CHAPTER TWO

A PROFILE OF HEPATITIS C

The existence of a viral cause of hepatitis other than Hepatitis A and Hepatitis B was first recognised in the early 1970s following the development of diagnostic blood tests for Hepatitis A and Hepatitis B. Once these two forms of hepatitis could be identified, there still remained a form of hepatitis which became known as non-A and non-B hepatitis. This term lasted for approximately 20 years. It was not until the late 1980s that a test was developed to detect a particular kind of hepatitis which quickly became known as Hepatitis C.

Since that time, significant advances have been made in understanding the epidemiologic patterns of HCV transmission and its natural history and modes of transmission, but as MacDonald, Crofts and Kaldor observe, "much remains to be learned in all these areas" (1996:137).

The following discussion looks at the Hepatitis C virus: what it is, how it spreads, how it manifests itself and how prevalent it is in the community. The Chapter also compares and contrasts Hepatitis C with another blood borne virus: HIV.

2.1 THE HEPATITIS C VIRUS

The current Hepatitis C epidemic is unlike many other epidemics where the consequences are fairly immediately obvious. With Hepatitis C there is a considerable lag period between the epidemic of infection and the epidemic of consequences (Wodak evidence, 2 October 1997). As Wodak explained to the Committee:

> *in the 1990s we are now only just beginning to see the consequences of the epidemic of Hepatitis C that we think probably began in Australia in a big way in the late 1960s when drug injecting took off* (Wodak evidence, 2 October 1997).

2.1.1 AETIOLOGY

The aetiologic agent of this form of infection is the Hepatitis C virus. The virus is relatively robust and can live for an appreciable period of time outside the body. Research presented at the Australian Society for Microbiology National Conference in late September 1998 suggested that the virus can remain infectious in a bloody syringe for up to three weeks (Ferrari, 1998).

The virus was originally identified by Chiron Corporation, a Californian based company.

- **The Chiron Patent**

Having discovered the Hepatitis C virus, Chiron Corporation took out a patent on the virus effectively giving the company a monopoly on research use of the virus. The

Australian Patents Office has granted Chiron Corporation a patent which relates to, amongst other things, the identification of the causative agent of the virus.

The use and treatment of living matter in patentable form is not new. It is often the basis for genetic engineering of food plants and animals. However, in this case, the Committee understands some Australian medical researchers were "incensed" over the government's decision to grant the patent (Mackenzie, 1994:9). The researchers believe such a patent makes HCV diagnostic tests more expensive and inhibits further research into vaccine and treatment development (Mackenzie, 1994:8). When the Committee questioned Professor Farrell on the impact the patent would have on Australian research he informed Members that:

> *it does place some impediments on research which could lead to a commercial outcome, particularly in the area of vaccines . . . and that has certainly been a concern to Australians because we have some really expert virologists in this country who would have been very interested* (Farrell evidence, 28 November 1997).

A representative from the Hepatitis C Council added that:

> *it has not been brought to our attention that there is a massive problem. Morally our belief is that it is a problem, but we have not come across practical difficulties which have prevented research* (Loveday evidence, 30 March 1998).

Court actions in the USA and the UK have involved litigation aimed at testing the legitimate extent of the Chiron patent. British courts ruled that the patent's claims regarding vaccines and cell cultures were not valid due to insufficient legal justification. The British courts found, however, that the patent was valid in regard to the diagnostic kits. Australian courts are still undecided on the validity of the patent. The Committee understands that a case is currently before the Federal Court in which Chiron is claiming that Murex Diagnostic (a diagnostic testing company) has infringed its patent (Mackenzie, 1994:9).

2.1.2 TRANSMISSION OF THE HEPATITIS C VIRUS

Hepatitis C transmission is predominantly parenteral. The primary modes of transmission for this blood borne virus include shared drug injecting equipment, infected blood products (prior to the screening of blood products in February 1990), unsterile skin penetration practices such as tattooing, ear/skin piercing and acupuncture, needlestick and "sharps" injuries and shared personal items that may contain blood such as toothbrushes and razors. Patient-to-patient (via contaminated anaesthetic circuitry) and surgeon-to-patient transmission (via percutaneous injury) have been demonstrated. Sexual transmission without blood contact appears rare but

the risk of blood exposure may be increased by sexual contact during menstruation and anal intercourse. Vertical transmission (mother-to-child) also appears to be rare. Each of these forms of transmission are discussed in further detail in Chapter Three. Arthropod vectors have not been identified (Sladden *et al*, 1997:290).

MacDonald, Crofts and Kaldor (1996:137) note that blood contact does not fully explain HCV transmission. They observe that there has been less certainty about the role of other routes of transmission and factors that modify the efficiency of transmission (MacDonald, Crofts, Kaldor, 1996:137).

Sladden and colleagues have conducted a population based, cross-sectional survey of notified HCV cases to determine the routes of Hepatitis C virus transmission (Sladden *et al*, 1997). The results of their survey, reproduced in Table One, show the routes of Hepatitis C transmission exposure. Non-parenteral transmission appeared to be minimal.

TABLE ONE
POTENTIAL HEPATITIS C TRANSMISSION EXPOSURE

	Number	Likely Primary Exposure	IDUs: Sharer	IDUs: Non-Sharer	Pre-'90 Transfusion	Dialysis	Needle Stick	Tattoos	Blood Splash	Post '90 Transfusion	Ear/Skin Piercing	Clinical Procedure
IDUs: **Sharer** **Non Sharer**	398 354 44	298 354 44										
Pre '90 Transfusion	70	30	30	10								
Dialysis	5	2	2	0	1							
Needlestick	67	6	51	8	9	0						
Tattoo	176	4	143	24	25	2	32					
Blood Splash	60	4	40	9	11	0	21	29				
O/S	13	3	8	0	4	0	2	4	0			
Post '89 Transfusion	14	2	10	0	0	1	3	5	2	0		
Ear/Skin Piercing	354	13	285	29	47	2	52	155	46	10	13	
Clinical Procedures	340	4	251	33	57	2	52	124	45	9	96	256
All Blood Exposures	1497	466										

Source: Sladden, Hickey, Dunn and Beard, 1997:291

The study identified injecting drug use as the mode of transmission for 85% of infections occurring in the Northern Rivers region. The second highest source of infection was blood transfusions prior to 1990. Sladden anticipates that the proportion of infections due to injecting drug use will increase now that the blood is screened for the Hepatitis C virus (Sladden evidence, 30 March 1998).

2.1.3 NATURAL HISTORY OF HEPATITIS C

The natural history of a disease refers to the way it evolves in individual persons and the way it affects their lives. An understanding of the disease's natural history is, therefore important. However, as Seeff has observed, the natural history of chronic Hepatitis C infection has not yet been fully defined (Seeff, 1997:26S). This could be because, as Hoffnagle notes, there is no single typical course or natural history of this specific disease, but rather a broad clinical spectrum of disease presentations and outcomes (Hoffnagle, 1997:15S).

The disease has been described as a "quiet" infection (Anlezark *et al*, 1997:81); a "silent" disease (Farrell evidence, 28 November 1997) which may have "a mixed picture from passivity to overt active behaviour" (Anlezark *et al*, 1997:81). As many as two-thirds of people with Hepatitis C do not know they have the infection even at fairly advanced stages of liver damage (Farrell evidence, 28 November 1997). The disease may manifest itself in either an acute or chronic form.

- **Acute Hepatitis C**

This form of the disease is commonly an asymptomatic and mild illness. Of 100 people exposed to the virus approximately 20 will clear it themselves within four to six weeks of infection. However, of this 100 approximately 80 - 85 will develop chronic Hepatitis C which for many will have long term health consequences.

- **Chronic Hepatitis C**

Chronic Hepatitis C is determined by persistently abnormal serum enzymes and/or viraemia (as measured by HCV RNA reactivity). Like the acute form of the illness, chronic Hepatitis C is also predominantly asymptomatic.

Once chronicity ensues, it is believed that the disease may range through incrementally advancing stages of histologically defined chronic hepatitis, progress to cirrhosis and other extrahepatic manifestations and culminate in the development of hepatocellular carcinoma (HCC) (Seeff, 1997:21S). Some authorities believe that these sequentially progressive changes are inevitable and will occur in most chronically infected persons provided they do not die first from another lethal illness. Others believe that only a proportion of infected persons will develop progressive disease. Attention should therefore focus on establishing as early as possible who is likely to show advancing

disease and on attempting to define factors that might be responsible for such progression (Seeff, 1997:21S).

