Review of: "Successful Cessation Programs that Reduce Comorbidity may Explain Surprisingly Low Smoking Rates among Hospitalized COVID-19 Patients"

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I've read your article with great interest. While it is quite explanatory, it has some issues that need to be adressed. First and formost, as the authors suggest, hospitalised patients, who are active smokers, tend to quit smoking when being hospitalised, or prior to their hospitalisation, since they've got a diagnosis. Thus, it is evident that whichever protection, nicotine/cotinine offers, it is vanished, since it is no longer administered. After all, all hospitals prohibit smoking. Apart from that, the prevelence of smoking in hospitalised patients is relatively lower compared to non-smokers, even with the chance of miscalculation, taken into account. If we assume that there is the same degree of exposure to the virus, then we should assume that smokers should be more often hospitalised, due to comorbidities, one of which is high P(CO2), raphly 6-13%. Thus I do agree that turning to pharmaceutical nicotine is more beneficial, decause there are far less comorbidities. As for nicotine, it's 'protective' nature exerts over the stimulation of α7nAchR and upregulation of ACE2R. It is certainly not an infection inhibitor, but an entry inhibitor, since those receptors, according to data, are vital for spike's interactions, through the RBD and the RBM. Moreover, nicotine tends to protect people from PD, and in fact, the decrease in smoking polulation is a cause for increase in PD patients, because the agonist effect on α4β2 nAchR and other nAchRs, located in the basal gangglia, tend to increase dopaminergic activity in the region. These nAchRs are mostly prosynaptic, thus an agonist of them would cause efflux of neurotransmiters, from the dopaminergic neuron, where they are located, especially dopamine. After all, the main region which is mostly affected by PD is the basal gangglia and specifically the substantia nigra pars compacta, which contains dopaminergic neurons. If nicotine keeps the dopamine levels in the basal gagglia, high enough, there is less chance for neurodegeneration, most often manifested in it's initial form as atrophy, due to decreased stimulation of receptors. The causes of PD are not known, but nicotine tends to have a protective role and it is believed that this is due to increased signaling. After all, PD appears more often among the elderly, excluding of course, the cases of genetic susceptibility. In the elderly population, dopamine tends to decrease naturally. Nicotine prevents essentially just that. For more info, I'll provide the references.

1. doi: 10.1016/j.toxrep.2020.04.012
2. DOI: 10.1016/S1474-4422(16)30230-7
3. PMID: 31733690
4. https://doi.org/10.1038/aps.2009.65