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Research in brief

Genetics and COVID-19
Scientists have identified a small group of rare structural gene variants found in people who are extremely ill with COVID-19 using optimal genome mapping of 52 patients. Seven such variants affecting 31 genes involved in innate immunity, inflammation, and other key pathways were found in nine of the sickest patients who shared common comorbidities such as diabetes and hypertension. The scientists say that more research is needed on the role of structural variants in host-virus interactions and that genetic variations would ideally be found with a blood assay.

Vimentin in SARS-CoV-2 entry
Extracellular vimentin, a structural protein widely expressed in endothelial cells in the vascular system, enables SARS-CoV-2 entry into human cells say researchers who found that knockdown of vimentin significantly reduced SARS-CoV-2 infection of human endothelial cells, whereas overexpression of vimentin with ACE2, the entry receptor for SARS-CoV-2, significantly increased the infection rate. The researchers also saw that the CR3022 antibody inhibited the binding of vimentin with the spike protein and neutralised SARS-CoV-2 entry into human cells.

Which mRNA vaccine is best?
There were fewer breakthrough SARS-CoV-2 infections and a lower risk of hospitalisation among those who received Moderna’s mRNA-1273 vaccine than among those who received Pfizer-BioNTech’s BNT162b2 vaccine, according to an analysis of the electronic health records of more than 637,000 fully vaccinated patients in the USA when the delta variant (B.1.617.2) was predominant. Incidents of breakthrough infections were included if the individual had not been previously infected with SARS-CoV-2 and had not received a booster dose. No significant difference was observed in mortality rates.

COVID-19 mortality in children
Sub-Saharan African children admitted to 25 hospitals with COVID-19 between March and December, 2020, were more likely to die than their peers in the USA and Europe. A study examined outcomes in 469 children aged between 3 months and 19 years who were hospitalised in the DR Congo, Ghana, Kenya, Nigeria, South Africa, or Uganda. Infants younger than 1 year had nearly five times the risk of death than adolescents aged 15–19 years. Children of all ages with comorbidities including high blood pressure, chronic lung diseases, haematological disorders, and cancer were also at higher risk of dying. According to the study’s lead author, the situation has changed little for children in Africa since the study was done.

Targeting HIV latency
Pembrolizumab, a cancer immunotherapy, was found to reverse HIV latency, which is the ability of the virus to hide in the cells of people receiving antiretroviral therapy. In a prospective clinical trial of pembrolizumab in people with cancer living with HIV, pembrolizumab was able to perturb the HIV reservoir in 32 people. Pembrolizumab is a monoclonal antibody that works by blocking PD-1 that is expressed by exhausted T cells. The researchers have approval for a clinical trial, which has been put on hold due to the COVID-19 pandemic, to understand how anti-PD-1 treatment works in people without cancer and to look at the effects of anti-PD-1 treatment in both the lymph nodes and the blood to try to find the safest dose.

EBV in multiple sclerosis
A study provides compelling evidence that multiple sclerosis is likely caused by infection with Epstein-Barr virus (EBV). Researchers did a study among more than 10 million young adults on active duty in the US military and identified 955 who were diagnosed with multiple sclerosis during their period of service. The team analysed serum samples taken biennially by the military. The risk of multiple sclerosis increased 32-fold after infection with EBV but was unchanged after infection with other viruses. Serum levels of neurofilament light chain, a biomarker of the nerve degeneration typical in multiple sclerosis, increased only after EBV infection. An EBV vaccine or targeting the virus with EBV-specific antiviral drugs could ultimately prevent or cure multiple sclerosis said the study’s senior author.

Gene editing in malaria
Researchers using gene-editing technology to delete the gene encoding CTL4 from Anopheles gambiae mosquitoes made them highly resistant to the malaria parasite and led to a big decrease in infection prevalence. The study’s researchers allowed no-CTL4 and intact-CTL4 mosquitoes to feed on human blood samples laced with Plasmodium falciparum parasites. When the concentration of parasites in the blood meal was low, mimicking typical conditions in the wild, only 19.7% of the no-CTL4 mosquitoes were infected, compared with 61.3% of the control mosquitoes. When the concentration of parasites in the blood meal was high, only 45.0% of the no-CTL4 mosquitoes harbour the parasites, compared with 97.3% of the intact-CTL4 mosquitoes. Malaria transmission modelling studies suggest this would translate into near-complete prevention of local mosquito-to-human transmission, said the senior author.

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