to manage relationships of authors with industry and other entities (RWI) are outlined in the methods section of each panel report. These policies were in effect when this effort began in 2008 and throughout the writing process and voting on recommendations, until the process was transferred to ACC/AHA in 2013. In the interest of transparency, the ACC/AHA requested that panel authors resubmit RWI disclosures as of July 2013. Relationships relevant to this guideline are disclosed in Appendix 1. None of the ACC/AHA expert reviewers had relevant RWI (Appendix 2). See Appendix 3 for a list of abbreviations used in the guideline.

Systematic evidence reports and accompanying summary tables were developed by the expert panels and NHLBI. The guideline was reviewed by the ACC/AHA Task Force and approved by the ACC Board of Trustees and the AHA Science Advisory and Coordinating Committee. In addition, ACC/AHA sought endorsement from other stakeholders, including professional organizations. It is the hope of the writing panels, stakeholders, professional organizations, NHLBI, and Task Force that the guidelines will garner the widest possible readership for the benefit of patients, providers, and the public health.

These guidelines are meant to define practices that meet the needs of patients in most circumstances and are not a replacement for clinical judgment. The ultimate decision about care of a particular patient must be made by the healthcare provider and patient in light of the circumstances presented by that patient. As a result, situations might arise in which deviations from these guidelines may be appropriate. These considerations notwithstanding, in caring for most patients, clinicians can employ the recommendations confidently to reduce the risks of atherosclerotic CVD events.

See Tables 2 and 3 for an explanation of the NHLBI recommendation grading methodology.

1.1. Scope of Guideline

See Table 4 for the Lifestyle Expert Work Group's CQs.

A healthy lifestyle is important in the prevention of CVD, the leading cause of morbidity and mortality worldwide. The intent of the Lifestyle Work Group (Work Group) was to evaluate evidence that particular dietary patterns, nutrient intake, and levels and types of physical activity can play a major role in CVD prevention and treatment through effects on modifiable CVD risk factors (ie, blood pressure [BP] and lipids). These ESs and recommendations may be used as appropriate in the management of hypercholesterolemia and hypertension. The target audience of the report is primary care providers.

This guideline is based on the Full Work Group Report, which is provided as an online-only data supplement to the

guideline. The [Full Work Group Report supplement](#) contains background and additional material related to content, methodology, evidence synthesis, rationale, and references and is supported by the NHLBI Systematic Evidence Review, which can be found at http://www.nhlbi.nih.gov/guidelines/cvd_adult/lifestyle/.

Diet and physical activity interventions of interest to the Work Group that were not included in this report because of time and resource limitations were the following: calcium, magnesium, alcohol, cardiorespiratory fitness, single behavioral intervention or multicomponent lifestyle interventions, the addition of lifestyle intervention to pharmacotherapy, and smoking. Outcomes of interest not covered in this evidence review were the following risk factors: diabetes mellitus (diabetes)- and obesity-related measurements, incident diabetes metabolic syndrome, high-sensitivity C-reactive protein, and other inflammatory markers. The Work Group was interested in reviewing the evidence for CVD outcomes in all of the CQs; however, the evidence for mortality and CVD outcomes was reviewed only in CQ2.

1.2. Methodology and Evidence Review

1.2.1. Scope of the Evidence Review

To formulate the nutrition recommendations, the Work Group used randomized controlled trials (RCTs), observational studies, meta-analyses, and systematic reviews of studies carried out in adults (≥ 18 years of age) with or without established coronary heart disease/CVD and with or without risk factors for coronary heart disease/CVD, who were of normal weight, overweight, or obese. The evidence review date range was 1998 to 2009. To capture historical data or more recent evidence, date ranges were changed for subquestions in some instances. The evidence date ranges are described clearly in each CQ section. The Work Group assessed the impact of both dietary patterns and macronutrient composition on plasma low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides and on systolic BP and diastolic BP over a minimum RCT intervention period of 1 month in studies performed in any geographic location and research setting.

Overall, the Work Group emphasized dietary patterns rather than individual dietary components. Patterns were characterized by habitual or prescribed combinations of daily food intake. Dietary patterns offer the opportunity to characterize the overall composition and quality of the eating behaviors of a population (eg, Mediterranean-style dietary [MED] pattern). Eating patterns consist of various combinations of foods that may differ in macronutrient, vitamin, and mineral compositions. The macronutrients saturated, *trans*, monounsaturated, and polyunsaturated fatty acids are particularly relevant for their effects on plasma lipids and lipoproteins. Dietary sodium and potassium are particularly relevant for their effects on BP. Epidemiological research has examined the dietary patterns of populations and identified associations between various patterns and CVD risk factors and outcomes. Intervention studies have tested *a priori* hypotheses involving prescribed dietary patterns specifically formulated on the basis of these data (eg, Dietary Approaches to Stop Hypertension [DASH] or MED

Table 1. Applying Classification of Recommendation and Level of Evidence

		SIZE OF TREATMENT EFFECT										
		CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/administered	CLASS IIa <i>Benefit >> Risk</i> Additional studies with <i>focused objectives</i> needed IT IS REASONABLE to perform procedure/administer treatment	CLASS IIb <i>Benefit ≥ Risk</i> Additional studies with <i>broad objectives</i> needed; additional registry data would be helpful Procedure/Treatment MAY BE CONSIDERED	CLASS III <i>No Benefit</i> or CLASS III <i>Harm</i> <table border="1" style="margin: 0 auto;"> <tr> <td></td> <td>Procedure/ Test</td> <td>Treatment</td> </tr> <tr> <td>COR III: No benefit</td> <td>Not Helpful</td> <td>No Proven Benefit</td> </tr> <tr> <td>COR III: Harm</td> <td>Excess Cost w/o Benefit or Harmful</td> <td>Harmful to Patients</td> </tr> </table>		Procedure/ Test	Treatment	COR III: No benefit	Not Helpful	No Proven Benefit	COR III: Harm
	Procedure/ Test	Treatment										
COR III: No benefit	Not Helpful	No Proven Benefit										
COR III: Harm	Excess Cost w/o Benefit or Harmful	Harmful to Patients										
ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	<ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Sufficient evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Greater conflicting evidence from multiple randomized trials or meta-analyses 	<ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Sufficient evidence from multiple randomized trials or meta-analyses 							
	LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	<ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Some conflicting evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Greater conflicting evidence from single randomized trial or nonrandomized studies 	<ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Evidence from single randomized trial or nonrandomized studies 							
	LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	<ul style="list-style-type: none"> Recommendation that procedure or treatment is useful/effective Only expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> Recommendation in favor of treatment or procedure being useful/effective Only diverging expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> Recommendation's usefulness/efficacy less well established Only diverging expert opinion, case studies, or standard of care 	<ul style="list-style-type: none"> Recommendation that procedure or treatment is not useful/effective and may be harmful Only expert opinion, case studies, or standard of care 							
Suggested phrases for writing recommendations		should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	COR III: No Benefit is not recommended is not indicated should not be performed/administered/other is not useful/beneficial/effective	COR III: Harm potentially harmful causes harm associated with excess morbidity/mortality should not be performed/administered/other						
Comparative effectiveness phrases [†]		treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B									

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Even when randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

†For comparative-effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

patterns). Population-based prospective cohort studies and RCTs suggest that there are healthier overall dietary patterns (foods and/or their constituent macronutrient, vitamin, and mineral combinations) that are associated with lower risk of chronic diseases, including CVD and risk factors such as type 2 diabetes and hypertension. We reviewed data exclusively on dietary intake rather than nutritional supplements provided in pharmaceutical preparations (eg, potassium pills), because nutritional supplements may not have similar effects and are not considered “lifestyle” interventions.

The Work Group focused on CVD risk factors to provide a free-standing Lifestyle document and to inform the Blood Cholesterol guideline⁴ and the hypertension panel. It also recognized that RCTs examining the effects on hard outcomes (myocardial infarction, stroke, heart failure, and CVD-related death) are difficult if not impossible to conduct for

several reasons (eg, long-term adherence to dietary changes). However, the Work Group also supplemented this evidence on risk factors with observational data on hard outcomes for sodium. The Work Group prioritized topics for the evidence review and was unable to review the evidence on hard outcomes for dietary patterns or physical activity.

For physical activity, substantial epidemiological evidence links higher levels of aerobic physical activity to lower rates of CVD and other chronic diseases, such as type 2 diabetes. Evidence indicates a dose-dependent inverse relationship between levels of physical activity and rates of CVD. The proposed mechanisms mediating the relationship between physical activity and decreased CVD rates include beneficial effects on lipids, lipoproteins, BP, and type 2 diabetes. The search for evidence related to physical activity and CVD included only systematic reviews and meta-analyses of RCTs or individual

Table 2. NHLBI Grading of the Strength of Recommendations

Grade	Strength of Recommendation*
A	Strong recommendation There is high certainty based on evidence that the net benefit† is substantial.
B	Moderate recommendation There is moderate certainty based on evidence that the net benefit is moderate to substantial, or there is high certainty that the net benefit is moderate.
C	Weak recommendation There is at least moderate certainty based on evidence that there is a small net benefit.
D	Recommendation against There is at least moderate certainty based on evidence that there is no net benefit or that risks/harms outweigh benefits.
E	Expert opinion (“There is insufficient evidence or evidence is unclear or conflicting, but this is what the Work Group recommends.”) Net benefit is unclear. Balance of benefits and harms cannot be determined because of no evidence, insufficient evidence, unclear evidence, or conflicting evidence, but the Work Group thought it was important to provide clinical guidance and make a recommendation. Further research is recommended in this area.
N	No recommendation for or against (“There is insufficient evidence or evidence is unclear or conflicting.”) Net benefit is unclear. Balance of benefits and harms cannot be determined because of no evidence, insufficient evidence, unclear evidence, or conflicting evidence, and the Work Group thought no recommendation should be made. Further research is recommended in this area.

*In most cases, the strength of the recommendation should be closely aligned with the quality of the evidence; however, under some circumstances, there may be valid reasons for making recommendations that are not closely aligned with the quality of the evidence (eg, strong recommendation when the evidence quality is moderate, such as smoking cessation to reduce cardiovascular disease risk or ordering an ECG as part of the initial diagnostic work-up for a patient presenting with possible MI). Those situations should be limited and the rationale explained clearly by the Work Group.

†Net benefit is defined as benefits minus risks/harms of the service/intervention. ECG indicates electrocardiogram; MI, myocardial infarction; and NHLBI, National Heart, Lung, and Blood Institute.

controlled clinical trials in adults (≥ 18 years of age) that were published from 2001 to 2011. For this CQ, the intervention was defined as physical activity interventions of any type.

Weight loss and maintenance are crucial for prevention and control of CVD risk factors. The Obesity Expert Panel simultaneously performed a systematic review of the evidence for weight management and CVD risk factors and outcomes.⁵ The primary intent of the Work Group's systematic review was to focus on the effects of diet and physical activity on CVD risk factors independent of effects on weight. Therefore, studies in which the primary outcome was weight loss or in which treatment was associated with $>3\%$ change in weight were excluded from the present review. However, the Work Group expects that recommendations from both evidence reviews will apply to many patients.

Because of limited resources and time, the Work Group could not review every study pertaining to lifestyle and CVD risk factors and outcomes. Priority was given to strong study design and a contemporaneous timeframe (1998 to 2009).

Table 3. NHLBI Quality Rating of the Strength of Evidence

Type of Evidence	Quality Rating*
<ul style="list-style-type: none"> Well-designed, well-executed† RCT that adequately represent populations to which the results are applied and directly assess effects on health outcomes. Meta-analyses of such studies. Highly certain about the estimate of effect. Further research is unlikely to change our confidence in the estimate of effect.	High
<ul style="list-style-type: none"> RCT with minor limitations‡ affecting confidence in, or applicability of, the results. Well-designed, well-executed nonrandomized controlled studies§ and well-designed, well-executed observational studies¶. Meta-analyses of such studies. Moderately certain about the estimate of effect. Further research may have an impact on our confidence in the estimate of effect and may change the estimate.	Moderate
<ul style="list-style-type: none"> RCT with major limitations. Nonrandomized controlled studies and observational studies with major limitations affecting confidence in, or applicability of, the results. Uncontrolled clinical observations without an appropriate comparison group (eg, case series, case reports). Physiological studies in humans. Meta-analyses of such studies. Low certainty about the estimate of effect. Further research is likely to have an impact on our confidence in the estimate of effect and is likely to change the estimate.	Low

*In some cases, other evidence, such as large all-or-none case series (eg, jumping from airplanes or tall structures), can represent high- or moderate-quality evidence. In such cases, the rationale for the evidence rating exception should be explained by the Work Group and clearly justified.

†“Well-designed, well-executed” refers to studies that directly address the question; use adequate randomization, blinding, and allocation concealment; are adequately powered; use intention-to-treat analyses; and have high follow-up rates.

