Study shows substantial increase in childhood diabetes rates during COVID-19 pandemic

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A study published last month in JAMA Network Open found that since the start of the COVID-19 pandemic, the rate of new cases of type 1 diabetes mellitus in children has increased far beyond expectations. The study pooled the data of 17 prior, published studies to arrive at the result. Because of the size of the pooled data analysis, the study authors could conclude with confidence that the increase is real, and that it is significant.

Type 1 diabetes is an incurable disease with dramatic consequences. It frequently degrades the functioning of the eyes, kidneys, and nerves, ultimately resulting in the failure of these organs in a significant proportion of patients. It predisposes the patient to atherosclerosis that can result in heart attacks and strokes.

In the past, type 1 diabetes shortened lifespan dramatically. Today, with modern treatments, life expectancy among sufferers of type 1 diabetes has lengthened considerably in more developed countries, but still falls approximately 8 years short of average according to recent studies. In less developed economies, the disease remains very dangerous for most patients.

The incidence rate, or the number of new cases of type 1 diabetes in children per year in the United States divided by the total population, increased by 14 percent in the first 12 months of the pandemic versus the 12 months prior, while the incidence rate during the second year of the pandemic was 27 percent higher than the pre-pandemic period.

The incidence rate of type 1 diabetes had been increasing by 3-4 percent every year prior to the pandemic, meaning that the 14 percent increase during the first year of the pandemic is 3.5 times greater than the higher bound of 4 percent. Thus, the increase in incidence rate far outstripped the expected increase based on pre-pandemic historical trends.

The study looked at two sequential 12-month periods of the pandemic to avoid suffering from a potential bias known as the "catch up effect." This effect is caused both by barriers to accessing care resulting from the shutdown of significant portions of the healthcare system and by patient and parent reluctance to seek care. The result of the effect would be delayed diagnosis of type 1 diabetes onset in children during those periods with a "surge" in diagnoses resulting from past missed diagnoses due to patients not visiting the healthcare system.

The study avoided catch up effects by covering two, sequential 12-month time intervals where healthcare systems reopened and reluctance to seek care diminished, especially during the second year of the pandemic. Thus, any delayed diagnoses would be accounted for in the numbers and were not a significant factor in explaining the significantly increased incidence.

The study also attempted to look at the incidence rate of type 2 diabetes in children before and during the pandemic, but the data on this disorder were too limited to conclude one way or the other that there was a significant increase. Only 10 prior studies included in the meta-analysis reported on type 2 diabetes, and only one of those reported the population size (i.e., the denominator) needed to calculate incidence rate.

The results obviously beg the question of whether COVID-19 is directly responsible for the increase, and if so, what is the biological mechanism by which it does so? Notably, the SARS-CoV-2 virus attaches to the ACE2 cellular receptor in order to enter cells. This receptor appears in significant quantity on the insulin-producing beta cells of the pancreas that are affected by type 1 diabetes.

This process of viral entry via ACE2 receptors is known to damage beta cells, resulting in abnormal glucose levels in many COVID-19 patients. It also can cause beta-cell "exhaustion," a state where otherwise normal-appearing beta cells fail to produce insulin.

Given that seroprevalence studies show that the vast majority of children globally have been infected with SARS-CoV-2 at least once, this would seem to be the leading candidate for explaining the massive increase in type 1 diabetes.

Another hypothesis is that COVID-19 has numerous impacts on the immune system, and type 1 diabetes is nearly

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always an auto-immune disease whereby the patient's immune system destroys insulin-producing beta cells in the pancreas. However, the details of how COVID-19 might induce this autoimmune reaction are unclear and demonstrating such a connection requires substantial further study.

Nevertheless, the pandemic erased the seasonal pattern of type 1 diabetes onset. Prior to the pandemic, the incidence rate of type 1 diabetes was greater during winter months and lower during summer months. During the pandemic, however, the incidence rate has not significantly varied from winter to summer months, and the seasonal pattern has not returned despite re-opening of the healthcare system and the complete ending of lockdowns. These observations suggest, but do not prove, a direct effect of COVID-19 on type 1 diabetes onset.

Another hypothesis is that the measures taken to control the pandemic might have increased behaviors associated with the development of type 1 diabetes. In particular, it is suspected that weight gain in young children is associated with a risk of autoimmunity and thus development of type 1 diabetes. Children under lockdown might have had reduced physical activity levels and increased obesity, which could potentially contribute to the observed increase in type 1 diabetes. The study was not designed to provide evidence in support of or against this hypothesis, however.

The study also looked at the frequency with which new type 1 diabetes cases were associated with a condition called diabetic ketoacidosis (DKA) at the time of initial diagnosis. DKA is a life-threatening condition nearly always requiring hospitalization. If a child has DKA at the time of diagnosis of their type 1 diabetes, it is a potential indicator of both severity and duration of illness prior to diagnosis.

The study found that the increase in DKA at diagnosis seems to be more likely related to delays in seeking and having access to care. For example, one study included in the meta-analysis from Germany found that the frequency of DKA at initial diagnosis did not vary regionally based on varying levels of COVID-19 transmission, suggesting that uniform lockdown regulations across Germany were the main factor. Nevertheless, the evidence is not conclusive and further study is required here, too.

A key limitation of the study is that, due to the lack of information in the prior studies it had available, it could not examine the impact of access to healthcare and reluctance to seek care. Relatively few of the prior studies included variables on socioeconomic status, which is typically a proxy for access to care, especially in the United States where healthcare is not guaranteed to all.

Another limitation of the study is that the prior studies only represented certain regions of the world including North America, Europe, Asia, and Australia, but not Africa. Some of this bias could have resulted from limiting to articles published in English.

An accompanying editorial to the study adds an additional hypothesis to the mix to account for any effects of COVID-19 on the increase. In particular, it mentions that COVID-19 infection might directly change the makeup of gut bacteria (also called the microbiome) in children, or the pandemic control measures might have indirectly affected the microbiome through various means. Regardless, there is significant evidence that a less diverse microbiome is associated with type 1 diabetes patients, and thus could be the mediating effect.

Given the potential enormous toll of future morbidity and mortality from excess type 1 diabetes, further elucidation of the causes of the increased incidence is imperative. The indifference of the capitalist ruling elites to the long-term effects of COVID-19 has already led to potentially hundreds of millions worldwide suffering from Long COVID.

The additional potential toll of increased type 1 diabetes compounds dramatically the crimes of the ruling class, for they bear responsibility no matter which hypothesis proves correct. The hypotheses of direct effects from COVID-19—either through direct viral injury to beta cells, through induction of autoimmunity against beta cells, or through viral-induced changes to the makeup of the gut microbiota—most obviously implicate the ruling class's "let it rip" policies.

However, the ruling class is no less implicated via the indirect hypotheses. If the haphazard, ill-considered, poorly implemented, and arbitrarily managed lockdowns are responsible for long periods of inactivity or reduced gut microbiome diversity as causes of the increased incidence of type 1 diabetes, then the ruling class is also criminally liable. From the very beginning of the pandemic, the experience in China and other countries proved that a global elimination strategy was possible, but the prioritization of capitalist profits overrode this public health program.

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