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Acute lung injury (ALI), and acute respiratory distress syndrome (ARDS) are two major forms of acute lung injury (ALI) that are caused by excessive pulmonary inflammation due to, for example, ischemia/reperfusion injury, pneumonia, or sepsis. ALI is a life-threatening disease that frequently progresses to ARDS, which has a mortality of approximately 40-50%. ARDS is characterized by a non-cardiogenic pulmonary edema associated with the accumulation of neutrophils and increased endothelial permeability. Approximately 34% of patients with ARDS die during their hospital stay, and the rate of death among patients who do not require mechanical ventilation ranges from 25 to 50% within the

first 2 months after ARDS onset. The hallmark of the progression of ALI is the accumulation of neutrophils in the alveolar space, which causes lung edema that develops into diffuse alveolar damage (DAD), i.e., an overt inflammatory response. The onset of ALI may be defined by the occurrence of DAD within 48-72 hours after the onset of injury. The pathogenesis of ALI and ARDS is thought to be linked to the migration of neutrophils and their subsequent release of proteases and oxygen radicals. Two major types of neutrophils are involved in the pathogenesis of ALI: one type is the classical or “primary” neutrophil and the other type is the previously poorly characterized “secondary” neutrophil. The classical neutrophil (also called “primary” neutrophil) is referred to as the “macrophage of the

lungs” because they are the predominant circulating myeloid cell in the blood. The classical neutrophil is

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