Surveillance of Viral Hepatitis in Hong Kong

2021 Report

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ABBREVIATIONS

AIDS	Acquired immune deficiency syndrome
Anti-HAV	Antibody against hepatitis A virus
Anti-HBc	Antibody against hepatitis B core antigen
Anti-HBs	Antibody against hepatitis B surface antigen
Anti-HCV	Antibody against hepatitis C virus
Anti-HDV	Antibody against hepatitis D virus
Anti-HEV	Antibody against hepatitis E virus
CHP	Centre for Health Protection
CI	Confidence interval
COVID-19	Coronavirus disease 2019
CRPVH	Community Research Project on Viral Hepatitis
DH	Department of Health
FHS	Family Health Service
FPAHK	Family Planning Association of Hong Kong
HBsAg	Hepatitis B surface antigen
HAV	Hepatitis A virus
HBV	Hepatitis B virus
HCC	Hepatocellular carcinoma
HCV	Hepatitis C virus
HCW	Health care worker
HDV	Hepatitis D virus
HEV	Hepatitis E virus
HIV	Human immunodeficiency virus
HKRCBTS	Hong Kong Red Cross Blood Transfusion Service
ICS	Immunisation coverage survey
IgG	Immunoglobulin G
IgM	Immunoglobulin M
ITC	Integrated Treatment Centre
MCHC	Maternal and Child Health Centre
MSM	Men who have sex with men
OR	Odds ratio
PHLSB	Public Health Laboratory Services Branch
PMH	Princess Margaret Hospital
PWH	Prince of Wales Hospital
PWID	People who inject drugs
QMH	Queen Mary Hospital
RNA	Ribonucleic acid
RT-PCR	Reverse transcription polymerase chain reaction
STI	Sexually transmitted infections
TPC	Therapeutic Prevention Clinic
WHO	World Health Organization
WPRO	Western Pacific Regional Office

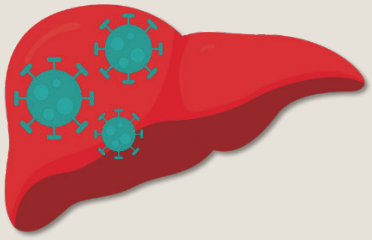
SURVEILLANCE 2021 AT A GLANCE

Number of reported cases of viral hepatitis

Hepatitis A
15

Hepatitis B
17

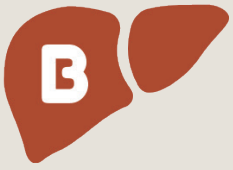
Hepatitis C
23

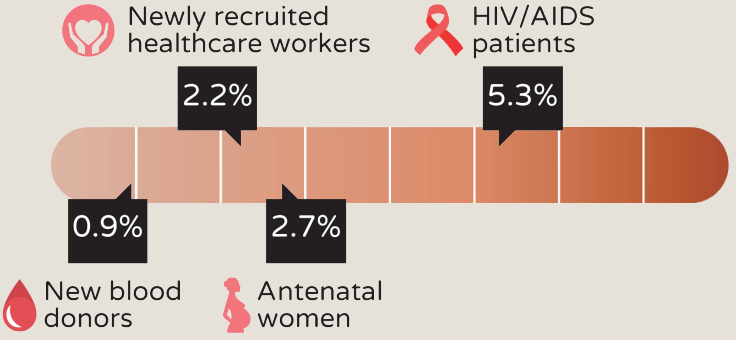


Hepatitis D
1

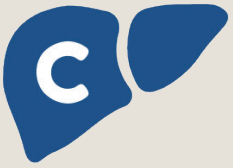
Hepatitis E
77

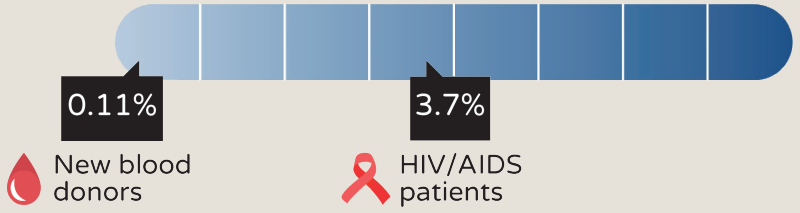
Prevalence of HBsAg





Prevalence of anti-HCV





Liver cancer statistics (2020)

Number of new cases
1735

Number of deaths
1530

Coverage of hepatitis B vaccination

Birth dose coverage
99.7%

Third dose coverage in pre-school children born in 2015 - 2017
99.3%

SURVEILLANCE MECHANISMS OF VIRAL HEPATITIS

1. Viral hepatitis is a statutory notifiable disease in Hong Kong. Voluntary reporting was started in 1966, and the disease has become notifiable since 1974. It was not until 1988 that the reported cases were classified by viral etiology, namely hepatitis A, hepatitis B, non-A non-B hepatitis and unclassified hepatitis. In 1996, non-A non-B hepatitis was further categorised into hepatitis C, hepatitis E and hepatitis (not elsewhere classified).

2. The extent of chronic viral hepatitis, notably hepatitis B and C, is determined by other mechanisms. This Report presents the latest findings from collation and analysis of viral hepatitis data obtained from the disease notification system, service statistics, seroprevalence studies and other research findings.

COMMENTARY

Hepatitis A

Acute Hepatitis A Virus Infection

3. Hong Kong was once of intermediate endemicity for hepatitis A virus (HAV) [1, 2]. After 1988 when viral hepatitis began to be reported according to etiologic agents, the largest epidemic of hepatitis A occurred in 1992, with over 3 500 cases reported to the Department of Health (DH) (Box 1). This represented a notification rate of 63 per 100 000 population (Box 8), and since then, a gradual declining trend in HAV incidence has been observed. This discernible decline in hepatitis A contributed to a parallel declining trend in overall reported viral hepatitis since 2002 (Box 4). The death rates from hepatitis A has been low, ranging between 0 and 0.15 per million population in the last two decades (Box 8).

4. From 2012 to 2021, there were a total of 658 hepatitis A reported cases and the annual number of cases peaked in 2015 at 138 and dropped to a record low of 15 in 2021 (Box 5). The male to female ratio was 1.5:1, with 74% aged below 45 years (Box 6, Box 7). Over the years, there has been an increase in the proportion of reported cases over 35 years old. Although the majority were still below 45 years of age, the proportion of reported cases that were aged 45 and above increased from less than 10% in early 2000s to 14% - 41% in the 2010s (Box 7). In 2021, the proportion of reported cases aged 45 and above was exceptional high at 80% (12/15), which shall be interpreted with caution given the small number of cases reported in that year.

5. In 2015, a review on 587 reported cases of hepatitis A from 2005 to 2014 was published by the Surveillance and Epidemiology Branch of Centre for Health Protection (CHP), DH. The majority (70%) of cases required hospitalisation, and two fatal cases were recorded. Both fatalities had multiple comorbidities. The majority (76%) of the patients acquired the disease locally. Most (92%) were sporadic cases and 22 small clusters affecting two to four patients were identified. Of these, at least 60% were clusters affecting members of the same household [3].

6. An increase in the number of cases was noted in 2015 when a total of 138 cases were reported. The majority (75%) of the cases was reported from February to June. The male to female ratio was 1.2 to 1, with a median age of 33 years (range: 3 to 83 years). There was no fatality. Except two cases studying in the same school and two cases from the same family, no epidemiological link was found. No single identifiable source could explain the upsurge of cases [3].

7. In late 2016, an unusual upsurge of acute HAV infection affecting men who have sex with men (MSM) with human immunodeficiency virus (HIV) infection was noticed. With retrospective investigations and prospective reporting, a total of 53 cases of laboratory-confirmed HAV infection with clinical symptoms among individuals identified as MSM were recorded between September 2015 and November 2017. The age range of the cases was 20 to 55 years (median: 33 years). Forty-five (84.9%) required hospitalisation and there were no fatalities. Thirty-seven cases (69.8%) were known to be HIV-positive attending one of the three designated public HIV clinics. The majority (96.2%) did not report history of hepatitis A vaccination. Eighteen (33.9%) reported travel history within the incubation period. Around one quarter of the cases had concurrent diagnosis of other sexually transmitted infections (STI) including syphilis, gonorrhoea and chlamydia infection. Among the cases with specimen available for laboratory analysis, forty-three (81.1%) had identical nucleotide sequences within the genotyping window. Apart from one cluster affecting two patients, who were sex partners residing together, no other epidemiological linkage could be found. No common food nor water source or social gathering was identified among these cases. Epidemiological investigations suggested that the outbreak was contributed by transmission by way of sexual contact between men, a high proportion of whom were HIV-infected [4]. Hepatitis A outbreaks among MSM communities were reported during the same period in some other regions with low HAV endemicity, including Taiwan [5], Europe [6] and both North and South America [7-8].

Prevalence of anti-HAV

8. In a territory-wide seroprevalence study on viral hepatitis, involving 10 256 participants recruited between February 2015 and July 2016, the crude and adjusted prevalence of antibodies against hepatitis A virus (anti-HAV) in Hong Kong was 65.2% (95% confidence interval [CI]: 64.2% - 66.1%) and 52.2% (95% CI: 51.3% - 53.2%) respectively [9]. The prevalence of anti-HAV found in this study was significantly lower than that (71.0%) in another previous local seroprevalence study ($P < 0.001$), conducted back in 2001 via telephone household survey (Community Research Project for Viral Hepatitis 2001, CRPVH) (Box 21) [2].

9. Observations from epidemiological studies signify an aging cohort effect with an overall decline in the prevalence of HAV infection. Anti-HAV positivity was less common across all age groups among subjects aged 30 or above in the seroprevalence study in 2015-16 [9] than the subjects in the same age groups in CRPVH conducted in 2001 [2]. Similar phenomenon that a lower anti-HAV prevalence among the subjects of the same age groups in a more recent study was observed, while comparing the findings of CRPVH 2001 with

those in another study conducted in late 1980s [10] or comparing the late 1980s findings with those of a late 1970s study on local HAV seroprevalence [11]. Together, these four studies suggest that age-specific prevalence of anti-HAV has right-shifted locally since 1980s. As of 2016, the prevalence of anti-HAV remained at low level (around 20%) among adults aged below 30 years old. However, an anti-HAV prevalence exceeding 80% could only be observed in people aged 60 years old or above in 2016, instead of those aged >=40 years in 2001, in the general Chinese population (Box 21).

10. Similar cohort effect has also been observed from the laboratory surveillance performed by Public Health Laboratory Services Branch (PHLSB) every five years since 2000. In the latest serosurvey conducted by PHLSB in 2020, an anti-HAV prevalence exceeding 80% was limited to those aged above 60, while the anti-HAV prevalence was higher than 80% in younger subjects aged above 40 in 2000 and 2005 or those aged above 50 in 2010 and 2015. The seroprevalence of anti-HAV was generally below 50% among those younger than 30 years old over the years (Box 22) [12]. In the last two serosurveys in 2015 and 2020, there was a significant increase in the seroprevalence of anti-HAV in the younger age group, most prominent among those aged 0 – 20. This finding may suggest an increase in the uptake of hepatitis A vaccination in the community, while the overall hepatitis A activity remained low in Hong Kong in the two decades before 2020. However, some limitations of the serosurvey, including relatively small sample size and potential bias from convenience sampling, should be noted while interpreting its results. Overall, with the declining prevalence of HAV infection, Hong Kong has changed from a region with intermediate to very low endemicity in the past three decades.

11. Besides an increasing prevalence with higher age, people born outside Hong Kong were generally more likely to test positive for anti-HAV, whereas a lower anti-HAV positivity rate was observed among people of non-labour work [2]. In the seroprevalence study 2015-16, anti-HAV positivity was more likely among the participants born in the mainland China, while those having lower monthly household income were more likely to be anti-HAV-positive [9].

12. From the telephone interview of the CRPVH 2001, some 11% of 4 564 subjects reported a history of HAV vaccination, about 80% of whom had completed the course. The uptake of vaccination in the general population remained low, as 5.9% of the participants in the seroprevalence study 2015-16 had received hepatitis A vaccination [9]. Both the low coverage of hepatitis A vaccination and the low circulating HAV in the community probably lead to a general decrease in anti-HAV prevalence over the years.

13. Cross-sectional surveys of anti-HAV at Kowloon Bay Integrated Treatment Centre (ITC), the HIV specialist clinic under DH, have been started since 2007. The subjects consisted of all new HIV/AIDS patients who first attended ITC between July 2007 and 2021 and convenience samples of all active HIV/AIDS patients who first attended ITC before July 2007 (Box 23). The prevalence of anti-HAV increased with age of HIV/AIDS patients, and the overall positivity rate among these patients tested between 2007 and 2021 appeared to be comparable with that of the data obtained from serosurvey in the general population in 2001 and 2016. Confounding factors, such as different levels of past infection, immunodeficiency in HIV patients, history of hepatitis A vaccination and difference in years of testing, may have affected the results. Compared with patients acquiring HIV via other routes, those infected via homosexual or bisexual routes were most vulnerable to subsequent HAV infection, as reflected by the lowest level of anti-HAV prevalence in this group of patients (Box 24). Indeed, the increased susceptibility had manifested itself during the upsurge of hepatitis A infection among MSM occurring in 2015 to 2017 [4]. As a result, the Scientific Committee on AIDS and STI and Scientific Committee on Vaccine Preventable Diseases extended their recommendation for hepatitis A vaccine to MSM in June 2017 [13].

Hepatitis E

Acute Hepatitis E Virus Infection

14. The annual notification of hepatitis E infection increased from 11 in 1996 to a record high of 150 in 2012 (Box 1). In the past five years, the number of reported cases of hepatitis E ranged from 43 to 85. A higher number of infections were usually reported from February to April (Box 16), but such seasonal pattern was less prominent in recent few years. Of 1 556 cases reported, 1 012 (65.0%, Box 17) were male, giving male to female ratio of 1.9:1. The majority was adults, most of whom were aged between 35 and 74 (Box 18). Fatalities were more common with acute hepatitis E than with acute hepatitis A, and there have been death cases attributable to acute hepatitis E infection each year since 2014 (Box 19). In 2021, four deaths were reported, giving a record high death rate at 0.54 per million population.

15. In 2011, the CHP reviewed all hepatitis E cases recorded between 2001 and 2010 [14]. Of the 524 cases, 78.2% were hospitalised with a median stay of seven days. A total of 12 cases were fatal (9 males and 3 females), and age ranged from 53 to 82 (median age 67.5 years). The case fatality rate was 2.3%, which was comparable with reported figures from other countries (0.2% - 4.0%) [15]. None of the fatal cases was pregnant. Most cases (99.4%) were sporadic infection, except a small family cluster involving three persons, and 87.4% acquired the disease locally. Epidemiological investigation did not identify any outbreak linked to a particular food premises.

16. The epidemiology of acute hepatitis E cases recorded by CHP was also reviewed in recent years [16, 17]. The latest review covered a total of 461 cases from 2013 to 30 September 2018, with age ranging from 15 to 96 years (median: 56 years). More males were affected than females (62.5% vs. 37.5%). More than half of the cases were recorded from January to April. Most of the cases (81.8%) acquired the infection locally. Some 399 (86.6%) patients required hospitalisation with a median length of stay of seven days. Nine fatal cases were recorded, among whom eight had underlying illnesses, giving a case fatality rate of 2.0%. The age of the deceased patients ranged from 49 to 81 years (median: 74 years). A significant proportion of the patients recalled consuming pig liver (28.6%) and shellfish (28.9%) during the incubation period. Notably, one case recorded in August 2018 acquired the infection from organ transplant, involving a single deceased person whose organs had been donated to five recipients in February 2018. Subsequent laboratory investigations found that the other four recipients also had hepatitis E virus (HEV) infection [18].

Clinical Epidemiology of HEV Infection

17. The epidemiology and clinical features of sporadic hepatitis E cases were compared with those of another enterically transmitted hepatitis, namely hepatitis A. Of 105 acute hepatitis A and 24 hepatitis E patients seen at Princess Margaret Hospital (PMH) in 2002, patients having hepatitis A were significantly younger (median age: 27 years) and had recent history of shellfish consumption while hepatitis E patients were older (median age: 53 year) and most had a recent travel history. Moreover, whereas hepatitis A was milder and recovery was uneventful, hepatitis E was more severe, associated with significant mortality and frequently complicated by protracted coagulopathy and cholestasis [19]. The higher disease severity for hepatitis E was also identified in a territory-wide cohort study, involving 1 068 cases of acute hepatitis A and 846 cases of acute hepatitis E from 2000 to 2016. As compared with hepatitis A patients, hepatitis E patients had more all-cause mortality (3.9% vs 0.6%; $P < 0.001$), liver-related mortality (2.0% vs 0.3%; $P < 0.001$) and hepatic events (2.8% vs 0.3%; $P < 0.001$) within 30 days from diagnosis [20].

18. A local study examined the epidemiology and genotype distribution of HEV infections from 57 laboratory-confirmed acute hepatitis E patients who were admitted to Prince of Wales Hospital (PWH) during 2002 and 2007. Almost all patients (56/57; 98%) were Chinese and most (48/57; 84%) had no history of travel during the prior 6 months. All cases were sporadic. No fulminant hepatitis was recorded and all patients recovered. Phylogenetic analyses of isolates from 46 patients showed that all except one belonged to genotype 4, and most were phylogenetically related to swine isolates reported from China. The remaining isolate was genotype 3 obtained from a woman who had no history of travel [21].

19. Apart from pregnancy, coinfection with hepatitis B virus (HBV) might be associated with more fulminant clinical outcome in patients infected with HEV. Among three cases of serious HEV infection with acute liver failure reported to DH in the first two months of 2012, one required liver transplantation and two passed away. One of the deceased patients was tested positive for chronic hepatitis B infection [22]. Moreover, a 10-year retrospective study on acute hepatitis E in local hospitals showed that patients with chronic hepatitis B acutely infected with HEV had a higher rate of liver failure, liver-related mortality and all-cause mortality, though the association was not statistically significant [23]. In another territory-wide cohort study from 2000 and 2016, coexisting chronic hepatitis B was found to be an independent risk factor for liver-related mortality in patients with acute hepatitis E (adjusted hazard ratio = 3.34; $P = 0.02$), as compared with acute hepatitis A patients [20].

HEV in High-risk Food Items

20. Given the evidence that suggests a zoonotic source of hepatitis E in overseas studies, the Centre for Food Safety conducted a risk assessment study titled "Hepatitis E Virus in Fresh Pig Livers" [24] to determine the HEV prevalence in fresh pig liver samples obtained in local markets. One hundred fresh pig liver samples were collected from pigs slaughtered between mid-January and May 2009. Sixteen (31%) out of 51 roaster pig (around four months old) liver samples were positive for HEV, while none of the 49 porker pig (around six months old) liver samples tested positive. Partial sequences of some HEV isolates from roaster pigs were identical to those from 7 among 48 local human cases. The findings suggest the possibility of roaster pigs as one of the sources of local human hepatitis E infections.

21. The genetic association between human HEV infection and HEV-contaminated high-risk food in Hong Kong was examined in a molecular epidemiological study by comparing local virus strains obtained from sera from 24 hepatitis E patients with those surveyed from five types of high-risk food items (lamb, oyster, pig blood curd, pig large intestine and pig liver) between 2014 and 2016 [25]. HEV RNA was detected in pig liver, pig intestine and oyster samples with prevalence of 1.5%, 0.4% and 0.2% respectively. Phylogenetic analysis showed that all sequenced human and swine HEV strains belonged to genotype 4 with close genetic relatedness. Again, the findings suggested that swine could be an important foodborne source of autochthonous human HEV infections in Hong Kong. The study also echoed the evidence of a major epidemiological shift in hepatitis E in Southern China driven by genotype switch from HEV-1 to HEV-4 over the past two decades [26].

Prevalence of HEV

22. In the CRPVH study conducted in 2001, 18.8% of adult subjects were found to have serologic evidence of HEV infection. People in the 40 - 49 years age group had the highest positivity rate of 24.1% (Box 25). Another local seroprevalence study on anti-HEV using 450 serum samples submitted for virological investigation in 2008 - 2009 in a local hospital found a higher rate of HEV IgG seropositivity at 28.7% [27]. The HEV IgG seropositivity rate increased from 8% among 1 - 10 years old to >56% among those aged over 80. The overall seropositivity rate was higher among male than female (32.9% vs 24.4%, p=0.048).

23. The overall anti-HEV seroprevalence had further risen in the past decade. A cross-sectional sero-epidemiological study conducted between February 2012 and May 2014 gave an overall anti-HEV seropositivity at 32.0% [28]. This community-based study involved a total of 1 539 participants sampled from different subpopulations, including healthy adults, pregnant women, patients with chronic liver disease, elderly people and frequent food handlers. Independent risk factors associated with anti-HEV seropositivity was older age (>35 years), no hand-washing practice after handling shellfish and lower education level. Prevalence of anti-HEV remained at a similar level at 33.3% (95% CI: 32.4% - 34.2%) in the territory-wide seroprevalence study in 2015-16 [9]. The study also found that hepatitis A and E shared similar risk factors, such as being born in mainland China and increasing age, and protective factor of higher family income. In both studies, male sex was associated with increased risk of acquiring HEV.

24. The HEV prevalence was also determined in Hong Kong blood donors [29]. Of 10 000 unlinked donation samples collected in March to May 2015, two were tested positive for HEV RNA. Genotype 4, the dominant genotype in circulation in Hong Kong, was identified in one of the two RNA-positive samples, while genotyping was unsuccessful for another one. Both samples were also positive for IgG and IgM anti-HEV. Anti-HEV seroprevalence was estimated as 15.8% among all donors. IgG anti-HEV positivity rate was higher in males, and increased with age from 3.1% for age group 16 - 20 to 43.1% for age group 51 - 60. The HEV RNA positivity rate at 0.02% found in the study was within the reported range in developed countries (0.01% - 0.08%).

25. Following the documentation of bloodborne transmission of HEV in recent years, a matched cohort study was conducted to assess the effects of age, gender and addictive injection use on HEV serostatus and concentration [30]. HEV IgG seroprevalence was 46.2% among 91 people who inject drugs, who underwent HCV load testing between 1 January 2018 and 31 October 2019, as compared with 22.0% in 91 age- and sex-matched organ donors. Increasing age and addictive injection use were significantly associated with HEV IgG positivity. The study results suggested that people who inject drugs were at increased risk for hepatitis E and prone to repeated HEV exposure and reinfection, indicated by higher HEV IgG concentrations.

Epidemiology of Human Infection of Rat HEV

26. The usual HEV causing human infection belongs to *Orthohepevirus A* (HEV-A), while *Orthohepevirus* genus has three other species circulating in different hosts, namely *Orthohepevirus B* in chickens, *Orthohepevirus C* (HEV-C) in rats and ferrets and *Orthohepevirus D* in bats. Cases of human infection with HEV-C (also known as rat HEV) were first reported in Hong Kong in 2018, involving a 56-year-old man having immunosuppressant for anti-rejection prophylaxis after liver transplant in May 2017 [31] and a 70-year-old woman on immunosuppressant for treatment of underlying disease [32]. Epidemiological investigation of the first two cases conducted by the CHP revealed that both cases resided in Wong Tai Sin District without travel history during the incubation period of usual HEV infection. The two patients could not recall having direct contact with rodents or their excreta, but one recalled having seen suspected rodent excreta in his residence. Based on the available epidemiological information, the source and the route of infection in these two immunocompromised patients could not be determined. The exact mode of transmission of rat HEV to humans is unknown at the moment.

27. To describe the epidemiological and clinical features of human HEV-C1 infection in Hong Kong, a territory-wide prospective study was conducted by screening blood samples from 2 860 patients with abnormal liver function or immunosuppressive conditions between 1 January 2017 and 31 July 2019 [33]. Of the eight identified infections, three had acute hepatitis, four had persistent hepatitis and one had subclinical infection without hepatitis. HEV-C1 hepatitis was generally milder than HEV-A hepatitis. One HEV-C1 isolate obtained from a rat captured in Wong Tai Sin District, where half of the identified cases resided, was closely related to the major outbreak strain in Hong Kong.

28. Another clinical-epidemiological investigation of human HEV-C1 infections found that HEV-C1 accounted for 8/53 (15.1%) reverse transcription PCR (RT-PCR) confirmed hepatitis E infections in Hong Kong between 1 August 2019 and 31 December 2020, raising the total number of HEV-C1 infections detected in the city to 16 [34]. These eight patients were elderly and/or immunocompromised, and half tested negative for HEV IgM. Among immunocompromised patients infected with HEV between January 2016 and December 2020 in Hong Kong, there were nine cases (9/21; 42.9%) of HEV-C1 infection. The proportion of patients who developed persistent hepatitis was similar between immunocompromised HEV-C1 patients (7/9; 77.8%) and HEV-A patients (10/12; 83.3%).

Hepatitis B

Acute Hepatitis B Virus Infection

29. The number of reported acute HBV infections has been decreasing over decades, from 137 cases reported in 2000 to 17 cases reported in 2021 (Box 1).

30. In an epidemiologic study of acute HBV infection conducted by DH and Hong Kong Red Cross Blood Transfusion Service (HKRCBTS), 149 of 351 eligible subjects recruited from 2000 to 2003 participated in risk factor assessment with or without blood screening. Repeat blood donors who tested positive for hepatitis B surface antigen (HBsAg) for the first time and were then confirmed IgM anti-HBc positive were reported as having acute HBV infection. There were 43 such clients, yielding a yearly incidence rate of HBV seroconversion in repeat donors between 3.5 and 9.4 per 100 000 population during the study period. Nearly 70% of the study subjects were male; 99% were Chinese and the mean age was 31 years. From standardised questionnaire interview by nurses, over half could not have risk factor of acute HBV infection determined, while sexual contact was assessed to be the commonest risk (85%) in the rest. Of 124 subjects who had hepatitis B screening at 6 months post-IgM anti-HBc positivity, 50% developed anti-HBs while 9.7% were positive for HBsAg. Although these results could suggest a higher rate of HBV chronicity than what was previously reported in the literature, they have to be interpreted with caution owing to the relative small number of samples, incompleteness of data and potential biases from the subjects sampling and other study design.

Seroprevalence of HBV Infection

31. The territory-wide seroprevalence study in 2015-16 gave a crude and age-and-sex-adjusted prevalence of HBsAg at 7.8% and 7.2% respectively in the general population [9]. A more recent household survey conducted between October 2018 and August 2019 also gauged a crude HBsAg prevalence of similar level at 8%, where 489 participants with mean age at 51 year-old were recruited [35]. Several features on the current pattern of HBV infection could generally be observed from the previous seroprevalence studies, including the CRPVH 2001 (Box 45), namely

(a) chronic HBV infection is in a general declining trend in community groups without apparent risk of contracting HBV,

(b) HBsAg prevalence is higher in older adults, and

(c) chronic HBV infection is commoner in male than female.

32. Seroprevalence of HBsAg in different communities are monitored continuously and the various adult communities can be categorised into three groups according to the risk of contracting HBV:

(a) without apparent risk: blood donors, pre-marital/ pre-pregnancy service users, antenatal women, police officers, new health care workers (HCW)

(b) with undetermined risk: clients seeking post-exposure management and tuberculosis patients

(c) with apparent risk: drug users, HIV/AIDS patients and female sex workers

33. A word of caution in the interpretation of data though, is that testing for HBV markers has been performed for a variety of reasons in different communities, with heterogeneous mix of population characteristics.

Seroprevalence of Adult Communities without Apparent Risks

34. The temporal decline of chronic HBV infection has been most obvious in new blood donors and police officers. For new blood donors, the HBsAg prevalence follows a continual falling trend since early 1990s, from 8.0% in 1991 to 0.9% in year 2021 (Box 27). The trend is even more obvious among the 16 - 19 years age group where the prevalence was as low as 0.33% in male and 0.31% in female in 2021 (Box 28, Box 29). A similar trend was observed among police officers where the HBsAg prevalence fell from 7.9% in 1997 to 1.8% in 2021 (Box 36), with a prevalence of 0.9% among those aged 30 or less (Box 35). A falling trend was generally observed in other community groups without apparent HBV risk (Box 26, Box 34).

35. The HBsAg prevalence in newly recruited health care workers as determined at pre-HBV vaccination screening also showed a generally decreasing trend (Box 37). The prevalence decreased from 4.6% in 2002 to 1.6% in 2021 among newly recruited male health care workers, while that for newly recruited female health care workers decreased from 5.2% to 3.0% over the same period.

36. The HBsAg prevalence in antenatal mothers has been decreasing from over 10% in the early 1990s to 2.7% in 2021 (Box 30). As compared with other groups without apparent risk, the overall HBsAg prevalence in antenatal mothers is higher and confounded by the place of birth. A study of 2 480 pregnant women attending the Maternal and Child Health Centre (MCHC) of DH in 1996 found an HBsAg prevalence at 13.1% in those born in mainland China

as compared to 8.4% in local mothers [36]. Data from Virus Unit, DH also showed a higher prevalence of 12.5% and 13.8% in the subset of non-resident expectant mothers versus the overall positivity rate of 8.5% and 8.6% in 2004 and 2005 respectively. The prevalence of HBsAg among antenatal mothers also varied significantly by age (Box 31, Box 32). The HBsAg prevalence among antenatal mothers younger than 25 years has been dropping to a low level (less than 1%) in 2021, as compared with those aged 35 years or above (more than 4%). The age-specific prevalence is in line with the findings in a retrospective cohort study, involving 10 808 young pregnant women aged 25 years or below born in Hong Kong and managed at a local hospital between 1998 and 2011 [37]. The HBsAg prevalence in the study ranged between 2.3% and 8.4%, with a significantly lower prevalence among those being born in and after 1984 (Odds ratio [OR]: 0.68, 95% CI: 0.58 - 0.80), when hepatitis B vaccination was given to neonates born to HBsAg-positive mothers.

37. The HBsAg prevalence of users of pre-marital check-up in The Family Planning Association of Hong Kong (FPAHK) decreased from 9.6% in 1991 to 6.5% in 2010. The prevalence has further dropped to 3.3% in 2021 among pre-marital or pre-pregnancy package service users (Box 33).

Seroprevalence of Adult Communities with Undetermined Risk

38. Of 827 tuberculosis patients attending Tuberculosis & Chest Clinics, DH between March and May in 2021, 64 (7.7%, Box 38) were detected HBsAg positive, with the highest prevalence rate in the middle age group (40 - 59 years old: 9.9%, Box 39) followed by the more elderly group (>= 60 years old: 8.1%, Box 39). The HBsAg positivity rate was usually higher in male clients than in female clients, but similar level of HBsAg prevalence was observed between males (7.6%) and females (7.9%) in 2021 (Box 38). Both the age (Box 39) and gender pattern (Box 38) were consistently observed over the last decade.

39. Among clients attending for post-exposure management in Therapeutic Prevention Clinic (TPC) at ITC of CHP, DH in 2021, HBsAg rate was low at 1.6% in both non-health care workers and health care workers (Box 40).

Seroprevalence of Adult Communities with Apparent Risk

40. The HBsAg prevalence in HIV/AIDS patients under care of DH was in the range of 5.3% to 10.7% in the past decade (Box 42). The HBsAg prevalence was highest among those patients who were drug users (15.2%), while the lowest HBsAg prevalence was observed in heterosexual female patients (5.5%) (Box 43). Due to underlying immunosuppression and shared routes of transmission, HIV/AIDS patients are more likely to be chronically infected with HBV [38].

41. The HBsAg prevalence in female sex workers attending the clinic of Action for REACH OUT tested between 2007 and 2011 ranged from 5.0% to 10.4% (Box 41), similar to that measured in 1995 - 1998 at 6.8%.

42. The data regarding prevalence of HBsAg in drug users was difficult to interpret because of the small number of subjects since 2006 (Box 44). Before 2006, the annual prevalence of HBsAg in drug users was exceeding 10%, except for the year 1996 and 1997.

43. Overall, the difference in HBsAg prevalence between groups with or without apparent risk of contracting HBV has not been prominent in the past few years.

Seroprevalence of Children

44. In 2009, an HBsAg seroprevalence study was conducted among 1 913 children aged 12 to 15 years who were born after the implementation of universal neonatal hepatitis B vaccination programme [39]. The seroprevalence of HBsAg was 0.78% (95% CI: 0.39 - 1.16%, Box 46). This result showed that Hong Kong had already achieved a time-bound goal set by the Western Pacific Regional Office (WPRO) of the WHO, which referred to reducing chronic HBV infection rate to less than 2% among children at least 5 years of age by the year of 2012. In July 2011, Hong Kong was verified by WPRO as having successfully achieved the goal of HBV control. Based on the same study, Hong Kong was also verified as of June 2013 as having met the goal of achieving a seroprevalence of less than 1%.

Genotypes of HBV and Their Disease Course

45. Different HBV genotypes have been identified with distinct geographic distribution and association with different clinical outcomes. Local studies indicated that genotype C was the commonest genotype and genotype B was the second. A study of 776 chronic hepatitis B patients seen at the University of Hong Kong Liver Clinic from 1999 to mid-2003 found that genotype C was the commonest (486, 62.6%), followed by genotype B (252, 32.5%), with a majority of genotype B belonging to subgroup Ba [40]. Another study of 426 chronic hepatitis B patients recruited consecutively from 1997 to mid-2000 at the Hepatitis Clinic of Prince of Wales Hospital (PWH) found a prevalence of 57% (242) and 42% (179) of genotypes C and B respectively [41].

46. A study of 49 HBV genotype C isolates from Chinese patients under the care of the PWH Hepatitis Clinic identified 2 distinct groups with different epidemiological distribution and virologic characteristics – 80% being genotype “Cs” (found mostly in Southeast Asia) and 20% “Ce” (predominated in Far East) [42]. In addition, subgenotype Cs appears to be more common in Hong Kong than other parts of China. In another analysis of a cohort of patients with HBeAg-negative chronic liver disease from three different parts of China (Beijing, Shanghai and Hong Kong), 69% of genotype C patients in Hong Kong belonged to subgenotype Cs whereas 97% of genotype C HBV in Shanghai and Beijing belonged to subgenotype Ce ($P< 0.0001$) [43].

47. Regarding the disease course of HBV infection, local studies suggested that patients infected with genotype C had a higher risk of cirrhosis and hepatocellular carcinoma (HCC) development [41, 44], as well as more severe histological fibrosis [45]. A recent meta-analysis concluded that HBV genotype C was associated with a higher risk of HCC than other major HBV genotypes [46]. Among HBV genotype C, subgenotype Cs appears to carry a worse prognosis than subgenotype Ce [43]. In a local study conducted by the Chinese University of Hong Kong, patients infected by subgenotype Cs had the lowest serum albumin and highest alanine aminotransferase levels compared with subgenotypes Ce and Ba. Moreover, patients infected by subgenotype Cs had more severe histological necroinflammation than subgenotype Ce [43]. However, the meta-analysis did not find significant difference in the risk of HCC between HBV-infected patients with subgenotype Ce and Cs [46].

48. Nevertheless, in a local study of 119 end-stage HBV-related liver disease patients requiring liver transplantation between September 1996 and August 2003, those with genotype B had significantly more pre-transplant acute flare and worse liver function while genotype C patients had a greater risk and severity of recurrence due to lamivudine-resistant mutants [47].

49. In a case-control study, it was concluded that HCC patients had a significantly higher prevalence of core promoter mutations and genotype C but the association with HCC was mediated via the former [48]. A study of 5 080 chronic HBV patients focusing on familial HCC found 22 such families, giving a prevalence of 4.3 families/1000 HBV carriers [49]. Age of onset of HCC was significantly younger in familial HCC than sporadic cases, and it progressively decreased down the generations, suggesting an anticipation phenomenon.

Co-infection with Hepatitis D Virus

50. Hepatitis D virus (HDV) is a defective RNA virus that can infect only individuals who have HBV. In Hong Kong, HDV superinfection has been rare among non-drug abusers. In a study in early 1990s, only one patient was found to be anti-HDV-positive after testing sera collected from 664 patients with chronic hepatitis B and 31 patients with acute hepatitis B between January 1988 and December 1990 [50]. In the territory-wide seroprevalence study in 2015-16, no cases of HDV infection were detected among 10 256 participants, when 803 of the participants were HBsAg-positive and almost all had no history of illicit intravenous drug use [9].

51. After the first ever hepatitis D case reported to the CHP (Box 1) in November 2020, there was another reported hepatitis D case in August 2021. While the first case was a male injecting drug user aged 65 and above, the second case was a man in his forties, without history of needle sharing during the incubation period. Both cases were discharged after hospitalisation for three days and two weeks respectively. As reported in the aforementioned study in the 1990s, anti-HDV could be more commonly detected in people who inject drug (PWID), who had HBV-related chronic liver disease (13/14; 93%) [50].

Hepatitis B Vaccination

52. The universal vaccination programme for newborns, increased vaccination coverage in adults, practice of universal precaution in health care settings, screening of blood donors and promotion of safer sex all contributed to the reduced HBV incidence in Hong Kong [51].

53. A local cohort study of 1 112 neonates born to HBsAg-positive mothers who received hepatitis B vaccine and hepatitis B immunoglobulin at different schedules demonstrated the long-term protective efficacy of immunisation [52, 53]. Upon completion of the vaccination schedules, 92.6% developed antibody against surface antigen (anti-HBs) seroconversion. Thirty-nine (3.5%) babies were tested positive for HBsAg and had become chronic carriers, 35 of which (89.7%) occurred before one year of age. The anti-HBs seroconversion rate dropped to 33.3% (203/610) at the 16th year of follow-up [52] and maintained at 37.4% (92/246) at the 30th year of follow-up [53]. Although 97 subjects developed anti-HBc seroconversion over the 30-year period, there was no new development of HBsAg positivity detected after the second year of follow-up. These findings demonstrated the long-term protective efficacy of neonatal hepatitis B immunisation among high-risk individuals up to at least 30 years.

54. In another local study comparing three different HBV vaccine regimens without boosters given to 318 HBV negative children recruited at age 3 months to 11 years and followed up annually, no subjects tested positive for HBsAg up to 22 years of follow-up (55 subjects). Seventy-two subjects were noted to have at least one episode of anamnestic responses with significant increase in anti-HBs titres. Three subjects had benign breakthrough HBV infection with isolated anti-HBc seroconversion [54].

