HIV, HEPATITIS B AND HEPATITIS C

DMD3 DENT5311

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LEARNING OUTCOMES

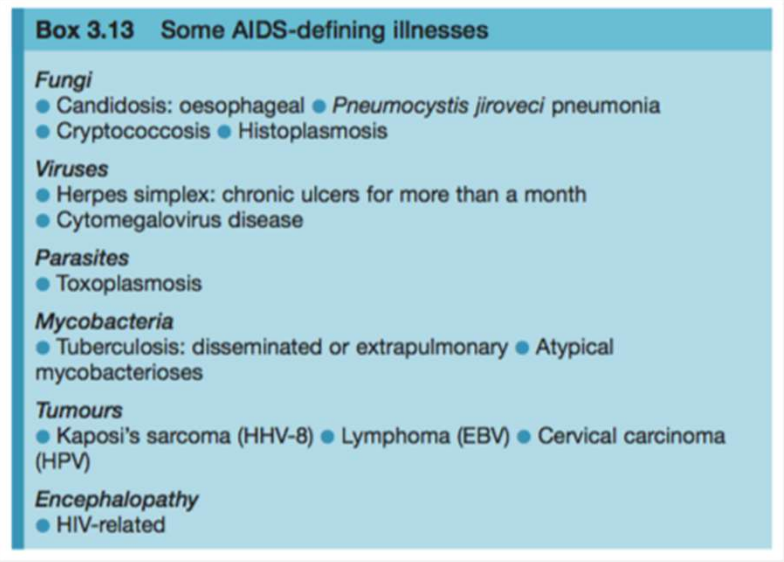
- 1. Outline key clinical features and disease progression of blood-borne viruses (HIV, Hepatitis B, Hepatitis C)
- 2. Appreciate the epidemiology of BBVs over time
- 3. Understand the evolution of BBV treatment over time
- 4. Describe BBV-related oral disease, and side-effects of medications
- 5. Understand BBV-transmission, risks and infection control
- 6. Discuss professional obligations under the Australian national guidelines for the management of healthcare workers living with BBV and healthcare workers who perform exposure prone procedures at risk of exposure to blood borne viruses

HIV AND VIRAL HEPATITIS

HUMAN IMMUNODEFICIENCY VIRUS AND ACQUIRED IMMUNODEFICIENCY SYNDROME

- **HIV Infection**: Infection with human immunodeficiency virus (HIV), a retrovirus, causes HIV disease and ultimately damages certain T lymphocytes, producing the acquired immune deficiency syndrome (AIDS).

- **AIDS** is a lethal infection, and the final and most advanced stage of infection caused by HIV. Defined as [1] HIV infection plus [2] one or more AIDS-defining (opportunistic) illnesses, and [3] a CD4 T-lymphocyte count <200 × 10^6/L (or a CD4+ T-cell percentage of total lymphocytes of <14%)



Box 3.13 Some AIDS-defining illnesses

Fungi
- Candidosis: oesophageal
- *Pneumocystis jiroveci* pneumonia
- Cryptococcosis
- Histoplasmosis

Viruses
- Herpes simplex: chronic ulcers for more than a month
- Cytomegalovirus disease

Parasites
- Toxoplasmosis

Mycobacteria
- Tuberculosis: disseminated or extrapulmonary
- Atypical mycobacterioses

Tumours
- Kaposi's sarcoma (HHV-8)
- Lymphoma (EBV)
- Cervical carcinoma (HPV)

Encephalopathy
- HIV-related

HUMAN IMMUNODEFICIENCY VIRUS AND ACQUIRED IMMUNODEFICIENCY SYNDROME

- Two main types of HIV:
  - **HIV-1** = most common. Prevalent throughout the world and responsible for the global HIV pandemic
  - **HIV-2** = geographic distribution much more limited, found primarily in West Africa. Less transmissible, slower disease progression
- Both infect cells expressing CD4 surface receptors, mainly the T-helper lymphocytes, and replicate within them, damaging them.

HUMAN IMMUNODEFICIENCY VIRUS AND ACQUIRED IMMUNODEFICIENCY SYNDROME

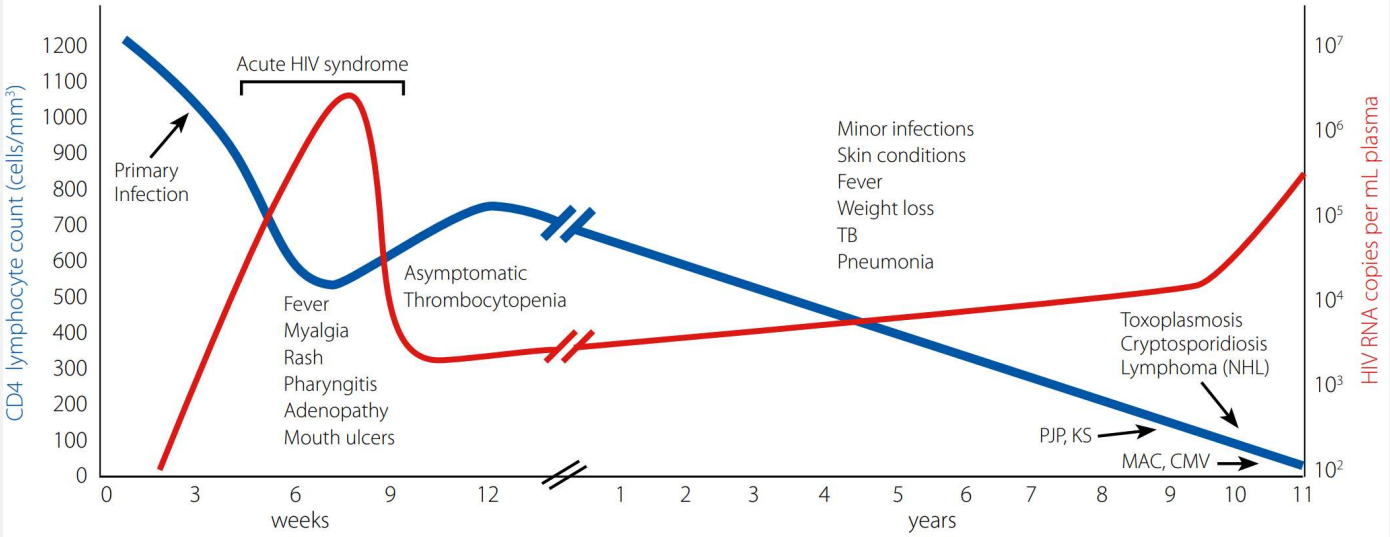
- Based on clinical features patients are distributed in three categories:
  - A: asymptomatic, acute infection or persistent generalised lymphadenopathy: >500 × 10^6/L
  - B: symptomatic, no acute or chronic conditions: 200–500 × 10^6/L
  - C: AIDS-defining illnesses: < 200 × 10^6/L
    - → vulnerable to opportunistic infections

Australian Centre for Disease Control | Australian Immunisation Handbook

Level of immunocompromise	Infants aged <12 months, $CD4^+$ count	Children aged 1–5 years, $CD4^+$ count	Adults and children aged ≥6 years, $CD4^+$ count
Not immunocompromised	≥1500 cells/μL	≥1000 cells/μL	≥500 cells/μL
Moderately immunocompromised	750–1499 cells/μL	500–999 cells/μL	200–499 cells/μL
Severely immunocompromised	<750 cells/μL	<500 cells/μL	<200 cells/μL

HUMAN IMMUNODEFICIENCY VIRUS AND ACQUIRED IMMUNODEFICIENCY SYNDROME

The various stages of untreated HIV infection depicting the development of different opportunistic infections with advanced immunodeficiency.



