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Within-herd spread of contagious bovine pleuropneumonia in Ethiopian highlands

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Abstract

Contagious bovine pleuropneumonia (CBPP) is a major threat for cattle health and production in Africa. This disease is caused by the small-colony type of *Mycoplasma mycoides* subspecies *mycoides* (*MmmSC*). Transmission occurs from direct and repeated contacts between sick and healthy animals. Veterinary services recently reported a resurgence of CBPP in the province of West Wellega, in the Ethiopian highlands. A research program was set up to estimate the epidemiological parameters of the within-herd infection spread. A follow-up survey was implemented in 71 sampled herds of the Boji district (West Wellega province). Fifteen herds were classified as newly infected and used in a serological- and clinical-incidence study. The overall 16-month cumulative sero-incidence risk was 34%. Clinical cases were recorded for 39% of the seropositive cattle; case-fatality risk was 13%. There was no evidence of benefit on infection spread of CBPP-control measures used locally by farmers (isolation or antibiotic treatments of sick animals). This might be related to a lack of power in the statistical analyses or to a quality problem for the medications used (and more generally, for health-care delivery in the Boji district).

Keywords: Contagious bovine pleuropneumonia; Herd monitoring; Within-herd incidence; Clinical signs; Ethiopia

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1. Introduction

Contagious bovine pleuropneumonia (CBPP) is a major threat for cattle health and production in Africa. A list-A disease in the classification of the World Organisation for Animal Health (OIE) (Lefèvre, 2000), it was reported from 17 countries in 2001 (OIE, 2003). The disease is caused by the small-colony type of *Mycoplasma mycoides* subspecies *mycoides* (*MmmSC*) (Cottew and Yeats, 1978; Nicholas and Bashidurin, 1995). In its acute form, general and respiratory signs are observed: polypnea, coughing, painful breathing (Curasson, 1942; Martel et al., 1985; Provost et al., 1987); a marbled pneumonia and an exudative pleurisy are the most-obvious lesions. Recovered cattle often have necrotic lung tissue, encapsulated in sequestra where mycoplasmas can survive.

Transmission occurs from direct and repeated contacts between sick and healthy animals. The involvement of chronic carriers in the perpetuation of the infection has been suggested by several authors (Mahoney, 1954; Martel et al., 1985; Provost et al., 1987; Egwu et al., 1996) but is still debated (Windsor and Masiga, 1977). Risk factors for its spread include high-density confinement in night housings and use of common grasslands and watering places (Provost et al., 1987). In Africa, between-zone or -country contagion essentially is related to cattle movements caused by trade, transhumance and social troubles (Roeder and Rweyemamu, 1995).

In past years, prevention of CBPP indirectly relied upon internationally funded rinderpest-control programs. Pan-African mass-vaccination campaigns were carried out, during which cattle were immunized against both rinderpest and CBPP. Rinderpest was nearly eradicated from Africa and most countries recently stopped vaccination (and increased rinderpest surveillance) to be officially recognized as free of rinderpest (Yaya et al., 1999). Without further support, most African veterinary services were unable to achieve mass vaccination against CBPP, or to implement specific disease surveillance. This situation is thought to be partly responsible for the reappearance of CBPP in countries where it had been eradicated (or, at least, kept under control) (Masiga et al., 1996; Windsor, 2000b).

Because it is unlikely that pan-African CBPP mass-vaccination campaigns will be funded in the near future, research priorities should focus on improving the potency of the vaccines and looking for alternative control strategies (at the farm, zonal and national levels) (OIE, 1994; Masiga and Domenech, 1995). Economic assessment of these strategies is not possible without good evaluations of the epidemiological processes of the infection (e.g. the dynamics of new cases). Unfortunately, longitudinal data on the within-herd spread of CBPP are rare in general (Bygrave et al., 1968) and absent for mixed crop–livestock systems. These systems are common in Africa (especially in the East African highlands) and characterized by small herds managed by individual farmers (Gryseels and Anderson, 1983; de Leeuw et al., 1995).

