Metadichol and Corona Virus

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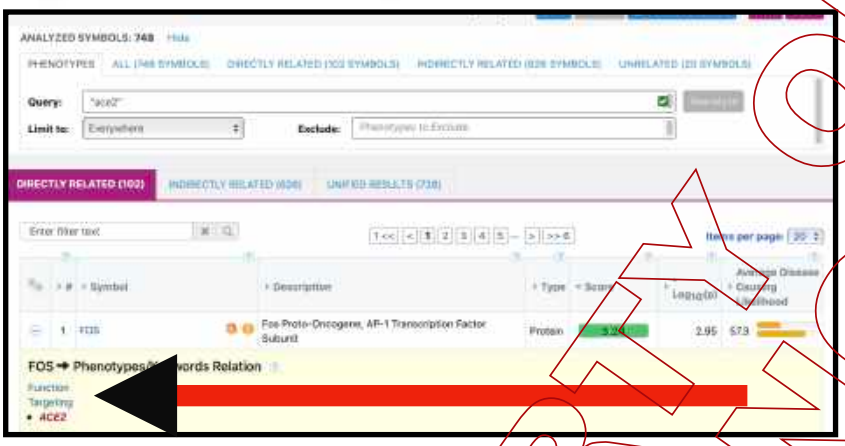
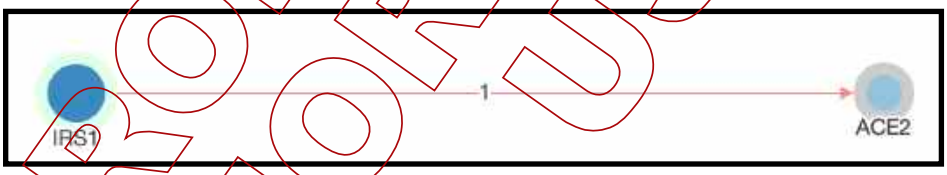


Metadichol expresses FOS gene that targets ACE2. Also IRS1 and IRS2 are over-expressed by Metadichol at picogram levels with stem cells. IRS1 directly controls ACE2 intact Metadichol expresses over 102 genes that can target ACE2.
With SARS (sudden acute respiratory syndrome), another coronavirus, researchers discovered that one of the ways the disease attaches itself is through an enzyme known as ACE2, a'functional receptor' produced in several organs (oral and nasal mucosa, nasopharynx, lung, stomach, small intestine, colon, skin, lymph nodes, thymus, bone marrow, spleen, liver, kidney, and brain). ACE2 is also "abundantly present in humans in the epithelia of the lung and small intestine, which might provide possible routes of entry for the SARS-CoV," while it was also observed "in arterial and venous endothelial cells and arterial smooth muscle cells" – which would include the heart. Evidence of both sudden collapses and neurological damage from footage pouring out of Wuhan, China




Journal of
Stem Cell Research & Therapy
Umbilical Cord Cells Treatment with Metadichol® IRS Proteins and GLUT4 Expression and Implications for Diabetes

