

BIFI-Talks 2021

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Networks Virology. Learning from SARS-CoV-2

Viruses are reproductive machineries formed mainly by genetic material (RNA or DNA) and proteins. In general, these proteins interact with specific proteins of the host forming vast networks of virus-host protein-protein interactions (PPI). Although a virus may infect mainly an organ or system of the host, the damages can propagate beyond it, transforming the affection into a multiorganic/multifunctional one. This is the case of SARS-CoV-2, which makes COVID-19 a multiorganic disease affecting a dozen of organs/systems in humans. Here, I develop the hypothesis based on facts that this propagation of extrapulmonary damages takes place via the PPI network of virus-host interactions. It considers that some viral proteins interact with specific human proteins highly expressed in the lungs. Then, these perturbed human proteins navigate outside the lungs via an exosomemediated transport network, which allows the inter-organ cross-talk. Due to the fact that these "perturbated" proteins interact with their partners in other organs they are capable to transmit such perturbations beyond the lungs. Therefore, there is a network of perturbators from the lungs interacting with a network of vulnerable proteins in other organs, which trigger a range of damages in them. We identify here the perturbators and vulnerable proteins in COVID-19, identifying 13 organs/systems that may be affected by the infection of SARS-CoV-2 and explain some of the extrapulmonary damages observed in clinics. We propose a series of drugs that can be repurposed to treat combinations of these damages in COVID-19 patients. Finally, at a lower size-scale we zoom on these SARS-CoV-2 proteins to investigate their topological structure, which form other networks of amino-acids interacting noncovalently among them. Using these techniques we are able to identify weak pints in such proteins that can be used as pharmacological targets.

DIA Y HORA: 16 DE ABRIL A LAS 12:30

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