mac: mycobacterium avium complex, **cmv:** cytomegalovirus, **ks:** kaposi sarcoma, **pjp:** pneumocystis jirovecii pneumonia, **tb:** tuberculosis

CD4+ Lymphocyte Count (Blue Line)
HIV RNA copies (Red Line)

- If untreated, progression to AIDS, occurs a ***median of 10 years*** after initial infection with HIV
- Moderately immunocompromised = **CD4 count <500/µL**
- Severely immunocompromised (AIDS) = **CD4 count <200/µL**

HIV – COMORBID CONDITIONS

- HIV/AIDS-defining cancers
  - Common
    - Kaposi sarcoma (can occur orally)
    - Non-Hodgkin lymphoma (can occur orally)
    - Cervical cancer
  - Less common
    - HPV oropharyngeal cancers, liver, lung, colon, recumt, anus, testes, skin



Diffuse large B-cell lymphoma (NHL)

Corti, M., Villafañe, M.F., Solari, R. *et al.* Non-Hodgkin Lymphomas of the Oral Cavity in AIDS Patients in a Reference Hospital of Infectious Diseases in Argentina: Report of Eleven Cases and Review of the Literature. *J Gastrointest Canc* **42**, 143–148 (2011).

HIV – COMORBID CONDITIONS

- HIV-associated neurocognitive disorders (HAND)
  - 3-level spectrum of cognitive, motor and/or mood problems
  1. Asymptomatic neurocognitive impairment (ANI)
  2. Mild neurocognitive disorder (MND)
  3. HIV-Associated dementia (HAD)

- Cardiovascular disease (~50% increased risk)
- Haematological disease: anaemia, thromobocytopenia, neutropenia
- Opportunistic or co-infections

VIRAL HEPATITIS

- **Hepatitis**: term for liver inflammation, and can result form a wide range of insults, from infections to drugs and poisons.
  - Features: jaundice, pale/fatty faces, itching, nausea, fatigue, weight loss, bleeding tendency, upper abdominal discomfort, fever

- **Viral hepatitis:** In healthcare, refers to infection by hepatitis B, C or D, which are transmitted parenterally.
  - Hepatitis B and C are the most relevant to health care, since delivery of health care has the potential to transmit viral hepatitis to both HCWs and patients.

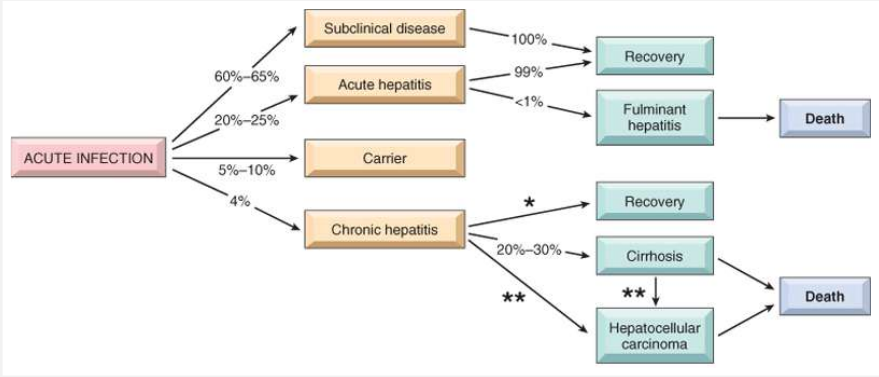




HEPATITIS B

- **Definition**: Inflammation of the liver caused by the parenteral infection with hepatitis B virus.
- The virus is spread through blood contact, and can spread from infected mother to child during birth.

- **Prognosis**: Depends on age when come into contact with hepatitis B. Can get an acute or chronic illness
  - Acute: may feel symptomatic for a short time, then recover. Some with acute hepatitis B naturally clear the virus.
  - Chronic: have virus for life. May not feel unwell, but over time can damage the liver. 6 month wait to find out whether acute hepatitis B becomes chronic.



Age (when you first come into contact with the virus)	**Acute hepatitis B**	**Chronic hepatitis B**
Infants under one year old	Most will not get any acute hep B signs	90% will get chronic hepatitis B if not given injection at birth and vaccinations
Children aged one to six years old	Most will not get any acute hepatitis B signs	30% will get chronic hepatitis B
Adults or children over six years old	Many will get acute hepatitis B signs	Less than 5% will get chronic hepatitis B

HEPATITIS B

CLINICAL FEATURES

- Malaise
- Weight loss
- Fatigue
- Upper abdominal discomfort
- Jaundice
- Pale/fatty faeces
- Bleeding tendency
- *Chronic liver disease in 5%

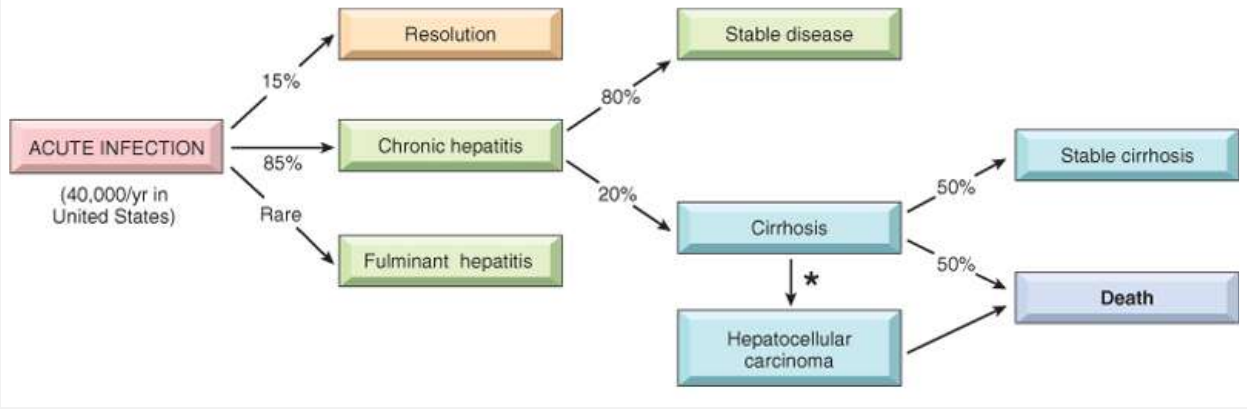
EXTRA-HEPATIC CLINICAL FEATURES

- Serum sickness-like syndrome
- Polyarteritis nodosa
- Glomerulonephritis
- Polyarthritis
- Non-Hodgkin lymphoma
- Cryoglobulinemic vasculitis
- Dermatological signs: Gianotti-Crosti syndrome (papular acrodermatitis), urticaria

HEPATITIS C

- **Definition**: Inflammation of the liver caused by the parenteral infection with hepatitis C virus. The virus can cause both acute and chronic hepatitis, ranging in severity from a mild illness to a serious, lifelong illness including slow progressive liver cirrhosis and cancer.

- **Prognosis**: Long-term impact of infection is highly variable (without treatment)
  - 15-25% will spontaneously clear virus without treatment
  - 75-85% will develop chronic hepatitis C infection (10-20% develop liver cirrhosis, 2-5% hepatocellular carcinoma)



HEPATITIS C

CLINICAL FEATURES

- Malaise
- Weight loss
- Fatigue
- Upper abdominal discomfort
- Jaundice
- Pale/fatty faeces
- Bleeding tendency
- *Chronic liver disease in >70%

EXTRA-HEPATIC CLINICAL FEATURES

- Aplastic anaemia
- Thyroid disease
- Polyarteritis nodosa
- Glomerulonephritis
- Peripheral neuropathy
- Autoimmune disease
- Sialadenitis
- Liver cancer

LIVER CIRRHOSIS

- *See GIT/Hepatology lecture*
- **Compensated liver cirrhosis** = stable phase of the disease, with good long-term prognosis if the cause of cirrhosis is treated.
- **Decompensated liver cirrhosis** =
  - associated with high mortality
  - defined clinically by the presence of complications: ascites, hepatic encephalopathy, variceal haemorrhage or nonobstructive jaundice.
- Patients may move between decompensated and compensated disease states, depending on response to treatments, acute events and disease progression.
- **Liver failure (end-stage cirrhosis)** = impairment of the synthetic or metabolic functions of the liver.

