Is there a Relationship between Suicide and Alcohol Related Mortality in Hungary? A Spatial Panel Approach

Introduction

Alcohol related mortality has been extremely high in Hungary for decades (Bosetti et al. 2007, Meslé 2004). According to the 2014 WHO Report, the number of Years of Life Lost due to excessive alcohol consumption is one of the highest in the world (WHO 2014:214). Hungary has always belonged to the group of nations characterized by high alcoholic beverage consumption and it is still one of the leading liver cirrhosis mortality countries in Europe and in the world (Elekes 2014). Mortality caused by chronic liver diseases (the majority of which are alcohol related) is one of the highest in Europe; in 2010 it was five times as high as the average of the EU-15 countries (Nagy et al. 2013).

Hungarian suicide rate has been outstandingly high for more than a century (Szanto et al., 2007). Despite the monotonous decrease observable since the end of the 1980s, Hungary has the second highest suicide rate in the European Union after Lithuania; Hungarian suicide rate is still among the top ones worldwide (WHO, Värnik 2012, Rihmer et al. 2013).

The relationship of these two self-destructive behaviours is highly relevant in a country where both have played a permanent and prominent role in the prevailing high levels of mortality (Józan 2008).

The relationship of alcohol and suicide

A multitude of studies have proven that alcohol use increases the risk of suicide both among alcohol dependents (Murphy & Wetzel 1990, Roy & Linnoila 1986) and also among non-alcohol-dependent, occasional drinkers (Borges & Rosovsky 1996, Young et al. 1994). Alcohol is a risk factor of suicide (Conner & Chiapella 2004, Hufford 2001, Kendall 1983).

Alcohol consumption increases the risk of suicide through two distinct mechanisms: chronic abuse and acute intoxication (Norström 1988, Landberg 2008, 2009, Ramstedt 2001, Skog 1991). Chronic abuse leads to self-destruction through the destructive social consequences of excessive alcohol consumption. Alcoholism exerts its influence indirectly through the inadequate fulfilment of social roles and the disapproval of the societal environment. Long-term alcohol use may lead to the erosion of kinship, friendship and collegial ties, to rejection by the social environment and finally to suicide. For this reason, the studies neglecting the role of alcoholism might overestimate for example the effect of unemployment or divorce (Rossow 1995). The other mechanism, acute intoxication, has a more direct and short-term effect. Acute intoxication reduces self-control, fosters psychological distress, enhances

suicidal impulses, especially in the case of those people who have already entertained suicidal thoughts (Landberg 2009).

The relationship of alcohol use and suicide has been investigated intensely both at micro and macro levels. A multitude of micro-level studies have demonstrated convincingly that people suffering from alcoholism commit suicide more often (Murphy & Welzel 1990, Inskip et al., 1998, Roy & Linnoila 1986). The values indicating the magnitude of lifetime risk, however, are significantly different. The causality of alcoholism and suicide is also supported by those retrospective and longitudinal studies, which shed light on the alcohol use of the victims at the time of their suicide and on earlier diagnosed alcohol dependence (Cherpitel et al 2004, Cullberg et al. 1988, Rossow & Amundsen 1995, Sher 2006, Welte et al., 1988).

The analyses investigating macro-level relationships are mostly based on time-series modelling; however, their results are less consistent than those of micro-analyses. Nevertheless, the studies supporting a positive relationship between alcohol consumption and suicide rate are probably in majority. It has been observed that the effect of alcohol consumption on suicide rates varies from country to country (Skog 1993, Norström 1988, 1995). The results of time-series models suggest a European north-south gradient in the alcohol consumption effect on suicide mortality (Norström – Ramstedt 2005). Dominantly significant relationships have been found in those so called "dry" countries where the consumption of spirits is preferred and where alcohol consumption is infrequent but heavy, characterised by binge drinking (intoxication-oriented drinking pattern), whereas in southern, dominantly "wet" countries the relationship is less apparent (Norström 1995, Landberg 2008). The north-south gradient were questioned by some studies conducted in post-socialist countries without the main premises have been rejected. These researches have also convincingly confirmed the population-level relationship between alcohol consumption and suicide rate several times (Landberg 2008, Ramstedt 2001, Skog-Elekes 1993, Pridemore-Chamlin 2006, Razvodovsky 2011).

