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Medical Understandings of Alcohol and the Liver in Twentieth-Century Britain

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Abstract

This thesis foregrounds the importance of medicine in shaping the public discussions on alcohol misuse. A wide range of medical and non-medical sources reveal the factors that contributed to the shifting medical understandings of alcohol and its relation to the liver, as well as exposing the subsequent impact of such shifts on the policy debates over alcohol and licensing in twentieth-century Britain. Instead of being a product of changing social and cultural attitudes towards drink, I argue that the medical knowledge of alcohol's causation of cirrhosis were formulated on a set of strict scientific criteria that took into account the available evidence. Although assumed today to be a direct outcome of heavy drinking, cirrhosis was shown in clinical and experimental studies from the middle of the century to be most likely caused by nutritional deficiency. The direct toxicity of alcohol to the liver was further demonstrated in the 1970s through the successful reproduction of cirrhosis in experimental animals. As the quintessential illness of the heavy drinker, cirrhosis was often at the heart of the dialogue surrounding alcohol use and harm in Britain. The variable extent to which drink was understood to be culpable for liver damage had a direct bearing on how different groups, including the temperance movement and the alcoholic beverage industry, exaggerated or downplayed alcohol's destructive properties. Contrary to the tendency of the existing historical literature to disassociate the perceptions and responses to alcohol use from the scientific knowledge on its effects on the body, this thesis shows that expert medical understandings of alcohol and the liver influenced how drink was conceptualised in relation to harm.

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Contents

List of Acronyms

ALD – Alcoholic Liver Disease
DTT – Direct Toxicity Theory
NDT – Nutritional Deficiency Theory
MDTT – Modern Direct Toxicity Theory
MP – Member of Parliament
HC – House of Commons
HL – House of Lords
BMA – British Medical Association
BMTA – British Medical Temperance Association
CCB – Central Control Board

NIH – National Institute of Health

Introduction

The twentieth century witnessed the relative significance of alcohol use 'ebb and flow' as a social problem in Britain.[1] The late nineteenth and early twentieth centuries saw the height of political hostilities between the temperance movement, a campaign that pushed for legislative solutions to the problem of drunkenness in society, and the liquor trade, the collective interests that benefitted from the sale of alcoholic beverages. This conflict eventually culminated in the implementation of some of the most stringent state controls ever imposed on alcohol and licensing in Britain during the First World War. The perceived effectiveness of these regulations resulted in the establishment of a peacetime settlement to preserve many of the wartime controls in a much more modest form.[2] Such developments produced an alcohol debate that was markedly diminished in its ferocity throughout the interwar and postwar eras, as the British seemingly drank in moderation to an extent never seen before. However, following a resurgence in alcohol consumption and the incidence of many of its perceived harms, the 'drink question' was once again brought back to the public agenda in the 1970s by campaign groups who called for alcohol use to be dealt as a public health issue.

Among the multiplicity of recognised harms known to result from the misuse of alcohol, one was present at every phase of the alcohol debate: liver disease. In the present day, cirrhosis is commonly understood to be one of the deadliest chronic diseases brought about by the long-term abuse of alcohol. As a debilitating and potentially life-threatening illness, cirrhosis has been used to highlight the deleterious effects that heavy drinking can have on health. While alcohol's precise relationship to many of its purported problems continue to be debated today,

[1] Craig Reinarman, 'The Social Construction of an Alcohol Problem: The Case of Mothers Against Drunk Drivers and Social Control in the 1980s', *Theory and Society* 17 (1988), p. 91.
[2] John Greenaway, *Drink and British Politics Since 1830: A Study in Policy-Making* (Basingstoke, 2003), pp. 111-2.

virtually all observers now agree that it possesses a direct toxic action in damaging the liver.[3] However, this was not always the case. Throughout the twentieth century, medical understandings of the culpability of alcohol in the development of cirrhosis underwent a striking series of evolutions. The causality between the two were questioned by medical professionals throughout the period, some even going as far as to argue that alcohol played nothing more than an indirect role in enabling some other factor in damaging the liver. Furthermore, the variable extent to which alcohol was understood to be responsible for causing cirrhosis inevitably influenced how drink was problematised in society. Hence, focusing specifically on the liver, this thesis highlights the integral role played by expert knowledge of physiological diseases in shaping the public discourses surrounding alcohol use in Britain. It also explores the variety of factors that contributed to dramatic shifts in the medical understandings of alcohol and its causation in cirrhosis.

Alcohol, the Liver, and Disease

An outline of the basic facts and assumptions concerning the liver as an organ, the action of alcohol on the liver, and different categories of liver damage will make the task of grasping the historical shifts in the causal understandings of cirrhosis less daunting. This section also touches on knowledge on cirrhosis predating the twentieth century to show that the intimate association between alcohol and liver disease was not without its pre-modern foundations. It should be noted, however, that the present knowledge on the disease would not be portrayed as the objective 'truth' or a necessary endpoint of past scientific developments. Modern-day medical understandings, much like their historical predecessors, are contingent to scientific assumptions and methodological norms that are prevalent today and continue to be open to change. As this thesis will explore, present understandings were shaped by changes in the scientific knowledge on cirrhosis causation that occurred throughout the past century or so.

The liver is the largest and heaviest internal organ in the human body, and is located adjacent to the stomach on the upper right side of the abdomen. Rather than being just a

[3] World Health Organization, *Global Status Report on Alcohol and Health 2014* (Luxembourg, 2014), p. 12.

random conglomeration of cells, the organ possesses a highly organised structure whereby different types of cells are elaborately arranged to maximise functional efficiency. As part of the gastrointestinal system, the liver performs multiple functions, including protein synthesis, the production of bile to aid digestion, and the storage of sugar, vitamins, and other chemicals. Another important function of the liver is its capacity to metabolise toxins such as alcohol. When alcohol enters the body, it gets absorbed into the bloodstream through the stomach and the small intestine, much of it eventually ending up in the liver to be detoxified. Here, the organ breaks down alcohol into a compound called acetaldehyde.[4] Owing to its toxic properties, the liver prioritises the metabolisation of acetaldehyde in the place of fat. Thus, the habitual consumption of alcohol can result in the infiltration of excess fatty acids in the liver cells. This condition is known as *fatty liver*, or *steatosis*, the earliest stage of *alcoholic liver disease* (ALD). Fatty liver itself is not seen to be a serious health condition because it is mostly asymptomatic and the damage incurred can be reversed upon the cessation of alcohol consumption. However, continued abuse of alcohol can lead to the development of *alcoholic hepatitis*, the second stage of ALD involving the inflammation of the organ. Alcoholic hepatitis is a serious condition that could result in death upon acute liver failure, although the damage, again, is still reversible once the patient chooses to abstain. Eventually, ALD may enter its final, irreversible stage in the form of *cirrhosis*. Cirrhosis has two components: excessive scar tissue infiltrating the liver as the outcome of ongoing damage over many years, and the disruption of the normally elaborate organisational structure of the liver resulting from its chaotic regeneration and repair. Morphologically, the surface of the liver becomes hardened and covered in lumps, or 'nodules'. Cirrhosis can lead to a myriad of symptoms associated with the diminished functional capability of the liver. The disruption of blood flow through the portal vein connecting to the liver results in ascites, the build-up of fluid in the abdomen. The incapacity of the liver to excrete bilirubin, a pigment produced by the breakdown of aging red blood cells, leads to the yellowing of the skin, a condition known as jaundice. Cirrhosis can also lead to a number of fatal complications: brain 'fog' or encephalopathy, often progressing to confusion and coma as

[4] Charles S. Lieber, 'Alcoholic fatty liver: its pathogenesis and mechanism of progression to inflammation and fibrosis', *Alcohol* 34.1 (2004), p. 9

a result of liver failure; the further development of liver cancer; and a heightened risk of developing infectious diseases due to a compromised immune system. The public concern over the incidence of cirrhosis can be partially explained by the often-devastating course of the disease. Indeed, cirrhosis is one of the leading causes of mortality among heavy drinkers, and ALD is seldom detected in its early stages since it does not become a serious 'problem' for the patient until symptoms such as ascites begin to develop. Unfortunately, it is often too late by then since the patient will already have a significantly reduced survival rate.[5]

This current medical orthodoxy paints a stark picture of the relationship between alcohol and liver disease. However, as it is the case with many chronic diseases, the relationship between cirrhosis and its most commonly recognised cause, alcohol, reveals itself to be more complex than that of a simple cause-and-effect relationship. Throughout the twentieth century, cirrhosis was never understood to be caused solely by alcohol, having been associated with other purported aetiologies such as syphilis, tuberculosis, and malaria.[6] Today, the disease is also known to be caused by obesity and chronic viral hepatitis, the latter of which accounts for just over half of all cirrhosis deaths worldwide.[7] In Europe, however, drink is responsible for the majority of its incidence due to higher levels of alcohol consumption over other continents.[8] Yet even then, cirrhosis has been found to develop in only a minority of heavy drinkers. Estimates on the prevalence of the disease among long-term abusers range anywhere between 10 to 35 per cent.[9] This variable predisposition was regularly suggested throughout the

[5] Julie Steen Pedersen, Flemming Bendtsten, and Søren Møller, 'Management of cirrhosis ascites', *Therapeutic Advances in Chronic Disease* 6.3 (2015), p. 124.

[6] Alexander Wheeler, and William R. Jack, *Wheeler's Handbook of Medicine and Therapeutics* (3rd edn, Edinburgh, 1908), p. 197; Arthur F. Hurst, 'Diseases of the Digestive System', in Frederick W. Price (ed.), *A Textbook of the Practice of Medicine* (5th edn, London, 1937), p. 701.

[7] Roger Williams, et al., 'Addressing liver disease in the UK: a blueprint for attaining excellence in health care and reducing premature mortality from lifestyle issues of excess consumption of alcohol, obesity, and viral hepatitis', *The Lancet* 384 (2014), p. 1953; Joseph F. Perz, et al., 'The contributions of hepatitis B virus and hepatitis C virus infections to cirrhosis and primary liver cancer worldwide', *Journal of Hepatology* 45.4 (2006), p. 529.

[8] Martin Blachier, Henri Leleu, Markus Peck-Radosavljevic, Dominique-Charles Valla, and Francoise Roudot-Thoraval, *The Burden of Liver Disease in Europe: A Review of Available Epidemiological Data* (Geneva, 2013) p. 5; Sonia Ratib, Joe West, and Kate M. Fleming, 'Liver cirrhosis in England – an observational study: are we measuring its burden occurrence correctly?', *BMJ Open* 7 (2017), pp. 1-7.

[9] Williams, et al., 'Addressing liver disease', p. 1956; Philippe Mathurin, and Ramon Bataller, 'Trends in the management and burden of alcoholic liver disease', *Journal of Hepatology* 62 (2015), p. S40; Gro Askgaard, et al.,

twentieth century as a reason to question the notion that cirrhosis could be explained just by the straightforward action of alcohol on the liver. Indeed, the reigning explanation since the 1970s has been that the drinker's susceptibility to the toxic action of alcohol on the liver is determined by the addition of individual genetic and environmental factors. Such complexities were at the heart of much of the historical debates that brought the presumed culpability of alcohol into question.

Alcohol is a multifaceted substance that has been tied to a wide variety of perceived problems. These include public disorder, economic costs, and excess mortality and morbidity of numerous mental and physical illnesses said to result from its excessive use. Concerning the mortality risks, the most recent World Health Organisation *Global Status Report on Alcohol and Health* (2014) states that alcohol is presently responsible for 5.9 per cent of all deaths worldwide, of which 74 per cent are specifically to do with diseases and not injuries incurred from alcohol-related violence, traffic accidents, and so on.[10] The report also recognises 200 different disease and injury conditions that alcohol is seen to be at least partly responsible for, such as alcohol poisoning, pneumonia, tuberculosis, diabetes, cardiovascular diseases, and various types of cancer. Outside of the harms that are categorically restricted to drinkers (alcohol use disorders, foetal alcohol spectrum disorder), gastrointestinal diseases like cirrhosis and pancreatitis are understood to have the strongest connection to heavy drinking.[11] Jürgen Rehm et al. estimates cirrhosis to be 'the most important single fatal chronic disease condition caused by alcohol consumption', being responsible for 15 per cent of all alcohol-related deaths worldwide.[12] Hence, the significance of cirrhosis among the numerous problems caused by drink cannot be understated.

'Alcohol drinking pattern and risk of alcoholic liver cirrhosis: A prospective cohort study', *Journal of Hepatology* 62 (2015), p. 1061.

[10] World Health Organization, *Global Status Report*, pp. 46-8.

[11] *Ibid.*, pp. 12, 46.

[12] Jürgen Rehm, Benjamin Taylor, Satya Mohapatra, Hyacinth Irving, Dolly Baliunas, Jayadeep Patra, and Michael Roerecke, 'Alcohol as a risk factor for liver cirrhosis: a systematic review and meta-analysis', *Drug and Alcohol Review* 29 (2010), p. 437.

The association between alcohol and liver disease possesses a long history that predates the twentieth century. Diseases of the liver, including cirrhosis, were already identified in Ancient Greece by Hippocrates and Erasistratus.[13] Galen, arguably the most influential doctor from classical antiquity, explained the pathogenesis (development) of cirrhosis through the lens of Hippocratic humoralism. He understood the hardening of the liver to be the indirect outcome of the consumption of 'heavy wine' causing the obstruction of blood flow between the intestine and the liver.[14] The knowledge on alcohol and liver damage was further articulated in the early modern period. During the sixteenth century, Jean Fernel and Andreas Vesalius noted the existence of a close association between heavy drinking and cirrhosis.[15] Later, Matthew Baillie devised the first official classification of cirrhosis as a disease entity in *The Morbid Anatomy of Some of the Most Important Parts of the Human Body* (1793), which stated that the pathogenesis of the disease 'would seem to depend upon the habit of drinking'.[16] Thomas Trotter and Benjamin Rush, early pioneers of the 'disease' concept of alcoholism, similarly recognised the liver as one of the organs that are negatively affected by the consumption of alcohol.[17] The word 'cirrhosis' was coined for the first time in the nineteenth century by René Laennec to denote the scarring of the liver.[18] Thus, the term 'Laennec's cirrhosis' was developed by Carl von Rokitansky in 1849 to refer to the specific variant of the disease believed to be caused by alcohol.[19] The notion that alcohol was directly responsible for cirrhosis of the liver became generally accepted across the rest of the nineteenth century, explaining why the

[13] Piero Amodio, 'Hepatic Encephalopathy: Historical Remarks', *Journal of Clinical and Experimental Hepatology* 5 (2015), p. S4.

[14] Jacques Jouanna, *Greek Medicine from Hippocrates to Galen: Selected Papers* (trans. Neil Allies, Leiden, 2012), p. 179; Thomas S. Chen, and Peter S. Chen, *Understanding the Liver: A History* (Westport, CN, 1984), pp. 123-4.

[15] John T. Galambos, 'Progress Report: Alcohol and Liver Disease', *New Series* 14.7 (1969), p. 477; Charles S. Lieber, 'The Metabolism of Alcohol', *Scientific American* 234.3 (1976), p. 25; Chen, and Chen, *Understanding the Liver*, p. 131.

[16] Matthew Baillie's *The Morbid Anatomy of Some of the Most Important Parts of the Human Body*, quoted in Roy Porter, 'The Drinking Man's Disease: The "Pre-History" of Alcoholism in Georgian Britain', *British Journal of Addiction* 80 (1985), p. 389.

[17] Chen, and Chen, *Understanding the Liver*, p. 132.

[18] William F. Bynum, 'Chronic alcoholism in the first half of the 19th century', *Bulletin of the History of Medicine* 42.2 (1968), p. 179; Ariel Roguin, 'Rene Theophile Hyacinthe Laennec (1781-1826): The Man Behind the Stethoscope', *Clinical Medicine & Research* 4.3 (2006), p. 234.

[19] Michael W. Mulholland, *Greenfield's Surgery: Scientific Principles and Practice* (Philadelphia, 2016), p. 946.

assumed causation of drink was deeply ingrained among most physicians in Britain before the First World War.[20] It is somewhat ironic, therefore, that while the connection between cirrhosis and alcohol had been observed since the dawn of Western medicine, it was during the flourishing of modern professional medicine in the twentieth century that this relationship was most effectively questioned.

Social Constructionism and the Epistemology of Disease Knowledge

In studying the history of disease knowledge and its impact on wider social developments, it is useful to outline some of the epistemological assumptions that form the groundwork of this thesis. Any historian of medicine would have to address the implications of the concept of *social constructionism*, an idea that has wielded tremendous influence in the humanities and the social sciences throughout the past five decades. Social constructionism challenges older positivist and empiricist assumptions by suggesting that knowledge is a construct rather than an objective representation of reality. Vivien Burr sets out four general presuppositions that underlie the approach: that assumed 'truths' in society should be critiqued; that knowledge is historically and culturally specific; that knowledge is a product of social processes; and that the knowledge itself has the agency to shape human behaviour.[21] Following a brief summary of the literature that constitutes this influential approach, the topic of this thesis will be conceptualised within a modified rendition of the social constructionist approach that effectively reconciles the emphasis on social and historical contingency with the recognition that rationality played a role in the formation of medical knowledge.

For historians of medicine, social constructionism can be summed up to three relevant strands of literature. First, constructionist approaches had a particular impact on historians in the form of what has commonly been referred to as 'postmodernism'. The influence of this intellectual approach on the profession began when Hayden White triggered a departure from the belief that historians were engaged in an objective uncovering of past events and ideas,

[20] Chen, and Chen, *Understanding the Liver*, pp. 131-2.
[21] Vivien Burr, *Social Constructionism* (2nd edn, Hove, 2003), pp. 2-5.

choosing instead to stress the literary and subjective nature of historical writing.[22] In the same year, Clifford Geertz pushed the discipline towards adopting an anthropological outlook that acknowledged the specificity of certain practices and beliefs across different cultures.[23] However, the individual who has most frequently been associated with postmodernism is Michel Foucault, whose theory of knowledge as a product of power relations had an enormous impact in reshaping the discipline.[24] Foucault was indirectly responsible for inspiring many works of cultural history, whereby a large segment of the historical profession became compelled to engage in a rigorous interrogation of the meanings and discourses found in primary sources.[25]

The understanding that knowledge is 'constructed' has also been salient particularly among sociologists of medicine. The term 'social constructionism' has its roots in the publication of *The Social Construction of Reality* (1966) by Peter L. Berger and Thomas Luckmann.[26] The theoretical outlook, when applied to medical knowledge, challenged the epistemologically privileged status of modern medicine and its alleged foundations in scientific objectivity. According to Peter Wright and Andrew Treacher, such assumptions neglected the agency of non-scientific social factors in the formation of medical knowledge.[27] One particular theory stemming from this school of thought that has been particularly thought provoking for historians was the medicalisation theory.[28] The idea, strongly associated with Foucault's work on the history of psychiatry, interprets the emergence of modern professional medicine as a

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[22] Hayden White, *Metahistory: The Historical Imagination in Nineteenth-Century Europe* (Baltimore, MA, 1973).
[23] Clifford Geertz, *The Interpretation of Cultures* (New York, 1973).
[24] Michel Foucault, *Madness and Civilization: A History of Insanity in the Age of Reason* (trans. Richard Howard, London, 1967); Michel Foucault, *The Order of Things: An Archaeology of the Human Sciences* (trans., London, 1970); Michel Foucault, *The Archaeology of Knowledge* (trans. A. M. Sheridan Smith, London, 1972); Michel Foucault, *The Birth of the Clinic: An Archaeology of Medical Perception* (trans. A. M. Sheridan Smith, London, 1973).
[25] Georg G. Iggers, *Historiography in the Twentieth Century: From Scientific Objectivity to the Postmodern Challenge* (Middletown, CT, 2005), pp. 118-33.
[26] Peter L. Berger, and Thomas Luckmann, *The Social Construction of Reality* (New York, 1966).
[27] Peter Wright, and Andrew Treacher, 'Introduction', in Peter Wright, and Andrew Treacher (eds.), *The Problem of Medical Knowledge: Examining the Social Construction of Medicine* (Edinburgh, 1982), pp. 3-5.
[28] Ludmilla Jordanova, 'The Social Construction of Medical Knowledge', *Social History of Medicine* 8.3 (1995), p. 367.

process whereby various aspects of life, such as depression and addiction, came under the control of medical experts.[29] The medicalisation theory has been most frequently applied to mental illnesses, many of which came to be seen as a by-product of the labelling of certain behaviours that digressed from the norm.[30]

Social constructionist assumptions were also adopted by a group of scholars who critiqued the assumed objectivity and rationality of modern science, a trend that originally stemmed from the philosophy of science following the publication of Thomas Kuhn's *The Structure of Scientific Revolutions* (1962).[31] Kuhn contended that scientific progress is guided, not by the rational accumulation and development of new facts and theories, but through 'paradigm shifts': periodic revolutions whereby fundamental assumptions in a particular scientific field or discipline are overturned and replaced by a new set of assumptions that are incommensurable with their predecessors. Kuhn's scepticism of the autonomy of scientific truth was furthered by a handful of important studies, such as Paul Feyerabend's opposition to the notion that science is guided by a universal method, the Edinburgh-based strong programme of the sociology of scientific knowledge, and Bruno Latour's anthropology of science.[32] This anti-positivist current inspired a number of interdisciplinary fields such as the history and philosophy of science (HPS) and science and technology studies (STS), both of which combines the conceptual analysis of science with the empirical study of past scientific ideas and practices. While scholars belonging to such fields have struggled to reconcile the philosopher's interest in the normative workings of science with the historian's indifference towards attempts to devise a universal explanation, ideas from philosophy have proven their use in providing a theoretical framework to interpret past scientific knowledge and practice.[33]

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[29] Sarah Nettleton, *The Sociology of Health and Illness* (2nd edn, Cambridge, 2006), p. 25; Colin Jones, and Roy Porter, 'Introduction', in Colin Jones, and Roy Porter (eds.), *Reassessing Foucault: Power, Medicine and the Body* (London, 1994), pp. 1-3.

[30] The most well-known example of this line of work is Peter Conrad's study on ADHD, in 'The Discovery of Hyperkinesis: Notes on the Medicalization of Deviant Behaviour', *Social Problems* 23.1 (1975), pp. 12-21.

[31] Thomas S. Kuhn, *The Structure of Scientific Revolutions* (Chicago, 1962).

[32] Kathryn Olesko, 'Historiography of Science', in J. L. Heilbron, James Bartholomew, Jim Bennett, Frederic L. Holmes, Rachel Laudan, and Giuliano Pancaldi (eds.), *The Oxford Companion to the History of Modern Science* (Oxford, 2003), pp. 366-70.

[33] Larry Laudan, 'The History of Science and the Philosophy of Science', in R. C. Olby, G. N. Cantor, J. R. R. Christie, and M. J. S. Hodge (eds.), *Companion to the History of Science* (London, 1990), pp. 47-59.

Admittedly, the term 'construct' has become one of the most commonly employed metaphors across the various disciplines in the humanities and the social sciences that study medicine. In spite of its frequent use, many scholars tend to take the word at face value without interrogating the potential implications that might be brought about by its connotations. For instance, the idea that a certain medical knowledge was 'constructed' may insinuate that the knowledge is somehow 'fabricated' by the scientist without any reference or relation to the material reality. In addition, while it is true that all attempts to understand a biological phenomenon is mediated by the individual outlook of the scientist as well as their social and cultural contexts, 'construction' inadequately accounts for the capacity of scientists to exercise their rationality in formulating descriptions and explanations of diseases. There exists, therefore, the need to devise a conceptual approach that strikes a middle ground between positivist empiricism on the one hand and social constructionism on the other in order to account for these shortfalls.[34]

Charles E. Rosenberg reflects on this problem by suggesting the notion of 'framing' as a more precise metaphor over 'construction' in approaching the history of medicine.[35] His book, *Framing Disease* (1997), is founded on the premise that the labelling of a specific pathological phenomenon as a 'disease' arises from the physician's practical imperative to comprehend its causes and symptoms to more effectively prevent, diagnose, and treat the illness.[36] The advantage of understanding diseases as being 'framed' rather than being 'constructed' is founded on how the metaphor accurately refers to the 'naming process' as a discursive conceptualisation of a specific pathological phenomenon rather than the ontological creation of the disease itself. The disease as a biological entity and the suffering derived from it are, indeed, very real and not necessarily 'constructed', although Rosenberg additionally sees this 'naming process' as also a social process involving the negotiation of competing interests that

[34] Richard Evans sought to do the same in trying to 'steer a middle course between the extremes of postmodernist hyper-relativism on the one hand, and traditional historicist empiricism on the other' in, *In Defence of History* (2nd edn, London, 2000), pp. 254-5.
[35] Charles E. Rosenberg, 'Introduction - Framing Disease: Illness, Society, and History', in Charles E. Rosenberg, and Janet Golden (eds.), *Framing Disease: Studies in Cultural History* (New Brunswick, NJ, 1997), pp. xiii-xxvi.
[36] *Ibid.*, pp. xv-vi.

seek to benefit from the parameters set by established definitions.[37] Robert Aronowitz takes the critique of the 'construction' metaphor further by labelling it a 'dated' manifestation of Foucault's reductionist understanding of modern medicine as nothing more than a means to control deviance.[38] Aronowitz adds that social constructionists have the habit of conveniently applying the notion to diseases that lack a clear somatic (physical) foundation, primarily that of psychological conditions.[39] While recognising that disease knowledge is founded on a 'result of negotiations among the different parties with a stake in the outcome', Rosenberg and Aronowitz stress the linguistic nature of the 'framing' process and acknowledge how scientific insight contributes to the formulation of medical knowledge. Beyond the scientific realm, however, disease knowledge becomes negotiated by a separate social process.[40]

'Alcoholic cirrhosis', along with its various historical incarnations such as 'hobnailed liver', 'gin drinker's liver', and 'Laennec's cirrhosis', is, itself, a linguistic framing of the manifestations of the condition that can be attributed to the causation of alcohol. Cirrhotic damage to the liver is a biologically real phenomenon that can be detected through blood tests, clinical examination, liver scans, and a microscopic examination of a liver biopsy. The decision to frame cirrhosis as an 'alcoholic' disease was founded on its clinical implications. Naming a condition based on its causal origin allows the clinician to target the aetiology (causation) to treat the disease by, in the case of alcoholic cirrhosis, encouraging the patient to withdraw from alcohol consumption upon diagnosis. Moreover, the recognition of the disease as among the most common pathological outcomes of drinking allows policymakers to argue the legitimacy of targeting alcohol consumption as a risk factor that warrants a preventative response. Thus, the understanding that 'alcoholic cirrhosis' was 'constructed' inadequately accounts for the practical advances derived from labelling a disease entity as an outcome of its assumed aetiology, alcohol. Although cirrhosis had always been understood as a disease that could be brought about by factors other than alcohol, the primary disagreement throughout the

[37] *Ibid.*, p. xxi.
[38] Robert Aronowitz, *Making Sense of Illness: Science, Society, and Disease* (Cambridge, 1998), p. 11
[39] *Ibid.*, p. 11.
[40] *Ibid.*, pp. 1, 11.

twentieth century was over the question of whether alcohol itself should even be regarded as a legitimate cause in the first place.

Additionally, the scientific debate over the causation of cirrhosis had much wider implications on the alcohol debate in Britain. According to Aronowitz, the 'meanings and connotations' of a particular framing produces 'winners and losers'.[41] During the interwar period, 'New Moderationist' alcohol researchers used the diminishing recognition of alcohol's role in the disease as a means to discredit the exaggeration of alcohol-related harm by the temperance movement. This process, in turn, allowed the New Moderationists to justify the promotion of moderate drinking at the expense of total abstinence as a means to reduce the incidence of drunkenness in society. Later in the 1970s, the experimental confirmation of alcohol as a direct toxin to the liver played a crucial role in allowing alcohol researchers and professional medical bodies to restart the discussion on the problem of alcohol misuse in British society. In both instances, the social and political context within a certain period had a greater impact on the *reception* of medical knowledge over that of the *creation* of knowledge itself.

The other advantage of Rosenberg's approach is its recognition of the possibility that scientists were engaging in rational exchanges when debating the nature of diseases. Social constructionist accounts often tend to undermine the historian's capacity to judge the relative merits of different theories in medicine.[42] In their seminal anthropological study of the scientific laboratory, Latour and Woolgar argue that scientific 'facts' are socially negotiated and constructed.[43] Although social factors admittedly factors into the formation of scientific knowledge, Latour and Woolgar neglect rationality as playing a role in the process. That scientific knowledge is borne out of its social settings as well as of the scientist's cognitive limitations does not necessarily imply that the knowledge itself is not a reasonably accurate description or explanation of the phenomena. As discussed before, the success of a certain

[41] *Ibid.*, p. 7.
[42] Nettleton, *The Sociology of Health and Illness*, p. 28.
[43] Bruno Latour, and Steve Woolgar, *Laboratory Life: The Construction of Scientific facts* (Beverly Hills, CA, 1979).

'framing' of a disease based on its purported aetiology can be dependent on its effectiveness in reaching a clinical goal. This echoes Larry Laudan's argument that rational progress in science can be assessed, not by its approximation to reality, but by its capacity to solve a given problem.[44] Therefore, to disregard rationality as a factor in the production of knowledge leads to a simplistic historical account in which scientific change is understood to be caused exclusively by factors that have no direct bearing on the science itself. Although the public dissemination of the knowledge admittedly was shaped by non-scientific factors, my thesis argues that, rather than being driven purely by social and political circumstances, scientists explained cirrhosis quite faithfully according to the methodologies and standards of proof current to the time.

Historiography

The historical formation of the medical knowledge of cirrhosis and its wider impact on the alcohol problem in modern Britain has direct implications to various historiographies. Starting with an explanation of how my thesis supplements previous accounts on the history of the changing understandings of alcoholic liver disease, this section moves onto the historiography of alcohol and politics in modern Britain and the wider study of alcohol and drugs across different periods and regions. The general theme that underlines the contributions of this thesis has to do with the importance of scientific evidence in precipitating change in the expert knowledge on liver disease and its relation to alcohol. Additionally, this thesis argues that scientifically informed understandings of cirrhosis aetiology, as part of the wider medical knowledge on physiological diseases that are tied to alcohol consumption, shaped how drink was conceptualised and approached within various debates over alcohol use in twentieth-century Britain.

The history of the science surrounding the causation of liver disease has attracted some interest from researchers working in health policy. The earliest historical account of the medical knowledge of the liver was written in 1984 by two American doctors, Thomas and

[44] Larry Laudan, *Progress and its Problems* (Berkeley, CA, 1977), pp. 11, 68

Peter S. Chen. It presents a teleological narrative that traces the changing understandings of numerous aspects of the liver from the time of Hippocrates.[45] Although the authors provide a reasonably accurate account of the shifting knowledge on cirrhosis aetiology, the book nevertheless reads more as a scientific literature review rather than a work of history. In addition to providing virtually no explanations on the numerous factors and outcomes that contributed to the changes, the book fails to account for the dissemination of expert knowledge outside of the realm of academic journals.

A much more analytical account of the history of liver disease was published a number of years later in the *British Journal of Addiction* by Denise Herd (1992).[46] The guiding thesis of Herd's paper states that the 'shifts in the medical and epidemiological paradigms of liver cirrhosis… were reflections of the changing social images of alcohol'. Focusing primarily on American society following the repeal of the prohibition in 1933, she explores how changing cultural attitudes towards alcohol consumption were 'mirrored' by the gradual de-emphasis on alcohol's toxicity to the liver in medicine and public health.[47] The 1940s saw the emergence of a new theory that attributed cirrhosis to malnutrition, while alcohol consumption was relegated to being nothing more than an indirect factor that ate up a large share of the drinker's caloric needs, thereby facilitating a deficiency in certain nutrients. Herd primarily attributes the dismissal of the alcoholic aetiology of cirrhosis to two primary factors: the diminishing influence of the American temperance movement in professional medicine owing to the perceived failure of prohibition, and the endorsement of the nutritional theory by a new generation of researchers led by E. M. Jellinek, who called for the medicalisation of alcoholism as a 'disease'. Although the article does not provide a precise account of how the 'shifts in medical and epidemiological paradigms' precisely interacted with the 'changing social images of alcohol', ambiguously describing the process as a 'reflection', it effectively infers that the

[45] Chen, and Chen, *Understanding the Liver.*
[46] Denise Herd, 'Ideology, history and changing models of liver cirrhosis epidemiology', *British Journal of Addiction* 87 (1992), pp. 1113-26.
[47] *Ibid.*, p. 1114.

abandonment of alcohol as a recognised aetiology of cirrhosis was precipitated by social, rather than scientific, developments.[48]

A year later, another article on the topic was published in the same journal by Brian S. Katcher (1993).[49] His paper, 'The post-repeal eclipse in knowledge about the harmful effects of alcohol', similarly states that post-prohibition American culture and the emergence of the disease concept of alcoholism triggered a reassessment of the causality between alcohol and cirrhosis, alongside other diseases such as cardiomyopathy, foetal alcohol syndrome, and oesophageal cancer. In contrast to Herd's article, Katcher attributes this change more to the period's predominant scientific paradigm on disease causation by arguing that, at the time, statistical associations between a presumed cause and a disease were deemed to be insufficient in demonstrating the existence of a causality.[50] Hence, the direct toxicity of alcohol was disputed by influential figures like Jellinek on the grounds that laboratory studies had largely failed to reproduce the disease in experimental animals.[51] However, in spite of his perceptivity to the contingent nature of the period's criteria for establishing disease causation, Katcher describes how alcohol researchers 'prematurely' abandoned alcohol as a recognised cause of cirrhosis.[52] He provides a noticeably progressivist narrative that marks a clear distinction between modern-day understandings and older explanations like the nutritional theory, just because the latter turned out to be, in his words, 'wrong'.[53]

In essence, both articles argue that the knowledge on cirrhosis was shaped to an extent by the liberalisation of cultural attitudes towards alcohol after the repeal of prohibition in 1933 and by the popularity of the disease concept of alcoholism. Herd and Katcher theoretically ground their arguments on Kuhn's 'paradigm shifts' by highlighting the impact of period-specific assumptions on disease causation, as well as the emergence of new disciplines such as

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[48] *Ibid.*, pp. 1116-7.
[49] Brian S. Katcher, 'The post-repeal eclipse in knowledge about the harmful effects of alcohol', *Addiction* 88 (1993), pp. 729-44.
[50] *Ibid.*, pp. 736-8.
[51] *Ibid.*, p. 736.
[52] *Ibid.*, pp. 731-2.
[53] Virginia Berridge, 'The relationship of science to policy: the need to look wider than alcohol', *Addiction* 89 (1994), pp. 534-5.

nutrition and psychology.[54] Although they are right to point to Jellinek as the intermediary who was responsible for endorsing a theory that downplayed the causation of alcohol, neither scholars pay much attention to the specific experimental and clinical studies that had contributed to the establishment of the nutritional theory. Furthermore, they attribute the demise of the theory and the re-establishment of alcohol as a recognised cause of cirrhosis in the 1970s to the statisticians who successfully demonstrated a strong association between the two.[55] This argument is inspired by the influential historiographical interpretation of lung cancer epidemiology by Allan Brandt and others, that the discovery of a statistical link between smoking and lung cancer by Richard Doll and Austin Bradford Hill in the 1950s legitimised epidemiology as a valid method of inferring disease causation.[56]

In regard to alcoholic liver disease, this thesis shows that scientists continued to believe throughout the century that evidence founded on animal experimentation was paramount to deciding if alcohol should or should not be deemed a cause of cirrhosis. While Katcher fails to recognise that this was still the case for cirrhosis even after Doll and Hill's studies, Herd provocatively implies that the medical scientists were somehow guided by the culture of post-prohibition America to hastily dismiss alcohol's culpability in various somatic diseases. Instead, I argue that these shifts were much more grounded in the science: the simultaneous existence and absence of certain kinds of evidence compelled specialists to reasonably, by the standards of scientific evidence of the time, abandon alcohol as the direct cause of liver damage on behalf of malnutrition, even though the theory itself was eventually falsified by the 1970s.

In contrast to Herd and Katcher's focus on the American experience, this thesis instead takes a look at the place of medicine in the drink question in Britain. The decision to focus exclusively on the United Kingdom is partly driven by the recognition that the public discourse

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[54] Herd, 'Ideology, history and changing models', p. 1122; Katcher, 'The post-repeal eclipse', p. 730.
[55] Herd, 'Ideology, history and changing models', p. 1119; Katcher, 'The post-repeal eclipse', p. 732.
[56] Allan Brandt, 'The Cigarette, Risk and American Culture', *Daedalus* 119 (1990), pp. 155-76; Allan Brandt, '"Just say No": Risk, Behavior, and Disease in Twentieth-Century America', in Ronald G. Walters (ed.), *Scientific Authority in Twentieth Century America* (Baltimore, 1997), pp. 82-98; John Burnham, 'American physicians and Tobacco Use: Two Surgeons General, 1929 and 1964', *Bulletin of the History of Medicine* 63 (1989), pp. 1-31; Colin Talley, Howard I. Kushner, and Claire E. Sterk, 'Lung Cancer, Chronic Disease Epidemiology, and medicine, 1948-64', *Journal of the History of Medicine and Allied Sciences* 59.3 (2004), pp. 334-5.

surrounding any substance is inevitably shaped by specific national circumstances. As Mark Schrad has noted, regulatory responses to the alcohol problem in the early twentieth century varied widely across the Western world due to the institutional contexts of each country.[57] Harry Levine groups Britain as among the nine 'temperance cultures' alongside Finland, Sweden, Norway, Iceland, Australia, New Zealand, Canada, and the United States: countries that had 'large, enduring temperance movements' that 'extended far beyond formal membership and achieved widespread acceptance and legitimacy in the larger society'.[58] In the early twentieth century, some of these countries, like the United States and Canada, chose to implement a nationwide prohibition on the sale of alcohol, while others, like Britain and Sweden, resorted to the partial nationalisation of the liquor industry. Even so, Britain, alongside much of Scandinavia, continues to be governed under a relatively restrictive control regime whereby alcohol is problematised and regulated to a greater extent than in other parts of Western Europe.[59] Even beyond the movement's decline in the twentieth century, the temperance campaigns had a lasting impact on the language and policy surrounding the alcohol problem in Britain.[60] Although more recent accounts by Paul Jennings and Thora Hands have justifiably sought to distance the historiography from the problematising discourses by choosing to highlight the 'ordinary' aspects of drinking, one could hardly deny that the temperance movement played a big part in shaping how alcohol was conceptualised within the wider public discourse.[61] This, in turn, influenced how the medical knowledge on much of the 'alcoholic' diseases was adopted within the alcohol debate.

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[57] Mark Schrad, *The Political Power of Bad Ideas: Networks, Institutions, and the Global Prohibition Wave* (Oxford, 2010).

[58] Harry G. Levine, 'Temperance Cultures: Concern about Alcohol Problems in Nordic and English-speaking Cultures', in Malcolm Lader, Griffith Edwards, and D. Colin Drummond (eds.), *The Nature of Alcohol and Drug-Related Problems* (New York, 1993), pp. 16-7.

[59] Michael Savic, Robin Room, Janette Mugavin, Amy Pennay, and Michael Livingston, 'Defining "drinking culture": A critical review of its meaning and connotation in social research on alcohol problems', *Drugs: Education, Prevention and Policy* 23.4 (2016), pp. 272-3.

[60] Henry Yeomans, *Alcohol and Moral Regulation: Public Attitudes, Spirited Measures and Victorian Hangovers* (Bristol, 2014), p. 10.

[61] Paul Jennings, *A History of Drink and the English, 1500-2000* (Abingdon, 2016), pp. 211-2; Thora Hands, *Drinking in Victorian and Edwardian Britain: Beyond the Spectre of the Drunkard* (Basingstoke, 2018), pp. 1-10.

Unlike the long-established historical literature surrounding the Victorian temperance movement, the alcohol problem in the twentieth century has only been thoroughly studied by historians in the past two decades or so.[62] Scholarly interest in the period has been a by-product of the resurgence of public concern surrounding alcohol use and harm in the late twentieth century. Thus, the earliest accounts authored by Rob Baggott (1990) and Betsy Thom (1999) proved to be particularly relevant for researchers in health policy. Baggott and Thom explore how the alcohol debate in postwar Britain was shaped by the ideas, interests, and values of the medical professional bodies, campaign groups, and policymakers that had a stake in the problem.[63] Both scholars articulate a particularly influential view that the conceptualisation of the alcohol problem shifted throughout the century from the 'moral' model promoted by the temperance movement towards a 'medical' model whereby alcohol use was increasingly approached through evidence-based knowledge derived from expert research in health and medicine.[64] The period was later approached by John Greenaway (2003) under the lens of Westminster and Whitehall politics, highlighting the 'elasticity' of the drink question in adjusting to the political and social contexts of any particular period.[65] A more multifaceted account of alcohol history in England was produced by James Nicholls (2009) in the form of a narrative that spans from the English Reformation to the present day.[66] Nicholls combines the strengths of many of the previous accounts by arguing that the historical debates on alcohol were seldom to do with alcohol itself, but a reflection of other political, economic, social, and intellectual developments.[67] Adding to the previous work, the most recent contribution was authored by Henry Yeomans (2014).[68] Yeomans reassesses the history of the alcohol problem

[62] Brian Harrison, *Drink and the Victorians: The Temperance Question in England 1815–1872* (London, 1971); A. E. Dingle, *The Campaign for Prohibition in Victorian England: The United Kingdom Alliance 1872–1895* (London, 1980); Lilian Lewis Shiman, *Crusade Against Drink in Victorian England* (London, 1988).
[63] Rob Baggott, *Alcohol, Politics and Social Policy* (Aldershot, 1990); Betsy Thom, *Dealing with Drink: Alcohol and Social Policy from Treatment to Management* (London, 1999).
[64] Baggott, *Alcohol, Politics and Social Policy*, p. 13; Thom, *Dealing with Drink*, p. 15.
[65] Greenaway, *Drink and British Politics*, p. 2.
[66] James Nicholls, *The Politics of Alcohol: A History of the Drink Question in England* (Manchester, 2009).
[67] *Ibid.*, p. 3.
[68] Yeomans, *Alcohol and Moral Regulation.*

in modern Britain through the idea of moral regulation, revealing a set of discourses that were neglected by older accounts.[69]

Perhaps to a greater extent than many of the older narratives, Yeomans presents a much more critical account of the various groups that saw alcohol as a problem. Yeomans challenges Baggott and Thom in arguing that the discourses surrounding alcohol use continue to be tinged with moral prejudices well into the present era.[70] This line of interpretation echoes the popular characterisation of the modern public health campaign as the 'new temperance movement' or the 'neo-temperance alliance', portraying the resurgent concerns over the rising incidence of alcohol misuse as part of an ideological legacy of the Victorian crusade against drunkenness.[71] While Yeomans does not entirely disregard the 'scientific' basis of many of the modern approaches, he somewhat downplays the extent to which they were founded on a set of innovative ideas, especially that of the identification of a statistical connection between overall levels of alcohol consumption and incidence of alcohol-related harm. Other scholars such as Nicholls and Jennings present a more balanced interpretation that recognises the discursive similarities between the temperance movement and the public health model while simultaneously highlighting the glaring differences between the two, such as on how the latter targets the outcomes of drinking rather than drinking itself.[72] Although social constructionist outlooks are unquestionably relevant to the critique of various 'scientific' approaches to health, they should not muddle the acknowledgement of rationality and innovation that grounded such approaches. Therefore, the argument of this thesis leans more towards Thom's account by showing that the scientific evidence on alcohol's harms on the body played a vital role in shaping many of the important ideas that contributed to the alcohol debate.[73]

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[69] Yeomans dedicates a section of a chapter on the previously understudied teetotal pledge movement during the First World War, in *Ibid.*, pp. 108-11.
[70] *Ibid.*, pp. 203-33;
[71] Robin Room, 'Alcohol Control and Public Health', *Annual Review of Public Health*, 5.1 (1984), p. 295; Greenaway, *Drink and British Politics*, pp. 178, 182.
[72] Nicholls, *The Politics of Alcohol*, pp. 206-7; Jennings, *A History of Drink and the English*, pp. 169-70.
[73] Thom, *Dealing with Drink*, pp. ix-x.

The question on the place of expert knowledge in the drink question constitutes a larger historiographical debate on the interaction between scientific knowledge and the social reputation of a wider cohort of psychoactive substances, or 'intoxicants' as referred to by Phil Withington.[74] Thus far, historians have generally argued that social attitudes and regulatory responses towards such substances have less to do with the objective harms brought about by their consumption and more with the subjective meanings that societies attach to them. According to the late addiction psychiatrist Griffith Edwards (2004), '[t]he physical reality of these drugs is manifest, but the symbolism that attaches is also a potent and sometimes toxic reality which is likely to colour the policy choices.'[75] Virginia Berridge reiterates this view in *Demons: Our Changing Attitudes to Alcohol, Tobacco & Drugs* (2013) in stating that the social and political responses to intoxication are seldom to do with the intrinsic properties of the intoxicant. A reputation of a certain drug is a product of the complex web of interaction between institutional, economic, ideological, and professional interests.[76] An apt example of this interpretation can be found in her seminal account of opium in Victorian society, in which Berridge (1981) argues that, rather than being founded solely on scientific objectivity, the new 'disease' theory of opium addiction that emerged in the late nineteenth century reflected class and moral prejudices carried by professional physicians.[77] Matthew Hilton (2000) similarly discusses how much of the early efforts to devise a public health response to the link between tobacco and lung cancer was sabotaged by the cultural legitimacy attached to the intoxicant, owing to the positive association between smoking and *bourgeois* individualism in postwar Britain.[78] A more favourable aspect of intoxication was highlighted by Erika Dyck (2012) in her work on LSD research in Saskatchewan during the middle of the twentieth century. In spite of its enormous therapeutic potential in treating conditions such as alcoholism and PTSD, studies

[74] Phil Withington, 'Starting the Conversation', in Jonathan Herring, Ciaran Regan, Darin Weinberg, and Phil Withington (eds.), *Intoxication and Society: Problematic Pleasures of Drugs and Alcohol* (Basingstoke, 2013), p. 2; Phil Withington, 'Introduction: Cultures of Intoxication', *Past & Present* 222.9 (2014), p. 12.
[75] Griffith Edwards, *Matters of Substance: Drugs, and Why Everyone's a User* (New York, 2004), p. xxxvii.
[76] Virginia Berridge, *Demons: Our Changing Attitudes to Alcohol, Tobacco, & Drugs* (Oxford, 2013), p. 5.
[77] Virginia Berridge, and Griffith Edwards, *Opium and the People: Opiate Use in Nineteenth-century England* (London, 1981), pp. 150-70.
[78] Matthew Hilton, *Smoking in British Popular Culture 1800-2000* (Manchester, 2000), pp. 2-3.

into the clinical use of LSD were outlawed because of the ensuing panic surrounding its non-medical, recreational use after the 1960s.[79] The tendency among many of these accounts has been to causally disassociate the social problematisation of a particular intoxicant with the scientific knowledge on its properties. Alcohol clearly possesses a reputation that differs from most illicit substances, some of which, including cannabis, continue to be prohibited in much of the world even though the evidence points to their relative lack of harm. Alcohol's status as a legal, ubiquitous intoxicant in British society meant that more affirmative or forgiving attitudes towards the substance were often balanced out by those that condemned it, each of which were heavily informed by the knowledge surrounding its purported benefits and harms to health. My exploration of the impact of liver disease research on the social problematisation of drink illustrates how both the emphasis and de-emphasis of alcohol's culpability in cirrhosis causation directly contributed to the respective exaggeration and minimisation of the harmful properties of the beverage.

In essence, this thesis takes a cue from James Kneale's and Shaun French's contention that medicine functions as a 'key site for the production of ideas about drink and drinkers'.[80] In many of the historical accounts on alcohol in modern Britain, the interest in the wider significance of alcohol dependence ('dipsomania', 'inebriety', 'alcoholism', 'alcohol use disorders', and so on) tended to cloud the importance of somatic diseases such as cirrhosis.[81] Rightfully so, addiction has been recognised by scholars such as Mariana Valverde to be one of the central historical themes in the politics of alcohol.[82] However, diseases of the body likewise attracted the interest of stakeholders in the alcohol debate at every stage of the century as a noteworthy signifier of the dangers brought about by habitual drinking.[83] Among the assortment of harms known to be caused by alcohol, cirrhosis was recognised as one of the

[79] Erika Dyck, *Psychedelic Psychiatry: LSD on the Canadian Prairies* (Winnipeg, 2012).
[80] James Kneale, and Shaun French, '"The Relations of Inebriety to Insurance": Geographies of Medicine, Insurance and Alcohol in Britain, 1840-1911', in Jonathan Herring, Ciaran Regan, Darin Weinberg, and Phil Withington (eds.), *Intoxication and Society: Problematic Pleasures of Drugs and Alcohol* (Basingstoke, 2013), p. 88.
[81] For instance, two entire chapters are dedicated on the medical knowledge surrounding alcohol dependence in Nicholls, *The Politics of Alcohol*, pp. 59-72, 161-79.
[82] Mariana Valverde, *Diseases of the Will: Alcohol and the Dilemmas of Freedom* (Cambridge, 1998), pp. 1-2.
[83] Thom, *Dealing with Drink*, pp. 5-7.

more pernicious outcomes of alcohol misuse due to its strong association to drink and its poor prognosis. Hence, this thesis shows that the expert knowledge on whether alcohol should be deemed to be responsible for such a deadly disease inevitably shaped how drink itself was understood within the wider discussion on alcohol use in Britain.

Methodology and Sources

This thesis examines the production, dissemination, and appropriation of medical knowledge in the twentieth century. In so doing, it uses a wide range of materials that reveal the knowledge produced by or distributed to different sections of society. After outlining some of the methodological assumptions that guide the thesis, this section describes the uses and implications of a variety of texts and images that account for the formulation of medical knowledge found in scientific journal articles, their dissemination within the wider professional medical community, and their impact on the alcohol debate. My study is primarily motivated by the examination of the developments surrounding a specific question, namely 'does alcohol cause cirrhosis?' In addition to providing a set of explanations on the various scientific attempts to answer this question, I explore how many of these answers were transmitted to the wider public discussions in order to bring medicine back into the history of drink question in modern Britain.

This thesis is driven by a methodology that takes into account Ludmilla Jordanova's suggestion that social historians of medicine should make use of the systematic and conceptual rigour of the history of science in examining the theoretical *content* of medical knowledge.[84] This proposition encourages the historian to pass some sort of judgement on past scientific knowledge and practices, a fulfilment of what Paul Forman understood as the historian's 'obligation to decide for ourselves what is the good of science, and by our historical research and writing to advent that good'.[85] The crucial recognition that not all knowledge is equally

[84] Jordanova, 'The Social Construction of Medical Knowledge', p. 374.
[85] Quoted by Hasok Chang, 'Practicing Eighteenth-Century Science Today', in M. Biagioli, and J. Riskin (eds.), *Nature Engaged: Palgrave Studies in Cultural and Intellectual History* (New York, 2012), p. 104.

valid allows for the assessment of the relative merits of one knowledge over another.[86] Therefore, in addition to looking at the wider impact of a piece of scientific knowledge within 'non-scientific' contexts, an examination of the theoretical content of the knowledge is paramount to understanding the ideas and practices of a particular epistemic community. It must, however, be stressed that the historian could plausibly assess the merits of past knowledge while simultaneously accounting for their contexts and conditions that are contingent to a specific period of history. More often than not, physicians who held sympathies towards the temperance movement from the first half of the twentieth century exaggerated the harmful qualities of alcohol by providing a somewhat simplistic, dishonest account of its role in damaging the liver. Such views contradicted the knowledge produced by non-temperance medical professionals who highlighted the nuances and complexities surrounding alcohol's causation in cirrhosis. Thus, acknowledging the relative poverty of the knowledge promoted by temperance doctors allows for the recognition that such accounts were products of their ideological and moral prejudices towards alcohol.

Based on this outlook, my thesis engages in the content and discourse analysis of the sources that reveal the knowledge of various epistemic communities in society. Recent scholars such as Stig Brorson and Hanne Andersen (2001) have pointed to the usefulness of the categorisation of the complex strata of scientific knowledge originally outlined by Ludwik Fleck in his 1935 study on the history of syphilis.[87] Fleck's typology contains four separate, but interdependent, sources of knowledge in modern science: 'journal science', 'handbook science', 'textbook science', and 'popular science'.[88] Concerning this thesis, the first three categories belongs to the wider cohort of ideas on cirrhosis aetiology that were shared among medical professionals, while 'popular science' directly refers to the wider public discourses on alcohol

[86] George Steinmetz, 'Critical Realism and Historical Sociology. A Review Article', *Comparative Studies in Society and History* 40.1 (1998), p. 171

[87] Ludwik Fleck, *Genesis and Development of a Scientific Fact* (trans. Frederick Bradley, and Thaddeus J. Trenn, Chicago, 1979); Stig Brorson, and Hanne Andersen, 'Stabilizing and Changing Phenomenal Worlds: Ludwik Fleck and Thomas Kuhn on Scientific Literature', *Journal for General Philosophy of Science/Zeitschrift für allgemeine* 32.1 (2001), p. 115.

[88] Dimitri Ginev, 'The Transcendental in Ludwik Fleck's Social Epistemology', *Social Epistemology* 29.4 (2015), p. 384.

use in Britain. Fleck understood 'journal science' to encompass the content of the research carried out by specialists within a scientific discipline, which, as the name suggests, can be found in academic journals. The knowledge created in 'journal science' is then transmitted to scientific handbooks and textbooks, a phase when ideas that were previously shared among the exclusive group of specialists is established as a 'scientific fact' within the wider discipline. 'Popular science', then, refers to the stage where the knowledge manifests itself within various social and political contexts outside of the realm of professional medicine.[89] As for the specific case of this thesis, it describes how the expert knowledge on alcohol and the liver was conveniently remoulded and filtered through to serve a set of narratives promoted by groups involved in the alcohol debate. Therefore, Fleck's typology gives a suitable account of the complex process involving the formation of expert medical knowledge in scientific journals, its dissemination among medical professionals through medical textbooks and general medical journals, and its eventual appropriation within the public discourse.

In modern Western societies, academic journals function as the point of origin for much of the knowledge shared among medical professionals. Hence, 'Journal science' specifically concerns the production of new scientific knowledge in the research performed by medical experts who belong to a closed group of specialists, or what Fleck referred to as an 'esoteric circle' of scientists.[90] Although academic journals are accessible to any individual or group subscribed to them, such publications are generally consumed by other professionals belonging to the same field or discipline. To explore the genealogies of how a certain idea was formulated before it had an impact outside of the 'esoteric circle', this thesis provides a detailed account of the content of such publications.

The journal articles that are relevant to this thesis could be categorised to three types: laboratory experiments, clinical studies, and epidemiological studies. Laboratory experiments involve the use of non-human animal subjects, allowing for the study of cause and effect between different variables within a carefully controlled environment. Scientists investigating the causation of cirrhosis in the twentieth century tended to use rodents, canines, or primates

[89] *Ibid.*, pp. 384-5.
[90] Fleck, *Genesis and Development of Scientific Fact*, p. 118.

in attempting to reproduce cirrhotic damage with the suspected cause. Clinical studies, on the other hand, differed from laboratory experiments in that they depend on human participants to understand the biomedical phenomena. Although the outcomes of such studies carry the advantage of being directly applicable to human biology, the use of human subjects imposes higher ethical constraints. Thus, much of the clinical research examined in this thesis were either observational studies of patients who had already developed cirrhosis or attempts to produce the earliest and non-fatal stage of alcoholic liver disease, fatty liver. On a much larger scale, the distribution of certain diseases within a defined population is the subject of an epidemiological study. Such methods were particularly useful in investigating how the incidence of cirrhosis was affected by numerous societal variables such as the overall *per capita* level of alcohol consumption, the availability of alcoholic beverages, occupational factors, and so on.

One subcategory of journal articles that functioned somewhat differently from the above three would be the scientific review. As perhaps the most useful source of scientific evidence, review articles seek to answer specific questions by drawing broad conclusions that consider other works of scientific research.[91] Because of this, scientific reviews tend to have a larger impact in the discipline compared to clinical, experimental, or epidemiological research articles, often by transmitting the knowledge produced by other researchers towards more widely available texts such as medical textbooks. In this thesis, review articles are used not only to determine the content of a scientific consensus that would have existed among specialists of the liver but to also assess the reception of an important research article.

It should be noted that 'journal science' features much more prominently in chapters 3 and 4, roughly pointing to the period after the 1930s, over chapters 1 and 2. The emergence of the nutritional theory in mid-century and its eventual demise in the 1970s constitutes the 'climax' of this thesis due to the peculiar state of how alcohol ceased to be acknowledged as a direct cause of cirrhosis. Additionally, the identification of specific articles from before the 1940s that had a wide impact in the field proved a challenge for a number of reasons. The

[91] Cynthia D. Murlow, 'The Medical Review Article: State of the Science', *Annals of Internal Medicine* 106.3 (1987), pp. 485-8.

journal articles found mostly in the last two chapters were selected from the bibliographies of numerous review articles and specialist textbooks on the liver and the digestive system. In contrast to the second half of the twentieth century, understandings of cirrhosis aetiology appeared to be much more heterogeneous in the first half, as references found in review articles were often inconsistent with other similar publications, while medical textbooks seldom contained references to journal articles to support their particular claims. Therefore, the professional knowledge on alcohol and the liver in the first two chapters are mostly generalised from textbooks of general medicine and general medical journals.

Hierarchically situated below 'journal science', two types of medical texts functioned to transmit the scientific knowledge to the wider medical profession in Britain. The first was the textbook of general medicine, considered by historians of medicine to be an exceptionally useful source to determine the content of the knowledge that was widely disseminated to medical practitioners and students. Fleck identified both 'handbook' and 'textbook science' as a stage whereby knowledge previously restricted to specialists became 'part of the established corpus of knowledge' within a discipline.[92] Due to their intended audiences, textbooks can also be taken to contain the most 'correct' and 'up-to-date' knowledge on a particular topic. However, these ideals were not always met. Because they merely functioned to compile the most recent developments in medicine, textbooks were, by their nature, already outdated by the time they were published.[93] In addition, the precise explanations of the causes of cirrhosis were not always identical. At specific points in the century, several textbooks expressed views on the topic that radically diverged or contradicted with one another. Hence, this thesis assesses the degree of uniformity in opinion at a certain period to determine whether a specific stance on cirrhosis aetiology, whether it be those that had attributed the disease to alcohol or to malnutrition, was truly dominant or not. Additionally, the thesis systematically traces the shifting understandings on the causation of cirrhosis across multiple editions of a single series of textbook to demonstrate change over time. In order to restrict the sample size, only a single edition of a particular series of textbooks is studied at an interval of every five years. Multiple

[92] Brorson, and Andersen, 'Stabilizing and Changing Phenomenal Worlds', pp. 116.
[93] *Ibid.*, p. 116.

editions of a total of 13 textbooks, spanning from the 4th edition of James M. Anders's *Text-book of the Practice of Medicine* (1900) to the 2nd edition of the *Oxford Textbook of Medicine* (1987), are explored throughout this thesis (see Appendix: Textbooks of General Medicine).[94] The popularity and usage of a particular textbook in the British medical profession is further demonstrated through the book reviews of each series of textbooks, referenced whenever the volume is referred to for the first time.

Functionally similar to medical textbooks, general medical journals also played a crucial role in disseminating specialist knowledge to the wider medical profession. *The Lancet* and *The British Medical Journal* served as the unofficial 'newspapers' in the milieu of physicians and medical researchers in Britain, having a large readership of medical practitioners, researchers, and patients.[95] Aside from original research, both journals contained medical news, editorials, columns, letters from readers, transcripts of lectures, and accounts of important gatherings and meetings in the world of medicine. One aspect that distinguished the journals from textbooks was that they were not necessarily relied on to provide the most 'accurate' knowledge on a particular subject. Understandably, the journals, depending on the author and subject of the article, gave a platform to various opinion on the aetiology of cirrhosis that did not conform to the status quo. Therefore, rather than revealing the 'facts' within a given period, their usefulness as a historical source is founded on their wide readership, providing space for debates between divergent understandings on a given topic.

Beyond the realm of professional medicine, 'popular science' strictly points to the collection of texts and images that were produced as part of the wider public discussion on alcohol use in Britain. Most of these sources were tied to specific groups in society that participated in the alcohol debate, including temperance groups, the alcoholic beverage industry, public health campaigners, and policymakers. Described in detail in chapter 1, the temperance movement was a decentralised campaign composed of numerous groups and

[94] James M. Anders, *A Text-book of the Practice of Medicine* (4th edn, London, 1900); D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrell (eds.), *Oxford Textbook of Medicine* (2nd edn, Oxford, 1987).
[95] Drummond Rennie, 'The Present State of Medical Journals', *The Lancet* 352.S18-22 (October 1998), pp. 18-22; Vikki Entwistle, 'Reporting Research in Medical Journals and Newspapers', *BMJ* 310.920 (April 1995), pp. 920-3.

individuals that were united in their opposition to the prevalence of drink in society. The movement produced material that cautioned against the harms brought about by alcohol. These included educational handbooks and pamphlets, medical monographs, and school curriculums. Within such texts, cirrhosis was described in a way to promote the movement's agenda, often by overstating of the culpability of alcohol in causing the disease.

The primary antagonist of the temperance movement was the alcoholic beverage industry. Although the contribution of the distillers is touched on at certain points in this thesis, the vast majority of the sources relating to the liquor trade were specifically derived from the brewing industry, partly because of their prominent role in the alcohol debate as well as the fact that beer consistently accounted for most of the alcohol consumed in Britain. In the early half of the century, drinks advertisement revealed the various discourses on the supposed dietary and medicinal benefits of alcohol that were promoted by the industry as a counter-narrative to the temperance condemnation of drink. The Brewers' Society, the leading trade association of the brewing industry, played an integral role in co-opting numerous public campaigns to defend their commercial interests against calls to strengthen restrictions on the sale of alcohol. The materials that were used in these campaigns can be found in several industrial archives across England. In chapter 4, the source base shifts towards internal sources within the board of the Society, many of which illustrated how the industry intervened in the medical profession to wage a war of ideas against public health campaigners in the late twentieth century. Although liver disease was not directly mentioned or addressed by industrial sources until the late twentieth century, depictions of drink as a dietary beverage in many of the adverts serve to contextualise the wider debate on alcohol and health in society.

The final category of sources belongs to the official policy debates on alcohol. Many of these texts were directly attributable to discussions that had taken place in Westminster and Whitehall, a collection that encompasses the *Hansards*, government white papers, and official reports. While such sources embodied the discourses exchanged in the Houses of Parliament, the government, and various branches of the civil service, there were other similar reports and monographs produced by professional medical bodies that were connected to New Moderationism in the interwar period and the public health model in the 1970s. Official reports

published by groups such as the Royal College of Physicians provided legitimacy to new medical knowledge upon their dissemination to the public, handing them the power to directly influence policy-making.[96]

Summary

In approaching the topic from the angle of the history of medicine, this thesis puts forward two broad arguments. First, evidence consistently underpinned the formation of expert medical knowledge on cirrhosis aetiology. The assumed causation of alcohol was challenged in the middle of the twentieth century based on two primary objections that were founded on a set of period-specific assumptions as to what constituted 'acceptable' evidence in demonstrating disease aetiology. While cirrhosis was believed by many scientists to be a direct outcome of alcohol misuse, the commonly held understanding that the disease developed in no more than a minority of heavy drinkers invalidated the notion that there existed a straightforward, linear causality. Moreover, medical professionals disputed whether alcohol should even be regarded a true toxin to the liver on the grounds that most laboratory studies had failed to experimentally reproduce alcoholic cirrhosis in animal subjects. The abandonment of the alcoholic aetiology on behalf of the nutritional theory was founded on such objections, indicating that the widespread acceptance of malnutrition as the primary cause of cirrhosis was not necessarily a case of 'bad' science but a sensible conclusion that conformed to an existing standard of scientific evidence. Scientists were thus seemingly able to exercise their rationality in devising a coherent aetiological explanation based on a rigorous assessment of the available information. Although the public dissemination of the knowledge of liver disease was undoubtedly shaped by the wider social context of the drink question, the formulation of the knowledge itself was manifestly the product of historically and socially contingent, but nonetheless reasonable, observations by medical professionals.

