

Alcohol and hepatitis C

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The public regard hepatitis C virus (HCV) infection as an ailment of substantial concern, as evidenced by the more than 12 million Google search results — many of which are from consumer groups. It is unsurprising that the public have such a view, as early medical evidence largely supported this. However, recent evidence demonstrates that the disease is more benign and subject to substantial heterogeneity.¹ Modifiable causes of heterogeneity, such as alcohol intake, remain under-publicised, but, as disease labelling generates anxiety, it is imperative that health care providers refashion the public's impression and understanding of the disease and the effect of alcohol. The reduction in morbidity, mortality and treatment costs of hepatitis C could be substantial.

In 2003–04, the Australian Government subsidised the recommended combination therapy for HCV (interferon and ribavirin) by \$86.4 million annually, and underwrote the employment of a large number of personnel to manage its administration in the states and territories.^{2,3} The pharmaceutical industry has actively supported the government, medical and general public's emphasis on the menace of HCV, implying the benefits of pharmacological treatment outweigh the risks and the opportunity costs of alternate and complementary strategies.

Early follow-up data supported an intensive pharmacological approach, with estimates of 20%–30% of individuals becoming cirrhotic in 20–30 years.¹ The focus on patients referred to tertiary units made the early studies unrepresentative, as they were:

- selected patients (referral bias), with
- more aggressive disease (selection bias), and
- more likely to have concurrent liver disease leading to referral (eg, alcohol) (selection bias).

More advanced disease, not surprisingly, led to poor outcomes. In contrast, recent large follow-up studies of patients with transfusion-associated and community-acquired hepatitis C showed much more benign courses with less excess mortality.¹ These studies, including two population-based studies, provide important insights into why prognosis is variable. A New South Wales study of more than 75 000 HCV notifications (1990–2002) followed up for an average of 4.6 years reported 4008 deaths (5.3%), with drug use, mental and behavioural disorders, and alcoholic liver disease increasing the death rate by 19, 15 and 8 times the background population risk, respectively.⁴ Findings from a study in the United Kingdom were similar and showed on follow-up that current alcohol consumption predicted liver-related mortality independently of initial liver biopsy findings.⁵

The high incidence of alcohol-related disorders alludes to a key issue in hepatitis C progression — its deleterious interaction with alcohol, first recognised more than 15 years ago.⁶ The evidence for alcohol's deleterious effects on HCV RNA levels, treatment response, and disease progression was sufficiently persuasive for the United States National Institutes of Health in 2002 and the American Gastroenterological Association in 2006 to issue statements advising that “abstinence is strongly recommended before and during antiviral therapy ... [as] even moderate levels of [alcohol] consumption may accelerate [liver] disease progression in some patients”.⁷ Clinical practice confirms HCV's

ABSTRACT

- Although early studies of hepatitis C indicated this is a serious disease, more recent evidence shows it can be relatively benign.
- A major determinant of hepatitis C prognosis is alcohol consumption.
- Promotion of alcohol abstinence among people with hepatitis C could result in substantial reductions in morbidity, mortality and treatment costs.

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interaction with alcohol, with most patients admitted to hospital with hepatitis C and advanced liver disease having a history of heavy alcohol consumption.

Despite the laboratory and clinical evidence, alcohol's significance has not permeated clinical practice.⁸ In 2000, NSW Health advice was: “People with hepatitis C *can* [our italics] consider the following actions to improve their health: stop drinking alcohol or cut down alcohol intake”.⁹

At the operational level, hepatitis C and drug and alcohol services await integration.

Progression to cirrhosis at 20 years is now reported to occur in 7% of community-acquired, 4% of adult and 2%–4% of childhood transfusion-associated HCV, with longitudinal studies attributing some of these events to HCV and alcohol consumption.¹

Little has been said about the harm the public's misconception generates. One of the earliest follow-up studies of the effect of hepatitis C diagnosis on quality of life followed up 34 individuals previously diagnosed with non-A, non-B hepatitis at a single Australian institution. Compared with 19 individuals (54%) who, 2 years later, were unaware of their revised diagnosis, the 15 who were aware reported significantly poorer quality of life in seven of eight quality-of-life scores (SF-36) compared with population controls.¹⁰ In other settings, diagnosis of well individuals has been reported to have an adverse psychological effect.¹¹ Hypertensive individuals identified through workplace screening programs have shown increased worry, greater sick leave and reduced perceived health, irrespective of whether their hypertension necessitated treatment.¹¹ To diminish patient anxiety, clinicians need to ensure patients are appropriately counselled and accurately informed.

The available evidence indicates that alcohol consumption is the strongest known modifiable determinant of hepatitis C outcome and that, with abstinence, the disease course is reassuringly benign, particularly for community-acquired infection.¹² Unless holistically managed, the “hype of hep C” may generate psychological harm in excess of treatment benefit for most patients. Future HCV studies need to quantify concurrent alcohol consumption to further our understanding of the total burden of illness from alcohol-associated hepatitis C in the community.

Competing interests

None identified.

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