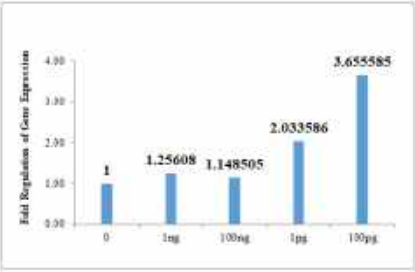
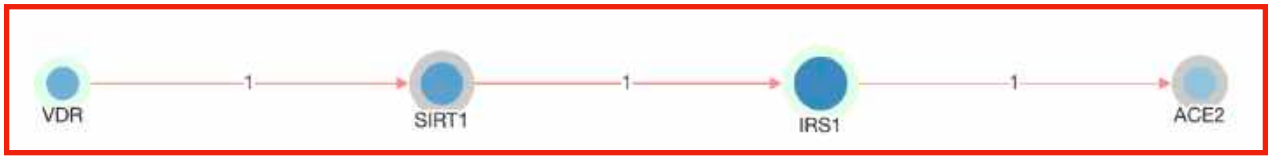
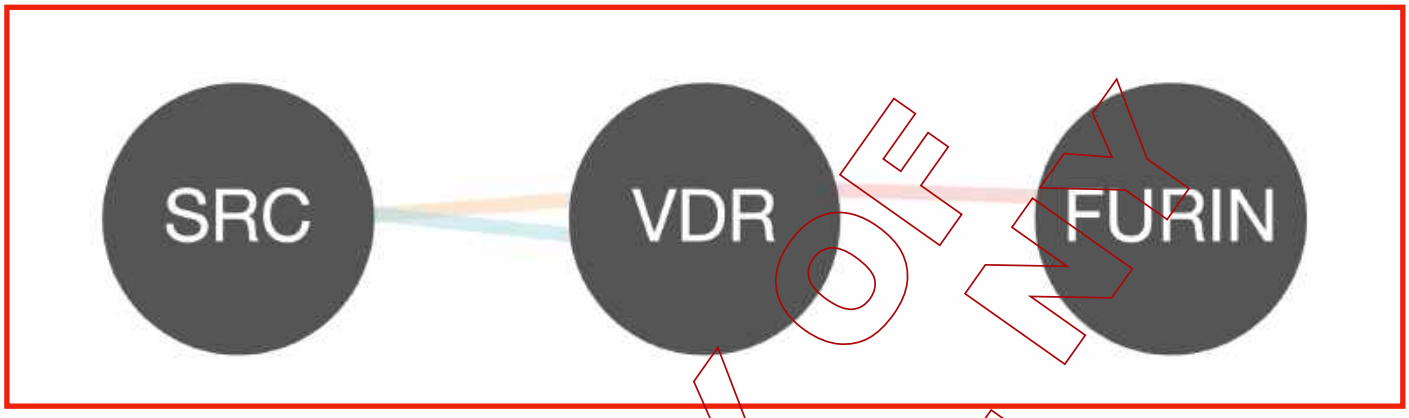


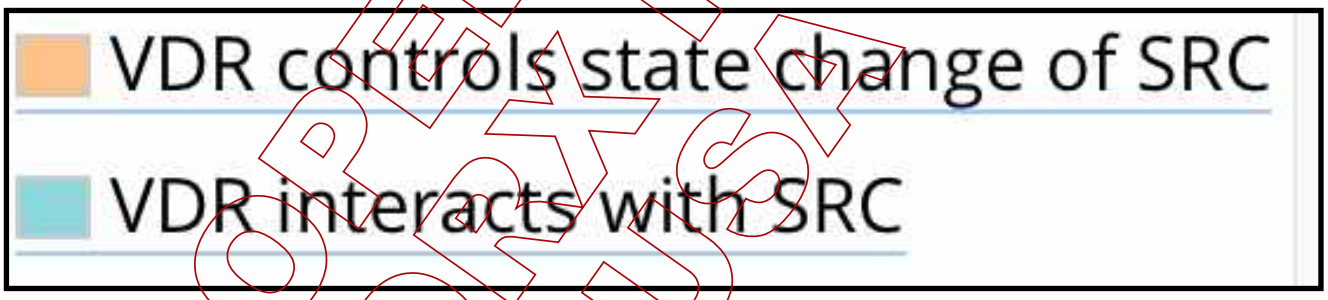
Figure 5: Relative expression of IRS1 gene in untreated and treated umbilical cord cells.





