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CONTENTS

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QUANTITATIVE METHODS

THE USE OF MULTI-LEVEL MODELLING IN VETERINARY EPIDEMIOLOGY

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The analysis of data from observational epidemiological studies of farmed animals presents particular problems because they are kept in groups and are consequently, not entirely independent of their conspecifics e.g. lambs born into one litter and sheep managed in one flock are more alike than lambs from different litters and sheep from different flocks, both because of genetic and environmental influences.

These groups (litters and flocks) are known as hierarchical clusters i.e. they contain 'lower level' elements (Goldstein, 1995): flocks are comprised of ewes, and ewes produce litters of lambs. A hierarchical structure also occurs when there are repeated measurements on one individual, these measurements form the lowest level of a hierarchy and the individuals , each with several measurements, form the next level. Where there are several levels of clusters the structure is known as a multilevel hierarchy.

A basic epidemiological principle is that the study sample should reflect the target population, so it seems intuitive that, if the population is hierarchically structured, the most precise estimations will result when the sample is also. It is therefore important to either account for clustering of data in the design and analysis of a study.

Because data from individuals within a cluster are not entirely independent, analysis of these data assuming independence flaunts the requirements of many statistical tests and can lead to an underestimation of standard errors and confidence intervals (McDermott & Schukken, 1994). There has therefore been an increased effort to include and to account for clustering of data. Several analytical approaches can account for one level of clustering e.g. random effects and mixed effects models (Curtis et al, 1993; Atwill et al, 1995; McDermott et al, 1994). There are now advanced techniques for handling multilevel hierarchical clusters. These are not yet widely used in epidemiology (McDermott, 1995). This paper presents an example of multilevel modelling used to analyse data from a prospective longitudinal study of housed lambs with unbalanced repeated measurements. The multilevel model investigated the effect of diarrhoea on the weight of lambs.

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MATERIALS AND METHODS

There were three flocks in this study, A, B and C, each flock was managed with a similar husbandry system. Ewes were housed, on straw or slats, in groups of 40 - 200 from two to six weeks before lambing in December or January, and fed *ad libitum* silage and a limited quantity of commercial concentrate ration. Once the entire litter was born the lambs and ewes were put into individual 'lambing' pens for approximately 24 hours. They were then moved into large straw - bedded, naturally ventilated barns where the lambs remained until they were finished. The lambs were fed *ad libitum* proprietary concentrates from 10 days old to slaughter. They were weaned when 6 - 8 weeks old and slaughtered between 10 and 26 weeks (median age; 14 weeks).

During the lambing season 1989 - 1990, three randomly selected cohorts of approximately 80 lambs, one from each of flocks A, B and C were examined each week. Each lamb was given an initial score of healthy or sick (lack of vigour with overt dullness) and was then examined using a defined clinical protocol. This included examining the perineum for signs of diarrhoea (wet or dry faecal soiling of perineal wool), and examining faecal consistency. Diarrhoea was recorded when liquid faeces were seen during the lamb's clinical examination. The lambs were weighed at the end of each examination.

Data were stored in a database (dBase III plus, Ashton Tate, Ashton Tate Corporation). Errors were checked by running frequency distributions of variables; outlying values and impossible codings were checked with the raw data and corrected where necessary.

The prevalence and exact 95% confidence intervals (Geigy, 1982) of diarrhoea were calculated. Univariate analysis was performed in Epi Info 6.0 (Dean et al., 1990). The association between ever having had an episode of diarrhoea and the mean weight gain from first to last examination was investigated using Student's t test.

MLn, a multi-level models programme (Woodhouse, 1995), was used to for more complex analysis of these longitudinal data with repeated measures. The outcome variable was lamb weight in kilograms. The independent variables tested as fixed effects were: flock, age in weeks, and quadratic and cubic values of age, litter size, sex and diarrhoea. The effect of diarrhoea was investigated further by creating six new dummy variables. Five of these examined the lag effect of an episode of diarrhoea at weekly intervals from the observed recording of diarrhoea for five weeks and the sixth variable was used to investigate whether there was any effect on the weight of lambs one week before an episode of diarrhoea.

There were three hierarchical levels in the model: (i) each examination (level 1) clustered by (ii) each lamb (level 2) clustered by (iii) each litter (level 3). Rather than confining the components of variation by examination, lamb and litter to simple variation (i.e. a constant variation for all values of the independent variables), complex variation (i.e. variation which altered and was therefore dependent upon the values of the independent variables) (Woodhouse G, 1995a) was investigated at all three levels: age and the quadratic of age were tested.

The age of the lambs was centred at 10 weeks so that the intercept values of the random variation at the different levels of the hierarchy, displayed in the output, were centred around the variation in lamb weight at 10 weeks rather than at birth.

The significance probability (p) was set at 0.05 for a two tailed test. Variables were left in the model when there was a significant ($p = < 0.05$) reduction in the likelihood ratio statistic (LRS) using Chi squared tables.

The final model was:

fixed effects
Lamb weight = constant + a(age) + b(age squared) + c(litter size) + d(sex) + e(farm) + f(diarrhoea) + g(diarrhoea-1) + h(diarrhoea-2) + i(diarrhoea-3) + j(diarrhoea-4)

random effects
level 1
variation in examinations = constant + k(covariance age) +l(age)
level 2
variation between lambs (controlling for litter effects) = constant + m(covariance age) + n(age) + o(covariance age squared) +p(covariance age and age squared) + q(age squared)
level 3
variation between litters = constant + r(covariance age) +s(age)

where: a - r = coefficients, diarrhoea-1 = diarrhoea in previous week, diarrhoea-2 = diarrhoea in two weeks previously etc. for diarrhoea-3 and diarrhoea-4.

The mean growth curve was estimated from the fixed effects in the model. Then, for each hierarchical level the total variation was calculated from a quadratic function of the constant, covariance and variance terms. For example, the total ewe level variation was estimated from the following equation:

ewe level variation = 6.69 + (2 x 0.74 x 'age') + 0.09 x 'age'$^{\wedge 0.5}$ (Woodhouse, 1995b).

The standard deviation was then estimated from the squared root of this equation and the upper and lower 95% growth curves calculated by multiplying the standard deviation by two and adding (upper) or subtracting (lower) the result to or from the mean growth curve respectively.

The 95% confidence limits for the growth of lambs by occasion, lamb and litter were then plotted against age in weeks.

RESULTS

There were a total of 2178 examinations. Each lamb had from one to 19 examinations. The proportion of cohort lambs examined by week of age ranged from 81% to 98.5%.

The risk of a lamb ever having diarrhoea during its life was 17.1% (13/76), 40.0% (30/75) and 21.3% (17/80) for flocks A, B and C respectively. Flock B had more lambs with diarrhoea than flocks A and C, although this was not significant (P = 0.17). The majority of lambs had one episode

of diarrhoea but three, six and five lambs, from flocks A, B and C respectively, had diarrhoea on two occasions. The mean weight gain in lambs from first to last examination was 23.7 kg for lambs with diarrhoea and 23.8 kg for lambs without ($p<0.85$). Diarrhoea was seen in lambs of all ages (Table 1), the peak prevalence was 3 - 5 weeks of age. None of the lambs observed with diarrhoea were clinically sick.

Table 1. Prevalence of diarrhoea by age of lamb

Age	Flock A		Flock B		Flock C	
(weeks)	% (prop^n)	95% CI	% (prop^n)	95% CI	% (prop^n)	95% CI
<1	0.0 (0/35)	0.0 - 10.0	0.0 (0/51)	0.0 - 7.0	3.0 (2/66)	0.4 - 10.5
1	1.7 (1/60)	0.0 - 8.9	1.6 (1/62)	0.0 - 8.7	0.0 (0/64)	0.0 - 5.6
2	1.6 (1/61)	0.0 - 8.8	1.6 (1/62)	0.0 - 8.7	1.5 (1/68)	0.3 - 10.2
3	0.0 (0/66)	0.0 - 5.6	18.8 (12/64)	10.1 - 30.5	4.1 (3/73)	0.9 - 11.5
4	11.3 (7/62)	4.7 - 21.9	23.1 (15/65)	13.5 - 35.2	14.7 (11/75)	7.6 - 24.7
5	11.1 (6/54)	4.2 - 22.6	6.5 (4/62)	1.8 - 15.7	2.7 (2/72)	0.3 - 9.7
6	0.0 (0/39)	0.0 - 9.0	2.6 (1/38)	0.1 - 13.8	2.1 (1/47)	0.0 - 11.3
7	0.0 (0/51)	0.0 - 7.0	0.0 (0/58)	0.0 - 6.2	1.5 (1/65)	0.0 - 8.3
8	0.0 (0/36)	0.0 - 9.7	2.4 (1/41)	0.1 - 12.9	2.0 (1/49)	0.1 - 10.9
9	0.0 (0/48)	0.0 - 7.4	0.0 (0/60)	0.0 - 6.0	0.0 (0/64)	0.0 - 7.3
10	0.0 (0/37)	0.0 - 9.5	2.2 (1/68)	0.1 - 11.8	1.4 (1/70)	0.0 - 7.7
11	0.0 (0/50)	0.0 - 7.1	0.0 (0/45)	0.0 - 7.9	0.0 (0/67)	0.0 - 5.4
12	2.9 (1/34)	0.1 - 15.3	0.0 (1/17)	0.0 - 19.5	0.0 (0/59)	0.0 - 6.1

% = percent, 95% CI = 95 percent confidence intervals, prop^n = proportion

From the multilevel model (Table 2), it can be seen that the constant was a 10 week old female, single lamb weighing 24.53 kg from flock A, without diarrhoea. The fixed effects: age (2.2 kg per week increase), the quadratic of age (0.04 kg per week increase), litter size (e.g. reduction of 2.1 kg in absolute weight for twins), sex (increase of 0.54 kg for male lambs versus female) and flock (e.g. flock C lambs were 1.5 kg heavier than flock A) all had significant effects on the weight of lambs.

Table 2. Multi-level model of the fixed effects affecting the weight of lambs.

Parameter	Estimate	Standard error	X^2 value	P value
Constant (centred at 10 weeks old)	24.53	0.414		
Age	2.20	0.043	5737.5	<0.001
Age squared	0.04	0.003	44.3.60	<0.001
Twin	-2.06	0.290	12.3	<0.001
Triplet	-2.54	0.360	17.6	<0.001
Quadruplet	-4.06	1.010	4.8	0.03
Male	0.54	0.180	6.6	0.01
Flock B	-0.22	0.290	7.2	0.007
Flock C	1.49	0.31	23.3	<0.001

Estimate = final estimate of variables effect on weight of lambs at end of model
Standard error = standard error of estimate
X^2 value = value as variable was fitted into model
P value = probability of chance effect

After accounting for these factors, there was a significant reduction in the LRS for the effect of diarrhoea on the weight of lambs. The weight of lambs with diarrhoea was less than that for unaffected lambs. Their weight continued to decrease each week for four weeks after an episode of diarrhoea (Table 3). After this there was no further reduction in the weight of lambs with diarrhoea. Once lambs had had diarrhoea they remained approximately 2 kg lighter than lambs which had not had diarrhoea (Figure 1). There was no effect on the weight of lambs in the week prior to an episode of diarrhoea.

Table 3. Effect of diarrhoea on weight of lambs during the week of diarrhoea and with up to four weeks lagging, after the removal of confounding factors

Variable	coefficient	s.e.	X^2 value	P value
Diarrhoea	-0.30	0.09	0.2	0.65
Diarrhoea - 1 week	-0.49	0.11	7.6	0.006
Diarrhoea - 2 week	-0.55	0.13	12.4	<0.001
Diarrhoea - 3 week	-0.40	0.15	5.5	0.02
Diarrhoea - 4 week	-0.35	0.17	4.6	0.03

Estimate = final estimate of variables effect on weight of lambs at end of model
Standard error = standard error of estimate
X^2 value = value as variable was fitted into model
P value = probability of chance effect

There was significant complex random variation at all three hierarchical levels. There was a significant reduction in the LRS when the variance and covariance of age (levels 1, 2 and 3) and the quadratic of age (level 2) with the constant and age terms respectively were included (Table 4).

Figure 1 Average growth curves for lambs with and without diarrhoea

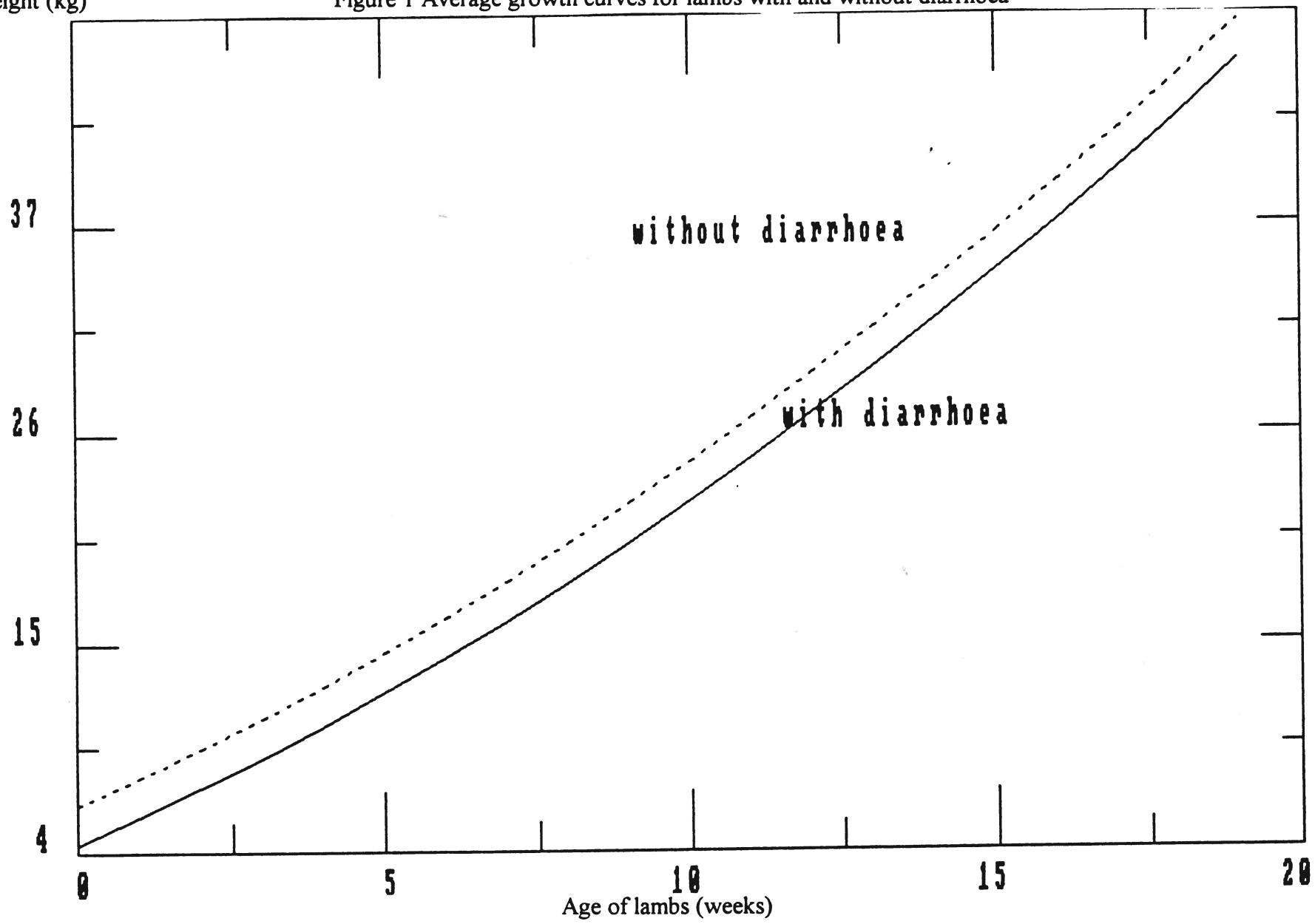


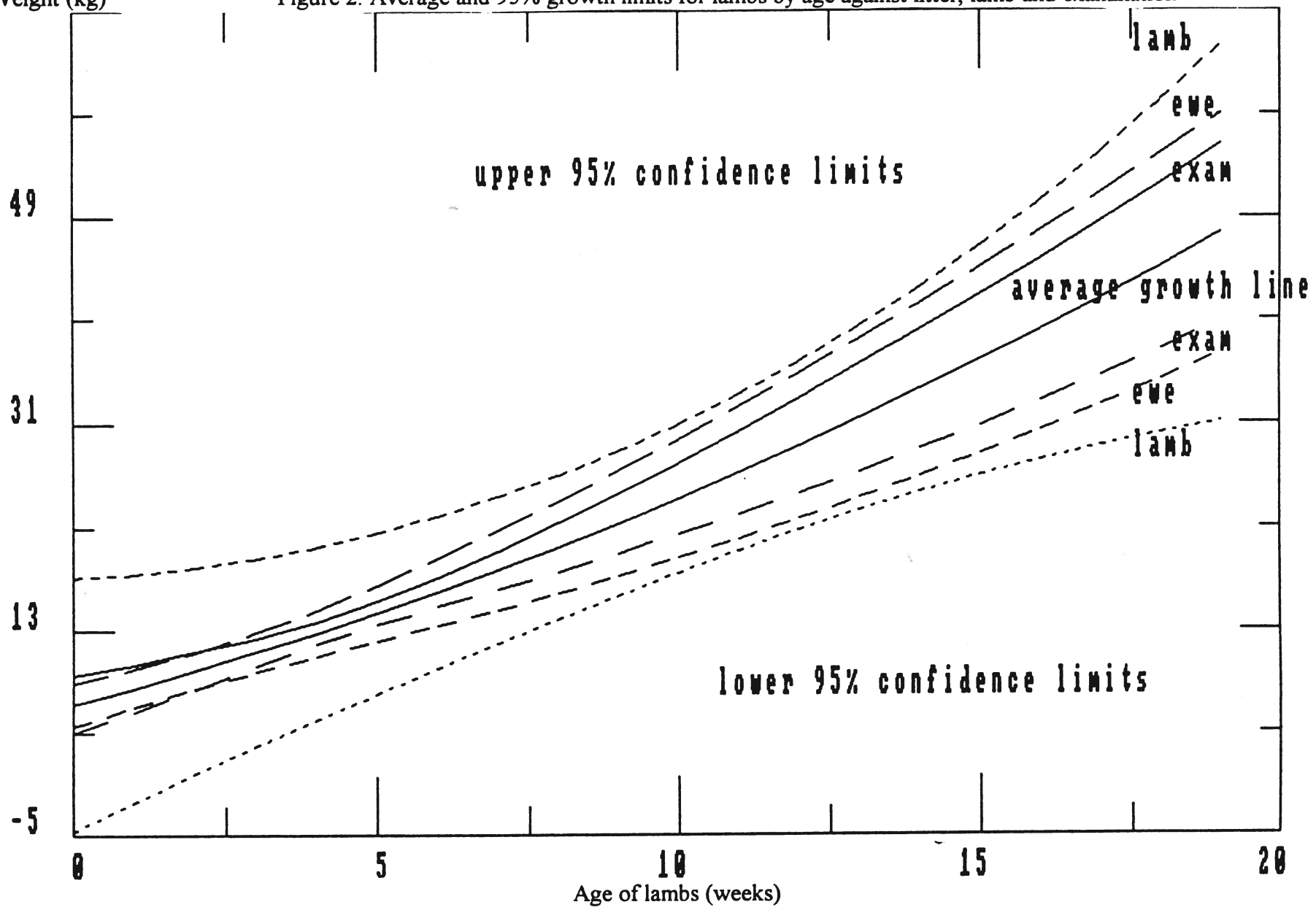
Table 4. Multi-level model of the random effects affecting the weight of lambs.

Parameter	Variance estimate	Standard error	X^2 value	P value
Ewe				
Constant	6.69	1.89		
Covariance of ewe and age	0.74	0.20		
Age	0.09	0.022	809.5	<0.001
Lamb				
Constant	10.48	1.65		
Covariance of lamb and age	0.37	0.16		
Age	0.14	0.27	492.5	<0.001
Covariance of lamb and age squared	0.07	0.01		
Covariance of age and age squared	0.01	0.002		<0.001
Age squared	0.002	0.0003	226.4	<0.001
Examination				
Constant	2.41	0.12		
Covariance of exam and age	0.39	0.02		
Age	0.07	0.004	191	<0.001

Variance estimate = final estimate of the variance around the mean growth line of lambs
Standard error = standard error of the variance estimate
X^2 value = value as variable was fitted into model
P value = probability of chance effect

The mean litter, lamb and examination growth curve by age for levels 3, 2 and 1 of the hierarchy was estimated (Figure 2). For each level in the model there were different 95% confidence limits about this line. At all three levels, this variation initially decreased and then increased with age (Figure 2). The variation in weight attributable to the examinations (level 1) diverged at each end of the growth curve. The main increase in variation occurred from week seven. The greatest variation occurred between individual lambs (level 2). As with level one, this was also divergent at both ends of the age range and at a minimum at 10 weeks of age. The litter variation was initially less than the variation observed between examinations. It became greater than the examination variation and continued to increase from when the lambs were two to three weeks old.

Figure 2. Average and 95% growth limits for lambs by age against litter, lamb and examination



DISCUSSION

The key finding in this analysis is that an episode of diarrhoea did reduce the weight of affected lambs, the mean growth curve indicated that they were approximately 2 kg lighter than lambs which had not had diarrhoea (Figure 1). This difference could not be detected in the simple analysis when the first and last weights of the lambs were compared, this is almost certainly because of the large variation in the weight of lambs (observed in this analysis at level two), e.g. at ten weeks of age the lambs weight ranged from 18.1 - 31.0 kg ($24.53 +/- 2 \times 10.48^{\wedge 0.5}$), the effect of confounding variables (the other fixed effects) and because the lambs were slaughtered when they were finished, which was dependent upon their weight and fat cover rather than their age.

It was very interesting to note that lambs with diarrhoea not only weighed less during the week that they had diarrhoea, but that this reduced absolute weight continued to increase for the subsequent four weeks. This indicates the real effect of diarrhoea on the health of a lamb. Farmers can now be advised that even when lambs are not overtly sick with diarrhoea, they will have a reduced weight for age and will therefore cost more to rear to slaughter. This should encourage the prevention of these apparently mild diseases and improve the health of lambs.

The variation observed at the different levels of a hierarchy are also extremely interesting and important and provide new interpretations from this study. The 95% confidence limits (Figure 2) represent the boundaries within which 95% of the population litter, lamb and examination growth curves are expected to fall. The ewe level variation (level 3) indicated that the litter effect did not disappear, or even decrease, at weaning, when the ewe and consequently the milk supply were withdrawn, rather the litter continued to exert an effect on the variation in the weight of the lambs after weaning and this effect increased as the lambs age increased. The litter effect will include birth weight, any environmental exposures attributable to the day of birth and management during rearing specific to each litter and genetic influences, both from sire and dam. These effects will all continue even in the absence of the ewe, consequently it is biologically plausible and interesting to note that the litter level variation continues after the ewe is withdrawn.

The majority of the variation in lamb weight occurred between lambs, at level 2, as one would expect. There are many factors at this level which were not and could not be estimated which would influence the growth of the lambs (e.g. colostral intake, sub-clinical disease, appetite, behaviour, activity). The variation can be explained in part by cohort lamb ascertainment. Several cohort lambs from flocks A and B were not identified until they were 2 - 3 weeks old (Green et al, 1994) and, at the end of the study 50% of the lambs were killed by 14 weeks (Green et al, 1995) so the sample size was lower at both ends of the age spectrum. This may explain the diverging confidence limits at the extreme ends of the growth curve.

The examination level variation quantitates the variation which the authors knew was likely to be present, because of the study design errors and also within lamb variation but could not previously estimate. The examination curves diverge from the mean growth curve from when lambs were six weeks of age. This is interesting since at approximately six weeks of age the rumen becomes functional, the rumen can weigh up to 20% of the live body weight of a sheep therefore the degree of rumen fill between examinations would affect the weight of a lamb considerably. This may account for some of the increase in the variation in weight by examination from six weeks (Figure 2). Also, lambs were weighed on clock scales with an accuracy of +/- 20 g until they weighed 20 kg; (at 7 - 10

weeks of age) they were then weighed on calibrated farm scales with an accuracy of 0.5 kg. The method used to weigh the lambs would have affected the variation in lamb weight by occasion.

One other possible effect which may account for some of the occasion variation is recording error (Rothman, 1986). As this study proceeded the researchers had to contend with physical tiredness and mental fatigue from a fairly repetitive task. It is possible, although we would like to think unlikely, that the degree of care taken when observing the lambs was reduced and consequently that the recording error increased. This non-differential misclassification would have increased the variation between examinations within lambs (level 1), between lambs (level 2) and between litters (level 3).

It is rewarding to have found an technique to analyse hierarchically clustered, unbalanced repeated measures data, which was arduous and time consuming to collect and which was not analysed to its full potential by other more conventional statistical methods. Clustered data can either be avoided by using only one member of a group in an investigation. However this prevents any estimation on the effect of grouping from being made e.g. if only one lamb from each litter had been examined, the litter variation could not have been estimated. Clustering can also be avoided by collecting data at a the higher cluster level Data must be interpreted at that same level. Any interpretation to lower levels, e.g. assuming that flock level data is applicable to individual sheep within a flock, may lead to an error known as the ecological or aggregation fallacy (Waltner Toews, 1985). Also, the cluster variable can be modelled as a fixed effect. This reduces the power of a study by (n-1) dummy variables from n clusters. It provides information on each element of the cluster e.g. in this study how flock A differs from flock B, but fails to provide any information on the overall variation attributable to the grouping i.e. the variation between flocks.

Using multi-level modelling avoided all of the above compromises and also enabled the unbalanced repeated measures data to be analysed. Standard techniques for handling repeated measures data, such as MANOVA (Crowder & Hand, 1990), rely on a balanced design (that is, equal numbers of repeated measures for each subject) or the subject is excluded. Balanced designs in an observational study are difficult to achieve, in this study lambs were missed for a variety of reasons (Green at al, 1994) and also died or lost their unique identifying number positioned in an ear tag. When a lamb's examination was missed, only the occasion of the missing examination, not the entire lamb data, were omitted from the analysis. This made efficient use of the data collected whether a lamb was examined once or on nineteen occasions.

The multi-level analysis indicated that an episode of diarrhoea does reduce the weight of lambs. The weight of lambs is also dependent upon their age, sex, the size of the litter and the flock of origin. Furthermore, there is significant complex random variation between litters, lambs and examinations which can be explained by biologically plausible, but unmeasured exposures.

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META-ANALYTIC REVIEW OF ELISA TESTS FOR THE DIAGNOSIS OF HUMAN AND PORCINE TRICHINELLOSIS: WHICH FACTORS ARE INVOLVED IN DIAGNOSTIC ACCURACY?

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Human trichinellosis in Europe in the past forty years has been closely associated with the consumption of raw or undercooked meat of wild animals (mainly wild boar), horses and pigs infected with the tissue nematode *Trichinella spp.* Therefore, the detection procedure of this parasite in meat at slaughter is of public health concern and, in countries of the European Union, regulated by the Directive 77/96/EEC. In Germany, the annual detection rate is up to three cases out of about 40 million pigs slaughtered per year (data for 1990-1995; Statistisches Bundesamt). The situation is similar in most European countries. With regard to the extremely low prevalence new EU regulations are anticipated that schedule the certification of '*Trichinella* free exploitation' to pig farms located in 'non-endemic areas' (no cases in the past decade, parasitological prevalence in indicator wild animals less than 0.1%, strict registration and identification system for farm pigs) under the condition of compulsory sanitary and logistic measures (Report of the working group on '*Trichinella* free areas' (non-endemic areas) at the request by the Scientific Veterinary Committee on Trichinellosis, 1996). Serological tests for *Trichinella* antibodies are to be conducted after three weeks quarantine for bought-in pigs from uncertified farms. This approach is encouraged by economic considerations since pigs from certified farms can be suspended from trichinoscopy according to Directive 64/433/EEC (§ 6.2).

Various modifications of the enzyme-linked immunosorbent assay (ELISA) for the detection of *Trichinella* antibodies in swine have been described. Moreover, a significant body of empirical knowledge exists concerning the diagnostic utility of such tests for human trichinellosis. From our previous experience with other serodiagnostic test systems, we infer that the diagnostic accuracy of *Trichinella* antibody ELISAs may also depend upon both inherent test properties and a set of biological and technical influencing factors (Greiner and

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Böhning, 1994; Greiner et al., 1997). Reliable estimates of the diagnostic performance criteria (sensitivity, specificity, predictive values) will be required for balancing public health and economic interests.

Based on a systematic literature review we use meta-analytic and multivariate methods with the objective to identify influencing factors for trichinellosis serology. Various methods have been described for the meta-analysis of diagnostic tests (MADT). We have compared inferences from summary measures of test accuracy based on summary receiver operating characteristic (sROC) analysis (Hurblut et al., 1991, Moses et al., 1993), Mantel-Haenszel summary odds ratios and standardised mean differences (SMD) (Hasselblad and Hedges, 1995).

MATERIALS AND METHODS

Literature retrieval

References were retrieved from the databases Medline, VetCD, BeastCD and CAB Helminthological Abstracts using the search string "[Trichin*] and [ELISA or ENZYME-LIN* or ENZYMELIN* or ENZYME-IMMUN* or IMMUNO-ASSAY or IMMUNOASSAY or IMMUNO-DIAGN* or IMMUNODIAGN* or EIA]". From these and from additional references collected by experts in trichinellosis only those references were included for analysis that describe a trichinellosis antibody ELISA in humans and/or farm pigs and were published between 1990 and 1995. Furthermore, inclusion required that the number of true positive, false positive, false negative and true negative test results (from now on referred to as a, b, c and d, respectively) were either indicated or derivable from the published data, that the positive subpopulation was not sampled before day 10 post infection and that, in the case of repeated measurements, the time interval in days to the preceding sampling date was at least 10 days. Studies were excluded from analysis if published in languages others than English, German or French. A list of publications excluded from this study can be obtained from the authors.

Data transcription

A set of variables was derived from primary studies, based on the information provided in the original publication, YEAR (publication year), SPECIES (0=human, 1=swine), ANTIGEN (coating antigen for the ELISA; 0=crude larval antigen or extract of larval antigen, 1=excretory/secretory (E/S) or purified antigen preparations), CONJUGATE (antibody class specificity of the anti species-enzyme conjugate; 0=whole Ig fraction, 1=IgM, 2=IgG, 3=IgE), TITER (dilution regime for serum samples; 0=one-point determination, 1=titration, 2=measures derived from titration curves), AVERAGE (whether or not sera were tested in duplicates; 0=no, 1=yes), STANDARD (whether or not internal standards were tested along with the samples; 0=no, 1=yes, 2=yes/used in a formula to express test results), SEW (selected cut-off optimises sensitivity rather than specificity; 0=no, 1=yes)[§], SPW (selected cut-off optimises specificity rather than sensitivity; 0=no, 1=yes)[§], DPI (sampling date for the positive control group in days

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[§] E.g., SEW=1 or SPW=1 was assumed if the cut-off value was established as confidence limit of only the positive or the negative reference population, respectively.

post infection), NPOS and NNEG (sample sizes of the positive and negative subpopulations, respectively), STATPOS (status of the positive subpopulation; 0=experimental infection, 1=arbitrary or convenience sample, 2=cases "difficult-to-classify", 3=combined strata with different characteristics or pooled reference groups, 4=representative sample of the target population), STATNEG (status of the negative subpopulation; 0=healthy controls, samples from a non-target population or unrelated diseases, e.g., atopic conditions, 1=other helminthic infections, 2=combined strata with different characteristics or pooled reference groups, 3=representative sample of the target population), RESUBST (whether or not the cut-off optimisation and evaluation was done using identical reference samples; 0=no, 1=yes). Each of the possible combinations of an estimate of sensitivity and specificity for one published test within one publication was considered as an analytical unit. For each study unit the empirical frequencies a, b, c and d were recorded.

Meta-analytic summary measures and definition of residuals

Summary receiver operating characteristic analysis (sROC): If – theoretically – the variation of sensitivity (Se) and specificity (Sp) between primary evaluation studies is only due to different cut-off values the ROC function (Eq. 1)

$$TPR=1/[1+\exp(-A/(1-B))((1-FPR)/FPR)^{(1+B)/(1-B)}], \qquad (1)$$

holds, where TPR=(a+0.5)/(a+c+1)≅Se and FPR=(b+0.5)/(b+d+1)≅(1-Sp)[§]. The parameters A and B can be estimated using unweighted (UW) and weighted least square linear regression $D_i=A+BS_i$ (Moses et al., 1993) with $D_i=\text{logit}(TPR_i)-\text{logit}(FPR_i)$ and $S_i=\text{logit}(TPR_i)+\text{logit}(FPR_i)$. Since D is equivalent to the logarithm of the odds ratio (ad/bc) and var(ln OR)=1/(a+0.5)+1/(b+0.5)+1/(c+0.5)+1/(d+0.5) (Hasselblad and Hedges, 1995) we can use the inverse variance (IV) (Irwig et al., 1994) or, alternatively, the square root of the total sample sizes (a+b+c+d) for each analytical unit (SS) as weights. A further weight is based on the inflation factor (IF) which is the inverse of the number of possible combinations of Se/Sp estimates per publication and test. Using the empirical values for TPR and FPR and the expected values TPR' and FPR' based on Eq. (1) we define residuals in direction of TPR and FPR (DSE=TPR-TPR' and DSP=FPR-FPR', respectively) and the summary measure DY=DSE+DSP on the analogy of Youden's index Y=Se+Sp-1. According to the weighting of the linear regression we obtain the residuals

$DYUW_i=DSE_{UW}+DSP_{UW}$,

$DYIV_i=DSE_{IV}+DSP_{IV}$,

$DYSS_i=DSE_{SS}+DSP_{SS}$,

$DYIF_i=DSE_{IF}+DSP_{IF}$.

[§] TPR=true positive rate, FPR=false positive rate. Note that the ½ correction is necessary to avoid zero values but leads to a slight underestimation of Se and Sp. Furthermore, we have excluded units with Se<0.5 or Sp<0.5 from sROC analysis (for justification see Moses et al., 1993). It was argued that both biases operate in opposite direction and therefore were tolerable.

Mantel-Haenszel odds ratios: From the 2x2 tables for each study unit we establish $OR_i=[TPR_i/(1-TPR_i)][(1-FPR_i)/FPR_i]$ which can be combined (we treat study units as strata of the Mantel-Haenszel procedure) as $ORMH=[\Sigma a_i d_i/(a_i+b_i+c_i+d_i)]/[\Sigma b_i c_i/(a_i+b_i+c_i+d_i)]$. The residuals are then given as

$$DOR_i=OR_i-ORMH.$$

Standardised mean difference (SMD): The standardised difference ($SMD=|\mu_1-\mu_0|/\sigma$; where μ_0, μ_1 and σ denote the means of the negative and positive reference population and the common standard deviation, respectively) reflects the degree to which a quantitative diagnostic test separates the readings for the negative and positive reference population. Hasselblad and Hedges (1995) have shown that SMD can be estimated using the Se and Sp of a test as $SMD_i\cong\sqrt{3}/\pi[logit(Se_i)+logit(Sp_i)]=\sqrt{3}/\pi[log(OR_i)]$. They have proposed the summary measure $SMD=\Sigma SMD_i w_i/\Sigma w_i$, where $w_i=1/var(SMD_i)=\pi^2/3[1/a'_i+1/b'_i+1/c'_i+1/d'_i]^{-1}$. The deviations of individual units to the summary measure can be expressed as

$$DSMD_i=SMD_i-SMD.$$

Multivariate analysis

The above mentioned residuals **R**={DYUW, DYIV, DYSS, DYIF, DOR, DSMD} can be regarded as dependent variables to study the impact of the explanatory factors X_1, X_2, X_3, ..., X_m. We use the z-transforms of the factors and obtain standardised coefficients for the models $\mathbf{R}=\beta_0+\beta_1X_1+\beta_2X_2+\beta_3X_3+ ...+\beta_mX_m+\varepsilon$. Alternatively, the linear model $D_i=A+BS_i+\varepsilon$ can be extended by the risk score vector $\mathbf{CZ}=\beta_1X_i+\beta_2X_2+\beta_3X_3+ ...+\beta_mX_m$. Only those variables were selected for inclusion into the multivariate models that showed an effect on at least one of the residuals **R** in a univariate analysis (Kruskal-Wallis test, $\alpha=0.1$).

RESULTS AND DISCUSSION

Characteristics of the primary studies

Twelve original publications that describe a total of 19 tests met the inclusion criteria and were used for analysis (Arriaga et al., 1995; Bruschi et al., 1990; Chan and Ko, 1990; Dzebenski et al., 1994; Gamble, 1995; Lind et al., 1991; Mahannop et al., 1992; Mahannop et al., 1995; Morakote et al., 1991; Morakote et al., 1992; Nöckler et al., 1995; Serrano et al., 1992). The total number of study units was 72 since nine studies reported more than one pair of sensitivity/specificity. Major technical variations of a given test (e.g., the detection of specific IgM, IgG and IgE antibodies using the same antigen) or evaluation using different reference populations were treated as different study units. The implication of such "multiple studies" for meta-analysis is discussed in more detail elsewhere (Greiner et al., manuscript in preparation). The distribution of characteristics among the studies included in this analysis is displayed in Fig. 1. Some of the variables may be regarded as surrogate variables. The publication year, e.g., may describe unobserved technical factors that may have been changing over the observation period.

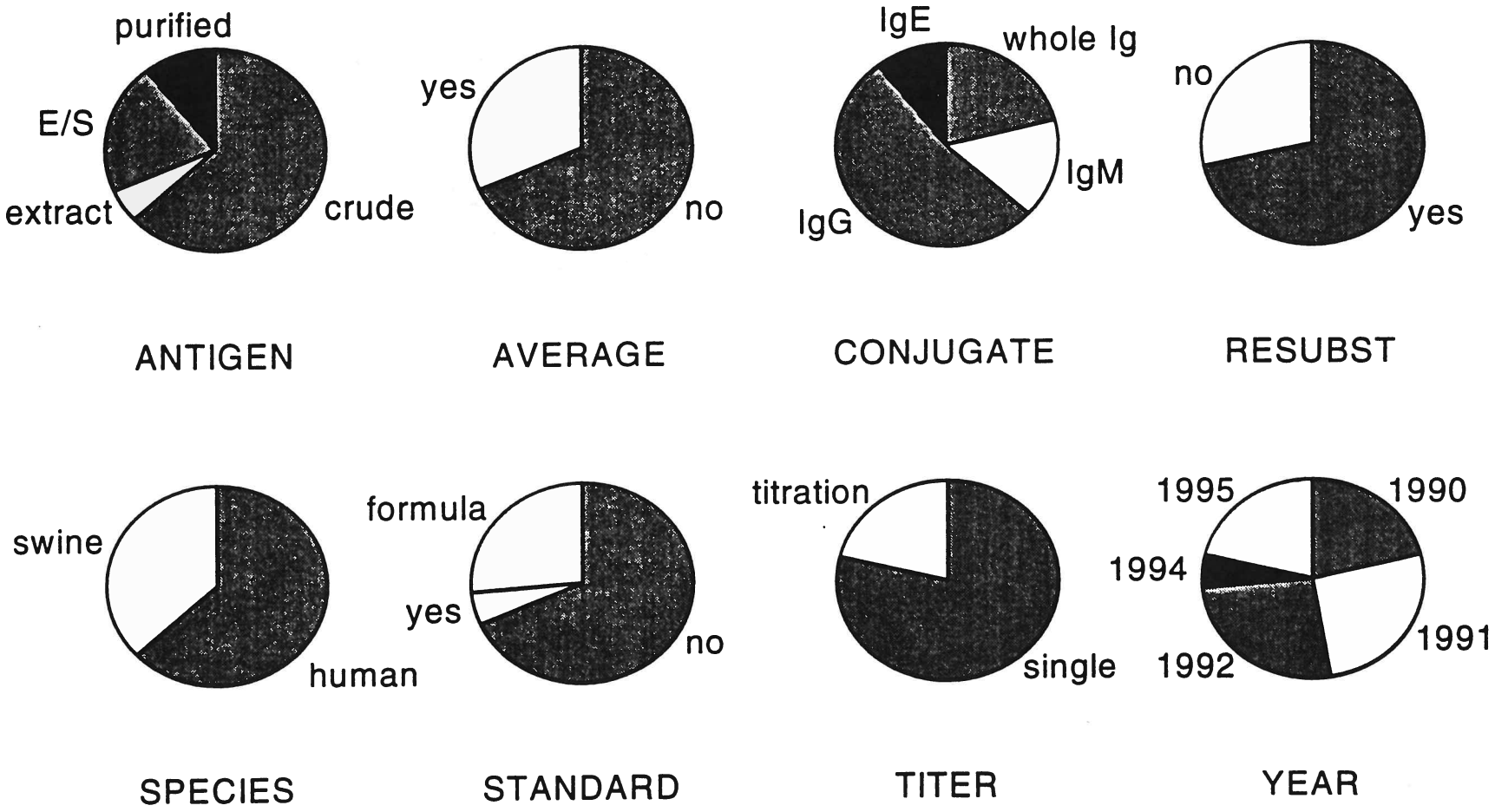


Fig. 1 Characteristics of *Trichinella* antibody ELISA evaluation studies published between 1990 and 1995 (19 tests described in 12 publications). Refer to Materials and Methods for further explanation of the variables.

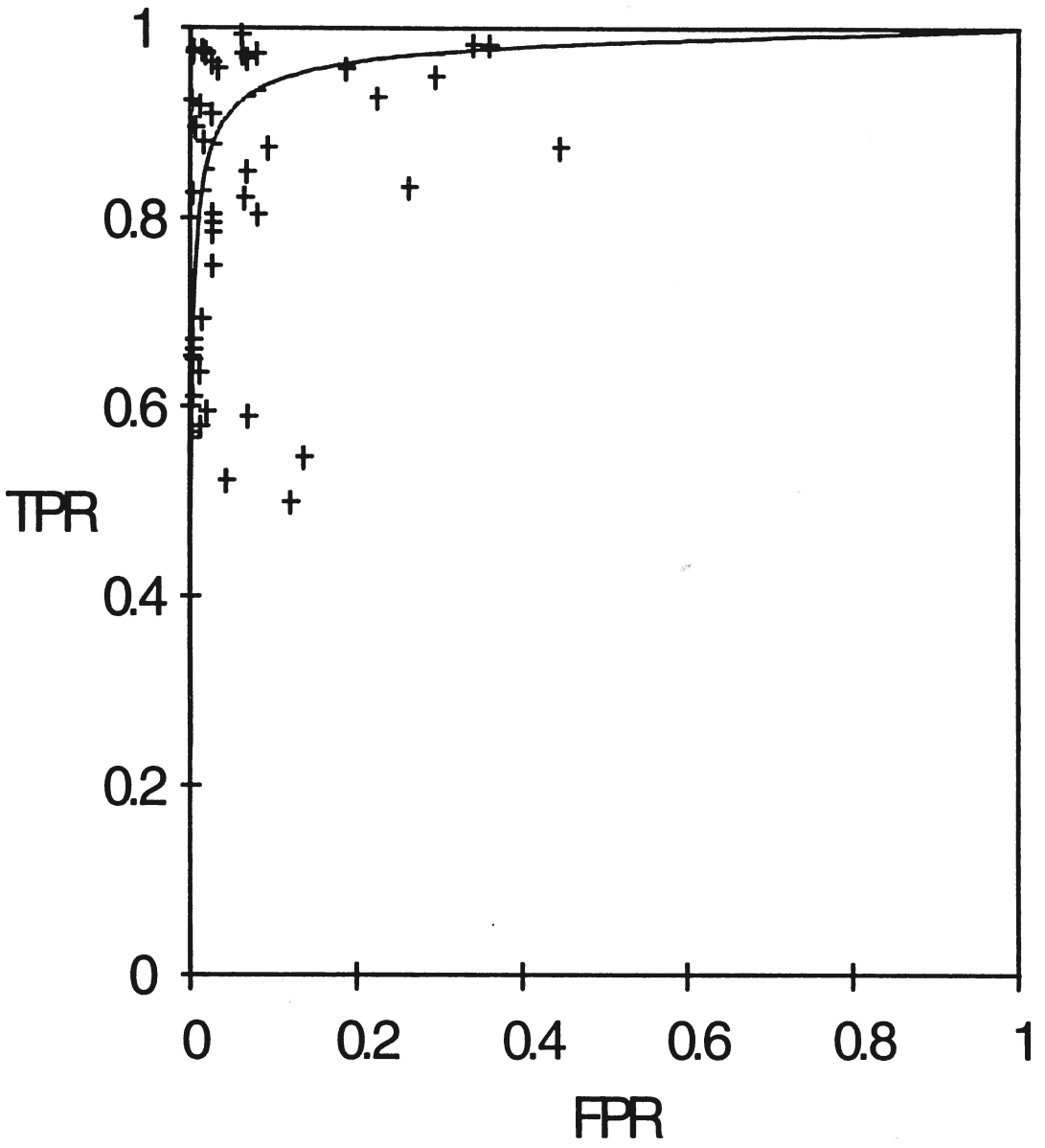


Fig. 2 Variability of the diagnostic accuracy of *Trichinella* antibody ELISA tests published between 1990 and 1995 (72 study units described in 12 publications). The measures FPR and TPR are asymptotically equivalent to (1-Sp) and Se, respectively (see text for further explanation). The graph of the sROC function is overlaid.

The variability of the diagnostic accuracy (Se, Sp) observed for the total of 72 analytical units can be seen from a plot in the ROC space (Fig. 2). This graphical representation is also the key issue of the sROC technique that aims at a summary measure for test accuracy. This summary measure can be expressed in terms of the parameters of an ROC function that is fitted to the empirical data.

Investigation of influencing factors

We have investigated the study characteristics for their impact on the residuals using explorative descriptive methods. For example, the publication year as well as the antigen and the conjugate used for the tests seemed to be important factors for the diagnostic accuracy as shown for DYUW in Fig. 3 and 4. Positive and negative values for the residuals reflect a diagnostic accuracy which is better or worse than the average, respectively. Any of the factors that revealed an effect on any of the residuals (Kruskal-Wallis test, α=0.1) was forced into multivariate models as explanatory variable for each of the residuals **R** as dependent variable.

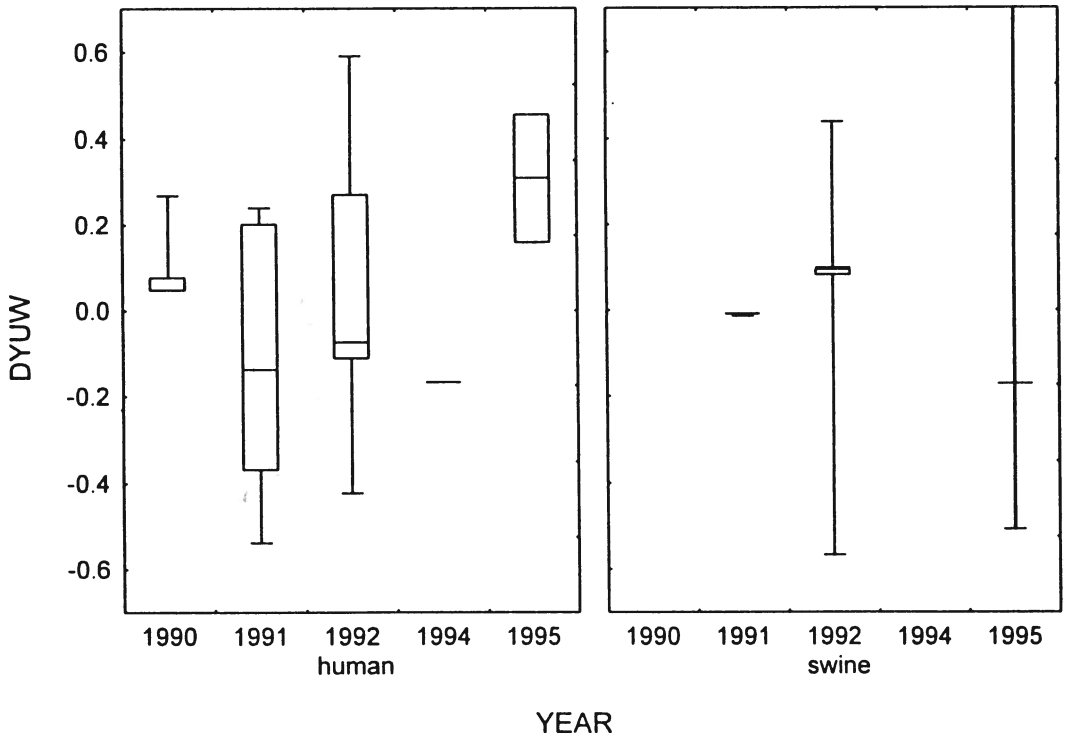
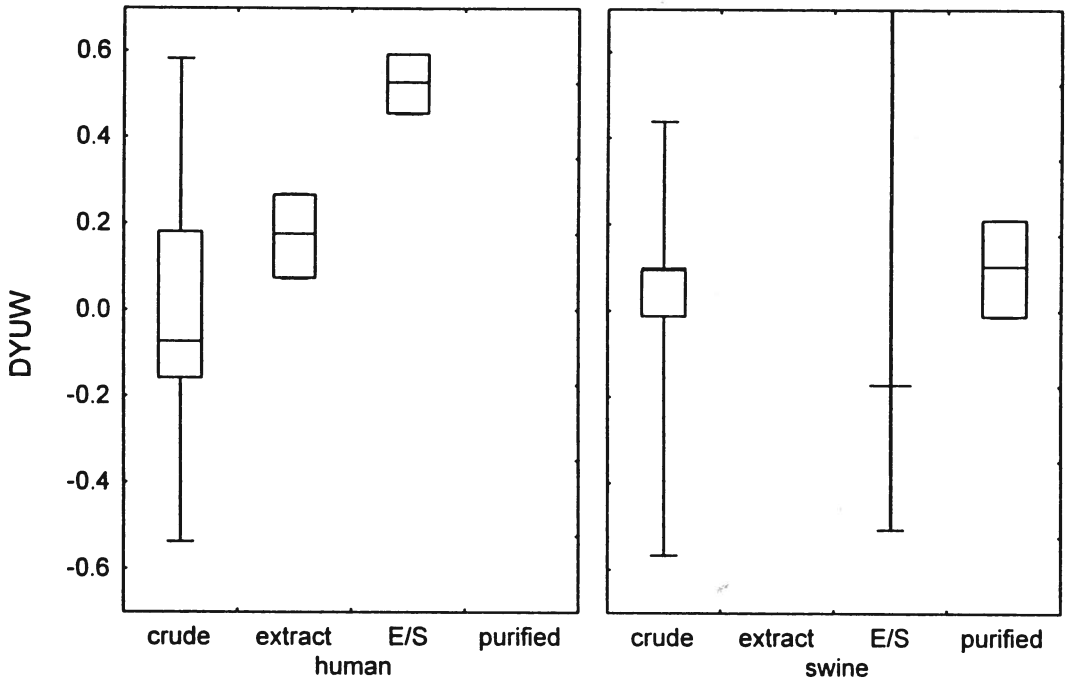


Fig. 3 Impact of the publication year on the diagnostic accuracy of *Trichinella* antibody ELISA in man and swine (based on 12 evaluation studies published between 1990 and 1995) in terms of deviation from a meta-analytic measure of test accuracy (DYUW). The five horizontal bars of the box plots represent the minimum, lower quartile, median, upper quartile and maximum value, respectively.



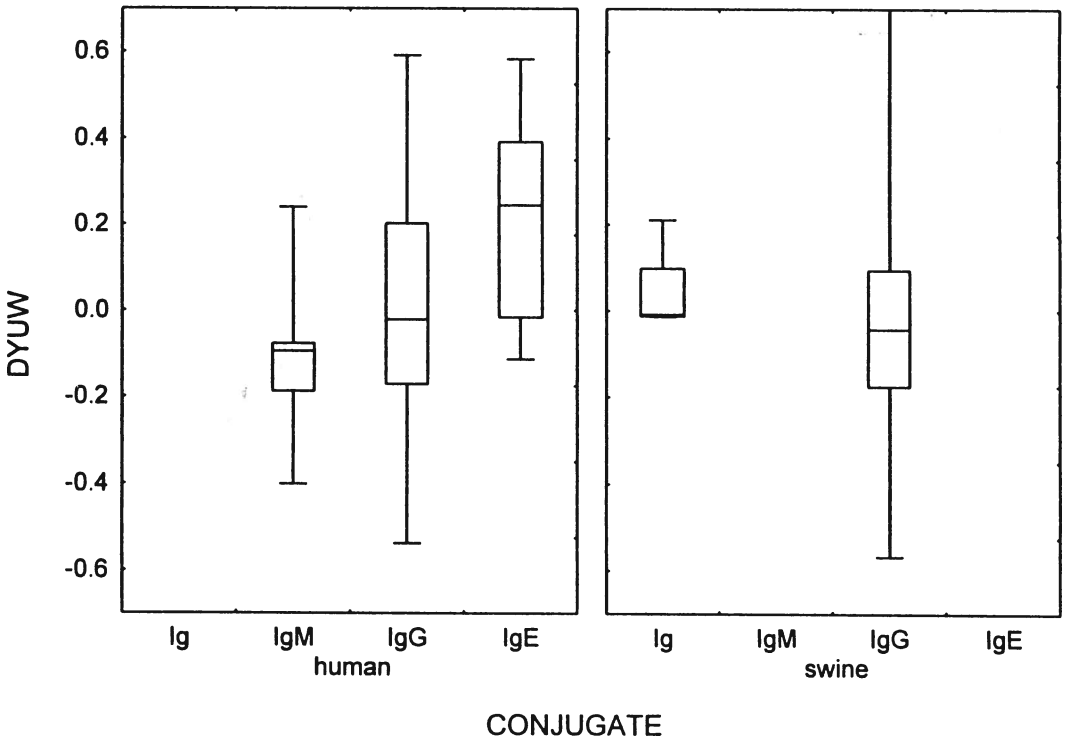


Fig. 4 Impact of the antigen and conjugate on the diagnostic accuracy of *Trichinella* antibody ELISA in man and swine (based on 12 evaluation studies published between 1990 and 1995) in terms of deviation from a meta-analytic measure of test accuracy (DYUW). The five horizontal bars of the box plots represent the minimum, lower quartile, median, upper quartile and maximum value, respectively.

Some of the study characteristics could be confirmed as influencing factors also in a multivariate analysis (Fig. 5). The size of the positive reference population (NPOS) and the time interval after infection (DPI) was positively associated with the diagnostic accuracy. Both effects were consistently significant ($p<0.05$) in all 7 multivariate models. The effect of sample size may be explained by unobserved and unknown confounding effects. The duration of infection prior to sampling is certainly a factor for the degree to which specific antibodies have been produced and, thus, a biologically plausible factor. This finding is in line with the suggestion of the working group on "*Trichinella* free areas" that serological tests of bought-in animals are to be conducted only after 3 weeks of quarantine.

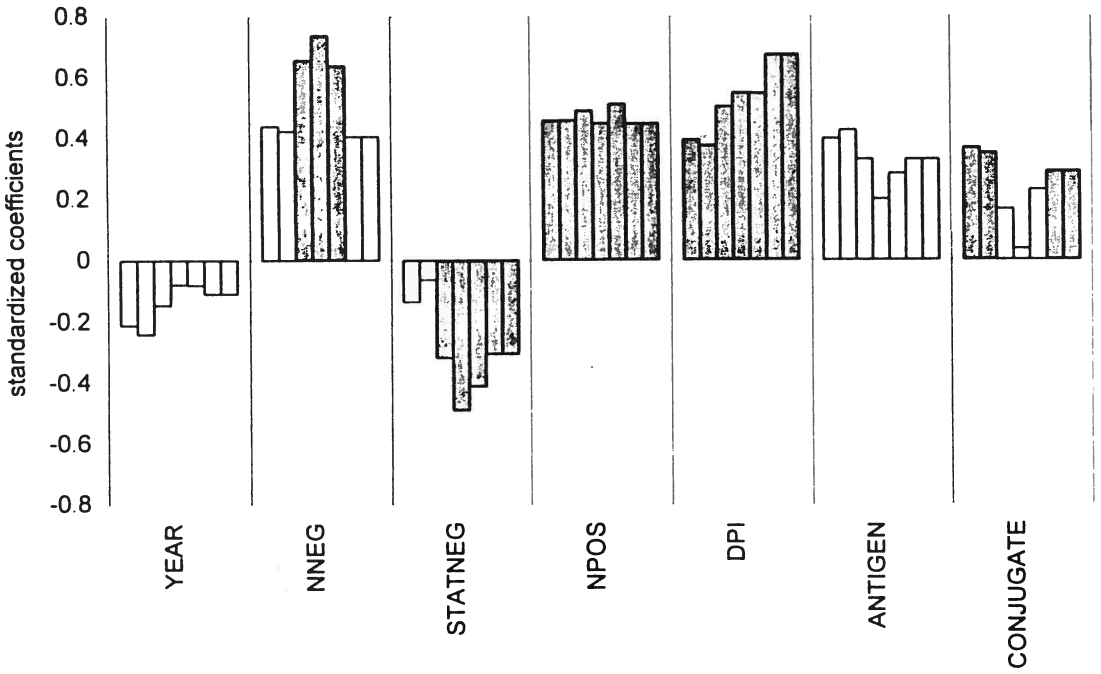


Fig. 5 Meta-analysis of *Trichinella* antibody ELISA in man and swine based on 12 evaluation studies published between 1990 and 1995. The impact of the study characteristics YEAR (of publication), NNEG and STATNEG (sample size and status of the negative reference population, respectively), DPI (days post infection), ANTIGEN (antigen preparation) and CONJUGATE (used for the test) is expressed in the form of standardised coefficients of multiple regression models where the residuals DYUW, DYIV, DYSS, DYIF, D, DOR and DSMD (shown in that order for each of the independent variables) were the dependent variables. Shaded bars represent significant ($p<0.05$) effects.

The significance of other effects was not consistent between the models. However, the direction of the coefficients for each variable was equivocal in all models. It may be assumed that some plausible factors failed to reach a significant p-value due to sample size limitations (note that the source data can be regarded as census data because all publications were considered that met the inclusion criteria). The status of the negative controls (STATNEG) might be a true factor. It is well established that the specificity of a diagnostic test depends on the selection of the negative reference population. Animals or patients with other helminthic infections are more likely to give false positive results than healthy controls. Our data partly

confirm this interpretation. We have expected that the choice of E/S or purified test antigen had a positive effect on test accuracy. This factor, however, was formally not significant. Due to sample size and other limitations associated with the meta-analytic approach we would not tempt to rule out a potential effect of the test antigen. Four of the models suggested that the detection of specific IgG and IgE antibodies is associated with a higher diagnostic accuracy than detection of IgM or all classes of antibodies which is already well recognised in laboratory practice. Two models (DSMD and DOR) gave equivocal results because they were both based on odds ratios. Further studies are required to investigate the power and bias of the proposed summary measures.

CONCLUSIONS

On the background of the anticipated application of serological *Trichinella* antibody tests in pigs, we have investigated factors that could have an impact on the diagnostic accuracy. The context of the decision making background is unfavourable given the low prevalence of trichinellosis in pigs and the high public health and economic risks associated with false negative test results. On the basis of a quantitative literature review using meta-analytic methods we could identify study design (sample size and the status of the negative reference population, time point of sampling for the positive controls) and technical features (specificity of the conjugate) as relevant factors.

Meta-analysis can be recommended as a powerful tool in the evaluation of diagnostic tests. Scarcity and heterogeneity in the source data may constitute serious limitations to the analytical techniques. However, heterogeneity, as far as it concerns factors under consideration, is of course acceptable. The identification of study design characteristics as factors for test accuracy is an important step towards the extrapolation of study results standardisation of evaluation studies.

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PUBLIC HEALTH

METHODOLOGICAL ISSUES IN A CASE-CONTROL STUDY OF BOVINE SALMONELLOSIS

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Great Britain is in the midst of an epidemic of salmonellosis in man and farm animals, mainly cattle, caused by a multiple antibiotic resistant strain of *Salmonella typhimurium* definitive type 104, which has risen to prominence in the last five years (Threlfall et al., 1994). It is now the second most common salmonella isolated from man and the most common cause of salmonellosis in cattle. This strain of salmonella has spread rapidly in the national cattle population and both beef and dairy herds have been affected. It causes relatively severe disease, the most common signs are scouring and loss of condition, and there is often a high mortality rate in both calves and adults. Another concern is that infection is difficult to treat as the strain is resistant to at least five commonly used antibiotics. It can also cause serious illness in people and although it is not yet clear what the main sources of infection are it is likely that infection can be transmitted through contaminated meat or milk. There is increasing evidence that direct contact with sick cattle can cause human disease (Wall et al., 1995).

In 1994, a MAFF funded case-control study of infection in cattle was initiated in order to identify possible sources of infection for cattle and to describe the epidemiology of infection within herds. Preliminary findings were recently published in Cattle Practice (Evans 1996). However, this paper discusses the methodological issues that arose in the design and analysis of the study. The main study findings are outlined and the effects of potential biases are considered. This is the first case-control study in GB to utilise a veterinary laboratory based control group and the validity of this reference population is discussed.

MATERIALS AND METHODS

Study population

The study or base population consisted of all cattle herds utilising the laboratory diagnostic services of veterinary investigation centres (VICs) in England and Wales (MAFF) and Scotland (SAC Veterinary Services).

Study design

Case-control methodology was employed in the study to identify risk factors associated with clinical disease due to multiple-resistant *S typhimurium* DT 104 infection in this population. Data were

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collected from May 1994 to October 1995. At each participating centre, in each month of the study, an approximately equal number of control herds as the number of identified cases were examined. Thus control herds were frequency matched to cases by location and season, thereby reducing the possible confounding effects of these variables. The study used a case to control ratio of 1:1.

Case definition

Cases were defined as clinically diseased herds with an isolation of *S typhimurium* DT 104 from a bovine animal, showing resistance to at least ampicillin (A), chloramphenicol (C), streptomycin (S), sulphonamides (Su) and tetracyclines (T). Repeat incidents reported in case herds were ignored.

Control definition

Control herds were randomly selected from all non-case cattle submissions to the veterinary laboratories. Herds with a recent history of salmonellosis were not eligible as controls.

Data collection

During the period of the study, all Group B salmonellas isolated from cattle during the course of normal diagnostic investigations were screened for multiple-resistant *Salmonella typhimurium* DT 104 by antimicrobial sensitivity testing. Herds were visited by MAFF/SOAFD veterinarians if the resistance pattern was indicative of this strain and information was gathered by questionnaire about herd exposure to possible risk factors for infection from a systematic sample of 85 % of the herds. Farms which were selected as controls were visited to obtain comparative information as soon as possible after their selection following the farmers agreement to participate in the study.

Data handling

Information was collected at both the herd and individual animal level and a number of data-sets were constructed for analysis.

1 Herd file (case and control farms) - information on exposure variables applicable to whole herd.

2 Primary case file (cases) - the first clinically affected animal was selected in each case herd (the primary case) and a file was created of exposure data specific to this animal (eg. origin of stock, feeding history, housing). Each herd had one primary case file. Calf rearers and cattle dealers were excluded due to low numbers as were the few farms where the primary case was a heifer, bull or fattening animal. Primary case files were grouped by cattle type, creating four case data-sets:- dairy cow (n = 193), suckler cow (n = 98), artificially reared calf (n = 81), suckler calf (n = 66).

3 Individual animal file (controls) - four comparison data-sets were formed of control herd data specific to the selected cattle types. Similar exclusions were used as for the primary case files. The Epi-Info RAN (x) command was used to randomly select one animal from each farm of each cattle type. Thus each control herd was able to contribute data only once per cattle type but was able to act as a control for multiple cattle types. For example, two files were produced from a beef suckler herd; one file specific to a randomly selected suckler cow and a second specific to a randomly selected suckler calf. Individual animal files were grouped by cattle type, creating four control data-sets:- dairy cow (n = 322), suckler cow (n = 217), artificially reared calf (n = 240), suckler calf (n = 133).

The resulting case and control data-sets were combined to form four case-control data-sets, each containing information at the herd level and the selected individual animal level. A fifth data-set contained herd-associated exposure data for all the case and control farms.

Data analysis

The essence of the analysis was the comparison of questionnaire information between case and control herds in the five data-sets in order to identify factors which either increased or decreased the risk of clinical disease associated with multiple-resistant *S typhimurium* DT 104.

The strategy of analysis was similar for each data-set. Initially each factor was examined on its own for association with disease (univariate analysis) using the computer package Epi-Info version 6 (Dean et al., 1994). However, factors were often related to one another so factors were examined together in order to identify those which were independently associated with disease (multi-variable analysis) by multiple logistic regression using EGRET (Anon, 1993). A statistical model was produced using a standard model building strategy (Breslow & Day, 1980) to show the influence of the most important factors on risk of disease.

Investigation of potential biases

1 Case definition
1.1 Strain type - data were analyzed excluding case herds where animals were infected with strains other than the epidemic strain R-type ACSSuT (England and Wales only).
1.2 Reporting laboratory - cases arising from reports by private laboratories were excluded as these herds may not have been regular users of State veterinary investigation centres.

2 Case census - case herds used in the analysis were compared with case herds which were not included in the study, due to staff resource limitations, using information obtained on a short report form (ZO4) completed for all cases of infection.

3 Reference population - control herds were selected from a population of herds submitting samples to veterinary laboratories for reasons other than salmonella. Data were analyzed excluding herds from the control group in each major diagnostic grouping in turn in order to investigate potential selection bias that may have arisen from differences in submission rates or from disease associations with risk factors for salmonella.

4 Misclassification - slurry samples were examined microbiologically in a small proportion of control herds in order to screen for salmonella infection.

RESULTS

There were 804 incident reports of bovine salmonellosis caused by multiple-resistant *Salmonella typhimurium* DT 104 during the study; 695 (86.4 %) herds completed a study questionnaire and 656 (94.4 %) of these fulfilled the case definition. The most common strain isolated was R-type ACSSuT. However, in England and Wales, strains with additional resistance to trimethoprim were detected in 13 % of herds and 2 % of herds were infected with strains with additional resistance to nalidixic acid.

Approximately 57 % of case herds were in England, 17 % in Wales and 26 % in Scotland. The

incidence was greatest in Scotland and the North East of England and lowest in the South of England using Agricultural Census data as denominators. Cases were more common in the summer months than the winter months and more common in dairy than beef herds when case data were similarly compared with the agricultural census.

There were 507 control herds of which 505 (99.6 %) fulfilled the control herd definition. Most (96 %) of the randomly selected control herds approached by MAFF/SOAFD agreed to participate in the study. Slurry samples were collected at 44 (9 %) of the control herd visits but salmonella was not isolated from any sample.

The results of the multi-variable analyses are summarised in tables 1 - 5. The model covariates accounted for 32 - 47 % of the variation in the dependent variable when measured by squared Pearson correlation with the predicted value as recommended by Mittlbock and Schemper (1996). Three other variables had significant effects on risk of disease in the model of herd associated risk factors but were not added to the model due to a large proportion of missing data. In summary, farms with many feral cats or where there was evidence of wild bird access to cattle feed stores were at increased risk of infection and purchasing stock directly from other farms rather than via markets or dealers reduced the risk of infection. Feral cats also significantly increased the risk of disease when added to the dairy cow model. Interactions are not reported here but are discussed in other publications.

Investigation of potential biases

1 Case definition

1.1 Strain type - 96 case herds in England and Wales were infected with strains other than R-type ACSSuT. When these were excluded from the analysis of herd associated risk factors the associations with cattle movement, maize and dead removal were no longer significant. However, all the main associations remained.

1.2 Reporting laboratory - there were 21 case reports arising from private laboratories (3.2 %) and exclusion of these cases from the models resulted in no major changes (all variables remained significant and the size of the odds ratios altered only very slightly).

2 Case census - case investigations were compared by region, herd type and size. A significant difference in the regional distribution of cases was seen but this was not surprising as only centres experiencing staff shortages reduced their rate of recruitment. A greater than average proportion of non-study cases were in S. England, E. Anglia, E. Midlands, N.W England and Scotland but all regions, except E.Anglia, included at least 80 % of cases in the study. The only difference in herd type was that study cases contained a smaller proportion of mixed herds than non-study cases reflecting the regional differences. However, there was no significant difference in herd sizes.

3 Reference population - the control herds fell into eight diagnostic categories (10 % of herds were diagnosed as suffering from diseases causing abortion, 8 % diseases of the digestive system, 5 % cases of mastitis, 3 % diseases of the respiratory system, 6 % other disorders and in 26 % of herds it was not possible to reach a diagnosis. In addition 23 % of herds submitted samples for the statutory testing of abortions for *brucella abortus* which were found to be negative and 19 % of herds submitted samples for routine monitoring or non-diagnostic testing. When each diagnostic grouping was excluded in turn from the main models only minor changes occurred, mainly within categorical variables, with no changes of significance.

4 Misclassification - slurry samples were collected at 44 (9 %) of control herd visits and salmonella was not isolated from any samples.

Table 1: Multi-variable analysis of risk factors for bovine salmonellosis due to multiple-resistant *S typhimurium* DT 104 in adult dairy cattle (n=515)

Exposure	Level of Exposure	Cases	Controls	Odds ratio[a]	95% confidence limits
Adult dairy animals purchased in last 3 months	Yes	53	50	2.79	1.60-4.88
Period of herd calving season	All year calving	29	51	1.00	-
	Spring/Summer	20	41	0.37	0.15-0.96
	Autumn/Winter	139	235	1.27	0.60-2.67
MAFF/SOAFD visit in calving season	Yes	92	161	2.40	1.28-4.51
Feeding of purchased dairy concentrate	Yes	145	285	0.16	0.08-0.33
Feeding of home produced wheat	Yes	9	36	0.31	0.12-0.81
Feeding of purchased maize	Yes	10	43	0.20	0.07-0.53
Feeding of purchased root crops	Yes	16	63	0.46	0.22-0.96
Home produced feed stored in silo	Yes	31	103	0.35	0.19-0.65
Sheep on farm	Yes	83	186	0.49	0.31-0.78
Disinfectant bootdip used	Yes	27	8	6.39	2.48-16.5
Visits by other farmers in last 3 months	Yes	85	216	0.39	0.24-0.62
Horse farm within 2 mile proximity	Yes	109	232	0.52	0.32-0.83

[a] Adjusted for the effect of all other variables in the model and season of herd visit, geographical location of herd and herd size.

Table 2: Multi-variable analysis of risk factors for bovine salmonellosis due to multiple-resistant *S typhimurium* DT 104 in adult suckler cows (n=266)

Exposure	Level of Exposure	Cases	Controls	Odds ratio[a]	95% confidence limits
Origin of stock	Homebred	6	43	1.00	-
	Homebred/purchased	37	110	1.53	0.52-4.51
	Purchased	33	37	5.03	1.58-16.0
MAFF/SOAFD visit in calving season	Yes	59	103	3.27	1.52-7.06
Feeding of minerals	Yes	21	23	2.80	1.19-6.57
Home produced feeds stored on floor	Yes	28	44	2.09	1.00-4.35
Disinfectant bootdip used	Yes	12	8	6.30	1.77-22.4
Visits by other farmers in last 3 months	Yes	38	131	0.31	0.15-0.65
Visits by AI technician in last 3 months	Yes	4	59	0.14	0.05-0.46
Public access to farm (bridleways, footpaths etc)	Yes	40	134	0.44	0.22-0.89
Sewage plant within a 2 mile proximity	Yes	18	77	0.43	0.20-0.92

[a] Adjusted for the effect of all other variables in the model and season of herd visit, and herd size.

Table 3: Multi-variable analysis of risk factors for bovine salmonellosis due to multiple-resistant *S typhimurium* DT 104 in artifically reared calves (n=289)

Exposure	Level of Exposure	Cases	Controls	Odds ratio[a]	95% confidence limits
Purchase of artificially reared calves in last 3 months	Yes	10	10	3.66	1.02-13.1
Time since most recent cattle purchase	Closed herd	13	56		
	> 90 days	26	116		
	70-90 days	4	11		
	56-69 days	1	8		
	42-55 days	1	12	(1.22)b	(1.03-1.44)b
	28-41 days	0	9		
	14-27 days	6	9		
	<14 days	11	6		
MAFF/SOAFD visit in calving season	Yes	42	120	2.61	1.11-6.11
Adult isolation facilities	Yes	43	191	0.34	0.15-0.78
Disinfectant bootdip used	Yes	13	6	7.92	2.34-26.9
Milking parlour disinfected	Yes	16	31	3.47	1.36-8.85
Visits for dead disposal in last 3 months	Yes	36	172	0.33	0.15-0.78
Equipment shared with other farms	Yes	18	120	0.27	0.12-0.60

[a] Adjusted for the effect of all other variables in the model and season of herd visit, geographical location of herd and herd size.

[b] Modelled as a trend over the categories coded as 0-7.

Table 4: Multi-variable analysis of risk factors for bovine salmonellosis due to multiple-resistant *S. typhimurium* DT 104 in suckler calves (n=171)

Exposure	Level of Exposure	Cases	Controls	Odds ratio[a]	95% confidence limits
Cattle types purchased in last 3 months	None	11	54	1.00	-
	Adults	4	7	1.77	0.20-15.7
	Youngsters	3	10	0.66	0.09-5.04
	Calves	21	25	10.3	2.93-36.1
	Mixed	8	28	1.86	0.49-7.14
MAFF/SOAFD visit in calving season	Yes	40	69	8.70	2.51-30.1
Cattle location	Pasture	20	83	1.00	-
	Housed	27	41	4.91	1.52-15.9
Visitor hygiene	Good	16	9	12.6	3.12-50.6
Visits by other farmers in last 3 months	Yes	22	88	0.22	0.08-0.63
Sewage plant within a 2 mile proximity	Yes	9	52	0.11	0.03-0.40

[a] Adjusted for the effect of all other variables in the model and season of herd visit, and herd size.

Table 5: Multi-variable analysis of herd - associated risk factors for bovine salmonellosis due to multiple-resistant *S. typhimurium* DT 104 (n=819)

Exposure	Level of Exposure	Cases	Controls	Odds ratio[a]	95% confidence limits
Herd Type	Dairy	178	264	1.00	---
	Suckler	123	132	0.81	0.49-1.36
	Mixed	43	42	1.52	0.85-2.73
	Calf rearer	21	7	3.17	0.98-10.3
	Dealer ± other	8	1	14.3	1.14-178

Cont...

Table 5: continued

	Closed herd	60	87	1.00	---
Time since	>90 days	133	201	1.12	0.68-1.84
most recent	70-90 days	18	25	1.66	0.73-3.77
cattle purchase	56-69 days	16	18	0.94	0.35-2.51
	42-55 days	19	29	0.65	0.27-1.58
	28-41 days	25	24	1.33	0.59-2.99
	14-27 days	30	23	2.38	1.07-5.29
	<14 days	72	39	2.58	1.36-4.92
Temporary movement of cattle in last 3 months	Yes	43	93	0.50	0.31-0.80
Cattle location	Pasture only	30	56	1.00	---
on farm	Housed/pasture	227	222	3.64	1.81-7.32
	Housed only	116	168	2.79	1.24-6.29
Recent use of location by other species	Yes	96	148	0.56	0.36-0.86
Mains drinking water for cattle	Yes	200	197	2.01	1.37-2.94
Purchased maize fed	Yes	5	20	0.14	0.04-0.57
Purchased roots fed	Yes	43	100	0.51	0.31-0.83
Disinfectant bootdip used	Yes	66	15	8.93	4.24-18.8
MAFF/SOAFD visit in calving season	Yes	235	236	2.48	1.66-3.69
Area for difficult calvings same as for ill cattle	Yes	203	238	1.51	1.06-2.16
Equipment shared with other farms	Yes	125	215	0.60	0.42-0.87
Cattle farm within 2 mile proximity	Yes	356	441	0.23	0.06-0.82
Public access to farm	Yes	229	333	0.48	0.32-0.72
Farm visitors (last 3 mths)					
Other farmers	Yes	186	304	0.54	0.38-0.78
Feed delivery	Yes	323	421	0.34	0.17-0.68
AI personnel	Yes	136	204	0.60	0.40-0.89
Dead disposal	Yes	192	278	0.60	0.41-0.89

[a]Adjusted for the effect of all other variables in the model and month of herd visit, geographical location of herd and herd size.

DISCUSSION

This study examined the influence of a number of farm management factors on risk of clinical disease in cattle herds due to multiple-resistant *S typhimurium* DT 104. Management factors were often correlated and it is likely that the epidemiology of this infection is complex with many potential routes of infection. For these reasons, multi-variable analyses were necessary to identify the factors which were independently associated with the risk of disease.

The multi-variable models were adjusted for the effects of the main confounding variables; geographical location, season (or month) of visit and herd size. Regional and seasonal variations in incidence were masked by the study design. However, when cases were compared to the agricultural census, disease was most common in the North of England and Scotland. Clinical disease was more common in the summer than the winter and this reflects the seasonality of reported human cases.

