

Modelling hepatitis C virus incidence, prevalence and long-term sequelae in Australia, 2001

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Background To plan an appropriate public health response to the hepatitis C virus (HCV) epidemic requires that estimates of HCV incidence and prevalence, and projections of the long-term sequelae of infection, are as accurate as possible. In this paper, mathematical models are used to synthesize data on the epidemiology and natural history of HCV in Australia to estimate HCV incidence and prevalence in Australia to end 2001, and project future trends in the long-term sequelae of HCV infection.

Methods Mathematical models of the HCV epidemic in Australia were developed based on estimates of the pattern of injecting drug use. Estimates of HCV infections due to injecting drug use were then adjusted to allow for HCV infections resulting from other transmission routes. Projections of the long-term sequelae of HCV infection were obtained by combining modelled HCV incidence with estimates of the progression rates to these outcomes.

Results It was estimated that there were 210 000 (lower and upper limits of 157 000 and 252 000) people in Australia living with HCV antibodies at the end of 2001, with HCV incidence in 2001 estimated to be 16 000 (11 000–19 000). It was estimated that 6500 (5000–8000) people were living with HCV-related cirrhosis in 2001, that 175 (130–210) people developed HCV-associated liver failure, and that there were 50 (40–60) incident cases of HCV-related hepatocellular carcinoma (HCC). It was estimated that in 2001 22 500 quality adjusted life years were lost to chronic HCV infection, the majority (77%) in people with early (stage 0/1) liver disease.

Discussion Model-based estimates were broadly consistent with other sources of information on the HCV epidemic in Australia. These models suggest that the prevalence of HCV-related cirrhosis and the incidence of HCV-related liver failure and HCC will more than triple in Australia by 2020.

Keywords Australia, drug abuse, epidemiology, hepatitis C, models

Hepatitis C virus (HCV) infection is the most common notifiable infectious disease in Australia. To the end of 2000, over 160 000

diagnoses of HCV were reported to State and Territory surveillance systems,¹ with approximately 65% in the age range 20–39 years, and an overall male to female ratio of 1.8:1.0.

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Studies of HCV risk factors in Australia indicate that around 80% of prevalent HCV infections were through injecting drug use,² in whom HCV prevalence has ranged from 50% to 70% since the early 1970s. Hepatitis C virus incidence among injecting drug users (IDU) in the 1980s and early 1990s has been estimated to be around 15% per year, with some evidence of a decline in HCV incidence in the late 1980s, coincident with the introduction of needle and syringe programmes in Australia primarily aimed at preventing HIV infection among IDU.³

Although information from HCV notifications and seroprevalence surveys among sentinel populations has provided a broad picture of the HCV epidemic in Australia, there are considerable limitations within these surveillance systems. For example, monitoring of recent levels of HCV transmission is problematic due to the generally asymptomatic nature of new HCV infection. Surveillance of the long-term consequences of chronic hepatitis C is also difficult. Unlike HIV/AIDS surveillance in Australia, advanced disease manifestations are not currently notifiable for HCV. Therefore, in 1998 a working group was established to estimate HCV incidence and prevalence, as well as the current and future burden of HCV disease. Initial estimates from the working group were that 190 000 people were living with HCV antibodies at the end of 1997, with 11 000 new HCV infections in 1997.⁴

To further assist in evaluation of HCV prevention strategies and assessment of treatment and care needs in Australia, a second round of HCV estimates and projections was undertaken. In addition to updated HCV estimates and projections, this paper presents estimates of the number of people with HCV by liver disease stage. As antiviral therapy for chronic hepatitis C is generally recommended for those who have progressed to moderate hepatic fibrosis, this information

is crucial for assessment of current and future treatment needs.

Methods

Modelling strategy

The models adopted are a refinement of the models developed by the previous working group,⁴ and were based on the following strategy:

- First, the number of people injecting drugs in Australia over the last three decades was estimated.
- Based on this pattern of injecting drug use, and estimates of HCV incidence among IDU derived from cohort studies, HCV incidence as a result of injecting drug use was estimated.
- These estimates of HCV incidence due to injecting drug use were then adjusted in accordance with epidemiological data to allow for HCV infections through other transmission routes, including receipt of blood or blood products.
- Estimates of the number of people experiencing long-term sequelae of HCV infection were then obtained from the estimated pattern of HCV incidence using rates of progression derived from cohort studies.

