On the Purported Efficacy and Safety of Face Mask Use to Stop Transmission of Covid-19

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INTRODUCTION

With increasing SARS-CoV-2 infection numbers across the globe, fear of a second wave of Covid-19 – the disease resulting from significant SARS-CoV-2 infection[1] – has produced yet another unique change in human behavior to attempt to combat this seemingly omnipresent threat: face mask usage. This subject has become the latest addition to the large pile of divisive subjects thrust into Western consciousness, furthering highly irrational public discourse. Unsurprisingly, individual opinion on the face mask issue coincides with the individual's political leaning[2], which is, of course, absurd. The truth of a matter is not subject to political affiliation. Despite this glaring reality, large swaths of lay persons, doctors, and scientists alike parrot with fervent devotion the latest television-intoned mantra of choice from the most appealing cult of personality of choice to whom they have surrendered their intellectual assent. This functional illiteracy manifests in the echoing of platitudes along the lines of, "I believe in science; therefore, I choose (insert desired action here)." Through application of one's intellect to the subject, one can avoid such dangers and transmute his or her blind faith in "scientism" into comprehension. Despite the superabundance of information in our world there is a corresponding scarcity of understanding. At the same time, however, it exposes a major flaw in modern science: one can find literature to support opposing positions on a great many subjects. To overcome this obstacle and be able to discern the correct position on any subject, one must come to understand the underlying principles at play. The goal of this work is to clarify important principles involved in face masking to halt SARS-CoV-2 transmission.

WHO TRANSMITS AND WHO IS SICKENED BY SARS-CoV-2?

Within our current face mask-mandate climate, the assumption is that anyone at any time is merely asymptomatic and thus everyone must wear a face mask. It is important to differentiate between a- and presymptomatic individuals. The words themselves offer a superficial understanding: asymptomatic individuals are without symptoms and will not develop them – will not get sick, and presymptomatic individuals are temporarily without symptoms but will eventually develop them – will eventually get sick. There is a distinct biological difference between a- and presymptomatic individuals. The asymptomatic individual exhibits a robust immune system response while the presymptomatic's flounders. The former has some immunological disposition which renders him/her capable of fighting off either a great many pathogens in general or SARS-CoV-2 in particular, and the latter lacks this capability. Yes, the presymptomatic was at one time asymptomatic, and while the difference between the two may be imperceptible to the human eye before the presymptomatic turns symptomatic, the invisible immunological difference exists. The distinction between the two cannot be made using the prevailing

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reverse transcription polymerase chain reaction (RT-PCR) detection methods[3]; indeed, the distinction between virulent and obliterated virus **cannot be made by this method**. RT-PCR detects minute fragments of viral RNA which must be amplified by orders of magnitude before its presence can be confirmed. Because the detected RNA fragment can derive from actively replicating viruses and destroyed viruses alike, a positive RT-PCR result alone is not evidence of an active infection. It is a distinct possibility, therefore, that asymptomatic individuals are testing positive for SARS-CoV-2 RNA and a conquered infection and thus are not spreading virions. It is possible that this is the case for a great many, if not all, asymptomatic individuals. This highly plausible scenario has been unexamined with scientific rigor despite the urgent need for it.

