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Coffee Consumption and Bladder Cancer Risk

Barbara D'Avanzo, Carlo La Vecchia, Silvia Franceschi, Eva Negri, Renato Talamini and Isabella Buttino

The relation between consumption of regular and decaffeinated coffee and other methylxanthine-containing beverages and bladder cancer was analysed in a case-control study in two different areas of northern Italy (555 cases and 855 controls). The multivariate relative risk (RR) adjusted for smoking, occupation and sociodemographic variables for coffee drinkers versus non-drinkers was 1.3 (95% CI 1.0–1.8). The RR was 1.2 for one cup of coffee per day, 1.4 for two, 1.5 for three and 1.4 for four or more ($P = 0.05$). RRs for current drinkers were 1.5 (0.9–2.4) for decaffeinated coffee, 0.9 (0.6–1.2) for tea, and 0.6 (0.3–1.4) for cola. With reference to duration of consumption of coffee, RRs were 1.2 for less than 30 years or 1.4 for 30 years or more. Coffee-related RRs were higher in the older age group and in ex-smokers. Among 105 cases and 338 controls who had never smoked, RRs were 1.9 for one or two cups per day, 1.8 for three and 1.5 for four or more (trend not significant). A higher prevalence of coffee drinking among bladder cancer cases than controls was confirmed, with no clear dose–risk relation.
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INTRODUCTION

OVER THE past 2 decades, more than 30 case–control studies of coffee and bladder cancer have been published, but the issue is still open to debate [1]. Compared with non-users of coffee, in fact, the relative risks (RRs) tend to be elevated in drinkers, but such increase is not consistently related to dose or duration. This suggests that the apparent association, although present in various studies, is possibly not causal, but at least partially due to some residual confounding. Smoking is the most likely confounding factor, but the differences between crude and adjusted RRs vary from one study to another, and misclassification of smoking or insufficient adjustment for it are unlikely to explain the results from all the studies. Internal inconsistencies have also emerged in some studies, especially as regards potentially different effects of coffee according to sex [2–4] and to smoking habits [5].

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Epidemiological data are much more scanty for tea and cola-containing beverages, and further information on them would be important both for the widespread use of these beverages worldwide, and the additional information they could provide on methylxanthines, which are the major common constituent.

To further investigate this issue we present here the data from a study conducted in two different areas of northern Italy. Interest of this report may lie in providing additional information useful in order to better understand the potential modifying effect or interaction of smoking or other covariates on the relationship between coffee and bladder cancer.

SUBJECTS AND METHODS

Since 1985 we have been conducting a case–control study of urological cancers in the greater Milan area and in the province of Pordenone, north-eastern Italy. The general design of this investigation has been described elsewhere [6].

Cases and controls were directly interviewed by specially trained interviewers; less than 3% of subjects approached (cases and controls) refused to answer. The structured questionnaire included a series of socio-demographic questions, detailed information on smoking habits, frequency of consumption of alcoholic beverages and of a few selected foods, history of

Table 1. Distribution of 555 cases of bladder cancer and 855 controls according to age, sex, education, smoking habits and alcoholic beverage consumption. Italy, 1985–1990

	Milan		Pordenone	
	Cases	Controls	Cases	Controls
Sex				
Males	302(83.0)	336(75.3)	160(83.8)	259(63.5)
Females	62(17.0)	111(24.7)	31(16.2)	149(36.5)
Age group				
<55	51(14.0)	120(26.8)	29(15.2)	141(34.6)
55–64	155(42.6)	176(39.4)	63(33.0)	122(30.3)
65–74	158(43.4)	151(33.8)	99(51.8)	145(35.5)
Education*				
≤6	211(58.0)	255(57.0)	134(70.2)	305(74.8)
7–11	101(27.7)	105(23.5)	38(19.9)	77(18.9)
≥12	52(14.3)	85(19.0)	19 (9.9)	26 (6.4)
Smoking habits*				
Never smokers	80(22.0)	181(40.5)	25(13.1)	157(38.5)
Ex-smokers	97(26.6)	114(25.5)	72(37.7)	124(30.4)
Current smokers	187(51.4)	152(34.5)	93(47.7)	127(31.1)
<15 cig/day	50(13.7)	48(10.7)	30(15.7)	54(13.2)
15–24	100(27.5)	71(15.9)	50(26.2)	53(13.0)
≥25	37(10.2)	33 (7.4)	13 (6.8)	20 (4.9)
Wine (glasses/day)				
<2	79(21.7)	113(25.3)	16 (8.4)	34 (8.3)
2 < 4	38(10.4)	65(14.5)	16 (8.4)	49(12.0)
4 < 6	109(29.9)	128(28.6)	50(26.2)	108(26.5)
≥6	138(37.9)	141(31.5)	109(57.1)	217(53.2)
Beer				
No	342(94.0)	404(90.4)	123(64.4)	265(65.0)
Yes	22 (6.0)	43 (9.6)	68(35.6)	143(35.0)
Spirits				
No	312(85.7)	378(84.6)	95(49.7)	234(57.4)
Yes	52(14.3)	69(15.4)	96(50.3)	174(42.6)