Estimates of trends in injecting drug use in Australia

In the models, IDU are divided into regular and occasional injectors. Regular IDU are defined as a person who has injected for at least 12 months, an average of 10 times per month with injecting in most months. Occasional IDU are all other people who have injected in the previous 12 months.

Data regarding the number of IDU in Australia are summarized in Table 1. The numbers of regular and occasional IDU

Table 1 Estimated number of injecting drug users and dependent heroin users in Australia

Method	Data source	Year	No. (lower, upper limits)	Reference
Estimates of number of injecting drug users				
Household surveys	National Drug Strategy Household Survey	1993	255 000 ever injected	5
		1995	70 000 injected in last 12 months	5
	1998	190 000 ever injected 85 000 injected in last 12 months	6	
Delphi	Expert opinion	1997	110 000 injected in last 12 months 100 000 regular IDU ^a (80 000–120 000) 175 000 occasional IDU (120 000–240 000)	4
Estimates of number of dependent heroin users				
Multiplier method	Various	1984–1987	34 000 (25 000–86 000)	28
		1988–1992	59 000 (49 000–150 000)	28
Back projection	Overdose deaths	1997	74 000	7
		1997	68 000	7
	Methadone entrants	1967	670 (460–1100)	29
		1977	7000 (5000–11 000)	
		1987	30 000 (19 000–51 000)	
Back projection	Methadone entrants	1997	67 000 (39 000–120 000)	
		1977	4000 (2000–6000)	29
		1987	29 000 (18 000–44 000)	
Capture-recapture	Methadone maintenance	1997	71 000 (47 000–109 000)	
		1995–1998	82 000 (68 000–109 000)	30
	Heroin arrests	1997–1998	86 000 (78 000–102 000)	

^a Intravenous drug users.

estimated from the National Drug Strategy Household Surveys^{5,6} were based on surveys of the general population. It is likely that both surveys gave underestimates of the total number of people who had injected drugs in the previous 12 months because IDU would probably be less likely to be contacted by such surveys, and even if contacted they may not have admitted to an illegal activity. However, a consistent feature of these survey results was an increasing number of people reporting injecting drug use, from 70 000 in 1993 to 110 000 in 1998.

Because of the lack of data on the numbers of IDU in Australia at the time of the previous projections, the Delphi technique was used to reach consensus estimates.⁴ The number of regular IDU in 1997 was estimated, using the Delphi method, to be 100 000, with lower and upper plausible limits of 80 000 and 120 000. The number of occasional IDU was estimated to be 175 000 (120 000–210 000).

Since 1998, further modelling, applied to national overdose deaths, New South Wales methadone maintenance therapy entrants, methadone maintenance therapy episodes, and arrests for heroin-related offences, has been used to estimate numbers of dependent heroin users in Australia (Table 1). The median number of dependent heroin users in Australia in 1997 was 74 000,⁷ with consistent evidence of increasing numbers of dependent heroin users over the previous 30 years.

The estimates of the numbers of dependent heroin users are plotted over time in Figure 1. A reasonable visual fit to these estimates was obtained by taking a consistent 8% annual increase in the number of dependent heroin users from 1970 to a total of 75 000 dependent heroin users in 1997. Prior to 1970 it was assumed there was a linear decline in the number of dependent heroin users to negligible numbers prior to 1960.

To include people injecting drugs other than heroin, it is necessary to inflate these estimated numbers of dependent heroin users to give total estimated numbers of regular injectors. Data from National Needle and Syringe Program (NSP) surveys indicate that around three-quarters of people attending NSP report heroin as the most recently injected drug.⁸ This suggests that the total

number of regular IDU in 1997 was around 100 000, consistent with estimates used in the previous HCV projections.

