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**National Disease Surveillance Centre,**

25-27 Middle Gardiner St
Dublin 1,
Ireland

Tel: +353 (0)1 **876 5300**
Fax: +353 (0)1 **856 1299**
info@ndsc.ie
www.ndsc.ie



# SARS Update

On 5th July 2003, the World Health Organisation (WHO) removed Taiwan from the list of areas with recent local transmission of severe acute respiratory syndrome (SARS). Taiwan was the last area to be removed indicating that the human chains of SARS virus transmission appear to have been broken worldwide. The chain of transmission is considered to be broken if no further cases have occurred within 20 days of the last probable case. However, global vigilance will continue as much remains unknown about SARS. Based on what is known about other coronaviruses it may be a seasonal disease and could return later in the year. The original source of the outbreak could still be in an animal or environmental reservoir or there may be cases undetected in countries with poor surveillance systems.

SARS is now known to have emerged in the Guangdong Province of China in mid-November 2002. From there it spread via an infected medical doctor from Guangdong to guests and visitors to a hotel in Hong Kong. They seeded outbreaks of cases in the hospital system in Hong Kong, Vietnam, Singapore and Toronto, and around the world via international air travel as visitors/guests of the hotel flew home or travelled for other reasons.

WHO issued a global alert on 12th March 2003. Areas with cases prior to this alert experienced the most devastating outbreaks. Health care staff took no precautions to protect themselves and these were the people who were initially most affected by SARS. They in turn spread it to the wider community. With the exception of Taiwan, all other areas experiencing imported cases after the alert took precautions and were able to prevent further transmission or reduce transmission to small numbers.

## Impact

SARS is a severe and readily transmissible disease. Symptoms are non-specific and common. The incubation period is up to 10 days. The overall fatality rate is 15% and can exceed 50% in persons over 65 years of age. It is caused by a new coronavirus unlike any other known human or animal virus in its family. There is no effective treatment and no vaccine against the disease.

The volume of international air travel allowed spread around the world with unprecedented speed. The close interdependence of economies and markets amplified the economic impact. The economic costs to countries around the world are still being calculated but the initial estimates for the Far East alone are US$ 30 billion. Instant electronic communication raised public concern but also allowed the establishment of networks of researchers, epidemiologists and clinicians to identify the causative agent, develop a case definition and investigate modes of transmission in record time. There were significant strains on the healthcare systems in the countries most affected by SARS.

The outbreak period continued from 1st November 2002 to 5th July 2003. A total of 8,437 probable cases and 813 deaths were reported from 29 countries around the world. In Ireland, there were several suspect cases. However, only one of these was subsequently diagnosed as a probable case. A review of cases is ongoing and the final outcome of probable cases is still unfolding.

## Control Measures

The control of SARS is the result of efforts by governments and health care staff supported by a well-informed and co-operative public. It involved the identification of cases, and their appropriate management including isolation, infection control, and contact tracing. Other measures included education and information to the public, travel alerts and restrictions.

Vietnam shows that immediate political commitment at the highest level can be decisive. It was the first country to contain the outbreak on 28th April 2003. The government of Vietnam went public straight away, alerted the health care system and border controls and set up a national task force with committees at local level to deal with the outbreak. They invited assistance from WHO very early on and created excellent coordination between government, the taskforce and WHO.

## Future Measures

To prepare for the next outbreak we need to strengthen the public health infrastructure. More epidemiologists and other public health specialists are needed along with surveillance systems with strong national, regional and global linkages. More investment in hospital infection control is required. There is also a need to develop the surge capacity of hospitals and public health systems. Priorities for research are the development of a rapid reliable diagnostic test, understanding modes of transmission and developing effective treatments.

Dr Brundtland, Director-General of WHO said “SARS is a warning, it pushed even the most advanced public health system to the breaking point. Those protections held, but just barely. Next time we may not be so lucky. We have an opportunity now, and we see the need clearly, to rebuild our public health protections. They will be needed for the next global outbreak, if it is SARS or another new infection.”

