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An evaluation of the impact of a large reduction in alcohol prices on alcohol-related and all-cause mortality: time series analysis of a population-based natural experiment

Kimmo Herttua,[1]* Pia Mäkelä[2] and Pekka Martikainen[1]

[1]Population Research Unit, Department of Sociology, University of Helsinki, Helsinki, Finland, [2]National Institute for Health and Welfare, Helsinki, Finland

*Corresponding author. Department of Sociology, P.O. Box 18, 00014 University of Helsinki, Finland. E-mail: kimmo.herttua@helsinki.fi

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Background We examined the effect of a large reduction in the price of alcohol that occurred in Finland in 2004 on alcohol-related and all-cause mortality, and mortality due to cardiovascular diseases (CVDs) from which alcohol-attributable cases were excluded.

Methods Time series intervention analysis modelling was applied to the monthly aggregations of deaths in Finland for the period 1996–2006 to assess the impact of the reduction in alcohol prices. Alcohol-related mortality was defined using information on both underlying and contributory causes of death. Analyses were carried out for men and women aged 15–39, 40–49, 50–69 and >69 years.

Results Alcohol-related deaths increased in men aged 40–49 years, and in men and women aged 50–69 years, after the price reduction when trends and seasonal variation were taken into account: the mean rate of alcohol-related mortality increased by 17% [95% confidence interval (CI) 1.5, 33.7], 14% (95% CI 1.1, 28.0) and 40% (95% CI) 7.1, 81.7), respectively, which implies 2.5, 2.9 and 1.6 additional monthly deaths per 100 000 person-years following the price reduction. In contrast to alcohol-related mortality, CVD and all-cause mortality decreased: among men and women aged >69 years a decrease of 7 and 10%, respectively, in CVD mortality implied 19 and 25 fewer monthly deaths per 100 000 person-years, and a decrease of 7 and 14%, respectively, in all-cause mortality similarly implied 42 and 69 fewer monthly deaths.

Conclusion These results obtained from the time series analyses suggest that the reduction in alcohol prices led to an increase in alcohol-related mortality, except in persons <40 years of age. However, it appears that beneficial effects in older age, when CVD deaths are prevalent, counter-balance these adverse effects, at least to some extent.

Keywords Alcohol drinking, commerce, economics, mortality, alcohol-related disorders, cardiovascular diseases

Introduction

Changes in alcohol prices have been documented to be inversely associated with changes in consumption and alcohol-related problems. Much of the evidence on this issue is based on cross-sectional state-level time series data,[1,2] and natural experiments have been called for.[1]

The changes in the Finnish alcohol policy in 2004 could be considered as a natural experiment. Following the deregulation of import quotas within the European Union (EU) on 1 January 2004, it became possible to import from other member countries unlimited amounts of alcoholic beverages for one's own use without paying further taxes. Finnish taxes on alcohol were reduced by an average of 33% on 1 March 2004: the off-premise retail price of spirits went down by 28–36%, wines by 3%, beers by 13% and other alcoholic beverages by 7–28%.[3] The reason for the tax cuts was that Estonia joined the EU on 1 May 2004, which had a great impact on the Finnish alcohol market because of the proximity of the two countries and the significantly lower prices in Estonia. Estimated total per capita alcohol consumption (recorded and unrecorded) was 9.4 l per inhabitant in Finland in 2003. The increase was estimated to be 10% in 2004, to 10.3 l, after which it has remained roughly on that level.[4]

Two previous studies have indicated that the reduction in alcohol prices in 2004 had an impact on alcohol-related deaths. In a register-based natural experiment, before–after comparison showed that a reduction in price was followed by substantial increases in alcohol-related mortality, particularly among those less privileged in society, and in chronic diseases associated with heavy drinking.[5] A time series analysis demonstrated that the price reduction resulted in eight additional alcohol-positive sudden deaths per week within 10 months of the reduction.[6]

Overall, aggregate-level research on the effect of alcohol prices on alcohol-related mortality is sparse. Two cross-sectional time series studies from the USA report contradictory findings on these effects: a study covering 30 states and years 1962–77 estimated that an increase in excise tax on spirits reduced mortality from liver cirrhosis,[7] whereas according to a later study covering years 1982–88 across 48 states, higher alcohol prices did not decrease mortality rates for alcohol-attributable primary causes of death.[8] This difference is mainly attributable to the inclusion of a larger number of independent variables in the latter study.[8] A recent study from Alaska reported that increase in alcohol excise tax rates were associated with immediate and sustained reductions in alcohol-related disease mortality in 1976–2004.[9]

In contrast, the relation between alcohol consumption and mortality has been investigated more extensively. A review of studies on mortality and population drinking covering most of the EU member states, Canada and the USA concluded that the association was statistically significant in terms of liver cirrhosis and other alcohol-related diseases in all countries, as was the association between consumption and mortality from accidents and homicide in about half of them.[10–14] Moreover, a 1-l increase in per capita consumption was associated with a stronger effect on mortality in northern Europe and Canada than in mid- and southern Europe.

Apart from these adverse effects on health, alcohol consumption may have beneficial effects as well. A large body of epidemiological evidence has concluded that low to moderate consumption is associated with a reduced risk of cardiovascular disease (CVD) and all-cause mortality at the individual level.[15–17] Hence, one could assess the beneficial effects by studying CVD mortality, and the net effect of consumption by using all-cause mortality as an outcome measure. Alcohol consumption confers cardiovascular protection predominantly through the elevation of high-density lipoprotein cholesterol, and the enhancement of insulin sensitivity.[15,18,19] However, an adverse effect of increased per capita consumption on all-cause mortality has been reported in time series studies from several countries.[20,21] In the main, no significant associations between alcohol consumption and mortality from ischaemic heart disease (IHD) were found in 15 European countries.[22] There are no time series studies directly addressing the relationship between alcohol prices and CVD or all-cause mortality. Moreover, beneficial effects of low to moderate consumption, even if the evidence is not as convincing as for CVD mortality, have been reported for some other diseases, such as dementia, diabetes and chronic obstructive pulmonary diseases (COPDs).[23–28]

The unique natural experiment in Finland in 2004 involving a substantial reduction in the full price of alcohol has thus given us the opportunity to directly evaluate changes in mortality when prices fall and consumption increases. Hence, the purpose of the present study was to evaluate, by means of time series analysis, the impact of the reduction in alcohol prices on alcohol-related mortality, mortality due to CVDs and all-cause mortality, stratified by sex and age. The study period extended from >8 years before to almost 3 years after the realization of the price reduction. Data on coronary operations were included as a control series in analyses of CVD mortality. Mortality tends to rise with increasingly cold temperatures from an optimum temperature value,[29,30] IHD being the biggest single cause of excess mortality in winter.[31,32] We therefore also conducted temperature-adjusted analyses of CVD and all-cause mortality.

