Abstracts and references

Selected by Tom Kiedrzynski

The aetiology, origins, and diagnosis of severe acute respiratory syndrome.

Poon LL, Guan Y, Nicholls JM, Yuen KY, Peiris JS.
Lancet Infect Dis. 2004 Nov;4(11):663-71.

Severe acute respiratory syndrome (SARS) is a new infectious disease that first emerged in Guangdong province, China, in November, 2002. A novel coronavirus was later identified in patients with SARS. The detection of the virus in these patients, its absence in healthy controls or other patients with atypical pneumonia, and the reproduction of a similar disease in a relevant animal model fulfilled Koch's postulates for implicating this coronavirus as the causal agent of SARS. The full genome sequence was determined within weeks of the virus's identification. The rapid progress in the aetiology, the development of laboratory diagnostic tests, and the defining of routes of viral transmission were facilitated through a unique WHO-coordinated virtual network of laboratories, which shared information on a real-time basis through daily teleconferences. Subsequent studies have indicated that the SARS coronavirus is of animal origin, that its precursor is still present in animal populations within the region, and that live-animal markets in southern China may have provided the animal-human interphase that allowed this precursor virus to adapt to human-human transmission. These findings underscore the potential for the re-emergence of SARS and the need for laboratory tests for early diagnosis. However, the low viral load in the respiratory tract makes early diagnosis of SARS a diagnostic challenge, although improvements in the sensitivity of molecular diagnostic methods continue to be made.

Subsequent studies have indicated that the SARS coronavirus is of animal origin, that its precursor is still present in animal populations within the region

Bats are natural reservoirs of SARS-like coronaviruses.

Li W, Shi Z, Yu M, Ren W, Smith C, Epstein JH, Wang H, Crameri G, Hu Z, Zhang H, Zhang J, McEachern J, Field H, Daszak P, Eaton BT, Zhang S, Wang LF.
Science. 2005 Oct 28;310(5748):676-9.

Severe acute respiratory syndrome (SARS) emerged in 2002 to 2003 in southern China. The origin of its etiological agent, the SARS coronavirus (SARS-CoV), remains elusive. Here we report that species of bats are a natural host of coronaviruses closely related to those responsible for the SARS outbreak. These viruses, termed SARS-like coronaviruses (SL-CoVs), display greater genetic variation than SARS-CoV isolated from humans or from civets. The human and civet isolates of SARS-CoV nestle phylogenetically within the spectrum of SL-CoVs, indicating that the virus responsible for the SARS outbreak was a member of this coronavirus group.

Severe acute respiratory syndrome coronavirus-like virus in Chinese horseshoe bats.

Lau SK, Woo PC, Li KS, Huang Y, Tsoi HW, Wong BH, Wong SS, Leung SY, Chan KH, Yuen KY.
Proc Natl Acad Sci U S A. 2005 Sep 27;102(39):14040-5.

Although the finding of severe acute respiratory syndrome coronavirus (SARS-CoV) in caged palm civets from live animal markets in China has provided evidence for interspecies transmission in the genesis of the SARS epidemic, subsequent studies suggested that the civet may have served only as an amplification host for SARS-CoV. In a surveillance study for CoV in noncaged animals from the wild areas of the Hong Kong Special Administration Region, we identified a CoV closely related to SARS-CoV (bat-SARS-CoV) from 23 (39%) of 59 anal swabs of wild Chinese horseshoe bats (Rhinolophus sinicus) by using RT-PCR. Sequencing and analysis of three bat-SARS-CoV genomes from samples collected at different dates showed that bat-SARS-CoV is closely related to SARS-CoV from humans and civets. Phylogenetic analysis showed that bat-SARS-CoV formed a distinct cluster with SARS-CoV as group 2b CoV, distantly related to known group 2 CoV. Most differences between the bat-SARS-CoV and SARS-CoV genomes were observed in the spike genes, ORF 3 and ORF 8, which are the regions where most variations also were observed between human and civet SARS-CoV genomes. In addition, the presence of a 29-bp insertion in ORF 8 of bat-SARS-CoV genome, not in most human SARS-CoV genomes, suggests that it has a common ancestor with civet SARS-CoV. Antibody against recombinant bat-SARS-CoV nucleocapsid protein was detected in 84% of Chinese horseshoe bats by using an enzyme immunoassay. Neutralizing antibody to human SARS-CoV also was detected in bats with lower viral loads. Precautions should be exercised in the handling of these animals.

Nipah virus: impact, origins, and causes of emergence.

Epstein JH, Field HE, Luby S, Pulliam JR, Daszak P.
Curr Infect Dis Rep. 2006 Jan;8(1):59-65.

