

Alpha1 MZ Foundation - Information & Research

News & Research Update

Sep 21, 2024

Dear Subscribers,

It is mid-September. The summer is over, and the days are getting shorter and shorter. The kids are back in school, and it will get colder. So, the conditions for any virus spreading around are improving daily! And we all know that Influenza and Coronavirus variants are ready at the starting line for a fast sprint.

Most of us are smart enough to get vaccinations, especially those with reduced lung capacity and liver issues. However, even if you are a bit younger and have no lung or liver-related issues (yet), getting your vaccinations is recommended to avoid infections and, as such, lung and liver damage, including liver damage-related morbidities. (Prevention of any inflammation is the key for all Alpha's)

But how sensitive are we to virus infections and, in this case, particularly to the coronavirus? For that purpose, we found a paper describing our sensitivity to the coronavirus for Alpha1 MS MZ and ZZ.

In this paper, the researchers describe the key actors of coronavirus cell entry, which is crucial for advancing knowledge of how a virus selectively infects specific populations of cells and the potential therapeutic (medication) targets. In this case, the role of alpha-1-antitrypsin (AAT) in inhibiting protease-mediated SARS-CoV-2 entry and they explored the implications of AAT deficiency on susceptibility to different SARS-CoV-2 variants.

The researchers demonstrated that Alpha1 Antitrypsin is the major serum protease inhibitor, strongly restricting entry of the SARS-CoV-2 virus into the cell.

It showed that persons with an AAT-deficient genotype showed a reduced ability to inhibit entry of both coronavirus types, Wuhan-Hu-1 (WT) and B.1.617.2 (Delta). However, there was no difference in inhibiting B.1.1.529 (Omicron) cell entry.

It was also noticed that the most severe AATD genotypes have reduced serum inhibitory potential against protease-mediated entry of WT SARS-CoV-2 Spike. Still, this reduction is not observed for heterozygotes of the Z and S alleles. (So the M is doing its work, apparently)

Their findings highlight the importance of further investigating the complex interplay between proteases, antiproteases, and spike glycoprotein activation in SARS-CoV-2 and other respiratory viruses to identify potential therapeutic targets and improve the understanding of these diseases.

This means that Alpha1 patients with a low alpha1 antitrypsin level (without the "M") are more likely to get a Covid infection, but there was no difference in likelihood concerning the Omicron version.

Based on this paper, you could conclude that Alpha1 Antitrypsin forms a line of defense against virus cell entry (the infection), and we already know that Alpha1 Antitrypsin is also very, very important in fighting the inflammation caused by a virus infection.

Note: This paper is not conclusive, but it provides information that Alpha1 Antitrypsin plays a defensive role against virus infections. Which lack of defense is something most of us are experiencing.

Here is the link to the paper: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7430570/>

And, like always, enjoy the ride !!

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