Jussi Puljula

ALCOHOL-RELATED TRAUMATIC BRAIN INJURIES BEFORE AND AFTER THE REDUCTION OF ALCOHOL PRICES

OBSERVATIONS FROM OULU PROVINCE AND NORTHERN OSTROBOTNIA
JUSSI PULJULA

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Observations from Oulu Province and Northern Ostrobothnia

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University of Oulu Graduate School; University of Oulu, Faculty of Medicine, Institute of Clinical Medicine, Department of Neurology, P.O. Box 5000, FI-90014 University of Oulu, Finland; National Graduate School of Clinical Investigation, Biomedicum Helsinki, Haartmaninkatu 8, P.O. Box 700, FI-00029 HUS, Finland
Oulu, Finland

Abstract

Traumatic brain injury (TBI) is an enormous health and economic problem. The proportion of alcohol involvement among subjects with TBI varies 34–51%. Acute alcohol intoxication increases the risk for head trauma compared with other parts of the body. Therefore, alcohol is a major, yet preventable risk factor for TBI. Alcohol taxes were reduced in 2004 and limits on tax-free imports from other EU countries were also removed. Within a year, total alcohol consumption increased by 10% in Finland. Alcohol-induced liver diseases and sudden deaths involving alcohol increased after the reduction. The effects of the reduction on the incidence and mortality of TBI were not known.

Data on all TBI subjects admitted to Oulu University Hospital (1999, 2007) were gathered, as well as data on all fatal TBIs among the residents of Oulu Province (1999, 2006, and 2007). A cohort of subjects with head trauma admitted to Oulu University Hospital in 1999 was followed up until the end of 2009. Alcohol involvement was based on positive alcohol measurement or alcohol-related cause of death on death certificate or hospital chart notes made by health care providers. The incidence and mortality rates of moderate-to-severe TBI and alcohol involvement between the observation years were compared. Cumulative survival rates were calculated to demonstrate the effect of alcohol price reduction on the mortality of the head trauma cohort during a 10-year follow-up.

The incidence of moderate-to-severe and fatal TBI was similar before and after the reduction of alcohol prices. After the reduction, the proportion of alcohol-related moderate-to-severe and fatal TBIs increased among middle-aged people, but decreased among young adults. The increase in TBIs among the middle-aged was mainly due to an increase in falls, whereas the decrease among young adults was due to a decreased number of suicides (particularly among young men). After the reduction of alcohol prices, harmful drinkers were significantly more likely to die than those who were not harmful drinkers.

The overall incidence of moderate-to-severe and fatal TBIs did not increase after the reduction of alcohol prices, but the proportions of alcohol-related TBIs due to falls increased among middle-aged people. Our observations demonstrate that harmful drinkers and middle-aged people were the groups which suffered the most TBI-related harm after the reduction of alcohol prices.

Keywords: alcohol drinking, alcohol price reduction, epidemiology, incidence, mortality, traumatic brain injury
Puljula, Jussi, Alkoholiin liittyvät traumautiset aivovammat ennen ja jälkeen alkoholin veronalennuksen. Havaintoja Oulun läänin ja Pohjois-Pohjanmaan alueelta

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Oulu

Tiivistelmä


Lievää vaikeampien aivovammojen ilmaantuvuus sekä alkohollin liukkaus on pysyviä samansuuruissa ennen ja jälkeen alkoholiin veronalennuksen. Veronalennuksen jälkeen alkoholiin liittyvien aivovammojen osuus lisääntyi keski-ikäisillä mutta vähäni nuorilla. Keski-ikäisillä havaittiin alkoholiin liittyvien aivovammojen lisääntyminen johtui pääasiallisesti sitä että kaatumisvammoja tapahti enemmän, kun taas nuorten kohdalla havaittiin vähenneminen johtui itsenemisen lisäksi rajat, jotka eivät käyttäneet alkoholia halullisesti.


Asiasanat: alkoholi, alkoholin veronalennus, epidemiologia, ilmaantuvuus, kuolleisuus, traumaattinen aivovamma
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Rovaniemi, September

Jussi Puljula
Abbreviations

BAC  blood alcohol concentration
DAI  diffuse axonal injury
DALY disability-adjusted life year
ER  emergency room
GCS  Glasgow Coma Scale
ICD  International Classification of Diseases
NHD  National Hospital Discharge Registry
PTA  post-traumatic amnesia
SFCD  Statistics Finland Causes-of-Death Registry
TBI  traumatic brain injury
List of Original Publications

This thesis is based on the following articles, which are referred to in the text by their Roman numerals.


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1 Introduction

Traumatic brain injury (TBI) is an enormous health problem around the world. It has been estimated that 10 million TBIs leading to hospitalisation or death occur annually (Hyder et al. 2007). TBI is one of the leading causes of death and permanent disability among young people (Bruns & Hauser 2003, Jennett 1996). In the US, 1.7 million TBIs occur annually, 1.365 million subjects are treated and discharged from emergency rooms (ERs), 275,000 subjects are hospitalised and 52,000 subjects die due to TBI (Faul et al. 2010). Besides the burden of individual health problems caused by mortality, morbidity and permanent disability, the economic impact on society is significant. Every year 3.7 million Europeans sustain TBI, and direct and indirect total costs exceed 33,000 million euros (Olesen et al. 2012).

The most significant and modifiable risk factor for TBI is alcohol drinking. Previous studies have reported that 34–51% of subjects with TBI are under the influence of alcohol upon admission to hospital (Corrigan 1995, Dikmen et al. 1995, Gurney et al. 1992, Vickery et al. 2008). Studies in Finland have observed alcohol involvement varying from 47% to 65% in subjects with head trauma (Honkanen & Visuri 1976, Savola et al. 2005). These studies show that acute intoxication increases the risk for head trauma compared with other parts of the body. It has also been shown that the risk for head trauma compared with other parts of the body increases with the degree of intoxication (Savola et al. 2005). Therefore, both irregular and regular heavy alcohol drinking are major risk factors for TBI.

The burden of alcohol-related diseases has been observed to be closely linked to per capita alcohol consumption in a defined population (Rehm et al. 2009). In Finland, annual total per capita alcohol consumption increased from 3.1 litres in 1965 to 10.0 litres in 2010 (National Institute for Health and Welfare 2011). There have been two major liberalisation events in alcohol policy in the past four decades which have significantly affected total per capita alcohol consumption in Finland. First, new alcohol legislation which liberated the sale of medium-strength beer in grocery stores and cafeterias was instituted in the beginning of 1969. Second, a major reduction of alcohol taxes was carried out in 2004 and limits on tax-free imports from other EU countries were also removed that same year (Mäkelä & Österberg 2009). Within a year, total alcohol consumption increased by 10% in Finland (National Institute for Health and Welfare 2011).
Some consequences of this increase in alcohol consumption for public health have been analysed. Sudden deaths involving alcohol as well as deaths from alcohol-induced liver disease increased significantly in Finland after the alcohol tax cut (Koski et al. 2007, Mäkelä & Österberg 2009). Total alcohol-related mortality increased in Finland, mainly due to liver disease, and it increased more among women (31%) than men (16%) (Herttua et al. 2008). Hospitalisation due to alcohol-related diseases has also increased significantly after the reduction of alcohol prices, mainly among subjects aged 50–69 (Herttua et al. 2011). However, studies reporting how changes in alcohol consumption affect occurrence of TBI are still lacking.

This study investigated recent trends in the epidemiology of TBI among people living in Oulu Province and Northern Ostrobothnia. The main focus was to determine temporal associations between alcohol consumption and TBI and to investigate the effects of the large reduction of alcohol prices on the number of TBIs.
2 Review of the literature

2.1 Traumatic brain injury (TBI)

2.1.1 Definition of TBI

TBI is defined as an alteration in brain function, or other evidence of brain pathology, caused by an external force (Menon et al. 2010). A Finnish guideline defines TBI as a verified head trauma resulting from one or more of the following: loss of consciousness (even short), dysfunction of memory regarding events before or after the trauma, any alteration in mental functions (disorientation, confusion), transient or permanent focal neurological deficit or an abnormal brain CT/MRI finding due to trauma (Aikuisiän aivovammojen käypää hoito 2008). The term head injury/trauma is frequently used in literature and usually these terms mean the same as brain injury. However, the term head injury or head trauma can also include subjects without brain injury, i.e. facial fractures or wounds/lacerations of the head.

2.1.2 Classification of TBI

TBIs have been classified in many different ways, depending on available data and the purpose of the classification (Saatman et al. 2008). In clinical practice, classification provides information on the localisation and anatomic features of TBIs. TBIs can be divided into four main categories: contusion, intracranial haematomas, subarachnoid haemorrhage and diffuse axonal injury (Saatman et al. 2008).

Cerebral cortical contusion is the hallmark for brain injury and can occur as haemorrhagic lesions, (Adams et al. 1980), ischemia or brain oedema (Bullock et al. 1991, Gaetz 2004). Macroscopic findings include areas of superficial damage consisting of streaks or groups of punctuate haemorrhages accompanied by variable amounts of necrosis or areas of necrosis without haemorrhage (Strich 1970). Intracranial haematomas can be divided into epidural, subdural and intracerebral. An epidural haematoma is located between the dura and the skull. An epidural haematoma is frequently caused by tearing of branches of the middle meningeal artery, and is sometimes caused by tearing of the dural sinuses or transdural veins (Simpson 1949). Subdural haematomas occur when bridging
veins of the dura or possibly cortical arteries rupture and blood extends to the
cortex under the dura (Gaetz 2004). Intracerebral haematoma is caused by a direct
rupture of a blood vessel inside the brain parenchyma (Gaetz 2004). Traumatic
subarachnoid haemorrhage occurs when a blood vessel ruptures inside the
subarachnoid space. Usually, diffuse brain injury is suspected when intracranial
haematomas or contusions are not found. Diffuse brain damage consists of axonal
injury, hypoxic brain damage, brain swelling and vascular injury (Adams et al.
1991). An early study showed diffuse degeneration of cerebral white matter in
severe dementia following head injury without fractures of the skull, intracranial
haematoma or lacerations of the brain (Strich 1956). The term diffuse axonal
injury (DAI) is used to describe this type of brain damage (Adams et al. 1991).
DAI, contusions, intracranial haematomas and subarachnoid haemorrhages
frequently occur simultaneously, and a skull/facial bone fracture may also be
present.

Classification according to physical mechanism is also possible. TBI can be
classified according to whether the brain is injured due to a direct strike to the
head or due to brain movement inside the skull, for example acceleration/deceleration (Saatman et al. 2008).

The International Classification of Diseases (ICD) has also been used for TBI
classification. ICD codes specify locations of injury, but the severity of injury
remains difficult to identify reliably from ICD codes (Jennett 1996). However,
ICD codes have frequently been used especially in epidemiological studies
(Carroll et al. 2004), despite the criticism of its poor detection of TBI (Deb 1999,
Leibson et al. 2011).

### 2.1.3 Severity of TBI

The most general classification of TBI is based on severity. Severity is divided
into mild, moderate and severe TBI (Vos et al. 2002). Severity can be defined by
the Glasgow Coma Scale (GCS) score, duration of unconsciousness, duration of
amnesia, abnormality in a brain CT/MRI, focal neurological symptoms or a
combination of these. The GCS score—introduced by Teasdale and Jennett in
1974—is an internationally used aid in clinical assessment of the level of
consciousness; see Table 1 (Teasdale & Jennett 1974).
Table 1. Glasgow Coma Scale (GCS).

<table>
<thead>
<tr>
<th>Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To speech/voice</td>
<td>3</td>
</tr>
<tr>
<td>To pain stimulus</td>
<td>2</td>
</tr>
<tr>
<td>No response at all</td>
<td>1</td>
</tr>
<tr>
<td>Motor</td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Localises to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdrawal to pain</td>
<td>4</td>
</tr>
<tr>
<td>Decorticate posture</td>
<td>3</td>
</tr>
<tr>
<td>Decerebrate posture</td>
<td>2</td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Verbal</td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused conversation</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensive speech</td>
<td>2</td>
</tr>
<tr>
<td>No response</td>
<td>1</td>
</tr>
</tbody>
</table>

Mild TBI

The definition of mild TBI varies considerably in different guidelines (Aikuisiän aivovammojen käypä hoito 2008, Batchelor & McGuiness 2002, Borg et al. 2004, Ingebrigtsen et al. 2000, National Center for Injury Prevention and Control 2003, Vos et al. 2002). The WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury recommends the following criteria for mild TBI: 1) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for health care; 2) one or more of the following were present: confusion or disorientation, loss of consciousness for up to 30 minutes, post-traumatic amnesia for less than 24 hours and/or other transient neurological abnormalities such as focal neurologic deficits, seizure and intracranial lesions not requiring surgery. These symptoms must not be due to drugs, alcohol or medications nor caused by other injuries or treatment for other injuries (Carroll et al. 2004).
Moderate-to-severe TBI

TBI can be defined as moderate by either duration of amnesia or GCS score or a combination of these. An early study suggests that TBI is moderate if the duration of posttraumatic amnesia ranges from 1 to 24 hours, severe if amnesia ranges from 1 to 7 days and very severe if amnesia lasts more than 7 days (Russell & Smith 1961). The European Federation of Neurological Societies guideline defines TBI as moderate with a GCS score of 9–12 and severe with a GCS score ≤ 8 (Vos et al. 2002). The Finnish Adult TBI guideline defines TBI as moderate with one or more of the following: 1) a GCS score of 9–12; 2) posttraumatic amnesia lasting from one day to seven days 3) an intracranial finding in a brain CT/MRI due to trauma not requiring surgery. Accordingly, TBI is severe if the GCS score is ≤ 8, posttraumatic amnesia lasts more than seven days or an intracranial brain CT/MRI finding requires neurosurgical operation (Aikuisiän aivovammojen käypä hoito 2008).

