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INVITED REVIEW

CELL DAMAGE IN INFLAMMATORY AND INFECTIOUS SITES MIGHT INVOLVE A COORDINATED "CROSS-TALK" AMONG OXIDANTS, MICROBIAL HAEMOLYSINS AND AMPIPHILES, CATIONIC PROTEINS, PHOSPHOLIPASES, FATTY ACIDS, PROTEINASES AND CYTOKINES (AN OVERVIEW)

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PROLOGUE

Voluminous literature exists on the mechanisms by which cells and tissues are destroyed in infectious and inflammatory sites. Microbial toxins,[1–5] their enzymes[6–9] and cell-wall components,[10–12] leukocyte and platelets-derived hydrolases and oxygen radicals,[13–18] cationic polypeptides,[19–24] arachidonic acid and metabolites, cytokines,[25,26] coagulation factors and fibrinolysin,[27] cytotoxic antibodies and complement components,[28] nitric oxide,[29] platelet activating factors,[30] killer lymphocytes,[31] as well as additional, still undefined, agonists have all been incriminated as putative agents capable of injuring cells. Special attention has, however, been devoted in the last decade to the role of reactive oxygen species (ROS)[13] as the main agonists responsible for causing tissue destruction in inflammatory conditions. Todate, over 100 human disorders have been connected with an excessive generation of ROS[13,14,15,32–47]

The pivotal role played by leukocyte-derived ROS, in cellular injury, was often supported by showing that their removal ameliorated and even totally prevented the initiation of cellular damage (see below). The exact nature of the ROS involved in the initiation of cellular damage is, however, still controversial. While superoxide has been advocated as the main toxic oxygen radical,[32] H_2O_2, OH· ROO·, NO and additional more exotic radicals were considered to be the main culprits.[14,19,33–47] Screening the

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