Early in the life of Covid-19, several reports were rapidly published detailing evidence of SARS-CoV-2 human-to-human transmission[4,5] and infection in a- and presymptomatic individuals[6–12]. Subsequent studies interrogated the potentiality of SARS-CoV-2 transmission from individuals displaying no symptoms with quick, small cohort analyses which were rapid-fired into public consciousness[13–22]. Of the studies which claim to provide evidence of SARS-CoV-2 transmission from asymptomatic carriers, Li et al[5], Zhang et al[14], Rothe et al[16], and Ye et al[21] have very limited cohort sizes (7, 5, 5, and 5 respectively) and mistakenly use the term asymptomatic when the appropriate term is presymptomatic. Li et al likewise had a small cohort (5) and detailed a supposed asymptomatic spreader, though this individual did not test positive for SARS-CoV-2 by RT-PCR until well after others in the cohort developed symptoms[19], the individual was assumed to be the spreader as he had traveled to Wuhan though the testing suggests he was infected by his family, not the other way around. Hu et al examined a somewhat larger cohort (24) and revealed 1 potential asymptomatic spreader[17]; however, this study suffered from a confounder which the others also share – the possibility of the infections coming from alternate, unknown sources cannot be eliminated[23]. A contract tracing study was recently performed to better address the question of SARS-CoV-2 transmission from a true asymptomatic[24]. This study traced an asymptomatic individual, positive for SARS-CoV-2 by RT-PCR, to 455 contacts and assessed these 455 for SARS-CoV-2. All contacts tested negative for the infection. The aforementioned inability of RT-PCR to distinguish between RNA from live and dead virus renders virologic evidence of asymptomatic transmission dubious[25–27], particularly in the case of an infant with high viral load who remained asymptomatic throughout hospitalization[28]. Moreover, all analyzed specimens collected from potential cases are extracted from oropharyngeal or nasopharyngeal swabs and not being actively expelled from them. The experiment required to prove asymptomatic transmission would involve isolating asymptomatic carriers with negative testers and assessment of the extent to which the virus infects the negative testers. This is, of course, a highly unethical experiment and will not be performed. There is no evidence to suggest SARS-CoV-2 spreads from asymptomatic infections, which is analogous to what is known about influenza[29].

A significant portion of the human population is already immune to Covid-19. Several examinations of antibodies against SARS-CoV-2 in populations have been conducted to determine the extent of SARS-CoV-2 spread[30–35]. These antibody studies have multiple problems: the lifetime of circulating antibodies against SARS-CoV-2 can wain within weeks[36], which appears to be different than the lifetime of IgG antibodies against the original severe acute respiratory syndrome-causing virus (SARS-CoV) which can persist for at least 12 years after infection[37], so infected individuals may be producing antibodies against SARS-CoV-2 antigens other than the antibody being tested for; despite a recent bioinformatic assessment of the antigenic dominance (85.3-90.9%) of the SARS-CoV-2 spike protein[38], actual antigen measurements from SARS-CoV-2 exposed individuals revealed only 27% T cell response to spike protein antigens, indicating T

cell reactivity to several other SARS-CoV-2 protein epitopes[39]; additionally, a preponderance of evidence from work on coronavirus outbreaks of recent memory, SARS and MERS, suggests immunity to these coronaviruses derives from $CD4^+$ and $CD8^+$ memory T cells[40–45], implying antibodies are not necessarily what confers immunity to coronaviruses and, therefore, serological antibody tests do not provide an accurate image of one's immune posture against SARS-CoV-2 infection. Congruent to the SARS and MERS literature, recent excellent work has demonstrated SARS-CoV-2-reactive T cell mechanisms to be invaluable to immunity to Covid-19[39,46–48]. These studies further revealed an intriguing discovery: healthy, SARS-CoV-2 unexposed individuals displayed T cell reactivity to SARS-CoV-2. Indeed, Grifoni et al[39] found $CD4^+$ T cells from 40-60% of blood donations from years previous to the SARS-CoV-2 pandemic to be reactive to SARS-CoV-2 antigens, Braun et al[47] discovered these cells in 34% of healthy donors, both likely from immunological cross-reactivity of coronaviruses responsible for the common cold and SARS-CoV-2, and Nelde et al[49] discovered these T-cell responses in 81% of unexposed individuals again citing heterologous immunity from exposure to circulating common cold coronaviruses. This makes perfect sense considering the large homology between common cold coronaviruses and SARS-CoV-2 protein amino acid sequences[50]. These findings indicate an existing immunity to Covid-19 and a relatively small leap to be made to achieve[51], or to have already achieved[52–54], the natural herd immunity threshold.

