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The Post-Repeal Eclipse in Knowledge About The Harmful Effects of Alcohol

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Abstract

National Prohibition in the USA (1919 to 1933) was followed by an era in which medical scientists played an important role in minimizing the harmful effects of alcohol. Cirrhosis, cardiomyopathy, adverse fetal effects, and esophageal cancer are examples of alcohol-related health problems that were well known at the beginning of the 20th century but were dismissed during the late 1930's and early 1940's, only to be rediscovered during the 1960's and afterwards. This eclipse in knowledge occurred because of skepticism about earlier claims that had been made in the name of scientific temperance and, most importantly, because of changing standards for medical evidence. The paradigm for disease causation that gave birth to modern medicine was based on microbiology and reinforced by hormone and nutrition discoveries. Most alcohol-related health problems are poorly explained by this paradigm. The more recent epidemiologic paradigm for noninfectious disease is more applicable to the health risks associated with heavy drinking. A transformation of knowledge about alcohol's relationship to disease has occurred.

Introduction

National Prohibition in the USA (1919–1933) was followed by an era in which medical scientists played an important and generally overlooked role in freeing alcohol from its reputation as a harmful substance. During the late 1930's and early 1940's, alcohol-related diseases that were well known at the beginning of the century, and are well documented today, were discounted in authoritative journals and books.

In a 1942 book for the lay public that was prepared under the auspices of the American Association for the Advancement of Science, Yale researchers Howard Haggard and E.M. Jellinek told their readers, “The diseases of chronic alcoholism are essentially nutritional disturbances.” They went on to explain that the cause of cirrhosis was unknown, that the enlarged and failing heart that was formerly known as “beer drinker's heart” (and is presently known as alcoholic cardiomyopathy) was due to excessive fluid intake, that temperance claims about the adverse effects of alcohol on offspring were baseless, and that cancers of the upper digestive tract were not associated with heavy alcohol consumption (Haggard & Jellinek, 1942, pp 177–214).

Haggard and Jellinek were by no means alone in minimizing the harmful effects of alcohol (Harding, 1939; Leary, 1938; Moore, 1941). Even Haven Emerson, who documented the public health benefits of Prohibition during the 1920's (Emerson, 1928) and who, as President of the American Public Health Association shortly after Repeal, urged greater

attention to alcohol-related problems (Emerson, 1934), was extremely even-handed and perhaps, by late-20th-century standards, overly skeptical in his judgement of alcohol's harmful effects in his role as editor of the highly-regarded *Alcohol and Man* (Emerson, 1932). If Emerson, a 'dry', who *Fortune* magazine (Anon., 1935) characterized as "a fanatical teetotaler," was easy on alcohol, 'wetter' writers were more generous in their assessment of alcohol's harmful effects. And, given the 'wetter' nature of the age, with alcohol "becoming identified with progressive, urban, middle-class lifestyles" (Room, 1984, p 298), 'wetter' treatments of alcohol were becoming more frequent.

Post-Repeal scientists were eager to distance themselves from temperance ideology and from the 19th-century and early 20th-century culture in which it had flourished (Room, 1984). Moreover, many of them no doubt felt threatened by the temperance movement, which was by no means dead after Repeal. For example, in 1938, bowing to the political weight of the temperance movement, the Virginia legislature felt compelled to burn copies of a scientific report on alcohol that it had commissioned from professors representing the state's two medical schools; the report had condoned moderate drinking (Roizen, 1991a). Because of the still-unresolved 'wet-dry' debate and because of the harsh economic realities of the Depression, several attempts at organizing funding for the study of alcohol problems met with failure during the 1930's (Roizen, 1991b). It was not until 1938 that the Research Council on Problems of Alcohol, an associated organization of the American Association for the Advancement of Science, finally became established as a scientific body that could credibly be accepted as being above the 'wet-dry' debate (Roizen, 1991a). The Research Council's Scientific Committee included some of the elite of American scientific medicine, towering figures like Hans T. Clarke, Philip Bard, Walter B. Cannon, Alan M. Chesney, Edward G. Conklin, and E.K. Marshall (Anon., 1938a; Harvey, 1981).

The Council's first major project, which began in 1939, was a thorough review of the literature on alcohol's effects on man, funded by a grant of \$25,000 from the Carnegie Corporation. The principal investigator was Norman Jolliffe, who was a member of the faculty of the New York University College of Medicine and who was conducting seminal research on the vitamin deficiencies that plagued many of the 7,000 to 9,000 alcoholics who were admitted to Bellevue Hospital each year. Jolliffe, who worked part time on the project, hired a biometrician named E.M. Jellinek to be the full-time scientific secretary for the project; this was Jellinek's entry into the alcohol field. The staff, which included multi-lingual physician readers, abstracted the tremendous amount of literature that had already been published during the century, typing the abstracts onto punch cards that were organized into the forerunner of today's computerized information databases. In 1940, Howard Haggard moved the project to his Applied Physiology Laboratory at Yale and obtained additional funding for a new publication, the *Quarterly Journal of Studies on Alcohol*.

Despite their impressive resources, Haggard and Jellinek were wrong in their pronouncements about the health consequences of heavy drinking. The purpose of this paper is to explain this post-Repeal "eclipse" in knowledge about alcohol's harmful effects. My central argument is that the context of medical knowledge during the post-Repeal era played an important role in determining how scientists evaluated various longstanding claims about alcohol's harmful effects. The post-Repeal understanding of disease causation came together

with the reaction against temperance ideology to produce a new minimization of alcohol's harmful effects and a greater emphasis on alcoholism as a disease.