The main risk factors for disease that were identified by this study are summarised below;

1 There was no apparent difference in risk between dairy and beef herds but cattle dealers, although only accounting for a small number of cases, were at increased risk of disease.

2 The introduction of cattle to the farm increased the risk of disease and the period of high risk was the first 4 weeks after purchase. The primary case usually occurred in a recently purchased animal. Purchase of stock from dealers increased the risk compared to purchasing from other farms.

3 There was an increased risk of disease occurring when cattle were housed, possibly indicating that persistently contaminated buildings may be a source of infection or that close confinement or stress when housed increased the risk of clinical disease.

4 A lack of isolation facilities for ill animals increased the risk of disease, providing further evidence that the environment is a source of infection or indicating a poor general approach to hygiene and disease control on these farms.

5 Wild birds and feral cats were shown to be possible vectors of this infection, particularly if they have access to cattle feed stores. Chronic carriage of multiple-resistant *S typhimurium* DT 104 has been previously reported in a cat (Wall et al., 1995) and wild birds may also harbour salmonella. The presence of other species of wildlife on farms were not associated with disease.

6 Cases were more common during the herds' calving period which may be due to impairment of liver activities at calving altering the balance of the intestinal flora in favour of salmonella growth.

7 The feeding of maize, wheat, root crops and dairy concentrate reduced the risk of disease in adult dairy cows. The reasons for this are unknown but may be related to differences in susceptibility to salmonella due to diet or nutritional stress.

8 Storing home produced feed in a silo reduced the risk whereas storage of feed on the floor increased the risk of disease. This may be related to differences in ease of access of wildlife to feed and resulting contamination of feed stuffs.

Cattle contact with other species of farm livestock did not increase the risk of disease and there was no indication that feed stuffs were a source of *S typhimurium* DT 104. The application of animal or human waste products to farm land was not associated with disease.

These findings are discussed in detail in other papers (Evans, 1996, Evans & Sayers - in press) and the present discussion centres on some of the methodological issues involved.

Five statistical models of infection were produced. Some risk factors were common to most or all cattle types (recent cattle purchase, calving association, use of boot dip) but some appeared to be unique (eg. dairy cow feed associations). It was considered appropriate to present results for each cattle type separately. There was no indication that major risk factors differed between strains.

The study identified risk factors associated with clinical salmonellosis. Stress was a contributing factor, thus some herds may have been asymptomatically infected for some time prior to the outbreak. Therefore, the source of infection may not have been detectable by the study as it assessed only the current and recent history of the herd. This potential bias might result in a dilution of the strength of detected associations. It seems likely that most case herds were recently infected as few herds (2.4 %) reported a recent history of salmonellosis and studies of outbreaks have shown that infection peaks 14 - 21 days after exposure then declines (Wray et al., 1987). Management factors identified in the analysis are a mixture of those responsible for the introduction of salmonella infection and those triggering clinical disease and models should be interpreted accordingly.

There were a number of implausible findings produced by this study. These were; the association with drinking water source, shared farm equipment, proximity to other farms and sewage plants, public access to farmland and farm visitors. A case control study examining a large number of factors is likely to produce a small number of significant associations with the outcome purely by chance. Residual confounding in the study or the identified factor being correlated with another management factor which has a true effect on the risk of disease but which was not measured by this study might also have been responsible for these findings. Unexplained associations may also be due to bias in the study. Cause effect reversal, where the disease leads to the exposure rather than vice versa, is likely to have accounted for the apparent increased risk of disease on farms using a disinfectant boot dip outside cattle buildings, applying high standards of visitor hygiene or milking parlour disinfection.

A moderate level of undetected salmonella infection in the control herds might result in bias in the study due to the misclassification of herd infection status. Therefore, a proportion of the control herds were screened for salmonella in order to assess the prevalence of infection. The method of screening was not highly sensitive. However, salmonella was not detected in any of the screened herds and other studies have found that the prevalence of asymptomatic herds is only about 1 % (Robinson & Bender, 1994). This level of misclassification is unlikely to affect the study results. In addition, the results of this study relate to clinically affected herds and it is unlikely that any control herds were clinically affected at the time of the MAFF/SOAFD visit.

The case-control study was an appropriate and rapid method to investigate risk factors for bovine salmonellosis. However, valid selection of subjects is always a concern. If cases are defined as those appearing in VICs during the study period, the definition of the base or reference population is secondary to the case selection and the challenge is to ensure that the case series and base sample are representative of the same population. The base must be the population in which each potential case, had it occurred, would have been included in the case series ie. the VICs catchment population. The base sample used in this study appeared to be valid as there was no indication of selection bias. A laboratory based study population was chosen for reasons of efficiency. The generalizability of the results has not been assessed but it is likely that most cattle herds, excluding small-holdings with only a few animals, regularly submit material to VICs. This is indicated by the fact that only 3 % of salmonella incidents were reported by private laboratories. However, it is recommended that a formal assessment of the VICs catchment population is undertaken as this population forms an efficient and convenient base for large scale epidemiological investigations. In addition, a very good response rate was achieved in this study which is vital to the validity of a study.

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HUMAN AND BOVINE TUBERCULOSIS IN THE HIV ERA

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ABSTRACT

Tuberculosis (TB) kills more than three million people annually. Human immuno-deficiency virus (HIV) is a potent trigger for progression from mycobacterial infection to clinical TB. In many countries, the incidence of HIV and TB is increasing.

The zoonotic potential of cattle infected with *Mycobacterium bovis* is well established. A person infected with *M.bovis* may transmit the organism to bovine and human contacts. In areas with a high prevalence of HIV infection and close contact between human and bovine populations, the risk of zoonotic transmission should be quantified to indicate whether specific control measures are required.

INTRODUCTION

The success of the veterinary profession in bringing about the control of TB in cattle in the USA was celebrated over fifty years ago as "Mans Greatest Victory Over Tuberculosis"(Myers 1941). In the last century, TB was the cause of one quarter of all human deaths in Northern Europe and the USA (Snider 1994). Up to 4% of pulmonary TB and 20% of extra-pulmonary TB was attributed to *M. bovis* of bovine origin, although this varied markedly both between and within countries. For example, in Denmark 50%-60% of pulmonary TB in rural areas was caused by *M. bovis* (Sigurrdson 1945). Outbreaks of *M. bovis* due to infection via unpasteurised, raw milk were common - in one outbreak in Sweden, 50 children had clinical sign of TB and a further 134 were tuberculin positive after drinking milk from one dairy of 22 cows. One of these cows was excreting tubercle bacilli in her milk but was clinically normal and three other cows had signs of pulmonary TB (Stahl 1937). Alleviation of poor housing, sanatoria, the advent of effective chemotherapy and widespread application of the BCG vaccine as well as the introduction of pasteurisation and cattle TB control programmes combined to reduce the impact of TB in economically favoured countries to current levels of approximately 40,000 deaths in 1990 (Kochi 1991). From an industrialised world viewpoint, therefore, it might seem that TB is a success story - a feared disease now conquered.

However, in most of the world, excluding Europe, North America, Australia, New Zealand and Japan, TB remains a devastating human disease. It was estimated that there were seven million

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new cases of TB and three million deaths in 1990 (Murray et al 1990) - this is approximately 7%of all deaths and 25% of all avoidable deaths in these areas. Although the largest number of TB cases were estimated to occur in Asia, the highest case rate was found in Sub-Saharan Africa (250 new cases per 100,000people per annum). Furthermore, the majority of these deaths (75%) are concentrated in the economically most active age group (15-59 years). Consequently, the social costs of this disease are severe. By 2000, it has been predicted that there may be in excess of ten million cases per year (Kochi 1994) and that the total number of TB deaths during the 1990's will have reached thirty million (Raviglione et al 1995). This increasing incidence of TB is associated with demographic and socio-economic factors, population movement, increasing drug resistance and the Human Immuno-deficiency Virus (HIV) epidemic. HIV infection increases susceptibility to infection with TB, increases the risk of progression from infection to disease and worsens the clinical prognosis for dually infected patients. In a cruel synergy, TB infection hastens the progression from HIV infection to clinical AIDS. Between 20%-67% of TB patients in Sub-Saharan Africa are also infected with HIV (De Cock et al 1992).

Although there is no doubt that most human TB cases are due to infection with *Mycobacterium tuberculosis* and most new infections follow contact with such cases, the possible role of *M. bovis* and cattle deserves consideration. In addition, the exposure of cattle to TB of human origin may have an impact from a veterinary viewpoint. In the following sections, the microbiology and epidemiology of TB in humans and cattle will be summarised. The relatively scanty information available from less developed nations will be outlined and the public health significance of *M. bovis* in these countries will be discussed.

MICROBIOLOGY

M. tuberculosis and *M. bovis* have been described as separate species (Karlson & Lessel 1970) but they are very closely related (Tsukamura 1976, Imaeda 1985) and so both may better be regarded as members of the *M. tuberculosis* complex, together with *M. africanum, M. bovis BCG* and *M.microti* (Grange & Yates 1994). Differentiation can be based on the criteria shown in the table below:

Species	Variant	TCH	NO_3	O_2	PZA	NIACIN
M. tuberculosis	Classical human	R	+	+++	S	+
	Asian Human	S	+	+++	S	+
M. africanum	Type I	S	-	+	S	+/-
	Type II	S	+	+	S	+/-
M. bovis	Classical bovine	S	-	+	R	-
	BCG	S	-	+++	R	-

TCH = susceptiblility to thiophene-2-carboxylic acid hydrazide; R = resistant, S = sensitive
NO_3 = nitratase activity, + = yes, - = no
O_2 = oxygen preference; +++ = acrobic, + = microaerophilic
PZA = pyrazinamide; S = susceptible; R = resistant
Niacin = ability to synthesise niacin; + = yes, +/- = variable, - = no

In addition, the growth of *M. tuberculosis* is enhanced by glycerol which suppresses *M. bovis* The growth of the latter is promoted by the addition of pyruvate to the culture medium. Since many medical laboratories as a routine use only media that contain glycerol and use aerobic conditions to isolate mycobacteria, there is a risk that *M. bovis* may not be isolated even if it were present in submitted material.

Epidemiologically, it is useful not only to know the species of mycobacteria involved in cases but also whether the same isolate is implicated in any particular series. Mycobacteria can also be identified by "genetic fingerprinting". A short sequence within the genome of *M. tuberculosis*, known as an insertion element, occurs in varying positions and numbers (Zainuddin & Dale 1989). When the mycobacterial DNA is cleaved by a restriction endonuclease, charged fragments of different lengths are created which can be separated by electrophoresis and visualised as a pattern of bands on a membrane. This technique is known as Restriction Fragment Length Polymorphism analysis (RFLP). The pattern, or fingerprint, of an individual isolate is relatively stable (Hermans et al 1990). The insertion element IS*6110* (also known as IS*986*) has been successfully used in epidemiological studies of *M. tuberculosis* infection in man in developed and developing countries (Godfrey-Faussett & Stoker 1992). Although multiple copies of IS*6110* are reportedly present in isolates of *M. bovis* from goats (Gutierrez et al 1995) and other non-bovine species (van Soolingen et al 1994), only a single copy at one site has been found amongst bovine isolates of *M. bovis* , which has hindered epidemiological studies of bovine TB. However, by combining probes for IS*6110* , IS*1081* and another, highly reiterated DNA element (pTBN12) 26 distinct RFLP types of *M. bovis* could be distinguished among 109 isolates from Northern Ireland (Skuce et al 1994). A similar study in Argentina used different probes to identify 30 different RFLP types of *M. bovis* (Romano et al 1996). These techniques promise to lead to new understanding of the transmission of mycobacteria between and within different species of mammals.

Detection of species specific DNA by polymerase chain reaction (PCR) technology also offers improved capability for diagnosis of infection and detection of low numbers of mycobacteria in materials such as milk and sputa. PCR has also been used to differentiate between *M. bovis* and *M. tuberculosis* (Rodriguez et al 1996, Herrera et al 1996).

Given the spectrum of properties which may be displayed by the members of the *M. tuberculosis* complex, the emphasis on the species involved in an infection may be of relatively little interest epidemiologically. Speculatively, there may be strains of tubercle bacilli in some situations which are particularly amenable to zoonotic transmission. RFLP analysis can provide an invaluable tool for the investigation of this hypothesis.

EPIDEMIOLOGY

M. tuberculosis is a human pathogen. Infection is usually via the respiratory route and it has been estimated that a patient with undetected pulmonary TB might infect as many as twenty susceptible contacts in two years. In many less developed countries, the majority of the adult population are infected with TB. Following infection, approximately 5% of people develop clinical disease within 5 years whilst the remainder maintain a latent infection. Five percent of latent infections become reactivated later in life (Murray et al 1990). HIV infection is associated with a ten-fold increase in the risk of progression from infection to disease. RFLP analysis has demonstrated that HIV positive patients are also susceptible to re-infection (Godfrey-Faussett & Stoker 1992). The clinical presentation of TB in HIV positive patients is often atypical (Elliot et

al 1990). However, patients with dual HIV and TB infections are capable of transmitting disease and high attack rates (30-40%) have been seen in nosocomial outbreaks (De Cock 1994). Cattle exposed to *M. tuberculosis* may develop a transient sensitivity to tuberculin (Karlson 1962). In areas such as sub-Saharan Africa where human TB is common, this may be a serious problem for the interpretation of bovine tuberculin tests. Indeed, in Kenya, it was reported that all positive tuberculin reactions in cattle could be explained by their exposure to TB-infected people (Waddington 1965). Although bovine infection is rare, it has been reported (Stenius 1938, Sigurrdson 1945, Lesslie 1960). In the rare instances where infection does occur, it is likely to be self-limiting and not contagious. Therefore, the greatest risk to cattle from *M. tuberculosis* is that of false positive tuberculin reactions following exposure to infected people. *M. tuberculosis* has been isolated from market milk, presumably as a result of contamination (Wehke & Berepubo 1989, Shah et al 1993).

In contrast to *M. tuberculosis*, *M. bovis* has a wide host range, encompassing many mammals (O'Reilly & Daborn 1995). Transmission within and between species is generally by the respiratory route. Oral transmission with much larger doses of the bacillus is also possible. Although *M. bovis* is frequently associated with extra-pulmonary TB in human cases, this reflects the historical risk of exposure to infected milk. *M. bovis* has been isolated from all forms of human disease and is clinically indistinguishable from disease caused by *M. tuberculosis*. In Britain prior to the control of bovine TB and the introduction of pasteurisation of milk, *M. bovis* was isolated from 20% of extra-pulmonary TB and 3% of pulmonary disease in humans - however, the greater incidence of pulmonary disease meant that numerically, most isolates were made from pulmonary cases (Francis 1947). Epidemiological studies demonstrated a strong correlation between exposure to TB infected cattle and the risk of human TB infection, but not to human disease or TB mortality (Magnus 1966, Sjogren & Sutherland 1974). This apparent paradox is explained by childhood exposure to *M. bovis* from milk, which often caused a latent infection and induced protection against later exposure to *M. tuberculosis*. This observation was used in the past as an argument against control of TB in cattle - but the immediate cost of this protective effect was a greater incidence of childhood TB, including tuberculous meningitis (Savage 1933). The protective advantage would probably be lost if a person became infected with HIV, which would predispose them to re-activation of a latent infection. Although it was generally assumed that any human infection with *M. bovis* was of bovine origin, reports exist throughout the literature which suggested human to human transmission (Collins & Grange 1983, Pritchard 1988, O'Reilly & Daborn 1995) and recent use of RFLP analysis has supported this hypothesis (van Soolingen 1994). Human infection with *M. bovis* has been transmitted to cattle, by both the respiratory route and following bovine exposure to infected urine in housing (Tice 1944, Baldwin 1968, Fourie 1952, Wilesmith 1983). Wildlife reservoirs of *M. bovis* also exist and may cause human and bovine infection (O'Reilly & Daborn 1995). RFLP analysis has demonstrated that in Argentina, most human TB cases caused by *M. bovis* are of bovine origin, whereas in The Netherlands they are from other sources (van Soolingen et al 1994). *M. bovis* has been isolated from HIV infected patients and has been transmitted between them (Houde & Dery 1988, Cornuz et al 1993, Dankner et al 1993, Albrecht et al 1995).

The risk of bovine infection with *M. bovis* is increased by various factors (Francis 1947, Szyfres 1972, Andrews & Johnstone 1988, Cook et al 1996, Griffin et al 1996, Sharma et al 1994), including the following:

- Prevalence is higher in dairy than beef systems
- Housing

- Intensification
- Increasing age
- Increasing herd size
- Congregation at eg. watering sites or in kraals overnight
- Contact with other infected herds
- Contact with other infected animals
- Breed - in particular, zebu type cattle have been reported to have a greater resistance to infection than *Bos taurus* breeds
- Poor nutrition, body condition and other forms of stress predispose to infection - but also reduce the capacity to show a positive tuberculin reaction.

Control of human TB depends upon the prompt identification of clinical cases, particularly of sputum positive pulmonary disease, and their effective treatment. This strategy has been successful and prior to the HIV epidemic, incidence rates of human TB were falling in many countries (Murray et al 1990). Implicit within this strategy is the assumption that transmission by infected individuals before they develop clinical signs is below the threshold necessary to maintain the disease. By contrast, veterinary programmes seek to remove infected cattle before they have developed clinical signs.

As the incidence of bovine TB falls, the relative importance of reservoir hosts - including human cases - increases. By analogy, the relative importance of TB of bovine origin as a source of human infection might be expected to increase as the risk of infection with *M. tuberculosis* is reduced.

DISCUSSION

In the economically developed world, bovine TB is no longer a significant public health risk. Control strategies are designed to eradicate infection with *M. bovis* in cattle - not to eliminate *M. bovis* nationally. The latter would be impractical due to the existence of other hosts for *M. bovis* , including badgers, possums, wild deer and humans. Therefore, continued surveillance is necessary. Maintenance of surveillance in the absence of final eradication represents a continuing economic cost nationally and owners of herds that are persistently re-infected or that suffer breakdowns will also incur financial losses. The emergence of *M. bovis* in farmed deer represents another area for vigilance. These challenges have helped to stimulate research into the role of wildlife reservoirs, the design of new diagnostic tests and the application of the technology of molecular genetics. Sporadic cases of human infection with *M. bovis* are still detected in the developed world, often amongst older patients, immigrants from less developed countries or following unusual exposures, eg. to wild animals or domesticated deer (O'Reilly & Daborn 1995). With the re-organisation and restructuring of the cattle industry that has followed the break up of the former USSR, there is a risk that former control measures will be prejudiced and there could be a resurgence of TB in cattle.

The situation in many less developed countries is very different. Although there may be evidence that *M. bovis* is present within a national herd, its frequency and distribution are seldom known. Compared to other major diseases - eg. rinderpest, foot and mouth disease, tick-borne diseases or internal parasitism - TB may have little impact on productivity of the cattle sector and consequently, have a low priority for hard-pressed veterinary budgets. However, losses are

incurred, as demonstrated by data on cause for abattoir condemnations eg in India, Mexico, Morocco, Ethiopia, Egypt, Tanzania, Madagascar (Anon 1994, Yehualaeshet 1995, Maity et al 1992, Sartirano et al 1993, Kambarage et al 1995). In addition to direct losses, presence of TB may preclude opportunities for trade.

As emphasised above, TB is a human disease and the human suffering which it causes is reflected in enormous economic costs both for treatment and control and through lost human productivity. In the absence of hard facts, it has been argued that there is little purpose in investigating bovine TB in less developed countries for the following reasons:

- Milk is boiled and/or processed before consumption, so the risk of transmission is negligible
- Local zebu breeds have an innate resistance to infection with TB
- The extensive nature cattle husbandry and the often unfavourable environment for survival of tubercle bacilli in the environment minimise the risk of infection
- There are relatively few reports of *M. bovis* from human TB cases in less developed countries
- Childhood exposure to *M. bovis* offers protection against exposure to *M. tuberculosis* later in life
- In comparison to the overwhelming risk of infection with *M. tuberculosis*, bovine TB is unimportant
- *M. bovis* infections are in any case susceptible to the same treatment regimes as *M. tuberculosis*
- Even if *M. bovis* were a problem, control measures would be too costly to institute

An alternative argument may be made for investigating whether *M. bovis* does have a public health significance in less developed countries today:

- *M. bovis* is present in many of these countries - but the prevalence is largely unknown in the bovine or human population
- Lack of laboratory isolates may be due to inappropriate culture techniques for *M. bovis*
- There may be an urban reporting bias, since access to suitable facilities may be limited for the rural poor
- Increased numbers of improved *Bos taurus* cattle have been introduced and many have been cross-bred with traditional breeds, which may lower innate resistance
- Many traditionally owned cattle are overstocked by Western standards and consequently suffer from poor nutrition and stress
- Personal observation of traditional milking practice in Zambia revealed that some raw milk is drunk before boiling. In addition, yoghurt culture is often continued by adding the milk obtained each day to that of the previous day. This might prevent sufficiently acid conditions developing to kill any mycobacteria that were present.
- As mentioned earlier, HIV infection could cause reactivation of *M. bovis*
- In many cities, there are increasing numbers of urban dairies, in which small numbers of cattle are housed and their milk is sold directly to the public. These environments could, if unhygienic, favour TB infection of cows and the dissemination of *M. bovis* to the local human population.

Whilst available evidence suggests that infection with *M. bovis* has only a minor role in human disease (Kleeberg 1984), this evidence is extremely limited. In many countries, particularly in Africa, routine diagnosis of pulmonary disease is confirmed only by sputum smear examination, which cannot differentiate between *M. tuberculosis* and *M. bovis*. *M. bovis* has been isolated from human cases in Argentina, Mexico, Zaire, Tanzania, Ethiopia, Nigeria, Egypt and Madagascar (Szyfres 1972, Anon 1994, Idigbe et al 1986, Menard et al 1995, Mposhy et al 1983). These various reports suggest that possibly no more than 1% of human TB in less developed nations is due to *M. bovis* - which could imply 25,000 - 30,000 human deaths each year. By analogy with experience from developed countries prior to control of bovine TB, these cases are probably not evenly distributed. Rather, they are likely to be associated with known risk factors for acquiring an infection of bovine origin, including:

- close contact with infected cattle
- consumption of milk or milk products containing unpasteurised milk or milk that has not been boiled
- occupational hazard - veterinary and agricultural staff, abattoir workers etc
- sharing an airspace with cattle
- living in a rural area.

The combination of a high risk of exposure to *M. bovis* with a high prevalence of HIV infection provides a novel epidemiological scenario worthy of investigation. Such a situation existed in the Monze district of Zambia and was the subject of a cross-sectional study in 1992-93 (Cook et al 1996). It was estimated that 10% of the population were HIV positive and it was known that 70% of TB patients in the local district hospital were concurrently HIV positive. The local tribal people, the *ba*-Tonga are traditional cattle keepers and they owned an estimated 200,000 animals - compared to a human population of 170,000. It was found that herds of households in which there had been a human TB case during the preceding 12 months were six times more likely to contain a tuberculin positive animal than herds which belonged to households without a TB case. The prevalence of tuberculin positive cattle was 7%. However, it was not possible to slaughter positive cattle, so their true status was unknown. Furthermore, no samples had been taken for culture from any of the cases encountered, so it was impossible to discover whether they had been infected with *M. bovis* or *M. tuberculosis*. If it is assumed that at least 70% of these reactions were indicative of infection with *M. bovis*, then this suggests that approximately 10,000 infected cattle were present in the area. These cattle share seasonal grazing on the Kafue river floodplain with red lechwe antelope from which *M. bovis* has been isolated. If results from developed countries with respect to increased risk of human TB as a result of exposure to infected cattle were applicable, then exposure to TB infected cattle in Monze district could be responsible for a 12% increase in the number of people infected with TB and a 1% increase in the annual risk of human tubercular infection amongst the rural population (Magnus 1966, Sjogren & Sutherland 1974). The introduction of test and slaughter schemes is probably not feasible in these areas due to expense, presence of wildlife reservoirs and cultural values. However, many people are very aware of the dangers of TB and the disease itself carries a cultural stigma in many countries (eg Liefooghe et al 1995). Therefore, they are likely to be receptive to appropriate animal and human health education messages and will wish to take an active part in protecting themselves and their cattle from the risk of TB.

Further research is needed to quantify the zoonotic importance of TB in the HIV era. This work should be a collaboration between veterinary and medical scientists. Since those areas where

risk is greatest are often also those which are under-resourced in veterinary and medical terms, specific studies are needed. Technological advances, particularly RFLP analysis and PCR methods, provide the means to detect infection and demonstrate whether or not these infections are shared by the bovine and human population.

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JOHNE'S AND CROHN'S:

INFLAMMATORY BOWEL DISEASES WITH A SIMILAR AETIOLOGY?

K.L. MORGAN*, B. CETINKAYA* AND K. EGAN**

Johne's disease or paratuberculosis is a chronic inflammatory bowel disease of ruminants first described in cattle by Johne and Frothingham (1895) in Dresden. The similarity between Johne's disease and the "regional ileitis - a clinical and pathological entity", decribed in human patients by Crohn and his colleagues (1932) was first recorded by Dalziel (1913). Dalziel, a Scottish surgeon, owned a herd of Belted Galloway cattle afflicted with Johne's disease and noted the resemblance between their lesions and those which he removed from human patients.

The chronic inflammatory reactions in both Crohn's and Johne's disease was suggestive of a mycobacterial aetiology. A small, slow growing acid-fast bacillus with fastidious growth requirements, *Mycobacterium johnei* (subsequently renamed *M. avium subsp, paratuberculosis* or *M. paratuberculosis*) was isolated from animals with Johne's disease and numerous acid fast organisms were identified in histological lesions. In contrast, attempts to isolate a mycobacteria from Crohn's disease proved unsuccessful until the early 1980's when Chiodini and colleagues (1984) isolated *M. paratuberculosis* from patients with Crohn's disease. This finding renewed interest in the possible association between Johne's disease and Crohn's disease. Specifically it posed the question, "Does *M. paratuberculosis* **cause** Crohn's disease".

As veterinarians we may find ourselves approaching this hypothesis with a scepticism born of images of histological sections of intestine stuffed with acid fast bacteria; a feature of Johne's disease but not of Crohn's disease. In an attempt to generate a more balanced view let us start by questioning the nature of this well established causal relationship between *M. paratuberculosis* and Johne's disease by re-examining the evidence on which it is based.

MYCOBACTERIUM PARATUBERCULOSIS AND JOHNE'S DISEASE

Early attempts to transmit the disease to cattle experimentally with *M paratuberculosis* proved disappointing, only 9 of 22 animals developed disease (McFadyean and Sheather 1916). Similar results were obtained with sheep and no transmission occurred in experimental animals animals (McFadyean and Sheather 1916). Later experimental studies established four important features of the relationship between *M. paratuberculosis* and Johne's disease:

- *age*: young ruminants, particularly sucking animals, could be infected easily whereas

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- *dose and route of infection:* no clinical disease developed in sheep given 10^3 organisms whereas a small proportion of those given 10^9 bacilli did; sheep infected intravenously did not develop disease, orally infected animals did;
- *long incubation period:* infection takes place in early life but disease does not develop until adulthood, usually after 18 months of age;
- *sub-clinical infection:* some animals become infected and show no clinical or pathological signs.

The presence of infection in the absence of gross lesions or clinical disease is a good starting point for the epidemiologist. If we were to examine "healthy" and diseased cattle for evidence of infection, what proportion of the controls would be infected? The answer is unknown. As is often the case with animal disease, current knowledge is based upon case and experimental studies. It is important to make this point because it is easy to point a disbelieving finger at the infected healthy patients in similar studies of Crohn's disease. They are also a feature of Johne's disease, as our most recent studies confirm (Cetinkaya et al 1996)

At the other end of the spectrum, severe chronic inflammatory lesions may occur in the intestine of adult ruminants in which *M. paratuberculosis* is either absent or present in very small numbers. This observation was first made by Stamp and Watt (1954). They examined 51 cases of ovine paratuberculosis and divided them into 4 groups. Their data are summarised in Table 1.

Table 1. The relationship between the severity of the lesions of Johne's disease and the presence of *M. paratuberculosis* (after Stamp and Watt 1954)

Group	Gross lesions		Histopathology	Bacterial culture
	Intestine	Mesenteric LN		
1	yellow thickened	oedematous	epitheliod cells large number [a]AFB	all positive
2	not yellow	oedematous	epitheliod cells lymphocytes smaller number AFB	all positive
3	no abnormality or cracking	enlarged oedematous "flecks"	giant epitheliod cells AFB in 5/21 cases	positive in 14/21 cases
4	thickened nodular	nodular	necrosis, calcification no AFB	positive in 4/9 cases

[a] AFB - Acid Fast Bacilli

So, if no acid fast bacilli are detected histopathologically or by culture are we correct in calling this Johne's disease? More recent studies of the lesions of Johne's disease have resulted in their classification into two types; tuberculoid and lepromatous. Tuberculoid lesions are characterised by small numbers of *M. paratuberculosis* (paucibacillary), focal aggregations of macrophages and many lymphoid cells whereas lepromatous lesions have a marked macrophage infiltration and numerous bacilli (pluribacillary) (Clarke 1994).

These observations serve to establish the point that although *M. paratuberculosis* is clearly involved in the causal web of Johne's disease, its precise position and influence is unclear. Almost 40 years after the disease was first described, articles were still appearing in veterinary journals questioning whether Johne's disease was infectious (Spicer 1934).

Prior to 1984 the absence of any convincing evidence of an infectious cause of Crohn's disease resulted in the development of a number of other causal hypotheses; genetic, psychogenic, dietary, immunological. These also have parallels in Johne's disease.

Familial association for Johne's disease

Examination of the family breeding chart from an infected herd suggests a familial association for Johne's disease (Fig 1.). It is now recognised that infection can be transmitted vertically, in utero and via colostrum and that faeco-oral transmission takes place in the neonatal period. Nevertheless not all offspring or generations developed disease. A similar pattern of disease frequency has been used as evidence for a genetic component cause of Crohn's disease. The importance of host genotype in the development of Johne's disease is unknown.

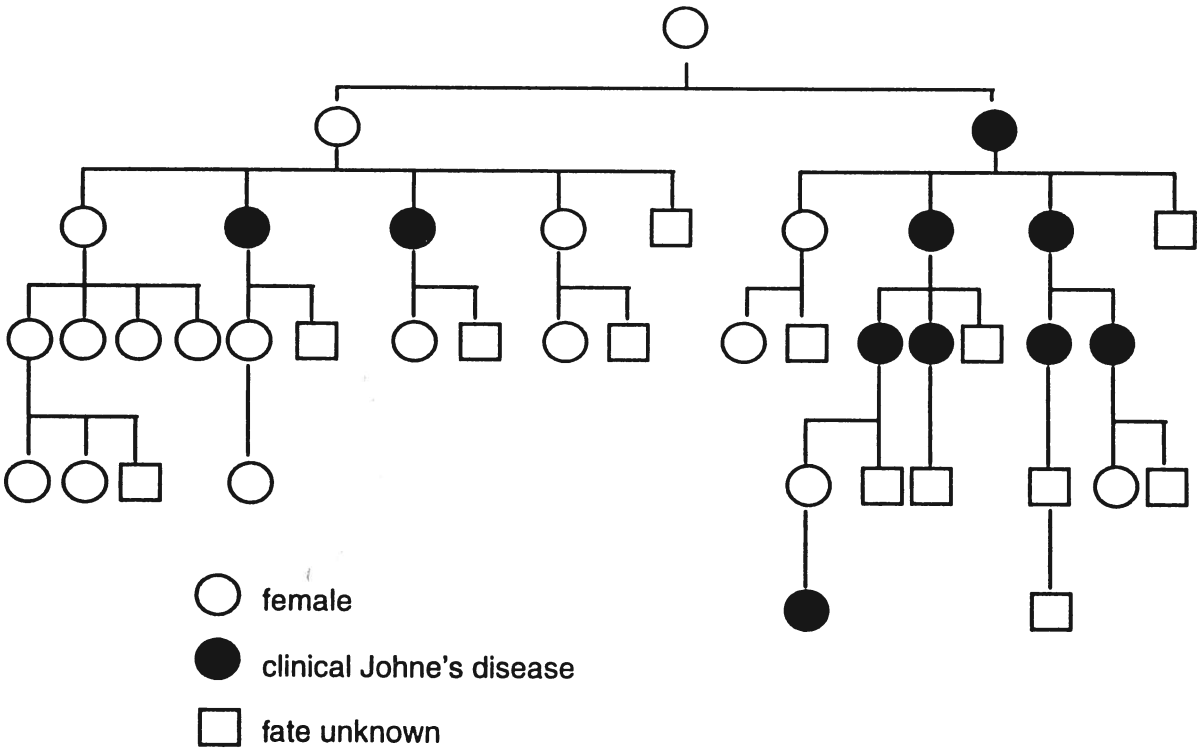

Fig.1 A family breeding chart from a herd with Johne's disease (after Mathews1947)

Psychogenic theories and Johne's disease

Stress is known to be associated with the development of clinical Johne's disease. Classically, the disease appears soon after parturition but it may also occur following transportation, a change in diet or environment.

Dietary theories and Johne's disease.

Crohn's disease has been successfully controlled by dietary manipulation. There are also reports in the literature of similar successes with Johne's disease (Spicer 1934). Removal of

cereals and increasing the mineral content of the diet was associated with the disappearance of clinical signs of disease in a number of herds.

Immunological theories

The immune response of the host plays a major role in the generation of the lesions of Johne's disease and Crohn's disease and represents one of the most exciting areas for the development of comparative research and an improved basic understanding of cell mediated immune responses in the ruminant intestine. A detailed review of the immune response to *M. paratuberculosis* may be found elsewhere (Chiodini 1996). It is worth emphasisng that the host immune response to *M. paratuberculosis* produces the characteristic lesions in the intestine rather than any direct toxic effect of the bacillus but it is also important to recognise that in many cases the immune response clears and eliminates the organism. The role of vaccination in preventing clinical disease in unclear. It does not prevent infection and may exert its effect by preventing the generation of a damaging immune response. The improvement in the clinical signs of cattle after the intravenous administration of johnin (a protein extract of *M. paratuberculosis*) have led some workers to suggest that the disease represents a hypersensitivity response to mycobacterial antigens. (Merkal et al 1970).

Having questioned and reviewed the evidence for and nature of the causal relationship between *M. paratuberculosis* and Johne's disease we now examine the evidence that *M. paratuberculosis* is associated with Crohn's disease.

MYCOBACTERIUM PARATUBERCULOSIS AND CROHN'S DISEASE

The revival of interest in a possible role for *M. paratuberculosis* in the aetiology of Crohn's disease stimulated the application of molecular biological techniques to this micro-organism. The identification of a specific insertion sequence IS900 which was present in multiple copies (15-20) in the genome of *M. paratuberculosis* was a major advance and allowed the development of a specific polymerase chain reaction (PCR) capable of detecting between 10-100 organisms (Green et al 1989, Sanderson et al 1992). This overcame one of the greatest problems associate with *M. paratuberculosis*, the difficulty in culturing it. Even in pluribacillary forms of Johne's disease in sheep, it has proved impossible to culture bacteria in a significant number of cases. *M. paratuberculosis* requires a minimum of 2 months to grow *in vitro* and in the case of some of the human isolates positive cultures have been obtained after years rather than months.

The use of DNA technologies also brought a new dimension to the classification of mycobacteria. The sequence homology between *M. paratuberculosis* and *M. avium* was so high (>90%) that it is now considered by some groups to be a subspecies of *M. avium, (M. avium. Subsp. paratuberculosis).* Current evidence suggests that the presence of IS900 is the only difference between them.

The relationship between *M. paratuberculosis* and Crohn's disease has been investigated using two approaches; an examination of the immune response to *M. paratuberculosis* and the presence of the micro-organism.

Crohn's disease and the immune response to *M. paratuberculosis*

Attempts to test the hypothesis using serological studies based on an ELISA have provided controversial results. There are reports of increased antibody levels to *M. paratuberculosis* (Thayer et al 1984, Elasaghier et al 1992) but a number of other studies have failed to confirm this association (Mcfadden and Houdayer 1988, Stainsby et al 1993). The equivocal nature of these results is not surprising. The absence of an antigen which is specific for *M. paratuberculosis* has hampered the control of Johne's disease in cattle. Most of the serological tests used suffer poor sensitivity (Table 2).

Table 2. The sensitivity and specificity of tests used in the diagnosis of Johne's disease

Test	Sensitivity for clinical disease %	Sensitivity for subclinical disease %	Specificity	Reference
[a]CFT	55	11-38	97-99	Sherman et al 1990 Sockett et al 1992
[b]AGID	41-97	19-27	88-100	Sherman et al 1984 Sockett et al 1992
[c]FAT	72		60	Goudswaard et al 1976
[d]ELISA	51-66	59	95	Sockett et al 1992

[a]CFT- complement fixation test; [b]AGID- agar gel immunodiffusion
[c]FAT- fluorescent anitbody test; [d]ELISA- enzyme linked immunosorbent assay

Recentlly a *M. paratuberculosis* recombinant polypeptide a362 has been used to measure the isotype specific serum antibody response. IgA titres showed a bimodal distribution with 36% of the patients suffering being in the highest peak. (Vannuffel et al 1994). The authors suggest that *M. paratuberculosis* may be involved in the aetiology of disease in these individuals.

Mycobacterial antigens stimulate a strong cell mediated immune response and its association with Crohn's disease has also been investigated. The results suggest that there is no association between *M. paratuberculosis* and Crohn's disease (Rowbotham et al 1995), however the same arguments raised against the ELISA results (antigen specificity and the difficulty in interpreting the response) also apply. In summary, little conclusive evidence for or against the hypothesis can be drawn from these results.

Crohn's disease and the presence of *M. paratuberculosis* in affected tissue

Mycobacterium paratuberculosis has been detected in resected tissue from patients with Crohn's disease by bacterial culture and PCR.

Bacterial culture: Since the first isolation of *M. paratuberculosis* from Crohn's disease patients (Chiodini et al 1984) a number of laboratories in different parts of the world have isolated this organism. In total 135 patients and 121 controls have been examined and *M. paratuberculosis* has been cultured from 27% (36) of the patients and 0.8% (1) of the controls. (Chiodini and Rossiter 1996). An important feature of these isolates is that in many of them (72%) the organisms were in a spheroblast or cell wall deficient form. These are not acid fast and can only be positively identified in culture using the IS900 specific PCR
PCR: In 1992, *M. paratuberculosis* DNA was detected in 65% (26/40) of patients with Crohn's disease compared with 4%(1/23) patients with ulcerative colitis and 12% (5/40) of controls without inflammatory bowel disease (Sanderson et al 1992). A number of groups have repeated these studies with mixed results. In total 211 patients, 163 controls and 110 patients with ulcerative colitis were examined in these studies. 28.9%(61/211) of patients with Crohn's disease displayed a positive PCR reaction compared with 13.6% (15/110) patients with ulcerative colitis and 16.5% (27/163) controls. Details of these results are shown in Table 3.

Table 3.The relationship between IS900 PCR positive tissues and Crohn's disease

Percentage positive (number positive/number tested) Crohn's disease	Ulcerative colitis	Controls	Reference
65 (26/40)	4 (1/23)	12.5 (5/40)	Sanderson et al 1992
72.3 (13/18)	20 (1/5)	29.2 (7/24)	Dell'Isola et al 1993
12.9 (4/31)	---	(0/30)	Fidler et al 1994
100 (10/10)	61.1 (11/18)	87.5 (14/16)	Suenaga et al 1995
100 (8/8)	100 (2/2)	(0/4)	Mishina et al 1996
(0/36)	(0/13)	(0/23)	Dumonceau et al 1996
(0/68)	(0/49)	(1/26)	Rowbotham et al 1996

These results remain controversial. The presence of *M. paratuberculosis* in non-inflammatory bowel disease controls is often considered to detract from the hypothesis but might be expected because in Johne's disease infection occurs in the absence of clinical disease.

An important feature of Johne's disease is that although infection occurs in the young animal clinical disease does not develop until early adulthood. If the similarity between Johne's disease and Crohn's disease encompassed not only the infectious agent but also the relationship between infection and disease, then it would account for two interesting epidemiological features of Crohn's disease that are often used in support of non-infectious causes: the rare occurrence of disease in husband and wife and the occurrence of disease in monozygotic twins who had lived apart since childhood. This hypothesis is developed in more detail elsewhere (Morgan 1987).

In order to test the hypothesis that Crohn's disease was associated with an environmental exposure during childhood, possibly to *M.paratuberculosis,* we studied a small Cotswold village of 2000 people with a "cluster" of 12 patients with Crohn's disease. Three study designs were used; cohort, case-control, and cross sectional survey.

We argued that if the occurrence of disease was associated with a childhood environmental exposure within the village during then the prevalence of disease in individuals who had moved away from the village to distant locations would also be elevated. We set about identifying and tracing all the individuals born in the village during the 20-year period from 1940-60, the time period when the greatest number (5) of cases had been born. A list of births was obtained from the parish baptism records and register of births. The electoral register was then examined to see how many of the cohort still lived in the village. Individuals who had left the village were traced via the Office of Population and Census Studies (OPCS). The last known address and National Health number were obtained and the individuals traced via family practitioner committees to their doctor. Individuals in the cohort were notified of the study by letter, asked to complete a short questionnaire and to participate in the study. The questionnaire was designed to identify individuals suffering from Crohn's disease, ulcerative colitis, or symptoms of chronic inflammatory bowel disease (chronic diarrhoea). These were included in a list of 11 diseases and clinical signs. Their general practitioner was also notified of the study. A total of 725 individuals were on the birth list, 88 were still on the electoral

register and 2 had Crohn's disease. Attempts were made to trace the remaining 637 individuals. At the time of writing, there was no trace of 119, this included 13 deaths, embarkations and adoptions. 349 patients had been traced, 6.3% (2/349) were identified as having had symptoms of inflammatory bowel disease but non of these was confirmed as Crohn's disease. No other cases of Crohn's disease were identified in the cohort. These results did not support the hypothesis.

A case-control study was carried out using 11 cases and three controls per case, matched by sex and nearest birth date. All the individuals who agreed to take part in the study were visited and interviewed using a questionnaire designed to collect information on events occurring during the first 10 years of life. Where possible parents were also visited to enable the collection of data about the first few years of life and the validation of other exposure variables. If parents were deceased an elder sibling was approached. These data have not been analysed.

The prevalence of Johne's disease on village farms during the period of interest was determined by interviewing the farmers using a questionnaire designed to obtain information on a large number of variables relating to farming and animal health. The farmers were unaware of the hypothesis. Of the 20 farmers who kept cattle at the time of interest 9 (45%) said they had had Johne's disease on the farm and three of these had village milk rounds. This appeared to be a high prevalence of disease but veterinarians who had been in practice in the 20 years period of the study they were not surprised by this figure. It became clear that the prevalence of Johne's disease within the UK cattle herd was unknown. Circumstantial evidence suggested that it had decreased but there had been no study since the 1950s a time when if was believed to be, economically, the most important disease of cattle; the proportion of affected farms varying from 7.8-9.5% (Smith 1954) and the proportion of affected cattle 3.8-4.5% (Withers 1959). In 1995 we set out to determine the prevalence and incidence of Johne's disease in the UK; the farm level risk factors associated with clinical disease and the prevalence of sub-clinical disease.

The farm prevalence and within herd incidence of paratuberculosis was determined by a self administered postal questionnaire sent to a stratified random sample of 3772 dairy herds in England and the Border regions of Wales in 1995. The response rate was 78.3% with 77.3% usable returns. Full details are reported elsewhere (Cetinkaya et al 1997).

Farmers were asked if they had seen a clinical case or Johne's disease on their farms "ever", within the last 10 years or in 1993 or 1994. The results are shown in Table 4.

When the overall proportion of farms affected was estimated using cases diagnosed by a veterinarian or VI centre, the only significant difference was in the prevalence "ever". It was estimated to be 11.4 % (95% CI 10.3-12.5) and was significantly different from that of 17.4 % (95% CI 16.1-18.7) obtained without consideration of the type of diagnosis ($p<0.001$).

The incidence rate of clinical disease was also estimated for all herds. It was the same in both years; 3.0/10,000 cow-years (60/238,745 and 72/251,210). The incidence rate for clinical disease in each region ranged from 1.5/100 cow years in the north in both years to 3.0/100 cow years in the central region in 1994 (Table 5), but the differences were not statistically significant ($p=0.8$ for 1993 and $p=0.07$ for 1994).

The prevalence of subclinical disease was estimated by examining three intestinal lymph nodes; the ileocaecal, jejunal and one from the mesenteric lymph chain collected from 1553 adult cull cows in three abattoirs in the south west of England for the presence of *M. paratuberculosis* by PCR and culture. Full details of this study have been reported elsewhere (Cetinkaya et al 1996).

Table 4. The proportion of farms affected with Johne's disease in each region

Region	Ever	(95% CI*)	Proportion % 1985-94	(95% CI)	°1993	(95% CI)	1994	(95% CI)
South	18.6	(16.4-20.8)	6.3	(4.8-7.8)	2.1	(1.2-3.0)	1.8	(1-2.5)
Central	17.5	(15.1-19.9)	4.5	(3.2-5.8)	1.4	(0.7-2.1)	1.2	(0.5-1.9)
North	15.8	(13.6-18.0)	3.7	(2.6-4.8)	0.8	(0.3-1.3)	1.1	(0.6-1.6)
Overall	17.4	(16.1-18.7)	4.9	(4.2-5.6)	1.5	(1.1-1.9)	1.3	(0.9-1.7)

* p=0.03
° p=0.054

Table 5. The incidence of clinical Johne's disease in each region in 1993 and 1994 (Number of cases/100 cow-years)

Region	1993		1994	
South	1.7	(26/1524)	2.4	(32/1311)
Central	1.8	(20/1115)	3.0	(25/823)
North	1.5	(14/947)	1.5	(15/1018)
Overall	1.7	(60/3586)	2.3	(72/3152

The proportion of samples giving a positive PCR product with the molecular size of 400 bp is shown in Table 6. Neither the differences between age groups nor abattoirs was statistically significant (p> 0.05).

Table 6. The proportion of PCR positive lymph nodes from cull cows and young cattle collected at each abattoir

Abattoir	Cull cows %	CI	Young cattle %	CI
Keynsham	2.6	(1.5-4.4)	2.2	(0.05-11.5)
Langport	5.2	(3.1-7.9)	2.8	(0.8-6.9)
Hatherleigh	3.3	(1.7-5.5)	---	
Total	3.5	(2.6-4.7)	2.0	(0.6-4.5)

Mycobacterial growth in culture was observed in 30 (58.8%) of the 51 PCR positive samples and 5 (22.7%) of the 22 PCR inconclusive samples. All these cultures were then confirmed as *M. paratuberculosis* by PCR. The proportion of subclinical Johne's disease estimated from culture was 2.3% (35/1553) (CL 1.6-3.1). There was no significant difference between this and the 3.3%obtained from PCR results (p>0.05). These results show that *M. paratuberculosis* exists within the UK cattle population mainly as a subclinical infection. None of the cull cows or

young cattle in the abattoir survey showed any gross pathological lesions of the small intestine or intestinal lymph nodes. Currently there is no diagnostic test which is sensitive and specific enough to identify these animals. Infected cows may shed M. paratuberculosis in their milk. The recent identification of M. *paratuberculosis* PCR products in 7% of retail milk samples, its ability to resist pasteurisation under and the growing evidence for a link between this organism and some cases of Crohn's disease makes Johne's an important disease.

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BOVINE SPONGIFORM ENCEPHALOPATHY

AN EXPLORATORY CASE-CONTROL STUDY OF RISK FACTORS FOR BOVINE SPONGIFORM ENCEPHALOPATHY ON SWISS DAIRY FARMS

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With 230 cases reported by 31 December 1996, Switzerland has experienced the second largest reported epidemic of bovine spongiform encephalopathy (BSE) in the world after the UK. The first case occurred in November 1990. In 1995 the peak of the Swiss epidemic was reached with 68 cases, followed by a decrease in 1996 (45 cases). The epidemic of BSE in the United Kingdom occurred in cattle after the inclusion of protein derived from infected tissues in their feed. Further epidemiological investigations supported the association of meat-and-bone meal as the only identifiable source of infection (Wilesmith et al., 1988, 1991, 1992). In the UK,from July 1988 the feeding of meat-and-bone meal of ruminant origin to ruminants was prohibited (referred to as the 'feed ban', Order 1988). In September 1990, the introduction of the specified bovine offals ban further prohibited the inclusion of ruminant-derived protein in feed used for non-ruminants (Order 1990).

After the first case of BSE in Switzerland had been confirmed, a ban on the feeding of protein derived from animal waste to ruminants was introduced. An observational study analysed the first 24 cases and led to the hypothesis that the BSE epidemic, like that in the UK, was associated with the consumption by cattle of contaminated feed concentrates which had been imported directly or indirectly from the UK (Hörnlimann et al., 1994). A case-control study was designed to investigate whether the occurrence of BSE in Switzerland was consistent with the UK experience and with the hypothesis of Hörnlimann et al. (1994).This paper presents descriptive data and preliminary results from univariate analyses carried out at the farm level.

MATERIALS AND METHODS

This investigation was conducted as a case-control study.

Definition of cases and controls

Case farms were defined as dairy farms on which BSE infection had been suspected and later confirmed by histological investigation (Reference Laboratory of

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the Institute of Animal Neurology, Veterinary Faculty, University of Berne) reported between November 1990 and 31 December of 1995. A total of 165 farms satisfied the criteria and consented to participate, and so could be included in the study.

Control farms were neighbourhood-matched (area with the same postal code) and randomly selected using a list of the Swiss Federal Office of Statistics. For each case farm, three control farms were initially selected, and each of them was contacted by mail and by 'phone and invited to participate in the study. In cases where none of the three potential control farms consented, additional addresses were selected if there were more dairy farms in the village. A total of 157 control farms were enrolled.

Consequently, the unmatched analysis contained 322 farms (165 case and 157 control farms). The matched-pairs analysis consisted of 142 pairs, where there was a case and a control in the same postal district. This sample size allowed the detection of an odds ratio (OR) >2.5 with 95% confidence and 80% power for the unmatched analysis (assuming a risk exposure of 70%). The paired analysis allowed the detection of an OR >3 with 95% confidence and 80% power.

In addition, the farm characteristics were compared with data of 113 dairy farms selected for an earlier health profile study (Staerk et al., 1996). These farms had been randomly selected through the Swiss Federal Office of Statistics and thus formed a second, unmatched control group representing the national dairy farm population.

Data collection

Data collection on case and control farms was based on interviews with the farmers, and was conducted on the farm using a standardised questionnaire (available in French or German). The interviews were undertaken by the first author and two well-trained and supervised interviewers. The questionnaire consisted of four parts :

1. The characteristics of the farm: (farm size, production system, calving accommodation), and herd management practices (calving management, policies for stock replacement and movement to summer pastures) Questions referred to the year of occurrence of BSE. For recent cases, this was the year of the survey (i.e. 1995). However, information on management practices was also obtained for all the previous years during which the BSE animal had lived on this farm.

2. The feeding management of cattle of all age classes: calves (up to three months of age), heifers (from four months up to the first calving) and adult milk cows. Whenever BSE animals were not homebred, the farms of origin were identified and the feeding history was also investigated for the time during which the animal stayed on these farms; special emphasis was placed on calfhood feeding.

3. The nature of the disease process in the individual BSE animal: Emphasis was placed on the recall of events before the onset of clinical signs, such as potential predisposing factors for the development of clinical signs (transportation, movement to summer pastures, preventive measures).

These data were included to facilitate an analysis at the individual animal level. This will be described in a subsequent paper.

4. Characteristics of the herd: Whenever possible, these data were obtained through the individual pedigree cards from breeding societies or through control sheets from the milk recording agency.

   In addition, all receipts for purchased cattle concentrates were examined, to obtain detailed information on feeding management practices on all farms that were investigated. Bills for all the years the BSE animal had lived on each case farm, and for the same time-period on their respective control farms, were copied.

Data management and analysis

Data from the questionnaires and record sheets were entered in the Microsoft Access Version 2.0 relational database (Microsoft Corporation, Redmont, WA, USA). Evaluation of risk factors was performed by analysing the data in a case-control study format with three subsets of data. The first set was the main data-set including all farms (referred to as series A, 322 farms), which was used for the unpaired analyses. In the second set, only complete pairs of case and control farms were analysed (series B, 142 pairs). The third data-set consisted of the paired farms with only homebred BSE animals (series C, 101 pairs). All statistics were calculated using STATA Version 5.0 (Statistics/Data Analysis; Copyright 1985-97, Stata Corporation, Texas).

RESULTS

Compliance

From November 1990 until 31 December of 1995, 189 BSE animals on 183 farms (four farms with 2 animals, one farm with 3 animals) were registered and fulfilled the entry criteria. One hundred and sixty five farms consented to participate, achieving a compliance rate of 90% among case farms.

Of the 611 potential control farms, randomly selected by the Federal Office of Statistics, 157 agreed to participate in the study. This low rate appeared to be mainly due to lack of interest or the fact that there was no longer any dairy farming at that address. In small villages with very few farms, it was occasionally impossible to find a control, therefore 8 farms with BSE animals could not be paired with a control.

Features of farms

Case and neighbourhood control dairy farms were located in all but five of the 26 Swiss cantons. A third of the BSE-affected animals were kept on two or more farms during their life span. In 80% of the dairy farms,milk production was the only source of income. Crop production was the second important production branch. Less than 10%

of case and control farms were not organised in a breeding society. The dominant breeds were Simmental, Red Holstein, their crossbreeds, and Swiss Brown - the main Swiss dairy breeds. The age distribution of the BSE-affected animals was, as expected, between four and six years of age (mean 5.15 years, median 5.17 years range 2.65-7.61)

To calculate the average age of lactating cows, the age of all the cows in the herd at the time of onset of the clinical signs of the BSE-affected animal was used. Animals younger than 18 months were excluded. The average age of milk cows was not statistically different between case and control farms (4.74 years ±(1.09) years vs 4.94 ± (1.20) years).

The farm characteristics of the case and control farms were also compared with data of 113 farms of an earlier health profile study (Staerk et al. 1996), because these farms were representative of all Swiss dairy farms, stratified by canton and minimal herd size. The distribution pattern between agricultural zones was the same in both studies.

Table 1 shows the average farm size, the average milk quota per year and the average number of milk cows on the case and control farms, as well as the dairy farms of the health profile study.

Case and control farms did not differ statistically in any of the three criteria. On the other hand, the farms of the sample for the health profile study differed significantly from cases and control farms: lower farm size (*t*-test: $p < 0.05$), lower milk quota compared with case farms (*t*-test: $p < 0.03$) and lower numbers of milk cows (*t*-test: $p<0.05$). The only exception was seen for the milk quota, which was not statistically significantly lower when compared with that of the control farms.

Farm Management

A large number of potential risk factors (more than 40 variables analysed at the farm level) were assessed in the questionnaire. Risk factors considered to be of most interest are shown in Tables 2 (continuous variables) and 3 (dichotomous variables).

An important finding related to the farm area. Case farms had a significantly larger farm area than control farms. The difference between the size of farms with homebred cases compared with control farms was even stronger (series B mean 24.35 ± 19.02 for case farms; 20.77 ± 11.23 for control farms p= 0.04; series C mean 27.17 ± 21.18 resp. 21.43 ± 12.22; p= 0.01). The presence of pigs was twofold more likely on homebred case farms than on control farms (OR 2.15; 95%CI CI 1.08 to 4.53, p=0.02). This association was not seen when comparing the other data-sets.

The use of organophosphate containing products (Galesan®, Neguvon®, Tiguvon®, Antigal®, Sebacil®, Galtox®) against ectoparasitices was reported as an aspect of herd management. The analysis of all three data-sets revealed no positive association of the occurrence of BSE with the use of any of these products.

Table 1. Comparison between dairy farms of case-control study and farms of health profile study [a]

	Case farms (1)			Neighbourhood control farms (2)			Health profile dairy farms (3)			Comparison between farms t-test p value		
Variables	No.	Mean	SD	No.	Mean	SD	No.	Mean	SD	1-2	1-3	2-3
Farm size (ha)	161	23.83	18.27	156	20.78	11.74	113	18.17	9.5	0.08	<0.01	0.05
Milk quota (kg/year) *10^3	159	81.2	53	156	76.8	43.5	113	68.9	33.5	0.42	0.03	0.11
Number of milk cows	159	18.65	9.42	156	17.28	7.87	113	15.5	6.08	0.16	<0.01	0.05

No. = Number of farms; SD = Standard deviation

[a] Data from a previous dairy health profile study (Staerk et al., 1996)

Table 2 Comparison of farm characteristics with the occurrence of BSE on Swiss dairy farms: continuous variables

Farm Characteristics unpaired (Serie A)	Cases			Controls				
Variable	No.	Mean	SD	No.	Mean	SD	t	P-Value
Farm size (ha)	161	23.83	18.27	156	20.78	11.74	-1.76	0.08
Milk contingent x 10^3	159	81.23	53.00	156	76.82	43.46	-0.81	0.42
Herd size of milk cows	159	18.65	9.42	156	17.28	7.87	-1.40	0.16
Mean age of milk cows	121	56.98	13.08	120	59.27	14.38	1.29	0.20
Proportion of silage feed (%) in forage [b]	82	47.00	21.07	92	39.00	20.76	-2.63	**0.01**
Proportion of succulent feed (%) in forage [c]	43	15.00	8.00	46	15.00	11.00	0.08	0.94
paired * (Serie B)	Cases			Controls				
	No.	Mean	SD	No.	Mean	SD	t	P- value [a]
Farm size	142	24.35	19.02	142	20.77	11.23	2.13	**0.04**
Milk contingent x 10^3	140	82.63	54.48	140	77.27	44.41	1.07	0.29
Herd size of milk cows	140	18.99	9.73	140	17.39	7.88	1.73	0.09
Mean age of milk cows	88	55.91	1.36	88	58.53	1.56	-1.35	0.18
Proportion of silage feed (%) in forage [b]	63	46.00	20.00	63	41.00	20.00	1.81	0.08
Proportion of succulent feed (%) in forage [c]	21	17.00	8.00	21	13.00	9.00	2.04	0.05
paired*: only homebred farms (Serie C)	Cases			Controls				
	No.	Mean	SD	No.	Mean	SD	t	P- value [a]
Farm size	101	27.17	21.18	101	21.43	12.22	2.56	**0.01**
Milk contingent x 10^3	100	89.46	56.29	100	79.21	46.72	1.62	0.11
Herd size of milk cows	101	20.16	9.97	101	18.03	8.36	1.88	0.06
Mean age of milk cows	61	56.24	1.67	61	58.84	1.94	-1.08	0.29
Proportion of silage feed (%) in forage [b]	46	44.00	19.00	46	41.00	19.00	1.04	0.30
Proportion of succulent feed (%) in forage [c]	15	17.00	9.00	15	14.00	9.00	1.50	0.15

[a] paired t-test; No=Number of farms; SD=Standard deviation

[b] On farms with silage; [c] On farms with no silage

Table 3 Comparison of farm characteristics with the occurrence of BSE on Swiss dairy farms: dichotomous variables

	Cases		Controls		unpaired (Serie A)					paired * Serie (B)				
	No.	(%)	No.	(%)	OR	CI-95%		chi	P-value	OR	CI-95%		chi	P- value [a]
Animals														
Fattening calves present	60	38%	64	41%	0.88	0.56	1.38	0.31	0.58	0.96	0.53	1.73	0.02	0.88
Fattening heifers present	28	17%	31	20%	0.85	0.48	1.49	0.32	0.57	0.88	0.42	1.84	0.12	0.73
Presence of pigs	81	50%	64	41%	1.43	0.92	2.33	2.58	0.11	1.48	0.85	2.63	2.12	0.15
Presence of poultry	78	48%	79	51%	0.91	0.58	1.40	0.20	0.66	0.84	0.49	1.45	0.42	0.52
Calving accomodation														
Calving at usual place in the stable	155	96%	145	93%	1.68	0.65	4.31	1.11	0.29	1.33	0.41	4.66	0.29	0.59
Calving on pasture possible	112	69%	117	75%	0.73	0.44	1.19	1.59	0.21	0.66	0.36	1.17	2.28	0.13
Removal of placenta in or on dung hill	124	77%	113	72%	1.24	0.75	2.05	0.71	0.40	1.44	0.76	2.80	1.45	0.23
Removal of placenta within one hour after calving	81	51%	81	52%	0.95	0.61	1.48	0.05	0.82	0.97	0.57	1.65	0.02	0.90
Antiparasitica														
Use of organophospate containing products [b]	61	42%	52	35%	1.29	0.81	2.07	1.16	0.28	1.17	0.65	2.14	0.32	0.57
Feed production and storage														
Silage storage	82	51%	96	62%	0.64	0.41	0.99	3.85	**0.05**	0.41	0.17	0.92	5.45	**0.02**
Succulent feed storage	43	28%	46	30%	0.89	0.55	1.46	0.20	0.65	0.78	0.40	1.51	0.61	0.43
Hay ventilation in barn for drying	118	74%	114	74%	0.99	0.60	1.63	0.00	0.96	1.31	0.65	2.69	0.68	0.41

[a] McNemar chi-square test : Unadjusted odds ratio (OR) with 95% confidence interval, chi-square and p-value;
[b] Galesan®, Neguvon®, Tiguvon®, Antigal®, Sebacil®, Galtox®; No. (%)= Number of exposed farms and percentage

Table 3: continued

	paired[a]: only homebred farms (Serie C)				
	OR	CI-95%		chi	P- value[a]
Animals					
Fattening calves present	1.05	0.53	2.08	0.03	0.87
Fattening heifers present	0.8	0.34	1.83	0.33	0.56
Presence of pigs	2.15	1.08	4.53	5.49	**0.02**
Presence of poultry	0.87	0.45	1.66	0.21	0.65
Calving accomodation					
Calving at usual place in the stable	1.00	0.23	4.35	0.00	1.00
Calving on pasture possible	0.94	0.44	1.98	0.03	0.86
Removal of placenta in or on dung hill	1.46	0.68	3.22	1.12	0.29
Removal of placenta within one hour after calving	0.88	0.47	1.63	0.19	0.66
Antiparasitica					
Use of organophospate containing products[b]	1.37	0.69	2.80	0.95	0.33
Feed production and storage					
Silage storage	0.28	0.08	0.78	7.35	**0.01**
Succulent feed storage	0.87	0.38	1.95	0.14	0.71
Hay ventilation in barn for drying	1.50	0.63	3.73	1.00	0.32

[a] McNemar chi-square test : Unadjusted odds ratio (OR) with 95% confidence interval, chi-square and p-value; [b] Galesan®, Neguvon®, Tiguvon®, Antigal®, Sebacil®, Galtox®; No. (%)= Number of exposed farms and percentage

On all study farms, 95% of all calvings occurred at the cow's usual place in the stable. Separate calving accommodation was exceptional. The placenta was deposited in or on the dung hill, within one hour in 50% of the farms. It is noteworthy that in about 25% of both cases and controls, farmers mentioned the removal of the placenta to sites where scavengers like foxes or birds of prey would have access to it.

Farms with silage storage were significantly less at risk of the occurrence of BSE than farms with no such feed preservation facility. Analysis showed a risk of OR=0.64 for series A, (95%CI: 0.41-0.99, p=0.05) the unmatched pairs, decreasing to OR=0.41 (95%CI CI: 0.17 - 0.92, p=0.02) in complete pairs and OR=0.28 (95%CI CI: 0.08 - 0.78, p=0.01) in the analysis restricted to homebred cases. Virtually all of the farms with silage storage used a elevator (silo) as storage container. No difference was observed when plastic-covered round bales were used instead. Further relevant associations could only be shown in the unmatched analysis, where the proportion of silage used in preparing cattle feed was significantly higher in case farms than in control farms (series A p=0.01).

Feeding Management

Tables 4 (dichotomous variables) and 5 (continuous variables) summarise the exploratory analysis of risk factors concerning the feeding management. For all the variables investigated in calf feeding, no differences between cases and control farms were observed. Data considered here were for calves being reared for either future milk-production or breeding. More than 95% of case and control farmers reared their calves with cow milk from their cow herd up to three months. Milk powder as a milk substitute was encountered in less than 20% and the use of high-nourishment powder even less (<10%). Special products for calf-rearing were used additionally in 70% of the farms, starting at about three weeks. The mean daily proportion of these products in the rearing feed did not differ between cases and controls.

Interestingly, 35% of case farms and 37% of the control farms were feeding concentrates or cereal mixtures designed for lactating cows to calves being reared. With regard to heifer feeding, neither the use of heifer-rearing feed nor feeding heifers with concentrates for cows was associated with BSE. The only significant association found was related to milk cow feeding. Feeding proprietary concentrates to milk cows appeared to be a BSE risk factor when compared with the feeding of cereal mixtures and protein supplements such as soya products (series B: OR=1.82; 95%CI: 0.98 - 3.51, p=0.04; series C: OR=2.67; 95%CI 1.2 - 6.51, p=0.01). Farmers using silage were not less likely to feed concentrates (series B OR=0.57 95%CI: 0.29-1.09, p= 0.07, series C OR=0.5, 95%CI: 0.22-1.08, p=0.06).

Only 5% of case and control farmers declared that they had ever used separate components like meat-and-bone meal (meat meal, bone meal, blood meal, tallow) from bovine sources in home-made mixtures. The small sample size did not allow further subgroup analysis. A third of farmers in both study groups were using fish meal or oil as supplemental feed. Feeding pig or poultry feed to cattle was found to be an exceptional feeding practice (2% of case and control farms).

Preliminary results for the feed products consumed in all age categories revealed a similar distribution pattern between case and control farms. Neither the products of

Table 4: Comparison of dichotomous feeding factors with the occurrence of BSE on Swiss dairy farms

Variable	unpaired: (serie A) Cases No.	%	Controls No.	%	OR	CI-95%		chi	P-value	paired * (serie B) OR	CI-95%		chi	P-value [a]
Calf feeding (up to 3 month)														
Cow milk	148	94%	152	97%	0.39	0.13	1.20	2.61	0.11	0.50	0.11	1.87	1.33	0.25
Milk powder	25	16%	26	17%	0.94	0.52	1.70	0.04	0.84	0.94	0.43	2.02	0.03	0.86
Nourishment powder	14	9%	11	7%	1.28	0.57	2.86	0.35	0.55	1.38	0.50	3.94	0.47	0.49
Calf rearing feed	118	75%	110	71%	1.23	0.75	2.02	0.69	0.41	1.42	0.82	2.50	1.72	0.19
Concentrates or cereal mixture designed for milk cows	55	35%	59	37%	0.90	0.57	1.42	0.22	0.64	0.81	0.46	1.41	0.64	0.42
Heifer Feeding (up to first lactation)														
Heifer rearing feed or concentrates	126	80%	110	71%	1.65	0.98	2.76	3.58	0.06	2.00	0.76	5.86	2.33	0.13
Concentrates or cereal mixture designed for milk cows	61	38%	50	31%	1.36	0.86	2.15	1.67	0.20	1.33	0.79	2.28	1.29	0.26
Concentrates or cereal mixture designed for milk cows to calves and heifers	47	30%	42	26%	1.17	0.72	1.90	0.39	0.53	1.15	0.66	2.00	0.28	0.60
Milk cow Feeding														
Cereal mixture	75	47%	78	53%	0.78	0.50	1.22	1.19	0.28	0.74	0.41	1.31	1.19	0.28
Protein supplement	38	24%	33	23%	1.08	0.63	1.83	0.07	0.79	1.09	0.59	2.01	0.08	0.77
Concentrates	113	71%	92	63%	1.44	0.89	2.33	2.24	0.13	1.82	0.98	3.51	4.08	**0.04**
Feeding silage, but no concentrates	23	15%	36	24%	0.57	0.32	1.01	3.66	0.06	0.57	0.29	1.09	3.27	0.07

Table 4: Continued

Use of home-made feed mixtures														
Ever home mixture with potential infectious material	8	5%	6	4%	1.30	0.46	3.67	0.23	0.63	.	.	.	.	.
Ever fish components	39	24%	40	26%	0.93	0.56	1.55	0.07	0.80	1.09	0.59	2.04	0.09	0.77
Use of non ruminant feed for cattle														
Ever used pig feed for cattle	4	2%	3	2%	1.30	0.32	5.31	.	1.00 [b]	.	.	.	.	.
Ever used poultry feed for cattle	4	2%	5	3%	0.78	0.22	2.72	.	0.75 [b]	.	.	.	.	.

[a] McNemar chi-square test: Unadjusted odds ratio (OR) with 95% confidence interval, chi-square and p-value
[b] Fisher's exact test (two sided) ; No. (%)= Number of exposed farms and percentage

Table 4: Continued

Variable	paired*: homebred farms serie C				
	OR	CI-95%		chi	P-value [a]
Calf feeding (up to 3 month)					
Cow milk	1.00	0.13	7.46	0.00	1.00
Milk powder	0.87	0.38	1.95	0.14	0.71
Nourishment powder	2.75	0.81	11.84	3.27	0.07
Calf rearing feed	1.85	0.90	3.95	3.27	0.07
Concentrates or cereal mixture designed for milk cows	0.87	0.45	1.66	0.21	0.65
Heifer Feeding (up to first lactation)					
Heifer rearing feed or concentrates	1.83	0.62	6.04	1.47	0.23
Concentrates or cereal mixture designed for milk cows	1.40	0.76	2.62	1.33	0.25
Concentrates or cereal mixture designed for milk cows to calves and heifers	1.29	0.70	2.39	0.75	0.39
Milk cow Feeding					
Cereal mixture	0.71	0.36	1.37	1.2	0.27
Protein supplement	1.12	0.55	2.29	0.11	0.74
Concentrates	2.67	1.20	6.51	6.82	**0.01**
Feeding silage, but no concentrates	0.5	0.22	1.08	3.67	0.06
Use of home-made feed mixtures					
Ever fish components	1.33	0.65	2.80	0.71	0.40

[a] McNemar chi-square test : Unadjusted odds ratio (OR) with 95% confidence interval, chi-square and p-value; No. (%)= Number of exposed farms and percentage

Table 5: Comparison of feeding factors with the occurrence of BSE on Swiss dairy farms : continuous variables

Calf feeding (up to 3 month) unpaired (Serie A)	**Cases**			**Controls**				
	No.	Mean	SD	No.	Mean	SD	t	p-value
Mean daily ratio of cow milk (l)	148	6.05	1.82	109	6.18	2.472	0.48	0.63
Mean daily ratio of milk powder (kg)	16	0.33	0.24	9	0.38	0.539	0.32	0.75
Mean daily ratio of nutrition powder (kg)	8	0.19	0.27	6	0.33	0.456	0.70	0.50
Mean daily ratio of rearing feed (kg)	123	0.55	0.40	81	0.61	0.418	0.96	0.34
paired (Serie B)	**Cases**			**Controls**				
	No.	Mean	SD	No.	Mean	SD	t	p-value[a]
Mean daily ratio of cow milk (l)	132	6.05	1.81	132	6.15	2.17	-1.00	0.32
Mean daily ratio of milk powder (kg)	16	0.33	0.24	16	0.33	0.24	.	.
Mean daily ratio of nutrition powder (kg)	8	0.19	0.27	8	0.19	0.27	.	.
Mean daily ratio of rearing feed (kg)	111	0.55	0.41	111	0.56	0.40	-1.44	0.15
paired homebred (Serie C)	**Cases**			**Controls**				
	No.	Mean	SD	No.	Mean	SD	t	p-value[a]
Mean daily ratio of cow milk (l)	99	5.96	1.87	99	6.08	2.34	-1.00	0.32
Mean daily ratio of milk powder (kg)	14	0.32	0.24	14	0.32	0.24	.	.
Mean daily ratio of nutrition powder (kg)	8	0.19	0.27	8	0.19	0.27	.	.
Mean daily ratio of rearing feed (kg)	86	0.59	0.43	86	0.59	0.43	-1.44	0.15

[a] paired t-test; No=Number of farms; SD=Standard deviation

particular companies nor a single type of feed product was found to be associated with the outcome of BSE. A large variety of different products was involved. More than 75% of them were mentioned on a single farm. Only 3% were mentioned in more than 10 farms.

DISCUSSION

The present study of risk factors was designed as a case-control study. Case and control farms were matched on the basis of the postal area code. Since postal districts are small in area in Switzerland, this procedure had the advantage that cases and controls were in the same agricultural zone. It could mean that many factors that were shown in the UK to be potential confounders (Wilesmith et al 1988, 1991, 1992) such as herd size, herd type and geographical location would have been similar in cases and controls. On the other hand, close matching could have led to a risk of overmatching, which would have obscured connections between the BSE risk and features of an agricultural zone. So far we could not identify such possible factors for the Swiss situation.

The sample size in the Swiss study was inevitably limited because the total number of BSE cases in Switzerland from 1990-1995 was only 189, and 10% of the farms involved did not participate in the study. They were mostly those where the case of BSE had occurred early in the epidemic and suibsequently had been very thoroughly investigated. The lower prevalence of BSE in Switzerland compared with the UK must be taken into account when comparing the results with those of UK studies.

In this study of a large number of potential risk factors for BSE in Switzerland, the main associations observed were a positive association with the feeding of proprietary feed concentrates to milk cows, and a negative association with the preservation of fodder on farms as silage.

The feeding of proprietary concentrates to milk cows constituted a high risk factor for BSE in contrast to feeding of cereal mixture and protein supplement. The practice of feed preservation in the form of silage on a farm was found in the paired analyses to be significantly negatively associated with the occurrence of BSE. This raises questions on the causal nature of the association, and the possible mechanism of protection,should it be causal. The possibility that farmers using silage gave less proprietary concentrates was also considered. However, no inverse association could be shown. Farmers using silage did not significantly differ in giving concentrated feed to cattle. The unpaired analyses showed that silage was included in a higher proportion of the cattle feed on case farms than on control farms, which did not support the negative association.

Studies in the UK did not include references to the feeding of milk cows. They did, however, show that a higher risk of BSE was associated with certain calf-feeding practices. In our study, at the exploratory, univariate level, none of the potential risk factors associated with calfhood feeding showed a relationship with the disease outcome. This is in clear contrast to the studies in UK, where feed related factors could be clearly established as risk factors (Wilesmith et al. 1988, 1991, 1992). However, as pointed out by Wilesmith et al. (1992) feeding proprietary concentrates can only be a proxy for BSE exposure.

In the feeding of calves, potential risk factors were the use of milk substitute, nourishment powder and rearing feed for calves. Swiss dairy calves were reared for three months with cow's milk, in contrast to commercial dairy herds in UK, where rearing calves did not receive their mothers' milk except for colostrum in the first days of life (MAFF, Press release, Aug.1996). In the Swiss epidemic, the use of nourishment powder was, in practice, found to be negligible. Calf-rearing feed was given at an early stage (from 3 weeks) as recommended by feed manufacturers (Anonymous, 1996), but there was no difference in this respect between case and control farms.

Potentially infectious material such as meat-and-bone meal, meat meal and others could have been bought separately for use in home mixtures. The procedure of mixing separate components to produce cattle feed requires appropriate tools and knowledge. Mixtures made on the farm did not appear to be used in the dairy farms investigated.

Most frequently, when a farmer used a mixture with single components from his own crop (so called "Lohnmischungen"), a miller was asked to produce it. The composition of these feeds regarding the protein source depended, like proprietary concentrates, on market situations, and therefore could vary very much.

Probably due to ease of handling, the supplemental feeding of fish components like meal or oil was more frequent. According to expert advice, the content of such products can be considered as pure fish and not as a mixture of protein from different sources;therefore it was considered as negligible as potential risk factor for BSE.

Feed intended for non-ruminants could still contain potential infectious material, so the study farmers were asked about feeding poultry or pig feed to cattle to investigate whether this was a common practice on dairy farms. It appeared to happen only on 2 to 3% of the farms. Based on these results, feeding pig or poultry feed products to cattle could be excluded as a risk with regard to disease potential.

The answers to interview questions that concerned banned practices could be subject to bias, and case farmers might have concealed these practices from the interviewers. On the other hand, since a majority of the case farmers experienced negative consequences (loss in direct market, stigma) after their BSE case became public, they also recognised the importance of detecting the risk factors and sources.

The use of organophosphate products on Swiss dairy farms were investigated as they have been suspected (Purdey 1996) as potential risk factors for the occurrence of BSE in the UK. There was no significant association between the use of at least one of these products on dairy farms and the occurrence of BSE in Switzerland.

Calving practices on the farm were also investigated. Placental tissues from BSE- affected cattle have not yet been shown to transmit the infection in the experimental transmissions that have been attempted (Fraser and Foster, 1994). However, the investigation of risk factors for cases born after the introduction of the feed ban (Hoinville et al, 1995), revealed that animals born within a week of the calving date of a subsequently affected animal on the same farm showed an increased risk for the development of BSE. However, there was no evidence that the observed associations were modified by the location of calving or by the time of removal of

placental tissues from the calving accommodation. In the Swiss study also, the calving practice or methods of placenta disposal showed no relationship with the occurrence of BSE.

