A CAEV epidemiological model for goat breeding

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ABSTRACT

In this paper we analyze the Caprine Arthritis Encephalitis virus disease. We construct a very general model for its epidemiology, for the case when the disease is caused only by a specific viral strain, called the genotype B. The model has only the endemic and the disease-free equilibria, with a transcritical bifurcation connecting the two. Eradication based on this analysis is possible only for very small herds, so that it can hardly be considered economically affordable. The study suggests that in absence of control measures new means of fighting the disease are needed, paving the road for further theoretical and field work.

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1. Introduction

We consider here a mathematical model for Caprine Arthritis Encephalitis virus disease (CAEV) affecting goats, of which the first cases have been reported in 1974, see [5]. As the name suggests, this disease manifests itself mainly like arthritis, the most frequent symptom, but also through other different forms such as pneumonia, which is rare and is associated to the first one, mastitis, leading to udder deformation, or also encephalitis or encephalomyelitis.

CAEV is an infectious pathology characterized by a long period of incubation and with a progressive cronic course. Its clinical signs are not immediately visible, although the goats are infected by the virus. For this reason the virus causing this pathology is named lentivirus. After the incubation period, the infected goats present deformations with enlargement of specific parts of the body. For the farmers, the disease represents an economic burden, since infected goats produce less milk, are weaker and more prone to contract other diseases.

No vaccine is available for this pathology. In order to control it, it is therefore necessary to apply a number of hygienic and sanitary measures to avoid the spread of the virus, see [13]. One of the most used techniques, named "test-and-slaughter", consists in selecting infected goats and directly slaughter them. This method represents a way to eradicate a virus but it can be used only if the infection prevalence is sufficiently low, allowing the survival of the flock. Another one is a virus eradication technique. It is implemented by removing sick goats from the breeding, thereby isolating them together with other infected goats of other breedings. Finally, newborns of infected mothers are not allowed to be weaned by their mothers, but put in stalls with other healthy mothers where they are raised. After weaning they rejoin the other goats in the flock. At this point however, they could of course contract the disease via a horizontal contact with individuals who are asymptomatic carriers.

CAEV is caused by different viral strains belonging to the Small Ruminant Lentivirus group (SRLV), members of the genus lentivirus of the family Retroviridae, able to infect both goats and sheep, see [14]. Based on a limited number of complete

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sequences, they were initially described as two different genetic groups evolving independently in sheep or goats. The ovine strains are closely related to each other and differ from the caprine strains. Over the past two decades, the description and phylogenetic analysis of many complete or partial sequences of caprine and ovine field samples isolated from various geographical regions have clearly highlighted the existence of a genetic continuum, with viruses that do not simply cluster according to the animal species they were isolated from, see [6].

The lentivirus genome is made up of two molecules of monocatenary RNA with positive polarity characterized by an extreme slowness of their replication processes. The long incubation period is the reason of the fact that symptoms of the infection show up only after many years from the contagion. Another example of known retrovirus is the HIV virus responsible of the acquired immunodeficiency syndrome (AIDS) in humans, see [7]. A virus belonging to the same group is responsible of the feline immunodeficiency (FIV), see [8].

To date, SRLVs have been classified into five genotypes. With the exception of genotypes C, D and E, which seem to be geographically restricted to limited areas, genotypes A and B have been described worldwide with well-known associated diseases. Recent veterinary studies have found two different virus genotypes that affect goats and lead to CAEV: the lentivirus genotype B and the lentivirus genotype E.

The genotype B is pathogenic, causing severe CAEV signs in goats. It can be transmitted not only in a vertical way from mother to offspring through the colostrum or the milk, see [11], but also in a horizontal way, through the blood or the saliva of infectious adult goats.

The lentivirus genotype E, whose prototype is known as Roccaverano strain, from the breeding site where it has been discovered for the first time, is particularly important because it is not pathogenic. This means that goats infected by the genotype E do not present any symptom and furthermore they do not pose any threat to the breedings. Field studies show that the genotype E lentivirus can only be transmitted vertically.

In this paper we analyze CAEV caused only by the genotype B, in order to study its complexity, severity and possible transmission ways. CAEV caused by genotype E could produce more complex effects like coinfection or superinfection, see [3], but since this is the first study in this area, see [12], we leave the more complex interactions to further investigations.