The scientific paradigm (Kuhn, 1970) for disease causation was narrower for post-Repeal scientists than it is for scientists living near the end of the 20th century; it was based on disease as a reaction to foreign bacteria or deficiency of some vital hormone or nutrient. Working within this paradigm during the first four decades of the 20th century, medical scientists produced seemingly miraculous treatments--sulfas, vitamins, adrenal hormones, and insulin. These medical scientists were skeptical of statistical data that could not be readily verified with laboratory evidence. They had not yet developed the epidemiological techniques that would eventually link smoking with lung cancer or high-fat diets with heart disease; they had not yet witnessed the emergence of a new paradigm for chronic diseases, based on epidemiology, that would link them with environmental causes (Susser, 1985). Also, discovery of the vitamins that were often lacking in the diets of chronic alcoholics occurred very close to Repeal, giving medical scientists a potent and inexpensive means for curing many alcohol-related diseases and thereby reinforcing their older, narrower paradigm for disease.

The epidemiologic paradigm for noninfectious disease that emerged in the 1950's, with its attendant concern for the identification and prevention of risk factors, was applied to smoking before it was reluctantly accepted for heavy drinking. The post-Repeal minimization of alcohol's harmful effects continued until the 1960's. In a book published toward the end of the era which is the focus of this paper, Morris Chafetz, who subsequently became the first director of the National Institute on Alcohol Abuse and Alcoholism, told his readers: "*there is no sound evidence whatsoever that alcohol causes permanent direct damage to the body*" (Chafetz, 1965, p 48, emphasis in the original text).

I shall elaborate on this theme of changing understanding of disease causation later, after presenting a series of four historical case examples of alcohol-related health problems: cirrhosis, cardiomyopathy, fetal alcohol syndrome, and esophageal cancer. These examples were selected because they are currently fairly well established and because they were specifically discounted by Haggard and Jellinek, who had intimate knowledge of the outcome of the Carnegie-funded review of the literature on alcohol.

Four Historical Examples

Cirrhosis of the Liver

Cirrhosis is moving toward the bottom of the top ten causes of death in the United States, but, along with alcohol-related car crashes, suicides, homicides, and esophageal cancer, it remains a major cause of alcohol-related mortality (MMWR, 1990a, 1990b, & 1990c). For example, in California during 1988, cirrhosis was the leading cause of alcohol-related deaths among those between the ages of 45 and 74 (Sutocky, 1990). Although alcohol-related liver disease can rightly be thought of as a significant public health problem and was identified at the beginning of the century as one of the ways in which "excessive and continued use of alcoholic drinks tends to produce disease and shorten life" (Billings, 1905), at mid-century these characterizations would have been considered too close to temperance ideology to

receive serious consideration by mainstream medical scientists. Thus, it is a good place to begin our series of historical case examples of alcohol-related health problems.

At the beginning of the century, in his pioneering textbook of medicine, William Osler proclaimed, “alcohol is the chief cause of cirrhosis of the liver” (Osler, 1909). Today, near the end of the century, Osler’s statement still stands. As a recent edition (the 22nd) of Osler’s text, now a multi-authored book with five editors, put it,

Cirrhosis related to alcoholism is the most common form of chronic liver disease found in the United States, and its incidence has roughly paralleled the per capita consumption of alcohol. During the Prohibition era, deaths from cirrhosis decreased and after the repeal of the law began to rise again. The majority (80 to 90 percent) of patients with alcoholic cirrhosis give a history of years of heavy alcohol use

(Mezey, 1988).

At mid-century, however, alcohol was thought to have a secondary role in the etiology of cirrhosis. Although cirrhosis was frequently associated with long-time heavy drinking, it was the diets of these heavy drinkers that were suspect (Patek, 1955). In 1967, a prominent hepatologist noted, “A few years ago protein deficiency in the chronic alcoholic would be given the prime role in the etiology of Laennec’s cirrhosis, but more recent concepts have come full cycle back to the view of Osler that alcohol is directly toxic” (Watson, 1967).

The idea that diet, not alcohol, might be the major cause of cirrhosis of the liver was popularized by a major review of the literature that was sponsored by the Research Council of Problems of Alcohol and funded by the Carnegie Corporation. After reviewing several dozens of autopsy studies, as well as unpublished data from Bellevue Hospital, Jolliffe and Jellinek (1941) observed that chronic inebriates were seven times more likely to die from cirrhosis of the liver than those who were temperate or abstinent. However, the majority of chronic inebriates did *not* develop cirrhosis; it apparently-occurred in only 8 per cent. They also reviewed vital statistics comparing cirrhosis death rates with per capita alcohol consumption rates, observing a close correlation between changes in the death rate from cirrhosis of the liver and changes in the consumption of alcoholic beverages. However, after reviewing the experimental studies, they concluded (prematurely) that alcoholic liver cirrhosis could not be produced experimentally in laboratory animals. As a result, they felt that the evidence established an association between excessive drinking and cirrhosis but that alcohol was not the direct cause. Given these constraints, the last section of their paper focused on just what the cause of cirrhosis might be. It was an extensive review of “recent etiological views on cirrhosis of the liver (1930–1941).” As might have been expected at a time when impressive discoveries in nutrition were being made--many of them in alcoholic populations--nutritional theories were quite prominent. Though no single etiological theory was deemed satisfactory, the authors’ bias is evident from the title they chose for their paper and subsequent book chapter: “Vitamin Deficiencies and Liver Cirrhosis in Alcoholism” (Jolliffe & Jellinek, 1941; Jolliffe & Jellinek, 1942).

From this point, the mid-century story of scientific inquiry relative to cirrhosis unfolds along three independent lines, each of them serving to diminish medical scientists’ long-standing

tendency to blame alcohol as the main cause of cirrhosis. The first, which will not be described in detail here, was an extension of Jolliffe and Jellinek's quest for an explanation for why some excessive drinkers developed cirrhosis. With mid-century advances in biochemistry, the role of nutrition in the etiology of cirrhosis continued to serve as a major area of focus. Some workers sought to abandon, "as unscientific, reference to any type of *liver cirrhosis* as 'alcoholic';" alcohol was thought to be toxic to livers that were "already damaged as a result of dietary deficiency" (American Foundation, 1955).