**The above information and further information on SARS is available on the WHO website at www.who.int/**

# Epidemiology of Hepatitis B Infection in Ireland

## Introduction

Hepatitis B infection is one of the most common causes of serious liver disease in the world. Acute infection is often asymptomatic (less than 10% of children and 30-50% of adults develop symptoms) and may lead to chronic infection. The risk of developing chronic infection varies inversely with age, occurring in 90% of infants infected at birth, 20-50% of children infected at 1-5 years of age and 1-10% of persons infected as older children or adults. Chronic infection leads to death from cirrhosis or hepatocellular carcinoma (HCC) in 15-25% of cases. Hepatitis B may be the cause of up to 80% of all HCC worldwide, and is second only to tobacco among known human carcinogens.[1] Hepatitis B infection is a vaccine preventable disease.

The major routes of hepatitis B virus transmission are through sexual or household contact with an infected person, perinatal transmission from mother to infant, injecting drug use and nosocomial exposure.

Ireland is considered a low prevalence country as the prevalence of hepatitis B surface antigen (HBsAg), a serological marker of chronic carriage, is below 2% in the general population.[2,3] Therefore vaccination is only recommended for individuals who are at increased risk of infection because of their occupation, lifestyle or other factors.[2]

## Methods

Data from various sources were used to describe the epidemiology of hepatitis B in Ireland.

- The National Disease Surveillance Centre (NDSC) collates data on all notifiable diseases including hepatitis B. Currently no case definitions exist for any of the notifiable diseases and therefore there is no requirement in the notification process to distinguish between acute and chronic cases of hepatitis B. While NDSC has aggregate data on hepatitis B from 1982, disaggregate data (including information on age and sex) has only been collected since mid-2000 when NDSC took over responsibility for the collation and analysis of the weekly notifications of infectious diseases. Data for 2002 are provisional.
- Data on hospital discharges containing a principal diagnosis of hepatitis B or any diagnosis of hepatitis B (principal and up to 5 secondary diagnoses) by sex and age group were obtained from the HIPE (Hospital In-Patient Enquiry) Unit of the Economic and Social Research Institute for the years 1999-2001.
- Data on the total number of liver transplants carried out in Ireland from 1997 to 2002, and the number with an aetiology of viral hepatitis were obtained from the National Liver Transplant Unit, St Vincent's University Hospital, Dublin.
- Data on the number of new cases of HCC by sex and age group were obtained from the National Cancer Registry of Ireland (NCRI) for the years 1994-1999.
- The Central Statistics Office (CSO) provided data on deaths from hepatitis B and primary liver cancer by sex for the years 1990-2002.

All rates were calculated using denominator data from the closest census year. For 1999, the 1996 census data were used.

## Results

***Incidence***

Since 1997, the number of notifications has increased nearly 15-fold. The data from 1988 to 2002 are illustrated in figure 1. The incidence rates increased from 8.7/100,000 in 2001 to 11.6/100,000 in 2002. The age standardised rates varied between health boards with the Southern Health Board (SHB) having the highest rate in 2002, followed by the Midland Health Board (MHB) (table 1).

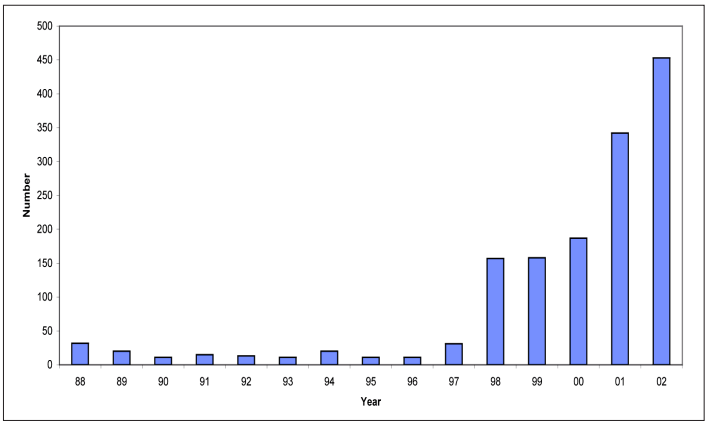



***Figure 1****. Number of hepatitis B cases notified 1988-2002*

***Table 1.*** *Number and age standardised incidence rate of hepatitis B notifications by health board, 2002.*

| Health board | Number of cases 2002 | Age standardised rate 2002 |
|---|---|---|
| **ERHA** | 127 | 7.9 |
| **MHB** | 53 | 24.7 |
| **MWHB** | 24 | 7.4 |
| **NEHB** | 15 | 4.5 |
| **NWHB** | 5 | 2.5 |
| **SEHB** | 63 | 15.3 |
| **SHB** | 164 | 29.1 |
| **WHB** | 2 | 0.6 |
| **Total** | **453** | **11.6** |

In 2001, there was a slight excess of male cases (54%) reported.[4] However, cases were nearly evenly distributed in 2002 (48% male).