The paper is organized as follows. In the next section we identify an extremely important information for the farmer, the so-called breeding's replacement rate, which gives a minimum threshold on the size of the breeding for it to persist in time. Below this value indeed, the farm cannot be kept on an economically sound basis. We then construct a very general model in Section 3, analyze its equilibria and assess their stability. The paper concludes with a final discussion and interpretation of the results.

2. Assessing the breeding's replacement rate

In this section, we introduce the basic demographic model considering an 'ideal breeding' with no pathogens affecting the goats.

Let us take as the time unit the year, which is useful in connection with the gestation time g and the interbirth interval i. The former is on average half year $g = \frac{1}{2}t$, precisely about 153 days. The latter indicates the time elapsing between subsequent deliveries. For the goats it is 1 year, because goats are on heat only in a specific period, after gestation and the nursing times, which overall take about a year. To assess the animal reproductive efficiency the following other fundamental non-negative parameters characterize the goat reproductive cycle, see [19].

The fertility $f < 1$ is the fraction of the pregnant goats, either inseminated or bound for covering. It is used to calculate the reproductive efficiency.

The reproductivity $p > 1$ gives the relationship between the number of newborns with respect to the number of pregnant goats. It represents the ability to give birth to more offsprings in any pregnancy.

The fecundity is the product between fertility and reproductivity: $\varphi = fp > 1$. It represents the relationship between the number of offsprings and the number of goats, either inseminated or bound for covering.

The average reproductive life l_r of a goat expresses the time in which the goat is able to bear, estimated to be around 10 years. For obvious economic reasons, usually the reproductive life of a goat coincides with its life span. Its reciprocal $\mu = l_r^{-1}$ describes the goat mortality, i.e. the rate at which goats die. Thus, specifically, $\mu = 10^{-1} = 0.1 < 1$. This parameter accounts for the fact that, generally, the first coupling for a goat occurs around 7–8 months of age. The newbirth rate $l = 1 - \mu_n < 1$ is the fraction of of live newborns. Here μ_n represents the newborns mortality rate.

The reproduction rate r describes the relationship between the number of inseminated goats and the number of live newborns. It is the product between fecundity and live birth rate, i.e. the product between fertility, reproductivity and live birth rate; thus $r = \varphi l = fpl > 1$. This parameter represents the real economic parameter for the farmer. In fact it is the synthesis of the previous parameters and therefore it is one of the most used in applications.

We are now ready to introduce the key parameter of the model, namely the replacement rate α_{farm}, which represents the rate of offsprings that need to be introduced into the breeding in order to replace the deceased or slaughtered goats in an ideal, disease-free environment. Note that after birth, the offsprings are divided between those to be raised and slaughter-house animals. In order to keep the breeding thriving, the fraction of those that will be raised must exceed α_{farm}.

Let now $G(t)$ denote the total goat population in the breeding. The goat population change in time is due to the fraction α of the newborns that are kept for raising from which the removed animals, due to natural mortality or slaughtering, must be subtracted

$$\frac{d}{dt}G(t) = \alpha r G(t) - \mu G(t). \tag{1}$$

The breeding usually operates at steady state, given that the capacity of the stalls is finite. Thus looking for an equilibrium of the above equation, we find

$$\alpha_{farm} = \frac{\mu}{r} = \frac{1}{l_r r}. \tag{2}$$

Eq. (2) shows that the replacement rate α_{farm} is directly proportional to the goat's mortality rate. Alternatively, the last fraction states that it is inversely proportional to the total number of live newborns produced by a single goat in her entire lifetime.

2.1. Replacement rate for different goat's breeds

The most interesting goat breeds for our study are the breedings for which the presence of the CAEV has been discovered, Fig. 1. For all of them, the mortality is assumed to be the same, $\mu = 0.1$, so that the reproductivity life turns out to be the same, namely $l_r = 10$ years, the most likely value in real situations.

In the literature, see [2], the reproductive parameters characterizing these breeds are found. They are summarized in Table 1.

The reproductive cycle providing deliveries between November and February is strongly conditioned by the annual cycle of pastures while the reproductive parameters are influenced by the altitude.

Using (2), the replacement rate for the sardinian race is

$$\alpha_{sard} = \frac{\mu}{r} = \frac{0.1}{1.128} \cong 0.0886 \cong 9\%, \tag{3}$$

meaning that to keep the goats population constant in a sardinian race goat breeding every year 9% of live newborns must enter the breed.

