Abstract S85 Table 1 30 and 90 day readmissions and A&E attendances by group

	Declined to attend PEPR n = 34	Completed PEPR n = 2	p value (Chi^2)
30 day readmission%(n=)	18% (n = 6)	0% (n = 0)	p = 0.046
90 day readmission%(n=)	29% (n = 10)	5% (n = 1)	p = 0.031
30 day A&E attendance%(n=)	15% (n = 5)	0% (n = 0)	p = 0.072
90 day A&E attendance%(n=)	38% (n = 13)	5% (n = 1)	p = 0.007

2013, Thorax). We secured funding to pilot PEPR in our local DGH population. Here, we present the initial five months data.

Aims and objectives This study aimed to investigate the impact of PEPR on exercise tolerance, QoL and health care utilisation in a local DGH population.

Methods Data were collected prospectively from successive patients referred for PEPR between December 2012 and May 2014. Outcome measures consisted of ISWT and QoL (CAT). Healthcare utilisation was measured through 30 and 90 day readmission and A&E attendance rates. Descriptive statistics and significance values were calculated in SPSS (version 22) using paired t-test and Chi^2.

Results 64 patients were referred to PEPR. 53% (n = 34) decline to attend, 15% (n = 10) failed to complete the programme. Subsequently 31% (n = 20) patients completed PEPR which is comparable to standard PR. Exercise tolerance was significantly improved (difference between the means 46 m 95% CI +/-33 m p = 0.009). QoL was significantly improved (difference between the means 5.4 95% CI +/-3.1 p = 0.002). Table 1 demonstrates the impact of PEPR on healthcare utilisation. Both 30 and 90 day readmissions were significantly reduced. 90 day A&E attendances were significantly reduced. Average LoS following readmission in the group who declined PEPR was 11 days compared to an average LoS following readmission in PEPR group of 1 day. Considering the savings associated with bed days alone and staffing expenses the cost benefit of PEPR was £21309 pa.

Conclusions Results suggest PEPR in a DGH population has a significant impact on QoL and exercise tolerance with reductions in healthcare utilisation and associated cost benefits.

Pulmonary infection: discovery science

S86 THE INFLAMMATORY RESPONSE TO STREPTOCOCCUS PNEUMONIAE IS EXAGGERATED BY THE POLYSACCHARIDE CAPSULE

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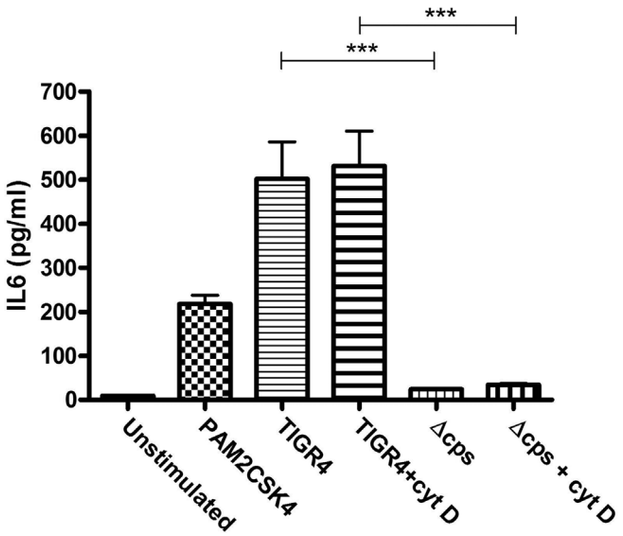
Streptococcus pneumoniae infections characteristically cause a high degree of inflammation. The *S. pneumoniae* polysaccharide capsule prevents opsonophagocytosis and is essential for virulence. The capsule might also be expected to reduce the host's inflammatory response by inhibiting bacterial interactions with pro-inflammatory signalling proteins eg toll-like receptors (TLR), but this has not previously been investigated. Using isogenic unencapsulated strains and *in vitro* and *in vivo* models of infection we have characterised capsule effects on the inflammatory response to *S. pneumoniae*.

Surprisingly, although the unencapsulated (Δ*cps*) *S. pneumoniae* strain was much more sensitive to phagocytosis by macrophages and induced a stronger NFκB response by human monocyte derived macrophages (MDMs) it caused similar levels of stimulation of a TLR2 reporter cell line as the encapsulated strain TIGR4. In addition, microarrays demonstrated increased transcription of pro-inflammatory cytokines by MDMs in response to TIGR4 compared to the Δ*cps* strain, and quantitative PCR and ELISAs confirmed stronger TNF, IL1β, and IL6 responses by MDMs to TIGR4. Furthermore, compared to the Δ*cps* strain the TIGR4 strain caused greater neutrophil recruitment and higher cytokine levels in the lungs in a mouse model of pneumonia, as well as higher serum cytokine levels with worse hypotension in a rat model of sepsis. Additional *in vitro* experiments excluded antibody, complement, pneumolysin, the inflammasome, and lectin-mediated signalling as mechanisms driving differences in inflammatory responses between TIGR4 and Δ*cps*. Expression of the TIGR4 capsule in *Streptococcus mitis* did not increase MDM or murine inflammatory responses. Notably, preventing phagocytosis with cytochalasin D did not alter differences in the inflammatory response between TIGR4 and the Δ*cps* strains, and *in silico* analysis suggested the Δ*cps* strain activated a wider range of transcription factors.



Abstract S86 Figure 1 Monocyte derived macrophages incubated with media or 10μM cytochalasin D for 30 min. Cells washed in PBS, then incubated with bacteria or controls for 6 h. Supernatants assayed for IL6 by ELISA

Overall, the data indicate that unencapsulated *S. pneumoniae* stimulate a wider range of host cell signalling pathways than encapsulated bacteria, some of which are likely to be anti-inflammatory. Hence the capsule, rather than reducing inflammation, causes increased pro-inflammatory responses and subsequent disturbances to host physiology during *S. pneumoniae* infection. Targeting the mechanisms responsible for capsule-dependent inflammation could offer novel treatment options for reducing the morbidity and mortality associated with *S. pneumoniae* infections.

S87 A FUNCTIONAL COMPARISON OF NEONATAL AND ADULT NEUTROPHIL RESPONSES TO RESPIRATORY SYNCYTIAL VIRUS

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S86 The Inflammatory Response To Streptococcus Pneumoniae Is Exaggerated By The Polysaccharide Capsule

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