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# PDMJ

Masks, false safety and real dangers, Part 3:

## Hypoxia, hypercapnia and physiological effects

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### Abstract

Wearing a mask causes physiological changes to multiple organ systems, including the brain, the heart, the lungs, the kidneys and the immune system. We examine changes in oxygen and carbon dioxide concentrations in masked airspace that is available to the airways over the first 45 seconds of wear. Our findings of reduced oxygen and increased carbon dioxide in a masked airspace are not inconsistent with previously reported data. We also consider the range of injuries known to occur to the above-named organ systems in a state of hypoxia and hypercapnia. As an excretory pathway, carbon dioxide release by cells throughout the body, and then past the alveoli and then the airways and orifices, has not been previously challenged by deliberate obstruction in the history of the animal kingdom, except for relatively rare human experiments. Self-deprivation of oxygen is also unknown in the animal kingdom, and rarely attempted by humans. We examine the physiological consequences of this experiment.

### MASKS and HYPERCAPNIA

#### Do masks cause systemic hypercapnia?

Airway obstruction is a long recognized cause of retention of carbon dioxide and respiratory acidosis. A sustained level of increased carbon dioxide stays inside of masked air, compared to room air, which in turn sustains a low level of hypercapnia. Rebreathing of exhaled air has been

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found to quickly elevate [CO<sub>2</sub>] in available air above 5000 ppm, and to increase arterial CO<sub>2</sub> concentration and to increase acidosis.<sup>1</sup> The mechanism of mask-induced hypercapnia may also include the moisture on a mask trapping carbon dioxide from exhalation. Some carbon dioxide diffuses in the air, more so if dry, but some portion of it, trapped by water vapor and mask moisture, would form a weak, unstable acid with water, for re-circulation to the airways and lungs. The mechanism is that retention of CO<sub>2</sub> causes an increase in PCO<sub>2</sub>. This is the primary disturbance in respiratory acidosis. It results in an increased concentration of both HCO<sub>3</sub><sup>-</sup> and H<sup>+</sup>, which is measured as a lower pH.

Masks increase respiratory drive and bronchodilation in mild hypercapnia, from sensitive chemoreceptors picking up changes in pH of cerebrospinal fluid. Ultimately in severe hypercapnia, respiratory drive is actually depressed.

Hypercapnia is widely recognized to be an independent risk factor for death.<sup>2 3 4 5</sup> A number of organ systems are negatively impacted, including the brain, heart, lungs, immune system and musculoskeletal system.<sup>6 7</sup>

### **How quickly do masks increase carbon dioxide in the masked airspace?**

We used a new calibrated carbon dioxide meter to measure ambient carbon dioxide in room air, and then inside the masked airspace of three different masks after donning each in turn. This experiment involved a disposable surgical mask, a N-95 mask and a cloth mask. We recorded carbon dioxide parts per million inside the masked airspace. The meter refreshed its readings at 5-second intervals, and we used those same intervals in recording CO<sub>2</sub> parts per million. The maximum CO<sub>2</sub> reading on the meter is 10,000 parts per million.

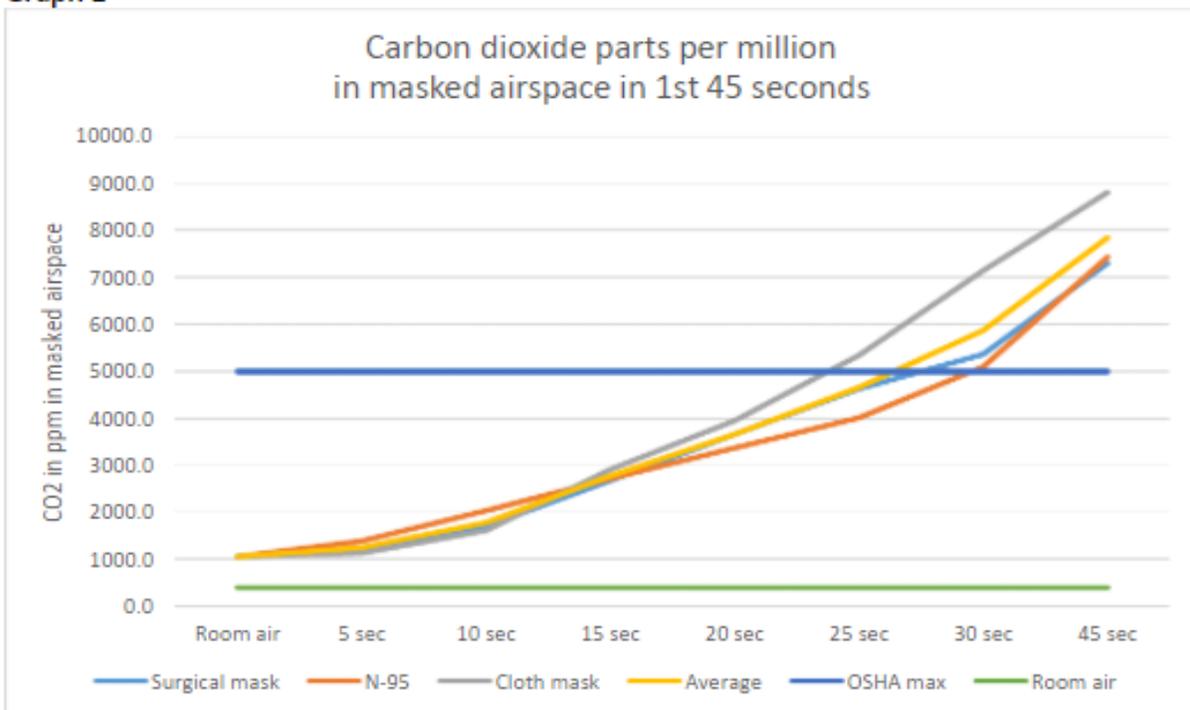
The table of those values are shown in Table 1, with the mean values shown for each 5-second interval in the first 45 seconds. After 45 seconds, the readings passed the maximum meter reading of 10,000 ppm [CO<sub>2</sub>], and were thereafter indeterminate from the meter.

