Smoking, type 2 diabetes and metabolic syndrome

Two of the articles in this issue of *Diabetes & Metabolism* are cross-sectional epidemiological studies which evoke the projected epidemic of type 2 diabetes [1]. They study two potential risk factors for diabetes, namely obesity and smoking [2, 3].

The relation between cigarette smoking and the presence of diabetes was studied in almost 30 000 consultants from the French IRSA Health Examination Centres [3]. Diabetes, as defined by hypoglycaemic treatment and/or a fasting plasma glucose \( \geq 7.0 \text{ mmol/l} \) was present in 437 (3.5%) of the 12 427 men and 263 (1.8%) of the 15 360 women diabetic.

Already there are number of prospective studies (the authors cite 8 articles) which have shown that smoking, particularly heavy smoking precedes incident diabetes, not only in Asiatic populations, but also in European and American populations. I would hazard to say, in contrast to the authors, that the populations in all but two of these studies are not so genetically different to a French population, and that any differences would be due to lifestyle.

Current smoking was associated with diabetes in men with an odds ratio of 1.5 (95% CI: 1.1-2.0) after adjustment for factors associated with diabetes, notably age, BMI, WHR and regular alcohol consumption. For women, the relation was not significant, with an odds ratio 0.9 (0.5-1.4). No results have been shown to test whether there is a significant difference between these odds ratios for men and women and it is possible that statistically, there is no difference.

In contrast, for former smokers, the odds ratios for the presence of diabetes are very similar in the two sexes, 1.3 (1.0-1.7) for the men and 1.5 (0.9-2.3) for the women.

This study thus confirms that smoking and diabetes are associated, but only in men. By design this study is not able to show the temporal nature of the relation. Further, as other studies tend to show that the incidence of diabetes is higher in heavy smokers, a stronger relation may have been found if the number of cigarettes smoked per day had also been studied. More evidence for a causal effect could have been obtained if glycaemic states (nормo-glycaemia, impaired fasting glucose, screened diabetes, drug-treated diabetes) had been studied.

The authors note four studies that have shown that insulin sensitivity is reduced in smokers, however two of the studies are experiments of the acute effects of smoking. One of the other two studies is a carefully analysed epidemiological study of non-diabetic men; insulin was indeed higher in the current and former smokers than in those who had never smoked [4]. In contrast, a study from French Health Examination Centres which included the IRSA centres, and recently published in *Diabetes & Metabolism* [5] showed that male smokers and non-smokers had equivalent insulin levels, whereas women who smoked had a significantly lower mean level of insulin than those who did not, despite equivalent BMI. More light on this relation is shed by the recent prospective analysis of incident hyperinsulinaemia in the Atherosclerosis Risk in Communities Study study [6], where starting to smoke was a risk factor for hyperinsulinaemia.

Prevention of smoking will not only reduce morbidity and mortality of cardiovascular diseases and cancer, but perhaps also the incidence of diabetes. Further a large American study [7], with 20 times more subjects than the present study, was able to show that quitting smoking had a beneficial effect: 10 years after men had stopped smoking the incidence returned to that of those who had never smoked, 5 years for women.

The second article comes from the French SU.VI.MAX population [2] where a simple (or even simplistic) questionnaire provided information about regular physical activity (as a binary variable) as well as about age, smoking (current, ex- or never) and educational level (primary, secondary, tertiary) and their relation with obesity (BMI \( \geq 30 \text{ kg/m}^2 \)) and abdominal adiposity, as defined by waist hip ratio (WHR) and waist circumference with thresholds used in the literature.

As expected in a population of middle aged individuals, obesity and central obesity increased with age. A higher education had a beneficial effect on obesity. It was also negatively associated with central obesity, but only as defined by the WHR and only in men.

Smoking is potentially modifiable. The relations between smoking and obesity were all positive but their significance differed according to sex. Further they differed according to whether obesity or central obesity (after adjusting for BMI) was being studied and then whether it was WHR or waist circumference. In general, smokers were more likely to be obese and have a central adiposity. However, former male smokers were more obese than smokers. Men and women who were current smokers had a higher WHR than non-smokers, but former smokers had a similar WHR to those who had never smoked. For waist circumference, smoking appear to have no effect. These results go against the usual notion that smoking is associated with a lower
BMI, which is often used as an argument in smokers, especially women, for continuing to smoke.

Regular physical activity was negatively associated with obesity and with abdominal obesity as characterised by the WHR but for the waist, this was only the case for women. Again this is may be due to the thresholds used, and it would be of interest to study the relation between the distributions of BMI, WHR and waist circumference in the active and the inactive to see if the former relation was due to the chosen thresholds.

Again prospective studies are required before any conclusions can be reached as to their causative nature. However, prevention of and stopping smoking and increasing physical activity can only be beneficial — with a possible lowering of the incidence of both diabetes and obesity.

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References


