

# National intervention study of mastitis control in dairy herds in England and Wales

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**An intervention study was carried out on 52 dairy farms in England and Wales to determine whether the implementation of a well-specified mastitis control plan in herds with an incidence of clinical mastitis of more than 35 cases per 100 cows per year would reduce the incidence of clinical mastitis, and also reduce the incidence of increases in the somatic cell counts of individual cows. A clearly defined plan for the diagnosis and control of mastitis was developed by two veterinary specialists from the research literature. The herds were randomly allocated to receive the plan either at the start of the study (intervention herds) or after one year (control herds). Data on mastitis management and the farm environment were collected during farm visits. After one year there was a significant 22 per cent reduction in the proportion of cows affected with clinical mastitis on the intervention farms compared with the control farms. There were also significant reductions of approximately 20 per cent in the incidence of clinical mastitis and in the occurrence of increases in the somatic cell counts of individual cows from below, to above 200,000 cells/ml.**

DESPITE much research to identify risk factors for clinical mastitis (Pearson and others 1972, Schukken and others 1990, Barkema and others 1998, 1999, Peeler and others 2000), the incidence of clinical mastitis in UK dairy herds (and worldwide) has not decreased significantly in the last 20 years (Wilesmith and others 1986, Booth 1988, Berry 1998, Kossaibati and others 1998, Bradley and Green 2001, Smith 2005). It has been estimated that the mean incidence of clinical mastitis is approximately 40 cases per 100 cows per year (Berry 1998, Kossaibati and others 1998, Bradley and Green 2001). This is likely to be a conservative figure because under- rather than over-reporting of clinical cases is probable. A recent study by Bradley and others (2007) indicated that the mean incidence is currently more than 50 cases per 100 cows per year.

An important limitation for improvements in the control of mastitis has been a lack of knowledge of the effectiveness of farm control strategies derived from studies of risk factors. There is a need to test whether the risk factors associated with clinical mastitis are causal, that is predictive rather than explanatory, and whether a control plan that uses these factors can be implemented and is effective in reducing the incidence of mastitis in commercial dairy herds.

Apart from methods of treatment, there has been little research that has used large-scale, controlled trials to measure the efficacy of strategies to control mastitis or other animal diseases. The effectiveness of a control programme is best tested by means of a controlled trial (Dohoo and others 2003), an approach that provides the highest standard of evidence to guide clinical decision making (Lavori and Kelsey 2002).

A problem with diseases such as mastitis is that the multi-factorial risks necessitate a control programme that involves a number of simultaneous management changes, because a change in only one risk factor may improve mastitis control only marginally (or not at all) if several other factors are contributing to an increased risk of the disease. A further problem is that, owing to the complex aetiology of mastitis, an intervention that may reduce the risk of infection with one pathogen may increase the risk of infection with another.

This paper presents the results of implementing a novel, detailed mastitis control plan on commercial dairy units in England and Wales. The specific hypothesis tested was that the implementation of the plan, developed from known associated risks for mastitis reported in the literature, would reduce the incidence of cows affected with clinical mastitis (ICA) and the total incidence of clinical mastitis (ICM) over a period of one year.

## MATERIALS AND METHODS

### Farm selection

Twenty-six herds were required in each of two groups to have an 80 per cent power and 95 per cent confidence of detecting a true difference in the ICA and ICM of more than 20 per cent between the intervention and control herds. This difference was considered biologically important, and 52 herds were therefore selected.

A database administered by National Milk Records (NMR), Chippenham, was used to identify 250 herds with a record of more than 35 cases of clinical mastitis per 100 cows during the previous 12 months. A recruitment letter was sent to these farmers inviting them to participate in the study and 68 of them responded positively. Monthly recordings of somatic cell counts (SCCs) from these herds were assessed, and 26 herds with an annual arithmetic mean SCC of more than 200,000 cells/ml and 26 with an annual arithmetic mean SCC of less than 200,000 cells/ml were selected. A final selection was made from farms located in one of three regions of England and Wales; a region to the north of a line from the Severn estuary to the Wash, and two regions to the south of this line divided to the east and west by a line joining Oxford and Portsmouth (Fig 1); one farm in south-west Wales was classified as being in the south-west region. Pairs of farms within these regions were selected with the closest arithmetic mean annual bulk milk SCC; one of each pair was selected at random as an intervention farm, the other herd becoming the matched control farm. Two groups of herds were thus selected as intervention and control herds, matched for SCC and region of the country.