A second line of inquiry suggested that environmental toxins might be the cause of cirrhosis. Thus, in 1950, Lilienfeld and Korns criticized Jolliffe and Jellinek's review and presented epidemiological data that appeared to dissociate alcohol entirely from cirrhosis. As they put it,

It is abundantly clear that none of the hypotheses suggested explain the data presented even reasonably well. However, the distribution of deaths from cirrhosis, according to age, population size and socioeconomic groups, suggests that the cumulative influence of occupation, per se, may well be a rather important factor in the epidemiology of this disease. These observations lead one to re-examine the animal experimental studies of the relation of various chemical poisons to cirrhosis and to suggest the possible influence of industrial toxic substances on human cirrhosis. This would appear to be a rather fertile field for epidemiological investigation by those engaged in industrial hygiene. With cirrhosis mortality climbing each year to higher levels in the United States, such an investigation is necessary, particularly since it might reveal certain practical control measures

(Lilienfeld & Korns, 1950).

Sixteen years later, with the mortality rate from cirrhosis continuing to climb, Milton Terris responded in detail to Lilienfeld and Korns' paper by providing an elaborate array of epidemiologic evidence, across time in the United States and among other countries and by social class and occupation, that re-established the close association between levels of alcohol consumption and mortality rates from cirrhosis (Terris, 1967). Around the same time, clinical and animal studies were being published that confirmed alcohol's role in the production of cirrhosis (Lieber, 1990).

A third line of inquiry into cirrhosis was concerned not with its etiology but with its utility in predicting the prevalence of alcoholism. Because of the clear association between alcoholism and cirrhosis, Jellinek devised a formula, which came to bear his name, that estimated the number of alcoholics in the population for any given year based on the number of deaths from cirrhosis for that year. The formula is: $A = (PD/K) R$, where A is the number of alcoholics, D is the number of cirrhosis deaths, and P, K, and R are proportionality constants. P stands for the percentage of cirrhosis deaths attributable to alcoholism, K is the percentage of all alcoholics who die from cirrhosis, and R is the ratio of all alcoholics to those with complications. These constants, however, were modified over the years to values that were divined to be more appropriate (Keller, 1962; Popham, 1970; Roizen & Milkes, 1980). The net effect of this preoccupation with counting alcoholics was obfuscation of what

was becoming an epidemic of liver cirrhosis (Herd, 1984). Age-adjusted cirrhosis rates rose steadily from Repeal to 1973 (Grant *et al.*, 1988).

Cardiomyopathy

Most of the recent attention to the relationship between alcohol and heart disease has focused on moderate drinking as a possible way of reducing the risk of coronary artery disease. Indeed, a recent editorial in the *American Heart Journal* worries about “booze bashing” on the part of “a new breed of prohibitionists” who, in their eagerness to increase alcohol taxation and educate the public about the potential dangers of alcohol, might deprive the public of the benefits of moderate alcohol consumption on the heart (Kaplan, 1991). However, this diversion of cardiologists’ attention away from the hazards of heavy drinking is likely to be only temporary, because the cardiotoxic effects of heavy drinking are well documented. The best evidence that persistently heavy drinking causes direct damage to the heart muscle comes from the work of Urbano-Marquez and his colleagues (1989), who compared 50 well-nourished, employed males entering an alcohol treatment program in Barcelona with 50 male physicians and medical assistants. The mean daily ethanol intake for the alcoholics was 286 gm over an average of 16 years; the intake for the controls was less than 20 gm. On the basis of skeletal muscle strength, skeletal and myocardial muscle biopsy, echocardiography, and radionuclide cardiac scanning, they were able to clearly demonstrate dose-related destruction of both skeletal and cardiac muscle (Urbano-Marquez *et al.*, 1989).

Although deaths from alcoholic cardiomyopathy--a condition in which the heart becomes markedly stretched in size and weakened in pumping ability--are now reported as vital statistics (MMWR, 1990a), the true prevalence is unknown because clinicians frequently fail to recognize it (Paul, 1989). Alcoholic cardiomyopathy is a common cause of congestive heart failure among heavy drinkers (Wang, 1987), and preclinical lesions are frequently found when autopsies are performed on alcoholics (Steinberg & Hayden, 1981), but it can be hard to differentiate clinically from other causes of congestive heart failure that are unrelated or only secondarily related to heavy drinking (Wang, 1990). Nevertheless, annual reported alcoholic cardiomyopathy mortality rates appear to vary with the per capita consumption of alcohol (Smith, 1990). Like cirrhosis, its relationship to alcohol was minimized from the late 1930’s until the 1960’s.

In 1909, at the spring meeting of the American Society for the Study of Alcohol and Other Drug Narcotics, Dr. B.C. Keister warned his colleagues about reports of alcohol-induced damage to the heart muscle:

Our friends of the “Fatherland,” the Germans, have made some recent pathological discoveries resulting from the much beer drinking [sic] of that country. It is known among the physicians connected with the large hospitals of Germany as “beer-drinkers’ disease,” and is recognized by a special condition of unhealthy enlargement of the heart...

It is reliably reported that one in every sixteen of the hospital patients in Munich die from this disease

(Keister, 1909).

The term “Munchener Bierherz” was used in 1884 to describe what we now call alcoholic cardiomyopathy (Ferrans, 1966).

In 1893, Graham Steell, reporting on a series of 25 alcoholic patients with heart failure, commented, “not only do I now recognize alcoholism as one of the causes of muscle failure of the heart, but I find it a comparatively common one.” However, the causes of heart disease were less clear early the century than they are today, and by 1906 the same author, writing in his textbook on the heart, noted that “dropsy is especially apt to occur in cases of the cardiac muscle failure of beer drinkers and of the disease known as beri-beri” (quoted in Ferrans, 1966, p 90). Beriberi is a disease of nutritional deficiency that was relatively unknown until the 19th century and whose precise cause was not determined until 1926, when thiamine (vitamin B₁), the “anti-beriberi vitamin,” was first isolated in pure form. By 1937, the chemical synthesis of thiamine had been worked out, thereby providing an inexpensive source (Cline *et al.*, 1937). In any case, during the 1930’s there was a great deal of productive research on the relationship between thiamine deficiency and beriberi heart disease, and because vitamin deficiencies were common among alcoholics, the role of alcohol in the etiology of heart failure came to be doubted (Paul, 1989).

