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Social Support, Type A Behavior, and Coronary Artery Disease

Sheldon Cohen
Carnegie Mellon University, scohen@cmu.edu

Karen A. Matthews
University of Pittsburgh - Main Campus

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Editorial

Social Support, Type A Behavior, and Coronary Artery Disease

SHELDON COHEN, PHD AND KAREN A. MATTHEWS, PHD

Although four community studies have demonstrated a relation between social ties and mortality, we still know relatively little about the possible role of social relationships in the etiology of the leading cause of death, coronary heart disease (CHD). Recognizing the importance of this issue, scientists at two conferences sponsored by the National Heart Lung Blood Institute enthusiastically endorsed funding work to investigate links between the social environment and CHD. (1, 2). An additional recommendation was to pursue empirical and theoretical connections between social supports and other risk factors for CHD, including the Type A behavior pattern.

In consequence, it is particularly exciting that this issue includes two articles on the relations between various conceptions of social support and coronary artery atherosclerosis (CAD), the disease underlying most CHD. At the cutting edge of this field, these two articles, by Blumenthal et al. (3) and Seeman and Syme (4), provide critical evidence relevant to a possible etiologic role of social relationships in CHD. In addition to estimating the risk associated with social ties independent of the major biological risk factors, both articles have the attractive feature of estimating risk in the context of another behavioral risk factor, Type A behavior. Finally, both of these studies use the most up-to-date and reliable method of measuring CAD—coronary arteriography.

In this editorial, we raise a series of theoretical and methodologic issues to consider when reading these articles. We focus on conceptual questions regarding social support and Type A, and on methodologic issues in cross-sectional angiography studies. An awareness of these issues should aid in the design of future work that builds on the fascinating leads provided by the two articles.

CONCEPTUALIZATION OF SOCIAL SUPPORT

Although the term "social support" is often used as if it refers to a commonly defined characteristic of the social environment, existing studies apply the term to a broad range of conceptualizations of social networks and the functions that they provide. As noted by Seeman and Syme, the distinction between social network structure and the functional or support

Dr. Cohen is from the Department of Psychology, Carnegie-Mellon University, Pittsburgh, PA 15213, and Dr. Matthews is from the Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA 15213. Reprint requests may be addressed to either Dr. Cohen or Dr. Matthews.

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characteristics of network ties (5, 6) is a necessary first discrimination. However, even within these categories there are diverse ranges of conceptions. An especially laudatory aspect of the Seeman and Syme article is their recognition that there are multiple conceptions of social support and that these conceptions may have independent associations with disease. Future studies should go one step further and provide specific tests of plausible psychosocial-biologic models that explain *how* each conception of social support influences the etiology of CAD. It is only with convincing models of how the social environment triggers biologic changes associated with disease pathogenesis that data from these studies can greatly contribute to understanding risk for CAD.

A discussion of specific alternative causal pathways linking various conceptions of support to CAD is beyond the scope of this editorial (5, 7, 8). However, three general points are critical to consider. First, some conceptions of social support are presumed to operate by directly (main effect) influencing the atherosclerotic process, while others operate by short-circuiting the atherosclerotic influence of psychosocial stressors (life stress × support interaction) (9). To adequately test the relative importance of these two models, measures of life stress should be included in study designs.

Second, effects of support on the pathogenesis of CAD may be mediated by influences of support on traditional risk factors. For example, social networks may discourage smoking, encourage low cholesterol diets, and aid in blood pressure control by enhancing compliance with medical regimens. Studies that control for standard risk factors may be underestimating the influence of support on disease by subtracting effects of risk that actually mediate the support-disease association (10). The Seeman and Syme study is noteworthy because it tested for the relations between social support and risk factors for CHD. Future studies should similarly address these possible pathways.

Third, it is important to choose conceptualizations of social support that are plausible risk factors in light of the long-term pathogenesis of CAD. For a risk factor to contribute to pathogenesis, one must assume either that it is stable over a significant period of the development of the disease, or that it is salient enough that short-term exposure is sufficient to influence the disease process. Although there is limited evidence on the temporal-stability of various conceptions of support, existing data suggest that satisfaction with support is relatively unstable; perceived availability of support is moderately stable; and integration in a social network may be stable over a year or more. Because atherosclerosis has a long developmental period, the appropriateness of relatively unstable support measures in this context is questionable.

CONCEPTUALIZATION OF TYPE A BEHAVIOR AND SOCIAL SUPPORT

Type A has been characterized as a behavior pattern that is elicited from susceptible individuals by an appropriately challenging, or stressful environment. Existing epidemiologic literature, including the present articles, have focused on the CHD risk associated with Type A classification *without* consideration of life stress. In consequence, the predicted interaction of Type A with appropriately eliciting situations has been ignored. It is possible that

this interaction is not important. Type As may almost always be under greater risk because they create and perceive more challenge than Bs (11); or the existing definition of Type A may be incorrect and As may be at greater risk irrespective of challenge (12). Studies including measures of life stressors that are presumed to elicit the Type A pattern would provide important information in this regard and perhaps improve the ability to predict disease.

