Familial clustering of adult mortality risk in Russia: the role of education, smoking and alcohol

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Abstract

Mortality increased substantially since 1991 among Russian working age men. From a 2002 nationally representative survey in the Russian Federation, we investigated the extent to which adult mortality clusters within sibships, and how far the distribution of high-risk behaviours, such as heavy alcohol and tobacco consumption, explains mortality clustering (or 'frailty') within family groups using information about the vital status, childhood conditions and socio-economic characteristics of sibs, reported by one family member, a design widely used in developing, but rarely in developed country demographic studies. We undertook a Cox proportional hazard gamma-frailty model analysis of the siblings (N=6,716) of survey informants. The effects of risk factors on mortality were similar as other studies. The overall clustering effects were much larger for men (theta=0.48) than for women (theta=0.22) but this excess was largely explained by measured socio-economic and behavioural risk factors; once these are accounted for, the magnitude of clustering among male and female sibs was similar. Smoking, drinking and, to a lesser extent, education made a substantial contribution to clustering of mortality in male sibs but unmeasured background familial factors remain important and are of a similar magnitude for men and women. While there was a positive association between risk factors among sibs, controlling for the characteristics of the informant sibs did little to account for the magnitude of clustering.

Keywords

Adult; Family Characteristics; Mortality; Russia

INTRODUCTION

There is evidence of familial clustering for mortality since the nineteenth century. Karl Pearson analyzed the correlations of parent/child and siblings' ages at death using information on members of the English Society of Friends and of the Friends' Provident Association and concluded that mortality of relatives is strongly correlated (Beeton and Pearson, 1899). This has been confirmed by later studies (Wyshak 1978; Carmelli, 1982; Gavrilov, Gavrilova, Olshansky, Carnes, 2002; Garibotti, Smith, Kerber, Boucher, 2006).

Over the last 15 years, Russia has been undergoing an unprecedented mortality crisis (Cornia, Paniccia, 2000; Brainerd, Cutler, 2004.). During the societal transformation that followed the fall of communism, Russian economic indicators, such as per capita income, fell sharply; life expectancy decreased dramatically between 1990 and 1994 and after a short recovery between 1995 and 1998 it has started to decline once more and life expectancy for men was still below 60 years in 2005 (Table 1).

Insert table 1

A number of studies have shown the importance of socio-economic factors -- such as education -- and health behaviours -- such as alcohol consumption and smoking -- on mortality in Russia (Notzon, Komarov, Savinykh, Hanson, Albertorio, 2003; Shkolnikov, Andreev, Leon, McKee, Meslé, Vallin, 2004). Such factors tend to cluster within families (Gleiberman, Harburg, Di Franceisco, Schork, 1991; Duncan, Duncan, Hops, 1996; D'Amico, Fromme, 1997; Bierut, Dinwiddie, Begleiter, Crowe, Hesselbrock, Nurnberger, Porjesz, Schuckit, Reich, 1998; Van Gundy, 2002; Nurnberger, Wiegand, Bucholz, O'Connor, Meyer, Reich, Rice, Schuckit, King, Petti, Bierut, Hinrichs, Kuperman, Hesselbrock, Porjesz, 2005), and would therefore be likely to lead to clustering of mortality within kin groups, but the extent and explanations for familial clustering in the context of the high and sharply increased adult mortality in Russia since 1990 remains unexplored.

In this paper, we focus on adult mortality from all causes, and we address four main questions. First, what is the magnitude of mortality clustering among adult Russians. Second, how far such clustering can be explained by similarities in socio-economic and lifestyle factors within families. Third, whether clustering varies between men and women. Finally, we also investigated how far information on the characteristics of an informant can be used as proxy information for other kin members.