The highest rates of hepatitis B notifications in both sexes occurred between the ages of 25 and 34 years (figure 2).

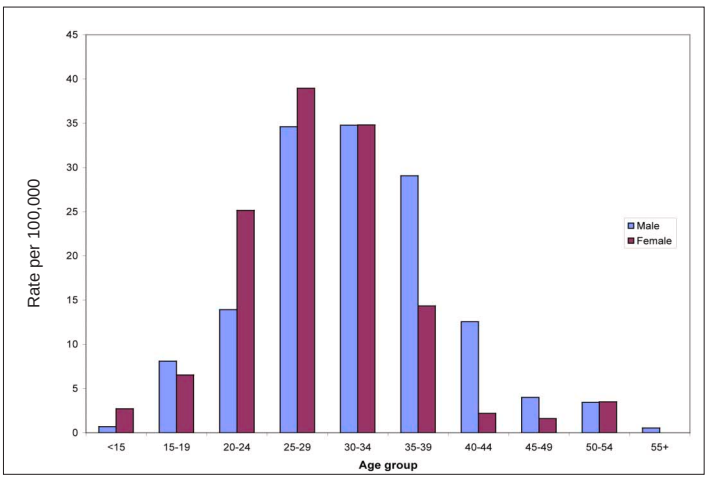



***Figure 2.*** *Age- and sex-specific rates of hepatitis B per 100,000 population, 2002.*

## Morbidity and Mortality Data

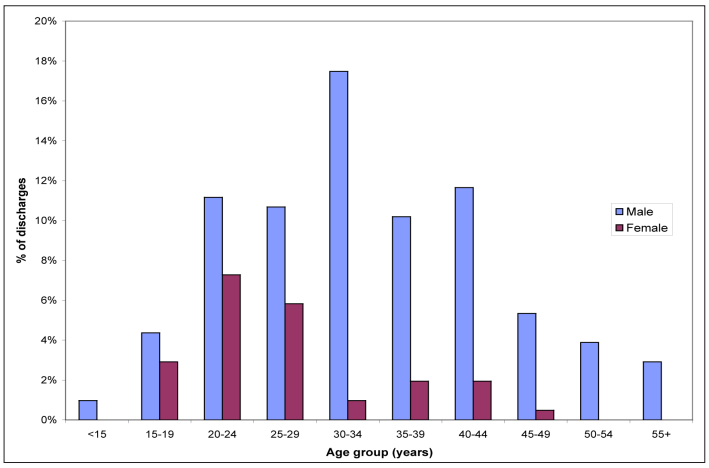
The number of hospital discharges which contained hepatitis B, as either the principal diagnosis or recorded anywhere in the diagnoses, between 1999 and 2001 can be seen in table 2.

Seventy-nine percent of the discharges containing a principal diagnosis of hepatitis B were male. The highest number of male cases occurred in the 30-34 year age group, with 78% between 20 and 44 years of age. The female cases tended to be younger, peaking in the 20-24 year age group, the majority of cases (61%) being between 20 and 29 years of age (figure 3).

***Table 2.*** *Total number of discharges containing a diagnosis of hepatitis B (1999-2001) and the average annual rates per 1000 discharges and per 100,000 population.*

| Hepatitis B diagnosis | Number of discharges (1999-2001) | Average rate /1000 discharges | Average rate /100,000 population |
|---|---|---|---|
| **Acute or unspecified** | | | |
| All diagnoses | 771 | 0.32 | 6.73 |
| Principal diagnosis | 125 | 0.05 | 1.09 |
| **Chronic** | | | |
| All diagnoses | 426 | 0.17 | 3.72 |
| Principal diagnosis | 81 | 0.03 | 0.71 |

Source: HIPE unit, ESRI




Source: HIPE unit, ESRI

***Figure 3.*** *Age and sex distribution of all hospital discharges with a principal diagnosis of hepatitis B, 1999-2001.*

There were 191 adult liver transplants carried out in Ireland between 1997 and 2002. Hepatitis B infection accounted for 3.1% of these.

