Human malaria originated in gorillas

Human malaria, caused by the parasite *Plasmodium falciparum*, originated in gorillas and not in chimpanzees, as originally thought. A recent study examined faecal samples from chimpanzees, bonobos and eastern and western gorillas from sub-Saharan Africa to screen for parasite infection. The authors identified *Plasmodium* spp. infection in chimpanzees and western gorillas, but not in bonobos or eastern gorillas, and observed that chimpanzees and western gorillas were, in most cases, infected with mixed species of the parasite, three of which had not been previously described. Phylogenetic analysis using mitochondrial DNA showed that *P. falciparum*, the human parasite, is most closely related to the western gorilla parasites. When the gorilla *Plasmodium* spp. parasite entered the human population and whether cross-species infections still occur are important questions that now need to be addressed. ***Nature*/Nature News**

Methuselah SIV

Simian immunodeficiency virus (SIV) has been around for more than 32,000 years, according to a study published in *Science*. Although it was thought that SIV has been infecting African non-human primates for a very long time, recent DNA-sequencing studies estimated that the virus is only a few hundred years old. To resolve this discrepancy, the authors sampled monkeys on Bioko Island, Equatorial Guinea, which separated from Africa ~10,000 years ago, when sea levels rose. They observed that SIV was present in 22 out of 79 monkeys from six species, and they identified four previously uncharacterized SIV isolates. The four SIV isolates were found to be phylogenetically related to the SIV strains infecting the same genus of monkey in mainland Africa, suggesting that they share a common ancestor; in fact, computer-modelling studies estimated the time of the last common ancestor to be between 76,794 and 28,077 years ago, depending to the sequence compared (amino acid versus nucleotide). Therefore, the low pathogenicity of SIV has taken thousands of years to evolve, which indicates that a similar evolution of HIV might not be expected to occur in a short time span. ***Science***

Urban immunity

Living in cities could have led to the development of resistance to some infections, in a form of natural selection to survive the proliferation of disease caused by overcrowding. A recent study used DNA sequencing to examine 17 human populations in Europe, Asia and Africa for the prevalence of an allele of the immune system gene *SLC11A1* that is known to provide protection against intracellular pathogens such as *Mycobacterium tuberculosis* and *Mycobacterium leprae*. The authors then compared their findings to historical and archaeological data of the earliest urban human settlements in each area. They found that the protective gene variant is present in a high proportion of the population in areas of the Middle East and Europe, where cities have been established for thousands of years; by contrast, individuals from areas where urbanization has been more recent (for example, in Africa) were less likely to carry the gene variant. The correlation was still strong when the authors accounted for the prevalence of the allele owing to shared history (for example, through interbreeding). Ian Barnes, an author in this study, called this "an elegant example of evolution in action", and commented that the study highlights the importance of "the development of cities as a selective force". ***Evolution*/BBC**

HIV-1 recognition

A pathway by which dendritic cells (DCs) detect HIV-1 and subsequently activate an antiviral immune response was described in a study published in *Nature*. DCs detect microorganisms and present antigen to T cells to initiate an immune response; however, they are largely resistant to infection by HIV-1 and instead induce the infection of $CD4^+$ T cells through a process known as *trans*-enhancement. By infecting DCs with HIV-1 along with SIV virus-like particles containing viral protein X (VPX; which is expressed by HIV-2 and SIV but not by HIV-1), the authors managed to overcome this resistance and achieve productive infection and activation of an antiviral immune response. This response depended on the interaction of the newly synthesized HIV-1 capsid and the host protein cyclophilin A (also known as PPIA), which led to the induction of interferon regulatory factor 3 and to DC activation. Importantly, the infected, activated DCs could promote T cell activation and did not induce *trans*-enhancement of surrounding T cells. Further studies and a better understanding of this intrinsic recognition pathway could potentially be used for vaccine development. ***Nature***

Outbreak news

Chikungunya. In September, two 12-year-old girls were diagnosed with chikungunya fever in Fréjus, southeastern France. The infection is caused by the mosquito-borne chikungunya virus, which causes symptoms such as fever, arthritis and rash. In both cases, the infection was autochthonous — that is, the two girls had acquired the virus in their native area, as they had not travelled to regions where chikungunya is endemic. This, along with cases of dengue fever in the same region, has caused concern, as the mosquito vector for both viruses, *Aedes albopictus*, was recently discovered in the area. Health authorities have asked doctors to be vigilant to ensure detection of further cases, and efforts to eliminate the mosquito are underway. **Le Monde**

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 our Twitter page (@NatureRevMicro).

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