In 1937, the authors of a major study on the subject found so many of their alcoholic patients to have edema that resolved with the administration of thiamine that they concluded, “There is valid evidence indicating that alcohol per se cannot be primarily responsible for the manifestations observed” (Weiss & Wilkins, 1937). A year later, in 1938, these findings were replicated by others, who concluded, “It would appear, then, that alcohol per se is probably not the cause of the cardiovascular disturbances which occur in the alcohol addict” (Goodhart & Jolliffe, 1938). There are substantial clinical differences between the heart failure caused by beriberi and that caused by alcohol, but these were not apparent at the time. Finally, experimental evidence that normal concentrations of alcohol have little acute effect on the heart muscle led the then-eminent pathologist and pharmacologist Arthur Grollman to conclude that “the so-called ‘beer-drinker’s heart’ is probably due to the plethora induced by large volumes of malted fluids rather than to their alcoholic content” (Moore, 1941, p 850).

Ironically, it was because of other advances in cardiology that interest in alcohol as a potential cause of heart disease lay fallow until the late 1950’s. Hypertension and coronary artery disease were found to be important causes of heart disease, and they became the primary focus in cardiology (Burch, 1964). Not until the 1960’s did it become intellectually fashionable to talk about primary myocardial (heart muscle) disease (as opposed to heart disease secondary to some sort of circulatory problem or valve defect) (Ferrans, 1966).

Fetal Alcohol Syndrome

The adverse effects that heavy maternal drinking can have on the fetus are so well known that they require no introduction. However, only 20 years have elapsed since the problem was rediscovered (Jones *et al.*, 1973).

Two hundred and fifty years ago, when the unrestrained production and consumption of inexpensive distilled beverage was causing what subsequently became known as the London

Gin Epidemic, there was widespread concern about the effects of excessive maternal drinking: “What must become of an infant who is conceived in gin?” asked Henry Fielding, “with the poisonous distillation of which it is nourished, both in the womb and at the breast” (quoted in Coffey, 1966, p 672; Rodin, 1981, p 1238). In 1726, the Royal College of Physicians told Parliament that gin was “too often the cause of weak, feeble and distempered children, who must be, instead of an advantage and strength, a charge to their country” (Royal College of Physicians, 1987). A physician writing in 1981 diagnosed fetal alcohol syndrome in the infant that is so prominently flying out of its mother’s arms in William Hogarth’s 1751 print, *Gin Lane*:

The facial appearance of the child is worthy of note. The eyes have a shorter than normal palpebral fissure, resulting in relatively round, “Orphan Annie” eyes. These are one of the features of the effects of maternal alcohol on fetal morphogenesis, described by Jones et al as part of the fetal alcohol syndrome.

Short palpebral fissures are not depicted by Hogarth in infants and young children in his other engravings

(Rodin, 1981).

It is not unusual to find vivid and accurate portrayals of medical syndromes in pre-20th-century literature. For example, Thomas Sydenham’s 17th-century description of gout is still quoted in contemporary textbooks. What is striking, however, is the contrast between the current (and 18th- and 19th-century) appreciation of the fetal alcohol syndrome and the certainty with which post-Repeal medical scientists ruled out the possibility that alcohol might be injurious to the fetus: “The fact is that no acceptable evidence has ever been offered to show that acute alcoholic intoxication...is the cause of any abnormality in the child,” said Haggard and Jellinek (1942, p 207). Keller, in 1955, wrote, “the old notions about children of drunken parents being born defective can be cast aside, together with the idea that alcohol can directly irritate and injure the sex glands” (quoted in Warner & Rosett, 1975, p 1411). This rejection needs to be considered in reference to the early-20th-century debate that preceded it.

The possible effects of parental drinking on offspring were widely debated during Prohibition, but in a context that seems quite foreign to contemporary thinking. In order to understand that context, we first need to recognize that throughout the 19th century and for much of the 20th century, there was little distinction between heredity and prenatal effects (Abel, 1990; Warner & Rosett, 1975). It was not until 1962 that the concept of teratogenesis (the production of physical defects in offspring in utero) gained widespread acceptance, after the maternal ingestion of the sedative thalidomide was shown to be a major cause of birth defects (Woollam, 1962).

The 1920’s, when Prohibition was in effect, were the peak years of the eugenics movement. Better Babies and Fitter Families contests were featured at state fairs. As one brochure proclaimed, “The time has come when the science of human husbandry must be developed, based on the principles now followed by scientific agriculture, if the better elements of our society are to dominate or even survive” (quoted in Kevles, 1985, p 61). Not surprisingly,

the eugenic effects of alcohol were a frequent theme of the ‘wet-dry’ debates that went on throughout Prohibition (Jones, 1961).

Briefly stated, ‘dry’ arguments were based on the idea of inheritance of acquired characteristics, which had not been completely replaced by modern views of heredity. From this perspective, statistical evidence suggesting a greater likelihood of mental and physical defects among the offspring of chronic inebriates supported the notion that alcohol was a racial poison, as articulated in this poster which was produced for classroom use by the National Education Association:

The children of drinking parents show a strong tendency toward weakened mentality--there are more idiots and inferior individuals among them. It is even probable that the germ plasm itself--that vital spark which continues on thru countless centuries--is so affected by alcohol that the children for generations to come suffer from the sins of the fathers. It is the race that counts and alcohol is an enemy of its advance.--Doctor K. Herman Bouman, noted Dutch scientist and psychiatrist

(quoted in Jones, 1963, p 160).

