National mortality data for liver cirrhosis are presented and evaluated with respect to a number of parameters. From the evidence the conclusion is offered that mortality from cirrhosis is directly related to per capita consumption of alcohol from wine and spirits. Consequences for public action based on these findings are cited.

EPIDEMIOLOGY OF CIRRHOSIS OF THE LIVER: NATIONAL MORTALITY DATA

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IN 1964, cirrhosis of the liver was the eleventh leading cause of death in the United States, and the fifth leading cause for men in the productive years from 25 to 64.[1] The mortality rate for the disease had slowly but steadily risen from 7.7 per 100,000 population in 1934 to 12.1 in 1964, an increment of close to 60 per cent.[1,2] Yet epidemiologists have paid very little attention to this important and ever increasing cause of death.

The lack of interest is surprising, not only because of the significance of cirrhosis of the liver as a major cause of mortality, but because of the peculiar history of the disease. From 1900 to 1914, the death rate varied between 13 and 15 per 100,000 population. It dropped precipitously during World War I to a low of seven in 1920, remained at a plateau of seven for a period of 14 years through 1933, and then began the uphill climb which in three decades would bring it back to the 1914 level (Figure 1). It should be noted, incidentally, that the sharp drop from 11.3 in 1948 to 9.2 in 1949 is an artifact resulting from the adoption of the Sixth Revision of the International List of Diseases and Causes of Death in the latter year. When the 1949 rate was determined on the basis of the Fifth Revision, the rate was found to be 11.3, which is identical with the 1948 rate.[1] Jolliffe and Jellinek[3] pointed out in 1941 that the rapid decline in the cirrhosis death rate from 1916 to 1920 corresponded with the period of the most effective state and wartime prohibition, that the plateau from 1920 to 1933 coincided with the period in which the Eighteenth Amendment was in effect, and that the gradual increase from 1933 to 1940 occurred after national prohibition was repealed. They considered it safe to conclude that a significant association exists between changes in the death rate from cirrhosis of the liver and changes in the consumption of alcoholic beverages.

This view was challenged in 1950 by Lilienfeld and Korns[4] on a number of counts. The purpose of the present paper is to utilize additional data which have become available during the past 15 years to evaluate the conclusion reached by Jolliffe and Jellinek.

Comparative Long-Term Trends

One criticism made by Lilienfeld and Korns was that in England and Wales

the cirrhosis death rate remained at a rather constant level during the 1930's even though the use of alcohol as a beverage had been resumed following its wartime diversion to other uses. Figure 1 presents the long-term trends for the United States, England and Wales, Canada, and the city of Paris.[1,2,5-8] In England and Wales and in Paris, just as in the United States, there was a very sharp drop in the cirrhosis death rate coincidental with World War I. However, only in the United States did the rate remain stationary during the period of American prohibition from 1920 to 1933. The rate climbed rapidly to new peaks in Paris during this period. In England and Wales it remained more or less the same until 1929, when it began a long period of decline to a low of two per 100,000 in 1942 through 1950; since then there has been a perceptible rise. In Canada, the rate rose from 1921 to 1933 and has continued to rise ever since.

Even more instructive are the subsequent data for Paris. In 1942 there began an unprecedentedly sharp fall in the cirrhosis death rate, from 35 in 1941 to a low of six in 1945 and 1946; it began to rise sharply again in 1948 and was back to normal by 1952. This was not merely a wartime phenomenon; the war was over in 1945. What happened was that from 1942 to 1948 wine was limited by rationing to one-half to one liter per week.[7]

These data appear to obviate a number of criticisms of the Jolliffe-Jellinek thesis. One is that deaths from a chronic disease would not be expected to decrease