VDR controls expression of FURIN

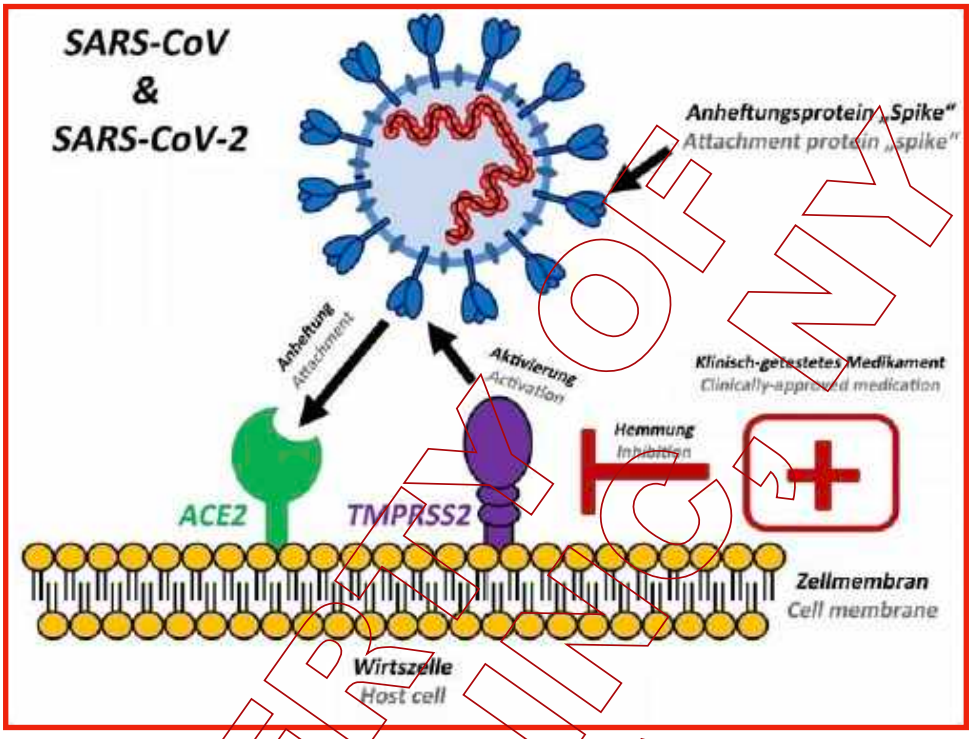
Data Sources
Molecular Signatures Database



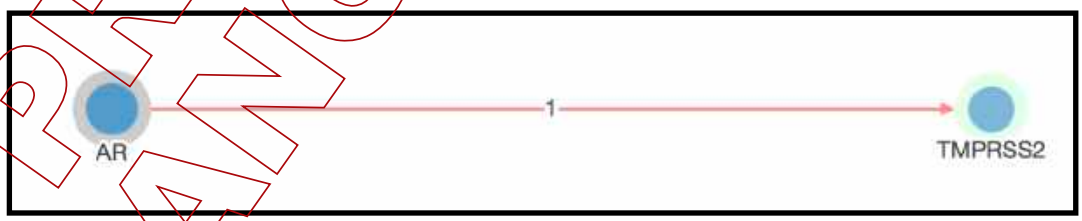
The virus uses the outreaching spike protein to hook on to the host cell, but normally this protein is inactive. The cleavage site structure's job is to trick the human furin protein, so it will cut and activate the spike protein. The activation causes a "direct fusion" of the viral and cellular membranes. Compared to the Sars virus's way of entry, this binding method is "100 to 1,000 times" as efficient, according d by Professor Li Hua from Huazhong University of Science and Technology in Wuhan, Hubei province, . Drugs targeting the furin enzyme, including HIV medicines, could have the potential to hinder the virus's replication in the human body,

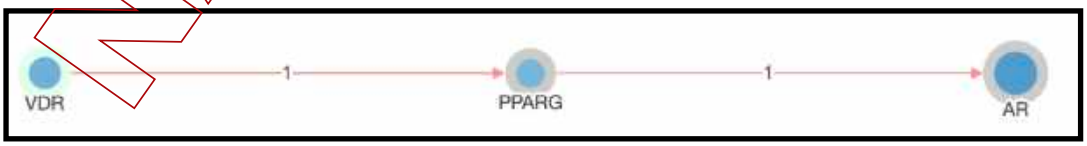
The attachment protein "spike" of the new coronavirus SARS-CoV-2 uses the same cellular attachment factor (ACE2) as SARS-CoV and uses the cellular protease TMPRSS2 for its activation. Existing, clinically approved drugs directed against TMPRSS2 inhibit SARS-CoV-2 infection of lung cells. A cellular protein called TMPRSS2, is crucial for the entry of the novel coronavirus into lung cells. The

results of the study show that the virus needs the protease, which is present in the body, to enter the cells, providing a new target for therapeutics.



Hoffmann, M et al. (2020). SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically-proven protease inhibitor. Cell, https://marlin-prod.literatumonline.com/pb-assets/journals/research/cell/CELL_S0092-8674%2820%2930229-4.pdf





Metadichol® can regulate ACE 2 through its expression of IRS1. In Addition VDR binding to SRC leads to regualtion of FURIN And Finally VDR controls expression of PPARG which controsl AR another Nuclear receptor that leads to TMPRSS2 and thus all elements needed are presnt to onhibit the Corona Virus by Metadichol whcih may trun out to be a safe novel agent to control the Corona virus.