Methods

Mortality data

Mortality data for the years 1996–2006 were obtained from Statistics Finland (permission CS-52-222-08). The monthly data were stratified by sex and 5-year age groups (15–19, . . . , 75–79, >80). Causes of death were classified according to the Finnish edition (FCD) of the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10).

Alcohol-related deaths were defined as those for which there was a reference to alcohol on the death certificate as the underlying or one of the contributory causes. Estimating alcohol-related mortality on the basis of both the underlying and contributory causes yields more versatile and comprehensive data than the standard method based solely on the underlying cause, particularly in Finland where death certificates record alcohol intoxication as a contributory cause more frequently and accurately than in most other countries.[33,34] Frequent use of medicolegal autopsy is one of the major factors enabling the proper attribution of alcohol intoxication as a contributory cause of death. Medicolegal autopsies were carried out in 98% of all accidental and violent deaths occurring among people aged <65 years in 2006,[35] and in >60% of all deaths in 1987–2003.[36]

The total pool of alcohol-related deaths used here consists of the following two main categories: (i) the underlying cause of death was an alcohol-attributable disease or fatal alcohol poisoning (ICD10 code X45); and (ii) the underlying cause was not alcohol related, but a contributory cause was an alcohol attributable disease or alcohol intoxication (ICD-10 code F100). The first group i.e. underlying causes constituted 42% of all alcohol-related deaths ($n = 41\,385$). Of all deaths in which the underlying cause was alcohol attributable, 43% referred to alcoholic liver disease (ICD-10 code K70), 26% to fatal alcohol poisoning (ICD-10 code X45), 13% to alcohol dependence syndrome (ICD-10 code F102), 7% to alcoholic cardiomyopathy (ICD-10 code I426), 5% to alcoholic diseases of the pancreas (ICD-10 code, Finnish Edition, K860), 3% to other mental and behavioural disorders due to alcohol (ICD-10 codes F101, F103-109) and 3% to a few rarely occurring categories (ICD-10 codes K292, G312, G4051, G621, G721). In the second group the underlying cause was suicide in 19%, CVD in 17% and accidental fall in 6% of the cases.

We used alcoholic liver diseases instead of all liver diseases because there does not seem to be any strong tendency to underreport alcoholic cases in Finland: for example, in 2006, 98% of deaths due to liver cirrhosis among men aged <65 years were classified as alcohol related on the death certificate.[35] The proportion of all alcohol-related deaths among men was 83%.

CVDs consist of the following categories: IHDs (ICD-10 codes I20–I25), other heart diseases excluding rheumatic heart diseases (ICD-10 codes I30–I425, I427–I52), cerebrovascular diseases (ICD-10 codes I60–I69) and other diseases of the circulatory system (ICD-10 codes I00–I15, I26–I28, I70–I99). Alcohol-attributable CVDs, i.e. cases that had alcohol-attributable contributory causes of death, were excluded due to their inclusion in alcohol-related deaths. The beneficial effects of the price reduction on CVD mortality could thus be distinguished from its detrimental effects. Furthermore, we also included a few other causes of death categories in the analysis for older persons on the assumption that they may benefit from moderate alcohol consumption.[23–28] These categories included dementia, diabetes and COPDs.

Data for the control series

We acquired two additional monthly datasets for the control series for the years 1996–2006. Data (also stratified by sex and age) on the number of coronary operations (including bypass operations and angioplasties) were obtained from the National Research and Development Centre for Welfare and Health, and were used as control series in the analyses of CVD mortality. The Finnish Meteorological Institute provided us with a dataset on monthly mean temperatures in three different places in the southern and central parts of Finland, in which 87% of the Finnish population lives. In order to form a control series, we first combined these measures into a single mean temperature, which we then converted into a categorized winter-cold variable with a value of 0 for temperatures >0°C, 1 for 0 to −2.99°C, 2 for −3 to −5.99°C, 3 for −6 to −8.99°C and 4 for −9°C and colder.

Statistical analysis

For the analyses, monthly deaths were converted to monthly mortality rates per 100 000 person-years. We used Box–Jenkins autoregressive integrated moving average (ARIMA) intervention time series analyses to model the monthly alcohol-related, CVD and all-cause mortality. The intervention was assumed to take place on 1 March 2004. We also conducted the analyses using 1 January and 1 May as intervention points (see the 'Introduction' section for the dates of the changes in alcohol policies). It appeared that the effect estimates were largest and in most cases the models were best identified when 1 March was used: the differences were small, however, and therefore we only show the results from the models with 1 March as an intervention point.

We also considered using monthly alcohol sales as a direct measure of consumption. However, there are four main problems with using such data to capture the mediating effects of change in consumption on mortality following a policy change: (i) sales data

cannot capture the effect on binge drinking or other types of drinking patterns; (ii) it cannot be broken down by sex and age; (iii) it does not capture changes in unrecorded consumption; and (iv) the reliable part of sales series regarding the timing of the change, i.e. sales to consumers, constitutes only 40% of all consumption. All in all, the benefits of incorporating sales series into a time series intervention of a sudden change in policy appear modest and we decided not to carry out such analyses.