*The sum of the strata does not add up to the total because of some missing values. †Values are expressed as no. (%).

selected diseases, family history for cancers of the urinary tract, reproductive and sexual habits, history of relevant occupational exposures and modalities of diagnosis, and specific information on consumption of coffee, tea and cola-containing beverages including the number of drinks per day and the total duration (in years) of the habit.

Cases

A total of 555 cases of bladder cancer aged 27–74 years (median age 63) were enrolled in the study. Of these, 364, aged 28–74 (303 males and 61 females), were recruited in the National Cancer Institute and a network including other major general hospitals and university clinics in the greater Milan area; 191 (160 males and 31 females) were recruited in the Pordenone Cancer Institute and General Hospital. Diagnosis of bladder cancer had to be assessed no more than one year before the interview and had to be histologically confirmed as invasive bladder cancer.

Controls

Controls were 855 subjects aged 27–74 years (median age 60) admitted to the same network of hospitals where cases were identified. In the Milan dataset there were 447 controls (336 males and 111 females). They were admitted for a wide spectrum of acute, non-neoplastic, non-urological or genital tract diseases (29% fractures and other traumatic conditions; 17% non-traumatic orthopaedic conditions; 13% medical disorders; 7% surgical conditions and 34% miscellaneous illnesses including skin, nose and throat conditions). The controls interviewed in Pordenone were 408 patients aged 32–78 (259 males and 149 females) selected according to similar criteria (17% fractures, traumas, contusions or dislocations; 25% non-traumatic orthopaedic conditions; 11% medical disorders; 16% surgical conditions; 14% skin conditions and 17% other miscellaneous illnesses).

Table 2. Distribution of 555 cases of bladder cancer and 855 controls according to methylxanthine-containing beverage consumption. Italy, 1985–1990

	Milan		Pordenone		All	
	Cases	Controls	Cases	Controls	Cases	Controls
Coffee (cups/day)						
0	53(14.6)	92(20.6)	18 (9.4)	43(10.5)	71(12.8)	135(15.8)
1	79(21.7)	110(24.6)	47(24.6)	102(25.0)	126(22.7)	212(24.8)
2	112(30.8)	115(25.7)	55(28.8)	134(32.8)	167(30.1)	249(29.1)
3	71(19.5)	73(16.3)	38(19.9)	76(18.6)	109(19.6)	149(17.4)
≥4	49(13.5)	57(12.8)	33(17.3)	53(13.0)	82(14.8)	110(12.9)
Duration* (years)						
<30	175(48.1)	220(49.2)	44(23.0)	178(43.6)	219(39.5)	398(46.5)
≥30	136(37.4)	129(28.9)	129(67.5)	187(45.8)	267(47.7)	316(37.0)
Decaffeinated coffee						
No	340(93.4)	426(95.3)	176(92.1)	387(94.9)	516(93.0)	813(95.1)
Yes	24 (6.6)	21 (4.7)	15 (7.9)	21 (5.1)	39 (7.0)	42 (4.9)
Tea						
No	323(88.7)	379(84.8)	166(86.9)	353(86.5)	489(88.1)	732(85.6)
Yes	41(11.3)	68(15.2)	25(13.1)	55(13.5)	66(11.9)	123(14.4)
Cola						
No	358(98.4)	436(97.5)	188(98.4)	395(96.8)	546(98.4)	831(97.2)
Yes	6 (1.6)	11 (2.5)	3 (1.6)	13 (3.2)	9 (1.6)	24 (2.8)

*The sum of the strata does not add up to the total because of some missing values.
Values are expressed as no. (%)