Studies at ecological level

Scientists in the 19th century have already theorized about the relationship between alcohol consumption and suicide. Morselli (1879[1975]: 288-289) emphasised the close and direct relationship between alcoholism and suicide; he based his conclusions on the similarities between the regional patterns of the two phenomena. In his classic work, Durkheim (1897[1951]) rejected the effect of alcohol on suicide. His firm dismissal might have played a role in that, for long decades, social sciences disregarded the examination of the relationship of alcoholism and suicide (Skog et al. 1995). However, the reanalysis of the French department data used by Durkheim indicated that alcohol consumption and suicide rates were far from being independent of each other (Skog 1991). Despite these long research traditions, the spatial investigation of the relationship of alcohol and suicide is quite moderate. The lack of data on alcohol consumption might have a major role in this, resulting in a low number of studies on the spatial analysis of alcohol-related mortality (Grigoriev et al. 2015, Hanson-

Wieczorek 2002). While deaths resulting from suicide can be determined quite easily, alcohol might contribute to the development of several dozens of different diseases (Corrao et al. 2000, Rehm et al. 2003), and determining the deaths caused by alcohol is also much more complicated. It is true for the majority of the low number of ecological studies available that they lack the necessary means of modelling spatial data. Ramstedt (2005) analysed time series data of the Canadian regions after World War 2. This study supported the relationship between the amount of alcohol consumed and suicide rates for all regions except for Quebec. The models using separate estimates identified significant regional differences. A Portuguese study examined regional cross-sectional data on deaths resulting from cirrhosis of the liver and raw suicide rates while controlling for familial and religious integration (childless households, Catholic marriages). Although the variables of social integration explained differences in suicides rates well, deaths from cirrhosis had no effect on them (Skog et al. 1995). Pridemore and Kim (2006) showed similarities in the spatial distribution of Russian alcohol-related mortality and suicides, but their conclusion were not based on statistical inferences. A Norwegian (aspatial) ecological regression study found significant relationships between the rate of alcohol consumption measured by sales figures and self-report and gender-specific suicide rates as well as suicide rates for the whole population. However, the effect disappeared with the exception of one after introducing control for social integration (Rossow 1995).

Hungarian evidences

Several Hungarian empirical studies have investigated the issues concerning alcohol and suicide (Kovács 2008). Recent autopsy examinations conducted in Hungary confirmed that alcohol-consumption is frequent among suicides. High blood alcohol levels (BAC >2,51‰) were characteristic of old-age (65 years and over) victims (Törő et al. 2009). A number of time-series analysis for different periods confirmed the significant relationship of alcohol consumption and suicide (Skog & Elekes 1993, Zonda et al. 2010), and deaths resulting from the cirrhosis of the liver and suicide (Paksi et al. 1995). However, results of the regional analyses of the issue are rather contradictory since they either did not find any (Andorka et al 1968) or found an unexpected negative relationship between suicide rate and deaths resulting from the cirrhosis of the liver, used as the proxy variable of alcohol consumption (Andorka 1990). Skog and Elekes's (1993) hypothesis of alternative solutions might provide an explanation for these results, which cannot be interpreted along causal accounts. According to the hypothesis, alcohol consumption and suicide are alternative behavioural responses to a single common cause, the state of anomie. The choice between alcohol and suicide depends on cultural traditions, which can be particularly well captured by space. Therefore, if the size of at-risk populations is roughly the same, but they respond to anomie differently, then alcohol consumption and suicide might correlate negatively.

Data & Methods

There are no regional data available concerning alcohol consumption; therefore, similarly to previous studies, mortality data were used as a proxy variable (Skog et al 1995). According to the literature, mortality caused by cirrhosis paints an accurate picture of the incidence of alcoholism (Skog 1985).

The demographic register of the Hungarian Central Statistical Office (Demo) provided the data used in the study on mortality and population. Age groups in the data set were created by covering five year periods (15-19, … 85-x). In order to reduce stochastic variability, data were aggregated for five year periods when calculating mortality rates, which enabled the analysis of seven independent (non-overlapping) periods (1980-1984, … 2010-2014). The different age patterns of the micro-regions were eliminated by using indirectly standardized mortality ratio (SMR); these relative values were then multiplied by a scalar, with the country specific directly standardised mortality rate (SDR) based on the age distribution of the European population in 1976. This measurement enabled us the spatio-temporal investigation of suicide and alcohol-related mortality.