**Table 1: Measured [CO2] in masked airspace**

	Room air	5 sec	10 sec	15 sec	20 sec	25 sec	30 sec	45 sec	60 sec	75 sec	90 sec
Surgical mask	1072	1298	1757	2256	2995	3215	3306	3074	3378	5483	7472
	1022	1086	1317	1667	2221	2792	3526	6479	7755	9964	>10000
	1074	1202	1502	2400	2619	2746	2948	4794	5994	8095	>10000
	1089	1211	1357	3090	5520	8230	9381	>10000			
	989	1265	1700	3257	4221	5412	6764	9465	>10000		
	1026	1363	2590	3392	4384	5377	6263	>10000			
<b>Mean</b>	<b>1045.3</b>	<b>1237.5</b>	<b>1703.8</b>	<b>2677.0</b>	<b>3660.0</b>	<b>4628.7</b>	<b>5364.7</b>	<b>7302.0</b>			
N-95	1050	1323	1834	2518	3184	4281	4689	7042	9684	>10000	
	1037	1517	3360	4133	4708	5315	5394	9082	>10000		
	1049	1475	1599	1800	1911	2773	6346	6563	>10000		
	1083	1292	1834	3312	3730	3901	4140	5692	7855	>10000	
	1073	1450	1975	2621	3820	4407	5629	7279	9240	>10000	
	1033	1266	1583	1926	2874	3392	4371	8921	>10000		
<b>Mean</b>	<b>1054.2</b>	<b>1387.2</b>	<b>2030.8</b>	<b>2718.3</b>	<b>3371.2</b>	<b>4011.5</b>	<b>5094.8</b>	<b>7429.8</b>			
Cloth mask	1084	1115	1718	2218	2725	3300	4914	6494	8410	>10000	
	1066	1057	1558	2467	3644	6369	8480	>10000			
	1050	1189	1686	3573	4400	5080	5768	8966	>10000		
	1062	1200	1685	4129	5848	7863	>10000				
	1051	1078	1430	2301	3580	5087	8555	>10000			
	1044	1115	1569	2772	3503	4321	5149	7385	9260	>10000	
<b>Mean</b>	<b>1059.5</b>	<b>1125.7</b>	<b>1607.7</b>	<b>2910.0</b>	<b>3950.0</b>	<b>5336.7</b>	<b>7144.3</b>	<b>8807.5</b>			

If we look at the time in which our readings did not yet exceed the maximum of the meter, then we have the following graph, Graph 1, of the average rise in carbon dioxide concentration inside the masked air for each mask, as [CO2] rose over the first 45 seconds of wear.

**Graph 1**



The blue horizontal line in Graph 1 represents the maximum allowable average CO<sub>2</sub> concentration in workspace air during an 8-hour work shift, by the Occupational Safety and Health Administration (OSHA) of the US Department of Labor.<sup>8</sup> The green horizontal line represents typical [CO<sub>2</sub>] in room air, which is 400 parts per million.

After donning each mask, we see that [CO<sub>2</sub>] in the masked airspace rose above acceptable OSHA limits within the first 30 seconds.

The concentration of carbon dioxide rises similarly during the time of wearing each kind of mask. These findings are consistent with known data on the carbon dioxide concentration of available airspace inside of a mask.<sup>9</sup>

Industrial workspace standards established by OSHA for carbon dioxide concentration in the workspace are for ambient room air, and these have been established since 1979. It is not the case that OSHA has mandated specific CO<sub>2</sub> concentrations for masked airspace. However, we examine these standards for available room air, and compare masked airspace to them, because in both cases we may consider [CO<sub>2</sub>] concentration in the air that is available to the airways and the lungs.

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The Food Safety and Inspection Service of the United States Department of Agriculture notes that carbon dioxide gas is used to euthanize both poultry and swine.<sup>10</sup> Concentration of this gas is therefore of concern regarding the use of masks by human beings. That government agency publishes the following warnings:

5,000 ppm = 0.5% is the OSHA Permissible Exposure Limit (PEL) for 8-hour exposure,<sup>11</sup> averaged over the workday. Each of our masks surpassed that level within the masked airspace in the first 25 to 30 seconds of wear.

At 10,000 ppm of short exposure, OSHA says there are typically no effects, possible drowsiness.

At 20,000 ppm, the Food Safety and Inspection Service advises: "Do not enter areas where CO<sub>2</sub> levels exceed 20,000 ppm until ventilation has been provided to bring the concentration down to safe levels." We should remember here that each of the masks we studied rose to half of this concentration within the first minute alone.

At 30,000 ppm = 3% [CO<sub>2</sub>], there is "moderate respiratory stimulation and increased heart rate and blood pressure."

At 40,000 ppm = 4%, OSHA finds [CO<sub>2</sub>] to be "immediately dangerous to life or health."<sup>12</sup>

Hypercapnia is known to rapidly cause intracellular acidosis in all cells in the body. There is no way to wall off the damage to only affect the lungs, due to constant gas exchange. That is, there is no known way to restrict hypercapnic effects to only the lungs.

The effects of hypercapnia progress in this order: Compensatory attempt at respiratory ventilation, labored breathing, hyperpnea; nervous system changes with changes in motor skills, visual acuity, judgment and cognition, cerebral vasodilation with increasing pressure inside the skull and headache, stimulation of the sympathetic nervous system, resulting in tachycardia, and finally, in case of extreme hypercapnia, central depression.<sup>13 14</sup>

### **Hypercapnia effects on the lungs and immune system**

Exhaled breath contains about 5% = 50,000 ppm carbon dioxide. This is more than 100 times the average of room air which is about 0.04% [CO<sub>2</sub>]. Exhaled [CO<sub>2</sub>] is 10 times the upper limit permitted by OSHA in ambient air. Yet each of us exhales this concentration with every breath. Should we re-breathe our own exhaled breath?

A study of healthy healthcare workers found increased [CO<sub>2</sub>] and decreased [O<sub>2</sub>] in the respiratory dead space inside a N95 filtering respirator while walking on a treadmill. Within one hour of use, these were "significantly above and below, respectively, the ambient workplace standards."<sup>15</sup> The exhalation valve of the N95 masks did not significantly change its impact on P(CO<sub>2</sub>).

