Tobacco smoking and bladder cancer in coffee non-drinkers

Data from a large Italian case-control study confirm that the tobacco-related relative risk of bladder cancer is higher in coffee non-drinkers than in coffee drinkers. However, given the correlation between tobacco and coffee, the population attributable risk was similar for coffee drinkers and non-drinkers. Lopez-Abente and Escolar, using data from a large Italian case-control study, suggested that the association between tobacco smoking and bladder cancer may be stronger among coffee non-drinkers than among usual coffee drinkers. This potential favourable interaction has some biological plausibility, and can be related to the inhibition of enzymes involved in the activation of carcinogenic aromatic amines, including cytochrome P450IA2.

To provide further information on the issue, we analysed data from a case-control study conducted in Italy between 1985 and 1992, whose main results on tobacco and coffee have been published elsewhere. Briefly, a total of 727 histologically confirmed, incident cases of bladder cancer (617 men, 110 women), aged 27–79 years (median age 63), were included in the study. They were recruited in the National Cancer Institute and in a network of other major general hospitals and university clinics in greater Milan and in Pordenone (northern east Italy). Controls were 1067 subjects (769 men, 298 women), aged 25–79 years (median age 60), admitted to the same network of hospitals of cases for acute, non-neoplastic, non-urological or genital tract diseases (23% were admitted for surgical conditions, 29% for fractures and traumatic conditions, 18% for non-traumatic orthopaedic disorders, and 30% for miscellaneous other illnesses). Less than 3% of subjects approached (cases and controls) refused the interview.

Data were collected by trained interviewers using a structured questionnaire, including information on sociodemographic factors, personal characteristics and lifestyle habits, history of selected diseases and of relevant occupational exposures. Questions on coffee drinking included average cups drunk per day and duration of use; questions on tobacco included smoking status (never smoker, ex-smoker or current smoker), average quantity smoked per day, and duration of the habit. Information on tobacco and coffee was satisfactorily reliable. We estimated the odds ratios (OR) and their 95% confidence intervals (CI) using unconditional multiple logistic regression, including terms for age, sex and study centre. Further adjustment for education and occupation did not materially change any of the results.

Table 1 shows the distribution of cases and controls, the corresponding OR and 95% CI according to tobacco smoking in strata of coffee drinking. Among cases, ever smokers were 84% among coffee drinkers and 78% among non-drinkers. Corresponding fractions among controls were 65% and 53%. Compared with never smokers, the OR for current smokers was 3.2 (95%CI 2.3 to 4.4) for coffee drinkers and 3.3 (95%CI 2.5 to 11.4) for non-drinkers. The test for heterogeneity was not significant. The relative risks for current smokers increased at higher levels of cigarettes per day in both coffee drinkers (OR=2.7 for <15, OR=3.8 for ≥15) and non-drinkers (OR=2.4 for <15 and 5.5 respectively). Trends in risk were observed in both strata also for duration of the habit, the OR being 3.6 (95%CI 2.6 to 5.1) in coffee drinkers and 6.1 (95%CI 2.8 to 13.6) in non-drinkers for smoking duration >30 years. When the analysis was restricted to male cases and 769 male controls, the OR for current smokers was 3.5 (95%CI 2.4 to 5.2) among coffee drinkers and 4.2 (95%CI 1.7 to 10.4) among coffee non-drinkers.

In conclusion, our study confirms that cigarette smoking is a risk factor for bladder cancer both in coffee drinkers and non-drinkers, and provides some support for the observation that coffee may attenuate the relative risk deriving from tobacco. Still, the population attributable risks were similar in coffee drinkers and non-drinkers. In fact, as coffee and tobacco consumption were positively correlated, risk fractions of bladder cancer attributable to smoking in this Italian population were 56% (95%CI 36% to 77%) among coffee non-drinkers and 53% (95%CI 43% to 64%) among coffee drinkers, confirming the prominent effect of smoking on bladder carcinogenesis in both coffee drinkers and non-drinkers.

Acknowledgments

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Table 1 Distribution of 727 cases of bladder cancer and 1067 controls according to smoking habits, in coffee and non-coffee drinkers, and odds ratios* (OR) and corresponding 95% confidence intervals (CI), Italy, 1985–1992

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coffee drinkers</th>
<th>Non-coffee drinkers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Controls</td>
</tr>
<tr>
<td>Tobacco</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smokers</td>
<td>101</td>
<td>323</td>
</tr>
<tr>
<td>Ex-smokers</td>
<td>215</td>
<td>267</td>
</tr>
<tr>
<td>Current smokers</td>
<td>322</td>
<td>324</td>
</tr>
<tr>
<td>Cigarettes/day (current smokers only)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;15</td>
<td>93</td>
<td>121</td>
</tr>
<tr>
<td>≥15</td>
<td>228</td>
<td>202</td>
</tr>
<tr>
<td>χ², trend</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Duration of smoking (y)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;15</td>
<td>28</td>
<td>76</td>
</tr>
<tr>
<td>16–30</td>
<td>141</td>
<td>207</td>
</tr>
<tr>
<td>&gt;30</td>
<td>350</td>
<td>288</td>
</tr>
<tr>
<td>χ², trend</td>
<td>p&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