In a Roccaverano breed instead, twin and triplet births are very frequent, so that p is larger. In this case we find

$$\alpha_{rocc} = \frac{\mu}{r} = \frac{0.1}{1.536} \cong 0.0651 \cong 6.5\%, \tag{4}$$

so that for keeping a constant goat population in a Roccaverano race goat breeding, 6.5% of the live newborns must be raised in the breed.

For the Saanen race, the replacement rate is

$$\alpha_{saan} = \frac{\mu}{r} = \frac{0.1}{1.368} \cong 0.073 \cong 7.3\%, \tag{5}$$

i.e. the goats population remains constant if 7.3% of Saanen live newborns yearly enter into the breed.

Comparing the different goat's race parameters from Table 1 we observe that $f_{sard} > f_{saanen} > f_{rocc}$, but $p_{sard} < p_{saanen} < p_{rocc}$ and also $l_{n-sard} < l_{n-saanen} < l_{n-rocc}$. In agreement with the above results, it therefore follows

$$\alpha_{sard} > \alpha_{saanen} > \alpha_{rocc}. \tag{6}$$

Thus to keep the total number of goats population constant, in a Sardinian race goat breeding more newborns need to be raised than what it is necessary in the Saanen or Roccaverano race goat breedings.

3. The model with an infective pathogen and incomplete vertical transmission

We consider now a breeding with an infective pathogen affecting the goats, the genotype B lentivirus responsible of the CAEV.

Fig. 1. Examples of goats of the Sardinian Race (left), the Roccaverano Race (center) and the Saanen Race (right).

Table 1
Parameters values for different goat breeds.

Parameter	Symbol	Sardinian	Roccaverano	Saanen
Fertility	f	92%	83%	90%
Reproductivity	p	130%	190%	160%
Fecundity	$\varphi = fp$	1.2	1.6	1.44
Offsprings mortality	m_n	6%	4%	5
Live natality	l_n	94%	96%	95%
Reproduction rate	r	1.128	1.536	1.368

We are interested in evaluating the replacement rate in order to maintain the total goat population constant for this particulary case. We also want to study under what parameter combinations the disease could disappear or become endemic. Infected goats produce less milk, are weaker and more prone to contract other diseases, so that, ultimately, their mortality rate should be higher than for healthy goats. For this reason we expect that the breeder in this case has to use a replacement rate α_{path} larger than the one α_{farm} of the ideal breeding. As mentioned earlier, the pathogenic genotype B lentivirus can be transmitted both in a vertical way, i.e. from the mother to the offspring via the colostrum or the milk, and in a horizontal way, from an infectious individual infecting other goats through the blood or the saliva. Let γ denote the rate of newborns getting the infection from the mother, possibly by feeding on infected milk.

As usual in epidemiology, see [4,10], the goat population G is partitioned into susceptibles S, asymptomatic infectious I_a and symptomatic infectious I_s individuals, obviously all nonnegative quantities. Thus

$$G(t) = S(t) + I_a(t) + I_s(t). \tag{7}$$

The asymptomatic individuals will eventually move in long time to the symptomatic class. As soon as the symptomatics are recognized as disease carriers, they are removed by the farmer from the breeding, in order to avoid further possible spreading of the infection.

We now introduce the disease-related parameters. The average number of contacts for unit time between a susceptible and the whole set of the infected individuals, $I_a(t)$ and $I_s(t)$, leading to new asymptomatic individuals is represented by β, while δ denotes the progression rate from the asymptomatic to the symptomatic class.

From the literature, the average number of goats per breeding is seen to be reasonably small; in fact in 2002, there were 2,530,466 goats in 91.463 farms in the whole US, see Table 1.9 in [15], with an average of about 28 goats per farm. In 2007 the goats increased of about 20%, but the number of farms is not available. Assuming that they remained the same, the average becomes about 33 goats per farm. Similar data are available for Piedmont, in Northwest Italy, see [16] and in particular [18]. In the year 2000, there were 3638 farms raising 46,176 goats, with an average of about 13 goats per farm. For Lombardy, [17], the situation was 50,627 goats in 2857 farms, with an average of 18 goats per farm. Further, looking specifically for the Roccaverano stock in [19], see point 19, we find that there are only a dozen or so of farms with around 100 goats, the largest one having 209 animals. The vast majority of the 68 farms has thus a relatively small number of goats. Similar values hold also for other provinces in Italy, where different stocks are raised, see once again [19]. From the above facts, in these situations mass action can thus be safely assumed for modelling the disease transmission process. We thus have

