Long-term study on the spread of caprine arthritis-encephalitis in a goat herd

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Abstract

Dynamics of the infection with caprine arthritis-encephalitis virus (CAEV) has been studied in a goat herd for 17 years, between 1994 and 2010. During the first eight years within-herd seroprevalence raised from 6.8% to 75.0%. Lack of any control measures in the herd and unlimited contact between kids and their dams led to annual increase of seroprevalence rate by 7.7%. Implementation of the control program based on weaning kids immediately after birth and rearing them on cow colostrum and milk allowed to reduce seroprevalence to 26.4% within 5 years, with an annual decline in seroprevalence rate by 9.8%. However control program was unable to eradicate the disease and in the next 4 years the disease turned endemic with prevalence rate ranging from 26.4 to 32.7%. It seems that horizontal transmission and other technical handicaps are limiting factors which, in field conditions, do not allow to reduce the prevalence rate in CAEV-infected herds below 25-30%.

Key words: caprine arthritis-encephalitis, seroconversion, seroprevalence, goat.

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Introduction

Caprine arthritis-encephalitis (CAE) is a viral disease endemic in many countries all over the world. Infection with caprine arthritis-encephalitis virus (CAEV) leads to seroconversion and lifelong presence of antibodies to CAEV, what is the hallmark of lifelong presence of the virus in host’s cells and tissues [1]. The time between infection and seroconversion varies considerably and in general it depends on the route of infection. In experimental conditions antibodies are detected in serum 4 weeks after oral or intravenous inoculation and 4-8 weeks after intramammary inoculation [2]. However, it has been documented that in natural conditions seroconversion can be delayed for many months [3].

It has been confirmed in several studies that trade and movement of goats is responsible for spreading the disease between different regions [4, 5]. Transmission between individual goats in a herd occurs easily from dams to their offspring by ingestion of contaminated colostrum or milk [6, 7]. It is the most important and efficient route of spreading the infection. Even single ingestion of CAEV-contaminated milk suffices to establish the infection, that is, in field condition any access of kids to contaminated colostrum or milk results in seroconversion [7]. In adult goats infection spreads by inhalation of respiratory secretions. This route of infection seems to be less effective and hence long direct contact between animals is necessary [8]. Since the virus is present in milk and intramammary infection has been documented [6, 7, 9] machine milking of goats could be a risk factor [6, 7]. However, clear evidences for a practical role of this route of infection are lacking [5]. Similarly, presence of CAEV in semen of infected bucks has been demonstrated [10] but sexual route of infection has not been confirmed [8]. There are no evidences that indirect contact plays role spreading the disease [5].
Several studies indicated that within-herd prevalence of CAE can vary considerably between herds. In large scale epidemiological studies performed in Poland it ranged from 4.2 to 80.0%, but in most herds was around 30-40% (median 33.3%) [11]. In one commercial dairy goat herd all adult animals were seropositive (authors’ unpublished data). In Norway within-herd prevalence varied from 11.5 to 97.3% in herds implementing eradication program [12, 13]. In South Australia in middle 1980-s 40-45% of annually tested goats were seropositive [14]. In herds diagnosed with CAE for the first time in Jordan overall prevalence was 8.9% [15]. The study performed in dairy goat herds in California showed that seroprevalence ranged from 38.3 to 80.1% [16]. It is obvious that within-herd prevalence increases along with time elapsing from the infection of a herd. One-year-long study carried out in Jordan determined the incidence for 2.4-5.3% [15]. However, there are no data available on the spread of CAE within a herd over longer period of time.

Given that oral route is the most effective in transmission of the infection between goats, weaning kids immediately after birth and rearing them in strict isolation on virus-free colostrum and milk should be proper and sufficient manner of CAE eradication. However, some evidences exist that such procedure is not fully effective in field conditions. It has been observed that even though seroprevalence rises much slower in herds applying this control program, the infection keeps spreading anyway [7, 13, 16]. It seems that direct contact between goats limits the effect of control program, however the dynamics of this process has not yet been determined.

