SOCIETY FOR VETERINARY EPIDEMIOLOGY AND PREVENTIVE MEDICINE

Proceedings of a meeting held at
the University of Edinburgh on
the 2nd, 3rd and 4th of April 1986

Edited by M.V.Thrusfield

ISBN 0 948073 06 3

ACKNOWLEDGEMENTS

Beecham Animal Health, Duphar Veterinary Limited, Glaxovet Limited, Pfizer Limited, MSD AGVET, Smith Kline Animal Health Limited and Upjohn Limited generously gave financial support towards the publication of these proceedings.

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THE EVALUATION OF DISEASE CONTROL SCHEMES

THE EVALUATION OF PIG HEALTH AND DISEASE CONTROL SCHEMES AT THE NATIONAL LEVEL

D. BASINGER*

National disease control in Great Britain effectively began in 1866 with the introduction of the Cattle Diseases Prevention Bill, aimed at the elimination of rinderpest. Central intervention then and since has been mainly influenced by economic and public health factors. The approach is always a fine balance between popular demand and political expediency. Procedures, whether geared to prevention, containment, elimination or amelioration, are dictated by current knowledge of the disease and its severity.

At present (1985), 23 animal diseases are subject to statutory control, 5 relating specifically to pigs. Since 1955, £203 million have been expended on slaughter compensation, of which £48 million again relate to pigs. Besides eradication programmes and other statutory controls, central government has, since the mid-1960s, also offered the farmer preventive medicine programmes in the form of the Poulty and Pig Health Schemes.

Government involvement in pig health

The five main areas of involvement can be summarised as follows:

1. Control of specific diseases, e.g. anthrax, atrophic rhinitis, swine fever, foot and mouth disease, swine vesicular disease, Teschen disease and Aujeszky's disease.
2. Miscellaneous Orders relating to export, import (including tests imposed before and after entry into G.B. and quarantine regulations) movement and sales of pigs and the processing of waste food.
3. Welfare recommendations and definitive guidelines.
4. Laboratory back-up to practitioner diagnoses and treatments at the local level (Central Veterinary Laboratory and Veterinary Investigation Centres).
5. Preventive medicine packages, in association with the Meat and Livestock Commission (MLC), e.g. the MAFF Pig Health Scheme (PHS).

Principles of disease control

The oft stated advantages of an island economy in terms of national herd health still apply. National control measures are therefore geared primarily to dealing with overseas threats. These threats are posed by the movement of live animals; of meat, meat products and meat contaminated materials such as wrapping cloths; of vectors - whether animals, birds or people; and of fomites,

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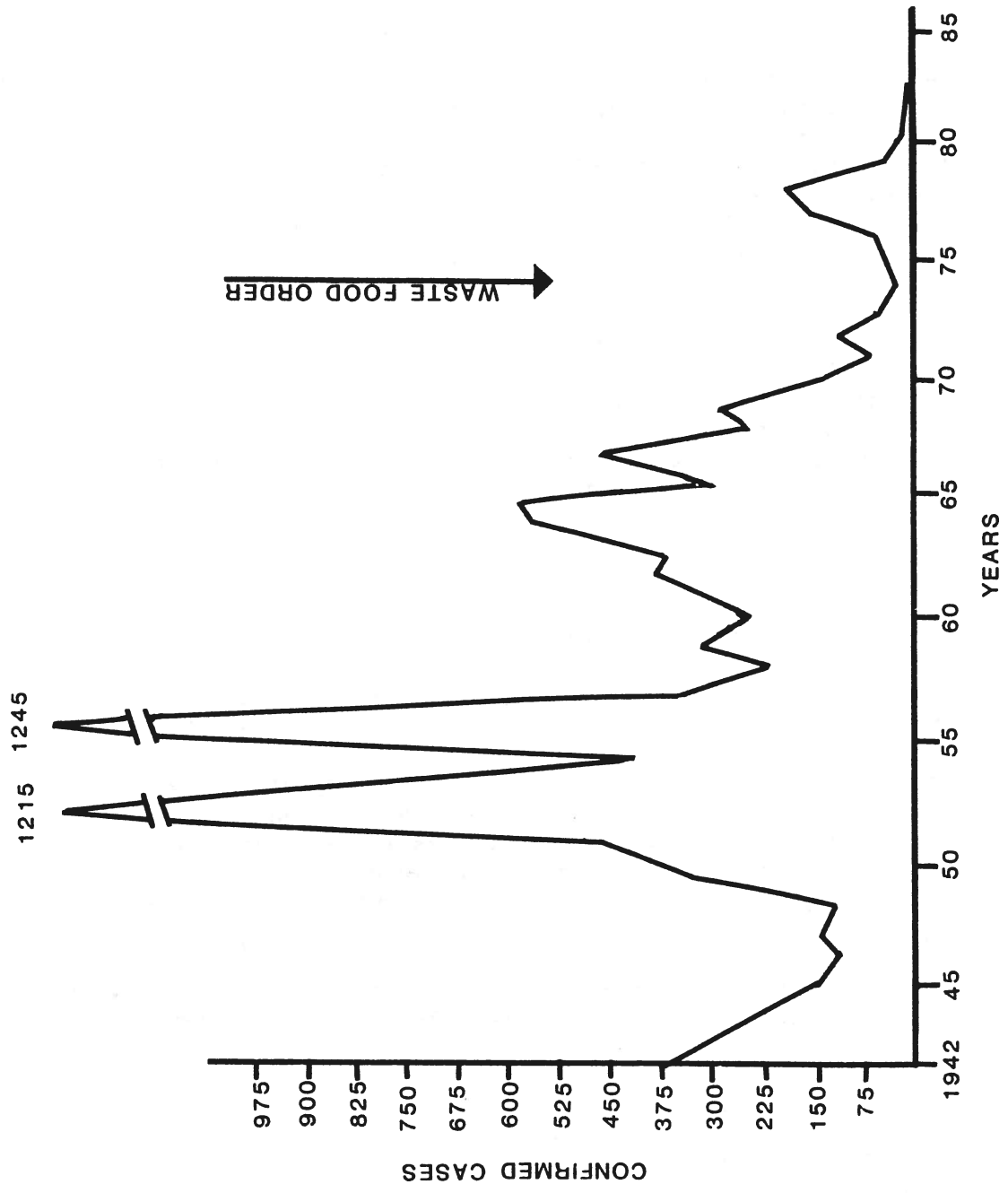


Figure 1. Incidence of anthrax (all species).

such as feedstuffs, litter and equipment. In addition, there is the problem of wind carriage.

Nationally, there are four lines of defence:

1. Prevention - stopping the entry of disease by Import/Quarantine Orders.
2. Containment - stopping spread within the country by:
   a) cutting the infected food chain (Waste Food Order 1973)
   b) restricting movement of infected or potentially infected animals (Movement and Sale of Pigs Order 1975).
3. Elimination - 'stamping out' known pockets of disease (eradication Orders).
4. Amelioration - subsidised use of vaccines etc.

The statutory control of animal diseases is regulated by Orders made under the Animal Health Act of 1981 (previously the Diseases of Animals Act). Diseases are made 'notifiable' and control measures instituted when they are significant enough to warrant government intervention. Influencing factors would include a threat to the national herd through economic 'clout', or the insidious nature of a disease, or its rapid spread; the public health risk; its ready identification either in the field or at the laboratory; and, equally important, the volume of public demand and political will.

An essential preliminary to any system of control is an effective monitoring programme whereby disease prevalence can be assessed. This entails accurate recording of all known outbreaks of the disease and the institution of random and selective herd samplings for the disease within the country's livestock industry.

National control methods

Control measures range from the simple to the Draconian. In all instances, final responsibilty rests with the owner to report the existence of suspected disease to the competent authority. This may result in confirmation plus recording (as in the first attempt at Aujeszky's disease control in 1979); confirmation plus recording plus movement restrictions (as in the first attempt at swine fever control in 1938); or confirmation plus recording plus movement restrictions plus carcase disposal and disinfection (as under the Anthrax Order of 1938). These simpler methods of control are compulsory; but involve no financial outlay except for veterinary input and slaughter for diagnosis. The success of any controls will depend on the soundness of the veterinary judgement which iniated them. When such measures work, as in the case of anthrax (Figure 1), there is no disputing their cost-effectiveness.

A variation of the simpler control methods, described above, is inducement. Initially, acceptance is always voluntary. Inducement policies have resulted in 'free' testing (as in the tuberculosis pre-eradication era) and vaccination schemes (e.g. brucellosis pr-eradication using S19 vaccine; swine fever pre-eradication using crystal violet vaccine; and Newcastle disease post- eradication). The financial outlay in these instances was confined to veterinary input, subsidised vaccines and herd bonuses. However, experience has taught that without quick and accurate recognition of vaccinal titres, vaccines have no place in national disease control schemes ultimately aimed at eradication. Figure 2 shows that swine fever spread faster afer the introduction of the crystal violet vaccination scheme in 1949

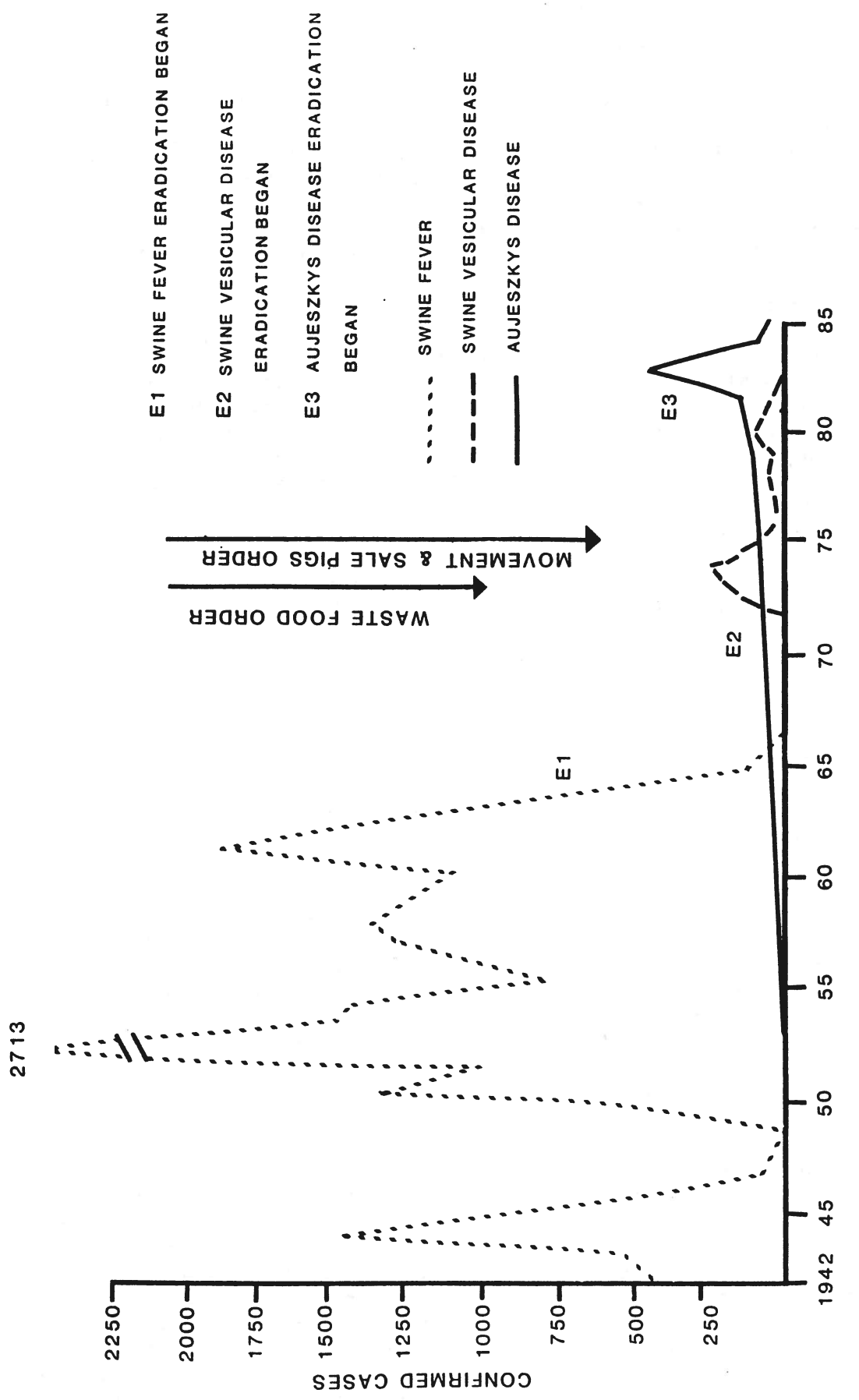


Figure 2. Incidence of swine fever, swine vesicular disease and Aujeszky's disease 1942-1984.

than it did before, helped no doubt by the increased numbers of 'carriers', it created. The usefulness of vaccines in ameliorative control schemes is however not disputed.