Hypercapnia has a number of damaging effects on the lungs. Those effects seem to begin with disruption of Na<sup>+</sup>-K<sup>+</sup>-ATPase, which leads to impaired alveolar fluid reabsorption. This results in alveolar edema, which in turn obstructs optimal gas exchange.<sup>16</sup> Hypercapnia also inhibits repair of alveoli by impairing proliferation of alveolar epithelial cells via inhibition of the citric acid cycle and resulting mitochondrial dysfunction.<sup>17</sup>

Cilia are made immotile by hypercapnia, along with mask changes in humidity and temperature in the upper airway. This leads to predisposing mask wearers to lower respiratory tract infections by allowing deep seeding of oropharyngeal flora.<sup>18</sup> The lower respiratory system is usually sterile because of the action of the cilia that escalate debris and microorganisms up toward the orifices. Impairment of this process, such as in hypercapnia, may partly explain a correlation of hypercapnia with increased mortality from pulmonary infections.

Hypercapnia correlates with increased mortality in hospitalized patients with community-acquired pneumonia.<sup>19</sup> This seems to be due to a number of factors, including that hypercapnia inhibits IL-6 and TNF as well as inhibiting immune cell function generally,<sup>20</sup> including alveolar macrophages.<sup>21</sup>

Hypercapnia was found to downregulate genes related to immune response. The researchers that had studied this in depth found that “hypercapnia would suppress airway epithelial innate immune response to microbial pathogens and other inflammatory stimuli.”<sup>22</sup> They also found suppressive effects of hypercapnia on macrophage, neutrophil and alveolar epithelial cell functions. Hypercapnia was found to decrease bacterial clearance in rats.<sup>23</sup>

In our previous paper in this series, we found a historical correlation with a hypercapnic practice, specifically mask-wearing, and a severe surge of bacterial pneumonia deaths.<sup>24</sup> This time period was mis-named the Spanish Flu, due to a number of reasons, too extensive for this paper. Dr. Anthony Fauci’s research team found that every cadaver exhumed from that time in 1918 – 1919 showed the cause of death was bacterial pneumonia, secondary to typical upper respiratory bacteria.<sup>25</sup>

Common and life-threatening diseases of impeded air flow include both obstructive disorders such as asthma, COPD, bronchiectasis and emphysema, as well as restrictive disorders, such as pneumothorax, atelectasis, respiratory distress syndrome and pulmonary fibrosis.

### Hypercapnia effects on the blood

Excess carbon dioxide is buffered exclusively in the intracellular fluid, especially in red blood cells. CO<sub>2</sub> crosses cell membranes by diffusion, and combines with water to convert to H<sup>+</sup> and HCO<sub>3</sub><sup>-</sup>. The hydrogen is then buffered by intracellular proteins such as hemoglobin and organic phosphates. The price paid by the red blood cells for this buffering is seen in the comparison of normal red blood cells on the left versus the damaged and depleted red blood cells on the right.

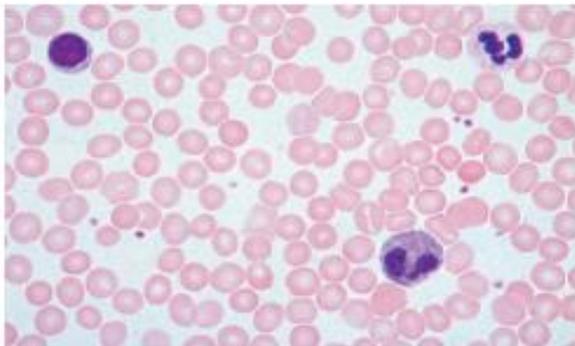


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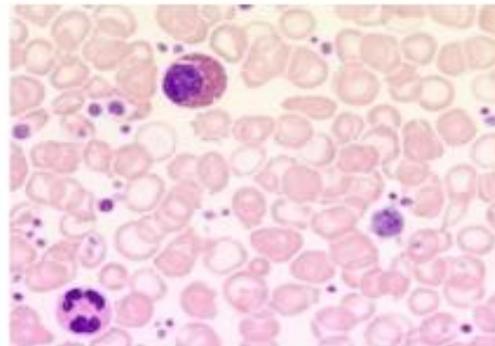


Photo from  
<http://img.medscapestatic.com/pi/meds/ckb/61/36661tn.jpg>

The above photo on the right demonstrates secondary polycythemia.. This is a known consequence of hypoxia. This abnormal blood finding may also correlate with dehydration from wearing a mask. The US National Institute of Occupational Safety and Health (NIOSH) says that "particular features of PPE can impose a physiological . . . burden on the healthcare worker." And "dehydration can be a significant problem while wearing PPE."<sup>26</sup> Individuals suffering from dehydration are at risk for relative erythrocytosis, which can manifest as polycythemia vera.<sup>27</sup> Polycythemia vera is an independent risk factor for other cancers, commonly treated with lifelong blood thinning medication. Polycythemia develops slowly over years. Are today's mask wearers at future risk of developing this blood cancer?

### Hypercapnia effects on the kidneys

The kidneys are tasked with compensating for the damage inflicted on the blood stream by respiratory acidosis. They must excrete hydrogen ions and reabsorb the newly made HCO<sub>3</sub><sup>-</sup>. The Henderson-Hasselbalch equation indicates the extent to which increased HCO<sub>3</sub><sup>-</sup> compensates for the acidic condition.

$$\text{pH}=\text{pK}+\log[\text{HCO}_3^-]/(\text{Pco}_2)$$

The  $[HCO_3^-]$  is a reflection of renal or metabolic compensation, whereas the  $PCO_2$  reflects the primary disturbance, where airway obstruction created an acidemia.<sup>28</sup>

The kidneys show decreased GFR and decreased urine output, as well as increased renal vascular resistance, as a result of hypercapnia.<sup>29</sup> Aciduria increases as a compensatory mechanism to excrete acid. This in turn damages tubules and has been shown to worsen kidney function in those with established chronic diseases.<sup>30</sup>

### **Hypercapnia effects on the cardiovascular system**

A hypercapnic patient may be warm, flushed and tachycardic. A bounding pulse and sweating may also be present. Arrhythmias may be present if there is significant hypoxemia. Arterial  $pCO_2$  above 90 mmHg is not compatible with life, because hypercapnia is necessarily accompanied by hypoxemia, in this case by  $pO_2 = 37$ .<sup>31</sup> It has been noted that masked patients are often found to be tachycardic, to be discussed more further on in this paper.