DATA AND METHODS

Subjects

We conducted a cross-sectional survey of a national sample of the Russian population, conducted in 3 waves in July, September and November 2002. The data were collected in collaboration with the Russian Centre for Public Opinion Research (VCIOM), under the direction of Professor Levada, and the New Russian Barometer survey program (The Centre for the Study of Public Policy, n.d.). The population sample was selected in a multi-stage process. The whole Russian Federation was first stratified into 22 regions; each region was further stratified into urban and rural areas and towns and settlements were randomly selected proportionately to population size. Within these locations, primary sampling units were randomly drawn and within these units, an address was randomly selected, and interviewers were instructed to seek a face-to-face interview with one eligible respondent at every n-th household. 11,776 households containing an eligible respondent were identified, and 7,172 informants provided information about their parents, eldest two siblings and first husbands (overall response rate 61 percent). Such a design borrows from demographers' indirect estimation methodology to estimate mortality in countries without vital statistics. A number of these indirect demographic methods using survey or census data, often called "Brass techniques" (Brass et al, 1968; Hill, Trussell, 1977), are used to estimate mortality from information on the survival of close kin (such as spouses and parents) where conventional data are unavailable. These methods use simple information on the number of close kin and on how many of them have died. We modified this method for literate and numerate populations, and showed that the method, based on spouses and siblings, is a useful tool to study mortality and its individual level determinants in Russia (Bobak et al, 2002, 2003; Murphy et al, 2006). The approach is quick and cheap but effective approach to assess levels and predictors of mortality in a population, especially in the application here which is concerned with clustering of mortality for which routine data sources are inadequate. Although the design is unusual in epidemiological contexts, the records on eldest siblings' vital status and their probabilities of death by certain ages are essentially un-influenced by the survival of respondents (we discuss deviations form this conclusion below). The information collected for a given eldest sibling by this method would be exactly the same as that obtained from any other valid data collection system. On the other hand, selection of a particular subject (eldest sibling) into the sample can be affected by several mechanisms. First, the probability of a person being included in the sample is proportional to the number of living siblings so that those with a large number of siblings are overrepresented (and no information is collected on only-children). However, we found that sibling group size effects

are very small and therefore we report unweighted data; the lack of relation with sibling group size leads us to infer that the exclusion of only-children does not bias the estimates, although this is not certain. The second issue is whether there are biases resulting from the fact that the eldest sibling is usually either the first or second birth. There are birth-order effects on infant and child mortality. However, we have no evidence that there are substantial birth-order effects among adults. Finally, because reports are available only from surviving respondents, correlated mortality between siblings would lead to a downward bias. We did simulations, which suggested that the effects on the estimated level of mortality are small, and in any case, our main interest is in differentials among risk categories as measured by relative risks.

This analysis is restricted to mortality of sibs only, who therefore form the group of subjects for our study, but using information provided by the survey informant; informant sibs were not included in the analyses since to do so would generate biases in estimates. While we could have included parents, less information was available about these, and we excluded husbands since we wanted to include information on childhood circumstances which was collected only about the informant's childhood circumstances (which we assume are similar to those of her or his sibs).

Measurements

Informants answered questions concerning their own age, sex, socio-economic characteristics, and social and political attitudes, childhood circumstances at age 15, such as whether hungry and availability of kitchen and toilet, and family size together with information on their sibs, including year of birth, whether they were alive or dead, and, if applicable, year and cause of death, together with details on lifestyle of siblings aged 20 years and older including the frequency of drinking vodka or other strong spirits (data on the consumption of other alcoholic beverages were not collected due to the predominance of vodka and spirit consumption in Russian drinking behaviour), smoking, highest education level, marital status and frequency of contact with informant (Bobak, Murphy, Pikhart, Martikainen, Rose, Marmot, 2002; Bobak, Murphy, Rose, Marmot, 2003; Nicholson, Bobak, Murphy, Rose, Marmot, 2005).

Statistical analyses

We fitted a series of models of increasing complexity to investigate how far patterns of risk behaviour are correlated within kin groups; how these influence mortality; how mortality clusters within families; and how far information from one person, the informant, can be used

to explain this pattern. Since we were interested in the determinants of adult mortality, only sibs who had reached 20 years of age were included in these analyses.