The third facet to national control is the attempted elimination of the causal organism, i.e. eradication. This 'go for broke' alternative, satisfying if it succeeds, can be attempted in two ways: by drug or chemical means (e.g. the sheep scab and warble fly schemes) and by slaughter, compensation and disposal of carcases. Slaughter programmes can themselves be conducted in two ways: on the one hand, by a series of herd tests and subsequent removal of reactors, until at least two negative herd tests are obtained; on the other hand, by the complete and immediate depopulation of the infected units and the slaughter of any contacts, with or without disinfection of premises and the use of area restrictions. Again, such a slaughter policy may be carried out with or without salvage of carcases, and with or without payment for consequential losses.

Factors necessary to the success of eradication schemes

1. The threat to the national herd must be properly assessed by in-depth studies of the disease, both at home and abroad.
2. The projected scheme must be economically viable, with a reasonable expectation of a successful conclusion i.e. the probable benefits outweigh the cost of the scheme.
3. The infective agent must be readily identifiable by quick, accurate laboratory/field tests.
4. The operators must have 'sufficient' epidemiological knowledge of the disease in respect of cause, spread factors, amenability to the control measures envisaged, persistence/survival of the organism, and the availability of safe and effective germicides.
5. It must be preceded by adequate monitoring and surveillance to assess geographical distribution and prevalence.
6. It must be launched early enough, before the disease becomes endemic.
7. The policy must be seen to be beneficial to the industry/country as a whole, i.e. preferable to 'living' with the disease or employing stop gap sanitary measures.

Success in itself will be judged on three counts:

1. Whether the organism has actually been eliminated'.
2. Whether, following elimination, we effectively prevent the re-introduction of the disease into the country.
3. whether money has been saved overall and is quantifiable.

In the final analysis, however, success will be measured in terms of customer confidence. This will be reflected in increased trade both at home and abroad.

Cost-effectiveness

Costing a disease control programme (its overall expenditure in administration, manpower, materials and services) is relatively simple. Relating these control costs to the real costs of the disease (sustained losses and projected losses) is far more difficult. No disease appears in

isolation on any farm. Other diseases and other factors will influence its progress. Losses associated with a specific disease (mortality, morbidity, depression of production and even profits) can at best be merely estimates. Even more ephemeral is the costing of what might have happened, but never did happen because of the course of action taken - which is the very essence of preventive medicine and eradication schemes. The best one can do is assess the price of living with a disease, with its probable losses and possible pattern of spread, including any necessary intervention to ameliorate its effects, (e.g. vaccination usage) and compare it broadly with the once-for-all cost over a restricted time period of the disease control scheme. If eradication is the control technique then the consequential losses of the affected farmer (loss of income from the time of depopulation to to the resumption of full production) also must be taken into consideration. This has been quoted as a general figure of £20,000 per herd per year on a 100 sow unit (Vinson & Muirhead, 1981).

Of the 9 pig diseases subject to statutory control (Table 1) , only one control programme might be adjudged a failure. Two others, Teschen disease and African swine fever, have yet to be put to the test. The failure was the attempted eradication of atrophic rhinitis by slaughter and compensation. It began in May 1954, following the introduction of infected Swedish Landrace pigs in the autumn of 1953. In the eight years that the policy continued (Figure 3) some 40 herds were slaughtered out at a compensation cost of £251,042 (£1.5 million updated to 1984). The revocation of the Order in 1962 was attributed to 'the decline in the number of outbreaks and because necessary control could be achieved by ordinary intelligent culling'. A MAFF press notice containing notes to journal editors (22/11/62) is of interest: 'Experience has shewn that the disease does not cause undue losses or have any significant effect on growth or food conversion rates and is not of such economic importance as had been feared.' What defeated the project of course was the lack of basic knowledge regarding aetiology, prevalence and the effects of intensification within the industry

Swine fever was a different story (Figure 2). The industry had lived with the disease for many decades before the eradication by slaughter policy began in March 1963, following the confirmation of 1878 cases in 1962. Its lowest ebb was indeed reached in 1949, when only 5 cases were confirmed and, in the ensuing twelve months, no cases were recorded whatsoever. This low incidence was apparently related to the drastic reduction in meat imports from countries where the disease was rife. The same year saw the introduction of the crystal violet vaccination scheme and the subsequent spread of the disease yet again. It is interesting to speculate what the cost of eradication might have been if it had been attempted at this time. The control programme ran 4 years, during which time 1693 herds were slaughtered out at a compensation cost of £5,681,598 (£30 million updated to 1984). Even so, with only 3 more outbreaks of the disease in 1971 (at a cost of £2257), the control policy has been fully vindicated. In terms of cost-effectiveness, the total cost of eradication over the three and a half year period plus the ongoing surveillance costs upto 1975 was £12.3 million (Ellis, 1976). Over the same period, the benefit was assessed at £50 million (an overall benefit:cost ratio of 4:1).

The Americans too have eradicated swine fever (hog cholera). It took them 16 years (from 1961 to 1978) at a cost of £70 million. Prior to this, disease costs were assessed at £25 million per year and thus over the eradication period would probably have amounted to £600 million. Savings were therefore estimated at £530 million, giving a benefit:cost ratio of 8:1 (Atwell, 1985).

Table 1 National disease control programmes 1938-1984.

Disease period	Eradication (years)	Number of confirmed cases	Cost of compensation[α]	1984 equiv.*	Outcome
In pigs:					
Anthrax	46 continuing	13,775	-	-	successful
Foot & Mouth	30 (until 1968)	5,929	£39,545,393	£197m	successful (1 further case 1981)
Swine fever	4 (until 1966)	1,693	£5,997,815	£30m	successful (3 further cases 1971)
Atrophic rhinitis	9 (until 1962)	40	£251,042	£1.5m	failure
Swine vesicular disease	11 (until 1982)	532	£14,575,079	£32m	successful
Aujeszky's disease	2 continuing	490	£24,898,529		successful
In other animals:					
Tuberculosis	35 continuing	208,279	£14,666,873	£71m	successful
Brucellosis	17 continuing	404,299	£83,407,231	£137m	successful
Newcastle disease	17	21,188	£27,486,288	£192m	failure
Sheep scab 1.:	14 (until 1952)	2,021	-	-	successful
2.:	12 continuing	886	-		?
E B L	3	-	£1,806,581	-	-

* Source: Central Statistical Office.

[α] Add on 15% approx. to cover staff input and administration costs.

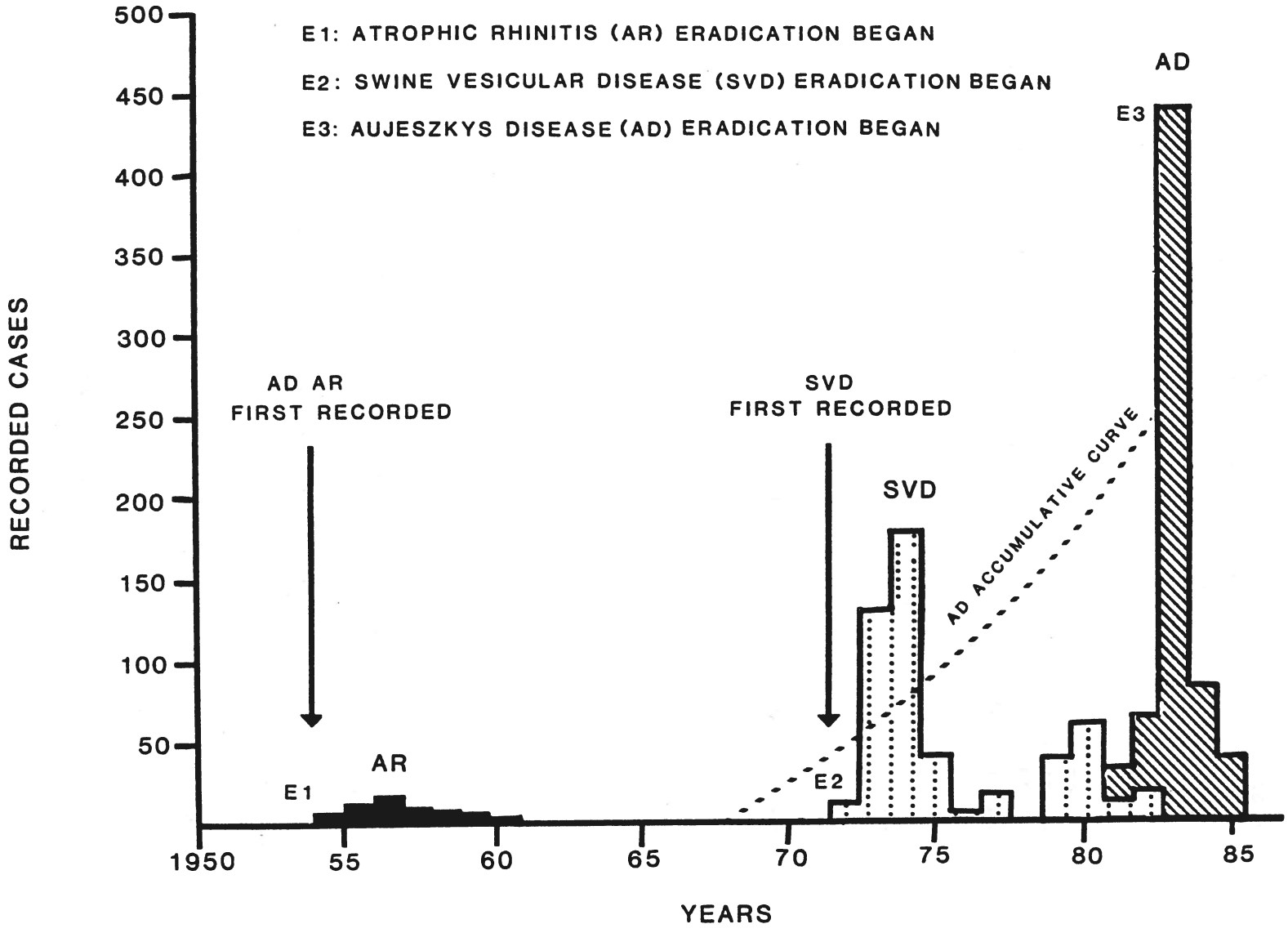


Figure 3. Swine disease eradication schemes 1950-1985.

ERRATUM

Page 9, paragraph 4, sentence 1 should read:

"Table 2 shows that over the thirty years 1955–1984 a total of £203 million were paid out in slaughter compensation, of which £48 million related to compensation for specific pig control programmes."

Finally, the successful pattern of eradication by slaughter, although still continuing, would appear to be repeating itself with regard to Aujeszky's disease. It could be argued that this has been achieved in spite of the protracted delay in implementing the ultimate control. Again, as with swine fever, it is interesting to speculate on the effect this may have on the total cost of the programme (Figure 4).

In 1983, at the start of eradication, there were 170 known infected herds in the country. In the preceding 12 months the disease had spread from 9 to 27 counties. By the end of 1983, 443 infected herds had been slaughtered out and another 11 herds partially depopulated. This was a strong indication that the disease had already entered the escalation phase, evidenced by rapid spread of infection, seen previously and since in countries like Holland and the USA. The sudden upward trend merely mirrored the picture abroad and firmly suggested that some 90% of the national herd would become infected in the period 1983-1993. Recognising that most of the country's pigs are concentrated in about 7,000 herds, some 6,000 of the largest herds might be expected to become infected before the end of the decade. Losses associated with this scale of infection would amount to at least £59.5 million (6,000 herds at £9,920 in estimated losses per herd: MAFF source 1981). To this, of course, must also be added the cost of voluntary vaccination over the same period. If carried out on the same scale as that in Holland, i.e. £3.5 million per year (Oirschut, 1985), another £35 million can be envisaged. This would give a probable loss figure £94.5 million. Set against this must be the actual cost of the programme. To date (1985), some £25 million has been paid out in infected herd valuations, plus a further £4 million on farmer consequential loss (paid out only during the first 6 months or so). To this must be added a further £4 million in bank interest charges incurred by the Pig Diseases Eradication Fund in the interim. This gives an overall total cost of £33 million. Subtract from this the £11 million obtained from the salvaged carcases and we are left with a real outlay of £22 million. Thus, if the trend continues, we can look for an overall benefit:cost figure of about 4:1 (cf. swine fever).