### **Hypercapnia effects on the central nervous system**

Central nervous system effects, such as headache, fatigue, dizziness and drowsiness are common effects of chronic obstructive pulmonary disease (COPD),<sup>32</sup> In this patient cohort we also see defects in proprioception, instability of posture and gait, as well as falls, with strong evidence that these result from hypercapnia.<sup>33</sup> There is a progressively increasing sedation from mask use and increased intracranial pressure. Headaches are a common complaint of mask wearers, and are found to be attributable to hypercapnia.<sup>34</sup> Increases in  $PCO_2$  lead to increases in cerebral blood flow and cerebral blood volume, as well as a resulting intracranial pressure.<sup>35</sup> These are consistent with findings through the rest of the body.

Slowed performance of reasoning tasks was observed at 20 minutes of inhaling 4.5% to 7.5%  $[CO_2]$ .<sup>36</sup> When subjects were exposed to 2,500 parts per million carbon dioxide in room air, it was found that their decision-making ability declined by 93%, which was comparable to being drunk or having a head injury.<sup>37</sup> At this same level of  $[CO_2]$ , it was also found that visual performance suffered.<sup>38</sup> We measured this same level of  $[CO_2]$  inside masked airspace at 15 seconds.

Even smaller  $CO_2$  concentrations had deleterious effects.  $CO_2$  exposure beginning at 1000 ppm affected cognitive performance, such as problem resolution and decision-making.<sup>39</sup> We measured 1000 ppm  $[CO_2]$  in masked airspace within the first few seconds of wear.



## **MASKS and HYPOXIA**

### **Masks create hypoxia in the wearers**

A study of 53 surgeons who were non-smokers and without chronic lung disease were shown to have a decrease in saturation of arterial pulsations (SpO<sub>2</sub>) when performing surgery while masked. Oxygen saturation decreased significantly after the operations in both age groups, with a greater decrease in surgeons over the age of 35.<sup>40</sup>

A study of 39 end-stage renal disease patients wearing N-95 masks for 4 hours during hemodialysis were found to have significantly reduced PaO<sub>2</sub> over that time. The average drop in PaO<sub>2</sub> was from a baseline PaO<sub>2</sub> of 101.7 to 15.8,  $p = 0.006$ . Respiratory rate increased from 16.8 to 18.8 respirations per minute,  $p < 0.001$ . Chest discomfort and respiratory distress were also reported by the subjects.<sup>41</sup>

### **Hypoxia is a health hazard**

Hypoxia is deadly. Each year, many workers are injured or die due to oxygen deficiency.<sup>42</sup> "There have been reports of workers who have opened a hatch to an O<sub>2</sub>-deficient atmosphere and died with only their head inside the hazard. The low level of O<sub>2</sub> resulted in a feeling of euphoria and the workers could not comprehend that they only needed to lean back out of the hatch to save their lives."<sup>43</sup>

The issue of mask wearing is especially critical for children. In children, any hypoxic condition is even more of an emergency than it is for an adult. This is partly due to their more horizontal ribs and barrel-shaped chest, resulting in children relying primarily on diaphragm muscles for breathing, not nearly so much intercostal muscles, as in adults. These diaphragm muscles have proportionately fewer type I muscle fibers, resulting in earlier fatigue.<sup>44</sup> Also, a child's tongue is relatively large in proportion to the size of the pharynx, and the epiglottis is floppy.<sup>45</sup> These anatomical differences make a child potentially more vulnerable than an adult to injury from hypoxic assault.

We consider it urgent for children to be released from mask "mandates," based on this information.

### **Hypoxia in masked airspace**

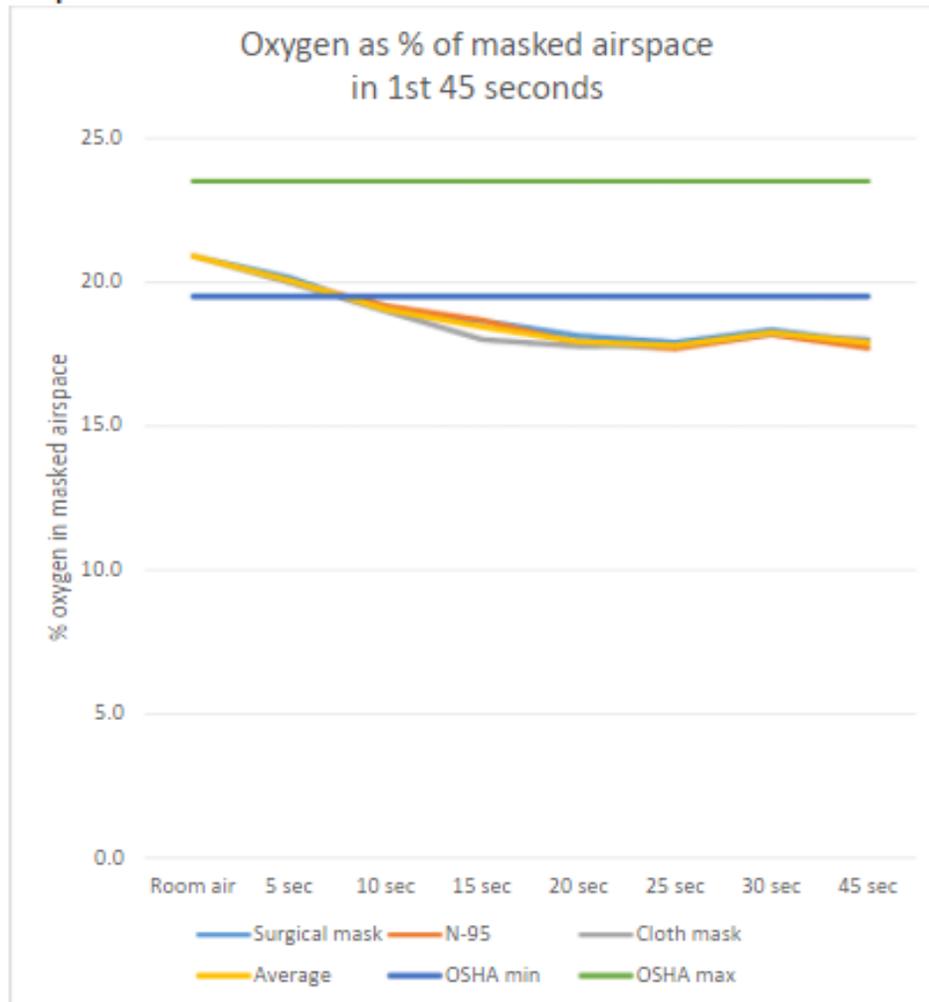
In order to determine the percent of oxygen in masked airspace, we ran 6 trials each for 45 seconds of 3 types of masks: a disposable surgical mask, a N-95 mask and a laundered cloth mask.