[96] Virginia Berridge, 'Passive smoking and its prehistory in Britain: policy speaks to science?', *Social Science & Medicine* 49 (1999), pp. 1184-6.

Second, expert medical understandings of alcohol and the liver had a direct bearing on how harm was conceived in relation to drink in British society. For most of the twentieth century, cirrhosis was discursively framed as the quintessential outcome of the long-term abuse of alcohol, an 'alcoholic's disease', much like the immediate association between smoking and lung cancer. As part of a campaign to discourage alcohol misuse, this framing was used by different groups to highlight the damage that alcohol was capable of inflicting on the drinker. Inversely, the scientific minimisation of alcohol's culpability in the disease contributed to the tendency to downplay the harmful effects of alcohol on the body. Thus, the varying degrees to which alcohol was problematised at separate points of the century was shaped by the medical understanding of alcohol's association to physiological illnesses, among which cirrhosis was routinely regarded as being the most significant.

The decision to structure this thesis as a chronological narrative reflects the need to present an accessible account that traces both the complex shifts in the medical knowledge on cirrhosis aetiology and its impact on the concurrent public debates on alcohol in British society. Chapter 1 looks at the very beginning of the twentieth century when alcohol was acknowledged by most medical observers to be a direct cause of cirrhosis, an understanding that will be referred to as the *direct toxicity theory* (DTT; all acronyms that appear henceforth pointing to a particular theory of cirrhosis aetiology were created for the purpose of this thesis). Considered within the context of the fierce political disagreement over licensing reform in Edwardian society, the chapter discusses how the DTT was used by the temperance movement to highlight the destructive properties of drink on the body. Chapter 2 moves onto the interwar period and examines how the toxic action of alcohol on the liver was progressively played down in the medical literature. Although there was a lack of a clear consensus over the precise action of alcohol on the liver, with some even suggesting that the causation was indirect, most observers began to sense that the relationship between the two was markedly more complex than it had been previously assumed under the DTT. The minimised disease culpability of alcohol contributed to the emergence of New Moderationism, an innovative approach to alcohol and health derived from the First World War that challenged the temperance movement's insistence on total abstinence by suggesting the promotion of moderation as the optimal

solution to the problem of drunkenness. As a continuation of the developments seen in the interwar period, chapter 3 traces the consolidation of the fragmented understandings of cirrhosis aetiology under the establishment of the *nutritional deficiency theory* (NDT) after the Second World War. A study of scientific journals illustrates the elaborate process whereby cirrhosis was framed as a disease borne out of the deficiency of key nutrients. The adoption of the NDT led to the total dismissal of the DTT, a development that coincided with the diminished problematisation of drink in postwar Britain. The final chapter examines the original research that facilitated the demise of the NDT during the 1960s and 70s. Upon the re-establishment of alcohol as the primary cause of cirrhosis, further studies into the individual and environmental factors that contribute to its pathogenesis led to the formulation of the *modern direct toxicity theory* (MDTT). Such developments were at the heart of the resurgent concerns over the growing incidence of problem drinking in the late twentieth century, when the recognition of a direct causality between alcohol and cirrhosis emerged as an integral component of the public health approach to consumption and harm. Hence, the history of the politics of alcohol is as much to do with medicine as it is with society and culture.

Chapter 1

Alcohol and the Liver in Edwardian Britain

During the Edwardian period (1900-1914), cirrhosis, commonly referred to at the time as the 'gin-drinker's' or 'hobnailed' liver, was situated in the alcohol debate as a routinely recognised outcome of heavy drinking. An exploration of some of the most influential medical texts from the period shows that liver disease was primarily framed as an outcome of the action of alcohol on the organ. For the purpose of this chapter, this loosely conceptualised understanding of cirrhosis is referred to as the direct toxicity theory (DTT).

Since the middle of the nineteenth century, the drink question was among the key issues that dominated national politics. The campaign against the ubiquitous phenomenon of drunkenness in British society was led by the temperance movement. Within a distinctly divisive climate around the politics of alcohol, liver disease was frequently flagged up by physicians who were sympathetic to the temperance cause to overstate the hazardous effects of alcohol on the body. Temperance medical texts often presented a deliberately simplified understanding of alcohol's relationship to cirrhosis that neglected many of the intricacies surrounding the aetiology. Some medical professionals, however, were reluctant to take the DTT at face value by highlighting such intricacies, an even smaller minority suggesting that alcohol played nothing more than an indirect role in enabling some other factor in damaging the liver. Such claims were based on a handful of shared ideas, one of which understood that cirrhosis, the most serious stage of liver disease, only ever seemed to develop in no more than a minority of heavy drinkers. Although the DTT was evidently acknowledged in much of the established medical literature, the precise degree to which one attributed liver disease to alcohol varied depending on the text, making it difficult to homogenise the period's predominant

medical understandings. Thus, the broad agreement over alcohol as a primary cause of cirrhosis was balanced by a variety of other perspectives that questioned it.

This chapter provides the entire thesis with a sense of change in the medical understandings of alcohol and the liver across the twentieth century, at the beginning of which alcohol was presumed by most to be directly responsible for cirrhosis. The first section explores the intellectual context of the Edwardian drink question. The polarisation between the liquor trade and the temperance movement can be appreciated in their distinctive discursive conceptualisations of alcohol itself, the former of which understood it to be a 'food' while the latter framed it an 'evil' and a 'poison'. The second section moves onto how cirrhosis and its relation to alcohol was perceived among medical texts that were not explicitly tied to the temperance movement, including textbooks of general medicine, general medical journals, and numerous medical monographs. The final section studies how this expert knowledge was received in the wider context of the alcohol debate. A detailed look at some of the major temperance medical texts at the time shows that liver disease was integral to the movement's wholesale condemnation of alcohol.

The Politics of Alcohol in Edwardian Britain

Before exploring the knowledge surrounding alcohol and the liver, it would be necessary to outline the context of the alcohol problem in Edwardian Britain. According to Nicholls, the drink question was a high-profile political and social issue during the late nineteenth and the early twentieth centuries, a period when the debate on drunkenness reached, in the words of Greenaway, 'heights of acrimony and fierce political dispute' previously unseen in modern British history.[1] Naturally, many of the texts studied in this chapter communicate a polarised set of discourses on alcohol that conformed to the main ideological division at the time. The temperance movement employed a language in its medical literature whereby alcohol was framed as an 'evil' and a 'poison' that did nothing but harm to the body. As explored in the third section, the exaggeration of the harmful effects of alcohol crucially shaped how much of

[1] Nicholls, *The Politics of Alcohol*, p. 130; Greenaway, *Drink and British Politics*, p. 73.

the same publications understood the aetiology of cirrhosis. Such claims were contradicted by a set of separate discourses promoted by the liquor trade in their beverage advertisements, many of which presented alcohol as an energising, nutritious beverage that aided one's health. In addition to studying the seemingly contradictory understandings of alcohol, this section assesses the extent of the temperance movement's impact in shaping the public discussions surrounding drink. Joanne Woiak highlights the substantial influence commanded by the 'medical temperance movement' in promoting abstinence to the wider public.[2] Indeed, articles found in *The British Medical Journal* show that temperance doctors were at the very heart of the medical conversation on alcohol in Britain, promoting their specific brand of the problematisation of alcohol.

The temperance movement emerged in the 1830s as a social campaign that fought against the pervasiveness of drunkenness in British society. The anti-drink movement took on many different forms, spanning from the 'moderate' temperance movement and its targeted problematisation of drunkenness and sprit consumption, to teetotalism and its promotion of abstinence from all alcohol.[3] Thus far, historians have generally agreed that teetotalism originated in the Preston Temperance Society in 1832 when Joseph Livesey led the first public pledge to abstain from all intoxicating beverages.[4] By the middle of the nineteenth century, the strategic priorities of the teetotal temperance movement shifted from encouraging individuals to take the initiative to abstain towards supporting the legislative ban on the sale of alcohol. This was precipitated by the establishment of the United Kingdom (UK) Alliance in 1852, a pressure group that wielded considerable power in national politics in pushing for prohibition. Unlike the blanket nationwide ban on alcohol most commonly associated with the prohibition

[2] Joanne Woiak, '"A Medical Cromwell to Depose King Alcohol": Medical Scientists, Temperance Reformers, and the Alcohol Problem in Britain', *Historie Sociale/Social History* 27.54 (1994), pp. 353-60.

[3] Throughout this chapter, the teetotal wing of the temperance movement would be simply referred to as the 'temperance movement' unless expressed otherwise. The moderate temperance movement was primarily a phenomenon of the early half of the nineteenth century. The anti-drink discourse of the Edwardian era was dominated by those who advocated abstinence and prohibition.

[4] Shiman, *Crusade against Drink*, p. 18; Robert Duncan, *Pubs and Patriots: The Drink Crisis in Britain during World War One* (Liverpool, 2013), p. 15; Aidan Turner-Bishop, 'Livesey, Joseph (1794-1884)', in Jack S. Blocker, Jr., David M. Fahey, and Ian R. Tyrrell (eds.), *Alcohol and Temperance in Modern History: An International Encyclopaedia* (Santa Barbara, CA, 2003), p. 383.

era of the United States, the UK Alliance advocated the 'local option', which allowed local jurisdictions to hold a plebiscite over whether licenses for the sale of alcoholic beverages should continue to be permitted. Gradually, the effort to enact temperance legislation courted the Liberal Party after Wilfrid Lawson, the president of the UK Alliance, was elected to the House of Commons alongside several other influential temperance campaigners who likewise joined as MPs for the party. David Lloyd George was one such example of a strong supporter of temperance who eventually became Prime Minister during the First World War, playing a pivotal role in enacting some of the most restrictive anti-drink legislation under wartime conditions.

Outside the sphere of parliamentary politics, temperance was one of the major Victorian reform movements that spread across the country that tied together a broad milieu of individuals under a common set of values and causes. Nicholls estimates that every town in Britain had a teetotal society by the 1840s.[5] Communities of shared lifestyles around sobriety were embodied in the movement's numerous social institutions, including fraternities, friendly societies, and temperance hotels. The largest temperance organisation of all was the Band of Hope, a loosely knit agglomeration of Christian educational societies for children centred on the teetotal pledge. The group's popularity peaked in 1908 when its membership reached three million.[6] The Band of Hope provided many social opportunities for its adult and youth members, such as temperance education in Sunday schools, tea meetings, musical performances, and organised trips to neighbouring cities and towns. The more moderate, non-abstaining section of the movement had a place in the Church of England Temperance Society, which had 200,000 members in 1899.[7]

The temperance movement's attitudes to alcohol was reflected in many of the discourses that the campaign employed in referring to drink itself. Aside from their standard warnings against its perceived harms on the body, the movement was instrumental in scapegoating alcohol as a vice responsible for virtually every recognised social problem at the

[5] Nicholls, *The Politics of Alcohol*, p. 111.
[6] Duncan, *Pubs and Patriots*, p. 18.
[7] *Ibid.*, p. 20.

time, including crime, corruption, poverty, economic inefficiency, and the breakdown of families. One such example of this tendency was the ubiquitous use of the word 'evil' when referring to alcohol or its effects. In an anti-drink book titled *The Curse* (1903), M. C. Sykes argued that 'evil results of its [alcohol] abuse and… its pernicious actions on the various organs in man'.[8] When commenting on the prevalence of heavy drinking amongst the youth, prominent suffragist and physician Mary Murdoch stated in 1911 that '[t]he evil begins at the public schools and universities' where 'a career of bright promise' can be 'cut off by the indiscriminate use of alcohol'.[9] Thomas Kelynack, a prominent member of the British Society for the Study of Inebriety, argued that appropriate knowledge on alcohol's effects on the body was necessary to 'ameliorate the wide-spread evil which now threatens us with national disaster'.[10] An international medical temperance manifesto published in 1902 also condemned 'the terrible evils which have resulted from the consumption of alcohol', which encompassed both 'the injury to the individual and the danger to the community'.[11] As a by-product of the strong evangelical foundations of the temperance cause, drink was represented as a source of profound immorality and wickedness from which absolutely nothing 'good' was derived from its consumption.[12]

The above manifesto additionally stipulated that 'even a small quantity of alcoholic liquor, either immediately or after a short while, prevents perfect mental action and interferes with the functions of the cells and tissues of the body, impairing self-control by producing progressive paralysis of the judgement of the will; and having other markedly injurious effects.'[13] The belief that drinking at all levels was either harmful or had the potential of being

[8] M. C. Sykes, *The Curse* (Barnsley, 1903), p. 5.
[9] Mary Murdoch, 'One of England's Foes', in National Brotherhood Council, *Fifty Doctors Against Alcohol: A Call to National Defence* (London, 1911), p. 187.
[10] T. N. Kelynack, *The Alcohol Problem in its Biological Aspect* (London, 1906), p. 7.
[11] Anon., 'A Medical Temperance Manifesto', *The British Medical Journal* 1.2142 (18 January 1902), p. 170.
[12] Shiman, *Crusade against* Drink, p. 4; It remains uncontroversial among historians that the British temperance movement depended on the support of nonconformist Protestants. According to Matthew K. McKean, and Gerald Wayne Olsen, '[e]vangelicals spearheaded the debate over teetotalism and religion, and a helped Christianize antidrink movements by 1873', a quote found in Matthew McKean, 'Evangelical Temperance (United Kingdom)', in Blocker, Jr., Fahey, and Tyrrell (eds.), *Alcohol and Temperance in Modern History*, p. 225.
[13] Anon., 'A Medical Temperance Manifesto', p. 170.

harmful was a central doctrine of teetotalism, a line of discourse intended to critique the moderate, anti-spirits temperance movement that only went so far as to oppose heavy drinking. In an address delivered by pathologist G. Sims Woodhead at a 1904 conference for the British Temperance League, the audience applauded when he remarked that '[f]rom a medical point of view I am fully convinced that at no period of our lives are we better for alcohol.'[14]

Central to this belief in the harmfulness of all forms of drinking was another discursive trope in which alcohol was labelled a 'poison', a word connoting a substance that possessed a level of toxicity capable of causing instantaneous, rather than gradual, harm. [15] This understanding of drink constituted the 'slippery slope' thesis, a doctrine of teetotalism that stipulated that all levels of consumption, including moderate, had the potential to lead towards drunkenness, alcoholism, and eventual self-destruction.[16] An educational pamphlet published by the Band of Hope described alcohol as a 'deadly poison' that is both 'useless and dangerous'.[17] The international medical temperance manifesto insisted that 'alcohol must be regarded a *poison*, and ought not to be classed among foods.'[18] At the National Temperance League breakfast at Brighton on 24 July 1913, physician Mary Scharlieb argued that childbearing women should abstain because 'alcohol was just as much a poison as opium, strychnine, or arsenic'.[19] Philp Snowden, a prominent MP for the Labour Party and a teetotaller, referred to alcohol as a 'brain poison' that 'diminishes the fighting power of the workman'.[20] The framing of alcohol as a 'poison' exemplified the wider tendency of the temperance

[14] G. Sims Woodhead, *Alcohol and Health: Public and Private* (Sheffield, 1903), p. 3.

[15] According to the third edition of the *Oxford English Dictionary*, the most frequently used definition of 'poison' as a literal noun was one that pointed to it as a '[m]aterial that causes illness or death when introduced into or absorbed by a living organism, *esp. when able to kill by rapid action and when taken in small quantity*'. This definition has been used in the English language since at least c. 1225. 'Poison, n.', *OED Online*, December 2016, Oxford University Press, http://www.oed.com/view/Entry/146669?rskey= UvLZNV&result=1&isAdvanced=false [accessed 11 January 2017).

[16] Yeomans, *Alcohol and Moral Regulation*, p. 50.

[17] W. Chandos Wilson, *Temperance Science Lessons. No. 3: The Band of Hope Teacher, "Why Abstain?"* (Manchester, 1898), pp. 3-4.

[18] Anon., 'A Medical Temperance Manifesto', p. 170.

[19] Mary Scharlieb, 'The National Temperance League', *The British Medical Journal* 2.2745 (9 August 1913), p. 328.

[20] Philip Snowden, *Socialism and the Drink Question* (London, 1908), p. 32. Snowden was one of the central figures of the newly formed Labour Party, later becoming the Chancellor of the Exchequer in the first Labour government under Ramsay MacDonald in 1924.

movement to overstate the harmful properties of alcohol. This discursive label opposed the widely accepted notion that alcohol, far from being a 'poison' capable of causing serious harm through rapid action, was harmless to one's own health when consumed in moderate quantities.

The movement's wholesale condemnation of alcohol was also intended to debunk the idea that certain alcoholic beverages were necessary for the maintenance of good health, a belief aggressively promoted by the liquor trade. A Band of Hope pamphlet from 1905 addressed such perceptions on stout and port, arguing that alcohol ought to be deemed an 'inefficient' food in providing energy and strength.[21] A similar handbook for 'young abstainers' dispelled the popular characterisation of beer as 'liquid bread' by pointing out that the beverage was 'much liquid and little food', unlike real bread.[22] This belief in the 'food value' of drink was both exploited and promoted by 'the Trade', the temperance representation of the monolith of organised interests of publicans, retailers, brewers, distillers, and other groups that profited from the sale of alcohol.[23] The anti-drink movement viewed the Trade as its antagonist and a bulwark against its legislative effort to restrict or prevent the distribution of alcohol. The reality, however, was much more complicated. The alcoholic beverage industry faced major financial challenges during the Edwardian period, especially due to falling sales and profits resulting from the recession that immediately followed the turn of the century.[24] The call for licensing reduction and the encroachment of the local option drove the industry towards further consolidation against the implementation of legislation that threatened the existence of the beverage industry.[25] Yet, unlike the temperance movement, there were no large-scale public campaigns that were collectively coordinated by the numerous beverage industries before the First World War.

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[21] W. Chandos Wilson, *Temperance Science Lessons. No. 5: A Band of Hope Manual. Setting forth Physiological, Financial, and Social Reasons WHY all should Fight the Drink* (Manchester, 1905), pp. 7-8.

[22] J. James Ridge, *The Guide to Temperance for Young Abstainers and more Advanced Students, and for use in Bands of Hope* (London, 1903), p. 19.

[23] Nicholls, *The Politics of Alcohol*, p. 132.

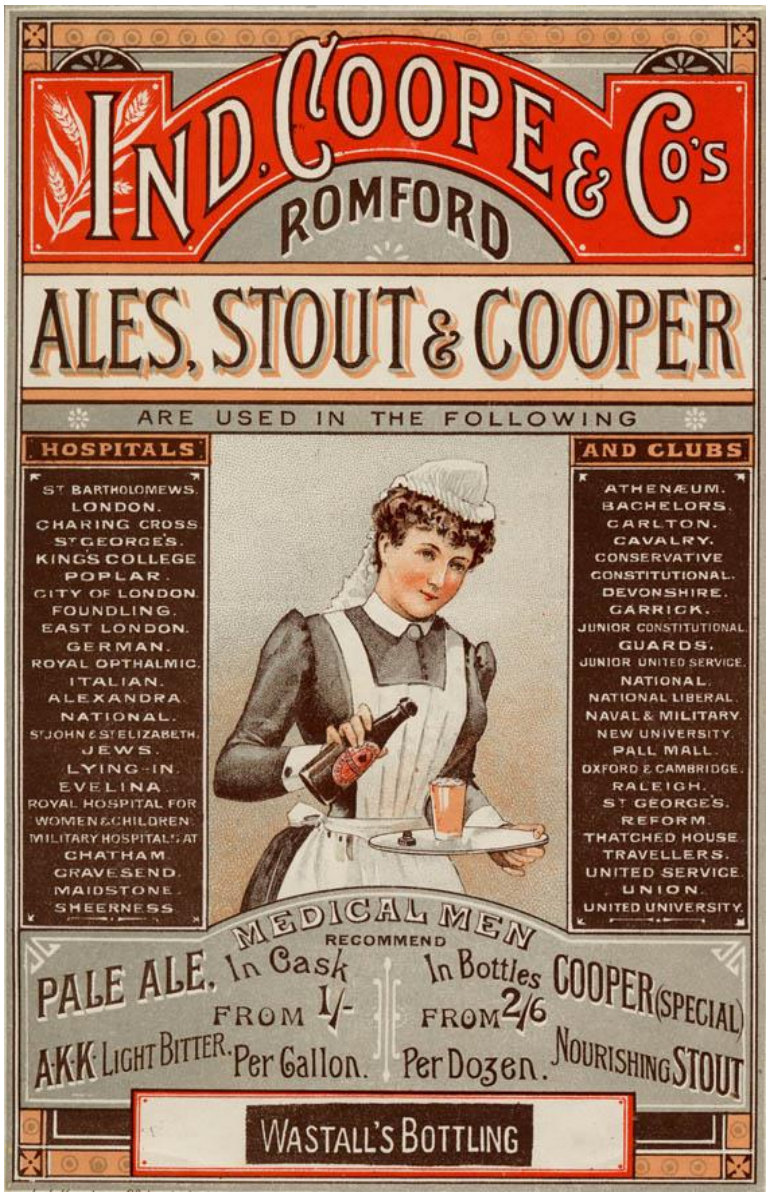
[24] In addition to the downturn in demand under the recession, the fall in sales was also caused by the rise of alternative products and leisure activities that gradually ate into consumer spending on alcohol. Greenaway, *Drink and British Politics*, p. 74.

[25] *Ibid.*, pp. 73-5; Duncan, *Pubs and Patriots*, pp. 35-6; Jennings, *A History of Drink and the English*, pp. 48-55.

The idea that certain alcoholic beverages possessed health-giving properties had been widely accepted in British society since at least the medieval period when ale and beer were consumed as ordinary components of the diet.[26] According to Hands, the general public continued to believe in the therapeutic qualities of drink well into the early twentieth century.[27] The Trade appealed to these popular understandings by conducting a war of publicity against the temperance movement through the use of advertisement. Beer, for instance, was promoted by the brewing industry under a distinctly medical set of tropes that directly contradicted the claim that it was a 'poison'. A characteristic example of this medical imagery could be found in a print advert produced by Ind. Coope breweries based in Burton-upon-Trent (see Figure 1). Arguing that their ales and stouts were recommended by 'medical men' for 'nourishment', the advert contained a list of hospitals across London that made use of their products to treat patients. A nurse depicted at the centre of the poster carried a serving tray with a glass of pale ale poured out of a bottle with an Ind. Coope label.[28] The use of the white-clad nurse, a gendered symbol that denotes the benevolence of a maternalistic, patient-centred medical care, was commonplace in many beer advertisements.[29]

[26] John Burnett, *Liquid Pleasures: A Social History of Drinks in Modern Britain* (London, 1999), pp. 111-32; Jennings, *A History of Drink and the English*, pp. 10-3; Yeomans, *Alcohol and Moral Regulation*, p. 6.
[27] Hands, *Drinking in Victorian and Edwardian Britain*, pp. 113-4.
[28] History of Advertising Trust: Image Gallery HAT62/3/621, 'Ind Coope Co Ale, Stout Cooper Print Advertising', 1900s.
[29] Another advert for James Calder & Co.'s 'strengthening invalid stout' similarly depicted a nurse handing the beverage poured out into a glass to a visibly ill male patient, found in Michael Jones, *Time, Gentlemen, Please! Early Brewery Posters in the Public Record Office* (Kew, 1997), p. 20.

FIGURE 1 'Ind Coope Co Ale, Stout Cooper Print Advertising', 1900s



Bass, another renowned brewery based in Burton, employed similar tropes in their newspaper advertisements to promote the 'nutritious' and 'wholesome' qualities of their pale ales.[30] The entire front page of the *Daily Mail* from 18 May 1910 was dominated by an advert titled 'Bass Notes, No. 8—The Dietetic Value of BASS', which stated that 'doctors speak emphatically of the wholesomeness and of the nutritive properties of a carefully brewed beer,

[30] Lesley Richmond, and Alison Turton, *The Brewing Industry: A Guide to Historical Records* (Manchester, 1990) p. 58

such as BASS'.[31] A similar advert from 1912 additionally noted that Bass produced 'pure beer, brewed solely from malt and hops'.[32] Bass also promoted its barley wine in 1910 by claiming the beverage was 'the best winter drink the Doctors recommend' that 'warms and nourishes' the body.[33] The frequent claim to wholesomeness and purity, strongly underlined by a set of 'clinical' discourses, reveal an unmistaken attempt by the brewers to associate their products with notions of plentiful energy and good dietary health. The adverts demonstrate that the Trade, along with the temperance movement, sought to attach their products to the authority of professional medicine.

Much like the brewers, distillers made similar claims about their spirits. Throughout the Edwardian era, Dewar was especially known to promote their 'White Label' whiskies for 'for their wholesomeness and their richness in nutrition' and 'carefully analysed by eminent medical men and other scientific authorities' for being 'absolutely pure, and a stimulant which may be taken in reason with benefit to health'.[34] White Horse whiskeys were similarly advertised for those who were 'careful for their health'.[35] An advert of the 'Pure Malt Whiskies' from W. & A. Gilbey's in *The Manchester Guardian* argued that '[o]ne can hardly exaggerate the importance to the general health of the community that the spirit consumed should be wholesome'.[36] Hence, claims that a certain alcoholic beverage provided good health was not restricted to beer at the time.

The food value of alcohol was similarly endorsed within debates over alcohol policy in Westminster. In the Houses of Parliament, identical health claims were often employed by pro-Trade politicians to argue for the defence of the interests of the brewing industry. The 'purity' and the 'wholesomeness' of beer was frequently mentioned as reasons to oppose legislation that restricted the availability of what some understood to be a nutritionally rich 'food' that should

[31] The National Brewery Centre: Scrapbook 89.1430.00 p. 37, 'Bass Notes, No. 8—the Dietetic Value of Bass', 18 May 1910.
[32] NBC: Scrapbook 89.1430.00 p. 108, 'Bass, the Standard Quality and Purity', 1912.
[33] NBC: Scrapbook 89.1430.00 p. 49, 'Bass No. 1 Barley Wine', 1910-1.
[34] 'Dewar's "White Label" Whisky', *Daily Mail,* 20 August 1903, p. 1; 'Dewar's "White Label" Whisky', *Daily Mail,* 6 December 1903, p. 1.
[35] 'Display Ad 17', *The Manchester Guardian*, 17 January 1912, p. 6.
[36] 'Display Ad 3', *The Manchester Guardian*, 24 February 1903, p. 5.

be freely available to the masses.[37] For instance, on 26 May 1905, Liberal Unionist MP Thomas Cochrane argued that the legislative attempt to force licensed premises to close on Sunday ignored the reality that 'most people in this country still regarded a certain amount of alcohol as an ordinary article of diet'.[38] Conservative MP Samuel Robert called for the government to rethink their plan on increasing import tariffs of black beer, which he believed to be 'more a food for invalids than ordinary beer'.[39] Such instances of parliamentary opposition to temperance legislation illustrate how the political right was generally more sympathetic to the interests of the liquor trade than the anti-drink movement.

Similar to the Trade's efforts to mould the public's perception of alcohol, the temperance movement exerted its own influence on the knowledge on alcohol and the body through the organs of the medical establishment. A study of the institutional presence of temperance doctors in organisations such as the British Medical Association (BMA), the most important professional body representing the interests of doctors across Britain, reveals that temperance ideology achieved a degree of scientific legitimacy among some leading medical professionals in Britain. Divorced from the perceived 'radicalism' of the early prohibitionists, temperance doctors saw themselves as legitimate authorities on matters of alcohol and health.[40] One notable organisation to emerge out of this trend was the Society for the Study of Inebriety, established in 1884 by Norman Kerr. Kerr and the Society called for the medicalisation of habitual drunkenness as a 'disease' within the framework of professional medicine, and later campaigned for the establishment of the earliest asylums to treat inebriety.[41] Although temperance doctors ultimately constituted no more than a minority of all medical professionals in Britain, the 'medical temperance movement' was undoubtedly an influential section of the wider anti-drink movement in the late nineteenth and early twentieth centuries.[42] A connection between the more moderate wings of the movement with the nation's wider community of

[37] HC Deb 13 December 1900, vol 88, cc698-9; 27 March 1901, vol 91, col 1441-507; 16 March 1906, vol 153, cc1541-83.
[38] HC Deb 26 May 1906, vol 146, col 1574.
[39] HC Deb 24 November 1914, vol 68, col 974.
[40] *Ibid.*, p. 341.
[41] Nicholls, *The Politics of Alcohol*, pp. 161-2; Berridge, *Demons,* pp. 62-8.
[42] Woiak, '"A Medical Cromwell"', pp. 338, 352.

medical professionals can be discerned by looking at some of the published accounts of meetings and events of temperance physicians, most of which can be found in *The British Medical Journal*, the officially certified journal of the BMA.

In 1879, the British Medical Temperance Association (BMTA) was established by the National Temperance League for the promotion of abstinence among practising doctors and research on alcohol and health. In the same year, the first 'temperance breakfast' of the BMTA was hosted and sponsored by the BMA as a gathering of doctors allied to the temperance campaign.[43] In July 1911, the BMA organised a large conference on the problems of alcohol consumption in Birmingham on behalf of the BMTA, attracting as many as 15,000 attendees. The event involved lectures delivered by 50 physicians that were represented by the BMA and the BMTA, many of whom argued for the benefits of abstinence over moderate drinking.[44] Thus, as an association of more than five hundred physicians sympathetic to the cause, the BMTA was deeply embedded within the structures of the BMA.[45]

The wide-ranging influence of temperance medicine was further confirmed by their role in the introduction of a school curriculum in 1909 to promote temperance across state schools in Britain. At a session chaired by the president of the BMA on 5 August 1905, members of the BMTA and the Society for the Study of Inebriety announced that they had managed to collect nearly 15,000 signatures from medical professionals from across Britain for a petition to introduce a provision for the 'instruction in hygiene and temperance' in primary education.[46] The council of the BMA played an instrumental role in submitting the signatures directly to the Board of Education in Whitehall. As an early public health measure, it pushed for the inclusion of the 'elementary scientific instruction in health subjects, including temperance' in primary schools to prevent the 'deterioration of the national physique'. Alongside the need to

[43] Anon., 'British Medical Temperance Association', *The British Medical Journal* 2.973 (23 August 1879), pp. 309-10.
[44] National Brotherhood Council, *Fifty Doctors Against Alcohol.*
[45] Kenneth Pearl, 'British Medical Temperance Association (BMTA)', in Blocker, Jr., Fahey, and Tyrrell (eds.), *Alcohol and Temperance in Modern History*, p. 112.
[46] Anon., 'The National Temperance League Breakfast' *The British Medical Journal* 2.2327 (5 August 1905), pp. 284-5.

promote personal hygiene, it argued that 'the evil effects of alcoholic indulgence are among the prime causes of the squalor and disease by which the poorer quarters of our large towns are disgraced; that in fact ignorance and intemperance are the causes of much of the poverty which makes slums possible.'[47]

The Board of Education eventually agreed in 1909 to introduce hygiene and temperance as compulsory subjects across state-funded schools. However, certain parts of the curriculum were augmented from what was originally suggested. The Board was suspicious of the dogma that alcohol had '"deleterious consequences when taken as a beverage in any conditions whatsoever"', stating that the belief 'rested on somewhat precarious foundations'. As a result, it was decided that the curriculum should promote the 'manifest advantages of abstemiousness' without endorsing the temperance movement's uncompromising rejection of moderate drinking.[48] At the same time, a clause in the curriculum that warned that excessive consumption had the capacity to turn individuals into a 'mental or physical wreck' and make it harder for them 'have long or healthy lives' was approved.[49] A section titled '[e]vil consequences of intemperance to the individual, to the home, and to the state' went as far as to state that alcohol caused 'moral injury to himself and great harm to others', portraying it as a harmful substance responsible for poverty, loss of 'self-control', ill health, and 'moral degradation'.[50] The introduction of a temperance curriculum to the Board of Education was an instance when the medical temperance movement was capable of exercising its power in affecting the national conversation surrounding drink. Confronted with the liquor trade and their continued insistence on the food value of alcohol, temperance doctors were key players in shaping the content of the debate over alcohol and health in Edwardian society.

[47] Anon., 'Instruction in Hygiene and Temperance', *The British Medical Journal* 1.2247 (23 January 1904), pp. 201-2.
[48] Anon., 'Hygiene and Temperance in Elementary Schools', *The British Medical Journal* 1.2528 (12 June 1909), p. 390.
[49] *Ibid.*, pp. 390-1.
[50] *Ibid.*, p. 391.

Edwardian Medical Understandings of Alcohol and the Liver

This section explores how cirrhosis aetiology was understood by the wider medical profession situated outside of the alcohol debate. In Edwardian Britain, alcohol was broadly perceived to be the primary cause of liver damage among the majority of medical experts. The predominant discourse that characterised cirrhosis as an 'alcoholic's disease' was closely tied to the DTT, which understood alcohol to have a direct role in harming the liver. However, a handful of doctors were not entirely convinced that this causality was as straightforward as it was commonly supposed by most medical observers. Doubts over the simple action of alcohol in harming the liver were based on legitimate grounds, many of which pointed to the inconsistent outcomes of past experimental studies in their attempts to demonstrate causation between alcohol and cirrhosis. Sceptics also remarked on how cirrhosis developed in only a minority of heavy drinkers, even though their excessive habits continued to be understood to directly result in serious liver damage. Thus, seeing how the topic was discussed in medical textbooks, journals, and monographs from the period, it becomes clear that the intricacies surrounding the pathogenesis of the disease received insufficient attention from most medical professionals at the time. While Herd and Katcher rightly highlight the existence of a widely held assumption at the beginning of the twentieth century that cirrhosis was a direct outcome of alcohol consumption, they underestimate the existence of sceptical voices that proved to be the antecedents of the eventual dismissal of alcohol's direct causation in the interwar period.[51]

In order to assess the level of knowledge on cirrhosis in Edwardian Britain, this section examines a total of 14 separate publications of five textbooks of general medicine published between 1900 and 1914 (see Figure 2; see Appendix: Textbooks of General Medicine for long-term shifts). Before the First World War, medical textbooks were written by a single author rather than being the product of multiple authors. This understandably restricted their capacity to provide the most 'accurate' knowledge on any given topic. The absence of referencing also makes it impossible to determine the sources with which the authors attributed their claims to.

[51] Herd, 'Ideology, history and changing models', pp. 1113-26; Katcher, 'The post-repeal eclipse', pp. 729-44.

Thus, the usefulness of the textbooks is primarily based on their wide readership, giving historians a sense of the established knowledge that were disseminated within the medical community as a whole rather than that of the content of the most recent advancements in medicine at the time.

FIGURE 2 Textbooks of general medicine, 1900~1915

	1900~05	1906~10	1911~15
James M. Anders, *A Text-book of the Practice of Medicine*	4th edition (1900) 'although the quantity necessary to produce the disease varies greatly in different individuals...by the side of alcoholism all other causes combined are comparatively insignificant.'	8th (1908) identical claim to 4th edn	11th (1913) identical claim to 4th edn
William Osler, *The Principles and Practice of Medicine*	4th (1901) 'Alcohol is the chief cause of cirrhosis... [o]ther poisons... play a minor role'		8th (1912) Alcohol 'produces definite changes in the liver' with '[d]egenerative changes in the liver cells
Frederick Taylor, *A Manual of the Practice of Medicine*	6th (1901) 'great majority of cases the cause of cirrhosis' is the '[e]xcessive use of alcohol', '[b]ut the simple theory of direct irritation by alcohol has not escaped criticism' and '[w]here alcoholic excess cannot be proved, a satisfactory explanation is rarely forthcoming.'	8th (1908) identical claim to 6th edn	9th (1911) identical claim to 6th edn
Alexander Wheeler, *Handbook of Medicine*	2nd (1903) 'By far the most important factor in producing this disease is the excessive use of alcohol, especially spirits'	3rd (1908) 'Of those causes that may affect the liver by way of the portal vein, alcohol, especially in the form of spirits, is certainly the most important.'	4th (1912) 'Alcohol is not the direct cause of cirrhosis, its specific action on the liver being to produce fatty change, but it lowers the resistance of the liver to the action of other poisons, or possibly even of micro-organisms, which are then free to set up connective tissue proliferation.'
Thomas Dixon Savill, *A System of Clinical Medicine*	1st (1903) 'cirrhosis of the liver must still be regarded as mainly the result of alcoholic excess' and 'Alcohol is undoubtedly the most usual cause of atrophic cirrhosis'	2nd (1909) identical claim to 1st edn	3rd (1912) identical claim to 1st edn

Before touching on the content of the textbooks, it would be necessary to show that the volumes discussed in this chapter were generally well received. Due to my inability to find the exact records for sales and circulation, I made use of book reviews in medical journals from

Britain, mostly from *The British Medical Journal* and the *Postgraduate Medical Journal*, as indicators for the reputation and popularity of each textbook in Britain. Although every textbook studied in this chapter, as well as in the rest of the thesis, were produced by an international group of authors mostly based in Britain and the United States, these book reviews show that such publications were heavily relied on by medical professionals in Britain. The most renowned textbook from the period was William Osler's *Principles and Practice of Medicine*. A review of its seventh edition stated that its 'general plan… is so familiar that it is not necessary to refer to it in this review', while another review from 1912 touted that it 'has become so universally accepted as "guide, philosopher, and friend" in matters of medical interests that he [Osler] practically sets the current of medical ideas'.[52] The textbook left the strongest legacy among all other volumes, indicative of how a review of its final edition from 1943 referred to it as 'the last great medical textbook by a single writer'.[53] In a posthumous article on Osler's legacy, W. B. Matthews additionally mentioned James M. Anders's *Textbook of the Practice of Medicine* as the 'only book that I found to have held its own with Osler'.[54] A 1908 review in *The British Medical Journal* described Anders's work as 'one of the best textbooks on medicine in the English language', while a later 1912 review argued that '[t]here is no better American work'.[55] Frederick Taylor's *Manual of the Practice of Medicine* was also referred to in a 1918 review as 'Olser's rival work'.[56] Taylor's textbook was particularly praised for containing the most recent knowledge on any particular subject, with one reviewer recommending it 'with the utmost confidence to all busy men who, in the bustle of work, desire to acquaint themselves with the main facts of up-to-date investigation in regard to any

[52] Anon. [review], 'William Osler, *The Principles and Practice of Medicine* (7th edn, London, 1909)', *The British Medical Journal* 2.2543 (25 September 1909), p. 883; Anon. [review], 'William Osler, *The Principles and Practice of Medicine* (8th edn, London, 1912)', *The British Medical Journal* 2.271 (7 December 1912), p. 1611.
[53] Anon. [review], 'Henry A. Christian, and William Osler, *The Principles and Practice of Medicine* (14th edn, London, 1942)', *The British Medical Journal* 1.4283 (6 February 1943), p. 163.
[54] W. B. Matthews, 'Osler Oration', *Journal of the Royal Society of Medicine* 75 (May 1982), p. 308.
[55] Anon. [review] 'James M. Anders, *A Textbook of the Practice of Medicine*', *The British Medical Journal* 1.2469 (25 April 1908), p. 993; Anon. [review], 'James M. Anders, *A Textbook of the Practice of Medicine*', *The British Medical Journal* 1.2682 (25 May 1912), p. 1186.
[56] Anon. [review], 'Frederick Taylor, *The Practice of Medicine* (11th edn, London, 1918)', *The British Medical Journal* 1.2984 (9 March 1918), p. 288.

disease'.[57] Additionally, Alexander Wheeler's *Handbook of Medicine*, which 'for many years was widely read by students in Great Britain', was praised as an 'ideal practical companion for the period of clinical clerking'.[58] Thomas Dixon Savill's *System of Clinical Medicine* presented itself to be the most controversial, with one reviewer pointing out that '[t]here is hardly a page in which one does not come across either an incorrect statement or a dogmatic opinion'.[59] In spite of that, later reviews credited its 'high reputation' among practitioners and that it was 'at least as good as and in many respects superior to' other textbooks.[60]

The existence of a consensus surrounding cirrhosis becomes immediately apparent when quantifying the proportion of various stances regarding the question of its aetiology. Cirrhosis was directly attributed to the action of alcohol in all 14 textbooks. Among them, 53 per cent (7 out of 13) provided a straightforward account of the causation. The 1901 edition of Osler's *Principles and Practice of Medicine* asserted that '[a]lcohol is the chief cause of cirrhosis of the liver' and that '[o]ther poisons, such as lead and the toxic products of faulty metabolism in gout, diabetes, rickets, and indigestion, play a minor role'.[61] The later 1912 edition more specifically argued that alcohol '[p]roduces definite changes in the liver' by producing '[d]egenerative changes in the liver cells'.[62] The 1903 edition of Wheeler's *Handbook of Medicine* identified alcohol as '[b]y far the most important factor in producing' cirrhosis, much like the subsequent 1908 edition that argued that, '[o]f those causes that may affect the liver by way of the portal vein, alcohol, especially in the form of spirits, is certainly the most

[57] Anon. [review], 'Frederick Taylor, *The Practice of Medicine* (7th edn, London, 1904)', *The British Medical Journal* 1.2298 (14 January 1905), p. 78; Anon. [review], 'Frederick Taylor, *The Practice of Medicine* (9th edn, London, 1911), *The British Medical Journal* 2.2657 (2 December 1911), p. 1478.

[58] Anon. [review], '*Wheeler and Jack's Handbook of Medicine* (revised by Robert Coope, 11th edn, Edinburgh, 1950)', *Postgraduate Medical Journal* 27. 307 (1 May 1951), p. 255; Derrick Dunlop [review], '*Wheeler and Jack's Handbook of Medicine* (revised by Robert Coope, 12th edn, Edinburgh and London, 1963)', *The British Medical Journal* 2.5353 (10 August 1963), p. 381.

[59] Anon. [review], 'Thomas D. Savill, *A System of Clinical Medicine* (2nd edn, London, 1905)', *The British Medical Journal* 2.2323 (8 July 1905), p. 75.

[60] Anon. [review], 'Agnes Savill (ed.), *A System of Clinical Medicine* (2nd edn, London, 1905)', *The British Medical Journal* 2.2323 (12 December 1925), p. 1131; Anon. [review], 'Agnes Savill, and E. C. Warner (eds.), *Savill's System of Clinical Medicine* (11th edn, 1939, London)', *Postgraduate Medical Journal* 16.215 (June 1940), p. 215.

[61] William Osler, *The Principles and Practice of Medicine* (4th edn, Edinburgh, 1901), p. 569.

[62] William Osler, and Thomas McCrae, *The Principles and Practice of Medicine* (8th edn, London, 1912), p. 397.

important'.[63] Similarly, three consecutive editions of Savill's *System of Clinical Medicine* from between 1903 and 1912 stated that 'cirrhosis of the liver must still be regarded as mainly the result of alcoholic excesses' and that '[a]lcohol is undoubtedly the most usual cause of atrophic cirrhosis'.[64] Passages that provided a simple explanation of cirrhosis and its relation to alcohol clearly constituted a majority of the textbooks published during the Edwardian period.

On the other hand, 43 per cent of the textbooks (6 out of 14) chose to highlight the intricacies surrounding alcohol's action on the liver without explicitly denying its status as the primary cause. The 1900, 1908, and 1913 editions of Anders's *Textbook of Medicine* stated that, 'although the quantity necessary to produce the disease varies greatly in different individuals… by the side of alcoholism all other causes combined are comparatively insignificant'.[65] The textbook recognised the role of differences in one's susceptibility to alcohol's toxic action, referring to how the incidence of the disease varied heavily across individuals. A similar argument concerning the DTT could be found in Taylor's *Manual of the Practice of Medicine*, three editions of which pointed to the existence of the 'widest individual differences' in the volume of alcohol necessary to produce cirrhosis. Taylor went on to mention that there were other likely explanations of the pathogenesis of cirrhosis apart from the action of alcohol. He stated that 'the simple theory of direct irritation by alcohol has not escaped criticism; and the following views have also been advanced: that the real irritant is some toxin produced in the mucus which results from the accompanying gastritis; that the irritant is not alcohol, but some other constituent of the liquid drunk.' The textbook nonetheless admitted that, '[w]here alcoholic excess cannot be proved, a satisfactory explanation is rarely forthcoming' and that '[e]xcessive use of alcohol, in the form of beer, wine or spirits' was still the 'great majority of

[63] Alexander Wheeler, and William R. Jack, *Wheeler's Handbook of Medicine and Therapeutics* (2nd edn, Edinburgh, 1903), p. 229; Wheeler, and Jack, *Wheeler's Handbook of Medicine*, p. 197.
[64] Thomas Dixon Savill, *A System of Clinical Medicine* (1st edn, London, 1903), pp. 450-1; Thomas Dixon Savill, *A System of Clinical Medicine* (2nd edn, London, 1909), pp. 373-4; Thomas Dixon Savill, *A System of Clinical Medicine* (3rd edn, London, 1912), pp. 362-3.
[65] Anders, *A Text-book of the Practice of Medicine* (4th edn), p. 889; James M. Anders, *A Text-book of the Practice of Medicine* (5th edn, London, 1902), p. 893; James M. Anders, *A Text-book of the Practice of Medicine* (8th edn, London, 1908), p. 927.

cases the cause of cirrhosis'.[66] Although most of the textbooks were in agreement over the DTT, some of the authors were correct to highlight the difficulties of assuming that alcohol was, without exception, the cause of cirrhosis.

The only textbook from the period that explicitly challenged this consensus was the 1912 edition of Wheeler's *Handbook of Medicine*, revised at the time by William R. Jack.[67] Jack boldly proclaimed that '[a]lcohol is not the direct cause of cirrhosis', further arguing that it only functioned to lower 'the resistance of the liver to the action of other poisons, or possibly even of micro-organisms, which are then free to set up connective tissue proliferation.'[68] Unlike to every other textbook studied for this section, Jack's volume understood drinking to be an indirect, rather than a direct, cause of the disease, whereas the *true* cause was attributed to other exogenous agents that enter the body. Although such views were clearly held only by a minority of physicians at the time, that there were other medical texts from the period that similarly disagreed with the direct culpability of alcohol is indicative of how Jack's opinion was not entirely baseless.

Unlike the clear consensus that had existed in most textbooks over alcohol's direct causation of cirrhosis, a minority of medical professionals expressed doubts over the DTT. Scepticism towards the role of alcohol was expressed by doctors who questioned the theory upon the recognition of many of the often-unaddressed scientific inconsistencies that made it difficult to assume the causality to be linear and direct. A wide variety of medical texts, including monographs, accounts of public lectures, and journal articles show that a handful of medical professionals had good reasons not to take the DTT at face value. Unsurprisingly, sceptical physicians tended to be more attuned to the specialised knowledge surrounding the complex nature of the causation of cirrhosis than authors of textbooks of general medicine. Although a more articulated set of objections towards the DTT did not emerge until the

[66] Frederick Taylor, *A Manual of the Practice of Medicine* (6th edn, London, 1901), p. 714; Frederick Taylor, *A Manual of the Practice of Medicine* (8th edn, London, 1908), p. 766; Frederick Taylor, *A Manual of the Practice of Medicine* (9th edn, London, 1908), p. 778.
[67] William R. Jack, *Wheeler's Handbook of Medicine* (4th edn, Edinburgh, 1912), p. 212.
[68] *Ibid.*, p. 212.

interwar era, the reasons that grounded them were often identical to those highlighted by the small number of critics from before the First World War.

Before touching on the critique of the DTT, it must be stressed that there still existed a broad agreement over the theory across general medical journals, even though to a lesser extent than in the textbooks. Articles in *The British Medical Journal* frequently affirmed the DTT, with one column from 1901 observing that 'there is a firmly rooted belief that alcohol is practically the sole cause'.[69] In response to the suggestion that cirrhosis was caused not by alcohol itself but by the acid sulphate found in wine, a column from 1907 stated that the theory was 'absurd', adding that '[t]here is no room to doubt that the cause of atrophic cirrhosis of the liver is the drinking of alcohol'.[70] An article from 1906 maintained that, ultimately, the most reliable approach to treating cirrhosis must be the maintenance of total abstinence from alcohol, while a *Lancet* report on a clinical study in the Boston City and Massachusetts hospitals identified alcohol as by far the most important factor in the causation of cirrhosis, based on a primary observational data that showed that 69 out of the 78 patients with the disease had admitted to abusing alcohol.[71] In 1900, the same journal published an account of the year's Lumleian lecture delivered at the Royal College of Physicians. In it, paediatrician W. B. Cheadle stated that doctors had the habit of neglecting other potential illnesses that result from alcoholism by paying too much attention to the liver. Understanding cirrhosis as 'only one phase of chronic alcoholism', physicians commonly talked about liver disease 'as if all the evil effect of the poison were concentrated upon the liver alone' and assumed that the illness was 'the single and only result' of heavy drinking. He, therefore, called for more attention to be paid to other harms, stating that '[t]he heart, the kidneys, the spleen, the pancreas, the blood-vessels, and the nervous

[69] Anon., 'The Etiology of Hepatic Cirrhosis', *The British Medical Journal* 1.2111 (15 June 1901), pp. 1502-3; W. Hale White, 'An Address on some Misconceptions with regards to Diseases of the Liver', *The British Medical Journal* 1.2201 (7 March 1903), pp. 533-7; James Barr, 'Presidential Address on Alcohol as a Therapeutic Agent', *The British Medical Journal* 2.2322 (1 July 1905), pp. 4-8.

[70] Anon., 'Plastered Wines', *The British Medical Journal* 2.2437 (14 September 1907), pp. 686-7.

[71] Anon., 'The Surgical Treatment of Ascites in Vascular Cirrhosis of the Liver', *The British Medical Journal* 2.2393 (10 November 1906), p. 1320; Anon., 'Cirrhosis of the liver', *The Lancet* 160.4130 (25 October 1902), pp. 1141-2.

system all suffer'.[72] Cheadle's remarks were indicative of how liver disease occupied a central place among the cohort of different diseases that were associated with alcohol.

The abundance of evidence surrounding the continued affirmation of the alcoholic aetiology of cirrhosis was balanced by those of other opinions that questioned it. A particular objection to the DTT was derived from the observation that cirrhosis was statistically rare among alcoholics. The reality that the vast majority of heavy drinkers, even after years of habitual consumption, were absolved from ever developing a cirrhotic liver was commonly referred to in order to question the assumption that alcohol was a toxin capable of directly causing harm on the organ. An article in *The British Medical Journal* discussed an autopsy study in which one of its authors, Arthur Voelcker, remarked that 'alcohol played an important part in the production of cirrhosis, but in what way was uncertain.'[73] In 1907, the journal received a correspondence from R. Welsh Branthwaite, a civil servant working for the Home Office, who expressed 'repeated doubt as to the existence of [a] relationship between' alcohol and cirrhosis because he had 'never met a single case of cirrhosis' throughout the 25 years that he had spent working at a reformatory for inebriates.[74] Similarly, Francis Hare, a superintendent of the Norwood sanatorium for inebriates in South London[75], suggested that 'doubts have been expressed as to the real frequency of the disease' among alcoholic patients.[76] In referencing a study by Frederick Walker Mott, Hare suggested that cirrhosis was much more common in general hospitals than in asylums because a 'large number of asylum patients who have been inebriates suffer from intolerance of alcohol, and that such can very seldom drink sufficient

[72] W. B. Cheadle, 'The Lumleian Lectures on Some Cirrhoses of the Liver: lecture 2', *The Lancet* 155.3997 (7 April 1900), pp. 986.
[73] Anon., 'A discussion on the pathology of cirrhosis of the liver in adults and young children', *The British Medical Journal* 2.2074 (29 September 1900), pp. 913-7.
[74] R. Welsh Branthwaite, 'Alcoholism and Hobnail Liver', *The British Medical Journal* 2.2445 (9 November 1907), pp. 1375-6.
[75] The Norwood sanatorium was a private medical establishment for rehabilitation from alcohol and drug addiction, catering mostly to wealthy patients. Matthew Smith, *Another Person's Poison: A History of Food Allergy* (New York, 2015), p. 59.
[76] Francis Hare, *On Alcoholism its Clinical Aspects and Treatment* (London, 1912), p. 106.

alcohol, extended over sufficient length of time, to produced advanced cirrhosis'.[77] Arguing that 'this well-known, and constantly looked for alcoholic complication is considerably less frequent than is commonly supposed', he concluded that cirrhosis is ultimately 'restricted to the class of chronic alcoholics'.[78] Although Hare did not necessarily deny that alcohol was directly responsible for the disease, his clinical findings were intended to encourage others to question the assumed interconnectedness between cirrhosis and alcoholism.

Other medical observers highlighted the virtual absence of a clear experimental demonstration of the mechanism of alcohol's toxicity on the liver in animal subjects. Ever since the mid-nineteenth century, the laboratory had been understood as the prime source of knowledge production for the establishment of disease aetiology. Animal experiments are useful in allowing scientists to test multiple variables within a carefully controlled environment without being restricted by the ethical constraints of a clinical study on human patients.[79] The necessity of experimental evidence in demonstrating causation was promoted most famously by bacteriologist Robert Koch, who established a criterion as part of his postulates that stipulated that the specific disease agent must be isolated in the diseased subject in order for the agent to be legitimately identified as a cause. Although Koch's postulates were initially intended to account for infectious diseases, the primacy of laboratory evidence proved to be enormously influential among doctors in the early half of the twentieth century in strengthening the standard of required scientific evidence for the demonstration of a causality between a factor and an illness.[80] The required evidence was that the administration of the putative factor—alcohol—to animals would result in the development of serious liver damage.

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[77] *Ibid.*, p. 106. Mott, an experienced biochemist and neuropathologist who co-founded the Maudsley Hospital, performed autopsy studies in two different institutions across a decade, first at the Claybury Asylum where only 1.8 per cent of alcoholics developed cirrhosis, and at the Charing Cross Hospital where the number was 7.7 per cent. F. W. Mott, 'A Discussion on Alcohol and Insanity', *The British Medical Journal* 2.2439 (28 September 1907), pp. 797-803.

[78] Hare, *On Alcoholism*, p. 106.

[79] Roy Porter, *The Greatest Benefit to Mankind: A Medical History of Humanity from Antiquity to the Present* (London, 1999), p. 320.

[80] *Ibid.*, p. 532.

In *Diseases of the Liver, Pancreas, and Suprarenal Capsules*, German physicians Heinrich Quincke and G. Hoppe-Seyler stated that '[t]he chief cause of cirrhosis of the liver is continued indulgence in alcoholic liquors' in addition to 'other concomitant features'.[81] The authors, however, proceeded to point out that all claims on the specific mechanism of causation must be denounced as 'conjectures' since 'no one has so far succeeded in producing experimental cirrhosis in animals by the administration of alcohol'.[82] A column in *The British Medical Journal* from 1914 similarly pointed out that the varying success in the laboratory reproduction of the disease made it difficult for generalisations to be made on its aetiology, arguing that '[t]he pathology of cirrhosis of the liver is by no means so clear that dogmatic utterances are permissible.' It went on to argue that '[e]ven the role played by alcohol in the production of cirrhosis has been challenged by capable pathologists, and, on the other hand, typical hob-nail livers have been observed in the etiology of which alcohol could not be definitely excluded.' The author thus concluded that '[i]t is still quite uncertain' how alcohol acts on the liver, even going as far as to hint that 'it acts… indirectly by damaging the gastro-intestinal canal'.[83] Such observers, therefore, believed that the failure to reproduce cirrhosis in animals was partly due to the inability of scientists to replicate human conditions for the experimental subjects. Humans were able to withstand years of heavy drinking to the point where their liver might suffer from cirrhosis, whereas animals rarely survived beyond the earliest stages of liver damage. The absence of sufficient experimental proof of alcohol's direct causation became more frequently highlighted throughout the rest of the twentieth century by later critics of the DTT.

Among the sceptical doctors that this chapter has examined thus far, Humphry Rolleston was perhaps the most influential physician in Edwardian Britain to directly oppose the consensus surrounding the DTT. Rolleston was one of the leading medical authorities in the early twentieth century who had published on a variety of topics in the discipline. Following

[81] H. Quincke, and G. Hoppe-Seyler, 'Diseases of the Liver', in Reginald H. Fitz, and Frederick A. Packard (eds.), *Diseases of the Liver, Pancreas and Suprarenal Capsules* (London, 1903), p. 692.
[82] *Ibid.*, p. 693.
[83] Anon., 'An Epitome of Current Medical Literature', *The British Medical Journal* 1.2782 (25 April 1914), pp. 68.

the First World War, his reputation allowed him to be elected as the president of both the Royal Society of Medicine and Royal College of Physicians and to become the personal physician to George V.[84] In a chapter on alcoholism in a textbook edited by himself, Rolleston premised that the toxicity of alcohol was often 'directly or indirectly responsible for the numerous morbid changes found in the organs and tissues of chronic alcoholics'.[85] Specifically concerning the liver, he then went on to argue that 'the failure to produce hepatic cirrhosis in animals by the administration of alcohol' indicated that 'alcohol is responsible for cirrhosis in an *indirect* fashion, namely, by setting up gastro-enteritis which gives rise to poisons possessing a sclerogenic effect on the liver.'[86] This assertion was further reinforced by his knowledge of how many of the autopsy studies had shown that cirrhosis was prevalent among only a minority of alcoholics.[87] This was the clearest endorsement of a theory in which alcohol was designated to playing an indirect role in allowing some other factor to cause liver damage.

In a separate monograph on the *Diseases of the Liver, Gall-bladder and Bile-ducts* (1905), Rolleston likewise argued that, '[w]ith regard to the question whether alcohol is the cause of cirrhosis, clinical and experimental evidence are hardly in accord.'[88] In addition to the statistical rarity of cirrhosis among hospitalised alcoholics, he reiterated that animal experiments, for the most part, had failed to reproduce liver damage beyond its early stages.[89] Therefore, Rolleston stated that, '[s]ince alcohol alone is not sufficient to account for cirrhosis either in man or animals, the undoubted association between alcoholism and cirrhosis must be explained in some other way.'[90] Much like in his previous book, he went on to suggest that it was more likely that alcohol acted indirectly on the liver by damaging some other organ, as 'alcohol has no specific action on the liver except [in causing] fatty degeneration'.[91] Among the variety of texts studied for this chapter, Rolleston's views on the question was the most explicit critique of the

[84] Anon., 'Sir Humphry Rolleston', *The British Medical Journal* 2.4369 (30 September 1944), pp. 452-4.
[85] H. D. Rolleston, 'Alcoholism', in Thomas Clifford Allbutt, and Humphry Davy Rolleston (eds.), *A System of Medicine: Volume II, Part I* (London, 1906), p. 916.
[86] *Ibid.*, pp. 916-7.
[87] *Ibid.*, p. 917.
[88] H. D. Rolleston, *Diseases of the Liver, Gall-bladder and Bile-ducts* (1st edn, London, 1905), p. 182.
[89] *Ibid.*, pp. 182-3.
[90] *Ibid.*, p. 183.
[91] *Ibid.*, pp. 183-4.

DTT. His capacity to account for the contradictions between the theory and the outcomes of some of the experimental and clinical studies proved to be a set of indictments that became much more influential after the First World War. In the meanwhile, the opinions of much of the textbooks and journal articles form the period shows that sceptical voices such as those of Rolleston's were still restricted to a minority. Although many of the doubts were founded on good reason, cirrhosis was regarded by most medical professionals to be a direct outcome of the action of alcohol on the liver.

Cirrhosis Aetiology and the Edwardian Drink Question

The disease knowledge on alcohol and liver disease played a pivotal role in shaping how alcohol was understood as a problematic substance in Edwardian Britain. Cirrhosis was framed as among the wide variety of health problems known to be associated with drink, and, unsurprisingly, politically charged references to alcoholic liver damage were almost exclusively produced by the temperance movement. Various medical texts attributed to the anti-drink movement show that the knowledge on alcohol's toxicity to the liver was used by temperance campaigners to overstate the effects of alcohol on the body, consequentially reinforcing their discursive framing of alcohol as a 'poison'. Unlike many of the medical texts explored in the previous section, many of the temperance medical literature gave a markedly straightforward account of liver damage, presenting a linear relationship between the cause and the disease that omitted the complexities grounding the pathogenesis of cirrhosis. At the other end of the spectrum, the period saw a limited set of discourses on alcohol and the liver from doctors who critiqued the teetotal temperance movement on behalf of their preference for moderation over abstinence. The diverse reception of the debates over cirrhosis aetiology shows that the knowledge on liver disease was an integral part of the alcohol debate during the early twentieth century.

The broadly held framing of cirrhosis as an 'alcoholic's disease' was adopted as part of the temperance movement's understanding that alcohol consumption had nothing but deleterious effects on the body. Unlike the assortment of medical and scientific texts explored

in the previous section, those studied here were either published by temperance organisations or authored by physicians who were explicitly tied to the movement. Many of the educational handbooks and monographs published by groups within the movement presented a narrative that subtly overstated the action of alcohol on the liver. This view was founded on their conceptualisation of alcohol as a 'poisonous' substance that caused immediate harm, an idea directly tied to the 'slippery slope' thesis that formed the ideological groundwork of teetotalism.[92] Based on this belief, temperance publications often provided an account of alcohol's causation as if drink was capable of incapacitating the organ without prolonged indulgence.

Such discourses were apparent in the Band of Hope's pamphlet-sized handbooks that were aimed at young readers. On its specific action on the liver, a volume of the *Temperance Science Lessons* understood alcohol to have a specific mechanism to 'kill numbers of the liver cells, and thus to render the organ less capable of doing its work'. Cirrhosis was described as an outcome of the ardent consumption of spirits, in which 'the liver shrinks and hardens, the outer skin being drawn into furrows with parts sticking up like the hob nails of boots'.[93] Although this particular account of the pathogenesis seems uncontroversial at first, it was then followed by a statement that '[i]t is very doubtful whether a drunkard could be found with a liver in a healthy condition, and it is quite certain that those who are in close contact with strong drink suffer enormously from liver diseases.'[94] This curious passage exemplified the movement's tendency to inflate the negative effects of drink by describing its effects on the liver under a deliberately ambiguous language. First, by using the term 'liver diseases', the text could be referring to the whole spectrum of liver damage that spans from fatty liver to cirrhosis, the former of which is ubiquitous among those who frequently consumes 'strong drink'. Fatty liver, however, is only the earliest stage of liver disease and rarely results in any serious problems, unlike cirrhosis. By stating that drunkards 'suffer *enormously* from liver diseases', the author

[92] Yeomans, *Alcohol and Moral Regulation*, p. 50.
[93] W. R. Edwards, *Temperance Science Lessons No. 2: Physiology, Showing the Effects of Alcohol on the Human Body* (Manchester, 1898), pp. 26-7.
[94] *Ibid.*, p. 27.

referred to 'liver diseases' to connote a set of illnesses that were serious enough to be a mortality risk. This passage thus failed to account for two crucial facts: that not all 'liver diseases' are the same in their capacity to cause harm, and cirrhosis, the highest and the most serious level of alcoholic liver disease, never occurs in no more than a minority of heavy drinkers.

