Behavior change and compliance: keys to improving cardiovascular health. Workshop VI. AHA Prevention Conference III

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Behavior Change and Compliance: Keys to Improving Cardiovascular Health

Workshop VI

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Many targets for cardiovascular disease interventions—eg, smoking, high serum cholesterol level, and high blood pressure—are more clear-cut and easily defined than stress. Rather than being a unitary construct that can be indexed by a single number or condition, stress results from personal and environmental factors and the interactions between those factors. Despite this complexity, recent research has shown that it is possible to define and measure specific sources of personal and environmental stress that appear to increase risk of cardiovascular disease. In this section we review (1) state-of-the-art research on the impact of these sources of stress on risk of cardiovascular disease, the mechanisms of impact, and the effects on risk of interventions to reduce stress; (2) gaps in research on stress and risk of cardiovascular disease; and (3) recommendations for public policies, educational programs, and research relevant to stress and cardiovascular disease risk.

State-of-the-Art Research

Two sources of chronic environmental stress appear to increase risk of cardiovascular disease: job strain and social isolation. Persons working in high-strain jobs, defined as high demands with low control over how the demands are met, had increased incidence of coronary heart disease in 12 of 14 studies and increased ambulatory blood pressure in eight cohort studies.[1] The association between social isolation (ie, low social support) and increased cardiovascular disease risk in healthy populations,[2] as well as poorer prognosis in patients with coronary heart disease has been well documented.[3] Another source of chronic stress linked to increased cardiovascular disease risk is low social class.[3,4]

Although harder to document, a growing body of evidence suggests that acute psychosocial stress may act as a trigger for acute coronary heart disease events in patients with atherosclerotic plaques that are vulnerable to rupture.[5,6] A biological mechanism for such effects is suggested by laboratory studies showing increased myocardial ischemia during mental challenge[7] and reduced ejection fraction during anger recall[8] in patients with coronary heart disease.

Personal sources of stress—psychological distress and depressive symptoms (not necessarily clinical depressive disorder)—have been associated with increased coronary heart disease risk in both healthy people[9] and patients with coronary heart disease.[10] A considerable body of epidemiological evidence indicates that a hostile personality profile—cynical attitudes toward others, frequent anger, and overt aggressive behavior—is another personal factor associated with increased incidence of cardiovascular disease.[11,12]

The stress factors identified above may contribute to increased cardiovascular disease risk by increasing sympathetic nervous system activity and reactivity. Both hostility[11,12] and social factors[13] might be involved in atherogenesis or the precipitation of acute coronary heart disease events through this biological pathway. More extensive evidence documents an indirect effect of stress on cardiovascular disease risk: increased levels of established risk factors (eg, cigarette smoking) in persons subjected to high stress from both personal[14,15] and environmental[16] sources.

Only a limited number of studies have evaluated the efficacy of interventions to reduce the effects of stress on cardiovascular disease, but the preliminary available evidence that interventions to reduce personal[17] and environmental[18] stress may reduce cardiovascular disease risk is encouraging. Pharmacological approaches also show promise in ameliorating the effects of stress on cardiovascular disease risk in animal models.[19]

Gaps in Research

The knowledge base for the role of stress factors in cardiovascular disease risk is far less extensive than that for established risk factors. The strongest and most consistent evidence documents the effect of social isolation, particularly in coronary heart disease patients. More data in larger samples will be required to establish the role of the stress factors identified above in cardiovascular disease risk.

Understanding of the mechanisms through which stress contributes to cardiovascular disease risk is similarly limited. Although there is no dispute that stress factors contribute to risk indirectly by increasing established risk factor levels, it remains to be seen whether any of the stress factors acts as an "independent" risk factor for cardiovascular disease, presumably by means of biological concomitants of acute and chronic stress.

Preliminary studies using psychosocial interventions in secondary prevention trials are promising, but the

studies are limited by sample size and methodological flaws.

Few data are available on the role of stress factors in cardiovascular disease risk among women and minority populations.

Recommendations

Public Policies

1. More resources should be allocated to support research on the role of stress factors in cardiovascular disease.

2. With the forthcoming changes in the health care system to improve the health of the population, provision should be made for the inclusion of preventive measures aimed at reducing the effect of stress factors on cardiovascular disease risk.

Educational Programs

1. The American Heart Association should help educate the public and the health care community about current understanding of the relation between personal and environmental stressors and cardiovascular disease risk.

2. The American Heart Association should assist in the development, implementation, and evaluation of stress reduction interventions in various settings, including the worksite* and community-based cardiac rehabilitation programs.

Research Needs

1. Research is needed to develop, and evaluate the benefits of, interventions to reduce the effect of stress factors on cardiovascular disease risk. Although secondary prevention efforts will be most informative early on, public health approaches to primary prevention by means of educational programs should begin soon. The outcomes of these intervention studies will not only help document the efficacy of the interventions but could add to the evidence that the targeted stress factors are contributing to cardiovascular disease risk.

2. Accelerated and expanded efforts are needed to increase understanding of the psychological, social, behavioral, and biological mechanisms whereby stress factors contribute to the etiology, course, and prognosis of cardiovascular disease. This research should include efforts to understand how stress factors interact, both with each other and with established risk factors, to increase risk.

3. Given the growing evidence for the role of stress factors in increasing cardiovascular disease risk, even if only an indirect contribution is clear at present, measures of the identified stress factors should be included in ongoing and planned population studies and clinical trials (eg, the tamoxifen trial in 15 000 women). This will not only enlarge the data base on the role of stress factors in cardiovascular disease risk but will also ensure that we take advantage of important opportunities to prevent cardiovascular disease.

*This is already under way under the auspices of the AHA's Program Committee.

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