During the period examined, two versions of the International Classification of Disease existed, between 1980 and 1995 ICD 9, then in 1996 ICD 10 was introduced. Defining cases of suicide is unproblematic. Deaths resulting from alcohol were defined in two ways. According to the narrower definition, only alcohol-related liver diseases (ICD 9: 571.0-571.3 and ICD 10: K70.0-K70.9) were taken into consideration. On the broader list (Table 1), those causes of death were listed where the disease affecting the organs can be directly attributed to alcohol; nevertheless, only those diseases were taken into account where direct correspondences can be found across the different ICD versions. The procedure applied is widespread in the United Kingdom (ONS 2015, Breakwell et al. 2007, Siegler et al. 2011), and it is also known in Hungary (Kovács 2008). The relationship between regional alcohol-related deaths defined based on this typology and alcohol consumption has been successfully supported in Great Britain (Robinson et al. 2015). The individual time series of alcohol-related and suicide mortality are presented in Tables 1 and 2.

Because of repeated nature of the data, the relationship of alcohol-related mortality and suicide was investigated with the help of a spatial panel model. Panel models offer several advantages over traditional approaches. As opposed to cross sectional models, the panel approach enables us to take into consideration specific effects in time besides the individual (random) effects and also ensures the control of invariant spatial effects (Elhorst 2014). Because of the higher number of observations and the higher degree of freedom, the estimates are more effective and the models can be more informative (Elhorst 2003). Spatial panel models can be fixed and random effect models. Spatial correlations can derive from two sources: they can derive from a data generated process, which can be described by the theory, or specification or measurement errors. In the first case, spatial autocorrelation can be modelled as the spatial diffusion process of the dependent variable, where the outcome of the response variable is influenced by the values of the neighbouring locations (spatial lag model), or it can be obtained through the idiosyncratic shocks (spatial error model).

Table 1.

ICD 9 and ICD 10 CODES

ICD 9	Codes	ICD 10	Codes
	Alcoholic mental diseases		
305.0	Alcohol abuse	F10.1	Alcohol abuse
303.0	Acute alcoholic intoxication	F10.22	Alcohol dependence with intoxication
291.0	Alcohol withdrawal delirium	F10.23	Alcohol dependence with withdrawal
		F10.24	Alcohol dependence with alcohol-induced mood disorder
291.3	Alcohol-induced psychotic disorder with hallucinationsű	F10.25	Alcohol dependence with alcohol-induced psychotic disorder
291.4	Idiosyncratic alcohol intoxication	F10.251	Alcohol dependence with alcohol-induced psychotic disorder with hallucinations
291.5	Alcohol-induced psychotic disorder with delusions	F10.250	Alcohol dependence with alcohol-induced psychotic disorder with delusions
291.9	Unspecified alcohol-induced mental disorders	F10.259	Alcohol dependence with alcohol-induced psychotic disorder, unspecified
291.1	Alcohol-induced persisting amnestic disorder	F10.26	Alcohol dependence with alcohol-induced persisting amnestic disorder
291.2	Alcohol-induced persisting dementia	F10.27	Alcohol dependence with alcohol-induced persisting dementia
		G31.2	Degeneration of nervous system due to alcohol
357.5	Alcoholic polyneuropathy	G62.1	Alcoholic polyneuropathy
	Cardiovascular disease		
425.5	Alcoholic cardiomyopathy	I42.6	Alcoholic cardiomyopathy
	Diseases of digestive system		
571.0	Alcoholic fatty liver	K70.0	Alcoholic fatty liver
571.1	Acute alcoholic hepatitis	K70.1	Alcoholic hepatitis
		K70.2	Alcoholic fibrosis and sclerosis of liver
571.2	Alcoholic cirrhosis of liver	K70.3	Alcoholic cirrhosis of liver
		K70.4	Alcoholic hepatic failure
571.3	Alcoholic liver damage, unspecified	K70.9	Alcoholic liver disease, unspecified
		K74.3	Primary biliary cirrhosis
		K74.4	Secondary biliary cirrhosis
571.6	Biliary cirrhosis	K74.5	Biliary cirrhosis, unspecified
571.8	Other chronic nonalcoholic liver disease	K74.6	Other and unspecified cirrhosis of liver
535.3	Alcoholic gastritis	K29.2	Alcoholic gastritis
577.1	Chronic pancreatitis	K86.0	Alcohol-induced chronic pancreatitis
		K86.1	Other chronic pancreatitis
	External causes of deaths		
E860.0	Accidental poisoning by alcoholic beverages	X45.0	Accidental poisoning by and exposure to alcohol
E950.0-E959.0	Suicide	X60.0-X84.0	Suicide (Intentional self-harm)

Figure 1.

