CORRECTION

Publisher Correction: Role of DAMPs in respiratory virus-induced acute respiratory distress syndrome — with a preliminary reference to SARS-CoV-2 pneumonia

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Genes & Immunity (2022) 23:245; https://doi.org/10.1038/s41435-022-00190-8

Correction to: *Genes & Immunity* https://doi.org/10.1038/s41435-021-00140-w, published online 17 June 2021

Abstract

When surveying the current literature on COVID-19, the "cytokine storm" is considered to be pathogenetically involved in its severe outcomes such as acute respiratory distress syndrome, systemic inflammatory response syndrome, and eventually multiple organ failure. In this review, the similar role of DAMPs is addressed, that is, of those molecules, which operate upstream of the inflammatory pathway by activating those cells, which ultimately release the cytokines. Given the still limited reports on their role in COVID-19, the emerging topic is extended to respiratory viral infections with focus on influenza. At first, a brief introduction is given on the function of various classes of activating DAMPs and counterbalancing suppressing DAMPs (SAMPs) in initiating controlled inflammation-promoting and inflammation-resolving defense responses upon infectious and sterile insults. It is stressed that the excessive emission of DAMPs upon severe injury uncovers their fateful property in triggering dysregulated life-threatening hyperinflammatory responses. Such a scenario may happen when the viral load is too high, for example, in the respiratory tract, "forcing" many virus-infected host cells to decide to commit "suicidal" regulated cell death (e.g., necroptosis, pyroptosis) associated with release of large amounts of DAMPs: an important topic of this review. Ironically, although the aim of this "suicidal" cell death is to save and restore organismal homeostasis, the intrinsic release of excessive amounts of DAMPs leads to those dysregulated hyperinflammatory responses—as typically involved in the pathogenesis of acute respiratory distress syndrome and systemic inflammatory response syndrome in respiratory viral infections. Consequently, as briefly outlined in this review, these molecules can be considered valuable diagnostic and prognostic biomarkers to monitor and evaluate the course of the viral disorder, in particular, to grasp the eventual transition precociously from a controlled defense response as observed in mild/moderate cases to a dysregulated life-threatening hyperinflammatory response as seen, for example, in severe/fatal COVID-19. Moreover, the pathogenetic involvement of these molecules qualifies them as relevant future therapeutic targets to prevent severe/fatal outcomes. Finally, a theory is presented proposing that the superimposition of coronavirus-induced DAMPs with non-virus-induced DAMPs from other origins such as air pollution or high age may contribute to severe and fatal courses of coronavirus pneumonia.

This article is part of the Special Issue "Immunology of cell death in cancer & infection", Guest Editor: Professor Abhishek D. Garg, Katholieke Universiteit Leuven, Belgium. It was unintentionally published in issue 22-3 (2021).

You can access the article via this link: https://www.nature.com/articles/s41435-021-00140-w. We apologise for the inconvenience.

Published online: 1 December 2022