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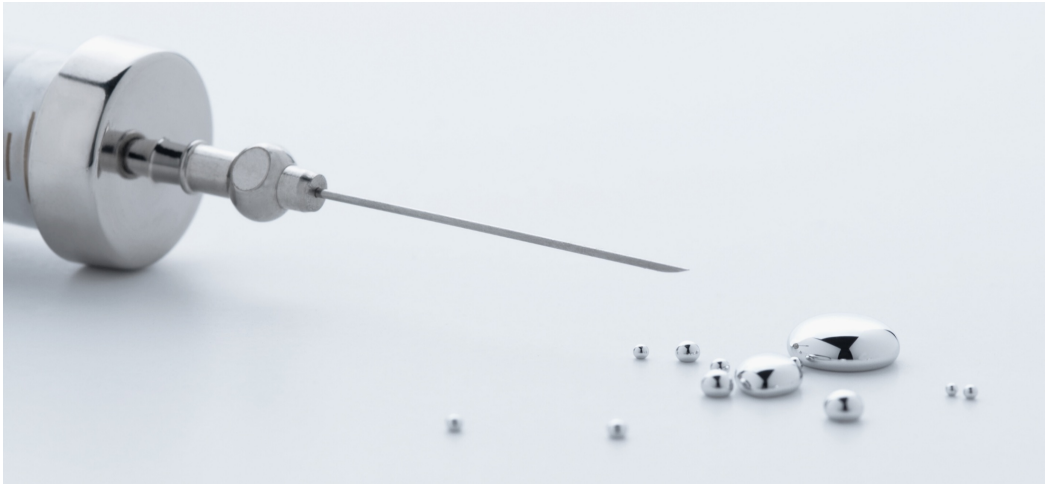

Mercury as a factor in COVID-19 mortality: hypothesis and evidence.

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Abstract

The majority of those infected with COVID-19 recover without serious complications. However, a small subset of the population is unable to recover from infection and tragically succumb. Conditions found in those that suffer poor COVID-19 outcomes are hypercoagulability, high levels of pro-inflammatory cytokines, and lymphopenia. These rare conditions can all be caused by mercury. In addition, men are more likely to die from infection than women. Mercury can account for this gender skew as it is potentiated by testosterone. The hypothesis is that mercury toxicity is causing a susceptibility to COVID-19 death. This hypothesis offers a mechanistic understanding of COVID-19 deaths. Mercury sets the stage by suppressing the immune system and causing hypercoagulability. COVID-19 infection then triggers coagulation leading to thrombosis and an increase in proinflammatory cytokines resulting in patient death.

Introduction

Coronavirus disease 2019 (COVID-19) is a global pandemic that is currently the focus of an unprecedented wave of research. A characterizing feature of COVID-19 is that the majority of people infected fight it off effectively with few or no symptoms presented.[1–5] The typical immune response is more than sufficient to overcome the virus. There is however a small section of the population that is not able to do that and tragically succumb. There is something different about this subset of the population.

Factors that increase one's risk for succumbing to the virus are lymphopenia, high levels of pro-inflammatory cytokines, hypercoagulability, age, being male, hypertension, obesity and diabetes.[6–9] All of these disparate factors can be attributed to mercury toxicity. Below I review each factor and the evidence that it can be linked to mercury. Then I analyze this evidence to develop a mechanistic model of COVID-19 mortality.

Methodology:

To identify the known factors that increase one's risk for COVID-19 death and acute respiratory distress syndrome (ARDS), a literature review via Google Scholar and PubMed was used. Each of the identified factors was cross-referenced to determine if heavy metal toxicity could cause the change. Surprisingly, all of the factors can be explained by mercury toxicity. The following is a brief summary of the risk factors and a summary of the role that mercury may play in them.

