# Review of: "Low incidence of daily active tobacco smoking in patients with symptomatic COVID-19"

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The data is clear and, as a report in late March on hospitalized patients in China pointed before[1], there is little doubt now that, as much as it is somehow undigestible to us physicians, there is a positive effect of current smoking on the clinical outcome of Covid-19 disease. As for the hypothesis, we agree that nicotine, through an as yet unknown mechanism could be responsible for that beneficial effect. There is however a possible exlanation that needs to be taken into consideration. Indeed, we have recently proposed that asthma, and atopia in general, can be in itself an independent protective factor for the Covid-19 disease[2]. The model we have put forward places the Th2 response as seen in those conditions at the center of this protective role. We argue that the counterbalance of the Th1/Th2 immune response in favor of the Th2 axis and its main effector cell, the eosinophil, , as seen in most asthma and atopia patients, would be a deterrent for the SARS-CoV2 virus infection as well as for the complications thereof (ARDS above all). In this regard, cigarette smoking is a well known activator of the Th2 immune axis and an inducer of tissue and systemic eosinophilia. Since nicotine has been shown to activate the Th2 branch of the immune response[3], it would be interesting to test if nicotine could mediate these effects through the tilting of the Th1/Th2 immune branches towards Th2.

## References

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