We charted the results as follows, showing the average for each type of mask, compared to OSHA workspace requirements for air available to the airways.

**Table 2 Measured [O2] in masked airspace**

	Room air	5 sec	10 sec	15 sec	20 sec	25 sec	30 sec	45 sec	60 sec
Surgical mask	20.9	20.2	19.2	19.1	18.5	19.1	18.4	18.1	17.7
	20.9	20.1	18.9	18.7	18.1	17.7	18.1	17.9	17.4
	20.9	20.3	18.7	18.1	18.1	18.2	17.9	17.6	17.7
	20.9	19.6	19.1	18.7	17.7	17.1	18.5	17.1	17.5
	20.9	19.8	19.1	18.9	18.2	17.4	18.7	18.6	16.7
	20.9	20.9	19.0	18.4	18.2	17.9	18.4	18.2	18.6
<b>Average</b>	<b>20.9</b>	<b>20.2</b>	<b>19.0</b>	<b>18.7</b>	<b>18.1</b>	<b>17.9</b>	<b>18.3</b>	<b>17.9</b>	<b>17.6</b>
N-95	20.9	20.0	19.1	18.1	17.7	18.2	18.4	17.2	17.4
	20.9	19.7	19.3	18.5	17.3	18.0	18.3	18.2	16.7
	20.9	19.6	18.1	18.6	18.4	18.2	17.8	17.5	17.1
	20.9	20.1	19.4	19.1	18.0	17.6	18.3	17.2	17.8
	20.9	19.8	19.3	19.0	17.8	16.9	18.1	18.2	17.4
	20.9	20.9	19.8	18.7	18.0	17.2	18.1	17.9	17.7
<b>Average</b>	<b>20.9</b>	<b>20.0</b>	<b>19.2</b>	<b>18.7</b>	<b>17.9</b>	<b>17.7</b>	<b>18.2</b>	<b>17.7</b>	<b>17.4</b>
Cloth mask	20.9	19.6	19.5	17.7	16.7	17.5	17.5	16.7	17.5
	20.9	20.1	19.2	17.2	17.1	16.9	17.1	17.0	17.4
	20.9	20.2	19.3	18.4	18.4	18.2	19.0	17.9	17.1
	20.9	20.0	18.9	18.6	19.0	19.8	19.3	18.8	18.7
	20.9	20.1	18.4	18.3	17.7	17.3	17.9	18.1	17.7
	20.9	19.9	18.6	17.8	17.7	17.0	18.5	19.5	17.1
<b>Average</b>	<b>20.9</b>	<b>20.0</b>	<b>19.0</b>	<b>18.0</b>	<b>17.8</b>	<b>17.8</b>	<b>18.2</b>	<b>18.0</b>	<b>17.6</b>

**Graph 2**



It can be seen from Graph 2 that all of the masks showed similar results, and that in each type of mask, available oxygen as a percentage of available air volume decreased to less than the OSHA required minimum of 19.5%<sup>46</sup> in less than 10 seconds of wear, and stayed below that threshold. Breathing seemed to be shallow until 30 seconds of wear. Then the wearer's responsive drawing of air through pores and side gaps and top gaps around the mask appeared to occur mostly at 30 seconds, but did not compensate adequately to return [O<sub>2</sub>] in the masked airspace back above the OSHA minimum requirement of 19.5% [O<sub>2</sub>] in available air.

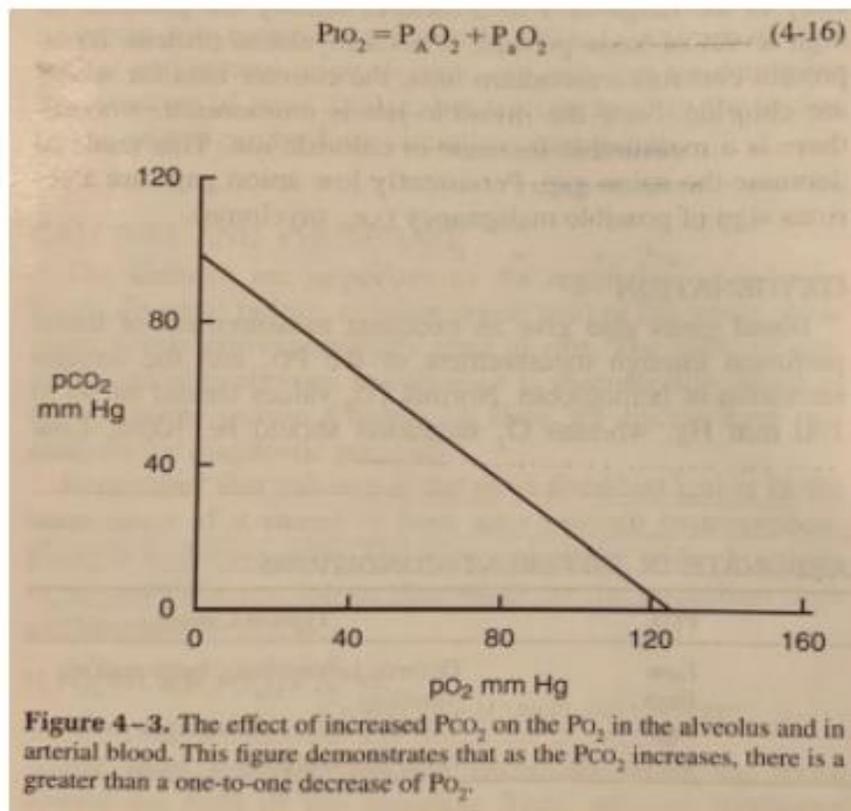
The above findings are consistent with known decrease of oxygen concentration in the airspace inside of masks.<sup>47</sup> The standards for oxygen concentration in airspace available to workers has been so strictly enforced by OSHA that in a low-oxygen workspace, employee access must be restricted by using locks or barriers. Oxygen-level monitoring is required before entry, and the space must meet OSHA oxygen-level standards during the entire time that it is in use.<sup>48</sup> Is the

