example, country-wide epidemics and the increasing seroprevalence of *Chlamydia pneumoniae* infection in Finland [4] have been accompanied by corresponding country-wide increased asthma medication use in both males and females and all age groups [5]. The growing body of clinical, epidemiological and therapeutic evidence implicating acute, reactivated and chronic infection by *C. pneumoniae* in asthma [6, 7] forms the basis for proposing the theory that a now waning, geographically heterogeneous worldwide *C. pneumoniae* pandemic is responsible for the recent unexplained observations.

According to this theory, genetic susceptibility involves polymorphisms influencing innate/adaptive immunity and other critical functions (*e.g.* attachment receptors, *etc.*) that affect host response to *C. pneumoniae* infection to promote asthma and lung remodelling. As an example, in a case–control study, NAGY *et al*. [8] reported a strong and significant interaction between *C. pneumoniae* immunoglobulin A antibody, a putative marker for chronic infection, and mannose-binding lectin (MBL) allele polymorphisms producing MBL deficiency in children with asthma. *C. pneumoniae* has been detected in more than half of bronchoalveolar lavage fluids of another group of asthmatic children [9], confirming that the organism is actually present. MBL deficiencies affect up to one quarter of human populations and, perhaps not coincidentally, 20–25% is the apparent ''upper limit'' of wheezing prevalence in epidemiological studies.

Acute primary *C. pneumoniae* infections have been documented to trigger new-onset asthma, which can be successfully rendered asymptomatic after antibiotic treatment [10], raising the possibility of early detection and prevention. Asthma is a strong risk factor for developing chronic obstructive pulmonary disease (COPD) [11] and chronic *C. pneumoniae* infection is also implicated in COPD [12], further suggesting the possibility of prevention of COPD by early recognition and treatment.

The Chlamydia-asthma theory is receiving growing attention among a segment of the public that is acutely aware of current limitations in asthma treatment (www.asthmastory.com). I suggest that now is the time for asthma researchers to promote the widespread critical evaluation that the Chlamydia-asthma theory deserves.

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From the authors:

We would like to thank D.L. Hahn for his interesting comments on our recent paper in the *European Respiratory Journal*. No conclusive explanations have been given thus far on the apparent levelling off of asthma prevalence in children. It has been suggested that the underlying cause of the asthma increase in past decades was due to changes towards a westernised lifestyle. However, it is unlikely that the recent observed plateau, or even decrease, would be due to a stabilisation in a westernised lifestyle. The most likely explanation to us seems that a prevalence plateau of all genetically predisposed children has been reached. This means that children who have a genetic predisposition become asthmatic due to relevant exposure. This relevant exposure could indeed be acute primary infections. Moreover, asthmatic children may be diagnosed earlier because of earlier symptom presentation followed by correct diagnosis and therapy. It has previously been shown that underpresentation of asthma symptoms will normally lead to underdiagnosis of asthma, resulting in underestimated asthma prevalence [1]. Prior to the 1980s, general practitioners in the Netherlands were reluctant to label asthmatic symptoms in children as having a diagnosis of asthma, since the social consequences and the impact of this diagnosis were far-reaching. Together with the steroid phobia in the general public at that time, this probably resulted in an underdiagnosis of asthma. This changed considerably due to the introduction and subsequent widespread use of inhaled corticosteroids in the following years. It is interesting to note that when we compared two identical surveys in Germany and the Netherlands from 1995 and 1997, in Dutch–German borderland, the asthma diagnosis was more prevalent in Dutch children with recent asthmatic complaints (50–60%), whereas >90% of the German children with recent asthmatic

complaints were diagnosed with bronchitis. This resulted in a more frequent use of inhaled steroids and bronchodilators in Dutch children as compared with German children [2].

We cannot exclude the fact that a possible geographically heterogeneous worldwide *Chlamydia pneumoniae* pandemic could contribute to changes in asthma prevalences in different countries. However, it seems unlikely to us that this would be the sole explanation, as not all asthmatics (established or newly diagnosed) have *C. pneumoniae* present in bronchoalveolar lavage fluid. Moreover, the widespread use of (macrolide) antibiotics has not prevented a clear increase in asthma prevalence. On the contrary, it seems that a decrease in hospitalisation and mortality is strongly associated with an increase in the use of inhaled steroids [3], and there is no indication that this is associated with the use of antibiotics.

However, it is certainly worthwhile to pay attention to the so-called Chlamydia-asthma theory proposed by D.L. Hahn and to investigate the presence of *Chlamydia pneumoniae* or other infectious organisms in new asthma patients.

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Nasogastric tube feeding is a cause of aspiration pneumonia in ventilated patients

To the Editors:

In a recent issue of the *European Respiratory Journal*, KOSTADIMA *et al*. [1] reported that early gastrostomy is associated with a lower frequency of ventilator-associated pneumonia (VAP) compared with nasogastric tube (NGT) feeding in patients who are mechanically ventilated due to stroke or head injury. Since VAP is the most frequent and serious intensive care unit (ICU)-acquired infection among patients undergoing mechanical ventilation, and is associated with a 20–30% increase in the risk of death, the preventive strategy for VAP in mechanically ventilated patients is important to reduce the length of an ICU stay and overall mortality [2].

Although the classic theories, including the gastropulmonary hypothesis, are important to understand the mechanisms of VAP, the recent advancement of the pathophysiology of nosocomial pneumonia and aspiration pneumonia are not fully discussed in the paper by KOSTADIMA *et al*. [1].

There is growing evidence that oropharyngeal dysphagia plays a critical role in aspiration pneumonia and VAP in mechanically ventilated patients [3, 4]. Brain injury, severe stroke and unconsciousness, due to sedatives and hypnotics, disturb the swallowing reflex. This results in the development of aspiration pneumonia in humans and animals [5]. However, nosocomial pneumonia and aspiration pneumonia are prevented by the improvement of the swallowing reflex after administration of angiotensin-converting enzyme (ACE) inhibitors [6]. The elevated levels of bradykinin and substance P by ACE inhibitors play a role in setting the threshold for the cough and swallowing reflex in humans, resulting in the reduction of occurrence of pneumonia. Although KOSTADIMA *et al*. [1] speculated about the underlying mechanisms of risk of VAP in the patients with NGT feeding, they did not assess the swallowing reflex and cough reflex. We have developed a novel diagnostic test for the risk of aspiration pneumonia [7, 8]. The simple swallowing provocation test can be applied for all the ventilated patients as it is very easy and can be performed on bedridden patients without requiring their cooperation. The assessment of the swallowing reflex is the clue to the underlying mechanisms of VAP in critically ill patients. As it has been suggested that nosocomial maxillary sinusitis increases the occurrence of VAP, microaspiration of oropharyngeal materials, including maxillary sinus, is a significant cause of VAP [9].

NGT feeding is known to be a significant cause of aspiration pneumonia in stroke patients [10]. Since the NGT bypasses the small amount of gastric contents through to the oropharynx, the materials can be easily aspirated into lower airways in dysphagic patients with stroke. The mechanism is not related to the percutaneous endoscopic gastrostomy (PEG). This evidence supports the fact that NGT feeding, but not PEG, is a significant cause of VAP in critically ill patients. Although feeding *via* PEG is a very straightforward way to reduce aspiration and aspiration-associated pneumonia, the improvement of the swallowing reflex must be a fundamental approach to reduce VAP in patients. As the PEG procedure using gastroscopic fibre may also be a risk for aspiration in unconscious patients, the indication of early gastrostomy for