Other volumes of the same series of handbooks presented a similarly obscure, dumbed-down account of alcohol's effect on the liver. A volume titled *"Why Abstain?"* discussed how 'heavy ardent spirit drinkers' often caused 'fatty degeneration' and 'hobnailed' liver.[95] Another handbook from 1905 described the development of liver disease by stating that alcohol had the property to make the liver 'loaded with unhealthy fat' in eventually incapacitating its function.[96] Neither texts were particularly willing to explain that fatty liver, by itself, is not a cause for medical concern, and the description of the pathogenesis of the acute condition implicitly misled the reader to think of it as a serious problem. The emphasis was placed on the harmfulness of alcohol's short-term effects on the liver, as it was additionally noted by W. H. Cologan and Francis Cruise in their educational *Temperance Reader* that the 'nature of the liver' changed even after 'small doses of alcohol', a passage that likewise failed to mention that alcoholic liver disease does not cause serious harm until its later stages.[97]

Other publications from the temperance movement made similar use of disease knowledge to condemn drink. In 1913, the Church of England Temperance Society published *The Voice of Doctors and the Verdict of Scientific Research in Relation to the Use of Alcohol*, an edited compilation of *ad verbum* quotes by professional doctors that were conveniently cherry-picked to lend scientific legitimacy to certain claims derived from the temperance cause.[98] The pamphlet rehashed a series of dogmas associated with the temperance cause with quotes such as: '"[a]lcohol is not essential, not only so, but it is absolutely deleterious to life"', '"there seems to be conclusive evidence that total abstinence is better than moderate drinking"', '"[t]hat

[95] Wilson, *Temperance Science Lessons No. 3*, p. 26.
[96] Wilson, *Temperance Science Lessons No. 5*, p. 28.
[97] W. H. Cologan, and Francis Richard Cruise, *The Temperance Reader* (Belfast, 1902), p. 50.
[98] C. I. Parish (ed.), *The Voice of the Doctors and the Verdict of Scientific Research in Relation to the Use of Alcohol* (London, 1913).

alcohol is necessary in illness and disease, is the greatest of all delusions"', and the most dramatic of all, '"[a]lcohol is the most dangerous chronic form of poison that the human race can make use of."'[99] The book contained a chapter on alcohol as 'a cause of disease', which presented liver disease as one of the numerous harms brought about by alcohol consumption. James Miller, a bacteriologist based in Edinburgh, was quoted for stating that '"[a]lcohol may produce diseases of the liver, it may produce heart disease"', while another quote by David Barcroft, a physician from London, argued that alcohol was responsible for '"gin drinker's liver, the beer drinker's fatty heart, and the spirit drinker's kidney."'[100] Most interestingly, a passage attributed to a town hall address delivered by pharmacologist W. A. Potts stated that even '"small quantities"' of alcohol had '"definite effects, not only on the kidney and liver, but also on the heart and the brain."'[101] The quote overstated the effect of alcohol by presenting the disease as if the '"definite effects"' on the liver caused by the consumption of '"small quantities"' of alcohol were somehow detrimental to one's health. Much like the earlier handbooks published by the Band of Hope, the passage omitted the fact that alcohol cannot seriously harm the liver until after years of heavy drinking. Aside from the likelihood that the pamphlet had deliberately misquoted Potts and other doctors out of context of a larger passage, the minimal references and citations to the origin of the quotes forces the reader to question the authenticity of the book itself.

A more comprehensive account of alcohol's effects on the liver was presented in *A Premier of the Physiological Action of Alcohol* by Edwin J. Norris, a physiologist based in Portsmouth.[102] The monograph was not produced by any specific temperance organisation, but the discourse employed in the content indicated that Norris was at least sympathetic of the movement. The book was intended for younger audiences, arguing that it is important to have 'some knowledge of the structure and functions of the body in health' to 'understand and appreciate the evil effects of alcohol.'[103] In dedicating an entire chapter on the liver, Norris

[99] *Ibid.*, pp. 5, 7, 15.
[100] *Ibid.*, pp. 10-1.
[101] *Ibid.*, p. 10.
[102] Edwin J. Norris, *A Primer of the Physiological Action of Alcohol* (London, 1900).
[103] *Ibid.*, p. v.

provided a detailed account of the pathogenesis of cirrhosis: '[l]ong continued irritation [by alcohol] produces an increase in quantity of the connective tissue, followed by hardening and consequent pressure upon the branches of both the portal vein and the liver cells… The whole liver may, after a long period of irritation by alcohol, become enlarged and its surface irregular.'[104] Although Norris gave a relatively fair account of the pathogenesis of the disease, clearly distinguishing cirrhosis and earlier forms of liver disease and pointing out that the former required the long-term abuse of alcohol, his solution was distinctly characteristic of the cause of teetotalism, stipulating that '[t]he advantages of complete abstinence are more than the simple removal of the cause from the tissues of the liver.'[105] Rather than promoting the moderate consumption of 'sensible' volumes of alcohol, with which the risk of cirrhosis would be minimised, Norris made use of his account of cirrhosis to promote abstinence as the only effective prevention to its pathogenesis.

Among many of the medical temperance texts that have been studied for this chapter, the one publication that stood tall in its legacy was a treatise from 1908 titled *Alcohol and the Human Body: An Introduction to the Study of the Subject, and a Contribution to National Health.*[106] The book was authored by Victor Horsley and Mary Sturge, two prominent members of the medical temperance movement. Horsely, in particular, was known as an authority in neurology who, on separate occasions, served as the president of both the National Temperance League and the British Medical Temperance Association. The book was a noted best-seller, having sold over 20,000 copies by 1915.[107] Much like Norris's monograph, Horsley and Sturge employed a relatively toned-down language when discussing the numerous harms brought about by alcohol on the human body. Nevertheless, the core theses of the book was characteristic of the temperance cause in stating that alcohol 'does not aid the human economy in any way popularly supposed' and that there was ample scientific evidence of the 'occurrence

[104] *Ibid.*, p. 27.
[105] *Ibid.*, p. 28.
[106] Victor Horsley, and Mary D. Sturge, *Alcohol and the Human Body: An Introduction to the Study of the Subject, and a Contribution to National Health* (London, 1908).
[107] David M. Fahey, 'Horsley, Sir Victor (1857-1916)', in Blocker, Jr., Fahey, and Tyrrell (eds.), *Alcohol and Temperance in Modern History*, p. 301.

of actual damage to the structure and functions of the different organs'.[108] Additionally, the book set forth a common temperance trope in arguing that 'it is plain that alcohol cannot… be regarded as a "food"' and was 'always included among the "poisons"… placed side by side with chloroform and ether and described as a narcotic poison.'[109]

Horsley and Sturge provided a more detailed account of alcohol's role in liver damage compared to other equivalent texts tied to the temperance cause. Alcohol, even in its 'moderate amounts', is 'practically entirely absorbed by the stomach' and carried straight to the liver through the blood-vessels on the stomach lining. They went onto state that, 'as the first organ in the path of the absorbed alcohol, we should expect the liver to be most affected by it' and argued that the organ was 'an excellent field for studying the action of alcohol upon cells in general'.[110] Describing alcohol as a 'poison' that deprived the cell of 'its nutrition, its growth, and its power of reproduction', its continued consumption results in '"fatty degeneration"', which the authors exaggeratedly believed made the liver 'incapable of performing the work which it ought to do.'[111] To its credit, the monograph later noted that, if alcohol was 'taken only for a short time' and was 'removed' from consumption, 'the condition of the liver will go back to what it was before irritation occurred'.[112] The continued consumption was said to lead to the proliferation of 'useless scar tissue', culminating in the production of 'the "drunkard's" or "hobnailed" liver'.[113] Liver disease was then understood to 'occur more frequently as a result of the frequent taking of small doses of alcohol… than as a result of indulging more freely'. The authors, however, warned that 'different people are affected in different ways by the action of alcohol' and that 'the irritant action of the alcohol upon the liver has time wherein to manifest itself'.[114] Besides their willingness to overstate of the harmful effects of fatty liver, Horsley and Sturge gave a relatively balanced account of the development of alcoholic liver disease, especially compared to those of the previous temperance texts. Their consideration of the

[108] Horsley, and Sturge, *Alcohol and the Human Body*, p. 4.
[109] *Ibid.*, pp. 13, 230.
[110] *Ibid.*, p. 237.
[111] *Ibid.*, pp. 239-40.
[112] *Ibid.*, p. 241.
[113] *Ibid.*, pp. 231-2.
[114] *Ibid.*, pp. 243-4.

impact of the duration of consumption and individual differences in susceptibility to the toxicity of alcohol was unusual for a temperance medical text, even though such considerations did not make them question the status of alcohol as a definite cause of cirrhosis.

Outside of the temperance movement, liver disease was mentioned on several occasions in the Houses of Parliament over matters concerning the drink question. On 16 March 1906, the House of Commons debated the Pure Beer Bill, which legislated a ban on the adulteration of beer. In it, Conservative MP George Courthrope supported the law based on his understanding that '[t]hey had the evidence of some great doctors that cirrhosis of the liver was caused by the drinking of beer in the manufacture of which sulphates had been used', a passage that curiously attributed liver damage, not to alcohol, but to other substances contained in beer.[115] A House of Lords debate on 25 November 1908 discussed the licensing bill proposed by H. H. Asquith's Liberal government, which was vehemently attacked by many peers for containing many 'provisions… which are known to be distasteful to the majority of the House'.[116] However, Arthur Winnington-Ingram, the sitting Bishop of London and one of the Lords Spiritual serving in the House, was among the minority who supported the bill as 'an old temperance worker for thirty years' who showed 'disgust at the amount of alcohol the British drink'. In making his case, Winnington-Ingram stated that an overly liberalised licensing regime harmed the youth, referencing a 'somewhat sensationally written' book titled *The Black Stain* by George R. Sims[117] that gave an account of 'the number of children whose liver have become hardened by gin given them by their parents in public-houses'.[118] Although it is difficult to confirm the truthfulness of the account of slum-dwelling children who had supposedly developed cirrhotic livers, the Bishop of London's contribution indicated the strong entrenchment of the understanding of cirrhosis as a disease brought about by drink.

On 10 April 1910, the House of Commons was in the midst of a debate over the introduction of the local option bill that allowed municipalities in Scotland to hold a

[115] HC Deb 16 March 1906, vol 153, col 1543.
[116] HL Deb 25 November 1908, vol 197, col 280-370.
[117] Unfortunately, I was unable to get access to this particular book for the purpose of this thesis.
[118] HL Deb 25 November 1908, vol 197, col 281.

referendum over the ban on alcohol licensing.[119] The legislation was enthusiastically supported by William Chapple, a physician and the MP for Stirlingshire, who argued that prohibition would save Scotland from the 'degradation, to the crime, to the disease, and to the premature death of the hundreds and thousands that surround us every day'.[120] To highlight the innumerable harms that resulted from alcohol, Chapple noted that physicians and coroners had the tendency to mistakenly attribute most alcoholic deaths to cirrhosis instead of other illnesses such as peripheral neuritis, Bright's disease, and delirium tremens.[121] This line of criticism received a response from Edward Marshall Hall, a Liberal Unionist MP who accused Chapple of mistakenly believing that cirrhosis was 'due entirely to alcohol'. Marshall Hall questioned 'whether there are any statistics which will prove that', as 'it often occurs in children of two years of age who can hardly have been intemperate'.[122] Indeed, although cirrhosis was most strongly associated with the toxicity of alcohol on the liver, it was known at the time that the disease could be caused by other factors besides alcohol.[123] Chapple irately responded to Marshall Hall, stating that he never intended to say that 'cirrhosis of the liver was due only to alcohol.'[124] Hence, this was clearly a misunderstanding on Marshall Hall's part, as Chapple was merely stated that cirrhosis was abused by medical professionals as a popular explanation of deaths attributable to alcohol to highlight the existence of other negative repercussions of drink. This exchange revealed that the likes of Marshall Hall were sensitive to the exaggerations of the harms of alcohol that were used to justify some of the more radical measures supported by the temperance movement. Such discussions demonstrated of how the knowledge on liver disease was used as a rhetorical device to support certain ideological positions concerning alcohol and its harms on society and the individual.

[119] HC Deb 10 April 1910, vol 16, col 1579-661.
[120] *Ibid.*, col 1642-3; Chapple famously authored a treatise in support of total abstinence, in W. A. Chapple, *How to Impress the Evils of Alcohol: Cases and Comments from a Doctor's Practice* (London, 1911).
[121] HC Deb 10 April 1910, vol 16, col 1642.
[122] *Ibid.*, col 1645.
[123] Julius L. Salinger, and Frederick J. Kalteyer, *Modern Medicine* (London, 1900), p. 521.
[124] HC Deb 10 April 1910, vol 16, col 1645.

At the other end of the spectrum in the politics of alcohol, the DTT was critiqued by a small group of Victorian and Edwardian doctors as part of their opposition to particular medical claims made by the temperance movement. The place of such doctors within the alcohol debate has been thoroughly explored by Kneale and French in their study of the 'Anstie's limit', an early scientific attempt to devise a 'safe' level of drinking.[125] During the 1860s, physician Francis E. Anstie created a metric to separate 'moderate' from 'excessive' drinkers by devising a daily limit up to which alcohol could be consumed without affecting one's longevity. Kneale and French refer to this theory as the forerunner of the conceptualisation of alcoholic 'units' that later emerged as part of the official alcohol guidelines in the 1980s. Anstie himself, who devised the limit as an indictment against the wholesale condemnation of moderation by teetotallers, believed that the human body benefitted from drink up until a certain point before it became a 'narcotic' and a 'depressant' under 'heavy' use.[126] In spite of the vehement opposition by temperance groups, the influence of Anstie's limit reached to such a degree that it was adopted by life assurance offices in Britain and the United States as a metric to quantify the level of consumption that would have been subjected to a higher premium upon the increased risk to mortality.[127]

A small handful of texts suggested that Anstie himself had his own views on liver disease. A column piece from 1872 written by George Johnson, a physician at King's College Hospital, referred to Anstie's 'conviction that the generally accepted doctrine of the intimate relation between alcoholic excess and cirrhosis of the liver, is erroneous', a view where 'few physicians of experience will be found to agree'.[128] In *The Practice of Medicine* (1901), James Tyson similarly remarked that Anstie was often 'disposed to deny that the abuse of alcohol ever produces cirrhosis'.[129] Similar statements that opposed the consensus surrounding alcohol's causation in cirrhosis were later echoed by some of Anstie's intellectual successors. Following

[125] James Kneale, and Shaun French, 'Moderate drinking before the unit: Medicine and life assurance in Britain and the US c.1860-1930', *Drugs: Education, Prevention, and Policy* 22.2 (2015), pp. 111-7.
[126] *Ibid.*, pp. 112-3.
[127] *Ibid.*, p. 114.
[128] George Johnson, 'The Elimination of Poisons', *The British Medical Journal* 1.586 (23 March 1872), p. 311.
[129] Tyson, *The Practice of Medicine*, p. 450.

Anstie's death in 1874, the recognition of the existence of a 'safe' level of alcohol consumption received the support of a handful of physicians, many of whom opposed the temperance belief that alcohol posed an immediate harm to the body. Joseph Mortimer Granville, the famed inventor of the electric vibrator, was one such doctor who took on Anstie's argument that moderate drinking was, in fact, beneficial to health over total abstinence.[130]

Another notable advocate of Anstie's approach was Dyce Duckworth, a medical authority on gout who worked as a consultant at St Bartholomew's hospital and as the personal physician to Edward VII.[131] Duckworth's standing on the drink question could be appreciated in a talk that he delivered in 1893, within which he argued there was 'no evidence to prove that a moderate consumption of alcoholic liquid taken with other food was injurious to the best health of the textures of the human body'.[132] As a fierce opponent of teetotalism, Duckworth echoed Anstie in a lecture delivered in 1907 at the London School of Clinical Medicine by presenting a theory that alcohol never went beyond having an indirect role in harming the liver. Although '[a]lcohol, improperly used, was formerly regarded as acting directly upon the hepatic cells and connective tissues of the liver… [p]athologists assert now that alcohol acts only indirectly on the liver by setting up gastroenteritis in the first instance'.[133] The mechanism was believed to involve alcohol indirectly damaging the liver through its action on the stomach, involving bacterial toxins produced by an alcohol-induced gastroenteritis. Furthermore, perhaps in response to the tendency of other physicians to neglect the impact of the duration of continued alcohol misuse, Duckworth correctly pointed out that cirrhosis did not develop until 'after many years of inordinate use of alcohol'. He stated that the likelihood that cirrhosis developed in heavy drinkers varied widely across individuals, noting that '[a]lcohol is a more toxic agent to some persons than others.'[134]

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[130] Kneale, and French, 'Moderate drinking before the unit', p. 113-4.
[131] Anon., 'Obituary', *The British Medical Journal* 1.3499 (28 January 1928), pp. 161-2.
[132] Quoted in Kneale and French, 'Moderate drinking before the unit', p.113.
[133] Dyce Duckworth, 'A Clinical Lecture on Portal Cirrhosis of the Liver', *The Lancet* 169.4354 (9 February 1907), p. 348.
[134] *Ibid.*, p. 348.

Much of the views that Duckworth presented on cirrhosis in the above passage falls in line with some of the ideas provided by the small handful of sceptical physicians from the previous section who questioned the consensus surrounding the DTT. His attribution of alcohol as an indirect cause of liver damage was virtually identical to what was theorised by Rolleston in 1906.[135] Furthermore, Duckworth's emphasis on the necessity of the long-term abuse of alcohol and individual variations to one's susceptibility to its toxicity was akin to the explanation of why the disease seemingly developed in only a minority of heavy drinkers. Therefore, it would be difficult to argue that his reasonable account of the pathogenesis of cirrhosis was necessarily motivated by his ideological sympathies for Anstie's approach to moderation. At the same time, in terms of the impact that Duckworth's views might have had, it cannot be denied that an endorsement of a theory that branded alcohol as an indirect cause would have potentially encouraged others to question the widely held assumption that alcohol directly harmed the liver. This would have delegitimised the scientific authority of the medical temperance movement and its tendency to overstate the toxic action of alcohol.

In retrospect, the medical knowledge on alcohol and the liver was influential enough to shape aspects of the public discourse on drink in Edwardian society. The temperance movement exploited the medical orthodoxy surrounding alcohol's direct toxicity to the liver to push forward their ideological agenda in highlighting the harms brought about by alcohol on the human body. Many of the temperance ideas on cirrhosis aetiology mirrored the discursive conceptualisation whereby alcohol was labelled a 'poison', a dumbed-down account of alcohol's effects on the liver that ignored the various complexities and nuances that contributed to the development of serious damage. Cirrhosis was also referenced to a lesser degree in parliamentary debates and by a handful of moderationist physicians who critiqued teetotalism. An exploration of the coverage of the public discourses surrounding alcohol and the liver demonstrate that the medical understandings on the causation of cirrhosis were a critical component of the wider political confrontations over drink in the early twentieth century.

[135] Rolleston, 'Alcoholism', in Allbutt, and Rolleston (eds.), *A System of Medicine*, pp. 916-7.

Conclusion

Liver disease manifested itself under numerous contexts within the heightened divisions over the drink question in Edwardian Britain. Many of the most influential medical texts claimed that cirrhosis was primarily caused by alcohol. The DTT was acknowledged by the majority of medical textbooks examined in this chapter, while temperance medical publications took advantage of this literature to strengthen their narrative that portrayed alcohol to have a direct, immediate harm to the body. A small number of physicians, however, challenged this straightforward understanding of alcohol's causation by highlighting the intricacies surrounding the pathogenesis of cirrhosis, some even suggesting that alcohol played nothing more than an indirect role in enabling some other factor to damage the liver. Although most of the medical sources clearly understood alcohol to be directly responsible for cirrhosis, the existence of more sceptical opinions means that the early twentieth century cannot be understood simply under the dominance of the DTT.

Chapter 2

New Moderationism and the Liver in Interwar Britain

Medical opinions on alcohol and its relationship to the liver gradually shifted after the First World War. In the original 1905 edition of *Diseases of the Liver, Gall-bladder and Bile-ducts*, Humphry Rolleston postulated that '[w]ith regard to the question whether alcohol is the cause of cirrhosis, clinical and experimental evidence are *hardly in accord*'.[1] A respected authority in the professional medical world, Rolleston was among the minority of physicians in Edwardian Britain who questioned the widely held assumption that cirrhosis was the direct outcome of the toxicity of alcohol. Rolleston understood that the lack of precise knowledge on the mechanism of alcohol's action on the liver owed to the inconsistencies in the outcomes of several of the experimental attempts to reproduce cirrhosis in animals.[2] He additionally suggested that an additional factor besides alcohol must contribute to the pathogenesis of the disease since many clinical studies had shown that cirrhosis developed in no more than a minority of long-term alcoholics.[3] In the subsequent 1929 edition of *Diseases of the Liver*, Rolleston instead argued that, '[w]ith regard to the question whether alcohol is the cause of cirrhosis, clinical and experimental evidence are *opposed*'.[4] A more dramatic shift had occurred between two separate editions of William Osler's *The Principles and Practice of Medicine*. Unlike the 1901 edition, which labelled alcohol as 'the chief cause of cirrhosis', the 1947 edition stated that 'there is now a considerable volume of evidence pointing to nutritional deficiency

[1] Rolleston, *Diseases of the Liver*, p. 182.
[2] Rolleston, 'Alcoholism', pp. 916-7.
[3] Rolleston, *Diseases of the Liver*, pp. 182-3.
[4] H. D. Rolleston, *Diseases of the Liver, Gall-Bladder and Bile-ducts* (3rd edn, London, 1929), p. 211.

as of greatest importance in the etiology of cirrhosis'.[5] Evidently, the wider conceptualisation of cirrhosis as a disease of alcoholism steadily fell out of favour among many medical professionals during the interwar period. The direct toxicity theory (DTT) was challenged by the emergence of alternative theories whereby the disease culpability of alcohol was either minimised or discredited. As the 1947 edition of Osler's textbook suggested, most observers by the end of the Second World War settled with malnutrition as the most likely cause of the disease. Thus, the interwar period marks the gradual transition from the predominance of the DTT in Edwardian Britain to the eventual ascendancy of the nutritional deficiency theory (NDT) in the 1940s.

This chapter explores the diminishing importance of alcohol within the shared understandings of cirrhosis aetiology and how such changes shaped the wider alcohol debate in interwar Britain. The shifting knowledge on alcohol and liver disease contributed to the emergence of *New Moderationism*, an innovative intellectual approach to alcohol and harm that conceptualised moderate drinking as essentially a harmless activity while simultaneously viewing heavy drinking as a clear detriment to health. New Moderationism owes its legitimacy to the perceived success of the government's controls on alcohol licensing during the First World War. The revelation that widespread drunkenness can easily be mitigated through the imposition of stringent restrictions on the availability of alcohol undermined any attempts to enact national prohibition in Britain.[6] In turn, the temperance movement's support of total abstinence gave way to the preference for promoting moderation to discourage heavy drinking.

These developments went hand-in-hand with efforts to repudiate the medical temperance movement's effort to exaggerate alcohol's capacity to cause bodily harm. In contrast to the general consensus over the DTT in Edwardian Britain, the interwar period was characterised by the existence of several competing theories on the aetiology of cirrhosis. While a growing number of medical professionals increasingly understood that alcohol played an indirect role in harming the liver, many more argued that its relationship to the organ was more

[5] Osler, *The Principles and Practice of Medicine* (4th edn), p. 596; Henry A. Christian, *The Principles and Practice of Medicine, originally written by William Osler* (16th edn, London, 1947), p. 741.
[6] Nicholls, *The Politics of Alcohol*, p. 158.

complicated than was previously supposed. In turn, the scientific decline of the DTT had a profound impact on the public discussions surrounding drink in interwar Britain. The diminished role of alcohol in the pathogenesis of its most recognised associated illness, cirrhosis, was adopted by the New Moderationists to question the temperance movement's overstatement of the dangers of alcohol consumption.

This chapter divides into three sections. First, it sets out the context of the drink question in interwar Britain and explores the theoretical claims of New Moderationism found in many of the key medical texts that framed the approach. The second section traces the changing understandings on alcohol and the liver among the professional medical community, found in medical textbooks and journals. The third and final section studies how such changes were reflected onto the ideas of the interwar moderationists by revisiting the texts explored in the first section of this chapter, many of which made explicit references to newer claims on alcohol's relation to the liver.

The Drink Question in Interwar Britain

Following over half a century of fierce disagreements between the temperance movement and the liquor trade, the politics of alcohol peaked during the First World War with the implementation of some of the most far-reaching regulations ever imposed on alcohol and licensing. The government established the Central Control Board (CCB) in 1915 to safeguard the efficiency of the war effort against the threat of drunkenness, enacting a myriad of regulations on the sale of alcohol in the home front. Outside of the numerous controls that were imposed on licensing, the CCB commissioned a set of scientific studies into the effects of alcohol on the human body. The results of such investigations instigated a major shift in the predominant medical and scientific approaches on alcohol towards what Woiak referred to as the 'new moderationist paradigm'.[7] The term 'moderationist' was first coined in the nineteenth century, used by prohibitionists and teetotallers as a pejorative against temperance agitators

[7] Woiak, '"A Medical Cromwell"', p. 360.

who went short of condemning all forms of drinks by tolerating the moderate consumption of beer.[8] As an alternative approach to alcohol and the body, the 'new moderationist paradigm', or New Moderationism, altered the nature of the alcohol debate by striking a middle ground between the temperance encouragement of total abstinence and the liquor trade's promotion of drink as a health beverage. It opted for a more nuanced view of alcohol and the body that recognised excessive drinking as being detrimental to health while acknowledging that small or moderate quantities of alcohol had little or no negative effects on the body. A column in *The Lancet* aptly summarised the core assumption grounding New Moderationism: '[n]o one would argue that an occasional glass of wine or beer would appreciably shorten life. On the other hand, no one doubts that intemperance shortens life.'[9] Therefore, the approach stipulated that the most effective solution to the problem of drunkenness was the promotion of moderate consumption. As the third section of this chapter explores, it was within this framework where the new knowledge on liver disease exerted its influence on the interwar discourse on alcohol. The scientific abandonment of alcohol's direct toxicity on the liver contributed to the marginalisation of the temperance narrative that exaggerated alcohol's capacity to harm the body.

While Woiak highlights the significance of the new paradigm within interwar medical understandings of alcohol, her article lacks a detailed exploration of the intellectual content of interwar moderationist thought.[10] This section intends to address such shortfalls by providing a theoretical description and analysis of New Moderationism through contents of monographs, journal articles, and government inquiries that contributed to its entrenchment in the interwar alcohol debate. While the liquor trade continued to promote the 'food value' of alcoholic beverages, moderationist assumptions gained traction within the interwar medical establishment as a favourable framework to understand the alcohol problem.

[8] Nicholls, *The Politics of Alcohol*, pp. 100-1.
[9] Anon. 'Alcohol and Longevity', *The Lancet* 203.5250 (12 April 1924), pp. 758-9.
[10] Woiak, '"A Medical Cromwell"', p. 360.

The contextual significance of the First World War in the history of the drink question in Britain cannot be understated.[11] In the immediate aftermath of Britain's declaration of war against Germany on 4 August 1914, the Houses of Parliament passed the Defence of the Realm Act, vastly expanding state power over public and private affairs to support the war effort. Among its immediate reforms, naval authorities were handed the responsibility to regulate opening hours of licensed premises that were in close proximity to Britain's key harbours and shipyards. Concerns over the impact of drunkenness on the homefront was further exacerbated in 1915 by David Lloyd George, who famously declared that '[d]rink is doing us more damage in the war than all the German submarines put together'.[12] This climate of panic led to the establishment of the CCB in May 1915, an independent body of the state entrusted with complete control over the nationwide production and distribution of alcohol.

Among the CCB's efforts to mitigate the disruptive impact of drunkenness on national efficiency, pubs were forced to close before 9:30 pm, customers were prohibited from buying drinks for others, beverages sold at licensed establishments were diluted, and alcohol duties were significantly raised. The CCB was also responsible for building hundreds of canteens in munition factories across the country as recreational spaces intended to rival drinking establishments. As an experiment to alter drinking habits, some pubs were nationalised for spatial renovation as well to allow the provision of cooked food and non-alcoholic beverages. The most peculiar policy of the CCB was the Carlisle scheme, which saw the entire liquor trade of the city in Cumbria, including pubs, off-licences, and breweries, placed under public ownership to micromanage alcohol pricing, licensing hours, and the drinking environment. Within the history of alcohol in Britain, Yeomans describes the wartime era as the 'apogee of the temperance movement' due to how the period saw the implementation of the most all-encompassing set of controls on drink.[13] Alongside other additional factors such as the wartime

[11] The most comprehensive account of Britain's wartime experience with alcohol control would be Duncan's *Pubs and Patriots*. Shorter, but similarly reliable, accounts could be found in Greenaway, *Drink and British Politics*, pp. 91-114; Nicholls, *The Politics of Alcohol*, pp. 150-60; Yeomans, *Alcohol and Moral Regulation*, pp. 97-127.
[12] Quote from Ian Spencer Hornsey, *A History of Beer and Brewing* (London, 2003), p. 581.
[13] Yeomans, *Alcohol and Moral Regulation*, p. 121.

disruption of the production of alcoholic beverages and the growing popularisation of non-alcoholic leisure pursuits, the policies of the CCB were seen to be responsible for the vast reduction in *per capita* levels of alcohol consumption during the war. This in turn led to the reduction in the incidence of drunkenness, mortality from cirrhosis, and other harms associated with drink.[14]

Following the end of the war in 1918, there was a general agreement, outside of some wings of the temperance movement, that Britain should not resort to prohibition.[15] This was especially noteworthy since other nations in the Western world were in the process of implementing a countrywide ban on alcohol during the same period, most notably in the United States with the passage of the Volstead Act on 28 October 1919.[16] The dismissal of prohibition as a solution to the alcohol problem crucially relied on the perceived efficiency of Britain's wartime controls. This sentiment was posthumously expressed in 1928 by Lord D'Abernon, the chairman of the CCB.

> The gain consisted in this: discovery that the drink traffic, so far from being uncontrollable, was eminently susceptible of control; that it could be regulated with precision; that definite results could be predicted with almost scientific accuracy. Contrary to previous experience—in defiance of expectation—it was found that the phenomenon of intemperance could be controlled by skilful legislation; that it could be regulated—even modulated—like the tones of a violin by a virtuoso.[17]

Evidently, the state was increasingly trusted as a body capable of controlling the problem of drunkenness, even within the medical establishment. *The British Medical Journal* reported on a council meeting of the British Medical Association (BMA) that took place on 16 April 1919,

[14] Greenaway, *Drink and British Politics*, pp. 111-2; Duncan, *Pubs and Patriots*, p. 207.
[15] Nicholls, *The Politics of Alcohol*, p. 158.
[16] Among the countries of Northern Europe and North America where temperance movements were influential, Britain, Ireland, and Sweden were exceptions for not undergoing total prohibition.
[17] Edgar Vincent D'Abernon, 'preface', in H. M. Vernon, *The Alcohol Problem* (London, 1928), p. vi.

where it was agreed that 'the medical profession as a whole is not convinced of the necessity for total prohibition'.[18] The declaration, signed by every doctor present at the meeting, further supported the preservation of 'reasonable restrictions' on licensing that did not 'interfere with the habits, customs, and pleasures of the people to an undue extent'.[19] This resulted in a settlement in the form of the 1921 licensing act, which moderated the CCB's controls by maintaining strict licensing hours between 11:30 am to 10:30 pm throughout much of the country, while beverage duties were raised above levels from before 1914.[20] Scotland was the glaring exception to this development. The municipal plebiscites that were promised by the passage of the local option bill of 1913 eventually took place in 1920. In the end, however, only a small handful of rural and suburban jurisdictions ended up voting in support of the ban on licensing, much of which were completely reversed within a matter of a decade.[21] Unsurprisingly, such developments led to the ideology of prohibitionism losing much of its traction during the period.

The ensuing interwar period was unanimously interpreted by historians to be the historical low-point in the intensity of the alcohol debate in modern Britain.[22] The diminishing influence of the temperance movement was signalled by the declining public interest in the alcohol problem. In the face of their dwindling support and membership, various temperance groups fractured into those that accepted the 1921 licensing act and those that continued to push for radical solutions, like the local option.[23] The new settlement was even accepted by the UK Alliance, formerly the vanguard for prohibition in the late Victorian period, alongside other moderate temperance groups that sought to appropriate the CCB's policies as 'temperance measures' and a by-product of their decades of struggle against 'demon drink'.[24]

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[18] Anon., 'Liquor Control', *The British Medical Journal* 1.3045 (10 May 1919), pp. 586-7.
[19] Anon., 'Liquor Control or Prohibition', *The British Medical Journal* 2.3053 (5 July 1919), p. 21.
[20] Greenaway, *Drink and British Politics*, p. 124.
[21] *Ibid.*, pp. 132-3.
[22] Greenaway, *Drink and British Politics*, pp. 130-49; Nicholls, *The Politics of Alcohol*, pp. 180-98. Even Yeomans described the interwar era as a 'point of low tide within efforts to morally regulate alcohol consumption' in *Alcohol and Moral Regulation*, p. 129-59.
[23] Yeomans, *Alcohol and Moral Regulation*, pp. 132-3.
[24] *Ibid.*, p. 120; Woiak, '"A Medical Cromwell"', p. 364.

The moderate consumption of alcohol was increasingly encouraged as the wartime controls were credited for the decline of the overall *per capita* level of alcohol consumption from 10.9 litres in 1900-4 to a meagre 4.2 litres in 1930-4.[25] In his second study of poverty in interwar York, social reformer Seebohm Rowntree confirmed this trend upon his discovery that the working classes were spending a noticeably smaller proportion of their salary on alcohol compared to the Edwardian era.[26] The royal commission on licensing in 1929-1931 observed that 'the present century has seen a distinct advance in sobriety' and that '[d]runkenness has gone out of fashion, and a drunken person is not tolerated as he used to be.'[27]

Within the shift towards a more favourable attitude towards alcohol, New Moderationism emerged as the dominant framework of understanding alcohol and its effects on the body. The origin of interwar moderationism was attributed by historians to the wartime research that were carried out under the sponsorship of the CCB, many of which played an instrumental role in precipitating the shift towards the encouragement of moderation over abstinence after the war.[28] The studies were performed by the members of the board's own scientific advisory committee, a group of experts who informed the CCB on the medical aspects of drink. One of the prominent scientists who joined the committee was H. M. Vernon, a respected industrial physician and psychologist hired to study the influence of alcohol on work efficiency.[29] His contributions included investigations into the impact of alcohol on activities such as typewriting and heavy lifting, from which Vernon observed that consumption, especially of spirits, caused a noted rise in the number of work-related errors and accidents.[30]

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[25] Yeomans, *Alcohol and Moral Regulation*, p. 130.

[26] B. S. Rowntree, *Poverty and Progress: A Second Survey of York* (London, 1941), pp. 360-9.

[27] Anon., *Royal Commission on Licensing (England and Wales) 1929-31, Report* (London, 1932), pp. 8-9.

[28] Woiak, '"A Medical Cromwell"', p. 362.

[29] Thomas Bedford, 'Obituary: H. M. Vernon, M.A., M.D.', *British Journal of Industrial Medicine* 8.2 (April 1951), pp. 96-7. The committee was chaired by D'Abernon and George Newman, the principal medical officer of the Board of Education. It also included pharmacologist A. R. Cushny, biochemist H. H. Dale, statistician M. Greenwood, philosopher W. McDougall, asylum pathologist F. W. Mott, physiologist C. S. Sherrington, and asylum superintendent W. C. Sullivan. Central Control Board, *Alcohol: Its Action on the Human Organism* (New York, 1918), pp. i-ii.

[30] Anon., 'Conference of the Society for the Study of Inebriety', *The British Medical Journal* 2.3069 (25 October 1919), p. 534.

Another leading member of the committee was Edward Mellanby, a nutritional scientist who later discovered rickets to be an illness caused by the deficiency of vitamin D.[31] Mellanby performed a wide variety of studies on blood-alcohol content, examining the effects that varying volumes of alcohol had on hand-eye coordination and physical exercise.[32] In demonstrating how food seemingly slowed down the absorption of alcohol in the body, Mellanby was responsible for providing scientific evidence to the widely held dictum that one should not drink 'on an empty stomach'. He additionally noted that the rate of absorption decreased when alcohol was consumed in the form of beer over spirits.[33] Both Vernon and Mellanby's studies marked a crucial distinction between certain forms of alcohol consumption based on the outcomes produced. Against the blanket condemnation of all drinking by the temperance movement, the work of the scientific advisory committee pushed the CCB to tackle certain kinds of drinking habits that were deemed to be 'harmful' over others.

The committee's work culminated in the publication of a report titled *Alcohol: Its Action on the Human Organism*, a book-length literature review of the existing medical knowledge on alcohol aimed at a public audience.[34] Partially legitimised by its association with the CCB and its success, the report enshrined some of the core assumptions that formed the basis of interwar moderationist thought. First, the book labelled the 'food versus poison' debate, a key intellectual battleground that divided the temperance movement and the liquor trade, as a false dichotomy, arguing that alcohol can both be a food *and* a poison and that the two are not mutually exclusive from one another.[35] While acknowledging that the caloric content of alcohol allowed it to be classified as a 'fuel' under some circumstances, the report recognised that its food value was restricted by the fact that it would have to be consumed to the point of causing harm for it to have any benefits. The authors simultaneously recognised alcohol as also a

[31] B. J. Hawgood, 'Sir Edward Mellanby (1884-1955) GBE KCB FRCP FRS: nutrition scientist and medical research mandarin', *Journal of Medical Biography* 18.3 (August 2010), pp. 150-7.
[32] Edward Mellanby, *Alcohol: Its Absorption into and Disappearance from the Blood under Different Conditions* (London, 1919).
[33] Anon., 'Conference for the Society', p. 534.
[34] Central Control Board, *Alcohol.*
[35] *Ibid.*, p. 2.

'poison' when consumed in excess, possessing a 'devitalising action on the tissues' that varied from 'relatively moderate impairment of the normal state of the organs to gross morbid changes which ultimately cause death'.[36] This assumption discursively established a crucial distinction between short-term, or acute, alcoholic harm, and long-term, or chronic, harm. This differentiation was seldom recognised by the temperance medical texts that had existed before the war, many of which labelled alcohol as a 'poison' without considering the benignity of its moderate consumption. In doing so, such texts exaggerated the harms of all forms of consumption to encourage total abstinence as the only viable alternative to alcoholism.[37] The question, therefore, was not to ask whether one should drink or not, but to ask how and how much one should drink. The report's recognition of alcohol as both a food and a poison was a reasonable objection to a highly inaccurate discursive binary that had characterised the debate between the temperance movement and the liquor trade. Indeed, alcohol *can* theoretically be understood as a 'food', albeit a very inefficient one in providing energy, while it is also a 'poison' for its capacity to bring about harm to the body when consumed in excess (a principle that similarly applies to practically all substances fit for human consumption, including fresh water).

Concerning the acute effects of alcohol, the CCB report stipulated that 'apart from the continued excessive use, the main effects of alcohol that have any real significance are due to its action on the nervous system'.[38] This statement was intended to counter another set of existing assumptions on drink. First, it challenged the temperance understanding of the 'poisonous' action of alcohol in which drink was seen to pose immediate harm to the body, even after a single session of heavy drinking. Second, it dismissed the belief in the 'stimulating properties of alcohol' promoted by the liquor trade as being 'purely subjective origin and illusory' owing to the 'sedative' effects that alcohol seemed to have on the nervous system.[39] Along with the food value of drink, the description of alcohol as a 'stimulant' was heavily promoted by the liquor trade. One pre-war slogan for Bass No. 1 Barley Wine promoted the

[36] *Ibid.*, p. 94.
[37] Anon., 'A Medical Temperance Manifesto', p. 170; Scharlieb, 'The National Temperance League', p. 328.
[38] Central Control Board, *Alcohol*, p. 125.
[39] *Ibid.*, p. 125.

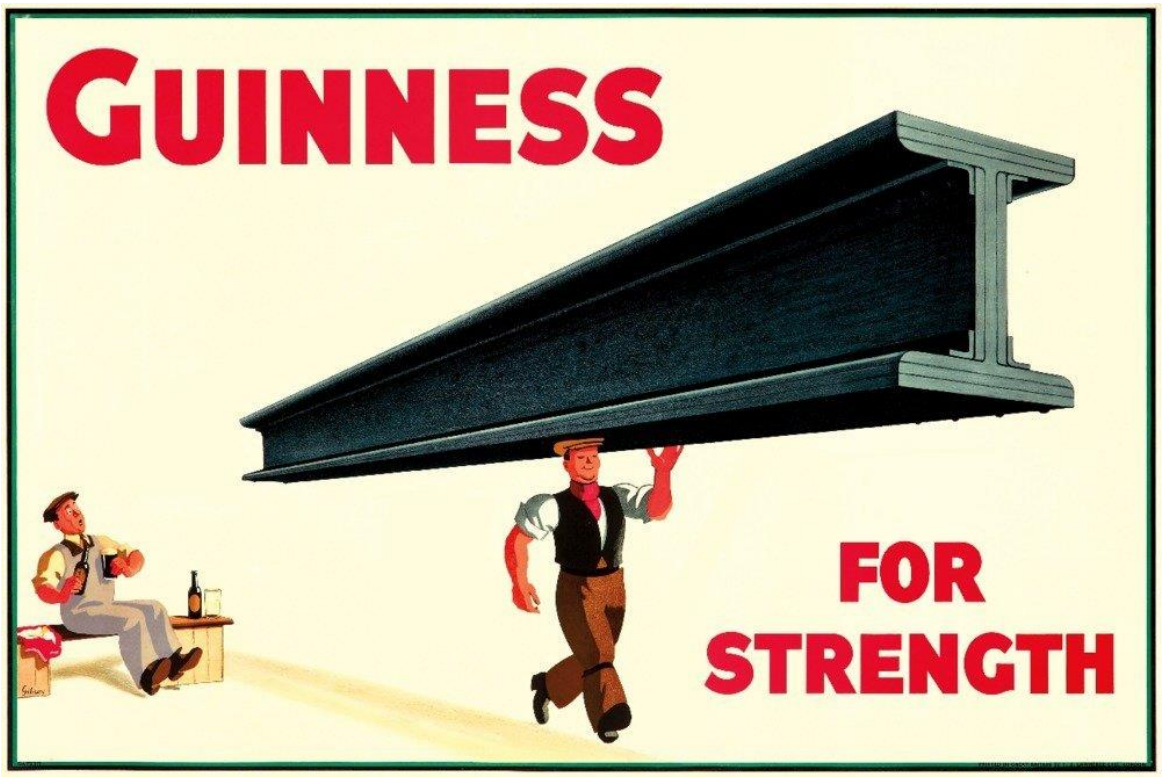
beverage for being 'quality, stimulating, nutritious, and invigorating'.[40] Another understanding of alcohol relating to its supposed nutritional qualities concerned its value as an energy drink. This characterisation of certain alcoholic drinks as an aid to physical exercise and labour reverberated into the interwar period, a trope most commonly associated with Guinness, which outspent every other beverage producer in its advertising campaigns.[41] Alongside the well-known slogan, 'Guinness is good for you', the company distributed adverts such as the those depicting a construction worker lifting a steel frame several times the size of his body after finishing a glass of the beverage (see Figure 3).[42] The CCB report thus discredited these medical tropes in arguing that alcohol's 'habitual use as an aid to work is physiologically unsound'.[43] This assertion was largely dependent on Vernon's research on alcohol's impact on physical exercise, whereby alcohol was seen to be 'not only useless or even detrimental in immediate effect, but is also likely to be, in its ultimate results, seriously injurious to health.'[44] This was based on his observation that alcohol, a 'sedative and narcotic drug', negatively influenced the drinker's coordination.[45] As a result, the report concluded that 'the ordinary use of alcohol should not only be moderate but should also be limited to the consumption of beverages of adequate dilution, taken at sufficient intervals of time to prevent a persistent deleterious action on the tissues'.[46]

The CCB's *Alcohol: Its action on the human organism* left its mark by radically altering the nature of the alcohol debate by establishing certain assumptions that formed the foundations of New Moderationism. While the report refrained from explicitly *promoting* moderate drinking, affirming that alcohol was 'in no way necessary for healthy life', it indirectly

[40] Burnett, *Liquid Pleasures*, p. 172; The National Brewery Centre, Scrapbook 89.1430.00, pp. 76-7.
[41] In 1933, Guinness spent £115,000 on adverts, while Bass came in second only with £54,000. Jonathan Reinarz, 'Advertising (United Kingdom)', in Blocker, Jr., Fahey, and Tyrrell (eds.), *Alcohol and Temperance in Modern History*, pp. 4-6.
[42] Victoria & Albert Museum, Prints & Drawings Study Room, level C, case Y, shelf 67, box 5, 'Guinness for Strength'.
[43] Central Control Board, *Alcohol*, p. iv.
[44] *Ibid.*, p. 130.
[45] Vernon, *Alcohol Problem*, pp. 157-8.
[46] *Ibid.*, p. iv.

recognised the pleasures of mild intoxication as a positive outcome of moderate consumption.[47] This phenomenon was part of the wider scientific tendency in the interwar period to recognise the pursuit of pleasure as a facet of human nature, due in part to the popularisation of psychoanalysis and its normalisation of sexual pleasure.[48] Indeed, the acknowledgement that alcohol consumption can benefit the drinker was a marked departure from the wholesale condemnation of drink by the temperance movement.

FIGURE 3 'Guinness for Strength', c. 1934



The conceptual development of New Moderationism continued after the end of the war. In 1923, Ernest H. Starling, a physiologist at the University College London, was suggested by the Royal Society of Medicine to author *The Action of Alcohol on Man* as an update to the CCB's report on the existing knowledge on alcohol and the body.[49] The book was politically charged from the outset, touting the CCB's control policies as the most effective deterrent to

[47] *Ibid.*, p. 132.
[48] Valverde, *Diseases of the Will*, p. 97.
[49] Ernest H. Starling, and Robert Hutchinson, *The Action of Alcohol on Man* (London, 1923).

drunkenness while simultaneously dismissing prohibition as 'a mistake and contrary to the permanent interest of the race.'[50] Starling channelled the central assumption of the CCB report, arguing that '[i]n moderation it is difficult to appreciate any harmful effect from its use, whereas when temperance is abandoned and alcohol is used immoderately, its effects are evil and fraught with disaster to the individual and damage to the community.'[51] The book proceeded to acknowledge the pleasurable aspects of drink by stating that alcohol possessed an 'indirect advantage to a man in contributing to a happy and healthy existence.'[52] It went on to describe how moderate drinking enhanced 'the man's sense of membership of the society' where 'the individual feels himself more kin with his fellow-men' under 'the operation of the spirit of charity, with its fruits of love, joy or pity'.[53] Starling's book was intriguing in how it framed alcohol as a Janus-faced object. In one instance, the 'immoderate' use of alcohol was labelled as an 'evil', while another associated its moderate use with health and contentment and a 'distinct advantage to the community as a whole'.[54] Again, New Moderationists believed that the important question was to ask *how* one ought to drink, not whether one should drink or not. Starling's double-sided depiction of drink was later reiterated by Bertrand Dawson, the personal physician to the royal family, who stated that alcohol 'might be a narcotic, but in moderation it added to the pleasure, the exhilaration, the happiness, and the gaiety of life'.[55] In a lecture delivered at the Royal Society of Medicine, physician W. E. Dixon endorsed the moderationist approach to drink by stating that, while alcohol was harmful to a minority of drinkers, for the majority, 'alcohol added to the joy and general agreeableness of life.'[56] The defining feature that set these discourses apart from many of the Edwardian medical texts on alcohol was the understandings that the pleasures derived from moderate consumption was

[50] *Ibid.*, p. v.
[51] *Ibid.*, p. 168.
[52] *Ibid.*, p. 140.
[53] *Ibid.*, pp.153-4.
[54] *Ibid.*, p. 155.
[55] Anon., 'Medical Notes in Parliament', *The British Medical Journal* 2.3265 (28 July 1923), pp. 157-8.
[56] W. E. Dixon, 'Alcohol: Its Use and Abuse. Lady Priestley Memorial Lecture', *The British Medical Journal* 1.3295 (23 February 1924), pp. 341-3.

essentially self-justifying in confirming its positive value, independent to their potential benefits to bodily health.

Much like the CCB's report, *The Action of Alcohol on Man* acknowledged that alcohol was 'without a doubt a food', but an insufficient one since it would have to be consumed in morbid excess for it to have any dietary benefits.[57] Furthermore, the sedative, narcotic properties of drink was highlighted over its food value, arguing that the popular belief that 'industrial drinking' was an aid to manual labour was 'founded on a pernicious illusion.'[58] It cited recent experiments that had shown that, although alcohol can be utilised as a food in the short term, it would ultimately be oxidised at the expense of physical energy.[59] Thus, the monograph concluded that alcohol is not 'good or bad in general', but that 'they have their value in their proper dose and appropriate conditions.'[60] The dismissal of alcohol as an efficient source of nutrition was complemented by the simultaneous rejection of the temperance trope that alcohol possessed a poisonous action on the body when consumed, even in small doses.

By the 1930s, the influence of New Moderationism was felt even at the highest levels of government. In 1929, Ramsay MacDonald's second Labour government appointed a royal commission into the alcohol problem in Britain. The report of the commission, published in 1932, positively attributed the recent decline of alcohol consumption and drunkenness to the control policies of the CCB and the 1921 Licensing Act, recognising how the experience of the war increased the public's faith in the ability of the state to control the problem of intemperance.[61] The report also reaffirmed many of the assumptions that were initially promoted by the CCB's *Alcohol: Its Action on the Human Organism*, a book acknowledged by the commission for being 'frequently referred to in the evidence and which appears to be accepted on all hands'.[62] The authors agreed that moderation should continue to be

[57] Starling, and Hutchinson, *Action of Alcohol on Man*, pp. 169-70.
[58] *Ibid.*, p. 170.
[59] *Ibid.*, pp. 60-70.
[60] *Ibid.*, p. 172.
[61] Henry Carter, 'The Drink Problem in Great Britain', *Annals of the American Academy of Political and Social Science* 168 (1932), p. 199.
[62] Anon., *Royal Commission*, p. 14.

encouraged instead of abstinence, arguing that the idea that moderate consumption was harmful to longevity was controversial at best, while the food value of alcohol was rejected in a statement that alcohol has 'no advantage over any other substance possessing the same properties'.[63] This conclusion was reiterated in the *Handbook of Suggestions on Health Education* by the Board of Education in 1934, which recommended that 'for practical everyday purposes alcoholic beverages cannot be regarded from a health point of view as a source of nourishment'.[64] The pleasurable qualities of drink was also recognised by the commission, stating that alcohol, 'temperately used, may have a legitimate value in causing relaxation of mental tension after a period of strain or worry'.[65] The CCB's scientific legacy is noted by how every principle that grounded the New Moderationist approach were disseminated and articulated to policymakers.

Although New Moderationism sought to dispel the purported dietary benefits of alcoholic beverages, other sources indicated that the belief nevertheless persisted in British society. This was the result of the concerted effort by some brewers and distillers to safeguard the health-promoting reputations of their products in response to the long-term financial decline of the beverage industry. Among average consumers, alcoholic beverages dropped from being the second to the fifth largest component of household expenditure between 1900 and 1949.[66] As a response, many of the largest brewers consolidated into larger conglomerates to survive the austere controls that were imposed on the sale of alcohol during and after the First World War.[67] Under the interwar licensing regime, prominent brewers such as Sydney Nevile and W. Waters Butler, both of whom previously belonged to the board of the CCB, widened the social appeal of the pub to a more middle-class clientele by reforming the traditional pub from a 'vilified drinking den' to a 'respectable social space' by providing more space, seating,

[63] *Ibid.*, pp. 16-8, 23.
[64] Board of Education, *Handbook of Suggestions on Health Education* (London, 1934), p. 47.
[65] *Ibid.*, p. 15.
[66] Ron Weir, 'Rationalization and Diversification in the Scotch Whisky Industry, 1900-1939: Another Look at "Old" and "New" Industries', *The Economic History* Review 42.3 (1989), p. 379.
[67] Nicholls, *The Politics of Alcohol*, p. 192.

and lighting.[68] The attempt by pub reformers to engineer 'sensible' drinking habits among pub-goers was emblematic of the spirit of New Moderationism. This development informed the collective decision within the liquor trade to promote the quality and the brand recognition of their products rather than by increasing the volume of sales.[69]

The liquor trade expanded their advertising campaigns in promoting the invigorating and nutritional qualities of alcoholic beverages to a much larger scale than during the Edwardian era. Besides the enormous success of the slogan 'Guinness is good for you', the Brewer's Society co-opted the 'beer is best' collective advertising campaign to counteract falling beer sales. The Society was formed in 1904 as a coalition of the London Brewers' Association, the Country Brewers' Society, and the Burton Brewers' Association, quickly emerging to become the most powerful trade group representing the British brewing industry. Since its foundation, the Society actively campaigned to defend the interests of brewers, particularly in the realm of public relations.[70] Sir Edgar Sanders, the chairman of the Society at the time, believed that it was crucial to 'get the beer-drinking habit instilled into thousands, almost millions, of young men who do not at present know the taste of beer'.[71] As one of the largest cooperative advertising schemes in Britain at the time, the 'beer is best' campaign was launched in 1933 through the nationwide distribution of newspaper adverts, posters, beermats, and music.[72] Its most notable aspect was the promotion of the purity of the ingredients and the health-giving properties of beer. A few illustrative examples of adverts produced by the campaign reiterated the claim that beer was an invigorating beverage that aided one's 'digestion', 'energy', 'appetite', and 'vitality'.[73] The beverage was also increasingly associated with physical fitness and manual labour, as shown in the set of adverts from the 1930s that

[68] David W. Gutzke, *Pubs and Progressives: Reinventing the Public House in England, 1896–1960* (DeKalb, IL, 2005).
[69] Greenaway, *Drink and British Politics*, p. 114.
[70] Ian Donnachie, 'Brewers' Society (BS)', in Blocker, Jr., Fahey, and Tyrrell (eds.), *Alcohol and Temperance in Modern History*, pp. 111-2.
[71] Nicholls, *The Politics of Alcohol*, p. 191. Isaac Foot, a Liberal MP and the father of later Labour leader Michael Foot, compared Sanders to Hitler for hampering down on the 'free press' with his mass advertising campaign, in *Blood Money? An Open Letter to Sir Edgar Sanders* (London, 1933).
[72] Reinarz, 'Advertising', p. 5; Brewers' Society Collection, MSS.420/BS/6/3/3, 'The Brewers' Society Collective Advertising Campaign', 6 February 1952.
[73] 'Beer is Best', *Daily Mail*, 6 February 1934, p. 5.

featured cricketers, and track and field athletes, both of which were accompanied by the claim that beer was a 'powerful source of energy' (see Figures 4).[74]

FIGURES 4 'Beer is Best', *Daily Mail*, 21 August 1934, and 27 February 1935

[74] 'Beer is Best', *Daily Mail*, 21 August 1934, p. 5; 'Beer is Best', *Daily Mail*, 27 February 1935, p. 5; 'Beer is Best', *Daily Mail*, 10 June 1935, p. 3.

Distillers additionally made extensive use of medical and dietary tropes to promote their products. White Horse whisky was advertised on its supposed benefits to health, with one advert suggesting that '[y]ou should always have a bottle of White Horse in your medicine chest' since there 'is no happier prescription for the refreshment of a tired body and mind at the end of the day'.[75] Gordon's Gin was promoted for its 'important properties which are considered by the medical profession to be most beneficial', claiming that its regular consumption made one 'feel braced and energetic' while clearing 'the system of impurities'.[76] Gordon's Orange and Lemon Gin were similarly purported to be a 'wholesome drink' that contained 'the essential vitamins only fresh fruit can give you'.[77] In many instances, beer and spirits advertisements

[75] 'White horse whisky', *Daily Mail*, 7 November 1931, p. 11.
[76] 'The 3 Weeks health plan', *Daily Mail*, 11 June 1936, p. 13.
[77] 'Display Ad 4', *The Manchester Guardian*, 10 December 1935, p. 5

were not promoting the healthiness the substance of alcohol itself. Each individual drink was advertised on the healthful qualities of the particular congeners, or chemicals that accompanied the ethanol in alcoholic beverages.

The food value of alcohol generally received a positive reception from the wider public. Various parliamentary debates at the time show evidence of the persisting belief in alcohol as a food among MPs, mostly within the Conservative Party. In 1923, Arthur Holbrook dismissed the attempt by Edwin Scrymgeour to introduce prohibition in Scotland by stating that alcohol was 'regarded as a food' by most people, while Reginald Applin declared in a separate debate that alcohol 'is very much better and more wholesome than many of the foods that are largely consumed.'[78] Lord Sydney Arnold, a radical member of the Liberal Party with sympathies to the temperance cause, critically remarked on how slogans such as '"Guinness is good for you"… has been so drilled into the public mind that a widespread illusion has been created that stout has some beneficial qualities not to be found in other intoxicating liquors.'[79]

The popularity of the belief in the health-giving properties of beer was explored further by Mass-Observation in *The Pub and the People*, an anthropological investigation into the general attitudes toward alcohol shared among residents of Bolton.[80] The observers noted the widespread circulation of posters, showcards, and billboards throughout the town containing slogans such as 'beer is best' and 'Guinness is good for you'. Such slogans were interpreted by the researchers to have played an instrumental role in shaping the 'mental attitude to their beer' among the town's residents.[81] The researchers also conducted a survey of drinkers to understand the most common motivations behind the decision to drink beer. 52 per cent of the respondents stated reasons to do with health, such as its 'beneficial effect in connection with work', 'nourishing' qualities, 'vitamins', and 'general health-giving properties'.[82] One resident believed that beer 'keeps your body in good health', while another purported to drink beer

[78] HC Deb 20 April 1923, vol 162, col 2487; 31 March 1933, vol 276, col 1365.
[79] HL Deb 28 March 1935, vol 96, col 415-6.
[80] Mass-Observation, *The Pub and the People: A Worktown Study* (London, 1943).
[81] *Ibid.*, pp. 26, 44.
[82] *Ibid.*, p. 42.

'because I cannot eat it.'[83] Evidently, the influence of the food value of alcoholic beverages prevailed within certain sections of the general public, in spite of the effort by the New Moderationists to dispel such beliefs.

In summary, the emergence of New Moderationism was a defining intellectual development within the drink question during and after the First World War. This section supplements Woiak's earlier introduction of the concept by providing a detailed overview and analysis of interwar moderationist thought through several of the key texts that had contributed to its establishment.[84] The polarised debates over drink between the temperance movement and the liquor trade during the Edwardian period was put to rest under the establishment of a middle ground that promoted the idea that alcohol can be both harmless and harmful depending on how it is consumed. Alcohol was increasingly understood to be both a 'food' and a 'poison' under this assumption, while later volumes highlighted the pleasurable qualities of drink, something that was seldom recognised by medical texts until then. Hence, New Moderationist ideas was a defining feature of the public discussion over alcohol following the First World War

Medical Understandings of Alcohol and the Liver after World War I

The medical knowledge on cirrhosis underwent notable changes after the First World War. Whereas most Edwardian physicians took cirrhosis for granted as an 'alcoholic's disease', most of the medical literature in interwar Britain began to question the DTT. This shift was triggered by a number of factors, including the growing understanding that cirrhosis was prevalent in no more than a minority of heavy drinkers and that previous laboratory studies had failed to provide consistent results in their attempts to reproduce cirrhosis in animals. While Herd correctly describes this shift as a process whereby '[t]he role of alcohol as a direct liver toxin was de-emphasized and increasing attention was paid to other etiological factors', her paper takes the transition from the DTT to the NDT at face value without providing an account of

[83] *Ibid.*, pp. 26, 42.
[84] Woiak, '"A Medical Cromwell"', p. 360.

what transpired in between.[85] The scepticism towards alcohol's role as a direct cause of liver damage manifested itself in the form of alternative theories that attributed the disease to other toxic agents, while others explained the close association between cirrhosis and alcohol by relegating the substance as an indirect cause, or an enabler, of other factors that directly harmed the liver. Although the culpability of alcohol was indeed downplayed, the continued ambivalence among many of the texts, refusing to give a conclusive aetiological claim or to provide a full account of the mechanism of pathogenesis, is indicative of how fragmented the understandings of alcohol and the liver were. Thus, the interwar period was characterised by the absence of a clear consensus over the exact cause of cirrhosis. Various aetiological theories of cirrhosis aside from the DTT were liberally discussed within textbooks of general medicine and general medical journals.

For this section, a total of 21 editions of ten different textbooks have been examined, spanning between 1915 and 1940 (see Figures 5; see Appendix: Textbooks of General Medicine for long-term shifts). They differed quite significantly from the Edwardian era in that there was a lack of a single explanation of the aetiology of cirrhosis that was generally accepted by a majority of the authors. Although 71 per cent (10 out of 14 textbooks) of the publications from between 1900 and 1914 confidently attributed alcohol as the direct cause of cirrhosis, the number fell to 38 per cent (8 out of 21) between 1915 and 1940. Whilst agreeing that cirrhosis was ultimately caused by alcohol, the proportion of textbooks that recognised the intricacies surrounding its aetiology witnessed a slight increase from 21 per cent (3 out of 14) to 29 per cent (6 out of 21) between the two periods. Most strikingly, the proportion of textbooks that explicitly referred to alcohol as an indirect cause of cirrhosis on behalf of some other factor rose from 7 per cent (1 out of 14) to 33 per cent (7 out of 21). Evidently, the notion that alcohol posed a direct harm to the liver clearly fell out of favour among most textbooks during the period.

FIGURES 5 Textbooks of General Medicine, 1916~1940

[85] Herd, 'Ideology, history and changing models', p. 1117.