As recently described, maternal transmission could occur at the rate of one per cent. Preliminary results provide some, albeit limited, evidence that there is an enhanced risk of vertical transmission in the last six months of the BSE incubation period (MAFF, Press release, Aug.1996, Wilesmith et al.submitted). In Switzerland, so far none of the mothers of BSE animals were reported with BSE. Possible associations between the date of calving and the onset of BSE will be analysed at the individual animal level and reported elsewhere.

Concluding, the findings of this study indicated that at the exploratory, univariate level of analysis feed the consumption of any particular feed and the quantities used, could not be seen to be directly associated with BSE. However, the findings of this study point to the need for detailed information on the nature and source of ingredients used for the feed products used in case and control farms. An in-depth analysis among the different feed manufacturers has therefore launched which will allow comprehensive multivariate analyses of the complex interactions indicated by the present exploratory analysis.

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AN EMPIRICAL MODELLING APPROACH TO ESTIMATE THE ECONOMIC EFFECTS OF BSE FOR LIVESTOCK INDUSTRIES

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BSE has influenced demand and prices for beef and other meat in most countries of the EU considerably. There exists a large temptation to relate all price movements to BSE. However, as prices always fluctuate over time, only a part of total fluctuation can be imputed to BSE. This research attempts - on request of a Dutch farmers' journal (Dokter, 1996) - to better discriminate between what should be considered as 'normal' fluctuations and what is due to the BSE affair. Four major meat categories in The Netherlands are taken into account, viz. beef, veal, pork and chicken.

An econometric modelling approach (Pindyck and Rubinfield, 1981; Maddala, 1992) was developed to describe statistically the price behaviour before the BSE affair, using product-specific explanatory variables (such as amount of supply, prices abroad and contagious disease outbreaks) as well as cross price relations (e.g. the price of pork and poultry to explain the price of beef) and a seasonal- and trend correction. Parameters were estimated based on data from 1988 or 1990 until February 1995. Data from February 1995 till February 1996 were used to validate the model. Predicted values from the models were compared with the actual monthly data. Results of the validation showed that for most products it was possible to closely simulate the actual values. Finally the best fitting model for each sector was re-estimated over a period until February 1996. These models were used to predict the prices in `business as usual` scenario for the first and most serious period of the BSE affair, i.e., from March till July 1996[3]. These predictions were compared with actual values to determine more precisely the BSE-related price effects.

1. METHOD

Estimations, for different categories, are carried out over a period depending on the maximum available data. Each equation is estimated over the period equal to the most limited data. Endogenous variables, meat prices, are explained by variables specific to each sector and some general variables. Examples of specific variables are supply and demand of meat and prices in the related markets while trend and seasonal correction are more general explanatory variables. Main explanatory variables in each estimation are shown below.

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[3] July was most recently available month at the time this study was conducted. When time passes, more recent months can be included.

- Beef sector
  - price of beef in the UK
  - average price of beef of main trading partners of The Netherlands
  - supply of beef in The Netherlands
  - price of pigs in The Netherlands
  - price of chicken in The Netherlands
  - trend
  - monthly seasonal correction
- Veal sector
  - average price of veal of main trading partners of The Netherlands
  - export demand of white and pink veal in The Netherlands
  - Dutch import of calves for fattening
  - year correction for '90/'91 (large imports from Eastern Europe)
  - correction for new situation in the veal market in 1995
  - trend
  - monthly seasonal correction
- Pork sector
  - supply in The Netherlands
  - price of pork in Germany
  - price of pork in Belgium
  - price of fodder
  - animal disease outbreaks
  - trend
  - monthly seasonal correction
- Poultry sector
  - supply in The Netherlands
  - trend
  - monthly seasonal correction

To get a complete data set several different sources are used. Definitions and sources of variables used are given in notations section.

Prices are corrected for inflation and equations are estimated over a minimum period of five years. When necessary, correction for autocorrelation of disturbances has been carried out.

2. BSE EFFECTS IN THE BEEF SECTOR

Beef price is estimated using monthly data from January 1990 with an explained fraction of variance of 0.97. Estimation results are given in table 1. Beef prices abroad positively influence Dutch beef price while the prices of chicken and pork have a negative influence but are not significant at the 10 percent level. Likewise, trend and Dutch supply of beef are not found to be significant. Amongst the seasonal dummies, only March up to July are significant and positive meaning that beef prices in those months are higher on average (approximately Dfl. 60 in March) than in December which is the reference month.

Table 1. Estimation results for price of beef (Dfl./carcass, 1990-1996)

Variable	Estimate	Standard error	t-value
Constant	0.390	466.47	0.00
Price UK.	0.096	0.07	1.39
Price B/G.[a]	1.099	0.14	7.95
Price pork	-0.124	0.22	-0.56
Price chicken	-217.982	203.05	-1.07
Trend	0.474	1.85	0.26
Supply	-0.001	0.00	-1.24
January	-7.937	17.24	-0.46
February	25.760	21.99	1.17
March[a]	60.016	22.39	2.68
April[a]	58.461	25.05	2.33
May[a]	68.883	27.26	2.53
June[a]	66.008	31.39	2.10
July	39.370	34.51	1.14
August	28.443	28.96	0.98
September	1.454	25.66	0.06
October	-31.046	23.66	-1.31
November	-29.243	19.63	-1.49

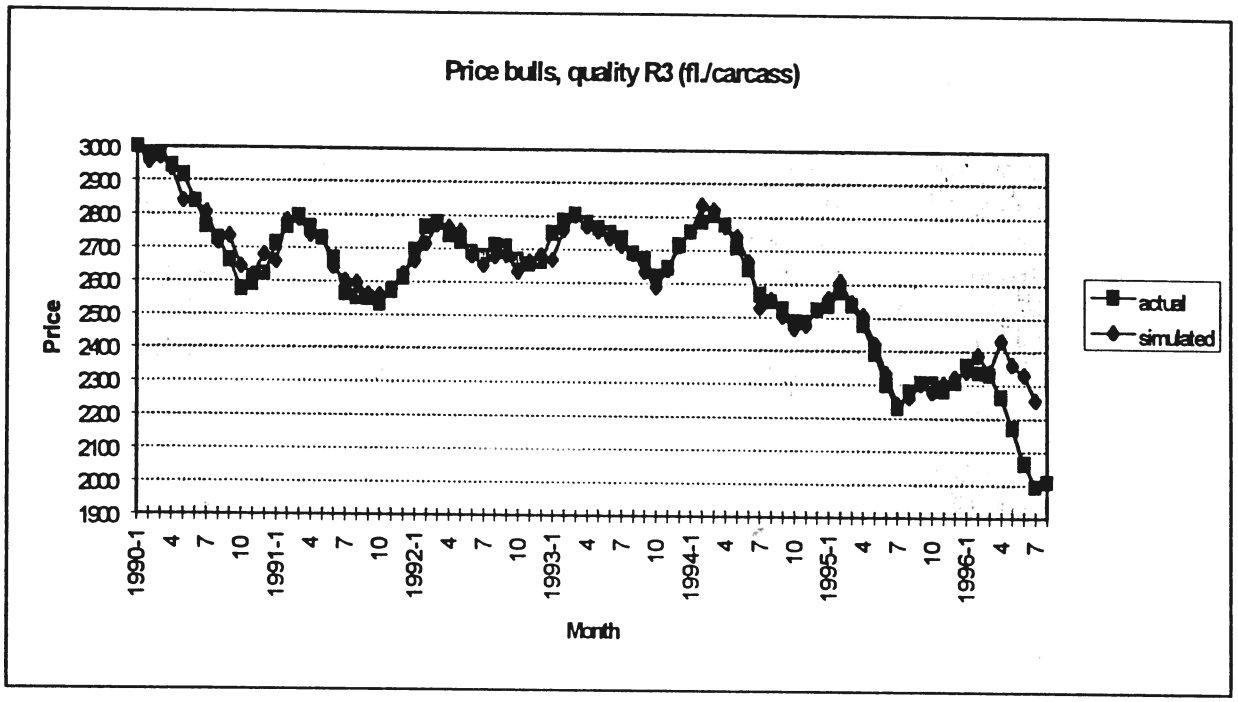
[a] Significant at the 10 per cent level

Since there are many different qualities of beef, beef quality R3 was chosen as the representative quality. Prices of different qualities vary in absolute level but tend to move together over time. Since R3 is the largest category, its price was used as the dependent variable in the beef price equation.

Figure 1 shows that until '94-'95 the price of beef was relatively stable around an average of approximately 2700 guilders per carcass and subsequently decreased. This development has been taken into account when calculating BSE-effects on the beef sector.

Beef prices decreased almost immediately after the beginning of the BSE crisis in March 1996 as can be seen from the actual prices in Fig. 1. The actual beef price ws nearly 12 per cent below the simulated beef price by July 1996.

Fig. 1 Actual and simulated price of beef quality R3



For an indication of the total damage to the beef sector, the price difference between actual and simulated price can be multiplied by the number of slaughterings in The Netherlands. The cumulative loss up to the end of July '96 was about 81 million guilders. It is assumed that the real number of slaughterings does not differ from what would have happened without this new concern about BSE. However, it might be possible that some slaughterings were postponed to a later month.

It must be taken into account that the loss is calculated using an average representative price (quality R3) and that total slaughterings are multiplied by this single price. Justification can be found in the fact that quality R3 is about average and the largest category by volume amongst all meat qualities.

3. EFFECTS ON THE VEAL SECTOR

The results in this sector must be treated with caution. The preceding year, before the BSE crisis, major changes took place in the veal sector. In particular, there was a strong increase in the concentration in the veal market. Furthermore, from March 1995 it was necessary to use another data set which may have caused data inconsistencies. Both facts could have influenced the price development from early 1995, see Fig. 2.

Coefficients in the estimation were allowed to differ before and after March 1995 to capture data discontinuity. The price of veal is estimated with an R-squared of 0.95 and important significant coefficients are: price of veal calves abroad, trend, imports of young calves and some monthly seasonal correction. Price of veal calves abroad positively influences Dutch veal

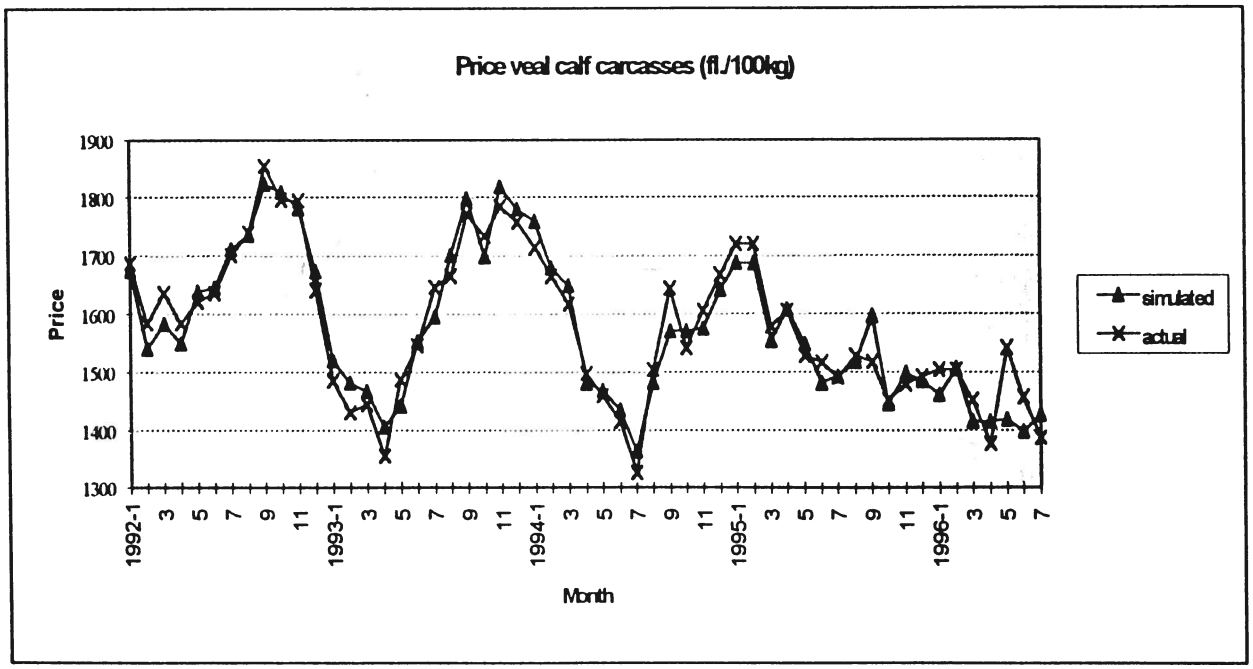
prices while imports of young calves have a negative influence. Trend has a positive influence on Dutch veal price before 1995 but negative afterwards. Prices of veal calves from March until June are significantly lower than in the reference month December.

The simulated price, showing what would have happened without the BSE crisis, reveals a gradual decline until the end of the simulation period. However, BSE has clearly brought some market disturbances into the veal market, see Fig. 2.

The removal from the market, necessary to regain consumer trust, of 60,000 calves imported from Britain is thought to have contributed to price increases in May and June in the veal sector due to the decrease in the supply of veal. The European Community has also tried to remove veal temporarily from the market by subsidising private storage. Contracts were made from May to June and The Netherlands strongly took advantage of this opportunity, 7,600 tons in The Netherlands compared to 11,500 tons for the EU as a whole. Stored veal is subsidised temporarily and starting in August, is slowly entering the market again which will probably decrease the actual price of veal.

In conclusion, until July there is no straightforward effect of the BSE crisis on the veal sector as real prices alternate below or above the simulated price, varying between approximately -3 to +9 per cent. The veal sector marginally gains from the BSE crisis but this positive effect will probably be negatively influenced after August when veal from private storage begins entering the market again.

Fig. 2 Actual and simulated price of veal



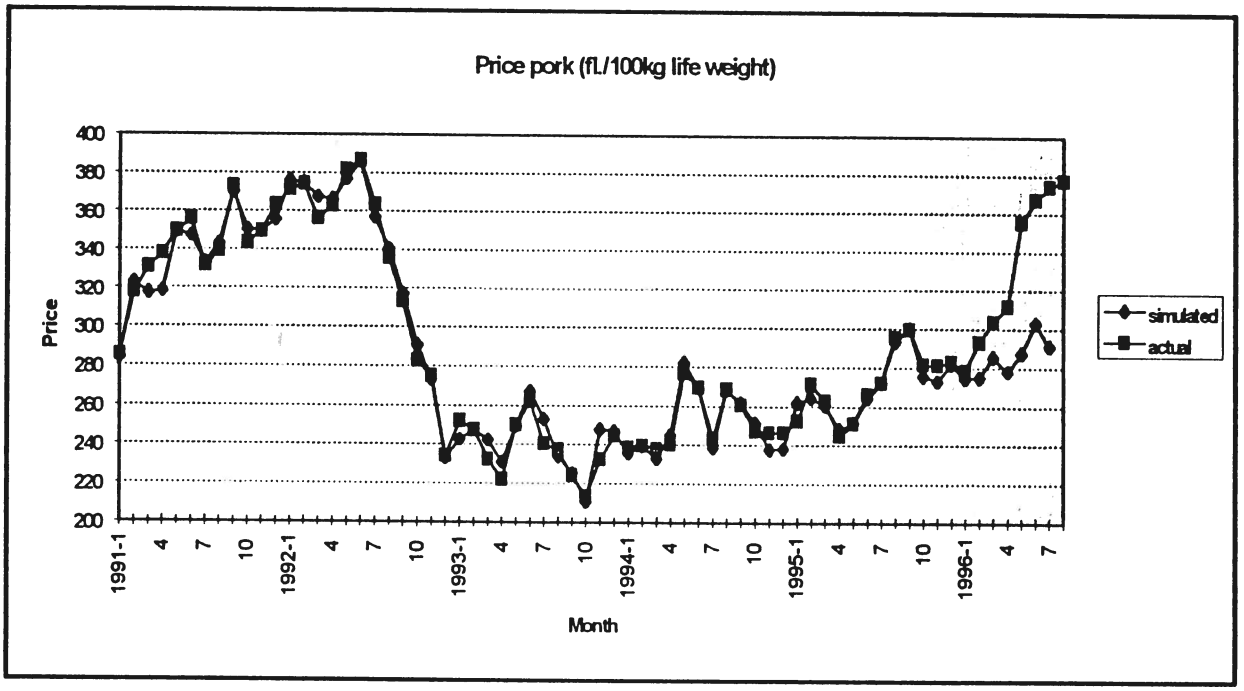
4. EFFECT ON THE PORK SECTOR

The price of pork is well estimated with an explained fraction of total variance of 0.99. Supply on the Dutch pork market has a negative influence on the price of pork while it is positively influenced by the German price of pork. Both the price of feed and monthly trend negatively influence the Dutch price of pork. Pork is more expensive in January, May and June than in the reference month December according to significant dummy corrections of those months.

Figure 3 shows that the price of pork (per 100 kg of live weight) decreased by almost 35 per cent in mid-1992 and subsequently stabilised around this relatively low level. From 1995 the actual pig price shows a gradual increase, probably due to a lower supply and persistently low margins in the pig sector. Starting from the BSE crisis the pork price experienced an extra boost upwards. Total price increases must not be fully attributed to the BSE crisis, a certain part of that price increase also would have happened without the crisis.

Taking into account the present increasing trend from 1995, the actual price of pork has experienced a strong extra increase due to the BSE crisis until July. The initial low price increase of 3.6 per cent in March had become a price increase of about 25 per cent by July, compared to the situation without the BSE scare (simulated price), see Fig. 3.

Fig. 3 Actual and simulated prices of pork



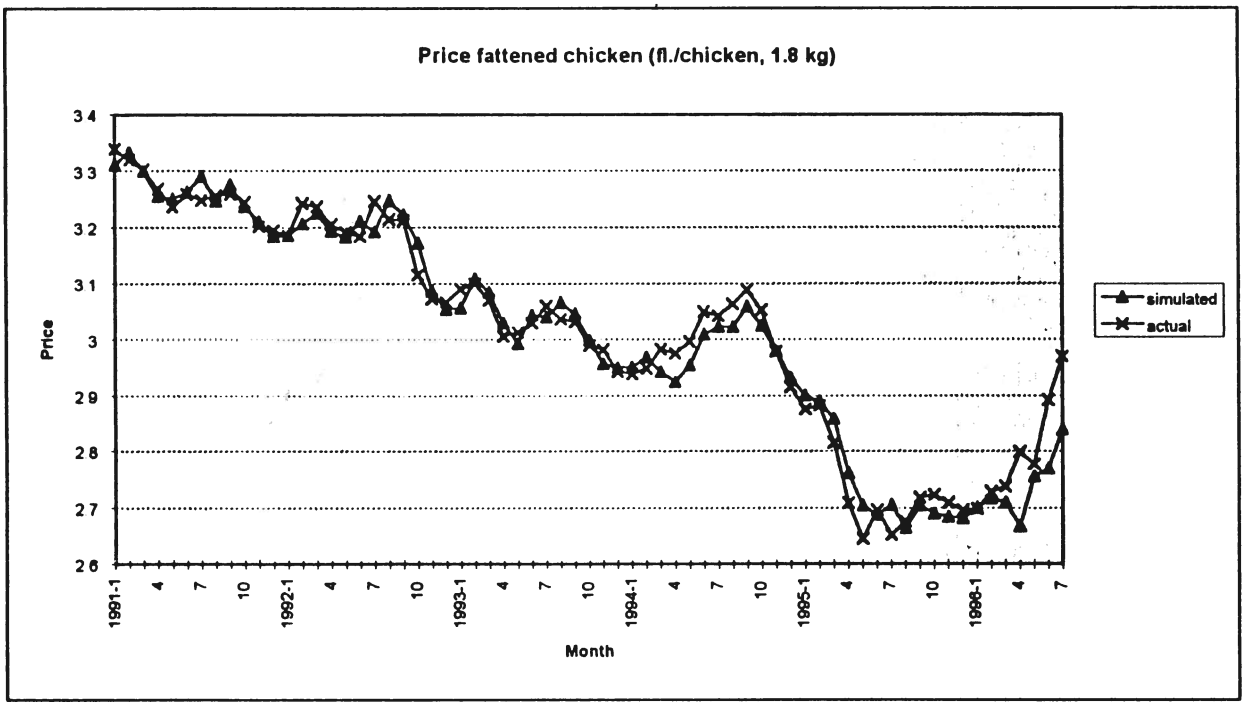
5. EFFECTS ON THE POULTRY SECTOR

Until mid 1994 the price of chicken showed a gradual decline followed by a large drop, and finally stabilised, see Fig. 4. The estimated equation has an R-squared of 0.98 in which trend has a negative significant influence and coefficients for February, August and September (monthly seasonal corrections) are positive.

For some months before the BSE crisis, price was stable at around 2.70 guilders per chicken. The BSE crisis caused a price increase above what would be expected without this crisis. Although differences between actual and simulated vary between subsequent months the actual price is on average 4 per cent higher than the simulated price.

Until July total extra earnings in poultry can be calculated by multiplying the price difference by the number of chickens produced. Of the four meats studied here, poultry has the shortest supply lag. Although the assumption of no BSE-induced supply change must be abandoned in the medium to long run for all four meats, the delay is shortest for poultry. Before the end of 1996, one would expect the BSE crisis to produce a quantity effect on chicken supply as well as a price effect.

Fig. 4 Actual and simulated prices of fattened chicken



6. CONCLUSION

Meat prices in different sectors can be well explained ($R^2 \geq 0.95$) by the models specified. Prices which would have happened without the BSE crisis are simulated using the best fitting model on the data up to February 1996. Initially all equations were tested for their forecasting ability.

The method used provides insight into most important explanatory variables of meat prices in different sectors. At the time this study was conducted, no other research had attempted to compare prices before and after the BSE crisis in this way.

This analysis allows to directly measure the effects on price, thereby making difficult estimations of changes in consumer demand or confidence unnecessary. This permits the calculation of revenue changes on the producer side only, due to the BSE crisis. Changes in the market due to BSE finally results in actual price changes and after comparing actual price and price simulated on the assumption that BSE did not happen yields the total BSE effecton price.

The total effect of the BSE crisis until July 1996 on Dutch main meat sectors is about 390 million guilders and is composed of:

- beef sector -80.9 million
- veal sector 15.2 million
- pork sector 393.6 million
- poultry sector 16.5 million

These effects, plus the compensation of about 46.5 million guilders paid by the European Union to support the beef sector results in extra earnings of roughly 390 million guilder. The largest loser until July 1996 is the beef sector while after July 1996 also the veal sector might lose as subsidised private stored meat will enter the market again. Until July the pork sector has the largest extra earnings and as this sector is relatively large in The Netherlands, this effect dominates the aggregate result. The poultry sector also has a positive revenue effect from BSE crisis.

The European subsidy for compulsorily killing and withdrawing from the market 60,000 calves of UK origin has not been taken into account as this compensation just covers the costs (losses) for producers.

Financial damage to the export of live breeding animals and young calfs sales from dairy farmers are not studied as these sectors are not primarily meat producers. However, these sectors have certainly suffered from the BSE crisis.

This study shows that the effects of the BSE crisis on Dutch meat producers can be quantified reliably in the short term. When important supply changes occur in the future these need also be analysed. Furthermore, this modelling approach can also be applied to other countries and conditions if data are available.

NOTATION:

Variables used in estimation:

N°	Source	Definition
1	Eurostat	Price/100kg dead weight, bulls (R3, >220kg) F, NL, G, UK
2	PVE	Supply beef (x1,000) in NL (slaughterings, im-/export)
3	WAU	Trend (monthly)
4	WAU	Monthly seasonal correction (dummy, reference=December)
5	Eurostat/PVE	Price calf carcasses guilders/100kg in F, I, B, NL
6	PVE	Export Dutch veal calves (white/pink)
7	PVE	Dutch imports young calves
8	WAU	Year correction `90/`91 (dummy)
9	WAU	Coefficient correction, through dummies, for modified market conditions in veal market from 1995
10	PVE	Price pigs/100 kg, live weight in NL
11	PVE	Dutch supply pigs (slaughterings, im-/export)
12	PVE	Price pigs Belgium
13	PVE	Price pigs Germany
14	LEI	Price sow standard sow concentrates(cents/kg)
15	RVV	Dummy for animal disease outbreak (Classical Swine Fever and Swine Vescular Disease)
16	PVE	Price chicken (Dfl./kg live weight)
17	PVE	Dutch supply chicken (x1,000 kg live weight)

Eurostat: European Statistical Service
PVE: Product Boards for Livestock, Meat and Eggs
LEI: Agricultural Economics Institute
RVV: Inspection for Livestock and Meat
WAU: Wageningen Agricultural University

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A NEW VARIANT OF CREUTZFELDT-JAKOB DISEASE IN THE UK

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Despite extensive epidemiological research, the cause of sporadic CJD is unknown and, in particular, there is no good evidence of a causal link with scrapie in sheep or goats (Brown et al., 1987). CJD occurs worldwide with a relatively constant incidence (Will, 1996), including countries that are free of scrapie such as Australia and New Zealand. Case control studies have failed to show any consistent environmental risk factor for CJD (Wientjens et al., 1996) and the rarity and the apparently random spatio-temporal distribution of cases indicates that case-to-case transmission is an unlikely explanation for sporadic cases of CJD (Will, 1993). Rarely, CJD has been transmitted through medical treatment or neurosurgical instruments (Brown et al., 1992) and about 10% of cases are associated with mutations of the prion protein gene (Windl et al., 1996). The incubation period is not known in sporadic cases, but in iatrogenic cases related to peripheral inoculation of human pituitary hormones the mean incubation period is about 13 years (Brown et al., 1992). In kuru, in which transmission from case to case was related to ritual cannibalism, the mean incubation period is not known but ranges from 4.5 to over 30 years.

Systematic national surveillance of Creutzfeldt-Jakob disease (CJD) was reinstituted in the UK in May 1990 following the epidemic of bovine spongiform encephalopathy (BSE) in the UK cattle population. The primary aim of surveillance has been to identify any change in the characteristics of CJD that might be attributable to transmission of BSE to the human population, although the possibility of such cross-species transmission had been judged to be remote (Southwood Committee, 1989).

Since 1990, surveillance of CJD has depended on direct referral of suspect cases from neurologists and neurophysiologists and identification of confirmed cases by neuropathologists. A number of changes in the characteristics of CJD have been identified. The number of cases per annum has approximately doubled in the 1990s in comparison with the 1980s, and this is largely due to an increase in the number of cases of CJD in the elderly (Will, 1996). The total numbers of cases of CJD in the UK in 1995 and 1996 are lower than the peak year of 1994. Four farmers have been identified with CJD who have had possible contact with BSE affected cattle. Since 1993 the surveillance of CJD has been harmonised in a number of European countries, including France, Germany, Italy, the Netherlands and the UK in order to allow comparison of data on CJD in countries with widely different incidence of BSE. Comparisons of the incidence of CJD between 1993-1995 and of the frequency of CJD in individuals employed in farming has demonstrated no excess in the UK in comparison with other countries (Alperovitch et al., 1994, Delasnerie-Laupretre et al., 1995). Up until late 1995 there was no convincing evidence of a change in CJD in the UK that might be related to BSE.

In November 1995 details of two cases of CJD, already known to the Unit, were published (Britton et al., 1995, Bateman et al., 1995). These cases were of particular concern because both were teenagers and only four cases of sporadic CJD in adolescents had been identified previously,

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although all were from countries where there could be no link with BSE. By March 1996, 10 cases of CJD in younger patients had been identified in the UK, with a median age of 29 years in comparison to a median age of 65 years in previous cases (Will et al., 1996). The clinical features in these cases were unusual for CJD with a prolonged duration of illness (median 12 months), prominent early psychiatric features, and persistent sensory disturbance in some cases. In contrast to classical CJD in which a "typical" EEG appearance is seen in about 60-70% of cases, the EEG was diagnostic in none of these cases. Crucially the neuropathological findings, although confirming the diagnosis of CJD, were unlike any previous case of CJD examined by the CJD Surveillance Unit. All cases exhibited widespread plaque formation throughout the cerebrum and cerebellum with smaller numbers in the basal ganglia, thalamus and hypothalamus. The morphology of the plaques was unusual with a dense core surrounded by a halo of spongiform change reminiscent of "florid" plaques sometimes seen in scrapie. The neuropathological appearances were thought to be novel and subsequent to the publication of these cases no previous case of CJD with similar appearances has been identified despite review of cases of CJD in Europe and elsewhere.

The occurrence of a novel form of CJD in a country with a high incidence of a potentially new risk factor for human prion disease (BSE) raised the possibility of a causal link between animal and human forms of prion disease. However other explanations for this 'cluster' of cases had to be considered. One early hypothesis was that these cases might represent a genetic form of CJD, but full sequencing of the prion protein gene in 8 cases excluded any of the mutations known to be associated with familial forms of human prion disease. All cases were homozygous for methionine at codon129 of the prion protein gene in comparison with about 80% methionine homozygosity in classical CJD (Windl et al., 1996). Description of the neuropathology in 14 published cases of CJD in patients aged less than 30 outside the UK did not suggest similar neuropathological changes to new variant CJD. The pathological appearances in a number of previous young cases of CJD in the UK, including a 16 year-old dying in 1980, were distinct from the new form despite processing and staining using similar techniques to the new cases. This evidence largely excluded the possibility that these cases were simply age related or that the neuropathological changes simply reflected the use of modern techniques. Ascertainment bias was carefully considered but was judged to be an unlikely explanation for this 'cluster' of cases not least because similar biases were likely to have influenced ascertainment in other European countries, but no similar cases had been identified in these countries. No case had a history of potential iatrogenic exposure and there was no evidence of common dietary or occupational exposure. In conclusion, a novel form of CJD had been identified in the UK and in the absence of any other explanation, it was judged that a causal link with BSE was the most plausible explanation for the occurrence of these cases.

Since the publication in April 1996 describing 10 cases of nvCJD, 4 further confirmed cases and one "probable" have been identified in the UK and one confirmed case in France. The clinicopathological phenotype in these cases is consistent with the original description, although one patient in the UK died at the age of 50 years extending the age range of nvCJD. The small numbers of cases identified since April 1996 has led to speculation that there are unlikely to be a significant number of subsequent cases even if there is a causal link with BSE. However mathematical modelling has indicated that there are a wide range of possible future scenarios and that it may be four or more years before the epidemiological evidence may lead to more accurate projections (Cousens et al., 1996). The crucial determinant to the future numbers of cases is the length and spread of the incubation period and extrapolation from the incubation period in iatrogenic CJD may be misleading because of the absence of a species barrier.

A causal link between nvCJD and BSE was suggested by the occurrence of a novel form of CJD in a country with a high incidence of a potentially new risk factor for human disease. Since the original description of nvCJD, no historical case with the similar neuropathological profile has been identified despite the review of a large number of cases of CJD, for example through the BIOMED1

programme for coordination of neuropathological analysis of CJD in the EU (coordinator Professor H Budka). On current evidence, nvCJD is a novel condition.

Through the BIOMED1 funded project for the coordination of surveillance for CJD in 5 Member States of the EU, it was possible in early 1996 to confirm that this form of CJD was not occurring in countries with a low potential exposure to the BSE agent and this comparative data was crucial to the hypothesis of a causal link. A case of nvCJD has subsequently been identified in France but it is known that food products, meat and bonemeal, and cattle incubating BSE may have been exported to France in the 1980s. Recent studies have provided supportive evidence for a causal link between BSE and nvCJD. The neuropathological profile in macaque monkeys inoculated with BSE is similar to nvCJD (Lasmézas et al., 1996) and analysis of protein subtypes in nvCJD has shown a pattern distinct from other forms of CJD and similar to protein subtypes found in BSE (Collinge et al., 1996). It is essential to extend this work by examining larger numbers and subtypes of CJD cases and to obtain comparative data from scrapie, but the balance of scientific evidence is consistent with the hypothesis that nvCJD is due to exposure to the BSE agent, presumptively through contaminated food products.

The incubation period in prion diseases is prolonged, often extending to many years, and is determined by a number of factors including the route of exposure, the amount of infectivity and the species barrier in cross species transmission. Although the incubation period in kuru may extend to 30 years or more, it is difficult to estimate the mean incubation period because it is only in rare cases that the date of a single exposure event can be defined, because in the majority of kuru victims there had been multiple contaminating events over many years. In iatrogenic CJD due to treatment with human pituitary-derived hormones the best estimate of incubation period is about 12-13 years (although cases with longer incubation periods may still occur) and the shortest incubation period has been 4.5 years. If BSE is transmitted to the human population by the oral route, it is likely that the incubation period would be at least as long as in iatrogenic CJD due to peripheral cross contamination and may well be longer because of transmission across a species barrier. If so, it is likely that cases of nvCJD reflect exposures that took place many years ago, probably in the 1980s. The amount of BSE infectivity that might have entered the human food chain at that time is not known but transmission studies have demonstrated infectivity in natural BSE in brain, spinal cord and retina and not in other tissues. Prior to 1989 when legal measures to exclude brain and spinal cord (and other tissues) from the human food chain were introduced, it is probable that the human population in the UK were exposed to high titres of infectivity for example through mechanically recovered meat. The extent of such exposure is not known but mathematical modelling has indicated that large numbers of cattle incubating CJD may have been slaughtered and consumed in the late 1980s (Anderson et al., 1996).

A causal link between BSE and nvCJD has not been proven but further evidence may come from laboratory transmission studies and continued epidemiological research, including comparisons of data from the UK and other countries. Such evidence may take years to obtain and in the interim it is essential to ensure the strict application of all legal measures to minimise current human exposure to the BSE agent, although the risks from BSE in the UK are now probably less than at any time since the early stages of the cattle epidemic.

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A STOCHASTIC APPROACH TO MODELLING BSE IN UK DAIRY HERDS

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It is now 10 years since the first case of BSE was reported. There have been more than 160,000 clinical cases on over 33,000 different farms. About 60 % of all dairy herds have had a least one clinical case of BSE whilst less than 20% of beef suckler herds have had a case (MAFF, 1996). Closer examination of these statistics indicates that about 95 % of all clinical cases were born on a dairy farm.

Study of individual dairy farm records of BSE incidence using DAISY herd data has indicated that there is significant variation in BSE clinical incidence rates between farms. Individual reports from farmers suggest that some of the first dairy farms to experience cases of BSE in 1986 went on to have a disproportionate number of cases in the following years. There has also been regional variation in BSE incidence which has been attributed to differences in the source of meat and bone meal (Wilesmith *et al.*, 1991).

The recent report that maternal transmission may occur in some cases (Anderson *et al.*, 1996) complicates any attempt to determine the future course of the BSE epidemic. The long incubation period of the disease, our limited understanding of maternal transmission rates and the low incidence of the disease in the cattle population make it difficult to predict when the last case of clinical BSE will occur.

Predictions of the future incidence of BSE and evaluation of control measures have previously been carried out using mathematical modelling techniques (Richards *et al.*, 1993; Stekel *et al.*, 1996; Anderson *et al.*, 1996). However, these predictions have been based on national models of the disease dynamics in the UK which do not consider the between-gherd variation in the clinical incidence of BSE. Anderson *et al* (1996) used such a model to make some preliminary predictions on the likely impact of a number of alternative control strategies.

This paper describes a model developed to simulate the dynamics of BSE at the individual herd level. The model is based on a stochastic herd model which enables the effect of chance on disease transmission to be demonstrated. This model also has the advantage that it can trace individual cattle and their offspring over the modelling period enabling a closer analysis of the significance of maternal transmission. The model has been used in this example to describe a typical UK dairy herd with clinical cases of BSE.

MATERIALS AND METHODS

Description of the population model

A stochastic herd model was first developed to describe population dynamics in a UK dairy herd. This model was based on previous herd models that have been developed by McLeod (1993) and Rushton (1996). Production parameters used in the model were taken from published data calculated from DAISY

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dairy herds (Kossaibati & Esslemont, 1996). These herds can be considered to be representative of intensive dairy production in the South of England.

The model was developed in Microsoft– Access– Basic 2.0 The herd model simulates the population turnover in a typical dairy cow herd with the individual animal being the unit of modelling and one day being the unit of time. It was designed to run a large number of simulations for each set of parameters from the year 1987 until 2015. Herd and disease predictions were then validated against real field data for the first 10 year period.

A simplified structure of the herd model is provided in Fig. 1. The model is able to simulate the effect of seasonal factors and management decisions on herd structure and output. It is equally relevant to both dairy and beef production systems and can also be used to simulate regional differences in herd dynamics. However, for the sake of simplicity, only the results of a typical UK dairy herd simulation are presented in this paper.

The model was initially run for 12 years starting with 20 six-month-old heifers using defined production parameters in order to generate the initial herd structure in 1987. The maximum herd size was limited to 80 adult dairy cattle and 40 replacement heifers. Adult cows were culled when heifers calved and entered the breeding herd. Surplus heifers and all male calves were assumed to be sold at birth.

Description of the disease model

The structure of the disease model is shown in Fig. 2. This disease model was superimposed upon the population model to describe a UK dairy herd infected with BSE. The stochastic nature of the model means that cows that develop disease within the herd are randomly selected given the age and year specific probability of disease occurring. Once the model has determined that a cow has clinical BSE, it works backwards to determine, using the Monte Carlo method, whether any of the cow's calves will also be infected.

The model uses incidence of clinical disease rather than infection rates as the main parameter to describe the disease epidemic. This is representative of the real field situation where the lack of a diagnostic tests for pre-clinical BSE means that clinical cases are used as the main indicator of infection. Reported clinical incidence figures for UK dairy cattle were used for the period 1987 to 1995 (Wilesmith, J., pers. comm.).

Least squares linear regression was then used to predict clinical incidence rates based on the assumption that the future age distribution of cases will follow a similar pattern to those observed in the 1987 birth cohort. The expression used year of birth and age of animal as independent variables with an r^2 value of 0.9848 (Equation 1). Projected figures from 1996 onwards are shown in italics in Table 1.

$$y = \exp(1.82799 + \beta 1 + \beta 2) \qquad \text{Eq. (1)}$$

Where :

y= age specific incidence rate
$\beta 1$ = coefficient for year of birth
$\beta 2$ = coefficient for age

In the model it was assumed that no new feed-based infection of cattle took place after 1993 due to the effective implementation of the meat and bone meal ban for ruminant feeds. Therefore any new infections from 1994 onwards were likely to be maternally derived. In order to estimate the number of these new maternal cases, a number of assumptions were made about the potential of an infected dam to infect her calf. In the original scenario, it was assumed that no maternal transmission took place (Table 2). Subsequent scenarios simulated the effect of the percentage of successful infections of calves that took place during the infective period. The lack of any reliable estimates of maternal transmission rates meant that these scenarios were selected arbitrarily using the work of Anderson *et al* (1996) as a guide.

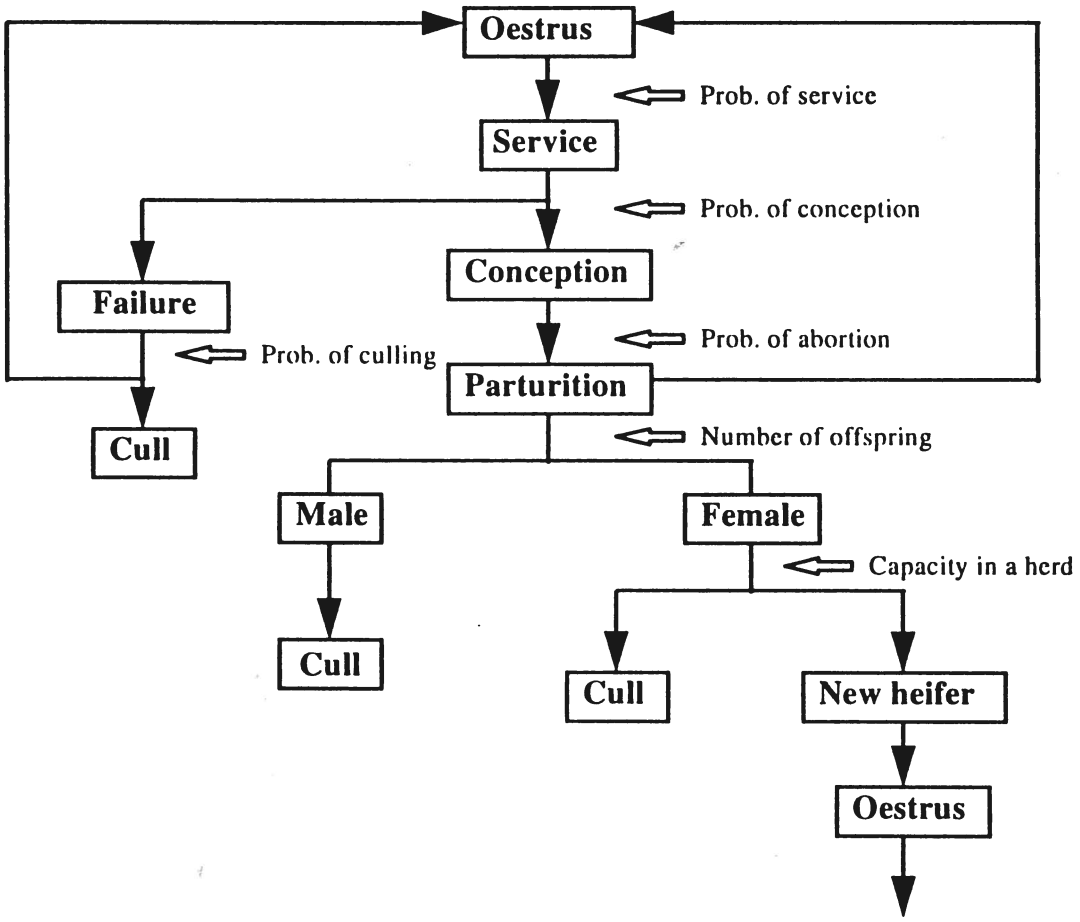

Fig. 1 Structure of herd model

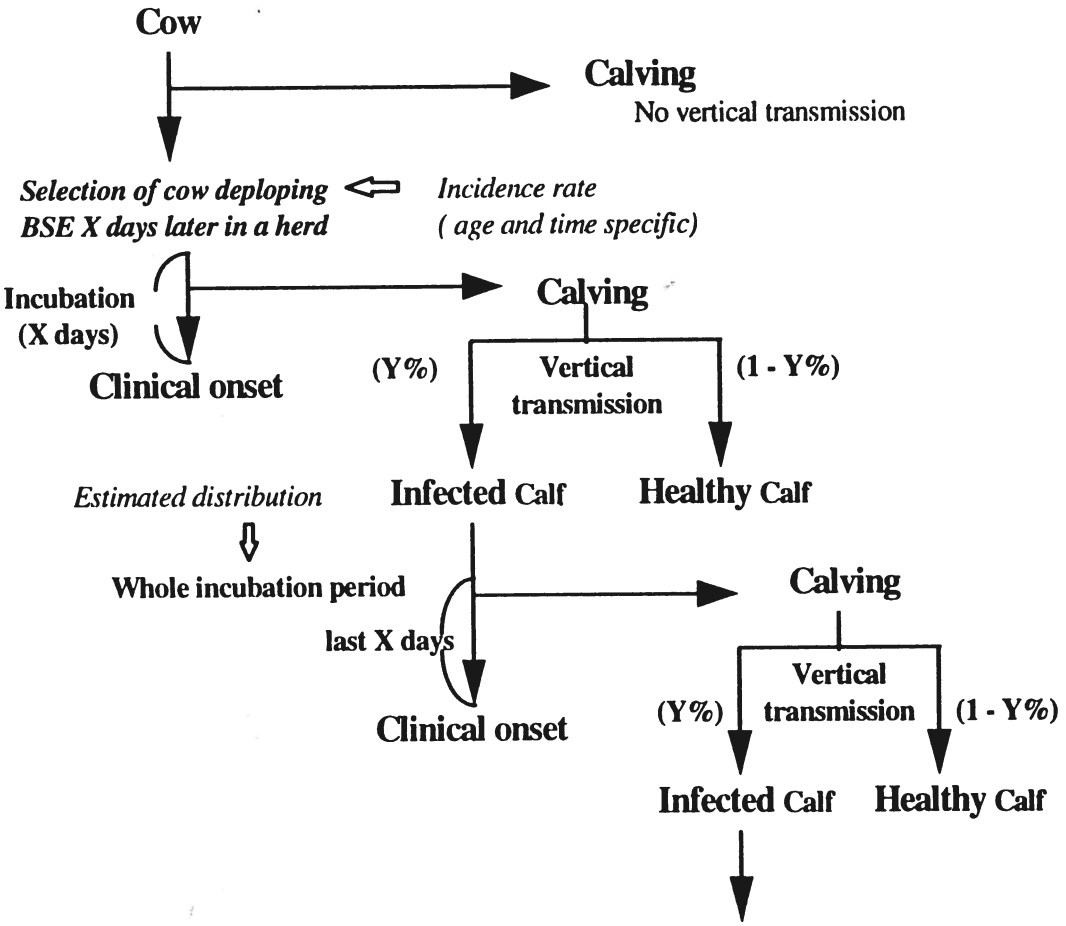

Fig. 2 Structure of disease model

Table 1 Age-specific BSE clinical incidence by year (%)

Age	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005
0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00										
1	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00										
2	0.09	0.06	0.06	0.08	0.03	0.02	0.01	0.01	0.01										
3	1.10	0.85	0.81	1.36	2.01	0.66	0.48	0.26	0.30	0.21									
4	2.60	3.02	3.21	3.86	5.74	6.82	3.40	2.30	1.40	1.06	0.90								
5	1.51	2.50	3.34	3.33	3.86	6.43	8.14	4.57	3.58	1.28	1.23	1.04							
6	0.35	0.84	1.54	1.95	1.89	2.70	4.95	5.84	3.54	1.60	0.79	0.76	0.64						
7	0.35	0.26	0.39	0.73	0.92	1.03	1.64	2.97	3.43	1.39	0.91	0.45	0.43	0.36					
8	0.00	0.09	0.16	0.18	0.35	0.60	0.75	1.17	1.50	1.60	0.72	0.47	0.23	0.22	0.19				
9	0.08	0.07	0.06	0.08	0.19	0.33	0.46	0.66	0.71	0.96	1.18	0.53	0.35	0.17	0.16	0.14			
10	0.00	0.07	0.02	0.06	0.08	0.08	0.35	0.53	0.45	0.53	0.79	0.98	0.44	0.29	0.14	0.14	0.11		
11	0.00	0.09	0.04	0.06	0.08	0.09	0.18	0.35	0.42	0.56	0.65	0.97	1.19	0.54	0.35	0.17	0.17	0.14	
12	0.00	0.00	0.03	0.02	0.06	0.09	0.07	0.14	0.35	0.45	0.52	0.61	0.91	1.12	0.51	0.33	0.16	0.16	0.13

Table 2 Maternal transmission scenarios

Scenario	Infective period during which dam can infect calf prior to developing clinical disease (days)	Percentage of successful infections of calf during infective period (%)
1	0	0
2	365	10
3	365	50
4	365	100

Table 3 Incubation period distribution by age

Year	0	1	2	3	4	5	6	7	8	9	10
Probability	0.00%	0.01%	3.80%	17.70%	25.40%	21.00%	13.70%	8.10%	4.60%	2.50%	1.40%

The model was run 100 times for each scenario to determine the number of maternal infections that could be expected in a typical herd. It was then possible to estimate if and when maternally infected calves would develop clinical disease. This was dependent on the incubation period distribution (Table 3) and the age specific culling rate. Incubation periods were derived by back calculation using national BSE data. Culling rates were taken from published DAISY data (Kossaibati & Esslemont, 1996).

RESULTS

The results of running the herd model 500 times without infection are shown in Fig. 3 with 95% confidence intervals marked. Data collected from DAISY and Holstein Friesan Society dairy herds are superimposed on this figure. The simulated herd age structure appears to fit quite well to the real situation observed in typical intensive dairy herds. In the simulation it was also noted that the herd structure quickly reached a state of equilibrium.

Figure 4 shows the total number of clinical BSE cases that the model predicted in 500 simulations under scenario 1 (no maternal transmission). This is equivalent to the number of clinical cases that would be expected in 500 herds or about 50,000 cattle. From this information it is possible to calculate clinical incidence rates. The predicted incidence rates for 1990 to 1995 have been compared with true incidence rates calculated from the MAFF data given in Table 1. In addition, to provide a different data source to validate the model, BSE clinical incidence rates were calculated for 15 DAISY herds which had experienced at least one case of BSE previously (Fig. 5).

The model was then run 100 times under each scenario to predict the influence of different maternal transmission rates on the future course of the epidemic. Figures 6a, b, c and d indicate when each of the 100 simulated herds were predicted to have had their last clinical case of BSE.

Using these projections it was then possible to calculate the probability that no further clinical cases would occur in any of the 100 herds by a specified year (Fig. 7). These results provide an indication of the time required for BSE to be eradicated in the National dairy herd. However, it is possible that additional simulations would extend the time that clinical disease could be detected and thus reduce the probability of disease eradication by a specified date.

The results presented here are a summary of simulations conducted to date. It is intended to continue this work with a larger number of simulations for each scenario and also to model the effect of different control strategies on the predicted course of the epidemic. This further research will take some time to complete given the fact that each simulation requires about 20 minutes of computer time on a Pentium 120 MHz computer.

DISCUSSION

Disease modelling can provide a means of testing current hypotheses about the dynamics of a disease in a population and also help to predict the future course of the disease. In the case of BSE, mathematical models could be useful in developing our understanding of the mechanisms of transmission. Predictive modelling of BSE incidence has also acquired a political significance given the pressure to completely eradicate the disease from the UK as quickly as possible.

The population model described here provides a tool to investigate disease dynamics within individual herds. The relevance of this model to the real situation is influenced by the validity of the relationships defined in the model and the accuracy of standard production parameters used. The basic output of the model fits closely with data available from the Holstein Friesan Society and DAISY herds.

Validation of the disease component of the model is rather more difficult. Comparison of clinical incidence rates predicted by the model with observed incidence rates reported by MAFF for UK dairy

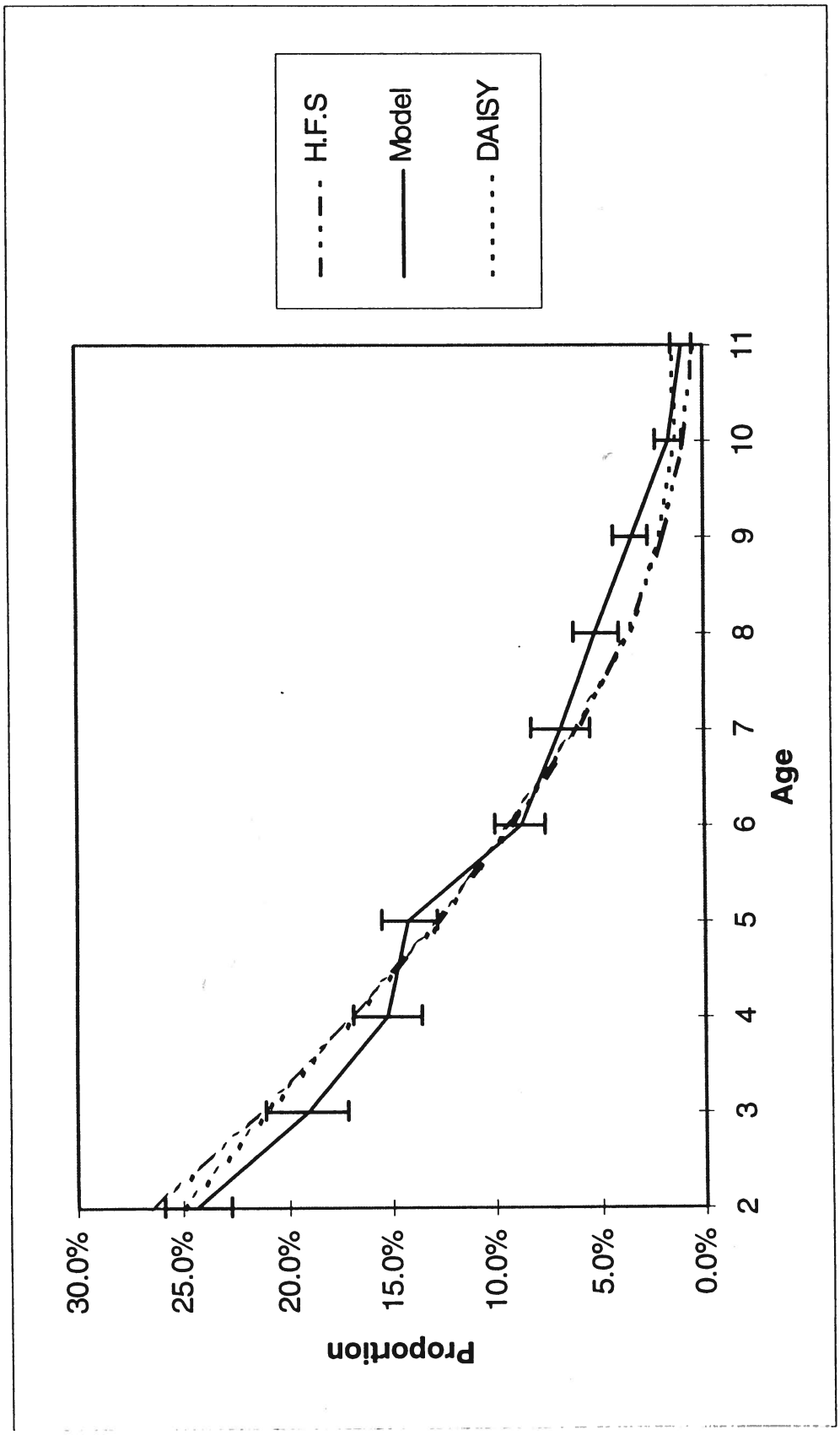

Fig. 3 Age structure of the herd based model when compared to national data collected from DAISY and the Holstein Friesan Society.

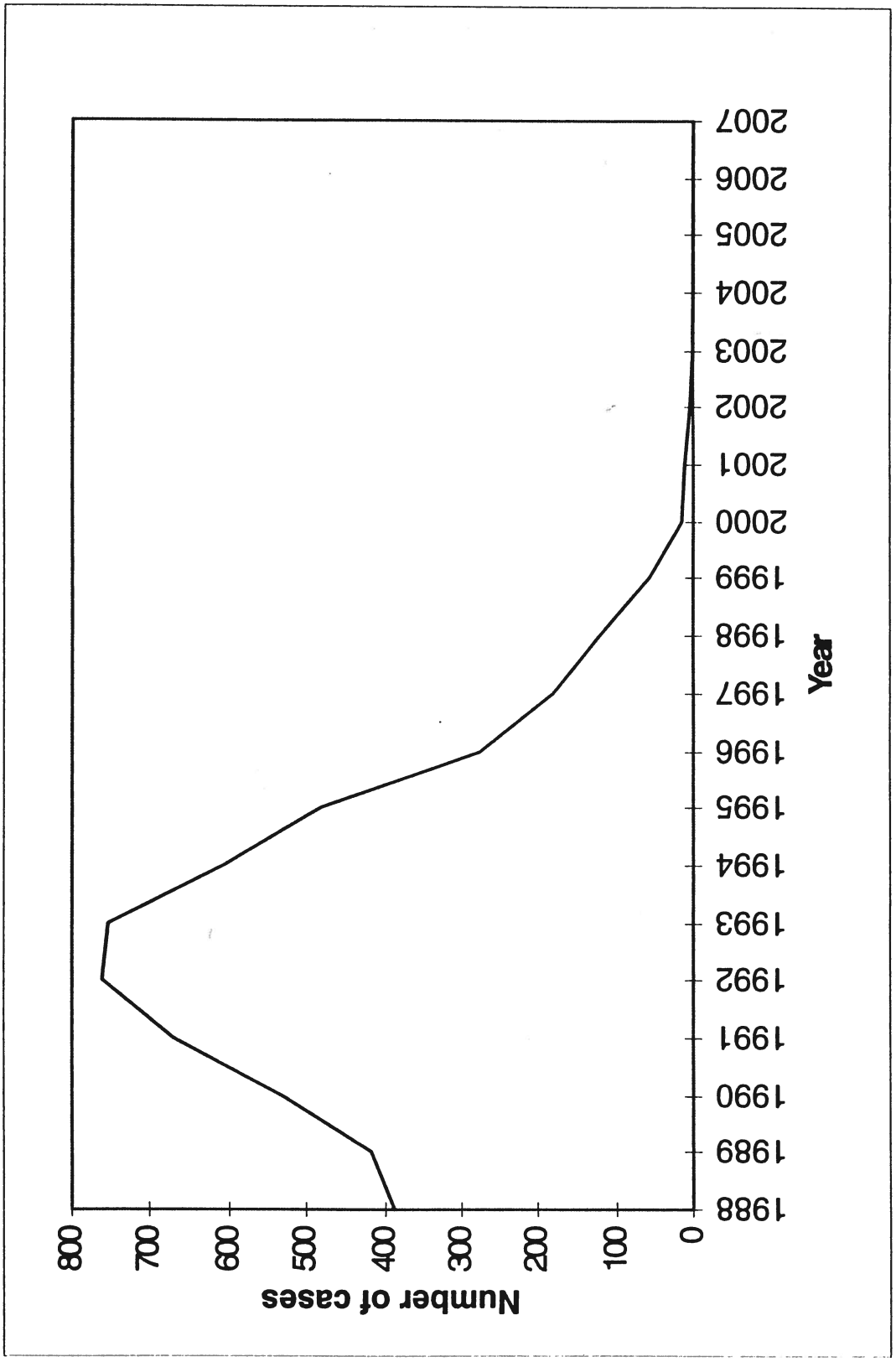

Fig. 4 Predicted total number of clinical cases by year of clinical disease onset in 500 simulated herds under scenario 1

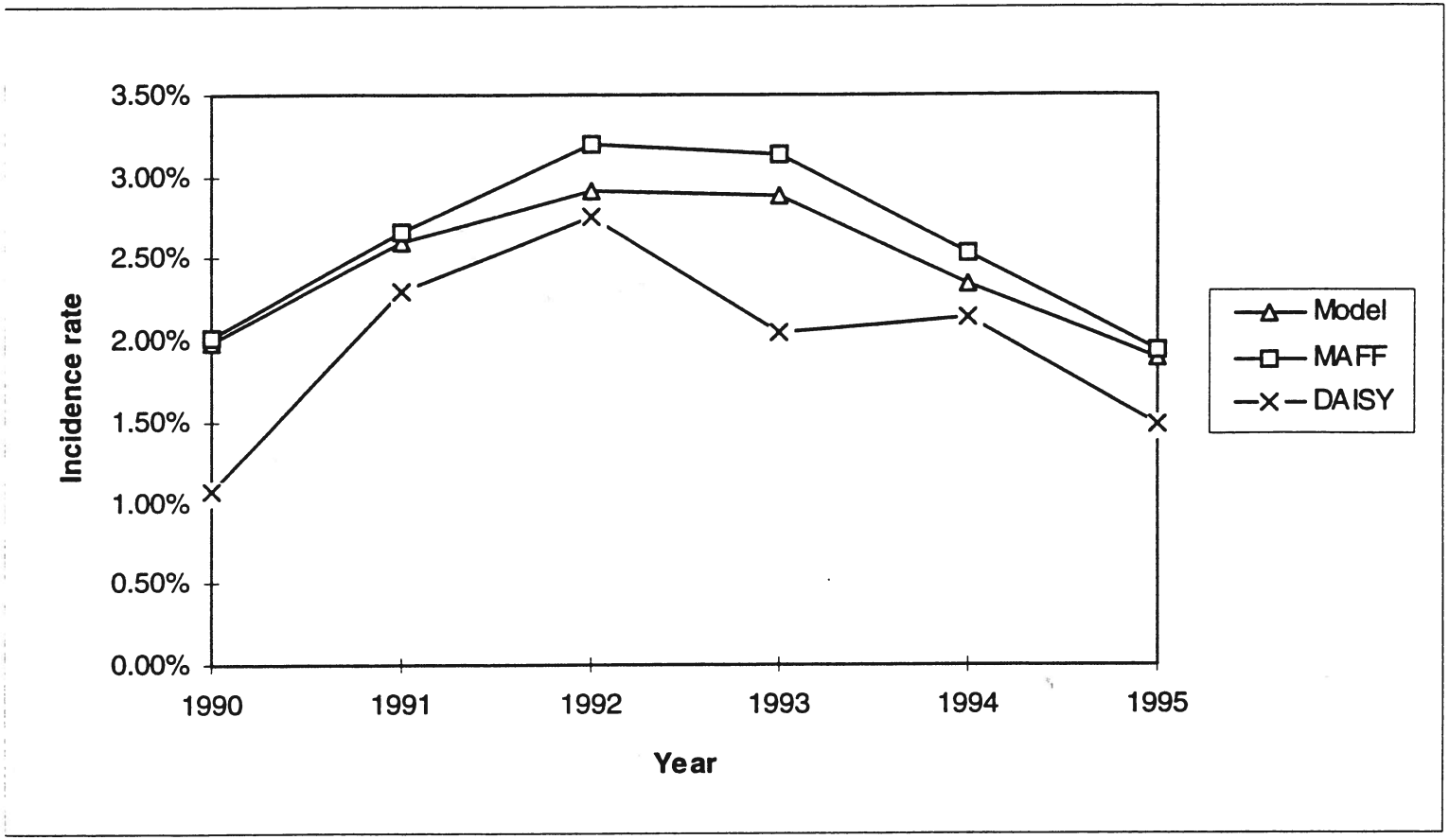

Fig. 5 Comparison of predicted clinical incidence with observed incidence calculated from MAFF BSE database and from DAISY herd records

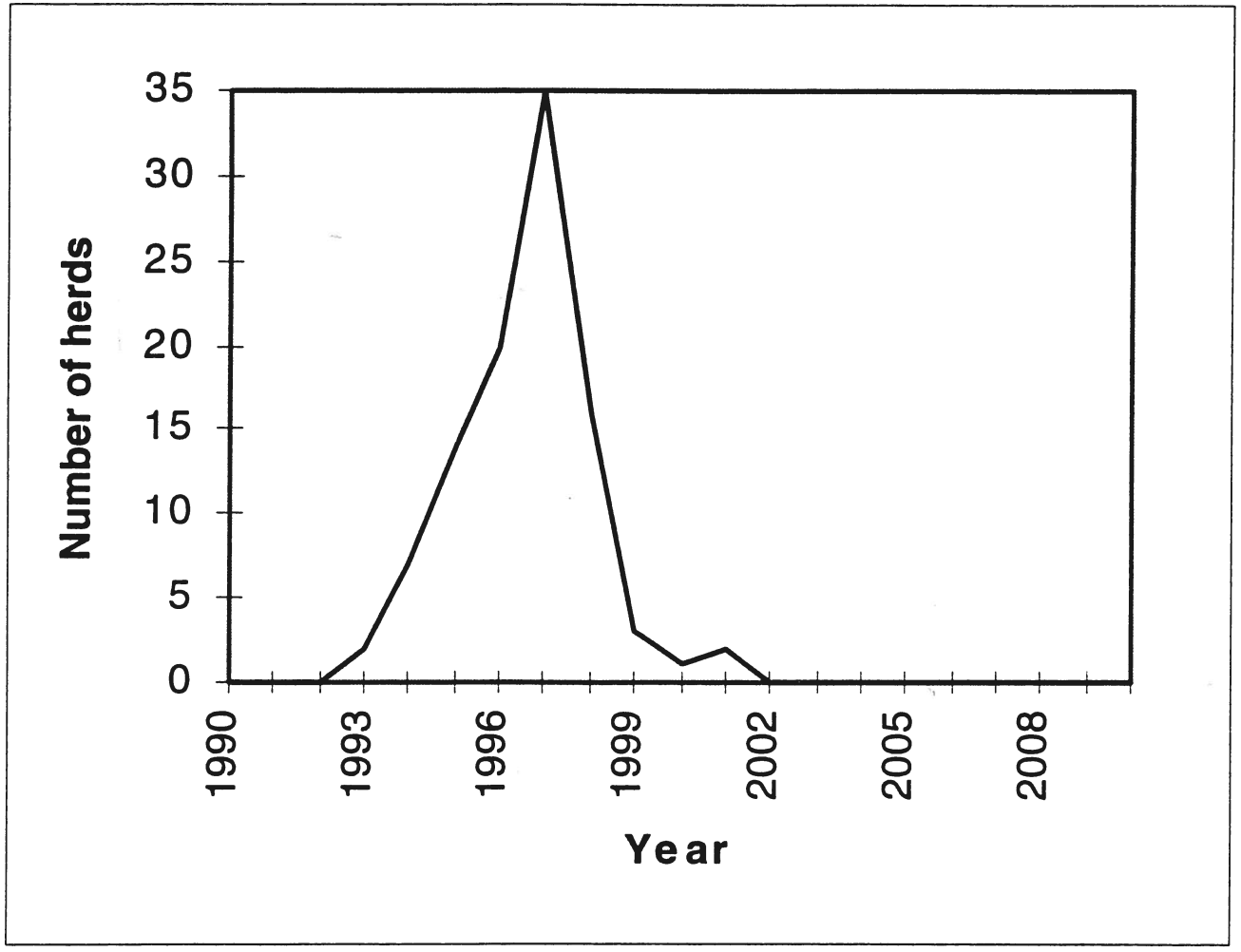

Fig. 6-a Distribution of 100 simulated herds by year that they will experience their last clinical case of BSE - Scenario 1 (no maternal transmission)

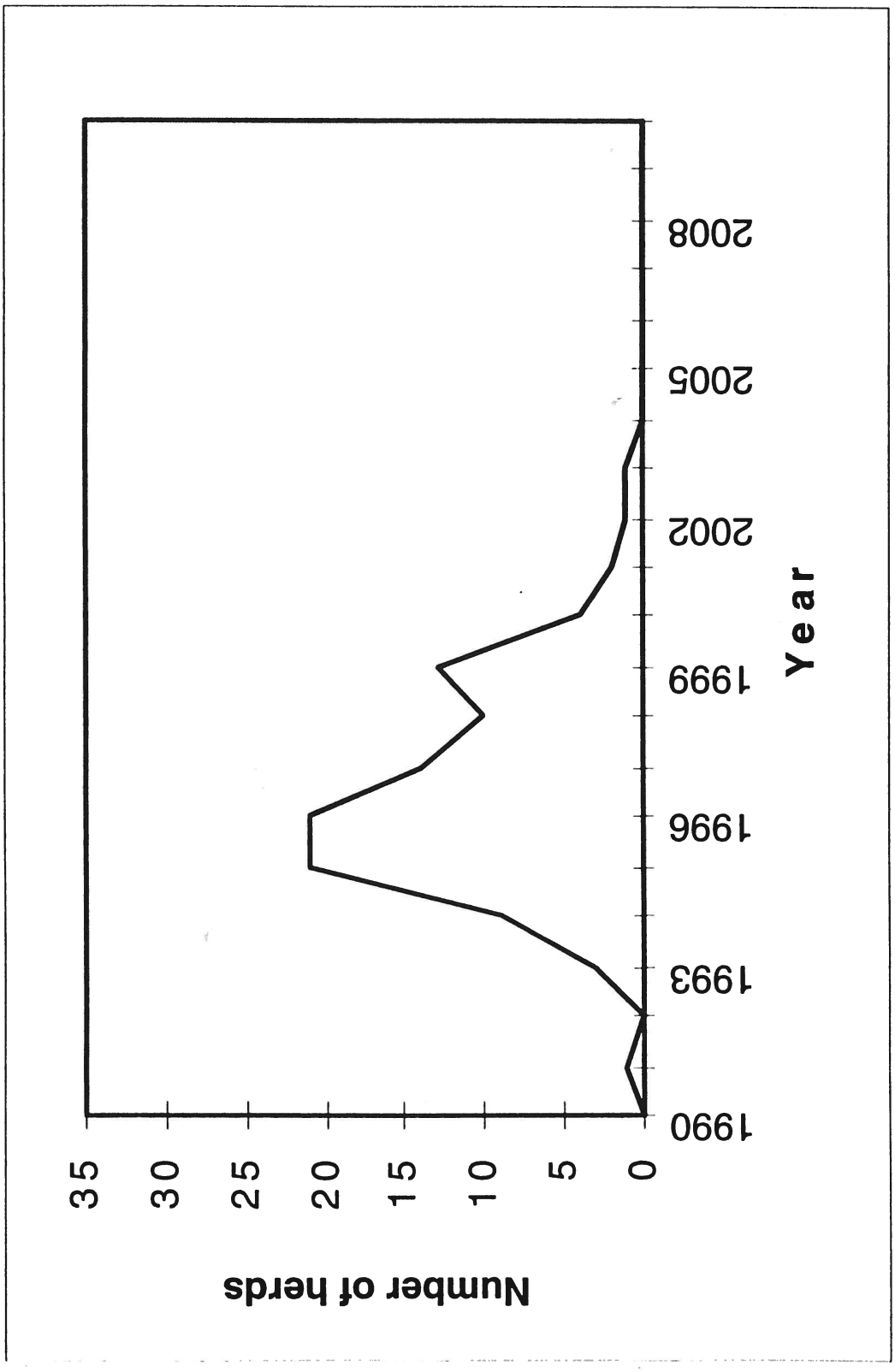

Fig. 6-b Distribution of 100 simulated herds by year that they will experience their last clinical case of BSE - Scenario 2 (10% maternal transmission)

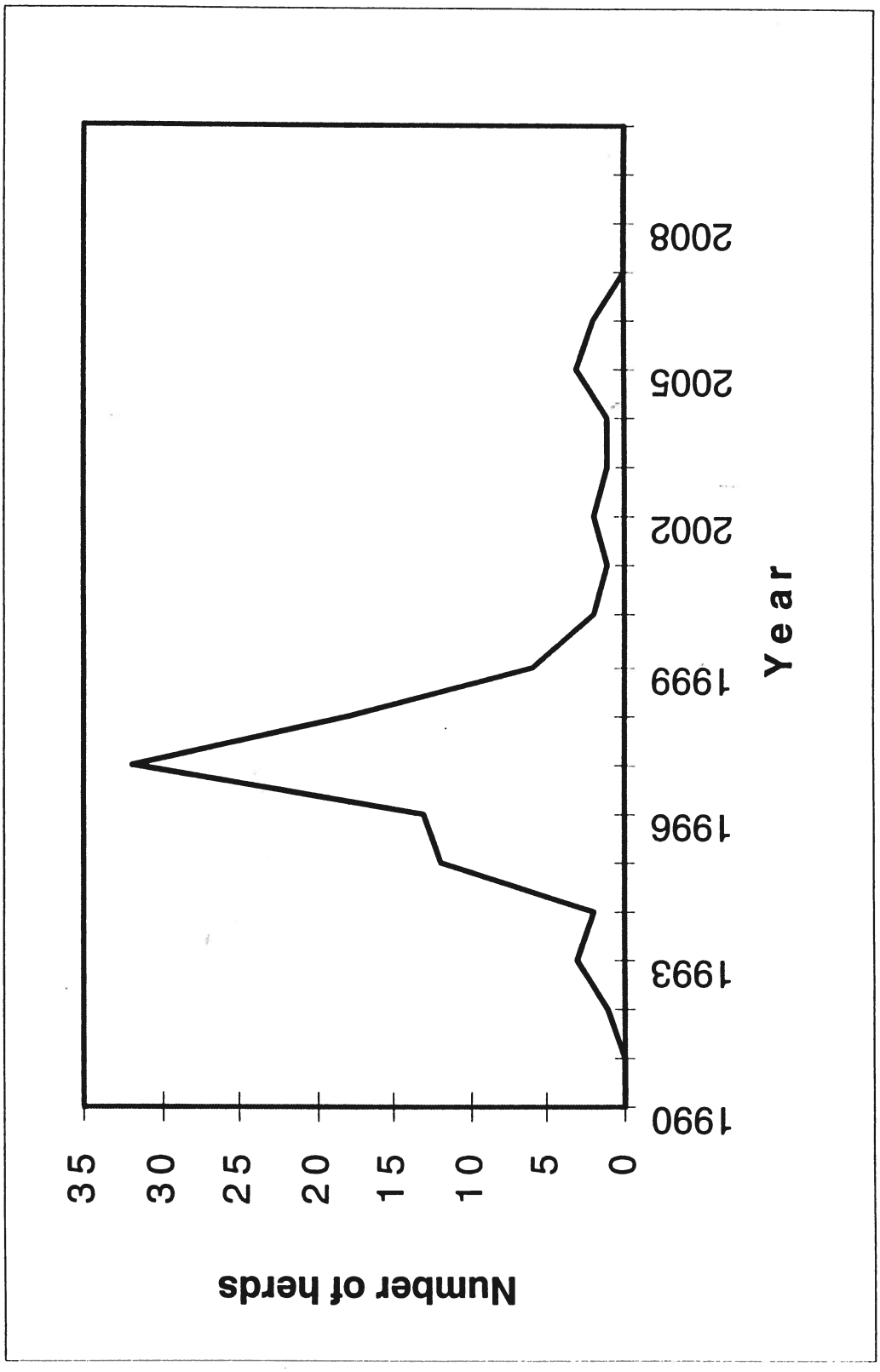

Fig. 6-c Distribution of 100 simulated herds by year that they will experience their last clinical case of BSE - Scenario 3 (50% maternal transmission)

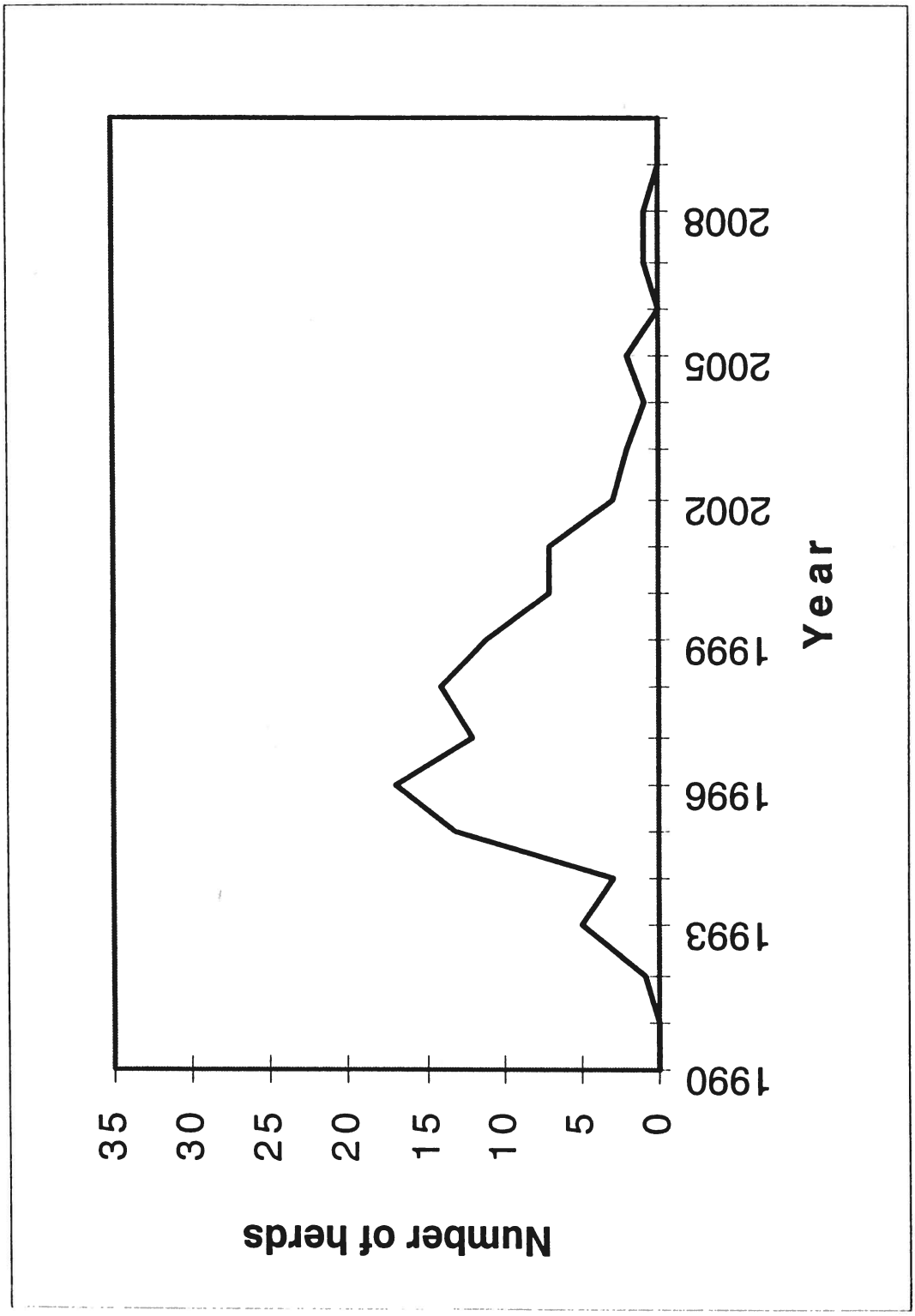

Fig. 6-d Distribution of 100 simulated herds by year that they will experience their last clinical case of BSE Scenario 4 (100% maternal transmission)

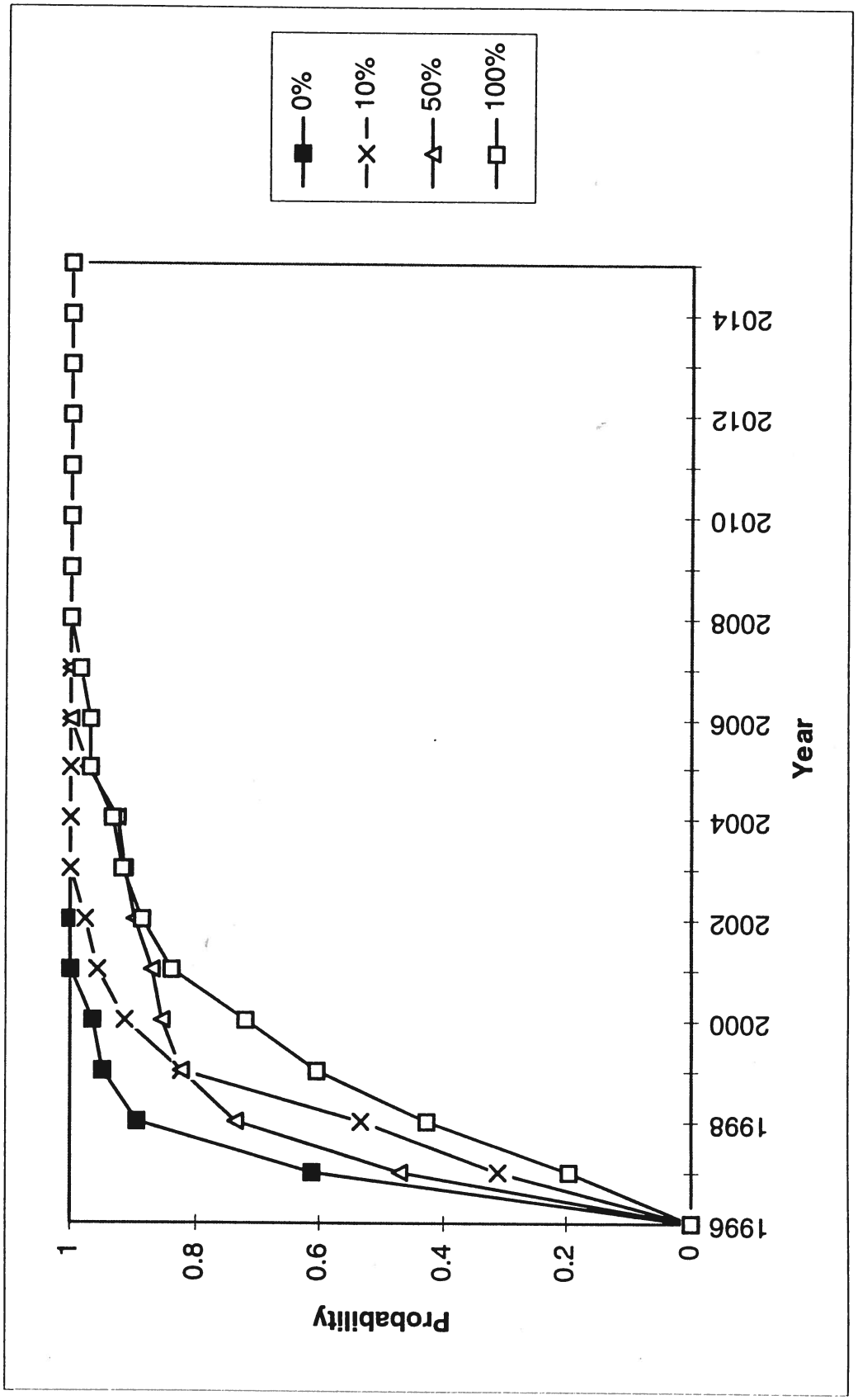

Fig. 7 The cumulative probability of clinical disease eradication in 100 simulated herds at a particular date given different maternal transmission scenarios (0, 10, 50 and 100% transmission during 1 year infective period)

herds suggest that the model slightly under-estimates the number of cases that would be expected in a herd. This may partly be due to the fact that clinical incidence rates used in the model are constant for each year when they would be expected to be a continuous variable in reality. Caution needs to be exercised in validating the model against official statistics as the original incidence figures used in the model are themselves extracted from the same data. Comparison with BSE incidence rates calculated from a small number of DAISY farms suggest that the model is overestimating incidence rates.