55. Universal neonatal hepatitis B vaccination programme has been in place in Hong Kong since 1988. The coverage for the birth dose of hepatitis B vaccine among infants born locally was consistently above 99% in the past decade (Box 47).

56. DH has been conducting immunisation coverage surveys (ICS) every two or three years starting from 2001 to determine the coverage of all vaccines under the Hong Kong Childhood Immunisation Programme. The surveys included children aged 2 to 5 years and attending pre-primary institutions including kindergartens and childcare centres. Results from ICS conducted in 2001, 2003, 2006, 2009, 2012, 2015 and 2018 confirmed high coverage of hepatitis B vaccination [55, 56, 57, 58, 59, 60, 61]. From the provisional data obtained in the

latest round of ICS conducted in 2021, the coverages of the first, second and third dose of hepatitis B vaccination were all exceeding 99% (Box 48).

57. Apart from universal neonatal hepatitis B vaccination programme, supplementary Primary 6 vaccination programme was introduced in 1998 to provide mop-up for primary school students who have not completed the primary series of immunisation. The coverage for three doses of hepatitis B vaccine had been consistently above 99% in the past decade but showed a slight decline since 2015/16 to about 98% for the third dose. Of note, this coincided with a change of survey methodology in 2015 and an underestimation of the actual coverage was possible (Box 49). With a high coverage of the neonatal hepatitis B vaccination programme, the number of Primary 6 students eligible for mop-up hepatitis B vaccination continued to decrease in the past decade, and they were mainly children born outside Hong Kong and cross-border students. In the school year 2019/2020 and 2020/2021, the number of students who did not receive the mop-up hepatitis B vaccination was higher, as compared with the previous years. The uptake rates of mop-up hepatitis B vaccination were significantly lower than those rates in previous years. It is postulated that some requiring mop-up did not return to Hong Kong in view of the border control measures amid Coronavirus disease 2019 (COVID-19) pandemic.

58. In the CRPVH 2001 study, about 16% of the telephone-interviewed subjects reported a history of hepatitis B vaccination, with a higher frequency in persons below 50 years of age. Some 83% of them reported having completed the vaccination course. Over 99% had the cost paid by them or borne by their employers. In another local survey by face-to-face questionnaire interview on over 1 900 adult Chinese, 58% (n=1 151) of the subjects had been tested for HBV during adulthood. Among those tested negative for HBV infection, 58% (n=506) of them reported subsequent hepatitis B vaccination [63]. Age, occupation, having children and family monthly income were independent factors associated with vaccination in the study. In the territory-wide survey in 2015-16, a quarter of participants reported having received hepatitis B vaccination, which significantly reduced the chance of positive HBsAg by 85% (OR: 0.15, 95% CI: 0.11 - 0.21) [9].

Hepatitis C

Current Situation of Hepatitis C

59. From 2002 to 2021, a total of 236 cases of acute hepatitis C virus (HCV) infection were reported to DH under the statutory notification system (Box 1). Of these, 23 (9.7%) were reported in 2020. An increasing trend in the number of reported cases was observed over the years, with a record high of 39 cases in 2016 (Box 12). A review conducted by the Centre for Health Protection [64] showed that among the 22 laboratory confirmed acute hepatitis C cases reported to DH from January 2008 to October 2011, there were 17 males and 5 females, most (86%) acquired the infection locally. The median age was 47.5 years. Majority (86%) was ethnic Chinese. Five (23%) of them reported history of injecting drug use while no particular risk factor was identified for the remaining cases.

60. Of the 39 cases in 2016, 31 were male (79%), with age ranged from 23 to 94 years (median: 42 years). Thirteen (33%) required hospitalisation and no fatalities were recorded. With regard to the potential risk exposures, one case reported having tattoo procedure, and two cases were identified as injecting drug users. Two cases reported having sex partners who were HCV carriers. Among the 31 male cases reported, 23 (74%) were known MSM. There was also one case, who had history of repeated hospital admissions and had received multiple transfusions of blood product during the incubation period. Epidemiological investigation and contact tracing did not identify other acute hepatitis C cases and the source of infection in this case could not be determined. For the rest of the cases, no epidemiological linkage was identified and all cases were regarded as sporadic. There have been overseas reports of rising incidence of sexual transmission of HCV among MSM [65]. Further study and monitoring is required of the possibility that this is also the case for Hong Kong.

61. Although HCV shares similar transmission routes with hepatitis B, the epidemiology of two infections are different in Hong Kong. While HBV is prevalent in the general population in Hong Kong, HCV prevails only in specific populations.

Prevalence of HCV in Populations without Apparent Risk

62. Findings of the seroprevalence studies of the entire spectrum of adult age groups further supported the low prevalence of HCV infection among general population in Hong Kong; given the overall positivity rate for anti-HCV at 0.5% in 382 subjects in 1988 [66], 0.3% in 936

subjects in 2001 (95% CI: 0.07% - 0.94%) (Box 52) and 0.5% in 10 256 subjects in 2016 (95% CI: 0.3% - 0.6%) [9].

63. Data from new blood donors who were mostly adolescents and young adults in the last decade suggested that HCV prevalence was around 0.1% locally, with the figure in 2021 being 0.11% (95% CI: 0.07% - 0.17%) (Box 50). An unusual increase in anti-HCV prevalence was noted in 2020 and 2021, and should be interpreted with the changes in the composition of new blood donors, when the proportion of those aged below 30 decreased from 67.2% in 2019 to 52.4% and 52.2% in 2020 and 2021 respectively (Box 51).

64. The trend of anti-HCV among blood donors has also been monitored. Some 180 000 - 260 000 new and repeated blood donors of HKRCBTS were tested for anti-HCV each year, among which the prevalence was consistently low at less than 0.1% since 2003 (Box 53). The annual number of anti-HCV cases among blood donors ranged between 17 and 43 in the past decade.

65. In an analysis of HCV-positive blood donors during the period from 2003 to 2010, of those with identifiable risk factors, history of blood transfusion (43.7%) was the most common risk factor, followed by intravenous drug use (34.9%) and tattoo (28.6%). The source of infection was unknown in more than half of the respondents in the study [67]. In another study, 14 (30%) HCV-infected blood donors recruited in 2014 - 2016 could be traced to a history of contaminated blood transfusion (n = 9) or injection drug use (n = 5). In donors without identifiable source of infection (n = 32, 70%), high-risk sexual behaviour, body piercing, intramuscular injection and vaccine inoculation abroad and having lived abroad for more than 3 months were associated with HCV infection [68].

Prevalence of HCV in Populations with Undetermined or Apparent Risk

66. From 2000 to 2021, 13 of 3 403 (0.4%) clients who attended the TPC at ITC of CHP, DH for post-exposure management were tested positive for anti-HCV. Eleven (84.6%) cases were non-HCW (Box 54).

67. A study published in the early 1990s has already shown that anti-HCV was more common in injecting drug users (117/175; 66.8%), haemophiliacs (14/25; 56.0%) and haemodialysis patients (3/65; 4.6%) requiring frequent blood/blood product transfusions but not persons at risk through sexual contact [66]. Other local studies also found a higher

infection rate among haemodialysis patients in 1990s (9/51; 18%) [69] and a higher anti-HCV positivity rate among haemophiliacs in a survey in 2011 (100/222; 45%) [70].

68. Injecting drug use has been an important route of HCV acquisition. An HCV seroprevalence study in 2006 conducted in methadone clinics targeting PWID echoed the high prevalence rate of HCV in this community [71]. Of 567 PWID participants recruited in 2006, the prevalence of anti-HCV was 85% (95% CI: 82.5% - 88.3%). Two other studies in 2010s, involving PWID recruited at their gathering places, gave a similar figure of anti-HCV prevalence at 81.7% (95% CI: 78.6% - 84.7%) among 622 subjects in 2011 [72] and 76.4% (95% CI: 73.1% - 79.6%) among 664 subjects in 2014 [73] respectively. In a prospective study initiated in 2021, the overall anti-HCV prevalence was 63% (54/86; 95% CI: 52 – 73%) among methadone clinic attendees, 64% (55/86) of whom had self-reported history of injecting drug use. Of 54 anti-HCV-positive study participants, 78% tested positive for HCV RNA [74]. Injection duration, current or recent injection, ever sharing injecting equipment and concomitant use of other drugs, such as midazolam, were independent factors associated with HCV infection in these studies. In the recent New Life New Liver Project, which provided targeted HCV screening and education to ex-PWID in the community, 73% of 365 subjects screened were anti-HCV positive. The number needed to screen to detect one patient with positive anti-HCV was 1.4 (95% CI: 1.3 - 4.6) [75].

69. HIV/AIDS patients, with a proportion being PWID, is another group with a comparatively high HCV prevalence (Box 55, Box 56). From 2000 to 2021, HCV/HIV coinfection among new patients attending ITC ranged from 1.5% to 24.8%. The decreasing trend of anti-HCV seroprevalence was largely attributed to the decreasing proportion of new patients acquiring HIV via injecting drug use. The prevalence rate appeared to be higher in male than female patients, likely related to the differential risk of parenteral and blood product exposure (Box 55). While HCV infection was present in 1.4 - 6.1% of HIV/AIDS patients infected due to sexual contact, HCV was nearly universal in patients infected through drug injection (Box 56). It should be noted that, among male patients who acquired HIV via heterosexual contact and tested anti-HCV positive, about three fifths (31 out of 55 subjects) had a past history of injecting drug use (Box 56).

70. There has been overseas data supporting sexual transmission of HCV among HIV-positive MSM [76]. The anti-HCV prevalence of subjects who contracted HIV via homosexual or bisexual contact in the ITC HIV/AIDS patient cohort has remained below 2% from screening since 2005. However, this figure has shown an increasing trend since 2012, with the cumulative number of individuals with HCV/HIV coinfection at the time of HIV diagnosis rising from 16 (1.3%) in 2013 to 70 (2.3%) in 2021 (Box 56).

71. From July to November 2013, ITC identified seven cases of recent HCV infection in Chinese HIV-positive MSM without history of injecting drug use [77]. Five of the seven cases were also diagnosed to have recent syphilis infection during the period. Phylogenetic analyses revealed that all cases belonged to the same genotype (genotype 3) although investigation showed no apparent linkage on their sexual exposure. An analysis on HIV-positive MSM attending ITC who had HCV seroconversion in the period 1999 - 2013 was subsequently performed [78]. Fourteen (1.1%) patients seroconverted, with an overall incidence rate of 0.22 per 100 patient-years. The incidence rate increased from 0.13 per 100 patient-years before 2002 to 0.19 per 100 patient years in 2002 - 2007 and 0.47 per 100 patient-years in 2008 - 2013. Compared with the non-seroconverters, the seroconverters were of higher education level and had prior history of STI. As reported in the latest retrospective study, a total of 420 records of HIV/HCV co-infections were identified in ITC between 1999 and February 2021, and the majority of the cases after 2013 were found in MSM [79]. The study also found that HIV/HCV co-infection cases in MSM were more likely to be younger, local residents, achieving HIV viral suppression and co-diagnosing with an STI at HCV diagnosis, and having a longer time lag between HIV and HCV diagnoses, as compared with those in non-MSM. The overall higher HCV prevalence, and the increasing incidence of HCV infection among HIV-positive MSM, coupled with the hastened liver disease progression in patients with HIV infection [80], would demand further attention.

72. A surveillance project for HCV in Hong Kong had been in place to monitor the trend of anti-HCV among selected in-patients, with the participation of the laboratories of Princess Margaret Hospital (PMH, joined since 2003) and Prince of Wales Hospital (PWH, joined since 2005). Among the selected hospital patients tested in the past eleven years, the overall anti-HCV prevalence was 1.8% (Box 57). Anti-HCV was most commonly found in drug users, of which 48.6% were found positive, followed by patients with history of blood transfusion at

8.1%. Overall, the male-to-female ratio of HCV positive subjects was about 2.5 to 1, with a mean age of 54.6 years old (Box 58).

Genotypes of HCV

73. Genotypic studies in Hong Kong has identified that 1b and 6a were the prevalent HCV genotypes locally, a scenario different from that in North America where 1a predominated [81, 82]. In an early study of 212 blood donors tested anti-HCV positive from 1991 to 1994, the commonest genotype found was 1b (58.8%), followed by 6a (27.0%) [83]. In another study of hospitalised patients with HCV testing for clinical indications, 1b was the commonest type found in patients with chronic liver diseases and chronic renal failure [84]. According to a local study of patients on renal replacement therapy, the predominant genotype was 1b, followed by 1a and 6a [85]. As reported in a recent territory-wide population-based study, the commonest HCV genotype was genotype 1 (48.8%), followed by genotype 6 (33.6%) and genotype 3 (10.8%) among 2 699 patients who were tested positive for anti-HCV between January 2005 and March 2017 in public hospitals in Hong Kong [86].

74. The commonest genotype in intravenous drug users was genotype 6. A retrospective analysis of 106 intravenous drug users and 949 non-drug users with samples collected between December 1998 and May 2004 also confirmed the significant high prevalence of genotype 6a in drug users (58.5%) followed by 1b (33.0%), in contrast to 63.6% for 1b and 23.6% for 6a in non-drug users [87]. Besides intravenous drug use, age and sex were independent factors associated with HCV genotypes in this study. Further phylogenetic analyses revealed that HCV 6a strains from Vietnam might be ancestral to Hong Kong counterparts, suggesting an association between the high predominance of HCV 6a infections and Vietnamese immigration during 1987 - 1997 in Hong Kong [88]. In a methadone clinic-based study published in 2011, out of 273 PWID with different periods of initiating injection, 52% had genotype 6a and 38% had 1b. Both genotypes 1b and 6a were prevalent among older injectors, while subtype 3a was more common in young injectors and those initiating injection more recently during 1995 - 2006. Moreover, phylogenetic analysis revealed no specific clustering of any subtype or genotype, which did not suggest any outbreak of HCV among the study population. The extensive use of methadone, widely available since 1980s, may have protected Hong Kong from the emergence of HCV clusters among injection drug users [89].

75. For the HIV-positive MSM attending ITC who were diagnosed with acute HCV infection between 2009 to 2014, genotype 3a was the most prevalent (63.6%), followed by 1a (18.2%) and 6a (9.1%). The high prevalence of genotype 3a in MSM was in stark contrast to its rarity among HCV-infected PWID in Hong Kong. Phylogenetic analyses revealed a monophyletic HCV-3a cluster with members all diagnosed between 2013 and 2014, and a homologous pair with HCV-6a genotype. However, there was no temporal or genetic clustering of the corresponding HIV sequences [90]. Molecular analyses of HCV sequences from 58 HIV-positive patients from ITC between 2010 and 2016 also showed no international network of HCV among HIV-positive MSM in the three Asia-Pacific cities, namely Hong Kong, Taipei and Tokyo [91]. An overview of all 420 HIV/HCV co-infected patients between 1999 and February 2021 in ITC found that MSM were more likely to be associated with HCV genotype 3, compared to genotypes 1 and 6 in non-MSM [79].

76. The natural history of 138 HCV genotype 1 patients (median age: 50 years) was compared with that of 78 HCV genotype 6 patients (median age: 46.5 years) by reviewing medical records of anti-HCV-positive patients in Queen Mary Hospital between 1991 and 2007 [92]. Both genotypes share a similar natural history based on liver biochemistry, HCV viral load, and probability of cirrhotic complications and mortality after a median follow-up period of over 5 years.

Liver Cancer

Major Morbidity and Mortality from Viral Hepatitis

77. Chronic HBV and HCV infection are important risk factors for cirrhosis and liver cancer. Globally 830 000 people died of liver cancer in 2020 [93], and HBV and HCV infection generally accounted for approximately 80% of liver cancer cases [94]. Local studies showed that 75 - 80% of hepatocellular cancers in Hong Kong were related to chronic HBV infection, and 3 - 6% of the cases were related to chronic HCV infection. HBV and HCV co-infection accounted for another 0.4 - 3% [95].

78. Among 76 liver transplants performed in Queen Mary Hospital (QMH) due to cirrhosis from 1999 to 2000, 51 and 7 were related to hepatitis B and C respectively [96]. Another case series report showed that more than half (800; 58.6%) of the 1366 patients undergoing liver transplantation between 1999 and 2019 in QMH had indications related to complications of chronic hepatitis B, including cirrhosis, HCC and severe flares; while the proportion of transplanted patients with chronic hepatitis B was declining from a peak of 76.7% in 2002 to 44.4% in 2019 [97].

79. According to the data from the Hong Kong Cancer Registry [98], liver cancer, including neoplasm of liver and intrahepatic bile ducts, was the fourth commonest cancer in men and eleventh commonest cancer in women in 2020. There were 1 735 newly registered cases of liver cancer, with 1 261 cases of males and 474 cases of females (male to female ratio was about 2.7 to 1) in 2020. There was a downward trend for the age-standardised incidence rate for both male and female in the past decade (Box 59, Box 60). The figures were 18.2 for male and 5.6 for female per 100 000 standard population in 2020.

80. In 2020, liver cancer was the third leading cause of cancer deaths in Hong Kong. There were 1 530 registered mortality from liver cancer. There was a downward trend for the age-standardised mortality rate for both sexes in the past decade (Box 61, Box 62). The figures were 15.3 for male and 4.4 for female per 100 000 standard population in 2020 [98].

SURVEILLANCE INFORMATION

Acute viral hepatitis

(Data source: Centre for Health Protection, Department of Health)

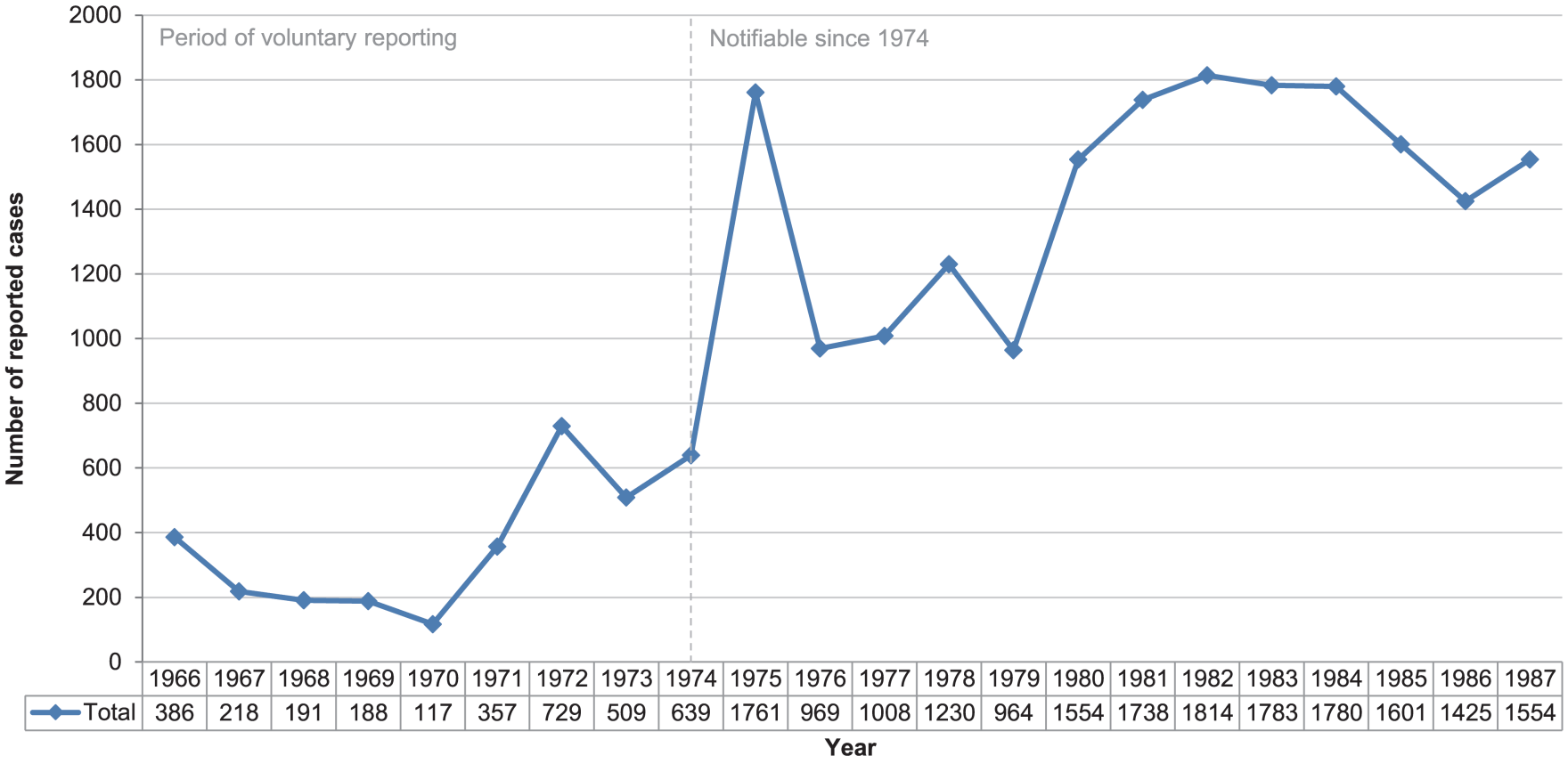
Box 1

Number of cases of viral hepatitis reported to the Department of Health between 1988 and 2021 (Data source: CHP, DH)

Year	A	B	NANB	C	D	E	Unclassified	Hepatitis (not elsewhere classified)	**Total**
1988	1187	250	465				496		**2398**
1989	618	136	154				324		**1232**
1990	1362	178	183				261		**1984**
1991	1297	150	200				154		**1801**
1992	3626	157	301				273		**4357**
1993	874	116	203				80		**1273**
1994	557	112	125				41		**835**
1995	491	102	55				18		**666**
1996	264	144	-	-	-	11	-	58	**477**
1997	595	100	-	-	-	4	-	37	**736**
1998	474	145	-	-	-	16	-	29	**664**
1999	426	152	-	-	-	8	-	31	**617**
2000	505	137	-	-	-	11	-	30	**683**
2001	494	134	-	-	-	26	-	23	**677**
2002	267	121	-	4	-	28	-	10	**430**
2003	107	98	-	-	-	19	-	8	**232**
2004	121	134	-	1	-	38	-	6	**300**
2005	64	105	-	1	-	34	-	0	**204**
2006	76	123	-	2	-	34	-	0	**235**
2007	69	74	-	1	-	65	-	0	**209**
2008	71	83	-	3	-	90	-	-	**247**
2009	64	80	-	3	-	73	-	-	**220**
2010	65	73	-	11	-	118	-	-	**267**
2011	46	70	-	5	-	119	-	-	**240**
2012	43	47	-	3	-	150	-	-	**243**
2013	44	40	-	10	-	90	-	-	**184**
2014	46	41	-	12	-	93	-	-	**192**
2015	138	29	-	14	-	84	-	-	**265**
2016	98	37	-	39	-	96	-	-	**270**
2017	117	33	-	18	-	64	-	-	**232**
2018	50	29	-	34	-	43	-	-	**156**
2019	79	28	-	17	-	85	-	-	**209**
2020	28	17	-	35	1	80	-	-	**161**
2021	15	17	-	23	1	77	-	-	**133**

Box 2

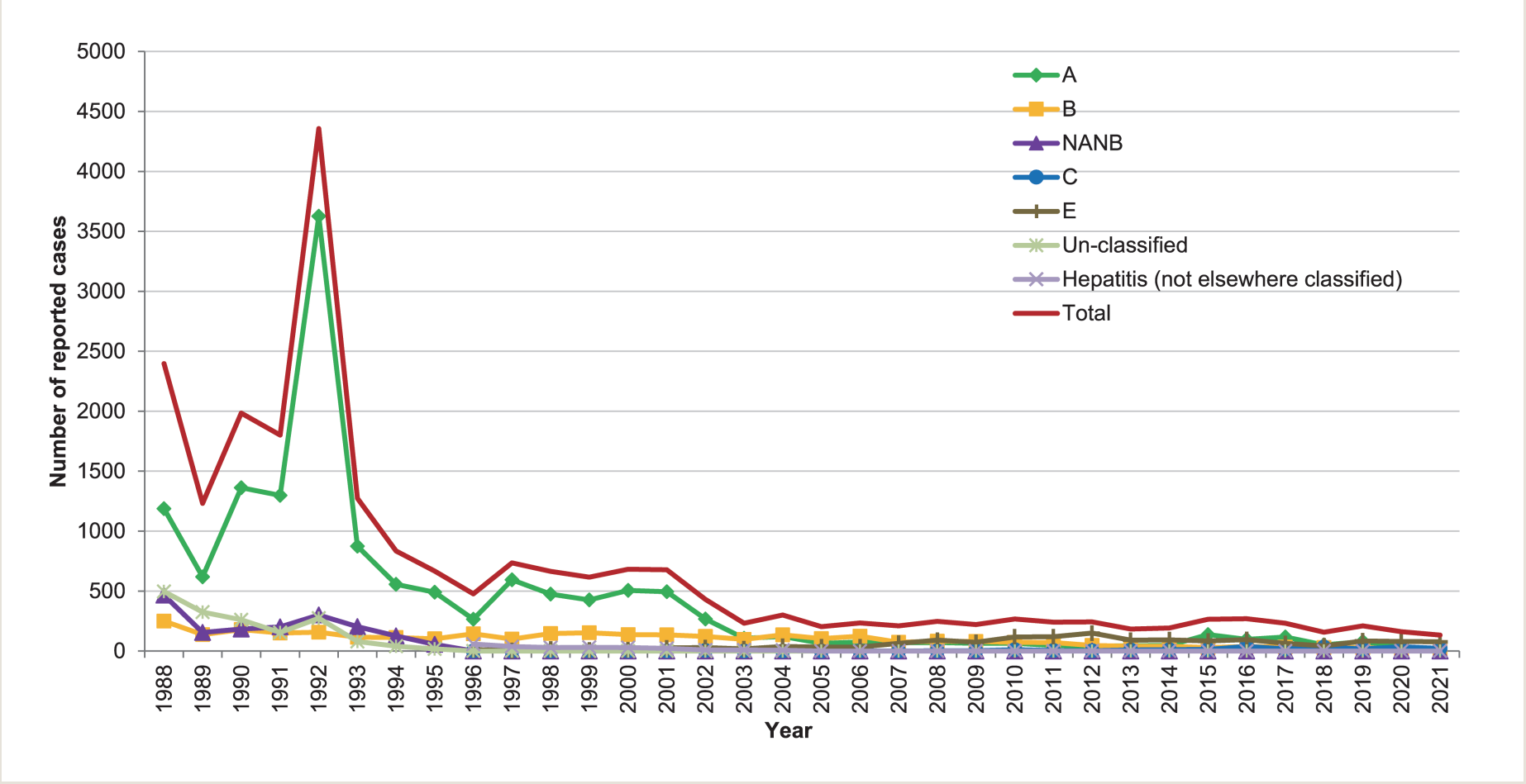
Reported cases of viral hepatitis from 1966 to 1987 by syndromic surveillance (Data source: CHP, DH)



	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987
Total	386	218	191	188	117	357	729	509	639	1761	969	1008	1230	964	1554	1738	1814	1783	1780	1601	1425	1554

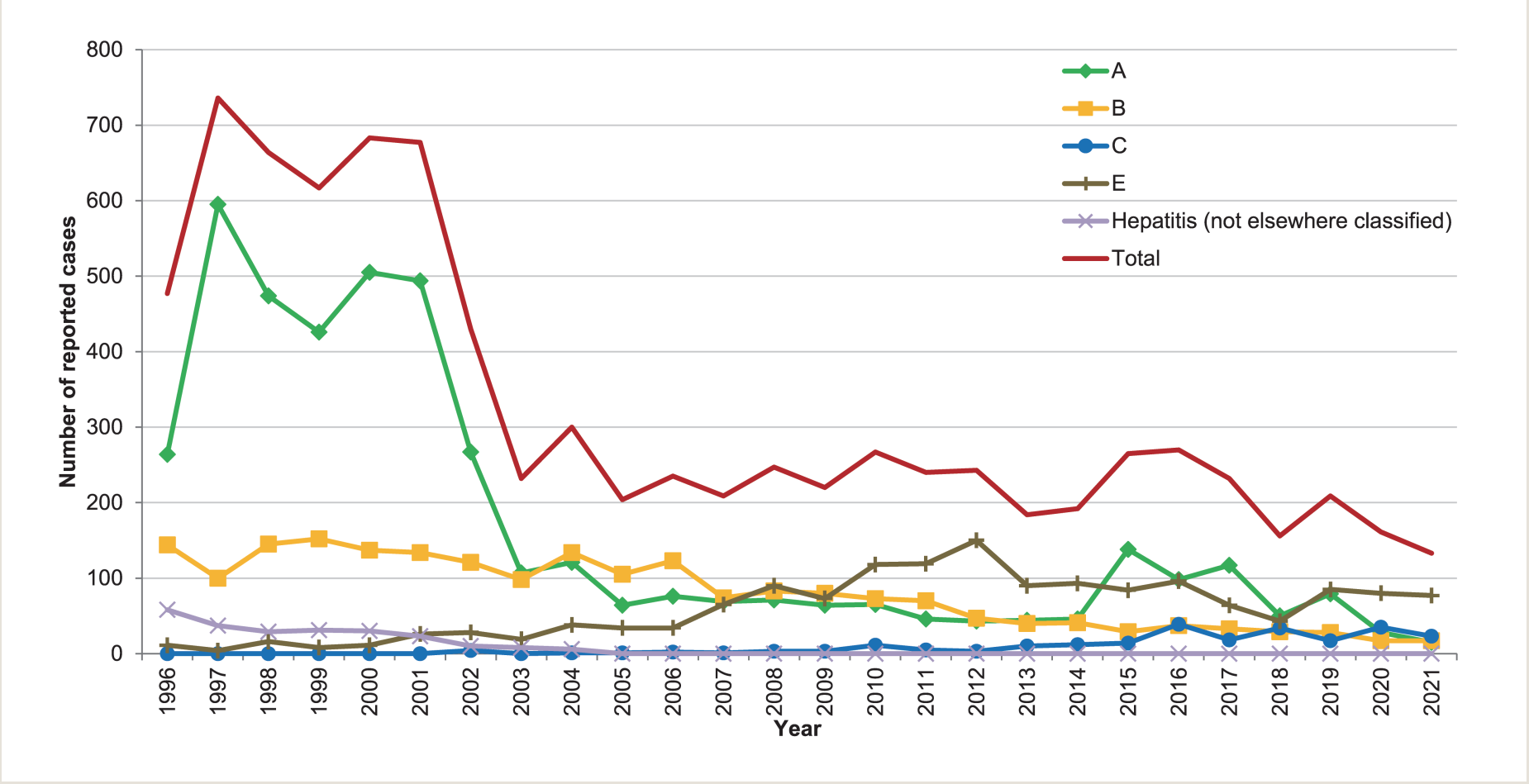
Box 3

Reported cases of viral hepatitis from 1988 to 2021 by viral etiology surveillance (Data source: CHP, DH)



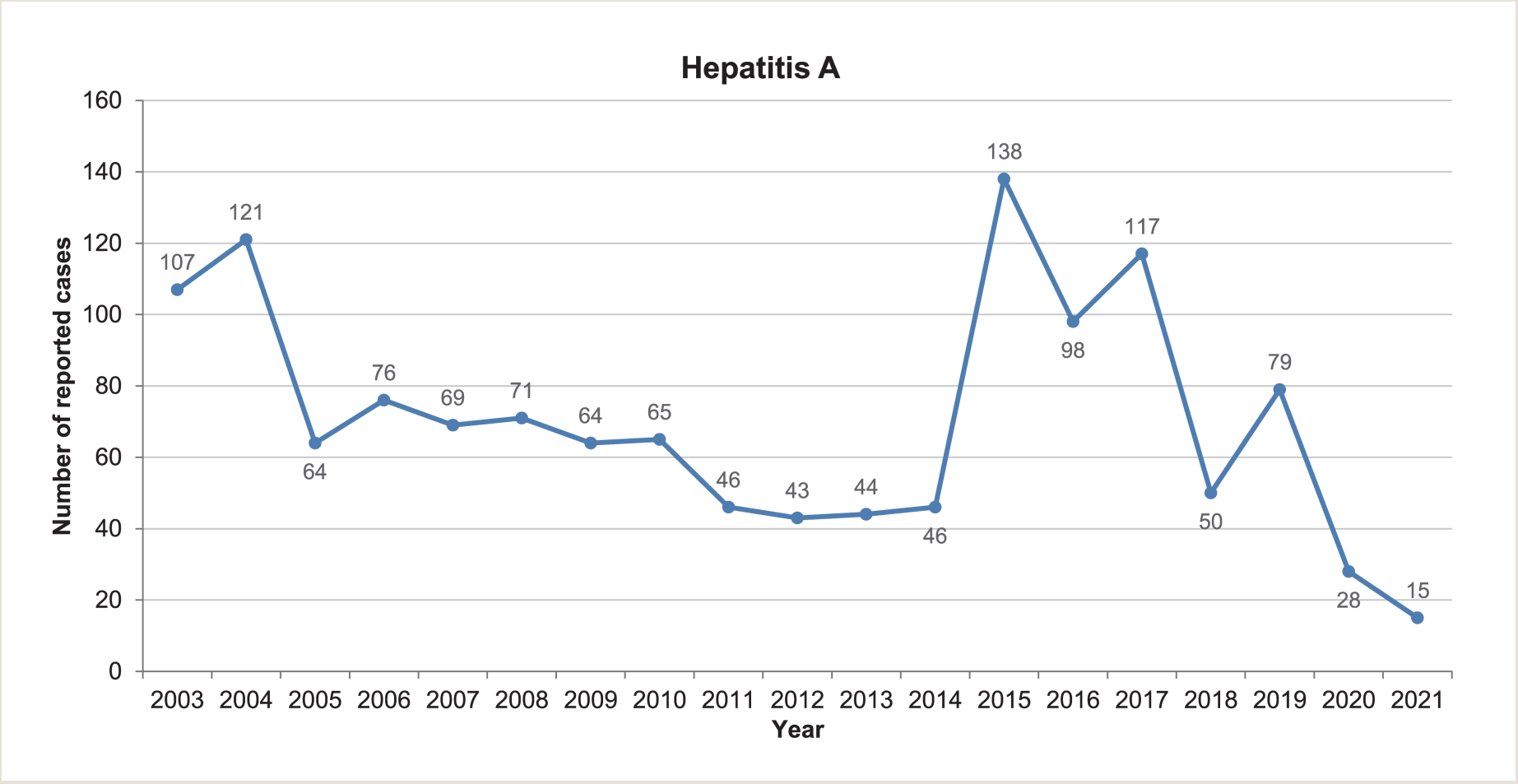
Box 4

Breakdown of viral hepatitis by etiology reported from 1996 to 2021 (Data source: CHP, DH)



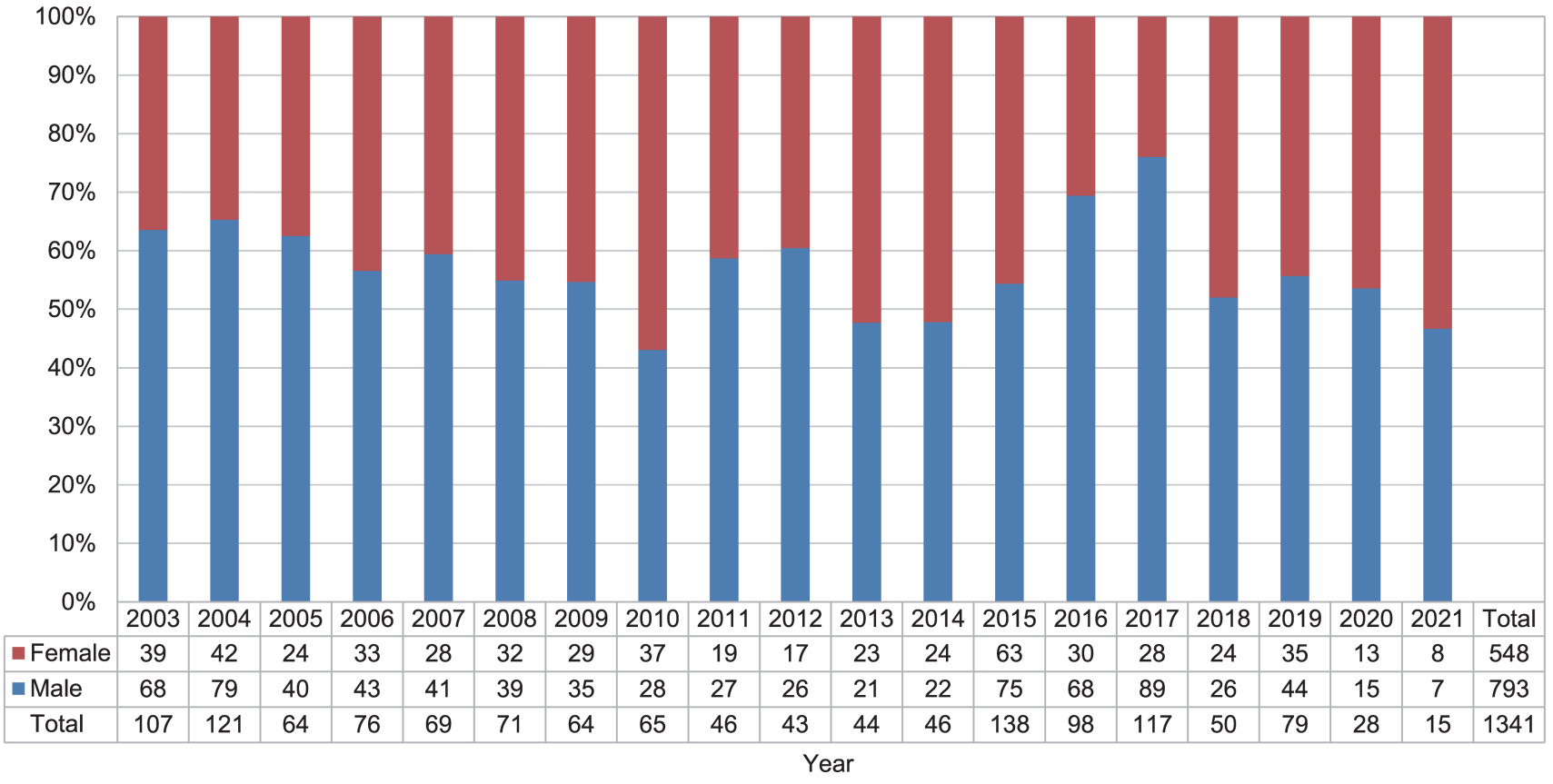
Box 5

Number of hepatitis A cases reported from 2003 to 2021 (Data source: CHP, DH)