Modelling assumptions

The incidence of HCV in Australia was modelled based on the following assumptions regarding the past pattern of injecting drug use in Australia:

- there were 100 000 regular IDU in 1997 (lower and upper limits, 80 000–120 000), with a constant net increase of 8% per year between 1970 and 2001, and with 5% stopping injecting each year.^{9,10}
- there were 175 000 occasional IDU in 1997 (lower and upper limits, 120 000–210 000), with a constant net increase of 8% per year between 1970 and 2001, and with 10% stopping injecting each year (based on Delphi study estimates⁴).
- there were negligible numbers of IDU prior to 1960, with a linear increase in the number of both regular and occasional IDU between 1960 and 1970.

Hepatitis C virus incidence was modelled based on these assumed patterns of injecting drug use. The models used, and other assumptions made, were identical to those adopted in the previous projections.⁴ Estimates of HCV prevalence were adjusted to allow for mortality related to HCV injecting drug use and unrelated to HCV or injecting (see section below).

Sensitivity analyses performed in the previous HCV projections indicated that the single most important source of uncertainty in estimates of HCV incidence and prevalence arose due to uncertainty in the number of regular and occasional IDU. Hence, lower and upper limits on the HCV epidemic were produced based on the upper and lower limits of the numbers of regular and occasional IDU.

Long-term sequelae of HCV infection

Estimates and projections were made for the three main long-term sequelae of HCV infection: compensated cirrhosis, liver failure, and hepatocellular carcinoma (HCC).

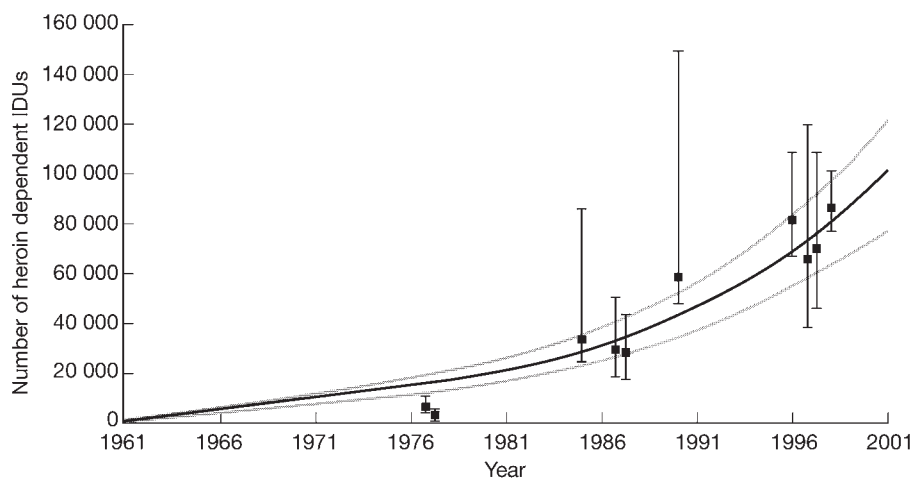


Figure 1 Modelled numbers of heroin dependent intravenous drug users 1961–2001

Best modelled estimates in black, lower and upper limits in grey (see text for details). See Table 1 for references. Vertical bars are upper and lower limits on points.

The progression rate to cirrhosis was modelled in the following way. It was first assumed that 75% of people exposed to HCV developed chronic infection.^{11,12} Of people with chronic HCV infection, it was assumed that one-third had normal alanine aminotransferase (ALT) values, one-third abnormal ALT values, and one-third abnormal ALT values with further covariates which would indicate that they would be at increased risk of progression (e.g. high alcohol intake). Annual rates of progression from stage 0/1 liver disease to stage 2/3 liver disease, and from stage 2/3 liver disease to cirrhosis are shown in Table 2.

