Patterns of alcohol consumption in middle aged men from France and Northern Ireland. the PRIME Study

European Journal of Clinical Nutrition (2000) 54, 321–328

www.nature.com/ejcn

Patterns of alcohol consumption in middle-aged men from France and Northern Ireland. The PRIME Study

P Marques-Vidal[1]*, D Arveiler[2], A Evans[3], M Montaye[4], A Bingham[5], JB Ruidavets[1], D McMaster[3], B Haas[2], P Amouyel[4] and P Ducimetière[5]

[1]*INSERM U518, Faculté de Médecine Purpan, Toulouse, France;* [2]*MONICA-Strasbourg, Laboratoire d'Epidémiologie et de Santé Publique, Strasbourg, France;* [3]*Belfast-MONICA, Division of Epidemiology, The Queen's University of Belfast, Belfast, UK;* [4]*MONICA-Lille, Institut Pasteur de Lille, Lille, France; and* [5]*INSERM U258, Hôpital Paul Brousse, Villejuif, France*

Objective: To assess the patterns of alcohol consumption in France and Northern Ireland.
Design: Four cross-sectional studies.
Setting: Sample of 50–59 y old men living in France and Northern Ireland, consuming at least one unit of alcoholic beverage per week.
Subjects: 5363 subjects from France and 1367 from Northern Ireland.
Interventions: None.
Results: Consumption of wine was higher in France whereas consumption of beer and spirits was higher in Northern Ireland. Alcohol drinking was rather homogeneous throughout the week in France, whereas Fridays and Saturdays accounted for 60% of total alcohol consumption in Northern Ireland. In both countries, current smokers had a higher consumption of all types of alcoholic beverages than non-smokers. Similarly, obese and hypertensive subjects had a higher total alcohol consumption than non-obese or normotensive subjects, but the type of alcoholic beverages differed between countries. In Northern Ireland, subjects which reported some physical activity consumed significantly less alcoholic beverages than sedentary subjects, whereas no differences were found in France. Conversely, subjects with dyslipidemia consumed more alcoholic beverages than normolipidemic subjects in France, whereas no differences were found in Northern Ireland. In France, total alcohol, wine and beer consumption was negatively related to socioeconomic status and educational level. In Northern Ireland, total alcohol, beer and spirits consumption was negatively related whereas wine consumption was positively related to socioeconomic status and educational level.
Conclusions: Alcohol drinking patterns differ between France and Northern Ireland, and also according to cardiovascular risk factors, socioeconomic and educational levels.
Sponsorship: Merck, Sharp & Dohme-Chibret (France), the NICHSA and the Department of Health and Social Service (Northern Ireland).
Descriptors: alcohol; epidemiology; wine; beer; socioeconomic status; drinking patterns
European Journal of Clinical Nutrition (2000) **54**, 321–328

Introduction

Moderate intake of alcohol has been shown to be inversely related to the incidence of coronary artery disease (Marmot *et al*, 1981; Kozarevic *et al*, 1982; Friedman and Kimball, 1986; Moore & Pearson, 1986; Lang *et al*, 1987; Jackson *et al*, 1991; Rimm *et al*, 1996; Camargo *et al*, 1997; Tunstall-Pedoe *et al*, 1997). Although some authors have found that the protective effect of wine is superior to that of other alcoholic beverages (Rosenberg *et al*, 1981; Renaud & De Lorgeril, 1992; Goldberg, 1995), those results have not been confirmed in other studies (Hennekens *et al*, 1979; Marques-Vidal *et al*, 1995). The mechanisms by which moderate alcoholic beverages consumption decreases coronary artery disease include the increase of high density lipoprotein levels (Gaziano *et al*, 1993; Marques-Vidal *et al*, 1995; Parker *et al*, 1996; Paunio *et al*, 1996), the modification of hemostatic parameters (Sumi *et al*, 1988; Owens *et al*, 1990; Hendricks *et al*, 1994; Pellegrini *et al*, 1996) and the inhibition of lipoprotein oxidation by phenolic components present in red wine (Anonymous, 1993; Frankel *et al*, 1993; Kinsella *et al*, 1993; Serafini *et al*, 1994; Mérillon *et al*, 1997). However, although there is definitely a biological effect of alcohol, differences in consumption patterns and in lifestyle can also partly account for the relationship between alcohol consumption and coronary artery disease. For instance, moderate drinkers tend to smoke less than other groups (Marques-Vidal *et al*, 1995), while hypertension control is inversely related to alcohol consumption (Lang *et al*, 1987). In addition, wine-drinking populations tend to have a healthier diet than those which consume other alcoholic beverages (Evans *et al*, 1995; Tjønneland *et al*, 1999), whereas the bingeing style of drinking beer has been associated with a higher

*Correspondence: P Marques-Vidal, INSERM U518, Faculté de Médecine, Département d'Epidémiologie, 37 Allées Jules Guesde, 31073 Toulouse cedex, France.
Guarantor: J Ferrières.
Contributors: PMV and JBR carried out the statistical analysis and wrote the article. JBR also collected and controlled all data from the Toulouse center. BH and DA collected and controlled the data from the Strasbourg center, and made corrections to the article. AE and DMcM collected and controlled the data from the Belfast center, and made corrections to the article (content and language). MM and PA collected and controlled all data from the Lille center, and commented on the article. AB and PD were in charge of merging, controlling and correcting the data sets from all participating centers, and contributed to the statistical analysis of the data.
Received 15 June 1999; revised 23 September 1999; accepted 5 November 1999

risk of death from any cause (including cardiovascular disease; Kozarevic *et al*, 1983; Kauhanen *et al*, 1997).