Nipah virus is an emerging zoonotic pathogen that causes severe febrile encephalitis resulting in death in 40% to 75% of human cases. Nipah virus is considered a biosafety level-4 pathogen and is listed as a select agent with high risk for public health and security due to its high mortality rate in people and the lack of effective vaccines or therapies. The natural reservoir for Nipah virus and related members of the genus Henipavirus are fruit bats of the genus Pteropus. Nipah virus emerged in Malaysia in 1998 as a porcine neurologic and respiratory disease that spread to humans who had contact with live, infected pigs. Research reviewed in this paper suggests that anthropogenic factors, including agricultural expansion and intensification, were the underlying causes of its emergence. Nipah virus has caused five subsequent outbreaks between 2001 and 2005 in Bangladesh. Here, it appears to have spilled over directly from bats to humans, and person-to-person transmission is evident suggesting a heightened public health risk.

In late 1998, a novel paramyxovirus named Nipah virus, emerged in Malaysia, causing fatal disease in domestic pigs and humans

Bat Nipah virus, Thailand.

Wacharapluesadee S, Lumlertdacha B, Boongird K, Wanghongsa S, Chanhome L, Rollin P, Stockton P, Rupprecht CE, Ksiazek TG, Hemachudha T.
Emerg Infect Dis. 2005 Dec;11(12):1949-51.

Surveillance for Nipah virus (NV) was conducted in Thailand's bat population. Immunoglobulin G antibodies to NV were detected with enzyme immunoassay in 82 of 1,304 bats. NV RNA was found in bat saliva and urine. These data suggest the persistence of NV infection in Thai bats.

Anthropogenic deforestation, El Nino and the emergence of Nipah virus in Malaysia.

Chua KB, Chua BH, Wang CW.
Malays J Pathol. 2002 Jun;24(1):15-21.

In late 1998, a novel paramyxovirus named Nipah virus, emerged in Malaysia, causing fatal disease in domestic pigs and humans with substantial economic loss to the local pig industry. Pteropid fruitbats have since been identified as a natural reservoir host. Over the last two decades, the forest habitat of these bats in Southeast Asia has been substantially reduced by deforestation for pulpwood and industrial plantation. In 1997/1998, slash-and-burn deforestation resulted in the formation of a severe haze that blanketed much of Southeast Asia in the months directly preceding the Nipah virus disease outbreak. This was exacerbated by a drought driven by the severe 1997-1998 El Nino Southern Oscillation (ENSO) event. We present data suggesting that this series of events led to a reduction in the availability of flowering and fruiting forest trees for foraging by fruitbats and culminated in unprecedented encroachment of fruitbats into cultivated fruit orchards in 1997/1998. These anthropogenic events, coupled with the location of piggeries in orchards and the design of pigsties allowed transmission of a novel paramyxovirus from its reservoir host to the domestic pig and ultimately to the human population.

Novel viral encephalitides associated with bats (Chiroptera)--host management strategies.

Field H, Mackenzie J, Daszak P.
Arch Virol Suppl. 2004;(18):113-21.

Several novel viruses recently described in bats of the genus Pteropus (sub-order Megachiroptera) in Australia and southeast Asia cause encephalitic disease in animals and humans. These viruses include Hendra virus and Nipah virus (genus Henipavirus, family Paramyxoviridae) and Australian bat lyssavirus (ABLV; genus Lyssavirus, family Rhabdoviridae). Broadly, strategies for disease prevention and control in the spillover host are directed at minimising direct or indirect contact with the natural host, improving farm-gate and on-farm biosecurity, and better disease recognition and diagnosis. Additional strategies for ABLV include the use of rabies vaccine for effective pre- and post-exposure prophylaxis in humans. Effective management strategies in the natural host are predicated on an understanding of the ecology of the disease in the natural host, and the identification and avoidance of factors putatively associated with emergence, such as habitat loss, land use change and demographic shifts. A possible future management strategy for ABLV in reservoir populations is immunisation using bait or plant-derived vaccination.

Emerging encephalitogenic viruses: lyssaviruses and henipaviruses transmitted by frugivorous bats.

Mackenzie JS, Field HE.
Arch Virol Suppl. 2004;(18):97-111.

Three newly recognized encephalitogenic zoonotic viruses spread from fruit bats of the genus Pteropus (order Chiroptera, suborder Megachiroptera) have been recognised over the past decade. These are: Hendra virus, formerly named equine morbillivirus, which was responsible for an outbreak of disease in horses and humans in Brisbane, Australia, in 1994; Australian bat

lyssavirus, the cause of a severe acute encephalitis, in 1996; and Nipah virus, the cause of a major outbreak of encephalitis and pulmonary disease in domestic pigs and people in peninsula Malaysia in 1999. Hendra and Nipah viruses have been shown to be the first two members of a new genus, Henipavirus, in the family Paramyxoviridae, subfamily Paramyxovirinae, whereas Australian bat lyssavirus is closely related antigenically to classical rabies virus in the genus Lyssavirus, family Rhabdoviridae, although it can be distinguished on genetic grounds. Hendra and Nipah viruses have neurological and pneumonic tropisms. The first humans and equids with Hendra virus infections died from acute respiratory disease, whereas the second human patient died from an encephalitis. With Nipah virus, the predominant clinical syndrome in humans was encephalitic rather than respiratory, whereas in pigs, the infection was characterised by acute fever with respiratory involvement with or without neurological signs. Two human infections with Australian bat lyssavirus have been reported, the clinical signs of which were consistent with classical rabies infection and included a diffuse, non-suppurative encephalitis. Many important questions remain to be answered regarding modes of transmission, pathogenesis, and geographic range of these viruses.

