A Prospective Study of Passive Smoking and Risk of Diabetes in a Cohort of Workers

The High-Risk and Population Strategy for Occupational Health Promotion (HIPOP-OHP) study

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OBJECTIVE — We investigated the impact of active smoking and exposure to passive smoke on the risk of developing diabetes.

RESEARCH DESIGN AND METHODS — Data were analyzed from a cohort of participants in the High-Risk and Population Strategy for Occupational Health Promotion Study (HIPOP-OHP) conducted in Japan from 1999 to 2004. Active and passive smoking status in the workplace was evaluated at baseline.

RESULTS — Of 6,498 participants (20.9% women), a total of 229 diabetes cases were reported over a median 3.4 years of follow-up. In the workplace, compared with zero-exposure subjects, the multivariable-adjusted hazard ratios of developing diabetes were 1.81 (95% CI 1.06–3.08, $P = 0.028$) for present passive subjects and 1.99 (1.29–3.04, $P = 0.002$) for present active smokers.

CONCLUSIONS — In this cohort, exposure to passive smoke in the workplace was associated with an increased risk of diabetes after adjustment for a large number of possible confounders.

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A positive association between active smoking and the incidence of diabetes has been identified (1–3). Only one study has shown a significant association between passive smoke and impaired glucose tolerance (4), and the association between exposure to passive smoke and the risk of developing diabetes has not been fully investigated. Here, we examine the relationship between exposure to passive smoke in the workplace or at home and the risk of developing diabetes in a large sample from a nonrandomized health promotion intervention study conducted at workplaces in Japan.

RESEARCH DESIGN AND METHODS

Analyses were performed using baseline and annual follow-up data from the High-Risk and Population Strategy for Occupational Health Promotion Study (HIPOP-OHP) conducted between 1999 and 2004 at 12 large-scale companies, excluding prevalent diabetes cases or those who did not report active or passive smoking status at baseline. Full-time employees at the worksites were enrolled, and then the worksites were nonrandomly assigned to either the intervention or control groups (13–20). All participants underwent an annual health check including blood testing at baseline and thereafter. A history of diabetes as well as lifestyle variables such as daily alcohol intake and smoking habits were evaluated using a self-administered questionnaire (5–7).

We constructed the following four categories by combining active smoking status and passive smoking status at workplace or at home as follows: *1*) "zero exposure" included those who never smoked and were not currently exposed to passive smoke; *2*) "past active only" included those who had smoked in the past but did not currently smoke and were not currently exposed to passive smoke; *3*) "present passive" included those currently exposed to passive smoke but who did not actively smoke, irrespective of past smoking; and *4*) "present active" included those who currently smoke irrespective of exposure to passive smoke.

A subject was considered diabetic if at least one of the following parameters was met: fasting blood glucose level ≥126 mg/dl (≥7.0 mmol/l), random plasma glucose level ≤200 mg/dl (≥11.1 mmol/l), or treatment with hypoglycemic medication (insulin or oral hypoglycemic agent). A self-reported history of diabetes was also accepted, since self-reported diagnosis of diabetes has been shown to be reliable (8) and has been used in many cohort studies (9,10).

Statistical analyses

We used the Cox proportional hazards model to analyze the association between passive smoking and incident diabetes cases. Person-time was calculated from the return of the baseline questionnaire until the date of the annual health check, at which the diagnosis of diabetes was

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Table 1—*Age-adjusted baseline characteristics of the study participants according to active and passive smoking status for men and women, aged 19–69 years (1999–2000, HIPOP-OHP, Japan)*

	Smoking status			
	Zero exposure	Past active only	Present passive	Present active
Participants (*n*)	2,129	779	690	2,900
Age (years)	36.9	41.7	39.3	37.9
Female (%)	45.5	6.4	30.6	4.5
BMI (kg/m^2)	22.3	22.7	22.7	22.9
Physical activity (MET h/week)	4.8	6.7	6.3	4.6
Alcohol (g/day)	11.2	22.7	15.7	26.9
Family history of diabetes (%)	20.1	16.1	18.5	18
Hypertension (%)	10.3	19.8	11.3	12.3
Health promotion intervention (%)	46.0	41.6	46.7	44
Frequency of sweetened beverage intake ≥1/day (%)	20.5	17.6	19.0	19.7
Frequency of vegetable intake <1/week (%)	47.3	45.9	46.7	47.2
Do not care about eating too much fat at all (%)	18.2	16.4	17.3	17.8

MET h, metabolic equivalent hours.

confirmed, or the end of the follow-up, whichever occurred first.

We evaluated the effect of active smoking and exposure to passive smoke on the risk of developing diabetes in a multivariable-adjusted model, adjusting for all variables listed in Table 1. Likelihood ratio tests were used to test statistical interactions between passive smoking status and sex, BMI, or assigned intervention.

RESULTS

Of the 6,498 participants (20.9% women), 44.6% were current smokers (average of 19.6 cigarettes smoked per day), while 12.6% reported exposure to passive smoke in the workplace. Approximately 32% of participants dropped out during the follow-up.

Age-adjusted baseline characteristics of the study participants are summarized in Table 1 by active smoking and exposure to passive smoke at workplace. In the workplace, compared with zero-exposure subjects, the multivariable-adjusted hazard ratios (HRs) for past active–only subjects, present passive subjects, and present active smokers were 1.15 (95% CI 0.66–2.03, $P = 0.62$), 1.81 (1.06–3.19, $P = 0.028$), and 1.99 (1.29–3.04, $P = 0.002$), respectively, in the analysis including all subjects; 1.23 (0.56–2.73, $P = 0.60$), 2.76 (1.38–5.50, $P = 0.004$), and 2.09 (1.14–3.82, $P = 0.017$), respectively, in the control group; and 1.19 (0.23–2.71, $P = 0.84$), 0.70 (0.25–1.92, $P = 0.50$), and 1.99 (1.07–3.70, $P = 0.03$), respectively, in the intervention group. We did not observe statistically significant interactions between exposure to passive smoke and sex ($P = 0.74$, 1 d.f., $\chi^2 = 0.60$), obesity ($P = 0.77$, 1 d.f., $\chi^2 = 0.08$), or health promotion intervention ($P = 0.087$, 1d.f., $\chi^2 = 0.60$). At home, the multivariable-adjusted HRs for past active–only subjects, present passive subjects, and present active smokers were 0.97 (0.59–1.60, $P = 0.92$), 0.80 (0.46–1.40, $P = 0.44$), and 1.42 (0.98–2.04, $P = 0.062$), respectively.

CONCLUSIONS

— In this 4-year prospective study conducted in the workplace, self-reported exposure to environmental tobacco smoke in the workplace and current active smoking at baseline were positively associated with an increased risk of developing diabetes, even after adjustment for a large number of possible confounders. To our knowledge, only one study has explored the association between exposure to passive smoke and subsequent risk of diabetes, which yielded similar results to our study, although not statistically significant (4). A possible limitation of our study is that the results might be underestimated by time-dependent confounding by smoking status; in fact, exposure to passive smoke in the workplace was not associated with the risk of diabetes in the intervention group, possibly due to lowered exposure to passive smoke by intervention. These findings add new evidence to support the need for measures to lessen environmental tobacco smoke in the workplace, especially in Asian populations, in which both the genetic susceptibility to diabetes (11,12) and smoking rate (13) are generally high.

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APPENDIX

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