Research brief

For more on the **virulence of Zika virus** see *PLoS Negl Trop Dis* 2019; **13:** e0007387

For more on **blocking structural changes in picornaviruses** see *PLoS Biol* 2019; **17:** e3000281

For more on **measuring vulnerability to viral outbreaks** see *Philos Trans R Soc Lond B Biol Sci* 2019; **374:** 20180265

For more on the **evolution of plague** see *Proc Natl Acad Sci USA* 2019; published online June 4. https://doi.org/10.1073/pnas.1820447116

For more on **antibodies against malaria** see *Sci Transl Med* 2019; **11:** eaav3963

For more on the **genetics of vaccine response** see *Cell Rep* 2019; published online June 11. http://dx.doi.org/10.1016/j.celrep.2019.05.053

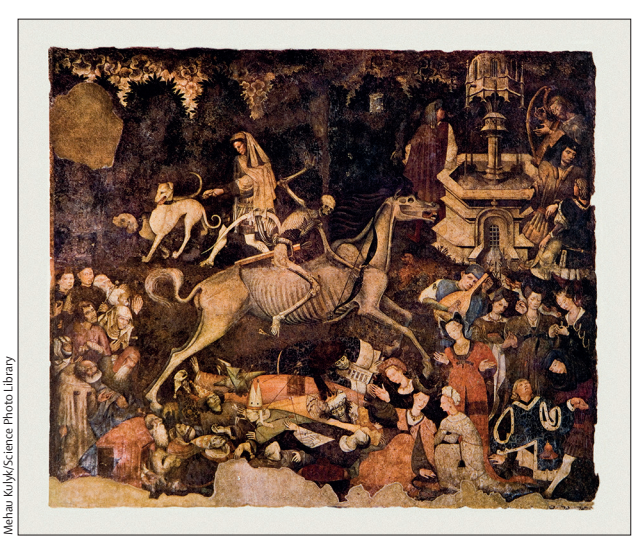
For more on a **transgenic fungus to kill mosquitoes** see *Science* 2019; **364:** 894–97

US comes up trumps

Findings from a study done in monkey cells and mice suggest that the American strain of Zika virus is more virulent than the other two subtypes: Pacific and southeast Asian. In monkey-derived Vero cells, the American subtype proliferated most (the southeast Asian type proliferated least). The American subtype also had the highest viral loads in the testes of mice (the southeast Asian type had the lowest) and, consequently, caused the most damage. The researchers posit that the virus acquired increased proliferative capacity and pathogenicity as it moved from southeast Asia through the Pacific islands to the Americas.

Slippery customers

Picornaviruses change shape to interact with host cells and can thus avoid targeting by drugs. It is why virologists have been unable to make antivirals against any of the subtypes that cause countless illnesses from upper respiratory tract infections to polio. A new drug-discovery study, though, might have identified a compound that wedges into a previously unknown indentation on the surface of the virus and prevents it from shape-shifting. The researchers think that the targeted structure is central enough to the core function of all picornaviruses that any resistance-conferring mutation would also make the viruses less viable.



Mehau Kulyk/Science Photo Library

***The Triumph of Death*, a fresco from Palazzo Scalfani in Palermo, Italy, painted in the 15th century as bubonic plague spread through Europe**

A model of vulnerability

A team of UK researchers has designed and tested a model that can assess the degree to which a country or region is vulnerable to outbreaks of zoonotic diseases such as Ebola virus disease and Lassa fever. The model incorporates a region's vulnerability in terms of the likelihood of impact (eg, number of cases) as well as its ability to cope with, or adapt to, an outbreak and turn the tide of an epidemic to reduce mortality or morbidity. They assessed their impact-*vs*-adaptive-capacity formula using data from epidemics of Lassa fever in Nigeria (2017–18) and Sierra Leone (2008–12), and of Ebola virus disease in Sierra Leone (2013–16).

Plague again

Rome wasn't built in a day. Nor, it seems, was the plague. Scientists have reconstructed eight distinct *Yersinia pestis* genomes sampled from human remains from 21 sites across Europe. For the first time, they found evidence that the first historically reported pandemic, the so-called Justinian plague of 541 CE, was present in the British Isles. They detected the same two virulence factors in the first and the second (14th to 18th century) epidemics, providing an example of potential convergent evolution of *Y pestis* during large-scale epidemics.

New antigens, new leads

An analysis of human plasma samples from more than 500 people with *Plasmodium falciparum* malaria from Cameroon, Burkina Faso, The Gambia, and Malawi has uncovered two new antigens on red blood cells infected with the parasite. The researchers showed that naturally produced antibodies against these antigens can target the parasite at two stages: the asexual reproductive stage and the subsequently matured gametocytes, the stage ingested and spread by mosquitoes. A vaccine that could stimulate such an immune response could thus block disease within an individual as well as its spread to others.

Personalised vaccines

Scientists have discovered genetic variants associated with differing levels of protection from vaccines in children. Researchers did a genome-wide association study of more than 3500 children in the UK and the Netherlands who had been given capsular group C meningococcal, *Haemophilus influenzae* type b, and tetanus toxoid vaccines. The associations need to be explored in a larger cohort, and once the level of response is untangled from genetic variation and other determinants of immunity persistence, such as age, sex, and ethnic origin, the findings might lead to personalised vaccination regimens that increase protection.

Spider *vs* malaria

A team of researchers from the USA and Burkina Faso have shown that a transgenic fungus can kill 99% of insecticide-resistant anopheline mosquitoes. The fungus *Metarhizium pingshaense* was engineered to produce a toxin from the venom of the Australian Blue Mountains funnel-web spider with a control switch so that the toxin is released within only mosquitoes so as not to cause harm to beneficial species such as honeybees. Still, given concerns around introducing transgenic organisms into the wild, the study was done within a screen-enclosed simulated village. In the face of mounting insecticide resistance, findings such as these should inform debate about the use of such technology to combat disease vectors.

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