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 diarrhoea 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 faeces for at least 10 days after infection and presented a seroconversion against rotavirus.

When between two and three months old, the same animals were reinoculated 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 faeces, but all of them possessed circulating antirotavirus antibodies.

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

It was already known that passively acquired circulating antirotavirus antibodies did not prevent rotavirus infection in calves (Mebus and others 1973, Snodgrass and Wells 1978). 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.

As 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.

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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, p288).

Although a preliminary study (Thomas 1973), which these authors quote, did not find evidence of respiratory syncytial virus infection our subsequent work (Stott and others 1980, 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 agreement with our findings in the UK and we would endorse their conclusion that 'more attention to the prevention of this infection is warranted'.

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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 border disease virus (Barlow and others 1980, Gardiner 1980, Barlow and Patterson 1982, Barlow and others 1983) and widened to include mucosal disease of cattle (Gardiner and others 1983).

We wrote: 'In view of the foregoing we conclude that sheep and cattle persistently excreting border disease or bovine virus diarrhoea 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 story of mucosal disease emerged from their investigation.

We write to put *The Record* straight.

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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 Malmquist (1968) and mentioning the contributions of other workers.

Had we been aware of the full content of the paper by Gardiner and others (1983) which was not published until after our paper had been accepted for publication, we would have been pleased to have quoted 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 Moredun 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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