The Committee understands experts in Australia to support the theory that only a proportion of those infected will develop progressive disease. Educational material produced by the Hepatitis C Council, for example, suggests that out of 80 people with chronic Hepatitis C:

- approximately 20 people will not develop symptoms and will remain well;
- approximately 40 people may develop some liver damage and will eventually experience symptoms;
- over 20 years, approximately 20 people will develop cirrhosis. After a further 5-10 years, ten of these people will develop liver cancer or liver failure which may result in a liver transplant (Hepatitis C Council of NSW, 1996).

A number of factors have been identified as possibly promoting progression of HCV-related chronic liver disease. These factors may be viral related, host related or extraneous to either virus or host. They are recorded in Table Two.

TABLE TWO
POSSIBLE FACTORS IN PROMOTING PROGRESSION OF HCV-RELATED CHRONIC LIVER DISEASE

CATEGORY	FACTORS
Virus-Related	Viral Dose Viral Genotype Quasispecies
Host-Related	Age Sex Race
Extraneous	Geography Smoking Environmental Chronic Alcoholism Viral Co-Infection

Source: Seeff, 1997:25S

2.1.4 CLINICAL FEATURES OF THE DISEASE

Common features of chronic Hepatitis C tend to be nonspecific, mild and intermittent. The most frequent symptom is fatigue, variably described as lethargy, malaise, lack of energy or stamina and easy fatiguability (Hoofnagel, 1997:17S). In the study by Sladden *et al* 36% of respondents reported fatigue (1998:509). Wodak and Crofts have described the fatigue experienced as "distressingly common and often precludes employment or home duties" (Wodak and Crofts, 1996:181). One HCV positive person who wrote to the Committee described it as:

> *I always want to sleep. By midday I'm back in bed. I sleep two hours get up for two hours then I'm tired again* (Submission 7),

while another told the Committee in evidence that:

> *fatigue is a terrible thing. I get up, I walk about for a while extremely dizzy and tired. I go back to bed and I try to do something else. I am just not functioning well at all* (Smart evidence, 26 February 1998).

Wodak informed the Committee that perhaps 40% of people with chronic Hepatitis C develop severe fatigue "at some stage in their lives . . . which is a very troublesome fatigue" (Wodak evidence, 2 October 1997). As he elaborated:

> *it means that people are unable to stay at work, they are unable to look after the kids at home or cook meals and so forth. This troubles a lot of people. It is difficult for the medical profession to deal with because it is hard to disentangle who is well, who is malingering, perhaps for social security reasons, and who is fatigued because they are depressed about their illness and who is fatigued because of the illness in the first place. This is going to be a problem that we are going to have to get better at grappling with* (Wodak evidence, 2 October 1997).

Other less frequent symptoms include nausea, poor appetite, muscle aches, arthralgias, feverishness, weakness, and weight loss (Hoofnagle, 1997:17S). Symptoms experienced by participants in the study by Sladden et al include nausea (21%), abdominal pain (21%), loss of appetite (13%), vomiting (6%) and jaundice (3%).

Psychological symptoms may also be experienced. A study on the morbidity of Hepatitis C conducted by Lee *et al* (1997) identified, for example, a high rate of depressive symptoms reported by patients with chronic Hepatitis C. They conclude that

> *fatigue and depression are commonly observed in chronic Hepatitis C and are likely to be codependent variables . . . Appreciation that many patients suffer from depression is particularly relevant because of the low rate of response to interferon therapy and the capability the drug has to induce or worsen depression* (Lee *et al*, 1997:191).

The consequences of these complications are discussed in further detail in Section 4.1 which looks at the impact of Hepatitis C.

2.1.5 GENOTYPES OF THE HEPATITIS C VIRUS

Genotype refers to the genetic constitution of an organism as opposed to the external appearance (or phenotype) of the organism or, the type species of a genus. The NHMRC considers there to be at least six and probably more than nine genotypes of Hepatitis C based on partial or complete genomic sequencing (NHMRC, 1997:1). Rawlinson suggested to the Committee that there are six major genotypes, "many" subtypes, and at least four other types (7 - 11) which may be different to genotypes 1-6 or may be sub-types of genotypes 1-6 (Rawlinson submission).

Each particular genotype of Hepatitis C has the potential to form quasispecies which are variant genomes of the same genotype, arising within a single patient, derived from the original infecting virus. Quasispecies diversity may increase with time and may contribute to interferon resistance (Sherman, 1996:9).

Research is beginning to identify specific characteristics of the various genotypes. For example, the major genotypes show distinct geographic clustering. Farrell informed the Committee that genotype distribution of the virus correlates "very closely with the ethnic background of the patient" (Farrell evidence, 28 November 1997).

HCV types 1 and 2 and their subtypes are distributed virtually worldwide including Europe, North America, Japan and Australasia. HCV type 3 has been reported in Europe, the United States, Thailand, India and Australia, but not Japan (MacDonald *et al*, 1996:138). HCV type 4 appears to be a Pan-African type (the principal genotype in Zaire and Egypt for example [Purcell, 1997:12S]) and type 5 has been found to be the principal genotype in South Africa (MacDonald *et al*, 1997 and Purcell, 1997:12S). Genotype 6 and its many variants have been found principally in Asia (Purcell, 1997:12S), though MacDonald *et al* narrow this to Hong Kong only (MacDonald *et al*, 1996:138).

Studies of HCV genotype distribution in Australia show genotypes 1 (both a and b) and 3 to be predominant (one study, for example, reported 49.5% type 1 and 35% type 3 [Swanson *et al*, 1997:75]). Given Australia's multicultural population, there are small numbers of genotype 4, mainly from Egypt which is otherwise very rare in Australia (Farrell evidence, 28 November 1997), and genotype 6a and 6c, all from South East Asia (McCaw *et al*, 1997).

Patterns of genotype distribution are also being identified according to the means by which people acquired the infection. Genotype 1b has, for example, been found to be more prevalent in those who acquired Hepatitis C through blood transfusions than injecting drug use (Sherman, 1996:9).

As will be discussed in Chapters Seven and Eight genotype is also considered a predictor of response to interferon therapy. Research to date has established that genotypes 1a (Bell *et al*, 1997:234) and 4 (Farrell evidence, 28 November 1997) respond poorly to interferon therapy, whereas patients with the genotype 3 can expect to have a 40 - 50 per cent cure rate with interferon (Farrell evidence, 28 November 1997). In his submission Farrell notes that:

> *there are certain groups in the community, eg those from particular ethnic backgrounds, who are very likely to have genotypes which respond poorly to interferon treatment. At present such patients are denied knowledge of this and thus they may often embark on treatment with less than a ten per cent chance of success*.

Not surprisingly then, Farrell concludes "if I had Hepatitis C I would certainly want to know my genotype" (Farrell evidence, 28 November 1997). The NHMRC however suggested that:

> *the immunological and pathological implications of HCV genotypic differences are as yet only partly understood [as] are the effects of interferon therapy on infection with different genotypes* (NHMRC, 1997:1)

2.1.6 OTHER FORMS OF HEPATITIS

As the Committee heard, there is "an alphabet" of hepatitis now from A to G (Dwyer evidence, 10 October 1997). Table Three compares and contrasts hepatitis A through to E and the features of these various viruses such as modes of transmission, 'at risk' groups, symptoms and treatment.

