Tropical Animal Health and Production, 31 (1999) 347–361

Impact of Mastitis Control Measures on Milk Production and Mastitis Indicators in Smallholder Dairy Farms in Kiambu District, Kenya

A.O. Omore[1,2*], J.J. McDermott[1], S.M. Arimi[3] and M.N. Kyule[3]
[1]International Livestock Research Institute, PO Box 30709, Nairobi; [2]Kenya Agricultural Research Institute, PO Box 57811, Nairobi; [3]Department of Public Health, Pharmacology and Toxicology, University of Nairobi, PO Box 29053, Nairobi, Kenya
**Correspondence: E-mail: a.omore@cgiar.org*

Omore, A.O., McDermott, J.J., Arimi, S.M. and Kyule, M.N., 1999. Impact of mastitis control measures on milk production and mastitis indicators in smallholder dairy farms in Kiambu district, Kenya. *Tropical Animal Health and Production*, **31(6)**, 347–361

ABSTRACT

Bovine mastitis and mastitis control were investigated on smallholder farms in central Kenya. After an initial observational study, a clinical trial to assess the impact of three different mastitis control strategies – (1) improved udder hygiene, (2) treatment of subclinical cases, and (3) a combination of these – was conducted on 100 randomly selected farms with 332 lactating cows. Before the implementation of control measures, the milk yield was low (mean 6.5 kg/day; median 6 kg/day) and somatic cell counts (SCC) were high, with 80% and 43% of cows having milk with SCC greater than 250×10^3 cells/ml and 600×10^3 cells/ml, respectively. Infectious pathogens were also commonly isolated, with 63% of cows being positive for pathogenic bacteria. Neither intervention strategy alone had any effect on mastitis indicators or milk yield. In combination, the measures had some impact, lowering the prevalence of contagious pathogens by 18%, but this was not reflected in a significantly increased milk yield, lowered SCC or reduced incidence of clinical mastitis.

Keywords: bacteria, cattle, dairy, economics, hygiene, mastitis, somatic cell counts, treatment

Abbreviations: CMT, California mastitis test; GEE, generalized estimating equation; IM, intramuscular; IMM, intramammary; LNML, natural logarithm of the lactation length in months; LNSCC, natural logarithm of SCC; SCC, somatic cell counts

INTRODUCTION

Bovine mastitis, especially in its subclinical form, is widely considered to be the most costly disease facing the dairy industry in temperate countries (Kingwill *et al.*, 1970; Philpot, 1976; Blosser, 1979; Raubertas and Shook, 1982; Schepers and Dijkhuizen, 1991). Estimates of the impact of mastitis and mastitis control measures vary widely (e.g. McDermott *et al.*, 1983; Rivard *et al.*, 1986; Goodger and Ferguson, 1987; Bartlett *et al.*, 1990; Schepers and Dijkhuizen, 1991). The differences in impact may result from differences in the farming system and in the level of production, assessment methods and the scope of the impact assessed.

Commonly used methods for assessing disease impact are observational studies and clinical trials. Clinical trials based on random allocation of animals or farms provide a better level of proof in testing alternative control methods than do observational studies, where the groups are self-selected by the farmer or investigator and thus more susceptible to confounding.

Most published estimates from clinical trials have compared the outcome of a control strategy with the situation before the start of the programme. Success and profitability for any control measure depends on the type of mastitis and the control measure implemented. Profitability from control programmes varies widely from net losses (e.g. McDermott *et al.*, 1983) to high positive gains (e.g. Dobbins, 1977; Goodger and Ferguson, 1987).

Progress in controlling mastitis has been quantified by reduction in the prevalence of subclinical mastitis or in the incidence of clinical mastitis. Improved milking management (including teat dipping) and antibiotic therapy reduce new infection rates and shorten the duration of infections, respectively (Philpot, 1969, 1975). Antibiotic therapy is best administered in the dry period to achieve a higher cure rate (Natzke, 1981; Eberhart, 1986), although therapy during lactation is also often practised despite the cost of discarded milk, the low efficacy of therapy during lactation (Philpot, 1969; Radostitis *et al.*, 1994), and the risk of antibiotic residues occurring. In addition, there is a lack of a reliable procedure for identifying mastitis infections (Philpot, 1969).

An additional determinant of success that may be overlooked in farmer-supervised trials is that of compliance. Adoption rates and compliance with recommended mastitis control measures have been shown to depend on the returns that farmers perceive and their understanding of the rationale behind the measures (Burton *et al.*, 1988; Williamson *et al.*, 1988). Farmer compliance may be poor for mastitis control, since concerted control efforts are usually necessary to reduce the mastitis rates (especially in herds with a high prevalence of chronic infections) before tangible results of increased milk yield and profit can be slowly realized.

On the basis of high infection rates and high somatic cell counts (SCC), mastitis has previously been reported to be an important problem in Kenya (Hamir *et al.*, 1978; Maina, 1984; Munene *et al.*, 1987; Ngatia, 1988). However, in a recent observational study, the associations between these mastitis indicators and milk yield in the low-producing smallholder herds have been found to be modest compared with those in temperate countries (Omore *et al.*, 1996a).

A clinical trial was conducted on smallholder dairy farms in central Kenya to more specifically assess the technical and financial impact of mastitis and to compare some recommended mastitis control programmes.

