Abstract S127 Table 1 Physiotherapy contact and rehabilitation milestone data

Parameter	
Physiotherapy contacts	
Total physiotherapy contacts	656 (100)
Contact only, assessment +/- advice provision	85 (12.9)
Respiratory treatments only	452 (68.9)
Combined respiratory treatment and exercise therapy	24 (3.7)
Exercise therapy only	95 (14.5)
Physiotherapy contacts/day	
Overall contacts	0.97 (0.83–1.9)
Contact only, assessment +/- advice provision	0.12 (0.0–0.22)
Respiratory treatments only	0.67 (0.5–0.87)
Combined respiratory treatment and exercise therapy	0.0 (0.0–0.05)
Exercise therapy only	0.1 (0.0–0.2)
Physiotherapy treatment time (mins)	
Respiratory treatments only	30 (30–40)
Combined respiratory treatment and exercise therapy	40 (30–60)
Exercise therapy only	60 (50–80)
Physiotherapy milestones	
Time to first contact (days)	2.0 (1.0–2.0)
Time to first exercise therapy activity (days)*#	8.0 (5.0–12.0)
Number of patients achieving SOEOB*	29 (58)
Time to achieve SOEOB (days)*	9.0 (5.5–12.0)
Highest exercise therapy activity level*	3 (0–4)

Data are reported as n (%) or median (interquartile range). n = 50. n = 19 did not receive exercise therapy during admission. *n = 31 received exercise therapy during admission. #Exercise therapy activities were categorised according to a standardised classification: 0. Passive Exercises, 1. Bed Exercises, 2. Passive sitting out of bed, 3. Sitting of edge of bed, 4. Standing, 5. Transfer to chair, 6. Marching on the spot, 7. Walking with assistance of 2, 8. Walking With assistance of 1, 9. Walking with gait aid, 10. Walking independently. Abbreviations: SOEOB = sitting over edge of bed.

Asthma – basic mechanisms

S128 DOES THE TIME OF DAY OF ALLERGEN CHALLENGE AFFECT THE DEGREE OF INFLAMMATORY RESPONSE IN THE MURINE LUNG?

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Introduction Circadian variations in immune parameters such as lymphocyte proliferation, antigen presentation and cytokine gene expression have been described. Recently, an association between the molecular circadian clock, immunity and inflammation has been recognised. To date research in this area has focussed on the innate immune response. However, the time at which the lung is exposed to an allergen might significantly affect the ability of the lung to mount an adequate immune response. Furthermore, this line of investigation might provide valuable insight into asthma, a common disease with a strong circadian rhythm.

Method We used a well-defined mouse model of allergic lung inflammation, the ovalbumin challenge model. After initial intra-peritoneal sensitisation, 4 groups of C57Bl/6 mice received ovalbumin challenge at one of four time points, repeated at the same time for 3 consecutive days. The timepoints used were: 1 am, 6 am, 1 pm or 6 pm. Measurements of airway hyper-responsiveness were recorded, bronchoalveolar lavage was performed and lungs were harvested for immunohistochemistry and for gene

Abstract S128 Figure 1 Lung section from c57BL/6 mice challenged with ovalbumin at different time points

analysis by PCR. Experiments were repeated in clock gene knock-out mice, *rev-erba*^{-/-}.

Results

- C57BL/6 mice challenged at 1 am develop increased AHR
- This suggests that allergic airway inflammation is under clock control
- *Rev-erba*^{-/-} mice show identical responses, suggesting that REV-ERB α is not critical to the development of airway inflammation in this model
- C57BL/6 mice challenged at 6 pm develop the most profound inflammatory response within the lung (Figure 1)
- This suggests that allergic inflammation within the lung is caused by a different mechanism to that within the airway, yet is also under clock control

Discussion Understanding the mechanism underlying clock control of allergic lung inflammation and its possible translation to asthma, provides a new therapeutic opportunity. Furthermore, targeting earlier stages in the circadian pathway might narrow the therapeutic window for timing of existing drug delivery, reducing drug dose and minimising side effects by giving shorter acting agents and the most efficacious time of day.

S129 INFLAMMATORY CYTOKINES INFLUENCE RESPIRATORY EPITHELIAL ANTI-VIRAL IMMUNE RESPONSES VIA INDUCIBLE EPIGENETIC CONTROL OF RIG1 EXPRESSION: A MODEL OF EARLY LIFE ORIGINS OF ASTHMA?

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The development of asthma is linked to early life environmental exposures and the occurrence of severe viral infections. Rapid maturation of adaptive immunity from a tolerant (Th2) to an anti-infective (Th1) state occurs in the neonatal period. We hypothesised that the airway inflammatory milieu, driven by the maturing immune response to environmental exposures may have important effects on the development of anti-viral innate immunity at the level of the epithelium. We studied whether the inflammatory environment of the airway epithelium modulates gene expression via epigenetic regulation of anti-viral genes as a model of the