### Data collection and visit times

Farm visits were made by two trained researchers to collect data and by two specialist veterinary surgeons to set up the management interventions. The first visit was made to each farm by a researcher between April and May 2004, before the first veterinary visit. A visit to initiate the intervention was made by one of the two veterinary surgeons in May or June, and a follow-up telephone call to the farmers was made in August by the same veterinary surgeon to encourage their compliance. A second visit was made to all the farms by the same researcher in October or November to record the farms' management procedures and a second visit was made to the herds by the same veterinary surgeon in October or November to discuss winter aspects

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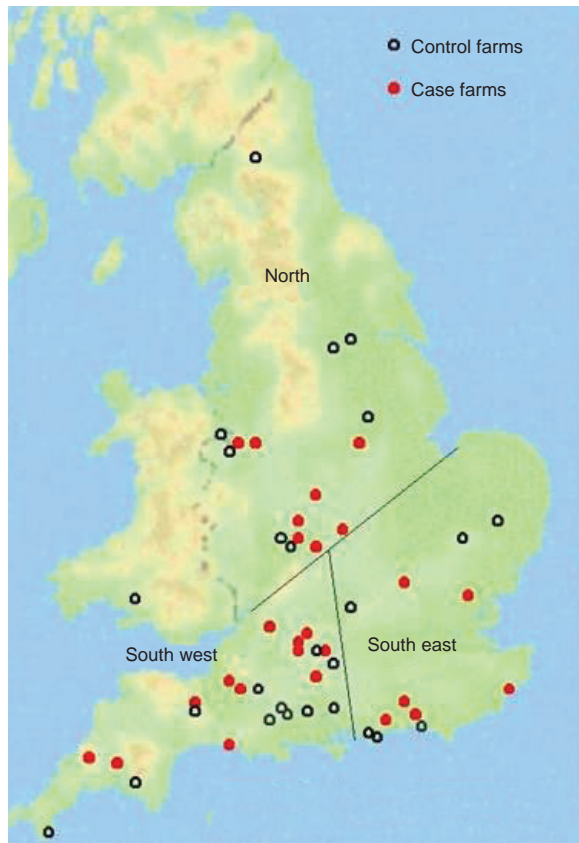
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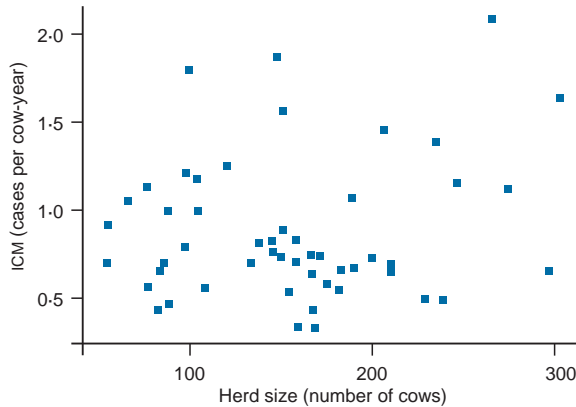
**FIG 1: Geographical location of the 52 farms in the study (Reproduced from Ordnance Survey map data by permission of the Ordnance Survey Crown copyright 2001)**

of the intervention. A further follow-up telephone call to the farmers was made by the veterinary surgeon in January 2005 to encourage their compliance. A final visit was made by a researcher to gather data from all the farms in April or May 2005.

Data on herd management procedures were collected by using structured, pre-tested questionnaires at interviews with the farmers, and from on-farm inspections of the cow housing and pastures, and by attendance at two milkings. The details collected in the questionnaires were in the following categories: general farm information, for example, herd size, yield and breed, staff, cow groups, bedding storage, feed and water (composition and provision), housing, pasture and tracks, fly control, milking plant and routines, pre- and post-milking yards and routines, management of calving cows and calves, management of youngstock, bio-security, and treatment regimens for the dry and lactating cows. Data from these visits were collated and used by the veterinary surgeons in preparing the intervention plan before their first visit.

**Intervention: a mastitis diagnosis and control plan**

**Preparing the intervention** The aim of the intervention was to use current research literature to develop a mastitis diagnosis and control plan (MDCP) and for two veterinary surgeons to implement it; the whole plan will be published on the Milk Development Council's website ([www.mdc.org.uk](http://www.mdc.org.uk)). It was not possible to carry out the intervention 'blind', because the farmers had to carry out the control procedures. The first stage of the control plan was to assess the patterns and types of mastitis in each herd.



