

Kimmo Herttua

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PRICE OF ALCOHOL ON ALCOHOL-RELATED
HARM IN FINLAND**

**A Natural Experiment Based on Register
Data**

Finnish Yearbook of Population Research
XLV 2010 Supplement

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ABSTRACT

Changes in alcohol pricing have been documented as inversely associated with changes in consumption and alcohol-related problems. Evidence of the association between price changes and health problems is nevertheless patchy and is based to a large extent on cross-sectional state-level data, or time series of such cross-sectional analyses. Natural experimental studies have been called for. There was a substantial reduction in the price of alcohol in Finland in 2004 due to a reduction in alcohol taxes of one third, on average, and the abolition of duty-free allowances for travellers from the EU. These changes in the Finnish alcohol policy could be considered a natural experiment, which offered a good opportunity to study what happens with regard to alcohol-related problems when prices go down. The present study investigated the effects of this reduction in alcohol prices on (1) alcohol-related and all-cause mortality, and mortality due to cardiovascular diseases, (2) alcohol-related morbidity in terms of hospitalisation, (3) socioeconomic differentials in alcohol-related mortality, and (4) small-area differences in interpersonal violence in the Helsinki Metropolitan area. Differential trends in alcohol-related mortality prior to the price reduction were also analysed.

A variety of population-based register data was used in the study. Time-series intervention analysis modelling was applied to monthly aggregations of deaths and hospitalisation for the period 1996–2006. These and other mortality analyses were carried out for men and women aged 15 years and over. Socioeconomic differentials in alcohol-related mortality were assessed on a before/after basis, mortality being followed up in 2001–2003 (before the price reduction) and 2004–2005 (after). Alcohol-related mortality was defined in all the studies on mortality on the basis of information on both underlying and contributory causes of death. Hospitalisation related to alcohol meant that there was a reference to alcohol in the primary diagnosis. Data on interpersonal violence was gathered from 86 administrative small-areas in the Helsinki Metropolitan area and was also assessed on a before/after basis followed up in 2002–2003 and 2004–2005. The statistical methods employed to analyse these data sets included time-series analysis, and Poisson and linear regression.

The results of the study indicate that alcohol-related deaths increased substantially among men aged 40–69 years and among women aged 50–69 after the price reduction when trends and seasonal variation were taken into account. The increase was mainly attributable to chronic causes, particularly liver diseases. Mortality due to cardiovascular diseases and all-cause mortality, on the other hand, decreased considerably among the-over-69-year-olds. The

increase in alcohol-related mortality in absolute terms among the 30–59-year-olds was largest among the unemployed and early-age pensioners, and those with a low level of education, social class or income. The relative differences in change between the education and social class subgroups were small. The employed and those under the age of 35 did not suffer from increased alcohol-related mortality in the two years following the price reduction. The gap between the age and education groups, which was substantial in the 1980s, thus further broadened. With regard to alcohol-related hospitalisation, there was an increase in both chronic and acute causes among men under the age of 70, and among women in the 50–69-year age group when trends and seasonal variation were taken into account. Alcohol dependence and other alcohol-related mental and behavioural disorders were the largest category in both the total number of chronic hospitalisation and in the increase. There was no increase in the rate of interpersonal violence in the Helsinki Metropolitan area, and even a decrease in domestic violence. There was a significant relationship between the measures of social disadvantage on the area level and interpersonal violence, although the differences in the effects of the price reduction between the different areas were small.

The findings of the present study suggest that that a reduction in alcohol prices may lead to a substantial increase in alcohol-related mortality and morbidity. However, large population group differences were observed regarding responsiveness to the price changes. In particular, the less privileged, such as the unemployed, were most sensitive. In contrast, at least in the Finnish context, the younger generations and the employed do not appear to be adversely affected, and those in the older age groups may even benefit from cheaper alcohol in terms of decreased rates of CVD mortality. The results also suggest that reductions in alcohol prices do not necessarily affect interpersonal violence. The population group differences in the effects of the price changes on alcohol-related harm should be acknowledged, and therefore the policy actions should focus on the population subgroups that are primarily responsive to the price reduction.

Keywords: alcohol drinking, price of alcohol, natural experiment, alcohol-related disorders, alcohol-related mortality, alcohol-related hospitalisation, cardiovascular disease, all-cause mortality, interpersonal violence, socioeconomic factors, education, income, social class, economic activity, neighbourhood characteristics.

LIST OF ORIGINAL PUBLICATIONS

- I. Herttua K, Mäkelä P, Martikainen P. Differential trends in alcohol-related mortality: A register-based follow-up study in Finland in 1987–2003. *Alcohol and Alcoholism* 2007; 42: 456–64.
- II. Herttua K, Mäkelä P, Martikainen P, Sirén R. The impact of a large reduction in the price of alcohol on area differences in interpersonal violence: a natural experiment based on aggregate data. *Journal of Epidemiology and Community Health* 2008; 62: 995–1001.
- III. Herttua K, Mäkelä P, Martikainen P. Changes in alcohol-related mortality and its socioeconomic differences after a large reduction in the price of alcohol: a natural experiment based on register data. *American Journal of Epidemiology* 2008; 168: 1110–18; discussion 1126–31.
- IV. Herttua K, Mäkelä P, Martikainen P. 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. *International Journal of Epidemiology*, 2009 Dec 7. Epub ahead of print.
- V. Herttua K, Mäkelä P, Martikainen P. The effects of a large reduction in alcohol prices on hospitalisations related to alcohol: a population-based natural experiment. Submitted.

1 INTRODUCTION

The World Health Organization has rated alcohol use eighth in the global risks for mortality and third for burden of disease as measured in disability-adjusted life years (WHO 2009). The price of alcohol has been reported to be associated with its consumption and alcohol-related problems on the population level: an increase in price tends to decrease consumption and related problems (Bruun et al. 1975; Edwards et al. 1994; Chaloupka et al. 2002; Babor et al. 2003; Trolldal and Ponicki 2005). Much of the evidence is based on cross-sectional state-level time-series data from the United States. The need for studies based on natural experiments, which take account of both change over time and differences between subpopulations, is obvious (Chaloupka et al. 2002; Peticrew et al. 2005). Natural experiments can be defined as studies which explore the consequences of a change (often abrupt) in policy or situation which was realised without a research or an evaluation in mind (Peticrew et al. 2005).

The reduction in the total price of alcohol realised at the beginning of the year 2004 in Finland was an event of interest even in the global context in terms of alcohol policy due to its uniqueness. It was expected to have considerable effects on alcohol consumption and alcohol-related harm in terms of mortality, morbidity and violent crime, for example.

Socio-demographic differentials in terms of gender, age and socioeconomic factors are well-established in alcohol-related problems and alcohol consumption (Edwards et al. 1994; Room et al. 2005). However, evidence is scant as far as the differential effects of changes in alcohol prices on harm are concerned.

The aim of this study is to investigate, in broad terms, the effects of the reduction in the price of alcohol in Finland in 2004, which was followed by an increase of 10 per cent in alcohol consumption, i.e. in a natural experimental setting, on health and crime in terms of interpersonal violence on the population level, and how these effects varied according to different socio-demographic factors and neighbourhood characteristics.

2 THE FINNISH CONTEXT – HISTORICAL BACKGROUND

2.1 A short history of Finland's alcohol policy until 1969

Finland – alongside Sweden and Norway – has a long history of an exceptionally restrictive alcohol policy on the European level. This is a consequence of the fact that the government has been a strong actor on the European scale in its implementation. The production of alcohol in terms of its manufacture, import, export, and wholesale and retail sale has been in the hands of the private sector in Europe, whereas it was monopolised for the most part in Finland after the abrogation of prohibition in 1932 until 1995 when a new alcohol law came into effect (Holder et al. 1998, 49–50).

Distilling spirits at home was prohibited and controls covering the manufacturing of spirits for commercial purposes were introduced in 1866. Legal trade in all alcohol except beer was ceased almost entirely of the end of 19th century, partly because landowners wanted to keep control over their workforce (Peltonen 1997, 45–59; Holder et al. 1998, 51–52). Local government on the county level, being under the control of landowners until the Civil War of 1918, was able to keep the countryside dry by prohibiting the sale of all alcohol including beer (Peltonen 1997, 54–58; Apo 2001, 208). Consequently, in 1901 beer was only available in the inns of six rural counties (Peltonen 1988, 17). However, the consumption of home-made distilled spirits was declining even before the ban of 1866 for economic, social and cultural-historical reasons (Apo 2001, 207). Dryness in the countryside is of significance because a major proportion of the Finnish people lived there even in the 1960s. It should also be noted that both on- and off-premise retail trade in alcoholic beverages was pursued in towns.

A law on prohibition, supported by an overwhelming parliamentary majority was considered on two separate occasions in the first decade of the 1900s. However, in both 1907 and 1909 the legislative proposals were not carried due to opposition in the senate, and were therefore never ratified by the tsar (Johansson 2000, 24). Despite the efforts of parliament to attract support, it was not until after the February Revolution of 1917 that the Kerensky government, on 29 May, adopted the Finnish prohibition law. The Prohibition came into effect two years later, in accordance with the 1909 proposal. Thus on 1 June 1919 a general, legislated spirits prohibition (production, import, transport, sales and storage) related to alcoholic beverages of over two per cent in volume was introduced in the new Republic of Finland (Johansson 2000, 24). The prohibition was short-lived where it succeeded.

There was total prohibition in Finland in 1919–1932, prohibition was rejected by referendum in Sweden in 1922, and in Norway partial prohibition affecting spirits and fortified wines was in effect in 1916–1923, and for spirits alone until 1927. Furthermore, Iceland and the Soviet Union also had prohibition in some measure in 1915–1935 and 1914–1925, respectively (Karlsson 2000, 296; Segal 1987, 118–121). Illegal forms largely replaced legal consumption in Finland, as large quantities of alcohol were smuggled in mainly from Germany, and also from Estonia at the beginning of the prohibition period. Regardless of several reforms comprising the introduction of more severe punishments and more strict control, alcohol abuse and its consequences were on the increase (Holder et al. 1998, 53). Moreover, when the severe economic crisis took hold at the turn of the 1920s and 1930s, economic arguments were put forward. Consequently, the repeal of prohibition was supported so that alcohol taxes could be introduced in order to build up the state coffers. Eventually, almost 70 per cent of the population voted in favour of repeal in the consultative referendum of December 1931, and the alcohol retail shops were reopened in April 1932 (Kallenautio 1981; Johansson 2000, 24–25).

A law regulating the trade of alcohol of more than 2.8 per cent by volume replaced the prohibition law. All trade was monopolised and turned over to the state company, Alko. An administrative board with extensive powers was appointed by the government to manage the company, which had autonomy in establishing price levels, among other things. The company handled imports and sales, whereas production and distribution could be transferred to the private sector. The production of malt drinks was handed over to privately owned breweries (Alkoholikomitea 1946). Furthermore, the state also controlled the production and trade of alcohol beyond the monopoly – such as beer brewing and some of the on-premise retail trade – very strictly by means of a licensing system (Holder et al. 1998, 53; Österberg and Karlsson 2002, 16). The peculiarities regarding the regulation included keeping the countryside dry by permitting on- and off-premise retail trade only in towns (Apo 2001, 213).

Reforming the 1932 alcohol law came under debate after World War II. However, it was not until 1968 when the legislation was liberalised to a great extent, even if the monopoly system basically remained untouchable. The most significant change concerned medium-strength beer (which contains less than 4.7 per cent alcohol by volume) when the new law came into effect at the beginning of 1969. After that, on- and off-premise retail trade in medium-strength beer was extended to cafeterias and grocery stores, which meant that the countryside was no longer dry. The minimum legal ages for alcohol purchase were also dropped from the previous uniform age of 21 to 18 years for beer, wine and other beverages with an

alcohol content of less than 22 per cent by volume, and to 20 years for stronger beverages (Holder et al. 1998, 54).

2.1.1 The origins of the restrictiveness in Finland's alcohol policy

Explanations of the essence of the Finnish alcohol policy are not straightforward. A few studies in the field of history (see e.g. Sulkunen 1985; Johansson 2000; Peltonen 2002) have addressed this intriguing phenomenon, which is unique in terms of restrictiveness – alongside Norway and Sweden – from the European perspective. As a matter of fact, it would not be possible to conduct this study if the Finnish alcohol policy was not as strongly in the hands of government in terms of using tax as a control instrument as it was, and in point of fact still is in the 2000s. In a comparison of alcohol-policy strictness of covering 15 western European countries in 2005, the Finnish policy was rated third after Norway and Sweden and sharing the same level, but with a large gap ahead of the fourth, France (Anderson and Baumberg 2006).

Common Nordic features

According to Johansson (2000, 17–19), Protestantism, a spirits culture and state instruments for implementation were the necessary factors behind the English-speaking and Nordic alcohol cultures, but fall short of explaining the establishment of the Nordic control systems. More factors are needed to account for the Nordic uniqueness in terms of restrictiveness (Johansson 2000, 17–19). The basic elements of the control systems and the principles behind eliminating private profit motives that were introduced and accepted in Finland (as well as in Norway) were a Swedish creation (Johansson 2000, 41–42). It is reasonable to assume that the introduction and preservation of the Nordic control systems were rooted in a temperance-friendly and politically influential labour movement, the feeble economic contribution of the alcohol industry, a low level of urbanisation and a dominant rural culture, the lack of an everyday drinking culture, and extensive political engagement among women (Johansson 2000, 34–40).

These roots of the Nordic control system warrant a brief explanation. First, the labour movement and social democracy played crucial roles in establishing and maintaining prohibition and alcohol-related restrictions. The social-democratic commitment to the modern project through state control and social intervention provided a framework and the methods for achieving a restrictive alcohol-control system (Sulkunen 1985; Johansson 2000, 34–35). Secondly, the relatively weak economic importance of the production, distribution and sale of alcohol created a space for state intervention and restriction (Sulkunen 1985; Johansson 2000, 42).

Thirdly, the rural culture and lifestyles dominated. The absence of inebriant-liberal urban environments lasted long into the post-war period, was significant in the establishment of restrictive alcohol-control policies, their preservation and social acceptance (Sulkunen 1985; Johansson 2000, 31, 42). Fourthly, due to the absence of an everyday drinking culture and social forms of drinking, alcohol never became a meaningful element of the symbolic language of society or its cultural system (Sulkunen 1985; Johansson 2000, 30–31). It was rather associated with transgressing boundaries, hedonism and intoxication, which required external control. The negative symbolic pictures of alcohol as the root of all evil painted by the temperance movement were strong, and provided political space for and the social acceptance of control, discipline and restriction (Sulkunen 1985; Johansson 2000, 42).

Finally, the role of woman has been crucial in terms of restrictiveness (Johansson 2000, 37–38). Women often constituted the majority of members in the temperance movement. They had lower alcohol consumption than men – as a matter of fact drinking was very unusual for them (Apo 2001, 241, 246, 359) – and many of the politically active women's organisations supported prohibition and other restrictions on alcohol. Women were clearly more in favour of prohibition than men. Still, their lower alcohol consumption and generally more positive position on prohibition and restriction is a general phenomenon and cannot explain the existence of control systems in the Nordic countries (Johansson 2000, 37–38). The essential difference is the strong political position Nordic women had (and still have) compared to the rest of world. For example, Finland was the first nation in Europe to introduce women's suffrage as early as in 1906. Overall, it could be argued that extensive organisation, mobilisation and political consciousness among Nordic women created a political space and more widespread acceptance of control-oriented decisions in the alcohol-related political arena (Rose 1996; Johansson 2000, 37–38).

Special Finnish features

A few studies in the field of social history shed further light on the origin of the restrictiveness of the Finnish alcohol policy. It is paradoxical that when alcohol consumption per capita was among the lowest in Europe at 1.1–2.9 litres in 1871–1968, the Spirit Problem was considered a big issue that was assumed to call for austere measures. Although sufferers from serious alcohol problems were few in number, the Finnish people were stigmatised for not being able to consume alcohol in a moderate and responsible way. The discriminatory concept of the Finnish inherited disposition for alcohol abuse was thus established at the beginning of the 20th century (see e.g., Peltonen 1997, 71–81; Apo 2001, 387).

According to Peltonen (1997, 71–81) and Apo (2001, 387–8), explanations for this concept which led to one of the most restrictive alcohol policies in the Western world are to be found in the threats and complications related to building the Finnish nation at the turn of the 20th century. Most of the nationalists belonging to the Swedish-speaking middle class had a remote and ambivalent relation to the peasantry. On the one hand they were idealised in the spirit of nationalistic writers such as Runeberg and Topelius, and on the other hand they were regarded as a backward and primitive mass, which only through the Enlightenment would become honourable citizens serving society. At the same time, Finland was in the grip of the most severe social problem in its history, namely poverty among the peasantry. The nation builders' biggest concerns, i.e. poverty and the backwardness of the people, resulted in the conception of a negative identity: Finland was less developed than other West-European nations. In order to overtake the others, it had to reject everything that prevented it from advancing towards virtuous citizenship and establishing a powerful fatherland. The model of virtuous citizenship originated in West-European idealism. It had to be applied in the strictest sense in the Finnish context due to the backwardness of the Finnish people (Peltonen 1997, 71–81; Apo 2001, 387–8).

The official discourses considered alcohol consumption among the masses the enemy of progress and the concrete symbol that crystallised the poverty and primitiveness of the people (Peltonen 1997, 81; Apo 2001, 388–389). The elite did not rely on people's ability to regulate their drinking, nor did not the labour party or the labour movement, which took an anti-alcohol stance in their programme. The most recent research has called into question the stereotypes picturing the lowest social classes as pauperised by heavy drinking. There is no evidence meeting the criterion of reliability on extensive alcohol abuse at the turn of the 20th century (Peltonen 1997, 11, 71–81; Apo 2001, 387–389).

2.2 The liberalisation of Finland's alcohol policy after 1969 – and the year 2004

The next turning point in the liberalisation process after 1969 was at the beginning of 1995. A new alcohol law was passed in order to bring Finland in line with the European Union in terms of alcohol policy and legislation. The minor legislative changes between 1969 and 1994 mainly increased availability, but the new law demolished the state monopoly on the production, import, export and wholesale trade of alcoholic beverages, but left the monopoly on off-license retail trade almost untouched except for beverages with an alcohol content of 4.7 per cent or less, which could also be sold in kiosks and service stations. The new law in

1995 also affected import quotas in the form of slight changes in the amount of alcohol travellers were allowed to import for their own use (Holder et al. 1998, 54–55; Alavaikko and Österberg 2000).

The year 2004 was a true milestone in the liberalisation process. The changes that occurred that year could be considered a natural experiment. Following the deregulation of import quotas within the European Union (EU) on January 1st it was 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 per cent on March 1st: the off-premise retail price of spirits went down by 28–36 per cent depending on the type, wines by three per cent, beers by 13 per cent, and other alcoholic beverages between seven and 28 per cent (Mäkelä and Österberg 2009). This was the first time after the mid 1970s when the real price indices for the sales of alcoholic beverages decreased. They were rising until the beginning of 1990s and then remained stable until 2004 (Stakes 2007). The reason for the tax cuts was that Estonia joined the EU on May 1st, and this had a great impact on the Finnish alcohol market because of the proximity of the two countries and the significantly lower price of alcohol in Estonia. All in all, these three changes brought about not only a large reduction in the nominal price of alcohol but also a significant reduction in the full price because, on top of the nominal price, the indirect costs fell due to the abolition of duty-free allowances for travellers. The estimated total per-capita alcohol consumption (recorded and unrecorded) was 9.4 litres per inhabitant in Finland in 2003. There was an estimated 10-per-cent increase in 2004, to 10.3 litres, after which consumption has remained roughly on that level (Stakes 2007).

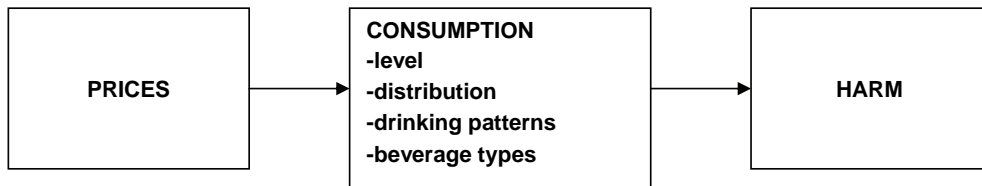
3 THE EFFECTS OF ALCOHOL PRICING ON ALCOHOL-RELATED HARM: THEORY AND EMPIRICAL EVIDENCE

This chapter reviews the recent research literature on the effects of alcohol pricing on alcohol-related harm. However, given that the main mediating factor between pricing and problems is consumption, research on the associations between pricing and consumption on the one hand, and between consumption and harm on the other, is also reviewed. Moreover, previous studies on socioeconomic differentials regarding these relationships are assessed. The theory and the empirical evidence are discussed in parallel.

3.1 Alcohol prices and consumption

Change in the price of alcohol does not necessarily entail any changes in the consequences of drinking, and it needs a mediating factor so as to have any effect. This mediating factor is alcohol consumption, which has at least four elements that may change in response to price changes and potentially affect alcohol-related harm (Figure 1). The first of these is the level of alcohol consumption, which is usually quantified as per-capita consumption on the population-level. The change in this aspect of consumption as a response to prices changes is the best documented (e.g., Edwards et al. 1994). Secondly, price changes potentially affect the distribution of consumption in the population. One of the few regularities in this distribution is its skewness (Ledermann 1956, 1964; Skog 1985, 1993; Duffy 1986; Edwards et al. 1994), measured in terms of the proportion of the population drinking more than twice the average consumption, for example (Edwards et al. 1994). This figure has been found to be markedly stable across drinking cultures, and typically varies between 10 and 15 per cent of the population (Skog 1985). The harmful effect of changes in per-capita consumption typically depends on how they are distributed in the population. Thirdly, changes in alcohol prices may have effects on drinking patterns, although such societal changes are typically slow (e.g., Edwards 1994). Fourthly, there may be changes in beverage choice, particularly in the case of differential price changes. All these patterns may be intertwined and may be in progress simultaneously.

Figure 1. Alcohol prices, consumption and harm.



There have been several systematic reviews of the association between alcohol prices and consumption. The main conclusion Edwards et al. (1994, 121) reached in their comprehensive review of alcohol issues was that, “Other things being equal, a population’s consumption of alcohol will to a lesser or greater but usually significant degree, be influenced by price”. Cook and Moore (2002), in turn, concluded that “Estimated elasticities for beer, wine, and spirits differ widely over time, place, data set, and estimation method, but one conclusion stands out: In almost every case the own-price elasticities are negative”. In other words, consumption tends to decrease when prices increase, and vice versa, when other factors (income, for instance) remain stable. A few recent meta-analyses have mainly confirmed these findings (Fogarty 2006; Gallet 2007; Booth et al. 2008; Wagenaar, Salois et al. 2009).

A natural experiment type of study from Sweden using price and sales data for the years 1984–1994 showed that consumers responded to price increases by altering their total consumption and by varying their brand choices (Gruenewald, Ponicki et al. 2006). In July 1992 the Swedish alcohol retail monopoly Systembolaget implemented a common schedule according to which all beverages were taxed per unit of liquid volume rather than as a percentage of the pre-tax price, as had previously been the case for spirits and wine. It appeared that consumer behaviour was quite responsive to changes in beverage prices: rather than simply lowering the quantity consumed drinkers appeared willing to switch to lower-cost brands in order to maintain their consumption level (Gruenewald, Ponicki et al. 2006). A price increase may thus induce consumers of high-quality brands to switch to less costly alternatives, or to switch purchases to venues in which alcohol is less costly, whereas those who were already drinking the lowest-priced beverages could only respond by changing quantity rather than quality (Gruenewald, Ponicki et al. 2006). This suggests that certain groups of consumers may be systematically more responsive to price changes than others, including those with fewer opportunities to reduce usage costs (Gruenewald, Ponicki et al. 2006). An earlier study, which was also based on data provided by Systembolaget, examined the effects on alcohol sales within the three beverage classes (beer, wine and spirits) in the

same situation as above, in other words the tax was purposely linked to alcohol content (Ponicki et al. 1997). The most notable effects of the taxation change were a substantial compression of the range of prices for spirits and wine and a corresponding expansion of the price spectrum for beer. This study also confirmed the finding that consumers may respond to tax changes by shifting away from beverage brands that become relatively more expensive (Ponicki et al. 1997).

Another event that could be called a natural experiment was when Switzerland implemented a reform of taxation on spirits in 1999 in accordance with the World Trade Organization agreement on the elimination of discriminatory duties on foreign spirits. This resulted, with the liberalised import of spirits due to the fiscal reform, in a reduction of 30–50 per cent in the retail price of foreign spirits. However, prices of domestic spirits did not change (Heeb et al. 2003). A longitudinal survey-based study examining the impact of this change and using a before/after design reported an increase in spirits consumption (domestic and foreign spirits were not separated) in all age groups except the over-60s, which persisted even after adjustment for significant correlates of spirits consumption (Kuo et al. 2003).

With regard to liberalisation on the Nordic level, a survey-based study examined short-term changes in alcohol consumption among subgroups of the population in Denmark, Finland and southern Sweden following large-scale decreases in alcohol taxation in Denmark and Finland in 2004, and large increases in travellers' allowances in Finland and Sweden (Mäkelä et al. 2008). Reported consumption decreased or remained the same among women and men in all three study sites. The relative changes were similar across the age, gender and income subgroups in all countries. In absolute terms, there was a differential trend by age in Denmark, Finland and southern Sweden, with the lower consumption level of the older age groups and the higher consumption level of the younger age groups converging. Women's and men's consumption converged in Finland and southern Sweden. These results did not confirm expectations: no increase in consumption larger than that in the control site (which was northern Sweden) was found in any of the countries or population subgroups. However, consumption information obtained from the survey diverged clearly from the per-capita statistics in Finland (Mäkelä et al. 2008).

There is little evidence of differential responsiveness in terms of consumption to the price of alcohol by sex and age. However, youths and young adults have been shown to be generally more responsive to price increases than older adults (Chaloupka et al. 2002). For example, a recent survey-based UK study found a strong relationship between teenagers' disposable income and their likelihood of

binge drinking (Bellis et al 2007). However, another survey-based study from the U.S. indicated that the drinking practices of male college students were generally insensitive to the price of beer, whereas underage drinking and binge drinking by female students did respond to price, even if the effects were generally small (Chaloupka and Wechsler 1996).

Earlier studies using cross-sectional survey data on alcohol consumption and individual characteristics linked to alcohol prices in the US have yielded contradictory results on whether price responsiveness is different in various user groups: Manning et al. (1995) concluded on the basis of survey data collected in 1983 that heavy (and light) drinkers were much less price-responsive than moderate drinkers, whereas a more recent study also based on survey data gave evidence of substantial price responsiveness among heavy drinkers with symptoms of alcohol abuse or dependence (Farrell et al. 2003). Methodological differences lie behind these discrepancies, at least to some extent (see Farrell et al. 2003).

In sum, it appears that there is evidence that alcohol consumption is affected by prices: higher prices are related to a lower level of consumption and lower prices to a higher level. In addition, consumers tend to respond to rising prices by shifting away from beverage types or brands that become relatively more expensive, or by switching purchases to venues in which alcohol is less costly. The question remains whether men and women, younger and older people, and heavy, moderate and light drinkers are equally sensitive to changes in price.

3.2 Alcohol consumption related to health and violence

More than 60 health consequences have been identified for which a causal link between alcohol consumption and the outcome can be assumed (Gutjahr et al. 2001; Rehm, Gmel et al. 2003; Rehm et al. 2003; Corrao et al. 2004; Murray and Lopez 1997; Murray et al. 2004). Most effects of alcohol on disease have been reported to be detrimental, but for certain patterns of drinking, a beneficial influence has been observed (Gutjahr et al. 2001; Rehm, Gmel et al. 2003; Rehm, Room et al. 2003; Corrao et al. 2004; Grønbaek 2009). Major directly alcohol-attributable diseases include alcohol liver disease, pancreatitis and alcohol dependence.

It is not only the level but also the pattern of drinking that matter in terms of linking alcohol consumption to its outcome. The same average volume of alcohol (e.g., two drinks a day) can be consumed in relatively small quantities (e.g., two drinks a day with meals) or in large quantities on a few occasions (e.g., two bottles of

wine on a single occasion every Friday), and there are different health implications. The data on the effects of drinking patterns is less abundant than data on overall consumption, but evidence is accumulating that such patterns affect the link between alcohol and both disease and mortality (Rehm, Rehn et al. 2003; Paljärvi et al. 2005, 2009). In other words, the effects of the average volume of consumption are somewhat moderated by the way alcohol is consumed, which in turn is influenced by the cultural context (Room and Mäkelä 2000; Rehm, Rehn et al. 2003).

3.2.1 Disease

Diseases directly attributable to alcohol

Although the relation between alcohol abuse and diseases directly related to alcohol is well-established on the individual level (Rehm et al. 2003; Corrao et al. 2004), research is sparse on the population level. The distinction between the two is worth clarifying here. On the individual level it is a question of the extent to which alcohol affects mortality risk, for example, whereas on the population (or aggregate) level the interest is in the extent to which changes in overall alcohol consumption in society affect mortality rates (Norström and Skog 2001). Implicit idea in the latter is that the level of alcohol consumption is something that can be affected by alcohol policy. It must be noted that for a population-level association between per-capita consumption and harm to exist there has to be a similar association on the individual level. However, this condition is not sufficient because there may also be other influencing factors, such as distribution of alcohol consumption, for instance, that may influence the association between population level consumption and harm. Consequently, findings obtained on the population level cannot and need not test the association on the individual level.

Before considering the population-level evidence, it is of great importance to briefly review some fundamental features based mainly on individual-level studies, of three major directly alcohol-attributable diseases: alcohol liver disease, pancreatitis and alcohol dependence. Alcohol liver disease is a major source of alcohol-related morbidity and mortality (e.g., Mann et al. 2003). The most prevalent types of alcoholic liver disease are fatty liver, alcoholic hepatitis and cirrhosis. The trend among people who continue to drink heavily is to progress from fatty liver to hepatitis to cirrhosis. However, the disorders may also occur simultaneously (Kirsch et al. 1995; Mann et al. 2003). It is estimated that between 10 and 15 per cent of alcoholics will develop cirrhosis (Anand 1999). The likelihood of developing alcoholic liver disease is, to a great extent, a function of both

the duration and the amount of heavy drinking (Lelbach 1974; Mann et al. 2003), and it is suggested that cirrhosis does not develop below an average daily intake of 30 grams (between two and three drinks) of alcohol (Bellentani and Tiribelli 2001; Mann et al. 2003). Furthermore, some studies have proposed that cirrhosis mortality is more strongly associated with the consumption of spirits than with other alcoholic beverages (Roizen et al. 1999; Kerr et al. 2000) and that consuming alcohol with food is less risky than consuming it in isolation (Bellentani and Tiribelli 2001). Cirrhosis mortality rates vary substantially among age groups: they are very low among the young but increase considerably in middle age, reaching a peak among people aged between 75 and 84 (Mann et al. 2003). Significant differences in the rates of alcoholic liver disease have also been found in men and women, and among different ethnic groups (Tuyns and Pequignot 1984; Stinson et al. 2001; Mann et al. 2003).

Another major directly alcohol-attributable disease, pancreatitis, (i.e., inflammation of the pancreas) takes two forms: acute and chronic. Acute pancreatitis is defined as an acute inflammatory process that frequently involves peripancreatic tissues and/or remote organ systems, whereas the chronic form leads to the progressive and irreversible destruction of exocrine and endocrine glandular pancreatic parenchyma which is substituted by fibrotic tissue. As a result, a series of morphologic and functional changes occur that produce several symptoms (Bornman and Beckingham 2001; Etemad and Whitcomb 2001; Strate et al. 2002; Witt et al. 2007; Spanier et al. 2008; Irving et al. 2009). The two most common etiological factors of acute pancreatitis are gallstones and alcohol abuse (Banks 2002; Whitcomb 2006; Kempainen and Puolakkainen 2007; Forsmark and Bailie 2007; Pandol et al. 2007), which together represent more than 80 per cent of cases (Irving et al. 2009). However, Lankisch et al. (2002) suggest that the risk of developing the condition among heavy drinkers (>60g per day for 20–30 years) is only two or three percent. With regard to chronic pancreatitis however, alcohol abuse is the major cause in Western countries, accounting for approximately 70–80 per cent of all cases (Etemad and Whitcomb 2001; Banks 2002; Dufour and Adamson 2003; Witt et al. 2007; Mayerle and Lerch 2007). Morbidity rates for acute, but not for chronic, pancreatitis increase with age (Lankisch et al. 2002; Tinto et al. 2002; Levy et al. 2006; Fagenholz et al. 2007). Most studies report that the median age for the first attack of acute pancreatitis is in the sixth decade of life, whereas the peak incidence of the chronic form is between the fourth and sixth decade (Irving et al. 2009). Male morbidity is higher than female morbidity for both forms of the disease. The dose-response relationship between the average volume of alcohol consumed and pancreatitis has been found to be approximately exponential, the threshold being about four daily drinks (Irving et al. 2009).

The third major category of directly alcohol-attributable diseases is alcohol dependence, also known as alcohol dependence syndrome or simply alcoholism. Despite the general conviction that it is a unitary phenomenon, there is ample evidence that people with alcohol dependence differ with respect to a variety of demographic, personal and clinical characteristics (Epstein et al. 2002; Windle and Scheidt 2004; Babor and Gaetano 2006; Leggio et al. 2009). Sufferers thus differ in many traits, such as age at the onset of heavy drinking (early or late), patterns of drinking (e.g., continuous or binge), rate of alcohol metabolism, sensitivity to intoxication, rapidity of progression to medical problems, and the presence or absence of co-occurring psychiatric illness (Leggio et al. 2009). Despite the heterogeneity, some average ages have been proposed regarding the course of alcohol dependence: the usual age at onset is 23–33 years, the usual age for seeking treatment is 40, and the usual age of death is 55–60 (Schuckit 2000).

On the population level, much of the evidence comes from a project entitled the European Comparative Alcohol Study (ECAS), which involved a number of time-series studies on alcohol sales and mortality due to different causes. Data covering the period from the 1950s to the mid-1990s were obtained from 14 Western European countries and Canada (in some cases) (Norström 2002).

One of the ECAS studies demonstrated a positive and statistically significant effect of changes in per-capita consumption in the period 1950–1995 on changes in cirrhosis mortality in 12 out of 14 Western European countries among men, and in nine countries among women. Moreover, when different age groups were analysed significant estimates were obtained in 29 out of 42 strata defined by three age groups and 14 countries for men, and in 20 out of 42 strata for women. Most of the significant estimates were found among persons aged 45–64 years (Ramstedt 2001). Another study involving the same countries tested whether there was a relation between alcohol consumption and pancreatic mortality. On average, depending on the model employed, statistically significant positive estimates were found in nine of the countries. The analyses did not produce a single positive estimate for Finland, Italy or Canada (Ramstedt 2004).

Cardiovascular diseases

Apart from its adverse effects on health, alcohol consumption may also have beneficial effects. There is a large body of epidemiological evidence that low-to-moderate consumption is associated with a reduced risk of cardiovascular and all-cause mortality on the individual level; a J-shape curve thus illustrates the relation between consumption and mortality (Wannamethee and Shaper 1999; Corrao et al. 2000; Rehm, Gmel et al. 2003; Rehm, Room et al. 2003; Reynolds et al.

2003; Freiberg and Samet 2005; O'Keefe et al. 2007). This association has several biologically plausible mechanisms with the dose-dependent effects of alcohol to increase levels of high-density lipoprotein cholesterol, to lower levels of low-density lipoprotein cholesterol and of plasma fibrinogen, inhibit platelet aggregation and enhance insulin sensitivity (Puddey et al. 1999; Agarwal 2002; Burger, Mensink et al. 2004; O'Keefe et al. 2007). Thus, alcohol reduces the risk of coronary vascular diseases by inhibiting the formation of atheroma and by decreasing the rate of blood coagulation (Agarwal 2002; Burger, Mensink et al. 2004).