The study design means that the data are clustered, with one informant potentially giving information on up to two sibs. We use a Cox model with right-censored and left-truncated data, but in addition, we assume that the hazard function also depends on an unobserved random variable ('frailty') which acts multiplicatively on the hazard and is the same for all sibs in the family unit, so that a large value of this shared frailty variable increases the mortality hazard as follows:

$\lambda_0(t| Z_i) = Z_i \lambda_0 (t) \exp (\beta' X_{ij})$

where $\lambda_0(t)$ is the baseline hazard function; X_{ij} is the covariate vector for the j^{th} sib in sibship i, and β' is the corresponding vector of regression parameters. We use maximum penalized likelihood estimation in a gamma-frailty model (Rondeau, Commenges, Joly, 2003). The frailty Z_i is assumed to be independently and identically distributed from a gamma distribution with mean one and unknown variance theta. Large values of theta signify a stronger positive relationship between sibs (i.e. higher degree of clustering of mortality) and greater heterogeneity between the family groups. We assume the censoring times to be independent of the failure times and of the frailties.

We calculated the Cox proportional hazard ratios (relative risks) to assess the effect of sibs' characteristics on their own risk of death from all causes. The proportional hazards assumptions were fulfilled, and the regression coefficients with and without the frailty term were very similar.

Since we relied on survey informants' reports about their relatives and we were particularly concerned with clustering within families, our models used cases where full information on all covariates used, including vital status and age, were available for sibs (i.e. the study subjects) and informants. This means that the same number of cases was used in all models, and therefore likelihood ratio tests were valid. There were 7,444 sib subjects aged 20 and over, but 728 were rejected due to missing or incomplete information on one or more covariates used (mainly relating to smoking and drinking patterns of sibs). The informant is not included as a subject in these models since they are by definition selected for being alive and to do so would otherwise lead to biases in the results (Trussell, Rodriguez, 1990; Murphy, Bobak, Nicholson, Rose, Marmot, 2006). We included sex of subject (i.e. the sib), since mortality levels differ between men and women, and birth cohort of the subject, since if

cohort effects exist, sibs who are likely to be born relatively close together would have correlated mortality experience as a result of their being from neighbouring cohorts (although we find that this cohort effect was weak and it did not affect our results).

The other covariates included were: (a) frequency of drinking, with non- or rare drinkers as the (low-risk) reference group; (b) frequency of smoking, with never or former smokers as the (low-risk) reference group; (c) education, with higher education as the (low-risk) reference group; and (d) informant's reported circumstances at age 15 as indicator of childhood circumstances for the whole sibship. We examined a number of indicators of childhood circumstances, such as father's smoking and drinking, both because they may be directly relevant to childhood circumstances, but also may be associated with similar patterns of behaviour among children. However, reported childhood difficulties at age 15 based on the question "When you were a child were things so difficult that you sometimes went to bed hungry?" provided the best indicator of early life conditions (Nicholson, Bobak, Murphy, Rose, Marmot, 2005), in part because a substantial fraction of information on fathers' smoking and drinking was missing (details available on request). Finally, we included sex of informant as a covariate since women tend to report more completely than men (Poulain, Riandey , Firdion, 1991; White, 1998). However, we note that since older people are more likely to have dead sibs than younger people, and that there are substantially more older women than men alive, the distributions of deaths reported by male and female informants differ so we do not present estimates of clustering of mortality within sibships according to sex of informant.

In total, 4,708 informants provided full information on themselves and on 6,716 of their sibs who had reached at least age 20 years, of which 992 cases were of two brothers, 996 of two sisters, 2,028 of mixed pairs and 2,700 of an only sib. Since we are interested in whether clustering is more important for males or females, we analysed male-male and female-female pairs separately. We note that information on two sibs was available only for sibships of at least size three (since the informant is in addition to the sib pair of subjects). Only sibs do not contribute to assessing familial clustering, but they were included to facilitate comparisons of our Cox model coefficients with other studies.