Box 6

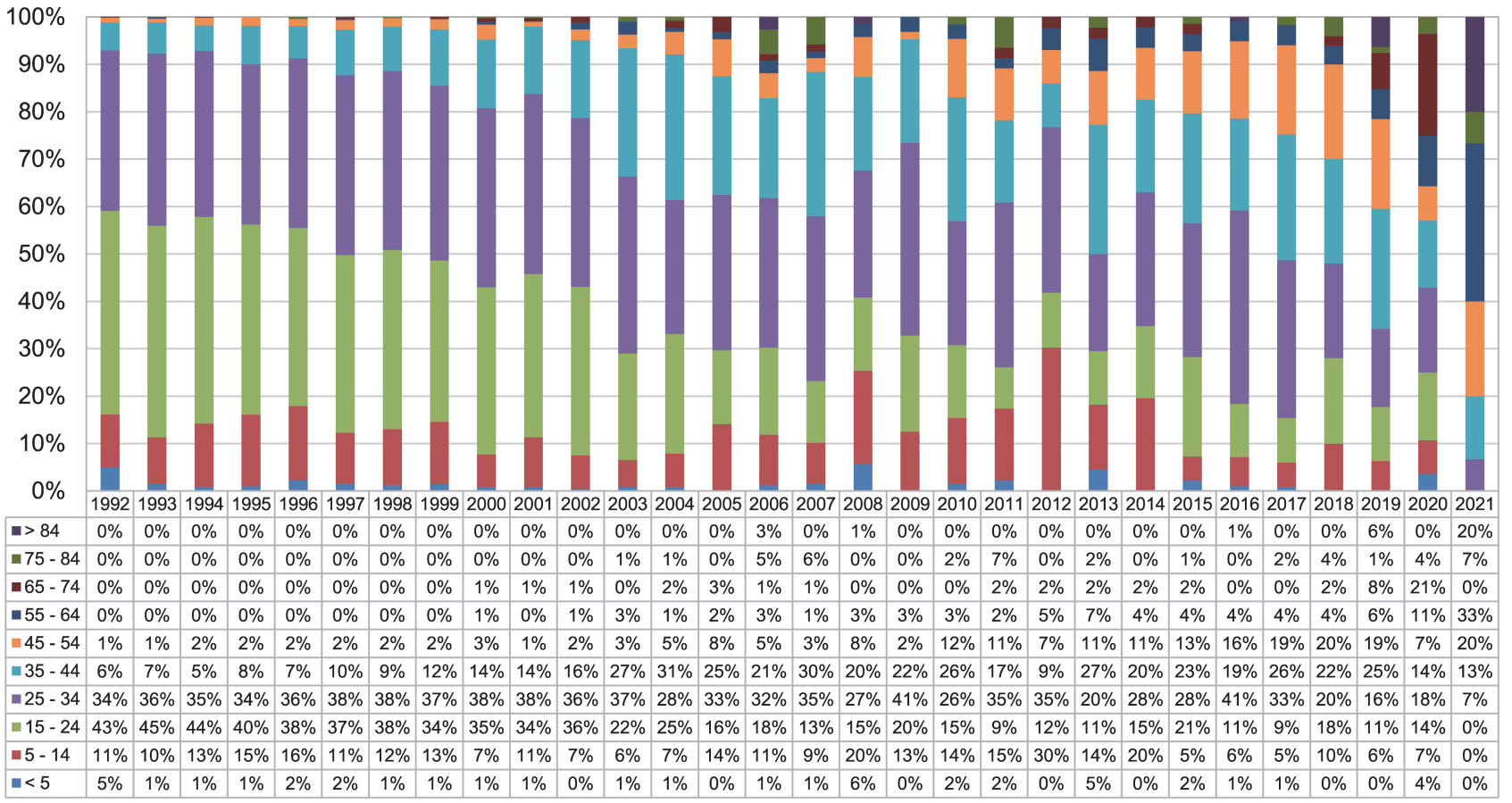
Sex distribution of hepatitis A cases reported from 2003 to 2021 (Data source: CHP, DH)



	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	Total
Female	39	42	24	33	28	32	29	37	19	17	23	24	63	30	28	24	35	13	8	548
Male	68	79	40	43	41	39	35	28	27	26	21	22	75	68	89	26	44	15	7	793
Total	107	121	64	76	69	71	64	65	46	43	44	46	138	98	117	50	79	28	15	1341

Box 7

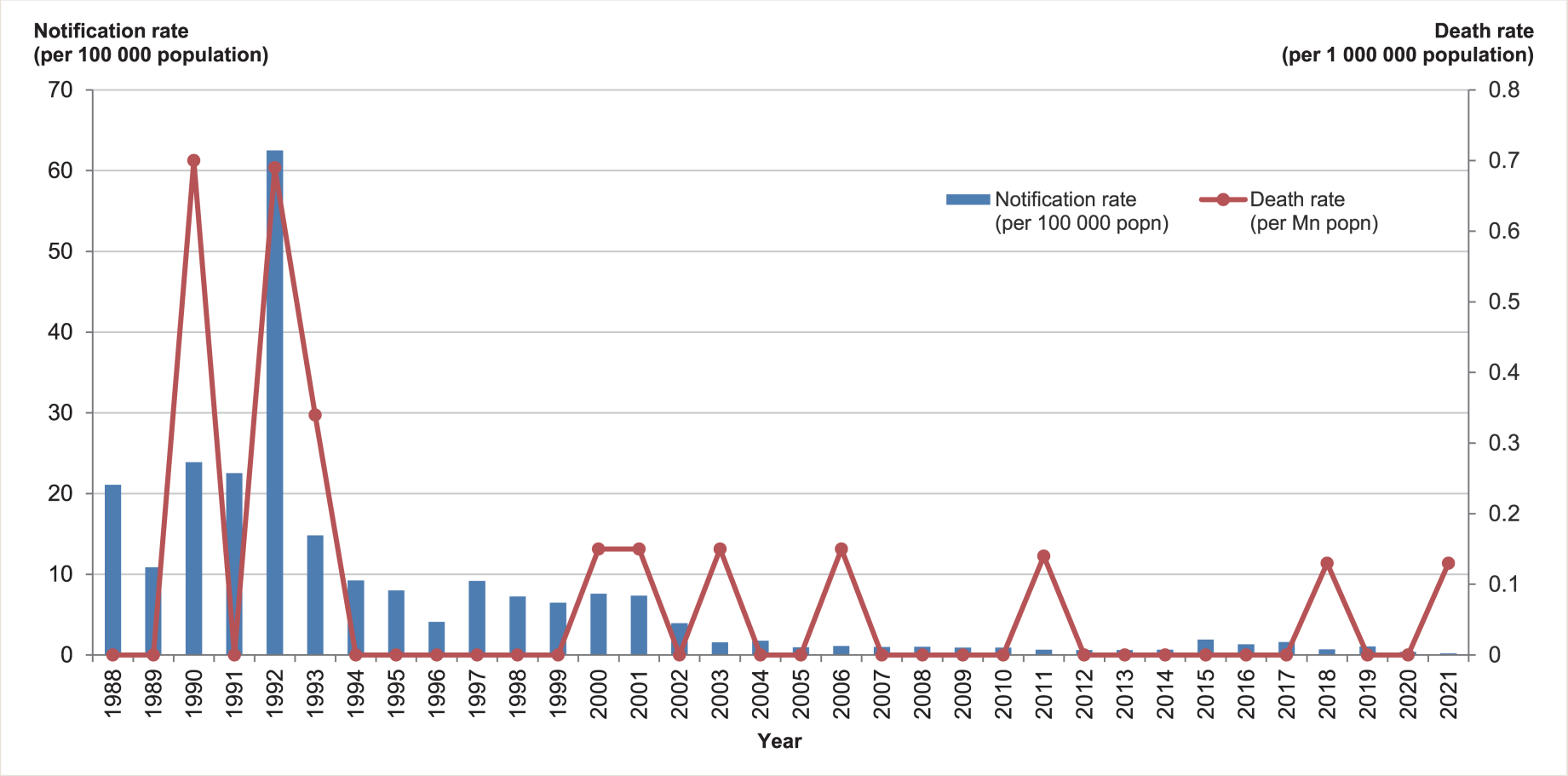
Age distribution of hepatitis A cases reported from 1992 to 2021 (Data source: CHP, DH)



	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
> 84	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	3%	0%	1%	0%	0%	0%	0%	0%	0%	0%	1%	0%	0%	6%	0%	20%
75 - 84	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	1%	1%	0%	5%	6%	0%	0%	2%	7%	0%	2%	0%	1%	0%	2%	4%	1%	4%	7%
65 - 74	0%	0%	0%	0%	0%	0%	0%	0%	1%	1%	1%	0%	2%	3%	1%	1%	0%	0%	0%	2%	2%	2%	2%	2%	0%	0%	2%	8%	21%	0%
55 - 64	0%	0%	0%	0%	0%	0%	0%	0%	1%	0%	1%	3%	1%	2%	3%	1%	3%	3%	3%	2%	5%	7%	4%	4%	4%	4%	4%	6%	11%	33%
45 - 54	1%	1%	2%	2%	2%	2%	2%	2%	3%	1%	2%	3%	5%	8%	5%	3%	8%	2%	12%	11%	7%	11%	11%	13%	16%	19%	20%	19%	7%	20%
35 - 44	6%	7%	5%	8%	7%	10%	9%	12%	14%	14%	16%	27%	31%	25%	21%	30%	20%	22%	26%	17%	9%	27%	20%	23%	19%	26%	22%	25%	14%	13%
25 - 34	34%	36%	35%	34%	36%	38%	38%	37%	38%	38%	36%	37%	28%	33%	32%	35%	27%	41%	26%	35%	35%	20%	28%	28%	41%	33%	20%	16%	18%	7%
15 - 24	43%	45%	44%	40%	38%	37%	38%	34%	35%	34%	36%	22%	25%	16%	18%	13%	15%	20%	15%	9%	12%	11%	15%	21%	11%	9%	18%	11%	14%	0%
5 - 14	11%	10%	13%	15%	16%	11%	12%	13%	7%	11%	7%	6%	7%	14%	11%	9%	20%	13%	14%	15%	30%	14%	20%	5%	6%	5%	10%	6%	7%	0%
< 5	5%	1%	1%	1%	2%	2%	1%	1%	1%	1%	0%	1%	1%	0%	1%	1%	6%	0%	2%	2%	0%	5%	0%	2%	1%	1%	0%	0%	4%	0%

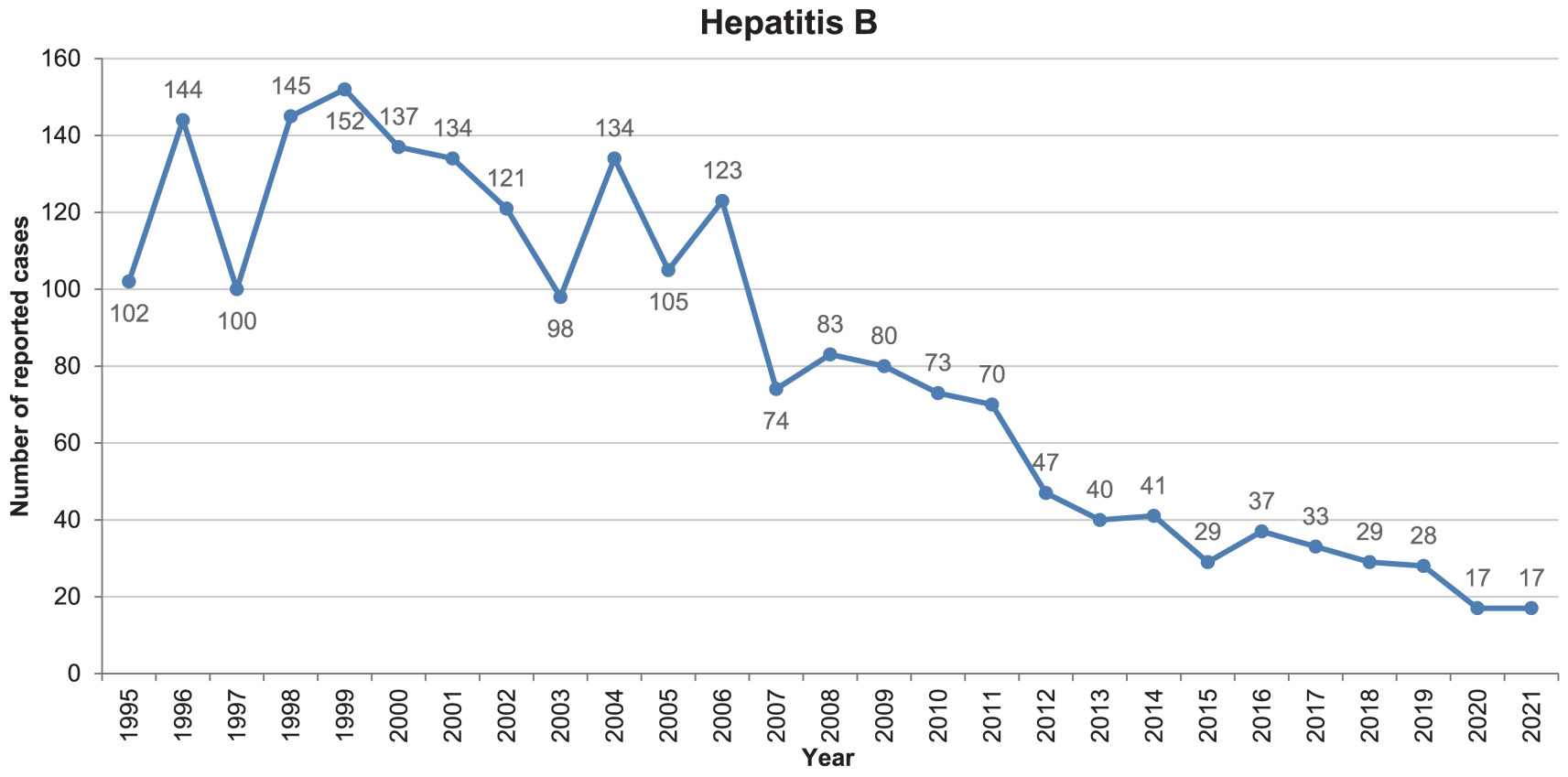
Box 8

Notification rates and death rates of hepatitis A from 1988 to 2021 (Data source: CHP, DH)



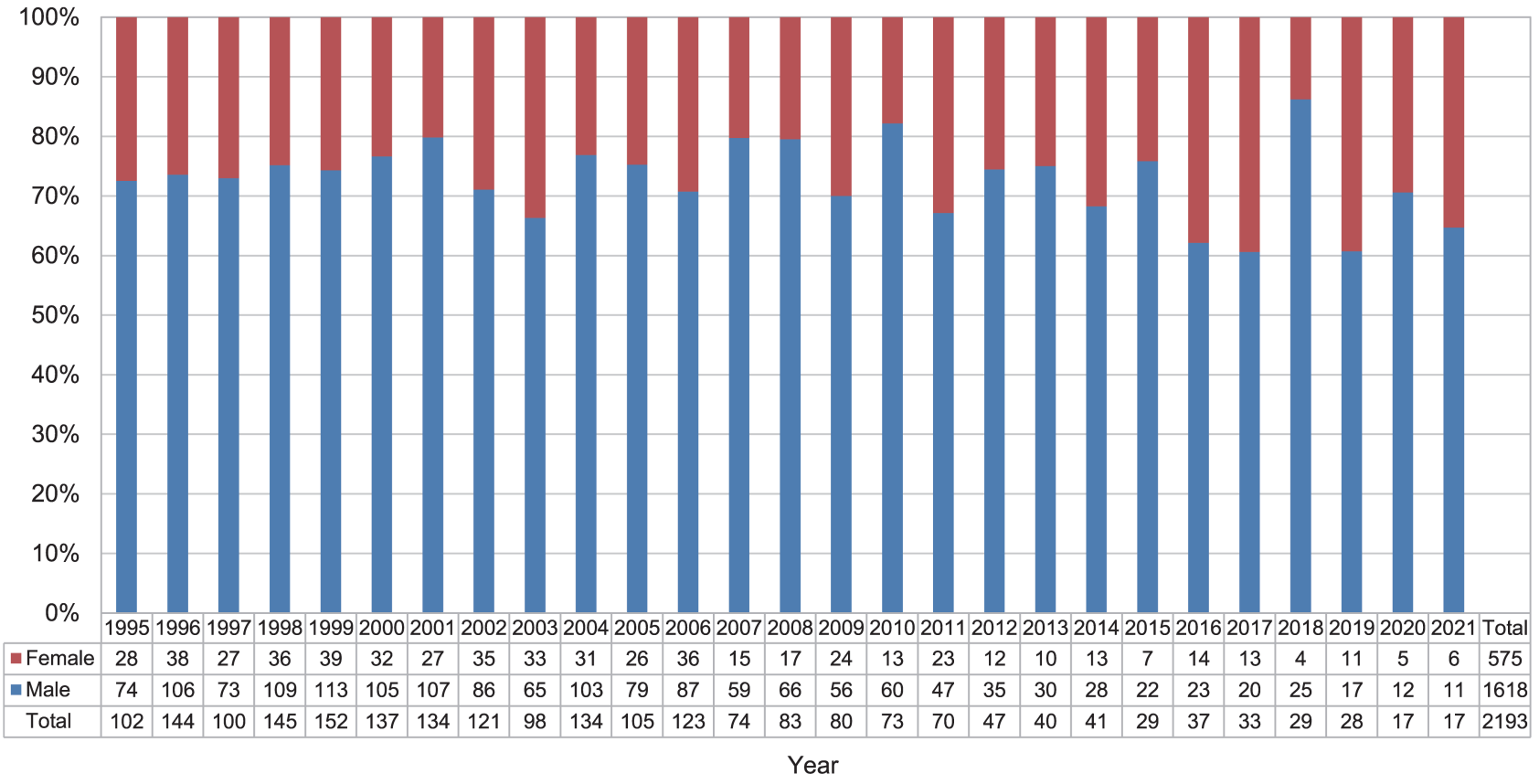
Box 9

Number of hepatitis B cases reported from 1995 to 2021 (Data source: CHP, DH)



Box 10

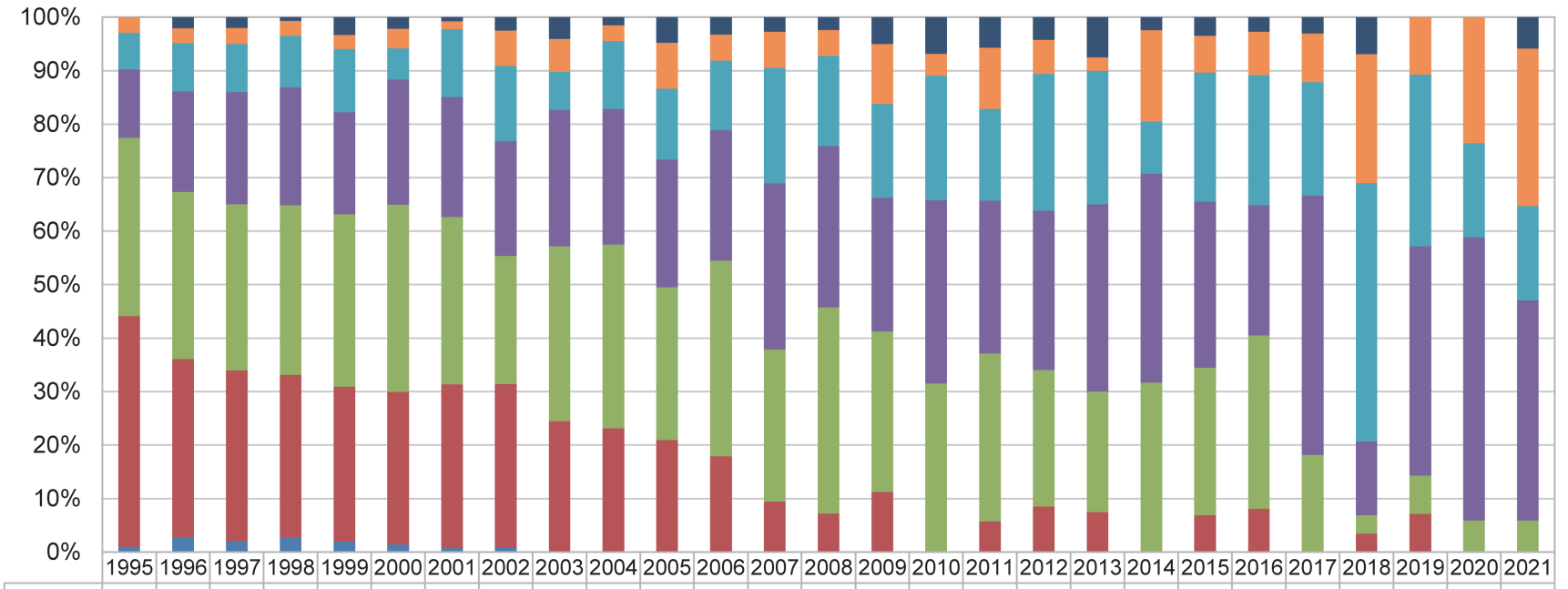
Sex distribution of hepatitis B cases reported from 1995 to 2021 (Data source: CHP, DH)



	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	Total
Female	28	38	27	36	39	32	27	35	33	31	26	36	15	17	24	13	23	12	10	13	7	14	13	4	11	5	6	575
Male	74	106	73	109	113	105	107	86	65	103	79	87	59	66	56	60	47	35	30	28	22	23	20	25	17	12	11	1618
Total	102	144	100	145	152	137	134	121	98	134	105	123	74	83	80	73	70	47	40	41	29	37	33	29	28	17	17	2193

Box 11

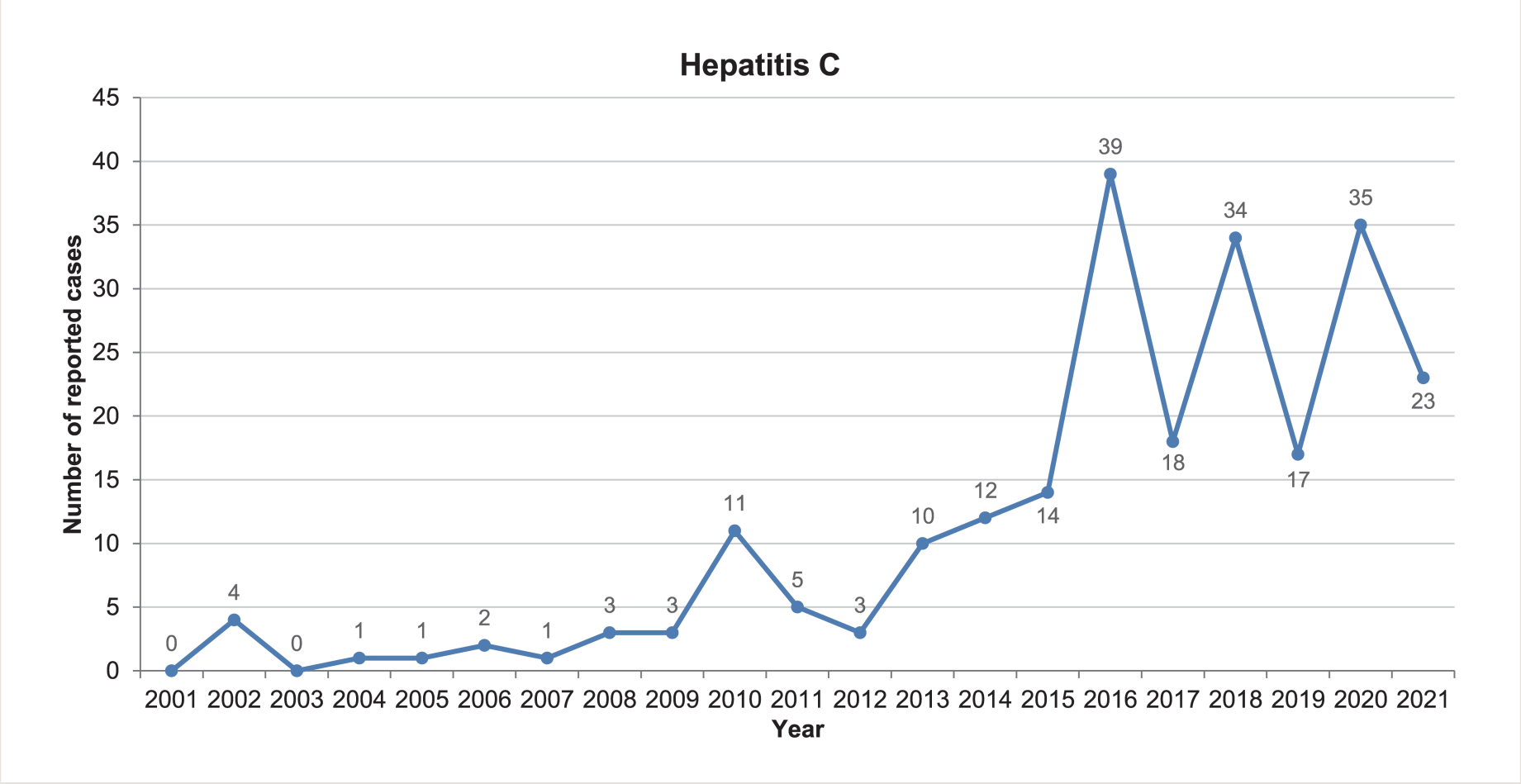
Age distribution of hepatitis B cases reported from 1995 to 2021 (Data source: CHP, DH)



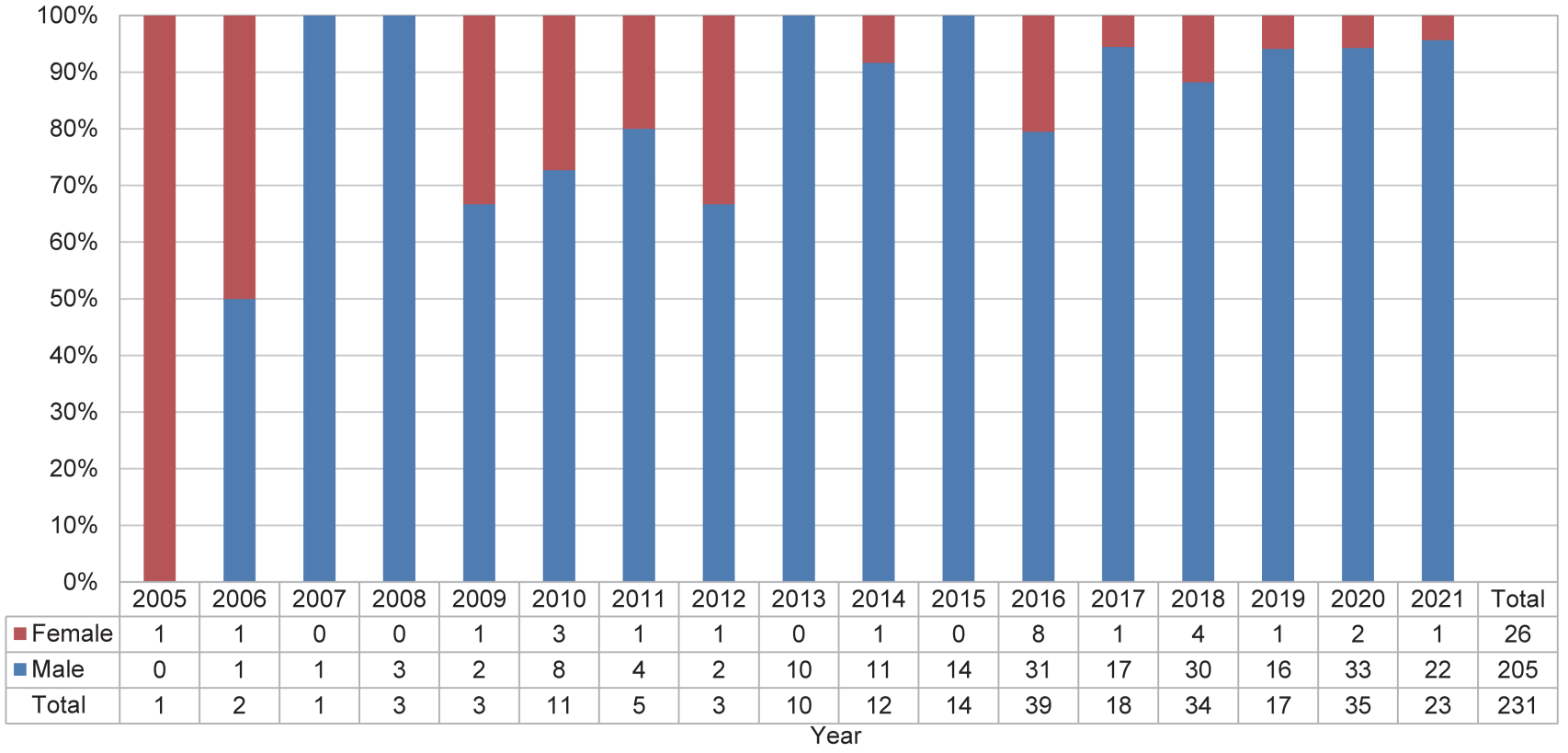
	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
>= 65	0%	2%	2%	1%	3%	2%	1%	2%	4%	1%	5%	3%	3%	2%	5%	7%	6%	4%	8%	2%	3%	3%	3%	7%	0%	0%	6%
55 - 64	3%	3%	3%	3%	3%	4%	1%	7%	6%	3%	9%	5%	7%	5%	11%	4%	11%	6%	3%	17%	7%	8%	9%	24%	11%	24%	29%
45 - 54	7%	9%	9%	10%	12%	6%	13%	14%	7%	13%	13%	13%	22%	17%	18%	23%	17%	26%	25%	10%	24%	24%	21%	48%	32%	18%	18%
35 - 44	13%	19%	21%	22%	19%	23%	22%	21%	26%	25%	24%	24%	31%	30%	25%	34%	29%	30%	35%	39%	31%	24%	48%	14%	43%	53%	41%
25 - 34	33%	31%	31%	32%	32%	35%	31%	24%	33%	34%	29%	37%	28%	39%	30%	32%	31%	26%	23%	32%	28%	32%	18%	3%	7%	6%	6%
15 - 24	43%	33%	32%	30%	29%	28%	31%	31%	24%	23%	21%	18%	9%	7%	11%	0%	6%	9%	8%	0%	7%	8%	0%	3%	7%	0%	0%
<1 - 14	1%	3%	2%	3%	2%	1%	1%	1%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%

Box 12

Number of hepatitis C cases reported from 2002 to 2021 (Data source: CHP, DH)



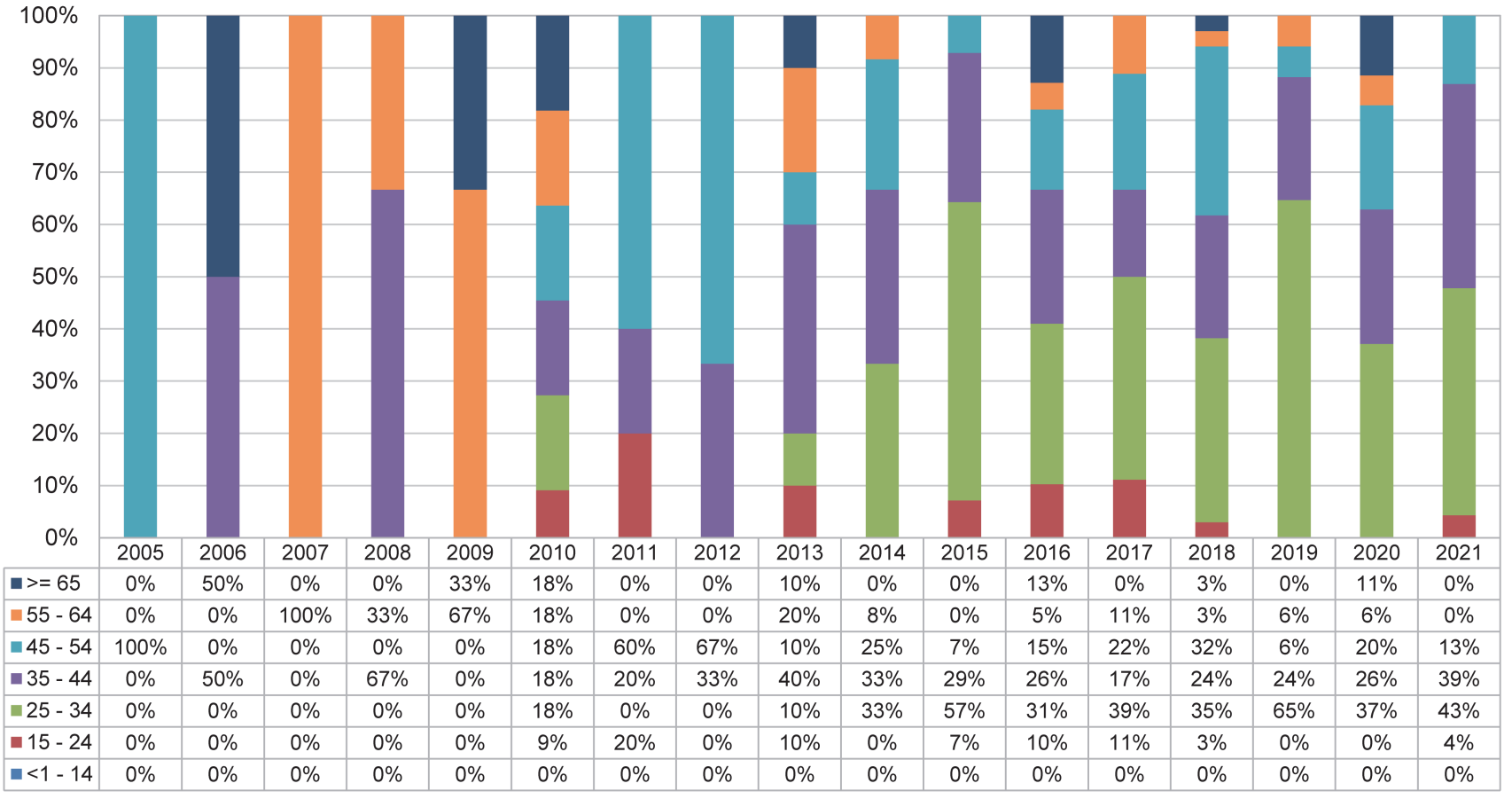
Box 13 Sex distribution of hepatitis C cases reported from 2005 to 2021 (Data source: CHP, DH)



	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	Total
Female	1	1	0	0	1	3	1	1	0	1	0	8	1	4	1	2	1	26
Male	0	1	1	3	2	8	4	2	10	11	14	31	17	30	16	33	22	205
Total	1	2	1	3	3	11	5	3	10	12	14	39	18	34	17	35	23	231

Box 14

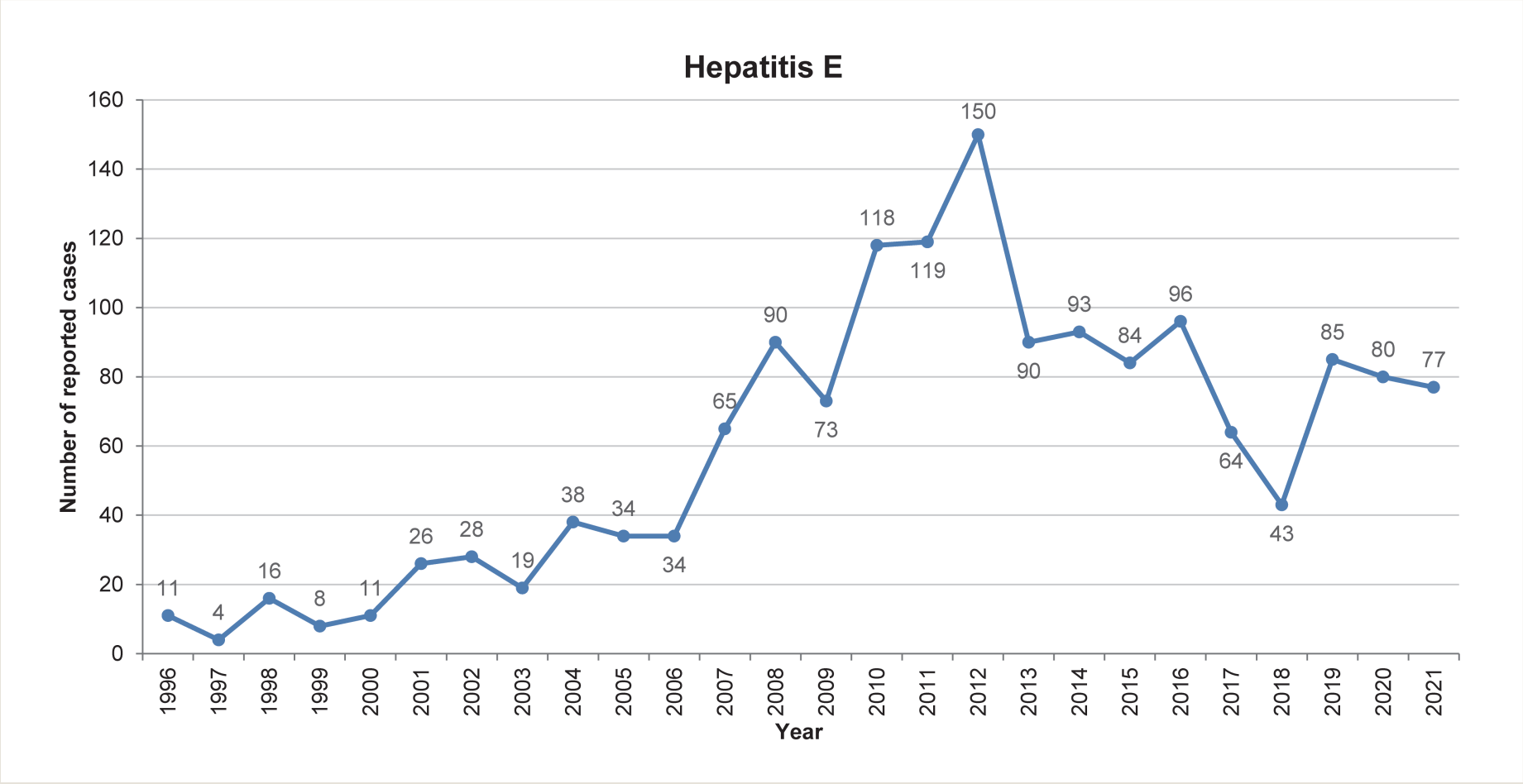
Age distribution of hepatitis C cases reported from 2005 to 2021 (Data source: CHP, DH)



