Incidence Trends and Risk Factors of Alcohol-Specific Deaths

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Abstract
A simple but questionable view holds that alcohol-related mortality is directly and closely related to the total consumption of alcohol in a country. To study how uniform this relationship is, trends and changes by age and diagnosis in alcohol-specific deaths were studied in Finland between the years 2007-2022, a period when both alcohol consumption and alcohol-specific mortality showed a decreasing trend. Alcoholic liver disease mortality trends deviated markedly from those for the other diagnoses. Among those aged 64 years or less, there was a consistent decreasing trend for the other categories while liver disease showed a slight increase after the initial decrease. Among the population aged 65 years or more both liver disease and other groups showed an increasing trend. Alcohol poisoning decreased in both age groups. Mortality in all other diagnostic groups decreased in the younger age group and increased in the older group. Age-adjustment or gender did not explain differences. The different mortality trends were not consistent with the assumed uniform effect of alcohol consumption on alcohol-related harms. Other risk factors than alcohol may play a significant role. These are discussed and need to be considered to yield better estimates than the simplistic assumption of a 100 % attributable fraction of alcohol in these diagnoses.

Keywords: Alcohol-Specific Mortality, Liver Disease, Alcohol Poisoning, Cardiomyopathy, Mental Disorders

1. Introduction
Alcohol-specific deaths are a group of etiologic diagnoses, that is, alcohol is mentioned in the disease name as a causal agent. A common view is that the total consumption of alcohol in a country determines how much harm is caused by drinking alcoholic beverages. If the above total consumption model is true, alcohol-related harms should move in concert with alcohol consumption over calendar time. This should be most evident for alcohol-specific deaths since these deaths are considered to be 100 % caused by alcohol [1]. All deaths in the categories comprising alcohol-specific diagnoses should follow a similar trend. The present analysis shows that this was not the case even if the overall trends in both total alcohol consumption and alcohol-specific mortality suggest the opposite in Finland, where alcohol consumption decreased steadily from 12 to 9 liters of 100% alcohol per capita and alcohol-specific mortality from 40 to 30 deaths per 100 000 of the population between the years 2007-2022.

2. Methodology
Alcohol-specific deaths were studied in Finland between the years 2007-2022. The above period was chosen since the trends of both alcohol consumption and alcohol-specific mortality started to decrease, after reaching a peak in the year 2007. Data were derived from the StatFin database kept by Statistics Finland (https://pxdata.stat.fi/PxWeb/pxweb/en/StatFin/). The three-digit ICD-10 codes were F10, G312, G4051, G6 21, G721, I426, K292, K70, K860, K852, 0354, P043, Q860 and X45. These are underlying causes of deaths, that is the disease or injury initiating the train of morbid events. Contributory causes of death were not included, because their causal role is unknown. The European standard population (ESP2012) was applied for age adjustment. Both age-adjusted and crude mortality showed a closely correlated decreasing trend, Because adjustment did not make any notable difference, the actual mortality rates for both genders combined will be presented. The proportion of male deaths was predominant (77 %) and male mortality was closely correlated with the respective female rates (r=0.87).

3. Results
Alcoholic liver disease was the largest group and its mortality trends deviated from the other diagnoses. Among those aged 64 years or less, there was a consistent decreasing trend for the other categories while liver disease showed a slight increase after the initial decrease (Figure 1). Among the population aged 65 years or more both liver disease and other groups showed an increasing trend (Figure 2). Alcohol poisoning was the next largest group,