Finally, most ecological studies on the relationship between alcohol intake and coronary artery disease have focused on the average intake of alcohol (St Leger *et al*, 1979; Renaud & De Lorgeril, 1992), whereas the pattern of intake (continuous or binge-drinking) has not been considered. In a previous report (Marques-Vidal *et al*, 1996), we assessed the alcohol consumption between myocardial infarction patients and healthy controls in France and in Northern Ireland. In this study, we use data from the PRIME Study (Prospective Epidemiological Study of Myocardial Infarction), which also involved France and Northern Ireland, to analyze the patterns of alcohol consumption and their relationship with socioeconomic variables in middle-aged men.

Methods

Population sampling

The PRIME Study (Prospective Epidemiological Study of Myocardial Infarction) was established in 1991 in the populations of four collaborating centers of Belfast (UK), Lille, Strasbourg and Toulouse (France). The target was to recruit 2500 men, aged 50–59 y, in each center and to follow them for a minimum of 5 y. The sample was recruited to match broadly the social class structure of the background population. The sampling frame was based on industry and various employment groups, and on health screening centers and general practice. Participation was voluntary. Subjects were informed of the aim of the study and those who agreed to take part were given a morning appointment and asked to fast for a period of a minimum of 10 h.

Personal and medical history

Self-administered questionnaires relating to demographic, socioeconomic factors and diet were completed at home by the participants and checked by the interviewer at the clinic. Data on level of education, occupational activity, personal and family history, tobacco and alcohol consumption, drug intake and physical activity were collected.

Blood pressure was measured once at the end of the examination after a 5 min rest in the sitting position. Measurements were performed with an automatic device (Spengler SP9), which also recorded heart rate. A standard cuff size was used, but a large cuff was available when necessary. At least three measuring devices were available at any time in each center and all three were used equally. In order to avoid systematic differences between centers, the devices were circulated between them. The devices were also recalibrated every 3 months in the co-ordinating center in Paris.

Subjects were considered as hypertensive if their systolic blood pressure was $\geq$ 160 mmHg and/or their diastolic blood pressure was $\geq$ 95 mmHg. Subjects were considered as having dyslipidemia if their cholesterol was $\geq$ 250 mg/dl and/or their triglycerides were $\geq$ 200 mg/dl. Obesity was defined as a body mass index $\geq$ 27 kg/m^2. Subjects were considered as having physical activity if they exercised at least once per week.

Blood sampling and assay procedures

Venous blood was collected between 9 and 10 a.m. into siliconized vacutainer tubes (Vacutainer, Becton Dickinson) containing EDTA. Aliquots of plasma were immediately transferred into plastic tubes and frozen at − 80°C. The frozen aliquots were then shipped in batches to the central laboratory in Lille. Plasma total cholesterol and triglyceride levels were measured by enzymatic methods using reagents from Boerhinger Mannheim (Mannheim, Germany). The inter-assay coefficients of variation for total cholesterol and triglyceride were 2% and 3%, respectively.

Alcohol consumption

Alcohol consumption was assessed by a questionnaire where the subject reported his mean consumption (in units) of wine, beer, cider and spirits for each day of the week. Intake of alcohol (expressed in ml of pure ethanol) was estimated from the average number of milliliters of ethanol in a serving of each type of alcoholic beverage: wine, 120 ml serving, 10% or 12% alcohol (vol/vol); beer, 120 ml serving, 5% alcohol, 250 or 330 ml serving, 6 or 8% alcohol; cider, 120 ml serving, 5% alcohol; spirits, 20 or 60 ml serving, 20% or 40% alcohol. The amount of alcohol was calculated as follows: serving volume (ml) × alcoholic degree.

Statistical analysis

Out of the 10,596 subjects initially included in the study, 593 were excluded from the analysis because of a personal history of angina pectoris, myocardial infarction or possible coronary heart disease, 1823 because of pharmacological treatment for hypertension or dyslipidemia and 1450 because they reported no consumption of alcoholic beverages. Statistical analysis was conducted using SAS (Cary, NC, USA) software. Data are presented as median (5th 95th percentiles). Comparisons were performed by non-parametric tests. Due to the number of tests performed, statistical significance was considered only for $P < 0.01$.

Results

Population and types of alcohol consumed

In all data from 6730 subjects who reported consuming at least one alcoholic beverage per week was analyzed (5363 in France and 1367 in Northern Ireland).

Mean consumption of alcohol (expressed in milliliters of ethanol per week) was slightly higher in Northern Ireland than in France (mean ± s.d.: 326 ± 333 *vs* 318 ± 249 ml/week, respectively; Kruskal–Wallis = 8.20, $P <$ 0.01). When total alcoholic consumption was separated into wine, beer and spirits, very significant differences were observed: wine accounted for most of the alcohol consumed in France, whereas beer and spirits accounted for most of the alcohol consumed in Northern Ireland (Table 1).

Table 1 Consumption of different types of alcoholic beverages among subjects consuming at least one unit per week. Results are expressed in ml of ethanol per week and as median (5th–95th percentiles)

	France (n = 5363)	*N. Ireland (n = 1367)*	*Test*
Wine	180 (0–570)	0 (0–101)	2166.8***
Beer	15 (0–336)	132 (0–788)	544.7***
Spirits	12 (0–104)	36 (0–369)	118.6***

Kruskal–Wallis test: *$P < 0.01$; ***$P < 0.001$.

Table 2 Consumption of different types of alcoholic beverages among subjects consuming at least one unit per week. Results are expressed in ml of ethanol per week and as median (5th–95th percentiles)

	Toulouse (n = 1767)	*Strasbourg (n = 1872)*	*Lille (n = 1724)*	*Test*
Total	252 (30–683)	270 (34–787)	282 (30–840)	34.0***
Wine	210 (0–588)	168 (0–525)	168 (0–630)	51.1***
Beer	0 (0–140)	36 (0–364)	26 (0–420)	725.7***
Spirits	12 (0–84)	12 (0–81)	24 (0–168)	187.7***

Kruskal–Wallis test: *$P < 0.01$; ***$P < 0.001$.

Drinkers from Toulouse had a lower total alcohol consumption than those from Lille and Strasbourg (Table 2). Also, when the types of alcoholic beverages were assessed, the consumption of wine was higher and the consumption of beer was lower in Toulouse than in the other two French centers (Table 2).