Standardized alcohol related mortality rates of subregions by NUTS2 regions

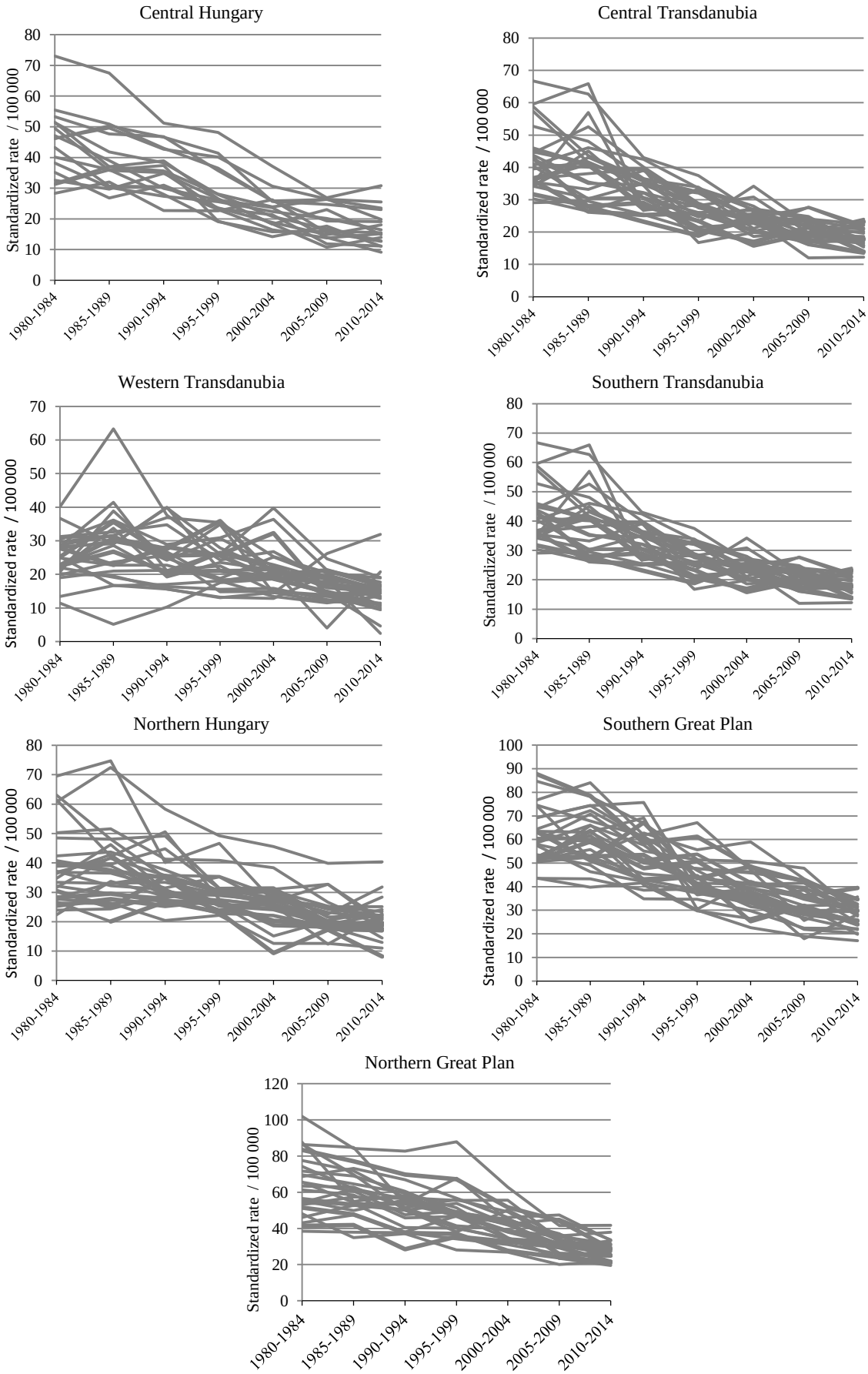
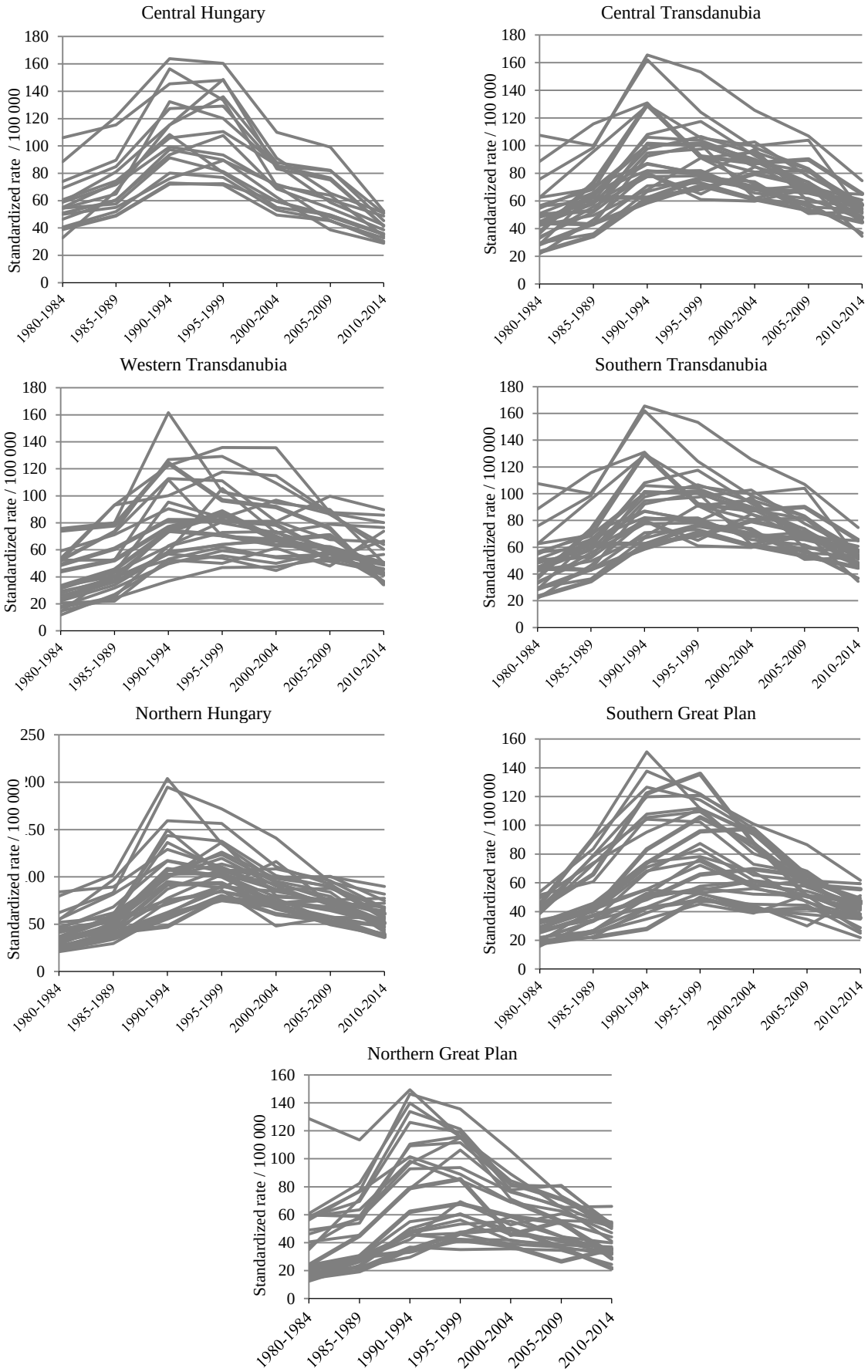