The NCRI recorded 147 cases of HCC between 1994 and 1999 (table 3), 79% of which were in males.

***Table 3.*** *Number of new cases of hepatocellular carcinoma registered and rate per 100,000 population, 1994-1999*

| Year | Number of cases | Rate /100,000 population |
|---|---|---|
| 1994 | 32 | 0.88 |
| 1995 | 25 | 0.69 |
| 1996 | 27 | 0.75 |
| 1997 | 19 | 0.52 |
| 1998 | 26 | 0.72 |
| 1999 | 18 | 0.50 |

Source: NCRI

Hepatitis B was recorded as the primary cause of death in 25 people between 1990 and 2002, all but five being male.

HCC is the most common primary liver cancer. Between 1990 and 2002, there were 248 deaths from primary liver cancer , 70% being male.

## Discussion

Using a combination of different data sources it is possible to get a broad, although incomplete, picture of the current burden of disease due to hepatitis B infection in Ireland. The epidemiology of hepatitis B has changed in recent years. From 1981 (when it was specified as a notifiable disease) until 1997 there were less than 2 cases per 100,000 population notified each year. Published data from the National Virus Reference Laboratory (NVRL) from 1970-1987 showed that the most common risk factor identified was injecting drug use, the male to female ratio was 25:1, and only 8.3% of cases were chronically infected.[5] Since 1997, the number of hepatitis B notifications has increased dramatically, with over 11 cases per 100,000 population in 2002. Many of the cases being reported now are chronically infected asylum seekers[6] and there are nearly as many female cases as there are male.

The hospitalised cases with a principal diagnosis of hepatitis B did not show the same sex distribution as notified cases, although the age distribution was similar. The numbers of cases with any diagnosis or a principal diagnosis of chronic hepatitis B both increased steadily over the three years, while the number of discharges with a principal diagnosis of acute hepatitis B decreased over the same time. However, data would be needed over a longer period of time in order to study these trends.

Although hepatitis B is a notifiable disease, under-reporting is common as demonstrated in 2000 by the discrepancy between the number of HBsAg positive samples detected by the NVRL (n = 470) and the substantially smaller number of hepatitis B cases reported to NDSC (n = 187).[4] Information currently reported on individual cases is inadequate. More detailed information, e.g. risk factor details, is required to monitor and inform prevention and control strategies and to plan services.

Laboratory data on hepatitis B are not routinely available. At present, laboratories are not obliged to notify notifiable diseases. In the case of a disease such as hepatitis B, whose definitive diagnosis requires laboratory confirmation, laboratory notification is essential to accurately estimate the incidence and prevalence of disease. A review of notifiable diseases and the process of notification carried out by NDSC at the request of the Department of Health and Children has recommended that laboratories should be specified as notifiers. Case definitions to cover both acute and chronic disease have also been proposed. If these recommendations are adopted and implemented, and the process of reporting facilitated by the Computerised Infectious Disease Reporting (CIDR) system which is currently under development, the result will be greatly improved information on the epidemiology of hepatitis B in Ireland.

**Aline Brennan and Lelia Thornton, NDSC**

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# INVASIVE GROUP A STREPTOCOCCAL INFECTION

There is recent concern that there has been an increase in the incidence of invasive group A streptococci (GAS) infections in Ireland. This concern is based on anecdotal reports from local centres of a perceived increase in invasive GAS infections in clinical practice. However, formal surveillance is not yet performed routinely, impacting on the ability to confirm this trend objectively.

In 2001, the incidence of invasive GAS disease was 3.5/100,000 population in the United States, 5.9% of which were streptococcal toxic shock syndrome (STSS) and 6.8% necrotising fasciitis. Death occurred in 10-13% of all invasive cases, 45% of STSS and 25% of necrotising fasciitis.[1] Worldwide rates of invasive disease increased from the 1980s to the 1990s but have been relatively stable over the past five years. The increase was associated with an increased prevalence of serotypes M-1 and M-3.[2] A European GAS surveillance programme (Strep-EURO) was launched on 1 September 2002 to develop a pan-European epidemiological perspective on severe GAS disease in Europe and to study pathogenic mechanisms, determine antibiotic susceptibility and apply molecular methods for typing and clonal identification of clinical isolates.[3] Plans for national surveillance for GAS in Ireland are at an advanced stage and a case definition for GAS has been included in the proposed changes to Infectious Disease legislation.

