yet still experienced ill health may do more to erode a sense of well-being than diagnosed disease (3). Furthermore, the limitations of statistical adjustment in situations where correlated covariates are measured imprecisely are well known (6). Adult occupational class is a crude index of social position across the life course and the exposures associated with this. A growing number of studies show that socioeconomic disadvantage at various life-course stages independently predicts cardiovascular disease mortality (7, 8) and is associated with adult psychosocial characteristics such as hopelessness and hostility (9). A categorization of smoking status into current, former, and never smokers similarly provides a limited index of lifetime tobacco exposure.

Even with these imprecise measures, confounding appeared to account for approximately half of the apparent protective effect of higher SOC on cardiovascular disease mortality in men (a rate ratio of 0.75 associated with higher SOC was attenuated to 0.87). Therefore, the probability of residual confounding in relation to most estimates appears strong. The nonspecific character of the association between SOC and both cancer and cardiovascular disease mortality is another indicator of its potentially artifactual basis. The importance of specificity as a criterion for establishing causality has perhaps been underestimated (10, 11). Interestingly, investigators in the EPIC-Norfolk Study previously reported a strong, apparently protective but nonspecific association of vitamin C levels with cardiovascular and other causes of death (12). Evidence from a randomized trial now strongly suggests that this was noncausal (13), and empirical data demonstrate the degree to which confounding by life-course socioeconomic circumstances could have generated such an artifact (14). All of these considerations suggest that some degree of caution is warranted when attempting to draw causal conclusions based on observational evidence of a general association between greater SOC and better health.

Finally, adopting a “positive psychology” toward one’s life’s circumstances is an admirable and attractive idea, but achieving this goal may be difficult for persons facing manifest disadvantage. Indeed, as clinicians and public health advocates, we need to ask ourselves whether it is even appropriate that they be counseled to do so.

REFERENCES


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THE AUTHORS REPLY

We thank Dr. Macleod and colleagues for their comments (1). Macleod et al. make a number of broad-ranging observations on our recent paper (2) and another, unrelated publication (3) that concern residual confounding, disease-specific associations, clarity in risk factor specification, and interpretation of results. These issues are central to the professional practice of public health observational epidemiology but are occasionally addressed with ideologically driven stridency in response to studies evaluating psychosocial factors as determinants of chronic disease (4).

Macleod et al. refer to residual confounding due to smoking status, social class, and undiagnosed ill health as likely explanations for the associations observed between sense of coherence (SOC) and mortality in our study. The large correlation observed between SOC and both smoking status and social class indicates a potential source of confounding. However, these observed correlations did not account for the associations with mortality. A substantial attenuation in effect size following these adjustments might suggest residual confounding as an explanation for the
remainder of the association. However, while these adjustments resulted in a 50 percent attenuation for the association between SOC and cardiovascular disease mortality in men, this was not true for the other eight results presented in the same table (table 3 (2)). We believe interpretation of study findings should be based on the balance of the evidence presented, not on a single result that may or may not be in conflict with the rest of the data. Undiagnosed ill health represents another potential source of residual confounding, and for this reason we included figure 1, which shows that the association with all-cause mortality was consistent across the duration of follow-up (up to 6 years) and, importantly, did not become attenuated over time. Residual confounding arising from imprecisely measured, unavailable, or unknown risk factors is an issue that is relevant to all observational epidemiologic studies. However, we would reiterate that, within the limitations of these data, we have shown that the association between SOC and mortality was observed after adjustment for traditional mortality risk factors, was unaffected by the exclusion of study participants identified as having chronic disease at baseline, was consistent by sex and time since follow-up, conformed to a dose-response relation, and was observed within strata of age, cigarette smoking history, and social class (2).

Furthermore, in our paper we do not dismiss the possibility of noncausal explanations. However, while the importance of specificity in establishing causality may have been underestimated, a lack of specificity does not imply that an association is noncausal (5, p. 25). We agree that caution is warranted in drawing conclusions based on observational data, but we reiterate that, to our knowledge, our paper is the first formal test of the hypothesis that a strong SOC is associated with reduced mortality. Our interpretation of this intriguing association remains cautious and requires confirmation.

Understanding of how and why a strong SOC is associated with reduced mortality is limited. Evidence is accumulating which suggests that personal coping dispositions, including SOC, may have an impact on the immune system (6–8), but evidence from community studies remains sparse (9). Antonovsky defined SOC as representing a global orientation that included cognitive, instrumental, and motivational components (10). His definition developed through recognition that adaptation could occur for some people even following the trauma of a concentration camp experience (11). It was designed to measure individual differences in adaptation to adverse experiences and disadvantage (12). Further investigation is now needed to improve our understanding of SOC and to establish whether or not Antonovsky’s ideas and those represented by the emerging psychological science of positive psychology and resilience (13, 14) can contribute to improved understanding of the considerable individual and subgroup variability in chronic disease.

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