Many important questions remain to be answered regarding modes of transmission, pathogenesis, and geographic range of these viruses.

Nipah virus encephalitis outbreak in Malaysia.
Lam SK, Chua KB.
Clin Infect Dis. 2002 May 1;34 Suppl 2:S48-51.

Emerging infectious diseases involving zoonosis have become important global health problems. The 1998 outbreak of severe febrile encephalitis among pig farmers in Malaysia caused by a newly emergent paramyxovirus, Nipah virus, is a good example. This disease has the potential to spread to other countries through infected animals and can cause considerable economic loss. The clinical presentation includes segmental myoclonus, areflexia, hypertension, and tachycardia, and histologic evidence includes endothelial damage and vasculitis of the brain and other major organs. Magnetic resonance imaging has demonstrated the presence of discrete high-signal-intensity lesions disseminated throughout the brain. Nipah virus causes syncytial formation in Vero cells and is antigenically related to Hendra virus. The Island flying fox (Pteropus hypomelanus; the fruit bat) is a likely reservoir of this virus. The outbreak in Malaysia was controlled through the culling of >1 million pigs.

Nipah virus encephalitis reemergence, Bangladesh.
Hsu VP, Hossain MJ, Parashar UD, Ali MM, Ksiazek TG, Kuzmin I, Niezgoda M, Rupprecht C, Bresee J, Breiman RF.
Centers for Disease Control and Prevention, Atlanta, Georgia, USA. vhsu@att.net
Emerg Infect Dis. 2004 Dec;10(12):2082-7.

We retrospectively investigated two outbreaks of encephalitis in Meherpur and Naogaon, Bangladesh, which occurred in 2001 and 2003. We collected serum samples from persons who were ill, their household contacts, randomly selected residents, hospital workers, and various animals. Cases were classified as laboratory confirmed or probable. We identified 13 cases (4 confirmed, 9 probable) in Meherpur; 7 were in persons in two households. Patients were more likely than nonpatients to have close contact with other patients or have contact with a sick cow. In Naogaon, we identified 12 cases (4 confirmed, 8 probable); 7 were in persons clustered in 2 households. Two Pteropus bats had antibodies for Nipah virus. Samples from hospital workers were negative for Nipah virus antibodies. These outbreaks, the first since 1999, suggest that transmission may occur through close contact with other patients or from exposure to a common source. Surveillance and enhancement of diagnostic capacity to detect Nipah virus infection are recommended.

Nipah virus outbreak in Malaysia.
Chua KB.
J Clin Virol. 2003 Apr;26(3):265-75.

Nipah virus, a novel paramyxovirus, closely related to Hendra virus emerged in northern part of Peninsular Malaysia in 1998. The virus caused an outbreak of severe febrile encephalitis in humans with a high mortality rate, whereas, in pigs, encephalitis and respiratory diseases but with a relatively low mortality rate. The outbreak subsequently spread to various regions of the country and Singapore in the south due to the movement of infected pigs. Nipah virus caused systemic infections in humans, pigs and other mammals. Histopathological and radiological findings were characteristic of the disease. Fruitbats of Pteropid species were identified as the natural reservoir hosts. Evidence suggested that climatic and anthropogenic driven ecological changes coupled with the location of piggeries in orchard and the design of pigsties allowed the spill-over of this novel paramyxovirus from its reservoir host into the domestic pigs and ultimately to humans and other animals.

Nipah encephalitis outbreak in Malaysia.
Tan CT, Wong KT.
Ann Acad Med Singapore. 2003 Jan;32(1):112-7.