	Hep A	Hep B	Hep C	Hep D	Hep E
What is it?	A virus that causes inflammation of the liver. Does not lead to chronic disease	A virus that causes inflammation of the liver. Can cause liver cell damage, leading to cirrhosis & cancer of the liver	A virus that causes inflammation of the liver. Can lead to cirrhosis and cancer of the liver	A virus that causes inflammation of the liver. Only infects those with HBV	A virus that causes inflammation of the liver. There is no chronic state.
Incubation period	15-50 days (30 day average)	4-26 weeks (8-12 weeks average)	2-26 weeks (7-9 weeks average)	4-26 weeks	2-9 weeks (40 days average)
How is it spread?	Faecal/oral routs through close person/person contact or ingestion of contaminated food or water	Contact with infected blood, seminal fluid, vaginal secretions. Sex contact, contaminated needles, tattoo/body piercing and other sharps instruments. Infected mother to newborn. Human bite	Contact with infected blood, contaminated needles, razors, tattoo/body piercing and other sharp instruments. Infected mother to new born.	Contact with infected blood and contaminated needles. Sexual contact with HDV infected person	Transmitted through oral/faecal route. Outbreaks associated with contaminated water supply
Symptoms	May have no symptoms. Adults may have light stools, dark urine, fatigue, fever and jaundice	May have no symptoms. Some people may have mild flu-like symptoms, dark urine, light stools, jaundice, fatigue and fever	Same as HBV	Same as HBV	Same as HBV
Treatment of Chronic Disease	Not applicable	Interferon is effective in up to 50% of those treated	Interferon is effective in 10-20% of those treated	Interferon with varying success	Not applicable
Vaccine	Two doses of vaccine to anyone over the age of 2 years	Three doses may be given to persons of any age	None available	None available	None available
Who is at risk?	Household or sexual contact with infected person or living in an area with HAV outbreak. Travellers to developing countries; homosexual men and IV drug users	Infant born to infected mother; those engaging in sexual activity with infected person or multiple partners; injecting drug users; emergency responders and health care workers; homosexual men and haemodialysis patients	Injecting drug users; blood transfusion recipients prior to 1990; health care workers; infants born to infected mothers; patients on haemodialysis.	Injecting drug users; homosexual men and those engaging in sexual activity with an HDV infected person	Travellers to developing countries
Prevention	Immune globulin or vaccination. Personal hygiene.	Vaccination and safe sex. Clean up blood spills; Do not share razors or toothbrushes	Clean up blood spills. Universal precautions. Wear gloves when touching blood. Do not share injecting equipment, razors or toothbrushes	Hep B vaccine to prevent HBV infection. Safe sex	Avoid drinking or using potentially contaminated water

Source: Hepatitis Foundation International website

2.1.7 HIV AND HCV: CONTRASTING EPIDEMICS

The causative link between HIV and AIDS was discovered in 1983, some five years before the discovery of HCV. The two diseases have many parallels and present similar challenge, but also differ in important ways. As Wodak suggested to the Committee in his submission it is "instructive" to compare Hepatitis C and HIV (Wodak submission).

Leeder succinctly identified the common features of these two diseases: both are infectious blood borne diseases; both went unrecognised for a period; both preferentially affect socially marginalised groups; the realisation of how they may impact upon society came slowly; both have been subject to much misinformation and "scaremongering"; both were spread through our collective blood supply and both have "demanded significant changes in social policy" (Leeder, 1997:15). Leeder cautions though that the similarities between diseases can be "beguiling" and "all too easily obscure the major differences" (Leeder, 1997:15). He considers it:

> *time to develop an awareness of Hepatitis C which recognises its uniqueness rather than seeing it as a shadow of AIDS. . . From the perspective of public health, Hepatitis C is a different beast to AIDS and we can't depend on identical strategies to defend it* (Leeder, 1997:15).

Mr Jack Wallace, Executive Officer of the Australian Hepatitis Council, on the other hand, has contrasted the two diseases in the following way:

> *Hepatitis C is not related to HIV, with the exception that they are both blood borne viruses and are transmitted through the sharing of injecting equipment. Hepatitis C generally affects people who inject or who have injected drugs, whose social environment and cohesion are completely different from gay men; Hepatitis C has not received the level or concentration of funding particularly for treatments and research that has been provided to HIV; the treatments and information for people infected with Hepatitis C are vastly different than for people with HIV. In short, the epidemiology, virology, short and long term physical effects, treatments, social environments and consequences of the diseases are different* (Wallace correspondence, 2 September 1998).

- **Modes of Transmission**

As has been discussed, the major mode of transmission of HCV in Australia is blood-to-blood contact, in particular sharing injecting equipment and paraphernalia. Unlike HIV, sexual transmission appears to be very uncommon (as will be discussed in Section 3.8.1) occurring only in conditions of viraemia.

By contrast, Australian HIV transmission occurs principally within the gay community with over 80% of new cases occurring among homosexually active men. The prevalence of HIV among injecting drug users is approximately 2% (Cregan, 1998:5).

The Committee understands Hepatitis C to be a more infectious virus (for blood-to-blood spread) than HIV by a factor of ten (Wodak evidence, 2 October 1997). Both Wodak and Kaldor informed the Committee that the risk of infection following an occupational needle stick injury for a health worker is 0.3% for HIV, 3% for Hepatitis C and 30% for Hepatitis B (depending on the immune response of the infected person) (Wodak submission, Kaldor evidence, 3 October 1997).

- **Natural History**

The natural history of HCV infection is less well defined than that of HIV. HCV seems to be a more slowly progressive disease than HIV with an estimate of 20-30% of people with chronic infection developing advanced liver disease within 20-25 years, and liver cancer developing in a small proportion of these people in the subsequent 5-10 years (Dore and Kaldor, 1996:32). Already chronic HCV is the most common underlying cause of liver disease in Australia for people needing liver transplants (Dore and Kaldor, 1996:32). Even if only one in ten people infected with HCV develop advanced liver disease, the cumulative number of cases will be several times higher than the cumulative number of AIDS cases in Australia.

Wodak informed the Committee that people with HIV get sick in a higher percentage of cases much more quickly than with Hepatitis C. Roughly 50% of people go from HIV to AIDS in about 12 years whereas only 20% of cases of Hepatitis C go on to develop cirrhosis within a 20 year period (Wodak evidence, 2 October 1997). However, as Wodak added:

> *if we multiply the far larger pool size by the small proportion that are getting sick, we nevertheless come to estimates of the number of people who are going to develop life-threatening complications of Hepatitis C which suggest that it is a problem of enormous public health magnitude* (Wodak evidence, 2 October 1997).

- **Population Groups**

Those most at risk of either HIV or HCV are young adults within two groups - homosexually active men and injecting drug users respectively. Both groups are often perceived to be outside mainstream Australian society yet the social positioning of these populations is different. By the early '80s the gay community movement had organisational structures and communication mechanisms in place that could (and did) take up the challenges that the HIV epidemic presented.

In contrast to the gay community, injecting drug users have not developed as a distinct community. In its submission to this inquiry, the National Centre for HIV Social Research noted this distinction:

> *the affected communities are very dissimilar. . . The gay community is relatively cohesive in comparison with those at major risk from HCV. Injecting drug users are more widely diffused, geographically and socially, and have so far been far less ready than gay men to form a coherent community* (National Centre for HIV Social Research submission).

In addition, many injecting drug users have also spent time in prison which could have contributed to the spread of HCV due to the difficulty in obtaining clean injecting equipment.

- **Incidence and Prevalence Rates**

As has been discussed, it is estimated that up to 150,000-200,000 Australians have Hepatitis C with the current rate of infection in the range of 8,000 - 10,000 new cases per year; about 10 to 20 times the respective estimates for the HIV epidemic (Dore and Kaldor, 1996:30). Wodak noted in his submission that

> *There are approximately ten times as many old (prevalent) and ten times as many new (incident) cases of Hepatitis C in Australia than HIV. However, a very high proportion of individuals with HIV infection develop life threatening complications after ten or twelve years . . . A smaller proportion of a far larger sized pool develop severe complications of Hepatitis C* (Wodak submission).

By the time the Hepatitis C epidemic was identified the baseline prevalence rate was 80% compared with a baseline prevalence for HIV of only 0.5% (Wodak evidence, 2 October 1997):

> *We went into this [HIV] epidemic with very low rates of HIV infection among our injecting drug users in this country. In other countries it was different. In the United States, for example, in some parts such as New York City, when we first became aware of AIDS . . . 50% of their drug injectors were already infected. It has taken them a long time to pare that back; in fact, they are still struggling. In this country HIV entered the drug injecting population much later. By then we already had tests available and baseline levels were very low* (Wodak evidence, 2 October 1997).

It is generally acknowledged that widespread access to clean injecting equipment introduced in the late '80s allowed Australia to keep HIV prevalence among injecting drug users to 1-2% (one of the lowest in the world). As long as the extent of HIV

infection in this population has remained low, occasional needle sharing among users has not led to significant spread of HIV. This is attributed to the 1:50 to 1:100 chance of being exposed to an injecting drug user with HIV. This ratio increases to 1:2 with respect to HCV.

