An empirical analysis of the relationship between the consumption of alcohol and liver cirrhosis mortality

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Abstract

The question whether intake of alcohol is associated with liver cirrhosis mortality is analyzed using aggregate data for alcohol consumption, alcohol related diseases and alcohol policies of 16 European countries. The empirical analysis gives support to a close association between cirrhosis mortality and intake of alcohol – and the latter also concerns each of the specific beverages, i.e. spirits, wine and beer, where other studies usually only find evidence of spirits and wine related to liver cirrhosis mortality.

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1 Introduction

Since the 1960s wine consumption has decreased dramatically, especially in the Southern European countries whereas the countries in the northern parts of Europe have experienced a substitution from beer and spirits toward wines. In this sense there has been a process of convergence in per capita consumption of wine among the European countries. Also for the total consumption of alcohol, i.e. the per capita consumption of beer, wine and spirits, a hypothesis of convergence seems to hold but generally, the aggregate level of alcohol consumption has been increasing in many European countries.

In the same time span the number of alcohol related diseases like e.g. liver diseases, have changed significantly and in the same direction as the developments in alcohol consumption and, naturally, this has raised concerns about health implications.

It's a well-established fact that especially high levels of alcohol intake are causing a number of health related diseases, and among those liver cirrhosis, which is also the focus in the present analysis as an indicator of alcohol-induced detrimental health effects. From medical literature it is claimed that a moderate level of alcohol consumption may even improve health conditions, primarily with positive effects for heart diseases, assuming the level of consumption is kept at a few drinks daily but a higher level of alcohol intake will imply negative net effects and thus, there is a so-called J-curve effect in relation to the health consequences of alcohol intake, de Lorimier (2000). But in most countries the level of alcohol consumption has exceeded just a few drinks, which may be healthy and thus health authorities in many countries come up with 'advisory guidelines', e.g. the weekly consumption is advised in the UK not to exceed 21 drinks (for men, less for women), as the negative effects seem to far outweigh the beneficial effects of population drinking.

2 Literature review and empirical findings

Numerous studies address the health related problems caused by the consumption of alcohol and there is no doubt about the clinical evidence concerning the harmful consequences from heavy drinking. Most of the empirical papers rely on individual micro data as an obvious point of departure concerning the choice of data-information. In these studies it is argued that causes are most easily detected by using person-specific information on both the alcohol intake as well as the health conditions (liver cirrhosis and alcohol related deceases). But also from aggregate data the alcohol-health connection has been detected in a number of empirical studies. Moreover it is suggested that the variation in the average level of alcohol

consumption also reflects the alcohol intake in the group of individual with a high-risk level of e.g. liver cirrhosis. The latter corresponds to the so-called 'collectivity theory of drinking cultures' saying that heavy drinkers consume in some social context and are not confined to isolated 'drinking environments' – otherwise, cirrhosis mortality should not be associated with the overall level of alcohol consumption in the population. An advantage associated with studies using aggregate data is that the policy-relevant effect – the impact from alcohol consumption to health or mortality effects – is estimated and thus quantified, Norström and Ramstedt (2005).

From a methodological or econometric point of view aggregate studies have used various models and estimation techniques of course depending on the nature of data used. Most studies use either cross-section or time-series data and the conclusions are similar in almost all cases, i.e. significant effects of harmful health consequences from consumption of alcohol are found. Using time series data covering fourteen European countries Ramstedt (2001) finds a significant effect on liver cirrhosis mortality rates and with a similar kind of data, Mills (2007) reaches the same conclusion for the UK. Norström and Ramstedt (2005) give a review of the empirical findings on the harmful effects of alcohol consumption. Furthermore they find that for fourteen Western European countries an increase in per capita alcohol consumption was found to be significantly related to liver cirrhosis – with the strongest effects in Northern Europe. A similar conclusion was reached for Canada as well as the individual provinces. For the USA only spirits were found to have a cirrhosis impact in one study. Another analysis, Kerr et al. (2000), including the USA in a panel data set with five English-speaking countries (USA, Canada, New Zealand, Australia, UK) finds liver cirrhosis to be associated with both the overall level of drinking and spirits consumption. By now, there is an overwhelming number of studies concerning the alcohol-cirrhosis connection and additional to the above-mentioned papers an overview of methodologies and empirical findings can be found in Kerr et al. (2000), Nordström and Skog (2001), Rehm and Gmel (2001), Room et al. (2005), Antoñanzas et al. (2008).