EPIDEMIOLOGY

MODES OF TRANSMISSION AND AT-RISK POPULATIONS

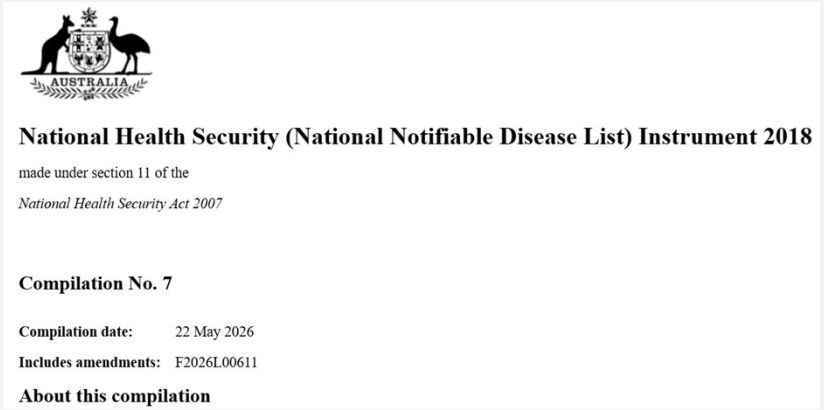
- **HIV** = blood, semen / vaginal /rectal fluids (sexual contact)
  - Injecting drug use
  - Vertically from mother to child
  - Men who have sex with men

- **Hep B** = blood, semen / vagina / rectal fluids (sexual contact).
  - Vertical transmission from mother to child at birth

- **Hep C** = blood
  - Strongly associated with risky injecting behaviour
  - Blood transfusion or transplant, or blood product for clotting disorder, before 1993 (Hep C)
  - Long-term renal dialysis

EPIDEMIOLOGY

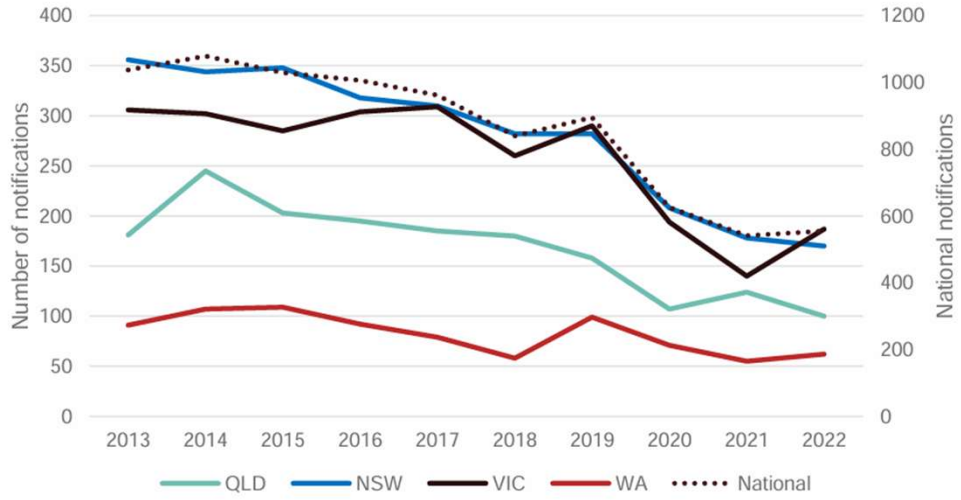




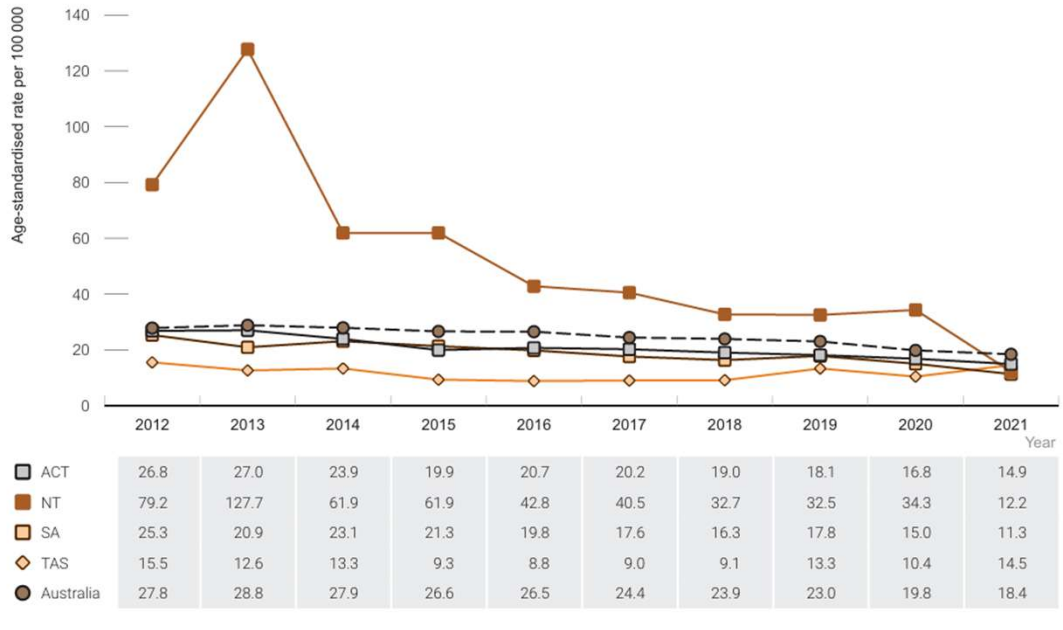
UNSW Kirby Institute

HIV/AIDS

Figure 1: HIV notifications by jurisdiction, 2013 – 2022



HEPATITIS B



	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
ACT	26.8	27.0	23.9	19.9	20.7	20.2	19.0	18.1	16.8	14.9
NT	79.2	127.7	61.9	61.9	42.8	40.5	32.7	32.5	34.3	12.2
SA	25.3	20.9	23.1	21.3	19.8	17.6	16.3	17.8	15.0	11.3
TAS	15.5	12.6	13.3	9.3	8.8	9.0	9.1	13.3	10.4	14.5
Australia	27.8	28.8	27.9	26.6	26.5	24.4	23.9	23.0	19.8	18.4

Source: National Notifiable Diseases Surveillance System

Most people living with chronic hepatitis B in Australia were born overseas and acquired hepatitis B at birth or in early childhood.

