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# Chapter V

## The Epidemiological Study of Alcohol-Related Problems

**Defining the Problems**

This report, to this point, has reviewed the use and immediate physiological effect of alcohol on humans. This helps one to understand the nature and immediate effect of the alcoholic beverages themselves; in other words the agent-man inter-relationships. A major factor which apparently causes alcoholic beverages to be controlled and taxed in a special manner is the presence of medical and social problems which appear to be related to alcohol's effects and its use or abuse. It is these overt negative reactions to alcoholic beverages which are of most concern to society. To deal effectively with these concerns requires one to have an appreciation of the relative seriousness of the different problems and their cause-effect relationships and, finally, to develop strategies which are acceptable and which will alter the cause-effect relationship in order to reduce the undesirable effects. This approach corresponds to the environmental aspects of the study.

The discussion which follows focuses on methods which are used in investigating such matters of concern to the health of a nation. Examination of the role of the environment by epidemiological methods is the study of the distribution (or variation) of a disease or condition between regions and populations and across time intervals in order to gain information (of the cause-effect relationships) on which to base preventive strategies. In other words, one attempts to investigate the causal factor or factors relating to a health problem by statistically relating the variation in the incidence of the problem to the variation in suspected causal factors (11,13,16,19). The statistical relationships also can often be supported by direct clinical evidence of the cause-effect relationship — but this is not always the case. Quite often the statistical evidence is not wholly accepted by policy-makers or the public until it has been irrefutably proven by directly observable tests.

In the case of some epidemiological studies, fairly direct relationships can be ascertained where a prominent cause-effect link appears to exist — as, say, in the instance of fluoridated water leading to reduced cavities in children's teeth or in the case of heavy smoking which leads to increased evidence of lung cancer. The epidemiological study of alcohol-related health problems is more complex. Large numbers of people consume alcoholic beverages without incurring any apparent disadvantages from them and apparently deriving satisfaction from drinking. However, there is a considerable minority who are adversely affected in many different ways. Often these disabilities are difficult to define in order to determine the extent and severity of the problem (4). They are also difficult to relate to their causative factors which may be inter-related in a complex way not yet understood.

Alcohol-related problems may be divided into acute effects and chronic effects. The acute effects may occur even though the individual is not a regular abuser of alcohol, or they may be manifestations of the longer-term overall condition called

alcoholism. The acute adverse effects of alcoholic beverage consumption can be described as:

—drunkenness and social disruption – culminating in arrests for drunkenness
—auto, industrial and other accidents, culminating in property loss, disability or death
—acts of violence, suicide and other anti-social problems.

The chronic effects are usually related to illness brought on by the effects of alcohol on the body, either by itself or in combination with the life-style of the drinker. The major chronic problems of sickness and death which are considered to be alcohol-related may be described as follows:

—alcoholism – (habitual drinking which causes severe social or health problems)
—cirrhosis of the liver
—delirium tremens and other alcoholic psychoses
—morbidity and mortality in excess of normal expectations from conditions such as heart disease
—other less frequent disorders as shown in Table 21.

It is difficult to place the different problems in perspective in terms of their seriousness. To the afflicted individual and his family it is serious even if his is the only case in the country, but in setting priorities for prevention and cure, the extent of the problem is important to policy-makers. In terms of the acute effects, the relative importance to Canadians seems to depend on the public awareness. As an example, automobile accidents are perceived as being serious – in 1970 they accounted for 3.5% of the Canadian death rate (over 5,000 deaths) (22) of which, it is estimated, half may be alcohol-related (5). However, there are few measures in Canada of other acute effects such as the extent to which violence, industrial accidents, etc. may be due to alcoholic beverage consumption.

In terms of the chronic conditions, it is estimated by some that perhaps 1 to 3 percent of the adult Canadian population are alcoholics (1). The heavy consumption of alcoholic beverages by these persons may result in extreme danger to themselves and severe social disruption to their families and the communities in which they live. In addition, most of the other chronic conditions (and many of the acute incidents) are incurred by alcoholics.

Liver cirrhosis is commonly associated with alcoholism and heavy drinking – this chronic disease caused 1,800 deaths in Canada in 1970 (22). Delirium tremens and other alcoholic psychoses are also commonly known and associated directly with alcoholism. These psychoses are a problem of morbidity; in Canada in 1970 thirty-three deaths from delirium tremens were reported (22). The foregoing diseases, the incidence of accidents and the increased mortality from more common diseases, all combine to shorten the lives of alcoholics by twelve years or more, according to some investigators. Table 22 illustrates the causes of death in some alcoholics in comparison to the normal expectancy in the general population. This study was done by Schmidt and de Lint (19) and followed up the mortality of 6,500 past alcoholic patients of the Addiction Research Foundation of Ontario. This is indicative of the types of illness affecting alcoholics, but care must be taken in



TABLE 21

ALCOHOL-RELATED DISORDERS

*Gastrointestinal*
- Esophagitis
- Esophageal carcinoma
- Gastritis
- Malabsorption
- Chronic diarrhea
- Pancreatitis
- Fatty liver
- Alcoholic hepatitis
- Cirrhosis (may lead to cancer of liver)

*Cardiac*
- Alcoholic cardiomyopathy
- Beriberi

*Skin*
- Rosacea
- Telangiectasia
- Rhinophyma
- Cutaneous ulcers

*Neurologic and psychiatric*
- Peripheral neuropathy
- Convulsive disorders
- Alcoholic hallucinosis
- Delirium tremens
- Wernicke's syndrome
- Korsakoff's psychosis
- Marchiafava's syndrome

*Muscle*
- Alcoholic myopathy

*Hematologic*
- Megaloblastic anemia

*Vitamin deficiency disease*
- Beriberi
- Pellagra
- Scurvy

*Metabolic*
- Alcoholic hypoglycemia
- Alcoholic hyperlipemia

Source: U.S. Department of Health, Education and Welfare. First Special Report to the U.S. Congress on Alcohol and Health. DHEW Publication No. (HSM) 72-9099. Washington, D.C.: U.S. Govt. Printing Office, 1971. p. 45.

TABLE 22

OBSERVED AND EXPECTED NUMBER OF DEATHS IN MEN AND WOMEN ALCOHOLICS BY MAJOR SPECIFIC CAUSES

| | MEN | | | WOMEN | | |
|---|---|---|---|---|---|---|
| | No. of Actual Deaths | No. of Expected Deaths | Ratio of Actual to Expected Deaths | No. of Actual Deaths | No. of Expected Deaths | Ratio of Actual to Expected Deaths |
| All Causes | 649 | 315.25 | 2.02 | 99 | 30.98 | 3.19 |
| Cancer of upper digestive and respiratory organs | 49† | 17.55 | 2.79 | 1 | 0.53 | 1.88 |
| Other malignant neoplasms | 28* | 40.56 | 0.69 | 9 | 9.47 | 0.95 |
| Alcoholism | 30† | 1.25 | 24.00 | 3 | 0.09 | 33.33 |
| Vascular lesions of central nervous system | 27 | 23.67 | 1.14 | 9* | 3.69 | 2.43 |
| Arteriosclerotic & degenerative heart disease | 230† | 128.86 | 1.74 | 28† | 6.82 | 4.10 |
| Pneumonia | 22† | 7.15 | 3.07 | 5† | 0.70 | 7.14 |
| Cirrhosis of the liver | 56† | 4.87 | 11.49 | 12† | 0.48 | 25.00 |
| Ulcer of stomach & duodenum | 13† | 3.66 | 3.55 | 1 | 0.15 | 6.66 |
| Accidents | 65† | 25.79 | 2.52 | 17† | 1.37 | 12.40 |
| Suicides | 47† | 7.80 | 6.02 | 4† | 0.46 | 8.69 |
| Other° | 72 | | | 10 | | |

*Significant at the .05 level. †Significant at the .01 level.

°These comprise: bacterial diseases (I.C.D. 053, 063), 2; diabetes mellitus (260), 8; allergic, endocrine, metabolic and nutritional diseases (241, 274, 286, 287, 289), 9; schizophrenic disorders (300), 1; epilepsy (353), 5; other diseases of the central nervous system (352 and 356), 4; chronic rheumatic heart disease (410–416), 10; other diseases of the heart (430–434), 4; hypertensive disease (443–447), 7; diseases of the arteries (450–456), 4; diseases of the veins (462), 2; bronchitis (502), 3; other respiratory diseases (519–527), 5; hernia (561), 2; intestinal obstruction (570), 2; other diseases of the intestines and peritoneum (578), 3; chronic nephritis (592), 2; infection of kidney (600), 3; hydronephrosis (601), 1; congenital malformation (759), 1; ill-defined condition (784), 1; homicides (982, 983), 3.

*Source:* Schmidt, W. and de Lint, J. *Causes of deaths of alcoholics.* Quart. J. Stud. Alc. 33: 1972.



ascribing this for use as a mortality indicator for all alcoholics (these were only the alcoholics who sought help and were already suffering severe medical disability at the time of their admission).

The authors note that the higher than expected mortality figures are not necessarily related directly to heavy alcohol consumption. For instance, the above average death rate from cancer of the upper digestive tract and respiratory organs may be related to the fact that alcoholics are usually heavy smokers. Similarly, the increased risk of death for arteriosclerotic heart disease might also be ascribed to smoking, rather than heavy alcohol use per se. The other diseases shown have similar complex relationships. The authors, in concluding, make the point that they have derived only preliminary statistical relationships which must be examined in more depth.

**A Framework for the Study of Alcoholism**

Alcoholism is the major alcohol-related problem in society. To aid in the analysis of the epidemiology of alcoholism and related problems, this report outlines a seven-stage system by which epidemiological research accumulates evidence. Based on these stages of development, the relative level of sophistication achieved in the epidemiology of alcoholism can be estimated and so help determine the degree of confidence to be placed in current hypotheses and recommendations:

1. identifying clearly a target disease or condition for study;
2. searching for and collecting relevant raw data;
3. producing disease distribution figures for various regions, populations, and times;
4. presenting selected trends, statistical associations, hypotheses;
5. rigorously testing promising hypotheses of cause-effect;
6. on the basis of hopefully strong evidence, designing preventive strategies and public policies;
7. continuing work on unresolved questions concerning the disease or condition under study.

It is not proposed that the foregoing represents a necessary progression, for in practice, work at any or all stages may be conducted simultaneously and stages may be skipped or their order reversed. Research is not nearly so neat an enterprise as is often believed (2). Nevertheless, research effort can be wasted by ignoring a stage or by adopting or slipping into an inappropriate research strategy.

The brief description which follows is designed to provide a simplified framework which will identify some of the major paths in the complex terrain of general epidemiology and, hopefully too, will help illuminate major areas in the relatively uncharted territory of the epidemiology of alcoholism. The aim in developing the framework is to untangle, in part, what Room refers to as "the snarl of accumulated literature" (20). Those seeking a comprehensive and detailed picture of epidemiological theory and methods can consult various references (11,13,16,19).

