

IN THIS ISSUE...

Fusidic Acid Resistant *Staphylococcus aureus* and Skin Disease

In this issue Alsterholm et al. (p. 52–57) present data from Sweden on fusidic acid resistant *Staphylococcus aureus* (FRSA) isolated from cases of impetigo and atopic dermatitis, respectively. They found high numbers of FRSA in impetigo, ranging from 75% in bullous impetigo to 32% in non-bullous impetigo. By contrast, significantly lower numbers of FRSA (6.1%) were detected in secondarily infected atopic dermatitis. Unfortunately, the FRSA strains were not genotyped. The high numbers, especially in bullous impetigo, could be due to infection by the well-described clone that has caused widespread outbreaks of impetigo in several European countries, including Norway, Sweden and Denmark, during the last 10 years. The low number of FRSA in secondarily infected atopic dermatitis may indicate that the impetigo clone is not a frequent colonizer of atopic dermatitis. The small number of other studies, published from other geographical areas, have shown FRSA levels in atopic dermatitis ranging from 6% to 50%.

All cases were seen at a single dermatological department after referral from general practitioners, and

only a minority of patients seen in the study period 2004 to 2008 were included. There may therefore be an important selection bias and the results are not necessarily true for impetigo and atopic dermatitis patients in primary care. However, it is important continuously to follow the development of fusidic acid resistance among *S. aureus* and to remind the medical community that restricted use of this antibiotic seems necessary to keep resistance rates as low as possible.

For atopic dermatitis it is a clinical impression that secondary infection with *S. aureus* promotes inflammation in flares, and that the addition of antiseptics or antibiotics leads to improved efficiency of treatment in the most severely infected cases. Interestingly, a recent Cochrane Review did not find any evidence that the addition of oral or local antibiotics was of benefit in atopic dermatitis. However, the available data are insufficient for firm conclusions to be drawn.

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