2.1.4 Epidemiology of TBI

The incidence of TBI varies widely in different parts of the world and its overall global incidence is not known. Worldwide incidence is hard to calculate, because subjects with mild TBI do not necessarily seek help, injury surveillance and reporting systems are different in different parts of the world, TBI might be underestimated in the context of multiple injuries and definitions of TBI vary widely (Hyder et al. 2007). Approximately 10 million TBIs leading to either death or hospitalisation occur annually (Hyder et al. 2007). This estimation does not represent overall incidence, because large proportions of TBI subjects are not hospitalised. A recent study from the US reported that 1.7 million subjects suffer TBI annually, 80.7% are treated at and discharged from emergency rooms, 16.3% are hospitalised and 3.0% die. The amount of people who get TBIs and don’t seek help is unclear (Faul et al. 2010). The reported incidences in Europe vary from 100 to 300/100,000 (Tagliaferri et al. 2006). However, greater overall rates of TBI were found in the United Kingdom (Yates et al. 2006) and the US (Leibson et al. 2011). A population-based study from western Sweden that includes data from a hospital’s emergency room, its discharge register, a regional neurosurgical clinic and a coroner’s record found overall TBI incidence to be 546/100,000 (Andersson et al. 2003). Two studies from Finland have reported annual incidences of TBI. A national register-based study reported overall incidence of 102/100,000, but this
study excluded subjects who succumbed to TBI before admission to hospital (Alaranta et al. 2002). A recent study investigated a defined population from Southeast Finland. It included all health centres and regional hospitals, and reported a crude annual incidence rate of 221/100,000 if all symptoms of brain injury after an external force to the head were taken into account (including dizziness, headache and nausea). However, if strict EFNS criteria for TBI (excluding nausea, headache and dizziness) were used, overall incidence decreased to 137/100,000 (Numminen 2011).

Age

A review of the epidemiology of TBI showed that admittance rates to hospital because of TBI are highest among subjects aged less than 30. Age-specific mortality rates from TBI peaks in subjects aged around 20 and over 70 (Jennett 1996). Population-based studies from the Nordic countries have confirmed these findings. The number of TBI patients was highest among subjects aged less than 30 (Andersson et al. 2003, Styrke et al. 2007). However, incidence rates per 100,000 were highest among subjects aged less than 30 and also over 70, especially among men (Styrke et al. 2007). A population-based study from Finland found that incidence rates were evenly distributed among subjects between the ages of 15–65, but began to significantly increase after the age of 65 years (Numminen 2011).

Sex

Age-specific admission and mortality rates are higher among men than women (Jennett 1996). A population-based study from Norway including hospital-treated TBI subjects observed that TBI incidence was higher among men than women (ratio 1.8:1) (Andelic et al. 2008). Incidence was higher among men, particularly among subjects aged 10–30 and over 60. A study from New Zealand observed that the incidence of hospitalised TBI was higher among men in every age group until the age of 75. After that incidence rates were evenly distributed by sex. The most significant sex difference in incidence was found at the age of 15–20 (Barker-Collo et al. 2009). In this age group men were particularly prone to TBI. According to a review of epidemiological TBI studies from Europe, the incidence rate is higher among men than among women. The incidence ratio between men
and women varied from 1.5 to 3:1 (Tagliaferri et al. 2006). Men in the US were approximately four times as likely as women to get a fatal TBI (Adekoya & Majumder 2004). In Finland, also, the incidence of TBI is higher among men. Particularly, the incidence of TBI among men aged over 75 is high in comparison with women (Numminen 2011).

2.1.5 External causes of TBI

Falls

The leading cause of TBI is falling. A recent study from the US reported that the annual average percentage of fall-related TBIs (including hospitalised subjects, ER visits and deaths) was 35.2% of all TBIs (Faul et al. 2010). However, fall-related TBI deaths accounted for only 16.7% of all TBI deaths (Coronado et al. 2011). In Finland, falls contributed 63% of all external causes of TBI among subjects who were hospitalised in 1991–2000 (Alaranta et al. 2002). The proportion of subjects with TBI caused by falling was 58% in a defined adult population from Southeast Finland (Numminen 2011). However, the mortality rate from fall-related TBI was not reported. Incidence rates of fall-related TBIs are highest among the elderly and children (Faul et al. 2010, Numminen 2011). Fall-related TBIs contributed to 50.3% of all unintentional fall-related deaths among subjects aged ≥ 65 in the US (Thomas et al. 2008).

Traffic

Traffic-related TBI percentages vary from 16% to 60% (Andersson et al. 2003, Faul et al. 2010, Murray et al. 1999, Styrke et al. 2007, Tagliaferri et al. 2006). Comparison between studies is difficult, because depending on the study, traffic as a cause of TBI could include/exclude bicycle collisions as well as pedestrian and other non-motor-vehicle collisions. In Finland, traffic accounts for 18–23% of all TBIs (Alaranta et al. 2002, Numminen 2011), about the same proportion as in the US (Faul et al. 2010). Mortality rates are not known in Finland, but in the US traffic collisions accounted for 31.4% of all TBI deaths during 1997–2007 (Coronado et al. 2011) and 34% during 1989–1998 (Adekoya & Majumder 2004).
**Intentional**

Intentional causes of trauma include suicides, suicide attempts, assaults, gunshots/firearms and other interpersonal violence. Intentional causes account for 6–28% (Faul et al. 2010, Leibson et al. 2011, Murray et al. 1999, Thornhill et al. 2000) of all TBIs. A study from the US reported firearm-related injuries (39%) being the leading cause of fatal TBI (Adekoya & Majumder 2004). In Finland, intentional injuries comprise only 4–8% of all TBIs (Alaranta et al. 2002, Numminen 2011). Intentional injuries are most common among young men.

**Sports**

The incidence of sports- and recreation-related TBIs in Finland is not known. However, a systematic review of TBIs in Europe reported that sports-related TBIs vary between 1% and 7% of all TBIs (Tagliaferri et al. 2006). In the US, annually 207,830 emergency room visits are due to sports- and recreation-related TBIs, as was reported to be the case during 2001–2005 (Centers for Disease Control and Prevention (CDC) 2007). It has been estimated that altogether 1.365 million subjects with TBI are treated and released annually from emergency rooms in the US (Faul et al. 2010). Approximately 15% of all TBIs occur due to sports and recreational activities. Another population-based study from the US reported 25% of all TBIs being due to sports (Leibson et al. 2011). Subjects aged 10–19 account for 70.5% of all sports- and recreation-related TBIs (Centers for Disease Control and Prevention 2011).

**2.1.6 Diagnosis of TBI**

Diagnosis of TBI is based on clinical examination, careful assessment of the length of amnesia, the duration and level of consciousness and brain CT/MRI findings (Borg et al. 2004, Vos et al. 2002, Vos et al. 2012). GCS score is a widely used tool for assessing impaired consciousness and coma (Teasdale & Jennett 1974), especially in severe TBIs. The relationship between GCS score and outcome has been reported. Subjects with a higher GCS score have been more likely to have a favourable outcome (Kim 2011, Labi et al. 2003, van der Naalt 2001). Admission GCS score may predict intracranial abnormalities and a need for neurosurgical intervention in subjects with TBI. Intracranial findings and a
need for neurosurgical intervention increased with a lower GCS score (Smits et al. 2007, Stiell et al. 2001). A history of altered consciousness or loss of consciousness (even short) seems to increase the risk for an intracranial finding and/or cranial fracture (Moran et al. 1994, Teasdale et al. 1990). However, the correlation between loss of consciousness (without an intracranial finding) and neuropsychological outcome remains controversial. A few studies suggest no relationship between loss of consciousness (LOC) and neuropsychological outcome (Hanlon et al. 1999, Lovell et al. 1999), but several studies report a chronic neurological and neuropsychological deficit after repetitive TBI (usually sports-related mild TBIs) (Gavett et al. 2011, McCrory 2011, Rabadi & Jordan 2001). Post-traumatic amnesia (PTA) is frequently caused by TBI. It has been suggested that the duration of post-traumatic amnesia predicts outcome better than GCS score in subjects with mild-to-moderate TBI (van der Naalt et al. 1999). In clinical practice, GCS score, duration of unconsciousness and PTA are usually evaluated, and both TBI severity and outcome are based on all of them.

2.2 Alcohol

2.2.1 Contribution of alcohol to harm

It is well known that alcohol causes major health problems around the world (World Health Organization (WHO) 2011). At the population level, the amount of harm due to alcohol depends on the total amount of alcohol consumed, but is also influenced by prevailing drinking patterns (Babor et al. 2010). Drinking pattern has been defined as a sequence of drinking behaviour that includes the kind and amount of alcoholic beverage consumed as well as the frequency, timing and context of drinking occasions (Babor et al. 2010). Total alcohol consumption and drinking patterns vary between different countries, but also in different subgroups inside the population. Total alcohol consumption consists of drinking occasions and also the volume consumed per occasion. Babor et al. define the different hazardous drinking patterns as follows (Babor et al. 2010): 1) Binge drinking is a pattern of heavy drinking that occurs over an extended period of time. In earlier population surveys this period was usually defined as more than one day of drinking at a time. More recently the term has been applied to drinking by young adults and has been defined by the number of alcoholic drinks (usually five or six) consumed on a single occasion. Sometimes the term episodic heavy drinking has
been used as a synonym for binge drinking. 2) Alcoholism is term traditionally used to identify chronic excessive drinking by individuals who are physically dependent on alcohol. Alcohol dependence syndrome is a term used in psychiatric diagnostic classifications to identify the co-occurrence of at least three of six alcohol-related symptoms associated with dependence on alcohol (increased tolerance, withdrawal signs, continued drinking despite harmful consequences, preoccupation with alcohol, impaired control over drinking and alcohol craving). Alcohol intoxication is a result of binge/episodic heavy drinking. 3) Alcohol intoxication has been defined as a more or less short-term state of functional impairment in psychological and psychomotor performance induced by the presence of alcohol in the body.

Binge drinking is a more popular drinking pattern in Finland and other Scandinavian countries than it is in Mediterranean countries. A European telephone survey including people aged 18–64 revealed that in Finland, 29% of all drinking occasions during the last 12 months were binge drinking occasions (at least a bottle of wine, 25 centilitres of spirits or four cans of beer) among men and 17% among women (Norström 2002). A survey of Finnish drinking habits from 2008 found that 31% of men reported being at least a little bit intoxicated at least once a week, suggesting that binge drinking is a popular drinking pattern. The corresponding figure for women was 12% (Huhtanen et al. 2011). The proportion of alcohol intoxication occasions (estimated alcohol content over 1 per mille of blood) of all drinking occasions was 22% among men and 14% among women (Mustonen et al. 2009). Savola et al. found that episodic heavy drinking is the most popular drinking pattern in subjects with trauma admitted to the emergency room of a university hospital in 1998–2000. Two hundred and seventy-three subjects out of 349 (78%) reported heavy drinking either regularly or irregularly (Savola et al. 2005).

Regular daily drinking causes different health problems than binge drinking. However, the effects can have interrelationships, as illustrated in Fig 1. It is sometimes difficult to distinguish between episodic heavy drinking and dependent drinking. These drinking patterns do not exclude each other.
Alcohol is a major risk factor for a wide range of diseases and injuries. A recent study estimated that 3.8% of all global deaths and 4.6% of global disability-adjusted life-years (DALY) were caused by alcohol (Rehm et al. 2009). Approximately 2.2 million alcohol-attributable deaths occur annually. Deaths due to alcohol are common in cardiovascular diseases (22% of all alcohol-attributable deaths), cancer (19.6%) and cirrhosis of the liver (15%). However, the most common cause of alcohol-attributable deaths are injuries (37.8% of all alcohol-attributable deaths)—26.8% from unintentional and 11% from intentional injuries. The disease burden of alcohol has also been estimated with the disability-adjusted life-years (DALY) measure. DALY measures years of life lost due to premature death together with life lost due to disability. It has been estimated that 4.6% of DALYs have been alcohol-attributable. The highest burdens of alcohol-attributable diseases are caused by neuropsychiatric disorders (36.4% of all alcohol-attributable DALYs), unintentional injuries (25.4%), intentional injuries (10.5%), cirrhosis of the liver (9.5%), cardiovascular diseases (9.5%) and cancer (8.6%) (Rehm et al. 2009).
It is well shown that alcohol is a major risk factor for deaths and diseases. Several studies have estimated the causality between alcohol consumption and mortality in a defined population. A large case-control study from Russia found that 52% of all deaths (among people aged 15–54) were alcohol-attributable—59% of all deaths among men and 33% among women (Zaridze et al. 2009). Preceding alcohol use and drinking pattern were clarified from family members. The association between preceding drinking pattern and risk for alcohol-related death was calculated. The highest risks for alcohol-related deaths were caused by accidents and violence (RR 5.94, 95% CI 5.35–6.59 among those who consumed > 3 bottles of vodka per week), alcohol poisoning (RR 21.68, 95% CI 17.94–26.20) and acute ischemic heart disease other than myocardial infarction (RR 3.04, 95% CI 2.73–3.39). An excessive risk for alcohol-related death was also observed in digestive tract cancer, liver cancer, tuberculosis, pneumonia, liver diseases, pancreatic diseases and illness-specific conditions. Alcohol poisoning, accidents, violence and cardiovascular mortality peaked on weekends and Mondays in Russia, and this was suggested to be due to binge drinking, which is a typical drinking pattern in Russia (Chenet et al. 1998).