Figure 2.

Standardized alcohol related mortality rates of subregions by NUTS2 regions



Results

Natural logarithms of the variables were used for the estimates. The rationale for the transformation is the following: according to the Jellinek formula, the number of alcohol-dependents is in linear relationship with the number of deaths caused by cirrhosis of the liver, and according to the Lederman estimate, changes in the per capita alcohol consumption are in quadratic relationship with changes in the number of alcoholics. It can be assumed that the relationship between changes in the number of deaths cause by cirrhosis of the liver and changes in alcohol consumption is also quadratic. Therefore, if we want to estimate the effect of the tendency to consume alcohol on alcohol-related mortality, we have to use logarithm values. As a result of the double log transformation, we can evaluate the elasticity of the variables in the model. The estimations were created using the splm package written in programming language R (Millo & Piras 2012). The choice between fixed and random effect models was carried out with the help of the spatial Hausman test, which recommended the use of random models in all cases. In the case of both independent variables, the random effect models estimated using temporal (ψ) and spatial (ρ) correlation (SEMSRRE) proved to be the most appropriate.

The estimates using the classic least squares method were negative and non-significant in the case of both predictors (Table 2-3). However, calculations using the spatial panel models resulted in the expected estimates. The elasticity of the models was positive in both cases. However, the magnitude of the estimates differed significantly. A 100 percent increase in the rate of alcoholic liver diseases resulted in a 5 percent increase in suicide rate. The effect in the case of alcohol-related mortality (Table 3) was more than double of this (12%). Besides, the model using estimates of alcohol-related mortality had a better fit, which was showed by the higher log-likelihood value.

Table 2.

Spatial panel regressions of suicide and alcoholic liver disease

Variables	Pooled (OLS)	Random effect spatial error model	Random effect spatial lag model	Random effect spatial error model + serial correlation	Random effect spatial lag model + serial correlation
Constant	3.624***	3.086***	0.713***	3.232***	1.334***
	(0.079)	(0.078)	(0.053)	(0.082)	(0.059)
Alcohol liver disease	-0.057**	0.093***	0.029*	0.050*	0.015
	(0.021)	(0.019)	(0.013)	(0.021)	(0.016)
ϕ		2.215***	2.236***	1.747***	1.559***
		(0.281)	(0.290)	(0.270)	(0.309)
ρ		0.786***		0.668***	
		(0.017)		(0.032)	
λ			0.760***		0.592***
			(0.018)		(0.038)
ψ				0.451***	0.569***
				(0.056)	(0.063)
Log-likelihood	-770.82	-161.19	-192.45	-119.94	-134.32
N	175	175	175	175	175
T	7	7	7	7	7

***$p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Table 3.

Spatial panel regressions of suicide and alcohol-related deaths

Variables	Pooled (OLS)	Spatial error Random effect model	Spatial lag model random effect model	Spatial error serial correlation Random effect	Random effect spatial lag model + serial correlation
Constant	5.353***	2.660***	0.560***	2.913***	1.247***
	(0.117)	(0.112)	(0.074)	(0.123	(0.088)
Alcohol-related mortality	-0.028	0.185***	0.051***	0.123***	0.032
	(0.028)	(0.030)	(0.017)	(0.029)	(0.021)
ϕ		2.260***	2.329***	1.831***	1.565***
		(0.286)	(0.289)	(0.272)	(0.307)
ρ		0.797***		0.693***	
		(0.017)		(0.030)	
λ			0.763***		0.595***
			(0.018)		(0.038)
ψ				0.407***	0.565***
				(0.056)	(0.063)
Log-likelihood	-773.78	-148.00	-190.50	-114.88	-133.61
N	175	175	175	175	175
T	7	7	7	7	7

***$p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Discussion

The validity of the relationship between alcohol and suicide was successfully supported with the help of spatial data. Instead of the hypothesis which considered suicide and alcoholism as alternative methods of frustration and stress management (Skog & Elekes 1993), it was the traditional causal model which provided a satisfactory explanation for the relationship of suicide and alcohol-related mortality. The public health relevance of the study is that by reducing alcohol consumption (alcohol-related mortality), mortality resulting from suicide will also decrease. Our results also point out that a broader definition of alcohol-related mortality provides a better estimation of suicide mortality. Consequently, when quantifying alcohol-related deaths, alcohol-related liver diseases should be supplemented by a more comprehensive list of causes of death.

Suicide is a complex phenomenon; an interaction of biological, psychological, socio-demographic and cultural factors, life events, life style contribute to it (Almasi et al 2009, Rihmer et al. 2013). The analysis of the relationship of alcohol and suicide often lacks a wider range of predictors; however, in such a case attention should be drawn to the possible bias of the omitted variables (Gujarati 2003). Moreover, it should also be emphasised that both alcoholism and suicide have strong gender-specific characteristics; thus, studies carried out for the genders separately might contribute to a better understanding of these two deviant behaviours.

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