Those at risk of serious illness after SARS-CoV-2 infection are overwhelmingly of older age (65+) and/or possess comorbid health complications like hypertension, diabetes, cardiovascular disease, or respiratory diseases[55–58]. There is nothing to suggest that younger individuals in good health are more likely to succumb to the ravages of SARS-CoV-2 infection than any other virus which circles the globe every other year. A recent study, conducted in the pre-face mask era, revealed school aged children and their teachers had very low anti-SARS-CoV-2 IgG seroprevalence of 0.6%[59], even amongst 23 of 24 participants who live with a household member who previously tested positive for SARS-CoV-2, suggesting extremely low prevalence of the virus in this population. Further, studies have now found no child-to-child[60] or child-to-adult[61] transmission of SARS-CoV-2. This demonstrates that our youth are not in imminent danger, nor are their teachers, from SARS-CoV-2. Schools should no longer be closed; our youth are a bulwark against Covid-19. Indeed, school closure was ineffective at slowing the spread of this virus[62] as children and teachers do not significantly contribute to Covid-19 transmission[63].

CAN A FACE MASK STOP THE SPREAD OF COVID-19?

Though SARS-CoV-2 may infect the body through the fecal-oral route[64,65] and fomite contact[66,67], aerosol transmission appears to be the primary mechanism for its transmission[68,69]. From this follows the theory behind face mask use: blocking or slowing virus-laden airflow from an individual will protect his or her fellow citizens as well as serve to protect the individual form virus-laden airflow originating from his or her fellow citizens. However, like other superficial theories emanating from the biomedical world, this theory crumbles when examined with any diligence beyond trusting a perfunctory platitude from "experts" on the television. The face mask question has been considered for decades and has been assessed in diverse arenas by sundry paradigms. The actual science is unequivocal: face masks do nothing to stop the spread of any type of infection. Results from studies performed in operating rooms[70–74], amongst clusters of healthcare professionals[75–77], and in broader communities[78,79] all report the same finding: face masks afford no protection against bacterial and viral infection. Several literature reviews further support the inability of face masks to stop these pathogen classes[80–83] or highlight the lack of evidence to suggest they do[84–89]. The reason for a face mask's inability to stop viral transmission is, quite simply, it cannot physically contain virus-laden aerosol particulates.

Viruses can penetrate face masks of all classes. N95 respirators, which are designed to filter 95% of aerosolized contaminants, do not live up to their name when confronted with viral assault[90], and afford no superior protection against respiratory illness and viral infection compared to surgical masks[91,92] which does not protect one against viral infection. Regarding homemade cloth masks, one randomized trial[76] found them to actually increase the rate of influenza-like illness – cloth mask use during influenza season may be particularly dangerous. The filtering efficiency of cloth masks is, of course, material-dependent but is generally quite low[76,93,94] and, like all face mask classes, relies on proper use. Proper use is hit-or-miss, to say the least, as mask wearing causes a number of annoyances to its wearer[95], particularly skin irritation and damage[96,97]. A study of surgical masks by Oberg et al[98] revealed all subjects failed an unassisted mask fit test and all but two failed after assistance. This same study further examined filtering efficiencies of surgical masks and found no surgical mask to provide adequate filtration efficiency to be viable protection against respiratory infection[98]. This is because a large fraction of virus-containing aerosols is sub-micron in size[99]. Yang et al[100] assessed aerosol particulates of varying sizes for their influenza A genomic content and discovered 64% of influenza A genome copies were present in aerosols less than 2.5 microns in size. This implies virus-harboring aerosols come in sizes small enough to readily penetrate surgical masks, cloth masks, and n95 respirators alike[90,93,98]. These small aerosols can remain in the indoor air for hours after exhalation[100] and can contain 10^3 to 10^7 viruses[101]. The number of viruses needed to infect a tissue culture 50% of the time ($TCID_{50}$) is between 100 and 1000 and the human infective dose is estimated to be between 2 to 3 times $TCID_{50}$[102–104]. This implies one inhaled virus-laden aerosol harbors well beyond the minimum infective dose to sicken a human[105]. This alone renders face masks useless. Further, it needs to be understood that most of the air exchange between the environment and the mask-wearer's lungs will not occur through the mask, but rather around it. One of the common inconveniences mask wearers experience is misting of glasses[95]. This phenomenon is caused by exhaled warm, moist air being deflected out above the mask and condensing on the glasses. This air likewise escapes below and out the sides of the mask. As the viral aerosols do not possess any face-mask homing mechanisms, the air escaping around the mask will carry viral-laden aerosols to the environment. The same principle applies to inhalation; if one is in an environment with aerosolized viral particles not only will these aerosols penetrate the face mask, but they will be readily inhaled around the face mask. Face masks protect neither the individual wearing it nor the others sharing the environment.