A Finnish death-register-based study including subjects aged 15–89 found that alcohol-related deaths accounted for 6% of all deaths—11% among men and 2% among women (Mäkelä 1998). Among deceased subjects aged 15–49, the alcohol-related proportion was over 40% in men and 15% in women. Another study from Finland reported the influence of alcohol on mortality among the population aged over 20 (Poikolainen et al. 2008). The percentage of alcohol-attributable deaths out of all deaths was 9.4% in men, the highest proportion in deaths being due to accidents and poisoning (2.9%), cirrhosis of the liver (2.2%) and cardiovascular diseases (2.0%). Overall, 2232 deaths among men were attributable to alcohol in 2002. In women the net effect of alcohol was protective (-2.3%), due to the protective effect against cardiovascular death. It was estimated that alcohol contributed to 724 deaths, but protected against 1311 deaths among women. When the analysis was performed among only people aged 20–64, the proportion of alcohol-attributable deaths was 22.5% in men and 11.2% in women (Poikolainen et al. 2008).

The proportion of alcohol-attributable deaths has been estimated to be 6.1% of all deaths in Spain (Yanez et al. 1993) and 3.5% of all deaths in Sweden, although the percentage is much higher (25%) among people aged < 50 (Sjögren et al. 2000). Some protective effects of alcohol consumption have been observed
in cardiovascular diseases (O’Keefe et al. 2007, Rehm et al. 2003, Rehm et al. 2009). Cardiovascular benefits are associated with light regular drinking, but not with regular or irregular heavy drinking.

It is essential to clarify how untoward effects of alcohol are associated with drinking pattern. In a Finnish study including subjects aged 15–69, distributions of alcohol-related harms were compared between the 10% of the population with the highest average alcohol consumption and the remaining 90% of drinkers. The study revealed that 70% of self-reported harms, 70% of alcohol-related hospitalisations, 64% of alcohol-related deaths and 64% of premature life-years lost before the age of 65 occurred among the 90% of drinking men who were not among the highest consumers. The corresponding figures for women were 64%, 60%, 93% and 98% (Poikolainen et al. 2007). Accordingly, although a great proportion of alcohol-related harm is carried by the 10% of the population with the highest average alcohol consumption, most of the harm is still carried by those who consume less. Those with the highest average alcohol consumption are mainly alcoholics and dependent on alcohol. The burden of alcohol-related harms is not restricted to alcoholics. Light and moderate drinkers may also sometimes engage in irregular heavy drinking. These occasions give rise to acute problems such as violence, accidents and injuries. An Australian study compared harm caused by chronic alcohol-related conditions with harm caused by acute alcohol-related conditions (Chikritzhs et al. 2001). Usually, chronic alcohol-related diseases such as alcoholic cirrhosis or digestive tract cancers require long-term daily alcohol consumption, and acute alcohol-related harms such as accidents and traumas tend to arise during binge drinking occasions. The Australian study observed that 42% of all alcohol-related deaths were caused by a chronic condition, 28% by an acute condition and 30% by mixed conditions. Estimation of person-years of life lost revealed that acute conditions accounted for 46%, chronic conditions for 33% and mixed conditions for 21% (Chikritzhs et al. 2001). The burdens of alcohol-related harms are distributed among both alcoholics and binge drinkers. The most convincing evidence of an association between binge drinking and alcohol-related harm is shown in traumas. The risk of injury is increased by an average daily volume of one drink, but also by drinking five or more drinks on one day more often than twice a year (Cherpitel et al. 1995). Spurling and Vinson found that the population-based proportion of injury among those who had consumed alcohol six hours prior to injury (8.5% to 10.6%) was higher than among those who were dependent on alcohol (4.0%) (Spurling & Vinson 2005). Several other emergency room studies have also found that acute

2.2.2 Definition and content of alcohol policy

Public policy decisions made by governmental or non-governmental authorities which influence alcohol-related harms (both health and social) are defined as alcohol policy (Babor et al. 2010). Alcohol consumption can be controlled by economic regulation, restricted availability, education and less visible marketing of alcohol beverages. Price controls and taxation are the most effective tools which influence alcohol consumption. Limitations can be set on the number of retail outlets, places of selling, trade hours or even days and minimum legal purchase age. Retail sale is monopolised in some parts of the world, like in Finland. Total prohibition was implemented in several countries in the early 1900s. Education and general knowledge about alcohol-related harm can be provided by both schools and the community. Special anti-alcohol campaigns and programmes that reduce alcohol-related harms could be arranged. Marketing of alcoholic beverages can be limited, for example, by restricting the time and place for marketing.

Affordability

As in any other commodities, the affordability of alcohol is determined by price and consumers’ incomes. With higher incomes and lower prices, affordability grows, and vice versa. Different methods have been used to calculate affordability, but the basic elements are income and the cost of alcoholic beverages (Seabrook 2010). The price of alcohol is one of the most effective tools for controlling alcohol consumption at the population level. Fundamental economic theory states that demand by the population and supply will set the price of a product. Therefore, an increase in the price of a product will decrease its demand, assuming that supply stays at the same level. There is strong evidence that an increase in alcohol price will result in a decline in consumption and that a decrease in price will result in an increase in consumption (Chaloupka et al. 2002, Cook et al. 2011, Edwards et al. 1994, Mäkelä & Österberg 2009, Stockwell et al. 2011). Therefore, the price of alcohol is the key element for controlling
consumption and taxation is the most widely used method for controlling the price of alcohol.

2.2.3 Effects of alcohol price on alcohol consumption

The effects of alcohol price policy can be measured by changes in total alcohol consumption. Economic elasticity is a measure which estimates how sensitive a product’s demand is to changes in price. A recent meta-analysis of 112 different studies summed up the effects of alcohol price or tax changes on alcohol sales and self-reported drinking. The study revealed that simple means of reported elasticity are -0.51 for total alcohol, -0.46 for beer, -0.69 for wine and -0.80 for spirits (Wagenaar et al. 2009). This means that a 1% increase in price results in a 0.51% decrease in total alcohol consumption and correspondingly, a 0.46% decrease in beer consumption, 0.69% in wine and 0.80% in spirits. Although the relationship between alcohol price and consumption varies around the world, the main result is that the price of alcohol is an effective tool for limiting total alcohol consumption (Chaloupka et al. 2002, Wagenaar et al. 2009).

2.2.4 Alcohol policy in Finland

Restrictions on availability, strict state monopolisation of alcohol sale, trade and production and high prices have been the basic elements of alcohol policy in Finland. Total prohibition was ratified in 1919 and repealed in 1932. After the repeal of prohibition the State Alcohol Monopoly (Oy Alkoholiliike Ab, ALKO) was established to control production, distribution and sales (Sariola 1954). There have been two major liberalisations of alcohol policy during the past four decades. First, new alcohol legislation instituted in the beginning of 1969 liberated the sale of medium-strong beer in grocery stores and cafeterias. This new law also allowed ALKO to establish retail shops in rural areas and lowered the legal age for purchasing alcoholic beverages with alcohol content under 22‰ from 21 to 18. Second, a major reduction of alcohol taxes was carried out in 2004 and limits on tax-free imports from other EU countries were removed that same year (Mäkelä & Österberg 2009). Alcohol taxes on spirits (44%), fortified wine (40%), table wine (10%) and beer (32%) were lowered on 1 March 2004 (Mäkelä et al. 2008) and within-EU traveller’s allowances came into effect on 1 May for travellers from Estonia, where alcohol was much cheaper than in Finland. These
and other minor changes resulted in changes in total alcohol consumption, illustrated in Figure 2.

![Graph showing alcohol consumption trends](image)

**Fig. 2. Total alcohol consumption (litres of 100% alcohol per capita) during 1965–2009.**

Alcohol data source: THL, www.sotkanet.fi

### 2.2.5 Effects of the alcohol tax cut of 2004 on health

The following chapter reviews current literature on alcohol-related health problems caused by the 2004 tax reduction in Finland. It has been suggested that expensive alcohol is associated with less alcohol-related harm (Anderson et al. 2009, Purshouse et al. 2010). On the other hand, reduction of alcohol price would increase alcohol-related harms. However, the increase in alcohol consumption after the alcohol tax reduction is not necessarily distributed equally among the population. For example, poor people may consume more if the price of alcohol decreases, but wealthy people’s alcohol consumption may not be influenced as much by price.
Morbidity and mortality

The reduction of the alcohol tax in 2004 resulted in an overall increase in alcohol-related mortality. Mortality rates were compared with those observed before (2001–2003) and after (2004–2005) the price reduction in a register-based study of Finns aged ≥ 15 years (Herttua et al. 2008). Men showed a 16% increase in alcohol-related deaths, whereas women showed a 31% increase. These increases were mostly due to alcohol-related chronic diseases, such as alcoholic liver disease, and they occurred particularly among middle-aged and elderly people. Deaths caused by alcoholic liver disease were 46% more frequent in 2004–2006 than in the early 2000s (Mäkelä & Österberg 2009). Deaths due to alcohol poisoning and alcohol dependency also increased more than expected. A slight increase in accidental and violent alcohol-related deaths occurred, especially among women. Time-series analysis from 1990–2004 revealed that alcohol-positive sudden deaths increased 17% within a year after the tax cut (Koski et al. 2007). The total number of alcohol-related hospitalisations (acute and chronic) also increased after the tax cut (Mäkelä & Österberg 2009). Hospitalisations were compared with those observed before (2001–2003) and after (2004–2006) alcohol tax reduction, and this increase was mostly due to liver diseases, alcohol intoxication and alcohol-induced psychotic disorders and it occurred mainly among people older than 45. Increases in hospitalisations were observed among subjects aged 55–64 (25%), ≥ 65 (11%) and those aged 45–54 (9%). Time-series intervention analysis from 1996–2006 revealed that acute alcohol-related hospitalisations (due to alcohol intoxication and alcohol poisoning) increased by 17% and 20% among men aged 40–49 and 50–69 and by 38% among women aged 50–69 after the alcohol tax reduction (Herttua et al. 2011). Chronic alcohol-related hospitalisations (due to alcohol-attributable disease other than intoxication and poisoning) increased by 16% among men aged 15–39, 11% among men aged 40–49 and 22% among men aged 50–69. Chronic hospitalisations increased only among women aged 50–69 (23%).

The increase in alcohol-related liver disease mortality and morbidity indicates that heavy drinkers carried a great burden of the alcohol price reduction. Risk of alcoholic liver diseases increases with the length of drinking history and the volume of daily doses (Lelbach 1975). Usually, several years of daily or almost daily alcohol consumption is needed for development of alcoholic liver disease. An Italian study found that the risk threshold for developing chronic alcoholic liver disease was a daily dose of 30 g of alcohol (one drink contains
approximately 12 g of ethanol), and the risk increased with increasing doses (Bellentani et al. 1997). The rapid increase in alcoholic liver diseases after the reduction of alcohol prices in Finland suggests that subjects with a habit of regular drinking increased their consumption after the tax cut.

A postal survey of randomly selected subjects probed differences in drinking patterns after the 2004 price reduction. The numbers of moderate-to-heavy regular and binge drinkers were observed to have increased among those aged > 44 (Helakorpi et al. 2010). Herttua et al. reported similar findings indicating that most of the increase in alcohol consumption and concomitant harm involved older people—men aged 55–59 and women aged 50–54 (Herttua et al. 2008). Young and employed people suffered less untoward effects of the alcohol tax reduction, and alcohol-related health problems did not increase among them. However, a survey study from Denmark, Norway, Sweden and Finland did not support the hypothesis of a clearer increase in alcohol consumption among heavy drinkers compared with other alcohol consumers after the tax cut (Mäkelä et al. 2008). But, the design was insufficient for gathering reliable data from the whole population and from all subgroups and did not manage to capture the overall increase in consumption in Finland.

The reduction of alcohol prices also influenced the sale of different beverages in Finland. The sale of unflavoured spirits (such as vodka) increased by 21% from 2003 to 2007. The sale of cognac and whisky increased by 28% and even by 69% for the most expensive whisky and cognac. Heavy drinkers usually consume cheaper unflavoured spirits and not the most expensive beverages, such as quality whiskies. These figures suggest that the alcohol tax cut increased alcohol purchasing also by others, not only heavy drinkers (Sandelin 2008).

Studies from Finland investigating the effects of alcohol tax reduction in 2004 report results only on a short-term basis (after two years). The data on long-term effects of alcohol price reduction in different subgroups, also other than heavy drinkers, are still missing. Some light-to-moderate drinkers may also have increased their alcohol consumption and these harms may be seen later on.
2.3 Alcohol and TBI

2.3.1 Effects of alcohol and trauma

Alcohol drinking usually results in acute alcohol intoxication (also called inebriation or drunkenness), and intoxication causes several effects which expose to traumas. According to ICD-10, acute alcohol intoxication is defined as (World Health Organization (WHO) 1993): 1. clear evidence of recent administration of alcohol; 2. evidence of dysfunctional behaviour in at least one of the following: disinhibition, argumentativeness, aggression, lability of mood, impaired attention, impaired judgment or interference with personal functional behaviour; 3. at least one of the following signs: unsteady gait, difficulty in standing, slurred speech, nystagmus, decreased level of consciousness, flushed face or conjunctival injection; 4. not accounted for by a medical disorder unrelated to alcohol use, and not better accounted for by some other mental or behavioural disorder. Most of the effects increase the risk for trauma.

The effects are usually dose-dependent, i.e. they are greater with higher blood alcohol concentrations: the more you drink the more symptoms you will get. However, continuous exposure to alcohol results in development of central nervous system tolerance to alcohol. Occasional drinkers without increased tolerance are more sensitive to the effects of alcohol and feel more inebriated after ingestion of the same amount of alcohol than are regular drinkers. It has been suggested that subjects who usually do not drink are particularly at risk for alcohol-related traumas if they engage in occasional drinking (Gmel et al. 2006). Age, sex and weight also influence the level of intoxication. Alcohol is a water-soluble molecule, and after absorption from the gastrointestinal tract it is rapidly distributed to all water-containing tissues, such as the brain and muscles. Women have a higher body fat content than men (lower percentage of water-rich tissues) and therefore the same amount of alcohol causes higher BAC in women than in men.