‡Limitations include concerns with the design and execution of a study that result in decreased confidence in the true estimate of the effect. Examples of such limitations include but are not limited to: inadequate randomization, lack of blinding of study participants or outcome assessors, inadequate power, outcomes of interest that are not prespecified for the primary outcomes, low follow-up rates, and findings based on subgroup analyses. Whether the limitations are considered minor or major is based on the number and severity of flaws in design or execution. Rules for determining whether the limitations are considered minor or major and how they will affect rating of the individual studies will be developed collaboratively with the methodology team.

§Nonrandomized controlled studies refer to intervention studies where assignment to intervention and comparison groups is not random (eg, quasi-experimental study design).

¶Observational studies include prospective and retrospective cohort, case-control, and cross-sectional studies.

NHLBI indicates National Heart, Lung, and Blood Institute; and RCT, randomized controlled trials.

However, there were instances in which the evidence review was extended beyond that timeframe. Landmark evidence on the effect of fatty acids on lipids was included back to 1990. The sodium evidence review included evidence through April 2012, and the physical activity meta-analysis review was extended to May 2011. Given the expertise of Work Group

Table 4. Critical Questions

Critical Questions:	
CQ1.	Among adults*, what is the effect of dietary patterns and/or macronutrient composition on CVD risk factors, when compared with no treatment or with other types of interventions?
CQ2.	Among adults, what is the effect of dietary intake of sodium and potassium on CVD risk factors and outcomes, when compared with no treatment or with other types of interventions?
CQ3.	Among adults, what is the effect of physical activity on BP and lipids when compared with no treatment or with other types of interventions?

*Those ≥ 18 years of age and < 80 years of age.

BP indicates blood pressure; CQ, critical question; and CVD, cardiovascular disease.

members and their familiarity with the literature in this field, the Work Group is confident that a broader review would not substantially change our conclusions or recommendations.

The results of the Work Group systematic review are the 10 lifestyle recommendations (8 dietary and 2 physical activity recommendations) (Table 5). Because the Work Group was convened to inform the development of clinical guidelines, and because most data meeting our criteria for review were derived from studies of high-risk populations, these recommendations are directed at patients with CVD risk factors (ie, abnormal lipids and/or prehypertension and hypertension). The majority of adults in the United States currently have ≥ 1 of these risk factors (33.5% with elevated LDL-C; 27.3%, hypertension; 31%, prehypertension; and 11.3%, diabetes), with risk factors increasing with age.⁶ The Work Group encourages heart-healthy nutrition and physical activity behaviors for all adults (Section 5.6) (Table 17).

For both BP and lipids, most studies of diet and/or physical activity exclude people taking antihypertensive or lipid-lowering medications. Although there is no direct evidence, it is reasonable to expect that the beneficial effects of these lifestyle recommendations apply to those taking such medications and that following these recommendations can potentially lead to better BP and lipid control in those taking medications and/or reduced medication needs. The recommendations apply to adults < 80 years of age with and without CVD.

1.2.2. CQ-Based Approach

The Work Group developed an initial set of questions based on their expertise and a brief literature review to identify topics of the greatest relevance and impact for the target audience of the guideline: primary care providers. Because of time and resource limitations, the Work Group prioritized the 3 CQs in Table 4.

The body of this report is organized by CQ. For each CQ:

- The rationale for its selection is provided, and methods are described.
- The ESs are presented, which include a rating for quality, a rationale that supports each item of evidence, and a statement. A detailed description of methods is provided in the NHLBI Lifestyle Systematic Evidence Review Report (http://www.nhlbi.nih.gov/guidelines/cvd_adult/lifestyle/).

The [Full Work Group Report supplement](#) presents documentation for search strategies and results from the search of the published literature.

- Recommendations include recommendation strength, accompanied by a summary of how the recommendation derives from the evidence and a discussion of issues considered by the Work Group in formulating the recommendation. The ACC/AHA COR/LOE ratings have also been added.

The ESs and recommendations are presented by CQ and grouped by topic:

- CQ1 presents evidence on dietary patterns and macronutrients and their effect on BP and lipids. The dietary recommendations for LDL-C lowering are described at the end of CQ1.
- CQ2 presents the evidence on the effect of dietary sodium and potassium intake on BP and CVD outcomes. The dietary recommendations for BP lowering are located at the end of CQ2.
- Finally, CQ3 presents evidence on the effect of physical activity on lipids and BP and physical activity recommendations for BP and lipid lowering. The physical activity recommendations for BP and lipid lowering are located at the end of CQ3.

It should be recognized that formulating recommendations derived from evidence reviews in response to CQs has some advantages as well as limitations. Because of its desire to adhere to the highest quality of evidence, the Work Group was restricted to using evidence that met inclusion/exclusion and quality criteria established by the Work Group in partnership with the methodologists. When the phrase “there is insufficient evidence” is used, the reader must distinguish between “insufficient” evidence where no studies meeting inclusion/exclusion and quality criteria were found to answer a CQ and “insufficient” evidence where RCTs or observational studies were conducted but the available data do not provide sufficient information to formulate a recommendation. This perspective is important because clinicians could see fewer recommendations derived from expert opinion. Given this perspective, the clinical and research community can identify research questions that need to be answered in the future to refine recommendations when updates to the guideline are written (Section 6).

1.3. Organization of Work Group

The Work Group was composed of 12 members and 4 ex-officio members, including physicians and experts in BP, blood cholesterol, obesity, and lifestyle management. The authors came from the primary care, nursing, pharmacology, nutrition, exercise, behavioral science, and epidemiology disciplines and also included senior scientific staff from NHLBI and the National Institutes of Health.

1.4. Document Reviews and Approval

A formal peer review process initially was completed under the auspices of the NHLBI and included 6 expert reviewers and representatives of federal agencies. This document was

Table 5. Summary of Recommendations for Lifestyle Management

Recommendations	NHLBI Grade	NHLBI Evidence Statements	ACC/AHA COR	ACC/AHA LOE
DIET				
LDL-C: Advise adults who would benefit from LDL-C lowering* to:				
1. Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limits intake of sweets, sugar-sweetened beverages, and red meats. a. Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes). b. Achieve this pattern by following plans such as the DASH dietary pattern, the USDA Food Pattern, or the AHA Diet.	A (Strong)	CQ1: ES4 (high), ES6 (low), ES8 (moderate), ES9 (moderate)	I	A
2. Aim for a dietary pattern that achieves 5%–6% of calories from saturated fat.	A (Strong)	CQ1: ES11 (high)	I	A
3. Reduce percent of calories from saturated fat.	A (Strong)	CQ1: ES11 (high), ES12 (moderate), ES13 (moderate)	I	A
4. Reduce percent of calories from <i>trans</i> fat.	A (Strong)	CQ1: ES14 (moderate), ES15 (moderate)	I	A
BP: Advise adults who would benefit from BP lowering to:				
1. Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limits intake of sweets, sugar-sweetened beverages, and red meats. a. Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes). b. Achieve this pattern by following plans such as the DASH dietary pattern, the USDA Food Pattern, or the AHA Diet.	A (Strong)	CQ1: ES1 (low) ES3 (high), ES5 (high), ES6 (low), ES7 (low), ES8 (moderate)	I	A
2. Lower sodium intake.	A (Strong)	CQ2: ES1 (high), ES2 (moderate), ES3 (high), ES4 (high), ES5 (high), ES8 (low), ES9 (low)	I	A
3. a. Consume no more than 2400 mg of sodium/d; b. Further reduction of sodium intake to 1500 mg/d can result in even greater reduction in BP; and c. Even without achieving these goals, reducing sodium intake by at least 1000 mg/d lowers BP.	B (Moderate)	CQ2: ES2 (moderate), ES3 (high)	Ia	B
4. Combine the DASH dietary pattern with lower sodium intake.	A (Strong)	CQ1: ES3 (high), ES5 (high), ES8 (moderate) CQ2: ES1 (high), ES2 (moderate), ES3 (high), ES4 (high), ES5 (high), ES6 (moderate)	I	A
PHYSICAL ACTIVITY				
Lipids				
1. In general, advise adults to engage in aerobic physical activity to reduce LDL-C and non-HDL-C: 3–4 sessions per wk, lasting on average 40 min per session, and involving moderate- to vigorous-intensity physical activity.	B (Moderate)	CQ3: ES1 (moderate), ES2 (moderate), ES5 (low)	Ia	A
BP				
1. In general, advise adults to engage in aerobic physical activity to lower BP: 3–4 sessions per wk, lasting on average 40 min per session, and involving moderate- to vigorous-intensity physical activity.	B (Moderate)	CQ3: ES1 (high)	Ia	A

*Refer to 2013 Blood Cholesterol Guideline for guidance on who would benefit from LDL-C lowering.⁴

ACC indicates American College of Cardiology; AHA, American Heart Association; BP, blood pressure; COR, Class of Recommendation; CQ, critical question; DASH, Dietary Approaches to Stop Hypertension; ES, evidence statement; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LOE, Level of Evidence; NHLBI, National Heart, Lung, and Blood Institute; and USDA, US Department of Agriculture.

also reviewed by 4 expert reviewers nominated by the ACC and the AHA when the management of the guideline transitioned to the ACC/AHA. The ACC and AHA reviewers' RWI information is published in this document (Appendix 2).

This document was approved for publication by the governing bodies of the ACC and AHA and endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American Pharmacists Association, American Society for Nutrition, American Society for Preventive Cardiology, American Society of Hypertension, Association of Black Cardiologists, National Lipid Association, Preventive Cardiovascular Nurses Association, and WomenHeart: The National Coalition for Women With Heart Disease.

2. Lifestyle Management Recommendations

See Table 5 for the Summary of Lifestyle Recommendations.

3. CQ1—Dietary Patterns and Macronutrients: BP and Lipids

See Table 6 for the CQ for BP and lipids with dietary patterns and macronutrients.

3.1. Introduction/Rationale

The importance of nutrition in modifying the risk of CVD has been repeatedly emphasized.^{7–11} Historically, the role of dietary components has been the predominant focus; however, foods are typically consumed in combinations rather than individually. Over the past few years, increasing attention has been given to dietary patterns and their relationship to health outcomes such as CVD.^{12–20}

In intervention studies, specific dietary patterns of defined macronutrient composition are identified on the basis of expert evidence and *a priori* hypothesis (eg, the DASH or MED patterns) and then are evaluated in RCTs. In observational studies, associations between intake and risk factors are assessed. Because of resource limitations, CVD morbidity and mortality outcomes were not included in the evidence review for this question. The charge of the Work Group was to inform the treatment of lipids and BP; therefore, those risk factors were the outcomes of focus.

3.2. Selection of Inclusion/Exclusion Criteria

Work Group members developed eligibility criteria based on a Population, Intervention, Comparator, Outcomes, Timing, and Setting (PICOTS) approach for screening potential studies for inclusion in this evidence review. The details of the PICOTS approach for CQ1 and Literature Search Yield, including summary tables, are available in the [Full Work Group Report supplement](#).

3.3. Literature Search Yield

3.3.1. Dietary Pattern/Macronutrient Composition Evidence
In all, 17 studies (28 articles) satisfied the final inclusion criteria and were rated to be of good or fair quality.^{21–48}

The Dietary Pattern Summary Tables (Tables B–1 through B–8) are available in the [Full Work Group Report supplement](#). The tables present summary data on the included

Table 6. CQ for Dietary Patterns and Macronutrients: BP and Lipids

CQ1:

Among adults, what is the effect of dietary patterns and/or macronutrient composition on CVD risk factors, when compared with no treatment or with other types of interventions?

BP indicates blood pressure; CQ, critical question; and CVD, cardiovascular disease.

studies organized by dietary pattern/macronutrient composition or subpopulations of interest, defined by age, sex, race, or comorbid condition. Some studies appear in more than 1 summary table because they address more than 1 corresponding macronutrient composition or dietary pattern comparison.

3.4. CQ1 Evidence Statements

3.4.1. Dietary Patterns

3.4.1.1. MED Pattern

MED pattern description (Table 7): There was no uniform definition of the MED diet in the RCTs and cohort studies examined. The most common features of diets in these studies were that they were higher in fruits (particularly fresh), vegetables (emphasizing root and green varieties), whole grains (cereals, breads, rice, or pasta), and fatty fish (rich in omega-3 fatty acids); were lower in red meat (emphasizing lean meats); substituted lower-fat or fat-free dairy products for higher-fat dairy foods; and used oils (olive or canola), nuts (walnuts, almonds, or hazelnuts), or margarines blended with rapeseed or flaxseed oils in lieu of butter and other fats. The MED patterns examined tended to be moderate in total fat (32% to 35% of total calories), relatively low in saturated fat (9% to 10% of total calories), high in fiber (27 to 37 g/d), and high in polyunsaturated fatty acids (particularly omega-3s).

3.4.1.2. DASH Dietary Pattern

DASH dietary pattern description (Table 8): The DASH dietary pattern is high in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, and nuts and is low in sweets,

Table 7. ESs for BP and Lipids With the MED Pattern

BP

ES1.