decreasing among both age groups. Mortality in other diagnostic groups decreased in the younger age group and increased in the older group (Table).
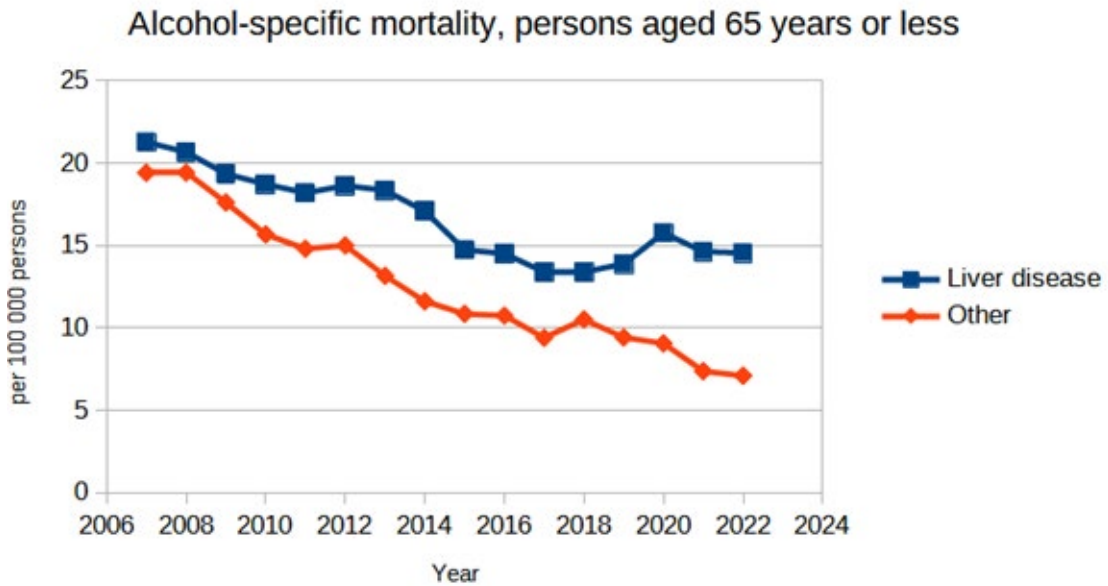

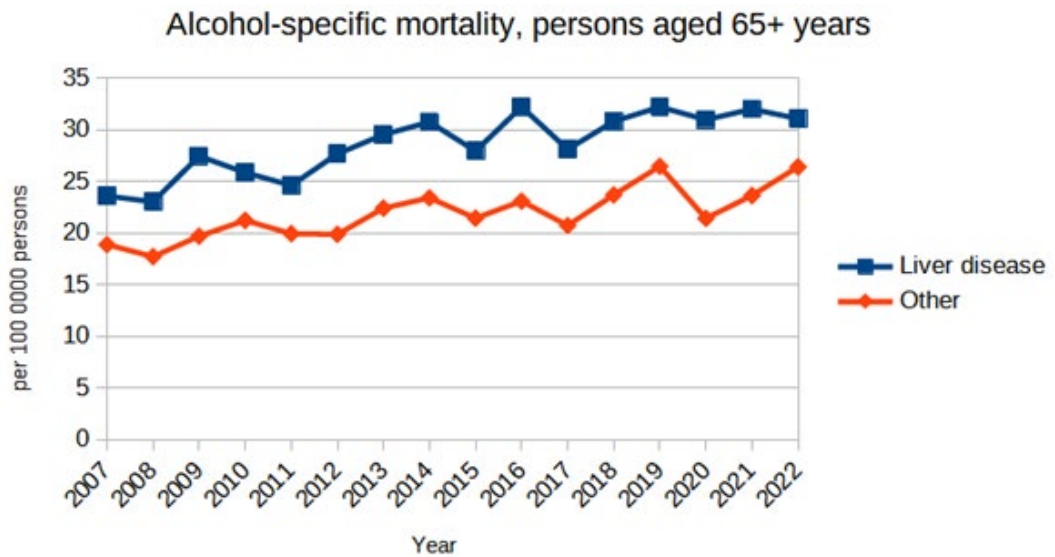**Figure 1:** Alcohol-Specific Mortality, per 100,000 Persons Aged 65 Years or Less, in 2007-2022 in Finland

Figure 2: Alcohol-Specific Mortality, per 100,000 Persons Aged 64 Years or more, in 2007-2022 in Finland

	Liver disease (K70)	Alcohol poisoning (X45)	Mental disorders (F10)	Cardiomyo-pathy (I426)	Other	Total
Under 65 years of age						
No. of deaths 2007	948	503	106	96	143	1796

Change % 2007 to 2022	*-34.4*	*-80.3*	*-36.7*	*-57.3*	*-32.9*	*-48.6*
65 years of age or older						
No. of deaths 2007	197	68	43	23	40	371
Change % 2007 to 2022	+103.5	-55.9	+320.9	+47.8	+137.5	+98.4

Table: Number of Alcohol-Specific Deaths (ICD-10 Codes) in 2007 and Percent Change from 2007 to 2022 by Age Group

4. Discussion

The alcohol-specific mortality trends were different for the liver disease group and the rest, as well as for the two age groups, those less than 65 years and those 65 years or older. Alcohol-specific deaths are a category of etiologic diagnoses, that is, alcohol is mentioned in the disease name as a causal agent. Therefore, alcohol is assumed to be a necessary cause. Not a sufficient cause, since death is caused by many factors. Alcohol may, or may not, be the decisive factor. The accuracy in ascertaining these deaths depends on the judgment of the cause-of-death determination and is often unknown. Attributions to alcohol may be under or over-estimated. Each revision of the ICD has provided more diagnoses with alcohol etiology which may have increased the number of alcohol-specific deaths.Other risk factors than alcohol may be related to alcohol-specific deaths. These have been earlier studied in toto (deaths and hospitalizations of all diagnoses). In addition to alcohol intake, smoking and marital status were found to increase the risk of occurrence [2]. However, focusing on the risk factors of separate diagnostic entities is likely to be a more elaborate strategy, but remains underutilized.

