Crop Switching for Improved Agricultural Sustainability in China

In a study published in *Nature*, Dr. WU Feng from the CAS Institute of Geographic Sciences and Natural Resources Research together with his collaborators demonstrated that strategic crop switching could notably improve environmental sustainability and boost farmer incomes in China (10.1038/s41586-023-05799-x). Using a crop-switching model, the team simulated scenarios to minimize resource use and environmental impacts while maximizing farmer incomes. The results confirmed the effectiveness of crop switching in combating environmental degradation and enhancing livelihoods, highlighting its potential to contribute to China's agricultural sustainable development targets. The study also emphasized the need for careful planning, policy support, and consideration of regional differences in implementing crop switching strategies. Despite these challenges, the research provides a promising roadmap for achieving China's sustainability goals while improving farmer incomes.

Illustration of a Chinese farmland during the transition phase of crop-switching. (Image created by Midjourney)

New MERS-like Coronavirus Discovered in Pangolins

A research team jointly led by Dr. SHI Zhengli and Dr. ZHOU Peng from the CAS Wuhan Institute of Virology and their collaborators have discovered a novel MERS-like coronavirus in Malayan pangolins, the world's most trafficked mammals (doi: 10.1016/j.cell.2023.01.019).

A Philippine Pangolin pup nudges its mother, rolled up in a protective ball. Photographed by Shukran888 (CC BY-SA 4.0) https://commons.wikimedia.org/w/index.php?curid=80616668

The virus, named *Manis javanica* HKU4-related coronavirus (MjHKU4r-CoV), was found in 11% of the tested animals by pan-CoV PCR and 12.8% tested seropositive, according to a new study published in *Cell* on February 16.

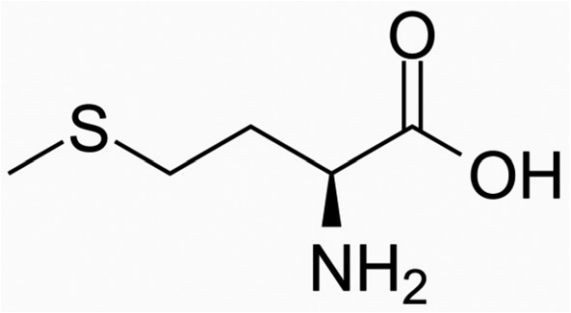
The MjHKU4r-CoV-1 strain uses human dipeptidyl peptidase-4 (hDPP4) as a receptor and host proteases for cell infection. Notably, the virus has a furin cleavage site –many viruses, including SARS-CoV-2 virus, use

furin to process their surface proteins and gain entry into host cells – and binds effectively to hDPP4 and have a broader host range. The study shows that MjHKU4r-CoV-1 is infectious and pathogenic in human airways and intestinal organs and in hDPP4-transgenic mice.

This finding highlights the importance of pangolins as a reservoir host of coronaviruses with the potential to emerge as human diseases. Public safety may be impacted by the potential for zoonotic transmission of bat coronaviruses through pangolins, emphasizing the need for continued monitoring and research into these animals and their role in virus transmission.

A Small Amino Acid, Big Impact: Methionine Restriction in Cancer Therapy

A recent study published in *Gut* (doi:10.1136/gutjnl-2022-326928) reveals a novel potential strategy for cancer immunotherapy: a methionine-restricted diet (MRD). A research team, led by Dr. JU Huaiqiang and Dr. XU Ruihua from the Sun Yat-sen University Cancer Center, and Dr. PIAO Hailong from the CAS Dalian Institute of Chemical Physics (DICP), discovered that a diet deficient in methionine, an essential amino acid, can reduce tumor growth and boost antitumor immunity. This is achieved by increasing the number and cytotoxicity of tumor-infiltrating CD^{8+} T cells, a type of immune cell that plays a crucial role in fighting cancer.



Methionine, an essential amino acid in humans, after converting into S-Adenosylmethionine (SAM) acts as the methyl group donor in DNA or RNA methylation, a process that has given rise to the science of epigenetics.

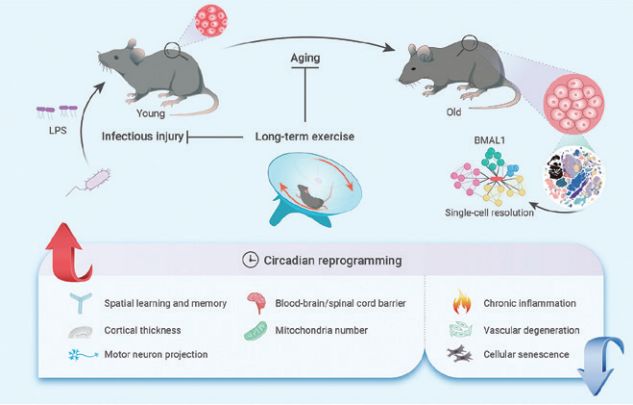
In addition to the diet, the researchers also identified a critical player in anticancer immunity: a protein called YTHDF1. High expression of YTHDF1 was found to correlate with poor prognosis and less effective immunotherapy outcomes for cancer patients. The study suggests that either a methionine-restricted diet or YTHDF1 depletion can inhibit tumor growth and synergize with PD-1 blockade, a type of immunotherapy, for better tumor control.

