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Resumo	This study evaluated the role of endogenous and exogenous annexin A1 (AnxA1) in the activation of the NLRP3 inflammasome in isolated peritoneal neutrophils. C57BL/6 wild-type (WT) and AnxA1 knockout mice (AnxA1$^{-/-}$) received 0.3% carrageenan intraperitoneally and, after 3 h, the peritoneal exudate was collected. WT and AnxA1$^{-/-}$ neutrophils were then stimulated with lipopolysaccharide, followed by the NLRP3 agonists nigericin or ATP. To determine the exogenous effect of AnxA1, the neutrophils were pretreated with the AnxA1-derived peptide Ac_{2-26} followed by the NLRP3 agonists. Ac_{2-26} administration reduced NLRP3-derived IL-1β production by WT neutrophils after nigericin and ATP stimulation. However, IL-1β release was impaired in AnxA1$^{-/-}$ neutrophils stimulated by both agonists, and there was no further impairment in IL-1β release with Ac_{2-26} treatment before stimulation. Despite this, ATP- and nigericin-stimulated AnxA1$^{-/-}$ neutrophils had increased levels of cleaved caspase-1. The lipidomics of supernatants from nigericin-stimulated WT and AnxA1$^{-/-}$ neutrophils showed potential lipid biomarkers of cell stress and activation, including specific sphingolipids and glycerophospholipids. AnxA1 peptidomimetic treatment also increased the concentration of phosphatidylserines and oxidized phosphocholines, which are lipid biomarkers related to the inflammatory resolution pathway. Together, our results indicate that exogenous AnxA1 negatively regulates NLRP3-derived IL-1β production by

	neutrophils, while endogenous AnxA1 is required for the activation of the NLRP3 machinery
Fomento	