Detection of antibodies of rinderpest and peste des petits ruminants viruses (Paramyxoviridae, Morbillivirus) during a new epizootic disease in Ethiopian camels (Camelus dromedarius)

F. ROGER, M. GUEBRE YESUS, G. LIBEAU, A. DIALLO, L.M. YIGEZU and T. YILMA

SUMMARY
A serological survey was designed to determine the antibody prevalence of rinderpest virus (RPV) and peste des petits ruminants virus (PPRV) in Ethiopian camels. That study was undertaken after the occurrence in 1995 of an apparently new highly contagious disease characterized by a rinderpest-like disease syndrome in the camel population. 90 dromedaries were distributed in groups based on three epidemiologically-defined regions. The first group was from a non-affected area, the second from sick and contact animals and the third from convalescent animals. The sera were analyzed for antibody to RPV and PPRV by competitive ELISA tests. Results showed a global seroprevalence of 7.8 % for PPRV antibodies and 21.3 % for RPV antibodies. None of the sera from the non-affected area was positive and the second and third groups had various positive rates. In accordance with several authors, the receptivity of the camel to these viruses appears to be a reality. However, its susceptibility to RPV and PPRV had never been confirmed, as well as its role as a potential reservoir of these viruses which cause two major diseases of ruminants. The hypotheses about the occurrence of an emerging infection in camels, caused by pathogens usually found in cattle, sheep and goats, are discussed.

KEY-WORDS: dromedary - serology - peste des petits ruminants - rinderpest - Ethiopia.

Introduction
In the aim to provide elements of investigations after the occurrence of a rinderpest-like disease affecting from 1995 the camel population in Ethiopia, we designed a preliminary serological survey for the detection of antibodies of rinderpest virus (RPV) and peste des petits ruminants virus (PPRV). These diseases normally affect cattle and small ruminants and are caused by closely related viruses [9, 11].

The epizootic, previously described [21, 25], was characterized by a highly contagious respiratory syndrome with a high rate of morbidity (over 90%) and a variable rate of mortality, which depended on the antibiotic treatment. All the camel population of Ethiopia, almost 2 millions of heads, had been affected in less than one year. Nomadic breeders of camels and the veterinary services had never encountered that type of disease.

The last outbreak of rinderpest was declared in 1994 in the North-East of Ethiopia and peste des petits ruminants is prevalent in Ethiopia for many years in sheep and goats [19, 23].
Material and methods

Sera collected from 90 dromedaries in three epidemiologically-defined regions were tested. The first group (G1) comprised 17 serum samples collected in August 1995 from Southern Ethiopia, an area considered non-affected by the disease, since the disease was confined to the northern part of the country. A second set (G2) was collected from 47 clinically sick or contact camels in Eastern Ethiopia in November 1995. A third group (G3) included 26 samples collected from convalescent camels in Northern Ethiopia one month after the outbreak ended in that region.

A competitive ELISA consisting of a monoclonal antibody to the PPR virus (PPRV) nucleoprotein (N) and recombinant PPRV N-protein as an antigen [14] was used for the quantification of PPRV antibodies in all 90 serum samples. A similar test was used for detecting RP virus (RPV) antibody that incorporated recombinant RPV hemagglutinin (H) protein and a specific anti-H monoclonal antibody [2] in 80 serum samples. The threshold value applied was 50% of competition. Moreover, the quantitative values of competition were registered.

Results

PPRV serology (n = 90) - Serum samples from the non-affected area (G1) were all negative for PPRV antibodies, but 7 samples in groups G2 and G3 were tested positive for PPRV antibodies.

The qualitative values (percentage of positive) for the distinct groups were G1 = 0.0 %, G2 = 6.4 % and G3 = 15.4 % (graph 1).

The quantitative mean values calculated for each group on the basis of all samples are significantly different (table I).

RPV serology (n = 80) - Serum samples from the non-affected area (G1) were all negative for RPV antibodies, but 17 samples in group G2 and group G3 were tested positive for RP antibodies.

The qualitative (percentage of positive) values for the distinct groups were : G1 = 0 %, G2 = 36.4 % and G3 = 4.5 % (graph 1).

The quantitative mean values calculated for each group on the basis of all samples were significantly different (table II).

Discussion

Recent serological surveys have indicated the receptivity of the camels to RPV [3, 12, 18], although clinical signs have not been observed [6, 7, 8, 22, 24]. A study in Egypt showed 4.2% of 142 healthy camels at a slaughterhouse were positive for PPRV antibodies by serum neutralization test [12]. However, a clinical disease in camels due to PPRV had not yet been established.

Considering the extremely rapid spread of the disease in Ethiopia, and in spite of the fact that the antibiotics had a positive effect, we can presuppose that the disease observed was initiated by a virus.