Summarized in Table 23 is the proposed seven-stage framework for organizing and evaluating epidemiological data on a particular disease or condition. Six stages are shown in the table and the seventh stage represents, of course, the need for continued research on unresolved problems. The table includes examples of both

TABLE 23

# FRAMEWORK FOR ORGANIZING AND EVALUATING EPIDEMIOLOGICAL DATA ON A PARTICULAR DISEASE OR CONDITION

| | | |
|---|---|---|
| 1.1 Target disease or condition identified and crude epidemiological model or theory implied. | 1.2 Disease or condition may be identified with varying degrees of clarity from vague to highly specific, and implicit epidemiological models may range from simple (one main cause, one main mechanism, and one main effect or disease) to very complex models assuming multiple causes, mechanisms and effects. | 1.3 The more vague or varied the definition of the target disease or condition the less likelihood there is of establishing strong support for any specific causal hypothesis.<br>Similarly, the more variable the causes, mechanisms, and effects the more difficult to establish strong support for any specific hypothesis. | 1.4 *Example:* Lung Cancer<br>Clarity of disease definition relatively high.<br>Implicit model:<br>multiple causes →<br>working through → → →<br>same mechanism →<br>to produce ↑ ↑ ↑<br>common effects cau. mech. ef. | 1.5 *Example:* Alcoholism<br>Clarity of disease definition relatively low.<br>Implicit model: →<br>one main cause → →<br>working through →<br>various mechanisms →<br>to produce ↑ ↑ →<br>various effects cau. mech. ef. |
| 2.1 Search for and collection of raw data from public records – data that is directly or indirectly relevant to target disease or condition. | 2.2 Must face questions concerning:<br>– reliability and accuracy of data<br>– comparability of data from different sources<br>– data gaps<br>– relevance of data to target disease or condition | 2.3 These questions arise because of frequently different and changing customs and standards of data collection, coding and retrieval, national, regional and temporal. | 2.4 *Example:* Lung Cancer<br>A relatively well standardized diagnostic category with data readily available through international sources (such as W.H.O.) as well as from regional sources for special studies. | 2.5 *Example:* Alcoholism<br>Major weakness in epidemiology of alcoholism is lack of standard definition or target variables.<br>As a result data collection, coding, and retrieval still relatively primitive and uncoordinated and research difficult to interpret. |
| 3.1 Production of disease distribution figures for different regions, populations, and times. | 3.2 Such figures serve to:<br>– help indicate the scope of the disease relative to others – its health "Cost" or threat.<br>– Also, if marked differences are noted between regions, populations, or time periods, these differences stimulate:<br>– generation of causal hypotheses<br>– critical examination of data base | 3.3 If possible figures are generated on:<br>*Incidence* (# of new cases arising in a given time period)<br>*Duration* (average duration of disease prior to cure or death)<br>*Prevalence* (# of cases in existence at a given point in time)<br>Calculated for different regions, ages, sexes, ethnic groups, etc. | 3.4 *Example:* Lung Cancer Graph<br>*Region:* England and Wales<br>*Population:* Males age 50–74<br>*Period:* 1911–1965<br>Shows rising incidence of disease and encourages investigators to isolate causal factors. | 3.5 *Example:* Alcoholism<br>Since no widely accepted and direct measure of alcoholism is established must rely heavily on one available indirect measure: liver cirrhosis.<br>→ →<br>→ →<br>→ → →<br>→ →<br>↑ ↑ ↑<br>cau. mech. ef. |
| 4.1 Extraction and critical examination of trends and statistical associations.<br>Generation of tentative hypotheses. | 4.2 If observed regional, temporal or population differences cannot be readily accounted for in terms of differences in diagnostic practices, or record keeping, and if not readily explicable on other grounds, then provide basis for serious examination. | 4.3 In terms of population differences, age, sex and ethnic variables are usually important either as control variables or cause-linked variables.<br>In terms of temporal differences, changes in diagnostic and record keeping practices are often important contributors to reported differences.<br>In terms of regional differences must try to sort out whether differences due to physical environment, social environment, diagnostic and record keeping practices, or various combinations. | 4.4 *Example:* Lung Cancer<br>Available data suggested:<br>– reliable increase in lung cancer rates over other cancer rates<br>– higher rates in urban areas<br>– higher rates among men than women<br>– higher rates among heavy smokers, asbestos workers, etc. | 4.5 *Example:* Alcoholism<br>Using readily available liver cirrhosis data as a crude estimate of alcoholism, data suggest:<br>→cirrhosis on increase<br>→rate higher among men<br>→rate higher in urban centers<br>→distribution appears to follow consumption levels for spirits and wine. |
| 5.1 Systematic testing of selected hypotheses arising out of four preceding stages. | 5.2 Data up to this stage is rarely strong evidence for any specific hypothesis. Rather the evidence is mild, open to a variety of interpretations, and based on:<br>– statistical associations and trends<br>– clinical reports<br>– anecdotal material. | 5.3 To provide strong evidence usually requires special studies of a large, representative group to determine:<br>a) what proportion of the sample<br>b) exposed to varying degrees of the suspected causal agent<br>c) suffer high risk morbidity or mortality consequences.<br>Note at stage 4 investigators don't know whether the people who are smoking are the same ones who are dying from lung cancer. They know only that both smoking and lung cancer are increasing in the country. | 5.4 *Example:* Lung Cancer<br>Doll & Hill, follow-up study of non-smokers and mild, moderate, and heavy smokers to determine risk of dying from lung cancer<br>Find strong direct relationship between lung cancer and number of *cigarettes* smoked | 5.5 *Example:* Alcoholism<br>No comparable study yet located. We need a follow-up study comparing mortality and morbidity in a large representative sample of individual people who drink different amounts and with clear beverage preferences. |
| 6.1 Public policy and preventive strategy stage. | 6.2 Once a significant factor in the "causal web" is located the problem becomes one of technical, social, and political feasibility of introducing preventive measures. | 6.3 Preventive measures are likely to be effective to the degree the technology is available and the change does not come in conflict with established habits and vested interests – water fluoridation is perhaps a good example. However, where strong habits or vested interests are involved change will be slow. | 6.4 *Example:* Lung Cancer<br>Even though the evidence linking some forms of lung cancer to cigarette smoking is very strong public policy changes are very mild. | 6.5 *Example:* Alcoholism<br>We are a long way from having strong evidence of the type we have linking lung cancer to amount and type of smoking – but if and when we do, preventive strategies will be at least as difficult, if not more difficult to implement. |

alcohol-related problems and problems relating to smoking for comparative purposes. These comparisons are made since epidemiological studies of smoking are among the most sophisticated and well known.

From the epidemiological viewpoint the implied cause-effect relationship leading to alcoholism may be summarized as one main cause (alcohol) working through a variety of mechanisms (genetic, psychological, social, etc.) to produce a wide variety of effects (delirium tremens, accidents, liver problems, social problems, addiction, etc.).

In investigating alcoholism, much basic work still remains to be done – certainly much more than is the case for smoking studies. This reconsideration of approach in the study of alcoholism is necessary because much of the current thinking regarding causes, mechanisms and effects is too vague to lead to the use of efficient research strategies. However, there is some evidence of increasing interest (18,26) in focusing on alcoholic beverage consumption, with important work now in progress in the U.S. Also, laboratory work originating in Sweden (9) of alcohol action in terms of blood level and behavioural indices is of great relevance and has been discussed earlier in this report.

## Quantitative Studies of Alcohol-Related Problems

In spite of the difficulty of defining alcoholism and studying its incidence epidemiologically, some quantitative studies have been carried out both on acute or immediate effects, as well as on long-term problems related to the use and abuse of each type of alcoholic beverage. As described earlier, most of the long-term or chronic effects are associated with either alcoholism or continuing heavy drinking over many years. Short-term complications may be associated with a moderate drinker who occasionally, or even once, participates in excess drinking which ends up in a drunkenness charge, an accident or violence. In both the short-term and long-term effects, epidemiological research has revealed some differences among beverage types and drinking patterns.

**Acute Problems**

The physiological studies described earlier relate directly to acute alcohol-related problems in that these problems are usually associated with the blood-alcohol level of the drinker. To the extent that the alcohol in lower strength beverages results in a lower intoxication effect on the drinker, one might expect fewer acute problems. The physiological studies carried out in Sweden and replicated in Canada have shown such differences; i.e. a significantly higher peak blood alcohol percentage is experienced when a drinker consumes the same amount of alcohol in distilled spirits rather than in beer. Although only a limited number of epidemiological studies by beverage type have been carried out, there are certain implications in terms of acute problems. Some of these studies are summarized below.

*Drunkenness*

Most studies of arrests for drunkenness related to the type of alcoholic beverage consumed, have been carried out in Europe. In the forefront of such research are investigators in the Scandinavian countries: Denmark, Finland, Norway and Sweden; but others, in countries such as the Netherlands and Poland, also have published data.



Nielsen (17) notes that while Denmark among the Scandinavian countries, has one of the highest per capita consumptions of absolute alcohol, it has the lowest consumption of distilled spirits and the lowest frequency of arrests for intoxication.

A study done by Patrik Tornudd in 1964 (25) shows that arrests for drunkenness were much lower in Denmark. The data are shown below and related to percent of alcohol consumed in beer:

| | Arrests in 1964 per 1,000 Inhabitants | Per Capita Consumption of Alcohol Imperial Gallons | Percent Consumed in Beer |
|---|---|---|---|
| Denmark | 4.8 | 1.131 | 72% |
| Norway | 11.5 | 0.592 | 45% |
| Sweden | 18.6 | 1.023 | 32% |
| Finland | 40.5 | 0.554 | 34% |

The data presented by these investigators indicate some differences in the apparent impairment effect of the different beverages. This in itself is probably not sufficient to fully explain the differences in drunkenness found in these countries. No doubt cultural differences, traditional beverage roles, differences in law enforcement, etc., are additional factors which affect the level of drunkenness and social disruption.

In a study conducted by the Finnish Foundation for Alcohol Studies on the frequency of drinking occasions and quantities consumed by type of beverage, Klaus Mäkelä (15) found that the use of each type of beverage is different. Over 70 percent of strong liquors were consumed on occasions when total consumption was over 10 centilitres of absolute alcohol (this would represent about nine ounces of Canadian whisky) whereas for beer it was only 36 percent.

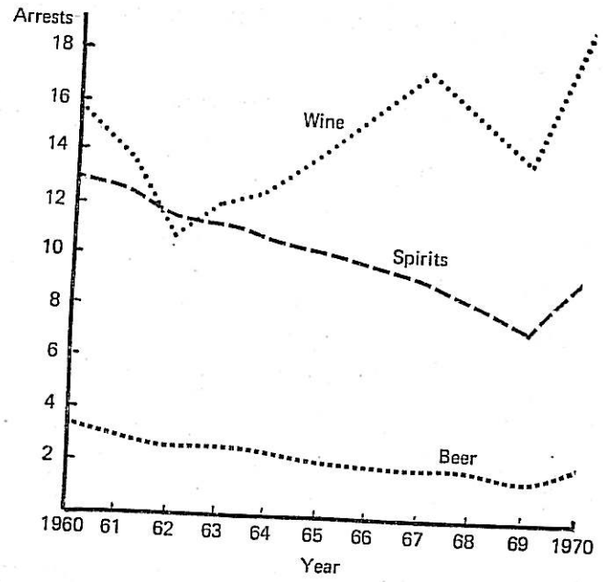
These Finnish data are borne out further by the published statistics of the Alcohol Monopoly on the arrests for drunkenness in urban areas according to the intoxicating compound used by the person arrested (3).