Alcohol consumption during the week
In France, total alcohol consumption was evenly distributed throughout the week, with a slight increase on weekends. Conversely, consumption of alcohol on Fridays and Saturdays accounted for almost two-thirds of total alcohol consumption in Northern Ireland (Figure 1). Similar patterns were observed when total alcohol consumption was separated into wine, beer and spirits (Figures 2–4).

Alcohol consumption and cardiovascular risk factors
In France, total alcohol consumption decreased from current smokers to ex-smokers and non-smokers. When total alcohol consumption was separated into wine, beer and spirits, similar patterns were observed (Table 3). In Northern Ireland, total alcohol consumption was also higher in current smokers than in ex- or non-smokers, but wine consumption was higher among ex- and non-smokers compared to current smokers, whereas the consumption of beer and spirits was higher among smokers (Table 4).

In both countries, obese subjects consumed more alcoholic beverages, but the type of beverage differed slightly: in France, obese subjects consumed more beer and spirits than non-obese (Table 3), whereas in Northern Ireland obese subjects consumed more wine and spirits than non-obese (Table 4).

In both countries, total alcohol and beer consumption were higher in hypertensive than in normotensive subjects (Tables 3 and 4). A higher consumption of spirits was also found in French hypertensive subjects, but not in their Northern Irish counterparts.

In France, subjects with dyslipidemia consumed significantly more alcoholic beverages (wine, beer and spirits) than normolipidemic subjects (Table 3), while no differences were found in Northern Ireland (Table 4). Conversely, in France, no differences were found between subjects engaging in physical activity and sedentary individuals (Table 2), whereas in Northern Ireland subjects engaging in physical activity consumed significantly less total alcohol, less beer and more wine than non-active, sedentary subjects (average

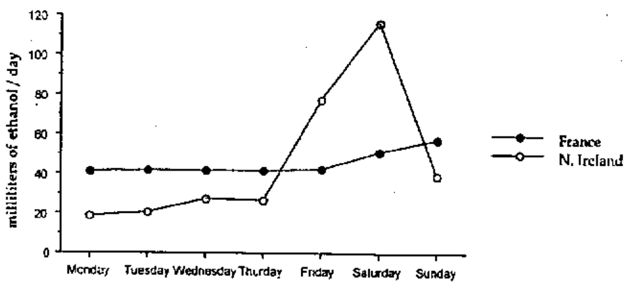

Figure 1 Total alcohol consumption according to the day of the week for France and Northern Ireland. Results obtained from the subjects consuming at least one unit per week, and expressed in milliliters of ehtanol per day.

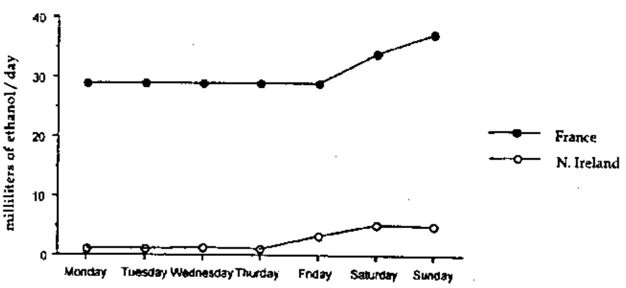

Figure 2 Wine consumption according to the day of the week for France and Northern Ireland. Results obtained from the subjects consuming at least one unit per week, and expressed in milliliters of ethamol per day.

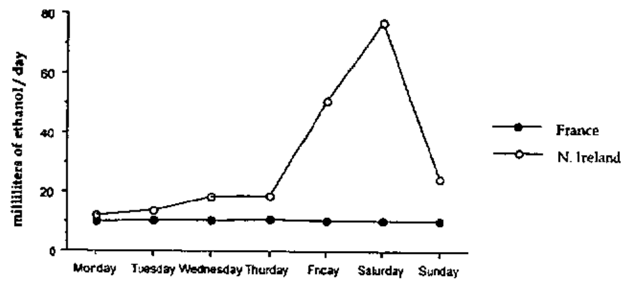

Figure 3 Beer consumption according to the day of the week for France and Northern Ireland. Results obtained from the subjects consuming at least one unit per week, and expressed in milliliters of ethanol per day.

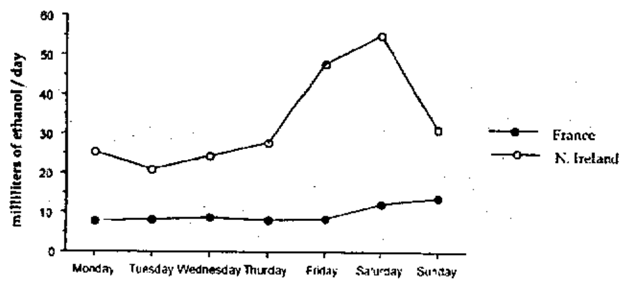

Figure 4 Spirit consumption according to the day of the week for France and Northern Ireland. Results obtained from the subjects consuming at least one unit per week, and expressed in milliliters of ethanol per day.