	1916~20	1921~25	1926~30
James M. Anders, *A Text-book of the Practice of Medicine*		14th (1922) Alcoholism is a 'causative factor operative in nearly all cases' but the 'influence of alcohol is undoubtedly exaggerated. Experimentally it is impossible to reproduce the picture of cirrhosis by feeding animals with alcohol in large amounts over long periods of time.'	
William Osler, *The Principles and Practice of Medicine*	9th (1920) Cirrhosis due to 'toxic action of alcohol'		12th (1930) identical claim to 9th edn
Frederick Taylor, *A Manual of the Practice of Medicine*		12th (1922) alcohol responsible for the 'great majority of cases' of cirrhosis, but some have downplayed the role of alcohol	
Alexander Wheeler, *Handbook of Medicine*	6th (1920) 'Alcohol is not the direct cause of cirrhosis, its specific action on the liver being to produce fatty change, but it lowers the resistance of the liver to the action of other poisons, or possibly even of micro-organisms, which are then free to set up connective tissue proliferation.'		8th (1927) identical claim to 6th edn
Thomas Dixon Savill, *A System of Clinical Medicine*	5th (1918) 'cirrhosis of the liver must still be regarded as mainly the result of alcoholic excess' and 'Alcohol is undoubtedly the most usual cause of atrophic cirrhosis'		8th (1930) While alcohol is 'undoubtedly the most usual cause of atrophic cirrhosis... Alcoholic excess is now known to be only one of the causes of cirrhosis of the liver. Syphilis is in some cases a predisposing factor, and so are many bacterial infections.'
Russell Cecil, *A Textbook of Medicine*			1st (1927) in spite of recent attempts to come up with other explanations, 'very strong clinical opinion still points to *alcohol* as the chief etiological factor. This poison may act directly on liver cells...'
Frederick W. Price, *A Textbook of the Practice of Medicine*		1st (1922) 'large majority of patients... have indulged excessively in alcohol' but also caused by 'excessive indulgence in highly seasoned foods'	
John Conybeare, *A Textbook of Medicine*			1st (1929) Alcoholism 'by far the most important, though by no means the only aetiological factor', possibly directly caused by chronic gastritis indirectly caused by alcohol

	1931~35	1936~40
William Osler, *The Principles and Practice of Medicine*		13th (1938) Cirrhosis 'occurs most commonly' in whiskey, gin, and brandy drinkers'
Frederick Taylor, *A Manual of the Practice of Medicine*		15th (1936) 'great majority' of cirrhosis 'dependent, wholly or in part, upon the excessive use of alcohol, but '[w]idest individual differences' in dosage required to produce the disease
Alexander Wheeler, *Handbook of Medicine*		10th (1937) identical claim to 4th edn
Thomas Dixon Savill, *A System of Clinical Medicine*	11th (1939) identical claim to 8th edn	
Russell Cecil, *A Textbook of Medicine*		4th (1938) identical claim to 1st edn
Frederick W. Price, *A Textbook of the Practice of Medicine*	4th (1934), 'It is probable, therefore, that alcohol produces cirrhosis of the liver indirectly by leading to gastroenteritis' but '[a] s ordinary gastroenteritis does not lead to cirrhosis, an additional factor must be present. This is probably the direct poisonous action of alcohol and occasionally of other toxins'	5th (1937) identical claim to 4th edn
John Conybeare, *A Textbook of Medicine*		4th edn (1939) identical claim to 1st edn
G. E. Beaumont, *Medicine Essentials for Practitioners and Students*	1st (1932) alcohol 'undoubtedly is a factor of great importance' though nature of irritant is not certain in all cases	
Derrick Dunlop, *Textbook of Medical Treatment*		2nd (1940) alcohol 'still by far the commonest cause of hepatic cirrhosis. Arguments as to how it acts do not concern us here.'

All five of the textbook series from chapter 1 were re-released during the interwar period as new editions. The changes in the language on alcohol and the liver across these volumes were subtle but significant. The 1920 and 1930 editions of Osler's renowned *Principles and Practice of Medicine* continued to identify cirrhosis as a disease caused by the 'toxic action of alcohol' in the same vein as how the 1912 edition argued that alcohol '[p]roduces definite changes in the liver'.[86] The 1938 edition, however, refrained from explicitly labelling alcohol as the cause of the disease by pointing out instead that cirrhosis 'occurs most commonly' in spirit drinkers, a likely response to the wider re-assessment of alcohol's direct causation.[87] The 1918

[86] Osler, and McCrae, *The Principles and Practice of Medicine* (8th edn), p. 397; William Osler, and Thomas McCrae, *The Principles and Practice of Medicine* (9th edn, London, 1920), p. 598; William Osler, and Thomas McCrae, *The Principles and Practice of Medicine* (12th edn, London, 1930), p. 566.
[87] Henry A. Christian, and Thomas McCrae (eds.), *The Principles and Practice of Medicine* (13th edn, London, 1938), p. 711.

edition of Savill's *System of Clinical Medicine* restated the sentiment of the original 1903 edition, that alcohol was 'undoubtedly the most usual cause of atrophic cirrhosis'.[88] This assertion was subtly revised in the 1930 edition, which designated alcohol to be 'only one of the causes of cirrhosis' alongside syphilis and other bacterial infections.[89] The 1922 and 1936 editions of Taylor's *Manual of the Practice of Medicine* continued to agree with its prior volumes that, while the theory of alcohol's direct toxicity remained controversial, it was safe to assume that cirrhosis is an outcome of drink.[90] There were, however, two clear anomalies. The 1912 edition of Wheeler's *Handbook of Medicine* was the sole exception among textbooks predating the First World War to label alcohol as an indirect cause, stating that alcohol 'is not the direct cause of cirrhosis' and that it only functioned to lower 'the resistance of the liver to the action of other poisons'.[91] The same argument was replicated throughout the subsequent three editions published after 1915.[92] Similarly, whereas all three of Anders's *Text-book of the Practice of Medicine* from before 1914 argued that 'by the side of alcoholism all other causes combined are comparatively insignificant', the 1922 edition was revised to state that the 'influence of alcohol is undoubtedly exaggerated' because cirrhosis was experimentally 'impossible to reproduce' in animal subjects.[93] The existence of these revisions in many of the new editions reflect a marked shift from the belief in cirrhosis as an 'alcoholic's disease' towards the acknowledgement of the complexity underlining alcohol's relationship with the liver.

The end of the First World War ushered in an 'era of multi-authored texts' in medical textbooks.[94] Aside from G. E. Beaumont's *Medicine Essentials for Practitioners and Students*, widely recognised by reviewers as 'one of the last major textbooks' to be written by a single

[88] Thomas Dixon Savill, *A System of Clinical Medicine* (5th edn, London, 1918), p. 370.
[89] Thomas Dixon Savill, *A System of Clinical Medicine* (8th edn, London, 1930), p. 388.
[90] E. P. Poulton, C. Putnam Symonds, and H. W. Barber, *Taylor's Practice of Medicine* (12th edn, Toronto, 1922), p. 458; E. P. Poulton, *Taylor's Practice of Medicine* (15th edn, London, 1936), p. 394.
[91] Jack, *Wheeler's Handbook of Medicine*, p. 212.
[92] John Henderson, and D. M. Dunlop, *Wheeler and Jack's Handbook of Medicine* (10th edn, Edinburgh, 1937), p. 287.
[93] Anders, *A Text-book of the Practice of Medicine* (4th edn), p. 889; Anders, *A Text-book of the Practice of Medicine* (8th edn), p. 927; James M. Anders, *A Text-book of the Practice of* Medicine (11th edn, London, 1913), p. 933; James M. Anders, and John H. Musser, *A Text-book of the Practice of Medicine* (14th edn, London, 1922), p. 892.
[94] Matthews, 'Osler Oration', p. 308.

author and an 'essential volume for medical students', four out of five of the new volumes embodied the shift towards further specialisation in the medical profession by designating the authorship of individual chapters and sections on specific systems, organs, and illnesses to the most knowledgeable experts in their appropriate fields.[95] Russell Cecil's *Textbook of Medicine* was renowned for being 'the best great textbook of medicine in the English language', 'a companion to countless physicians throughout the world', and '[u]p to now among the most popular postgraduate texts for the British physician'.[96] Frederick W. Price's *Textbook of the Practice of Medicine* was another volume known for its quality, considered by reviewers to be 'one of the foremost textbooks of its kind in this country', a 'standard medical reference for most physicians trained in Britain', and one which 'finds most favour with both undergraduates and postgraduates'.[97] Also well known for its reliability was John Conybeare's *Textbook of Medicine*, with which reviews noted that it 'has gained wide acceptance' and 'already won a firm place' as 'one of the most popular textbooks of medicine with students and teachers'.[98] All three volumes were touted later in 1975 by endocrinologist Raymond Hoffenberg as the '[b]ig and classical volumes' in medical textbooks, while Oxford neurologist W. B. Matthews labelled them to be the spiritual successors of Osler and Anders.[99] The fifth volume to come out of the

[95] Anon. [review], 'G. E. Beaumont, Medicine: Essentials for Practitioners and Students (1st edn, London, 1932)', *The British Medical Journal* 2.3748 (5 November 1932), p. 839; Anon., 'George Ernest Beaumont', *The Lancet* 303.7863 (11 May 1974), p. 943; Porter, *The Greatest Benefit to Mankind*, pp. 11-2.

[96] Derrick Dunlop [review], 'Paul B. Beeson (eds.), *Cecil-Loeb Textbook of Medicine* (12th edn, London, 1967), *The British Medical Journal* 4.5574 (4 November 1967), p. 286; R. W. Lamont-Havers, 'Dr. Russell L. Cecil: An Appreciation', *Canadian Medical Association Journal* 93 (21 August 1965), p. 374; Alex Paton [review], 'D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrell, *Oxford Textbook of Medicine* (Oxford, 1983)', *The British Medical Journal* 286.6370 (26 March 1983), pp. 1030-1.

[97] Anon. [review], 'Donald Hunter (ed.), *Price's Textbook of the Practice of Medicine* (9th, Oxford, 1956)', *The British Journal of Tuberculosis* 51.2 (1957), p. 204; Anon. [review], 'Ronald Bodley Scott (ed.), *Price's Textbook of the Practice of Medicine* (12th edn, Oxford, 1978)', *Postgraduate Medical Journal* 55.642 (1 April 1979), p. 288; Victor Bloom [review], 'Ronald Bodley Scott (ed.), *Price's Textbook of the Practice of Medicine* (12th edn, Oxford, 1978)', *Journal of the Royal Society of Medicine* 72 (January 1979), p. 82.

[98] Walter C. Tobie [review], 'John Conybeare (ed.), *Textbook of Medicine* (8th edn, Baltimore, MA, 1946)', *The Quarterly Review of Biology* 24.3 (September 1949), p. 260; Anon. [review], 'J. J. Conybeare (ed.), *Textbook of Medicine* (7th edn, Edinburgh, 1945)', *The British Medical Journal* 1.4447 (30 March 1946), pp. 487-8; L. J. Witts [review], 'John Conybeare, and W. N. Mann (eds.), *Textbook of Medicine* (11th edn, Edinburgh, 1954)', *The British Medical Journal* 2.4900 (4 December 1954), pp. 1338-9.

[99] R. Hoffenberg, 'Has the text book a future? Textbooks and the teacher', *The British Medical Journal* 4.5997 (13 December 1975), p. 627; Matthews, 'Osler Oration', p. 308.

end of the interwar era was the *Textbook of Medical Treatment*, the first edition of which was edited by Edinburgh-based physician Derrick Dunlop. The volume was an instant classic, commended by the *Postgraduate Medical Journal* for its 'enduring and richly deserved popularity' among students and practitioners and as a quality textbook touted, by the publication of its tenth edition in 1966, for having 'the flavour of a well-blended carefully matured scotch whisky'.[100] All five volumes maintained their high reputation among textbook reviews in varying degrees throughout the interwar period.

Among the new series of textbooks, three out of five maintained throughout the period that alcohol played a direct role in causing cirrhosis, albeit with some reservations. Both the 1927 and 1938 editions of Cecil's *Textbook* argued that 'very strong clinical opinion still points to alcohol as the chief etiological factor' where the 'poison may act directly on the liver cells'. This was stated in consideration of the 'attempts, especially in recent years, to explain portal cirrhosis on the basis of infection, or "subinfection" and intoxications other than with alcohol', indicating that the emergence of other potential aetiological explanations warranted some mention for the readers.[101] Beaumont's *Medicine Essentials* recognised alcohol as 'a factor of great importance', even though the 'nature of the irritant' was not certain in all cases.[102] Dunlop's *Textbook* also expressed uncertainty. The statement that alcohol is 'still by far the commonest cause of hepatic cirrhosis' was followed by the caveat that the '[a]rguments as to how it acts do not concern us here'.[103] The language found in Beaumont and Dunlop's textbooks indicate that the authors were avoiding controversy by toeing the line through the affirmation of the DTT while simultaneously refraining from providing a full, conclusive account of its aetiology. Although medical textbooks were expected to provide as much

[100] D. S. L. [review], 'D. M. Dunlop, L. S. P. Davidson, and J. W. McNee (eds.), *Textbook of Medical Treatment* (5th edn, Edinburgh, 1949)', *Postgraduate Medical Journal* 25.283 (1 May 1949), p. 224; R. I. S. Bayliss [review], 'Derrick Dunlop, and Stanley Alstead (eds.), *Textbook of Medical Treatment* (10th edn, Edinburgh, 1966)', *The British Medical Journal* 1.5498 (21 May 1966), p. 1285.

[101] Russell L. Cecil (ed.), *A Text-Book of Medicine* (1st edn, London, 1927), p. 740; Russell L. Cecil (ed.), *A Text-Book of Medicine* (4th edn, London, 1938), p. 789.

[102] G. E. Beaumont, *Medicine: Essentials for Practitioners and Students* (London, 1932), p. 71.

[103] J. W. McNee, and D. Smith 'Diseases of the Liver, Gallbladder and Biliary Tract, Pancreas and Peritoneum', in D. M. Dunlop, L. S. P. Davidson, and J. W. McNee (eds.), *Textbook of Medical Treatment* (2nd edn, Edinburgh, 1940), p. 610.

information as possible on the 'facts' of modern medicine, their refusal to go into detail on among the most recognised diseases of alcoholism suggested that the authors were amply aware that its aetiology was being reassessed at the time.

Along with Wheeler's *Handbook of Medicine*, the remaining two volumes suggested that alcohol played an indirect role in causing liver damage. The original 1922 edition of Price's *Textbook* refrained from specifically discussing the aetiology of cirrhosis. Instead, it referred to its association with alcohol, stating how a 'large majority of patients [of cirrhosis] … have indulged excessively in alcohol'.[104] However, the later 1934 edition suggested the likelihood that 'alcohol produces cirrhosis of the liver indirectly by leading to gastroenteritis' as 'the poisons produced in the stomach and intestines are absorbed and pass to the liver'. The section presented some confusion in its subsequent supposition that the poisonous action of gastrointestinal toxins worked in tandem with 'the direct poisonous action of alcohol and occasionally of other toxins, such as that of malaria, on the liver cells'.[105] Rather than interpreting it as an explicit endorsement of alcohol's indirect action, this passage ought to be understood as a conjecture on the likely mechanism of the pathogenesis of cirrhosis. Conybeare's *Textbook* similarly attributed alcohol as an indirect factor that enabled gastritis to cause liver damage, even if the author identified drink to be 'by far the most important, though by no means the only aetiological factor'.[106] In their suppositions on the causation of cirrhosis, neither volumes were particularly confident in their belief that alcohol played an indirect role. Much like Beaumont and Dunlop's volumes, many of the interwar textbook authors were characterised by indecisiveness rather than certainty, with the language allowing for the possibility that alcohol might be still considered a direct toxin. In spite of that, the noted tendency towards recognising the likelihood that alcohol was nothing more than an indirect factor to cirrhosis indicated a clear shift in the predominant medical knowledge at the time.

[104] Frederick W. Price (ed.), *A Textbook of the Practice of Medicine* (1st edn, London, 1922), pp. 567-605.
[105] Hurst, 'Diseases of the Digestive System', in Price (ed.), *A Textbook of the Practice of Medicine*, p. 702.
[106] J. J. Conybeare (eds.), *A Textbook of Medicine* (1st edn, Edinburgh, 1929), p. 633; J. J. Conybeare (eds.), *A Textbook of Medicine* (4th, Edinburgh, 1939), pp. 455-9.

The question concerning the aetiology of cirrhosis was explored in greater detailed in *The Lancet* and *The British Medical Journal*, the two leading medical journals in Britain renowned for their coverage of a wide array of developments in general medicine. At the beginning of the 1920s, several articles highlighted the likely role of endogenous factors that predisposed individuals to cirrhosis in order to explain why only a fraction of heavy drinkers developed a cirrhotic liver. However, it was unclear exactly what sorts of predisposing factors, whether it be genetic, sexual, or environmental, were implied in many of these texts. While physicians were aware of how heredity (i.e. traits passed from one generation to another) shaped one's susceptibility to certain diseases, modern genetics, tracing back to Gregor Mendel's famous pea plant studies in 1866, was still too underdeveloped to give a reasonably accurate account of the specific factors that contributed to serious liver damage. Before genetics was studied at a molecular level in 1943 by the isolation of the DNA, human genetics in pathology was understood through patterns of inheritance that traced the incidence of a certain illness in the family history of a patient.[107] This approach was first established in 1902 by Archibald Garrod in his study of alkaptonuria (a rare disorder that prevents the body from processing certain chemicals, causing urine go dark) as a recessive illness, which led to the nascent medical interest in 'inborn errors'.[108] In order to explain the statistical variation in the incidence of cirrhosis across a sample of individuals, medical texts casually mentioned the role of individual predispositions without the accompaniment of a detailed explanation of the nature of the predisposition itself. As explored in chapter 4 of this thesis, genetic factors to the causation of cirrhosis was not seriously looked into by most specialists until the late twentieth century.

Both journals took interest in a publication of a new monograph in 1923 by Norwegian physician Søren Laache, which highlighted the role of individual susceptibilities to serious liver

[107] Porter, *The Greatest Benefit to Mankind*, pp. 586-7 Oswald; T. Avery, Colin M. MacLeod, and Maclyn McCarty, 'Studies on the Chemical Nature of the Substance Inducing Transformation of Pneumococcal Types: Induction of Transformation by a Desoxyribonucleic Acid Fraction Isolated from Pneumococcus Type III', *The Journal of Experimental Medicine* 79.2 (February 1944), pp. 137-58.
[108] Peter S. Harper, *A Short History of Medical Genetics* (Oxford, 2008), pp. 172-3

damage.[109] Laache observed that only 34.3 per cent of cirrhosis patients ever had a history of alcoholism, leading him to conclude that the development of the disease 'probably depend on a certain predisposition without which alcohol cannot provoke cirrhosis'.[110] Although Laache acknowledged that alcohol might have a direct action on the liver, he argued for the existence of an additional factor that contributed to variations across individuals in its toxicity and the pathogenesis of cirrhosis. This notion was taken further by Victor Scheel, a liver pathologist from Denmark, who similarly highlighted the role played by individual predispositions to explain the statistical rarity of cirrhosis among heavy drinkers.[111] Evidently, suggestions on the impact of individual factors in the extent of alcohol's toxicity to the liver that came from Scandinavian physicians like Laache and Scheel were influential enough to receive some coverage in British medical journals. The doubts held by a handful of Edwardian physicians over the straightforward belief that cirrhosis was directly caused by alcohol without any exception persisted well into the interwar era.

The Lancet later published several articles that declared that liver damage was likely attributable to metal poisoning. Rather than proposing an additional aetiology to the disease, this suggestion explicitly challenged the notion that alcohol played any role in the causation of cirrhosis. A 1928 editorial discussed how F. B. Mallory argued that 'the recent decline in the frequency of the [liver] disease is due as much to giving up copper cooking vessels as to moderation in alcohol.'[112] Mallory was an eminent pathologist from the United States who reached this conclusion after discovering traces of copper in bootlegged liquors in Boston during the prohibition. He subsequently succeeded in producing liver damage in rabbits that were fed copper acetate, resulting in the identification of copper poisoning as a causative agent to liver disease.[113] Mallory extended the same conclusion to phosphorus in a separate study from 1933.[114] In the present day, it is recognised that haemochromatosis, or iron overload, is a

[109] Anon., 'Aetiology and Prognosis of Cirrhosis of the Liver', *The Lancet* 201.5205 (2 June 1923), p. 1122.
[110] Anon., 'Epitome of Current Medical Literature', *The British Medical Journal* 2.3263 (14 July 1923), p. 5.
[111] Anon., 'Dr Victor Scheel', *The British Medical Journal* 1.3289 (12 January 1924), p. 90.
[112] Anon., 'Cirrhosis of the Liver', *The Lancet*, 211.5462 (5 May 1928), pp. 922-3.
[113] Anon., 'Copper and the Human Organism', *Journal (American Water Works Association)* 21.2 (February 1929), pp. 262-3.
[114] F. B. Mallory, 'Phosphorus and Alcoholic Cirrhosis', *American Journal of Pathology* 9 (1933), pp. 557-67.

known genetic condition which may co-exist with alcoholic excess as a cause of cirrhosis. Yet, Mallory was erroneous to disregard alcohol's toxicity simply because they identified another potential causative agent to liver damage. His conclusions, however, were a symptom of the wider tendency within the medical sciences to contest alcohol's assumed status as the primary aetiology of cirrhosis at the time.

Founded on a similar premise, a research article from 1930 by G. Marshall Findlay tied cirrhosis to manganese poisoning in an experiment that reproduced liver lesions that resembled cirrhosis in some mammals through the injection of large doses of manganese chloride.[115] A. E. Boycott and G. R. Cameron additionally argued in a 1930 *Lancet* article that 'vegetarian teetotallers' were more susceptible to cirrhosis than alcoholics were because they had a 'higher risk of taking manganese than heavy drinkers'.[116] To them, 'there are many difficulties in the way of believing that the liver damage is caused directly by the alcohol' in spite of the close association.[117] This conclusion was suggested as a response to the need to explain the pathogenesis of cirrhosis using a theory that at least had some experimental basis. Although the line of inquiry into the possible role of manganese poisoning failed to gain much traction, the coverage of such controversial theories by *The Lancet* indicates the existence of an interest in mainstream medical practice to challenge and overcome the traditional understanding of cirrhosis as an 'alcoholic's disease'.

The supposition that alcohol merely played a secondary, indirect role in enabling bacterial toxins from the stomach to damage the liver surfaced in the latter half of the interwar period. Although this aetiological theory was highlighted by the likes of Rolleston and Duckworth in the Edwardian period, it was seldom taken seriously by most medical professionals in Britain at the time.[118] An editorial in *The British Medical Journal* noted the growing traction behind the knowledge of alcohol as an indirect factor, understood to weaken

[115] G. Marshall Findlay, 'The Experimental Production of Biliary Cirrhosis by Salts of Manganese', *British Journal of Experimental Pathology* 5.2 (April 1924), pp. 92-9.
[116] A. E. Boycott, and G. R. Cameron, 'Manganese in Foodstuffs: Its possible relation to cirrhosis of the liver', *The Lancet* 216.5592 (1 November 1930), p. 959.
[117] *Ibid.*, p. 959.
[118] Duckworth, 'A Clinical Lecture', p. 348; Rolleston, 'Alcoholism', pp. 916-7.

the resistance of liver cells to toxins and infections.[119] In the 1931 annual Lumleian lectures at the Royal College of Physicians, William Willcox asserted that cirrhosis was caused by alcohol 'permitting the passage of toxins of bacterial origin through the gastro-intestinal mucous membrane, and so on to the liver. It was these toxins, not the alcohol, which caused the progressive hepatic fibrosis.'[120] Alcohol was understandably assumed to only play an indirect role, not least because there seemed to be no precise experimental articulation of the mechanism of alcohol damaging the liver at the time, as there was only real evidence for a strong association between the two.

Although Herd's argument that theories attributing cirrhosis to the indirect action of alcohol gained traction applies to Britain's case after the First World War, her paper fails to provide an account of the ideas and theories that tied together the slow demise of the DTT with the emergence of the NDT.[121] This section has thus explored the variety of other potential explanations that were suggested by the interwar medical professionals to account for some of the shortfalls of straightforwardly attributing cirrhosis to alcohol. While most medical texts at the time did not explicitly reject alcohol as the primary cause of the disease, a large portion of them were increasingly reluctant to take the DTT at face value, some choosing instead to recognise the complexity underlying the causal relationship between alcohol and liver damage. This is in reflection of how the interwar era was an important period of transition for the aetiological understandings of cirrhosis, witnessing a process through which the faith in alcohol's direct culpability in liver damage was slowly abandoned on behalf of a multiplicity of other explanations and theories.

Cirrhosis Aetiology and New Moderationism

This final section brings the first two sections together by explaining how the changing medical knowledge on alcohol and the liver was appropriated in the sphere of interwar moderationism.

[119] Anon., 'Epitome of Current Medical Literature', *The British Medical Journal* 1.3340 (3 January 1925), p. 1.
[120] William Willcox, 'Toxic Jaundice Lumleian Lectures', *The British Medical Journal* 1.3665 (4 April 1931), p. 596.
[121] Herd, 'Ideology, history and changing models', p. 1117.

Within the context of the end of the prohibition in the United States, Katcher states that 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'.[122] The same trend broadly applies to the case of Britain after the First World War, where the shift towards the conception of alcohol as having an indirect action on the liver had a far-reaching impact on interwar moderationist ideas. However, this section supplements Katcher's original argument by clearly showing that New Moderationist texts made many of the same claims on cirrhosis aetiology that were found in medical texts explored in the previous section. Furthermore, the diminishing legitimacy of the belief in alcohol's direct causation was conveniently used to downplay the general toxic qualities of alcohol as part of the effort by New Moderationism to distance the public discussion away from the exaggeration of the hazards of drink by the temperance movement. New understandings of alcohol and the liver were referred to in the medical literature of interwar moderationism as a constituent of the wider knowledge on alcohol and its effects on the human body. This section traces references of alcohol and the liver within many of the texts touched on in the first section to study the implications and the motivations behind such references to the aetiological debate.

The likelihood that alcohol played nothing more than an indirect role in causing liver damage was not explicitly referred to in the earliest New Moderationist texts. The CCB's *Alcohol: Its Action on the Human Organism* from 1918 maintained that cirrhosis was a 'frequent cause of death' for alcoholics.[123] The report attributed this claim to the evidence that the mortality rate from cirrhosis was 'sixfold' among occupations associated with the liquor trade over other industries.[124] It stated, however, that 'an isolated bout of drunkenness does not leave any lasting after-effects on the liver' as chronic liver damage results from the long-term abuse of alcohol, contradicting many of the pre-war temperance accounts in which alcohol was understood to have an immediate action in critically harming the organ.[125] This reflects a

[122] Katcher, 'The post-repeal eclipse', p. 730.
[123] Central Control Board, *Alcohol*, pp. 99-100.
[124] *Ibid.*, p. 121. The report cited statistics from the Actuarial Society of America.
[125] *Ibid.*, p. 100.

notable distinction established between the acute and chronic harms of alcohol within interwar moderationist discourse. When emphasising 'the existence of wide individual variations in susceptibility to the injurious effects of alcohol', the report highlighted the growing awareness among medical professionals that alcohol's toxic action on the liver did not occur in a vacuum from other associated factors.[126] Alcohol's aetiological relationship to cirrhosis was understood to be far more complicated than the straightforward conception of the disease as an 'alcoholic's disease', citing many cases where 'persons have drunk what would be generally regarded as dangerously excessive quantities of alcohol for years, and have yet shown no signs of being the worse in health'.[127]

The report also commented on the difficulties of establishing a causality between alcohol and many of the illnesses tied with its consumption. This nuanced scepticism is noteworthy in how similar statements were seldom found in any of the equivalent medical literature on alcohol from the Edwardian period, many of which presumed without question that 'alcoholic' diseases were inevitably caused by alcohol itself.[128] Because habitual drunkards often engaged in other 'deleterious influences besides that of alcoholic excess', the report warned that 'it may be a matter of considerable difficulty to assess the due importance of the alcoholic factor in the causation of the disease conditions which are found in specially frequent association with intemperance'.[129] For instance, the 'general paralysis of the insane' (paralytic dementia) was highlighted as a common 'alcoholic' condition that was recently revealed to be caused by syphilis, a venereal disease that commonly developed in heavy drinkers owing to the diminution of self-control and self-awareness during a drunken bout of sexual intercourse.[130] The effort to scrutinise the assumed causation of alcohol in conditions that were often recognisable among alcoholics was part of the initiative to reassess the temperance movement's tendency to exaggerate the outcomes of drinking, especially that of moderate consumption.

[126] *Ibid.*, p. 97.
[127] *Ibid.*, p. 97.
[128] Horsley, and Sturge, *Alcohol and the Human Body*; Cheadle, 'The Lumleian Lectures', pp. 985-90; Hare, *On Alcoholism its Clinical Aspects.*
[129] *Ibid.*, pp. 94-5.
[130] *Ibid.*, p. 95.

The report thus signalled the growing tendency to view many 'alcoholic' diseases as having a complex relationship with alcohol itself as well as the emerging understanding that cirrhosis too is an indirect, rather than a direct, result of drink.

Almost immediately after the war, the theory that relegated alcohol as an indirect factor to cirrhosis was highlighted in Starling's 1923 treatise, *The Action of Alcohol on Man.* Although cirrhosis was frequently tied to the 'immoderate use of ardent spirits', the book noted that 'it has not been possible to reproduce with any certainty this condition by the experimental administration of alcohol to animals, and it may occur in rare cases in men who have not indulged in any alcoholic excess.'[131] By explaining the mechanism of liver damage, Starling understood that alcohol caused the 'destruction of the lining membrane of the alimentary canal, rendering this less efficient in the absorption of food and more prone to admit the passage of toxins produced in the process of digestion, or in the micro-organisms which abound in the contents of the gut'.[132] Therefore, cirrhosis was 'probably caused by the action of toxins or allied irritant substances absorbed from the alimentary canal and is only indirectly due to the action of alcohol'.[133]

It must be noted that Starling's account was published within a matter of few years after the end of the First World War. This is especially revealing, since, among the textbooks examined for this thesis, the theory of the indirect action of alcohol on the liver was endorsed only by the 1912 and 1920 editions Wheeler's *Handbook of Medicine* up until the publication of the book. This hinted at the possibility that Starling explicitly chose to frame the aetiology of cirrhosis under this specific theory, even if it had not been accepted among most medical professionals. While some physicians had been aware that animal experiments had largely failed to reproduce cirrhosis with alcohol, the notion alcohol played an indirect role in allowing other toxins to damage the liver was still undoubtedly controversial at the time. Thus, not only does the book demonstrate that the direct toxicity of alcohol on the liver was questioned by New Moderationism from early on; figures like Starling were purposefully inclined to adopt

[131] Starling, and Hutchinson, *The Action of Alcohol*, p. 147.
[132] *Ibid.*, p. 147.
[133] *Ibid.*, p. 147.

new models of disease causation to counter the overstatement of alcohol's harmful properties by the medical temperance movement.

The interest in adopting the theory based on the indirect action of alcohol on the liver was not isolated to Starling's book. A lecture titled 'alcohol: its use and abuse' delivered on 18 February 1924 at the Royal Society of Medicine by Dixon, a Cambridge-based pharmacologist, noted the diminishing medical support for the DTT.[134] The talk was laced with moderationist language, endorsing the benefits of moderate consumption of alcohol whilst simultaneously condemning its abuse. Dixon dispelled many of the '[m]isrepresentations, exaggerations, contradictions, and delusions' of pre-war understandings in which 'alcohol had been extolled as the elixir vitae, and on the other denounced as a deadly poison.'[135] The understanding that 'cirrhosis was always caused by alcohol' was further labelled as a 'misrepresentation' produced by prohibitionist 'bias'. 'Alcohol was not the direct cause of this condition' since, according to Dixon, only a quarter to half of all cases were associated with drink.[136] This statement presents itself to be somewhat misleading since a specific agent does not have to be the sole cause of a disease in order for it to be considered a direct cause. Although Dixon also failed to specify what he suspected as the direct cause of cirrhosis, his lecture nevertheless embodied an effort within New Moderationism to discredit the broadly held conceptualisation of cirrhosis as a disease of alcoholism.

Additionally, the decline of the DTT attracted the attention of sections of the medical temperance movement which were increasingly susceptible to the influence of New Moderationism. Interwar accounts of temperance breakfasts of the British Medical Temperance Association (BMTA) revealed that the group had largely abandoned their uncompromising support for total abstinence on behalf of moderation. A meeting that took place in 1931 discussed the need for medical professionals to promote the 'legitimate uses of alcohol' and 'the extent to which it was right to use for pleasure that with other might abuse or

[134] Dixon, 'Alcohol: its use and abuse', pp. 341-3.
[135] *Ibid.*, p. 341.
[136] *Ibid.*, p. 343.

misuse'.[137] This was a noted shift from the wholesale condemnation of all uses of alcohol that coloured much of the 'temperance breakfasts' predating the First World War. Quite surprisingly, a conference hosted by the National Temperance League on 30 November 1937 invited Rolleston to deliver a lecture on the pathological effects of alcohol. In true moderationist fashion, Rolleston characterised alcohol as a 'food, but a jealous food' for its inefficiency in providing nutrition, while suggesting that 'what was needed in regard to alcohol was education, not prohibition' as a solution to the problem of drunkenness, a statement that would have no doubt been deemed extremely controversial among temperance agitators during the Edwardian period.[138] Concerning the liver, Rolleston argued that alcohol 'might cause cirrhosis indirectly, and so act on the stomach and intestines that poisonous bodies would be formed and carried to the liver'.[139] This explanation is identical to the one found in Starling's *Action of Alcohol on Man*, which understood drink as an indirect factor that enabled other toxins to damage the liver. The extent of the influence of New Moderationism on the medical temperance movement was outlined by the group's willingness to tolerate Rolleston as an invited speaker to one of their largest annual gatherings, even if he had been consistently suggesting that alcohol might not be the direct cause of cirrhosis since before the war began.

While many temperance groups were willing to forego their drive for total abstinence on behalf of New Moderationism, some campaigners were unwilling to surrender the idea that cirrhosis was a disease brought about by drink. The most profound critique of interwar moderationism came from Courtenay Weeks, a surgeon and the director of the National Temperance League.[140] Weeks's *Alcohol and Human Life* was published in 1928 as a revised edition of Horsley and Sturge's *Alcohol and the Human Body*, a widely circulated temperance medical treatise from 1908 discussed in detail in the first chapter of this thesis.[141] Although Weeks's monograph was among the last texts that defended the old anti-drink cause against

[137] Anon., 'National temperance league breakfast', *The British Medical Journal* 2.3648 (15 August 1931), p. 134.
[138] Anon., 'Alcohol, Nutrition, and Fitness: Medical Aspects of a Social Problem', *The British Medical Journal* 2.4014 (11 December 1937), p. 1183.
[139] *Ibid.*, p. 1183.
[140] Woiak, '"A Medical Cromwell"', p. 364.
[141] Courtenay C. Weeks, *Alcohol and Human Life* (2nd edn, London, 1938), p. viii.

New Moderationism, it nonetheless admitted that 'anything but the very strictest *moderation* must inevitably have serious results', indicating that total abstinence was no longer seen as the be all and end all.[142] However, in spite of the apparent reduction in drunkenness after the First World War, Weeks warned his fellow temperance activists not to be complacent against the 'ghastly evil of alcoholism… the greatest single enemy Britain has to fear, the one which allies itself with all the mars and spoils of human life'.[143] Evidently, much of the language found in the text was still strongly embedded in the discursive tradition of the anti-drink movement.

Concerning the aetiology of cirrhosis, Weeks decried the 'tendency in some quarters to minimise the importance of alcohol, and in nearly all quarters a recognition that alcohol may be allied with syphilis and certain other toxic conditions in the production of cirrhosis of the liver'.[144] He observed that '[m]uch stress is laid upon the indirect of action of alcohol', citing the increasing tendency to blame gastritis and foreign non-alcoholic toxins as the direct causes of liver damage.[145] However, Weeks's detailed counterargument against the aetiological theory of indirect action shows that he had misunderstood the nature of the debate. As evidence in support of the DTT, he pointed out that cirrhosis deaths were statistically more prevalent among professionals who worked in close proximity to alcoholic beverages, and noted that overall cirrhosis mortality rates had fallen during and after the war alongside the decline of the amount of alcohol consumed by the population.[146] This statement outlined how Weeks was oblivious to the fact that the statistical association between cirrhosis and alcohol consumption continued to be widely accepted. He failed to recognise that many of his contemporaries were increasingly aware that this association did not necessarily translate to the conclusion that alcohol played a direct role in harming the liver.

Nevertheless, Weeks's anxiety over the reassessment of the aetiology of cirrhosis not only indicates the far-reaching influence of the aetiological debate on the wider discourse on

[142] *Ibid.*, p. 206.
[143] *Ibid.*, p. viii.
[144] *Ibid.*, p. 208.
[145] *Ibid.*, p. 208.
[146] *Ibid.*, pp. 210-6.

drink at the time but also of the alienation of more radical temperance opinions within professional medicine. Indeed, a review of his book in *The Lancet* criticised Weeks for his 'enthusiastic conviction that alcohol is wholly evil' and suggested a 'little more discretion' in approaching the topic.[147] Another scathing review accused him of producing 'propaganda', stating that had 'Dr Weeks contented himself with a plain statement of facts he might perhaps have carried greater conviction.'[148] Thus, medical texts on alcohol that were written explicitly from a radical temperance standpoint were increasingly discredited within the wider medical profession in Britain.

The impact of the aetiological shift in interwar moderationist thought was not purely restricted to the closed group of medical professionals. The most noteworthy case in which the abandonment of alcohol's direct toxicity had an impact on interwar moderationist discourse was in the royal commission on licensing in 1929-31. In the report, the pathogenesis of cirrhosis was described as follows: 'drinking, particularly of spirits, may cause chronic inflammation of the stomach, which in its turn may lead to disease of the liver'.[149] Here, alcohol is explicitly understood as an indirect cause of liver damage through gastritis. The report added that '[m]ost authorities appear to be satisfied that chronic alcoholic poisoning is prominent in the causation of cirrhosis of the liver, though there is a difference of opinion as to the degree of its responsibility, and the disease may arise from other causes.'[150] The question concerning the aetiology of cirrhosis was touched on in greater detail in *A Review of the Effects of Alcohol on Man* (1931), a monograph separately published by the medical committee of the royal commission.[151] It stated that '[t]here is considerable clinical and experimental evidence that alcohol may, at least indirectly, cause definite disorder of liver function. That it may itself directly cause permanent structural changes is less certain.'[152] Animal experiments were

[147] Anon., 'Reviews and Notices of Books', *The Lancet* 213.5521 (22 June 1929), pp. 1306-8.
[148] Anon., 'Alcohol in Hospital Practice', *The Lancet* 227.5872 (14 March 1936), p. 617.
[149] Anon., *Royal Commission*, p. 15.
[150] *Ibid.*, p. 15.
[151] Kathleen Kitchin, and D. Harcourt Kitchin (eds.), *A Review of the Effects of Alcohol on Man* (London, 1931), p. 7.
[152] W. D. Newcomb, and T. C. Hunt, 'The Changes in the Body Caused by Alcohol', in Kitchen, and Kitchen, *A Review of the Effects*, p. 169.

successful in producing fatty liver, the earliest stage of alcoholic liver disease, while cirrhosis, the latest stage, was 'almost impossible to reproduce' with alcohol alone.[153] The review, therefore, concluded that alcohol 'can rarely be more than an associated agent in the causation of the condition' since the 'exact role which it plays is not fully explained, and its importance in this respect has probably been much exaggerated.'[154] Although the medical committee refrained from going as far as to write off alcohol's potential causation, the conclusion that alcohol played nothing more than an indirect role highlighted an intellectual process involving the rejection, not just of direct toxicity, but of the previously dominant framework of cirrhosis as the 'alcoholic's disease'. The recognition of this aetiological shift in the royal commission is an indication that the medical debate on alcohol and the liver was part of the broader scientific recognition of New Moderationism within the wider political discourse on alcohol. This is a significant development where the medical reassessment of alcohol's relationship to the liver informed policymaking at the highest levels of the government.

From the CCB's *Alcohol: Its Action to the Human Organism* in 1918 to the royal commission on licensing in 1932, the aetiology of cirrhosis and the likelihood that drink only played an indirect role in the disease was acknowledged in every core text that contributed to New Moderationist Thought. Katcher's observation, that the minimisation of the harms of alcohol through renewed understandings of disease causation went hand-in-hand with the reaction against temperance ideology in post-repeal America, is largely applicable to the British case.[155] The declining belief in the DTT was adopted by the interwar moderationists as a way to reassess the temperance movement's overstatement of alcohol's deleterious harms on the body that previously prevailed in the Edwardian drink question.

Conclusion

The interwar era was a critical transitionary period when the belief in alcohol's direct toxicity on the liver was progressively replaced by an aetiological model that understood liver damage

[153] *Ibid.*, pp. 171-2.
[154] *Ibid.*, p. 174.
[155] Katcher, 'The post-repeal eclipse', p. 730.

to be an indirect outcome of drink, predating the eventual emergence of the NDT in the 1940s. In spite of that, the set of texts studied for this chapter suggested that there was a lack of a clear consensus over the exact aetiology of cirrhosis or the precise mechanism of alcohol's action on the liver. A model that attributed alcohol as an indirect cause of cirrhosis gained in popularity, but the reigning view was characterised by a growing awareness that alcohol's relationship with the liver was far more intricate than the simple cause-and-effect understanding that predominated before the First World War. This development turned out to play a vital role in the wider discursive shift towards the promotion of moderate drinking in the drink question. The diminishing importance of drink in liver disease was used to discredit the temperance movement's overstated harms of alcohol. Thus, the rise of New Moderationism was heavily informed by the slow demise of the DTT.

Chapter 3

The Nutritional Deficiency Theory

As the interwar era came to a close, the various aetiological theories of cirrhosis that existed in the decades prior to the Second World War were replaced by a single, predominant explanation: the nutritional deficiency theory (NDT). The NDT framed cirrhosis as a disease borne out of the lack of certain nutrients in the body, of which heavy drinking was seen to play an indirect role; the ingestion of excessive volumes of alcohol reduces one's appetite by taking up a large portion of the individual's caloric intake, thereby depriving the individual of an adequate diet.[1] The theory was legitimised by a set of clinical and experimental studies that seemingly accomplished the reproduction of liver damage through the provision of a deficient diet, some even succeeding in the prevention and treatment of liver disease through the use of supplements containing the necessary nutrients. By the 1950s, these studies allowed the NDT to emerge as the dominant conceptual framework in regard to alcoholic liver disease. Such developments were simultaneously abetted by general failure among older experimental studies in their attempts to reproduce cirrhosis in experimental animals through the use of alcohol.

This chapter illustrates a case in which medical scientists reached the 'wrong' conclusions for the right reasons and through the right methods. Although the NDT was eventually superseded by subsequent discoveries, it was apparent that the decision to attribute cirrhosis to malnutrition was logically sound in relation to the knowledge available at the time. Moreover, the abandonment of alcohol as a direct cause of cirrhosis was justified by a widely accepted view, which stipulated that one required experimental evidence in addition to a strong

[1] Gerald Klatskin, 'The Role of Alcohol in the Pathogenesis of Cirrhosis', *Yale Journal of Biology and Medicine* 26 (1953), pp. 33-4.

statistical association to infer that a specific factor was responsible for causing a disease. Within the British medical profession, the NDT was unanimously acknowledged as the most likely explanation of the pathogenesis of cirrhosis, while the notion that alcohol was a direct toxin to the liver was dismissed in virtually all avenues of medical opinion. What is interesting is that this major scientific shift had a decidedly limited impact on public discussions surrounding alcohol use in Britain, even though parliamentary debates and texts produced by surviving temperance groups showed that the nutritional framing of liver damage had attracted some interest. Much like that of the inherent uniqueness of the scientific dismissal of alcohol on behalf of malnutrition as a recognised cause of cirrhosis, the period's strong inclination towards moderation and moderate drinking was reflected in the peculiar developments that occurred within the toned-down debates over alcohol in postwar society.

The chapter begins by describing the original research that had contributed to the scientific emergence of the NDT. The second section moves onto how the NDT and its combined dismissal of alcohol's direct toxicity was legitimised within the context of the international popularisation of the disease concept of alcoholism. The last two sections studies how these scientific changes were received in Britain. The first explores the impact of the shift on the British medical profession by looking how the knowledge was received in textbooks of general medicine and general medical journals. The final section assesses the impact of the scientific debate on the wider public discussions on alcohol in Britain after the Second World War.

The Scientific Emergence of the Nutritional Deficiency Theory

The establishment of the NDT as the prevailing aetiological understanding of cirrhosis was largely owed to a set of clinical and experimental studies performed throughout the 1930s and 40s. Part of this section's argument builds on Herd and Katcher's accounts, both of whom correctly attribute the dismissal of the theory of alcoholic causation to the inability to experimentally demonstrate cirrhosis as an outcome of heavy alcohol exposure.[2] The

[2] Herd, 'Ideology, history and changing models', p. 1114; Katcher, 'The Post-Repeal Eclipse', p. 736.

abandonment of the direct toxicity theory (DTT) was deemed to be logically sound due to the general failure within past laboratory studies to reproduce cirrhosis in animal livers through the provision of alcohol. However, neither author pays much attention to the reasons why the scientists specifically decided on malnutrition as the most plausible cause of the disease, especially when there were other theories at the time that similarly relegated alcohol to being an indirect factor. This section addresses this question by studying the key experimental and clinical studies in peer-reviewed journals of medicine between the 1930s and the 1950s. A detailed examination of their content reveals that the interest in nutritional health was triggered by the successful production of a cohort of liver diseases, including cirrhosis, in animal subjects through the introduction a dietary regimen deficient in certain nutrients. Later experiments demonstrated that severe liver damage could even be treated through the ingestion of the very same nutrients. As a result, not only was alcohol almost entirely dismissed as a direct toxin to the liver, the discursive framing of cirrhosis as the 'alcoholic's disease' virtually disappeared from much of the specialist medical literature.

The methodology of this section warrants some explanation. This is the first time in the thesis in which the content of the original research found in scientific journals is discussed in such detail, involving an exploration of the content of the discovery and the justification of new scientific claims. The rise of the NDT was undoubtedly the most noteworthy development in the history of the medical understandings of alcohol and the liver in the twentieth century. The historical emphasis on experimental evidence in inferring disease causation has featured prominently in the first two chapters. During the Edwardian period, contrarians such as Humphry Rolleston challenged the DTT by highlighting the experimental failure to reproduce cirrhosis in animal livers through the use of alcohol.[3] The methodological distinction between the original research explored in this section and other previously explored medical texts, such as textbooks of general medicine and general medical journals, rests on Fleck's differentiation between 'journal science' and 'textbook science'. The former describes the production of original scientific knowledge within a closed group of specialists and experts in a particular

[3] Rolleston, 'Alcoholism', pp. 916-7.

field. The latter describes the moment whereby the knowledge becomes established as a 'scientific fact' in the wider discipline, allowing for its dissemination for practical use (i.e. clinical practice and medical education).[4]

Thus, this section explores the origins of the NDT in academic journal articles, most of which were of studies performed by scientists based in North America. Although the original scientific contributions to the knowledge on liver disease was scattered throughout the Western world in the first half of the century, the period saw a markedly unipolar concentration of high-impact research from the United States. This development was partly contributed to by the migration of European medical experts to America from before, during, and after the Second World War. Indeed, several of the most influential contributors in liver disease research discussed in the last two chapters of this thesis were themselves Jewish refugees who escaped the rise of Nazi Germany in Europe.[5] As explored in the third section of this chapter, the British medical profession was heavily receptive to the knowledge produced by such figures. In order to identify the most relevant research articles from the period, I have sought out the bibliographies of specialist textbooks on the liver and literature reviews.[6]

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[4] Ilana Lowy, 'Ludwik Fleck on the social construction of medical knowledge', *Sociology of Health & Illness* 10.2 (1988), pp. 145-7.

[5] For more on the rise of American science in the postwar era and the role of migration, see Joseph Manzione, '"Amusing and Amazing and Practical and Military": The Legacy of Scientific Internationalism in American Foreign Policy', *Diplomatic History* 24.1 (2000), pp. 21-7.

[6] In chronological order of publication, the specialist textbooks include: Mitchell A. Spellberg, *Diseases of the Liver* (New York, 1954); Fenton Schaffner, Sheila Sherlock, and Carroll M. Leevy, *The Liver and Its Diseases* (New York, 1974); Neil McIntyre, and Marsha Y. Morgan, 'Nutritional Aspects of Liver Disease', pp. 108-33, and Mikko P. Salaspuro, and Charles S. Lieber, 'Alcoholic Liver Disease', pp. 735-73, in Ralph Wright, K. G. M. M. Alberti, Stephen Karran, and G. H. Millward-Sadler (eds.), *Liver and Biliary Disease: Pathophysiology, Diagnosis, Management* (London, 1979); Harold O. Conn, and Colin E. Atterbury, 'Cirrhosis', in Leon Schiff, and Eugene R. Schiff (eds.), *Diseases of the Liver* (6th edn, Philadelphia, 1987), pp. 725-86. The literature reviews include: Norman Jolliffe, and E. Morton Jellinek, 'Vitamin Deficiencies and Liver Cirrhosis in Alcoholism, Part VII Cirrhosis of the Liver', *Quarterly Journal of Studies on Alcohol* 2 (1941), pp. 544-83; Howard T. Karsner, 'Morphology and Pathogenesis of Hepatic Cirrhosis', *American Journal of Clinical Pathology* 13 (1943), pp. 596-606; Russell S. Boles, Robert S. Crew, and William Dunbar, '"Alcoholic Cirrhosis"', *The Journal of the American Medical Association* 134.8 (1947), pp. 670-3; Klatskin, 'The Role of Alcohol', pp. 23-37; Gerald Klatskin, 'Effect of alcohol on the liver', *The Journal of the American Medical Association* 170.14 (1959), pp. 1671-6; Gerald Klatskin, 'Newer concepts of cirrhosis', *JAMA Archives of Internal Medicine* 104.6 (1959), pp. 899-902; Gerald Klatskin, 'Experimental Studies on the Role of Alcohol in the Pathogenesis of Cirrhosis', *The American Journal of Clinical Nutrition* 9 (1961), pp. 439-45; P. E. Steiner, 'Evolution of Research in the Etiological Types of Cirrhotic Diseases of the Liver, 1931-1961', *Pathobiology* 27.5 (1964), pp. 890-924; Galambos, 'Progress Report', pp. 477-90; H. M. Sinclair, 'Nutritional

As explored in the previous chapter, the DTT experienced gradual decline throughout the interwar period. In 1934, Virgil H. Moon, a pathologist based in Philadelphia, authored an influential review article titled 'experimental cirrhosis in relation to human cirrhosis', which discussed a range of aetiological theories of the disease.[7] According to the review, studies had been carried out on the potential hepatotoxicity of a set of inorganic poisons that included phosphorous, lead, manganese, and copper. Such substances, however, had largely failed to cause the development of cirrhosis without the aid of alcohol, leading Moon to conclude that '[i]t seems improbable that inorganic poisons… constitute an important factor in the etiology of human cirrhosis'.[8] Yet, many of the attempts that made use of the primary culprit, alcohol, had shown that '[n]o cirrhosis has been reported in experiments with alcohol in animals other than rabbits.'[9] Here, Moon remarked on the tendency of some scientists to selectively experiment on species, like rabbits, that were more susceptible to liver damage than other animals such as dogs, rats, and guinea pigs. Arguing that the 'belief that cirrhosis is caused by alcohol has not received experimental support', Moon suspected that alcohol most likely played only a contributory or a predisposing role to liver damage.[10] Another paper by Russell S. Boles and Jefferson H. Clark (1936) agreed with this analysis, stating that, since 'it is generally accepted that alcohol alone will not produce cirrhosis', it ought to be completely disregarded as a legitimate cause of the disease.[11] Evidently, the DTT had lost much of its credibility among most specialists by the late 1930s.

So how did nutritional deficiency come to be accepted as the most likely cause of cirrhosis in the place of alcohol? The study of nutrition emerged in its modern form in the late nineteenth and early twentieth centuries. At the time, diseases such as beriberi, pellagra, scurvy,

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Aspects of Alcohol Consumption', *Proceedings of the Nutrition Society* 31.2 (1972), pp. 117-23; Lawrence Feinman, and Charles S. Lieber, 'Liver disease in alcoholism', *The Biology of Alcoholism* 3 (1974), pp. 303-338.

[7] Virgil H. Moon, 'Experimental Cirrhosis in Relation to Human Cirrhosis', *Archives of Pathology* 18 (1934), pp. 381-424.

[8] *Ibid.*, pp. 387-95.

[9] *Ibid.*, pp. 396-402.

[10] *Ibid.*, pp. 419-21.

[11] Russell S. Boles, and Jefferson H. Clark, 'The Role of Alcohol in Cirrhosis of the Liver: A Clinical and Pathologic Study based on four thousand autopsies', *The Journal of the American Medical Association* 107.15 (1936), pp. 1200-3.

and rickets were increasingly understood to be brought about by the absence of vital elements in the body, a new paradigm of disease causation that defected from the dominant association of illnesses with positive agents such as toxins and microorganisms.[12] Nutritional science matured as a field of medicine during the interwar period when many of the vitamins that we know of in the present day were isolated.[13] The two World Wars and the Great Depression also contributed to the growing importance of expert knowledge on nutrition and health within the policies surrounding public health, social welfare, and agriculture.[14] In turn, this period witnessed a process whereby many diseases that were previously believed to be caused directly by alcohol were now considered to be nutritional in nature, a discovery founded on the frequent observation that many alcoholics were visibly malnourished.[15] This was true of polyneuropathy, pellagra, and stomatitis, all of which continue to be acknowledged today as nutritional diseases.[16] Therefore, Herd and Katcher believe that the popular inclination to explain the cause of many illnesses under their dietary origins partly shaped how cirrhosis came to be understood as an illness of deficiency.[17] However, it would be difficult to prove that this trend, a phenomenon described as *nutritionism*[18], was directly responsible for the emergence of the NDT, since there seemed to be no explicit indication of the influence of such tendencies in any of the sources discussed in this chapter. At the same time, one cannot deny that the discovery

[12] Porter, *The Greatest Benefit*, pp. 551-2.
[13] Lee Russell McDowell, *Vitamins in Animal Nutrition: Comparative Aspects to Human Nutrition* (Amsterdam, 2012), p. 398.
[14] Josep L. Barona, 'Nutrition and Health: The international context during the inter-war crisis', *Social History of Medicine* 21 (2008), pp. 87-9; Rima D. Apple, *Vitamania: Vitamins in American Culture* (New Brunswick, NJ, 1996).
[15] G. R. Minot, M. B. Strauss, and S. Cobb, '"Alcoholic" polyneuritis; dietary deficiency as a factor in its production', *New England Journal of Medicine* 208 (1933), pp. 1244-9; M. A. Blackenhorn, and T. D. Spies, 'Oral complications and chronic alcoholism', *The Journal of the American Medical Association* 107 (1936), pp. 641-2; S. Weiss, and R. W. Wilkins, 'Nature of the cardiovascular disturbance in nutritional deficiency states (beriberi)', *Annals of Internal Medicine* 11 (1937), pp. 104-48; J. M. Askey, 'The use of vitamins in the treatment of alcoholic diseases', *California and Western Medicine* 51 (1939), pp. 294-6.
[16] Robert Goodhart, and Norman Jolliffe, 'The Role of Nutritional Deficiencies in the Production of Cardiovascular Disturbances in the Alcohol Addict', *American Heart Journal* 15.5 (1938), p. 569.
[17] Herd, 'Ideology, history and changing models', p. 1117; Katcher, 'The Post-Repeal Eclipse', p. 739.
[18] Georgy Scrinis, 'On the ideology of nutritionism', *Gastronomica* 8.1 (2008), p. 39; Tenna Jensen, 'The importance of age perceptions and nutritional science to early twentieth-century institutional diets', *Social History of Medicine* 30.1 (2017), p. 158.

of vitamins in the preceding decades provided a new avenue of research that allowed medical scientists to conceptualise liver damage as an outcome of the lack of nutrients by the late 1930s and early 1940s. The relation between the wider context of the rise of nutritional science and the debate on the aetiology of cirrhosis is explored in greater detail in the beginning of the following section of this chapter.

The supposition that liver damage was caused by the deficiency of key nutrients was an accident borne out of the discovery of insulin by a handful of Canadian scientists at the University of Toronto. In the summer of 1921, Frederick Banting made history with his medical student, Charles Best, when they successfully devised a new treatment method of diabetes. Banting and Best successfully isolated a hormone called insulin, extracted at the time from the pancreas removed from dogs, which functioned to reduce high blood glucose levels when injected to the body.[19] After receiving a portion of Banting's reward from the Nobel Prize in Medicine in 1923, Best completed his education to become a professor of physiology by 1929 at his *alma mater*.[20] There, he continued to make use of depancreatised dogs as his primary subjects to indulge in his newfound interest in fatty liver, the earliest stage of alcoholic liver damage. The pancreas has two components. The endocrine pancreas functions to regulate the body's blood sugar level through the production of insulin, while the exocrine pancreas secretes enzymes into the small intestine to aid the breakdown of carbohydrates, proteins, and fats in food. Among such fats, Best identified one—lecithin—to be the fat that prevented the production of fatty liver.[21] He further isolated choline as the specific nutrient within lecithin that carried this specific property.[22] The absence of a pancreas, therefore, compromises the dog's capacity to metabolise lecithin, resulting in the failure to produce choline, the absence of which led to the excess accumulation of fat in the liver. Thus, the question remained whether

[19] Frederick Grant Banting, and Charles Herbert Best, 'Pancreatic extracts', *The Journal of Laboratory and Clinical Medicine* 7.8 (1922), pp. 464-73.
[20] Anon., 'C. H. Best', *The British Medical Journal* 1.6117 (8 April 1978), p. 927.
[21] C. H. Best, J. M. Hershey, and M. Elinor Huntsman, 'The effect of lecithin on fat deposition in the liver of the normal rat', *The Journal of Physiology* 75.1 (1932), pp. 56-66.
[22] C. H. Best, and M. Elinor Huntsman, 'The effects of the components of lecithin upon the deposition of fat in the liver', *The Journal of Physiology* 75.4 (1932), pp. 405-12.

fatty liver could be prevented or alleviated through the introduction of a diet that contained adequate amounts of choline.

In 1933, Best performed an experiment in which a group of depancreatised dogs were provided with a daily regimen that contained excess fat.[23] The control group from which choline was withheld from quickly developed fatty liver, while the experimental group receiving choline did not, in spite of the continued consumption of a fat-based diet. Based on this, Best argued that choline effectively functioned to prevent early stages of liver damage.[24] He further confirmed this through a study of the morphological changes of the livers of diabetic dogs and normal rats. In both animals, the addition of choline to a fat-based diet seemingly prevented the deposition of fat in the liver.[25] While none of Best's studies at this stage commented on their implications on the specific aetiology of liver disease relating to alcohol, his research indicated that the deficiency of key nutrients played an important role in the production of liver damage.

The interest in studying the development of liver disease in depancreatised dogs was further taken up by Charles Lloyd Connor and I. L. Chaikoff, two physiologists based at the University of California Medical School in San Francisco. In a 1938 study, four of the 16 depancreatised dogs with fatty liver developed 'extensive cirrhosis' after being kept alive for over a year.[26] This established deficiency-caused fatty liver as a necessary precondition for the further development of cirrhosis, which itself was brought about in a later study through the ingestion of alcohol.[27] The study also mentioned nutritional deficiency for the first time within the context of a debate surrounding the assumed causation of alcohol in cirrhosis. Laboratory studies on depancreatised dogs crucially paved a path towards the recognition of malnutrition

[23] C. H. Best, G. C. Ferguson, and J. M. Hershey, 'Choline and Liver Fat in Diabetic Dogs', *The Journal of Physiology* 79.1 (1933), pp. 94-102.
[24] *Ibid.*, pp. 99-101.
[25] D. L. MacLean, and C. H. Best, 'Choline and liver fat', *British Journal of Experimental Pathology* 15.4 (1934), pp. 193-9.
[26] I.L. Chaikoff, C.L. Connor, and G.R. Biskind, 'Fatty infiltration and cirrhosis of the liver in depancreatized dogs maintained with insulin', *The American Journal of Pathology* 14.1 (1938), pp. 101-110.
[27] C. L. Connor, and I. L. Chaikoff, 'Production of Cirrhosis in Fatty Livers with Alcohol', *Experimental Biology and Medicine* 39.2 (1938), pp. 356-9.

as a potential causative factor in alcoholic liver disease. Still, the notion that alcohol was a direct cause was not entirely abandoned as of yet. Connor cautioned in an article published in 1939 that, although 'large amounts of alcohol are necessary' in the production of cirrhosis, it nonetheless required the existence of an additional factor like nutritional deficiency, as scientists have uniformly failed to reproduce the disease through the use of alcohol alone.[28] He was also sceptical of studies that had allegedly succeeded in treating alcoholic liver disease with high-vitamin diets, suggesting that the improved prognosis might also be the partial outcome of the drinker ceasing consumption after being admitted to hospital.[29]

The impact of nutritional deficiency on the liver was further expanded on by subsequent studies. An experiment performed by Paul György and Harry Goldblatt on rats sustained on a diet deficient in vitamin B complex, a supplement including all eight variations of B vitamin, resulted in the development of 'various pathological changes in the liver', including fatty liver, necrosis, haemorrhage, and fibrosis.[30] They discovered that the addition of yeast, which contained a rich supply of vitamin B2, prevented hepatic injury, leading them to conclude that 'liver changes are of nutritional origin and should be correlated to deficiency of a part of the vitamin B2 complex'.[31] Later, Arnold R. Rich and John D. Hamilton, two pathologists based at the Johns Hopkins Hospital, performed what turned out to be the first study that succeeded in reproducing cirrhosis through the provision of an inadequate diet.[32] It involved three separate experiments in which a group of rabbits were kept on a diet lacking in a variety of nutrients. Among the 15 rabbits that were deficient in yeast, which contains choline, all but one ended up developing cirrhosis.[33] The importance of choline in the prevention of liver damage was further confirmed by a later paper by György and Goldblatt, which discussed

[28] Charles L. Connor, 'The etiology and pathogenesis of alcoholic cirrhosis of the liver', *The Journal of the American Medical Association* 112.5 (1939), pp. 387-90.
[29] Charles L. Connor, 'Cirrhosis of the Liver', *Quarterly Journal of Studies on Alcohol* 1 (1940), p. 98.
[30] Paul György, and Harry Goldblatt, 'Hepatic injury on a nutritional basis in rats', *The Journal of Experimental Medicine* 70.2 (1939), pp. 185-92.
[31] *Ibid.*, p. 191.
[32] Arnold R. Rich, and John D. Hamilton, 'The Experimental Production of Cirrhosis of the Liver by means of a Deficient Diet', *Bulletin of the Johns Hopkins Hospital* 66 (1940), pp. 185-98.
[33] *Ibid.*, pp. 192-5.

their nine-year study on the effect of a wide variety of dietary factors on the livers of a total of 1,922 rats.[34] In addition to how a high-fat diet worked to enhance the production of cirrhosis, a deficiency of choline and methionine, compounds that encourage the breakdown of fats, was increasingly understood to be responsible for the development of cirrhosis.[35]

A new set of experiments on rats conducted by Floyd S. Daft, W. H. Sebrell, and R. D. Lillie at the National Institute of Health (NIH) at Bethesda, Maryland continued the research on the role of diet in the production of cirrhosis. In their first paper from 1941, a diet low in protein not only produced cirrhosis in rapidity, the introduction of a low-fat regimen with the addition of specific fat-related substances such as choline, methionine, and casein, so called 'lipotropic factors', seemingly prevented the production of the lesion.[36] The authors, therefore, argued that 'the diet here is the essential factor' in the production of cirrhosis, an explicit supposition that hepatic damage was primarily the result of nutritional deficiency and not alcohol.[37] The research group's second paper, titled 'cirrhosis of the liver in rats on a deficient diet and the effect of alcohol', looked into the pathological effects of alcohol over water on rat livers on a low protein diet. The paper not only observed that 'alcohol gives an additional insult to liver tissue injured by a dietary deficiency… No statement can be made at the present time as to the nature of the deficiency or deficiencies in this diet which permit or cause the development of hepatic cirrhosis.'[38] Here, the text was constructed on the assumption that cirrhosis was a disease of deficiency, while alcohol was represented as a secondary factor that merely exacerbated the disease. Although this was the first among similar studies to make such a claim, a later article by the same group of researchers cautioned against the universal applicability of this assumption when arguing that the 'experimental hepatic cirrhosis of rats

[34] Paul György, and Harry Goldblatt, 'Further observations on the production and prevention of dietary hepatic injury in rats', *The Journal of Experimental Medicine* 89.2 (1948) pp. 245-68.
[35] *Ibid.*, p. 265.
[36] Floyd S. Daft, W. H. Sebrell, and R. D. Lillie, 'Production and Apparent Prevention of a Dietary Liver Cirrhosis in Rats', *Experimental Biology and Medicine* 48.1 (1941), pp. 228-9.
[37] *Ibid.*, p. 228.
[38] R. D. Lillie, F. S. Daft, W. H. Sebrell Jr., 'Cirrhosis of the Liver in Rats on a Deficient Diet and the Effect of Alcohol', *Public Health Reports* 56.24 (1941), p. 1957.

has no counterpart in the usually described varieties of hepatic cirrhosis in man'.[39] The conclusion on the dietary nature of experimental cirrhosis was counterbalanced by a modest admission that the results at the time were applicable to animal subjects and not humans.