Several reviews and other studies on the individual level have contributed to the specification of the relation between consumption and mortality. A J-shaped relation was observed for ischemic heart disease with a minimum relative risk of 0.80 at 20 g/day, a significant protective effect at up to 72 g/day, and a significant increased risk at 89 g/day in a meta-analysis on 156 studies (Corrao et al. 2004). A meta-analysis on experimental studies suggested that thirty grams of alcohol a day would cause an estimated reduction of 25 per cent in the risk of ischemic heart disease (Rimm et al. 1999), whereas another meta-analysis concluded that the risk was lowest among men drinking up to 30 and women drinking 10–20 grams of alcohol/day (Burger, Brönstrup et al. 2004). The beneficial effects have been found to be more pronounced among older men (Burger, Brönstrup et al. 2004). A Whitehall II Cohort Study examining the relationship between consumption and both ischemic heart disease and all-cause mortality found that the optimal frequency of drinking was between once or twice a week and daily, after adjustment for average volume consumed per week. Those drinking twice a day or more had a more than twofold increased risk of mortality compared to those drinking once or twice a week. Drinking only once a month or only on special occasions had a 50-per-cent increased risk of mortality (Britton and Marmot 2004). Another study from the Whitehall II Cohort found a significant cardioprotective benefit of moderate drinking compared with abstinence or heavy drinking among those with poor health behaviours (little exercise, poor diet and smokers). No additional benefit from alcohol was found among those with the healthiest behaviour profile (Britton et al. 2008). A recent review concluded that it is not only the quantity, but also drinking patterns and genetic factors that may influence the relation between alcohol consumption and cardiovascular diseases (Djoussé and Gaziano 2008).

However, there are few population-level studies on the association between alcohol consumption per capita and cardiovascular diseases. An ECAS time-series study on consumption and ischaemic heart disease mortality in the period 1950–1995 reported a random distribution of insignificant negative and positive alcohol-effect estimates. A slight indication of a cardioprotective effect among 30–44-year-old

women in high-consumption countries was observed. Unlike in the other ECAS studies, no pooled estimates were presented (Hemström 2001).

Not all researchers are convinced by the evidence on the cardioprotective effects of alcohol. One group set out to show that there may be a systematic error in prospective epidemiological mortality studies reporting “light” or “moderate” regular use of alcohol to be “protective” against coronary heart disease. It has been suggested that people decrease their alcohol consumption as they age and become ill or frail, or increase their intake of medications, and some abstain from alcohol altogether. If these people are included in the abstainer category in prospective studies it is reasoned that it is not the absence of alcohol that elevates their risk of ischemic heart disease but rather their ill health. The authors call for studies on ischemic heart disease mortality that use lifelong abstinence as the reference point for estimating ischemic heart disease protection (Fillmore et al. 2007; Stockwell et al. 2007). Accordingly, a prospective cohort study from Australia indicated that, compared with life-time abstention, regular daily alcohol intake was associated with a lower risk of mortality due to cardiovascular disease and ischemic heart disease among women but not among men (Harriss et al. 2007). Poikolainen et al. (2005), in turn, evaluated whether confounding by several known or suspected coronary-heart-disease risk factors such as body mass index, smoking or physical activity was likely to explain the lower disease risk among light alcohol drinkers compared with never-drinkers. They concluded that none of the risk factors studied was a likely candidate for an unknown confounder. These results thus rule out several alternative explanations of the alcohol and coronary heart disease association between light drinkers and never-drinkers (Poikolainen et al. 2005).

There is also a large body of individual-level epidemiological evidence demonstrating a J-shaped or U-shaped association between alcohol consumption and stroke, which implies that low-to-moderate levels of consumption have a protective effect on cerebral vasculature, whereas heavy consumption predisposes to both hemorrhagic and non-hemorrhagic stroke (Gill et al. 1986, 1988, 1991; Shaper et al. 1991; Reynolds et al. 2003; Mukamal et al. 2005). This protective effect was detected in both younger and older groups (65 years as a dividing age), among men and women, and among whites, blacks and Hispanics (Sacco et al. 1999).

Diabetes mellitus, dementia and respiratory diseases

Evidence of the association between alcohol use and diabetes mellitus comes purely from individual-level studies. A meta-analysis based on 32 studies comparing abstinence with moderate consumption (one to three drinks per day) found that moderate consumption was associated with a 33-to-56-per-cent lower incidence

of diabetes and a 34-to-55-per-cent lower incidence of diabetes-related coronary heart disease. Compared with moderate consumption, heavy consumption (more than three drinks a day) may be associated with up to a 43-per-cent increased incidence of diabetes (Howard et al. 2004). Another meta-analysis investigated the relationship between alcohol consumption and long-term complications of type 2 diabetes. The authors concluded that, as with findings covering the general population, moderate alcohol consumption is associated with a lower risk of total mortality and ischemic heart disease in type-2 diabetic populations (Koppes et al. 2006). Moreover, beneficial effects of low-to-moderate consumption have been reported with regard to some other conditions such as dementia and respiratory diseases, particularly chronic obstructive pulmonary diseases (Tabak, Smit, Heederik et al. 2001; Tabak, Smit, Räsänen et al. 2001; Ruitenberg et al. 2002; Mukamal et al. 2003; Doll et al. 2005; Deng et al. 2006).

3.2.2 Accidents and violence

Evidence on the association between alcohol consumption and both accidents and violence is scarce and weak on the population level, and is mostly based on time-series analyses. A few ECAS time-series studies based on data from 14 European countries found that total alcohol sales were positively and statistically significantly associated with homicide rates in 21 per cent of the strata defined by these countries, age and sex (Rossow 2001), and with fatal accidents in 21 per cent of the strata, too (Skog 2001). Another ECAS study found that alcohol sales were positively associated with suicide rates in 14 per cent of the strata also defined by age, sex and country (Ramstedt 2001).

A Canadian study covering the period 1968–1991, reported included only 24 time points, found a positive relationship between alcohol sales and homicide rates (Mann et al. 2006). An association between alcohol consumption and both homicide and assault rates was also found in Sweden in the period 1870–1984 (Lenke 1990), and alcohol consumption was reported to be one of the four indicators that explained an increase in assault rates in Finland in 1950–2000 (Sirén 2002). Furthermore, an increase in alcohol consumption of one litre per capita in Norway predicted an increase of eight per cent in the violence rate between 1911 and 2003 (Bye 2007).

Individual-level studies contribute to the literature to some extent. According to two empirical reviews of retrospective and prospective cohort studies, alcohol (and drug) use disorders are strongly associated with suicide (Harris and Barraclough 1997; Wilcox et al. 2004). Lyn Exum (2006) concluded from her review of experi-

mental studies that alcohol consumption increases aggressive behaviour. This effect is not uniform, however, but is instead moderated by factors (e.g., situational factors such as emotional state, level of inebriation, and perceived retaliation from the victim) commonly found in real-world accounts of intoxication-related violence (Exum 2006). According to another review approximation, in countries in which alcohol is commonly used over 50 per cent of assailants have been drinking prior to their offence (Roizen 1997). It has also been suggested that binge drinking rather than alcohol consumption as such may contribute to an increased risk of interpersonal violence (Richardson and Budd 2003; Graham et al. 2006).

A meta-analysis of 22 studies revealed that evidence on the relationship between alcohol consumption and violence between intimate partners is weak: many studies are based on feeble design and may be biased by the publication of positive results. Consequently, the authors concluded that there was not enough empirical evidence to support the introduction of preventive policies based on male alcohol consumption as a risk factor in the particular case of partner violence (Gil-González et al. 2006). According to another study research findings support the assumption that drinking is involved in or associated with much social harm (including unintentional injury, aggression and violence), but do not offer evidence that it causes these effects. Methodological flaws characterise much of the research in this area (Gmel and Rehm 2003).

According to a meta-analysis of 28 studies conducted in 16 countries between 1984 and 2002 drinking within six hours prior to the injury was reported by 21 per cent of the injured patients sampled. The estimated (random) pooled relative risk for patients who had consumed alcohol within six hours prior to injury was 5.69 (95% CI: 4.04–8.00), ranging from 1.05 in Canada to 35.00 in South Africa. The effect size was not homogeneous across the studies: there was a higher relative risk for injury in societies with riskier consumption patterns. There was also a lower relative risk among heavier drinkers (Borges et al. 2006). Another meta-analysis supported these results, and further found no strong association between indicators of alcohol-related disorders and injury (Cherpitel 2007).

3.2.3 All-cause mortality

A couple of time-series analyses on the population level focused on the association between alcohol consumption and all-cause mortality. It was concluded in one such study based on historical data from Prussia, France and Sweden that a one-litre increase in per-capita alcohol consumption would increase mortality among middle-aged men by about one per cent (Norström 1996). An ECAS time-series

study investigating the association between alcohol sales and all-cause mortality in 14 European countries reported significantly positive effect estimates in three of them (France, the Netherlands and West Germany; a one-litre increase was expected to be followed by an increase of about one per cent in mortality) or eight of them (an increase of from one to four per cent) depending on the model employed, and in 17 of 56 age- and country-specific cases (Norström 2001). Another time-series study based on Canadian data for the years 1950–1998 revealed a significant alcohol effect that implied a 2.9-per-cent increase in mortality given a one-litre increase in consumption. When cigarette sales were included in the model the alcohol effect was still statistically significant but markedly reduced, to 1.7 per cent (Norström 2004).

A number of individual-level studies have contributed to the literature by specifying the association between alcohol consumption and all-cause mortality. With regard to weighing the risks of moderate alcohol consumption against its benefits at tolerable upper intake levels different estimates have been set. A meta-analysis set the estimates at 10–12 g/day for healthy women and 20–24 g/day for healthy men in the adult population (Burger, Brönstrup et al. 2004). Another meta-analysis concluded that for the detrimental conditions mentioned above, significant increased risks were associated with ethanol intake of 25 g per day (Corrao et al. 2004). It was found in yet another meta-analysis of 34 studies that alcohol consumption, up to four drinks a day among men and two drinks among women, was inversely associated with total mortality, the maximum protection (i.e. risk reduction) being 18 per cent among the women (99% CI: 13%–22%) and 17 per cent among the men (99% CI: 15%–19%). Higher doses of alcohol were associated with increased mortality (Di Castelnuovo et al. 2006).

A study conducted in the UK based on mortality and survey data from England and Wales produced evidence of a direct dose-response relation between alcohol consumption and the risk of death among women aged 16–54 and among men aged 16–34 (White et al. 2002). At older ages the relation is U shaped. The level at which the risk is lowest increased with age, reaching three units (27 g) a week among women aged over 65 and eight units (72 g) a week among men. The level at which the risk increases by five per cent above this minimum is eight units (72 g) a week among women aged 16–24 and five units (45 g) a week among men, increasing to 20 and 34 units (180 g and 306 g) a week in women and men aged over 65, respectively. This reflects the steeper slope of the risk curve for young men compared with young women (White et al. 2002).

3.2.4 Gender and age differences in alcohol consumption and related harm

Gender differentials are universal in terms of alcohol consumption and its consequences; only the magnitude of the male excess varies (e.g. Nolen-Hoeksema 2004). However, in many countries the gender gap has narrowed in terms of both volume and patterns of drinking, which has also decreased the gender differences in alcohol-related harm. A number of types of explanations for this difference have been proposed. Psychosocial and cultural (or psychological and social-structural) explanations of gender differences are applicable to drinking rather than to its harmful consequences, whereas biological factors seek to explain why women are more sensitive or reactive to alcohol use in terms of alcohol-related harm (e.g. Wilsnack et al. 2000; Nolen-Hoeksema 2004; Holmila and Raitasalo 2005).

Studies on psychological explanations focus on needs, reasons and motivations in relation to drinking (Holmila and Raitasalo 2005), whereas cultural or social-structural explanations are used particularly in order to assess gender differences in drinking control (Wilsnack et al. 2000; Holmila and Raitasalo 2005). According to some studies the greater social sanctions against drinking for women than for men is the main reason why women do not drink more than men (e.g., Gomberg 1988; Nolen-Hoeksema 2004).

On the biological level alcohol has different effects on the female and the male body (e.g. Nolen-Hoeksema 2004; Holmila and Raitasalo 2005). Due to the larger average content of lipids and the smaller average content of water in women's bodies, the same amount of alcohol for the same body weight, consumed during the same length of time leads to higher blood-alcohol levels among women than among men (e.g. Mumenthaler 1999; Ramchandani et al. 2001; Holmila and Raitasalo 2005). Furthermore, gender differences in alcohol metabolism (Lieber 2000), in pharmacokinetics of alcoholism (Baraona et al. 2001), and in its effect on brain volumes (Hommer et al. 2001) have also been put forward as biological reasons for women's greater vulnerability to the effects of alcohol.

Age is of major significance in alcohol consumption in terms of regularity of use and drinking patterns. In many countries, patterns of sporadic heavy drinking in young adulthood tend to give way in middle age to more regular consumption and fewer episodes of heavy drinking, and in turn to much lighter drinking at older ages (Edwards et al. 1994, 45). The effect of drinking on alcohol-attributable consequences in both detrimental and beneficial terms also varies according to age, as mentioned previously. As with gender differences, biological, psychosocial and cultural explanations also apply to age.

On the whole, there is a relatively abundant body of research on the association between alcohol consumption and both health and violence. With regard to directly alcohol-attributable diseases, it is well-established that the risk increases in line with an increase in consumption. The safe amount of daily consumption appears to vary to some extent depending on the disease. There is strong evidence of cardioprotective effects of alcohol on the individual level, whereas research is scarce on the population level. Furthermore, evidence concerning the effects of alcohol consumption on accidents and violence, and also on all-cause mortality, is scanty and weak on the population level. It is obvious that the drinking pattern is an important element of consumption in terms of determining whether the outcome is harmful or beneficial in nature.

3.3 The association between alcohol prices and the consequences of drinking

Research on the association between alcohol pricing and health and other measures of harm is based mainly on aggregate-level data, but is scanty overall. Moreover, there are hardly any studies on socio-demographic differentials regarding this relation. Much of the evidence comes from the US and is based on cross-sectional aggregate-level data, or time-series of such cross-sectional analyses. The few natural experiments have addressed various causes of death or self-reported problems, and some of them have serious limitations in terms of data and methodology. Other studies related to price and health are based purely on cross-sectional data from the US and mainly address traffic fatalities, although a few other outcome measures have been investigated. Discussion in terms of the effects of various policy measures is accentuated in the studies on traffic fatalities. Research on violent crimes is also scanty and inconsistent.

3.3.1 Natural experimental studies on changes in the full price of alcohol and harm

A frequently cited example of research offering historical evidence of the association between changes in the full price of alcohol and the consequences comes from Denmark. Although Denmark remained neutral during World War I, the blockade caused a substantial shortage of many commodities. It was mainly for this reason that the tax on alcoholic beverages increased dramatically, and consumption decreased, according to estimates of alcohol sales, from about 10 litres per capita during the period 1911–1915 to 2.2 litres in 1918. Skog (1993) concluded in a time-series study that per-capita alcohol consumption in 1911–1924 was probably related to the suicide rate in Denmark: the number of suicides decreased by 19

per cent in 1916–1920 compared to 1911–1915. However, the period in question (1911–24) on annual basis is very short, given that at least 50–100 observations are required for accurate time-series analysis (Yaffee 2000). Moreover, war is a very exceptional state of affairs as such, not to speak of WW1, with numerous factors that might contribute to suicide rates. Again according to the Danish data from the years 1911 to 1931, the immediate reduction in sales of distilled spirits from 1916 to 1918 following the rise in prices was counteracted by an adjustment in the opposite direction during the following years. Data on mortality from delirium tremens, alcohol psychosis and liver cirrhosis confirmed this pattern (Norström and Ramstedt 2005; Skog and Melberg 2006).

A classic example of rapid change in mortality due to a policy act regarding a change in the full price of alcohol was illustrated in data from Paris during the Second World War. Rationing (0.5–1 litre of wine per week) was introduced in 1942 because of an extreme shortage of alcoholic beverages, and consequently, according to estimates, there was a dramatic reduction of 80 per cent or even more in consumption during the war. Liver-cirrhosis mortality decreased by 50 per cent in one year, and after five years it was more than 80 per cent below the 1941 level (Edwards et al. 1994, 82).

A more recent episode regarding changes in alcohol policy was documented in Russia. According to data from Moscow state alcohol sales decreased by 38 per cent in 18 months following the 1985 anti-alcohol campaign, and deaths from liver cirrhosis and alcohol poisoning, and blood-positive violent deaths were estimated to have decreased by 33, 51, and 51 per cent, respectively. It has been estimated that total alcohol consumption began to increase again in 1987 and continued in all subsequent years, although it was especially high in 1992–93 at the time of the introduction of market reforms in Russia. An increase in blood-alcohol-positive violent deaths was estimated to have begun in 1987, before the increases in other deaths (Nemtsov 1998). The reliability of the Russian data on mortality and alcohol consumption has been questioned, however (Leon et al. 1997; Rehm 2009; Zaridze et al. 2009).

The effects of the 1999 tax reform of foreign spirits in Switzerland was examined in a before/after design: a randomly selected sample of 4,007 residents aged 15 years or older participated in a baseline survey three months before the tax reform, and 73 per cent of those in the follow-up survey 28 months after it. Self-reported alcohol-related problems increased significantly at follow-up, particularly among the younger age groups who showed a preference for spirits over other alcoholic beverages (Mohler-Kuo et al. 2004).

Three recent time-series analyses have addressed the effects of policy changes on different outcomes. One of them focused on the same change of alcohol policy in Finland in 2004 as the present study, but lacked age and gender stratification, covered a time period of ten months after the price change, and focused on a limited set of alcohol-related causes, namely alcohol-positive sudden deaths. According to the findings, the price reduction resulted in eight additional alcohol-positive sudden deaths per week within ten months, which represents a 17-per-cent increase compared to the weekly average of 2003 (Koski et al. 2007). Another study evaluated the effects of tax increases on alcoholic beverages in 1983 and 2002 on alcohol-related disease mortality from 1976 to 2004 in Alaska (Wagenaar, Maldonado-Molina et al. 2009): reductions in mortality of 29 and 11 per cent, respectively, were found. Another paper investigated changes in violent assaults and hospitalisation due to acute alcohol intoxication in Denmark between 2003 and 2005 after changes in alcohol policies (the excise tax on spirits in Denmark was lowered by 45 per cent and duty free allowances of alcohol for travellers were increased) introduced in 2003 and 2004 (Bloomfield et al. 2009). No significant increase in hospitalisation was found among persons older than 15 years or in violent assaults in general, whereas there was an increase of 26 per cent in the hospitalisation rate due to intoxication among people aged 15 or younger (Bloomfield et al. 2009).

3.3.2 Other studies on alcohol prices and harm

On top of natural experimental studies, a number of other studies have examined the association between alcohol prices or taxes and alcohol-related harm. Most of them are based on cross-sectional data or time-series of such cross-sectional analyses.

Chronic diseases

Two cross-sectional time-series studies from the US reported contradictory findings on the effect of alcohol prices on alcohol-related mortality. According to one of them an increase in the liquor excise tax of one dollar (1967 prices) per proof gallon would reduce the liver-cirrhosis mortality rate by 5.4 per cent in the short run, and perhaps by twice that amount in the long run. This estimate was based on an analysis of annual state-level data, covering 30 states and the years 1962–1977, with state excise taxes and per-capita income as the covariates (Cook and Tauchen 1982). The other study, covering the years 1982–1988 and 48 states, found that higher prices did not decrease the mortality rates for alcohol-attributable primary causes of death (Sloan et al. 1994). This difference was mainly attributable to the inclusion of a larger number of independent variables in the latter study (Sloan et al. 1994).

Traffic fatalities

The US population-level evidence on the effects of alcohol pricing on alcohol-related mortality is based mainly on cross-state studies on the association between beer taxes and traffic fatalities, and in many cases beer taxes are set against other alcohol-regulation policies such as the minimum legal drinking age, for example. However, the findings are conflicting. According to one study based on data for 48 states over the 1982 through 1988 time period on the impact of beer taxes and a variety of alcohol-control policies on motor-vehicle fatality rates, most of the regulations had little or no impact. By way of contrast, higher beer taxes were associated with reductions in crash deaths, and this result was relatively robust across specifications (Ruhm 1996). A more recent study reported that raising either beer taxes or the minimum legal drinking age in isolation led to fewer traffic fatalities among young people (Ponicki et al. (2007).

Some recent studies have introduced numerous specifications, and have reported contradictory findings, regarding this relationship. Young and Likens (2000) analysed data from 48 states over a nine-year study period, investigating the relationship between motor-vehicle fatalities and alcohol taxes, prices, and various drinking laws. They found that none of the beer-tax or price coefficients were statistically significant. The magnitudes of the estimated effects were much smaller than those reported in some previous studies. Seatbelt laws, the minimum legal drinking age, and dram-shop laws typically had statistically significant, negative relationships with fatalities. The results of another study focusing on teenagers demonstrated the significance of cross-state heterogeneity and the relatively small and statistically insignificant effects of beer taxes on teen drinking and youth traffic fatalities (Dee 1999). With regard to the relationship between beer taxes and traffic fatalities over time, it has been found that this relation is not robust across data periods, and that it reflects missing variable biases (Mast et al. 1999). The authors found it surprising that most studies on alcohol-related traffic fatalities attribute importance to beer taxes as a policy variable because they have only a minor impact on prices in the U.S., and thus on consumption. Furthermore, heavy drinkers appeared to be the least responsive to prices in this respect (Mast et al. 1999).

Other accidents and violence

Another cross-sectional study from the US covering the period 1976–1999 reported that increases in the excise tax on beer were associated with a reduced number of male suicides among youths and young adults aged 10–24 years, but had no impact on female suicides (Markowitz et al. 2003).

According to the results of one of the few studies directly assessing the relation between the price of alcohol and violent crime, which was based on cross-sectional state-level comparisons in the US, a 10-per-cent increase in beer tax would reduce the incidence of rape by 1.3 per cent and robberies by 0.9 per cent, but would have little impact on homicides and assaults (Cook and Moore 1993). Studies based on national surveys and cross-sectional data in the US also suggest that an increase in the price of alcohol, as measured by a weighted average of the price of beer, wine and spirits, would reduce the probability of violence aimed at wives, assault and alcohol- or drug-involved assault, but not rape or robbery (Markowitz 2000; 2005). A study from the UK found that high regional violence-related injury rates correlated with a low real price of alcohol as measured by the price of beer: rates of violence were higher during the summer months and on days of major sporting events (Sivarajasingam et al. 2006).

All in all, studies addressing the association between alcohol prices and harm are few and far between. A few natural-experiment studies, based mainly on historical data, suggest that higher prices are associated with fewer alcohol-related problems and vice versa. However, much of this evidence suffers from methodological limitation. Moreover, the evidence on the effects of pricing on chronic diseases, traffic fatalities and other accidents and violence, based mainly on cross-state studies from the US, remains weak and inconsistent.

3.4 Inequalities in alcohol consumption and the consequences

3.4.1 Socioeconomic differentials

Alcohol consumption tends to be more adverse, in terms of the prevalence of heavy drinking and drinking patterns, in lower socioeconomic groups, but cultural and gender differences exist (Kunz and Graham 1998; Kuntsche et al. 2004; Kuntsche et al. 2006). For example, according to a study based on survey data conducted in eight European countries in the 1990s and early 2000s, women in countries with a strong social-welfare system tend to drink more heavily if they employed, have a lower level of formal education, and a non-traditional family role, whereas in countries with weak social-welfare systems heavy drinking is associated with a high level of education, and the effects of family roles and employment status are small (Kuntsche et al. 2006). With regard to responsiveness to alcohol prices it could be hypothesised that groups with fewer means, such as those of lower socioeconomic status, would be more affected.

Large socioeconomic differences in alcohol-related mortality, and also in life expectancy and all-cause mortality, are well documented (Mackenbach et al. 1997, 1999; Mäkelä 1999; Martikainen et al. 2001; Steenland et al. 2004; Huisman et al. 2005; Mäki and Martikainen 2009). Occupational social class has been found to be a risk factor for alcohol-related mortality in the UK, particularly among men (Harrison and Gardiner 1999; Metcalfe et al. 2005), whereas Nordic studies have reported alcohol-related mortality rates that are 1.9–3.2 times higher among male manual workers than among non-manual employees in middle-age (Mäkelä 1999; Norström and Romelsjö 1998; Hemström 2002). Furthermore, some recent studies report increased socioeconomic inequalities in alcohol-related mortality over time according to social class and education (Valkonen et al. 2000; Najman et al. 2007). US studies on socioeconomic differences in alcohol-related mortality are sparse, but it has been reported that socioeconomic differences in alcohol-related motor-vehicle crashes are marked by education, income and language group (Braver 2003; Romano et al. 2006). There are also a few studies have also reporting a clear negative socioeconomic gradient in the rate of alcohol-related hospitalisation, implying that rates in lower-status groups are 2.7–3.6 times higher than in higher-status groups (Poikolainen 1982, 1983; Romelsjö and Diderichsen 1989; Mäkelä et al. 2003; Metcalfe et al. 2005).

With regard to the explanations for socioeconomic differentials in health and mortality in general, one of the most important milestones was the publication of “Inequalities in Health. The Black Report” in 1980. A shortened version of the report was published in 1982, making it widely available (see Townsend and Davidson 1982). Although there are more recent overviews, some of which are critical (Vågerö and Illsley 1995), the explanatory framework presented in the Black Report could also apply to socioeconomic differences in alcohol use and its consequences. The report suggests four types of explanation: (1) artefactual, (2) theories of natural or social selection, and (3) materialist or structuralist, and (4) cultural/behavioural explanations, the two last-mentioned being causal in nature (Townsend and Davidson 1982, 104–15). Artefactual explanations attribute the association between education and alcohol-related mortality, for example, to measurement bias and errors. The relevance of such explanations has diminished along with advancements in data collection and methodology.

According to explanations based on natural selection (or direct selection), a person is a member of a low social class because of personal characteristics such as alcohol abuse. Consequently, this would be evident in higher rates of alcohol-related mortality among persons of low social class than among those in the higher classes. Thus alcohol abuse influences social class rather than vice versa.

This “hard” version of selection “explains away” observed inequalities in health in accordance with social class as nothing meriting social concern or collective intervention (Macintyre 1997). Explanations based on social (or indirect) selection, a “soft” version of selection, assume that processes of social selection (such as recruitment into a social class) may contribute to the production of health gradients by social class (Macintyre 1997). There would thus be one or a number of other social factors that influence both health and socioeconomic position. For example, childhood in a family suffering from severe parental alcohol problems may contribute to a person’s later susceptibility to alcohol abuse and, at the same time, exclude him or her from higher education and further higher social positions, and be harmful to health.

Causal effects of social class on health are considered to be mainly indirect, in other words they are mediated through numerous other health determinants. The materialist/structuralist explanation emphasises the role of economic and associated socio-structural factors in the distribution of health and well-being (Townsend and Davidson 1982, 106). In other words, physical and material conditions of life, which are determined by social class, produce class gradients in health, and deprivation in terms of income and wealth produces deprivation in health (Macintyre 1997). According to the “softer” version, the conditions of life that are determined by social-class position, and which may influence health, include psychosocial as well as physical factors, and social as well as economic capital (Macintyre 1997). This “softer” version has attracted support among a number of researchers (e.g., Morris 1990; Smith et al. 1990; Davey Smith et al. 1994), although its applicability to empirical studies has been questioned (Vågerö and Illsley 1995).

According to the cultural/behavioural explanation, there are observable social-class gradients in health (e.g., alcohol-related mortality or morbidity) but these are completely attributable to health-damaging behaviours (heavy drinking), or in “softer terms”: certain health-damaging behaviours have a social-class gradient and this contributes to the social-class gradient in ill health (alcohol-related mortality or morbidity). In other words, behaviours do not explain away class differences, but contribute to them, and push the explanatory task further back to questioning why such behaviours are persistently more common in poorer groups (Macintyre 1997). Finally, it must be noted that the two main types of explanation, selection and causal, are not incompatible, even within a single study context (Kasl and Jones 2000, 120).

The Black Report’s explanatory framework, like other general frameworks, is often modified for specific study purposes. For example, in a more specific context

Martikainen, Brunner and colleagues (2003) suggested that at least four factors could be put forward to explain socioeconomic differences in dietary patterns, which may also be applicable, at least to some extent, in the context of alcohol consumption and its consequences: 1) Socioeconomic aggregation of unhealthy behaviour; 2) Material hardship; 3) Socioeconomic differences in perceptions of control over health; and 4) Socioeconomic variations in contextual influences.

The first of the above explanations is applicable in the context of socioeconomic differences in alcohol use and harm. Heavy drinking, smoking, an unhealthy diet and insufficient physical exercise are more prevalent among the lower social classes (e.g. Crespo et al. 1999; Salmon et al. 2000; Dowler 2001; Droomers et al. 2001; Hanson and Chen 2007; Padrão et al. 2007; Bécue-Bertaut et al. 2008), which may partially explain socioeconomic differences in alcohol-related mortality and morbidity, and in health in general. With regard to material hardship, a lack of money, for instance, may affect socioeconomic differences in alcohol consumption and harm in at least three ways. First, people of low socioeconomic status may purchase cheaper beverages, which may be of lower quality. For example, harmful consequences of drinking surrogate alcohol have been reported in Russia (McKee et al. 2005). Secondly, if someone cannot afford to go to a restaurant, he or she drinks at home where the informal control may be less effective. Thirdly, drinking, and heavy drinking in particular, may be a way of coping with the material adversities and hardships of daily life.

On the more psychosocial level, the perception of control over health is also a feasible potential explanation of socioeconomic differences in alcohol-related harm. Control over different domains of life, and health in particular, may be an incentive to consume alcohol moderately, and this may differ by socioeconomic status. It was found in Sweden, for example, that young men with heavy alcohol consumption had an increased risk of developing alcoholism if they then worked in an environment characterised by low control (Hemmingsson and Lundberg 2001). This finding was not attributable to the selection of heavy drinkers into low-control jobs. In terms of contextual influences, Martikainen, Brunner and colleagues (2003) referred to ethnicity and household effects. In the context of socioeconomic differences and alcohol-related harm, however, it would be more accurate to refer to the norms and attitudes of a certain social class or level of education. Permissive attitudes to binge drinking and beverage preferences, for instance, have been reported to be associated with social class (Kuntsche et al. 2004; Mortensen et al. 2005).

3.4.2 Neighbourhood characteristics

In recent years an increasing number of studies have focused on the neighbourhood or area effects – a specific type of a contextual effect – on health and crime. Explanations for the socioeconomic inequality observed in community settings usually refer to one of two neighbourhood features: the characteristics of the people living in it or the integral characteristics of the places in which they live (Gruenewald, Freisthler et al. 2006). The former include factors such as area-level poverty, family structure and residential mobility (Sampson et al. 1997, 2002), and the latter factors such as the proportion of abandoned housing and the level of retail activity (Felson 1987). The use of population- rather than place-related characteristics may work better in the European welfare setting in which cities are better equipped to control their development by means of town planning than in the US context (Ostendorf et al. 2001).

The underlying mechanisms linking neighbourhood characteristics and various outcomes remain partly uncharted, however. Neighbourhood differences are not “naturally” determined, but rather result from social and economic processes influenced partly by specific policies (Diez Roux 2002). It has been suggested that spatial embeddedness, internal structural characteristics and social organisational processes are all important in understanding neighbourhood-level variations in health and crime (Rossow 2001). A review examining social processes related to health-related outcomes and problem behaviour covering over 40 studies emphasised neighbourhood ties, social control, mutual trust, institutional resources, disorder and routine activity patterns in tandem with traditional characteristics such as poverty and residential instability (Sampson et al. 2002).

4 STUDY DESIGN, SCOPE AND OBJECTIVES

4.1 The natural experiment as a research design

Natural or quasi-experimental research concerns naturally occurring instances of observable phenomena in which the study subjects experience of an 'exposure' of interest changes, and that approximate or duplicate the properties of a controlled experiment. In contrast to laboratory experiments, these events are not created by scientists, but yield data that can nonetheless be used to make strong causal inferences. A natural experimental design is commonly employed in studies in which it is intended to identify the effect of an intervention (Petticrew et al. 2005). In other words, it is a study that explores the consequences of a change in policy or situation that came about without consideration of research or an evaluation. Such natural experiments are rare in the field of alcohol studies, and most of them examine the effects of temporary disruptions in the supply of alcoholic beverages, such as caused by strike action (Österberg and Säilä 1991, 12).

According to Wagenaar, Maldonado-Molina and colleagues (2009) an appropriate natural experimental design has a number of benefits: (1) it allows researchers to eliminate many confounding factors that threaten the causal interpretation of an observed relationship without having to identify, measure and statistically control for all possible confounding variables; (2) it could stop debate about which of the numerous specific control variables to include; and (3) it may prevent researchers from introducing biases into the study by including only the control variables for which operational measures are available.

4.2 Scope and objectives

The overall aim of this study, which could be regarded as a natural experiment, was to investigate the effects of the reduction in alcohol prices that was realised at the beginning of the year 2004 in Finland on mortality attributable to different causes, morbidity related to alcohol, and violent crime. An additional aim was to assess how these effects varied according to different socio-demographic and area factors. Furthermore, in order to put these findings into a broader perspective, the study also focused on the trends in alcohol-mortality prior to the price reduction.

The main goals listed according to the various sub-studies were as follows:

1. To examine how alcohol-related mortality and the population sub-group differences changed in Finland in 1987–2003 – prior to the policy-driven price change – when there were substantial changes in economic conditions and alcohol consumption (Study I).
2. To investigate the extent to which the reduction in alcohol prices in 2004 and the subsequent increase in consumption affected alcohol-related mortality overall and in different age and socioeconomic groups. Specifically, the aim was to find out whether the effect varied with (i) sex, age and cause of death, (ii) education and occupational social class, and (iii) household income and economic activity (Study III).
3. To evaluate, by means of time-series analysis, the impact of the reduction in alcohol prices on alcohol-related mortality, mortality attributable to cardiovascular diseases and all-cause mortality, stratified by sex and age over the period 1996–2006 (Study IV).
4. To document and assess, by means of time-series analysis, the change in hospitalisation related to alcohol, stratified by sex and age, after the reduction in alcohol prices over the period 1996–2006 (Study V).
5. To assess to what extent the changes in the full price of alcohol in 2004 affected the change in rates of interpersonal violence and in two indicators of less severe disorderly conduct, and how these changes varied on the small area level in the Helsinki Metropolitan area (Study II).

5 DATA AND METHODS

5.1 Data

5.1.1 Studies on mortality (Studies I, III and IV)

Study populations

The analyses in all of the sub-studies on mortality are based on the linkage of census or population-registration data on persons certified as dead according to a unique personal identification number. All the analyses comprised all Finns aged 15 and above.

In Study I the data from the 1985, 1990, 1995 and 2000 censuses were linked individually to records from the death register for the years 1987–90, 1991–95, 1996–2000 and 2001–03, respectively. The analysis comprised a total of 33.7 million person-years among men and 36.4 million person-years among women.

Two study periods were defined in Study III: 2001–2003 before and 2004–2005 after the price reduction. Longitudinal register data from the employment statistics for 2000 and 2003 were linked individually by means of personal identification codes to the records from the death register. The men in the study population represented about 10.4 million and the women 11.1 million person-years. The data for Studies I and III were in the form of a cross-tabulation of person-years and numbers of deaths according to the variables of interest.

The focus of the analyses in Study IV was on monthly time-series data on alcohol-related, cardiovascular disease and all-cause mortality rates for the years 1996–2006.

Alcohol-related mortality

Deaths were classified according to the Finnish edition (FCD) of the International Classification of Diseases and Related Health Problems, Ninth Revision (ICD-9) (Study I) and Tenth Revision (ICD-10) (Studies I, III and IV). Alcohol-related deaths were defined as those in which alcohol was referred to as the underlying or one of the contributory causes in the death certificate.

The total pool of alcohol-related deaths used comprised the following two main categories: 1) the underlying cause of death was an alcohol-attributable disease (see below) or alcohol poisoning, and 2) the underlying cause was not alcohol-related, but a contributory cause was an alcohol-attributable disease or alcohol intoxication (ICD10 code F100). Alcohol-attributable diseases included alcohol

dependence syndrome (ICD10 code F102), other mental and behavioral disorders due to alcohol (F101, F103-109), alcoholic cardiomyopathy (I426), alcoholic liver disease (K70), alcoholic diseases of the pancreas (FCD K860), and a few rarely occurring categories (K292, G312, G4051, G621, G721).

Two mutually exclusive cause-of-death categories were formed for the analysis in Study III: acute and chronic alcohol-related causes. Deaths in the chronic category were those of which the underlying cause was alcoholic or an alcohol-attributable disease was a contributory cause, whereas in the acute category the underlying cause was not alcoholic, but alcohol intoxication was a contributory factor. Deaths of which one contributory cause was intoxication and another was an alcohol-attributable disease were included in the acute-causes category.