Taken together, these assumptions combine so that of all people exposed to HCV, 5.3% and 15.3% are estimated to develop cirrhosis by 20 and 40 years respectively, with 7.1% and 20.4% of people with chronic HCV developing cirrhosis by 20 and 40 years respectively. This is consistent with a recent systematic review of current evidence regarding progression rates to cirrhosis,¹³ and with an accelerating rate of disease progression with increasing duration of HCV infection.¹⁴

Rates of developing liver failure or HCC from cirrhosis were assumed to be 4% and 1% respectively.¹⁵ It was further assumed that HCC could develop following liver failure, but not *vice versa*. Hepatitis C virus-related mortality following cirrhosis was taken to be 1.5% per annum.¹⁶

Estimates of the numbers of people with long-term sequelae of HCV infection were obtained by combining the estimated pattern of HCV incidence with the assumed progression rate distributions. Mortality unrelated to HCV due to injecting drug use, both before and after cirrhosis, was assumed to be 1% per year.^{9,10} Background mortality was based on Australian Bureau of Statistics (ABS) life tables, assuming that the mean age at HCV seroconversion among IDU was 25 years,⁸ and that there were two male HCV-infected IDU for each female HCV-infected IDU.¹⁵ Lower and upper limits were obtained based on the lower and upper limits of HCV incidence. The best estimates and upper limits were obtained by assuming that numbers of IDU continued to increase at 8% per annum until 2020. Lower limits were obtained by assuming that the lower limit of HCV incidence in 2001 continued at this lower level until 2020. In any event, because of the protracted progression rate distributions to cirrhosis, liver failure, and HCC, the projected numbers of long-term sequelae depend primarily on HCV infections that have already occurred by 2000.

Loss of quality of life associated with HCV infection

Although there may be quality of life impairment and health care costs for people who are HCV antibody positive but do not

have chronic HCV, we have taken the conservative approach of assuming impairment only in cases of chronic HCV.

Definitions for disease states and associated quality of life adjustments are as follows:

- Chronic HCV, Stage 0/1 and 2/3 fibrosis, undiagnosed (0.94)
- Chronic HCV, Stage 0/1 and 2/3 fibrosis, diagnosed (0.82)
- Compensated cirrhosis, undiagnosed (0.84)
- Compensated cirrhosis, diagnosed (0.74)
- Liver failure (0.32)
- HCC (0.10)

These quality of life adjustments were partly based on previous published estimates from a panel of hepatologists.¹⁷ However, recent studies indicate no significant difference in quality of life based on either degree of hepatic inflammation (as measured by ALT/AST) or extent of hepatic fibrosis.¹⁸ Therefore, we have used the same quality of life adjustment for diagnosed stage 0/1 and stage 2/3 chronic hepatitis. Undiagnosed categories have higher quality of life estimates for two reasons. First, symptomatic disease is often a reason for HCV testing. Second, recent evidence suggests that quality of life impairment increases following diagnosis of HCV.¹⁹ We have combined the quality of life adjustments from¹⁷ for ascites (0.35), variceal haemorrhage (0.28), and hepatic encephalopathy (0.30), to produce a category for liver failure (0.32). We have assumed that all people with liver failure and HCC are aware of their HCV status.

Results

Modelled estimates of hepatitis C virus prevalence and incidence in 2001

The modelled pattern of past HCV incidence is shown in Figure 2. Overall, the pattern of HCV incidence is consistent with a gradually increasing rate of HCV infections over the last three decades. The apparent plateau in HCV incidence in the late 1980s is a combination of the assumed decreasing HCV incidence in IDU, and the gradual decrease in HCV transmissions through receipt of blood products initially due to screening of donors aimed at reducing HIV transmission.

The best estimate of the number of people living with HCV antibodies to the end of 2001 was 210 000 (lower and upper limits, 157 000–252 000), with HCV incidence in 2001 estimated to be 16 000 (11 000–19 000). Of all HCV infections, 83% were estimated to be due to injecting drugs, 5% due to receipt of blood, and 12% due to other transmission routes. Of incident HCV infections in 2001, 91% were estimated to be through injecting drug use, 0% to receipt of blood, and 9% due to other reasons.

