SCIENTIFIC RESEARCH GROUP

NEWSLETTER

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1. ENVIRONMENTAL TOBACCO SMOKE

K H Svendsen, L H Kuller, M J Martin, J K Ockene. Effects of passive smoking in the Multiple Risk Factor Intervention Trial. Amer. J. Epidemiol. 126(5): 783-795 (1987).

Male participants in the Multiple Risk Factor Intervention Trial who reported that they had never smoked tobacco products were classified according to the smoking status of their wives. There was no difference in plasma thiocyanate levels between the two groups; however, men whose wives smoked were reported to have higher mean levels of expired carbon monoxide. The authors also claimed lower levels of lung function in that group. The relative risks that were calculated for men whose wives smoked compared with men whose wives did not smoke, were for coronary heart disease death 2.11, for fatal or nonfatal coronary heart disease event 1.48, and for death from any cause 1.96.

R L Repace, A H Lowry. Predicting the lung cancer risk of domestic passive smoking. Amer. Rev. Respir. Dis. 136:1308 (1987).

Some comments on this letter by Peter Lee, an independent consultant, are enclosed. The authors claim that estimates by the American National Academy of Sciences of a 30% increase in risk in lung cancer for non-smokers whose wives smoke are consistent with their own model-based estimates and 'validate' their findings. Peter Lee suggests that the letter is misleading and the estimate of 5,000 death is likely to be 2 or 3 orders of magnitude too high.

P Martin. Passive smoking; A Crawford. Passive smoking. New Zealand Med. J. 100(835): 696-7 (1987).

An exchange of letters between Peter Martin, a respiratory specialist in New Zealand, and Allan Crawford, a consultant to the Australian tobacco industry. Dr Crawford had challenged an article of Dr Martin's about passive smoking; Dr Martin responds by questioning the integrity of consultants to the tobacco industry.

M H Venters, L I Solberg, T E Kottke, M Brekke, T F Pechacek, R H Grimm. Smoking Patterns among social contacts of smokers, ex-smokers and never-smokers: the doctors helping smokers study. Prevent. Med. 16: 626-635 (1987).

Peter Lee's comments also accompany this paper, in which US estimates of concordance (i.e. the tendency for smokers to marry smokers) are provided. Estimates of concordance of smoking habits between social contacts are also given, which may be important for studies based on exposure to ETS in social situations.

M J Jarvis, H Tunstall-Pedoe, C Feyerbend, C Vesey, Y Salojee. Comparison of tests used to distinguish smokers from non-smokers. Amer. J. Pub. Health 77:1435-1438 (1987).

The authors discuss purported biochemical markers of smoke exposure and their use to distinguish between smokers and non-smokers in a group of hospital outpatients. Comments by Peter Lee are provided. Interestingly, of 121 patients reporting to be non-smokers, 21 (17%) had plasma cotinine above a level considered incompatible with non-smoking status.

2. THEORIES OF ADDICTION, DEPENDENCE ETC.

H C Porchet, N L Benowitz, L B Sheiner. Pharmacodynamic model of tolerance: application to nicotine. J. Pharmacol. Exp. Ther. 244(1): 231-236 (1988).

The authors investigated the development of tolerance to nicotine following repeated infusions, and then examined the regaining of sensitivity to the substance. They proposed a joint pharmacokinetic - pharmacodynamic theory of tolerance which they tested by taking blood samples as a pharmacokinetic measure and heart rate as a pharmacodynamic measure. They suggested that tolerance to nicotine could not be explained purely on a pharmacokinetic level; however, since the studies were carried out in vivo in humans it was not possible to take a direct measure of pharmacodynamic tolerance e.g. receptor number and down-regulation. This part of the paper is therefore highly speculative and does not advance any further the knowledge of mechanisms underlying pharmacodynamic tolerance. It is suggested that, for nicotine, a half-life of tolerance development and regression is 35 min., indicating to the authors that 4-5 half-lives (approx. 3 hours) after smoking a cigarette, nearly full sensitivity should have been gained. The authors suggest that the interval at which smokers smoke cigarettes may be determined by the kinetics of regression of tolerance.

T W Lombardo, J R Hughes, J D Fross. Failure to support the validity of the Fagerstrom tolerance questionnaire as a measure of physiological tolerance to nicotine. Addictive Behaviours 13(1): 87-90 (1988).

The Fagerstrom Tolerance Questionnaire was developed with the stated aim of measuring "dependence" on nicotine among smokers. There has been some controversy in the scientific literature as to whether this questionnaire does indeed measure what it claims to measure. In the present study it is indicated that the scale does not predict tolerance, and previous studies suggested that it did not measure physical dependence. The authors therefore claim that the scale in fact measures "behavioural dependence" (i.e. persistence of the smoking behaviour) rather than physical dependence.

3. SMOKING, OESTROGEN AND ENDOMETRIAL CANCER

4. SMOKING AND ALZHEIMER'S/PARKINSON'S DISEASE

H Sershen, A Hashim, A Laitha. Behavioural and biochemical effects of nicotine in an MPTP- induced mouse model of Parkinson's Disease. Pharmacol. Biochem. Behav. 28:299-303 (1987).