In the Ethiopian highlands, cattle are the cornerstone of the agricultural system (draft power, milk, meat, manure, etc.) (Laval and Workalemahu, 2002). Veterinary services recently reported CBPP cases in the province of West Wellega (Laval, 2002). A research program was set up to estimate the epidemiological parameters of infection spread, build simulation models of CBPP dynamics and use them to test different strategies for disease control. Our goals in the present paper were to estimate the within-herd

CBPP-spread parameters in newly infected herds, and to assess the effect of different disease management strategies (as actually implemented by the local farmers) on these parameters.

2. Material and methods

2.1. Studied area and agricultural system

We first identified a CBPP-infected area; the western part of Ethiopia was indicated as such by the national veterinary services. A preliminary survey was conducted and the Boji district was selected (Fig. 1). Mixed crop–livestock farming is the dominant agricultural system, providing a subsistence economy at the farm level. Few towns and villages are found in this area; farms are scattered in the countryside—making it difficult to implement mass-vaccination campaigns. Cattle are mostly of the Horro breed, an intermediate Sanga-zebu type (Alberro and Haile-Mariam, 1982). The weaned cattle (>9 months) are kept at night in open temporary paddocks (called *della*) built around the farms (Lesnoff et al., 2002). Suckling calves are kept away from the main herd. They have no contact with the mature animals except during milking. Cattle exchanges (e.g. for loaning contracts) between farmers are frequent (Laval, 2002; Lesnoff et al., 2002).

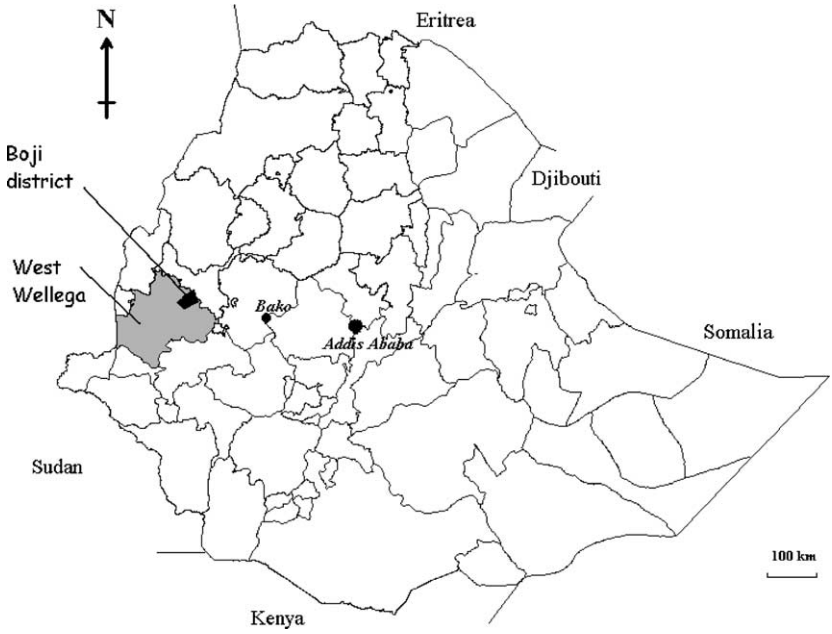

Fig. 1. Location of the Boji district (West Wellega, Ethiopia).

2.2. Herd sampling and monitoring

Information meetings were hold with veterinary-services staff and peasant associations to explain the goal of the survey. All the herds of the farmers who volunteered to be involved in the study were visited. They were selected according to three primary criteria: (i) they should gather at least five owned animals (excluding borrowed cattle) in the della at the start of the follow-up, to ensure survey continuity at the herd level; (ii) newly infected herds were searched for by interviewing farmers (for reports of recent cattle deaths caused by respiratory disease) and slide-agglutination tests on any cattle presenting respiratory signs (Turner and Etheridge, 1963); (iii) farms should be located $\leq$20 km from Bila (the main "town" of the Boji district, where the surveyors were settled) (most of the surveys were done on foot). Secondarily, other herds (all with a della and presumably free of CBPP) were selected around Bila.