Table 3 Alcohol consumption according to different cardiovascular risk factors in France. Results are expressed in ml of alcohol per week and as median (5th–95th percentiles)

	Total alcohol	*Test*	*Wine*	*Test*	*Beer*	*Test*	*Spirits*	*Test*
Smoking status:								
non-smoker ($n=1464$)	204 (24–630)		134 (0–473)		12 (0–260)		12 (0–72)	
ex-smoker ($n=2402$	269 (31–735)	283.8	180 (15–588)	124.5	15 (0–315)	51.9	16 (0–100)	113.5
current smoker ($n=1496$)	350 (42–900)	***	210 (0–630)	***	24 (0–446)	***	20 (0–138)	***
Obesity:								
obese ($n=2085$)	284 (31–827)	15.4	204 (0–630)	4.1	20 (0–357)	13.1	18 (0–123)	23.5
non-obese ($n=3277$)	258 (30–730)	***	180 (0–525)	NS	15 (0–327)	***	12 (0–96)	***
Physical activity:								
yes ($n=2722$)	271 (30–770)	0.6	210 (0–588)	4.3	15 (0–322)	0.9	12 (0–98)	0.8
no ($n=2641$)	264 (32–768)	NS	168 (0–552)	NS	15 (0–360)	NS	14 (0–108)	NS
Hypertension:								
yes ($n=957$)	331 (36–886)	39.8	210 (0–630)	0.9	36 (0–440)	103.6	18 (0–134)	10.4
no ($n=4406$)	258 (30–738)	***	180 (0–540)	NS	12 (0–312)	***	12 (0–96)	**
Dyslipidemia:								
yes ($n=1529$)	313 (36–865)	47.3	210 (0–630)	10.9	24 (0–420)	33.9	18 (0–140)	27.7
no ($n=3834$)	255 (30–726)	***	180 (0–525)	***	15 (0–300)	***	12 (12–96)	***

Kruskal–Wallis test: NS, not significant; $*P<0.01$; $**P<0.005$; $***P<0.001$.

consumption for wine, mean ± s.d.: 21 ± 50 *vs* 16 ± 46 ml/week, respectively, Kruskal–Wallis = 14.4, $P<0.001$; Table 4).

Alcohol consumption and socioeconomic variables
In France, single, widowed or separated subjects had higher total alcohol consumption than married or cohabiting subjects. This higher consumption was due chiefly to an increase in the consumption of beer, whereas the consumption of wine and spirits was comparable. Similar results were found for Northern Ireland, where single, widowed or separated subjects consumed more beer and less wine than married or cohabiting subjects. Although the consumption of spirits tended to be higher among single subjects in Northern Ireland, no significant differences were found between the two groups (Tables 5 and 6).

Table 4 Alcohol consumption according to different cardiovascular risk factors in Northern Ireland. Results are expressed in ml of alcohol per week and as median (5th–95th percentiles)

	Total alcohol	*Test*	*Wine*	*Test*	*Beer*	*Test*	*Spirits*	*Test*
Smoking status:								
non-smoker ($n=352$)	171 (24–825)		0 (0–101)		77 (0–693)		28 (0–241)	
ex-smoker ($n=474$)	231 (43–794)	55.4	0 (0–108)	40.5	128 (0–660)	40.4	43 (0–369)	16.5
current smoker ($n=540$)	278 (57–1153)	***	0 (0–86)	***	165 (0–923)	***	57 (0–426)	***
Physical activity:								
yes ($n=664$)	206 (36–792)	20.4	0 (0–108)	14.4	102 (0–650)	26.9	28 (0–341)	2.3
no ($n=703$)	256 (36–1035)	***	0 (0–86)	***	154 (0–858)	***	43 (0–369)	NS
Obesity:								
obese ($n=484$)	256 (38–924)	7.6	0 (0–86)	16.1	145 (0–768)	2.7	57 (0–398)	11.6
non-obese ($n=883$)	225 (36–934)	*	0 (0–108)	***	128 (0–792)	NS	28 (0–312)	***
Hypertension:								
yes ($n=223$)	303 (48–1035)	18.2	0 (0–101)	4.2	198 (0–922)	12.6	43 (0–398)	2.9
no ($n=1144$)	222 (36–915)	***	0 (0–101)	NS	128 (0–717)	***	36 (0–341)	NS
Dyslipidemia:								
yes ($n=618$)	239 (43–878)	2.3	0 (0–108)	0.0	132 (0–717)	1.0	43 (0–341)	1.1
no ($n=749$)	222 (33–954)	NS	0 (0–86)	NS	128 (0–792)	NS	28 (0–384)	NS

Kruskal–Wallis test: NS, not significant; $*P<0.01$; $**P<0.005$; $***P<0.001$.

Table 5 Alcohol consumption according to socioeconomic variables in France. Results are expressed in ml of alcohol per week and as median (5th–95th percentiles)

	Total alcohol	*Test*	*Wine*	*Test*	*Beer*	*Test*	*Spirits*	*Test*
Marital status:								
married/cohabiting ($n=4738$)	264 (30–744)	8.2	180 (15–567)	0.3	15 (0–315)	5.4	13 (0–100)	3.24
other ($n=624$)	294 (42–912)	*	180 (0–630)	NS	20 (0–504)	NS	12 (12–120)	NS
Professional status:								
active ($n=4254$)	260 (30–744)		180 (0–540)		15 (0–315)		14 (0–99)	
sick leave ($n=167$)	321 (44–924)	21.5	210 (0–672)	4.1	15 (0–420)	16.5	9 (0–113)	7.12
retired ($n=533$)	291 (30–731)	***	210 (0–588)	NS	20 (0–312)	***	14 (0–90)	NS
inactive ($n=408$)	336 (30–928)		210 (0–630)		25 (0–500)		12 (0–132)	
Years of education:								
≤ 7 ($n=292$)	336 (30–905)		210 (0–672)		0 (0–420)		12 (0–108)	
7–11 ($n=2628$)	291 (42–912)	76.2	204 (0–630)	23.3	24 (0–416)	98.3	16 (0–113)	10.6
11–15 ($n=1439$)	264 (30–717)	***	180 (0–525)	***	15 (0–280)	***	14 (0–96)	NS
≥ 15 ($n=1002$)	219 (30–656)		165 (0–504)		0 (0–210)		12 (0–96)	

Kruskal–Wallis test: NS, not significant; $*P<0.01$; $**P<0.005$; $***P<0.001$.