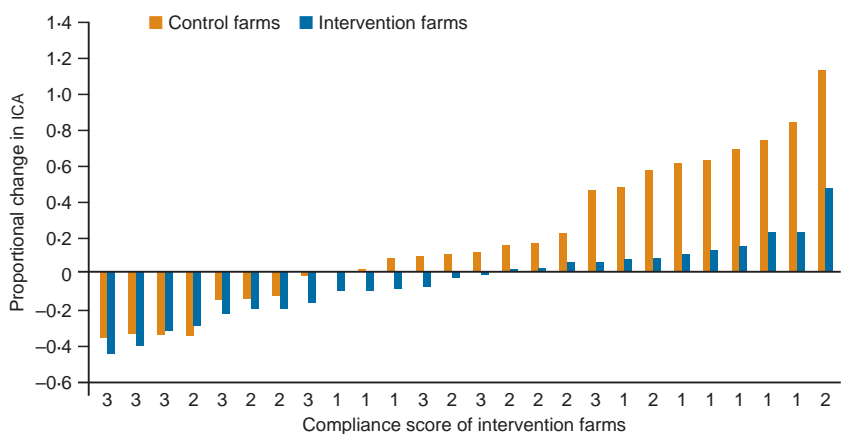
**FIG 2: Relationship between the incidence of clinical mastitis (ICM) and the size of the herds before the intervention**

**Assessment of mastitis patterns** At the beginning of the study bacteriological cultures were performed on a cohort of 10 cows with high SCCs and 10 cases of clinical mastitis on each farm, and the herds were categorised according to the following mastitis patterns. First, whether the pathogens were predominantly environmental organisms (such as *Escherichia coli* and *Streptococcus uberis*), or predominantly contagious organisms (such as *Staphylococcus aureus* and non-environmental streptococci), or if there were approximately equal proportions of environmental and contagious pathogens. Secondly, whether new infections occurred mainly during the dry period, mainly during lactation or equally in both (Bradley and Green 2005), and thirdly, whether the clinical cases occurred predominantly in summer, winter or equally in both seasons.

On the basis of these farm patterns of mastitis, control measures were developed by a structured process. The existing farm control measures were compared with the MDCP and the measures not currently being undertaken were highlighted. The MDCP had a hierarchy of three levels of importance attached to each control measure, termed 'could', 'should', and 'must', representing increasing levels of importance. The emphasis of the plan was different for the different categories of mastitis patterns, and, in accordance with these differences a set of between 10 and 20 management changes were recommended from the plan for each farm.

**Monitoring of compliance**

The compliance of the farmers was measured in terms of the proportion of the recommendations made from the MDCP that were actually implemented, as determined during fol-



**FIG 3: Proportional changes in the number of cows first affected in lactation/the number of cow-years at risk (ICA) after the implementation of the mastitis control plan, in ascending order of the size of the change, in relation to the compliance scores of the intervention farms**

**TABLE 1: Mean proportional changes in ICA, ICM and SCCNI after implementation of the mastitis control plan on the 26 intervention farms, on the intervention farms with different compliance scores, and on the 26 control farms**

	Control farms	Intervention farms	Intervention farms with a compliance score of		
			1 (n=9)	2 (n=9)	3 (n=8)
ICA	0.19	-0.04	0.07	-0.01	-0.20
ICM	0.18	-0.04	0.09	0.00	-0.21
SCCNI	0.16	-0.04	0.08	-0.05	-0.17

ICA Number of cows first affected in lactation/number of cow-years at risk, ICM Total number of cases of clinical mastitis/number of cow-years at risk, SCCNI Somatic cell count new infection

low-up visits both by observation and by questioning the farmers; a score of 1 was given when less than one-third of the recommendations were applied, 2 when between one and two-thirds were applied and 3 when more than two-thirds were applied. These scores were calculated at the end of the study, before the incidence of clinical mastitis on each farm was calculated.

### Statistics and analysis

A cow with a first case of clinical mastitis during lactation in a specified risk period was defined as a 'newly affected' cow, and the ICA over that period for herd 'i' was calculated as:

$$ICA_i = \text{Number of cows first affected in lactation/number of cow-years at risk}$$

Similarly, the ICM<sub>i</sub> was calculated as:

$$ICM_i = \text{Total number of cases of clinical mastitis/number of cow-years at risk}$$

The changes in ICA and ICM between year 1 (the 12 months before the intervention) and year 2 (the 12 months following the intervention) were calculated for both the intervention and control farms. The control farms shared the date of intervention with their matched farm, even though no intervention was carried out.