- Counseling to eat a MED pattern, as compared with minimal advice to consume a low-fat dietary pattern, in free-living middle-aged or older adults (with type 2 diabetes or at least 3 CVD risk factors) reduced BP by 6–7/2–3 mm Hg. In an observational study of healthy younger adults, adherence to a MED pattern was associated with lower BP (2–3/1–2 mm Hg).

Strength of Evidence: Low

Lipids

ES2.

- Counseling to eat a MED pattern, compared with minimal or no dietary advice, in free-living middle-aged or older adults (with or without CVD or at high risk for CVD) resulted in no consistent effect on plasma LDL-C, HDL-C, and triglycerides, in part because of substantial differences and limitations in the studies.

Strength of Evidence: Low

BP indicates blood pressure; CVD, cardiovascular disease; ES, evidence statement; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; and MED, Mediterranean-style dietary pattern.

Table 8. ESs for BP and Lipids With the DASH Pattern

BP
ES3.
<ul style="list-style-type: none"> When all food was supplied to adults with BP 120–159/80–95 mm Hg and both body weight and sodium intake were kept stable, the DASH dietary pattern, compared with a typical American diet of the 1990s, lowered BP by 5–6/3 mm Hg. <p><i>Strength of Evidence: High</i></p>
Lipids
ES4.
<ul style="list-style-type: none"> When food was supplied to adults with a total cholesterol level <260 mg/dL and LDL-C level <160 mg/dL and body weight was kept stable, the DASH dietary pattern, compared with a typical American diet of the 1990s, lowered LDL-C by 11 mg/dL, lowered HDL-C by 4 mg/dL, and had no effect on triglycerides. <p><i>Strength of Evidence: High</i></p>
DASH DIETARY PATTERN SUBPOPULATIONS
Subpopulations and BP
ES5.
<ul style="list-style-type: none"> When all food was supplied to adults with BP 120–159/80–95 mm Hg and body weight was kept stable, the DASH dietary pattern, compared with the typical American diet of the 1990s, lowered BP in women and men, African-American and non-African-American adults, older and younger adults, and hypertensive and nonhypertensive adults. <p><i>Strength of Evidence: High</i></p>
Subpopulations and Lipids
ES6.
<ul style="list-style-type: none"> When all food was supplied to adults with a total cholesterol level <260 mg/dL and LDL-C level <160 mg/dL and body weight was kept stable, the DASH dietary pattern, compared with a typical American diet of the 1990s, lowered LDL-C similarly in subgroups: African-American and non-African-American adults and hypertensive and nonhypertensive adults. <p><i>Strength of Evidence: Low</i></p>
ES7.
<ul style="list-style-type: none"> When all food was supplied to adults with a total cholesterol level <260 mg/dL and LDL-C level <160 mg/dL and body weight was kept stable, the DASH dietary pattern, compared with a typical American diet of the 1990s, lowered HDL-C similarly in subgroups: African-American and non-African-American adults, hypertensive and nonhypertensive adults, and men and women. <p><i>Strength of Evidence: Low</i></p>

BP indicates blood pressure; DASH, Dietary Approaches to Stop Hypertension; ES, evidence statement; HDL-C, high-density lipoprotein cholesterol.

sugar-sweetened beverages, and red meats. The DASH dietary pattern is low in saturated fat, total fat, and cholesterol. It is rich in potassium, magnesium, and calcium, as well as protein and fiber.

3.4.1.3. DASH Variations

DASH variations description (Table 9): In OmniHeart (Optimal Macronutrient Intake Trial for Heart Health), 2 variations of the DASH dietary pattern were compared with DASH: one that replaced 10% of total daily energy from carbohydrate with protein, and another that replaced the same amount of carbohydrate with unsaturated fat. These patterns were studied in an adequately powered crossover trial of 164 adults in which the participants were given all of their daily food.

3.4.2. Dietary Fat and Cholesterol

See Table 10 for ESs for saturated fat, *trans* fat, and dietary cholesterol.

3.5. Diet Recommendations for LDL-C Lowering*

The following diet recommendations for LDL-C lowering are based on the ESs from CQ1 on dietary patterns and fatty acids. Diet recommendations for BP lowering are based on CQ1 and CQ2 and are located after the CQ2 ESs. The physical activity and lipids ESs and recommendations are located in CQ3.

1. Advise adults who would benefit from LDL-C lowering to:

- Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, non-tropical vegetable oils, and nuts; and limits intake of sweets, sugar-sweetened beverages, and red meats.
 - Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes).
 - Achieve this pattern by following plans such as the DASH dietary pattern, the US Department of Agriculture (USDA) Food Pattern, or the AHA Diet.

NHLBI Grade: A (Strong); ACC/AHA COR: I, LOE: A

Rationale: This recommendation is based largely on studies of the DASH dietary pattern (DASH and DASH-Sodium), which provided the highest-quality evidence for a dietary pattern causing improvements in BP and lipid profiles (Tables 8 and 9). The LDL-C-lowering effect has been demonstrated in men and women, African Americans and non-African Americans, and in adults of all ages (Table 8, ES6). The evidence suggests that the effects of the recommended dietary pattern persist as long as the pattern is consumed.

The caloric (energy) intake should be appropriate for the individual (eg, restricted for those attempting weight loss). Patients should also be encouraged to adapt the recommended dietary pattern to their personal and cultural preferences. Materials are available to assist patients in achieving the recommended dietary pattern at different calorie levels (Table 9). The 2010 US Department of Health and Human Services Dietary Guidelines for Americans recommend the USDA Food Pattern and the DASH dietary pattern.⁴⁹ Overall, the recommended dietary pattern is consistent with the AHA Diet⁵⁰ and the USDA Food Pattern.⁴⁹ The USDA Food Pattern offers lacto-ovo vegetarian and vegan adaptations. Therefore, this recommendation is consistent with other national guidelines. Clinicians should be familiar with the recommendations, advise their patients to adopt them, and provide easy access to information (Table 11). Dietary planning and nutritional counseling are often facilitated by referral to a nutrition professional.

2. Advise adults who would benefit from LDL-C lowering to:

- Aim for a dietary pattern that achieves 5% to 6% of calories from saturated fat.

NHLBI Grade: A (strong); ACC/AHA COR: I, LOE: A

Rationale: As described in Table 10, ES11 there is strong evidence that reductions in LDL-C were achieved when dietary

*Refer to the 2013 Blood Cholesterol Guideline⁴ for guidance on who would benefit from LDL-C lowering.

Table 9. ESs for DASH Variations/Glycemic Index/Load Dietary Approaches

BP

ES8.

- In adults with BP of 120–159/80–95 mmHg, modifying the DASH dietary pattern by replacing 10% of calories from carbohydrates with the same amount of either protein or unsaturated fat (8% monounsaturated and 2% polyunsaturated) lowered systolic BP by 1 mmHg compared with the DASH dietary pattern. Among adults with BP 140–159/90–95 mmHg, these replacements lowered systolic BP by 3 mmHg relative to DASH.

Strength of Evidence: Moderate

Lipids

ES9.

- In adults with average baseline LDL-C level of 130 mg/dL, HDL-C level of 50 mg/dL, and triglyceride level of 100 mg/dL, modifying the DASH dietary pattern by replacing 10% of calories from carbohydrates with 10% of calories from protein lowered LDL-C by 3 mg/dL, HDL-C by 1 mg/dL, and triglycerides by 16 mg/dL compared with the DASH dietary pattern. Replacing 10% of calories from carbohydrates with 10% of calories from unsaturated fat (8% monounsaturated and 2% polyunsaturated) lowered LDL-C similarly, increased HDL-C by 1 mg/dL, and lowered triglycerides by 10 mg/dL as compared with the DASH dietary pattern.

Strength of Evidence: Moderate

ES10.

- There is insufficient evidence to determine whether low-glycemic diets versus high-glycemic diets affect lipids or BP for adults without diabetes. The evidence for this relationship in adults with diabetes was not reviewed.

Strength of Evidence: Insufficient

BP indicates blood pressure; DASH, Dietary Approaches to Stop Hypertension; ES, evidence statement; HDL-C, high-density lipoprotein cholesterol; and LDL-C, low-density lipoprotein cholesterol.

patterns were followed in which saturated fat intake was reduced from 14% to 15% of calories to 5% to 6%. As previously noted, these studies did not isolate the effect of saturated fat on LDL-C lowering. Intake of saturated fat has decreased in the United States over the past few decades and is currently estimated at 11% of energy in the US population ≥ 2 years of age.⁵¹ However, this level of saturated fat intake is higher than that tested in the DASH and DELTA (Dietary Effects on Lipoproteins and Thrombogenic Activity) trials (5% to 6%) and is not consistent with consuming a diet rich in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, legumes and nuts, and vegetable oils and limited in sweets, sugar-sweetened beverages, and red meat. Given the current average intake of saturated fat at 11% of calories, it would be beneficial for those who would benefit from LDL-C lowering to decrease saturated fat intake to 5% to 6% of calories.

3. Advise adults who would benefit from LDL-C lowering to:

- Reduce percentage of calories from saturated fat.

NHLBI Grade: A (strong); ACC/AHA COR: I, LOE: A

Rationale: Reducing saturated fat intake lowers both LDL-C and HDL-C. Because the absolute effect tends to be greater for LDL-C than HDL-C, reducing saturated fat intake has a beneficial effect on the lipid profile. Given that reducing saturated fat intake lowers LDL-C regardless of whether the saturated

fat is replaced by carbohydrate, monounsaturated fatty acids, or polyunsaturated fatty acids, the Work Group does not specify which of these 3 macronutrients should be substituted in place of saturated fat. However, favorable effects on lipid profiles are greater when saturated fat is replaced by polyunsaturated fatty acids, followed by monounsaturated fatty acids, and then carbohydrates. It is important to note that there are various types and degrees of refinement of carbohydrates. Substitution of saturated fat with whole grains is preferable to refined carbohydrates. For American adults who eat more saturated fat than the current average, some reduction is warranted, and adhering to a “heart-healthy” dietary pattern from dietary recommendation No. 1 for LDL-C lowering will likely result in a reduction of saturated fat.

4. Advise adults who would benefit from LDL-C lowering to:

- Reduce percentage of calories from *trans* fat.

NHLBI Grade: A (strong); ACC/AHA COR: I, LOE: A

Rationale: Reducing intake of *trans* fatty acids lowers LDL-C, with little or no effect on HDL-C or triglycerides levels. The direction of the relationship between *trans* fatty acids and LDL-C is consistent, regardless of whether the *trans* fatty acids are replaced by carbohydrates, monounsaturated fatty acids, or polyunsaturated fatty acids. Using 2003 to 2006 NHANES (National Health and Nutrition Examination Survey) data, intake of *trans* fat from partially hydrogenated oils was estimated at a mean of 1.3 g/d to 1.6 g/d among the US population ≥ 2 years of age.⁵² Although the intake level appears low, certain subgroups within the US population may still be consuming relatively high levels of *trans* fatty acids. For this reason, the Work Group recommends that emphasis continue to be placed on the reduction of *trans* fat in the diet. Even if intake of *trans* fat from partially hydrogenated oils decreases, naturally occurring *trans* fatty acids in the form of ruminant fat from meat and dairy products may still be present in small amounts in the US diet. Adhering to the recommendation to reduce dietary sources of saturated fat (meat and dairy fat) will result in additional reductions in *trans* fat intake.

4. CQ2—Sodium and Potassium: BP and CVD Outcomes

See Table 12 for the CQ on BP and CVD outcomes with sodium and potassium.

4.1. Introduction and Rationale

Vitamins and minerals typically are consumed in foods. However, it is sometimes possible to isolate the effect of individual minerals to determine the effects on health outcomes. Therefore, the Work Group decided that a systematic review was warranted to determine the individual effects of the minerals sodium and potassium, which are associated with CVD risk factors and outcomes. Other minerals, such as calcium and magnesium, were also considered but were not included in the systematic review because their consumption is limited to relatively few specific foods or food groups (eg, calcium and dairy products); furthermore, it was unlikely that a

Table 10. ESs for Dietary Fat and Cholesterol

Saturated Fat

ES11.

- When food was supplied to adults in a dietary pattern that achieved a macronutrient composition of 5%–6% saturated fat, 26%–27% total fat, 15%–18% protein, and 55%–59% carbohydrate compared with the control diet (14%–15% saturated fat, 34%–38% total fat, 13%–15% protein, and 48%–51% carbohydrate), LDL-C was lowered 11–13 mg/dL in 2 studies and 11% in another study.

Strength of Evidence: High

ES12.

- In controlled feeding trials among adults, for every 1% of energy from SFA that is replaced by 1% of energy from carbohydrate, MUFA, or PUFA:
 - LDL-C is lowered by an estimated 1.2, 1.3, and 1.8 mg/dL, respectively.
 - HDL-C is lowered by an estimated 0.4, 1.2, and 0.2 mg/dL, respectively.
- For every 1% of energy from SFA that is replaced by 1% of energy from:
 - Carbohydrate and MUFA:
 - Triglycerides are raised by an estimated 1.9 and 0.2 mg/dL, respectively.
 - PUFA:
 - Triglycerides are lowered by an estimated 0.4 mg/dL.

