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Poly(ADP-ribose) polymerase-1 inhibitor in carrageenan-induced lung inflammation in mice (Article)

Ahmad, S.F.^a , Zoheir, K.M.A.^{a,b}, Ansari, M.A.^a, Korashy, H.M.^a, Bakheet, S.A.^a, Ashour, A.E.^a, Al-Shabanah, O.A.^a, Al-harbi, M.M.^a, Attia, S.M.^{a,c} ^aDepartment of Pharmacology and Toxicology, College of Pharmacy, King Saud University, Riyadh, Saudi Arabia^bDepartment of Cell Biology, National Research Centre, Cairo, Egypt^cDepartment of Pharmacology and Toxicology, College of Pharmacy, Al-Azhar University, Cairo, Egypt

Abstract

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Increasing indication is unveiling a role for poly(ADP-ribose) polymerase (PARP)-1 in the regulation of inflammatory/immune responses. The aim of the present study was to determine the potential anti-inflammatory effects of PARP-1 inhibitor 5-aminoisoquinolinone (5-AIQ) to explore the role of PARP-1 inhibitor in a mouse model of carrageenan-induced lung inflammation. A single dose of 5-AIQ (1.5mg/kg) was administered intraperitoneally (i.p.) 1h before λ -carrageenan (Cg) administration. We assessed the effects of 5-AIQ treatment on CD25⁺, GITR⁺, CD25⁺GITR⁺, IL-17⁺ and Foxp3⁺ cells which were investigated using flowcytometry in pleural exudates and heparinized blood. We also evaluated mRNA expressions of IL-6, TNF- α , IL-1 β , IL-10, CD11a, I-selectin (CD62L), ICAM-1, MCP-1, iNOS and COX-2 in the lung tissue. We further examined the effects of 5-AIQ on the key mediators of inflammation, namely COX-2, STAT-3, NF-kB p65, PARP-1, I κ B- α and IL-4 protein expression in the lung tissue using western blotting. The results illustrated that the numbers of T cell subsets, IL-17⁺ cytokine levels were markedly increased and Foxp3⁺ production decreased in the Cg group. Furthermore, Cg-induced up-regulation of adhesion molecules, pro-inflammatory mediators and chemokine expressions. Western blot analysis revealed an increased protein expressions of COX-2, STAT-3 NF-kB p65 and PARP-1 and decreased I κ B- α and IL-4 in the Cg group. PARP-1 inhibitor via 5-AIQ treatment reverses the action significantly of all the previously mentioned effects. Moreover, histological examinations revealed anti-inflammatory effects of 5-AIQ, whereas Cg-group aggravated Cg-induced inflammation. Present findings demonstrate the potent anti-inflammatory action of the PARP-1 inhibitor in acute lung injury induced by carrageenan. © 2014 Elsevier Ltd.

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 monocyte chemotactic protein 1
 nicotinamide adenine dinucleotide adenosine diphosphate ribosyltransferase 1
 nicotinamide adenine dinucleotide adenosine diphosphate ribosyltransferase 1 inhibitor
 STAT3 protein transcription factor FOXP3 tumor necrosis factor alpha unclassified drug
 5-aminoisoquinolinone autacoid cell adhesion molecule cyclooxygenase 2 cytokine
 enzyme inhibitor forkhead transcription factor Foxp3 protein, mouse
 glucocorticoid induced tumor necrosis factor receptor inducible nitric oxide synthase
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 Tnfrsf18 protein, mouse

EMTREE medical terms:

adult animal experiment animal model animal tissue antiinflammatory activity
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5-aminoisoquinolinone; Carrageenan; Cell Adhesion Molecules; Cyclooxygenase 2; Cytokines; Enzyme Inhibitors; Forkhead Transcription Factors; Foxp3 protein, mouse; Glucocorticoid-Induced TNFR-Related Protein; Inflammation Mediators; Interleukin-17; Interleukin-2 Receptor alpha Subunit; Isoquinolines; Nitric Oxide Synthase Type II; Poly(ADP-ribose) Polymerases; RNA, Messenger; Tnfrsf18 protein, mouse

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