Letters

Repeated bovine rotavirus infection and excretion in calves
From Dr A. Schwers and others

SIR,—Rotavirus infection is one of the main causes of diarrhea in newborn calves, but asymptomatic rotavirus excretion is also frequently observed in older calves. Such viral excretion is most probably caused by reinfection rather than by prolonged virus shedding.

In order to test this hypothesis, three colostrum-deprived newborn calves were infected orally soon after birth with different isolates of cell culture bovine rotavirus. All of them shed virus in the feces for at least 10 days after infection and presented a seroconversion against rotavirus.

When between two and three months old, the same animals were inoculated with the same (one calf) or another (two calves) bovine rotavirus isolate than was used for the first infection. At the time of reinfection, none of the animals were still excreting rotavirus in the feces, but all of them possessed circulating rotavirus antibodies.

The three calves excreted rotavirus for a few days after reinfection, as shown by counterimmunoelectrophoresis and virus isolation, although they did not present any clinical signs.

It was already known that passively acquired circulating rotavirus antibodies did not prevent rotavirus infection in calves (Mebus and others 1973, Snodgrass and Wells 1976). Active immunity following virus infection also seems insufficient to prevent virus multiplication after reinfection.

This absence of protection does not reflect antigenic differences between serotypes of rotavirus, as reinfection with the same virus isolate that was used for the first infection was followed by virus shedding. It is most probably related to the site of virus replication, which is limited to the epithelial cells of the gut (McNulty 1978), avoiding contact with circulating antibodies.

The presence of circulating antibodies is not sufficient to prevent reinfection by the same virus isolate, the existence of different serotypes of bovine rotavirus (Naik and Butler 1982, Murakami and others 1983) does not necessarily play a role in the phenomenon of repeated contamination of calves.

Re-excretion of rotavirus by calves of several months of age is likely to play a role in the persistence of rotavirus infection in a farm.

A. SCHWERS
M. MAENHOUDT
P.-P. PASTORET
Faculty of Veterinary Medicine
University of Liège
45 rue des Vétérinaires
B-1070 Brussels

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Virus infections in bovine respiratory disease
From Mr L. H. Thomas and Dr E. J. Stott

SIR,—Dr Verhoeff and Dr van Nieuwstadt are to be congratulated on their comprehensive article on the epidemiology and clinical features of three virus infections in young cattle on Dutch dairy farms (VR, March 24, p588).

Although a preliminary study (Thomas 1973), which these authors quote, did find evidence of respiratory syncytial virus infection in our subsequent work (Stott and others 1982), Thomas and others (1982) demonstrated that respiratory syncytial virus and parainfluenza type 3 virus were significantly associated with respiratory disease and that the weight of evidence was stronger for respiratory syncytial virus.

We are pleased that the results obtained by Verhoeff and van Nieuwstadt in the Netherlands are in line with our findings in the UK and we endorse their conclusion that 'more attention to the prevention of this infection is warranted'.

L. H. THOMAS
E. J. STOTT
Institute for Research on Animal Diseases
Compton
Berks

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Mucosal disease of cattle
From Dr R. M. Barlow and others

SIR,—As your editorial (VR, March 31, p305) suggested, the paper by P. L. Roeder and T. W. Drew is of considerable interest. The interest however lies chiefly in that it illustrates an epidemiological concept which has already been described for the nearly identical condition in sheep which occurs following the establishment of persistent infection with bovine disease virus (Barlow and others 1980, Gardner 1980, Barlow and Patterson 1982, Barlow and others 1983) and widened to include mucosal disease of cattle (Gardner and others 1983).

We wrote to the authors of the foregoing to conclude that sheep and cattle persistently excreting border disease or bovine virus diarrhea viruses are likely to be specifically immunotolerant to a particular strain of virus as a result of transplacental infection with that strain in early pregnancy. In such animals the virus appears to exist in equilibrium with the host in some attenuated form. Pathogenicity may be reasserted following specific homologous reinfection or following some unknown event, the consequence being mucosal disease in cattle and a similar syndrome in sheep.

Roeder and Drew omitted to mention this work and it is therefore incorrect to suggest that the epidemiological study of mucosal disease emerged from their investigations.

We write to put The Record straight.

R. M. BARLOW
A. C. GARDINER
P. F. NETTLETON
Moreton Research Institute
408 Gilzent Road
Edinburgh EH17 7JH

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Mr P. L. Roeder and Mr T. W. Drew, Central Veterinary Laboratory, Weybridge, write—in presenting our paper on mucosal disease (VR, March 31, p309) it was our intention to increase the general awareness of the mucosal disease syndrome by describing our experiences with naturally occurring mucosal disease and relating these to epidemiological studies performed by other workers.

It was not our intention to claim primogeniture for the concept that persistent infection established by early fetal infection was fundamental to the pathogenesis of mucosal disease, nor did we so, clearly attributing that concept to Malnquist (1968) and mentioning the contributions of others. Had we been aware of the full content of the paper by Gardner and others (1983) which was not published until after our paper had been accepted for publication, we would have been pleased to have given it more extensively since clearly it made a significant contribution to knowledge. We would also like to have referred to another publication, by Liess and others (1983), which described the superinfection of persistently infected cattle.

Knowing that the Moreton work was in press we took care to indicate that work and to include a reference to it (Barlow and others 1983) although obviously it was not emphasised to the authors' satisfaction.

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