Strength of Evidence: Moderate

ES13.

- In controlled feeding trials among adults, for every 1% of energy from carbohydrate that is replaced by 1% of energy from:
 - MUFA:
 - LDL-C is lowered by 0.3 mg/dL, HDL-C is raised by 0.3 mg/dL, and triglycerides are lowered by 1.7 mg/dL.
 - PUFA:
 - LDL-C is lowered by 0.7 mg/dL, HDL-C is raised by 0.2 mg/dL, and triglycerides are lowered by 2.3 mg/dL.

Strength of Evidence: Moderate

Trans Fat

ES14.

- In controlled feeding trials among adults, for every 1% of energy from *trans* monounsaturated fatty acids replaced with 1% of energy from:
 - MUFA or PUFA
 - LDL-C is lowered by 1.5 mg/dL and 2.0 mg/dL, respectively.
 - SFA, MUFA, or PUFA:
 - HDL-C is increased by an estimated 0.5, 0.4, and 0.5 mg/dL, respectively.
 - MUFA or PUFA:
 - Triglycerides are decreased by an estimated 1.2 and 1.3 mg/dL.

Strength of Evidence: Moderate

ES15.

- In controlled feeding trials among adults, the replacement of 1% of energy as *trans* monounsaturated fatty acids with carbohydrate decreased LDL-C levels by 1.5 mg/dL and had no effect on HDL-C cholesterol and triglyceride levels.

Strength of Evidence: Moderate

Dietary Cholesterol

ES16.

- There is insufficient evidence to determine whether lowering dietary cholesterol reduces LDL-C.

Strength of Evidence: Insufficient

ES indicates evidence statement; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; and SFA, saturated fatty acid.

recommendation to increase or decrease consumption of the mineral rather than the food could be implemented.

In contrast, sodium was reviewed as a single nutrient because little sodium is found naturally in food, and it is added to foods primarily during preparation, at preservation, and/or at the time of consumption. Therefore, it is theoretically possible to alter sodium intake without altering intake of specific foods or overall dietary pattern. In addition, potassium was reviewed as a single nutrient because it has been hypothesized that dietary potassium intake may lower BP independent of other nutrients or foods. In addition, the effect of sodium on BP may be modulated by concomitant potassium intake.

Most of the clinical trial evidence pertains to effects of minerals on risk factors (ie, BP and plasma lipids) that are relevant, intermediate outcomes for CVD. In addition, data primarily from observational studies provide evidence on the

effects of dietary sodium and potassium on outcomes that are CVD events.

4.2. Selection of Inclusion/Exclusion Criteria

Work Group members developed eligibility criteria on the basis of a PICOTS approach for screening potential studies for inclusion in the evidence review. The PICOTS approach for CQ2 and other detailed methods are in the NHLBI Lifestyle Systematic Evidence Review report.

CQ2 was established to examine studies that assessed the impact of sodium and potassium on BP and cardiovascular morbidity and mortality. The studies included adults with or without established CVD; with or without CVD risk factors; with or without tobacco use; and who were of normal weight, overweight, or obese. In addition, intervention sample sizes were required to be at least 50 for biomarker and risk factor

Table 11. Resources and Information for Dietary Planning

DASH Eating Plan

- Your Guide to Lowering Your Blood Pressure With DASH (http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/new_dash.pdf)
- Your Guide to Lowering Your Blood Pressure With DASH Brochure (http://www.nhlbi.nih.gov/health/public/heart/hbp/dash/dash_brief.pdf)

AHA Diet and Lifestyle Recommendations

- AHA Diet and Lifestyle Recommendations Article (http://www.heart.org/HEARTORG/GettingHealthy/Diet-and-Lifestyle-Recommendations_UCM_305855_Article.jsp)
- AHA Diet and Lifestyle Recommendations 2006 Scientific Statement (<http://circ.ahajournals.org/content/114/1/82.full.pdf>)¹¹

Dietary Guidelines for Americans

- 2010 Dietary Guidelines for Americans (<http://www.cnpp.usda.gov/DGAs2010-PolicyDocument.htm>)⁴⁹
- 2011 Dietary Guidelines for Americans Brochure (<http://www.cnpp.usda.gov/Publications/MyPlate/DG2010Brochure.pdf>)
- USDA Food Patterns (<http://www.cnpp.usda.gov/Publications/USDAFoodPatterns/USDAFoodPatternsSummaryTable.pdf>)

AHA indicates American Heart Association; DASH, Dietary Approaches to Stop Hypertension; and USDA, US Department of Agriculture.

studies and 500 for cardiovascular morbidity and mortality. Because a separate Obesity Expert Panel reviewed evidence on the effect of weight loss on CVD risk factors and outcomes, the Work Group excluded studies in which weight change was >3%.

4.3. Literature Search Yield

In all, 34 studies (46 citations) satisfied the CQ2 inclusion criteria and were rated as good or fair quality.^{31,32,46,47,53–94}

The CQ2 summary tables are available in the [Full Work Group Report supplement](#). The tables present data on the studies used in the evidence review organized by mineral (sodium or potassium), outcomes (BP or CVD outcomes), sodium subquestions (overall results, different levels of sodium, sodium and other dietary changes), and subpopulations (sex, Summary Table C–4a; race/ethnicity, Summary Table C–4b; age, Summary Table C–4c; and hypertension status, Summary Table C–4d). Some studies appear in more than 1 summary table because they address more than 1 corresponding mineral or subquestion.

4.4. CQ2 Evidence Statements

See Table 13 for the CQ2 ESs for sodium and BP.

4.4.1. Sodium and BP

A note about the unit of measure presented for dietary and urinary sodium: sodium is presented in studies in millimoles (mmol), grams (g), and milligrams (mg). The Work Group chose to convert the sodium results to milligrams for the ESs, recommendations, and rationales so that data from different studies would be displayed in a consistent unit. Also, US dietary recommendations and the Nutrition Facts labels on food products display sodium in milligrams, and this unit (mg) will be easier for healthcare providers to communicate with patients. Urinary and dietary sodium are portrayed in the original units from each published study in the CQ2 summary tables (C–1 to C–8).

Table 12. CQ for Sodium and Potassium: BP and CVD Outcomes

CQ2:

Among adults, what is the effect of dietary intake of sodium and potassium on CVD risk factors and outcomes, when compared with no treatment or with other types of interventions?

BP indicates blood pressure; CQ, critical question; and CVD, cardiovascular disease.

4.5. Diet Recommendations for BP Lowering

1. Advise adults who would benefit from BP lowering to:
 - a. Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limits intake of sweets, sugar-sweetened beverages, and red meats.
 - i. Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes).
 - ii. Achieve this pattern by following plans such as the DASH dietary pattern, the USDA Food Pattern, or the AHA Diet.

NHLBI Grade: A (strong); ACC/AHA COR: I, LOE: A

Rationale: This recommendation is based largely on studies of the DASH dietary pattern (DASH and DASH-Sodium), which provided the highest-quality evidence that this food-based dietary pattern improves lipid profiles and BP (Tables 8 and 9, CQ1 ES3–ES9). This evidence was supplemented by studies of low quality in which various adaptations of the MED pattern were tested and also found to reduce BP (Table 7, CQ1 ES1). The evidence suggests that the effects of the recommended dietary pattern persist as long as the pattern is consumed. The BP-lowering effect has been demonstrated in adults with hypertension and prehypertension and is evident in men and women, African-American and non-African-American adults, and older and younger adults (Table 8, ES5). The dietary pattern's effect on BP is independent of changes in weight and sodium intake. The magnitude of effect is sufficient to prevent progression from prehypertension to hypertension, promote nonpharmacological BP control in those with hypertension, and supplement pharmacological BP lowering.

The caloric (energy) intake should be appropriate for the individual (eg, restricted for those attempting weight loss). Patients should also be encouraged to adapt the recommended dietary pattern to their personal and cultural preferences. Materials are available to assist patients in achieving the recommended dietary pattern at different calorie levels (Table 9). The 2010 US Department of Health and Human Services Dietary Guidelines for Americans recommend the USDA Food Pattern and the DASH dietary pattern.⁴⁹ Overall, the recommended dietary pattern is consistent with the AHA Diet⁵⁰ and the USDA Food Pattern.⁴⁹ The USDA Food Pattern offers lacto-ovo vegetarian and vegan adaptations. Therefore, this recommendation is consistent with other national guidelines. Clinicians should be familiar with the recommendations, advise their patients to adopt them, and provide easy access to information (Table 11).

Table 13. CQ2 ESs for Sodium and BP

Overall Results of Sodium and the Effect on BP:

What Is the Overall Effect of Dietary Intake of Sodium on BP?

ES1.

- In adults 25 to 80 years of age with BP 120–159/80–95 mmHg, reducing sodium intake lowers BP.

Strength of Evidence: High

Comparison of Different Levels of Sodium Intake:

What Is the Effect of Different Levels of Dietary Sodium Intake on BP?

ES2.

- In adults 25 to 75 years of age with BP 120–159/80–95 mmHg, a reduction in sodium intake that achieves a mean 24-h urinary sodium excretion of approximately 2400 mg/d, relative to approximately 3300 mg/d, lowers BP by 2/1 mmHg. A reduction in sodium intake that achieves a mean 24-h urinary sodium excretion of approximately 1500 mg/d lowers BP by 7/3 mmHg.

Strength of Evidence: Moderate

ES3.

- In adults 30–80 years of age with or without hypertension, counseling to reduce sodium intake by an average of 1150 mg/d reduces BP by 3–4/1–2 mmHg.

Strength of Evidence: High

Sodium in Subpopulations:

What Is the Effect of Sodium on BP in Subgroups Defined by Sex, Race/Ethnicity, Age, and Hypertension Status?

ES4.

- In adults with prehypertension or hypertension, reducing sodium intake lowers BP in women and men, African-American and non-African-American adults, and older and younger adults.

Strength of Evidence: High

ES5.

- Reducing sodium intake lowers BP in adults with either prehypertension or hypertension who are eating either the typical American diet or the DASH dietary pattern. The effect is greater in those with hypertension.

Strength of Evidence: High

Sodium and Dietary Pattern Changes:

What Is the Effect of Sodium on BP in the Context of Dietary Pattern Changes?

ES6.

- In adults 22–80 years of age with BP 120–159/80–95 mmHg, the combination of reduced sodium intake plus eating the DASH dietary pattern lowers BP more than reduced sodium intake alone.

Strength of Evidence: Moderate

Sodium in the Context of Other Minerals and BP:

What Is the Effect of Sodium on BP in the Context of Other Single Minerals?

ES7.

- There is insufficient evidence from RCTs to determine whether reducing sodium intake plus changing dietary intake of any other single mineral (eg, increasing potassium, calcium, or magnesium) lowers BP more than reducing sodium intake alone.

Strength of Evidence: Insufficient

Sodium and Congestive Heart Disease/CVD Outcomes:

What Is the Effect of Dietary Intake of Sodium on CVD Outcomes?

ES8.

- A reduction in sodium intake of approximately 1000 mg/d reduces CVD events by about 30%.

Strength of Evidence: Low

ES9.

- Higher dietary sodium intake is associated with a greater risk of fatal and nonfatal stroke and CVD.

Strength of Evidence: Low

ES10.

- There is insufficient evidence to determine the association between sodium intake and the development of HF.

Strength of Evidence: Insufficient

ES11.

- There is insufficient evidence to assess the effect of reducing dietary sodium intake on cardiovascular outcomes in patients with existing HF.

Strength of Evidence: Insufficient

Potassium and BP and Congestive Heart Disease/CVD Outcomes:

What Is the Effect of Dietary Intake of Potassium on BP and CVD Outcomes?

ES12.

- There is insufficient evidence to determine whether increasing dietary potassium intake lowers BP.

Strength of Evidence: Insufficient

ES13.

- In observational studies with appropriate adjustments (eg, BP, sodium intake), higher dietary potassium intake is associated with lower stroke risk.

Strength of Evidence: Low

ES14.

- There is insufficient evidence to determine whether there is an association between dietary potassium intake and congestive heart disease, HF, or cardiovascular mortality rate.

Strength of Evidence: Insufficient

BP indicates blood pressure; CVD, cardiovascular disease; DASH, Dietary Approaches to Stop Hypertension; ES, evidence statement; HF, heart failure; and RCT, randomized controlled trial.

Dietary planning and nutritional counseling are often facilitated by referral to a nutrition professional.