The method used involves a two-phase process. The aim in the first phase is to identify a descriptive model that best captures seasonality, time trends and the autocorrelation inherent in the series.[37,38] The intervention component is added in the second phase in order to obtain the impact assessment model that allows causal attribution of changes in time series to given events (for further details see Box *et al*.[38]). Unlike other methods, time series analysis can detect trends and seasonal variation that have a tendency to bias assessment of the impact on an outcome measure. Moreover, time series intervention analysis can reveal changes that may differ in terms of both the onset and the duration of the effects they produce[38]. We hypothesized that the reduction in alcohol prices could have three possible effects on the outcome measures: (i) abrupt and permanent, (ii) abrupt and temporary and (iii) gradual and permanent. We rejected the latter two after fitting the models as neither of them was essentially better in terms of model identification or impact strength, possibly because they may need a longer time period after intervention before they are identifiable.

Outliers may distort specification of the ARIMA model and its parameter estimates in time series analyses unless they are properly taken into account.[39,40] In the analyses of cardiovascular mortality, a dummy variable was added as a regression parameter to the models for persons aged >69 years (January 1996 and 2000, and December 2003). These outliers hindered model specification, but their presence or absence in the models did not largely affect the results. The annual salient wintertime peaks in all-cause mortality among the oldest, and a peak in December 2004 due to the tsunami in Thailand (171 Finns died) among those aged <70 years were treated in the same manner. Control series (i.e. coronary operations) were added to the models in the analyses of cardiovascular mortality. Variance appeared to change over time in all the series, particularly after the price reduction. A natural log transformation was therefore applied. The maximum likelihood estimation method in Stata, Version 10 (Stata Corporation, College Station, TX, USA) was used for all the time series analyses.

Results

The time series for monthly alcohol-related, cardiovascular and all-cause mortality rates for men and women are displayed in Figures 1–3. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004. There appeared to be a clear age-specific pattern in the overall trends, which was similar among both men and women. Alcohol-related mortality rates fluctuated or were stable among persons <50 years of age, but were mainly increasing among the older groups. As far as cardiovascular and all-cause mortality were concerned, the trend was mostly declining in all age and sex groups.

We used ARIMA modelling in our formal assessment of the effect of the price reduction on alcohol-related mortality (see Table 1). These models identified a clear increase in three groups. Among men aged 40–49 years, and men and women aged 50–69 years, the impact parameters (0.15, 0.13 and 0.33) suggested that the price reduction produced an increase of 17% [95% confidence interval (CI) 1.5, 33.7], 14% (95% CI 1.1, 28.0) and 40% (95% CI 7.1, 81.7), respectively, in the alcohol-related mortality rate, which implies an increase of 2.5, 2.9 and 1.6 monthly deaths per 100 000. Among men aged >69 years and women aged 40–49 years, the models provided point estimates of 9% (95% CI −3.8, 23.5) and 11% (95% CI −1.8, 24.6), respectively.

Table 2 shows the analysis of the effects of the price reduction in terms of change in cardiovascular mortality (alcohol-attributable cases excluded). There was a clear decrease in mortality in four subgroups: among men and women aged 40–49 years and >69 years, the estimated decrease was 21% (95% CI −32.2, −7.2), 24% (95% CI −40.5, −3.1), 7% (95% CI −12.4, −0.7) and 10% (95% CI −13.5, −6.5), respectively, which implies a decrease of 1.2, 0.5, 19.1 and 24.8 monthly deaths per 100 000 person-years, respectively. When coronary operations were added into the models as control series the estimates of the impact remained essentially the same (data not shown). In an additional analysis, we examined the effect of the price reduction on mortality due to IHD, which seems to show the beneficial effects of alcohol most clearly. Estimates of the effect on IHD mortality were larger than those for mortality including other CVD categories (data not shown): among men and women aged >69 years, the estimated decrease was 8.9% (95% CI −12.5, −5.1) and 12.5% (95% CI −15.1, −8.2), respectively, in IHD mortality, whereas it was 6.6% (95% CI −16.2, 4.2) and 8.1% (95% CI −14.4, −1.4) in mortality due to other CVD categories. Table 3 shows the ARIMA analysis of the effect of the price reduction on all-cause mortality. The models estimate the impact to be 2-fold: there was a clear decrease in mortality among the youngest and oldest of both sexes, whereas there was no substantial change in the other groups. The estimated impacts of −0.07 and −0.15 among men and women aged >69 years suggest that there was a decrease in mortality of 7% (95% CI −13.0, −1.5) and 14% (95% CI −19.5, −8.2) after the price