Compensation for consequential loss during the first 6 months, the salvage of carcases from infected herds and the funding of the scheme itself by the pig industry, through a 30 pence levy on all slaughtered pigs (i.e. £5 million per year) - all these things make this national disease control campaign different from its predecessors. If it proves a success, as seems likely, and if the lessons are learned, it could well form the pattern for the future.

Table 2 shows that over the thirty years 1955-1984 a total of £203 million were paid out in slaughter compensation for specific pig control programmes. Of this latter sum, half was paid for by the pig industry in the control of Aujeszky's disease. A more meaningful comparison can be made by bringing all outlays to 1984 values. The revised total sum then stands at £646 million, with specific pig diseases accounting for £78.5 million of that amount.

PREVENTIVE MEDICINE PROGRAMMES

MAFF Pig Health Scheme (PHS)

The government sponsored Pig Health Scheme has been in existence since November 1968. It is a continuous exercise in preventive medicine, originally

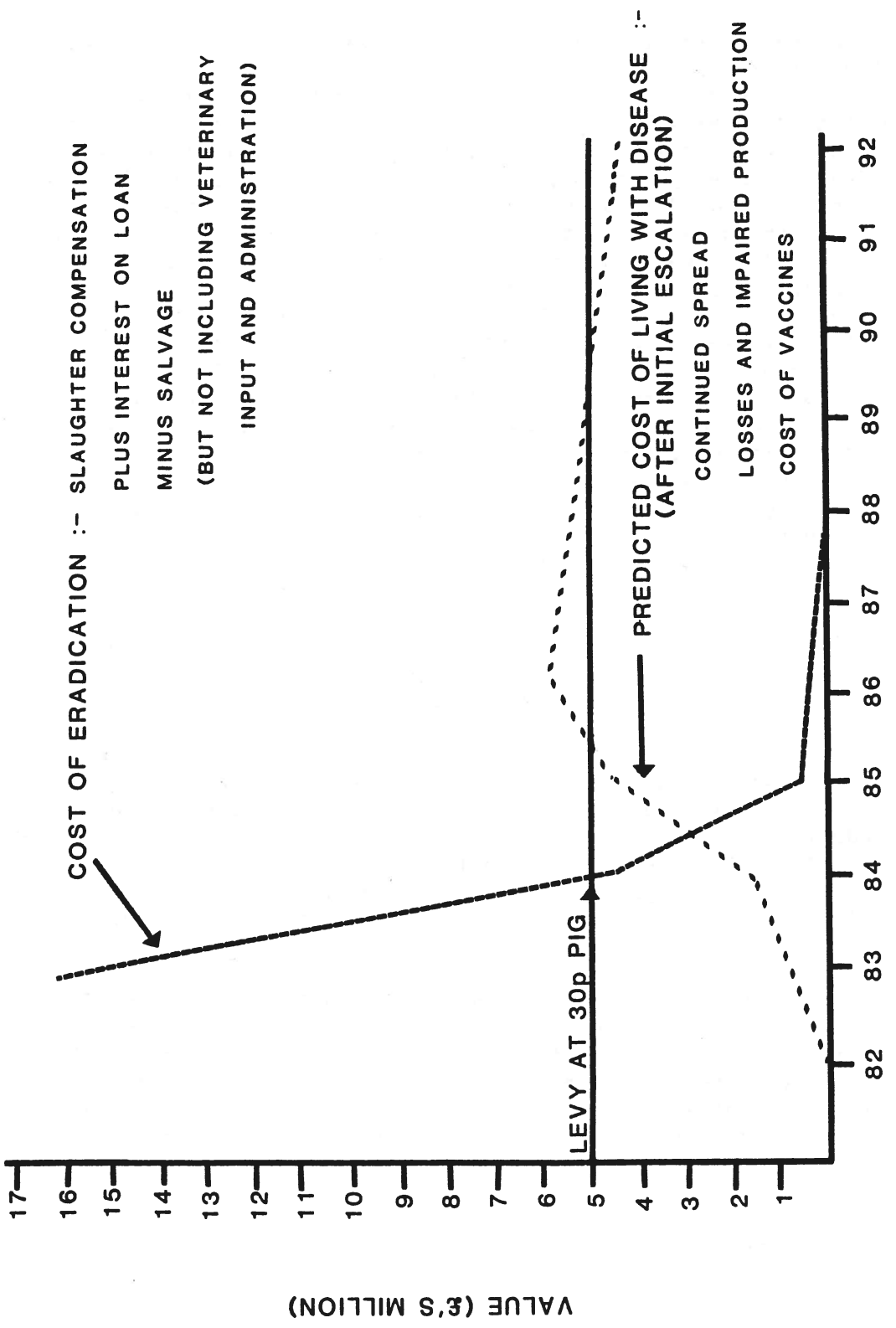


Figure 4. Comparative costs of Aujeszky's disease eradication 1982-1992.

catering for the needs of some 250 Meat and Livestock Commission (MLC) registered herds. These herds already conformed to an approved genetic improvement scheme (the Pig Improvement Scheme: PIS). As one would expect, there is great diversity within the herds regarding both management and size. At the end of 1984, there were 245 herds ranging from 6 to 1562 sow units and accounting for 6.5% of the national sow herd. These herds are serviced by 30 state veterinary officers (PHSVOs) and 170 local veterinary surgeons (Basinger, 1985a,b). Herds can be either independent or company components. The variations indicated have dictated the need for a broad based flexible scheme.

Table 2. Cost of national disease control 1955-1984.

Year	Actual compensation	1984 equiv.		Year	Actual compensation	1984 equiv.	
1955	£1,285,159	£8.5m)		1971	£605,176	£2.5m	
1956	£2,743,051	£19m)		1972	£2,014,431	£8m)	
1957	£2,702,539	£16m)	A.R.$^{\alpha}$	1973	£6,275,454	£25m)	
1958	£3,510,301	£21m)	£250,163	1974	£9,861,469	£35m)	
1959	£5,395,181	£33m)	(£1.5m)	1975	£12,179,282	£30m)	
1960	£7,383,091	£45m)		1976	£16,200,082	£33m)	S.V.D.$^{\delta}$
				1977	£10,783,168	£22m)	£14.5m
				1978	£10,722,116	£19.5m)	(£32m)
1961	£5,488,723	£33m	N.D.$^{\gamma}$	1979	£10,963,699	£15m)	
1962	£9,387,156	£60m		1980	£10,031,783	£12.5m)	
1963	£5,051,424	£30m)				)	
1964	£1,903,289	£12m)	S.F.$^{\beta}$			)	
1965	£867,164	£5m)		1981	£3,292,538	£3.75m)	
1966	£1,293,633	£7.5m)	£,997,815	1982	£2,231,764	£2.5m)	
1967	£24,805,569	£100m	(£30m)	1983	£23,717,239	-)	A.D.$^{\zeta}$
1968	£2,636,861	£12.5m	F.M.D.$^{\epsilon}$	1984	£8,230,536	-)	
1969	£526,810	£2.5m					
1970	£799,532	£3m					
				TOTAL (slaughter compensation):		£203m (£646m)	

Bracketed years are those when a significant amount of compensation resulted from diseases of pigs. (Source: MAFF Animal Health reports 1950-1984).

$^{\alpha}$Atrophic rhinitis compensation over the period. 1984 equivalent in ()

$^{\beta}$Swine fever compensation over the period. 1984 equivalent in ()

Years where a significant amount of compensation resulted from particular diseases are indicated:

$^{\gamma}$Newcastle disease

$^{\delta}$Swine vesicular disease

$^{\epsilon}$Foot and mouth disease

$^{\zeta}$Aujeszky's disease

Aim - this is to control and reduce disease incidence and severity within the member herds, thus enabling full advantage to be taken of genetic improvement, so increasing the profitability of the industry and its potential for pig export.

Membership - the scheme presently is restricted to herds participating in the new MLC genetic improvement programme and other herds, of nucleus or multiplier status, engaged in the sale of breeding stock. A key factor to acceptance is the herd owner's ability to provide such facilities to enable his veterinary advisers to accurately assess the disease/health situation in that herd at any given time. In addition, herds must be free from spreading clinical disease and be serologically negative for Aujeszky's disease at the time of entry. The probationary period lasts for not less than 6 months. If satisfactory, full membership is then granted. No herd is removed from membership soley because of disease problems, faulty management or deficient housing. Only failure to grapple with such problems would occasion revocation. Restrictions on these herds are sparingly imposed and relate generally to spreading disease which might imperil other herds.

Servicing - the scheme is serviced by selected specialist veterinary officers (PHSVOs) and the owners' veterinary surgeons (VSs). Back-up is provided by the Central Veterinary Laboratory, the Veterinary Investigation Centres and other ADAS specialist disciplines. This demands a close working relationship between all interested parties. To date, all input has been provided free of charge; however, some form of charging is scheduled by April 1987. For 18 years, the scheme has been managed regionally, with policy decisions made centrally. Since 1975, a national supervisory function has been exercised by the National Pig Adviser, in charge of the PHS Central Records Office (CRO) at Cambridge. Herd servicing and all other activities are monitored by the CRO and PHS herd/membership lists, and data analysis tables are produced annually.

Primary objectives - these are more numerous than the stated aim would suggest. They account for the differences between the PHS and other collective preventive medicine schemes. They are:

1. To reduce health hazards to MLC testing stations and to prospective purchasers.
2. To monitor disease levels within the herds and to improve on existing situations.
3. To correlate management systems in these herds with disease incidence and severity.
4. To study specific diseases by herd investigations and surveys (and so arrive at national policies regarding them, e.g. Aujeszky's disease and swine dysentry).
5. To obtain and disseminate information and build up professional expertise.
6. To improve the national herd overall.

Thus, apart from direct farmer benefit, the PHS has always had an educational and research role to fulfil.

Function - the main function is to reduce disease incidence to an acceptable level within the member herds. What is acceptable will depend entirely on the disease, its effects on the the herd and its potential threat to other herds. While low grade infections may well be tolerated, at least initially, the presence of insidious and potentially explosive diseases, e.g. Aujeszky's disease, most certainly would not.

The function of the scheme therefore falls into four sequential categories:

1. To prevent disease coming onto the farm by adopting strict security pracautions with regard to persons, vehicles and animals. The provision of an isolation unit is an integral part of this regime.
2. To contain disease already present by reduction of stress and challenge. The nature of each disease will determine whether a policy of amelioration or eradication should be embarked on and what prophylactic measures, if any, should be routinely used.
3. To prevent disease being transmitted to other farms by effective treatment and testing procedures and, if necessary, by imposed or voluntary restrictions.
4. To improve overall production and, therefore, profit.

Farmer benefits - the overriding value of the PHS is the constant herd surveillance it provides. 'Crisis only' visits are replaced by routine veterinary assessments. From the latter stem the programmes, tailor made to suit each unit, for herd health improvement. The components are:

1. A minimum of 4 advisory visits a year, with interim visits as required.
2. Maintenance of accurate herd recording, covering production and disease variables. Individual identification of pigs from three weeks of age consequently is mandatory.
3. Investigations of all herd health problems, with advice tendered on disease control.
4. Critical assessment of management and buildings, established and projected.
5. Routine monitoring for disease on-farm and at slaughter and, particularly, inspection of significant culls and casualties.
6. Full and detailed investigations into all 'sales off ' complaints.
7. Provision of a detailed herd asessment each quarter, which may be used for sales promotion (as can membership of the scheme itself).

Whereas other schemes place emphasis on freedom from specified diseases, the PHS is primarily concerned with establishing herd disease profiles. Accepting that no herd is indeed free of disease per se, it allows purchasers to be selective and to buy safely with respect to their own indigenous health status.