Figure 15 shows the number of arrests, classified as to each beverage, divided by Finland's total alcohol consumed annually in each of three beverages. The reader will note that the arrests for drunkenness of those drinking wine were highest per thousand litres of alcohol consumed in Finland (18 arrests per thousand litres of annual consumption in 1970), arrests of spirits drinkers were also high (9-10 arrests per thousand litres of alcohol consumed in spirits), and lowest for beer (only 2-3 arrests per thousand litres of alcohol consumed in beer in 1970). Beer sales represent 48 percent of the total alcohol sold, while those arrested for drunkenness after consuming beer represented only 11 percent of such arrests. The relatively higher rate of arrests related to wine drinking is not explainable by the alcoholic strength of the beverage alone and this requires further investigation.

According to Gadourek (7), differences in the short-term effects of each type of alcoholic beverage have also been found in the Netherlands. The per capita

FIGURE 15

FINLAND: NUMBER OF ARRESTS FOR DRUNKENNESS PER 1,000 LITRES OF ALCOHOL IN WINE, SPIRITS AND BEER




Based on Tables 19 and 23, Alcohol Statistics, Extract from the Alko Statistical Year Book, 1970, Helsinki, pp. 28 and 34.

consumption of distilled spirits dropped from 1.14 gallons in 1910 to 0.55 gallons in 1930. During this same period, the percentage of law violations under the influence of alcohol dropped from 10.7 percent to 4.1 percent. He finds there is a close correlation between these two variables. In a later period, between 1952 and 1956, the rate of public drunkenness decreased from 119 per 100,000 to 85 per 100,000 and this in spite of the fact that beer consumption (the major alcoholic beverage consumed in the Netherlands) and wine consumption had increased substantially as well as total alcohol consumption.

A. Swiecicki of Poland (23) has pointed out that although Italy has twice the per capita alcohol consumption of Poland, because of drinking patterns which prevail in Italy the amount of intoxication there is less than half that in Poland. He concludes that when spirits are consumed in large quantities, as in Poland, the harmful social effects are considerably worse than they are when drinking is in the form of low percentage alcoholic beverages drunk every day but in small doses, even though the annual consumption may be as high.

While the sociological environment of each country differs, the pattern in regard to social disruption arising from drunkenness shows some consistency – those areas where most alcohol is consumed in beer appear to have fewer arrests for public drunkenness than areas where other alcoholic beverages are consumed. The



statistical relationship of wine and arrests for drunkenness is not clear, based on the data available. Further study is required of both the amount of alcohol consumed in each beverage at each sitting and the differences in relative incidence of social disruption.

*Industrial and Other Accidents*

A search of the literature has failed to find any research on the relationship of type of alcoholic beverage consumed and risk of industrial and other accidents. *Alcohol and Health* (26) describes the general relationship of blood alcohol levels and accident cases as follows:

*. . . One study of over 300 consecutive fatalities resulting from nonhighway accidents that were seen by the coroner of Sacramento County, Calif., reveals that alcohol plays a major role in deaths from unintentional injuries. Among all these cases, 58 percent of the fatalities had at least one indication of being an alcohol abuser or alcoholic individual; 30 percent were known to be heavy drinkers or alcoholic persons. The study also showed that death from alcohol-related injuries occurs more often at home or during recreational events, than at work. Only 18 percent had taken place at work while 50 percent had taken place at home.*

*The role of alcohol in nonfatal accidents has been investigated by Wechsler and Associates. The Breathalyzer test was used to measure alcohol levels in patients admitted to the emergency service of a general hospital. Comparisons were made between the alcohol levels of patients in home accident injuries, and those admitted for injury resulting from transportation accidents, occupational accidents, fights, or assaults, as well as patients with medical conditions unrelated to accidental injuries.*

*Among accident victims, patients with injuries incurred in transportation had the highest frequency of positive blood alcohol levels (30 percent), and patients involved in accidents at home had the next highest (22 percent). Occupational accident cases had the lowest proportion with positive readings (16 percent).*

*Accidents and injuries result from a complex pattern of antecedent conditions. The role of alcohol as a causitive agent in many accidents may be suggestive but cannot be proved from statistical studies showing the association of drinking with accidents. The same conditions or personality characteristics which give rise to heavy drinking may also contribute to accident proneness, for example.*

Canadian studies carried out in Edmonton under the direction of Gilbert (8) have shown that 25 percent of patients being admitted to the emergency room of a large city hospital had been drinking, and that the average blood-alcohol concentration of these drinking cases was 0.15 percent. The sample indicated that almost 75 percent of these drinking patients had suffered lacerations, bruises, bumps or falls which appeared to be related to their state of intoxication.

The relationship of alcoholic beverage consumption and accidents may include socio-economic factors, since 28 percent of labourers and unemployed persons treated had positive blood-alcohol readings, while only 18 percent of other occupational groups showed positive readings. No data was gathered on the type of alcoholic beverage consumed, during the course of this Canadian study.

### *Traffic Accidents*

Most alcohol-related accident research has been conducted with regard to traffic accident risks. The risk of traffic accident connected with blood-alcohol levels has been briefly described earlier in this report. The exponential nature of the rise in risk as blood-alcohol levels rise and the concept of a threshold level of impairment were discussed. Studies of this type have shown that the risk of accident and particularly fatal accident is extremely high at blood-alcohol levels of 0.15 g/% and over – a level not normally attained by ordinary social drinkers. However, even at lower levels there is an increased risk attendant with drinking.

Lucas et al (14) reported on the results of a statistically valid Toronto Traffic Risk Study and related it to a study carried out by Holcomb in Evanston (10). Table 24 summarizes the relative accident risk due to increased blood-alcohol levels – the U.S. study showing a steeper rise in risk as blood-alcohol levels rise.

In all such studies, the presence of increased blood-alcohol levels increases the risk of accident. However, the type of beverage consumed has not been examined. To the extent that lower strength beverages tend to result in a lower blood-alcohol level, then they may be regarded as less hazardous. This is an area which requires further study to validate this hypothesis.

TABLE 24

THE ACCIDENT HAZARD ASSOCIATED WITH VARIOUS CONCENTRATIONS OF ALCOHOL

| % of Alcohol in Blood | Accident Drivers | | Non-Accident Drivers | | Ratio of % Accident to Non-Accident Drivers | Hazard | |
|---|---|---|---|---|---|---|---|
| | No. | % of Group | No. | % of Group | | | |
| *Toronto study* | | | | | | | |
| 0.0 up to 0.05 | 328 | 77.5 | 1,839 | 91.3 | 0.85 | | 1.0 |
| 0.05 up to 0.10 | 30 | 7.1 | 109 | 5.4 | 1.31 | | 1.5 |
| 0.10 up to 0.15 | 17 | 4.0 | 39 | 1.9 | 2.10 | | 2.5 |
| 0.15 and over | 48 | 11.3 | 28 | 1.4 | 8.10 | | 9.7 |
| Totals | 423 | | 2,015 | | | | |
| *Evanston study* | | | | | | | |
| No alcohol | 144 | 53.4 | 1,538 | 87.9 | 0.6 | 1.0 | |
| Trace up to 0.06 | 39 | 14.2 | 133 | 7.6 | 1.9 | 3.2 | 1.0 |
| 0.07 up to 0.10 | 28 | 10.4 | 56 | 3.2 | 3.3 | 5.5 | 4.7 |
| 0.11 up to 0.14 | 22 | 8.2 | 16 | 0.94 | 8.7 | 14.5 | 12.4 |
| 0.15 and above | 37 | 13.8 | 7 | 0.45 | 33.1 | 55.0 | 46.7 |
| Totals | 270 | | 1,750 | | | | |

*Source: Quantitative studies of the relationship between alcohol levels and motor vehicle accidents.* Lucas et al (1955), Proceedings of the Second International Conference on Alcohol and Road Traffic, The International Committee on Alcohol and Road Traffic, Toronto, 1955.

## Long-Term Alcohol-Related Problems

As described earlier, the long-term alcohol-related problems are usually associated with either alcoholism or heavy and continued drinking over a long period of years. The difficulty of studying variations in the level of alcoholism itself has already been discussed. The relationships of certain long-term effects with the type of alcoholic beverage consumed are discussed below.

### *Delirium Tremens and Other Alcoholic Psychoses and Neuroses (Including Deaths due to Alcoholism)*

Delirium tremens and other alcoholic psychoses are not usually a cause of death in Canada (33 deaths in 1970) (22) although the incidence of illness may not be low. Deaths classified as due to alcoholism are not easily definable – they are classified as a neurotic disease under the "mental illness" category and amounted to 256 deaths in Canada in 1970 (22). Little has been done in Canada to relate these morbidity and mortality figures to the type of alcoholic beverage.

A detailed study was conducted in Denmark on the effects of the drop in consumption of spirits after the very sharp increase in the tax on aquavit at the end of World War I. The frequency of delirium tremens cases dropped greatly, as shown below.

| Year | Cases of Delirium Tremens | Per Capita Consumption of Absolute Alcohol in Litres | Beer Consumption as a Percentage of Total Consumption |
|---|---|---|---|
| 1916 | 781 | 6.7 | 25% |
| 1918 | 51 | 1.6 | 70% |
| 1920 | 56 | 3.0 | 80% |
| 1935 | 28 | 2.4 | 75% |
| 1950 | 22 | 3.3 | 80% |
| 1960 | 61 | 4.2 | 70% |

In summing up the results of the tax increase, Johannes Nielsen of the Institute of Psychiatry, Århus State Hospital, Risskov, Denmark, who coordinated and wrote the report "Delirium Tremens in Copenhagen" (17) stated:

*The 35 fold rise in tax on "Akvavit" in 1917-1918, causing a fall to 1/16th of the frequency of delirium tremens is probably the best example ever given of the importance of alcohol-political planning in fighting alcoholism and the most serious complications of alcoholism such as delirium tremens and death . . .*

*Alcoholism is always a disease whatever type of liquor or beverage is used, but the frequency of complications and of serious sequelae of alcoholism depends to a great extent on the consumption of distilled spirits and not so much on the consumption of beer and wine. This fact ought always to be taken into consideration in the planning of alcohol-tax manipulations as it was in Denmark in 1917, so that the tax on distilled spirits ought to be comparatively higher than the tax on wine and beer . . . (this) was clearly shown in Denmark in 1917-1918 when the tax on distilled spirits rose from 0.60 kr. to 20.60 kr. per litre, and the frequency of*

*delirium tremens decreased from 27.2 per 100,000 to 1.7, and deaths from alcoholism from 12.0 per 100,000 population to 1.6.*

The type of research done in Denmark tends to be supported by world-wide clinical experience which indicates that delirium tremens cases are most associated with higher strength alcoholic beverages.

*Cirrhosis of the Liver*

Liver cirrhosis is the one example of a disease related to chronic high-alcohol intake that is susceptible to statistical analysis because mortality figures exist for a variety of countries. It has also been widely used as an indicator of the prevalence of alcoholism in different countries, although the relative importance of prolonged and excessive alcohol consumption in the etiology of this disease is yet to be established and may well vary appreciably from one country to another. Whether or not the risk of liver cirrhosis for an individual relates to his choice of a particular alcoholic beverage (12), it is of interest to examine whether the beverage mix in a population has any bearing on liver cirrhosis death rates.