Strong associations have been observed between acute intoxication and several types of trauma (Cherpitel et al. 1995, Honkanen et al. 1975, Honkanen & Smith 1991, Mcleod et al. 1999, Nurmi-Lüthje et al. 2007). Some diseases that occur after prolonged heavy drinking may also predispose to traumas, for example, alcoholic cerebellar atrophy. Purkinje cells in the cerebellum are particularly vulnerable to damage due to chronic alcohol consumption (Andersen 2004, Gilman et al. 1990), and atrophy in the cerebellum results in impaired
balance and gait. Chronic long-term heavy alcohol intake also causes peripheral neuropathy, which may cause severe problems in gait and balance and therefore expose subjects to traumas.

2.3.2 Alcohol and the epidemiology of TBI

Alcohol involvement is high in subjects with TBI. A majority of the studies on this have been done in the US. Alcohol involvement has been found in 34% to 48% of subjects with TBI admitted to emergency rooms (Dikmen et al. 1995, Gurney et al. 1992, Vickery et al. 2008). Two systematic reviews found that the proportion of alcohol involvement varies from 36% to 51% (Corrigan 1995, Parry-Jones et al. 2006). A case-control study from the UK reported alcohol involvement in 42% of subjects with TBI (Swan et al. 2004). A recent study from Norway observed alcohol involvement in 39% of subjects with moderate-to-severe TBI (Andelic et al. 2010). In a Swedish study, head traumas resulting in disturbed consciousness were identified and the alcohol involvement rate evaluated by either blood sampling, medical data or self-reporting revealed that only 17% (75/449) were under the influence of alcohol (Styrke et al. 2007). A few studies from Finland have estimated alcohol involvement among subjects with TBI. An early emergency room study from Finland included 1012 trauma subjects, and alcohol involvement was determined from their blood (Honkanen & Visuri 1976). Alcohol involvement varied widely according to the type of trauma. Subjects with head trauma were significantly (p < 0.05) more frequently intoxicated (47%) than subjects with other types of trauma. Alcohol involvement was seen in 38% of subjects with trunk or neck trauma, 25% of subjects with upper extremity trauma and 39% of subjects with lower extremity trauma. A large emergency room register-based study from Finland (Honkanen & Smith 1991) included 22,717 subjects aged 15–64 with primary trauma in only one body region. The overall alcohol involvement rate was 17%. Forty-seven percent (1879/3993) of male subjects with head trauma were under the influence of alcohol. The odds for head trauma were 8.3 among male subjects under the influence of alcohol compared with sober subjects. For women, the corresponding odds for head trauma were 7.0. A total of 385 consecutive trauma subjects aged 16–49 were investigated in the Oulu emergency room using blood/breath alcohol measurement. Subjects with head trauma were significantly more likely to be under the influence of alcohol than those with other types of trauma (65% versus
All these studies show that acute intoxication increases the risk for head trauma, in particular, compared with other parts of the body. It has also been demonstrated that the risk for head trauma increases according to the degree of intoxication. Honkanen and Smith divided trauma subjects into four different alcohol intoxication groups (sober, slight, moderate and heavy). Classification was based on either clinical evaluation or a breath test. It was found that the odds for head trauma increased with the degree of intoxication (Honkanen & Smith 1991). Savola et al. found that the risk of getting a head trauma compared with other traumas increased significantly after blood alcohol content exceeded 1.5 g/l. The age- and sex-adjusted odds ratios (OR) for head traumas compared with other traumas were 1.24, 95% CI 0.55–2.01 (BAC 0.1–0.99 g/l); 1.64, 95% CI 0.71–3.77 (BAC 1.00–1.49 g/l); 3.20, 95% CI 1.57–6.53 (BAC 1.50–1.99 g/l) and 9.23, 95% CI 4.79–17.79 (BAC > 1.99 g/l) (Savola et al. 2005).

### 2.3.3 Alcohol-related TBI mortality

Six million deaths caused by injury occur annually in the world (World Health Organization (WHO) 2008). TBIs and other nervous system traumas are frequently (33–62%) found to be the main cause of death (Acosta et al. 1998, Evans et al. 2010, Shackford et al. 1993, Söderlund et al. 2009). However, little is known about the contribution of alcohol, particularly to fatal TBI. A medico-legal autopsy study from Sweden including unnatural deaths (ICD-9 E-codes) reported that alcohol-related deaths amounted to 33% among women and 48% among men, but percentages of fatal alcohol-related TBIs were not reported (Sjögren et al. 2006). A study of 753 consecutive deaths in a US trauma centre reported that 51% of deaths were due to central nervous system injuries (Stewart et al. 2003). Forty-nine percent of all deaths were associated with blood alcohol content ≥ 0.01 or positive toxicology screening.

There is strong evidence that acute alcohol intoxication exposes subjects to traumas. It increases the occurrence of trauma and the risk for prehospital death. Tien et al. studied the association between BAC and mortality from TBI among 1158 consecutive subjects with severe TBI who were admitted to a trauma centre in Canada. Four hundred and three subjects (35%) died during hospital treatment. One hundred and thirty-four (33%) of deceased subjects had alcohol in their blood upon admission. The risk of death was significantly lower in subjects with low to moderate BAC (0.1–2.3 g/l) than in sober subjects. On the other hand,
subjects with high BAC (> 2.3 g/l) had a higher risk of death than sober subjects (Tien et al. 2006). This study did not include subjects who died on the scene and did not survive to hospital. Another study from Canada did not observe any significant differences in in-hospital, 90- and 365-day mortality between subjects who were under the influence of alcohol (at any level) and sober subjects (Shandro et al. 2009). Some studies have suggested that alcohol could be a neuroprotective agent in subjects with moderate-to-severe TBI (Salim et al. 2009a, Salim et al. 2009b). One review concluded that there are studies showing increased mortality from TBI in alcohol-intoxicated subjects, studies reporting no significant difference between intoxicated and sober subjects as well as studies showing decreased mortality in intoxicated subjects (Opreanu et al. 2010). The review revealed that these studies have several limitations, which should be kept in mind when interpreting the results. A comprehensive population-based medico-legal study of TBI mortality is needed to demonstrate whether there is an association between alcohol and mortality from TBI.

2.3.4 Sex- and age-specific differences

Only a few studies have reported alcohol involvement among subjects with TBI according to sex. Alcohol involvement varies 13–43% among men with all types of trauma and 6–24% among women (Honkanen et al. 1975, Honkanen & Smith 1991, Peppiatt et al. 1978). An early study of head trauma subjects from Glasgow observed that 62% of men and 27% of women were under the influence of alcohol (Galbraith et al. 1976). One hundred and sixty-seven out of 398 (42%) men and 24 out of 122 (20%) women with TBI were identified as being under the influence of alcohol (Gurney et al. 1992). Swan et al. reported alcohol involvement among 78/170 (46%) men and 14/51 (27%) women with TBI (Swan et al. 2004).

The highest TBI incidences have been found among children and young adults (Andersson et al. 2003, Jennett 1996, Winqvist et al. 2007). TBI mortality peaks among subjects aged 20 and over 70 (Jennett 1996). An ER study from the US found that 45% of 25–34-year-old TBI patients (86/191) were intoxicated (Gurney et al. 1992). The corresponding figures were 24.1% (46/191) for subjects aged 18–24, 22.5% (43/191) for subjects aged 35–49, 5.8% (11/191) for subjects aged 50–64 and 2.6% (5/191) for subjects aged over 65. Another head trauma study from the US found that high BAC levels were most frequent among
subjects aged 25–44 (Kraus et al. 1989). There are no Finnish data on alcohol involvement according to age in subjects with TBI.

### 2.3.5 Alcohol and different causes of TBI

In Finland, 58–63% of TBIs are caused by falls, 18–23% by traffic, 4–8% by assault and the rest by other causes (Alaranta et al. 2002, Numminen 2011). However, the distribution of causes varies widely in different parts of the world. For example, the proportion of TBIs caused by traffic ranges from 24% to 90% and that caused by assaults, from 1% to 45% (Jennett 1996).

Some external causes of TBI are frequently associated with alcohol drinking, whereas others are not (sports injuries). TBIs due to assaults and falls show the highest proportion of alcohol involvement. In some countries, TBIs caused by motor vehicle collisions are also often associated with alcohol. A recent study from the US found that the proportion of intoxicated subjects was 55% in assault-related, 46% in fall-related and 35% in traffic-related severe TBIs. (Talving et al. 2010). TBI subjects are frequently tested for BAC in the ER. A study from San Diego, US, identified 2649 hospitalised or deceased subjects with TBI aged ≥ 15 within one year (Kraus et al. 1989). Forty-four percent (1155/2649) of those subjects were tested for blood alcohol. The BAC testing rate varied from 87% of firearm trauma to 47% of motor vehicle accidents, 43% of assaults and 33% of falls. Two-thirds of the tested subjects were positive for alcohol if TBI was caused by motor vehicle accidents, the corresponding figures for assaults (60%), falls (44%) and firearms (35%) being lower. Another study from the US found that TBI caused by assaults showed the highest proportion of alcohol involvement (Gurney et al. 1992). Thirty-six out of 53 (68%) TBI subjects with assaults being the external cause were intoxicated, whereas the corresponding figures for traffic- (117/345, 34%), and fall-related TBIs (34/91, 37%) were lower.

### 2.3.6 Temporal distribution of alcohol-related TBIs

Several studies have shown that alcohol-related traumas accumulate on weekends (Luke et al. 2002, O'Sullivan & O'Conor 2003, Oikarinen et al. 1992, Peppiatt et al. 1978, Schepens et al. 1998, Smith et al. 1989). An early study observed that 47% of TBI admissions to a city hospital occurred on Fridays and Saturdays and the subjects were more often under the influence of alcohol (62%, 266/431) than were those admitted on other days (43%, 210/487) (Galbraith et al. 1976). A
Finnish study found seasonal variations in the number of alcohol-associated mandibular fractures (Oikarinen et al. 1992). More alcohol-associated mandibular fractures occurred during late summer and autumn and fewer in the spring. However, we lack Finnish data on the seasonal distribution of alcohol-related TBIs.
3 Purpose of the study

The specific aims were to assess

1. Effect of alcohol consumption on weekday and seasonal occurrences of TBI (paper I)
2. Accuracy of TBI diagnosis among subjects with craniofacial fractures and does the subject’s alcohol intoxication hamper diagnosis of TBI (paper II)
3. Effect of alcohol price reduction on TBI mortality and morbidity among a defined population from Northern Finland (paper III and IV)
4. Effect of alcohol price reduction on the mortality of harmful drinkers among a head trauma cohort during a 10-year follow-up (paper V)
4 Subjects and methods

The study protocol was approved by the Ethical Committee of the Northern Ostrobothnia Hospital District and the Oulu Provincial Government (later the Regional State Administrative Agency).

4.1 Subjects

4.1.1 Subjects in the emergency room

All subjects with head trauma ranging from small wounds to severe brain injuries (including subjects with craniofacial fractures and multiple traumas) admitted to the emergency room of Oulu University Hospital during 1999 were included. Acute head traumas were identified from the line listing kept every day at the emergency room during 1999. A study nurse checked the line listing daily and picked up those subjects whose notes in the hospital record suggested head trauma. In addition, our hospital discharge registry was checked weekly to identify cases of head trauma which were not observed already on the basis of the ER line listing. This method was used to identify as many head trauma subjects as possible during 1999. Subjects with head traumas, including ICD-10 diagnoses S06.0–S07.9 (TBI) and S02.0–S02.91 (craniofacial fracture) admitted to Oulu University Hospital during 2007 were included. Subjects with minor head trauma without these diagnoses were excluded during 2007. During both years data were collected from 1 January to 31 December. The data were gathered from hospital charts and they included information on sex, age, symptoms, time of trauma, cause of trauma, alcohol involvement, GCS score (extracted from symptoms of hospital charts, if not mentioned by the treating physicians), specialty of the treating physician, radiological investigations, place of residence, length of hospital stay and both the initial and final diagnoses.

We also gathered data on all deaths due to head trauma (ICD-10: S00.0–S09.9) recorded by the Department of Forensic Medicine, University of Oulu, during 1999, 2006 and 2007. All medico-legal autopsies within the province of Oulu are carried out in the Department of Forensic Medicine, University of Oulu. In Finland, a medico-legal autopsy is performed on all those who die because of trauma or some other unnatural cause (Act on the Inquest into the Cause of Death, 459/1973, Finnish Law). Data included information on age, sex, underlying and
contributing causes of death, external causes of trauma, precise time of death, estimated time from trauma to death and alcohol involvement.