Independent variables

The independent variables in Study I included sex, five-year age groups (15–19, ..., 90–94, 95+), and education split into the following categories: basic (10 years or less), intermediate (11–12 years), high (13+ years).

In Study III the independent variables derived from employment statistics were gender, five-year age-groups (15–19, ..., 75–79, 80+) and four indicators of socio-economic position. The four educational categories were based on the highest achieved educational level obtained from the national Register of Completed Education and Degrees: basic education, secondary education, lower tertiary and higher tertiary. Occupational social class was divided into six categories: upper white-collar employees, lower white-collar employees, skilled workers, unskilled workers, the self-employed and other. Unemployed and retired persons were classified according to their previous occupations, and people taking care of households were categorised according to the occupation of the head of the household (Valkonen et al. 1993, 14–16). Income was measured as household disposable income per consumption unit and divided into quintiles, with quintile boundaries defined for men and women combined in the year 2000. Income comprised all taxable income received by family members after taxes had been subtracted, including wages, capital income and taxable income transfers. Different weights were used for adults and children in the calculation of household-consumption units: for the first adult, 1.0; for other adults, 0.7; and for children, 0.5. This corresponds to the OECD equivalence scale (OECD 1982). The information on different sources of income came from the registers of the Finnish Tax Administration and the Social Insurance Institution. Economic activity included five categories: employed, unemployed for a period of 25 months or more during the previous three years, unemployed for less than 25 months, pensioner, and other.

The monthly data were stratified by gender and five-year age-groups (15–19,...,75–79, 80+) in Study IV.

5.1.2 Hospitalisation (Study V)

Monthly time-series data on hospital utilisation attributable to alcohol-related diagnoses were obtained from the Finnish Hospital Discharge Register, which gathers comprehensive information on individual patients in all Finnish public and private hospitals. The monthly data for the years 1996–2006 were stratified by gender and five-year age-groups (15–19,...,75–79, 80+). Diagnoses were classified according to the Finnish edition (FCD) of the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10). Causes of hospitalisation related to alcohol were defined as those with a reference to alcohol in the primary diagnosis. There were almost 283,000 such incidences, of which 27 per cent referred to alcohol dependence syndrome (ICD-10 code F102), 20 per cent to other mental and behavioural disorders due to alcohol (ICD-10 codes F101, F103-109), 1.7 per cent to alcohol poisoning (ICD-10 code X45), 10 per cent to alcoholic liver diseases (ICD-10 code K70), 0.2 per cent to alcoholic cardiomyopathy (ICD-10 code I426), nine per cent to alcoholic diseases of the pancreas (ICD-10 code, Finnish Edition K860), 0.9 per cent to alcoholic gastritis (ICD-10 code K292), 28 per cent to alcohol intoxication (ICD-10 code F100), 0.2 per cent to maternal care for (suspected) damage to foetus from alcohol (ICD-10 code O354), and 3.7 per cent to other alcoholic diseases (ICD-10 codes G312, G4051, G621, G721). Men accounted for 81 per cent of all hospitalisations related to alcohol.

5.1.3 Area level interpersonal violence study (Study II)

Study sites and period

The sample for this aggregate-level study comprised 86 small areas (tracts) from the Helsinki Metropolitan area, of which 33 belonged to Helsinki, 27 to Espoo and 26 to Vantaa and a small municipality within Espoo (Kauniainen). The populations ranged from 486 to 36,522, with a mean of 10,981, and the tracts were based on the administrative-area division of the municipalities, which is used for policy purposes (Helsinki Region Statistics Database 2007; Kauniainen Statistics Database 2007).

The study period extended from the beginning of 2002 until the end of 2005. The total period was divided into two sub-periods: (1) before the change in the pricing of alcohol (2002–2003) and (2) after the change (2004–2005). The investigation of two symmetric periods in the analysis and allowed to potential bias due to seasonal variations in interpersonal violence.

Area-level crime and socio-demographic characteristics

Data on crime and socio-demographic characteristics were obtained from administrative databases. The main outcome measure was interpersonal violence, but two less severe indicators of disorderly conduct were also included in the analyses. The data on interpersonal violence and disorderly conduct were obtained from three sources at the Helsinki Police Department. The first covered crimes recorded by the police that were specified as offences against the Penal Code, and the following outcome measures were included in the analysis: assault and battery including the subgroups assault in private homes and in public places, robbery and extortion, disturbance of the domestic peace and rape. The second source of data comprised emergency call-outs related to domestic violence, disturbing behaviour and vandalism, and emergency responses in total. Approximately 80 per cent of these cases were reported to the police emergency centre by the public. Finally, a particular outcome measure was included: being taken into custody due to alcohol intoxication was recorded as a police task but not as a crime. The data consisted of the number of various acts by tract and month in the years 2002–2005.

Six of the seven socio-demographic characteristics were measures of social disadvantage: for persons aged 15 years or more they included the proportion of people with a basic education, a mean personal income (€ 1,000), manual-class membership and on the unemployment register (the two latter being characteristics of the labour force), the proportion of single-parent families, and the proportion of homes that were not owner-occupied. A measure of residential instability (outmigration) was also used. All but one of the area-level socio-demographic characteristics were measured in 2002–2003, and manual-class membership was measured in 2000.

5.2 Analyses

Study I focused on trends in mortality by cause and age, assessed in terms of annual crude death rates per 100,000 person-years. Cause-specific mortality was calculated without age standardisation because it did not affect the results due to the short observation period. The effect of differences in the age distribution was controlled for in the comparison of the educational groups by calculating directly age-adjusted mortality rates, with the combined population of men and women in 1987–2003 as the standard population.

In Study II, the frequencies and rates per risk population per year and per 1,000 persons were calculated for all the different categories of interpersonal violence and disorderly conduct. Multiple linear regression analyses were conducted on

these rates in order to evaluate the association between the area characteristics and assault, assault in private homes, domestic violence, and custody due to alcohol intoxication. In order to take account of the correlatedness of the data over time the Stata 'cluster' option was used, which affects the estimated standard errors and the variance-covariance matrix of the estimators, but not the estimated coefficients. Weighting was used in the linear regression analysis in order to take account of the population size in the tracts.

Two different models were estimated. First analyses were conducted separately for each one of the area characteristics, which were chosen on the basis of earlier studies and were thus established as relevant in theoretical terms (Stafford et al. 2004; Martikainen et al. 2007). Secondly, a backward selection regression procedure, with an incremental removal significance criterion of $p > 0.05$, was used to determine the best model with suitable independent variables. The change over time was tested by means of linear regression focusing on the interaction between time and the area characteristics. Time was used here as a categorical variable and area characteristics as a continuous variable.

For Study III, deaths and person-years were cross-tabulated according to year, socio-economic indicators and other variables of interest. The statistical analyses were based on the Poisson regression model, with the cell in the cross-tabulation taken as the unit of analysis. Mortality ratios obtained by means of Poisson regression were used to assess the relative differences between the age groups and the different socioeconomic characteristics. An experiment with negative binomial regression was conducted to account for any over-dispersion in the data. However, the point estimates, and test values and confidence intervals were very similar to those obtained from Poisson regression, which was therefore preferred. Calendar period–social class interaction terms were included in the models in order to determine the relative effect of the price reduction on alcohol-related mortality by any one socioeconomic variable, and likelihood ratio tests were used to derive the p-values.

Box-Jenkins autoregressive integrated moving average (ARIMA) intervention time-series analyses were used in Studies IV and V to model the monthly alcohol-related, cardiovascular disease and all-cause death and hospitalisation rates. This method involves a two-phase process. The aim in the first phase is to identify a descriptive model that best captures the seasonality, time trends and autocorrelation inherent in the series (Box et al. 1994). The intervention component – here time of the price change – is added in the second phase in order to obtain the impact-assessment model that allows better causal attribution of changes in time-series to given events. 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 (McCleary and Hay 1980).

Maximum likelihood was used to estimate the model parameters. For the model selection, Akaike's and Schwarz's Bayesian Information Criterion was computed for each one, which are statistics used to evaluate the goodness of fit and parsimony of a candidate model. Time series may be influenced by outliers that are not in accordance with most observations in them. The presence of outliers in a time series could affect the specification of the model and distort the estimates of the ARIMA model parameters. The ways of dealing with outliers are diverse and context-specific (Chatfield 2004). Dummy variables were added as a regression parameter in the analyses of mortality and hospitalisation, if outliers hindered the model specification. Their presence or absence did not largely affect the results. Variance appeared to change over time in all of the series, particularly after the price reduction, and natural log transformation was therefore applied to the series.

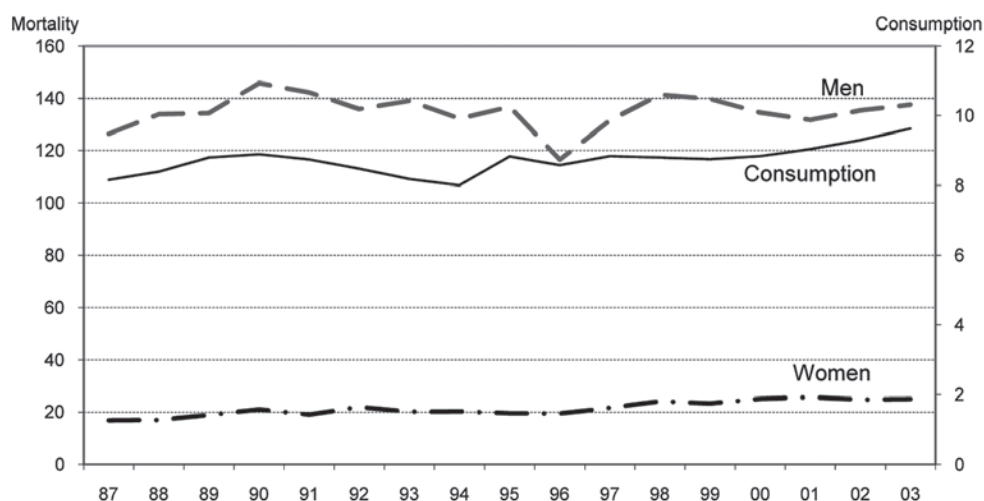
6 RESULTS

6.1 Differential trends in alcohol-related mortality before the reduction in alcohol prices in 1987–2003 (Study I)

The annual alcohol-related mortality rate comprising deaths of which alcohol was the underlying or a contributory causes averaged 3,140 (2,681 for men and 459 for women) in the period of study (1987–2003) peaking in 2003 (3,581 deaths). Alcohol-related mortality accounted for an average 11.2 per cent of all deaths among men and 1.9 per cent among women. Men accounted for 85 per cent of all alcohol-related deaths.

Total alcohol-related mortality among both men and women mainly followed the economic cycles and the trend in alcohol consumption in 1987–2003 (Figure 2). There was an increase in mortality, as well as in alcohol consumption, in the economic boom of the late 1980s, and corresponding decreases during the recession. Among men there was another high point in mortality in 1998, succeeded by a slight decrease and another slight increase at the end of the study period, whereas for women the highest rate of 2001 was followed by a slight decrease towards the end of the study period.

Figure 2. Alcohol consumption (litres of pure alcohol per capita among people aged 15 years and older) and age-adjusted alcohol-related mortality per 100,000 person-years among Finnish men and women in 1987–2003



As far as alcohol-related mortality according to different age groups is concerned, mortality increased among men between the late 1980s and early 1990s in all age groups, after which the trends began to diverge (Table 1; Figure 3 in Study I). There was a decrease of approximately 30 per cent among men under 45 from the beginning until the end of study period. Among the older age groups which clearly exhibited the highest level of alcohol-related mortality at the outset, there was a considerable increase: 15 per cent among those aged 45–59 years and over 31 per cent among the over-60s.

The causes most responsible for the increase in 1997–2003 among men aged 45 and older were alcohol-attributable diseases, which contributed 39 per cent of the increase, and intoxication-related cardiovascular diseases, accidents and violence at over 20 per cent each. The decrease in alcohol-related mortality among younger men was largely due to a significant decrease in intoxication-related accidents and violence, and also in alcohol-attributable diseases.

This divergence by age was also prominent among women. Alcohol-related mortality was low and stable throughout the study period among the youngest. It increased first but remained stable after 1990–91 among those aged between 30 and 44, and increased by 42 and 91 per cent, respectively, in the 45–59 and the oldest groups. Closer examination for the period 1997–2003 revealed that among women aged 45 and older, intoxication-related accidents and violence and alcohol-attributable diseases were responsible for a good 35 per cent each of the increase, and that among women below the age of 45, the decrease in alcohol-related mortality was due to a decrease in intoxication-related accidents and violence. Hence, the trends in alcohol-related mortality were associated with economic cycles and alcohol consumption among both men and women aged 45 years and over.

Education turned out to be an important factor in terms of producing diverging trends in mortality. Age-adjusted alcohol-related mortality was highest among men with the least education, and lowest among the most highly educated over the whole period. The level in the latter group remained stable throughout the study period, but increased among those with intermediate and basic education. Among women the difference between the basic and intermediate educational classes was, in relative terms, similar over the whole period. Absolute mortality differences increased rapidly, too, but there was a decrease in relative differences between those with a basic education and the highly educated because of a rapid proportional increase in alcohol-related mortality among the latter.

Table 1. Alcohol-related mortality per 100,000 person-years by age group, and age-adjusted alcohol-related mortality by education in 1987–2003, Finnish men and women aged 15+

	1987-1988		1990-1991		1994-1995		2002-2003		Change ^a	
	No. of Deaths	Deaths per 100,000	No. of Deaths	Deaths per 100,000	No. of Deaths	Deaths per 100,000	No. of Deaths	Deaths per 100,000		
Men										
Age group, years										
15-29	503	46.2	541	51.8	406	40.7	310	31.4	-14.8	-32
30-44	1460	117.8	1621	129.2	1342	112.7	899	82.7	-35.1	-30
45-59	1654	203.3	1900	224.3	2085	210.0	2730	233.0	29.8	15
60+	1132	168.6	1347	188.9	1441	190.6	1968	222.3	53.7	32
Education										
Basic	3131	157.5	3473	180.0	3252	172.3	3192	192.6	35.1	22
Intermediate	1427	112.5	1594	132.2	1525	125.0	2003	135.3	22.8	20
High	188	59.7	342	64.8	498	63.9	712	64.4	4.6	8
Total	4749	124.5	5409	140.2	5274	133.9	5907	142.9	18.4	15
Women										
Age group, years										
15-29	52	5.0	60	6.0	56	5.8	45	4.8	-0.2	-4
30-44	170	14.4	234	19.5	213	18.6	185	17.6	3.2	22
45-59	270	32.3	274	32.0	355	35.9	533	46.0	13.7	42
60+	177	16.0	252	22.1	212	18.2	383	30.6	14.6	91
Education										
Basic	496	21.7	567	26.1	518	28.5	626	40.3	18.6	86
Intermediate	154	19.5	197	15.5	233	17.1	371	24.3	4.8	24
High	20	5.3	58	13.3	84	12.1	149	13.9	8.6	163
Total	669	16.1	820	19.5	836	19.6	1146	26.0	9.9	62

^a Change from 1987–1988 to 2002–2003.

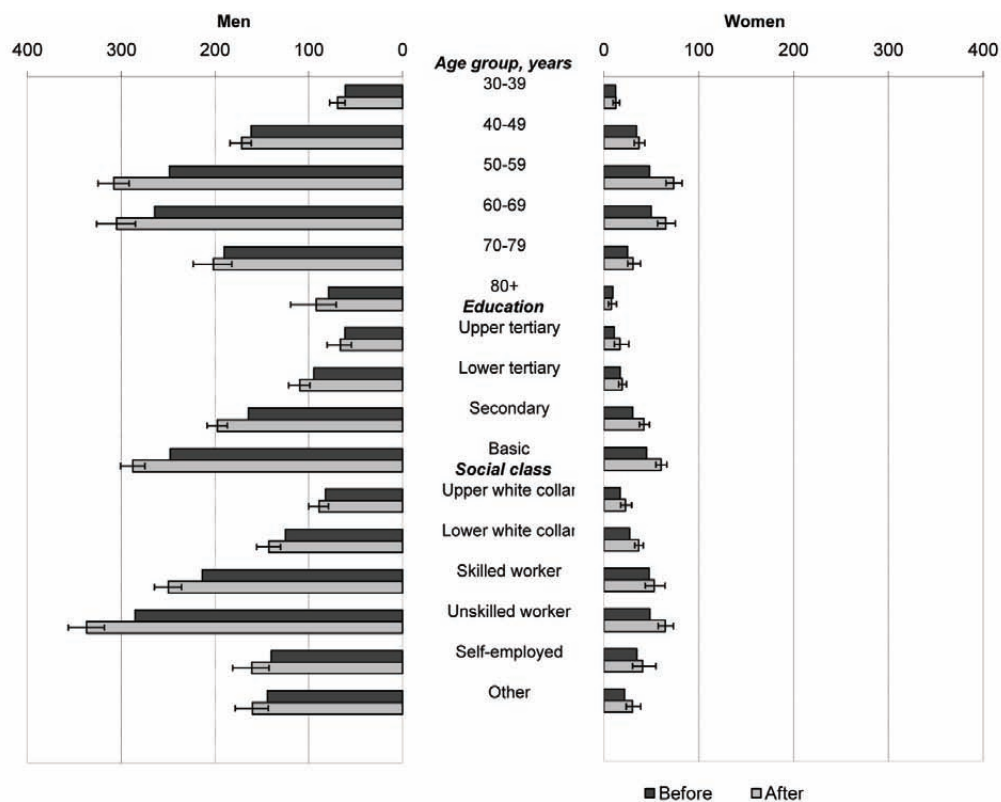
6.2 Changes and socioeconomic differences in alcohol-related mortality (Study III)

The increase in alcohol-related deaths from 2001–2003 (before the price reduction) to 2004–2005 (after the price reduction) was approximately 16 per cent (95% CI: 12.1, 19.4) (or 22 deaths per 100,000 person-years) among men and 31 per cent (95% CI: 22.0, 40.0) (or 8 deaths per 100,000 person-years) among women. Deaths due to chronic causes constituted 81 per cent of all alcohol-related deaths among men and 86 per cent among women in 2001–2003, increasing by 21 and 32 per cent, respectively, whereas the increases in deaths due to acute causes were seven and 27 per cent, respectively. Chronic causes comprised 82 per cent – and alcoholic liver diseases alone 39 per cent (38 and 41 per cent among men and women, respectively) – of the total increase in alcohol-related mortality. The increase in intoxication-related causes was mostly due to cases in which the underlying cause of death was cardiovascular disease or an accidental fall.

Before the reduction in the price of alcohol, alcohol-related mortality was clearly highest among men and women aged 50–69 (Figure 3; Figure 1 in Study III). The increase following the price reduction was highest in this age group, whereas among persons under the age of 35 the change was marginal (Figure 1 in Study III).

Among the over-30s the age-adjusted alcohol-related mortality rates in 2001–2003 and the change after the price reduction were clearly higher in absolute terms in the lower educational and social-class categories (Figure 3; Table 1 in Study III): before the price reduction both men and women with a basic education had a 3.5 (95% CI: 3.1, 4.0) and 4.1 (95% CI: 3.0, 5.8) times higher alcohol-related mortality rate than those with upper-tertiary education, for example. The rate increased by 39.9 versus 4.8 (non-significant) deaths per 100,000 person-years among men and 15.4 versus 6.0 among women. In terms of social class, there was an increase of 51.9 and 6.9 (non-significant) deaths per 100,000, respectively, among male unskilled workers and upper-white-collar employees, for example. The changes according to education and social class were quite similar among the younger (30–59) and older (60+) adults (results not shown).

Figure 3. Alcohol-related mortality per 100,000 person-years among men and women by age group and various socioeconomic indicators among persons aged 30+ years before (2001–2003)^a and after (2004–2005)^b the price in Finland^c



a. Unstandardised.

b. Obtained by multiplying the mortality rate before the change by the percentage change as estimated from the model.

c. 95-per-cent confidence intervals applied to the calculation of the estimates for 2004–2005.

The 2001–2003 rates varied strongly according to economic activity among persons aged 30–59 years (Table 2; Table 2 in Study III): the male and female long-term unemployed had 12-fold and 17-fold rates of age-adjusted alcohol-related mortality, respectively, compared with those who had a job. The price reduction had a marginal effect on the rates among the employed, whereas there was a large increase among the long-term unemployed and early-age pensioners.

TABLE 2. Alcohol-related mortality before (2001–2003) and after (2004–2005) the reduction in the price of alcohol by economic activity, Finnish men and women aged 30–59 years

	2001–2003			Change from 2001–2003 to 2004–2005			
	Deaths per year N	Deaths per 100,000 ^a	Age-adjusted relative rate	95% CI	% ^b	95% CI	Deaths per 100,000 ^c
MEN							
Economic activity					0.001 ^d		
Employed	530	59.9	1.00		2.9	-4.7, 11.1	1.7
Unemployed 25 mo+	477	783.7	12.11	11.28, 13.01	21.2	11.7, 31.5	166.1
Unemployed. other	153	336.9	5.54	4.99, 6.15	30.1	14.2, 48.1	101.4
Pensioned	482	619.1	8.15	7.58, 8.76	27.1	18.0, 36.9	167.8
Other	156	259.5	4.50	4.06, 4.99	21.5	6.3, 38.8	55.8
Total	1798	159.1			19.2	14.4, 24.1	30.5
WOMEN							
Economic activity					0.011 ^d		
Employed	97	11.7	1.00		8.0	-9.4, 28.7	11.8
Unemployed 25 mo+	89	218.3	16.68	14.12, 19.70	49.9	24.8, 79.9	16.9
Unemployed. other	36	57.1	5.03	4.03, 6.28	81.3	39.8, 135.1	99.2
Pensioned	98	162.1	11.11	9.43, 13.09	36.6	16.3, 60.5	152.3
Other	36	33.3	3.41	2.73, 4.29	54.0	18.3, 100.4	70.9
Total	356	32.2			33.6	22.4, 45.8	10.8

^a Unstandardised.

^b Derived from the age-adjusted interaction term between each variable and period.

^c Derived from the age-adjusted interaction term between each variable and period.

^d p-value for the difference in change obtained from a model that includes an interaction term between each variable and period.

Alcohol-related mortality rates and household income were inversely related before the price reduction (Table 2 in Study III). The patterns of change were similar in all income groups, with the exception of the lowest quintile. When household income, age and economic activity were introduced into the same model, the association was greatly reduced: for example, the relative rate in the lowest quintile decreased from 6.26 to 1.66. The differences between the income groups clearly decreased as well, but they did not disappear, nor did they become linear. Income-related changes were also examined separately among the employed on the one hand and the unemployed and pensioners on the other (data not shown). The patterns of relative change were not substantially different in these groups among men or women, even if the absolute changes were much higher among the unemployed and pensioners.

The patterns of socioeconomic differences in the change in mortality attributed to both chronic and acute causes and all alcohol-related causes were very similar among the over-30s (data not shown). In the case of acute causes however, the

differences in change – both in absolute and relative terms – were marginal according to education in both genders, and according to social class and income among women.

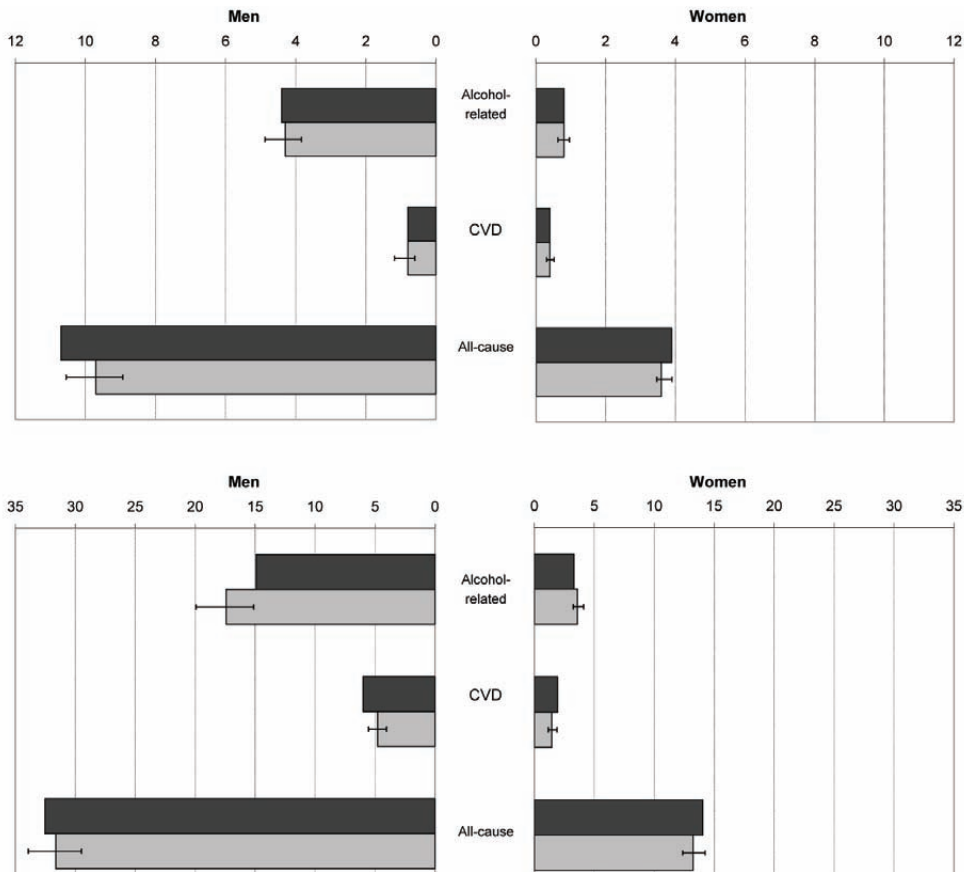
6.3 Time-series analysis of changes in alcohol-related and all-cause mortality (Study IV)

Figures 1–3 in Study IV depict the monthly rates of alcohol-related, cardiovascular diseases (alcohol-attributable cases excluded) and all-cause mortality among men and women in the different age groups. There appeared to be a clear age-specific pattern in the overall trends prior to the price reduction, which was similar among both men and women: alcohol-related mortality fluctuated or were stable among the under-50s, but was mainly increasing among the older groups. With regard to cardiovascular and all-cause mortality the trend was mostly downwards in all age and sex groups.

ARIMA time-series modelling was used in the formal assessment of the effect of the price reduction on alcohol-related, CVD and all-cause mortality in the 15–39, 40–49, 50–69 and 70+ age groups (Figures 4 and 5; Tables 1–3 in Study IV). In the first of these groups all-cause mortality among men and women decreased by 9.2 (95% CI: -16.5, -1.4) (one death per 100,000 person-years) and 8.3 (95% CI: -11.3, -0.0) (0.3 deaths per 100,000 person-years) per cent, respectively, after the price reduction (Figure 4; Tables 1–3 in Study IV). The change was marginal in the other categories.

There was an increase in, alcohol-related mortality increased of 17 (95% CI: 1.5, 33.7) and 11 (95% CI: -1.8, 24.6) per cent, respectively among men and women aged 40–49 years, which implied a monthly increase of 2.5 and 0.3 per 100,000 person-years (Figure 4; Tables 1–3 in Study IV), whereas cardiovascular-disease mortality decreased by 21 (95% CI: -32.2, -7.2) and 24 (95% CI: -40.5, -3.1) per cent, respectively, implying 1.2 and 0.5 fewer deaths per month. The change in all-cause mortality was not statistically significant.

Figure 4. Monthly rates of alcohol-related, CVD and all-cause mortality per 100,000 person-years before and after the price reduction, men and women aged 15–39 (upper figures) and 40–49 (lower figures) ^{a,b}



a. Obtained by multiplying the mortality rate before the change by the percentage change as estimated from the natural logarithmic ARIMA time-series models.

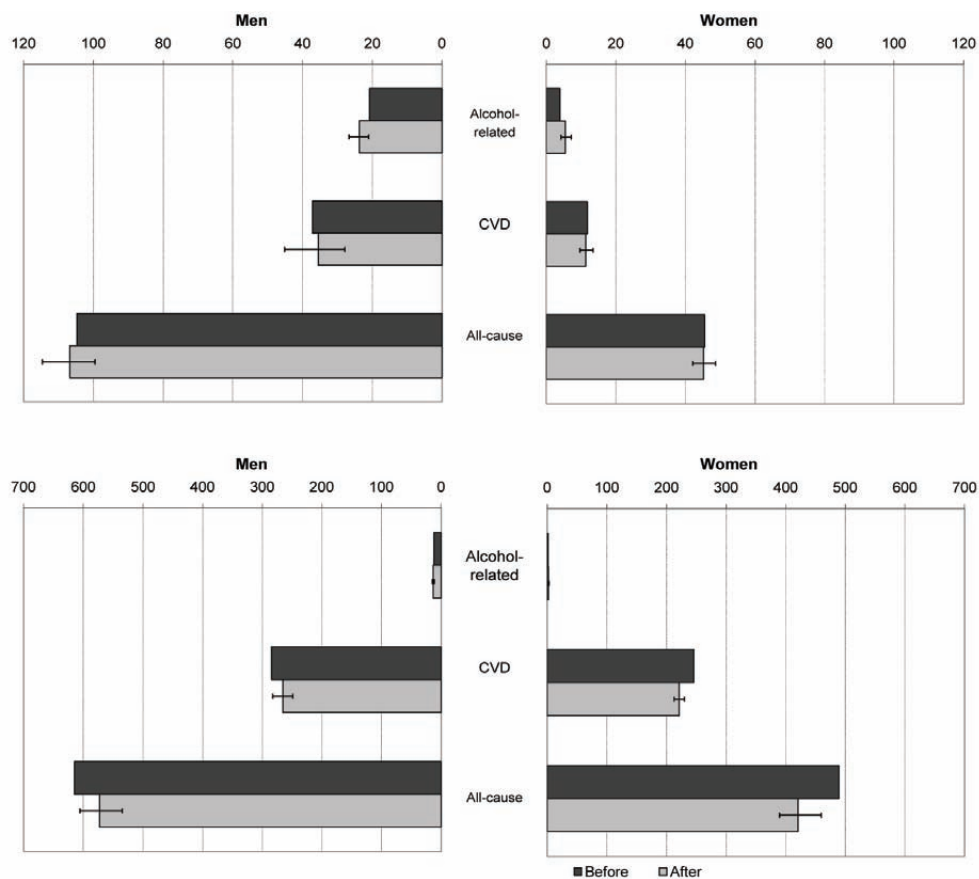
b. 95-per-cent confidence intervals applied to the change estimates derived from the models.

Among men and women aged 50–69 years (Figure 5; Tables 1–3 in Study IV), the impact parameters (0.13 and 0.33) suggested that the price reduction produced an increase of 14 (95% CI: 1.1, 28.0) and 40 (95% CI: 7.1, 81.7) per cent, respectively, in the alcohol-related mortality rate, which implies an increase of 2.9 and 1.6 deaths per 100,000 per month. In contrast, the change was marginal in CVD and all-cause mortality.

The change in alcohol-related mortality was not statistically significant among men and women aged over 69 years but the estimated decrease in CVD mortality was seven (95% CI: -12.4, -0.7) and 10 (95% CI: -13.5, -6.5) per cent, respectively,

which implies a monthly decrease of 19.1 and 24.8 deaths per 100,000 person-years, respectively (Figure 5; Tables 1–3 in Study IV). There was a decrease in all-cause mortality of seven (95% CI: -13.0, -1.5) and 14 (95% CI: -19.5, -8.2) per cent after the price reduction, which implies a monthly decrease of 42 and 69 deaths per 100,000, respectively.

Figure 5. Monthly rates of alcohol-related, CVD and all-cause mortality per 100,000 person-years before and after the price reduction, men and women aged 50–69 (upper figures) and 70+ (lower figures) ^{a b}



a. Obtained by multiplying the mortality rate before the change by the percentage change as estimated from the natural logarithmic ARIMA time-series models.

b. 95-per-cent confidence intervals applied to the change estimates derived from the models.

The analyses on cardiovascular mortality were performed again so that coronary operations were added into the models as control series. However, the estimates of the impact remained essentially the same (data not shown). The effect of the price reduction on mortality due to ischemic heart disease was examined

separately given the indications in the literature that it is the cause of death on which the effect may be the most clearly beneficial. The estimates were larger than for mortality in the other CVD categories (data not shown): among men and women aged over 69 the estimated decrease in mortality due to ischemic heart disease was 8.9 (95% CI: -12.5, -5.1) and 12.5 (95% CI: -15.1, -8.2) per cent, respectively, and it was 6.6 (95% CI: -16.2, 4.2) and 8.1 (95% CI: -14.4, -1.4) per cent, respectively, in the other CVD categories. Moreover, including winter cold as a control variable affected the results only marginally.

The net monthly sum of alcohol-related deaths estimated to be attributable to the price reduction and CVD deaths estimated to have been prevented by it was 18 saved lives per 100,000 person-years among men aged over 69, and 25 among women of the same age. This net sum was clearly smaller than the estimated decrease in the model for all-cause mortality (42 and 69 fewer deaths, respectively) (Table 4 in Study IV). It could be concluded on the basis of these and some additional analyses regarding this gap between CVD and all-cause mortality that the decrease in CVD and COPD mortality accounted for 55 per cent of the decrease in all-cause mortality among men and 38 per cent among women. Moreover, a decrease in dementia and diabetes mortality accounted for an additional 12 per cent among women.

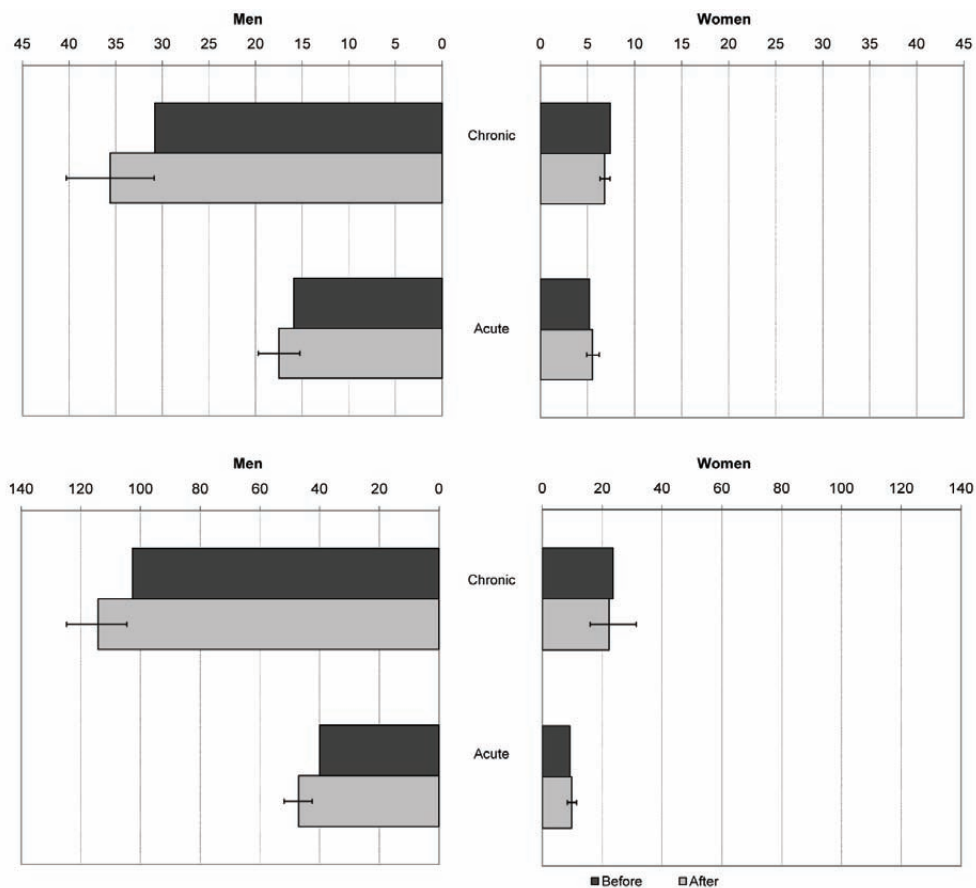
6.4 Time-series analysis of hospitalisation related to alcohol (Study V)

Figures 1–2 in Study V depict monthly rates of hospitalisation related to chronic and acute alcohol-attributable causes for men and women according to different age groups. The overall trends prior to the price reduction in the former were mainly downwards among the under-50s, whereas there was no distinct trend among those over 50 (Study V, Figures 1–2). With regard to acute causes there was no distinctive trend among the under-50s and a mainly increasing trend among the over-50s.