$$\frac{d}{dt}S(t) = \alpha r S(t) + (1 - \gamma)\alpha r(I_a(t) + I_s(t)) - \mu S(t) - \beta S(t)(I_a(t) + I_s(t)),$$

$$\frac{d}{dt}I_a(t) = \beta S(t)(I_a(t) + I_s(t)) + \gamma \alpha r(I_a(t) + I_s(t)) - (\delta + \mu)I_a(t), \tag{8}$$

$$\frac{d}{dt}I_s(t) = \delta I_a(t) - m I_s(t).$$

The first equation contains the dynamics of the susceptibles. Their newborns come either from healthy parents, at rate αr, first term, or from infected parents, at the same rate. But in this case only a fraction $1 - \gamma$ of them does not acquire the disease after being born, see the discussion below to better illustrate this point on vertical transmission. The class is subject to natural mortality μ, third term. The last term models the infection process. Sound individuals can get it at rate β either from asymptomatic or symptomatic individuals.

In the second equation we find the asymptomatic individuals. They enter this class either via horizontal transmission, first term, or by vertical transmission, second term. Here only a fraction γ of newborns from infected parents actually gets the disease, as explained more at length below. The last term accounts for the loss of individuals either by natural mortality, or by progression into the symptomatic class, at rate δ. Note that we explicitly assume that no disease-related mortality exists for individuals in the asymptomatic phase of the disease, in view of the extremely long evolution of this disease.

The symptomatic class evolution is described by the last equation. In it we find recruitments from the asymptomatic class, first term, and losses due to mortality, represented by the parameter m that indicates natural plus disease-related mortality.

Note that to understand the meaning of the parameter γ we need to look at what actually happens in the farm. In this pathology, all the newborns from infected mothers acquire the disease by drinking the infected milk. Therefore, if countermeasures are not taken, there is full vertical disease transmission. The farmer, however, can remove the newborns from their mothers and put them in stalls with only healthy goats. They are raised without acquiring the disease and, when weaned,

they rejoin the other animals in the breeding, as new susceptibles. We introduce γ to better assess the farmer's behavior. The two extremes correspond to the two possible opposite behaviors. When $\gamma = 0$, all the newborns from infected mothers are removed, while $\gamma = 1$ represents the situation of 100% vertical transmission, no offspring is removed from its mother and all newborns from infected mothers will eventually become infected. The model is general enough to accomodate for intermediate situations, for $0 < \gamma < 1$.

The model can be rewritten introducing the fractions of infected individuals instead of the densities $I_a(t)$ and $I_s(t)$, namely

$$i_a(t) = \frac{I_a(t)}{G(t)}, \quad i_s(t) = \frac{I_s(t)}{G(t)},$$

to get

$$
\begin{aligned}
\frac{d}{dt}G(t) &= (\alpha r - \mu)G(t) - (m - \mu)G(t)i_s(t), \\
\frac{d}{dt}i_a(t) &= \beta G(t)(i_a(t) + i_s(t))(1 - i_a(t) - i_s(t)) + (\gamma - 1)\alpha r i_a(t) + \gamma \alpha r i_s(t) - \delta i_a(t) + (m - \mu)i_a(t)i_s(t), \\
\frac{d}{dt}i_s(t) &= \delta i_a(t) - (m + \alpha r - \mu)i_s(t) + (m - \mu)i_s(t)^2.
\end{aligned}
\tag{9}
$$

Then the total breeding population is usually kept at a constant value $G(t) = N$, in view of the finite size of the farm. Therefore, from now on, the constant N represents the fixed size of the breeding. From the steady state of the first equation in the system (9), instead of solving for an equilibrium, we rather determine α, thus finding the replacement rate for the diseased model, α_{path}, as a function of $i_s(t)$. Using (2) it can be written as

$$\alpha_{path} = \frac{\mu}{r} + \frac{m - \mu}{r}i_s(t) = \alpha_{farm} + \frac{m - \mu}{r}i_s(t) > \alpha_{farm}. \tag{10}$$