The change in the incidence of clinical mastitis between the years was divided by the initial rate of mastitis to give the proportional change in ICA (or ICM) after the mastitis control intervention. The proportional changes in ICA and ICM were used as the main outcomes for the statistical analysis for several reasons; first, they partially compensated for the higher numerical reductions in clinical mastitis that were generally observed with higher starting levels of mastitis; secondly, they provided a realistic and informative biological interpretation, that is, the change in the incidence of clinical mastitis that could be expected as a proportion of the starting level in the herd; and thirdly, the proportional changes in ICA

**TABLE 2: Linear regression models of the proportional changes in ICA and ICM with different intervention and compliance categories**

Variable	Coefficient	Standard error	P
<b>Model 1: proportional change in ICA for intervention and control farms*</b>			
Intercept	0.57	0.12	
Control farm	Reference category		
Intervention farm	-0.22	0.08	0.01
<b>Model 2: proportional change in ICA for farms with different compliance scores*</b>			
Intercept	0.58	0.12	
Control farm	Reference category		
Compliance score 1	-0.07	0.11	0.52
Compliance score 2	-0.23	0.11	0.04
Compliance score 3	-0.36	0.11	<0.01
<b>Model 3: proportional change in ICM for intervention and control farms†</b>			
Intercept	0.43	0.12	
Control farm	Reference category		
Intervention farm	-0.20	0.09	0.044
<b>Model 4: proportional change in ICM for farms with different compliance scores†</b>			
Intercept	0.45	0.12	
Control farm	Reference category		
Compliance score 1	-0.04	0.13	0.75
Compliance score 2	-0.18	0.13	0.17
Compliance score 3	-0.39	0.14	<0.01

\* Confounding covariate included: year 1 ICA

† Confounding covariate included: year 1 ICM

ICA Number of cows first affected in lactation/number of cow-years at risk, ICM Total number of cases of clinical mastitis/number of cow-years at risk

and ICM had an approximately normal distribution across the herds and this provided for straightforward model building and interpretation, and also for a good model fit (see below).

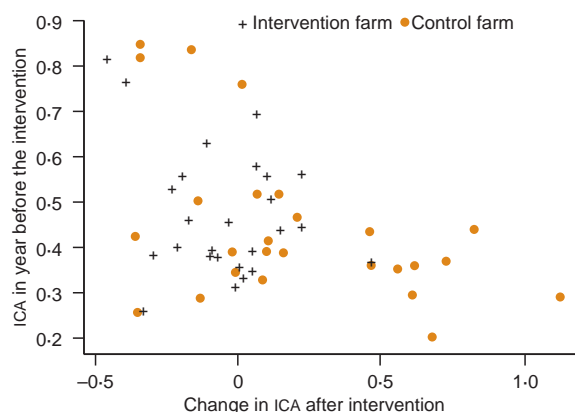
Descriptive statistics and an exploratory univariable analysis were carried out by using Minitab (Version 13.3; Minitab). Analyses of variance and Kruskal-Wallis tests were used for parametric and non-parametric continuous data, respectively (Petrie and Watson 1999). For clarity in the descriptive analyses, parametric data were summarised by using mean estimates, and non-parametric data were summarised by using median estimates. Statistical modelling was carried out by using conventional linear regression (McCullagh and Nelder 1989). The models took the general form:

$$Y_i = \alpha + \beta_1 I_i + \beta_2 C_i + e_i$$

where  $Y_i$  is the response for farm 'i';  $\alpha$  is the model intercept;  $I_i$  is a binary covariate for identifying farm 'i' as either an intervention or control;  $\beta_1$  is a coefficient for  $I_i$ ;  $C_i$  is a vector of confounding covariates;  $\beta_2$  is a coefficient for  $C_i$ ; and  $e_i$  is the residual error.

In each model, the mean annual milk yield per cow, and the mean herd size (both  $\log_{10}$  transformed to normalise the data) and the ICM in year 1 were tested as potential confounders. A probability of  $P < 0.05$  was considered significant. The fit of the models was checked by using plots of standardised residuals, assessments of homoscedasticity and investigations of the effects of data points with large influence or leverage values (Dohoo and others 2003). The models were recalculated after the omission of influential herds to determine whether the biological inference would be changed.

