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Short Communication

Asbestosis Situation Impacted by COVID-19 Pandemic

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Asbestos-fiber inhalation, a known cause of fibrogenic and neoplastic pulmonary injury, such as asbestosis [1,2] that triggers the development of this lung disease after more than 15 years of exposure [3-6]. These various serum biomarkers, such as 1B, interleukin (IL)-18, RANTES, tumor-necrosis-factor (TNF)-alpha (Figure 1) [6], (Figure 2 and 3) [7] with sustained inflammation in non-ill-exposed-asbestos persons [3], including immune-system dysregulation, chronic inflammation and carcinogenesis [8]. In 2020, a study was conducted in Italy compared to the period 2015 - 2019 demonstrated decreased deaths of asbestosis and COVID-19 among people below 80 years of age, whereas demonstrating increasing trend in persons with 80 and above with a relative mortality risks of 1.17 for asbestosis and 1.10 for malignant pleural mesothelioma (MPM) (Figure 4) [9]. A recent study revealed no difference of the various follow-up variables, such DLCO diffusion, the 6-minute walk test, computed tomography (CT) changes, and spirometry parameters [10]. In multivariate analysis, asbestos is not related to the COVID-19 severity, but in univariate analysis, it was related [10].

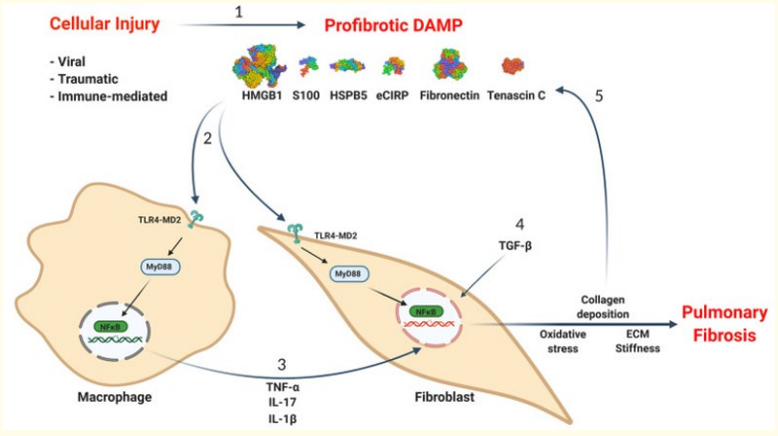

Figure 1: *Demonstrating the interplay of DAMPs, TLR4, and proinflammatory cytokines in pulmonary fibrosis centered around macrophages and fibroblasts. (1) Injury to the cells either from a viral infection, chemical/mechanical trauma, or immune-mediated damage causes the release of DAMPs in the microenvironment. (2) DAMPs stimulate and activate macrophages and fibroblasts through a TLR4-MD2 → MyD88-mediated pathway. (3) Activated macrophages release proinflammatory cytokines such as TNF-α, IL-17, and IL-1β in the tissue microenvironment that, (4) along with TGF-β, activate fibroblasts to become profibrotic and deposit collagen and ECM components like fibronectin and tenascin-C. This causes stiffness of ECM and oxidative stress in the microenvironment, which (5) causes the release of more DAMPs leading to the vicious cycle of pulmonary fibrosis. DAMP: Damage-Associated Molecular Patterns; HMGB1: High-Mobility Group Box 1; eCIRP: Extracellular Cold-Inducible RNA-Binding Protein; HSPB5: Heat Shock Protein B5; TLR4: Toll-Like-Receptor 4; MD2: Myeloid Differentiation Factor 2; MyD88: Myeloid Differentiation Primary Response 88; NF-κB: Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells; ECM: Extracellular Matrix [6].*