- **Community (and political) Response**

Australia's response to the HIV epidemic has been described as "prompt, innovative and courageous" (Wodak evidence, 2 October 1997). Wodak attributes that to the multi-party political support:

> *I cannot help but contrast the national and international reaction to Hepatitis C with the . . . reaction to HIV. In this country, we are very aware of the benefits of that tremendous response. Whenever I talk in public about Australia's reaction to HIV I always emphasis that one of the reasons why Australia is now in the fortunate position to have controlled that epidemic is really because in the early years of the epidemic Australia managed to have a bipartisan approach or a multi-party approach* (Wodak evidence, 2 October 1997).

Wodak elaborated further:

> *It was an approach in which the state and commonwealth boundaries, for once, did not trip us up and those complexities were contained. I cannot emphasise enough the importance of that . . . I do not think we will be able to develop a response commensurate with the magnitude of this epidemic unless we follow the same principles of developing and strengthening the multi-party political approach and also work out a way for the Commonwealth and the states to work together rather than in competition* (Wodak evidence, 2 October 1997).

Kaldor also compares the response to HIV to that of HCV from the perspective of planning at the national level and research. He noted that:

> *With HIV we still have a national strategy which has allocated to it a clear and significant component of research funding that is tied into the strategic objectives of the national strategy. With Hepatitis C we do not really have a national strategy. Hepatitis C has been attached to the National HIV Strategy under the framework of related diseases and it is still being discussed* (Kaldor evidence, 3 October 1997).

He went on further to comment that:

> *At this stage it is not quite clear what HIV strategic moneys are allowed to be allocated for Hepatitis C. That argument is unfortunate because it*

leads to what might be called 'turf battles' between different diseases. That is unfortunate when all that is required is a consolidated effort to address these very important health problems (Kaldor evidence, 3 October 1997).

- **Conclusion**

In their comparison of the HIV and HCV epidemics, Dore and Kaldor summarise the two diseases in tabular form which has been reproduced in Table Four.

TABLE FOUR
SUMMARY OF HIV AND HCV

	HIV	HCV
Start of epidemic	early 1980s	early 1970s
Peak of new infections	1984-1985	unknown
Estimated cumulative infections	15,000-20,000	150,000 - 200,000
Prevalence infection	0.1%	1.0%
Male:female ratio	17.0:1.0	1.7:1.0
Estimated new infections/year	500	8,000 - 10,000
Cumulative attributable deaths	5000	unknown

Source: Dore and Kaldor, 1996:30

2.2 EPIDEMIOLOGY

In many respects, Hepatitis C is still a relatively new disease. This is reflected in a number of ways including the limited availability of accurate prevalence and incidence estimates on the number of people with the virus. Wodak, for example informed Members that estimates of the numbers of HCV+ people are still "fairly inexact" and that there is "still some imprecision" about the exact prevalence and incidence figures (Wodak evidence, 2 October 1997).

ANCARD's submission to review of the Highly Specialised Drugs Program considered surveillance of HCV infections in Australia to be by no means as advanced as that of HIV and in need of "greater attention". As the submission noted, "in estimates of incidence and prevalence, only a very broad-brush approach is possible" (ANCARD submission to the review of the Highly Specialised Drugs Program, attachment to their submission to this Inquiry).

In its submission to the Inquiry, NSW Health cited six reasons for the lack of accurate prevalence and incidence data. These reasons included:

1. no population prevalence surveys have been done;

2. only a minority of people become ill when they first acquire the virus so infection often goes unnoticed, untested and unnotified;

3. available antibody tests cannot discriminate between a person with a new infection and one with a longstanding disease;

4. many people are ignorant of being infected due to the long latent period before symptoms commence;

5. available incidence estimates come from surveys of high risk populations who have been serially tested (eg prisoners and clients of agencies such as the Kirketon Road Centre which targets Kings Cross sex workers and at-risk youth); and

6. blood donor data probably underestimates population prevalence because the risk behaviour declaration screens out those most likely to be infected (NSW Health submission).

In addition, routine diagnostic antibody tests for Hepatitis C have only been available since 1990. Successive generations of tests have resulted in improved sensitivity and specificity with the result that comparisons of prevalence estimates over time have to be made cautiously. In addition, not only do antibody tests not discriminate between new infections and long standing ones as NSW Health suggested in Point 3 above, but the tests do not provide information on infectiousness or viral load.

During the last few months of this Inquiry the Hepatitis C Virus Projections Working Party released its report, *Estimates and Projections of the Hepatitis C Virus Epidemic in Australia* (1998). The Hepatitis C Virus Projections Working Group was formed under the auspices of, and reports to, the Australian National Council on AIDS and Related Diseases (ANCARD) Hepatitis C Sub-committee. Membership of the Working Group included clinicians, epidemiologists, statisticians, mathematical modellers, health economists and representatives from the Commonwealth, State and Territory Health Departments and the Australian Hepatitis Council. The objectives of the Working Group were:

- to provide consensus estimates of HCV incidence and prevalence in Australia;

- to obtain projections of the long-term sequelae of HCV infection;

- to identify gaps in research and surveillance relevant to these projections;

- to recommend a mechanism for updating and improving estimates over time; and

- to recommend mechanisms for monitoring the incidence and prevalence of complications of chronic HCV infections (such as cirrhosis, hepatocellular carcinoma, death) (HCV Projections Working Group, 1998:2).

The estimates and projections proposed by the Working Group are included in the following discussion.

2.2.1 PREVALENCE OF HEPATITIS C

Prevalence refers to the total number of people in a population who have the disease at a given point in time, or as Kaldor informed Committee Members, it is the "pool of infection that already exists" (Kaldor evidence, 3 October 1997). Prevalence depends on two factors: the incidence and the duration of the disease.

Direct estimates of HCV prevalence for the Australian population as a whole are difficult to calculate because studies of HCV prevalence have been conducted in specific populations (such as injecting drug users or prisoners), none of which may be considered to be representative (Hepatitis C Virus Projections Working Group, 1998:5).

An estimated 1-2% of the world population is chronically infected with HCV (Schering-Plough submission).

- **Prevalence of Hepatitis C in Australia**

There appears to be general consensus that the prevalence of the disease is, as of 30 June 1996, 150,000 - 200,000. These figures were given in evidence by Professor Batey (evidence, 27 October 1997), and in submissions made by Professor Wodak and organisations such as the Hepatitis C Council, and NUAA. Prevalence has also been cited as being between 0.5-1.0% of the total Australian population which equates to one in every 100 Australians carrying the virus (Hepatitis C Council of NSW, 1996:5).

The Hepatitis C Virus Projections Working Group estimated the prevalence of Hepatitis C in 1995 to be 170,000 - 195,000 with a lower limit of 130,000 and an upper limit of 230,000 (1998:6). Using modelled patterns, the Group's preferred estimate of the number of prevalent infections at the end of 1997 was 196,000 (with a lower limit of 149,000 and an upper limit of 234,000) (1998:8).

However, despite this apparent agreement on the disease's prevalence, it is generally recognised that accurate statistics are not available. Professor Farrell, for example, has observed that:

> *We do not even have accurate statistics on the real prevalence of Hepatitis C in the general Australian community . . . No studies have been done into the prevalence of the virus in the wider community. The current estimate of 0.5 - 1.0% is probably an underestimate as it is based on the results of routine blood donors. Community prevalence studies in France and the USA have found infection rates two to five times higher than this* (Farrell, 1997).

Similarly, Dr Kaldor acknowledges that:

> *there has been virtually no prevalence survey done of a group that might be considered representative of the wider population. The only group that has been regularly surveyed is blood donors and they are by no means representative because a wide range of factors have to be excluded for one to be a blood donor. The prevalence among regular blood donors is now around one in 1,000 so it is well down below these levels . . . So we have no really good idea about the different demographic clustering or patterns of Hepatitis C* (Kaldor evidence, 3 October 1997).

Clearly there is a need for monitoring of the prevalence of Hepatitis C on an ongoing basis to, as it were, keep a finger on the pulse of the epidemic. While the Hepatitis C Virus Projections Working Group's study is an initial attempt to get a consensus on the magnitude of the Hepatitis C epidemic, it is not the definitive study. The Committee therefore firmly believes further prevalence studies are required at both the national and state level.