Table 6 Alcohol consumption according to socioeconomic variables in Northern Ireland. Results are expressed in ml of alcohol per week and as median (5th–95th percentiles)

	Total alcohol	*Test*	*Wine*	*Test*	*Beer*	*Test*	*Spirits*	*Test*
Marital status:								
married/cohabiting ($n = 1162$)	222 (36–87))	24.8	0 (0–101)	13.0	115 (0–666)	24.4	39 (0–312)	0.6
other ($n = 205$)	330 (43–1485)	***	0 (0–86)	***	198 (0–1056)	***	28 (0–426)	NS
Professional status:								
active ($n = 1194$)	222 (33–870)		0 (0–106)		102 (0–660)		43 (0–369)	
sick leave ($n = 27$)	288 (51–1386)	32.8	0 (0–144)	30.1	230 (0–495)	51.5	0 (0–284)	11.7
retired ($n = 24$)	189 (51–979)	***	0 (0–130)	***	143 (0–717)	***	14 (0–578)	*
inactive ($n = 122$)	332 (85–1485)		0 (0–29)		264 (0–1155)		0 (0–312)	
Years of education:								
≤ 7 ($n = 10$)	212 (102–600)		0 (0–58)		77 (0–429)		156 (0–596)	
7–11 ($n = 918$)	264 (43–1048)	67.0	0 (0–72)	206.5	176 (0–896)	128.6	28 (0–369)	3.6
11–15 ($n = 282$)	183 (28–623)	***	0 (0–144)	***	51 (0–512)	***	43 (0–284)	NS
≥ 15 ($n = 157$)	143 (26–648)		24 (0–144)		26 (0–358)		28 (0–398)	

Kruskal–Wallis test: NS, not significant; $*P < 0.01$; $**P < 0.005$; $***P < 0.001$.

In both countries, professionally active subjects drank less alcohol, whereas the highest consumption levels were found for subjects on sick leave or inactive (unemployed). In France, the increase in total alcohol consumption was mainly due to an increase in the consumption of beer, while the consumption of wine and spirits was comparable (Table 5). In Northern Ireland, the higher total alcohol consumption was accounted for by higher consumption of beer and spirits, whereas the consumption of wine was higher for professionally active and retired subjects (Table 6).

In France, total alcohol consumption decreased when the educational level of the subject (expressed as number of years of education) increased. This lower intake was found for wine and beer, whereas no difference was found for spirits (Table 5). A similar pattern was found for total alcohol consumption in Northern Ireland, but in this country only the consumption of beer was lower in more well-educated subjects, whereas the consumption of wine showed a positive association with educational level (Table 6). Again, no differences were found for the consumption of spirits, although a trend towards a lower consumption among more well-educated subjects was observed.

Discussion

Several studies have shown that moderate alcohol consumption is inversely related to the incidence of coronary artery disease (Rimm *et al*, 1996; Camargo *et al*, 1997; Tunstall-Pedoe *et al*, 1997). Not taking into account the types of alcoholic beverages consumed might be misleading, since in this study two populations which possess very different coronary artery disease rates have in fact the same total alcohol consumption, but differ considerably regarding the type of alcoholic beverages consumed. Our data also indicate that even within France, a country characterized by a high relative intake of wine, the total amount and the consumption of the different types of alcoholic beverages vary considerably according to the geographical region, probably due to local factors such as cultural and nutritional heritage (Trichopoulou & Lagiou, 1997). Those findings are in agreement with another study, which showed that the nutritional intake of subjects living in Toulouse differs from that of subjects living in Strasbourg (Ruidavets *et al*, 1993). Thus, considering the relationships between coronary artery disease and alcohol consumption *per se* and not taking into account the general nutritional pattern of the population studied might lead to spurious results, since differences in alcohol consumption might also reflect differences in nutritional intake, which can influence cardiovascular risk factors. For instance, Toulouse has a higher consumption of wine than Belfast, but also a higher consumption of vegetables and fruit (Evans *et al*, 1995), which possess a beneficial effect towards coronary artery disease (Ness & Powles, 1997). The quality of the overall nutritional intake in France (and not only of its alcoholic beverages) has also been suggested as possible determinant of the 'French paradox' (Drewnowski *et al*, 1996). Finally, it should be stressed that the impact of shifting a single nutrient or food in the diet appears to have less effect on coronary artery disease (Caggiula *et al*, 1996; He & Whelton, 1997) than a complete change in nutritional lifestyle (de Lorgeril *et al*, 1996). Taken together, our data indicate that focusing on a single nutrient or food (alcohol or alcoholic beverages) without taking into account the pattern of consumption and the nutritional background of the population studied might lead to inconsistent results.

The relationships between alcohol consumption and coronary artery disease are further complicated by the fact that the nutritional intake of a population can vary with time (Welsh & Marston, 1982). Thus, it has been shown that wine consumption has been decreasing in Southern Europe whereas it has been increasing in Northern Europe, the opposite being true for the consumption of beer (Marques-Vidal, unpublished observations; Simpura *et al*, 1995; Knibbe *et al*, 1996; Gual & Colom, 1997). Considering that coronary artery disease has been decreasing in all European countries (Sans *et al*, 1997), one would infer from ecological correlations that wine consumption is negatively related to coronary artery disease in Northern European countries, whereas it is beer (and not wine) consumption that is negatively related to coronary artery disease in Southern, wine-producing European countries. Also, most studies which have found an inverse relationship between wine and coronary artery disease were carried out in populations whose alcohol intake was almost exclusively due to wine consumption (Farchi *et al*, 1992), whereas studies which found an inverse relationship between beer or spirits and coronary artery disease have concerned populations with a high intake of those types of alcohol (Yano *et al*, 1977; Rimm *et al*, 1991). Thus, assessing the relationships between alcoholic beverages and coronary artery disease without taking into account the relative amount of each beverage type in the population studied and even the overall consumption trends might lead to inconsistent results.