RESULTS

Basic descriptive statistics are given in table 2. Almost two thirds of the informants were female, in part due to higher response rates for women and more surviving women at older ages. Among informants' siblings, however, the numbers of men and women were similar.

The average year of birth was later among informants than their siblings, since information was requested on older sibs and because older informants were more likely to report on two sibs which were both included in the siblings distribution, whereas they were included only once in the informants' distribution. Siblings had somewhat lower education and higher proportion of smokers and drinkers than informants, but the differing sex and age composition of the two populations partially explains these differences.

Insert table 2

Table 3 shows the effect of four major factors on individual-level mortality risk, education, drinking, smoking and living conditions at age 15. We present results for each of the three sibship types of the subjects and an overall model including all subjects (i.e. also including only a single sib). The mixed and all sibship models are adjusted for sex of subject and all models include adjustment for birth cohort, separately for each sex of sib where appropriate.

Insert table 3

Although the standard errors are large in some cases, the coefficients are in the expected direction with clearly higher relative risks associated with drinking, smoking and lower educational level. Gradients are similar in magnitude for male and female sibships (although considerably fewer women are in the higher risk groups for smoking and drinking, so the population attributable risk is lower for women). The effect of poor childhood circumstances is to increase adult mortality risk especially for males but the coefficient is not statistically significant at 5 percent level (although close to in all models including male subjects).

Table 4 shows the theta frailty parameter (i.e. the variance) of the gamma distribution among members of the kin group, showing the degree of clustering within sibships obtained from the same set of models shown in table 3, together with values obtained from a model including only age and sex, and one where the characteristics of the informant rather than the corresponding value for the subject was used in the regression (this is discussed later). Although cases with only one sibling do not contribute to the estimation of clustering, the values obtained by confining the model to the 4,016 cases of sib pairs were only trivially different from those in the final column. The gamma distribution in the full sample of siblings had a theta parameter of 0.41 if no socio-economic covariates are included, and by definition, the value averaged all individuals was one; however, members of some sibships will have relative risks of mortality above this average value, and some below it with the spread of values given by the theta parameter. To illustrate the magnitude of estimated

family clustering of mortality, if the population is divided into four quartiles according to the (unobserved) level of risk, when compared with the overall value of 1.0, the average relative risk of a person in the top quartile of risk is 1.88, which is 5.5 times that of a person in the bottom quartile of risk, 0.34. The frailty parameter was considerably lower for sister-sister pairs than for the other three sibship types suggesting that familial factors are more important for males than for females. The inclusion of sibs' individual covariates from table 3 considerably reduced the frailty parameter in models that include males; for brother-brother pairs, the value dropped by 60 percent, from 0.48 to 0.20. There was virtually no change for sister-sister pairs so that the estimated frailty parameters after the inclusion of siblings' covariates were similar, between 0.20 and 0.30 in the four cases shown.

Insert table 4

At least part of this clustering seems to be due to members of a given family having similar characteristics to each other in terms of common backgrounds for both socio-economic factors and for behaviours; the characteristics of the informant (who is not included in the analysis) will reflect this common background and is sometimes used as a proxy for information on sibs when such information is unavailable. Therefore the final row of table 4 shows results obtained when the values of the informant for the variables of table 3 rather than those of the sib were used. We find that controlling for the characteristics of the informant does add marginally to the explanation of clustering of mortality, but rather little when compared to information on the subject.