This is an important result, validating the farmer's intuition. It means that when the breeding is affected by a pathogen, more offsprings must be raised than in the case of a disease-free farm. From (10), the replacement rate is directly proportional to the infected symptomatic fraction in the population. The higher the latter, the more newborns need to be kept in the breeding. On the other hand, this mathematical formulation corresponds to the daily routine for a farmer, thus giving it a sound theoretical background. Using (10) into the system (9), we obtain the final form of the model

$$
\begin{aligned}
\frac{d}{dt}i_a(t) &= -\beta N i_a^2 + (\gamma(m - \mu) - \beta N)i_s^2 + (\gamma(m - \mu) - 2\beta N)i_a i_s + (\beta N + \mu\gamma - \mu - \delta)i_a + (\beta N + \mu\gamma)i_s, \\
\frac{d}{dt}i_s(t) &= \delta i_a - m i_s.
\end{aligned}
\tag{11}
$$

In view of the definitions of the variables as fractions, of the restriction $i_a + i_s \leqslant 1$, coming from (7) and the nonnegativity of S, the dynamics of (11) evolves entirely in the standard unit simplex $\Sigma = \{(i_a, i_s), 0 \leqslant i_a + i_s \leqslant 1\}$ with vertices given by the origin and the two unit points on the coordinate axes.

The equilibria of (11) can be explicitly evaluated. The isoclines are the straight line $\delta i_a - m i_s = 0$, from the second equation, and the conic section

$$-\beta N i_a^2 + (\gamma(m - \mu) - \beta N)i_s^2 + (\gamma(m - \mu) - 2\beta N)i_a i_s + (\beta N + \mu\gamma - (\mu + \delta))i_a + (\beta N + \mu\gamma)i_s = 0. \tag{12}$$

The latter is in fact a hyperbola. Calculating its invariants, we find indeed

$$
\begin{aligned}
I_3 &= \frac{\beta N}{16}(\beta N + \mu\gamma)^2(\mu + \delta)[\gamma(m - \mu)(\beta N + \mu\gamma - \mu - \delta) + \beta N(\mu + \delta)], \\
I_2 &= -\frac{\gamma^2(m - \mu)^2}{4} < 0
\end{aligned}
$$

and $I_1 = -2\beta N + \gamma(m - \mu)$. Note that I_3 in general does not vanish, unless

$$N = \frac{\gamma(m - \mu)((\mu + \delta) - \mu\gamma)}{\beta(\gamma(m - \mu) + (\mu + \delta))},$$

while I_1 vanishes only for

$$N = \frac{\gamma(m - \mu)}{2\beta}.$$

In summary thus, the hyperbola crosses the origin, and if $I_3 \neq 0$, it is not degenerate. Further it is an equilateral hyperbola if $I_1 = 0$.

The equilibria are in any case easily explicitly calculated. They are the origin and the point $C \equiv (i_a^C, i_s^C)$ with

$$i_a^C = \frac{m\beta N(\delta + m) + m\mu\gamma(\delta + m) - m^2(\delta + \mu)}{(m + \delta)(\beta N(m + \delta) - \delta\gamma(m - \mu))}, \quad i_s^C = \frac{\delta}{m}i_a^C.$$

This equilibrium is feasible if and only if $0 \leqslant i_a^C \leqslant 1$, i.e. for either one of the alternative conditions

$$0 < N < \frac{\delta\gamma(m-\mu)}{\beta(m+\delta)} \equiv D_\gamma, \quad N_\gamma \equiv \frac{m(\mu+\delta)}{\beta(m+\delta)} - \frac{\mu\gamma}{\beta} < N. \tag{13}$$

Note that $D_\gamma \leqslant N_\gamma$ since this is equivalent to the condition $(\delta+\mu)\gamma \leqslant \delta+\mu$, which in view of the assumption on $\gamma < 1$, is always satisfied. Further, for $\gamma = 1, D_\gamma = N_\gamma$ so that the endemic equilibrium with total vertical transmission is always feasible.

The Jacobian matrix of the system (11) is:

$$J = \begin{pmatrix} J_{11} & J_{12} \\ \delta & -m \end{pmatrix}, \tag{14}$$

where $J_{11} = -2\beta N i_a + (\gamma(m-\mu) - 2\beta N)i_s + \beta N + \mu\gamma - \mu - \delta$ and $J_{12} = 2(\gamma(m-\mu) - \beta N)i_s + (\gamma(m-\mu) - 2\beta N)i_a + \beta N + \mu\gamma$.