### 4.1.2 Follow-up cohort

Altogether 832 cases with head trauma, including also children and the oldest old, were admitted to Oulu University Hospital emergency rooms in 1999. During the follow-up and after re-checking the cohort, we found three subjects with duplicate recordings of the same trauma, and the duplicates were excluded. Two subjects with suspected head trauma had an intracranial haemorrhage, which in later checking appeared to be a spontaneous cerebral haemorrhage. These cases were also excluded from the cohort. We formed a cohort of the remaining 827 subjects and followed them up until the end of 2009. The data on these subjects were identified using social security numbers from the National Hospital Discharge Registry (NHD). We did not use only register data from NHD but also checked data from the hospital charts. Eight hundred and twenty-seven (n = 827) subjects had complete data and were included in the follow-up cohort. The NHD registry includes all diagnoses recorded at discharge and the date/duration of treatment in any hospital for in-patient, out-patient and emergency room visits (a few hospitals were lacking psychiatric out-patient data from 1999–2004). The registry also includes the diagnoses and duration of treatment for in-patient visits to health centres (but not for out-patient or emergency room visits). All 827 subjects were identified from the Statistics Finland Causes-of-Death Registry (SFCD) for possible death until the end of 2009. Altogether 160 subjects died during the follow-up period. We gathered data on causes of death from the Statistics Finland Causes-of-Death Registry, and that data included both the underlying and contributory causes of death (ICD-10), time of death and whether the causes of deaths were determined by medico-legal autopsy, medical autopsy or clinical examination. A summary of all the studies, the number of subjects included in each study, the type of material and the aims of each study are summarised in Table 2.
<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects with</th>
<th>Year</th>
<th>Number</th>
<th>Material</th>
<th>Aim(s) of the study</th>
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<td>head trauma</td>
<td>1999</td>
<td>832</td>
<td>All subjects admitted to the emergency room of Oulu University Hospital</td>
<td>To see the effect of alcohol consumption on weekday and seasonal distribution of head traumas</td>
</tr>
<tr>
<td>II</td>
<td>TBI and craniofacial fracture</td>
<td>1999, 2007</td>
<td>111</td>
<td>All subjects with craniofacial fractures admitted to the emergency room of Oulu University Hospital</td>
<td>To evaluate the diagnostic accuracy of TBI among subjects with craniofacial fractures and to see whether the subjects' intoxication hampered the diagnosis of TBI</td>
</tr>
<tr>
<td>III</td>
<td>fatal TBI</td>
<td>1999, 2006, 2007</td>
<td>318</td>
<td>Inhabitants of Oulu Province with fatal TBI</td>
<td>To see whether alcohol price reduction in 2004 influenced TBI mortality</td>
</tr>
<tr>
<td>IV</td>
<td>moderate-to-severe TBI</td>
<td>1999, 2007</td>
<td>261</td>
<td>Inhabitants of Northern Ostrobothnia with moderate-to-severe TBI</td>
<td>To see whether alcohol price reduction in 2004 influenced the incidence of moderate-to-severe TBI</td>
</tr>
<tr>
<td>V</td>
<td>head trauma</td>
<td>1999–2009</td>
<td>827</td>
<td>All subjects belonging to Study I except 5 cases that were excluded</td>
<td>To see whether alcohol price reduction in 2004 influenced mortality among a cohort of head trauma subjects during a 10-year follow-up</td>
</tr>
</tbody>
</table>
4.2 Definitions

4.2.1 TBI

In paper I, altogether 832 subjects with head trauma were included. These 832 subjects included all head traumas from minor wounds/lacerations to more severe TBIs. In paper I, the severity of the head trauma/TBI was divided into three different categories modified from the EFNS guideline (Vos et al. 2002). Moderate-to-severe TBIs included subjects with brain contusion, diffuse axonal injury, traumatic subarachnoid haemorrhage and traumatic intracranial haematomas regardless of GCS score or surgery requirement, whereas mild TBIs included subjects with a GCS score of 13–15 and LOC < 30 min or PTA < 1 hour in the absence traumatic intracranial findings on a brain CT/MRI. Subjects with LOC > 30 min or PTA > 1 hour or a GCS score under 13 without brain CT/MRI findings were also classified as having moderate-to-severe TBI. Subjects without a brain CT/MRI were also classified as having mild TBI if other criteria for mild TBI were fulfilled. We also used a third category which included head traumas with a GCS score of 15 and no LOC or no PTA. These subjects were classified as having head trauma without brain injury (category 0 according to the EFNS guideline). The same classification of head trauma/TBI was used in our follow-up study (paper V).

The severity of TBI was classified differently in papers II and IV. In paper II, we used the criteria recommended by the WHO collaboration centre task force on mild traumatic brain injury (Carroll et al. 2004). TBI was classified as mild if the GCS score was 13–15 and one or more of the following symptoms were present: confusion or disorientation, loss of consciousness for up to 30 minutes, post-traumatic amnesia for less than 24 hours or if other transient neurological abnormalities such as focal neurologic deficits, seizures or intracranial lesions not requiring surgery were observed. Moderate-to-severe TBI was diagnosed in subjects with a GCS score under 13 or one or more of the following symptoms: loss of consciousness over 30 minutes, post-traumatic amnesia over 24 h or intracranial abnormality requiring surgery. In paper IV, which included only moderate-to-severe TBI subjects, the severity of TBI was classified simply according to GCS score. Subjects with a GCS score lower than 13 on admission were classified as having moderate-to-severe TBI.
4.2.2 Alcohol involvement

Subjects with TBI are often under the influence of alcohol. However, alcohol is not necessarily measured from every trauma patient. Usually, blood alcohol is measured from all subjects who are unconscious at presentation to the ER. Measured alcohol was considered to be positive if its concentration in blood or breath was 10 mg/dl (0.1‰) or higher. In Finland the generally used alcohol unit is per mille (1‰ = 100 mg/dl = 22 mmol/l). Some subjects were judged to be under the influence of alcohol (in the absence of alcohol measurement) by health care providers who noted it on hospital charts. Therefore, we classified that the trauma was alcohol-related if either alcohol measurement was positive or the subject was noted to be under the influence of alcohol according to hospital charts. The same classification was used to define alcohol-related trauma among the deceased subjects, but we also took into account alcohol measured from urine or other tissues or whether alcohol was considered to be a contributory cause of death according to the forensic death certificate (paper I, III, IV, V). In paper II, the term alcohol-related was based only on positive BAC.

The term harmful alcohol use has been defined by the WHO as a pattern of alcohol use that causes damage to health (WHO 1992). Accordingly, the diagnosis requires that actual damage should have been caused to the mental or physical health of the user. In our follow-up study (paper V), harmful drinking was defined as having occurred if the follow-up data obtained from our hospital charts or NHD included any diagnosis of alcohol-related disease (including mental and behavioural disorders due to alcohol, alcoholic hepatitis, acute and chronic alcohol liver cirrhosis or acute and chronic alcohol pancreatitis) or if there was any diagnosis on hospital charts of acute alcohol intoxication or harmful use of alcohol immediately preceding admittance to the ER, health centre or hospital during the follow-up. We defined a subject as a harmful drinker even with one alcohol-related medical event. According to the WHO definition, the term harmful drinking does not require repeated alcohol-related medical events (WHO 1992). We excluded alcohol-related index trauma in 1999 as harmful drinking, because we used it and harmful drinking later on as separate parameters in our analyses. Accordingly, harmful drinkers in paper V include only subjects who had recorded health problems directly related to alcohol after the index trauma (i.e. after the year 1999). However, harmful drinkers did not include subjects who had an alcohol-related disease or intoxication as a contributing cause of death in the death certificate obtained from SFCD if they did not have any alcohol-related
visits to a hospital or health centre during the follow-up period according to the NHD data.

### 4.2.3 Other definitions

Age was divided into five different categories: < 15, 15–34, 35–54, 55–74 and > 74 years. External causes of traumas were falls, traffic, intentional, sports and other. In paper IV intentional causes were divided into suicide and assaults.

### 4.3 Methods and statistical analyses

All the analyses were performed using SPSS, version 10.0–20.0 (all papers), Medstat software, version 2.2 in paper I and CIA statistical software, version 1.0 (paper III and IV). Fischer’s exact 2-tailed or Pearson’s $\chi^2$ tests were used for comparison of categorical variables. Continuous variables between groups were compared with Student’s t-test. Annual mortality and incidence rates were calculated as the number of cases per 100,000 population. However, because this material is rather small in size, we prefer to use proportions rather than incidence figures. Small changes are more easily observable within a group than if related to a population.

In paper I, the number of head traumas in alcohol-related and sober subjects according to day and month were compared. The excess risk for head trauma related to alcohol was obtained by calculating the difference between these two groups:

$$\text{ARR (Alcohol related risk)} = \text{Risk}_{\text{use of alcohol}} - \text{Risk}_{\text{sober}}.$$  

Weekday distribution of head trauma was compared with the weekly pattern of alcohol consumption in Finland. Confidence intervals (CI) for percentages and relative risk ratios were calculated (paper I).

In paper II, subjects with craniofacial fracture(s) with symptoms and/or signs of TBI were identified. Clinical symptoms of TBI were gathered from notes on hospital charts and compared with the recorded diagnoses at discharge. Logistic regression analysis was used to determine ORs and 95% CIs of variables predicting TBI to remain unrecorded. The test for significance was based on changes in log (partial) likelihood. In a final logistic model, all of the considered possible predictors were fitted simultaneously to provide adjusted estimates. A
two-tailed $P$ value of less than 0.05 was considered statistically significant. The significance of alcohol involvement to omission of TBI diagnosis was also investigated.

In paper III, changes in the number and alcohol involvement of fatal TBI were observed during 1999, 2006 and 2007. Age-adjusted annual mortality rates were calculated as deaths per 100,000 persons in the population. Poisson’s log linear regression models were used to estimate the differences in mortality rates (TBI, alcohol-related TBI, cause of trauma) between different factors (sex, age and year groups). Final models were stratified by age groups (15–34, 35–54 and 55–74) and by calendar periods (1999 and 2006–2007). Logistic regression analysis was used to determine which factors were independently associated with alcohol involvement in fatal TBI. A forward stepwise method selected variables in descending order of strength of association with alcohol involvement ($p \leq 0.05$ to enter, $p > 0.1$ to exit). Age, sex, observation year and external cause of trauma were included in the final model.

In paper IV, subjects with moderate-to-severe TBI (among the population of Northern Ostrobothnia) were identified in 1999 and 2007. Incidence rates, mortality and alcohol involvement in moderate-to-severe TBIs before and after the reduction of alcohol prices were calculated. Logistic regression analysis was used to determine which factors were independently associated with fatal TBI. A forward stepwise method selected variables in descending strength of association with fatal TBI ($p \leq 0.05$ to enter, $p > 0.1$ to exit). Factors in the model included age as a continuous variable, sex, year of observation, external causes of trauma and alcohol-related trauma. A similar analysis was performed to find out which factors were associated with alcohol-related TBI.

In paper V, the cohort of head trauma subjects admitted to our hospital during 1999 were followed up until death or the end of 2009. Data from NHD, SFCD and hospital charts were collected. Hazard ratios (HR) and 95% confidence intervals (CI) were calculated using the Cox proportional hazard model to find out the independent predictors for death during the follow-up period. The Kaplan-Meier method was used to estimate the cumulative survival rates of alcohol-related deaths and non-alcohol-related deaths. The curves were compared using the log-rank test.
5 Results

5.1 Effect of alcohol consumption on weekday and seasonal occurrence of head trauma (paper I)

In paper I, all 832 subjects with head trauma admitted to Oulu University Hospital emergency rooms in 1999 were included. Two hundred and ninety-seven (35.7%) of the subjects were under the influence of alcohol. Head traumas seemed to accumulate on weekends. We found an 11.5% (95% CI 6.77 to 16.31) increase in head traumas during weekends, assuming that occurrence should be evenly distributed by weekday. The weekend increase was mainly due to alcohol-related head traumas. This is illustrated by Figure 3, where we can see that the proportion of sober subjects with head traumas is evenly distributed by weekday, whereas the proportion of those under the influence of alcohol with head traumas is significantly more frequent on weekends. The excess risk for head trauma related to alcohol was 10.3% (95% CI 1.4 to 19.2) on Fridays, 23.9% (95% CI 15.4 to 32.4) on Saturdays and 21.9% (95% CI 13.2 to 30.5) on Sundays. The overall excess risk related to alcohol from Friday to Sunday was 27.3% (95% CI 14.2 to 40.2) among women and 20.3% (95 CI 12.3 to 28.34) among men.

Fig. 3. Percentage distribution of head trauma patients admitted to the ER of Oulu University Hospital by sex and weekday; striped area = alcohol-related cases. (Paper I, published by permission of Oxford University Press)
Alcohol consumption accumulates on weekends in Finland. The popular drinking pattern in Finland is binge drinking and the drinking occasions of Finnish people aged 15–69 accumulate on Fridays, Saturdays and Sundays (Metso et al. 2002). The weekday distribution of alcohol-related head traumas is very similar to the weekday distribution of drinking occasions in Finland, as seen in Figure 4.

![Figure 4](image)

**Fig. 4.** Percentage distribution of alcohol-related head trauma patients and drinking occasions (general population) in Finland by weekday. (Paper I, published by permission of Oxford University Press)

Seasonal variations in the proportion of sober and intoxicated subjects were also observed. Sober subjects’ head traumas occurred evenly throughout the year, whereas those of intoxicated subjects peaked significantly in July. July is the most common holiday season in Finland and the amount of alcohol sold in July is rather high. The excess risk related to alcohol was 7.9% (95% CI 3.3–12.5) in July, being 16.1% (95% CI 4.8–27.5) for women and 5.3% (95% CI 0.1–10.6) for men.

### 5.2 Craniofacial fractures and accuracy of TBI diagnosis (paper II)

In paper II we identified all the subjects with craniofacial fractures admitted to Oulu University Hospital in 1999 (n = 102) and 2007 (n = 92). After reviewing
the hospital charts, we found that 111/194 subjects (57%) had TBI based on symptoms and signs indicating TBI—40 in 1999 and 71 in 2007. Fifty-one of the TBIs (46%) had remained unrecorded at discharge. Independent predictors for TBI remaining unrecorded were the specialty of the treating physician being something other than neurology or neurosurgery (OR 29.2, 95% CI 8.0–106.3) and falling as the cause of trauma (OR 4.0, 95% CI 1.0–15.4). However, alcohol involvement was not a predictor for TBI diagnosis remaining unrecorded (See paper II, Table 4). Altogether, blood/breath alcohol content was positive in 37/111 (33%) subjects. A greater proportion of subjects were under the influence of alcohol in 2007 (26/71, 37%) than in 1999 (11/40, 28%), however the difference was not statistically significant. Alcohol was present in 12/24 (50%) of intentional injuries, in 15/48 (31%) of falling accidents and in 10/33 (30%) of traffic accidents, but in none of those with sports injuries. Alcohol intoxication of the subject did not influence the diagnostic accuracy of TBI (See paper II, Table 4).