Concerning the beverage-specific relations concerning cirrhosis the results from numerous studies differ relatively much where some studies claim wine and spirits are both linked to the harmful effects and other studies only finding one of the beverages most likely to cause cirrhosis; for a survey see Kerr et al. (2000). A very thorough, recent study using time-series data, Mills (2007), finds wine to be associated with cirrhosis in the UK, and with beer and spirits being of only minor importance. Heavy consumption of all three kinds of alcoholic beverages may cause liver cirrhosis and thus, there should be no beverage-specific effects – unless the drinking patterns of the beverages are differing and therefore causing

the adverse health effects. The latter may be the case where spirits is primarily consumed by (elderly) men and without the intake of food, where wine is much more likely to be enjoyed to regular meals.

At the individual level cirrhosis mortality is usually caused by heavy drinking during a number of years. Naturally, this will differ from case to case with a lot of individual variation, where e.g. women have a higher risk of developing liver cirrhosis for a given level of alcohol intake. Despite this gender-specific difference almost ninety per cent of the cirrhosis mortality cases are due to the male population. As discussed in Kerr et al. (2000) and Norström (1989), Norström and Ramstedt (2005) many of the studies using aggregate data often end up with an empirical specification of short lags from alcohol consumption to liver cirrhosis which appear inconsistent with the usually long-developing process of liver cirrhosis at the individual level. Therefore, the present study will also focus on the question of beverage-specific cirrhosis mortality effects as well as the question of using (long) lags of the alcohol consumption variable in the empirical estimations.

3   Data and modelling the alcohol-health relationship

In line with other studies data used in the present analysis are collected from the WHO Statistical Information System (WHOSIS, www.who.int). The liver cirrhosis mortality is measured as the number of deceases per 100.000 persons in the total population and the alcohol consumption variables measured in litres of pure alcohol per capita (+15 years). The data for alcohol consumption also include the specific beverages and cover the period 1963 to 2003, and the data set has been extended back in time to 1961 using data from World Drink Trends (1999) in order to have a pair of decades of alcohol consumption data prior to 1980 (the cirrhosis mortality data are available from the late 1970's) in order to include long lags in the model. The data for liver cirrhosis generally cover the period 1977 to 2003 (with some variation because of missing values) for sixteen European countries: Austria, Belgium, Denmark, Finland, France, Greece, Germany, Ireland, Italy, the Netherlands, Norway, Portugal, Switzerland, Spain, Sweden and the UK.

A balanced panel data set including these sixteen members are constructed with the time dimension represented by three periods, 1980, 1990 and 2000. This allows for using a lag structure of the recent ten years without overlapping use of information in the explanatory alcohol variable. This only leaves 48 time-country observations for the estimations, and therefore an unbalanced panel data set covering all the available data during the period from 1977 to 2003 are also included in the analysis – which results in a reasonable number of observations (409). Having the two panel data sets the robustness of the estimation results

as a consequence of choosing data can be evaluated. Data are presented in table 1 and figure 1 shows examples of disaggregated data for selected countries. The following notation is used in the analysis:

A, average consumption of pure alcohol, litres per capita (+ 15 years) annually, and **A10** is the average level representing the most recent decade (i.e. 10 years). A similar notation is applied in the case of beer (B, B10), wine (W, W10) and spirits (S, S10). Sources: The WHO and World Drink Trends (1999, 2005).

LC: annual mortality rates of liver cirrhosis (per 100.000 inhabitants), Source: The World Health Organization (the WHOSIS database).

POL: a measure of the 'restrictiveness' alcohol policies, measured on a 1–20 scale, and constructed from a number of variables measuring separate dimensions of alcohol policies (an increasing value indicates a more restrictive alcohol policy). Source: Karlsson and Österberg (2001).

Y: real per capita income (in constant PPP-$). Source: The Penn World Tables (version 6.2).