INTRODUCTION: Between September 1998 and June

1999, there was a severe outbreak of viral encephalitis among the pig farm workers in Malaysia. METHODS: This is a review of the published literature related to the outbreak with the focus on human diseases. RESULTS: The encephalitis was caused by a newly discovered paramyxovirus related to Hendra virus, later named Nipah virus. There were 265 patients with acute encephalitis. The disease is thought to spread from pig to man through close contact. The risk of human-to-human spread is thought to below. The disease affected mainly adult Chinese males, half of whom had affected family members. The disease presented mainly as acute encephalitis with a short incubation period of less than two weeks, with the main symptoms of fever, headache, and giddiness followed by coma. Distinctive clinical signs include segmental myoclonus, areflexia and hypotonia, hypertension, and tachycardia. Initial cerebrospinal fluid was abnormal in 75% of patients. Serology was helpful in confirming the diagnosis. Magnetic resonance imaging showed distinctive changes of multiple, discrete, and small high signal lesions, best seen with fluid-attenuated inversion recovery (FLAIR) sequences. Mortality was high at 40% and death was probably due to severe brainstem involvement. The main necropsy finding in acute encephalitis was that of disseminated microinfarction associated with vasculitis and direct neuronal involvement. Ribavirin was able to reduce the mortality by 36%. Relapse encephalitis was seen in 7.5% of those who recovered from acute encephalitis, and late-onset encephalitis in 3.4% of those with initial non-encephalitic or asymptomatic diseases. The mean interval between initial illness and the onset of the complication was 8.4 months. The relapse and late-onset encephalitis which manifested as focal encephalitis arose from recurrent infection. CONCLUSION: Nipah virus, a recently discovered paramyxovirus, causes a unique encephalitis with high mortality as well as relapse and late-onset encephalitis. The infection is mainly spread from pigs to man.

Risk factors for Nipah virus infection among abattoir workers in Singapore.

Chew MH, Arguin PM, Shay DK, Goh KT, Rollin PE, Shieh WJ, Zaki SR, Rota PA, Ling AE, Ksiazek TG, Chew SK, Anderson LJ.

J Infect Dis. 2000 May;181(5):1760-3.

During 10-19 March 1999, 11 workers in 1 of 2 Singaporean abattoirs developed Nipah-virus associated encephalitis or pneumonia, resulting in 1 fatality. A case-control study was conducted to determine occupational risk factors for infection. Case patients were abattoir A workers who had anti-Nipah IgM antibodies; control subjects were randomly selected abattoir A workers who tested negative for anti-Nipah IgM. All 13 case patients versus 26 (63%) of 41 control subjects reported contact with live pigs (P=.01). Swine importation from Malaysian states concurrently experiencing a Nipah virus outbreak was banned on 3 March 1999; on 19 March 1999, importation of Malaysian pigs was banned, and abattoirs were closed. No unusual illnesses among pigs processed during February-March were reported. Contact with live pigs appeared to be the most important risk factor for human Nipah virus infection. Direct contact with live, potentially infected pigs should be minimized to prevent transmission of this potentially fatal zoonosis to humans.

Epidemiology of sexually transmitted diseases: the global picture.

De Schryver A, Meheus A.

Bull World Health Organ. 1990;68(5):639-54.

Sexually transmitted diseases (STD) are now the commonest group of notifiable infectious diseases in most countries, particularly in the age group of 15 to 50 years and in infants. Their control is important considering the high incidence of acute infections, complications and sequelae, their socioeconomic impact, and their role in increasing transmission of the human immunodeficiency virus (HIV). The worldwide incidence of major bacterial and viral STD is estimated at over 125 million cases yearly. STD are hyperendemic in many developing countries. In industrialized countries, the bacterial STD (syphilis, gonorrhoea, chancroid) declined from the peak during the Second World War till up to the late fifties, then increased during the sixties and early seventies, and they have been decreasing again from the late seventies till the present. In the industrialized world, diseases due to Chlamydia trachomatis, genital herpes virus, human papillomaviruses and human immunodeficiency virus are now more important than the classical bacterial ones; both groups remain major health problems in most developing countries. Infection rates are similar in both women and men, but women and infants bear the major burden of complications and serious sequelae. Infertility and ectopic pregnancies are often a consequence of pelvic inflammatory disease, and are preventable. Sexually transmitted diseases in pregnant women can result in prematurity, stillbirth and neonatal infections. In many areas 1-5% of newborns are at risk of gonococcal ophthalmia neonatorum, a blinding disease; congenital syphilis causes up to 25% of perinatal mortality. Genital and anal cancers (especially cervical cancer) are associated with viral sexually transmitted diseases (genital human papillomavirus and herpes virus

Contact with live pigs appeared to be the most important risk factor for human Nipah virus infection

infections). Urethral stricture and infertility are frequent sequelae in men.

PIP: Sexually transmitted diseases (STDs) are now the most common group of identifiable infectious diseases in many countries, especially among those ages 15-50 and in infants. Their control is important considering the high incidence of acute infections, complications and sequelae, their socioeconomic impact, and their role in increasing transmission of the human immunodeficiency virus (HIV). THe worldwide incidence of major bacterial and viral STDs is estimated to be over 125 million cases yearly. STDs are hyperendemic in many developing countries. However, in industrialized countries, the bacterial STDs such as syphilis, gonorrhea, chancroid declined from their peak during WW II until the late 1950s, increased during the 1960s and early 1970s, and have again decreased since that time. In the industrialized world, diseases due to Chlamydia trachomatis, genital herpes virus, human papillomaviruses, and HIV are now more significant than the classical bacterial ones; both groups remain major health problems in most developing countries. Infection rates are similar in both men and women, but women and infants bear the major burden of complications and serious sequelae. Infertility and ectopic pregnancy are often a result of pelvic inflammatory disease and are preventable. STDs in pregnant women can result in prematurity, stillbirth, and neonatal infections. In many areas, 1-5% of newborns are at risk of gonococcal ophthalmia neonatorum, a disease that blinds and congenital syphilis causes up to 25% of perinatal mortality. Genital and anal cancers (especially cervical cancer) are associated with viral STDs (genital human papillomavirus and herpes virus infections). Urethral stricture and infertility are frequent sequelae in men. (author's modified)

