DISEASE WATCH | IN THE NEWS

Dysentery on the run

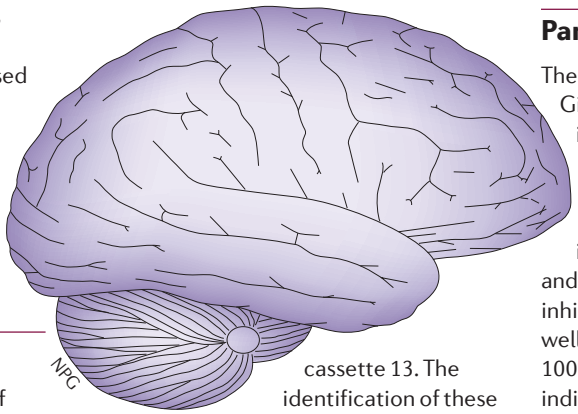
A drug that is already approved to treat rheumatoid arthritis is active against the protozoan parasite *Entamoeba histolytica*, the causative agent of amoebic dysentery in humans. Dysentery, or amoebiasis, is the fourth leading cause of mortality worldwide, causing ~70,000 deaths each year (mostly in developing countries). Current approaches for treating amoebic dysentery rely on the use of metronidazole; however, this drug has adverse side effects, and resistance in *E. histolytica* is thought to be on the increase. A high-throughput screen of US Food and Drug Administration (FDA)-approved and non-FDA-approved drugs identified the arthritis drug auranofin as being ten times as active as metronidazole against *E. histolytica* in culture. Furthermore, auranofin decreased parasite numbers and reduced disease symptoms in mouse and hamster infection models. Auranofin is thought to work by inhibiting the *E. histolytica* thioredoxin reductase, resulting in enhanced sensitivity to reactive oxygen species. **BBC/*Nature Med.***

Gonorrhoea fears grow

The number of newly diagnosed cases of gonorrhoea increased by 25% in England during 2011, according to the UK Health Protection Agency. Gonorrhoea is a sexually transmitted infection (STI) caused by the Gram-negative bacterium *Neisseria gonorrhoeae* and can result in infertility in infected individuals. The 25% increase in diagnosed cases for gonorrhoea is particularly troubling against an overall increase of just 2% for all STIs. In addition, resistance to first-line antibiotics is spreading among *N. gonorrhoeae* isolates, prompting fears that the infection could become untreatable. A spokesperson from the UK sexual health charity the Terrence Higgins Trust said, "These figures must act as a wake-up call, not only to sexually active people but also to the government and public health services." **BBC**

Malaria gets cerebral

Three papers have identified the parasite molecules that are expressed on the surface of infected erythrocytes during cerebral malaria. In patients with cerebral malaria (the most severe consequence of a *Plasmodium falciparum* infection), infected erythrocytes accumulate in the microvasculature of the brain, causing life-threatening inflammation. The adhesin *P. falciparum* erythrocyte membrane protein 1 (PfEMP1) is expressed on the surface of infected erythrocytes and binds to human receptors on the vascular endothelial cell surface to mediate attachment. Multiple variants of PfEMP1 are known to exist and are associated with the virulence of infection. The identity of the variants associated with cerebral malaria was unknown, but now three different approaches have identified PfEMP1 variants with distinct types of amino-terminal domains, consisting of domain cassette 8 or domain cassette 13. The identification of these variants provides new drug targets for combating cerebral malaria.



In addition, a fourth study has shown that administration of the host innate defence regulator peptide IDR-1018 together with standard antimalarial drugs increases the survival of mice suffering from cerebral malaria by reducing the inflammation associated with fatality.

Proc. Natl Acad. Sci. USA*/*Sci. Transl. Med.

Setting the scene for SIV

The number of T helper 17 (T_H17) cells in the blood and intestinal tissue prior to infection with simian immunodeficiency virus (SIV) can limit viral replication, according to a new study. Some individuals infected with HIV (or SIV) resist disease progression, whereas others progress more rapidly; however, the mechanisms underlying this phenomenon are incompletely understood. Although viral and host genetic factors are known to have an important role in the speed of disease progression, the status of the host's immune system prior to infection and its role in disease progression have been difficult to assess. The latest findings show that SIV infection of rhesus macaques with high numbers of T_H17 cells in their blood and intestinal tissue resulted in peak and set-point viral loads of one log lower than those in infected rhesus macaques with low T_H17 cell numbers. Furthermore, depletion of T_H17 cells prior to infection led to higher viral loads for 6 months following infection.

These findings suggest that the composition of the host immune system prior to viral infection will affect disease progression and should therefore be taken into account when developing therapeutics and vaccines to target HIV. **EurekAlert/*Sci. Transl. Med.***

Partnership for HCV drugs?

There have been renewed calls for Gilead Sciences, Inc. to enter one of its hepatitis C virus (HCV) drugs into Phase III clinical trials in combination with a drug from Bristol-Myers Squibb Co. The drugs, daclatasvir (an NS5A inhibitor from Bristol-Myers Squibb) and GS-7977 (a nucleotide polymerase inhibitor from Gilead Sciences), performed well together in a mid-stage trial, with a 100% response rate in previously untreated individuals infected with the most common form of HCV. However, Gilead Sciences is pursuing combinations of GS-7977 with its own NS5A inhibitor rather than continuing into Phase III trials with daclatasvir.

An estimated 170–180 million individuals worldwide are infected with HCV, and current treatments that rely on the use of pegylated interferons and ribavirin can have adverse side effects that result in poor adherence to treatment regimes. **Reuters**

Outbreak news

Measles. The number of measles cases diagnosed in Ukraine has increased sharply, with 63 reported cases in a 24-hour period in May 2012, according to the Ukraine Ministry of Health. The rise in the number of cases is of particular concern ahead of the Euro 2012 football championship, which will be hosted by Ukraine and Poland and will see many thousands of fans travelling to the country. **forUm/Outbreaknews.com**

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 his Twitter page (@Ojcius).