Mortality among "never smokers" living with smokers: two cohort studies, 1981-4 and 1996-9

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Few studies have examined the association between passive smoking and all cause mortality; most of these have had limited study power.[1–4] We present results from two population cohorts of adults who had never smoked ("never smokers"), followed for three year mortality according to household exposure to secondhand smoke.

Participants, methods, and results

The two cohorts comprised all New Zealand adults aged 45-74 years who responded to the 1981 and 1996 censuses and who identified themselves as never smokers, lived in a private dwelling (that is, not a prison, hospital, or other institution), and had provided data on smoking status for all household members aged 15 and over (87.0% of never smokers in 1981 and 85.3% in 1996).

Never smokers living in households with one or more current smokers were regarded as being exposed to secondhand smoke in the home; those living in households with no current smokers were regarded as not exposed. Cohort members were followed for mortality in the three years after the census by means of anonymous probabilistic linkage with a national register of mortality records.[5] Record linkage was complete for 71.0% of eligible mortality records during 1981-4 and for 78.2% during 1996-9. Data were weighted to adjust for potential linkage bias.[5]

We calculated mortality and standardised for age and ethnicity using the 1996 census population as the standard. We used Poisson regression to adjust for age, ethnicity, marital status, and socioeconomic position, using a more restricted cohort with full demographic data (82.3% of the 1981 cohort and 89.9% of the 1996 cohort).

In both cohorts and sexes, mortality among never smokers was greater in those living in households with a current smoker (table).

Comment

Among adults who had never smoked we found a modest but consistent association between exposure to secondhand smoke in the home and mortality. This association persisted after adjustment for age, ethnicity, marital status, and socioeconomic position. The finding of about 15% excess mortality in never smokers exposed to secondhand smoke at home is consistent with the previous largest study in this area.[1]

Mortality and mortality rate ratios were standardised by age and ethnicity, and further adjustment for marital status and socioeconomic position altered the results only slightly. This suggests that these factors were not important confounders (independent of age and ethnicity). We could not adjust directly for lifestyle characteristics as these data are not included in the census. However, lifestyle factors are unlikely to act as important confounders when there is no confounding by socioeconomic position.

We considered exposure to secondhand smoke in the home only. Our inability to measure exposure in other settings introduces a degree of exposure misclassification; mortality rate ratios will probably be underestimated as a consequence. We suspect that this misclassification will be greater for the 1981-4 cohort,

A security statement about the New Zealand census-mortality study (NZCMS) is on bmj.com

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All cause mortality among adults who have never smoked, by household exposure to secondhand smoke, 1981-4 and 1996-9

Cohort	No of deaths	Person years	Standardised mortality (per 100 000 a year)	Rate ratio (95% confidence interval)	
				Standardised*	Adjusted†
1981-4					
Men:					
No exposure	3240	211 852	1530.4		
Exposure	846	57 344	1683.6	1.10 (0.99 to 1.22)	1.17 (1.05 to 1.30)
Women:					
No exposure	4902	435 423	1009.8		
Exposure	1200	138 675	1050.4	1.04 (0.96 to 1.13)	1.06 (0.97 to 1.16)
1996-9					
Men:					
No exposure	3684	387 292	1024.6		
Exposure	687	63 244	1198.3	1.17 (1.05 to 1.31)	1.16 (1.04 to 1.30)
Women:					
No exposure	4026	578 216	671.6		
Exposure	756	100 507	854.8	1.27 (1.15 to 1.41)	1.28 (1.16 to 1.42)

Raw numbers are randomly rounded to a near multiple of three, as per Statistics New Zealand's protocol. (Mortality and regression analyses are calculated using exact counts.)
*Standardised by age (five year age bands) and ethnicity (Maori, Pacific, and neither Maori nor Pacific).
†Adjusted for age, ethnicity, marital status, and socioeconomic position (that is, education, labour force status, household equivalised income, household car access, housing tenure, and small area deprivation index).

What is already known on this topic

Few studies have examined the link between exposure to secondhand smoke and mortality

What this study adds

Adults who had never smoked and who lived with smokers had about 15% higher mortality than never smokers living in a smoke-free household

This study strengthens the case for a causal association between secondhand smoke and mortality

as smoking and exposure to secondhand smoke outside the home were more prevalent in New Zealand in the early 1980s. This may explain the apparently stronger association between household exposure and mortality in the 1996-9 cohort compared with the 1981-4 cohort.

The results from this study add to the weight of evidence of harm caused by passive smoking and support steps to reduce exposure to other people's smoke—in the home and in other settings.

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Contributors: SEH conceived the study, analysed the data, and drafted the manuscript. TAB conceived and led the New Zealand census-mortality study (NZCMS) from which data for this study were drawn; advised on study design, data analysis, and interpretation; and contributed to the manuscript. AW and IK advised on the design, analysis, and interpretation of the study and contributed to the manuscript. SEH and TAB will act as joint guarantors for this paper.

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Effectiveness of nicotine patches in relation to genotype in women versus men: randomised controlled trial

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The overall effectiveness of nicotine replacement therapy could be greater if the therapy were targeted at those most likely to respond. Variants of the dopamine D2 receptor (*DRD2* 32806 C/T) have been implicated in the initiation and maintenance of smoking,[1 2] and these variants may also be related to response to nicotine replacement therapy.[3] Additionally, mechanisms of nicotine addiction may differ in men and women.[4] With this evidence in mind, we examined whether the response to nicotine replacement therapy is modified by sex and genotype.

Participants, methods, and results

A randomised controlled trial of nicotine patches in 1991-2 recruited 1686 heavy smokers (≥15 cigarettes a day).[5] The participants wore patches for 12 weeks. Abstinence from smoking was confirmed at one week by expired carbon monoxide concentration ≤10 ppm, and at 12, 24, and 52 weeks by salivary cotinine concentration ≤20 ng/ml (89% of cases) or by expired carbon monoxide concentration ≤10 ppm.

In 1999-2000, we contacted 1532 of the 1625 participants still alive; the mean time from trial to follow up was 8.3 years. In all, 752/1532 (49%) gave a blood sample from which *DRD2* 32806 was successfully typed. Reported abstinence at follow up was confirmed by plasma cotinine concentration ≤20 ng/ml. Throughout, non-respondents were assumed to be smoking.

Participants were older than non-participants (mean age at entry to trial, 43.0 years *v* 41.5 years; P=0.002), more likely to be female (59% (445/752) *v* 53% (410/780); P=0.01), and more likely to have quit for a year in the trial (11% (82) *v* 4% (33), P<0.0001); 744 (99%) reported their racial background as white.

The variant T allele of the dopamine D2 receptor *DRD2* 32806 (CT or TT genotype) was found in 41% (183/445) of women and 41% of men (127/307). Within each sex, there was no difference between the genotype groups in age, number of cigarettes a day, or dependency score.

We measured effectiveness of the patches by the relative odds of abstinence for active and placebo patches over five cumulative time periods: one week, 12 weeks, 24 weeks, 52 weeks, and to follow up. Treatment by genotype and sex, and their interaction, was examined in a full logistic regression model. The three way interaction by genotype by sex was significant for all time periods (P=0.009, P=0.03, P=0.006, P=0.006, P=0.004 respectively), and we therefore analysed the data for men and women separately.

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