Initially, ‘wets’ cast doubt on the idea of alcohol-induced germ damage by pointing out that the dominant races on the planet were drinkers. If alcohol was harmful to race, then how could hard-drinking Englishmen have come to dominate India, which was peopled with nondrinking Hindus and Muslims? Later in the debate, ‘wets’ drew ammunition from laboratory studies which showed that alcoholized animals were more likely to produce malformed offspring in the first generation but that subsequent generations were more robust as a result of the alcohol exposure. Thus, alcohol was deemed to have a positive eugenic effect in selecting out the weaker of the species, and Prohibition was anti-eugenic because it protected the weak and defective. As Charles Stockard, a professor at Cornell argued,

There are a certain class of weak mediocre people or individuals who don’t seem to amount to anything and you want to take them on your lap...and coddle their mediocrity, and humor them, but alcohol, if used in a eugenic way, will prevent such people from even being born...

We can’t look at this from an ethical or humanitarian standpoint; we’ve got to consider it on a scientific basis. If you go to breed horses or dogs or pigs or any of those things, you must, and do, go at it scientifically. If you don’t do that soon...we shall eventually go even below the level of mediocrity; and that is not what you want to do to the human race

(quoted in Jones, 1963, pp 164–5).

By the time post-Repeal scientists had organized themselves to investigate alcohol problems, the eugenics movement was in shambles, discredited by its poor science and underlying racism. Furthermore, the eugenic practices of the Nazis made the entire subject repugnant. Therefore, near the end of the War, when Yale psychologist Anne Roe (1944) found similar social adjustment and drinking patterns among 36 offspring of alcoholics and 25 offspring of normal parents, all of whom had been raised in foster-homes, it was with

some triumph that she was able to conclude (despite design flaws) that environment and not alcohol was the cause of problems among the offspring of alcoholics. The modern alcoholism movement had succeeded in distancing itself from the uncomfortable science of the past.

Esophageal Cancer

The association between heavy drinking and cancer is well documented, at least epidemiologically, but it does not usually receive as much attention as other alcohol-related health problems, such as cirrhosis of the liver or fetal alcohol syndrome (NIAAA, 1990). Relationships between cancer mortality and alcohol consumption have been shown in comparisons among abstainers and drinkers, in retrospective and prospective studies, and in data correlating per capita consumption of alcohol and age-adjusted cancer mortality (Driver and Swann, 1987; Schoenberg, 1971; Turner and Anderson, 1990; Tuyns, 1990). Esophageal cancer is an important contributor to overall alcohol-related mortality. For example, in reports for the United States as a whole and for individual states such as California and Wisconsin, the number of alcohol-attributed deaths from esophageal cancer approaches that for alcoholic cirrhosis of the liver (MMWR, 1990a and 1990b; Sutocky, 1990).

Although the possibility of a link between alcohol and cancer received less prominence in early-20th-century scientific literature than that between alcohol and other health problems, it was by no means unknown. Speaking before the Royal College of Surgeons in 1910, Sir Alfred Gould listed age, chronic irritation, prolonged exposure to x-rays, and alcohol as major factors in the etiology of cancer:

The influence of alcohol on the incidence of cancer is really important. Dr. Tatham's statistics of the mortality of various occupations from certain diseases revealed the fact that cancer is twice as frequent among brewers and London publicans as among clergymen, and that the cancer incidence in any trade varies with the attendant habits as regards alcohol

(Gould, 1910).

Similar associations were noted by other early-20th-century scientists (Hoffman, 1915; Horsely & Sturge, 1915; Gilford, 1925).

However, there is no experimental model for alcohol carcinogenesis, perhaps because alcohol is a *cocarcinogen*--it facilitates the activity of carcinogens that are present in tobacco smoke, in various foods, or in other environmental sources. This lack of experimental evidence was of paramount importance during the 1930's, 1940's, and 1950's, when cancer researchers downplayed epidemiologic evidence that could not be supported by evidence from the laboratory (Patterson, 1987). Therefore, earlier reports associating alcohol with cancer did not meet the standards for medical evidence that existed at the end of Prohibition, allowing the conclusion that "no connection can be shown to exist between the susceptibility to, or the incidence of, cancer and the use or abuse of alcohol in any form whatever, in spite of the fact that a few statistical enquiries have appeared to point towards such a relationship" (Emerson, 1932, p 197). Haggard and Jellinek (1942) drew a similar conclusion, criticizing

the investigators who “have attempted to show that cancer of the esophagus and stomach may be caused by irritation from strong alcoholic beverages. These assertions are based entirely on statistical studies of death rates.” They classified the alleged relationship between alcohol and cancer as one of several fallacies about alcohol, concluding that, if anything, it appeared “that cancer is less frequent among chronic alcoholics” (Haggard & Jellinek, 1942, pp 192–194).

Diverse Problems with Shared Historical Themes

None of the problems described here are apparent in most heavy drinkers. Even cirrhosis of the liver, which is among the best documented of the alcohol-related diseases, fails to occur in nine out of ten heavy drinkers (Grant, 1988). Such a relationship might be dismissed as “merely statistical”—and it was. When E.M. Jellinek plotted cirrhosis deaths against per capita alcohol consumption in 1939 for 45 states, he found a linear relationship, but he could not bring himself draw a straight line in his figure. Although he acknowledged the “straight line relationship,” he qualified its meaning, and he cautioned: “This correlation should not be used as an argument that individuals who drink much are liable to die from cirrhosis of the liver” (Jolliffe & Jellinek, 1942, p 294). Jolliffe and Jellinek’s thorough review of the literature contained an abundance of statistical evidence, from clinical studies and from general mortality studies, that linked heavy drinking with cirrhosis of the liver, but the lack of a good experimental model was a major impediment to accepting that statistical evidence as anything more than a “sidelight:”

Even the most valid statistics, vital or clinical, cannot go beyond the point of indicating the association or nonassociation between cirrhosis of the liver and inebriety. These investigations in no way answer the question whether or not there is direct causation of cirrhosis of the liver by alcohol. Even statistics of 100 per cent validity showing the association could not be interpreted in the terms that cirrhosis is caused by alcohol. The statistics merely indicate that among chronic alcoholics the occurrence of, as well as the death from, liver cirrhosis is significantly greater than among temperate persons. Etiologic theories must be based on physiologic experiment and physiologic reasoning. The statistical sidelights aid etiologic research only inasmuch as they show that a connection between inebriety and cirrhosis of the liver cannot be rejected and that, therefore, etiologic investigations of the question are not vain undertakings

(Jolliffe & Jellinek, 1942, p 295).