MATERIALS AND METHODS

Study population

The clinical trial was conducted with farmer members of two dairy societies (Kikuyu and Nderi) in Kiambu District, central Kenya. The societies had a combined member-

ship of 1325 dairy farmers (555 in Kikuyu and 770 in Nderi). The sampling frame was built from active society members who were delivering milk at the time of sampling. The selection of these two societies was justified from an earlier observational study which found that the prevalence of mastitis was similar across farms in each of the dairy societies in Kiambu District (Omore *et al.*, 1996a). The selection of these dairy societies greatly reduced study costs, because of their proximity to the Veterinary Faculty of the University of Nairobi, without seriously affecting the applicability of the results to the rest of the District. The areas covered by Kikuyu and Nderi Societies are in the south of Kiambu District, at a mean elevation of about 1500 m.

Trial groups

Three intervention strategies – improved management practices; therapy for cases of subclinical mastitis; and a combination of both – were compared with a nonintervention group in which no control measures were introduced. Regular field visits were conducted every 2 months to ensure that the intervention measures were being implemented correctly. During these visits, data were collected on milk production and cases of clinical mastitis. In addition, cow-side California mastitis tests (CMT) were performed and samples were collected for bacterial culture and SCC. Data on the cost of drugs, veterinary fees and discarded milk were also collected.

Farms were allocated to four trial groups in a completely randomized design. Each group consisted of 25 farms, making a total of 100 farms (50 farms per dairy cooperative society) with 322 cows. The mastitis control measures implemented for each group are outlined below.

Group A: Improved management practices. The management and hygiene practices employed were: (a) hand and udder washing with disinfectant before milking; (b) post-milking teat dipping; and (c) proper milking technique (Schalm *et al.*, 1971). Iodophor udder wash and teat dip (Coopers Ltd, Nairobi) were used.

Group B: Therapy of subclinical cases. This involved treatment of subclinical cases of mastitis, if either a mastitis pathogen had been isolated at the previous sampling or the CMT was positive following a high SCC ($>300\,000$ cells per ml of quarter milk sample) and CMT at the previous visit was positive. Oxytetracycline 10% (Coopers Ltd, Nairobi, Kenya) was used as the first line of treatment and administered intramuscularly (IM). As with other tetracyclines, less resistance has previously been found to occur againt oxytetracycline than other commonly used drugs in Kenya (Rege *et al.*, 1989; Mulei, 1990). If there was no response, as determined by persistent high cell counts and bacteriology at the next sampling, an antibiotic sensitivity test was done to choose the next drug, which was administered either IM or by the intramammary route (IMM). The implicit assumption was that a cow that tested positive on two consecutive visits had the same persistent infection.

Group C: Improved management practices and therapy of subclinical cases. This was a combination of programmes A and B.

Group D: Nonintervention group. This group had no mastitis control measures applied.

Farms were allocated to the appropriate group at the first visit. Farmers asked to implement improved management practices (groups A and C) were trained and provided with the necessary materials (e.g. a towel for each cow and iodophor teat dip) at the second visit. Treatment of infected cows (groups B and C) commenced at the second visit after collection of samples. Response to interventions was monitored from the third visit onwards.

Clinical cases of mastitis were treated using a standard treatment protocol in all four groups. When a farmer suspected a case, he called project staff (who were also veterinary clinicians), university ambulatory staff or a qualified local veterinary clinician. The clinician made a confirmative diagnosis based on clinical examination, udder palpation and CMT. Pre-treatment milk samples were collected for bacteriology and SCC. As in subclinical cases, tetracyclines were used as the first line of treatment. If the initial treatment failed, which was determined by a lack of response in 3–5 days, an antibiotic sensitivity test was done to choose the next drug. Farmers were asked not to use any other antibiotic during the study and to report all cases of clinical mastitis to a responsible clinician. Evaluation of the response to treatment was determined both within 5 days and at the next 2-monthly visit from the clinical response, CMT, SCC and bacteriology. The data gathered were used to estimate the rate of occurrence and recurrence of clinial mastitis and its duration.

Compliance with udder hygiene measures was assessed by the rate of use or non-use of the teat dip supplied. One farmer in group A and two farmers in group C did not comply with the routine measures for udder washing and teat dipping and they were subsequently reallocated into groups D and B, respectively.

Data collection

The first author and two technicians visited all the farms every 2 months from May 1993 to April 1994, inclusive. Introductory visits were conducted together with dairy society officials.

During the first farm visit, an initial farm survey and individual-cow questionnaires were administered by an animal health assistant conversant with the local language. Questions were asked covering farm management, including feeding, housing, mastitis control measures and general disease history. The initial cow questionnaire covered history, individual animal health, treatments and productivity (including age, breed, milk production levels and reproductive performance). A general physical examination and an examination of the udder and teats were conducted for each cow. A cow-side CMT was carried out and quarter milk samples were aseptically collected in sterile 20 ml glass tubes from each lactating cow. Samples were kept in a cold box and transported within 6 h of collection to the laboratory at the Department of Public Health, University of Nairobi.

On subsequent 2-monthly visits, a follow-up survey was administered, in which the health and production events (e.g. milk yields, calving and treatments) occurring in the

previous 2 months were recorded. Physical examination, sampling and laboratory tests were carried out as above. In cases where project or university ambulatory service staff were not available to attend to a clinical mastitis case, the farmers were reimbursed any costs paid to private veterinarians upon production of receipts.

Samples for bacteriology were streaked onto blood and MacConkey agar plates and incubated at 37°C for 24 h. Isolated organisms were identified to genus and/or species level using a standard protocol (National Mastitis Council, 1987). Staphylococci were subdivided into coagulase-positive and coagulase-negative classes. Coagulase-positive staphylococci were assumed to be *S. aureus*. *Streptococcus agalactiae* were differentiated from other streptococci.