MPTP is a substance that has been shown to produce deficits in pathways that contain the neurotransmitter dopamine in a brain area called the nigrostriatum in humans, other primates and mice. It has been suggested that is is a useful model of Parkinson's Disease because the damage and symptoms produced by the compound are very similar. The present study was aimed at testing whether nicotine had a therapeutic effect in such a model. However since their model was based on the symptom of reduced locomotor activity produced by MPTP, some confounding could have occurred due to the intrinsic locomotor stimulant effect of nicotine which could explain its ability to bring activity levels in MPTP-treated mice back to control levels. This could happen with any other drug with intrinsic stimulatory properties on locomotor activity, and would thus be no indication of therapeutic effect. No other compounds were used as a control for this. In this case, therefore the animal model selected was inappropriate to the compounds used and no adequate control was provided to aid interpretation. The authors acknowledge to US Council for Tobacco Research for support for the work.

5. HEART DISEASE

6. RESPIRATORY/LUNG DISEASE

D Trichopoulos, A Hatzakis, E Wynder, K Katsouyanni, A Kalandidi. Time trends of tobacco smoking, air pollution and lung cancer in Athens. Environ. Res. 44: 169-178 (1987).

The study was aimed at evaluating whether air pollution had affected lung cancer incidence in Athens. The lung mortality figures between Athens and the rest of Greece were compared after standardisation for tobacco effects and any differences were assumed to be due only to air pollution. This may or may not be a valid assumption. The authors cliam that although a strong effect of tobacco smoking on lung cancer mortality is visible, this is not so for air pollution.

7. SMOKING AND MENTAL ILLNESS

8. MOLECULAR BIOLOGY

L Andreasson, G Bjorlin, M Hocherman, R Korsgaard, E Trell. Laryngeal cancer, aryl hydrocarbon hydroxylase inductibility and smoking. ORL 49:187-192 (1987).

The authors note that the tendency of respiratory tract cancer to aggregate in families has been tentatively explained by studies on the ability to induce the enzyme aryl hydrocarbon hydroxylase (AHH). The function of AHH has been suggested to be to convert polycyclic aromatic hydrocarbons into a carcinogenic form which is able to bind to DNA, thus presumably altering genetic information. It has been suggested in man that genetically-determined differences in AHH inducibility exist between individuals. The authors suggest that in 58 cases with laryngeal cancer (56 of whom were smokers) there was a high representation of patients with a high level of AHH. They claim that smokers with a high AHH level run a fourfold risk of developing laryngeal cancer as compared to non-smokers with low AHH levels. However, there is no evidence to suggest that smokers in general have higher AHH levels than the general population.

9. COMPONENTS OF CIGARETTE SMOKE

10. SMOKING AND REPRODUCTION/PREGNANCY

T R Martin, M B Bracken. The association between low birth weight and caffeine consumption during pregnancy. Amer. J. Epidemiol. 126:873-821 (1987).

In addition to studies claiming that smoking or even passive smoking during pregnancy may lead to low birth weight, many other studies have suggested that similar effects can be measured for consumption of alcohol and caffeine. In the present study it is suggested that even 1-150 mg caffeine daily (e.g. up to 1.5 cups of coffee daily) could increase the relative risk for low birthweight by 1.4, and 4.6 for over 300 mg (3 cups of coffee daily). The authors measured decreases in mean birth weight at 6-105g.

A D McDonald, J C McDonald, B Armstrong, N Cherry, C Delorme, A D Nolin, D Robert. Occupation and pregnancy outcome. Br. J. Ind. Med. 44:521-526 (1987).

A large sample of pregnant women attending a Montreal hospital were interviewed about occupational, social and personal characteristics and followed up for pregnancy outcome. The authors report that statistically significant excesses of spontaneous abortion were observed in nursing aides, saleswomen, and women in food and beverage service. Stillbirth was associated with agriculture and horticulture, leatherwork and certain sales occupations; congenital defects were found more frequently in women in childcare, service occupations and manufacture of metal and electrical goods; and low birth weight was observed more frequently in chambermaids, cleaners, janitors and women employed in the manufacture of food, drink, metal and electrical goods, and clothing.

11. GASTROENTEROLOGY

12. PHARMACOLOGY/NICOTINE

13. SMOKING AND BODY WEIGHT

14. MISCELLANEOUS

D LeGrady et al. Coffee consumption and mortality in the Chicago Western Electric Company Study. Amer. J. Epidemiol. 126:803-812 (1987).

The authors examined the relationship between coffee consumption and 19-year mortality from all causes, from coronary heart disease, and from non-coronary causes in 1,910 white males aged 40-56 years in 1957-1958. Mortality from all causes was greatest in both the lowest coffee intake group (0-1 cups daily) and the highest group (6+ cups daily). The increased mortality in the 6+ cups group was claimed to be due to an excess risk of coronary heart disease, while in the lower group it was claimed to be due to non-coronary causes (non-coronary heart disease and cancer). The increased risk of coronary heart disease in the high coffee consumption group was found both in smokers and non-smokers.

15. OTHER CANCERS

16. BLOOD COMPONENTS