Table 1: Liver cirrhosis, alcohol consumption and alcohol policy in Europe

	1980			2000		
	LC	A	POL	LC	A	POL
Austria	29.2	13.8	6.0	19.7	11.1	7.0
Belgium	12.9	13.5	8.5	11.8	10.3	11.5
Denmark	11.4	11.5	7.0	14.0	11.7	8.5
Finland	6.8	7.9	18.5	11.5	8.6	14.5
France	28.9	19.2	9.5	13.4	12.9	12.5
Germany	-	14.0	6.0	17.5	12.4	8.0
Greece	10.6	13.2	2.0	5.0	9.4	7.0
Ireland	4.7	10.5	12.0	4.3	13.7	12.0
Italy	32.9	16.7	12.0	14.1	9.0	13.0
Netherlands	5.1	11.5	11.0	5.1	10.1	13.0
Norway	5.8	5.9	19.0	5.0	6.3	17.0
Portugal	33.2	14.9	4.0	15.7	13.0	8.0
Spain	24.2	18.5	4.5	10.8	11.5	10.0
Sweden	11.2	7.1	18.5	5.2	6.0	16.5
Switzerland	12.6	13.4	-	7.5	11.1	-
UK	4.6	9.6	14.0	9.7	10.4	13.0
Average	15.6	12.6	10.2	10.6	10.5	11.4
Std.err.	*(2.80)*	*(0.95)*	*(1.42)*	*(1.23)*	*(0.56)*	*(0.84)*

Sources: The World Health Organization (the WHOSIS database), World Drink Trends (1999, 2005), Karlsson and Österberg (2001).

Table 1 exhibits a clear decline in the average level of liver cirrhosis mortality from 1980 to 2000 and there appears to be a rather close correlation between cirrhosis and the intake of alcohol. For the strictness of the alcohol policy in

Europe there seems to be some convergence with approximately the same average value of the policy-variable, but with a decreasing standard error – similar to the decline in the variation of the cirrhosis and alcohol consumption levels.

Figure 1: Liver cirrhosis and wine consumption in France and the UK, 1977-2003/04 (log values of indices)
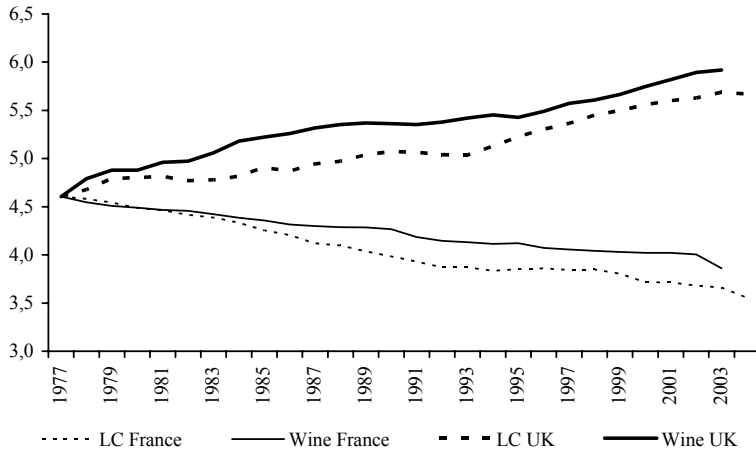
Source: The WHOSIS database (www.WHO.int).

As mentioned before, most of the papers dealing with the estimation of the cirrhosis-alcohol relationship set up an empirical model including only these two variables – which figure 1 will also indicate will work well, but when setting up complete (theoretical) models other variables should be included controlling for unobserved heterogeneity. The most important factors entering a complete model are shown in (1), (2) and (3):

$$A = f(Y, Prices of alcohol, POL) \tag{1}$$

$$POL = f(A, LC) \tag{2}$$

$$LC = f(A, Y) \tag{3}$$

The first relationship is from standard economic theory prediction alcohol demand influenced by income and prices – and also by the alcohol policies, i.e.

whether a liberal or restrictive alcohol policy is preferred by the authorities. Simultaneously, the alcohol policy is influenced by the general level of alcohol consumption – assuming this is associated with harmful social and health effects – and of course, the direct observable health consequences like e.g. mortality from liver cirrhosis. Finally, the latter is to a high degree determined by the intake of alcohol, although cirrhosis may also be caused by other factors – just as alcohol may cause additional health problems besides cirrhosis mortality, cf. Rehm et al. (2003), Nordström and Ramstedt (2005) and Day (2006). The level of real income might also be expected to influence the liver cirrhosis mortality where increasing wealth is associated with a better health system and therefore reducing mortality from cirrhoses, e.g. due to liver transplants.