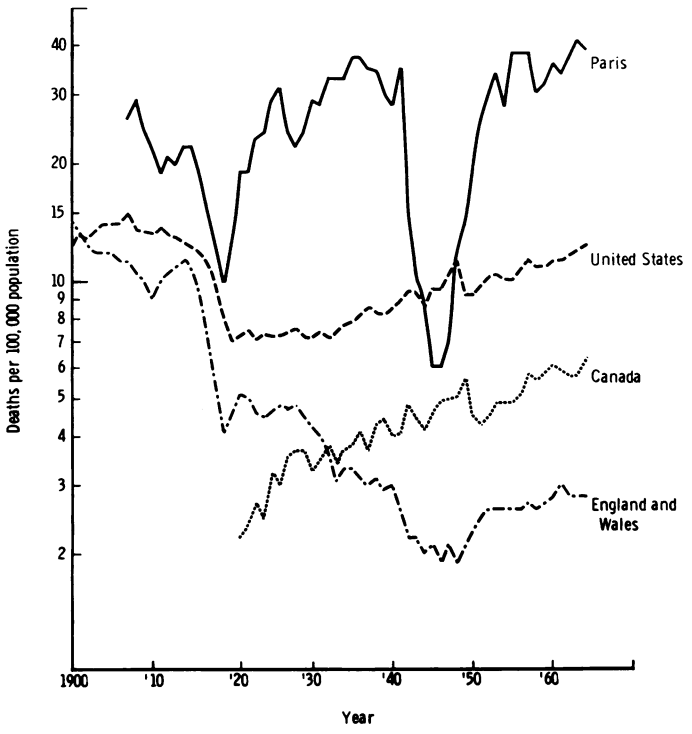

Figure 1—Death rates, cirrhosis of the liver, United States, England and Wales, Canada, and Paris, 1900-1964

Table 1—Death rates by age, cirrhosis of the liver, United States, 1900-1960

Age (yrs)	Deaths per 100,000 population						
	1900	1910	1920	1930	1940	1950	1960
Under 1	4.3	0.8	1.0	0.5	1.7	1.2	0.8
1–4	0.3	0.5	0.3	0.2	0.2	0.1	0.2
5–14	0.6	0.4	0.2	0.2	0.2	0.1	0.1
15–24	1.3	0.9	0.5	0.4	0.3	0.3	0.3
25–34	8.8	5.3	1.3	1.5	1.8	2.1	2.9
35–44	27.0	18.1	4.5	6.1	7.2	9.3	11.8
45–54	48.7	38.3	12.9	14.0	16.5	19.1	27.6
55–64	75.2	63.2	28.1	26.8	28.0	27.3	32.7
65–74	97.4	87.4	53.2	43.0	38.9	33.5	37.4
75–84	91.7	96.8	78.1	57.5	52.1	36.9	32.1
85 and over	61.9	84.4	76.3	64.4	58.3	39.0	28.8

so sharply within a brief space of years when the incriminated etiologic agent was temporarily removed. The fact is, however, that in every instance the cirrhosis death rate did fall rapidly when the availability of alcohol was sharply diminished; this occurred during World War I in the United States, England and Wales, and Paris; it occurred again in Paris in 1942. Furthermore, the Paris rate rose sharply after 1948; apparently the cirrhosis death rate responds fairly rapidly to changes in availability of alcoholic beverages. This phenomenon is consistent with the clinical course of the disease. In many cases the cirrhotic process can be halted and decompensation prevented by avoiding further use of alcohol. Conversely, resumption of heavy alcohol use after a period of abstinence can decompensate a previously injured liver in a relatively short period of time.

Another criticism was that the low cirrhosis death rate in the United States during prohibition may have been due to reluctance on the part of physicians to certify cirrhosis as a cause of death, because such reports might incriminate the patient with illegal alcohol consumption. This explanation, however, cannot account for the decline in cirrhosis mortality during World War I in England and Wales, where alcohol consumption was reduced by measures other than prohibition.