Early in the pandemic several nations, the Center for Disease Control, and the World Health Organization were recommending against the use of face masks by the general public for stopping the spread of SARS-CoV-2[106]. Despite the reality of mask science (see above), ignored recommendations against mask use to stem the Covid-19 tide[107,108], and estimations like that made by the Norwegian Institute of Public Health that 200,000 people would have to wear a face mask to stop one single infection[109], the latter pandemic months have seen face mask mandates appear in nations, corporations, and businesses across the globe. The "scientific rationale" provided for these mandates comes from studies like Zhang el al[69] which boldly claims face mask mandates alone saved thousands of lives in Italy and New York city yet bases its analyses on patently false assertions like face mask mandates being the only regulatory measure issued at the time[110]. In addition, the study is riddled with methodological shortcomings and scientists around the globe have called for its retraction from its journal[110]. Lyu et al[111] attempt to link state face mask mandates to lower daily Covid-19 cases between April 8 and May 15. This study fails to account for compliance in both mandate and non-mandate states and should have collected additional months of data as, even in areas without mask mandates, face masking increased throughout the summer as did SARS-CoV-2 infection counts in many states. Leung et al[112] assessed a face mask's capacity to inhibit viral

exhalation from coronavirus and influenza infected individuals, but was unable to detect any exhaled, face mask or not, from the majority of participants and those which did shed virus, shed very little. The truth is, face mask mandates fly in the face of real science. If these mandates were merely desperate, no-harm-no-foul, misguided attempts to stymy Covid-19 they could perhaps be forgivable; however, the reality is, once again, different than what it seems at first as mask wearing is certainly not harmless.

DOES A FACE MASK AFFECT THE HEALTH OF THE WEARER?

An aspect of mask wearing which is of paramount importance yet neglected by media and TV "experts" alike is the detrimental effects chronic mask wearing can have on the wearer. Though the main negative health outcome discussed in this section revolves around the induction of subnormal levels of blood oxygen, hypoxia, caused by wearing a face mask for an extended duration, it is certainly not the only detrimental effect masks inflict upon their wearers. If an individual is symptomatic, his or her face mask will retain some expelled virus which he or she can then re-inhale. Besides hampering the sick individual's attempt to aid his or her swamped immune system by reducing his or her viral load, the danger of inhalation of the virus from the face mask into the nasal passages increases the possibility of SARS-CoV-2 entering the brain by way of the olfactory nerves, a brain entry pathway utilized by other coronaviruses[113]. Glial cells and neurons express angiotensin converting enzyme 2[114], one of the SARS-CoV-2 receptor proteins[115], which the virus uses to enter these cells to produce the nervous system complications witnessed in some Covid-19 cases[116–118]. Mask wearing also reduces one's capacity to breathe through nasal passages after mask removal[119], reduces work rate performance[120], and significantly increases one's susceptibility to headaches[121,122], though this latter effect likely derives from hypoxia[123].