Data storage and analysis

Laboratory results, farm management and individual animal-level and quarter-level data were entered, stored, screened and corrected in PANACEA (Pan Livestock Services Ltd, University of Reading, UK).

Changes in milk production and SCC, and the prevalence and incidence of contagious and environmental mastitis pathogens were compared between the three intervention groups and the nonintervention group. Isolated bacteria were grouped according to their usual means of spread (National Mastitis Council, 1987). *Staphylococcus aureus* and *Streptococcus agalactiae* were grouped together as contagious pathogens. Gram-negative bacteria and streptococci other than *S. agalactiae* were grouped together as environmental pathogens. Other bacterial isolations were grouped as minor pathogens.

Descriptive statistics for milk production, SCC, including the proportion of cows with elevated cell counts ($>250\times10^3$ and $>600\times10^3$ cells/ml, respectively) and the proportion of cows with positive cultures of bacterial pathogens, were estimated for each visit to each of the four groups of farms. The former threshold for cell counts was recommended by Dohoo and Meek (1982), and the latter was arrived at during the observational study (Omore *et al*., 1994).

Two strategies were used to evaluate the technical impact of mastitis control measures. The first was simply to compare, for visits 3–6, the average milk yield, proportions of cows with elevated SCC and contagious or environmental pathogens between the intervention group and the nonintervention group. This analysis was repeated independently for cows with the highest (upper 25th centile) SCC, the highest (upper 25th centile) milk yield and the lowest (lower 25th centile) milk yield. The second strategy was to include mastitis control groups as independent variables in repeated-measures models of the different outcomes of interest (milk yield, natural logarithm of SCC (LNSCC), contagious and environmental pathogens). This was done using the generalized estimating equation (GEE) approaches of Liang and Zeger (1986) and Zeger and Liang (1986).

The GEE models ignored the effects of herd clusters (due to small herd size) and fitted an autoregressive (AR-1) correlation pattern between successive visits. Independent variables for inclusion in GEE models were first screened using the forward

stepwise-selection option in Proc REG ($p < 0.05$ for entry and retention) in SAS statistical software (PC-SAS for Windows version 6.12, SAS Institute, Cary, NC, USA) and by serially removing or forcing back and re-testing individual variables. The screened variables were grazing methods (zero vs semi-zero or free); milk yield; natural logarithm of lactation length in months (LNML); breed (exotic or zebu); contagious mastitis pathogens (present or absent); environmental mastitis pathogens (present or absent); season (rainy or dry); and covariance adjustment for pre-implementation values (visits 1 and 2). Age of cow, natural logarithm of SCC (LNSCC) and intervention group were forced into all GEE models.

The assessment of the costs of the mastitis control measures included: (1) estimates of overall milk yield losses associated with SCC mastitis; (2) clinical mastitis rates; (3) the costs of the control programmes (costs of drugs, chemicals and materials); and (4) the costs associated with treatments for clinical mastitis. Clinical mastitis treatment costs were calculated from data collected on the cost of drugs, veterinary fees and discarded milk. The financial impact was evaluated using a partial analysis.

RESULTS

The average number of cows in each herd was 3.3 (median 2, range 1–9). Ninety-eight farms were followed over the complete 1-year trial period. Of the 325 cows on these farms, 198 were followed during the whole period and 127 for only part of the period, owing to purchase or calving after the start of the study, sale or death. Overall, data were collected on 1605 cow-visits in 3210 cow-months (260 cow-years). A total of 5150 milk samples were collected, processed for cow-side CMT tests and SCC, and cultured for bacteriology. However, owing to missing values and the requirement of at least two observations for inclusion in the GEE model, only 1292 and 552 quarter-level and cow-level observations, respectively, were retained for that analysis.

Descriptive statistics

Table I and Figure 1 display the average values for outcome measures by trial group and visit. The mean milk yield on all farms before the implementation of the intervention programmes (visits 1 and 2) was 6.5 kg/day (median 6 kg/day). The average SCC for both visits was high, with 80% and 43% of cows having milk with SCC greater than 250×10^3 cells/ml and 600×10^3 cells/ml, respectively. Rainfall was much lower than average during the 12-month clinical trial period, with a mean of only 710 mm being recorded in Kikuyu (Kabete Agro-meteorology Station) and Nderi (Muguga Forest Station). This was in the lower range of mean annual rainfall of 600–2500 mm. The severe drought resulted in shortage of forage and had a significant impact on the milk yields between the second and fifth visits (Figure 1).

TABLE I
Average values for milk yield, milk SCC and bacterial isolations during the clinical trial on smallholder dairy farms in Kiambu District[a]