If we assume that what we believe about recent trends in injecting drug use continue, with an annual 8% increase in the total number of IDU, for the foreseeable future, the total number of people living with HCV in 2020 is estimated by the current models to be 836 000. A much more conservative scenario can be obtained by basing projections on the current lower limits on number of IDU, and to assume that the total number of IDU remains contained at the lower limit levels estimated in 2001. Under this scenario, the models estimate there would be 321 000 people living with HCV infection in 2020.

The modelled best estimates of number of people living with HCV by stage of disease from 1961 to 2001 are shown in Figure 3.

Table 2 Annual rates of liver disease progression

	Stage 0/1 to stage 2/3	Stage 2/3 to cirrhosis
Not chronic HCV ^a	0%	0%
Chronic HCV, normal ALT ^b	1%	1%
Chronic HCV, abnormal ALT	2%	2%
Chronic HCV, abnormal ALT and further cofactors	3%	3%

^a Hepatitis C virus.

^b Alanine aminotransferase.

Note: Stage 0 = no hepatic fibrosis; stage 1 = minimal hepatic fibrosis; stage 2 = moderate hepatic fibrosis; stage 3 = severe hepatic fibrosis; stage 4 = cirrhosis.

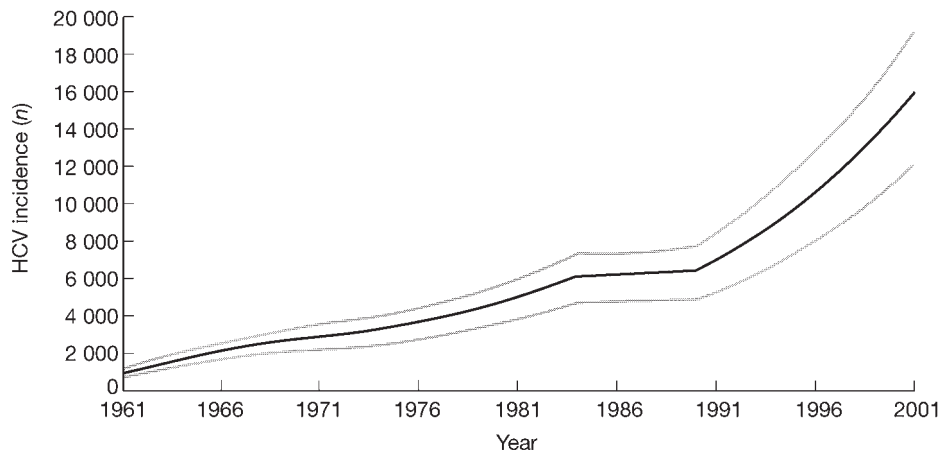


Figure 2 Modelled hepatitis C virus incidence 1961–2001
Best estimates in black, lower and upper limits in grey (see text for details).

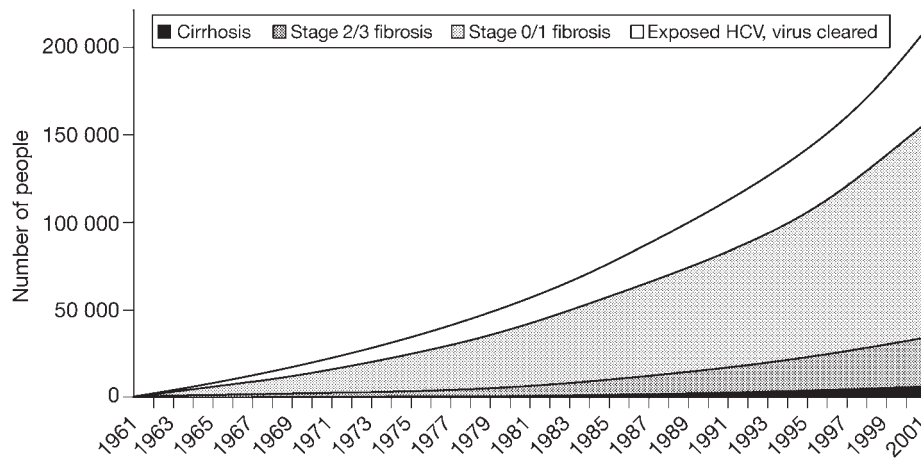


Figure 3 Estimated number of people living with hepatitis C virus by stage of disease 1961–2001

At the end of 2001 these models estimate that there was a total of 210 000 (lower and upper limits, 157 000–252 000) people living with HCV in Australia. Of these, 53 000 (39 000–64 000) people had been exposed to HCV but had cleared the virus and were not chronically infected, 124 000 (92 000–149 000) were chronically infected with HCV with stage 0/1 liver disease, 27 000 (20 000–32 000) were chronically infected with HCV with stage 2/3 disease, and 6500 (5000–8000) were living with cirrhosis.