One of the more controversial aspects of face masking is whether it induces hypoxia. Virtually all "fact checking" websites deny hypoxia is caused by face mask wearing but cite no scientific publication and usually justify their position with a quote from one of those dubious "experts". Scientific examination of the relationship between face mask use and blood oxygen has demonstrated mask use to cause hypoxia. Beder et al[124] analyzed the blood oxygen saturation of surgeons after they completed their operations. This study revealed a time dependent effect on loss of hemoglobin oxygen saturation as longer surgery times (surgical mask wearing) were associated with greater oxygen deprivation, reaching statistical significance at 60-120 minute surgeries and further decreasing thereafter[124]. Johnson et al[125] probed face mask wearing and oxygen consumption by exercising individuals and found face mask-induced airway resistance caused maximum oxygen deficits to be reached more rapidly. There are no studies that demonstrate face masking has no effect on blood oxygen concentration.

Hypoxia produces many negative health outcomes: hypoxia causes activation of general inflammation[126]; it can induce liver inflammation[127] and injury[128,129]; it can produce mutation to mitochondrial DNA which damages mitochondrion function[130]; it promotes fibrosis of multiple tissues[131–133]; it contributes to a plethora of autoimmune disorders including rheumatoid arthritis[134–136], inflammatory bowel disease[137,138], psoriasis[139,140], and systemic sclerosis[141,142]; hypoxia engenders atherosclerosis[143–145]; it promotes glucose intolerance[146]; it can increase lipid synthesis to bring about hyperlipidemia[147,148]; it alters neuromuscular signaling and output[149,150]; hypoxia dysregulates neurotransmitter production[151]; it diminishes endothelial progenitor cell blood concentration, impairing the body's ability to heal internal injury[152]; it is carcinogenic as hypoxic microenvironments enhance tumor growth and metastasis[153–157]. But this is not all. The hypoxia pathology most germane to the current world situation is its modulation of the immune system.

Hypoxia triggers adaptation to both the innate and adaptive arms of the immune system. The important molecular immunobiological effect of decreased blood oxygen involves gene expression-controlling, oxygen sensing, and ubiquitously expressed proteins aptly named hypoxia inducible factors (HIFs)[158–161]. These proteins are associated with all the above-named hypoxic physiological maladies and direct macrophage and T cell function[162]. HIFs shift the metabolic status of immune cells in which they are expressed to an increased glycolytic (glucose breakdown for energy production) phenotype[160,163]. While this metabolic switch may increase the antimicrobial capacity of macrophages[164–166], HIF expression in macrophages brought on by chronic hypoxic microenvironments can contribute to inflammatory diseases[166,167] and increase tumor angiogenesis[168–170]. HIFs also influence T cell differentiation. HIFs drive T cell differentiation to the regulatory T cell (Treg) phenotype[171] which can contribute to autoimmune diseases[172]. Treg dominance suppresses $CD4^+$ T cell activity[173] which, as stated earlier, is the immunological mechanism present in large swaths of the population unexposed to SARS-CoV-2 which provide them immunity against Covid-19 antecedent to a SARS-CoV-2 infection. Thus, wearing a face covering actually blunts one's natural immunity to Covid-19 and ability to combat SARS-CoV-2 infection. If all this were not enough, there is more: face mask-induced hypoxia actually replicates aspects of SARS-CoV-2 infection. It was recently discovered that SARS-CoV-2 infected macrophages release large quantities of proinflammatory cytokines to cause T cell dysfunction and lung epithelia death – all by way of HIF biochemistry[174]. Not only is a face mask utterly useless in stopping the spread of SARS-Cov-2, it may actually encourage its pathology. It may encourage all the above-named pathologies. No study has been performed to assess the health outcomes of face masking, yet it has been foisted, with the assumption of safety and without informed consent, upon entire societies of people.

WHAT CAN ONE DO TO FIGHT SARS-CoV-2?