Variable	Trial group[b]	Visit number and date (months and year) 1 5–6/93	2 7–8/93	3 9–10/93	4 11–12/93	5 1–2/94	6 3–4/94
Mean milk yield (kg/day)[c]	A	7.25 (0.35)	5.78 (0.46)	4.77 (0.30)	5.74 (0.43)	6.92 (0.66)	7.85 (0.59)
	B	7.60 (0.75)	6.36 (0.65)	6.33 (0.58)	5.88 (0.45)	6.12 (0.39)	7.38 (0.51)
	C	7.04 (0.50)	6.62 (0.61)	4.80 (0.43)	6.38 (0.43)	6.07 (0.45)	6.47 (0.40)
	Control	6.51 (0.36)	5.06 (0.41)	5.39 (0.67)	5.73 (0.54)	7.16 (0.69)	7.36 (0.56)
Median SCC $\times 10^3$ of cow samples (average of SCC of quarter samples)	A	745	485	457	573	475	482
	B	842	508	655	568	600	536
	C	728	518	648	585	452	432
	Control	750	529	499	503	432	406
Clinical mastitis incidence (percentage per 2 months)	A	–	2.4	3.8	5.1	0	2.9
	B	–	1.6	1.6	0	1.8	1.8
	C	–	1.3	2.7	2.8	4.1	1.4
	Control	–	1.4	0	0	0	3.6
Percentage with a quarter having SCC $>600 \times 10^3$ cells/ml and CMT >0 (in parentheses)[d]	A	68 (69)	45 (55)	55 (64)	65 (70)	57 (70)	61 (66)
	B	70 (70)	48 (55)	56 (56)	53 (55)	75 (74)	57 (61)
	C	63 (63)	36 (50)	58 (58)	59 (64)	52 (57)	52 (52)
	Control	68 (71)	57 (60)	62 (75)	55 (66)	49 (66)	55 (60)
Percentage quarters with contagious pathogens	A	73	80	58	70	60	65
	B	64	70	71	72	73	61
	C	59	65	64	49	38	48
	Control	78	70	75	66	60	63
Percentage quarters with environmental pathogens	A	58	55	79	59	78	49
	B	42	60	58	59	70	59
	C	34	40	62	59	64	42
	Control	67	50	76	50	51	48

[a]Number of observations was in the range 43–73 for each trial group and visit

[b]Trial group A = improved hygiene practices; B = therapy of subclinical cases; C = combination of A and B

[c]Standard errors for means of yield are given in parentheses

[d]CMT was evaluated as follows: 0, negative; 1, weak; 2, distinct positive; 3, strong positive

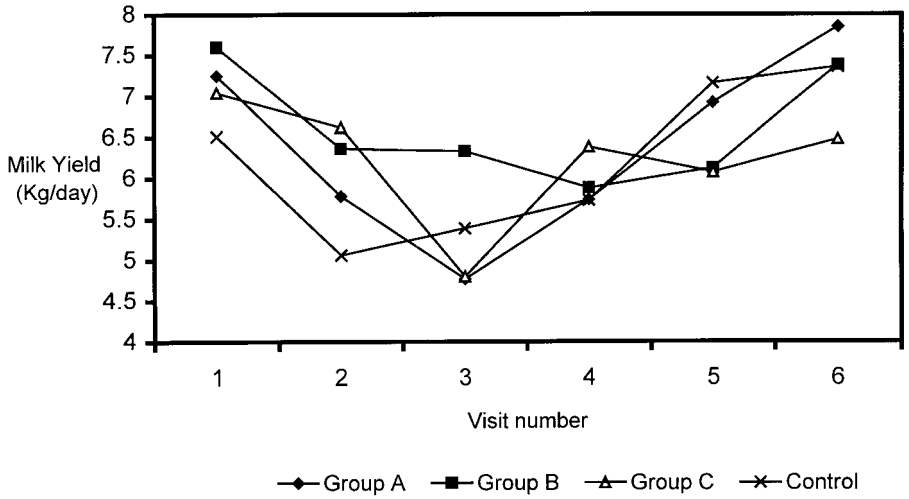

Figure 1. Patterns of mean milk yields by trials group and visit number from May 1993 to April 1994 for cows on smallholder farms in Kiambu District, Kenya. Group A, improved hygiene practices; B, therapy of subclinical mastitis cases; C, combination of A and B

Comparisons of outcomes between the trial groups and the control group

Table II shows simple comparisons of the changes in outcome measures due to mastitis control. There were no significant ($p > 0.05$) changes in the values of any of the variables between the intervention and the nonintervention groups. Analyses of the subset of cows in the highest 25th centiles of SCC (above 1256×10^3 cells/ml) and milk yield (above 8.3 kg/day) and of those in the lowest 25th centile of milk yield (below 3.5 kg/day) did not yield significant results ($p > 0.05$). The overall incidence of clinical mastitis was 10.8 per 100 cow-years at risk and was not significantly different between the groups ($p > 0.05$).

Multivariable regression models

None of the farm-level or cow-level variables screened for association with milk yield (grazing method, breed, bacterial pathogens) were significant in the final GEE model. Only rainy season and, as expected, increasing lactation length (LNML) were significantly ($p < 0.05$) associated with higher and lower milk yield, respectively (Table III). A unit increase in LNSCC lowered milk yield modestly ($p = 0.07$). None of the mastitis control interventions significantly ($p > 0.05$) increased the milk yield.