The successful production of cirrhosis in animals under a deficient diet further underlined the scientific legitimacy of the NDT. This was supported by a set of studies that produced marked improvements in the condition of the liver through dietary treatment. In a 1941 study on the 'treatment of dietary liver cirrhosis in rats with choline and casein', the NIH scientists observed that the consumption of a diet containing choline and casein led to an 'extensive regeneration of liver cells' after a biopsy revealed that 'fat had completely disappeared from the liver cells' in most of the subjects.[40] Based on this study, the authors stated that 'a clinical trial of choline and casein therapy in human liver cirrhosis should be considered.'[41] The suggestion was put into practice by Arthur J. Patek and Joseph Post. The pair of New York-based scientists conducted a clinical study on alcoholic patients with cirrhosis that made use of a highly nutritious diet containing abundant doses of vitamin B complex.[42] The research was carried out on an observation that a large proportion of the patients were clearly malnourished, some of whom had 'histories of having subsisted entirely on alcoholic liquor for intervals of several days and of having refused all food' during long periods of heavy consumption. The authors thus argued that alcohol was 'a predisposing factor rather than a primary etiologic agent' to cirrhosis, wherein its heavy consumption deprived the individual of an adequate diet by making up a large portion of their caloric intake.[43] The results of the study were remarkable. 72 per cent of the experimental group receiving a dietary regimen survived the first six months of the trial, compared to only 57 per cent of the control group from whom the regimen was

[39] R. D. Lillie, L. L. Ashburn, W. H. Sebrell, F. S. Daft and J. V. Lowry, 'Histogenesis and Repair of the Hepatic Cirrhosis in Rats Produced on Low Protein Diets and Preventable with Choline', *Public Health Reports* 57.14 (1942), p. 507.
[40] J. V. Lowry, Floyd S. Daft, W. H. Sebrell, L. L. Ashburn, and R. D. Lillie, 'Treatment of dietary liver cirrhosis in rats with choline and casein', *Public Health Reports* 56.46 (1941), pp. 2218-9.
[41] *Ibid.*, p. 2219.
[42] Arthur J. Patek, Jr., and Joseph Post, 'Treatment of Cirrhosis of the Liver by a Nutritious Diet and Supplements rich in Vitamin B Complex', *Journal of Clinical Investigation* 20.5 (1941), pp. 481-505.
[43] *Ibid.*, pp. 481-3.

withheld. By the end of the trial in its second year, 45 per cent of the experimental group survived as opposed to the 21 per cent of the control group.[44] Based on this long-term study, it was understood that a nutritious diet containing vitamin B complex concentrates was effective in treating cirrhosis. Whereas Patek and Post were cautious about passing judgements on the aetiology, stating that 'the relation of nutritional deficiency to cirrhosis is not clear' and 'could be either direct or indirect', the study aptly demonstrated that the life expectancy of patients suffering from cirrhosis could be lengthened when certain dietary requirements were met.[45] An improved prognosis upon the ingestion of a nutrient implied that a shortage of the specific nutrient might contribute to the causation the illness in the first place.

The perceived effectiveness of dietary treatment to cirrhosis was further strengthened after the Second World War. In 1948, Charles Best authored a paper with E. A. Sellers and C. C. Lucas in *The British Medical Journal* that observed that an addition of pure choline chloride to a minimal dietary regimen led to vast improvements in the livers of rats which had previously been damaged by the toxin, carbon tetrachloride. Based on this, the authors concluded that 'the presence of the lipotropic agents is essential for the repair of the damaged liver under the conditions of our experiments'.[46] The support for the dietary treatment of liver damage eventually reached to such a level of acceptance that some scientists even went as far to question whether if it was even necessary to abstain from alcohol to improve one's prognosis as long as the patient subsisted on a healthy diet. A team at Harvard University led by Gerald B. Phillips studied the variable effects of total abstinence and a diet containing choline on the liver of three alcoholic patients.[47] The paper noted marked improvements in the morphology of the livers, which they understood were 'related to the provision of an adequate diet and that a subsidiary, if any, role was played by withdrawal of alcohol and rest in the hospital.'[48] In spite of the

[44] *Ibid.*, p. 493.
[45] *Ibid.*, p. 493.
[46] E. A. Sellers, C. C. Lucas, and C. H. Best, 'The Lipotropic Factors in Experimental Cirrhosis', *The British Medical Journal* 1062 (1948), pp. 1064-5.
[47] Gerald B. Phillips, George J. Gabuzda Jr, and Charles S. Davidson, 'Comparative effects of a purified and an adequate diet on the course of fatty cirrhosis in the alcoholic', *Journal of Clinical Investigation* 31.4 (1952), pp. 351-6.
[48] *Ibid.*, p. 356.

problematic methodology of the study, which was based on an extremely limited sample of only three subjects, it made a radical supposition that entirely attributed cirrhosis to nutritional deficiency while discounting any role that alcohol may have played in directly damaging the liver.

By the end of the Second World War, the NDT was accepted as the dominant explanation for the aetiology of cirrhosis among the esoteric group of scientists who had a special interest in liver disease. The dominance of the NDT was signalled by a controversy surrounding one of the last papers from the period that actively opposed the tendency to blame liver disease among alcoholics to dietary factors. In 1947, C. T. Ashworth, a pathologist based in Texas, published a paper on the 'production of fatty infiltration of liver in rats by alcohol in spite of adequate diet'. As the title suggests, the article successfully produced fatty liver in rats by combining alcohol with an adequate intake of casein.[49] The subjects were divided into four groups: rats receiving a low casein diet with alcohol, rats receiving a high casein diet with alcohol, rats receiving a low casein diet without alcohol, and rats receiving a high casein diet without alcohol.[50] Among them, fatty liver developed in the first three groups that were exposed to either alcohol, a deficient diet, or both. The most significant changes were seen in the second group, where the presence of a high casein diet seemingly failed to prevent alcohol from causing fatty liver. This led to the supposition that alcohol was the 'only apparent variable' that 'exerts an effect which permits the accumulation of fat within the liver cells, and that this effect operates separately from that of extrinsic deficiency of lipotropic factors.'[51] In spite of this, Ashworth did not explicitly insist that alcohol should be identified as a direct toxin to the liver. Rather, he was intending to question the emerging consensus that attributed malnutrition as the primary cause of liver disease. Ashworth admitted that '[i]t remains to be shown whether the liver cell injury produced by alcohol is due to a direct toxic action, to the effect of the degradation products of alcohol, or whether alcohol interferes with the chemical processes

[49] C. T. Ashworth, 'Production of Fatty Infiltration of Liver in Rats by Alcohol in spite of adequate diet', *Experimental Biology and Medicine* 66.2 (1947), pp. 382-5.
[50] *Ibid.*, p. 383.
[51] *Ibid.*, pp. 384-5.

normally called into play in the metabolism of fat.'[52] Nevertheless, his paper was unusual at the time for challenging the emerging orthodoxy surrounding the NDT.

Two years later, Ashworth's provocative study received a direct rebuttal from Charles Best. In 1949, Best and his team of researchers at Toronto, including W. Stanley Hartroft, C. C. Lucas, and Jessie H. Ridout, published a paper titled 'liver damage produced by feeding alcohol or sugar and its prevention by choline' in *The British Medical Journal.*[53] The article began with a detailed dissection on Ashworth's problematic methodology. Best noted that all four groups of rats experienced drastic weight-loss in Ashworth's experiment, bringing into question whether the dietary regimen was truly adequate.[54] In addition, Ashworth's imprecise dosing of alcohol fed to the rats was suspected to have caused the rats to go into a comatose state.[55] The most problematic aspect of his paper was that it took no consideration of the likelihood that alcohol provided excess calories to the two experimental groups over the two control groups.[56] This would have distorted the total caloric intake of the subjects and may have inadvertently affected their livers. Best, therefore, improved on Ashworth's shortfalls by using an isocaloric pair-feeding method, ensuring similar total caloric intake. While the experimental group was fed an alcohol-rich diet, the control group consumed a diet that contained identical caloric portions of table sugar (sucrose) instead of alcohol.[57] The paper also included a study of the effects of the addition of various lipotropic substances, including choline, methionine, and casein, on the production of fatty liver in rats consuming either alcohol or sugar water. The authors hypothesised that the 'pathological changes in the liver produced in these experiments are attributed to an imbalance of calories and vitamins, particularly to an induced inadequacy of lipotropic factors consequent upon the increased calorie intake.'[58] They performed a

[52] *Ibid.*, p. 384.
[53] C. H. Best, W. Stanley Hartroft, C. C. Lucas, and Jessie H. Ridout, 'Liver Damage Produced by Feeding Alcohol or Sugar and its Prevention by Choline', *The British Medical Journal* 2.4635 (1949), pp. 1001-6.
[54] *Ibid.*, p. 1001; Ashworth, 'Production of Fatty Infiltration', p. 383.
[55] Best, Hartroft, Lucas, and Ridout 'Liver Damage', p. 1005; Ashworth, 'Production of Fatty Infiltration', p. 382.
[56] Best, Hartroft, Lucas, and Ridout 'Liver Damage', p. 1001.
[57] *Ibid.*, pp. 1001-2.
[58] *Ibid.*, p. 1002

complex experiment lasting 177 days that involved 12 different experimental (alcohol) and control (sugar) groups across 188 male rats.[59]

The results of the experiment showed that an inadequate supply of choline and methionine did indeed result in fatty liver, while their adequate supply protected the liver from damage. The hepatic damage in the sugar-consuming control group was 'so similar in character and extent to those produced by an isocaloric amount of alcohol that they are indistinguishable.'[60] These results led to the bold assertion that 'there is no more evidence of a specific toxic effect of pure ethyl alcohol up on liver cells than there is for one due to sugar.'[61] Although Best warned that his 'findings are, of course, not necessarily applicable to alcoholism in human subjects', his contribution consequently undermined the notion that alcohol was a direct toxin to the liver, instead strengthening the idea that it merely played an indirect role in increasing the choline requirement by augmenting one's caloric intake.[62]

The results of the above study were later replicated in 1954 by a team of hepatologists at Yale University led by Gerald Klatskin. They studied the potential role of alcohol in indirectly damaging the liver by reducing food consumption to the point of the deprivation of key lipotropic substances, a concept 'based on the well-known fact that malnutrition is a frequent complication of chronic alcoholism'.[63] Klatskin and his colleagues were able to produce a spectrum of liver diseases in rats that encompassed fatty liver and mild fibrosis.[64] Under a condition where all groups were fed a strict isocaloric diet akin to Best's experiment, Klatskin noticed that the condition of the liver was considerably worse when alcohol was ingested alongside a diet deficient in lipotropic factors, compared to those that consumed alcohol with an adequate diet.[65] It was thus argued that choline and methionine worked to 'abolish the effects

[59] *Ibid.*, pp. 1002-4.
[60] *Ibid.*, p. 1006.
[61] *Ibid.*, p. 1006.
[62] *Ibid.*, p. 1006.
[63] Gerald Klatskin, Willard A. Krehl, and Harold O. Conn, 'The effect of alcohol on the choline requirement I. Changes in the rat's liver following prolonged ingestion of alcohol', *The Journal of Experimental Medicine* 100.6 (1954), p. 605.
[64] *Ibid.*, pp. 605-614.
[65] *Ibid.*, pp. 613-4.

of both alcohol and sucrose supplements', leading to the conclusion that 'alcohol increases the choline requirement of the rat'.[66]

Ashworth's attempt to undermine the NDT as the primary explanation of cirrhosis unexpectedly resulted in the further legitimisation of the aetiological theory. Best's stark conclusion that alcohol was no more toxic to the liver than sugar water reverberated among other specialists in the field, shown by how the result of his article had become frequently referenced in subsequent publications as evidence that cirrhosis was caused by malnutrition.[67] Later in 1971, Paul Devenyi described Best's article as a 'classic work' in which 'the role of nutritional deficiencies that accompany alcoholism has been emphasized.'[68] It is also worth noting that the article was far more likely to have been read by medical professionals based in Britain over other studies because it was published in *The British Medical Journal.* As explored in the rest of this chapter, the NDT's wider influence outside of the restricted circle of scientists was evident not long after the Second World War.

The sheer complexity underlining how numerous experimental and clinical studies legitimised the NDT has not been properly accounted for by either Herd nor Katcher, both of whom choose to focus on the popularisation and appropriation of the theory without discussing its scientific origins.[69] Central to the dismissal of alcohol's direct toxicity to the liver was the general failure among laboratory studies to reproduce cirrhosis in animal livers with alcohol alone. On the other hand, a set of studies conducted between the 1930s to the 1950s were seemingly successful in producing liver damage through the ingestion of a diet deficient in key nutrients. The theory was further reinforced by the demonstration that an adequate dietary regimen could effectively treat severe liver damage by improving the prognosis of

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[66] *Ibid.*, p. 614.

[67] With 305 citations recorded on Google Scholar, Best, Hartroft, Lucas, and Ridout 'Liver Damage', pp. 1001-6 is the most cited article among all articles referenced in this section, according to 'Best: Liver damage produced by feeding alcohol or sugar and its prevention by choline', *Google Scholar*, https://scholar.google.co.uk/scholar?cites=3891102858103154 26&assdt=2005&sciodt=0,5&hl=en [ace- ssed 26 October 2017]. The paper was later cited in several influential literature reviews, including Sheila Sherlock, 'Cirrhosis of the Liver', *Postgraduate Medical Journal* 26.299 (1950), p. 473; Klatskin, 'Experimental studies', p. 440; Galambos, 'Progress Report', p. 477.

[68] Paul Devenyi, 'Alcoholic Liver Disease', *Canadian Family Physician* 17.7 (1971), p. 34.

[69] Herd, 'Ideology, history and changing models', p. 1114; Katcher, 'The Post-Repeal Eclipse', p. 736.

patients suffering from cirrhosis. The identification of dietary deficiency as the likely cause of cirrhosis was thus founded on a sound observation of the evidence available at the time. As a result, alcohol was largely dismissed as its cause, severely undermining the framing of cirrhosis as an 'alcoholic's disease' under the DTT.

E. M. Jellinek and the Zenith of the Nutritional Deficiency Theory

Having looked at the studies that contributed to the formation of the NDT, this section explores how the theory was legitimised for a broader group of alcohol researchers under the emergent conceptualisation of alcoholism as a 'disease'. The abandonment of alcohol as a direct toxin to the liver was promulgated in the early works of E. M. Jellinek, who was undoubtedly among the most influential alcohol researchers from the middle of the twentieth century. Jellinek's ideological interest in playing down the physiological harms of alcohol on behalf of a focus on alcoholism as a psychological condition is stressed by both Herd and Katcher to be the primary motivation behind his rejection of alcohol's direct toxicity to the liver.[70] Although Jellinek can correctly be understood as a key figure behind the popularisation of the NDT, the chief reasoning underlining his rejection of the direct toxicity theory, namely that scientists had largely failed to reproduce cirrhosis in animals with alcohol, was by no means novel. Jellinek thus argued that alcohol should be demoted to being understood as an indirect cause on the principle that, in order to establish disease causation, one must always demonstrate it through experimental means. Therefore, more so than his supposed ideological opposition to the exaggeration of alcohol's harms to the liver, Jellinek's objections were founded on perfectly reasonable scientific grounds. This interpretation is also in direct contradiction to Katcher's presentist judgement that Jellinek's decision to abandon the DTT was 'premature' on the grounds that the NDT turned out to be wrong several decades later.[71] The success of the NDT was also owed to the prestige and prominence of Jellinek within the international debate on alcohol use after the Second World War. Ironically, Jellinek's rejection of alcohol as the primary

[70] Herd, 'Ideology, history and changing models', p. 1117; Katcher, 'The Post-Repeal Eclipse', pp. 731-2.
[71] Katcher, 'The Post-Repeal Eclipse', pp. 731-2

cause of liver damage was declared simultaneously alongside his recognition of the existence of a strong statistical association between alcohol consumption and cirrhosis. This observation eventually culminated in the creation of the Jellinek formula, which estimated the prevalence of alcoholism in society by looking at cirrhosis mortality rates. This section focuses on several of Jellinek's earliest works on alcoholism and attempts to understand the reasons behind his rejection of alcohol as a direct toxin to the liver. It also assesses the wider impact of Jellinek's contribution to the scientific entrenchment of the NDT.

As correctly pointed out by Katcher, the end of federal prohibition in the United States with the repeal of the Eighteenth Amendment of the United States Constitution in 1933 was a major watershed in the history of American medical discourses surrounding alcohol.[72] These underwent their own rendition of New Moderationism whereby physicians and scientists dispelled the temperance movement's tendency to overstate the harms of alcohol consumption. Much like in Britain, interwar American texts critical of prohibition were sceptical of the notion that cirrhosis was the direct outcome of alcohol. An early critique predating the implementation of national prohibition was authored by John Koren in 1916, who outwardly attacked the temperance movement as 'equally unscientific and guilty of untruths' when it came to their claims on the effects of alcohol on the human body.[73] Koren added that the 'doctrine of the supremacy of alcohol among the factors in cirrhosis of the liver is largely mere assertion' and suggested 'that excessive drinking predisposes to this particular disease as to several others'.[74] Later, Haven Emerson, a renowned public health researcher from New York, edited a collection of articles in the form of *Alcohol and Man: The Effects of Alcohol on Man in Health and Disease* (1933), which challenged commonly held misunderstandings on alcohol that were disseminated to the public during the prohibition.[75] Concerning the aetiology of cirrhosis, two separate chapters in the book downplayed the causation of alcohol by arguing

[72] *Ibid.*, pp. 729-30.
[73] John Koren, *Alcohol and Society* (New York, 1916), p. 3.
[74] *Ibid.*, p. 37.
[75] Haven Emerson (ed.), *Alcohol and Man: The Effects of Alcohol on Man in Health and Disease* (New York, 1933); Katcher, 'The Post-Repeal Eclipse', pp. 729-30; Charles Bolduan, 'Haven Emerson, the public health statesman', *American Journal of Public Health and the Nation's Health* 40.1 (1950), pp. 1-4.

that 'there are some pathologists who firmly state that alcohol is not responsible for the disease' and that alcohol 'may make the liver more vulnerable to other harmful agents.'[76] Perhaps the most radical of all the anti-prohibition literature was *Liquor: The Servant of Man* (1940), authored by two American physicians. The book argued that '[t]he conclusion by practically all unbiased workers is that even the extensive use of alcohol in itself does not cause liver cirrhosis.'[77] This assertion was part of the book's overarching assumption that '[t]here has been found no evidence that prolonged and copious use of beverage alcohol causes *any disease*', and that alcohol, 'if taken in anything remotely approaching customary amounts, is harmless to the body and in many cases beneficial.'[78] The rejection of alcohol's direct toxicity to the liver was evidently entrenched in the moderationist discourse in the United States as it was in Britain.

It was within these intellectual circumstances of the post-prohibition era where Jellinek made his contribution to the knowledge on alcohol. In 1939, Jellinek was introduced to the world of alcohol research through Norman Jolliffe, a psychiatrist based in Bellevue Hospital in New York City. Jolliffe received a generous grant from the Research Council on Problems of Alcohol to complete an extensive review of the existing scientific knowledge on the impact of alcohol on the human body. To head this project, he hired Jellinek as the lead director and investigator.[79] Jolliffe himself was already renowned for his work on diseases of deficiencies that were commonly observed in malnourished alcoholics.[80] He famously demonstrated that polyneuritis, a painful inflammation of the peripheral nerves, was caused by the shortage of

[76] George B. Wallace, 'The Pharmacological actions of alcohol', and Harrison S. Martland, 'The Pathology of acute and chronic alcoholism', in Emerson, *Alcohol and Man*, pp. 56-7, 220.
[77] Walton Hall Smith, and Ferdinand C. Helwig, *Liquor: The Servant of Man* (Boston, 1940), p. 34.
[78] *Ibid.*, pp. 67, 221.
[79] Penny B. Page, 'Jellinek, Elvin Morton (1890-1963)', in Blocker, Jr., Fahey, and Tyrrell (eds.), *Alcohol and Temperance in Modern History*, pp. 338-9
[80] Norman Jolliffe, and Robert Goodhart, 'Beriberi in Alcohol Addicts', *The Journal of the American Medical Association* 111 (1938), pp. 380-4; Goodhart, and Jolliffe, 'The Role of Nutritional Deficiencies in the Production of Cardiovascular Disturbance', pp. 569-81; Norman Jolliffe, 'The Influence of alcohol on the adequacy of the B vitamins in the American diet', *Quarterly Journal of Studies on Alcohol* 1 (1940), pp. 74-84; Norman Jolliffe, 'Treatment of neurophychiatric disorders with vitamins', *The Journal of the American Medical Association* 117 (1941), pp. 1496-500.

vitamin B1 and not, as it had been previously believed, alcohol.[81] Continuing this line of research, he was interested in how many of the diseases that were tied to alcohol dependence turned out to be nutritional in nature. These included pellagra and scurvy, deficiency diseases still understood as such today.[82] To further this investigation, Jolliffe joined Jellinek as the first author in a key section of the influential review that discarded alcohol's direct role in damaging the liver.

In 1941, the two researchers authored 'vitamin deficiencies and liver cirrhosis in alcoholism' as an article in the newly established *Quarterly Journal of Studies on Alcohol*, of which Jellinek was appointed as an associate editor.[83] The paper was part of a wider cohort of reviews that analysed a total sum of three thousand empirical studies on alcohol's relation to various bodily disturbances.[84] As noted by both Herd and Katcher, the article was the most significant exposition of the rejection of alcohol's direct toxicity to the liver, thereby allowing the NDT to emerge as the most likely explanation of the pathogenesis of cirrhosis.[85] It also legitimised the reputation of the interdisciplinary journal among alcohol researchers, publishing several of the most important papers in the field across the next decade under the editorship of Howard Haggard. The article was ambitious in its scope, studying almost two hundred articles on the topic that encompassed literature reviews, population studies, clinical studies, and animal experiments.

Jolliffe and Jellinek's review opened with an observation that the liver 'occupied a paramount role in research in alcoholism' but continued to be 'the most misunderstood issue in alcohol literature.'[86] The early half of the article discussed the clinical and autopsy studies that had touched on a variety of questions, including the controversy surrounding the

[81] Norman Jolliffe, C. N. Colbert, and Phillip M. Joffe, 'Observations on the etiologic relationship of vitamin B (B1) to polyneuritis in the alcohol addict', *American Journal of Medical Sciences* 191 (1936), pp. 515-26; Apple, *Vitamania*, p. 5. Today, polyneuropathy, much like cirrhosis, is known to be caused by the direct action of alcohol.
[82] Goodhart, and Jolliffe, 'The Role of Nutritional Deficiencies in the Production of Cardiovascular Disturbance', p. 596.
[83] Jolliffe, and Jellinek, 'Vitamin Deficiencies', pp. 544-83.
[84] Herd, 'Ideology, history and changing models', p. 1117; Penny Booth Page, 'E. M. Jellinek and the evolution of alcohol studies: a critical essay', *Addiction* 92.12 (1997), p. 1620.
[85] Herd, 'Ideology, history and changing models', p. 1117; Katcher, 'The Post-Repeal Eclipse', pp. 731-2.
[86] Jolliffe, and Jellinek, 'Vitamin Deficiencies', p. 544.

commonality of cirrhosis among chronic alcoholics, as well as the proportion of cirrhosis deaths attributable to the causation of alcohol.[87] The authors observed that cirrhosis was on average seven times as common among chronic alcoholics than in the rest of the abstinent and moderate-drinking population.[88] The paper was also drawn to statistical studies that looked at the prevalence of cirrhosis in the general population across certain periods in history when the distribution of alcohol was dramatically curtailed. For instance, outcomes of the federal ban on alcohol under the American prohibition, with its enactment resulting in falling *per capita* levels of consumption and its subsequent repeal increasing them, was understood to have noticeably affected the incidence of alcohol-related diseases in the United States.[89] Jolliffe and Jellinek juxtaposed changing mortality rates from cirrhosis alongside those of other non-alcoholic diseases to better understand the role played by the availability of alcohol. Following the implementation of prohibition, both cirrhosis and tuberculosis mortality rates decreased. However, following its repeal, cirrhosis mortality rates began to rise again while tuberculosis mortality rates continued to fall, clearly indicating that the availability of alcohol played some sort of role in the incidence of cirrhosis.[90] The same phenomenon was observed in England and Wales during the First World War among women, who, unlike men, were not mobilised in large numbers to the frontlines. The rapid rise in the price of alcohol from wartime disruption and from the subsequent restrictions imposed on the liquor trade under the CCB led to a decline in both *per capita* levels of consumption and deaths from cirrhosis.[91] Thus, the authors recognised that 'a significant association exists between changes in the death rate from cirrhosis of the liver and the changes in the consumption of alcoholic beverages'. They added that the 'consistency of these phenomena definitely indicates that a certain portion of deaths from cirrhosis of the liver is related to alcoholic habits.'[92] These observations inadvertently

[87] *Ibid.*, pp. 546-52.
[88] *Ibid.*, p. 562.
[89] *Ibid.*, p. 558.
[90] *Ibid.*, pp. 558-60.
[91] *Ibid.*, pp. 561-2.
[92] *Ibid.*, pp. 558, 561.

strengthened the association between alcohol consumption and cirrhosis, the culmination of which was the 'Jellinek formula', explored in greater detail later in this section.

The second half of the review turned its attention to the experimental literature. Following a review of a large collection of studies published between 1887 and 1939, among which methodologies widely varied in their choice of experimental animal, the volume of the alcohol ingested, and the duration of the experiments, Jolliffe and Jellinek reached a surprising conclusion that 'neither an acceptance nor a rejection of the alcoholic etiology is permissible.'[93] While the direct causation of alcohol was understood to be largely unsubstantiated by the experimental literature, they argued that it was equally difficult to entirely disregard alcohol as a cause due to the inconsistent methodology of the studies.[94] The paper then pointed to the emergence of a new understanding that suggested that cirrhosis might be caused by a deficiency in key nutrients, which in turn was indirectly caused by alcoholism. It referred to a paper by Connor and Chaikoff, which established fatty liver as a necessary precondition for cirrhosis, as one of 'the most important advances in the knowledge of the pathogenesis of cirrhosis of the liver in chronic alcoholism'.[95] At a point where they completed their paper before its publication in 1941, the authors endorsed Connor's research as an inspiration to take the inquiry into the aetiology of cirrhosis further.[96] Rather than directly endorsing the new nutritional model of cirrhosis aetiology, Jolliffe and Jellinek concluded that, while 'direct causation through alcohol is ruled out, none of the numerous etiological theories of indirect causation can be accepted at present as sufficiently documented.'[97]

The paper's neutral stance on the exact aetiology of cirrhosis was balanced out by the following passage that transpired to be Jolliffe and Jellinek's most profound contribution to the debate.

[93] Jolliffe, and Jellinek, 'Vitamin Deficiencies', pp. 568-71.
[94] *Ibid.*, pp. 567, 571-3.
[95] *Ibid.*, p. 580; Connor, and Chaikoff, 'Production of Cirrhosis'.
[96] Jolliffe, and Jellinek, 'Vitamin Deficiencies', p. 580; Connor, 'The etiology and pathogenesis'.
[97] Jolliffe, and Jellinek, 'Vitamin Deficiencies', p. 580.

> Even the most valid statistics, vital or clinical, cannot go beyond the point of indicating the association or non-association 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 from 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. Etiological theories must be based on physiological experiment and physiological reasoning.[98]

The authors essentially articulated that a statistical association, no matter how close it may be, is still no evidence of causality, eloquently building up to an assertion that disease aetiology must always be demonstrated through experimentation. The passage embodied a principle in causal inference that was widely held at the time by many physicians.

The scientific supremacy of the laboratory has deep roots in the development of modern scientific medicine. Experimental medicine was conceived in the middle of the nineteenth century by French physiologist Claude Bernard, who promoted animal testing as a standard means to understand the biomedical phenomena. In contrast to the use of human participants in a clinical trial, laboratory experiments provide a strictly controlled environment where variables are freely testable and measurable.[99] Concerning the use of model organisms in understanding human physiology, Bernard argued that 'experiments on animals are entirely conclusive for the toxicology and hygiene of man. The effects of these substances are the same on man as on animals, save for differences in degree.'[100] Later, the centrality of experimentation to aetiology was further promoted by Koch, who postulated that the specific microorganism must be experimentally isolated within the diseased subject in order for causality to be

[98] *Ibid.*, p. 566.
[99] Porter, *The Greatest Benefit*, pp. 341-39.
[100] Quoted in Rachel Hajar, 'Animal Testing and Medicine', *Heart Views* 12.1 (2011), p. 42.

established between an infective microorganism and a disease.[101] This principle was immediately applied to understand the causation of non-infectious diseases. The causative agent should be manifest—and this was the case for cirrhosis, based on its clear association to alcoholic excess. Furthermore, another of Koch's postulates stated that the putative causative agent should actually cause the disease when introduced to a healthy subject.[102] This specific postulate was not met in regard to cirrhosis: the introduction of alcohol in an experimental animal seemingly failed to produce the disease. Insofar as the experimental phenomena observable in animals were still applicable to humans, laboratory experiments were deemed essential to demonstrating a causative relationship between an agent and a disease.

Jolliffe and Jellinek's emphasis on the importance of the experimental demonstration of the aetiology of cirrhosis contrasted to their relative indifference towards epidemiology. The poverty of epidemiological evidence in the realm of medicine in the 1940s has been the subject of the historiography on the discovery of an association between cigarette smoking and lung cancer by Doll and Hill in the 1950s. Allan Brandt, John Burnham, and Colin Talley et al. refers to the Doll and Hill case as a watershed when population studies began to be taken seriously in medicine as a suitable method to understand the relationship between a disease and its purported cause.[103] According to Dorothy Porter, epidemiology achieved a 'new legitimate authority' in post-Second World War medicine as a result of the lung cancer studies.[104] This development was further accentuated by the formation of the Bradford Hill criteria in 1965, which deemed experimental evidence non-essential to establishing causality as long as the association met a set of rigorous standards, such as the idea that greater exposure to the presumed cause should result in greater incidence of the disease.[105] When one takes into

[101] Porter, *The Greatest Benefit*, p. 432.
[102] *Ibid.*, p. 432.
[103] Brandt, 'The Cigarette, Risk and American Culture', pp. 115-76; Burnham, 'American Physicians and Tobacco Use: Two Surgeons General', pp. 1-31l; Talley, Kushner, and Sterk, 'Lung Cancer, Chronic Disease Epidemiology, and medicine', p. 332.
[104] Dorothy Porter, *Health, Civilization and the State: A history of public health from ancient to modern times* (Abington, 1999), p. 300.
[105] Nicola Crichton, and Anne Mulhall, 'Epidemiology and health', in Jennie Naidoo, and Jane Willis (eds.), *Health Studies: An Introduction* (2nd edn, Basingstoke, 2008), p. 93. Hill positioned experimental demonstration as the eighth most important criteria in establishing causation, outlining that '[o]ccasionally it is possible to appeal to

account the intellectual environment prior to Doll and Hill, when medical scientists were still inclined to acknowledge the necessity of animal testing under Koch's postulates, the seemingly paradoxical nature of Jolliffe and Jellinek's passage—that, although the association between alcohol and cirrhosis was self-evident, the absence of experimental evidence meant that direct causation could not be established—does not seem too far-fetched.

Jolliffe and Jellinek's review article is understood by Herd and Katcher to be the cornerstone study that discredited the DTT, allowing the NDT to emerge as the predominant theory.[106] The paper was influential among medical scientists who specialised in cirrhosis aetiology. Alongside Moon's 1934 literature review on the aetiologies of cirrhosis and Best's 1949 sugar water experiment, Jolliffe and Jellinek's review was widely cited as a standard reference to demonstrate the assertion that alcohol merely played an indirect role in damaging the liver.[107] In his 1971 review, Devenyi identified the paper as 'one of the most often quoted references' concerning the indirect role of alcohol in the causation of cirrhosis. As it will be discussed in chapter 4, the review also sparked a separate debate over whether an association even existed between alcohol and cirrhosis.[108]

Additionally, it is worth noting that a large part of the paper's positive reception and impact could be attributed to the authority and stature of Jellinek in alcohol research after the

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experimental, or semi-experimental, evidence' in Austin Bradford Hill, 'The Environment and Disease: Association or Causation?', *Proceedings of the Royal Society of Medicine* 58.5 (1965), p. 298.

[106] Herd, 'Ideology, history and changing models', p. 1117; Katcher, 'The Post-Repeal Eclipse', pp. 731-2.

[107] Boles, Crew, and Dunbar, '"Alcoholic Cirrhosis"', p. 670; Klatskin, 'The role of Alcohol', p. 23; Klatskin, 'Effect of alcohol on the liver', p. 1671; John R. Seeley, 'Death by liver cirrhosis and the price of beverage alcohol', *Canadian Medical Association Journal* 83 (24 December 1960), p. 1366; Devenyi, 'Alcoholic Liver Disease', p. 34; Feinman, and Lieber, 'Liver disease in alcoholism', p. 303; Werner K. Lelbach, 'Cirrhosis in the alcoholic and its relation to the volume of alcohol abuse', *Annals of the New York Academy of Sciences* 252.1 (1975), p. 85; M. Harvey Brenner, 'Trends in Alcohol Consumption and Associated Illnesses', *The American Journal of Public Health* 65.12 (1975), p. 1279; Salaspuro, and Lieber, 'Alcoholic liver disease', in Wright, Alberti, Karran, and Millward-Sadler (eds.), *Liver and Biliary Disease*, p. 735; Conn, and Atterbury, 'Cirrhosis', in Schiff, and Schiff (eds.), *Diseases of the Liver*, p. 737.

[108] Abraham M. Lilienfeld, and Robert F. Korns, 'Some Epidemiological Aspects of Cirrhosis of the Liver: A Study of Mortality Statistics', *American Journal of Epidemiology* 52.1 (1950), p. 65; Milton Terris, 'Epidemiology of Cirrhosis of the Liver: National Mortality Data', *American Journal of Public Health and the Nation's Health* 57.12 (1967), p. 2076; Kettil Bruun, Griffith Edwards, Martti Lumio, Klaus Mäkelä, Lynn Pan, Robert E. Popham, Robin Room, Wolfgang Schmidt, Ole-Jørgen Skog, Pekka Sulkunen, and Esa Österberg, *Alcohol Control Policies in Public Health Perspective* (Forssa, 1975), p. 43.

Second World War. Jellinek was responsible for the establishment of what was to become the most important institute for interdisciplinary research on alcohol throughout the next two decades. The Yale Centre for Alcohol Studies emerged as the harbinger of research, professional training, and the public dissemination of knowledge on alcoholism. Jellinek's collaboration with Alcoholics Anonymous through the Yale Plan Clinics additionally allowed him to become as an authority figure in the research on the treatment of alcoholism and its promotion as a 'disease'. His subsequent appointment as a consultant to the World Health Organisation further propelled his international reputation in spreading awareness on the problems associated with drink.[109]

A year after the publication of his literature review with Jolliffe, Jellinek co-authored a monograph with Haggard titled *Alcohol Explored*, a popular summary of the existing knowledge on alcohol and its effects on the body that increased Jellinek's public reputation as an alcohol researcher.[110] The book simplified and expanded on the ideas promoted in their previous set of reviews for *The Quarterly Journal of Alcohol Studies*, and contained some of the most far-reaching statements on alcohol-related harms. The authors remarked that 'the most striking fact in regard to the bodily diseases of chronic alcoholism is that none are specifically limited to individuals who use alcohol in excess; the diseases may, and do occur in those who use no alcohol. They are not, therefore, the direct result of poisoning by alcohol'.[111] Arguing that 'the habit of inebriety is a contributing factor' to such diseases, this passage inferred that *all* physiological diseases believed to be caused by alcohol were only associated with it indirectly.[112] Furthermore, Haggard and Jellinek went on to make an even more extraordinary generalisation that '[t]he diseases of chronic alcoholism are essentially nutritional disturbances'.[113] This soundbite was followed by a disclaimer that the discovery of the nutritional origins of many of the physiological diseases of alcoholism had 'led to curious

[109] Page, 'E. M. Jellinek and the evolution of alcohol studies', p. 1619.
[110] Howard W. Haggard, and E. M. Jellinek, *Alcohol Explored* (New York, 1942); Page, 'E. M. Jellinek and the evolution of alcohol studies', p. 1621-2.
[111] Haggard, and Jellinek, *Alcohol Explored*, p. 106.
[112] *Ibid.*, p. 106.
[113] *Ibid.*, pp. 177-8.

conclusions among both those who strongly champion alcohol' by inferring that alcohol was, therefore, completely harmless.[114] Jellinek was thus well aware of the dangers of alcohol abuse and recognised that illnesses that were indirectly caused by heavy drinking can be as much of a hazard as those that were directly caused by it.

Concerning cirrhosis, Haggard and Jellinek pointed out that animal experiments had 'no success except in demonstrating the fact that cirrhosis is not due to the direct action of alcohol' and that 'many physicians have suggested that there is no connection at all between inebriety and cirrhosis of the liver'.[115] However, the book refrained from fully endorsing deficiency as being responsible for the disease by modestly admitting that there 'is as yet no certain knowledge of what causes cirrhosis of the liver in the inebriate'.[116] In a volume of the Yale Centre's *lay supplement* pamphlets, written for educational purposes, Jellinek cautiously pointed out that cirrhosis 'may, in part at least, be due to lack of vitamins'.[117] Much like Jolliffe and Jellinek's review, *Alcohol Explored* avoided the pitfalls of decisively validating the NDT, even if their generalisation that the 'diseases of chronic alcoholism are essentially nutritional disturbances' contributed to the abandonment of alcohol as a direct cause of cirrhosis alongside a wider effort to downplay the toxic effects of alcohol on the body. This was likely intended to open the path for other medical scientists to continue their investigation into other potential aetiologies of liver disease.

Ironically, aside from his critique of alcohol's direct toxicity to the liver, Jellinek's other contribution in his review with Jolliffe was the recognition of a strong statistical association between the consumption of alcohol and the incidence of cirrhosis. As a by-product of this observation, Jellinek suggested that it might be possible to calculate the prevalence of alcoholism in a society when taking into account statistical data on the incidence of cirrhosis. In his new position as a consultant for the WHO, Jellinek devised what came to be known as

[114] *Ibid.*, pp. 177-8.
[115] *Ibid.*, p. 189.
[116] *Ibid.*, p. 192.
[117] E. M. Jellinek, *Alcoholic Beverages as a Food and their Relation to Nutrition: Lay Supplement No. 8* (New Haven, CT, 1942), p. 4.

the 'Jellinek estimation formula', or simply the 'Jellinek formula', in 1951. Jellinek argued that if one could determine the proportion of alcoholics who developed cirrhosis within a country, an estimate of the number of alcoholics could be calculated by dividing the number of cirrhosis sufferers by this proportion.[118] For instance, since roughly 25 per cent of alcoholics in the United States suffered from complications relating to alcohol, the number of alcoholics in society can be estimated by dividing the number of those suffering from alcohol-related complications by one quarter.[119] The Jellinek formula was immediately put to use by the WHO to calculate the epidemiology of alcoholism in multiple countries across the world.[120] Although it received a barrage of criticism from its inception, with Jellinek himself admitting the methodological flaws of the formula, it was nevertheless the only scientific means available at the time to measure the incidence of alcoholism.[121] This was one instance when the scientific debate on alcohol and liver disease had a much broader influence outside of the limited confines of the medical sciences.

The impact of the scientific acceptance of the NDT and Jellinek's rejection of alcohol's direct toxicity to the liver can be further appreciated from the literature that followed. Some reviews attempted to disassociate cirrhosis from alcohol entirely when referring to the two as a relationship founded on direct causation. Helen Marshall's 1941 literature review of 135 articles published between 1929 and 1940 on alcohol's various effects on the human body omitted any reference to liver disease under a section titled the 'physiological effects of alcohol', only mentioning 'blood, brain, kidneys' as the primary victims of the toxic action of alcohol.[122] Karsner's overview of the various typologies of liver cirrhosis included 'Laennec's cirrhosis', 'fatty cirrhosis', 'biliary cirrhosis', and so on, but not 'alcoholic cirrhosis', arguing the 'so-called alcoholic cirrhosis' must be disregarded since '[i]t is now realized that alcohol conditions other

[118] Page, 'E. M. Jellinek and the evolution of alcohol studies', p. 1626.
[119] E. M. Jellinek, and M. Keller, 'Rates of alcoholism in the United States of America, 1940-1948', *Quarterly Journal of Studies on Alcohol* 13 (1952), pp. 49-59.
[120] Page, 'E. M. Jellinek and the evolution of alcohol studies', p. 1626.
[121] Robin Room, 'Alcohol Control and Public Health', *Annual Review of Public Health* 5 (1984), p. 295.
[122] Helen Marshall, 'Alcohol: a critical review of the literature, 1929-1940', *Psychological Bulletin* 38.4 (1941), pp. 196-200.

factors that lead to cirrhosis instead of being a direct cause.'[123] Similarly, Abraham Wilensky's review on 'the pathogenesis and mechanism of cirrhosis of the liver' argued that '[t]he term alcoholic cirrhosis mentioned only to be condemned because it indicates a causal relationship which is not satisfactorily established'.[124] The demotion of alcohol as a mere indirect factor in the causation of cirrhosis precipitated a shift in the language surrounding cirrhosis. By then, the previously dominant framing of cirrhosis as an 'alcoholic's disease' was abandoned altogether.

Thanks in part to Jellinek, the NDT established its hegemony in the medical sciences. Several important literature reviews after 1950 acknowledged deficiency as the primary cause of the disease. Sheila Sherlock, at the time a young consultant working at the Hammersmith Hospital in London who eventually became one of the key pioneers in hepatology, wrote a detailed piece in the *Postgraduate Medical Journal* on the existing medical understandings on cirrhosis. In it, she extrapolated from Best's 1949 paper and Jolliffe and Jellinek's 1941 review that 'alcohol poisoning alone will not lead to cirrhosis, and the relation between alcoholism and cirrhosis is probably indirect.' In explaining the actual mechanism of the causation of cirrhosis, Sherlock articulated that, since '[m]ost alcoholics take a poor general diet which is particularly deficient in protein and lipotropic factors… [a]n imbalance between caloric intake and the supply of essential food factors results'. Therefore, 'cirrhosis is dietetic' and can be 'prevented by ensuring that alcoholics took not only adequate calories (alcohol) but also essential amino acids and lipotropes.'[125] Similarly, Charles S. Davidson's 'profile' of cirrhosis from 1957 attributed the disease to the deficiency of choline and methionine.[126] Mark Keller also tied cirrhosis and other conditions relating to alcohol to a 'faulty nutrition rather than the direct action of alcohol on organs and tissues'.[127] By the 1950s, virtually all scientists with a stake in

[123] Karsner, 'Morphology and Pathogenesis', pp. 571, 576.
[124] Abraham O. Wilensky, 'The Pathogenesis and Mechanism of Cirrhosis of the Liver', *American Journal of Digestive Diseases* 13.11 (1946), p. 367.
[125] Sherlock, 'Cirrhosis', pp. 473-4.
[126] Charles S. Davidson, 'Cirrhosis of the Liver', *Disease-a-Month* 3.9 (1957), p. 16.
[127] Mark Keller, 'Alcoholism: Nature and Extent of the Problem', *Annals of the American Academy of Political and Social Science* 315 (1958), p. 8.

the issue seemingly acknowledge malnutrition to be the primary cause of liver disease in the place of alcohol.

In summary, Jellinek and his colleagues at the Yale Centre for Alcohol Studies played a crucial role in the legitimisation and the popularisation of the NDT as a scientific consensus. However, in contrast to what Herd and Katcher understood as Jellinek's ideological interests in downplaying the toxic properties of alcohol against the crumbling edifice of temperance science in post-prohibition America, a careful analysis of Jolliffe and Jellinek's 1941 literature review and Haggard and Jellinek's 1942 monograph reveals that alcohol's direct toxicity to the liver was rejected owing to the theory's insufficient experimental foundations.[128] This objection was founded on a predominant principle at the time that, no matter how close the statistical association, disease aetiology can only be established when demonstrated through animal experimentation. Aside from his critique of the experimental basis of direct causation, Jellinek's conclusions paradoxically strengthened the association between alcohol and cirrhosis, contributing to the creation of the Jellinek formula. Although nutritional deficiency was not explicitly endorsed by Jellinek and his colleagues as the definitive explanation of cirrhosis aetiology, the scientific hegemony of the NDT after the Second World War was partially attributable to the international prestige of Jellinek in the field of alcohol research.

The Nutritional Deficiency Theory and the British Medical Profession

Having traced the scientific shift towards the NDT on the aetiological understanding of cirrhosis, it is now time to explore how the new consensus was received among medical practitioners in postwar Britain through the examination of textbooks of general medicine and general medical journals published between the early 1940s and the late 1960s. These are useful primary sources that show an understanding of the content of the knowledge that were disseminated across the medical profession in Britain. The association of nutritional deficiency to cirrhosis was omnipresent across both texts, even if only a minority of them went as far as to state that cirrhosis was unequivocally caused by nutritional deficiency. There was a tendency

[128] Herd, 'Ideology, history and changing models', p. 1117; Katcher, 'The Post-Repeal Eclipse', pp. 731-2.

to point out that malnutrition was the most probable, rather than a definite, cause of cirrhosis, often by referencing many of the experimental research discussed in the first section of this chapter. This was in reflection of the growth of scientific restraint within the language found in many of the medical texts, especially textbooks. Ultimately, the widespread influence of the NDT meant that virtually all had abandoned the notion that alcohol was a direct toxin to the liver.

As a reliable category of texts to understand the predominant medical knowledge within a certain period, I have taken a look at multiple editions of 10 different series of textbooks, a total of 32 publications, from between 1941 and 1970 to study the reception of the NDT (see Figures 6; see Appendix: Textbooks of General Medicine for long-term shifts). Among the 10 textbooks, two are previously unmentioned in this thesis: T. R. Harrison's *Principles of Internal Medicine* and Stanley Davidson's *The Principles and Practice of Medicine.*[129] Both textbooks were chosen because of their high usage and popularity among British medical professionals. Since its original publication, Harrison's *Principles of Internal Medicine* has maintained its high reputation.[130] A 1983 review of its 10th edition described it as 'among the most popular postgraduate texts' alongside Cecil's *A Textbook of Medicine,* while another review praised it as 'essential reading for hospital physicians'.[131] Davidson's *Principles and Practice of Medicine* caught the attention of some reviewers when it was first published in 1952 as a collaborative product within medical school at the University of Edinburgh.[132] Later

[129] T. R. Harrison, Paul B. Beeson, George W. Thorn, William H. Resnik, and M. M. Wintrobe (eds.), *Principles of Internal Medicine* (1st edn, London, 1951); L. S. P Davidson (ed.), *The Principles and Practice of Medicine: A Textbook for Students and Doctors* (1st edn, Edinburgh, 1952).
[130] Preeti N. Malani [review], 'Dan Longo, Anthony Fauci, Dennis Kasper, Stephen Hauser, and Joseph Loscalzo (eds.), *Harrison's Principles of Internal Medicine* (New York, 2011)', *JAMA* 308.17 (7 November 2012), p. 1813.
[131] Paton [review], 'D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrel', pp. 1030-1; C. W. H. Havard [review], 'Kurt J. Isselbacher, Raymond D. Adams, Eugene Braunwald, Joseph B. Martin, Robert G. Petersdorf, and Jean D. Wilson (eds.), *Update IV: Harrison's Principles of Internal Medicine with CME Examination* (New York, 1983)', *The British Medical Journal* 287.6389 (6 August 1983), p. 417.
[132] L. J. Witts [review], 'Stanley Davidson, The Principles and Practice of Medicine (Edinburgh, 1952)', *The British Medical Journal* 2.4797 (13 December 1952), p. 1297.

reviews of the textbook commented on how it was 'widely used by students preparing for the final examination' and that it 'has justifiably become a classic'.[133]

The noticeable trend across much of the textbooks published between 1941 and 1970 was the diminishing emphasis on alcohol in their descriptions of the aetiology of cirrhosis. Compared to 71 per cent of all textbooks from the Edwardian era (1900-1914) and 38 per cent from the First World War and the interwar period (1915-1940), not a single textbook in this chapter labelled alcohol as a 'cause' or a 'direct cause' without referring to the complexities of its precise aetiology, especially over the continued lack of knowledge over alcohol's exact mechanism on the organ.[134] In contrast, alcohol was often understood as either a 'factor' or an 'indirect factor' to the disease in 68 per cent (22 out of 32) of all postwar textbooks, a marked shift compared to the 33 per cent of textbooks in the interwar period and 7 per cent in the Edwardian period with a similar description. The most peculiar postwar development was the emergence of publications that refrained from mentioning alcohol at all in relation to cirrhosis (16 per cent; 5 out of 32), whereas every single textbook published between 1900 and 1940 referred to alcohol in some form or another. Although most textbooks from the postwar era continued to discuss the causation of cirrhosis alongside alcohol, its diminishing importance was reflected in its general disregard as a direct toxin to the liver.

[133] D. V. Hubble [review], 'Stanley Davidson, *The Principles and Practice of Medicine* (3rd edn, Edinburgh, 1956)', *The British Medical Journal* 1.5021 (30 March 1957), p. 751; Anon. [review], 'John MacLeod (ed.), *Davidson's Principles and Practice of Medicine* (12th edn, Edinburgh, 1978)', *Postgraduate Medical Journal* 54.637 (September 1978), p. 637.

[134] The possible exception may be the 1952 and 1963 editions of Wheeler's *Handbook of Medicine* where it is subtly implied that deficiency makes the liver more vulnerable to 'the action of these slow poisons' such as alcohol, though even here, alcohol is understood to work in tandem with other factors in damaging the liver. Robert Coope, *Wheeler and Jack's Handbook of Medicine* (11th edn, Edinburgh, 1952), p. 266; Robert Coope, and C. A. Clarke, *Wheeler and Jack's Handbook of Medicine* (12th edn, Edinburgh, 1963), p. 283.

FIGURES 6 Textbooks of General Medicine, 1941~1970

	1941~45	1946~50	1951~55
William Osler, *The Principles and Practice of Medicine*	14th (1942) Cirrhosis 'occurs most frequently' in whiskey, gin, and brandy drinkers but 'not always due to alcohol' and can be caused by syphilis	16th (1947) ' just how the alcohol causes cirrhosis remains unknown' while 'there is now a considerable volume of evidence pointing to nutritional deficiency as of greatest importance in etiology of cirrhosis'	
Alexander Wheeler, *Handbook of Medicine*		11th (1952) liver 'made more vulnerable to the action of these slow poisons (like alcohol) by nutritional deficiencies (whereby it loses the protective action of adequate protein and glycogen and possibly the vitamin B complex), or by earlier infections or poisonings (e.g. syphilis, infectious hepatitis, treatment with arsenicals).'	
Thomas Dixon Savill, *A System of Clinical Medicine*		13th (1950) 'Alcohol undoubtedly predisposes to atrophic cirrhosis' and 'dietetic factor may play a part'	
Russell Cecil, *A Textbook of Medicine*	6th (1944) 'Clinical and experimental studies strongly suggest that a direct relationship exists between nutritional deficiency and the development of portal cirrhosis. Whether alcoholism... simply predisposes to the dietary deficiency... or whether it lends an added toxic effect is not clearly established.'	7th (1947) while ' in the large majority of cases there is no known hepatotoxin', experimental studies strengthen '[t]he possibility that nutritional deficiency might play an etiologic role'	
Frederick W. Price, *A Textbook of the Practice of Medicine*		7th (1947) alcohol probably produces cirrhosis 'only when the liver is abnormally vulnerable owing to constitutional inferiority, vitamin or other dietetic deficiencies, and the simultaneous or previous action of the toxins of such infections as syphilis, malaria, amoebiasis, and the virus of infective hepatitis' and 'cirrhosis very rarely develops experimentally in animals	
John Conybeare, *A Textbook of Medicine*		9th (1949) 'The aetiology is not understood, but it is possible that it depends on a combination of factors which have to do with qualitative dietary insufficiencies... Alcohol, in producing a chronic gastritis, would thus play a definite role in augmenting this process in a susceptible individual.'	
G. E. Beaumont, *Medicine Essentials for Practitioners and Students*	4th (1942) 'The nature of the irritant is not certain in every case, but alcohol is usually considered to be a factor of some importance.'	5th (1948) 'The nature of the irritant is not certain in every case, but alcohol is usually considered to be a factor of some importance, possibly because the diet is low in protein and in vitamin B.'	
Derrick Dunlop, *Textbook of Medical Treatment*		4th (1946) 'alcohol is still by far the commonest cause of hepatic cirrhosis. Arguments as to how it acts do not concern us here, but recent work of Himsworth adds greater weight to the belief that secondary defects in nutrition are the essential cause of cirrhosis rather than any direct action of alcohol itself.'	
T. R. Harrison, *Principles of Internal Medicine*			1st (1951) 'coexistence of alcoholism and nutritional deficiencies in patients' and 'animal experiments point to lipotropic deficiency as a cause of cirrhosis, but 'it would be unwise to translate this experience directly to humans'
Stanley Davidson, *The Principles and Practice of Medicine*			1st (1952) 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency' due to experiments on animals, while 'alcohol itself does not produce cirrhosis of the liver'

	1956~60	1961~65	1966~70
Alexander Wheeler, Handbook of Medicine		12th (1963) identical claim to 11th edn	
Thomas Dixon Savill, A System of Clinical Medicine		14th (1964) 'Although alcohol plays a part in a number of cases... the condition may develop in those who are strict teetotallers.' 'Other factors are a lack of animal protein, an excess of fat in the diet and vitamin B deficiency.'	
Russell Cecil, A Textbook of Medicine	10th (1959) the nutritional deficiency theory has been 'greatly strengthened by a number of experimental studies which... established the importance of dietary factors' in cirrhosis, with beneficial results from diet therapy	11th (1963) 'Just how alcoholism, malnutrition, or both, produce hepatic injury in unknown', but alcohol itself 'has not been conclusively proved to have a directly injurious action upon the liver cells'	12th (1967) Very little known on factors that determine individual susceptibility to cirrhosis, but importance of 'genetic and dietary factors'
Frederick W. Price, A Textbook of the Practice of Medicine	9th (1956) 'Cirrhosis never develops experimentally in animals as a result of chronic alcohol poisoning... It is probable that the main factor concerned is the low-protein diet which alcoholics often take, combined with their high calorie intake in the form of alcohol.'		10th (1966) Cirrhosis caused by 'dietary deficiencies acting indirectly by interfering with the nutrition of liver cells'
John Conybeare, A Textbook of Medicine	12th (1957) 'aetiology is not understood' but 'combination of factors which have to do with qualitative dietary insufficiencies' brought about by 'insufficient protein in the diet with a consequent lack of lipotrophic factors, among which are methionine and choline'	13th (1961) Alcohol 'is a factor of the greatest importance, but the way in which this causes cirrhosis is uncertain. Experimental work and tropical experience show that disease was a 'nutritional defect' indirectly caused by alcohol by neglecting consumption of foods containing 'protective' substances like choline and methionine. Direct toxicity of alcohol on liver cells has not been demonstrated.	
G. E. Beaumont, Medicine Essentials for Practitioners and Students	7th (1958) the 'question whether alcohol exerts a direct toxic effect on the liver is not finally decided', but cirrhosis can be caused by 'either directly from protein nutritional deficiency, or indirectly because excessive consumption of alcohol results in a lowered protein intake, or causes a gastro-enteritis which interferes with the digestion and absorption of protein.'	8th (1962) identical claim to 7th edn	
Derrick Dunlop, Textbook of Medical Treatment	7th (1958) known causes of cirrhosis include alcohol, deficiency, and poisons, but aetiological effect of alcohol is unclear. Possibility that drinking is an indirect factor to deficiency.		10th (1966) known causes of cirrhosis include alcohol, deficiency, and poisons, but aetiological effect of alcohol is unclear. Possibility that drinking is an indirect factor to deficiency.
T. R. Harrison, Principles of Internal Medicine	3rd (1959) Cirrhosis 'thought to be the consequence of a specific type of malnutrition usually related to chronic alcoholism... It is generally believed that alcohol is 'not a hepatotoxin' and that its effects on the liver are secondary to an associated nutritional disturbance.' Specific nature of deficiency and mechanism of pathogenesis are 'still uncertain'	4th (1962) identical claim to 4th edn	6th (1970) 'epidemiologic studies have implicated' alcoholism as the cause of cirrhosis, but 'there is still no definitive evidence that alcohol by itself leads to cirrhosis'. While a 'contributing factor to the evolution of cirrhosis... malnutrition per se does not lead to Laennec's cirrhosis' but 'a combination of chronic alcohol ingestion plus impaired nutrition leads to liver cell damage and Laennec's cirrhosis'.
Stanley Davidson, The Principles and Practice of Medicine	4th (1959) identical claim to 1st edn	7th (1964) identical claim to 1st edn	9th (1968) 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency' due to experiments on animals, while 'alcohol itself does not produce cirrhosis of the liver'

In contrast to the waning emphasis on alcohol, nutritional deficiency abruptly emerged as the prominent point of reference. In contrast to the interwar period, when no single theory of cirrhosis aetiology was noticeably dominant over others, postwar textbooks almost unanimously (94 percent; 30 out of 32) pointed to dietary deficiency as either the factor or the cause of the disease. Only two of the textbooks from the period refrained from mentioning the role of the diet, although both were published in 1942 when the NDT was still in the process of establishing its foothold. That not a single textbook between 1900 and 1940 referred to nutrition is a remarkable indicator of how the NDT was adopted as the legitimate framework of cirrhosis causation within a matter of a few years. Out of all textbooks from the postwar era, 63 per cent (20 out of 32) specifically attributed deficiency as either a likely (47 percent; 15 out of 32) or a definite (16 percent; 5 out of 32) cause of cirrhosis, while the rest referred to deficiency as just another factor alongside many others. However, when examining the textbooks from within a limited period between 1951 and 1965, the proportion that saw nutritional deficiency as a cause increased to an overwhelming majority of 88 per cent (14 out of 16). Based on this, one can argue that the NDT did not achieve its greatest influence until the 1950s and that the consensus surrounding it did not exist until at least the second half of the 1940s.

The impact of the shift towards the NDT is reflected in how many of the textbooks changed their opinion overtime from editions published before the 1940s. A set of revised editions of Osler's *Principles and Practice of Medicine* by Henry A. Christian aptly illustrates this shift. Whereas both the 1938 and the 1942 editions straightforwardly pointed out that cirrhosis 'occurs most commonly' among drinkers of spirits without explicitly labelling alcohol as the direct cause, the 1947 edition argued instead that 'just how the alcohol causes cirrhosis remains unknown' and that 'there is now a considerable volume of evidence pointing to nutritional deficiency as of greatest importance in the etiology of cirrhosis of liver in man'.[135] A

[135] Christian, and McCrae (eds.), *The Principles and Practice of Medicine*, p. 711; Henry A. Christian, *The Principles and Practice of Medicine, originally written by William Osler* (14th edn, London, 1942), p. 757; Christian, *The Principles and Practice of Medicine* (16th edn), p. 741.

similar shift had occurred between two editions of Dunlop's *Textbook of Medical Treatment*. The 1940 edition argued that 'alcohol is still by far the commonest cause of hepatic cirrhosis'.[136] The later 1946 edition, however, restated the same passage with an added side-note: '…but recent work of Himsworth adds greater weight to the belief that secondary defects in nutrition are the essential cause of cirrhosis rather than any direct action of alcohol itself.'[137]

A set of more gradual changes can be appreciated across multiple editions of Cecil's *Textbook of Medicine*. The 1938 edition maintained that 'very strong clinical opinion still points to alcohol as the chief etiological factor'. The subsequent 1944 edition, however, shifted towards the view that '[c]linical and experimental studies strongly suggest that a direct relationship exists between nutritional deficiency and the development of portal cirrhosis.'[138] Cecil's textbooks continued to reference the existing scientific consensus over the NDT without explicitly stating that deficiency was without a doubt the cause of cirrhosis. The 1947 edition argued that it 'might play an etiologic role' while the 1959 edition pointed out that the NDT has been 'greatly strengthened by a number of experimental studies'.[139] Price's *Textbook of the Practice of Medicine* underwent similar changes in its stance. Whereas the 1947 edition continued to argue that alcohol potentially worked alongside other numerous factors in damaging the liver, as cirrhosis 'very rarely develops experimentally in animals as a result of chronic alcohol poisoning', the subsequent 1956 edition stated that 'the main factor concerned is the low-protein diet which alcoholics often take' because cirrhosis '*never* develops

[136] J. W. McNee, and D. Smith 'Diseases of the Liver, Gallbladder and Biliary Tract, Pancreas and Peritoneum', in Dunlop, Davidson, and McNee (eds.), *Textbook of Medical Treatment* (2nd edn), p. 610.
[137] J. W. McNee, 'Diseases of the Liver, Gallbladder and Biliary Tract, Pancreas and Peritoneum', in D. M. Dunlop, L. S. P. Davidson, and J. W. McNee (eds.), *Textbook of Medical Treatment* (4th edn, Edinburgh, 1946), p. 514.
[138] Herbert K. Detweiler, 'The Cirrhoses of the liver', in Cecil (ed.), *A Text-Book of Medicine* (4th edn), pp. 789-96; Herbert K. Detweiler, 'The Cirrhoses of the Liver', in Russell L. Cecil, and Foster Kennedy (eds.), *A Textbook of Medicine* (6th edn, London, 1944), pp. 761-2.
[139] Donald C. Balfour, 'Diseases of the Digestive System', in Russell L. Cecil, Walsh McDermott, and Harold G. Wolff (eds.), *A Textbook of Medicine* (7th edn, London, 1947), pp. 861-2; Paul A. Di Sant'Agnese, 'Diseases of the Digestive System', in Russell L. Cecil, and Robert F. Loeb (eds.), *A Textbook of Medicine* (10t h edn, Philadelphia, 1959), pp. 880-1.

experimentally in animals as a result of chronic alcohol poisoning'.[140] By 1966, the textbook effectively endorsed the NDT by conceded that cirrhosis was caused by 'dietary deficiencies acting indirectly by interfering with the nutrition of liver cells'.[141] While there had been noticeable changes among textbooks in integrating the recent aetiological shift towards the NDT, many of the authors were hesitant to label malnutrition as the conclusive cause of the disease. Contrasted to how many of the pre-1940 textbooks stated with certainty that cirrhosis was caused by alcohol, the language of the publications discussed in this chapter is characterised by scientific restraint, preferring to state that the evidence accumulated from recent studies seemed to designate deficiency as merely the more probable cause than alcohol.

