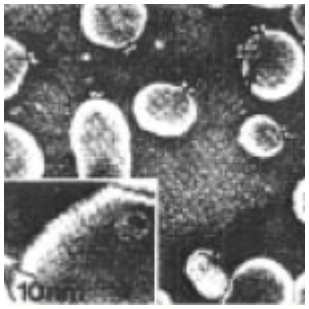
Why older people may succumb more readily to pneumonia



Age is known to be related to the levels of increased inflammatory cytokines in the blood and tissues. General inflammatory conditions are linked to diminished anti-bacterial immunity and overall health. In the most recent edition of PLoS Pathogens, Puchta and colleagues highlight the epidemiological link between older adults who have higher than age-average levels of inflammatory cytokines and "at increased risk of acquiring, becoming hospitalized with and dying of *Streptococcus pneumoniae* pneumonia".

The authors explore why this is the case by mimicking age-related inflammation using a mouse model. They show that increased Tumour Necrosis Alpha (TNF) causes premature release of monocytes from the bone marrow and that these immature monocytes are, in turn, capable of producing more inflammatory cytokines (notably IL-6 and TNF) when stimulated with bacterial products. The authors then went on to colonize the animals with *S. pneumonia* and found that chronic exposure to TNF with increasing age resulted in a decrease in the maturity of circulating monocytes. This was directly linked to susceptibility to infection and the mice could no longer clear the bacteria.

When they removed the effect of TNF with drugs, or removed the inflammatory cytokines, antibacterial immunity was restored. The authors thus conclude that monocytes not only contribute to age-associated inflammation, but are also affected by these conditions and cannot provide anti-bacterial immunity due to the resulting altered stage of monocyte "maturity and function". Although this is an experimental model, immature monocytes are also found in older humans with elevated inflammation and this study possibly provides a potential mechanism to age-associated susceptibility to pneumonia.

Puchta, A. et al. 2016. TNF Drives Monocyte Dysfunction with Age and Results in Impaired Anti-pneumococcal Immunity. *PLOS*.