*Estimates from multiple logistic regression including terms for quinquennia of age, study centre and sex. †Reference category.
Newcastle Heart Project

In our recently published analysis of Newcastle Heart Project data we concluded that in South Asians diabetes or impaired glucose tolerance was associated with an increased likelihood of reporting sibling but not parental diabetes. In contrast, in Europeans diabetes or impaired glucose tolerance were associated with both sibling and parental diabetes. In response to feedback, we reanalysed our data to assess whether awareness of a diagnosis of diabetes influenced reporting and to reconsider the possible influence of our direct standardisation procedure.

In brief, the Newcastle Heart Project studied an age and sex stratified population sample of 684 South Asians (139 Indian, 369 Pakistani, 120 Bangladeshi) and 825 European men and women aged 25–74 years in Newcastle upon Tyne, UK. Diabetes was defined as a plasma glucose sample >7.8 mmol/l, following World Health Organisation (1985) criteria. Respondents prescribed oral hypoglycaemic agents or insulin were classified as having diabetes. Full details of the study have been published. Because of the small numbers clear cut patterns did not emerge from age specific data. We were therefore reliant on age standardised analysis. Table 1 shows typical results. In Europeans the age/sex adjusted prevalence of parental diabetes rose across the categories of normal glucose tolerance, impaired glucose tolerance and previously undiagnosed diabetes (p=0.017 for linear trend across these categories). Parental diabetes was more frequent in those already known to have diabetes than those with newly diagnosed diabetes. In South Asians the equivalent associations were less clear cut. There was no appreciable difference in reporting between those with newly diagnosed or previously known diabetes.

In Europe the prevalence of sibling diabetes showed a strong positive relation with respondent diabetes tolerance (p<0.001 for linear trend) There was little difference in the prevalence of sibling diabetes between those with newly diagnosed and known diabetes. Once again, in South Asians the equivalent associations were less clear cut. Those with known diabetes were more likely than those with newly diagnosed diabetes to report diabetes in their siblings.

Even after excluding respondents with known diabetes, we found that among South Asians there was a clear relation between impaired glucose tolerance and diabetes in respondents and reported diabetes in parents and siblings. The relation with parental and sibling diabetes in South Asians was weaker. Exclusion of those with known diabetes also weakened the previously reported association with diabetes in South Asian siblings, raising the possibility that awareness of a diagnosis of diabetes may influence reporting of family histories of diabetes both in Asians and in Europeans.

This study shares with the majority of previous reports the weakness that it is dependent on respondent reports rather than biochemical testing for family diagnoses of diabetes. It is also limited where there are ethnic differences in family reporting. The relatively large number of missing responses (which may reflect uncertainty about family history) also urge caution in interpreting these results.

With these caveats, we conclude that familial clustering of diabetes is more clear cut in Europeans than in South Asians. Larger studies with biochemically verified information about parental and sibling diabetes may help to elucidate the causal basis of ethnic variations in diabetes.

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Table 1 Diabetes in parents and siblings, by respondent glucose tolerance status

