meta-analyses of passive smoking and coronary heart disease have been addressed elsewhere in the literature. Specifically, results in male and female patients are sufficiently homogeneous to allow their combination.[3,4] The various studies can be, and were, given meaningful quality scores.[3,4] The questions of misclassification of smoking status and exposure status have been adequately dealt with, as has publication bias.[3] Adjustment has been made for potential confounders,[2-4] and positive trends with dose were found for 16 of 22 studies without the inclusion of the nonexposed group.[4]

Bailar is also worried that the pooled relative risk of coronary heart disease associated with passive smoking is large as compared with the risk associated with active smoking. The ratio of excess risks, active to passive, is about $(1.93-1)\div(1.24-1)$, or 3.9, when the risk for active smokers is measured against that for nonexposed persons who have never smoked. This is not unusual for an anatomical site that is not in direct contact with tobacco smoke and for which the dose–response curve is convex upward.[5] For comparison, the best study we have on breast cancer and active and passive smoking[6] found an active-to-passive ratio of excess risks of only $(3.0-1)\div(2.3-1)$, or 1.5. Although the data are fewer, similar low ratios appear to hold for other noncontact cancer sites, such as the cervix, liver, and brain, and for lymphoma and leukemia. Probably these ratios are low because so many of the entities in environmental tobacco smoke that cause coronary heart disease and cancer are in the vapor phase[7]; they therefore are deposited more completely in the lung and are harder to clear than particle deposits. The toxins must be cleared into the body fluids, where they can circulate to distant sites, such as the heart or breast.

Bailar appears to prefer a "thoughtful review of the usual type."[1] If so, he should read the review by Kritz et al.,[8] which covers the same ground as the study by He et al.[2] and others,[3,4] but without the meta-analysis. Of course, Kritz et al. also conclude that environmental tobacco smoke causes coronary heart disease.

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To the Editor: Many readers would dispute Bailar's conclusion that "we still do not know . . . whether exposure to environmental tobacco smoke increases the risk of coronary artery disease." The evidence available to mid-1997 on this topic was reviewed by Australia's National Health and Medical Research Council (NHMRC).[1] The NHMRC considered 22 analyses from 16 studies; 17 of the 22 analyses indicated some increase in the risk of coronary events among nonsmokers with exposure to environmental tobacco smoke, and in 8 of them the results were statistically significant. Rather than undertake a quantitative meta-analysis, the NHMRC summarized the data in terms of a median relative risk and corresponding interquartile range. The median estimate of 1.24 (interquartile range, 1.02 to 1.62) is entirely consistent with the pooled estimate of 1.25 (95 percent confidence interval, 1.17 to 1.32) derived by He et al. and was supported by findings of excess risks of mortality from all causes in seven of eight prospective studies of passive smoking.

The report from the NHMRC also examined the relation between passive smoking and coronary heart disease in light of the criteria proposed by Hill[2] and concluded that "all the evidence put together is reasonably coherent."[1] Like Bailar, the NHMRC drew attention to the relatively large excess risk of coronary heart disease associated with passive smoking as compared with the risk attendant on active smoking, but it cited evidence that platelet function in nonsmokers is particularly sensitive to exposure to environmental tobacco smoke.[3]

Whatever the limitations of meta-analysis, the abundant evidence that passive smoking causes harm to health can no longer be ignored.

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2. Hill AB. The environment and disease: association or causation? Proc R Soc Med 1965;58:295-300.
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To the Editor: In his editorial, Bailar uses several flawed arguments. His concern about reporting bias might be appropriately applied to the case–control studies, but not to the cohort studies (10 of the 18 studies analyzed). Moreover, when the two types of studies were analyzed separately by He et al., the conclusions were strikingly similar. The possibility of publication bias was also raised. This is often a valid criticism of meta-analyses, but it appears misdirected in this instance. Specifically, of the 18 studies reviewed, 7 found a significant association and 11 did not. If anything, the bias here is likely to be very small. In addition, random reporting errors, also mentioned by Bailar, are likely to attenuate and not spuriously strengthen these associations.

Bailar considers the range of relative risk of about 1.0 to 2.2 in He et al.'s study to be "very small." He believes that uniformity in results is "not necessarily good." Such comments are only his opinion, and not facts. Finally, Bailar criticizes the use of a "multiplicative model," but meta-analysis must use the models of the original studies and