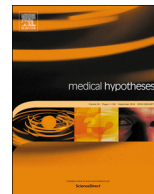




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Letter to Editors

Oxytocin as a potential defence against Covid-19?

To the editor,

According to the research advances on COVID-19, we propose that oxytocin (OT) a nonapeptide hormone acting in the body and the brain, constitutes a biological target against coronavirus infection, especially in high-risk population, with underlying conditions including diabetes, high blood pressure, cardiovascular issues and obesity.

OT exerts a dual effect by mobilizing the immune defense potential, and by suppressing pathogenic responses due to over-reactions of the innate immunity. In human, increase in plasma OT levels reported in the early phases of infectious disease, can limit the excessive pro-inflammatory and oxidative stress reactions, by decreasing interleukins levels in the macrophages [1]. OT exerts a metabolic functional role in cardiovascular disease (by regulating, heart rate, blood pressure, muscle contraction), in diabetes (via glucose uptake and insulin secretion), and obesity (food intake and satiety), in gastric injury (by anti-ulcer properties), and in osteoporosis (with bone formation and resorption) (see [Supplementary data](#)). These effects can be explained by the presence of local OT producing cells (brain, heart, gastrointestinal tract), and by extensive expression of OT receptors.

Of particular interest to Covid-19, is the nitric oxide (NO), which is a key signalling molecule acting as a host response modulator in viral infections (see [Supplementary data](#)). In humans, activation of the OT receptor, which are expressed by endothelial cells in the pulmonary artery, produce a vasodilatory effect via stimulation of the nitric oxide (NO) pathway [2]. In animal models of acute lung injury, OT exposure reduces the expression of inflammatory proteins in the lung tissue [3]. Literature also reveals that viral infections in human (including influenza) attenuate OT receptor expression, indicating a key role for the OT system for human health [4]. As OT secretion and levels seems to adjust to pathogen threat and infection, to elicit initial adaptive inhibitory responses and to restore the host homeostasis, OT administration, which is safely deliverable in humans (by nasal spray or intravenous injection) (see [Supplementary data](#)) could be used as a prospective therapeutic agent for Covid-19 viral replication and infection.

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Prior presentation

No data from this manuscript were presented in a previous scientific meeting.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.mehy.2020.109785>.

References

- [1] Wang P, et al. Oxytocin-secreting system: a major part of the neuroendocrine center regulating immunologic activity. *J Neuroimmunol* 2015;289:152–61.
- [2] Thibonnier M, et al. Human vascular endothelial cells express oxytocin receptors. *Endocrinology* 1999;140(3):1301–9.
- [3] An X, et al. Protective effect of oxytocin on LPS-induced acute lung injury in mice. *Sci Rep* 2019;9:2836.
- [4] Liu Y, Conboy I. Unexpected evolutionarily conserved rapid effects of viral infection on oxytocin receptor and TGF- β /pSmad3. *Skeletal Muscle* 2017;7:7.

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