The overall herd-sample size (71 herds, 1600 animals) (Lesnoff et al., 2002) was determined by the available financial, human and material means. Selected herds were monitored for 16 months (from July 2000 to January 2001) according to the Panurge method (Faugère et al., 1991). Each animal was ear-tagged. Herds were visited fortnightly, to record demographic events (entry, birth, mortality and offtake), general condition, disease signs and animal health interventions—all by direct observations of the enumerators or by reporting of farmer observations (when events occurred between two visits). In case of death, a post-mortem diagnosis was established by the veterinary supervisors according to the clinical signs and a necropsy (whenever possible). For necropsies, lungs and chest cavity were examined. Blood samples were quarterly collected from all animals to determine their CBPP sero-status. Any animal showing respiratory signs and any animal entering a herd (loans, purchases) also were blood-sampled.

All information were entered and edited in relational database-management systems specifically designed for herd monitoring and serological data (Juanès and Lancelot, 1999; Chavernac et al., 2002).

For each CBPP-affected herd, a retrospective survey was conducted at the end of the follow-up period to reinforce the quality of the follow-up data on the animal health interventions. The combined information showed two important control measures implemented by the farmers to manage CBPP clinical cases: separation of sick animals from the rest of the herd (thereafter referred as "isolation") and treatment with antibiotics. Two CBPP-control strategies were defined according to these practices (Laval, 2002): herds with complete isolation or antibiotic treatment (coded "C"), and herds with partial or null isolation and no antibiotic treatment (coded "P/N"). Herds for which the strategy remained unknown were coded "UNK".

2.3. Sero-incidence study and definition of CBPP clinical cases

Sera were tested with a competitive enzyme-linked immuno-sorbent assay (cELISA) test (Le Goff and Thiaucourt, 1998). Tests were carried out at the National Animal Health Research Centre (Sebata, Ethiopia) and at CIRAD-EMVT (FAO Reference Laboratory for CBPP, Montpellier, France). Serological results were reported as a percentage of inhibition (PI). A herd was considered as CBPP infected if (i) at least one serum tested >50% or

(ii) a necropsy revealed acute CBPP lesions during the monitoring. Within such a positive herd:

(a) Individual tests with PI $>$ 40% were considered as positive. The cELISA test-sensitivity was 86% for this PI-threshold (unpublished report from CIRAD, Montpellier, France). The effect of the specificity (98%) was ignored.
(b) A CBPP-infected animal was defined by the identification of at least one positive test.
(c) Within CBPP-infected animals, a clinical case was defined by the presence of at least two respiratory signs (cough, nasal discharge and dyspnoea), or one respiratory sign plus general signs (poor body condition, painful breathing, appetite loss). The duration of the clinical disease was defined as the number of weeks when signs were observed in the animal after the onset of the disease.

At the herd level, the onset of CBPP (time "zero" for the sero-incidence risk study) was defined as the date of the first clinical case (observed by the farmer or the enumerator), confirmed by at least one CBPP seroconversion or positive necropsy. When the date of the first clinical case in a herd was reported >2 months before the beginning of the follow-up period for this herd, the onset date was considered as unknown (to avoid inaccurate information). After a preliminary data exploration, 15 herds were finally classified as newly infected (*della* sizes averaged over the follow-up period: mean $=$ 17.5 animals, range $=$ 7.6, 26.6) and used in the present study.

2.4. Data analysis

Logistic-binomial regression models were used to analyze the sero-incidence data (uncorrected by the cELISA test-sensitivity) from the 15 newly CBPP-infected herds. For each herd, the follow-up period was discretized into four successive 4-months intervals starting at the CBPP onset date. The response was the sero-incidence of CBPP among the cattle present in the *della* at the beginning of each period, i.e. the number of positive seroconversions during the period, over the number of seronegative cattle at the beginning of the period. Elapsed time after CBPP onset (discretized in 4-month period) was the explanatory variable.