2. Advise adults who would benefit from BP lowering to:
a. Lower sodium intake

NHLBI Grade: A (strong); ACC/AHA COR: I, LOE: A

Rationale: There is strong and consistent clinical trial evidence that reducing sodium intake lowers BP. This BP-lowering effect has been demonstrated in adults with hypertension and prehypertension, in men and women, in African-American and non-African-American adults, and in older and younger adults. Trials contributing to this evidence include well-controlled feeding studies as well as studies in which participants were counseled to lower sodium intake. The effect of reducing sodium intake on BP is independent of changes in weight. The magnitude of effect is sufficient both to prevent progression from prehypertension to hypertension and to promote nonpharmacological BP control in those with hypertension. Observational data also suggest that lower sodium intake is associated with lower risk of cardiovascular events in people with and without hypertension, which is hypothesized to occur through reductions in BP.

3. Advise adults who would benefit from BP lowering to:

- a. Consume no more than 2400 mg of sodium/d;**
- b. Further reduction of sodium intake to 1500 mg/d can result in even greater reduction in BP;**
- c. Even without achieving these goals, reducing sodium intake by at least 1000 mg/d lowers BP.**

NHLBI Grade: B (moderate); ACC/AHA COR: IIa, LOE: B

Rationale: One well-conducted trial demonstrated clinically meaningful lowering of BP when sodium was reduced to 2400 mg/d, with lower BPs achieved when sodium intake was reduced to 1500 mg/d. Reductions of 1000 mg/d were shown to be beneficial in trials, and observational studies estimated significant reductions in relative risk associated with changes in sodium intake of about 1000 mg/d. This recommendation is directed at the two thirds of the US adults who have prehypertension or hypertension, for whom reducing sodium intake can prevent or improve control of hypertension and potentially reduce cardiovascular events.

The Work Group acknowledges that the recommendation to reduce sodium intake to ≤ 2400 mg/d differs slightly from other current dietary recommendations—specifically, the 2010 Dietary Guidelines for Americans and the Institute of Medicine Dietary Reference Intakes; both of these publications recommend 2300 mg/d as the upper limit of intake for adults. Although the impact on behavior of a difference between intakes of 2400 mg of sodium/d versus 2300 mg of sodium/d would be minimal, these recommendations are based on the strongest clinical trial evidence available: the achieved level of 2400 mg/d from the DASH-Sodium trial (estimated from average urinary sodium excretion) (Table 11, CQ2 ES2).

The strength of this recommendation is graded “moderate” because fewer clinical trials were used to devise the

2400-mg and 1500-mg goals than the large number of trials used to inform the overall recommendation on sodium (dietary recommendation No. 2 for BP lowering), which is graded “strong.”

Reducing sodium intake can be challenging for an individual because of the ubiquitous nature of sodium in the American food supply. Educational materials with strategies to help patients lower sodium intake are provided by several federal and private sources.^{49,95–98} Ultimately, however, significant changes in sodium intake among US adults may require changes both in individual behavior and in food manufacturing and processing.

4. Advise adults who would benefit from BP lowering to:
a. Combine the DASH dietary pattern with lower sodium intake.

NHLBI Grade: A (strong); ACC/AHA COR: I, LOE: A

Rationale: Both a healthy dietary pattern, as exemplified by DASH, and reduced sodium intake independently reduce BP. However, the BP-lowering effect is even greater when these dietary changes are combined. In the 60% of US adults with prehypertension or hypertension, simultaneously implementing dietary recommendations No. 1 and No. 2 for BP lowering can prevent and control hypertension more than either intervention alone.

5. CQ3—Physical Activity: Lipids and BP

See Table 14 for the CQ for physical activity and lipids and BP.

5.1. Introduction/Rationale

Large bodies of observational data show an association between higher levels of physical activity and lower rates of many chronic diseases, including CVD, and enhanced longevity.^{99–101} Furthermore, an inverse dose-response relation exists, with increasing higher levels of activity associated with commensurately lower rates of CVD in a curvilinear fashion.^{102,103} In a recent analysis, it was estimated that by eliminating physical inactivity, 6% of coronary heart disease worldwide may be eliminated, and life expectancy of the world population may be increased by 0.68 years.^{104,105}

Among the mechanisms proposed to mediate the relationship between physical activity and decreased CVD rates are beneficial effects of exercise on lipid profile and BP.¹⁰⁶ In one study, it was estimated that the beneficial effects of physical activity on BP and development of hypertension explained some 27% of the activity-related reduction in observed CVD rates, while 19% and 16% of the reduction in CVD rates could be explained by the beneficial effects of physical activity on traditional lipids and novel lipids, respectively.

In the remainder of Section 5, the Work Group elaborates on findings from meta-analyses of the effects of physical activity on changes in lipid profile and BP.

5.2. Selection of Inclusion/Exclusion Criteria

Because of resource limitations, the Work Group included only systematic reviews and meta-analyses of RCTs or

Table 14. CQ for Physical Activity: Lipids and BP

CQ3:
Among adults, what is the effect of physical activity on BP and lipids when compared with no treatment or with other types of interventions?

BP indicates blood pressure; and CQ, critical question.

controlled clinical trials published from 2001 through 2011. Detailed inclusion/exclusion criteria are available in the [Full Work Group Report supplement](#).

5.3. Literature Search Yield

A total of 26 systematic reviews and meta-analyses were identified that met inclusion/exclusion criteria and were rated as good or fair quality.^{105,107–130}

The CQ3 subcommittee members next identified the included systematic reviews and meta-analyses that contained detailed data on BP outcomes. They identified 11 studies with data on BP outcomes. Ten meta-analyses and 1 systematic review examined the effects of aerobic exercise. One systematic review looked at the effects of resistance training. The CQ3 subcommittee members next identified the included systematic reviews and meta-analyses that contained detailed data on lipid outcomes. They identified 14 studies with data on lipid outcomes, including 10 meta-analyses and 4 systematic reviews.

The next step in the evidence review process for systematic reviews and meta-analyses was to develop ESs and recommendations from the included studies and present them to the full Work Group for consideration and voting. Because each of these systematic reviews and meta-analyses summarizes evidence from several studies, NHLBI staff and Work Group members determined that the development of formal evidence tables and summary tables of individual articles was unnecessary. CQ3 subcommittee members developed evidence tables that are available in the [Full Work Group Report supplement](#) (CQ3 Summary Tables: Summary Table D–1: Aerobic Exercise and LDL-C, Summary Table D–2: Resistance Exercise and LDL-C, Summary Table D–3: Aerobic Exercise and HDL-C, and Summary Table D–4: Resistance Exercise and HDL-C) to summarize the evidence on physical activity and lipids.

5.4. CQ3 Evidence Statements

5.4.1. Physical Activity and Lipids

See Table 15 for the CQ3 ESs for physical activity and lipids.

This section examines evidence supporting the use of physical activity alone (ie, not in combination with other interventions, such as dietary interventions or weight loss) versus no physical activity or other type of intervention for improvements in selected blood lipids (HDL-C, LDL-C, triglycerides, and non-HDL-C). The *2008 Physical Activity Guidelines Advisory Committee Report* was used as the starting point for evidence review.⁹⁹ Additionally, a systematic search identified 8 meta-analyses from 2001 onward and 5 systematic reviews rated fair to good that addressed this question and were included as the evidence base.

5.4.2. Physical Activity and BP

This section examines evidence supporting the use of physical activity alone (ie, not in combination with other interventions,

Table 15. ESs for Physical Activity and Lipids

Aerobic Exercise Training and Lipids

ES1.

- Among adults, aerobic physical activity, compared with control interventions, reduces LDL-C 3–6 mg/dL on average.

Strength of Evidence: Moderate

ES2.

- Among adults, aerobic physical activity alone, compared with control interventions, reduces non-HDL-C 6 mg/dL on average.

Strength of Evidence: Moderate

ES3.

- Among adults, aerobic physical activity alone, compared with control interventions, has no consistent effect on triglycerides.

Strength of Evidence: Moderate

ES4.

- Among adults, aerobic physical activity alone, compared with control interventions, has no consistent effect on HDL-C.

Strength of Evidence: Moderate

Resistance Exercise Training and Lipids

ES5.

- Among adults, resistance training, compared with control interventions, reduces LDL-C, triglycerides, and non-HDL-C by 6–9 mg/dL on average and has no effect on HDL-C. Typical interventions shown to reduce LDL-C, triglycerides, and non-HDL-C and to have no effect on HDL-C include resistance physical activity programs that average 24 wk duration and include ≥ 3 d/wk, with 9 exercises performed for 3 sets and 11 repetitions at an average intensity of 70% of 1 maximal repetition.

Strength of Evidence: Low

ES indicates evidence statement; HDL-C, high-density lipoprotein cholesterol; and LDL-C, low-density lipoprotein cholesterol.

such as dietary interventions or weight loss) versus no physical activity or other types of intervention for BP reduction. The *2008 Physical Activity Guidelines Advisory Committee Report* was used as the starting point for evidence review.⁹⁹ Additionally, a systematic search identified 15 meta-analyses from 2001 onward and reviews rated fair to good that addressed this question. Details of the search are provided in the [Full Work Group Report supplement](#).

5.4.2.1. Aerobic Exercise Training and BP

See Table 16 for the ES for aerobic exercise training and BP.

5.4.2.2. Resistance Exercise Training and BP

The *2008 Physical Activity Guidelines Advisory Committee Report* focused on data from a meta-analysis of 9 RCTs of resistance training that included 341 subjects.¹³¹ However, given the limited parameters of the systematic search described previously for CQ3 (Section 5.3), only 1 review was identified. A

Table 16. ES for Aerobic Exercise Training and BP

ES1.

- Among adult men and women at all BP levels, including individuals with hypertension, aerobic physical activity decreases systolic and diastolic BP, on average by 2–5 mm Hg and 1–4 mm Hg, respectively. Typical interventions shown to be effective for lowering BP include aerobic physical activity of, on average, at least 12 wk duration, with 3–4 sessions per wk, lasting on average 40 min/session and involving moderate- to vigorous-intensity physical activity.

Strength of Evidence: High

BP indicates blood pressure; and ES, evidence statement.

qualitative review of clinical trials—randomized, nonrandomized, and uncontrolled studies—examined resistance exercise training in relation to metabolic health among patients with type 2 diabetes.¹²⁷ Ten of these studies assessed BP. Investigators concluded that resistance exercise training resulted in beneficial changes in systolic BP, with benefits in diastolic BP observed less frequently. (The magnitude of reduction was not specified.) Thus, the review of evidence did not provide consistent evidence on resistance exercise training for BP reduction.

5.4.2.3. Combination of Aerobic and Resistance Exercise Training and BP

There have been no published meta-analyses or reviews specifically examining the effect of a combined regimen of aerobic exercise and resistance training on BP. However, in some of the meta-analyses and reviews described above, studies with aerobic and resistance components were included in pooled data related to aerobic exercise training.^{113,114}

5.5. Physical Activity Recommendations

- 1. In general, advise adults to engage in aerobic physical activity to reduce LDL-C and non-HDL-C: 3 to 4 sessions per week, lasting on average 40 minutes per session, and involving moderate- to vigorous-intensity physical activity.**

NHLBI Grade: B (moderate); ACC/AHA COR: IIa, LOE: A

Rationale: This recommendation was based on evidence from meta-analyses and reviews published from 2001 onward and rated fair to good. This is also consistent with the findings of the literature review conducted for the *2008 Physical Activity Guidelines Advisory Committee Report*, in which it was found that it may require 12 metabolic equivalent task-hours per week of exercise to favorably influence LDL-C. The amount of physical activity recommended above for reducing LDL-C and non-HDL-C is congruent with the amount of physical activity recommended in 2008 by the federal government for overall health: “Most health benefits occur with at least 150 minutes (2 hours and 30 minutes) a week of moderate-intensity physical activity, such as brisk walking. Additional benefits occur with more physical activity.”⁹⁹

- 2. In general, advise adults to engage in aerobic physical activity to lower BP: 3 to 4 sessions per week, lasting on average 40 minutes per session, and involving moderate- to vigorous-intensity physical activity.**

NHLBI Grade: B (moderate); ACC/AHA COR: IIa, LOE: A

Rationale: This recommendation was based on evidence from meta-analyses and reviews rated fair to good that were published from 2001 onward, as well as the *2008 Physical Activity Guidelines Advisory Committee Report*. The amount of physical activity recommended above for lowering BP is congruent with the amount of physical activity recommended in 2008 by the federal government for overall health: “Most health benefits occur with at least 150 minutes (2 hours and 30 minutes) a week of moderate-intensity physical activity,

such as brisk walking. Additional benefits occur with more physical activity.”⁹⁹ It is worth noting that the present recommendation is congruent with (ie, expends approximately the same amount of energy) but not identical to the 2008 federal guidelines. This is because the present recommendation is based on a review of meta-analyses of exercise in relation to BP only (hence, the specific regimens as used in the clinical trials), whereas the 2008 federal guidelines targeted overall health (ie, not just BP). Additionally, the 2008 federal guidelines for overall health make it clear that any amount of physical activity is healthful (“Some physical activity is better than none”), and that there is a dose-response relationship (“For most health outcomes, additional benefits occur as the amount of physical activity increases through higher intensity, greater frequency, and/or longer duration”).

