Study adds to growing evidence of link between COVID-19 and type 1 diabetes in children

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A new study adds to the growing body of evidence that COVID-19 is responsible for the increased onset of type 1 diabetes mellitus in children. Type 1 diabetes is an autoimmune disease, which means that the immune system attacks the body's own cells. Autoimmunity is typically mediated by the development of antibodies that erroneously recognize cellular proteins as foreign. These antibodies are called "autoantibodies" for short.

The study found that the development of antibodies to COVID-19 was associated with the concurrent or subsequent development of the autoantibodies that are characteristically seen in type 1 diabetes. These autoantibodies—collectively referred to as "islet autoantibodies"—attack insulin-producing beta cells in the pancreas. Eventually the body cannot produce enough insulin, which results in blood glucose levels soaring to unhealthy and even dangerous levels.

The power of the study derives from the fact that it has followed children longitudinally since 2018 for the purposes of studying type 1 diabetes. It has collected blood samples from the children at least every six months from enrollment at age 4-7 months until the age of 6.5 years. More frequent collection occurred from enrollment until age 2 years. Thus, the study could pinpoint the timing at which islet autoantibodies and SARS-CoV-2 virus antibodies appeared in the children's blood.

The study had two key additional strengths. First, it enrolled children from four countries, so its results were much less likely to be impacted by unique circumstances or populations in any one country. Second, it also looked at the development of antibodies to an influenza virus relative to the onset of islet autoantibodies. Comparing against influenza, which has not been associated with type 1 diabetes onset, served as a control to rule out potential other, undetected effects and a possible general effect of viral infections.

The children in the study who developed SARS-CoV-2 antibodies had a risk of developing multiple islet autoantibodies that was a staggering 3.5 times higher than children who did not. It is known that approximately 70 percent of children who develop multiple islet autoantibodies will progress to type 1 diabetes mellitus within 10 years. Thus, the vast majority of these children would be expected to develop type 1 diabetes.

In the influenza virus comparison, 101 children developed influenza virus antibodies. No children in the study developed islet autoantibodies concurrently with or after developing influenza antibodies. Thus, the expected lack of association with influenza virus was abundantly confirmed. The association between infection and type 1 diabetes is therefore not a general viral phenomenon but very likely specific to SARS-CoV-2.

The study also computed incidence rates of developing islet autoantibodies, SARS-CoV-2 antibodies and influenza antibodies. The longitudinal tracking of the children over a years-long time interval was the key to enabling these calculations.

The incidence rates, during various time intervals, for developing SARS-CoV-2 antibodies were consistent with the temporal course of the pandemic. For example, the incidence rate was highest—81.7 per 100 person-years—during the Omicron variant surge from January to June 2022. It was zero prior to the pandemic and 4.4 per 100 person-years in its first phase from July to December 2020.

The incidence rates for developing islet autoantibodies did not vary significantly over time. The incidence rate for the development of influenza antibodies was 13.4 per 100 person-years prior to the pandemic, dropped dramatically to 4.0 during the first phase of the pandemic (during temporary lockdowns, mask mandates, initial school closures, etc.) and then rose again to 11.8 from January to June 2022, following the universal implementation of the criminal “let it rip” policies of the ruling class.

For children who developed SARS-CoV-2 antibodies, their subsequent incidence rate for developing islet autoantibodies was 7.8 per 100 person-years. This compares to an incidence rate of 4.0 for children who were negative for SARS-CoV-2 antibodies. The incidence rate ratio was therefore 2.3, which means that one would expect 2.3 times as many children per year to develop islet autoantibodies who are positive for SARS-CoV-2 antibodies versus children who are negative.

The attributable proportion of SARS-CoV-2 infection to the development of islet autoantibodies was 57 percent. That means that of all the children with SARS-CoV-2 antibodies who subsequently developed islet autoantibodies, approximately 57 percent of them would not have developed islet autoantibodies if they had not been infected.

The study also looked at the age at which children were infected and developed islet autoantibodies. The median age at which children developed SARS-CoV-2 antibodies was 18 months. Of the children who developed islet autoantibodies (n=60), 92 percent were positive by 24 months and 100 percent were positive by 30 months. One-third of them (n=20) have already progressed to type 1 diabetes.

The association between SARS-CoV-2 antibodies and development of islet autoantibodies was most pronounced at age 12 to 16 months. For these children, the incidence rate of developing islet autoantibodies (concurrent with or subsequent to developing SARS-CoV-2) was 36.5 per 100 person-years versus 4.4 for similarly aged children who were negative for SARS-CoV-2 antibodies, or an incidence rate ratio of 8.2.

One caution in interpreting the study is that the children enrolled in the original study of type 1 diabetes were selected for as already being at high risk for the disease. Thus, the study results show that in children already at high risk for developing type 1 diabetes, SARS-CoV-2 infection increased their risk even further. Whether SARS-CoV-2 infection increases risk for children not otherwise at high risk for the disease is a subject for future research.

Also, although the study included four countries, the four countries were all in Europe and thus still not representative of the full genetic variation of humanity worldwide. The four countries were the United Kingdom, Germany, Poland, and Sweden.

Nevertheless, the study shows a clear temporal relationship between SARS-CoV-2 infection and development of islet autoantibodies, which was not seen with influenza virus. Also, the age of children most affected is 12 to 16 months, a vulnerable population generally.

The results add to the evidence of the criminality of the ruling class, who have unleashed a novel virus far more dangerous than seasonal influenza on the world’s population, including its most vulnerable members. Robbing children of future potential by saddling them with not only the typical Long COVID sequelae, but now also a chronic disease with severe morbidity and mortality, is a monstrous act.