A model based on (1), (2) and (3) will be rather demanding with respect to data requirements – besides the econometric problems related to endogeneity, i.e. the difficulties in finding valid instruments. Therefore a partial relationship of (3) is assumed appropriate for estimation purposes:

$$Log(LC_{it}) = \alpha + \beta A_{it} + \delta Y_{it} + \varepsilon_{it} \qquad (4)$$

In the empirical literature a semi-log relationship is assumed, based on a convex function between the risk of liver cirrhosis and alcohol consumption, cf. Ramstedt (2001) and Mills (2007), and only the present level of alcohol consumption is usually included as an explanatory variable. The latter may seem contradictory to *a priory* expectations as it takes years to develop a serious liver cirrhosis, enough to also imply mortality. Therefore, the average level of alcohol consumption during the last decade (the A10 variable) is included as an alternative explanatory variable[2]. The income variable appearing in (4) was found to be close to zero and very insignificant in all cases which is why this variable is excluded in the empirical results presented.

Figure 2 and figure 3 give scatter plots of the data for the balanced data set for the sixteen countries (the time dimension being 1980, 1990 and 2000) and a rather close correlation appears between cirrhosis and alcohol consumption – quite in line with the pure time series data in figure 1. A closer inspection of the graph does indicate that the decade-long average alcohol consumption (A10) in figure 3 does seem to correlate most closely to the liver cirrhosis variable – which is also more consistent with expectations.

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[2]Also longer lags have been investigated for and models with decreasing weights for lagged alcohol consumption have been included in the estimations – but the results do not differ much from the obtained results when using the average level of alcohol consumption during the most recent decade (A10).

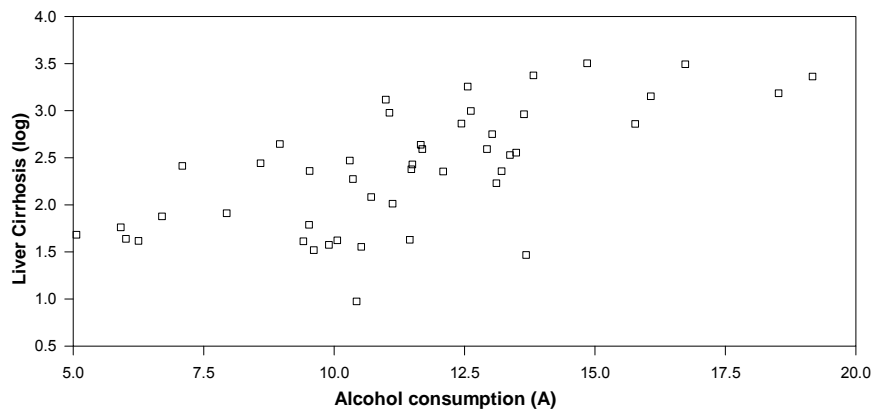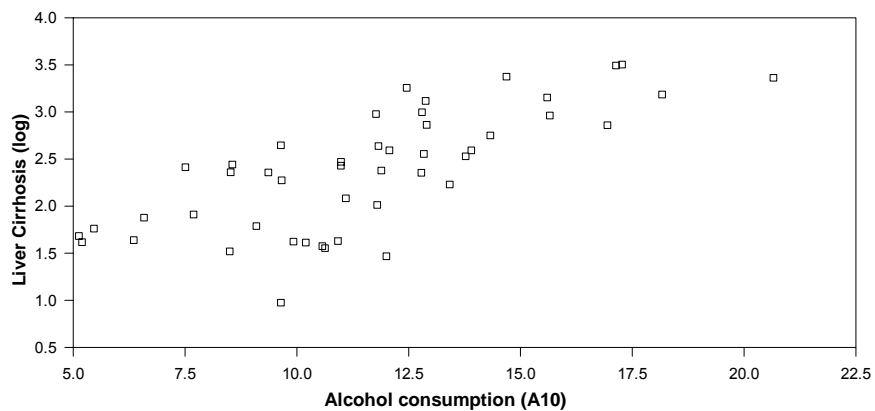Figure 2: Plot of liver cirrhosis (log values) and alcohol consumption (A)