Cost effectiveness - putting a value on prevention, i.e. costing losses that do not happen, is, as mentioned previously, inevitably difficult. The PHS, because of its many objectives, is even harder to assess. An attempt must compare sets of values: actual cost, including staffing, administration and laboratory services; and actual value, in terms of cost of providing alternative health back-up, health contribution to genetic gain (10% of the MLC claim), herd improvement (in respect of reproductive efficiency and post-natal survival) and its educational value to PHSVOs and VSs. Research value and export contribution are impossible to calculate. Nevertheless, recent assessments carried out (MAFF sources 1983) show that the known calculable value of the PHS is so in excess of the known output figures (£4 million and £290,000 respectively) that were the input trebled and the output halved, the scheme would still be cost effective. At that point in time, the scheme was costing about £1,174 per herd or £6.20 per sow. Tables 3 and 4 show production and disease trends in the health scheme in recent years.

Table 3. Comparison of production figures PHS/Ridgeon's Cambridge Management Scheme, 1984 figures.

	PHS Eastern Region*					Ridgeon's Cambridge Management				
Year	1980	1981	1982	1983	1984	1980	1981	1982	1983	1984
No. of farms	58	70	79	77	79	146	151	161	144	151
Av. no. sows	127	133	151	160	150	118	139	146	139	147
Litters/sow/year	2.05	2.11	2.06	2.15	2.17	2.14	2.19	2.21	2.24	2.22
Live pigs born/litter	10.03	10.29	9.95	10.18	10.13	10.3	10.4	10.3	10.5	10.4
Weaners/litter	9.14	8.98	9.03	9.02	9.06	8.8	8.8	8.8	9.0	9.1
Pigs born dead/litter	0.61	0.61	0.62	0.63	0.59	-	-	-	-	-
Pigs/sow/year	18.33	18.74	18.94	19.38	19.85	18.9	19.3	19.5	20.1	20.2
Conception rate of first service (%)	89.7	87.7	87.2	89.3	90.49	-	-	-	-	-
Conception rate to A.I. (%)	82.7	81.4	73.2	80.2	81.42	-	-	-	-	-

*Source: Basinger (1985a,b)

The future

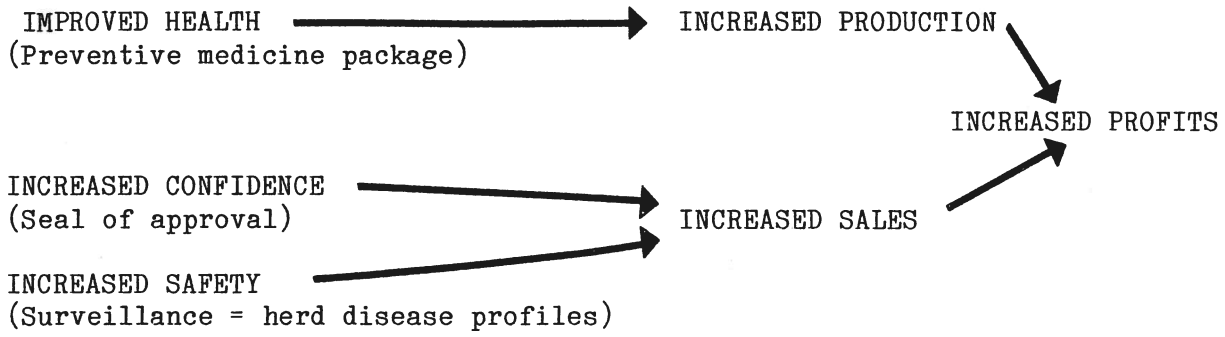
With the termination of PIS in January 1984 and the intoduction of a newer, smaller Nucleus/Supernucleus scheme (approximately 26 herds), the links with MLC have weakened. The principle of charging for state sponsored services, in general from 1987, will also affect the current scheme. Its survival will depend largely on the percentage recoupment of costs demanded by government and what the industry is able or prepared to pay. That a national scheme is still wanted by the pig industry is certain. The loss of the national scheme, if nothing else, would put exporters at disadvantage, compared to competitors in countries where national pig health schemes are not only extant but also are subsidised quite heavily, for example, Sweden (1951), Denmark (1970), Finland (1964), Holland (1965), Bavaria (1962) and Ulster (1972), as well as Switzerland, South Africa and the Canadian Provinces.

Table 4. PHS recorded incidence of disease*.

Year	1978	1979	1980	1981	1982	1983	1984
Number of herds	239	241	236	251	248	247	245
Disease	**Number of herds affected at the end of each year**						
Colibacillosis	118	111	121	83	102	59	69
Mange	131	129	125	91	68	52	68
Enzootic pneumonia	99	89	100	77	63	51	78
Parvivirus 59e	26	30	36	38	45	36	48
Atrophic rhinitis	45	26	28	14	20	9	16
Swine erisipelas	21	12	10	6	9	5	4
Swine dysentry	5	0	3	2	3	1	3
Aujeszky's Disease	1	5	6	5	2	0	0
Streptococcal meningitis	18	21	19	14	2	9	10
PIA	5	4	2	0	0	2	1
Transmissible gastro-enteritis	1	0	1	2	0	1	0

*Source: Basinger (1985a,b)

Basis for a revised Pig Health Scheme



There are two requirements of a 'national, scheme. The first is a valid declaration that herds are indeed members of a nationally backed and recognised scheme (this should induce charisma,impartiality and bigger laboratory back-up). The second is an equally valid herd health assessment programme based on regular on-farm, slaughterhouse and laboratory monitoring. The health base-line, and any variations above that level, will largely be dependent on purchaser herd requirements at home and abroad. The need to impose a specific pathogen free system on all members would probably be neither necessary nor desirable. The prime necessity, as in the current scheme, would be the establishment and maintenance of meaningful herd disease

profiles for all herds.

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PREVENTIVE MEDICINE SCHEMES FOR PIGS : PAST, PRESENT AND FUTURE

T.W. HEARD*

There is a little known book, published in 1920, in a collection called the Smallholders Library, by C.A. Pearson Ltd., entitled "Pigs: How to make them pay." In Chapter IX, headed "Health in the Piggery," the following two paragraphs are worthy of repetition:-

HOW DISEASE IS SPREAD

> "There are so many ways of carrying disease that the pig-keeper who has the advantage of breeding his own stock must score a decided advantage and largely minimise his risks.
>
> We have heard of disease even being carried by miller's sacks, which, when emptied, were in a certain case thrown on a spot near the piggery. Being returned to the mill and filled again, they became a very effective means of spreading disease in several places. Indeed, we have it on the authority of responsible parties that in a certain district it was only by insisting on the wholesale destruction of a large number of sacks that the spread of disease was checked.
>
> Rats are another source of spreading disease. These rodents will travel a long way to the pig trough, and in our opinion have have often been the means of carrying disease. Cold, damp sties or, on the other hand, close, low, stuffy, over-warm pens are a certain cause of cold. Fogs in the autumn and cold east winds in the spring are dangers to be looked for and guarded against. In such cases, good food and nursing are requisite."

TO WARD OFF DISEASE

> "The most successful preventive measures for all diseases will consist mainly in sedulously avoiding contagion through bringing in animals from districts in which disease has made its appearance, or even allowing persons from such districts to come in contact with healthy pigs. The pig pens should always be kept perfectly clean and frequently lime-washed, and a small proportion of strong carbolic acid should be mixed with the latter. If disease should appear in a district, carbolic acid in larger quantities and increased cleansings should be the order of the day."

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Not only does this infer that the pig was just as abused in the "good old days" as it is suggested that it is today, but that the basic requirements of good, preventive medicine techniques were well understood even then.

Certain aspects should be particularly noted:-

1) The concept that disease is spread.

2) That disease can be carried by inanimate objects such as sacks.

3) That vermin are capable of transmitting disease, (e.g. Swine Dysentery or Aujeszky's Disease).

4) Be wary of cold, dampness and stuffiness.

5) Control of animal movement, as well as contacts, is necessary.

6) Disinfection and cleaning helps to prevent disease.

By 1920 a number of substantial preventive medicine schemes had been catalogued. After the discovery of Foot and Mouth Disease in August 1839 at Stratford, London, the disease rapidly spread so that by 1870, 52,164 outbreaks had been recorded. This catastrophe triggered the forming of the Contagious Diseases (Animals) Act of 1869, and it took many years to get the problem fully under control.

Since then Swine Fever, Atrophic Rhinitis, Rabies, Anthrax, Salmonellosis, Swine Vesicular Disease and, more recently, Aujeszky's Disease. have been added to the list of diseases being controlled by state intervention. The principles behind these controls are similar and can be summarized as follows:-

1) Diagnosis.
2) Prevention of spread by quarantine, movement controls, or slaughter.
3) Elimination of infection by slaughter, vaccination, natural resolution. or medication.
4) Cleaning and disinfection of premises and monitoring of residual stock.

While all this government sponsored activity was preventing the spread of disease within the boundaries of these islands, strenuous attempts were maintained in order to keep out new infections which were likely to be imported from other countries. Import controls, port inspections, international treaties and agreements, product and population monitoring and intelligence gathering are all the tools used in this procedure.

At home on the farm a new form of preventive medicine was evolving. (Heard 1969). The origins of this evolution were the Specific Pathogen Free pig movement from the USA, the Ontario Department of Agriculture schemes in Canada, and two projects that originated from the School of Veterinary Medicine at Cambridge, UK. These last two took the form of:-

1) A Certification of freedom from Enzootic pneumonia scheme run by the Pig Health Control Association.

2) The availability of a Hysterectomy Centre which operated on pregnant sows and produced "Minimal Disease" piglets for prospective farmers to rear.

It is interesting to note that the Pig Health Control Association still exists in 1986, with 50 participating herds, and with certification programmes for six diseases:-

1) Enzootic pneumonia.
2) Atrophic Rhinitis.
3) Aujeszky's Disease.
4) Mange.
5) Swine Dysentery.
6) Streptococcal Meningitis.

The Hysterectomy Centre did not survive, although some of the herds it helped to establish were responsible for the development of many of the largest UK pig breeding companies, including the Pig Improvement Company and the Northern Pig Development Company.

The relevance of these procedures to preventive medicine is substantial, for the ability to test for the presence of a disease on a continuing basis. and the production of animals free of certain diseases by hysterectomy have produced herds which have advantages in growth and feed conversion efficiency. It must be stated that there is also a substantial improvement in the pigs' welfare when freed from the effects of such diseases.

In order to keep these pigs healthy, barriers have been constructed around farms to keep the pigs in, and other animals, men, vermin and dangerous objects, out. These barriers need not necessarily take the form of a physical fence, geographical features such as hills, rivers, or urban communities serve as well, if not better. Perhaps the most important feature of all is the provision of a poorly accessible site.

The UK pig industry encompasses many farms that are free from the scourges of a number of diseases such as:-

Mange,
Swine Dysentery.
T.G.E..
Epidemic Diarrhoea,
Enzootic pneumonia,
Atrophic Rhinitis,
Streptococcal Meningitis,
Lice.

They maintain this freedom by preventing infection from entering their premises, and they only purchase replacement pigs from sources of known similar health status. (Heard 1978). "Known" is the key word in this sentence, and a great deal of effort is put into assessing the health status of a herd. Current veterinary knowledge makes use of production records, clinical inspection, post-mortem examination, serological testing and tissue and faeces sampling. The future will see increasing reliance

on the use of slaughterhouse material and the development of serological tests that can identify the presence or absence of many infections.

Responsible herds may use a health data report form, such as the one shown in Fig. 1, to record the presence and absence of disease. The advantage of this format is that it allows the criterion for freedom from infection to be set by the signatory, and by this process complete flexibility is available.

FIG. 1

PIG HERD HEALTH STATUS REPORT

Farm: Date:

Condition	Test performed	Result
Swine Pox	.....	
H. Pleuropneumoniae	.....	
Epidemic Diarrhoea/TGE	.....	
Aujeszky's Disease	.....	
Vomiting and wasting disease	.....	
Enzootic Pneumonia	.....	
Parvovirus/Smedi	.....	
Cl. Infections	.....	
Dysentery	.....	
Atrophic/Infectious Rhinitis	.....	
Streptococcal Meningitis	.....	
Erysipelas	.....	
Adenomatosis	.....	
Greasy Pig Disease	.....	
Internal Parasites	.....	
Mange/Lice	.....	
Any other health problem	.....	