RECOMMENDATION 1:

That the Minister for Health urge his Federal counterpart to commission population **prevalence** studies of Hepatitis C at the national level to determine the prevalence of Hepatitis C in the general Australian community. The Committee further recommends that such studies be conducted on a regular and ongoing basis.

The prevalence of Hepatitis C has been estimated among various specific population groups in Australia including injecting drug users, prisoners and recipients of blood. These studies will be reviewed in Sections 3.1, 3.2 and 3.3 respectively.

Despite the lack of prevalence surveys, certain characteristics are becoming evident. It was suggested to the Committee that the prevalence of Hepatitis C is, for example, specific in terms of age and ethnicity. Farrell, for example, identified Hepatitis C as being common in Arabic, Italian, and South-east Asian communities (Farrell evidence, 28 November 1997). In giving evidence to the Committee he cited research from Italy suggesting that the prevalence of Hepatitis C amongst younger people (30 years and

below) is between 1 - 1.4 per cent while the prevalence for those aged 40 years and over ranges from 12 - 20 per cent (Farrell evidence, 28 November 1997). He considers Australian prevalence rates amongst Italian communities to reflect a similar trend. He concludes that:

> *something happened 40 or 50 years ago, or longer, that gave those people Hepatitis C and it is almost certainly inappropriate medical behaviours and particularly use of glass syringes and non-disposable needles* (Farrell evidence, 28 November 1997).

In both his submission and during the course of evidence, McCaughan also noted national differences. He observed that the epidemiology of the infection in Australia and the US is "totally different" to the epidemiology in Italy and Japan (McCaughan evidence, 23 March 1998). While many HCV cohorts in Australia are in their early forties (reflecting injecting drug use many years ago) in Italy and Japan the average of the infected cohort is 70 years. As McCaughan observed, that cohort comes from iatrogenic use of glass syringes and non-disposable needles 30 to 40 years ago (McCaughan evidence, 23 March 1998).

Kaldor, in evidence before the Committee, identified certain demographic clusterings of Hepatitis C based on anecdotal observations rather than empirical evidence. He spoke of high representations of people from some Middle Eastern and Vietnamese backgrounds with advanced stages of Hepatitis C as evidenced in those attending liver clinics with severe liver damage (Kaldor evidence, 3 October 1997).

- **Prevalence of Hepatitis C in NSW**

Given the lack of general population prevalence studies, the Committee did not receive any indication of prevalence estimates for the state. Information provided referred rather to notification rates which are reviewed in Section 2.2.4. The Committee considers it essential that NSW conduct ongoing population prevalence studies of the Hepatitis C epidemic at the state level. Such studies are to be in addition to the national prevalence studies proposed in Recommendation 1. The Committee considers it important that studies be conducted at both the national and state level to provide as accurate and complete a picture of Hepatitis C as is possible. It is not anticipated that the studies will duplicate each other, but rather provide different levels of detail all of which will be necessary to appreciate more fully the scope of the disease.

RECOMMENDATION 2:

That the Minister for Health commission population **prevalence** studies of Hepatitis C at state level to determine the prevalence of Hepatitis C in New South Wales. The Committee further recommends that such studies be conducted on a regular and ongoing basis.

2.2.2 INCIDENCE OF HEPATITIS C

Incidence refers to the number of **new** cases of a disease within a population over a period of time and reflects only the rate of disease occurrence. Incidence is expressed in terms of XX per 100 person years.

While prevalence studies indicate the cumulative impact of a disease, only incidence studies can identify where infection is currently occurring and allow the effectiveness of current prevention strategies to be assessed (van Beek *et al*, 1998). In appearing before the Committee, Kaldor stressed the importance of knowing incidence rates:

> *Incidence is crucial . . . if you are thinking about prevention. If you do not know what is going on with incidence you do not know what is happening about your prevention efforts* (Kaldor evidence, 3 October 1997).

- **Incidence of Hepatitis C in Australia**

Up until the release of the Hepatitis C Virus Projections Working Group's report in August 1998, there was general consensus amongst the research literature, evidence taken and submissions received that the incidence of Hepatitis C in Australia is 8,000 -10,000 new infections per year. It would appear that this estimate was originally proposed by Crofts, Hopper *et al* in a 1993 study drawing upon estimates from preliminary results of their longitudinal study of injecting drug use (the Victorian Injecting Drug Use Cohort Study, VICS).

While the Crofts, Hopper *et al* estimate referred to the incidence of Hepatitis C amongst injecting drug users it does seem to have become a generally recognised "benchmark" statistic and was cited by the NHMRC (1997:2). It was also suggested to the Committee in evidence given by representatives from NSW Health (Wilson evidence, 3 October 1997) and in the submission made by Hepatitis C Council.

The Hepatitis C Virus Projections Working Group estimated HCV incidence in 1997 to be 11,000 with a lower limit of 8,500 and an upper limit of 13,500 (1998:8). These different figures were appropriately described by Dwyer as "rubbery" (Dwyer evidence, 10 October 1997).

Batey, Crofts and Wodak all suggested to the Committee that there are 6,000 - 8,000 new cases per annum (Batey evidence, 27 October 1997; Crofts evidence, 28 November 1997; Wodak evidence, 2 October 1997). In his submission, Wodak explained the difference between the figures 6,000-8,000 and 8,000-10,000:

> *there are probably 8,000 to 10,000 cases of acute Hepatitis C infection each year and about 80% of these go on to develop chronic infection; hence the estimate of 6,000 to 8,000 new cases of chronic infection each* year (Wodak submission).

These current incidence estimates equate to approximately one new case of Hepatitis C in Australia per hour (Wodak evidence, 2 October 1997). Given such a statistic, ANCARD considers there to be "no sign that incidence is slowing" (ANCARD submission to Highly Specialised Drugs Program - attachment to submission). The NHMRC considers trends in Hepatitis C incidence to be "impossible" to discern (1997:2).

As with estimates of prevalence, the Committee considers there to be scope for incidence studies to be conducted at the national level and proposes the Minister for Health approach his federal counterpart urging such studies to be undertaken.

RECOMMENDATION 3:

That the Minister for Health urge his Federal counterpart to commission population **incidence** studies of Hepatitis C at the national level to determine the incidence of Hepatitis C in the general Australian community. The Committee further recommends that such studies be conducted on a regular and ongoing basis.

- **Incidence of Hepatitis C in New South Wales**

Evidence presented to the Committee on New South Wales' share of the national rate of Hepatitis C was limited and varied. In evidence before the Committee, NSW Health suggested, for example, that:

> *the NSW share of this [national incidence rate] is a guesstimate but it could be as high as 40 per cent to 50 per cent annually, given the profile of the high-risk groups* (Wilson evidence, 3 October 1997).

Wodak estimated that at least one-third of all national cases reside in New South Wales (Wodak submission).

The only regional incidence estimates presented to the Committee came from the submission from the A.W. Morrow Gastroenterology and Liver Centre, based at Royal

Prince Alfred Hospital. That submission suggested there are at least 250 new HCV infections each year in the Central Sydney Area Health Service.

The fact that no expert could, with any degree of accuracy, inform the Committee of the incidence of this disease in the state is indicative of the limited data available on this disease. Such a situation has major implications for planning and funding of health and community services and, as Kaldor has identified, assessment of preventative strategies.

RECOMMENDATION 4:

That the Minister for Health commission population **incidence** studies of Hepatitis C at state level to determine the incidence of Hepatitis C in New South Wales. The Committee further recommends that such studies be conducted on a regular and ongoing basis.

2.2.3 ESTIMATES OF THE LONG-TERM SEQUELAE OF HEPATITIS C INFECTION

As was discussed in Section 2.1.3 approximately 80-85% of those who come in contact with the Hepatitis C virus and do not clear it within the first few months go on to develop chronic Hepatitis C which may manifest itself in a number of ways, the most extreme form being hepatocellular cancer and ultimately liver failure. It is therefore important to know not only the incidence and prevalence of HCV infection, but the prevalence and incidence of those people living with the disease.