Figure 3: Plot of liver cirrhosis (log values) and alcohol consumption (A10)

Suggestion :in these two figures and the following, is it possible to distinguish the three periods by using 3 different symbols ? Software Excel permits such a presentation. This procedure can give an interesting view of the phenomena when panel data are used.

The alcohol policies in Europe have traditionally been more restrictive in the northern countries, cf. table 1, and there is a clear, inverse relationship between the 'strictness' of the policy and the level of alcohol consumption as exhibited in figure 4.

Figure 4: Plot of alcohol policy (POL) and alcohol consumption (A)
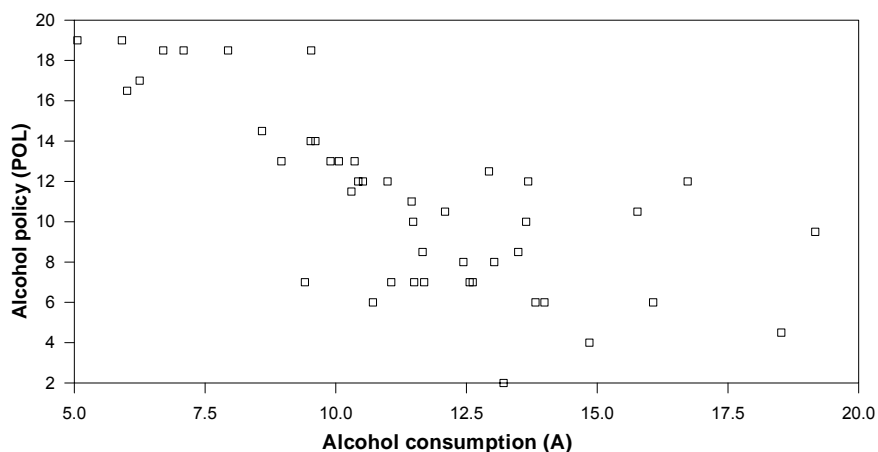
The causal link between alcohol policy and alcohol consumption is most likely bi-directional and also reflecting e.g. the national differences with a less restrictive policy in the wine-drinking countries in contrast to the spirits- and beer-drinking countries in Scandinavia, where the most restrictive regulations have been imposed since the beginning of the last century. With a bi-directional causation – and a lot of other factors influencing alcohol consumption – there is no direct empirical evidence saying that a strict alcohol policy has a positive influence in the sense of reducing cirrhosis mortality via a reduction in alcohol consumption.

An interesting conclusion in some studies is that cirrhosis mortality is highly related to the consumption of wine, e.g. Mills (2007) – or spirits in other studies, e.g. Kerr et al. (2000) and Stokkeland (2006), but beer is usually assumed to be less harmful. This result contradicts anecdotic knowledge among people and from time to time found in the press. Figure 5 gives a scatter plot of liver cirrhosis and the share of wine in total alcohol consumption – for the same data presented

in figure 2 and figure 3 - and the correlation coefficient is 0.72 between these two variables.

