Sesso et al. (1) recently reported a lack of association of physical activity with cardiovascular disease (CVD) for older women by using an analysis strategy that included only a single measure of physical activity in 1962 and a subsequent 31-year follow-up for CVD. In 1996, three of us (2) reported on a relatively strong association between physical activity and all-cause as well as CVD mortality for subjects tracked over a similar period (1965–1994). Unlike Sesso et al., we measured physical activity at three time points over the follow-up period; however, we did not examine results separately by gender.

To test the hypothesis that associations for women between physical activity and subsequent CVD would be strengthened with more frequent assessments, we repeated our earlier analyses but focused only on women and included exclusions and coding of potential confounders to make our comparisons as similar as possible to those performed by Sesso et al. (1) (shown in their table 3). Thus, we focused on women aged 35–75 years at baseline, excluded those with self-reports of heart disease or stroke, and included adjustments for age, diabetes, high blood pressure, body mass index as a continuous variable, and cigarette smoking. Following the methods of Sesso et al., we included two sets of analyses: one adjusting only for age and the other adding all other variables. A total of 2,027 women were eligible, and their mean age was 50 years. Over the 28-year follow-up period, 190 cardiovascular deaths occurred. Person-years totaled 36,172.

Because we had three possible assessment periods (1965, 1974, and 1983), we were able to compare baseline-only assessment of physical activity with results obtained when all assessment periods were included in the statistical models. As before, we used Cox proportional hazards models with the time-dependent covariate option for comparisons in which changes in physical activity during follow-up were included (3). Deaths were included through 1994. Subjects who did not die of CVD were censored at loss to follow-up, at death from causes other than CVD, or at the end of 1994.

Our measure of physical activity is similar to that used by Sesso et al. (1), except that we did not convert our values to metabolic equivalencies. Instead, we constructed a scale from three questions on frequency of physical exercise, taking part in active sports, and taking long walks or swimming. Responses to these questions were never, sometimes, or often and were scored as 0, 2, or 4, respectively. The resulting physical activity scale ranged from 0 to 12. Mean scores were 3.8 in 1965 and 1974, followed by an increase to 4.2 in 1983. However, there was considerable individual variation; for example, only 28 percent of the women who survived to 1983 reported the same level of physical activity in 1983 as in 1965.

Our results, shown in table 1, indicate a much weaker association for women aged 35–75 years between physical activity and cardiovascular mortality when only baseline data are included compared with the results obtained when up to three assessment periods are included. Although we used cardiovascular mortality rather than incidence of disease as an outcome, these analyses strongly suggest that the weak association between physical activity and CVD reported by Sesso et al. (1) was caused by using only a single assessment of activity at baseline over a very long follow-up period.

**REFERENCES**


**THREE OF THE AUTHORS REPLY**

We appreciate the comments and data from Strawbridge et al. (1) regarding our recent article on physical activity and the risk of cardiovascular disease (CVD) in women (2). We agree that use of a single measurement of physical activity at baseline in 1962, with 31 years of subsequent follow-up for CVD, is not the optimal way to test the hypothesis that
physical activity is associated with the risk of CVD. We did address this issue among the acknowledged limitations of our study, noting the importance of including multiple longitudinal measurements of physical activity (3), as is done with other risk factors for chronic disease (4). In addition, we assessed physical activity as a time-dependent variable updated on the 1980 questionnaire, finding a marginally significant inverse association with CVD risk in a multivariate model (p = 0.076) with a relative risk of 0.72 for each 100 kcal/week increase in physical activity.

Here, we expand our previous analyses and provide more detailed information on findings when physical activity was updated. We used Cox proportional hazards models to update physical activity in 1980. We found that, as noted previously, the possible inverse association between physical activity and risk of CVD was somewhat strengthened for a subset of 962 women who returned both the 1962 and 1980 questionnaires. The relative risks for women expending <500, 500–999, and ≥1,000 kcal/week were 1.00 (reference), 0.72 (95% confidence interval: 0.43, 1.19), and 0.66 (95% confidence interval: 0.42, 1.03), respectively. Had we considered time-dependent models including all 1,562 women and updating physical activity for only those who provided data in 1980, the relative risks for women expending <500, 500–999, and ≥1,000 kcal/week would have been 1.00 (reference), 0.77 (95% confidence interval: 0.52, 1.13), and 0.71 (95% confidence interval: 0.50, 1.00), respectively. This assumes that women who were not censored through 1980 but did not provide updated information on physical activity maintained the physical activity levels consistent with their 1962 baseline value.

How time-dependent models change the interpretation of results must be considered carefully. In these two studies, physical activity was measured at least 9 years apart, still enabling the models to reflect a more “chronic” effect. However, in studies with frequent exposure assessments of shorter duration, the “acute” effect modeled in time-dependent models may be less appropriate for chronic diseases. Instead, the cumulative average method may better reduce within-person variation when multiple exposure measurements are used to capture long-term lifestyle and dietary patterns (4).

REFERENCES


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ERRATA

RE: “RELATION BETWEEN OBESITY AND BREAST CANCER IN YOUNG WOMEN”

Table 1 of this article contains a minor error. In this table, the 95% confidence interval for age at menarche was published as 1.64, 0.93. The correct confidence interval is 1.64, 1.93.

The Journal regrets the error.

REFERENCES


RE: “EPIDEMIOLOGY OF GESTATIONAL WEIGHT GAIN AND BODY WEIGHT CHANGES AFTER PREGNANCY”

The authors of this paper, which appeared in Epidemiologic Reviews, were inadvertently omitted from the beginning of the article. The authors are Eric P. Gunderson and Barbara Abrams.

The Journal and the printer regret the error.

REFERENCES