ARIMA modelling was used in the analyses in order to formally evaluate the impact of the reduction. In the case of chronic causes there was an estimated increase of 16 per cent (95% CI: 2.2, 30.9) and a decrease of eight per cent (95% CI: -14.7, -0.4) (4.8 more and 0.6 fewer monthly hospitalisations per 100,000 person-years), respectively, among men and women aged between 15 and 39 (Figure 6; Tables 1–2 in Study V), and an increase in the rate of acute causes of 10 per cent (95% CI: -1.3, 24.1) among the men, whereas the change was far to be statistically significant among the women.

There was an increase in hospitalisation due to both chronic and acute causes among 40–49-year-old men: the impact estimates suggested an increase of 11 (95% CI: 1.9, 21.5) and 17 (95% CI: 6.2, 29.9) per cent respectively, which implies a monthly increase of 11.5 and 7.0 hospitalisations per 100,000 person-years, respectively (Figure 6; Tables 1–2 in Study V). The change was marginal with regard to women.

Figure 6. Monthly rates of acute and chronic hospitalisation per 100,000 person-years before and after the price reduction, men and women aged 15–39 (upper figures) and 40–49 (lower figures) ^{a b}



a. Obtained by multiplying the hospitalisation rate before the change by the percentage change as estimated from the natural logarithmic ARIMA time-series models.

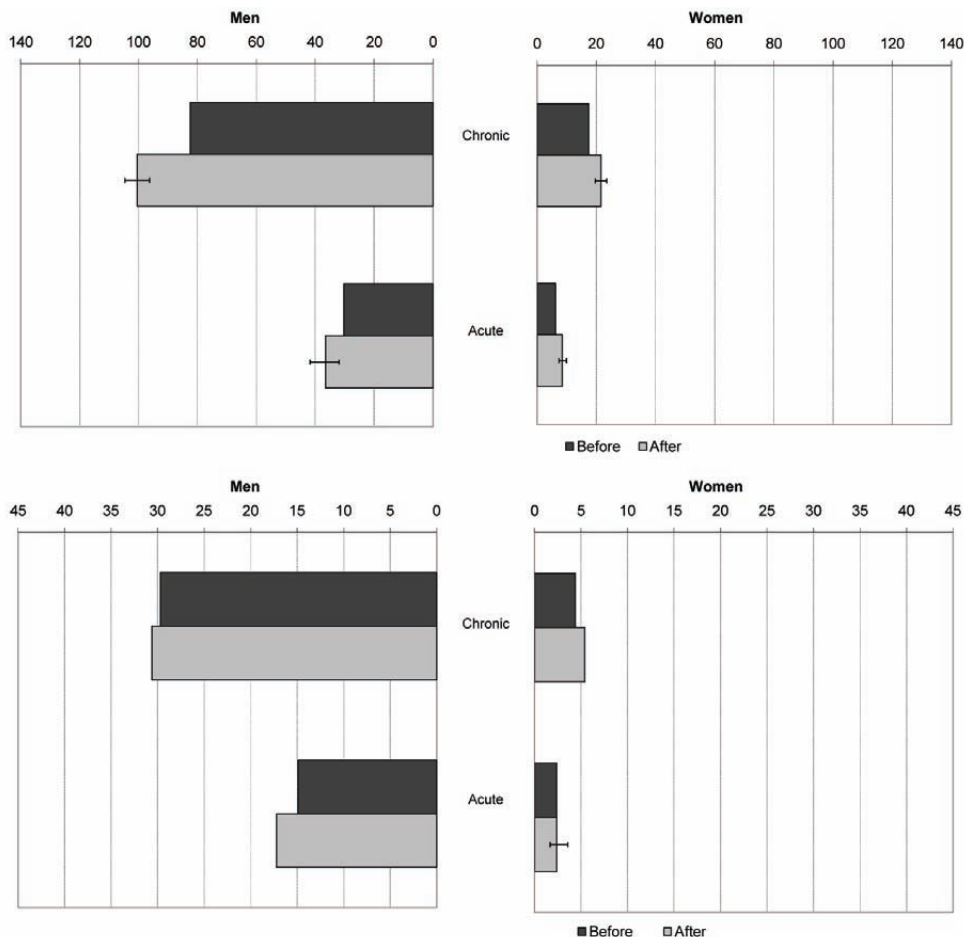
b. 95-per-cent confidence intervals applied to the change estimates derived from the models.

The hospitalisation rates clearly increased among men and women aged 50–69 years, and due to both chronic and acute causes (Figure 7; Tables 1–2 in Study V). The estimated increases in chronic cases were 22 (95% CI: 16.9, 27.0) and 23 (95% CI: 12.1, 34.1) per cent among men and women, respectively, which implies a monthly increase

of 18.0 and 4.0 hospitalisations per 100,000 person-years. The respective figures for acute causes were 20 (95% CI: 5.3, 37.7) and 38 (95% CI: 19.5, 59.1) per cent, an increase in the monthly hospitalisation rate of 6.2 and 2.3, respectively.

The change in the hospitalisation rate of the over-69-year-olds was not statistically significant (Figure 7; Tables 1–2 in Study V), although the increases of 23 per cent (95% CI: -5.5, 60.7) among women due to chronic causes and 15 per cent (95% CI: -3.5, 37.5) among men due to acute causes were not far from significant.

Figure 7. Monthly rates of acute and chronic hospitalisation per 100,000 person-years before and after the price reduction, men and women aged 50–69 (upper figures) ^{a b} and 70+ (lower figures)



a. Obtained by multiplying the hospitalisation rate before the change by the percentage change as estimated from the natural logarithmic ARIMA time-series models.

b. 95-per-cent confidence intervals applied to the change estimates derived from the models.

Separate analyses were also conducted for hospitalisation due to mental and behavioural disorders due to alcohol and other chronic causes (data not shown). All the impact estimates of mental and behavioural disorders (except among women aged 15–39) turned out to be larger than in the other chronic categories.

6.5 Changes in area-level variation in interpersonal violence (Study II)

The effects of the price reduction on interpersonal violence and the variation on the small-area level were investigated in the Helsinki Metropolitan area. The changes in the rates of interpersonal violence and disorderly conduct from 2002–03 to 2004–05 were mainly marginal (Table 3; Table 2 in Study II), the decrease of 14 per cent in violating domestic peace being the only statistically significant change. The decreases of 12 and 7 per cent, respectively, in robbery and domestic violence were close to significant.

Table 3. Distribution of delinquency and delinquency rates a before (2002–2003) and after (2004–2005) the change, Helsinki Metropolitan area^b

	Before		Change		
	N	Rate	Rate	%	p value ^c
Assault	10,246	6.33	0.28	4.5	0.161
Private homes	3,061	1.89	0.07	4.0	0.447
Public places	5,426	3.35	0.13	3.9	0.548
Robbery	1,054	0.65	-0.08	-12.4	0.054
Violating domestic peace	1,355	0.84	-0.11	-13.6	0.001
Rape	139	0.09	-0.02	-18.0	0.180
Domestic violence ^d	7,838	4.84	-0.33	-6.8	0.069

^a Amount of delinquency by risk-population and year x 1000.

^b Population aged 15 years and above 809,086.

^c p value for the change in delinquency rate from linear regression.

^d A category of emergency call-outs.

Two different regression models were used to assess the effect of area characteristics, i.e. mainly measures of social disadvantage, prior to the price reduction on the three types of interpersonal violence that were chosen for further analysis on the basis of their importance or observed associations in the preliminary analyses (Table 4; Table 3 in Study II). When all the area characteristics were added into the model one by one (model not shown), all except outmigration and manual class (i.e. five out of seven) had a significant relationship with all three types of interpersonal violence. However, when all measures of social disadvantage were entered simultaneously the effects overlapped so that only the characteristics

shown in Table 4 remained significant (Table 4; Table 3 in Study II). When other significant factors were controlled for, the assault rate was higher in tracts with a higher proportion of people with a basic education and outmigration, whereas other factors lost significance. The controlled model for assault in private homes revealed a higher crime rate where there was a higher proportion of single-parent families. As far as domestic violence was concerned, the final model indicated a higher rate in tracts with high proportions of manual-class membership and unemployment.

Table 4. The effects of area characteristics on interpersonal violence before the change (2002–2003) in the Helsinki Metropolitan area, linear regression ^{a b}

	Coefficient	Robust SE	Beta	p value
Assault^c				
Basic education (%)	2.62	0.53	0.38	<0.001
Outmigration	3.10	0.93	0.38	0.001
Assault private homes				
Single-parent family (%)	0.78	0.10	0.65	<0.001
Domestic violence				
Manual class (%)	1.12	0.25	0.34	<0.001
Unemployment	6.40	0.99	0.63	<0.001

^a Area characteristics were measured as continuous in the linear regression.

^b Multivariate model. All significant area characteristics are included in the analysis.

^c Excluding the centre of Helsinki.

Table 5 (Table 4 in Study II) confirms the higher levels of all types of interpersonal violence in low-status than in high-status tracts before the changes in the pricing of alcohol. The change from 2002–03 to 2004–05 on two different indicators of interpersonal violence varied according to the area characteristics. The change in the incidence of assault in high-status tracts was marginal. According to the interaction analysis, change in the assault rate according to area measures was statistically significant only in the manual class: there was a higher increase in tracts with a high proportion of manual-class inhabitants. The difference in the direction of change was similar among those with a low educational level, although not statistically significant.

Changes in incidences of assault in private homes did not differ by area characteristics (Table 4 in Study II). As far as domestic violence was concerned there was some indication that the change was more favourable in the tracts with higher unemployment and lower incomes. A similar pattern also emerged for the other

characteristics (except outmigration), i.e. the rate of domestic violence decreased most in the lower-status tracts, but the change was not statistically significant. The fact that many of the seemingly substantial differences remained non-significant was at least partly attributable to the distribution of the categories: intermediate categories covered 60 per cent of the population.

Table 5. Annual delinquency rates (per 1,000) before and after the price change of alcohol, and the p values for the linear regression interaction (bivariate models) for area characteristics and time in the Helsinki Metropolitan area ^a

	Assault ^b			Domestic violence		
	Before	Change %	p value	Before	Change %	p value
Basic education (%)			<i>0.082</i>			<i>0.123</i>
Low	3.1	-3.9		2.3	-6.0	
Intermediate	5.5	3.4		4.9	-2.4	
High	8.7	19.3		7.8	-14.5	
Manual class (%)			<i>0.048</i>			<i>0.383</i>
Low	2.5	0.9		2.1	-1.1	
Intermediate	5.8	2.0		4.9	-5.5	
High	8.0	20.6		8.3	-10.3	
Unemployment			<i>0.248</i>			<i>0.016</i>
Low	2.3	0.3		2.3	-0.3	
Intermediate	5.8	6.8		4.7	-1.7	
High	8.2	10.2		8.8	-16.6	
Income			<i>0.248</i>			<i>0.022</i>
High	2.8	0.2		2.3	-3.2	
Intermediate	6.0	8.0		4.9	-1.7	
Low	7.4	8.2		8.4	-17.1	
Outmigration			<i>0.431</i>			<i>0.070</i>
Low	3.0	-1.6		3.3	-8.3	
Intermediate	5.4	8.2		5.9	-7.9	
High	8.6	8.7		4.1	-0.9	

^a Area characteristics were measured as continuous variables in the linear regression.

^b Excluding the centre of Helsinki.

7 DISCUSSION

Natural experiments have been called for (Chaloupka et al. 2002) in research on the association between the pricing of alcohol and alcohol-related harm. The present study investigated the effects of the 2004 reduction in alcohol prices on alcohol-related and all-cause mortality, alcohol-related hospitalisation and interpersonal violence in Finland, and how these varied according to socio-demographic factors and neighbourhood characteristics. A rare natural experimental design was thus used. Time-series analysis, which takes account of trends and seasonal variation allowed examination of the effects of the price reduction on mortality and hospitalisation. The before/after design contributed to the study regarding the investigation of the socioeconomic differences in mortality and the differences in interpersonal violence. Differential trends in alcohol-related mortality before the price reduction were also examined.

7.1 The effects of the price reduction on specific causes of mortality and hospitalisation

7.1.1 Alcohol-related mortality and hospitalisation

Alcohol-related deaths after the price reduction increased among men aged 40–49 years and among men and women aged 50–69 years when trends and seasonal variation were taken into account: the mean rates increased by 17, 14 and 40 per cent, respectively, which implies 2.5, 2.9 and 1.6 additional deaths per month per 100,000 person-years.

The chronic hospitalisation rate increased among men below the age of 70. It was highest among the 50–69-year-olds at 22 per cent, which implies a monthly increase of 18.0 hospitalisations per 100,000 person-years, and there was an 11 and a 16 per cent (11.5 and 4.8 per month) increase among those aged 40–49 and 15–39, respectively. Among the women there was an increase of 23 per cent (4.0 per month) in the 50–69-year-olds, and a decrease in the under-40s. Acute hospitalisations increased by 17 and 20 per cent (6.2 and 7.0 per month) among men aged 40–49 and 50–69, respectively, and by 38 per cent among women aged 50–69 (2.3 per month).

The post-price-reduction increase in alcohol-related mortality was mainly attributable to chronic causes: chronic deaths accounted for more than 80 per cent of the total increase in alcohol-related mortality. With regard to alcohol-related

hospitalisation, chronic causes constituted more than 70 per cent of the total increase. It is of major significance that the increases in both alcohol-related mortality and hospitalisation were mainly attributable to chronic rather than acute causes. One would rather expect that an abrupt price reduction would be followed, if any change, an increase in acute rather than chronic causes of mortality and morbidity. Previous evidence of such an association is scanty and based mainly on historical data, but sudden fluctuations after price changes in both types of causes have been reported (Skog 1993; Edwards et al. 1994; Nemtsov 1998; Norström and Ramstedt 2005; Skog and Melberg 2006). A previous time-series analysis of alcohol-positive sudden deaths recorded a 17 per cent increase in 2004 compared to 2003 (Koski et al. 2007).

According to the before/after analysis, alcoholic liver diseases alone constituted 39 percent of the increase in total alcohol-related mortality after the price reduction. This increase with a concurrent increase of 1.2 litres in per-capita alcohol consumption is higher than would be expected on the basis of a previous time-series analysis of the longer-term connection between liver cirrhosis mortality and per-capita consumption in Finland, but it is in line with the effect size observed for Sweden in 1950–1995 (Ramstedt 2001). These findings are also in accord with those of an earlier U.S. study based on annual state-level data on alcohol sales and mortality from 30 states in 1962–1977 and concluding that increases in excise taxes on distilled spirits would reduce deaths from liver cirrhosis (Cook and Tauchen 1982). As far as the relation between alcohol consumption and disease is concerned, one must consider that not only present but also past consumption impinges on the risk of alcohol-attributable disease. It has been suggested that the latency period for liver cirrhosis could be very long – 20 years of excessive drinking may be needed (Skog 1980). However, there was evidence of an instantaneous response to changes in consumption on the aggregate level with regard to cirrhosis mortality, in France during WWII and in Russia after 1990, for example (Edwards et al. 1994; Nemtsov 1998). However, one must be cautious in making interpretations based on these historical and contemporary Russian data on alcohol consumption and mortality (Leon et al. 1997; Rehm 2009; Zaridze et al. 2009). This seeming contradiction in the case of a rapid increase in cirrhosis-related mortality is best understood in terms of the water-glass analogy: those who died from cirrhosis in that short period after the price reduction already had their water glass almost full, and the increased consumption took a higher number of previous heavy drinkers over the rim.

As far as hospitalisation due to chronic causes is concerned, the increase was almost entirely attributable to mental and behavioural disorders, whereas the

changes related to diseases of the liver and pancreas, even if mostly positive, could not be separated from random changes. These findings are somewhat in line with those reported in earlier studies from Sweden covering the period 1969–2001 and Holland over the period 1970–1994, according to which the trends in liver-disease hospitalisation followed the trends in consumption only to some extent or not at all (Stokkeland et al. 2006; Adang et al. 1998). Furthermore, a study conducted in Finland covering the period 1969–1975 reported mostly larger increases in hospitalisation due to mental and behavioural disorders (increases in admission rates ranging from 1.7 to 2.1 among men and 2.3 to 2.4 among women) than to cirrhosis and pancreatitis (increases ranging from 1.2 to 2.0 among men and from 1.0 to 1.4 among women) following an increase in alcohol sales per capita from 2.9 litres in 1968 to 6.5 litres in 1974 (Poikolainen 1980). Nevertheless, the association between consumption and hospitalisation was not formally estimated. A time-series study based on quarterly data conducted in the Stockholm area covering the period 1980–1994, however, reported only a weak and non-significant relation between alcohol sales and hospital admissions on account of alcoholism, alcohol intoxication and alcohol psychosis, whereas the relation was significant with regard to cirrhosis admissions (Leifman and Romelsjö 1997). Differences in the scope and length of the follow-up and in methodological features, or differences in how the consumption change translated into changes in drinking patterns or changes in the distribution of consumption, may account for the discrepancies between the findings reported in this and the current study.

There was also an increase in hospitalisation attributable to acute causes and differences on at least two factors, apart from the magnitude of the change, in comparison with chronic causes. First, it appears that there was slightly a more systematic increase in acute than in chronic causes among women below the age of 70, but not among men. This could be attributable to the changes in drinking patterns reported in a recent Finnish survey-based study: the number of binge-drinking occasions increased by 63 per cent among women between 2000 and 2008, whereas it remained unchanged among men (Mustonen et al. 2009). Secondly, chronic alcohol-related hospitalisation could be regarded as an indicator of more severe adverse alcohol-attributable problems. For example, hospitalisation due to alcohol intoxication may certainly be a serious event as such, but it does not necessarily imply frequent excess alcohol drinking and its consequences. The formation of chronic and acute categories for the analysis thus improved the validity of the study in this respect.

It is worth considering the different nature of the two categories of chronic hospitalisation with regard to mortality. Mental and behavioural disorders primarily

reflect mental health, which is not necessarily captured in studies on mortality, whereas the correspondence between mortality and hospitalisation for diseases of the liver and pancreas should be closer. It is thus not inconsistent to claim that the increase in hospitalisation was due to an increase in mental and behavioural disorders rather than in diseases of the liver and pancreas, which together with alcoholic cardiovascular disease accounted for much of the increase in mortality after the price reduction. However, it must be noted that there was also an increase in hospitalisation due to other chronic causes (of which around 80 per cent were diseases of the liver and pancreas) but it could not be observed as precisely as with alcohol-attributable mental and behavioural disorders in which the number of hospitalisations was 2.5-times higher.

Overall, the attribution the increase in hospitalisations was mainly to mental and behavioural disorders is important for three reasons. First, hospitalisation for these causes predict premature mortality: it has been found that a person with alcohol dependence syndrome is likely to die 15 years earlier than the average member of the general population (Moos et al. 1994), the leading causes of death being heart disease, cancer, accidents and suicide (Schuckit 2000). Secondly, this finding may explain some of the above-mentioned discrepancy related to men below the age of 40: hospitalisation increased (73 per cent of the chronic cases were attributable to these such and behavioural disorders) but mortality did not. One contributing factor here is the time lag: shorter latency periods have been found for alcohol-related mental and behavioural disorders than for other major alcohol-related diseases. According to Schuckit (2000), the average alcoholic first experiences a clustering of major alcohol-related life problems in the mid-20s to early 30s, and first seeks treatment in their early 40s – after more than a decade of difficulties. It is thus likely that by the age of about 30 approximately half of those who fulfil the criteria for alcoholism have already reached that stage (Schuckit 2000). Liver cirrhosis, for example, requires up to 20 years of excessive drinking (Skog 1980). Thirdly, alcohol dependence is often combined with other severe mental disorders: a study conducted in the US showed that 37 per cent of those afflicted had a comorbid mental disorder (Regier et al. 1990). The increase in alcohol abuse among younger men is somewhat in accord with the results of a survey-based study showing a respective increase in the proportion of heavy drinkers of 25 and seven per cent among men aged 15–29 and 30–49 between 2000 and 2008, even if the change was not statistically significant (Mäkelä et al. 2009). Moreover, it remains unclear whether there has been any specific change since 2004. On the other hand, alcohol consumption generally declined in these age groups and the estimated proportion of abstainers among 15–29-year-olds doubled (Mäkelä et al. 2009).

7.1.2 CVD and all-cause mortality

The time-series analysis revealed beneficial effects of the price reduction in terms of a clear decrease in CVD mortality in the over-70s. Negative point estimates were found for men and women in most other age groups, too. Coronary operations and winter cold were included as control series in the models, but their effects were marginal.

There is extensive evidence that light to moderate drinking is associated with cardioprotective benefits on the individual level (Wannamethee and Shaper 1999; Corrao et al. 2000; Agarwal 2002; Rehm et al. 2003; Reynolds et al. 2003; O'Keefe et al. 2007). Most studies report J- or U-shaped curves, meaning that light to moderate drinkers are less at risk than abstainers, and heavy drinkers run the highest risk (e.g., O'Keefe et al. 2007). Definitions of light to moderate drinking in cardioprotective terms vary from 10–20 g for women and 20–72 g for men per day (Rimm et al. 1999; Corrao et al. 2004).

A larger effect on deaths due to ischemic heart disease than to other cardiovascular diseases lends credence to these results, given that cardioprotective effects have been reported to be most obvious in ischemic heart disease (O'Keefe et al. 2007), most epidemiological cohort studies showing reductions in risk of approximately 30 to 35 per cent from light to moderate drinking (Kabakambe et al. 2005; Mukamal et al. 2005). Previous population-level evidence on this association is scarce, however. This finding in the present study is somewhat at odds with the results of a cross-European time-series study on the relation between alcohol consumption and ischemic-heart-disease mortality conducted in 15 European countries in 1950–1995: it reported a random distribution of insignificant negative and positive alcohol-effect estimates (Hemström 2001). Differences in the scope of the data account, at least to some extent, for the discrepancy in findings. For example, the current study did not include alcohol-attributable cases (17 per cent of all cardiovascular-disease deaths) in the analyses of cardiovascular-disease mortality because they were included in the models of alcohol-related deaths. Moreover, the cross-European study was limited to the under-75s and covered smaller consumption changes overall, and thus may not have had the capacity to identify effects that are mainly characteristic of 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, have fostered moderate consumption and in at least some parts of the population. This is in concord with two recent Finnish surveys reporting an increase in alcohol consumption in the 2000s especially among those aged 50–69 (Mäkelä et al. 2009), and a slight

increase among the over-65s (Sulander et al. 2006) whose drinking is reported to be primarily low-to-moderate, (Mäkelä et al. 2009) and thus beneficial in nature. There was no increase in consumption among the under-50s.

The estimated effect of the 2004 price reduction on all-cause mortality was beneficial to people over 69 years of age, as expected on the basis of the aforementioned results and the prominence of cardiovascular mortality at older ages. The point estimates were negative, even if statistically non-significant, in the younger age groups, too. The J- or U-shaped association between low-to-moderate alcohol intake and all-cause mortality at all ages is well-established (Shaper et al. 1988; Boffetta and Garfinkel 1990; Klatsky et al. 1992; Doll et al. 1994; Grønbaek et al. 1994; Fuchs et al. 1995; Thun et al. 1997; Yuan et al. 1997; Baglietto et al. 2006; Di Castelnuovo et al. 2006). A recent meta-analysis of individual epidemiological studies revealed an association between moderate daily consumption and a mortality reduction of 18 per cent (Di Castelnuovo et al. 2006).

Previous time-series research has reported an association between a one-litre increase or decrease in consumption and a corresponding increase or decrease of between 1.3 and three per cent in total mortality in separate analyses of 25 and 14 European countries and Canada (Her and Rehm 1998; Norström 2001, 2004). Alcohol sales were used as a proxy for per-capita consumption in all of these studies. It seems from the present study that the one-litre increase in per-capita consumption that occurred in Finland in 2003–2004 led to very little change in all-cause mortality among persons aged below 70, but a decrease among the older population. In the study of 14 European countries (Norström 2001), the only one of these studies stratified by age, the estimates among covering those aged 70 or more were mainly non-significant, being very close to zero in medium- and high-consumption countries, and small but positive in low-consumption countries. The present study is the first aggregate-level time-series analysis to show a clear protective effect of changes in alcohol prices on mortality among the over-69-year-olds. One reason for the discrepancy with earlier studies could lie in the estimation of the effects of a single abrupt and large policy change rather than of numerous often smaller incremental changes over a longer follow-up period. Moreover, annual data used in the earlier studies are short-term according to time-series criteria (Yaffee 2000), but long-term in terms of historical time involving a risk of numerous uncontrolled confounding factors.

Cardiovascular-disease mortality only partially captures the estimated overall beneficial effects of the tax change on all-cause mortality among those aged over 69. The current study also found evidence of a decrease in mortality due to chronic

obstructive pulmonary diseases among both men and women in this age group, 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 at all ages: research on the first-mentioned cause is scarce (Beaglehole and Jackson 1992; Tabak et al. 2001ab; Agarwal 2002; Ruitenberg 2002; Mukamal et al. 2003; Howard et al. 2004; Deng et al. 2006; O’Keefe et al. 2007). There was no observable beneficial or harmful effect of the tax change on malignant neoplasms, i.e. there was no apparent protective effect for this broad group of causes of death on which alcohol consumption is not expected to have such an effect. With respect to possible confounders it should be noted that the tax cuts in 2004 were specific to alcohol, but the opening of borders also applied to cigarettes but this had very little effect on smoking rates (Sulander et al. 2006; Helakorpi et al. 2009). The change around the year 2004 has echoed pre-existing favourable trends with regard to physical activity and diet (Sulander et al. 2006; Helakorpi et al. 2009). However, the possibility remains that the estimated reduction in all-cause mortality in particular, but also in CVD, after the tax intervention of 2004 was attributable to lower risk factors and better treatment, or to an improvement in winter conditions not captured by the variables used in this study. Nevertheless, the time-series method should make such confounding relatively unlikely, and the possible influence of coronary operations and low winter temperatures were controlled for.

Care should be taken in interpreting the favourable changes in CVD and all-cause mortality associated with a reduction in the price of alcohol. It is worth pointing out that alcohol-related death is specific – as this is the case already by definition – to alcohol and thus understandably responsive to changes in alcohol prices, whereas improvements in CVD mortality and other causes of death may come through a multitude of other modifiable factors such as an improved diet, increased physical activity and smoking cessation. For example, in Finland from the 1980s until the mid-1990s, favourable trends regarding risk factors including smoking, blood pressure and cholesterol, explained 53–72 per cent and improved treatment 23 per cent of the declining trends in CVD mortality (Laatikainen et al. 2005).

One could question the possibility of showing observable beneficial effects within such a short period after the price reduction. However, little is known about the amount of exposure time that is needed in order to achieve cardioprotective or other benefits, and there is no reason to assume that it is long on the population level. Liver-cirrhosis mortality is a good case in point: despite its long latency period it may respond almost instantaneously to changes in consumption. There is evidence of this not only in a few previous studies (Edwards et al. 1994; Nemtsov 1998) but also in this one (Study III).

7.2 The differential effects of the price reduction on mortality and hospitalisation

7.2.1 Age and gender differentials

Two main findings of this study were that rates of alcohol-related mortality and morbidity increased considerably on the whole after the reduction in alcohol prices, and the effects of the reduction on these rates varied widely among the population subgroups.

Time-series analysis revealed an increase in alcohol-related mortality among both men and women aged 40 years or more, and virtually no change among younger people, whereas Study I showed a declining trend in alcohol-related mortality in the 1990s and early 2000s among the under-45s. On the other hand, alcohol-related hospitalisations increased by 16 per cent after the price reduction among men aged under 40. On the basis of both the mortality and hospitalisation studies it could be said that the mortality impact was highest, in both relative and absolute terms, among persons aged 50–69 years.

The finding that the level of alcohol-related mortality did not increase after the price reduction but, if anything, decreased among younger people is of great interest because alcohol consumption is generally more responsive to increases in price among youths and young adults than among older people (Chaloupka et al. 2002). The implication is that this is also true when the price goes down. The observed increase in alcohol-related hospitalisation among men aged under 40 does not change the fact that the price reduction mainly affected the over-50s.

The different developments according to age group would appear to be accounted for by the varying changes in alcohol consumption: survey-based studies have shown an increase among the over-50s after the mid 1990s, and also after 2004, whereas it was stable or decreased among younger Finns (Helakorpi et al. 2007, 2009; Mäkelä et al. 2009). The increase was particularly large and harmful in terms of drinking patterns among the 50–69-year-olds between 2000 and 2008 (Mäkelä et al. 2009). These results suggest that younger generations may be adopting less damaging alcohol-consumption patterns, even if recent surveys do not fully confirm this (Mäkelä et al. 2009; Mustonen et al. 2009). Overall, the phenomenon (i.e. a decrease in alcohol consumption and alcohol-related harm among younger generations) rather seems to be indicative of a longer-term change than a consequence of the price reduction.

With regard to gender differences in the effects, the increase in alcohol-related mortality and hospitalisation, on the whole, was larger among men than among women, consistently in absolute terms, and in most cases also in relative terms. The larger relative increase among women in some cases could be attributable in part to the consistently much lower rates before the price reduction. Study I confirms this pre-price-reduction trend. Gender differences in alcohol consumption and the related problems are universal and well-documented (see e.g., Plant 1990; Wilsnack et al. 2000). However, the large increases in both alcohol-related mortality and hospitalisation among women aged 50–69 years is indicative of an increase in alcohol abuse. Two recent survey-based studies confirm this: the proportion of heavy drinkers increased from 0.3 to 1.5 per cent (Mäkelä et al. 2009), and the proportion of binge-drinking occasions increased from three to five per cent between 2000 and 2008 among women aged 50–69 years (Mustonen et al. 2009). Gender convergence in this respect has also been reported elsewhere (e.g., McPherson et al. 2004).

7.2.2 Socioeconomic differentials

There is ample evidence of socioeconomic differences in health (Mackenbach et al. 1997, 1999, 2008; Martikainen et al. 2007; Leinsalu et al. 2009; Stirbu et al. 2009). For example, a study based on data from 22 European countries reported substantially higher mortality rates in groups of a lower socioeconomic status, and it was only the magnitude of the inequalities between the groups that varied across countries (Mackenbach et al. 2008).

The effects of the price reduction on alcohol-related mortality were assessed in the present study according to four socioeconomic factors, which implies a broad definition of socioeconomic status: on top of the three conventional factors of education, household income and social class, economic activity was also used. In addition, Study I examined educational differentials in trends of alcohol-related mortality prior to the price reduction.

The most salient finding was a huge gap between the employed and those without work among the 30–59-year-olds. There was a particularly strong increase among the long-term unemployed and early-age pensioners, which was mostly attributable to chronic causes, but deaths from acute causes also increased to some extent. At least two relevant but interlaced explanations for this divergence between the employed and others could be offered. First, for reasons to do with selection and causal processes (Martikainen and Valkonen 1996, 1998), those drinking heavily before the price reduction are overrepresented among the unemployed and early-

age pensioners, and chronic conditions may more quickly respond to an increase in consumption in the form of an increase in deaths. Secondly, the unemployed and early-age pensioners include more poor individuals whose drinking may have previously been restricted by the higher price of alcohol. With regard to acute causes, the mortality rates of the long-term unemployed increased but remained almost unchanged among the employed. This could indicate that hazardous alcohol consumption has not increased substantially among employed individuals.

The finding that the increase in alcohol-related mortality after the price reduction was, in absolute terms, higher in the lower educational and socioeconomic groups is in accordance with the long-term trends observed in Study I and in two earlier studies conducted by Mäkelä and colleagues in the Finnish context (1997, 2000): the pre-existing differential trends were clearly more detrimental to people of a lower socioeconomic and educational status than in 1987–2003. Moreover, as reported in Study I, in the past 20 years economic fluctuations have not been associated with alcohol-related mortality among better educated men and women. In the lowest educational group, alcohol consumption and related mortality appeared to follow economic cycles, increasing during upturns and decreasing during downturns. Differences in harmful alcohol consumption mainly explain these results. According to one survey both men and women with the lowest level of education increased their annual binge drinking occasions in the economic upturn of the 2000s, whereas the more highly educated kept it stable or even reduced it (Mäkelä et al. 2009). Furthermore, changes in alcohol-related mortality by socioeconomic indicators could be partly attributable to beverage preferences: the beverages consumed by the lower educated were affected by the price changes to a greater extent than those consumed by the more highly educated (Metso et al. 2002). Those with a lower education are more likely to consume spirits than the more highly educated, and spirits were most affected by the price reduction (Metso et al. 2002; Mäkelä and Österberg 2009).

Although household income was inversely associated with mortality before the price reduction, the effect was not the largest among the poorest. One might assume that if, on the whole, alcohol-related mortality duly increased, it would happen among poor people whose consumption may have been restricted the most by the higher price. The lack of a perfect gradient in change according to household income could reflect the possibility that alcohol is still too expensive for the poorest. In any case, this peculiarity in the results diminished when economic activity was controlled for.

7.3 The effects of the price reduction on interpersonal violence

It appeared that the price reduction had only a marginal effect on interpersonal violence in the Helsinki Metropolitan area, and there was even a decrease in the incidence of domestic violence. This was not surprising, for at least for two reasons. First, the increase in alcohol consumption in adverse terms that followed the price reduction was due largely to the increase among heavy drinkers, who constitute a relatively small population group (Skog 1985; Mäkelä et al. 2009). Secondly, there is scant and weak evidence of any association between alcohol prices and consumption and interpersonal violence. As Gmel and Rehm (2003) put it, although drinking is involved or associated with many social problems (including aggression and violence against others), there is no clear evidence that it causes them. Moreover, much of the research in this area is methodologically flawed (Gmel and Rehm 2003; Gil-González et al. 2006). These findings also support the results of a recent study based on time-series analysis conducted on the national level in Finland indicating no increase in violent crimes in 2004 (Sirén and Lehti 2006).

These effects on interpersonal violence were investigated on the area level according to six measures of social disadvantage and one measure of residential instability. The effects of price change on the rates of interpersonal violence were small, as were the differences in impact between high-, intermediate- and low-status areas, although a clear gradient was evident before the price reduction. This is in accord with previous studies demonstrating a significant relationship between measures of area-level social disadvantage and interpersonal violence (Kawachi et al. 1999; Gruenewald et al. 2006). However, there was an adverse development in assault in low-status tracts characterised by a high proportion of manual-class membership and a low educational level. The role of education in creating inequality and contributing to other socio-structural characteristics is well established on the individual level (Ross and Wu 1995; Braveman et al. 2005). With regard to domestic violence, not only did the rate decrease on the whole, but also the decrease was largest in the low-status tracts in which the rate was highest before the price reduction. This favourable trend could be attributed to the increasing public intolerance of domestic violence, which has catalysed actions within society in terms of legislation, for instance, in order to reduce it. Hence, the declining trend does not necessarily relate to the price reduction.

Most previous studies on the association between interpersonal violence and disadvantage have been conducted in the US and in metropolitan contexts, in which

disadvantage is likely to be concentrated to a much larger extent than in a Nordic welfare setting in which the state intervenes more in market processes in order to reduce poverty (Esping-Andersen 2000). Finland is one of the most egalitarian societies measured in terms of income inequality (Atkinson 2000). Accordingly, the current study strengthens the relationship between measures of area-level disadvantage and interpersonal violence in acknowledging the existence of such an association exists even in a Nordic welfare state.