Data analysis and control for confounding

RRs of bladder cancer and their approximate 95% confidence intervals (CI), according to various measures of coffee consumption and other methylxanthine-containing beverages, were obtained first from data stratified for sex and decade of age (plus, when required and centre) using the Mantel–Haenszel procedure [7]; tests for linear trend in proportions were computed according to the Mantel test [8]. Unconditional multiple logistic regression was used in order to account simultaneously for the effect of a number of major identified potential confounders [9]. Included in the regression equation were terms for age, sex, educational level, smoking (never smokers, ex-smokers since 9 years or less, ex-smokers since 10 years or more, current smokers of less than 15, 15 to 24 and 25 or more cigarettes per day), alcohol drinking (in tertiles) and occupation (previously defined as related to bladder cancer).

RESULTS

Distributions of cases and controls according to sex, age, level of education, tobacco and alcohol consumption are shown in Table 1, divided for the two study centres of Milan and Pordenone. Cases were, in general, less educated than controls and more frequently smokers (the RR for current smokers compared to never smokers adjusted for sex, age and centre was 3.1, 95% CI 2.3–4.2). With regard to alcoholic beverages, although levels were substantially higher in Pordenone than in Milan, no significant difference was observed between cases and controls.

Table 2 shows the distribution of consumption of methylxanthine-containing beverages. Overall 13% of cases were non-coffee drinkers as compared with 16% of controls. Most cases and controls consumed between one and three cups of coffee per day, and only 15% of cases and 13% of controls consumed

Table 3. Relative risks (and 95% confidence intervals) of 555 cases of bladder cancer and 855 controls according to level and duration of regular coffee consumption, decaffeinated coffee, tea and cola. Italy 1985–1990

	Milan		Pordenone		All	
	M-H*	MLR†	M-H*	MLR†	M-H*	MLR†
Coffee						
Non-drinkers	1‡	1‡	1‡	1‡	1‡	1‡
Drinkers	1.6	1.4	1.2	1.1	1.4	1.3
	(1.1–2.3)	(1.0–2.1)	(0.7–2.2)	(0.6–2.0)	(1.0–1.8)	(1.0–1.8)
Cups/day						
1	1.2	1.2	1.2	1.1	1.1	1.2
	(0.7–1.8)	(0.8–1.9)	(0.6–2.2)	(0.5–2.1)	(0.8–1.6)	(0.8–1.7)
2	1.8	1.7	1.1	0.9	1.3	1.4
	(1.2–2.8)	(1.1–2.7)	(0.6–2.1)	(0.4–1.8)	(0.9–1.9)	(0.9–2.0)
3	1.8	1.5	1.6	1.2	1.5	1.5
	(1.1–2.8)	(0.9–2.6)	(0.8–3.3)	(0.6–2.6)	(1.0–2.2)	(1.0–2.2)
≥4	1.7	1.4	1.9	1.5	1.7	1.4
	(1.0–2.8)	(0.8–2.4)	(0.9–3.9)	(0.7–3.2)	(1.1–2.6)	(0.9–2.2)
χ^2_1	8.63‖	2.39	4.07§	1.32	9.09‖	3.77
Duration (years)						
Non-drinkers	1‡	1‡	1‡	1‡	1‡	1‡
<30	1.6	1.5	0.8	0.7	1.2	1.2
	(1.1–2.4)	(1.0–2.3)	(0.4–1.5)	(0.4–1.5)	(0.9–1.7)	(0.9–1.7)
≥30	1.6	1.5	1.7	1.3	1.5	1.4
	(1.1–2.5)	(1.0–2.3)	(0.9–3.0)	(0.7–2.5)	(1.1–2.1)	(0.9–2.2)
χ^2_1	4.13§	2.44	8.18‖	3.35	6.28§	5.94§
Decaffeinated coffee						
Non-drinkers	1‡	1‡	1‡	1‡	1‡	1‡
Drinkers	1.5	1.3	1.9	1.7	1.6	1.5
	(0.8–2.7)	(0.7–2.5)	(0.9–3.8)	(0.8–3.8)	(1.0–2.5)	(0.9–2.4)
Tea						
Non-drinkers	1‡	1‡	1‡	1‡	1‡	1‡
Drinkers	0.7	0.8	0.9	0.9	0.8	0.9
	(0.5–1.1)	(0.5–1.5)	(0.5–1.5)	(0.5–1.7)	(0.6–1.1)	(0.6–1.2)
Cola						
Non-drinkers	1‡	1‡	1‡	1‡	1‡	1‡
Drinkers	0.7	0.7	0.5	0.6	0.6	0.6
	(0.3–1.7)	(0.2–1.9)	(0.1–2.2)	(0.1–2.1)	(0.3–1.2)	(0.3–1.4)

*Mantel–Haenszel estimates adjusted for sex and age.