In many areas, 1-5% of newborns are at risk of gonococcal ophthalmia neonatorum, a disease that blinds and congenital syphilis causes up to 25% of perinatal mortality

Decreasing incidences of gonorrhea- and chlamydia-associated acute pelvic inflammatory disease. A 25-year study from an urban area of central Sweden.

Kamwendo F, Forslin L, Bodin L, Danielsson D.
Sex Transm Dis. 1996 Sep-Oct;23(5):384-91.

BACKGROUND AND OBJECTIVES: Acute pelvic inflammatory disease (PID) affects women in their reproductive years and is often a complication of a sexually transmitted disease (STD), particularly Neisseria gonorrhoeae and Chlamydia trachomatis. Infertility, ectopic pregnancy, and chronic lower abdominal pain are common long-term sequelae to acute PID. Through different preventive measures, endemic N. gonorrhoeae is almost eliminated, and C. trachomatis has been reduced almost fourfold in Sweden. GOALS: To investigate variations in STD-associated acute PID and the extent to which this influenced the yearly incidences of patients hospitalized for this complication during a 25-year-period. STUDY DESIGN: Hospital records of 2499 patients admitted and treated for acute PID from January 1, 1970 to December 31, 1994 were analyzed for infection with N. gonorrhoeae. Routine laboratory diagnosis for C. trachomatis infection started June 1, 1980. Detailed statistical analysis for chlamydial-associated PID in this study, therefore, covers the period January 1, 1981 to December 31, 1994 and includes 1030 patients. RESULTS: Gonorrhea occurred in 42% of patients with acute PID in 1970 and decreased continuously to zero in 1988 and beyond. Concomitant urogenital chlamydial infection reduced almost fourfold from 28.4% in 1985 to 7.7% in 1994. Yearly admissions for acute PID fluctuated slightly (< or = 16%) in the early 1970s and early 1980s but increased greatly (> 60%) in the middle and late 1970s; the highest was 180 per year in 1976. This coincided with high incidence rates of gonorrhea in the general population, and probably of genital C. trachomatis infection as well, coupled with an increased use of intrauterine contraceptive device in nulliparous women. The largest increase in admissions for acute PID was in the 15- to 29-year-old group. A steady decrease started in 1987 and reached the low figure of 26 admissions in 1994. The greatest decrease occurred in the 15- to 19-year-old group, from the relative age distribution of 28.9% in the period 1970 to 1974 to 12.9% in 1990 to 1994. Yearly admissions for the > or = 35-year-old group remained almost constant during the study period, but the relative age distribution shifted from second lowest (excluding those 14 years or younger, totaling 15 admissions for the entire study period), 9.1% at the beginning of the study period, to the second largest, 24.9% at the end of it. The study also showed that the total and relative rates of recurrence decreased.
CONCLUSIONS: Measures aimed at reducing incidences of gonorrhea and genital chlamydial infection will reduce the incidences of one of the most serious complications of these STDs, acute PID, and, in turn, its long-term sequelae.

The frequency of salpingitis and ectopic pregnancy as epidemiologic markers of Chlamydia trachomatis.

Bjartling C, Osser S, Persson K.
Acta Obstet Gynecol Scand. 2000 Feb;79(2):123-8.

BACKGROUND: To study the incidence of non-gonococcal salpingitis, gonococcal salpingitis and

ectopic pregnancy in a defined population over a 28-year period on the assumption that the frequency of salpingitis and ectopic pregnancy may indirectly illustrate the epidemiological pattern of Chlamydia trachomatis. DESIGN: A retrospective epidemiological study. SETTING: University hospital with an urban catchment area. PATIENTS: Five thousand two hundred and thirty-three patients admitted to the hospital between 1969 and 1996 with a diagnosis of ectopic pregnancy, non-gonococcal salpingitis, or gonococcal salpingitis. RESULTS: The frequencies of both non-gonococcal and gonococcal salpingitis increased steeply early in the period under study, rising to a peak in the early 1970s, then decreasing throughout the period except for the last 3 years when a slight increase was seen again. The frequency of ectopic pregnancy showed a steady increase, peaking in the late 1980s and early 1990s and then declining at the end of the study period. While the introduction of more sensitive pregnancy tests and programs for assisted fertility would increase the rate of ectopic frequency the decline during the 'nineties cannot be accounted for in this way. The peak of salpingitis cases in the early 'seventies seems to be mirrored exactly by the peak of ectopic pregnancies fifteen years later in the late 'eighties. CONCLUSION: The frequencies of salpingitis and of ectopic pregnancy can probably be used to estimate the incidence of preceding Chlamydia trachomatis. Thus the incidence of C. trachomatis has probably declined since the early 'seventies like that of N. gonorrheae.