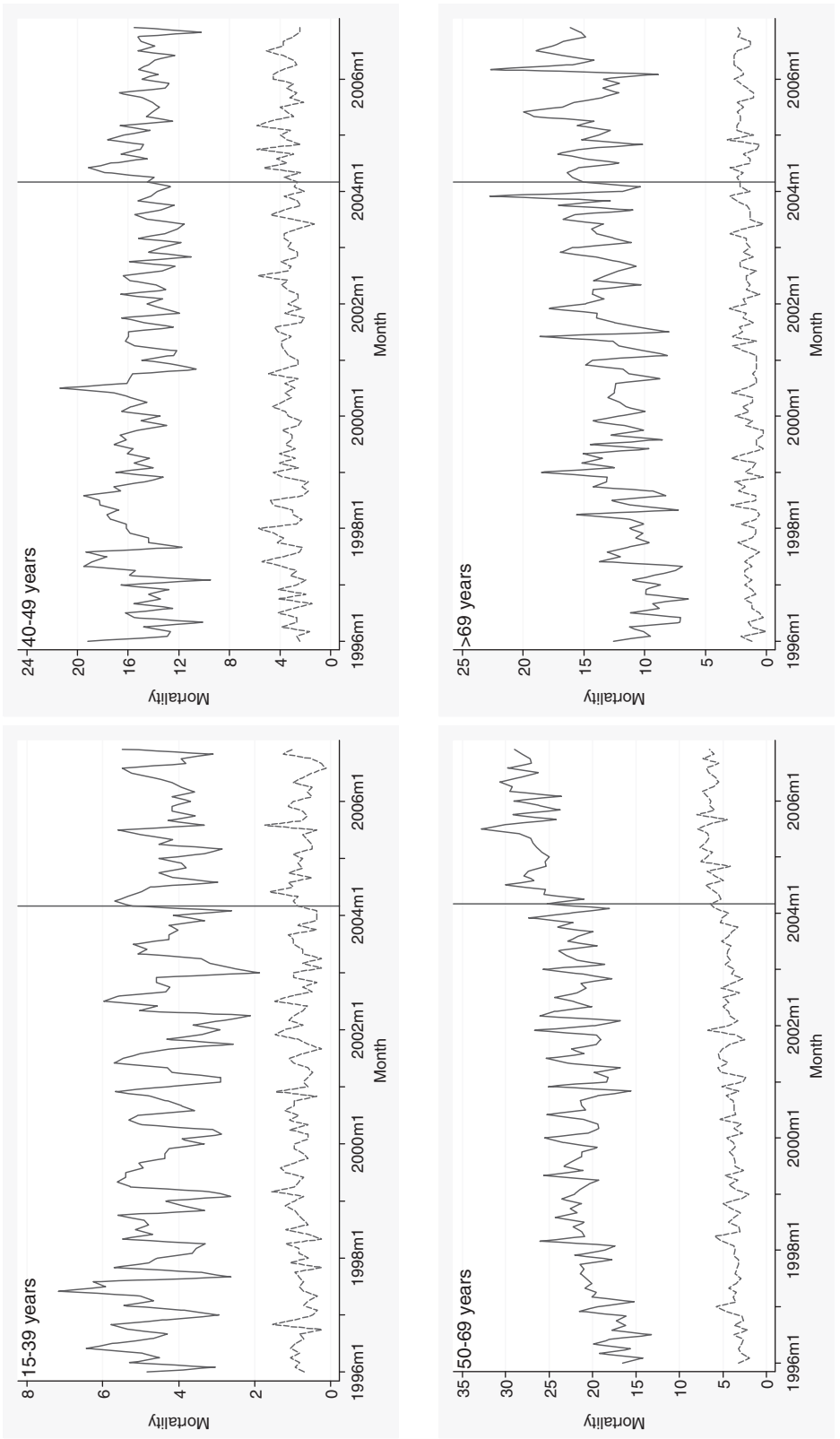

Figure 1 Monthly alcohol-related mortality rates per 100 000 person-years among men (solid line) and women (dashed line), aged 15–39, 40–49, 50–69 and >69 years, Finland, 1996–2006. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004

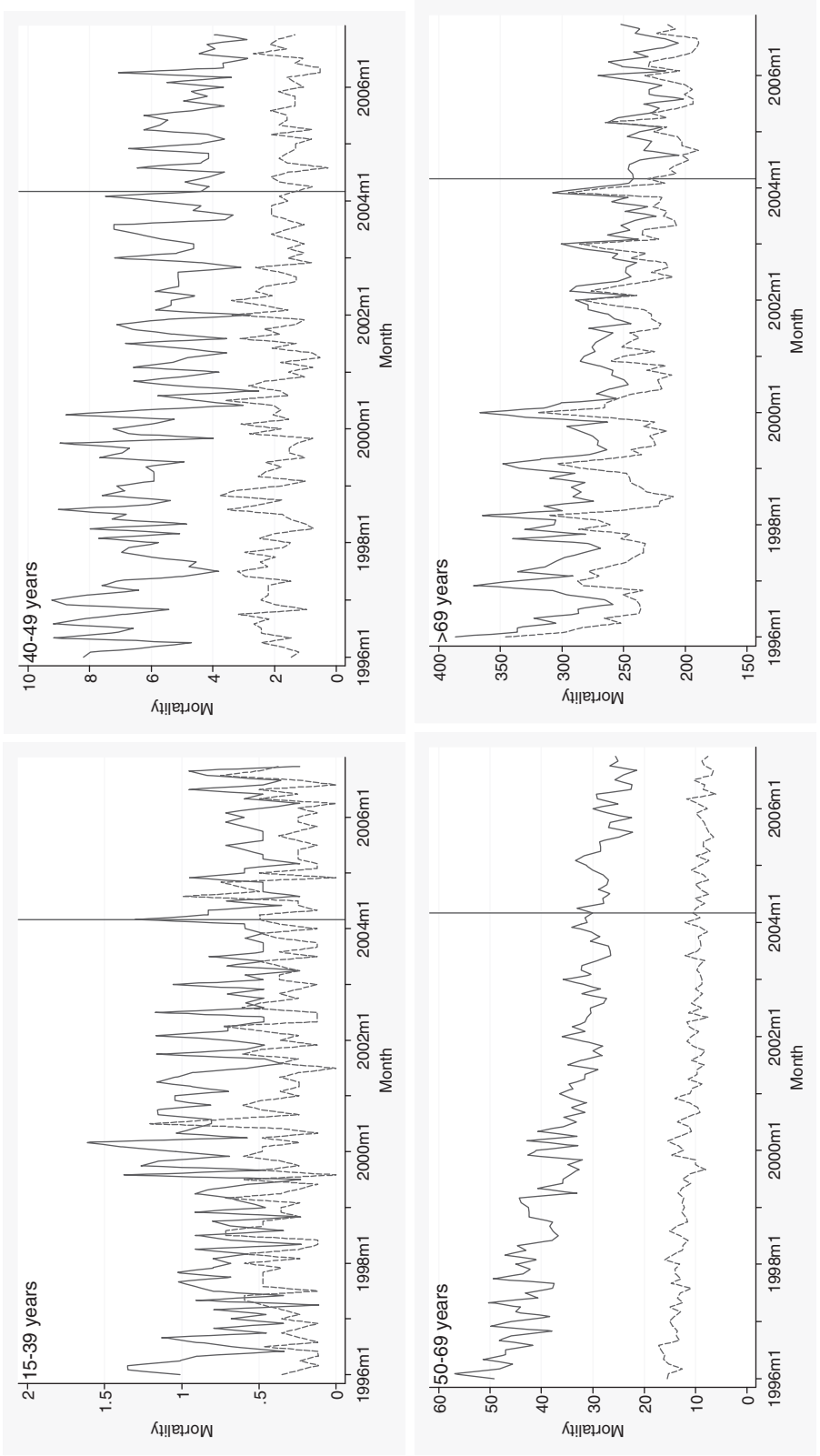

Figure 2 Monthly rates of CVD mortality per 100 000 person-years among men (solid line) and women (dashed line), aged 15–39, 40–49, 50–69 and >69 years, Finland, 1996–2006. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004