In other textbooks, the NDT was adopted with greater subtlety. Savill's *System of Clinical Medicine* never attributed deficiency as a cause and labelled it as just another factor alongside alcohol in its 1950 and 1964 editions.[142] Conybeare's *Textbook of Medicine* expressed uncertainty over the precise aetiology of cirrhosis in its 1949, 1957, and 1961 editions, although the latter two highlighted the importance of 'qualitative dietary insufficiencies' and the 'lack of lipotropic factors'. The 1961 edition specifically pointed out that experiments have demonstrated that cirrhosis was primarily a 'nutritional defect' indirectly caused by alcohol.[143] The 1942 edition of Beaumont's *Medicine Essentials for Practitioners and Students* stated that the 'nature of the irritant is not certain in every case, but alcohol is usually considered to be a factor of some importance.'[144] The subsequent 1948 edition addressed the development of the experimental knowledge of cirrhosis and deficiency by augmenting on the previous passage: '…but alcohol is usually considered to be a factor of some importance, *possibly because the diet*

[140] Arthur Hurst, 'Cirrhosis of the Liver', in Frederick W. Price (ed.), *A Textbook of the Practice of Medicine* (7th edn, Oxford, 1947), pp 729-36; Thomas Hunt, 'Diseases of the Digestive System', in Donald Hunter (ed.), *Price's Textbook of the Practice of Medicine* (9th edn, London, 1956), p. 675.
[141] Thomas Hunt, 'Diseases of the Digestive System', in Ronald Bodley Scott (ed.), *Price's Textbook of the Practice of Medicine* (10th edn, London, 1966), p. 560.
[142] E. C. Warner (ed.), *Savill's System of Clinical Medicine* (13th edn, London, 1950), p. 428; E. C. Warner (ed.), *Savill's System of Clinical Medicine* (14th edn, London, 1964), p. 492.
[143] J. J. Conybeare (eds.), *A Textbook of Medicine* (9th edn, Edinburgh, 1949), 345; J. J. Conybeare (eds.), *A Textbook of Medicine* (12th edn, Edinburgh, 1957), p. 356; John Conybeare, and W. N. Mann (eds.), *Textbook of Medicine* (13th edn, Edinburgh, 1961), pp. 406-7.
[144] G. E. Beaumont, *Medicine: Essentials for Practitioners and Students* (4th edn, London, 1942), p. 78.

is low in protein and in vitamin B.'[145] However, by the 1958 edition, the textbook conceded that cirrhosis can be caused by 'either directly from protein nutritional deficiency, or indirectly because excessive consumption of alcohol results in a lowered protein intake, or causes a gastro-enteritis which interferes with the digestion and absorption of protein.' At the same time, the potential role of alcohol as a liver toxin was not entirely disregarded, signalled by the statement that the 'question whether alcohol exerts a direct toxic effect on the liver is not finally decided'.[146]

The new textbooks that emerged after 1945 also addressed the prominence of the NDT. Four editions of Davidson's *Principles and Practice of Medicine* (1952, 1959, 1964, and 1968) consistently argued that 'alcohol itself does not produce cirrhosis' and observed that there is 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency'.[147] On the other hand, multiple editions of Harrison's *Principles of Internal Medicine* presented a nuanced account of the precise impact of nutritional deficiency. Its first edition from 1951 observed that experimental studies had identified the absence of lipotropic factors as the cause of liver damage in animal subjects, but warned that 'it would be unwise to translate this experience directly to human cirrhosis'.[148] The 1958 and 1962 editions, both containing a chapter on liver disease authored by Klatskin, pointed out that cirrhosis was '[t]hought to be the consequence of a specific type of malnutrition usually related to chronic alcoholism and/or faulty dietary habits… However, the nature of the deficiency and the precise mechanism responsible for the development of cirrhosis are still uncertain.'[149] Although both Davidson's and Harrison's

[145] G. E. Beaumont, *Medicine: Essentials for Practitioners and Students* (5th edn, London, 1948), p. 83.
[146] G. E. Beaumont, *Medicine Essentials for Practitioners and Students* (7th edn, London, 1958), pp. 85-6.
[147] Davidson (ed.), *The Principles and Practice of Medicine* (1st edn), pp. 753-4; L. S. P Davidson (ed.), *The Principles and Practice of Medicine: A Textbook for Students and Doctors* (4th edn, Edinburgh, 1959), pp. 842-3; Stanley Davidson (ed.), *The Principles and Practice of Medicine: A Textbook for Students and Doctors* (7th edn, Edinburgh, 1964), p. 992; Stanley Davidson (ed.), *The Principles and Practice of Medicine: A Textbook for Students and Doctors* (9th edn, Edinburgh, 1964), pp. 1021-2.
[148] Daniel Harvey Labby, 'Diseases of the Liver', in Harrison, Beeson, Thorn, Resnik, and Wintrobe (eds.), *Principles of Internal Medicine* (1st edn), p. 1482.
[149] Gerald Klatskin, 'Diseases of the Liver', in T. R. Harrison, Raymond D. Adams, Ivan L. Bennett, Jr., William H. Resnik, George W. Thorn, and M. M. Wintrobe (eds.), *Principles of Internal Medicine* (3rd edn, New York, 1958), p. 1500; Gerald Klatskin, 'Diseases of the Liver', in T. R. Harrison, Raymond D. Adams, Ivan L. Bennett, Jr., William H. Resnik, George W. Thorn, and M. M. Wintrobe (eds.), *Principles of Internal Medicine* (4th edn, New York, 1962), p. 1685.

textbooks acknowledged the role of nutritional deficiency, the latter disputed the direct applicability of recent experimental evidence in explaining the nature of liver damage in humans. Along with the rest of the textbooks of general medicine, the reception of the NDT was coloured by a great degree of modesty over their attribution of dietary deficiency as the cause. At the same time, the emerging orthodoxy of the NDT is clear when one considers how every single one of the textbooks considered this aetiological shift in some form or another.

The wider impact of the specialist research discussed in the first section of this chapter can be appreciated in *The Lancet* and *The British Medical Journal*. The scientific emergence of the NDT received ample coverage in both journals throughout the 1940s. As early as 1943, an editorial in *The British Medical Journal* declared that '[c]hronic alcohol poisoning is now almost off the list of cirrhogenic agents, and room on it is now being found for dietary factors'.[150] An editorial on *The Lancet* referred to György and Goldblatt's article from 1942 to show that fatty liver in rats was caused by a deficiency in casein.[151] A later *Lancet* article also commented that '[e]xperimental work has proved beyond doubt that nutritional deficiency may lead to a diffuse fibrosis of the liver indistinguishable from Laennec's cirrhosis' and that researchers like Patek and Post had succeeded in extending the prognosis of cirrhosis patients through dietary therapy.[152] Patek's contributions were cited again in a 1947 *Lancet* article, which pointed to his successful use of vitamin B for treatment.[153] The same article also referred to the contributions of Charles Best in highlighting the dietary nature of liver damage.[154] A later

[150] Anon., 'Cirrhosis of the Liver', *The British Medical Journal* 1.4283 (6 February 1943), p. 165; The paper cited the following articles for reference: Paul György, and Harry Goldblatt, 'Observations on the conditions of dietary hepatic injury (necrosis, cirrhosis) in rats', *The Journal of Experimental Medicine* 75.4 (1942), pp. 355-68; Patek, and Post, 'Treatment of cirrhosis', pp. 481-505; Lowry, Daft, Sebrell, Ashburn, and Lillie, 'Treatment of dietary liver cirrhosis', pp. 2216-9.

[151] Anon., 'Diet and Hepatitis', *The Lancet* 243.6293 (8 April 1944), pp. 471-2; György, and Goldblatt, 'Observations on the conditions', pp. 355-68.

[152] Anon., 'Dietary Treatment of Hepatic Cirrhosis', *The Lancet* 253.6542 (15 January 1949), pp. 110-1; Arthur J. Patek Jr., Joseph Post, Oscar D. Ratnoff, Harold Mankin, and Robert W. Hillman, 'Dietary treatment of cirrhosis of the liver', *The Journal of the American Medical Association* 138.8 (23 October 1948), pp. 543-9.

[153] Anon., 'Diet and Cirrhosis', *The Lancet* 250.6474 (27 September 1947), pp. 472-4; Arthur J. Patek, 'Dietary treatment of Laennec's cirrhosis with special reference to early stages of the disease', *Bulletin of the New York Academy of Medicine* 19.7 (1943), p. 498.

[154] 'Diet and Cirrhosis', pp. 472-4.

British Medical Journal editorial from 1949 also touted the significance of Best and his team's rat experiments on the comparative effects of alcohol and sugar water on the liver and the effective prevention of its damage by choline. Their research made it 'possible to classify dietary factors into lipotropic and alipotropic, the latter enhancing the deposition of fat in the liver and the former preventing it or facilitating its removal when present'. The editorial annotated the paper's most well recognised soundbite, concluding with the following rhetorical question: 'Is alcohol toxic to the liver? The answer is, No.'[155] The leading medical journals in Britain at the time not only showed that the editors were abundantly aware of the scientific shift towards the NDT but also that many of the studies that were mostly performed in North America were well-received across the Atlantic.

The Lancet and *The British Medical Journal* additionally expanded on the wider discourses surrounding the discovery of the dietary nature of cirrhosis by publishing reports on numerous academic conferences where many of the aforementioned specialists converged and discussed their research. Such articles reveal the existence of a global network of academic researchers who, owing to the enormous growth of commercial air travel after the Second World War, converged in major cities across the world to share their knowledge on the topic.[156] Following the trend towards further specialisation in the discipline, the period saw hepatology grow out of gastroenterology (the study of the digestive system as a whole) as an independent field of medicine that focused on illnesses tied to the liver, gallbladder, pancreas, and the biliary tract. Hepatology matured following the establishment of the American Association for the Study of Liver Disease in 1948 and the subsequent formation of the International Association for the Study of the Liver in 1958. In the United States, the field was primarily established by Hans Popper, a renowned American pathologist of Viennese Jewish origin, while in the Britain, Sheila Sherlock, the first even female professor of medicine in the country, played an integral role in setting up a dedicated liver unit at the Royal Free Hospital School of Medicine in

[155] Anon., 'Alcohol and Cirrhosis of the Liver', *The British Medical Journal* 2.4635 (5 November 1949), pp. 1030-1; Best, Hartroft, Lucas, and Ridout 'Liver Damage', pp. 1001-6.

[156] The Conference of Liver Injury at New York City in January 1948 was attended by Hoffbauer, Best, Goldblatt, György, Patek, Sebrell, Davidson, and others. F. W. Hoffbauer (ed.), *Liver Injury* (1st edn, New York, 1948).

London.[157] The following accounts of the earliest conferences dedicated to liver disease research reveal that several of the medical scientists responsible for establishing the NDT were slowly integrating into the emerging international community of hepatologists.

In 1950, a congress on liver disease hosted in London was attended by György and Hartroft, who presented papers on the impact of nutritional deficiencies on the liver.[158] At a 1955 meeting of the American Association for the Study of Liver Diseases at Chicago, Charles S. Davidson, a noted hepatologist, argued that 'dietary deficiency was the most important factor in the protein malnutrition of alcoholic liver disease.'[159] Later in 1959, a conference on alcoholism and cirrhosis in New York chaired by Popper hosted a roundtable on the aetiology of the disease. Reiterating the results of his renowned study from 1949, Best stated in a talk that the heavy consumption of alcohol indirectly damaged the liver by depriving the drinker of an adequate diet. Although Klatskin largely concurred with Best's assertion that experimental studies had demonstrated that alcohol reduced food intake and increased the choline requirement, he nonetheless questioned the applicability of such results on the human liver by speculating if there might be an 'additional factor in the human disease'. The delegates of the conference came to an agreement that, while causation between alcohol and cirrhosis was not demonstrated as of yet, there was a clear epidemiological association between the two, and that the abstinence from alcohol in addition to a 'normal diet' led to noticeable improvements.[160] There were several other meetings and conferences across the 1950s and the 1960s that brought together many of the dominant researchers in the field.[161]

In summary, the shift towards the NDT directly shaped the dominant medical understandings in the medical profession of postwar Britain. Textbooks of general medicine

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[157] Chen, and Chen, *Understanding the Liver*, pp. 184-5.

[158] Anon., 'An International Congress on Liver Disease', *The Lancet* 258.6696 (29 December 1951), p. 1215.

[159] Anon., 'American Association for the Study of Liver Diseases', *The Lancet* 266.6904 (24 December 1955), pp. 1322-3.

[160] Anon., 'Alcoholism and Hepatic Cirrhosis', *The Lancet* 274.711 (12 December 1959), p. 1077.

[161] Anon., 'Symposium on Liver Diseases', *The Lancet* 265.6869(23 April 1955), pp. 859-61; Anon., 'The Liver', *The Lancet* 270.6985 (13 July 1957), pp. 89-90; Anon., 'Conferences: Liver Disease', *The Lancet* 271.7010 (4 January 1958), p. 45; Anon., 'World Congress of Gastroenterology', *The British Medical Journal* 2.5087 (5 July 1958), pp. 40-2; Anon., 'Conferences: Liver Disease', *The Lancet* 274.7110 (5 December 1959), pp. 1021-2; Anon., 'Conferences: Liver Disease', *The Lancet* 275.7133 (14 May 1960), pp. 1066-8; Anon., 'Gastro-Enterology', *The British Medical Journal* 2.5366 (9 November 1963), p. 1190.

unanimously addressed the significance of the newfound association between liver disease and nutritional deficiency while abandoning the notion that cirrhosis was an illness primarily caused by alcohol. However, many of the sources provided a noticeably restrained description of its causation, whereby a handful textbook authors often went as far as to state that the recent studies pointed to deficiency as nothing more than the most likely cause of cirrhosis and that alcohol merely played an indirect role in its pathogenesis, avoiding the risks of decisively concluding that cirrhosis was without a doubt a disease of nutritional disturbance.

The Drink Question and the Liver in Postwar Britain

The period spanning between the Second World War and the emergence of the public health model in the 1970s has been recognised as a comparatively uneventful time in the politics of alcohol, an exceptional 'twilight zone' within the history of the alcohol problem in modern Britain. Overall *per capita* levels of alcohol consumption dropped to an all-time low during the 1940s and 50s, even lower than that of during the Great Depression.[162] When considered alongside the interwar era, the changes that had occurred between 1918 and the 1970s only receive a single chapter in the accounts provided by Nicholls and Yeomans.[163] Along with the diminished intensity of the problematisation of alcohol, cultural perceptions of drink shifted further towards liberalisation, a development recognised by S. J. D. Green to be a part of the wider process of secularisation in British attitudes to pleasure-seeking activities.[164] However, similar to the rise of interwar moderationism, many of the texts studied in this section show that the relative lack of political confrontations over drink during the period was in many ways as noteworthy as its existence. The decline of public interest in liver disease was signalled by the demise of the DTT and of the conceptualisation of cirrhosis as an 'alcoholic's disease'. While historically low levels of alcohol consumption meant that the incidence of cirrhosis, alongside other chronic diseases tied to drink, was rarely highlighted in most texts as a serious problem,

[162] Nicholls, *The Politics of Alcohol*, p. 196.
[163] *Ibid.*, pp. 180-98; Yeomans, *Alcohol and Moral Regulation*, pp. 129-57.
[164] S. J. D. Green, *The Passing of Protestant England: Secularisation and Social Change, c.1920–1960* (Cambridge, 2011), pp. 142-6.

a handful of temperance and parliamentary sources perceptively referred to the disease and its troubled association with alcohol in a myriad of unique ways. In order to understand the significance of the limited, but nonetheless relevant, reception of the NDT within the toned-down public discussions on alcohol use, this section relies on a diverse array of primary sources connected to the liquor trade, the temperance movement, and parliamentary politics. Although it is difficult to establish historical causation between the diminished scientific emphasis on alcohol as a cause of liver disease and the rise of more permissive attitudes towards drink, the preference for the NDT over the DTT undoubtedly correlated with the continued propensity towards moderation in postwar Britain.

Many of the postwar developments were long-term outcomes of the entrenchment of New Moderationism within political discourses surrounding alcohol use. Unlike the First World War, the alcohol debate during the Second World War receives limited historiographical interest, for which there are no equivalent studies to Duncan's *Pubs and Patriots.*[165] The differences between the two wars can be appreciated by how pub attendance and beer production actually grew between 1939 and 1945, while drunkenness was rarely problematised due to the continued enforcement of strict controls derived from the 1921 licensing act.[166] However, the second half of the twentieth century witnessed the early steps towards the deregulation of this control regime that traced its origins to the CCB during the First World War. The 1949, 1953, and 1964 licensing acts allowed the sale of alcohol in a wider variety of premises, expanded the availability of alcohol, and decentralised the issuing of alcohol licenses. Jennings interpreted the 1961 licensing act, which gave supermarkets the right to retail alcoholic beverages, as a watershed in the path towards further liberalisation.[167] Although postwar governments were unconcerned with tackling alcohol consumption as a whole, the period witnessed targeted debates over specific kinds of drinking, such as alcoholism

[165] Duncan, *Pubs and Patriots.* The likely exception would be Brian Glover, *Brewing for Victory: Brewers, Beer and Pubs in World War II* (Cambridge, 1995), although the popular account is largely disregarded by most academic historians.
[166] Yeomans, *Alcohol and Moral Regulation*, pp. 138-44.
[167] Jennings, *A History of Drink and the English*, pp. 200-1.

as a 'disease', underage consumption, and drunk driving.[168] Such features thus point to the significance of the intrinsic peculiarities of the period within the wider history of the drink question in modern Britain.

During the first half of the twentieth century, the association of alcoholic beverages with medical and dietary symbolism was the dominant trope deployed by the alcoholic beverage industry as an antithesis to the moral condemnation of drink by the temperance movement. Alongside a marked intensification in the application of this association with good health, unpublished material from the Brewers' Society and a handful of beverage advertisement show that the brewing industry increasingly made use of other discursive devices. Although liver disease itself, or any chronic disease for that matter, was never mentioned in such sources, recent scientific discoveries, that alcohol could potentially deprive the drinker of a good diet rather than provide them, failed to deter the industry from continuing to associate beer consumption to nourishment.

As an outcome of the dramatic reduction of *per capita* levels of consumption following the Second World War, the liquor trade was in an increasingly tenuous financial position. As a reaction to the decline of consumer demand for drinks, brewers promoted beer as a symbol of Britishness and the preferred beverage for 'respectable' consumption.[169] This strategy reflected how Britain's national drinking habits were increasingly associated with moderation. An article in *The Lancet* concerning the 'facts and figures on alcoholism' (1952) estimated that, according to the Jellinek formula, England had the lowest proportion of alcoholics among a cohort of 11 countries in Western Europe and Latin America, with Switzerland, Chile, and France leading the top three.[170] To adjust to these austere conditions, brewers further consolidated to larger conglomerates in which eight companies came to dominate 60 per cent of the market share by 1961, compared to ten having 40 per cent of the share in 1940.[171]

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[168] Yeomans, *Alcohol and Moral Regulation*, pp. 150-4.
[169] *Ibid.*, p. 193.
[170] H. Pullar-Strecker, 'Facts and figures on alcoholism', *The Lancet* 259.6707 (15 March 1952), pp. 555-7.
[171] Nicholls, *The Politics of Alcohol*, p. 194.

A 1950 memorandum by the Brewers' Society on 'the problem of declining beer consumption' voiced the sense of panic felt within the industry.[172] The authors expressed dismay at the twenty per cent decline in beer consumption from 1945 to 1949. Although austerity continued in the period that immediately followed the Second World War, the industry expected a recovery in sales from growing incomes, historically low levels of unemployment, and the return of millions of young men from the frontlines. The memorandum attributed this trend to the rise of counter-attractions to alcohol consumption, a development referred to as 'the population deliberately and voluntarily choosing other things instead of beer'.[173] A more optimistic account of the changing social attitudes towards beer was published by Guinness in the form of their 'report on drinking habits' (1950).[174]

The authors also noted with great interest of the shift towards more sociable, moderate drinking practices in Britain. Pubs had gradually become a space where both men and women of all age groups congregated to socialise rather than for the specific purpose of consuming alcohol, a phenomenon described by the report as a 'move away from drink as an end in itself'. It was observed that an '[o]verwhelming [numbers of] people say that the reason they go to pubs etc. is because of the company in them', with the pub becoming increasingly tied to higher social prestige and respectability at the expense of its prior association with public drunkenness. The authors conducted a survey asking a sample of both pub-goers and the general public on the reasons why they drank beer. A hundred per cent of both groups of respondents stated that 'taste' was the primary reason, while only 40 per cent of the public and 34 per cent of pub-goers, a minority in both cases, pointed to 'health'.[175] The report thus noted that, compared to the data acquired by the previous Mass-Observation study in which 52 per cent of respondents had pointed to reasons relating to health to be the main motivation for beer consumption, the proportion of respondents who tied the two together had noticeably declined.[176] The authors

[172] Brewers' Society Collection, MSS.420/BS/7/1/4 'The problem of declining beer consumption', March 1950.
[173] *Ibid.*
[174] Brewers' Society Collection, MSS.420/BS/7/1/2, 'A report on drinking habits', August 1948.
[175] *Ibid.*
[176] Mass-Observation, *The Pub and the People*, p. 86.

nevertheless agreed that advertising slogans such as 'beer is best' had a noticeable impact in promoting the association of beer with physical health, arguing that 'there can be no doubt at all that the phraseology has entered into everyday language and epitomises health attitudes.'[177]

Such investigations only partially explain why brewers were so adamant to preserve the discursive association of beer with dietary health in advertisement. In order to counteract falling sales, the Brewers' Society embarked upon another large-scale advertising initiative similar to the 'beer is best' campaign in the interwar period, this time by more directly tying the beverage to the maintenance of a healthy diet. In 1952, the Society devised a campaign based around a new slogan: 'good wholesome beer'.[178] Its description of beer as being 'wholesome' explicitly conjured up an image that specifically promoted its benefits on one's dietary health, much like 'Guinness is good for you'. The objective of the campaign, simply stated, was to stimulate an 'increase in the consumption of beer mainly through licensed premises'.[179] As part of the initiative, the Society contracted the Jerome Music Company in 1954 to produce a pop song titled after the slogan.[180] The recordings were widely distributed in bulk as background music for many of the brewer-owned pubs across Britain.[181]

Throughout the 1950s, the Society embedded the 'good wholesome beer' message within many of their advertisements, many of which contained an assortment of discursive tropes employed by the brewing industry. Although the slogan itself referred to the standard association of beer with a good diet, the industry was noticeably willing to branch out towards other useful tropes beyond those specifically tied to health. Indeed, unlike the first half of the twentieth century, distillers had almost unanimously abandoned the association of spirits with

[177] Brewers' Society Collection, MSS.420/BS/7/1/2, 'A report on drinking habits', August 1948.
[178] Brewers' Society Collection, MSS.420/BS/6/3/3 Collective advertising campaigns, 1949-1954, 'The Brewers' Society Collective Advertising Campaign, Brief for Advertising Agents', 6 February 1952.
[179] Brewers' Society Collection, MSS.420/BS/6/3/3 Collective advertising campaigns, 1949-1954, 'Brewers' Society Advertising, Confidential', 1955.
[180] The song was performed by the Stargazers, a British vocal group founded by Cliff Adams and Ronnie Milne.
[181] Brewers' Society Collection, MSS.420/BS/6/3/3 Collective advertising campaigns, 1949-1954, 'The Brewers' Society Collective Advertising Campaign, Brief for Advertising Agents', 6 February 1952.

health.[182] Beer adverts found in the *Daily Mail* often contained the slogan within messages such as 'a healthy liking for fresh air and good wholesome beer', 'after a good healthy exercise you can't beat good wholesome beer', and '[w]hen you drink good wholesome beer, that cheerful phrase "Good Health" really means something!'.[183] A 1955 advert promoted the pub as a respectable space for moderate drinking by claiming that the 'friendliest place in the whole wide world is just near your home. The Local. It's the best place of all to meet your friends. To take your wife.'[184] Another advert with the slogan from 1956 appealed to patriotism by depicting three members of the Queen's Guard, an internationally recognised symbol of 'Britishness', uttering the slogan 'good wholesome beer' (see Figure 7) alongside a caption that stated that '[t]here's no other country on earth that can beat us when it comes to brewing beer. Draught or bottled, good British beer is the best long drink in the world!'[185]

[182] The possible exceptions are Teacher's Highland Cream, promoted as a thirst-quenching beverage, in 'Wm. Teacher & Sons Ltd', *Daily Mail*, 21 December 162, p.5. Gordon's Orange and Lemon Gin was also promoted as being 'invigorating' in one advert, in 'Gordon's Orange Gin Lemon Gin', *Daily Mail*, 14 November 1952, p. 4.
[183] 'Good wholesome beer!', *Daily Mail*, 4 June 1955, p 3; 'Beer', *Daily Mail*, 14 June 1958, p. 8; 'Beer', *Daily Mail*, 6 September 1958, p. 5.
[184] 'Good wholesome beer!', *Daily Mail*, 4 June 1955, p 3.
[185] *Ibid.*, p. 10.

FIGURE 7 'Good wholesome beer!', *Daily Mail*, 4 June 1955

Other interesting tropes were found in some of the promotional material that were produced by the Brewers' Society for its own collective campaigns between the 1940s and the 1960s. The standard association of beer with bodily health and energy was employed in a set of visual adverts, including a 1956 comic featuring a competitive rowing team uttering the slogan, and a 1955 sketch with a businessman, an engineer, a farmer, and a mailman going to work above a large cloud that contained the catchphrase, 'revive on it, thrive on it, feel more alive on

it – good wholesome beer'.[186] Several other materials combined the patriotic symbolism with the association to moderate consumption. A newspaper advert from 1960 directed at foreign visitors to Britain featured an image of four drinkers enjoying a beer together in a pub (see Figure 8). It was accompanied by a text that advised on '[h]ow to meet the British at their friendliest', suggesting foreigners to '[p]ut the Local high on your list of attractions when you come to Britain. And don't forget to sample the beer. Many people say it's the best long drink in the world!'[187] Under the title 'People and Pubs', a similar image from 1960 featured a photograph of four individuals enjoying a drink (see Figure 9), accompanied by a claim '[t]hat "after-work pint" is looked forward to all over Britain'.[188] Another photograph depicted a younger man wearing a shirt, a tie, and a suit chatting with a shorter middle-aged man wearing a flat cap, a picture that attempted to communicate the idea that the pub and beer facilitated sociable interactions beyond class and generational boundaries (see Figure 10).[189] The above three pictures all portrayed the pub as a respectable space for socialisation, lubricated by the mild intoxication of beer drinking. Additionally, they featured least one woman in the photograph, indicating that the Society was interested in promoting the space as an amicable environment for both genders. Thus, the brewing industry sought to counteract against falling profits by not only by continuing to promote the health-giving properties of their products, but by seeking to reconstruct the institutional image of the pub as a sociable, accessible environment in line with the perceived respectability and moderation of British drinking habits.

[186] Brewers' Society Collection, MSS.420/BS/6/3/9, 'Collective advertising campaigns', 1968-1983.
[187] *Ibid.*
[188] *Ibid.*
[189] Brewers' Society Collection, MSS.420/BS/6/4/4, 'Mounted photographs of people in pubs', 1939-1950s.

FIGURE 8 Brewers' Society, 'Collective advertising campaigns', 1968-1983

FIGURE 9 Brewers' Society, 'Collective advertising campaigns', 1968-1983

FIGURE 10 Brewers' Society, 'Mounted photographs of people in pubs', 1939-1950s

Unlike the liquor trade, the postwar temperance movement was more receptive to the changing medical understandings on alcohol and liver disease. Their influence on the alcohol debate had been on a long-term decline since the end of the First World War, forcing many of the temperance groups to retain some social significance by transforming into health education bodies.[190] Although the temperance movement always had a function of warning against the dangers of alcohol consumption, they turned to focusing much more on education over their prior interest in pushing for legislative reform.[191] Some of the texts show that the promotion of abstinence was still a priority to the extent of it being framed as a preferred lifestyle choice rather than as a moral compulsion.

In 1948, the Band of Hope published a collection of allegorical stories depicting a set of conversations on alcohol between a father, a son, and a doctor.[192] In it, the author dispelled some of the commonly held beliefs about alcohol that were often promoted by the liquor trade. The son described an advert that he saw as 'trying to tell us that we can work better with the

[190] Thom, *Dealing with Drink*, pp. 20-2.
[191] Yeomans, *Alcohol and Moral Regulation*, p. 228.
[192] Robert Taylor, *Burst Bubbles: The Truth about Alcohol Explained to Boys and Girls* (London, 1948).

aid of a glass of beer', to which the father responded '[d]on't believe everything you read', accompanied by a doctor's advice that '[t]his Beer is Best business is bunkum'.[193] The doctor further cautioned that drinking 'won't stop there' at moderation, adding that 'alcohol is a drug. People who take it grow to want more and more… no one can know until it is too late whether he will be one of the three'.[194] In spite of their continued promotion of abstinence, the dialogue exhibited a tone that was noticeably modest, abject of the fearmongering language on the 'poisonous' and 'evil' qualities of drink apparent in many of the temperance literature discussed in the previous two chapters of this thesis. In spite of the shift in discursive tone, the publication was nonetheless indicative of how the temperance movement was still preoccupied with providing what were essentially a set of counter-narratives against the liquor trade.

A stronger message against the dangers of alcohol was exhibited by an educational pamphlet that transcribed an address delivered by physician Kilsby D. Evans at the annual meeting of the temperance collegiate association in London in 1957.[195] The speaker supported the implementation of compulsory temperance education, arguing that children should be notified that alcohol was a 'dreadful scourge' and a 'poison'.[196] Evans additionally critiqued the normalisation of moderate drinking in postwar British society and how phrases such as 'go for a pint' has become so ingrained in daily life.[197] A separate pamphlet titled *Teetotal Cranks?* (1962), distributed by the UK Alliance, similarly condemned the normalisation of moderate consumption by referring to drunkenness as 'the greatest of Britain's internal problems'.[198] It argued that drink contributed to 'crime; sexual immorality; road and other accidents; ill-health of the body and mind; industrial absenteeism', all of which added up to 'an immense evil, which every Christian ought to resist; and his resistance ought to be based upon personal abstinence'.[199] Although the title of the pamphlet was intended as a rebuttal of the commonly held stereotype of temperance activists as a band of bible-thumping killjoys who derived

[193] *Ibid.*, p. 10.
[194] *Ibid.*, p. 14.
[195] Kilsby D. Evans, *Temperance Education* (Bedford, 1957).
[196] *Ibid.*, p. 15.
[197] *Ibid.*, p. 6.
[198] UK Alliance, *Teetotal Cranks?* (London, 1962), p. 5.
[199] *Ibid.*, p. 5.

pleasure from interfering with the habits of ordinary people, the abnormally harsh and outdated language of its content seemed to have only strengthened this generalisation.

Curiously enough, the scientific demise of alcohol's direct toxicity to the liver and the emergence of the NDT was even acknowledged by a handful of other temperance texts from after the 1950s. A 1958 monograph from the Temperance Collegiate Association hinted at the acceptance of this shift when discussing how 'medical scientists have become more and more of the opinion that many of the bodily ills from which human beings suffer are brought about by wrong feeding.' It referred to how scurvy, rickets, and beriberi, diseases formerly believed to be brought about by alcohol consumption, were found to be caused by the deficiency of 'some important substances'.[200] A lecture delivered by physician Amy M. Fleming at the Alliance House in London, the official headquarters of the UK Alliance, addressed the scientific demise of the DTT by arguing that cirrhosis, alongside other conditions, were 'not determined only by the extent and duration of the excessive drinking but are influenced by other associated factors.'[201] The talk was an occasion with which Fleming was handed the honour of giving the annual Sir R. Murray Hyslop Memorial Lecture, delivered previously by prominent temperance figures such as W. McAdam Eccles and Courtenay Weeks. She then proceeded to mention how alcoholic neuritis, another disease discovered to be directly caused by deficiency and indirectly caused by alcohol, was 'more prone to occur in countries where nutritional deficiencies are common' and 'can be completely prevented regardless of the extent of alcoholism by taking an adequate intake of Vitamin B.'[202] Although Fleming did not explicitly attribute this statement to the aetiology of cirrhosis, the mere fact that the two claims were made adjacent to one another suggests that the speaker acknowledged that medical professionals had already abandoned alcohol as a direct cause of liver disease. Surprisingly for a talk delivered to an audience composed mostly of temperance activists, Fleming also spoke critically of the 'legislative control of the distribution and sale of alcohol' and advocated for the establishment

[200] Walter Shawcross, *Healthy, Wealthy and Wise* (5th edn, Cardiff, 1958), p. 25.
[201] Amy M. Fleming, *Alcohol and Health Development* (Cardiff, 1958), pp. 7-8.
[202] *Ibid.*, p. 8.

of addiction therapy centres for the prevention of alcoholism.[203] This discourse signalled the extent to which the UK Alliance, previously the most powerful prohibitionist group in the nineteenth century, adapted to the changing social and scientific attitudes towards alcohol in postwar Britain.

In 1964, the UK Alliance reprinted an alcohol education syllabus originally produced by the Queensland Temperance League for use in secondary schools across Britain.[204] The curriculum contained what turned out to be the most moderated language towards drink exhibited in any of the temperance texts from the period. In spite of its intended goal of informing schoolchildren of the harmfulness of alcohol, the syllabus began with a statement that '[a]lcohol is one of the most useful chemicals known to mankind. Without it many of our industrial processes would not be possible', an unapologetically positive portrayal of drink that would seldom have been found in temperance texts from several decades before.[205] Although the proceeding sections discussed the myths surrounding the utility of moderate drinking for physical and mental efficiency, it never referred to alcohol as a 'poison' or an 'evil' throughout, possibly so that the syllabus would appear more impartial and less propagandistic to the wider, non-temperate public.[206] Its explicit position on alcohol's role in liver damage was stated as follows: '[t]he effect of alcohol in the stomach is responsible for nutritional disturbances and it is recognized as a contributing factor in causing cirrhosis of the liver.'[207] It later added that '[m]any of the nutritional diseases associated with alcoholism, although perhaps not due directly to the effects of alcohol itself are the result of dietary deficiencies common to the alcoholic.'[208] These passages directly acknowledged the NDT as the most likely explanation of alcohol's effects on the liver, even though, despite the recognition of its indirect nature, alcohol's inherent harmfulness to the human body was highlighted. The syllabus refused to allow the NDT to diminish or downplay the harms of alcohol on the body in concluding with

[203] *Ibid.*, p. 9.
[204] Queensland Temperance League, *A Syllabus of Alcohol Education for Secondary School Pupils* (Brisbane, 1964).
[205] *Ibid.*, p. 5.
[206] *Ibid.*, pp. 5-21.
[207] *Ibid.*, p. 10.
[208] *Ibid.*, p. 22.

bold letters that 'EVERY DRINKER RUNS THE RISK OF ALCOHOLISM'. This was followed by a disclaimer that '[t]he final choice and decision is up to teach one to make for himself and herself (to drink)', a notable departure from the UK Alliance's previous insistence on prohibition.[209] One can judge that this was still a reasonable, scientifically legitimate argument since a hazardous substance still ought to be deemed a hazard even if it causes damage through indirect means.

The NDT continued to be influential among temperance circles late into 1978 when it was referred to in an educational pamphlet distributed by the Teachers' Advisory Council on Alcohol and Drug Education. The list of authors included Derek Rutherford from the National Council of Alcoholism, an organisation originally set up by the Church of England Temperance Society.[210] Concerning the action of alcohol on the liver, the pamphlet admitted that the relationship was a 'very complicated and technical matter; it is difficult to determine which effects are due to alcohol and which to a faulty diet.'[211] Evidently, the example demonstrated how many of the texts mentioned in this section seemingly recognised the shift towards the NDT while simultaneously toning down the language towards drink to a level that would have been almost unheard of several decades earlier. This shift in the rhetoric used by temperance groups after the Second World War was part of their attempt to preserve their institutional relevance in a society that increasingly tolerated most forms of drinking. By acknowledging the NDT, the movement in turn sought to maintain their scientific legitimacy without resorting to the overstatement of the harmful properties of alcohol.

The influence of the shift from an aetiology based on alcohol's direct action on the liver to the NDT was more clearly reflected in the debates surrounding alcohol in high politics. Transcribed parliamentary debates in the *Hansards* show that there were mixed views on the causation of cirrhosis in relation to alcohol within both the House of Commons and the House of Lords. While the interest in alcohol and health was at an all-time low in national politics

[209] *Ibid.*, p. 25.
[210] S. Caruana, James C. P. Cowley, and Derek Rutherford, *Teaching About Alcohol and Drinking* (London, 1978).
[211] *Ibid.*, p. 24

during the first couple of decades after the Second World War, a set of references to liver disease illustrate the wider impact of the scientific debates on cirrhosis aetiology within the discussions surrounding drink in Britain.[212]

The simultaneous influence of the NDT and the rejection of alcohol's direct toxicity to the liver can be appreciated in how cirrhosis was referenced in relation to alcohol and other intoxicants. The clearest evidence of the reception of the NDT in high politics was in its earliest mention in a House of Commons debate that took place on 16 March 1950. Herbert Morrison, the Deputy Prime Minister to Clement Attlee's postwar Labour government, received a question on the newly established National Health Service from Albert Raymond Blackburn, a Labour backbencher. Based on a set of recent studies conducted under the supervision of the Medical Research Council, Blackburn enquired on 'the use of lipotropic agents for treatment of cirrhosis of the liver and undue obesity; and when such agents will be made available for general use.' Morrison responded that '[i]nvestigations into the action of these substances are being made by members of the Council's staff, both in this country and in the tropics. Ordinary diets contain ample amounts of them; but poor diets may contain insufficient amounts and then cirrhosis of the liver may follow.' He assured the backbencher that '[i]n conjunction with a rectified diet, the substances have been used in the treatment of that condition… They are available.'[213] The debate revealed that the dietary treatment of cirrhosis was in the process of being put to use by the NHS, only within a matter of few years after the NDT was established as a scientific consensus.

Other comments made in Parliament concerned issues indirectly related to liver disease, many of which inferred that the DTT no longer bore scrutiny. A House of Lords debate on the Road Traffic Bill against drink driving on 27 April 1961 made this very insinuation. Viscount Hailsham, an influential member of the Conservative Party who served as its chairman, questioned the reliability of venepuncture as a means to understand the driver's level of intoxication, arguing for the possibility that the method might falsely register acetone found in

[212] Yeomans, *Alcohol and Moral Regulation*, pp. 150-4.
[213] HC Deb 16 March 1950, vol 472, col 67-8W.

the bloodstream as part of the individual's blood-alcohol level. He added that '[i]t is also possible that other conditions, such as severe liver disease, may be associated with increased amounts of acetone and aceto-acetic acid in the bloodstream.'[214] The knowledge of acetone as an aetiology or factor to liver disease has not been suggested by any of the professional medical texts thus far. Still, the comment is indicative of how Hailsham sought to distance liver disease, previously understood as a common malaise of the alcoholic, from its association with alcohol itself.

A later discussion on cigarette smoking and lung cancer in the House of Lords on 16 July 1963 made other peculiar references to the liver. The debate over the obligation of the state to spread awareness on the dangers of smoking briefly shifted to the topic of heavy drinking. Baroness Edith Summerskill, a Labour peer and a former cabinet minister in Attlee's government, enquired 'would not the noble Lord agree that, while heavy spirit drinking does not necessarily always cause cirrhosis of the liver, nevertheless heavy spirit drinking does not promote good health?'[215] The passage made the perceptive case that the belief in the health-giving properties of spirit consumption was plainly false. In spite of that, Summerskill felt predisposed to clarify that cirrhosis was *not* an outcome of what she believed to be the practice of consuming a potentially hazardous substance, indicating that the peer was attempting to paint the fairest representation of hard liquor in the event of being accused of exaggerated its harmfulness. Thus, her question shows how she had assumed that that her colleagues might have shared the scepticism towards the notion that cirrhosis was always caused by alcohol, reflecting a wider tendency at the time to downplay alcohol's harmful properties on the body.

Although the above examples from the *Hansards* directly or indirectly addressed the dismissal of the causation of alcohol to liver disease, other passages pointed to the opposite case. A 1962 House of Lords discussion on the rise of lung cancer mortality rates provoked Ian George Eden, the Baron of Auckland, to argue that it does not take 'one of common sense' to abstain from an activity like smoking, knowing that it does 'tremendous, and probably fatal,

[214] HL Deb 27 April 1961, vol 230, col 1050.
[215] HL Deb 16 July 1963, vol 252, col 113.

damage'. He additionally noted that an '[e]xcess of alcohol can cause cirrhosis of the liver, excess of sweet-eating or other foodstuffs can cause tooth decay and stomach upsets.'[216] In a later House of Lords debate on alcoholism in 1965, Tory peer Lord Hawke advocated for stronger controls on alcohol by pointing to the example of France, a nation with a much higher rate of alcoholism and lax restrictions on the sale of drink. He pointed out how '[o]nly in recent years has it been discovered that, instead of being drunk, they [heavy drinkers in France] all have cirrhosis of the liver; and they are trying to do something about that.'[217]

A House of Commons debate on the criminalisation of cannabis on 16 July 1970 also referred to cirrhosis in association with alcohol. Reginald Maudling, the Home Secretary in Edward Heath's Conservative government, questioned the measure on behalf of the protection of personal freedoms, stating that '[i]t is deplorable to see people drinking themselves into cirrhosis or smoking themselves into lung cancer, but nobody proposes that either activity should be prohibited by law.' Reginald Paget, a Labour backbencher from Northampton, concurred by pointing to the hypocrisy of 'those of us who get our lift and our level of inebriation through alcohol' lecturing young drug users not to consume cannabis, stating that '[y]ou may be able to get drunk on pot. You may over-indulge in it. You can equally over-indulge in alcohol, and if you do the results are a good deal worse. "Pot" will not give you cirrhosis of the liver. Pot will not give you the D.T.s (delirium tremens).'[218] Admittedly, the strong association between alcohol and cirrhosis was not entirely abandoned by all, even though some MPs and peers had clearly been receptive to the rejection of alcohol's direct toxicity to the liver.

This section explored the impact of the NDT and the abandonment of alcohol's direct toxicity on the liver within the wider public discourse on alcohol after the Second World War. The period witnessed a noticeably toned-down discussion over the problem of drink in British society. Brewers' adverts and commercial campaigns illustrated a concerted effort within the liquor trade to adjust to a period that experienced the lowest *per capita* levels of alcohol

[216] HL Deb 22 March 1962, vol 238, col 718, 745.
[217] HL Deb 02 December 1965, vol 270, col 1426.
[218] HC Deb 16 July 1970, vol 803, col, 1753, 1801

consumption in modern history. During which, a discourse that combined the dietary and patriotic associations to beer were promoted alongside an attempt to institutionally rebrand the pub as a space for sensible, moderate drinking habits. In the face of their diminishing relevance, the temperance movement also moderated their language over the harms of alcohol. Curiously, however, both temperance and parliamentary texts responded in varying degrees to the scientific establishment of nutritional deficiency as the predominant aetiological explanation of cirrhosis. There were mixed receptions of this shift as some observers recognised the scientific demise of alcohol as the primary cause of liver damage, while others continued to refer to the disease in direct association to drink. Overall, the impact the NDT on the moderated discourses surrounding alcohol use reveal the exceptional qualities of the alcohol debate in postwar Britain.

Conclusion

Following the crescendo of the interwar era, the period during and after the Second World War was the climax in the history of the medical knowledge on alcohol and the liver in the twentieth century. The period witnessed the greatest extent to which alcohol was disassociated from its most archetypal physiological disease, cirrhosis, when the assumed model of causation founded on alcohol's direct toxicity to the liver was replaced by a new explanation that attributed the illness to the deficiency of certain nutrients borne out of a poor diet. Not only was the NDT supported by numerous experimental and clinical studies that succeeded in producing liver damage with the provision of a deficient diet, the theory was popularised after it was endorsed by Jellinek as part of his effort to promote the disease concept of alcoholism. Based on the knowledge available at the time, especially that of the general failure in the experimental reproduction of alcoholic cirrhosis, the NDT was doubtless the most logical explanation that was provided by medical professionals to explain the pathogenesis cirrhosis, even if it was eventually disproven. The framing of cirrhosis as a disease of deficiency predominantly replaced other potential aetiological explanations in texts tied to the alcohol debate, even

among many of the temperance propaganda where one would assume that alcohol's assumed toxicity to its most commonly associated organ would be vigorously defended.

Chapter 4

Alcoholic Cirrhosis in the Late Twentieth Century

For roughly three decades, the nutritional deficiency theory (NDT) remained the generally accepted explanation of the pathogenesis of cirrhosis until contested by a set of new clinical and experimental studies that emerged in the 1960s and 70s. Charles S. Lieber, a gastroenterologist and a nutritionist based in New York, succeeded for the first time in producing liver damage through the use of alcohol on both human and animal subjects, thereby effectively demonstrating alcohol as the primary cause of cirrhosis. Thus, the demise of the NDT in the mid-1970s showed that evidence founded on laboratory experimentation continued to be the cornerstone of inferring causation in liver disease. However, unlike the direct toxicity theory (DTT) from the early twentieth century, the new *modern direct toxicity theory* (MDTT) took into account individual and environmental factors to explain why cirrhosis developed in no more than a minority of heavy drinkers. Although alcohol continued to be seen as an indispensable cause, additional elements such as occupation, race, gender, and genetics were understood to play a part in one's susceptibility to serious alcoholic liver damage.

As a result, the British medical profession promptly acknowledged the alcoholic causation of cirrhosis and the multifactorial nature of its aetiology. The additional re-establishment of an association between alcohol and liver disease also contributed to the emergence of the new public health approach to alcohol consumption and harm in the 1970s. Liver disease, much more so than ever before, was at the epicentre of public debates on alcohol misuse in British society, whereby both public health campaigners and the alcoholic beverage industry heavily exploited the new scientific knowledge on alcohol and cirrhosis as a means to legitimise their respective narratives on the alcohol problem.

The first two sections of this chapter explore a set of experimental, clinical, and epidemiological journal articles that contributed to the shifting scientific consensus from the NDT to the MDTT. The third section uncovers the reception of the new knowledge on cirrhosis aetiology among professional physicians in Britain by looking at both general medical journals and textbooks of general medicine. The last section studies the impact of the changing understandings of alcohol and the liver on the wider public debate on alcohol misuse in Britain throughout the 1970s and 80s. This involves the study of numerous health reports published by professional bodies that were tied to the public health model, and unpublished material from the brewing industry.

The Decline and Fall of the Nutritional Deficiency Theory

The demise of the NDT as the predominant understanding of cirrhosis aetiology was largely precipitated by the eventual demonstration of alcohol's direct toxicity on the liver. During the 1950s, some medical scientists had already begun to question the notion that malnutrition was responsible for serious liver damage. The NDT was eventually undermined by set of a clinical and experimental studies performed by Lieber, who successfully produced the disease through the combined use of alcohol and a nutritionally sufficient diet. These studies demonstrated that alcohol was capable of damaging the liver regardless of the alleged protective effects of an adequate diet. Both Herd and Katcher argue that the decline of the theory was also brought about by a set of epidemiological studies that strengthened the association between alcohol and the liver.[1] This view corresponds to the historiographical interpretation surrounding the gradual acceptance of epidemiology as a legitimate means to infer disease causation following Doll and Hill's lung cancer research in the 1950s and the Framingham Heart Study on the epidemiology of cardiovascular disease in the 1960s.[2] However, a closer look at the content and reception of Lieber's work shows that the discrediting of the NDT and the confirmation of alcohol's direct action on the liver ultimately depended on the demonstration of alcohol's

[1] Herd, 'Ideology, history and changing models', p. 1119; Katcher, 'The Post-Repeal Eclipse', p. 732.
[2] Brandt, 'The Cigarette, Risk and American Culture', pp. 155-76; Burnham, 'American physicians and Tobacco Use', pp. 1-31; Talley, Kushner, and Sterk, 'Lung cancer', pp. 367-8; Aronowitz, *Making Sense of Illness*, pp. 133-4.

capacity to produce cirrhosis in experimental animals, rather than the establishment of a close statistical relationship between the two. Much like the first section of the previous chapter, this section traces the narrative of scientific change through 'journal science'.[3] The significance of clinical and experimental studies in confirming alcohol's direct toxicity on the liver can only be understood by studying the contents of original research found in scientific journal articles. The most relevant journal articles have been identified from the bibliographies of several important textbooks and literature reviews.[4]

In the 1950s, Gerald Klatskin authored a series of literature reviews that highlighted some of the theoretical inconsistencies of the NDT. His first article (1953) expressed awe at the 'body of evidence' that had emerged in support of the theory, so much so that 'for a number of years no dissenting voices were heard, a remarkable state of affairs in the history of the alcoholic cirrhosis problem'.[5] Indeed, Klatskin himself contributed to the entrenchment of the NDT when he performed a rat experiment that replicated Charles Best's sugar water study.[6] Nonetheless, he was disposed to warn that 'this has led to the erroneous impression in some quarters that the problem has been solved, and that further investigation is no longer needed'.[7] Klatskin highlighted four points of objection towards the NDT that suggested a disassociation between cirrhosis and what was then believed to be it primary aetiology, malnutrition. The first two was attributed to his own clinical observations: not all alcoholics suffering from cirrhosis

[3] Fleck, Genesis and Development of Scientific Fact, p. 118.

[4] In chronological order of publication, the specialist textbooks include: Sheila Sherlock, *Diseases of the Liver and Biliary System* (3rd edn, Oxford, 1963); Schaffner, Sherlock, and Leevy, *The Liver and Its Diseases*; Sheila Sherlock, *Diseases of the Liver and Biliary System*; Wright, Alberti, Karran, and Millward-Sadler, *Liver and Biliary Disease*; Schiff, and Schiff, *Diseases of the Liver*. Literature reviews include: Steiner, 'Evolution of Research in Etiological Types', pp 890-924; Galambos, 'Progress report', pp. 477-90; Sinclair, 'Nutritional Aspects', pp. 117-23; Feinman, and Lieber, 'Liver disease in alcoholism', pp. 303-38; Charles S. Lieber, 'Liver Disease and Alcohol: Fatty Liver, Alcoholic Hepatitis, Cirrhosis, and their Interrelationships', *Annals New York Academy of Sciences* 252 (1975), pp. 63-84; Lelbach, 'Cirrhosis in the alcoholic', pp. 85-105; Nancy K. Mello, 'Animal Models for the Study of Alcohol Addiction', *Psychoneuroendocrinology* 1 (1976), pp. 347-57; Allan D. Thomson, 'Alcohol and Nutrition', *Clinics in Endocrinology and Metabolism* 7.2 (1978), pp. 405-28; Arthur J. Patek, Jr., 'Alcohol, Malnutrition, and Alcoholic Cirrhosis', *The American Journal of Clinical Nutrition* 32 (1979), pp. 1304-12; Esteban Mezey, 'Alcoholic Liver Disease: roles of alcohol and malnutrition', *The American Journal of Clinical Nutrition* 33 (1980), pp. 2709-18; Sheila Sherlock, 'Current Problems in Alcoholic Liver Disease', *Alcohol & Alcoholism* 18.2 (1983), pp. 99-118.

[5] Klatskin, 'The Role of Alcohol', pp. 23

[6] Klatskin, Krehl, and Conn, 'The effect of alcohol on the choline requirement', p. 605.

[7] Klatskin, 'The Role of Alcohol', p., 28.

suffered from malnutrition or a nutritional deficiency, and cirrhosis was seldom common among malnourished non-alcoholics who may or may not have had other chronic diseases. Similar to the second objection, the third stated that cirrhosis was either rare or non-existent among those who were starved or unfed for long periods.[8] As examples, Klatskin referenced two separate studies that discussed the conditions of American prisoners of war in Japanese camps and German prisoners in British camps who survived the Second World War, both of whom underwent a state of severe long-term malnourishment *without* developing cirrhosis.[9] The final point stated that cirrhotic livers displayed clinical, functional, and visible improvement even when kept on a 'suboptimal' diet devoid of nutrients that were believed to be necessary in maintaining a healthy liver.[10] Based on these objections, Klatskin argued that 'the effects of alcohol in the liver should not be dismissed as being purely secondary to a reduction in food intake even though it is recognized that protein deficiency itself can produce cirrhosis.'[11] Although he acknowledged the overwhelming clinical and experimental evidence that pointed to diet having a major role in the pathogenesis, Klatskin suggested that there should be further studies into producing a more precise account of the mechanism grounding the interaction between alcohol, diet, and liver disease.

Klatskin's scepticism towards the NDT and its rejection of alcohol's causation strengthened further in the subsequent years. In his 1959 review article, Klatskin labelled alcoholism as an 'important but by no means the only, etiological factor', arguing that it had the capacity to 'potentiate the hepatotoxic effects of certain agents in the pathogenesis'.[12] The

[8] *Ibid.*, p. 28.

[9] Gerald Klatskin, W. T. Salter, and F. D. Humm, 'Gynecomastia due to malnutrition. 1. Clinical Studies', *American Journal of Medical Sciences* 213 (1947), pp. 19-30; Sheila Sherlock, and Veryan Walshe 'Effect of under-nutrition in man on hepatic structure and function', *Nature* 161 (1948), p. 604.

[10] Klatskin, 'The Role of Alcohol', p. 29; Gerald Klatskin, and R. Yesner, 'Factors in the treatment of Laennec's cirrhosis. 1. Clinical and histological changes observed during a control period of bed-rest, alcohol withdrawal, and a minimal basic diet', *Journal of Clinical Investigation* 28 (1949), p. 723; R. D. Eckhardt, N. Zamchek, R. L. Sidman, G. J. Gabuzda, Jr., C. S. Davidson, 'Effect of protein starvation and of protein feeding on the clinical course, liver function, and liver histochemistry of three patients with active fatty alcoholic cirrhosis', *Journal of Clinical Investigation* 29 (1950), p. 227

[11] Klatskin, 'The Role of Alcohol', p. 35.

[12] Klatskin, 'Effect of Alcohol on the Liver', p. 1671-5.

importance of alcohol was stressed with greater confidence in his final review from 1961 in which he observed that 'there is an impressive body of evidence implicating alcohol as an important etiologic factor in cirrhosis', even though 'the precise way in which alcohol affects the liver is unknown'.[13] While conceding that alcohol's known role was still restricted to its 'nutritional and metabolic effects' on the liver, Klatskin insisted that 'they do not necessarily imply that the effects of alcohol on the liver are solely due to limitation of the dietary intake', echoing the set of objections raised in his 1953 review.[14] Klatskin's scepticism signalled many of the later scientific developments in the laying the groundwork for other observers to take his critique of the NDT even further.

Around the same period, controversy erupted over whether fatty liver, the earliest stage of liver disease, itself played an aetiological role in the pathogenesis of cirrhosis in spite of the continued consumption of alcohol. Before the Second World War, two experimental studies by Charles Connor and I. L. Chaikoff in 1938 successfully reproduced cirrhosis in both normal and depancreatised dogs through the provision of a diet maintaining high levels of fatty liver, leading them to suppose that fatty liver itself was a necessary stepping-stone for the development of further liver damage.[15] W. Stanley Hartroft, a St. Louis-based pathologist who had previously worked alongside Charles Best on the 1949 sugar-water experiment, published a paper in 1954 that took this supposition further in arguing that fatty liver brought about by the deficiency of choline should be deemed among the aetiologies of cirrhosis.[16] Hartroft argued that, '[a]s the liver becomes increasingly fatty, fibrosis makes its appearance. Our evidence indicates that formation and rupture of fatty cysts is… the direct cause of fibrosis'.[17] Claiming that the phenomenon was directly applicable to human biology, he sought to

[13] Klatskin, 'Experimental Studies on the Role of Alcohol', p. 439.
[14] *Ibid.*, p. 441.
[15] Chaikoff, Connor, and Biskind, 'Fatty infiltration and cirrhosis', pp. 101-10; Connor, and Chaikoff, 'Production of cirrhosis in fatty livers', pp. 356-9.
[16] W. Stanley Hartroft, 'The Sequence of Pathologic Events in the Development of Experimental Fatty Liver and Cirrhosis', *Annals of the New York Academy of Science*s 54.7 (1954), pp. 633-45.
[17] *Ibid.*, p. 640.

strengthen the NDT in inferring that cirrhosis could be brought purely about by malnutrition if deficiency-caused fatty liver itself could lead to cirrhosis without the aid of alcohol.[18]

Hartroft's provocative argument later received a direct response from Carroll M. Leevy in 1962.[19] Leevy performed a large clinical study that involved a complete physical examination, survey of the nutritional history, and liver biopsy of 270 patients who had been suffering from 'significant fatty liver'. Within this sample, the paper observed that fatty liver progressed to cirrhosis only if the patient continued drinking, while patients who stopped drinking did not experience further liver damage. Therefore, Leevy labelled alcohol as the indispensable factor in the development of cirrhosis from earlier stages of liver disease, disregarding the possibility that fatty liver itself played any aetiological role.[20] His use of human patients was additionally understood as an effective indictment against Hartroft's assertion that his observation on animal subjects directly applied to humans. These conclusions were subsequently endorsed by a 1969 review article by Galambos, who stated that there seemed to be no legitimate evidence to date that fatty liver itself was a causative agent for cirrhosis in humans.[21] While the study did not disprove the causation of nutritional deficiency per se, Leevy's study laid the groundwork for later studies that attributed a full range of liver diseases to the action of alcohol.

By the 1960s, a notable shift occurred in the language concerning the relationship between liver disease, alcohol, and diet, further distancing itself from the nutrition-based discourse that had dominated the previous two decades. This trend was observable in P. E. Steiner's ambitious review article on the 'evolution of research in the etiological types of cirrhotic diseases of the liver, 1931-1961' (1964).[22] Steiner presented his review of 231 articles on the various types of cirrhosis as a 'sequel' to Virgil Moon's similarly ambitious article from 1934.[23] Since the publication of Moon's review, Steiner noted that substantial progress that had

[18] *Ibid.*, p. 633.
[19] Carroll M. Leevy, 'Fatty Liver: a study of 270 patients with biopsy proven fatty liver and a review of the literature', *Medicine* 41.3 (1962), pp. 249-78.
[20] *Ibid.*, pp. 250, 272-3.
[21] Galambos, 'Progress Report', pp. 481.
[22] Steiner, 'Evolution of Research', pp. 890-924.
[23] *Ibid.*, p. 891; Moon, 'Experimental cirrhosis', pp. 381-424.

been made on the knowledge on cirrhosis: a clearer understanding of the epidemiological prevalence of the disease, a more accurate knowledge of the mechanism of its pathogenesis, and diagnostic innovations through the use of a needle biopsy. In spite of that, the 'elusivity of its specific etiology' continued to baffle Steiner.[24] Although '[m]uch additional evidence that the heavy usage of alcoholic beverages may sometimes result in cirrhosis has accumulated… the exact mechanism remains unknown.'[25] Steiner observed that alcohol's aetiology was based purely on statistical and clinical associations at the time, while '[t]he failure to produce cirrhosis in experimental animals with ethyl alcohol alone… has caused some critics to deny the relationship.'[26] However, based on how robust the association was between heavy drinking and cirrhosis, how the risk of cirrhosis seemed to increase with the quantity of alcohol, and how cirrhotic livers tended to experience histological improvements when alcohol was withheld, Steiner concluded that '[t]he alcoholic beverage is the *sine qua non* in the equation; without these cirrhosis would not occur.'[27] Although Steiner refrained from identifying alcohol as a definite direct cause of cirrhosis, he nonetheless supported the view that alcohol was 'the *sine qua non*', or the indispensable or essential, cause in the pathogenesis of cirrhosis. This view could be juxtaposed to how the review stopped short of applying the same principle to nutritional deficiency, which he regarded as a cause demonstrated only in animal subjects and not in humans.[28]

The 1960s additionally saw the publication of a series of new studies that signalled the further demise of the NDT. At the heart of these investigations was a nutritionist and gastroenterologist named Charles S. Lieber. Lieber was born on 13 February 1931 in Antwerp to Jewish parents. After fleeing the German invasion and spending most of the duration of the Second World War as a refugee in Switzerland, he returned to Belgium to complete his medical education at the University of Brussels. Lieber later obtained a grant to work in the United

[24] Steiner, 'Evolution of Research', p. 891.
[25] *Ibid.*, p. 892.
[26] *Ibid.*, p. 893.
[27] *Ibid.*, p. 893-4.
[28] *Ibid.*, p. 909.

States alongside Charles S. Davidson in the Boston City Hospital. By the 1960s, he landed several prominent positions in New York City as a professor at the Mount Sinai School of Medicine and a research director on liver disease and nutrition at both the Bellevue Hospital and the Bronx Veterans Affairs Medical Centre.[29] Alongside Leonore M. DeCarli, Emanuel Rubin, Lawrence Feinman, and others, a large portion of Lieber's career at New York was dedicated to investigating the toxicity of alcohol to the liver.

In an interview in 2001, Lieber recalled the time when he, as a medical student, felt 'puzzled' by how the 'dogma', that 'liver disease caused by alcohol was due to malnutrition and not to any toxicity of alcohol'. This contradicted with how 'many of my cirrhotic patients in Belgium had a rather good diet and did not appear to suffer from malnutrition'.[30] In an essay from 2002, Lieber attributed this 'dogma' to 'the experiments of Best and Hartroft, who had observed that in rats given alcohol as part of their liquid diet, no liver damage resulted when the diet was adequate'.[31] He was referencing Best's influential rat experiment from 1949, which maintained that alcohol was no more harmful to the liver than isocaloric amounts of sugar water.[32] This paper inspired Lieber to make his first major contribution to the field by formulating the Lieber-DeCarli liquid diet. The new dietary regimen was intended to supplant the methodological flaws of Best's experiment, unable to introduce volumes of alcohol that were large enough to cause serious liver damage owing to its failure to overcome the animal's natural aversion towards alcohol. Lieber suggested that this aversion could be overcome by combining hazardous amounts of alcohol with an entirely liquid diet containing adequate amounts of nutrition. The Lieber-DeCarli liquid diet supplanted 36 per cent of its caloric

[29] For more on Lieber's life and career, see Anon., 'Conversation with Charles S. Lieber', *Addiction* 96 (2001), pp. 955-72; Steven Schenker, 'Special tribute: Charles S. Lieber, M. D. (1931-2009)', *Hepatology* 49.6 (2009), pp. 1785-6.

[30] 'Conversation with Charles S. Lieber', p. 958.

[31] Charles S. Lieber, 'Alcohol and the Liver: Metabolism of Alcohol and Its Role in Hepatic and Extrahepatic Diseases', in Jeremy Hugh Baron, and Henry D. Janowitz (eds.), *Gastroenterology and Hepatology at the Mount Sinai Hospital, 1852-2000* (New York, 2002), p. 83.

[32] Best, Hartroft, Lucas, and Ridout, 'Liver damage produced by feeding alcohol', pp. 1001-6.

content with pure alcohol, twice the volume of what was used by Best.[33] Thus, the animal subjects would have no choice but to consume alcohol to stave off its hunger.