	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
>= 65	0%	50%	0%	0%	33%	18%	0%	0%	10%	0%	0%	13%	0%	3%	0%	11%	0%
55 - 64	0%	0%	100%	33%	67%	18%	0%	0%	20%	8%	0%	5%	11%	3%	6%	6%	0%
45 - 54	100%	0%	0%	0%	0%	18%	60%	67%	10%	25%	7%	15%	22%	32%	6%	20%	13%
35 - 44	0%	50%	0%	67%	0%	18%	20%	33%	40%	33%	29%	26%	17%	24%	24%	26%	39%
25 - 34	0%	0%	0%	0%	0%	18%	0%	0%	10%	33%	57%	31%	39%	35%	65%	37%	43%
15 - 24	0%	0%	0%	0%	0%	9%	20%	0%	10%	0%	7%	10%	11%	3%	0%	0%	4%
<1 - 14	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%

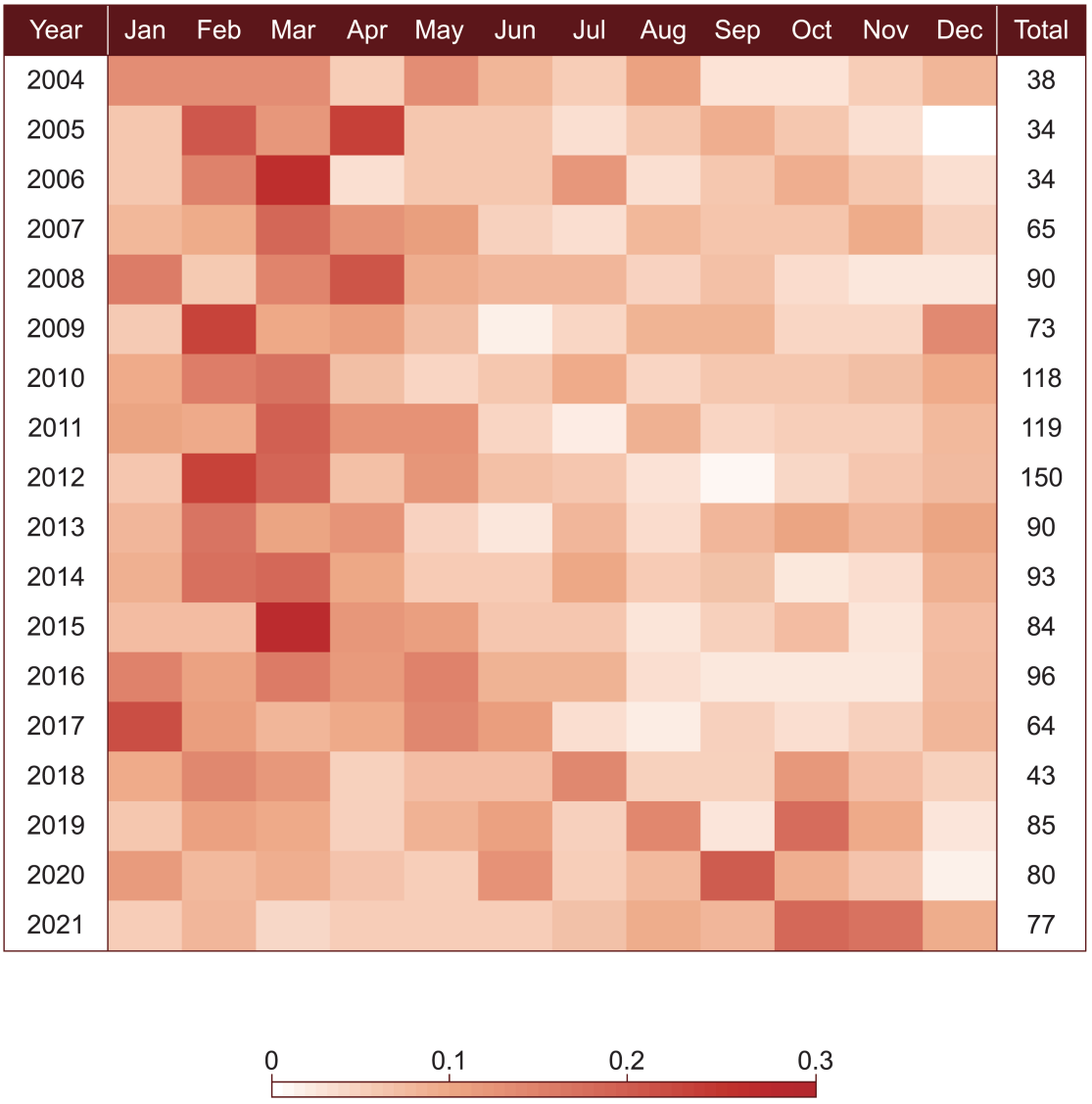
Box 15

Number of hepatitis E cases reported from 1996 to 2021 (Data source: CHP, DH)



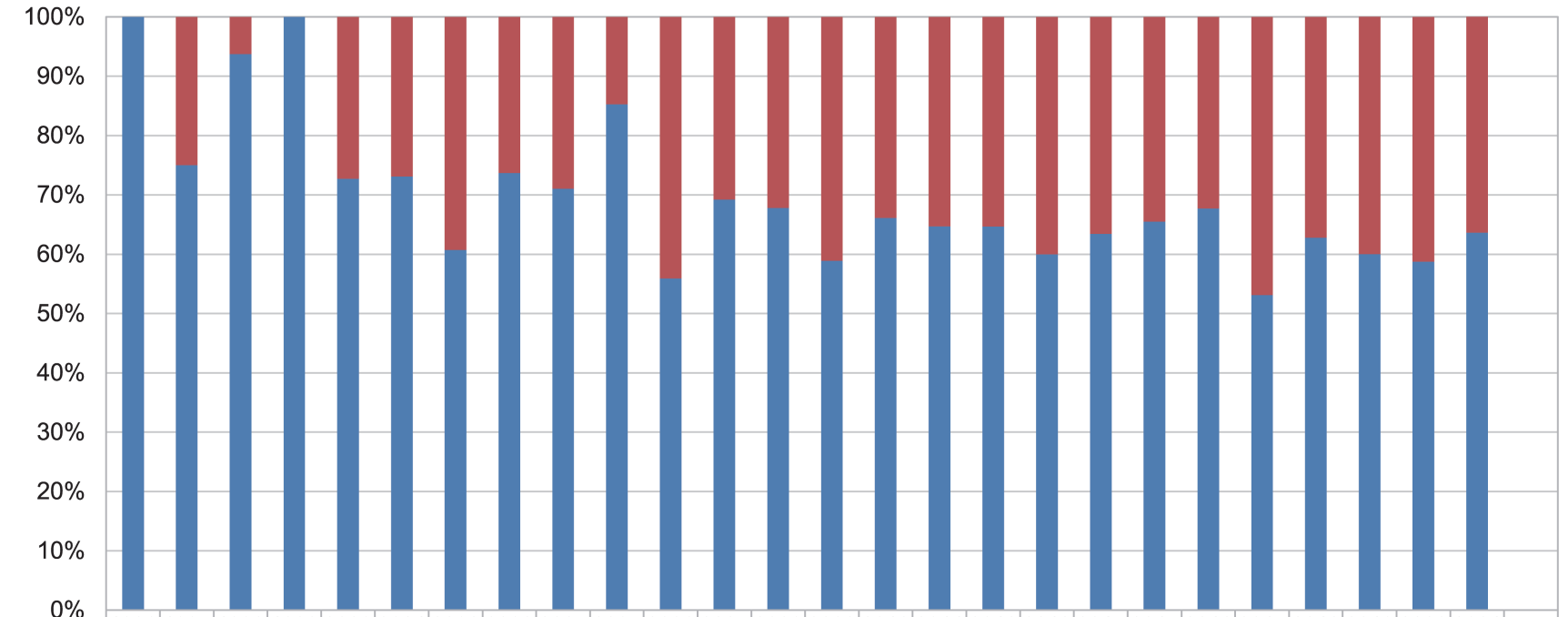
Box 16

Seasonal distribution of reported cases of hepatitis E by month from 2004 to 2021 (Data source: CHP, DH)



Box 17

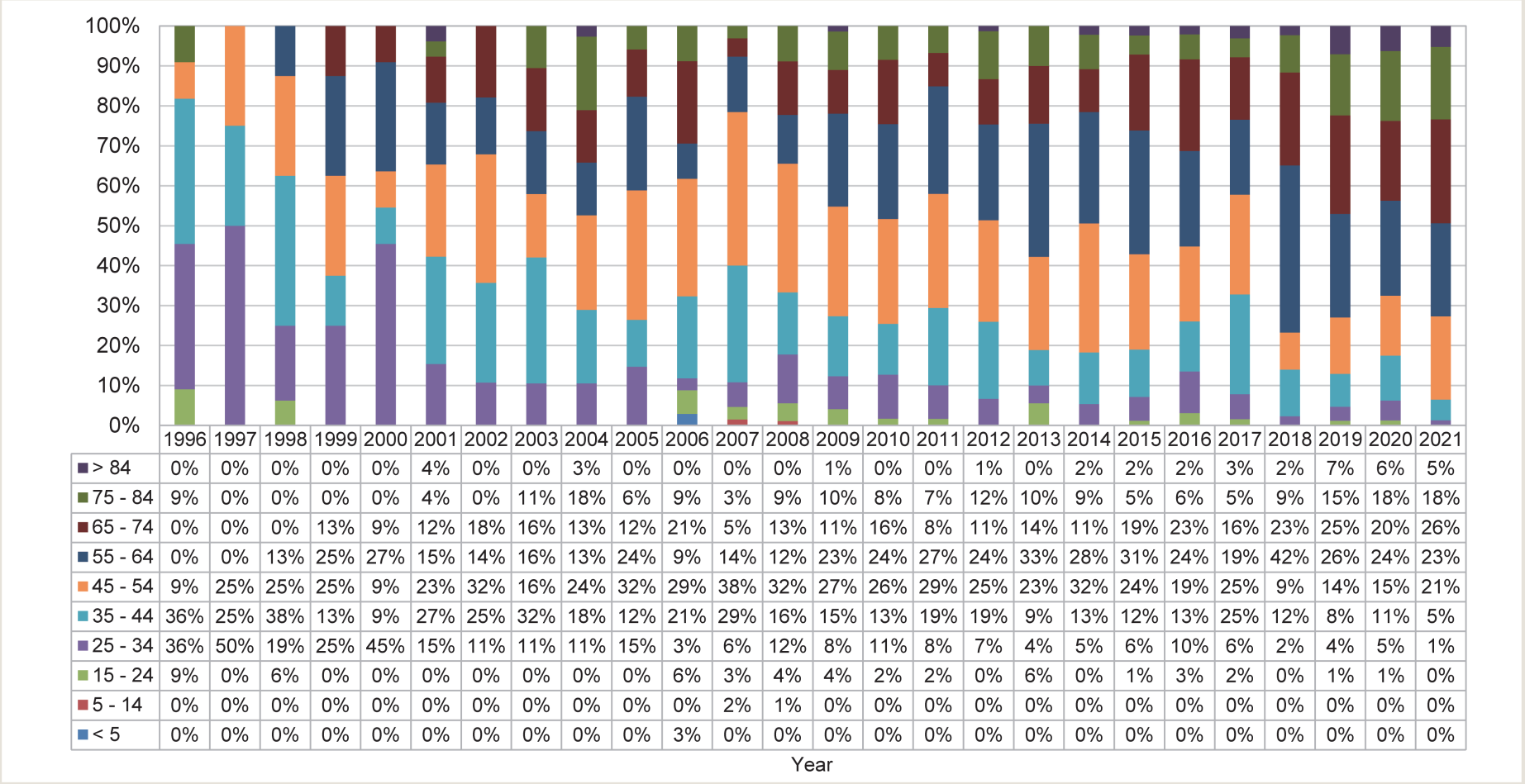
Sex distribution of hepatitis E cases reported from 1996 to 2021 (Data source: CHP, DH)



	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	Total
Female	0	1	1	0	3	7	11	5	11	5	15	20	29	30	40	42	53	36	34	29	31	30	16	34	33	28	544
Male	11	3	15	8	8	19	17	14	27	29	19	45	61	43	78	77	97	54	59	55	65	34	27	51	47	49	1012
Total	11	4	16	8	11	26	28	19	38	34	34	65	90	73	118	119	150	90	93	84	96	64	43	85	80	77	1556

Box 18

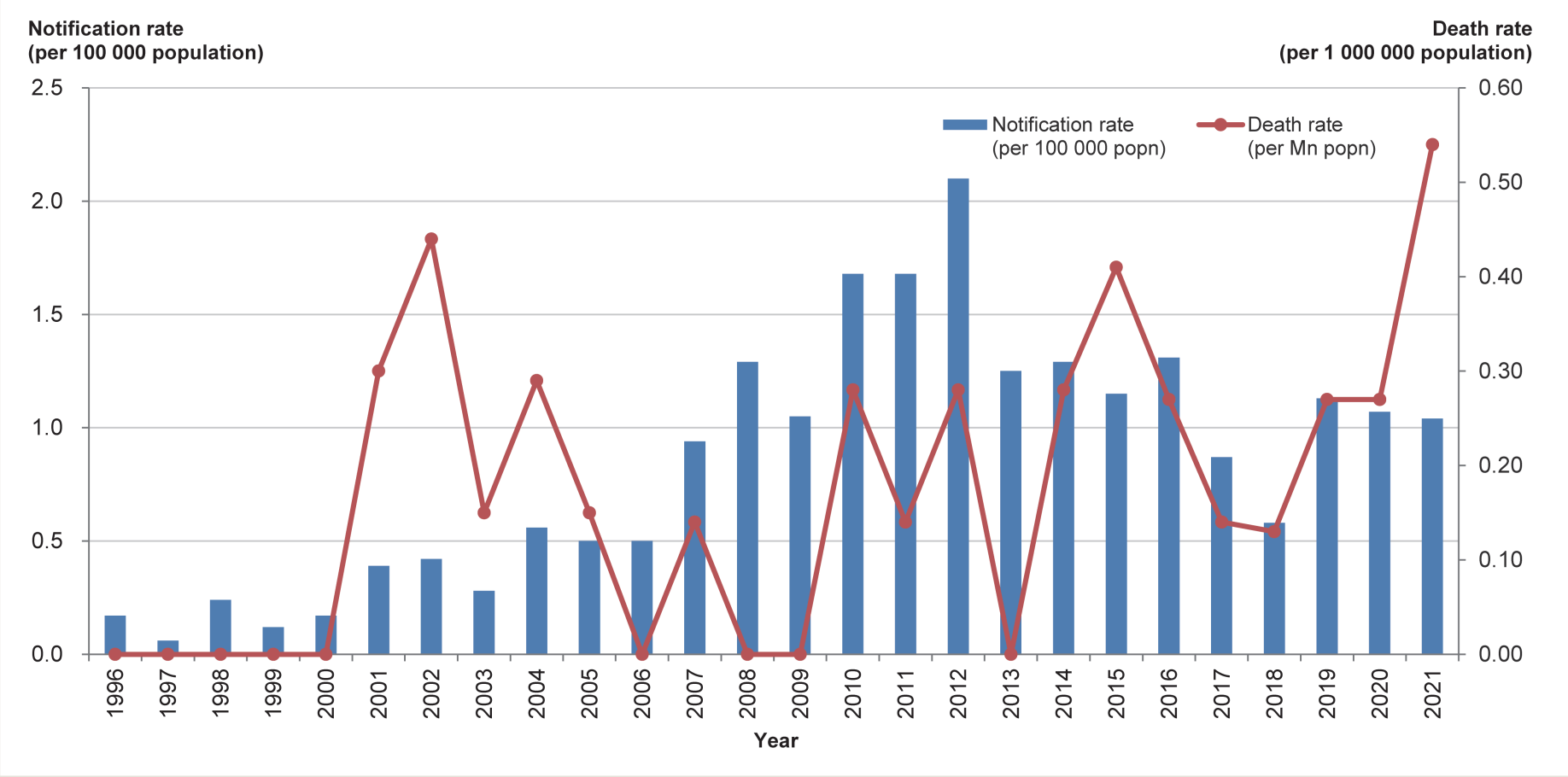
Age distribution of hepatitis E cases reported from 1996 to 2021 (Data source: CHP, DH)



	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
> 84	0%	0%	0%	0%	0%	4%	0%	0%	3%	0%	0%	0%	0%	1%	0%	0%	1%	0%	2%	2%	2%	3%	2%	7%	6%	5%
75 - 84	9%	0%	0%	0%	0%	4%	0%	11%	18%	6%	9%	3%	9%	10%	8%	7%	12%	10%	9%	5%	6%	5%	9%	15%	18%	18%
65 - 74	0%	0%	0%	13%	9%	12%	18%	16%	13%	12%	21%	5%	13%	11%	16%	8%	11%	14%	11%	19%	23%	16%	23%	25%	20%	26%
55 - 64	0%	0%	13%	25%	27%	15%	14%	16%	13%	24%	9%	14%	12%	23%	24%	27%	24%	33%	28%	31%	24%	19%	42%	26%	24%	23%
45 - 54	9%	25%	25%	25%	9%	23%	32%	16%	24%	32%	29%	38%	32%	27%	26%	29%	25%	23%	32%	24%	19%	25%	9%	14%	15%	21%
35 - 44	36%	25%	38%	13%	9%	27%	25%	32%	18%	12%	21%	29%	16%	15%	13%	19%	19%	9%	13%	12%	13%	25%	12%	8%	11%	5%
25 - 34	36%	50%	19%	25%	45%	15%	11%	11%	11%	15%	3%	6%	12%	8%	11%	8%	7%	4%	5%	6%	10%	6%	2%	4%	5%	1%
15 - 24	9%	0%	6%	0%	0%	0%	0%	0%	0%	0%	6%	3%	4%	4%	2%	2%	0%	6%	0%	1%	3%	2%	0%	1%	1%	0%
5 - 14	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	2%	1%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
< 5	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	3%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%

Box 19

Notification rates and death rates of hepatitis E from 1996 to 2021 (Data source: CHP, DH)



Seroprevalence of hepatitis A

Box 20

Prevalence of anti-HAV in studies/testing between 1978 and 2009 (Data sources: multiple sources)

Age groups	1978	1987	1989	1993^	1995	1996		1998	2000	2001	2001	2002	2003	2004	2005	2006	2007	2008	2009
0 – 20	12.9% (0 – 10) 44.8% (11 – 20)	5.3% (0 – 10) 17.1% (11 – 20)	6.8% (0 – 10) 11.2% (11 – 20)	59.4% (M) 53.3% (F)	8.3%	- (0 – 10) 7.0% (11 – 20)	6.1%	5.4%	9.3%	4.58%	- (0 – 10) 12.5% (11 – 20)	5.3%	10.3%	14.7%	15.4%	20.0%	14.3%	16.7%	25.0%
21 – 30	75.0%	53.8%	58.8%	59.4% (M) 53.3% (F)	11.3%	-	11.8%	7.6%	17.5%	13.2%	26.8%	12.6%	13.2%	21.0%	28.2%	25.8%	19.4%	26.3%	30.3%
31 – 40	82.9%	85.1%	83.5%	59.4% (M) 53.3% (F)	49.0%	-	37.7%	40.8%	35.0%	41.3%	53.2%	46.7%	52.4%	43.8%	35.7%	50.0%	37.5%	47.4%	36.4%
>40	91.1%	94.7%	91.1% (41 – 50) 93.9% (>50)	94.5% (M) 91.0% (F)	70.5%	-	58.6%	66.7%	60.0%	71.1%	88.3% (41 – 50) 97.7% (>50)	58.1%	100.0%	50.0%	72.7%	80.0%	62.5%	71.4%	26.7%
Data source	A	B	C	D	E	F	E	E	E	E	G	E	E	E	E	E	E	E	E

^Figure is the average of age 0 – 40

Data sources:

A. *Study on left-over sera of 362 subjects, by Tsang et al of the University of Hong Kong [11]*

B. *Study on stored sera of 702 healthy subjects, by Chin et al of the University of Hong Kong [10]*

C. *Study on 1028 serum samples collected from individuals attending a health exhibition, by Lim et al of Department of Health. [99]*

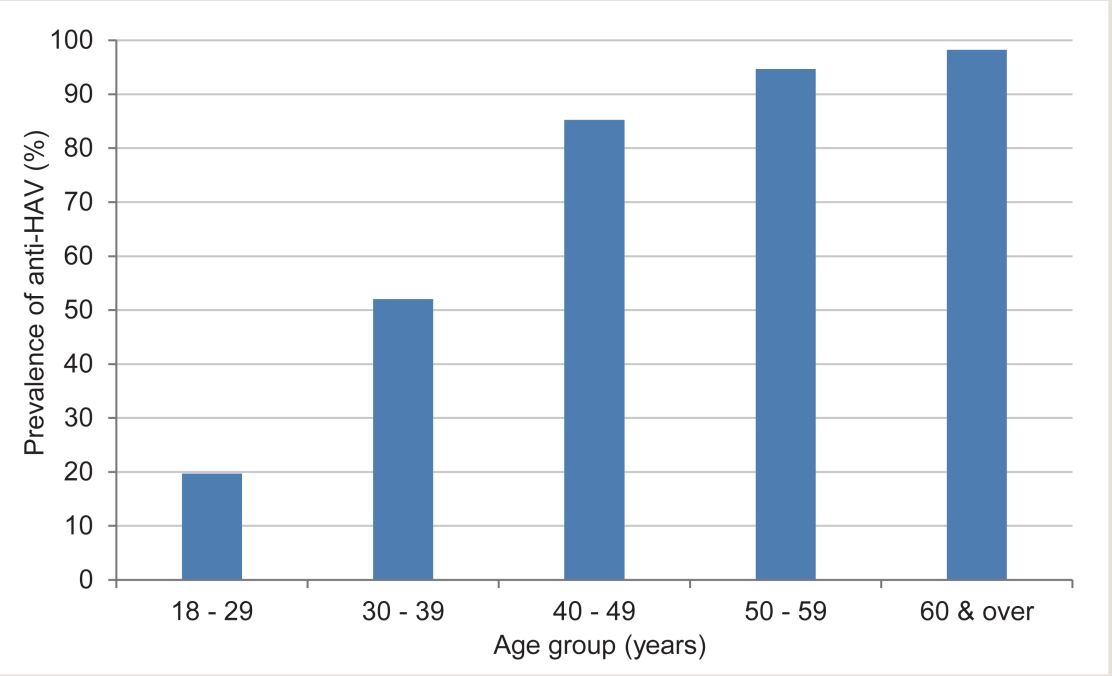
D. *Seroprevalence results reported in the press by Lai et al of the University of Hong Kong. [100]*

E. *Pre-vaccination screening on students and staff of City University of Hong Kong: 553 (1995), 669 (1996), 608 (1998), 395 (2000), 592 (2001), 371 (2002), students and staff of Baptist University of Hong Kong 240 (2001), 259 (2002), 153 (2003), 55 (2004), 77 (2005), 53 (2006), 54 (2007), 70(2008),63(2009) and students and staff of Lingnan University 125 (2003), 84 (2004). [Data from CHC-Group Medical Practice]*

F. *Seroprevalence study in school children by Lee et al of the Chinese University of Hong Kong. [101]*

G. *Community Research Project on Viral Hepatitis 2001. [2]*

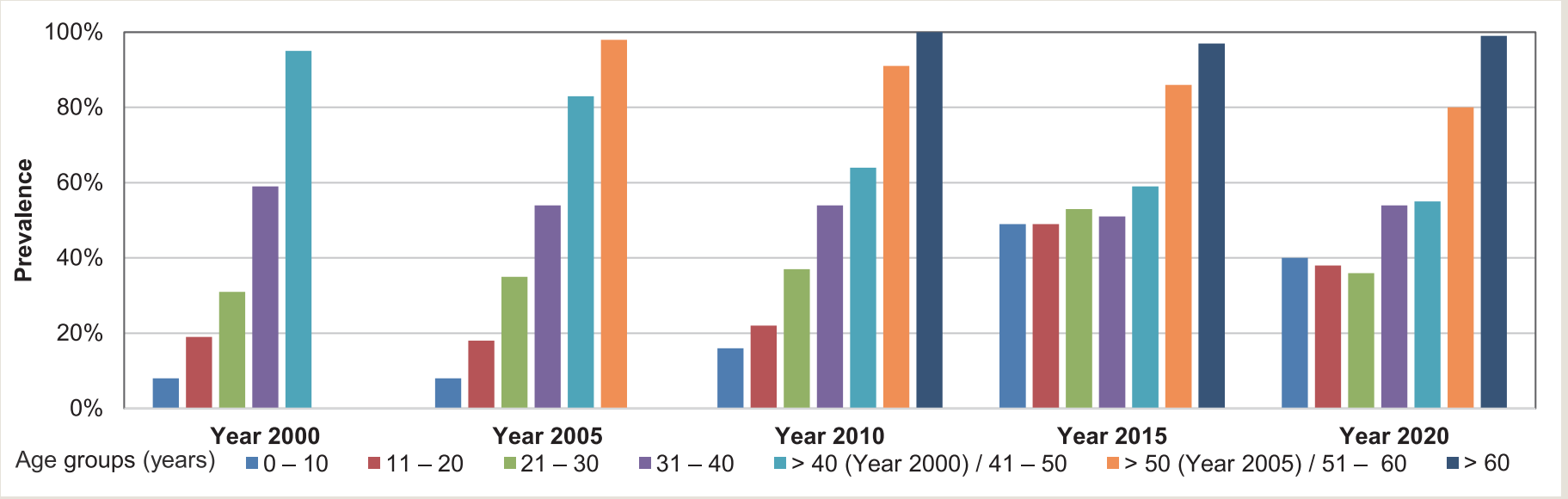
Box 21 Prevalence of anti-HAV in participants of Community Research Project for Viral Hepatitis in 2001 (Data source: DH)



Age group	No. Tested	Anti-HAV +ve (%)
18-29	137	27 (19.7%)
30-39	223	116 (52.0%)
40-49	291	248 (85.2%)
50-59	170	161 (94.7%)
60 & over	115	113 (98.3%)
All	**936**	**665 (71.0%)**

Box 22

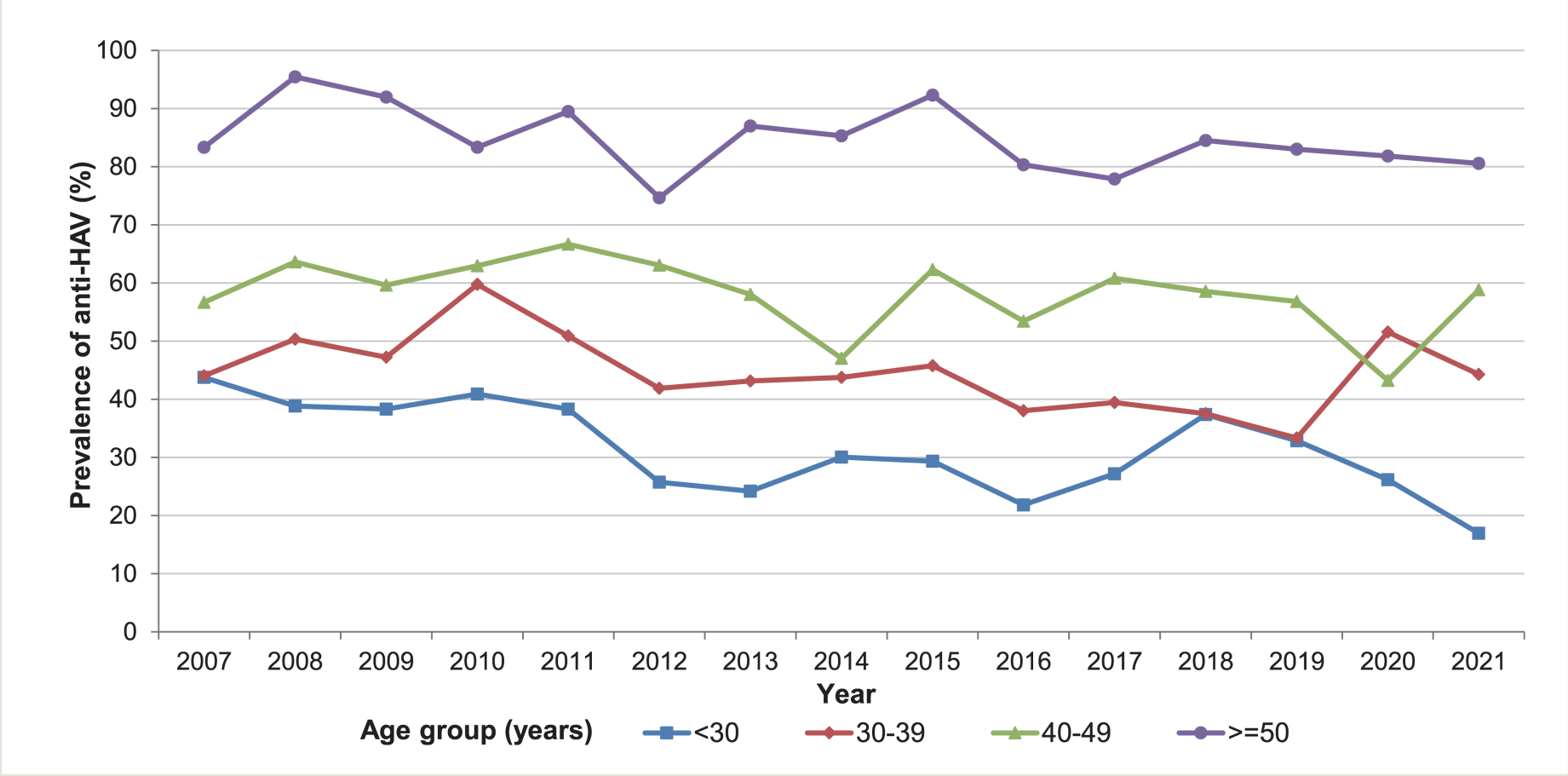
Prevalence of anti-HAV in individuals with blood collected for serological diagnosis of conditions unrelated to hepatitis (Data source: PHLSB, CHP, DH)



Year	Age groups (years)													
	0 – 10		11 – 20		21 – 30		31 – 40		> 40 (Year 2000) / 41 – 50		> 50 (Year 2005) / 51 – 60		> 60	
	No. tested	%	No. tested	%	No. tested	%	No. tested	%	No. tested	%	No. tested	%	No. tested	%
2000	420	8	190	19	200	31	190	59	100	95	-	-	-	-
2005	200	8	181	18	187	35	200	54	100	83	100	98	-	-
2010	96	16	100	22	100	37	95	54	100	64	100	91	100	100
2015	160	49	162	49	122	53	127	51	99	59	70	86	58	97
2020	89	40	99	38	97	36	99	54	101	55	100	80	100	99

Box 23

Prevalence of anti-HAV at baseline screening of HIV/AIDS patients attending ITC from Jul 2007 to 2021 (Data source: ITC, CHP, DH)

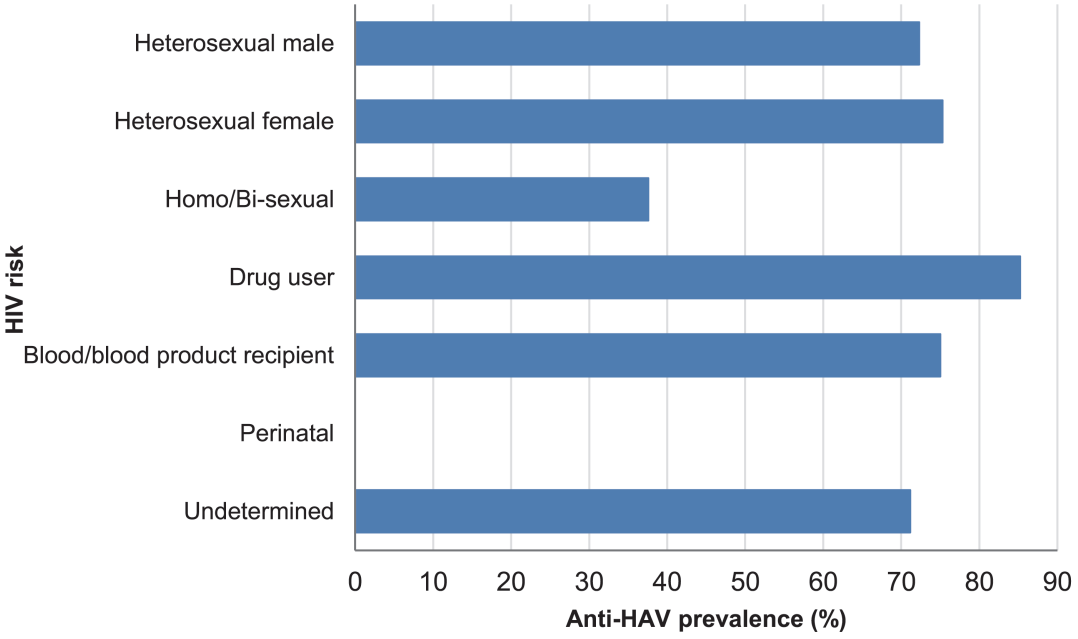


Box 23

Prevalence of anti-HAV at baseline screening of HIV/AIDS patients attending ITC from Jul 2007 to 2021 (Data source: ITC, CHP, DH) (continued)

Year	Age group									
	< 20		20 – 29		30 – 39		40 – 49		>= 50	
	No. tested	Anti-HAV +ve (%)	No. tested	Anti-HAV +ve (%)	No. tested	Anti-HAV +ve (%)	No. tested	Anti-HAV +ve (%)	No. tested	Anti-HAV +ve (%)
2007 Jul-Dec	0	0 (0.0%)	64	28 (43.8%)	202	89 (44.1%)	30	17 (56.7%)	12	10 (83.3%)
2008	2	1 (50.0%)	101	39 (38.6%)	282	142 (50.4%)	77	49 (63.6%)	44	42 (95.5%)
2009	2	0 (0.0%)	58	23 (39.7%)	91	43 (47.3%)	52	31 (59.6%)	25	23 (92.0%)
2010	3	0 (0.0%)	41	18 (43.9%)	82	49 (59.8%)	54	34 (63.0%)	42	35 (83.3%)
2011	2	0 (0.0%)	45	18 (40.0%)	57	29 (50.9%)	66	44 (66.7%)	38	34 (89.5%)
2012	6	0 (0.0%)	64	18 (28.1%)	105	44 (41.9%)	111	70 (63.1%)	75	56 (74.7%)
2013	5	2 (40.0%)	90	21 (23.3%)	102	44 (43.1%)	112	65 (58.0%)	123	107 (87.0%)
2014	8	1 (12.5%)	135	42 (31.1%)	96	42 (43.8%)	68	32 (47.1%)	68	58 (85.3%)
2015	13	6 (46.2%)	113	31 (27.4%)	118	54 (45.8%)	69	43 (62.3%)	65	60 (92.3%)
2016	4	0 (0.0%)	106	24 (22.6%)	121	46 (38.0%)	58	31 (53.4%)	56	45 (80.4%)
2017	10	4 (40.0%)	115	30 (26.1%)	109	43 (39.4%)	74	45 (60.8%)	86	67 (77.9%)
2018	2	1 (50.0%)	97	36 (37.1%)	64	24 (37.5%)	41	24 (58.5%)	97	82 (84.5%)
2019	3	1 (33.3%)	67	22 (32.8%)	69	23 (33.3%)	44	25 (56.8%)	53	44 (83.0%)
2020	1	0 (0.0%)	64	17 (26.6%)	64	33 (51.6%)	37	16 (43.2%)	33	27 (81.8%)
2021	1	1 (100.0%)	58	9 (15.5%)	61	27 (44.3%)	34	20 (58.8%)	36	29 (80.6%)