A sample of twenty-nine countries, for which apparently reliable data on liver cirrhosis death rates and consumption of beer, wine and spirits were available, was selected from statistics of the World Health Organization (27) and Dutch Distillers Association (6). The basic consumption data were corrected, where necessary, to reflect the true alcohol content in the beverages. These data are analyzed herewith.

As shown in Figure 16 and Table 25, there appears to be a relationship between liver cirrhosis death rate and annual per capita consumption of alcohol. However, it is equally apparent that the liver cirrhosis death rate can only be roughly predicted from total alcohol consumption. For example, New Zealand, with a per capita consumption of alcohol of 1.56 gallons per year, had a liver cirrhosis death rate of 2.7 per 100,000 population, less than one-fifth that of the U.S.A., which consumed considerably less alcohol per capita, 1.28 gallons per year.

STEP 1 – (i) CATEGORIZATION BY BEVERAGE PREFERENCE

As a first step we may extract from the twenty-nine countries two groups which are mainly beer and wine drinking countries.

a) Beer countries (Consuming more than 70 percent of their alcohol in the form of beer)

| Beer Countries | Population (millions) | Liver Cirrhosis Deaths | Consumption (Million Gallons of Absolute Alcohol) | | | |
|---|---|---|---|---|---|---|
| | | | Beer | Wine | Spirits | Total |
| (1) Australia: | 11.810 | 604 | 15.234 | 2.362 | 2.716 | 20.312 |
| (4) Denmark: | 4.839 | 368 | 4.162 | 0.677 | 1.065 | 5.904 |
| (5) England: | 48.301 | 1,350 | 36.709 | 3.381 | 8.694 | 48.784 |
| (17) New Zealand: | 2.729 | 73 | 3.302 | 0.273 | 0.682 | 4.257 |
| | 67.679 | 2,395 | 59.407 | 6.693 | 13.157 | 79.257 |

FIGURE 16

LIVER CIRRHOSIS DEATH RATE
RELATED TO TOTAL ALCOHOL CONSUMPTION PER CAPITA
FOR 29 COUNTRIES
LINE OF BEST FIT

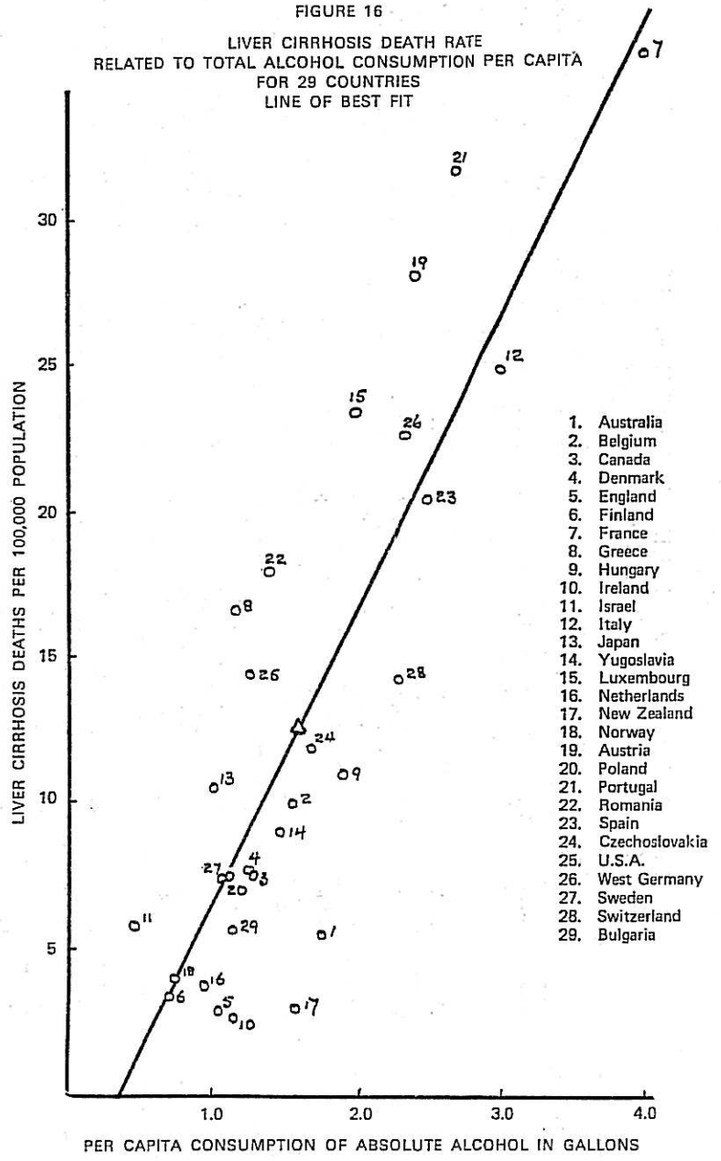


TABLE 25

PREDICTED LIVER CIRRHOSIS DEATH RATES
SELECTED INTERNATIONAL COUNTRIES 1967

| Country | | Per Capita Consumption of Absolute Alcohol in Gallons | | | | Liver Cirrhosis Deaths per 100,000 population | | | | Share of Total Consumption | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Name | Code | Beer | Wine | Spirit | Total | Actual | Estimated | Deviation | | Beer | Wine | Spirit |
| | | | | | | | | | % | % | % | % |
| New Zealand | 17 | 1.21 | 0.10 | 0.25 | 1.56 | 2.7 | 12.2 | −9.5 | −77.8 | 77.7 | 6.2 | 16.1 |
| Ireland | 10 | 0.68 | 0.05 | 0.43 | 1.16 | 2.6 | 8.1 | −5.5 | −67.9 | 59.0 | 3.9 | 37.1 |
| Australia | 1 | 1.29 | 0.20 | 0.23 | 1.72 | 5.1 | 13.9 | −8.8 | −63.2 | 75.0 | 11.6 | 13.4 |
| England | 5 | 0.76 | 0.07 | 0.18 | 1.01 | 2.8 | 6.6 | −3.8 | −57.5 | 75.3 | 6.9 | 17.8 |
| Netherlands | 16 | 0.49 | 0.11 | 0.36 | 0.96 | 3.5 | 6.1 | −2.6 | −42.3 | 51.0 | 11.5 | 37.5 |
| Bulgaria | 29 | 0.28 | 0.49 | 0.37 | 1.14 | 5.5 | 7.9 | −2.4 | −30.4 | 24.6 | 43.0 | 32.5 |
| Hungary | 9 | 0.55 | 0.90 | 0.41 | 1.86 | 10.8 | 15.3 | −4.5 | −29.5 | 29.5 | 48.5 | 22.0 |
| Switzerland | 28 | 0.84 | 1.02 | 0.40 | 2.25 | 14.0 | 19.3 | −5.3 | −27.6 | 37.2 | 45.1 | 17.7 |
| Canada | 3 | 0.75 | 0.10 | 0.45 | 1.30 | 7.2 | 9.5 | −2.3 | −24.5 | 57.6 | 7.7 | 34.6 |
| Yugoslavia | 14 | 0.19 | 0.70 | 0.57 | 1.46 | 8.9 | 11.1 | −2.2 | −20.1 | 12.8 | 48.1 | 39.2 |
| Belgium | 2 | 1.06 | 0.28 | 0.21 | 1.55 | 9.9 | 12.1 | −2.2 | −18.5 | 68.5 | 18.0 | 13.5 |
| Denmark | 4 | 0.86 | 0.14 | 0.22 | 1.22 | 7.6 | 8.7 | −1.1 | −12.8 | 70.5 | 11.5 | 18.1 |

TABLE 25—Continued

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Czechoslovakia | 24 | 1.01 | 0.37 | 0.29 | 1.67 | 11.8 | 13.3 | −1.5 | −11.6 | 60.6 | 22.0 | 17.4 |
| Italy | 12 | 0.11 | 2.51 | 0.35 | 2.97 | 24.8 | 26.7 | −1.9 | −7.0 | 3.7 | 84.5 | 11.8 |
| Sweden | 27 | 0.40 | 0.14 | 0.59 | 1.13 | 7.4 | 7.8 | −0.4 | −5.5 | 35.3 | 12.6 | 52.1 |
| Spain | 23 | 0.33 | 1.61 | 0.51 | 2.45 | 20.3 | 21.4 | −1.1 | −4.9 | 13.5 | 65.7 | 20.8 |
| France | 7 | 0.45 | 2.98 | 0.55 | 3.98 | 35.7 | 37.0 | −1.3 | −3.6 | 11.3 | 74.9 | 13.8 |
| Poland | 20 | 0.30 | 0.12 | 0.66 | 1.08 | 7.1 | 7.3 | −0.2 | −2.6 | 27.8 | 11.1 | 61.1 |
| Finland | 6 | 0.25 | 0.10 | 0.31 | 0.67 | 3.2 | 3.0 | 0.2 | 5.5 | 37.6 | 15.8 | 46.6 |
| Norway | 18 | 0.33 | 0.05 | 0.31 | 0.70 | 3.8 | 3.3 | 0.5 | 13.7 | 47.6 | 7.8 | 44.6 |
| Germany | 26 | 1.40 | 0.41 | 0.49 | 2.30 | 22.6 | 19.8 | 2.8 | 14.1 | 60.9 | 17.8 | 21.3 |
| Austria | 19 | 1.13 | 0.84 | 0.40 | 2.37 | 28.0 | 20.5 | 7.5 | 36.4 | 47.7 | 35.4 | 16.9 |
| Portugal | 21 | 0.09 | 2.44 | 0.11 | 2.64 | 31.8 | 23.3 | 8.5 | 36.5 | 3.4 | 92.4 | 4.2 |
| Luxembourg | 15 | 0.80 | 0.89 | 0.25 | 1.94 | 23.3 | 16.1 | 7.2 | 44.5 | 41.3 | 45.9 | 12.9 |
| United States | 25 | 0.59 | 0.10 | 0.59 | 1.28 | 14.1 | 9.3 | 4.8 | 51.2 | 45.9 | 8.0 | 46.1 |
| Japan | 13 | 0.27 | 0.39 | 0.33 | 1.00 | 10.4 | 6.4 | 4.0 | 62.0 | 27.5 | 39.3 | 33.2 |
| Romania | 22 | 0.17 | 0.84 | 0.38 | 1.39 | 17.9 | 10.5 | 7.4 | 70.9 | 12.5 | 60.2 | 27.3 |
| Greece | 8 | 0.10 | 0.97 | 0.05 | 1.12 | 16.5 | 7.7 | 8.8 | 114.1 | 9.0 | 86.5 | 4.5 |
| Israel | 11 | 0.12 | 0.10 | 0.22 | 0.44 | 5.6 | 0.7 | 4.9 | 689.8 | 26.6 | 23.3 | 50.2 |
| Mean | | 0.58 | 0.66 | 0.36 | 1.60 | 12.6 | 12.6 | −0.0 | | | | |
| Standard Deviation | | 0.39 | 0.79 | 0.15 | 0.77 | 9.4 | 7.9 | 4.9 | | | | |