space of available airflow to the human airways any less important to protect from low ambient O<sub>2</sub>, simply because it is the very small space between the mask and the respiratory orifices?

The United States Code of Federal Regulations in paragraph (d) of 29 CFR 1910.134 “requires the employer to evaluate respiratory hazard(s) in the workplace, identify relevant workplace and user factors, and base respirator selection on these factors.” This “shall include a reasonable estimate of employee exposures to respiratory hazard(s) . . .” Exceptions are permitted “if the employer can meet the difficult evidentiary burden of showing that the oxygen content can be controlled reliably enough to remain within the ranges specified . . .”<sup>49</sup> Does this leave employers liable for injuries to workers who wear masks?

### Hypoxia accompanies hypercapnia

Retention of carbon dioxide reduces oxygen availability, as in COPD. As CO<sub>2</sub> builds up in alveoli, the available volume for oxygen in the airspace is reduced. “For each increment in the PaCO<sub>2</sub>, there will be a more than one-to-one decrease in the PaO<sub>2</sub>, which will result in severe oxygen deficits, as illustrated in the following graph.”<sup>50</sup>



From J Henry. Clinical Diagnosis and Management by Laboratory Methods. 19<sup>th</sup> ed. WB Saunders Co. © 1996.

## **Hypoxia effects on the brain**

Hypoxia, which is the lack of oxygen available to the respiratory system and to the tissues generally, stimulates mitochondria to generate reactive oxygen species (ROS). All body tissues are vulnerable to ROS, but the brain is especially vulnerable. ROS damage lipids, protein and DNA. The brain is 60-70% lipids and low in antioxidants, and is therefore especially vulnerable to ROS damage.<sup>51</sup> For the immature brain, the problem is even worse. Poorly developed scavenging systems and the high availability of free iron leave the child's brain, especially neurons and oligodendrocytes, vulnerable to the oxidative damage of free radicals.<sup>52</sup>

A biochemical mechanism of hypoxia damage to the brain is that hypoxia stimulates a kallikrein – bradykinin – nitric oxide pathway.<sup>53</sup> As a result, the blood-brain barrier can become more permeable. Extravasation of plasma proteins and brain edema may result.<sup>54</sup>

Neurologist and neurophysiologist Dr. Margarite Friesz-Brisson says this about forcing masks on children: “The child needs the brain to learn, and the brain needs oxygen to function. We don't need a clinical study for that. This is simple, indisputable physiology.” She warns of a “tsunami of dementia” in the future, because of oxygen deprivation from wearing masks today. She points out long-recognized physiology that re-inhaling our exhaled air creates a state of oxygen deficiency and an excess of carbon dioxide.

Normalization is a phenomenon observed in medicine in which the individual adapts to disadvantageous conditions. Mask wearers may believe that they have become accustomed to wearing a mask. However, the effects of degenerative processes in the brain accumulate during a state of oxygen deprivation.<sup>55</sup>

## **Cardiovascular effects of hypoxia**

It is established that mask wearers work harder at breathing and have greater inspiratory flow than unmasked individuals. This in turn increases sympathetic vasoconstrictor outflow to limb skeletal muscle. After donning a mask, even at rest, mean arterial blood pressure increased by 12 mmHg, and heart rate increased by 27 BPM.<sup>56</sup> Cardiac output is increased and so is prolongation of the QT interval. Vasoactive effects include systemic arterial vasodilation and pulmonary arterial vasoconstriction. It has been found that even at low workloads, in a hypoxic environment, there is not only increased heart rate and blood pressure, but also aortic pressure and left ventricular pressures increase, which in turn promote cardiac overload and coronary demand.<sup>57</sup>

Let us now look at the mechanisms of how this happens. When there is hypoxic assault on the body, hemoglobin is the first sensor. The red blood cells are stimulated to produce nitric oxide, which causes vasodilation and increased blood flow. Hypoxia decreases the threshold needed

for this to happen.<sup>58</sup> This vasodilation is a protective effect on the tissues from hypoxic assault, and as a result, the individual becomes tachycardic and agitated.

### **Hypoxia effects on erythropoiesis**

Mask wearing results in loss of available oxygen transport to the tissues. This in turn results in increased erythrocyte production. If hypoxia persists, then free 2,3-DPG is depleted. This leads to increased glycolysis. This leads to production of more 2,3-DPG, which reduces oxygen affinity for hemoglobin. As a result, oxygen is released to the tissues away from vital organs, such as the brain, liver, kidneys and heart. Low oxygenation stimulates production of erythropoietin, which results in more red blood cell production.<sup>59</sup>