In 1933, Lancefield classified Beta-haemolytic streptococci into serogroups based on M-protein precipitin reactions and found that most strains pathogenic for humans belonged to serogroup A (*S. pyogenes*). The M-protein, a cell membrane protein has been identified as a major virulence factor. GAS may be further subdivided on the basis of antigenic differences in the M-protein molecule and on the basis of nucleotide differences in the *emm* gene, which encodes the molecule.[4] More than 90 serotypes of GAS have been identified.

Importantly GAS elaborates a number of pyrogenic exotoxins which have a role in its pathogenicity. They are responsible for the rash in scarlet fever and play a role in STSS.

GAS causes a range of diseases in humans including pharyngitis, scarlet fever, soft tissue infections, rheumatic fever and poststreptococcal glomerulonephritis. Invasive GAS infection, including necrotising fasciitis, bacteraemia and infection at normally sterile sites carries a significant mortality, especially when complicated by STSS.

STSS is characterised by isolation of GAS from a normally sterile site associated with hypotension and two of the following: renal impairment, coagulopathy, liver dysfunction, Adult Respiratory Distress Syndrome, rash, soft tissue necrosis.[5] STSS has been reported in persons of all ages both in those with predisposing medical conditions and the immunocompromised but also in the immunocompetent with no predisposing factors. Portals of entry for invasive GAS include the pharynx, skin and vagina in 50% of cases and can be related to surgical procedures. Infection may rarely occur secondary to streptococcal pharyngitis, and viral infections such as influenza and varicella have provided portals of entry. Factors predisposing to invasive GAS and STSS include young or old age, diabetes, HIV and immunosuppression, alcohol abuse, intravenous drug use, surgical procedures, trauma, viral infection, contact with an infected patient, non-steroidal anti-inflammatory medication and a high prevalence of GAS in the community. Nosocomial spread has been described. Serotypes M-1 and M-3 are particularly associated with outbreaks of invasive infection and hence it is important that isolates from invasive infection are serotyped.

The priority in the management of suspected deep-seated GAS infection is rapid aggressive surgical debridement. Patients may need fluid and pressor therapy for shock. Initial broad-spectrum antibiotic therapy should be switched to high-dose penicillin (in the non-penicillin allergic patient) and clindamycin as soon as GAS is confirmed. Clindamycin has the additional effect of suppressing M-protein and exotoxin production by GAS. Penicillin alone can be ineffective in severe deep infections with large amounts of bacteria, as penicillin-binding proteins are not expressed in the stationary-phase growth of GAS. Intravenous immunoglobulin has been used successfully in the treatment of STSS and in one comparative observational study the mortality was halved (from 67% mortality in the control group to 34% in the group that received IVIG).[6] It is believed that the IVIG neutralises circulating GAS exotoxins. It should be given early and in more than one dose.

In view of the high morbidity and mortality associated with invasive GAS infection it is a cause for concern that there has been an increase in the perceived incidence of infection. National surveillance data would help clarify these suspicions and provide a better understanding of the pattern of this serious infection.

**Drs Peter Coakley and Colm Bergin, St. James's Hospital**

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## Salmonella Monthly Report (June 2003):

Strains are allocated to months based on the date of receipt of the isolate from the referring laboratory. These figures are provisional as work may not be finished on particular strains at the time of publication. Data are provided courtesy of Prof Martin Cormican and Dr Geraldine Corbett-Feeney, INSRL.

| Health Board | E | M | MW | NE | NW | SE | S | W | Total |
|---|---|---|---|---|---|---|---|---|---|
| S.Agona | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| S.Braenderup | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| S.Dublin | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | **2** |
| S.Enteritidis | 4 | 1 | 2 | 0 | 0 | 4 | 5 | 3 | **19** |
| S.Hadar | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | **2** |
| S.Javiana | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| S.Newport | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | **1** |
| S.Saintpaul | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | **1** |
| S.Typhi | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | **2** |
| S.Typhimurium | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 2 | **7** |
| S.Virchow | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| **Total** | **9** | **3** | **6** | **1** | **1** | **6** | **5** | **7** | **38** |