†Multivariate logistic regression equation includes terms for age, sex, education, smoking habits [never smoker, ex- (since <10 and ≥10 years), current smoker: <15; 15–24; >24] alcohol drinking and exposure to occupation at risk (chemical industry, dyestuff, painting, pharmaceutical and coal/gas).

‡Reference category. §$P < 0.05$; ‖$P < 0.01$.

four or more cups per day. Decaffeinated coffee was consumed by 7% of the cases versus 5% of the controls and cola-containing beverages by 2% of the cases versus 3% of the controls. These proportions were similar in the two study centres. The corresponding sex- and age-adjusted and multivariate relative risks are reported in Table 3. The multivariate RR for coffee drinkers vs. non-drinkers was 1.3 (95% CI 1.0–1.8). The point estimate was 1.2 for one cup per day, 1.4 for two, 1.5 for three and 1.4 for four or more cups of coffee per day. The trend in risk was of borderline statistical significance ($\chi^2_1 = 3.77$, $P = 0.05$). With reference to duration of use, the RR was 1.2 for 30 years or less and 1.4 for more than 30 years. The RR was 1.5 (95% CI 0.9–2.4) for decaffeinated coffee, 0.9 (95% CI 0.6–1.2) for tea, and 0.6 (95% CI 0.3–1.4) for cola, respectively. The data from the two study centres were closely comparable (Table 3).

Coffee-related risks were further examined in strata of sex, age group, education and smoking status. The trends in risk were comparable in males and females, and in less and more educated subjects. There was some tendency for the coffee-related risks among older subjects and ex-smokers to be higher, although these interactions were not significant.

Multivariate relative risks for selected measures of coffee consumption were further examined in 105 bladder cancer cases and 338 controls who had never smoked (Table 4). Although the point estimates for subsequent levels of daily intake were above unity, the highest risk estimates were observed in the lowest exposure categories and the trend in risk was not significant.

Table 4. Relative risks (and 95% confidence intervals) of 105 cases of bladder cancer and 338 controls who had never smoked according to level and duration of coffee consumption. Italy, 1985–1990

	Cases	Controls	RR*
Non-drinkers	18(17.1)	68(20.1)	1†
Drinkers (cups/day)			1.9(1.0–3.4)
1	34(32.4)	88(26.0)	1.9(1.0–3.9)
2	13(12.4)	100(29.6)	1.9(1.0–3.8)
3	6 (5.7)	57(16.9)	1.8(0.8–4.1)
≥4		25 (7.4)	1.5(0.5–4.5)
χ^2_1			1.27
Drinkers (years)			
<30	43(41.0)	164(48.5)	1.5(0.8–3.0)
≥30	44(41.9)	106(31.4)	2.3(1.2–4.5)
χ^2_1			6.53‡

Values are expressed as no. (%)
*Multivariate regression equation includes terms for age, sex, education, alcohol consumption, centre and exposure to occupation at risk (chemical industry, dyestuff, paint, pharmaceutical and coal/gas).
†Reference category. ‡$P < 0.05$.

DISCUSSION

The results of this study again confirm that the prevalence of coffee drinking is higher among bladder cancer cases than controls, in the absence, however, of any clear dose–risk relationship. Part of this apparent association was explained by a positive correlation between coffee and cigarette smoking, but even after careful allowance for smoking the risk of bladder cancer was still higher, although not significantly, in coffee drinkers. A similar pattern of risk emerged in lifelong non-smokers, despite the too limited absolute number of cases to allow any definite inference.

Besides a lower tobacco consumption, those who were coffee drinkers in this study were somewhat less educated and tended to report lower intake of alcoholic beverages. None of these covariates, however, explained the coffee and bladder cancer relationship.