5.3 Effect of alcohol tax reduction on TBI mortality (paper III)

All subjects with fatal TBI during 1999, 2006 and 2007 in Oulu Province were included and analysed in paper III. Altogether 318 deceased subjects were identified and their medico-legal autopsy reports were reviewed. TBI mortality rates were similar during those years, and the annual average rate was 22.8/100,000. Alcohol involvement did not change significantly during the years. In 2006–2007 the deceased subjects were significantly older than in 1999. Falling as a cause of trauma was significantly more frequent in 2006–2007 than in 1999 (see Table 3). Fall-related mortality increased significantly from 5.9/100,000 population in 1999 to 9.8/100,000 population in 2006–2007 (a difference of 3.89 95% CI 0.90–6.9).
Table 3. Demographic data on subjects with fatal TBIs in Oulu Province by year.

<table>
<thead>
<tr>
<th>Variable</th>
<th>1999</th>
<th></th>
<th>2006</th>
<th></th>
<th>2007</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of cases,</td>
<td>Mortality</td>
<td>No. of cases,</td>
<td>Mortality</td>
<td>No. of cases,</td>
<td>Mortality</td>
</tr>
<tr>
<td></td>
<td>n(%)</td>
<td></td>
<td>n(%)</td>
<td></td>
<td>n(%)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>104</td>
<td>22.8</td>
<td>110</td>
<td>23.5</td>
<td>104</td>
<td>22.1</td>
</tr>
<tr>
<td>Alcohol-related</td>
<td>56 (54)</td>
<td>12.3</td>
<td>51 (46)</td>
<td>10.9</td>
<td>52 (50)</td>
<td>11.1</td>
</tr>
<tr>
<td>Men</td>
<td>82 (79)</td>
<td>35.8</td>
<td>81 (74)</td>
<td>34.4</td>
<td>84 (81)</td>
<td>35.5</td>
</tr>
<tr>
<td>Age, years (mean ± SD)</td>
<td>47±22</td>
<td></td>
<td>53±22*</td>
<td></td>
<td>54±22*</td>
<td></td>
</tr>
<tr>
<td>External cause of death</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Falling</td>
<td>27 (26)</td>
<td>5.9</td>
<td>44 (40)*</td>
<td>9.4</td>
<td>48 (46)**</td>
<td>10.2‡</td>
</tr>
<tr>
<td>Traffic</td>
<td>23 (22)</td>
<td>5.0</td>
<td>17 (15)</td>
<td>3.6</td>
<td>17 (16)</td>
<td>3.6</td>
</tr>
<tr>
<td>Intentional</td>
<td>44 (42)</td>
<td>9.6</td>
<td>38 (35)</td>
<td>8.1</td>
<td>32 (31)</td>
<td>6.8</td>
</tr>
<tr>
<td>Other</td>
<td>10 (10)</td>
<td>2.2</td>
<td>11 (10)</td>
<td>2.4</td>
<td>7 (7)</td>
<td>1.5</td>
</tr>
</tbody>
</table>

* p < 0.05, **p < 0.01 for difference between 1999 and the index year;  
Mortality = cases / 100,000 population  
‡Difference between the years 1999 and 2007 is 4.3 (95% CI 0.6–7.9); only significant difference in mortality

The characteristics of the subjects according to alcohol involvement are shown in Table 4. Alcohol-related fatal TBIs occurred significantly more frequently among men, p < 0.001. Subjects with fatal TBIs were significantly older in 2006–2007 than in 1999 (mean difference 7.0 years, 95% CI 1.88–12.05, p = 0.007) and the difference was seen both in alcohol-related and non-alcohol-related fatal TBIs. However, the difference was statistically significant only in alcohol-related TBIs (mean difference 7.8 years, 95% CI 2.2–13.5, p = 0.007). Significant changes between the observed years were also seen in causes of traumas. The proportion of falls as a cause of trauma increased significantly (p < 0.05) in alcohol-related TBIs from 1999 (12/56, 21%) to 2006–2007 (43/103, 42%). Alcohol-related mortality due to falls increased from 2.6/100,000 in 1999 to 4.6/100,000 in 2006–2007 (difference 2.0 95% CI -0.7–4.0). A greater proportion of deaths from falls was also seen in non-alcohol-related TBIs, from 15/48 (31%) in 1999 to 49/111 (46%) in 2006–2007, but the difference was not significant. Falls were more commonly (p = 0.011) alcohol-related among middle-aged people (35–54 yrs) in 2006–2007 (14/15, 93%) than in 1999 (2/6, 33%). On the other hand, the proportion of alcohol-related fatal TBIs caused by intentional injuries decreased from 33/56 (59%) in 1999 to 43/103 (42%) in 2006–2007.
Table 4. Sex, age and external cause of trauma by year among subjects with alcohol-related and non-alcohol-related fatal TBI

<table>
<thead>
<tr>
<th>Variables</th>
<th>1999 Alcohol</th>
<th>2006 Alcohol</th>
<th>2007 Alcohol</th>
<th>1999 No alcohol</th>
<th>2006 No alcohol</th>
<th>2007 No alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>56</td>
<td>51</td>
<td>52</td>
<td>48</td>
<td>59</td>
<td>52</td>
</tr>
<tr>
<td>Men, n (%)</td>
<td>52 (93)</td>
<td>42 (82)</td>
<td>49 (94)</td>
<td>30 (63)</td>
<td>39 (66)</td>
<td>35 (67)</td>
</tr>
<tr>
<td>Mean age, y (SD)</td>
<td>40 ± 19</td>
<td>46 ± 17</td>
<td>50 ± 15**</td>
<td>54 ± 22</td>
<td>59 ± 24</td>
<td>58 ± 26</td>
</tr>
<tr>
<td>Cause of trauma, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Falling</td>
<td>12 (21)</td>
<td>19 (37)</td>
<td>24 (46)**</td>
<td>15 (31)</td>
<td>25 (42)</td>
<td>24 (46)</td>
</tr>
<tr>
<td>Traffic</td>
<td>6 (11)</td>
<td>4 (8)</td>
<td>2 (4)</td>
<td>17 (36)</td>
<td>13 (22)</td>
<td>15 (29)</td>
</tr>
<tr>
<td>Intentional</td>
<td>33 (59)</td>
<td>20 (39)*</td>
<td>23 (44)</td>
<td>11 (23)</td>
<td>18 (31)</td>
<td>9 (17)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (9)</td>
<td>8 (16)</td>
<td>3 (6)</td>
<td>5 (10)</td>
<td>3 (5)</td>
<td>4 (8)</td>
</tr>
<tr>
<td>Mortality (cases/100,000)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Falling</td>
<td>2.6</td>
<td>4.1</td>
<td>5.1</td>
<td>3.3</td>
<td>5.3</td>
<td>5.1</td>
</tr>
<tr>
<td>Traffic</td>
<td>1.3</td>
<td>0.9</td>
<td>0.4</td>
<td>3.7</td>
<td>2.8</td>
<td>3.2</td>
</tr>
<tr>
<td>Intentional</td>
<td>7.2</td>
<td>4.3</td>
<td>4.9</td>
<td>2.4</td>
<td>3.8</td>
<td>1.9</td>
</tr>
<tr>
<td>Other</td>
<td>1.1</td>
<td>1.7</td>
<td>0.6</td>
<td>1.1</td>
<td>0.6</td>
<td>0.9</td>
</tr>
</tbody>
</table>

* p < 0.05; **p < 0.01 for difference between proportions in 1999 and the corresponding index year

Mortality from TBI was not significantly different between the observation years. Average annual mortality was 22.8/10,000. TBI mortality rates by age and alcohol involvement are shown in Table 5. The highest mortality rate was observed among the oldest old (aged > 74). Their mortality rate increased from 48.7/100,000 in 1999 to 58.3/100,000 in 2007, whereas at the same time mortality rates decreased among middle-aged people and young adults. The mortality rate of men aged 15–34 decreased from 46.9/100,000 in 1999 to 28.0/100,000 in 2007. The most significant decrease occurred in alcohol-related subjects: the alcohol involvement fraction decreased among young male adults from 23/30 (77%) in 1999 to 8/18 (44%) in 2007 (p = 0.024), but not among women. Alcohol-related mortality of men aged 15–34 decreased significantly from 35.9/100,000 in 1999 to 12.5/100,000 in 2007 (difference 23.5 95% CI 6.5–40.5). On the other hand, the alcohol involvement fraction increased among the middle aged (35–54) from 15/31 (48%) in 1999 to 20/22 (91%) in 2007. At the same time, both the overall mortality rate and the fraction which was not related to alcohol decreased among middle-aged people.
<table>
<thead>
<tr>
<th>Age group</th>
<th>&lt; 15</th>
<th>Difference</th>
<th>15–34</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>number of cases</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>34</td>
</tr>
<tr>
<td>population</td>
<td>95,396</td>
<td>92,736</td>
<td>92,637</td>
<td>121,679</td>
</tr>
<tr>
<td>mortality</td>
<td>2.1</td>
<td>2.2</td>
<td>1.1</td>
<td>-1.0 (-4.6–2.6)</td>
</tr>
<tr>
<td>alcohol-related</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td>ARM</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7.3</td>
</tr>
<tr>
<td>AIF (%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7.3</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>number of cases</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>30</td>
</tr>
<tr>
<td>population</td>
<td>48,677</td>
<td>47,541</td>
<td>47,572</td>
<td>64,012</td>
</tr>
<tr>
<td>mortality</td>
<td>2.1</td>
<td>2.1</td>
<td>0</td>
<td>-2.1 (-6.1–2.0)</td>
</tr>
<tr>
<td>alcohol-related</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>23</td>
</tr>
<tr>
<td>ARM</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>35.9</td>
</tr>
<tr>
<td>AIF (%)</td>
<td>0</td>
<td>0</td>
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<td>7.3</td>
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<td>Women</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>number of cases</td>
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<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>population</td>
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<td>45,195</td>
<td>45,065</td>
<td>57,667</td>
</tr>
<tr>
<td>mortality</td>
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<td>2.2</td>
<td>2.2</td>
<td>0.08 (-6.0–6.1)</td>
</tr>
<tr>
<td>alcohol-related</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>ARM</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3.5</td>
</tr>
<tr>
<td>AIF (%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7.3</td>
</tr>
</tbody>
</table>

AIF = alcohol involvement fraction, mortality = cases/100,000, ARM = alcohol-related mortality (alcohol-related cases/100,000)

Difference in mortality from 1999 to 2007; 95% CI/100,000/ per year
<table>
<thead>
<tr>
<th>Age group</th>
<th>35–54</th>
<th>Difference</th>
<th>55–74</th>
<th>Difference</th>
<th>&gt; 74</th>
<th>Difference</th>
</tr>
</thead>
</table>

### All

<table>
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<th></th>
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<th></th>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>number of cases</td>
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### Men

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AIF = alcohol involvement fraction, mortality = cases/100,000, ARM = alcohol-related mortality (alcohol-related cases/100,000)

Difference = Difference in mortality from 1999 to 2007; (95% CI/100,000/year)
Death rates by age and observation years as well as proportions of the deceased who were under the influence of alcohol are illustrated in Figure 5. The lowest death rate was observed among children and the highest among the oldest old (> 74 years). None of the children had an alcohol-related fatal TBI, and only a few were seen among old people. The rates between the observation years were not significantly different among children or very old people. The proportion of young adults (15–34 years) decreased from 34/104 (33%) in 1999 to 44/214 (21%) in 2006–2007 (p = 0.018), whereas the proportion of elderly people (55–74 years) increased from 25/104 (24%) in 1999 to 75/214 (35%) in 2006–2007 (p = 0.047).

Fig. 5. TBI deaths by age and year. Cross-hatched area = alcohol-related.

TBI deaths by sex, age and year are illustrated in Figure 6. Death rates were rather constant among the middle-aged, but an increase was observed after the age of 74. Among men aged 15–34, mortality from TBI was higher in 1999 than in 2006–2007. This was due to higher alcohol-related mortality in 1999 than in 2006–2007, as illustrated in Figure 7.
Fig. 6. TBI deaths by sex, age and year; reference year 1999 boldfaced.
Associations between observation year, age group, cause of trauma and alcohol involvement were analysed using log linear Poisson regression analyses. People aged 55–74 had higher TBI mortality without interaction with observation year than those aged 15–34 ($Z = -4.24$, $p < 0.001$) or 35–54 ($Z = -3.64$, $p < 0.001$). However, TBI mortality was higher than expected among subjects aged 15–34 in 1999 (interaction, $Z = 1.93$, $p = 0.054$). Alcohol-related TBI mortality was significantly greater among those aged 55–74 than among those aged 15–34 ($Z = -3.19$, $p < 0.01$). We also analysed the association between TBI mortality and causes of trauma. Among those aged 15–34 and 35–54, fall-related TBI mortality was significantly lower than expected (without interaction) in comparison with those aged 55–74 ($Z = -3.24$, $p < 0.01$ and $Z = -4.10$, $p < 0.01$, respectively). TBI mortality due to intentional injury was almost significantly higher than expected among those aged 15–34 in 1999 (interaction, $Z = 1.92$, $p = 0.055$).

Independent predictors for alcohol involvement in fatal cases were found with logistic regression analyses. Male sex (OR 2.9, 95% CI 1.5–5.7) and fall-
related (OR 9.4, 95% CI 3.6–24.5) and intentional (OR 6.9, 95% CI 3.1–15.5) injuries were significant (p < 0.01) independent predictors for sustaining an alcohol-related fatal TBI. Older age protected against alcohol-related fatal TBI (OR per year 0.97, 95% CI 0.95–0.98, p < 0.01), although the overall mortality rate of the oldest old was highest. Observation year was not a predictor for alcohol-related fatal TBI.