Age, Sex, and Race

Table 1 indicates that the death rate from cirrhosis of the liver is somewhat higher in infancy than in childhood but does not reach significant levels until adult life. The rate in adults rises continuously with age up to 75 years. The rise in the crude death rate in the United

Table 2—Death rates by age and sex, cirrhosis of the liver, United States, 1960

Age (yrs)	Deaths per 100,000 population	
	Male	Female
Under 1	0.8	0.9
1–4	0.2	0.1
5–14	0.1	0.2
15–24	0.2	0.3
25–34	3.1	2.7
35–44	14.4	9.3
45–54	36.8	18.6
55–64	48.5	17.9
65–74	57.4	20.1
75–84	45.1	22.0
85 and over	40.0	21.7
Total	15.3	7.5

States between 1930 and 1960 was due entirely to increases in the specific rates for ages 25 to 64; the rates for the groups aged 65 and over declined during this period.

The crude cirrhosis death rate in males is approximately twice that in females. Before adult life, on the other hand, there is no sex difference in mortality (Table 2). These facts are consistent with the hypothesis that alcohol consumption is an important factor in liver cirrhosis in adults. They are consistent also with the fact that cirrhosis of the liver in infants and children is due to a variety of other causes such as hepatitis, mucoviscidosis, Wilson's disease, and galactosemia.

The crude death rates by sex and color for the period 1910 to 1964 are shown in Figure 2. It is evident that the end of prohibition in 1933 did not result in the same rise in the cirrhosis death rate in nonwhites that occurred in whites. The rise did not begin to occur in nonwhites until about 1948. (The sharp drop in rates for all four sex-color groups from 1948 to 1949 is an artifact caused by the change from the Fifth to the Sixth Revision of the International List of Diseases and Causes of Death.) Lilienfeld and Korns had available only the death rates through 1947; these data indicated that while the rates for whites had been increasing since 1933, the rates for nonwhites had declined after 1930.

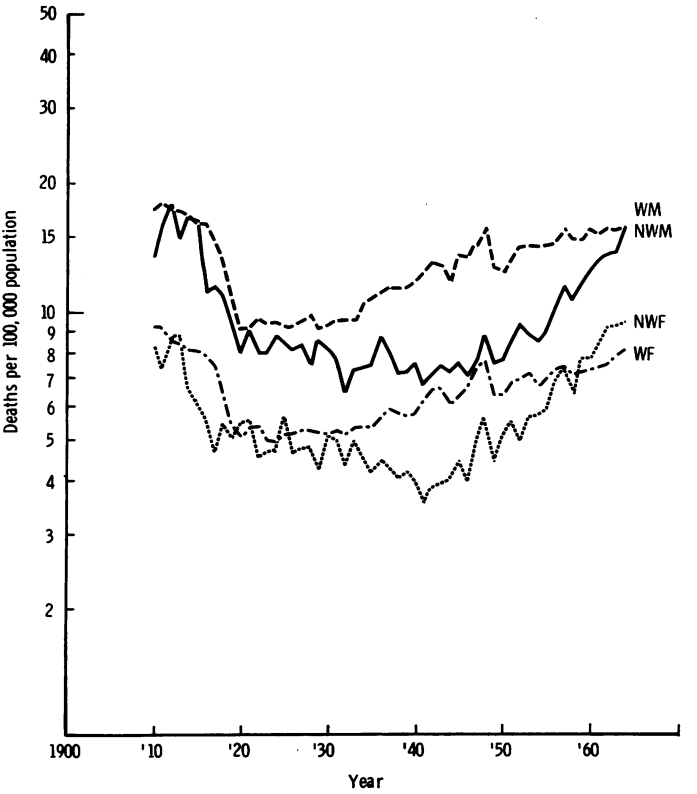

Figure 2—Death rates, cirrhosis of the liver, by sex and color, United States, 1910-1964

Table 3—Age-adjusted death rates by sex and color, cirrhosis of the liver, United States (adjusted to total US 1940 population as the standard), 1915-1964