Downward trend likely related to:

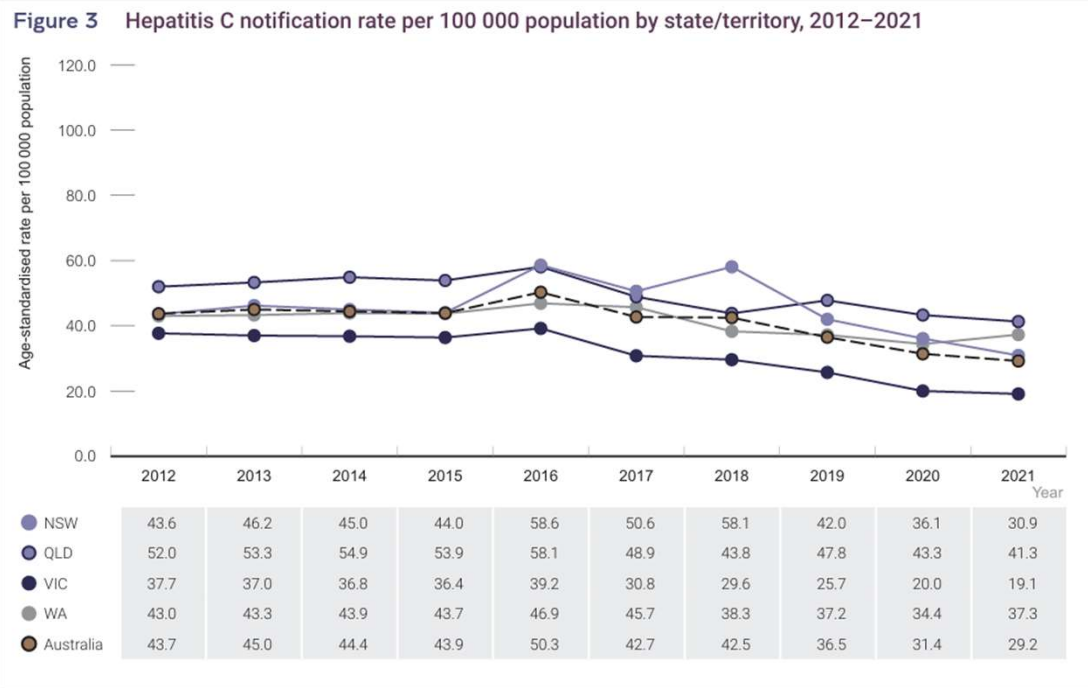
- Vaccination programs being introduced in countries that many Australian migrants emigrate from
- Maternal screening and vaccination of infants born to women with Hep B

Notification rate among ATSI peoples was more than twice as high compared with the non-Indigenous population

- Reflective of higher prevalence of chronic Hep B among ATSI peoples

https://www.kirby.unsw.edu.au/sites/default/files/documents/Annual-Surveillance-Report-2022_HBV.pdf

HEPATITIS C



	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
NSW	43.6	46.2	45.0	44.0	58.6	50.6	58.1	42.0	36.1	30.9
QLD	52.0	53.3	54.9	53.9	58.1	48.9	43.8	47.8	43.3	41.3
VIC	37.7	37.0	36.8	36.4	39.2	30.8	29.6	25.7	20.0	19.1
WA	43.0	43.3	43.9	43.7	46.9	45.7	38.3	37.2	34.4	37.3
Australia	43.7	45.0	44.4	43.9	50.3	42.7	42.5	36.5	31.4	29.2

https://www.kirby.unsw.edu.au/sites/default/files/documents/Annual-Surveillance-Report-2022_HCV.pdfv

Uptake of hepatitis C treatment is resulting in a population level decline of hepatitis C transmission.

- In March 2016, DAA regimens became subsidised by the Pharmaceutical Benefits Scheme (PBS) for hepatitis C treatment.
- Uptake of DAA therapies was initially high but has since steeply declined.

No declines in hepatitis C notifications amongst ATSI peoples

- suggests inequity in the availability of DAAs as well as resources directed toward harm reduction.
- Hepatitis C notification rates remain several-fold higher among ATSI people compared to non-Indigenous people.

TREATMENT OVER TIME

HIV

- **No cure** for HIV infection
- Several anti-retroviral drugs available, though costly and often associated with resistance and/or adverse reactions
  - nucleoside reverse transcriptase inhibitors (NRTI), non-nucleoside reverse-transcriptase inhibitors (NNRTI), protease inhibitors (PI), integrase inhibitors, entry inhibitors
  - Multiple drugs required → high pill-burden → poor-compliance
- Advent of specific anti-retroviral agent combinations 1996 = highly active anti-retroviral therapy (HAART)
  - Especially increased antiviral activity
  - Reduced pill-burden → improved compliance
- Usually takes ***12-24 weeks of therapy for viral suppression*** below limits of detection

HIV

- **Combination class agents:**
- Atripla = Efavirenz, emtricitabine, tenofovir
- Eviplera = rilpivirine, emtricitabine, tenofovir
- Stribild = elvitegravir, cobicistat, tenofovir, emtricitabine
- Triumeq = abacavir, dolutegravir, lamivudine
- Biktarvy = bictegravir, emtricitabine, tenofovir

Box 20.1 *Main antiretroviral agents*

1. Reverse transcriptase inhibitors

Nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs)

- Abacavir (ABC)
- Didanosine (ddI)
- Emtricitabine (FTC)
- Lamivudine (3TC)
- Stavudine (d4T)
- Tenofovir (TDF)
- Zidovudine (AZT, ZDV)

Non-nucleotide reverse transcriptase inhibitors (NNRTIs)

- Delavirdine (DLV)
- Efavirenz (EFV)
- Etravirine (ETR)
- Nevirapine (NVP)
- Rilpivirine (RPV)

2. Protease inhibitors (PIs)

- Atazanavir (ATV)
- Darunavir (DRV)
- Fosamprenavir (FPV)
- Indinavir (IDV)
- Lopinavir (LPV)
- Nelfinavir (NFV)
- Ritonavir (RTV)
- Lopinavir/ritonavir (LPV/r)
- Saquinavir (SQV)
- Tipranavir (TPV)
- Amprenavir (APV)

3. Integrase inhibitor

Raltegravir (RAL)

4. Entry inhibitor

Fusion inhibitors

- Enfuvirtide (ENF, T-20)

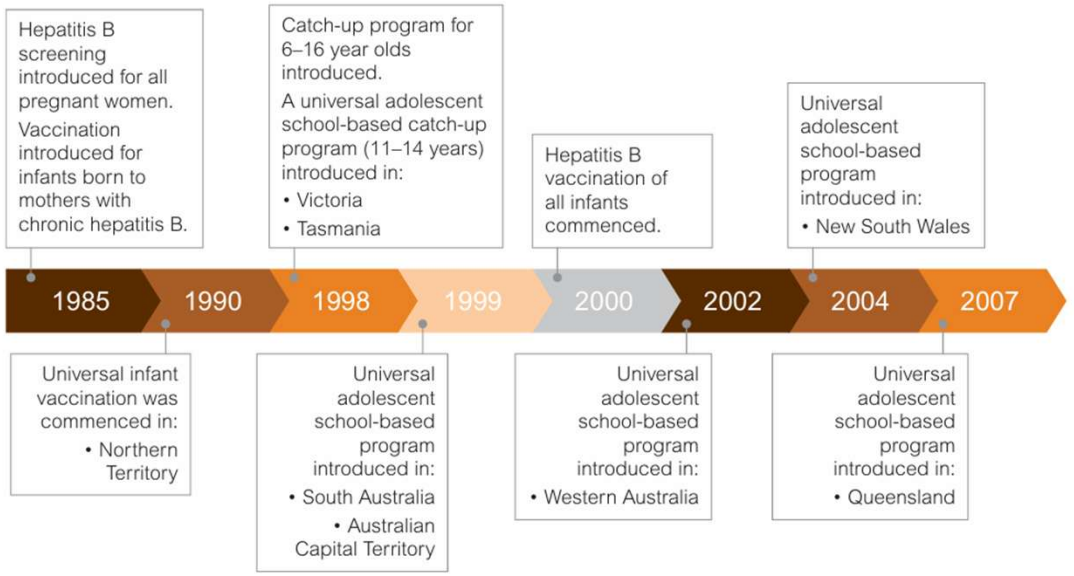
CCR5 antagonist

- Maraviroc (MVC)

