Streptococcus pyogenes in a similar manner to that observed in studies with *Streptococcus pneumoniae*.

Methods Human positively-isolated CD14+ monocyte-derived macrophages (MDM), an established model for alveolar macrophages, were incubated with an optimised dose of carbon particulates. Phagocytosis of *Streptococcus pneumoniae* and *pyogenes* was assessed by bacterial culture of macrophages lysates. Autologous CD3+ T cells were co-cultured with infected MDM for 24 h. Expression of cell surface markers, T cell activation and uptake of FITC beads were assessed by flow cytometry.

Results Phagocytosis of *Streptococcus pneumoniae* and *pyogenes* by carbon loaded MDM was impaired compared to non-carbon loaded MDM (18.8% reduction, p = 0.01). Phagocytosis of FITC beads was also shown to be reduced by the carbon loaded MDM (10% reduction, p = 0.03).

Carbon loading decreased MDM surface expression of the phagocytic receptor CD36 ($p < 0.01$) and decreased the surface expression of antigen presentation molecules HLA-ABC and HLA-DR (p = 0.04 and 0.03 respectively). Addition of *S. pyogenes* increased expression of HLA-DR only on the cell surface of carbon loaded MDM (p = 0.02). Preliminary results suggest that autologous T cells express IFNγ in response to coculture with infected MDM and that this response is enhanced in carbon loaded MDM.

Conclusion We conclude that carbon exerts immunomodulatory effects on MDM phagocytosis of *Streptococcus pyogenes* and subsequent antigen presentation. Further investigation of this subject in the context of both chronic lung infection and RHD is therefore warranted.

REFERENCE

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S73 VIRUSES ASSOCIATED WITH COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN – A LARGE PROSPECTIVE STUDY IN THE POST-PREVENAR 13 ERA

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10.1136/thoraxjnl-2016-209333.79

Introduction and objectives Community-acquired pneumonia (CAP) is a common childhood illness and frequent reason for hospital admission. CAP may be caused by bacteria or viruses. Many studies have relied on administrative records that typically provide poor quality retrospective aetiological data. We have performed a large prospective study to investigate the aetiology of CAP in the 'post-Prevenar 13 era'. We wished to describe the current epidemiology of viral CAP and compare this with data from studies that used similar methodology in the same population in 2009–2011 (post-Prevenar 7) and 2001–2002 (pre-Prevenar 7).

Methods Children aged under 16 years admitted to a large paediatric centre, between January 2015 and June 2016, with a chest radiograph meeting WHO criteria were eligible. Following informed consent nasopharyngeal aspirate, nasal swab, sputum or endotracheal secretions were collected. Influenza A and B, Human Rhinovirus, Human Metapneumovirus, Adenovirus, Parainfluenza 1 – 4 and Respiratory Syncytial Virus (RSV) were tested using real-time PCR assays. Comparisons were made using appropriate non-parametric tests.

Results A total of 148 children were recruited, median age 2.7 years, IQR 1.5–5.3. Samples from 63 participants (43%) were positive for a virus and 7 cases had co-infection with 2 viruses. Children that tested positive for a virus were significantly younger (median 2.1 years, IQR 1.2–3.2, versus 3.7 years, IQR 1.9–6.9, p = 0.0005). When compared with previous studies there was no significant change in the virus detection rate. The breakdown of individual viruses is shown in the table, no significant changes were identified apart from a reduction in RSV.

Conclusions These results represent the most comprehensive data available on viruses associated with CAP in children in the UK in the 'post-Prevenar 13 era'. They confirm that there has not been a significant shift in the viruses associated with children requiring admission to hospital. Our results also suggest that the introduction of the live attenuated intranasal influenza vaccine to children, which began in 2013, is yet to have a significant effect on the frequency of influenza detected in children with CAP. Future surveys will be important for ongoing evaluation of the viral aetiology of CAP.

REFERENCE

1 Elemraid MA, *et al*. Aetiology of paediatric pneumonia after the introduction of pneumococcal conjugate vaccine. *Eur Respir J* 2013;**42**(6):1595–603.

Abstract S73 Table 1 Demographic data, evidence of sensitisation and clinical parameters

	2001–2002 study[1]			2009–2011 study[1]			2015–2016 study			
Virus	**Number tested (percentage positive)**	**<5 years**	**5–16 years**	**Number tested (percentage positive)**	**<5 years**	**5–16 years**	**Number tested (percentage positive)**	**<5 years**	**5–16 years**	**Frequency change (2009–2011 study to 2015–2016 study)**
RSV	213 (15.0%)	29	3	147 (21.1%)	30	1	147 (10.2%)	15	0	p = 0.015
Influenza A and B viruses	213 (6.1%)	9	4	149 (12.1%)	11	7	147 (10.2%)	12	3	p = 0.713
- H1N1		*NT*	*NT*	*141 (5.0%)*	*4*	*3*	*147 (4.8%)*	*6*	*1*	*p = 1*
Adenovirus	213 (6.1%)	11	2	145 (6.9%)	10	0	147 (10.2%)	11	4	p = 0.404
Parainfluenza 1–4	158 (3.2%)	5	0	141 (4.3%)	5	1	147 (6.1%)	9	0	p = 0.599
Human metapneumovirus	48 (0%)	0	0	141 (0.7%)	1	0	147 (3.4%)	5	0	p = 0.214
Rhinovirus	Not tested	NT	NT	141 (8.5%)	10	2	147 (7.5%)	8	3	p = 0.829
Total		54	9		67	11		60	10	

NT = not tested, note: children with bronchiolitis were excluded