<table>
<thead>
<tr>
<th>Diabetes in Parents</th>
<th>Normal</th>
<th>IGT</th>
<th>New diabetes</th>
<th>Known diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>European Total and % don’t know/missing</td>
<td>649 (27)</td>
<td>129 (29)</td>
<td>27 (44)</td>
<td>19 (16)</td>
</tr>
<tr>
<td>Parental diabetes % (95% CI)*</td>
<td>8.2 (6.0 to 11.1)</td>
<td>11.7 (6.5 to 20.3)</td>
<td>27.9 (10.6 to 56.0)</td>
<td>42.9 (21.3 to 67.6)</td>
</tr>
<tr>
<td>OR (95% CI) for parental diabetes*</td>
<td>1.00</td>
<td>1.5 (0.7 to 3.1)</td>
<td>4.4 (1.3 to 15.1)</td>
<td>8.5 (2.9 to 24.8)</td>
</tr>
<tr>
<td>South Asian Total and % don’t know/missing</td>
<td>386 (14)</td>
<td>140 (14)</td>
<td>56 (11)</td>
<td>102 (23)</td>
</tr>
<tr>
<td>Parental diabetes % (95% CI)*</td>
<td>17.9 (13.8 to 22.9)</td>
<td>28.7 (20.8 to 38.2)</td>
<td>25.6 (14.5 to 41.1)</td>
<td>26.3 (16.8 to 38.7)</td>
</tr>
<tr>
<td>OR (95% CI) for parental diabetes*</td>
<td>1.00</td>
<td>1.8 (1.1 to 3.1)</td>
<td>1.6 (0.7 to 3.4)</td>
<td>1.6 (0.9 to 3.2)</td>
</tr>
<tr>
<td>Sibling diabetes European Total and % don’t know/missing</td>
<td>563 (19)</td>
<td>109 (28)</td>
<td>25 (28)</td>
<td>15 (9)</td>
</tr>
<tr>
<td>Diabetes in siblings % (95% CI)*</td>
<td>3.5 (2.1 to 5.7)</td>
<td>6.6 (2.9 to 14.1)</td>
<td>25.1 (10.3 to 49.5)</td>
<td>27.7 (11.0 to 54.3)</td>
</tr>
<tr>
<td>OR (95% CI) for sibling diabetes*</td>
<td>1.00</td>
<td>1.9 (0.8 to 4.9)</td>
<td>9.2 (2.9 to 28.7)</td>
<td>10.5 (3.1 to 35.2)</td>
</tr>
<tr>
<td>South Asian Total and % don’t know/missing</td>
<td>372 (10)</td>
<td>130 (7)</td>
<td>53 (4)</td>
<td>97 (13)</td>
</tr>
<tr>
<td>Diabetes in siblings % (95% CI)*</td>
<td>9.8 (7.0 to 13.6)</td>
<td>15.0 (9.6 to 22.5)</td>
<td>16.4 (8.7 to 28.7)</td>
<td>28.3 (19.4 to 39.3)</td>
</tr>
<tr>
<td>OR (95% CI) for sibling diabetes*</td>
<td>1.00</td>
<td>1.6 (0.9 to 3.0)</td>
<td>1.8 (0.8 to 4.0)</td>
<td>3.6 (2.0 to 6.7)</td>
</tr>
</tbody>
</table>

*Adjusted for sex and age in five categories.

References


BOOK REVIEWS

Disease, knowledge and society


The purpose of this book is to provide critical analyses of disease, treatment and care as socially structured practices. The authors are Danish and Norwegian social scientists and sociologists—and occasional historians, psychologists and biologists—who belong to Disease and Society Network. The book consists of papers presented at the seminars of this network.

The papers have a wide scope, ranging from highly theoretical (for example, social construction of diagnosis, philosophy of science critique of the randomised clinical trial) to highly empirical approaches (for example, user participation in employees’ strain disorders intervention programme, observations on the role of food in hospitals). They also cover a wide variety of disciplines ranging from a historical overview of public health impacts of medicine to psychoneuroimmunology, which studies the relations between behaviour, the hormone system, the nervous system and the immune system in the human body. One of the starting points is the medical science’s inability to deal with patients instead of diseases. Critique of the randomised clinical trial as the gold standard in medical science is also one of the key issues. Somewhat surprisingly the book does
not take up such topics as health inequalities, empowerment or disease prevention/health promotion.

I found Hans Wadskjær’s analysis of the modern institutionalised medicine as a dise-ase system particularly interesting. The suc-cess of this system in society, its self referent nature and the power, prestige and status associated with it has led it to cooperate with another powerful system, the economic sys-tem in order to avoid the constraints put upon it by the political system. Even though there are exceptions to the rule, modern medicine seems to be engaged in providing incredibly sophisticated services to small, mainly affluent groups. The impact this development in the society should be of interest both for health researchers and social scientists. It is in this discourse this book finds its place.

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Perspectives on health inequity

The book presents the views of experts of different backgrounds on causes and solutions of health inequity. These views are given as a reaction on data on health inequalities using the 1994 New South Wales Health Promotion Survey (New South Wales is a large region in the south east of Australia, including Sydney). The presented survey data focus on health differences by sociodemographic char-acteristics, and by education, employment status and a not further specified socioeco-nomic classification based on usual resi-dence. No attention was paid to differences by income levels or by the often used manual/non-manual work distinction. The presented rates were corrected for age differ-ences but unfortunately not for differences by gender. It is clear that there are health equalities. Health inequity is not the same as health inequality. Equity, as is said in the book, describes differences that are unfair and unjust, which are largely beyond the individual’s control and that are avoidable. The presented data give, however, not insight in the proportion that is “unfair and unjust, beyond the individuals control and that are avoidable”.

The perspectives from the point of view of health promotion, behavioural epidemiology, general practitioners, health economics, sociology, public policy and the commentar-ies of representatives from a governmental organisation, the university, the social service council and a minority commission give a nice overview how we can think about health equalities. Perspectives from the point of view of ethics could have made it more complete.

Intervention strategies could include (1) strengthening individuals in disadvantaged circumstances, (2) strengthening disadvan-taged communities, (3) improving access to essential facilities and services and (4) en-couraging macro-economic and cultural change. But, it is clear that we are still at the beginning of a very long road to address health inequities.

The book can help students, public health workers and (junior) researchers to begin ning to understand the complex field on health equalities and inequities.

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