The aim of our study was to evaluate the dynamics of the spread of CAEV infection between goats within a herd and to evaluate the influence of control method based on weaning kids immediately after birth and rearing them in isolation on CAEV-free colostrum and milk on the reduction of prevalence rate.

Material and methods

The study was carried out in the herd established in 1993 by purchasing goats from many small Polish herds. In 1994 breeding goats from France and Holland were imported to that herd. CAE was diagnosed in the herd by a serological method in 1996. The retrospective survey based on serum samples collected in 1994 allowed to confirm that the infection had already been present then [17]. Subsequently, the disease was confirmed in this herd by CAEV isolation [18]. The study has lasted 17 years, from 1994 till 2010. In this period number of adult goats in a herd varied from 32 to 99 (Table 1). From 1994 till 2002 there were no restrictions in rearing kids so they were in constant contact with their dams. In 2002 the control program was implemented. It consisted in weaning all kids immediately after birth and rearing them on cow colostrum and milk (or milk replacers based on cow milk) in strict isolation from adult goats. Seropositive and seronegative adult goats were separated from each other and yearlings used to be combined with the seronegative group. As a rule seronegative goats were machine-milked before seropositive ones. However, this regime showed up to be problematic on that farm and many violations of separating and milking goats in the proper order have been observed during the study.

To avoid possible bias due to persistent maternal immunity in kids only adult goats (12 months of age or more) were included in the study. All goats in the age of 12 months and above have been tested for antibodies to CAEV once a year (in October-November) using ELISA tests (ELISA Checkit CAEV/MVV, Dr. Bommeli AG and Pourquier ELISA Maedi-Visna/CAEV Serum Verification, Institut Pourquier). Prevalence of the infection was calculated as the proportion of seropositive goats to all tested animals in the herd and plotted as the epidemic curve [19]. The average yearly increase and decrease of seroprevalence in the herd was calculated as a geometric mean of modulus of differences in seroprevalence between consecutive years in a given period.

Results

Within-herd seroprevalence is illustrated as the epidemic curve in Figure 1. At the beginning of the study seroprevalence was as low as 6.1% and within 8 years it reached the maximum of 75.0%. The geometric mean of annual increase in seroprevalence in this period was 7.7%. Starting from 2002 (the year when control program was started) seroprevalence stopped growing and from 2003 it began to decline gradually, eventually reaching 26.4% in 2007. The geometric mean of annual decrease in seroprevalence (calculated as modulus) in this period was 9.8%. In years 2007-2010 seroprevalence was relatively stable varying from 26.4 to 32.7% (Table 1).

Discussion

CAE is usually introduced to a herd with newly purchased goats and in many cases importation of goats from abroad is responsible for dissemination of the disease [4, 5]. Our study was conducted in a herd established in 1993 by gathering goats from many small farms from different regions of Poland. That time indigenous goat population in Poland was rather small and isolated [20] and therefore unlikely to have contact with CAEV [4, 21-23]. Moreover, in 1994 breeding goats from France and Holland were imported to the studied herd. In both these countries CAEV had been present since at least 1980-s [24, 25]. All goats in the herd were serologically tested several months after introduction of imported individuals. As all imported goats present at the time of sampling in the herd were CAE-positive and within-herd seroprevalence was very low (Table 1).
it is very likely that the disease was dragged to the herd with imported goats.

The analysis of epidemic curve (Figure 1) allowed to divide the studied period into 4 distinct stages: 1994-2002 when the prevalence was increasing (progression of the epidemic), 2002-2003 when the prevalence was the highest (peak or plateau of the epidemic), 2003-2007 when it was decreasing (regression of the epidemic) and 2007-2010 when the prevalence was stable (endemic).