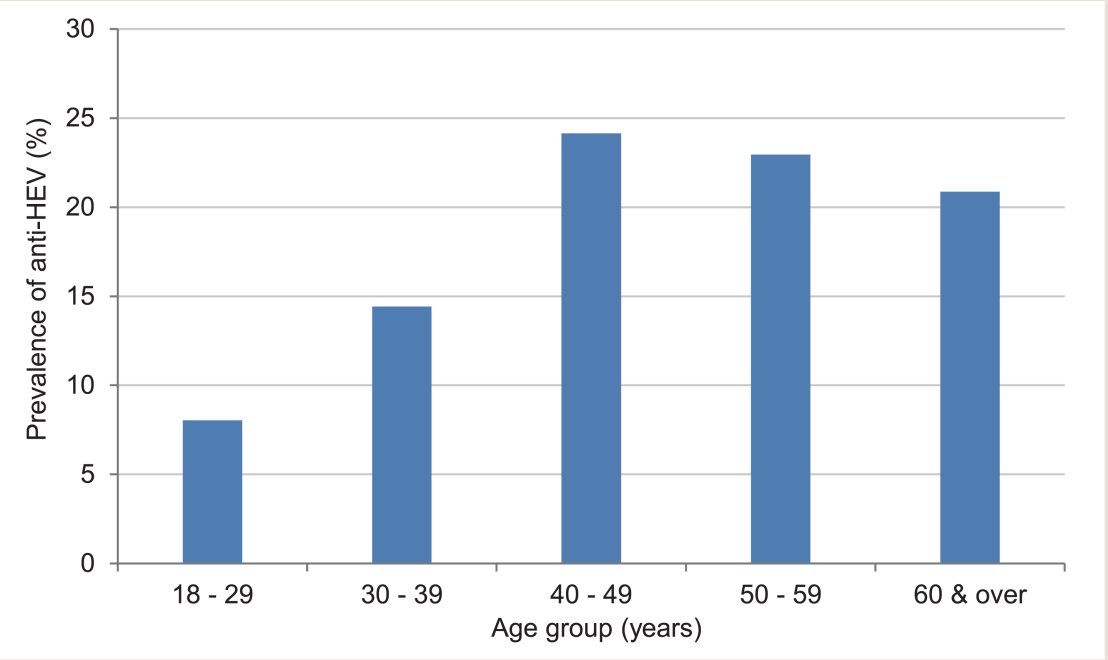
Box 24 Prevalence of anti-HAV per HIV risk at baseline screening of HIV/AIDS patients attending ITC from Jul 2007 to 2021 (Data source: ITC, CHP, DH)



HIV risk	No. tested	Anti-HAV +ve (%)
Heterosexual male	851	615 (72.3%)
Heterosexual female	554	417 (75.3%)
Homo/Bi-sexual	2987	1122 (37.6%)
Drug user	203	173 (85.2%)
Blood/blood product recipient	28	21 (75.0%)
Perinatal	9	0 (0.0%)
Undetermined	52	37 (71.2%)
Total	**4684**	**2385 (50.9%)**

Seroprevalence of hepatitis E

Box 25 Prevalence of anti-HEV in participants of Community Research Project for Viral Hepatitis in 2001 (Data source: DH)



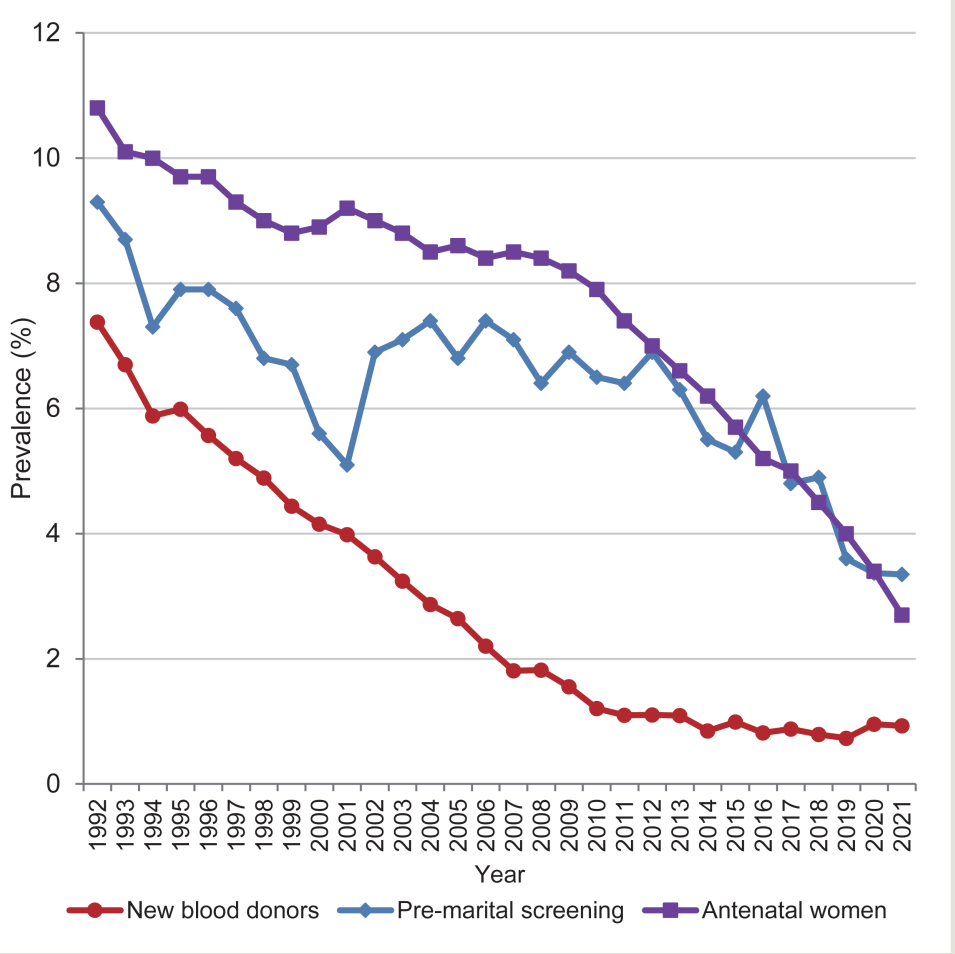
Age group	No. Tested	Anti-HEV +ve (%)
18-29	137	11 (8.0%)
30-39	222	32 (14.4%)
40-49	290	70 (24.1%)
50-59	170	39 (22.9%)
60 & over	115	24 (20.9%)
All	**934**	**176 (18.8%)**

Seroprevalence of hepatitis B

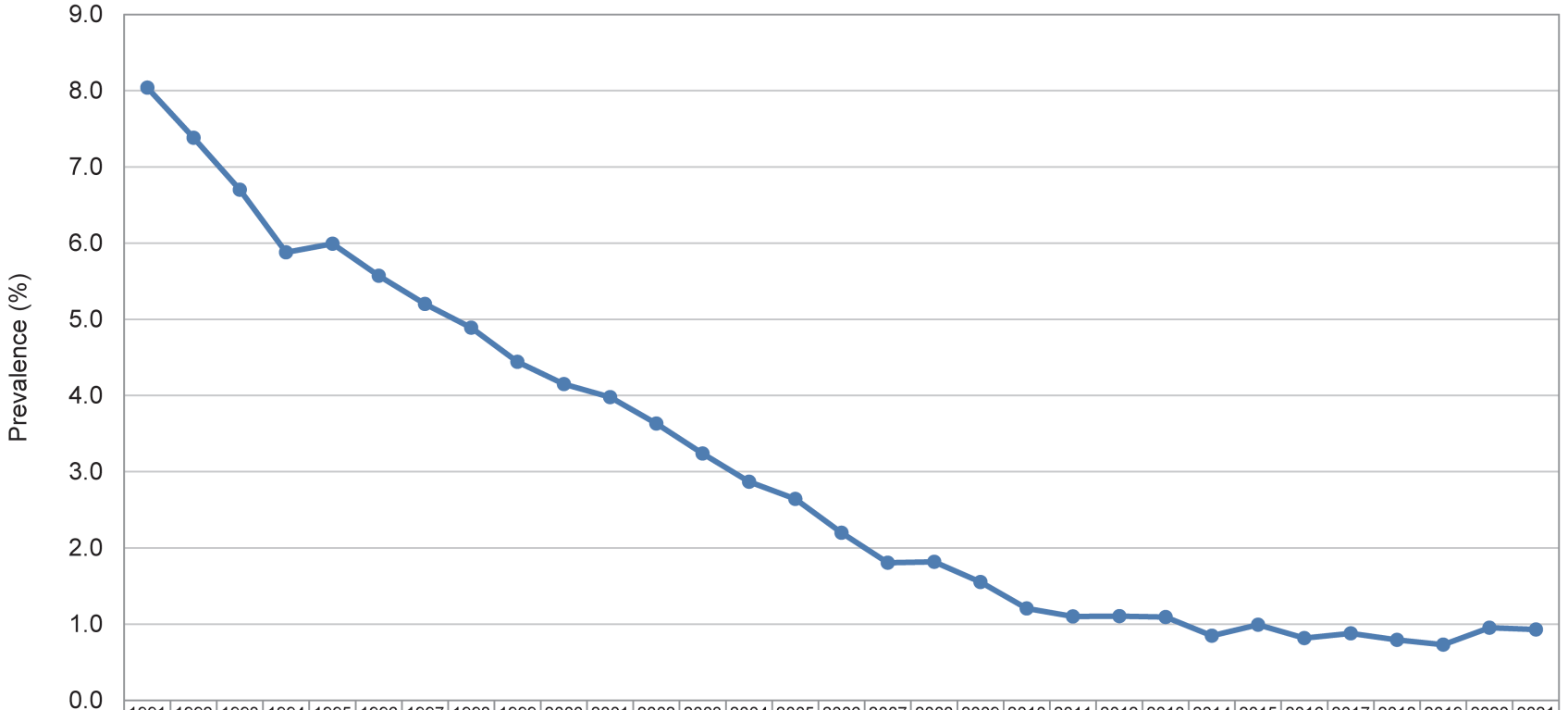
Box 26

HBsAg prevalence in new blood donors, pre-marital screening and antenatal women from 1992 to 2021 (Data source: HKRCBTS, FPAHK, FHS and PHLSB, CHP, DH)

Year	New blood donors	Pre-marital screening	Antenatal women
1992	7.4	9.3	10.8
1993	6.7	8.7	10.1
1994	5.9	7.3	10.0
1995	6.0	7.9	9.7
1996	5.6	7.9	9.7
1997	5.2	7.6	9.3
1998	4.9	6.8	9.0
1999	4.4	6.7	8.8
2000	4.2	5.6	8.9
2001	4.0	5.1	9.2
2002	3.6	6.9	9.0
2003	3.2	7.1	8.8
2004	2.9	7.4	8.5
2005	2.6	6.8	8.6
2006	2.2	7.4	8.4
2007	1.8	7.1	8.5
2008	1.8	6.4	8.4
2009	1.6	6.9	8.2
2010	1.2	6.5	7.9
2011	1.1	6.4	7.4
2012	1.1	6.9	7.0
2013	1.1	6.3	6.6
2014	0.8	5.5	6.2
2015	1.0	5.3	5.7
2016	0.8	6.2	5.2
2017	0.9	4.8	5.0
2018	0.8	4.9	4.5
2019	0.7	3.6	4.0
2020	1.0	3.4	3.4
2021	0.9	3.3	2.7



Box 27 **HBsAg prevalence in new blood donors from 1991 to 2021 (Data source: HKRCBTS)**

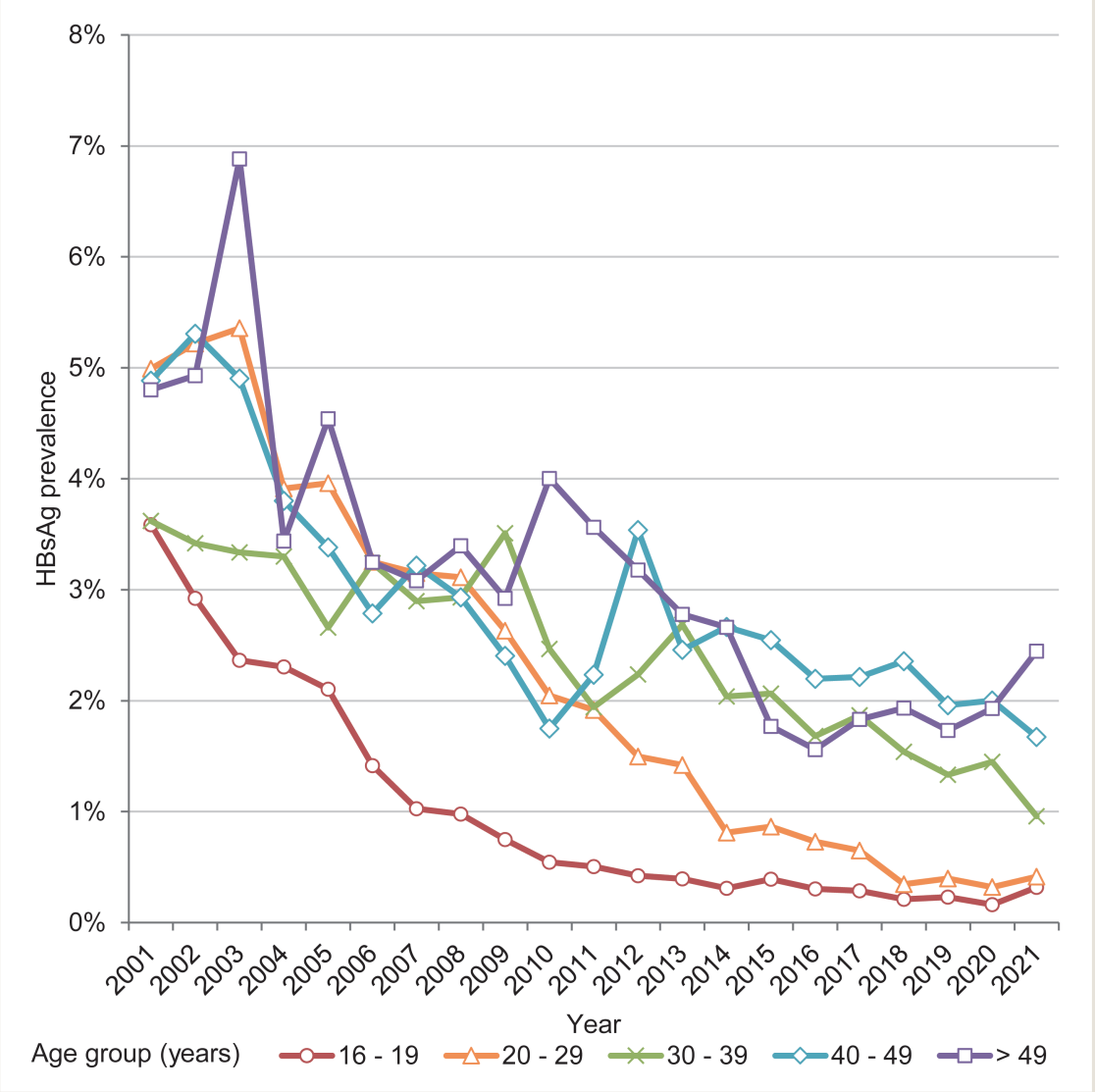


	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
% HBsAg +ve	8.0	7.4	6.7	5.9	6.0	5.6	5.2	4.9	4.4	4.2	4.0	3.6	3.2	2.9	2.6	2.2	1.8	1.8	1.6	1.2	1.1	1.1	1.1	0.8	1.0	0.8	0.9	0.8	0.7	1.0	0.9

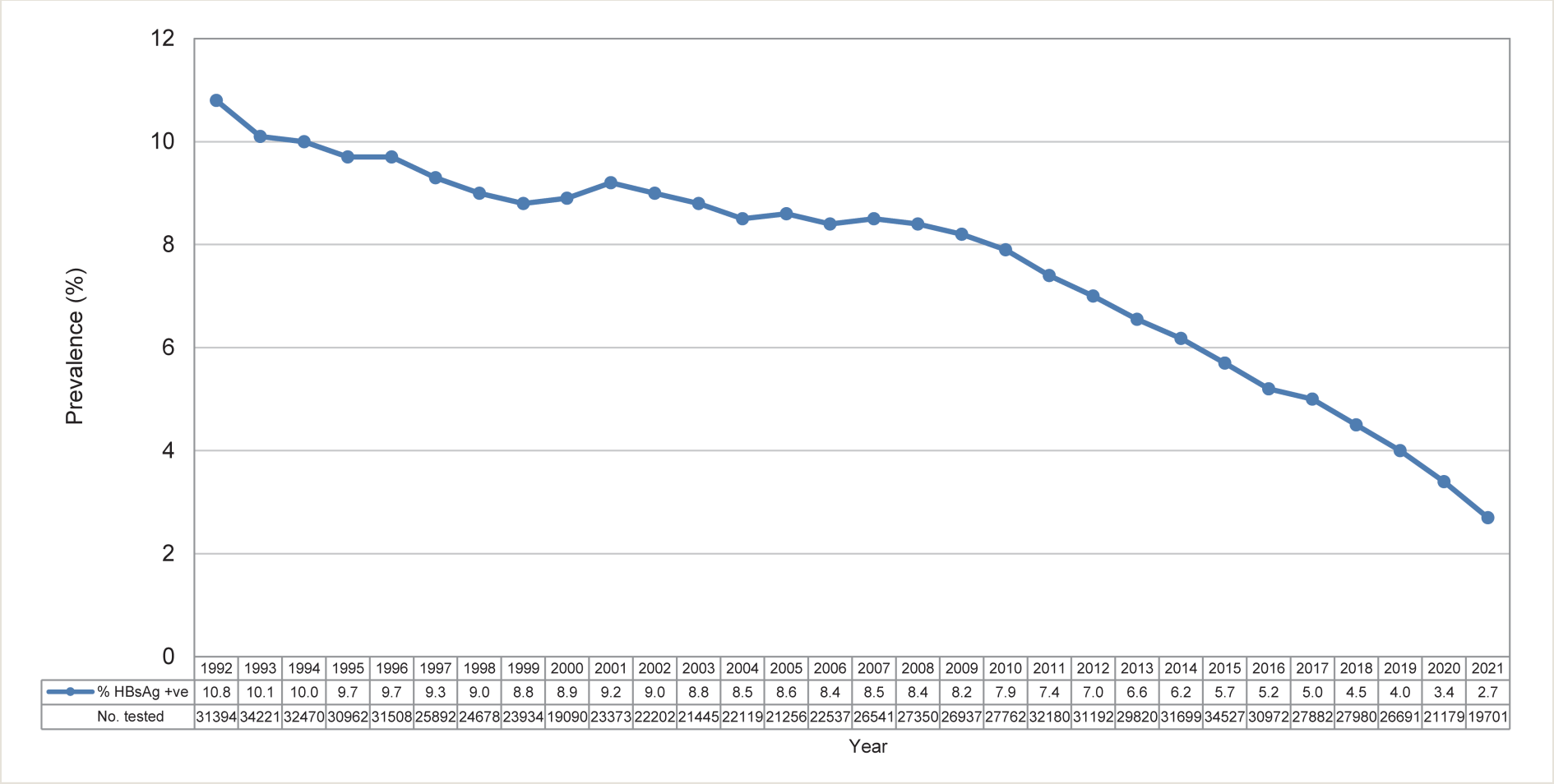
Box 28 HBsAg prevalence and its sex and age breakdown in new blood donors in 2021 (Data source: HKRCBTS)

Age group	Male		Female		Total	
	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)	**No. tested**	**HBsAg +ve (%)**
16-19	2424	8 (0.33%)	3243	10 (0.31%)	5667	18 (0.32%)
20-29	2207	15 (0.68%)	2631	5 (0.19%)	4838	20 (0.41%)
30-39	1606	23 (1.43%)	2463	16 (0.65%)	4069	39 (0.96%)
40-49	1182	29 (2.45%)	2165	27 (1.25%)	3347	56 (1.67%)
>49	745	23 (3.09%)	1463	31 (2.12%)	2208	54 (2.45%)
Total	**8164**	**98 (1.20%)**	**11965**	**89 (0.74%)**	**20129**	**187 (0.93%)**

Box 29 HBsAg prevalence among new blood donors by age, from 2001 to 2021 (Data source: HKRCBTS)



Box 30 **HBsAg prevalence in antenatal women from 1992 to 2021 (Data source: FHS and PHLSB, CHP, DH)**



	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
% HBsAg +ve	10.8	10.1	10.0	9.7	9.7	9.3	9.0	8.8	8.9	9.2	9.0	8.8	8.5	8.6	8.4	8.5	8.4	8.2	7.9	7.4	7.0	6.6	6.2	5.7	5.2	5.0	4.5	4.0	3.4	2.7
No. tested	31394	34221	32470	30962	31508	25892	24678	23934	19090	23373	22202	21445	22119	21256	22537	26541	27350	26937	27762	32180	31192	29820	31699	34527	30972	27882	27980	26691	21179	19701

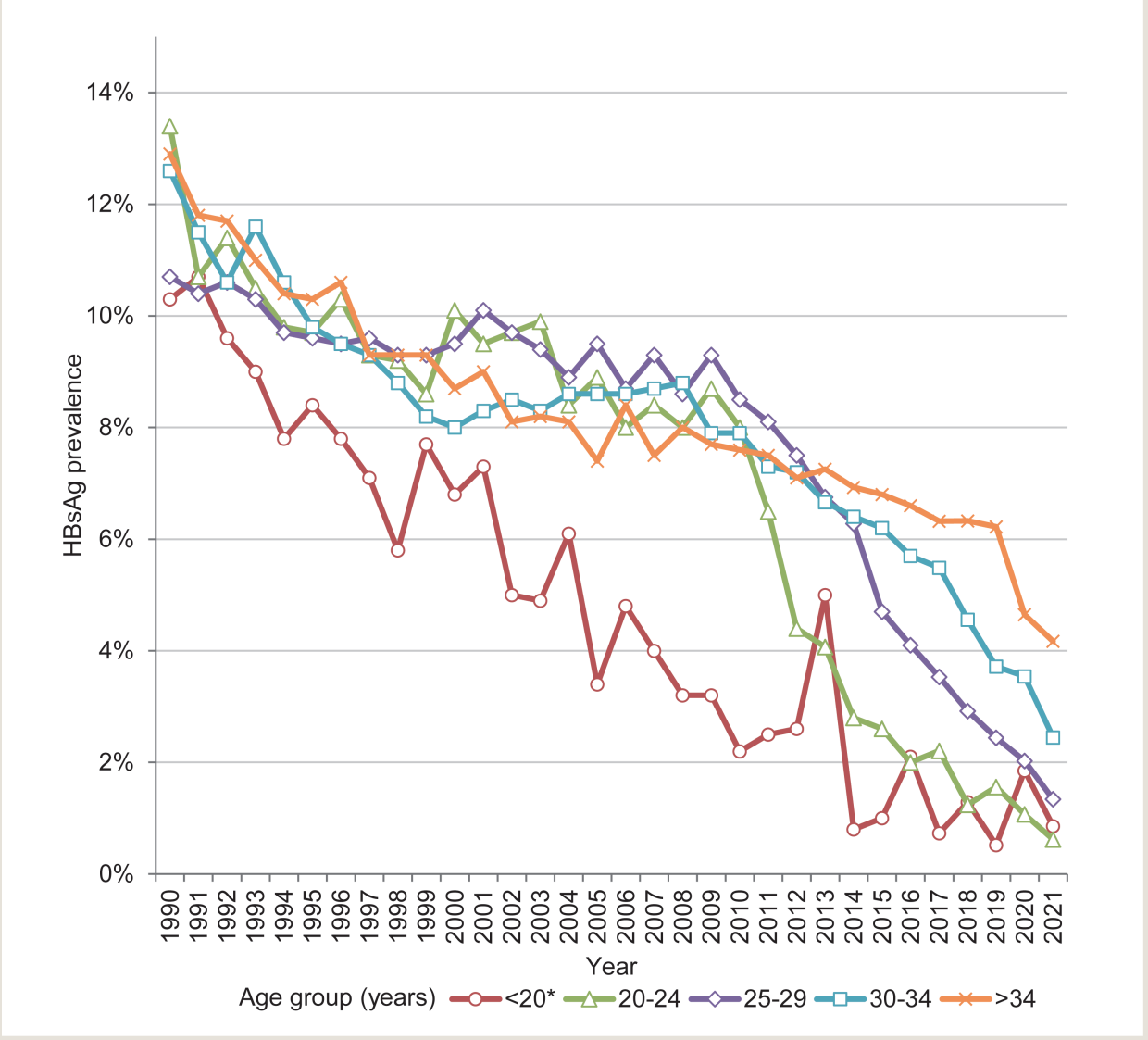
Box 31 HBsAg prevalence and age breakdown of antenatal mothers from 1990 to 2021 (Data source: FHS and PHLSB, CHP, DH)

Year	No. tested (% HBsAg +ve) according to age group of antenatal mothers				
	<20*	20-24	25-29	30-34	>34
1990	1044 (10.3%)	4671 (13.4%)	15228 (10.7%)	7639 (12.6%)	2780 (12.9%)
1991	987 (10.7%)	4620 (10.7%)	13151 (10.4%)	8168 (11.5%)	3063 (11.8%)
1992	928 (9.6%)	5065 (11.4%)	13093 (10.6%)	8788 (10.6%)	3470 (11.7%)
1993	984 (9.0%)	5589 (10.5%)	12345 (10.3%)	9395 (11.6%)	3798 (11.0%)
1994	951 (7.8%)	5723 (9.8%)	11590 (9.7%)	10158 (10.6%)	3998 (10.4%)
1995	922 (8.4%)	4979 (9.7%)	10619 (9.6%)	10112 (9.8%)	4283 (10.3%)
1996	842 (7.8%)	4765 (10.3%)	10137 (9.5%)	9759 (9.5%)	5908 (10.6%)
1997	902 (7.1%)	4207 (9.3%)	8895 (9.6%)	7982 (9.3%)	3897 (9.3%)
1998	911 (5.8%)	3887 (9.2%)	8507 (9.3%)	7418 (8.8%)	3851 (9.3%)
1999	794 (7.7%)	3777 (8.6%)	8068 (9.3%)	7196 (8.2%)	3975 (9.3%)
2000	618 (6.8%)	2974 (10.1%)	6466 (9.5%)	5818 (8.0%)	3192 (8.7%)
2001	659 (7.3%)	3516 (9.5%)	8330 (10.1%)	6936 (8.3%)	3915 (9.0%)
2002	484 (5.0%)	2829 (9.7%)	9120 (9.7%)	6351 (8.5%)	3414 (8.1%)
2003	548 (4.9%)	2880 (9.9%)	7614 (9.4%)	6789 (8.3%)	3602 (8.2%)
2004	510 (6.1%)	2854 (8.4%)	7161 (8.9%)	7732 (8.6%)	3856 (8.1%)
2005	445 (3.4%)	2753 (8.9%)	6063 (9.5%)	7869 (8.6%)	4114 (7.4%)
2006	516 (4.8%)	2590 (8.0%)	6271 (8.7%)	8637 (8.6%)	4514 (8.4%)
2007	520 (4.0%)	2929 (8.4%)	7301 (9.3%)	10232 (8.7%)	5551 (7.5%)
2008	533 (3.2%)	2968 (8.0%)	7652 (8.6%)	10354 (8.8%)	5838 (8.0%)
2009	434 (3.2%)	2830 (8.7%)	7444 (9.3%)	10156 (7.9%)	6071 (7.7%)
2010	446 (2.2%)	2903 (8.0%)	7817 (8.5%)	10211 (7.9%)	6385 (7.6%)
2011	447 (2.5%)	2898 (6.5%)	9010 (8.1%)	12273 (7.3%)	7552 (7.5%)
2012	463 (2.6%)	2467 (4.4%)	8161 (7.5%)	12664 (7.2%)	7437 (7.1%)
2013	423 (5.0%)	2237 (4.1%)	7526 (6.8%)	12466 (6.7%)	7168 (7.3%)
2014	366 (0.8%)	2252 (2.8%)	7901 (6.3%)	13488 (6.4%)	7692 (6.9%)
2015	409 (1.0%)	2439 (2.6%)	8589 (4.7%)	14434 (6.2%)	8656 (6.8%)
2016	328 (2.1%)	2123 (2.0%)	7580 (4.1%)	13018 (5.7%)	7923 (6.6%)
2017	274 (0.7%)	1897 (2.2%)	6624 (3.5%)	11476 (5.5%)	7611 (6.3%)
2018	233 (1.3%)	1698 (1.2%)	6376 (2.9%)	11647 (4.6%)	8026 (6.3%)
2019	193 (0.5%)	1474 (1.6%)	5948 (2.4%)	11333 (3.7%)	7743 (6.2%)
2020	162 (1.9%)	1031 (1.1%)	4394 (2.0%)	9291 (3.5%)	6301 (4.7%)
2021	116 (0.9%)	811 (0.6%)	3960 (1.3%)	8586 (2.4%)	6228 (4.2%)

** Figures before year 2010 refer to age group 15-19; figures in year 2010 and thereafter refer to age group <20*

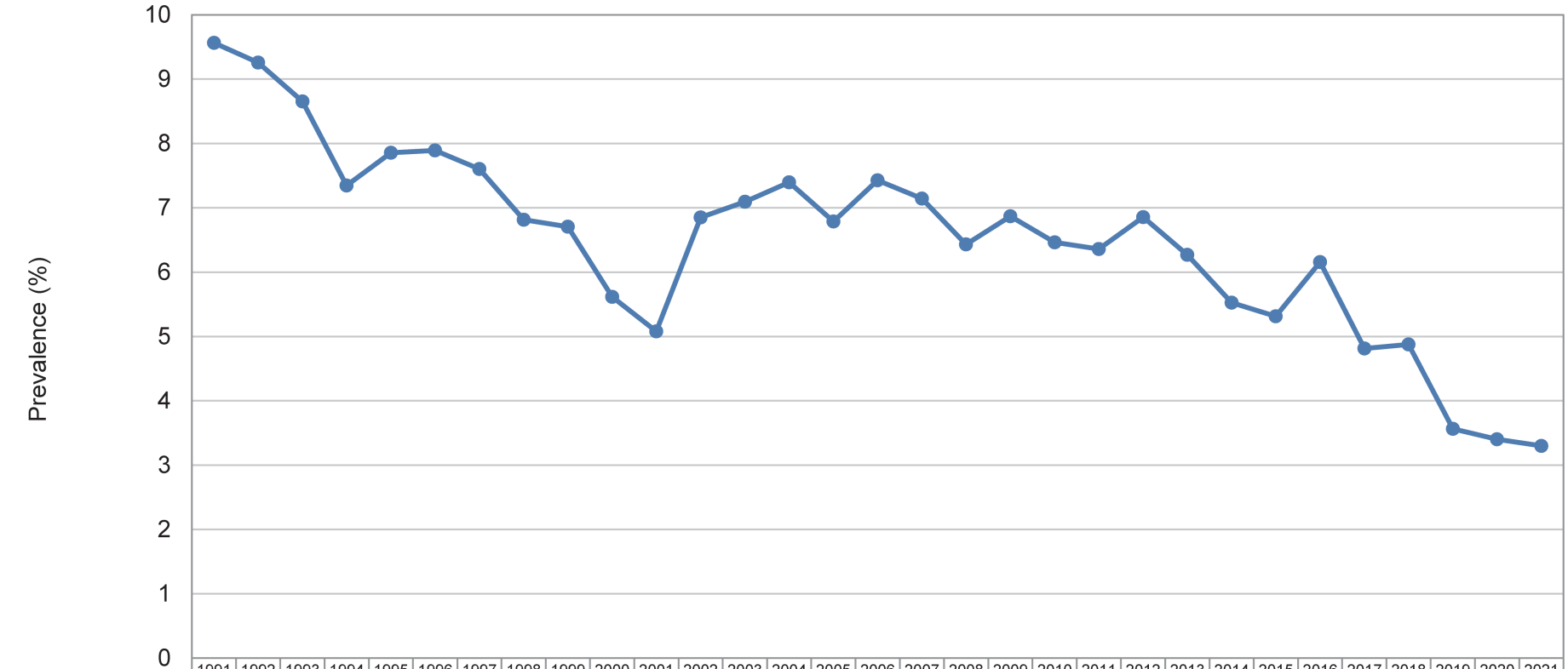
Box 32

HBsAg prevalence among antenatal mothers by age, from 1990 to 2021 (Date source: FHS and PHLSB, CHP, DH)



** Figures before year 2010 refer to age group 15-19; figures in year 2010 and thereafter refer to age group <20*

Box 33 HBsAg prevalence from the FPAHK's clinical services (Data source: FPAHK)



	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
% HBsAg +ve	9.6	9.3	8.7	7.3	7.9	7.9	7.6	6.8	6.7	5.6	5.1	6.9	7.1	7.4	6.8	7.4	7.1	6.4	6.9	6.5	6.4	6.9	6.3	5.5	5.3	6.2	4.8	4.9	3.6	3.4	3.3
Total no. of cases	19142	18445	19193	16466	16798	19959	17109	13163	12686	15348	16611	15077	13489	13773	11772	11831	9787	10669	9553	14137	13163	12191	13850	13117	11325	11091	10101	10315	8776	6027	6930

Note: 1991-2010 only contain pre-marital check-up
Start from 2011 contain both pre-marital and pre-pregnancy check-up

Box 34 HBsAg prevalence in other selected populations from 1990 to 2021 (Data sources: DH)

Year	Police officers	Health care workers
1990	-	-
1991	-	6.2
1992	-	-
1993	-	4.4
1994	-	-
1995	-	7.0
1996	6.1	4.2
1997	7.9	-
1998	7.4	-
1999	6.4	2.2
2000	5.6	5.4
2001	5.9	6.0
2002	5.3	5.0
2003	4.6	5.2
2004	4.9	5.3
2005	4.2	5.4
2006	4.6	4.9
2007	-	3.9
2008	-	3.8
2009	-	5.1
2010	-	4.6
2011	-	2.5
2012	3.0*	4.3
2013	2.8	3.9
2014	2.6	2.5
2015	2.8	3.2
2016	1.9	3.5
2017	1.4	3.1
2018	2.3	3.5
2019	1.2	2.7
2020	2.2	2.2
2021	1.8	2.2

** For a period between Mar-Dec 2012*

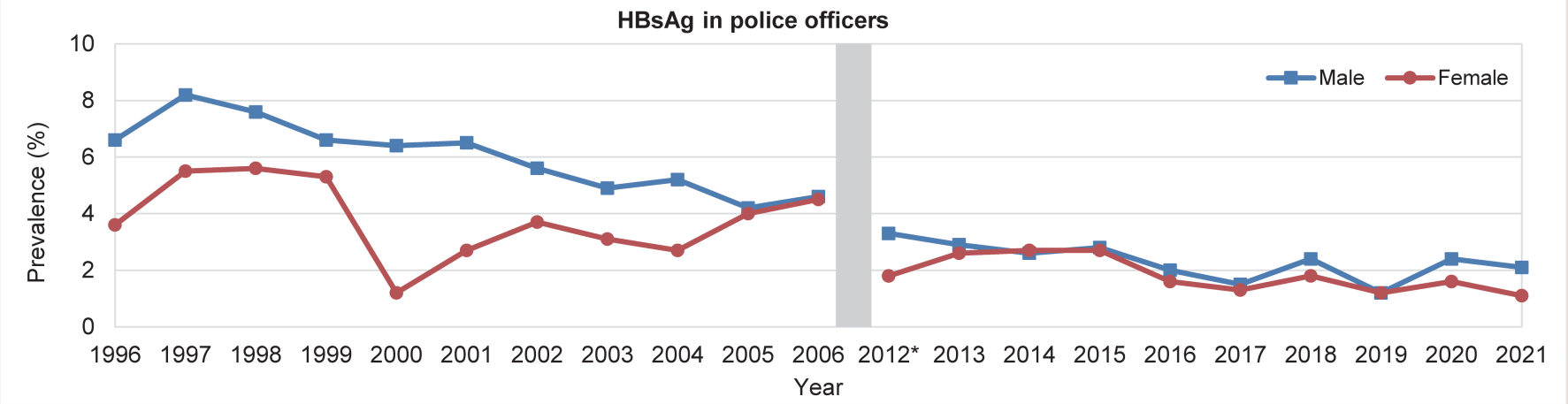
Box 35 Prevalence of hepatitis B markers in police officers, by age from 1996 to 2006 and 2012 to 2021 (Data source: DH)

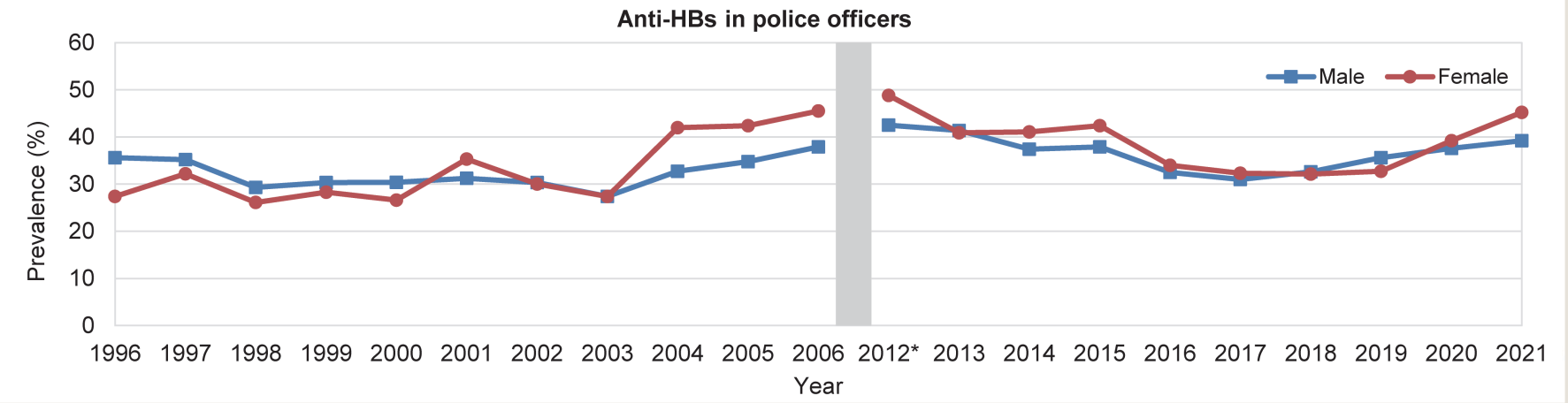
Year	Age group														
	≤20			21-30			31-40			41-50			>50		
	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)
1996	17	0.0	35.3	733	4.8	24.4	1155	6.8	32.9	544	5.9	49.6	44	18.2	40.9
1997	15	6.7	46.7	1494	6.1	25.4	2081	7.3	35.0	999	11.4	46.6	110	13.6	55.5
1998	387	5.9	20.7	969	5.5	25.0	828	8.3	30.8	356	12.4	40.4	60	6.7	51.7
1999	270	4.4	24.1	799	6.1	27.5	428	6.8	31.8	202	8.9	42.1	22	9.1	40.9
2000	72	4.2	22.2	746	6.4	24.3	460	4.3	31.3	242	5.8	44.6	24	4.2	45.8
2001	68	4.4	30.9	602	5.8	28.4	339	5.6	30.7	225	6.2	40.0	45	8.9	48.9
2002	145	4.8	29.7	697	4.9	25.3	443	3.6	29.6	307	9.1	37.5	52	3.8	61.5
2003	72	1.4	16.7	702	4.8	22.9	505	4.6	26.5	357	5.0	38.1	38	2.6	42.1
2004	8	0.0	37.5	466	5.2	35.6	441	3.4	28.6	321	5.9	39.6	57	8.8	31.6
2005	80	1.3	52.5	791	3.8	32.7	533	4.3	31.0	427	4.2	43.3	105	8.6	45.7
2006	0	-	-	39	0.0	51.3	86	5.8	36.0	90	4.4	36.7	24	8.3	41.7
2012*	267	0.7	20.2	1169	2.1	47.3	122	6.6	53.3	203	5.9	47.8	71	11.3	43.7
2013	393	0.0	24.4	1635	2.7	43.8	95	4.2	57.9	133	11.3	46.6	62	3.2	46.8
2014	456	0.7	24.8	1789	1.9	37.8	188	6.4	48.9	280	6.4	51.1	114	6.1	46.5
2015	455	0.9	24.8	2077	2.4	38.9	221	5.4	50.7	309	5.5	46.9	122	4.1	47.5
2016	428	0.5	17.3	2250	1.6	33.2	154	5.2	53.2	125	7.2	49.6	54	3.7	42.6
2017	391	0.5	21.2	2594	1.3	31.7	182	2.2	46.7	13	38.5	30.8	3	0.0	66.7
2018	332	2.1	27.7	1908	1.9	31.1	176	6.3	53.4	7	0.0	85.7	1	0.0	100.0
2019	274	0.7	33.2	1475	0.8	32.5	217	4.6	49.8	32	0.0	59.4	3	0.0	100.0
2020	149	0.0	34.2	1021	1.7	32.5	360	4.2	52.5	80	3.8	48.8	7	14.3	57.1
2021	157	0.0	31.8	973	1.0	34.6	492	3.0	53.5	136	5.1	61.0	15	6.7	60.0