"Methionine and YTHDF1 play a critical role in anticancer immunity through regulating the functions of T cells," the researchers explained. "Targeting methionine metabolism or YTHDF1 could be a potential new strategy for cancer immunotherapy."

Exercise: The Fountain of Youth at a Cellular Level

On January 5, 2023, a new study in *The Innovation* reveals the profound impact of exercise on the body at a cellular level (doi:10.1016/j.xinn.2023.100380). The study, entitled "A single-cell transcriptomic atlas of exercise-induced anti-inflammatory and geroprotective effects across the body," offers a fresh view on the well-known saying, "Exercise is good for you."

The team used young and old mice undergoing a year-long exercise program. They analyzed the changes across 14 different tissues/organs at a single-cell level. The findings were impressive. Exercise protected tissues



An atlas of age-, tissue-, and cell-type-specific benefits of long-term exercise. (Credit: *The Innovation*)

from infection, with younger animals showing more pronounced effects. Older subjects also reaped benefits, with a significant decrease in inflammaging (chronic low-grade inflammation linked with aging) and signs of tissue rejuvenation. The most notable improvements were in the central nervous system and systemic vasculature.

The study also found that exercise helps reset circadian rhythms through the circadian clock protein BMAL1, delaying aging and aiding recovery from infectious damage. This highlights the role of exercise in restoring the youthful circadian clock network, paving the way for further research into the relationship between exercise, aging, and immune responses across the entire organism.

Stealthy Invaders: How SARS-CoV-2 Evades Our Defenses and Spreads

On January 6, 2023, a recent *Cell Discovery* study, jointly spearheaded by scientists from the CAS Shanghai Institute of Materia Medica, Wuhan Institute of Virology and Kunming Institute of Zoology, reveals a new way that the SARS-CoV-2 virus, responsible for the COVID-19 pandemic, evades our immune system and spreads. The virus uses a mechanism involving extracellular vesicles (EVs) to transmit itself from cell to cell, effectively hiding from neutralizing antibodies.

The researchers found that the SARS-CoV-2 virus induces the formation of EVs in infected cells. These EVs, which are much larger than previously reported virus-generated vesicles, contain large amounts of live virus particles. The virus uses these vesicles as a Trojan horse, hiding inside them to escape detection by neutralizing antibodies. Once inside a new cell, the virus can then establish an infection, all the while remaining hidden from the immune system. It's like the virus is using a stealth bomber to get past our defenses. This discovery provides a new perspective on how the virus resists current antibody therapies and vaccines, and it could potentially guide the development of future antiviral treatments.

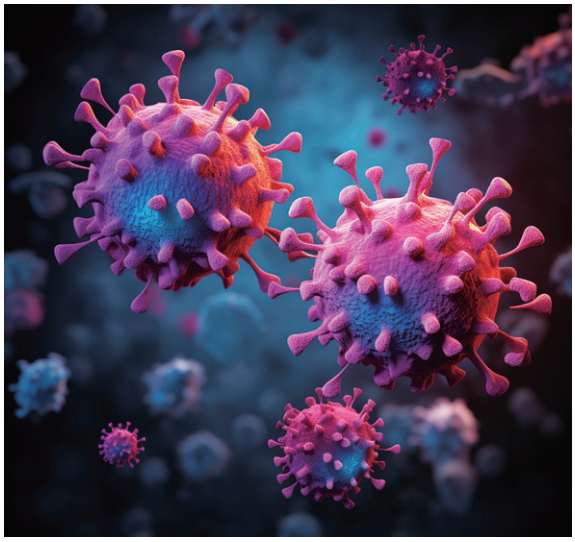
How SARS-CoV-2 Hijacks Our Immune Cells

In a study published in the journal *Signal Transduction and Targeted Therapy* on February 27, a team of scientists led by Dr. CHEN Jianfeng from the CAS Shanghai Institute of Biochemistry and Cell Biology and Dr. JIANG Shibo from Fudan University has discovered a new mechanism by which the SARS-CoV-2 virus, responsible for COVID-19, infiltrates and dysregulates our immune cells (doi: 10.1038/s41392-023-01348-0). The researchers found that the virus uses certain proteins, known as integrins, on the surface of T cells to gain entry and disrupt their normal functioning.

The study reveals that the virus's spike protein, which it uses to bind to cells, has three potential integrin-binding motifs. These motifs allow the virus to latch

onto the integrins on T cells, a type of white blood cell crucial for immune responses. Once inside, the virus causes the T cells to become hyperactivated, leading to an overproduction of proinflammatory cytokines, a condition known as a cytokine storm, often seen in severe COVID-19 cases.

Given that Integrins act as SARS-CoV-2 receptors on T cells and mediate entry and dysregulation of T cells by SARS-CoV-2, blocking this interaction may be a strategy for preventing SARS-CoV-2 entry into T cells and associated immune dysregulation in COVID-19 patients. This discovery provides a new perspective on the virus's ability to infect various tissues and cells, including those with low expression of the previously identified SARS-CoV-2 receptor, ACE2. It also sheds light on the mechanisms behind the severe immune response seen in some COVID-19 patients.



New study shows that SARS-Cov-2 uses certain proteins, known as integrins, on the surface of T cells to gain entry and disrupt their normal functioning. (Credit: *Pixabay*)