5.6. Heart-Healthy Nutrition and Physical Activity Behaviors

See Table 17 for information on heart-healthy nutrition and physical activity behaviors.

Overall, the Work Group encourages heart-healthy nutrition and physical activity behaviors for the entire US adult population as stated in the *2010 Dietary Guidelines for Americans* and the *2008 Physical Activity Guidelines for Americans*. The recommendations in Table 17 are a consensus of the Work Group, not a guideline, and are generally consistent with the *2010 Dietary Guidelines for Americans* and the *2008 Physical Activity Guidelines for Americans*.

6. Gaps in Evidence and Future Research Needs

6.1. Diet

The extensive work of the Work Group served an additional purpose, and that was to identify important gaps in the knowledge of how lifestyle impacts CVD risk reduction.

Table 17. Heart-Healthy Nutrition and Physical Activity Behaviors

Heart-Healthy Nutrition and Physical Activity Behaviors

The adult population should be encouraged to practice heart-healthy lifestyle behaviors, including:

- Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limits intake of sodium, sweets, sugar-sweetened beverages, and red meats.
 - Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes).
 - Achieve this pattern by following plans such as the DASH dietary pattern, the USDA Food Pattern, or the AHA Diet.
- Engage in 2 h and 30 min per wk of moderate-intensity physical activity, or 1 h and 15 min (75 min) per wk of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity aerobic physical activity. Aerobic activity should be performed in episodes of at least 10 min, preferably spread throughout the wk.¹³²
- Achieve and maintain a healthy weight. Refer to the 2013 Obesity Expert Panel Report for recommendations on weight loss and maintenance.⁵

AHA indicates American Heart Association; DASH, Dietary Approaches to Stop Hypertension; and USDA, US Department of Agriculture.

Additional research is needed on the following topics related to diet:

- Interaction between dietary modification and statin treatment.
- Relative effects of saturated fats, monounsaturated fatty acids, polyunsaturated fatty acids, *trans* fatty acids, omega-3 fatty acids, and types of carbohydrates on lipids, inflammation, microbiome, and other newer potential CVD risk factors.
- Relative effects of naturally occurring fiber (cereal [whole grains] and vegetable/fruit) and supplemental fiber on lipids, inflammation, microbiome, and other newer potential CVD risk factors.
- Effects of dietary cholesterol on LDL-C and HDL-C over the current ranges of cholesterol and saturated fat intakes (5th and 95th percentiles).
- Effects of minerals in combination (other than sodium) on BP.
- High-density lipoprotein function in studies that modify HDL-C by changes in diet.
- Is the minimal effect of dietary carbohydrate on plasma triglycerides harmful?
- The effect of sodium reduction in patients with diabetes, heart failure, and chronic kidney disease.
- Effect of dietary pattern and sodium intake in adults taking BP-lowering or lipid-lowering medications (eg, effects on BP/lipids, achieving BP/lipid goals, medication needs/costs, outcomes).
- Effect of dietary pattern and sodium intake in adults with CVD (eg, after myocardial infarction; after stroke; with coronary artery disease, heart failure, or chronic kidney disease).
- Strategies for effectively (and cost-effectively) implementing these evidence-based recommendations. (How can primary care providers, health systems, public health agencies, local and federal government, community organizations, and other stakeholders help patients adopt these diet and sodium intake recommendations?)
- Increased understanding of racial/ethnic/socioeconomic factors that may influence (a) effect of dietary pattern and sodium on BP and lipids, (b) adoption of diet/sodium recommendations, and (c) method of diet assessment.

6.2. Physical Activity

Additional research is needed on the following topics related to physical activity:

- Improved understanding of whether exercise performed at a lower intensity or dose, or different modes of exercise, can impact these outcomes.
- Further understanding the characteristics of individuals for whom exercise of a certain dose or intensity can reduce LDL-C and non-HDL-C.
- Understand the source of the inconsistent findings to better understand under what conditions exercise can increase HDL-C or decrease triglycerides.
- Define the optimal dose of exercise that will result in the desired changes in lipids and lipoproteins and whether

exercise performed at a lower intensity or dose, or different modes of exercise, can impact these outcomes.

- Further understand the characteristics of individuals for whom exercise of a certain dose, intensity, or mode can increase HDL-C or reduce triglycerides.
- Clarify the shape of the dose-response curve between physical activity and BP.
- Expand the limited data on whether resistance exercise training lowers BP and whether a combination of aerobic and resistance exercise training offers any added BP lowering, compared with aerobic exercise only.
- Determine how diet and physical activity behave synergistically with regard to lipids and BP.
- Determine the effect of physical activity in adults taking BP-lowering and/or lipid-lowering medications (eg, effects on BP/lipids, achieving BP/lipid goals, medication needs/costs, outcomes).
- Determine the effect of physical activity in adults with CVD (eg, after myocardial infarction; after stroke; with coronary artery disease, heart failure, or chronic kidney disease).
- Determine strategies for effectively (and cost-effectively) implementing these evidence-based recommendations. (How can primary care providers, health systems, public health agencies, local and federal government, community organizations, and other stakeholders help patients adopt these physical activity recommendations?)
- Increase understanding of racial/ethnic/socioeconomic factors that may influence (a) effect of physical activity on BP and lipids and (b) adoption of physical activity recommendations.

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References

- Institute of Medicine (US) Committee on Developing Trustworthy Guidelines. *Clinical Practice Guidelines We Can Trust*. Washington, DC: The National Academies Press; 2011.
- Gibbons GH, Harold JG, Jessup M, et al. The next steps in developing clinical practice guidelines for prevention. *J Am Coll Cardiol*. 2013;62:1399–400.
- Gibbons GH, Shurin SB, Mensah GA, et al. Refocusing the agenda on cardiovascular guidelines: an announcement from the National Heart, Lung, and Blood Institute. *J Am Coll Cardiol*. 2013;62:1396–8.
- Stone NJ, Robinson JG, Lichtenstein AH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(suppl 2):S1–S45.
- Jensen MD, Ryan DH, C.M. A, et al. 2013 ACC/AHA/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation*. 2014;129(suppl 2):S102–S138.
- Centers for Disease Control and Prevention (CDC). National Center for Health Statistics (NCHS). National Health and Nutrition Examination Survey Data. Hyattsville, MD: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2005–2008.
- Micha R, Kalantarian S, Wirojatana P, et al. Estimating the global and regional burden of suboptimal nutrition on chronic disease: methods and inputs to the analysis. *Eur J Clin Nutr*. 2012;66:119–29.
- Mehio Sibai A, Nasreddine L, Mokdad AH, et al. Nutrition transition and cardiovascular disease risk factors in Middle East and North Africa countries: reviewing the evidence. *Ann Nutr Metab*. 2010;57:193–203.
- Srinath Reddy K, Katan MB. Diet, nutrition and the prevention of hypertension and cardiovascular diseases. *Public Health Nutr*. 2004;7:167–86.
- Diet, nutrition, and the prevention of chronic diseases. Report of a WHO Study Group. *World Health Organ Tech Rep Ser*. 1990;797:1–204.
- American Heart Association Nutrition Committee; Lichtenstein AH, Appel LJ, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006;114:82–96.
- Eilat-Adar S, Mete M, Fretts A, et al. Dietary patterns and their association with cardiovascular risk factors in a population undergoing lifestyle changes: The Strong Heart Study. *Nutr Metab Cardiovasc Dis*. 2013;23:528–35.
- Flock MR, Kris-Etherton PM. Dietary Guidelines for Americans 2010: implications for cardiovascular disease. *Curr Atheroscler Rep*. 2011;13:499–507.
- Mozaffarian D, Appel LJ, Van Horn L. Components of a cardioprotective diet: new insights. *Circulation*. 2011;123:2870–91.
- Hercberg S, Castetbon K, Czernichow S, et al. The Nutrinet-Santé Study: a web-based prospective study on the relationship between nutrition and health and determinants of dietary patterns and nutritional status. *BMC Public Health*. 2010;10:242.
- Kant AK. Dietary patterns: biomarkers and chronic disease risk. *Appl Physiol Nutr Metab*. 2010;35:199–206.
- Iqbal R, Anand S, Ounpuu S, et al. Dietary patterns and the risk of acute myocardial infarction in 52 countries: results of the INTERHEART study. *Circulation*. 2008;118:1929–37.
- Nettleton JA, Schulze MB, Jiang R, et al. A priori-defined dietary patterns and markers of cardiovascular disease risk in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr*. 2008;88:185–94.
- Mikkilä V, Räsänen L, Raitakari OT, et al. Major dietary patterns and cardiovascular risk factors from childhood to adulthood. The Cardiovascular Risk in Young Finns Study. *Br J Nutr*. 2007;98:218–25.
- Panagiotakos DB, Pitsavos C, Stefanadis C. Dietary patterns: a Mediterranean diet score and its relation to clinical and biological markers of cardiovascular disease risk. *Nutr Metab Cardiovasc Dis*. 2006;16:559–68.
- Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. *Arterioscler Thromb Vasc Biol*. 1992;12:911–9.
- Mensink RP, Zock PL, Kester ADM, et al. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr*. 2003;77:1146–55.
- Mozaffarian D, Clarke R. Quantitative effects on cardiovascular risk factors and coronary heart disease risk of replacing partially hydrogenated vegetable oils with other fats and oils. *Eur J Clin Nutr*. 2009;63(Suppl 2):S22–33.
- Tang JL, Armitage JM, Lancaster T, et al. Systematic review of dietary intervention trials to lower blood total cholesterol in free-living subjects. *BMJ*. 1998;316:1213–20.
- Jula A, Marniemi J, Huuopponen R, et al. Effects of diet and simvastatin on serum lipids, insulin, and antioxidants in hypercholesterolemic men: a randomized controlled trial. *JAMA*. 2002;287:598–605.
- Michalsen A, Lehmann N, Pithan C, et al. Mediterranean diet has no effect on markers of inflammation and metabolic risk factors in patients with coronary artery disease. *Eur J Clin Nutr*. 2006;60:478–85.
- Wolever TMS, Gibbs AL, Mehling C, et al. The Canadian Trial of Carbohydrates in Diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. *Am J Clin Nutr*. 2008;87:114–25.
- Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med*. 1997;336:1117–24.
- Sacks FM, Appel LJ, Moore TJ, et al. A dietary approach to prevent hypertension: a review of the Dietary Approaches to Stop Hypertension (DASH) Study. *Clin Cardiol*. 1999;22:III6–10.
- Obarzanek E, Sacks FM, Vollmer WM, et al. Effects on blood lipids of a blood pressure-lowering diet: the Dietary Approaches to Stop Hypertension (DASH) Trial. *Am J Clin Nutr*. 2001;74:80–9.
- Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med*. 2001;344:3–10.
- Harsha DW, Sacks FM, Obarzanek E, et al. Effect of dietary sodium intake on blood lipids: results from the DASH-sodium trial. *Hypertension*. 2004;43:393–8.
- Erlinger TP, Miller ER 3rd, Charleston J, et al. Inflammation modifies the effects of a reduced-fat low-cholesterol diet on lipids: results from the DASH-sodium trial. *Circulation*. 2003;108:150–4.
- Ginsberg HN, Kris-Etherton P, Dennis B, et al. Effects of reducing dietary saturated fatty acids on plasma lipids and lipoproteins in healthy subjects: the DELTA Study, protocol 1. *Arterioscler Thromb Vasc Biol*. 1998;18:441–9.
- Gardner CD, Coulston A, Chatterjee L, et al. The effect of a plant-based diet on plasma lipids in hypercholesterolemic adults: a randomized trial. *Ann Intern Med*. 2005;142:725–33.
- Jenkins DJA, Kendall CWC, McKeown-Eyssen G, et al. Effect of a low-glycemic index or a high-cereal fiber diet on type 2 diabetes: a randomized trial. *JAMA*. 2008;300:2742–53.
- Estruch R, Martínez-González MA, Corella D, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. *Ann Intern Med*. 2006;145:1–11.
- Appel LJ, Sacks FM, Carey VJ, et al. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA*. 2005;294:2455–64.
- Núñez-Córdoba JM, Valencia-Serrano F, Toledo E, et al. The Mediterranean diet and incidence of hypertension: the Seguimiento Universidad de Navarra (SUN) Study. *Am J Epidemiol*. 2009;169:339–46.
- Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*. 2006;295:655–66.
- Tinker LF, Bonds DE, Margolis KL, et al. Low-fat dietary pattern and risk of treated diabetes mellitus in postmenopausal women: the Women's Health Initiative randomized controlled dietary modification trial. *Arch Intern Med*. 2008;168:1500–11.
- Yusof BNM, Talib RA, Kamaruddin NA, et al. A low-GI diet is associated with a short-term improvement of glycaemic control in Asian patients with type 2 diabetes. *Diabetes Obes Metab*. 2009;11:387–96.
- Svetkey LP, Simons-Morton D, Vollmer WM, et al. Effects of dietary patterns on blood pressure: subgroup analysis of the Dietary Approaches to Stop Hypertension (DASH) randomized clinical trial. *Arch Intern Med*. 1999;159:285–93.
- Moore TJ, Vollmer WM, Appel LJ, et al. Effect of dietary patterns on ambulatory blood pressure: results from the Dietary Approaches to Stop Hypertension (DASH) Trial. DASH Collaborative Research Group. *Hypertension*. 1999;34:472–7.
- Conlin PR, Chow D, Miller ER 3rd, et al. The effect of dietary patterns on blood pressure control in hypertensive patients: results from the Dietary Approaches to Stop Hypertension (DASH) trial. *Am J Hypertens*. 2000;13:949–55.
- Bray GA, Vollmer WM, Sacks FM, et al. A further subgroup analysis of the effects of the DASH diet and three dietary sodium levels on blood pressure: results of the DASH-Sodium Trial. *Am J Cardiol*. 2004;94:222–7.