Modelled estimates of long-term sequelae of HCV infection

The modelled number of people living with cirrhosis from 1990 to 2020 is shown in Figure 4. In 2001 the models estimate that there were: 6500 (5000–8000) people living with cirrhosis, 175 (130–210) new cases of HCV-related liver failure, 50 (40–60) HCV-related cases of HCC, and 1000 (750–1200) cumulative HCV-related deaths.

For all these measures of the long-term sequelae of HCV infection, the models predict there will be at least a threefold increase by 2020.

Estimates of reduced quality-adjusted life years (QALY) associated with hepatitis C virus infection

In Australia, after allowing for up to 10% duplicate reporting of HCV diagnoses, an estimated 70% of people living with HCV are diagnosed. Table 3 outlines the estimates of diagnosed chronic HCV by stage of liver disease. It was assumed that proportions of diagnosed chronic HCV would increase with disease stage to reach 100% for advanced liver disease complications (HCC, liver failure).

In total, it is estimated that 22 500 (17 000–27 000) QALY were lost to HCV infection in Australia during 2001. Even though the QALY adjustments are modest in early stage HCV disease, the majority of the estimated QALY lost to HCV infection during 2001 were in either stage 0/1 disease (73% of QALY lost) or stage 2/3 disease (18% of QALY lost).

Discussion

The estimates of HCV prevalence and incidence presented here are based on a refinement of earlier models, and findings are broadly consistent.⁴ Current estimates are that there were

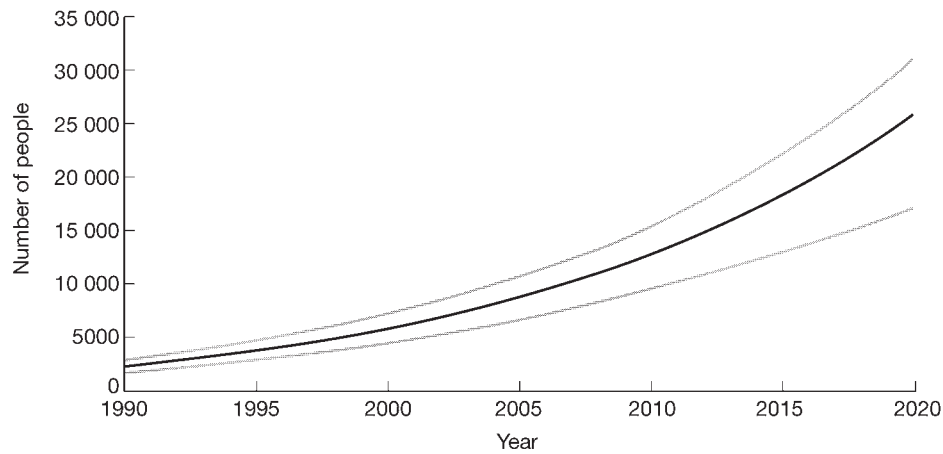


Figure 4 Estimated number of people living with hepatitis C virus-related cirrhosis 1990–2002
Best estimates in black, lower and upper limits in grey (see text for details).