Thus the incidence of C. trachomatis has probably declined since the early 'seventies like that of N. gonorrheae.

Assessing the impact of national anti-HIV sexual health campaigns: trends in the transmission of HIV and other sexually transmitted infections in England.

Nicoll A, Hughes G, Donnelly M, Livingstone S, De Angelis D, Fenton K, Evans B, Gill ON, Catchpole M.
Sex Transm Infect. 2001 Aug;77(4):242-7.

OBJECTIVE: To assess the impact of the sexual component of AIDS and HIV campaigns on transmission of HIV and other sexually transmitted infections (STIs). DESIGN: Comparison of time series data. SETTING: England, 1971-1999. OUTCOME MEASURES: HIV transmission and diagnoses among men who have sex with men (MSMs), rates of attendances and specific STI diagnoses (per 100 000 total population) at genitourinary medicine (GUM) clinics. RESULTS: Awareness of AIDS and campaigns in 1983-4 among homosexual men coincided with substantial declines in transmission of HIV and diagnoses of syphilis among MSMs. During general population campaigns in 1986-7 new GUM clinic attendances requiring treatment fell by 117/10(5) in men and 42/10(5) in women. Rates for gonorrhoea fell by 81/10(5) and 43/10(5) and genital herpes by 6/10(5) and 4/10(5), respectively. Previous rises in genital wart rates were interrupted, while rates of attendances not requiring treatment (the "worried well") increased by 47/10(5) and 58/10(5) for men and women, respectively. Since 1987 diagnoses of HIV among MSMs have not declined, averaging 1300-1400 annually. Following a period of unchanging rates there have been substantial increases in GUM attendances requiring treatment, notably for gonorrhoea, syphilis, and viral STIs since 1995. CONCLUSIONS: Self help initiatives and awareness among homosexual men in 1983-4 contributed significantly to a fall in HIV transmission among MSMs, and the general campaigns of 1986-7 were associated with similar effects on all STI transmission. Both effects seem to have occurred through changing sexual behaviour, and probably contributed to the UK's low national HIV prevalence. Bacterial STI incidence has increased significantly since 1995 and there is no evidence that recent prevention initiatives have reduced HIV transmission among MSMs, hence sexual health initiatives need to be comprehensively reinvigorated in England.

Epidemic polyarthritis (Ross River) virus infection in the Cook Islands.

Rosen L, Gubler DJ, Bennett PH.
Am J Trop Med Hyg. 1981 Nov;30(6):1294-302.

An epidemic of Ross River virus infection occurred in the Cook Islands early in 1980 and affected the majority of the inhabitants of Rarotonga, the most populated island in the group. This represents the easternmost extension of the virus which, until 1979, was believed limited to Australia, New Guinea, and the Solomon Islands. The clinical manifestations of Ross River disease, predominantly polyarthritis, did not differ significantly from those observed previously in Australia. However, unlike the experience in Australia, where Ross River virus has never been isolated from a patient with polyarthritis, the agent was recovered from the serum of one-half of approximately 100 such patients with serologically proven infections. It is not known if this latter observation is the result of a change in the virus, the different virus isolation technique employed, or other factors. It was found that the incubation period of the disease could be as short as 3 days--much less than previously suspected. Ross River virus was isolated from six pools of Aedes polynesiensis mosquitoes collected in nature and it appeared that this species was the most probable vector on Rarotonga. In view of the widespread distribution of Ae. polynesiensis on islands, in the eastern Pacific it would not be surprising if Ross

River virus occurs in other previously unaffected areas in the future.

Ross River virus disease reemergence, Fiji, 2003-2004.

Klapsing P, MacLean JD, Glaze S, McClean KL, Drebot MA, Lanciotti RS, Campbell GL.

Emerg Infect Dis. 2005 Apr;11(4):613-5.

We report 2 clinically characteristic and serologically positive cases of Ross River virus infection in Canadian tourists who visited Fiji in late 2003 and early 2004. This report suggests that Ross River virus is once again circulating in Fiji, where it apparently disappeared after causing an epidemic in 1979 to 1980.

Ross River virus (Togaviridae: Alphavirus) infection (epidemic polyarthritis) in American Samoa.

Tesh RB, McLean RG, Shroyer DA, Calisher CH, Rosen L. Trans R Soc Trop Med Hyg. 1981;75(3):426-31.