47. Vollmer WM, Sacks FM, Ard J, et al. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-sodium trial. *Ann Intern Med.* 2001;135:1019–28.
48. Tonstad S, Sundfjor T, Seljeflot I. Effect of lifestyle changes on atherogenic lipids and endothelial cell adhesion molecules in young adults with familial premature coronary heart disease. *Am J Cardiol.* 2005;95:1187–91.
49. US Department of Agriculture and US Department of Health and Human Services. *Dietary Guidelines for Americans*, 2010. 7th Edition. Washington, DC: US Government Printing Office; December 2010.
50. Lichtenstein AH, Appel LJ, Brands M, et al. Summary of American Heart Association Diet and Lifestyle Recommendations revision 2006. *Arterioscler Thromb Vasc Biol.* 2006;26:2186–91.
51. Wright JD, Wang C-Y. Trends in intake of energy and macronutrients in adults from 1999–2000 through 2007–2008. *NCHS Data Brief.* 2010:1–8.
52. Doell D, Folmer D, Lee H, et al. Updated estimate of trans fat intake by the US population. *Food Addit Contam Part A Chem Anal Control Expo Risk Assess.* 2012;29:861–74.
53. Whelton PK, Appel LJ, Espeland MA, et al. Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). TONE Collaborative Research Group. *JAMA.* 1998;279:839–46.
54. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The Trials of Hypertension Prevention, phase II. The Trials of Hypertension Prevention Collaborative Research Group. *Arch Intern Med.* 1997;157:657–67.
55. Appel LJ, Espeland MA, Easter L, et al. Effects of reduced sodium intake on hypertension control in older individuals: results from the Trial of Nonpharmacologic Interventions in the Elderly (TONE). *Arch Intern Med.* 2001;161:685–93.
56. Espeland MA, Kumanyika S, Yunis C, et al. Electrolyte intake and non-pharmacologic blood pressure control. *Ann Epidemiol.* 2002;12:587–95.
57. Svetkey LP, Simons-Morton DG, Proschan MA, et al. Effect of the dietary approaches to stop hypertension diet and reduced sodium intake on blood pressure control. *Hypertens (Greenwich).* 2004;6:373–81.
58. Kumanyika SK, Cook NR, Cutler JA, et al. Sodium reduction for hypertension prevention in overweight adults: further results from the Trials of Hypertension Prevention Phase II. *J Hum Hypertens.* 2005;19:33–45.
59. Cook NR, Kumanyika SK, Cutler JA, et al. Dose-response of sodium excretion and blood pressure change among overweight, nonhypertensive adults in a 3-year dietary intervention study. *J Hum Hypertens.* 2005;19:47–54.
60. Hu J, Jiang X, Li N, et al. Effects of salt substitute on pulse wave analysis among individuals at high cardiovascular risk in rural China: a randomized controlled trial. *Hypertens Res.* 2009;32:282–8.
61. China Salt Substitute Study Collaborative Group. Salt substitution: a low-cost strategy for blood pressure control among rural Chinese. A randomized, controlled trial. *J Hypertens.* 2007;25:2011–8.
62. Charlton KE, Steyn K, Levitt NS, et al. A food-based dietary strategy lowers blood pressure in a low socio-economic setting: a randomised study in South Africa. *Public Health Nutr.* 2008;11:1397–406.
63. Obarzanek E, Proschan MA, Vollmer WM, et al. Individual blood pressure responses to changes in salt intake: results from the DASH-Sodium trial. *Hypertension.* 2003;42:459–67.
64. Ard JD, Coffman CJ, Lin P-H, et al. One-year follow-up study of blood pressure and dietary patterns in dietary approaches to stop hypertension (DASH)-sodium participants. *Am J Hypertens.* 2004;17:1156–62.
65. Cappuccio FP, Kerry SM, Micah FB, et al. A community programme to reduce salt intake and blood pressure in Ghana [ISRCTN88789643]. *BMC Public Health.* 2006;6:13.
66. Cook NR, Cutler JA, Obarzanek E, et al. Long term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of the trials of hypertension prevention (TOHP). *BMJ.* 2007;334:885–8.
67. Chang H-Y, Hu Y-W, Yue C-S, et al. Effect of potassium-enriched salt on cardiovascular mortality and medical expenses of elderly men. *Am J Clin Nutr.* 2006;83:1289–96.
68. Cook NR, Obarzanek E, Cutler JA, et al. Joint effects of sodium and potassium intake on subsequent cardiovascular disease: the Trials of Hypertension Prevention follow-up study. *Arch Intern Med.* 2009;169:32–40.
69. Nagata C, Takatsuka N, Shimizu N, et al. Sodium intake and risk of death from stroke in Japanese men and women. *Stroke.* 2004;35:1543–47.
70. Tuomilehto J, Jousilahti P, Rastenyte D, et al. Urinary sodium excretion and cardiovascular mortality in Finland: a prospective study. *Lancet.* 2001;357:848–51.
71. Umesawa M, Iso H, Date C, et al. Relations between dietary sodium and potassium intakes and mortality from cardiovascular disease: the Japan Collaborative Cohort Study for Evaluation of Cancer Risks. *Am J Clin Nutr.* 2008;88:195–202.
72. Alderman MH, Cohen H, Madhavan S. Dietary sodium intake and mortality: the National Health and Nutrition Examination Survey (NHANES I). *Lancet.* 1998;351:781–5.
73. Cohen HW, Hailpern SM, Fang J, et al. Sodium intake and mortality in the NHANES II follow-up study. *Am J Med.* 2006;119:e7–14.
74. Cohen HW, Hailpern SM, Alderman MH. Sodium intake and mortality follow-up in the Third National Health and Nutrition Examination Survey (NHANES III). *J Gen Intern Med.* 2008;23:1297–302.
75. He J, Ogden LG, Vupputuri S, et al. Dietary sodium intake and subsequent risk of cardiovascular disease in overweight adults. *JAMA.* 1999;282:2027–34.
76. He J, Ogden LG, Bazzano LA, et al. Dietary sodium intake and incidence of congestive heart failure in overweight US men and women: first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. *Arch Intern Med.* 2002;162:1619–24.
77. Yang Q, Liu T, Kuklina EV, et al. Sodium and potassium intake and mortality among US adults: prospective data from the Third National Health and Nutrition Examination Survey. *Arch Intern Med.* 2011;171:1183–91.
78. O'Donnell MJ, Yusuf S, Mente A, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. *JAMA.* 2011;306:2229–38.
79. Marniemi J, Alanen E, Impivaara O, et al. Dietary and serum vitamins and minerals as predictors of myocardial infarction and stroke in elderly subjects. *Nutr Metab Cardiovasc Dis.* 2005;15:188–97.
80. Takachi R, Inoue M, Shimazu T, et al. Consumption of sodium and salted foods in relation to cancer and cardiovascular disease: the Japan Public Health Center-based Prospective Study. *Am J Clin Nutr.* 2010;91:456–64.
81. Liang W, Lee AH, Binns CW. Dietary intake of minerals and the risk of ischemic stroke in Guangdong Province, China, 2007–2008. *Prev Chronic Dis.* 2011;8:A38.
82. Gardener H, Rundek T, Wright CB, et al. Dietary sodium and risk of stroke in the Northern Manhattan study. *Stroke.* 2012;43:1200–5.
83. Ekinci EI, Clarke S, Thomas MC, et al. Dietary salt intake and mortality in patients with type 2 diabetes. *Diabetes Care.* 2011;34:703–9.
84. Stolarz-Skrzypek K, Kuznetsova T, Thijs L, et al. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. *JAMA.* 2011;305:1777–85.
85. Strazzullo P, D'Elia L, Kandala N-B, et al. Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies. *BMJ.* 2009;339:b4567.
86. Walker J, MacKenzie AD, Dunning J. Does reducing your salt intake make you live longer?. *Interact Cardiovasc Thorac Surg.* 2007;6:793–8.
87. Fang J, Madhavan S, Alderman MH. Dietary potassium intake and stroke mortality. *Stroke.* 2000;31:1532–7.
88. Bazzano LA, He J, Ogden LG, et al. Dietary potassium intake and risk of stroke in US men and women: National Health and Nutrition Examination Survey I epidemiologic follow-up study. *Stroke.* 2001;32:1473–80.
89. Al-Delaimy WK, Rimm EB, Willett WC, et al. Magnesium intake and risk of coronary heart disease among men. *J Am Coll Nutr.* 2004;23:63–70.
90. Ascherio A, Rimm EB, Hernán MA, et al. Intake of potassium, magnesium, calcium, and fiber and risk of stroke among US men. *Circulation.* 1998;98:1198–204.
91. Green DM, Ropper AH, Kronmal RA, et al. Serum potassium level and dietary potassium intake as risk factors for stroke. *Neurology.* 2002;59:314–20.
92. Weng L-C, Yeh W-T, Bai C-H, et al. Is ischemic stroke risk related to folate status or other nutrients correlated with folate intake?. *Stroke.* 2008;39:3152–8.
93. Geleijnse JM, Witteman JCM, Stijnen T, et al. Sodium and potassium intake and risk of cardiovascular events and all-cause mortality: the Rotterdam Study. *Eur J Epidemiol.* 2007;22:763–70.
94. Iso H, Stampfer MJ, Manson JE, et al. Prospective study of calcium, potassium, and magnesium intake and risk of stroke in women. *Stroke.* 1999;30:1772–9.
95. National Heart, Lung, and Blood Institute. Your guide to lowering your blood pressure with DASH. Bethesda, MD: National Heart, Lung, and Blood Institute. Available at: <http://catalog.nhlbi.nih.gov/catalog/product/Your-Guide-to-Lowering-Your-Blood-Pressure-with-DASH/06-4082?sortBy=4>; 2013. Accessed January 28, 2014.
96. Centers for Disease Control and Prevention (CDC). Most Americans should consume less sodium. Atlanta, GA: Centers for Disease Control and Prevention. Available at: <http://www.cdc.gov/salt/>; 2013. Accessed January 28, 2014.
97. US Food and Drug Administration. Sodium reduction. Silver Spring, MD; 2013. Available at: <http://www.fda.gov/Food/IngredientsPackagingLabeling/FoodAdditivesIngredients/ucm253316.htm>. Accessed January 28, 2014.

