DISEASE WATCH | IN THE NEWS

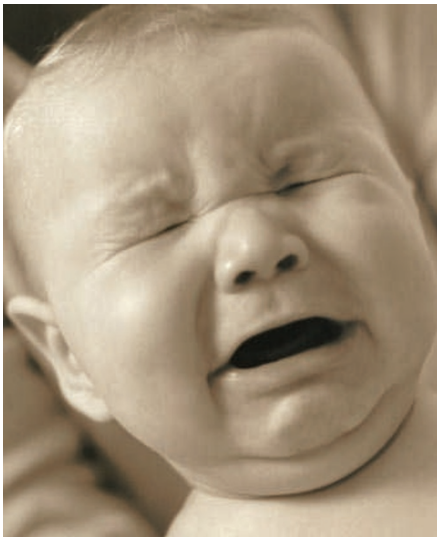
Varying progress on polio

Many experts think that the 2012 target for the eradiction of polio is likely to be missed, but hope has been renewed following the remarkable success of anti-polio programmes in Nigeria and India, two global hotspots for polio infection. In Nigeria, improved funding and management of public health programmes coupled with better ties between such programmes and local Muslim leaders, who had previously obstructed efforts to combat polio, have resulted in only 2 children becoming paralyzed with polio so far this year compared with 123 during the same period last year. Meanwhile, in India, tracking of the virus to the vast flood plain in the state of Bihar has allowed for a more targeted vaccination strategy that has contributed to the states of Uttar Pradesh and Bihar reporting no cases of infection since January 2010, the first time that no new infections have been reported for 4 consecutive months. Globally, such efforts have contributed to a 75% drop in new cases compared with the same period in 2009. However, although this news is welcome, it is tempered by the recent detection of type 1 polio virus in diagnostic samples in Tajikistan. Although Tajikistan is in Central Asia geographically, the WHO groups the country into its Europe region, which makes this the first outbreak in the region since it was certified polio free in 2002. ***NY Times*/WHO**

Details on emerging fungal threat

The pathogenic fungus *Cryptococcus gattii* has emerged over the past decade in the Pacific Northwest of the United States and Canada. Historically, *C. gattii* has been associated with eucalyptus trees in tropical and subtropical climates, making its appearance in temperature regions particularly alarming, as it indicates an expansion in the endemic ecology of this pathogen. *C. gattii* can be classified into four discrete molecular types (VGI–VGIV), with the VGII type being responsible for 95% of the infections seen in the United States and Canada. A new subtype of the fungus, VGIIc, has become a major source of illness in Oregon, USA, infecting 21 people. Writing in *PLoS Pathogens*, Joe Heitman and colleagues now show that this subtype is clonal. VGIIc and the related subtype VGIIa, which first appeared in 1999 in Vancouver, Canada, are sexually fertile and support ongoing recombination in the global VGII population. Furthermore, in macrophage and mouse infection models, the VGIIa and VGIIc isolates are highly virulent. Unlike non-outbreak *C. gattii* isolates, which cause infection at low incidence in immunocompromised individuals, the emerging *C. gattii* subtypes infect healthy individuals and therefore pose a serious threat to public health. ***PLoS Pathog*/ABC**

Scale of RSV disease burden revealed



Respiratory syncytial virus (RSV) is the most common cause of viral lung infection in children, causing mild flu-like symptoms in most cases but also leading to serious illness and death in babies that are born prematurely or that have a congenital heart condition. However, the global burden of disease that can be attributed to RSV was unknown. Reporting in *The Lancet*, Nair *et al.* now address this issue using data from a systematic review of studies published between January 1995 and June 2009 as well as the results of 10 unpublished population-based studies. Their findings indicate that an estimated 33.8 million episodes of RSV-associated acute lower-respiratory infection occurred in children under 5 years of age worldwide in 2005, with at least 3.4 million episodes requiring hospital admission. They go on to estimate that in the same year 66,000–199,000 children under 5 years of age died as a result of RSV infection and that 99% of these deaths occurred in developing countries. RSV is therefore the third most common cause of mortality associated with acute lower-respiratory infection, behind *Streptococcus pneumoniae* and *Haemophilus influenza* type b infections, highlighting the need to accelerate the development of treatment and prevention strategies. ***Lancet*/BBC**

HIV blunts *Salmonella* response

The association between infection with HIV and non-typhoidal strains of *Salmonella* has long been appreciated, but the mechanistic basis underlying the fatality of this co-infection has remained unclear. Although salmonellae are facultative intracellular pathogens and the host innate immune system is important for controlling *Salmonella* infections, it was recently observed that antibody-induced complement-mediated killing has a protective role against bacteraemia in African children. Molyneux and colleagues set out to determine whether antibody-mediated responses might be involved in the impaired host defences against non-typhoidal *Salmonella* strains in HIV-infected individuals. They found that the lack of bactericidal activity observed in HIV-infected individuals resulted from an overproduction of immunoglobulin G specific to lipopolysaccharide (LPS) of *Salmonella enterica* subspecies *enterica* serovar Typhimurium. Excess LPS-specific antibodies blocked complement-mediated killing of S. Typhimurium, and killing was restored by either genetically shortening the LPS or removing the LPS-specific antibodies from the serum. These findings suggest that the aberrant immune response to salmonellae in HIV-infected individuals results in competition between blocking antibodies and killing antibodies. ***Science***

Outbreak news

Norovirus. According to figures from the UK Health Protection Agency, confirmed cases of norovirus infection have almost doubled in Britain since January 2010 compared with the same 3 month period last year. The agency predicts that more than 1 million people could be hit by norovirus infection this year, with a highly infectious strain and the coldest winter for decades the potential causes. **Telegraph**

In the News was compiled with the assistance of David Ojcius, University of California, Merced, USA. David's links to infectious disease news stories can be accessed on Connotea (http://www.connotea.org), under the username NatureRevMicrobiol.