HEPATITIS B

- Effective immunisation available as part of the childhood schedule

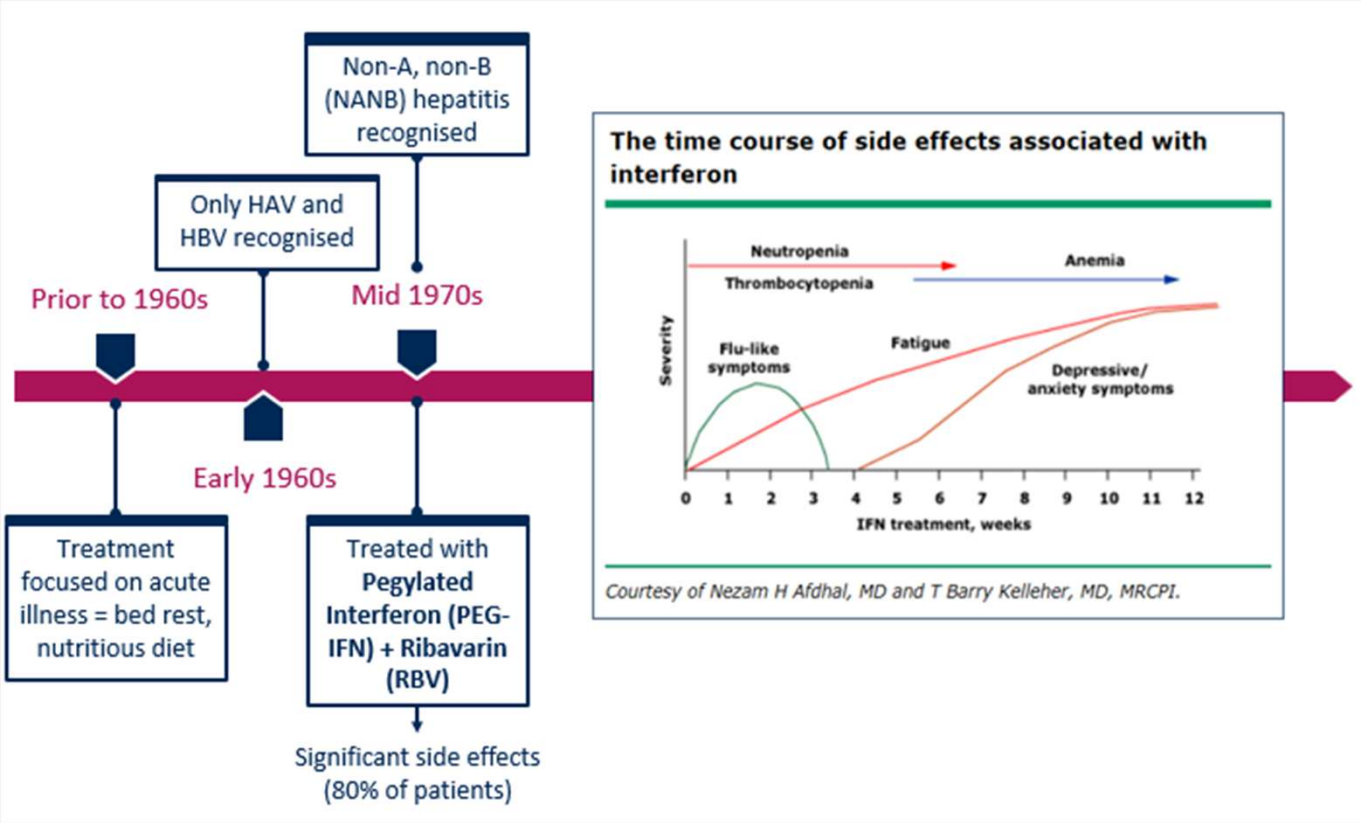
Figure 10 Roll-out of hepatitis B vaccination in Australia, by year



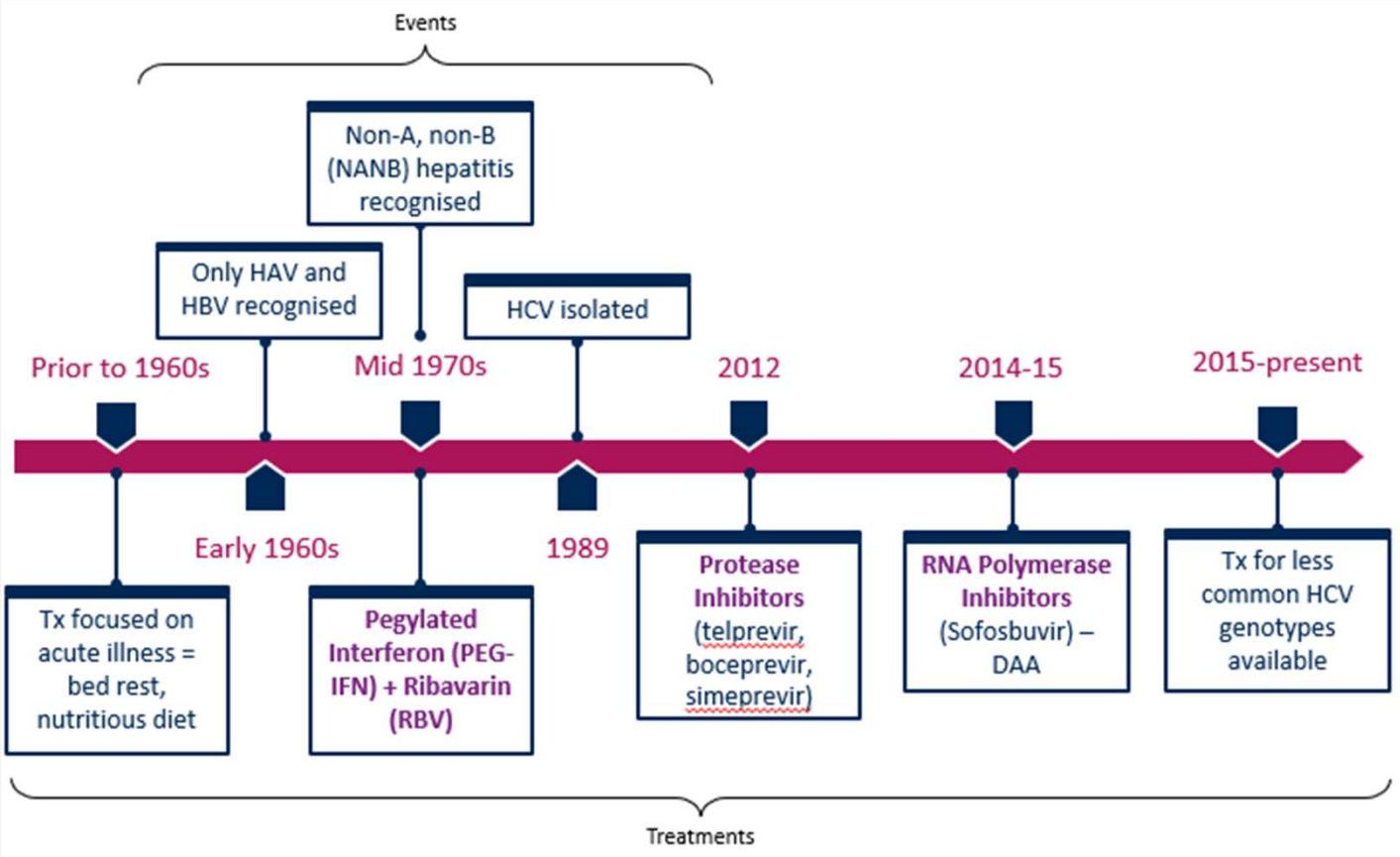
HEPATITIS B

- BUT **no cure** for hepatitis B
  - Persists in infected hepatocytes as covalently closed circular DNA (cccDNA)
- Currently, there is no evidence of benefit to support treating everyone who lives with hepatitis B.
- Focus of acute hepatitis B: supportive care (e.g. rest, proper nutrition, hydration) and monitoring liver function
- Medications help an individual live well with the virus, but are not a cure.
  - Usually only need to take medications if signs of cirrhosis or ongoing liver damage (even if have chronic hepatitis B)
  - Reduce likelihood of liver disease or hepatocellular carcinoma
  - Need to be taken daily

