REFERENCES

1 Cates EC, Fattouh R, Johnson JR, *et al.* Modeling responses to respiratory house dust mite exposure. *Contrib Microbiol* 2007; 14: 42–67.
2 Swirski FK, Sajic D, Robbins CS, *et al.* Chronic exposure to innocuous antigen in sensitized mice leads to suppressed airway eosinophilia that is reversed by granulocyte macrophage colony-stimulating factor. *J Immunol* 2002; 169: 3499–3506.
3 Van Hove CL, Maes T, Joos GF, *et al.* Prolonged inhaled allergen exposure can induce persistent tolerance. *Am J Respir Cell Mol Biol* 2007; 36: 573–584.
4 Maunsell K, Wraith DG, Cunnington AM. Mites and house-dust allergy in bronchial asthma. *Lancet* 1968; 1: 1267–1270.
5 Platts-Mills TA, Wheatley LM. The role of allergy and atopy in asthma. *Curr Opin Pulm Med.* 1996; 2: 29–34.
6 Roche N, Chinet TC, Huchon GJ. Allergic and nonallergic interactions between house dust mite allergens and airway mucosa. *Eur Respir J* 1997; 10: 719–726.
7 Hatzivlassiou M, Grainge C, Kehagia V, *et al.* The allergen specificity of the late asthmatic reaction. *Allergy* 2010; 65: 355–358.
8 Haddad el-B, Underwood SL, Dabrowski D, *et al.* Critical role for T cells in Sephadex-induced airway inflammation: pharmacological and immunological characterization and molecular biomarker identification. *J Immunol* 2002; 168: 3004–3016.
9 Penido C, Castro-Faria-Neto HC, Vieira-de-Abreu A, *et al.* LPS induces eosinophil migration *via* CCR3 signaling through a mechanism independent of RANTES and Eotaxin. *Am J Respir Cell Mol Biol* 2001; 25: 707–716.
10 Johnson JR, Wiley RE, Fattouh R, *et al.* Continuous exposure to house dust mite elicits chronic airway inflammation and structural remodeling. *Am J Respir Crit Care Med* 2004; 169: 378–385.

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Risk factors of community-acquired pneumonia in children

To the Editors:

In a recent issue of the *European Respiratory Journal*, TEEPE *et al.* [1] published their interesting observations of determinants of community-acquired pneumonia (CAP) in children in primary care. The authors included 107 children with either radiologically or clinically diagnosed CAP treated as outpatients in four Dutch healthcare centres in 1999–2008, and compared the potential determinants of CAP between the cases and 321 controls from the same area with no CAP during the study period. In adjusted analyses, lower age (OR 1.14), asthma history (OR 3.57) and the number of previous visits for upper respiratory tract infections (URTIs) (OR 1.80 for one or two episodes and 2.46 for more than three episodes) were independently associated with CAP. The authors concluded that the association between CAP and the number of URTIs can be explained by infection susceptibility of the individuals [1].

In the discussion, the authors mentioned that their study was the first to explore the determinants of CAP in children in primary care. Actually, their study was the second one.

As part of the Savo Pneumonia Study performed in 1981–1982 in Eastern Finland [2, 3], we also analysed risk factors for CAP in children aged 3 months to 15 yrs [4]. The design of the study was prospective and strictly population-based. During a surveillance period of 12 months, all CAP cases were registered in a small manufacturing town and three rural municipalities. Chest radiographs were studied in all clinically presumptive cases, and only radiologically confirmed CAP cases were included in the analyses. The incidence of CAP was 36 per 1,000 per yr for <5 yrs and 16 per 1,000 per yr for 5–15-yr-old children [2]. 51% in the younger and 11% in the older age group were treated in hospital. *Pneumococcus* caused 28% of the cases overall [3], and *Mycoplasma* <10% at <5 and >50% at >5 yrs of age, over 80% of mycoplasmal cases being treated at home [5].

To evaluate the possible risk factors for paediatric CAP, identical standardised questionnaires were sent to the parents of the 201 children with CAP and to 250 controls from the same four municipalities. In all, 176 (88%) cases and 233 (93%) controls answered.

In adjusted analyses, significant risk factors for CAP were a history of recurrent (at least three) URTIs within 12 months (OR 5.5), a history of wheezing at any age (OR 5.3) and a history of otitis media and tympanocentesis before 2 yrs of age (OR 3.6) in <5-yr-old children. The significant risk factors in 5–15-yr-old children were a history of recurrent URTIs within 12 months (OR 5.5) and a history of wheezing at any age (OR 5.3).