Note: Data were not available from 2007-Feb 2012
** For a period between Mar-Dec 2012*

Box 36

Prevalence of hepatitis B markers in police officers, by sex from 1996 to 2006 and 2012 to 2021 (Data source: DH)





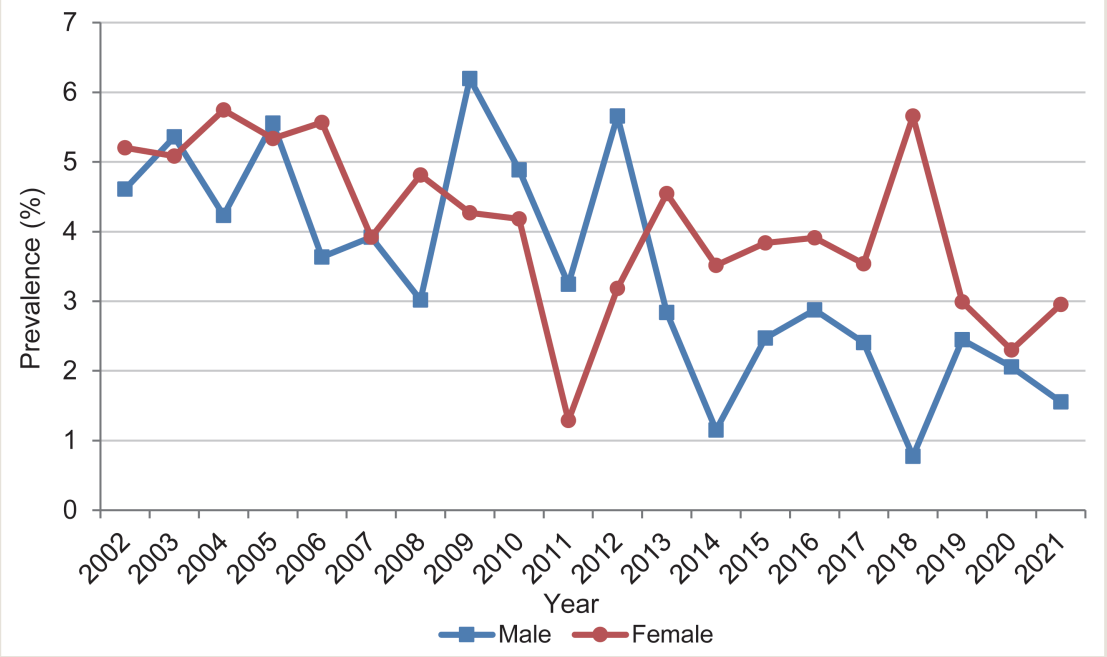
Note: Data were not available from 2007-Feb 2012
** For a period between Mar-Dec 2012*

Box 36 **Prevalence of hepatitis B markers in police officers, by sex from 1996 to 2006 and 2012 to 2021 (Data source: DH) (continued)**

Year	Male			Female			All		
	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	**No. tested**	**HBsAg +ve (%)**	**Anti-HBs +ve (%)**
1996	2080	138 (6.6%)	740 (35.6%)	413	15 (3.6%)	113 (27.4%)	**2493**	**153 (6.1%)**	**853 (34.2%)**
1997	4227	346 (8.2%)	1489 (35.2%)	472	26 (5.5%)	152 (32.2%)	**4699**	**372 (7.9%)**	**1641 (34.9%)**
1998	2316	177 (7.6%)	678 (29.3%)	284	16 (5.6%)	74 (26.1%)	**2600**	**193 (7.4%)**	**752 (28.9%)**
1999	1399	93 (6.6%)	424 (30.3%)	322	17 (5.3%)	91 (28.3%)	**1721**	**110 (6.4%)**	**515 (29.9%)**
2000	1300	83 (6.4%)	395 (30.4%)	244	3 (1.2%)	65 (26.6%)	**1544**	**86 (5.6%)**	**460 (29.8%)**
2001	1058	69 (6.5%)	330 (31.2%)	221	6 (2.7%)	78 (35.3%)	**1279**	**75 (5.9%)**	**408 (31.9%)**
2002	1374	77 (5.6%)	416 (30.3%)	270	10 (3.7%)	81 (30.0%)	**1644**	**87 (5.3%)**	**497 (30.2%)**
2003	1415	69 (4.9%)	388 (27.4%)	259	8 (3.1%)	71 (27.4%)	**1674**	**77 (4.6%)**	**459 (27.4%)**
2004	1105	58 (5.2%)	361 (32.7%)	188	5 (2.7%)	79 (42.0%)	**1293**	**63 (4.9%)**	**440 (34.0%)**
2005	1613	68 (4.2%)	562 (34.8%)	323	13 (4.0%)	137 (42.4%)	**1936**	**81 (4.2%)**	**699 (36.1%)**
2006	195	9 (4.6%)	74 (37.9%)	44	2 (4.5%)	20 (45.5%)	**239**	**11 (4.6%)**	**94 (39.3%)**
2012*	1494	49 (3.3%)	635 (42.5%)	338	6 (1.8%)	165 (48.8%)	**1832**	**55 (3.0%)**	**800 (43.7%)**
2013	1812	52 (2.9%)	751 (41.4%)	506	13 (2.6%)	207 (40.9%)	**2318**	**65 (2.8%)**	**958 (41.3%)**
2014	2267	59 (2.6%)	847 (37.4%)	560	15 (2.7%)	230 (41.1%)	**2827**	**74 (2.6%)**	**1077 (38.1%)**
2015	2563	71 (2.8%)	972 (37.9 %)	621	17 (2.7%)	263 (42.4%)	**3184**	**88 (2.8%)**	**1235 (38.8%)**
2016	2450	49 (2.0%)	796 (32.5%)	561	9 (1.6%)	191 (34.0%)	**3011**	**58 (1.9%)**	**987 (32.8%)**
2017	2477	36 (1.5%)	768 (31.0%)	706	9 (1.3%)	228 (32.3%)	**3183**	**45 (1.4%)**	**996 (31.3%)**
2018	1913	46 (2.4%)	623 (32.6%)	511	9 (1.8%)	164 (32.1%)	**2424**	**55 (2.3%)**	**787 (32.5%)**
2019	1582	19 (1.2%)	563 (35.6%)	419	5 (1.2%)	137 (32.7%)	**2001**	**24 (1.2%)**	**700 (35.0%)**
2020	1191	29 (2.4%)	448 (37.6%)	426	7 (1.6%)	167 (39.2%)	**1617**	**36 (2.2%)**	**615 (38.0%)**
2021	1291	27 (2.1%)	506 (39.2%)	522	6 (1.1%)	236 (45.2%)	**1813**	**33 (1.8%)**	**742 (40.9%)**

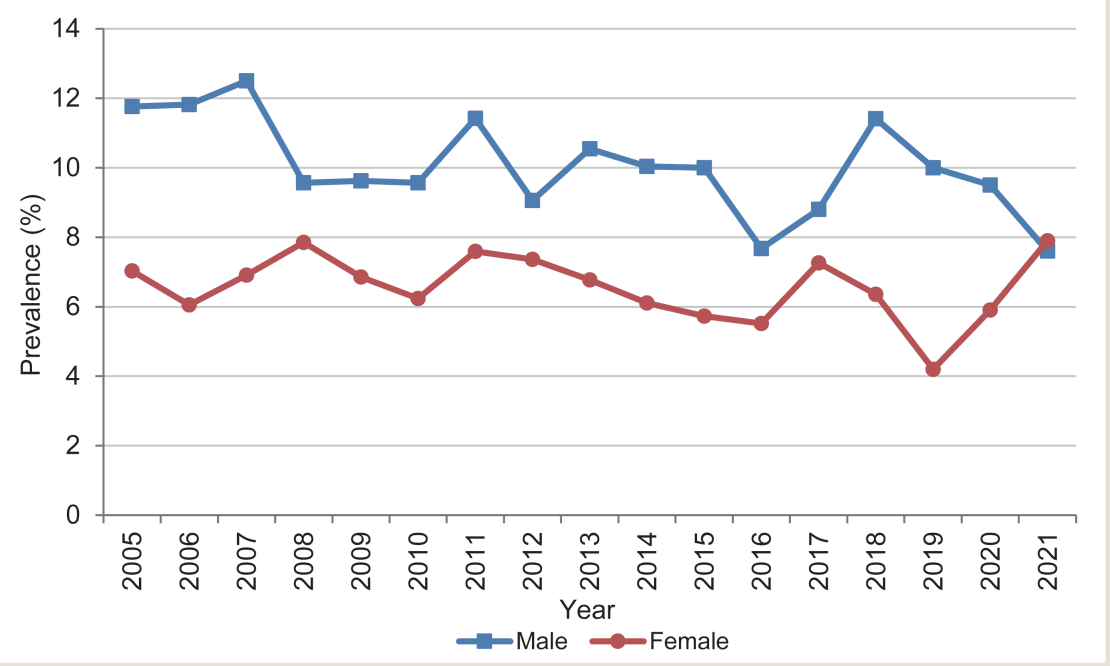
Note: Data were not available from 2007-Feb 2012;
** For a period between Mar-Dec 2012*

Box 37 HBsAg prevalence in newly recruited health care workers of DH from 2002 to 2021 (Data source: DH)



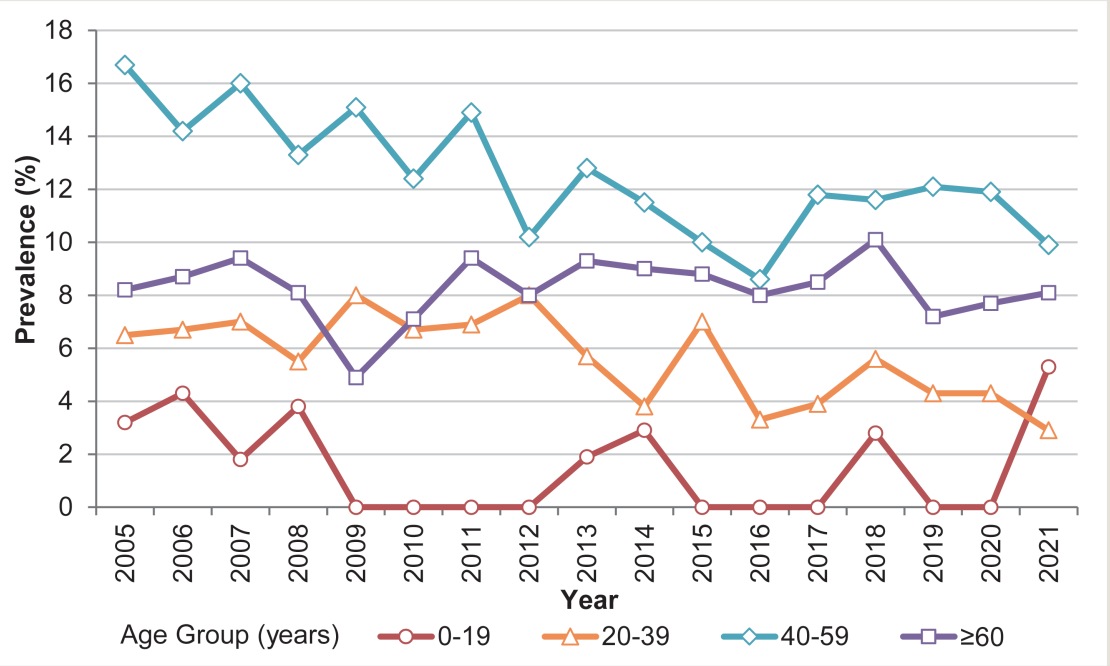
Year	Male		Female	
	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)
2002	499	23 (4.6%)	730	38 (5.2%)
2003	373	20 (5.4%)	531	27 (5.1%)
2004	307	13 (4.2%)	644	37 (5.7%)
2005	396	22 (5.6%)	956	51 (5.3%)
2006	220	8 (3.6%)	449	25 (5.6%)
2007	204	8 (3.9%)	102	4 (3.9%)
2008	232	7 (3.0%)	187	9 (4.8%)
2009	226	14 (6.2%)	328	14 (4.3%)
2010	307	15 (4.9%)	239	10 (4.2%)
2011	370	12 (3.2%)	233	3 (1.3%)
2012	318	18 (5.7%)	377	12 (3.2%)
2013	282	8 (2.8%)	418	19 (4.5%)
2014	261	3 (1.1%)	370	13 (3.5%)
2015	324	8 (2.5%)	391	15 (3.8%)
2016	278	8 (2.9%)	409	16 (3.9%)
2017	291	7 (2.4%)	452	16 (3.5%)
2018	258	2 (0.8%)	318	18 (5.7%)
2019	245	6 (2.4%)	234	7 (3.0%)
2020	243	5 (2.1%)	391	9 (2.3%)
2021	450	7 (1.6%)	440	13 (3.0%)

Box 38 HBsAg prevalence in tuberculosis patients treated at chest clinics, by sex from 2005 to 2021 (March to May) (Data source: Tuberculosis and Chest Service, CHP, DH)



Year	Male		Female		Total	
	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)	**No. tested**	**HBsAg +ve (%)**
2005	442	52 (11.8%)	242	17 (7.0%)	**684**	**69 (10.1%)**
2006	821	97 (11.8%)	446	27 (6.1%)	**1267**	**124 (9.8%)**
2007	768	96 (12.5%)	420	29 (6.9%)	**1188**	**125 (10.5%)**
2008	648	62 (9.6%)	382	30 (7.9%)	**1030**	**92 (8.9%)**
2009	759	73 (9.6%)	438	30 (6.8%)	**1197**	**103 (8.6%)**
2010	669	64 (9.6%)	353	22 (6.2%)	**1022**	**86 (8.4%)**
2011	674	77 (11.4%)	382	29 (7.6%)	**1056**	**106 (10.0%)**
2012	651	59 (9.1%)	367	27 (7.4%)	**1018**	**86 (8.4%)**
2013	664	70 (10.5%)	369	25 (6.8%)	**1033**	**95 (9.2%)**
2014	598	60 (10.0%)	393	24 (6.1%)	**991**	**84 (8.5%)**
2015	560	56 (10.0%)	314	18 (5.7%)	**874**	**74 (8.5%)**
2016	534	41 (7.7%)	308	17 (5.5%)	**842**	**58 (6.9%)**
2017	500	44 (8.8%)	303	22 (7.3%)	**803**	**66 (8.2%)**
2018	666	76 (11.4%)	425	27 (6.4%)	**1091**	**103 (9.4%)**
2019	571	57 (10.0%)	312	13 (4.2%)	**883**	**70 (7.9%)**
2020	423	40 (9.5%)	288	17 (5.9%)	**711**	**57 (8.0%)**
2021	511	39 (7.6%)	316	25 (7.9%)	**827**	**64 (7.7%)**

Box 39 HBsAg prevalence in tuberculosis patients treated at chest clinics, by age from 2005 to 2021 (March to May) (Data source: Tuberculosis and Chest Service, CHP, DH)



Year	Age group							
	0-19		20-39		40-59		≥60	
	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)
2005	31	1 (3.2%)	168	11 (6.5%)	204	34 (16.7%)	281	23 (8.2%)
2006	47	2 (4.3%)	314	21 (6.7%)	402	57 (14.2%)	504	44 (8.7%)
2007	57	1 (1.8%)	287	20 (7.0%)	374	60 (16.0%)	470	44 (9.4%)
2008	26	1 (3.8%)	256	14 (5.5%)	316	42 (13.3%)	432	35 (8.1%)
2009	45	0 (0.0%)	275	22 (8.0%)	370	56 (15.1%)	507	25 (4.9%)
2010	34	0 (0.0%)	224	15 (6.7%)	315	39 (12.4%)	449	32 (7.1%)
2011	35	0 (0.0%)	259	18 (6.9%)	303	45 (14.9%)	459	43 (9.4%)
2012	32	0 (0.0%)	261	21 (8.0%)	315	32 (10.2%)	410	33 (8.0%)
2013	54	1 (1.9%)	228	13 (5.7%)	320	41 (12.8%)	431	40 (9.3%)
2014	34	1 (2.9%)	211	8 (3.8%)	313	36 (11.5%)	433	39 (9.0%)
2015	30	0 (0.0%)	187	13 (7.0%)	260	26 (10.0%)	397	35 (8.8%)
2016	25	0 (0.0%)	180	6 (3.3%)	222	19 (8.6%)	415	33 (8.0%)
2017	35	0 (0.0%)	153	6 (3.9%)	237	28 (11.8%)	378	32 (8.5%)
2018	36	1 (2.8%)	197	11 (5.6%)	311	36 (11.6%)	547	55 (10.1%)
2019	11	0 (0.0%)	163	7 (4.3%)	248	30 (12.1%)	461	33 (7.2%)
2020	22	0 (0.0%)	140	6 (4.3%)	210	25 (11.9%)	339	26 (7.7%)
2021	19	1 (5.3%)	138	4 (2.9%)	252	25 (9.9%)	418	34 (8.1%)

Box 40

Prevalence of hepatitis B markers in persons attending Therapeutic Prevention Clinic of ITC for post-exposure management, from 2000 to 2021 (Data source: ITC, CHP, DH)

Year	Health care workers			Non- Health care workers			**Total**		
	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)	**No. tested**	**HBsAg +ve (%)**	**Anti-HBs +ve (%)**
2000	77	5 (6.5%)	56 (72.7%)	217	20 (9.2%)	91 (41.9%)	294	25 (8.5%)	147 (50.0%)
2001	103	2 (1.9%)	78 (75.7%)	313	20 (6.4%)	143 (45.7%)	416	22 (5.3%)	221 (53.1%)
2002	99	9 (9.1%)	62 (62.6%)	252	22 (8.7%)	133 (52.8%)	351	31 (8.8%)	195 (55.6%)
2003	96	6 (6.3%)	66 (68.8%)	201	24 (11.9%)	81 (40.3%)	297	30 (10.1%)	147 (49.5%)
2004	66	4 (6.1%)	41 (62.1%)	182	15 (8.2%)	97 (53.3%)	248	19 (7.7%)	138 (55.6%)
2005	49	3 (6.1%)	31 (63.3%)	206	13 (6.3%)	99 (48.1%)	255	16 (6.3%)	130 (51.0%)
2006	54	6 (11.1%)	33 (61.1%)	289	15 (5.2%)	151 (52.2%)	343	21 (6.1%)	184 (53.6%)
2007	54	1 (1.9%)	45 (83.3%)	228	18 (7.9%)	88 (38.6%)	282	19 (6.7%)	133 (47.2%)
2008	54	2 (3.7%)	39 (72.2%)	235	20 (8.5%)	111 (47.2%)	289	22 (7.6%)	150 (51.9%)
2009	56	1 (1.8%)	41 (73.2%)	297	22 (7.4%)	138 (46.5%)	353	23 (6.5%)	179 (50.7%)
2010	47	1 (2.1%)	33 (70.2%)	245	10 (4.1%)	137 (55.9%)	292	11 (3.8%)	170 (58.2%)
2011	54	1 (1.9%)	35 (64.8%)	270	12 (4.4%)	159 (58.9%)	324	13 (4.0%)	194 (59.9%)
2012	70	2 (2.9%)	54 (77.1%)	311	16 (5.1%)	173 (55.6%)	381	18 (4.7%)	227 (59.6%)
2013	82	1 (1.2%)	64 (78.0%)	313	15 (4.8%)	149 (47.6%)	395	16 (4.1%)	213 (53.9%)
2014	79	3 (3.8%)	58 (73.4%)	330	9 (2.7%)	180 (54.5%)	409	12 (2.9%)	238 (58.2%)
2015	85	1 (1.2%)	66 (77.6%)	311	10 (3.2%)	172 (55.3%)	396	11 (2.8%)	238 (60.1%)
2016	118	2 (1.7%)	82 (69.5%)	343	12 (3.5%)	155 (45.2%)	461	14 (3.0%)	237 (51.4%)
2017	83	1 (1.2%)	56 (67.5%)	350	2 (0.6%)	186 (53.1%)	433	3 (0.7%)	242 (55.9%)
2018	82	2 (2.4%)	53 (64.6%)	347	4 (1.2%)	165 (47.6%)	429	6 (1.4%)	218 (50.8%)
2019	115	2 (1.7%)	86 (74.8%)	376	8 (2.1%)	194 (51.6%)	491	10 (2.0%)	280 (57.0%)
2020	74	0 (0.0%)	49 (66.2%)	358	4 (1.1%)	197 (55.0%)	432	4 (0.9%)	246 (56.9%)
2021	123	2 (1.6%)	92 (74.8%)	376	6 (1.6%)	194 (51.6%)	499	8 (1.6%)	286 (57.3%)
Total	**1720**	**57 (3.3%)**	**1220 (70.9)**	**6350**	**297 (4.7%)**	**3193 (50.3%)**	**8070**	**354 (4.4%)**	**4413 (54.7%)**

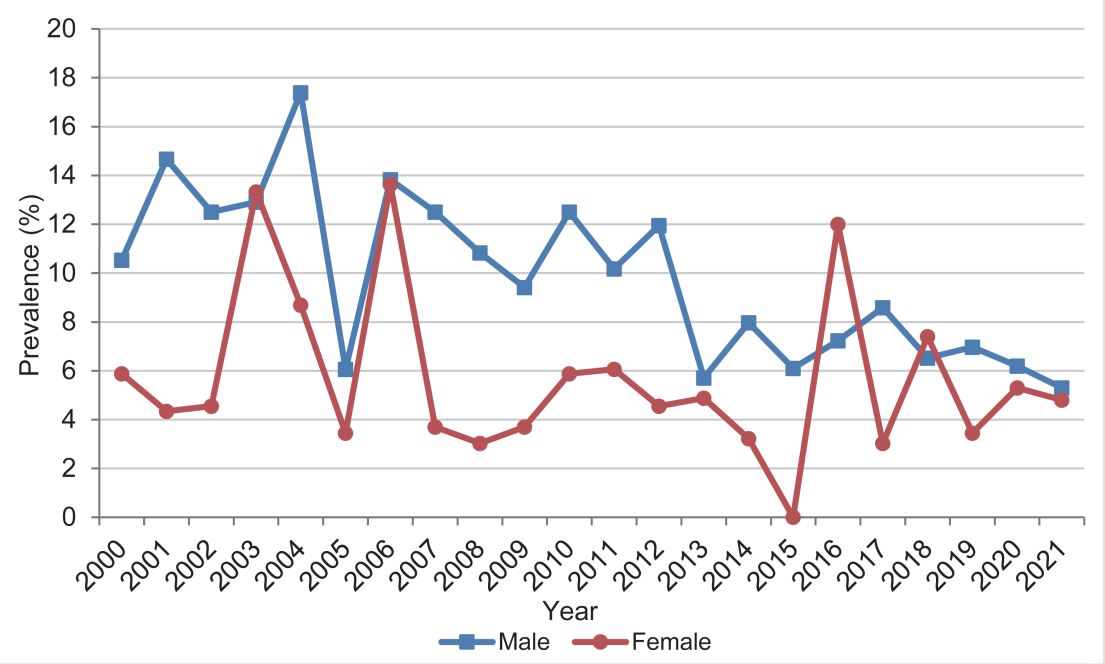
Box 41

HBsAg prevalence in drug users, female sex workers and HIV/AIDS patients attending ITC from 1992 to 2021 (Data sources: PHLSB, Social Hygiene Service, ITC, CHP, DH and Action for REACH OUT)

Year	Drug users	Female sex workers	HIV/AIDS patients attending ITC
1992	13.9	-	-
1993	14.4	-	-
1994	12.9	-	-
1995	10.5	6.8^	-
1996	8.7	6.8^	-
1997	6.6	6.8^	-
1998	10.0	6.8^	-
1999	11.2	-	-
2000	11.4	-	9.5
2001	11.6	-	12.2
2002	12.7	-	11.2
2003	10.1	-	13.0
2004	-	-	15.9
2005	-	-	5.6
2006	-	-	13.8
2007	-	10.4*	11.5
2008	-	9.0	9.7
2009	-	6.5	8.6
2010	-	5.0	11.3
2011	-	7.2**	9.5
2012	-	-	10.7
2013	-	-	5.6
2014	-	-	7.5
2015	-	-	5.6
2016	-	-	7.6
2017	-	-	8.1
2018	-	-	6.6
2019	-	-	6.5
2020	-	-	6.1
2021	-	-	5.3

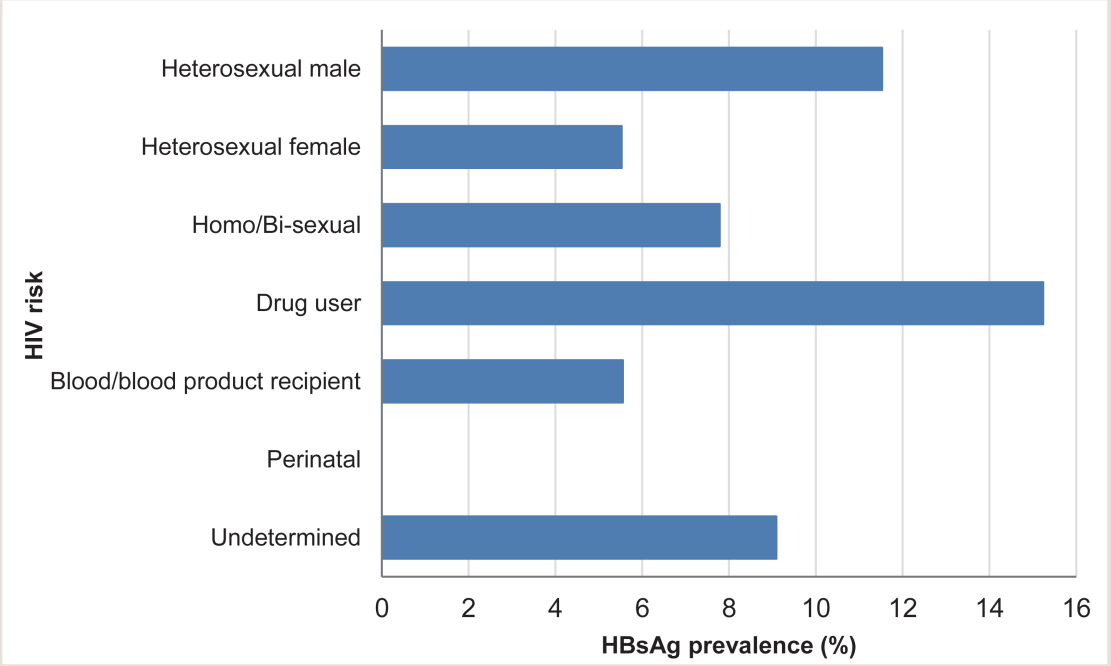
**For a period between Aug-Dec 2007;*
*** For a period between Jan-Jul 2011;*
^Figure is the average of 1995-1998

Box 42 Prevalence of HBsAg at baseline screening of HIV/AIDS patients attending ITC from 2000 to 2021 (Data source: ITC, CHP, DH)



Year	Male		Female		Total	
	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)	**No. tested**	**HBsAg +ve (%)**
2000	57	6 (10.5%)	17	1 (5.9%)	**74**	**7 (9.5%)**
2001	75	11 (14.7%)	23	1 (4.3%)	**98**	**12 (12.2%)**
2002	112	14 (12.5%)	22	1 (4.5%)	**134**	**15 (11.2%)**
2003	93	12 (12.9%)	15	2 (13.3%)	**108**	**14 (13.0%)**
2004	115	20 (17.4%)	23	2 (8.7%)	**138**	**22 (15.9%)**
2005	132	8 (6.1%)	29	1 (3.4%)	**161**	**9 (5.6%)**
2006	188	26 (13.8%)	22	3 (13.6%)	**210**	**29 (13.8%)**
2007	216	27 (12.5%)	27	1 (3.7%)	**243**	**28 (11.5%)**
2008	203	22 (10.8%)	33	1 (3.0%)	**236**	**23 (9.7%)**
2009	170	16 (9.4%)	27	1 (3.7%)	**197**	**17 (8.6%)**
2010	160	20 (12.5%)	34	2 (5.9%)	**194**	**22 (11.3%)**
2011	167	17 (10.2%)	33	2 (6.1%)	**200**	**19 (9.5%)**
2012	226	27 (11.9%)	44	2 (4.5%)	**270**	**29 (10.7%)**
2013	263	15 (5.7%)	41	2 (4.9%)	**304**	**17 (5.6%)**
2014	301	24 (8.0%)	31	1 (3.2%)	**332**	**25 (7.5%)**
2015	328	20 (6.1%)	26	0 (0.0%)	**354**	**20 (5.6%)**
2016	304	22 (7.2%)	25	3 (12.0%)	**329**	**25 (7.6%)**
2017	326	28 (8.6%)	33	1 (3.0%)	**359**	**29 (8.1%)**
2018	230	15 (6.5%)	27	2 (7.4%)	**257**	**17 (6.6%)**
2019	201	14 (7.0%)	29	1 (3.4%)	**230**	**15 (6.5%)**
2020	178	11 (6.2%)	19	1 (5.3%)	**197**	**12 (6.1%)**
2021	169	9 (5.3%)	21	1 (4.8%)	**190**	**10 (5.3%)**

Box 43 Prevalence of HBV infection per HIV risk at baseline screening of HIV/AIDS patients attending ITC from 2000 to 2021 (Data source: ITC, CHP, DH)



HIV risk	No. tested	HBsAg +ve (%)	Anti-HBs +ve (%)
Heterosexual male	911	105 (11.5%)	429 (47.1%)
Heterosexual female	561	31 (5.5%)	241 (43.0%)
Homo/Bi-sexual	2994	233 (7.8%)	1623 (54.2%)
Drug user	269	41 (15.2%)	128 (47.6%)
Blood/blood product recipient	18	1 (5.6%)	6 (33.3%)
Perinatal	9	0 (0%)	2 (22.2%)
Undetermined	55	5 (9.1%)	28 (50.9%)
Total	**4817**	**416 (8.6%)**	**2457 (51.0%)**

Box 44

Prevalence of hepatitis B markers in drug users from 1990 to 2010 (Data source: PHLSB, CHP, DH)

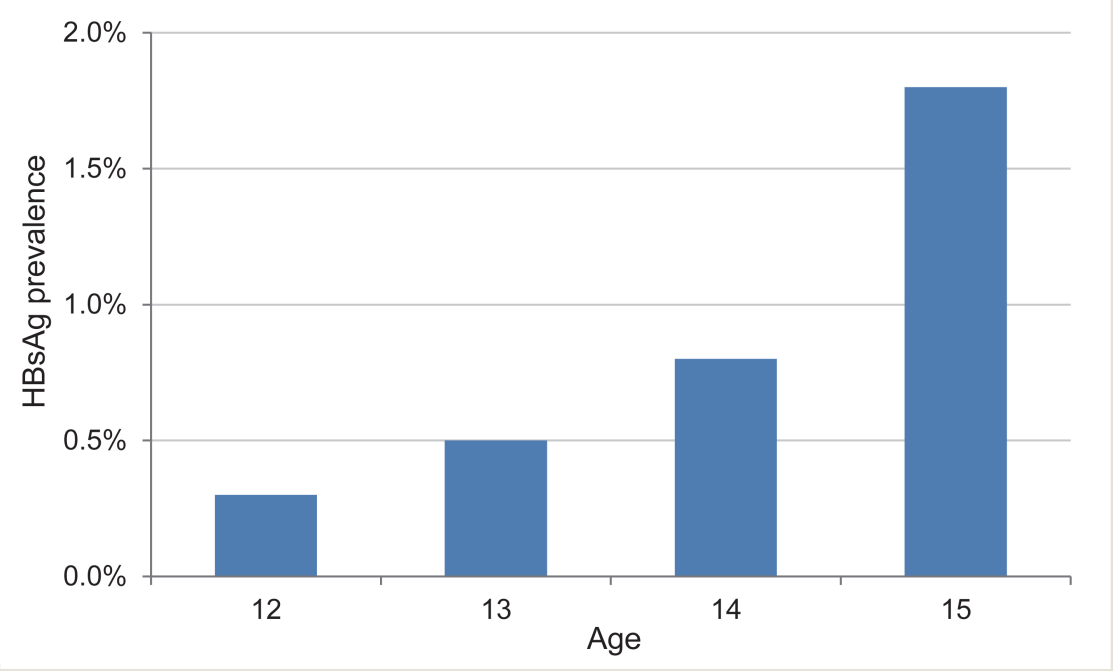
Year	No. tested	HBsAg (%+ve)	Anti-HBs (%+ve)	Anti-HBc* (%+ve)	Any marker (%+ve)
1990	1067	13.4	59.0	15.7	90.8
1991	1517	14.4	54.4	20.5	89.3
1992	832	13.9	49.0	21.4	84.4
1993	744	14.4	43.4	16.4	69.2
1994	607	12.9	38.1	13.5	64.1
1995	190	10.5	36.8	12.1	58.9
1996	358	8.7	43.0	12.6	62.8
1997	290	6.6	36.2	15.9	53.4
1998	290	10.0	43.4	7.9	59.3
1999	725	11.2	44.8	13.8	67.2
2000	892	11.4	42.5	15.8	67.8
2001	654	11.6	41.3	17.3	70.2
2002	553	12.7	43.0	16.6	72.3
2003	198	10.1	42.4	12.6	65.2
2004	45	11.1	57.8	4.4	73.3
2005	26	11.5	46.2	11.5	69.2
2006	6	33.3	50.0	16.7	100.0
2007	11	0.0	81.8	9.1	90.9
2008	7	28.6	28.6	14.3	71.4
2009	11	9.1	72.7	9.1	90.9
2010	12	8.3	58.3	8.3	75.0

**Anti-HBc was not tested in specimens that were HBsAg positive*

Box 45 Prevalence of HBsAg in participants of Community Research Project on Viral Hepatitis in 2001 (Data source: DH)

Age Group	Male		Female		**Total**	
	No. tested	HBsAg +ve (%)	No. tested	HBsAg +ve (%)	**No. tested**	**HBsAg +ve (%)**
18-30	72	6 (8.3%)	87	6 (6.9%)	**159**	**12 (7.5%)**
31-40	93	5 (5.4%)	144	20 (13.9%)	**237**	**25 (10.5%)**
41-50	100	20 (20.0%)	183	10 (5.5%)	**283**	**30 (10.6%)**
51 & Over	111	8 (7.2%)	146	7 (4.8%)	**257**	**15 (5.8%)**
Total	**376**	**39 (10.4%)**	**560**	**43 (7.7%)**	**936**	**82 (8.8%)**

Box 46 HBsAg prevalence by age among children aged 12 to 15 years in 2009 (Data source: unpublished data of DH)



**The overall seroprevalence of HBsAg was 0.78%.*

Vaccination coverage of hepatitis B

Box 47 Estimated coverage of birth-dose hepatitis B vaccine between 2010 and 2021 (Data source: DH and Census and Statistics Department)

Year	No. of first-dose hepatitis B vaccine administered to newborn babies at public and private hospitals	Number of live births	Birth-dose coverage
2010	88 148	88 584	99.5%
2011	95 113	95 451	99.6%
2012	91 073	91 558	99.5%
2013	56 565	57 084	99.1%
2014	61 813	62 305	99.2%
2015	59 520	59 878	99.4%
2016	60 522	60 856	99.5%
2017	56 403	56 548	99.7%
2018	53 506	53 716	99.6%
2019	52 603	52 856	99.5%
2020	42 876	43 031	99.6%
2021	36 849	36 953	99.7%

Box 48

Hepatitis B immunisation coverage among children aged 2 to 5 by year of birth (Data source: ref 55 - 61 & unpublished DH data)