Because animals were clustered in herds, and repeated observations were made on the same herds, within-herd individual responses were likely to be correlated—violating the independence assumption needed in ordinary logistic-regression (e.g. McDermott et al., 1994). Three statistical models were used to overcome this problem. The first was obtained by fitting an ordinary logistic-regression (OLR) and multiplying the resulting variance–covariance matrix of the fixed effects by the variance-inflation factor (VIF). The VIF was defined as the sum of the squared Pearson residuals divided by the residuals' degrees of freedom (McCullagh and Nelder, 1989). The other two models were generalized linear mixed-effect models (GLMM): the fixed effects were the time categories as described above, and herd was the random effect (i.e. herd was related to the intercept of the regression equation). In other words, a population mean was defined by the fixed effects, and herd-specific trajectories were parallel (on the logit scale) to this base line. Parameters of the GLMM were fitted either with the adaptative Gaussian quadrature (AGQ) (Pinheiro and Bates, 1995), or a Monte Carlo Markov chain (MCMC) algorithm (Zeger and Karim, 1991).

Table 1
CBPP cELISA sero-incidence data (uncorrected by the test-sensitivity) for zebu cattle in 15 CBPP newly infected herds from West Wellega (Ethiopia)

Herd	Size[a]	Four-month time period sero-incidence data							
		$0 \le t < 4$ months		$4 \le t < 8$ months		$8 \le t < 12$ months		$12 \le t < 16$ months	
		N[b]	INC_1 (%)[c]	N	INC_2 (%)	N	INC_3 (%)	N	INC_4 (%)
1 (C)[d]	23.7	22	36	16	13	16	0	20	10
2 (C)	19.0	17	18	17	0	18	0	20	5
3 (C)	17.5	19	5	15	7	15	7	15	0
4 (C)	25.4	21	5	24	8	19	0	23	0
5 (C)	14.1	19	58	2	0	3	0	2	0
6 (P/N)	13.1	14	14		12	25		9	44
7 (P/N)	19.1	22	14	18	6	21	5	–[e]	–
8 (P/N)	10.0	10	20	10	0	9	22	6	0
9 (P/N)	15.0	16	50	10	10	9	33	5	0
10 (P/N)	20.3	34	35	–	–	–	–	–	–
11 (P/N)	9.1	9	22	6	0	8	0	6	0
12 (P/N)	7.6	10	20	8	13	6	0	5	0
13 (UNK)	21.0	18	27	25	0	24	0	4	25
14 (UNK)	21.5	22	5	22	5	18	0	22	9
15 (UNK)	26.6	25	0	27	19	22	14	22	5

[a] Average della sizes (animals >9 months old) over the follow-up period.
[b] Number of seronegative animals in the herd at the beginning of the period.
[c] Sero-incidence risks (proportions of the positive seroconversions in the seronegative animals).
[d] CBPP-control strategies implemented by the farmer (C: herds with complete isolation or antibiotic treatment, P/N: herds with partial or null isolation and no antibiotic treatment, UNK: unknown).
[e] No data available.

The latter was used because no Gaussian assumption on random effects was necessary to draw inference.

Graphical analyses and tests were carried out on the estimated random herd-effects, to investigate two potential risk factors for CBPP incidence (Table 1): herd size (handled as a continuous variable) and disease-control strategy.