Today, statistical evidence carries more weight. The standards of medical evidence for disease etiology changed in the decades that followed (Burnham, 1989; Susser, 1985). In order to understand that change, which I believe is central to understanding why medical scientists were skeptical about the role of alcohol in the etiology of disease, it is necessary to look at the larger historical context of scientific medicine.

Changing Intellectual Contexts for Scientific Medicine

Shryock’s (1979) history of medicine, which is a standard text, begins with Benjamin Rush, who is familiar to alcohol scholars as the father of the temperance movement (Levine,

1978). Shryock begins with Rush because he “was so unfortunate as to be one of the last leaders of a medicine not entirely divested of the medieval tradition; while the very next generation witnessed the metamorphosis of this old art into a modern science.” Rush’s system of medicine characterized all disease as sharing a common basis (excessively stimulated nervous tissue), which, in turn, required the same treatment (aggressive bloodletting and purging). His treatments were, as Shryock put it, “if good for anything, literally ‘good for what ails you,’ since by definition the same thing ailed everyone” (Shryock, 1979).

Rush lived in an era when there was general disappointment that science had not produced any tangible benefits in medicine; it was an era when medicine was referred to as “the withered arm of science” (Shryock, 1979). The specific causes of diseases were unknown; they were attributed to miasmas or to constitutional imbalances. Medicine had not yet acquired a scientific paradigm (Kuhn, 1970) for disease.

Medicine acquired a paradigm for disease in 1876, when Koch inoculated a healthy animal with anthrax bacilli and then recovered the same organisms from the sick and infected animal. Later he found the cholera bacillus. Before the end of the century, Koch identified the microorganism that caused tuberculosis, then the single leading cause of death. The implications were enormous--there were specific causes for specific diseases. Pasteur, Koch, and others developed effective vaccines. Sanitation measures aimed at controlling the spread of infectious diseases were implemented. Sulfonamides were developed during the 1930’s, and penicillin became available during the 1940’s.

Between 1900 and 1942, life expectancy for males increased from approximately 50 to 64 years (Shryock, 1979). This increase in longevity dramatically changed the face of disease. Heart disease and cancer became the leading causes of death. Although their ranking in mortality statistics has not changed during the past half century, they are understood quite differently today. In 1942, when Haggard and Jellinek’s *Alcohol Explored* was first published, heart disease and cancer were generally thought of as degenerative diseases whose greater prevalence was to be expected with the greater number of people surviving to older age. Today, we think of coronary heart disease, which is the most frequent type of heart disease, in terms of risk factors like age, sex, blood pressure, serum cholesterol, tobacco use, and exercise habits. Likewise, risk factors have been identified for many forms of cancer. During the past half century, a new paradigm for chronic disease, based on epidemiology, has evolved. Many diseases are now seen as having environmental causes; hence, they are viewed as being preventable. However, the techniques that are central to this paradigm for chronic disease are relatively new, at least in terms of the historical span that is the subject of this paper. The first textbook to describe these epidemiologic techniques was not published until 1960 (Susser, 1985).

When the Framingham Heart Study, which established the idea of cardiovascular risk factor scores and is the prototype of the cohort study, was set up in 1947, its intention was to determine the incidence rate of heart disease. Not until 1949 was the design modified to determine “factors influencing the development of heart disease” (Susser, 1985).

The change in standards of medical evidence that came with the epidemiologic paradigm for noninfectious disease can be seen in the history of lung cancer. At the beginning of the century, when most tobacco was being either chewed or smoked as cigars or pipes (which are not inhaled), lung cancer was a rare disease that occurred primarily among the Central European miners of radium-rich ores (Johnson, 1985; Osler, 1907). As a result of the steep and steady increase in cigarette consumption that began after World War I (and continued until the first Surgeon General's Report in 1964), lung cancer became increasingly more common (Patterson, 1987; Slade, 1989). When Ochsner and DeBaakey reported on their experience with pneumonectomy in cancer of the lung in 1939, they observed, based on clinical impression, that the increasing incidence of lung cancer was related to the practice of inhaling tobacco smoke. However, Ochsner's old teacher, Evarts Graham, commented, "Yes, there is a parallel increase between the sale of cigarettes and the incidence of cancer of the lung, but there is also a parallel increase between the sale of nylon stockings and cancer of the lung" (quoted in Burnham, 1989, p 18). By 1950, Graham felt differently. Together with Ernest Wynder, he published one of the early case-control studies that established the association between smoking and lung cancer (Wynder & Graham, 1950). In 1985, the *Journal of the American Medical Association* republished their paper as a "Landmark Article" (Wynder & Graham, 1985), though the journal's editor had been reluctant to publish the paper in 1950 (Susser, 1985). And its landmark status was not immediately apparent; the journal continued its publication of advertisements for cigarettes until 1954 (Lundberg, 1985).

If medical scientists were slow to accept this new kind of evidence about disease causation as applied to cigarettes, they were even more skeptical when it came to alcohol.

Skeptical Alcohol Researchers

Many post-Repeal researchers were skeptical about alcohol's harmful effects because, unlike their abstinent parents, they were moderate drinkers. Moderate drinking was one of the major symbols of the cultural divide between the Victorian age and the modern age. Many of those who attended college during the 1920's drank, despite Prohibition. Scientists, like other members of their social class, tended to be moderate drinkers. For these individuals, temperance ideology was quintessentially unmodern (Johnson, 1973; Room, 1984). One of the central tenants of temperance thought was the idea that moderate drinking was dangerous. As late as the 1930's, this idea had been an impediment to the funding of any sort of alcohol education campaign that condoned moderate drinking; it had even been the basis for the burning of a scientific report to the Virginia Legislature (Roizen, 1991a). Since most researchers were moderate drinkers, they were personally at odds with temperance ideology. Outrageous claims about alcohol's harmful effects had been the stock in trade of the "scientific temperance" curricula that the WCTU had successfully mandated for public schools in nearly every state. Hence, post-Repeal researchers were naturally skeptical about alcohol's harmful effects.