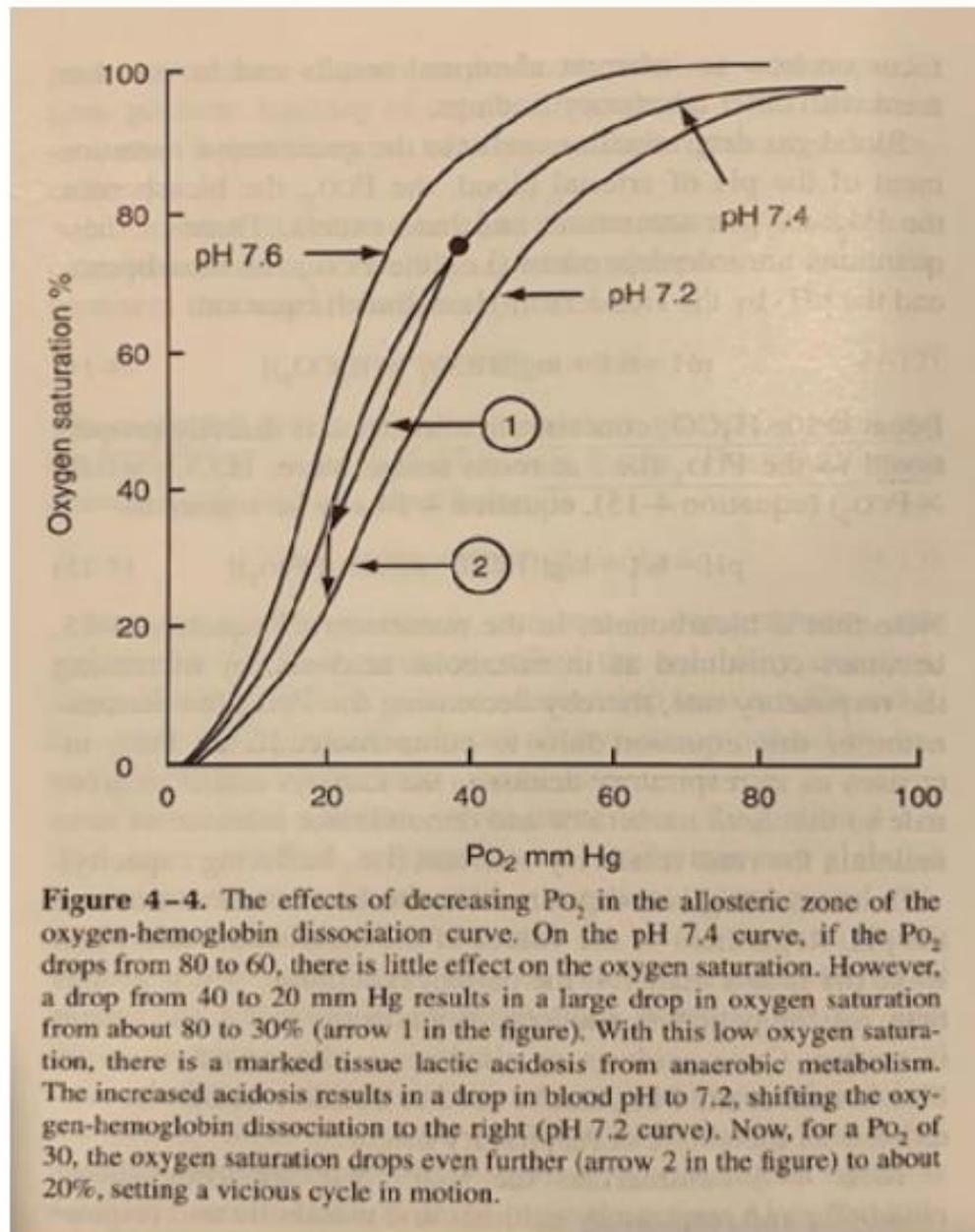
Why would we deliberately expose ourselves to persistent hypoxia, which leads to tissue hypoxia in vital organs and increased red blood cell production? Conditions featuring erythroid hyperplasia include but are not limited to: acute myeloid leukemia, congenital dyserythropoietic anemia, microangiopathic hemolytic anemia and sideroblastic anemia.<sup>60</sup> In turn, these can increase risk of polycythemia vera, a disease of thick blood from overproduction of red blood cells. In fact, loss of oxygen is the most common cause of polycythemia vera.<sup>61</sup>

### **Hypoxia and the gastrointestinal tract**

Hypoxia and hypoxia-dependent signaling pathways are becoming better-appreciated in their role in intestinal disease. Tissue hypoxia is recognized as a feature of inflammatory bowel disease.<sup>62</sup> Although intestinal tissue averages 7% [O<sub>2</sub>], hypoxic stress occurs in infection and inflammation, states which are characterized by oxygen demand being higher than supply.<sup>63</sup> As a result of induced hypoxia, the delicate balance of commensal bacteria on the one hand and limitation of pathogenic bacterial access to tissues on the other is vulnerable to new disruption.

### **Hypoxia and cancer risk**

When there is resistance to inspiratory and expiratory flow, respiratory acidosis and increased lactate levels have been found.<sup>64</sup> At the [O<sub>2</sub>] levels we measured in the masked airspace, at 17%, higher levels of lactic acid accumulated.<sup>65</sup> This is no surprise given the understanding we have of the metabolic initiation of cancer from Nobel Prize biochemist Otto Warburg. He found that the removal of oxygen initiates the destruction of respiration in cells, and that this process leads to formation of cancer.<sup>66</sup> As tissue oxygenation drops, cells resort to anaerobic glycolysis, which ends the glycolytic pathway with conversion of pyruvate to lactic acid. A marked increase in tissue lactic acidosis results. When oxygen saturation lowers to 30%, blood pH drops to 7.2, which shifts the oxygen-hemoglobin dissociation curve to the right, and sets a vicious cycle in motion, as seen here.



From J Henry. Clinical Diagnosis and Management by Laboratory Methods. 19<sup>th</sup> ed. WB Saunders Co. © 1996.

Warburg showed that cancer cells live in hypoxic conditions, and that an initial assault on normal cells leads to hypoxia that in turn damages mitochondria, which is the first step in the cancerous process. He found “the root cause of cancer is oxygen deficiency. . . . Cancer cannot survive in the presence of high levels of oxygen.”<sup>67</sup>

Hypoxia also negatively impacts the mobility of natural killer cells,<sup>68</sup> which are one of the strongest defenses of the immune system against cancer.