Table 3 Estimated quality of life years lost to hepatitis C virus (HCV) infection in Australia in 2001 by stage of liver disease

	Percentage	Person years in 2001 (lower/upper limits)	QALY ^a adjustments	QALY lost (lower/upper limits)
Mild chronic HCV				
Undiagnosed	40%	50 000 (37 000–60 000)	0.94	3000 (2200–3600)
Diagnosed	60%	74 000 (55 000–89 000)	0.82	13 400 (9900–16 000)
Moderate chronic HCV				
Undiagnosed	25%	7000 (5000–8000)	0.94	400 (300–500)
Diagnosed	75%	20 000 (15 000–24 000)	0.82	3600 (2700–4300)
Compensated cirrhosis				
Undiagnosed	20%	1000 (800–1300)	0.84	160 (130–200)
Diagnosed	80%	4100 (3200–5100)	0.74	1100 (800–1300)
Liver failure^b				
Diagnosed	100%	1300 (1000–1600)	0.32	880 (680–1100)
Hepatocellular carcinoma				
Diagnosed	100%	50 (40–60)	0.10	45 (35–55)
Total		210 000 (157 000–252 000)		22 500 (17 000–27 000)

^a Quality adjusted life year.

^b Person years of liver failure in 2001 calculated as cumulative incidence of liver failure and hepatocellular carcinoma (HCC) minus cumulative HCV-related mortality, which assumes average duration of survival with HCC is around 12 months.

210 000 (157 000–252 000) people living with HCV at the end of 2001, compared with the previous report's estimated 190 000 (140 000–240 000) at the end of 1997. Hepatitis C virus incidence is estimated here to be 16 000 (11 000–19 000) in 2001 compared with 11 000 (8500–13 500) in 1997 in the previous report. Current estimates are that HCV incidence in 1997 was 12 000 (9000–14 000). Current estimates of the long-term sequelae of HCV infection are lower than previous estimates. The number of people living with cirrhosis is currently estimated to be 6500 in 2001 compared with 8500 in 1997 (previously estimated), and HCC incidence is now estimated at 50 cases in 2001 compared with 80 in 1997 (previously estimated). Estimates

of long-term sequelae are lower in our current estimates than previously because, following a systematic review of the literature,¹³ we now believe rates of progression from HCV infection to cirrhosis are slower than we had previously assumed. However, a consistent feature of both the present and previous projections is that long-term sequelae are projected to increase quite rapidly over the next two decades.

Our current estimates of HCV prevalence and incidence are based on the assumption that the trends in injecting drug use which appear to have occurred during the mid 1990s continue through 2001. This is clearly a highly uncertain assumption. There has been recent evidence of a heroin drought in

Australia from the last quarter of 2000 through to early 2002.²⁰ This may have resulted in fewer people injecting heroin, although there have been anecdotal reports that any decrease in injecting of heroin has been replaced by increased injecting of amphetamines and cocaine. Certainly, a decrease in opioid overdose deaths from a peak of 958 in 1999 to 725 in 2000 has been reported,²¹ suggesting some decrease in injecting opioids. The number of needles and syringes distributed through NSP has also been reported to have decreased. If there was a decrease in the number of people injecting drugs in 2001, then the best estimates of HCV incidence and prevalence may be too large. The modelled estimate of HCV incidence in 2001 is particularly sensitive to assumptions regarding the number of IDU, and the best estimate of 16 000 needs to be interpreted as particularly uncertain.

The estimated 210 000 people living with HCV during 2001 is corroborated by other data, and so appears more robust. To the end of 2000 there have been over 160 000 HCV diagnoses notified through State/Territory health departments. With 10% duplicate reporting, this corresponds to around 30% of people living with HCV being undiagnosed. Furthermore, HCV prevalence among antenatal patients has been estimated to be 1.1% in 1995²² and 1.3% in 1999.²³ Direct estimates of HCV prevalence extrapolated from these estimates of HCV prevalence in antenatal patients would be of the order of 200 000 people living with HCV in the late-1990s.⁴