The mastitis indicators of LNSCC and infectious mastitis pathogens showed different associations with various factors. LNSCC was associated with only two variables: age and contagious pathogens ($p < 0.05$). Both contagious and environ-

TABLE II
Changes in outcome measures relative to nonintervention after implementation of mastitis control measures (visits 3–6) for cows on smallholder dairy farms in Kiambu District[a]

Outcome measure	Improved milking hygiene (group A)	Therapy of subclinical cases (group B)	Improved milking hygiene and therapy of subclinical cases (group C)
Milk yield (kg/day)	–0.05 (0.57)[b]	0.09 (0.55)	–0.38 (0.53)
Natural logarithm of somatic cell counts (LNSCC)	0.14 (0.12)	0.21 (0.12)	0.21 (0.12)
Prevalence of contagious pathogens (% year)	–3.0 (9.0)	3.0 (9.1)	–17.0 (9.7)
Prevalence of environmental pathogens (% year)	11.0 (9.6)	6.0 (10.2)	1.0 (10.0)
Incidence of clinical mastitis (% year)[c]	10.6 (6.6)	2.2 (5.6)	11.1 (6.7)

[a]Number of observations in each trial group was between 150 and 237
[b]Standard error in parentheses
[c]The average number at risk was between 59 and 75

mental pathogens were positively associated with LNSCC ($p < 0.05$) and negatively associated with rainy season ($p < 0.05$) and with the combined interventions of milking hygiene and therapy of subclinical cases (group C) ($p < 0.05$) (Table III). The biggest impact was the reduction in the prevalence of contagious pathogens by 18% (1.60/9.06) under the combined intervention of milking hygiene and therapy of subclinical cases (Table III).

Financial losses

The GEE multivariate model for milk yield (Table III) showed that approximately 5% (0.32/6) of median milk yield is lost for every unit increase in LNSCC. Taking the lowest 10th centile (equivalent to a geometric SCC of individual cows of 280 000 cells/ml or a logarithmic scale score of about 5.6) as a realistic target, the loss in daily milk

TABLE III
Generalized estimating equation (GEE) models of the impact of intervention on milk yield and pathogenic bacteria for 552 milk recordings and 1292 quarter-level observations of cows on smallholder dairy farms in Kiambu District

Parameter	Estimate	SE–Naive	SE–Robust	*z*–Robust
(a) GEE model for daily milk yield (kg)				
Intercept	12.83	1.26	1.49	8.63
Age (years)	0.07	0.07	0.08	0.84
Natural logarithm of lactation length (LNML)	–2.46	0.19	0.21	–11.53
Natural logarithm of somatic cell counts (LNSCC)	–0.32	0.19	0.19	–1.78
Rainy season	1.14	0.24	0.26	4.44
Improved milking hygiene (group A)	–0.32	0.56	0.59	–0.54
Therapy of subclinical cases (group B)	–0.19	0.57	0.67	–0.24
Combination of groups A and B (group C)	–0.69	0.52	0.54	–1.27
(b) GEE model for LNSCC (unit)				
Intercept	5.43	0.10	0.10	52.66
Age (years)	0.06	0.02	0.02	3.76
Natural logarithm of month of lactation (LNML)	0.01	0.03	0.03	0.18
Contagious pathogens	0.94	0.09	0.14	6.74
Improved milking hygiene (group A)	0.07	0.09	0.09	0.79
Therapy of subclinical cases (group B)	0.11	0.09	0.11	1.06
Combination of groups A and B (group C)	0.05	0.08	0.09	0.58
(c) GEE model for contagious pathogens (unit)				
Intercept	–9.06	0.90	0.89	–10.23
Covariate for pre-implementation values (visits 1 and 2)	1.26	0.54	0.44	2.85
Age (years)	0.05	0.56	0.06	0.91
Natural logarithm of somatic cell counts (LNSCC)	1.10	0.14	0.14	7.70
Rainy season	–0.88	0.33	0.32	–2.72
Improved milking hygiene (group A)	0.03	0.44	0.45	0.06
Therapy of subclinical cases (group B)	0.05	0.43	0.45	0.12
Combination of groups A and B (group C)	–1.60	0.54	0.61	–2.61
(d) GEE model for environmental pathogens (unit)				
Intercept	–8.10	0.76	0.85	–9.55
Covariate for pre-implementation values (visits 1 and 2)	1.26	0.38	0.40	3.16
Age (years)	0.03	0.46	0.05	0.60
Natural logarithm of somatic cell counts (LNSCC)	0.98	0.12	0.13	7.36
Rainy season	–0.97	0.28	0.29	–3.38
Improved milking hygiene (group A)	0.13	0.38	0.40	0.34
Therapy of subclinical cases (group B)	0.21	0.37	0.41	0.50
Combination of groups A and B (group C)	–0.98	0.41	0.47	–2.07

yield from the median cell count of 553 000 cells/ml was calculated to be 0.20 kg per cow. This represented a decrease of 4.4% (80 kg) in yield or KShs 1049 loss per cow per year (Table IV). Milk yield losses were recorded with SCC below 280 000 cells/ml, but these losses were considered unrecoverable on a herd basis in the smallholder dairy production system studied.

As there were no significant increases in milk yield brought about by the interventions, only the costs of the different programmes were considered in the partial analysis (Table IV). The average total cost of drugs, fees and discarded milk incurred for each case of clinical mastitis (adjusted to end-1996 figures) was estimated to be Ksh 1010. At the annual incidence of clinical mastitis of 10.8%, this translates into Ksh 109 per cow per year.