Many studies on the effects of alcohol on coronary artery disease have used total alcohol consumption or the consumption of various types of alcohol, without taking into account the pattern of consumption (see (Rimm *et al*, 1996) for a review). Not taking into account the pattern of consumption might be misleading, as we have seen in this study that two populations with the same total alcohol consumption differ regarding drinking habits (continuous or binge-drinking). Indeed, the effect of pattern alcohol consumption on cardiovascular risk factor levels has seldom been studied. Binge drinking has been associated with a higher pressor effect (Moreira *et al*, 1998; Rakic *et al*, 1998), although this effect appears to be time-dependent. Consumption of wine with the evening meal has also been associated with an increase in plasminogen activator activity early in the next morning (Hendricks *et al*, 1994; Muller & Fugelsang, 1994). Further, in Northern Ireland, alcohol is mostly consumed outside meals, whereas in France it is mostly consumed with meals, and it has been shown that the pharmacological effect of ethanol can be influenced by meals (Jones *et al*, 1997). Finally, since a binge-type consumption of alcohol appears to be deleterious regarding cardiovascular diseases (Kauhanen *et al*, 1997), it could be speculated that differences in the pattern of consumption of alcoholic beverages (irrespective of the type) could also partly explain the differences in coronary artery disease between France and Northern Ireland.

In both countries, a higher consumption of beer was found for subjects who were single or separated, who were on sick leave or inactive, or who were less well educated. Those results are in agreement with other studies (Burke *et al*, 1995; Westin, 1995; Tjonneland *et al*, 1999), and indicate that the consumption of beer is positively associated with a low socioeconomic level. Since a low socioeconomic level is associated with a poorer health status and a higher incidence of cardiovascular disease (Lang *et al*, 1988, 1997; Lang & Ducimetière, 1995), our findings might explain why in some studies the consumption of beer had no protective effect against cardiovascular disease. A lower consumption of alcohol was also found among subjects who were professionally active, with a higher socioeconomic status and married. Those findings are in agreement with other studies (French & Zarkin, 1995; Heien, 1996) and might also partly explain the beneficial effect of moderate drinking, as the incidence of cardiovascular disease is lower in those groups. Still, those findings cannot be extrapolated to all populations, since it has been shown that in some countries alcohol consumption is positively related with financial status (Balabanova & McKee, 1999). Finally, the positive relationship between wine drinking, and the negative relationship between beer consumption with socioeconomic status found in Northern Ireland could be explained by economic constraints, the price of wine being usually higher than the price of beer in countries which are not wine producers. This might explain why in most studies conducted in Anglo-American countries the consumption of wine was found to be protective, whereas no effect was found for beer (Rosenberg *et al*, 1981; Goldberg, 1995).

The lower consumption of beer and spirits and the higher consumption of wine found in physically active subjects in Northern Ireland could be explained by a healthier lifestyle, as it has been suggested previously (Evans *et al*, 1995; Tjonneland *et al*, 1999). The lack of such a relationship in France awaits further investigation, but this could eventually indicate that alcohol consumption does not preclude a healthy lifestyle among French subjects.

In this study, several cardiovascular risk factors (smoking, obesity, hypertension and dyslipidemia) were positively related to a higher consumption of alcoholic beverages. Those relationships are in agreement with other previous studies (Marques-Vidal *et al*, 1995), indicating that alcohol consumption is related to cardiovascular risk factor levels. Nevertheless, some care should be taken when establishing those relationships, since it has been shown that socioeconomic status also influence the levels of cardiovascular risk factors (Fouriaud *et al*, 1984). For instance, the higher consumption of beer among hypertensive subjects in both countries could be due to a lower socioeconomic status rather than to a direct effect of beer on blood pressure levels, although this hypothesis remains to be assessed. Thus, since overall *per capita* alcohol consumption of beverage-specific consumption may be related to other important correlates of coronary artery disease (such as cardiovascular risk factors, dietary intake or socioeconomic status), a crude comparison of disease rates between countries with differing average alcohol consumption might produce misleading results (Westin, 1995; Tjonneland *et al*, 1999).

Finally, it should be stressed that, whereas 90% of French subjects reported consuming at least one unit of alcoholic beverage per week, only 60% of Northern Irish subjects did so. This difference indicates that some caution should be taken when comparing the effect of alcohol consumption among populations, since this effect would be much more widespread in the French population than in the Northern Irish. Also, the high frequency of reported teetotalers in Northern Ireland would lead to a lower mean individual consumption of alcoholic beverages relative to other countries.

In summary, our results indicate that total alcohol consumption is a poor indicator of the drinking characteristics for a given population and that the drinking pattern (continuous or binge drinking) together with the nutritional intake of the population should also be considered. The data also indicate that, even when the types of alcohol are taken into account, their consumption varies according to geographical factors, health and socioeconomic status.

Acknowledgements—We would like to thank Dominique Courbon for her valuable help in the statistical analysis of the data. The PRIME Study is supported by grants from Merck, Sharp & Dohme. We thank the following organizations which allowed the recruitment of the PRIME subjects: the Health screening centers organized by the Social Security of Lille (Institut Pasteur), Strasbourg, Toulouse and Turcoing; Occupational Medicine Services of Haute-Garonne, the Urban Community of Strasbourg, the Association Inter-entreprises des Services Médicaux du Travail de Lille et environs; the Comité pour le Développement de la Médecine du Travail; the Mutuelle Générale des PTT du Bas-Rhin; the laboratoire d'Analyses de l'Institut de Chimie Biologique de la Faculté de Médecine de Strasbourg. Thanks are also due to the DHSS (Northern Ireland) and the Northern Ireland Chest, Heart and Stroke Association.

References

Anonymous (1993): Ethanol stimulates apo A-I secretion in human hepatocytes: a possible mechanism underlying the cardioprotective effect of ethanol. *Nutr. Rev.* **51**, 151–152.