Year of Survey	Year of Birth	First dose (%)	Second dose (%)	Third dose (%)
2001	1995	99.5	99.5	99.1
	1996	99.1	99.0	98.6
2003	1997	99.5	99.3	99.1
	1998	99.9	99.9	99.6
	1999	100	100	99.7
2006	2000	99.9	99.8	99.6
	2001	99.9	99.9	99.6
	2002	99.9	99.8	99.5
2009	2003	99.9	99.8	99.5
	2004	99.9	99.9	99.8
	2005	99.7	99.7	99.5
	2006	100	100	99.7
2012	2006	99.6	99.5	99.0
	2007	99.8	99.8	99.3
	2008	99.8	99.8	99.3
	2009	100	100	98.8
2015	2009	99.7	99.6	99.2
	2010	99.6	99.6	99.2
	2011	99.6	99.5	99.2
	2012	100	100	99.2
2018	2012	100	100	99.8
	2013	100	99.9	99.5
	2014	99.9	99.8	99.7
2021*	2015	99.9	99.9	99.5
	2016	99.7	99.6	99.2
	2017	99.8	99.5	99.1

*Provisional figures

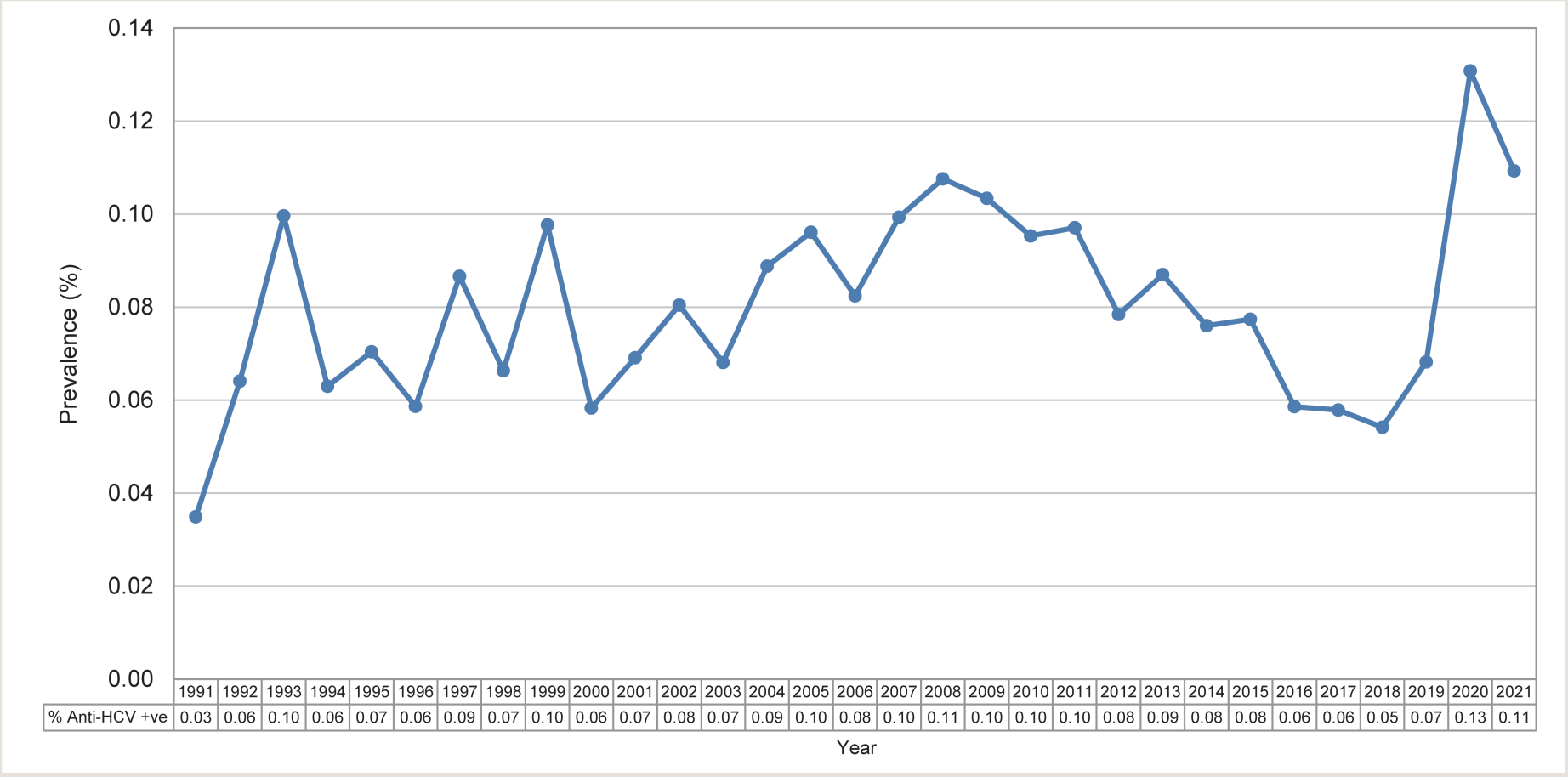
Box 49 Cumulative statistics of the supplementary hepatitis B vaccination programme for Primary 6 students from the school years 2003 to 2021 (Data source: DH)

	2003-2004	2004-2005	2005-2006	2006-2007	2007-2008	2008-2009	2009-2010	2010-2011	2011-2012	2012-2013	2013-2014	2014-2015	2015-2016	2016-2017	2017-2018	2018-2019	2019-2020	2020-2021
Cumulative no. of Primary 6 students	86208	83974	83164	81818	77273	73757	67310	63332	63394	57487	54845	52013	51009	52848	55660	59481	59339	58710
First Dose																		
Cumulative no. eligible for vaccination	10625	8433	6648	6351	6204	5165	4698	3736	2509	2376	1992	1797	982	710	483	407	429	889
Cumulative no. administered	10519	8313	6591	6262	6095	5043	4520	3563	2318	2237	1810	1606	729	588	346	218	148	105
Acceptance rate (at the present campaign)	99.0%	98.6%	99.1%	98.6%	98.2%	97.6%	96.2%	95.4%	92.4%	94.1%	90.9%	89.4%	74.2%	82.8%	71.6%	53.6%	34.5%	11.8%
Coverage (for the whole Primary 6 population)	99.9%	99.8%	99.9%	99.9%	99.9%	99.8%	99.7%	99.7%	99.7%	99.8%	99.7%	99.6%	98.4%	98.6%	98.5%	98.5%	98.0%	98.2%
Second Dose																		
Cumulative no. eligible for vaccination	10626	8545	6710	6392	6243	5165	4698	3787	2573	2432	2033	1825	1025	753	540	443	453	918
Cumulative no. administered	10341	8185	6573	6278	6068	4969	4398	3516	2286	2203	1718	1578	675	589	384	224	151	87
Acceptance rate (at the present campaign)	97.3%	95.8%	98.0%	98.2%	97.2%	96.2%	93.6%	92.8%	88.8%	90.6%	84.5%	86.5%	65.9%	78.2%	71.1%	50.6%	33.3%	9.5%
Coverage (for the whole Primary 6 population)	99.7%	99.6%	99.8%	99.8%	99.8%	99.7%	99.5%	99.6%	99.5%	99.6%	99.4%	99.5%	98.2%	98.6%	98.5%	98.5%	98.0%	98.1%
Third Dose																		
Cumulative no. eligible for vaccination	11222	9300	7397	6986	6741	5575	5032	4104	2825	2692	2283	2096	1307	1071	965	938	711	1116
Cumulative no. administered	10069	8478	6965	6607	6273	4817	4409	3526	2344	2232	1777	1708	835	839	734	579	320	186
Acceptance rate (at the present campaign)	89.7%	91.2%	94.2%	94.6%	93.1%	86.4%	87.6%	85.9%	83.0%	82.9%	77.8%	81.5%	63.9%	78.3%	76.1%	61.7%	45.0%	16.7%
Coverage (for the whole Primary 6 population)	98.7%	99.0%	99.5%	99.5%	99.4%	99.0%	99.1%	99.1%	99.2%	99.2%	99.1%	99.3%	97.9%	98.4%	98.3%	98.2%	97.8%	98.0%

Seroprevalence of hepatitis C

Box 50

Anti-HCV prevalence in new blood donors from 1991 to 2021 (Data source: HKRCBTS)



	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021
% Anti-HCV +ve	0.03	0.06	0.10	0.06	0.07	0.06	0.09	0.07	0.10	0.06	0.07	0.08	0.07	0.09	0.10	0.08	0.10	0.11	0.10	0.10	0.10	0.08	0.09	0.08	0.08	0.06	0.06	0.05	0.07	0.13	0.11

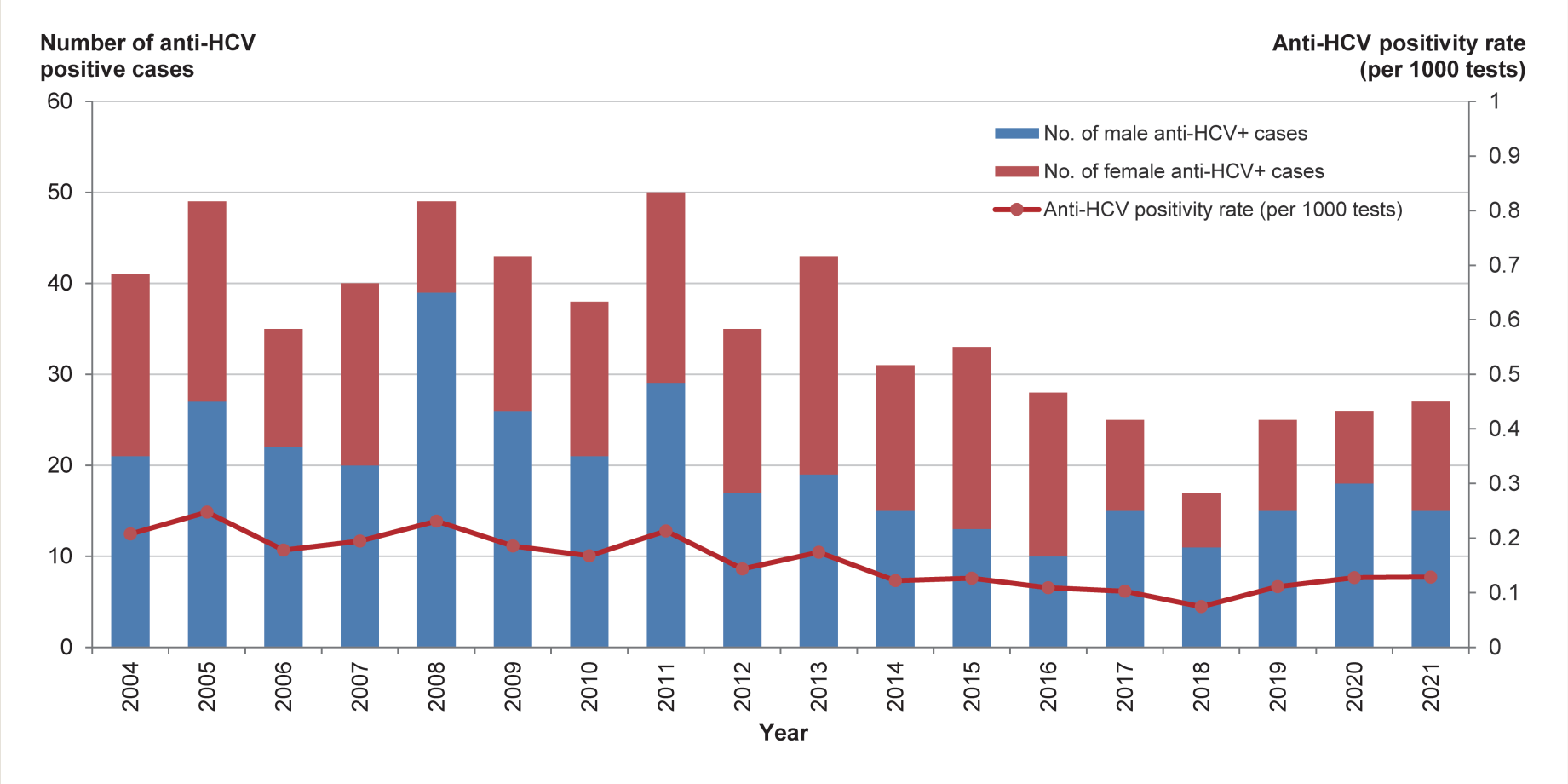
Box 51 Anti-HCV prevalence and its sex and age breakdown in new blood donors in 2021 (Data source: HKRCBTS)

Age group	Male		Female		**Total**	
	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	**No. tested**	**Anti-HCV +ve (%)**
16-19	2424	0 (0.00%)	3243	1 (0.03%)	5667	1 (0.02%)
20-29	2207	1 (0.05%)	2631	0 (0.00%)	4838	1 (0.02%)
30-39	1606	4 (0.25%)	2463	2 (0.08%)	4069	6 (0.15%)
40-49	1182	5 (0.42%)	2165	3 (0.14%)	3347	8 (0.24%)
>49	745	3 (0.40%)	1463	3 (0.21%)	2208	6 (0.27%)
Total	**8164**	**13 (0.16%)**	**11965**	**9 (0.08%)**	**20129**	**22 (0.11%)**

Box 52 Prevalence of anti-HCV in participants of Community Research Project on Viral Hepatitis in 2001 (Data source: DH)

Age group	No. Tested	Anti-HCV +ve (%)
18-29	137	0 (0.0%)
30-39	223	1 (0.4%)
40-49	291	0 (0.0%)
50-59	170	2 (1.2%)
60 & over	115	0 (0.0%)
All	**936**	**3 (0.3%)**

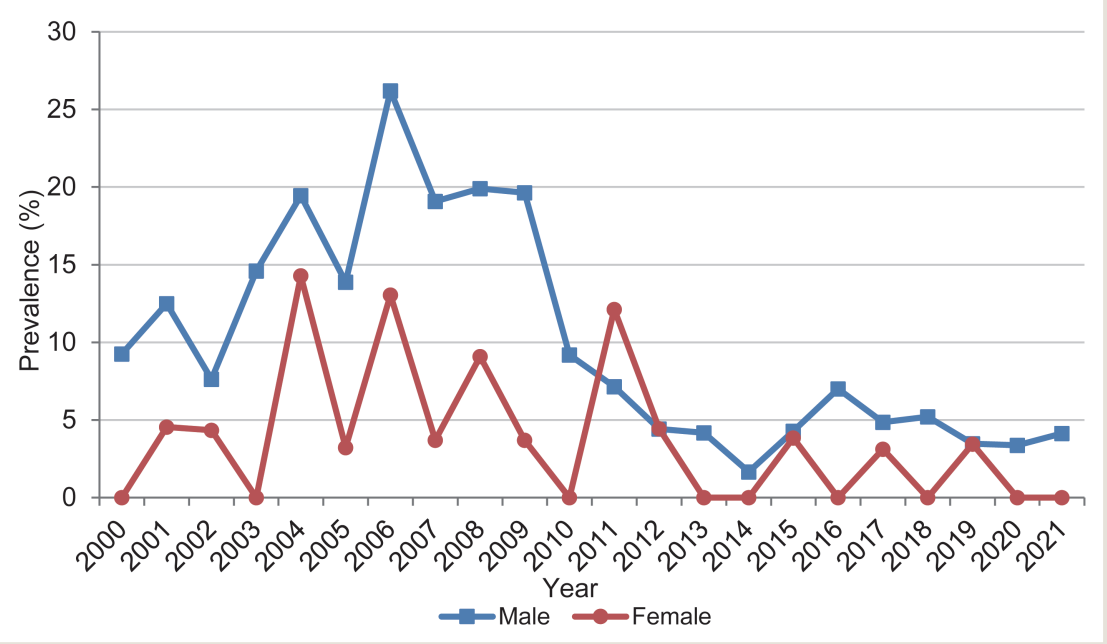
Box 53 **Prevalence of anti-HCV from screening of blood donors from 2004 to 2021 (Data source: HKRCBTS)**



Box 54 **Prevalence of anti-HCV in persons attending Therapeutic Prevention Clinic of ITC for post-exposure management, from 2000 to 2021 (Data source: ITC, CHP, DH)**

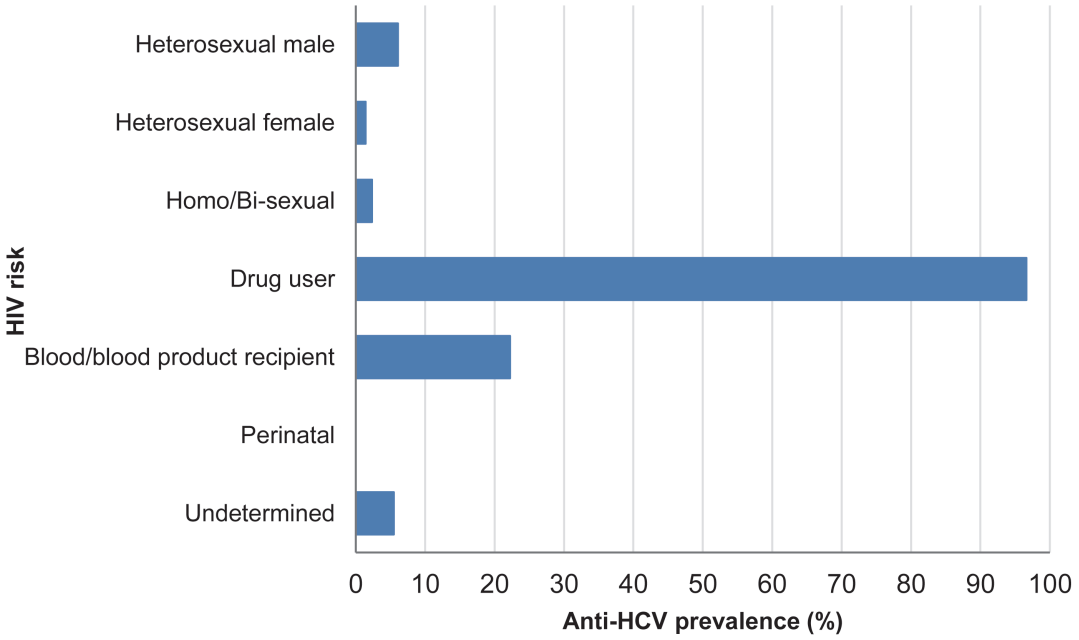
Year	Health care workers		Non- Health care workers		**Total**	
	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	**No. tested**	**Anti-HCV +ve (%)**
2000	15	0 (0.0%)	20	1 (5.0%)	35	1 (2.9%)
2001	22	0 (0.0%)	50	1 (2.0%)	72	1 (1.4%)
2002	27	0 (0.0%)	50	1 (2.0%)	77	1 (1.3%)
2003	18	0 (0.0%)	43	0 (0.0%)	61	0 (0.0%)
2004	17	0 (0.0%)	40	0 (0.0%)	57	0 (0.0%)
2005	10	0 (0.0%)	57	0 (0.0%)	67	0 (0.0%)
2006	33	0 (0.0%)	139	0 (0.0%)	172	0 (0.0%)
2007	36	0 (0.0%)	118	0 (0.0%)	154	0 (0.0%)
2008	23	0 (0.0%)	126	3 (2.4%)	149	3 (2.0%)
2009	25	0 (0.0%)	161	1 (0.6%)	186	1 (0.5%)
2010	25	0 (0.0%)	131	0 (0.0%)	156	0 (0.0%)
2011	17	0 (0.0%)	145	0 (0.0%)	162	0 (0.0%)
2012	37	0 (0.0%)	154	0 (0.0%)	191	0 (0.0%)
2013	26	0 (0.0%)	162	1 (0.6%)	188	1 (0.5%)
2014	29	0 (0.0%)	157	0 (0.0%)	186	0 (0.0%)
2015	34	0 (0.0%)	150	0 (0.0%)	184	0 (0.0%)
2016	47	1 (2.1%)	145	1 (0.7%)	192	2 (1.0%)
2017	38	0 (0.0%)	165	0 (0.0%)	203	0 (0.0%)
2018	41	0 (0.0%)	172	0 (0.0%)	213	0 (0.0%)
2019	66	0 (0.0%)	172	0 (0.0%)	238	0 (0.0%)
2020	38	0 (0.0%)	189	1 (0.5%)	227	1 (0.4%)
2021	46	1 (2.2%)	187	1 (0.5%)	233	2 (0.9%)
Total	**670**	**2 (0.3%)**	**2733**	**11 (0.4%)**	**3403**	**13 (0.4%)**

Box 55 Prevalence of anti-HCV at baseline screening of HIV/AIDS patients attending ITC from 2000 to 2021 (Data source: ITC, CHP, DH)



Year	Male		Female		Total	
	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	**No. tested**	**Anti-HCV +ve (%)**
2000	54	5 (9.3%)	15	0 (0.0%)	**69**	**5 (7.2%)**
2001	72	9 (12.5%)	22	1 (4.5%)	**94**	**10 (10.6%)**
2002	118	9 (7.6%)	23	1 (4.3%)	**141**	**10 (7.1%)**
2003	89	13 (14.6%)	14	0 (0.0%)	**103**	**13 (12.6%)**
2004	108	21 (19.4%)	21	3 (14.3%)	**129**	**24 (18.6%)**
2005	137	19 (13.9%)	31	1 (3.2%)	**168**	**20 (11.9%)**
2006	187	49 (26.2%)	23	3 (13.0%)	**210**	**52 (24.8%)**
2007	215	41 (19.1%)	27	1 (3.7%)	**242**	**42 (17.4%)**
2008	201	40 (19.9%)	33	3 (9.1%)	**234**	**43 (18.4%)**
2009	168	33 (19.6%)	27	1 (3.7%)	**195**	**34 (17.4%)**
2010	163	15 (9.2%)	33	0 (0.0%)	**196**	**15 (7.7%)**
2011	168	12 (7.1%)	33	4 (12.1%)	**201**	**16 (8.0%)**
2012	226	10 (4.4%)	45	2 (4.4%)	**271**	**12 (4.4%)**
2013	264	11 (4.2%)	40	0 (0.0%)	**304**	**11 (3.6%)**
2014	301	5 (1.7%)	31	0 (0.0%)	**332**	**5 (1.5%)**
2015	327	14 (4.3%)	26	1 (3.8%)	**353**	**15 (4.2%)**
2016	300	21 (7.0%)	25	0 (0.0%)	**325**	**21 (6.5%)**
2017	330	16 (4.8%)	32	1 (3.1%)	**362**	**17 (4.7%)**
2018	230	12 (5.2%)	27	0 (0.0%)	**257**	**12 (4.7%)**
2019	201	7 (3.5%)	29	1 (3.4%)	**230**	**8 (3.5%)**
2020	178	6 (3.4%)	19	0 (0.0%)	**197**	**6 (3.0%)**
2021	169	7 (4.1%)	21	0 (0.0%)	**190**	**7 (3.7%)**

Box 56 Prevalence of anti-HCV per HIV risk at baseline screening of HIV/AIDS patients attending ITC from 2000 to 2021 (Data source: ITC, CHP, DH)



HIV risk	No. tested	Anti-HCV +ve (%)
Heterosexual male	906	55* (6.1%)
Heterosexual female	557	8 (1.4%)
Homo/Bi-sexual	2992	70 (2.3%)
Drug user	268	259 (96.6%)
Blood/blood product recipient	18	4 (22.2%)
Perinatal	9	0 (0.0%)
Undetermined	55	3 (5.5%)
Total	**4805**	**399 (8.3%)**

**31 out of 55 had a history of injecting drug use*

Box 57

Prevalence of anti-HCV from clinical testing of patients in 2 hospital clusters under Hospital Authority from 2011 to 2021 (Data source: PMH Microbiology Laboratory and PWH Microbiology Laboratory)

Category	2011		2012		2013		2014		2015		2016		2017		2018		2019		2020		2021		Overall	
	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)	No. tested	Anti-HCV +ve (%)
(a) Screening																								
Pre-transplant	80	0 (0.0%)	96	0 (0.0%)	82	0 (0.0%)	111	1 (0.9%)	118	0 (0.0%)	108	0 (0.0%)	128	0 (0.0%)	90	0 (0.0%)	75	1 (1.3%)	48	0 (0.0%)	69	1 (1.4%)	1005	3 (0.3%)
Drug users	84	61 (72.6%)	103	53 (51.5%)	112	63 (56.3%)	114	66 (57.9%)	124	51 (41.1%)	81	41 (50.6%)	87	38 (43.7%)	103	40 (38.8%)	90	35 (38.9%)	90	39 (43.3%)	80	32 (40.0%)	1068	519 (48.6%)
Needlestick injuries	559	4 (0.7%)	592	6 (1.0%)	610	4 (0.7%)	537	6 (1.1%)	494	3 (0.6%)	516	5 (1.0%)	667	9 (1.3%)	614	2 (0.3%)	678	7 (1.0%)	674	11 (1.6%)	790	13 (1.6%)	6731	70 (1.0%)
Haemodialysis/ peritoneal dialysis	2251	34 (1.5%)	2452	34 (1.4%)	2449	37 (1.5%)	2569	34 (1.3%)	2535	48 (1.9%)	2613	34 (1.3%)	3557	60 (1.7%)	3021	44 (1.5%)	2713	33 (1.2%)	2526	33 (1.3%)	2645	33 (1.2%)	29331	424 (1.4%)
Post-renal transplant	722	18 (2.5%)	737	17 (2.3%)	718	16 (2.2%)	692	15 (2.2%)	863	18 (2.1%)	541	6 (1.1%)	708	9 (1.3%)	611	6 (1.0%)	636	5 (0.8%)	432	4 (0.9%)	396	2 (0.5%)	7056	116 (1.6%)
Haematology (pre-chemotherapy)	399	1 (0.3%)	415	4 (1.0%)	444	2 (0.5%)	472	2 (0.4%)	489	4 (0.8%)	533	2 (0.4%)	687	6 (0.9%)	622	2 (0.3%)	615	2 (0.3%)	655	5 (0.8%)	711	5 (0.7%)	6042	35 (0.6%)
Rheumatology (pre-methotrexate)	464	2 (0.4%)	449	2 (0.4%)	471	4 (0.8%)	580	3 (0.5%)	689	5 (0.7%)	730	5 (0.7%)	1285	3 (0.2%)	1310	8 (0.6%)	1501	6 (0.4%)	1484	2 (0.1%)	1713	5 (0.3%)	10676	45 (0.4%)
History of blood transfusion	168	19 (11.3%)	197	17 (8.6%)	275	28 (10.2%)	224	22 (9.8%)	222	15 (6.8%)	166	14 (8.4%)	292	16 (5.5%)	222	18 (8.1%)	211	18 (8.5%)	238	16 (6.7%)	211	13 (6.2%)	2426	196 (8.1%)
Pre-vaccination	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	5	0 (0.0%)	0	0 (0.0%)	5	0 (0.0%)
Subtotal (a)	4727	139 (2.9%)	5041	133 (2.6%)	5161	154 (3.0%)	5299	149 (2.8%)	5534	144 (2.6%)	5288	107 (2.0%)	7411	141 (1.9%)	6593	120 (1.8%)	6519	107 (1.6%)	6152	110 (1.8%)	6615	104 (1.6%)	64340	1408 (2.2%)
(b) *Clinical indication	8196	293 (3.6%)	9815	308 (3.1%)	10911	323 (3.0%)	11229	316 (2.8%)	12360	351 (2.8%)	15472	383 (2.5%)	15889	329 (2.1%)	15208	338 (2.2%)	16028	302 (1.9%)	15307	278 (1.8%)	18289	302 (1.7%)	148704	3523 (2.4%)
(c) Others or unknown	8835	132 (1.5%)	9026	131 (1.5%)	9615	136 (1.4%)	11213	150 (1.3%)	10836	107 (1.0%)	10701	125 (1.2%)	15527	171 (1.1%)	18844	179 (0.9%)	19100	182 (1.0%)	19027	166 (0.9%)	21781	179 (0.8%)	154505	1658 (1.1%)
Total (a+b+c)	**21758**	**564 (2.6%)**	**23882**	**572 (2.4%)**	**25687**	**613 (2.4%)**	**27741**	**615 (2.2%)**	**28730**	**602 (2.1%)**	**31461**	**615 (2.0%)**	**38827**	**641 (1.7%)**	**40645**	**637 (1.6%)**	**41647**	**591 (1.4%)**	**40486**	**554 (1.4%)**	**46685**	**585 (1.3%)**	**367549**	**6589 (1.8%)**

**includes suspected hepatitis, work up for liver function derangement and others*

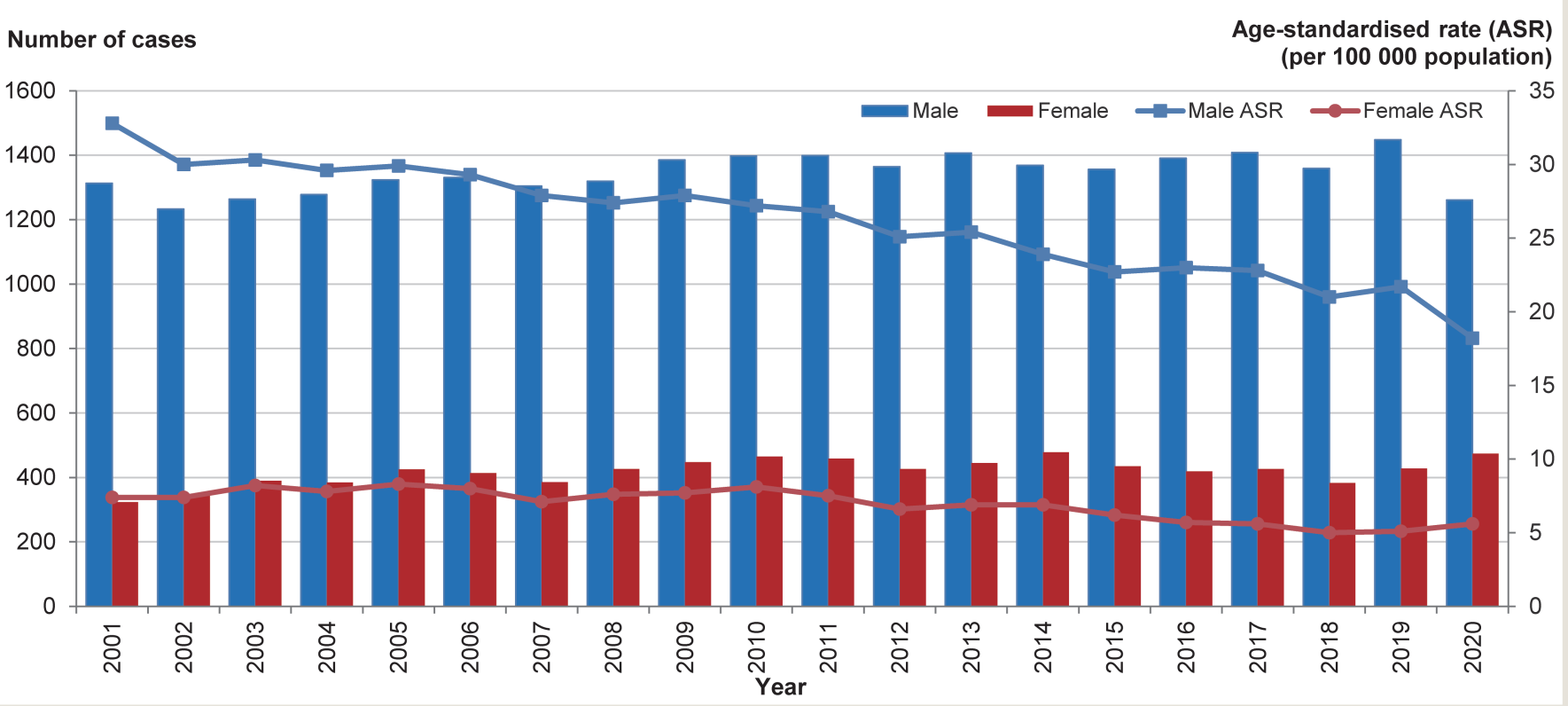
Box 58 Characteristics of anti-HCV positive subjects detected in 2 hospital clusters under Hospital Authority from 2008 to 2021 (Data source: PMH Microbiology Laboratory and PWH Microbiology Laboratory)

		2008 (n=494)	2009 (n=542)	2010 (n=537)	2011 (n=565)	2012 (n=574)	2013 (n=616)	2014 (n=615)	2015 (n=602)	2016 (n=615)	2017 (n=641)	2018 (n=638)	2019 (n=592)	2020 (n=554)	2021 (n=585)	**Overall (n=8170)**
		No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	No. (%)	**No. (%)**
Lab	PMH	208 (42.1%)	273 (50.4%)	271 (50.5%)	280 (49.6%)	298 (51.9%)	279 (45.3%)	297 (48.3%)	354 (58.8%)	372 (60.5%)	340 (53.0%)	363 (56.9%)	312 (52.7%)	300 (54.2%)	295 (50.4%)	**4242 (51.9%)**
	PWH	286 (57.9%)	269 (49.6%)	266 (49.5%)	285 (50.4%)	276 (48.1%)	337 (54.7%)	318 (51.7%)	248 (41.2%)	243 (39.5%)	301 (47.0%)	275 (43.1%)	280 (47.3%)	254 (45.8%)	290 (49.6%)	**3928 (48.1%)**
Sex	Male	339 (68.6%)	389 (71.8%)	384 (71.5%)	405 (71.7%)	421 (73.3%)	445 (72.2%)	425 (69.1%)	421 (69.9%)	443 (72.0%)	439 (68.6%)	460 (72.2%)	419 (70.8%)	409 (73.8%)	432 (73.8%)	**5831 (71.4%)**
	Female	155 (31.4%)	153 (28.2%)	153 (28.5%)	160 (28.3%)	153 (26.7%)	171 (27.8%)	190 (30.9%)	181 (30.1%)	172 (28.0%)	201 (31.4%)	177 (27.8%)	173 (29.2%)	145 (26.2%)	153 (26.2%)	**2337 (28.6%)**
	Unknown	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.2%)	1 (0.2%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	**2 (<0.1%)**
Age at diagnosis	Mean	52.0	55.1	52.9	52.5	52.5	52.4	53.2	55.0	55.5	56.3	56.2	56.4	56.7	56.9	**54.6**
	S.D.	16.9	16.7	16.2	15.8	15.6	15.9	15.7	15.1	15.1	15.1	15.2	14.6	15.0	15.2	**15.6**
	Range	0 – 88	1 – 102	0 – 90	0 – 90	0 – 99	0 – 113	0 – 95	1 – 95	0 – 97	0 – 94	0 – 99	0 – 96	0 – 96	0 – 99	**0 – 113**
Category	Pre-transplant	0 (0.0%)	1 (0.2%)	2 (0.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	1 (0.2%)	**6 (0.1%)**
	Drug users	66 (13.4%)	93 (17.2%)	75 (14.0%)	61 (10.8%)	53 (9.2%)	63 (10.2%)	66 (10.7%)	51 (8.5%)	41 (6.7%)	38 (5.9%)	40 (6.3%)	35 (5.9%)	39 (7.0%)	32 (5.5%)	**753 (9.2%)**
	Needlestick injuries	6 (1.2%)	5 (0.9%)	5 (0.9%)	4 (0.7%)	6 (1.0%)	4 (0.6%)	6 (1.0%)	3 (0.5%)	5 (0.8%)	9 (1.4%)	2 (0.3%)	7 (1.2%)	11 (2.0%)	13 (2.2%)	**86 (1.1%)**
	Pre-haemodialysis/ peritoneal dialysis	31 (6.3%)	34 (6.3%)	36 (6.7%)	34 (6.0%)	34 (5.9%)	37 (6.0%)	34 (5.5%)	48 (8.0%)	34 (5.5%)	60 (9.4%)	44 (6.9%)	33 (5.6%)	33 (6.0%)	33 (5.6%)	**525 (6.4%)**
	Post-renal transplant	21 (4.3%)	19 (3.5%)	25 (4.7%)	18 (3.2%)	17 (3.0%)	16 (2.6%)	15 (2.4%)	18 (3.0%)	6 (1.0%)	9 (1.4%)	6 (0.9%)	5 (0.8%)	4 (0.7%)	2 (0.3%)	**181 (2.2%)**
	Haematology	5 (1.0%)	2 (0.4%)	6 (1.1%)	1 (0.2%)	4 (0.7%)	2 (0.3%)	2 (0.3%)	4 (0.7%)	2 (0.3%)	6 (0.9%)	2 (0.3%)	2 (0.3%)	5 (0.9%)	5 (0.9%)	**48 (0.6%)**
	Pre-methotrexate	1 (0.2%)	5 (0.9%)	1 (0.2%)	2 (0.4%)	2 (0.3%)	4 (0.6%)	3 (0.5%)	5 (0.8%)	5 (0.8%)	3 (0.5%)	8 (1.3%)	6 (1.0%)	2 (0.4%)	5 (0.9%)	**52 (0.6%)**
	History of blood transfusion	18 (3.6%)	32 (5.9%)	21 (3.9%)	19 (3.4%)	17 (3.0%)	28 (4.5%)	22 (3.6%)	15 (2.5%)	14 (2.3%)	16 (2.5%)	18 (2.8%)	18 (3.0%)	16 (2.9%)	13 (2.2%)	**267 (3.3%)**
	Clinical Indication	215 (43.5%)	216 (39.9%)	262 (48.8%)	293 (51.9%)	308 (53.7%)	323 (52.4%)	316 (51.4%)	351 (58.3%)	383 (62.3%)	329 (51.3%)	338 (53.0%)	302 (51.0%)	278 (50.2%)	302 (51.6%)	**4216 (51.6%)**
	Others or unknown	131 (26.5%)	135 (24.9%)	104 (19.4%)	133 (23.5%)	133 (23.2%)	139 (22.6%)	150 (24.4%)	107 (17.8%)	125 (20.3%)	171 (26.7%)	180 (28.2%)	183 (30.9%)	166 (30.0%)	179 (30.6%)	**2036 (24.9%)**

Liver cancers

(Data source: Hong Kong Cancer Registry, Hospital Authority)

Box 59 Number of new liver cancer cases and age-standardised incidence rate by gender from 2001 – 2020 (Data source: Hong Kong Cancer Registry, Hospital Authority)