3. Results

3.1. CBPP serological incidence

Ninety-nine seroconversions were observed during the survey period (Table 1). The statistical analysis of the sero-incidence provided similar results with the different logistic-regression models (Table 2). The time effect was negative and significant ($P < 0.01$ at the most): the sero-incidence risk decreased with time. The estimated herd-effects were not distributed as Gaussian (Fig. 2, left plot), so a confidence interval based on the distribution quantiles was used (rather than tests) to assess the statistical significance of the herd-effect variance. The 95% confidence interval of this variance was (0.12; 1.42) (GLMM–MCMC

Table 2
Coefficients for three logistic-regression models of CBPP sero-incidence for zebu cattle in 15 CBPP newly infected herds from West Wellega (Ethiopia). Sample sizes were 278, 212, 197 and 155 cattle during the first, second, third and fourth 4-month periods after CBPP introduction

	OLR + VIF[a]				GLMM − AGQ[b]				GLMM − MCMC[c]			
	b (−1.27)[d]	S.E. (0.21)	d.f. (52)	P[e] ($<10^{-3}$)	b (−1.40)	S.E. (0.23)	d.f. (14)	P ($<10^{-3}$)	b (−1.42)	S.E. (0.25)	d.f. (14)	P ($<10^{-3}$)
$5 \leq t < 8$ months	−1.17	0.43	52	0.009	−0.99	0.31	14	0.006	−1.01	0.31	14	0.006
$8 \leq t < 12$ months	−1.30	0.46	52	0.007	−1.13	0.33	14	0.004	−1.14	0.33	14	0.004
$12 \leq t < 16$ months	−1.78	0.61	52	0.005	−1.58	0.43	14	0.002	−1.63	0.44	14	0.002
Random-effect variance	–	–	–		0.42	0.23	14	0.094	0.54	0.35	14	0.144

[a] Ordinary logistic-regression + variance-inflation factor (VIF = 2.2, $P < 10^{-3}$).
[b] Generalized linear mixed model fitted with adaptative Gaussian quadrature.
[c] Generalized linear mixed model fitted with Monte Carlo Markov chain.
[d] Values within parenthesis indicate intercept.
[e] Student's t-test with degrees of freedom (d.f.) (null hypothesis: true value for $\beta = 0$).

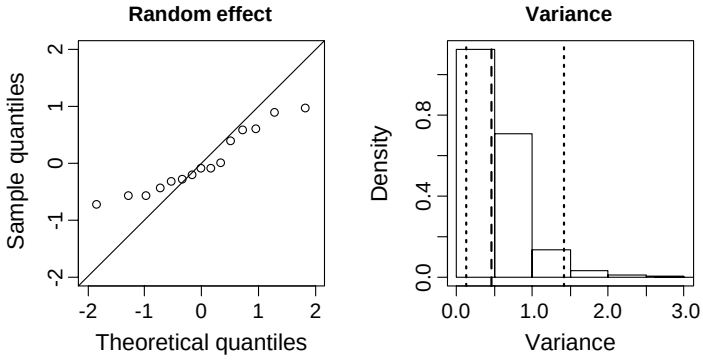

Fig. 2. Normal quantile plot for the estimated random herd-effect (left plot) and probability distribution of their variance (right plot) estimated from the MCMC logistic-regression model of CBPP sero-incidence for zebu cattle in 15 CBPP newly infected herds from West Wellega (Ethiopia). On the left plot, the departure from the bisecting line indicated that the estimated herd-effects were not distributed as Gaussian. On the right plot, the vertical dashed line was drawn at the median of the variance probability distribution (0.46) and the vertical dotted lines delimited the 95% confidence interval of the variance (0.12; 1.42). The non-inclusion of the threshold-value zero in the confidence interval showed evidence of a significant random herd-effect variance.

model, Fig. 2, right plot) and the estimated VIF was 2.2 (H_0: VIF $= 1$, $P < 10^{-3}$)—which both confirmed the necessity to account for clustering within herd.