5.4 Effect of alcohol price reduction on moderate-to-severe TBI (paper IV)

Two hundred and sixty-one subjects with moderate-to-severe TBI were identified in Northern Ostrobothnia—126 subjects in 1999 and 135 in 2007. Baseline data on the subjects is shown in Table 6. The overall incidence of moderate-to-severe TBI increased slightly but insignificantly from 1999 to 2007. Falling as a cause of trauma increased significantly from 11.2/100,000 in 1999 to 17.6/100,000 in 2007 (difference 6.4 95% CI 1.0–11.8). Incidence rates of alcohol-related moderate-to-severe TBI remained rather much the same (difference 1.6 95% CI -4.0–7.1).

Table 6. Characteristics of 261 subjects with moderate-to-severe TBI. (Paper IV, published by permission of Wiley)

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<td>103</td>
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<td>32</td>
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<td>41</td>
<td>46</td>
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<td>34(25)</td>
<td>34(37)</td>
<td>28(27)</td>
<td>8(23)</td>
<td>6(19)</td>
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<td>35–54</td>
<td>32(25)</td>
<td>38(28)</td>
<td>27(30)</td>
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<td>43(34)</td>
<td>56(41)</td>
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<td>37(36)</td>
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<td>19(59)</td>
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<td>48(47)</td>
<td>16(45)</td>
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<td>Alcohol-related, n (%)</td>
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63
Different proportions and incidences were observed between alcohol-related and non-alcohol-related TBIs by observation year, age group and cause of trauma (see Table 7). From 1999 to 2007, alcohol-related traumas decreased among young adults, but increased among middle-aged people. However, significant heterogeneity of alcohol-related TBIs was not observed by age and year. The number of both alcohol-related and non-alcohol-related falls increased from 1999 to 2007. The incidence of alcohol-related falls was significantly higher in 2007 than in 1999 (difference 5.2 95% CI 1.7–8.8). The incidence of alcohol-related suicides decreased significantly from 1999 to 2007 (difference 3.8 95% CI 0.5–7.0).
Table 7. Alcohol-related and non-alcohol-related TBIs by observation year, age group and external cause of trauma. (Modified from paper IV, published by permission of Wiley)

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<th>Difference</th>
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<td>Incidence</td>
<td>Number, n(%)</td>
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Incidence = cases/ 100,000 population

Difference = Difference in observed incidence from 1999 to 2007 (95% CI/100,000/year)
A multivariate regression analysis between different factors and alcohol-related TBI revealed that male sex (OR 4.04 95% CI 1.86–8.75), age 35–54 (OR 2.61 95% CI 1.22–5.60), suicide (OR 3.15 95% CI 1.20–8.22), assault (OR 13.61 95% CI 1.30–143.16) and falling (OR 2.58 95% CI 1.04–6.39) as a cause of trauma were predictors for alcohol-related TBI compared with non-alcohol-related TBI. One hundred and fifty-one subjects died from TBI and an autopsy was done on all of them (medico-legal autopsy). Alcohol involvement fraction was significantly (p < 0.001) higher among subjects who died (83/156, 53%) than among subjects with non-fatal TBI (30/105, 29%). Alcohol involvement was a significant and independent predictor for fatal TBI (OR 2.17 95% CI 1.18–3.98). However, there was no significant difference between observation year and alcohol involvement fraction among those who died. On the other hand, a greater proportion of subjects with non-fatal TBI were under the influence of alcohol (p = 0.018) in 2007 (22/58, 38%) than in 1999 (8/47, 17%).

5.5 Effect of alcohol price reduction on long-term mortality of harmful drinkers (paper V)

Subjects who were admitted to the ER of Oulu University Hospital because of head trauma in 1999 formed a cohort which was followed up for 10 years. Cohort included 827 subjects, of whom 160 (19%) died before the end of 2009. According to hospital and/or NHD records, 101 of the subjects were recorded as being harmful drinkers after 1999. These harmful drinkers were slightly older (p = 0.059), more frequently men (0 < 0.001) and had less severe index head injury in 1999 (significant heterogeneity p = 0.002). They were more often under the influence of alcohol (p < 0.001) on admission for the index trauma and more frequently had seizures (p < 0.001) and mental illness (p < 0.001) in their medical history.

During the follow-up, significantly (p < 0.001) more deaths occurred among them than among those who were not recorded as being harmful drinkers. Death certificates showed that alcohol was much more frequently (p < 0.001) coded as an underlying or contributing cause of death among them than among the other subjects belonging to the cohort. They also died more often while being under the influence of alcohol (p < 0.001).

We identified significant independent predictors for death with the Cox proportional hazards regression model. Age and severity of the index TBI were
significant predictors. Alcohol involvement in index trauma did not predict death, whereas being a harmful drinker during the follow-up was a powerful predictor.

The proportions of alcohol-related deaths during the follow-up in 2000–2009 are shown in Fig. 8. Included are subjects who were alive at the beginning of 2000. It appeared that the proportion of deaths which occurred under the influence of alcohol increased significantly (p = 0.005, test for linear trend) year by year. Simultaneously, per capita alcohol consumption increased in Oulu Province as well as in all of Finland.

![Fig. 8. Proportion of alcohol-related deaths in the cohort (bars and left axis, %) and documented per capita 100% alcohol consumption (line and right axis, litres) in Oulu Province. (Paper V, published by permission of Karger)](image)

Life table analysis (Fig. 9) revealed different rates of death between harmful drinkers (diagnosed in 1999–2009, n = 101) and those who were not harmful drinkers. Harmful drinkers showed a significantly ($\chi^2 = 10.31; p = 0.001$) lower cumulative survival rate. Their death rate increased significantly after the alcohol price reduction on 1 March 2004. Because 16/101 harmful drinkers were diagnosed after the alcohol price reduction, we did a separate analysis in which
these 16 subjects were considered non-harmful drinkers. Even in this analysis, harmful drinkers showed a significantly ($\chi^2 = 15.11; p = 0.001$) lower cumulative survival rate (Fig. 10).

Fig. 9. Reduction of alcohol price on 1 March 2004 associated with an increase in the cumulative death rate of subjects who were recorded as being harmful drinkers from 1999 until the beginning of 2010 (n = 101). (Paper V, published by permission of Karger)
Fig. 10. Cumulative death rate of subjects who were harmful drinkers already before the alcohol tax reduction (n = 85).
6 Discussion

6.1 Epidemiological observations

No significant increases were observed in either the incidence of moderate-to-severe TBIs or in TBI mortality from 1999 to 2007. The overall incidence of moderate-to-severe TBI was 34.1/100,000 in 1999 and 34.6/100,000 in 2007. Also mortality from TBI remained rather constant throughout the observed years. The overall TBI mortality figures for 1999, 2006 and 2007 were 22.8/100,000, 23.5/100,000 and 22.1/100,000. These figures are in accordance with the rate of TBI deaths reported previously from Finland (21.2/100,000) (Sundstrom et al. 2007). Similar figures have also been found in the US (23.6/100,000) (Adekoya & Majumder 2004). Although significant changes in overall incidence and mortality were not found, subjects with fatal TBIs were significantly older in 2006–2007 than in 1999. Mortality from TBIs decreased from 28/100,000 in 1999 to 18/100,000 in 2007 among young adults, but increased among people aged 55–74 from 31/100,000 in 1999 to 41/100,000 in 2007. Also the incidence of moderate-to-severe TBIs among young adults was lower in 2007 than in 1999, but the difference was not statistically significant. Falling as a cause of moderate-to-severe and fatal TBI increased significantly from 1999 to 2006–2007. On the other hand, the proportion of intentional and traffic collisions as causes of TBI decreased.

6.2 Alcohol consumption and occurrence of TBI

Earlier reviews have demonstrated the significant role that alcohol consumption plays in predisposing subjects to TBI (Corrigan 1995, Parry-Jones et al. 2006). In our study, alcohol involvement in fatal TBIs varied from 46% to 54%, depending on the year of observation. A Swedish autopsy study including all fatal injuries observed that alcohol-related injuries accounted for 40% of all deaths, but figures particularly for fatal TBIs were not reported (Sjögren et al. 2006). A study of 753 consecutive deaths in a US trauma centre observed that 49% of all deaths were associated with alcohol and 51% of all deaths were due to nervous system injuries (Stewart et al. 2003). Only a few alcohol involvement studies concentrating on fatal TBIs have been published. A Canadian trauma centre study of 403 fatal TBI subjects found that 33% of the subjects had alcohol in their blood on admission...
(Tien et al. 2006). A multicentre study from the US reported that 37% of in-hospital fatal TBIs were alcohol-related (Shandro et al. 2009). An Irish autopsy record study found that only 13% of fatal TBIs due to falls and road traffic accidents had occurred under the influence of alcohol, but only 28% of this population were tested for blood alcohol content (O'Toole et al. 2009). Our study comprehensively represents all fatal TBI subjects regardless of the place of death (including subjects who succumbed outside the hospital and in-hospital subjects), and therefore the proportion might be slightly higher than reported in earlier publications. We also included subjects with alcohol as a contributing cause of death, not only subjects with documented alcohol content.

The proportions of alcohol-related moderate-to-severe TBIs (of all moderate-to-severe TBIs) were 41% in 1999 and 45% in 2007. In 1999, head traumas involved alcohol in 36% of the subjects brought to our ER. These proportions are in accordance with earlier reports where alcohol involvement has been found in 34% to 48% of subjects with TBI admitted to emergency rooms (Dikmen et al. 1995, Gurney et al. 1992, Vickery et al. 2008).

It is well known that alcohol-related traumas accumulate on weekends (Luke et al. 2002, O'Sullivan & O'Conor 2003, Oikarinen et al. 1992, Peppiatt et al. 1978, Schepens et al. 1998, Smith et al. 1989). In line with these earlier studies, we found an alcohol-related excess risk of 21.1% among head traumas during weekends. The occurrence of alcohol-related head traumas paralleled the weekly rhythm of alcohol consumption in Finland. The finding that alcohol-related head traumas were common also on Sundays reflects heavy drinking during Saturday nights. In Finland, drinking occasions accumulate on Friday and Saturday nights. Alcohol-related traumas also accumulate on Sundays, although the heavy drinking occasions usually start on Friday and continue over Saturday until the early morning of Sunday. This discrepancy should be noted when interpreting results which compare the occurrence of a weekly rhythm in head traumas and drinking occasions.

### 6.3 Alcohol price reduction and TBI

Alcohol price reduction was implemented on 1 March 2004. As a consequence, total estimated per capita alcohol consumption increased about 10% in Finland within a year. From 1999 to 2007, recorded alcohol consumption increased 25% in Northern Ostrobothnia and even 29% in Oulu Province. Because we wanted to
see the long-term effects of increased alcohol consumption, we decided to compare data gathered from those years, i.e. from 1999 and 2007.

We observed that the increase in alcohol consumption did not significantly influence overall TBI mortality and incidence of moderate-to-severe TBIs. The reduction of alcohol prices was observed to increase both acute and chronic alcohol-related hospitalisations and alcohol-related deaths (Mäkelä & Österberg 2009). This increase was mainly due to alcoholic liver diseases, alcohol intoxication and alcohol-induced psychotic disorders. The increase in alcoholic liver diseases after the reduction of alcohol prices suggests that at least subjects with the habit of regular heavy alcohol use increased their consumption further. However, the reduction of alcohol prices did not seem to enhance interpersonal violence rates (Herttua et al. 2008). Binge drinking is more clearly associated with traumas than is regular drinking (Gmel et al. 2006), which is quite understandable, because regular drinkers develop central nervous system tolerance to alcohol, whereas occasional drinkers do not. In our catchment area, binge drinking is the prevailing drinking pattern among trauma patients, and only a small proportion of the TBI population is comprised of dependent drinkers (Savola et al. 2005).

Although it is well-known that alcohol drinking is frequently associated with the occurrence of acute TBI, the alcohol price reduction did not increase the overall incidence of alcohol-related TBIs. Perhaps the price reduction influenced the drinking of regular and habitual heavy drinkers more than that of occasional drinkers. Our study supports this and is in accordance with earlier studies, where the burden of alcohol harms after the reduction of alcohol prices was observed to concentrate around typical diseases of regular heavy users of alcohol, i.e. liver cirrhosis (Herttua et al. 2008, Mäkelä & Österberg 2009).

6.4 Age, sex and alcohol-related TBI

Alcohol-related moderate-to-severe and fatal TBIs did not increase after the reduction of alcohol prices. However, age-specific changes were observed among the deceased. Among middle-aged people (35–54 yrs), the proportion of alcohol-related fatal cases increased from 48% in 1999 to 91% in 2007; a significant increase was observed among men, but not among women. At the same time, a significant decrease in the alcohol involvement fraction of fatal cases was observed among men aged 15–34—from 77% in 1999 to 44% in 2007—but not among women. Similar trends were seen also in incidence rates of moderate-to-
severe TBIs, where alcohol-related moderate-to-severe TBIs decreased among young adults, but increased among middle-aged people. However, the changes in incidence rates were not statistically significant. Although total per capita alcohol consumption increased after the reduction of alcohol prices in Finland, the increase in alcohol consumption was not equally distributed among the population. Mäkelä et al. reported that older age groups were more influenced by the alcohol price reduction than younger adults (Mäkelä et al. 2008). Another study reported that increased alcohol consumption was seen particularly among middle-aged and less educated people (Helakorpi et al. 2010). The increase in alcohol consumption by older age groups could possibly be a reason for the increased occurrence of alcohol-related TBIs among middle-aged people in our study.