HEPATITIS C


Non-A, non-B (NANB) hepatitis recognised
Only HAV and HBV recognised
Prior to 1960s
Mid 1970s
Early 1960s
Treatment focused on acute illness = bed rest, nutritious diet
Treated with Pegylated Interferon (PEG-IFN) + Ribavarin (RBV)
Significant side effects (80% of patients)
The time course of side effects associated with interferon
Neutropenia
Thrombocytopenia
Anemia
Severity
Flu-like symptoms
Fatigue
Depressive/ anxiety symptoms
0 1 2 3 4 5 6 7 8 9 10 11 12
IFN treatment, weeks
Courtesy of Nezam H Afdhal, MD and T Barry Kelleher, MD, MRCPI.

HEPATITIS C



Interferon-free breakthrough: 2014

Possible to cure a severe, life-threatening chronic infection disease within 8-12 weeks in nearly all patients!

HEPATITIS C

- **General Management**:
  - Lifestyle advice (tobacco and alcohol cessation, high CHO diet, maintaining health body weight and diet, advise on sexual transmission)
  - Bed rest
  - Offer hepatitis A and B immunisations
  - Antiviral drugs
    - Traditionally PEG-IFN and ribavirin
    - Now include direct acting antivirals (DAAs - protease inhibitors and RNA polymerase inhibitors)
  - Liver transplant → corticosteroid or immunosuppressive therapy

SUMMARY – TREATMENTS OVER TIME

HIV	Hep B	Hep C
• Treatment provided earlier • Less side effects from treatment • Reduced pill burden → increased compliance • People living well with HIV (stable, managed)	• Fewer people diagnosed due to childhood immunisation schedule • Not much progress with treatment options • No cure	• Much improved side-effect profile from treatment • Only 2-3 months of treatment (c.f. 6-12 months) • Aim of treatment: achieve SVR (sustained virologic response) • Good success rate of HCV cure

BBV ORAL DISEASE AND MEDICATION SIDE-EFFECTS

HIV – ORAL MANIFESTATIONS

Group 1 lesions strongly associated with HIV infection	Group 2 lesions less commonly associated with HIV infection	Group 3 lesions seen in HIV infection
Candidosis • Erythematous • Pseudomembranous	Bacterial infections • Mycobacterium avium-intracellulare • Mycobacterium tuberculosis	Bacterial infections • Actinomyces israelii • Escherichia coli • Klebsiella pneumonia
Hairy leukoplakia	Melanotic hyperpigmentation	Cat-scratch disease
Kaposi's sarcoma	Necrotizing (ulcerative) stomatitis (Figure 5)	Drug-reactions • Ulcerative • erythema multiforme • lichenoid • toxic epidermolysis
Non-Hodgkin's lymphoma	Salivary gland diseases • Dry mouth due to decreased salivary flow rate • Unilateral or bilateral swelling of major salivary glands	Epithelioid (bacillary) angiomatosis
Periodontal disease • Linear gingival erythema • Necrotizing gingivitis • Necrotizing periodontitis	Thrombocytopenic purpura	Fungal infections other than candida • Cryptococcus neoformans • Geotrichum candidum • Histoplasma capsulatum • Mucoraceae (mucormycosis, zygomycosis) • Aspergillus flavus
	Ulceration NOS (not otherwise specified)	Neurological disturbances • Facial palsy • Trigeminal neuralgia
	Viral infections • Herpes simplex virus • Human papillomavirus lesions ♦ Condyloma acuminatum ♦ Focal epithelial hyperplasia ♦ Verruca vulgaris • Varicella zoster virus ♦ Herpes zoster ♦ Varicella	Viral infections • Cytomegalovirus • Molluscum contagiosum

Leao JC, Ribeiro CM, Carvalho AA, Frezzini C, Porter S. Oral Complications of HIV Disease. Clinics. 2009;64:459-70

HIV – ORAL MANIFESTATIONS



Figure 1 - Severe pseudomembranous candidosis (Thrush) in HIV disease



Figure 2 - Oral hairy leukoplakia – this finding can also be a feature of iatrogenic immunosuppression



Figure 3 - Kaposi's sarcoma of the right buccal vestibule in an undiagnosed HIV-infected patient



Figure 4 - Necrotizing gingivitis in HIV disease



Figure 5 - Necrotic oral mucosal ulceration, possibly associated with CMV in severe HIV disease

Leao JC, Ribeiro CM, Carvalho AA, Frezzini C, Porter S. Oral Complications of HIV Disease. Clinics. 2009;64:459-70

HIV – MEDICATION SIDE EFFECTS

Oral and systemic side effects of antiretroviral drugs (ARDs)

Oral side effect	Class of drug	Name of drug
Erythema multiforme	NRTIs	*Zidovudine (ZVD)Abacavir (ABC)Didanosine (DDI)Zalcitabine*
	NNRTIs	*EfavirenzDelaviridineNevirapineSaquinav*
Hyperpigmentation	NRTIs	*Zidovudine (ZVD)*
Lipodystrophy	NRTIs	*Stavudine*
	PIs	*SaquinavirRitonavir*
Xerostomia	NRTIs	*Lamivudine (3TC)Didanosine*
	PIs	*SaquinavirIndinavirNelfinavirRitonavir*
Parotid lipomatosis	PIs	*IndinavirRitonavirSaquinavirNelfinavirA*
Cheilitis	PI	*Indinavir*
Perioral paresthesia	PIs	*RitonavirAmprenavir*
Taste disturbances	PIs	*IndinabirRitonavir*
Facial edema	PIs	*Ritonavir*
Ulcers	NRTIs	*AbacavirZalcitabineNevirapine*
Lip enlargement	EIs	*Enfuvirtide*

NRTI = nucleoside analog reverse transcriptase inhibitors; **NNRTI** = non-nucleoside analog reverse transcriptase inhibitors; **PIs** = protease inhibitors; **EI** = entry inhibitors

Leao JC, Ribeiro CM, Carvalho AA, Frezzini C, Porter S. Oral Complications of HIV Disease. Clinics. 2009;64:459-70

HIV

ORAL FEATURES

- Uncommon in well-managed and stable patients
- Perioral paraesthesia
  - Rare adverse reacti
- Increased risk of oral disease (immunocompromise)
  - Opportunistic infections
  - Periodontal disease
  - Oral hairy leukoplakia
  - Oral cancer
  - HIV-related salivary gland hypofunction

DENTAL IMPLICATIONS

- Immunocompromised:
  - Caution with proceed with dental treatment if
- Antiretroviral drug interactions
  - Many commonly prescribed drugs (e.g. benzodiazepines can reach high levels toxicity)
  - Consult HIV specialist before prescribing drugs
- Spread
  - Standard precautions
  - Needlestick injury policy

HIV

- ACCESS:
  - Safe, non-judgemental, empathetic environment
  - Many HIV-infected individuals continue to have concerns about privacy.
  - Stigma and social implications of their infection may prevent them from accessing general dental services

- COMMUNICATION / CONSENT / EDUCATION:
  - Maintain medical confidentiality
  - Increased risk of opportunistic infections (e.g. oral thrush), periodontal disease, cancer (Kaposi sarcoma / NHL) → preventive advice, routine recalls
  - HIV-associated neurocognitive disorders (HAND)