We hypothesize that the presence of PPRV and RPV antibodies in camels can be due:

- **either** to an immune humoral response to these viruses that expressed only the passage and an immune receptivity of the camel, without any clinical or sub-clinical expressions. In that case, we have to emphasize that the detection of these antibodies allow revealing the circulation of the RPV and PPRV among the cattle and small ruminants. Thus, in that hypothesis, the disease observed has been caused by other(s) pathogen(s).

- **or** to susceptibility to a morbillivirus closely related to RPV or PPRV, which was virulent for the camels. This virulence could be foreseen taking into account that morbilliviruses have an immunosuppressive effect [11] that led to secondary bacterial infections effectively treated by the antibiotics. Indeed, during this epizootic, *Streptococcus equi subsp. equi* have been isolated from sick camels in Afar region [25] and *Mannheimia haemolytica* from camels in Somali region [4]. Within the morbillivirus genus and related viruses, several emerging diseases have been recently described. The existence of mild strains of rinderpest in domestic cattle has had devastating effects in African buffaloes, eland and lesser kudu in Africa [1, 13, 17]. PPRV has been isolated during an outbreak in Indian buffaloes [10]. New morbilliviruses with devastating effects have been described in marine mammals [15]. Canine distemper virus caused mortalities in lions of East Africa [20]. Viruses belonging to the *Paramyxoviridae family*, initially classified as morbilliviruses, were recently described in diseases declared in horses and human [16] and pigs and human [3]. In that context, we presuppose that this new disease could result from an interspecies transfer of PPRV or RPV strains from cattle or goats and sheep to camel or through the emerging of a new morbillivirus, serologically related to PRV and/or RPV.

The first consequence of these results is that the camels could play a role in the epidemiology of these major diseases in Africa. Considering the necessary programs of eradication of rinderpest and control of *Peste des petits ruminants*, camel populations should be integrated in the epidemiological surveys. They could especially be used for the epidemiological surveillance, during the process of the status evaluation of rinderpest and as sentinels of the disease.

Microbiological, pathological and epidemiological investigations are in progress to precise the etiology of this apparently new disease of camels.

Acknowledgement

The authors are very grateful to Dr Pascal BONNET, CIRAD-EMVT, who provided valuable references on camel diseases and to Dr Fekadu KEBEDE, from the Kombolcha Regional Laboratory, Ethiopia, for his field collaboration.

References


Table I. — Values of competition among the three groups for PPR antibodies (N-protein).

<table>
<thead>
<tr>
<th>Group</th>
<th>G1</th>
<th>G2</th>
<th>G3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size</td>
<td>17</td>
<td>47</td>
<td>26</td>
</tr>
<tr>
<td>Mean for all sample *</td>
<td>0.247</td>
<td>0.323</td>
<td>0.391</td>
</tr>
<tr>
<td>Variance for all samples **</td>
<td>0.013</td>
<td>0.011</td>
<td>0.017</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.000</td>
<td>0.095</td>
<td>0.195</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.435</td>
<td>0.525</td>
<td>0.690</td>
</tr>
<tr>
<td>Mean for positive samples</td>
<td>-</td>
<td>0.513</td>
<td>0.619</td>
</tr>
<tr>
<td>Variance for positive samples</td>
<td>-</td>
<td>0.002</td>
<td>0.007</td>
</tr>
</tbody>
</table>

* ANOVA : F = 8.14 ; p < 0.001
** Variances are homogeneous with 95 % confidence (Bartlett’s test for homogeneity)

Table II. — Values of competition among the three groups for RP antibodies (H-protein).

<table>
<thead>
<tr>
<th>Group</th>
<th>G1</th>
<th>G2</th>
<th>G3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size</td>
<td>14</td>
<td>44</td>
<td>22</td>
</tr>
<tr>
<td>Mean for all sample *</td>
<td>0.229</td>
<td>0.399</td>
<td>0.243</td>
</tr>
<tr>
<td>Variance for all samples **</td>
<td>0.013</td>
<td>0.052</td>
<td>0.038</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.113</td>
<td>0.032</td>
<td>0.000</td>
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<tr>
<td>Maximum</td>
<td>0.477</td>
<td>0.835</td>
<td>0.836</td>
</tr>
<tr>
<td>Mean for positive samples</td>
<td>-</td>
<td>0.658</td>
<td>0.836</td>
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<tr>
<td>Variance for positive samples</td>
<td>-</td>
<td>0.006</td>
<td>0.000</td>
</tr>
</tbody>
</table>

* Non parametric Kruskal-Wallis H = 11.5 ; p < 0.01
** Bartlett’s test shows the variances in the samples differ (non parametric test is preferred rather than ANOVA)