Furthermore, epidemiologic thinking had not, by and large, been employed in the understanding of noninfectious diseases, and, as noted above, the techniques had not yet been invented (Susser, 1985). The epidemiologic evidence linking heavy drinking with

cirrhosis of the liver and with cardiomyopathy was eventually supported by experimental evidence. In the cases of fetal alcohol syndrome and cancer, however, the idea of alcohol's role as an etiologic factor rests on the strength of epidemiologic evidence alone. The standards of medical evidence have changed.

Moreover, successes in the application of laboratory science to bedside medicine had been so dazzling that anything that could not be proven in the laboratory or with a clinical trial--anything "merely statistical"--was suspect. In addition to the triumphs over infectious disease noted above, there had been tremendous advances in endocrinology and nutrition. One of the most dramatic of these medical advances occurred during the midst of Prohibition with the discovery of insulin. Before insulin, diabetics could be kept from coma and death only by near-starvation. Diabetic children of the pre-insulin era appear in photographs with the emaciated look of concentration camp victims. Insulin awakened diabetics from coma and allowed them to gain weight and lead productive lives (Bliss, 1982). The medical miracle that followed insulin was the discovery and synthesis of the B vitamins, whose effects in those with pellagra and beriberi were equally as dramatic. These vitamins became widely available within a few years after Repeal, and one of the segments of the population that needed them most was the urban alcoholic.

Success with Vitamins

In an era before the addition of vitamins to baked goods, and during the Depression, when malnutrition was widespread, vitamin deficiencies were common among alcoholics, who received most of their caloric intake from whiskey. More than 20 per cent of these alcoholics, even when sober, were unable to move their eyes freely, staggered when they walked, were unable to maintain their balance, or complained of burning of the soles of their feet. These symptoms, which are the polyneuropathy of Wernicke's disease, a manifestation of beriberi, as well as the heart failure and edema of the so-called "wet beriberi," responded to the administration of vitamin B₁ (thiamine). Mental symptoms among these alcoholics were often improved or reversed with the administration of either thiamine or another B vitamin, niacinamide. Their sore mouths and swollen tongues responded to niacinamide. When vitamins became available, it appeared that they might be the cure for all alcohol-related diseases (Askey, 1939; Blackenhorn & Spies, 1936; Goodhardt & Jolliffe, 1938; Jolliffe & Goodhardt, 1938; Jolliffe, 1940; Jolliffe, 1941; Jolliffe & Wortis, 1941; Hibbs, 1946; Minot, 1933; Weiss & Wilkins, 1937).

The chemical synthesis of vitamin B₁ (thiamine), which was described in 1937 (Cline *et al.*, 1937), provided an abundant and inexpensive source of the vitamin. Among the first to receive the newly available vitamin, supplied courtesy of Merck and Co., were the alcoholics who were admitted to Bellevue Hospital in New York, where Norman Jolliffe was evaluating their response. Jolliffe's picture appeared in *Time* magazine along with a report on his success and his alleged recommendation that vitamin B₁ be added to whiskey (Anon., 1938b). The addition of vitamins to alcoholic beverages was given serious consideration but ultimately rejected because of the possibly misleading impression of curative or therapeutic effect that the vitamin content on the label might imply (Anon., 1940/1941). Jolliffe calculated the ratio of vitamin B₁ to calories in typical diets and

established recommendations for daily requirements (Jolliffe, 1940). However, when the lay publication *Science News Letter* reported on his work, it emphasized his comment that better diets in 1840 allowed Americans of that era to consume more of their calories as whiskey. The large-type headline for this report was “1840 Americans Could Drink Quart of Whisky a Day: Harm from Alcohol Comes from Increasing Calories in Proportion to B₁ Intake; Diet Worse Now” (Anon., 1940).

Norman Jolliffe made important contributions to the field of nutrition, as a teacher and researcher, as the editor of a major textbook, and as instigator of the policy of supplementing bread products with vitamins. Later in his life, he became concerned with the relationships among dietary fat, serum cholesterol, and the incidence of coronary artery disease (Williams, 1963). His last publications, which appeared after his death, in 1961, from the complications of diabetes, were papers describing the effects of dietary control in reducing serum cholesterol and a new chapter in the second edition of his nutrition text on dietary treatment of hypercholesterolemia (Jolliffe, 1961, 1962a, and 1962b). He embraced the new paradigm for chronic disease, but by then he had left the alcohol field, and there was still no alcohol research that embodied this new perspective. Jolliffe was important to the history of alcohol research because he was the principle investigator in the Carnegie-funded review of the literature on the effects of alcohol on man, which was the first major alcohol research project after Repeal (Roizen, 1991a). And it was he who hired both Mark Keller and E.M. Jellinek, researchers who would go on to become important personalities in the new research on alcoholism.

Optimism About Alcoholism

In his 1931 book for the lay public, psychiatrist Karl Bowman explained,

There is no evidence that the average, normal individual is harmed in any way by the use of moderate amounts of alcoholic beverages. It is true that certain unstable individuals are affected unfavorably by the use of alcohol and should leave it entirely alone. There is also no question but that the excessive use of alcohol is harmful both mentally and physically

(Bowman, 1931).

By 1939, Bowman was quite familiar with the impressive results of Jolliffe's vitamin research; as Professor of Psychiatry at New York University (NYU) College of Medicine and Director of the Division of Psychiatry of New York's Department of Hospitals, he oversaw Jolliffe's research in NYU's Psychiatric Medical Service at Bellevue Hospital (Cattell, 1938). More importantly, as Chairman of the Executive Committee of the Research Council on Problems of Alcohol, Bowman had participated in the selection of Jolliffe as principle investigator of the Carnegie-funded literature review on the effects of alcohol on man (Roizen, 1991a).