Balabanova D & McKee M (1999): Patterns of alcohol consumption in Bulgaria. *Alcohol Alcohol.* **34**, 622–628.
Burke V, Puddey IB & Beilin LJ (1995): Mortality associated with wine, beers, and spirits. *Br. Med. J.* **311**, 1166–1167.
Caggiula AW, Watson JE, Kuller LH, Olson MB, Milas NC, Berry M & Germanowski J (1996): Cholesterol-lowering intervention program. *Arch. Intern. Med.* **156**, 1205–1213.
Camargo CA, Stampfer MJ, Glynn RJ, Grodsetin F, Gaziano JM, Manson JE, Buring JE & Hennekens CH (1997): Moderate alcohol consumption and risk for angina pectoris or myocardial infarction in U.S. male physicians. *Ann. Intern. Med.* **126**, 372–375.
de Lorgeril M, Salen P, Martin JL, Mamelle N, Monjaud I, Touboul P & Delaye J (1996): Effect of a Mediterranean type of diet on the rate of cardiovascular complications in patients with coronary artery disease. *J. Am. Coll. Cardiol.* **28**, 1103–1108.
Drewnowski A, Henderson SA, Shore AB, Fischler C, Preziosi P & Herchberg S (1996): Diet quality and dietary diversity in France: implications for the French paradox. *J. Am. Diet. Assoc.* **96**, 663–669.
Evans AE, Ruidavets JB, McCrum EE, Cambou JP, McLean R, Douste-Blazy P, McMaster D, Bingham A, Patterson CC, Richard JL, Mathewson ZM & Cambien F (1995): Autres pays, autres coeurs? Dietary patterns, risk factors and ischaemic heart disease in Belfast and Toulouse. *Q. J. Med.* **88**, 469–477.
Farchi G, Fidanza F, Mariotti S & Menotti A (1992): Alcohol and mortality in the Italian rural cohorts of the Seven Countries Study. *Int. J. Epidemiol.* **21**, 74–81.
Fouriaud C, Jacquinet-Salord MC, Degoulet P, Aimé F, Lang T, Laprugne J, Mai J, Oeconomos J, Phalente J & Prades A (1984): Influence of socioprofessional conditions on blood pressure levels and hypertension control. *Am. J. Epidemiol.* **120**, 72–86.
Frankel EN, Kanner J, German JB, Parks E & Kinsella JE (1993): Inhibition of oxidation of human low-density lipoprotein by phenolic substances in red wine. *Lancet* **341**, 454–457.
French MT & Zarkin GA (1995): Is moderate alcohol use related to wages? Evidence from four worksites. *J. Health Econ.* **14**, 319–344.
Friedman LA & Kimball AW (1986): Coronary heart disease mortality and alcohol consumption in Framingham. *Am. J. Epidemiol.* **124**, 481–489.
Gaziano JM, Buring JL, Breslow JL, Goldhaber SZ, Rosner B, VanDenburgh M, Willett W & Hennekens CH (1993): Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. *New Engl. J. Med.* **329**, 1829–1834.
Goldberg DM (1995): Does wine work? *Clin. Chem.* **41**, 14–16.
Gual A and Colom J (1997): Why has alcohol consumption declined in countries of southern Europe? *Addiction* **92** (Suppl. 1), S21–S31.
He J & Whelton PK (1997): Role of sodium reduction in the treatment and prevention of hypertension. *Curr. Opin. Cardiol.* **12**, 202–207.
Heien D (1996): The relationship between alcohol consumption and earnings. *J. Stud. Alcohol* **57**, 536–542.
Hendricks HFJ, Veenstra J, Velthuis-te Wierick EJM, Shaafsma G & Kluft C (1994): Effect of moderate dose of alcohol with evening meal on fibrinolytic factors. *Br. Med. J.* **308**, 1003–1006.
Hennekens CH, Willet W, Rosner B, Cole DS & Mayrent SL (1979): Effects of beer, wine, and liquor in coronary deaths. *JAMA* **242**, 1973–1974.
Jackson R, Scragg R & Beaglehole R (1991): Alcohol consumption and risk of coronary heart disease. *Br. Med. J.* **303**, 211–216.
Jones AW, Jonsson KA & Kechagias S (1997): Effect of high-fat, high-protein, and high-carbohydrate meals on the pharmacokinetics of a small dose of ethanol. *Br. J. Clin. Pharmac.* **44**, 521–526.
Kauhanen J, Kaplan GA, Goldberg DE & Salonen JT (1997): Beer binging and mortality: results from the Kuopio ischaemic heart disease risk factor study, a prospective population based study. *Br. Med. J.* **315**, 846–851.
Kinsella JE, Frankel E, German B & Kanner J (1993): Possible mechanisms for the protective role of antioxidants in wine and plant foods. *Food Technology* **00**, 85–89.
Knibbe RA, Drop MJ & Hupkens CL (1996): Modernization and geographical diffusion as explanations for regional differences in the consumption of wine and beer in the European Community. *Substance Use Misuse* **31**, 1639–1655.
Kozarevic D, Vojvodic N, Gordon T, Kaelber CT, McGee D & Zukel WJ (1982): Drinking habits and coronary heart disease: the Yugoslavia cardiovascular disease study. *Am. J. Epidemiol.* **116**, 748–758.
Kozarevic D, Vojvodic N, Gordon T, Kaelber CT, McGee D & Zukel WJ (1983): Drinking habits and death. The Yugoslavia cardiovascular disease study. *Int. J. Epidemiol.* **12**, 145–150.
Lang T & Ducimetière P (1995): Premature cardiovascular mortality in France: divergent evolution between social categories from 1970 to 1990. *Int. J. Epidemiol.* **24**, 331–339.
Lang T, Degoulet P, Aimé F, Devries C, Jacquinet-Salord MC & Fouriaud C (1987): Relations between alcohol consumption and hypertension prevalence and control in a French population. *J. Chron. Dis.* **40**, 713–720.
Lang T, Pariente P, Salem G & Tap D (1988): Social, professional conditions and arterial hypertension: an epidemiological study in Dakar, Senegal. *J. Hypertens.* **6**, 271–276.
Lang T, Ducimetière P, Arveiler D, Amouyel P, Cambou JP, Ruidavets JB, Montaye M, Meyer V & Bingham A (1997): Incidence, case fatality, risk factors of acute coronary heart disease and occupational categories in man aged 30–59 in France. *Int. J. Epidemiol.* **26**, 47–57.
Marmot MG, Rose G, Shipley MJ & Thomas BJ (1981): Alcohol and mortality: a U-shaped curve. *Lancet* **1**, 580–583.
Marques-Vidal P, Cambou JP, Nicaud V, Luc G, Evans A, Arveiler D, Bingham A & Cambien F (1995): Cardiovascular risk factors and alcohol consumption in France and Northern Ireland. *Atherosclerosis* **115**, 225–232.
Marques-Vidal P, Ducimetière P, Evans A, Cambou JP & Arveiler D (1996): Alcohol consumption and myocardial infarction: a case–control study in France and Northern Ireland. *Am. J. Epidemiol.* **143**, 1089–1093.
Mérillon J-M, Fauconneau B, Teguo PW, Barrier L, Vercauteren J & Huguet F (1997): Antioxidant activity of the stilbene astringin, newly extracted from *Vitis vinifera* cell cultures. *Clin. Chem.* **43**, 1092–1093.
Moore RD & Pearson TA (1986): Moderate alcohol consumption and coronary artery disease. A review. *Medicine* **65**, 242–267.
Moreira LB, Fuchs FD, Moraes RS, Bredemeier M & Duncan BB (1998): Alcohol intake and blood pressure: the importance of time elapsed since last drink. *J. Hypertens.* **16**, 175–180.
Muller CJ & Fugelsang KC (1994): Take two glasses of wine and see me in the morning. *Lancet* **343**, 1428–1429.
Ness AR & Powles JW (1997): Fruit and vegetables, and cardiovascular disease: a review. *Int. J. Epidemiol.* **26**, 1–13.
Owens CK, McIntire LV & Lasslo A (1990): Ethanol inhibition of thrombus formation on collagen- coated glass. *Thromb. Haemost.* **63**, 510–516.
Parker DR, McPhillips JB, Derby CA, Gans KM, Lasater TM & Carleton RA (1996): High-density-lipoprotein cholesterol and types of alcoholic beverages consumed among men and women. *Am. J. Public Health* **86**, 1022–1027.
Paunio MJV, Gref CG & Heinonen OP (1996): Serum high density lipoprotein cholesterol, alcohol, and coronary mortality in male smokers. *Br. Med. J.* **312**, 1200–1203.
Pellegrini N, Pareti FI, Stabile F, Bursamolino A & Simonetti P (1996): Effects of moderate consumption of red wine on platelet aggregation and haemostatic variables in healthy volunteers. *Eur. J. Clin. Invest.* **50**, 209–213.
Rakic V, Puddley IB, Burke V, Dimmitt SB & Beilin LJ (1998): Influence of pattern of alcohol intake on blood pressure in regular drinkers: a controlled trial. *J. Hypertens.* **16**, 277–285.
Renaud S & De Lorgeril M (1992): Wine, alcohol, platelets and the French paradox for coronary heart disease. *Lancet* **339**, 1523–1526.
Rimm EB, Giovannucci EL, Willett WC, Colditz GA, Ascherio A, Rosner B & Stampfer MJ (1991): Prospective study of alcohol consumption and risk of coronary heart disease in men. *Lancet* **338**, 464–468.
Rimm EB, Klatsky A, Grobbee D & Stampfer MJ (1996): Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits? *Br. Med. J.* **312**, 731–736.
Rosenberg L, Slone D, Shapiro S, Kaufman DW, Miettinen OS & Stolley PD (1981): Alcoholic beverages and myocardial infarction in young women. *Am. J. Public Health* **71**, 82–85.
Ruidavets JB, Cambou JP, Arveiler D, Nuttens MC, Bingham A, Richard JL, Salomez JL & Ducimetière P (1993): Différences inter-régionales de consommation alimentaire: project MONICA. *Cah. Nutr. Diét.* **28**, 105–109.
Sans S, Kesteloot H, Kromhout D & on behalf of the Task Force (1997): The burden of cardiovascular diseases mortality in Europe. *Eur. Heart J.* **18**, 1231–1348.
Serafini M, Ghiselli A & Ferro-Luzzi A (1994): Red wine, tea, and antioxidants. *Lancet* **344**, 626.
Simpura J, Paakkanen P & Mustonen H (1995): New beverages, new drinking contexts? Signs of modernization in Finnish drinking habits from 1984 to 1992, compared with trends in the European Community. *Addiction* **90**, 673–683.

