Wearing face masks do no harm

The principal mode of SARS-CoV-2 transmission involves viral entry through the respiratory tract. This generally occurs when an infected individual coughs, sneezes, or speaks, generating aerosols carrying the virus. At the onset of the pandemic, there was rudimentary knowledge about the virus, and policies were developed based on the best available evidence. Thus, guidelines regarding wearing face masks differed among countries and over time. At present, scientists are convinced that masks are paramount in reducing viral spread.

As the pandemic ravages the world relentlessly, there has been a setback in government measures to contain the virus globally. A large number of protestors have taken to the streets against the compulsory use of face masks. They have condemned mandatory mask-wearing as *"an infringement of freedom"*, *"violation of religious rights"*, or a part of a broader conspiracy that *"COVID-19 is a hoax"*. Some have proclaimed that mask-wearing engenders*"more harm than good"* due to rebreathing CO_2, with some even considering that *"masks are lethal."*

Researchers at Florida recruited 15 house-staff physicians without any pulmonary conditions and 15 patients with COPD. They monitored their $EtCO_2$ and SpO_2 throughout the time they were wearing surgical masks, and arterial blood gases before and after a 6-minute walking test with masks on. They found that gas exchange was not significantly affected by the use of masks, even in those with severe lung impairment. They feel that dyspnea experienced by some people wearing masks does not stem from hypercarbia or hypoxemia; rather, it occurs from a restriction of airflow, especially when higher ventilation is needed on exertion.

This research, although small, dispels some misconceptions surrounding the use of face masks in the context of the surging COVID-19 pandemic.
(Annals of the American Thoracic Society 2 October 2020)

Classical music improves epilepsy: The Mozart effect

Epilepsy is a common disorder, affecting approximately 0.64% of the world population, resulting in substantial neurologic, cognitive, psychological, and social consequences. Treatment frequently involves multi-drug therapy, but seizures persist in about 30% of them. Among the non-pharmacological approaches for drug-resistant epilepsy, there is an evolving interest in non-invasive forms of neurostimulation such as music therapy. A recent meta-analysis has shown that listening to Mozart's piano music can reduce the frequency of epilepsy.

The music compositions of Wolfgang Amadeus Mozart date way back to the 18th century. It was in 1993 that Francis Rauscher claimed that listening to Mozart's music improved the spatiotemporal senses of normal subjects. However, clinicians have always treated the 'Mozart Effect' with some scepticism.

More recently, two Italian researchers, Gianluca Sesso and Federico Sicca, systematically reviewed existing research works and found that listening to Mozart led to a remarkable decrease in epileptic seizures (31%–66%) and interictal epileptiform discharges. These effects occurred after a single music session and were sustained after a prolonged treatment duration. Sonatas for two pianos, K448 and K545, had an exceptionally positive effect. The researchers believe that Mozart's sonatas might have distinctive rhythmic structures specifically suited to working on epilepsy.

The highly congruous results of this meta-analysis firmly imply that Mozart's music could be an effective non-invasive method for improving clinical outcomes in epilepsy, especially in difficult-to-treat ones. Nevertheless, the exact mechanisms of the Mozart effect on the brain should be understood to use this method in clinical settings.
(European College of Neuropsychopharmacology Congress September 2020, Clinical Neurophysiology April 2020)

SARS-CoV-2 spike protein allays pain

A group of researchers based at the University of Arizona Health Sciences have reported that SARS-CoV-2 spike protein could relieve pain.

Numerous biological pathways signal the human body to perceive pain, one among which is the vascular endothelial growth factor-A (VEGF-A)/neuropilin signaling pathway. VEGF-A binds to the receptor neuropilin and kicks off a cascade of events causing neuronal hyperexcitability and resulting in pain.

Neuropilin-1 is the second receptor proposed for SARS-CoV-2 in some studies; angiotensin converting enzyme-2 being the first. The research team found that the SARS-CoV-2 spike protein binds to neuropilin at the same location as VEGF-A, thereby hindering VEGF-A from binding to it. In a series of experiments in the laboratory and in rodent models, spike protein reversed VEGF-A induced pain signaling.

This research finding could perhaps explain how SARS-CoV-2 reduces pain in some patients and stays under the radar. According to the U.S. Centers for Disease Control and Prevention, 50% of COVID-19 transmission occurs before symptom onset and 40% of infections are asymptomatic. This research also paves the way to explore a novel class of non-opioid therapeutics for pain targeting the VEGF-A/neuropilin pathway that would reduce opioid abuse.

This finding could have important implications at a time we arewaging a war against the COVID-19 pandemic and the opioid epidemic.
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JERIN SEKHAR AND GOURI RAO PASSI*
*gouripassi@gmail.com