7.4 Methodological considerations

The present study has several methodological strengths. First, the natural experiment as a research design has a number of benefits regarding the need, or as a matter of fact the lack of need, to use control variables (as discussed in Chapter 4.1). Secondly, the investigation of the effects of the price reduction was based on numerous data sets, which enabled a versatile and accurate approach to the research problem. Not only were all the major data sets for each of the five studies different, additional data sets were used in several studies in order to improve the validity of the analyses. For example, data on hospitalisation contributed significantly to the analyses of the relationship between the price reduction and health outcomes, and an additional set on climate improved the validity of the time-series analysis of mortality. The different data sets also facilitated analysis of numerous different outcomes. Thirdly, the use of different methods contributed to the versatility and accuracy of the study. For example, the before/after design in the studies on mortality made it possible to examine socioeconomic differentials in changes in mortality, whereas the use of time-series analysis allowed investigation of the impact of the price reduction without the inherent biases related to trends and seasonality. Fourthly, the data sets on mortality attributable to alcohol-related causes are unique in many ways: they are based on both underlying and contributory causes of death, and the death certification has good coverage and reliability. For example, death certificates record alcohol intoxication as a contributory cause more frequently and accurately than in most other countries (Mäkelä et al. 2008; Lahti and Penttilä 2001). The frequent use of medicolegal autopsy in Finland is one of the major factors enabling the proper attribution of alcohol intoxication as a contributory cause of death (Lahti and Penttilä 2001; Lahti and Vuori 2002). Furthermore, data on mortality and hospitalisation cover the total population and it does not suffer from self-report bias and non-response.

Despite these methodological strengths, caution should be exercised in interpreting the results of the study. One big question emerges: to what extent can these results be generalised? As mentioned in Chapter 2 with regard to the Finnish

alcohol policy and its historical background, the high overall alcohol taxation as imposed in Finland, Norway and Sweden is all but unique in the Western world (Anderson and Baumberg 2006). Consequently, it is open to question whether the findings of the current study, at least in terms of the overall increase in alcohol-related mortality and morbidity following the price reduction, can be generalised to countries with a lower degree of restrictiveness in terms of lower taxes and, therefore prices. It is difficult to imagine that a reduction in the price of wine in France, Germany or Italy, for example, where a litre of table wine may cost less than one euro, would have any effect on consumption and, further, on harm. On the other hand, it is probable that the results are broadly applicable to countries that restrict the price and availability of alcohol, which in addition to the Nordic countries include at least most Anglo-Saxon countries such as Ireland, the UK, the USA and Canada (European Commission 2009; Ontario Ministry of Finance 2009; Tax Foundation 2009). Secondly, the assessment of the causal contribution of alcohol on the individual level remains problematic on the basis of causes of death listed on death certificates, which could lead to some overestimations. In some cause-of-death categories the role of alcohol is clear and simple, whereas in others it is more complex and ambiguous. For example, the association between alcohol and suicide is complex (Inskip et al. 1998; Norström et al. 2002; Wilcox et al. 2004). Thirdly, despite the sufficient number of time points in order to conduct analyses accurately in the time-series analyses (Yaffee 2000), a yet longer study period after the price reduction would have yielded more information on the impact. However, excise taxes on alcohol have since been raised three times, which would have made it difficult or impossible to identify further consequences of this price reduction with any degree of accuracy with a longer study period. Furthermore, this relatively short study period made it possible to avoid the time-lag problem, which often affects studies on alcohol consumption and harm (Norström and Skog 2001).

8 CONCLUSION

Epidemiological studies on alcohol provide little and mostly inconsistent evidence of the effects of alcohol pricing on health and crime (Cook and Moore 1993, 2002; Sloan et al. 1994; Dee 1999; Mast et al. 1999; Chaloupka et al. 2002; Farrell et al. 2003; Mohler-Kuo et al. 2004; Ponicki et al. 2007). Research on socioeconomic variation in these effects is even more scant. Furthermore, much of the evidence is based on cross-sectional area-level time-series data (Chaloupka et al. 2002; Trollid and Ponicki 2005). The analyses of the natural experiment presented in this study contribute to this research literature.

Alcohol-related mortality in Finland increased strongly after the price reduction in 2004, but the increase was confined to certain population subgroups: it was largest among persons aged 50–69 years but did not affect the under-35s. A clear gradient was found for education and social class, whereas income did not turn out to be so important as a determinant. The unemployed and early-age pensioners were affected much more than the other groups. The increase was mainly attributable to liver cirrhosis, which is associated with long-term heavy drinking. All in all, the price reduction affected the less privileged in particular. Furthermore, the rate in alcohol-related hospitalisation increased among men under 70 and women aged 50–69, even when trends and seasonal variation were taken into account. The increase was mainly due to an increase in alcohol dependence syndrome and other alcohol-related mental and behavioural disorders.

There were indications that the reduction in alcohol prices may even have had beneficial effects in terms of mortality. People in older age groups appear to have benefited from cheaper alcohol in terms of decreased rates of CVD mortality in particular. Improvements in unobserved risk factors and treatment may have affected the decreased rates of CVD and all-cause mortality to some extent. The effects of the price reduction on all-cause mortality were marginal among younger people. Similarly, the impact was minimal as far as interpersonal violence was concerned.

When it comes to price responsiveness, one of the issues of interest in the field of alcohol research is whether it is different in various user groups. Previous studies have yielded contradictory results: Manning et al. (1995) concluded on the basis of cross-sectional data that heavy (and light) drinkers were much less responsive to prices than moderate drinkers, whereas a more recent study, also based on cross-sectional data, gave evidence of substantial price responsiveness among heavy drinkers with symptoms of alcohol abuse or dependence (Farrell et al. 2003). From the evidence of the current study it seems that heavy drinkers

were very responsive to the price reduction, whereas moderate drinkers were not in adverse terms. The following arguments support these conclusions. First, the large increase in alcohol-related mortality was mainly attributable to chronic causes, particularly liver diseases. As mentioned above, the latency period for liver cirrhosis is long: up to 20 years of excessive drinking (Skog 1980). Consequently, an instant effect on liver cirrhosis implies an effect on heavy drinkers who have severely damaged livers to begin with. Secondly, the increase with regard to acute causes in alcohol-related mortality was marginal: there was no substantial increase in accidental drowning or traffic fatalities, for example. Thirdly, in spite of the increase in alcohol-related mortality, CVD and all-cause mortality decreased in older ages. Low-to-moderate drinking has been linked to the cardioprotective effects of alcohol (Wannamethee and Shaper 1999; Corrao et al. 2000; Agarwal 2002; Rehm et al. 2003; Reynolds et al. 2003; O'Keefe et al. 2007), and thus the price reduction may have fostered moderate consumption and its beneficial effects. Fourthly, the increase in alcohol-related hospitalisation was due to chronic rather than acute causes even if the differences were smaller than in alcohol-related mortality. Finally, there was no significant increase in interpersonal violence in the Helsinki Metropolitan area.

The Finnish health policy has aimed to reduce inequalities in health (Ministry of Social Affairs and Health 2001). One of the main contributions of the current study to the literature on alcohol research is the finding that the increase in alcohol-related mortality was confined to certain disadvantaged population subgroups such as the long-term unemployed and early-age pensioners – subgroups that already before the tax changes included a relatively large proportion of heavy drinkers. In this respect the aims of the health policy have not been realised: the gap between the socially disadvantaged and those better positioned in society has broadened. The government has reacted to the increase in alcohol-related harm by raising taxes three times within a short period after 2007. As a result, alcohol sales decreased in Finland in 2008, whereas imports particularly from Estonia, increased (THL 2009). The repercussions of these recent policy changes remain to be assessed, but it is unlikely that these actions will reduce differentials.

One could conclude on the basis of the results of the present study that price of alcohol should be further raised in order to prevent further harm. However, the use of taxes or prices as a control measure in health policy is problematic in a number of respects. First, there is no doubt about the efficiency of using taxes as a controlling measure in Finland or in similar context – in the short run. However, as a policy it is short-sighted because it does nothing to develop the alcohol culture in a more positive and responsible direction. The century-long restric-

tive alcohol policy has not taught Nordic people to drink responsibly. One could hardly consider the Finnish (or Norwegian or Swedish) policy successful given the prevalent harmful drinking and alcohol-related problems. On the contrary, it is possible that this sort of policy, which was introduced in the spirit of prohibition has rather prevented the Finnish drinking culture from coming more responsible and health-promoting or at least less harmful. It would be worth contemplating whether priority should be given to policy, which would have more profound and thus longer-lasting effects on the drinking culture. The government should strive to remove the causes of social disadvantage instead of focusing on the symptoms. The traditional alcohol policy could thus be called into question. As a matter of fact, it could be replaced, at least to some extent, with a better social policy. Investment in education, for example, would be positive and far-sighted in terms of social policy (and thus of alcohol policy) in that it could provide a set of cognitive resources with the broad potential to influence health (Lynch and Kaplan 2000, 22). This study reported more favourable trends in alcohol-related harm among younger generations, which could be considered as indicators of a new alcohol culture that is less detrimental. The adoption of such a culture could be partly attributable to the generally higher educational level of young people.

Furthermore, a price-oriented alcohol policy could be considered unfair because it is implemented on the terms of problem drinkers. The protection of disadvantaged people in society has frequently been used as justification for using high prices and low availability as a control mechanism. This principle is a well-intentioned and fundamental in a welfare society. However, the price for this short-lived protection may be high, because the more expensive beverages and thus the assumed benefits (of many of which are not easy to measure) associated with moderate alcohol consumption may remain inaccessible to many. In addition, the question remains whether cheaper alcohol would be a totally bad thing even for problem drinkers. To the extent that lower prices would mean more money to purchase food and other basics in order to live a little better which would thus have concrete benefits in human terms.

Finally, it must be emphasised here that drinking too much alcohol is alcohol abuse. As Abraham Lincoln (Ellison 2002) put it, 'it has long been recognised that the problems with alcohol relate not to the use of a bad thing, but to the abuse of a good thing'.

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ORIGINAL PUBLICATIONS

DIFFERENTIAL TRENDS IN ALCOHOL-RELATED MORTALITY: A REGISTER-BASED FOLLOW-UP STUDY IN FINLAND IN 1987–2003

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Abstract — **Aims:** To assess to what extent alcohol-related mortality has changed by age, sex and education in Finland in 1987–2003, a period which saw two periods of economic growth, separated by a severe depression (1991–1995). **Methods:** A register-based follow-up study of all over 15-year-old Finnish men and women. Age, sex and education of the participants were measured at the time of the 1985, 1990, 1995 and 2000 censuses. Follow-up for mortality was for 1987–2003. The outcome measure was alcohol-related mortality, which was defined using information on the underlying and contributory causes of death. **Results:** Among men and women aged 45 years and over, the trends in alcohol-related mortality were associated with economic cycles. Among those aged less than 45 years, alcohol-related mortality decreased from the early 1990s, but intoxication-related accidents and violence still contributed largely to premature mortality. The unfavourable trend for older men resulted from an increase in mortality due to directly alcohol-attributable diseases, alcohol-related diseases of the circulatory system and accidents and violence, and for older women from an increase due to intoxication-related accidents and violence, and alcohol-attributable diseases. Alcohol-related mortality was higher in lower educational groups, and among women the educational gap widened towards the end of the study period. **Conclusions:** This study shows that trends in both economic conditions and per capita consumption of alcohol are not associated with trends in alcohol-related mortality in all population subgroups. In health policy more attention should be paid to divergent trends in gender, age and education specific alcohol-related mortality.

INTRODUCTION

Alcohol is a major determinant of premature death and population subgroup differences in mortality. It has been estimated that alcohol contributes 3.2% to global mortality (Rehm *et al.*, 2004). These types of estimates of alcohol's contribution are most often produced at the national level, and only seldom have differences between other population subgroups than age and sex been shown. The standard procedure for estimating alcohol-related mortality is based on risk ratios and survey data on consumption, and special studies on the contribution of alcohol to injuries and violence (e.g. Mokdad *et al.*, 2004; Murray *et al.*, 2004). This approach may yield a good estimate at the population level, but it is not suitable for studying differences between subgroups of the population. In Finland, the death register provides exceptionally extensive and reliable data on the contribution of alcohol on deaths at the individual level, which enables comparisons between population subgroups.

Previously it has been found, particularly in the Nordic countries, that alcohol-related harms have closely followed changes in per capita alcohol consumption (see Edwards *et al.*, 1994; Norström *et al.*, 2002). On the other hand, it has been reported that in Sweden alcohol-related deaths in 1994–2002 did not increase despite an increase in estimated total alcohol consumption (Holder *et al.*, 2005).

Our observation period, 1987–2003, was characterized by two periods of economic growth (1987–1991 and 1995–2003), separated by a severe depression (1991–1995) that involved large scale unemployment. In the late 1980s, the unemployment rate in Finland was ~5%. However, unemployment in Finland began to increase in late 1990. By the end of 1992, unemployment had climbed to 15%, and by early

1994 a peak figure of about 19% was recorded. Long-term unemployment became more common and redundancy also affected the well-educated labour force and white-collar occupations although to a lesser extent than less educated and blue-collar workers (Martikainen and Valkonen, 1996). The total consumption of alcohol rose in the periods of growth and declined in the course of the depression (Hein and Vuorjoki, 1998; Österberg, 2005). Similarly, deaths from alcohol-related diseases and poisoning increased along with the rapid economic boom of 1987–1991, and decreased during the recession (e.g. Valkonen *et al.*, 2000).

According to previous studies (e.g. Mackenbach *et al.*, 1997; Martikainen *et al.*, 2001; Huisman *et al.*, 2005) there are large differences between sexes and socio-economic groups in premature mortality and life expectancy. In many countries a significant part of these differences have been shown due to alcohol (Mäkelä, 1998; Mäkelä *et al.*, 1997; Mackenbach *et al.*, 1999). According to the global burden of disease estimates alcohol-related deaths are 10 times more frequent among men than among women (Rehm *et al.*, 2004). Socio-economic inequalities in mortality due to alcohol-related diseases among manual labourers were ~90% higher than among non-manual employees in Sweden (Norström and Romelsjö, 1998), whereas in Finland alcohol-related mortality was about three times higher in lower as compared to higher educational and occupational classes (Mäkelä, 1999).

The two most commonly used indicators of socio-economic status are education and occupation based social class. Education and social class are not simply interchangeable, but emphasize different dimensions of social stratification. For the most part, studies on the socio-economic differences in alcohol-related mortality are based on occupational social class (e.g. Mäkelä *et al.*, 1997; Hemström, 2002; Pensola and Martikainen, 2004; Ljung *et al.*, 2005). One of the purposes of this study is to extend the analyses of differential alcohol-related mortality to the examination of education.

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As a measure of socio-economic status education reflects the experiences of youth, a time when educational qualifications are usually obtained. From the point of view of this study it is thus particularly relevant and it is also the time of life when the basis for many habits and behaviours are laid.

The aim of this study is to assess how alcohol-related mortality and its population sub-group differences changed in Finland in 1987–2003 when there were substantial changes in economic conditions, unemployment, and alcohol consumption. However, we do not aim to use information on economic cycles explicitly, e.g. in time series analysis, but use information on changes in unemployment rates as a tool for interpretation. First, we will study trends in alcohol-related mortality by cause and also to assess whether alcohol-related mortality changes with alcohol consumption as can be expected on the basis of literature (Edwards *et al.*, 1994). Second, we will study population differences according to sex, age and education, and their changes in alcohol-related mortality. Alcohol-related mortality is defined using information on the underlying and contributory causes of death. The Finnish death certification practices and cause of death validation result in good quality data on alcohol-related underlying as well as contributory causes of death (Lahti and Penttilä, 2001), which makes the data unique from an international point of view.

DATA AND METHODS

Study population

The data in this study were obtained from Statistics Finland (permission CS-53-483-05). The data are registered data from the 1985, 1990, 1995, and 2000 census which were linked individually to records from the death register for the years 1987–90, 1991–95, 1996–2000, and 2001–03, respectively. The analysis comprises all over 15-year-old Finns, altogether 33.7 million person-years among men and 36.4 million person-years among women.

Alcohol-related mortality

The follow-up for mortality was from 1987 to 2003. In 1987, the Finnish Classification of Diseases 1987 (FCD) was introduced and good quality data on alcohol as a contributory cause of death became available. The basis of the FCD is on the 9th Revision of the International Classification of Diseases (ICD), but it is more detailed due to use of five-digit codes, and some categories have codes different to those of the ICD. In 1996–2003, ICD 10 was used. The Finnish classification also includes some specifications to the international classification in this version.

Alcohol-related deaths were defined as those that have a reference to alcohol in the death certificate's underlying or contributory causes of death. Estimating alcohol-related mortality on the basis of the underlying and contributory causes of death 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 of death more frequently and accurately than in most other countries (Mäkelä, 1998, 2000; Lahti and Penttilä, 2001). Frequent use of medicolegal autopsy is one of the major factors that enables a proper use of alcohol intoxication as a contributory cause of death.

Medicolegal autopsies were carried out in >97% of all accidental and violent deaths among people aged under 75 years in 1987–1993 (Mäkelä, 1998), where as it was >60% of all deaths among people aged <65 years in 1987–2003.

The pool of alcohol-related deaths used here consists of the following two main categories of death: (i) the underlying cause of death is an alcohol-attributable disease (see below) or fatal alcohol poisoning, and (ii) the contributory cause of death is alcohol-attributable disease or alcohol intoxication. The two categories were defined to be mutually exclusive. Below, we use the term 'intoxication-related' for deaths where alcohol intoxication is a contributory cause of death. Alcohol-attributable diseases were: alcohol dependence syndrome (ICD10 code F102; FCD9 code 303), other mental and behavioural disorders due to use of alcohol (F101, F103–109; 291), alcoholic cardiomyopathy (I426; 4255A), alcoholic liver disease (K70; 5710–5713), alcoholic diseases of the pancreas (K860, K8600; 5770D–F, 5771C–D), and additionally some rarely occurring categories (K292, G312, G4051, G621, G721; 3575A, 5353A). We used alcoholic liver cirrhosis instead of all liver cirrhosis, because there does not seem to be any strong tendency to underreport the alcoholic cases in Finland: for example, in 2004 among men aged 15–64 years, 97% of deaths due to liver cirrhosis were classified as alcoholic in the death certificate (Statistics Finland, 2005a).

In the figures for the period 1987–95, we combined the deaths from fatal alcohol poisoning and alcohol dependence syndrome, because a part of the cases defined as alcohol poisoning under ICD9 were defined as alcohol dependence under ICD10.

Measurement of explanatory variables

Sociodemographic data came from censuses in 1985, 1990, 1995, and 2000. These variables included sex, 5 year age groups (15–19, . . . , 90–94, 95+), and education. For education, the following categories were used: basic (10 years or less), intermediate (11–12 years), and high (13+ years).

Methods

Trends in mortality by cause and age were assessed by calculating crude death rates per 100 000 person-years. Age standardization did not affect the results on cause-specific mortality due to the short observation period. When comparing educational groups, the effect of differences in the age distribution was controlled by calculating age-adjusted mortality rates, using combined population of men and women in 1987–2003 as the standard population. Per capita alcohol consumption was calculated as the sum of recorded consumption and estimated unrecorded consumption—based on survey data on the consumption of privately imported, smuggled or home-made beverages (Österberg, 2000)—per inhabitants aged 15 or more (an unpublished table by STAKES 2005). Unemployment rate was used as an indicator for economic trend (Statistics Finland, 1997a, b, 1999a, b, 2005b, c, d).

RESULTS

When alcohol-related mortality comprises not only underlying but also contributory causes of death, the average annual

Table 1. Person-years at risk, number of alcohol-related deaths, and proportion of alcohol-related deaths of all deaths by age, education and period in 1987–2003, Finnish men and women aged 15+

	Men			Women		
	Years at risk %	Alcohol related deaths N	Alcohol-related of all deaths %	Years at risk %	Alcohol-related deaths N	Alcohol-related of all deaths %
Age						
15–29	25.6	3405	35.5	22.7	419	14.4
30–44	29.9	11 002	43.2	26.6	1763	19.4
45–59	25.1	18 409	29.1	23.2	3294	12.8
60+	19.5	12 755	4.1	27.5	2333	0.6
Education						
Basic	47.6	27 369	9.0	49.8	4929	1.4
Intermed	35.6	14 219	21.1	32.6	2113	4.3
High	16.8	3983	10.7	17.6	767	3.1
Period						
1987	5.6	2291	9.6	5.7	338	1.4
1988–89	11.3	4952	10.2	11.5	711	1.5
1990–91	11.4	5409	11.1	11.5	821	1.7
1992–93	11.6	5265	10.8	11.6	874	1.7
1994–95	11.7	5275	11.2	11.7	836	1.7
1996–97	11.9	5009	10.5	11.9	893	1.8
1998–99	12.0	5812	12.0	11.9	1045	2.1
2000–01	12.2	5651	11.9	12.1	1145	2.3
2002–03	12.3	5907	12.4	12.1	1146	2.3
Total	100	45 571	11.2	100	7809	1.9
Person-years (1000s)			33 724			36 382

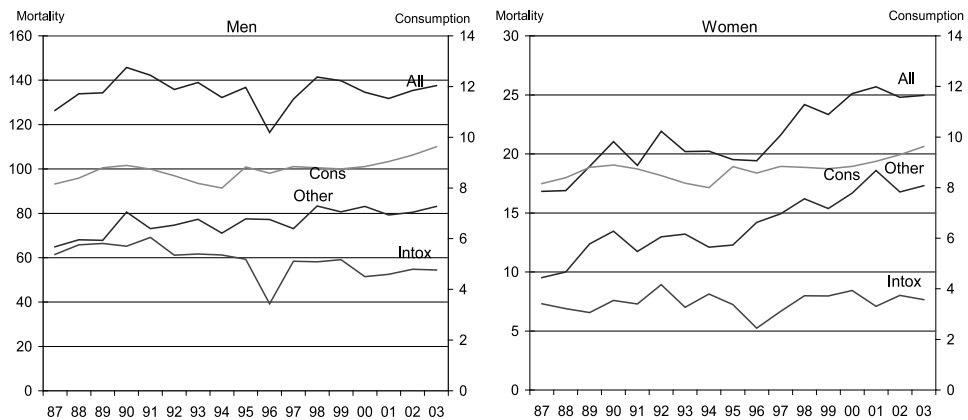


Fig. 1. Alcohol consumption (litres of pure alcohol per capita 15 years and older) and age-adjusted alcohol-related mortality per 100 000 person-years for Finnish men and women in 1987–2003: total alcohol-related mortality, intoxication-related deaths, and other alcohol-related deaths.

number of alcohol-related deaths was 3140 (2681 for men and 459 for women) in the period of study 1987–2003 in Finland, with the peak in 2003 (3581 deaths). Alcohol-related deaths encompassed on average 6.5% (11.2% among men, 1.9% among women) of all deaths (Table 1). Of all alcohol-related deaths 85% were among men.

Trends in alcohol-related mortality by cause and alcohol consumption

Among men, total alcohol-related mortality increased along with the economic boom of the late 1980s, as did alcohol

consumption, and hit record level in 1990 (Fig. 1). During the recession, alcohol-related mortality decreased along with consumption, until the lowest level was observed in 1996. In that year, ICD10 was introduced, which resulted in changes in diagnosing causes of death (Lahti and Vuori, 2002). This may explain the large drop in both total alcohol-related mortality among men and alcohol intoxication as the contributory cause of death in both sexes in 1996. The results for 1996 should thus be interpreted cautiously.

Among men, alcohol-related mortality had another high point in 1998 and was succeeded by a slight decrease and a slight increase at the end of the study period. Alcohol

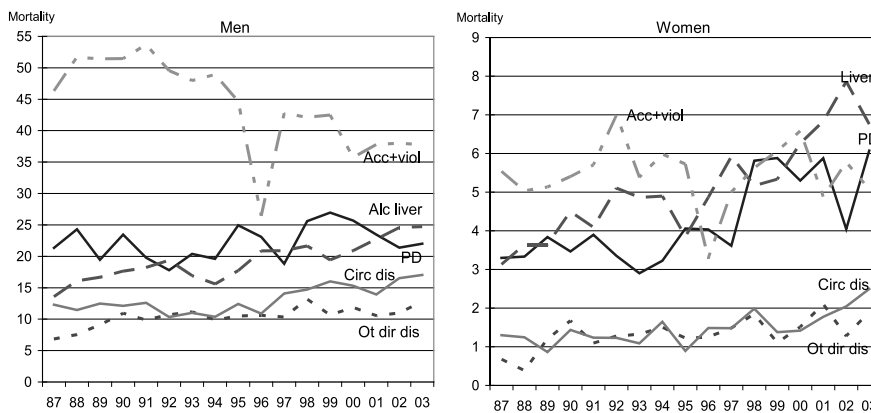


Fig. 2. Alcohol-related mortality per 100 000 person-years by specific causes of deaths, Finnish men and women in 1987–2003. Based on underlying cause: Alc liver (= alcoholic liver diseases), Ot dir dis (= other directly alcohol-attributable diseases), PD (= poisoning and dependence). Based on contributory cause: Acc+viol (= intoxication-related accidents and violence), Circ dis (= diseases of the circulatory system).

consumption was stable in the late 1990s, and did not exceed the record of 1990 until 2001. Intoxication-related deaths followed the changes of total mortality quite closely, although declining slightly, representing an average 44% of all alcohol-related deaths, whereas other alcohol-related causes of death increased slightly.

Among women, the trend of total alcohol-related mortality was to some extent different from men: total alcohol-related mortality increased more clearly during the economic boom of the late 1980s and even more strongly after 1996 until a slight decrease towards the end of the study period, while it was relatively stable during the recession of the early 1990s. In other words, the increase in total consumption was associated with a larger increase in alcohol-related mortality among women than men, and the increase was due to other alcohol-related causes than intoxication. The proportion of intoxication-related deaths was clearly lower among women than among men, i.e. an average of 34% of all alcohol-related deaths. Women's alcohol-related mortality was ~13% of that of men at the beginning of the study period and at the end of the period it was 18%.

Among men the decomposition of total alcohol-related mortality shows that intoxication-related accidental and violent deaths decreased strongly (Fig. 2). The trend for alcoholic liver diseases, as well as for the intoxication-related diseases of the circulatory system roughly followed the development in alcohol consumption. The trends of the other categories did not display strong trends. The relation between two major alcoholic causes of death changed remarkably. At the beginning of the study period there were 3.1 times more deaths from intoxication-related accidents and violence than from alcoholic liver disease, while towards to the end of the study period this ratio had decreased to 1.7.

Among women, almost all specific alcohol-related causes contributed to the increase in alcohol-related mortality. The only exception was intoxication-related accidental and violent deaths.

Trends in alcohol-related mortality by age

Among men, alcohol-related mortality increased between late 1980s and early 1990s in all age-groups after which the trends began to diverge (Fig. 3). For men under 45 years alcohol-related mortality decreased ~30% from the beginning until the end of study period. In older age-groups among whom alcohol-related mortality was at the outset clearly the highest, mortality increased considerably: in the age-group 45–59 years alcohol-related mortality increased by 15% and among those aged 60+ by 31%.

Alcohol-attributable diseases contributed 39% and intoxication-related diseases of circulatory system and intoxication-related accidents and violence >20% each of the increase among men aged 45 and older in 1997–2003 (the period in which ICD10 was used). Among men <45 years the decrease in alcohol-related mortality was above all due to a significant decrease in intoxication-related accidents and violence but also in alcohol-attributable diseases.

This divergence by age was also prominent for women. Alcohol-related mortality was low and stable throughout the study period among the youngest. In the age-group 30–44 years, alcohol-related mortality increased first but remained stable after 1990–1991. In the age-group 45–59 years, alcohol-related mortality increased by 44% and among the oldest by 94%. A closer examination for the period 1997–2003 revealed that among women aged 45 years and older, intoxication-related accidents and violence and alcohol-attributable diseases were responsible for a good 35% each of the increase. Among women aged <45 years, the decrease in alcohol-related mortality was due to a decrease in intoxication-related accidents and violence.

Trends in alcohol-related mortality by education

Among men, age-adjusted alcohol-related mortality was highest among lowest educated men and lowest among highest educated men (Fig. 3). During the study period, alcohol-related mortality of the highest group was stable, but mortality increased in the intermediate and basic educational groups.

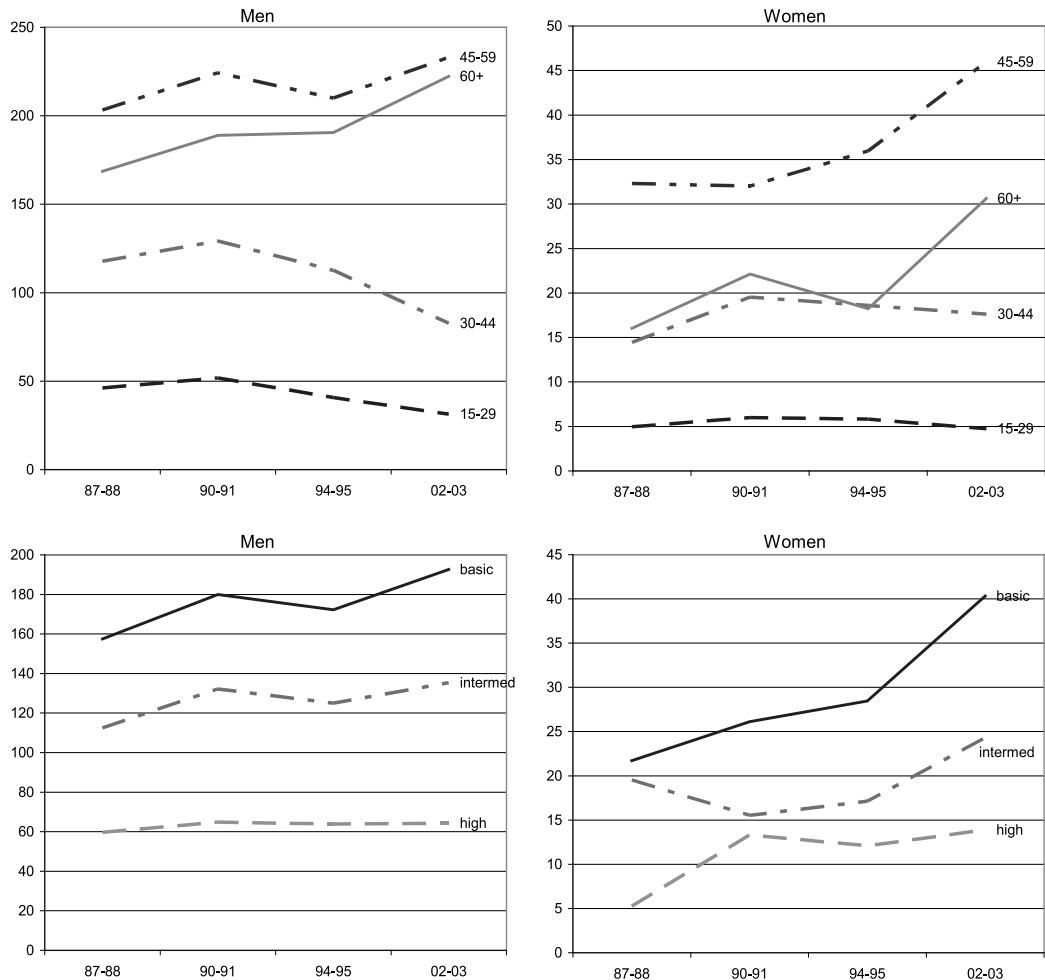


Fig. 3. Alcohol-related mortality per 100 000 by age group (upper figures) and age-adjusted alcohol-related mortality per 100 000 by education (lower figures) in 1987–2003, Finnish men and women aged 15+.

Among women, the difference between basic and intermediate educational classes was, in relative terms, similar to that of men. Also among women absolute mortality differences increased rapidly during the study period. However, relative differences between the basic educated and highly educated women decreased, because of a rapid proportional increase in alcohol-related mortality among the higher educated.

DISCUSSION

The aim of this study was to assess to what extent alcohol-related mortality has changed by age, sex, and education in Finland in 1987–2003, and to assess previous empirical

findings about the association between economic cycles and alcohol-related mortality.

When alcohol as a cause of death comprises not only underlying but also contributory causes an average of slightly over 3000 alcohol-related deaths occurred annually in Finland in the period 1987–2003 which is in agreement with the results that were reported in earlier studies for the period 1987–1995 (Mäkelä, 2000). The proportion of men of all alcohol-related deaths was 85%.

Trends in alcohol-related mortality

Although early studies (e.g. Brenner, 1979) have been criticized on methodological grounds later studies have mainly

validated the counter-cyclical association with mortality (see Ruhm, 2003; Tapia Granados, 2005, and comments in the same issue). The association between economic cycles and alcohol-related mortality may partly be mediated by concurrent changes in alcohol consumption. It has been shown that alcohol consumption increases during economic expansion while the probability of being drinker remains unchanged (Johansson *et al.*, 2006). In numerous studies, it has been reported that alcohol-related harm follows the changes of alcohol consumption at the population level (see e.g. Edwards *et al.*, 1994; Norström *et al.*, 2002).

Among Finnish men, the trend in alcohol-related mortality was associated with economic booms and recessions until mid 1990s, but after that the clear association ended. The economic boom began in 1995, involving decreasing unemployment from 18% in 1994 to <10% in 1999, and continued until the end of the study period, but total alcohol-related mortality mainly decreased after 1998. The trend for different causes of alcohol-related deaths was different. Intoxication-related accidental and violent deaths decreased strongly whereas alcoholic liver diseases and diseases of the circulatory system that had alcohol-related contributory causes increased.

The difference in cause-specific trends is accounted for by diverging trends for those aged 45 years and over and under 45 years. Among men aged 45 years and over, the trend in alcohol-related mortality was mainly increasing even after the mid 1990s. Among them even intoxication-related accidental and violent deaths increased. Together with directly alcohol-attributable diseases and alcohol-related diseases of the circulatory system these causes were responsible for almost 90% of the increase in alcohol-related mortality among men aged 45 years and over. Among young men, the development was favourable due to a decrease in intoxication-related accidents and violence, and alcohol-attributable diseases. Intoxication-related accidents and violence still contributed largely to premature mortality. Unemployment level was highest among those aged <30 years, and it remained relatively high (~15%) even in early 2000s. Additionally, the jobs of young people were more often precarious due to their more frequent fixed-term nature (Sutela *et al.*, 2001). Hence, even though national economy improved in the late 1990s, due to continued labour market and economic disadvantage among the young, alcohol-related mortality did not increase.

Due to lack of accurate consumption data by population subgroups it is somewhat difficult to evaluate to what extent the association between economic cycles and alcohol-related mortality is mediated by alcohol consumption. The distribution of alcohol consumption by sex and age can, however, be assessed through estimations derived from surveys. According to Helakorpi *et al.* (2005), alcohol consumption after the mid 1990s increased among men aged 45 years and over, whereas it was stable or decreased among the younger. Consequently, the association between economic cycles, alcohol consumption, and alcohol-related mortality were consistent among men aged 45 years and older, whereas among younger there was consistency only in the association between alcohol consumption and alcohol-related mortality. In summary, among the older, alcohol consumption increased during an upturn in economy (when unemployment level decreased and earnings increased) and alcohol-related mortality increased, whereas among the young, alcohol consumption

decreased or remained stable (when high unemployment level and precarious jobs restrained expenditure) and alcohol-related mortality decreased.

Among women, alcohol-related mortality during the study period was mainly slowly increasing. Almost all of the specific alcohol-related causes were responsible for the increase. As among men, alcohol-related mortality increased only in the two oldest age-groups. Among them, intoxication-related accidents and violence and alcohol-attributable diseases covered three-quarters of the increase. Like among men, the economic depression affected those aged <30 years the most, and the association between alcohol-related mortality and economic cycles was weaker than among them. Also among women the mediating role of alcohol is supported by a survey. Metso *et al.* (2002) report a slight increase in women's consumption in Finland from the early 1990s to 2000. According to an unpublished table from the Health behaviour and health among the Finnish adult population—surveys in 1982–2005 of the National Public Health Institute by Helakorpi, consumption increased among women aged 45 years and over just as among men, while it decreased or was stable among the younger. Thus like among men, the economic cycles resulted in increased consumption and increased alcohol-related mortality merely among women aged 45 years and older.

Socio-economic differences

The studies on socio-economic differences in alcohol-related mortality are most commonly based on occupational social class. Education lays stress on a different dimension of social stratification than occupation. Education reflects the experiences of early life, a time when educational qualifications are usually obtained and it is also the time when the basis for many attitudes and behaviours are established—as, for example, the relationship to alcohol—that may last till later life. The specific nature of education is knowledge and other non-material resources that are likely to promote healthy lifestyles. Occupational social class, on the other hand, mirrors experiences and exposures in adult life, and its positions indicate status and power, and reflect material conditions related to paid work (e.g. Lahelma *et al.*, 2004). Education is one of the criteria that define social class (e.g. Dahl, 1994) by providing formal qualifications that contribute to the socio-economic status of destination through occupation and income (Lahelma *et al.*, 2004).

