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Genetic commonality between COVID-19 and AlzheimerÃ¢â‚¬â„¢s disease

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The severity of the novel coronavirus or COVID-19 infection in individuals varies owing to the population’s diversity on a genetic level. A single nucleotide change in the DNA sequences of our genes can result in different variants of the same gene. This may or may not cause any disease but is definitely capable of changing the function of a gene. Thereby, in some cases, causing a disorder or increasing the risk of it. Simply put, we all have genes with different combinations of variations or DNA sequences. Further, 2’-5’-oligoadenylate synthetase 1 (OAS1) is a gene that exists in two types in humans; one that can detect and hinder the growth of the virus that causes COVID-19 infection, SARS-CoV-2, while the other form can not do so. It was reported that the hospitalised patients with the variant of the gene which cannot sense SARS-CoV-2 were more likely to have severe outcomes with the virus. Such variations or forms of one gene can dictate our ability to deal with such viruses. This does not mean that other already existing conditions like diabetes or hypertension will not contribute to the severity. However, studying the robustness and diversity of our immune system can lead to novel discoveries. One particular genetic variant of OAS1 can multiply the risk for Alzheimer’s disease (a neurodegenerative disease that causes the death of neurons) ultimately leading to dementia. Interestingly, this variation is similar to what contributes to severe COVID-19 outcomes. Identifying such related genetic mechanisms in innate immune cells can help pick out biomarkers to measure or track the diseases and target sites for novel drug treatment. However, it would be too early to say whether every individual with COVID-19 infection will exhibit symptoms of dementia later in life.

Keywords: COVID-19, AlzheimerÃ¢â‚¬â„¢s disease, SARS-CoV-2, Gene variant, 2Ã¢â‚¬â„¢5Ã¢â‚¬â„¢-oligoadenylate synthetase-1

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