The Lieber-DeCarli liquid diet was immediately put to use in a 1963 study on the hepatic impact of alcohol combined with a nutritionally adequate diet. As expected, the researchers successfully developed fatty liver in experimental groups that were fed alcohol unlike the control groups that consumed an isocaloric dosage of carbohydrates.[34] The subsequent paper published in 1965 replicated this study by introducing the Lieber-DeCarli liquid diet on both 5 human patients and 32 groups of six rats.[35] Both groups were fed an intake of essential vitamins and minerals that exceeded the recommended daily dosage. Special attention was also paid to ensure the consumption of at least 90 per cent of the provisioned regimen for both humans and rats to maintain a healthy body weight.[36] Initially, the livers of the human subjects showed no signs of fatty liver following the first 'control period' when carbohydrates were consumed in the place of alcohol. However, fatty livers of varying degrees developed during the 'experimental period' when the carbohydrates were replaced by alcohol.[37] The results were largely identical for rats, among which the alcohol-fed experimental group were more disposed to developing fatty liver over the control group.[38] Lieber, therefore, concluded that the study 'incriminates ethanol itself as a direct etiologic factor in the pathogenesis of the alcoholic fatty liver, independent of nutritional deficiencies.'[39] Outside of its conclusion, the significance of this early study is attributable to the unique methodology involving the simultaneous production of fatty liver in both a clinical and a laboratory setting under more or less identical conditions.

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[33] C. S. Lieber, 'Pathogenesis and treatment of alcoholic liver disease: progress over the last 50 years', *Annales Academie Meicae Bialostocensis* 50 (2005), p. 8.

[34] Charles S. Lieber, Don P. Jones, J. Mandelson, and Leonore M. DeCarli, 'Fatty liver, hyperlipemia and hyperuricemia produced by prolonged alcohol consumption, despite adequate dietary intake', *Translations of the Association of American Physicians* 76 (1963), pp. 289-300.

[35] Charles S. Lieber, Don P. Jones, and Leonore M. DeCarli, 'Effects of Prolonged Ethanol Intake: Production of Fatty Liver despite adequate diets', *Journal of Clinical Investigation* 44.6 (1965), pp. 1009-21.

[36] *Ibid.*, pp. 1009-11.

[37] *Ibid.*, p. 1011.

[38] *Ibid.*, p. 1020.

[39] *Ibid.*, p. 1020.

Thereafter, Lieber continued his investigations into alcohol and liver damage. A pair of clinical studies from 1968, performed in collaboration with Rubin, demonstrated that fatty liver could be produced in human subjects maintained on an adequate diet, even among non-alcoholics. The first study was carried out on five alcoholic volunteers who were given alcohol and a diet high in protein and low in fat for 18 consecutive days. Every single subject developed fatty liver, exhibiting 'that alcohol, even when given with an amount of dietary protein well in excess of recommended levels, can nevertheless produce a fatty liver in man'. The authors, however, noted that, 'for a given alcohol intake, the severity of the hepatic lesions may be aggravated by dietary deficiencies' as it has been demonstrated on rats before.[40] Moreover, they hinted at the likelihood that alcoholic volunteers were potentially 'more susceptible to an alcoholic insult than an average nonalcoholic', leading to a subsequent study that replicated the same results on volunteers who had no previous history of alcoholism.[41] Lieber and Rubin recruited eight male and four female medical students at Mount Sinai between the ages of 19 to 32, all of whom were 'well nourished' with no prior self-reported episodes of alcohol abuse.[42] The experimental group of seven volunteers consumed alcohol with a nutritionally ample diet, while five others in the control group were fed the same diet with isocaloric amounts of carbohydrates instead of alcohol.[43] As a result, most of the experimental group underwent 'occasional periods of euphoria', while 'other clinical signs of alcohol intoxication, such as slurred speech and ataxic gait (abnormal, uncoordinated physical movements resulting from intoxication), were absent', indicating that alcohol was not consumed to excess.[44] As for their livers, the 'data demonstrate that in normal, nonalcoholic people alcohol produces a fatty liver and ultrastructural changes that are independent of nutritional factors.' Remarkably, the morphological infiltration of fat in the organs of the experimental subjects who had no previous

[40] Charles S. Lieber, and Emanuel Rubin, 'Alcoholic fatty liver in man on a high protein and low fat diet', *The American Journal of Medicine* 44.2 (1968), pp. 200-5.
[41] *Ibid.*, p. 205; Emanuel Rubin, and Charles S. Lieber, 'Alcohol-induced hepatic injury in nonalcoholic volunteers', *New England Journal of Medicine* 278.16 (1968), pp. 869-876.
[42] Rubin, and Lieber, 'Alcohol-induced hepatic injury', p. 896.
[43] *Ibid.*, p. 896.
[44] *Ibid.*, p. 871.

history of alcoholism was 'no less than that in alcoholic persons' from their preceding study.[45] The paper thus concluded that even moderate consumption was capable of causing fatty liver, stating that 'alcohol can rapidly produce liver injury when taken in amounts equivalent to those consumed not only by recognized alcoholic persons but by many "social" drinkers as well'.[46]

Lieber's demonstration of alcohol's direct culpability in fatty liver received a robust reply from Hartroft, who previously worked with Best as the second author of the 1949 sugar water experiment. Hartroft, attempting to defend the NDT, published a couple of articles that made a set of claims on alcohol and liver disease that directly challenged Lieber's attempt to reconnect the two as a relationship founded on direct causation. Hartroft's 1967 paper insisted on the validity and applicability of the results of his animal experiments on humans, arguing that '[t]he cirrhotic livers of choline-deficient rats… share many features with those of chronic alcoholics and offer an acceptable model in which to test the effect of alcohol'.[47] In a study on 50 rats that developed fatty liver from a deficient diet, Hartroft investigated whether the use of the Lieber-DeCarli liquid diet across 3 months led to any improvements in the structure and function of the organ.[48] The study showed noticeable hepatic improvements among most subjects and, most importantly, demonstrated that the inclusion of alcohol in the 'super diet' failed to prevent the recovery. Based on this, Hartroft argued that the 'concept championed by Lieber et al. advocating alcohol itself as a direct hepatotoxic agent in the production of fatty liver independent of associated nutritional disturbance appears… difficult to reconcile with the results of this and other recently published experiments from our laboratories'. According to him, this was because the inclusion of alcohol, if it were a hepatotoxin, should have inhibited hepatic recovery.[49] Hartroft then reached the astounding conclusion that 'it affords hope for achieving some recovery of liver function *in even those alcoholics who cannot give up spirits* if

[45] *Ibid.*, p. 872.
[46] *Ibid.*, pp. 874-5.
[47] Akira Takada, Eduardo A. Porta, and W. Stanley Hartroft, 'Regression of Dietary Cirrhosis in Rats Fed Alcohol and a "Super Diet" Evidence of the Nonhepatotoxic Nature of Ethanol', *The American Journal of Clinical Nutrition* 20.3 (1967), p. 213.
[48] *Ibid.*, p. 219.
[49] *Ibid.*, p. 223.

only they can be induced to consume simultaneously high protein diets containing abundant vitamins and essential food factors.'[50] The same study was replicated later in 1972 when the caloric proportion of alcohol was raised from 36 to 50 per cent, which also led to a conclusion that liver damage depended on the quality of the diet and not the presence of alcohol.[51] As the most vocal champion of the NDT, Hartroft was confident that an adequate diet could potentially lead to a better prognosis for alcoholic patients of liver disease, even if they continued to drink

The debate between advocates of the NDT and its opponents culminated in 1973 in an exchange of letters published in *Science* in which Hartroft and his colleague Eduardo A. Porta went head-to-head with Feinman and Lieber over the applicability of using rat experiments to understand liver damage in humans.[52] Hartroft and Porta vigorously stated their case by referring to a long list of studies published since the early 1940s that had, in their view, confirmed the detrimental effects that dietary changes had to the liver and the curative outcomes of dietary therapy. Concerning Lieber's recent successes in the reproduction of fatty liver through the combined use of alcohol and an adequate nutrition, Hartroft suspected that 'the animal diets used… were not adequate to protect the animal livers from the caloric burden imposed by alcohol'.[53] This supposition, however, was curiously not backed-up by an explanation of precisely *what* the diets where inadequate of. In response, Feinman and Lieber commented on the methodological flaws of Hartroft's own studies. They argued that humans and rats differed in their 'hepatic choline oxidase activity', pointing to how human livers were not as negatively affected by the deficiency of choline as in rats.[54] Furthermore, they brought attention to a prior study by Lieber and DeCarli from 1966 that showed that prolonged ingestion of alcohol eventually resulted in the production of fatty liver in rats, in spite of the

[50] *Ibid.*, p. 223.
[51] Eduardo A. Porta, Osvaldo R. Koch, and W. Stanley Hartroft, 'Recovery from chronic hepatic lesions in rats fed alcohol and a solid super diet', *The American Journal of Clinical Nutrition* 25.9 (1972), pp. 881-896.
[52] W. S. Hartroft, Eduardo A. Porta, Lawrence Feinman, and Charles S. Lieber, 'Fibrogenic Effect of Alcohol in Rat Liver: Role of Diet', *Science* 179.4071 (1973), pp. 406-7.
[53] *Ibid.*, p. 406-7.
[54] *Ibid.*, p. 407.

application of a diet that contained ample amounts of choline.[55] Therefore, Lieber and Feinman concluded that 'there are no diets known, no matter how superb by traditional nutritional criteria, that are "adequate" enough to fully protect the liver against the distinct effects of alcohol we have enumerated.'[56] The dispute between the two camps concerned whether rats were an appropriate model organism to understand the susceptibility of human livers to certain kinds of nutritional deficiencies, as well as over the limits of the protective effects of choline on the liver against the toxicity of alcohol. Hartroft's critique was ultimately undermined by his inability to specifically point out exactly what the nutrient was that was supposedly missing in Lieber's dietary regimen.

The controversy over the applicability of hepatic changes found in rodents to human pathology precipitated a shift in Lieber's experimental approach. To account for the deficiencies of rat experiments highlighted by his exchange with Hartroft, Lieber turned his attention to non-human primates, an expensive but a genetically much closer order of mammals to humans. This methodological approach was partly inspired by two prior studies. The first, performed by Frederick W. Hoffbauer and F. George Zaki, underlined pronounced differences between baboons and rats in their hepatic susceptibility to certain kinds of malnutrition.[57] The paper noted that baboons, unlike rats, did not develop anything worse than fatty liver, even after two years of being fed a choline-deficient diet.[58] The second study by Boris H. Reubner similarly discussed observable ultrastructural differences in the livers of humans, rats, and rhesus monkeys in their response to choline deficiency.[59] The author observed how the deficiency-caused fatty liver that had developed in rhesus monkeys 'resembled human nutritional (alcoholic) liver disease in many respects'.[60] Lieber, DeCarli, Rubin, and two other

[55] Charles S. Lieber, and Leonore M. DeCarli, 'Study of agents for the prevention of the fatty liver produced by prolonged alcohol intake', *Gastroenterology* 50.3 (1966), pp. 316-22.
[56] Hartroft, Porta, Feinman, and Lieber, 'Fibrogenic Effect of Alcohol', pp. 406-7.
[57] Frederick W. Hoffbauer, and F. George Zaki, 'Choline Deficiency in Baboon and Rat Compared', *Archives of Pathology* 79 (1965), pp. 264-9.
[58] *Ibid.*, p. 368.
[59] Boris H. Ruebner, Joseph Moore, Robert B. Rutherford, Arnold M. Seligman, and George D. Zuidema, 'Nutritional cirrhosis in rhesus monkeys: Electron microscopy and histochemistry', *Experimental and Molecular Pathology* 11.1 (1969), pp. 53-70.
[60] *Ibid.*, p. 65.

researchers thus conducted their first experiment on baboons in 1972 to understand the varying effects of long-term alcohol use and the restriction of protein.[61] The study revealed that baboons that were fed alcohol developed 'fat accumulation in the liver with striking ultrastructural changes even in the presence of an adequate diet', while on the other hand, the restriction of protein had 'no striking effects'.[62] The results were consistent with Rubin and Lieber's prior clinical study on human subjects. Naturally, Lieber and his colleagues were then pushed towards attempting to produce cirrhosis, the highest level of liver damage. Therefore, because it was clearly unethical to develop later stages of liver disease in human patients, the baboon was a promising experimental model for Lieber's proposed experiments, possessing a morphologically identical liver, a relatively long life expectancy, and a higher tolerance to alcohol than rodents. The following articles published in the mid-1970s transpired to be the nail in the coffin of the NDT.

The first paper published in 1974 by Rubin and Lieber was titled 'fatty liver, alcoholic hepatitis and cirrhosis produced by alcohol in primates', a result of an expensive long-term study performed at a Veterans Administration laboratory just outside of New York City.[63] As the title suggested, Lieber and Rubin achieved the first successful experimental production of the entire spectrum of alcoholic liver disease, including fatty liver, hepatitis, and cirrhosis, through the provision of alcohol alongside an adequate diet. The diet contained ample amounts of vitamin B12, thiamine, and choline chloride among other nutrients.[64] Across a duration of nine months to four years, all 13 of the baboons in the experimental group developed fatty liver at a certain point, four developed hepatitis, and two developed cirrhosis. Lieber and Rubin remarked that the 'ultrastructural changes in the liver cells were also remarkably similar to those seen in human alcoholic hepatitis in volunteers fed alcohol' and that 'the failure to produce alcoholic hepatitis and cirrhosis when alcohol is fed to rats not only may represent a

[61] Charles S. Lieber, L. M. DeCarli, H. Gang, G. Walker, and E. Rubin, 'Hepatic effects of long term ethanol consumption in primates', *Medical Primatology, Part 3* (1972), pp. 270-278.
[62] *Ibid.*, p. 275.
[63] Emanuel Rubin, and Charles S. Lieber, 'Fatty liver, alcoholic hepatitis and cirrhosis produced by alcohol in primates', *New England Journal of Medicine* 290.3 (1974), pp. 128-135.
[64] *Ibid.*, pp. 129-30.

species difference but also may reflect the short life of the rat and the inability to administer as much alcohol as is consumed by chronic alcoholics'.[65] Thus, the benefits of using primates as the model organism were abundantly clear, as cirrhosis in humans develops as a result of the habitual consumption of harmful volumes of alcohol across many years. Unlike baboons, rats were prone to die long before further liver damage was reproducible.[66] Based on the results of the study, the authors not only stipulated that the patients diagnosed with severe liver disease should immediately abstain from alcohol but that 'it would be appropriate for the physician to warn the alcoholic patient that a nutritious diet will not prevent the development of alcoholic hepatitis and cirrhosis'.[67]

Lieber and Rubin were joined by DeCarli in a later study from 1975 that continued their research on alcoholic liver disease in primates.[68] This time, they raised the dosage of alcohol to 50 per cent of the total caloric content of the nutritionally sufficient diet, the results of which were signs of physical dependence and severe withdrawal symptoms among the 15 baboons in the experimental group.[69] A detailed histological study of the livers of each baboon showed that every single one of the experimental subjects developed some level of fatty liver markedly worse than those of the control groups receiving an isocaloric non-alcoholic regimen, while five went as far as to develop cirrhosis.[70] The paper thus concluded 'that despite the evidence produced before indicating that malnutrition can cause liver damage, *alcohol itself is an indispensable etiologic agent for the development of the typical complications observed in the alcoholic.*'[71] Lieber's ability to reproduce a range of liver diseases, especially cirrhosis, through the combined use of alcohol and a plentiful diet that contained every nutrient believed to be necessary for maintaining a healthy liver was a milestone in the history of the medical knowledge of alcohol

[65] *Ibid.*, p. 134.
[66] *Ibid.*, p. 134.
[67] *Ibid.*, p. 134.
[68] Charles S. Lieber, Leonore M. DeCarli, and Emanuel Rubin, 'Sequential Production of Fatty Liver, Hepatitis, and Cirrhosis in Sub-Human Primates Fed Ethanol with Adequate Diets', *Proceedings of the National Academy of Sciences of the United States of America* 72.2 (1975), pp. 437-41.
[69] *Ibid.*, p. 437.
[70] *Ibid.*, pp. 438-40.
[71] *Ibid.*, p. 440.

and the liver. By successfully isolating alcohol as the specific agent that harmed the liver, the two experimental studies on baboons invalidated the NDT.

Lieber's baboon experiments accomplished another major contribution to the knowledge of alcoholic liver disease. His 1975 paper with Rubin and DeCarli discussed how '[t]he experimental reproduction of the lesions of alcoholic hepatitis and the demonstration in an experimental model of its transition to cirrhosis support the hypothesis that alcoholic hepatitis is a precursor of the cirrhotic lesion.'[72] The mention of hepatitis, or the inflammation of the liver most commonly attributed to viral infection, was a new development that stemmed from Lieber's study. It was previously assumed that alcoholics were merely able to produce fatty liver, fibrosis, and cirrhosis, and an earlier paper by Rubin and Lieber challenged this by suggesting that hepatitis was a possible stepping-stone for fatty liver to develop into cirrhosis.[73] The significance of the baboon experiments rested on the fact that '[t]he entire constellation of histologic features characteristic of human alcoholic hepatitis has been produced for the first time in an experimental model'.[74] In essence, the baboon experiments inadvertently led to the establishment of the present model of 'alcoholic liver disease' as a three-step process involving the development of fatty liver, hepatitis, and cirrhosis.

Understandably, Lieber's studies had an enormous impact among specialists of liver disease. Already in 1975, the fifth edition of Sherlock's *Diseases of the Liver and Biliary System*, a celebrated textbook among gastroenterologists, noted that deficiency was now considered a mere 'possible factor' than a definitive cause of cirrhosis.[75] Unlike the third edition of Sherlock's textbook, the fifth also added a dedicated chapter on 'Alcohol and the Liver', signalling the resurgence of the discursive association between the two.[76] Sherlock cited Rubin and Lieber's 1968 study on non-alcoholic volunteers and the 1974 study on baboons to demonstrate that

[72] *Ibid.*, p. 440.
[73] Emanuel Rubin, and Charles S. Lieber, 'Experimental Alcoholic Hepatitis: A New Primate Model', *Science* 182.4113 (1973), pp. 712-713.
[74] *Ibid.*, p. 713.
[75] Sheila Sherlock, *Diseases of the Liver and Biliary System* (5th edn, Oxford, 1975), p. 430.
[76] *Ibid.*, pp. 445-60; Sherlock, *Diseases of the Liver.*

'[p]ure alcohol is undoubtedly directly hepatotoxic even in the presence of an adequate diet.'[77] Nancy K. Mello's 1976 review on the 'animal models for the study of alcohol addiction' also praised Rubin and Lieber for providing '[p]erhaps one of the most important recent contributions to research on alcoholism' through their 'development of the first animal model of alcohol-induced cirrhosis'.[78] The demise of the NDT was acknowledged even within the official organs of the WHO. A 1977 article on 'The morphology of cirrhosis: definition, nomenclature, and classification' published in the *Bulletin of the World Health Organization* recognised alcoholism alongside viral hepatitis as among the 'established etiological associations' of cirrhosis, while malnutrition was listed under 'debatable etiological factors'. The article added that '[i]t is doubtful if malnutrition by itself is ever a cause of cirrhosis in man. Protein deficiency, as seen in kwashiorkor, produces gross fatty change in the liver, but it does not lead to chronic liver disease.'[79] Later review articles and specialist textbooks published several years after the baboon experiments continued to attribute the establishment of alcohol's direct hepatotoxicity and the diminished aetiological importance of malnutrition to Lieber's studies.[80] At the same time, frequent references to malnutrition show that the role played by nutritional deficiency continued to be recognised to an extent, more as an additional factor to early stages of liver damage rather than as an indispensable cause. The NDT, which explicitly attributed malnutrition as the specific aetiology of cirrhosis, was entirely abandoned.

Charles S. Lieber's death on 1 March 2009 was reported by a handful of national newspapers in the United States. In *The New York Times*, Lieber was commemorated as 'a clinical nutritionist who upset scientific dogma by showing that alcohol in excess can cause

[77] *Ibid.*, pp. 449.

[78] Mello, 'Animal Models for the Study of Alcohol Addiction', p. 353.

[79] P. P. Anthony, K. G. Ishak, N. C. Nayak, H. E. Poulsen, P. J. Scheuer, and L. H. Sobin, 'The morphology of cirrhosis: definition, nomenclature, and classification', *Bulletin of the World Health Organization* 55.4 (1977), p. 524.

[80] Thomson, 'Alcohol and Nutrition', p. 412; McIntyre, and Morgan, 'Nutritional Aspects of Liver Disease', in Wright, Alberti, Karran, and Millward-Sadler (eds.), *Liver and Biliary Disease,* p. 118.; Mezey, 'Alcoholic Liver Disease', pp. 2709, 2714; Sherlock, 'Current Problems in Alcoholic Liver Disease', p. 104; Harold O. Conn, and Colin E. Atterbury, 'Cirrhosis', in Schiff, and Schiff (eds.), *Diseases of the Liver*, pp. 736-7.

cirrhosis despite an adequate diet'.[81] His 'findings upset conventional medical belief that cirrhosis was due to the poor nutrition commonly linked to alcoholism, not alcohol'.[82] This tribute aptly characterises Lieber's scientific contributions, whereby nutritional deficiency was discredited on behalf of the direct culpability of alcohol in cirrhosis. Although the decline of the NDT was primarily attributed by Herd and Katcher to the epidemiological studies that re-established the association between alcohol and cirrhosis, a detailed examination of the content of the original research demonstrates that its demise ultimately depended on the experimental reproduction of the disease in animal subjects. The scientific restoration of alcohol as a legitimate substance capable of damaging the liver played a central role in the wider reattribution of cirrhosis as a disease of alcohol consumption.

The Modern Direct Toxicity Theory: Cirrhosis as a Multifactorial Illness

The continued interest in diet as a predisposition, rather than a cause, of cirrhosis signalled the interest in other allied factors that predisposed individuals to further liver damage. The 1975 edition of Sherlock's *Diseases of the Liver and Biliary System* regarded malnutrition as a 'possible factor' to cirrhosis, while Patek argued that a deficient diet might make the liver more susceptible to the toxic action of alcohol.[83] Aside from nutrition, Esteban Mezey pointed to the causation of 'genetic, environmental, or nutritional' factors.[84] Furthermore, Allan D. Thomson highlighted the 'decisive importance' of individual susceptibility and other variables besides the toxicity of alcohol to the liver to explain why cirrhosis developed in no more than a minority of lifelong alcoholics.[85] Rather than resolving the long-contested question over its aetiology, the demise of the NDT and the demonstration of alcohol as the direct cause of cirrhosis paved a path towards a new set of unanswered questions on the complex web of causation surrounding the disease.

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[81] Lawrence K. Altman, 'Charles Lieber, Who Studied Alcohol as a Toxin, is Dead at 78', *The New York Times*, 10 March 2009, p. A29.
[82] *Ibid.*, p. A29.
[83] Sherlock, *Diseases of the Liver*, p. 430; Patek, 'Alcohol, Malnutrition, and Alcoholic Cirrhosis', pp. 1309-10.
[84] Mezey, 'Alcoholic Liver Disease', p. 2709.
[85] Thomson, 'Alcohol and Nutrition', p. 412.

The scientific framework of cirrhosis as a combined outcome of alcohol and other predisposing factors is referred to in this thesis as the modern direct toxicity theory (MDTT). As a theory founded on the direct action of alcohol on the liver, the MDTT crucially differed from the straightforward monocausal model of alcoholic causation of the DTT in its framing of cirrhosis as a multifactorial disease. The extent to which the liver could be harmed was understood to be determined by factors such as the volume and duration of alcohol consumption, genetic susceptibilities to the hepatotoxic action of alcohol, and various external or internal predispositions associated with class, occupation, gender, race, and so on. This was founded on a widely held contention that the disease was statistically prevalent in only a minority of lifelong heavy drinkers. No matter how much they drank over the course of ten or more years, most never went as far as to developing a cirrhotic liver, leading one to suspect that there must be an additional factor at play that put individuals at risk of the highest level of alcoholic liver damage. This notion is an example of a process in modern medicine described by Aronowitz as a shift from an 'ontological' view of disease whereby an illness was understood to exist as an independent entity to the patient, towards a 'holistic' view that took into account the individual and environmental contexts of its pathogenesis. The increasing reliance on population studies was part of a wider trend of identifying certain 'risk factors' to cirrhosis.[86] In order to understand the development of the knowledge on the determinants that correlated to a higher incidence of alcoholic cirrhosis, this section focuses on a loosely related collection of clinical and epidemiological studies from the second half of the twentieth century. In addition to the experimental demonstration of alcohol's direct toxicity to the liver, the framing of cirrhosis as a multifactorial disorder was directly adopted as an established understanding of the disease within the British medical profession as well as shaping the wider discussion on alcohol and public health that unfolded in the 1970s.

An interest in additional determinants that contributes to the incidence of cirrhosis could be traced to earlier debates on the epidemiological association between alcohol consumption and cirrhosis deaths. The particular exchange between two articles, one by

[86] Aronowitz, *Making Sense of Illness*, pp. 7-9.

Abraham M. Lilienfeld and Robert F. Korns (1950) and another by Milton Terris (1967), was understood by Herd and Katcher to be the specific instance when the causation of alcohol was re-established in the middle of the twentieth century.[87] The debate highlighted the importance of factors relating to the social class, income, and occupation of patients who had suffered from cirrhosis, inadvertently leading the way to a wider set of questions concerning the risks associated with the disease.

The article authored by Lilienfeld and Korns was intended as a critique of the notion that 'the association between alcoholism and cirrhosis is definitely established', a quote that they attributed to Jolliffe and Jellinek in their pioneering review article from 1941.[88] Within its summary of the various epidemiological studies of cirrhosis in relation to gender and race, the paper's most contentious claim concerned the impact of socioeconomic status and occupation. Pointing to a case study at Buffalo, New York, an industrial city situated in the manufacturing heartlands of the American Northeast, Lilienfeld and Korns noted a 'disproportional concentration of deaths from cirrhosis' among male residents of lower socioeconomic status over women.[89] This gender disparity led them to suspect that the toxic substances with which the workers, mostly men, were exposed to on a daily basis may have role in causing cirrhosis.[90] Although industrial pollution was not explicitly endorsed by the paper, it nevertheless suggested that scholars should look into it as a 'fertile field for epidemiological investigation'.[91]

The suggestion that alcohol and cirrhosis might not even be associated received a response from Terris in a review article from 1967.[92] In it, Terris noted a contradiction between the claim that a high mortality rate from cirrhosis was inversely correlated to males in lower-status occupations with a separate study in England and Wales, where the frequency of cirrhosis

[87] Herd, 'Ideology, history and changing models', p. 1119; Katcher, 'The Post-Repeal Eclipse', p. 732; Lilienfeld, and Korns, 'Some Epidemiological Aspects', pp. 65-81; Milton Terris, 'Epidemiology of Cirrhosis of the Liver: National Mortality Data', *American Journal of Public Health and the Nation's Health* 57.12 (1967), pp. 2076-88.
[88] Lilienfeld, and Korns, 'Some epidemiological aspects', p. 67; Jolliffe, and Jellinek, 'Vitamin Deficiencies', pp. 544-83.
[89] Lilienfeld, and Korns, 'Some Epidemiological Aspects', p. 74-6.
[90] *Ibid.*, p. 77.
[91] *Ibid.*, p. 77.
[92] Terris, 'Epidemiology of cirrhosis', pp. 2076-88.

was greatest among those of 'more comfortable backgrounds'.[93] Another study demonstrated that mortality rates from cirrhosis in England were higher among those in the service sector over manufacturing. The correlation was especially strong in professions in the recreational industry where workers were much more exposed to alcoholic beverages.[94] Furthermore, the correlation between cirrhosis and the service industry was greater than in the manufacturing sector, leading Terris to conclude that alcohol clearly possessed a stronger association to liver disease than industrial toxins. Thus, more than two decades after a similar supposition was made by Jolliffe and Jellinek, later observers subsequently noted that the publication of Terris's review article as the decisive moment when this association was finally set to stone.[95] Aside from the impact that the article had had on the statistical knowledge on alcohol and cirrhosis, the exchange between the two papers was illustrative of how useful epidemiology was in highlighting the importance of social determinants in aiding the identification of the specific causative agent and of how such determinants contributed to the pathogenesis of an illness. Terris's article paved the way towards a wider interest in additional factors that predisposed individuals to becoming more susceptible to alcoholic liver damage.

Following the demonstration of the direct toxicity of alcohol in the 1970s, a small number of physicians began to suspect the importance of additional predispositions to the pathogenesis of cirrhosis. The reality that cirrhosis only ever developed in no more than a minority of heavy drinkers loomed large in the minds of many hepatologists, forcing some like Klatskin to suspect that 'differences in individual susceptibility to the effects of chronic alcoholism or that other associated factors play a significant role' whereby 'endogenous [internal] factors may be important in determining whether or not cirrhosis develops'.[96] In another review article, Klatskin enquired whether variations among individuals in their susceptibility 'are genetically determined or are due solely to environmental factors, such as the

[93] *Ibid.*, p. 2082.
[94] *Ibid.*, pp. 2082-4.
[95] Feinman, and Lieber, 'Liver disease in alcoholism', p. 303; Brenner, 'Trends in Alcohol Consumption', p. 1279; Lelbach, 'Cirrhosis in the alcoholic', p. 105; Room, 'Alcohol Control and Public Health', pp. 293-4.
[96] Klatskin, 'Effect of alcohol on the liver', p. 1671.

diet.'[97] Investigations into the determinants behind cirrhosis were evidently motivated by a need to explain the statistical rarity of cirrhosis among heavy drinkers.

While it had been known that cirrhosis was predicated on the persistent, long-term consumption of large volumes of alcohol, some scholars sought to devise an estimate of the point in which alcohol consumption began to pose a serious risk to the liver. Werner K. Lelbach, a physician based in West Germany, published a study in 1975 addressing whether the probability of developing cirrhosis increased with the volume of alcohol consumed.[98] His research was founded on two premises. First, Lieber's baboon experiments showed that the prolonged consumption of alcohol doubtless had a 'deleterious effect on the liver'.[99] Second, in spite of the demonstration of alcohol as the direct cause, cirrhosis only developed in a minority of heavy drinkers, a proportion that ranged, according to Jolliffe and Jellinek, between 2.4 to 28 per cent.[100] Lelbach, therefore, stated that there existed an 'unresolved discrepancy' between high rates of alcoholism among sufferers of cirrhosis and the low incidence of cirrhosis among alcoholics.[101] After studying the clinical and histological data of 526 male alcoholics who underwent voluntary withdrawal treatment in a sanatorium, an association between the intake of alcohol and the incidence of cirrhosis was noted.[102] Based on the results, Lelbach concluded that, '[u]p to a certain intake per time unit, alcohol is readily metabolized and completely disposed of by several physiologic mechanisms. However, if the ingestion of alcohol exceeds the capacity of these systems, the pharmacologic or toxic aspect of this molecule becomes manifest' in causing the pathogenesis of cirrhosis.[103] Thus, the causation of alcoholic cirrhosis was marked by a dose-response relationship in which the risk of developing cirrhosis increased when the volume of alcohol consumed surpassed a certain threshold. While Lelbach admitted that '[t]he specific factors predisposing an alcoholic are biologically still an enigma… individual

[97] Klatskin, 'Newer concepts of cirrhosis', p. 899.
[98] Lelbach, 'Cirrhosis in the alcoholic', pp. 85-105.
[99] *Ibid.*, p. 98.
[100] *Ibid.*, pp. 85.
[101] *Ibid.*, p. 100.
[102] *Ibid.*, pp. 100-1.
[103] *Ibid.*, p. 99.

susceptibility—whatever its biological counterpart may be—plays an equally important role' to the alcohol intake.[104]

Lelbach's inquiry was later expanded on by a Paris-based nutritionist named G. Péquignot.[105] In 1978, Péquignot calculated the '[r]elative risk of ascetic cirrhosis' to 'different levels of daily alcohol consumption' by conducting a questionnaire on the food and alcohol consumption habits of 184 hospitalised male cirrhotic patients and a control group of non-hospitalised 778 males.[106] The study showed that the likelihood of developing the disease decreased on average by 80 per cent if the drinker refrained from consuming more than 40 grams of pure alcohol per day, equivalent to five units of alcohol under the present UK health guidelines. Although Péquignot refrained from interpreting these results as an objectively 'safe' level of drinking, admitting that it was 'out of the question in a nature of this study' to make such judgements, both his and Lelbach's studies allowed for the possibility of devising a parameter of consumption whereby it would begin to pose a serious risk to health.[107] Evidence towards the notion that one had to satisfy a certain minimum intake of alcohol to develop cirrhosis highlighted the inherent complexity of the disease, failing to be reduced to a straightforward understanding that was merely based on the toxic action of alcohol on the liver.

The interest in the multifactorial nature of cirrhosis culminated in a set of studies that sought to identify the specific endogenous factors that exposed individuals to alcoholic cirrhosis. In the 1970s and 80s, studies into such factors were carried out by Roger Williams, a prominent hepatologist based at the King's College Hospital known among hepatologists for carrying out the first successful liver transplant in Britain in 1968. Williams is known for establishing the Liver Unit in 1973, the first intensive care unit for patients with liver disease in the country.[108] Here, he and his colleagues performed studies that looked into the genetic and gender predispositions that potentiated serious alcoholic liver damage.

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[104] *Ibid.*, p. 91, 99.
[105] G. Péquignot, A. J. Tuyns, and J. L. Berta, 'Ascitic Cirrhosis in Relation to Alcohol Consumption', *International Journal of Epidemiology* 7.2 (1978), pp. 113-20.
[106] *Ibid.*, p. 113-4.
[107] *Ibid.*, p. 119.
[108] Eunice X. Xu, 'Living legend in surgery: Professor Roger Williams', *Hepatobiliary Surgery and Nutrition* 3.6 (2014), pp. 423-4.

The first point of interest for Williams was how genetics contributed to the wide variations across individuals in their susceptibility to liver disease. At the time, the role of heredity in cirrhosis had been studied only by a handful of researchers. A paper from 1966 by a team at Copenhagen looked into the association between cirrhosis and different genes that corresponded to the production of certain types of antibodies. Anticlimactically, the authors found 'no evidence of a familial occurrence of liver cirrhosis.'[109] A separate article from 1968 on a clinical study of cirrhosis patients at Baltimore looked into the correlation of the disease with specific genetically determined traits. It showed that cirrhosis was strongly associated with white people with brown hair and fair skin, and with black people belonging to the Duffy blood group.[110] It concluded that there was 'a broad multifactorial base for susceptibility to cirrhosis and an increased frequency of these factors in Whites compared with non-Whites'.[111] Williams made his own contributions upon his recognition that the interest in genetic predispositions were limited at the time. His investigations were primarily immunogenetic in nature, inspired by his previous work on liver transplantation that involved the identification of genes that signalled the tissue compatibility between the donor and the recipient of the organ.[112]

In 1976, Williams published his first study that sought to isolate the prevalent gene among patients who developed alcoholic cirrhosis.[113] After 'typing' the tissues of the participants, he and his colleagues identified that 45 per cent of cirrhotic patients carried HLA-B8, a variant of one of the human leukocyte antigen (HLA) group of genes, as opposed to its lower prevalence among 25 per cent of non-cirrhotic control group.[114] The incidence of this gene variant among patients who developed cirrhosis indicated that genetically determined

[109] P. Elling, P. Ranløv, and P. Bildsøe, 'A Genetic Approach to the Pathogenesis of Hepatic Cirrhosis: a clinical and serological study', *Acta Medica Scandinavica* 179.5 (1966), p. 533.
[110] N. C. R. W. Reid, P. W. Brunt, W. B. Bias, W. C. Maddrey, B. A. Alonso, and F. L. Iber, 'Genetic Characteristics and Cirrhosis: a controlled study of 200 patients', *The British Medical Journal* 2.5603 (1968), pp. 463-5.
[111] *Ibid.*, p. 465.
[112] Clyde F. Barker, and James F. Markmann, 'Historical overview of transplantation', *Cold Spring Harbor Perspectives in Medicine* 3.4 (2013), pp. 1-18.
[113] R. J. Bailey, N. Krasner, A. L. W. F. Eddleston, Roger Williams, D. E. H. Tee, Deborah Doniach, L. A. Kennedy, J. R. Batchelor, 'Histocompatibility antigens, autoantibodies, and immunoglobulins in alcoholic liver disease', *The British Medical Journal* 2 (1976), pp. 727-9.
[114] *Ibid.*, pp. 727-8.

immune responses might play an important role in the pathogenesis of the disease.[115] Williams continued his investigation on HLA-B8 in a later paper from 1978, the results of which reiterated that the gene was more frequent among patients who developed cirrhosis than among a control group of patients who never went beyond fatty liver or fibrosis.[116] The authors concluded that 'it is possible that there may be a more direct link between this genetic marker and the toxic effect of ethanol on the liver'.[117] Eventually in 1982, an even stronger association between alcoholic cirrhosis and HLA-B8 was established in a paper where he observed that carrying the gene did indeed 'enhance the rate of development of liver damage' by about 50 per cent.[118] Although Williams was open to the possibility that HLA-B8 might also play a role in altering the individual's drinking habits, his investigation succeeded in identifying the specific gene that (at least mildly) correlated with serious liver damage. As the first serious set of long-term studies on the genetic predispositions of cirrhosis, Williams shed more light on the notion that alcohol worked alongside certain inborn factors in harming the liver.

Williams was also interested in gender-related factors in liver disease. In 1977, he and his colleagues at the Liver Unit published paper that explained how the rapid rise in cirrhosis deaths among women was tied to the growth of female alcohol consumption.[119] Using a survey of 293 patients with alcoholic liver disease, the study demonstrated that women not only had a 'significantly higher incidence of alcoholic hepatitis' than men did, the prognosis was significantly lower.[120] In a later article from 1981, the same team of researchers reviewed the existing knowledge on the question of whether 'women develop alcoholic liver disease more readily than men'.[121] The article suspected that '[d]ifferences in body size and composition are

[115] *Ibid.*, p. 728.
[116] A. L. W. F. Eddleston, and Roger Williams, 'HLA and liver disease', *British Medical Bulletin* 34.3 (1978), p. 299.
[117] *Ibid.*, pp. 299-300.
[118] J. B. Saunders, A. D. Wodak, A. Haines, P. R. Powell-Jackson, B. Portmann, M. Davis, and Roger Williams, 'Accelerated development of alcoholic cirrhosis in patients with HLA-B8', *The Lancet* 319.8286 (1982), pp. 1381-4.
[119] *Ibid.*, p. 1499.
[120] N. Krasner, M. Davis, B. Portman, and Roger Williams, 'Changing pattern of alcoholic liver disease in Great Britain: relation to sex and signs of autoimmunity', *The British Medical Journal* 1.6075 (1977), pp. 1497-500.
[121] J. B. Saunders, M. Davis, and Roger Williams, 'Do women develop alcoholic liver disease more readily than men?', *The British Medical Journal* 282 (1981), pp. 1140-3.

partly responsible for the greater susceptibility of women, but differences in immune reactivity between the sexes may also play a part.'[122] Between 1959 and 1970, in the midst of a context in which alcoholic cirrhosis was primarily understood as a 'disease of middle-aged and elderly men' in Britain, statistical reports from district hospitals and specialist units had shown that the male-female ratio of the incidence of the disease was five to one. However, more recent epidemiological studies saw the ratio narrow to two to one in 1980, simultaneously reflecting the disproportionate rise in convictions from drunkenness and admissions for treatment from alcohol addiction among women.[123] More interestingly, a larger proportion of female alcoholics developed alcoholic hepatitis or cirrhosis over men.[124] Williams understood this as an outcome of the physiological differences between the two sexes. Aside from the fact that women are physically smaller on average, the rate of absorption of alcohol is affected by having a higher body fat percentage. Since alcohol 'diffuses slowly' in fat owing to the tissue's poorer blood supply, the 'administration of alcohol will result in higher systematic blood concentrations in women than in men and might reasonably be expected to cause more liver damage'.[125]

The above article took into account other possible factors, such as variations in drinking practices and habits between the genders that would doubtless impact the duration and volume of consumption. The evidence was accumulating that women, whatever the reason, were much more at risk of severe alcoholic liver damage. Williams thus concluded that '[g]reater emphasis must be placed on designing abstinence programmes specifically for female patients, on earlier detection of liver disease, and on educating women about hazardous drinking levels.'[126] Additionally, he supported the establishment of separate guidelines for women in their recommended limit of alcohol intake.[127] Alongside his genetic and gender studies, Williams played an instrumental role in spearheading the earliest investigations into how individual susceptibilities potentiated the pathogenesis of cirrhosis. The conceptual formation of the

[122] *Ibid.*, p. 1140.
[123] *Ibid.*, p. 1140.
[124] *Ibid.*, p. 1141.
[125] *Ibid.*, p. 1142.
[126] *Ibid.*, p. 1140.
[127] *Ibid.*, p. 1143.

MDTT depended on the framing of the disease as an outcome of numerous factors that exposed the liver to the toxic action of alcohol.

The Modern Direct Toxicity Theory and the British Medical Profession

The shift towards the MDTT was progressively acknowledged by the British medical profession after the 1970s. As revealed in both the general medical journals and textbooks of general medicine, the NDT was all but abandoned within mainstream medical practice, with many of the texts placing a particular emphasis on Lieber's studies in precipitating its demise. Furthermore, alcoholic cirrhosis was often understood in relation to the additional role of individual and environmental determinants. Partly due to the continued ambiguity over the exact factors that put individuals at risk of developing cirrhosis, a large proportion of the texts showed restraint by expressing uncertainty over the precise mechanism of the pathogenesis of the disease. Therefore, the understandings that were disseminated across the medical profession show that the contested knowledge on cirrhosis causation was far from being settled.

The impact of the shift from the NDT to the MDTT was immediately evident in its reception in *The Lancet* and *The British Medical Journal.* A handful of articles that predated Lieber's 1974 baboon experiments had already shown sympathy towards the idea that alcohol played a direct role in damaging the liver. In 1966, a *British Medical Journal* column remarked that the question over the aetiology of cirrhosis 'baffled the experts for years, and… the affirmative indicates that the relation between alcohol and cirrhosis is a complex one.' While it was conceded that alcohol was 'probably not a true hepatotoxin', the article critiqued the NDT for failing to account for the fact that 'many alcoholic cirrhotics are well nourished'.[128] Later in 1968, an article in *The Lancet* discussed that, although 'the widely accepted view has been that the hepatic injury in alcoholism is the outcome of nutritional deficiencies... doubts have arisen whether malnutrition is the true cause.'[129] Concerning the recent developments in the clinical treatment of patients with cirrhosis, another column admitted that 'the outstanding problem is

[128] Anon., 'Any Questions', *The British Medical Journal* 1.5488 (12 March 1966), p. 659.
[129] Anon., 'Alcohol and the Liver', *The Lancet* 292.7569 (21 September 1968), pp. 670-1.

its aetiology and pathogenesis', adding that '[e]ven the role of malnutrition in this sequence is not unequivocally established' since 'some patients with alcoholism gets cirrhosis without any obvious associated dietary deficiency'.[130] These articles show that the late 1960s was the NDT's last stand. The near-unanimous acceptance of alcohol over malnutrition as the likely cause of the disease by the mid-1970s was thus not entirely without its antecedents, even within general medical journals.

The journals additionally took notice of the gradual accumulation of evidence in support of the notion that alcohol possessed a direct toxic action on the liver. The shift towards an aetiological theory that attributed cirrhosis to alcohol over malnutrition was frequently mentioned in reference to Lieber's clinical and experimental studies. Already in 1968, *The Lancet* took note of Rubin and Lieber's clinical studies as an indication that 'alcohol is directly poisonous to the liver'.[131] In the same year, *The British Medical Journal* pointed to how Lieber 'stressed the importance of alcohol as a toxic agent in the production of fatty liver or acute alcoholic hepatitis', while deficiency was 'now thought to be less important'.[132] By 1978, *The Lancet* argued that Lieber's baboon studies demonstrated that alcohol 'has a direct toxic effect on the liver'.[133] Another article from 1978 credited the baboon experiments for successfully producing 'alcoholic hepatitis and cirrhosis' that were 'morphologically similar to that seen in man'. It additionally commended Lelbach and Péquignot's dosage studies for demonstrating the positive association between the volume of alcohol and the extent of liver damage, which in turn provided 'strong evidence in support of a direct hepatotoxic effect of alcohol'.[134]

The journals were also interested in the studies surrounding individual and environmental susceptibilities. A column from 1978 admitted that, while alcohol could confidently be understood as a direct hepatotoxin, there still was very little knowledge on why alcoholic hepatitis and cirrhosis developed in only a minority of heavy drinkers.[135] *The British*

[130] Anon., 'Natural History of Cirrhosis', *The Lancet* 291.7551 (18 May 1968), p. 1076.
[131] Anon., 'Alcohol and the Liver', pp. 670-1.
[132] Anon., 'Slow Progress in Cirrhosis', *The British Medical Journal* 2.5603 (25 May 1968), pp. 445-6.
[133] Anon., 'Towards Prevention of Alcoholic Liver Disease', *The Lancet* 312.8083 (12 August 1978), pp. 353-4.
[134] Anon., 'Chronic Effects of Alcohol', *The British Medical Journal* 2.6134 (5 August 1978), pp. 381-2.
[135] Anon., 'Towards Prevention of Alcoholic Liver Disease', p. 354.

Medical Journal highlighted the importance of the social determinants of alcohol consumption, whereby an individual's likelihood of developing liver disease was understood to be shaped by alcohol's 'availability and cost and his income'. However, the author pointed out that, although the 'average intake of alcohol necessary to cause damage is becoming established… the nature of individual susceptibility is still imperfectly understood.'[136] A column titled 'how does alcohol damage the liver?' in *The British Medical Journal* similarly noted that it was 'unknown' why the likelihood of developing cirrhosis varied across individuals. As a partial response to this question, the author cited a study by Péquignot that indicated that the susceptibility towards alcoholic liver damage varied between men and women.[137] Instead of presenting a simplistic account of scientific change where alcohol ousted deficiency as the acknowledged cause of cirrhosis, articles in general medical journals show that the complex nature of its multifactorial causation was aptly disseminated throughout the British medical profession.

The shift towards the MDTT was abundantly discussed in textbooks of general medicine as well. In this chapter, multiple editions of seven different series of textbooks from between 1966 and 1990, a total of 18 publications, have been examined to assess the impact of the demise of the NDT (see Figures 11; see Appendix: Textbooks of General Medicine for long-term shifts). The wider reception and impact of every textbook studied in this section was accounted for in the previous chapters, except for one: the *Oxford Textbook of Medicine*.[138] This specific textbook was immediately renowned upon its maiden publication in 1983. A massive project jointly produced by 281 contributors, three-quarters of whom were based in Britain, *The British Medical Journal* praised it as 'a brand new phoenix arising from the ashes of Price' that stood alongside other giants such as Harrison's *Principles of Internal Medicine* and Cecil's *Textbook of Medicine*. The textbook was additionally referred to as the 'latest jewel of scholarship from England's oldest university, and one at least has made a resolution to try to

[136] Anon., 'Chronic Effects of Alcohol', pp. 381-2.
[137] Anon., 'How Does Alcohol Damage the Liver?', *The British Medical Journal* 2.6154 (23-30 December 1978), pp. 1733-4.
[138] D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrell (eds.), *Oxford Textbook of Medicine* (1st edn, Oxford, 1983).

read it right through, like the Bible, a few pages at a time'.[139] Another review from 1987 referred to its second edition as 'a remarkably successful textbook', stating that '[a]ll medical libraries, from teaching hospital to postgraduate centre in the district hospital, should possess a copy, as should any well run general practice'.[140]

FIGURES 11 Textbooks of General Medicine, 1966~1990

	1966~70	1971~75	1976~80
Russell Cecil, *A Textbook of Medicine*	12th (1967) Very little known on factors that determine individual susceptibility to cirrhosis, but importance of 'genetic and dietary factors'		15th (1979) identical claim to 12th edn
Frederick W. Price, *A Textbook of the Practice of Medicine*	10th (1966) Cirrhosis caused by 'dietary deficiencies acting indirectly by interfering with the nutrition of liver cells'	11th (1973) 'cirrhosis is due to the harmful effects of alcohol on the liver', while actual factor producing it is 'unknown' even if some liver damage is 'guaranteed' if you drink	12th (1978) identical claim to 11th edn
John Conybeare, *A Textbook of Medicine*		16th (1975) Alcohol included among 'Toxins' with Iron and Copper as possible aetiologies of cirrhosis, does not include nutritional deficiency	
Derrick Dunlop, *Textbook of Medical Treatment*	10th (1966) known causes of cirrhosis include alcohol, deficiency, and poisons, but aetiological effect of alcohol is unclear. Possibility that drinking is an indirect factor to deficiency.	12th (1971) Cirrhosis covers variety of liver disorders that can be 'viral infection, alcohol or nutritional deficiency', but aetiology 'is unknown in many instances'	
T. R. Harrison, *Principles of Internal Medicine*	6th (1970) 'epidemiologic studies have implicated' alcoholism as the cause of cirrhosis, but 'there is still no definitive evidence that alcohol by itself leads to cirrhosis'. While a 'contributing factor to the evolution of cirrhosis... malnutrition per se does not lead to Laennec's cirrhosis' but 'a combination of chronic alcohol ingestion plus impaired nutrition leads to liver cell damage and Laennec's cirrhosis'.	7th (1974) 'epidemiologic studies have implicated' alcoholism as the cause of cirrhosis. While a 'contributing factor to the evolution of cirrhosis... malnutrition per se does not lead to Laennec's cirrhosis' but 'a combination of chronic alcohol ingestion plus impaired nutrition leads to liver cell damage and Laennec's cirrhosis'.	8th (1977) identical claim to 7th edn
Stanley Davidson, *The Principles and Practice of Medicine*	9th (1968) 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency' due to experiments on animals, while 'alcohol itself does not produce cirrhosis of the liver'	11th (1974) 'It is generally agreed that dietary deficiency plays a part in the genesis of some cases of hepatic cirrhosis.'	

	1981~85	1986~1990
Russell Cecil, A Textbook of Medicine	17th (1985) 'Ethanol is an hepatotoxin' but nutrition also seems to play some role	
T. R. Harrison, Principles of Internal Medicine	10th (1983) identical claim to 7th edn	
Stanley Davidson, The Principles and Practice of Medicine	13th (1981) 'The mechanism whereby alcohol damages the liver is unknown; it is now, however, accepted, as a direct liver toxin in man and in other primates'	
Oxford Textbook of Medicine	1st (1983) Alcohol and hepatitis B account for majority of aetiology in western world, alcohol's aetiology taken for granted without discussion	2nd (1987) 'The vast majority are due to alcohol, hepatitis B, or non-A, non-B hepatitis' but '[s]ince only a proportion of heavy drinkers develop alcoholic liver disease, it has been suggested that susceptible individuals might metabolize alcohol abnormally.'

[139] Paton [review], 'D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrell (eds.)', pp. 1030-1.

[140] Christopher Booth [review], 'D. J. Weatherall, J. G. G. Ledingham, and D. A. Warrell (eds.), *Oxford Textbook of Medicine* (2nd edn, Oxford, 1987)', *The British Medical Journal (Clinical Research Edition)* 295.6597 (29 August 1987), pp. 546-7.

The earliest textbooks from the period studied in this chapter reveal that many authors were already in doubts of the NDT before 1970s. In contrast to how the 1963 edition of Cecil's *Textbook of Medicine* admitted that '[j]ust how alcoholism, malnutrition, or both produces hepatic injury is unknown' while arguing that alcohol 'has not been conclusively proved to have a directly injurious action upon the liver cells', the subsequent 1967 edition entirely omitted the latter passage while highlighting the importance of 'genetic and dietary factors'.[141] This editorial decision reflected a sense that it was no longer feasible to categorically disregard the causation of alcohol, even if its direct toxicity was still uncertain at the time. Although the 1970 edition of Harrison's *Principles of Internal Medicine* acknowledged that 'there is still no definitive evidence that alcohol by itself leads to cirrhosis', it conceded that recent epidemiological studies had strengthened the association between cirrhosis and heavy drinking to the extent that it was no longer unreasonable to suppose that alcohol could be a 'major cause of Laennec's cirrhosis'.[142] The last textbook that attributed cirrhosis to nutritional deficiency without mentioning alcohol was the 1974 edition of Davidson's *Principles and Practice of Medicine.* The volume stated that 'dietary deficiency *plays a part* in the genesis of some cases of hepatic cirrhosis', a deliberately ambiguous statement that refused to conclusively endorse either alcohol or malnutrition.[143] This, however, stood in contrast to its previous 1968 edition, which suggested not only that there was 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency' but also that 'alcohol itself does not produce cirrhosis of the liver'.[144] There was a general sense of uncertainty and doubt in textbooks that were published during and after the late-1960s over the nutritional aetiology that had dominated the previous couple of decades. Much like the general medical journals, this suggests that the full confirmation of the direct toxicity of alcohol following Lieber's 1974 study should not have come as much of a surprise to the authors and readers of the textbooks.

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[141] Sleisenger, 'Diseases of the Digestive System', p. 1039; Jefferies, 'Diseases of the Liver', p. 983.
[142] William A. Tisdale, and Kurt J. Isselbacher, 'Cirrhosis', in Maxwell M. Wintrobe, George W. Thorn, Raymond D. Adams, Ivan L. Bennett, Eugene Braunwald, Kurt J. Isselbacher, and Robert G. Petersdorf (eds.), *Harrison's Principles of Internal Medicine* (6th edn, New York, 1970), p. 1547.
[143] John MacLeod (ed.), *Davidson's Principles and Practice of Medicine* (11th edn, Edinburgh, 1974), p. 1035.
[144] Davidson (ed.), *The Principles and Practice of Medicine*, pp. 1021-2.

As nutritional deficiency was gradually abandoned as the sole or primary cause of cirrhosis, textbooks published after 1970 shifted towards attributing the disease to the toxicity of alcohol. 77 per cent (10 out of 13) of those published post-1971 specified alcohol as the most likely cause of cirrhosis, often by describing the substance as a 'toxin' to the liver. While arguing that nutrition might still play some secondary role, the 1985 edition of Cecil's *Textbook of Medicine* labelled alcohol as the 'hepatotoxin' and the 'most common cause of liver disease in the Western world'.[145] The 1983 edition of the *Oxford Textbook of Medicine* simply pointed out that alcohol and hepatitis B accounted for the majority of cirrhosis in the Western world without going into much detail on alcohol's causal mechanism of its pathogenesis, indicating that the author took the hepatotoxicity of alcohol for granted to the extent that an explanation was not even warranted.[146]

Some of the textbooks, however, went short of labelling alcohol as the indisputable aetiology of cirrhosis, preferring instead to point to it as the most likely cause. The final edition of Conybeare's *Textbook of Medicine* from 1975 listed alcohol alongside iron and copper as one of the likely 'toxins' responsible for causing cirrhosis.[147] All three consecutive editions of Harrison's *Principles of Internal Medicine* from between 1974 and 1983 remarked on how '[m]any epidemiologic studies have implicated chronic alcoholism as a major cause of Laennec's cirrhosis', adding that patients 'should understand clearly that neither nutritious diet nor added vitamins will protect his liver against the effects of further alcohol'.[148] Although most of the textbooks were comfortable with the notion that alcohol possessed a direct toxic action

[145] Thomas D. Boyer, 'Cirrhosis of the Liver', in James B. Wyngaarden, and Lloyd H. Smith (eds.), *Cecil Textbook of Medicine* (17th edn, London, 1985), p. 836.

[146] S. Sherlock, 'Cirrhosis of the Liver', in Weatherall, Ledingham, and Warrell (eds.), *Oxford Textbook of Medicine* (1st edn), p. 12.182.

[147] R. P. H. Thompson, 'The Liver and the Gall Bladder', in W. N. Mann (ed.), *Conybeare's Textbook of Medicine* (16th edn, London, 1975), p. 347.

[148] William A. Tisdale, J. Thomas LaMont, and Kurt J. Isselbacher, 'Cirrhosis', in Maxwell M. Wintrobe, George W. Thorn, Raymond D. Adams, Eugene Braunwald, Kurt J. Isselbacher, and Robert G. Petersdorf (eds.), *Harrison's Principles of Internal Medicine* (7th edn, New York, 1974), pp. 1540-2; J. Thomas LaMont, and Kurt J. Isselbacher, 'Cirrhosis', in George W. Thorn, Raymond D. Adams, Eugene Braunwald, Kurt J. Isselbacher, and Robert G. Petersdorf (eds.), *Harrison's Principles of Internal Medicine* (8th edn, New York, 1977), p. 1604; J. Thomas LaMont, Raymond S. Koff, and Kurt J. Isselbacher, 'Cirrhosis', in George W. Thorn, Raymond D. Adams, Eugene Braunwald, Kurt J. Isselbacher, Joseph Martin, Jean Wilson, and Robert G. Petersdorf (eds.), *Harrison's Principles of Internal Medicine* (10th edn, New York, 1983), pp. 1804-5.

on the liver, the ambiguity over their stance on the aetiology of cirrhosis could be interpreted as an indication of scientific restraint, an acknowledgement of the fact that the question was far from being settled.

Indeed, seeing as how many of the textbooks took note of the multiplicity of factors that contribute to cirrhosis, the aetiology of the disease was clearly understood to be far more complicated than one that could be simply be attributed to the action of alcohol alone. Remarkably, only 3 out of 13 (23 per cent) textbooks published after 1971 described cirrhosis through the action of alcohol without referring the continued uncertainties over the mechanism of the pathogenesis or the contribution of additional factors. While most textbooks from the period attributed cirrhosis to alcohol, the 1981 edition of Davidson's *Principles and Practice of Medicine* recognised alcohol 'as a direct liver toxin in man and in other primates'. It additionally pointed out that the 'mechanism whereby alcohol damages the liver is unknown' because cirrhosis develops in no more than a small proportion of heavy drinkers.[149] Both the 1973 and 1978 editions of Price's *Textbook of the Practice of Medicine* argued that 'cirrhosis is due to the harmful effects of alcohol on the liver', even though the actual factor that contributed to its production was 'unknown'.[150] The second edition of the *Oxford Textbook of Medicine* from 1987 argued that, '[s]ince only a proportion of heavy drinkers develop alcoholic liver disease, it has been suggested that susceptible individuals might metabolize alcohol abnormally, or that they may show an immunological reaction to cell constituents altered by alcohol or its metabolites resulting in cell death.'[151] Factors other than alcohol itself were referred to by the authors as a way explain that the disease, while predominantly attributable to the toxicity of alcohol, only developed among a minority of heavy drinkers.

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[149] N. D. C. Finlayson, and John Richmond, 'Diseases of the liver and biliary tract', in John MacLeod (ed.), *Davidson's Principles and Practice of Medicine* (13th edn, Edinburgh, 1981), p. 401.

[150] A. E. Read, 'Diseases of the Liver, Gallbladder and Pancreas', in Ronald Bodley Scott (ed.), *Price's Textbook of the Practice of Medicine* (11th edn, London, 1973), p. 642; A. E. Read, 'Diseases of the Liver, Gallbladder and Pancreas', in Ronald Bodley Scott (ed.), *Price's Textbook of the Practice of Medicine* (12th edn, London, 1973), p. 573.

[151] R. Wright, 'Cirrhosis of the Liver', in Weatherall, Ledingham, and Warrell (eds.), *Oxford Textbook of Medicine* (2nd edn), p. 12.228.

Journals and textbooks suggest, then, that the scientific shift from the NDT towards the MDTT shaped the dominant professional medical understandings on alcohol and the liver in Britain after the 1970s. The authors of these texts were receptive to the establishment of alcohol as a direct toxin to the liver and the profound scientific interest developing around the multifactorial nature of cirrhosis. The transformative impact of Lieber's clinical and experimental studies were frequently highlighted, especially his baboon experiments from 1974. Many of the textbooks additionally refused to settle the question on the aetiology of cirrhosis with a description based purely on the straightforward causation of alcohol. Instead, the disease was understood as an intricate outcome of the direct toxic action of alcohol working alongside other individual and environmental factors. Many of the texts thus showed restraint by acknowledging the continued uncertainties over the predispositions that contribute to cirrhosis. Admittedly, the medical knowledge on the precise aetiology of the disease was still incomplete, even after the decisive demonstration of alcohol's direct culpability in the disease.

Politics, Public Health, and the Liver

The establishment of the MDTT had a profound impact on the wider public discussion on alcohol in Britain during the 1970s and 80s. Much more so than previously, the question on the relationship between alcohol and the liver manifested itself in a myriad of ways outside the immediate realm of professional medicine. Following a prolonged period of negligible conflicts over drink that had been going on since the end of the First World War, an alliance of medical experts and health campaigners reopened the dialogue on the alcohol problem in the 1970s. The group approached alcohol misuse as a public health issue by calling for the implementation of stricter controls on the availability of alcoholic beverages to reduce consumption and harm. Liver disease was at the heart of this new debate. A renewed understanding of cirrhosis as a disease most commonly associated with alcohol heralded a wider discussion on the various problems caused by drink. A statistical model whereby cirrhosis deaths were tied to *per capita* levels alcohol consumption formed the basis of the total consumption model, the theoretical

pillar of the public health approach that justified the problematisation of all forms of drinking, both moderate and excessive.

In reaction to the renewed interest in the alcohol problem, the beverage industry instead chose to promote educational solutions to encourage moderate, sensible attitudes towards alcohol. Internal materials of the Brewers' Society reveal that the industry sought to adapt to the shifting aetiological understanding of cirrhosis by attempting to disassociate beer from the disease, and, most surprisingly, by funding Roger Williams's studies on the genetic and gender susceptibilities towards cirrhosis. Thus, the shift towards the MDTT presented a unique opportunity whereby certain aspects of the new knowledge on cirrhosis was selectively highlighted by both sides to promote their narrative on the alcohol problem. Public health campaigners depended on the scientific establishment of alcohol's direct toxicity to the liver to target consumption to prevent the growing prevalence of problem drinking. On the other hand, the brewing industry took advantage of the framing of cirrhosis as a multifactorial disease as a way to downplay the disease culpability of their products, stressing the importance of factors other than alcohol itself in causing the disease.

Following the 1960s, Britain underwent a sustained increase in its *per capita* levels of alcohol consumption, accompanied by a sharp rise in deaths from cirrhosis.[152] This marked growth in consumption was attributable to a number of factors that included the exponential growth of disposable incomes under the postwar economic boom, the gradual liberalisation of licensing laws after the 1960s, and the rise of alcohol consumption among women. These simultaneous developments resulted in the resurgence of public concerns over the social and economic costs of alcohol misuse. Within this context, a new approach to alcohol policy based on the epidemiology of consumption and harm across whole populations emerged. According to Thom, this shift was signalled by the publication of a 1971 Home Office report, which framed the alcohol problem under the growing statistical incidence of 'alcohol-related harm' in relation to the increase of overall levels of consumption.[153] The previously dominant approach based on

[152] Nicholls, *The Politics of Alcohol*, p. 204.
[153] Thom, *Dealing with Drink*, pp. 7-8; Anon., *Habitual Drunken Offenders: Report of the Working Party* (London, 1971).

the disease concept of alcoholism, promoted by E. M. Jellinek and his colleagues at the Yale Centre for Alcohol Studies, advocated the treatment of individual alcoholics as the paramount solution to the alcohol problem. Instead, the new public health approach called for prevention by targeting all drinkers as being potentially 'at risk' of a wide variety of health and social problems tied to alcohol.[154] This model was inspired by an earlier observation made by Sully Ledermann, the statistician who discovered the existence of a direct relationship between *per capita* levels of alcohol consumption and the level of alcohol misuse.[155] If the overall levels of consumption in a population doubled, the number of drinkers engaging in 'problem drinking', as well as mortality rates from alcohol-related illnesses, could potentially multiply by a factor of four or eight. Contrasted to Jellinek's disease concept of alcoholism, which understood any form of 'problem drinking' to be restricted to a set minority of drinkers at any point, Ledermann's model suggested that all drinkers were potentially at risk of becoming a 'problem drinker'. This led to the conclusion that an effective policy should focus on preventative measures such as stricter licensing laws, a ban on beverage advertising, and higher alcohol taxes, all of which would be implemented under the goal of reducing overall levels of both heavy and moderate consumption to minimise the incidence of 'problem drinking'. This assumption formed the basis of the total consumption model, inspiring a network of epidemiologists, social scientists, physicians, civil servants, and surviving temperance groups to converge around the public health model as a legitimate approach to tackling alcohol misuse.