This study showed that among both men and women, mortality differences were substantial between educational groups. Alcohol-related mortality was clearly higher in lower educational groups, and among both men and women the absolute gap widened in the study period but the changes in time were somewhat different for men and women. Among men, large differences between educational groups remained during the whole study period, but among women the absolute gap in mortality increased rapidly towards the end of the study period.

The trend in mortality in the lowest educational group followed economic cycles more closely than trends in other educational groups. The unemployment level was clearly highest in the lowest educational group during the whole study period. Those who belong to this group earn less, and during recession they earn even less because of their larger

unemployment rate, and economic availability is entangled with drinking habits. The adverse development among the lowest educated can be mediated by alcohol consumption which increased more rapidly in the lower educational groups at the beginning of the 21st century (Helakorpi *et al.*, 2005). In summary, it thus appears that in the past 20 years economic fluctuations were not associated with alcohol-related mortality among better educated men and women. In the lowest educational group, alcohol consumption and alcohol-related mortality seemed to follow economic cycles: during upturn (when unemployment decreased and earnings increased) consumption and mortality increased, during depression (when unemployment level increased and earnings decreased) consumption and mortality, on the contrary, decreased.

There are certain similarities and differences in alcohol-related mortality when these results are set to comparison with previous findings where the indicator for socio-economic status was occupational social class (see Mäkelä, 2000). The order of the hierarchical educational and occupational categories in alcohol-related mortality is similar. However, alcohol-related mortality among unspecialized blue-collar men is one-third larger than men in the lowest educational group. The very high alcohol-related mortality in the unspecialized blue-collar class could be accounted for by differences in total consumption or in drinking habits. There is no, however, accurate data available on trends in consumption or drinking habits by occupational social class. Furthermore, it is likely that the relative position on the social hierarchy of unspecialized blue-collar workers is lower than that for those with basic education. Unspecialized blue-collar workers constituted about 8% of men aged 50–54 years in the mid 1990s, while the corresponding proportion for the basic educated was 49%. In any case these differences show that education and occupational social class are not interchangeable, and thus support the use of different dimensions of socio-economic status in analyses of differential alcohol-related mortality. The trends are approximately similar in both categorizations until the end of 1995, after which no data exist for occupational social class.

Alcohol-related burden of disease

In the global context, alcohol-related burden of disease is substantial. It is found that 3.2% of global mortality is estimated to be accounted for by alcohol. According to Rehm *et al.* (2004) one-third of the global burden of alcohol-related mortality is due to unintentional injuries, one-fifth to malignant neoplasm, and 13–15% to cardiovascular diseases (percentage includes both beneficial and detrimental effects), intentional injuries and other noncommunicable diseases (i.e. type 2 diabetes and liver cirrhosis) each. According to our study 6.5% of total mortality was related to alcohol in Finland. One-third of the national burden of alcohol-related mortality was due to accidental and violent deaths (i.e. unintentional and intentional injuries), one-fifth to directly alcohol-attributable diseases (including liver cirrhosis), and 11% diseases of the circulatory system (including cardiovascular diseases).

In the estimates by Rehm *et al.* (2004) several diseases related to alcohol could not be accounted for because of lack of population level data on risk ratios and consumption. In this study we could avoid these restrictions by relying on

data on underlying as well as contributory causes of death at the individual level. Additionally, Rehm *et al.*'s study included an estimate of the protective effects of consumption, which was not done in this study. By design then our approach is likely to yield a higher estimate. Therefore, despite the fact that alcohol-related mortality rate in Finland according to this study is two times higher than global rate derived from Rehm *et al.* (2004) it is not possible to make a firm conclusion that alcohol-related mortality is actually higher in Finland than elsewhere. A fuller understanding of the issue would require a more careful comparison that is beyond the scope of this study, but our results do highlight the possibility that the estimates of the contribution of alcohol that are based on more traditional data may be underestimates.

Considerations on data

The data we used is unique in many ways; it is based on both underlying and contributory causes of death, and the death certification has good coverage and reliability (see Lahti and Penttilä, 2001), it covers the total population of Finland and it does not suffer from self-report biases and non-response. However, when interpreting the results of this study the following considerations should be made. The assessment of the causal contribution of alcohol at the individual level remains problematic. Consequently, this may lead in some parts to overestimations of alcohol as a cause of death. In some of the cause of death categories, the role of alcohol is clear and simple, and in the other death categories it is more complex and ambiguous. As an example of the latter outcome measures is suicide. The association between alcohol and suicide is complex. Many studies (see Wilcox *et al.*, 2004) have yielded evidence for an association of suicide with alcohol use disorders. Those with alcohol dependence have a lifetime risk of suicide that is estimated to be 7% (Inskip *et al.*, 1998). According to Norström *et al.* (2002) two main ways in which alcohol might imply an elevated suicide risk have been suggested. One concerns the destructive social consequences of chronic abuse, and the other that intoxication may trigger suicidal impulses. The role of alcohol remains unclear in many cases. The fact that those committing suicide were intoxicated at the moment of suicide does not prove that alcohol causally contributed to the death. When writing alcohol-related contributory causes (e.g. intoxication or alcohol dependence) to the death certificate, the certifying doctor should believe that these causes actually contributed to the death and did not merely co-exist, but it is not possible to assess the true causality in individual cases. Similarly, the fact that someone had a high blood alcohol concentration at the moment of a traffic accident does not prove that the accident would not have happened if the driver had been sober. It is thus possible that our levels of alcohol-related mortality for accident and violent causes are overestimates.

CONCLUSIONS

These results confirm and quantify the important contribution of alcohol use to premature mortality and educational and gender differences in mortality. Among men aged <45 years, the development in alcohol mortality has been very favourable

mainly due to a decrease in intoxication-related accidents and violence. However, it should be remembered that alcohol-related deaths, and particularly intoxication-related accidents and violence, still have a large impact on premature mortality in these age groups. Among older men, the contribution of alcohol on burden of disease has increased. Among women, alcohol-related mortality has remained at a much lower level compared to men, but the rising trend in alcohol-related mortality in older age groups gives reason to be aware of increasing risks.

The association between economic cycles and alcohol-related mortality, possibly mediated by alcohol consumption, still seems to exist, but only in certain population subgroups and due to certain alcohol-related causes of death, namely among those aged 45 years and over and in lower educational groups, and for alcohol-attributable diseases, intoxication-related accidents and violence, and additionally among men due to alcohol-related diseases of the circulatory system. In summary, national economic fluctuations seem to have different effects on different subgroups, partly because economic conditions in all subgroups do not follow to the same extent developments in national economy. Thus harm in alcohol-related mortality in all subgroups does not necessarily follow the trends in per capita consumption.

The results of our study have relevance for attempts at achieving the Finnish health policy goals of reducing alcohol-related harm, and reducing socio-economic and gender differences in mortality (MSAH, 2001). Many of the trends documented in this study are contrary to the set targets. Our results are also an important reference for further follow-up that extends beyond 2004, the year of substantial changes in pricing and availability of alcohol in Finland brought about by EU jurisprudence.

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The impact of a large reduction in the price of alcohol on area differences in interpersonal violence: a natural experiment based on aggregate data

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ABSTRACT

Background: This paper examines the effect of a drastic reduction in the price of alcohol that occurred in Finland in 2004 on interpersonal violence in the Helsinki Metropolitan Area, and how these changes varied at the small-area level.

Methods: This study comprised 86 administrative tracts from the Helsinki Metropolitan Area. Data pertaining to the social structure of the tracts and interpersonal violence were collected from archival sources in the cities and the police in 2002–2005, and analysed using regression analysis.

Results: Interpersonal violence rates did not increase after a large reduction in alcohol prices and an increase in consumption. For domestic violence, the rate even decreased. There was a significant relationship between measures of social disadvantage and interpersonal violence. A low educational level and a high outmigration level were the most salient factors. The differences in impact of the reduction in alcohol prices on interpersonal violence between high-, intermediate- and low-status areas were small.

Conclusions: It would appear that a radical reduction in the price of alcohol and an increase in consumption do not necessarily lead to detrimental consequences in interpersonal violence or to an adverse development in areas of social disadvantage.

The link between alcohol and interpersonal violence is well established.¹ According to a review approximation, in countries in which alcohol is commonly used, over 50% of assailants have been drinking prior to their offence.² However, the consumption of alcohol as such does not necessarily involve an increased risk of interpersonal violence, but binge drinking seems to do so.^{3,4}

The price of alcohol, through taxation in particular, is one of the central tools of alcohol policy. It is widely recognised that it affects the level of alcohol consumption and hence also of alcohol-related problems.⁵ Whether alcohol prices also affect the level of interpersonal violence is a question of interest. Evidence shows that lower alcohol prices are associated with higher consumption and higher rates of interpersonal violence, but population-level studies on this relationship are sparse, and natural experiments for examining this issue have been called for.⁶ One of the few studies in which the focus was directly on the price of alcohol and violent crimes suggested, on the basis of cross-sectional state-level comparisons in the USA, that a 10% increase in beer tax would reduce rapes by 1.3% and robberies by 0.9%, but would

have little impact on homicides and assaults.⁷ On the whole, little is known about the effects of price reductions, as opposed to price increases, on interpersonal violence.

Several studies have focused on the aggregate-level association between alcohol consumption and violence. In Sweden, a relationship between alcohol consumption and homicide and assault rates in 1870–1984⁸ was found and, in Finland, alcohol consumption was one of the four indicators that explained an increase in assault rates in 1950–2000.⁹ In Norway, an increase in alcohol consumption of one litre per capita predicted an increase of 8% in the violence rate in 1911–2003¹⁰ and, in five European countries (including Finland) and in Ontario, Canada, homicide rates were positively related to alcohol sales or consumption.^{11,12}

Alcohol use and interpersonal violence are both individual acts, and they are influenced by individual characteristics. However, the environment in which people live also matters. A number of studies have provided evidence that neighbourhood characteristics are related to interpersonal violence,^{13,14} whereas there is little direct evidence about the association between neighbourhoods and heavy or binge drinking. Indirect evidence in terms of alcohol-related problems, however, shows that this relationship exists.¹⁵ Explanations for the rates of interpersonal violence observed in community settings usually focus on one of two neighbourhood features: the characteristics of the people living in the neighbourhoods or the integral characteristics of the places in which they live.¹⁶ The former include factors such as area-level poverty, family structure and residential mobility,¹⁷ and the latter factors such as the proportion of abandoned housing and the level of retail activity.¹⁸ The use of population- rather than place-related characteristics works better in the European welfare setting where cities are better equipped to control their development by means of town planning than in the US context.¹⁹

However, the underlying mechanisms linking neighbourhood characteristics and various outcomes have remained uncharted for the most part. Neighbourhood differences are not “naturally” determined, but rather result from social and economic processes influenced by specific policies.²⁰ It has been suggested that spatial embeddedness, internal structural characteristics and social organisational processes are all important in understanding neighbourhood-level variations in rates of violence.¹¹ A review examining social processes

related to problem behaviour and health-related outcomes covering over 40 studies emphasised neighbourhood ties, social control, mutual trust, institutional resources, disorder and routine activity patterns more than traditional characteristics such as poverty and residential instability.¹⁵

THE FINNISH AND THE HELSINKI METROPOLITAN AREA CONTEXT

Pricing and availability have been two major tools of the authorities in controlling alcohol consumption in the Nordic countries.⁵ In Finland, the state alcohol off-premise retail company has a monopoly on alcoholic beverages other than those with a low alcohol content, such as beer and cider with an alcohol content of less than 4.7% by volume. Alcoholic beverages have been heavily taxed, and the amount of alcohol that people are allowed to import from abroad has been strictly limited.

The year 2004 was a milestone in Finnish alcohol policy. On 1 January, it became legal to import practically unlimited amounts of alcoholic beverages for one's own consumption due to the deregulation of import quotas within the European Union. On 1 March, taxes on alcohol were reduced by an average of 33%. The off-premise retail price of spirits went down by an average of 36%, wines by 3% and other alcoholic beverages by between 13 and 28%. The reason for the tax cuts was that Estonia was to join the European Union on 1 May, after which the deregulation of import quotas would also apply to alcohol imports from Estonia. This was expected to lead to a considerable increase in imports because of the proximity of the two countries and the significantly lower price of alcohol in Estonia. Tax cuts were aimed at restraining these imports. A unique natural experiment—such as that carried out in Finland in 2004, involving a radical reduction in the full price of alcohol (including both retail price and indirect costs)—has thus opened up the possibility of assessing the causality between the price of alcohol and interpersonal violence.

The increase in total alcohol consumption (the sum of recorded and estimated unrecorded consumption) in Finland was estimated to be 9.6% in 2004 and a further 1.9% in 2005.²¹ There are no data available on the total consumption in the Helsinki Metropolitan Area, but recorded sales increased by 5.4% (7.3% at the national level) in 2005 compared with 2003 (National Product Control Agency for Welfare and Health, unpublished table). If the ratio between recorded sales and unrecorded consumption was similar in the Helsinki Metropolitan Area to that in the whole country, this would translate to an increase of 8.7% in total consumption. Owing to its proximity to the Estonian capital (which means that imports may have increased more than average) and its roles as a centre of nightlife and tourism (which means that part of the changes in on-premise sales may be attributable to changes in the consumption of visitors to Helsinki), sales statistics do not necessarily reflect consumption by the metropolitan inhabitants, or its changes, reliably. Even if there is uncertainty in the Helsinki Metropolitan Area about the extent of the increase in alcohol consumption—the central intervening variable between alcohol price and violence—it is of great interest to examine what effects the price change had on interpersonal violence and also whether the effects varied by type of area.

Helsinki is the capital of Finland and had a population of 559 000 on 1 January 2005. Espoo and Vantaa are neighbouring cities of Helsinki that had 227 000 and 185 000 inhabitants respectively. Helsinki, Espoo, Vantaa and Kauniainen constitute the Helsinki Metropolitan Area with a population of 972 000,

which was 18.6% of the total population of Finland in 2005. Finland is characterised by an ethnically homogeneous population and a low rate of economic inequality.²² An expressed objective of local housing and city planning policies in Helsinki is to prevent and reduce socioeconomic segregation among residential areas.²³ As a result of the national context and local policies, the Helsinki Metropolitan Area does not feature areas that could be described as “truly disadvantaged” or “hypersegregated”.^{24–25} A previous study has, however, reported differences in mortality between areas in the Helsinki Metropolitan Area.²⁶

The main purpose of this study was to assess to what extent the changes in the full price of alcohol affected the change in rates of interpersonal violence and two indicators of less severe disorderly conduct (custody due to alcohol intoxication and disturbing behaviour), and how these changes varied at the small-area level in the Helsinki Metropolitan Area. We had three research questions, the first of which was more general in nature. We examined whether:

1. measures of area-level social disadvantage were associated with interpersonal violence rates
2. the reduction in the price of alcohol was associated with interpersonal violence
3. the effects of the price reduction on interpersonal violence were associated with measures of area-level social disadvantage.

METHODS

Study sites and period

The sample for this study comprised 86 small areas (tracts) from the Helsinki Metropolitan Area, of which 33 belonged to Helsinki, 27 to Espoo and 26 to Vantaa and a small municipality within Espoo (Kauniainen). The tracts had populations ranging from 486 to 36 522, with a mean of 10 981, and were based on the administrative area division of the municipalities, which is used for policy purposes. We obtained data on the socio-demographic characteristics from administrative databases.^{27–28}

The study period extended from the beginning of 2002 until the end of 2005. The total period was divided into two subperiods: (1) before the change in the pricing of alcohol (2002–2003) and (2) after the change (2004–2005). We thus had two symmetric periods to investigate in our analysis and could avoid potential bias due to the seasonal variation in interpersonal violence. A before–after comparison is more appropriate here than an interrupted time series analysis because time series analysis needs a clearly longer study period than is available in this study. Time series analysis requires at least 100 observations in order to detect a moderate effect of an intervention;²⁹ but we only have access to 48 measurement points in our data.

Interpersonal violence and disorderly conduct

Our main outcome measure was interpersonal violence, but we also included two less severe indicators of disorderly conduct in our analyses. Data on interpersonal violence in the administrative areas were obtained from the Helsinki Police Department. The size of these areas was smaller on average in the Helsinki Metropolitan Area than in the municipal administrative areas but, by merging them, we obtained corresponding data for the purpose of analysis and also achieved correspondence in terms of area units. The data on interpersonal violence and disorderly conduct consisted of the number of various acts by tract and month in the years 2002–2005.

The data on interpersonal violence and disorderly conduct were obtained from three sources in the Helsinki Police Department. First, we considered crimes recorded by the police that were specified as offences against the Penal Code. The following outcome measures from this source were included in the analysis: assault and battery, including the subgroups assault in private homes and in public places, robbery and extortion, disturbance of domestic peace and rape. The second source of data comprised emergency call-outs related to domestic violence, disturbing behaviour and vandalism, and emergency responses in total. Approximately 80% of these cases were reported to the police emergency centre by the public. Third, we included a particular outcome measure: taken into custody due to alcohol intoxication was recorded as a police task but not as a crime.

Area characteristics

The choice of seven sociostructural area characteristics was based on prior knowledge obtained from previous studies on area differences in association between alcohol and crimes,³⁰ as well as from studies on the effect of neighbourhood characteristics on mortality in the Helsinki Metropolitan Area.²⁶ Six of the area characteristics were measures of social disadvantage: for those aged 15 years and over, we measured the proportion of people with basic education, mean personal income (€1000), manual class membership and unemployment (the last two being characteristics of the labour force), the proportion of single-parent families and the proportion of homes that were not owner-occupied. We also used a measure of residential instability (outmigration). All the area characteristics were measured in 2002–2003, except for manual class membership in 2000. All the area characteristics are relatively stable over time, and the ranking of neighbourhoods on these variables has changed very little. The area characteristic “proportion of 15–29 year olds” was left out because of its very minor effect.

Analyses

The frequencies and rates per risk population per year and per 1000 persons were calculated for all the different categories of interpersonal violence and disorderly conduct. Multiple linear regression analyses were performed using Stata, version 8, in order to evaluate the association between the area characteristics and assault, assault in private homes, domestic violence and custody due to alcohol intoxication. The choice of these outcome measures was based on their importance or their observed associations in the preliminary analyses. In order to take account of the correlatedness of the data over time, we used the “cluster” option, which affects the estimated standard errors and variance-covariance matrix of the estimators, but not the estimated coefficients.³¹ Weighting was used in linear regression analysis in order to take account of differences in the size of small-area populations.

We used two different models in assessing the association between area-level social disadvantage and interpersonal violence and disorderly conduct. First, we conducted the analyses separately for each of the area characteristics, chosen on the basis of earlier studies, which established their relevance in theoretical terms.^{26,27} Second, we used a backward selection regression procedure, with an incremental removal significance criterion of $p > 0.05$, to determine the best model with suitable independent variables.

We excluded the centre of Helsinki from the analyses of assault and custody due to intoxication because of its role as the

centre of amusements and nightlife in the Helsinki Metropolitan Area. If we had not done so, it would have biased the estimates of assault and custody rates in areas of concentrated disadvantage, as the centre of Helsinki represents a “good” tract with its low unemployment rate and high mean income, among other things.

We calculated rates for three indicators of interpersonal violence and for custody due to alcohol intoxication and changes in them over time according to the high, intermediate and low categories in the area characteristics. The high and low categories each covered 20% of the population aged 15 years and over and the intermediate category 60%. The change over time was tested by means of linear regression focusing on the interaction between time and area characteristics. Time was used here as a categorical variable and area characteristics as a continuous variable with values of 1 for the high-status category, 2 for the intermediate and 3 for the lowest status category.

RESULTS

The effect of the price reduction on interpersonal violence and disorderly conduct

Table 1 gives the descriptive statistics of the area characteristics used in the analysis.

The change in rates for interpersonal violence and disorderly conduct from 2002–2003 to 2004–2005 varied according to the type of crime or recorded task (table 2). Custody due to alcohol intoxication, assault and battery together with its subgroups (assault in private homes and public places and disturbing behaviour) increased by 3–5%, whereas domestic violence decreased by 7% and robbery, violating domestic peace and rape by over 10%. Emergency call-outs in total remained at the level that prevailed before the price reduction. However, all the estimates of change, except in the case of violating domestic peace, were statistically insignificant.

Area characteristics and interpersonal violence

Four dependent variables were chosen for further analyses on the basis of their importance or their observed associations in the preliminary analyses. The effect of area characteristics on three types of interpersonal violence and custody due to intoxication before the price reduction was examined by fitting two different regression models (table 3). When all the measures of social disadvantage were added into the model one by one (model 1), all except outmigration and manual class had a significant relationship to all four outcome variables. However, when all measures of social disadvantage were put in simultaneously, the effects overlapped (model 2). When we controlled for other factors that remained significant in the model, we found that the assault rate was higher in tracts with a higher proportion of people with basic education and

Table 1 Descriptive data on area characteristics in the Helsinki Metropolitan Area in 2002–2003*

	Mean	SD	Minimum	Maximum
Basic education (%)	34.1	7.4	12.6	57.7
Manual class (%)	25.9	8.7	9.9	51.6
Unemployment (%)	7.2	2.8	2.1	17.2
Income (€1000)	26.7	7.2	13.3	56.2
Single-parent family (%)	20.5	11.4	4.1	49.3
Not owning a home (%)	47.8	16.6	15.8	92.5
Outmigration (%)	18.2	6.3	5.6	48.8

*Manual class in 2000.

Table 2 Distribution of delinquency and delinquency rates* before (2002–2003) and after (2004–2005) the change, Helsinki Metropolitan Area

	Before		After		Change		p Value†
	n	Rate	n	Rate	Rate	%	
Custody	43699	27.01	45040	27.83	0.83	3.1	0.567
<i>Crimes</i>							
Assault	10246	6.33	10704	6.61	0.28	4.5	0.161
Private homes	3061	1.89	3182	1.97	0.07	4.0	0.447
Public places	5426	3.35	5637	3.48	0.13	3.9	0.548
Robbery	1054	0.65	923	0.57	-0.08	-12.4	0.054
Violating domestic peace	1355	0.84	1171	0.72	-0.11	-13.6	0.001
Rape	139	0.09	114	0.07	-0.02	-18.0	0.180
<i>Emergency call-outs</i>							
Domestic violence	7838	4.84	7306	4.51	-0.33	-6.8	0.069
Disturbing behaviour	45428	28.07	46863	28.96	0.89	3.2	0.421
Total emergency call-outs	460526	284.60	462345	285.72	1.12	0.4	0.921
Population aged 15+	809086		809086				

*Amount of delinquency/risk population/year × 1000.

†p value for the change in delinquency rate from linear regression.

outmigration, while other factors lost their significance. The controlled model for assault in private homes revealed a higher crime rate in tracts with a higher proportion of single-parent families. As far as domestic violence was concerned, the final model indicated a higher rate in tracts with a high proportion of manual class membership and unemployment. The controlled model for custody due to intoxication revealed a higher rate in tracts with people of a lower educational level and a higher outmigration rate.

The price reduction, area characteristics and interpersonal violence

Table 4 confirms the findings shown in table 3 that rates of all types of interpersonal violence were higher in low-status than in high-status tracts before the changes in the pricing of alcohol. Different indicators of interpersonal violence changed differently, depending on the area characteristics, from 2002–2003 to 2004–2005. We observed very little change in incidences of assault in high-status tracts. Interaction analysis showed that variations in change according to area measures were proportionally significant only in the manual class: the assault rate increased more in tracts with a high proportion of inhabitants of that status. The difference in the direction of change was similar for those with a low educational level, although not statistically significant.

Changes in incidences of assault in private homes did not differ by area characteristics. As far as domestic violence was concerned, there was some indication that the change was more favourable in tracts with higher unemployment and lower income. A similar pattern also emerged for other area characteristics (except outmigration), ie the rate of domestic violence decreased most in lower status tracts, but the change was not statistically significant.

The change in custody due to alcohol intoxication was the most adverse in tracts with high outmigration, and was close to zero in those with low outmigration. The manual class and home ownership variables showed the same pattern, but the association between the change and the categories of area characteristics was not statistically significant. That many of the seemingly sizeable differences remained non-significant was at least partly due to the distribution of the categories: intermediate categories covered 60% of the population.

DISCUSSION

This study showed that a drastic reduction in the cost of alcohol did not increase the rates of interpersonal violence and disorderly conduct in the Helsinki Metropolitan Area. Several studies that have examined the effects of price increases⁶ have uniformly suggested that changes in the price of alcohol have consequences in terms of alcohol-related harm, such as interpersonal violence. A reduction in the price of foreign-produced spirits in Switzerland has been reported to have been followed by increased self-reported alcohol-related problems (the effects on violence were not examined).³² Previous studies⁵ have raised expectations that the association between price and interpersonal violence is two-way, ie if an increase in the price of alcohol is observed to have been followed by a lower level of interpersonal violence, a decrease would result in higher levels. According to the findings of this study, this does not necessarily seem to be true.

It remains unclear why the rates of interpersonal violence did not increase in the Helsinki Metropolitan Area after a rapid change in pricing and availability and an increase in consumption. We have identified two possible factors that may explain this. First, in the Finnish context, the association between alcohol consumption and violence is not as strong as was expected on the basis of earlier studies. This factor is supported by a recent study based on time series analysis conducted at the national level, which did not demonstrate an increase in violent crimes in 2004.³³ Second, there is some evidence that the increase in alcohol consumption that followed the price reduction was especially strong among heavy drinkers. For example, increase in custody due to intoxication resulted from an increased number of arrests among “regulars”, while the number of arrests among the more occasional arrestees did not increase.³⁴ In addition, after the price reduction, alcohol-related mortality increased particularly strongly in the groups that had already experienced the highest levels of alcohol-related mortality, ie males living alone, without a job and those on disability pensions.³⁵ The increase was also mainly due to alcoholic liver cirrhosis.³⁶ It may thus be that the possible increase in the case of interpersonal violence due to increased consumption is confined to a relatively limited group of heavy drinkers. Further studies should try to identify these population subgroups.

Table 3 The effects of area characteristics on interpersonal violence and custody due to alcohol intoxication before the change (2002–2003) in the Helsinki Metropolitan Area (the centre of Helsinki excluded except for assault in private homes and domestic violence), linear regression

	Model 1				Model 2				$R^2 = 0.38$
	Coefficient	Robust SE	Beta	Probability	Coefficient	Robust SE	Beta	Probability	
<i>Assault</i>									
Basic education (%)	2.25	0.46	0.33	0.000	2.62	0.53	0.38	0.000	
Manual class (%)	2.25	0.36	0.38	0.000					
Unemployment	7.72	0.94	0.43	0.000					
Income	-2.63	0.66	-0.37	0.000					
Single-parent family (%)	1.66	0.24	0.37	0.000	3.10	0.93	0.38	0.001	
Not owning a home (%)	1.11	0.25	0.36	0.000					
Outmigration	2.67	1.30	0.33	0.043					
	Model 1				Model 2				$R^2 = 0.42$
<i>Assault in private homes</i>									
Basic education (%)	0.52	0.25	0.28	0.038					
Manual class (%)	0.29	0.22	0.18	0.190					
Unemployment	2.68	0.58	0.55	0.000					
Income	-0.49	0.20	-0.26	0.017					
Single-parent family (%)	0.78	0.10	0.65	0.000	0.78	0.10	0.65	0.000	
Not owning a home (%)	0.29	0.09	0.35	0.003					
Outmigration	0.28	0.24	0.13	0.238					
	Model 1				Model 2				$R^2 = 0.69$
<i>Domestic violence</i>									
Basic education (%)	2.83	0.46	0.73	0.000					
Manual class (%)	2.52	0.36	0.77	0.000	1.12	0.25	0.34	0.000	
Unemployment	8.73	1.01	0.86	0.000	6.40	0.99	0.63	0.000	
Income	-2.40	0.51	-0.60	0.000					
Single-parent family (%)	1.38	0.30	0.55	0.000					
Not owning a home (%)	0.89	0.22	0.52	0.000					
Outmigration	1.05	0.69	0.23	0.134					
	Model 1				Model 2				$R^2 = 0.18$
<i>Custody</i>									
Basic education (%)	7.84	3.38	0.18	0.023	10.15	4.49	0.23	0.027	
Manual class (%)	9.13	2.89	0.24	0.002					
Unemployment	28.21	5.10	0.25	0.000					
Income	-12.35	3.78	-0.27	0.002					
Single-parent family (%)	6.11	1.19	0.21	0.000	19.11	8.64	0.37	0.030	
Not owning a home (%)	6.02	1.90	0.31	0.002					
Outmigration	17.48	9.45	0.34	0.068					

Area characteristics were measured as continuous in the linear regression.

Model 1. Bivariate model. Each of the area characteristics was analysed separately.

Model 2. Multivariate model. All significant area characteristics are included in the analysis.

This study provides support for previous work^{16, 37} demonstrating a significant relationship between measures of area-level social disadvantage and interpersonal violence. It extends these findings by specifying and quantifying these relationships. The association was found to be different with regard to different types of interpersonal violence and for different area characteristics: a low educational level and a high outmigration level were the most salient. The role of education in creating inequality and contributing to other sociostructural characteristics is well established at the individual level.^{38, 39} Moreover, a low educational level among individuals is reported to have an association with other types of harm, such as alcohol-related mortality.⁴⁰ The findings of this study correspond with previous findings on the association between residential instability and interpersonal violence.¹⁵ It is also noteworthy that rates of assault in private homes were strongly related to a high proportion of single-parent families, and rates of domestic violence to a high proportion of unemployment and manual class membership when other characteristics were held constant.

Most previous studies on the association between interpersonal violence and disadvantage have been conducted in the USA

and in metropolitan contexts in which disadvantage could be assumed to be concentrated to a much larger extent than in a Nordic welfare setting, where the state intervenes more in market processes in order to reduce poverty⁴¹. Finland is one of the most egalitarian societies measured by income inequality.⁴² Accordingly, the current study extends the relationship between measures of area-level disadvantage and interpersonal violence by providing support for the observation that this association exists even in a Nordic welfare state.

Overall, the differences in impact of the reduction in alcohol prices on interpersonal violence and disorderly conduct between high-, intermediate- and low-status areas were small. An adverse development in assault occurred in low-status tracts according to manual class membership and educational level, and in custody due to intoxication according to outmigration and manual class. In terms of domestic violence, however, the results were encouraging in two ways: the rate decreased on the whole, and most in the low-status tracts in which it was highest, although the association between time and area characteristics was statistically significant only in the case of unemployment and income. This favourable trend could be

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Table 4 Annual delinquency rates (per 1000) before and after the change in the pricing of alcohol, and linear regression interaction (bivariate models) for area characteristics and time in the Helsinki Metropolitan Area (the centre of Helsinki excluded except for assault in private homes and domestic violence)

	Assault		Assault in private homes		Domestic violence		Custody due to intoxication	
	Before (change %)	p Value	Before (change %)	p Value	Before (change %)	p Value	Before (change %)	p Value
Basic education (%)		0.082		0.516		0.123		0.823
Low	3.1 (-3.9)		1.3 (5.1)		2.3 (-6.0)		15.6 (-0.7)	
Intermediate	5.5 (3.4)		2.1 (2.2)		4.9 (-2.4)		20.7 (10.8)	
High	8.7 (19.3)		2.2 (7.8)		7.8 (-14.5)		38.2 (-0.8)	
Manual class (%)		0.048		0.753		0.383		0.170
Low	2.5 (0.9)		1.3 (-0.8)		2.1 (-1.1)		11.3 (-6.6)	
Intermediate	5.8 (2.0)		2.1 (2.1)		4.9 (-5.5)		22.9 (8.6)	
High	8.0 (20.6)		2.2 (11.7)		8.3 (-10.3)		35.2 (2.7)	
Unemployment		0.248		0.951		0.016		0.994
Low	2.3 (0.3)		0.9 (6.6)		2.3 (-0.3)		9.7 (2.6)	
Intermediate	5.8 (6.8)		2.0 (3.2)		4.7 (-1.7)		24.7 (7.6)	
High	8.2 (10.2)		2.8 (4.9)		8.8 (-16.6)		30.6 (1.0)	
Income		0.248		0.855		0.022		0.908
High	2.8 (0.2)		1.3 (1.7)		2.3 (-3.2)		11.5 (-3.4)	
Intermediate	6.0 (8.0)		2.0 (4.2)		4.9 (-1.7)		25.7 (9.2)	
Low	7.4 (8.2)		2.7 (4.4)		8.4 (-17.1)		26.4 (-1.3)	
Single-parent family (%)		0.740		0.646		0.150		0.459
Low	2.6 (-0.8)		0.8 (12.3)		2.2 (1.4)		9.9 (3.1)	
Intermediate	5.5 (15.5)		1.9 (5.8)		5.2 (-5.2)		23.3 (5.3)	
High	8.7 (-6.2)		3.4 (-1.3)		7.4 (-12.7)		34.2 (6.4)	
Not owning a home (%)		0.154		0.796		0.537		0.151
Low	2.7 (-2.5)		1.1 (5.5)		2.5 (-2.1)		9.5 (1.4)	
Intermediate	5.6 (11.6)		1.8 (8.2)		5.1 (-7.6)		23.7 (3.7)	
High	8.3 (1.7)		3.3 (-4.2)		7.5 (-6.6)		33.7 (10.1)	
Outmigration		0.431		0.607		0.070		0.044
Low	3.0 (-1.6)		1.2 (5.9)		3.3 (-8.3)		10.3 (-0.5)	
Intermediate	5.4 (8.2)		2.2 (4.7)		5.9 (-7.9)		19.6 (2.4)	
High	8.6 (8.7)		2.0 (0.3)		4.1 (-0.9)		45.6 (10.6)	

Area characteristics were measured as continuous variables in the linear regression.

interpreted as a result of increased public intolerance of domestic violence, which has catalysed actions within society, in terms of legislation for instance, in order to reduce it.

Two issues need to be considered in the interpretation of our findings. First, the data on interpersonal violence that we used included only crimes about which the police had information, ie there was a report of an offence. Many criminal events fail to enter the records, however: they may not be known to the police at all, or the police may not record them as crimes for a variety of reasons.⁴⁵ However, the coverage of police records is high as regards severe crimes, such as the acts of interpersonal violence included in our analyses. Second, there are factors that affect the compilation of statistics on the number of crimes, such as changes in police control and in legislation. Our study period was too short to witness any remarkable changes in the readiness to report crimes, however, and there were no significant changes in legislation or police control concerning the types of crime used in our study during the study period. The frequent variation in police control was one reason for excluding drunken driving in our analyses, for instance.

Conclusions

Although previous studies indicate that the price of alcohol is a major tool affecting alcohol-related problems, this was not found in the case of interpersonal violence when alcohol prices decreased and consumption increased. Furthermore, the differences in the change in interpersonal violence between high- and low-status tracts were small. Consequently, it would

appear that a drastic reduction in the price of alcohol does not necessarily lead to detrimental consequences in interpersonal violence or to an adverse development in areas of social disadvantage. However, additional studies on other types of harm, such as mortality and morbidity, are needed in order to reach a fuller understanding of the impact of the availability of cheaper alcohol.

What is already known on this subject

- ▶ An increase in the price of alcohol is associated with a decrease in the rate of alcohol-related problems
- ▶ Little is known about the effects of price reductions of alcohol on interpersonal violence, and how this varies in urban areas with different characteristics

What this study adds

- ▶ We use a natural experimental design in Finland that occurred when taxes on alcohol were reduced and the total price of alcohol fell greatly in 2004
- ▶ The results show that a large reduction in alcohol prices and an increase in consumption do not necessarily lead to detrimental consequences in interpersonal violence or to an adverse development in areas of social disadvantage

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Original Contribution

Changes in Alcohol-Related Mortality and its Socioeconomic Differences After a Large Reduction in Alcohol Prices: A Natural Experiment Based on Register Data

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The authors examined the effect of a large reduction in the price of alcohol in Finland in 2004 on alcohol-related mortality by age and socioeconomic group. For this register-based study of Finns aged ≥ 15 years, data on independent variables were extracted from the employment statistics of Statistics Finland. Mortality follow-up was carried out for 2001–2003 (before the price reduction) and 2004–2005 (after). Alcohol-related causes were defined using both underlying and contributory causes of death. Alcohol-related mortality increased by 16% among men and by 31% among women; 82% of the increase was due to chronic causes, particularly liver diseases. The increase in absolute terms was largest among men aged 55–59 years and women aged 50–54 years. Among persons aged 30–59 years, it was biggest among the unemployed or early-age pensioners and those with low education, social class, or income. The relative differences in change between the education and social class subgroups were small. The employed and persons aged < 35 years did not suffer from increased alcohol-related mortality during the 2 years after the change. These results imply that a large reduction in the price of alcohol led to substantial increases in alcohol-related mortality, particularly among the less privileged, and in chronic diseases associated with heavy drinking.

alcohol drinking; alcohol-related disorders; commerce; economics; education; income; social class; socioeconomic factors

Abbreviation: ICD-10, *International Statistical Classification of Diseases and Related Health Problems*, Tenth Revision.