An outbreak of Ross River virus infection (epidemic polyarthritis), which occurred in American Samoa between August 1979 and January 1980, is described. On the basis of a serological survey performed near the end of the epidemic, it is estimated that at least 13,500 people were infected. Ross River virus was isolated from the blood of a single polyarthritis patient. Plaque reduction neutralization tests, using this virus strain, were done on 393 human and 143 animal sera collected on Tutuila island. Over-all, 43.8% of the people sampled had evidence of infection. Sera from 100 adult residents of the same island, collected in 1972, had no Ross River antibody, suggesting recent introduction of the virus. In contrast to the human serological data, the prevalence of Ross River antibodies among animals was relatively low. Dogs and pigs had the highest rates with 20% and 15%, respectively. Results of this study suggest that the Ross River virus cycle during the epidemic in American Samoa involved primarily humans and mosquitoes with animals less frequently infected. These observations plus the recent introduction of Ross River virus into new areas of the South Pacific suggest that a major change has occurred in the epidemiology of epidemic polyarthritis.

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[Laboratory diagnosis of epidemic polyarthritis (Ross River virus) in New Caledonia]

[Article in French]

Panon G, Le Gonidec G, Fauran P.

Bull Soc Pathol Exot Filiales. 1983 Dec;76(5 Pt 2):755-60.

During an outbreak of arboviral diseases in the South-West Pacific islands, from 1979 to 1980, patients sera examined in New Caledonia revealed 43 cases of epidemic polyarthritis due to Ross River virus. Viral strains were obtained from 16 of these patients. After isolation of the virus in newborn mice, the inoculation of C6/36, PS-EK and Vero cells allowed the identification of these strains by hemagglutination inhibition and complement fixation tests. This was the first time that cases of polyarthritis caused by RR virus were observed in New Caledonia, Vanuatu, Wallis and Futuna.

Vector competence of geographic strains of Aedes albopictus and Aedes polynesiensis and certain other Aedes (Stegomyia) mosquitoes for Ross River virus.

Mitchell CJ, Gubler DJ.

Am Mosq Control Assoc. 1987 Jun;3(2):142-7.

The vector competence of geographic strains of Aedes albopictus and Ae. polynesiensis and Fiji strains of Ae. pseudoscutellaris and Ae. aegypti was assessed for Ross River (RR) virus, the etiologic agent of epidemic polyarthritis. Strains of Ae. polynesiensis from Fiji, Rarotonga, Somoa and Tahiti were the most susceptible to infection per os (MID50 less than or equal to 10(1.2) Vero cell plaque-forming units [PFU]/blood meal). Virus transmission data were variable, but all strains except the one from Fiji transmitted virus at 14 to 21 days postinfection. Shanghai and Hawaii Ae. albopictus and Fiji Ae. pseudoscutellaris were also highly susceptible to per os infection with RR virus (MID50 10(2.0) to 10(2.6) PFU). Singapore and Sri Lanka Ae. albopictus and Fiji Ae. aegypti were the least susceptible (MID50 10(4.0) to 10(4.2) PFU). With one exception, virus transmission rates for Ae. pseudoscutellaris and Ae. aegypti and the four strains of Ae. albopictus ranged from 52 to 100%. A total of 4,718 third- and fourth-instar larvae from the second gonotrophic cycle of potentially infected females were tested for RR virus in 39 pools. Infection rates in parental females ranged from 87 to 100% in Ae. albopictus, Ae. pseudoscutellaris and Ae. polynesiensis and 40 to 48% in Ae. aegypti. Virus was not isolated from larval progeny.

Transmission of Ross River virus by Aedes polynesiensis and Aedes aegypti.

Gubler DJ.

Am J Trop Med Hyg. 1981 Nov;30(6):1303-6.

Laboratory studies were carried out with two geographic strains of Aedes polynesiensis and one strain of Aedes

aegypti to determine whether they could transmit Ross River virus (RRV). Both species were shown to be good vectors of RRV, but Ae. polynesiensis was the most susceptible. Ae. polynesiensis represents a new vector for this virus and the epidemiologic implications of RRV spread by both mosquito species are discussed.

[Arboviral diseases in South-West Pacific islands (author's transl)]

[Article in French]
Le Gonidec G, Fauran P.
Med Trop (Mars). 1981 Jan-Feb;41(1):85-92.

Islands of the south-west Pacific area belong to the melanesian group, excepted Niue, Tonga, Wallis and Futuna which are polynesian. Through New Guinea, there is a geographic relation to the eastern part of Australia, rich of 42 arbovirus types. Dengue and Ross River fever are the most important arboviral diseases in the region; both affect islanders after introduction of virus by travellers to localities where efficient vectors are present. Dengue types 1, 2 and 4 were isolated from man and from mosquitoes in this area. Successive outbreaks, transmitted by Aedes aegypti or by Ae. polynesiensis, resulted in thousands of cases with a few fatal hemorrhagic forms. Ross River virus, responsible of epidemic polyarthritis, evaded in 1979, from Australia to Fidji, Wallis, Futuna and New Caledonia. Suspected vectors are Aedes vigilax and Culex annulirostris. Aedes aegypti and Ae. polynesiensis are also possible carriers. Murray Valley encephalitis virus caused severe outbreaks in Australia and fatal cases in New Guinea; it is a possible invader when Culex annulirostris is abundant. Expansion of arboviral diseases is a major epidemiological problem in south-west Pacific islands. Research work on pathogeny and vectors control must be intensified in this area.