For a more complete information, give the correlation coefficient in the previous cases (fig 2 to 4)

Figure 5: Plot of liver cirrhosis (log values) and share of wine in total alcohol consumption
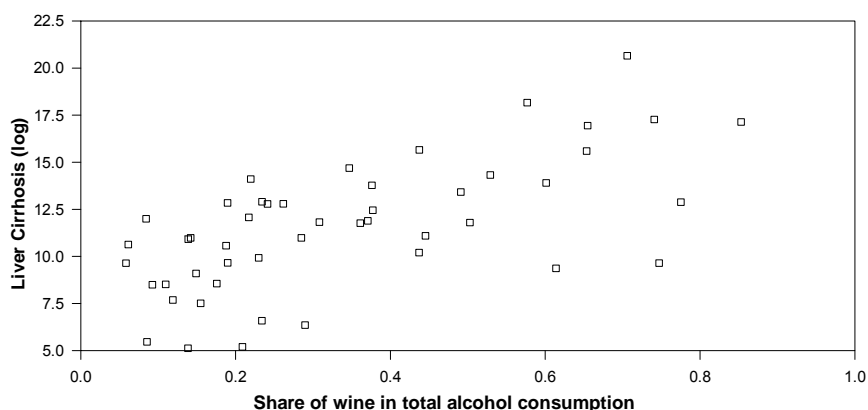
A high share of wine in the total alcohol consumption is found in Southern Europe (and mostly pronounced in the beginning of the period analyzed) but also for the recent years in some northern European countries where the overall level of alcohol has also increased – via increased wine consumption. Thus, a relatively high level of liver cirrhosis mortality is appearing simultaneously with a high share of wine in the total alcohol consumption. The interpretation that wine is causing liver cirrhosis may be erroneous in the sense that it is the alcohol content, not the drink *per se* that causes the detrimental health consequences. Therefore, all kinds of alcohol assumable should cause cirrhosis and other diseases, although the drinking habits (e.g. wine enjoyed with a meal) might also be of influence.

4 Estimating the relationship between alcohol consumption and liver cirrhosis

The two panel data sets presented in part 3 are used for the estimation of model (4) and in the econometric methodology follows the commonly used techniques in relation to panel data analysis. The data for the sixteen European countries are pooled and in the present case the β–parameter in (4) represents the impact from alcohol intake on cirrhosis mortality, which may be assumed identical across

countries. Note that the alcohol-parameter is not of a traditional behavioural character but to a large extent determined by medical causes – although there are individual differences concerning this 'impact parameter' – and no differences should be expected when using aggregate data. In order to investigate the robustness of estimating the equation (4), including the question of pooling the data for the sixteen countries, a battery of different econometric methodologies is applied. The parameter estimates are reported in table 2 – for both variables concerning alcohol consumption, i.e. A and A10 - and the income variable from (4) is deleted from the model as it showed up to be insignificant in all cases.

Table 2: Parameter estimates for the balanced panel data set (N=16, T=3)

Method	$\hat{\beta}(A)$	SSR	$\hat{\beta}(A10)$	SSR
OLS	0.137* *(0.023)*	10.29	0.135* *(0.019)*	8.56
Between	0.145* *(0.044)*	3.03	0.137* *(0.034)*	2.49
Fixed Effects (Within)	0.106* *(0.024)*	1.27	0.120* *(0.025)*	1.18
Random Effects	0.125* *(0.016)* [0.29]	1.37	0.136* *(0.017)* [0.39]	1.30
SUR	0.130 *(0.070)*		0.132* *(0.062)*	
Differences	0.105* *(0.022)*	1.94	0.133* *(0.023)*	1.64

Notes: SSR is the squared sum of residuals. Standard errors in parenthesis and indicated by * if significant at least the five per cent level. For the random effect estimates the *p*-values of a Hausman-test is reported in [ ]-parenthesis. SUR is the seemingly unrelated regression and 'Differences' is first differences of each individual series.

The results from table 2 reveal surprisingly identical parameter estimates no matter whether a simple OLS, a Between or Within estimator is applied – indicating that biased parameter estimates due to unobserved heterogeneity might not be a major problem. Also, using data in first differences – due to take care

of heterogeneity in the time dimension – does not differ much from the results from e.g. using the fixed effects estimator. For the random effects estimator the Hausman-test does not reject this specification of the error-structure of the model – consistent with the (nearly) identical parameter estimates from the Between and Within estimators. Treating the pooled panel data as a system of equations (SUR) results in parameter estimates identical to the before-mentioned techniques. Evaluated from the sum of residual squares (SSR) the fixed effects and random effects estimators seem to perform best and the alcohol consumption variable A10 (the ten year average level of alcohol intake) does also seem to fit the model marginally better than the present level of alcohol consumption (A).