Editor's note: An invited commentary on this article appears on page 1126.

The price of alcohol is evidently associated with alcohol consumption and alcohol-related problems on the population level: An increase in price tends to decrease consumption and related problems (1, 2). Much of the evidence on this issue is based on time-series analyses; however, such analyses do not yield information on the differential effects of a change in price on population subgroups. The need for studies based on natural experiments, which take account of both change over time and differences between subpopulations, is obvious (1). Here we report what happened to alcohol-related mortality and its socioeconomic differences in Finland after a large reduction in the price of alcohol.

Large socioeconomic differences in alcohol-related and all-cause mortality are well documented (3–5). In the United Kingdom, occupational social class has appeared to be a risk factor for alcohol-related mortality and hospitalizations, particularly in men (6, 7), while in Nordic studies, investigators have reported rates that are 1.9–3.2 times higher among male manual workers than among nonmanual employees (4, 8, 9). Additionally, in a few recent studies, researchers have reported increased inequality over time according to social class and education (10, 11). US studies on socioeconomic differences in alcohol-related mortality are sparse, but a few have found that socioeconomic differences in alcohol-related motor vehicle crashes are marked by education, income, and language group (12, 13).

Differences in alcohol consumption contribute to the socioeconomic differences in alcohol-related mortality and

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other alcohol-related problems. Consumption tends to be more adverse, in terms of the prevalence of heavy drinking and drinking patterns, in lower socioeconomic groups, but cultural and sex differences exist (14–16). However, there is little evidence on the issue of potential differential responsiveness to price. Youths and young adults have been shown to be generally more responsive to price increases than older adults (1). Correspondingly, it could be hypothesized that other groups with fewer means, such as lower socioeconomic groups, would also be more affected by the price of alcohol. Age is also, in other respects, an essential factor in the relation between socioeconomic indicators, alcohol consumption, and alcohol-related mortality. From the life-course perspective, the most crucial dividing lines can be drawn between persons who have not fully established their socioeconomic status (i.e., youths and young adults) and older persons and between persons of working age and those in retirement.

Alcohol-related mortality can be divided into acute and chronic causes. The former include deaths related to alcohol intoxication and the latter deaths due to the chronic effects of long-term heavy drinking, liver cirrhosis being the most notable example. Chronic causes have also been found to be quickly responsive to a sudden decrease in consumption (17). We expected our results on socioeconomic differences in acute versus chronic causes to be informative in terms of interpreting the process of change.

The year 2004 was a milestone in Finnish alcohol policy. On January 1, it became legal to import from other European Union countries virtually unlimited amounts of alcoholic beverages for one's own use, because of the deregulation of import quotas within the European Union. On March 1, taxes on alcohol were reduced by an average of 33%. The off-premise retail price of spirits went down by an average of 36%, the price of wine by 3%, the price of beer by 13%, and the price of other alcoholic beverages by 17%–25% (18). The reason for the tax cuts was that Estonia joined the European Union on May 1; this had a great impact on the Finnish alcohol market because of the proximity of the two countries and the significantly lower price of alcohol in Estonia.

In 2003, estimated total per capita alcohol consumption (recorded and unrecorded) in Finland was 9.4 L per inhabitant. The increase in consumption was estimated to be 10% in 2004 and a further 2% in 2005 (19). Thus, this unique natural experiment involving a large reduction in the full price of alcohol (including the retail price and indirect costs, both of which were lowered by the abolishment of travelers' allowances) gave us the opportunity to directly assess what happens to alcohol-related mortality and its socioeconomic differences when prices fall and consumption increases.

Earlier studies had raised expectations that the reduction of alcohol prices would increase alcohol-related mortality, particularly in lower socioeconomic groups and possibly among youth and young adults, and that this increase would be due to acute causes rather than chronic causes. We investigated the extent to which the reduction in alcohol prices and the subsequent increase in consumption affected alcohol-related mortality overall and in different age and socioeconomic groups. Specifically, we examined whether the effect

varied with 1) sex, age, and cause of death; 2) education and occupational social class; and 3) household income and economic activity.

MATERIALS AND METHODS

Study population

The data were obtained from Statistics Finland. Two study periods were defined: 2001–2003 for the period before the price reduction and 2004–2005 for the period after the price reduction. The data were longitudinal register data from employment statistics for 2000 and 2003 and were linked individually by means of personal identification codes to records from the death register. The analysis comprised all Finns aged 15 years or more. Altogether, the men in the study population lived approximately 10.4 million person-years and the women 11.1 million person-years.

Alcohol-related mortality

Deaths were classified according to the Finnish Edition of the *International Statistical Classification of Diseases and Related Health Problems*, Tenth Revision (ICD-10). Alcohol-related deaths were defined as those in which alcohol was referred to as the underlying cause or one of the contributory causes on the death certificate. Estimating alcohol-related mortality on this basis yields more comprehensive data than the standard method, which is 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 (20, 21). The frequent use of medicolegal autopsy in Finland is one of the major factors enabling the proper attribution of alcohol intoxication as a contributory cause of death. Medicolegal autopsies were carried out for 98% of all accidental and violent deaths occurring among people under age 65 years in 2005 (20) and for more than 60% of all deaths occurring in 1987–2003 (10). In general, Finnish mortality data are assessed to be exceptionally reliable and valid due to functional legislation and death certification practices (20, 21).

The total pool of alcohol-related deaths used here consisted of the following two main categories: 1) the underlying cause of death was an alcohol-attributable disease (see below) or fatal alcohol poisoning and 2) the underlying cause was not alcohol-related, but an alcohol-attributable disease or alcohol intoxication was a contributory cause (ICD-10 code F100) (the underlying cause was then a disease of the circulatory system in 29% of the cases in 2001–2005, suicide in 22%, and an accidental fall in 10%). Alcohol-attributable diseases were: alcohol dependence syndrome (ICD-10 code F102), other mental and behavioral disorders due to alcohol use (ICD-10 codes F101 and F103–109), alcoholic cardiomyopathy (ICD-10 code I426), alcoholic liver disease (ICD-10 code K70), and alcoholic diseases of the pancreas (ICD-10, Finnish Edition, code K860), as well as a few rarely occurring additional categories (ICD-10 codes K292, G312, G4051, G621, and G721). 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 2005, among men under age 65 years, 99% of deaths due to liver cirrhosis were classified as alcoholic on the death certificate (22). It is also unlikely that illicit drug use and hepatitis C virus infection could significantly contribute to socioeconomic differences in mortality due to liver cirrhosis because of their internationally low levels in Finland (23).

We formed 2 mutually exclusive cause-of-death categories for the analysis: acute and chronic alcohol-related causes. Deaths due to chronic causes consisted of deaths for which the underlying cause was alcoholic or for which an alcohol-attributable disease was a contributory cause; deaths due to acute causes were those for which the underlying cause was not alcoholic but alcohol intoxication was a contributory factor. Deaths for which one contributory cause was intoxication and another was an alcohol-attributable disease were included in the acute-causes category.

Independent variables

The independent variables derived from employment statistics were sex, 5-year age group (15–19, . . . , 75–79, and ≥ 80 years), and socioeconomic characteristics. The 4 educational categories were based on the highest level of education achieved, obtained from the national Register of Completed Education and Degrees: basic education, secondary education, lower tertiary education, and higher tertiary education. Occupational social class was divided into 6 categories: upper white-collar, lower white-collar, skilled worker, unskilled worker, self-employed, and other. Unemployed and retired persons were classified according to their previous occupation, and persons running households were categorized according to the occupation of the head of the household. Income was measured as household disposable income per consumption unit and was divided into quintiles, with quintile boundaries defined for men and women combined in the year 2000. Income comprised all taxable income received by family members after taxes had been subtracted, including wages, capital income, and taxable income transfers. Different weights were used for adults and children in the calculation of household consumption units: for the first adult, 1.0; for other adults, 0.7; and for children, 0.5. Economic activity included 5 categories: employed, unemployed for a period of 25 months or more during the previous 3 years, unemployed for less than 25 months, pensioner, and other. The information on different sources of income came from the registers of the Finnish Tax Administration and the Social Insurance Institution.

Statistical analysis

Mortality ratios and 95% confidence intervals obtained by means of Poisson regression (Stata, version 8; Stata Corporation, College Station, Texas) were used to assess the relative differences between the age groups and the different socioeconomic characteristics. We also experimented with negative binomial regression to account for any overdispersion in our data. However, the point estimates and the test values and confidence intervals were very similar to those

obtained from Poisson regression. Since the crude mortality rates and their ratios more closely corresponded to those obtained from Poisson regression, we preferred to use it. In order to determine the relative effect of the price reduction on alcohol-related mortality according to any one socioeconomic variable, we included calendar period–social class interaction terms in the models, and we used likelihood ratio tests to derive the *P* values.

RESULTS

Between 2001–2003 and 2004–2005, numbers of alcohol-related deaths increased by approximately 16% (95% confidence interval: 12.1, 19.4) (or 22 deaths per 100,000 person-years) among men and by 31% (95% confidence interval: 22.0, 40.0) (or 8 deaths per 100,000 person-years) among women. In 2001–2003, chronic deaths made up 81% of all alcohol-related deaths among men and 86% among women. This was an increase of 21% among men and 32% among women, whereas the increases in acute deaths were 7% and 27%, respectively. Chronic deaths constituted 82% of the total increase in alcohol-related mortality, and alcoholic liver diseases alone constituted 39%. Numbers of deaths due to alcoholic liver diseases increased by 38% and 41% among men and women, respectively. The increase in intoxication-related causes was mostly due to cases in which the underlying cause of death was a disease of the circulatory system or an accidental fall.

Before the reduction in the price of alcohol, alcohol-related mortality was clearly highest among men aged 45–74 years (Figure 1). Following it, the increase was highest (more than 25%) in the age groups 55–59 years and 65–69 years, while among males under age 35 years, the observed number of alcohol-related deaths was slightly smaller after the price reduction.

Among persons aged 30 years or more, both age-adjusted alcohol-related mortality rates in 2001–2003 and the change in mortality rates were clearly higher in absolute terms in the lower educational and social-class categories (Table 1). Interaction analysis revealed that the proportional changes differed by age but not by education or social class. Changes by education and social class were quite similar in the age groups 30–59 years and 60 years or more (results not shown).

Among persons aged 30–59 years, the rates varied strongly by economic activity in 2001–2003 (Table 2): The male and female long-term unemployed had 12-fold and 17-fold rates of age-adjusted alcohol-related mortality, respectively, compared with those who had a job. The price reduction had no effect on mortality rates among employed persons, while the long-term unemployed and early-age pensioners suffered a large increase.

Before the price reduction, alcohol-related mortality rates were inversely related to household income. The patterns of change were similar, with the exception of the lowest quintile (the age-adjusted model in Table 2). When household income, age, and economic activity were introduced into the same model, the association between income and mortality was greatly reduced: For example, the relative rate in the lowest quintile decreased from 6.26 to 1.66 (data not shown).

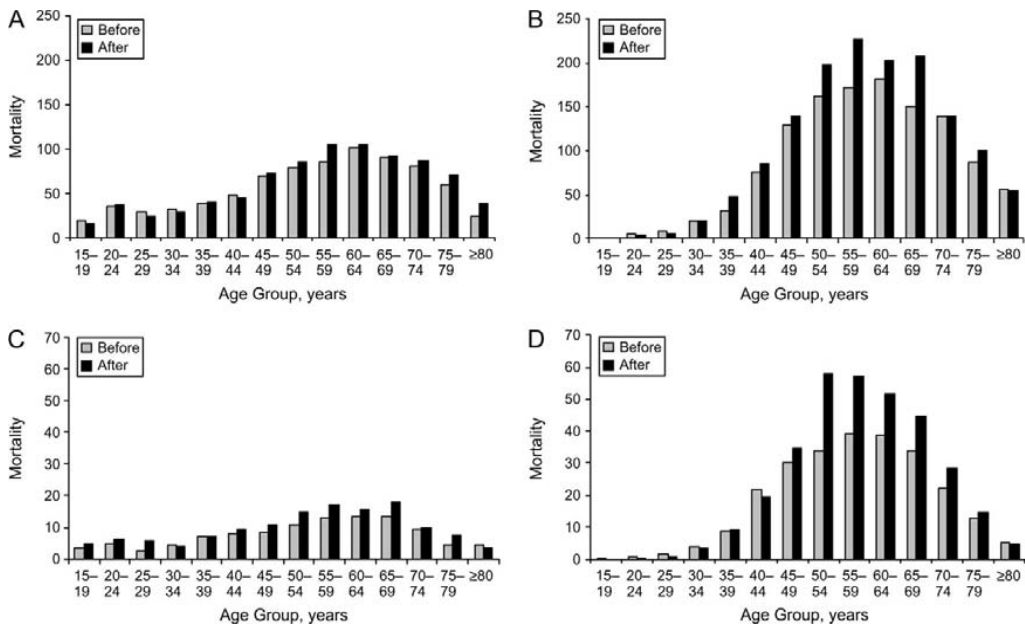


Figure 1. Numbers of acute and chronic alcohol-related deaths per 100,000 person-years among men and women aged ≥ 15 years, by age group, before (2001–2003) and after (2004–2005) a reduction in the price of alcohol, Finland, 2001–2005. A) acute mortality among men; B) chronic mortality among men; C) acute mortality among women; D) chronic mortality among women.

The differences between the income groups clearly decreased as well, but they did not disappear, nor did they become linear (Table 2, model adjusted for age and economic activity). We also examined the changes by income separately among the employed on the one hand and the unemployed and pensioners on the other (data not shown). Among males, the patterns of relative change were not substantially different in these groups, even if the absolute changes were much higher among the unemployed and pensioners. Among females, as well, the absolute changes were much higher among the unemployed and pensioners, but there were no observable differences in the patterns of relative change between these groups.

The change in mortality due to chronic causes by socio-economic characteristics in men and women aged 30 years or more (data not shown) was very similar to the pattern of change due to all alcohol-related causes as shown in Tables 1 and 2. Similarly, for mortality from acute alcohol-related causes, the patterns of change by social class and income among men and by economic activity among men and women were similar to those reported in Tables 1 and 2. In contrast, the changes did not differ by education among men or women or by social class or income among women.

DISCUSSION

Our study produced evidence of a substantial increase in alcohol-related mortality, among both men and women, after the reduction in the price of alcohol in Finland. These findings are in accordance with a study on the Finnish alcohol tax cuts and alcohol-positive sudden deaths in which blood-alcohol concentration data were subjected to time-series analysis: There was a 17% increase in alcohol-positive deaths in 2004 as compared with 2003 (24). This increase in mortality could be seen as the continuation of a 30-year trend associated with rising alcohol consumption (10, 20). However, the increase after the price reduction clearly exceeded the average annual increase of 0.8% in males and 2.6% in females that was observed in 1998–2003. Since the level of alcohol-related mortality was so much lower in women than in men, the increase in relative—but not absolute—terms exceeded that of men.

The mortality impact in the first 2 years after the price reduction was highest in subpopulations that already had the highest rates: Even in 2001–2003, the rates were highest among men and women aged 50–69 years, and the increase in absolute terms was highest in this age group as well. Among persons aged 35 years or less, the level of alcohol-related

Table 1. Crude and age-adjusted rates of alcohol-related mortality among men and women aged ≥ 30 years before (2001–2003) and after (2004–2005) a reduction in the price of alcohol, according to socioeconomic indicators, Finland, 2001–2005

	2001–2003					Change from 2001–2003 to 2004–2005		
	% of Person-Years	No. of Deaths per Year	Deaths per 100,000 Person-Years ^a	Age-Adjusted Relative Rate	95% CI	% ^b	95% CI	Deaths per 100,000 Person-Years ^c
<i>Men</i>								
Age group, years						0.012 ^d		
30–39	22.8	217	60.9	1.00		13.5	0.8, 27.8	8.2
40–49	24.9	630	161.3	2.65	2.42, 2.90	6.3	0.0, 14.1	10.2
50–59	24.4	951	248.5	4.08	3.75, 4.44	23.9	17.4, 30.8	59.4
60–69	15.0	621	264.4	4.34	3.97, 4.75	15.4	7.8, 23.5	40.7
70–79	9.7	290	190.2	3.12	2.82, 3.46	6.0	–4.3, 17.4	11.4
≥ 80	3.3	40	78.6	1.29	1.07, 1.57	16.9	–9.9, 51.7	13.3
Education						0.625 ^d		
Upper tertiary	7.9	76	61.4	1.00		7.8	–11.3, 31.1	4.8
Lower tertiary	18.2	270	94.5	1.48	1.28, 1.71	16.0	4.7, 28.5	15.1
Secondary	35.8	921	164.2	2.71	2.37, 3.10	20.3	13.9, 27.0	33.3
Basic	38.2	1,483	247.7	3.52	3.08, 4.03	16.1	11.0, 21.5	39.9
Social class						0.777 ^d		
Upper white-collar	16.5	212	82.0	1.00		8.4	–3.7, 22.0	6.9
Lower white-collar	18.4	360	124.7	1.54	1.40, 1.70	14.2	4.4, 24.9	17.7
Skilled worker	25.5	854	213.5	2.56	2.35, 2.79	17.0	10.4, 24.0	36.3
Unskilled worker	19.7	879	285.1	3.43	3.14, 3.74	18.2	11.6, 25.1	51.9
Self-employed	9.1	200	139.9	1.57	1.40, 1.76	14.9	1.8, 29.6	20.8
Other	10.8	245	144.0	1.79	1.61, 1.99	11.1	–0.5, 23.9	16.0
Total	100	2,749 ^e	175.3			17.1	13.4, 21.0	30.0
<i>Women</i>								
Age group, years						0.002 ^d		
30–39	19.9	42	12.2	1.00		1.8	–23.0, 34.5	0.2
40–49	22.1	131	34.5	2.82	2.30, 3.44	7.3	–8.0, 25.1	2.5
50–59	22.0	183	48.0	3.93	3.24, 4.77	52.7	36.0, 71.5	25.3
60–69	15.1	130	49.9	4.08	3.34, 4.98	30.6	13.1, 50.8	15.3
70–79	13.1	56	24.8	2.03	1.61, 2.56	23.8	0.0, 55.1	5.9
≥ 80	7.8	13	9.4	0.77	0.53, 1.11	–16.1	–50.0, 40.9	–1.5
Education						0.288 ^d		
Upper tertiary	6.4	12	10.8	1.00		56.0	0.4, 142.3	6.0
Lower tertiary	21.3	63	17.1	1.49	1.04, 2.13	11.5	–9.9, 38.0	2.0
Secondary	31.9	166	30.2	2.51	1.79, 3.52	39.8	23.5, 58.3	12.0
Basic	40.5	314	44.9	4.13	2.95, 5.77	34.2	22.0, 47.6	15.4
Social class						0.571 ^d		
Upper white-collar	13.8	41	17.2	1.00		31.9	2.3, 70.1	5.5
Lower white-collar	38.7	182	27.2	1.52	1.25, 1.84	34.9	19.5, 52.2	9.5
Skilled worker	9.8	81	47.8	3.06	2.46, 3.80	10.6	–9.0, 34.5	5.1
Unskilled worker	21.4	179	48.4	2.72	2.23, 3.31	33.5	18.0, 51.1	16.2
Self-employed	5.4	32	34.5	1.88	1.44, 2.46	17.6	–12.9, 58.7	6.1
Other	10.9	41	21.7	1.51	1.18, 1.95	38.4	7.4, 78.1	8.3
Total	100	555 ^e	32.1			30.6	21.7, 40.0	9.8

Abbreviation: CI, confidence interval.

^a Unstandardized.^b Derived from the age-adjusted interaction term for the interaction between each variable and period.^c Obtained by multiplying the mortality rate before the change by the percentage of change.^d *P* value for difference in change obtained from a model that included an interaction term for the interaction between each variable and period.^e Differences result from rounding.

Table 2. Rates of alcohol-related mortality among men and women aged 30–59 years before (2001–2003) and after (2004–2005) a reduction in the price of alcohol, according to economic activity and household disposable income per consumption unit, Finland, 2001–2005

	Change from 2001–2003 to 2004–2005											
	2001–2003					Age-Adjusted					Adjusted for Age and Economic Activity	
	% of Person-Years	No. of Deaths per Year	Deaths per 100,000 Person-Years ^a	Age-Adjusted Relative Rate	95% CI	% ^b	95% CI	Deaths per 100,000 Person-Years ^c	% ^b	95% CI	Deaths per 100,000 Person-Years ^c	
Men												
Economic activity						0.001 ^d						
Employed	78.4	530	59.9	1.00			–4.7, 11.1	1.7				
Unemployed for ≥25 months	5.4	477	783.7	12.11	11.28, 13.01	2.2	11.7, 31.5	166.1				
Unemployed for <25 months	4.0	153	336.9	5.54	4.99, 6.15	30.1	14.2, 48.1	101.4				
Pensioned	6.9	482	619.1	8.15	7.58, 8.76	27.1	18.0, 36.9	167.8				
Other	5.3	156	259.5	4.50	4.06, 4.99	21.5	6.3, 38.8	55.8				
Income quintile ^e						<0.001 ^d						
1 (highest)	28.7	222	68.6	1.00		17.2	5.6, 30.1	11.8	13.5	2.3, 26.0	9.3	
2	22.8	250	96.7	1.56	1.40, 1.73	17.5	5.3, 31.0	16.9	2.5	–8.1, 14.4	2.4	
3	18.1	257	125.9	2.19	1.97, 2.43	78.8	61.7, 97.7	99.2	44.8	30.9, 60.1	56.4	
4	14.1	380	238.0	4.18	3.80, 4.60	64.0	50.6, 78.6	152.3	37.4	26.6, 49.6	89.0	
5 (lowest)	14.2	586	365.7	6.26	5.72, 6.84	19.4	10.4, 29.2	70.9	22.1	12.8, 32.1	80.8	
Total	100	1,798	159.1			19.2	14.6, 24.1	30.5				
Women												
Economic activity						0.011 ^d						
Employed	75.5	97	11.7	1.00		8.0	–9.4, 28.7	0.9				
Unemployed for ≥25 months	3.7	89	218.3	16.68	14.12, 19.70	49.9	24.8, 79.9	108.9				
Unemployed for <25 months	5.7	36	57.1	5.03	4.03, 6.28	81.3	39.8, 135.1	46.4				
Pensioned	5.5	98	162.1	11.11	9.43, 13.09	36.6	16.3, 60.5	59.3				
Other	9.7	36	33.3	3.41	2.73, 4.26	54.0	18.3, 100.4	18.0				
Income quintile ^e						0.063 ^d						
1 (highest)	26.6	43	14.7	1.00		17.8	–6.8, 48.9	2.6	19.9	–6.8, 49.0	2.9	
2	23.4	48	18.6	1.41	1.11, 1.79	61.2	28.3, 102.5	11.4	47.1	17.1, 84.9	8.7	
3	19.1	55	26.1	2.23	1.77, 2.81	81.5	46.2, 125.3	21.2	50.6	21.2, 87.0	13.2	
4	15.4	84	49.0	4.42	3.57, 5.47	78.2	49.1, 112.9	38.3	58.2	32.4, 89.1	28.5	
5 (lowest)	14.5	113	70.2	6.60	5.38, 8.09	48.4	25.1, 76.1	34.0	57.3	32.5, 86.7	40.2	
Total	100	356	32.2			33.6	22.4, 45.8	10.8				

Abbreviation: CI, confidence interval.

^a Unstandardized.

^b Derived from the age-adjusted interaction term for the interaction between each variable and period.

^c Obtained by multiplying the mortality rate before the change by the percentage of change.

^d *P* value for difference in change obtained from a model that included an interaction term for the interaction between each variable and period.

^e One category ("income unknown") is not shown.

mortality did not increase; if anything, it decreased. This is very noteworthy, because alcohol consumption is generally more responsive to increases in price among young adults than among older adults (1); this has raised expectations that this is also true when the price of alcohol goes down. This gap between older and younger persons could additionally be seen as the continuation of a previous trend (10): There was a favorable trend in the 1990s and early 2000s among men under age 45 years, mainly due to a decrease in intoxication-related accidents and violence, while total alcohol-related mortality increased among older men. The different developments would appear to be accounted for by increased alcohol consumption among men aged 45 years or more after the mid-1990s, and also after 2004, whereas consumption was stable or decreased among younger Finns (25). Drinking habits on the population level change slowly, but these results suggest that younger generations may be adopting less damaging alcohol consumption patterns. The phenomenon at hand seems to be rather more an accentuation of a longer-term change than an isolated consequence of the price reduction. However, one must be cautious when making inferences about alcohol consumption on the basis of alcohol-related mortality data.

Alcoholic liver diseases alone constituted 39% of the increase in total alcohol-related mortality after the price reduction. The recorded increase in liver-disease deaths with a concurrent increase of 1.2 L in per capita alcohol consumption is clearly higher than would be expected on the basis of a time-series analysis of the longer-term connection between liver cirrhosis mortality and per capita consumption in Finland, but it is in line with the effect size observed for Sweden in 1950–1995 (26). With regard to the relation between alcohol consumption and disease, one must consider that not only present consumption but also past consumption impinges on the risk of alcohol-attributable disease. It has been suggested that the latency period for liver cirrhosis could be very long—20 years of excessive drinking may be needed (27). However, there is also previous evidence on an instantaneous response to changes in consumption on the aggregate level in cirrhosis mortality—for example, in France during World War II and in Russia after 1990 (17, 28). This seeming contradiction in the case of a rapid increase in cirrhosis mortality is best understood in terms of the water-glass analogy: Persons who died from cirrhosis during that short period after the price reduction already had their water glass almost full, and the increased consumption took an increased number of previous heavy drinkers over the rim.

As far as the impact of the price reduction on subpopulation differences in alcohol-related mortality is concerned, the most salient finding is a huge chasm between the employed and the unemployed among people aged 30–59 years. Mortality increased strongly after the price reduction, particularly among the long-term unemployed and pensioners. This was mostly due to chronic causes, but deaths due to acute causes also increased to some extent. At least two relevant but interlaced explanations for this divergence should be taken into account. Firstly, because of both selection and causal processes (29), heavy drinkers are overrepresented in the population of unemployed and early-age

pensioners, and chronic conditions may more quickly respond to an increase in consumption among them. Secondly, the unemployed and early-age pensioners include more poor persons whose drinking may have previously been restricted by the higher price of alcohol.

The change in alcohol-related mortality due to acute causes as opposed to chronic causes was slight. Here, too, the most salient differentials were for economic activity: The mortality rates of the long-term unemployed increased, but rates remained almost unchanged for the employed. This may indicate that hazardous alcohol consumption has not increased substantially among employed persons.

The finding that the increase in alcohol-related mortality was higher, in absolute terms, in the lower educational and socioeconomic groups was not a surprise, given the preexisting adverse trend for persons of lower socioeconomic and educational status in Finland in 1987–2003 (10, 18). Additionally, men with the lowest level of education increased their alcohol consumption in the 2000s, while the more highly educated kept it stable (25).

The observed results concerning changes in alcohol-related mortality according to socioeconomic indicators may be partly attributable to beverage preferences. It was the price of spirits in particular, and to some extent the price of beer (but not of wine), that went down. According to survey-based results, men with a basic education consumed 61% of their total ethanol intake as beer, 26% as spirits, and only 4% as wine, while the corresponding proportions for the highly educated were 51%, 15%, and 25% (30). These results indicate that the beverages consumed by the less educated were affected by the price changes to a greater extent than the beverages consumed by the more highly educated.

The finding that the effect of the price reduction was not greatest among the poorest in household-income terms, although they had the highest mortality rates before the price reduction, was undoubtedly at odds with expectations. One might assume that if, on the whole, alcohol-related mortality were to increase because of the price reduction, it would happen among poor people, since their alcohol consumption may have previously been restricted by the higher price. Finally, the lack of a perfect gradient in change according to household income could be attributed to the possibility that alcohol is still too expensive for the poorest persons in Finland. This peculiarity in the results decreased when we controlled for economic activity.

Earlier studies yielded contradictory results on whether price responsiveness differs in various alcohol user groups: Manning et al. (31) concluded on the basis of cross-sectional data that heavy (and light) drinkers were much less responsive to prices than were moderate drinkers, while a more recent study produced evidence of substantial price responsiveness among heavy drinkers with symptoms of alcohol abuse or dependence (32). In the current study, the large increase in mortality due to liver diseases indicates that heavy drinkers, at least, were very responsive to the price reduction.

This unique natural experiment has shown that the relation between changes in the price of alcohol and alcohol-related mortality in Finland is not the same in all subpopulations. It

appears that the socially disadvantaged and heavy drinkers have been most sensitive to the price decreases, whereas grievous problems measured in terms of mortality have not increased among those better positioned in society. Hence, it may be claimed that high prices protect the worst-off members of the population against alcohol-related problems and that alcohol price cuts may impose the biggest burden on persons who already suffer the most from alcohol-related harm. More time is needed, however, to assess the full effect of the reduction in the price of alcohol on alcohol-related mortality.

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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

IV

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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

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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 9.41 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 produce³⁸. 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 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

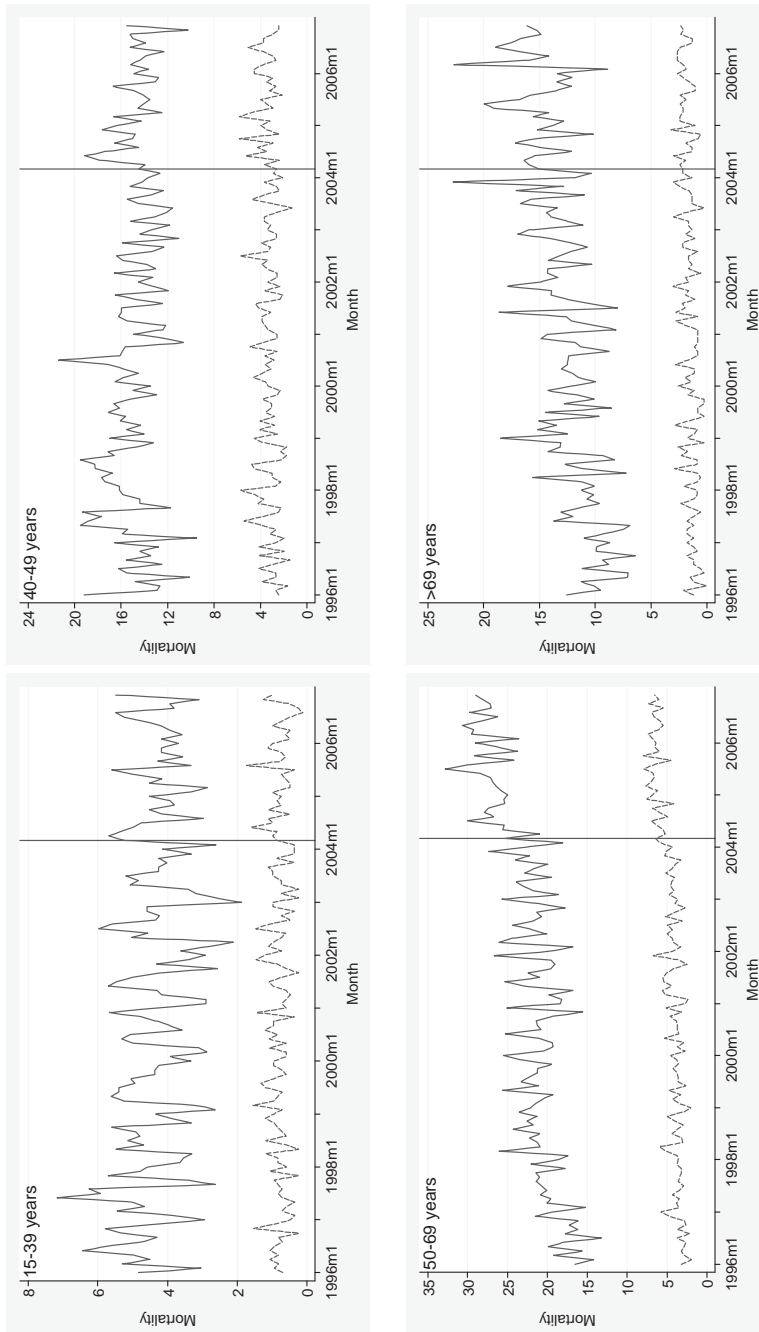


Figure 1 Monthly alcohol-related mortality rates per 100 000 person-years among men (solid line) and women (dashed line), aged 15–39, 40–49, 50–69 and >69 years, Finland, 1996–2006. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004

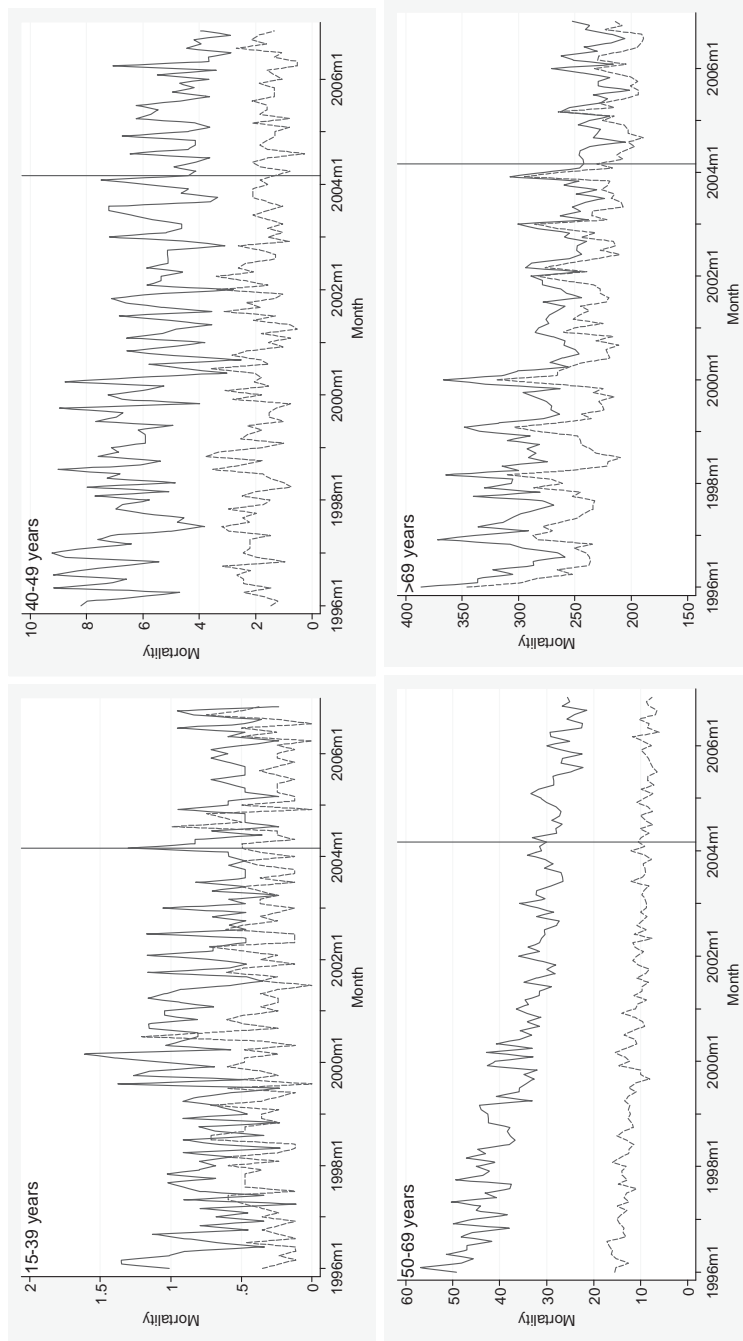


Figure 2 Monthly rates of CVD mortality per 100 000 person-years among men (solid line) and women (dashed line), aged 15–39, 40–49, 50–69 and >69 years, Finland, 1996–2006. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004

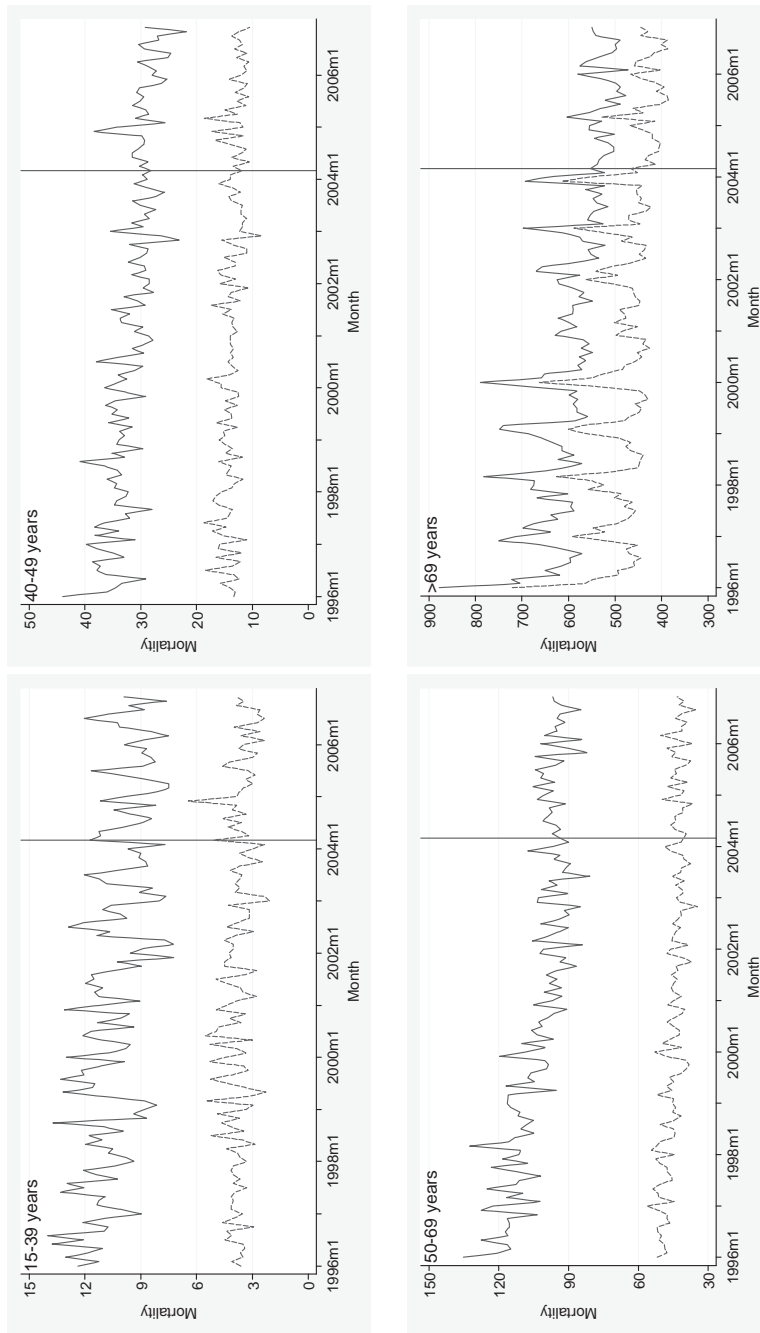


Figure 3 Monthly all-cause mortality rates per 100 000 person-years among men (solid line) and women (dashed line), aged 15–39, 40–49, 50–69 and >69 years, Finland, 1996–2006. The vertical line indicates the realization of the reduction in alcohol prices on 1 March 2004

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			

^aDerived from exp(estimate).

