



LETTER TO THE EDITOR

To The Editor:

I read with keen interest the article entitled “**Statin Drug Therapy May Increase COVID-19 Infection**” published on 1st April 2020.

The author was excellent in pointing out the importance of lipid rafts for the entry of enveloped viruses like the SARS-CoV-2. Lipid rafts are indeed vital for viral cell endocytosis. However, he has also pointed out that statins, via the upregulation in LDL receptor, may increase cholesterol content in cell membrane resulting in more lipid rafts. But studies have shown that statins destabilize lipid rafts by decreasing membrane cholesterol rather than increasing it.¹ This is in strong contraindication to the author's assumption. Statins may thus even decrease the viral endocytosis and further decrease the infectivity of SARS-CoV-2. Its anti-inflammatory pleiotropic action may even be of aid in many respiratory diseases including pneumonia², a severe complication of COVID-19.

The author has also mentioned the directly proportional interrelationship between statin intake and COVID-19 infection taking the example of Italy, the UK, the USA on one hand and India on the other. But the study shows that people with cardiovascular disease, as well as diabetes, are at risk of developing severe COVID infection³ for which statins are frequently prescribed. Statins may even alleviate the cardiac injury associated with COVID 19. So the approach of associating statin use rather than underlying cardiovascular diseases with COVID-19 infectivity seems very one dimensional if not a bit irrelevant.

SARS-CoV-2 is a novel virus; more research is needed to understand its pathogenesis and relation with a statin. However, we have known statins for a while now and know that it benefits more than it harms in many instances. Thus patients on statins should continue with their therapy because abrupt withdrawal with no conclusive evidence can cause more harm than good.

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