infectious and parasitic (1.57, 1.07 to 2.29), genitourinary (1.46, 1.04 to 2.04), circulatory (1.07, 1.01 to 1.12), and external (non-medical) (1.17, 1.00 to 1.57) causes and decreased for deaths attributed to in situ, benign and unspecified neoplasms (0.60, 0.57 to 0.99). There was no clear relation between chemical exposure group and cause-specific mortality. The mortality of each group was lower than that of the general population (SMR 0.88, 0.85 to 0.90; 0.82, 0.80 to 0.84 respectively). 3457 cancers were reported in Porton Down veterans and 3380 in non-Porton Down veterans. While overall cancer morbidity was the same (the RR 1.00, 95% CI 0.95 to 1.05), Porton Down veterans had higher rates of ill-defined malignant neoplasms (1.12, 1.02 to 1.22), in situ neoplasms (1.45, 1.06 to 2.00) and those of uncertain or unknown behaviour (1.52, 1.01 to 1.75).

Conclusions: Mortality was slightly higher in Porton Down than non-Porton Down veterans. With the lack of information on other important factors, such as smoking or service overseas, it is not possible to attribute the small excess mortality to chemical exposures at Porton Down. Overall cancer morbidity in Porton Down veterans was no different from that in non-Porton Down veterans.

Friday 11 September
Parallel session C
Smoking

049 CAN NATIONAL SMOKING PREVALENCE BE MONITORED USING PRIMARY CARE MEDICAL RECORDS DATA?
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Background: Databases of electronic primary care records are widely used for research, but not currently as a source of national statistics on lifestyle issues such as smoking. There has been little contemporary research conducted into the quality of smoking data held within primary care, particularly since the introduction of the Quality and Outcomes Framework. This research is vital to assess the potential for using these large, longitudinal databases to monitor smoking trends.

Objectives: To compare smoking data recorded within The Health Improvement Network database (THIN) with the accepted "gold standard" for measuring smoking prevalence, to investigate the potential of using THIN data to track changes in smoking prevalence.

Methods: For 2000 to 2006, the annual prevalence of current, ex and never-smoking in THIN was determined, taking patients’ most recent smoking-related Read codes for that year as indicative of their smoking status. These figures were compared with the expected prevalence calculated using indirect standardisation based on age, sex and country-specific smoking rates from the corresponding General Household Survey (GHS).

Results: There was generally good agreement between recording of current smoking in THIN and the expected prevalence as predicted using GHS smoking rates. For example, in 2006 the GHS-predicted prevalence of current smoking in the THIN population was 23.4% for men (women 20.7%), with 22.6% of men (19.8% women) actually being recorded as current smokers in their medical records. The recording of ex and never-smoking within THIN was less complete—for men the recorded prevalence of both ex and never smoking was approximately 10 percentage points lower than would be expected using GHS rates, and for women 5 percentage points lower. 17.4% of men and 8.0% of women in THIN in 2006 had no smoking status recorded in their electronic medical records.

Conclusions: These results suggest that primary care medical records within THIN can be used to identify current smokers possibly with enough accuracy for use in monitoring smoking prevalence nationally. However, recording of ex and never-smokers is less complete.

050 THE IMPACT OF IMPLEMENTATION OF SMOKE-FREE LEGISLATION IN ENGLAND ON COTININE LEVELS IN ADULTS
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Objective: To investigate the impact of the implementation on 1st July 2007 of smokefree legislation in England on tobacco smoke exposure and cotinine levels in non-smoking adults.

Design: Cross-sectional survey.


Participants: Nationally-representative sample of 5330 (2585 male) self-reported non-smokers (never or ex-smokers) aged 16+ interviewed in the 2007 Health Survey for England; 3183 cotinine-validated non-smokers aged 16+ (1441 men) with a saliva sample.
Main Outcome Measures: Mean weekly duration of self-reported tobacco smoke exposure; geometric mean salivary cotinine. Cotinine is an excellent marker of exposure to tobacco. Low levels indicate exposure to other people’s smoke; 12 ng/ml is the best cut-off for personal tobacco use. Analyses adjusted for the complex (stratified, clustered) sampling design and weighted for non-response to interview and saliva sample, as appropriate.