Risk factors for liver disease may include hepatitis infections, smoking, obesity, proton-pump inhibitor medication to decrease stomach acidity, malnutrition, some environmental poisons, like aflatoxin, and perhaps genetic factors [3,4]. C-type lectin (mincle) may contribute to inflammation and fibrosis once the intestinal barrier becomes leaky in the advanced stages of chronic liver disease [5]. Notably, in a large series of biopsy-confirmed alcoholic liver disease patients, the presence of excess weight for at least 10 years was a risk factor for cirrhosis, alcoholic hepatitis, and steatosis and consumption of 20 cigarettes/day has been found to relate to 4-fold risk increase among women in England [6,7]. Coffee consumption has decreased the risk of cirrhosis [8,9]. Fatal alcohol poisoning is alcohol-induced, but adequate evidence is often unavailable. The average lethal blood alcohol concentration is about 0.5 % but variation depends on innate and acquired tolerance. The blood alcohol level may decrease from the maximum concentration to the postmortal level for many hours between the onset of drinking and the moment of death. The time from the peak to the moment of death is seldom known [10,11]. Psychoactive drugs and conditions inducing inhalation of vomit complicate the ascertainment. Therefore, determining the underlying cause of death must often be based on a probability estimate after ruling out other possible causes.

The group alcohol-related disorders in the mental, behavioral, and neurodevelopmental disorders (ICD-10 chapter F10) is heterogeneous. It includes alcohol-related intoxication, abuse, dependence, withdrawal, dementia, psychotic disorder, and several alcohol-induced mental disorders, such as anxiety and depression, among others. Alcohol dependence may be the pivotal disorder that leads to several others. Risk factors and developmental causal paths for alcohol dependence are many and varied [12]. Major possible risk factors, either externalizing or internalizing mental problems, have been found in about 50 % of a large Swedish representative sample [13]. Cardiomyopathies comprise a heterogeneous group of diseases, often of genetic origin or caused by several heart and circulatory conditions (acquired cardiomyopathy). The main types are hypertrophic, dilated, and arrhythmia-induced cardiomyopathies [14]. In Finland, approximately one-third of hypertrophic cardiomyopathy is explained by the four most common genetic variants [15].

The role of alcohol as a causal agent is difficult to ascertain because both self-reports and reports from significant others are inaccurate, usually underestimate alcohol intake (but sometimes overestimate it) and variations in inaccuracy may be great [16-18]. Therefore, the under-representation of alcohol-specific deaths among deaths is sometimes suspected. In the present series, this does not seem likely since the proportion of alcohol-specific liver disease deaths (ICD-10 code K70) was as much as 86 % of all liver disease deaths (K70–K76). Other possible explanations for these observations exist. The age-group differences suggest that younger birth cohorts may drink less, have healthier drinking patterns, or have fewer other risk factors. Unfortunately, the present data are too sparse for further study of this possibility. Cause-of-death diagnostics could differ, but it is not likely because no major changes in the organization and personnel of cause-of-death examination have been observed during this short observation period. The association between alcohol-specific deaths is commonly thought to be linear, causal, and strong. Consequently, decreasing the total consumption is assumed to decrease harm. Decreasing consumption by tax hikes and limitation of supply of the beverages is thought to be strong and is marketed as a "best buy", by WHO and several other organizations [19]. This appealing and simple view seems to be false because of contrary scientific evidence [20]. The present

findings support the latter view, suggesting that other risk factors are important.

5. Conclusion
While "alcohol-specific" subcategories of disease and injury entities may be useful for basic cause-of-death statistics, differences in the present study call for studies on the other risk factors than alcohol in these subgroups. These should be considered if we want better estimates than the crude assumption of a 100 % attributable fraction of alcohol on the role of alcohol in these diagnoses. Diagnostic entities should not be split into alcohol-related and not-related etiologic categories in such studies.

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