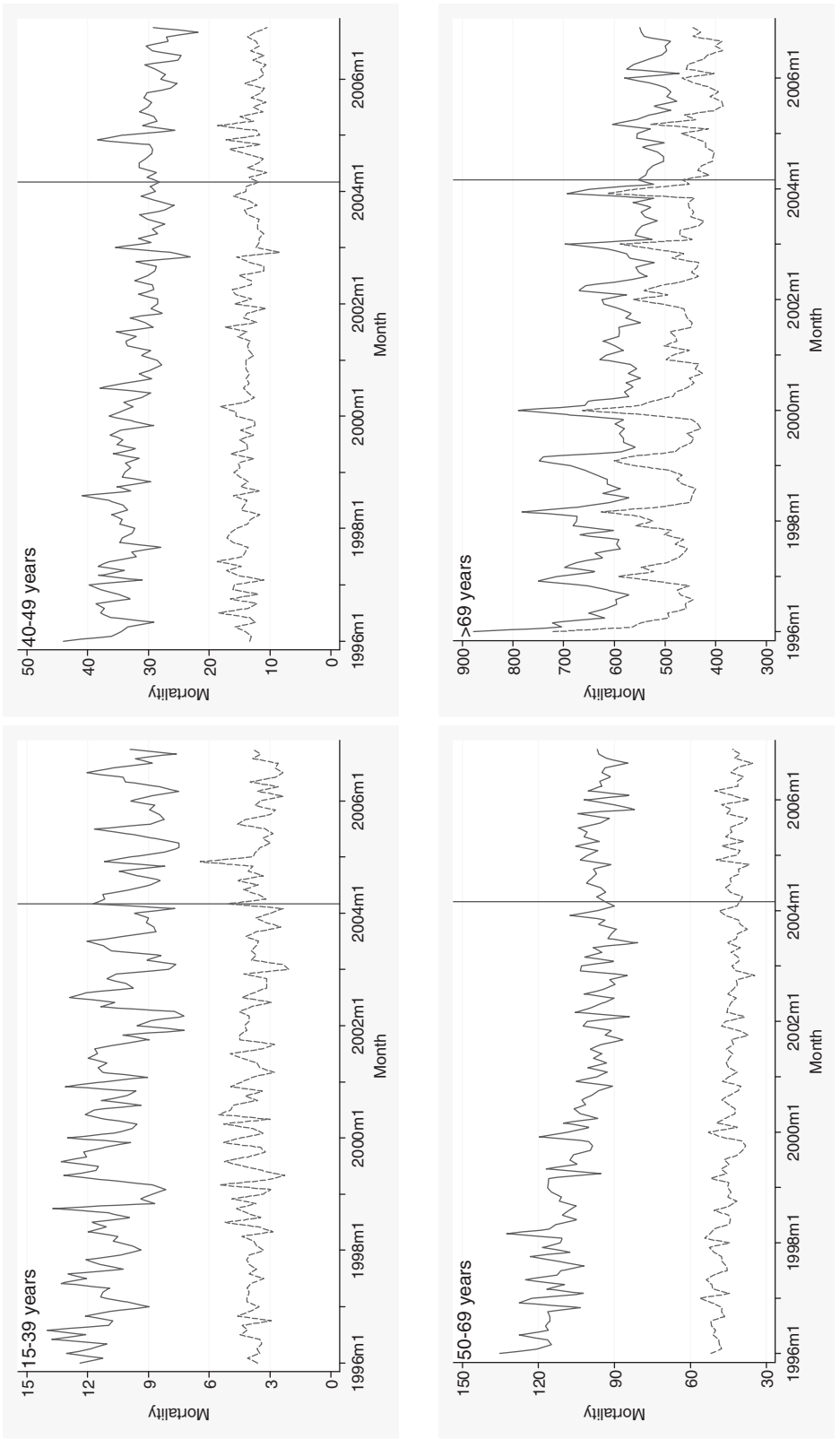

Figure 3 Monthly all-cause mortality rates per 100 000 person-years among men (solid line) and women (dashed line), aged 15–39, 40–49, 50–69 and >69 years, Finland, 1996–2006. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004

Table 1 The effects of the reduction in alcohol prices on alcohol-related mortality by sex and age, natural logarithmic ARIMA models

	Monthly average	Estimate	Change		
			Percent level[a]	95% CI	Deaths per 100 000[b]
Men					
15–39 years	37				
Impact		−0.02	−1.6	−12.7 to 11.0	−0.1
Noise and Q(24)		ARIMA(0,0,0)$(0,1,1)_{12}$; Q(24) = 12.29; $P = 0.976$			
40–49 years	59				
Impact		0.15	16.5	1.5 to 33.7	2.5
Noise and Q(24)		ARIMA(0,1,1)$(1,0,0)_{12}$; Q(24) = 19.28; $P = 0.737$			
50–69 years	136				
Impact		0.13	13.8	1.1 to 28.0	2.9
Noise and Q(24)		ARIMA(0,1,1)$(1,0,0)_{12}$; Q(24) = 26.63; $P = 0.322$			
>69 years	26				
Impact		0.09	9.0	−3.8 to 23.5	1.1
Noise and Q(24)		ARIMA(0,0,0)$(2,1,0)_{12}$; Q(24) = 16.46; $P = 0.871$			
Women					
15−39 years	7				
Impact		−0.03	−3.2	−21.8 to 19.3	0.0
Noise and Q(24)		ARIMA(0,0,0)(0,1,1)12; Q(24) = 25.75; $P = 0.366$			
40–49 years	20				
Impact		0.10	10.6	−1.8 to 24.6	0.3
Noise and Q(24)		ARIMA (0,1,1); Q(24) = 17.46; $P = 0.828$			
50−69 years	29				
Impact		0.33	39.5	7.1 to 81.7	1.6
Noise and Q(24)		ARIMA (0,1,1); Q(24) = 18.92; $P = 0.753$			
>69 years	6				
Impact		0.21	23.1	−18.4 to 84.8	0.3
Noise and Q(24)		ARIMA (0,1,1); Q(24) = 27.42; $P = 0.285$			

[a]Derived from exp(estimate).
[b]Obtained by multiplying the mortality rate before the change by the percentage change.
Q(24) is the Portmanteau test for residual autocorrelation with 24 lags.

reduction, which implies a decrease of 42 and 69 monthly deaths per 100 000, respectively. Among men and women aged 15–39 years there were decreases of 9% (95% CI −16.5, −1.4) (one death per 100 000) and 8% (95% CI −11.3, −0.0) (0.3 deaths per 100 000), respectively, in all-cause mortality, whereas the change was marginal among men and women aged 40–69 years.