Year	Deaths per 100,000 population			
	White male	White female	Nonwhite male	Nonwhite female
1915	21.5	10.7	27.9	16.4
1920	11.2	6.3	10.9	8.0
1925	10.9	6.2	11.4	8.9
1930	10.4	5.8	11.5	7.7
1935	11.3	5.7	9.9	5.7
1940	11.7	5.6	9.2	5.0
1945	11.9	5.9	8.5	5.4
1950	11.6	5.8	8.9	5.9
1955	13.0	6.1	10.6	6.8
1960	14.4	6.6	14.9	9.1
1964	15.0	7.3	19.5	11.3

They doubted therefore that the trend of cirrhosis death rates could be accounted for by changes in alcohol consumption, since prohibition and repeal should have had similar effects on consumption by both whites and nonwhites.

Now that we have the data for the subsequent years, it becomes clear that what occurred up to 1948 was a prolonged lag in the rates in nonwhites, and that their rates have since climbed more sharply than the rates in whites. In fact, while nonwhites have not been able to achieve equality with whites in most areas of American life, they have apparently succeeded in this one. They have succeeded all too well; in 1964 the age-adjusted cirrhosis death rate for nonwhite males was 30 per cent higher than for white males, while for nonwhite females it was 55 per cent higher than for white females (Table 3).

Why was there a lag in the nonwhite rates from 1930 to 1948, and why have they risen so sharply since? In the absence of data on alcohol consumption of whites and nonwhites, we can only speculate as to the reasons. One possible explanation is that nonwhites were hit harder by the depression and were therefore less able to obtain alcoholic beverages. Another factor which may help explain the sharp rise since 1948 is the tremendously accelerated urbanization of the nonwhite population in the forties and fifties.[9]

Urban-Rural Differences

In 1960, mortality from cirrhosis of the liver in urban areas in the United States was more than twice the rate in rural areas. Mortality in metropolitan counties was double the rate in nonmetropolitan counties. The urban-rural

Table 4—Mortality rates, cirrhosis of the liver, by size of community, United States, 1960

	Deaths per 100,000 population		
	Total	Urban	Rural
United States	11.3	14.6	6.2
Metropolitan counties*	13.8	16.0	7.0
Nonmetropolitan counties	7.0	9.4	5.6

* Counties containing at least one city of 50,000+ population, and contiguous counties which are socially and economically integrated with the central cities.

Table 5—Standardized mortality ratios, cirrhosis of the liver, men age 20 to 64 years, by occupation level, United States, 1950

Occupation level	Standardized mortality ratio		
	Total	White	Nonwhite
I. Professional workers	90	—	—
II. Technical, administrative, and managerial workers, except farm	88	88	—
III. Clerical, sales, and skilled workers	105*	107*	54
IV. Semiskilled workers	118*	122*	85
V. Laborers, except farm	148	158*	121
Agricultural workers	51	51	54

* Proportionate mortality ratio significantly higher than 100.

difference was found in both types of counties, but was smaller in the nonmetropolitan counties (Table 4).

Social Class

Lilienfeld and Korns divided the city of Buffalo into four economic quarters and found that the cirrhosis death rate in males increased with decreasing economic status. Similarly, Pearl, Buechley and Lipscomb[10] reported that in three California cities—Los Angeles, San Francisco, and Oakland—there was an inverse relationship of both male and female cirrhosis death rates with occupational level of census tracts.

National data on mortality by social class are presented in Table 5. The standardized mortality ratio (SMR) compares the tabulated number of deaths in an occupation group from a specific cause with the number to be expected if the age-specific death rates from that cause for the total male population of working age had prevailed in that occupation group.