Risk estimates and their confidence intervals were similar with the three regression models (Table 3). Results of the GLMM–MCMC model were used in the rest of this section. Sero-incidence risks decreased from 20% in period 1 ($t < 4$ months after CBPP onset) to 5% in period 4 (Table 3). However, risks in periods 3 and 4 (i.e. $8 \leq t < 12$ months and $12 \leq t < 16$ after CBPP onset) still were significantly >0. The 16-month cumulative risk was 34% (25; 48) (95% confidence interval in brackets), or 40% (29; 56) after correction for the cELISA test-sensitivity.

Table 3
CBPP sero-incidence and cumulative sero-incidence risks (%) (uncorrected by the test-sensitivity) estimated from three logistic-regression models for zebu cattle in 15 CBPP newly infected herds from West Wellega (Ethiopia)

Period	OLR + VIF[a]		GLMM − AGQ[b]		GLMM − MCMC[c]	
	Incidence[d]	Cumulative incidence[d]	Incidence	Cumulative incidence	Incidence	Cumulative incidence
$0 \leq t < 4$ months	22 (16; 30)	22 (16; 30)	20 (13; 29)	20 (13; 29)	20 (13; 28)	20 (13; 28)
$4 \leq t < 8$ months	8 (4; 15)	28 (21; 37)	8 (5; 14)	27 (19; 37)	8 (4; 14)	26 (18; 37)
$8 \leq t < 12$ months	7 (3; 15)	33 (26; 43)	7 (4; 12)	32 (23; 43)	7 (4; 13)	31 (22; 44)
$12 \leq t < 16$ months	5 (2; 13)	36 (29; 47)	5 (3; 8)	36 (26; 47)	5 (2; 10)	34 (25; 48)

Sample sizes were 278, 212, 197 and 155 cattle during the first, second, third and fourth 4-month periods after CBPP introduction.

[a] Ordinary logistic-regression + variance-inflation factor.

[b] Generalized linear mixed model fitted with adaptative Gaussian quadrature.

[c] Generalized linear mixed model fitted with Monte Carlo Markov chain.

[d] Estimated population mean and 95% confidence interval.

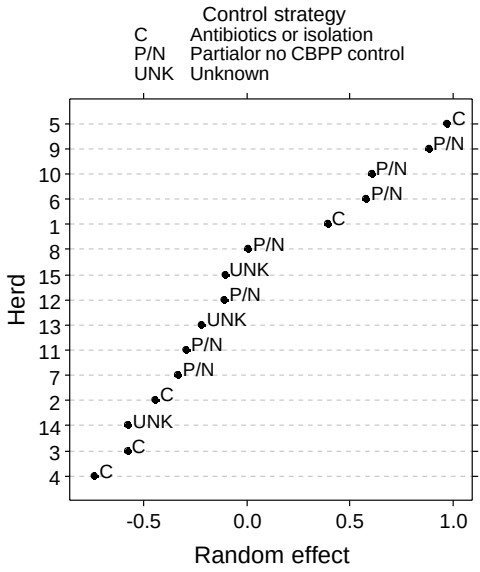

Fig. 3. Relation between CBPP-control herd status and herd-effect estimated from the MCMC logistic-regression model of CBPP sero-incidence for zebu cattle in 15 CBPP newly infected herds from West Wellega (Ethiopia). Estimated herd-effects were ranked from the lowest (lowest herd CBPP sero-incidence) to the highest (highest herd CBPP sero-incidence). Herd identifiers in the vertical axis corresponded to Table 1. No clustering of herd-effects by CBPP-control strategies "C" and "P/N" was apparent—indicating no evidence of strategy effect on the sero-incidence risk.

A negative trend was observed ($r = -0.28$) between herd-effect and herd size, but the linear correlation coefficient was not significant (randomization test, $B = 2000$ permutations, $P = 0.64$). Moreover, no evidence of a CBPP-control strategy effect was shown. No clustering by CBPP-control strategies "C" and "P/N" was apparent in the estimated herd-effects (Fig. 3). The difference of the mean herd-effects between strategies "P/N" and "C" was 0.271 on the logit scale (which represented a variation of 6% in the cumulative sero-incidence risk) and was not significant (randomization test, $B = 2000$ permutations, $P = 0.43$).