Kaldor suggested to the Committee that it is important to think not just about the incidence and prevalence of the virus, but the incidence and prevalence of the Hepatitis C illness. As he noted:

> *up to now most of the activity and monitoring has gone to . . . try to see where the virus itself is, but what impacts on people is the illness that is caused by the virus . . . we have not concentrated much effort to really find out what Hepatitis C is doing in the population as far as death rates go* (Kaldor evidence, 3 October 1997).

He went on to tell Members that:

> *when one looks at the pattern of illness, this is where we have limited information . . . As far as I am aware, there has been no comprehensive effort so far to put together a picture of the incidence of Hepatitis C-related cirrhosis and Hepatitis C liver cancer, or indeed, the prevalence or these conditions in people living with Hepatitis C. They are the most severe manifestations and probably the easiest ones to monitor. It is*

> *even harder to monitor the broader symptoms such as fatigue and other less severe symptoms that are associated with the infection. There is still a great deal of work required, similarly with mortality. I do not think anyone has tried to sort through deaths to assess what proportion are really Hepatitis C related to come up with an estimate of what the mortality of this condition is in Australia* (Kaldor evidence, 3 October 1997).

Since Kaldor appeared before the Committee the Hepatitis C Virus Projections Working Group has released its report. The estimates and projections contained in this report go some way to addressing Kaldor's concerns regarding the incidence and prevalence of, for example, Hepatitis C related cirrhosis, liver cancer and deaths.

Building upon the prevalence figures for the Hepatitis C virus discussed in Section 2.2.2, the Working Party has calculated that, based on direct estimates of the prevalence of HCV antibody, there were probably 130,000 to 145,000 people living with chronic HCV in 1995 (lower limit 100,000; upper limit, 175,000) (Hepatitis C Virus Projections Working Group, 1998:9). Based on modelled estimates, the Group estimated that there were 147,000 people living with chronic HCV in 1997 (lower limit of 112,000; upper limit of 176,000) (Hepatitis C Virus Projections Working Group, 1998:9).

Taken together, the Working Group concludes that these estimates and projections suggest that in Australia in 1997 there were around 190,000 people living with antibody to HCV (140,000 to 240,000). Of these people it was estimated that:

- 47,000 (35,000 to 60,000) had cleared their HCV infection;
- 134,000 (101,000 to 176,000) were living with chronic HCV infection and therefore at risk of developing cirrhosis;
- 8,500 (4,000 to 13,000) were living with HCV-related cirrhosis, the majority probably asymptomatic and undiagnosed;
- 80 (40 to 130) people developed HCV-related hepatocellular carcinoma (HCC) during 1997; and
- to the end of 1997, 3,000 (1,450 to 4,550) had died prematurely as a result of their HCV infection (Hepatitis C Virus Projections Working Group, 1998:10).

The Working Group also made projections for estimated incidence of both cirrhosis and hepatocellular carcinoma due to HCV infection through to 2010. These data are presented in Figures One and Two below.

FIGURE ONE
ESTIMATED NUMBER OF PEOPLE LIVING WITH CIRRHOSIS DUE TO HCV INFECTION
1980 TO 2010

FIGURE TWO
ESTIMATED INCIDENCE OF HEPATOCELLULAR CARCINOMA DUE TO HCV INFECTION 1980 TO 2010

Brown and Crofts recognised the need for prospective data on the prevalence and incidence of HCV-related sequelae (Brown and Crofts, 1998:388). Until such data are available, they argue, a full appreciation of the impact Hepatitis C will have upon the community as a whole and specifically the economic impact of Hepatitis C cannot be made.

Fully aware of the limited data available on the incidence and prevalence of Hepatitis C related diseases (such as cirrhosis and HCC) and deaths, the ANCARD Hepatitis C Sub-committee has identified clinical based morbidity registers and studies and monitoring of hepatocellular carcinoma incidence and associations as research priorities (Hepatitis C Virus Projections Working Group, 1998:32). The Committee appreciates the value to be gained from introducing such measures and therefore fully supports these priorities. Such measures go some way to addressing the current shortfalls in data identified by Brown and Crofts.

RECOMMENDATION 5:

That the Minister for Health urge his federal counterpart to establish systems to monitor Hepatitis C related cirrhosis and hepatocellular carcinoma at the national level.

RECOMMENDATION 6:

That NSW Health establish systems to monitor Hepatitis C related cirrhosis and hepatocellular carcinoma in New South Wales.

RECOMMENDATION 7:

That at the next Australian Health Ministers' Council the Minister for Health urge his federal, state and territory counterparts to establish clinical based morbidity registers to monitor Hepatitis C deaths.

RECOMMENDATION 8:

That NSW Health establish a clinically based morbidity register to monitor Hepatitis C related deaths in New South Wales.

2.2.4 NOTIFICATION OF HEPATITIS C

In addition to the limited prevalence and incidence data that are available, Departments of Health at both the state/territory and federal levels collect data on the numbers of Hepatitis C cases reported. The Committee is reporting these data separate to incidence and prevalence data as it believes notification data are not, as yet, a complete and true reflection of Hepatitis C cases, merely the results of those tested.

Hepatitis C became a notifiable disease in all Australian states and territories in 1990. From that time, it rapidly emerged as the most frequently notified infectious disease in the nation.

During the course of giving evidence to the Committee, Kaldor informed Committee Members that a secondary way of assessing incidence is to look at acute infections as recorded in notification data. He noted that, if done systematically, notification data should give a national picture of incidence (Kaldor evidence, 3 October 1997). He identified two problems:

> *It is believed that only about five per cent of people who acquire Hepatitis C get that illness: there is nothing to pick up in 95 per cent of people so*

> *that has to be multiplied by a factor of 20. Even if they do get the illness, it is not necessarily picked up as Hepatitis C by doctors and does not necessarily get reported along the public health reporting chain to health departments* (Kaldor evidence, 3 October 1997).

Kaldor concluded that:

> *it is an ideal system in theory but in practice it does not produce the incidence figures we would like to see for retracking what is going on with incidence at a population level* . . . (Kaldor evidence, 3 October 1997).

In using notification data, Kaldor has further cautioned that:

> *national reporting of Hepatitis C diagnoses provides a somewhat incomplete picture of the occurrence of HCV infection in Australia. Because of variable patterns of testing for HCV antibody, it is not possible to estimate population prevalence of HCV exposure from the diagnosed cases. Similarly, although national case reporting now requires the separate reporting of newly acquired or acute Hepatitis C infections, it is likely that only a small proportion of such cases will actually be recognised and reported. Furthermore, national case reporting has only provided limited details on the modes of HCV transmission in Australia, and no indication at all of the extent of illness and mortality caused by HCV infection* (Kaldor, 1997:47).

Associate Professor McCaughan made similar comments in his submission to the Committee. In it he noted that:

> *unfortunately, current notification data does not allow differentiation between incident or prevalent cases. As this crucial information and other epidemiological information is currently unknown, estimates have to be made to identify the extent of Hepatitis C infection in the community* (McCaughan submission).

The Hepatitis C Council commented that there is "huge room for improvement" in the standardisation of notification data (Loveday evidence, 30 March 1998). As Mr Loveday explained to Members:

> *we had the situation of national reporting being unavoidably confused by the fact that the States and Territories report differently to the Commonwealth Health Department. All States and Territories other than NSW and South Australia report non-specific notifications. These are Hepatitis C diagnoses and the person who has been tested might have contracted Hepatitis C 10 or 25 years ago. Those are called non-specific*

> *notifications or unspecified notifications. South Australia and NSW report only incident cases, which are new cases* (Loveday evidence, 30 March 1998).

As a result of these different reporting practices the total number of known Hepatitis C cases in Australia is "underplayed" (Loveday evidence, 30 March 1998).

The NHMRC notes that, because of chronicity of HCV infection and the asymptomatic nature of most acute infections, the notification system reflects changing trends in testing patterns and distinguishes "poorly" between prevalence and incident cases (NHMRC, 1997:2).

- **Notification of Hepatitis C in Australia**

Each state and territory collects surveillance data on Hepatitis C. These data are collated nationally each fortnight by the National Notifiable Diseases Surveillance System (NNDSS) under the auspices of the Communicable Diseases Network of Australia and New Zealand (CDNANZ) and published in *Communicable Diseases Intelligence* (the notification publication of the Commonwealth Department of Health and Family Services).