This is a typical hospital-based case–control study and has, therefore, all the relative strengths and weaknesses. It is conceivable, for instance, that coffee drinking in hospital patients differs from that of the general population [10], although bias should be limited by the fact that we had carefully excluded digestive tract disorders and any other condition potentially influencing coffee drinking habits. As for other potential biases, we were largely reassured by the practically complete participation, and by the observation that the major known risk factors for bladder cancer (smoking and occupational exposures) consistently emerged in the present study. Residual confounding by smoking due to misclassification is possible [11], as the positive but weak correlations between number of cigarettes and number of cups of regular coffee per day suggest ($r = 0.18$ in all the subjects, 0.22 in cases, and 0.15 in controls), but is probably not of substantial importance in view of the similarity of the pattern of risk in lifelong non-smokers [5, 12, 13]. Confounding by other correlates of coffee drinking may also have contributed to the observed relationship, although allowance for a large number of identified covariates (including social class, dietary indicators and occupation) did not appreciably modify the risk estimates. Probably the most convincing evidence of the absence of substantial bias is, *a posteriori*, the consistency between the results of the present study and the accumulated evidence, arising from studies from different parts of the world, on the coffee–bladder cancer relation [1]. This enables us confidently to exclude a strong association between coffee and bladder cancer, while at the same time suggesting that coffee drinking represents an indicator of risk [1]. Whether this indicator is aspecific or includes some aspects of causality is still open to debate.

The lack of clear dose–risk relationship in the overall dataset as well as in lifelong non-smokers reduces the likelihood of a causal association, although it cannot totally exclude it.

Tea consumption (not analysed here as a continuous variable, because of the modest amount both of consumers and of cups per day) showed a relative risk below 1, although the upper confidence limit reached 1.5, but tea consumption in our sample could also be too limited to produce any effect possible to be reflected in an elevation in risk [2, 4].

The elevated relative risks related to intake of decaffeinated coffee suggest that caffeine is unlikely to be the only cause of the emerging positive association of coffee with bladder cancer in determining the increasing of the risk. The elevation in risk for decaffeinated coffee is not explainable in terms of correlation with regular coffee consumption, since in the two samples under study the correlation coefficient between the two types of coffee was −0.03 ($r = -0.08$ in Milan and 0.05 in Pordenone).

In biological terms, the large number of substances other than caffeine contained in coffee have a wide spectrum of direct as well indirect metabolic activities, and it is conceivable that even small amounts of coffee may modify the levels of carcinogens or anticarcinogens in the bladder endothelium, because most substances or metabolites are secreted through the urinary tract and are consequently in direct contact with the bladder mucosa [14, 15]. Alternatively, but less likely, coffee drinking may have some influence on diagnostic pattern of bladder cancer, thus systematically interfering with the likelihood of interview. These

and other possible sources of error or bias may, at first sight, appear mere speculations. However, after the amount of epidemiological research which has systematically tended to reproduce, with remarkable consistency, an association between coffee and bladder cancer, even apparently less plausible hypotheses should be considered, and, if possible, tested in future research.

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The Effect of Family History of Cancer, Religion, Parity and Migrant Status on Survival in Colorectal Cancer

Data from the Melbourne Colorectal Cancer Study

Gabriel A. Kune, Susan Kune and Lyndsey F. Watson

The association between 5-year survival and several risk factors was investigated in 705 histologically confirmed, new cases of colorectal adenocarcinoma as one aspect of a comprehensive population-based study of large bowel cancer incidence, aetiology and survival—the Melbourne Colorectal Cancer Study. 5-year survival was not influenced by the previously determined risk of a family history of colorectal cancer in near-relatives. Similarly, other previously determined risk factors of religion, number of children, age at birth of first child and migrant status did not influence survival.
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INTRODUCTION

COLORECTAL CANCER remains one of the commonest cancers in the world and is particularly common in developed societies. An understanding of the factors which determine survival in this cancer is particularly relevant to enable accurate prognostication in an individual case and also in the study of the natural history of colorectal cancer.

This communication is based on data derived from the Melbourne Colorectal Cancer Study which is a large, comprehensive, epidemiological and clinicopathological investigation of large bowel cancer incidence, aetiology and survival [1, 2]. This study has three main arms, the incidence study, the case-control study and the survival study. The incidence study determined the demographic variables, the case-control study examined all the hypothesised aetiological and risk factors such as diet and heredity, and the survival study examined survival in this group of patients.

The data for this paper are derived from both the case control and the survival arms of the study. The aim of this communication was to examine whether certain factors which are known risks for this cancer, such as a family history of

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