TABLE IV
Average cost in KShs of intervention programmes and subclinical and clinical mastitis per cow per year in Kiambu District, Kenya

Cost item	Group A[a]	Group B[a]	Group C[a]
Control of subclinical mastitis			
Iodophor teat dip	300.00	–	300.00
Oxytetracycline treatment IM	–	93.00	93.00
Other IM or IMM treatment	–	55.80	55.80
Towels	24.00	–	24.00
Loss associated with high somatic cell counts	1 049.00	1 049.00	1 049.00
Clinical mastitis[b]	109.00	109.00	109.00
Total cost	1 482.00	1 306.80	1 630.80
Gross margin[c]	22 709.00	22 884.20	22 560.20
Percentage loss from intervention strategy	1.4	0.6	2.0
Percentage loss associated with high somatic cell counts	4.4	4.4	4.4
Percentage loss from clinical mastitis	0.5	0.5	0.5

[a]Group A, improved hygiene practices; B, therapy of subclinical mastitis cases; and C, combination of A and B

[b]Losses calculated based on project-provided cost of drugs, professional fees, discarded milk and the annual incidence rate of 10.8%

[c]Gross margin without control of Ksh 23 142.00 per cow (Omore, 1997) less the total cost of the intervention programme and mastitis in Kiambu District

DISCUSSION

The estimates of mastitis indicators, milk yield and their associations were similar to those recorded during an observational study conducted from July 1991 to June 1992 (Omore *et al.*, 1996a). Contagious pathogens, predominantly *S. aureus*, were significantly associated with LNSCC in both studies.

It is difficult to make direct comparisons of the results obtained from this intervention study with those from other mastitis impact studies for the reasons cited earlier (sampling differences, method of assessment and scope of impact assessed). Factors such as decreased milk quality, changed milk composition, decreased feed intake and replacement costs from premature culling were not considered in our study because of difficulties in obtaining reliable measurements of their importance in the local smallholder production system. For example, although decreased milk quality (due to high SCC) and changed nutrient composition are appropriate factors for an economic analysis in dairy production systems in temperature countries (Schepers and Dijkhuizen, 1991), local payment schemes do not take them into consideration in Kenya.

In addition, the smallholder dairy production farms in this study were quite different from, and the cows in them produced much lower milk yields than, the dairy production systems in temperate countries, where nearly all the other studies on the technical and financial impact of controlling mastitis have been conducted. However, the percentage milk loss associated with increasing LNSCC was similar to those from temperate countries (Raubertas and Shook, 1982; Bartlett *et al.*, 1990). It is probable that a greater reduction in the prevalence of bacterial pathogens would be reflected in lower LNSCC. However, Schalm and colleagues (1971) and Radostitis and colleagues (1994) have both noted that the SCC often does not fall back to its pre-infection level when infection is eliminated, and the lack of impact of a decreased prevalence of bacterial pathogens on LNSCC on these farms should be seen in this context.

The poor response to the intervention methods employed in this trial could be due to a number of factors. First, the antibiotic therapy may not have been particularly effective, since the main contagious pathogen present was *S. aureus*. The response to treatment during lactation in cases of *S. aureus* is usually poor. Radostitis and colleagues (1994) explained that, in chronic *S. aureus* mastitis, the bacteria survive intracellularly in leukocytes walled off in small abscesses of mammary ducts. Dry cow therapy, rather than treatment of chronic cases during lactation, may be a more appropriate control strategy for mastitis in these circumstances (Brown *et al.*, 1998). This would necessitate a longer study to assess impacts in the subsequent lactation. One important factor that would need to be considered is that many smallholder farmers in Kenya do not give cows a specific dry period. Secondly, the 2-monthly visits may have been too far apart to monitor the interventions successfully and would have allowed re-infection of cured cows from a chronically infected herd mate. Thirdly, the improved hygiene interventions were mainly targeted at the control of contagious pathogens; however, suboptimal hygiene in the housing would have been a constant source of re-infection that may have overwhelmed the intervention methods employed.

It appears that other factors limit the potential impact of mastitis control on increasing milk yield. Poor nutrition (Omore *et al.*, 1996b), made even more severe by the prolonged drought during the trial, was probably the most important factor limiting milk yields and may have swamped any improvement brought about by mastitis control. Other factors, such as the long average calving interval of 20 months (Odima *et al.*, 1994; Staal *et al.*, 1998) may also have played a role. Long calving intervals and low milk yields seem to be interlinked constraints and need to be resolved together (Tanner *et al.*, 1998).

The main impact of the interventions was the reduction in the prevalence of infectious pathogens by the combination of improved milking hygiene and therapy of subclinical mastitis cases. This combination might have greater potential benefits when the other factors limiting daily milk yield, such as nutrition and long calving intervals, are improved. At present milk production levels, the costs of these control measures cannot be justified.

Given the weak association between bacterial infection and milk yield (Omore *et al.*, 1996a), it was not surprising that the intervention measures did not have any significant impact on milk yield. Although dry-cow therapy could have been more successful in reducing the infection rate (Natzke, 1981; Eberhart, 1986), it is unlikely that a significant increase in milk yield would have resulted given the serious shortage of feed during the study period. In view of the low levels of milk production on smallholder dairy farms in other parts of East Africa (Omiti and Staal, 1996; Omore *et al.*, 1998), it must be concluded that mastitis control programmes based on mastitis indicators developed in temperate dairy systems are not economically justified locally. However, the economics of mastitis control deserves to be re-examined on a subset of smallholder farms that, owing to improved nutrition and breeding inputs, have achieved higher average milk production levels.

ACKNOWLEDGEMENTS

The authors thank the Kenya Government and the International Foundation for Science for financial assistance. The technical and professional assistance of Drs Lawrence Kariuki and Ngatia Matemo and technician Kaiga Agagwa during data collection in the field was highly appreciated, as were the tireless laboratory services provided by Desterio Ouma, Nathan Magomere, Peter Karanja, John Mathenge, Amos Kimotho and Tirus Munyua. The study relied on the generous cooperation of the study farmers and dairy cooperative staff.