Between 1991 and 1997 over 57,000 diagnoses of HCV infection from all states and territories (excluding New South Wales and South Australia) were reported to the NNDSS maintained by the Commonwealth Department of Health and Family Services (Hepatitis C Virus Projections Working Group, 1998:2). A further 46,900 HCV diagnoses were reported in NSW and 7,500 in South Australia making a total of over 110,000 HCV diagnoses in Australia to the end of 1997 (Hepatitis C Virus Projections Working Group, 1998:2). NSW and South Australia are the only two states that base the data they provide to the NNDSS on confirmed recent infections rather than all diagnosed cases (some of which may be infections contracted years ago).

Notification data obtained from the National Centre for Disease Control (a unit of the Commonwealth Department of Health and Family Services) and broken down by state/territory and year has been obtained by the Hepatitis C Council and are reproduced in Table Five. The data are for 1993 to 1996 only and therefore not as complete as the data reported by the Hepatitis C Virus Projections Working Group.

TABLE FIVE
HEPATITIS C NOTIFICATIONS IN AUSTRALIA, 1993-1996

STATE/TERRITORY	1993	1994	1995	1996	TOTAL	TOTAL %
NSW	6,722	9,357	8,393	9,294	33,766	44%
Victoria	2,659	3,523	4,506	4,597	15,285	20%
Queensland	3,049	3,177	2,920	2,884	12,030	16%
South Australia	1,912	2,281	1,215	1,201	6,609	9%
Western Australia	1,176	1,416	1,268	1,230	5,090	7%
ACT	285	428	330	270	1,313	2%
Northern Territory	212	301	309	217	1,039	1%
Tasmania	161	53	268	291	773	1%
TOTAL	16,176	20,536	19,209	19,984	75,905	100%

Source: Hepatitis C Council of NSW, 1997

The Council advised that the following qualifications apply to the data:

- some notifications will represent the second or third time someone has had an HCV antibody test; and

- some notifications may represent false positive test results, although most of these would have occurred prior to 1992 when the HCV antibody tests were less sensitive and specific.

The Council cautions these figures must be considered as approximates, but because the margin of error (estimated to be 5%) applies equally across all states, comparisons can be made.

Given the different reporting mechanisms of New South Wales and South Australia and the remainder of the country, the Committee is not convinced that the quality of available notification data is as high as it could be. The Hepatitis C Council commented on the current situation in its submission:

> *the current situation of inconsistent reporting is highly unsatisfactory. There would be a natural tendency on the part of a reader who is not fully informed of all the facts to take, at face value, total national figures as*

> *reported in national surveillance publications. Figures published in such reports thus exclude the state with the largest number of HCV notifications in Australia. National figures reporting is thus highly misleading* (Hepatitis C Council supplementary submission).

The Committee can see little benefit in the current mechanism and wishes to see it rectified.

The Committee understands that earlier this year NSW Health contacted the National Centre for Disease Control and, in the "interests of national uniformity", requested that data on all NSW Hepatitis C notifications be published in *Communicable Diseases Intelligence* (O'Donoughue correspondence, 2 April 1998). However, as of August 1998 the publication still has NSW and SA unspecified cases as being "not notifiable". The confusion continues despite attempts by NSW Health to rectify the situation.

The 1997 Hepatitis C NHMRC report recognised current problems in the surveillance of Hepatitis C due to, amongst other factors, variance in surveillance methodology between the different jurisdictions. The report called for uniform data collection for Hepatitis C to improve Hepatitis C surveillance and recommended that States and Territories address this issue "as a priority" (NHMRC, 1997:31). The Hepatitis C Council's submission called upon this Committee to make a strong recommendation for consistency in national reporting of notification statistics (Hepatitis C Council supplementary submission).

The Committee fully supports both the NHMRC recommendation and the Hepatitis C Council's request. It therefore wishes to see action be taken at the federal level to ensure all states and territories standardise their reporting of Hepatitis C notifications.

RECOMMENDATION 9:

That the Minister for Health urge his Federal counterpart to institute standardised procedures for the notification of Hepatitis C across all states and territories of Australia.

- **Notification of Hepatitis C in NSW**

As Table Five shows, NSW has almost one-half (44%) of the Hepatitis C cases reported nationally in the period 1993-96. Notification data from 1991 to 1997 are provided in Table Six according to Area Health Services.

TABLE SIX
HCV NOTIFICATIONS ACCORDING TO NSW AREA HEALTH SERVICE

AREA HEALTH SERVICE	HCV NOTIFICATIONS CUMULATIVE TO 1997[1]	
	Number	% New South Wales
South Eastern Sydney	8,190	18.0
Western Sydney	4,646	10.2
South West Sydney	5,349	11.7
Central Sydney	6,088	13.4
Northern Sydney	3,416	7.5
Central Coast	1,852	4.1
Hunter	3,044	6.7
Wentworth	1,458	3.2
Illawarra	2,226	4.9
Northern Rivers	3,314	7.3
Mid North Coast	1,410	3.1
Mid Western	1,259	2.8
Southern	1,113	2.4
New England	808	1.8
Greater Murray	963	2.1
Macquarie	328	0.7
Far West	105	0.2
New South Wales	**45,569**	**100.0**

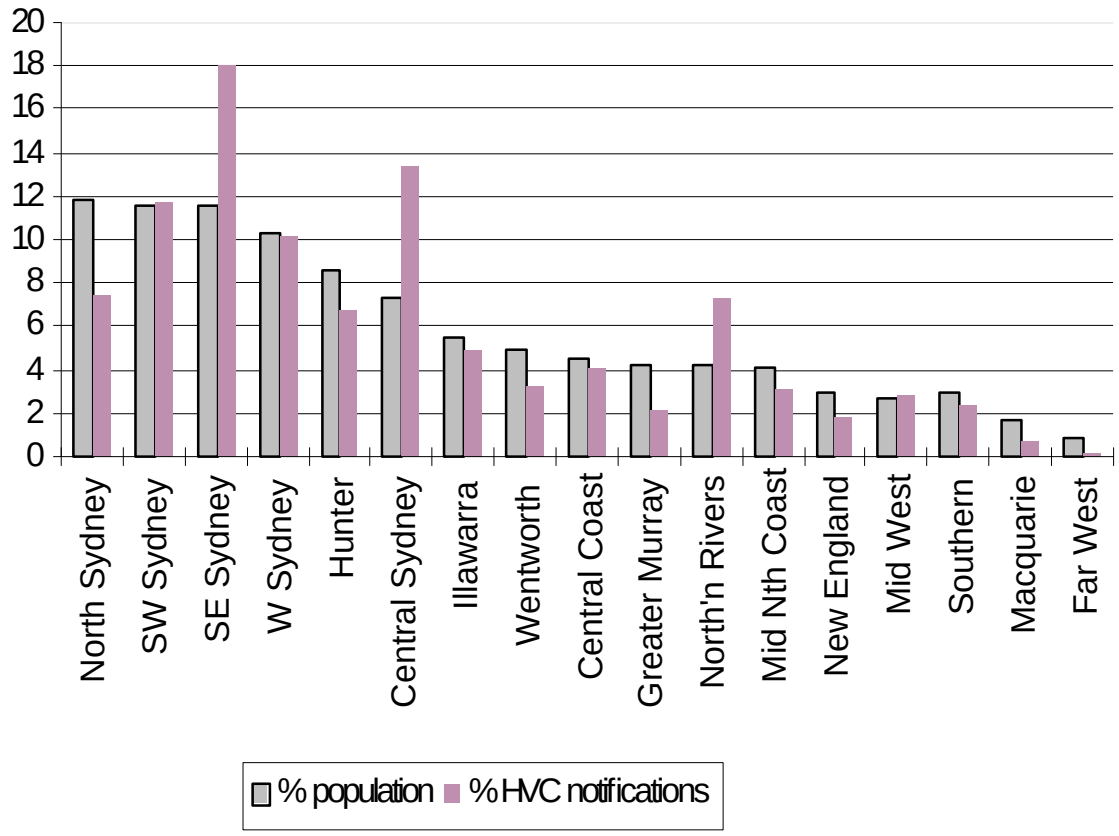
[1] Hepatitis C notifications is the cumulative number of positive Hepatitis C antibody tests notified to the Health Department since testing commenced in 1991

Source: NSW Parliament, 1998

As Table Six shows, the five Area Health Services with the highest notification rates are South Eastern Sydney (18%); Central Sydney (13.4%); South Western Sydney (11.7%); Western Sydney (10.2%); and Northern Sydney (7.5%).

