Since the large preponderance of patients with fucosidosis are of Italian extraction, I wonder whether this could be regarded as a legacy of the Roman legions, and would suggest that further searches for fucosidosis could be made most profitably in areas that formed part of the Roman Empire.

Craig Phadrig Hospital,
Inverness. PAULA H. GOSLING.

CAR VACUUM CLEANER FOR BUG CAPTURE

SIR,—Chagas' disease is the commonest cause of heart-failure and sudden cardiac arrest in Brazil. For its successful control the contact between man and the blood-sucking bugs that transmit *Trypansoma cruzi* must be broken. Further studies on domestic bug ecology are needed, and during the course of our work on house demolition of homes containing *Triatoma infestans,* an important bug vector of Chagas' disease, we have had difficulty in catching all the young instars. Underneath a chicken nest, for example, one may encounter many first and second instars which move rapidly to disappear among the rubble. One of us (C. H. C.) suggested suction to overcome this problem. We have found the small vacuum cleaners used in cars to be useful. The degree of suction is sufficient to secure undamaged the first and second instars which are captured by a net gauze (size 2 mm) placed anterior to the fan of the instrument. Third instars and older stages, however, can secure themselves against the suction force and have to be individually collected with forceps. Despite many attempts no-one has succeeded in designing a trapping method for these bugs. We still have to resort to individual manual capture.

C. H. COSTA
P. D. MARSDEN
N. J. ALVARENGA
A. SHELLEY
C. C. CUBA.

House Demolition Team,
Chagas Study Group,
University of Brasilia,
Brazil.

CALF DIARRHŒA CORONAVIRUS

SIR,—We would like to propose an alternative explanation for Sharpel and Mebus's findings.[1] We have adapted the neonatal calf diarrhœa coronavirus [2] (N.C.D.C.) to the suckling mouse brain. Electron micrographs of negatively stained and thin section tissues of infected brain have demonstrated morphological characteristics shared with other members of the coronaviridæ. The adaptation of N.C.D.C. to the suckling mouse brain has facilitated comparative serological testing with human coronavirus strain OC43 by the use of common host reagents and systems. Using specific immune animal sera, coronavirus OC43 and N.C.D.C. were shown by hæmagglutination inhibition, complement fixation, and serum neutralisation tests to be distinct, but revealing the closest antigenic relationship between human and animal coronavirus strains thus far tested in our laboratory.

Of 37 paired (acute and convalescent) sera found positive to OC43 (fourfold or greater antibody rises) in a sero-epidemiological survey of children with upper respiratory illness,[3] 20 pairs (54%) were positive to N.C.D.C. The geometric mean antibody titres in acute and convalescent sera increased from 5 to 60 for OC43 and 14 to 38 for N.C.D.C. In addition, 14 paired sera from adults with known respiratory illness due to coronavirus B814, 229E, and OC43 (virus isolation or administration and/or fourfold or greater antibody rises) were tested for N.C.D.C. antibody rises; 0/2 with B814, 0/7 with 229E, and 4/5 with OC43 infections were found positive to N.C.D.C. Thus we have also confirmed the findings of Sharpel and Mebus that N.C.D.C. does not serologically cross-react with 229E.

Preliminary evidence suggests that N.C.D.C. antibody in humans represents a heterologous response to OC43 infection. However, further studies are needed to exclude the possibility of a heterotypic response to as yet unknown or uncharacterised human strains related to enteric infection, as suggested by Sharpel and Mebus.

Respiratory Virology Branch,
Virology Division,
Center for Disease Control,
Atlanta, Georgia 30333,
U.S.A.

H. S. KAYE
W. B. YARBROUGH
C. J. REED.

RECURRENCE AND HERPES SIMPLEX

SIR,—Cold sores are too often regarded as relatively uninteresting clinically. Nevertheless, the herpes-simplex virus and our understanding of host responses to it is proving a most exciting topic for laboratory research. Professor Lehner and his colleagues (July 12, p. 60) have presented a stimulating hypothesis to explain the latency of herpes-simplex virus and are to be congratulated on drawing the attention of the general medical public to this rapidly advancing field.

One point of interest not discussed by Lehner et al. is what "turns off" the centrifugal flow of activated virus down the neural pathway during the course of an active lesion. I would suggest that this may be where cell-mediated immunity plays a major role. Murine nerve ganglia with activated herpes virus show lymphocytic infiltration both when transplanted [1] and in situ, and this may also be true for the trigeminal ganglion of man. These cells may have been attracted by the chemotactic stimuli from overtly infected cells,[2] and there is in-vitro evidence to suggest that lymphocytes can restore a state of latency in herpes-virus cultures.[3] In vivo, this may happen before the infected neurone dies. Diminution in this function of cell-mediated immunity would explain the prolonged and often asymptomatic excretion of herpes-simplex virus into the mouth of renal-transplant recipients.

As Lehner et al. point out, it seems very likely that cell-mediated immunity is responsible for limiting the cutaneous spread of infection, but I remain to be convinced that it plays a major role in the prevention of recrudescent infections.

The evidence documenting defects in some lymphokine activities in relation to a recrudescent herpetic infection is appealing, but I think it is important to remember that we still have little idea of the importance of these factors in vivo. This is of particular note when that old-fashioned indicator of cell-mediated immunity, the skin test, has consistently been shown to be positive using herpes-virus antigen in patients with recurrent lesions.[4,5] On a more specific level, we do not know, for example, the importance of interferon production in recovery or protection from any form of herpes infection.[6] I am much more impressed with the regular reproducibility of lesions in some people, so that they occur on every skiing trip or with every bout of fever. It seems unlikely that the defect in the production of a particular lymphokine would occur at the right time to just precede every such episode.

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