To investigate the efficacy of the MDCP further, changes in the SCC of individual cows were also examined. A somatic cell count new infection (SCCNI) was defined as an increase in SCC in consecutive recordings from below, to above 200,000 cells/ml (Bradley and Green 2005); cows in which the counts

**FIG 4: Relationship between the ratio of the number of cows first affected in lactation/the number of cow-years at risk (ICA) in the year before the mastitis control plan was implemented and the change in the ICA after it was implemented**

**TABLE 3: Linear regression models of the proportional changes in SCCNI with different intervention and compliance categories**

Variable	Coefficient	Standard error	P
<b>Model 5: proportional change in SCCNI for intervention and control farms</b>			
Intercept	0.16	0.06	
Control farm	Reference category		
Intervention farm	-0.21	0.08	0.02
<b>Model 6: proportional change in SCCNI for farms with different compliance scores*</b>			
Intercept	0.27	0.18	
Control farm	Reference category		
Compliance score 1	-0.08	0.11	0.62
Compliance score 2	-0.26	0.12	0.04
Compliance score 3	-0.30	0.13	<0.01

\* Confounding covariate included: log herd size  
SCCNI Somatic cell count new infection

were below 200,000 cells/ml at the last recording of one lactation and above 200,000 cells/ml at the first recording of the next lactation were included as an SCCNI. The number of SCCNIs was calculated for each farm as a proportion of SCCs that were originally below 200,000 cells/ml (and eligible to become an SCCNI) for the year before the intervention and the year of the intervention. The change in SCCNI between years was expressed as a proportion of the initial rate of SCCNI to give the proportional change in SCCNI following the mastitis control intervention. Statistical modelling was carried out by using the methods described above.

The impact of individual management recommendations on the proportional changes in ICA, ICM and SCCNI after the intervention was investigated by assessing the change in each of these parameters, depending upon whether each management recommendation was introduced. A non-parametric Kruskal-Wallis test was used to assess the statistical associations (Petrie and Watson 1999) and a probability  $P < 0.05$  was considered significant.

## RESULTS

The geographical location of the farms is shown in Fig 1. The median herd size was 156 cows. The intervention herds (median herd size 167) were slightly larger than the control herds (median 149) but the difference was not significant. The median milk yield of the 52 herds was 8500 litres per cow per year and the yield in the control herds (median 8650) was not significantly higher than in the intervention herds (median 8350).

### Incidence of clinical mastitis before the intervention

The median ICA in the 52 herds in the year before the intervention was 0.41 cows affected per cow-year, and there was no significant difference between the control herds (median 0.39) and the intervention herds (median 0.44). The median ICM in the 52 herds in the year before the intervention was 0.75 clinical cases per cow-year, and there was no significant difference between the control farms (median 0.73) and the intervention farms (median 0.79). There was no correlation between the initial incidence of clinical mastitis and the size of the herds (Fig 2).

### Compliance by intervention farms

Of the 26 intervention farms, eight complied with more than two-thirds of the recommended changes (score 3) nine complied with between one-third and two-thirds (score 2) and nine complied with less than one-third (score 1) of the recommended changes.

**TABLE 4: Number (%) of intervention farms with different compliance scores that made management changes specified by the mastitis diagnosis and control plan**