In giving evidence before the Committee, the Hepatitis C Council reformatted the data presented in Table Six to demonstrate notifications as they relate to each Area Health Service's population. These data are reported in Figure Three below.

FIGURE THREE
AREA HEALTH SERVICE POPULATION (%) AND HCV ANTIBODY POSITIVE NOTIFICATIONS (%) 1991 - 1997



The Area Health Services where Hepatitis C notifications outstrip population are South Eastern Sydney, Central Sydney and Northern Rivers. While these data are based on notifications only, these regions clearly have specific service delivery needs that will be discussed later in the report.

In commenting on these data Mr Loveday noted that:

> *you would normally expect Hepatitis C being so widespread throughout the general community that it would be pretty much a 50-50 basis in proportion with the total population, but there are three areas where this is wildly out. The biggest is South Eastern Sydney, which has a much bigger share of notifications, Central Sydney and Northern Rivers and the converse of this is that Northern Sydney, which has a much higher general population as a pro rata basis to the total, has a smaller share. This is the first indication we have seen as a result of these figures . . . that could possibly guide the provision of resource allocation* (Loveday evidence, 30 March 1998).

Studies analysing the notification rates at the regional level are limited. As far as the Committee could ascertain, the only published work that teases out official notification data at the regional level appears to be that done by Sladden (based at the Northern Rivers Institute of Health and Research) and colleagues. Over the past five years, an average of approximately 550 cases have been reported in that region (Sladden evidence, 30 March 1998). Sladden *et al* (1997:290) have calculated the rate of Hepatitis C notification in the north coast to be 201 per 100,000 residents. They note that such a rate is double the state's rate of 103 per 100,000 and nearly three times the Australian average (74/100,000). In evidence Sladden informed the Committee that he has:

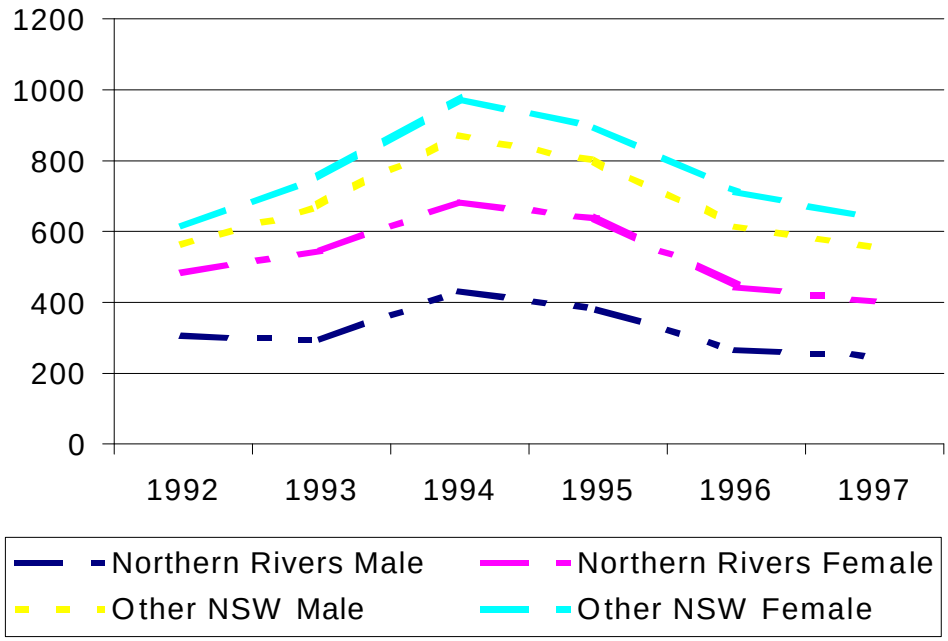
> *estimated that about 0.2 per cent of the population in the Northern Rivers area is infected. That is about one in 500 people, as opposed to about 0.1 per cent in the New South Wales population as a whole. So it is about double the state average* (Sladden evidence, 30 March 1998).

Data provided to the Committee by Sladden demonstrate the notification rates for males and females in the Northern Rivers area as compared to other NSW residents over time (1992-1997). In commenting on the data to the Committee, Sladden noted that:

> *the rates appear to be coming down over the last three years but they remain above the New South Wales average. Now they are about 50 per cent above. We do not have a long enough trend to say whether this is a fluctuation or a continued downward trend* (Sladden evidence, 30 March 1998).

With regard to the 1994 peak, Sladden considers it could be "just a testing artifact" due to a lot of people getting tested that year for the first time (Sladden evidence, 30 March 1998). These data are recorded in Figure Four.

FIGURE FOUR
HEPATITIS C STANDARDISED NOTIFICATION RATES PER 100,000
NORTHER RIVERS AND OTHER NSW RESIDENTS, 1992-1997



Source: tabled material, Sladden evidence, 30 March 1998

Sladden described the Hepatitis C situation in the Northern Rivers region as a "double whammy" in that:

> *a lot of previous injectors have retired and are no longer injecting, but they still have Hepatitis C. We also have a high rate of current injecting drug use, with the second highest provision of needles in any area of the state. So there is a lot of continued drug use and a lot of current infection going on* (Sladden evidence, 30 March 1998).

2.2.5 CONCLUSION

Given available data, what then can be said about the incidence and prevalence of Hepatitis C in Australia? The Committee feels it can say with a certain degree of confidence that:

- in Australia one person each hour of each day contracts the Hepatitis C virus which, in New South Wales equates to one person every three hours (Wodak evidence, 2 October 1997). This adds up to 8,000 - 10,000 new Hepatitis C cases each year. Of this number 6,000 - 8,000 will go on to develop chronic Hepatitis C;

- approximately one in every 100 Australians has the Hepatitis C virus with up to 200,000 cases of Hepatitis C in Australia;

- 90,000 of these cases have been tested (Batey evidence, 27 October 1997) of whom approximately 76,000 tested positive.

The discrepancy between the estimated 150,000 - 200,000 people with Hepatitis C and the actual number of reported cases (76,000) is of great concern. The discrepancy highlights the number of people who remain undiagnosed. As the Hepatitis C Council observed:

> *this may be because of a lack of public awareness about HCV and limited medical practitioner knowledge. It is also likely that a number of people have not yet developed symptoms and have not yet presented for medical assistance* (Hepatitis C Council submission).

The issues of limited public awareness and limited medical practitioner knowledge will be discussed further in Sections 10.6 and 8.3.3 respectively.

The second discrepancy of concern to the Committee relates to the figures proposed by the Hepatitis C Virus Projections Working Group. The concerns do not relate to the quality of work produced by the Working Group, but rather the large range given in upper and lower limits for both incidence and prevalence data. The Working Group, for example, suggested the incidence of Hepatitis C in 1997 to be 11,000 with lower and upper limits of 8,500 and 13,500 respectively. A range in the order of 100,000 was suggested for Hepatitis C prevalence with the lower range proposed as being 130,000 and an upper range of 230,000. Such a range is very broad and a fair reflection of the uncertainties in the point estimates (Law correspondence, 29 October 1998).

The discussion above has also demonstrated to Members current inadequacies in the appreciation of governments at both the federal and state/territory level to the epidemiology of Hepatitis C. As Farrell admitted to the Committee:

> *I regard the knowledge about epidemiology and the investment of government in understanding epidemiology to be totally inadequate* (Farrell evidence, 28 November 1997).

The Committee trusts that the recommendations it has forwarded will go some way to redressing the current inadequacies on the part of government to understand and appreciate the epidemiology of the Hepatitis C virus.

2.3 CONCLUSION

The discussion in this chapter has sought to provide an aetiological and epidemiology context for the rest of the report. Various features of the Hepatitis C virus have been identified and discussed along with a thorough analysis of the virus' incidence and prevalence in Australia, and where possible, New South Wales. Evidence received highlighted difficulties in ascertaining, with any degree of accuracy, current rates of Hepatitis C. The Committee sees a need for regular and ongoing prevalence and incidence studies to be conducted at both the state and national level and has recommended accordingly.