REFERENCES

Bartlett, P.C., Miller, G.Y., Anderson, C.R. and Kirk, J.H., 1990. Milk production and somatic cell counts in Michigan dairy herds. *Journal of Dairy Science*, **73**, 2794–2800

Blosser, T.H., 1979. Economic losses from and the national research program on mastitis in the United States. *Journal of Dairy Science*, **62**, 119–127

Brown, D.F., Ardaya, D., Ribera, H., Cueller, A.M. and Kerby, P.J., 1998. Mastitis control programme in the developing dairy industry of tropical lowland Bolivia. *Tropical Animal Health and Production*, **30**, 3–11

Burton, M.J., Williamson, N.B., Brown, W.B. and Baumann, L.E., 1988. Mastitis control measures used in some Minnesota dairy farms. *Preventive Veterinary Medicine*, **5**, 225–232

Dobbins, C.H., 1977. Mastitis losses. *Journal of the American Veterinary Medical Association*, **170**, 1129–1132

Dohoo, I. and Meek, A.H., 1982. Somatic cell counts in bovine milk. *Canadian Veterinary Journal*, **23**, 119–125

Eberhart, R.J., 1986. Management of dry cows to reduce mastitis. *Journal of Dairy Science*, **69**, 1721–1732

Goodger, W.J. and Ferguson, G., 1987. Benefits and costs of a control program for an epizootic of *Staphylococcus aureus* mastitis. *Journal of the American Veterinary Medical Association*, **190**, 1284–1287
Hamir, A.N., Gehring, W. and Muhammed, S.I., 1978. The incidence of bovine mastitis in Kenya. *Bulletin of Animal Health and Production in Africa*, **26**, 55–61
Kingwill, R.G., Neave, F.K., Dodd, F.H. and Wilson, C.D., 1970. The effect of mastitis control system on levels of subclinical and clinical mastitis in 2 years. *Veterinary Record*, **87**, 91–125
Liang, K.Y. and Zeger, L.S., 1986. Longitudinal data analysis using generalised linear models. *Biometrika*, **77**, 13–22
Maina, P.W., 1984. *A study of bovine mastitis control in Nyeri, Kenya*, (MSc thesis, University of Nairobi, Kenya)
McDermott, M.P., Erb, H.N., Natzke, R.P., Barnes, F.D. and Bray, D., 1983. Cost and benefit of lactation therapy with SCC as indication for treatment. *Journal of Dairy Science*, **66**, 1198–1203
Mulei, C.M., 1990. Micro-organisms associated with clinical mastitis in dairy cows in Kabete area of Kiambu District in Kenya. *Bulletin of Animal Health and Production in Africa*, **38**, 331–334
Munene, J.N., Kariuki, D.I., Munyua, S.J.M., Gachuiri, C. and Wahome, G., 1987. Incidence and cost of mastitis in Kenya: projections from Kabete area. *Proceedings of Animal Production Society of Kenya (APSK)*, **17**, 114–117
National Mastitis Council, 1987. *Laboratory and Field Handbook on Mastitis*, (WD Hoard, Fort Atkinson, USA)
Natzke, R.P., 1981. Elements of mastitis control. *Journal of Dairy Science*, **64**, 1431
Ngatia, T.A., 1988. *Aetiology and pathology of subclinical mastitis in dairy cows*, (PhD thesis, University of Nairobi)
Odima, P.A., McDermott, J.J. and Mutiga, E.R., 1994. Reproductive performance of dairy cows on smallholder dairy farms in Kiambu District, Kenya: design, methodology and development constraints. *Proceedings of the 7th ISVEE Symposium, Nairobi, Kenya. The Kenya Veterinarian*, **18**, 366
Omiti, J. and Staal, S., eds, 1996. *The Ugandan dairy sub-sector: a rapid appraisal*, (Research Report by the National Agricultural Research Organization (Uganda), the Ministry of Agriculture, Animal Industry and Fisheries (Uganda) and the International Livestock Research Institute, (Kenya), Nairobi, Kenya)
Omore, A.O., 1997. *Epidemiology and economics of mastitis in the smallholder dairy sector of Kiambu District, Kenya*, (PhD thesis, University of Nairobi)
Omore, A.O., McDermott, J.J. and Arimi, S.M., 1994. How do you diagnose subclinical mastitis in low producing dairy cows? *Proceedings of the 7th ISVEE Symposium, Nairobi, Kenya. The Kenya Veterinarian*, **18**, 116–118
Omore, A.O., McDermott, J.J., Arimi, S.M., Kyule, M.N. and Ouma, D., 1996a. A longitudinal study of milk somatic cell counts and bacterial culture from cows on small holder dairy farms in Kiambu District, Kenya. *Preventive Veterinary Medicine*, **29**, 77–89
Omore, A.O., McDermott, J.J. and Gitau, G.K., 1996b. Factors influencing production on smallholder dairy farms in central Kenya. *Proceedings of the 5th KARI Scientific Conference, Nairobi, Kenya.*
Omore, A.O., Staal, S. and Thorpe, W., eds, 1998. *The Tanzanian dairy sub-sector: a rapid appraisal. Main report* (Volume 3), (Research Report of the Ministry of Agriculture and Co-operatives (Tanzania), Sokoine University of Agriculture (Tanzania) and the International Livestock Research Institute (Kenya), Nairobi Kenya.
Philpot, W.N., 1969. Role of therapy in mastitis control. *Journal of Dairy Science*, **52**, 708
Philpot, W.N., 1975. Prevention of infection hygiene. *International Dairy Federation Bulletin Document*, **85**, 155
Philpot, W.N., 1976. Mastitis controlled yield in profits. *Proceedings of Ruakura Farmers Conference, Hamilton, New Zealand*, 80
Radostitis, O.M., Leslie, K.E. and Fetrow, J., 1994. Mastitis control in dairy herds. *Herd Health: Food Animal Production Medicine*, 2nd edn, (WB Saunders, Philadelphia), 229–276
Raubertas, R.P. and Shook, G.E., 1982. Relationship between lactation measures of somatic cell concentration and milk yields. *Journal of Dairy Science*, **65**, 419–425
Rege, J.E.O., Mutwiri, G.K. and Mulei, C.M., 1989. Antibiotic resistance patterns of bacterial organisms implicated in bovine mastitis and some canine infections. *Bulletin of Animal Health and Production in Africa*, **37**, 319–326
Rivard, G., Benoit, M. and Marcoux, R., 1986. Les pertes économiques dues à la mammite: une méthode d'appréciation. *Médicine Vétérinaire Québec*, **16**, 93–95
Schalm, O.W., Carrol, E.J. and Jain, N.C., 1971. *Bovine Mastitis*, (Lea and Febiger, Philadelphia)
Schepers, J.A. and Dijkhuizen, A.A., 1991. The economics of mastitis and mastitis control in dairy cattle: a critical analysis of estimates published since 1970. *Preventive Veterinary Medicine*, **10**, 213–224