Management change	Compliance score		
	3	2	1
<b>Improved milking machine function</b>			
Correct machine faults identified by expert	4 (50.0)	6 (66.7)	4 (44.4)
Improve cluster alignment during milking	0	0	1 (11.1)
Reduce linear slip during milking	0	0	1 (11.1)
<b>Improved milking machine hygiene</b>			
Wash clusters if become dirty during milking	1 (12.5)	1 (11.1)	0
Improve full machine wash after milking	2 (25.0)	0	0
<b>Changes to teat cup liners</b>			
Change dump liners when changing other liners	1 (12.5)	1 (11.1)	0
Change all liners at least every 2500 milkings	2 (25.0)	1 (11.1)	0
Maintain liners in good condition	0	0	1 (11.1)
<b>Improved teat preparation before milking</b>			
Start pre-dipping	5 (62.5)	2 (22.2)	1 (11.1)
Dry teats if washed	3 (37.5)	1 (11.1)	0
Wash teats if dirty	3 (37.5)	0	0
Change milking preparation order to: wash and dry (if dirty); forestrip; pre-dip; dry; milk	2 (25.0)	0	0
<b>Improved milking of infected cows</b>			
Milk cows with clinical mastitis and high SCCs separately	3 (37.5)	2 (22.2)	0
Milk cows with clinical mastitis and high SCCs using a separate cluster	1 (12.5)	0	0
Clearly mark high SCC cows	0	2 (22.2)	0
Disinfect clusters and claw piece after milking cows with clinical mastitis	2 (25.0)	2 (22.2)	0
<b>Improved milking routine</b>			
Apply cluster <60 secs after preparation	3 (37.5)	1 (11.1)	0
Take approximately 1 minute from teat preparation to attaching the units to the cow	1 (12.5)	0	0
Wear disposable gloves	1 (12.5)	0	1 (11.1)
Reduce over-milking	2 (25.0)	0	1 (11.1)
<b>Improved post milking teat disinfection (PMTD)</b>			
PMTD >10 ml/cow for dip or 15 ml for spray	1 (12.5)	2 (22.2)	0
PMTD <30 secs after units off	0	2 (22.2)	0
Improve PMTD coverage	4 (50.0)	2 (22.2)	3 (33.3)
Check sprays working at each milking	1 (12.5)	0	0
Monitor post dip use	0	1 (11.1)	0
<b>Improved mastitis detection</b>			
Foremilk	0	0	1 (11.1)
Check in-line filters	1 (12.5)	1 (11.1)	0
<b>Improvements pre- and post milking</b>			
Improve ease of access to parlour	1 (12.5)	0	0
Improve post-milking yard hygiene	0	1 (11.1)	0
Stand for 30 minutes after milking	0	1 (11.1)	0
<b>Improved milking cows' environment: lactating cow stocking</b>			
Increase milkers' space allowance to >1.25 m <sup>2</sup> /1000 litres (mean herd yield)	1 (12.5)	0	0
Improve pasture rotation of milking cows	0	1 (11.1)	0
<b>Improved milking cows environment: lactating cow hygiene</b>			
Increase straw usage to over 250 kg per cow per month while housed	0	0	1 (11.1)
Clean out winter yards at least monthly	0	0	1 (11.1)
Apply clean bedding to yards daily	0	2 (22.2)	0
Scrape milking cow passageways twice daily	0	1 (11.1)	0
Improve hygiene of cubicle beds	0	1 (11.1)	2 (22.2)
Improve cubicle structure	2 (25.0)	0	0
Improve housing for milkers in summer	1 (12.5)	0	0
Reduce poaching of milking cows' pasture	2 (25.0)	0	0
Manage gateways used by milking cows	3 (37.5)	0	0
Ensure fly control for milking cows	0	0	1 (11.1)

### Changes in the incidence of clinical mastitis

The median ICA in the year after the implementation of the intervention plan was 0.54 cases per cow-year on the control farms and 0.43 on the intervention farms. The median ICM after the intervention was 0.94 cases per cow-year on the control farms and 0.75 on the intervention farms.

The proportional changes in ICA and ICM after the intervention are shown in Table 1. Overall, the mean ICA increased by 19 per cent on the control farms and decreased by 4 per cent on the intervention farms. The mean ICM increased by approximately 18 per cent on the control farms and decreased by 4 per cent on the intervention farms. The reductions in both ICA and ICM increased with increasing compliance with the plan (Table 1).

The proportional changes in ICA in the individual herds, and their compliance level, are shown in Fig 3; there was some

TABLE 4: continued

Management change	Compliance score		
	3	2	1
<b>Improved dry cow environment: transition cow stocking</b>			
Improve pasture rotation	2 (25.0)	1 (11.1)	0
Increase space allowance to >1.25 m <sup>2</sup> /1000 litres (mean herd yield)	2 (25.0)	0	0
<b>Improved dry cow environment: transition cow hygiene</b>			
Scrape transition cows' alleys twice daily	1 (12.5)	0	1 (11.1)
Bed transition cows daily	0	0	1 (11.1)
Spread transition cow bedding evenly	0	1 (11.1)	0
Clean out transition cow yards monthly	0	1 (11.1)	0
<b>Improved dry cow therapy (DCT)</b>			
Correct administration technique	5 (62.5)	7 (77.8)	5 (55.6)
Do not dry off and foot trim together	0	1 (11.1)	0
Dry off abruptly	1 (12.5)	2 (22.2)	1 (11.1)
Start differential DCT or seal teat internally*	0	3 (33.3)	2 (22.2)
Reduce yield pre dry-off	2 (25.0)	3 (33.3)	0
Dry period 42-70 days	0	1 (11.1)	0
<b>Changes to minerals/vitamins daily intake</b>			
Increase vitamin E to 550 iu/day for milkers	3 (37.5)	5 (55.6)	1 (11.1)
Increase vitamin E to 1200 iu/day for dry cows	3 (37.5)	3 (33.3)	0
Increase vitamin A to 83,000 iu/day for dry cows	0	1 (11.1)	0
Increase vitamin A to 75,000 iu/day for milkers	0	1 (11.1)	0
Increase selenium to 6.6 mg/day for milkers	1 (12.5)	1 (11.1)	1 (11.1)
<b>Changes to water supply</b>			
Use mains drinking water	1 (12.5)	0	0 (0.0)
Test mains water	0	1 (11.1)	0 (0.0)
Water in post milk yard ad libitum	0	0 (0.0)	1 (11.1)
<b>Improvement to the treatment of clinical mastitis</b>			
Treat all clinical cases including doubtful cases	3 (37.5)	1 (11.1)	2 (22.2)
<b>Bedding storage</b>			
Cover all straw	1 (12.5)	1 (11.1)	0
<b>Improvements to calving cow environment</b>			
Scrape calving area alleys daily	0	0	1 (11.1)
Bed calving cows daily	0	0	1 (11.1)
Clean out calving yards every four weeks	0	0	1 (11.1)
Improve calving area drainage	0	1 (11.1)	1 (11.1)
<b>Changes to calf management</b>			
Reduce cross suckling	1 (12.5)	0	0
Remove calf <24 hours after birth	0	1 (11.1)	0
<b>Youngstock</b>			
Heifers in clean dry environment	1 (12.5)	0	0