HIV

- SURGERY
  - **Pre-operative:** understand disease control
    - Immunocompromised: CD4 <200 cells/mm^3, absolute neutrophil count <$1x10^9$/L
      - Trend is more important than any single count
      - CD4% can be a more stable indication of whether there has been a change in the immune system (12-15% is equivalent to 200 cells/mm^3)
      - ?surgical antibiotic prophylaxis
    - Viral load: if compliant with treatment, should be undetectable
    - Platelet count
  - **Peri-operative:**
    - Some antiviral drugs can enhance benzodiazepine activity
  - **Post-operative:**
    - caution prescribing – drug interactions with antiviral medications

- SPREAD
  - See upcoming slides – standard precautions

HEPATITIS B

ORAL FEATURES

- Possible gingival bleeding, petechiae, ecchymoses
- Icteric patients: Discolouration of posterior palate and FoM along lingual fraenum

DENTAL IMPLICATIONS

- Liver cirrhosis → liver failure *(see GIT Hepatology lecture)*
  - Profound immune compromise associated with end-stage cirrhosis
  - Hepatic encephalopathy
- Bleeding risk (*see Bleeding Disorders lecture)*
- Drug metabolism
  - Dosage
  - Hepatotoxic drugs (avoid sedatives, NSAIDs ; do not exceed max dose of paracetamol)
- Spread
  - Standard precautions
  - Needlestick injury policy

HEPATITIS C

ORAL FEATURES

- Possible gingival bleeding, petechiae, ecchymoses
- Icteric patients: Discolouration of posterior palate and FoM along lingual fraenum
- Sialosis, sialadenitis, hyposalivation, sicca syndrome
- Chronic/untreated Hep C: Association with NHL, lichen planus, periodontal disease

DENTAL IMPLICATIONS

- Liver cirrhosis → liver failure *(see GIT Hepatology lecture)*
  - Profound immune compromise associated with end-stage cirrhosis
  - Hepatic encephalopathy
- Bleeding risk (*see Bleeding Disorders lecture)*
- Drug metabolism
  - Dosage
  - Hepatotoxic drugs (avoid sedatives, NSAIDs ; do not exceed max dose of paracetamol)
- Spread
  - Standard precautions
  - Needlestick injury policy

VIRAL HEPATITIS

- ACCESS:
  - if known to be incubating hepatitis, active clinical hepatitis, or in convalescent stages → defer elective dental treatment where possible until recovery.
  - Incubation/acute phase: hospital environment, emergency care only
  - Decompensated liver disease: hospital environment, emergency care only

- COMMUNICATION / CONSENT / EDUCATION:
  - Hepatic encephalopathy: advanced cirrhosis
  - Preventive dental care important with Hep C due to reduced salivary flow (outcome of major restorative treatment may be poor)

VIRAL HEPATITIS

- SURGICAL
  - Pre-operative:
    - check for jaundice or deranged LFTs – avoid elective dental treatment, consult physician
    - Prothrombin time test and platelet counts
    - ?surgical antibiotic prophylaxis if end-stage cirrhosis (profound immunocompromise)
  - Peri-operative:
    - LA: minimise lignocaine, ideally articaine
    - CS: caution benzodiazepines
  - Post-operative
    - Caution hepatotoxic drugs

- SPREAD
  - See upcoming slides – standard precautions

BBV TRANSMISSION, RISKS AND INFECTION CONTROL

RISK OF TRANSMISSION RATES

- **Hep B** = 1:3
  - positive source to an unvaccinated individual
- **Hep C** = 1:30 (2008)
  - likely less now with effective HCV therapy (closer to 1:200 to 1:50)
  - Range of transmission risk is 0-10% (average 1.8-1.9%)
- **HIV** = 1:300 (2014)
  - likely less now with ART and PEP



PREVENTION

HIV

- No vaccine available
- PrEP

Hep B

- Childhood immunisation schedule

Hep C

- No vaccine available

PREVENTION

- safe and appropriate use of health-care injections and procedures;
- safe handling and disposal of needles and medical waste;
- harm-reduction services for people who inject drugs, such as needle exchange programmes, substance use counselling
- testing of donated blood for the BBVs
- practicing safe sex by using barrier methods such as condoms.



HIV

- PrEP = Pre-Exposure Prophylaxis
  - Daily use of antiretroviral medications by HIV-negative individuals at increased risk of HIV exposure

- PrEP should be considered for all people at risk of HIV, including:
  - Gay, bisexual and men who have sex with men
  - Trans and gender diverse populations
  - Heterosexuals who may have sex with people at a higher risk of HIV, or have partners with an unknown HIV status
  - People who inject drugs
  - People who have arrived from overseas country with high-prevalence of HIV, who may not be connected to health services

HEPATITIS B

Table 1. Serological tests that constitute the diagnostic panel for HBV		
HBsAg	Hepatitis B surface antigen	Active infection (acute or chronic)
Anti-HBs	Hepatitis B surface antibody	Immunity (vaccination or infection)
Anti-HBc	Hepatitis B core antibody	Infection (past or current)



Table 2. Interpretation of HBV serology[3]		
HBsAg Anti-HBc Anti-HBs	Negative Negative Negative	Susceptible (consider vaccination)
HBsAg Anti-HBc Anti-HBs	Negative Positive Positive	Resolved HBV infection
HBsAg Anti-HBc Anti-HBs	Negative Negative Positive	Vaccinated
HBsAg Anti-HBc IgM anti-HBc* (high titre) Anti-HBs	Positive Positive Positive Negative	Acute HBV infection*
HBsAg Anti-HBc IgM anti-HBc* Anti-HBs	Positive Positive Negative Negative	Chronic HBV infection*
HBsAg Anti-HBc Anti-HBs	Negative Positive Negative	Various possibilities (see 'What if the result is inconclusive?')
* Anti-HBc IgM can also be present (usually at a lower titre) in a flare of CHB		

PROFESSIONAL OBLIGATIONS

AHPRA REGISTRATION AND RENEWAL

Exposure Prone Procedures

Before answering these questions, you need to read the Board's Guidelines – Registered health practitioners and students in relation to blood borne viruses. You can access them from the Ahpra renewal page (https://www.ahpra.gov.au/Registration/Renewal.aspx).

These guidelines are for practitioners and students who perform exposure-prone procedures and require you to comply with the Communicable Diseases Network Australia (CDNA) guidelines *Australian national guidelines for the management of healthcare workers living with blood borne viruses and healthcare workers who perform exposure prone procedures at risk of exposure to blood borne viruses*. (https://www.health.gov.au/resources/collections/cdna-national-guidelines-for-healthcare-workers-on-managing-bloodborne-viruses) This includes testing for HIV, Hepatitis C and Hepatitis B at least every three years.

Exposure-prone procedures (EPPs) are procedures where there is a risk of injury to the healthcare worker resulting in exposure of the patient's open tissues to the blood of the healthcare worker. These procedures include those where the healthcare worker's hands (whether gloved or not) may be in contact with sharp instruments, needle tips or sharp tissues (spicules of bone or teeth) inside a patient's open body cavity, wound or confined anatomical space where the hands or fingertips may not be completely visible at all times.

* (required) **Do you perform exposure-prone procedures?**

Yes | No

* (required) **During the preceding period of registration, have you complied with the Australian national guidelines for the management of healthcare workers living with blood borne viruses and healthcare workers who perform exposure prone procedures at risk of exposure to blood borne viruses?**

Yes | No

* (required) **If your registration is renewed, do you intend to perform exposure-prone procedures?**

Yes | No

* (required) **If your registration is renewed, do you commit to comply with the Australian national guidelines for the management of healthcare workers living with blood borne viruses and healthcare workers who perform exposure prone procedures at risk of exposure to blood borne viruses?**

Yes | No

If your circumstances change during the upcoming period of registration and you are no longer able to comply with the CDNA guidelines, you need to stop performing exposure prone procedures.

