Reply to: "Letter to the Editor: Concerning an Article Comparing the Efficacy of Local and Systemic Treatment of Clinical Mastitis"

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My publication (Soback et al., 1990) was criticized in a recent Letter to the Editor (Pyörälä, 2005).

Pyörälä claimed that the paper should not be referenced in the report of Sérieys et al. (2005) or, for that matter, anywhere. However, Pyörälä fails to describe the alleged errors in the Soback et al. (1990) paper. I find the Soback et al. (1990) paper, for which I am the principal and corresponding author, basically flawless with considerable scientific merit. Pyörälä goes on to claim that the authors themselves have refuted the study, which is inconsistent with the truth. Pyörälä also disregarded our subsequent and only published results on systemic norfloxacin dry-cow treatment (Saran et al., 1995), which did not refute the Soback et al. (1990) paper. By 1995, <3% of Israeli cows had subclinical *Staphylococcus aureus* mastitis, and the research was abandoned as logistically impractical.

I also disagree with Pyörälä's criticism concerning the report of Sérieys et al. (2005), which I found a worthy study. Correlation between susceptibility testing and mastitis therapy outcome has been shown only for very few compounds. Susceptibility testing cannot, therefore, be considered a prerequisite for successful treatment, especially with the antibiotics used in the Sérieys et al. (2005) study. The β -lactamase production was emphasized by Pyörälä, and two reports were quoted to support the criticism. The study by Sol et al. (2000) contradicts Pyörälä's suggestion. The antibiotics used in that study were not β -lactamase susceptible, and no resistance was encountered rendering β -lactamase production irrelevant. Not surprisingly, the β -lactamase production in the study correlated with more chronic infection. Systemically treated cows were excluded from this study.

The paper from Pyörälä's own group (Taponen et al., 2003) suffers from fundamental errors. Staphylococcal β -lactamase-negative pathogens were treated with systemic administration of 20 mg of procaine penicillin/kg, and the β -lactamase producers were treated with 7 mg of amoxicillin/clavulanic acid/kg (clavulanic acid has no antibacterial effect). The procaine penicillin dose was 3 times higher than the amoxicillin dose for no obvious reason as was the co-administered intramammary dose (500 + 300 mg vs. 200 mg). Furthermore, the dose, dosing interval, and the susceptibility breakpoints for clavulanic acid in mastitis treatment have not been determined. To determine the β -lactamase production effect, both groups should have been treated with the same antibiotic (β -lactamase resistant). To establish the difference in efficacy of the two treatments, the cows should have been assigned to the study randomly, disregarding β -lactamase production. Therefore, the assigned groups in the study were under no circumstances comparable. Subsequently, these results cannot be used to support Pyörälä's criticism.

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