Results

Lymphopenia: a below normal number of lymphocytes, has been of interest since the outset of the pandemic.[10] Tan et al (2020) documented that lymphopenia predicts the severity of COVID-19 disease.[7] Lymphopenia is associated with COVID-19 death and acute respiratory distress syndrome (ARDS).[11] Gallais et al (2020) illustrate a mechanism for lymphopenia leading to poor patient outcome because viral-specific T cells are critical in fighting COVID-19 infection.[5] One would expect those with low T cell counts to fair worse. Mercury causes T cell death.[12,13]

Cytokine response: COVID-19 deaths appear to be related to the immune system launching a "cytokine storm" wherein excessive levels of cytokines are released triggering a chain of events that can kill the patient.[8] Mercury alters the cytokine response by increasing proinflammatory cytokines. Intriguingly, the relationship is even more specific than that; interleukin-1β (IL-1β), IL-6, IL-8 cytokines are at the heart of COVID-19 induced cytokine storms, these are the exact cytokines that mercury increases.[14–17]

Hypercoagulability: Endemen et al (2020) found that COVID-19 is linked to hypercoagulability.[6] This conclusion is supported by cohort studies that found hypercoagulability and a severe inflammatory state in COVID-19 patients.[18,19] Hypercoagulability is one of the known effects of mercury.[20–22] Viral infections activate the blood coagulation system.[23] Additionally, coagulation stimulates the cytokine response and the expression of IL-1β genes.[24,25]

Gender skew: Men are more likely to die from COVID-19 infection than women.[26,27] Although there has been much speculation as to why, currently there is no known mechanism. Mercury offers a mechanism for the gender skew because it affects males and females differently. Males are more sensitive to mercury because it is potentiated by testosterone.[28–31] The combination of mercury and testosterone should make men more vulnerable to COVID-19 death and therefore skew the deaths toward males.

Age: The risk of COVID-19 related death increases with age.[32] Seniors have significantly higher mercury levels in their blood than young people because each year you typically retain more than you excrete.[33]

Hypertension: Hypertension is a common COVID-19 comorbidity.[9] Heavy metals increase risk for hypertension.[34–38] Mercury causes hypertension by inactivating catecholamine-0-methyltransferase, which increases serum and urinary epinephrine, norepinephrine, and dopamine. This effect increases blood pressure.[34]

Selenium deficiency: Selenium deficiency is associated with COVID-19 deaths.[39] Mercury depletes selenium.[40]

Diabetes: Diabetes is a common comorbidity of COVID-19.[9] Mercury has a demonstrated link with diabetes.[41–44]

Obesity: Obesity is a common comorbidity of COVID-19.[9] Mercury has a demonstrated link with obesity.[45]

Discussion

My hypothesis is that bioaccumulation of mercury is making certain individuals susceptible to COVID-19 mortality. A hypothetical mechanism is illustrated in Figure 1. Mercury induced hypercoagulation is touched off by COVID-19 infection. Coagulation induces proinflammatory cytokine response exacerbated by mercury causing a cytokine storm.

