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RESEARCH LETTER
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Acute respiratory distress syndrome is associated with impaired alveolar macrophage efferocytosis

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Shareable abstract (@ERSpublications)
ARDS patients have decreased alveolar macrophage efferocytosis, which is associated with increased alveolar inflammation, and may contribute to worse clinical outcomes, including mortality. Upregulation of efferocytosis may offer a therapeutic strategy. https://bit.ly/2Q7REdM

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To the Editor:

Acute respiratory distress syndrome (ARDS) is an inflammatory disorder of the lungs, with sepsis as the predominant aetiology. Despite advances in ventilation strategies, mortality for moderate to severe ARDS remains at 40–46% [1]. ARDS is associated with neutrophil influx into alveoli. Persistently high neutrophil and low alveolar macrophage (AM) numbers in bronchoalveolar lavage (BAL) fluid are associated with greater mortality [2]. While the inflammatory alveolar environment of early ARDS initially delays apoptosis, these neutrophils ultimately undergo apoptosis within alveoli [3]. Efficient efferocytosis of apoptotic neutrophils by AMs is critical for resolution of inflammation [3]. Apoptotic neutrophils may accumulate in ARDS due to defective AM efferocytosis and/or overwhelmed efferocytosis capacity, then undergo secondary necrosis, releasing inflammatory mediators into the alveolar space [4]. This may contribute to the prolonged inflammation observed in ARDS. No study has previously assessed AM efferocytosis in ARDS; however, monocyte-derived macrophages (MDMs) from ARDS patients do have impaired efferocytosis [5]. We investigated whether ARDS patients have impaired AM efferocytosis and increased alveolar neutrophil apoptosis.