A standardized mortality ratio significantly above 100 for a particular cause of death may occur as a result of overstatement of the "usual" occupation reported on death records in comparison with the "current" occupation reported in the census enumeration. The effects of errors of this type can be eliminated by the use of the proportionate mortality ratio (PMR). This ratio compares the tabulated number of deaths from a specific cause in an occupation group with the number to be expected if, for each age group, the deaths from that cause bore the same proportion to all deaths in this occupation group that prevailed in the total male population of working age. Since the proportionate mortality ratio for a specific cause involves no use of census data, the finding of a proportionate mortality ratio which is signifi-

Table 6—Standardized mortality ratios, cirrhosis of the liver, men age 20 to 64 years, by social class, England and Wales, 1949-1953

Social class	Standardized mortality ratio
I. Professional occupations	207
II. Intermediate occupations	152
III. Skilled occupations	84
IV. Partly skilled occupations	70
V. Unskilled occupations	96

Table 7—Admission rates per million males age 20 and over to mental hospitals, by social class and diagnostic group, England and Wales, 1953

Social class	Alcoholism + alcoholic psychosis	Schizophrenia	Manic-depressive reaction	Psychoneuroses, all forms	Behavior, character and intelligence disorders, all forms
I	152	164	430	164	217
II	65	170	369	201	85
III	26	332	392	294	91
IV	18	365	442	260	89
V	45	872	707	407	253

cantly higher than 100 strengthens the probability that a high standardized mortality ratio reflects an increased mortality risk from that cause.[11]

The data for the United States presented in Table 5 confirm the findings of the Buffalo and California studies; mortality from cirrhosis of the liver is inversely related to occupation level.

In England and Wales, on the other hand, the situation is entirely different. There mortality from cirrhosis of the liver is greatest in the upper classes (Table 6).[12] This class difference in England and Wales also holds true for admissions to mental hospitals for alcoholism and alcoholic psychosis, and differs from the social class distribution of other types of mental disease (Table 7).[13]

Occupation

Lilienfeld and Korns were led by their findings on urban-rural and social class differences to consider that occupation per se may be an important factor in the epidemiology of cirrhosis of the liver, and to suggest the possible etiologic role of industrial toxic substances. However, the data on cirrhosis mortality by social class in England and Wales cast doubt on this line of reasoning. If industrial toxins play a significant etiologic role in cirrhosis of the liver, one would expect industrial workers to have higher mortality rates than the professional classes in England and Wales as well as in the United States.

Additional data that have become available since the publication of Lilienfeld and Korns's paper make it possible to evaluate their hypothesis more fully. Table 8 indicates that the highest mortality from cirrhosis of the liver occurs

Table 8—Standardized mortality ratios, cirrhosis of the liver, men age 20 to 64 years, by major occupation group, United States, 1950

Major occupation group	Standardized mortality ratio
Professional, technical, and kindred workers	90
Managers, officials, and proprietors, except farm	111*
Clerical and kindred workers	99*
Sales workers	108
Craftsmen, foremen, and kindred workers	93
Operatives and kindred workers	96
Service workers, except private household	184*
Laborers, except farm and mine	154

* Proportionate mortality ratio significantly higher than 100.

Table 9—Occupation groups with high standardized mortality ratios, and proportionate mortality ratios significantly higher than 100, cirrhosis of the liver, men age 20 to 64 years, United States, 1950

Occupation group	Standardized mortality ratio
Waiters, bartenders, and counter workers	392
Longshoremen and stevedores	342
Cooks, except private household	286
Meat cutters, except slaughter and packing house	258
Bakers	219
Other service workers, except private household	209
Laundry and dry cleaning operatives	206
Barbers, beauticians, manicurists	182
Managers, officials, and proprietors (n.e.c.)* wholesale and retail trade	145
Operatives and kindred workers (n.e.c.)* food and kindred products	133
Other specified operatives and kindred workers	129
Salesmen and sales clerks (n.e.c.)* retail trade	127

* Not elsewhere classified.