Results: Most adult non-smokers reported no-one smoked in the home most days (95% before and 95% after 1st July 2007). Non-smokers’ mean self-reported exposure to tobacco smoke fell from 4.2 hrs (95% CI 3.6 to 4.9) before to 2.0 hrs (1.5 to 2.4) after 1st July in men and from 3.5 hrs (2.9 to 4.1) to 1.4 hrs (1.0 to 1.7) in women (both p<0.001). Exposure was inversely related to age-group but fell most in those with the highest exposure: from 5.9 hrs (4.9 to 6.9) to 2.8 hrs (2.3 to 3.3, p<0.001) aged 16–34 yrs; from 3.4 hrs (2.9 to 4.0) to 1.4 hrs (1.0 to 1.7, p<0.001) aged 55–64 yrs; and 2.1 hrs (1.5 to 2.7) to 0.9 hrs (0.4 to 1.4, p = 0.002) aged 65+. Similar falls occurred in all three NS-SEC groups: professional/managerial from 4.2 hrs (3.5 to 5.0) to 1.7 hrs (1.5 to 2.1); intermediate from 3.9 hrs (2.9 to 4.8) to 1.7 hrs (1.4 to 2.1); and routine/manual from 7.9 hrs (6.7 to 9.1) to 4.9 hrs (4.0 to 5.8) (all p<0.001). Overall, the proportion with undetectable salivary cotinine levels rose from 32% to 46% of cotinine-validated non-smokers. Geometric mean cotinine levels in cotinine-validated non-smoking adults fell from 0.20 ng/ml (95% CI 0.18 to 0.22) in the first half of 2007 to 0.14 ng/ml (0.13 to 0.15) after 1st July 2007 in men and from 0.19 ng/ml (0.17 to 0.21) to 0.12 ng/ml (0.11 to 0.13) respectively in women (both p<0.001). As with self-reported exposure, levels before July 2007 were highest in the youngest age-group, who experienced the largest falls: from 0.23 ng/ml to 0.15 ng/ml aged 16–34 yrs, p<0.001; 0.17 ng/ml to 0.11 ng/ml aged 35–64 yrs, p<0.001; and 0.17 ng/ml to 0.14 ng/ml aged 65+, p = 0.001. Similar, significant falls occurred in all three NS-SEC groups.

Conclusion: The legislation has been successful in its primary aim, to reduce the exposure of non-smokers to tobacco smoke pollution. It has decreased absolute inequalities.

052 WEIGHT CHANGE OVER EIGHT YEARS IN RELATION TO BASELINE BODY MASS INDEX IN A COHORT OF CONTINUING AND QUITTING SMOKERS

Objective: To examine the effect of body mass index (BMI) on weight change over 8 years in a cohort of continuing smokers and a cohort that quit and remained abstinent.

Design: 8 year prospective cohort study.

Data Source: Participants smoking >15 cigarettes daily enrolled in a clinical trial of nicotine patch or placebo in Oxfordshire general practices and were reviewed 8 years later.

Population: 832 male and female participants. Abstainers were 85 participants who were biochemically proven abstinent at 3, 6, 12 months and 8 years. 613 people were smoking at each follow-up, 26 relapsed and 116 quit after 1 year.

Statistical Methods: Means, SDs, and 95% CIs were calculated for change in weight by smoking status. Linear regression analysis, using baseline BMI as an effect modifier, was used to investigate whether the effect of smoking status on weight change was dependent on baseline BMI in smokers and continuous abstainers. Modelling proceeded with separate regression equations for smokers and abstainers. Confounding variables were adjusted for.

Results: Abstainers gained 8.79 kg (SD 6.36, 95% CI 7.42 to 10.17). Smokers gained 2.24 kg (6.65, 95% CI 1.7 to 2.77). Relapsers gained 3.25 kg (7.16, 95% CI 0.32 to 6.24). Later abasters gained 8.53 kg (8.04, 95% CI 6.85 to 9.81). The difference in weight gain (6.56 kg, 95% CI 5.05 to 8.06, p<0.001) between abstainers and smokers was modified by baseline BMI. In abstainers a positive quadratic relationship of BMI fit best, resulting in a J-shaped curve. In persistent smokers there was a negative linear relationship of BMI (p<0.001). The model predicted that abstainers with a baseline BMI of 18 would gain 6 kg, with a BMI of 23 gain 5 kg, and with a BMI of 33 gain 14 kg more than would have been the case had they continued smoking for eight years.

Conclusions: Obese smokers who continue smoking are likely to not change or lose weight over eight years while obese people who quit are likely to gain the most weight. Weight gain is not as harmful as continuing smoking, but weight gain prevention interventions for obese people trying to stop smoking are needed.