^bObtained by multiplying the mortality rate before the change by the percentage change. Q(24) is the Portmanteau test for residual autocorrelation with 24 lags.

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-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

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			

^aDerived from exp(estimate).

^bObtained by multiplying the mortality rate before the change by the percentage change. Q(24) is the Portmanteau test for residual autocorrelation with 24 lags.

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 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

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) ₁₂ ; 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			

^aDerived from exp(estimate).^bObtained 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

^aDerived from time series analyses.

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 ≥ 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 (≤ 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 years⁴¹ and among those aged 50–69 years⁴² 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 ≥ 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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The effects of a large reduction in alcohol prices on hospitalisations related to alcohol: a population-based natural experiment

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Running head: Alcohol price reduction and hospitalisations.

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Abstract

Aims. Finland experienced a large reduction in alcohol prices in 2004 due to the lowering of alcohol taxes by about one-third and the abolition of duty-free allowances for travellers from the EU. We examined the effects of these changes on alcohol-related hospitalisations. **Design.** Time-series intervention analyses of monthly aggregations of hospitalisation for acute and chronic causes among men and women aged 15-39, 40-49, 50-69 and over 69 years. **Setting.** Finland, 1996-2006. **Findings.** After the price reduction the chronic hospitalisation rate for men increased among those below the age 70. It was largest among those aged 50-69 years: 22% which implies an increase of 18.0 monthly hospitalisations per 100 000 person-years, and there was an 11% and a 16% (11.5 and 4.8 monthly hospitalisations) increase among those aged 40-49 and 15-39, respectively. Among the women the rate increased by 23% (4.0 monthly hospitalisations) in the 50-69 year-olds, and decreased in the under-40s. The increase in all of the population groups was mainly due to an increase in mental and behavioural disorders due to alcohol. Acute hospitalisations increased by 17% and 20% (6.2 and 7.0 per month) among men aged 40-49 and 50-69 years, respectively, and by 38% among women aged 50-69 years (2.3 per month). **Conclusions.** The results, obtained in a natural experimental setting when trends and seasonal variation had been taken into account, suggest that the reduction in alcohol prices led to increases in alcohol-related hospitalisation in certain population groups in Finland.

Keywords. Alcohol drinking, commerce, economics, alcohol-related disorders, hospitalisation.

INTRODUCTION

Background and the context of the study

Evidence suggests that changes in alcohol prices are inversely associated with changes in consumption and alcohol-related problems. Research on the alcohol consumption and the harm it causes is fairly plentiful, but studies on the effects of alcohol prices on the damage, particularly in a natural experimental setting, are called for [1, 2]. This paper presents new epidemiological evidence based on a natural experiment on the association between a policy-driven abrupt and substantial change in alcohol prices and alcohol-related harm in terms of hospitalisation.

The changes in the Finnish alcohol policy in 2004 could be regarded as a natural experiment. On January 1 it became legal to import virtually unlimited amounts of alcoholic beverages from other EU countries for one's own use due to the deregulation of import quotas within the Union. Taxes on alcohol were reduced by an average of 33% on March 1: 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 May 1 which affected the Finnish alcohol market to a significant extent because of the proximity of the two countries and the essentially lower price of alcohol in Estonia. The estimated total per-capita alcohol consumption in Finland (recorded and unrecorded)

was 9.4 litres per inhabitant in 2003. The estimated increase was to be about 10% in 2004, after which it has remained more or less on that level [4].

Alcohol prices and alcohol-related mortality and morbidity

The effects of changes in alcohol prices on mortality and morbidity have been assessed in Finland and elsewhere. Recent research has shown that the reduction in prices in 2004 had a considerable effect on alcohol-related mortality. The results of a register-based study indicated, following a before-after comparison, 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 further demonstrated a subsequent substantial increase in alcohol-related mortality among persons aged 40 years or more [6]. This study also reported, however, that all-cause mortality decreased among persons over age of 69, when deaths from cardiovascular disease are prevalent. One of the few studies on mortality apart from this natural experiment, a cross-sectional time-series investigation covering the years 1982-1988 across all 48 states in the U.S. gave no evidence that higher alcohol prices decreased mortality rates for alcohol-attributable primary causes of death [7].

Studies addressing the effect of alcohol prices on alcohol-related morbidity are few and far between. There was one study using state-level data for the years 1975-1985 that reported an inverse relationship between industrial injuries and beer taxes [8]. In addition, according to an individual-level study conducted in Switzerland a reduction of 30-50% in the retail price of foreign spirits resulted in an increase in self-reported alcohol-related problems, measured on items taken from the Alcohol Use Disorder Identification Test (AUDIT), particularly among younger age groups who consumed more spirits [9].

Alcohol consumption and alcohol-related morbidity and mortality

Population-level research on the relation between alcohol consumption and alcohol-attributable morbidity is more plentiful but the evidence is not consistent. Furthermore, this relation is not formally analysed in most of the studies. A 1987 cross-sectional study in the US found a significant correlation between state-level consumption and alcohol-related hospitalisations among elderly people [10]. When consumption increased 2.2-fold between 1968 and 1974 in Finland due to the liberalisation of the alcohol policy, cause-specific hospitalisations increased 1.2-2.1-fold among men and 1.0-2.4-fold among women, liver cirrhosis being the least affected cause [11]. A time-series study conducted in 1980-1994 in Sweden reported a significant relation between alcohol sales and hospital admissions due to cirrho-

sis, but not for admissions due to alcoholism, alcohol psychoses and intoxication in 1980-1994 in Sweden [12], whereas other studies from Sweden and Holland have found that the trends in hospitalisation for liver disease followed only partially or not at all the trends in consumption [13, 14]. According to an Australian study conducted in 1971-1984, hospitalisation among men hospitalisation among men due to acute and chronic alcohol pancreatitis was related to consumption [15] whereas a Finnish investigation in 1987-2007 found that hospitalisation due to acute alcohol pancreatitis did not follow the trends in alcohol consumption whereas it did for liver cirrhosis did [16].

The relation between alcohol consumption and mortality on the population level has attracted slightly more research attention, particularly in a project entitled ECAS, the European Comparative Alcohol Study, which comprised a number of time-series studies covering the period from the 1950s to the mid-1990s and 14 European countries (and Canada in some cases). These studies investigated the association between per-capita alcohol consumption and mortality from different causes, producing a number of estimates obtained from different models and diversely stratified by sex and various numbers of age groups. In 12 out of these 14 countries statistically significant positive estimates were found for cirrhosis mortality among men, and in nine of them among women [17]. In nine of countries, on average, depending on the model employed, statistically significant positive estimates were found for pancreatitis mortality [18]. Moreover, in 21% strata defined by 14 countries, age, and sex, were found significant positive estimates for mortality both from accidents and homicide [19, 20], and in 14% for suicide mortality [21].

Aims of the study

In general, alcohol-related morbidity is much less frequently studied than alcohol-related mortality, which is due at least in part to the lack of data of acceptable quality [22]. However, they are equally important for several reasons. When the focus is on the effects of the price change, morbidity as an outcome measure contributes to a more comprehensive health effect by expanding the scope from the most extreme measure. The effect in terms of public health and the national economy is even more significant in that the number of hospitalisations is almost ten times the number of deaths. Furthermore, when the development of alcohol-related problems is understood as a continuum, hospitalisations are one step ahead of deaths, and consequently their analyses could detect an increase in problems among younger people, for example, earlier than mortality analysis would do.

The present study focused on the effects of a substantial reduction in alcohol prices on alcohol-related hospitalisation in a rare natural experimental setting. More specifically, the aim of this study was to document and assess, by means of time-series analysis, a potential change in chronic and acute alcohol-related hospitalisations, stratified by sex and age, following the lowering of alcohol prices in Finland in 2004. This was the first study examining these effects in such a setting.

METHODS

Data

Hospital discharge data for the years 1996-2006 were obtained from the National Institute for Health and Welfare (formerly the National Research and Development Centre for Welfare and Health). The monthly data were stratified by sex and five-year age groups (15-19, ..., 75-79, 80+).

Hospital diagnoses were classified according to the Finnish edition (FCD) of the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10).

Data on hospital utilisation attributable to alcohol-related diagnoses were obtained from the Finnish Hospital Discharge Register, which gathers comprehensive information on individual patient hospitalisations in all Finnish public and private hospitals. We defined the causes related to alcohol as those with a reference to alcohol in the primary diagnosis. In all there were almost 283 000 hospitalisations due to alcohol, of which 27% referred to alcohol dependence syndrome (ICD-10 code F102), 20% to other mental and behavioural disorders due to alcohol (ICD-10 codes F101, F103-109), 1.7% to alcohol poisoning (ICD-10 code X45), 10% to alcohol liver diseases (ICD-10 code K70), 0.2% to alcoholic cardiomyopathy (ICD-10 code I426), 9% to alcoholic diseases of the pancreas (ICD-10 code, Finnish Edition K860), 0.9% to alcoholic gastritis (ICD-10 code K292), 28% to alcohol intoxication (ICD-10 code F100), 0.2% to maternal care for (suspected) foetal damage (ICD-10 code O354), and 3.7% to other alcoholic diseases (ICD-10 codes G312, G4051, G621, G721). Men comprised 81% of all of these hospitalisations.

We formed two mutually exclusive cause-of-hospitalization categories for the analysis: acute (30% of all cases) and chronic (70%) alcohol-attributable causes. The former comprised hospitalisations following a primary diagnosis of alcohol intoxication or alcohol poisoning, and the latter a primary diagnosis of alcohol-attributable disease. The chronic category was assumed to be a stronger and a more reliable marker of adverse alcohol use. It is noteworthy that two thirds of the chronic causes were mental or behavioural disorders related to alcohol abuse (ICD-10 codes F101-109) by definition. Due to inconsis-

tencies in earlier studies regarding the association between consumption and hospitalisation for different alcohol-related causes [11-16], we conducted additional analyses separately for alcohol dependence syndrome and other mental and behavioural disorders due to alcohol (below we use the term 'mental and behavioural disorders'), and other chronic causes related to alcohol. 41% of hospitalisations in the last-mentioned categories were due to alcohol liver diseases and 37% to diseases of the pancreas.

Statistical analysis

For the analyses we converted monthly hospitalisations to rates per 100 000 person-years. We used autoregressive integrated moving average (ARIMA) intervention time-series analyses to model these monthly alcohol-related hospitalization rates. We assumed the intervention took place on 1 March 2004. Estimation of the models 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 before the intervention [23]. The intervention component is added in the second phase in order to obtain the impact-assessment model that allows the causal attribution of changes in time series to given events. Unlike other methods, time-series analysis can detect trends and seasonal variation, which have a tendency to bias the assessment of the impact on an outcome measure.

There are several alternatives to the pure jump (i.e. zero-order step function) in modelling the intervention function: 1) the pulse function, which best characterises a purely temporary effect, 2) the gradually changing function and 3) the prolonged impulse function [24]. We rejected these models after fitting them because none were essentially better in terms of model identification than the pure jump.

Outliers may distort specification of the ARIMA model and its parameter estimates unless they are properly taken into account [25, 26]. A dummy variable was added to the models as a regression parameter in several series because the outliers hindered the model specification. Their presence or absence did not, however, significantly affect the results. Variance was stabilised by means of natural log transformation in all the series since it appeared to change over time.

RESULTS

Figures 1 and 2 show the time series for monthly hospitalisation rates related to chronic and acute alcohol-attributable causes (raw data) for men and women. The vertical line indicates the realisation of the reduction in alcohol prices on March 1, in 2004. Seasonal variation is apparent in most subgroups but

the direction of the trend or drift is age and sex-specific. With regard to chronic causes there was a mainly declining trend among persons under the age of 50, but no distinct trend among persons aged 50 years or more. As to acute causes, there was no distinctive trend among persons under 50 years of age, but there was a general increase in hospitalisations among the older groups.

Table 1 presents a formal assessment of the impact of the reduction in alcohol prices on the rates of hospitalisation due to chronic alcohol-attributable causes. The rate increased in every age group under the age of 70 among the men, being the largest in both absolute and relative terms among those aged 50-69: an impact parameter of 0.198 indicates an increase of 22% in the mean rate, which implies an increase of 18.0 monthly hospitalisations per 100 000 person-years. Among men aged 15-39 and 40-49 the estimates implied an increase of 16% and 11% (4.8 and 11.5 monthly hospitalisations per 100 000 person-years), respectively.

As far as the women were concerned the estimates indicated an increase of 23% among those aged 50-69, implying an increase of 4.0 monthly hospitalisations per 100 000, whereas the rate decreased by 8% among those aged 15-39 years (0.6 fewer monthly hospitalisations).

Separate analyses of hospitalisations due to mental and behavioural disorders and other chronic causes reveal a clear-cut division between the two hospitalization categories (table not shown). All the impact estimates of mental and behavioural disorders except among women aged 15-39 years were clearly larger than those of other chronic categories. There was a statistically significant percentage increase in hospitalisations due to mental and behavioural disorders of 15.1 (95% CI: 3.0, 28.8), 24.0 (95% CI: 17.4, 31.1), 21.8 (95% CI: 7.5, 38.0) and 29.2 (95% CI: 18.2, 41.2) among men aged 40-49, 50-69 and more than 69 years, and women aged 50-69 years, respectively, and a decrease of 24.7% (95% CI: -32.5, -16.1) among women aged 15-39. As far as other chronic causes were concerned, the only statistically significant impact estimate was for women aged 50-69 years: hospitalisations increased by 22.5% (95% CI: 9.6, 36.8). However, the point estimates were also positive in all the other groups (ranging from 4.9% to 20.7%) except for men aged over 69 (-8.9%) and women aged 40-49 (-2.8%) years.

Table 2 depicts the assessed effect of the price reduction on hospitalisations related to acute alcohol-attributable causes. The impact parameters of 0.161 and 0.186 among men aged 40-49 and 50-69 indicate an increase of 17% and 20%, respectively, in the mean number of hospitalisations implying a re-

spective monthly increase of 7.0 and 6.2 per 100 000. Among women aged 50-69 there was an increase of 38% (2.3 additional hospitalisations per 100 000).

DISCUSSION

Summary of the main results

Hospitalisations due to chronic and acute alcohol-related causes constituted 1.3% and 0.6%, respectively, of the over 15 million hospitalisations in Finland during the study period of 1996-2006. The increase after the price reduction was largest in both in absolute and relative terms, and for both chronic and acute alcohol-related causes among persons aged 50-69 years. As a matter of fact, this was the only female age-group in which there was any statistically significant increase, whereas there was also some measurable increase among younger men.

Variations between chronic and acute hospitalisations

The increase in chronic hospitalisations was almost entirely attributable to mental and behavioural disorders, whereas the change in hospitalisations due to liver and pancreatic disease, although mostly positive, could not be separated from the random changes. These findings are somewhat in accordance with the results of earlier studies from Sweden in 1969-2001 and Holland in 1970-1994, which found that the trends in hospitalisations due to liver disease followed the trends in consumption only to some extent, or not at all [13, 14]. Furthermore, a Finnish study in 1969-1975 reported mostly larger increases in hospitalisations due to mental and behavioural disorders (increases in admission rates ranging from 1.7 to 2.1 among men and 2.3 to 2.4 among women) than to cirrhosis and pancreatitis (increases in rates ranging from 1.2 to 2.0 among men and from 1.0 to 1.4 among women) following an increase in alcohol sales per capita from 2.9 litres in 1968 to 6.5 litres in 1974 [11]. Nevertheless, the association between consumption and hospitalisation was not formally estimated. Another study conducted in the Stockholm area, however, reported only a weak and non-significant association between alcohol sales and hospital admissions for alcoholism, alcohol intoxication and alcohol psychosis, whereas the association was significant with regard to cirrhosis admissions [12]. Differences in the scope and length of the follow-up and in methodological features, or in how the consumption change translated into changes in drinking patterns or in the distribution of consumption, may account for the discrepancies between the results of this and our study.

Hospitalisations attributable to acute causes increased in some measure as well, but there were at least two differing factors, on top of the magnitude of the change, in comparison with the chronic causes. First, it appears that there was a slightly more systematic increase in acute than in chronic causes among women aged less than 70, but not among the men. This finding may be attributable to the changes in drinking patterns reported in a recent survey: the number of binge-drinking occasions increased by 63% among women between 2000 and 2008 in Finland whereas it remained unchanged among men [27]. Secondly, chronic alcohol-related hospitalisation could be regarded as an indicator of more severe adverse alcohol-attributable problems than acute events. For example, a hospitalisation due to alcohol intoxication may certainly be a serious event as such, but it does not necessarily imply that the hospitalised person frequently experiences excess alcohol consumption and its consequences. The formation of chronic and acute analytical categories, thus, improved the validity of the study in this respect.

Gender differences

On the whole, the increase in acute and chronic hospitalisation rates was larger among men than among women, consistently in absolute terms, and in most cases also in relative terms. The larger relative increase among women aged 50-69 years may be partially attributable to the much lower level of alcohol-related hospitalisation (one-fifth of the men's rate in both the acute and chronic categories) before the price reduction. The much smaller proportion of women in both the level of and increase in hospitalisation is indicative of the fact that gender differences still exist with regard to alcohol consumption in terms of volume and drinking patterns: according to a recent Finnish survey women consumed around 25% of all alcohol sold, and the proportion of heavy drinkers was 1.6% in 2008, and 7.3% among men in 2008 [28]. However, even if the survey showed a greater increase in binge drinking among women than among men, it did not reveal any essential gender differences in changes in total consumption between 2000 and 2008.

Comparisons with previous studies on mortality

These findings could be compared with the two studies reporting the impact of the same price reduction on alcohol-related mortality. For the population as a whole there was no systematic difference between the effects on hospitalisation and on mortality, i.e. it cannot be argued that the impact was greater on the former than on the latter, or vice versa. It was reported in a study employing a before-after design and time-series analysis that the increase in alcohol-related mortality was largest in absolute terms

among persons aged 50-69 years [5, 6], which was also the case for alcohol-related hospitalisations. An increase in consumption seems to account for the greatest impact in this age group: consumption increased the most among them at least between 2000 and 2008, according to a recent survey [28].

It is noteworthy that alcohol-related hospitalisation increased by 16% among men aged less than 40 years, even if alcohol-related mortality did not [5, 6]. This finding indicates an increase in harmful drinking among this age group, at least to some extent. For women aged less than 50 years, in contrast, the results of this study are in accord with these earlier findings on alcohol-related mortality in that the change was marginal with regard to alcohol-related outcomes [5, 6]. Hence, it appears that harmful drinking did not increase in the younger female age groups after the price reduction to the extent that it would be reflected in hospitalisation or mortality.

It is important to consider the different nature of the two categories of chronic hospitalisation in comparison with mortality. Mental and behavioural disorders primarily reflect mental health, which cannot necessarily be easily captured in studies on mortality, whereas the correspondence between mortality and hospitalisation for diseases of the liver and pancreas should be better. It is not, therefore, inconsistent that the increase in hospitalisations was due to an increase in mental and behavioural disorders rather than in diseases of the liver or pancreas, causes that – together with alcoholic cardiovascular disease – accounted for a major part of the increase in mortality after the price reduction [5]. Overall, the finding that the increase in hospitalisations was mainly attributable to mental and behavioural disorders is important for two reasons. First, hospitalisations for these causes predicts premature mortality: it has been found that a person with alcohol dependence syndrome is likely to die 15 years earlier than the average age of death among the general population [29], the leading causes of death being heart disease, cancer, accidents and suicide [30]. Secondly, it may partially explain the above-mentioned discrepancy that among men aged less than 40 years: hospitalisations increased (73% of the chronic cases were attributable to these mental and behavioural disorders) but mortality attributable to alcohol-related causes did not. One factor behind this is the time lag: shorter latency periods have been found for alcohol-related mental and behavioural disorders than for other major alcohol-related diseases. According to Schuckit the average person with alcoholism first demonstrates the clustering of major alcohol-related life problems in the mid-20s to early 30s, and most people with alcoholism present for treatment in their early 40s – after more than a decade of difficulties [30]. It is, thus, likely that by the age of about 30, approximately half of those who fulfil the criteria for alcoholism have already reached that stage [30]. For liver cirrhosis, for example, 20 years of excessive drinking may be needed [31]. The increase in alcohol abuse among younger men is in accord, to some extent, with the results of a survey-

based study showing an increase in the proportion of heavy drinkers of 25% and 7%, respectively, among men aged 15-29 and 30-49 between 2000 and 2008, even if the change was not statistically significant [28]. Moreover, it remains unclear whether there has been any specific change since 2004. On the other hand, alcohol consumption generally declined in these age groups and the estimated proportion of abstainers doubled among 15-29-year-olds [28].

Conclusions

This was the first study examining the effects of a reduction in alcohol prices on alcohol-related hospitalisation in a natural experimental setting. The results obtained by means of time-series analysis suggest consequent increases among men under 70 and among women aged 50-69 years in Finland even when trends and seasonal variation were taken into account. The increases were mainly attributable to an increase in alcohol dependence syndrome and other alcohol-related mental and behavioural disorders. The study produced additional evidence that the price of alcohol may affect alcohol-related health problems in certain population sub-groups.

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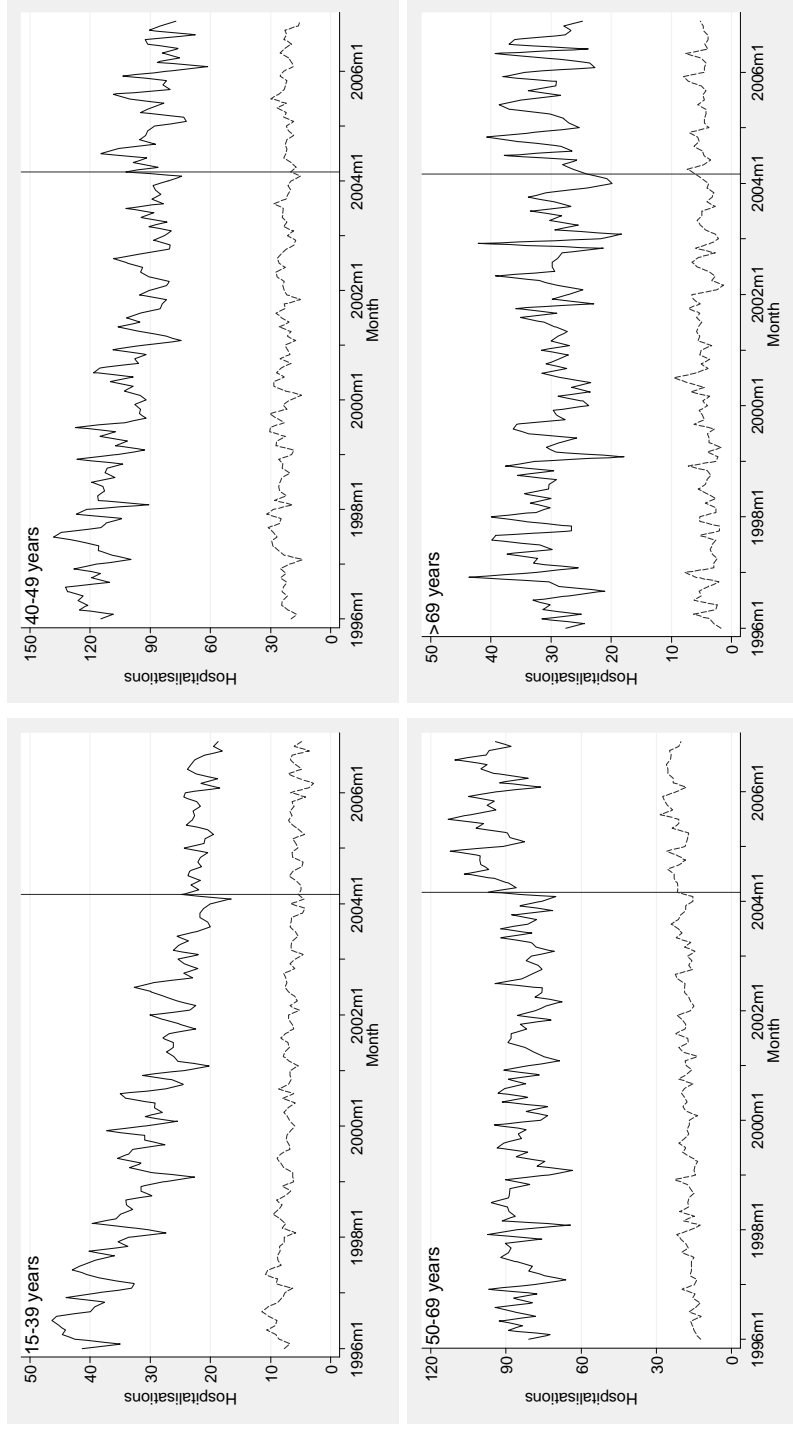


Figure 1 Monthly hospitalisation rates related to chronic alcohol causes per 100 000 person-years among men (solid line) and women (dashed line) aged 15-39 years, 40-49 years, 50-69 years, and >69 years, Finland, 1996-2006. The vertical line indicates the realisation of the reduction in alcohol prices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

Table 1 The effects of the reduction in alcohol prices on hospitalisations related to chronic alcohol-attributable causes by sex and age, natural logarithmic ARIMA models.

	Hospitalisations per month	Change				Hospitalisations per 100 000 ^b
		Estimate	% in level ^a	Lower CI	Upper CI	
Men						
15-39 years	246					
Impact		0.145	15.6	2.2	30.9	4.8
Noise		ARIMA (1,1,1)(1,0,1) ₁₂ ; Q(24)=19.43; P=0.729				
40-49 years	396					
Impact		0.107	11.2	1.9	21.5	11.5
Noise		ARIMA (0,1,1)(1,0,0) ₁₂ ; Q(24)=26.17; P=0.345				
50-69 years	518					
Impact		0.198	21.9	16.9	27.0	18.0
Noise		ARIMA (0,1,1)(2,0,0) ₁₂ ; Q(24)=24.30; P=0.445				
>69 years	59					
Impact		0.030	3.1	-5.6	12.5	0.9
Noise		ARIMA (0,0,1)(1,0,0) ₁₂ ; Q(24)=18.71; P=0.768				
Women						
15-39 years	58					
Impact		-0.081	-7.8	-14.7	-0.4	-0.6
Noise		ARIMA (0,0,0)(0,1,2) ₁₂ ; Q(24)=28.50; P=0.239				
40-49 years	90					
Impact		-0.055	-5.4	-32.6	32.8	-1.3
Noise		ARIMA (0,1,1)(1,0,0) ₁₂ ; Q(24)=17.99; P=0.804				
50-69 years	120					
Impact		0.204	22.6	12.1	34.1	4.0
Noise		ARIMA (0,0,1)(0,1,1) ₁₂ ; Q(24)=21.49; P=0.610				
>69 years	16					
Impact		0.209	23.3	-5.5	60.7	1.0
Noise		ARIMA (1,0,0); Q(24)=20.57; P=0.664				

CI = confidence interval.

^a Derived from exp(estimate).

^b Obtained by multiplying the hospitalisation rate before the change by the percentage change.

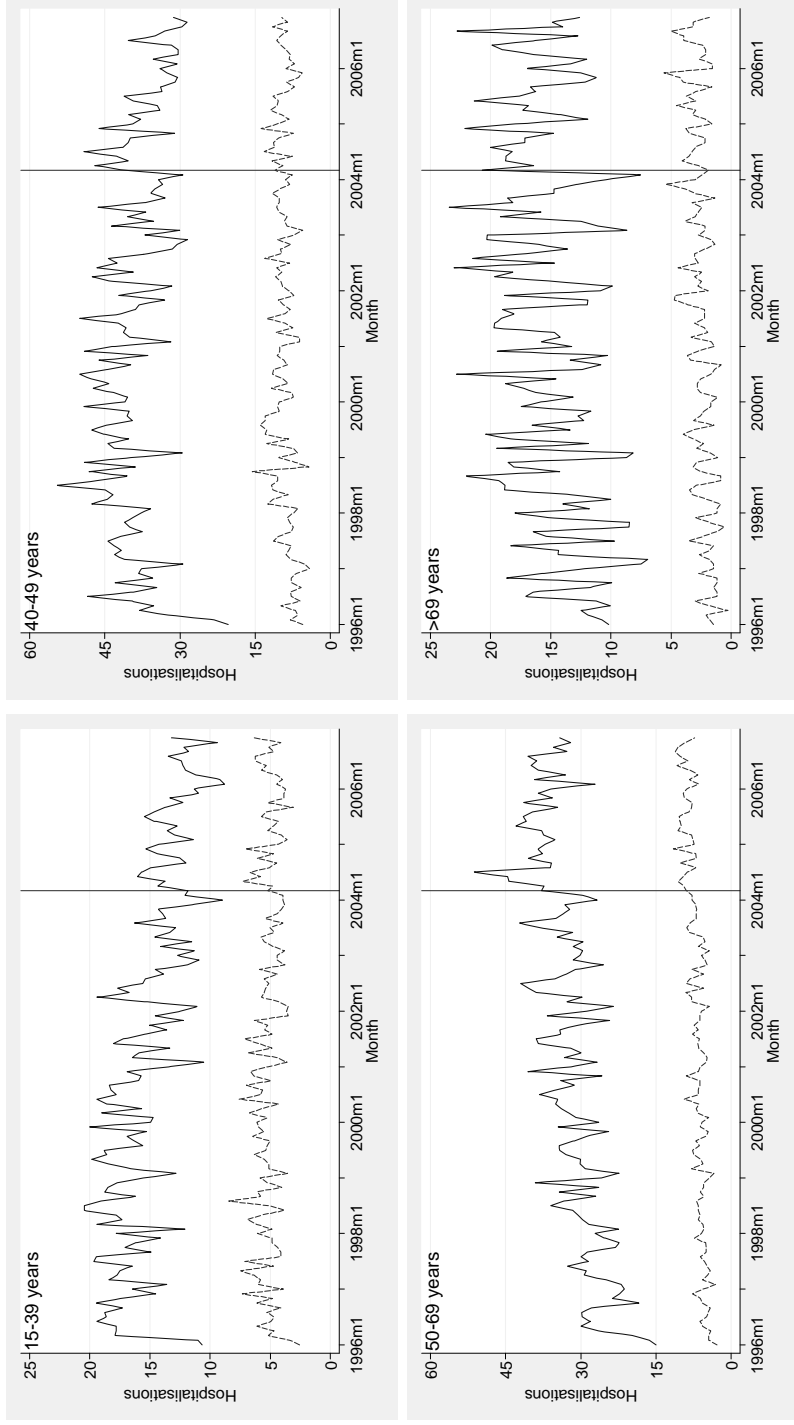


Figure 2 Monthly hospitalisation rates related to acute alcohol causes per 100 000 person-years among men (solid line) and women (dashed line) aged 15-39 years, 40-49 years, 50-69 years, and >69 years, Finland, 1996-2006. The vertical line indicates the realisation of the reduction in alcohol prices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

Table 2 The effects of the reduction in alcohol prices on hospitalisations related to acute alcohol-attributable causes by sex and age, natural logarithmic ARIMA models.

	Hospitalisations per month	Change			Hospitalisations per 100 000 ^b	
		Estimate % in level ^a	Lower CI	Upper CI		
Men						
15-39 years	130					
Impact		0.097	10.2	-1.3	24.1	1.6
Noise		ARIMA (0,1,1)(1,1,0) ₁₂ ; Q(24)=21.46; P=0.612				
40-49 years	156					
Impact		0.161	17.4	6.2	29.9	7.0
Noise		ARIMA (0,1,1)(1,1,0) ₁₂ ; Q(24)=18.24; P=0.791				
50-69 years	196					
Impact		0.186	20.4	5.3	37.7	6.2
Noise		ARIMA (0,1,1)(1,1,0) ₁₂ ; Q(24)=23.04; P=0.517				
>69 years	30					
Impact		0.141	15.2	-3.5	37.5	2.3
Noise		ARIMA (0,0,1)(1,0,0) ₁₂ ; Q(24)=23.41; P=0.496				
Women						
15-39 years	43					
Impact		0.058	6.0	-6.0	19.5	0.3
Noise		ARIMA (0,0,0)(1,1,0) ₁₂ ; Q(24)=21.53; P=0.608				
40-49 years	36					
Impact		0.059	6.0	-9.2	23.9	0.6
Noise		ARIMA (1,0,0)(1,0,0) ₁₂ ; Q(24)=19.41; P=0.729				
50-69 years	44					
Impact		0.321	37.9	19.5	59.1	2.3
Noise		ARIMA (0,0,1)(0,1,1) ₁₂ ; Q(24)=19.37; P=0.732				
>69 years	9					
Impact		0.019	1.9	-30.3	49.0	0.0
Noise		ARIMA (0,0,1)(2,1,0) ₁₂ ; Q(24)=20.36; P=0.676				

CI = confidence interval. Q(24) is the Portmanteau test for residual autocorrelation with 24 lags.

^a Derived from exp(estimate).

^b Obtained by multiplying the hospitalisation rate before the change by the percentage change.