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In vivo Pharmacology: Acute Tobacco Smoke Induced Pulmonary Neutrophilia In Mice

Species, strain, sex:	mouse, BALB/c, male
No. of animals per group:	n=8
Pharmacological control:	roflumilast
Routes of administration:	upon request
Treatment mode:	prophylactic, therapeutic
Duration of dosing:	upon request

Main read-outs:

- total and differential cell count in bronchoalveolar lavage fluid (BALF)

Facultative read outs:

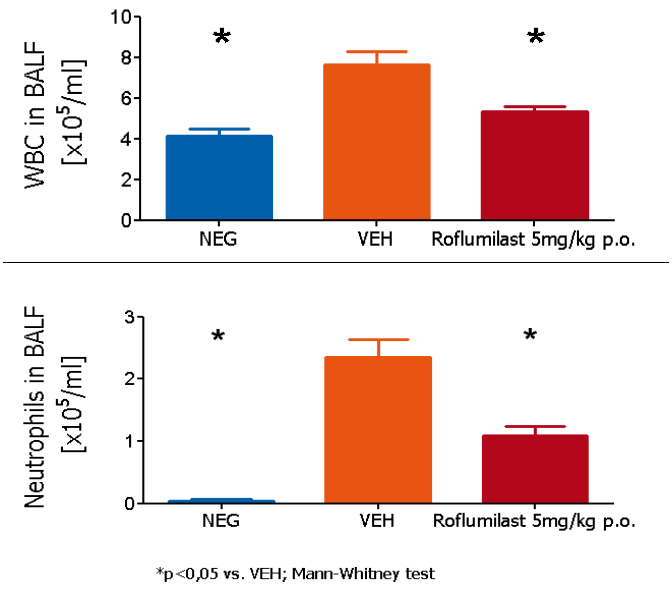
- inflammatory mediators in lung homogenate
- histopathology
- immunohistochemistry

Smoking-associated COPD is characterized by inflammation, changes affecting small airways, and emphysema.

In the present model, animals are exposed to tobacco smoke for 3 days. Inflammation is assesed through analysis of BALF cell count, cytokines in the lung homogenate and lung histology. Changes affecting epithelial cells are studied in depth employing immunohistochemistry.

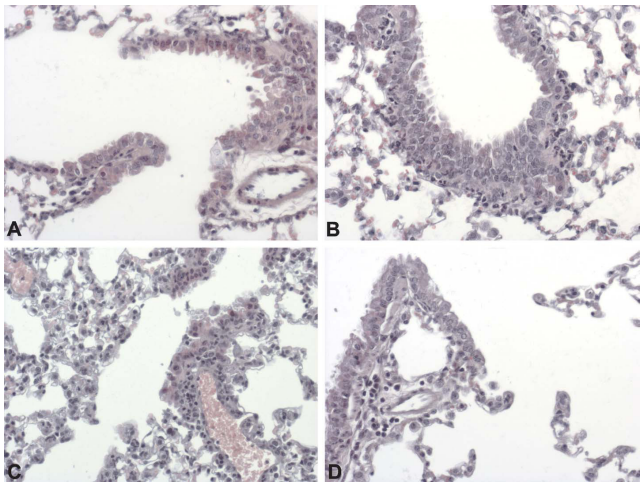
A recovery period can be included in the study upon request.

Effect of roflumilast on BAL cell influx in acute tobacco smoke pulmonary neutrophilia



*p<0,05 vs. VEH; Mann-Whitney test

Tobacco smoke induced inflammation in lungs during a 7day recovery period



(A) 1 day (B) 4 days (C) 5 days (D) 7 days post smoking; Tox Pathol (2012) 40:1169

References

Čužić S, Bosnar M, Dominis Kramarić M, Ferenčić Ž, Marković D, Glojnarić I and Eraković Haber V. Claudin-3 and Clara Cell 10 kDa Protein as Early Signals of Cigarette Smoke-Induced Epithelial Injury along Alveolar Ducts. *Toxicol Pathology* (2012) 40:1169

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