[Chikungunya virus: its recent spread to the southern Indian Ocean and Reunion Island (2005-2006)]

[Article in French]
Chastel C.
Bull Acad Natl Med. 2005 Nov;189(8):1827-35.

Chikungunya virus (alphavirus, Togaviridae) is transmitted by mosquitoes of the Aedes genus and responsible for a dengue-like acute disease characterized by severe arthralgias sometimes persisting during months and eventually years. Its geographical distribution is large, including west, central, east and southern tropical Africa, India and South-eastern Asia. Since 2005 february, Chikungunya disease invaded a number of islands in southern Indian Ocean, namely Comoro, Mauritius, Seychelles and Reunion islands. In this French department, it was responsible for 110000 to 200000 infections, neurological disorders, many deaths and some congenital infections not previously observed during preceding epidemics.

Isolation of chikungunya virus from Aedes aegypti mosquitoes collected in the town of Yawat, Pune District, Maharashtra State, India.

Mourya DT, Thakare JR, Gokhale MD, Powers AM, Hundekar SL, Jayakumar PC, Bondre VP, Shouche YS, Padbidri VS.
Acta Virol. 2001;45(5-6):305-9.

Chikungunya (CHIK) virus is prevalent throughout Southeast Asia and Africa. It has caused numerous large outbreaks in India. No active or passive surveillance has been carried out since the last epidemic occurring in 1971. During a recent outbreak of Dengue (DEN)-like illness in eastern India, Aedes aegypti mosquitoes collected from the affected area were positive for CHIK virus. Evidence of dual infection with CHIK and DEN typel virus was also obtained. A widely circulating low-virulent CHIK virus is a possible explanation for the epidemiological pattern of the CHIK virus disease in this region.

the potentially severe impact of pandemic influenza on population health and health sector capacity provides a strong case for health authorities to intensify preparatory efforts and to strengthen health sector infrastructure

Estimating the impact of the next influenza pandemic on population health and health sector capacity in New Zealand.

Wilson N, Mansoor O, Baker M.
N Z Med J. 2004 Mar 11;118(1211):U1346.

AIM: To estimate the impact of the next influenza pandemic on population health and health sector capacity in New Zealand. METHOD: Population data for New Zealand was used with the software package 'FluAid' (CDC, Atlanta). Additional data was used to provide estimates of impacts on health sector capacity. RESULTS: For incidence rates in the 15% to 35% range for the first pandemic wave, the modelling results give a range of 1600 to 3700 deaths attributable to pandemic influenza. The estimated range of hospitalisations was between 6900 and 16,200. The estimated number of cases of illness requiring medical consultation ranged from 325,000 to 759,000. For the peak week of an 8-week epidemic (35% incidence scenario), it was estimated that 42% of all public hospital beds would be required at least for some proportion of the week and

that the average general practitioner would be consulted by around 80 people with influenza.
CONCLUSION: This modelling work has a number of limitations and so these results could still substantially over- or under-estimate the impact of the next influenza pandemic. Nevertheless, the potentially severe impact of pandemic influenza on population health and health sector capacity provides a strong case for health authorities to intensify preparatory efforts and to strengthen health sector infrastructure.

Containing pandemic influenza at the source.

Longini IM Jr, Nizam A, Xu S, Ungchusak K, Hanshaoworakul W, Cummings DA, Halloran ME.
Science. 2005 Aug 12;309(5737):1083-7.

Combinations of targeted antiviral prophylaxis, pre-vaccination, and quarantine could contain strains with an R(0) as high as 2.4.

Highly pathogenic avian influenza A (subtype H5N1) is threatening to cause a human pandemic of potentially devastating proportions. We used a stochastic influenza simulation model for rural Southeast Asia to investigate the effectiveness of targeted antiviral prophylaxis, quarantine, and pre-vaccination in containing an emerging influenza strain at the source. If the basic reproductive number (R0) was below 1.60, our simulations showed that a prepared response with targeted antivirals would have a high probability of containing the disease. In that case, an antiviral agent stockpile on the order of 100,000 to 1 million courses for treatment and prophylaxis would be sufficient. If pre-vaccination occurred, then targeted antiviral prophylaxis could be effective for containing strains with an R0 as high as 2.1. Combinations of targeted antiviral prophylaxis, pre-vaccination, and quarantine could contain strains with an R(0) as high as 2.4.

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Any mind that is capable of real sorrow is capable of real good
(Bloomsbury Dictionary of Proverbs – 1994)