3.2. Clinical cases and case-fatality

Clinical cases were recorded for 39% of the 99 seropositive cattle. For animals surviving the disease, the median duration of the clinical signs was 3 weeks (mean = 4, range = 1, 11). The difference in the clinical-phase duration between untreated and treated animals was 1.2 weeks and not significant (Kolmogorov–Smirnof test, $P = 0.73$).

The CBPP case-fatality risk was 13% (3; 23) and no antibiotic treatment effect was observed (death of 3/20 untreated animals and 2/19 treated animals, Fisher's exact test $P = 1$).

4. Discussion and conclusions

One of the major difficulties of the study was to identify newly CBPP-infected herds. From our field investigations, including discussions with the local veterinary-services staff and farmers, we believed that CBPP was resurgent in the Boji district. However, the observed clinical CBPP cases were relatively rare in the area. Further difficulties were the insidious nature of the disease and the lack of sensitivity and specificity of the pen-side diagnostic test (Turner and Etheridge, 1963). To assess the CBPP status of the 71 monitored herds, the cELISA test threshold was set higher at the herd level (PI = 50%) than at the animal level (PI = 40%). These thresholds were retained to avoid false-positive herds in the sub-sample selected for the sero-incidence survey (specificity >99% when PI threshold = 50%) and still have a reasonable sensitivity and specificity at the animal level for the serological follow-up data (specificity = 98% and sensitivity = 86% when PI threshold = 40%).

Concerning the disease impact, the yearly cumulative CBPP incidence (36% after the test-sensitivity correction) and the case-fatality risk (13% of clinical cases) represented a small effect on the herd demography. For example, an estimate of the annual CBPP-specific mortality risk was 36% × 39% × 13% = 1.8% (on average, 0.32 deaths per year for an infected herd of 18 cattle). This effect was not detected in statistical analyses when the CBPP-free and infected herds were compared (Lesnoff et al., 2002). However, other cattle-productivity components such as animal draught, milk yield, manure and loaning contracts should be accounted for in an overall productivity analysis to assess the economic effect of CBPP at the farm level (Laval, 2002; Laval and Workalemahu, 2002).

The cumulative incidence risk was lower than those reported from experimental challenges (Turner, 1954; Hudson and Turner, 1963; Yaya et al., 1999; Wesonga and Thiaucourt, 2000) or from a field outbreak in a pastoral herd (Bygrave et al., 1968) (>70%). It seems biologically unlikely that this was related to the natural resistance of animals and other causes have to be considered. In experiments, disease-free animals are confined continuously with highly infectious animals. This was not the case in Boji, where animals were only confined at night in open paddocks. The variability in *MmmSC* strains' infectivity, or an effect due to the small herd size (infection control may be easier than in large herds) might also be involved. Finally, recovered-immune or vaccinated animals might have been introduced in the area through commercial or loaning flows—thus reducing the true proportion of susceptible cattle (CBPP serological tests do not detect all vaccinated animals—none of them if vaccination had been performed more than 3 months previously—nor all recovered animals as antibody titers do not always persist a long time in infected animals) (Yaya et al., 1999).

Incidence risks observed in periods 3 and 4 ($8 \leq t < 12$ months and $12 \leq t < 16$ months after CBPP onset) were significantly above 0 ($P < 0.01$). This was not in agreement with the reported overall outbreak durations (Hudson and Turner, 1963; Bygrave et al., 1968; Provost et al., 1987). In the absence of re-infection, most of the new CBPP seroconversions should occur within 6–7 months after the initial introduction. The late seroconversions observed during the survey might have resulted from infections of susceptible cattle newly introduced in the herd (by purchase, loan or incoming of young stock). These infections could have been generated by contacts with CBPP chronic carriers (which might present time-delimited infectious phase, although this hypothesis remains unproven) (this point was discussed in Lesnoff et al., 2004) or by unobserved contacts with neighboring and

non-monitored infected herds. As herds were open, the late seroconversions might also have resulted from secondary CBPP introductions occurring through importation of undetected infected cattle (possibly including young moving into the *della*). In case of such secondary introductions, the 'real' overall cumulative sero-incidence risk could take any value (on average) between 26% (periods 1 and 2) and 34% (periods 1–4).