As Ron Roizen's recent research has shown, Karl Bowman played a pivotal role in the birth of the modern alcoholism movement. In 1939, when other sources of funding for alcohol research were not forthcoming, he encouraged the Council to accept funds from the distilling industry. In order to avoid any charges of bias in the Council's work, he limited the

scope of its research to alcoholism, a problem of concern to both ‘wets’ and ‘drys’ (Roizen, 1991a).

Bowman’s decision to abandon other lines of alcohol research was pragmatic, but I will argue that it reflected his genuine optimism that it was the right choice. After all, Jolliffe and others were providing evidence suggesting that most, if not all, of the adverse mental and physical effects of heavy drinking were secondary to vitamin deficiency. His own research on the effects of vitamin B₁ injections in alcoholics with Korsakoff psychosis (recent memory loss, confabulation, and disorientation) had shown promising results (Bowman *et al.*, 1939). Furthermore, a new drug called amphetamine (Benzadrine) seemed to be of great value in the treatment of alcoholism (Bloomberg, 1939).

Psychiatry was at a crossroads. In 1940, more than two-thirds of the members of the American Psychiatric Association were providing custodial care for chronic patients in public mental hospitals. During the 1930’s, the populations of these hospitals were decreasing because of the decline in mental illness caused by neurosyphilis and pellagra. Also, the rise of scientific medicine had underscored the backwardness of custodial care (Grob, 1987; Valenstein, 1986). As Grob (1987, p 412) put it, “Under such circumstances it was understandable that twentieth-century psychiatrists attempted to redefine the concepts of mental disease and therapeutic interventions, as well as the very context in which they practiced.” In fact, psychiatry’s history after the War was to unfold in ways that placed alcoholism at the periphery of its concerns (Joint Commission, 1961; Grob, 1987; Romano, 1990).

In 1939, however, alcoholism appeared to be a bright spot in psychiatry’s future, and the Council’s most active members were psychiatrists. By 1940, the modern alcoholism movement was centered at Yale, in Howard Haggard’s Laboratory of Applied Physiology, and, after 1944, enthusiasm for the disease concept of alcoholism was fueled by Alcoholics Anonymous member Marty Mann, who applied her considerable energy and public relations expertise to popularizing the disease concept. Although Howard Haggard did not share Marty Mann’s conviction that the physiological basis for alcoholism was an established scientific fact, he was sympathetic to the idea. And so was E.M. Jellinek. Jellinek encouraged Haggard to employ Mann, who saw herself as a modern day Dorothea Dix, as the “wheelhorse” of the movement to accept the “scientific approach” to alcoholism (Johnson, 1973).

Alcoholism versus Moderate Drinking

Today, an “alcohol problems” perspective is replacing the earlier preoccupation with a unified disease concept (Moore & Gerstein, 1981; Room, 1983; Institute of Medicine, 1990). Any of the alcohol-related health problems described in this paper can occur without the glaring social and psychological consequences that are typically associated with alcoholism. Admittedly, the risk for alcohol-related health problems is quite small or even negligible with moderate levels of intake, such as one or two drinks per day. But E.M. Jellinek was more liberal in defining moderate drinking:

We cannot arrive at a definite standard, especially not in relation to quantity. I should like to quote a passage from Dr. Haven Emerson: "Alcohol, if used with the above precautions, should be taken only after the day's work of mental and physical effort has been completed, and only if inactivity or at least no responsibility or demand for skilled performance is reasonably assured for the next couple of hours."

Dr. Emerson is opposed to the use of alcohol. Presumably he states these criteria for its moderate use because of the realization that as long as we live in a drinking world such a statement may be useful. Quantity, in itself, does not determine moderate use, although, naturally, there is a limit above which there can be no question of moderation. What Dr. Emerson means is that one can speak of moderation only when the moderate intake will not interfere with the responsibilities of the drinker; that is, when the question of efficiency is not involved, and when the only aim is relaxation

(Jellinek, 1943).

By such standards one could engage in some fairly heavy drinking patterns--for example, a liter of wine every night with dinner, with additional drinks on the weekends--and still be a moderate drinker. From the standpoint of health risks, as defined by post-Repeal scientists, such a drinking pattern could be acceptable, provided one had a balanced diet. Not until fairly recently did the journalist Leonard Gross (1983) formulate the question, "How much is too much?" Heavy drinking is an important public health problem in its own right, regardless of whether or not the diagnosis of alcoholism can be applied.

Conclusions

Post-Repeal medical scientists created a new image for alcohol. They said it did not cause disease, overturning earlier claims that had been made in the name of temperance. They promoted the view that alcohol was relatively harmless, except in a few individuals, who, for unknown reasons, were alcoholics. The relationship of alcohol to disease became like that of sugar to diabetes. And alcoholism research promised to turn up something as effective as insulin. In the introduction to his penetrating series of essays on turn of the century science, Charles Rosenberg commented,

Anthropologists have long been aware of the centrality of medical theory and practice in the understanding of a particular culture's beliefs. Indeed, it is probably fair to say that the use of disease as a sanction in enforcing behavioral norms is almost universal and that the speculative etiologies which justify such social usage are always consistent with a cultures's most fundamental patterns of belief

(Rosenberg, 1976).

Was there really an eclipse of knowledge about alcohol's harmful effects? In a practical sense, there was. But viewed within the context of the history of medical science, there has been a transformation of knowledge about alcohol. What was plausible before Prohibition, and implausible afterwards, became established with the aid of a new kind of medical evidence.

The risks and benefits associated with various levels of alcohol consumption are still being established (Babor *et al.*, 1987; Kendell, 1987). Unfortunately, the epidemiology of noninfectious diseases is relatively new, and studies of poor scientific quality are frequently published in peer-reviewed journals, only to be amplified by the news media as newfound hazards of everyday life (Feinstein, 1988).

Persistently heavy drinking, however, has been clearly established as an important cause of many commonly occurring diseases. The historical consequences of these findings are still unfolding.

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