Year	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Female	324	343	390	385	425	414	386	426	447	465	459	426	445	478	435	419	426	383	428	474
Male	1313	1233	1264	1278	1324	1331	1304	1319	1385	1398	1399	1364	1407	1369	1356	1391	1408	1359	1448	1261
Total	**1637**	**1576**	**1654**	**1663**	**1749**	**1745**	**1690**	**1745**	**1832**	**1863**	**1858**	**1790**	**1852**	**1847**	**1791**	**1810**	**1834**	**1742**	**1876**	**1735**

Box 60

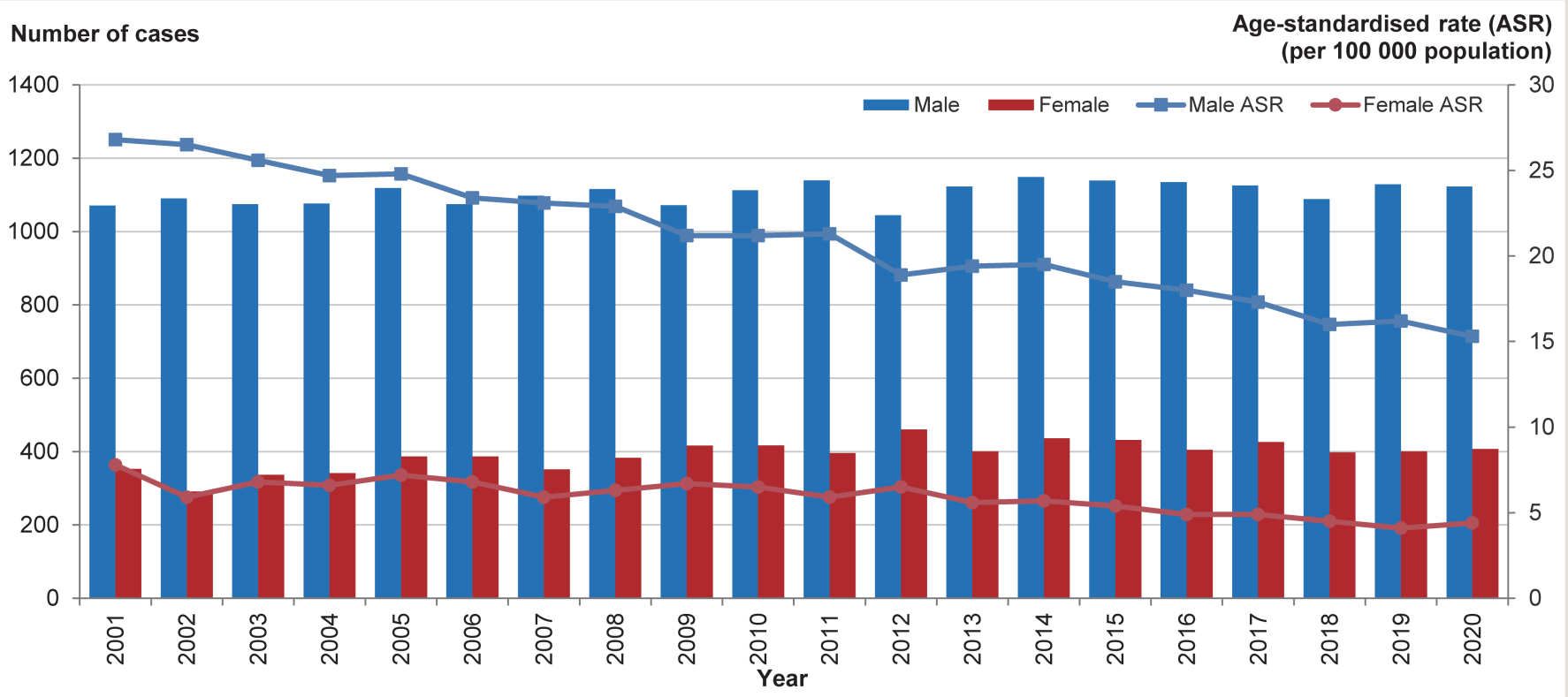
Number of new liver cancer cases and incidence rate by age and gender, from 2001 – 2020 (Data source: Hong Kong Cancer Registry, Hospital Authority)

Year	0-19						20-44						45-64						65+						Crude rate			ASR		
	Male		Female		**Total**		Male		Female		**Total**		Male		Female		**Total**		Male		Female		**Total**		Male	Female	**Total**	Male	Female	**Total**
	N	I	N	I	**N**	**I**	N	I	N	I	**N**	**I**	N	I	N	I	**N**	**I**	N	I	N	I	**N**	**I**	CR	CR	**CR**	ASR	ASR	**ASR**
2001	4	0.5	1	0.1	**5**	**0.3**	130	9.5	26	1.7	**156**	**5.3**	590	76.9	86	12.1	**676**	**45.7**	589	169.3	211	52.0	**800**	**106.2**	40.0	9.4	**24.4**	32.8	7.4	**20.1**
2002	4	0.5	2	0.3	**6**	**0.4**	130	9.7	17	1.1	**147**	**5.1**	534	67.1	79	10.5	**613**	**39.5**	565	157.6	245	58.5	**810**	**104.2**	37.6	9.9	**23.4**	30.0	7.4	**18.6**
2003	6	0.8	2	0.3	**8**	**0.5**	110	8.4	25	1.6	**135**	**4.7**	581	70.5	100	12.6	**681**	**42.1**	567	154.5	263	61.4	**830**	**104.4**	38.8	11.2	**24.6**	30.3	8.2	**19.1**
2004	2	0.3	1	0.1	**3**	**0.2**	121	9.4	18	1.2	**139**	**4.9**	554	64.6	91	10.9	**645**	**38.1**	601	159.2	275	62.3	**876**	**107.0**	39.1	10.9	**24.5**	29.6	7.8	**18.5**
2005	2	0.3	0	0.0	**2**	**0.1**	110	8.7	21	1.4	**131**	**4.7**	605	67.5	110	12.4	**715**	**40.1**	607	157.8	294	65.3	**901**	**107.9**	40.6	12.0	**25.7**	29.9	8.3	**18.9**
2006	6	0.8	1	0.1	**7**	**0.5**	88	7.1	21	1.4	**109**	**3.9**	637	68.5	109	11.8	**746**	**40.2**	600	152.6	283	61.7	**883**	**103.6**	40.7	11.5	**25.4**	29.3	8.0	**18.4**
2007	2	0.3	1	0.2	**3**	**0.2**	83	6.8	13	0.8	**96**	**3.5**	621	64.7	95	9.8	**716**	**37.1**	598	148.3	277	59.1	**875**	**100.3**	39.7	10.6	**24.4**	27.9	7.1	**17.2**
2008	1	0.1	1	0.2	**2**	**0.1**	90	7.5	24	1.6	**114**	**4.2**	636	64.0	135	13.2	**771**	**38.3**	592	144.6	266	56.2	**858**	**97.2**	40.1	11.6	**25.1**	27.4	7.6	**17.2**
2009	2	0.3	2	0.3	**4**	**0.3**	87	7.4	20	1.3	**107**	**4.0**	695	68.0	131	12.3	**826**	**39.6**	601	143.8	294	61.1	**895**	**99.6**	42.2	12.1	**26.3**	27.9	7.7	**17.5**
2010	0	0.0	4	0.7	**4**	**0.3**	78	6.7	23	1.5	**101**	**3.8**	711	67.9	140	12.6	**851**	**39.5**	609	142.4	298	60.7	**907**	**98.7**	42.4	12.5	**26.5**	27.2	8.1	**17.3**
2011	6	0.9	3	0.5	**9**	**0.7**	85	7.4	22	1.5	**107**	**4.0**	694	65.0	122	10.7	**816**	**36.9**	614	140.1	312	62.0	**926**	**98.4**	42.4	12.2	**26.3**	26.8	7.5	**16.8**
2012	2	0.3	1	0.2	**3**	**0.2**	69	6.0	25	1.6	**94**	**3.5**	654	60.6	108	9.2	**762**	**33.9**	639	140.1	292	55.7	**931**	**95.0**	41.0	11.1	**25.0**	25.1	6.6	**15.5**
2013	6	1.0	2	0.3	**8**	**0.7**	64	5.6	19	1.2	**83**	**3.1**	698	64.3	126	10.6	**824**	**36.2**	639	134.5	298	54.7	**937**	**91.9**	42.3	11.6	**25.8**	25.4	6.9	**15.8**
2014	3	0.5	1	0.2	**4**	**0.3**	69	6.0	17	1.1	**86**	**3.2**	644	59.2	130	10.8	**774**	**33.7**	653	131.7	330	58.1	**983**	**92.4**	40.9	12.3	**25.5**	23.9	6.9	**15.0**
2015	1	0.2	2	0.3	**3**	**0.3**	51	4.4	14	0.9	**65**	**2.4**	621	57.2	107	8.7	**728**	**31.5**	683	131.3	312	52.5	**995**	**89.3**	40.3	11.1	**24.6**	22.7	6.2	**14.1**
2016	1	0.2	2	0.4	**3**	**0.3**	64	5.6	9	0.6	**73**	**2.7**	679	62.6	118	9.5	**797**	**34.2**	647	119.2	290	46.8	**937**	**80.6**	41.2	10.6	**24.7**	23.0	5.7	**13.9**
2017	3	0.5	3	0.5	**6**	**0.5**	71	6.2	17	1.1	**88**	**3.3**	618	56.8	111	8.7	**729**	**30.9**	716	126.1	295	45.6	**1011**	**83.2**	41.5	10.7	**24.8**	22.8	5.6	**13.7**
2018	1	0.2	2	0.4	**3**	**0.3**	48	4.2	15	1.0	**63**	**2.4**	587	53.7	91	7.0	**678**	**28.3**	723	121.9	275	40.7	**998**	**78.7**	39.8	9.5	**23.4**	21.0	5.0	**12.5**
2019	1	0.2	1	0.2	**2**	**0.2**	60	5.3	7	0.5	**67**	**2.6**	594	54.2	115	8.7	**709**	**29.3**	793	127.7	305	43.2	**1098**	**82.7**	42.3	10.5	**25.0**	21.7	5.1	**12.9**
2020	3	0.5	2	0.4	**5**	**0.5**	27	2.4	10	0.7	**37**	**1.4**	515	47.3	109	8.2	**624**	**25.8**	716	110.3	353	48.2	**1069**	**77.4**	36.9	11.7	**23.2**	18.2	5.6	**11.5**
Average	**3**	**0.4**	**2**	**0.3**	**5**	**0.3**	**82**	**6.8**	**18**	**1.2**	**100**	**3.7**	**623**	**62.5**	**111**	**10.3**	**734**	**35.4**	**638**	**137.9**	**288**	**54.3**	**926**	**93.2**	**40.5**	**11.1**	**24.9**	**25.7**	**6.7**	**15.9**

Notes:
I: Incidence rate per 100 000 population
N: Number of new cases by selected age groups
ASR: Age-standardised rate (per 100 000 population) is calculated based on the reference standard population used
CR: Crude rate per 100 000 population

Box 61

Number of liver cancer deaths and age-standardised mortality rate by gender from 2001 – 2020 (Data source: Hong Kong Cancer Registry, Hospital Authority)



Year	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Female	353	291	337	341	387	387	351	383	416	417	396	460	401	436	432	405	426	398	401	407
Male	1071	1090	1075	1076	1119	1075	1098	1116	1072	1113	1140	1045	1123	1149	1139	1135	1126	1089	1129	1123
Total	**1424**	**1381**	**1412**	**1417**	**1506**	**1462**	**1449**	**1499**	**1488**	**1530**	**1536**	**1505**	**1524**	**1585**	**1571**	**1540**	**1552**	**1487**	**1530**	**1530**

Box 62

Number of liver cancer deaths and mortality rate by age and gender from 2001 – 2020 (Data source: Hong Kong Cancer Registry, Hospital Authority)

Year	0-19						20-44						45-64						65+						Crude rate			ASR		
	Male		Female		**Total**		Male		Female		**Total**		Male		Female		**Total**		Male		Female		**Total**		Male	Female	**Total**	Male	Female	**Total**
	N	I	N	I	**N**	**I**	N	I	N	I	**N**	**I**	N	I	N	I	**N**	**I**	N	I	N	I	**N**	**I**	CR	CR	**CR**	ASR	ASR	**ASR**
2001	3	0.4	2	0.3	**5**	**0.3**	101	7.4	16	1.0	**117**	**4.0**	434	56.6	74	10.4	**508**	**34.3**	533	153.2	261	64.4	**794**	**105.4**	32.6	10.3	**21.2**	26.8	7.8	**17.2**
2002	3	0.4	1	0.1	**4**	**0.3**	98	7.3	15	1.0	**113**	**3.9**	425	53.4	51	6.7	**476**	**30.7**	564	157.3	224	53.5	**788**	**101.4**	33.2	8.4	**20.5**	26.5	5.9	**16.1**
2003	2	0.3	0	0.0	**2**	**0.1**	80	6.1	15	1.0	**95**	**3.3**	436	52.9	69	8.7	**505**	**31.2**	557	151.8	253	59.0	**810**	**101.8**	33.0	9.7	**21.0**	25.6	6.8	**15.9**
2004	2	0.3	0	0.0	**2**	**0.1**	66	5.1	15	1.0	**81**	**2.9**	428	49.9	69	8.2	**497**	**29.3**	580	153.6	257	58.2	**837**	**102.2**	32.9	9.7	**20.9**	24.7	6.6	**15.4**
2005	0	0.0	1	0.1	**1**	**0.1**	93	7.4	17	1.1	**110**	**3.9**	432	48.2	75	8.5	**507**	**28.5**	594	154.4	294	65.3	**888**	**106.4**	34.3	10.9	**22.1**	24.8	7.2	**15.8**
2006	2	0.3	0	0.0	**2**	**0.1**	49	3.9	12	0.8	**61**	**2.2**	420	45.2	64	6.9	**484**	**26.1**	604	153.6	311	67.8	**915**	**107.4**	32.9	10.8	**21.3**	23.4	6.8	**14.8**
2007	3	0.4	0	0.0	**3**	**0.2**	57	4.7	7	0.5	**64**	**2.3**	470	49.0	62	6.4	**532**	**27.6**	568	140.8	282	60.1	**850**	**97.5**	33.4	9.7	**21.0**	23.1	5.9	**14.2**
2008	1	0.1	0	0.0	**1**	**0.1**	68	5.7	17	1.1	**85**	**3.1**	480	48.3	82	8.0	**562**	**27.9**	567	138.5	284	60.0	**851**	**96.4**	33.9	10.4	**21.5**	22.9	6.3	**14.3**
2009	2	0.3	0	0.0	**2**	**0.2**	43	3.7	10	0.7	**53**	**2.0**	442	43.3	95	8.9	**537**	**25.7**	585	140.0	311	64.7	**896**	**99.7**	32.6	11.3	**21.3**	21.2	6.7	**13.7**
2010	0	0.0	0	0.0	**0**	**0.0**	35	3.0	15	1.0	**50**	**1.9**	474	45.3	89	8.0	**563**	**26.1**	604	141.2	313	63.8	**917**	**99.8**	33.8	11.2	**21.8**	21.2	6.5	**13.6**
2011	1	0.2	1	0.2	**2**	**0.2**	52	4.5	8	0.5	**60**	**2.2**	462	43.3	72	6.3	**534**	**24.1**	625	142.6	315	62.6	**940**	**99.9**	34.5	10.5	**21.7**	21.3	5.9	**13.2**
2012	0	0.0	1	0.2	**1**	**0.1**	50	4.3	10	0.7	**60**	**2.2**	431	39.9	95	8.1	**526**	**23.4**	564	123.7	354	67.6	**918**	**93.7**	31.4	12.0	**21.0**	18.9	6.5	**12.4**
2013	3	0.5	1	0.2	**4**	**0.3**	38	3.3	13	0.8	**51**	**1.9**	437	40.3	82	6.9	**519**	**22.8**	645	135.8	305	56.0	**950**	**93.1**	33.7	10.4	**21.2**	19.4	5.6	**12.1**
2014	2	0.3	0	0.0	**2**	**0.2**	48	4.2	11	0.7	**59**	**2.2**	469	43.1	71	5.9	**540**	**23.5**	629	126.8	354	62.3	**983**	**92.4**	34.4	11.2	**21.9**	19.5	5.7	**12.2**
2015	1	0.2	1	0.2	**2**	**0.2**	37	3.2	6	0.4	**43**	**1.6**	427	39.4	76	6.2	**503**	**21.8**	674	129.6	349	58.7	**1023**	**91.8**	33.8	11.0	**21.5**	18.5	5.4	**11.6**
2016	1	0.2	1	0.2	**2**	**0.2**	39	3.4	7	0.5	**46**	**1.7**	445	41.1	75	6.0	**520**	**22.3**	650	119.7	322	51.9	**972**	**83.6**	33.6	10.2	**21.0**	18.0	4.9	**11.0**
2017	3	0.5	0	0.0	**3**	**0.3**	32	2.8	8	0.5	**40**	**1.5**	409	37.6	70	5.5	**479**	**20.3**	682	120.1	348	53.8	**1030**	**84.8**	33.2	10.7	**21.0**	17.3	4.9	**10.7**
2018	0	0.0	1	0.2	**1**	**0.1**	39	3.4	11	0.7	**50**	**1.9**	351	32.1	62	4.8	**413**	**17.3**	699	117.8	324	48.0	**1023**	**80.7**	31.9	9.8	**20.0**	16.0	4.5	**9.9**
2019	0	0.0	0	0.0	**0**	**0.0**	35	3.1	3	0.2	**38**	**1.4**	386	35.2	67	5.1	**453**	**18.7**	706	113.7	331	46.8	**1037**	**78.1**	33.0	9.8	**20.4**	16.2	4.1	**9.7**
2020	0	0.0	1	0.2	**1**	**0.1**	25	2.3	2	0.1	**27**	**1.0**	363	33.3	67	5.0	**430**	**17.8**	735	113.2	337	46.0	**1,072**	**77.6**	32.9	10.0	**20.5**	15.3	4.4	**9.5**
Average	**1**	**0.2**	**<1**	**0.1**	**2**	**0.2**	**54**	**4.5**	**11**	**0.7**	**65**	**2.4**	**431**	**43.2**	**73**	**6.8**	**504**	**24.4**	**618**	**133.7**	**306**	**57.6**	**925**	**93.0**	**33.3**	**10.4**	**21.1**	**20.7**	**5.8**	**12.9**

Notes:
I: Mortality rate per 100 000 population
N: Number of death cases by selected age groups
ASR: Age-standardised rate (per 100 000 population) is calculated based on the reference standard population used
CR: Crude rate per 100 000 population

REFERENCES

1. Gust ID. The epidemiology of viral hepatitis. In: Vyas GN, Dienstag JL, Hoofnagle JH, editors. Viral Hepatitis and Liver Disease. Orlando: Grune & Stratton; 1984. p. 415-21.

2. Wong KH, Liu YM, Ng PS, et al. Epidemiology of hepatitis A and hepatitis E infection and their determinants in adult Chinese community in Hong Kong. J Med Virol 2004; 72(4):538-44.

3. Poon C, Ho B. Update on hepatitis A in Hong Kong. Communicable Diseases Watch 2015; 12(14):65-6.

4. Ho B, Wong B, Chan K. Summary on the recent upsurge of hepatitis A infection among MSM in Hong Kong. Communicable Diseases Watch 2017; 14(24):96-7.

5. Chen WC, Chiang PH, Liao YH, et al. Outbreak of hepatitis A virus infection in Taiwan, June 2015 to September 2017. Euro Surveill 2019; 24(14):1800133.

6. Ndumbi P, Freidl GS, Williams CJ, et al. Hepatitis A outbreak disproportionately affecting men who have sex with men (MSM) in the European Union and European Economic Area, June 2016 to May 2017. Euro Surveill 2018; 23(33):1700641.

7. Latash J, Dorsinville M, Del Rosso P, et al. Notes from the Field: Increase in Reported Hepatitis A Infections Among Men Who Have Sex with Men - New York City, January-August 2017. MMWR Morb Mortal Wkly Rep 2017; 66(37):999-1000.

8. Rivas V, Barrera A, Pino K, et al. Hepatitis A outbreak since November 2016 affecting men who have sex with men (MSM) in Chile connected to the current outbreak in MSM in Europe, situation up to October 2017. Euro Surveill 2018; 23(9):18-00060.

9. Liu KS, Seto WK, Lau EH, et al. A territorywide prevalence study on blood-borne and enteric viral hepatitis in Hong Kong. J Infect Dis 2019; 219(12):1924-33.

10. Chin KP, Lok AS, Wong LS, et al. Current seroepidemiology of hepatitis A in Hong Kong. J Med Virol 1991; 34(3):191-3.

11. Tsang CW, Chan CL. Epidemiology of viral hepatitis in Hong Kong. In: New trends in peptic ulcer and chronic hepatitis-Part II. Chronic Hepatitis. Tokyo: Excerpta Medica; 1987. p. 43-50.

12. Centre for Health Protection, Department of Health. Seroprevalence rates of hepatitis A virus antibodies. (Available at https://www.chp.gov.hk/en/statistics/data/10/641/701/3936.html, accessed 28 November 2022)

13. Scientific Committee on AIDS and STI and Scientific Committee on Vaccine Preventable Diseases. Statement on hepatitis A vaccination and men who have sex with men. June 2017 (Available at https://www.chp.gov.hk/files/pdf/statement_on_hav_and_msm_201706.pdf, accessed 28 November 2022)

14. Wong PS. Review of hepatitis E infection (2001-2010). Communicable Diseases Watch 2011; 8(1):1-2.

15. Kamar N, Bendall R, Legrand-Abravanel F, et al. Hepatitis E. Lancet 2012; 379(9835):2477-2488.

16. Choi D. Update of hepatitis E infection in Hong Kong. Communicable Diseases Watch 2017; 14(13):52-3.

17. Yim J. Review of hepatitis E infection in Hong Kong. Communicable Diseases Watch 2018: 15(20):85-6.

18. Sridhar S, Cheng VC, Wong SC, et al. Donor-derived genotype 4 hepatitis E virus infection, Hong Kong, China, 2018. Emerg Infect Dis 2019; 25(3):425-33.

19. Chau TN, Lai ST, Tse C, et al. Epidemiology and clinical features of sporadic hepatitis E as compared with hepatitis A. Am J Gastroenterol 2006; 101(2):292-6.

20. Lai JC, Wong GL, Yip TC, et al. Chronic hepatitis B increases liver-related mortality of patients with acute hepatitis E: a territorywide cohort study from 2000 to 2016. Clin Infect Dis 2018; 67(8):1278-84.

21. Lam WY, Chan RC, Sung JJ, et al. Genotype distribution and sequence variation of hepatitis E virus, Hong Kong. Emerg Infect Dis 2009; 15(5):792-4.

22. Leung J. Update on hepatitis E infection in Hong Kong. Communicable Diseases Watch 2012; 9(5):17-8.

23. Chow CW, Tsang SW, Tsang OT, et al. Comparison of acute hepatitis E infection outcome in patients with and without chronic hepatitis B infection: a 10 year retrospective study in three regional hospitals in Hong Kong. J Clin Virol 2014; 60(1):4-10.

24. Centre for Food Safety, Food and Environmental Hygiene Department. Hepatitis E virus in fresh pig livers. Risk Assessment Studies Report No. 44. December 2010. (Available at https://www.cfs.gov.hk/english/programme/programme_rafs/files/RA_44_HEV_pig_liver_e.pdf, accessed 28 November 2022)

25. Chan MC, Kwok K, Hung TN, et al. Molecular epidemiology and strain comparison between hepatitis E viruses in human sera and pig livers during 2014 to 2016 in Hong Kong. J Clin Microbiol 2017; 55(5):1408-15.

26. Sridhar S, Lo SK, Xing F, et al. Clinical characteristics and molecular epidemiology of hepatitis E in Shenzhen, China: a shift toward foodborne transmission of hepatitis E virus infection. Emerg Microbes Infect 2017; 6(12): e115.

27. Chiu DM, Chan MC, Yeung AC, et al. Seroprevalence of hepatitis E virus in Hong Kong, 2008-2009. J Med Virol 2013; 85(3):459-61.

28. Chan DP, Lee KC, Lee SS. Epidemiology of hepatitis E infection in Hong Kong. Hong Kong Med J 2017; 23 Suppl 5(4):31-5.

29. Tsoi WC, Zhu X, To AP, et al. Hepatitis E virus infection in Hong Kong blood donors. Vox Sang 2020; 115(1):11-7.

30. Sridhar S, Chew NF, Situ J, et al. Risk of hepatitis E among persons who inject drugs in Hong Kong: a qualitative and quantitative serological analysis. Microorganisms 2020; 8(5):675.

31. Sridhar S, Yip CC, Wu S, et al. Rat hepatitis E virus as cause of persistent hepatitis after liver transplant. Emerg Infect Dis 2018; 24(12):2241-50.

32. Leung YH. Human infection of rat hepatitis E virus (HEV). Communicable Diseases Watch 2018; 15(23):100-101.

33. Sridhar S, Yip CC, We S, et al. Transmission of rat hepatitis E virus infection to humans in Hong Kong: a clinical and epidemiological analysis. Hepatology 2021; 73(1):10-22.

34. Sridhar S, Yip CC, Lo KH, et al. Hepatitis E virus species C infection in humans, Hong Kong. Clin Infect Dis 2022; 75(2):288-296.

35. Wong NS, Lee SS, Poon CM, et al. Awareness of hepatitis B virus infection status in the general population in Hong Kong. Int J Infect Dis 2020; 101 Suppl 1:471.

36. Kwan LC, Ho YY, Lee SS. The declining HBsAg carriage rate in pregnant women in Hong Kong. Epidemiol Infect 1997; 119(2):281-3.

37. Lao TT, Sahota DS, Law LW, et al. Age-specific prevalence of hepatitis B virus infection in young pregnant women, Hong Kong Special Administrative Region of China. Bull World Health Organ 2014; 92(11):782-9.

38. Cooley L, Sasadeusz J. Clinical and virological aspects of hepatitis B co-infection in individuals infected with human immunodeficiency virus type-1. J Clin Virol 2003; 26(2):185-93.

39. Tam YH. Hong Kong achieves goal of hepatitis B control verified by the World Health Organization Western Pacific Region. Communicable Diseases Watch 2011; 8(15):62-3.

40. Yuen MF, Sablon E, Tanaka Y, et al. Epidemiological study of hepatitis B virus genotypes, core promoter and precore mutations of chronic hepatitis B infection in Hong Kong. J Hepatol 2004; 41(1):119-25.

41. Chan HL, Hui AY, Wong ML, et al. Genotype C hepatitis B virus infection is associated with an increased risk of hepatocellular carcinoma. Gut 2004; 53(10):1494-8.

42. Chan HL, Tsui SK, Tse CH, et al. Epidemiological and virological characteristics of 2 subgroups of hepatitis B virus genotype C. J Infect Dis 2005; 191(12):2022-32.

43. Zhu L, Tse CH, Wong VW, et al. A complete genomic analysis of hepatitis B virus genotypes and mutations in HBeAg-negative chronic hepatitis B in China. J Viral Hepatol 2008; 15(6):449-58.

44. Chan HL, Tse CH, Mo F, et al. High viral load and hepatitis B virus subgenotype Ce are associated with increased risk of hepatocellular carcinoma. J Clin Oncol 2008; 26(2):177-82.

45. Chan HL, Wong GL, Tse CH, et al. Hepatitis B virus genotype C is associated with more severe liver fibrosis than genotype B. Clin Gastroenterol Hepatol 2009; 7(12):1361-6.

46. Wong GL, Chan HL, Yiu KK, et al. Meta-analysis: The association of hepatitis B virus genotypes and hepatocellular carcinoma. Aliment Pharmacol Ther 2013; 37(5):517-26.

47. Lo CM, Cheung CK, Lau GK, et al. Significance of hepatitis B virus genotype in liver transplantation for chronic hepatitis B. Am J Transplant 2005; 5(8):1893-900.

48. Yuen MF, Tanaka Y, Mizokami M, et al. Role of hepatitis B virus genotypes Ba and C, core promoter and precore mutations on hepatocellular carcinoma: a case control study. Carcinogenesis 2004; 25(9):1593-8.

49. Chan AO, Yuen MF, Lam CM, et al. Prevalence and characteristics of familial hepatocellular carcinoma caused by chronic hepatitis B infection in Hong Kong. Aliment Pharmacol Ther 2004; 19(4):401-6.

50. Lok AS, Wong A, Sporton S, et al. Hepatitis D virus superinfection remains a rare occurrence in non-drug abusers in Hong Kong. J Hepatol 1992; 14(2-3):332-4.

51. Ho CF, Wong KH, Chan CW, et al. Current pattern and course of acute hepatitis B virus infection in Hong Kong. J Gastroenterol Hepatol 2004; 19(5):602-3.

52. Young BW, Lee SS, Lim WL, et al. The long-term efficacy of plasma-derived hepatitis B vaccine in babies born to carrier mothers. J Viral Hepatol 2003; 10(1):23-30.

53. Lin AW, Wong KH. Long-term protection of neonatal hepatitis B vaccination in a 30-year cohort in Hong Kong. J Hepatol 2013: 59(6):1363-4.

54. But DY, Lai CL, Lim WL, et al. Twenty-two years follow-up of a prospective randomized trial of hepatitis B vaccines without booster dose in children: final report. Vaccine 2008; 26(51):6587-91.

55. Tse W, Mok T. Survey on immunization coverage among children aged two to five. Public Health & Epidemiology Bulletin 2002; 11(2):13-8.

56. Tse WK, Yeung SW. Immunization coverage among children aged two to five: an update. Public Health & Epidemiology Bulletin 2004; 13(1):7-15.

57. Wu T, Chan SK, Kung KH, et al. Immunization coverage among children aged two to five: findings of the 2006 survey. Public Health & Epidemiology Bulletin 2007: 16(4):57-67.

58. Chan D, Chan SK, Wong SC, et al. Immunisation coverage among children aged two to five: findings of the 2009 Immunisation Survey. Public Health & Epidemiology Bulletin 2010; 19(3):53-63.

59. Chan D. Immunisation coverage for children aged two to five: findings of the 2012 Immunisation Survey. Communicable Diseases Watch 2014; 11(13):55-7.

60. Chan D. Immunisation coverage for children aged two to five: findings of the 2015 Immunisation Survey. Communicable Diseases Watch 2017; 14(6):23-6.

61. Chan D. Immunisation coverage of vaccines under the Hong Kong Childhood Immunisation Programme - findings of the 2018 Immunisation Survey on Preschool Children. Communicable Diseases Watch 2019; 16(13):62-4.

62. Tam YH. Hong Kong achieves goal of hepatitis B control verified by the World Health Organization Western Pacific Region. Communicable Diseases Watch 2011; 8(15):62-3.

63. Chung PW, Suen SH, Chan OK, et al. Awareness and knowledge of hepatitis B infection and prevention and the use of hepatitis B vaccination in the Hong Kong adult Chinese population. Chin Med J (Engl) 2012; 125(3):422-7.

64. Lin A, Tam YH. Hepatitis C in Hong Kong, 2008 to 2011. Communicable Diseases Watch 2011; 8(25):103-4.

65. Hagan H, Jordan AE, Neurer J, et al., Incidence of sexually transmitted hepatitis C virus infection in HIV-positive men who have sex with men. AIDS 2015; 29(17):2335-45.

66. Chan GC, Lim W, Yeoh EK. Prevalence of hepatitis C infection in Hong Kong. J Gastroenterol Hepatol 1992; 7(2):117-20.

67. Wong HK, Lee CK, Leung JN, et al. Risk factor analysis of hepatitis C virus infection among Chinese blood donors in Hong Kong. Transfus Med 2012; 22(2):133-6.

68. Wong NS, Lee CK, Ng SC, et al. Prevalence of hepatitis C infection and its associated factors in healthy adults without identifiable route of transmission. J Viral Hepat 2018; 25(2):161-70.

69. Chan TM, Lok AS, Cheng IK, et al. Prevalence of hepatitis C virus infection in hemodialysis patients: a longitudinal study comparing the results of RNA and antibody assays. Hepatology 1993; 17(1):5-8.

70. Au WY, Lee V, Kho B, et al. A synopsis of current haemophilia care in Hong Kong. Hong Kong Med J 2011; 17(3):189-94.

71. Lee KC, Lim WW, Lee SS. High prevalence of HCV in a cohort of injectors on methadone substitution treatment. J Clin Virol 2008; 41(4):297-300.

72. Wong NS, Chan PC, Lee SS, et al. A multilevel approach for assessing the variability of hepatitis C prevalence in injection drug users by their gathering places. Int J Infect Dis 2013; 17(3):e193-8.

73. Chan DP, Lee KC, Lee SS, et al. Community-based molecular epidemiology study of hepatitis C virus infection in injection drug users. Hong Kong Med J 2017; 23 Suppl 5(4):27-30.

74. Wong NS, Chan DP, Chan CP, et al. Point-of-care hepatitis C reflex testing and treatment referral in methadone clinic settings in Hong Kong-a pilot study. IJID Reg 2022; 5:8-12.

75. Wong GL, Chan HL, Loo CK, et al. Change in treatment paradigm in people who previously injected drugs with chronic hepatitis C in the era of direct-acting antiviral therapy. J Gastroenterol Hepatol 2019; 34(9):1641-7.

76. Centers for Disease Control and Prevention (CDC). Sexual transmission of hepatitis C virus among HIV-infected men who have sex with men -- New York City, 2005--2010. MMWR Morb Mortal Wkly Rep 2011; 60(28):945-50.

77. Lin A, Wong P, Lo J. A case series of hepatitis C infection and syphilis among HIV positive men who have sex with men. Communicable Disease Watch 2014; 11(5):18-9.

78. Lin AW, Wong KH, Chan K. More safer sex intervention needed for HIV-positive MSM with higher education level for prevention of sexually transmitted hepatitis C. J Int AIDS Soc 2014;17(4 Suppl 3):19663.

79. Kwan TH, Wong BC, Wong KH, et al. Hepatitis C co-infection in people living with HIV – epidemiologic differences between men who have sex with men MSM and non-MSM. Front Public Health 2022; 10:925600.

80. Monga HK, Rodriguez-Barradas MC, Breaux K, et al. Hepatitis C virus infection-related morbidity and mortality among patients with human immunodeficiency virus infection. Clin Infect Dis 2001; 33(2):240-7.

81. Gower E, Estes C, Blach S, et al. Global epidemiology and genotype distribution of the hepatitis C virus infection. J Hepatol 2014; 61(1 Suppl):S45-57.

82. Polaris Observatory HCV Collaborators. Global prevalence and genotype distribution of hepatitis C virus infection in 2015: a modelling study. Lancet Gastroenterol Hepatol 2017; 2(3):161-176.

83. Prescott LE, Simmonds P, Lai CL, et al. Detection and clinical features of hepatitis C virus type 6 infections in blood donors from Hong Kong. J Med Virol 1996; 50(2):168-75.

84. Wong DA, Tong LK, Lim W. High prevalence of hepatitis C virus genotype 6 among certain risk groups in Hong Kong. Eur J Epidemiol 1998; 14(5):421-6.

85. Chan TM, Lau JY, Wu PC, et al. Hepatitis C virus genotypes in patients on renal replacement therapy. Nephrol Dial Transplant 1998; 13(3):731-4.

86. Hui YT, Wong GL, Fung JY, et al. Territory-wide population-based study of chronic hepatitis C infection and implications for hepatitis eliminiation in Hong Kong. Liver Int 2018; 38(11):1911-9.

87. Zhou DX, Tang JW, Chu IM, et al. Hepatitis C virus genotype distribution among intravenous drug user and the general population in Hong Kong. J Med Virol 2006; 78(5):574-81.

88. Zhou X, Chan PK, Tam JS, et al. A possible geographic origin of endemic hepatitis C virus 6a in Hong Kong: evidences for the association with Vietnamese immigration. PLoS One 2011: 6(9):e24889.

89. Chan DP, Lee SS, Lee KC. The effects of widespread methadone treatment on the molecular epidemiology of hepatitis C virus infection among injecting drug users in Hong Kong. J Med Virol 2011; 83(7):1187-94.

90. Chan DP, Lin AW, Wong KH, et al. Diverse origins of hepatitis C virus in HIV co-infected men who have sex with men in Hong Kong. Virol J 2015; 12:120.

91. Sun HY, Uemura H, Wong NS, et al. Molecular epidemiology of acute HCV infection in HIV-positive patients from Hong Kong, Taipei, Tokyo. Liver Int 2019; 39(6):1044-51.

92. Seto WK, Lai CL, Fung J, et al. Natural history of chronic hepatitis C: genotype 1 versus genotype 6. J Hepatol 2010; 53(3):444-8.

93. Sung H, Ferlay J, Siegel RL, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71(3):209-49.

94. Perz JF, Armstrong GL, Farrington LA, et al. The contributions of hepatitis B virus and hepatitis C virus infections to cirrhosis and primary liver cancer worldwide. J Hepatol 2006; 45(4):529-38.

95. Yuen MF, Hou JL, Chutaputti A, et al. Hepatocellular carcinoma in the Asia Pacific Region. J Gastroenterol Hepatol 2009; 24(3):346-53.

96. Lo CM, Fan ST, Liu CL, et al. Ten-year experience with liver transplantation at Queen Mary Hospital: retrospective study. Hong Kong Med J 2002; 8(4):240-4.

97. Fung J, Mak LY, Chan A, et al. Trends in liver transplantation for chronic hepatitis B in an era of highly potent antiviral therapies. Liver Transpl 2021; 27(1):134-139.

98. Hong Kong Cancer Registry, Hospital Authority. Liver Cancer in 2020. (available at https://www3.ha.org.hk/cancereg/pdf/factsheet/2020/liver_2020.pdf, accessed on 11 November 2022)

99. Lim WL, Yeoh EK. Hepatitis A vaccination. Lancet 1992; 339(8788):304.

100. Lai CL. Hepatitis A risk heightened. Data quoted in United Daily News dated 10 June 1994.

101. Lee A, Cheng F, Lau L, et al. Changing hepatitis A epidemiology among Hong Kong Chinese adolescents: what are the implications? Public Health 1999; 113(4):185-8.