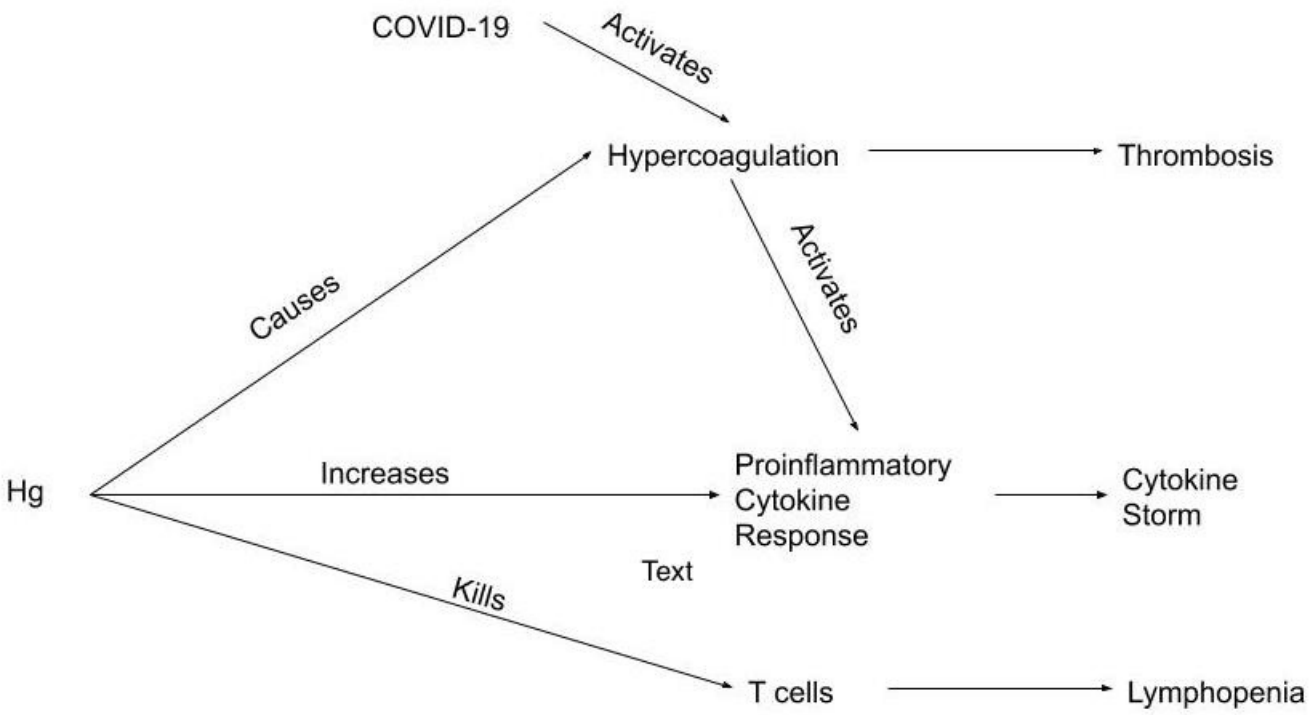

Figure 1. Illustrated is the hypothesized mechanism by which mercury (Hg) toxicity is interacting with COVID-19 to lead to patient mortality. Mercury is known to cause hypercoagulation and a proinflammatory cytokine response. COVID-19 touches off hypercoagulation which activates the proinflammatory cytokine response. Mercury is an immunosuppressant that kills T cells and could account for the observed lymphopenia in patients with poor outcomes.

Mercury has become an ubiquitous toxin in the modern world and each year our exposure to it increases.[46] We ingest it from seafood, dental amalgams, cosmetics, rice, corn syrup and vaccines.[47–53] Mercury is released into the biosphere from the burning of fossil fuels, gold mining, food processing and many other sources. As the mercury in the biosphere increases so does the mercury in our food, air and water. The mercury content in tuna is rising at four percent annually.[54] Six to eight percent of women of childbearing age have a mercury burden that is of concern. That number jumps to fifteen percent when accounting for transplacental mercury transportation.[55] Since the consumption of mercury containing products varies widely between individuals, the bioaccumulation of mercury also varies widely. In addition, some individuals are poor at excreting mercury once it is in their system.[56] That means certain individuals have very high levels of mercury relative to the rest of the population. Although mercury is recognized as the most toxic heavy metal, it is usually accompanied by other toxic heavy metals. Arsenic and lead are also infamous for immunosuppresion.[57–62] This is important because the bioaccumulation of multiple heavy metals potentiates the effect of mercury.[63–66] As our exposure to these immunosuppressive heavy metals continues to rise we should expect that infections that may have previously been harmless will become more serious. Mercury exposure is already shown to increase the virulence of viral infections.[67] It

would be no surprise that a highly contagious coronavirus combined with global mercury contamination would lead to a pandemic.

Coronavirus are frequently innocuous, as is COVID-19 to the majority of people it infects. That opens the possibility that what we are seeing is not a new deadly strain of virus but a virus interacting with an acquired immune disorder. What may be different is not the lethality of the virus but the fact that it is new, highly contagious, spreading rapidly, thereby triggering an avalanche of fatalities amongst those with a heavy metal induced immune disorder.

Science creates falsifiable hypotheses. If mercury is making people susceptible to COVID-19, those with an adverse reaction (e.g. thrombosis and cytokine storm) should have higher levels of mercury than those who do not have an adverse reaction. This is an easily testable and falsifiable hypothesis. Further research should compare heavy metal levels in the two groups. In addition to testing for mercury it should test for substances known to potentiate the effect of mercury such as other heavy metals and testosterone.

If it turns out that mercury is causing a vulnerability to COVID-19 then we have gained an invaluable tool for identifying who is at risk and a method to proactively reduce risk. In addition to saving priceless human life, this discovery could save the world economy tens of trillions of dollars.

Conclusions

The evidence suggests that mercury toxicity could make COVID-19 patients vulnerable to ARDS and death. Mercury can account for all of the disparate factors that are associated with poor COVID-19 outcomes.

Further research is needed to explore the hypothesis that mercury may be a critical factor in the COVID-19 pandemic.

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