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Correction

Correction: Unpuzzling COVID-19: tissue-related signaling pathways associated with SARS-CoV-2 infection and transmission

OPEN ACCESS

The authors of the original article "Unpuzzling COVID-19: tissue-related signaling pathways associated with SARS-CoV-2 infection and transmission" (Clin Sci (Lond) (2020) 134(16), DOI: 10.1042/CS20200904) would like to correct Figures 2 and 3 of their article as they have noted that these were inverted at article submission. The figures are presented with their correct order here.

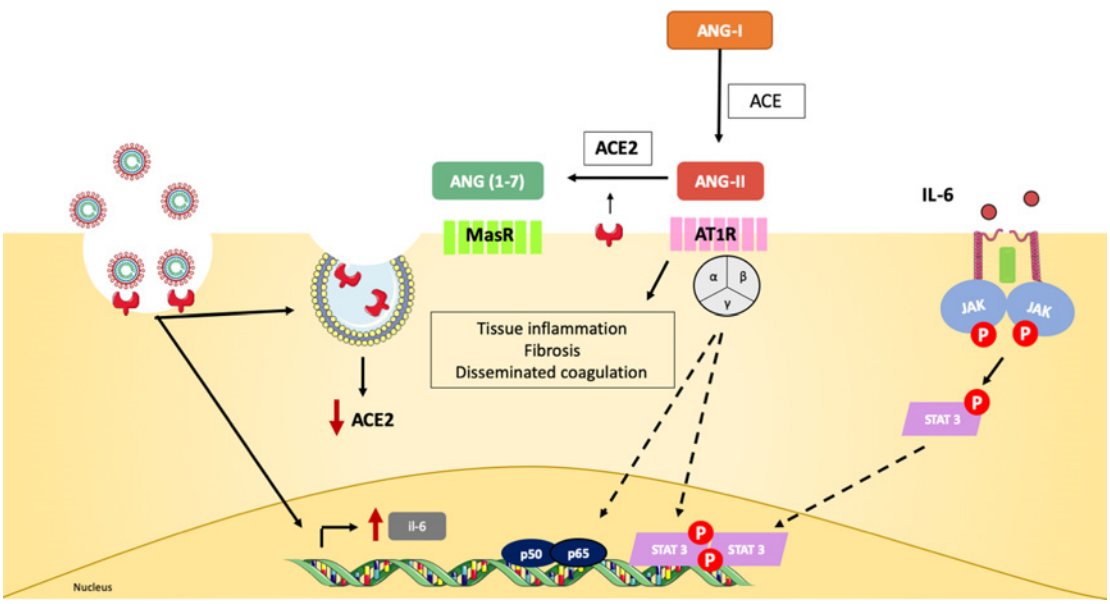

Figure 2. Canonical ACE2 pathway links multiple organ damage in COVID-19

SARS-CoV-2 infection down-regulates ACE2 expression and leads to the production of pro-inflammatory mediators, such as IL-6 [1]. Angiotensin-I (Ang-I) is converted into Ang-II by the ACE in the extracellular space. ACE2 is able to further cleave Ang-II to Ang(1-7), which binds MasR receptors on the cell surface and promotes anti-inflammatory, vasodilation and anti-fibrotic effects [1]. Since ACE2 is down-regulated during viral infection, this event will lead to the accumulation of Ang-II and binding to AT1R receptors on cellular membrane. AT1R signals through JAK-STAT and induces fibrosis, pro-inflammatory gene expression and vasoconstriction [2,3]. Multiple organs express ACE2 and are target for SARS-CoV-2. As they lose ACE2-mediated protection, Ang-II signaling contributes to the pathological findings observed in COVID-19 patients, such as disseminated coagulopathy and acute tissue damage [4].

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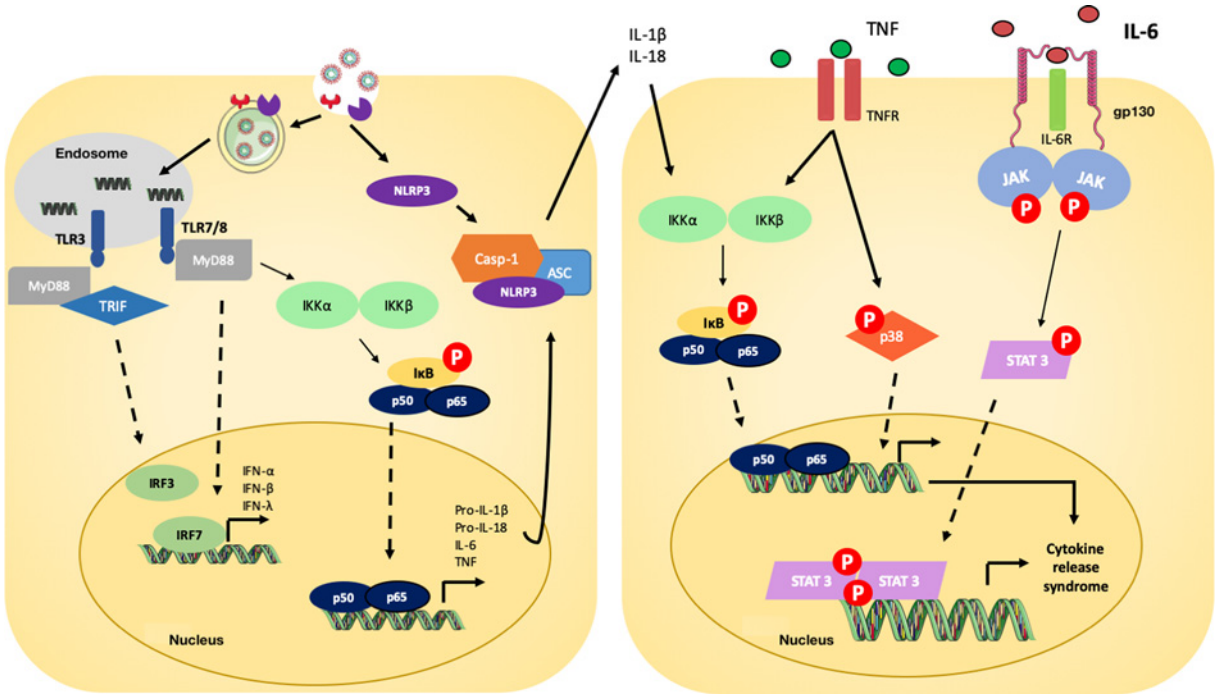

Figure 3. Signaling pathways involved in COVID-19 pathophysiology

Toll-like receptors (TLRs) 3 and TLR 7/8 recognize SARS-CoV-2 RNA and initiate the inflammatory cascade via type I and type II IFN gene expression and NF-κB nuclear translocation [5,6]. Via NF-κB, the expression of multiple pro-inflammatory genes is stimulated, including pro-IL-1β, pro-IL-18, TNF and IL-6 [7–9]. The virus is also recognized by cytoplasmic NLRP3, which forms, together with ASC and caspase-1 (Casp-1), the inflammasome complex that will cleave and release mature forms of IL-1β and IL-18 [10]. The cytokines IL-1β, IL-18 and TNF bind to specific receptors and promote further NF-κB nuclear translocation and phosphorylation of p38 MAPK, which will lead to great expression of pro-inflammatory cytokines and chemokines [11,12]. IL-6, an important player in COVID-19, binds IL-6R and gp130 receptors to activate JAK/STAT-3 pathway and then contribute to the CRS observed in COVID-19 patients [13].

The authors apologise for any inconvenience caused to the reader.

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