CDNA NATIONAL GUIDELINES FOR HEALTHCARE WORKERS 2018

These Guidelines provide information and recommendations for all healthcare workers (HCWs), in particular:

- o HCWs who perform exposure prone procedures (EPPs)
- o HCWs living with a blood borne virus (BBV)
- o doctors treating HCWs with a BBV



AUSTRALIAN NATIONAL GUIDELINES FOR THE MANAGEMENT OF HEALTHCARE WORKERS LIVING WITH BLOOD BORNE VIRUSES AND HEALTHCARE WORKERS WHO PERFORM EXPOSURE PRONE PROCEDURES AT RISK OF EXPOSURE TO BLOOD BORNE VIRUSES

RISKS OF TRANSMISSION BETWEEN HCW AND PATIENT

Table 1: Risk of BBV transmission per exposure episode from untreated infected HCW to patient and untreated infected patient to HCW (in the absence of additional risk management).

Blood Borne Virus	Risk of infected HCW to patient transmission	Risk of infected patient to HCW transmission
Hepatitis B virus	0.2% - 13.19%	1% - 62%*
Hepatitis C virus	0.04% - 4.35%	0% - 7%
Human immunodeficiency virus	0.0000024% - 0.000024%	0.3%

* There is a wide variability in infectiousness of people with hepatitis B reported in the literature and this depends on their hepatitis B e-antigen status.

CDNA NATIONAL GUIDELINES FOR HEALTHCARE WORKERS 2018

- **All healthcare workers:**
  - should be encouraged to undertake regular testing for blood borne viruses
  - have the right to access confidential testing, counselling, support and treatment
  - should be vaccinated against hepatitis B virus.

CDNA NATIONAL GUIDELINES FOR HEALTHCARE WORKERS 2018

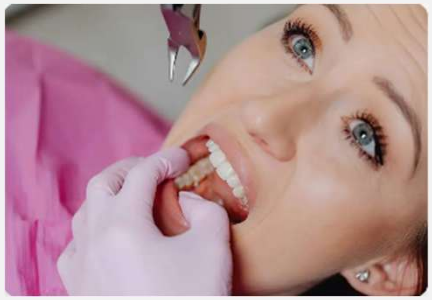
- **Healthcare workers living with a blood borne virus:**
  - must have appropriate and ongoing medical care
  - must be tested for the respective blood borne virus viral load levels, as well as for other blood borne viruses, in accordance with the National Guidelines
  - must comply with the listed blood borne virus criteria in the National Guidelines

EXPOSURE PRONE PROCEDURES

The National Guidelines define exposure prone procedures as:

"procedures where there is a risk of injury to the healthcare worker resulting in exposure of the patient's open tissues to the blood of the healthcare worker. These procedures include those where the healthcare worker's hands (whether gloved or not) may be in contact with sharp instruments, needle tips or sharp tissues (spicules of bone or teeth) inside a patient's open body cavity, wound or confined anatomical space where the hands or fingertips may not be completely visible at all times."

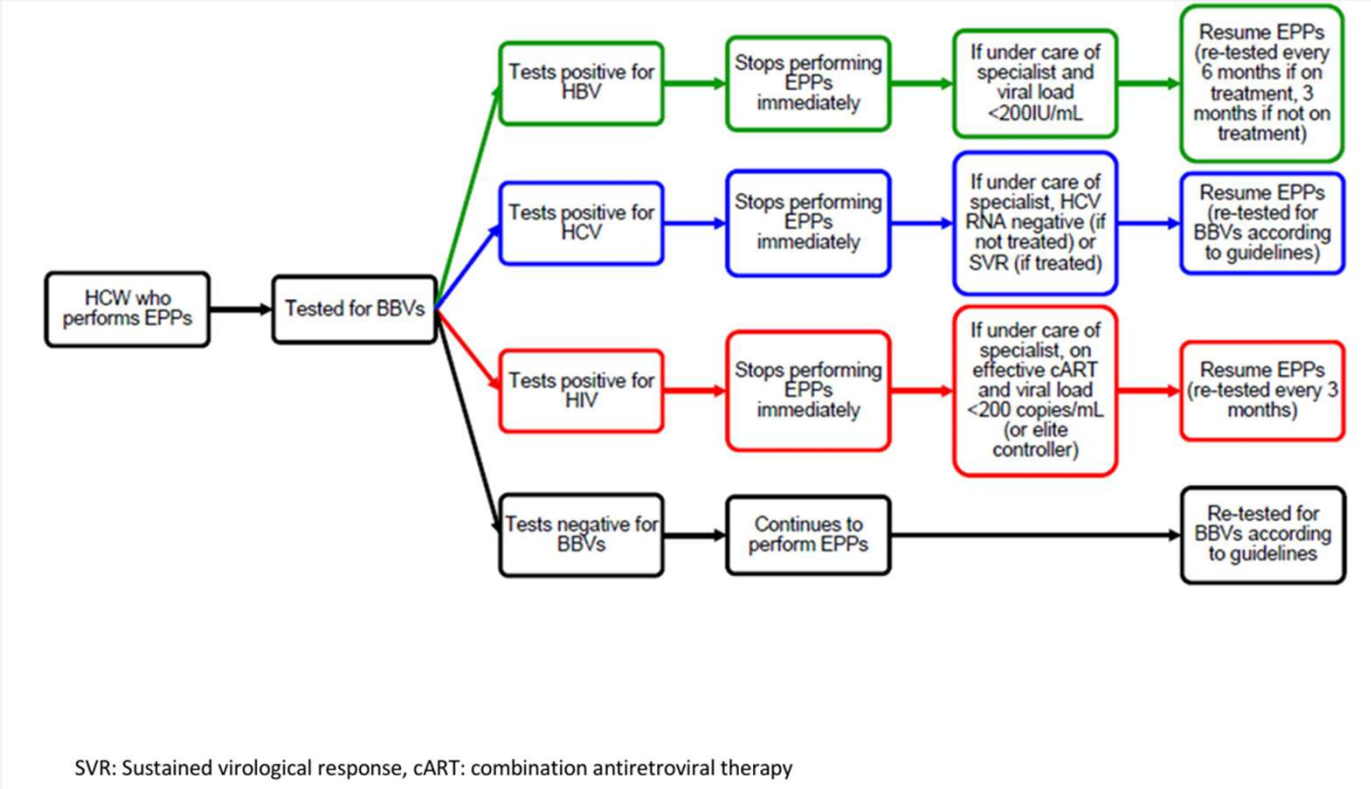
➔ **Operative dentistry!**



CDNA NATIONAL GUIDELINES FOR HEALTHCARE WORKERS 2018

- **Healthcare workers who perform exposure prone procedures:**
  - must take reasonable steps to know their blood borne virus status and should be tested for blood borne viruses at least once every 3 years
  - must declare when applying for renewal of registration that they are complying with, and have been tested in accordance with the National Guidelines
  - should understand their obligation to report their blood borne virus status, if required, under jurisdictional legislation and/or policies
  - should understand their obligation to report all sharps injuries, whether or not there was a risk of patient exposure

BBV TESTING REQUIREMENTS FOR HCW'S WHO PERFORM EPP'S



SVR: Sustained virological response, cART: combination antiretroviral therapy

QUESTIONS?

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RESOURCES

- Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM) = https://ashm.org.au/
- Kirby Institute UNSW = https://www.kirby.unsw.edu.au/
- CDNA National Guidelines for healthcare workers on managing bloodborne viruses = https://www.cdc.gov.au/resources/collections/cdna-national-guidelines-healthcare-workers-bbv