Including winter cold as a control variable affected the results only marginally: the adjusted impact estimate was −0.068 (95% CI −0.135, −0.000) compared with the unadjusted estimate of −0.069 (95% CI −0.133, −0.007) among men aged >69 years for CVD mortality, and −0.139 (95% CI −0.211, −0.067) compared with −0.146 (95% CI −0.228, −0.063) among women aged >69 years for all-cause mortality, for example.

Table 4 gives a summary of the results. We calculated a simple sum of lost lives due to alcohol-related deaths and saved lives due to its beneficial effects on CVD: there were 18.1 monthly saved lives per 100 000 among men aged >69 years and 24.6 among women of the same age, whereas the estimated decrease from the model on all-cause mortality clearly exceeded this net sum. In other groups the monthly saving or loss of lives was −0.2 to 1.2 per 100 000. In order to investigate this gap between CVD and all-cause mortality, we conducted time series analyses of the effect of the price reduction on mortality due to COPD, dementia and diabetes among persons aged >69 years (data not shown). Mortality due to COPD decreased by 14.9% (95% CI −27.2, −0.4) in men and 17.9% (95% CI −31.9, −0.9) in women that implied a decrease of 4.1 and 1.0 monthly deaths per 100 000 person-

Table 2 The effects of the reduction in alcohol prices on mortality due to CVDs (alcohol-attributable cases excluded) by sex and age, natural logarithmic ARIMA models

	Monthly average	Estimate	Change		
			Percent level[a]	95% CI	Deaths per 100 000[b]
Men					
15–39 years	6				
Impact		0.06	6.3	−25.5 to 51.5	0.0
Noise and Q(24)		ARIMA (0,1,1); Q(24) = 26.13; $P = 0.347$			
40–49 years	23				
Impact		−0.23	−20.7	−32.2 to −7.2	−1.2
Noise and Q(24)		ARIMA $(0,0,0)(0,1,1)_{12}$; Q(24) = 23.24; $P = 0.506$			
50–69 years	205				
Impact		−0.05	−4.5	−25.0 to 21.6	−1.7
Noise and Q(24)		ARIMA (2,1,0); Q(24) = 12.89; $P = 0.968$			
>69 years	533				
Impact		−0.07	−6.7	−12.4 to −0.7	−19.1
Noise and Q(24)		ARIMA $(0,1,1)(1,0,0)_{12}$; Q(24) = 25.25; $P = 0.392$			
Women					
15–39 years	3				
Impact		−0.01	−1.3	−24.8 to 29.7	0.0
Noise and Q(24)		ARIMA (0,0,1); Q(24) = 11.74; $P = 0.983$			
40–49 years	7				
Impact		−0.28	−24.1	−40.5 to −3.1	−0.5
Noise and Q(24)		ARIMA (0,1,1); Q(24) = 25.01; $P = 0.389$			
50–69 years	68				
Impact		−0.04	−4.1	−18.7 to 13.2	−0.5
Noise and Q(24)		ARIMA (0,1,1); Q(24) = 25.10; $P = 0.400$			
>69 years	840				
Impact		−0.11	−10.1	−13.5 to −6.5	−24.8
Noise and Q(24)		ARIMA $(0,1,1)(2,1,0)_{12}$; Q(24) = 20.22; $P = 0.684$			

[a]Derived from exp(estimate).
[b]Obtained by multiplying the mortality rate before the change by the percentage change.
Q(24) is the Portmanteau test for residual autocorrelation with 24 lags.

years, respectively, whereas dementia and diabetes increased by 8.5% (95% CI 0.9, 16.6; 3.8 more monthly deaths) in men but decreased by 6.9% (95% CI −12.1, −1.4; 8.4 fewer monthly deaths) in women. Hence, a decrease in CVD and COPD mortality accounted for 55% of the decrease in all-cause mortality in men and 38% in women. Moreover, a decrease in dementia and diabetes mortality accounted for an additional 12% in women.

Discussion

Time series analyses of the impact of the reduction in alcohol prices in Finland in 2004 show that alcohol-related mortality increased among both men and women aged ⩾40 years, whereas there was virtually no change among younger people. The increase was strongest in relative terms among women aged 50–69 years, and in absolute terms among men of the same age. These findings are in accordance with those reported in two earlier studies examining the effects of the same price reduction on alcohol-related mortality. One of these, in which a before–after design was adopted,[5] also focused on socio-economic differentials and showed that alcohol-related mortality increased particularly strongly among the long-term unemployed and pensioners. As far as differences according to age were concerned, the same study showed that the increase in absolute terms was largest among persons aged 50–69 years, whereas those <35 years of age did not suffer from increased mortality during the 2 years after the change. The impact of the price reduction appeared to be smaller in our current

Table 3 The effects of the reduction in alcohol prices on all-cause mortality by sex and age, natural logarithmic ARIMA models