Staal, S.J., Chege, L., Kenyanjui, M., Kimari, A., Lukuyu, B., Njubi, D., Owango, M., Tanner, J., Thorpe, W. and Wambugu, M., 1998. *Characterisation of Dairy Systems Supplying the Nairobi Milk Market*, (International Livestock Research Institute, Nairobi, Kenya)

Tanner, J.C., McCarthy, N.A. and Omore, A.O., 1998. Why shorten calving intervals on smallholder dairy farms in East Africa? *Proceedings of the KARI/APSK/ILRI/BSAS Conference on Food Lands and Livelihoods: Setting Research Agendas for Animal Science. Nairobi, Kenya*, 190–191

Williamson, N.B., Burton, M.J., Brown, W.B., Baumann, L.E. and Farnsworth, R.J., 1988. Changes in mastitis management practices associated with diet education, and the effects of adoption of recommended mastitis control procedures on herd milk production. *Preventive Veterinary Medicine*, **5**, 212–223

Zeger, L.S. and Liang, K.Y., 1986. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics*, **42**, 121–130

(Accepted: 24 February 1999)

Impact des mesures de contrôle contre la mastitite sur la production laitière et sur les paramètres indicateurs de mastitites chez les petits éleveurs laitiers du district de Kiambu au Kenya

Résumé – La mastitite bovine et les contrôles de celle-ci furent étudiés chez les petits éleveurs dans la partie centrale du Kenya. A la suite d'une étude initiale, d'un projet expérimental pour déterminer l'impact de 3 approches différentes pour le contrôle de la mastitite (à savoir: (1) meilleur hygiène des mamelles, (2) traitement des cas sub-cliniques et (3) une combinaison des 2 autres) une étude fut menée dans 100 fermes représentant 332 vaches laitières.

Avant le développement des mesures de contrôle, la production laitière était faible (en moyenne 6,5 kg/jour avec une medianne à 6 kg/jour) et un comptage des cellules somatiques (SCC) élevé avec un lait ayant des SCC au dessus de 250×10^3 cellules/ml et au dessus de 600×10^3 cellules/ml respectivement pour 80% et 43% des vaches. Des pathogènes infectieux furent souvent isolés avec 63% des vaches étant infectées par des bactéries pathogènes. Une approche basée uniquement sur une intervention n'eut aucun effet sur les cas de mastitites ou sur la production de lait. Une combinaison des 2 approches eut des effets bénéfiques comme la réduction de 18% des agents infectieux (sans pour autant que la production de lait s'en trouve améliorée ou en réduisant le nombre de SCC ou l'incidence des cas cliniques de mastitites).

Impacto de las medidas de control de mastitis en la producción de leche e indicadores de mastitis en los pequeños productores de leche del distrito de Kiambu, Kenia.

Resumen – La mastitis bovina y el control de mastitis han sido investigados en granjas de pequeños productores de Kenia central. Tras un estudio inicial de observación, se realizó una prueba clínica para averiguar el impacto de tres estrategias diferentes para el control de la mastitis: (1) Mejora de la higiene de las ubres, (2) Tratamiento de casos subclínicos, y (3) Una combinación de ambos, en 100 granjas seleccionadas aleatoriamente con 332 vacas lactantes.

Antes de la implementación de las medidas control, la producción de leche era baja (media de 6,5 Kg/día; mediana de 6 kg/día) y el recuento de células somáticas (SCC) era alto, con un 80% y un 43% de las vacas productoras de leche con un SCC mayor de 250×10^3 células/ml y 600×10^3 células/ml, respectivamente. También se han aislado patógenos infecciosos en un 63% de las vacas positivas en bacterias patógenas. La estrategia de intervención por sí sola no ha tenido ningún efecto en los indicadores de mastitis o en la producción de leche. Al combinar las medidas aparece cierto impacto, disminuyendo la prevalencia de patógenos contagiosos en un 18%, pero no se refleja en un aumento significativo de la producción de leche, en una disminución del SCC o en una reducción de la incidencia de mastitis clínica.