in service workers rather than in industrial workers. Laborers have a high SMR but not a significantly high PMR, while operatives and kindred workers show no excess of mortality. Furthermore, as Table 9 indicates, the highest mortality from cirrhosis occurs in waiters, bartenders, and counter workers, that is, workers with maximum exposure to alcoholic beverages, while practically all the occupation groups with high mortality ratios are in occupations which do not involve exposure to industrial toxins.[14]

Similarly, Table 10 demonstrates that for the major industry groups, the highest mortality from cirrhosis of the liver occurs in entertainment and recreation services, personal services, and wholesale and retail trade, while there is no excess of mortality in manufacturing. Furthermore, as indicated in Table 11, practically all the industries with high mortality ratios are unlikely sources of industrial toxins. It is significant that the three industries with the highest cirrhosis mortality are eating and drinking places, hotels and lodging places, and entertainment and recreation services.[15]

Alcohol Consumption

Table 12 shows that the differences in cirrhosis mortality in the United States, Canada, and the United Kingdom are associated with differences in the apparent consumption of alcohol from spirits

Table 10—Standardized mortality ratios, cirrhosis of the liver, men age 20 to 64 years, by major industry group, United States, 1950

Major industry group	Standardized mortality ratio
Agriculture, forestry, and fisheries	53
Mining	87
Construction	106
Manufacturing	87
Transportation, communication, and other public utilities	130
Wholesale and retail trade	143*
Finance, insurance, and real estate	96
Business and repair services	125
Personal services	152*
Entertainment and recreation services	191*
Professional and related services	87
Public administration	109

* Proportionate mortality ratio significantly higher than 100.

Table 11—Industries with high standardized mortality ratios, and proportionate mortality ratios significantly higher than 100, cirrhosis of the liver, men age 20 to 64 years, United States, 1950

Industry	Standardized mortality ratio
Eating and drinking places	368
Hotels and lodging places	210
Entertainment and recreation services	191
All other personal services	186
Food and dairy products stores, and milk retailing	159
Legal, engineering, and miscellaneous professional services	154
State and local public administration	148
Printing, publishing, and allied industries	141
All other retail trade	139
Other food industries	122

and wine. No such association exists for beer; the apparent consumption of alcohol from beer is similar in the three countries.[1,5,6,9,16-23]

The trends of cirrhosis mortality[1,2] and of alcohol consumption from spirits and wine in the United States[9,16] are given in Figure 3. Both are relatively stable from 1900 to World War I, when they drop sharply. Furthermore, the rise in the cirrhosis death rate since 1933 is associated with an increase in alcohol consumption from spirits and wine.

Unlike the United States, cirrhosis mortality in Canada has risen constantly since 1922.[6] Figure 4 indicates that this rise is associated with a long-term increase in alcohol consumption from spirits and wine.[17,18] It should be noted that the decline in apparent consumption in the early 1930's was not associated with a change in the cirrhosis mortality rate. However, at least part of this decline may be an artifact, since there is reason to believe that a good portion of the apparent consumption in the late 1920's was diverted to the United States.

The data for the United Kingdom[5,19-23] are presented in Figure 5. Both the cirrhosis mortality rate and the alcohol consumption from spirits and wine show some decrease from 1900 to World War I, a sharp drop during that war, then a long-term decline in the period between the two wars, a rapid fall early in World War II, and a continuing postwar rise.

The trends for these three countries indicate that the cirrhosis mortality rate is associated with per capita alcohol consumption from spirits and wine. Furthermore, the usual criticisms of such temporal associations are hardly applicable, since it is difficult to conceive of any other factor which would follow the same trends. Diagnostic practices, the

Table 12—Death rates, cirrhosis of the liver, and apparent consumption of absolute alcohol by type of beverage, United States, Canada, and United Kingdom, 1960