	Monthly average	Estimate	Change		
			Percent level[a]	95% CI	Deaths per 100 000[b]
Men					
15–39 years	89				
Impact		−0.10	−9.2	−16.5 to −1.4	−1.0
Noise and Q(24)		ARIMA $(0,0,1)(0,1,1)_{12}$;Q(24) = 16.61; $P = 0.865$			
40–49 years	126				
Impact		−0.03	−2.8	−9.3 to 4.3	−0.9
Noise and Q(24)		ARIMA $(0,0,0)(0,1,1)_{12}$; Q(24) = 21.45; $P = 0.612$			
50–69 years	612				
Impact		0.02	2.0	−4.9 to 9.5	2.1
Noise and Q(24)		ARIMA $(0,1,1)(1,0,0)_{12}$; Q(24) = 20.74; $P = 0.659$			
>69 years	1164				
Impact		−0.07	−7.2	−13.0 to −1.5	−42.4
Noise and Q(24)		ARIMA $(0,0,2)(2,1,0)_{12}$; Q(24) = 20.98; $P = 0.640$			
Women					
15–39 years	31				
Impact		−0.09	−8.3	−11.3 to 0.0	−0.3
Noise and Q(24)		ARIMA $(0,0,0)(0,1,1)_{12}$; Q(24) = 27.19; $P = 0.296$			
40–49 years	53				
Impact		−0.06	−5.5	−11.9 to 1.4	−0.8
Noise and Q(24)		ARIMA $(0,0,0)(2,1,0)1_2$; Q(24) = 17.26; $P = 0.837$			
50–69 years	281				
Impact		−0.01	−0.6	−7.5 to 6.8	−0.3
Noise and Q(24)		ARIMA $(0,1,1)(2,0,0)_{12}$; Q(24) = 22.41; $P = 0.559$			
>69 years	1681				
Impact		−0.15	−14.0	−20.4 to −6.1	−68.5
Noise and Q(24)		ARIMA $(1,1,0)(2,0,0)_{12}$; Q(24) = 16.65; $P = 0.863$			

[a]Derived from exp(estimate).
[b]Obtained by multiplying the mortality rate before the change by the percentage change.
Q(24) is the Portmanteau test for residual autocorrelation with 24 lags.

Table 4 Summary evaluation of the reduction in the price of alcohol, monthly mortality rates per 100 000 person–years before (1996–February 2004) and the change[a] after the reduction (March 2004–06)

	Alcohol-related mortality			CVD disease mortality			All-cause mortality		
	Before	Change	95% CI	Before	Change	95% CI	Before	Change	95% CI
Men (years)									
15–39	4.4	−0.1	−0.6 to 0.5	0.8	0.01	−0.2 to 0.4	10.7	−1.0	−1.8 to −0.1
40–49	14.9	2.5	0.2 to 5.0	6.0	−1.2	−1.9 to −0.4	32.5	−0.9	−3.0 to 1.4
50–69	20.8	2.9	0.2 to 5.8	37.2	−1.7	−9.3 to 8.0	104.7	2.1	−5.1 to 10.0
>69	12.1	1.1	−0.5 to 2.8	284.4	−19.1	−35.3 to −2.0	615.0	−42.4	−79.9 to −9.2
Women (years)									
15–39	0.8	0.0	−0.2 to 0.2	0.4	0.0	−0.1 to 0.1	3.9	−0.3	−0.4 to 0.0
40–49	3.3	0.3	−0.1 to 0.8	2.0	−0.5	−0.8 to −0.1	14.0	−0.8	−1.7 to 0.2
50–69	3.9	1.6	0.3 to 3.2	11.8	−0.5	−2.2 to 1.6	45.5	−0.3	−3.4 to 3.1
>69	1.5	0.3	−0.3 to 1.2	245.8	−24.8	−33.2 to −16.0	489.1	−68.5	−99.8 to −29.8

[a]Derived from time series analyses.

study, since our method took account of trends and seasonal variation, and was based on a longer follow-up. A previous time series analysis covering a more limited set of alcohol-related causes, namely alcohol-positive sudden deaths, and with no age stratification, recorded an impact of eight additional deaths per week within 10 months of the price reduction,[6] more than twice our estimate of three additional acute deaths per week. This difference may be attributable to the longer study period after the price reduction in our study. Our findings are also in accord with those of an earlier US study based on annual state-level data on alcohol sales and mortality from 30 states in 1962–77 and concluding that increases in excise taxes on distilled spirits would reduce deaths from liver cirrhosis.[7]

A beneficial effect of the alcohol tax cuts due to a decrease in CVD mortality was estimated for men and women aged 40–49 and $\geqslant$70 years. Negative point estimates were recorded in most of the other groups, too. The effect was estimated to be largest in the former age group in relative terms and in the latter age groups in absolute terms. Including coronary operations as a control series in the models did not affect this finding.

There is an extensive body of data showing that light to moderate drinking ($\leqslant$1 drink daily for women and 1–2 drinks for men) is associated with cardioprotective benefits.[15,19] A larger effect on deaths due to IHD compared with other CVD causes lends credence to these results since cardioprotective effects have been reported to be most obvious in IHD.[15] However, a random distribution of insignificant negative and positive alcohol effect estimates was found in a time series study examining the association between alcohol consumption and mortality from IHD conducted in 15 European countries in 1950–95.[22] Differences in the scope of data account, at least partially, for the discrepancy in findings between this and our study. In contrast to the cross-European study, we did not include alcohol-attributable cases (17% of all CVD deaths), which were included in the models on alcohol-related deaths, in our analyses of CVD mortality. Furthermore, Hemström's study examined only age groups <75 years and covered smaller consumption changes overall, and may thus not have had capacity to detect effects that are mainly present in the older population.

