21N-pm03 MAPK regulated endocytosis of neutrophils in differentiated human neutrophils and mouse primary neutrophils

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associated with respiratory and cardiovascular diseases. We previously reported the biological effects of PM in vivo, and although neutrophils play an important role in initiating inflammation, few reports have focused on the relationship between PM inhalation and immune responses. To investigate mechanisms for phagocytosis of PM in neutrophils, well-established human promyelocytic leukemia cell line, HL-60, was used for preparation of differentiated neutrophils, and

applied for elucidation of detail mechanisms. [Methods] For preparation of differentiated neutrophils, HL-60 cells were treated with DMSO (1.4%) or ATRA (1 µM) for 3 days. Flow cytometry was used to analyze phagocytosis. Western blotting was used to analyze expression of proteins.

[Results] To study molecular events, MAPK inhibitors (U0126 for MEK, SB239063 for p38, and INK inhibitor) were used. Inhibition of MEK can reduce phagocytosis of PM (size from 0.3 to 1 uM) and bio-particles (S. aureus, invitrogen) in DMSO or ATRA-differentiated HL-60.

[Conclusion] HL-60 can be differentiated into neutrophil like cells both by DMSO or ATRA. MAPK has important roles in this process.