It would appear from this preliminary analysis that if maternal transmission occurs, it would have a significant effect on the duration of the epidemic. This is in contradiction to the projections of Anderson *et al.* (1996) which suggested that maternal transmission would not lengthen the duration of the epidemic. The model predicts that with a 50% maternal transmission rate one out of a hundred herds with clinical disease in 1987 would still have a clinical case in 2006. This compares with the last case occurring in 2001 if no maternal transmission occurred. Ideally the model should be run many more times to represent all UK herds with clinical disease in 1987 in order to make any prediction as to when the last British herd would be likely to be free of clinical disease.

The significance of maternal transmission to the long term course of the epidemic are clear from the output of this model. Increasing the maternal transmission rate extends the time required to eradicate the disease from the modelled herds. It is likely that with additional simulation occasional cases will occur well into the next century. This emphasises the importance of further research to determine the exact mechanism and significance of maternal transmission in cattle.

Caution needs to be exercised in extrapolating the results of the model to predict national trends in disease incidence. Our limited understanding of the infectious agent and the different ways that it is transmitted make accurate predictions of future incidence difficult. It is therefore more appropriate to make estimates of the relative risks or probabilities of the disease still being present at any given time. Preliminary estimates suggest that there is a 5% chance that the last case will occur in the year 2001 if no maternal transmission occurs whilst, with a 50% maternal transmission rate, there is a 5 % probability that the last infected herd will be found in 2006. To make these predictions of probability more reliable a far larger number of simulations will be required.

The structure of the model lends itself to testing the impact of different disease control strategies. The ability of the model to identify and follow individual infected animals enables an evaluation of slaughter policies targeted at individual animals. The potential effect of a policy to slaughter offspring of cows with clinical BSE could be quite easily evaluated. In addition, given an estimate of the risk of birth cohorts being infected, it should be possible to predict the impact of the present birth cohort slaughter policy.

At present the model is limited in value due to the large number of assumptions that have had to be made in its construction. In particular, the model indicates that more accurate estimates of maternal transmission rates would be necessary in order to predict the probability of eradicating the disease by a specific date. Ideally, the proposed model should be refined so that infection rates are used to calculate clinical incidence rates rather than the other way around. This requires an estimate of distribution of age of infection and incubation intervals. The model may even be adapted to describe the dynamics of BSE in beef herds, other management systems or even different regions of the country.

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ANIMAL WELFARE

A POTENTIAL APPROACH TO SUPPORT ANIMAL WELFARE PROMOTION IN A DANISH VETERINARY PRACTICE CONTEXT

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In recent years a need to assess and promote animal welfare in any given herd has emerged. Because proper disease handling and effective disease prevention are important components of welfare promotion in livestock herds, the practicing veterinarian can play a potentially very important role to support welfare promotion in the future.

The growing concern for animal welfare in society in general has initiated considerable debate among animal welfare researchers about methodology and interpretation of data in relation to measurements of welfare (Rushen and Passillé, 1992). However, close contact with Danish veterinary practitioners working in the field indicates that most veterinarians are more interested in suggestions on how to solve health (and welfare?) problems and see part of the information produced by welfare research as disconnected from real life. We, therefore, need to ask the following questions to people involved in animal welfare issues: Are the welfare promotion activities relevant to the people who are working with the problems in everyday life? Is welfare research providing useful information?

The diagnostic work or health management support on farm level provided by the practicing veterinarian basically is (an implicit) combination of scientific quantitative knowledge with experience from previous work and detailed information obtained from visits at the individual farm. This process includes a basic understanding of the human factor and implementation of various qualitative assessments. The success of the veterinarian as a business man depends on his or her ability to combine these different sources of information in order to institute correct therapy and to produce efficient and practically relevant suggestions to health and production strategies. Although not always fully realized, these elements are evident in the daily work of the practicing veterinarian and may constitute important tools for welfare promotion on the herd level. It is the purpose of this paper to describe the Danish veterinary practice context and suggest options for active involvement of the practicing veterinarian in systematic welfare promotion seen from the veterinarian's point of view.

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THE DANISH LIVESTOCK HERD AND VETERINARY PRACTICE STRUCTURE

The number of livestock herds with full time employed owners in Denmark has declined from 80000 at the beginning of the eighties to 40000 in 1994/1995. This decline is expected to continue ending up with approximately 20000 herds with major livestock production (10,000 dairy and 10,000 swine units) at the end of the century (Landbrugsraadet, 1996). However, due to the international trend towards free trade there is reason to believe that this decline will be even more dramatic . Despite these structural changes in the herds, individual ownership is still predominating in Denmark.

The Danish practicing veterinarian can foresee equally extensive changes. At present more than 50% of the practices working with livestock are owned by one veterinarian with 1-2 assisting veterinarians . In order to satisfy the needs for specialization in the advisory system, the units gradually become larger. A practice typically serves 40-150 dairy herds while the number and size of swine herds varies according to the degree of specialization in the practice. Beef production is negligible. Due to a very restrictive legislation, lay use of labeled drugs is made conditional on advisory agreements with monthly visits by the veterinarian. Without advisory agreements lay use of systemic antibiotics is either prohibited (dairy cows) or restricted to veterinary diagnosed diseases and to time intervals of 35 days (calves, pigs). Recording of disease treatment and drug use is mandatory. Production, reproduction, and disease treatment records from dairy herds are collected and available in a national database. Production and reproduction data are available from most swine herds.

HOW TO INVOLVE THE VETERINARIAN ACTIVELY IN WELFARE RESEARCH AND PROMOTION

The authors' personal experience from working in veterinary practice and information gathered through close collaboration with veterinary practitioners in research projects indicate that welfare assessment and promotion can be integrated in the current Danish veterinary practice context in the following sequence of activities: 1) Systematic clinical observations at regular herd visits initiated by farmer calls for disease treatment or production management decision support. 2) Systematic (quantitative) epidemiological analysis of available data. 3) Systematic collection of "soft (qualitative) data" related to disease handling strategies. 4) Synthesis of information through a combination of phases 1-3 to identify obvious disease related welfare problems at the individual animal level (e.g., estimate the severity and duration of pain and discomfort preceding a case of involuntary culling) and, most importantly, identify options to prevent similar problems in the future. 5) Confront the farmer with these welfare problems and suggest practical ways to reduce severity and frequency of the problems. Such suggestions are expected to efficiently visualize animal welfare problems, suggest hypotheses related to causes of these problems, and restart the process at phase one.

CLINICAL OBSERVATIONS AND RECORDINGS

Systematic and regular clinical cross-sectional examinations are suggested as the core tool for welfare promotion because they will provide concrete evidence about important welfare problems at the individual animals. The applicability of this concept is not limited to welfare issues but is highly relevant in health management work in general where the pendulum may have swung too far towards (superficial?) flock diagnoses (Markusfeld, 1996).

Our experience with health related welfare problems indicate that physical injuries, poor body condition, and leg problems constitute the potentially most important indicators of animal welfare (Baadsgaard et al., 1996, Enevoldsen et al., 1990). The fat cow syndrome and the thin sow syndrome are disease complexes which should be diagnosed at an early stage and subsequently prevented. Leg disorders and deep skin lesions seriously affect the animal due to the degree of pain and to the chronic nature of these diseases. These findings will have important consequences for production, health and welfare. Consequently there is a need for the development of a practically applicable methodology in order to generate these indicators reliably on the herd level. Although evaluation of clinical symptoms is very important in the diagnostic process and in the assessment of welfare related problems as well, the use of systematic clinical examinations of disease manifestations within the herd apparently is not generally accepted as a useful tool in welfare research. The reasons for this may be that the methodology is not yet developed with regard to application under practical conditions, and veterinarians are in general not motivated for or prepared to generate data in a systematic fashion (they often prefer to focus on single cases!). Traditional scoring systems have been applied to the evaluation of both body condition (Charette et al., 1996, Ferguson et al., 1994) and clinical manifestations (Enevoldsen et al., 1994, Gjein and Larssen, 1995) and tests of reliability have been carried out (Charette et al., 1996, Ferguson et al., 1994).

Examples: To demonstrate the use of systematic clinical examinations and some of the problems related to these as a tool for welfare promotion, experiments in dairy herds and swine herds were carried out involving practicing veterinarians. Each clinical observation was given a score value from 1 to 5 indicating decreasing degree of welfare. The value 1 refers to normal state, 2 refers to clinical symptoms with minor negative (welfare) effects on the animal, 3 refers to clinical symptoms with considerable effect on the animal, 4 refers to severe clinical symptoms and a definite need for corrective action, and 5 to a very severe or fatal case. For the body condition measurements, the score value 1 = very thin and 5 = fat . The number of observations, their distribution within the score values, and change in distribution over time provide relevant measurements of the degree of pain and discomfort due to disease in the herd. Tables 1 and 2 show cross tabulations of the body condition scores in a swine herd and the lameness scores in two dairy herds conducted by 2 different veterinarians and tables 3 and 4 show cross tabulations of repeated recordings made one veterinarian in a swine herd.

Table 1. Cross tabulations of the body condition scores in a swine herd conducted by two different veterinarians

Body condition score	1	2	3	4	5
1	0	0	0	0	0
2	4	12	2	0	0
3	0	5	21	6	0
4	0	0	1	6	1
5	0	0	0	0	0

Table 2. Cross tabulations of the lameness scores in two dairy herds conducted by two different veterinarians

Lameness score	1	2	3	4	5
1	132	6	1	0	0
2	14	13	1	0	0
3	11	6	2	0	0
4	1	9	2	4	0
5	0	0	0	0	0

Table 3 . Cross tabulations of repeated recordings of body condition made by one veterinarian in one swine herd on the same day.

Body condition	1	2	3	4	5
1	2	2	0	0	0
2	1	3	4	0	0
3	1	2	6	3	0
4	0	0	2	2	0
5	0	0	0	0	0

Table 4 . Cross tabulations of repeated recordings of lameness made by one veterinarian in one swine herd on the same day.

Lameness	1	2	3	4	5
1	6	2	1	0	0
2	2	0	1	0	0
3	1	3	3	4	0
4	0	3	2	2	1
5	0	0	0	0	0

The study showed a fairly consistent pattern for body condition between veterinarians and for repeated recordings while observations of lameness were considerably different between veterinarians and to a smaller extent for repeated recordings. In this study, observer differences could probably have been minimized if the veterinarians had examined a number of cows and sows together prior to the experiment. A comprehensive recording guide was presented before the examinations but it is impossible to foresee all types of manifestations.

In spite of the marked individual differences in the evaluation the essential question is: Can two veterinarians agree upon the level in a herd? Or even more important: Can a change in the herd level be detected by one veterinarian? The better precision of the repeated recordings indicates that this is the case. Furthermore, the results and our experience with welfare assessment (Jensen and Sørensen, 1996) and health recordings (Baadsgaard et al., 1996) indicate that other conditions such as: the use of loose housing systems or tied stall systems, group size, level of hygiene, stocking rate and floor design also has a major impact on the reliability of the recordings.

The conclusion from this preliminary analysis of the data is that the records must be seen in a local context. This makes comparisons between veterinarians and farms meaningless or at least very difficult to carry out. On the herd level, the differences between veterinarians may in some situations be of significant importance e.g. identification of emerging sub-clinical disease problems. The between observer differences thus reflect the difficulties in establishing what can be agreed upon as an "acceptable" level of clinical manifestations in the herd. For the identification of welfare relevant problems it is important to emphasize that these observations constitute one of several local contextual sources of information. Thus, the need for a critical view on data quality when using farmer and veterinary recorded diagnoses and disease treatments in health management and research and the need for further development of the methodology have been further substantiated.

ANALYSIS OF AVAILABLE DATA

In traditional herd health management, evaluation of the efficiency of production is carried out through an analysis of production and management indicators. Important indicators in swine herds are: average daily weight gain and number of piglets born, died, or weaned per sow year. Examples of important indicators in cattle herds are: calving interval, 305 day milk production, and calf mortality. Some production parameters mayb also serve as symptoms of welfare problems (e.g. sudden decrease in milk production and involuntary culling) while others may serve directly as welfare parameters (e.g., calf survival, mortality before weaning, pathologic changes at the meat inspection).

The potentials and problems related to combining various sources of information can be illustrated by an example from one of the aforementioned dairy herds(120 cows). The level of recorded disease treatments during a period of 12 months were as follows:

Leg disorders	11
Mastitis	71
Metabolic disorders	15

Due to large individual differences in the use of diagnostic criteria by farmers and veterinarians, these "typical" results produced only little information about what was actually going on on the farm. Do these data reflect an intense mastitis control to reduce somatic cell counts which per se is economically important? Have the cows been treated successfully or are they still suffering from chronic and painful conditions? Clearly there is a need to provide a more in-depth description of such a herd. A (local?) veterinarian probably has more intuitive knowledge about these matters but usually such knowledge is difficult to communicate. Consequently, there is a need to do research on how to obtain and describe such situations in a meaningful way.

The case study methodology

Case studies may be a useful tool for studies of such phenomena (Enevoldsen and Gröhn, 1996). For practitioners, single concrete examples often (usually) form the starting point for exchange of valuable experiences and information. In veterinary research, however, case studies have not gained much acceptance. However, even the single case study can provide concrete, practical and context dependent information and allow the observer (scientist) to learn the intricate details of how a problem is tackled rather than averaging the procedures across a number of cases (Kennedy, 1979). That is , it can even be possible to generalize from a single case. In traditional quantitative studies, the important detailed information may be overlooked or neglected because statistical techniques do not apply and because the information is difficult to present in traditional publication formats. These problems are, however, characteristics of real life and not a problem of case studies as a research methodology (Flyvbjerg, 1991). A major case study concept is the "critical case" which can allow logic conclusions like "if this is valid for this case, it is valid for most (or all) cases" (Flyvbjerg, 1991). Such (welfare relevant) cases are simple to construct from herd data files combined with systematic clinical observations, and interviews with the owner. Table 5 provides a list of such cases from the abovementioned dairy herd. Such a list both demonstrates the "level of welfare"and the potentials for health and welfare promotion.

Table 5. Critical cases within one dairy herd and anamnestic information covering a period of 12 months

Cow no.	Occurrence	Possible preventive action
404	A very high yelding cow. Birth induction due to beginning lameness symptoms. Milk fever in nursery box during the night and she died just before the vet arrived in the morning	++
2333	Summer mastitis discovered at calving. After calving laminitis and swollen joints and increasing problems on the slatted floor. At last unable to rise and euthanized. Not separated from the herd. Two treatments recorded.	+++
2306	Treated for traumatic indigestion. Increasing lameness symptoms and finally euthanized. Not separated from the herd. Two treatments recorded.	+++
2153	Chronic diarrhoea and finally euthanized in a cachectic condition. One treatment recorded.	++
2378	Heifer with severe birth problems and wobbling hind part.After introduction to the herd physical injuries to the legs and septicemia. Not separated from the herd. Finally euthanized. One treatment recorded.	+++
2125	Calved in the cubicle stall among the dry cows. Uterus prolapse and died one hour after treatment due to bleeding to death.	+++

The validity and precision of the information derived from these cases are indisputable and certainly they render possibilities for generalizations about animal welfare issues on some very important management criteria in this herd. These generalizations are valid for other age groups in this particular herd e.g., many dead born calves and many disease problems among the calves. In swine herds similar analysis can be carried out.

SYSTEMATIC COLLECTION OF SOFT QUALITATIVE DATA (MANAGEMENT)

The importance of the management factor in health management and in welfare issues is well known (Enevoldsen and Gröhn, 1996, Johannesson et al., 1996, Rushen and Passillé, 1992, Sandøe et al., 1996). Traditionally, the approach to study this factor has been dominated by the use of quantitative methods listing several measurements of management routines relevant for the assessment of welfare. Examples of such welfare-relevant routines are, for instance (Sandøe et al., 1996): stocking rate, water supply, chain lengths in tie stalls, ventilation efficiency, disease and parturition handling, and frequency of regrouping. Although there is no doubt about the relevance of such parameters in relation to welfare, this strategy has several short-comings. First, there is a considerable lack of knowledge about the effect on welfare of a numerous of these details. Second, numerous dynamic interactions between these factors occur on the individual farm and may affect welfare in an unpredictable direction. Third, many of the parameters are very difficult and time consuming to measure and one may end up with numerous measurements but only limited understanding of the importance of the results. Finally, from the authors' experience with veterinary practice the whole concept of management rarely fit into diagrams, questionnaires or rigid action plans for management. The understanding of the management concept involves the whole process of learning, including the final steps: intuition, feelings, and a holistic and synchronous perception of problems and problem-solving. It implies a type of knowledge about numerous preceding problems or cases that is difficult to formalize. This is essentially what good management and management support is all about. To gain insight into this (new) field and generate new theory from such qualitative data, we need qualitative methods (Meek, 1991). It is difficult to quantify qualitative concepts.

However, it is the authors' experience that detailed knowledge about critical procedures related to management can be derived from regular herd visits through close contact with the production site and the staff. Therefore, the veterinarian's work constitutes a unique opportunity to obtain information about what is going on the farm. If collected and communicated properly, the practical value may far exceed what can be derived from questionnaires, and technical recordings. However, little research has been done to systematically utilize the practicing veterinarians knowledge about herd management.

SYNTHESIS OF INFORMATION (IDENTIFICATION OF PROBLEMS)

The estimation of the severity and frequency of disease problems involves a combination of the preceding three phases: systematic clinical examinations, quantitative and qualitative analysis of available data, and qualitative assessment of critical management procedures revealed through the case study. The combination of methodologies in the study of the same phenomenon is called triangulation (Denzin, 1978). Several advantages are related to the concurrent use of both methods e.g.: improved quality of data for quantitative methods, formulation of new theories, achieving deeper insight, and contextual understanding (Lunde and Ramhøj, 1995). During the process, the following steps need to be considered: 1) What are the major relevant measurements/observations on the individual farm? 2) Given a certain observation, it must be decided whether it is a

real problem or just due to chance. 3) Given a certain problem, are there any options for prevention available at all? 4) Are there any options for prevention available in the actual context? Table 6 shows examples of clinical disease problems and their evaluation in a welfare context through the application of phase 1-3.

Table 6. Examples of clinical disease problems and their evaluation in a welfare context.

	Duration		
	Long	Short	Very short
Severe pain	Severe sole ulcer with exposed pododerm	Effectively treated foul in the foot	Teat amputation
Moderate pain	Sole ulcer of light degree	Left-sided abomasal displacement with operation	Effectively treated mastitis
Weak pain	Contusion of the hock without lameness	Uncomplicated diarrhoe	Injection without local irritation
Discomfort	Tail mange	High yielding cows dried off abruptly	Hoof trimming

Comments to the examples: Clinical manifestations may vary in intensity, duration and incidence. The consequences for the animals may vary according to the housing constraints and management - the lesion or the etiology itself tells us only little about the whole story in relation to welfare.

CONFRONTATION WITH THE FARMER

In general, the practicing veterinarian avoids the use of evaluations or interpretations of observations in absolute terms. For instance, usually it will not be relevant to state in all herds that a certain incidence rate of cows with skin lesions is "unacceptable" because the term unacceptable is irrelevant to the farmer unless means to change the state of interest have been identified. Furthermore, as pointed out earlier, individual perceptions and judgments make a common agreement upon an "acceptable" level of disease in a given herd very difficult to establish. Another important characteristic is that the first judgment given by the consultant concerning some state primarily is meant as a "suggestion", as a starting point for dialogue between the farmer and the consultant.

This dialogue will usually lead to more detailed observation concerning some areas (e.g. more specific information about the individual cows with skin lesions). These new observations may lead to an adjustment of the initial evaluations; usually a consensus can be obtained. In this process the veterinarian's (epidemiological) work is complicated by the vagaries of human behaviour such as why farmers behave as they do or say they do.

A thorough understanding of the human factor (the farmer and his value orientation) implies humanistic research methodologies such as Grounded Theory (Lunde and Ramhøj, 1995, Meek, 1991). The objective is not to test hypotheses but to create theory about phenomena relevant for health and welfare promotion, "grounded" in qualitative data usually through text analysis of interviews (Kvale, 1996). In veterinary epidemiology, the methodology has been applied (Boland and Morris, 1988) but it is striking how limited acceptance the study of human behaviour has gained in animal health and welfare research.

CONCLUSION

Insight into the real world problems in veterinary practice has indicated that there is an urgent need to consider other paradigms in welfare research. An alternative approach to combine herd health management and welfare research has been suggested. That approach adds qualitative scientific methods (e.g., case study techniques and text analysis of interviews) to the traditional (purely?) quantitative approaches like systematic clinical examinations and analysis of data.

Basically the structure of the process is well-known to practitioners and not really different from integrated herd health programs. The essential difference is that freedom from pain and discomfort is formulated as an explicit management goal. Such an approach is practically applicable today if the veterinarian wants to actively include welfare aspects in his services to clients. The current suggestion is experience based, however. There is a clear need for research to support this process with guidelines for performing the clinical "welfare examinations" and analysing and interpreting data in a welfare context. Such research is currently underway and will, hopefully, make the process more operational and provide estimates of the "welfare value" of the different types of information available to a practicing veterinarian in a typical commercial context.

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HEALTH AS A PARAMETER FOR ASSESSING DAIRY HERD WELFARE: ADVANTAGES AND DISADVANTAGES

L. ALBAN* AND J.F. AGGER*

Despite the lack of consensus on a definition on animal welfare it is commonly agreed that behaviour, physiology, and health constitute important groups of parameters for assessing animal welfare (Simonsen, 1993). In this paper, focus is on health as a parameter for welfare. Various definitions of animal welfare are presented. Next, advantages and disadvantages associated with use of prevalence and incidence data for assessment of herd welfare are listed. The association between disease and welfare is discussed with a presentation of a welfare scale. Finally, considerations are presented regarding requirements for an ideal welfare parameter, and the assessment of welfare on the herd level. The paper is one of the results of a study which in part aimed at developing a method to assess welfare in Danish dairy herds.

WELFARE DEFINITIONS

It is commonly agreed that welfare can be applied meaningfully to sentient beings only (Broom, 1996; Duncan, 1996; Holtug, 1996). To most people talking about the welfare of an earthworm makes no sense - yet to some it does. Some scientists go further and argue that the cognitive aspects - which would say the feelings - are the most central elements of welfare and that we have to develop ways to "ask" the animals what they feel (Duncan, 1996). Contrary to this, Broom (1996) argues that feelings are just one aspect of an individual's biology and that it is not logical not to include the functional mechanisms when defining welfare. Additionally, Sandøe (1996) mentions that when "we know what welfare is, we still need to develop operational definitions which will indicate how scientists should measure welfare in practice".

To our opinion, Simonsen (1993) seems to combine the two different approaches in his definition of welfare: the feelings and the biological functioning. Simonsen sees welfare as a concept incorporating multiple attributes and defines welfare as the sum of negative and positive experiences that an animal has. Simonsen regards pain, fear, and frustration as major negative experiences, and joy, play, and satisfied expectations as positive experiences. We consider Simonsen's definition closest to reality.

Furthermore, we believe that an assessment of animal welfare cannot be carried out independently of ethical considerations - which is also suggested by Rollin (1996) and Sandøe et al. (1996). However, this implies that we somehow put a threshold value on how little welfare we will accept, i.e. the matter of setting our ethical borderline.

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ADVANTAGES AND DISADVANTAGES ASSOCIATED WITH USE OF PREVALENCE AND INCIDENCE DATA

In livestock farms, disease data from surveillance systems or from observational studies are an obvious source of information for assessment of animal welfare, because they are relatively easy to collect. Classical designs of observational studies are either cross-sectional, cohort, or case-control (Martin et al., 1988). Data from cross-sectional studies or case-control studies are prevalence data while data from cohort studies and surveillance systems are incidence data. Some of the advantages and disadvantages of these two kinds of data will be discussed with reference to the farmer-owned nationwide surveillance system for production diseases in Danish dairy herds, named the Central Danish Cattle Disease Recording Scheme. The data are stored in the Danish Cattle Database together with information on production and breeding. Data from case-control studies will not be discussed.

Incidence data

Incidence data describe the situation in a herd during a specified period of time. Veterinary treatments and owner treatments are examples of incidence data of relevance for welfare. Since 1989, veterinary treatments of Danish dairy cows in an increasing number of herds have been reported routinely to the Danish Cattle Data Base (Agger & Alban, 1995; Alban, 1995; Alban 1996). The purpose of reporting veterinary treatments is - among others - to form the basis of herd health management schemes and breeding for better health. This signifies, that data are readily available for use in assessment of welfare on herd level.

This kind of data imply several disavantages, some of which will be discussed. A very low recording level may occur if the veterinarian forgets to report his treatments, or if the farmer deals with a case without registrering it on a barn sheet. Furthermore, the threshold of calling the veterinarian varies among farmers. Some farmers do not hesitate to call the veterinarian upon suspicion of disease while others require assistance for severe cases only. Even within a herd there may be different thresholds of calling the veterinarian, since a farmer may be more concerned with a high producing dairy cow than with a lower producing one.

Usually the farmer is more inclined to require veterinary treatment for conditions which he thinks has a substantial impact on his economy. Conditions like scabies and neglected claw health are rarely seen by the veterinarian, and hence, rarely recorded. Therefore, data on veterinary treatments represent only one source of information regarding the welfare in a herd. To get a better description and understanding of the welfare in a herd, these data should be combined with other sources of information.

To minimize the risk of a very low recording level, the veterinarian should inform the farmer about the importance of all cases being registered, and the veterinarian should keep a high registration level. The animals in a herd where the farmer has a low threshold for calling the veterinarian do not necessarily have poorer welfare than the animals in a herd where the farmer has a high threshold. Therefore, one should be cautious in comparing herds. Instead, the herd could be used as its own reference over time.

Prevalence data

Health data of relevance to welfare recorded by use of a cross-sectional study are e.g. data on 1) the claw health recorded at the day of claw trimming, and 2) a systematic, visual inspection of leg disorders, skin injuries, and presence of ectoparasites. A common feature of the examples is that they provide information on conditions which are not routinely reported to the Disease Recording Scheme because usually, the farmer does not call the veterinarian for these conditions. Other examples of prevalence data of relevance for welfare could be: 1) inspection of the housing environment to get information on

possible causal factors that impact on disease, and 2) the getting up and lying down behaviour.

Data from a cross-sectional study represent the status at the moment of the observer's presence in the stable. Causal studies based on cross-sectional designs should be carefully interpreted, since the exposure-response time relationship is not always clear. However, this depends on whether the exposure variable is constant or varies over time. In addition, responses (condions) which were present in the past may not be detected. For behavioural responses, completely different results might be achieved depending on what time of the day the observation is done. For acute health conditions, the prevalence may vary from week to week - or even day to day, e.g. in a herd no calves may show symptoms of enzootic pneumonia on a certain date, while the majority of the calves are ill the next day.

For chronic conditions, the prevalence may vary from month to month, e.g. in October, at the end of the pasture season only a few tie stall cows may suffer from chronic tarsal cellulitis, and in February when the cows have been tied up during the winter months a third of the cows may show varying degrees of tarsal cellulitis (Fig.1). Furthermore, as Willeberg notes (1991) prevalence data tend to overestimate chronic conditions. To sum up, it may be problematic to use prevalence data for exposure as well as for response factors that vary over time. Therefore, prevalence data should be combined with other sources of information on the herd health.

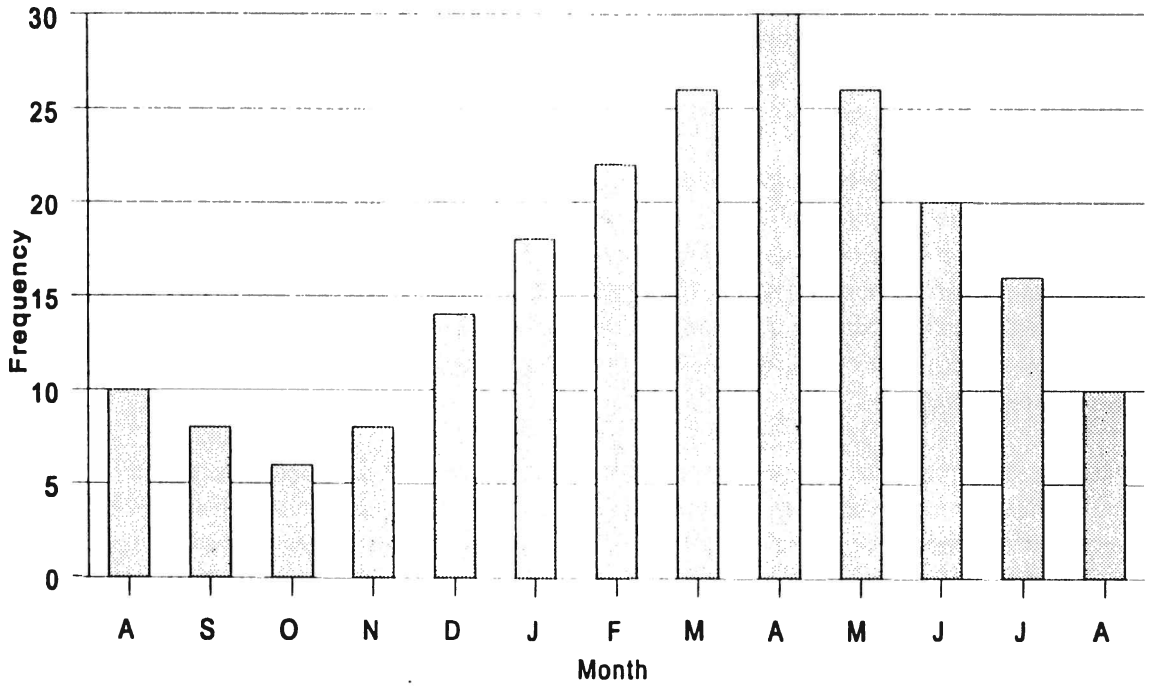

Figure 1. The monthly prevalence of tarsal cellulitis (hock lesions) in a tied dairy herd

Sample size and data quality

The study of a few herds or animals often requires one person only. Hereby, the inter-observer variation is avoided. However, some intra-observer variation will always exist. This may be the case for data based on a registration of the claw health at the day of trimming and data based on a systematic, clinical examination of the dairy cows. Large studies, on the contrary, usually involve more registrants, and inter-observer variation cannot be avoided. This is the case for data on veterinary treatments, where ususally more people are involved in recording disease data from a dairy herd, because in most veterinary practices more veterinarians collaborate and share duties. Therefore, analysis of data from surveillance systems, ikke the Danish Cattle Data Base, should always be carried out with caution.

THE ASSOCIATION BETWEEN HEALTH AND WELFARE

In Simonsen´s definition of welfare, pain is regarded as a negative experience. Hence, diseases which are associated with pain are likely to have a negative influence on welfare. It can be speculated that some diseases - which may be of major concern to the farmer - are of little, or no, relevance to the animal, e.g. increased somatic cell count in the milk. In effect, it is necessary not just to focus on whether disease is present or not. Attention should rather be drawn to the degree of pain associated with the disease. Furthermore, the duration of the pain should be considered, since undoubtly short-lasting pain is preferable to long-lasting pain - as suggested by Morton & Griffith (1985) and in accordance with Broom & Johnson (1993).

Willeberg (1991) argues that on the herd level the fraction of affected animals in a population must be considered, because in most cases a rare condition has smaller impact on a herd´s welfare than a more common condition. Willeberg suggests that the impact of a given disease on a herd´s welfare can be written as:

Welfare impact of a disease on a herd = Pain * Duration * Fraction of affected individuals

We agree with this proposition. This leads to the question of how to assess an individual´s pain. Sandøe et al. (1996) argue that since pain is a mental state, it can only be assessed indirectly. Broom (1996) recommends to use physiological measures, effects of analgesics, presence of neuromas, or the degree of aversion an animal may show. We do not know how the welfare impact of different diseases are related, e.g. additively, multiplacatively or otherwise. Nor do we know whether the feeling of pain is modified over time, as also discussed by Broom & Johnson (1993).

Even though we may never gain the full insight to how an individual feels pain, investigation into e.g. endorphins will probably reveal important, new information. As a preliminary attempt, we have ranked some of the most common diseases among dairy cows according to their welfare impact on the individual based on general, veterinary knowledge (Table1). Levels for pain and duration can be set to any number of values. We have used three levels for pain, and two for duration. We have used the term "discomfort" where we found that was a more correct description of what we believe the cow feels. We have ranked diseases like laminitis, severe sole ulcer, and traumatic reticulitis in the top, and mild mastitis and increased somatic cell count in the bottom. The ranking can be discussed and is ment to demonstrate the difference between economically important diseases and diseases of importance to the dairy cow primarily.

DEVELOPMENT OF A SCALE FOR WELFARE

Level of measurement

A scale is a structure of numbers that enables one to classify, order, or measure a set of observations and make statistical inferences from them.

Scaling has been used in the physical sciences for centuries, such as scales for weight and distance. In the social and behavioural and biological sciences scaling is a more recent device. In the last decades, measurement and scaling has been developed and used extensively in psychology and sociology enhancing greatly the development of psychometry and sociometry. Compared to this, the metrics of clinical medicine - i.e. clinimetry - is in the early stages of its development into a theoretical science (Feinstein 1977). The development of a theoretical science would seem to be virtually impossible unless its variables can be measured adequately (Torgerson 1958, quoted from Margenau 1950).

Table 1. A preliminary assessment of the welfare impact of some diseases in dairy cows

Condition	Degree of pain	Duration
Laminitis	Very painful	Weeks
Severe sole ulcer (black)	Very painful	Weeks
Arthritis	Very painful	Weeks
Amputation of claw	Very painful	Weeks
Traumatic reticulitis	Very painful	Weeks
Necrotised mastitis	Very painful	Weeks
Double sole	Has been very painful	Weeks
Teat lesions	Very painful	Days
Acute mastitis	Very painful	Days
Dead/euthanized	Has often been painful	Varying duration
Severe hock lesions	Moderate pain	Weeks
Severe heel horn erosion	Moderate pain	Weeks
Sole ulcer (yellow or red)	Moderate pain	Weeks
Severe over-grown claws	Moderate discomfort	Weeks
Scabies or lice	Moderate discomfort	Weeks
Chronic mastitis	Moderate pain	Days
L- displaced abomasum	Moderate pain	Days
Milk fever	Moderate pain	Days
Ketosis	Moderate pain	Days
Prolapsed uterus	Moderate pain	Days
Endometritis	Minor pain	Days
Mild mastitis	Minor pain	Days
Increased som. cell count	Discomfort?	Varying duration

The main reason for a lack of development of scales in the clinical science is that clinical observations are generally subjective statements and judgements and of a categorical nature, e.g. severity of pain or dyspnoe (Feinstein 1977). They are soft data in contrast to hard data that are objective measurements and are generally of a continuous nature, e.g. age, weight, and serum cholesterol. Hard data are more reliable for statistical procedures. This may be one of the main reasons for the extensive use of paraclinical data, e.g. in clinical chemistry. Paraclinical data are, however, often not appropriate for the goal of a clinical study, e.g. a welfare study.

To construct a scale requires that the parameter the scale is to present, can be measured. Four levels of measurements are generally accepted today as proposed by Stevens (1951). These are 1) the nominal scale, 2) the ordinal scale, 3) the interval scale, and 4) the ratio scale. The nominal scale is the lowest and the ratio scale is the highest level of measurement. According to the level of measurement different levels of statistical procedures can be applied to the data.

So which level is reasonable to achieve for a scale measuring welfare? We feel the ordinal level would be reasonable, because we for most situations are able to judge if an animal feels different in one situation

compared to another. However, this is partly based on the analogy postulate, based on which we assume that animals percieve in the same way as humans. For example, it is generally assumed that the cows feel well and in some ways benefit positively when groomed, and when lying in a clean, dry and soft tie stall or cubicle, and also that animals feel more uncomfortable the more severe pathological stage we observe (Agger & Alban 1996). Use of preference studies and operant conditioning are ways of achieving further information on these subjects.

An example of clinimetry

In a study on the possible association between teat lesions and mastitis and lameness, Agger (1980) developed a scale for the assumed levels of pain associated with lameness. Based on literature and clinical observations in a prospective, longitudinal study in a dairy herd, each disease was described in groups of progressing pathological stages and associated pain. A scale ranging from 0-10 was used with 0 as no pain or lameness, and 10 as maximum pain and maximum lameness, i.e. the cow can not walk on the leg due to lameness. Each pathological stage was ascribed a pain level, e.g. the 4'th stage of chronic necrotic pododermatitis was given the value 5. This stage has so severe horn destruction that corium is not covered with horn; and corium may be infected. There is both mechanical pain when the cow puts weight on the affected claw and also chemical pain due to the infection. All pain levels from all limb diseases in the animal were "summed up" to one pain score, i.e. one common estimate for the pain level due to limb diseases in that cow at that point in time. However, this scaling approach was not verified to another measure of neither pain nor "total lameness".

Assuming a persistent pain level for each disease, this level may be multiplied by the length of the disease period (e.g. 10 days). This is an estimate of that disease's negative welfare impact in that cow during that period. This may be transformed to the herd level, accounting for the pain of all the cows - in accordance with Willeberg´s formula. Such measures may be repeated over time for the same herd. Hereby, the herd acts as its own reference. The scale will theoretically range from zero to + infinity. However, what is the quantity of one pain unit? We do not know at the moment!

Obviously this scale is basically on the ordinal level, since the difference between two pain levels can not be estimated quantitatively - all we can say is that one pathological stage is more or less painful than another. However, the summation of pain units means the data are handled as if they were measured on an interval or ratio scale. Comparisons among herds may not be relevant due to mathematical and statistical problems.

REQUIREMENTS FOR AN IDEAL WELFARE PARAMETER

Logically, it should be possible to select a number of variables which can express the welfare status in a herd. Parameters for both health and behaviour are necessary for a broader statement about the welfare status, but for the present only health parameters will be considered. To make a welfare assessment operational, the number of parameters should be restricted. Therefore, a carefull selection is necessary. We believe that four requirements for an ideal welfare parameter should be met: 1) the parameter should represent a welfare status, 2) it should be quantitative, 3) it should be unambigously related to a causal path, and 4) it should be meaningfull to the farmer and the veterinarian.

The first requirement indicates the importance of the parameter representing a general state in the herd and not an extreme situation. As an example, the calf mortality may be high after introduction of BVD in a herd, but this does not signify that the welfare of the calves is low in general. The selection of what to record at which time should be considered very carefully, as discussed in subsection *Prevalence data*.

The second requirement implies that an ordinal scale can be used for the parameter, i.e. one end of

the scale is the best and the other end is the worst. In general, it is possible to sum up the number of diseased animals. No diseased animals is the best, and the worst is when all animals are diseased. Hence, the second requirement is fulfilled. But, strictly speaking, this may in fact not be the situation, since diseases vary in intensity among individuals. This "summing up" is an approximation which may be more or less good. As an example, cases of mastitis may differ in severity from an increase in somatic cell count, to mild cases associated with minor pain to seriously painful, necrotic mastitis. This is demonstrated in Table 1, where mastitis is listed four times and sole ulcer twice.

The third and fourth requirement signify that knowledge of what influences on a parameter is imperative if attempts to improve welfare are to be made. For example: if it is intented to reduce the amount of stress in a pig herd, it is not appropriate to monitor the level of aggression since - for the time being - we do not know how to interpret this parameter (Ladewig, pers.comm.). Therefore, information regarding the level of aggressions does not give the farmer an opportunity to act.

CONSIDERATIONS REGARDING ASSESSMENT OF WELFARE IN A DAIRY HERD

To make an assessment of health related aspects of dairy herd welfare, we suggest to use information regarding: 1) veterinary treatments during a year, 2) clinical examinations carried out three to four times a year, and 3) claw health monitored at the day of claw trimming. It may seem very time consuming to collect all this information. Unfortunately, we find that it is doubtfull whether a welfare assessment, based on less information, can be used to improve welfare. Imagine, if a clinician should base a health certificate for export, based on one, two or three parameters only!

We believe that a welfare product label, based on system parameters solely, is no guarantee for improved welfare. As Sandøe et al. (1996) point out, measurement of the system is a measure of the risk of decreased welfare only, and not a measure of welfare itself. It is commonly known that stockmanship is of outmost importance. The animals in a poor system - but with a talented, caring farmer - may be better off than the animals in a better housing system with a bad farmer.

Furthermore, if the intention is to improve welfare, focus should be on the farmer, because he is the main actor on a livestock farm. Therefore, it is important that the parameters which are included in a welfare assessment are meaningful for the farmer and the veterinarian. In principle, this implies that the parameters included should meet the requirements for an ideal parameter. Hereby, the welfare assessment can be used as a basis for decisions to improve the welfare.

Currently, an official welfare product label is being introduced for beef and pig production in Denmark (Anon., 1996). It is disappointing, that the label is based on system parameters only. Even though we find it could have been done better, the intention for making the label is positive. Furthermore, we agree with the requirements which form part of the label. Therefore, we find that the label is a good beginning.

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A POSSIBLE STRESS REACTION IDENTIFIED IN A GROUP OF HEIFERS IN A CONTROLLED EXPERIMENT

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Within the constraints of a particular husbandry system and the genetic potential of the cow milk yield is adjusted according to the quantity and quality of feed available. Factors such as disease, and nutritional problems such as excessive concentrate or excessively fat cows also affect milk yield (MacRae *et al*, 1988; Roberts *et al*, 1981). The assessment of stress has been reviewed and the imposition of stress affects both productivity and behaviour (Broom and Johnson, 1993). In order to alter the shape of a lactation curve or long term trends in body weight, condition score and appetite, a stress will need to be imposed over a protracted period. The identification of chronic stress is based largely on changes in behaviour and is difficult to quantify.

This paper describes changes in production and metabolic parameters which appear to have been associated with chronic stress in a group of experimental animals and may be of use in the recognition and quantification of chronic stress.

MATERIALS AND METHODS

The duration of the study was from approximately 6 weeks before calving to 12 weeks after calving.

Experimental animals

Forty pedigree Holstein heifers had been reared conventionally at an average growth rate of 0.68 kg/day and calved down at approximately two years of age between October 1994 and January 1995. The summer of 1994 had been spent at grass but supplementary concentrate had been fed during the late summer due to lack of grass.
The heifers were blocked by sire, within sire by lifetime weight gain, within live weight gain by ascending PIN value and each block of four heifers was randomly allocated to one of four treatment groups.

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Treatments

The experimental design was 2 x 2 factorial. The primary treatments were housing in cubicles (C) with rubber mats and shavings bedding or in straw yards (S) on deep wheat or barley straw. The heifers were housed approximately 6 weeks before calving in cubicles or straw yards according to their allocated experimental groups but all heifers were kept in straw yards for a period of 5-7 days around calving. The feeding passages, feed barriers and distance walked to the milking parlour were similar for all four groups. The cubicle housing was sub-divided by gates to separate the groups and there was only a single entrance to the cubicle passageway from the feeding passage for both cubicle housed groups. The feeding and cubicle passageways were cleaned twice daily with automatic scrapers.

The secondary treatments were the post-calving nutrition and two alternative complete diets were fed. These two diets contained 60% (H) and 30% (L) concentrate on a dry matter basis respectively, mixed with the same grass silage and fed *ad-libitum*. Group intakes were recorded daily. The concentrate feed was home-mixed and compounded from rolled wheat, sugar beet feed, molasses, extracted soya bean meal, fishmeal and a vitamin and mineral supplement. The proportions of ingredients in the concentrate are given in Table 1 and the proximate analyses of the two lactation diets are presented in Table 2. The pre-calving diet fed from the time the heifers were housed was the same for all groups and was a complete diet of grass silage, sodium hydroxide-treated straw, brewers grains and a small quantity of the home-mixed concentrate, the proportions of ingredients are presented in Table 3.

The four experimental groups were designated: CH (cubicles, high concentrate diet), CL (cubicles, low concentrate diet), SH (straw yard, high concentrate diet) and SL (straw yard, low concentrate diet).

The heifers were footbathed in an antibiotic solution of Lincospectin (Upjohn Ltd) or Erythrocin (Sanofi Animal Health Ltd) every 2 weeks to prevent digital dermatitis.

Table 1. Formulation of concentrate feed ingredients.

Ingredient	Quantity (kg/tonne as fed)
Rolled wheat	610
Molassed sugar beet feed	100
Molasses	100
Extracted soya bean meal	140
Fish meal	25
Mineral and vitamin supplement	25

Table 2. Proximate analysis of complete diets

	High concentrate diet	Low concentrate diet
Silage:Concentrate	40:60	70:30
Oil g/kg DM	27.0	29.0
Crude Protein g/kg DM	179.0	174.0
Neutral Detergent Fibre g/kg DM	285.0	359.0
Starch plus sugar g/kg DM	326.0	226.0
Metabolisable Energy MJ/kg DM	12.5	12.1

Table 3. Formulation of pre-calving complete diet

Ingredient	Quantity (kg as fed)
Grass silage	4-10[a]
Sodium hydroxide treated straw	3.5
Brewers grains	10.0
Concentrate (see table 1)	1.5

[a]The grass silage content was adjusted according to dry matter content and energy concentration based on silage analysis.

Examinations and monitoring

Locomotion scoring according to the system developed by Manson & Leaver (1988) was carried out weekly from approximately 3 weeks before to 12 weeks after calving and a locomotion score greater than 2 was interpreted as mild lameness. The clinical condition of the heifers was noted during the locomotion scoring procedure.

Group feed intakes together with the number of animals in the group were recorded daily. The variation in calving dates between and within the groups together with the large potential variation in feed intakes between individuals precluded meaningful statistical analysis of the differences in feed intake between groups.

Live weight and condition score were measured once a month from calving.

Individual milk yields were recorded daily throughout the experiment.

Blood samples were collected for estimation of plasma β-hydroxybutyrate (βHB) and non esterified fatty acid (NEFA) concentrations during the first, third and sixth week of lactation. Proprietary kits were used for the estimation of βHB (Sigma Diagnostics) and NEFA (Alpha Laboratories) in a random access analyser (RA-XT, Technicon Instruments Corporation).

Haptoglobin concentration was estimated in plasma samples (Lewis *et al* 1989, Skinner *et al* 1991) collected weekly from approximately 3 weeks before until 3 months after calving to assess the changes in acute phase protein concentration. The baseline haptoglobin concentration was defined as the mean concentration during the second and third weeks prior to calving and a significant rise in haptoglobin concentration (acute phase response) was defined as an increase of greater than 100% of the baseline value.

For continuous variables statistical analysis of the differences between groups was by analysis of variance. Pearson chi-square analysis was used to analyse group differences in condition score change and chi-square analysis with Yates correction was used to analyse the incidence of mild lameness.

RESULTS

Feed intake

The highest daily feed intake of complete diet was consistently recorded for CH (Fig. 1; Table 4). The heifers housed in straw yards ate an unquantified amount of bedding straw in addition to their complete diet.

Table 4. Estimated individual daily feed dry matter intakes (kg/head/day)

Housing	Concentrate level	Daily feed intake
Cubicles	High	14.5
	Low	12.1
Straw Yards	High	13.2
	Low	12.6

Liveweight change

Heifers fed the low concentrate diet lost more weight during the first month of lactation ($P < 0.01$) and gained less body weight during the second and third months of lactation with a larger overall mean body weight loss (Table 5; $P < 0.001$). There were no significant interactions between housing and feeding treatments although group CH had the lowest mean liveweight loss during the first month of lactation, gained the most liveweight during the second and third months of lactation and was the only group with a positive mean daily liveweight change over the first three months of lactation (Table 5). There was a trend for greater weight loss for heifers in straw yards (Table 5) which was not statistically significant.

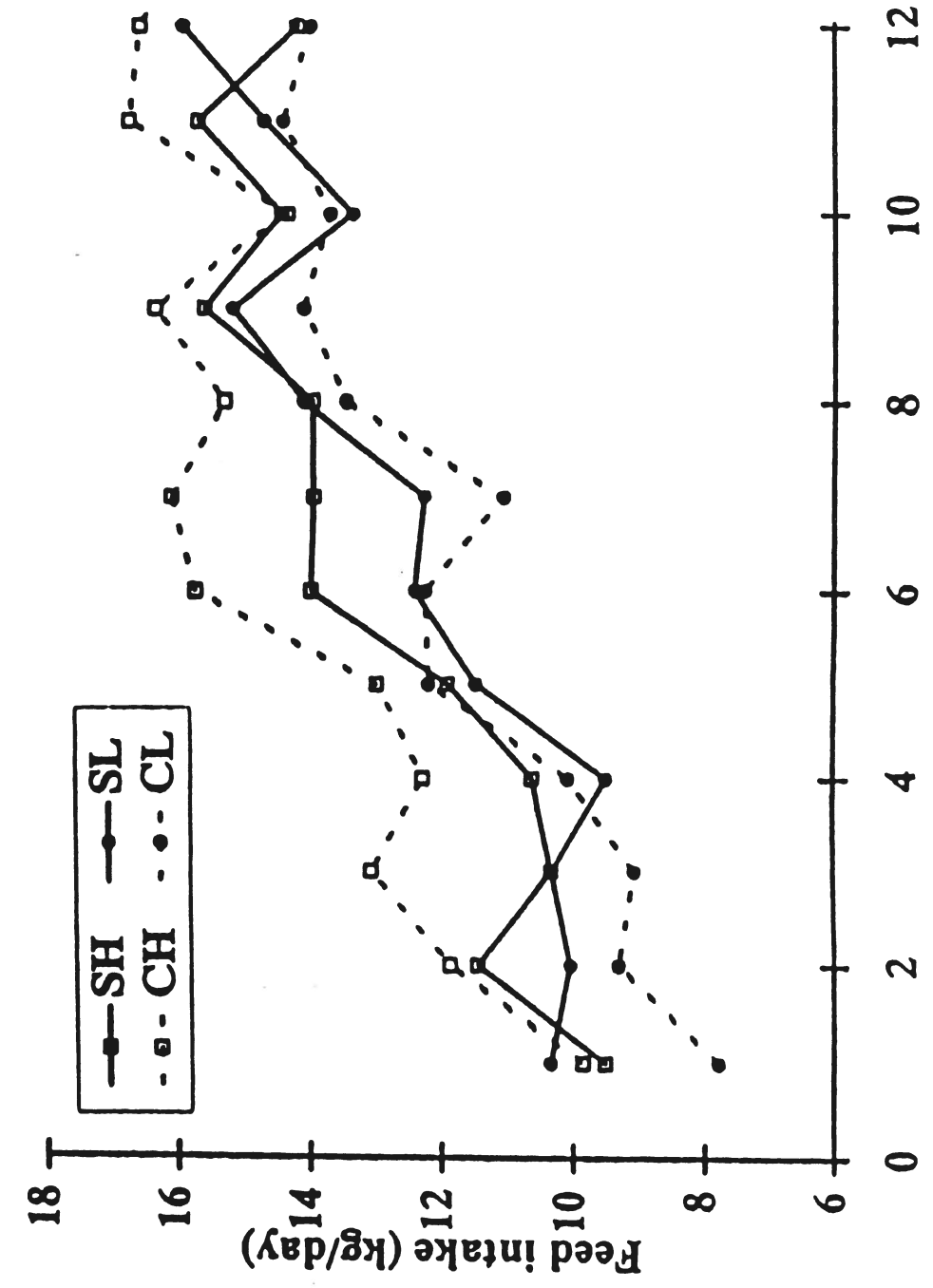

Fig. 1 Group mean daily feed intakes (kg/head/day)

Condition score

Condition scores were within the range 1.0 to 4.0 for all groups throughout the study. There was a significant interaction between housing and diet in the effect on condition score change over the first three months of lactation ($P < 0.05$) with the high concentrate diet and cubicle housing associated with a smaller loss of condition score (Table 6). However, in the first month of lactation there was a trend for group CH to lose more body condition than group SH.

Milk yield

There was a trend throughout the study for heifers housed in straw yards to produce more milk and this attained significance ($P < 0.05$) at five weeks post partum (Fig. 2). The heifers fed the high concentrate diet tended to produce more milk during the third month of lactation. During the first month of lactation heifers fed high or low concentrate diets produced similar quantities of milk but the lowest yields were recorded for group CH.

Table 5. Least square means of change in liveweight (kg)
Interactions: diet and housing

Housing	Concentrate level	Lactation period (weeks) 1-4	4-8	8-12	1-12	Mean daily liveweight change
Cubicles	High	-23	20	12	8	0.09
	Low	-56	8	2	-41	-0.48
Straw Yards	High	-33	15	5	-11	-0.13
	Low	-44	1	1	-42	-0.50
s.e.m.		7.8	4.5	3.8	10.2	0.12
F. probability		NS	NS	NS	NS	NS
Effect of Diet						
Concentrate level						
	High	-28	17	8	-1	-0.02
	Low	-50	5	2	-41	0.49
s.e.m.		5.5	3.2	2.7	7.2	0.09
F. probability		<0.01	0.01	NS	<0.001	<0.001
Effect of Housing						
Housing System						
Cubicles		-40	14	7	16	-0.19
Straw Yards		-39	8	3	26	-0.30
s.e.m.		5.5	3.2	2.7	7.2	0.09
F. probability		NS	NS	NS	NS	NS

Table 6. Least square means of the change in condition score

		Lactation period			
Housing	Concentrate Level	1-4	4-8	8-12	1-12
Cubicles	High	-0.4	-0.2	0.1	-0.5
	Low	-0.5	-0.2	0.1	-0.7
Straw Yards	High	-0.1	-0.2	-0.4	-0.7
	Low	-0.6	-0.3	-0.1	-1.0
Pearson chi-square		7.89	4.5	10.3	12.7
Degrees of freedom		6	6	6	6
Probability		NS	NS	NS	<0.05

Metabolic profiles

During the first week of lactation all groups contained at least one individual with plasma NEFA concentration greater than the normal reference range of less than 600 µmol/l but for treatment CH six of the heifers had NEFA concentrations less than 200 µmol/l. The normal range for βHB in lactating cows is less than 1.2 mmol/l and all groups were within this range with no differences related to treatments. The interaction between diet and housing was not significant (Table 7) but both cubicle yards ($P < 0.05$) and the high concentrate diet (Table 8, $P < 0.01$) were associated with lower blood NEFA concentrations.

During the third week of lactation plasma NEFA (non-significant trend only) and βHB (Table 9, $P < 0.05$) concentrations were higher for heifers fed the low concentrate diet but βHB concentrations only exceeded 1.2 mmol/l for three heifers.

During the sixth week of lactation plasma NEFA concentrations were similar for all four groups and were all less than 600 µmol/l while plasma βHB concentrations were higher for diet L ($P < 0.05$) but only one heifer exceeded 1.2 mmol/l.

Haptoglobin

Haptoglobin concentrations increased in all four groups around calving independent of the imposed treatments (Fig. 3). For all groups approximately half the individuals were classed as non-responders (less than twice the baseline value) around calving. The proportion of non-responders was in excess of 75% for most of the remainder of the study except for group CH in week five post-partum when there was a significant interaction ($P < 0.05$) between housing and diet with a high group mean haptoglobin concentration and only three non-responders (Table 10).

Table 7. Interactions of plasma NEFA and βHB concentrations: 1st, 3rd and 6th weeks of lactation

Housing	Concentrate Level	NEFA (μmol/l) Wk 1	Wk 3	Wk	βHB (mmol/l) Wk 1	Wk 3	Wk 6
Cubicles	High	256	258	247	0.5	0.5	0.5
	Low	488	331	243	0.5	0.7	0.7
Straw Yards	High	437	323	298	0.6	0.4	0.6
	Low	608	390	254	0.6	0.9	0.9
s.e.m.		72.9	57.1	32.0	0.07	0.13	0.11
F.probability		NS	NS	NS	NS	NS	NS

Table 8. Effects of housing and diet on NEFA concentrations: 1st week of lactation

	NEFA (μmol/l)
Effect of Housing	
Cubicles	372
Straw Yards	523
s.e.m.	51.5
F. probability	0.05
Effect of Diet	
High concentrate	347
Low concentrate	548
s.e.m.	51.5
F. probability	0.01

Table 9. Effect of diet on βHB concentration: 6th week of lactation

	βHB (mmol/l)
Effect of Diet	
High concentrate	0.5
Low concentrate	0.8
s.e.m.	0.08
F. probability	<0.05

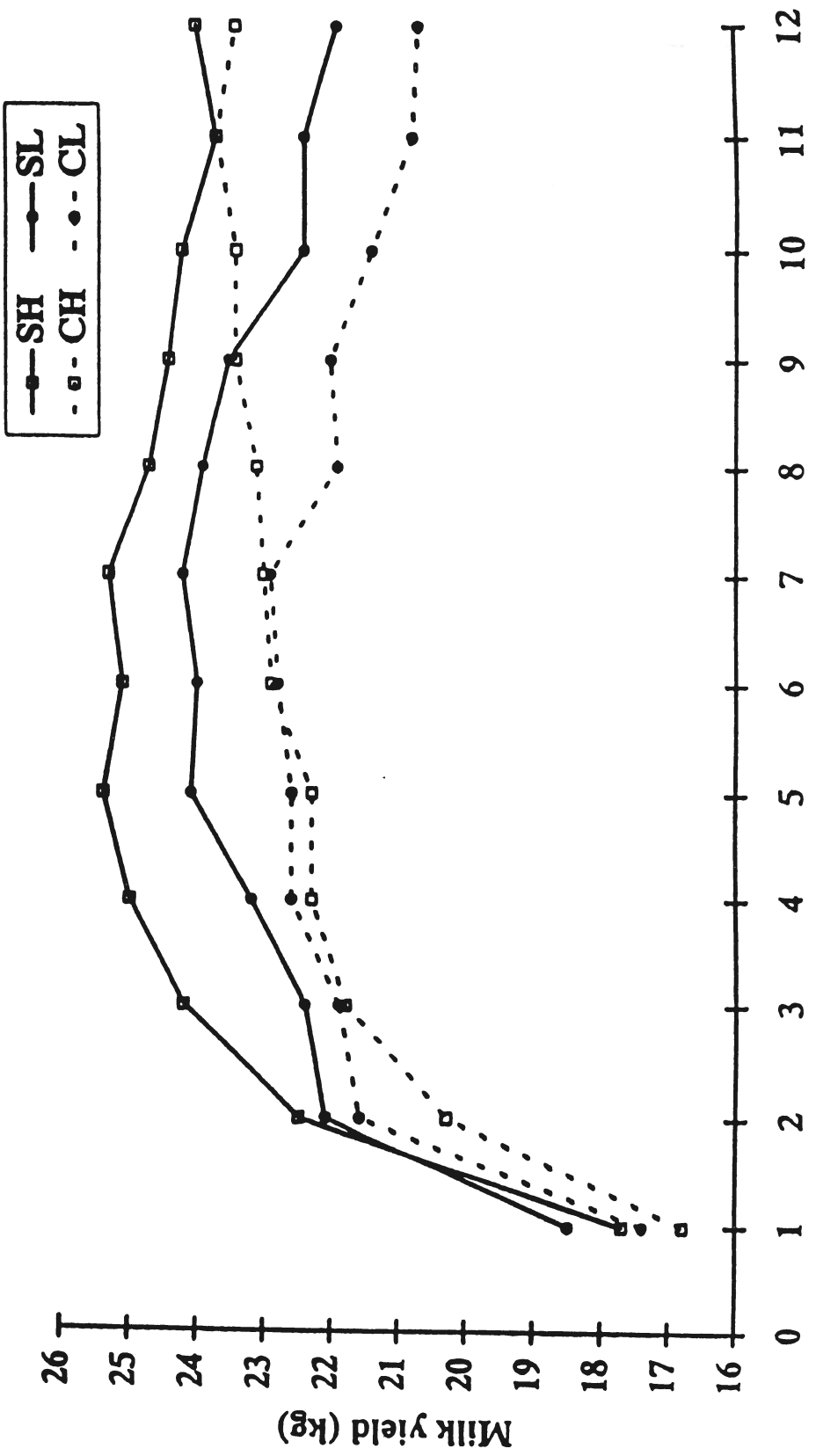

Fig. 2 Daily Milk Yield

Fig. 3 Haptoglobin Concentrations g/l

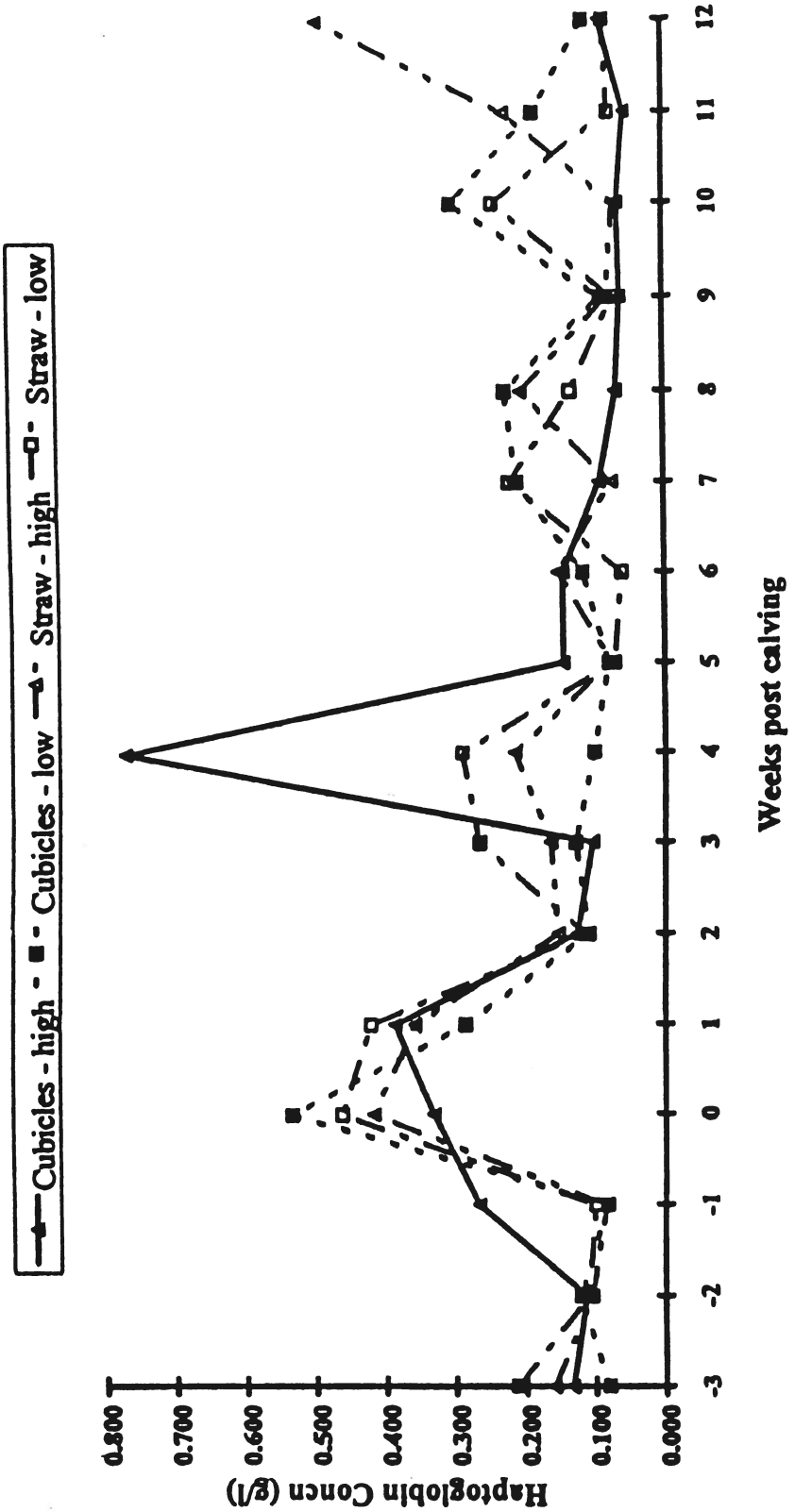

Table 10. Interaction of housing system and diet on haptoglobin concentration: week 5 of lactation.

Housing system	Concentrate level	Haptoglobin concentration (g/l)
Cubicles	High	0.804
	Low	0.003
Straw Yards	High	0.057
	Low	0.178
s.e.m.		0.2030
F. probability		<0.05

Clinical observation

During the first month of lactation the heifers in group CH were dirty with extensive dung adherence to the upper legs and body, their demeanour suggested they were depressed or dejected and when locomotion scoring was carried out it was relatively easy to walk up to and touch them compared with the other groups of heifers. These signs were alleviated during the second month of the experiment.

The high concentrate diet was associated with a higher incidence of mild lameness (Table 10; P <0.05), but the incidence was similar for groups CH and SH and the prevalence of sore feet was low in all groups until several weeks after calving.

Table 11. Incidence of heifers with mild lameness during the first 12 weeks of lactation. Interactions of housing and diet

		Incidence of locomotion scores >2.0
Interaction of housing & diet		
Cubicles	High concentrate	7
	Low concentrate	3
Straw Yards	High concentrate	7
	Low concentrate	2
Effect of diet		
High concentrate		14
Low concentrate		5
Probability[b]		P<0.05

[b] Chi square with Yates correction

DISCUSSION

A syndrome of relatively poor milk production occurred during the first month of lactation in group CH. This was accompanied by the lowest loss of body weight, with plasma NEFA concentrations which suggested that heifers in CH mobilised less body fat than the other groups immediately after calving with feed consumption records indicating that CH also had the highest daily intake of complete diet. A moderate haptoglobin response occurred independently of treatments affecting approximately 50% of all groups during the two weeks around calving but was succeeded by a haptoglobin response in the fifth week of lactation which was restricted to CH and affected more than half of the group. The demeanour of the heifers in group CH was also abnormal relative to the other groups during this period, they were generally dejected and the extensive faeces contamination of upper legs and bodies suggested that they had avoided lying in the cubicles. The milk production of the heifers in group CH had started to improve relative to the other groups by week 7 of lactation and by week 12 was similar to group SH. It is likely that this syndrome was caused by chronic stress.

The milk production of the heifers was highly variable and consequently the differences in milk yield between the groups were not usually significant. However, the shape of the lactation curves (Fig. 2) suggest that the milk yield for CH was comparatively depressed during the first month of lactation and subsequently recovered. The two groups housed in straw yards both produced lactation curves with a significantly ($P < 0.05$) higher yield than the cubicle housed groups for week five of lactation, suggesting that the cubicle yards used in this experiment had a negative effect on milk production.

The acute phase response in CH five weeks after calving, presented an apparent interaction between the high concentrate diet and cubicle housing which was consistent with the trend of depressed milk production. The delay in the acute phase response until a month after calving suggests this may have been an effect rather than a cause of the syndrome because other abnormalities were present from immediately after calving.

The diet containing a higher level of concentrates was expected to produce higher milk yields regardless of the housing system, unless a confounding factor such as rumen acidosis, or an independent factor such as infectious disease adversely affected milk production. The two groups housed in straw yards ate an unquantified amount of long bedding straw in addition to their complete diet but there was no access to straw in the cubicle yards, therefore rumen fermentation or rumination could also have been influenced by the housing system. Rumen pH and volatile fatty acid analyses were not carried out so there was no definitive information on rumen fermentation but the apparent benefit of the straw yard housing was also present for heifers fed the low concentrate diet which contained 70% silage and it is unlikely that significant chronic acidosis would occur on such a diet or even on the high concentrate diet which contained 40% silage. Therefore if access to straw was important it was more likely to have been the influence of the long fibre on rumination than simply the addition of fibre to the diet.

Body condition scores were always within the accepted normal range and should not have adversely affected appetite or production. The high concentrate diet was associated with lower liveweight loss during the first month of lactation ($P < 0.01$) and although weight loss

for CH was slightly less than for SH the effect of housing was not significant. Condition score change followed a different pattern during the first month of lactation with SH showing smaller loss of condition than CH although the effects of diet and housing were not significant. This apparent contradiction between loss of body weight and condition score may have been accounted for by differences in gut fill which could have been consistent with the higher group feed intakes recorded for CH than for SH and might have been related to the small but unquantified difference in diet arising from access of SH to bedding straw. However the apparently very low plasma NEFA concentrations for CH during the first week of lactation suggest that fat mobilisation was significantly less for CH immediately after calving, the depressed production syndrome had already commenced during the first week of lactation and the condition score data may be misleading. Hence some factor other than fat mobilisation may have caused a hidebound appearance and thereby the trend for apparently greater loss of condition score for CH than for SH. The abnormally depressed and dirty clinical appearance of CH during this period could have caused the heifers to appear hidebound and influenced condition scoring.

Alleviation of the syndrome from approximately week 6 occurred with no specific intervention suggesting that either the stress or constraint on production was transitory or alternatively that the heifers adapted to it.

Calving dates for CH spanned approximately four weeks but six of the heifers calved in the same week and it is possible the stress was independent of lactation and related to a confounding factor such as exposure to infection. The clinical appearance of depression and faecal soiling of heifers in group CH was consistent with a variety of enteric infections but the restriction of the syndrome to CH in spite of direct contact with the adjacent group CL suggests the syndrome was not directly contagious. The additional stress for CH could also not be attributed to lameness because the incidence of lameness was similar for CH and SH and was generally delayed several weeks after calving whereas the production syndrome was apparent from calving.

The comparatively severe faecal contamination of the upper legs and bodies of heifers in CH was probably caused by either cubicle rejection or restricted access to the cubicles. The design of both cubicle yards was similar suggesting that yard design alone was not responsible. The presence of gates subdividing the shed into groups restricted the movement of animals in both cubicle yards because of a single entry point to the cubicle passageway for each group and it was more likely that submissive heifers were bullied and excluded from their beds in the cubicle yards than in the straw yards. It is possible that the syndrome was precipitated by the presence of one or two dominant animals in CH which caused a hierarchical problem which was by chance not duplicated in CL.

CONCLUSIONS

Changes in milk production including the shape of lactation curves, physiological abnormalities recognised from metabolic and acute phase protein profiles and deviation from expected bodyweight, condition score and appetite may all contribute to the recognition of chronic stress. An unusually low level of fat mobilisation in early lactation may be equally as

significant for assessing the welfare of dairy cows as the excessive mobilisation of fat. The assessment of parameters such as condition score is subjective and should, if possible, be supported by confirmatory data, justifying the use of comprehensive profiles which take account of nutrition, production and biochemistry.

The improved performance of group CH during the second two months of lactation does not imply that the level of stress during the first month was acceptable but it would have been difficult to confirm that the group was stressed without comparison with the other experimental groups. This suggests there is a need for development of biomarkers of chronic stress.

The apparent interaction between cubicle housing and the high concentrate diet in this syndrome may indicate that adverse effects of diet and housing can be cumulative. This is consistent with anecdotal reports that well managed cows can tolerate a temporarily imposed stress better than poorly managed (highly stressed) cows.

Hierarchical stress may have contributed to the problem in group CH. Straw yards may allow cattle to tolerate higher levels of hierarchical stress than cubicle yards especially if the cubicle yards permit only restricted circulation and access to the cubicles.

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MONITORING BROILER WELFARE DURING REARING AND LOADING

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Welfare has been defined as the five freedoms: freedom from thirst, hunger and malnutrition; appropriate comfort and shelter; prevention, or rapid diagnosis and treatment of injury and disease; freedom to display most normal patterns of behaviour; and freedom from fear (Farm Animal Welfare Council). According to this definition, it is not possible to talk about animal health without entering the area of animal welfare. When we monitor animal health, which we do mainly for economical reasons when it comes to production animals, we also indirectly monitor aspects of animal welfare. When enforcing a surveillance programme in order to improve animal health and thereby producer economy, it will in many cases also improve animal welfare.

In practice, most monitoring programmes in the farm animal production industry are aiming at preventing disease. A lot of diseases can obviously be prevented by housing the animals in a stimulating environment with a proper climate where they have access to water and suitable feed, which means that such factors are usually included in animal health and welfare monitoring programmes. It is also common to include prevention of so called 'production diseases', i.e. diseases that are more or less a result of the ways that we keep farm animals for production purposes. Examples are metabolical diseases due to intensive feeding regimes or inflammations in the reproductive organs or mammary glands linked to high production. A programme that claims to monitor welfare should also include prevention of contagious diseases, although this aspect is surprisingly often forgotten. Contagious diseases may cause pain, suffering and death in the animals, which is certainly a welfare issue. There are also contagious diseases that will not cause disease in infected animals, but may be a threat to consumer heath if they reach the food market. Most probably, this has been the main reason for including measures against infectious diseases in some control programmes.

BROILER REARING - THE SWEDISH SITUATION

In the beginning of 1986, the broiler consumption in Sweden was approximately 4.2 kg per person and year. This roughly corresponded to the local production, and imports and exports were low. There had been some debate about the way broilers were reared, broiler farming was often used as an example of 'factory production of animals', implying extensive welfare problems. Visitors were not welcome to the farms, as it was generally believed that if consumers saw how the birds were reared nobody would want to buy the meat. Most broiler farmers were not too proud of their work and did not make any large profits. The producers national branch organisation, called the Swedish Poultry Meat Association (SPMA), lived a rather quiet life. Although being the central organisation for 97 % of the broiler producers and all ten major poultry abattoirs, their influence was limited.