One is not helpless in the face of impending infection unless one chooses to be. As opposed to the immune-suppressing action of face masking, one can engage in a variety of health-oriented behaviors which will support one's immune system. As simulated sunlight has been observed to reduce viral populations by 90% in 8 minutes[175], spending some time outside will help slow transmission as well as boost one's vitamin D levels. Vitamin D deficiency is associated with an increased risk of SARS-CoV-2 infection[176] and Covid-19 severity[177–180]; therefore, vitamin D supplementation has been suggested as a viable treatment for any stage of the viral infection[181–186]. Likewise, vitamin C is another ostensible treatment for Covid-19[187,188] and is being assessed to that end[189–191]. Vitamin A has been used in a regimen with other nutritional and oxidative therapies that has achieved a flawless success rate[192] (in an open-minded, scientific culture this method would see mass adoption). Vitamins A[193], C[194], and D[195] support multiple immune system biochemistries, and augmenting one's diet with these vitamins will help ward off all potential sickness-inducing pathogens, not just SARS-CoV-2. The same can be said for zinc[196,197] and selenium[198,199] which have also been proffered as weapons against SARS-CoV-2 infection[200]. One does not necessarily have to supplement these vitamins and minerals if one eats a diet rich in them[201]. It ought to be these foods one regularly consumes in addition to eschewing hyper-processed, sugar-dense fast foods which can scarcely be called food and which are a massive contributor to[202,203] the now perpetual obesity epidemic[204–206]. Obesity and its panoply of associated metabolic disorders induce immune system dysfunction and render one susceptible to infectious diseases[207], including Covid-19[208]. In like manner, the post-sugar consumption hyperglycemic state blunts the innate immune response[209] and increases the likelihood of SARS-CoV-2 infection taking hold[174]. True healthful behaviors will do lightyears more good for one's immune system and overall health than donning a face mask. If adopted by more and more

people, this improvement in individual health will morph into improved societal health, and healthy people and societies have no need to cower before health threats.

ON FREE CHOICE OF THE WILL

In the name of healthcare, preventative medical procedures are being forced upon the fearful, the ignorant, and the unwilling with near unanimous support of the "expert" class and with a complete absence of safety trials. Those with eyes to see recognize this is not the first time. The healthcare industry is, by very conservative estimates, the third leading cause of death in the United States of America[210,211]. Chronic illness is exploding[212], life expectancy is waning[213], and the last years of life have become a horror show of hospital visits, pharmaceuticals, and special care facilities. And where are the cures? One would expect the mountainous sums of money pumped into healthcare and biomedical research over the course of generations to have manufactured health improvement and longevity increases, yet the opposite is true. This only comes to be when the public "expert" class (doctors, scientists) is inculcated in myopic ignorance and applies its hyperspecialized knowledge sets to solve increasingly reductionist questions. When one reads headlines in the mold of, "Researchers discover (insert discovery here) and are closer to curing (insert disease here)," one ought to shake his or her head – our grandparents were reading the same drivel. Of course, not all doctors and scientists are substandard, but all doctors and scientists need to be challenged.

That which makes one human, is the ability to reason, to discern, to will. To be able to choose other than what even the most seductive of stimuli would have one choose. Yet, what we as a people have become, is a species which has been trained to respond in formulaic manner according to that ever-immutable law of nature, monkey-see-monkey-do, to stimuli emitted from the powerful technological amalgamation of Pavlovian and Skinnerian thought: the television. Its dictates, we follow and its presentation of reality, we accept. All cogitation which originates from inspiration outside its purview is considered anathema. Even the most strident opposition to its false reality wilts before the psychic driving repetition, repetition … repetition. In his 1931 book, *The Scientific Outlook*[214], Bertrand Russell describes a future where entire societies will be unable to think outside of "expert" approved opinions. Though we are not yet there, we are moving, lock step, ever so dangerously close. In order to avoid falling into that abyss, one must resist falling prey to the scientifically designed and targeted circuses which flow from personal digital media devices, one must question experts, not concede to them without thought, and one must exercise intellectual freedom to discern truth.

Asymptomatic individuals are simply healthy individuals. They are not a "case" of anything, particularly if diagnosed using a worthless test[215], and are incapable of spreading SARS-CoV-2; they should absolutely not be required to wear a face mask. The face mask does nothing to stop viral transmission but does vitiate physical health. Healthy living on the other hand, will benefit one far more than reliance on the prevailing medical establishment.

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