In order to examine possible pathways of family influence, we use education as an example of a socio-economic factor, and smoking and drinking alcohol as examples of behaviours. For a variable to act through a familial pathway, we expect the characteristics of family members to be positively correlated, and to investigate this, we calculated partial rank correlations between adult siblings of the same sex (including cases where the pairs are both subjects or informant and subject to maximise the number of cases) controlling for birth cohort which were as follows: 0.06 in males and 0.02 in females for education; 0.26 in males and 0.23 in females for smoking; and 0.18 in males and 0.27 in females for alcohol consumption, when these variables were coded according to increasing levels of education and consumption of alcohol and tobacco. The correlation for educational level is small compared with the other two variables. This suggests that behaviours may be more important for familial clustering of mortality than education. However, in the final row of table 4 when using informants' data as proxies for that of subjects, about half the time, men will be reporting on their sisters or *vice versa*, but the sample numbers were too small to analyse

the preferred case of sibships where all three members were of the same sex. Therefore, to address the issue of how far information on informants provides useful information about their sibs, in order to maximise comparability in table 5, we fitted models to subsets of the data set where there were at least two members of the same sex among the maximum of the three sibs involved, i.e. only excluding cases where a subject had the same sex as neither the informant nor the other subject sib. If the informant and sib were of the same sex, the socio-economic and behavioural information of the informant was used in the regression, otherwise we used information of the other sib. Therefore in table 5 we show the estimated effect of the characteristics of a sib of the same sex as compared with the same information of the subject on proportional hazards ratios for mortality of males and females. Coefficients based on another sib's characteristics provided little useful information on risk factors for subjects' mortality, since the coefficients were generally closer to one than those based on subject's information, none were statistically significant and, in a number of cases, were not in the expected direction. One consequence of the lack of explanatory power of informant's information is that including it in addition to that of subjects provides no improvement in model fit (not shown).

Insert table 5

Table 6 shows the change in log likelihood for the models of table 5 when these variables were included in addition to basic age and sex variables (for each of the four populations, results are based on the same number of cases and since all variables are trichotomised, the degrees of freedom are the same so changes may be compared directly). The importance of both behavioural factors, especially for men, is clear cut and, in particular, they are much more important than education, whether considered as an individual or familial characteristic.

Insert table 6

DISCUSSION

We analysed data collected in this population based study in Russia, by approaches based on reports by relatives more commonly used in developed countries. The population information obtained is not completely representative of the whole population, for example it excludes only children (Gakidou, King, 2006), but other studies have shown that potential biases are unlikely to affect the interpretation of our results (Trussell, Rodriguez, 1990; Murphy, Bobak, Nicholson, Rose, Marmot, 2006). Data reported by informants on relatives

produced overall mortality estimates consistent with official statistics, and estimated effects of socioeconomic and behavioural characteristics such as education, smoking and alcohol consumption which were all in the expected direction (Cornia, Paniccia, 2000; Brainerd, Cutler, 2004.). A number of studies have shown the importance of socio-economic factors -- such as education -- and health behaviours -- such as alcohol consumption and smoking -- on mortality in Russia (Notzon, Komarov, Savinykh, Hanson, Albertorio, 2003; Shkolnikov, Andreev, Leon, McKee, Meslé, Vallin, 2004)) but it is also possible to investigate the extent of familial factors in a relatively cheap and quick way. However, potential limitations need to be considered since the findings are reliant on the accuracy of information provided by siblings. Frequent contact with siblings was associated with higher reported mortality in siblings, probably due to increased contact with sick relatives and to under-reporting of mortality in sibs with less contact. While the levels of alcohol and tobacco consumption appear consistent with other sources, and the results for respondents and sibs are similar, it is not possible to rule out that respondents might over-estimate consumption in dead relatives. However, these are unlikely to seriously bias the results and might be expected to reduce rather than to increase the estimated level of familial clustering.

We find that adult mortality was clustered within kin groups, especially among men. However, the magnitude of the estimated clustering effect was substantially reduced when educational level, smoking, drinking and childhood circumstances were included in the analysis. These factors explained a substantial fraction of the clustering among brothers, but they did not seem to contribute much to explaining clustering among sisters. We therefore conclude that clustering of these risk factors within sibships can explain a substantial part of the overall mortality clustering only among adult males. The main reason why women show lesser reductions after including these covariates is due to the fact that much smaller proportions of women smoke and drink compared with men: for example, 89 percent of female sibs were reported as non-smokers compared with 35 percent of male sibs, and the proportion of non- or rare drinkers was 94 percent compared with 65 percent. However, once these factors are controlled for, the remaining shared frailty values are similar for sibs, whether of the same or different sexes.