At the origin, from the Routh Hurwitz conditions we find

$$N < \frac{\mu+\delta+m-\mu\gamma}{\beta} = N^{\mathrm{tr}}, \quad N < \frac{m(\delta+\mu)}{\beta(m+\delta)} - \frac{\mu\gamma}{\beta} = N^{\mathrm{det}} \equiv N_\gamma.$$

Further, comparing the values of the relevant quantities, we find that $N^{\mathrm{tr}} > N^{\mathrm{det}}$, so that the disease-free equilibrium is locally asymptotically stable just for $N < N_\gamma$.

For the endemic equilibrium C again we use the Routh–Hurwitz conditions, which amount to $-\mathrm{tr}(J_{|c}) = -J_{11}^C + m > 0$, giving

$$N > \frac{\delta\gamma(m-\mu)}{\beta(m+\delta)} \equiv D_\gamma$$

and $\det(J_{|c}) = -mJ_{11}^C - \delta J_{12}^C > 0$, which leads to

$$N > \frac{m(\mu+\delta)}{\beta(m+\delta)} - \frac{\mu\gamma}{\beta} \equiv N_\gamma.$$

Now, the equilibrium C is stable if $N > N_\gamma$, which is the opposite condition for the stability of the origin. This means that for any given value of the breeding population N only one of the two equilibria can be reached. The actual outcome is determined by this threshold value N_γ. In case $D_\gamma < N < N_\gamma$, C is infeasible. For $N < D_\gamma$, C is feasible, but unstable. Since for $N > N_\gamma$ the equilibrium C is not only stable, but becomes feasible, while for the same condition the origin becomes instead unstable, we are in presence also of a transcritical bifurcation. The results are summarized in Table 2.

Further, we can show global stability of the endemic equilibrium C, following the technique used in [1].

As already previously remarked, first of all note that the trajectories of (11) lie in the unit simplex Σ which is a compact set. Its boundaries cannot be crossed by the system's trajectories, since the coordinate axes are solutions of (11) and therefore cannot be crossed in view of the existence and uniqueness theorem for ordinary differential equations.

Secondly, we show that no cycles can exist in Σ. Consider the function $B(i_a, i_s) = (i_a i_s)^{-1}$. Observing that $i_s - 1 \leqslant 0$, because i_s is a fraction, we have

$$M = \frac{\partial}{\partial i_a}\left[B(i_a, i_s)\frac{di_a}{dt}\right] + \frac{\partial}{\partial i_s}\left[B(i_a, i_s)\frac{di_s}{dt}\right] = -\frac{\beta N}{i_s} - \frac{m\gamma i_s}{i_a^2} + \frac{\beta N i_s}{i_a^2} + \frac{\mu\gamma i_s}{i_a^2} - \frac{\beta N}{i_a^2} - \frac{\mu\gamma}{i_a^2} - \frac{\delta}{i_s^2}$$

$$= -\frac{\beta N}{i_s} - \frac{m\gamma i_s}{i_a^2} - \frac{\delta}{i_s^2} + \frac{\beta N + \mu\gamma}{i_a^2}(i_s - 1) < 0.$$

Thus $D < 0$ and by Dulac's theorem no periodic orbit of (11) can exist in Σ.

When the equilibrium C is locally asymptotically stable, the origin is unstable, and since no periodic orbit can exist in Σ, by the Poincaré–Bendixson theorem, see [9], it follows that C must also be globally asymptotically stable.

In summary we have the following result:

Theorem *For $N > N_\gamma$, the endemic equilibrium C of the system (11) is globally asymptotically stable.*

Table 2
Equilibria of system (11).