The negative, i.e. beneficial point estimates found in the current study suggest that cheaper alcohol may, in addition to its harmful effects, also have fostered moderate consumption and its beneficial effects in at least some parts of the population. According to recent surveys, alcohol consumption in the 2000s has increased among persons aged >65 years[41] and among those aged 50–69 years[42] whose drinking is reported to be primarily low to moderate,[42] and thus beneficial in nature. In contrast, consumption did not increase among persons aged <50 years. Little is known about the amount of exposure time that is needed to achieve cardioprotective or other beneficial effects but there is no reason to assume that it is long at the population level. Liver cirrhosis mortality is a good point of comparison, which, despite of its long latency period, may respond almost instantaneously to changes in consumption.[5,43,44]

The estimated effect of the 2004 price change on all-cause mortality was beneficial in males and females aged >69 years as would be expected on the basis of the aforementioned results and the prominence of cardiovascular mortality at older ages. A recent meta-analysis of individual epidemiological studies suggested a J-shaped association between alcohol intake and total mortality demonstrating that moderate daily consumption was associated with a mortality reduction of 18%.[45] Previous time series research found an association between a 1-l increase or decrease in consumption and a corresponding increase or decrease of 1.3–3% in total mortality rates in separate analyses of 25 and 14 European countries and Canada, respectively.[20,46,47] Alcohol sales were used as a proxy for per capita consumption in all these studies. The results of the present study showed that the 1-l increase in per capita consumption in Finland that occurred in 2003–04 was associated with very little change in all-cause mortality among persons aged <70 years but a decrease among older Finns. In the study of 14 European countries,[46] the only one of these studies stratified by age, the estimates among persons aged $\geqslant$70 years were mainly non-significant and very close to zero in medium- and high-consumption countries, and small positive in low-consumption countries. This is the first aggregate level time series study to show a clear protective effect of changes in alcohol prices on mortality among those aged >69 years. One reason for the discrepancy with earlier studies may be that we estimate the effects of a single abrupt and large policy change rather than numerous, often smaller incremental changes over a longer follow-up period as is done in earlier studies. Moreover, annual data used in the earlier studies are short in terms of time series criteria,[39] but long in terms of historical time involving a risk of numerous uncontrolled confounding factors.

CVD mortality only partially captures the estimated overall beneficial effects of the tax change on all-cause mortality among those aged >69 years. We found evidence that in this age group there was a decrease also in COPD in both men and women, and further in mortality due to diabetes and dementia in women—all causes that have been associated with a protective effect of moderate alcohol consumption[15,19,23–28,48] research on the first-mentioned being scarce. No beneficial or harmful effect of tax change was observed for malignant neoplasms, i.e. a protective effect did not appear for this neutral cause of

death. With respect to possible confounders it can be said that the tax cuts in 2004 were specific to alcohol, but the opening of borders applied to cigarettes, too. However, this had very little effect on smoking rates. With regard to physical activity and diet, the change has echoed pre-existing favourable trends.[41,49] However, the possibility remains that the estimated reduction in all-cause mortality in particular, but also CVD, after the tax intervention of 2004 resulted from improvements in risk factors and treatment, even if the time series method should make such confounding relatively unlikely and despite the fact that we controlled for coronary operations.

One must be somewhat cautious in any interpretation of the favourable changes in CVD and all-cause mortality associated with a reduction in the price of alcohol. It is important to emphasize that alcohol-related death is specific (by definition) to alcohol and thus, understandably, responsive to changes in price of alcohol, whereas improvements in CVD mortality and other causes of death can be achieved through a multitude of other modifiable factors, such as an improved diet, physical activity and smoking cessation. For example, in Finland from the 1980s to the mid-1990s, risk factors including smoking, blood pressure and cholesterol, explained 53–72% and improved treatments 23% of the declining trends in CVD mortality.[50]

Conclusions

These results of the time series analyses show that the reduction in alcohol prices that occurred in Finland led to an increase in alcohol-related mortality, except among persons <40 years of age. It appeared, on the other hand, that those in the older age groups benefitted from cheaper alcohol in terms of decreased rates of CVD mortality in particular. Improvements in unobserved risk factors and treatment may have also affected the decreased rates of CVD and all-cause mortality to some extent. Accordingly, it is suggested that future comprehensive analysis of reductions in the price of alcohol should examine both the detrimental and beneficial consequences.

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KEY MESSAGES

- The reduction in alcohol prices that occurred in early 2004 in Finland led to an increase in alcohol-related mortality among persons aged 40–69 years.
- Beneficial effects in older age, when CVD deaths are prevalent, counter-balance these adverse effects, at least to some extent.
- Improvements in unobserved risk factors and treatment may have also affected the decreased rates of CVD and all-cause mortality in some measure.
- Future comprehensive analysis of reductions in the price of alcohol should examine both the detrimental and beneficial consequences.

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Commentary: Sinners, preachers and natural experiments

Mark Petticrew

Department of Social & Environmental Health Research, Faculty of Public Health & Policy, LSHTM, 15-17 Tavistock Place, London WC1H 9SH, UK. E-mail: mark.petticrew@lshtm.ac.uk

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Two papers in this issue demonstrate the utility of observational methods for evaluating the health effects of policies.[1,2] These are helpful as they show the value of such approaches and also identify some of the methodological and other challenges. They are also timely because debates about the need for more evaluations of public health policies and other types of intervention research have moved on significantly in the past 10 years. Macintyre recently noted that commentaries from the 1990's had pointed to the lack of robust evidence to support social and public health policies in the UK.[3] These highlighted the need for more robust and relevant evidence, and noted the lack of evaluations, particularly around health inequalities. This debate about gaps in the evidence base seems to have developed rapidly in recent years into discussions about the methodological implications of such gaps, and the challenges in producing new, reliable evidence. One important piece of methodological guidance to public health researchers in the UK emerged in 2000: this was the first edition of the Medical Research Council's (MRC) Guidance on complex interventions, which focused on the development and evaluation of complex public health interventions, and randomized controlled trials in particular.[4] The second edition of this guidance, which appeared 8 years later, however also considered the place of other types of evaluative research, including the use of time series analyses for evaluations of the impact of natural experiments, with detailed examples.[5]

The MRC has followed this up even more recently by exploring the need for guidance on the evaluation of natural experiments, and a recent report of a workshop makes the point that in some circumstances an ideal study design will not be possible. Observational methods will therefore be necessary, though the findings may often need treated with caution.[6] Although the biases in observational designs are well known, it is likely that in many cases the only evidence available will be this type of weaker evidence. Nonetheless, it plays an essential part. In public health, study design is frequently confounded with intervention type—so placing restrictions on the study designs that can be used in the interests of methodological rigour also inadvertently places a restriction on the types of intervention that can be evaluated; the best becomes the enemy of the good.[7] For example, a systematic review which examined the effects of transport-related interventions to improve physical activity found that population-level interventions were less likely than individual-level interventions to have been studied using the most rigorous study designs.[8] This review would have missed almost all