Although the Blumenthal et al. study did not test for the interactive effect of Type A and challenging environments, it did test a provocative hypothesis stemming from a similar concern about the absence of measurement of environmental variables in Type A studies: Type As who experience a supportive environment would have less CAD than the remaining Type As. The Blumenthal et al. study provides support for this hypothesis. However, data from the Seeman and Syme study was not supportive. Neither measure of perceived emotional support nor any other of the support measures used in the latter study interacted with Type A.

Blumenthal et al. argue that social support reflects an environmental influence that protects persons from the pathogenic consequences of being a Type A. The assumption that social support is an environmental factor can be questioned. For example, social ties may act as a proxy for personality factors that influence the ability to make and maintain ties, e.g., social skills or neuroticism, and perceptions of support may result from personality driven distortions of the social environment (13–15). Thus, Blumenthal et al. may have identified in their analyses the subset of Type As whose supportive environment, or whose (mis)perceptions of support from their environment are attributable to stable individual differences in perceptual styles or social skills.

Whether or not social support in that study was an environmental or personality variable, the hypothesis that support buffers Type A persons has merit. Assume that the risk of CAD for Type As is increased only under challenge. Because high levels of perceived emotional support reduce the extent of perceived challenge and life stress (9, 16), they should also reduce the overall risk associated with Type A. The Blumenthal et al. study assessed perceptions of emotional support, and hence their results can be viewed as consistent with this hypothesis. However, neither the Blumenthal study's more general prediction that support will provide protection for Type As, nor our argument that support reduces risk by reducing the extent of perceived challenge adequately explains the interaction that was actually found in that study. In particular, they both fail to address the question of why CAD risk for Type Bs increases with higher support levels. Limited space prevents us from proposing speculative explanations for this effect. However, unexpected responses by Type Bs are not uncommon in this literature, and research on how Bs understand and cope with the world might help explain relative differences between As and Bs in disease risk.

It has been suggested that because of their hostility, Type As do not develop adequate social networks and that, at least to some degree, their increased risk for disease is caused by this isolation (5, 12). This hypothesis was not supported by either of the angiographic studies. Neither study found associations between perceived social support and Type A or between social support networks and Type A and Cook-Medley Hostility scores. It is possible,

however, that in a general population study, such a relation would be obtained (see below).

Finally, both studies reported that after adjustment for traditional risk factors, Type As assessed by the Structured Interview (SI) had more CAD than Type Bs. Previous studies using other assessment tools (questionnaires) have been unsuccessful in this context, whereas roughly half the studies using the SI have demonstrated an association of Type A and CAD (17). The reliability of the Type A effect across these two studies lends further support to the etiologic importance of Type A as measured by the SI.

CONSEQUENCES OF STUDY DESIGN

It is important to review the strengths and weaknesses of cross-sectional angiographic research to properly interpret the data from both studies. Cross-sectional angiography studies provide a good tool for the initial examination of relations between hypothesized psychosocial risk factors and coronary artery disease. They provide a "hard" biologic marker of coronary atherosclerosis and hence do not suffer from the diagnostic and ascertainment biases characteristic of studies using clinic outcomes. Angiographic studies also provide a semicontinuous disease measure, an opportunity to study risk factors in association with the natural history of CAD, and (in most cases) a thorough risk factor assessment associated with hospitalization for this procedure.

There are, however, a number of disadvantages associated with such designs (18, 19). The most serious problems result from biases in angiographic referral procedures that constrain the variances of risk factors and the extent of coronary artery occlusion, and bias the representativeness of the sample. People in these samples who are found to have CAD may not be representative of the population of CAD cases. Excluded are persons who fail to seek medical care in response to symptoms, those whose initial clinical manifestations were fatal, and symptomatic persons who are not referred for angiography because they do not have high levels of the traditional risk factors. Moreover, those who are relatively free of disease may not be representative of the "healthy" population as a whole. Instead, these persons manifest the relatively rare symptoms and elevated risk factor protocol required for angiographic referral. The influences of these biases are compounded when they are associated with risk factors being evaluated in the study. For example, persons with strong support networks may be overrepresented in such samples because concern shown by network members increases their likelihood of seeking treatment after symptom onset.

As a result of these biases, cross-sectional angiographic studies must be interpreted with caution. Failure to find an association between a risk factor and CAD must be interpreted in light of relatively severe constraints on variance in both risk factor and disease outcome. Success in finding an association is evidence for persons with CAD manifest in symptoms (but not in fatal events) being different than those with symptoms but no CAD. Such a relation may or may not be relevant to discriminating between the sick and healthy in the general population. Moreover, as in all cross-sectional studies, angiographic findings are open to alternative causal inferences. For example, persons with more serious CAD may alienate their social networks by overusing available aid while attempting to cope with previous symp-

toms. Hence, the disease might cause less available support rather than the support causing less disease.

CONCLUSIONS

The two angiography studies reported in this issue provide provocative data on the risk for CAD posed by various conceptions of social support and by Type A behavior. This work should not, however, be viewed as an end in itself but rather as an impetus for further studies that provide more detailed specifications and tests of models linking various conceptions of social support to CAD pathogenesis. Replication of the cross-sectional studies reported in this issue, longitudinal studies of persons undergoing repeated angiographic procedures, and conceptually sophisticated prospective studies of social support and Type A behavior as risks in CHD incidence are all necessary before we can establish the combined roles of social supports and Type A in CAD risk.

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