Estimated: Liver Cirrhosis deaths per 100,000 population according to Figure 16.

b) Wine countries (Consuming more than 70 percent of their alcohol in the form of wine)

| Wine Countries | Population (millions) | Liver Cirrhosis Deaths | Consumption (Million Gallons of Absolute Alcohol) | | | |
|---|---|---|---|---|---|---|
| | | | Beer | Wine | Spirits | Total |
| (7) France: | 49.866 | 17,784 | 22.440 | 148.601 | 27.426 | 198.467 |
| (8) Greece: | 8.716 | 1,434 | 0.872 | 8.455 | 0.436 | 9.763 |
| (12) Italy: | 53.493 | 13,265 | 5.884 | 134.267 | 18.723 | 158.874 |
| (21) Portugal: | 9.415 | 2,997 | 0.847 | 22.973 | 1.036 | 24.856 |
| | 121.490 | 35,480 | 30.043 | 314.296 | 47.621 | 391.960 |

These two groups could be thought of as two amalgamations of countries with the following characteristics:

| Group | Population (millions) | Liver Cirrhosis Deaths | Consumption (Million Gallons of Absolute Alcohol) | | | |
|---|---|---|---|---|---|---|
| | | | Beer | Wine | Spirits | Total |
| Beer | 67.679 | 2,395 | 59.407 | 6.693 | 13.157 | 79.257 |
| Wine | 121.490 | 35,480 | 30.043 | 314.296 | 47.621 | 391.960 |
| | 189.169 | 37,875 | 89.450 | 320.989 | 60.778 | 471.217 |

From these basic data we may derive the following ratios:

| Group | Liver Cirrhosis Deaths per 100,000 population | Consumption of Absolute Alcohol | | Liver Cirrhosis Deaths per Million Gallons of Absolute Alcohol Consumed |
|---|---|---|---|---|
| | | Total in Millions of Gallons | Per capita in Gallons | |
| Beer | 3.539 | 79.257 | 1.171 | 30.218 |
| Wine | 29.204 | 391.960 | 3.226 | 90.519 |
| | 20.218 | 471.217 | 2.491 | 80.377 |

It may be seen from these figures that the wine group has both a higher cirrhosis death rate, a higher per capita absolute alcohol consumption, and a higher number of liver cirrhosis deaths per million gallons of alcohol consumed. It is unclear from these data at this stage whether the lower cirrhosis death rate depends upon beverage preference, total consumption or other factors.

### Step 1 – (ii) Categorization by Cultural Background

In order to study some of the factors other than total consumption and beverage mix, it is also instructive to group those countries which have more homogeneous cultural and economic backgrounds. Three international groups were considered. Assuming for this test that there are no differences except beverage-mix consumption between members of the groups, one might expect each of them to have the same liver cirrhosis death rate per gallon of alcohol consumed. The expected number of liver cirrhosis deaths may then be compared with the actual number occurring, (see Table 26) in order to arrive at the following analysis:

TABLE 26
LIVER CIRRHOSIS DEATHS BY GROUPS OF COUNTRIES
CATEGORIZED BY CULTURAL BACKGROUND

| Country | Total Consumption in millions of gallons | Per Capita Consumption of Absolute Alcohol in gallons | Liver Cirrhosis Deaths | | | Share of Total Consumption | | |
|---|---|---|---|---|---|---|---|---|
| | | | Expected | Actual | Difference | Beer % | Wine % | Spirits % |
| *English Speaking Countries* | | | | | | | | |
| New Zealand | 4.257 | 1.56 | 147 | 73 | −74 | 77.7 | 6.2 | 16.1 |
| England | 48.784 | 1.01 | 1,687 | 1,350 | −337 | 75.3 | 6.9 | 17.8 |
| Australia | 20.312 | 1.72 | 702 | 604 | −98 | 75.0 | 11.6 | 13.4 |
| Ireland | 3.362 | 1.16 | 116 | 76 | −40 | 59.0 | 3.9 | 34.1 |
| Canada | 26.321 | 1.30 | 911 | 1,460 | +549 | 57.6 | 7.7 | 34.6 |
| Group | 103.036 | 1.20 | 3,563 | 3,563 | | 70.4 | 7.8 | 21.9 |
| Liver cirrhosis death rate per million gallons of absolute alcohol consumed = 34.58 | | | | | | | | |
| *Scandinavian Countries* | | | | | | | | |
| Denmark | 5.904 | 1.22 | 358 | 368 | +10 | 70.5 | 11.5 | 18.1 |
| Norway | 2.611 | 0.69 | 158 | 142 | −16 | 47.6 | 7.8 | 44.6 |
| Finland | 3.079 | 0.66 | 187 | 149 | −38 | 37.6 | 15.8 | 46.6 |
| Sweden | 8.890 | 1.13 | 538 | 582 | +44 | 35.3 | 12.6 | 52.1 |
| Group | 20.484 | 0.95 | 1,241 | 1,241 | | 47.4 | 11.9 | 40.6 |
| Liver cirrhosis death rate per million gallons of absolute alcohol consumed = 60.58 | | | | | | | | |
| *Latin Countries* | | | | | | | | |
| Spain | 79.857 | 2.45 | 7,029 | 6,617 | −412 | 13.5 | 65.7 | 20.8 |
| France | 198.467 | 3.98 | 17,466 | 17,784 | +318 | 11.3 | 74.9 | 13.8 |
| Italy | 158.874 | 2.87 | 13,982 | 13,265 | −717 | 3.7 | 84.5 | 11.8 |
| Portugal | 24.856 | 2.64 | 2,186 | 2,997 | +811 | 3.4 | 92.4 | 4.2 |
| Group | 462.054 | 3.18 | 40,663 | 40,663 | | 8.6 | 77.5 | 13.9 |
| Liver cirrhosis death rate per million gallons of absolute alcohol consumed = 88.00 | | | | | | | | |

Note: Due to rounding, percentages do not necessarily add to 100.0%.

a) English-Speaking Countries (assuming for the purpose of this study that Canada can be so described)

There is a significant difference between the number of liver cirrhosis cases occurring within the countries comprising this group. The deviation of the actual number of deaths varies from the expected number, with the negative deviations occurring most often in those countries with a high share of total consumption of alcohol in the form of beer. It is reasonable to assume that these may be related since, for instance, a high beer consumption country (England: 75.3%) has a much lower rate of liver cirrohsis deaths than does a moderately low country such as Canada (57.6%). This supports the previous inference that there is a connection between beverage mix and cirrhosis and that beer countries appear to have a lower cirrhosis death rate. This connection has been indicated by Terris in his paper *Epidemiology of cirrhosis of the liver: National mortality data* (24) where he examined English, Canadian and other data.

It should be noted that England's crude death rate exceeds Canada's mainly because of age distribution differences (see Figure 19). The values of this rate and that of liver cirrhosis for both Canada and England are:

| Country | Crude Deaths Per 100,000 Population | Liver cirrhosis deaths Per 100,000 Population | Ratio |
|---|---|---|---|
| Canada | 740 | 7.2 | 0.97 |
| England | 1,130 | 2.8 | 0.25 |

Thus cirrhosis is shown to be a much more frequent cause of death in Canada than it is in England and the difference between the two countries thus appears even more marked.

b) Scandinavian Countries

The beverage mix does not appear to be affecting the liver cirrhosis death rate within this group of countries. There is a difference between countries, but no apparent relation with the mix of beverage. Sweden has a greater-than-expected rate, Denmark is only slightly above, Norway is somewhat below and Finland has a much lower rate than expected. In this case even when comparing the two high consumption countries, Sweden and Denmark, there is only a slightly higher death rate for Sweden (a spirits drinking country) – but, of course, Sweden does have a lower per capita consumption.

c) Latin Countries

Once again, the beverage mix is not wholly the explanation for the differences between the members of the group, although, in this group the countries all tend to have very high death rates and other than Portugal there is little deviation from expectations. In the case of Portugal the death rate is almost 40 percent higher than expected – which could be due to the understatement of consumption figures, or to the fact that they consume 92 percent of their alcohol in the form of wine.

Summary

Of the three international groups, only one has differences within the group which could be attributable to beer consumption. The differences within the other two groups need other explanations. However, the between-groups variation is indicative:

| Group | Per Capita Consumption of Absolute Alcohol in Gallons | Liver Cirrhosis Deaths per Million Gallons of Absolute Alcohol Consumed | Share of Total Consumption | | |
|---|---|---|---|---|---|
| | | | Beer % | Wine % | Spirits % |
| English-speaking | 1.20 | 34.58 | 70.4 | 7.8 | 21.9 |
| Scandinavian | 0.95 | 60.58 | 47.4 | 11.9 | 40.6 |
| Latin | 3.18 | 88.00 | 8.6 | 77.5 | 13.9 |

As the relative proportion of beer consumed by the groups is reduced, the frequency of liver cirrhosois deaths per million gallons of absolute alcohol consumed increases markedly. As the consumption per capita does not rise in the same proportion, this cannot be fully explained by this factor.

STEP 2 – REGRESSION ANALYSIS

A method of assessing the statistical relationship between the liver cirrhosis death rate and per capita consumption of alcohol is to determine a line of best fit to see to what extent it is possible to predict the cirrhosis death rate for a specific country from its total alcohol consumption.

*STAGE 1 ANALYSIS*

The first assumption is that liver cirrhosis is linearly related to alcohol consumption and may be expressed by a straight line whose equation is:

Liver cirrhosis rate = constant + coefficient × total alcohol consumption + all other factors

i.e. $LC = c + mT + e$

The quantity $m$ may be thought of as the rate of increase in cirrhosis for a unit increase in consumption. The value $e$ represents all the other factors which could conceivably affect the liver cirrhosis rate other than the total consumption of alcohol and will necessarily vary with individual countries.

With least squares regression analysis the values for c and $m$ are found to be:

| | Value | Standard Error | F – ratio | F – prob |
|---|---|---|---|---|
| c | −3.78 | ±2.171 | | |
| $m$ | 10.25 | ±1.228 | 69.71 | 0.0000 |

$R^2 = .721$

These results (see Figure 16) show that there is a statistically significant increase in liver cirrhosis death rate with increase of total per capita alcohol consumption. The value of $R^2$ indicates that 72.1 percent of the total variance of liver cirrhosis is accounted for by a linear relation of liver cirrhosis to alcohol consumption. The probability (F – prob) of this relation being due to chance is less than 1:10,000.