The remaining reasons for clustering of mortality risk includes a range of common genetic, cultural, geographic, ethnic and shared environmental factors which surveys such as this can only provide limited information upon. Nevertheless, the recognised importance of smoking and drinking for male mortality (which are more important than education or childhood circumstances in our models) is clear and family patterns of drinking and smoking are important mechanisms that account of familial clustering which leads to excess mortality

among men. The determinants of risky behaviour are rooted to a large extent in earlier experiences and so attempts to address such issues need to be targeted not only to individuals, but also to the wider family and societal relations they are embedded in.

While this study highlights the importance of familial factors, it also shows that care must be taken in the ways in which information about kin groups is incorporated. We compared results that included characteristics of the informant and sib since we were interested to examine how far variables such as education, smoking and drinking behaviour of one sib could act as proxy for the characteristics of another (usually) non-co resident sib. Although we found that both behaviours and outcomes were positively correlated within family units, we also found that information relating to another sib provided little useful information about the relative risks on socio-economic factors, even if the information used is about the next closest sib of the same sex. Graham and colleagues (Graham, Fitzmaurice, Bell, Cairns, 2004) used the living informant's poverty status to draw conclusions about her sisters' maternal mortality using Demographic and Health Survey (DHS) data for a number of developing countries under the assumption that they have the same poverty status as their informant sibling. This assumption was not tested, but in our application, such an assumption would lead to misleading conclusions, especially for the educational level variable, even though we use variables which are positively correlated within sibships, suggesting that very high correlations are needed before reliable results can be drawn if proxy data from relatives is used.

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TABLE 1. Official estimates of life expectancy at birth (from the Health for All database)

	Male	Female
1980	61.44	74.03
1981	61.69	73.30
1982	62.36	73.81
1983	62.26	73.58
1984	61.72	73.03
1985	62.75	73.32
1986	64.88	74.37
1987	64.93	74.39
1988	64.66	74.32
1989	64.23	74.59
1990	63.79	74.42
1991	63.44	74.31
1992	62.02	73.77
1993	58.91	71.88
1994	57.62	71.18
1995	58.30	71.71
1996	59.77	72.52
1997	61.02	72.96
1998	61.39	73.27
1999	59.98	72.49
2000	59.15	72.36
2001	59.08	72.28
2002	58.88	72.03
2003	58.68	71.89
2004	59.08	72.36
2005	58.98	72.40

Source: accessed from WHO European Office Health for All database http://hfadb.who.dk/hfa/ on 21 September 2007.

TABLE 2. Distribution (percentages) of the population of informants and siblings.

Variable	Category	Informants* (n=4,708)	Siblings (n=6,716)
Sex	Male	38	49
	Female	62	51
Year of birth	Before 1941	28	33
	1941-59	33	33
	1960-86	39	33
Drank†	Rarely/never	82	79
	Sometimes	15	15
	Often	3	6
Education	University	23	17
	Intermediate	58	64
	Elementary	19	19
Smoked‡	Former/never	67	62
	Sometimes	19	20
	Heavy	14	18
Difficulties at age 15	None/occasionally	93	92
	Yes	7	8
Survival status	Alive	100	81
	Dead	-	19

Notes * With at least one sib aged 20 years or over.
† Rarely/never is Never or Monthly or less; Sometimes is Fortnightly or Weekly; Often is Most Days or Daily
‡ Sometimes is Daily or Occasionally

TABLE 3. Cox proportional hazard ratios* for mortality of siblings by their characteristics, adjusted for age and sex of sib (where appropriate) and informant.

