Dear Editor,

Dental patients presenting with Sjogren’s syndrome (SjS) can be chronically debilitated by pain, dryness, fatigue, depression, anxiety and face an increased risk of lymphoma. By late 2019 research from Newcastle, UK had defined four SjS patient groups [1]:

- Those with highest symptom burdens and high lymphocyte counts benefited from hydroxychloroquine.
- Those suffering oro-ocular dryness and fatigue benefitted from rituximab.
- Patients with the lowest symptoms had the highest IgG levels.
- Pain and fatigue dominated a fourth group.


The gender bias in SjS exceeds 9 female: 1 male, whereas in Covid-19, with an equal infection rate, the morbidity and mortality is predominantly male. The following questions arise from such a relationship:

- In SjS, epithelial aridity and mucin alteration create a deranged host microbiome. Could a pathologically viscid milieu create an unassailable barrier to SARS-CoV2?
- In SjS, could viral transport be mitigated by reduction in nasolacrimal, oropharyngeal, and tracheal fluid flow towards the lungs with lowered viral carriage towards those susceptible cells with the threshold for viral binding to ACE 2 receptors not being reached?
- With Covid-19 infections, morbidity and mortality arises in 20% of cases from: Multicellular Interferon Specific Hypoactivity (MISH) and: Macrophage Activated Systemic Haemophagocytosis (MASH). The cytokine storm kills non-infected bystander cells, but this is predominantly in men with underlying connective tissue diseases (rarely reported in SjS) [3]. As female immunity is moderated by oestrogen, could the gender bias of SjS confer an advantage?
- Hepatitis C associated with SjS modulated immunity alters p53 localization and increases IL8 levels with neutrophils attracted towards an altered biome [3,4]. In SjS could such a mechanism mitigate SARS-CoV2 risks of leucopenia, hyper-reactivity and coagulability causing organ failure?
- Notwithstanding such complications in Covid-19, systemic auto-immune conditions increase the risk of hospitalisation, but anti-rheumatic drug use does not [5]. Could the immediate risk to patients with SjS from Covid-19 already be mitigated iatrogenically, or might the numbers of sub-clinical cases of SjS patients with Covid-19 be an underestimate, due to the social stigmatisation and isolation SjS patients already endure?

Even if acute Covid-19 complications are alleviated by such mechanisms, for both SjS and Covid-19; chronic fatigue, depression and the risk of neoplasia still follow from virally initiated auto-immunity and this is a risk regardless of mitigating co-factors.

Notwithstanding the paucity of research at the cross-roads of these two illnesses, the complications and consequences for patients with SjS remain unbearable.

Dental research with our medical colleagues must build on what is known if the relationship between Sjogren’s syndrome and Covid-19 is to be better understood.

References