The morbidity risk (proportion of clinical cases within infected animals) (39%) fell within the 30–70% range reported in Africa (Turner, 1954; Hudson and Turner, 1963; Bygrave et al., 1968; Provost et al., 1987; Masiga et al., 1996). Case-fatality risks reported in the literature are highly variable (10–80%) (Provost et al., 1987; Egwu et al., 1996; Masiga et al., 1996). These risks are often difficult to compare, because denominators (e.g. total of animals in the herd, infected animals or clinical cases) are not clearly described in the studies. Nevertheless, the observed case-fatality risk in Boji was in the lowest part of this range. This was in agreement with Provost et al. (1987) concerning the zebu breeds. Moreover, other observations (not detailed here) showed that CBPP was not the first cause of mortality in infected herds (predation by wild animals and anthrax were the most frequent causes identified in the survey).

Globally, comparison with other field results was limited by the lack of detailed reports on CBPP outbreaks (Windsor, 2000a). This highlights the need to define standardized methods to investigate CBPP outbreaks, and to popularize them in veterinary services and diagnostic laboratories involved in CBPP control. International control programs should also devote substantial resources to support training and research in this area. Data from these reports would allow us to obtain more-precise estimates of within-herd incidence risks and other epidemiological parameters. Participatory rural appraisal might provide useful health information from wide areas (Catley and Mohammed, 1996; Catley and Leyland, 2001). However, on a smaller scale, we believe that herd follow-up surveys (such as the one described in the present study) provide accurate information (especially for incidence data). Such longitudinal studies should be undertaken in purposely selected areas to give epidemiologists and veterinary services a more-precise picture of the field epidemiology of CBPP in Africa.

The between-herd variability in CBPP incidence risks observed in the study was difficult to interpret. Isolation of sick animals by reducing the contacts between infected and healthy animals might reduce within-herd spread of the disease. Treatments might reduce both incidence and case-fatality risks. However, no significant effect of the CBPP-control measures, as implemented by the farmers, was observed in this study. Moreover, antibiotic treatments did not reduce the case-fatality risk and the average duration of signs. This might be related to a lack of power in the statistical analyses due to the small number of infected herds in the sample or to confounding factors (for example, the priority given by the farmers to the treatment of the most-severely affected animals). On the other hand, it might reflect a quality problem for the medications used, and more generally, for health-care delivery in the Boji district. Most of the cattle treated with antibiotics during the follow-up survey received a single injection of a 10% oxytetracycline suspension (purchased on the informal market), and administered intra-muscularly at a dose of 10–20 ml per cattle by the farmers themselves. Such a treatment protocol was probably not appropriate to ensure CBPP recovery.

Confronted with an emergency situation, farmers adopted a private and individual management of the CBPP—relying on isolation or the use of antibiotics (9 of the 15 selected

farmers). Antibiotic treatments are not officially recommended because it might result in chronic *MmmSC* carriers (Provost, 1996). However, the Boji district is far from any major city and farms are scattered in the countryside. Roads and power facilities are poor. In these conditions, it is difficult for the public veterinary services to implement mass-vaccination campaigns. For the farmers, antibiotics were thus an applicable control-measure to reduce CBPP mortality, although their effect was not apparent in the study sample. Clinical trials should be undertaken to better investigate the therapeutic effect of antibiotics on CBPP and, if antibiotics are recommended, to practical treatment guidelines ensuring efficiency at the individual level (clinical recovery), clinical recovery, and safety from an epidemiological viewpoint (prevention of long term carrier state).

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