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Editorial

# Prediction of the temperature response to antibiotic treatment in children with pneumonia

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Nielsen et al. [1] present in this issue an innovative retrospective study that attempts to define the population of children with pneumonia who will respond to antimicrobilal therapy. This was done by correlating the clinical, laboratory and radiological variables available at the time of admission to the hospital with the temperature response to intravenous antibiotic (mainly penicillin) treatment. Since typical and atypical bacterial and viral cultures as well as other methods of diagnosis were not routinely performed, a rapid reduction in fever following initiation of therapy signified in this study the presence of bacterial infection. What allows such an analysis, according to the authors, is that antimicrobial resistance of *Streptococcus pneumoniae* and of *Haemophilus influenzae* is practically nonexistent in Denmark. However, the absence of conclusive identifications of the pathogens reduces the significance of these conclusions. What also detracts from the validity of the analysis is that atypical pathogens (i.e. Mycoplasma, Chlamydia) are not susceptible to penicillin. Furthermore, penicillin is not the most effective antimicrobial even against non-beta lactamase producing *Haemophilus influenzae*. What also confounds the analysis is the possibility that patients with a mixed viral and bacterial infection may not defervesce following antimicrobial therapy.

The investigators identified a number of variables that independently predicted rapid resolution of the fever. These variables were: young age; more than three days of disease before admission; administration of oral antibiotics prior to admission; an elevated initial temperature; elevated white blood cell count; absence of wheezing and of atelectasis; the presence of a sharply delineated and/or spherical pulmonary infiltrate, and general ill appearance only in the absence of chest wall retractions. However, they concluded that these prediction features explained only less than half of the temperature responses and were too imprecise to be clinically useful. It was also not possible to identify a subgroup that would not benefit from antibiotic treatment. Of great interest was the fact that the physical findings, C-reactive protein concentration, white blood cell differential count and the number of pulmonary infiltrates were not helpful at predicting the rate of defervescence.

The low level of predictive performance can be partially explained by the changes over time of the predictive radiological and laboratory changes. Because the children were seen at in different stages of their illnesses, their presentations were not uniform. Another factor that may influence the results is the un-

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known rate of spontaneous defervescence of each bacterial pathogen. The retrospective nature of the study also weakens the ability to evaluate the data.

The findings of Nielsen et al. [1] are in concordance with most other investigators that attempted to correlate the diagnostic potential of C-reactive protein, leukocyte counts and radiological findings with the microbiological etiology of pneumonia. All these previous studies came to the same conclusion that the clinical and laboratory information available at admission can not assist in the differentiation between bacterial and non-bacterial pneumonia [2–5].

The development of better rapid methods of identification of bacterial and viral pathogens using specific and sensitive genetic markers may provide the clinicians with tools that may assist in the identification of those children that require antimicrobial or antiviral therapy. Prospective studies that identify the etiology of the pulmonary infection and correlate them with the clinical and laboratory features are warranted, as they are more likely to lead to definite clinical conclusions.

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