	Cirrhosis of liver (deaths per 100,000)	Absolute alcohol consumption (US gal per capita)			
		Spirits	Wine	Beer	Total
United States	11.3	0.60	0.15	0.77	1.52
Canada	6.1	0.44	0.12	0.84	1.40
United Kingdom	2.9	0.20	0.08	0.78	1.06

incidence of infectious or serum hepatitis, and exposure to industrial toxins or other possible factors could not be expected to show all these peculiar patterns of change: the sharp drop in World War I, the plateau from 1920 to 1933, and the constant rise since then in the United States; the continuing increase in Canada from 1921 to 1964; and, in the United Kingdom, the rapid fall in World War I and early in World War II, the long-term decline between the two wars, and the subsequent postwar rise.

These data provide, therefore, direct confirmation of Jolliffe and Jellinek's hypothesis that a significant association exists between changes in the death rate from cirrhosis of the liver and changes in the consumption of alcoholic beverages.

American versus British Experience

In 1914, the cirrhosis mortality rate was 12.5 in the United States; it fell to seven in 1920 and remained at this rate through 1933, when it began a continuous rise to 12.1 in 1964. If the conclusion is correct that the cirrhosis mortality rate is directly related to alcohol consumption from spirits and wine, then we may surmise that prohibition was successful in maintaining per capita consumption of spirits and wine at the low level reached in 1920. With repeal came a gradual increase in consumption, with the result that the cirrhosis mortality rate in 1964 is almost identical with the high rate of 1914.

The British experience was quite different. In 1914, the cirrhosis mortality rate in the United Kingdom was ten. It fell to five in 1920, then gradually declined to a low of two in the 1940's, and has risen since to a rate of three in 1963. Thus, while the United States came full circle, ending up in the same place where it began 50 years ago, the British succeeded in lowering their cirrhosis mortality rate by 70 per cent during the same

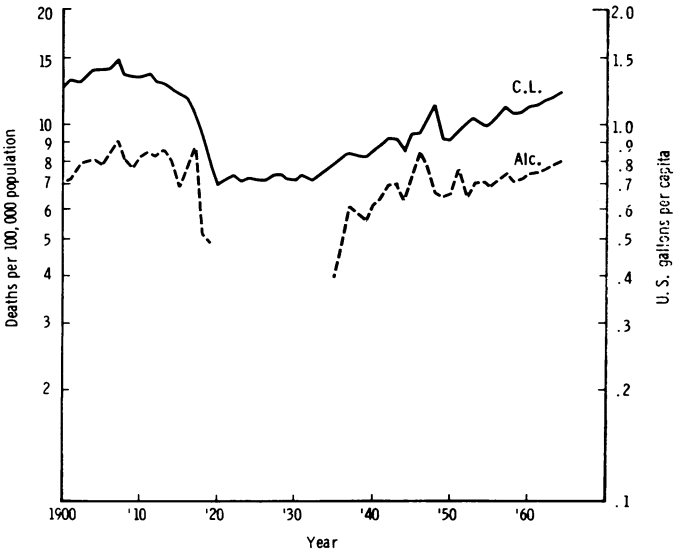

Figure 3—Death rates, cirrhosis of the liver, and apparent per capita consumption of absolute alcohol from spirits and wine, United States, 1900-1964