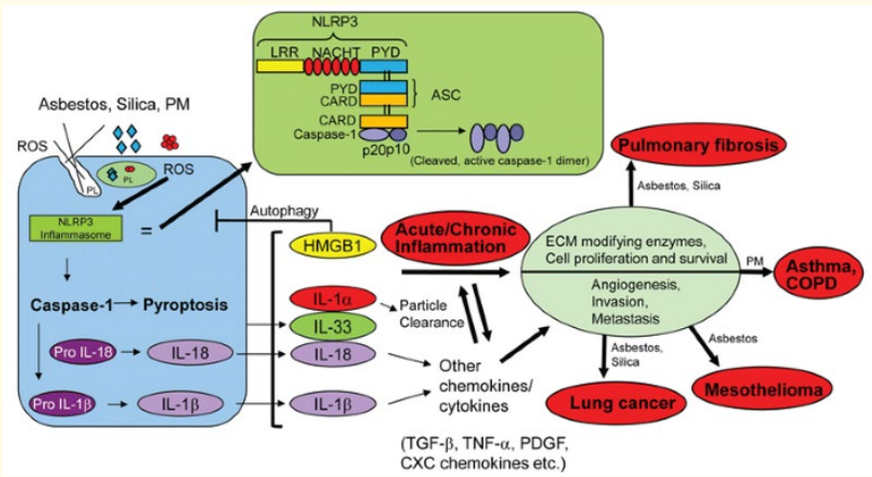

Figure 2: *Demonstrating a general schematic diagram describing the components, assembly, and biologic events linked to activation of the NLRP3 inflammasome. Exposure to asbestos, erionite, CS, or PM may prime and activate the NLRP3 inflammasome via multiple mechanisms and the mechanisms. These fibers and particles induce dose-related damage to the cell membrane at high concentrations, are phagocytized, and can rupture phagolysosomes (PL). These processes and iron-dependent reactions on the particle surface may result in the elaboration of ROS via many routes. Inflammasome-associated caspase-1 activation leads directly to the maturation and secretion of IL-1β and IL-18. Another important function of inflammasomes is the induction of caspase-1 dependent pyroptosis which is a form of cell death characterized by both apoptosis and necrosis. This results in release of IL-1β and IL-18 as well as other inflammatory mediators such as IL-1α and HMGB1. These chemokines and cytokines either directly or indirectly lead to acute and chronic inflammation, the latter resulting in various particle and fibre-associated lung and pleural diseases. Although IL-33 is sometimes released after pathogenic particle exposures, it is unclear whether or not it plays a critical role in IL-1β maturation or production [7].*

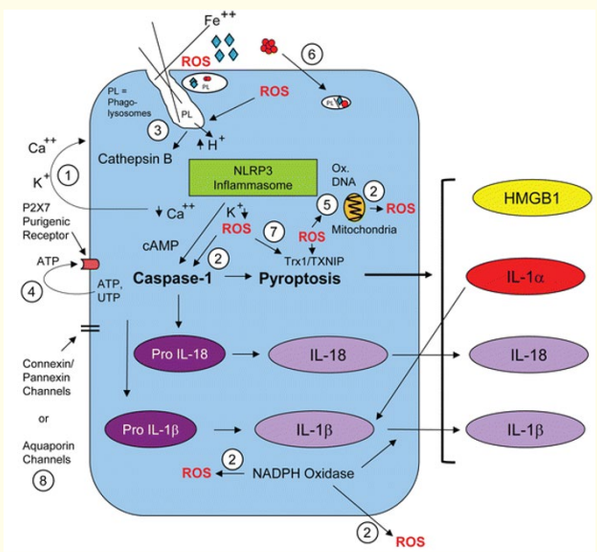

Figure 3: *Demonstrating the general mechanisms of inflammasome activation by pathogenic particles and fibers. Note that the complex cascades of specific proteins in these pathways are not presented. The numbers refer to individual pathways that include: 1) modifications in intracellular levels and export of K+ and Ca++; 2) formation and release of ROS both intracellularly and extracellularly that destabilize phagolysosomes, activate caspase-1 and 3) phagolysosomal disruption and increases in intracellular H+. In addition to these classical pathways observed with diverse agents, studies with pathogenic particles and fibres show inflammasome priming or activation by: 4) release of ATP, ADP, and increases in purigenic receptor signaling; 5) elaboration of ROS by a multiplicity of pathways including generation mitochondrial oxidants/antioxidant enzymes and oxidation of mitochondrial DNA; 6) dose-dependent particle uptake; 7) Trx-1 oxidation and TXN1P disassociation; and 8) increases in cell volume via particle uptake, aquaporin channels and/or modulation of connexin/pannexin gap junctions that also are cytoskeletal organization proteins linked to both inflammation and immune regulation [7].*

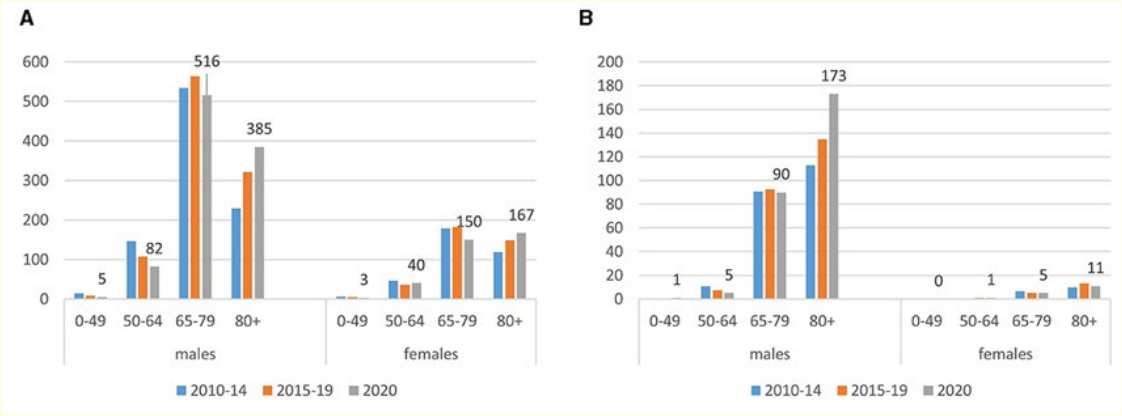

Figure 4: *Demonstrating number of deaths involving malignant pleural mesothelioma [MPM: (A)] and asbestosis (B), by sex and age group, Italy, 2020. Deaths and the average annual number of deaths observed in 2010 - 2014 and 2015 - 2019 [9].*

In conclusion, nevertheless, independent relationship between asbestos exposure and COVID-19 severity is still to be defined.

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