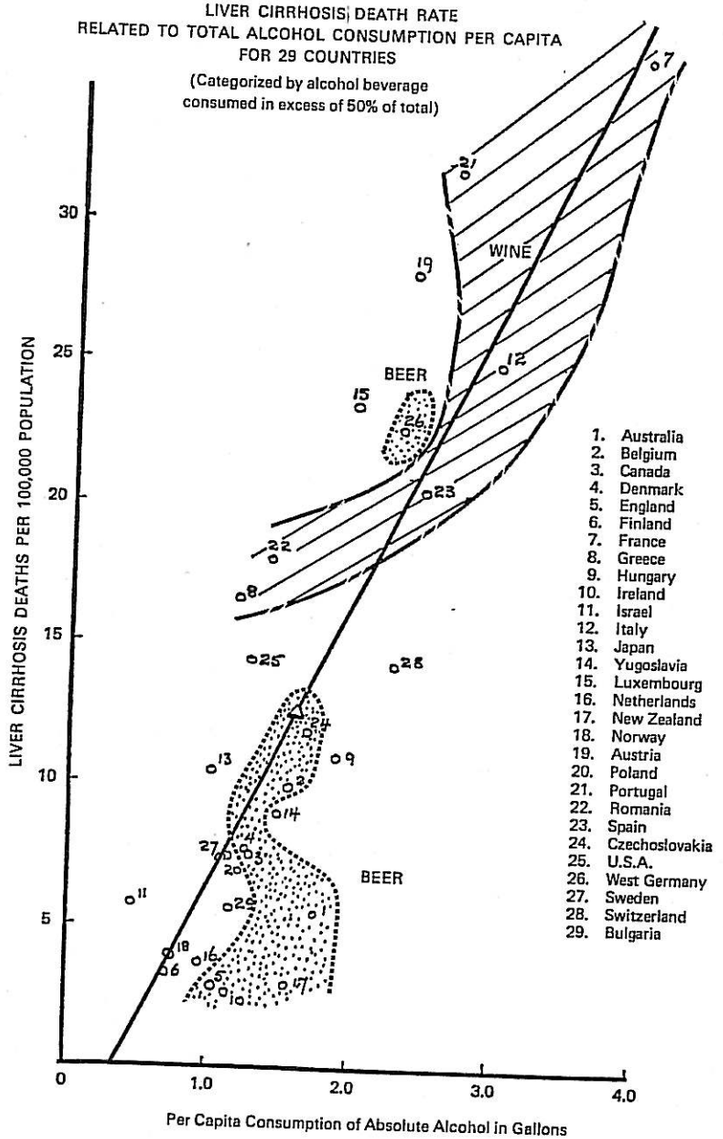
The slope of the line would indicate that liver cirrhosis increases by 10.25 cases per 100,000 population for every additional gallon of pure alcohol consumed per year per person. The deviations between observed liver cirrhosis and liver cirrhosis predicted by the equation are shown in Table 25.

There remain large deviations (shown in Table 25) between observed liver cirrhosis deaths and liver cirrhosis deaths predicted by the equation. These deviations could well reflect factors such as accuracy and completeness of diagnosis of the disease, general health and nutritional status of the community, percentage of drinkers and distribution of alcohol consumption in the drinking population. Nevertheless, it is notable that all countries with more than 50 percent of alcohol consumption in the form of beer, with the sole exception of West Germany, have an observed rate of liver cirrhosis lower than the predicted (see Figure 17).

FIGURE 17

LIVER CIRRHOSIS DEATH RATE
RELATED TO TOTAL ALCOHOL CONSUMPTION PER CAPITA
FOR 29 COUNTRIES

(Categorized by alcohol beverage consumed in excess of 50% of total)






*STAGE 2 ANALYSIS*

One way of testing the hypothesis that these deviations are related to the beverage mix is to expand the equation already used to include the per capita consumptions of beer, wine and spirits for the individual countries as follows:

$$LC = c + mT + a_bB + a_wW + a_sS + e$$

where $a_b$, $a_w$ and $a_s$ are coefficients and B, W and S are per capita consumptions of alcohol in the form of beer, wine and spirits respectively. On this basis, the values for c, $m$, etc. are:

| | Value | Standard Error | F-ratio | F-prob |
|---|---|---|---|---|
| c | .23 | ±2.58 | | |
| $m$ | 5.56 | ±2.22 | 6.26 | .018 |
| $a_w$ | 5.31 | ±2.17 | 6.01 | .020 |

$R^2 = .773$

The terms for $a_b$ and $a_s$ are not significant; thus it appears that consumption of alcohol in the form of wine is associated with more cirrhosis ($p = .020$) than consumption in the form of beer or spirits.

*STAGE 3 ANALYSIS*

In order to ascertain the relative values of the coefficients pertaining to each of the beverages individually it is necessary to omit the term for total consumption ($mT$). That is, on the basis of the model

$LC = c + m_bB + m_wW + m_sS + e$, the values of the constants are:

| | Value | Standard Error | F-ratio | F-prob |
|---|---|---|---|---|
| $c$ | 2.21 | ±1.98 | | |
| $m_b$ | 5.59 | ±2.39 | 5.56 | .0249 |
| $m_w$ | 10.88 | ±1.19 | 85.69 | .0000 |

$R^2 = .768$

The term $m_s$ is not signicant ($p = .46$). Thus, whereas the previous analysis (stage 2) indicated that there is no reason to believe that the amount of liver cirrhosis related to alcohol consumption is different for spirits and beer, it now appears that variations in per capita consumption of wine and beer are reflected in liver cirrhosis death rates while variations in per capita consumption of spirits are not so reflected.

However, this conclusion is in fact not valid, as shown below.

*STAGE 4 ANALYSIS*

The countries in Table 25 vary considerably in population, from Luxembourg (population 335,000) upwards; if countries are arbitrarily grouped together to make larger jurisdictions (e.g. EEC, EFTA, Commonwealth, Mediterranean), or divided (U.S.A. into states, Canada into provinces) to give more data points, coef-

ficients found by the foregoing analysis are considerably altered. This difficulty can be avoided by weighting each set of data points according to the population of the political entity. In principle, such weighting allows breakdown of countries into smaller jurisdictions without altering the final result if, within countries, there is a uniform distribution of consumption of beverages and liver cirrhosis. To the extent that variation exists within countries including this information by breakdown and weighting will add to the accuracy and validity of results.

Conversely, failure to break down countries into smaller units simply represents absence of information (in effect, it is assumed that within each jurisdiction LC, B, W and S are constant). With weighting according to population and now including data for 50 U.S. states, the District of Columbia, and the 10 Canadian provinces, (see Table 27), one finds with the original model used in stage 1, the result becomes:

$$LC = c + mT + e$$

| | Value | Standard Error | F-ratio | F-prob |
|---|---|---|---|---|
| $c$ | −1.20 | ±1.47 | | |
| $m$ | 9.42 | ± .48 | 385.2 | 0.0000 |

$R^2 = .8175$

Thus, 82 percent of the variance of liver cirrhosis deaths is now accounted for by a linear relation between liver cirrhosis and alcohol consumption.

*STAGE 5 ANALYSIS*

Proceeding further, with the expanded model as in stage 2, based on the 88 jurisdictions and taking into account the consumption of wine, beer and spirits as well as total consumption, and weighting as in stage 4, the new results are:

$$LC = c + mT + a_bB + a_wW + a_sS + e$$

| | Value | Standard Error | F-ratio | F-prob |
|---|---|---|---|---|
| c | −3.09 | ±1.60 | | |
| $m$ | 9.15 | ± .48 | 369.5 | 0.0000 |
| $a_s$ | 5.09 | ±1.99 | 6.5 | 0.0119 |

$R^2 = .8305$

The terms for $a_b$ and $a_w$ are not significant ($p = .14$), i.e., there is no significant difference in the extent by which the liver cirrhosis death rate alters with variations in wine and beer consumption. However, for each per-gallon increment of alcohol consumed per capita per year in the form of spirits there are 5.09 more liver cirrhosis deaths (per 100,000 population) than for the same increment of alcohol taken in the form of wines or beer. The probability for the term $a_s$ not being greater than zero is only .0119. This means that there is only one chance in ninety that differences in liver cirrhosis between jurisdictions are not related (directly or indirectly) to differences in beverage mix. Alcohol in the form of spirits is associated with *more* cirrhosis than the same amount of alcohol in the form of beer or wine.

*STAGE 6 ANALYSIS*

To obtain separate coefficients for beer and wine one now omits from the equation of stage 5, the term for total alcohol consumption ($mT$). Thus:

$$LC = c + m_bB + m_wW + m_sS + e$$

| | Value | Standard Error | F-ratio | F-prob |
|---|---|---|---|---|
| c | −2.48 | ±1.68 | | |
| $m_b$ | 7.84 | ±1.19 | 43.72 | 0.0000 |
| $m_w$ | 9.17 | ± .48 | 372.53 | 0.0000 |
| $m_s$ | 14.52 | ±1.95 | 55.35 | 0.0000 |

$R^2 = .8334$

That is, liver cirrhosis is significantly and positively correlated with the consumption of each beverage.* The difference between spirits and other beverages is now even greater than that found in the stage 5 analysis (+5.35 as against wine, +6.68 as against beer).

*STAGE 7 ANAYLSIS*

The above equation still does not account at all completely for variation of liver cirrhosis between countries, thus, for England and Wales, where there is a heavy preponderance of beer in the beverage mix, the predicted liver cirrhosis per 100,000 population is 7.0 whereas observed liver cirrhosis is 2.8, and for Australia and New Zealand the discrepancies are even larger. In order to obtain an equation more descriptive of the relation between liver cirrhosis and beverage consumption the data were fitted to a still more complex model in which was included the possibility that the coefficients for beer and spirits in the above equation are themselves functions of the consumption pattern in the country. On this basis a rather complex equation** is derived, which is characterized by a slope of alteration of liver cirrhosis with beer that is a rather steep function of spirits consumption. Conversely the slope of liver cirrhosis versus spirits increases rapidly with beer consumption, but becomes less as wine consumption increases. With the Canadian consumption pattern for 1967, it is found that the slope of liver cirrhosis versus beer alcohol is 8.6, the slope of liver cirrhosis versus alcohol in wine is 19.2 and of liver cirrhosis versus alcohol in spirits is 22.6. Thus, it would appear from this equation that the differences between beverages are even more marked than appeared in the previous analysis. The tables below show how liver cirrhosis rates might be expected to change with alteration of both beer and spirit consumption, with total consumption held constant, according to the equations re-

*A correlation of liver cirrhosis with beer, wine or spirits alone gives quite different values, because of the negative correlation between beer and wine. This fact is taken into account by the multiple regression analysis of the Stage-6 model.

**$LC = -.48 + 6.82S + 27.00W - 4.18B + 28.47B \times S - 10.23\ W \times S - 13.72W^2 + 3.28W^3 - 5.12S^2$.
$R^2 = 0.912$: coefficients of B, W, B × S, W × S, $W^3$ and $S^2$ are all significant at the 1% level; others must be included for the final regression equation – these coefficients become highly significant if "higher" terms are omitted.

