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### Proteinase activated receptor 2 (PAR2) modulation of murine airway function

Black, Kimberly; MacKenzie, Andrew; Dunning, Lynette; Crilly, Anne; Brzeszczyska, Joanna; McGarvey, Lorcan; Thornbury, Keith; Goodyear, C.S.; Lockhart, John; Litherland, Gary

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**British Association for Lung Research | Summer Meeting**

“Lung injury and Repair”

University of Cambridge, Selwyn College

10<sup>th</sup>-12<sup>th</sup> September 2019

**Abstract Submission Form – Deadline Friday 5th July 2019**

Please write clearly in block capitals

First Name	Kimberly
Surname	Black
Address	Institute of Biomedical and Environmental Health Research, University of the West of Scotland, Paisley Campus, Paisley. PA1 2BE.
Tel	0141 848 3000
E-mail	kimberly.black@uws.ac.uk
Title	Ms
Authors	K. Black, A. MacKenzie, L. Dunning, A. Crilly, J. Brzezczynska, L. McGarvey, K. Thornbury, C.S. Goodyear, J.C. Lockhart, G.J. Litherland

Abstract (see notes to authors)

**PROTEINASE ACTIVATED RECEPTOR 2 (PAR2) MODULATION OF MURINE AIRWAY FUNCTION**

K. Black<sup>1</sup>, A. MacKenzie<sup>1</sup>, L. Dunning<sup>1</sup>, A. Crilly<sup>1</sup>, J. Brzezczynska<sup>1</sup>, L. McGarvey<sup>2</sup>, K. Thornbury<sup>3</sup>, C.S. Goodyear<sup>4</sup>, J.C. Lockhart<sup>1</sup>, G.J. Litherland<sup>1</sup>

<sup>1</sup>Border & REgions Airways Training Hub, Institute of Biomedical and Environmental Health Research, School of Health & Life Sciences, University of the West of Scotland, Paisley PA1 2BE. <sup>2</sup>Centre for Experimental Medicine, Wellcome-Wolfson Institute for Experimental Medicine, School of Medicine, Dentistry and Biomedical Sciences, Queens University Belfast, Belfast, Northern Ireland. <sup>3</sup>Smooth muscle Research Centre, Dundalk Institute of Technology, Dundalk, Ireland. <sup>4</sup>Institute of Infection, Immunity & Inflammation, University of Glasgow, Glasgow, Scotland.

Hyperactivity, inflammation and hyperplasia/hypertrophy of airway smooth muscle (ASM) limit airflow and are key features of chronic obstructive pulmonary disease (COPD). Proteinase activated receptor-2 (PAR2) is a key modulator of inflammatory responses in respiratory disease such as asthma, and promotes ASM relaxation. However, the role of the receptor in ASM in conditions such as COPD is not well understood<sup>1</sup>.

The aim of this study was to use immunohistochemistry and an *ex vivo* murine airway myograph assay to confirm the presence in murine lung of PAR2 as a functional airway modulator.

PAR2 (detected using Alomone APR-32 antibody) was present on both murine airway and lung tissue. Following exposure to a disease relevant challenge (oxidative stress), PAR2 activation with trypsin (10 U ml<sup>-1</sup>), was observed to induce a higher relaxation in airway segments pre-contracted with acetylcholine (1 μM) using wire myography. Specifically, tracheal segments subjected to oxidative stress showed a significantly higher percentage relaxation (mean ± SEM; 53.8%±10.3%) compared with control (30.8%±7.9%; *p*=0.05; *n*=4). A higher percentage relaxation was also observed in bronchial segments (oxidative 55.1%±10.7% vs. control 33.9%±1.2%; *p*=0.07; *n*=4). The trypsin-induced relaxation was confirmed to be PAR2-dependent since relaxation to trypsin was significantly reduced in tracheal and bronchial tissue derived from PAR2-knockout mice.

Taken together this data confirms that PAR2 is present and suggests the receptor contributes functionally to the modulation of ASM tone in mice.

1. Sokolova E, Reiser G. A novel therapeutic target in various lung diseases: Airway proteases and protease-activated receptors. *Pharmacol Ther.* 2007;115(1):70–83.

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- Introduction to the study
- Methods
- Results, including data
- Conclusions

#### **Format**

All abstracts must:

- Be 10-point Arial font
- Be no more than 250 words
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**Abstracts should be emailed to Andrew Savage (ads85@medschl.cam.ac.uk) by 17.00h on Friday 5 July, 2019.**