	Brothers (n=992)	Sisters (n=996)	Mixed (n=2,028)	All (n=6,716)
Drank				
Rarely/never	1.0	1.0	1.0	1.0
Sometimes	1.59 (1.16-2.17)	2.09 (0.81-5.35)	1.01 (0.74-1.38)	1.26 (1.06-1.50)
Often	1.40 (0.92-2.12)	1.55 (0.35-6.95)	2.06 (1.45-2.93)	1.70 (1.37-2.10)
Education				
University	1.0	1.0	1.0	1.0
Intermediate	1.53 (1.03-2.26)	1.53 (0.63-3.74)	1.22 (0.87-1.70)	1.15 (0.95-1.40)
Elementary	1.44 (0.90-2.32)	1.37 (0.51-3.65)	1.44 (0.97-2.12)	1.37 (1.10-1.71)
Smoked				
Former/never	1.0	1.0	1.0	1.0
Sometimes	1.52 (1.08-2.16)	1.58 (0.47-5.26)	1.97 (1.44-2.70)	1.78 (1.48-2.13)
Heavy	1.98 (1.42-2.77)	NA (-)	1.89 (1.36-2.63)	1.86 (1.54-2.24)
Difficulties at age 15				
None/occasionally	1.0	1.0	1.0	1.0
Yes	1.40 (0.97-2.01)	0.93 (0.57-1.52)	1.27 (0.96-1.68)	1.16 (0.98-1.37)

Notes: * using penalised gamma frailty Cox proportional hazards model
95% confidence intervals shown in brackets.

TABLE 4. Theta coefficients of frailty, indicating degree of clustering of mortality in families.

Controlling for:	Brothers (n=992)	Sisters (n=996)	Mixed (n=2,028)	All (n=6,716)
age and sex	0.48 (0.21)	0.22 (0.22)	0.48 (0.16)	0.41 (0.11)
age, sex and covariates of subject *	0.20 (0.18)	0.25 (0.23)	0.30 (0.14)	0.23 (0.09)
age, sex and covariates of informant†	0.41 (0.20)	0.20 (0.22)	0.46 (0.16)	0.40 (0.10)

Notes: all values adjusted for age and for sex in analyses of mixed sibling sets and in all subjects
Standard errors shown in brackets.
* variables as in Table 3
† variables as in Table 3 but relating to informant

TABLE 5. Cox proportional hazards of mortality of sibs based on information on subject or on same sex informant or other sib

	Male sibs (n=1,875)		Female sibs (n=2,421)	
	Subject	Informant/other sib	Subject	Informant/other sib
Drank				
Rarely/never	1	1	1	1
Sometimes	1.67 (1.31-2.12)	1.24 (0.97-1.59)	1.67 (0.89-3.12)	1.08 (0.53-2.22)
Often	1.94 (1.43-2.62)	1.24 (0.84-1.82)	1.84 (0.79-4.28)	1.23 (0.61-2.46)
Education				
University	1	1	1	1
Intermediate	1.14 (0.83-1.56)	1.22 (0.89-1.67)	1.09 (0.69-1.71)	0.79 (0.57-1.10)
Elementary	1.31 (0.90-1.92)	1.10 (0.77-1.58)	1.09 (0.65-1.81)	0.87 (0.62-1.22)
Smoked				
Former/never	1	1	1	1
Sometimes	1.71 (1.31-2.24)	0.88 (0.67-1.17)	1.84 (0.95-3.56)	1.27 (0.77-2.08)
Heavy	2.04 (1.58-2.63)	1.29 (1.00-1.66)	1.67 (0.67-4.17)	1.84 (0.85-3.99)

Notes: 95% confidence intervals shown in brackets.
All estimates adjusted for age of subject

TABLE 6. Change in log likelihood for Cox proportional hazards of mortality based on information on subject or same sex informant or other sib

	Male sibs (n=1,875)		Female sibs (n=2,421)	
	Subject	Informant/ other sib	Subject	Informant/ other sib
Drank	13.2*	1.7	2.0	0.2
Education	1.0	0.9	0.1	1.0
Smoked	16.2**	3.6	1.9	1.4

Note: All estimates adjusted for age of subject
* P<0.01; **P<0.001