St Leger AS, Cochrane AL & Moore F (1979): Factors associated with cardiac mortality in developed countries with particular reference to the consumption of wine. *Lancet* **1**, 1017–1020.

Sumi H, Hamada H, Tsushima H & Mihara H (1988): Urokinase-like plasminogen activator increased in plasma after alcohol drinking. *Alcohol Alcohol.* **23**, 33–43.

Tjønneland A, Grønbæk M, Stripp C & Overvad K (1999): Wine intake and diet in a random sample of 48763 Danish men and women. *Am. J. Clin. Nutr.* **69**, 49–54.

Trichopoulou A and Lagiou P (1997): Healthy traditional mediterranean diet: an expression of culture, history, and lifestyle. *Nutr. Rev.* **55**, 383–389.

Tunstall-Pedoe H, Woodward M, Tavendale R, A'Brook R & McCluskey MK (1997): Comparison of the prediction by 27 different factors of coronary heart disease and death in men and women of the Scottish heart health study: cohort study. *Br. Med. J.* **315**, 722–729.

Welsh SO & Marston RM (1982): Review of trends in food use in the United States, 1090 to 1980. *J. Am. Diet. Assoc.* **81**, 120–128.

Westin S (1995): Mortality associated with wines, beers, and spirits. Study confounded by lack of correction for social class. *Br. Med. J.* **311**, 1167.

Yano K, Rhoads GG & Kagan A (1977): Coffee, alcohol, and risk of coronary heart disease among Japanese men living in Hawaii. *New Engl. J. Med.* **297**, 405–409.