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Molecular Damage and Repair Mechanisms in COPD

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Message from the Guest Editor

There is a growing list of molecules that can act as DAMPs, but they all have two things in common: 1) they are released into the extracellular space upon cellular damage and cell death, and 2) they can trigger a pro-inflammatory response by activation of pattern recognition receptors (PRRs). DAMPs can be derived from various sub-cellular origins, like the mitochondrion, the cytoplasm, the nucleus, the cytoskeleton or even the extracellular matrix. Furthermore, DAMPs can bind to and activate a plethora of PRRs, including Toll-like receptors (TLRs), the receptor for advanced glycation end-products (RAGE) or purinergic receptors.

The goal of this Special Issue is to gather evidence for the various roles of DAMPs and their receptors in the pathophysiology of COPD. Which DAMPs are associated with emphysema, or chronic bronchitis? Which DAMPs are related to the development of exacerbations? Does the remodeled and damaged extracellular matrix in COPD patients lead to the increased release of ECM-DAMPs? And can we use specific inhibitors for DAMPs or their receptors as treatment for COPD patients? These are all questions that could be addressed in this Special Issue of *Cells*.



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Special Issue



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