In the middle of 1986, a professor in meat hygiene gave a talk at the Nordic Veterinary Congress in Stockholm (Bergquist & Danielsson-Tham, 1986). She talked about the prevalence of *Campylobacter* in broiler meat and the infection risks for the consumers. One of the major national newspapers that covered the meeting found the story interesting, and printed it the next morning (Granestrand, 1996). Within a couple of weeks, broiler consumption was decreased by 40 %. The broiler industry was devastated. Their

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reputation was a catastrophe, as broiler meat consumption was now not only considered to be unethical, but also dangerous.

THE CARE PROGRAMME - MONITORING AND REGULATION

A sanitation programme was soon enforced and the prevalence of *Campylobacter* decreased quickly, but still the actual consumption of broiler meat was low. The industry realised that something had to been done as soon as possible to restore consumer confidence in broiler products. Together with production advisors, veterinarians and representatives from the Swedish National Board of Agriculture (SBA) the SPMA created something that was named 'The Care Programme', a regulatory programme for broiler producers.

The Care Programme aimed at improving the general standard of rearing, considering both housing facilities, equipment, management and stockmanship. Based on a set protocol, an evaluation was made of the animal area, economic area and management practices on every broiler farm in the country. The first evaluation was made by the same person, a man called the 'national standardiser', in co-operation with the local veterinary inspector and the poultry manager at each slaughterhouse. Operations judged to maintain high standards have since then been inspected by the 'national standardiser' every second or third year under the assumption that no unexpected changes have occurred, whereas operations that received lower scores have been inspected yearly. Persons believing that their operations have been unfairly judged have been able to appeal the decision by contacting the branch's central reference group, which also includes representatives from the SBA.

Considered under the programme category of 'animal area' are factors such as heating and ventilation systems, feed and water dispensers, building material, rodent security, and the ability to rapidly receive or load birds. Both the equipment itself and the standard of maintenance are evaluated. Considered under the category of 'economic area' are auxiliary power supply, alarm system and medicine-administering equipment, general hygiene conditions including sanitary barriers, among other things. Judged under the category of 'management' are animal-environment care (stockmanship), litter and air quality, record keeping practices, production results, slaughter quality (downgradings and rejections), etc. The evaluation of stockmanship is based in part on how often and carefully the stockperson sorts out and destroys sick birds. All in all, a total of 31 items are scored from 1 (poor) to 4 (very good). These scores are then weighted by multiplying them by a factor ranging from 1 to 9, depending on the relative importance of the item concerned. The total score for each broiler house is presented as a percentage of the total possible score. Every house is judged separately; thus, it is possible for a given producer to receive different scores for different buildings at the same farm.

GIVING THE FARMERS ECONOMICAL MOTIVES TO FOLLOW THE PROGRAMME

All producers who are members of the SPMA get their houses evaluated, but there is no formal way of forcing any producer to improve his housing or stockmanship standard. Instead, the programme is built on the 'reward' theory: improvements should pay in economical terms. This is done by connecting the total score received in the programme to the maximum stocking density allowed at time of slaughter in each broiler house. The higher the score, the higher stocking density of birds that is allowed. Farmers standing outside the regulatory programme are not allowed to exceed a stocking density of 20 kg/m^2 in their houses. The maximum stocking density allowed for farms within the programme is now 36 kg/m^2 (25 birds/m^2), and anything between those two values is set in relation to the score achieved.

In a compilation from November 1996, concerning 562 inspected broiler houses, none of them had received less than 65 % of the total possible score, which is the cut-off level for allowing a stocking density of 25 kg/m^2 instead of 20 kg/m^2, and 94 % had received 80 % or more of the total score, allowing a

stocking density of 30 kg/m^2 or more. Thirtyfive percent were allowed to have 36 kg/m^2. The annual consumption of broiler meat in Sweden is now 7.7 kg per person and year, i.e. an 83 % increase since 1986 when the programme started.

In the beginning, the programme was not entirely popular among the producers. Some felt that nobody else should interfere with how they reared their birds, and that this welfare thing was all rubbish. During the first year of the programme, 10 to 15 % of the producers left the broiler industry and the SPMA. These were mainly producers who had old-fashioned broiler houses and who were not interested in making investments to improve their standards. This automatically lead to an average improvement on the national basis, as the worst farms were shut down. Any new producers entering the industry get substantial instructions about the programme and a visit from the 'national standardiser' before their first batch of birds arrive. Since the start of the programme the housing standard has improved gradually and all new houses have been built aiming at maximum scores. This means that the number of farmers being allowed to rear their broilers at high stocking densities has increased. However, the farmers are aware of the fact that if they do not keep up their management standard as well, their total score will be decreased at the next inspection.

WELFARE IMPLICATIONS

It could of course be discussed if a farmer by improving the conditions for the birds should be allowed to worsen the birds' environment by applying a higher stocking density.

The scientific results on the effect of stocking density on bird welfare are confusing, not to say contradictory. It is very difficult to compare different investigations on stocking density, as there is a confusion between 'number of birds per area' and 'total bodyweight of birds per area'. In both cases, the age and weight at slaughter is essential information if any comparisons are to be made. Also, the international variation in what is considered as a 'high' stocking density is very large. There have been investigations on how stocking density affects production traits (such as growth rate and feed utilisation) and the environment (ventilation needs). From an animal welfare point of view, however, the health and behaviour of the birds are more interesting.

In general, with increasing stocking density, the plumage becomes dirtier and feathering deteriorates (Shanawany, 1988; Gordon, 1992). These problems can be attributed to the fact that as bird density increases the litter becomes dirtier and more moist (Proudfoot et al., 1979; Blokhuis & van der Haar, 1990) and the risk of birds climbing over each other increases (Proudfoot & Hulan, 1985; Gordon, 1992). The moist litter and elevated ammonia concentrations increase the incidence of 'breast blisters' and foot pad dermatitis (Greene et al., 1985; McIlroy et al., 1987).

Broilers can suffer from many types of leg problems, all of which can make it difficult for the bird to reach feed and water, and in many cases are considered to be painful (Kestin et al., 1992). Leg problems often have a complicated aetiology, with several interacting factors, such as genetic predisposition, feed composition, infections, and insufficient physical activity all affecting the incidence (FAWC, 1992; Kestin et al., 1992). With an increase in stocking density birds tend to move around less, and Grashorn & Kutritz (1991) showed that broilers were more plagued by leg problems when kept at a higher stocking density (29 birds/m^2) than at a lower one (20 birds/m^2).

A majority of published studies have noted no effects of stocking density on mortality, even at levels that should be regarded as extreme (see for example Scholtyssek & Gschwindt-Ensinger, 1983; Shanawany, 1988). Mortality merely reflects the number of animals becoming so sick that they die prior to slaughter and thus is a measure of little value when judging a bird's state of health from an animal welfare point of view, unless the cause of death is known.

It is always difficult to assess the degree of stress that animals experience, especially over longer periods. Stress in chickens has been studied primarily in connection with handling and transport and, consequently, little is known about stress during the rearing period. Neither Siegel (1960) nor Bolton et al. (1972) noted any differences in size between adrenal glands from young chickens kept at a high stocking density (approx. 22 birds/m^2) and those kept at a lower density (11 birds/m^2).

Considering the world-wide interest in broiler farming, it is surprising how few studies that have been carried out on the behaviour of broilers. There are, however, several studies on the birds' movement patterns and the use of total available area. The movement patterns have an important influence on the chickens' feed and water consumption, and thus indirectly affect their health. Lewis & Hurnik (1990) documented a large individual variation in movement patterns. As stocking density and age (body weight) increase, the birds spend less time in motion, move shorter distances and devote less time to pecking and scratching in the litter. This has been ascribed to the fact that as stocking density increases, it becomes more difficult for the birds to perform these behaviours without bumping into one another. This problem is most pronounced towards the end of the rearing period, when the birds are largest (Lewis & Hurnik, 1990; Newberry & Hall, 1990).

When evaluating research on the effects of stocking density it is almost impossible to avoid the effect of group size, as changes in the stocking density is usually achieved by changing the number of birds on a given area. If not, the confounding factor will instead be the total available floor area. Feeding and drinking space per bird is another common confounder. It is also very rare to see investigations where the ventilation capacity has been adjusted to the stocking density. The idea behind the 'Care Programme' is that the improvements in facilities, e.g. alarm systems, ventilation capacity or maximum distance to feed and water dispensers, and improvements in management, should clearly overrule any disadvantages from the increase in stocking density.

THE FOOT-HEALTH PROGRAMME

There have, however, been some serious discussions about the welfare implications of the Care Programme. Veterinarians and welfare activists have been worried that the limit has already been reached, and that there is no proper control of the birds' state of health from an animal welfare point of view. It has been suggested that the disease recording done by the meat inspectors at the abattoir has been focused only on meat hygiene aspects, and that signs of general unthriftiness or maltreatment in broiler flocks have either not been recorded or not followed up. As a result of discussions on this issue, the SBA in 1994 decided that they wanted a new parameter entered into the Care Programme. This parameter, at that time undefined, should somehow give information on the rearing standard during the birds' entire life. It should be easy to standardise, cheap to monitor and not require any major extra resources. Thus the Broiler Foot-Health Programme was created, as a part of the Care Programme.

There were several reasons for choosing the birds' foot-health status as an indicator of their welfare during the rearing period. The main reason was that foot-pad dermatitis is closely correlated to the rearing conditions. Foot-pad dermatitis is a type of contact dermatitis where lesions appear on the plantar regions of the broilers' feet (Greene et al., 1985). The lesions are thought to be caused by a combination of moisture and chemical irritants in the litter (Nairn & Watson, 1972; Harms et al., 1977; Greene et al., 1985; Martland, 1985; McIlroy et al., 1987). The lesions may be mild, showing only hyperkeratosis and discoloration. In severe cases, however, swelling and erosions or ulcers can be seen (Martland, 1985).

Previous studies had indicated that foot-pad dermatitis could be a wide spread health problem in Swedish broiler flocks (Ekstrand et al., *in press*). This was considered to be an animal welfare issue, as severely affected birds are likely to suffer from their condition, which meant that there was a good reason for trying to reduce the disease frequency. A number of risk factors for wet litter, such as litter depth and material (Shanawany, 1992; Ekstrand et al., *in press*), feed composition (McIlroy et al., 1987), stocking

density (McIlroy et al., 1987; Tucker & Walker, 1992; Gaardbo Thomsen, 1993), enteric *Campylobacter* infections (Neill et al., 1984), climatic conditions (Payne, 1967; McIlroy et al., 1987) and type of water equipment (Elson, 1989; Lynn & Elson, 1990; Tucker & Walker, 1992) have been identified. The measures that should be taken in order to avoid the risk factors for foot pad dermatitis are in good agreement with what is mentioned in the Care Programme, which means that farmers that have designed their broiler houses according to the programme will have good opportunities to avoid foot pad dermatitis in their flocks, if they also manage to look after the equipment and animals well. A good farmer should for example not only have sophisticated computer-controlled ventilation equipment but also have enough knowledge and interest to regulate the ventilation according to the growth of the birds, the outdoor air humidity and the condition of the litter. If not, the equipment will not always be able to save his broilers from getting foot-pad dermatitis in the Swedish climate.

The broiler foot-health surveillance programme was also developed in order to improve the knowledge about the occurrence of the disease and its spread in the population. The programme includes classification of foot-pad lesions and recording of flock prevalence at slaughter. For each flock information on producer, compartment, breed, feed manufacturer, region, abattoir, date of slaughter, age at slaughter, planned stocking density and reached stocking density is recorded. The surveillance programme also contains an advisory system. From every slaughtered flock 100 single feet are taken out for gross examination at the abattoir. The foot-pad lesions are assigned to three different classes: 0 = No remark: no lesions, only mild hyperkeratosis, no discoloration or scars; 1 = Mild lesions: superficial lesions, discoloration of the foot-pad; and 2 = Severe lesions: deep lesions, ulcers, scabs. Each flock is then given a total score, relating to different 'remark' levels.

When the programme was introduced, the official veterinary inspectors at the abattoirs were brought together and educated in the background and pathology of the lesions. They were also trained in the classification of broiler foot-health status. Each veterinarian was equipped with photographic material showing typical cases of different severity, including instructions on how to score 'borderline' cases. The veterinary inspectors are allowed to delegate the classification of the foot-health status to their assistants but have to countersign the report for each flock.

THE ADVISORY PART OF THE FOOT-HEALTH PROGRAMME

The different levels of flock prevalences leading to different 'remark' levels are connected to an advisory programme as a part of the surveillance programme. After the slaughter of each flock the producer is informed about the scores. The first time a producer delivers a broiler flock which is given any kind of 'remark' he is contacted by the advisor at the abattoir. If he continues to deliver flocks with the same level of lesions the rearing conditions of the following flocks are followed up. If no improvement is noticed the maximum allowed stocking density is decreased gradually, until the problems are solved. When the producer is able to deliver flocks classified as 'without remark', the stocking density is increased gradually again.

All data on background and foot-health scores have been reported to the central 'national standardiser' at the Swedish Poultry Meat Association. The programme is monitored by the national authorities, and summaries of the results have been presented twice yearly to the Broiler Committee of the National Board of Agriculture. The foot-health programme is now an integrated part of the Care Programme. The mean prevalence of severe foot pad dermatitis has decreased from 11 % in 1994 to 6 % in 1996.

WELFARE DURING LOADING - AN EXAMPLE OF THE PRE-TESTING PROCEDURE

Quite substantial research has been made in the area of broiler welfare during transport. For example, Nicol and co-workers (1991) have evaluated the effects of vehicle noise and horizontal motion on bird

behaviour. Physical injury, thermal stress and other aspects of stress during transport have also been rather thoroughly investigated (see for example Nicol & Scott, 1990).

In practice, monitoring the transport of birds is usually a matter of keeping records. The abattoirs in Sweden require the drivers to keep transport journals regarding loading time, departure time, arrival time and any extraordinary events during the process of transportation, such as ventilation failure or traffic delays. The proportion of birds being dead on arrival at the slaughterhouse is also recorded, and if any driver or any specific truck deliver a too high proportion of dead birds, an investigation is carried out.

The loading process has so far been less regulated. In most countries, manual catching of broilers has traditionally been carried out by catching the birds by a leg and carrying them together, three or four birds in each hand, to be placed in crates (Gerrits et al., 1985; Moran & Berry, 1988; Bayliss & Hinton, 1990). In some countries, including Sweden, the catchers have been advised to take the bird around the body and hold each pair of birds upright together with both hands on the way to the crates in order to reduce the risk of injuring the birds and thus downgradings (Gerrits et al., 1985; Parry, 1989).

In October 1995 a Swedish broiler company decided to import an automatic broiler catching machine from Finland. Such machines have been developed for several reasons. Manual catching is expensive and often rough and may cause injury to the birds. Apart from animal welfare considerations, the demand for good quality meat means that rejection rates must be kept at a low level. The poor working conditions for the manual catching teams are also an important factor.

The Swedish statutory on animal welfare demands that all new technical systems and new technical equipment for animal housing must be pre-tested from animal welfare point of view, before accepted for commercial use. The definition of 'new technique' is rather wide. Examples of recent testing areas are any loose housing systems for laying hens aiming at stocking densities exceeding 9 birds/m^2, and sand as litter material in cubicles for dairy cows. The broiler catching machine, which has a three-rotor sweeping pick-up head and two conveyor belts, was classified as 'new technique' by the SBA. The Department of Animal Hygiene at the University of Agriculture was given the commission of designing a study and carrying out the pre-testing. The aim of the study was to identify and compare the distribution of carcass rejection rates for damage related to handling during manual and mechanical catching of broilers, in order to provide information for an evaluation of the catching machine from an animal welfare point of view.

The results of this study are presently undergoing publication process. In summary, it can be said that the effect on the level of birds found dead on arrival at the slaughterhouse initially varied considerably regardless of catching method. During the last three-month period of the one-year study the mechanically caught flocks showed significantly higher frequencies of dead birds on arrival than manually caught flocks. During the same period, when running-in problems should be regarded as solved, the prevalence of bruises was still significantly higher in the mechanically caught flocks, whereas the prevalence of fractures did not differ significantly between the two groups. This suggests that there are still opportunities for further improvements of the machine, although it has now been accepted for commercial use in accordance with the Swedish animal welfare legislation. The acceptance of the machine is conditioned. The SBA requires the abattoir to regularly report rejection levels for both manually and mechanically caught flocks to the authorities involved. The board also requires the personnel that handles the machine to be properly trained for the task in order to minimise bird injury, and have set the maximum conveyor belt speed to 0.8 metres per second.

OTHER MONITORING PROGRAMMES

In the beginning of this paper it was said that the prevention of contagious diseases should partly be regarded as an animal welfare issue. Preventing the infectious organisms from getting close to the birds is the most efficient way of doing this, and therefore the Care Programme contains instructions on sanitary

barriers and hygiene measures. Still, it will always be necessary to check the status of production flocks, at least with regard to possible human pathogens such as *Salmonella* and *Campylobacter*. Such testing programmes are currently carried out in Sweden. Every broiler flock is tested for *Salmonella* spp. 10 days prior to slaughter (faecal samples) and for *Salmonella* (random samples) and *Campylobacter* at slaughter. This should be regarded as a meat hygiene issue, in order to maintain a certain standard of human welfare. The prevalence of these diseases in Sweden are low, each year an average of 2-6 broiler flocks are tested positive for *Salmonella*, and 12-14 % of the flocks are tested positive for *Campylobacter*.

This way of thinking has also influenced other parts of the Swedish animal production. In 1992, the Federation of Swedish Farmers (LRF) formulated a consumer policy called the 'On our way (to the world's cleanest agriculture)' programme. The main elements are good environment and animal care aiming at a sustainable agriculture. This policy is based on a number of different programmes concerning dairy, beef, pig and poultry production. Examples are programmes against *Salmonella*, the ban on growth-promoting use of antibiotics and a restrictive use of herbicides. Another example is the ethically based ban on using the carcasses from animals found dead for feed for other animals, and the decision (already in 1986) not to use meat- and bonemeal in ruminant feed. The 'On our way' programme also includes codes for good management practice on farms, and guidelines on environmentally friendly ways of handling waste.

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RISK FACTORS ASSOCIATED WITH SKIN ABRASIONS AND SOLE BRUISING IN PREWEANING PIGLETS

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Traumatic injuries on the limbs and feet of piglets are a common problem in most herds in the UK (MAFF, 1981). Results from a postal survey carried out by MAFF (1981) estimated that 57.7% of the herds which reared piglets on solid floors had piglets with injuries (leg or feet) and 36.8% of the herds which used perforated floors had injured piglets.

Limb injuries in piglets include abrasions, wounds or necrosis of the skin and hairless patches. Limb damage was reported for the first time in 1965 (Penny et al.), when several outbreaks of knee necrosis were described with a prevalence of 53% among the farms involved. Clinical observations (Penny et al., 1971) indicated that suckling piglets had superficial traumatic skin lesions which occurred in decreasing prevalence on the knees, fetlock, hock, elbow, and coronet. The most common foot lesions described in this age group ranged from bruised soles and heels (Penny et al., 1971) to total removal of the hoof wall and ascending bacterial infection (Smith, 1979).

Such damage is associated with surface roughness, the coefficient of friction (Christison & Farmer, 1983) and surface temperature (Phillips, et al., 1992) on solid floors, whilst floor properties such as the size of the slots (in relation to foot size), sharp edges of slats (Smith & Mitchell, 1976), and flooring material (i.e. plastic, aluminium, galvanized metal) (Lindenmann et al., 1985) were associated with lesions on perforated floors. Knee damage was less frequent on fully perforated floors with plastic covered woven wires than solid floors, but punched metal floors gave a damage level similar to solid floors (Furniss et al., 1986). When different types of perforated floors only were compared in experimental studies, piglets on plastic-coated expanded metal or plastic coated wire had less foot lesions than those on galvanized expanded metal and galvanized woven wire (Lindenmann et al., 1985). However, comparison of the results of these studies is complicated because there is no standardization of injury scoring protocols or instrumentation for measuring floors (Webb & Nilsson, 1982). Other possible aetiological factors in foot and limb damage include genetics and nutrition (Kovacs & Szilagyi, 1973). Whether there are breed differences that interact with mineral deposition to various part of the claw that may affect foot strength, is still unknown.

The present epidemiological study was performed to estimate the prevalence and distribution of the most common skin and foot lesions in preweaning piglets and identify potential environmental and managemental risk factors associated with these lesions.

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MATERIALS AND METHODS

Farm selection

Thirteen breeding farms participated in this study. They were convenience selected (Martin et al., 1987) from a list provided by a large pig specialist abattoir in south west England. Selection was based on a farmer's willingness to participate in the study, and that they were contracted to supply 50-200 pigs per week to the abattoir.

Piglets selection

It was estimated that examining 4% of the pig population which was on the farm at the time of visit, would provide information on approximately 400 each of preweaning, weaning and growing finishing pigs. Thus, by using an infinite herd size and an expected prevalence of 50%, the prevalence of lesions could be estimated with +/- 5% accuracy (Epi Info 6.03, 1996).

The farmer was contacted by telephone at least 3 days prior to the farm visit, and asked to give the researchers, the precise number of preweaning piglets which were on the farm at that time. The number of piglets which had to be examined from the whole population in each farm was then calculated, and a list of random numbers selected using Minitab (Minitab Inc.)

Data on the piglets and their environment were collected on two separate recording sheets, by two researchers. One of the researchers counted (from left to right) and marked the selected piglets (FMH) and the other examined the piglets limbs and feet and the farrowing pen (NM).

Data collection on the farm

Information collected from the farmer: A personal interview-based questionnaire was designed and used to collect information on the herd, breeds used and general information on the construction of the farrowing buildings. The interview was taken by one of the researchers (NM) after a short inspection of the buildings with the stock owner.

On farm examination of piglets: Counting always started from the pen nearest to the door and from the left row of pens if there was more than one row in the building. Piglets were marked, restrained and examined for skin and foot lesions, using a pre-tested protocol. The lesions observed are defined below:
Skin abrasions: the skin surface on the cranial surface of the limb was worn away.
Sole bruising: congestion and bruising of the solar corium presenting as a dark red pigmentation on the volar surface of the foot.

The lesions were recorded as a present or absent and their location i.e. lateral and medial digit and left or right and front or hind foot was recorded.

The date of birth, litter size, number of pigs born alive, weight (using suspended scales, Salter, 235-10S, weighing from 0 - 20 kg in 20 g increments) and sex were also recorded.

Data collected from the farrowing pen :The following definitions were used:
Wear of the floor surface: Damage or change to the floor surface in the course of normal use. This was scored in front of the pop hole, in front of the drinker or the feeder, in the lying area, in the dunging area, between joints (the area in the pen where the solid concrete floor adjoined with the perforated floor).
Wetness of the floor surface: Surface covered in water, urine or wet food. This was scored in front of the pop hole, in front of the drinker or the feeder, in the lying area and in the dunging area.
Aggregate exposed: Aggregate exposed on the solid concrete floor surface.
Steps and their location in the pen: A raised flat surface in the pen (e.g. a block). Its exact location in the pen (i.e. in front of the pop hole, in front of the drinker, in front of the feeder, in the lying, in the dunging area) was recorded.
Difference in height between the solid and the perforated area: A difference in the height between the solid and the perforated area was measured in cm.

Data Input

The data were loaded onto a database (Foxprow 3.2, Microsoft) when the farm visits were completed. Two different databases were set up: one consisted of data collected from each selected farrowing pen and the other containing data from the piglet examinations. In the databases each row corresponded to one pen or piglet respectively. Skin abrasions at different sites (i.e. left and right limb, carpal, metacarpal, digital lesions) and sole bruising on each digit (8 digits per piglet) were placed in separate columns. Information on floor materials and the management of each farrowing building was also entered onto the database by column. Errors were checked by running frequency distributions of the variables, unusual values and obviously incorrect coding were re-checked from the recording sheets.

Data Analysis

Univariate analysis was performed in Epi Info 6.03 (Dean et al. 1996). Frequency distributions were used to describe qualitative data, and means and standard errors to summarize quantitative data.

A pig was defined as affected with skin abrasions if it had at least one fore limb affected with one lesion located at any of the following sites: carpus, metacarpus, digit. The proportion of piglets with skin abrasions on the front limbs was calculated as shown below:

$$\text{proportion of piglets with skin abrasions} = \frac{\text{number of piglets with at least one skin abrasion on any site on front limbs}}{\text{total number of piglets examined (264)}}$$

The prevalence of lesions at each site was calculated as below, using the carpus as an example:

$$\text{prevalence of carpal skin abrasions} = \frac{\text{number of piglets with at least one carpal skin abrasion}}{\text{total number of piglets examined (264)}}$$

A pig was defined as affected with sole bruising if it had at least one affected digit on the front or hind feet.

$$\text{proportion of piglets with sole bruising} = \frac{\text{number of piglets with at least one digit affected with sole bruising}}{\text{total number of piglets examined (264)}}$$

Exact 95% confidence intervals for binomially distributed data were calculated using a program written in Foxprow 3.2 (Microsoft). The confidence limits were calculated as: 95% CI= $p \pm 1.96$ s.e., where s.e.=$\sqrt{\{ p (1-p)/n\}}$, (p= number of cases, n= number of animals examined).

The relative risk (RR) and 95% confidence intervals for the relative risk were calculated to estimate the strength of the associations between skin abrasions, sole bruising and the environmental or managemental exposures. For the simple and the multiple analysis the critical probability for significance was set at 0.05 for a two-tailed test.

Forward stepwise logistic regression was performed to determine factors associated with an increased or reduced risk of skin abrasion and sole bruising in EGRET (EGRET, 1991). Two logistic regression models were developed one with skin abrasions as the outcome variable and the other with sole bruising. All the explanatory variables that were significant at a level of $P \leq 0.25$ in the univariate analysis, were tested in the model. Each explanatory variable was fitted and then removed from the logistic regression model and the explanatory variable that led to the greatest reduction in the likelihood ratio statistic (LRS) was left in and then the remaining variables were re-tested in the model. The criteria for final inclusion in the model was a significant reduction in the LRS at the 5% level ($P \leq 0.05$).

RESULTS

A total of 264 piglets from 231 litters were examined and consequently 231 individual farrowing pens were measured. Two piglets and their pens were not examined. There were 130/264 (49.2%) male piglets and 134/264 (50.7%) female piglets examined.

Description of the farms

The farm visits took place between 25/08/1995 and 30/11/1995 and were accomplished in 29 visits. The farms were located in 3 counties. Eight of the farms were situated in Wiltshire, 4 in Avon, and 1 farm in Hampshire. There were 2 nucleus, 3 multiplier and 8 commercial units (breeder to finishing). The breeds used were: Large White (3 farms), Large White X Landrace (6 farms), Large White X Duroc (1 farm), Large White X Landrace X Duroc (1 farm) and Large White X Landrace X Hampshire (2 farms). The mean number of sows per farm at the time of visit was 322 and this ranged from 123-1000 sows. The mean number of preweaning piglets per farm was 523, and ranged from 159-1176 piglets. In 10/13 (76.9%) herds sows were kept in farrowing crates, while 1/13 (15.4%) herds kept sows in solari farrowing pens, 1/13 (7.7%) in farrowing crates and solari pens and 1/13 (7.7%) outdoors. There were 5/13 (38.5%) herds which weaned at 28 days of age, 3/13 (23.1%) herds at 21 days, 1/13 herds weaned piglets at each of 23, 24 and 26 days. The age of the piglets involved in this study ranged from 1-30 days (mean age: 11 days, s.e. 0.5 days). The weight of the piglets ranged from 1.20-7.8 kg (mean weight: 3.2 kg, s.e. 0.1 kg).

Prevalence and distribution of skin abrasion and sole bruising

Skin abrasions were located on three aspects of the front limbs: the carpus 95/264 (36.0%), the metacarpus 10/264 (3.8.0%) and the digit 18/264 (6.8%) (Table 1). All the piglets which had metacarpal or digital skin abrasions had a carpal skin lesion on the same limb. There were 95/264 (36.0%) piglets with at least one skin abrasion on any of the three sites of the front limbs.

There were 133/264 (50.4%) piglets with at least one digit with sole bruising, 86/264 (32.6%) piglets had at least one lesion on each foot and 58/264 (22.0%) piglets had all eight digits affected (Table 2).
There were 44/130 (33.8%) male and 51/134 (38.8%) female piglets affected with skin abrasions. 74/130 (56.9%) males and 59/134 (44.0%) females were affected with sole bruising. No significant difference was found between the prevalence of the lesions and sex.

Table 1: Prevalence and distribution of skin abrasions (total number of piglets examined = 264)

Lesions	Number	Prevalence (95% CI)
carpal skin abrasion	95	36.0 (30.2-41.8)
metacarpal skin abrasions	10	3.8 (1.5-6.1)
digital skin abrasions	18	6.8 (3.8-9.8)
skin abrasions on the front limbs	95	36.0 (30.2-41.8)

95% CI = 95% exact confidence intervals

Table 2: Prevalence of sole bruising (total number of piglets examined = 246)

Lesions	Number	Prevalence (95% CI)
at least one digit with sole bruising	133	50.4 (44.4-56.4)
at least one digit with sole bruising on each foot	86	32.6 (27.0-38.2)
all eight digits affected with sole bruising	58	22.0 (17.0-27.0)

95% CI = 95% exact confidence intervals

Univariate analysis

Age pattern of skin abrasions and sole bruising: Skin abrasions and sole bruising were seen in piglets from one day old to 28 days old. The modal age for the skin abrasions and sole bruising was 10 and 5 days respectively.

Breed: A significant difference in the prevalence of sole bruising and breeds was found with cross breeds Large White X Landrace, Large White X Duroc having less lesions than pure Large White.

Skin abrasions

Floor type: Piglets on concrete and round weld mesh floor (RR 1.95, 95% CI 1.26-3.03) or concrete and flat metal rods (RR 2.05, 95% CI 1.19-3.53) or concrete and part metal panels (RR 1.71, 95% CI 1.10-2.67) had a significantly higher prevalence of skin lesions than those kept on totally solid concrete floor (Table 3).
Bedding: Piglets on wood shavings had a significantly higher prevalence of skin abrasions (RR 1.72, 95% CI 1.01-2.94) than those kept on no bedding. Piglets were less likely to have skin abrasions when kept on deep bedding (straw or shavings) (RR 0.72, 95% CI 0.55-0.96) or when bedding covered the whole farrowing pen (RR 0.54, 95% CI 0.38-0.76) than those with no bedding (Table 7).

Floor characteristics: A worn floor surface in front of the drinker (RR 1.41, 95% CI 1.04-1.92) or at the point where the solid floor adjoined the perforated floor (RR 1.74, 95% CI 1.19-2.54) was associated with a higher prevalence of skin abrasions (Table 4). Piglets in pens with a wet floor surface in the dunging area were less likely to have skin abrasions than those on dry surfaces (RR 0.80, 95% CI 0.67-0.95) (Table 4).

Floor construction: Piglets kept on floors made from farm-mixed concrete had a higher prevalence of lesions when compared with those on floors made from ready-mixed concrete (RR 1.60, 95% CI 1.19-2.15) (Table 5). Piglets in pens with a top screed > 40 mm thick were less likely to have skin abrasions than those on floors with a top screed < 40 mm (RR 0.71, 95% CI 0.53-0.95) (Table 5).

Presence of steps in the farrowing pen: Piglets in pens with a step in front of the pop hole had a significantly higher prevalence of skin abrasions than those in pens with no step or a step elsewhere (RR 1.33, 95% CI 0.98-1.81) (Table 6).

Difference in the height between the solid and the perforated area: Piglets in pens with a difference in the height between the solid and the perforated area of > 2 cm were more likely to have skin abrasions (RR 2.05, 95% CI 1.45-2.91) than those kept in pens with a height between the solid and the perforated area of < 2 cm (Table 6).

Table 3: The effect of floor type on skin abrasions and sole bruising

Variables	Exposed	Piglets with skin abrasion			Piglets with sole bruising		
		N.	RR (95% CI)	P	N.	RR (95% CI)	P
Totally solid concrete	91	25	1.00 reference category		31	1.00 reference category	
Part concrete &							
Round weld mesh	41	22	1.95 (1.26-3.03)	0.006	37	2.65 (1.96-3.59)	<0.001
Flat metal rods	16	9	2.05 (1.19-3.53)	0.046	14	2.57 (1.83-3.61)	<0.001
Punched metal panels	51	24	1.71 (1.10-2.67)	0.029	22	1.27 (0.83-1.94)	0.372
Plastic slats	16	4	0.91 (0.37-2.27)	1.00*	10	1.83 (1.14-2.95)	0.060
Perforated floors**	27	10	1.35 (0.74-2.44)	0.474	13	1.41 (0.87-2.29)	0.121
Totally plastic slats	9	1	0.40 (0.06-2.64)	0.439	6	1.96 (1.14-3.37)	0.072*

N. = number of piglets with lesions
RR (95% CI)= relative risk and (95% confidence intervals for the relative risk)
* = Fisher's exact two tailed p value, P = significance probability
** more than one type of perforated floors

Sole bruising

Floor type: Piglets on concrete and round weld mesh floor (RR 2.65, 95% CI 1.96-3.59) or concrete and flat metal rods (RR 2.57, 95% CI 1.83-3.61), or concrete and plastic slats (RR 1.83, 95% CI 1.14-2.95) had a significantly higher prevalence of sole bruising than those kept on totally solid concrete floors (Table 3).

Bedding: The provision of straw was associated with a lower prevalence of sole bruising (RR 0.55, 95% CI 0.36-0.84). Pigs on deep bedding (straw or shavings) (RR 0.44, 95% CI 0.31-0.62) or on bedding which covered the whole farrowing pen (RR 0.40, 95% CI 0.26-0.60) were less likely to have sole bruising than those on no bedding (Table 7).

Floor construction: Piglets on floors made from farm-mixed concrete, were more likely to have of sole bruising than those on floors made from a ready-mixed concrete (RR 2.64, 95% CI 1.66-4.18) (Table 5).

Difference in the floor height between the solid and the perforated area: Piglets in pens with a difference in height between the solid and the perforated area of > 2 cm had a higher prevalence of lesions (RR 1.57, 95% CI 1.21-2.02) than those in pens with level floors or a difference in the height between the solid and the perforated area of < 2 cm (Table 6).

Table 4: The effect of different floor characteristics on skin abrasion and sole bruising

Variables	Exposed	Piglets with skin abrasion			Piglets with sole bruising		
		N.	RR (95% CI)	P	N.	RR (95% CI)	P
Wear in front of drinker	48	25	1.41 (1.04-1.92)	0.016	30	1.40 (0.95-2.05)	0.089
Wear between joints*	43	26	1.74 (1.19-2.54)	<0.001	22	1.02 (0.73-1.42)	0.956
Wetness in dunging area	95	25	0.80 (0.67-0.95)	0.020	45	0.91 (0.71-1.17)	0.545
Exposed aggregate	11	5	1.18 (0.68-2.04)	0.728	9	2.80 (0.80-9.88)	0.068

N. = number of piglets with lesions
RR (95% CI)= relative risk and (95% confidence intervals for the relative risk)
P = significance probability
* the point where the solid floor adjoined the perforated floor

Table 5: The effect of floor construction on skin abrasion and sole bruising

Variables	Exposed	Piglets with skin abrasion			Piglets with sole bruising		
		N.	RR (95% CI)	P	N.	RR (95% CI)	P
Ready mixed concrete	121	40	1.00 reference category		52	1.00 reference category	
Farm mixed concrete	74	43	1.60 (1.19-2.15)	0.001	58	2.64 (1.66-4.18)	<0.001
Top screed <40 mm	85	47	1.00 reference category		58	1.00 reference category	
Top screed > 40 mm	81	30	0.71 (0.53-0.95)	0.027	43	0.68 (0.46-1.00)	0.065

N. = number of piglets with lesions
RR (95% CI)= relative risk and (95% confidence intervals for the relative risk)
P = significance probability

Table 6: The effect of pen design on skin abrasion and sole bruising

Variables	Exposed	Piglets with skin abrasion			Piglets with sole bruising		
		N.	RR (95% CI)	P	N.	RR (95% CI)	P
No step or step elsewhere	204	71	reference category		105	reference category	
Step in front of pop hole	47	24	1.33 (0.98-1.81)	0.050	28	1.20 (0.83-1.75)	0.400
Level floor surface	157	51	1.00 reference category		78	1.00 reference category	
Difference in height 1-2 cm	67	26	1.19 (0.82-1.74)	0.448	34	1.02 (0.77-1.36)	1.00
Difference in height 2.1-3 cm	27	18	2.05 (1.45-2.91)	0.001	21	1.57 (1.21-2.02)	0.012

N. = number of piglets with lesions
RR (95% CI)= relative risk and (95% confidence intervals for the relative risk)
P = significance probability

Table 7: The effect of bedding on the development of skin abrasions and sole bruising and its association with bedding

Variables	Exposed	Piglets with skin abrasion			Piglets with sole bruising		
		N.	RR (95% CI)	P	N.	RR (95% CI)	P
No bedding	54	12	1.00 reference category		34	1.00 reference category	
Straw	52	14	1.21 (0.62-2.37)	0.736	18	0.55 (0.36-0.84)	0.006
Shavings	167	64	1.72 (1.01-2.94)	0.045	88	0.84 (0.65-1.07)	0.245
Deep bedding	123	56	0.72 (0.55-0.96)	0.04	34	0.44 (0.31-0.62)	<0.001
Sparse bedding	87	43	0.78 (0.58-1.05)	0.162	43	0.78 (0.58-1.05)	0.162
Bedding only in creep	118	68	0.92 (0.71-01.18)	0.621	54	0.73 (0.55-0.97)	0.053
Bedding over whole pen	92	31	0.54 (0.38-0.76)	0.001	23	0.40 (0.26-0.60)	<0.001

N. = number of piglets with lesions
RR (95% CI)= relative risk and (95% confidence intervals for the relative risk)
P = significance probability

Logistic regression analysis

When the above exposures were fitted into the logistic regression model the following remained significant.

Skin abrasions: An increase in the prevalence of skin abrasions was associated with solid concrete and round weld mesh floors (OR 3.05, 95% CI 1.37-6.82) (when compared with all the other floor types), wear at the point where the solid floor was adjoined to the perforated floor (OR 4.14, 95% CI 1.93-8.85) and sparse wood shavings in the pen (OR 2.10, 95% CI 1.10-4.16). A decrease in the prevalence of skin abrasions was associated with a wet dunging area (OR 0.50, 95% CI 0.26-0.95) (Table 8).

Sole bruising: An increase in the prevalence of sole bruising was associated with solid concrete and round weld mesh floors (OR 48.37, 95% CI 5.97-392.0) (compared with all the other floor types), and with exposed aggregate (OR 5.03, 95% CI 1.10-23.95). Both sparse (OR 0.19, 95% CI 0.04-0.86) and deep straw bedding (OR 0.10, 95% CI 0.01-0.83) reduced the prevalence of sole bruising (Table 9).

Table 8: Logistic regression model of factors associated with the presence of skin abrasion

Variable	Coefficient	Odds ratio (95% CI)	P value (LRS)
Constant	-1.150		
Wearing where solid adjoined perforated area	1.420	4.14 (1.93-8.85)	<0.001 (10.95)
Concrete and round weld mesh floor	1.117	3.05 (1.37-6.82)	0.003 (34.45)
Sparse shavings	0.742	2.10 (1.10-4.16)	
Deep shavings	0.619	1.86 (0.83-4.13)	0.051 (5.97)
Wet dunging floor surface	-0.069	0.50 (0.26-0.95)	0.033 (4.56)

(95% CI) = 95% confidence intervals for the odds ratio, LRS= Likelihood ratio statistic

Table 9: Logistic regression model of factors associated with the presence of sole bruising

Variable	Coefficient	Odds ratio (95% CI)	P value (LRS)
Constant	-0.112		
Concrete and round weld mesh floor	3.879	48.37 (5.97-392.0)	<0.001 (31.27)
Sparse straw	-1.680	0.19 (0.04-0.86)	
Deep straw	-2.280	0.10 (0.01-0.83)	<0.001 (14.59)
Aggregate exposed	1.616	5.03 (1.10-23.95)	0.021 (5.33)

(95% CI) = 95% confidence intervals for the odds ratio, LRS= Likelihood ratio statistic

DISCUSSION

This is the first cross sectional study on the risk factors associated with the prevalence of skin and foot lesions in preweaning piglets in the UK.

The variable most consistently associated with a high prevalence of skin abrasions and sole bruising was the part concrete and part round weld mesh flooring. During suckling, relatively small areas of the front legs support the body weight and take the abrasions created by the thrusting action of piglets struggling to maintain their position at the udder. This may concentrate the impact on a small weight bearing surface and therefore cause more mechanical destruction of the skin tissue. The fact that carpal skin abrasions were the most prevalent supports the hypothesis that the body weight is distributed over the area of the carpal joint especially while suckling and therefore the greater the pressure per unit area the greater the risk of skin structural failure.

The high prevalence of sole bruising may be attributable to two factors: the large void size and the shape of the slats. When a piglet is walking or standing on slats, it has to support its weight on a smaller surface area than on solid floors. This increases the force which the weight bearing surface of the foot takes and therefore increases the risk of sole bruising. This will be exacerbated when the slats

are rounded and the weight bearing width of the slats is reduced. The rounded surface of the slats may also fail to provide a grip for the piglets while walking and consequently slipping could increase the risk of skin injury or sole bruising. High pressure under the pig's foot also occurs when piglets walk on an uneven surface such as steps, uneven floor surfaces and exposed aggregate and these were important risk factors for sole bruising in this study. Conversely, a reduction in the prevalence of sole bruising was found when straw was present. Straw provides a more resilient substrate for all animals to walk and rest on. In this study, deep bedding (straw or shavings) and bedding which covered the whole farrowing pen, was associated with a low prevalence of sole bruising and skin abrasions.

However, skin abrasions were more prevalent in piglets kept on wood shavings than on no bedding. Shavings move when piglets scrabble whist suckling, particularly when they are sparse. Tissue damage may occur if sharp particles protruding from the floor surface shear off skin tissue or penetrate the skin. Histopathology carried out on these lesions indicated that the lesions are localized i.e. the infection and damage appeared to move from a superficial to the deeper structures of the carpus (Green, personal communication). The rougher the floor surface is the more likely it is that the weight bearing surface of the limbs would be abraded. An increase in the prevalence of skin lesions was shown in piglets kept in pens with a worn surface in front of the piglets' drinker and where the solid floor adjoined the perforated floor. Furthermore, piglets in pens with a step in front of the pop hole or a difference in the height between the solid and the perforated area of more than 2 cm had a higher prevalence of skin abrasions. It is possible that when piglets walk over a step they fall and slide on the floor surface on their knees or they scuff their knees as they step up.

Piglets on floors made from farm-mixed concrete had higher prevalence of skin abrasions and sole bruising than those on ready-mixed concrete. There is a possibility that farm-mixed concrete was not of a good quality since the consistency and mix proportions may vary from load to load compared to the standard mix proportions used to prepare the ready-mixed mixtures. A factor influencing the quality of the concrete mixture is the water added to the mixture (Barnes & Mander, 1986), the more water added the weaker and less durable the concrete will be.

Floor screeds on top of an insulated layer which are 30-35 mm thick will remain cold (Baxter, 1984). With solid floors, if they are to be considered thermally effective for pigs, the insulation layer must be as near to the surface as possible. A top concrete screed more than 40 mm thick was associated with a lower prevalence of skin abrasions. Farrowing pens are often equipped with a radiant heat source to warm the piglets creep area and this can raise the floor temperature above the air temperature (Phillips et al., 1992). Simulation studies predict that such warm floor surfaces will cause appreciably more leg injuries to piglets because frictional heat build-up on warm surface can increase the skin temperature to a point where skin strength begins to deminish (Phillips et al., 1992). It is possible that with a top screed more than 40 mm thick the floor temperature is not raised and therefore piglets are less likely to damage their skin or piglets move to a bedded area of the pen in preference to a cold surface. Piglets will not lie on a wet cold area of the floor unless heat stressed (Baxter, 1984), consequently they will lie away from the excretory area. Similarly, if there is a spillage of water around the drinker, pigs will avoid that wet floor area unless the floor under the drinker is slatted. A wet floor surface in the dunging area and around the drinker was associated with a lower prevalence of skin abrasions. Animals also have difficulty in finding a sure footing when the floor surface becomes wet (Robertson & Anderson, 1979) and therefore they avoid walking or resting on these surfaces. The majority of the totally solid floors had wet dunging surfaces and all had from a little to an ample amount of bedding, so it is likely that the piglets preferred resting on a dry bedded area than the wet, cold area.

The representiveness of these results is dependent to a great extent on the representiveness of the herds involved in this study. The sow herds for this study, were not randomly selected. However, the study herds were similar to herds in the UK, in the terms of the herd size and type of housing. The

average number of sows in breeding herds in the UK is estimated 343 sows (MLC, 1996) and it is similar to the mean number of sows kept by the farms in this study (322). 7/13 (53.8%) study farms used totally solid floors and 30.8 % (4/13) used part concrete and part perforated floors. Data from previous reports (MLC, 1986) estimate that 65.7% of the pig herds in the UK use totally solid floors in the farrowing accommodation and 29.0% part concrete and perforated floors.

The majority of the data on herd environment was recorded in a simple and reliable manner, particularly since clear and precise definitions were used to record the skin abrasions, sole bruising, and the environment. It should be a definite advantage that data were collected by one individual (NM) since observer bias was avoided. This applies particularly to observations, such as the assessment of the condition of the floor or the examination of piglet hooves and limbs, which although classified descriptively, required a uniform judgment.

Recall bias and other similar sources of error must be considered in all field surveys, involving stock owner interviews (Backström, 1973). In order to eliminate such effects as much as possible, all fundamental data were related to the situation current at the time of inspection. A short inspection of the buildings with the stock owner took place before the interview, in all the farms. So, considerable care was taken to combine the interview with the inspection of the stock, and to formulate questions on management and housing in a manner satisfactory from the veterinary point of view, but at the same time comprehensive and clear to the stock owner. The greatest attention was paid to questioning technique since the majority of the questions were short and closed (Vaillancourt et al., 1991) and had the same format for all types of accommodation.

This study has shown that piglets kept on concrete and round weld mesh floors had a higher prevalence of skin abrasions and sole bruising. Sparse shavings increased the risk of skin abrasions, while deep or sparse straw decreased the risk of sole bruising. Wearing of the surface at the point where solid floor adjoined the perforated floor was associated with an increased risk of skin abrasions while exposed aggregate in the farrowing pen increased the risk of sole bruising. These traumatic lesions are seen at a very early age and they are highly prevalent. A question must remain over the fulfillment of piglets' comfort and behavioural needs.

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OPEN SESSION

PRE-HARVEST FOOD SAFETY - AN EPIDEMIOLOGICAL APPROACH TO REDUCING FOOD BORNE PUBLIC HEALTH RISKS

TH. BLAHA*

Although mandatory meat inspection has been regarded as sufficient to guarantee safe pork over almost 100 years, new approaches to food safety and pork quality are becoming necessary. There are four major reasons for this need:

1) The consumer's concerns with food safety are increasing:

   It is true that meat has never been as safe as today, but the perception of the risks due to meat is that there are more risks to human health than ever. This general recognition is highly supported by the media. The urban consumer does not differentiate between commodities or diseases so that reports on BSE and E. coli O:157 H:7 do not only have an adverse impact on beef, but on meat in general. The concerns with food safety in meat focus mainly on pathogens, antimicrobial and chemical residues, and hormones.

2) Modern agriculture is more and more often being attacked by the medical society and consquently by the public:

   The latest and most serious attack is that of the Director General of the World Health Organisation (WHO), who stated in his World Health Report 1996: "....Making matters worse are modern types of food production. Antimicrobials are used in meat production to increase growth, but not usually in sufficient amounts to kill microbes. Drug-resistant bacteria are then passed through the food chain to the consumer."

3) Food safety issues are increasingly used as marketing tools, nationally and internationally:

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Nationally: Advertisements for meat utilise food safety concerns more and more often e.g. the grocery chain "Whole Foods Market" in several major cities of the USA advertises: "....Our fresh meat and meat products come from animals raised naturally without hormones and antibiotics...." It is obvious that such statements create new consumer demands and increase the distrust in meat without such "labels".

Internationally: Trade barriers that prevent national meat industries from getting access to international markets are more and more based on food safety concerns. The Danish salmonella control programme is successfully used to increase pork exports from Denmark. The Japanese requirements for pork imports into Japan are mainly referring to food safety issues.

4) The traditional mandatory meat inspection is still indispensable, but unable to control and prevent the emerging foodborne pathogens that nowadays pose risks to human health.

   In the days of the so-called classical zoonoses, diseases such as tuberculosis and brucellosis caused both clinical diseases that could be recognized at farm level and lesions that could be recognized during meat inspection at slaughter. The emerging pathogens of today such as salmonella, toxoplasma, trichinella, campylobacter and yersinia are only detectable through targeted monitoring systems, since they do not cause clinical symptoms in affected animals nor lesions that could be helpful to recognize contaminated carcasses.

The majority of the real and perceived reasons for the increased concerns with the safety and quality of pork apply to the pre-harvest area of the food production chain. Thus, the traditional mandatory meat inspection as a post-harvest food safety measure has no potential for major improvements in the safety and quality of pork. Therefore:

1) Pre-harvest food safety programmes implementing the rules of the HACCP (Haphazard Critical Control Points) concept at farm level from breeding to the slaughterhouse gate have to be added to the existing HACCP programmes from the slaughterhouse to the retailer. Quality assurance systems including pre-harvest food safety at farm level are the precondition for any certification procedure.

2) Governmental food safety programmes and market-driven food safety/pork quality programmes must be coordinated.

   All of this means that the swine industry is facing remarkable changes in the years to come, which is both a challenge and an opportunity for pork producers, packers/processors and the allied industry as well as for the veterinary profession. Competitiveness of pork production will be more dependent on the reliability of the safety and quality of meat than on quantity and price.

   The role of the pork producer is changing from just raising pigs to being an indispensable part of the food production chain that supplies a product which is

the basis for the production of a wholesome, safe and high quality pork. Likewise the veterinarian's focusing on herd health/productivity changes to focusing on supporting the pork producer in order to provide pigs with quality properties to meet the demands of packers/processors, wholesalers, retailers and the consumer. Apart from consistent herd health management, the swine practitioner will more and more be involved in on-farm pathogen control and on-farm residue prevention programmes, monitoring systems and verification procedures as demonstrated in Fig. 1.

The Veterinarian's Role in Food Animal Practice Changing over Time

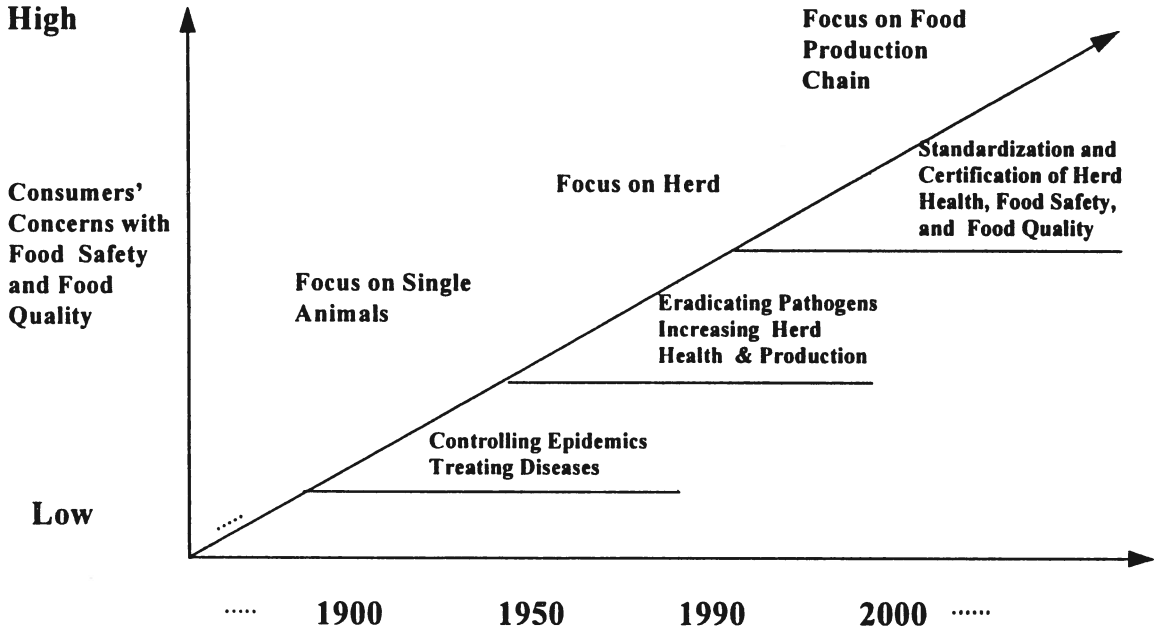

Fig. 1

To take advantage of this development i.e. to turn the new challenges due to upcoming food safety/quality concerns into an opportunity, it is necessary to introduce epidemiological methods for data collection and processing and analyzing. The implementation of an information feedback system, as demonstrated in Fig. 2 on the next page, is needed to have the management tool at hand that combines data from the slaughter plant (disease-related lesions, slaughter deficiencies, and monitoring results) with data on animal health (diseases, pathogens, drug use) and the performance of the herds of origin.

Information Feedback System "Pig HEALTH"

along the pork chain for improving animal health and production, product quality, and food safety.

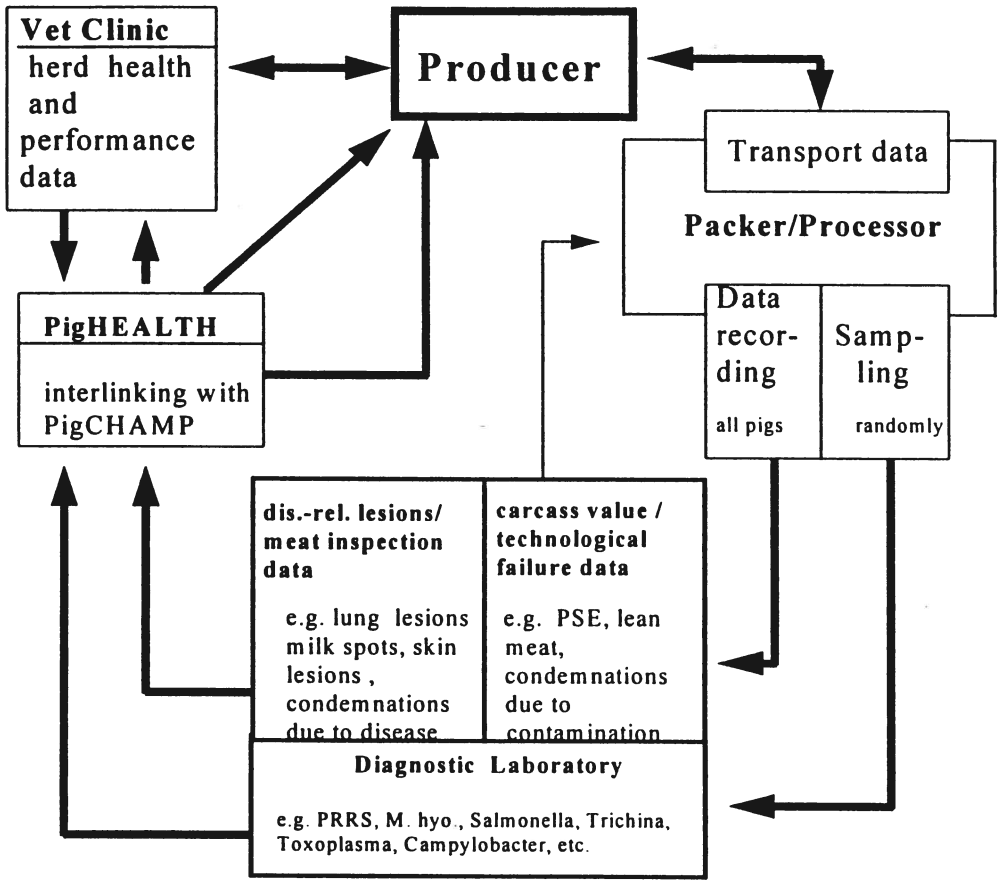

Fig. 2

The criteria that are mentioned in Fig. 2 are not complete, but suitable to start a distinguished pre-harvest food safety programme. However, once such an information system has been implemented, it is quite easy to include additional food safety/quality data to the system such as data on:

- pre-harvest residue risk reduction
- trichinella-free certification
- pre-harvest emerging pathogen reduction
- animal well-being improvement
- environmental controls

Since the majority of the data to be recorded, processed and turned into valuable information that makes on-farm food safety programmes possible are more or less "veterinary data", the swine practitioner using epidemiological methods plays the key role in developing effective quality assurance systems "from conception to consumption".

Producing safe pork which is certified will make our clients a competitive, publicly accepted and appreciated component of the food production chain. The swine practitioner will play an active role in this process as a catalyst. But he will also be the indispensable adviser and verifier that supports "his" pork producers' group to be always one step ahead of the rest, by which it will not only "stay in business", but even gain and maintain market leadership.

METHODS FOR EVALUATION OF INTERVENTION STRATEGIES TO CONTROL *CRYPTOSPORIDIUM* IN DRINKING WATER SUPPLIES

R.S. BARWICK AND H.O. MOHAMMED*

Cryptosporidiosis is a gastrointestinal disorder caused by the parasite, *Cryptosporidium parvum* (Dubey et al., 1990). This disease can be transmitted through water and has been implicated in several water-borne disease outbreaks throughout the world, including North America and the United Kingdom (Goldstein et al., 1996; Hayes et al., 1989; Joseph et al., 1991; Leland et al., 1993; MacKenzie et al., 1994; Richardson et al., 1991; Rush et al., 1990; Smith et al., 1989; Solo-Gabriele & Neumeister, 1996).

Cryptosporidium is a coccidian which can infect the small intestine of most mammals, including humans (Dubey et al., 1990). The organism invades the host who then sheds an infective oocyst into the environment and into the water through faeces (Dubey et al., 1991). *Cryptosporidium parvum* and *Cryptosporidium muris* are the species most commonly associated with infection in humans and other mammals. Cryptosporidiosis can cause gastroenteritis and severe diarrhoea in both immunocompetent and immunocompromised individuals. *Cryptosporidium spp.* has been found to be prevalent in bovine populations (Anderson & Hall, 1982; Garber et al., 1994; Moore & Zeman, 1991; Ongerth & Stibbs, 1989; Xiao et al., 1993) and contamination of the environment by these populations is possible through excretion and through the process of manure spreading on farmland. Runoff from the land contaminated by these sources serves as a vehicle through which the *Cryptosporidium* oocysts can travel into water sources. By infecting the environment, it is recognized that bovine operations serve as a potential source of exposure of the human population to *Cryptosporidium*. As a zoonotic pathogen, *Cryptosporidium* is a threat to humans in drinking water.

Studies have determined the *Cryptosporidium* oocyst is present in drinking water supplies throughout parts of the world (LeChavellier, et al., 1991). Because *Cryptosporidium* has also been shown to resist pressures such as temperature and disinfectants, it has great potential to contaminate and survive in the environment (Campbell, et al., 1982; Fayer & Nerad, 1996; Robertson, et al., 1992). It is also able to survive common drinking water treatments such as chlorination (Korich, 1990; Smith & Rose, 1990). It is therefore of great concern as to how to prevent this parasite from infecting community drinking water. Currently, filtration of water supply systems is the common preventative measure although several of the Cryptosporidiosis outbreaks have been in filtered water supplies (Goldstein et al., 1996; Hayes et al., 1989; Leland et al., 1993; MacKenzie et al., 1994; Richardson et al., 1991; Solo-Gabriele & Neumeister, 1996).

Public awareness about waterborne diseases was heightened through media coverage of outbreaks of Cryptosporidiosis in the United States. As a result, several complaints from the public about the quality of drinking water or illness were forwarded from the engineering department of the water-borne disease sections. Due to the high cost of intervention in water supply systems, and the limited resources, careful decision analysis needs to be employed to determine what is necessary to provide the most cost effective strategy to assure water quality for communities. In due process, many factors need to be taken into consideration, such as the probability that *Cryptosporidium* exists in the drinking water supply, how likely it is to cause an outbreak, and how effective are the

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available water treatments. Communities who do not currently filter their water supply are faced with this difficult decision. It is known that current chemical treatment of drinking water is not effective in eliminating oocysts, however, as mentioned earlier, *Cryptosporidium* was reported in filtered water supplies. Currently, the New York City Watershed serves approximately 13 million people. New York City does not have a filtration system and uses chemical treatment as a prevention strategy. The city is under pressure from the EPA and the residents to provide the most efficient water purification methods, but also have to maintain a feasible prevention strategy. It would cost New York City approximately 8 billion dollars to design and build a water filtration system and additional millions to run it year to year. But when faced with *Cryptosporidium* outbreaks such as Milwaukee had in 1993, where an estimated 403,000 people were sick from a filtered water system, it is hard to conclude that filtration would be the solution. A decision-tree analysis is a method that can use complex information including the probabilities that the organism causes a problem and the cost of the possible outcomes.

We carried out a study to evaluate the cost-effectiveness of two intervention strategies to prevent waterborne diseases, namely Cryptosporidiosis, in water supply systems. We employed the decision-tree analysis approach to compare filtration to treatment of water supply systems in preventing outbreaks of Cryptosporidiosis.

MATERIALS AND METHODS

Construction of the decision-tree

The decision-tree analysis technique was used to estimate the expected value of testing information (Weinstein, et al., 1980). The decision-tree is composed mainly of decision alternatives (decision node), probability values associated with each decision outcome, and monetary values associated with each decision outcome. Each decision is represented by a decision or a choice node, generally indicated by a small square. At this point, a decision is made and lines or paths are drawn to indicate a choice and consequences associated with this choice. If the consequence is to make another decision, another square is drawn, or if the consequence has uncertainty associated with it then a chance node (or a circle) is drawn which is indicated by a circle. At each chance node there is potentially an infinite number of outcomes. From all chance and choice nodes, all possible results must be included and they must be mutually exclusive. The probabilities of all branches from one chance node must sum to 1.

Included in the tree were the decision alternatives of using a filtration system, or not. If the choice is made not to use a filtering system, the alternative on decisions must be made which are either to treat the water or not to treat (Figure 1). We differentiated these systems based on filtration and not chemical treatment. Therefore when we refer to a system as being filtered, we do not mean to imply there is no chemical handling of the water, but when we refer to a system as being treated only, we mean there is no filtration system used. If there is no treatment, this indicates that there is no filtration or chemical treatment of any kind.

Estimation of probabilities

Probabilities were determined based on the frequency the event occurred in these populations. For this study, events were defined as; 1) if the drinking water supply was filtered, treated, or had no preparation for consumption; 2) if a *Cryptosporidium* oocyst was detected in the water after it had been processed for consumption, either chemically treated, or filtered; 3) if this water supply system was implicated in an outbreak of Cryptosporidiosis.

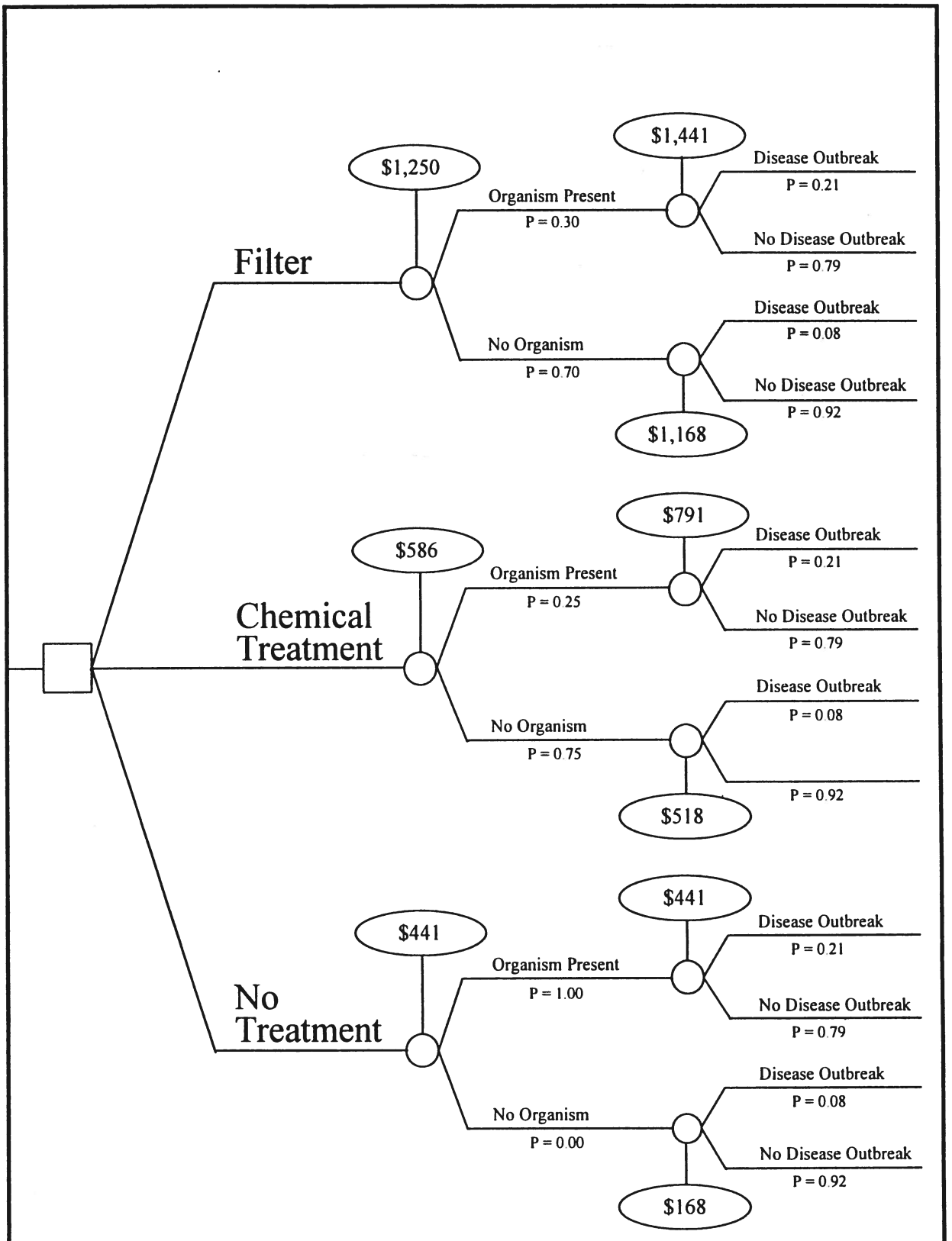

Figure 1-- Decision analysis of cost ($) of filtration water system vs. chemically treated water vs. no water treatment

For example, the probability that a filtered system had at least one sample with one oocyst detected in its finished water was determined by the proportion of systems that detected an oocyst out of the total number of filtered water systems from published studies (Goldstein et al., 1996; Hayes et al., 1989; Joseph et al., 1991; LeChavellier, et al., 1991; Leland et al., 1993; MacKenzie et al., 1994; Richardson et al., 1991; Rush et al., 1990; Smith et al., 1989; Solo-Gabriele & Neumeister, 1996). The probability that a filtration system did not detect a *Cryptosporidium* oocyst in its finished water was the proportion of filtration systems that tested *Cryptosporidium* free, to the total number of filtered drinking water systems included in these studies.

For those systems that did not have a filtration system and only chemically treated the drinking water, the probability that an oocyst was detected was the number of facilities testing positive for *Cryptosporidium* divided by the total number of chemical treatment systems included in this analysis. There was only one water supply that was not filtered or treated and that was included in this analysis as an untreated system (Solo-Gabriele & Neumeister, 1996).

Probabilities of disease outbreak were determined in a similar process. To determine the probability that a water supply system would have an outbreak if an oocyst was detected in finished water, the proportion of drinking water supplies testing positive for *Cryptosporidium* in their finished water was divided by the total number of drinking water supplies implicated in outbreaks. Moving down the tree, the probability that no disease outbreak occurred when an organism was found in finished waters, was the number of facilities with an organism present but no disease outbreak divided by the total number of systems with an organism detected in the finished water. Probabilities for the rest of the tree were constructed in a similar manner.

Cost considerations

Cost estimates were based on predictions of the cost and different treatment strategies to drinking water supplies. The cost of general chemical treatment of a drinking water source including flocculation, sedimentation, and chlorination, was estimated at $350 per person, through conversations with water supply experts. The average cost of implementation of a filtration system, was estimated at $1,000 per person (Bai, et al., 1995). This cost was determined by dividing the estimated cost of filtering the New York City water supply by the population of consumers of that water supply. The cost of illness was also considered and that was estimated at $2,100 per person based on a 3 day hospital stay, the cost of laboratory tests and fluids, and was determined through conversations with several hospital administrators.

Expected monetary loss

The expected monetary loss (EML) associated with each outcome was computed as the sum of the probabilities of and outcome (p_i) multiplied by the cost associated with that outcome (C_i):

$$EML = \sum_{i=1}^{n} (p_i C_i)$$

For example, the expected monetary loss associated with the use of a filter system is computed as:

((cost of filtration per person + cost of hospitalization) X (likelihood of an outbreak)
+ (cost of filtration per person) X (1 - likelihood of an outbreak))

The results of these computations gives the EML associated with the installation of a filtration system and the potential of detecting an organism in the water supply system.

Sensitivity Analysis:

Because there is uncertainty associated with estimates of costs of filtration we carried out a sensitivity analysis to evaluate the impact of changes in filtration costs on the decision outcome. Estimates of the cost of filtration were revised by two fold increment and 50% reduction and the EML associated with each intervention was recalculated. The efficacy of each strategy in reducing the likelihood of contamination of the water supply system was kept constant.

RESULTS

The result of the decision analysis comparing these strategies for controlling the contamination of the water supply system with *Cryptosporidium spp.* and managing the risk of Cryptosporidiosis in humans is shown in Figure 1. The EML associated with the installation of a filtration system was computed following the upper branch of the tree as:

$$[\{(\$1{,}000 + \$350)(0.21) + (\$1{,}000)(0.79)\}(0.3)] + [\{(\$1{,}000 + \$350)(0.08) + (\$1{,}000)(0.92)\}(0.7)] = \$1{,}250$$

The results of the computations of the EML is presented in Figure 2. Similar computations were made for the use of chemical treatment and no treatment strategies. The EML associated with the use of commercial treatment is $586 and the expected monetary loss associated with no treatment strategy was $441 (Figure 2).

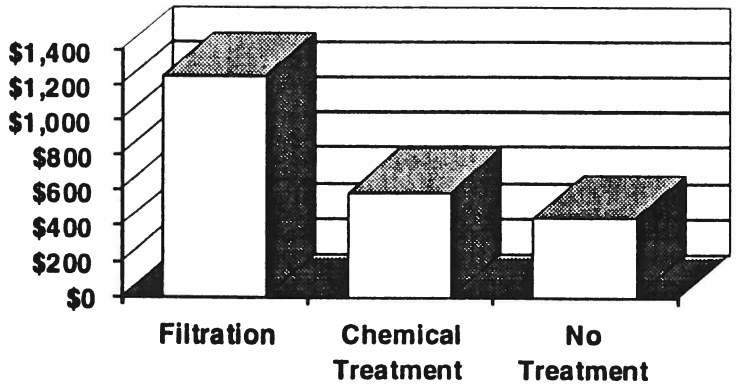

Figure 2. The expected monetary losses for the three intervention strategies

In comparing these strategies for the contamination control and risk management it appeared that no treatment has the least EML and thus is the strategy of choice. However, we caution the readers not to misinterpret the results of our analysis because estimate for the risk of contamination for this strategy was obtained from a single study that was conducted on a community that has a small risk of contamination.

Sensitivity analysis

Estimates for the costs of the three interventions were obtained from different sources. To assess the impact of the cost of filtration on the outcome we revaluated the decision tree using different estimates of the cost. The results of the sensitivity analysis are shown in Table 1. The EML associated with the filtration system increased by 4-fold in comparison with the chemical treatment strategy where the cost of filtration was doubled. Even when the cost of filtration was reduced by 50%, the EML for filtration remained high.

Table 1. Results of sensitivity analyses on expected monetary losses associated in change in cost of water treatment option

Expected Monetary Losses (EML)	Estimated cost of filtration $1000/person	Estimated cost of filtration $2000/person	Estimated cost of filtration $500/person
EML of filtration	$1,250.00	$2,250.00	$750.00
EML of chemical treatment	$586.00	$586.00	$586.00
EML of no treatment	$441.00	$441.00	$441.00

DISCUSSION

The evaluation of an intervention strategy from the policy perspective involves the comparison to an alternative intervention and the demonstration that a proposed strategy does more good than harm when implemented in usual circumstances. Such an evaluation requires the consideration of the effectiveness of these strategies as well as the resources needed to implement these strategies. The decision tree analysis is one of the approaches that allow for the evaluation of the decision making process and provides policy making with a tool that helps to support choices (Weinstein, et al., 1980). It is widely used in the medical decision making community and also in veterinary medicine (Carpenter & Norman, 1983; Mohammed, et al., 1990; Weinstein, et al., 1980; White & Erb, 1980).

The three strategies we evaluated here are the common practices that used to control and prevent water-borne diseases. OUI analysis showed that the use of a filtration system has the highest EML at $1,250 and chemically treating the water has an EML of $586, less than half that of a filtration system. This may indicate that chemical treatment is the most feasible solution. Chlorination, the most commonly used chemical water treatment, if implemented properly, has been proven to be an effective intervention strategy to control bacterial and viral contaminants, however, several studies have questioned its efficacy against *Cryptosporidium parvum* (Korich, 1990; Smith & Rose, 1990). Lippy (1986) reviewed the factors that influence the efficacy of chlorination in inactivating bacteria, virus, and protozoans. The study identified the concentration of chlorine, contact time, pH, temperature and interfering substance, organic and inorganic, as the factors which affect the efficacy of the intervention. It can also be seen in Figure 1 that probability of detecting *Cryptosporidium* in a chemically treated drinking water supply is at 25% after filtration. This inefficacy of chlorination has led some to insist that filtration should be the strategy of choice to prevent water-borne diseases.

The use of the filtration strategy is not without its short comings. Several studies have reported on outbreaks due to Cryptosporidiosis in filtered water supply systems (Goldstein et al., 1996; Hayes et al., 1989; Leland et al., 1993; MacKenzie et al., 1994; Richardson et al., 1991; Solo-Gabriele & Neumeister, 1996). Although filtration is the common strategy of choice by several large communities still some cities are hesitant to implement it. The main prohibitive factors in

implementing filtration is the cost. We calculated the expected monetary loss to be $1,250 per person with the use of a filtrated water system. In addition, the probability that *Cryptosporidium* is detected in a filtered water supply is still as high as 30%.

Although our analysis has shown that no intervention strategy has the least EML we would like to caution the reader about the interpretation of these results. Estimates of the probabilities for the presence of the organism in the water supply system under different intervention strategies were obtained from published literature (Goldstein et al., 1996; Hayes et al., 1989; Joseph et al., 1991; LeChavellier, et al., 1991; Leland et al., 1993; MacKenzie et al., 1994; Richardson et al., 1991; Rush et al., 1990; Smith et al., 1989; Solo-Gabriele & Neumeister, 1996). It would have been desirable to obtain these estimates from surveillance or cross-sectional studies. The estimate for the presence of the organism in water supply systems for filtration treatment strategy was obtained from a cross-sectional study which surveyed several water supply systems in small communities (LeChavellier, et al., 1991) combined with estimates for the presence of organisms from reports of outbreaks that occur in large communities (Goldstein et al., 1996; Hayes et al., 1989; Leland et al., 1993; MacKenzie et al., 1994; Solo-Gabriele & Neumeister, 1996). In contrast, the estimate for the presence of the organism in a water supply system that had no treatment strategy was obtained from one report of a Cryptosporidiosis outbreak to a very small population (Solo-Gabriele & Neumeister, 1996). It would not be a wise strategy to recommend to a large community not to treat the drinking water supply or use a filtration system.

Our sensitivy analysis showed that the filtration strategy has the highest EML in comparison to the other two intervention strategies in spite of the reduction in its cost by 50%. This evaluation is based on the cost estimates of the implementation of these strategies and the estimates of their efficacy in reducing or controlling the risk of contamination of the water supply system with *C. parvum.* This analysis explicitly demonstrates the resource implications of the allocation of decisions in the context of direct costs only, and ignoring the potentially immense indirect costs. However, society and policymakers often have difficulty in accepting this information. Furthermore, political pressures may lead to inefficient decisions and choices that are perceived to provide satisfactory outcomes. In the short run these choices might survive but in the long run society should be made aware of the opportunity cost of the money spent on the political choices.