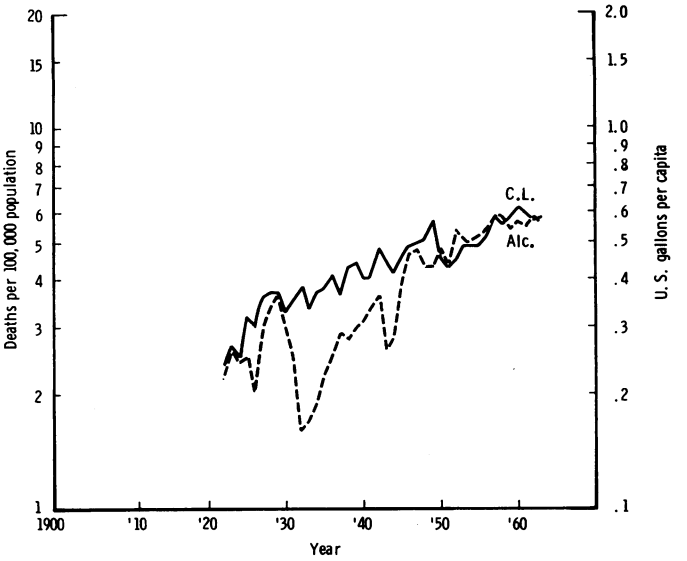

Figure 4—Death rates, cirrhosis of the liver, and apparent per capita consumption of absolute alcohol from spirits and wine, Canada, 1922-1963

period. This remarkable achievement occurred despite the fact that there was no prohibition in the United Kingdom. How did it happen?

The answer is to be found in the history of British social policy on alcoholic beverages in the period during and after World War I. Wartime measures included a sharp curtailment in the amount of alcohol available for consumption, drastic restriction of the hours of sale, and marked increases in taxes on alcoholic beverages. With the end of the war, the limitations on the available quantity of alcohol were removed, but the hours of sale were extended to only half the prewar time of opening, while taxation on alcoholic beverages was increased even further.[24] As Wilson[25] pointed out in 1940, "The high duty on spirits since 1918 has had a marked effect on consumption, as was inevitable when the selling price was raised from 31*s.* 6*d.* per proof gallon to its present price of 130*s.* a gallon. The consumption per head has fallen in England from 0.59 gallons in 1913 to 0.205 in 1936, and in Scotland from 1.42 gallons in 1913 to 0.36 in 1936."

This also explains the peculiar fact that mortality from cirrhosis of the liver is greatest in the lowest social class in the United States and in the highest social class in England and Wales (Tables 5, 6). Spirits have been taxed out of the reach of the lower social classes in the United Kingdom, where only the well-to-do can really afford the luxury of dying from cirrhosis of the liver.

Prevention

Although cirrhosis of the liver can result from hepatitis and other causes, the evidence presented in this paper appears to leave little doubt that cirrhosis mortality rates are directly related to per capita consumption of alcohol from spirits and wine. Prevention, therefore, requires measures to lower such con-

sumption. The methods used in the United Kingdom which have succeeded in reducing mortality from cirrhosis of the liver are, first, progressive increases in taxation of alcoholic beverages in order to decrease their availability to increasing proportions of the population and, second, restriction of the hours of sale of alcoholic beverages.

The relationship of price to consumption of alcohol cannot be considered a peculiarly British phenomenon. Seeley[26] has shown that during the period 1935-1956, in both the province of Ontario and in Canada as a whole, there was an inverse relationship between the relative price of beverage alcohol on the one hand, and alcohol consumption and cirrhosis mortality on the other.

Programs for control of cirrhosis of the liver which are limited to health education and treatment of the alcoholic are not enough. The British example demonstrates that governmental fiscal and regulatory measures can be effective in reducing alcohol consumption and lowering mortality from cirrhosis of the liver. Recognition of the significance of their experience is an urgent matter, for cirrhosis of the liver is now the eleventh leading cause of death in the United States, and the death rate rises with every passing year.

Summary

This paper evaluates national mortality data for cirrhosis of the liver with regard to a number of parameters: long-term trends, age, sex and race, urban-rural differences, social class and occupation, and alcohol consumption. The evidence strongly supports the conclusion that cirrhosis mortality is directly related to per capita consumption of alcohol from spirits and wine.

The experience of the United Kingdom is cited to indicate that governmen-

Figure 5—Death rates, cirrhosis of the liver, and apparent per capita consumption of absolute alcohol from spirits and wine, United Kingdom, 1900-1963

tal fiscal and regulatory measures to reduce per capita alcohol consumption can markedly lower mortality from cirrhosis of the liver.

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