Depression screening in patients with heart disease in reply
Thombs, B.D.; de Jonge, Peter; Ziegelstein, R.C.

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other nutrients collected at baseline to evaluate the potential modifying effect of vitamin E or C treatment on cardiovascular disease and cancer. Male physicians in PHS II were predominantly well nourished, with adequate dietary and/or supplemental intake of vitamin C on randomization. Whether this had a clinically meaningful effect on the saturation of circulating immune cells and increases in plasma vitamin C remains to be seen yet counters the assertions from vitamin C proponents that mega-doses beyond that tested in PHS II are required to prevent cardiovascular events and other outcomes. Despite many promising mechanisms linking vitamins and chronic disease, PHS II and other trials of individual vitamin supplements have yet to provide any compelling rationale to recommend their use.

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Depression Screening in Patients With Heart Disease

To the Editor: The American Heart Association (AHA) recently recommended routine screening for depression in patients with coronary heart disease.1 Shortly thereafter, Dr Thombs and colleagues2 published a systematic review of the research on depression screening and clinical outcomes in cardiovascular patients. They found no studies that evaluated whether routine screening for depression produces better outcomes in patients with heart disease. They concluded that “the adoption of depression screening in cardiovascular care settings . . . would not be likely to benefit patients in the absence of significant changes in current models of care.” We disagree with some of their conclusions.

Symptoms of depression double the risk for mortality and other cardiac events in patients with coronary disease. The risk is even greater for patients with an interview-based clinical diagnosis of depression.3 Regardless of whether treating depression can improve cardiac outcomes, depression is a cardiac risk marker and its presence warrants more aggressive cardiac care and secondary prevention efforts. Even risk markers that are untreatable, such as age, compel physicians to increase efforts to achieve optimal levels of other more readily treatable risk factors. In addition, depression should be recognized because it is one of the strongest predictors of nonadherence with medical treatment regimens,4,5 indicating the need for careful monitoring.

Thombs et al noted that placebo-controlled antidepressant trials have demonstrated only modest efficacy in cardiac patients and offered this as another reason to forgo screening. Yet, as they also noted, antidepressants are just as efficacious in cardiac patients as in depressed but otherwise medically well psychiatric patients. Depression is a debilitating, often chronic, disorder. Patients with heart disease should not be deprived of the benefits of treatment, even if they are only modestly effective.

Finally, the authors concluded that “there are patients with serious and potentially life-threatening depression in most cardiovascular care settings. For these patients, physicians should provide appropriate treatment, referral, or both.” It is not clear how physicians will identify these patients if they do not screen for depression.

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In Reply: We agree with Dr Carney and colleagues that depression is an important condition that is associated with increased risk of morbidity and mortality in patients with heart disease and that warrants effective response by clinicians. The recent AHA science advisory concluded that routine depression screening in heart disease patients would accomplish that, but this recommendation was not based on a careful review of potential benefits and harms that is customary for a statement of this type. Carney et al do not find fault with the quality or rigor of our systematic review. Yet they support the AHA recommendation even though our review did not find any clinical trial that assessed whether depression screening improves depression or cardiac outcomes in heart disease patients or any other evidence that depression screening would likely benefit patients with cardiovascular disease.

Carney et al question how physicians will identify patients with depression and heart disease if they do not screen for depression. Unfortunately, there are many examples of screening programs implemented based solely on the desire to do good or out of fear and concern rather than on evidence of benefit to patients. Many such programs are later found to be unhelpful or even to cause harm. Recommending routine depression screening is not just a cry for greater awareness; it is a call for a significant deployment of health care resources when there are already considerable health care expenditures and a limited number of mental health clinicians. A higher standard must apply to such a recommendation, one that directly links screening with improved mental health or cardiac outcomes and one that weighs the magnitude of potential benefits and harms, typically based on a systematic review of the literature. The call for routine screening in patients with cardiovascular disease falls well short of this standard.

It is not clear how many patients would be detected by routine screening who would otherwise not be identified and who have severe enough symptoms of depression to benefit from treatment. Even if some otherwise unrecognized patients would be identified, others would be inaccurately labeled and potentially inappropriately treated in the absence of sufficient mental health resources to address high numbers of false-positive screens. These potential harms, as well as those resulting from treatment even when appropriate (eg, medication adverse effects), must be balanced against modest improvements in depressive symptoms to assess the overall effects of depression screening. The potential effect of misdirecting limited resources to patients inappropriately identified as depressed by screening, rather than addressing gross inadequacies in the routine care of already identified depressed patients, is daunting.

To call for routine screening is to make a huge leap across a wide gap in current evidence. We advise looking carefully before making that leap, recommend further study of the potential harms and costs of routine screening, and urge reconsideration of the AHA science advisory.

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Triglycerides, Depression, and Risk of Ischemic Stroke

To the Editor: In their prospective cohort study, Dr Freiberg and colleagues found that patients with elevated nonfasting triglyceride levels had an increased adjusted relative risk for ischemic stroke. The estimation of the hazard ratio was performed using Cox regression adjusted for age, sex, and other major cardiovascular risk factors such as hypertension, total cholesterol level, and smoking. However, depression as a risk factor was not included in the multivariate model, although some studies have demonstrated an association between stroke and mood disorders.

Depressed patients may have an increased risk of cardiovascular disease, and depression may be a predictor of recurrent cerebrovascular events, leading to higher morbidity and mortality rates. Furthermore, depression, through mechanisms not yet elucidated, may affect other