\* Bismuth subnitrate in an oily base (Orbeseal; Pfizer Animal Health)

overlap between the control and intervention farms in the magnitude of the changes in ICA. The relationship between the proportional change in ICA and the ICA in the year before the intervention is shown in Fig 4; higher initial levels of ICA were generally associated with a larger proportional reduction in ICA; the pattern was similar for ICM.

### Descriptive statistics of the incidence of SCCNI after the intervention

The proportional changes in SCCNI in the intervention and control herds are shown in Table 1. Overall, the median number of SCCNI increased by 16 per cent in the control herds and decreased by approximately 4 per cent in the intervention herds. As with ICA and ICM, higher levels of compliance with the intervention resulted in greater reductions in SCCNI.

### Models of change in the incidence of clinical mastitis

The statistical models of the changes in ICA and ICM, including confounding covariates (models 1 to 4), are given in Table 2. There was a significant 22 per cent reduction in the proportion of cows affected with clinical mastitis on the intervention farms compared with the control farms ( $P<0.05$ , model 1) (Table 2), there were also significant reductions in ICA of 23 per cent in the herds with a compliance score of 2, and 36 per cent in the herds with a compliance score of 3, in comparison with the control herds ( $P<0.05$ , model 2) (Table 2).

There was a significant reduction in the ICM of 20 per cent on the intervention farms compared with the control farms ( $P<0.05$  model 3) (Table 2) and a significant reduction in

ICM of 39 per cent in the herds with a compliance score of 3, compared with the control farms ( $P<0.05$ , model 4) (Table 2). The fit of models 1 to 4 to the observed data was good and the omission of data points with high leverage or influence values did not affect the biological inferences made from any of the models.

### Models of change in somatic cell counts

The statistical models of the changes in SCCNI (models 5 and 6) are shown in Table 3. There were no confounding covariates and there was a reduction in the SCCNI of 21 per cent on the intervention farms compared with the control farms ( $P<0.05$ ). After accounting for the confounding covariate, herd size, there were reductions in the SCCNI of 26 per cent in the herds with a compliance score of 2, and 38 per cent in the herds with a compliance score of 3, compared with the control herds ( $P<0.05$ ). The fit of models 5 and 6 was good.

### Management changes undertaken by the intervention farms

Seventy-two different management changes were made by the 26 intervention farms (Table 4). The eight herds that had a compliance score of 3 made a total of 80 management changes (some changes were carried out by more than one farm), a mean of 10 per farm. On average, the nine farms with a compliance score of 2 made 7.8 management changes, and the nine farms with a compliance score of 1 made 4.2 changes. No individual management change was significantly associated with a reduction in the overall proportional change in ICA, ICM or SCCNI.

### DISCUSSION

The results of the present study show that it is possible to make a large improvement in mastitis control on dairy farms in the course of one year. The success of the intervention plan depended upon the accurate identification of the mastitis risk factors by using the MDCP, and also upon the correct implementation by the farmer of the changes recommended. Both of these elements were achieved in the herds that were successful in reducing the incidence of mastitis.

This is the first controlled intervention study in the UK, and possibly worldwide, to examine the effect of a clearly specified control plan for clinical mastitis. Such an approach is necessary to quantify the true effect of the potential risk factors identified. There have been few intervention studies in the field of animal health, except in discrete areas such as treatments or individual management practices such as teat dipping. This may be because of the difficulties of conducting them, but such studies are essential for robust inferences to be drawn about the effectiveness of disease control measures.