Some trends were observed in earlier studies on alcohol-related deaths after the reduction of alcohol prices. Alcohol-related deaths increased among people over 40, but younger people did not sustain increased alcohol-related deaths after the reduction of alcohol prices (Herttua et al. 2008). Acute alcohol-related (alcohol intoxication and poisoning) hospitalisations increased 17% among men aged 40–49, 20% among men aged 50–69, but not among men aged less than 40. Chronic alcohol-related (alcohol-attributable diseases other than intoxication and poisoning) hospitalisations increased 16% among men aged 15–39, 11% among men aged 40–49 and 22% among men aged 50–69. Among women aged 50–69, chronic hospitalisations increased 23%, whereas they decreased 8% among those aged 15–39 (Herttua et al. 2011). A Finnish Adolescent Health and Lifestyle Survey (including high school students aged 14–18) reported that drinking for intoxication at least once a month decreased from 27% in 1999 to 21% in 2007 among boys and from 22% in 1999 to 18% in 2007 among girls (Raisamo et al. 2011). Similar findings were observed in a Finnish drinking habit survey (Huhtanen et al. 2011). Drinking at least once a month (at least six drinks on a single occasion) decreased from 35% in 2000 to 26% in 2008 among men aged 15–19 and from 50% in 2000 to 36% in 2008 among men aged 20–29. The corresponding figures for women aged 15–19 were 24% in 2000, decreasing to 17% in 2008. Women aged 20–29 showed an increase from 20% in 2000 to 25% in 2008. In our study, the proportion of alcohol-related fatal TBIs decreased from 77% in 1999 to 44% in 2007 among men aged 15–34, but we cannot speak about a decrease among women, because only 3 women died from alcohol-related TBIs during these years. A greater proportion of moderate-to-severe TBIs among
people aged 15–34 were alcohol-related in 1999 (40%) than in 2007 (23%), and this decrease was particularly seen among men. These observations in age-specific alcohol-related TBIs may partly be explained by simultaneous changes in drinking habits.

6.5 Causes of alcohol-related TBI

There were significant changes in the causes of alcohol-related TBIs during the observation years. The proportion of alcohol-related fatal TBIs due to falls increased significantly from 1999 (21%) to 2007 (46%), and this occurred particularly among middle-aged and elderly people. A similar trend was seen in the incidence of moderate-to-severe TBIs from 3.6/100,000 in 1999 to 8.8/100,000 in 2007. Middle-aged and elderly people increased their alcohol consumption more than young people after the alcohol price reduction and that could at least partly explain the observed increase in the alcohol involvement fraction in moderate-to-severe TBIs among middle-aged and elderly people. If alcohol drinking becomes more popular among elderly people, we may have a huge problem with alcohol-related TBIs due to falls in the future. In Finland, the so-called baby boom generation (born after World War II) will be over 70 within a few years. The incidence and mortality of fall-related health problems will rise along with age. It has been estimated that every third person aged over 65 will fall annually (World Health Organization (WHO) 2007). Subjects over 65 years with fall-related TBIs also have more co-morbid conditions (such as hypertension, diabetes mellitus, cardiac arrhythmias and other neurological diseases) than subjects who sustain TBI due to traffic collisions (Coronado et al. 2005). These co-morbid conditions may contribute to worse outcomes after TBI among them.

In our study, the proportion of intentional injuries as a cause of trauma in moderate-to-severe and fatal TBIs was smaller in 2007 than in 1999. This decrease was observed in suicides among young men. This is in accordance with earlier observations. The number of suicides in Finland has been decreasing during the past two decades. A suicide prevention programme was instituted in the early 1990s, and during this programme mortality from suicides decreased 27% in men and 12% in women from 1997 to 2006 (Hiltunen et al. 2009). One of the main interventions in this suicide programme was an effort to decrease alcohol consumption among suicidal patients.
6.6 Effect of alcohol on diagnostic accuracy

The number of unrecorded TBI diagnoses among subjects with craniofacial fracture was high. Almost half (46%) of the craniofacial fracture subjects with TBI did not have the correct diagnosis documented in hospital discharge records. Similar results were reported in a study which determined how often emergency room patients meeting mild TBI criteria were diagnosed by the ER physician (Powell et al. 2008). In that study, 56% of mild TBIs did not have the diagnosis at discharge. In another report, 24/47 (51%) of head trauma patients in a large district and teaching hospital lacked a correct TBI diagnosis at discharge (Moss & Wade 1996). It has been suggested that subjects having other severe traumas and those with minor or trivial traumas are more likely to have an unrecorded TBI diagnosis. Davidoff et al. reported that plastic surgery, oral surgery and otolaryngology services frequently (19–27%) missed the diagnosis of mild TBI (Davidoff et al. 1988), and this was confirmed in our study, which found that non-neurological specialists frequently missed recording a TBI diagnosis. Independent predictors for an unrecorded TBI diagnosis were a specialty of the treating physician other than neurologist/neurosurgeon and falling as the cause of trauma.

It has been suggested that alcohol intoxication of the subject may influence the accuracy of GCS assessment because high alcohol concentration reduces the level of consciousness (Brickley & Shepherd 1995). This may result in overestimation of the severity of TBI and unnecessary investigations and treatments. On the other hand, a low GCS score may mistakenly be estimated to be due to alcohol intoxication and TBI may be unrecognised. Previous findings on the association between BAC and GCS score are controversial. An early study reported that a depressed level of consciousness occurred with high BAC (over 200 mg/100 ml) among subjects without severe TBI. Nevertheless, a significant number of patients with a low GCS score had severe TBI (Galbraith et al. 1976). Jagger et al. reported alcohol intoxication as a source of bias in the clinical classification of TBI severity (Jagger et al. 1984). Brickley and Shepherd found that neurological deficits were strongly associated with BAC and suggested that severe intoxication (more than 240 mg/100 ml) reduces the GCS score by 2–3 points (Brickley & Shepherd 1995). Several other studies fail to support these findings. A study from a neurosurgical unit found that previous alcohol intake did not influence the level of consciousness and it was suggested that a GCS score ≤ 8 was more likely caused by trauma than by high BAC (Nath et al. 1986). Some
results have been reported later on suggesting that alcohol intoxication does not result in a clinically significant decrease in the GCS score (Sperry et al. 2006, Stuke et al. 2007). In our study, alcohol did not hamper the diagnostic accuracy of TBI. Subjects under the influence of alcohol did not have a lower proportion of recorded TBI diagnosis than sober subjects. Failure to have a recorded TBI diagnosis was influenced by the specialty of the treating physician and the cause of trauma rather than by the fact that the subject was intoxicated.

6.7 Alcohol price reduction and mortality of harmful drinkers

It was a striking finding to observe that the mortality of harmful drinkers increased significantly soon after the alcohol price reduction was implemented in 2004. In our study, harmful drinking was frequently (12%) observed after the index trauma, i.e. during the follow-up period of 10 years. Harmful drinkers were significantly more likely to die than other subjects during the follow-up period.

A recent follow-up study from Scotland investigated long-term (13-year follow-up) mortality of subjects with TBI (McMillan et al. 2011). Subjects with TBI were compared with subjects having other injuries and with community controls. More than one year after the index trauma, deaths were 2.8 times more likely to occur among the subjects with TBI than among the community controls. Subjects with non-head injuries also showed an increased rate of late mortality, but the risk of death was greater after TBI than after other types of trauma. The rate of death was also higher after mild TBIs and especially among young adults. Previous admissions for brain illness, mental health treatment, a history of physical disability, habitual excess use of alcohol and living alone were associated with an increased risk of death.

Our study demonstrated that the alcohol price reduction and concomitant increase in alcohol consumption resulted in elevated mortality rates among harmful drinkers in a head trauma cohort. We also found that among those who were not identified as harmful drinkers, 9% of deaths occurred under the influence of alcohol and 17% had some alcohol-related cause of death recorded on the death certificate. Accordingly, alcohol-related harms easily remain unrecognised. Mcmillan et al. concluded that the reason for vulnerability to death after head trauma, especially among young adults, remains unclear and requires further consideration (McMillan et al. 2011). Our study suggests that alcohol consumption may be one potential risk factor for long-term mortality among subjects with head trauma.
6.8 Strengths and limitations

Several strengths of this study can be mentioned. This university hospital is the only trauma centre in this area with neurosurgical services, and all subjects with symptoms and signs suggesting TBI are usually admitted to this hospital. Accordingly, all subjects with moderate-to-severe TBIs within this defined area should be included in this study. In Finland, a medico-legal autopsy is required for all traumatic deaths according to the law, and therefore we were able to include all those who died from TBI regardless of whether the subject succumbed at home, on the scene or whether he/she was first treated in a hospital, health centre or other health care facility. The study represents the whole population of a defined area and reliably includes all subjects with moderate-to-severe TBIs.

The alcohol price reduction implemented in 2004 gave us an opportunity to investigate the effects of alcohol policy on TBI in a natural context. The data on subjects with TBI were collected systematically using a structured form before and after the alcohol price reduction, and case ascertainment was based on hospital charts and comprehensive medico-legal autopsy records, and not only on register-based data.

The follow-up data on our head trauma cohort were gathered from our own hospital records and supplemented with data obtained from the National Hospital Discharge Registry and the Statistics Finland Causes-of-Death Registry. These registries included all the recorded diagnoses and underlying and contributing causes of death and the data included visits to hospitals and inpatient treatment at health centres all over Finland. A majority (90%) of the subjects belonging to our cohort were living in our hospital catchment area and, therefore, their hospital charts were easily available and reviewed for case ascertainment. The follow-up was long enough to allow evaluation of the long-term changes in TBI mortality after the reduction of alcohol prices. Earlier studies have reported short-term changes in alcohol-related harms after the alcohol price reduction (Herttua et al. 2008, Herttua et al. 2008, Herttua et al. 2011, Koski et al. 2007), but we were able to see effects over a longer time period.

Several weaknesses should also be noted. A great majority of TBIs are mild and it has been evaluated that 82% of all medically attended subjects with TBI are not admitted to hospital (Jennett 1996). Many subjects with a mild TBI may not even seek medical attention, and the proportion of people suffering from TBI without seeking help is unclear (Faul et al. 2010). This happens also in our
catchment area. The number of subjects with mild TBI in our area who did not seek help is not known. Accordingly, we were not able to expand our observations to also include mild TBIs, and our observations cannot be generalised for all TBIs.

A limitation of our study may also be the rather small sample size. We cannot rule out the possibility that a small number of cases may have influenced the statistical power of the analyses, particularly when the analyses were almost significant. Accordingly, with a larger sample size the statistical power may have improved and some of the non-significant results may have become significant.

Alcohol involvement may have remained undocumented in hospital charts or alcohol measurement may have been neglected in some cases by health care providers. To minimise bias, alcohol-related contributory causes of death and notes of inebriation by health care providers were also taken into account. Some subjects may be admitted to hospital several hours after the trauma and alcohol attribution may be underestimated. The time lag from trauma to death may also be so long that alcohol measurement is not reasonable. Alcohol involvement accumulates on weekends, which is well-known among health care providers. This may cause self-amplified bias, i.e. alcohol will be measured more often on weekends and this may result in a higher proportion of alcohol-related traumas on weekends.

Harmful drinking was defined as having occurred if any medical event leading to a visit to a hospital or health centre during the follow-up period carried the diagnosis of harmful drinking, alcohol intoxication or any diagnosis of an alcohol-related disease (i.e. alcohol withdrawal seizures, for example). Probably first episodes of harmful drinking sometimes remained unrecorded in hospital charts, leading to underestimation of harmful drinkers. Therefore, in our first survival analyses, subjects were coded for harmful drinking during the whole follow-up period (1999–2010), not only for the period before the alcohol tax reduction. The main finding of this analysis was that harmful drinkers’ mortality increased significantly after the alcohol tax reduction. Because some of the subjects (n = 16) were recorded as being harmful drinkers after the alcohol tax reduction and because the tax reduction may have caused an increase in the number of harmful drinkers among the population, we repeated our analysis by placing these 16 subjects in the control group. This analysis confirmed the finding of the previous one, i.e. also in this analysis harmful drinkers showed increased mortality after the alcohol tax reduction.
Although, the data were collected the same way in each observation year, the before-after design has methodological weaknesses. Seasonal changes and possible long-term trends among the population cannot be controlled in the before-after design. The number of subjects in this study is rather small and random changes during the year may influence the incidence and mortality rates. There may also be other unknown risk factors for TBI and changes in those may have influenced the number of TBIs during the observation years. In addition to alcohol tax reduction, also economic booms and recessions have effects on alcohol consumption at the population level (Babor et al. 2010). In Finland, total alcohol consumption follows economic cycles (Huhtanen et al. 2011). After a severe depression (1991–1995) alcohol consumption and economic wealth begin to rise again. Unfortunately, we were unable to take into account education level, employment status, deprivation, ethnic group, income level and marital status. These parameters are usually not available in hospital records and to gather reliable data on them requires personal interviews of the subjects. Other possible unknown factors which may influence the occurrence of TBI and other variables which influence alcohol consumption should be taken into consideration when interpreting these results.
7 Conclusions

1. The overall incidence of moderate-to-severe TBIs did not increase after the reduction of alcohol prices.
2. Deaths from TBI were more commonly alcohol-related after the alcohol price reduction among middle-aged men, but not among women. The increase was mainly due to an increase in alcohol-related falls. At the same time, alcohol-related mortality from TBIs decreased among young men, but not among young women. The decrease among young men was mainly due to a decrease in suicides. Similar trends were seen also in the incidence rates of moderate-to-severe TBIs. Alcohol-related moderate-to-severe TBIs decreased among young adults, but increased among middle-aged people.
3. Harmful drinkers among head trauma subjects were significantly more likely to die than others after the reduction of alcohol prices.
4. Alcohol did not hamper the diagnostic accuracy of TBI. There was no significant difference in diagnostic accuracy between subjects who were under the influence of alcohol and sober subjects. Independent predictors for TBI diagnosis remaining undocumented were the specialty of the treating physician and the cause of trauma, but not inebriation of the subject.
5. The occurrence of head traumas occurring under the influence of alcohol clearly accumulated on weekends, and alcohol-related head traumas increased the total number of head traumas. The occurrence of alcohol-related head traumas also peaked in the most popular vacation month of July. Head traumas of sober subjects were evenly distributed according to the day of the week and the season of the year.
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