The above results are my assessment of the situation within the limitations of the tests performed.

Veterinary Surgeon

I have dwelt on these aspects in some detail as they are central to current preventive medicine beliefs. The approach can be summarised as follows:-

1) Know what diseases are present in the pig population.

2) Ensure that a farm barrier separates the population from external health problems.

3) Purchase stock of compatible health, from reliable sources.

4) Control and eliminate the health problems in the controlled population.

The key to success is the provision of an adequate barrier around the pigs, and continued monitoring of the health status of the pigs involved. The barrier has to be mental as well as physical, and a high level of motivation has to be instilled in stockmen, farm staff and owners.

Some of the features used can be listed:-

1) Chain link fencing.
2) Locked gates.
3) Loading bays.
4) Separation of delivery lorries from pigs.
5) Separation of dung removal activities from pig area.
6) Showering and changing requirements for staff.
7) Monitoring of health status of purchased feed and water supply.
8) Bird-proofing and rodent control.
9) Isolation of site with difficult access.
10) No pig purchases, save from monitored sources, or by hysterectomy-type procedures.

These techniques are designed to keep diseases out and the procedures are well-known. This leaves the problem of controlling and minimising the effect of diseases present within the herd. The economic effect can be substantial.

The improvement in productivity resulting from 50g per day extra growth over 150 day fattening period is, at 1986 prices, £7.50 a pig, or £ 15,000 for 100 sow herd rearing its progeny to bacon weight. Many producers, with the help of their veterinarians, are successful in gaining this type of advantage. They have gained it by eliminating Swine Dysentery, by negating the effects of Parasitic Worms, by controlling Mange, and by vaccinating against diseases such as Swine Erysipelas, Parvovirus and Haemophilus pleuropneumoniae. With commercial solvency of continuing importance to many producers many of the schemes being implemented are not reported in the literature, even though they are practised on a widespread basis.

The techniques used in these preventive medicine schemes may be of some interest.

Swine Dysentery

By herd medication, using a variety of products active against Treponema hyodystenteria, it has been demonstrated that infection can be suppressed to such an extent that cross-infection is prevented. When the herd has been medicated for a sufficient length of time for the fattening animals present in the herd at the commencement of the medication programme to have been slaughtered, it will normally be found that the new generation of animals that has taken their place are free from the disease.

In 47 herds where such a programme has been carried out by the author, 80% have been successful after the first attempt, 23% after the second, 30% after the third, whilst only 2 herds (4%) have failed to achieve success.

Parasitic Worms

In-feed worming procedures continued over a period of 7 to 14 days produce a population of pigs that have no worm eggs in their faeces, and where the chances of ingesting infected worm eggs are remote. The worm free faeces have acted as a "cleansing" agent and swept worm egg contaminated faeces out of the pig sty. By repeated treatments given to coincide with the first appearance of worm eggs in the faeces, the worm burden in the population can be kept extremely low, and worming treatments will only be required occasionally. The author has calculated that when using Thiophanate for 14 days (Nemafax 14) in 119 herds the interval between treatments after three initial treatment periods had stretched to 283 days.

Mange

When establishing a new herd on a clean site the use of the products Phosmet (Porect) or Ivomectin (Ivermectin) before the pigs are moved to the site, and two weeks after they arrive, will eliminate sarcoptes scabeii from that population. This is a most economical and effective method of establishing a mange free herd. Future work will no doubt concentrate on removing mange from existing herds.

Vaccination programmes

Some vaccination programmes are more effective than others and in many cases the criteria of success is not the vaccine, but the timing of its use. Vaccination of the adult breeding herd against Swine Erysipelas on a regular twice-yearly basis is one such technique that has stood the test of time. Annual vaccination of breeding stock with Parvovirus vaccine (Suvaxyn Parvo) looks as if it will be similarly successful, and the use of Haemophilus pleuropneumoniae vaccines in rearing animals has been shown to be an effective method of controlling the economic effects of this disease.

My own organisation has been active in a number of these control programmes. Salmonella control was reported by Heard, Jennett and Linton (1968). Mange control by Heard (1968), Swine Dysentery eradication by

Tasker et al (1981). Many other projects have been published. Muirhead (1984) published work on the use of computers to study the epidemiology of diseases on the farm, and Alexander (1981) described a medication technique (MEW) for establishing specific pathogen free populations of pigs.

With all this activity about us, some might be tempted to feel complacent, but they would be ill-advised to do so. The wide diversity of the subjects mentioned suggests that there exists considerable fragmentation of purpose. This fragmentation exists because many individuals are trying to solve the problems that confront them in their local environment. What we must seek for the future is the development of national and international data bases where health problems and health status, together with their economic cost, can be collated.

The economic cost of Streptococcal meningitis to the pig farm is low, despite the spectacular nature of the disease. The aborting sow carries a small economic penalty compared to the herd where the farrowing rate has dropped to 80% Many farms, some with the help of veterinary advisors, have now installed computers which can record the prevalence of disease, as well as the cost of production and the level of output. Relating this data to disease control is the coming challenge for the veterinary surgeon. Eliminating mange from a 100 sow breeder/feeder herd might cost £600, but could be worth £2000 a year. Eliminating Swine Dysentery is even more cost effective. The continuing control of worms, such as Ascaris, the cause of Milk Spot Liver, is a normal requirement for any progressive farm. Vaccination programmes for the control of Erysipelas and E. coli have long been accepted techniques. More recently, Parvovirus has been added to the list, and future development of vaccination programmes against Haemophilus pleuropneumoniae, Pasteurella and Clostridium perfringens will become very relevant. All of these programmes need to be evaluated within the national and international context so that we can accurately cost the benefit of the procedures envisaged. For example, a simple routine, such as "all in - all out" piggeries, with cleaning, disinfection and fumigation carried out between batches has never been properly evaluated, and the benefits are largely a matter of speculation. It is ironic that although the Smallholders Library in 1920 was well aware of the procedure, today we still cannot cost it for our producers.

SUMMARY

In the past, preventive medicine has centred on the grand national schemes for disease control. The development of specific programmes for health monitoring, the introduction of specific pathogen free barrier-maintained herds has allowed the establishment of populations of pigs freed from the ravages of many common pig diseases. The use of advanced diagnostic techniques, computerised data gathering and economic analyses will transform the world of future preventive medicine by selecting the diseases that can be most effectively controlled. The UK pig industry would be well advised to invest in a national data gathering programme to further these objectives and to base its health research effort on the diseases that are prevalent, economically expensive, and which are likely to be controllable, if it is to stay abreast of the competition.

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THE PROGRESS AND ECONOMIC BENEFITS OF A MASTITIS CONTROL PROGRAMME

R.W. BLOWEY*

The monitoring of herd and flock performance using numerical indices has now become an integral part of preventive medicine programmes. Records are used not only to assess whether a problem exists, but also in the analysis of that problem (Blowey 1984; Blowey in press). This paper describes the progress of a mastitis control scheme, including the importance of records and their analysis. Possible causes of the reduction in mastitis incidence are discussed and an attempt is made to quantify some of the financial benefits produced.

Collection of Data: Since 1979 the incidence of mastitis has been recorded in a group of 22 herds associated with a West Country veterinary practice. Herds were supplied with a small, loose-leaf booklet, in which each cow which developed mastitis was allocated a separate page. Date, quarter(s) affected, treatment used and results of bacteriology (if carried out) were recorded. Details of this recording system have been described in a previous paper (Blowey 1983). Twice each year the books were recalled from the farms and subjected to a simple manual analysis, from which the basic criteria of mastitis incidence were extracted. An example of the herd analysis covering a 12-month period is given in Table One (Blowey 1984). As with any field survey of this kind, and particularly one run as part of a general practice, there are many sources of possible error in the data. For example, during the period of recording some herds were dispersed and others, for a variety of reasons, discontinued their recording. Not every farm submitted its book for analysis every six months and although this was, fortunately, relatively infrequent, the 'league table' of results for the remaining herds was not necessarily held back while awaiting the data from the final participating herds. A few new herds joined the recording scheme, replacing those which had discontinued. Overall, however, the total number of herds being recorded remained constant at approximately 22. Tube usage for a given 12-month period was calculated from the sales invoices and this was related to the number of cases of mastitis recorded (Table One: tube usage per cow and per case treated). This gave some check on the accuracy of the data, because if tube usage per case treated became extremely high, e.g. an average of eight tubes per case or above, then it is possible that the farmer concerned was not recording all the cases of mastitis which occurred.

Results: Table Two shows the variation in herd size and performance for the

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Table 1. A 'league table' derived from mastitis data analysis.

Herd number	Number of cows	Rolling mean cell count ($\times 10^3$)	Mean herd yield (annual sales) (litres)	For whole herd				For mastitic cows		
				% cows affected	Cases per 100 cows	Tubes used per cow	Tubes used per case	Average number of cases	% which recurred	Quarters per cow
1	65	321	5800	38	60	3·5	5·7	1·6	15	1·4
2	81	745	6000	21	30	0·9	2·9	1·5	12	1·3
3	46	275	5850	19	40	4·1	9·4	2·2	45	1·2
4	134	267	5178	21	30	1·2	4·6	1·2		1·2
6	95	346		29	70	9·8	13·5	2·5	33	1·6
7	110	333	6500	27	70	2·7	3·8	2·6	40	1·6
8	132	272	6036	28	60	1·8	2·9	2·2	29	1·6
9	50	441		30	70	2·1	3·1	2·2	36	1·4
10	60	210		18	60	1·7	2·8	3·4	35	2·2
11	110	314		25	50	1·3	2·3	2·2	41	1·3
12	80	456		34	40	1·9	4·6	1·2	3	1·1
13	107	264	7121	4	5	0·2	3·8	1·2	20	1·0
14	123	252	6000	20	40	3·3	8·6	1·9	15	1·6
15	110	557		35	60	4·6	7·7	1·7	20	1·3
16	116	305	6127	27	50	1·4	2·9	1·7	16	1·5
17	95	668		20	30	3·5	11·6	1·5	14	1·3
18	127	236		22	40	1·7	3·8	2·0	36	1·3
19	105	217		30	70	4·1	6·0	2·2	28	1·6
20	65	304	5847	45	50	2·3	4·2	1·2	–	–
21	88	323	5097	29	80	2·6	3·3	2·6	34	1·7
22	110	332	7090	44	90	2·5	2·8	2·1	22	1·6
23	100	177	5500	18	30	0·6	2·2	1·5	15	1·3
·Mean		346		26·5	51	2·6	5·1	1·9	25	1·4

[From Blowey (1984)]

Table 2. The average performance of herds being monitored for mastitis

Periods of Records	No.herds being recorded	Herd Size	Rolling Mean cell count ($x10^3$)	Annual milk sales (ltrs)	% cows affected	Cases per 100 cows	Tubes used per cow	Tubes used per case treated	% cases which recurred
Oct '79-Sept'80	22	87.8	346	6011	26.5	51.0	2.6	5.1	25.0
March'81-Feb'82	22	101.0	310	5921	25.8	45.9	2.4	5.6	14.9
April'83-March'84	16	123.8	255	5651	23.6	42.7	1.9	4.2	13.1
April'84-March'85	23	11.5	243	5479	19.6	31.7	2.1	7.1	10.3

period September 1979 to March 1985. Each set of figures consists of a full twelve-month recording period and gives the mean performance of the herds being recorded at that time. The figures indicate that there has been a gradual, but definite, decline in mastitis, although 1982/3 the results obtained were similar to the initial three-year mean performance. A similar trend is seen in each of the indices used to express mastitis incidence. (The precise definitions of the terms used are described in a previous paper, Blowey 1984). The proportion of cows in each herd affected by mastitis during a twelve-month recording period has fallen from 26.4% to 19.6% and the case incidence from 51.0 to 31.7 cases per 100 cows per year. The rolling mean cell count has also decreased, from 346 to 243 x 10^3 per ml. A correlation between subclinical mastitis incidence and cell count has been described by other workers, e.g. Wilson and Richards (1980) and the current figures reflect a decrease in clinical mastitis paralleled by a falling cell count. In addition, the proportion of cows which required a second or subsequent treatment during a twelve-month period fell from 25% to 10.3% of first treatments.