During initial 8 years of the study (1994-2002) the disease took an epidemic course as no control measures were being applied. The prevalence was growing slowly over the first 4 years and more rapidly over the following 4 years. The mean annual increase of prevalence for the entire stage was 7.7%. The study performed in Jordan in a similar epidemiological situation (first report of the disease in a country) showed that the incidence of new cases per sampling time (study time – one year, 4 samplings with three-month intervals) for all goats in a herd (including kids) ranged from 2.4 to 5.3% [15]. Due to the differences in methodology results of both studies cannot be simply compared. However, low incidence rate observed in herds in Jordan is likely to indicate recent introduction of the disease and so it could be the initial phase of an epidemic. The observation correlates with the first 4 years of our study.

The increase of seroprevalence abruptly ceased when the eradication program was launched in 2002 (peak or plateau of the epidemic). Since 2003 the prevalence had been decreasing evenly by 9.8% per year. It is worthwhile to underline that eradication of the disease was only slightly faster than its spread (annual rate of 9.8 vs. 7.7%). These observations accord with the results of an earlier study showing that weaning kids immediately after birth and rearing them on cow colostrum and milk is a proper method of reducing transmission of CAEV infection in a goat herd [26]. However, serological results from the last stage of our study (years 2007-2010) show that such control program is insufficient to eradicate the disease from a herd – only stable endemic state can be achieved, what is consistent with several earlier reports [2, 13, 16, 27].

In the last stage of our study seroprevalence stabilized at approximately 30% and the disease turned endemic, even though the above described control program has carried on. This can be partially explained by a horizontal transmission of the infection. Results of other studies show that keeping seronegative kids in direct contact with seropositive ones for 20 weeks leads to a seroconversion in 10% of

### Table 1. Seroprevalence of CAE in the goat herd in consecutive years during the study

<table>
<thead>
<tr>
<th>Year</th>
<th>Total number of goats in the herd</th>
<th>Number of seropositive goats</th>
<th>Seroprevalence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1994</td>
<td>49</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>1996</td>
<td>99</td>
<td>17</td>
<td>17.2</td>
</tr>
<tr>
<td>1998</td>
<td>64</td>
<td>17</td>
<td>26.6</td>
</tr>
<tr>
<td>1999</td>
<td>53</td>
<td>22</td>
<td>41.5</td>
</tr>
<tr>
<td>2001</td>
<td>47</td>
<td>32</td>
<td>68.1</td>
</tr>
<tr>
<td>2002</td>
<td>36</td>
<td>27</td>
<td>75.0</td>
</tr>
<tr>
<td>2003</td>
<td>32</td>
<td>24</td>
<td>75.0</td>
</tr>
<tr>
<td>2004</td>
<td>39</td>
<td>28</td>
<td>71.8</td>
</tr>
<tr>
<td>2006</td>
<td>40</td>
<td>14</td>
<td>35.0</td>
</tr>
<tr>
<td>2007</td>
<td>53</td>
<td>14</td>
<td>26.4</td>
</tr>
<tr>
<td>2008</td>
<td>52</td>
<td>14</td>
<td>26.9</td>
</tr>
<tr>
<td>2009</td>
<td>53</td>
<td>15</td>
<td>28.3</td>
</tr>
<tr>
<td>2010</td>
<td>49</td>
<td>16</td>
<td>32.7</td>
</tr>
</tbody>
</table>

![Fig. 1. Epidemic curve depicting the change of CAE seroprevalence in the goat herd during the study (an arrow indicates the beginning of the control program)](image-url)
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Kids [7]. Similar method of CAE control based on weaning kids immediately after birth and rearing them in isolation on virus-free milk was unable to prevent spreading the disease and resulted in seroconversion in 5% of animals within 8 weeks [7]. It should be stressed that our study was conducted in field conditions and, as it was mentioned before, the rules of the control program has not always been strictly obeyed. Anyway it seems that horizontal transmission between adult goats is an important factor contributing to the spread of CAE in goat herds. It can be concluded that, together with technical handicaps mentioned above, the control program does not allow to reduce the prevalence rate in CAEV-infected herds in field conditions below 25-30%.

References