However, there were considerable variations between both intervention and control farms, in the changes observed in the incidence of mastitis. Some intervention herds made little or no improvement and some control herds made reasonable improvements. One component of this variability was the level of compliance of the intervention farmers with the MDCP; variations in compliance resulted in considerable differences in the success of the plan, indicating that motivation and encouragement to implement the control measures are essential elements of mastitis control and are likely to be important in implementing any disease control scheme.

On the control farms, there was an overall increase in the incidence of mastitis between years one and two of the study, with the ICA, ICM and SCCNI increasing by 16 per cent to 19 per cent. The reasons for the increase are not clear but it could be a result of changes in unknown factors, such as climate. There was also considerable variation between the control farms in the individual variability of ICA between years one and

two (Fig 3). Again, the causes are not clear, and the variation may simply be due to year-to-year variability on farms on which mastitis control is unfocused. The control farms were not prevented from making management changes, and they may have made changes that resulted either in an increase or decrease in the incidence of mastitis.

Individual management changes made by the farmers were not significantly associated with changes in the incidence of mastitis, implying that the overall improvements were a result of a cumulative effect of a number of changes. This may explain why the farms with lower compliance scores were less successful in controlling mastitis; too few changes were made for a significant difference to be detected. This is crucial to understanding the results of the study and hence the likely impact of the MDCP. There was an overall benefit from the intervention plan because of the improvements in the herds that complied with at least one-third of the recommendations, and more from the herds that complied with at least two-thirds of the recommendations. A sensible interpretation is that to be sure of making an improvement, farmers need to comply fully with the MDCP and not select only the management practices from the plan with which they agree or which may be easily implemented.

The MDCP was based on existing information, and the findings therefore suggest that there may be sufficient knowledge to reduce the current incidence of mastitis in the UK, but that its application, and also further education, knowledge transfer and motivation may remain essential to achieving improved mastitis control. The question of who is responsible for coordinating the control of mastitis and other endemic diseases, and thereby improving the life of farm animals is difficult, but it needs to be considered if improvements, particularly in the common endemic conditions, such as mastitis and lameness, are to be made. If the results of this study are applicable to UK dairy farms, then it should be possible to reduce the incidence of clinical mastitis (in herds with an ICM of more than 35 cases per 100 cows per year) by the order of 20 per cent in one year. This would have health, welfare and financial implications for the dairy industry and consideration should be given to how this type of plan could work on a nationwide basis. The MDCP provides a guide for veterinary advisors to follow in order to achieve such results.

A detailed economic assessment of the intervention was not the aim of the study, but some simple economic evaluation is of interest. If the current 'average' cost of a case of clinical mastitis is accepted as approximately £175 (Kossaibati 2000), then after one year of implementing the MDCP a 100-cow dairy farm with a starting ICM of 60 cases per 100 cows per year would be expected, on average, to prevent 12 cases, a saving of £2100. With full compliance with the MDCP approximately 18 cases would be prevented, with a financial saving of £3150. These estimates give an indication of the potential savings in the control of clinical mastitis alone, and therefore a guide to what expenditure would be sensible to achieve a cost benefit from the MDCP. If it would cost more to implement the plan than the potential savings, it may not be worthwhile. However, such a calculation does not include the improvement in the animals' welfare or the savings possible from controlling SCCs such as improved milk yield and increased milk value. Most of the management changes undertaken as part of the MDCP were relatively cheap and involved no large capital expenditure (Table 4), suggesting that in many cases improved mastitis control may not necessarily be expensive. This conclusion agrees with previous reports that the monitoring and control of mastitis is generally cost effective (Blowey 1986, Morin and others 1993, Yalcin and Stott 2000).

The target population in the study was identified from NMR, so that information on clinical mastitis from a wide geographical base of farms could be used for selection pur-

poses and so that SCC records were available from all the farms. Of the 250 farms initially identified with a recorded ICM of more than 35 cases per 100 cows per year, 68 (27.2 per cent) responded positively to be included in the study. It is uncertain whether the 52 farms selected were representative of the farms with an incidence of clinical mastitis of more than 35 cases per 100 cows per year in England and Wales as a whole. It is inevitable that field studies of this nature will suffer from selection bias to some extent, because the farms volunteering to participate are likely to be different from the whole target population; however, the size of the herds and their average annual milk yield were similar to published national data (Milk Development Council [MDC] 2006), providing some evidence that they were reasonably representative of the target population.

The implementation of a well specified mastitis control plan led to a reduction of approximately 20 per cent in cases of clinical mastitis and somatic cell counts within a year. This result has important health, welfare and financial implications for the dairy industry and consideration should be given to how such disease prevention strategies could be put into practice on a larger scale.

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