The overall decrease in antibiotic tube usage (milking cow preparations only), from 2.6 to 2.1 tubes used per cow per year, was less than expected and this was associated with the very high tube usage per case treated (7.1) in the final recording period (Table Two). This latter figure was elevated by three individual herds, all of which had a very high tube usage (11 to 13 tubes per case treated), partly due to the purchase of a large quantity of intramammary preparations at the end of the recording period. However, the possibility that the three herds concerned did not record all the cases of mastitis which occurred should not be overlooked.

Discussion: The results indicate a considerable decrease in mastitis incidence over the six-year period 1979-85. It is not possible to be sure of the cause of this reduction in mastitis incidence and it is likely that several factors are involved. Firstly, we would consider that the specific mastitis advisory visit paid to the farm once or twice each year must be having a significant effect. The details of the work carried out at this visit have been described in a previous paper (Blowey 1984). Secondly, the simple discipline of recording is in itself likely to be beneficial, in that it increases the general awareness of mastitis and identifies those persistant recurrent mastitis cases which need to be culled. Thirdly, the level of yield may have had an effect, either directly or indirectly, in that higher levels of yield may have been achieved by higher levels of concentrate feeding. This in turn could have led to a greater incidence of fatty liver and/or acidosis, which may have decreased the cow's resistance to infection (Reid 1982/3). Table Two certainly indicates that there was a steady decline in average yield over the period of recording, although nationally yields increased by 5.0%, from 4715 to 4950 litres over the first three recording periods shown in Table Two (UK Dairy Facts & Figures (1984)). The greatest improvement in mastitis incidence was seen in 1984/85. It is highly likely that this was associated with a more rigorous culling policy which was the result of the need to reduce herd production by 9% following the imposition of the EEC Milk Quotas in April 1984. Older cows and those which have had mastitis are likely to have been among the first to be culled. Table Two shows that there was a reduction in both herd size (number of cows) and herd production, although milk yields had been falling steadily throughout the six-year period.

Estimates of the cost of mastitis vary. Figures extrapolated from the data of Wilesmith et al (in press) give the average cost of a single case of mastitis as £50, whereas using estimates from our own practice (Blowey in

press) a figure of £38 per case is obtained.

Taking £38 to be the cost of an average case of mastitis, the financial saving for the herds concerned was considerable. Case incidence fell by almost 20 cases, from 51.0 to 31.7 cases per 100 cows per year (Table Two) and this represents a reduction in losses from mastitis of 19.7x£38=£733 per 100 cows per year. Almost all the herds concerned were implementing standard control measures (teat dipping, dry cow therapy etc.) prior to the start of the recording scheme and hence the only additional expenditure which they incurred was related to the veterinary advisory visits. On average, a herd is visited probably one and a half times each year, with a cost per visit (travel and professional time) amounting to approximately £40. This gives an annual cost of £60 per year to produce a saving in mastitis of £733 per year, which is clearly a sound investment. However, as noted earlier, we do not consider that veterinary input was the only cause of the reduction in mastitis incidence. As far as we were aware, the practice acted as the only source of regular advice on mastitis control; none of the herds concerned participated in the MMB technician service.

The results from this six-year monitoring and the financial benefits obtained have been discussed with the farms concerned and have been used at 'client evenings' to show a wider audience the advantages to be gained from controlling mastitis. Only by producing such data are we likely to maintain the farmer's enthusiasm for continuing with the control programme and we hope that this will be of long-term benefit to both the farms and the practice involved.

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PREVENTIVE MEDICINE SCHEMES FOR POULTRY

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I have been asked by your Conference organisers to look at preventive medicine as applied to a broiler and a layer enterprise. Many of the details are the same in both types of operation. There are some 465 million broilers and 34 million layers in the U.K. Preventive medicine schemes must consider as a total package the size of the site, the geographical situation, house design, security, hygiene, environmental control, chick source, nutrition, as well as the administration of medicaments and vaccines.

Since the end of the second world war, and more especially since the early Sixties, there have been greater changes in the methods of keeping poultry than most other types of livestock. The Poultry Industry led the way in intensiveism, succumbed to many of the problems and has mastered some of them. It is not uncommon to have up to 30,000 birds in one house and 300,000 birds on one site, although the average is somewhat less than this. However, the tendency is towards large sites, even though productivity tends to fall with the increase in size. This is due to increased disease incidence, not necessarily with obvious clinical symptoms, behavioural stresses amongst the birds and reduced manpower. Birds like other stock are responsive to the effects of management.

HOUSING

New sites should be as far away as possible from existing sites, possible new developments and wild bird migratory routes. They should ideally be sheltered from prevailing winds to avoid ventilation and insulation problems. Sites should be situated away from roads down which lorryloads of manure from other sites pass or along which poultry are transported to processing plants. They should not be situated near any geographical feature or physical objects that would impede ventilation nor access by vehicles.

The house design has a great influence on the efficacy of ventilation at bird level and the ease, and hence the efficacy, of terminal clean out. Smooth, uninterrupted, internal finishes to well insulated walls and ceilings are important. The inside of bulk feed bins and overhead water tanks should be capable of being cleaned.

Site security necessitates restricting visitors and lorries to a minimum. Protective clothing should be provided for visitors. Some poultry breeders have installed showers. Ideally food should be blown over a perimeter fence to avoid lorries going onto the site. All culled or dead birds should be collected by outsiders from the site entrance, not from individual houses. The houses should be vermin and wild bird proof.

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Hygiene during the flock's life, providing clean feed and water, removing dead birds and obnoxious fumes, is just as important as inter-batch clean outs.

Experience, often economically painful has shown that when keeping large numbers of poultry together, the best results are obtained by adopting the "all in" "all out" management system. This means that following the completion of a flock's life the site is depleted of all birds; the houses and equipment are thoroughly cleaned, washed, repaired, disinfected, fumigated and kept empty for several days before being refurnished and before any stock is brought onto the farm. It takes about 3 weeks to empty, clean out and refill most sites. This leads to the elimination or at least a great reduction in the number of any pathogenic organisms. After the first few weeks, birds on such sites, have a similar level of immunity to the various diseases which allows them to respond to vaccination, uniformly. With mixed ages some birds do not respond as they receive vaccines at the wrong time.

The majority of broilers, fattening turkeys, ducks and replacement laying birds are now reared on "all in, all out" sites.

The alternative system is a continuous production, multi-age site, where only individual houses are depopulated and cleaned at any one time. This allows on-site infections to be continually recycled through young susceptible birds, for example Marek's Disease, the respiratory viruses of Newcastle Disease, Infectious Bronchitis and Infectious Laryngo-tracheitis as well as bacterial infections including Mycoplasma, Pasteurella and Salmonella.

Large laying units have become an exception to this requirement for an "all in, all out" policy because of the commercial necessity to have an even production of eggs of various sizes available throughout the year. These sites are restocked with 18/20 week old birds that have been fully vaccinated against the prevalent diseases.

Good environmental control becomes more important where large numbers of birds are kept in so called controlled environment houses, where respiratory problems can be greater than when small numbers of birds are kept extensively. It is vitally important that these houses are provided with alarm systems that if anything goes wrong, with the ventilation system, action can be taken immediately. Birds soon die when the temperature and humidity rise within a house. It is essential to provide fresh air to the birds and to remove water vapour, heat, carbon dioxide and ammonia. This must be done at bird level, which can be difficult, amongst these large concentrations of birds, unless the ventilation system is adequately designed for that particular house. Ventilation is the most misunderstood, often badly designed , wrongly installed and misused facet in the management of so called controlled environment houses. The air circulation in the house must be uniform otherwise birds tend to collect into certain areas and thus compound stocking problems. Many people over restrict ventilation in order to maintain the temperature of the house and improve feed conversion. This can be extremely detrimental to the birds who should always be given a minimum of 3 cubic feet of fresh air per second, per tonne of

food eaten per day. Poor ventilation is the cause of many respiratory problems in broilers and egg production or egg quality problems in laying flocks, during the winter months.

House insulation together with ventilation has a great bearing on the state of litter in deep litter houses. A minimum of 6 inches of litter should be placed on a damp-proof concrete floor surrounded by insulated walls. Uniform temperatures and air movement help to maintain good dry litter conditions and an even distribution of birds over the floor. Drinking points should be managed to avoid splashing and leaks. Maintaining the litter helps to avoid high levels of ammonia which at 40 p.p.m. can aggravate respiratory problems. Dry litter reduces the possibility of heavy coccidial challenges.

Where birds are kept in cages there are fewer problems with ammonia but the same concepts apply to the ventilation requirements.

SOURCE OF CHICKS

Obviously the source of day old chicks is very important. These should be obtained from a clean hatchery whose stock is free from Mycoplasma gallisepticum and Mycoplasma synoviae. All the commercial breeding stock and hatcheries in U.K. are free from the species specific Salmonella pullorum and gallinarum. They should also be free from the other serotypes though this may not always be so as far as the more exotic serotypes are concerned. The parent stock from which the chicks are derived should have been vaccinated against Newcastle Disease, Infectious Bronchitis, Avian Encephalomyelitis and Gumboro Disease to ensure that the chicks are protected by maternal antibodies for the first few weeks of life.

NUTRITION

The provision of a balanced, well formulated, diet is essential and enables birds to grow, withstand stresses, overcome infections and react optimally to vaccinations. Vast increases in performance have occurred over the past two decades, due to the improved genetical material, diet and management. Forty-nine day old broilers in 1964 weighed 2.6 lb; in 1985 they weighed 4.8 lb. However, the production of "least cost formulations"; human errors in the mills; faulty storage of some raw materials; lead to varied problems on the farm. These include reduced production, wet litter, deficiencies and toxicities, the exact cause of which can be very difficult to detect.

All that I have mentioned so far is applicable to all species of poultry in all types of situation.

I now move on to specific medication and vaccination schedules for broiler and laying flocks. These are tailored to possible disease challenges in the area of the farm. Many are given for the owner's peace of mind as an insurance against diseases that may occur with catastrophic results.

MEDICATION

Preventive medication involves the use of anti-coccidial, anti-histomonad and antibacterial treatments in the feed.

All birds reared on litter require some anti-coccidial medication in order to control the Eimeria parasite and prevent mortality due to an acute outbreak and/or poor growth performance caused by subclinical infections. Birds reared in cages are not given an anticoccidial. There are many different drugs available. The most efficient, widely used ones in the broiler industry belong to the Ionophore group of compounds - Lasolocid (Roche), Monensin (Elanco), Narasin (Elanco), Salinomycin (Hoechst). They are given on a continuous basis from day old to within 3-5 days of slaughter. Improved growth and feed conversion equivalent to double the cost of the drug are recorded. It costs about £2.70 per tonne for their inclusion. 450 million broilers consume 2 million tons of feed including £5.4 million worth of coccidiostat!! There are other, slightly cheaper, anti-coccidial drugs but they have all run into resistence problems. An anti-coccidial vaccine is on the horizon, developed from attenuated strains of all the main Eimeria to which broilers are susceptible - 5-6 species. This may be cheaper!

Anti-histomonad (anti-blackhead) preventive medication is mainly used in turkey flocks, although it is occasionally used in rare replacement pullet deep litter sites that have a problem with Blackhead. When preventive medication is necessary either Dimetridazole or Nifursol is fed on a continuous basis from day old through to caging on the laying site. Blackhead is one of the developing disease problems being experienced by those farmers who have started keeping laying birds on free range systems.

It costs about £5.00 per tonne to medicate with Dimetridazole. A pullet eats 8 pounds of feed from day old to caging at 18 weeks. An 18 week old pullet is valued at £2.15. So before using any anti-Blackhead medication one has to be certain to save 3 birds per tonne of food used. Any effect on egg production is much more complicated to ascertain.

Similar calculations have to be done for those sites that require routine preventive antibacterial medication. These are mainly multiage broiler sites that continually run into problems with E. Coli septicaemia at 4 - 5 weeks of age. Again the cost of medication must be set against the possible benefits. It costs about £7.00 per tonne to medicate with a therapeutic dose of Furazolidone and about £8.00 per tonne with Chlortetracycline, the most commonly used drugs. It costs about 23p to produce each live weight pound of broiler - £1.10 per bird.

VACCINATION

Vaccination programmes are designed to prevent or reduce mortality and morbidity in the vaccinated birds, their progeny or both. They are designed so that a number of live or dead vaccines are administered at suitable intervals during a relatively short period of time.