TABLE 27

PER CAPITA CONSUMPTION OF ALCOHOL IN BEER, SPIRITS AND WINE AND LIVER CIRRHOSIS DEATH RATE

Canada/United States of America 1967–1968

| Province/State | Per Capita Consumption of Absolute Alcohol In Gallons | | | | Liver Cirrhosis Deaths per 100,000 Population |
|---|---|---|---|---|---|
| | Beer | Wine | Spirits | Total | |
| Alberta | .721 | .124 | .463 | 1.308 | 5.17 |
| British Columbia | .762 | .164 | .614 | 1.540 | 8.94 |
| Manitoba | .723 | .102 | .435 | 1.260 | 6.26 |
| New Brunswick | .475 | .088 | .347 | .910 | 4.20 |
| Newfoundland | .470 | .019 | .291 | .780 | 3.12 |
| Nova Scotia | .527 | .089 | .433 | 1.049 | 3.87 |
| Ontario | .794 | .104 | .525 | 1.423 | 7.98 |
| Prince Edward Is. | .408 | .072 | .477 | .957 | 4.32 |
| Quebec | .806 | .103 | .333 | 1.242 | 7.67 |
| Saskatchewan | .644 | .106 | .412 | 1.162 | 4.35 |
| Alabama | .318 | .042 | .367 | .727 | 7.00 |
| Alaska | .577 | .150 | 1.090 | 1.817 | 9.50 |
| Arizona | .716 | .168 | .551 | 1.435 | 14.30 |
| Arkansas | .378 | .072 | .307 | .757 | 7.70 |
| California | .637 | .343 | .821 | 1.801 | 21.50 |
| Colorado | .637 | .161 | .667 | 1.465 | 15.50 |
| Connecticut | .607 | .188 | .929 | 1.724 | 17.90 |
| Delaware | .674 | .136 | 1.004 | 1.814 | 14.40 |
| Dist. of Columbia | .851 | .453 | 2.743 | 4.047 | 41.70 |
| Florida | .626 | .168 | .974 | 1.768 | 16.50 |
| Georgia | .382 | .072 | .517 | .971 | 8.80 |
| Hawaii | .570 | .102 | .686 | 1.358 | 8.10 |
| Idaho | .607 | .050 | .412 | 1.069 | 7.70 |
| Illinois | .716 | .139 | .802 | 1.657 | 15.90 |
| Indiana | .588 | .059 | .397 | 1.044 | 11.50 |
| Iowa | .618 | .031 | .401 | 1.050 | 8.40 |
| Kansas | .453 | .050 | .390 | .893 | 8.40 |
| Kentucky | .517 | .046 | .483 | 1.046 | 9.40 |
| Louisiana | .615 | .160 | .513 | 1.288 | 9.80 |
| Maine | .686 | .052 | .588 | 1.326 | 11.70 |
| Maryland | .749 | .133 | .761 | 1.643 | 18.00 |
| Massachusetts | .686 | .164 | .821 | 1.671 | 18.20 |
| Michigan | .798 | .116 | .584 | 1.498 | 14.40 |
| Minnesota | .637 | .086 | .678 | 1.401 | 9.10 |
| Mississippi | .375 | .034 | .326 | .735 | 6.10 |
| Missouri | .652 | .102 | .592 | 1.346 | 12.60 |
| Montana | .813 | .074 | .562 | 1.449 | 11.60 |



TABLE 27—Continued

| Province/State | Per Capita Consumption of Absolute Alcohol In Gallons | | | | Liver Cirrhosis Deaths per 100,000 Population |
|---|---|---|---|---|---|
| | Beer | Wine | Spirits | Total | |
| Nebraska | .734 | .072 | .566 | 1.372 | 9.80 |
| Nevada | .959 | .320 | 1.727 | 3.006 | 22.70 |
| New Hampshire | .896 | .127 | 1.375 | 2.398 | 12.80 |
| New Jersey | .708 | .208 | .847 | 1.763 | 17.50 |
| New Mexico | .611 | .191 | .476 | 1.278 | 16.40 |
| New York | .708 | .225 | .851 | 1.784 | 24.20 |
| North Carolina | .356 | .059 | .517 | .932 | 8.80 |
| North Dakota | .659 | .058 | .558 | 1.275 | 7.70 |
| Ohio | .701 | .102 | .491 | 1.294 | 12.90 |
| Oklahoma | .420 | .084 | .461 | .965 | 12.80 |
| Oregon | .659 | .198 | .551 | 1.408 | 12.60 |
| Pennsylvania | .768 | .110 | .495 | 1.373 | 14.50 |
| Rhode Island | .802 | .202 | .719 | 1.723 | 18.10 |
| South Carolina | .408 | .072 | .629 | 1.109 | 7.10 |
| South Dakota | .521 | .074 | .543 | 1.138 | 8.50 |
| Tennessee | .446 | .045 | .333 | .824 | 7.40 |
| Texas | .719 | .089 | .423 | 1.231 | 10.90 |
| Utah | .360 | .062 | .322 | .744 | 7.80 |
| Vermont | .794 | .202 | .993 | 1.989 | 10.10 |
| Virginia | .562 | .116 | .600 | 1.278 | 11.60 |
| Washington | .682 | .180 | .641 | 1.503 | 13.40 |
| West Virginia | .468 | .052 | .382 | .902 | 12.20 |
| Wisconsin | 1.019 | .112 | .693 | 1.824 | 11.10 |
| Wyoming | .667 | .084 | .663 | 1.414 | 12.10 |

*Source:* Canadian consumption data based on figures appearing in: Dominion Bureau of Statistics. *The Control and Sale of Alcoholic Beverages in Canada 1967* (*Fiscal Year Ended March 31, 1968*). Catalogue No. 63–202. Ottawa: Queen's Printer, 1969. Liver cirrhosis death rates in Canada – two-year moving average centred at 1967 based on figures appearing in: Dominion Bureau of Statistics. *Causes of Death, Canada. Provinces by Sex and Canada by Sex and Age.* Catalogue No. 84–203. Ottawa: Queen's Printer, for selected years. United States consumption data based on figures appearing in: *Brewing Industry in the United States. Brewers Almanac 1971* Washington, D.C.: United States Brewers Association, Inc., 1971. Liver cirrhosis death rates in the United States in: U.S. Department of Commerce. *Statistical Abstracts of the United States, 1972.* Washington, D.C.: U.S. Govt. Printing Office, 1972, p. 60.

*Note:* In Canada consumption figures have been converted to alcohol at 5% absolute alcohol by volume for beer, 16% for wine, and 40% for spirits. In the United States, they have been converted at 4.5% for beer, 17% for wine and 45% for spirits, which are the strengths used for conversion purposes in: Efron, V. and Keller, M. *Selected Statistics on Consumption of Alcohol (1850–1968) and on Alcoholism (1930–1968).* New Brunswick, N.J.: Publications Division, Rutgers Center of Alcohol Studies, 1970.

sulting from the stage 6 and stage 7 analysis. It is here assumed that whatever factors keep liver cirrhosis lower in Canada than predicted* will remain constant.

| Per Capita Consumption of Absolute Alcohol in Gallons | | | | Predicted Change in Liver Cirrhosis Death Rate, 1967 = 100 | |
|---|---|---|---|---|---|
| Beer | Wine | Spirits | Total | Stage 6 Equation | Stage 7 Equation |
| .55 | .10 | .65 | 1.3 | 112.3 | 114.2 |
| .65 | ,, | .55 | ,, | 106.2 | 110.4 |
| .75 | ,, | .45 | ,, | 100 | 100 |
| .85 | ,, | .35 | ,, | 93.8 | 83.0 |
| .95 | ,, | .25 | ,, | 87.7 | 59.3 |
| .55 | .10 | .75 | 1.4 | 125.7 | 128.3 |
| .65 | ,, | .65 | ,, | 119.6 | 128.3 |
| .75 | ,, | .55 | ,, | 113.4 | 121.8 |
| .85 | ,, | .45 | ,, | 107.2 | 108.5 |
| .95 | ,, | .35 | ,, | 101.1 | 88.7 |
| 1.05 | ,, | .25 | ,, | 94.9 | 62.2 |
| .65 | .10 | .75 | 1.5 | 132.9 | 145.3 |
| .75 | ,, | .65 | ,, | 126.8 | 142.5 |
| .85 | ,, | .55 | ,, | 120.6 | 133.7 |
| .95 | ,, | .45 | ,, | 114.4 | 117.1 |
| 1.05 | ,, | .35 | ,, | 108.3 | 94.4 |

On the basis of either equation, a shift in consumption from spirits to beer is predicted to be associated with a fall in liver cirrhosis.

The equations also, of course, predict a rise in liver cirrhosis deaths with increase of consumption in any or all of the three beverages, and cannot account for the failure of Canadian liver cirrhosis rates to follow figures for total alcohol consumption (Figure 20), nor for other anomalies such as the fact that there does not exist a large difference in liver cirrhosis death rates between Ontario and Quebec, as the equations would predict from the consumption data. In terms of the present analysis such unexplained phenomena must be subsumed as "error" terms; the equations can be employed predictively only on the assumption that these terms do not alter.

*It will be apparent from Table 27 that liver cirrhosis death rates in Canadian provinces are consistently lower than in all those U.S. states showing equal or lower consumption of all three beverages. This anomaly cannot be reconciled by the regression analysis and, as a result, equations found by weighted least squares fitting must always over-predict Canadian liver cirrhosis death rates.

STEP 3 – TIME SERIES ANALYSIS

The statistical study to this point has been concerned with comparing differences among countries at one point in time – a cross sectional analysis based on 1967 data. It may also be instructive to examine the time series relationship of liver cirrhosis to beverage consumption within one country. The country chosen is the United Kingdom, since the beverage mix has changed sufficiently over the years (from 74 percent beer in 1920 to 86 percent during the war years – 1944 – and in 1968, back to 73 percent) to highlight differences. This change in beverage mix has been plotted against the liver cirrhosis death rate for the years 1914 to 1968 in Figure 18 (much of the data are drawn from Terris' work).

Figure 18 shows that within the period 1931 to 1958 (the years when beverage mix changed most) the liver cirrhosis death rate first declined to its lowest level, while beer consumption (and total consumption) was increasing. Then, as the liver cirrhosis death rate began to rise back to the present levels, beer consumption fell. During the same period, the pattern of consumption of wine and spirits was substantially opposite to beer consumption patterns. Terris (23) concluded that differences in cirrhosis mortality are associated with differences in the apparent consumption of alcohol from spirits and wine. No such association exists for beer.

If one examines the results over the total 54-year period from 1914 to 1968, there seems to be a relationship between cirrhosis death rates and total combined alcohol consumption. However, if one isolates the 1931 to 1958 period when beer consumption diverged from the path which other beverages took there seems to be a poor relationship – in fact an inverse association between beer consumption and the liver cirrhosis death rate. This illustrates the difficulty of drawing definitive conclusions from an examination of time series data alone.