98. American Heart Association (AHA). Sodium (salt or sodium chloride). AHA. Dallas, TX: AHA. Available at: http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyDietGoals/Sodium-Salt-or-Sodium-Chloride_UCM_303290_Article.jsp#; 2013. Accessed January 28, 2014.
99. Physical Activity Guidelines Advisory Committee. Physical Activity Guidelines Advisory Committee Report, 2008. Washington, DC: US Department of Health and Human Services; 2008:1–683
100. Warburton DE, Charlesworth S, Ivey A, et al. A systematic review of the evidence for Canada's Physical Activity Guidelines for Adults. *Int J Behav Nutr Phys Act*. 2010;7:39.
101. World Health Organization. Global recommendations on physical activity for health. Geneva, Switzerland: World Health Organization; 2010:1–60
102. Shiroma EJ, Lee IM. Physical activity and cardiovascular health: lessons learned from epidemiological studies across age, gender, and race/ethnicity. *Circulation*. 2010;122:743–52.
103. Sattelmair J, Pertman J, Ding EL, et al. Dose response between physical activity and risk of coronary heart disease: a meta-analysis. *Circulation*. 2011;124:789–95.
104. Lee I-M, Shiroma EJ, Lobelo F, et al. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. *Lancet*. 2012;380:219–29.
105. Guo X, Zhou B, Nishimura T, et al. Clinical effect of qigong practice on essential hypertension: a meta-analysis of randomized controlled trials. *J Altern Complement Med*. 2008;14:27–37.
106. Mora S, Cook N, Buring JE, et al. Physical activity and reduced risk of cardiovascular events: potential mediating mechanisms. *Circulation*. 2007;116:2110–8.
107. Kelley GA, Kelley KS, Tran ZV. Walking and resting blood pressure in adults: a meta-analysis. *Prev Med*. 2001;33:120–7.
108. Hamer M, Chida Y. Active commuting and cardiovascular risk: a meta-analytic review. *Prev Med*. 2008;46:9–13.
109. Whelton SP, Chin A, Xin X, et al. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med*. 2002;136:493–503.
110. Lee MS, Pittler MH, Guo R, et al. Qigong for hypertension: a systematic review of randomized clinical trials. *J Hypertens*. 2007;25:1525–32.
111. Kelley GA, Sharpe Kelley K. Aerobic exercise and resting blood pressure in older adults: a meta-analytic review of randomized controlled trials. *J Gerontol A Biol Sci Med Sci*. 2001;56:M298–M303.
112. Jolly K, Taylor RS, Lip GYH, et al. Home-based cardiac rehabilitation compared with centre-based rehabilitation and usual care: a systematic review and meta-analysis. *Int J Cardiol*. 2006;111:343–51.
113. Thomas DE, Elliott EJ, Naughton GA. Exercise for type 2 diabetes mellitus. *Cochrane Database Syst Rev*. 2006;CD002968.
114. Asikainen T-M, Kukkonen-Harjula K, Miilunpalo S. Exercise for health for early postmenopausal women: a systematic review of randomised controlled trials. *Sports Med*. 2004;34:753–78.
115. Taylor RS, Brown A, Ebrahim S, et al. Exercise-based rehabilitation for patients with coronary heart disease: systematic review and meta-analysis of randomized controlled trials. *Am J Med*. 2004;116:682–92.
116. Kelley GA, Kelley KS, Tran ZV. Aerobic exercise and lipids and lipoproteins in women: a meta-analysis of randomized controlled trials. *J Womens Health (Larchmt)*. 2004;13:1148–64.
117. Kelley GA, Kelley KS, Vu Tran Z. Aerobic exercise, lipids and lipoproteins in overweight and obese adults: a meta-analysis of randomized controlled trials. *Int J Obes (Lond)*. 2005;29:881–93.
118. Kelley GA, Kelley KS, Tran ZV. Walking and Non-HDL-C in adults: a meta-analysis of randomized controlled trials. *Prev Cardiol*. 2005;8:102–7.
119. Kelley GA, Kelley KS, Tran ZV. Exercise, lipids, and lipoproteins in older adults: a meta-analysis. *Prev Cardiol*. 2005;8:206–14.
120. Kelley GA, Kelley KS, Tran ZV. Walking, lipids, and lipoproteins: a meta-analysis of randomized controlled trials. *Prev Med*. 2004;38:651–61.
121. Kelley GA, Kelley KS. Aerobic exercise and HDL2-C: a meta-analysis of randomized controlled trials. *Atherosclerosis*. 2006;184:207–15.
122. Kelley GA, Kelley KS. Effects of aerobic exercise on lipids and lipoproteins in adults with type 2 diabetes: a meta-analysis of randomized-controlled trials. *Public Health*. 2007;121:643–55.
123. Kelley GA, Kelley KS. Impact of progressive resistance training on lipids and lipoproteins in adults: a meta-analysis of randomized controlled trials. *Prev Med*. 2009;48:9–19.
124. Kelley GA, Kelley KS. Impact of progressive resistance training on lipids and lipoproteins in adults: another look at a meta-analysis using prediction intervals. *Prev Med*. 2009;49:473–5.
125. Bravata DM, Smith-Spangler C, Sundaram V, et al. Using pedometers to increase physical activity and improve health: a systematic review. *JAMA*. 2007;298:2296–304.
126. Kodama S, Tanaka S, Saito K, et al. Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: a meta-analysis. *Arch Intern Med*. 2007;167:999–1008.
127. Gordon BA, Benson AC, Bird SR, et al. Resistance training improves metabolic health in type 2 diabetes: a systematic review. *Diabetes Res Clin Pract*. 2009;83:157–75.
128. Keogh JW, Kilding A, Pidgeon P, et al. Physical benefits of dancing for healthy older adults: a review. *J Aging Phys Activ*. 2009;17:479–500.
129. Orozco LJ, Buchleitner AM, Gimenez-Perez G, et al. Exercise or exercise and diet for preventing type 2 diabetes mellitus. *Cochrane Database Syst Rev*. 2008;CD003054.
130. Lin JS, O'Connor E, Whitlock EP, et al. Behavioral counseling to promote physical activity and a healthful diet to prevent cardiovascular disease in adults: a systematic review for the US Preventive Services Task Force. *Ann Intern Med*. 2010;153:736–50.
131. Cornelissen VA, Fagard RH. Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. *J Hypertens*. 2005;23:251–9.
132. US Department of Health and Human Services. 2008 Physical Activity Guidelines for Americans. Washington, DC: US Department of Health and Human Services:1–61. Available at: <http://www.health.gov/PAGuidelines>; 2008. Accessed January 28, 2014.

KEY WORDS: AHA Scientific Statements ■ cardiovascular disease ■ blood cholesterol ■ blood pressure ■ nutrition ■ dietary patterns ■ dietary fats ■ dietary sodium ■ physical activity

Appendix 1. Author Relationships With Industry and Other Entities (Relevant)—2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk

Committee Member	Employment	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Expert Witness
Robert H. Eckel, Co-Chair	University of Colorado, Anschutz Medical Campus—Professor of Medicine, Professor of Physiology and Biophysics; and Charles A. Boettcher II Chair in Atherosclerosis	2008–2012: • Foodminds 2013: • Foodminds	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None
John M. Jakicic, Co-Chair	University of Pittsburgh—Chair and Professor of Physical Activity and Weight Management Research Center	2008–2012: • Alere Wellbeing • JennyCraig • Nestle Nutrition 2013: • Calorie Control Council	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: • Body Media—PI 2013: • Body Media—PI	2008–2012: None 2013: None
Jamy D. Ard	Wake Forest University—Assistant Professor of Epidemiology and Prevention; Weight Management Center—Co-Director	2008–2012: • Arena Pharmaceuticals • Nestle Healthcare Nutrition • OPTIFAST Division • Vivus 2013: • Eisai • Nestle Healthcare Nutrition • OPTIFAST Division • Vivus	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None
Janet M. de Jesus, Ex-Officio	NHLBI—Nutritionist, Division for the Application of Research Discoveries	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None
Nancy Houston Miller	Stanford University School of Medicine, Department of Cardiology—Associate Director, Stanford Cardiac Rehabilitation Program	2008–2012: None 2013: • California Walnut Board	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None
Van S. Hubbard, Ex-Officio	National Institute of Diabetes and Digestive and Kidney Diseases—Director, NIH Division of Nutrition Research Coordination	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None
I-Min Lee	Harvard University—Professor of Medicine, Harvard Medical School	2008–2012: • Virgin HealthMiles 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None

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Appendix 1. Continued

Committee Member	Employment	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Expert Witness
Alice H. Lichtenstein	Tufts University, USDA Human Nutrition Research Center on Aging—Senior Scientist and Director, Cardiovascular Nutrition Laboratory Friedman School; Stanley N. Gershoff Professor of Nutrition Science and Policy	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	None	None	None
		2013:	2013:	2013:	2013:	2013:
		None	None	None	None	None
Catherine M. Loria, Ex-Officio	NHLBI—Nutritional Epidemiologist	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	None	None	None
		2013:	2013:	2013:	2013:	2013:
		None	None	None	None	None
Barbara E. Millen	Boston Nutrition Foundation—Chairman; Millennium Prevention—President	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	• Boston Nutrition Foundation* • Millennium Prevention*	None	None
		2013:	2013:	2013:	2013:	2013:
		None	None	• Boston Nutrition Foundation* • Millennium Prevention*	None	None
Cathy A. Nonas	New York City Department of Health and Mental Hygiene—Senior Advisor, Bureau for Chronic Disease Prevention and Tobacco Control	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	None	None	None
		2013:	2013:	2013:	2013:	2013:
		None	None	None	None	None
Frank M. Sacks	Harvard School of Public Health, Department of Nutrition—Professor of Cardiovascular Disease Prevention; Brigham and Women's Hospital—Senior Physician and Professor of Medicine	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	None	None	• Federal Trade Commission; Unilever, Keebler
		2013:	2013:	2013:	2013:	2013:
		None	None	None	None	None
Sidney C. Smith, Jr	University of North Carolina—Professor of Medicine; Director, Center for Cardiovascular Science and Medicine	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	None	None	None
		2013:	2013:	2013:	2013:	2013:
		None	None	None	None	None
Laura P. Svetkey	Duke University, Duke Hypertension Center—Professor; Duke Hypertension Center—Director; Clinical Research, Sarah W. Stedman Nutrition and Metabolism Center—Director	2008–2012:	2008–2012:	2008–2012:	2008–2012:	2008–2012:
		None	None	None	None	None
		2013:	2013:	2013:	2013:	2013:
		None	None	None	None	None

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Appendix 1. Continued

Committee Member	Employment	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Expert Witness
Thomas A. Wadden	University of Pennsylvania Perelman School of Medicine—Professor of Psychology, Psychiatry; Center for Weight and Eating Disorders—Director	2008–2012: • Alere Wellbeing • BMIQ • Novo Nordisk • Orexigen • Vivus 2013: • Novo Nordisk • Orexigen	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: • Novo Nordisk • Nutrisystem • Weight Watchers 2013: None	2008–2012: None 2013: None
Susan Z. Yanovski, Ex-Officio	National Institute of Diabetes and Digestive and Kidney Diseases, Division of Digestive Diseases and Nutrition—Co-Director, Office of Obesity Research	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None	2008–2012: None 2013: None

This table reflects the relevant healthcare-related relationships of authors with industry and other entities provided by the panels during the document development process (2008–2012). Both compensated and uncompensated relationships are reported. These relationships were reviewed and updated in conjunction with all meetings and conference calls of the Work Group during the document development process. Authors with relevant relationships during the document development process recused themselves from voting on recommendations relevant to their relationships. In the spirit of full transparency, the ACC and AHA asked Work Group members to provide updates and approve the final version of this table, which includes current relevant relationships (2013). To review the NHLBI and ACC/AHA's current comprehensive policies for managing relationships with industry and other entities, please refer to http://www.nhlbi.nih.gov/guidelines/cvd_adult/coi-rwi_policy.htm and <http://www.cardiosource.org/Science-And-Quality/Practice-Guidelines-and-Quality-Standards/Relationships-With-Industry-Policy.aspx>. *Per ACC/AHA policy:* A person is deemed to have a significant interest in a business if the interest represents ownership of $\geq 5\%$ of the voting stock or share of the business entity, or ownership of $\geq \$10\,000$ of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. Relationships that exist with no financial benefit are also included for the purpose of transparency. Relationships in this table are modest unless otherwise noted.

*Significant relationship.

ACC indicates American College of Cardiology; AHA, American Heart Association; NHLBI, National Heart, Lung, and Blood Institute; NIH, National Institutes of Health; PI, primary investigator; and USDA, US Department of Agriculture.

Appendix 2. Expert Reviewer Relationships With Industry and Other Entities—2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk

Reviewer	Employment	Representation	Consultant	Speaker's Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Nancy Albert	Cleveland Clinic Foundation—Sr. Director of Nursing Research and CNS, Kaufman Center for Heart Failure	ACC/AHA Task Force on Practice Guidelines	<ul style="list-style-type: none"> • BG Medicine • Gambro • Medtronic 	None	None	None	None	None
Gerald Fletcher	Mayo Medical School Mayo Clinic Jacksonville—Professor of Medicine	ACC/AHA	None	None	None	None	None	None
Frederick Kushner	Heart Clinic of Louisiana—Medical Director; Tulane University Medical Center—Clinical Professor	ACC/AHA	Federal Drug Administration Science Board†	None	None	None	None	None
Linda Van Horn	Northwestern University Feinberg School of Medicine—Professor, Preventive Medicine; Associate Dean, Faculty Development	ACC/AHA	None	None	None	None	None	None

This table represents the relationships of reviewers with industry and other entities that were self-disclosed at the time of peer review. It does not necessarily reflect relationships with industry at the time of publication. To review the NHLBI and ACC/AHA's current comprehensive policies for managing relationships with industry and other entities, please refer to http://www.nhlbi.nih.gov/guidelines/cvd_adult/coi-rwi_policy.htm and <http://www.cardiosource.org/Science-And-Quality/Practice-Guidelines-and-Quality-Standards/Relationships-With-Industry-Policy.aspx>.

†No financial benefit.

ACC indicates American College of Cardiology; and AHA, American Heart Association.

Appendix 3 Abbreviations

BP = blood pressure
COR = Class of Recommendation
CQ = critical question
CVD = cardiovascular disease
DASH = Dietary Approaches to Stop Hypertension
ES = evidence statement
HDL-C = high-density lipoprotein cholesterol
LDL-C = low-density lipoprotein cholesterol
LOE = Level of Evidence
MED = Mediterranean-style diet
NHLBI = National Heart, Lung, and Blood Institute
PICOTS = Population, Intervention, Comparator, Outcomes, Timing, and Setting
RCT = randomized controlled trial
RWI = relationships with industry and other entities
USDA = US Department of Agriculture