General consideration:

Programmes are many and varied depending on geographical location, possible disease challenge and the type of stock to be vaccinated. Different vaccines are available in different countries.

In devising a programme, one must consider:-

(a) The general health of the flock, - the effect of concurrent disease such as Mycoplasmosis and the local pattern of disease. It is obvious that vaccination is required only for those diseases that are likely to challenge the flock. Although vaccination must be regarded as an insurance this can be taken to extremes when gullible farmers are persuaded by very persuasive sales people to vaccinate for everything possible. It is not unknown for turkeys to be given Infectious Bronchitis vaccine when they do not succumb, as a species, to this viral infection. Flocks have been vaccinated for Infectious Laryngotracheitis when there was none nor had there been any in the area.

(b) The benefit of vaccination against potential loss.

(c) The influence of vaccination or disease in the previous generation on the maternal antibody status.

(d) The availability of labour and the practicality of a particular method of vaccination in relation to the design of the house and the position of equipment within it.

(e) The possible need for rapid protective cover of the birds, for example in an epizootic, and also the danger of reaction to the vaccine, which can have disastrous commercial consequences.

The following vaccines are available in this country:-

Live attenuated vaccines:	- Newcastle Disease - "HB1"
	- Infectious Bronchitis - "H120"
	- "H52"
	- Infectious Bursal Disease (Gumboro)
	- Marek's Disease
	- Avian Encephalomyelitis (Epidemic Tremor)
	- Infectious Laryngotracheitis
	- Pox
Inactivated Bacterins	- Pasteurella multocida
	- Erysipelas rhusiopathiae
	- Haemophillus gallinarum
Inactivated oil based vaccines:	- Newcastle Disease
	- Infectious Bronchitis
	- Infectious Bursal Disease
	- Egg Drop Syndrome and combinations of the above.

There are several methods of administering these vaccines:-

Live Vaccines:	-	Eye drop
	-	Coarse Spray
	-	Aerosol
	-	Drinking water
	-	Injection
Inactivated Vaccines:	-	Injection of individual birds.

The eye drop method is self explanatory. Its great advantage is that each bird receives a full dose of vaccine but it is very labour intensive.

Coarse sprays are now used only for day old vaccination, either in the hatchery or on the farm. The object with the eye drop and coarse spray is to ensure contact between the vaccine virus and the Harderian gland in the medial canthus of the eye. 1,000 birds are given 1,000 doses of vaccine in half a litre of water, using a commercial pressure sprayer (standard garden equipment).

Aerosol sprays are used with older birds, 1,000 doses being distributed via the aerosol machine in 40 cc. of water. This route ensures the widest distribution of the respiratory virus vaccines - along the bird's respiratory tract. It ensures the most rapid response of any route. However, it can lead to severe side effects in the birds when other diseases are present, when too fine a droplet or cold water is used, and when a less attenuated vaccine is given before a more attenuated one.

The water route is the most widely used but is the least effective method. Effective distribution and uptake of vaccine is difficult. Many birds may be missed.

Marek's Disease vaccine, the only live vaccine given by injection, is given to all replacement pullets and some broilers at day old in the hatchery.

The disease situation in the area dictates the speed of response to vaccination required and the frequency of application. One has to take into account the possible blocking effect of maternal antibodies on the primary vaccination of young flocks and the necessity to produce an anamnestic response either by the application of another live vaccine or the administration of an inactivated vaccine. The injection of individual birds with an inactivated vaccine is obviously labour intensive. These vaccines have improved enormously over the last decade with the utilisation of oil based adjuvants. In general they obviate the necessity for any further vaccination during lay.

The ultimate measure of efficiency of any programme will be based on resistance of birds to challenge, and their serological response to the vaccines administered.

VACCINATION OF BROILERS

The average life span of broilers is only 7 weeks and with the high stocking densities employed, it is most important to prevent respiratory disease. Infectious bronchitis is the disease for which vaccination is most likely to be required. The most effective programme is considered to be the use of coarse spray vaccine at day old in the hatchery, occasionally followed by a second dose of live vaccine in the drinking water at 18 days. The aerosol method of application is not generally used for broilers, because of the dangers of vaccine reaction resulting in increased mortality from secondary bacterial infection, e.g. colisepticaemia or reduced growth rate. Newcastle Disease is not a problem in the U.K. today. Past experience has shown that it can be controlled by aerosol vaccination. Broilers are therefore rarely vaccinated for this infection.

Infectious bursal disease is also important in broilers and considerable research effort has been successfully directed at the immunisation of parents, which should give protection to the broiler by means of maternal antibody for the first 3 - 4 weeks. Vaccination of the broiler is therefore not generally practised. However, we now see some flocks succumb to I.B.D. between 4 and 7 weeks.

Broilers in Great Britain are not usually vaccinated against Marek's Disease. However, on certain continuous production sites there is a heavy build up of infection and mortality will occur between 5 and 7 weeks if hatchery vaccination is not carried out.

Table 1. Vaccination programme for commercial broilers (UK)

Age - (days)	Vaccine	Route
1	Infectious Bronchitis H120	Coarse spray
(18	Infectious Bronchitis H120	Water / Spray)
	In Newcastle Disease endemic areas or emergency situation.	
1	Hitchner B1 + IB.	Eye drop or coarse spray
12	Hitchner B1	Drinking water or aerosol spray
24	Hitchner B1	Drinking water or aerosol spray
35	Hitchner B1	Drinking water or aerosol spray

VACCINATION OF LAYERS

These birds are usually housed in cages at 18 weeks of age, having been reared and vaccinated either on litter or in cages. Infectious Bronchitis is the most important disease. There are many variations in the programme used for immunisation. A successful immunising programme

should give protection to the respiratory tract in the growing pullet and layer, to the reproductive tract in the adult and give no respiratory reaction. A highly attenuated vaccine, H120, is given as a primary vaccination followed by a less attenuated H52 or an inactivated vaccine, as a booster vaccination. Many cage rearing systems utilise nipple drinkers. This route cannot be recommended for administration of live vaccines so the aerosol method is to be preferred. Often birds of different ages are kept on one site and it may be inappropriate to use H52 vaccine when younger birds are present on the farm. For some as yet unexplained reason H52 interferes with the response of the inactivated vaccine. For the second immunising dose the less virulent H120 strain is used in these circumstances.

Laying birds are vaccinated with Newcastle Disease vaccines during rearing as vaccination of laying stock in the face of an outbreak could in itself produce severe drops in egg production.

Egg drop syndrome 76 (EDS) vaccine may be used for commercial layers particularly if the disease has already been diagnosed on site or if the disease is on neighbouring farms. This condition is re-emerging as a problem in free range birds.

The replacement pullets receive an injection of Marek's Disease vaccine in the hatchery as day olds.

Table 2. Vaccination Programme for Layers (UK)

Age	Vaccine	Route
1 day	Marek's Disease	Intramuscular injection
1 day	Infectious Bronchitis H120	Coarse spray
3 weeks	Newcastle Disease HB1	Drinking water
5 weeks	Infectious Bronchitis H120	Aerosol
5 weeks	Newcastle Disease HB1	Aerosol
14 weeks	Avian encephalomyelitis	Drinking water
18 weeks	Infectious Bronchitis (inactivated) Newcastle (inactivated) E.D.S. (inactivated)	Intramuscular injection.

This programme would cost approximately 8p per bird. It costs 3.5p to produce one Grade A egg. One can easily lose more than 3 good eggs with any of the diseases. If Newcastle Disease is endemic then more vaccination with HB1 becomes necessary.

On some sites other infections have to be taken into account.

Infectious Laryngotracheitis - Eyedrop vaccination at 6 weeks if it is a problem on the rearing farm, repeated when moving to the cages. If

it is only a problem on the laying site, vaccination is carried out by eye drop at housing on the laying site. Water and spray applications can be either ineffective or downright dangerous.

Pasteurella - requires two injections at 4 - 6 weeks intervals with the first one about 8 weeks.

Pox vaccine is given by wing web stab at 9 weeks.

You will see that it can be very difficult to fit in all the vaccinations if the flocks have to be protected against all the diseases. Siting of a new operation is obviously very important.

Sometimes layers are put into an induced moult and rest after the first year of lay. To ensure continued protection, they are usually revaccinated for avian encephalomyelitis, Infectious Bronchitis and Newcastle Disease just before they come back into lay.

Vaccination programmes have remained essentially the same over the past five years, but now greater emphasis is being placed on the use of inactivated vaccines, which give longer lasting immunity, for the final vaccination for many diseases in laying birds.

Vaccines will continue to be improved and modified to suit the needs of an evolving industry and hence programmes will change in subtle fashion to incorporate new ideas and scientific information. The old requirements of site selection, security, hygiene, nutrition, however, will still remain.

Some typical costs:-

Table 3.	Jan./Mar. 1985	Broilers
	Chicks	18.446p
	Feed	78.445p
	Litter	1.066p
	Clean out	1.044p
	Medication	0.037p
	Vaccination	0.037p
	Cost per lb. live weight	22.938p
	Price received per lb. live weight	26.24p
	Margin per lb. live weight	2.926p

Table 4.	Jan./Mar. 1985	Layers
	Vaccination of a laying flock	8p per bird
	Cost of production per dozen eggs	42p
	Cost of point of lay pullet	£2.15
	Bonus for barn lay/ free range eggs per dozen	5-10p

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EPIDEMIOLOGY AND PREVENTIVE MEDICINE IN BEEF PRODUCTION

THE EPIDEMIOLOGY OF BOVINE VIRUS DIARRHOEA VIRUS

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Bovine virus diarrhoea is widespread in cattle populations throughout the world, and has been associated with a range of diseases. As well as causing enteric disease it is an important cause of congenital infections, the outcome of which can vary from fetal death to the birth of apparently healthy but persistently infected calves (Van Oirschot, 1983). A greater understanding of the epidemiology, pathogenesis and clinical aspects of BVDV infection has emerged during this decade and the newer findings have been encompassed in several recent reviews (Brownlie, 1985; Duffell and Harkness, 1985; Nettleton et al., 1985; Bielefeldt Ohmann and Babiuk, 1986).

Information on the epidemiology of BVDV infections in Britain has come largely from data collected by the Veterinary Investigation Services from specimens submitted by practitioners and from detailed herd investigations. In Scotland, the Moredun Research Institute provides a cattle and sheep virus diagnostic service in support of the nine Scottish Veterinary Investigation Centres, and this paper contains details of investigations involving BVDV in the years 1980 to 1985. The results are discussed in relation to findings from other countries and conclusions drawn about the present state of knowledge of the epidemiology of BVD virus.

MATERIALS AND METHODS

Specimen collection and submission

Virtually all the specimens tested were submitted by the nine veterinary investigation laboratories, three each of which are attached to the East, West and North Scottish Colleges of Agriculture respectively. They had been collected by veterinary practitioners or veterinary investigation officers according to standard guidelines (Snodgrass et al., 1980).

Serology

Heat inactivated serum samples were tested for the presence of anti-BVDV

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antibodies in a microneutralisation test using 100 $TCID_{50}$ per well of the NADL strain of BVDV. Results were expressed as the reciprocal of the dilution corresponding to the 50 per cent endpoint of neutralisation as calculated by the Karber method.

Virus isolation

Specimen filtrates were examined for the presence of cytopathic viruses during two weekly passages in secondary bovine embryonic kidney cells, previously screened for the absence of latent BVDV. After 14 days, cultures were frozen and thawed and passed into tubes containing bovine embryonic kidney cells grown on coverslips. Seventy-two hours later the coverslips were fixed in acetone and stained by an indirect immunofluorescent method using a hyperimmune serum against the NADL strain of BVDV produced in a gnotobiotic calf. Samples producing specific fluorescence in this test were considered positive for BVDV. Positive isolates which produced characteristic cytopathic changes after 14 days in culture were classified as cytopathic. Specificity of the cytopathic effect of some selected isolates was confirmed by microneutralisation tests using specific antiserum raised against the NADL strain of BVDV.

Herd Investigations

The methods used have been published in full (Barber et al., 1985), but basically the herd investigations were carried out in order to detect animals persistently infected with BVDV following infection in-utero. Serological screening of groups of animals within each herd was followed by attempted virus isolation from the blood of cattle with reciprocal serum neutralisation titres of less than 22.