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INDICATORS OF INCOMPLETE DISEASE SURVEILLANCE OF CLINICAL MASTITIS ON A LARGE NATIONAL DAIRY DATABASE

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Despite the increasing and already extensive use of second source databases for epidemiologic research, examples of formal data quality evaluations are few. A framework for evaluation of such types of data for research has recently been proposed by Soerensen et al. (1996). They define secondary data in research as data which have not been collected with a specific research purpose. Such data are often collected for management and administrative purposes. An example of how useful this type of data can be is the study on mortality in Danish dairy cows as related to production information, using time series analysis of data from 1960-1990 at the national level (Agger 1983, Agger and Willeberg 1991). The use of already existing data may reduce research time and costs, and big databases make it possible to study large samples of animals and herds. This improves the representativeness. One of the disadvantages is that the methods of data collection are not under the control of the researcher. Also, data are often impossible to validate. Second source data are almost never complete. However, the importance of occurrence of e.g. over- and underreporting of disease and of different types of errors depends on the purpose of the study.

The internist requires knowledge of the normal range of values for proper interpretation of a clinical chemistry or serologic test performed on an individual patient. In a similar manner, the field epidemiologist attempting to diagnose a herd disease problem requires a reference value regarding the normal or usual range of disease incidence. While such measures of normal disease incidence rarely perfectly apply to the herd of interest, they nevertheless allow a crude comparison where no other more applicable comparison is possible. While perhaps it is true that there is no such thing as a "normal" farm, it is also true that there is no such thing as a "normal" patient, and yet the use of reference values in clinical medicine has become so widely accepted that normal ranges are routinely pre-printed on virtually all laboratory report forms.

It is the responsibility of the field epidemiologist to provide accurate and interpretable measures of normal disease incidence for utilization by veterinary practitioners. Disease surveillance has been described as the "eyes and ears" of the field epidemiologist, and the way in which the epidemiologist "takes the pulse" of the population. Although impressive statistical modelling of the analytical epidemiologist may attract more attention from colleagues, the basic descriptive epidemiology of disease surveillance data has repeatedly played a major role in most disease control programs and practical policy decisions.

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To generate measures of disease surveillance, limited resources invariably dictate that mastitis field epidemiologists must choose between actively managed data collection methodologies conducted on a relatively small number of intensively monitored volunteer farms, or a more inclusive but less-controlled data collection methodology applied to a larger but usually more representative group of herds. Both approaches have their advantages and disadvantages. The intensive system usually provides accurate and well documented data through frequent and time-consuming farm visits by the investigators. Laboratory confirmation of diagnoses is often possible if, for example, milk from clinical mastitis cases is cultured. Such systems are often termed 'active disease surveillance' in that the investigators actively pursue disease reports through frequent interviews with dairy producers or provision of a service or supplies to encourage disease reporting. Such data collection protocols must usually be reserved for volunteer dairy producers who keep excellent cow records, have high management skills, are not secretive about their herd records, and are willing to collect additional data for the researcher or modify normal management procedures to accommodate the needs of the research protocol. Unfortunately, these restrictions often make intensively collected databases unrepresentative of the general population of dairy herds (sampling bias), and studies based on such databases may therefore lack external validity. Also, because of the considerable effort necessary to collect such databases, the total number of herds involved has rarely exceeded 100 herds.

In contrast, large passively collected observational databases may be biased by non-compliance with study protocol regarding disease reporting, poor standardization regarding case definitions, and high variance in diagnostic acumen. However, such databases often can include hundreds or sometimes thousands of herds, making it possible for epidemiologists to study herd-specific disease risk factors such as management procedures and environmental factors. A major limitation of such passively-collected extensive databases is that some means must be employed to identify and exclude from study those herd records which are incomplete due to disease misclassification, usually in the form of under-reporting.

Neither the intensive (active) or the extensive (passive) approach to disease surveillance can include the records of producers who refuse to participate, either by actively refusing intensive protocols, or by spelling passive disease collection systems with undependable and erratic reporting. Nevertheless, if such non-compliant producers can be identified for exclusion, extensively collected databases may be more representative of the general population of dairy farms than highly intensive surveillance databases. Also, intensively-collected databases rarely include a sufficiently large number of herds for meaningful analysis if study objectives relate to herd-specific risk factors.

The quality of herd management is likely associated with completeness of disease reporting. Dairy producers with well-managed herds are likely to be diligent regarding disease recording and have higher diagnostic acumen (ability) than dairy producers with poorly-managed farms. Rates of disease on poorly-managed farms may appear low only because of under-reporting and non-recognition of clinical mastitis. This differential misclassification bias will cause problems for researchers attempting to relate management practices to disease rates. Unless non-compliant herds are removed from the database, herds with superior management practices will likely show higher rates of mastitis than do herds with inferior management practices.

The objective of the following study is to provide information regarding the applicability of clinical mastitis incidence causal studies in farms participating in the Danish Cattle Data Base. Data screening techniques are used to identify herds suspected of non-reporting so that these farms can be excluded from causal epidemiologic studies. The study is based on the comparison of data on reported cases of clinical mastitis with data on monthly individual cow somatic cell counts estimated as a part of production monitoring at the cow level.

Comparison with other disease surveillance information is often made difficult by different def-

initions of mastitis incidence. To facilitate comparisons, we have calculated our measures of mastitis incidence in three different manners.

MATERIALS AND METHODS

The Danish Cattle Data Base was established in 1980 and now centralizes data regarding disease, production, reproduction, inheritance, and other animal events. However, the present disease monitoring system was not initiated until 1989 by the Danish Agricultural Advisory Center. Three counties known for good record keeping and good cooperation between farmers, veterinarians and farmers associations were selected for systems development. In 1991 this computerised disease recording scheme was made nationwide, and now includes 92% of all dairy herds in milk control societies (Andersen 1996, personal communication).

The 2146 herds included in the study originated from the three areas as mentioned above. A management questionnaire was completed for each herd, as described by Alban and Agger (1996 a, b) and Agger and Alban (1996). The disease study period was from July 1, 1993 to July 1, 1994.

The data have been restricted to a monitoring period from July 1, 1993 to July 1, 1994. Mastitis reports were entered into the database by both the dairy producers and by the practicing veterinarians. Of the 68,788 mastitis reports analyzed in this study, 77% were reported by the veterinarian and only 23% were reported by the dairy producer. Specific codes for various types of mastitis (acute mastitis, mastitis secondary to teat lesion, necrotic, unspecified mastitis, summer mastitis, and mastitis in a dry cow) were all combined into a single mastitis category for this analysis.

The number of days at risk of obtaining mastitis were calculated for each individual cow during the period of July 1, 1993 to June 30, 1994. Heifers began accumulating days at risk when they had their first calf and cows purchased during the monitoring period began accumulating days at risk on their date of purchase. Cows leaving the herd for any reason accumulated days at risk up until their cull date.

The disease incidence rate (true rate, incidence density rate) was calculated in three distinct ways:

M1. ((Number of mastitis cases) / (total number of cow-days at risk)) * 365 days * 100 cows. A case of mastitis was defined as a single mastitis report or series of reports separated from each other by less than 14 days. If at least 14 days elapsed after a mastitis report, any subsequent mastitis report was defined as the beginning of a new mastitis case. Cows were not considered at risk of obtaining a new case of mastitis during an ongoing case of mastitis or for 14 days after the last mastitis report of a mastitis case.

M2. ((Number of lactations affected with mastitis) / (total number of cow-days at risk)) * 365 days * 100 cows. This measure of mastitis incidence reflects the number of lactations affected by mastitis. Because we monitored the herds for 12 months, individual cows could contribute parts of 1, 2, or 3 lactations to the database and could therefore contribute 0, 1, 2, or 3 mastitic lactations to the numerator. For example, a cow with one calving in the middle of the monitoring period may have been in her 2nd and 3rd lactations during year-long study, and could therefore be counted as having at most two lactations affected by mastitis. Cows reported as having mastitis did not contribute days at risk for the M2 denominator for the remainder of their affected lactation.

M3. ((Number of cows with mastitis) / (total number of cow-days at risk)) * 365 days * 100 cows. This measure of mastitis incidence counted the number of cows which were affected at anytime during

the period of time they were being monitored. As such, cows with one or more reported mastitis treatments contributed a "1" to the numerator and cows with no mastitis contributed a "0". Individual cows with mastitis did not accumulate days at risk after their first reported mastitis treatment.

The following data screening methods were used to identify herds which showed evidence of under-reporting or non-reporting of clinical mastitis:

Method 1. All cows in the study were tested monthly for SCC. For each cow in each herd, we counted the number of milk tests with at least one SCC over 1 million cells per ml, and computed the percentage of these SCC tests which also had at least one mastitis report for this cow within 30 days. This percentage was used as an index on which to identify cows at high risk of having shown clinical mastitis for which no clinical mastitis reports had been submitted. Herds with a value of under 2 percent on the above index were suspected of under-reporting clinical mastitis and were therefore excluded from the calculations of mastitis incidence. Herds with less than 5 SCC tests over 1 million were included in the calculations of mastitis incidence since the above index was deemed unreliable due to small numbers.

Method 2. All producers were asked on the management questionnaire to estimate how many cases of mastitis occurred among their cows during the recent month. Herds with a calculated M2 incidence of at least 100% of this questionnaire estimate were included in the database.

RESULTS

Preliminary results of this study are reported here, some of which will be elaborated further on in the near future.

Table 1. Mastitis incidence rates (cases /100 cow-years) by data screening method and definition of mastitis.

Screening method and definition of mastitis	Number of cases	Cow days at risk	Incidence rate (cases/100 cow-years at risk)
Unscreened:			
Mastitis 1	44,202	38,463,249	41.95
Mastitis 2	36,021	34,582,687	38.02
Mastitis 3	33,613	33,939,766	36.15
Method 1:			
Mastitis 1	42,990	32,502,076	48.28
Mastitis 2	34,893	28,733,562	44.32
Mastitis 3	32,518	28,113,324	42.21
Method 2:			
Mastitis 1	41,382	33,119,817	45.61
Mastitis 2	33,594	29,513,028	41.54
Mastitis 3	31,311	28,921,968	39.52

Table 1. shows the mastitis incidence rates according to data screening method and definition of mastitis. The removal of herds due to screening method 1 in particular and also to method 2, as compared to unscreened herds clearly change the calculated incidence rates, due to eliminating the herds with no or very low disease recording. This change is further detailed in Figs. 1 and 2.

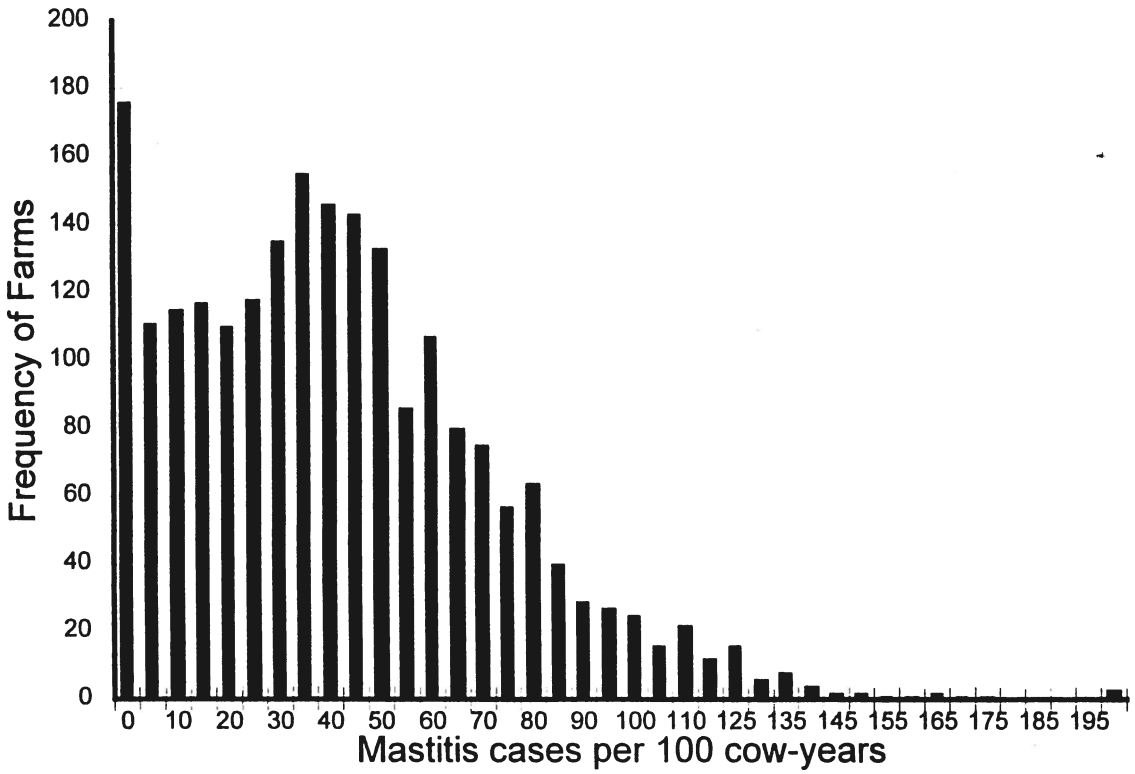

Figure 1. Frequency distribution of herd mastitis incidence rate (M1) in cases per 100 cow-years at risk. No data screening was employed (n=2146 herds).

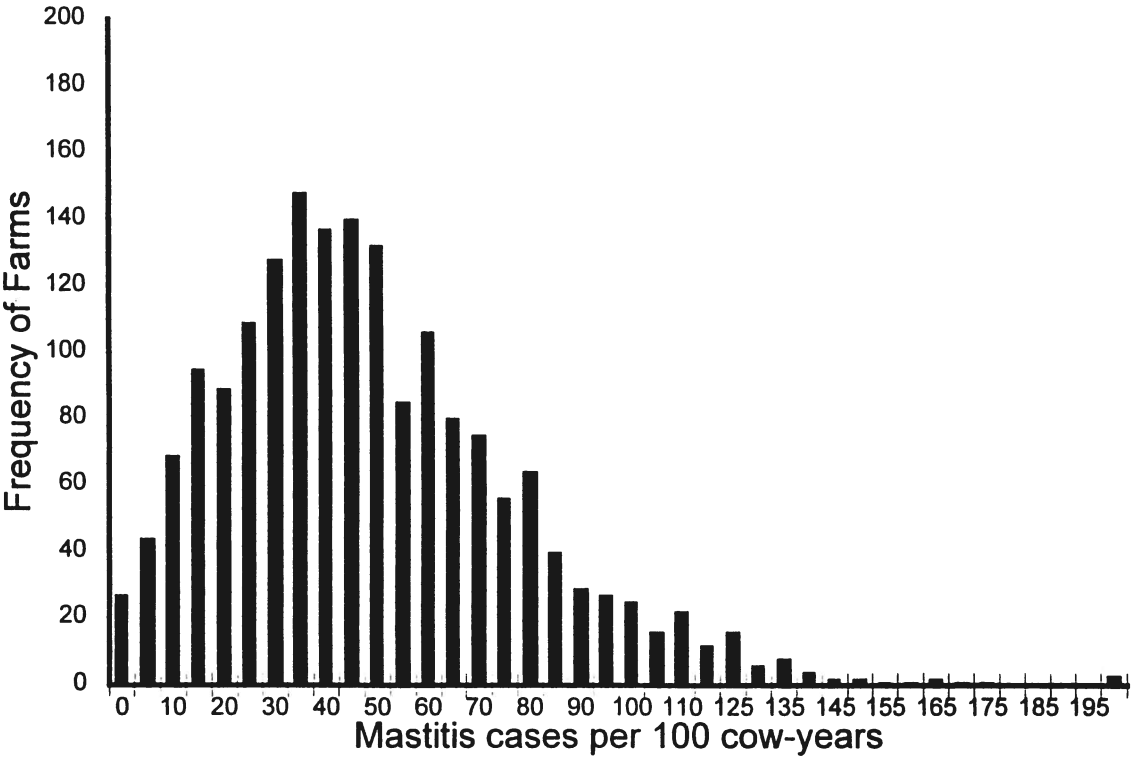

Figure 2. Frequency distribution of herd mastitis incidence rate (M1) in cases per 100 cow-years at risk. Herds judged to have inadequate mastitis reporting, according to screening method 1, have been excluded (n=1802).

The distributions of unscreened herds (Fig. 1) and after the application of screening method 1 (Fig. 2) show a dramatic change towards a more normal distribution slightly skewed to the right.

DISCUSSION

We believe that our technique of monitoring all cows present during a one-year monitoring period provides a representative sample of cow days at risk. Most cow-specific mastitis determinants (age, season of calving, days post-partum) will be appropriately represented in the database. For example, while it is true that some cows were monitored while in early lactation, and therefore presumably at higher risk of obtaining mastitis, such cows will be balanced with herdmates which contributed days at risk that were primarily late in lactation. Our study should be considered as having monitored all cows present during a representative sample of 365 days for which both the numerator (onset with mastitis) and the denominator (at risk of having onset with mastitis) were accessed and tabulated on a daily basis.

Many studies have used estimates of population size and disease duration to approximate the denominator days at risk. We believe that the availability of powerful statistical programs makes such estimates unnecessary, because the actual number of days at risk can now be computed on a daily basis for each cow being monitored.

Two screening techniques were used to identify herds suspected of under-reporting so that they could be excluded from the study. The SCC test can be considered "hard" data, in that it is not subject to subjective determination of a human observer. As such, it is a good objective measurement for use in evaluating mastitis reporting. Cows with a SCC of over 1 million cells per ml are at high risk of showing some signs of clinical mastitis. While it may be possible for milk with a SCC of this magnitude to have come from a cow showing no clinical signs, such cows certainly have a very high risk of showing clinical signs. Our index is based on the biological supposition that, given a cow with a SCC of 1 million, it is reasonable to assume that sometime in the previous 30 days or subsequent 30 days there would have been some clinical signs of mastitis which were exhibited in this particular cow. The second screening method basically assesses the repeatability of the disease reporting, by asking the farmer on the management questionnaire to state how many cows had mastitis in the previous month. As such, it is probably preferable to use method 1 which is based on an objective and independent measure of clinical mastitis.

There are many reasons for incomplete reporting of mastitis by dairy producers in Denmark and elsewhere. Some farmers are apathetic to disease control issues or lack the time to keep complete records. In Denmark, veterinarians are required for antibiotic treatment of mastitis. Because computerized veterinary records are included in this database, mastitis cases are reported even if the farmer never reports a single mastitis case.

The incidence rates reported in the current study are higher than most, but not all, reported rates from other developed countries. Miltenburg et al. (1996) reported 12.7 quarter cases per 100 cow-years at risk in the Netherlands, Bartlett et al. (1992) reported 38.4 cases per 100 cow-years at risk in Ohio, U.S.A.. Rates of 54.6 and 41.2 cases per 100 cow-years at risk have been reported from England and Wales (Miltenburg et al. 1996). Several factors could account for higher rates reported in Denmark. 1. Danes have a long tradition for reporting production and health data and enjoy considerable industry support for the Danish Cattle Data Base. 2. Veterinarians, rather than farmers, reported most of the mastitis cases in Denmark. 3. The screening procedures to remove non-reporting herds from the database increased the calculated incidence rates. 4. The direct calculation of days at risk for each cow, with the adjustment for mastitic cows not being at risk of developing a new mastitis case, acted to decrease total days at risk, thereby increasing the calculated incidence rates.

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THE TREATMENT AND PROGNOSIS OF MASTITIS ASSOCIATED WITH SHOCK (TOXIC MASTITIS): A FIELD STUDY

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Coliform organisms (gram negative, lactose fermenting bacteria of the family enterobacteriaceae) are currently one of the most important causes of clinical bovine mastitis and coliform mastitis is an important cause of death in adult dairy cows (Menzies et al, 1995). Over 1/4 of mastitis isolates have recently been reported to be coliforms (Booth 1993) and in well managed, low somatic cell count herds, coliforms are often the commonest cause of clinical mastitis (Erskine et al, 1988, Hogan et al, 1989, Schukken et al 1989, Guterbock, 1995). The clinical manifestations of the disease have been classified (Anderson 1989) as follows: TYPE I (Local); acute 1/4 infection only, TYPE II (Systemic); acute 1/4 infection with systemic signs (eg. pyrexia, loss of appetite, reduced milk yield) and TYPE III (Toxic); acute 1/4 infection with systemic signs and the onset of shock (eg. sunken eyes, severe weakness/recumbency, low body temperature). Around 10-15% of clinical coliform infections are thought to result in toxic (Type III) mastitis (Anderson 1989). Mastitis associated with shock is not always caused by coliform organisms however, and although they are thought to be the major cause of the disease (Smith & Hogan 1993), other mastitis pathogens, such as Staphlococcus aureus, can cause mastitis with toxic signs (Blood & Radostits, 1989).

Evaluating the severity of toxic mastitis in the field provides a challenge to the veterinary practitioner. Little information has been reported on the natural history or clinical findings in field cases of toxic mastitis and there are no well established methods to predict the outcome of the disease. A method of predicting survival would mean clinical decisions could be made quickly and accurately which should be of benefit to the welfare of the cow.

Toxic mastitis is particularly difficult to treat successfully and the variety of treatment protocols that exist reflects the uncertainty in this area. Commonly suggested treatments include antibacterials, anti-inflammatories, fluid therapy, and oxytocin with regular stripping of the affected quarter (Anderson, 1989, Jones, 1990, Lohius, 1990, Erskine et al, 1993) but up to 80% of cases die despite intensive therapy (Blood et al, 1989). There have been few controlled field studies to assess the efficacy of the different treatments and it is therefore difficult for the clinician to decide which are most likely to be successful.

Experimentally, severe and prolonged coliform mastitis has been shown to occur when there is a poor host cellular response to infection (Hill 1981). It has been suggested that in selecting for low somatic cell counts (e.g.by culling cows with persistently high counts) there is a possibility of selecting for a poor cellular response to infection (Shook 1993). While the concept that low somatic cell counts may act as a predisposing factor to coliform mastitis is not new (Jones 1976), little research has been performed in the field to study this possibility.

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In light of these uncertainties, we decided to undertake a field study of toxic mastitis. Our aims were i. to examine the possibility of predicting outcome, ii. to assess the efficacy of three commonly used treatments and iii. to discover if there was an association between toxic mastitis and herd bulk milk somatic cell counts (HBMSCCs).

MATERIALS AND METHODS

The study was carried out at two adjoining veterinary practices in mid Somerset. Farmers presented clinical cases for treatment over a three year period, January 1991 to January 1994. Cows accepted into the study fitted the following case definition; recumbent or severely weak with signs of shock (sunken eyes, raised pulse, raised respiratory rate, severe depression) and obvious inflammatory changes in the udder including an abnormal secretion.

A protocol was designed prior to the study and veterinarians were briefed regarding its use. At the initial presentation of a case, the cow and farms identity and the date and time were recorded. The historical details collected were; age of cow , previous calving date, length of time since onset of mastitis (as perceived by the farmer) and the length of time in recumbency (if appropriate). The clinical variables measured were; rectal temperature, respiratory rate, pulse rate, skin tent time of upper eyelid, assessment of water intake (scored on a scale 1-5 where 1=normal to 5=none), assessment of appetite (scored on a scale 1-5 where 1=normal to 5= anorexic), faecal consistency (scored on a scale 1-5 where 1=hard and firm, 3=normal, 5=watery) and depression (scored on a scale 1-5 where 1=normal to 5=moribund). The position of the affected quarter(s) was recorded. Each affected quarter was classified as hot, swollen or painful or a combination of these. The secretion from clinically affected quarters was classified as clotted or watery. Any concurrent disease diagnosed during the examination was recorded.

A heparinised blood sample was taken from each cow before treatment. Milk samples were taken aseptically from all quarters following a standard technique (Cripps, 1990), frozen and then posted to a laboratory.

Each cow was allocated to treatment group A, B or C. This was done by randomly distributing the questionnaires to each veterinarian with the treatment group marked on each. The treatments used in the three groups were; Group A, fluid therapy and flunixin, Group B, fluid therapy only and Group C, flunixin only. All groups received the same combination of parenteral and intramammary tetracyclines, intravenous calcium boroglucanate and intramuscular oxytocin.

Each cow was attended twice by a veterinarian within an 18-24 hour period. The fluids administered to groups A and B were made up by adding a powder containing 42.5g Sodium Chloride, 1g Calcium Chloride, 1.5g Potassium Chloride and 1g Sodium Bicarbonate (isotonic powder, Micro Biologicals Ltd), to 5 litres of clean, warm tap water. The fluids were given intravenously, 30 litres at the first visit and, unless a complete recovery had been made, 15 litres at the second visit. Fluid was administered under pressure through a 12 gauge catheter using a 7½ litre garden weedkiller spray pump (Killerspray (professional 12) Hozelock, Birmingham). 30 litres were administered in 45 - 60 minutes. 1000mg (20ml)Flunixin meglumine (Finadyne, Scherring-Plough) was administered intravenously to cows in groups A and C at each visit. All groups were injected with 2000mg (20ml) oxytetracycline (Engemycin, Mycofarm). This was administered intravenously by the veterinarian and continued intramuscularly by the farmer every 24 hours for 5 days. 80ius oxytocin (Oxytocin-S, Intervet) were given intramuscularly

to all cows at the initial visit to facilitate milk withdrawal from the affected quarter(s) which were then stripped out. Each farmer was advised to strip the affected quarter(s) every two hours and chlortetracycline (420mg) and hydrocortisone (2mg) intramammary tubes (Aureomycin, Cyanamid) were administered by the farmer after the last stripping of the day, for 5 consecutive days. 400ml of 40% calcium borogluconate was given by slow intravenous injection at the first visit to all groups. Advice was also given to each farmer that a clean, warm environment should be provided with food and water available at all times.

The outcome of each case was monitored. A cow was recorded as having survived if it did not die or was not humanely destroyed as a direct result of the disease. The survivors were separated into those which returned to the milking herd and those which did not but were sold for human consumption at a later date. Recovery of affected quarter(s) was not monitored.

A farm with a cow with toxic mastitis in the study was defined as a case farm. A control farm was selected for each case farm at the end of the 3 year period. This was done by randomly selecting from all farms in the practice which had not had a cow with mastitis included in the study. The arithmetic mean HBMSCC was calculated retrospectively for both case and control farms for the month in which the case occurred either using information from Genus Animal Health or from the farm monthly milk statement . The HBMSCCs could then be compared for case and control farms in the month in which the case occurred.

Blood samples were analysed in the practice laboratory using a centrifuge and Unifast 2 Sclavo analyser with standard techniques (Kerr 1989, Unifast 2 Sclavo analyser operator manual 1989). The packed cell volume, blood urea, and total plasma protein concentrations were estimated before treatment, in order to assess the state of hydration of each cow. At the end of the study a heparinised blood sample was taken from a group of 48 healthy adult cattle. From these normal values for PCV were measured to provide a comparison with the PCV results from toxic cows in the study. Bacteriological examination of the milk samples was performed at the Institute for Animal Health, Compton .

Data were collated and analysed on microcomputer. Epi-Info 5 (CDC, Atlanta, GA, USA), Minitab (Minitab inc., State College, PA, USA) and Genstat (NAG Ltd. Oxford) software were used for analysis . The treatment groups were compared using one way analysis of variance, Kruskall-Wallis, and chi-squared tests as appropriate. The null hypothesis was that there was no difference between groups. The case and control farm HBMSCCs were compared using a Wilcoxon 1 sample non-parametric test. Unconditional logistic regression analysis was carried out on cow survival, the independent variables were; age, time since previous calving, duration of mastitis and recumbency at first veterinary visit, temperature, pulse rate, respiratory rate, eyelid skin tent time, PCV, blood urea and total plasma protein. Following this statistical investigation, the ability of the independent variables to predict the outcome was examined both for single predictors and for combinations. This was done by estimating from the model the probability of each individual case surviving and comparing this with the actual outcome. From this the probability of survival (sensitivity) or death (specificity) was computed. The critical probability was taken to be 0.05.

RESULTS

Survival

54 cows were accepted into the study, 18 cows in each treatment group. Of these 29 (53.7%, 95% confidence interval = 39-67%) cows survived and 25 (46.3%) died. 24 survivors returned to the milking herd and 5 did not but were later culled for human consumption. The number of cows which survived in groups A and B was 10 (55.6%) and in group C, 9 (50%); this was not significantly different ($p>0.10$). The number of cows which returned to milk in groups A, B and C was 7, 8, and 9 respectively which also was not significantly different ($p>0.10$). Since there were no differences between treatment groups, the cases were considered as one group for the development of the prognosis model.

Temporal Presentation

23 cases (42.6%) were presented between January and June 1991 (Figure 1).

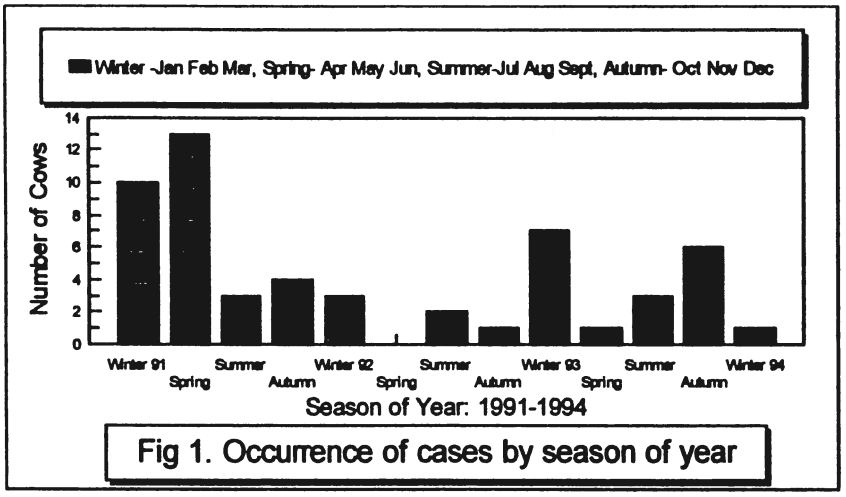

Fig 1. Occurrence of cases by season of year

The commonest time of day for presentation was between 8.00am and 10.00 am and no cases were presented between 9.00pm and 8.00 am (Figure 2).

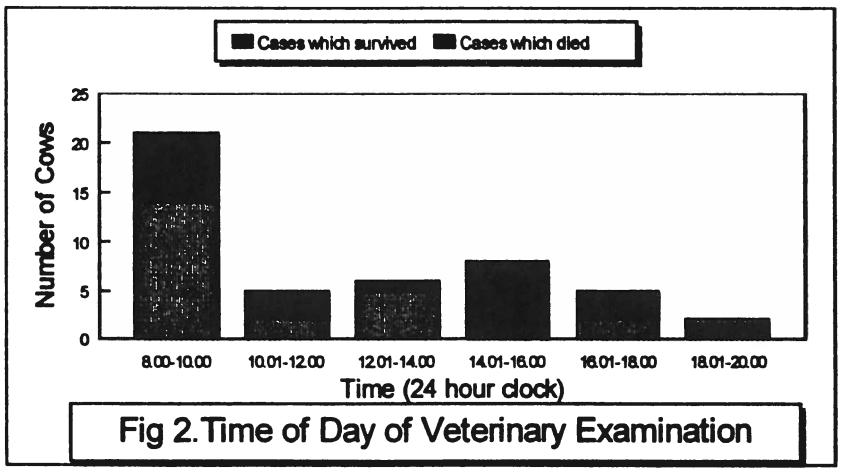

Fig 2.Time of Day of Veterinary Examination

20/37 (54.1%) of cows had calved within 7 days of initial examination. The occurrence of cases diminished with increased time from calving (Figure 3).

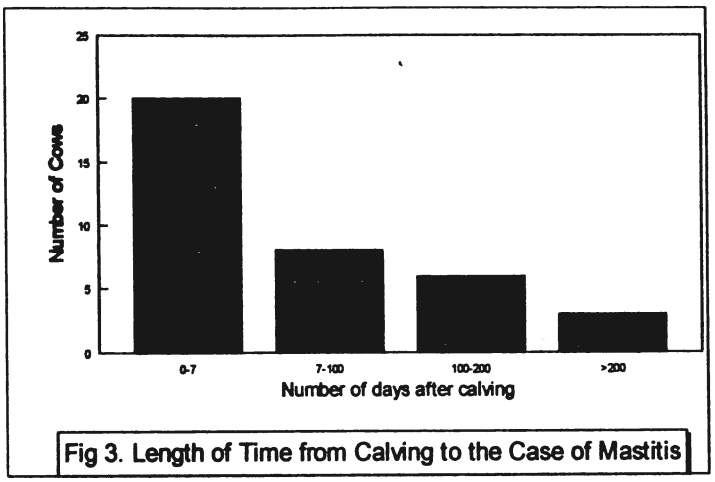

Fig 3. Length of Time from Calving to the Case of Mastitis

The length of time between the onset of mastitis and veterinary visit was very variable . The shortest time recorded was 3 hours but in 2 cases the onset of mastitis was 48 hours prior to the visit. The mean time was 16.0 hours (Figure 4).

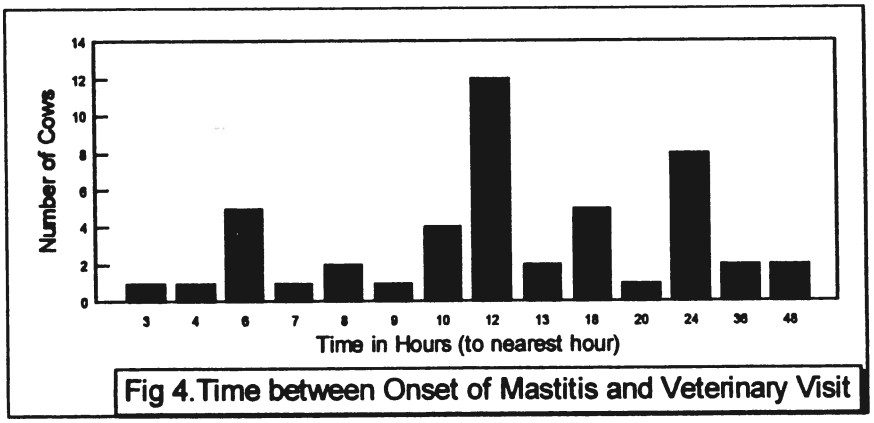

Fig 4.Time between Onset of Mastitis and Veterinary Visit

Clinical Presentation

The mean age of cows in the study was 7.8 years with a range of 2-14 years (figure 5).

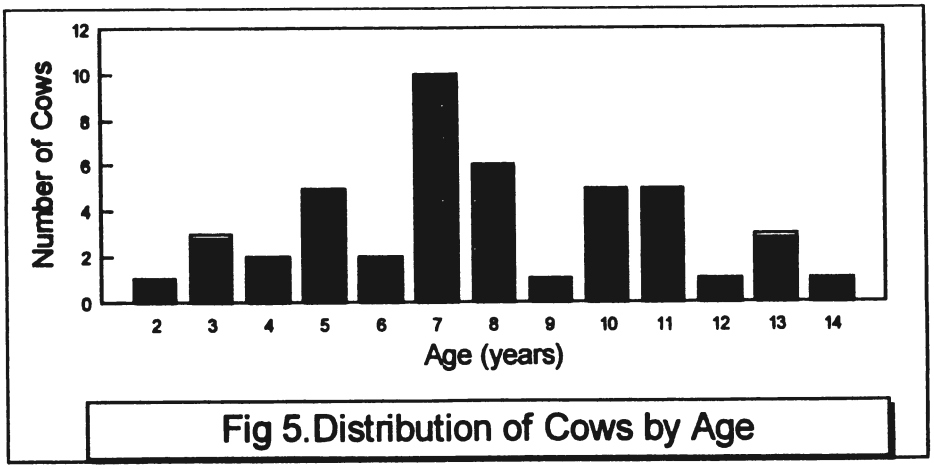

Fig 5.Distribution of Cows by Age

40/50 (80%) cows were recumbent at the time of veterinary visit and 10 (20%) were standing but severely weak. The mean time in recumbency for recumbent cows prior to the

veterinary visit was 6.4 hours. The mean rectal temperature of cows that died was 100.3^{0}F and those that survived was 101.9^{0}F, (p=0.003) . The mean eyelid skin tent time for cows that died was 6.7 seconds and for those that survived was 3.6 seconds, (p=0.01). Cows which died were significantly more depressed than those that survived, (p=0.018). There were no other significant differences in clinical presentation between cows which survived and died (Table 1).

Table 1. A summary of the clinical and historical details recorded before treatment

CLINICAL INFORMAT'N		ALL GROUPS		BY OUTCOME				p value
		n		Survived	n	Died	n	
AGE (Years)	Mean	45	7 8	7 4	27	8 6	18	ns
	S D		3	3 2		2 7		
Time since calved (days)	Median	37	6	37 5	20	3	17	ns
	Inter 1/4s		3-10	3 5-97		2-110		
Time since onset of mastitis (hr)	Mean	47	16	16 9	25	15	22	ns
	S D		10 2	10		10 6		
Time in recumbency (hrs)	Mean	50	6 4	5	26	7 9	24	ns
	S D		6 6	6		7 4		
Rectal Temp (^{0}F)	Mean	50	101 2	101 9	27	100 3	23	p=0.003
	S D		1 9	1 6		2		
Pulse Rate (beats/min)	Mean	46	96 8	94 8	25	99 1	21	ns
	S D		16 9	16 9		17 1		
Resp Rate (per min)	Mean	47	39 5	38 1	24	40 9	23	ns
	S D		21 1	18 7		23 7		
Eyelid Skintent Time (secs)	Mean	48	5 1	3 6	25	6 7	23	p=0.01
	S D		3 9	1 4		5 1		
Score for Appetite (scale1-5)	Median	52	5	5	27	5	25	ns
	Inter 1/4s		4-5	4-5		4-5		
Score for Water Intake (scale1-5)	Median	50	5	5	25	5	25	ns
	Inter 1/4s		4-5	4-5		4-5		
Score for faecal consistency	Median	45	4	4	26	4	19	ns
	Inter 1/4s		3-5	3-4		3-5		
Score for Depression (scale1-5)	Median	54	4	3	29	4	25	p=0.018
	Inter 1/4s		3-4	3-4		3-5		

The clinical measurements made at the initial examination were not significantly different between treatment groups (p>0.10), except the score for depression, for which

group C was significantly lower (less depressed) than groups A and B (p=0.01) .

Udder Changes

18 (33%) cows had more than one quarter affected and hind quarters were more frequently affected than fore quarters. The number of quarters judged to be clinically affected per case did not differ significantly in cows which survived or died. Description of the affected quarters indicated that 49.4%, 51.9% and 49.4% of quarters were classified as hot, swollen and painful respectively. The secretion was classified as watery in 88.6% of cases .

Concurrent Disease

Six cows were considered to be suffering from hypocalcaemia and of these, three cows survived and three died. One cow which had been treated previously for hypocalcaemia by the herdsman and two cows found by the veterinary surgeon to be suffering from metritis subsequently died. One cow was recorded with 'puffy eyes' and died.

Laboratory Results

The PCV results from the clinically healthy cohort of cows showed a significant difference from the diseased cows in the study. The range for healthy cows was 24-38% with a mean of 31.4% and the range for diseased cows was 29-60% with a mean of 42.6% (p<0.01). Packed cell volume was significantly greater in cows which died than those which survived (p<0.001) (Table 2). There were no significant differences between treatment groups in any of the laboratory measurements (p>0.10).

Table 2. Summary of the laboratory results for cows with toxic mastitis

LABORATORY TEST		ALL GROUPS		BY OUTCOME				Significance Survived vs died (p value or ns=not sig)
		n		Survived	n	Died	n	
PCV (%) Normal=24-46	Mean	53	42.6	39.5	29	46.4	24	p<0.001
	S.D.		6.5	5.3		6		
Blood Urea (mmol/litre) Normal= 2.0-6.6	Mean	49	5.8	5.4	27	6.3	22	ns
	S.D		3.1	2.7		3.6		
Total Plasma Protein (g/litre) Normal =53-75	Mean	50	76.3	73.5	27	79.5	23	ns
	S.D		18.9	18.4		19.3		

Bacteriology Results

Bacteriological examination of the milk samples resulted in organisms being isolated from clinically affected and apparently unaffected quarters. Coliform organisms (gram negative, lactose fermenting bacilli of the family Enterobacteriaceae) were not identified further. There were 11 cows in which a potential pathogen was isolated in pure growth from clinically affected quarters only, 10 produced a coliform organism and 1 Streptococcus

uberis . In 12 cows a pure growth of a coliform organism was found in clinically affected as well as in non-clinical quarter(s). In 7 cows a pure growth of a potential pathogen was found in quarters not observed to be clinically affected at the time of sampling with no growth found in the clinical quarters. Of these, 6 cases produced a coliform organism and 1 Streptococcus uberis. Therefore, in a total of 30 cows a pure culture of a potential pathogen was found in one or more quarters and in 28 (93%) of these a coliform organism was cultured. In 11 cases samples were contaminated and 7 cases gave no bacterial growth . In 6 cases results were not obtained because samples were not taken or they were damaged.

HBMSCC Results (Table 3)

In 8 instances it was not possible to obtain HBMSCCs for a case farm which left 46 farms for which control farms were matched. The mean and median values and interquartile ranges are shown in table 3. Case farms had a significantly lower HBMSCC in the month of the case than control farms ($P<0.002$).

Table 3. Summary of bulk milk somatic cell count results.

	MEAN (1000s/ml)	MEDIAN (1000s/ml)	25% ILE (1000s/ml)	75%ILE (1000s/ml)
Case farms	246.6	231	154	344
controls farms	406.6	347	234	563

Further Statistical Analysis

Rectal temperature (TEMP), eyelid skintent time (STT) and packed cell volume (PCV) were significant predictors of survival. The accuracy of predicting survival (sensitivity) and of predicting death (specificity) with these variables is shown below.

Equation 1.Relationship of probability of death (D) with TEMP, STT and PCV.

$$\log^e (D/1-D) = 67.5 - 0.775 \times TEMP + 0.2172 \times PCV + 0.298 \times STT$$

46 cases eligible, correct prediction of survival (sensitivity)=84%, correct prediction of dying (specificity)=71%

Equation 2.Relationship of probability of death (D) with TEMP and STT.

$$\log^e (D/1-D) = 72.9 - 0.739 \times TEMP + 0.342 \times STT$$

47 cases eligible, correct prediction of survival (sensitivity)=84%, correct prediction of dying (specificity)=72%

The predicted probability of death calculated from the model using TEMP and STT as predictors (equation 2) is displayed in Table 4.

Table 4. The predicted likelihood of death calculated from the model using TEMP and STT as predictors (equation 2)

STT (Sec)	0	1	2	3	4	5	6	7	8	9	10
TEMP (°F)											
96	0.88	0.91	0.93	0.95	0.97	0.96	0.98	0.99	0.99	0.99	1
96.5	0.83	0.87	0.91	0.93	0.95	0.96	0.97	0.96	0.99	0.99	0.99
97	0.77	0.83	0.87	0.9	0.93	0.95	0.96	0.97	0.98	0.99	0.99
97.5	0.7	0.77	0.82	0.87	0.9	0.93	0.95	0.96	0.97	0.98	0.99
98	0.62	0.69	0.76	0.82	0.86	0.9	0.93	0.95	0.96	0.97	0.98
98.5	0.53	0.61	0.69	0.76	0.81	0.86	0.9	0.92	0.95	0.96	0.97
99	0.44	0.52	0.6	0.68	0.75	0.81	0.86	0.89	0.92	0.94	0.96
99.5	0.35	0.43	0.51	0.6	0.68	0.75	0.81	0.85	0.89	0.92	0.94
100	0.27	0.34	0.42	0.51	0.59	0.67	0.74	0.8	0.85	0.89	0.92
100.5	0.2	0.26	0.34	0.41	0.5	0.58	0.66	0.74	0.8	0.85	0.89
101	0.15	0.2	0.26	0.33	0.41	0.49	0.58	0.66	0.73	0.79	0.84
101.5	0.11	0.15	0.19	0.25	0.32	0.4	0.49	0.57	0.65	0.73	0.79
102	0.08	0.11	0.14	0.19	0.25	0.32	0.4	0.48	0.56	0.65	0.72
102.5	0.05	0.08	0.1	0.14	0.19	0.24	0.31	0.39	0.47	0.56	0.64
103	0.04	0.05	0.07	0.1	0.14	0.18	0.24	0.31	0.38	0.47	0.55
103.5	0.03	0.04	0.05	0.07	0.1	0.13	0.18	0.23	0.3	0.38	0.46
104	0.02	0.03	0.04	0.05	0.07	0.1	0.13	0.17	0.23	0.29	0.37
104.5	0.01	0.02	0.03	0.04	0.05	0.07	0.09	0.13	0.17	0.22	0.29
105	0.01	0.01	0.02	0.02	0.03	0.05	0.07	0.09	0.12	0.17	0.22
105.5	0.01	0.01	0.01	0.02	0.02	0.03	0.05	0.06	0.09	0.12	0.16
106	0	0.01	0.01	0.01	0.02	0.02	0.03	0.05	0.06	0.09	0.12

DISCUSSION

There were no significant differences found in survival rates between the three treatment groups. This indicates that either the treatments fluid therapy, flunixin meglumine or both given together, had equal effects, or that the difference between them was too small to be detected by this study. No synergistic effects of fluids and flunixin meglumine were shown. One problem of studying the treatment of a relatively low incidence disease is that it is difficult to collect sufficient numbers of cases to detect small differences between treatment groups. It is possible that there was an insufficient number of cases in this study to detect a small difference in survival rate between treatment groups. Fluid therapy and flunixin meglumine have been widely recommended for the treatment of toxic mastitis and are commonly used in the field in the same way as in this study. Whilst they may be as effective as any current alternative therapy, this study has demonstrated that there is great scope and requirement for new, more successful treatments. Although hypertonic solutions may have a role to play, it seems uncertain whether they will significantly improve survival rates above those achieved when larger quantities of isotonic fluids are given, since it is considered that no clear benefits of hypertonic solutions have been established over equimolar amounts of sodium salts administered as isotonic solutions (Erskine & others 1993). This is one area in which there is a need for more research. With an overall survival rate of a little over 50% in this study, the question has to be raised, "is treatment improving survival rate at all?" This could be determined by a clinical trial using a control group which received either no treatment or which was not treated with flunixin or fluid therapy. In practice, however, we consider that this would be an infringement of cow

welfare. Survival rates of untreated cows in field conditions are therefore not known and any benefit of fluid or flunixin meglumine therapy has not been demonstrated.

It should be useful to the practitioner to have guidance with prognosis, particularly using the clinical measurements rectal temperature and eyelid skin tent time. The logistic regression model predicted survival with an accuracy of 84% and death with an accuracy of 72%. This is substantially better than 50% which would be obtained by chance, but it is not 100% accurate. It should be noted that the model has not been validated using an external data set. Problems involving validation of this type of data have been discussed (Chamberlain 1991). However, use of the table of survival rates at different rectal temperatures and eyelid skin tent times may give guidance regarding prognosis which can aid the veterinary surgeon in how he/she proceeds with the patient. The inclusion of PCV as well as rectal temperature and eyelid skin tent time did not further improve the accuracy of the model, although PCV itself differed significantly between cows which survived and died. It is also interesting that most of the results of PCV for cows with toxic mastitis fell within the 'normal' range generally quoted for PCV in cattle; 24-46%, (Blood and Radostits 1989) and it was because of this that the PCV of a healthy cohort of cows was measured. The significant difference between the diseased and healthy groups of cows clearly shows this 'normal' range can be difficult to interpret.

Farms which had a case of severe toxic mastitis had significantly lower herd bulk milk somatic cell counts than matched control farms in the month in which the case occurred. There are several reasons why this association may have occurred. Firstly, there is experimental evidence to suggest that individual cow somatic cell counts may influence the likelihood of coliform mastitis. Thirty years ago it was demonstrated experimentally that a high somatic cell count could protect the bovine udder from coliform infection (Schalm 1964). A pre-existing artificially induced somatic cell count of 200,000-350,000 per ml was seen to give partial protection from coliform infection while counts of 500,000 or more gave complete protection. As the somatic cell counts of cows within a herd are reduced (and hence the HBMSCC) it may be that more of them reach a level of susceptibility and the probability of coliform infection increases. A proportion of these would succumb to endotoxic shock. Secondly, it may be that the association between HBMSCC and toxic mastitis is not causal but due to a common factor linking them. For example high yielding cows may be more prone to toxic mastitis and also tend to be in well managed herds which have lower HBMSCCs . Therefore a causal relationship cannot be proven from this study although an association has been shown . Thirdly, inadvertent selection bias during the study could have lead to an association being found. Selection bias would occur if case and control farms were equally likely to have a case of toxic mastitis but case farms were more likely to call in a veterinarian. It is also possible that control farms were equally likely to have a case but that these were more likely to die before veterinary attention could be summoned. With a personal knowledge of the farms and the fact that control farms call for veterinary attention for other emergency conditions, these are considered unlikely.

Bacteriological examination of milk samples in this study was not rewarding .There was a high proportion of contaminated samples (11/54 cases) which may reflect the difficulty of maintaining sterility when sampling recumbent cows. There were also many cases (19) in which a pure growth of potential pathogens were found in apparently normal quarters . These may represent early or subclinical infection or possibly contamination. Their importance is uncertain. The cases in which no bacterial growth was found in any quarter may have been due to the pathogen having been removed by the host before sampling or possibly due to death of bacteria in the samples before culture. Coliforms, however, were the most common isolate, which tends to support the fact that they are considered the most common cause of toxic mastitis (Smith & Hogan 1993).

It is important in a study of this type to assess the validity of the data. Eight veterinary surgeons were involved with data collection which could have introduced variation, although the use of a standard questionnaire with mostly objective measurements will have minimised this. Cases were not accepted into the study unless the protocol was followed exactly and they fitted the case definition. There were no significant differences found between treatment groups in the objective clinical and laboratory measurements made and this suggests that severity of cases was spread evenly between the groups, and that the randomisation was successful. The subjective score for depression given by the veterinary surgeon at first examination however, was significantly lower for group C than for the other groups. This is in contrast to all other measurements and it is difficult to assess whether it is a true effect, a chance effect or whether the decision on a score was influenced by the treatment which the veterinary surgeon knew he/she was about to administer. Such subjective scores are of little value in the field since different veterinarians are likely to interpret each patient in a different way. Problems were found with information being either omitted from the questionnaire or occasionally being illegible. This was due to veterinary surgeon error and a few questions relating to history and clinical examination were sometimes overlooked. Missing data reduces numbers and therefore the power of statistical analysis. There was an unavoidable variation in the nursing ability of herdsmen. This may be a factor affecting survival but it is hoped that good and poor nursing was distributed evenly throughout treatment groups. There was also unavoidable variation in the treatments administered by the farmer prior to the veterinarians first examination. Since the type of drug and protocol for such treatments vary widely, such differences are difficult to allow for statistically. It was considered that each cow in the study would be judged by the findings at the first veterinary visit and that treatments prior to this would be discounted. It is unlikely that such treatments caused variation between treatment groups since cows were randomly distributed into the treatment groups. The dose rates of all treatments were standardised throughout the study because it was felt that estimation of cow weight may have introduced errors. Since all cows except one (a Jersey) were adult Holstein/Friesians it is unlikely that a variation in body weight would have caused any difference in survival between treatment groups. Antibiotics, calcium and oxytocin were used on all cows as they are commonly recommended to treat severe mastitis (Anderson 1989, Blood & Radostits 1989, Erskine et al 1993), and are regularly used in the field. Since they were used consistently, they would not have caused a variation in outcome between treatment groups unless unusual drug interactions had occurred, and this is considered unlikely.

This investigation has found that the treatments fluid therapy, flunixin meglumine or a combination of both, in addition to antibacterials, oxytocin and calcium boroglucanate, resulted in a similar chance of survival. With the survival rate being a little over 50%, the identification of more successful therapeutic regimes or preferably, reliable preventive measures is essential. It was possible to predict survival for cows with toxic mastitis with an accuracy of 84% using the clinical measurements rectal temperature and eyelid skintent time. Herds with a cow with toxic mastitis were more likely to have lower HBMSCCs in the month of the case than control herds. It is possible that low HBMSCCs lead to an increased susceptibility to this type of mastitis but cause and effect cannot be proven from this investigation.

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EFFECTS OF SUBCLINICAL MASTITIS ON MILK YIELD OF DAIRY COWS

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Mastitis is considered to be the most costly health disorder in dairy herds. The assessment of the economic worth of control plans for mastitis has to be supported by reliable evaluations of the economic losses caused by the disease. Decrease in milk production is one of the components of economic losses due to subclinical mastitis.

Literature published since the 1960's about the effect of subclinical mastitis on milk yield is quite abundant (Bartlett et al., 1990; Batra, 1986; Cameron & Anderson, 1993; Dohoo & Martin, 1984; Fabre et al., 1990; Forster et al., 1966; Jones et al., 1984; Miller et al., 1993; Salsberg et al., 1984; Ward & Schultz, 1972). Estimates of effect show large variations between studies. These variations may be due to differences not only in populations studied, but also in mastitis indicators (California Mastitis Test; Somatic Cell Counts) considered and in methods used for data analysis. No recent estimation of the effect of subclinical mastitis on milk yield is available in French conditions.

The aim of this study was to combine a critical analysis of estimates published with the quantification of the effect of the individual somatic cell counts on milk yield at a test day in France.

MATERIALS AND METHODS

Data

105 dairy farms were randomly chosen from those enrolled in the Milk Recording Scheme (Loire Atlantique area, France) between 1994 and 1995. At each test day, individual Somatic Cell Counts (SCC) (x1,000 cells/ml) and milk yield were recorded for each lactating cow. A total of 62,494 records were considered during the study period.

In these herds, clinical cases of mastitis occurring between two consecutive test days were recorded and assigned to the latter one. To avoid confusion between effect of SCC and

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effect of clinical mastitis on milk yield, records with a clinical case of mastitis and/or with SCC over 1,200,000 cells/ml were excluded from the analysis. In addition, records after 305 DIM were not considered. The resulting dataset consisted of 53,900 records from 6535 Holstein dairy cows. Records from primiparous cows accounted for 33% of the data. A summary of descriptive characteristics of cows at test day is provided in Table 1.

Table 1. Summary of cow characteristics at test day
(53,900 cow-records)

	All cow-records (n = 53,900)		Primiparous cow-records (n = 17,827)		Multiparous cow-records (n = 36,073)	
Characteristics[1]	Mean	SD	Mean	SD	Mean	SD
SCC	152	194	123	157	166	209
Milk yield	24.5	7.3	21.7	5.2	25.9	7.8
DIM	152	83	156	83	149	83

[1] SCC = Somatic Cell Count (in 1,000 cells/ml), Milk yield = test day milk per cow (in kg),
DIM = days in milk at test.

Definition of variables

The statistical unit was the test day. The milk yield at each test day was used as the dependent variable. SCC, stage of lactation, test month and parity were assumed to influence the milk yield at each test day (Bartlett et al., 1990). SCC were converted into natural logarithm (LNSCC) (Ali & Shook, 1980). DIM was split into 4 categories (<50 d post-partum (pp), [50-100], [100-150], ≥150 d pp). Test months were explicitly considered (from 1 to 12). Primiparous and multiparous cows were considered in separate models.

Strategy of analysis and model building

The data set was analyzed using the general linear models procedure (GLM procedure, SAS Inc., 1989). The model used to investigate the relationships between SCC and milk yield at test day was:

$$Y_{ijk} = m + LNSCC + H_i + S_j + M_k + e_{ijk}$$

where:

Y_{ijk} = test day milk yield,
m = overall mean,
LNSCC = natural logarithm of SCC (x1,000 cells/ml),
H_i = effect of herd i (random effect, 105 classes),

S_j = effect of stage of lactation j (4 classes),
M_k = effect of month of test day k (12 classes),
e_{ijk} = residual.

As a first step, the model described above was run separately for primiparous and multiparous cows (models A and B). Resulting estimates were compared to those found in the literature.

As a second step, an interaction term between LNSCC and DIM was added to the models A and B (models C and D respectively). This procedure was used because models A and B only gave average estimates of the relationship between SCC and milk yield whatever the stage of lactation considered. Due to the known relationship between SCC and stage of lactation, an assumption was made that the effect of SCC on milk yield could vary over the lactation.

In addition at each step, the continuous variable LNSCC was replaced in all models by a categorical variable (CSCC) with 24 classes of SCC with a range of 50,000 cells/ml, resulting in models A', B', C', D'. This procedure aimed to assess the relevance of the logarithmic transformation of SCC in models A, B, C, D.

The reduction in milk yield associated with SCC (described by LNSCC or CSCC) was expressed in % of average milk yield (AMY) produced by control cow-records with SCC lower than 100,000 cells/ml. When SCC was included as LNSCC (in models A, B, C, D), the reduction in milk yield (in %) was:

$$RMY = 100 \times \frac{-\beta \times (LNSCC - LNSCC_{50})}{AMY}$$

where:

RMY was the reduction in milk yield in %,
β was the estimate of effect of LNSCC,
LNSCC was the natural logarithm of a given SCC,
LNSCC50 was the natural logarithm for SCC equal to 50,000 cells/ml,
AMY was the average milk yield produced by control cow-records (primiparous or multiparous) with SCC lower than 100,000 cells/ml.

When SCC was included as CSCC (in models A', B', C', D'), the reduction in milk yield (in %) was:

$$RMY = 100 \times \frac{-(\beta_{CSCC} - \beta_{CSCC0\text{-}100})}{AMY}$$

where:

RMY was the reduction in milk yield in %,

β_{CSCC} was the estimate attached to a given category of CSCC,
$\beta_{CSCC0\text{-}100}$ was the estimate attached to CSCC for SCC from 0 to 100,000 cells/ml,
AMY was the average milk yield produced by control cow-records (primiparous or multiparous) with SCC lower than 100,000 cells/ml.

RESULTS

Models without interaction (models A, B, A', B')

French data: The estimate coefficients associated with LNSCC were - 0.429 (SE = 0.033) and - 1.079 (SE = 0.026) for primiparous (model A) and multiparous (model B) cows respectively. All variables included in models A and B were significantly ($P < 0.0001$) associated to milk yield at test day. The respective R^2 associated with models A and B were 0.41 and 0.57 ($P < 0.0001$). When SCC is increased two-fold (e.g., from 100 to 200,000; from 400 to 800,000 cells/ml), the reduction in milk yield is equal to 1.34 and 2.68% for primiparous and multiparous cows respectively. Figures 1 and 2 display the reduction in milk yield (in %) due to SCC (described as LNSCC and CSCC) for primiparous and multiparous cows.

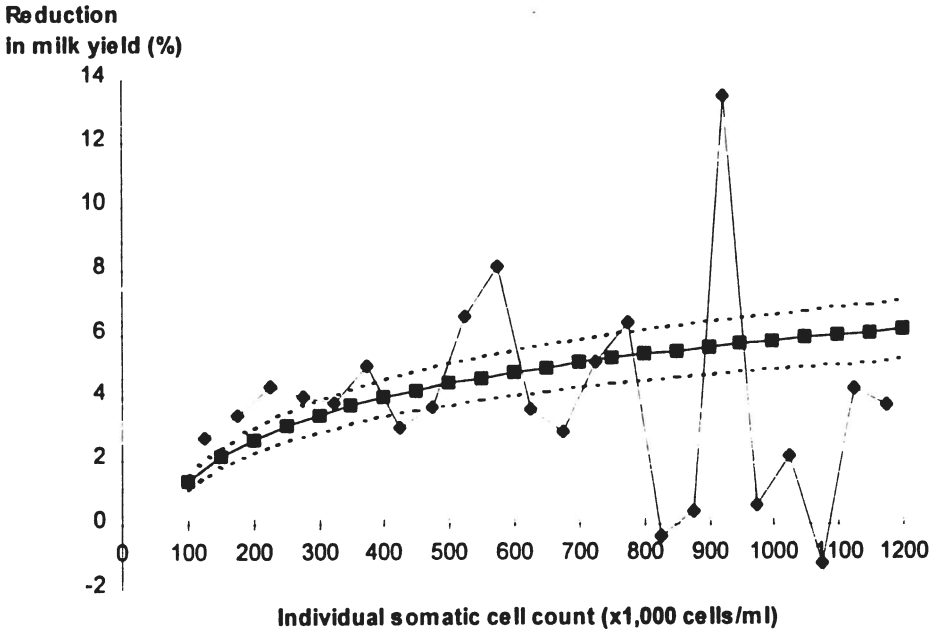

Fig. 1 Reduction in milk yield for primiparous cows expressed in % of average milk yield produced by control cow-records
square: SCC continuous (model A); lozenge: SCC categorical (model A')
95% CI of the reduction (model A) is displayed in dotted line

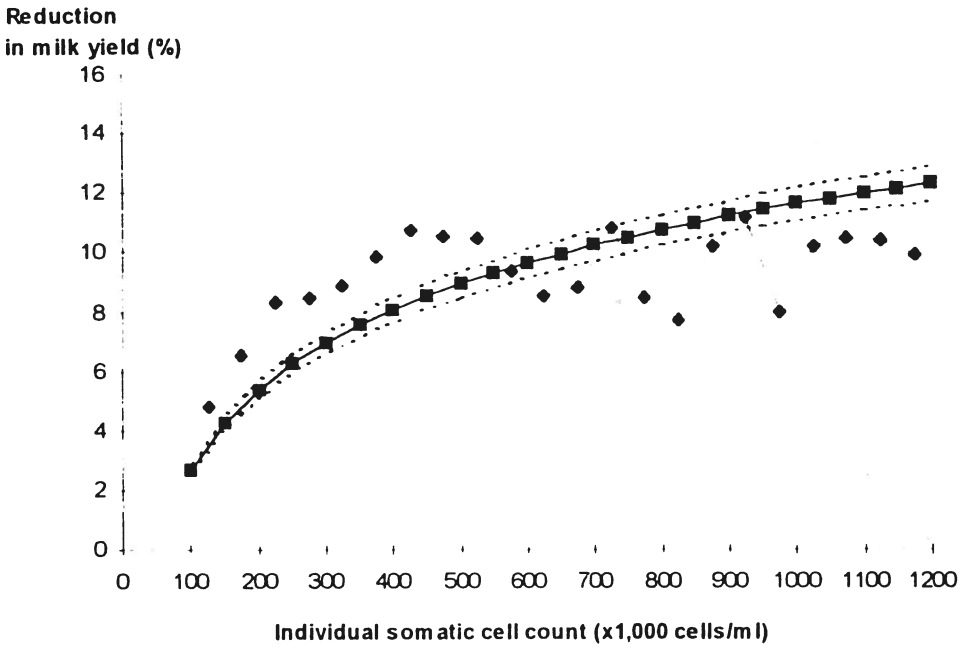

Fig. 2 Reduction in milk yield for multiparous cows expressed in % of average milk yield produced by control cow-records

square: SCC continuous (model B); lozenge: SCC categorical (model B')

95% CI of the reduction (model B) is displayed in dotted line

Comparison with the literature: Figures 3 and 4 display the reduction in milk yield (in %) due to SCC (described as LNSCC) from French data and published studies for primiparous and multiparous cows respectively.

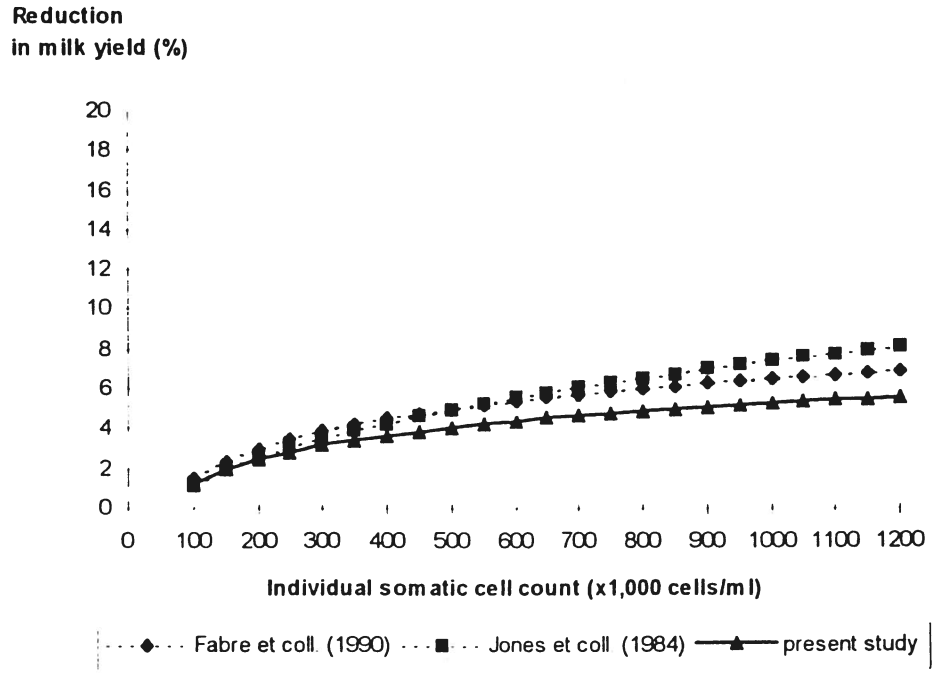

Fig. 3 Reduction in milk yield for primiparous cows expressed in % of milk yield
French data (continuous line) - Published studies (dotted lines)

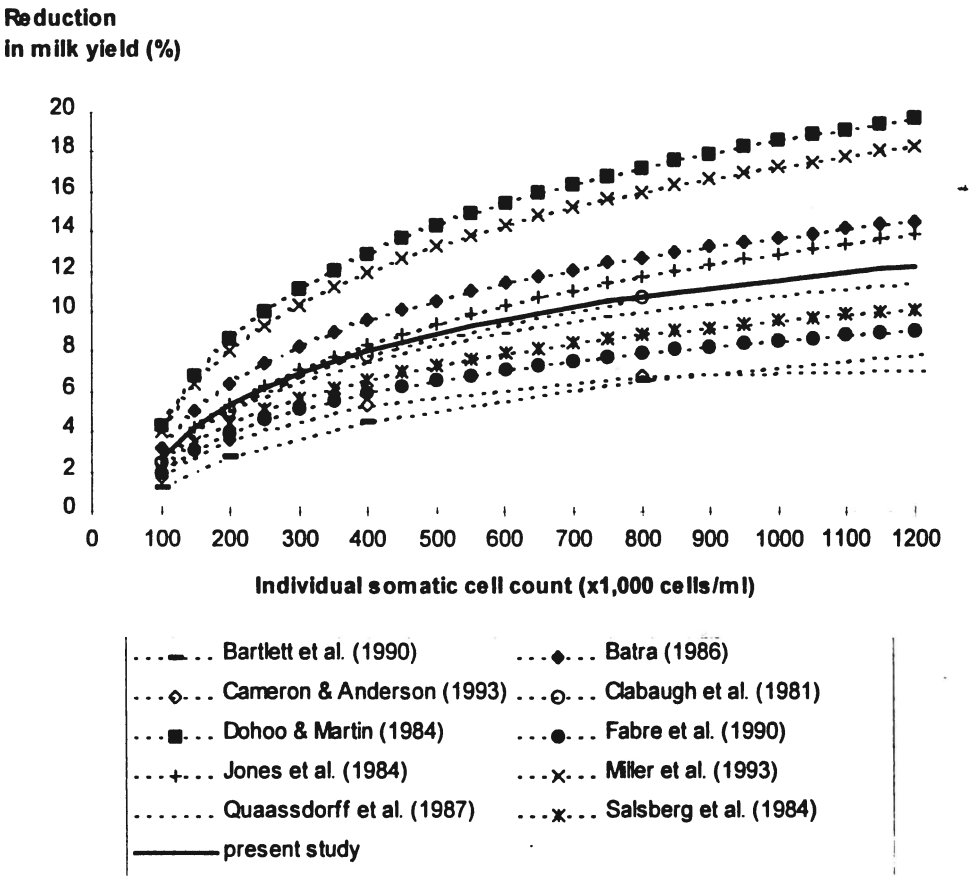

Fig. 4 Reduction in milk yield for multiparous cows expressed in % of milk yield
French data (continuous line) - Published studies (dotted lines)

Models with interaction between SCC and stage of lactation (models C, D, C', D')

Primiparous cows: the interaction term between LNSCC (or CSCC) and stage of lactation (in 4 classes) did not contribute significantly ($P>0.25$) to the models (models C and C').

Multiparous cows: the interaction term between LNSCC (or CSCC) and stage of lactation (in 4 classes) contributed significantly ($P<0.0001$) to the models (models D and D'). However, the contributions of LNSCC (or CSCC) in the three first classes of lactation stage (<50 d pp, [50-100], [100-150]) were deemed not different (based on estimates). Therefore models D and D' were rerun with stage of lactation split into 2 classes (<150 d pp, ≥150 d pp). The interaction term was again significant in both models. The estimate coefficient associated with LNSCC before day 150 pp was -0.938 (SE = 0.07). The estimate coefficient associated with LNSCC after day 149 pp was -1.690 (SE = 0.04). All variables included in model D were significantly ($P<0.0001$) associated to milk yield at test day. The R^2 associated with model D was 0.52 ($P<0.0001$). When SCC is increased two-fold (e.g., from 100 to 200,000; from 400 to 800,000 cells/ml), the reduction in milk yield is equal to 2.09 and 5.06% respectively before and after 150 days pp. Figure 5 displays the reduction in milk yield (in %) due to SCC (described as LNSCC and CSCC) depending upon the stage of lactation.

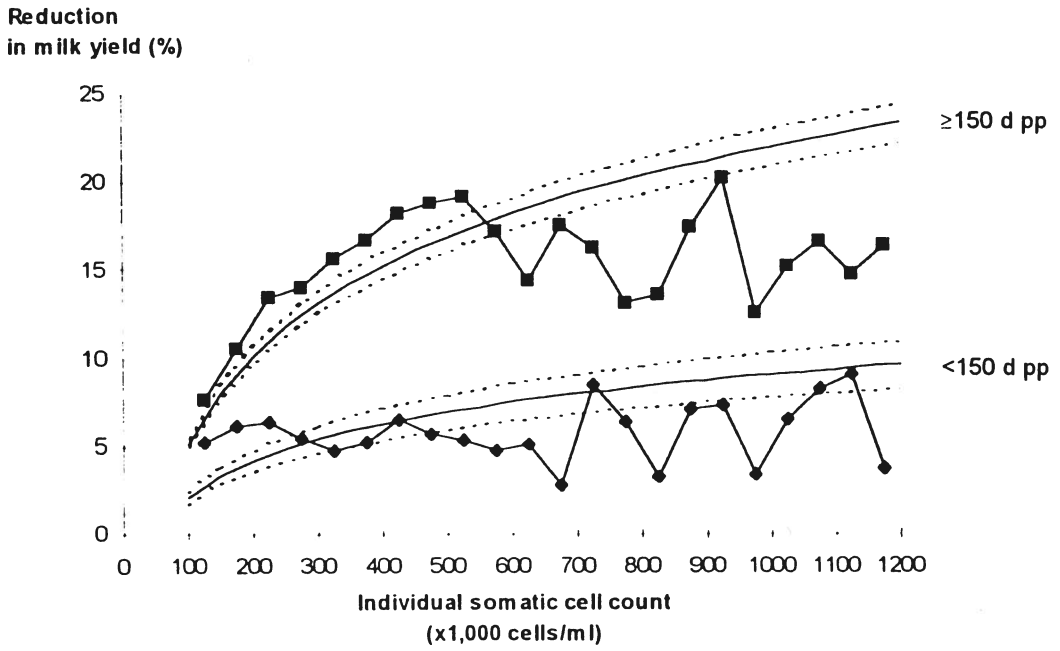

Fig. 5 Reduction in milk yield for multiparous cows in early or late lactation expressed in % of average milk yield produced by control cow-records
square: SCC continuous (model D); lozenge: SCC categorical (model D')
95% CI of the reduction (model D) is displayed in dotted line

DISCUSSION

In both primiparous and multiparous cows, losses in milk yield started to occur at low levels of SCC (>100,000 cells/ml). The reduction in milk yield increased with increasing SCC. The reduction in milk yield estimated from French data appeared to fit the central tendency of those already published in both primiparous and multiparous cows (Bartlett et al., 1990; Batra, 1986; Cameron & Anderson, 1993; Clabaugh et al., 1981; Dohoo & Martin, 1984; Fabre et al., 1990; Jones et al., 1984; Miller et al., 1993; Quaassdorff et al., 1987; Salsberg et al., 1984).

The effect of SCC in primiparous cows was lower than in multiparous cows as previously described by Jones et al. (1984) and Fabre et al. (1990).

No previous studies available to us included an interaction term between SCC and stage of lactation, despite a described relationship between SCC and stage of lactation (Wood & Booth, 1983). Effect of SCC on milk yield appeared to be much lower in early lactation than later (≥150 d pp). High SCC due to long lasting infections may be more frequent. To assess this assumption, dynamics of SCC in successive test days could be included in the models.

The differences in effects between primiparous and multiparous cows and the significant contribution of the interaction term to the models enhanced the hypothesis that the investigation of the effect of SCC on milk yield should consider different types of cows according to parity, stage of lactation (or other covariables).

To investigate the effect of SCC on milk yield, the natural logarithm of SCC was chosen as an independent variable in the present study, in agreement with all previous studies available to us. The replacement of the natural logarithm of SCC by a categorical variable of SCC aimed to assess the relevance and the limits of this common transformation. There is a general agreement between models (the parametric ones with logarithm transformation, and the non parametric ones with classes), that confirms the global relevance of this transformation. However, a detailed analysis of figures 1, 2 and 5 showed that, in comparison to the non parametric models, the parametric models resulted in under-estimated losses in milk yield for SCC below 600,000 cells/ml. In addition, the parametric models resulted in a continuous increase of losses with increased SCC above 600,000 cells/ml, whereas the non parametric ones appeared to show a relative stability of losses in milk yield due to SCC above this value. This latter result was contrary to those from Jones et al (1984) and Bartlett et al (1990), who concluded that quadratic and cubic effects of LNSCC contributed significantly to the reduction in milk yield. In their studies, the quadratic and cubic effects resulted in a higher increase rate in losses when SCC increased. For high SCC values, they did not conclude in stability of losses.

This study suggests that further research is needed to investigate the effect of SCC on milk yield. First, the comparison of non parametric and parametric models leads to conclude that the logarithm transformation of SCC which is commonly used may not be appropriate over a range of SCC from 50 to 1,000,000 cells/ml. To account for these limits, a strategy might exist for using a nonlinear model between milk yield and SCC (e.g. gamma function). Evidence that effect of SCC depends on parity and lactation stage suggests that interaction terms for covariables should be tested when different biological effects can be assumed. In this study, the fact that data were clustered was not accounted for (test days within lactations, lactations within cows, cows within herds). The use of mixed models may improve the goodness-of-fit of models (Goldstein et al, 1995).

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AN ECONOMIC ASSESSMENT OF THE IMPACTS OF MASTITIS CONTROL PROCEDURES USED IN SCOTTISH DAIRY HERDS

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INTRODUCTION

Many studies have assessed the cost-benefit of mastitis control programmes in controlled experiments (Gill *et al.*, 1990) and have generally shown substantial economic returns over the cost of the complete mastitis programmes (Natzke *et al.*, 1972; Schepers and Dijkhuizen. 1991). However, several authors (*e.g.* Howard *et al.*, 1987,;Gill *et al.*, 1990) stress that the results obtained in the field may be different from those obtained under controlled experiments, due to differences in managerial ability of the producers, farming systems and environmental conditions. Moreover, cost-benefit analysis of complete mastitis control programmes does not provide information to determine the economically optimum disease-disease control mix, because some control procedures generating negative net returns are masked by the positive effect of other procedures. It is, therefore, information about the marginal effect of each procedure in mastitis control programmes on the revenue loss from mastitis which is essential for guidance on economically efficient mastitis control (McInerney *et al.*, 1990).

The primary purpose here is to evaluate marginal cost-benefits of mastitis control procedures being used in Scottish dairy herds, and to investigate the technical and economic efficiencies of mastitis control strategies for Scottish dairy farmers with high bulk tank somatic cell (BTSCC).

METHODOLOGY

Data

The association between somatic cell count (SCC) and milk yield was examined using SMRA (Scottish Milk Recording Association) monthly individual cow records from 328,628 cows in 756 herds between April 1994 and March 1995. To establish the relationship

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between SCC and mastitis control procedures, The Scottish Milk Marketing Board's (SMMBs) farm census relating to mastitis control procedures applied by 2187 milk producers in Scotland in May 1993 was merged with the SMRA production records. This information was also merged with the SMMBs' BTSCC penalty records of Scottish herds between November 1993 and October 1994 in order to establish the relationship between BTSCC penalty paid by Scottish farmers and their use of mastitis control procedures. Full description of the records and their manipulation can be found in Yalcin (1996).

