Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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May IL-17 have a role in COVID-19 infection?

Dear Editor,

SARS-Corona-Virus-2 related disease (COVID-19) which started in Wuhan, China on December 2019, is spreading rapidly throughout the world [1]. COVID-19 clinical spectrum is variable and may lead to respiratory failure, multiorgan and systemic manifestations [2]. COVID-19 patients showed lower leukocyte numbers, abnormal respiratory findings, and increased levels of plasma pro-inflammatory cytokines such as interleukin (IL)1-β, IL1RA, IL6, IL8, IL10 with Th1-Th17 cells appearing as the main source [3]. That is why recently tocilizumab, a monoclonal antibody blocking IL-6 action has been shown to be effective in COVID-19 subjects with cytokine storm risk [4]. Indeed, pro-inflammatory cytokine storms have been observed in critically COVID-19 patients which progress to multiple organ dysfunction and death [4,5]. Therefore, cytokine storms early treatment and prevention is of crucial importance. In this context, the debate whether blocking other cytokines could reduce COVID-19 impact is growing [6]. Particularly, a cytokine that may be related to IL-6 in the context of viral infection is IL-17. Indeed, Hou et al. found in murine viral models that the excessive IL-6 level promotes the generation of Th17 cells, and the resulting IL-6 and IL-17 synergistically promote viral persistence by protecting virus-infected cells from apoptosis [7]. Another example is represented by influenza virus infection where high-mortality acute lung injury is associated with an increase in neutrophils in the airspace promoted by IL-17 [8]; IL-17 receptor antagonist (RA) knockout mice recruited fewer neutrophils to the airway in response to influenza A virus with a decreased mortality and lower immune-related lung injury [8]. Targeting IL-17 in acute lung injury due to viral infection could be beneficial also because blocking IL-17 did not impair influenza virus clearance. Indeed, main Th17 cell effector cytokines were up-regulated in laboratory-confirmed A(H3N2) influenza infected humans supporting that increased amounts of IL-17 may be implicated in influenza pathogenesis and immune control, predicting disease severity [9]. A recent review showed that IL-17 functions are crucial in different settings of viral infections with its targeting being an effective alternative treatment to suppress viral infections and minimizing tissue pathology in several human diseases [10]. Finally, Huang et al. observed that IL-17 increased in intensive-care COVID-19 patients vs non intensive-care and controls with Zumla et al. hypothesizing that blocking IL-17 could have the potential to improve COVID-19's aberrant immune response and acute respiratory distress syndrome-related mortality [3,11]. Indeed, a Chinese clinical trial evaluating an anti-IL17 drug approved for psoriasis (ixekizumab) is already running [12]. Despite the effect of blocking IL-17 on Th2 response should be deeper investigated since SARS-CoV-2 also seems to stimulate Th-2 cytokines production (IL-4 and IL-10) that suppress Th1/Th17 mediated inflammation [3], taken together all these data further underline the need of investigating on IL-17 blocking role in COVID19 which appears as a potential promising therapeutic target.

Conflict of interest

All authors declare to not have conflict of interest as well as funding sources to declare.

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