The conclusion from table 2 is that alcohol consumption does have a significant influence on cirrhosis mortality and the values of the β-parameter (0.12 for the fixed effects estimator) is also in accordance with other studies, e.g. Ramstedt (2001) using pooled data in differences. With the intention of investigating the beverage-specific effects on mortality the procedure of using pooled data – estimated by fixed or random effects for a ten-year average level of alcohol intake – seems to be a reasonable methodology and the results are presented in table 3 and table 4 for the balanced and unbalanced data sets, respectively.

Table 3: Parameter estimates for the balanced panel data set (N=16, T=3)

	Fixed Effects		Random Effects	
$\hat{\beta}$: Alc.(A10)	0.120*	*(0.025)*	0.136*	*(0.017)*
SSR	1.18		1.30	
$\bar{R}^2$	0.90		0.93	
Hausman-test	-		0.73	[0.39]
$\hat{\beta}$: Beer (B10)	0.017	*(0.078)*	0.062	*(0.042)*
Wine (W10)	0.114*	*(0.027)*	0.135*	*(0.019)*
Spirits (S10)	0.138	*(0.106)*	0.113*	*(0.035)*
SSR	1.18		1.80	
$\bar{R}^2$	0.89		0.90	
Hausman-test	-		2.46	[0.48]

Notes: All variables included in the estimates represent an average value of the consumption levels during a ten-year period for total alcohol and the respective drinks (beer, wine and spirits). A constant is included in the RE estimations, and in parenthesis the standard errors of the parameter estimates are reported, with a * indicating significance at least the five per cent level. For the RE models a Hausman specification test is reported with *p*-values in parenthesis.

The conclusions from table 3 and table 4 are very similar in the sense that the level of the parameter estimates does not differ much – although the data sets differ in size – and that both wine and spirits significantly influence on cirrhosis

Table 4: Parameter estimates for the complete (unbalanced) panel data set (N=16, T=25)

	Fixed Effects		Random Effects	
$\hat{\beta}$: Alc. (A10)	0.119*	*(0.008)*	0.128*	*(0.006)*
SSR	11.34		12.64	
$\bar{R}^2$	0.93		0.93	
Hausman-test	-		3.38	[0.07]
$\hat{\beta}$: Beer (B10)	0.045	*(0.024)*	0.043*	*(0.020)*
Wine (W10)	0.120*	*(0.008)*	0.122*	*(0.007)*
Spirits (S10)	0.115*	*(0.028)*	0.109*	*(0.016)*
SSR	10.72		13.75	
$\bar{R}^2$	0.93		0.92	
Hausman-test	-		1.15	[0.76]

Notes: All variables included in the estimates represent an average value of the consumption levels during a ten-year period for total alcohol and the respective drinks (beer, wine and spirits). A constant is included in the RE estimations, and in parenthesis the standard errors of the parameter estimates are reported, with a * indicating significance at least the five per cent level. For the RE models a Hausman specification test is reported with *p*-values in parenthesis.

mortality and with the same parameter values. Concerning beer, the influence is more ambiguous and the any harmful effects seem to be of a much smaller magnitude compared to wine and spirits, which is in accordance with the literature, e.g. Mills (2007). The random effects estimator results in a significant estimate also for beer (in case of the complete data set) and from a medical point of view all beverages should be expected to influence negatively on liver cirrhosis mortality due to the content of alcohol – no matter what the specific drink might be.

5   Conclusion

The empirical evidence from analyzing the relationship between alcohol consumption and liver cirrhosis mortality at an aggregate level in sixteen European countries is leaving no doubt about the detrimental health effects of alcohol intake. At the individual level liver cirrhosis is a consequence of sustained heavy drinking during several years and the present regression analysis of (panel) data covering approximately twenty-five years also gives support to a model where alcohol consumption during the most recent decade is included as an explanatory variable concerning cirrhosis mortality. There has been some uncertainty in the literature whether all the specific beverages influence on cirrhosis mortality, predominantly spirits and wine are assumed to have harmful effects, but in the analysis also beer

shows up with a significant impact, although the parameter estimate is smaller than found in the case of wine and spirits.

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