Models

Multiple regression analysis was used to estimate the technical impacts of mastitis control procedures on herd average milk yield loss and BTSCC Penalty. The statistical analyses were carried out using the GENSTAT Statistical Package, Version 3.1 (Payne *et al.*, 1994). The models were:

$$AMY_i = \mu + HERD_i + lnWASCC_i + lnWASCC_i^2 + ALAC_i + ASTAGE_i + SEASON_i \quad [1]$$

$$lnWASCC_i = \mu + MCP + MILKSYS + REGION + SEASON_i + AMY_i + ALAC_i + ASTAGE_i + I \quad [2]$$

$$PMP\% = \mu + MCP + MILKSYS + REGION + I + lnBTSCC \quad [3]$$

where:

AMY_i	Herd average milk yield (litre/cow/day) in test month i
μ	Overall mean
$HERD_i$	Effect of $Herd_i$
$lnWASCC_i$	Weighted herd average SCC in test month i in a natural log form
$lnWASCC_i^2$	Quadratic form of $lnWASCC_i$
$ALAC_i$	Herd average lactation number in test month i
$ASTAGE_i$	Herd average stage of lactation in test month i (Month)
$SEASON_i$	Season in test month i (Summer, Winter)
MCP	Mastitis control procedures (post milking teat disenfection (PMTD), dry cow therapy (DCT), udder preparation (UP), milking machine test (MMT), use of individual cow recording service (ICSCCREC) and number of men employed in the milking unit (MEN)).
MILKSYS	Milking systems (Byre, Parlour)
REGION	Milk Marketing Boards in Scotland (Scottish Milk Marketing Board (SMMB), North of Scotland Milk Marketing Board (NOSMMB) and Aberdeen and District Milk Marketing Board (A&DMMB)).
I	Interactions amongst the selected MCP and other variables related to mastitis control
PMP%	Percentage of months in a year in which the BTSCC penalty was paid.
lnBTSCC	Arithmetic mean BTSCC from November/93 to October/94 in natural log form.

Notes:

1) + sign does not refer to the direction of relationships.

2) The equations 1, 2 and 3 will be called milk yield, SCC and BTSCC penalty equation respectively in the rest of the text.

The Estimation Procedures: Regression estimations above were based on the herd average figures on the grounds that there were no known differences amongst cows within herds in terms of the application of mastitis control procedures considered in this study. As mastitis control procedures affect milk yield indirectly by decreasing mastitis infection (somatic cell counts), the effects of the mastitis control procedures on AMY were estimated as a two-stage process; firstly, determining the effect of WASCC on AMY (the milk yield equation), then the effect of the procedures on the WASCC (the SCC equation). Since the quadratic term of WASCC was derived from the multiplication of the lnWASCC term by itself, these two terms were naturally highly correlated. In order to reduce the correlation between linear and quadratic terms of lnWASCC, the mean value of lnWASCC was subtracted from each actual value of lnWASCC before it was squared. The impact of lnWASCC on AMY was corrected according to other variables which cause variation in AMY. These variables were ALAC, ASTAGE and SEASON. Some of the important confounding variables which cause variation in AMY, such as farming system and feeding policy could not be included in the equation due to the lack of data on these variables. For this reason, they were included in the equation as an overall herd effect.

The SCC equation (equation 2) establishes the relationship between lnWASCC and mastitis control procedures (PMTD, DCT, MMT, UP, ICSCCREC). The estimates were corrected according to the other available variables causing variation in herd average somatic cell counts (ALAC, ASTAGE, AMY, SEASON and REGION). The statistical significance of interactions amongst PMTD, DCT and MMT and UP and their interactions with MILKSYS and SEASON were tested.

The BTSCC penalty equation (equation 3) establishes the relationships between the likelihood of Scottish farmers paying BTSCC penalty and mastitis control procedures. Since the likelihood of paying BTSCC penalty is mainly determined by the level of herd BTSCC, the linear term of the BTSCC (in natural logarithmic form) was included in the model to separate the effect of mastitis control procedures on PMP% from that of BTSCC. The BTSCC penalty is a problem for herds whose BTSCC level is above 400,000 cells/ml. Therefore, to investigate the impact of mastitis control procedures on PMP%, only herds whose average BTSCC level was greater than 300,000, and lower than 600,000 cells/ml, were considered on the grounds that herds below 300,000 cells/ml were rarely faced with the BTSCC penalty problem, and herds above 600,000 cells/ml were always faced with it.

RESULTS and DISCUSSION

Effect of SCC on Milk Yield

The regression results for AMY (l/day/cow) as a function of lnWASCC (The milk yield equation) and other production characteristics are presented in Table 1. As expected, the estimated AMY was negatively correlated with lnWASCC. The linear and the quadratic terms of the lnWASCC were significant ($P< 0.01$). The model explained 79.4 per cent of the variation in test day AMY of which the effect of lnWASCC accounted for 10.6 per cent.

Table 1. Estimated regression model of AMY against lnWASCC and other production characteristics, SMRA herds, 1994-95 (The milk yield equation)

	Dependent variable: Test day AMY (l/cow/day)		
	Estimate	SE	t-value
Constant	21.686	0.740	29.30**
lnWASCC linear	-1.002	0.108	-9.28**
lnWASCC Quadratic	-0.277	0.103	-2.68**
ALAC#	0.9375	0.094	9.97**
ASTAGE#	-1.4724	0.026	-57.32**
SEASON summer	1.5632	0.058	27.15**

Adj. R^2 : 79.4 % (Includes herd effect whose coefficient estimates are not presented in the table)
** significant at P<0.01; # Herd Averages

Estimated AMY loss from a unit change in lnWASCC varied according to herd level WASCC. It was 0.56 l/cow/day at lnWASCC equals to 5 (WASCC = 148,000 cells/ml), 1.67 l/cow/day in herds where lnWASCC was 6 (equals to BTSCC penalty threshold of 400,000 cells/ml) and 2.23 l/cow/day where lnWASCC was 7 (maximum WASCC of score of 1,097,000 cells/ml in the data set). Average milk yield loss (This was derived by weighting the estimated milk yield losses in each SCC category by the number of herds in each SCC category) from a unit change in lnWASCC was calculated as 1.01 l/cow/day. These finding are in fair agreement with the 0.44-2.09 litre/cow/day per unit increase in individual cow SCC reported by a variety of authors (Raubertas and Shook. 1982, Jones *et al* 1984; Dohoo *et al* 1984; Salsberg *et al* 1984; Batra, 1986; Barlett *et al* 1990).

Effect of Common Mastitis Procedures on SCC

Amongst the interaction terms tested those between DCT and MILKSYS, MMT and MILKSYS, and PMTD and UP were found to be significant at P<0.05 (Table 2). Use of UP and regular use of ICSCC recording service were associated with a higher lnWASCC than their alternatives. PMTD lowered lnWASCC, but UP had a detrimental effect on the effectiveness of PMTD. These differences were all statistically significant (P<0.05). Effects of DCT and MMT varied according to milking systems. Under the byre system the difference in lnWASCC with and without the application of DCT was not statistically significant (P>0.05), whereas, the herds using DCT under a parlour system had significantly (P<0.01) lower lnWASCC than those not using DCT. Use of MMT was significantly (P<0.01) associated with a lower lnWASCC in parlour systems, whereas it was significantly (P<0.01) associated with a higher lnWASCC in herds having byre systems. The model explained 21.4 per cent of the variation in the lnWASCC.

Table 2 Estimated regression coefficients for the effects of mastitis control procedures and other production variables on lnWASCC in SMRA herds (The SCC equation).

Dependant variable: Test day lnWASCC			
	Estimate	SE	t-value
Constant	5.1	0.116	44.12
ALAC	0.0963	0.0115	8.36**
ASTAGE	0.04131	0.00673	6.14**
SEASON summer	0.1165	0.0145	8.03**
AMY	-0.02735	0.00215	-12.71**
REGION NOSMMB	0.1379	0.0266	5.18**
REGION ADMMB	0.2667	0.051	5.23**
ICSCCREC regular#	0.0793	0.0138	5.75**
MILKSYS parlour	0.362	0.107	3.40**
PMTD yes	-0.1687	0.0346	-4.88**
DCT yes	0.0016	0.0732	0.02
UP yes	0.078	0.0357	2.19*
MMT yes	0.2223	0.0399	5.57**
MEN >1	0.0305	0.0159	1.92
MILKSYS parlour .DCTyes	0.250	0.1	-2.50**
MILKSYS parlour .MMTyes	-0.2827	0.0449	-6.29**
PMTD yes .UP yes	0.1181	0.04	2.95**

Adj. R^2 21.4 %; * Significant at P<0.05, ** significant at P<0.01, # greater than 9 records in a year

The higher lnWASCC from use of udder preparation was not an unexpected result. Several previous studies reported a similar finding, particularly UP with bucket & reusable cloth. Gunn *et al* (1994) found a highly significant (P<0.001) increase in the BTSCC of herds using UP either with continuous water only or bucket & reusable cloth in Scottish dairy herds. Logue (1995) also reported significantly higher BTSCC figures for herds using a reusable cloth for udder preparation amongst dairy herds in the Isle of Man. Gill *et al* (1990), and Miller (1991) reported positive but not statistically significant differences in SCC level between using or not using a reusable cloth in Ontario (Canada), and Michigan (USA) dairy farms respectively. Howard *et al* (1987), on the other hand, reported a significantly higher SCC score in cows even under the application of individual paper towel in Texas (USA) dairy herds. Howard *et al* (1987)'s findings may be based on the fact that udder quarters are independent. Infection established in one quarter can be spread to the other quarters even when farmers use a single paper towel for each cow. The findings of this study as well as the others mentioned above call into the question the efficacy of UP methods. UP with continuous water or bucket and reusable cloth investigated in this study was not only the most costly practice (the opportunity cost of labour alone for this practice was calculated to be £9.2-12.2 depending on the methods used) but also encourages the spread of the infection and had a detrimental effect on PMTD, yet it was a widely adopted procedure amongst Scottish farmers (79.1 per cent in 1993). This study showed that, apart from UP, other widely used

mastitis control procedures (PMTD, DCT and MMT) appeared to be effective and their effects were essentially additive in reducing somatic cell counts.

The regression results showed that herds frequently receiving ICSCC records had higher SCC, a result which appears to be in contradiction to advice of veterinarians that ICSCC tests are very useful as indeed is the subsequent detailed bacteriology investigation(s) of such high ICSCC cows. However, it is highly likely that the adoption of this procedure by farmers in response to a BTSCC problem is the reason for this apparently anomalous result. Gunn *et al* (1994) reported that amongst 57 high BTSCC penalty herds in Scotland in December 1993, 67 per cent were regular users of the ICSCC recording service.

Unexpected effects of MMT and DCT were found in byre systems. In part the data was difficult to interpret because of small blocks, but in addition herds having byre system are small and the farmers tend to be less hygiene conscious (Logue *et al.*, 1993; Gunn *et al.*, 1996). Thus a swith ICSCC testing we suspect that many such herds adopted MMT in response to the BTSCC problem, and, furthermore we had no information as to whether the herds had taken corrective action if their milking machine was found to be faulty as a result of the MMT. Similar argument can be made for DCT. It would also have been good to know information on quality of applications and type of antibiotic used for DCT. Inadequate hygiene and/or cheap but less effective antibiotic may result in failure of DCT (Blowey and Edmondson, 1995).

The SCC regression equation also showed that while the mastitis control procedures were important, other elements accounted for a greater proportion of the variation in herd mean SCC. For instance, the bacteriological status of the udder is the main determinant of the level of SCC and can explain 24 per cent of the variation in ICSCC (Brolund, 1985). Fenlon *et al* (1995) found a very high correlation between number of *Streptococci* found in bulk tank milk and BTSCC in Scottish herds, but this correlation was found to be much smaller in the case of *S. aureus* infections. The regression analyses, however, made no allowance for the bacterial status of the udder due to lack of data. It is important to appreciate that mastitis control procedures have very different outcomes, depending upon the predominant cause of mastitis in the herd. For instance, the elimination rate of *S. agalactia* cases by antibiotic therapy is very high, and it can be eradicated from entire herds (West, 1979), whereas, antibiotic treatment, even in the dry period, is not very effective against *S. aureus* cases (Logue *et al.*, 1995).

Because the farm census on mastitis control procedures was limited to 'yes' and 'no' options, detailed analysis on the quality and frequency of farmers' use of mastitis control procedures could not be conducted in this study. The efficacy of PMTD, for example, depends on a number of factors, such as the frequency of its application, whether dip cups or sprays are used, how efficiently the teats are covered, which disinfectant is used, etc. Similarly, the farm census data available only allowed comparison on whether use of complete DCT was effective. However, the type of antibiotic is one of the main determinants of the efficacy of DCT (Blood and Rodostits, 1989). Moreover, the efficacy of applying DCT to the whole herd regardless of which cows have a mastitis problem has been questioned in recent studies. Howard *et al* (1987), Gill *et al* (1990) and Miller (1991) reported insignificant association between complete DCT and SCC in spite of the fact that in the study of Gill *et al*

(1990) selective DCT was significantly associated with lower SCC. As the extensive use of antibiotics has implications, not only on farm profitability (extra cost, risk of antibiotic residue penalty), but also on consumer concerns about food quality and safety, it is important to investigate the efficacy of DCT in detail (Gill *et al.*, 1990).

There was on average a gap of 1.5 years between MMB's farm census on mastitis control and other production data used in this study. However, the changes in the use of mastitis control procedures within 1.5 years does not seems to have been dramatic enough to change the statistical relationship between lnWASCC and mastitis control procedures since the same relationship was found when mastitis control variables recorded in May 1993 were regressed on mean BTSCC in May 1993, and the results of this study are well in agreement with the findings reported by a variety of authors.

Effect of Common Mastitis Control Procedures on BTSCC Penalty

The results of the BTSCC penalty regression equation are presented in Table 3. Amongst the mastitis control procedures only the association of DCT with PMP% was found to be statistically significant at $P<0.01$. Although, PMTD and MMT were again associated with a lower level of BTSCC penalty, their effects on the BTSCC penalty were not statistically significant at $P<0.05$. As expected, BTSCC was highly correlated with BTSCC penalty status of the herds. No interactions amongst the mastitis control procedures was found to be significant at $P<0.05$ in the BTSCC penalty equation.

Many mastitis control procedures are aimed at preventing the spread of infections, i.e. decreasing the incidence of mastitis. DCT, however, also aims to cure (shorten the duration of infection). In herds which have high incidence of mastitis, the infection has already been widely spread. In this situation, at least in the short term, preventive measures are less effective since the level of infection is high. Since the data in the BTSCC penalty equation was based on high BTSCC herds, this probably explains why only dry cow therapy significantly decreased the percentage of months in a year in which the EU threshold was exceeded.

Table 3 Estimated regression coefficients for the effects of mastitis control procedures and BTSCC on PMT% in Scottish dairy herds, November 93-October 94 (the BTSCC penalty equation).

Dependent variable: % of Months in a year in which BTSCC Penalty paid			
	Estimate	SE	t-value
Constant	-924.2	28.1	- 32.84**
ICSCCREC irregular	1.18	2.71	0.44
ICSCCREC regular	4.39	2.83	1.55
MILKSYS parlour	3.19	1.95	1.64
PMTD Yes	-2.35	1.94	-1.21
DCT Yes	-11.27	3.81	-2.96**
UP yes	4.17	2.25	1.85
MMT Yes	-2.8	2.03	-1.38
MEN two	-1.68	1.71	-0.98

Dependent variable: % of Months in a year in which BTSCC Penalty paid			
	Estimate	SE	t-value
REGION NOSMMB	1.69	2.77	0.61
REGION ADMMB	3.09	4.44	0.70
LnBTSCC	162.96	4.69	34.73**

Adj. R^2 : 86.2 %; ** Significant at P<0.01

ECONOMIC ANALYSIS

Marginal Revenues (MR) of each mastitis control procedure were obtained by using the appropriate regression equations to predict the reduction in lnWASCC and hence the saving in milk yield value and BTSCC penalty. The marginal costs of the mastitis control procedures considered in the economic analysis were obtained from McInerney *et al* (1990) but were updated for the inflation rate (Retail Price Index) in Britain. Unlike McInerney *et al* (1990), opportunity costs of labour incurred in the application of the procedures were also taken into account. A fuller description of the economic analysis can be found in Yalcin (1996).

The marginal economic outcomes of some selected mastitis control procedures (those associated with a lower lnWASCC in the regression analyses) in Scottish dairy herds facing high SCC problems[1] (those whose WASCC > 400,000 cells/ml) are given in Table 4. The results of the economic analysis show that applications of PMTD, DCT and MMT can be cost-effective provided that the quality of the applications are as recommended and/or the protection they give are not undermined by the antagonistic effect of other procedures. The dairy farmers that used a PMTD and did not apply any udder preparation method, and those that used a DCT and MMT in parlour systems obtained a net revenue of £1.4, £3.9 and £1.1 respectively per £1 invested in these mastitis control procedures. Since no significant interaction was detected between PMTD, UP and milking system, the PMTD and UP columns in Table 4 represent all farms. But the figures were substantially the same for herd using parlour system.

Table 4 Marginal cost-benefit of selected mastitis control procedures

Mastitis Control Strategies	**PMTD**	**PMTD with UP**	**DCT in parlour**	**MMT in Parlour**
AMY loss saved due to the strategies (l/year)	85.98	25.77	126.52	30.71
AMY loss saved due to the strategies (£/cow/year)*	15.13	4.54	22.27	5.41
Decrease in BTSCC Penalty due to the Strategies(%)	2.40	2.40	11.30	2.80
Value of BTSCC penalty saved (£/cow/year)	0.87	0.87	4.10	1.02
Marginal Revenues from the strategies (£/Cow/Year)	16.00	5.41	26.37	6.42
Marginal Cost of the strategies (£/cow/year)	11.53	20.68	6.70	5.84
Net Marginal Revenues from the strategies(£/cow/year)	4.47	-15.20	19.67	0.58
Return from £1 investment	1.39	0.26	3.94	1.10

* Considered the cost of extra feeding incurred due to increased milk yield

[1] Note that the result of economic analysis would be different for herds which are not facing the BTSCC penalty problem

The Technical and Economic Efficiency of Scottish Dairy Farmers in the Use of Mastitis Control Procedures: Further to the above mentioned marginal cost-benefit analyses, the total revenue loss and the total mastitis control expenditure under all possible combinations of PMTD, DCT and MMT under different milking systems or udder preparation (depending on the interactions) were calculated. This allowed construction of the loss-expenditure frontier McInerney & Turner (1989) developed. With the aid of the loss-expenditure frontier, the technical and economic efficiencies of Scottish dairy farmers facing high BTSCC problems in the use of mastitis control practices were examined. The results are presented in Table 5 and Figure 1.

Each point plotted on the graph in Figure 1 depicts the level of the revenue loss and the control expenditure incurred under a particular mastitis control strategy presented in Table 5. The line matching the lowest points is the loss-expenditure frontier[2] defined by McInerney & Turner (1989). This line illustrates the level of technically minimum attainable revenue loss (technical efficiency) under different levels of mastitis control expenditure. As can be seen from the figure, the shape of the line is concave to the origin. This illustrates that as mastitis control expenditure increases, the revenue loss decreases but at a decreasing rate. This is the evidence of the existence of the economic law of 'diminishing marginal return' on mastitis control expenditure (McInerney *et al.*, 1990).

Table 5 The revenue loss , the control expenditure and the total economic cost in different mastitis control strategy combinations

Mastitis control strategies	No of herds*	Control expenditure (£)	Revenue loss(£)	Total costs (£)
1. None	2	0.0	90.2	90.2
2. MMTbyre only	1	5.8	109.1	115.0
3. MMTparlour only	2	5.8	83.8	89.6
4. DCTbyre only	3	6.7	86.2	92.9
5. DCTparlour only	1	6.7	63.8	70.5
6. UP only	14	9.2	96.1	105.2
7. PMTD only	1	11.5	74.2	85.7
8. DCTbyre+MMTbyre	4	12.5	105.2	117.7
9. DCTparlour+MMTparlour	6	12.5	57.4	69.9
10.MMTbyre+UP	11	15.0	115.0	130.0
11.MMTparlour+UP	9	15.0	89.7	104.7
12.DCTbyre+UP	37	15.9	92.1	108.0
13.DCTparlour+UP	18	15.9	69.7	85.6
14.MMTbyre+PMTD	0	17.4	93.1	110.5
15.MMTparlour+PMTD	4	17.4	67.8	85.1

[2] The last point was not included in the loss-expenditure frontier since the disease control expenditure at this level includes UP that increases revenue loss from the disease.

16.DCTbyre+PMTD	4	18.2	70.2	88.5
17.DCTparlour+PMTD	4	18.2	47.8	66.0
18.UP+PMTD	17	20.7	84.9	105.6
19.DCTbyre+MMTbyre+UP	28	21.7	111.1	132.8
20.DCTparlour+MMTparlour+UP	24	21.7	63.3	85.0
21.DCTbyre+MMTbyre+PMTD	7	24.1	89.2	113.2
22.DCTparlour+MMTparlour+PMTD	15	24.1	41.4	65.5
23.MMTbyre+UP+PMTD	11	26.5	103.8	130.3
24.MMTparlour+UP+PMTD	8	26.5	78.5	105.0
25.DCTbyre+UP+PMTD	29	27.4	80.9	108.3
26.DCTparlour+UP+PMTD	41	27.4	58.5	85.9
27.DCTbyre+MMTbyre+UP+PMTD	41	33.2	99.9	133.1
28.DCTparlour+MMTparlour+UP+PMTD	113	33.2	52.1	85.3

* The Scottish dairy herds whose BTSCC level was greater than 400,000 cells/ml in the 1993 SMMBs' Farm Census.

The dairy farmers on the frontier attaining the lowest revenue loss from subclinical mastitis at a given expenditure level were those who used 1) DCT in parlour only, 2) DCT and MMT in parlour system, 3) DCT in parlour + PMTD with UP, and 5) PMTD with no UP, and DCT and MMT in parlour system. As can be seen from Table 5, the minimum revenue loss of £41.4 cow/year occur due to subclinical mastitis under technically the best control strategy considered in this study. On the other hand, the points above the cost-expenditure frontier represent the dairy farmers whose revenue loss from subclinical mastitis were unnecessarily high for their mastitis control expenditure level.

The economically optimum disease losses/disease control expenditure in this study was attained by herds who used DCT, PMTD and MMT under parlour system. This is the point where total cost (the revenue loss + the control expenditure) of subclinical mastitis was minimum (£65.5/cow/year) amongst the high BTSCC herds (since both axes of the graph are in the same unit (£), the economic optimum is attained at a point where the tangency of the line which is 45^0 to both axes touches the loss-expenditure frontier-McInerney *et al.*, 1990). By taking this figure as a baseline avoidable and unavoidable components of the total cost from subclinical mastitis at both cow, and herd level in high BTSCC herds were calculated, and presented in Table 6.

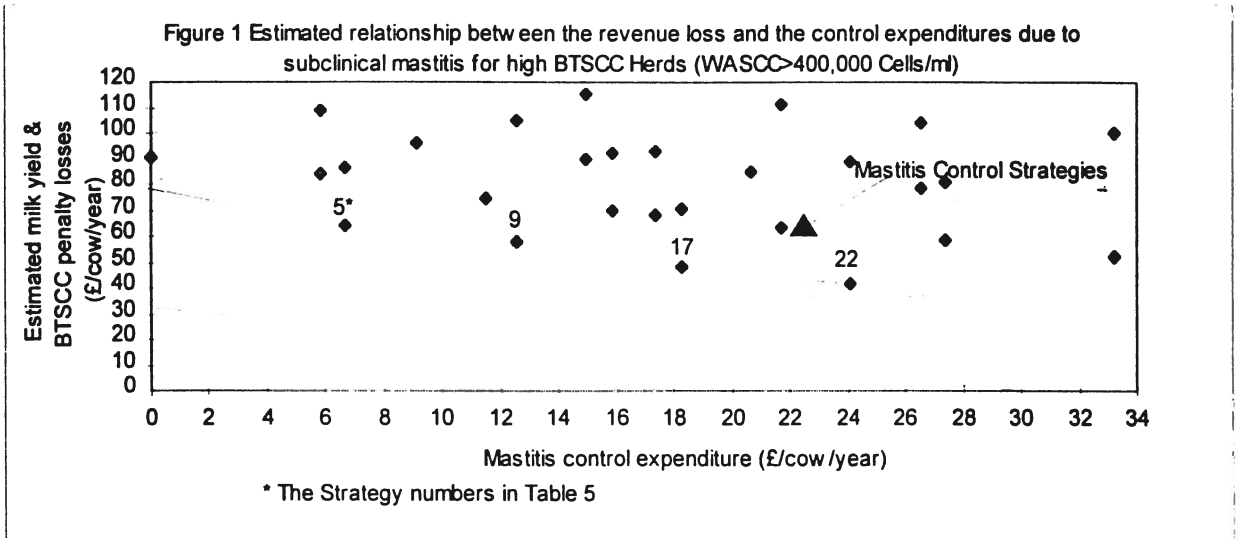

Table 6 Annual average total, avoidable and unavoidable costs from subclinical mastitis in Scottish dairy herds facing high SCC problems [1]

	Minimum Cost	Average Cost[&]	Avoidable costs (£)	Avoidable costs (%)
Cow	65.5	100.0[*]	34.5	34.5
Herd[#]	6,419.0	9,800.0	3,381.0	34.5

1 The costs include milk yield depression adjusted by feed costs saved, BTSCC penalty and expenditure on mastitis control.

& In order to derive this figure the total costs of subclinical mastitis under each mastitis control strategy were weighted by number of Scottish herds using these strategies in the farm MMBs' farm census in 1993 (see Table 3.5).

* Of which £75.3 and £24.8 are the revenue loss and control expenditure respectively.

\# Scottish average herd size of 98 cows/herd was used.

As can be seen from the table, in spite of the fact that subclinical mastitis costs on average £100/cow/year, 34.5 percent of these costs can be avoided by using the best control procedures considered in the analysis. However, these figures vary considerably for individual farms, giving a lot of scope for improvement in this area.

In comparison with DCT, PMTD and MMT gave lower net return. However, the revenue items from mastitis control included only savings in milk yield losses and BTSCC penalty from subclinical mastitis. The other two important revenue items, namely savings due to reduction in forced culling due to persistent clinical cases and cost of clinical mastitis treatment could not be considered in the cost benefit analysis. Therefore, the marginal returns from above reported mastitis control procedures should be treated as minima.

Previously, McInerney *et al* (1990) established the loss-expenditure frontier for subclinical mastitis in the UK and the Netherlands. Using this framework they evaluated the technical and economic efficiencies of UK dairy farmers using one of the 18 different mastitis control strategies. They reported that minimum cost of mastitis is attained by the herds using PMTD the whole year, DCT for all cows and annual testing of milking machines. In this group of herds cost of subclinical mastitis was reported to be £30 /cow/year, that is the

minimum level attainable with the control techniques available and under the economic conditions assumed in their research. They calculated the average cost of subclinical mastitis as £38/cow/year, (excluding cost of clinical cases and forced replacement due to mastitis) of which 27 % was avoidable by employing the most economically efficient approach. Although the proportion of avoidable cost in the total cost of subclinical mastitis in their research (27 %) was close to that reported in this study (30 %), the average cost of subclinical mastitis they reported is less than half that reported here. This appears to be due to the fact that the analysis in this study is based on herds with BTSCC problems, whereas the study of McInerney *et al* (1990) covered all herds in the UK. However, the inputs used in the study of McInerney *et al* (1990) were mostly derived from rough approximations of different findings in the literature since the main objective of their study was to test whether the economic law of 'diminishing marginal returns' applied to mastitis control expenditure. The annual average revenue loss of £67.6/cow found in this study is similar to the estimate of £30-60 reported by Logue *et al* (1995) in Scotland, and to those of £42 and £84 made elsewhere in the UK (Esslemont and Peeler, 1993). The BTSCC penalty cost calculation was based on the 1995 figures. The latest change in the milk hygiene structure in Scotland has resulted in much higher losses from subclinical mastitis in herds facing BTSCC penalty. Gunn *et al* (1996) estimated that under the present market situation in Scotland if a producer reduces his BTSCC from over 4000,000 to under 250,000 cells/ml, the increase in annual gross margin could be as much as £100/cow.

CONCLUSION

This study highlighted 4 important points in terms of technical and economic aspects of mastitis control in dairy herds.

Firstly, interaction exists between mastitis control procedures. Therefore, the efficacy of each mastitis control procedure should not be investigated in isolation as has been the case in many previous studies.

Secondly, application of conventional water based udder preparation methods (particularly with bucket and re-usable cloth but even with continuous water alone) were found to be contraindicated.

Thirdly, the study demonstrated that the overall cost benefit analysis approach is not a suitable tool for economically grounded decision support on mastitis control in dairy herds. The marginal analysis of mastitis control procedures shows that dairy producers could waste some of their investment on mastitis control unless they are aware of whether each mastitis control procedure they apply generates a net economic gain.

Finally, the majority of Scottish dairy herds facing the high BTSCC problem are not operating a maximum efficiency in terms of mastitis control practices. Potentially an average Scottish dairy farmer with a high BTSCC could reduce the costs resulting from subclinical mastitis by £34.5/cow/year by attaining the required economic efficiency in the use of mastitis control practice considered in this study.

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GUIDING DECISIONS ON METHODS AND RESPONSIBILITIES FOR EPIDEMIC DISEASE PREVENTION AND CONTROL: PERSPECTIVES FROM ENVIRONMENTAL AND INSURANCE ECONOMICS

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In a stimulating and provocative paper, Davies (1996) considers the role of the public sector in controlling epidemic diseases of livestock. In his review, Davies addresses the question of "whether it is right that the substantial costs involved in controlling epidemics should continue to be borne by the public sector" which, in turn, "brings into question the role of the public sector in controlling epidemic diseases of livestock". He argues that the contemporary livestock industry is subject to market disciplines which should, in theory, force it to adopt efficient husbandry, trading practices, and controls associated with preventive medicine. However, on a cautionary note, he asks whether there is a feasible alternative to public sector responsibility for epidemic disease control. Specifically, the public sector currently a) regulates livestock movements and takes remedial action when disease outbreaks occur, and b) bears the cost of those actions and protects the industry through compensation payments. Davies concludes that "it is difficult to conceive of a private sector arrangement that will result in effective remedial action" so that "the case for the public sector to continue with this responsibility is overwhelming." Also, "the case for maintaining compensation payments is irrefutable" but, by contrast, " present arrangements do little or nothing to force the livestock industry to accept the disciplines of preventive medicine." The solution to neglect of preventive measures is, according to Davies, some form of insurance system.

This analysis raises important issues. In an era when public sector activity, and especially its budgetary implications, is in many political circles regarded as something akin to mortal sin, there is a danger that Davies' conclusions may gain influence before there is sufficient reflection on the validity of his conclusions. Fundamentally, the issues raised require careful analysis from an economic point of view. This involves much more than comparison of the financial costs and who should bear them. The *rightness* of public sector involvement in Davies' terms should be recast in a way amenable to investigating questions of *economic efficiency*. This paper is an initial exploration of some important economic principles which need to be addressed. To begin with, we draw on environmental economics to illustrate the distinction between private and social optima in economic efficiency, and how a tax (levy) on livestock production may, in principle, encourage a move towards a socially optimal level of

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epidemic disease. Then the implications of insurance schemes are reviewed before drawing conclusions which set the context for the empirical research which ought to follow.

EPIDEMIC DISEASE AND THE ECONOMICS OF POLLUTION

From the perspective of an individual livestock producer, disease in the herd or flock is equivalent, in its economic effects, to that of a negative input (Howe, 1985). Insofar as the effects of the disease are contained within the individual herd or flock, lost output, reduced quality of product, and additional costs incurred in taking remedial action are all private in the sense that they are internal to that business, and that business alone. For epidemic disease, the costs are much more extensive. As disease spreads from an original source, other livestock enterprises and farm businesses are affected. In other words, there are effects on other producers which are external to the original source. What is more, the farmer at the original source does not compensate those others who are affected. In economic terms, when a) an activity by one agent causes a loss of welfare[1] to another agent, and b) the loss of welfare is uncompensated, an *external cost* (also known as a *negative externality* or *external diseconomy*) is said to exist. Although our context is epidemic disease, these principles are immediately familiar from the analysis of sources of pollution and its effects within the framework of environmental economics.

The optimal level of external cost

It might be concluded that an ideal scenario would be where external costs were reduced to zero. In practice, of course, this is typically a technical impossibility for epidemic diseases. In exceptional cases, rapid remedial action in the event of an isolated outbreak may cut down the spread, perhaps confining the outbreak to the source farm alone. More generally, inability to exert perfect control over the spread of disease even if it is single source means that someone, somewhere, suffers external costs. In any event, economic theory shows that reducing an externality to zero, even where technically possible, is unlikely to be in the best interests of society. That is because costs are also incurred in the effort to reduce externalities, and these must be weighed against the benefits resulting from their reduction. A key issue, therefore, concerns identification of the optimal level of externality. The relevant principles are illustrated with the help of Figure 1, which draws on elementary pollution economics.

Figure 1 presents a simple stylised model of the key relationships. The horizontal axis, labelled 'level of economic activity', broadly coincides either with scale of livestock production on an individual farm or across a number of farms in a given geographical area (perhaps a country or a sub-region of a country). It is asserted that, as a general rule, the more dense the livestock population the greater the risk of disease given production techniques including current practices for disease control. Assuming that producers aim to maximise profits (net revenue), output will increase to OQ_1 where line MNPB meets the horizontal axis.

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[1] Welfare in its specific economic sense of social well-being.

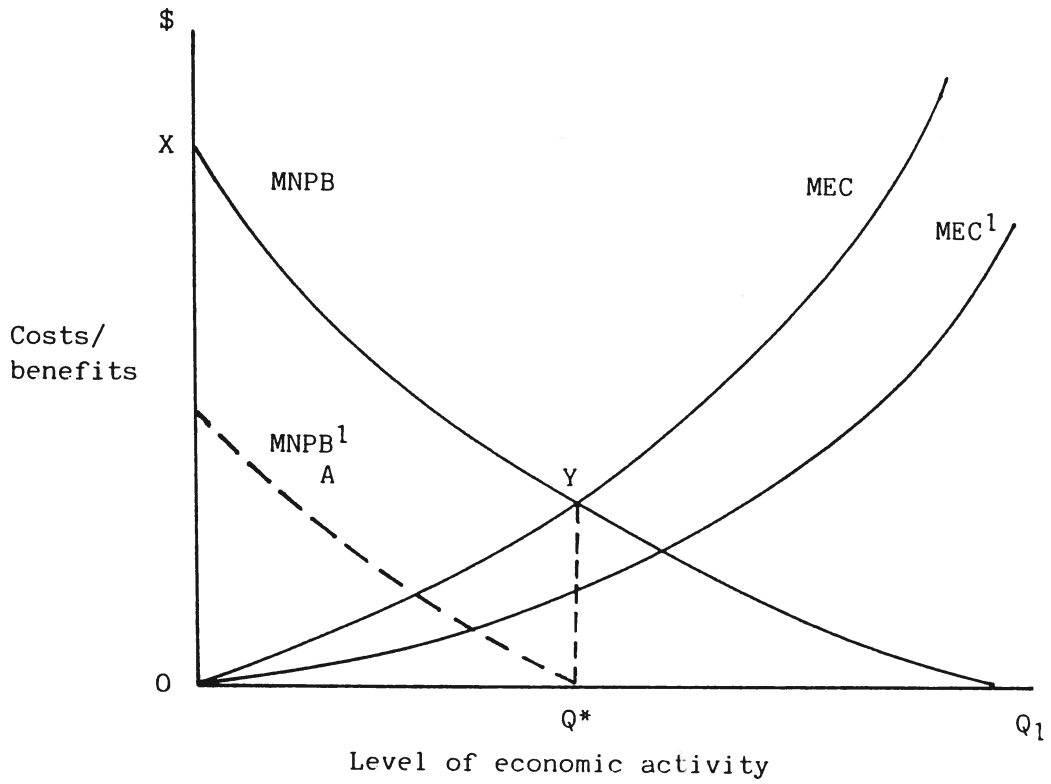

Fig. 1 The optimal level of external cost

MNPB, the schedule of marginal net private benefit, is explained as follows. Every increment of output earns additional (marginal) revenue in return for the outlay of additional (marginal) production costs. The difference between these magnitudes is therefore the marginal net (private) benefit. Because of the familiar law of diminishing marginal returns, marginal outlays on resources are associated with positive but declining marginal revenues until a point is reached where the two equate, i.e. at OQ_1. Since cumulative net marginal private benefits equal total net private benefits, it follows that total net benefits are maximised at OQ_1. Thus, left to pursue their private interests, individual livestock producers will aim to produce at OQ_1 because they take no heed of external costs imposed on third parties.

MEC represents marginal external costs associated with different levels of production. In this specific instance, the MEC schedule reflects the *expected* external costs of disease given the probabilities of outbreaks associated with different scales of livestock production. In that sense, disease outbreaks are like random pollution events such as oil spillage from a wrecked tanker or radiation emission from a nuclear accident. By contrast, environmental economics problems more usually concern MEC schedules which relate, say, to factories emitting a constant stream of pollutants whose magnitude is directly proportional to the scale of production.

As is common in economics, the location which defines an optimum is where marginal benefits equal marginal costs. The intersection of MNPB with MEC therefore defines Q* as the optimal volume of production which coincides with the optimal level of externality, OYQ*. It follows that OXY, summarised as area A, is the socially optimal total net benefit. This

assumes that we do not choose to weight the gains or losses of any one livestock farmer more than any other. This seems on the one hand to be intuitively reasonable, since it is commonly in the nature of major epidemic diseases that herds initially infected occur by chance, so that today's 'polluter' may be tomorrow's 'sufferer'. On the other hand, the assumption is not so reasonable if there was scope on a source farm to implement preventive measures which would have reduced the risk of initial infection. Such distributional effects should be taken into account when disease control policies are designed.

Since $Q^* < Q_1$, this suggests that the socially optimum scale of production is below the private optimum. Broadly, this indicates the need to reduce the density of susceptible livestock numbers. Would this matter to consumers of livestock products? It might if trade barriers prevented importation of substitute products from other countries because a reduction in domestic supplies, other things remaining equal, would then be expected to raise domestic consumer prices.[2] With freer trade, and on the realistic assumption for many countries that the scale of importation is insufficient to make much difference to international market prices, Davies (op.cit. page 80) is correct. Preoccupations with assuring food security which so influenced earlier generations is not nowadays of any great concern. We would simply find alternative sources for our livestock products.

Taxation for optimal production

Where an externality exists, the usual policy recommendation is to internalise the external cost so that the private optimum level of output coincides with the social optimum. To achieve this end, the recommended policy instrument is a per unit production tax equal to Q*Y. Each livestock unit produced would therefore be taxed with the effect of shifting the MNPB schedule towards the origin. In relation to Davies' discussion of the role of the public sector in epidemic disease control, imposition of a tax (the term 'levy' may be considered more palatable) is attractive. It serves two desirable functions. First, by virtue of its negative impact on marginal net private benefits it provides a disincentive to production which, in itself, has positive impacts on the incidence of epidemic diseases. Thus spontaneous market behaviour in response to imposition of the tax has benefits for disease control. Second, the tax revenue is a budgetary gain to the exchequer. If it were so wished, these tax revenues could be earmarked for state expenditures devoted to improving still further the scope for epidemic disease control.[3]

This is an important consideration, because a headage tax (levy) encourages disease reduction only by reducing output and not by stimulating the use of preventive measures. It would be preferable if disease (pollution) could be diminished by reducing the amount of disease (emissions) per unit of output produced, because then disease reduction could be

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[2] In practice, the extent to which consumers experienced price increases would depend, among other things, on what proportion the raw material livestock products accounted for in the retail price of a given final consumer product.

[3] i.e. a hypothecated tax.

achieved without sacrificing so much output. While in the environmental field such desirable changes in production processes can be encouraged by taxing pollutant emissions, the corresponding option is not feasible for disease control. That is because disease externalities are not predictably emitted in conveniently measurable units. Thus a headage levy would need to be supplemented by other incentives to encourage implementation of better preventive practices. In terms of Figure 1, the impact of better prevention would be to cause the MEC schedule to be shifted downwards to the right.

A headage tax nevertheless has the virtue of administrative simplicity, and may nowadays be politically feasible even if unpopular with livestock producers. Of course, there might be an incentive for some farmers to cheat by rearing more livestock than officially declared for the purposes of taxation, and so there would have to be effective enforcement with penalties for evasion. Moreover, a flat rate tax might be subject to the criticism that it is unfair since all producers are taxed at the same rate regardless of the extent of the preventive measures each takes. Indeed, the financial burden of the tax might deter some producers from making expenditures for disease prevention. Whether or not such considerations are of material significance can be assessed only by making empirical estimates of the financial implications of the tax relative to prevention outlays.

Empirical considerations

In practice, there would also be practical difficulties in setting the tax level. For the optimal per unit tax it is necessary to locate point Y. This requires knowledge of the MNPB and MEC schedules over sufficient range in the neighbourhood of Y. If livestock producers are all intent on maximising their private net benefits as would be expected, any empirical information relating to profitability - say from enterprise costs data - would tend to be located in the neighbourhood of Q_1, not Y. Thus identifying the MNPB empirically may be an obstacle to definition of the social optimum.[4] In turn, identifying MEC requires knowledge of disease incidence typically associated with different scales of livestock production, and a full economic assessment of the consequent social costs. This appears to be a feasible, if challenging, proposition for intensive livestock production such as pigs. That said, lack of perfect knowledge about the MNPB and MEC schedules does not actually preclude the introduction of a headage levy. Whenever a tax is introduced it is without full information and therefore its effects can never be fully known beforehand.

The optimal level of precaution

A limitation of the framework presented above was that incentives to take preventive measures were essentially treated as a secondary issue. As was noted earlier, though, disease outbreaks have a counterpart in pollution accidents, in that epidemic disease is not a persistent feature of the production process but rather a potential hazard. The literature on environmental

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[4] Perhaps this is not an insurmountable obstacle, because comparable problems in environmental economics can be addressed by the application of linear programming techniques.

hazards is therefore illuminating in relation to precautionary/preventive decisions for disease control.

When a producer stands to lose personally from a hazard occurring then there is a private incentive to take some precaution.[5] In the familiar way, economic principles show that a producer acting in private interest and with no obligations to others will expend resources on precautionary measures up to the point where the marginal cost of precaution equals the marginal expected benefits of precaution. These benefits are the marginal expected damages to own production which are avoided. The privately optimal level of precaution is given by point OP_1 in Figure 2, where the schedule for marginal cost of precaution (MCP) intersects that for marginal private expected benefits (MPEB). However, whilst this is privately optimal, if there are externalities associated with the hazard then it is not socially optimal. Since the social impact is greater than the private impact, the marginal social expected benefits of precaution are higher than the private. Correspondingly, the socially optimal level of precaution will be at point OP*, where MCP intersects the marginal social expected benefits schedule (MSEB), a higher level than what private interest would dictate. Thus the market cannot be relied upon to induce sufficient levels of precaution.

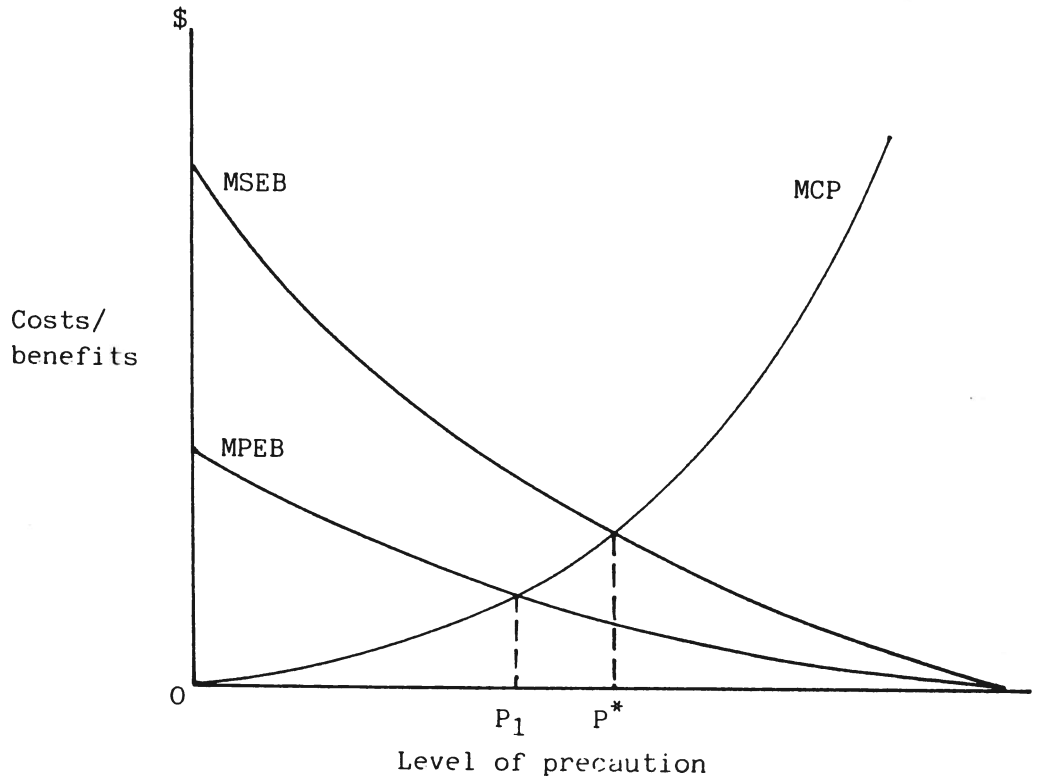

Fig. 2 The optimal level of precaution

In hazardous situations with potentially large impacts on society, producers can be prompted to take precautions by making them liable for the damages they cause to others.

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[5] Currently, with almost full compensation for livestock losses from state funds, there is very little private incentive.

Again, this is equivalent to a process of internalising external costs. When the payouts a producer might have to make are potentially high, he will typically aim to take out insurance cover against adverse outcomes. In fact, even when there is no liability for causing damage to others a producer might seek insurance to cover losses arising in his own business. Currently, incentives for livestock farmers to take out insurance to cover even private losses from epidemic disease are curtailed whenever they are compensated from the public purse. It can not be assumed that such policies will be maintained indefinitely.

INSURANCE FOR EPIDEMIC DISEASE CONTROL

A principal conclusion from Davies' discussion is that some form of insurance system is likely to provide appropriate incentives to induce livestock producers to accept the disciplines of preventive medicine. It is to examination of this contention that the paper now turns. There are essentially two themes to the discussion. First, it has to be established if insurance is an *efficient* instrument to cause livestock producers to protect themselves against the adverse effects of livestock diseases. Second, and growing out of the first, is the question of whether, as a result of individuals insuring, there are social benefits by virtue of an expected reduction in external costs. As part of the analysis we also address an issue overlooked by Davies, but relevant to his theme of the role of the public sector i.e. whether insurance should be left to the private sector or the responsibility of some agency acting on behalf of the state.

Insurance and the individual

A private insurance market will emerge when circumstances allow both insurants (in this case livestock producers) and insurers (insurance companies) to benefit from a policy. The insurant benefits from being able to reduce risk by taking out a policy. Interpreted slightly differently, this is equivalent to their purchasing greater certainty. With insurance, a farmer can counter the adverse consequences of livestock disease by paying a premium which entitles him to recover financial compensation for the disbenefits of an outbreak. In the simplest case, therefore, even if a farmer suffers the effects of disease-induced economic losses, receiving compensation from an insurer effectively should leave the business in exactly the same economic position as if the outbreak had never occurred.[6]

The financial premium paid by a livestock producer will be determined by a) the probability of a disease outbreak in his livestock enterprise and b) the consequent damage. For an *actuarially fair premium*, the premium per dollar (or whatever) of coverage will exactly reflect the actual probability of a disease outbreak. In such circumstances, the income of the insurance company will equal the expected payments. A simple example will clarify this point. Suppose that an insurance company takes the view that the typical annual cost of disease outbreaks in a livestock enterprise is $50000, but that it will cover its claims by setting premium of $100 for each livestock farmer who takes out a policy to cover such losses. It

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[6] The issue is inevitably complicated where, for example, pedigree breeding stock are slaughtered to the detriment of long-term economic performance of the business.

follows that for every payout of $50000 the company must receive 500 premia of $100 each. In other words, for every outbreak in an individual herd or flock, 499 must remain disease-free. Clearly, this implies that the insurance company has evidence which says that the chances of any individual livestock enterprise suffering from disease in a year is 1/500 or 0.2%. So, for example, a larger scale livestock enterprise which, say, might be expected to suffer losses of $100,000 from outbreaks would pay a premium of $200 per annum if the premium was actuarially fair.

Any risk averse livestock producer will want to avoid the private costs of disease and will *always* choose to be completely insured if offered actuarially fair insurance. But a private insurance company has to pay its staff, cover fixed costs, and make a return on capital, and thus it must accrue more revenue on average than it pays out.[7] Therefore, in practice, insurance must necessarily be actuarially *unfair*. Now the sum of money any insurant is prepared to pay as a premium to guard against risk will depend on their degree of risk aversion. In general, the greater the sum any individual is prepared to pay to get rid of a risk, the greater that person's degree of risk aversion. Thus, even when confronted with actuarially unfair insurance, sufficiently risk averse people will take out a policy.

The relevance of these observations is as follows. In the context of epidemic livestock disease, the 'economic liberal' may argue that livestock producers should meet full responsibility for disease-induced losses for themselves. In other words, abolish state compensation payments and force livestock producers into covering themselves by insurance. Some less risk averse producers may indeed choose not to insure if a) they judge their premium to be excessive given b) their assessment (well-informed or otherwise) of the probability of outbreak on their holding and how much damage would be caused. The costs of a wrong decision could well be bankruptcy, but it is their choice to take the gamble if they so wish. However, the fewer insurers the lower the revenue accruing to the insurance company, and therefore the higher the premia charged to those livestock producers who do wish to insure. The outcome in terms of the proportion of farmers insuring will be determined by their degree of risk aversion, the premium, and so each individual producer's risk premium i.e. the sum they are prepared to pay to eliminate the risk.

For the insurers, the decision to become involved in insuring livestock producers must depend on the availability of reliable data on disease incidence, the private costs of outbreaks, and the willingness of farmers to take out insurance given the premia which are implied. Conceivably, insurance companies may find that getting involved with livestock disease insurance is more trouble than it is worth. It is well known that competitive insurance markets have not emerged to cover all areas of risk in the economy, and the main reasons for this are the problems of *moral hazard* and of *adverse selection*. It is worth looking at these two problems in greater detail to consider whether they would also constrain the development of insurance for losses from livestock disease.

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[7] In the UK the margin is typically about 30%.

Moral hazard: The problem of moral hazard arises when someone knows that they will be compensated for the effects of an unfavourable event, such as livestock disease, if and when it actually occurs, and as a consequence they are less vigilant about preventing that event happening. The magnitude of the moral hazard problem depends on the type of insurance. For example, it is negligible for life insurance but significant for motor insurance. In agriculture, moral hazard has been found to be an important limitation on crop insurance because, once insured, farmers have an incentive to put less effort into crop protection (Chambers, 1989). In the case of livestock disease, this implies that the risk of livestock disease could actually increase because farmers insure. In other words, insurance might have precisely the opposite effect to that anticipated by Davies i.e. instead of preventive measures being increased through insurance, they may actually decline.

One solution to the moral hazard problem is to introduce so-called *co-insurance* and *deductible* schemes. A familiar example of this is the 'driver excess' clause in vehicle insurance policies, i.e. of a total claim, a certain specified sum is met by the claimant before the greater residual part is met by the insurance company. Thus some of the risk is passed back to the insurant, who has an incentive to be more careful to avoid making claims at all. In other words, a proportion of the private costs of the occurrence of disease remains with the originator. The remainder is externalised in the sense that insurance payouts are generated from the premia paid by farmers who do not suffer the disease in the usual, direct sense, but exchange the penalty of compensating the afflicted (with their own insurance premium) for the right to claim compensation for themselves under similar circumstances. It would be expected that the higher the excess the greater the incentive for a producer to try to avoid disease. On the other hand, this sum must not be so high as to discourage insurance participation. An obvious solution to this problem is to make insurance compulsory. This has the additional potential benefit of reducing disease incidence if side conditions are also imposed, such as when preventive measures have to be implemented as a condition of the policy. Compulsion with side conditions is not as harsh as it sounds if insurants benefit by having premia reduced as a reward for implementing preventive measures.

Adverse selection: When insurers have limited information about their potential customers, and therefore charge the same premium to all, the adverse selection problem can arise. Suppose that an insurance company charges the same premium to all insurants. For those producers who are 'good risks' this will be an actuarially unfair premium, whereas those who are 'bad risks' are content to get cheap cover for being a relatively high risk. Unless the 'good risks' are sufficiently risk averse to go on insuring they will stop, leaving only the 'bad risks'. Again, this has been found to be a problem with crop insurance where farmers whose expected yields are typically low purchase insurance (Skees and Read, 1986). Since this bias is wholly to the disadvantage of insurers, the insurance market ceases to exist. Solutions to the adverse selection problem for the insurers are a) to try to discover the real risks of producers in different groups, b) to attempt to learn about relative risks over time by offering 'no-claim discounts', and c) to offer a variety of insurance contracts so that producers subject to different degrees of perceived risk choose the contract which best suits their individual circumstances i.e. self-selection. In the context of livestock disease, there is a clear role for epidemiological data to inform specification of insurance policies e.g. system of production, regional/local livestock population density, livestock marketing policy (contracts with processors or sales through auction markets), regularity of interventions by veterinarians, identification of risk factors in herds of different sizes, and so on. Scope for avoiding adverse selection will vary

according to the state of knowledge about specific epidemic livestock diseases, and that implies a key role for epidemiologists.

'Free riding': It should also be noted that problems may arise if some producers choose not to take out insurance. In fact they may be less likely to do so if they consider that expenditures by neighbours on measures for protection against disease reduces the risk to themselves without making any changes to their own practices. In other words, they would 'free ride' and gain the external benefits from the decisions of others. Potentially, therefore, there may remain geographical 'hot spots' of high disease risk from such farmers. Solutions to this would be to make insurance compulsory or for government to legislate to make observation of certain husbandry practices compulsory, with or without the obligation for farmers to insure as well.

Insurance and the public good

The primary, though not exclusive, focus of the above discussion was on insurance aimed at reducing risk for individual producers. With epidemic diseases there is also concern for limiting the external costs imposed on third parties. Why should insurance have the effect of providing incentives to individuals to protect not just themselves but also other people?

In principle, this wider objective can be achieved very simply by making producers legally liable for the costs they impose on others for the hazard created by their own actions. Precedents exist in the field of environmental law, with the result that insurance premiums are increased to meet higher expected payouts from disasters. In practice, problems can arise. Insurance companies have been reluctant to insure against environmental hazard because they have insufficient information about the extent of the risk they are taking on. There are two particular areas of uncertainty which confront them, one technical and the other legal.

Technical and legal uncertainty: Technical uncertainty arises from incomplete knowledge of the technical consequences of any event. In the case of a disease outbreak, the technical knowledge required is essentially epidemiological, and so the current state of epidemiological knowledge is critical if insurance companies are to consider extending policy to cover external costs as well as purely private costs. Legal uncertainty relates to the initially unknown chances of a producer being found liable for damages to others as a result of litigation. Clearly, the outcome in any specific instance of litigation will be influenced by the extent and reliability of technical information pertaining to the case. Thus legal and technical uncertainty are linked. Over time, legal uncertainty may be reduced by case law, but until then protracted legal wrangles might be expected. For example, there could be disputes as to the extent to which an individual is able and responsible for protecting themselves against the carelessness of others. The prospect of potentially expensive court cases might deter insurers offering policy cover where liability is unclear, except at prohibitively large premiums. Some clarification through government statute might reduce this problem, but the complexity of livestock disease could prevent a clear indisputable delineation of responsibility. Therefore it remains questionable whether the external costs can be internalised in this way.

Additional resource requirements: Nonetheless, insofar as there is scope for implementing measures to reduce disease incidence (feet dips, isolated animal houses, efficient heating and ventilation in building design, etc.), insurance companies have an interest in encouraging farmers to implement them, because the fewer and less serious the outbreaks, the lower the

payouts. Of course, this may mean additional expenditures by farmers seeking insurance because inadequate provision may cause them to be classified as 'bad risks'. To be a 'good risk' may involve sizeable capital investment on the farm before they can get insured. Also, insurance companies will need the capacity to police farm practice. Conceivably this may not be an excessive burden, because claims in the event of disease may be void if farmers are discovered not to have made suitable provision for minimising on-farm risk.

Private or public?: To this point it has been assumed that if insurance was promoted for compensation and prevention in relation to epidemic livestock disease, then it would be provided by the private sector. Attention has, however, also been drawn to potential constraints which might limit private insurance companies' interest. These constraints will be significant if there are serious problems of moral hazard, adverse selection and unclear legal liability. We now return to these potential obstacles and consider whether they are likely to be better addressed under a state insurance scheme rather than in the private sector.

In the case of moral hazard, it is difficult to see how a state insurance scheme would be in a better position to address the problem than a private scheme. Making producers bear some of the risk and insisting that certain preventive measures are applied seems the only feasible solution. Arguably, a system of public insurance may achieve some economies of scale in monitoring and enforcing preventive measures and, therefore, may have an advantage in combatting moral hazard. As for adverse selection, this could be overcome within a private scheme if insurance were made compulsory so the insurance companies would receive premia from the 'good risks' as well as the 'bad'. Of course this is unfair on the 'good risks' who would be over-insuring, although they might benefit from the existence of a comprehensively insured industry and any additional preventive measures which might result. In fact, it would be fairer on the 'good risk' producers to have a compulsory private scheme rather than a state scheme. At least then there would be competition in the provision of insurance, with companies seeking to differentiate risk between producers in an endeavour to capture the custom of the 'good risks'. However, even with compulsory insurance the potential problem of uncertain liability remains.

Since the necessary existence of actuarially unfair insurance in the private sector leads to underinsurance, it can be argued that there is a case for state implementation of an actuarially fair system. Thus full compensation would be given for all affected livestock, with the benefits of minimising the risk of diseased stock being off-loaded onto the market. This could be achieved by setting premia which, on average, cover payouts but not administration costs. These administration costs would have to be met by taxpayers. Whilst in one respect the risk of disease spread consequently would be reduced, at the same time full compensation would accentuate the moral hazard problem. A careful appraisal of the relative importance of these two contradictory effects would be necessary.

CONCLUSIONS

This paper examined the implications for economic efficiency of two approaches to financing the control and prevention of livestock epidemic disease - a headage tax (levy) on livestock, and insurance against the losses arising from livestock disease.

In principle, a headage levy would generate funds from within the livestock industry which could be earmarked for both compensating producers for disease-induced losses and for promoting better preventive measures. To the extent that the intensity of livestock production is a factor which increases the risk of disease, the levy would reduce total disease costs by penalising volume of production. Nevertheless, overall a levy provides little direct incentive for individual producers to increase preventive measures. Almost certainly, supplementary state regulations would be required to encourage measures for disease prevention except where producers were capable of making effectively costless managerial improvements. In other words, since the effect of the tax is to reduce the scale of production, efficiency gains would have to be sought to compensate for lost profits. Any improvements which required financial outlays, say in terms of fixed capital investment, would have to be made compulsory and, preferably, subject to state subsidy from tax revenues.

A private insurance market could develop if existing state compensation schemes were withdrawn. However, problems of moral hazard, adverse selection and uncertain liability may all help to obstruct the development of such a private market. Also, unless insurance was made compulsory some producers may choose not to insure. Even with compulsion there remains the possibility of underinsurance, because a private insurance company has to charge actuarially unfair premia to cover its administration and profit margins. On the other hand, private insurance should encourage better preventive measures to be taken by producers but only so long as the producers take out an insurance policy. There is still no guarantee that the optimal level of precaution will be achieved through private insurance. For all of these reasons, private insurance may provide imperfect incentives for epidemic disease control. A state insurance scheme might be the only feasible option if the market is unprofitable for private companies, because actuarially fair premia could be set so long as administration costs were subsidised. Such taxpayer costs would nevertheless be expected to be lower than existing costs of financing epidemic disease control because of a) producers' financial contribution through premia, and b) side-conditions of the insurance scheme intended to encourage better disease prevention.

A compulsory state insurance scheme could allow premia to be varied according to degrees of risk, with lower premia charged on those producers who take greater precautions. If premia truly reflected the expected damages from individual enterprises to others within the industry then, in principle, the socially optimal level of precaution could be achieved. But since the required information is likely to be incomplete, the best that can be achieved in practice is a move towards the optimum. There are also additional administrative and monitoring costs associated with premium differentiation, so the efficiency gains from differentiation need to be assessed in the light of these extra costs. Conceivably, a simple premium based on the number of livestock held might be the most pragmatic option, in which case the effects of the policy would be identical with the headage levy. Evidently this simplified approach would need to be complemented with state regulations to improve prevention, but not at a cost which makes society worse off in terms of net economic welfare.

The complexity of the relationships set out, even at the level of general principles, shows that the issues raised by Davies require most careful consideration. Most importantly, it is clear that principles can take us only so far. There are trade-offs to be considered at every stage, e.g. between scale of premia and number of participants insuring, the typical magnitude of private/social costs consequent on disease outbreaks, the revenues which need to be raised, and

so on. These are all fundamentally empirical questions, and there is considerable scope for veterinary epidemiologists and economists to combine their talents to investigate these issues in response to Davies' important lead.

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TOWARDS AN INTEGRATED DAIRY INDUSTRY DATABASE

D.F. KELTON and K.D. LISSEMORE[1]

Increased global competition in the dairy sector has put pressure on dairy farmers to produce milk more efficiently, while concurrently responding to concerns about animal welfare, food quality and safety, and the impact of animal agriculture on the environment. This in turn has stimulated an interest in better understanding the complex relationships between the biology of the dairy farm (milk component production, feed utilization, animal health, food product quality, etc.) and the financial performance of the farm enterprise (net dairy enterprise income per kilogram of fat quota shipped, milk income over feed costs, etc.). Applied, broad-based research to investigate these relationships requires access to data collected from many farm operations, over a long time period. Over the last ten years progress towards an integrated dairy industry database has been made in the province of Ontario, Canada. Many groups and individuals have played a role in this effort, and while considerable progress has been made, there is still much to be done.

WHY AN INTEGRATED DAIRY INDUSTRY DATABASE

As early as October 1981, at a national Symposium on Animal Disease Recording, members of the Canadian animal agriculture community recognized a need for an animal health information system. Such a system was envisaged to involve a partnership between farmers, farm organizations, veterinarians and government agencies, and should safeguard the health of our animal populations, and thereby ensure the high quality of the food that they produce. One approach to the development of such a system was through the establishment of a sentinel herd network to monitor animal health, a concept that has been under discussion in Ontario for some time. Such a network could be one key component of a larger, integrated national health surveillance system that is crucial to meeting Canada's responsibility as a member of the World Trade Organization (WTO). This disease surveillance initiative has recently been revived, driven in part by the new world trading order governed by the General Agreement on Tariffs and Trade (GATT) and the North American Free Trade Agreement (NAFTA), and was the focus of the Animal Disease Surveillance Symposium held in Ottawa in October, 1996. Recent experiences with Bovine Viral Diarrhea (BVD) in Ontario, and with Bovine Spongiform Encephalopathy (BSE) in the United Kingdom, were used to demonstrate the potentially catastrophic impact that animal health issues can have on trade in

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animal derived food products. During that symposium it became apparent that there is a need, now more than ever, to have an active animal health surveillance system in place, to maintain and expand our international trading position. It is also apparent that with budget reductions at all government levels, the support of such a system will have to be borne, to a large extent, by the animal commodity groups. The Ontario dairy industry is looking to make use of existing data gathering systems to fill at least part of the disease surveillance role.

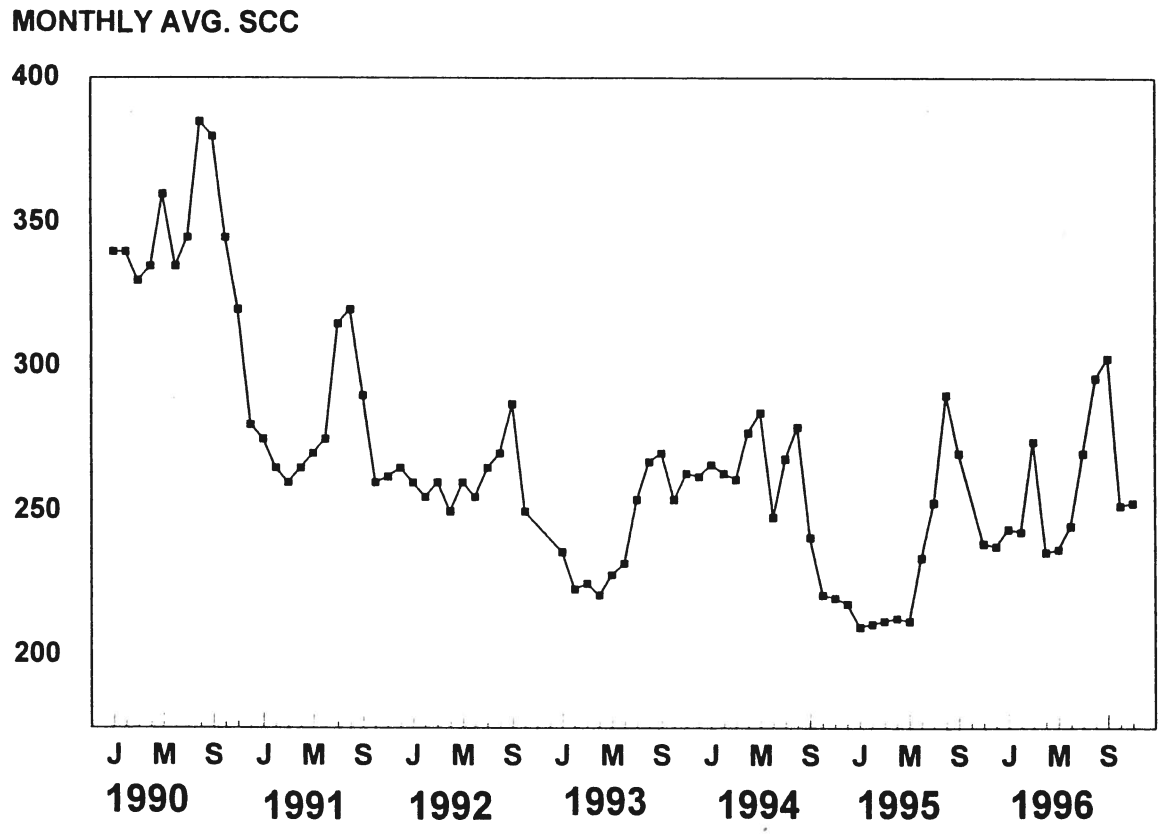
Falling milk prices and shrinking profit margins in the United States have stimulated an interest in the financial factors that drive dairy enterprise efficiency and profitability (Zweigbaum, 1989). In Ontario, perhaps largely because of the steady milk price guaranteed by our national dairy supply management system, the interest in dairy enterprise profitability has been slower to develop. Nonetheless, in anticipation of increased global competition in dairy production, mainly as a result of the NAFTA and GATT agreements, there is mounting interest in defining the characteristics of profitable Ontario dairy farms (Core, 1995; Nicholson, 1993). Attempts to identify health and production characteristics of "profitable and efficient" dairy operations have been frustrated by a lack of availability of comprehensive databases to investigate these issues. While large and significant dairy databases exist, they are very narrow in the scope of information that they contain. The data is collected by various dairy organizations (milk recording agencies, breed associations, artificial insemination cooperatives, government agencies, etc.) to serve the specific needs of their membership or constituents. These data are stored in various locations, are largely inaccessible for analysis, and are not easily linked. Increasing interest in collaboration among dairy agencies may facilitate the establishment of permanent linkages to move data among the existing systems, and make it more useful from an applied research perspective.

The research mission of the Department of Population Medicine at the University of Guelph is "to discover and disseminate knowledge regarding the management of health and productivity of animal populations, and the interrelationships of animals with humans and the environment." In support of this mission there is a strong reliance on expertise in field-based quantitative observational studies. However, on-farm data collection has always been a major challenge to mounting long-term field-based research efforts. Dairy producers, and their veterinarians, consider themselves to be data users, rather than data gatherers. Attempts to have producers use new, and additional, data capture systems has met with considerable resistance and eventually decreasing compliance. Exit questionnaires from major research projects have repeatedly emphasized the need to reduce duplicate collection of farm data by having one credible agency, such as the Ontario Dairy Herd Improvement (DHI) Corporation, as the primary data gatherer (Lissemore, 1992; Kelton, 1995). Considerable emphasis is currently being placed on working with industry partners to use existing data systems more efficiently, and thereby decrease the large expenses involved in establishing prospective data collection systems for relatively short-term research investigations.

Ontario prides itself in the production of milk and milk products of superior quality, and has had a provincial milk quality surveillance system in place for 28 years to support that claim. Bulk tank somatic cell counts (SCC) from every milk producer in the province are reported monthly. Concerns over elevated SCC's resulted in the implementation of the SCC penalty program by the Dairy Farmers of Ontario (DFO), formerly the Ontario Milk Marketing Board (OMMB), in August 1989. The SCC penalty program had a profound and

demonstrable effect on the provincial average bulk tank SCC (BTSCC) (Figure 1). The improvements made through the penalty program have been well documented (Schukken et al.,1992a; Schukken et al., 1992b). The steady decline in the provincial average BTSCC continued through 1994, and has been attributed to improved udder health through the implementation of control programs against mastitis caused by contagious organisms. However, the downward trend in BTSCC has not continued into 1995 and 1996. In fact, there is some evidence that the provincial BTSCC is on the rise again. There have been anecdotal reports that the increase in the provincial cell count is due to an increase in mastitis caused by environmental organisms, however there is currently no evidence to support this. There is a definite need to have a system in place to demonstrate the validity of such hypotheses, so that timely and appropriate actions can be taken to deal with the problems, if they can be clearly identified and defined.

Figure 1. Monthly mean bulk tank somatic cell count for the province of Ontario, from 1990 to 1996.



PROGRESS TOWARDS AN INTEGRATED DATABASE

Since 1989 we have been working with partners in the dairy industry and the provincial government to establish an integrated dairy industry database, utilizing existing data where possible, supplemented with limited additional on-farm data collection. Existing databases that have been identified as potentially important constituents of such an integrated system

include: milk quality and shipment data compiled monthly, from each of Ontario's approximately 8,000 licenced milk producers, by the Dairy Farmers of Ontario (DFO); milk and component production, somatic cell count and removal data collected by Ontario DHI, at the cow level, on approximately 5,500 of Ontario's 8,000 dairy farms (enrolment is optional); farm and dairy enterprise financial performance data collected annually from approximately 600 dairy farms by the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA), through the Ontario Farm Management Analysis Project (OFMAP) (enrolment is optional); breeding information for artificially inseminated dairy cattle served by one of two provincial bull studs; and cow and sire genetic evaluations for production and type, compiled semi-annually by the Canadian Dairy Network (CDN) and the Centre for Genetic Improvement of Livestock (CGIL). Electronic links between some of these existing dairy industry databases are currently being established, and mechanisms to facilitate the collection of additional data (specifically health data) on a broad basis are being investigated. The ultimate goal is to establish a national integrated dairy industry database, with cow and herd level data pertaining to production, health, genetics, reproduction, farm financial performance, and milk quality. By utilizing existing databases as much as possible, the database will have a foundation of many years of data (at least in several key areas). The database, once established, will be used to generate summary benchmark information for the dairy industry, to provide data for research into factors driving production efficiency and profitability, and to re-parameterize dairy herd/cow management models currently being developed in Canada and abroad.

A pilot project, using data collected from a purposive sample of approximately 110 Ontario dairy farms was conducted from 1990 to 1992. The objective was to investigate the relationships among herd level measures of management, productivity, health and profitability, in order to begin to define the financial and biological characteristics of profitable Ontario dairy farms (Kelton, 1995; Kelton and Martin, 1996). Existing production, SCC, removal, financial, milk quality and shipment data were collected through the cooperation of Ontario DHI, the DFO, and OMAFRA. Farm demographic and management information was supplied by the dairy producers, while additional information, particularly cow and heifer health, reproduction and herd management data were obtained through the ongoing collaboration of 27 veterinary practitioners. Of 107 dairy farms which completed the first year of the study, only 56 had complete financial, production and health information available. Figure 2 illustrates the range in profitability, as measured by *dairy enterprise net income per cow,* represented by this group of farms. Of 97 farms completing the second year of the study, only 39 had complete data. Analysis of the available data suggested that more profitable dairy farms produce more milk per cow, have lower feed costs per cow, and have better udder health. Unfortunately, due to the cross-sectional nature of the data, inference could not be made about the causal relationships between the biological factors and the farms' financial performance. The relatively short and finite study period, and the small number of farms that contributed data to both years of the study, may also have precluded the identification of important temporal relationships among these measures. It was concluded that future studies should include more farms, each contributing data over a much longer period of time, in order to better elucidate the impact of changes in health, management and productivity on measures of dairy farm profitability and production efficiency.

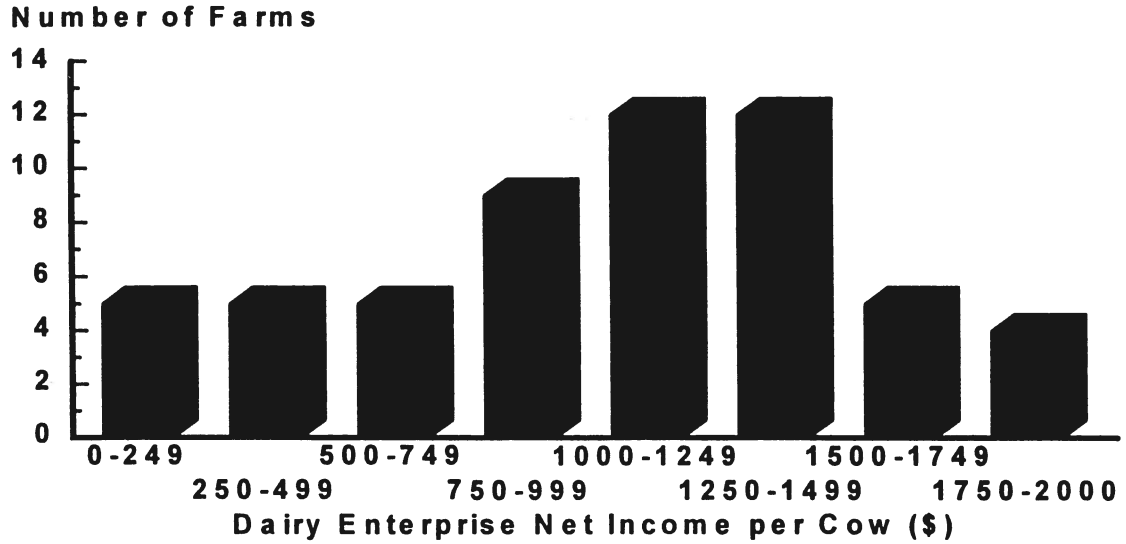


Figure 2. Distribution of dairy enterprise net income per cow (in Canadian dollars) for the 56 Ontario dairy farms which had complete financial data available for year 1 (1990).

The success of a broad-based initiative such as an integrated industry database depends on strong support from dairy producers and their representative organizations. In 1989, the First Ontario Dairy Producer Organizations Conference was held to address topics of concern and interest to the province's dairy producers. Out of that initial conference, two industry-wide committees were formed. One committee was mandated to address the coordination of dairy extension activities across the province, while the other was established to determine how best to assess cow and herd profitability in Ontario. At the Fourth Ontario Dairy Producer Organizations Conference, held in February 1992, a recommendation was made jointly by the chairs, on behalf of the two committees, that an integrated dairy industry database be established. The expressed desire for such a system stemmed from the realization that all aspects of production, management, health and finances must be evaluated together to get a true insight into the overall profitability of the Ontario dairy enterprise. Given the expression of interest and the spirit of cooperation that existed among the dairy industry partners in the province, there was perceived to be an opportunity to develop a system that will enable the creation of an integrated dairy industry database, containing both herd level and individual animal data, through the electronic collection of data already existing and in the possession of industry partners. The system needed to be flexible enough to allow later addition of data from additional herds, data from new sources (such as on-farm computer systems) and new variables which may become of interest to the industry. The system was to be utilized to produce annual dairy industry summary information, which would serve as the basis for dairy industry health and productivity benchmarks. In addition, the database would be used to identify the major determinants of profitability for Ontario dairy farms and to monitor changes in those determinants. This program would encourage the efficient use of existing resources by using an integrated long-term approach to data gathering and use. By having

this system in place, the lag time between issue identification and the commencement of data gathering would be markedly decreased. Funding for this initiative has become a high priority within OMAFRA's research support contract with the University of Guelph. This has made resources available to begin to implement this initiative.

A major challenge to the establishment of the integrated database has been the lack of uniformity among systems used to collect animal health and farm financial information. Previous work in the Ontario swine industry has emphasized the importance of reliable financial data in farm level economic analyses (Deen, 1993). The financial data incorporated into a dairy industry database must be collected in a uniform and consistent manner. Initially, farm financial data is being collected through the Ontario Farm Management Analysis Project (OFMAP) of the Ontario Ministry of Agriculture, Food and Rural Affairs. This program has been in operation since 1946 and uses standardized methodologies and definitions consistent with published industry recommendations (Farm Accounting Standardization Manual, 1991; Financial Guidelines for Agricultural Producers, 1993). Currently, efforts are underway to augment the annual financial information generated by OFMAP with monthly cost of production information, using the Ontario DHI field service staff.