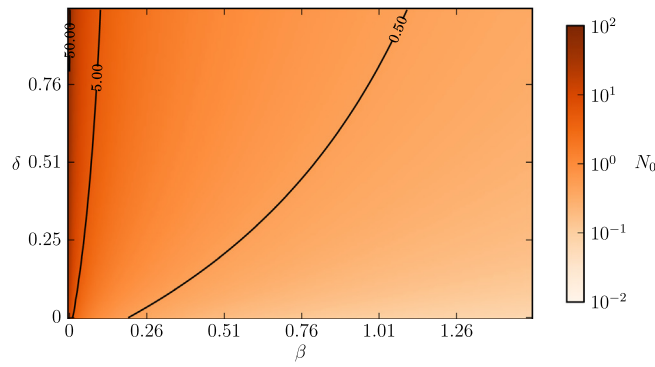| Condition	O	C	Bifurcation
$N < D_\gamma$	Stable	Unstable	
$D_\gamma < N < N_\gamma$	Stable	Infeasibile	
$N = N_\gamma$			Transcritical at $O = C$
$N_\gamma < N$	Unstable	Stable	

Fig. 2. The graphic representation of the critical value $N_\gamma = \frac{\mu+\delta}{\beta(\delta+1)}$ for $\gamma = 0$, at which the two system's equilibria interchange their stability. The level curves for N are plotted against β and δ on the horizontal axes.
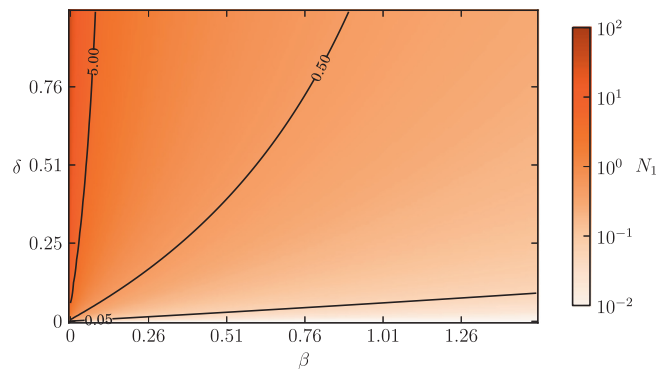

Fig. 3. The graphic representation of the critical value $N_\gamma = \frac{\delta+\mu}{\beta(\delta+1)} - \frac{\mu\gamma}{\beta}$ for $\gamma = 1$, at which the two system's equilibria interchange their stability. The level curves for N are plotted against β and δ on the horizontal axes.
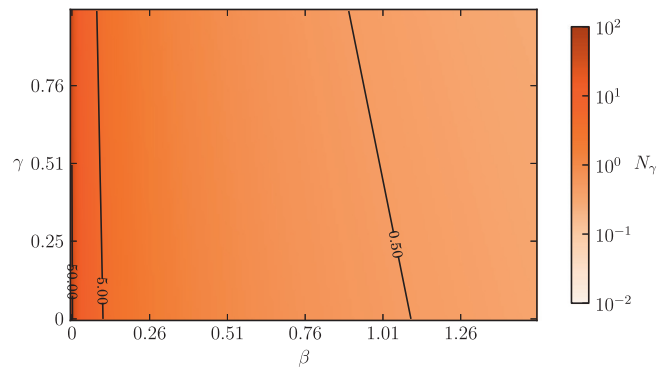

Fig. 4. The graphic representation of the critical value $N_\gamma = \frac{\delta+\mu}{\beta(\delta+1)}$, upon which the feasibility and stability of the equilibria depend. The level curves for N are plotted against β and γ.

4. Discussion

We have found the disease-free and the endemic disease points as possible system's equilibria. Further, there is a trans-critical bifurcation, at which the origin and the point C interchange their stability, when the parameter N attains the critical value N_γ. Thus, in these conditions, since the point C is globally asymptotically stable, the disease remains endemic and all the trajectories tend to this equilibrium, independently of their initial conditions.

To better understand the meaning of the bifurcation condition, we represent this critical value N_γ as a function of two variables, the parameters β and δ, for the particular values of $\gamma = 0$ and $\gamma = 1$ in the Figs. 2 and 3. Observe that m is fixed

at the value 2. The maximum value reached by N is about 80 for $\delta = 1$ in both cases and the same value is also attained in the whole range of $\gamma \in [0, 1]$, see Fig. 4.

The model shows that the system evolves toward a stable state and that the disease is bound to remain endemic, although in principle there could be the possibility of its eradication. But the origin is a stable equilibrium only for a very small N, i.e. for breedings of size so small, that they can hardly be considered as proper breedings, since they would not be economically viable. This result therefore implies that to fight the epidemics, in absence of a proper vaccine, measures aimed at culling the infected goats, and removing the newborns from infected mothers are at present the only possibility to keep the epidemics in check. Work in progress is aimed at finding other alternative possible strategies for disease control.

Acknowledgments

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