In line with the study by TEEPE *et al.* [1], wheezing tendency and susceptibility to respiratory infections were the only significant determinants of paediatric CAP in our prospective, population-based study [4]. Young age is an indisputable determinant of paediatric CAP, as also seen in our incidence figures. Interestingly, the urban *versus* rural place of residence and passive smoking were not associated with the risk of paediatric CAP [4].

In conclusion, pneumonia and other respiratory infections seem to cluster in the same children in the populations of high-income Western countries. Therefore, further studies should focus on the role of host factors in respiratory infections, including CAP in children. The CAP studies should be powered enough to allow the monitoring of the numerous environmental confounding factors, as well as age- and microbe-specific stratified analyses. The designs of the studies should be prospective and population-based to represent the whole spectrum of the disease, and in optimal cases should

continue for several years with different epidemiological features. The selection and number of controls is of utmost importance. In an optimal study, both healthy controls and controls with non-pneumonic respiratory infections matched for age and sex from the same area should be considered. Genetic epidemiology offers the frame of reference for such studies.

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REFERENCES

1 Teepe J, Grigoryan L, Verheij TJM. Determinants of community-acquired pneumonia in children and young adults in primary care. *Eur Respir J* 2010; 35: 1113–1117.
2 Jokinen C, Heiskanen L, Juvonen H, *et al.* Incidence of community-acquired pneumonia in the population of four municipalities in eastern Finland. *Am J Epidemiol* 1993; 137: 977–988.
3 Heiskanen-Kosma T, Korppi M, Jokinen C, *et al.* Etiology of childhood pneumonia: serologic results of a prospective, population-based study. *Pediatr Infec Dis J* 1998; 17: 986–991.
4 Heiskanen-Kosma T, Korppi M, Jokinen C, *et al.* Risk factors for community-acquired pneumonia: A population-based case–control study. *Scand J Infect Dis* 1997; 29: 281–285.
5 Korppi M, Heiskanen-Kosma T, Kleemola M. Incidence of community-acquired pneumonia in children caused by *Mycoplasma pneumoniae*: Serological results of a prospective, population-based study in primary health care. *Respirol* 2004; 9: 109–114.

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Vital capacity in lying position: important in Duchenne patients

To the Editors:

It was with great interest that we read the paper of LO MAURO *et al*. [1] entitled ''Abdominal volume contribution to tidal volume as an early indicator of respiratory impairment in Duchenne muscular dystrophy'' in a recent issue of the *European Respiratory Journal*. This paper very elegantly explained the contribution of the abdominal volume to tidal volume if patients with Duchenne disease are getting older and change from sitting to supine position. Especially in figure 6 of that manuscript, one can see very clearly the different contribution of ribcage and abdomen (diaphragm) if the patient is getting older. Despite the important contribution of this paper in this field, we would like to make two comments.

First, while the kinematic analysis was performed in both sitting and supine position, the pulmonary function tests were performed in sitting position only. This is a pity, as we know that a drop in vital capacity (VC), when a patient goes from sitting to supine position, is a sign of diaphragm paralysis. In contrast to optoelectronic plethysmography, spirometry in both positions is a simple test that can be performed in every hospital. Therefore, it would be interesting to know if the drop in the VC shows the same pattern in the different patients used in this study. For a physician taking care of these patients, it would be important to know whether he can rely on the VC in different positions to know whether the diaphragm is impaired or not.

Secondly, in this paper the assessment of nocturnal hypoxaemia is presented as clinically relevant and seems to be more pronounced in the patients with a smaller change in abdominal volume. While this might be true, the point of nocturnal hypoventilation is missed in the discussion. It was shown in the paper by WARD *et al.* [2] that nocturnal hypoventilation, with normocapnia during daytime, is an important indicator of respiratory impairment. They showed that even in these patients chronic ventilation is of benefit and should be started at this moment. Therefore the paper could have been even more informative if data on nocturnal carbon dioxide were included.

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REFERENCES

1 Lo Mauro A, D'Angelo MG, Romei M, *et al.* Abdominal volume contribution to tidal volume as an early indicator of respiratory impairment in Duchenne muscular dystrophy. *Eur Respir J* 2010; 35: 1118–1125.
2 Ward S, Chatwin M, Heather S, *et al.* Randomised controlled trial of non-invasive ventilation (NIV) for nocturnal hypoventilation in neuromuscular and chest wall disease patients with daytime normocapnia. *Thorax* 2005; 60: 1019–1024.

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