For over a quarter century, Guy Crittenden was editor of *HazMat Management*, an award-winning occupational health and safety journal. That journal routinely published articles regarding masks and compliance with health and safety laws. He has several major concerns with mask use by the public.<sup>69</sup> One of them is that the disposable surgical masks are sterilized with ethylene oxide, a known carcinogen.<sup>70</sup> Another is that the disposable surgical masks and N-95 respirators are woven with polytetrafluoroethylene (PTFE),<sup>71 72</sup> PTFE is made using perfluorooctanoic acid (PFOA), a known carcinogen. PFOA has been associated with cancer of the breast,<sup>73</sup> testicles, liver and pancreas.<sup>74</sup> As noted above, inspiratory flow is greater in mask wearers, which brings these compounds deep into the lungs.

### **Hypoxia and immune function**

During a state of hypoxia, the body produces hypoxia-inducible factor-1 (HIF-1). HIF-1 is known to lower T-cell function.<sup>75</sup> CD-4 T-cells have been observed to decline in this process, and they are known to fight viral infections.<sup>76</sup> This raises concerns about whether masks can function as desired during the COVID-19 era. The sudden increase of widespread masking throughout much of the world in 2020 has been motivated by a desire to limit or control the spread of the SARS-CoV-2 virus that is associated with COVID-19. As we have demonstrated, the hypoxia caused by mask-wearing defeats the objective of anti-viral strategy. As we showed in our previous paper in this series, mask use is correlated with higher, not lower, incidence of COVID-19.<sup>77</sup>

### **Other effects of masks**

Masks have been observed to create skin damage in 526 of 542 = 97% of healthcare workers studied. The affected sites were especially the nasal bridge, but also hands, cheeks and forehead. Longer exposure worsened outcome.<sup>78</sup>



### **US FDA definition of a “medical device”**

According to the United States Food and Drug Administration (FDA), a medical device has a specific definition, and it is defined as follows:

*“An instrument, apparatus, implement, machine, contrivance, implant, in vitro reagent, or other similar or related article, including a component part or accessory which is: recognized in the official National Formulary, or the United States Pharmacopeia, or any supplement to them,*

*-Intended for use in the diagnosis of disease or other conditions, or in the cure, mitigation, treatment, or prevention of disease in man or other animals, or*

*- intended to affect the structure or any function of the body of man or other animals, and which does not achieve its primary intended purposes through chemical action within or on the body of man or other animals and which is not dependent upon being metabolized for the achievement of its primary intended purposes.”<sup>79 80</sup>*

According to the FDA, a prescription for use of a medical device falls to state laws and regulations that determine who can write a prescription for a medical device in each state.<sup>81</sup> The FDA defers to the states regarding who can write a valid prescription. At this time in the United States at least, there are no known prescription rights granted to anyone who does not already hold a license to practice medicine.

However, also in the United States at this time, there are prominent politicians, as well as elected and appointed government leaders, who are “mandating” that citizens in their jurisdictions wear masks when in public.

We submit that a face mask is an apparatus ostensibly intended for prevention of disease, and therefore that it fits within the FDA definition of a medical device, although it is commonly sold over the counter, with no prescription. Therefore, is there now a situation in the United States, and throughout the world, of political leaders prescribing medical devices, including for complete strangers, without so much as a medical consult? Are these same political leaders practicing medicine without a license? If so, are they liable for injuries through these actions, and will they be prosecuted for their actions?

And if these political leaders are prescribing a medical device, without informed consent, then is it also the case that the same politicians and government officials are violating US federal laws regarding informed consent? The US Code of Federal Regulations (CFR) Title 21, Subchapter A, Part 50, Subpart B discusses the requirements of informed consent. Certainly, the same officials are in violation of the Universal Declaration of Human Rights and the Nuremberg Code, which are internationally honored guiding documents on citizens’ freedom from medical coercion and medical experimentation.

We therefore urge that people everywhere consider this definition of medical device, and to consider if they want their political leaders and / or their news media to practice medicine on them, without prior medical training, a license to practice medicine, or even so much as an individualized clinical consult.

## **Conclusion**

Our first paper in this series on the false safety and real dangers of masks examined the loose particulate and loose fibers that we found on new masks of various kinds at 40 times and greater magnification, along with the consideration of possible consequences of inhalation of such debris.

Our second paper in this series examined microbial challenges from masks, the dysregulation and imbalance of microbiota in the respiratory tract, and the consequences of such imbalances throughout the body. We showed that face masks are more likely to trap and re-circulate respiratory droplets and microbes, with incubation and proliferation of the same, inside the masked airspace and airways, which increases – not decreases – the risk of infections for major respiratory pathogens, bacterial, fungal and viral.

This paper, the third in our series, focuses on physiological changes induced by hypoxia and hypercapnia. Our findings of reduced oxygen and increased carbon dioxide in a masked airspace are not inconsistent with previously reported data.<sup>82 83</sup> Evidence of damage to multiple organ systems from the documented levels of [O<sub>2</sub>] and [CO<sub>2</sub>] in available airspace between a facemask and the airways are cited above and are abundant in the medical literature.

The pathological triad of micro-particles as long-term hazards, and bacterial and fungal infections as mid-term hazards, as well as injury from hypoxia and hypercapnia in the short-term, are expected to have synergistic results in endangering the health of masked people. Because of the extensive risk to mask wearers documented in these three papers, we urgently recommend that no adult or child be coerced to wear a mask under any circumstances. We further recommend that facemask hazards be published prominently and that masks only be worn by adults who choose to do so, and only with freely given informed consent, with full knowledge of their hazards, and that their use be prohibited for children, adult students and workers.

If on the other hand, widespread use of masks and mandating the same continue, then the question arises, from the data shown herein, whether morbidity and mortality from mask-wearing will exceed those of COVID-19 or other known infectious diseases. What will be the long-term effects of mask wearing if it continues? And will we be able to distinguish mask injury from COVID-19 or other pathologies? The evidence presented here, in summary of clinical data from around the world, show that masks can accelerate morbidity and mortality in those who are already ill, and that masks can sicken healthy people. Before masks are forced

[https://pdmj.org/papers/masks\\_false\\_safety\\_and\\_real\\_dangers\\_part3/](https://pdmj.org/papers/masks_false_safety_and_real_dangers_part3/)

onto school children and workers, why are animal studies not being done with all-day masking, to investigate safety issues? How much of an increase in mask-related illnesses will we have to observe before mask “mandates” end?

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