The validity of the inferences drawn from any database depends on many issues, not least the quality of the data collected. This applies particularly to health data. These data are needed to further investigate the genetic component of disease occurrence and resistance, to describe, compare and investigate disease occurrence on a national and regional basis, to modify management practices that promote animal health, and to monitor the health status of the national dairy herd. A major impediment to this initiative is the lack of national standards for disease definition and presentation. Use of disparate disease definitions makes it difficult to pool or compare disease rates from various regions of the country. A recent review of published reports of the incidence of common peri-parturient diseases demonstrated considerable variability in the eight conditions considered (Figure 3). Obvious reasons for the differences included a lack of uniformity in case definition, and differences in the calculation and interpretation of incidence rates.

Steps are being taken to establish standards for the definition and reporting of economically and biologically important diseases of dairy cattle. A report is currently being prepared in response to the Canadian dairy industry's increased interest in the recording of clinical disease data. The major objective is to introduce guidelines and national standards for dairy cattle disease recording and presentation. Eight clinically identifiable diseases of economic importance to the Canadian dairy industry have been identified. These diseases include milk fever, retained placenta, metritis, ketosis, left displaced abomasum, cystic ovarian disease, lameness and mastitis. Standardized definitions for these diseases have been created through consultation with industry partners. Since some of these conditions do not necessarily require veterinary intervention, the definitions are based primarily on clinically apparent signs, which will allow the dairy producer and/or the veterinarian to make the appropriate diagnosis. Used in a broad based data recording scheme, this will facilitate the uniform capture of most, if not all, clinically relevant events.

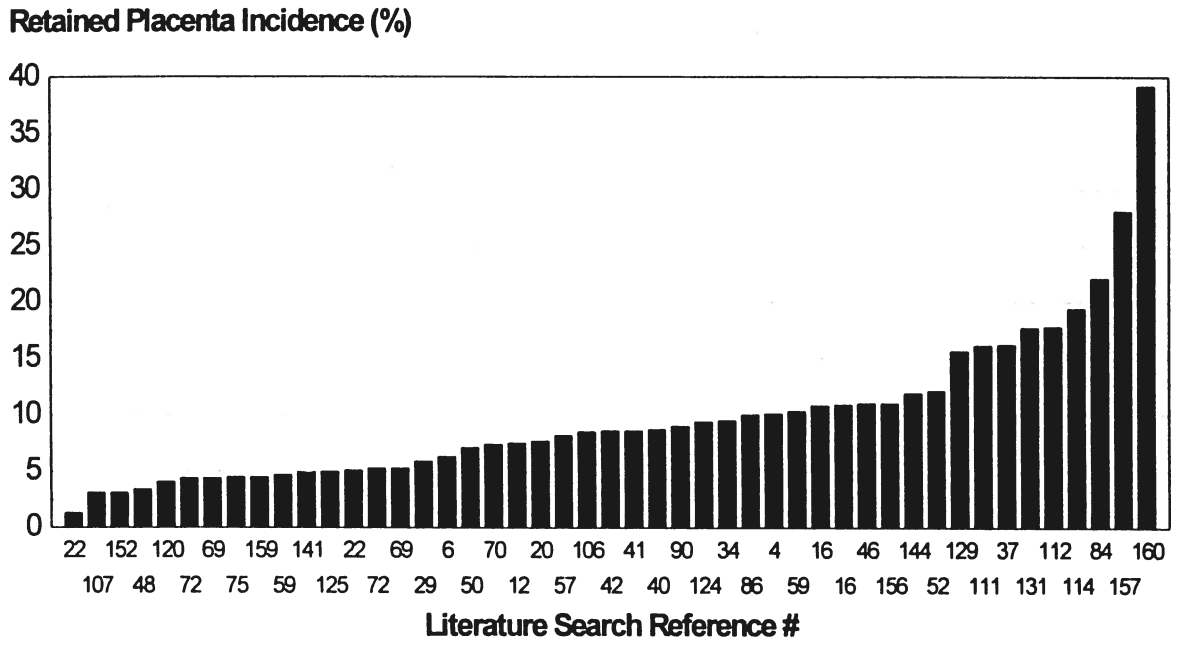


Figure 3. Distribution in lactational incidence of retained placenta as determined through a search of the veterinary literature from 1979 to 1995.

Disease frequency is usually reported either as incidence (the rate of occurrence of new cases of disease per unit of time) or prevalence (the proportion of animals that are diseased at any single point in time). All of the diseases described in the commissioned report have been summarized and reported extensively. In most cases, the measure of disease frequency has been some form of incidence. However, there is considerable variation in the reported incidence for each disease. This variability is likely due to geographical, management and breed differences in the populations of dairy cows/herds under study and in the manner in which incidence was calculated. For example, many studies report **incidence** calculated as a true rate, *cases per cow-year at risk*, while others report **lactational incidence** calculated as a risk (cumulative incidence) rate, *affected lactations per 100 lactations at risk.*

For each disease described in the commissioned report, two approaches to summarization and reporting have been proposed. The guidelines for **retrospective analysis** should be used when historical data is being analyzed for purposes such as genetic evaluation. In such a context, the data being used would include all lactations completed (lasting 305 days or more) or terminated (lasting less than 305 days but terminated by death or culling). In most instances, the recommendation is to report *lactational incidence* as a *risk (cumulative incidence) rate*, presented as *percent of affected lactations* (ie. affected lactations per 100 lactations at risk). **Current analysis** is likely to be used for *status monitoring* (comparing

current performance parameters against herd or group goals) and *trend monitoring* (tracking changes in a parameter over time) (Fetrow, 1994). The calculation should account for all events, and all animals at risk, for a recent period of time, usually 1 to 3 months. In most cases the recommendation is to report *incidence* as a *true rate*, presenting *cases per cow-time at risk*, in order to account for additions to and removals from the population under scrutiny.

PLANS FOR THE FUTURE

The Canadian milk recording system is rapidly changing. Over the last two years, the number of regional DHI services has decreased from seven to four, and the number of dairy record processing centres (DRPC) has decreased from four to two. Concurrently, the remaining DHI services have become aggressive in offering a wider variety of services to their producer clients. Beyond the traditional recording of milk and component production, and SCC's, these agencies are offering cost of production services and expanded data collection and storage through the adoption and marketing of a commercial herd management software computer program. The impending changes to the data gathering and transfer systems utilized by Ontario DHI and Promark Technologies (the DRPC serving the western half of Canada) offer a unique opportunity to easily augment the type of data being collected and stored. By implementing a versatile commercial herd management software package, DairyComp 305[2] (DC305), Ontario DHI, Western Canadian DHI Services and British Columbia DHI Services will be able to broaden the types of data that they collect and store. Of particular interest will be the ability to collect and store animal health information, including details pertaining to peri-parturient, metabolic and clinical mastitis events. The use of this existing system will facilitate the inclusion of disease data in the integrated dairy industry database without the necessity of implementing an additional data gathering system.

Ontario's dairy industry is among the largest in Canada, and is looking to provide leadership in the growth and sustainability of dairy production in Canada. As the next step in the movement towards an integrated dairy database, a proposal has been drafted for the establishment of a sentinel herd network within the dairy industry, with the purpose of maintaining the health of our provincial dairy herd, and further enhancing the quality of the milk leaving our dairy farms. Benefits to be derived from such a system include continued consumer confidence in the safety of milk and milk products, establishing a pre-eminent national and international quality status for our milk products to maintain domestic markets and expand into new national and international markets as they become available, and to provide our animal health professionals with current, useful information to maintain the health of our dairy cattle population. The major stakeholders in this initiative are the Ontario dairy producers, and the agricultural communities in which they live. The major benefit will be long-term sustainability of jobs through improvements in animal health, milk quality, consumer confidence and export market potential.

Utilizing the expanded data collection opportunities presented by the implementation of DC305, the goal will be to establish a network of sentinel herds and veterinarians for the

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[2]Valley Agricultural Software, 2861 South "K" Street, Tulare, California, USA, 93274.

routine collection of health and production data. These herds will be identified through dairy veterinary practitioners and would likely have the following characteristics: a herd size of 50 or more adult dairy cows; predominantly free-stall housing; enrolled in official milk recording through Ontario DHI; predominantly registered purebred cattle; willing to share farm financial data; and be willing project participants / cooperators. The goal will be to identify approximately 50 herds across Ontario. This sentinel herd network will allow the industry to monitor changes in animal health, to formulate responses to these changes and to measure the impact that such changes have had. Furthermore, this will serve as a model for the development of one component of an active animal health surveillance system that is crucial to Ontario's active participation in world trade. Data will also be available to quantify, and continue to update, the economic impact (direct and indirect costs) of clinical and subclinical disease events on the profitability of Ontario dairy farms. This will allow producers to increase farm efficiency through a better understanding of the true economic impact that disease has on their dairy enterprise.

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ADAPTING PARTICIPATORY APPRAISAL (PA) FOR THE VETERINARY EPIDEMIOLOGIST: PA TOOLS FOR USE IN LIVESTOCK DISEASE DATA COLLECTION

A.P. CATLEY*

In the early 1970s it was recognised that formal systems of inquiry were of limited value when working with rural communities in developing countries. Over-use of questionnaire surveys, "rural development tourism" and poor cost-effectiveness were identified as some of the key problems with formal methods of data collection, particularly questionnaire surveys (Chambers, 1983). In response to these problems, a system called Rapid Rural Appraisal (RRA) was developed in the 1980s (McCracken et al., 1988). Rather than attempting to collect quantitative data on problems identified by researchers, RRA focused on farmers' perceptions of priority problems and was chararacterised by a reliance on qualitative data and avoidance of statistical analysis.

In the late 1980s RRA evolved into Participatory Rural Appraisal (PRA). PRA facilitated the participation of communities in the analysis and solving of problems, and encouraged project beneficiaries to plan and take action. Definitions and levels of "participation" within the context of sustainable agricultural development are discussed by Pretty (1994), with self-mobilisation representing the ultimate level of participation. RRA and PRA now form part of a family of approaches including Participatory Learning and Action (PLA), Rapid Assessments Procedures (RAP) and Rapid Rural Systems Analysis (RRSA). As a key feature of all these systems is participation, this paper uses the term "Participatory Appraisal" which relates to data collection tools which might be used in any of the above systems.

Participatory methods are now widely used by development projects in both rural and urban areas of the Third World. Recent work includes efforts to combine participatory and formal approaches (Turton et al., 1996) and quantification of data generated by PRA tools for statistical analysis (de Villiers, 1996). In developed countries there is also interest in participatory approaches as exemplified by the use of RRA in forestry programmes (Inglis and Lussignea, 1995) and human health work in Scotland (Murray et al., 1994).

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Initial interest in participatory approaches in the livestock sector included a review of informal survey methods in relation to community participation (Leyland, 1991) and later, a description of rapid appraisal methodologies (Ghirotti, 1993). During the early 1990s PA was widely used by community-based animal health projects (Kirsopp-Reed, 1994) and pastoral development projects (Waters-Bayer and Bayer, 1994). These projects had close links with project beneficiaries and could demonstrate understanding of local veterinary knowledge, skills and perceptions, often called "ethnoveterinary medicine". In contrast, the use of PA in bilateral or multilateral-funded veterinary aid programmes and government veterinary services has been limited. The reluctance to use participatory data collection methods is often related to claims that PA is not "scientific" and does not produce data which can be analysed statistically.

Formal methods such as questionnaire surveys are frequently used by government services and researchers for collecting data from livestock owners.Work in Zambia (McCauley et al., 1983), Sudan (Perry et al., 1984) and Afghanistan (Schreuder et al., 1996) indicated that although livestock owners could provide useful information on disease incidence, mortality or production losses, the questionaire methodology was time-consuming and based on the researchers' priorities rather than those of the respondents. Attention to enumerator bias was limited in these studies.

PA has been used to good effect by community-based animal health projects and consequently, there is now potential for incorporating PA methods into conventional animal health data collection systems. This paper aims to outline PA tools which are relevant to veterinary epidemiology and discusses the role of qualitative data in formal surveys. Methodologies and results of two PA tools are described in order to show how the tools might be adapted in order to yield quantitative data for statististical analysis.

PA TRAINING FOR VETERINARY EPIDEMIOLOGISTS

The PA tools outlined below include tools which can be modified by veterinary epidemiologists to yield numerical data. However, before readers attempt to adapt or standardise PA tools, they should gain practical experience of participatory methods and essentially, receive training in PA. A typical PA training course would include the following topics:

- Introduction to the background, development and approach of PA.
- Attitudinal aspects of PA.
- Understanding bias and rural development tourism.
- Communication skills such as listening skills, non-verbal communication and appropriate behaviour.
- Identification of social groups and informants, including key informants.
- Managing group interviews including group dynamics and dominant talkers.
- Use of secondary data sources and direct field observation.
- Interviewing skills, particularly semi-structured interviews and use of open rather than closed questions.

PA TOOLS

PA collects information using a "toolkit" comprising diagramming, mapping, scoring, ranking and interviewing tools. Some PA tools which have been used by veterinarians in developing countries are shown in Table 1. In a typical PA survey, the combination of tools allows cross-checking or "triangulation" of results while researchers are still in the field. Results are also cross-checked by working with both men and women, and using informants of varying experience, skills, age, social status or wealth . When investigating subjects such as livestock disease, local "experts" can be identified who are respected by their communities for possessing specialist knowledge. PA calls these experts "key informants".

Table 1. Some PA tools for use in veterinary epidemiology and economics

Information required	PA tools and methods[a]
System boundary	Natural resource maps
Social organisation	Social mapping, venn diagram
Wealth groups	Wealth ranking
Relative livestock ownership	Proportional piling
Role of livestock in household economy	Livelihood analysis
Preferred types of livestock reared	Livestock species scoring
Income from livestock	Proportional piling
Marketing structure	Flow diagrams, service maps
Veterinary services	Service map, venn diagrams
Animal husbandry	Seasonal calendars[b], mobility maps[c]
Resources available to livestock	Natural resource maps
History of livestock diseases	Timelines
Priority livestock diseases	Livestock disease scoring
Seasonal variations in livestock disease	Seasonal calendars
Relative mortality rates	Proportional piling
Livestock productivity	Progeny histories, seasonal calendars

[a]Semi-structured interviews can provide information on all topics
[b]Particularly useful for showing breeding management and feeding management
[c]To show livestock movements in pastoral and agropastoral systems

To date, most PA tools have been used to produce qualitative not quantitative data and PA survey results are presented in a descriptive (e.g. interviews, direct observation, diagrams) rather than numerical (e.g. tables, graphs, statistics) form. However, virtually any qualitative data can be transformed into numbers. If this transformation occurs at an early stage in the data collection process, descriptive information is summarised in numerical form and subsequent analysis is governed by statistical rules (Moris and Copestake,1993). In the PA toolkit, some tools require informants to score items or illustrate proportional relationships between items. These tools produce numberical data at an early stage in the methodology.

Scoring tools

In livestock surveys, scoring tools can be used to compare and prioritise items such as livestock species or livestock diseases. The tools provide information on preferences and encourage informants to not only state opinions, but also explain the reasoning behind the decisions they make.

Method: Scoring tools involve three main stages. The methodology outlined below is described in much greater detail in account of a tool called livestock-disease scoring (Catley and Mohammed, 1996).

Stage 1 - Identification of items to be scored. Ask the informants to name the items under investigation. For example, if investigating preferences for species of livestock reared ask the question, "Which types of livestock do you keep?". If investigating animal health problems, the researcher can limit the number of items by questions such as "What are the six most important livestock diseases in your animals throughout the year?" Write the items named by the informants on to separate pieces of card using the local language. Check that at least one informant is literate. If all informants are illiterate use different objects to represent each named item e.g. when investigating livestock species a stone could represent a cow, a leaf could represent a goat, and so on.

Stage 2 - Pair-wise comparison of the named items. First, choose two items (represented as name cards or objects) and ask the question "Which of these two is most important and why?" The informants will prioritise the items and provide reasons for their decision. Record the response and repeat the question until each item has been compared with every other item. At the end of the pair-wise comparison the researchers should have recorded a list of "indicators" or factors used by the informants to compare the different items.

Stage 3 - Scoring of items verses indicators. Place the name cards or objects in a row on the ground. Collect a pile of stones using 5 stones per item as a guide to the number of stones needed e.g. if 6 items are being scored, 30 stones are required. Remind the informants of the first indicator mentioned during the pair-wise comparison; ask them to distribute the stones according to degree of relationship between this indicator and each of the items represented by the name cards or objects. All stones must be used. After the stones have been allocated to each item, check the scoring with the informants and allow them to alter the scoring if they wish. Record the final number of stones allocated to each item, collect the stones and then repeat the scoring for each of the indicators. In the example below, the indicator "resistant to drought" has been scored by Somali herders for 5 livestock species (see Fig.1 for full results).

Examples of results: Figure 1 shows how livestock species were scored by a group of three herders in northern Somalia. These informants mentioned 42 indicators during the pair-wise comparison of 5 livestock species and each indicator was scored using 25 stones. The indicators included both positive and negative attributes of livestock. Livestock species scoring is best conducted at an early stage in a survey as local perceptions of the role and relative importance of livestock types can assist researchers to cross-check information on animal health problems.

Figure 2 shows the results of a livestock-disease scoring which was based on the question "What were the 6 most important livestock diseases in your area during the last year?" The informants were a group of 5 herders in northern Somalia and each indicator was scored using 30 stones. The results include local perceptions of disease causality, epidemiology, production losses and economics. Seasonal bias of the results can be cross-checked using seasonal calendars (see later).

Variations in the method: Like most PA tools, scoring tools are flexible and can adapted by the researchers to suit particular needs. Obvious variations include altering the number of items to be scored, altering the number of stones used, or adding the researchers' own indicators to those of the informants. An overall score for each item can be obtained from the informants or by adding all the scores for each item. If the latter method is used, the scores of indicators which reflect problems or unfavourable aspects of a particular item should be given a negative value. For example, in Fig.1 indicators 7, 11, 14-16, 22-24, 27, 33-36 and 39 would be recorded as negative values if the scores for each livestock type were summed.

Standardisation of scoring tools: Scoring tools might be standardised by defining the number of items to be scored and the number of stones to be used for the scoring of indicators. Specific indicators could be defined by the researchers and added to those produced by the informants. Replication of a standardised scoring tool would allow statistical analysis of results using using tests for non-parametric data such as Kruskal-Wallis followed by Dunns Multiple Comparisons Test.

Seasonal calendars

Seasonal calendars are diagrams constructed by informants which illustrate seasonal variations of events under investigation. A number of different events can shown on a single diagram e.g. livestock breeding management, incidence of important livestock diseases, feeding management and livestock sales. If a survey is focused on animal health problems, a seasonal calendar showing the incidence of livestock diseases is useful for cross-checking the results of livestock-disease scoring.

Method: In order to use seasonal calendars the researchers should understand and use local descriptions of seasons and months.

Stage 1 - Draw a horizontal line on the ground to represent 1 year. The line should be at least 1 metre in length. Divide the line according to local definitions of month and season.

Stage 2 - It is useful (though not essential) to choose rainfall as the first event to be

Figure 1 **Example of livestock species scoring, northern Somalia**
(Source: Ahmed Aden and Catley, 1993)

Indicators provided by informants	camel	sheep	goat	cattle	donkey
1. Resistant to drought	11	6	4	1	3
2. Can travel long distances	15	2	3	2	3
3. Availability of milk	13	2	4	6	0
4. Breeding not controlled	3	3	13	3	3
5. Use for blood money compensation	25	0	0	0	0
6. Paying dowry	14	4	2	5	0
7. Difficult for women to look after	9	3	3	9	1
8. Only men can benefit	12	6	2	4	1
9. Easy to sell	2	16	4	3	0
10.Convenient food for hospitality	0	17	8	0	0
11.Used to assist newly married man	0	16	9	0	0
12.Rapid growth (to maturity)	2	7	11	5	0
13.Quality of milk (taste)	9	2	6	8	0
14.Susceptibility to disease	1	8	2	7	7
15.Frequency of watering needed	1	2	4	11	7
16.Susceptibility to cold	1	2	13	7	2
17.Sale value	10	4	1	7	3
18.Animal power (for ploughing)	3	0	0	15	7
19.Ghee production	0	3	7	15	0
20.Use of hides and skins	6	3	2	14	0
21.Use as burden animal	18	0	0	0	7
22.Cause of disputes (between herders)	14	3	1	7	0
23.Ownership is a security risk	20	1	0	4	0
24.Bad effect on herder literacy	14	2	8	1	0
25.Good effect on religious life	1	18	3	3	0
26.Requires forest area	11	0	10	4	0
27.Bad affect on marriage prospects	13	1	4	7	0
28.Quantity of meat	12	2	4	7	0
29.Quality of meat (taste)	5	5	13	2	0
30."Meat keeps us full"	15	3	1	6	0
31.Used for earning additional income	10	0	0	9	6
32.Used for domestic tasks	8	0	0	0	17
33.Noisy	2	0	0	0	23
34.Produces bad dung	0	0	0	0	25
35.Dirty/unclean animal	0	0	0	0	25
36.Cause of injury to herder	5	0	5	10	5
37.Amount of fat	5	12	2	6	0
38.Able to live on open plains	9	14	0	0	2
39.Special timing of watering needed	3	3	9	8	3
40.Overall score for meat	7	10	5	3	0
41.Nutritional value of milk	10	3	4	8	0
42.Overall score for milk	14	1	3	7	0

Figure 2 **Example of livestock-disease scoring, northern Somalia** (Source: Catley and Mohammed, 1996)

Indicators provided by informants	**DISEASES** **Nairobi sheep disease**	**respiratory disease in camels**	**intestinal helminthiasis all species**	**surra**	**ulcerative balanoposthitis in sheep**	**camel pox, sheep and goat pox**
Importance indicators						
reduced local sale value	0	4	4	5	7	10
reduced export sale value	0	0	0	0	11	19
disease causes poverty	19	0	3	0	0	8
disease causes death	15	0	6	0	0	9
disease causes recumbency	7	0	23	0	0	0
disease causes emaciation	0	0	17	13	0	0
disease causes abortion	0	24	0	0	0	6
disease damages skin	0	0	0	0	0	30
disease is spread by ticks	30	0	0	0	0	0
disease affects different species	0	0	0	0	0	30
disease reduces milk yield	0	12	6	12	0	0
disease makes meat inedible	4	0	8	0	0	18
disease cannot be treated	0	0	0	15	0	15
overall importance scoring	9	0	5	3	3	10
Difference indicators						
disease occurs in hot weather	10	0	0	0	0	20
disease is contagious	0	11	0	2	4	13
disease is spread by worms	0	0	30	0	0	0
disease affects mainly sheep	22	0	0	0	8	0
disease causes subcut. oedema	0	0	16	0	6	8
disease causes diarrhoea	8	0	22	0	0	0
disease causes bloody diarrhoea	9	0	21	0	0	0
disease causes coughing	11	19	0	0	0	0
reduced breeding potential	0	0	0	0	30	0
black lymph nodes, post mortem	21	0	0	0	0	9
thin watery blood, post mortem	0	6	6	11	0	7
congested meat, post mortem	15	0	15	0	0	0
can vaccinate against disease	0	0	0	0	0	30

illustrated on the calendar. Take a stick of around 30cm in length and explain to the informants that the stick represents the month which receives the most rain in a year. Ask the informants to place the stick against the month which receives the most rain.

Stage 3 - Take a second stick of around 30cm in length. Explain to the informants that the stick represents the month which receives the second most rain in a year. Ask them to break the stick according to the amount of rain received in the second wettest month, and place the stick against the appropriate month. At this stage the informants will often compare the length of the second stick with the first, and break the second stick accordingly. Repeat this procedure until rainfall throughout the year has been illustrated using sticks. An alternative method uses piles or rows of stones to illustrate rainfall.

Stage 4 - Ask the informants to illustrate on the diagram the occurrence of the events under investigation. Events might be the livestock diseases identified during a livestock-disease scoring. The informants can simply draw on the ground to show the events or use sticks, stones or other natural materials.

Examples of results: Examples of seasonal calendars are shown in Figure 3 and Figure 4. In Fig.3, stones were used by informants to show rainfall, livestock births, disease incidence and livestock sales. The diagram was constructed by a group of 11 pastoralists in northern Somalia. Figure 4 shows seasonal tick infestation of livestock as perceived by informants in one area in northern Somalia. Sticks have been used to illustrate rainfall and piles of stones indicate seasonal variations in infestation by different types of ticks. In this example, the number of stones used was not specified by the researchers.

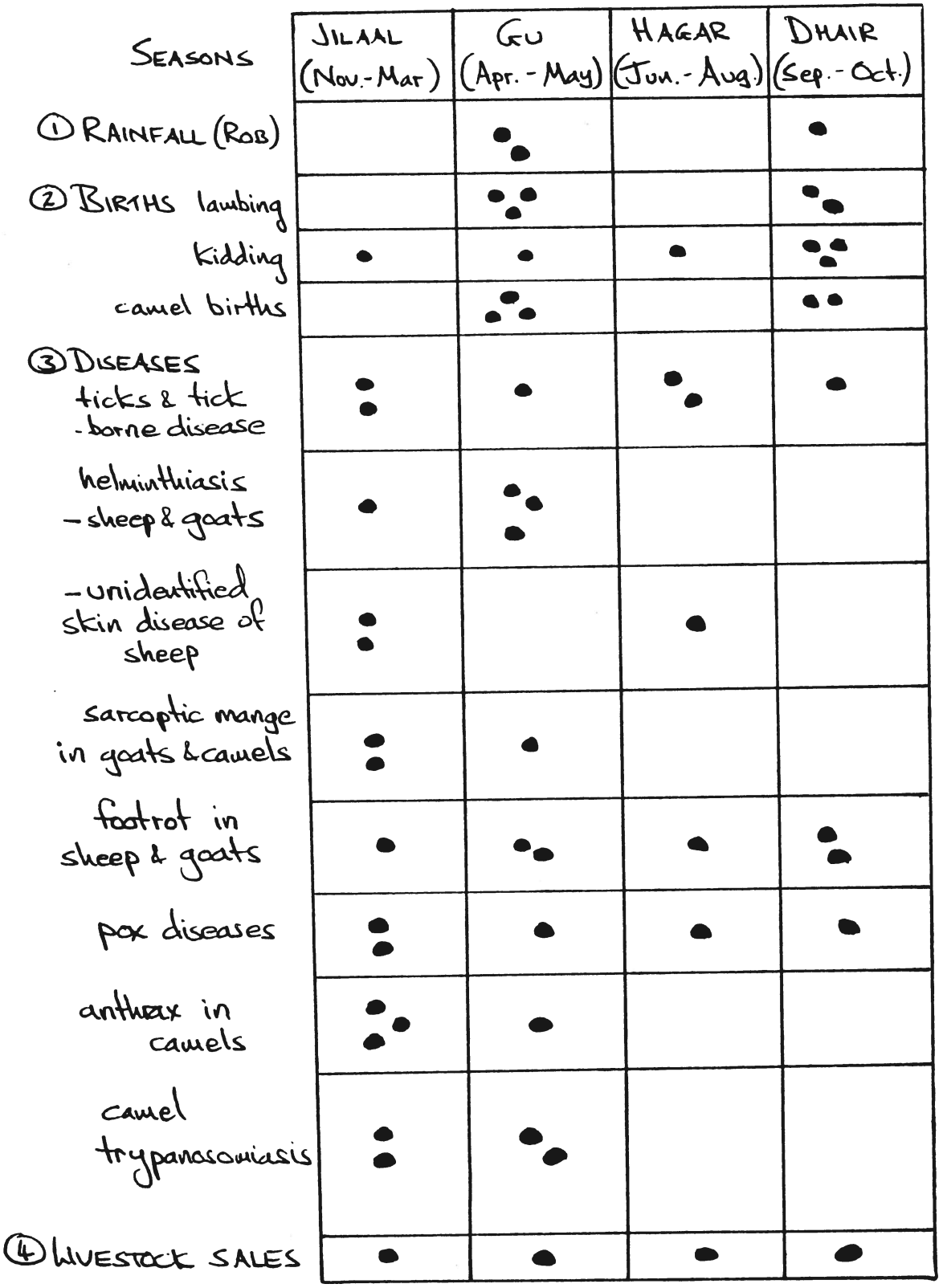
Standardisation of seasonal calendars: Seasonal calendars which use piles of stones to show the timing of events are similar to scoring tools and therefore some standardisation is possible. Note that the low number of stones (maximum 3 stones per item) used in Fig.3 may have limited the sensitivity of the tool because only scores or ranks of 0,1,2, or 3 were possible. In Fig.4, up to 18 stones per item (type of tick) were used. If the number of stones and seasons was fixed, results from repeated seasonal calendars could be analysed using Kruskal-Wallis tests and Dunns Multiple Comparison Test.

When sticks are used to show rainfall a "qualitative bar chart" such as that shown in Fig.4. can result. This diagram indicates trends in rainfall and was copied on to paper by measuring the sticks and producing a scaled drawing. Initially, local names and definitions of months and seasons should be used but later these can be altered to the Gregorian calendar as necessary. The events which are scored according to season can be defined and standardised by the researchers.

CONCLUSIONS

The use of PA is increasing in a range of sectors in both the developing and developed world. PA methods continue to evolve, including specialised PA tools which are adapted to meet the needs of particular disciplines. Regarding surveys in developing countries which rely on data derived from livestock owners, PA has numerous advantages over

Figure 3 **Seasonal calendar showing rainfall, livestock births, disease incidence and livestock sales, northern Somalia.**
(Source: Hadrill and Yusuf, 1994)



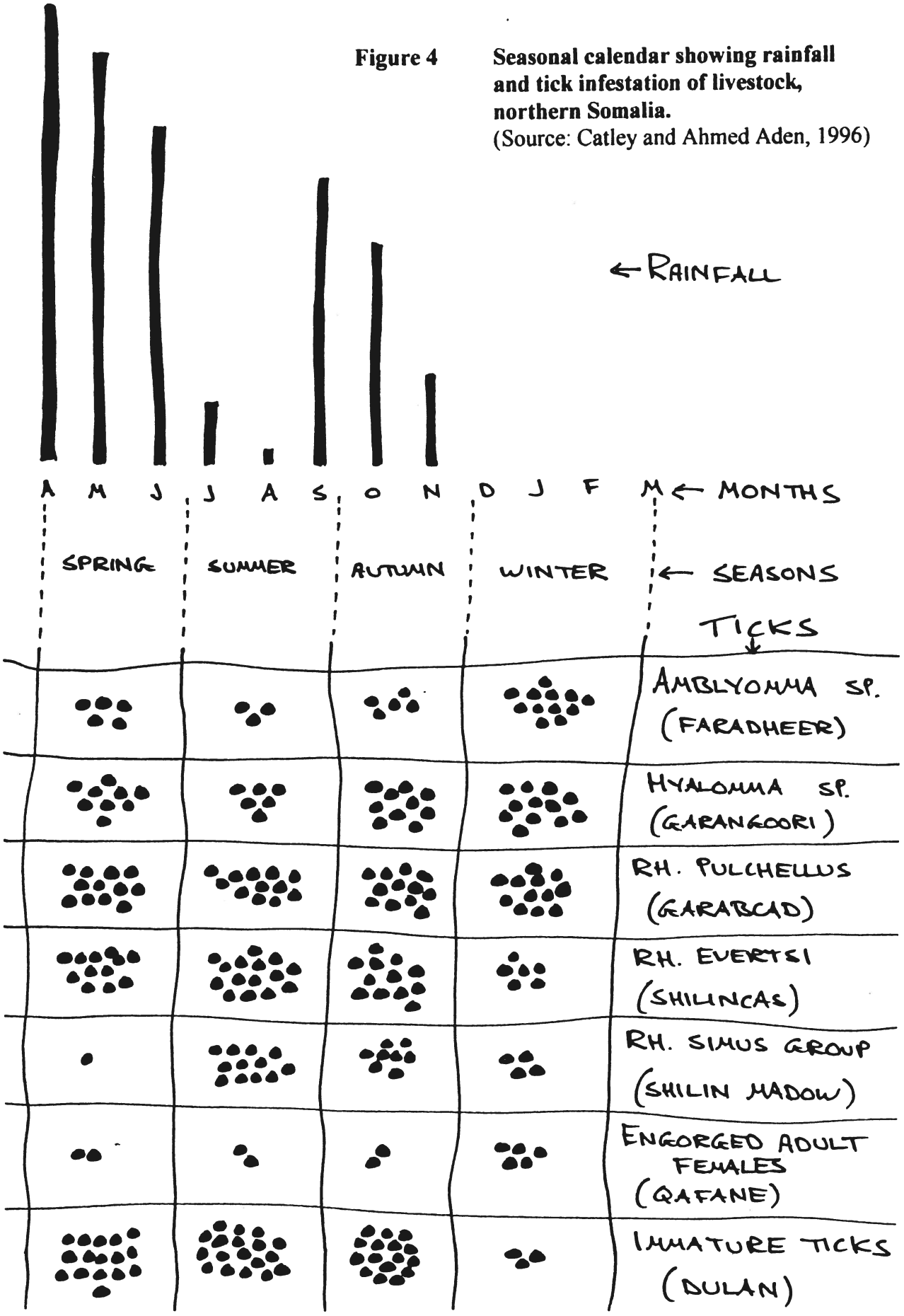


Figure 4 **Seasonal calendar showing rainfall and tick infestation of livestock, northern Somalia.**
(Source: Catley and Ahmed Aden, 1996)

questionnaires. The design of conventional questionnaire formats and interview protocols can be a lengthy and difficult process (Putt et al., 1988) whereas PA tools involve simple checklists of key words. PA tools are flexible and can be modified in the field, and triangulation allows cross-checking of results at the research site. In terms of quality of data, perhaps the most important aspect of PA is that it creates good rapport and trust between interviewers and informants. Unlike questionnaire surveys during which informants may be either suspicious or bored, livestock surveys using PA tend to be lively and enjoyable events which rely on livestock owners actively teaching outsiders about local problems and practices. The relative costs of questionnaire and PA surveys have not yet been determined.

Within the field of veterinary epidemiology and economics there are opportunities for using PA methods, particularly in developing countries. Although unmodified PA tools generate qualitative data, this data can compliment more formal systems of inquiry and may be acquired with limited resources. The use of standardised PA tools which produce quantitative data is another option for veterinary epidemiologists, although training in general PA approaches and methods is recommended before existing tools are modified. This paper provides a very brief overview of PA and presents results of PA tools from only one country. For further information on participatory methods, readers are advised to consult literature produced by the Sustainable Agriculture Programme of the International Institute for Environment and Development, London.

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RELATIONSHIP BETWEEN INDIVIDUAL SOMATIC CELL COUNT AND BACTERIOLOGY: A DETAILED STUDY ON A LOW BULK MILK SOMATIC CELL COUNT FARM

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Bulk milk somatic cell count is world wide used as a tool to monitor the mastitis status of a farm. Therefore, and because the somatic cell count is relatively easy to measure, measurements of individual cow somatic cell counts (CSCC) have become common practice on many dairy farms. Cows with a new or chronic infection can thus be detected (Reneau, 1990). Many factors are associated with an increase in CSCC (Leslie et al., 1983; Reneau, 1990). A large variation in CSCC between cows and even within cows exists (Schepers et al., 1997). Various authors suggest the use of CSCC for screening purposes before bacteriological culturing (Dohoo & Leslie, 1991; Erskine, 1992; Reneau, 1986). However, a considerable number of cows with an increased CSCC did show negative results in bacteriological culturing (Dohoo & Leslie, 1991; Emanuelson et al., 1987; Emanuelson & Wever, 1989; Kristula et al., 1992; McDermott et al., 1982; Poutrel & Rainard, 1982; Reneau, 1986; Sheldrake et al., 1983). This invokes a discussion on the relation between bacteriological culturing and increased somatic cell counts.

Therefore, by day to day monitoring of the udder health status, the objectives of this study are to gain increased knowledge in the bacteriological status of the milk in relation to CSCC.

MATERIALS AND METHODS

Data collection

The study is carried out on an experimental farm from February 9 1995 until March 15

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1995 (10 weeks). The farm had a low bulk milk somatic cell count (bulk milk somatic cell count < 250,000 cells/ml) for more than 2 years. All cows milked during the experimental period were in the study. In total, 88 cows were in the study. During each milking from each cow, a whole milk sample was taken to determine the CSCC. These samples were transported to the laboratory and CSCC was determined within 24 hours, using the Fossomatic.

Twice weekly, with intervals of 3 and 4 days respectively, quarter premilk samples were taken in duplo for determination of quarter milk bacteriology. All the duplo quarter milk samples were taken aseptically by trained personnel of the Animal Health Service. The udder was cleaned with one towel per cow, whereafter the teat orifice was disinfected by wiping them with tissues soaked with 70% isopropyl alcohol. The first two squirts of milk were discarded before the sample was collected. One sample was taken for bacteriological examination the other sample was frozen for possible extended bacteriology.

The collected samples were incubated according to the standard procedures of the International Dairy Federation within 24 hours of collection. An inoculum of 0.01 ml is spread on a sheep blood agar plate and on Edward's Medium for selective isolation of streptococci (groups A, B, C, G, E and L). The crystal violet in the Edward's Medium inhibits growth of *Staphylococcus aureus*. Incubation temperature is 37 °C and plates are examined after 24-48 hours incubation. Strains of *S. aureus* producing a_1, a and b or b haemolysis are usually coagulase positive. A sample is considered *S. aureus* positive from a single pure colony on. A single colony or, preferably a loopful of growth from a 24 hours blood agar culture, is emulsified in 1.0 ml of pooled citrated rabbit plasma diluted 1:5 with physiological saline. Clothing of the plasma is examined after 1, 2, 4, 8 and 24 hours incubation at 37 °C. Any degree of clothing of plasma is accepted as confirmation for *S. aureus*.

For the extended bacteriological culturing the milk samples were frozen by -20 °C. After thawing and incubation, an inoculum of 0.01 ml was spread on a sheep blood agar plate. After this procedure the samples were cultured and examined as described above.

Data processing

Summarizing definitions: Prevalence and incidence rate (cases per 100 cow days at risk) were calculated. When a pathogen was found in a cow, that cow was withdrawn from the at-risk population for 14 days. Based on the complete set of CSCC measurements, per cow the average CSCC was calculated at a natural logarithm scale. According to their average CSCC, cows were classified in 4 somatic cell count classes: smaller than 100,000 cells/ml, between 100,000 and 250,000 cells/ml, between 250,000 and 500,000 cells/ml, and larger than 500,000 cells/ml. Cows were also classified according to the number of milkings with an increased (> 250,000 cells/ml) CSCC. Three classes were created: no milkings with an increased CSCC, between 0 and 1.5 % of the milkings had an increased CSCC and more than 1.5 % of the milkings had a CSCC larger than 250,000 cells/ml. Also the frequency of diagnosis of pathogens was determined per cow.

Definition of increased CSCC: Cows were defined to have an increased CSCC during a

milking when the CSCC of that milking was higher than either 250,000 or 500,000 cells/ml. Because of the variability in CSCC, attentions were not only made for an increase of the CSCC during one milking, but also when CSCC was increased for two or three consecutive milkings. Moreover, attentions were also given when the CSCC was increased for two milkings in row, following two consecutive milkings with a CSCC below the threshold. The first 7 days after calving were not used in the calculation of the attentions, nor in the calculation of the mean CSCC.

Statistical analyses: Using two-way tables, bacterial infections were related to the mean CSCC and occurrence of high CSCC. For pathogens with more than 5 occurrences the effect of bacterial infection rate on the mean CSCC was tested with the following linear regression model:

$$Y_i = \mu + IR_i + E_i \quad (1)$$

where
Y_i = mean CSCC of cow i (on log scale)
μ = overall mean
IR_i = bacterial infection rate for cow i
E_i = residual error

To determine the effect of bacterial infection rate on the occurrence of high CSCC, logistic regression was used comparable to Eq. (1), with the percentage of high CSCC as dependent, and bacterial infection rate as independent variable.

The relation between CSCC attentions and bacteriology is analyzed for the first bacteriological sample taken on or after a CSCC attention and for the last bacteriological sample taken before the CSCC attention. In the period between two consecutive bacteriological samplings only one CSCC attention could be given. The analyses were carried out using two-way tables, which were evaluated using the Pearson chi-square statistic.

All analyses were carried out with the Genstat 5 statistical software (Genstat 5 Committee, 1993)

Table 1. Prevalence and incidence rate of mastitis during the study period

Pathogen	Prevalence (%)	Incidence rate (cases/100 days at risk)
Staphylococcus aureus	1.71	0.21
Streptococcus dysgalactiae	2.49	0.60
Escherichia coli	0.2	0.06
Streptococcus uberis	0.78	0.19
Actinonymec pyogenes	0.07	0.02
Corynebacterium bovis	2.71	0.50
Coagulase-negative staphylococci	0.07	0.02

Pathogen	Prevalence (%)	Incidence rate (cases/100 days at risk)
Lactobacillus lactis	0.07	0.02
Enterococci	0.50	0.12
Contagious pathogens	4.20	0.81
Environmental pathogens	1.07	0.27
Clinical mastitis	0.36	0.10

RESULTS AND DISCUSSION

During the study period, five cows got a case of clinical mastitis. Results from these cows have been removed from the analyses during the first 14 days on and after the occurrence of the clinical mastitis. Table 1 presents the prevalence and incidence rate of the various pathogens. *Corynebacterium bovis* had the highest prevalence (2.71 %) while *Streptococcus dysgalactiae* gave the highest incidence rate (0.60 cases/100 cow days). This indicates that *C. bovis* had more repeated diagnoses. Compared to other studies (Erskine et al., 1988; Lam et al., 1996) on farms with a low bulk milk somatic cell count, the high incidence of *S. dysgalactiae* and the low incidence of *Escherichia coli* were remarkable.

The number of cows which had one or more infections with a specific pathogen can be found in Table 2. For example, there were four cows which had one or more *S. aureus* infections and two cows which had an infection with *S. dysgalactiae*. For most types of pathogens, there was a significant relation between the occurrence of bacterial infection and the mean CSCC and between the occurrence of bacterial infection and the frequency of high CSCC (Table 2). There was no significant relation between infection with *S. dysgalactiae*, *C. bovis* and CSCC (either frequency of high CSCC or mean CSCC). *S. aureus*, *Streptococcus uberis* and enterococci had a positive relation with the mean CSCC and with the frequency of high CSCC. Enterococci are generally regarded as a minor pathogen, but these results suggest that enterococci have an effect on CSCC. The fact that *S. dysgalactiae* is not associated with an increased CSCC is remarkable, since *S. dysgalactiae* is regarded as a major pathogen.

Table 2. Mean CSCC and frequency of high CSCC in relation to bacteriology. Each cow is classified according to CSCC and the occurrence of 1 or more cases of bacterial infection.

Pathogen present		Mean CSCC (1,000 cells/ml)					High CSCC (% of milkings)			
no(0) or yes (1)		< 100	100-250	250-500	> 500	P[a]	0	0-1.5	>1.5	P[a]
S. aureus	0	49	17	3	2	*	49	7	15	
	1	2	0	2	0		0	1	3	
S. dysgalactiae	0	37	14	3	1		37	7	11	
	1	14	3	2	1		12	1	7	

Pathogen present		Mean CSCC (1,000 cells/ml)					High CSCC (% of milkings)			
no(0) or yes (1)		< 100	100-250	250-500	> 500	P[a]	0	0-1.5	>1.5	P[a]
S. uberis	0	50	14	4	1	**	47	8	14	**
	1	1	3	1	1		2	0	4	
C. bovis	0	36	8	5	2		32	5	14	
	1	15	8	5	2		17	3	4	
Enterococci	0	51	16	4	0	***	49	8	14	***
	1	0	1	1	2		0	0	4	
Contagious	0	36	14	1	1	**	37	6	9	*
	1	15	3	4	1		12	2	9	
Environmental	0	48	13	4	1	**	46	7	13	*
	1	3	4	1	1		3	1	5	
Minor	0	35	7	4	0	*	32	5	9	*
	1	16	10	1	2		17	3	9	

[a] Significance level: * $P < 0.10$, ** $P < 0.05$, *** $P < 0.01$

Table 3 shows the results of bacteriological culturing after an increase of CSCC. Attentions based upon increased CSCC during three consecutive milkings is not presented, since it did not give any better results. It can be seen that a low percentage of *S. dysgalactiae* and *C. bovis* infections were associated with increased CSCC. The highest percentage of found pathogens was for *S. aureus* and enterococci. This is consistent with the earlier observations. It is apparent from Table 3 that even with a very low threshold (CSCC > 250,000 cells/ml for only one milking) not all infections with major pathogens could be detected. Moreover, a relatively high precentage of the attentions did not give a positive bacteriological result, varying from 78 % for a threshold of 250,000 cells/ml during one milking to 70 % for a threshold of 500,000 cells/ml for two consecutive milkings. Increasing the threshold gives a lower percentage of detected bacterial infections but also a lower negative bacteriology rate. So for screening purposes, a low threshold should be used.

Table 3. Number of pathogens found in the first set of quartersamples taken after an increase in CSCC. N is the total number of a specific pathogen that were cultured.

		Threshold					
Pathogen	N	CSCC > 250,000 cells/ml			CSCC > 500,000 cells/ml		
		1 milking high	2 milkings high	2 milkings low and 2 milkings high	1 milking high	2 milkings high	2 milkings low and 2 milkings high
S. aureus	24	19 (79 %)	18 (75 %)	2 (8 %)	16 (67 %)	12 (50 %)	5 (21 %)
S. dysgalactiae	35	13 (37 %)	10 (29 %)	2 (6 %)	10 (29 %)	7 (20 %)	1 (3 %)
E. coli	3	0 (0 %)	0 (0 %)	0 (0%)	0 (0 %)	0 (0%)	0 (0%)
S. uberis	11	7 (64 %)	6 (55 %)	2 (18 %)	4 (36 %)	4 (36 %)	4 (36 %)
A. pyogenes	1	1 (100 %)	0 (0%)	0 (0%)	0 (0 %)	0 (0%)	0 (0%)
Major pathogens	74	40 (54 %)	34 (46 %)	6 (8 %)	30 (41 %)	23 (31 %)	10 (14 %)
C. bovis	38	3 (8 %)	0 (0%)	0 (0%)	0 (0 %)	0 (0%)	0 (0%)
Coag-neg. staph.	1	1 (100 %)	1 (100 %)	0 (0%)	1 (100 %)	0 (0%)	0 (0%)
Enterococci	7	7 (100 %)	6 (86 %)	1 (14 %)	6 (86 %)	5 (71 %)	1 (14 %)
L. lactis	1	1 (100 %)	1 (100 %)	0 (0%)	0 (0 %)	0 (0%)	0 (0%)
Minor pathogens	47	12 (26 %)	8 (17 %)	1 (2 %)	7 (15 %)	5 (11 %)	1 (2 %)
Negative	1301	196 (78%)[a]	134 (76%)[a]	52 (89 %)[a]	98 (73 %)[a]	64 (70 %)[a]	36 (77 %)[a]
No of attentions		248	176	59	135	92	47
P[b]		***	***		***	***	***

[a] Calculated as percentage of the total number of attentions
[b] Significance level: *** $P < 0.01$

The results in Table 3 are results, as would be found when CSCC is used for screening purposes. Bacteriological results on or after the occurrence of an increased CSCC. A possible explanation for the occurrence of negative bacteriological results, could then be that the increase in CSCC is caused by a pathogen and that at the moment of milk sampling the pathogen was already removed from the udder. Therefore, in Table 4 bacteriological results of milk samples taken before and after the increase of CSCC are presented. When compared to the results presented in Table 3, these results show approximately the same percentages of found pathogens. However, the percentage of negative milk samples after an increase in CSCC was lower. For a threshold of 250,000 cells/ml during one milking, 66 % of all attentions gave a negative bacteriological result before or after the attention. This percentage was 49 % for a threshold of 500,000 cells/ml during 2 consecutive milkings. The latter threshold is so strict that there is not a high probability that the increase of CSCC is coincidental. However, a large percentage of the milk samples associated with high CSCC still gave a negative bacteriological culturing. This might be explained by the fact that not all bacteria could be found by bacteriological culturing. However, extended bacteriological culturing hardly gave better results.

Table 4. Number of pathogens found in the first set of quartersamples taken after an increase in CSCC in the last set of quartersamples taken before an increase of CSCC. N is the total number of a specific pathogen that were cultured before or after an increase in CSCC.

		Threshold					
Pathogen	N	CSCC > 250,000 cells/ml			CSCC > 500,000 cells/ml		
		1 milking high	2 milkings high	2 milkings low and 2 milkings high	1 milking high	2 milkings high	2 milkings low and 2 milkings high
S. aureus	34	27 (79 %)	26 (76 %)	7 (21 %)	23 (68 %)	19 (56 %)	10 (29 %)
S. dysgalactiae	57	21 (37 %)	16 (28 %)	2 (4 %)	15 (26 %)	11 (19 %)	2 (4 %)
E. coli	6	1 (17 %)	1 (17 %)	1 (17 %)	1 (17 %)	1 (17 %)	1 (17 %)
S. uberis	21	14 (67 %)	12 (57 %)	3 (14 %)	10 (48 %)	8 (38 %)	5 (24 %)
A. pyogenes	2	1 (50 %)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)
Major pathogens	120	64(53 %)	55 (46 %)	13 (11 %)	49 (41 %)	39 (33 %)	18 (15 %)
C. bovis	66	6(9 %)	1 (2 %)	1 (2 %)	0 (0 %)	0 (0 %)	0 (0 %)
Coag-neg. staph.	2	2 (100 %)	2 (100 %)	1 (50 %)	2 (100 %)	1 (50 %)	1 (50 %)
Enterococci	11	11 (100 %)	9 (82 %)	5 (45 %)	8 (73 %)	7 (64 %)	1 (9 %)
L. lactis	2	2 (100 %)	2 (100 %)	0 (0 %)	0 (0 %)	0 (0 %)	0 (0 %)
Minor pathogens	81	21 (26 %)	14 (17 %)	7 (9 %)	10 (12 %)	8 (10 %)	2 (2 %)
Negative	1204	163 (66%)[a]	107 (61%)[a]	42 (68 %)[a]	76 (56 %)[a]	45 (49 %)[a]	24 (55 %)[a]
No of attentions		248	176	62	135	92	44
p[b]		***			***		

[a] Calculated as percentage of the total number of attentions
[b] Significance level: *** $P < 0.01$

Also other studies, regarding the use of CSCC for screening purposes, presented rather high percentages of milk samples with negative bacteriological results after an increase in SCC (Dohoo & Leslie, 1991; Emanuelson et al., 1987; Erskine, 1992; Reneau, 1986). The farm used for this study is a low bulk milk somatic cell count farm. Prevalence of pathogens is generally low at this type of farm. Because attentions were also based upon repeated milkings with increased CSCC, a coincidental increase cannot be used as explanation for the low predictive value. Moreover, looking at bacteriological culturing before an increase in CSCC did not show much better results. This might be an indicatioin that either the bacteriological research was not sufficient to determine the pathogens or that there were other reasons for the increase in CSCC in these cows. It has been demonstrated that the shedding pattern of *S. aureus* makes it difficult to determine bacterial infection with *S. aureus* with a single milk sample (Adams et al., 1992; Sears et al., 1990). It is not expected on the farm of this study (low bulk milk somatic cell count) that there are many cows with S. *aureus* infections. And besides, even with consecutive milk samples, we could not determine bacteriological contamination in many quarters.

Until now, high CSCC is mostly explained by bacteriological contamination. Although descriptions are made on the relation between milk production, age, lactation stage, stress and feeding on CSCC, not many other possibilities are given for a large increase of CSCC (Leslie et al., 1983). Research into other causative organisms besides bacteria could not be found. The results of the study described in this paper give evidence that it might be interesting to search for other causes for increased CSCC on low bulk milk somatic cell count farms besides bacteria.

The study described in this paper is carried out on only one farm. Because the incidence and prevalence rate do not differ very much from results found in studies on commercial dairy farms, the results of this study might be generalized to commercial dairy farms with the same type of mastitis status. However, since it is only one farm, results should be treated with caution. Still there is a lack of knowledge in the explanation of increased CSCC on farms with a low bulk milk somatic cell count. Especially with the growing number of this type of well managed dairy farms, it is important to gain that knowledge.

CONCLUSIONS

There is a relationship between CSCC and bacteriological contamination. However, in many cases, increased CSCC is not accompanied by positive bacteriology. A threshold of 250,000 cells/ml should be used to screen cows for bacteriological sampling. Increased CSCC might not only be caused by bacteriological infections, but it seems that, at least on a low BMSCC farm, there are also other mechanisms leading to increased CSCC.

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A DISEASE MANAGEMENT INFORMATION SYSTEM FOR PROFITABLE BROILER PRODUCTION

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INTRODUCTION

World production of poultry meat represents 23% of all meat production and is increasing annually. During the past twenty five years world poultry meat production and consumption has trebled with higher production (20.6 kilograms per head) in countries with a developed market economy than production (3.6 kilograms per head) in those countries with a developing market economy. Important factors in the growth of the poultry industry are the attractiveness and aesthetic acceptability of poultry meat to consumers and the highly competitive costs of production. The increased size of the poultry meat industry, to satisfy increased consumer demand, has been made possible by the introduction of intensive production systems utilising new technologies. For example, in the United Kingdom (UK), the poultry meat industry is concentrated in less than twenty large integrated organisations most of which have control of broiler parent flocks, hatcheries, as well as the broiler generation production. Most integrations also control the processing of the final broiler product. Effective disease control at all levels in such intricate, integrated organisations has been an essential component of the growth and economic success of the industry (Law and Payne, 1990).

Numerous attempts have been made to estimate the economic losses caused by disease in poultry meat production. In general, the prevalence and severity of disease, both viral and bacterial, is greater in developing countries where management systems are often less intensive and less well controlled. In the UK, mortality has been estimated to cause a minimum loss of 7% of the value of the industry, while the inclusion of losses due to reduced productivity, condemnations and the cost of prophylaxis and treatment has been estimated to increase the overall losses to approximately 20% (Biggs, 1982). However, a wide range in the prevalence and severity of disease between individual flocks and between poultry organisations is common and therefore the opportunity for effective disease control to reduce substantially the overall cost of poultry meat production is always present (Jones et al., 1978).

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In a large poultry organisation, the monitoring of the performance of broiler flocks is thus important for its' effective management. Production parameters which affect the performance of individual producers and/or houses must be identified and management methods modified to optimise production. In addition, the occurrence of disease incidents can have serious implications for profitable broiler production for both efficient and inefficient producers and requires both identification and economic assessment. Preventative measures against such disease incidents may be costly and in excess of the actual cost of the disease.

This paper describes a computerised management information and retrieval system for production data from individual broiler flocks, controlled centrally by a large poultry organisation in Northern Ireland. The system was initiated in 1983 by the Veterinary Sciences and Biometrics Divisions of the Department of Agriculture for Northern Ireland. This paper highlights how this system has been used to qualify and quantify parameters of a number of economically important diseases in commercial broiler flocks.

Data Collection

Broiler organisation structure

The broiler organisations monitored by the computer system consists of several types of physical locations which are widely distributed, as is common with other large integrated poultry organisations involved in broiler production. To maintain a minimal disease status, breeder flocks tend to be situated in rural areas, remote from any other poultry enterprises. Conversely, hatcheries, processing plants and central administrative units tend to be located in populous areas from which employees can be recruited. Broiler production takes place on many individual units situated on farms throughout a wide area. Information generation occurs at all of these locations in the integrated structure which is diagramatically represented in Fig. 1.

Broiler production data

When the data collection system was designed by McIlroy et al. (1988), the problem of widely distributed information locations had to be overcome and involved detailed discussions with the organisation and an intimate knowledge of the practical handling of the production information on a daily basis. Furthermore, the feasibility of generating and collecting any additional information required by the system had also to be taken into consideration.

For meaningful interpretation of the production information, the method of collecting data had to ensure that such data could be associated uniquely with a specific broiler flock and/or farm site. In the organisation, some of the data collected is associated with several broiler flocks on a particular farm site. On these multiple house sites, different proportions of flocks within different houses are often removed together, thereby not allowing the collection of data from individual flocks from specific broiler houses.

Although essential broiler production data information is generated at several physical locations represented in Fig. 1, a single EXCEL spreadsheet file is used to collect all the

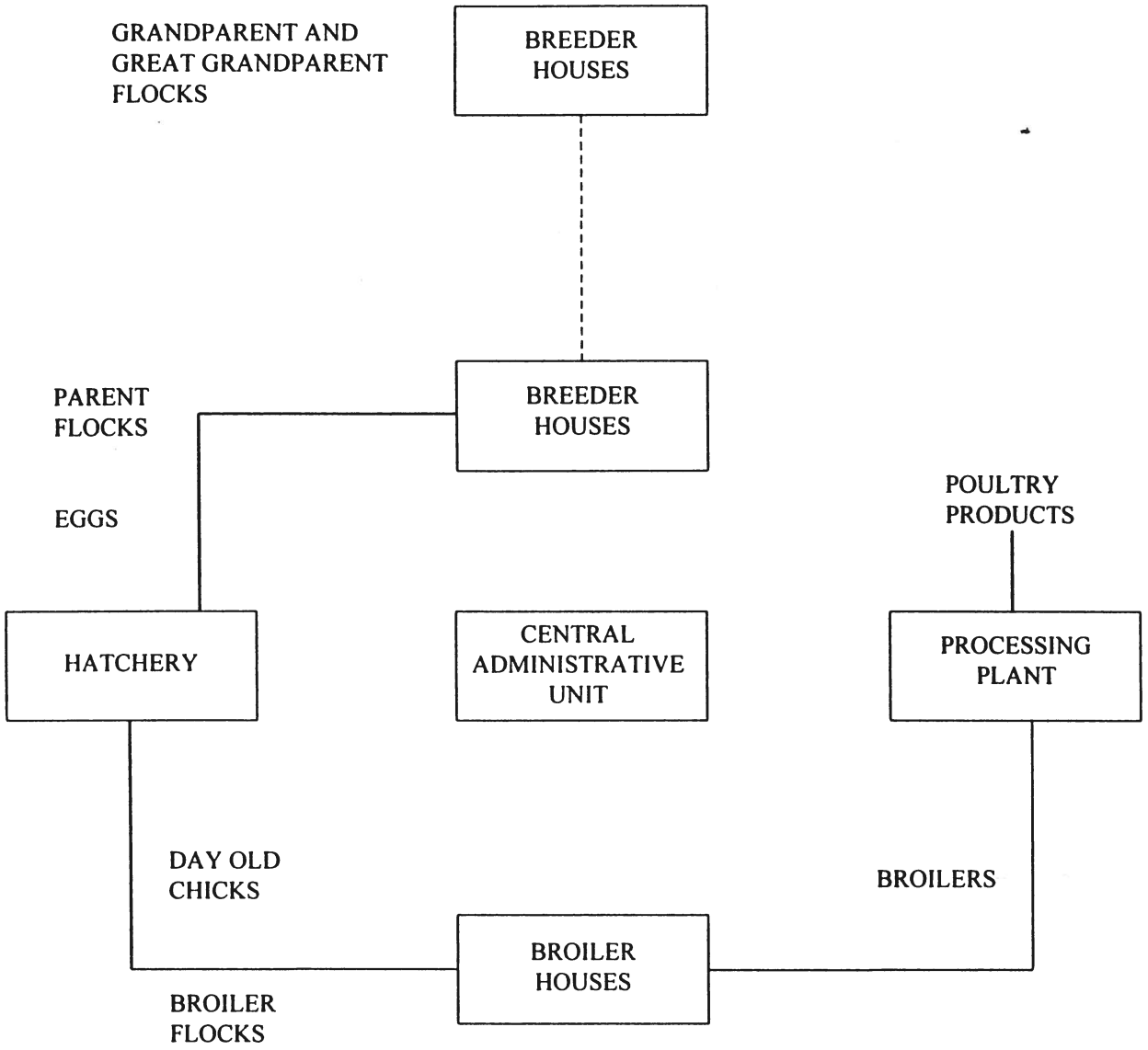

Fig. 1. The structure of a large broiler organisation

production data relating to any broiler flock. The existing management structure within the organisation is used to collate data at the central administrative unit for transfer to the spreadsheet.

The production data and the locations where generated are given below.

Location	Production Data
Breeder flock	Breed of bird Producer and house code Age of flock
Hatchery	Sex of birds Number of birds placed Date when birds placed
Broiler flocks	Producer and house code Stocking density Mortality Type of litter used Number and age of birds removed Date house started and finally cleared Condition of litter Occurrence of disease incidents Preventative measures employed Corrective measures employed Meal manufacturer Amount of gas used Amount of water used
Processing plant	Factory code Average weight per bird Grading information (number of Grade A, B and rejects) Presence of contact dermatitis lesions (hock/breast burns) Percentage Bruising
Central administrative unit	Food conversion ratio Net income (actual income minus cost of feed and day old chicks) Target grading information Target food conversion ratio Target average weight per bird Target net income

The condition of litter in individual broiler flocks is recorded as either dry (ie. the absence of poor litter conditions during the production cycle) or poor (ie. the presence of poor litter

conditions during the production period). Additional information is obtained on the occurrence of incidents when litter appeared to deteriorate very rapidly; in particular, the time during production when such acute outbreaks occurred.

The sex of flocks is categorised as being either male, female, as hatched or mixed sexes. The high stocking density flocks are stocked at 0.48 square feet or less per bird and the low density flocks stocked at 0.49 square feet or greater per bird. The long average age of removal flocks were retained for 47 days or more and the short average age of removal flocks were retained for 46 days or less. These stocking density and age of removal categories are those used by the broiler organisation.

Commercial targets for net income per 1000 birds, food conversion ratio, average weight per bird and mortality are available from the organisation and these are used to take into consideration other production parameters which would be expected to have an effect on the achievable result. The difference between the target value and the actual value achieved by broiler flocks is used to analyse the independent effect each parameter had on profitable broiler production. For reasons of agreed confidentiality results achieved for net income per 1000 birds, food conversion ratio, average weight per bird and mortality are reported as mean percentage deviation values from the expected values.

House information

Information relating to individual broiler houses is also collated onto a spreadsheet. The information collected is age of house, condition of house, size of house, type of ventilation, type and thickness of insulation, type of feeder and type of drinker. This house information is held in a separate file under the individual house code and can be integrated with the broiler flock production database.

Breeder flocks

Information on individual breeder flocks, as with broiler flocks, is generated from several physical locations also represented in Fig. 1. Data is collated and collected from the hatchery in the organisation. Data from individual breeder flocks is collected on a weekly basis and overall average production figures for the entire production cycle are also collected.

The production data and the locations where generated are given below.

Location	Production data
Breeder flocks	Producer and house code
	Type of feeding system
	Breed of bird
	Date day-old chicks placed
	Date house finally cleared
	Total number of point-of-lay pullets
	Age of birds at first egg
	Stage in laying period
	Length of laying period
	Meal manufacturer
	Daily feed per 100 birds
	Amount of gas/electricity used
	Preventive measures employed
	Corrective measures employed
	Occurrence of disease incidents
	Percentage mortality
Hatchery	Length of setting period
	Percentage egg production
	Percentage hatchable eggs
	Actual hen house average
	Percentage fertility
	Percentage hatchability
	Net return per point-of-lay pullet
Processing plant	Factory code
	Average weight of carcase - pullets
	- cockerels

Computer System

When the system was first established by McIlroy et al. (1988), several types of computer configuration were considered and evaluated with respect to the potential benefits expected. In view of the large volumes of data to be processed, validated and analysed, the use of personal computers located at the central administrative units of the organisations was not considered to be practical or feasible. Furthermore, it was considered that the control of the system should reside with the specialist veterinary and computer expertise available from the Department of Agriculture and absent from the poultry organisation.

Hardware

The system was initially mounted on a VAX 6320 superminicomputer but has subsequently been transferred to a DEC ALPHA computer currently utilising 512 megabyte RAM and 20 gigabyte hard disk for backing store. This computer has a very powerful, interactive operating system which facilitates rapid analysis and retrieval of data via several distributed PC terminals and incorporates user friendly editing facilities.

Software

DEC Alpha ORACLE

The principle software package available on the computer is ORACLE (ORACLE Corporation, California). ORACLE is designed to be used both by people with little or no computer experience and by computer professionals. It operates effectively in commercial, technical, scientific, industrial and educational environments. Typical tasks which ORACLE performs range from answering a casual question to creating a complex report. In ORACLE, data structures are defined and stored separately from programs. It is possible to write any number of programs that use the same data structure without having to redescribe the data each time. ORACLE is a tool for managing data and can perform the following functions:-

(1) Define data in a way that fits your information management needs.

(2) Store and modify data.

(3) Retrieve data and display it on a PC, record it in a file, or print it on paper.

(4) Format data in reports.

(5) Represent data in graphs.

(6) Use forms to format a VDU for input and display of data.

(7) Use a Graphical interface to correct typing and syntax errors.

(8) Obtain access to data files distributed across a network.

Creating an ORACLE information management application is a two-phase process. In the first phase, the data to be used in the application is described. This needs to be performed only once to establish a foundation on which to build the application. In the second phase, ORACLE statements are used to process the data associated with these definitions. Although ORACLE incorporates a powerful retrieval package, it had to be specially configured to meet the analysis requirements of the system. Special programs were written to allow the transfer of data for use in ORACLE. The use of ORACLE enables the formation of extract files of categorised subsets of production data before subsequent analysis by other software packages. ORACLE procedures are defined sequences of ORACLE commands and statements which are stored in a common data dictionary for later use.

GENSTAT

Routine statistical analyses, such as chi-squared tests, Student's t-tests, analysis of variance and regression/correlation analysis of subsets of production data are carried out using GENSTAT (Rothamstead Experimental Station, Herts, UK). GENSTAT is a very general computer program for statistical analysis, with all the facilities of a general-purpose statistical package. All the usual analyses are readily available using the standard GENSTAT commands

or directives. However, GENSTAT is not just a collection of pre-programmed commands for selecting from fixed recipes of available analyses. It has a very flexible command language which easily allows the writing of specific programs to cover the occasions when the standard analyses do not give exactly what is required, or when a new technique is developed. Programs can be formed into procedures to simplify their future use or to make them easily available to other users.

GENSTAT has many features for performing calculations and for manipulating data. The statistical facilities in GENSTAT are divided into four areas: regression, analysis of designed experiments, multivariate and cluster analysis, and time series. The regression facilities in GENSTAT enable simple and multiple linear regressions to be performed. Separate or related regression lines may be fitted where data are partitional into groups. GENSTAT can perform analysis of variance for virtually all the standard experimental designs and associated clinical trials. Commands (directives) are available for all the standard multivariate techniques. Time series analysis may also be performed.

Other software features

Software was specially written to link the data-sets containing production information with a database containing weather variables such as temperature, relative humidity, rainfall and wind speed. The database is updated monthly and contains all daily weather information recorded by the Meteorological Office (Aldergrove Station) and computerised at their headquarters in Belfast, Northern Ireland. This software provides comprehensive facilities for examining climatic trends over a series of years and calculating seasonally adjusted means for each month of the year. Monthly values may be combined (eg. bi-monthly, quarterly, etc.) and moving averages produced for analysis. The program also enables autocorrelation structures to be rigorously examined. In addition, linear combinations of weather variables may be correlated over any selected time period with corresponding production data subsets.

Separate files were created to store the production information which was additional to that routinely collected on the survey forms. Broiler house information and disease status are held in such files and can be merged with the main database for analysis with flock production information. A special program was written to facilitate the merging of these files and enables the system to be completely open-ended and flexible with respect to any additional production information which may be considered important at any particular time.

Reports

A wide variety of reports are produced by the system, comprising management reports sent monthly to the poultry organisation and more detailed statistical reports for analysis. The management reports provide the principal method of interaction with the poultry organisation. Regular liaison meetings are used to discuss these reports and identify areas of production which may be manipulated by management and the subsequent effects of such manipulations. Ad hoc reports are produced on request on an interactive basis. Statistical reports are used to identify trends and highlight any observations of possible interest. These are also considered at the regular liaison meetings with a view to the inclusion of additional production information in the management reports. Reports may also be presented in a graphical form utilising

histograms, pie charts, scatter diagrams, etc., and enable the presentation of complex production trends in a visual and more easily perceptible manner.

Results and Discussion

Whereas other computerised systems for profitable animal production in dairy and pig herds utilise information generated continuously on a single production unit, the system described here requires information from many locations. Furthermore, production information relating to individual broiler flocks is generated over a comparatively short period of time (7-9 weeks) and must be compared with the information generated in other broiler flocks over the same time period, as well as information previously generated by other flocks in any particular broiler house. This clearly involves a complex method of collecting and coding production information for useful analysis and may be the reason why such a comprehensive system has not previously been reported.

The system has currently collated and analysed production data from 6000 flocks incorporating 120,036,579 birds over a 13 year period. As it is anticipated that the system will continue indefinitely, effective analysis of this volume of data would not have been possible using a personal computer. Furthermore, the sophisticated statistical and retrieval packages necessary for the analysis of the data require a virtual memory computer system such as the DEC ALPHA range. The multitude of production parameters involved in broiler production and the complex interactions present necessitates that preliminary analysis is carried out by specialist staff in the Department of Agriculture and only relevant results distributed to the poultry organisation. Also, as this is still a relatively new area for profitable broiler production, the system has to be inherently flexible for the inclusion of additional production information and its subsequent merging with the existing database. These additional factors also militate against the use of local personal computers running software packages similar to those currently in use for dairy and pig herd production systems.

When the system was set up, little information was available on the interdependence of production parameters and their quantitative effect on overall profitable broiler production. Data were initially analysed using a wide range of intervals of small strata size to identify trends and highlight all observations of possible interest. This analysis involved the production of means, standard deviations and frequency distributions of individual parameters from all broiler flocks. Subsequent analysis involved the pooling of strata into gradations meaningful to the industry. Parameters were then cross-clarified to assess interdependence using Fisher's Exact test. Where considered relevant, selected production parameters were examined by month and year both over all broiler flocks and individual houses. Furthermore, individual production parameters were correlated with other parameters over time after autocorrelation structures had been taken into account.

Net income per 1000 birds is generally considered within the broiler industry to be the most accurate economic index of production and was used as the base-line when assessing the effect that other production parameters had on profitable production. Of particular interest was the seasonality pattern of net income in broiler flocks. Net income recorded during the winter months (October to April inclusive) was, on average 9% ($P<0.001$) less than during the summer months. Further analysis of net incomes showed that some producers consistently

recorded poor performance throughout the year and led to the initiation of a survey of individual flocks with regard to the presence or absence of disease. For example, the presence, by histopathological examination, of necrosis of lymphocytes and/or atrophy in bursae has been assessed. The lesions in all cases were typical of Infectious Bursal Disease virus. Analysis demonstrated a statistically significant difference of 10% ($P<0.001$) between net incomes from houses where the disease had been identified and those in which it had not. Assessing the cost effectiveness of available control measures against this disease was subsequently investigated as a consequence of this finding. The results have been reported by McIlroy et al. (1989).

A factor which was found to have a major effect on profitable production was the recorded presence or absence of poor litter conditions in the houses during the production period. The presence of poor litter was recorded in two categories, the gradual deterioration of litter conditions during the production period and the occurrence of incidents when litter conditions deteriorated rapidly, usually within 24 hours. Net income of flocks in the latter category was, on average, 20% ($P<0.001$) less than the income achieved in flocks where poor litter was not recorded. Flocks which had gradual litter deterioration were also significantly less economic (8%; $P<0.005$) than those which did not record poor litter.

Another production parameter associated with the occurrence of poor litter was the presence of dermatitis lesions on the hock and breast area of processed broilers. The presence of such lesions has serious implications for fulfilling daily contract requirements for fresh whole broiler carcasses, as birds may be down-graded, especially if the breast area is affected. In view of the serious nature of this condition to the broiler industry, a special statistical analysis was carried out to determine the association of other production parameters on the incidence of the condition. A major conclusion from this analysis was that lesions, especially the more economically important breast lesions, occur most frequently when male birds are kept at a high stocking density in the presence of poor litter conditions. Furthermore, the average age of removal in such flocks appears to influence the incidence of the condition. Discussions at liaison meetings have led to management modifying the stocking density of males in many incidences and the removal of such birds as soon as possible after the occurrence of poor litter conditions. Current results indicate that such management procedures and others identified from the survey are effective in reducing the incidence of dermatitis lesions.

Many production parameters were correlated with the weather variables held in the meteorological database. Significant correlations were found with some production parameters. Of particular interest was the highly significant correlation of 0.60 ($P<0.001$) found between the average incidence of dermatitis lesions and the level of relative humidity. Assuming a dependency relationship between relative humidity percentage (x) and the percentage mean incidence of dermatitis lesions (y), a regression analysis was performed. The equation obtained was $y = -31.9+0.65x$. The standard error of the regression coefficient was 0.14. This result is of special concern for broiler production in Northern Ireland, a geographic area with high humidity especially in the winter months. Discussions with the poultry organisation have led to the suggestion that when cold, damp, climatic conditions prevail outside, the maintenance of adequate ventilation, temperature and humidity in broiler houses may be difficult. Under such environmental stresses litter may deteriorate rapidly resulting in

an increased incidence of dermatitis lesions. The poultry organisation has subsequently advised broiler producers to monitor carefully environmental factors such as temperature, humidity, ventilation and litter especially during cold, damp, climatic conditions.

Other recent uses of the system have included research to assess the economic effects of clinical and subclinical Chicken Anaemia Agent Infection on profitable Broiler Production. Current work includes an assessment of the effect on profitability of vaccination regimes for Marek's disease.

This unique computerised system is considered by the management of the poultry organisation to be a major benefit for monitoring and optimising broiler production parameters. They consider time spent by staff collecting and collating information to be cost effective, when compared with the benefits achieved. The system has recently been expanded to include other production areas such as types of feeding regimes and also to categorise producers into sets depending on the profitability of their production. This dynamic, interactive system will continue to monitor broiler production within this organisation and may be expanded to include other broiler organisations in Northern Ireland and internationally.

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ELECTION

8. The election of office bearers and ordinary committee members will take place at the Annual General Meeting. Ordinary members of the Executive Committee will be elected for a period of three years. Retiring members of the Executive Committee will be eligible for re-election. Members will receive nomination forms with notification of the Annual

General Meeting. Completed nomination forms, including the signatures of a proposer, seconder, and the nominee, will be returned to the Secretary at least 21 days before the date of the Annual General Meeting. Unless a nomination is unopposed, election will be by secret ballot at the Annual General Meeting. Only in the event of there being no nomination for any vacant post will the Chairman take nominations at the Annual General Meeting. Tellers will be appointed by unanimous agreement of the Annual General Meeting.

FINANCE

9. An annual subscription will be paid by each member in advance on the first day of May each year. The amount will be decided at the annual general meeting and will be decided by a simple majority vote of members present at the Annual General Meeting.

10. The Honorary 'Treasurer will receive, for the use of the Society, all monies payable to it and from such monies will pay all sums payable by the Society. He will keep account of all such receipts and payments in a manner directed by the Executive Committee. All monies received by the Society will be paid into such a bank as may be decided by the Executive Committee of the Society and in the name of the Society. All cheques will be signed by either the Honorary Treasurer or the Honorary Secretary.

11. Two auditors will be appointed annually by members at the Annual General Meeting. The audited accounts and balance sheet will be circulated to members with the notice concerning the Annual General Meeting and will be presented to the meeting.

MEETINGS

12. Ordinary general meetings of the Society will be held at such a time as the Executive Committee may decide on the recommendations of members. The Annual General Meeting will be held in conjunction with an ordinary general meeting.

GUESTS

13. Members may invite non-members to ordinary general meetings.

PUBLICATION

14. The proceedings of the meetings of the Society will not he reported either in part or in whole without the written permission of the Executive Committee.

15. The Society may produce publications at the discretion of the Executive Committee.

GENERAL

16. All meetings will be convened by notice at least 21 days before the meeting.

17. The President will preside at all general and executive meetings or, in his absence, the Senior Vice-President or, in his absence, the Junior Vice-President or, in his absence, the Honorary Secretary or, in his absence, the Honorary Treasurer. Failing any of these, the members present will elect one of their number to preside as Chairman.

18. The conduct of all business transacted will be under the control of the Chairman, to whom all remarks must be addressed and whose ruling on a point of order, or on the admissibility of an explanation, will be final and will not be open to discussion at the meeting at which it is delivered. However, this rule will not preclude any member from raising any question upon the ruling of the chair by notice of motion.

19. In case of an equal division of votes, the Chairman of the meeting will have a second and casting vote.

20. All members on election will be supplied with a copy of this constitution.

21. No alteration will be made to these rules except by a two-thirds majority of those members voting at an annual general meeting of the Society, and then only if notice of intention to alter the constitution concerned will have appeared in the notice convening the meeting. A quorum will constitute twenty per cent of members.

22. Any matter not provided for in this constitution will be dealt with at the discretion of the Executive Committee.

April, 1982
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Corrected January 1997