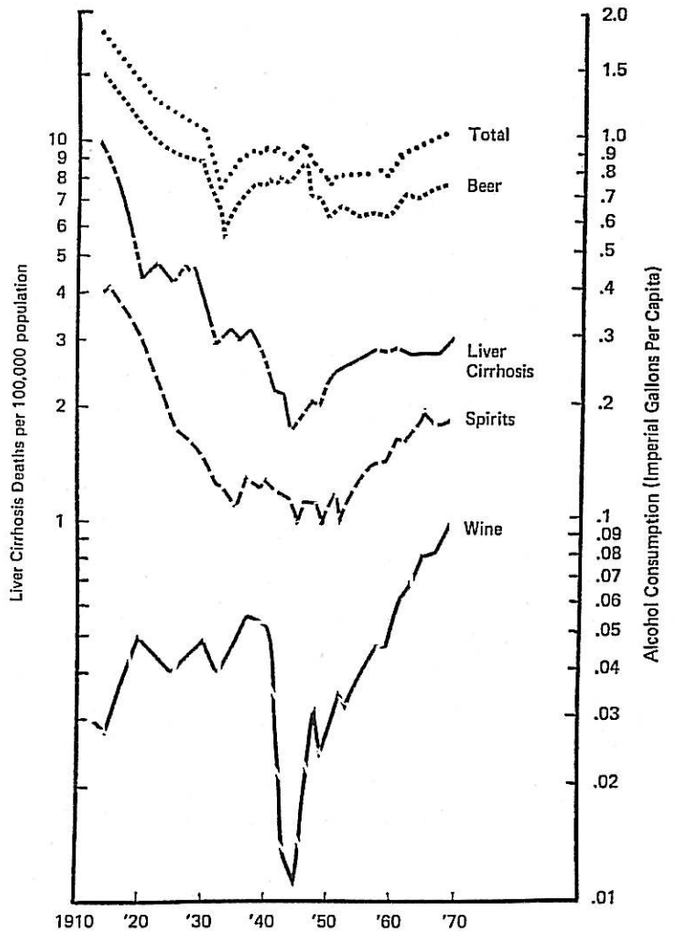
Carrying this review one step further, one might question whether this 1931-1958 lowering of the liver cirrhosis death rate was coincidental, either because all death rates were low during this period, or because the proportion of people in the 45-plus age group, who are most susceptible to the disease, decreased during this period. To test these possibilities, the crude death rates and the above proportion were compared to the liver cirrhosis death rates and the results were plotted in Figure 19. It may be seen from the plot that neither of the above reasons explain the decrease in the liver cirrhosis death rate. In fact the proportion of older people was increased during this period and the crude death rate was not lower.

This, on the whole, seems to bear out the Terris contention that the consumption of spirits and wine are more likely to be a cause of liver cirrhosis than is the consumption of beer. It is of interest that the equation from the Stage-7 analysis based on differences among countries predicts that English cirrhosis levels should correlate solely with spirits and wine consumption.

On the other hand when one examines the data from Paris over the period of the Second World War, the relationship between per capita consumption of absolute alcohol and liver cirrhosis death rate appears to be a much closer one. This is shown in Terris (23) where the liver cirrhosis death rate fell substantially when the availability of alcohol was curtailed.

FIGURE 18

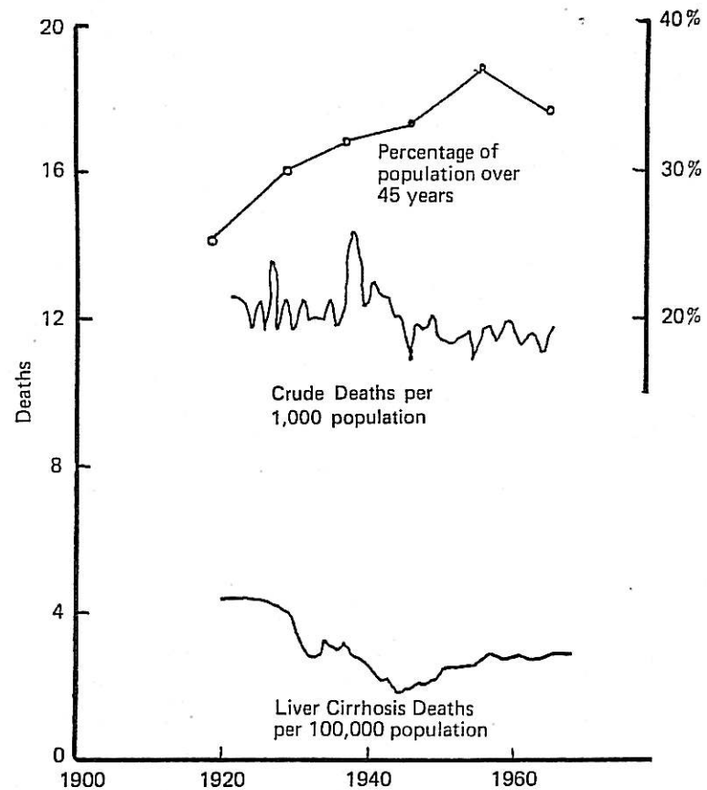
LIVER CIRRHOSIS DEATH RATES AND CONSUMPTION OF ABSOLUTE ALCOHOL IN BEER, WINE AND SPIRITS, UNITED KINGDOM 1914 – 1968




*Note*: The data from which the above was plotted were extracted from Terris, M., Amer. J. Public Health 57: 2076-2088, 1967.

FIGURE 19

DEATH RATES AND POPULATION TREND UNITED KINGDOM, 1925 – 1968



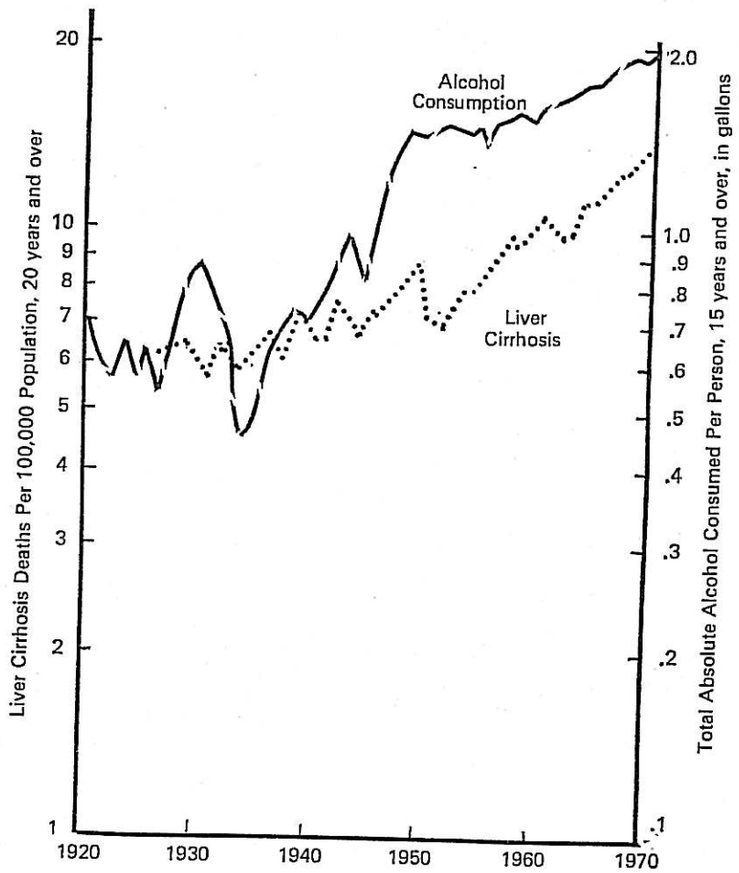

*Source*: Appendix B of Hickling-Johnston: *A Critical Evaluation of Epidemiological Studies Relating Alcoholic Beverages Consumption, by Type, with Various Alcohol Related Problems.* Unpublished report. Toronto: 1972.

Finally, Figure 20 contains Canadian data that illustrates a major counter-example to the relationship between average per capita consumption of alcohol and liver cirrhosis deaths. From 1926-1936 consumption of alcohol first increased by 69 percent and then fell by 47 percent; during the same period the rate of liver cirrhosis deaths showed very little variation.

In short, the data from these three countries suggest that an extremely mixed picture results from any attempt to explain liver cirrhosis death rates over time. It seems clear that factors other than alcohol consumption alone must be examined in order to give a more complete picture.

FIGURE 20

CANADA: COMPARISON OF TRENDS IN
LIVER CIRRHOSIS DEATH RATE AND ALCOHOL CONSUMPTION




*Source*: Alcoholism and Drug Addiction Research Foundation Statistics. Alcohol consumption: Dominion Bureau Statistics. *Control and Sale of Alcoholic Beverages.* Ottawa: Queen's Printer for selected years.



**Summary**

The quantitative studies indicate that different types of alcoholic beverages have different effects. This is true of both acute and chronic effects. Studies of arrests for drunkenness and drinking occasions indicate that the presence of wine and spirits as a factor is disproportionately high while beer is rarely implicated. This can be attributed to the lower intoxication effect of beer as well as to the fact that more alcohol is normally consumed on an occasion when higher strength beverages are being served. In other acute incidents, such as industrial and non-traffic accidents, alcohol is often a factor but little is known of its precise role.

In traffic accidents – an area of high concern to the general public – alcohol is estimated to be a factor in half of the Canadian fatalities. Studies in Canada, the United States and elsewhere indicate a rapidly rising risk of traffic accident as the blood-alcohol level rises. While little has been accomplished in isolating the type of alcoholic beverage most heavily implicated, the potential for harm would appear to bear a relationship to the impairment effect of the beverage and the amount consumed per drinking occasion. On the basis of the evidence on impairment effect included in this report, it seems probable that beer is less implicated in acute incidents such as traffic accidents. Studies to test this inference are necessary.

Examination of statistics concerning delirium tremens and liver cirrhosis – two diseases associated with chronic use of alcohol – indicates between-beverage differences. Investigators agree that distilled spirits are most usually implicated in cases of delirium tremens. In Denmark, a drastic reduction in spirits consumption during the First World War resulted in a reduction in delirium tremens incidence to about one-tenth of pre-war figures.

An examination of liver cirrhosis is important because statistics on cirrhosis mortality are available and often used as an indicator of the prevalence of alcoholism in a population. A cross-sectional analysis of alcohol consumption by beverage type and cirrhosis deaths in twenty-nine countries indicated that total consumption and liver cirrhosis deaths are generally related, but that there are differences between beverages and countries.

In analyzing international statistics to determine whether or not beverage mix in a population as well as total alcohol consumption is reflected in liver cirrhosis death rates, we have followed a number of previous investigators but with rather different results. Regression analysis has indicated that liver cirrhosis deaths rise about 50 percent more steeply with consumption of alcohol in the form of spirits than with alcohol consumed in wine or beer. A linear regression analysis does not account fully for the relatively low rates of liver cirrhosis found in countries (England, New Zealand, Australia) where more than 75 percent of alcohol consumption is in the form of beer; subsequent analysis (Stage-7 regression analysis) indicates that the rate of rise of liver cirrhosis deaths with consumption of any particular beverage is itself dependent upon how much is consumed by the population of each of the three alcoholic beverages. How this may arise is open to speculation. An alternative *a priori* possibility would be that alcoholics who drink beer are much less prone to liver damage than those who consume spirits. However, the results of Lelbach (12) indicate that this is not the case. One must conclude that the relation between liver cirrhosis deaths and alcohol consumption depends critically upon the drinking habits in a community, which are reflected in what, as well as how much, people

drink. The differences among beverages are supported by Terris' study of liver cirrhosis in England over time which showed that cirrhosis mortality there is related to levels of wine and spirits consumption but is not related to beer consumption.

Data from England, Paris and Canada suggest that an extremely mixed picture results from any attempt to explain liver cirrhosis death rates over long time periods. It seems clear that factors other than alcohol consumption alone must be examined to give a more complete picture.

Further research is needed on prospective and retrospective studies of selected groups of drinkers who drink different amounts of different beverages, especially in order to resolve the question as to whether the beverage differences which exist that have not yet been amenable to investigation, result in different risks of alcohol-related problems and in particular, alcoholism.

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