We have projected the numbers of people living with HCV in 2020 based on two scenarios. First, assuming current increasing trends in injecting drug use continue through 2020, we estimate there will be 836 000 people living with HCV in 2020. Second, assuming injecting drug use remains at the lower limit of best estimates in 2001 we estimate 321 000 in 2020. The numbers of people living with HCV in 2020 are likely to lie somewhere in between these two scenarios. It is difficult to argue that numbers of IDU will continue to increase by 8% per annum for a further two decades because the available population sizes of young adults in Australia are too small. Similarly, in the face of strong evidence of a continuing increase in the number of IDU throughout the 1990s, it is difficult to argue that the number of IDU will remain contained at the lower limit of best estimates in 2001. Thus the total of 836 000 people living with HCV in 2020 can be viewed as an upper limit on the likely numbers, and 321 000 a lower limit. The only realistic possibility that the number of people living with HCV in 2020 will be below this range is if a HCV vaccine becomes available, if HCV transmission among IDU is markedly reduced, or if anti-HCV treatments improve very rapidly to the point where a large proportion of people who are at risk of transmitting HCV (predominantly current IDU) can access treatment and clear their HCV infection and are thus no longer infectious.

In Australia, both HCC incidence and mortality has increased over the last two decades, particularly in men.²⁴ The reported annual number of cases of HCC has gradually increased over the last decade or more, from 215 cases in 1983 to 603 cases in 1998.²⁵ The proportion of HCC cases due to HCV infection is

uncertain. In one study of cases of HCC in Sydney between 1990 and 1993, 5 of 9 (56%) cases tested were HCV infected.²⁶ A second study in Victoria in 1991 to 1992 found that 7 of 24 (29%) cases tested were HCV infected.²⁷ However, in both studies rates of testing for HCV infection were low, and the actual rate of HCV infection in cases of HCC would be somewhat lower than these rates if testing was selectively done in cases thought to be at an increased risk of HCV infection. Based on our projections of HCC, we estimate that 7% (5–8%) of HCC cases in 1998 were due to HCV infection. Some authors suggest that HCC rates following cirrhosis should be higher than the 1% per annum we assumed. If HCC rates following cirrhosis are increased to 2% per annum, then the estimated HCV-related HCC incidence in 2001 is increased to 100 cases, with a corresponding 14% of HCC cases in 1998 due to HCV infection.

Both the current and previous estimates of the HCV epidemic in Australia are based on essentially simple models. These models cannot aim to capture all the complexities of the injecting drug use and HCV epidemic. For example, injecting behaviours are probably quite different depending on the drug injected, and consequently HCV incidence probably also differs according to drug injected. Further, certain subgroups of IDU, for example indigenous Australians, may have different injecting behaviours than other subgroups. There may also be sex and age differences. Unfortunately, epidemiological data are simply not available to allow more complex models to be specified with any certainty, and such models were not attempted. In our models we have sought to capture the essential features of the HCV epidemic in Australia, without undue model complexity leading to large numbers of unverifiable assumptions. It is somewhat reassuring that our models are consistent with available epidemiological data on risk factors for HCV, and where there are corroborative data on HCV prevalence, these data are consistent with our modelled estimates. As new data become available more complex modelling may be possible, leading to further refinements in our estimates. It should also be noted that the upper and lower limits we give on our estimates correspond to plausible ranges based on sensitivity analyses, and should not be interpreted as formal confidence intervals.

An important addition to our current results are estimates of the QALY lost through HCV infection in 2001. We estimate that a total of 22 500 QALY were lost in 2001, the majority in people with stage 0/1 (77%) and stage 2/3 (18%) chronic HCV infection. Even though mortality associated with HCV infection is currently at modest, though not inconsequential, levels in Australia, these estimates suggest that lost quality of life through HCV infection is a larger problem, affecting all stages of chronic HCV infection.

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KEY MESSAGES

- Using mathematical models it is estimated that there were 210 000 people living with antibodies to hepatitis C virus (HCV) in Australia in 2001.
- Hepatitis C virus incidence was estimated to be 16 000 new infections in 2001, and increasing over time largely as a result of continuing increases in injecting drug use in Australia throughout the 1980s and 1990s
- In 2001 it was estimated that there were 6500 people living with HCV-related cirrhosis. Cirrhosis, liver failure, hepatocellular carcinoma, and deaths as a result of HCV were all projected to at least triple by 2020.
- In 2001 it was estimated that 22 500 quality adjusted life years were lost to HCV, the majority in people with little or no liver damage.

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