Later in 1984, Robin Room, a sociologist and one of the early contributors to the formation of the public health approach, pointed to Terris's 1967 review article on the epidemiological studies on cirrhosis, explored in the second section of this chapter, as one of the earliest conceptual foundations for the public health approach.[156] In order to argue that 'cirrhosis mortality rates are directly related to per capita consumption of alcohol', Terris foregrounded the example of the Central Control Board's alcohol control policies in Britain

[154] Thom, *Dealing with Drink*, pp. 1-7.
[155] Sully Ledermann, *Alcool, Alcoolisme, Alcoolisation: Données Scientifiques de Caractère Physiologique, Économique et Social* (Paris, 1956).
[156] Room, 'Alcohol Control and Public Health', pp. 2076-88.

during the First World War, which involved 'progressive increases in taxation of alcoholic beverages to decrease their availability' and the 'restriction of the hours of sale of alcoholic beverages.'[157] He added that '[t]he British example demonstrates that governmental fiscal and regulatory measures can be effective in reducing alcohol consumption and lowering mortality from cirrhosis', whereas '[p]rograms for control of cirrhosis of the liver which are limited to health education and treatment of the alcoholic' were seen to be inadequate.[158]

In line with Terris's suggestions, Room described the new public health approach as having three primary features: first, the discursive shift from a focus on 'alcoholism' to 'alcohol-related problems', a category that encompassed a wide variety of mental, physical, and social harms; second, a re-emerging interest in tackling the growing incidence of physiological illnesses of alcohol, such as cirrhosis; and third, a preference for alcohol control policies, 'legal or regulatory measures affecting the production, distribution and sale of alcohol', as effective preventative measures against these 'alcohol-related problems'.[159] Room's acknowledgement of the significance of Terris's article is indicative of how the scientific dispute over the relationship between alcohol and cirrhosis shaped the conceptual formation of the public health model. The indirect impact of alcohol's availability on the incidence of cirrhosis justified their support for alcohol control policies to reduce overall levels of consumption.

The origins of the total consumption model as the bedrock of the public health approach can be attributed to the publication of an influential World Health Organisation report from 1975 titled *Alcohol Control Policies in Public Health Perspective*, posthumously referred to as the 'purple book'.[160] The WHO report was a product of a collaborative interdisciplinary project led by Kettil Bruun, a pioneering alcohol researcher of the Finnish Foundation for Alcohol Studies. The Foundation was a body established in 1950 under the funding of Alko, the national alcohol retail monopoly in Finland, for the promotion of research on alcohol-related problems.[161] Other organisations affiliated with the project included the

[157] Terris, 'Epidemiology of cirrhosis', pp. 2086-7.
[158] *Ibid.*, p. 2087.
[159] Room, 'Alcohol Control and Public Health', pp. 2094-6, 301.
[160] Bruun, et al., *Alcohol Control Policies.*
[161] K. Brofoss, 'Finnish social alcohol research and alcohol policy', *Nordisk Alkoholtidskrift* 11 (1994), pp. 61-2.

WHO Regional Office for Europe and the Addiction Research Foundation of Ontario in Canada, and the report itself was jointly authored by researchers based in Britain, Canada, the United States, Finland, and Norway. Aside from Robin Room himself, at the time a PhD student in sociology at the University of California, Berkeley, the long list of authors included Griffith Edwards, a prominent addiction researcher who played a pivotal role in promoting the public health model in Britain.[162] Rather than being a product of physicians specialising in physiological disorders, the authors of the 'purple book' were composed of sociologists like Bruun, psychiatrists like Edwards, and other experts in statistics and health economics. Paradoxically, however, the report reinstated the notion that alcohol misuse should be approached on account of its wide assortment of harms, including health problems such as liver disease.

The central premise of the WHO report stated that, since 'changes in the overall consumption of alcoholic beverages have a bearing on the health of the people in any society', the 'control of alcohol's availability becomes a public health issue'.[163] The authors believed that 'the long-term effects of alcohol use on health' acted as the primary justifier for the need for such policies.[164] As an example, they pointed to cirrhosis as 'both an index and a serious consequence of heavy alcohol use', and additionally as a 'rapidly increasing' problem in many Western countries that 'has become one of the leading causes of death among middle-aged males.'[165] A variety of statistical studies across Europe indicated the relationship between overall consumption and cirrhosis deaths to be 'remarkably close in nearly all cases'.[166] Cirrhosis deaths dropped when the availability of alcohol was withheld in instances such as the enactment of prohibition in Canada, Finland, and the United States, as well as the severe supply shortage of alcohol in Paris during both world wars.[167] Therefore, 'the rate of death from cirrhosis usually rises and falls with the level of alcohol consumption in general populations'

[162] Bruun, et al., *Alcohol Control Policies*, p. 4.
[163] *Ibid.*, pp. 12-3.
[164] *Ibid.*, p. 15.
[165] *Ibid.*, p. 28.
[166] *Ibid.*, p. 40.
[167] *Ibid.*, pp. 43-4.

regardless of 'cultural differences with respect to drinking pattern, social norms and the like'.[168] Thus, cirrhosis was frequently referred to as a typical 'problem' associated with alcohol misuse, being both statistically measurable and highly responsive to changes in *per capita* levels of consumption within a given period.

The report also justified its use of cirrhosis deaths as a pillar of the total consumption model by describing the disease as a widely accepted outcome of heavy drinking. Describing cirrhosis as 'one of the leading causes of death among heavy drinkers', the authors observed that '[t]he aetiological importance of long-term heavy alcohol intake *per se* would seem to have been established beyond doubt'.[169] 'In addition to a large body of epidemiological and clinical evidence' that included a reference to Lelbach's article from 1974, the report cited Rubin and Lieber's baboon experiment from 1974 in stipulating that 'recent experimental work has convincingly shown that a direct effect of alcohol is mainly responsible rather than a nutritional deficiency.'[170] Furthermore, it pointed to a 1974 paper by Péquignot that estimated that 'an average daily intake of between 40 to 60 grams was potentially cirrhogenic.'[171] While the authors did not entirely discard the role of malnutrition, stating how it 'may well heighten susceptibility to the alcohol effect', this passage demonstrated that Lieber's experiments and Lelbach and Péquignot's dose-response studies played a pivotal role in reconstructing the reputation of cirrhosis as the emblematic disease of alcohol misuse.[172]

The general spirit of the WHO report was carried on in Britain by a set of monographs published by a collection of prestigious professional associations that sided with the public health approach. At the beginning, psychiatrists acted as the harbinger of this movement when the Royal College of Psychiatrists published *Alcohol and Alcoholism* in 1979 under the

[168] *Ibid.*, p. 44.
[169] *Ibid.*, p. 26.
[170] *Ibid.*, p. 26; Werner K. Lelbach, 'Organic pathology related to volume and pattern of alcohol use', *Research Advances in Alcohol and Drug Problems* 1 (1974), pp. 93-198; Rubin, and Lieber, 'Fatty Liver, Alcoholic Hepatitis and Cirrhosis', pp. 128-135.
[171] Bruun, et al., *Alcohol Control Policies*, p. 27; G. Péquignot, 'Les problèmes nutritionnels de la société industrielle', *La Vie Médicale au Canada Français* 3 (1974), pp. 216-25.
[172] Bruun, et al., *Alcohol Control Policies*, p., p. 26.

authorship of a committee headed by Edwards.[173] Much like that of the 1975 WHO report, which actively 'tried as far as possible to be objective' in avoiding the use of a morally laden language, the committee of psychiatrists argued that their report was 'based on the most recent evidence' and dismissed the opinions of those who 'exaggerate the dangers of alcohol and see its use as an unmitigated evil'.[174] In spite of that, it went on to state that '[a]lcohol is also a drug which can miserably wreck or destroy life, and which exacts these costs on a devastating scale.'[175] This passage was intended to cautiously provoke the reader with a judgement that a certain social practice, drinking, was more dangerous than it had been presumed. To tackle the growing incidence of alcohol problems, *Alcohol and Alcoholism* recommended the implementation of higher alcohol taxes and the establishment of a guideline stipulating a safe drinking limit.[176] Concerning the effects of alcohol on the liver, the report explained how an overall increase in alcohol consumption within a population led to a steady increase in mortality from cirrhosis as well as the proportion of the cirrhosis deaths that were directly attributable to alcohol.[177] Seven years later, the College published another report titled *Alcohol: Our Favourite Drug* (1986), which additionally argued that '[a]lmost everyone is aware that excessive drinking can sometimes result in the development of cirrhosis'. Cirrhosis was referenced as a statistically verified disease that had a close, parallel association with *per capita* levels of consumption.[178] Throughout the 1980s, the Royal College of Psychiatrists continued to play an influential role, most notably in 1987 when they advised the Department of Health to stipulate 21 alcoholic units per week for men and 14 units for women as the recommended 'safe' level of consumption.[179]

The Office of Health Economics, a respected consultancy established in 1962 by the Association of the British Pharmaceutical Industry, contributed to the debate with their own

[173] The Royal College of Psychiatrists, *Alcohol and Alcoholism: The Report of a Special Committee of the Royal College of Psychiatrists* (London, 1979).
[174] Bruun, et al., *Alcohol Control Policies*, p. 14; The Royal College of Psychiatrists, *Alcohol and Alcoholism*, p. 2.
[175] The Royal College of Psychiatrists, *Alcohol and Alcoholism*, p. 1.
[176] *Ibid.*, p. 140.
[177] *Ibid.*, pp. 79-80.
[178] The Royal College of Psychiatrists, *Alcohol: Our Favourite Drug* (London, 1986), pp. 92-3.
[179] Yeomans, *Alcohol and Moral Regulation*, p. 220.

publication: *Alcohol: Reducing the Harm* (1981).[180] The book endorsed the total consumption model by arguing that the 'limitation of global consumption by price is the best proven alcohol harm control policy.'[181] Pointing to liver damage as 'the type of physical harm most likely to be thought of as being caused by drinking by members of the public', it understood cirrhosis as 'the most frequently used indicator of alcohol problems in the community', even if 'it plays only a relatively small part in the global burden of alcohol related morbidity and mortality'.[182] Here, cirrhosis was employed as a means to start a discussion on the set of other health problems that result from alcohol misuse. The disease itself was given the prestige of being the central component of the epidemiological knowledge on alcohol and physical harm in pushing for the reduction of overall levels of consumption.

By the late 1980s, the Royal College of Physicians joined the debate when they published *A Great and Growing Evil: The Medical Consequences of Alcohol Abuse* (1987), signalling the growing importance of the wider medical profession within public health coalition.[183] The temperance-tinged title of the report, provocatively labelling the alcohol problem as an 'evil', was indicative of its introductory hook. The authors highlighted the professional body's historical record of accomplishment on the drink question by referencing the instance when the College submitted a report in 1729 to the House of Commons on the Gin Craze. They then added that 'we have once again been led to write a report because of the increasing number of patients we see who are damaged by alcohol', a passage that remarkably equated the present alcohol problem with one of fiercest moral panics in Britain's history with drink.[184] Much like the previous report by the Office of Health Economics, cirrhosis mortality rates were employed as a narrative device to guide the reader towards a discussion on the incidence of other alcohol-related problems such as oesophageal and pancreatic cancer, traffic deaths, assault, and alcohol poisoning.[185] The fourth chapter of the report explored some of the nuances surrounding the

[180] Office of Health Economics, *Alcohol: Reducing the Harm* (Luton, 1981).
[181] *Ibid.*, p. 8.
[182] *Ibid.*, p. 23.
[183] The Royal College of Physicians, *A Great and Growing Evil: The Medical Consequences of Alcohol Abuse* (London, 1987).
[184] *Ibid.*, p. 1.
[185] *Ibid.*, pp. 23-4.

causation of cirrhosis. The authors explained that most 'would accept that both malnutrition and alcohol toxicity play an important part in damaging the liver but would probably disagree about the relative contribution that each of them makes.' Although nutritional deficiency 'may potentiate the effect of alcohol', the fact that an adequate diet 'would not succeed in preventing the development of cirrhosis unless they also reduce their alcohol intake' implied that 'alcohol itself plays a key role in the development of liver injury'.[186] The report also recognised how 'genetic, constitutional, and environmental' factors contribute to one's susceptibility to liver damage, referring to the works of Lelbach and Péquignot.[187] As a product of the authorship of expert physicians, it was hardly surprising that a text published as late as in 1987 delved into the controversy surrounding the aetiology of alcoholic cirrhosis. This indicated the importance of the shifting scientific knowledge on cirrhosis, including that of the experimental and clinical studies on its direct causation, the epidemiological studies on its statistical association with alcohol, and the ongoing investigations on the role of individual susceptibilities, all of which contributed to the formation of the conceptual foundations of the new public health approach.

The medical understandings of alcohol and the liver had a similarly notable impact on the alcoholic beverage industry. For the first time in the twentieth century, the scientific changes concerning the knowledge of cirrhosis aetiology was directly and vividly addressed by the alcohol industry in the 1970s and the 1980s. The Brewers' Society, which had previously focused on large-scale cooperative advertising projects such as the 'beer is best' and the 'good wholesome beer' campaigns, underwent a notable shift in its strategy in constructing a counter-narrative against the public health approach. *Sensible Drinking*, a pamphlet distributed by the Society in 1982, embodied the industry's stance on the issue by advocating educational solutions that encouraged responsible drinking as the optimal solution to the drink question.[188] In opposition to the problematisation of all forms of alcohol consumption, the brewing industry continued to emphasise how alcohol provided 'the most enjoyment and benefit' when

[186] *Ibid.*, pp. 54-5.
[187] *Ibid.*, p. 40.
[188] The Brewers' Society, *Sensible Drinking* (London, 1982).

consumed 'sensibly'.[189] The industry's narrative on the alcohol problem in the late twentieth century thus shared discursive similarities with its previous iterations in their encouragement and accentuation of the culture of moderation.

In 1972, the Society established its own medical advisory committee of physicians to protect the industry's interests against potential problems that it faced from professional medicine. A memorandum recommending the establishment of the committee stipulated the need for the Society to use 'accepted published work' to circumvent the dissemination of what they deemed as fallacious, scientifically unsubstantiated understandings of medical problems associated with beer consumption. The committee was additionally relied on to advise the industry on 'the elimination or containment of the problem if a rebuttal is not possible, all done with the absolute minimum of publicity.'[190] As the representative body of an industry that retails the most widely consumed alcoholic beverage in Britain, the Brewers' Society will be examined as a case study to understand the industry's reaction to the public health model. Unlike the tenuous collection of internal documents of groups like the Scotch Whisky Association and the Wine and Spirit Association, much of which are scattered throughout regional archives in Britain, a centralised collection of minutes of confidential meetings within the Society can be found in the Modern Records Centre at the University of Warwick.

The shifting focus from 'treatment' to 'prevention' under the public health approach was seen as a legitimate hazard to the interests of the brewing industry. Although the national consumption of beer experienced a long-term resurgence after the 1960s, the global recession between 1979 and 1983 forced sales to drop by 12 per cent.[191] Thus, a concerted legislative campaign that called for further restrictions on the availability of alcohol was seen as a threat to the industry's profit margins. Corporate members of the Brewers' Society correctly attributed this effort to the popularisation of Ledermann's total consumption model. The first

[189] *Ibid.*, p. 2.

[190] Brewers' Society Collection, MSS.420/BS/4/41/2, 'Memorandum from rear-admiral C. D. Madden to the Survey Committee: The need for a medical advisory group for the brewing industry in the United Kingdom', January 1972.

[191] Brewers' Society, MSS.420/BS/6/3/9, 'Brewers' Society News Release, '"You should've been in the pub last night"', 24 March 1983.

meeting that took place in May 1979 on 'the control theory of alcoholism' discussed potential 'courses open to the drinks industries to defend themselves against this threat', one of which involved a strategy to prove 'that its fundamental assumptions are incorrect, that a rise in *per capita* consumption of alcohol does not necessarily mean an increase in heavy drinkers'.[192] In a later meeting, Derrick Holden-Brown, chairman of Allied Lyons breweries, called for the Society to come up with 'solutions' to the alcohol problem 'which would not harm the industry'. Holden-Brown was convinced that legislation seeking to increase the price of alcoholic beverages 'would not provide remedies' since 'there was no doubt that the moderate use of alcohol was not only not harmful, but beneficial'.[193] Ledermann's total consumption model was understood to be 'oversimplified and misused', as it would be 'pure superstition to assume that people *en masse* behave in a manner which fits a precise mathematical pattern' in which 'the complex problem of alcohol misuse can be solved simply by pricing alcohol beyond the means of the majority'.[194] The 1975 WHO report was also scathingly critiqued by a member of the Society who attacked the belief that 'changes in *per capita* consumption produce changes in the proportion of excessive consumers' was 'both unrealistic and illogical'.[195]

In addition to their attempts to denounce the total consumption model, members of the Society appealed to Britain's exceptionalism in its national drinking culture. At a Society luncheon in October 1979, it was agreed that the conclusions reached by Ledermann's studies in France did not apply to Britain since the two nations had completely different drinking cultures and habits. The argument was oblivious to the results obtained from studies that took place in other parts of the Western world in which similar statistical correlations were noted between the *per capita* level of consumption and the prevalence of problem drinkers.[196] This line of critique was taken up by Holden-Brown, who remarked on how Britain's low *per capita* levels of consumption relative to the rest of Europe proved that '[t]he British do not drink in

[192] Brewer's Society, MSS.420/BS/4/61/14, 'The Control Theory of Alcoholism', 11 May 1979.
[193] Brewers' Society, MSS.420/BS/4/61/14, 'Notes on meeting at the Brewers' Society on Wednesday, 11th July to discuss social and health related aspects of alcohol consumption', 1979.
[194] Brewers' Society, MSS.420/BS/4/61/14, G. Winstanley, 'Notes for Chairman: Lederman', 1 October 1979.
[195] Brewers' Society, MSS.420/BS/4/61/14, John C. Duffy, 'Notes on the "Ledermann Equation" and the "distribution of consumption" theories of alcohol consumption', 3 October 1979.
[196] Brewers' Society, MSS.420/BS/4/61/14, 'notes of a luncheon meeting at 30 Portland Place, W.I.', 8 October 1979.

an extreme way'. In doing so, he argued that brewers were responsible 'to combat these problems and at the same time to protect the harmless pleasure experienced by the vast majority of regular drinkers.' In addition to being convinced of the health-giving properties of the moderate consumption of beer, '[w]e in the UK are perhaps particularly entitled to be proud of the fact that the character and traditions of the pub are powerful reasons why Britain has less abuse of alcohol than many other countries.' Thus, Holden-Brown suggested educational measures as the most favourable solution to alcohol misuse, arguing that it was more compatible with how drinking was predominantly practiced as a respectable social activity in Britain.[197]

A later meeting on 8 July 1980 discussed the next possible course of action for the Society to tackle problem drinking under their own terms. In considering Holden-Brown's suggestion, the members of the committee agreed that the Society should prioritise a portion of its funds to promoting alcohol education. This was in spite of their surprising admission that 'it is doubtful whether education *en masse* or individually is particularly effective in preventing alcohol abuse', showing that their preference for educational solutions was not driven by humanitarian motivations. The funding of alcohol education was understood to allow the brewing industry to be 'seen by the government and the general public to be making a genuine and effective effort' in providing solutions to the alcohol problem. This was part of a strategy whereby the Society 'should seek to influence the package sooner rather than later' by pushing for solutions that went short of harming their own financial gain.[198] The outcome of this committee decision was outlined in a pamphlet published two years later, according to which a large proportion of the Society's budget was dedicated to promoting 'sensible' attitudes to drinking through a myriad of educational schemes, including the production of posters and pamphlets, the development of school curriculums, public campaigns, and professional

[197] Brewers' Society, MSS.420/BS/4/61/14, Derrick Holden-Brown, 'Social Problems - The UK position', 5 October 1979.
[198] Brewers' Society, MSS.420/BS/4/62/1, 'R. Pryor, to Social Problems of Alcohol Committee of Brewers' Society, "Policy on Alcohol Problems and Employment"', 8 July 1980.

support for alcoholism counselling.[199] The enthusiasm over alcohol education thus formed a key component of the brewers' campaign to promote their own image as a socially responsible industry.[200] The explicit way in which the members of the Society discussed their strategy in opposing the threat of the public health model was indicative of how the industry, in all of its newfound interest in alcohol-related problems and the knowledge surrounding it, was ultimately concerned about their bottom line. The promotion of educational solutions was also seen as a useful tactic to contest the public health narrative that called for stringent regulations on the sale of alcohol.

The minutes of the medical advisory committee of the Society also revealed that the industry was interested in the implications of the shift towards the recognition of alcohol as a direct cause of cirrhosis. In a confidential meeting that took place on 13 January 1975, the committee briefly discussed a paper by Lieber titled 'hepatic and metabolic effects of alcohol' (1966).[201] Theo Crawford, a pathologist and a committee member, noted that Lieber's article 'disposed of the idea that malnutrition resulting from high alcohol intake was the sole source of pathogenesis of liver injury', further acknowledging that 'alcohol itself was incriminated' in causing cirrhosis.[202] Even though the article in question failed to specifically state this, that the committee took notice of Lieber in 1975, a year after his seminal baboon studies, indicated that they were reflecting on the demise of the NDT and the confirmation of alcohol's direct causation of cirrhosis. Following a later committee discussion over a *British Medical Journal* column titled 'how does alcohol damage your liver?' (1978), members additionally noted that '[i]t was usually accepted that damage to the liver was the direct toxic effect of alcohol or its metabolites'.[203]

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[199] The Brewers' Society, *Action Against Alcohol Abuse: A Guide to Projects Funded by the Brewing Industry* (London, 1982), pp.16-7, 36-8.

[200] Baggott, *Alcohol, Politics and Social Policy*, pp. 61-2.

[201] Charles S. Lieber, 'Hepatic and metabolic effects of alcohol', *Gastroenterology* 50.1 (1966), pp. 119-31.

[202] Brewers' Society Collection, MSS.420/BS/4/41/3, 'Medical Advisory Committee private and confidential meeting, minutes', 13 January 1975.

[203] Brewers' Society Collection, MSS.420/BS/4/41/5, 'Minutes, 29 January 1979, Medical Advisory Group', 29 January 1979; Anon., 'How does alcohol damage the liver?', pp. 1733-4.

The existence of overwhelming evidence implied that the committee of industry-representing physicians had no choice but to accept the idea that serious liver damage can be caused by the direct toxic action of alcohol. Much like those who refused to acknowledge alcohol as a direct hepatotoxin prior to Lieber's baboon experiments, physicians working for the Brewers' Society followed the criteria that disease causation had to be demonstrated in a laboratory setting on experimental animals for it to be credible, even if the association between the two had already been established. There were observable similarities with the case of the American tobacco industry's reaction to Doll and Hill's epidemiological studies on cigarette smoking and lung cancer in the 1950s, when equivalent trade groups such as the Tobacco Institute insisted that a statistical association between the two, no matter how robust, does not demonstrate causality.[204] However, unlike the surprising revelation of a link between tobacco and lung cancer, the aetiological relation between alcohol and liver disease possessed the advantage of not being an entirely new piece of knowledge abruptly introduced to the public without any pre-existing historical foundations, especially now that scientists had experimentally validated the causal relationship.

Following the recognition of the direct toxicity of alcohol, the brewing industry attempted to directly influence the knowledge on liver disease. Initially, the Society questioned the degree to which their own products were implicated in liver damage. While Crawford reiterated that that 'there was no doubt that excessive drinking caused ill-health, and cirrhosis of the liver was an accurate indicator of the extent of the problem in the country', he exonerated the brewing industry of its responsibility of worsening the incidence of cirrhosis by suggesting that British beer was generally weaker in alcoholic strength than those found in the Continental Europe, and that national beer consumption engulfed other stronger beverages like wine and spirits.[205] In a separate meeting on 22 October 1975, the committee highlighted other factors

[204] Talley, Kushner, and Sterk, 'Lung Cancer', p. 370.
[205] Brewers' Society Collection, MSS.420/BS/4/41/3, 'Minutes, 14 July 1975 of Medical Advisory Committee', 14 July 1975.

such as viral hepatitis as confirmed aetiologies of cirrhosis to discredit the use of cirrhosis mortality rates to estimate the prevalence of alcohol misuse.[206]

On 22 April 1977, the medical advisory committees of the various Anglophone national brewing associations from Britain, Australia, Canada, and the United States convened to discuss the implications of the most recent developments in medical knowledge. This was the first meeting involving the Brewers' Society where members suggested that the industry should invite experts on the liver to give talks on its relationship to alcohol consumption. The list of potential candidates for the event included figures such as Lelbach and Sherlock, with some members pointing out that a focus on the epidemiology of cirrhosis should 'provide useful clues as to factors enhancing the effect of alcohol'.[207] Later that year, the committee discussed a paper authored by Sherlock that argued that the incidence of chronic liver disease was much higher among women than in men.[208] The Society's interest in the multifactorial nature of cirrhosis embodied attempts by the industry to downplay the extent to which alcohol was responsible for causing its most frequently recognised chronic disease. By highlighting individual susceptibilities and other allied factors that contribute to its pathogenesis, it allowed the industry to argue that their products alone could not have caused the disease without the aid of these factors.

The medical advisory committees of the four national brewing industries converged again in Toronto on 4 October 1978.[209] The delegates of the conference asked Harvey Brenner, a physician based in Johns Hopkins Hospital, to speak on 'alcoholic beverage differences and their related health effects'. Brenner was invited because his research showed 'a positive

[206] Brewers' Society Collection, MSS.420/BS/4/41/3, 'Minutes, 22 October 1975 of Medical Advisory Committee', 22 October 1975.
[207] Brewers' Society Collection, MSS.420/BS/4/41/4, 'International Medical Group (US Brewers Association, Brewers' Association of Canada, Australian Associated Brewers, Brewers' Society) Notes of a meeting held on Friday 22 April 1977 in Brewers' Society', 22 April 1977.
[208] Brewers' Society Collection, MSS.420/BS/4/41/4, 'Minutes, 18 July 1977 of Medical Advisory Committee', 18 July 1977.
[209] Brewers' Society Collection, MSS.420/BS/4/41/4,'International Medical Advisory Conference, Toronto, attended by 4 great national medical advisory groups for brewers', 4-6 October 1978.

association [of cirrhosis] with wines and spirits but not beer'.[210] His epidemiological study demonstrated that cirrhosis mortality rates were visibly lower among 'beer drinking nations' in Northern Europe than those that predominantly consumed both beer and spirits, while the 'wine drinking nations' of Southern Europe had by far the highest numbers. He went as far to suggest that 'beer could inhibit cirrhosis because of the large fluid intake', which the committee members deemed 'not [an] irrational supposition' since 'when consumed it left little room for the ingestion of large quantities of other forms of alcohol'.[211] Based on the account found in the detailed minutes of the conference, Brenner's talk failed to consider how his typology of European nations to their beverage of choice fared in comparison to a ranking of counties based on the actual *per capita* level of consumption of pure alcohol. This should have been deemed a necessity since it had been amply demonstrated that the *per capita* level of consumption was precisely what tied cirrhosis deaths, as well as other statistically measurable problems of alcohol misuse, to the restriction of the supply of alcohol. Nonetheless, the conclusions of the talk were welcomed by the brewers as further confirmation that the brewing industry should not be indicted for the worst excesses of alcoholic liver disease.

The medical advisory committee's interest in the individual susceptibilities to alcoholic cirrhosis reached such a degree that they even took a keen interest in funding the studies on gender and genetic predispositions conducted by Roger Williams. Indeed, two of Williams's own publications from the early 1980s acknowledged the financial support of both the Department of Health and Social Security and the Brewers' Society.[212] A set of minutes from June 1979 provide an account of how the collaboration started with Holden-Brown's personal interactions with Williams. On the several occasions that they met, Holden-Brown expressed how he 'had been impressed by' Williams and his 'well-balanced attitude to drink'. The brewer

[210] Brewers' Society Collection, MSS.420/BS/4/41/4, ''Preliminary meeting of the Medical Advisory Groups of the Brewers' Associations of Australia, Canada, United Kingdom, and the United States of America', 4 April 1978.
[211] Brewers' Society Collection, MSS.420/BS/4/41/4,'International Medical Advisory Conference, Toronto, attended by 4 great national medical advisory groups for brewers', 4-6 October 1978.
[212] National Archives: Department of Health files, JA 367/22, 'Study of factors determining pattern of alcoholic liver disease: Dr R Williams', 1 January 1977-31 December 1981; Saunders, Davis, and Williams, 'Do women develop alcoholic liver disease', p. 1143; Saunders, Wodak, Haines, Powell-Jackson, Portmann, Davis, and Williams, 'Accelerated development of alcoholic cirrhosis', p. 1384.

additionally pointed to how the physician came off as being 'very presentable' in a televised interview.[213] The Society transcribed the interview in question, which impressed the members of the committee with the 'neutral' language that Williams used when discussing growth of the alcohol problem in Britain. In it, Williams warned that the 'amount of alcohol taken on average by the populace is steadily increasing each year, and with this there is a parallel increase in the amount of liver associated diseases.' He admitted that '[t]he problem is that the amount that a patient can safely drink, a person can safely drink, varies from person to person' and that he and his team were 'doing research into what determines safe limits for individuals, because everybody likes… to drink, and they want to be able to drink safely and wisely."[214] The medical advisory committee's enthusiasm over the researched carried out at the Liver Unit was founded on its capacity 'to develop means of measuring the extent of alcohol consumption for individuals before harm was caused to the liver', potentially contributing to the creation of a parameter whereby consumption could safely be encouraged before it posed a health risk. Additionally, funding such a project 'could bring substantial benefits to the industry, such as useful publicity, helpful facts and the support of a prominent doctor' whose 'attitude was favourable towards the Industry and it might at some stage be appropriate to invite him to join the Society's Medical Advisory Group'.[215]

Aside from the prestige gained from making a financial contribution to legitimate lines of scientific inquiry into the harms of alcohol, the primary motivations that drove the Society to fund the Liver Unit's research was later clarified in an account of an international meeting of the four Anglophone national brewing associations in October 1979. Hedley Atkins, a prominent London-based surgeon and a member of the committee, articulated on the implications of garnering a more detailed knowledge of what makes one susceptible to developing alcoholic cirrhosis.

[213] Brewers' Society Collection, MSS.420/BS/4/41/5, 'Extract from survey minutes of meeting', 13 June 1979.
[214] The interview in question: Brewers' Society Collection, MSS.420/BS/4/41/5, 'New Liver Disease Unit', 13 June 1979.
[215] Brewers' Society Collection, MSS.420/BS/4/41/5, 'Minutes on the Social Problems of Alcohol by Executive Committee', 13 June 1979.

> With such techniques it should be possible to evolve procedures for determining what is safe for an individual to drink without developing hepatic damage, and to set limits to consumption. It may be possible to separate patients with hepatitis or cirrhosis into a 'high-risk' group who all have HLA-B8 and develop severe liver disease with low alcohol consumption, and a 'low-risk' group in which the effect does not occur. Such an analysis may lead to a clearer estimate of how much alcohol is safe for an individual to take without the risk of liver damage.[216]

Hedley added that '[i]n addition to improving our understanding of the mechanism of liver injury, it is hoped that this approach would form the basis for a blood test to screen patients for immunologically mediated liver injury.'[217] An innovation in the identification of specific genes, pointing to HLA-B8 in this instance, allowed for medicine to isolate and target individuals that were more susceptible to cirrhosis while exonerating the rest of the population to continue drinking on a regular basis without fear of developing severe liver damage. The brewers thus believed that Williams provided the opportunity to intellectually discredit the drive to reduce all forms of consumption by scientifically demonstrating that only a specific, easily identifiable group were at risk of cirrhosis, while promoting the understanding that the majority of the drinking population had little chance of developing the most prevalent, infamous killer among the diseases of drink.

The Liver Unit's work was the largest beneficiary among all projects supported by the Brewers' Society at the time, receiving a total of £107,000 between 1980 and 1983.[218] A confidential medical advisory committee meeting on 18 July 1983 admitted that Williams received a 'disproportionate share of the research funds available', which, in its first year, constituted 32 per cent of the budget that the Society appropriated to funding research on

[216] Brewers' Society Collection, MSS.420/BS/4/41/5, 'Professor Sir Hedley Atkins' Introduction to Crystal Ball Session International Medical Advisory Groups' Conference', October 1979.
[217] *Ibid.*.
[218] The Brewers' Society, *Action Against Alcohol Abuse*, pp. 8-9.

alcohol-related problems.[219] Adding to how the members of the Society explicitly touted his work as a potentially beneficial investment, these numbers show that the industry was keenly interested in highlighting the multifactorial nature of alcoholic cirrhosis to counter the total consumption model.

The use of science funding by the beverage industry in its public relations battle against public health campaigners has largely been neglected by most of the historical accounts on the period.[220] The nature of this particular case of industry-sponsored medical research can be understood more effectively when juxtaposed to the historiography of the controversy over smoking and lung cancer during the 1950s and 60s. Against the tendency by older accounts to portray the tobacco industry as a 'villain', more recent accounts by Talley et al and Berridge have argued that the reality was far more complicated.[221] The tobacco industry provided reasonable objections towards the policy implications of Doll and Hill's studies by stating that an association between a factor and an illness, no matter how close, did not prove causation.[222] Indeed, the internal documents of the medical advisory committee of the Brewers' Society show that they were not interested in fabricating scientific falsehoods on alcohol-related harm. The Society sponsored legitimate lines of scientific inquiry into alcohol and liver disease that were carried out by Williams, who rightfully looked into how the aetiology of cirrhosis could not be solely attributed to alcohol itself. Although highlighting the importance of factors other than alcohol allowed the industry to downplay the extent to which their products were responsible for the disease, they nonetheless contributed to the expansion of the knowledge on the specific endogenous factors that made individuals more susceptible to the disease.

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[219] Brewers' Society, MSS.420/BS/4/61/14, 'Letter from Director General of Brewers' Society to Chairman and Vice chairman on "social and health related aspects of alcohol consumption", 6 July 1979; Brewers' Society Collection, MSS.420/BS/4/41/6, 'Medical Advisory Group meeting confidential minutes', 18 July 1983; National Archives: Department of Health files, JA 367/23, 'Progress report - Inter-relationship between socio-economic and genetic factors in determining the pattern of alcoholic liver disease and response to management', November 1978-December 1981.

[220] The exception would be Baggott, *Alcohol, Politics and Social Policy*, pp. 61-2, which very briefly touched on the Brewers' Society's funding of research on alcohol problems.

[221] Talley, Kushner, and Sterk, 'Lung Cancer'; Virginia Berridge, *Marketing Health: Smoking and the Discourse of Public Health in Britain, 1945-2000* (Oxford, 2007), pp. 4-5; 22.

[222] Talley, Kushner, and Sterk, 'Lung Cancer', pp. 368-70.

The confrontation between the public health coalition and the beverage industry had a peculiar effect on policymakers in Westminster and Whitehall, where the impact of the call to strengthen alcohol controls proved to be limited. Historians have pointed out that policymakers during the late-1970s and the 1980s were generally reluctant to raise duties on beverages to restrict the availability of drink.[223] Although the period saw heightened panics over alcohol's culpability in the 'epidemic' of football hooliganism and other forms of anti-social behaviour, the drinks lobby largely overpowered the demand to address the growing incidence of alcohol-related health problems.[224] The Brewers' Society compiled some revealing survey results. A Society-commissioned Market & Opinion Research International (MORI) poll conducted from 1978 to 1981 showed that 66 per cent of the general public was unconvinced that 'drunkenness' was an urgent issue, while only 11 per cent saw it as a 'serious problem'. Similar results were shown in a separate 1982 MORI study on opposition MPs in the House of Commons, 2 per cent of whom considered 'alcohol problems' to be a 'serious question' facing the country as opposed to unemployment (77 per cent), inflation (41 per cent), industrial relations (35 per cent), and so on.[225]

Although the outgoing Labour government commissioned a Department of Health report on the growing problem of alcohol misuse, its publication was controversially sabotaged by the new Conservative government of the 1980s, which was disposed to continue the path of liberalising the licensing regime dating back to the First World War.[226] Andrew Gamble's characterisation of Thatcherism as a paradoxical idealisation of the 'free economy and a strong state' aptly describes how the government's conservative 'tough on crime' approach towards drunkenness and public order coexisted alongside the neoliberal unwillingness to implement control legislation that potentially harmed to the beverage industry.[227] At the beginning, the newly elected government hinted at an interest in tackling the growing incidence of alcohol

[223] Nicholls, *The Politics of Alcohol*, pp. 207-8; Greenaway, *Drink and British Politics*, p. 175; Jennings, *A History of Drink*, p. 201.
[224] Yeomans, *Alcohol and Moral Regulation*, p. 167; Jennings, *A History of Drink*, p. 170.
[225] Brewers' Society, MSS.420/BS/4/61/22, 'Annexure results of surveys by MORI', 1982.
[226] Virginia Berridge, and Betsy Thom, 'Research and policy: what determines the relationship?', *Policy Studies* 17.1 (1996), p. 29.
[227] Andrew Gamble, *The Free Economy and the Strong State: The Politics of Thatcherism* (Basingstoke, 1988).

misuse. Immediately following the Tory landslide in the general election of 1979, George Young, an MP who served as the parliamentary Undersecretary of State to the Department of Health and Social Security in the Conservative government, delivered a speech to the National Council on Alcoholism that embodied the spirit of the public health approach. He argued for the 'vital role of prevention… to keep the fleet of social drinkers out of the minefield of alcohol misuse', calling for 'the adjustment of a nation's lifestyle to improve its health' through the use of fiscal measures to reduce the availability of alcohol.[228] Young's trailblazing speech, however, was short-lived in its impact. The Conservative Party's official stance in the alcohol debate was manifested in a later 1981 report, *Drinking Sensibly: Prevention and Health*.[229] The title of the report suggested that the government followed the narrative favourable to the beverage industry by endorsing education and self-control as the solution to the alcohol problem. Similar to the earliest public health initiatives to tackle smoking, the government's stance embodied what Berridge refers to as 'systematic gradualism', an industry-friendly harm reduction strategy that had a strong preference for health education over state control of individual consumption.[230] Based on the assumption at the time that most people drank responsibly and that individual drinkers were capable of making rational choices over their drinking habits, *Drinking Sensibly* set out that '[t]he aim of this booklet is not to stop people drinking but to encourage sensible attitudes towards the use of alcohol'.[231]

The changing medical understandings of alcohol and cirrhosis was additionally referred to by policymakers. *Drinking Sensibly* talked of cirrhosis as 'probably the best known long-term physical consequence of excessive drinking… a progressive and potentially lethal disease in which damaged cells are replaced by fibrous tissues'.[232] The mention of 'cirrhosis' noticeably increased during the period within the parliamentary debate. The disease was referred to at a total of 91 times in the 1980s, double that of the 43 times during the 1970s, in a reflection of the

[228] Brewers' Society, MSS.420/BS/4/61/14, 'Alcohol Misuse: Growing pressure for government action, says Sir George Young', report by Department of Health and Social Security, 10 July 1979.
[229] Department of Health and Social Security, *Drinking Sensibly: Prevention and Health* (London, 1981).
[230] Berridge, *Marketing Health*, p. 81.
[231] *Ibid.*, p. 7.
[232] *Ibid.*, p. 13.

growing political interest surrounding alcohol-related issues. Cirrhosis mortality rates were also frequently mentioned as a reliable indicator of the extent of the alcohol problem in Britain.[233] A 1987 debate in the House of Lords referred to the 'damage to the liver' as '[t]he most obvious effect' of alcohol abuse.[234] Evidently, alcohol's causative relation with cirrhosis was taken for granted, even if the drive to tackle alcohol misuse as a public health issue had a negligible impact in national politics at the time.

This section explored the impact of the shifting medical understandings on alcohol and the liver in directly shaping the wider public discourse surrounding consumption and harm in Britain in the 1970s and 80s. Renewed concerns over the rising incidence of alcohol misuse in Britain was spearheaded by a new professional coalition that rallied under the public health model, targeting the reduction of overall levels of consumption by pushing for more stringent controls on the availability of alcoholic beverages. The beverage industry, led by the Brewers' Society, reacted to these developments by pushing for more education-based solutions to the alcohol problem through the continued encouragement of moderation. Alcoholic liver disease was at the heart of this confrontation. The establishment of direct causation and association between alcohol and cirrhosis formed the conceptual foundation of the total consumption model, whereby the knowledge on the statistical occurrence of the disease was set out as a starting point for a further discussion into other alcohol-related harms that grew in incidence alongside the rise of *per capita* levels of consumption. In turn, the brewing industry saw the growing scientific interest in the various contributory factors to the pathogenesis of cirrhosis as an opportunity to downplay the hepatotoxic culpability of alcohol while welcoming the prospect of isolating consumers who were at a greater risk to tailor consumption models at the individual level. In essence, both sides of the alcohol debate chose to underscore different components of the MDTT to suit their own ends.

[233] HC Deb 25 May 1971, vol 818, col 47-9W; HC Deb 27 February 1976, vol 906, col 838; HL Deb 15 March 1979, vol 399, col 850-1WA; HC Deb 31 October 1979, vol 972, col 558-9W; HC Deb 12 November 1979, vol 973, col 472-3W.
[234] HL Deb 25 November 1987, vol 490, col 647-80.

Conclusion

The 1970s witnessed the re-establishment of alcohol as the agent understood to be the direct cause of cirrhosis. Nutritional deficiency was replaced by the direct toxic action of alcohol as the primary aetiology of the disease following the successful experimental production of liver damage through the combined use of alcohol and a nutritionally adequate diet. While alcohol was firmly established as a legitimate aetiology to cirrhosis, continued uncertainties over the statistical rarity of the disease among heavy drinkers inspired physicians like Williams to study the individual susceptibilities that predisposed drinkers to serious liver damage. Thus, alcoholic cirrhosis was increasingly understood as a multifactorial disease under the MDTT. These radical shifts in the expert medical knowledge on alcohol and the liver proved to be influential in both the wider medical profession and the alcohol debate in Britain. Cirrhosis, with its restored reputation as an archetypal disease of the heavy drinker, was central to the formation of the language surrounding alcohol misuse after the 1970s within both the public health coalition and the alcohol beverage industry.

Conclusion

In the early 2000s, alcohol and the liver came under the national spotlight amid the media outcry surrounding the last remaining years of Northern Irish football legend George Best. Best was diagnosed with severe liver damage after struggling with alcohol dependence for most of his adult life, placing him in a position to receive a liver transplant in 2002 under the supervision of Roger Williams. Controversy erupted when Best started drinking again after the operation, having faced multiple charges for drunken assault and drink driving in 2003. On 25 November 2005, he died after his liver failed to protect him from lung infection and multiple organ failure.[1] Aside from the widespread outpouring of grief for the passing of a footballer whom many deemed to be one of the greatest of the twentieth century, Best's legacy was attacked by those who pointed to his alcoholism and to allegations of domestic violence by his second wife. Many struggled to sympathise with a public figure who, even after being given a second chance to live, continued to drink excessively.[2] Nigel Heaton, the surgeon who performed Best's liver transplant, argued on reflection that patients who have a history of abusing alcohol should not be considered for a liver transplantation due to the short supply of liver donors.[3] Best's inability to stop drinking was thus seen by segments of the public as a failure of individual will. Although some of the criticisms surrounding his life were justified, rarely before has there been an instance in which alcoholic liver disease was moralised to such a degree.

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[1] Alan Bairner, "'In balance with this life, this death': mourning George Best', *International Review of Modern Sociology* 32.2 (2006), p. 298.

[2] *Ibid.*, pp. 300-1.

[3] Ian Sample, 'Alcohol abusers should not get transplants, says Best surgeon', *The Guardian Online*, 5 October 2005, https://www.theguardian.com/science/2005/oct/05/drugsandalcohol.medicineandhealth [accessed 6 September 2018].

Indeed, the controversy surrounding the death of the footballer was in many ways a partial symptom of the further escalation of the alcohol debate in the last couple of decades. In the closing years of the twentieth century, overall *per capita* levels of alcohol consumption increased to levels unseen since the Edwardian period.[4] The new millennium saw the emergence of a widespread concern surrounding the perceived epidemic of 'binge' drinking in town centres across the country.[5] Public health campaigners have also began to push for a minimum price to be imposed on every unit of alcohol to drive down the affordability of cheap beverages.[6] Citing various studies that suggested a stronger link between alcohol consumption and the risk of developing various cancers, Sally Davies, the chief medical officer of health for England, declared in 2016 that '[d]rinking *any level* of alcohol regularly carries a health risk for anyone'.[7] This potentially signalled a return to recommendations of total abstinence in the problematising discourses on alcohol.

In the midst of such developments, Berridge perceptively noted a trend where liver specialists have begun to play an increasingly prominent role within the public health coalition.[8] Alcohol Health Alliance UK, an umbrella organisation of over 50 charities, think tanks, and professional medical bodies united in their support for tougher regulations on alcohol pricing and licensing, was founded in 2007 by two prominent hepatologists, Ian Gilmore and Nick Sheron.[9] In spite of his previous ties to the alcoholic beverage industry in the 1980s, Williams has also become one of the leading voices within the alcohol policy community. As the man in charge of *The Lancet* standing commission into liver disease, Williams recommends the introduction of minimum unit pricing on alcohol to counteract the growing incidence of liver disease.[10] Although public health campaigners has had limited success in

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[4] Mark Tettenborn, *British Beer & Pub Association (BBPA) Statistical Handbook 2015* (London, 2016), p. 29.
[5] Nicholls, *The Politics of Alcohol*, pp. 223-48.
[6] In May 2018, Scotland became the first country in the United Kingdom to implement minimum unit pricing (MUP).
[7] Department of Health, 'New alcohol guidelines show increased risk of cancer' (8 January 2016), https://www.gov.uk/government/news/new-alcohol-guidelines-show-increased-risk-of-cancer [access- ed 5 September 2018].
[8] Berridge, *Demons*, p. 226.
[9] Alcohol Health Alliance UK, 'About the Alcohol Health Alliance' (2015), http://ahauk.org/about/ [accessed 5 September 2018]. Gilmore currently chairs the organisation.
[10] Roger Williams, et al., 'Addressing liver disease in the UK', pp. 1953-4.

counteracting the noticeable rise in alcohol consumption and cirrhosis deaths in the past few decades, one could reasonably argue that liver disease, alongside other chronic illnesses, has become much more integral to the discussions surrounding the alcohol problem in Britain.

In many respects, the continued significance of the liver within the present alcohol debate cannot be fully appreciated without looking at the historical backdrop. At the beginning of the twentieth century, cirrhosis was widely acknowledged by medical professionals as a direct outcome of alcohol use, an understanding that was exploited by the temperance movement to overstate the harmful qualities of drink. The perceived success of the Central Control Board in tackling drunkenness during the First World War instigated a major shift with the emergence of New Moderationism. The new approach, which called for the promotion of moderate drinking in the place of the temperance movement's call for abstinence and prohibition, was heavily informed by how the toxic action of alcohol on the liver was gradually downplayed in the medical literature. By the middle of the twentieth century, alcohol was all but abandoned as a direct cause of cirrhosis upon the emergence of the nutritional deficiency theory. The re-conceptualisation of cirrhosis as primarily a nutritional disease occurred alongside the relative pacification of the politics of alcohol in postwar Britain, a period that saw minimal confrontations over the drink question. Eventually, however, a new set of clinical and experimental studies in the 1960s and 70s successfully demonstrated alcohol to be a direct toxin to the liver. As a result, liver disease and its established association to alcohol came to play a major role in the renewed public concerns over alcohol misuse in the late twentieth century.

Thus, in describing the history of cirrhosis, an illness that has been ubiquitously tied to alcohol consumption, this thesis has shown that its most troubled career took place in the twentieth century, a distinctly volatile period when the presumed causation of alcohol in damaging the liver was vigorously interrogated. Looking at how cirrhosis had been associated with a multiplicity of potential causes throughout the century, theories into its aetiology were conceived according to the existing criteria of scientific evidence. Medical professionals questioned the direct toxicity of alcohol on the liver on the grounds that the causality was yet to be established experimentally. Furthermore, the fact that cirrhosis developed in no more than a minority of heavy drinkers implied that the causality was not at straightforward as some

had portrayed. Therefore, the collective decision to reject the causation of alcohol in favour of malnutrition in the middle of the century was an instance in which the 'wrong' conclusion was reached for the right reasons. Instead of being predominantly shaped by non-scientific social and cultural factors, medical scientists generally produced reasonable approximations of the aetiology of cirrhosis that took into account of the available information.

This thesis has additionally stressed the historical significance of medicine in shaping the alcohol debate in Britain. Expert knowledge on alcohol and its relation to illnesses such as cirrhosis played an integral role in how alcohol consumption was conceptualised in relation to harm within the public discourse. The framing of cirrhosis as an 'alcoholic's disease' proved to be useful at separate points in the twentieth century in highlighting the damage that alcohol was capable of inflicting on the individual. On the opposite end of the spectrum, the scientific minimisation of alcohol's responsibility in causing the disease contributed to wider efforts to downplay the substance's harmful properties. Hence, the degree to which drink was seen as a problem throughout the century was determined to an extent by the changing understandings of alcohol's relative culpability in damaging the liver. In approaching the history of alcohol, its medical and scientific dimensions should thus be regarded as being equally as significant as its social and cultural aspects.

Appendix

Textbooks of General Medicine

The following table outlines the long-term shifts in the aetiological knowledge of cirrhosis across multiple editions of textbooks of general medicine, spanning from 1900 to 1990. Each column contains the edition number, the year of publication, and an abbreviated quote outlining the publication's particular stance on what causes cirrhosis.

	1900~05	1906~10	1911~15	1916~20	1921~25	1926~30
James M. Anders, *A Text-book of the Practice of Medicine*	4th edition (1900) 'although the quantity necessary to produce the disease varies greatly in different individuals...by the side of alcoholism all other causes combined are comparatively insignificant.'	8th (1908) identical claim to 4th edn	11th (1913) identical claim to 4th edn		14th (1922) Alcoholism is a 'causative factor operative in nearly all cases' but the 'influence of alcohol is undoubtedly exaggerated. Experimentally it is impossible to reproduce the picture of cirrhosis by feeding animals with alcohol in large amounts over long periods of time.'	
William Osler, *The Principles and Practice of Medicine*	4th (1901) 'Alcohol is the chief cause of cirrhosis... [o]ther poisons... play a minor role'		8th (1912) Alcohol 'produces definite changes in the liver' with '[d]egenerative changes in the liver cells	9th (1920) Cirrhosis due to 'toxic action of alcohol'		12th (1930) identical claim to 9th edn
Frederick Taylor, *A Manual of the Practice of Medicine*	6th (1901) 'great majority of cases the cause of cirrhosis' is the '[e]xcessive use of alcohol', '[b]ut the simple theory of direct irritation by alcohol has not escaped criticism' and '[w]here alcoholic excess cannot be proved, a satisfactory explanation is rarely forthcoming.'	8th (1908) identical claim to 6th edn	9th (1911) identical claim to 6th edn		12th (1922) alcohol responsible for the 'great majority of cases' of cirrhosis, but some have downplayed the role of alcohol	
Alexander Wheeler, *Handbook of Medicine*	2nd (1903) 'By far the most important factor in producing this disease is the excessive use of alcohol, especially spirits'	3rd (1908) 'Of those causes that may affect the liver by way of the portal vein, alcohol, especially in the form of spirits, is certainly the most important.'	4th (1912) 'Alcohol is not the direct cause of cirrhosis, its specific action on the liver being to produce fatty change, but it lowers the resistance of the liver to the action of other poisons, or possibly even of micro-organisms, which are then free to set up connective tissue proliferation.'	6th (1920) identical claim to 4th edn		8th (1927) identical claim to 4th edn
Thomas Dixon Savill, *A System of Clinical Medicine*	1st (1903) 'cirrhosis of the liver must still be regarded as mainly the result of alcoholic excess' and 'Alcohol is undoubtedly the most usual cause of atrophic cirrhosis'	2nd (1909) identical claim to 1st edn	3rd (1912) identical claim to 1st edn	5th (1918) Identical claim to 1st edn		8th (1930) While alcohol is 'undoubtedly the most usual cause of atrophic cirrhosis... Alcoholic excess is now known to be only one of the causes of cirrhosis of the liver. Syphilis is in some cases a predisposing factor, and so are many bacterial infections.'
Russell Cecil, *A Textbook of Medicine*						1st (1927) in spite of recent attempts to come up with other explanations, 'very strong clinical opinion still points to *alcohol* as the chief etiological factor. This poison may act directly on liver cells...'
Frederick W. Price, *A Textbook of the Practice of Medicine*					1st (1922) 'large majority of patients... have indulged excessively in alcohol' but also caused by 'excessive indulgence in highly seasoned foods'	
John Conybeare, *A Textbook of Medicine*						1st (1929) Alcoholism 'by far the most important, though by no means the only aetiological factor', possibly directly caused by chronic gastritis indirectly caused by alcohol

	1931~35	1936~40	1941~45	1946~50
William Osler, *The Principles and Practice of Medicine*		13th (1938) Cirrhosis 'occurs most commonly' in whiskey, gin, and brandy drinkers'	14th (1942) Cirrhosis 'occurs most frequently' in whiskey, gin, and brandy drinkers but 'not always due to alcohol' and can be caused by syphilis	16th (1947) ' just how the alcohol causes cirrhosis remains unknown' while 'there is now a considerable volume of evidence pointing to nutritional deficiency as of greatest importance in etiology of cirrhosis'
Frederick Taylor, *A Manual of the Practice of Medicine*		15th (1936) 'great majority' of cirrhosis 'dependent, wholly or in part, upon the excessive use of alcohol, but '[w]idest individual differences' in dosage required to produce the disease		
Alexander Wheeler, *Handbook of Medicine*		10th (1937) identical claim to 4th edn		11th (1952) liver 'made more vulnerable to the action of these slow poisons (like alcohol) by nutritional deficiencies (whereby it loses the protective action of adequate protein and glycogen and possibly the vitamin B complex), or by earlier infections or poisonings (e.g. syphilis, infectious hepatitis, treatment with arsenicals).'
Thomas Dixon Savill, *A System of Clinical Medicine*	11th (1939) identical claim to 8th edn			13th (1950) 'Alcohol undoubtedly predisposes to atrophic cirrhosis' and 'dietetic factor may play a part'
Russell Cecil, *A Textbook of Medicine*		4th (1938) identical claim to 1st edn	6th (1944) 'Clinical and experimental studies strongly suggest that a direct relationship exists between nutritional deficiency and the development of portal cirrhosis. Whether alcoholism... simply predisposes to the dietary deficiency... or whether it lends an added toxic effect is not clearly established.'	7th (1947) while ' in the large majority of cases there is no known hepatotoxin', experimental studies strengthen '[t]he possibility that nutritional deficiency might play an etiologic role'
Frederick W. Price, *A Textbook of the Practice of Medicine*	4th (1934), 'It is probable, therefore, that alcohol produces cirrhosis of the liver indirectly by leading to gastroenteritis' but '[a]s ordinary gastroenteritis does not lead to cirrhosis, an additional factor must be present. This is probably the direct poisonous action of alcohol and occasionally of other toxins'	5th (1937) identical claim to 4th edn		7th (1947) alcohol probably produces cirrhosis 'only when the liver is abnormally vulnerable owing to constitutional inferiority, vitamin or other dietetic deficiencies, and the simultaneous or previous action of the toxins of such infections as syphilis, malaria, amoebiasis, and the virus of infective hepatitis' and 'cirrhosis very rarely develops experimentally in animals
John Conybeare, *A Textbook of Medicine*		4th edn (1939) identical claim to 1st edn		9th (1949) 'The aetiology is not understood, but it is possible that it depends on a combination of factors which have to do with qualitative dietary insufficiencies... Alcohol, in producing a chronic gastritis, would thus play a definite role in augmenting this process in a susceptible individual.'
G. E. Beaumont, *Medicine Essentials for Practitioners and Students*	1st (1932) alcohol 'undoubtedly is a factor of great importance' though nature of irritant is not certain in all cases		4th (1942) 'The nature of the irritant is not certain in every case, but alcohol is usually considered to be a factor of some importance.'	5th (1948) 'The nature of the irritant is not certain in every case, but alcohol is usually considered to be a factor of some importance, possibly because the diet is low in protein and in vitamin B.'
Derrick Dunlop, *Textbook of Medical Treatment*		2nd (1940) alcohol 'still by far the commonest cause of hepatic cirrhosis. Arguments as to how it acts do not concern us here.'		4th (1946) 'alcohol is still by far the commonest cause of hepatic cirrhosis. Arguments as to how it acts do not concern us here, but recent work of Himsworth adds greater weight to the belief that secondary defects in nutrition are the essential cause of cirrhosis rather than any direct action of alcohol itself.'

	1951~55	1956~60	1961~65	1966~70
Alexander Wheeler, *Handbook of Medicine*			12th (1963) identical claim to 11th edn	
Thomas Dixon Savill, *A System of Clinical Medicine*			14th (1964) 'Although alcohol plays a part in a number of cases... the condition may develop in those who are strict teetotallers.' 'Other factors are a lack of animal protein, an excess of fat in the diet and vitamin B deficiency.'	
Russell Cecil, *A Textbook of Medicine*		10th (1959) the nutritional deficiency theory has been 'greatly strengthened by a number of experimental studies which... established the importance of dietary factors' in cirrhosis, with beneficial results from diet therapy	11th (1963) 'Just how alcoholism, malnutrition, or both, produce hepatic injury in unknown', but alcohol itself 'has not been conclusively proved to have a directly injurious action upon the liver cells'	12th (1967) Very little known on factors that determine individual susceptibility to cirrhosis, but importance of 'genetic and dietary factors'
Frederick W. Price, *A Textbook of the Practice of Medicine*		9th (1956) 'Cirrhosis never develops experimentally in animals as a result of chronic alcohol poisoning... It is probable that the main factor concerned is the low-protein diet which alcoholics often take, combined with their high calorie intake in the form of alcohol.'		10th (1966) Cirrhosis caused by 'dietary deficiencies acting indirectly by interfering with the nutrition of liver cells'
John Conybeare, *A Textbook of Medicine*		12th (1957) 'aetiology is not understood' but 'combination of factors which have to do with qualitative dietary insufficiencies' brought about by 'insufficient protein in the diet with a consequent lack of lipotrophic factors, among which are methionine and choline'	13th (1961) Alcohol 'is a factor of the greatest importance, but the way in which this causes cirrhosis is uncertain. Experimental work and tropical experience show that disease was a 'nutritional defect' indirectly caused by alcohol by neglecting consumption of foods containing 'protective' substances like choline and methionine. Direct toxicity of alcohol on liver cells has not been demonstrated.	
G. E. Beaumont, *Medicine Essentials for Practitioners and Students*		7th (1958) the 'question whether alcohol exerts a direct toxic effect on the liver is not finally decided', but cirrhosis can be caused by 'either directly from protein nutritional deficiency, or indirectly because excessive consumption of alcohol results in a lowered protein intake, or causes a gastro-enteritis which interferes with the digestion and absorption of protein.'	8th (1962) identical claim to 7th edn	
Derrick Dunlop, *Textbook of Medical Treatment*		7th (1958) known causes of cirrhosis include alcohol, deficiency, and poisons, but aetiological effect of alcohol is unclear. Possibility that drinking is an indirect factor to deficiency.		10th (1966) known causes of cirrhosis include alcohol, deficiency, and poisons, but aetiological effect of alcohol is unclear. Possibility that drinking is an indirect factor to deficiency.
T. R. Harrison, *Principles of Internal Medicine*	1st (1951) 'coexistence of alcoholism and nutritional deficiencies in patients' and 'animal experiments point to lipotropic deficiency as a cause of cirrhosis, but 'it would be unwise to translate this experience directly to humans'	3rd (1959) Cirrhosis 'thought to be the consequence of a specific type of malnutrition usually related to chronic alcoholism... It is generally believed that alcohol is 'not a hepatotoxin' and that its effects on the liver are secondary to an associated nutritional disturbance.' Specific nature of deficiency and mechanism of pathogenesis are 'still uncertain'	4th (1962) identical claim to 4th edn	6th (1970) 'epidemiologic studies have implicated' alcoholism as the cause of cirrhosis, but 'there is still no definitive evidence that alcohol by itself leads to cirrhosis'. While a 'contributing factor to the evolution of cirrhosis... malnutrition per se does not lead to Laennec's cirrhosis' but 'a combination of chronic alcohol ingestion plus impaired nutrition leads to liver cell damage and Laennec's cirrhosis'.
Stanley Davidson, *The Principles and Practice of Medicine*	1st (1952) 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency' due to experiments on animals, while 'alcohol itself does not produce cirrhosis of the liver'	4th (1959) identical claim to 1st edn	7th (1964) identical claim to 1st edn	9th (1968) 'much evidence to suggest that hepatic cirrhosis may result from dietetic deficiency' due to experiments on animals, while 'alcohol itself does not produce cirrhosis of the liver'

	1971~75	1976~80	1981~85	1986~1990
Russell Cecil, *A Textbook of Medicine*		15th (1979) identical claim to 12th edn	17th (1985) 'Ethanol is an hepatotoxin' but nutrition also seems to play some role	
Frederick W. Price, *A Textbook of the Practice of Medicine*	11th (1973) 'cirrhosis is due to the harmful effects of alcohol on the liver', while actual factor producing it is 'unknown' even if some liver damage is 'guaranteed' if you drink	12th (1978) identical claim to 11th edn		
John Conybeare, *A Textbook of Medicine*	16th (1975) Alcohol included among 'Toxins' with Iron and Copper as possible aetiologies of cirrhosis, does not include nutritional deficiency			
G. E. Beaumont, *Medicine Essentials for Practitioners and Students*				
Derrick Dunlop, *Textbook of Medical Treatment*	12th (1971) Cirrhosis covers variety of liver disorders that can be 'viral infection, alcohol or nutritional deficiency', but aetiology 'is unknown in many instances'			
T. R. Harrison, *Principles of Internal Medicine*	7th (1974) 'epidemiologic studies have implicated' alcoholism as the cause of cirrhosis. While a 'contributing factor to the evolution of cirrhosis... malnutrition per se does not lead to Laennec's cirrhosis' but 'a combination of chronic alcohol ingestion plus impaired nutrition leads to liver cell damage and Laennec's cirrhosis'.	8th (1977) identical claim to 7th edn	10th (1983) identical claim to 7th edn	
Stanley Davidson, *The Principles and Practice of Medicine*	11th (1974) 'It is generally agreed that dietary deficiency plays a part in the genesis of some cases of hepatic cirrhosis.'		13th (1981) 'The mechanism whereby alcohol damages the liver is unknown; it is now, however, accepted, as a direct liver toxin in man and in other primates'	
Oxford Textbook of Medicine			1st (1983) Alcohol and hepatitis B account for majority of aetiology in western world, alcohol's aetiology taken for granted without discussion	2nd (1987) 'The vast majority are due to alcohol, hepatitis B, or non-A, non-B hepatitis' but '[s]ince only a proportion of heavy drinkers develop alcoholic liver disease, it has been suggested that susceptible individuals might metabolize alcohol abnormally.'

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