RESULTS

Prevalence of BVDV

The prevalence of BVDV in Scotland can be estimated from the number of cattle with neutralising antibody in their sera. Paired sera from a wide age range of cattle are commonly examined for the presence of BVDV antibodies during investigations into the cause of enteric disease, abortions and respiratory disease. Although biased, these sera represent a consistent source of material from a wide cross-section of the cattle population. The results of tests on groups of fewer than 20 animals are shown in Table 1. Seropositive animals were taken as those which had a reciprocal antibody titre greater than 4 in at least one of their sera.

From the Table it is clear that BVDV is widespread in all areas of the country with 60-70% of cattle having antibodies; there was some evidence of

Table 1. Prevalence of BVD virus antibodies in Scottish cattle 1980-1985

	East	West	North
1980	195/288[a](68%)	94/147(64%)	192/252(76%)
1981	137/218 (63%)	173/265(65%)	219/296(74%)
1982	134/207 (65%)	195/262(74%)	206/308(67%)
1983	124/216 (57%)	147/263(56%)	197/313(63%)
1984	340/641 (53%)	220/351(63%)	304/478(64%)
1985	314/558 (56%)	209/317(66%)	416/653(64%)
Totals	1244/2128(58%)	1038/1605(65%)	1535/2300(67%)
Total for whole of Scotland		3817/6033(63%)	

[a] Number of seropositive animals/Number of animals tested (% seropositive)

Table 2. Isolation rates of BVDV from disease outbreaks.

	East	West	North
1980	10/331[a](3%)	7/143(5%)	13/483(3%)
1981	15/247 (6%)	8/147(5%)	10/327(3%)
1982	13/351 (4%)	11/178(6%)	24/302(8%)
1983	21/404 (5%)	12/192(6%)	32/403(8%)
1984	53/725 (7%)	22/301(7%)	31/471(7%)
1985	41/578 (7%)	43/309(14%)	58/511(11%)

[a] Number of outbreaks from which BVDV was isolated/Number of outbreaks investigated (% isolation rate).

a reduction in the number of seropositive animals between 1980 and 1985 in the East and North.

Isolation of BVDV from disease outbreaks

BVDV was isolated commonly from clinical specimens. The virus isolation rates from disease outbreaks in which fewer than 20 animals were sampled are shown in Table 2. There was little difference between the isolation rates in each year from the three areas, but a gradual improvement in the overall isolation rate with a noticeable increase in 1985.

The association of virus isolation with disease syndromes for the whole of Scotland is shown in Table 3, from which it can be seen that BVDV was isolated most successfully from cattle with enteric disease. Outbreaks of neonatal diarrhoea in calves younger than 1 month old were not included in these figures, so that all the isolates were from older animals the majority of which were showing clinical symptoms of mucosal disease. Virus isolations from outbreaks of congenital disease, principally abortion cases, were made consistently but at a low frequency, as were isolations from cases of respiratory disease. Isolations from other syndromes were largely from calves with 'ill-thrift', some of which had skin lesions. The detection of these chronically infected calves with nebulous symptoms increased from 1983 onwards as the submission of blood samples for virus isolation became more common following the recognition of the role of persistently infected cattle in the spread of BVDV (see also below).

Cytopathogenicity of BVDV isolates

Isolates of BVDV were cytopathic (CP) or noncytopathic (NCP) in tissue culture. In some disease outbreaks both CP and NCP viruses were isolated from different animals or even from different tissues collected from the same animal. The relationship between cytopathogenity of BVDV isolates and clinical diseases from which they were recovered is shown in Table 4. Isolates from cases of congenital and respiratory disease were predominantly NCP and this was a consistent finding over all 6 years. Isolates from cases of enteric disease, however, were predominantly cytopathic for the first four years after which NCP isolates were more common. This does not represent a startling shift in the pathogenesis of BVDV but is again a reflection of the success of diagnosing clinical disease in the live animal. Before 1983 the majority of diagnoses resulted from virus isolation from post-mortem tissues, and cytopathic virus was isolated from 64% of such outbreaks. The equivalent figure for post-mortem tissues between 1983 and 1985 was 70% confirming the strong association of CP virus with tissues of animals dying of mucosal disease. Between 1983 and 1985 confirmation of BVDV infection in live animals with enteric disease by the detection of viraemia was used commonly. Isolates from blood were predominantly NCP (69%) although CP virus was recovered from 31% of outbreaks in which animals were showing evidence of disease.

Table 3. Isolation rates of BVDV from different clinical syndromes

	Clinical syndrome			
	Enteric	Congenital	Respiratory/Ocular	Other or unknown
1980	17/187[a](9%)	5/ 89(6%)	7/630(1%)	1/51
1981	22/186 (12%)	6/ 80(8%)	5/412(1%)	0/43
1982	26/182 (14%)	7/ 84(8%)	15/542(3%)	0/23
1983	48/290 (16%)	4/110(4%)	4/548(1%)	9/51
1984	75/524 (14%)	8/235(3%)	11/631(2%)	12/107
1985	104/493 (21%)	11/264(4%)	13/529(2%)	14/112

[a] Number of outbreaks from which BVDV was isolated/Number of outbreaks investigated (% isolation rate).

Table 4. Cytopathogenity of BVDV isolates from different clinical syndromes

		Clinical syndrome			
		Enteric	Congenital	Respiratory/Ocular	Other or unknown
1980	CP	11	2	2	1
	NCP	5	2	4	0
	Mixed	1	1	1	0
1981	CP	12	0	1	0
	NCP	7	5	4	0
	Mixed	3	1	0	0
1982	CP	11	0	2	0
	NCP	7	6	10	0
	Mixed	8	1	3	0
1983	CP	25	0	0	1
	NCP	21	4	4	8
	Mixed	2	0	0	0
1984	CP	26	1	4	0
	NCP	44	7	7	11
	Mixed	5	0	0	1
1985	CP	31	0	3	3
	NCP	68	10	9	11
	Mixed	5	1	1	0

CP : Only cytopathic BVDV isolated
NCP : Only non-cytopathic BVDV isolated
Mixed : Both cytopathic and non-cytopathic BVDV isolated

Mixed viral infections

Although BVDV was usually isolated on its own, dual infections with bovine herpesvirus 1 (Greig et al., 1981), paravaccinia virus (bovine papular stomatitis virus), enterovirus, and respiratory syncytial virus have also been made over the last six years.

Herd investigations into the frequency of persistent BVDV infections

As well as the epidemiological surveillance provided by the diagnostic service, more detailed herd investigations have been conducted by the Scottish Veterinary Investigation Service, often following confirmation of BVDV infection in a herd with enteric and/or congenital disease. Studies have been completed on 2 herds in which the serological and virological status of all animals over 4 months old was determined. In a closed dairy herd of approximately 200 milking animals BVDV was associated with congenital disease, pneumonia and enteric disease. Of 241 calves examined 12 were shown to be persistently viraemic as a result of congenital infection (Barber et al., 1985). In a suckler herd of 182 animals 10 persistently infected calves were identified.

In addition, groups of more than 20 animals on 13 farms from which BVDV had been isolated from dying animals were blood tested. All groups had a high prevalence of antibodies ranging from 73 to 100%. Viraemic animals were detected on seven of the farms with the highest frequency being in a group of 69 calves which contained 8 viraemic calves.

Overall results from the 15 farms showed that 39(3%) of these 1137 selected cattle were viraemic and almost certainly persistently infected with BVDV.

DISCUSSION

The prevalence of BVDV in Scotland, as judged by the detection rate of serum antibodies in more than six thousand cattle, was 63% a figure which is remarkably close to the 61% and 62% of two comparable surveys carried out in England and Wales (Phillip 1973; Harkness et al., 1978).

The data provided by the virus isolation results from six years of laboratory tests on specimens from the field require cautious interpretation. Because of the selective nature of the samples they do not reflect accurately the incidence of disease conditions caused by BVDV. They do, however, contribute to an understanding of the epidemiology of the virus and demonstrate very clearly why BVDV was considered for many years to be principally a cause of enteric disease in cattle. Two enteric syndromes were described originally. Bovine virus diarrhoea was very contagious with moderate clinical disease and pathological changes and had a high morbidity and low mortality, while mucosal disease (MD) was sporadic, had a low morbidity, was not highly contagious but affected animals had severe clinical

signs and pathological lesions and the mortality rate was high. These two names have been sustained because for some outbreaks they remain clinically and pathologically valid. Not all outbreaks conform to the high morbidity/low mortality or high mortality/low morbidity patterns, however, and the two syndromes can be considered to represent overlapping extremes at either end of a range of disease (for review see Pritchard, 1963). Experience at our laboratory (see also Snodgrass et al., 1980) has shown that while MD is commonly confirmed by virus isolation, BVD seldom is; only seroconversion to BVDV has occasionally helped to confirm clinically suspicious outbreaks of BVD. Viruses recovered from Scottish cases of MD include both CP and NCP isolates with a predominance of CP virus. Similar findings in border disease infections of sheep led to the proposition that persistent infection resulting from intrauterine exposure predisposed lambs to a condition analogous to MD in cattle (Barlow et al., 1983; Gardiner et al., 1983). Recent reports from other laboratories have provided similar findings and it is now recognised that MD occurs only in those cattle congenitally infected with NCP virus, and severe fatal MD has been experimentally produced by superinfecting such animals with CP virus (Liess et al., 1983; Brownlie et al., 1984; Roeder and Drew, 1984; Bolin et al., 1985b; McClurkin et al., 1985). Thus early fetal infections producing calves persistently infected with BVDV hold the key to the epidemiology of the virus. While virus isolation results, and also detection of BVDV antibodies in foetal fluids, from field material occasionally implicate this agent as a cause of congenital disease, they clearly underestimate the efficiency with which this is occurring.

Detailed herd investigations have given a greater insight into the prevalence of BVDV in infected herds. In the fifteen herds investigated in Scotland the number of surviving persistently infected calves was high at 3% of the 1137 cattle examined. This could have been influenced by the selected nature of the herds, although a similar study in the USA revealed a frequency of persistent infection of 1.7% (Bolin et al., 1985a). In a less biased sample of abattoir cattle in Denmark 0.9% persistently infected animals were detected, with the equivalent figure in infected herds being 10.5% (Meyling, 1984). Bovine virus diarrhoea virus is clearly very efficient at crossing the placenta to cause congenital infections. This can occur following infection of suspectible cows during pregnancy but it has also been recognised that persistently infected cows can themselves produce persistently infected offspring so that familial lines of BVDV infected cattle may be established (Littlejohns 1982; Straver et al., 1983).

Finally, no consideration of the epidemiology of BVDV can ignore possible reservoirs of infection outside the cattle population. The virus is serologically related to the viruses of border disease in sheep and hog cholera in pigs and the three agents have been classified as pestiviruses. The natural host range of pestiviruses is restricted to ruminants and pigs. Among domestic animals experimental cross-infection has been demonstrated between cattle, sheep, pigs and goats (Ward, 1971; Phillip and Darbyshire, 1972; French et al., 1974; Gibbons et al., 1974; Taylor et al., 1977). Interspecies infection, by some pestivirus strains at least, is therefore a possibility. In Scotland, a pestivirus has been isolated from red deer (Nettleton et al., 1980) and there is serological evidence of widespread infection in a variety of wild ruminants which should also be considered potential reservoir hosts (Hamblin and Hedger, 1979; Karstad, 1981).

Cattle, however, are the principal hosts of pestiviruses, and although recent studies have revealed new and fascinating insights into the epidemiology of BVDV, much is still to be learned about the vagaries of this agent which has clearly established an excellent long-term relationship with its major host. There is a need for more detailed information on the economic effects of BVDV, and effective control measures to limit these effects will have to be considered carefully.

CONCLUSION

The high prevalence of animals with serum antibodies to BVDV has confirmed that the virus is widespread in cattle populations throughout the world. This ubiquity has hindered the elucidation of the epidemiology of BVDV until quite recently when a combination of experimental infections, routine epidemiological surveillance and detailed herd investigations has revealed the fascinating natural history of the virus.

Many infections with BVDV are sub-clinical but the virus has been associated with a range of disease conditions. The most serious consequences of infection occur when BVDV infects susceptible cattle during early pregnancy. Ensuing fetal infection may cause abortion, mummification or stillbirth, but the low pathogenicity of many virus strains ensures the birth of live calves. Some calves have congenital defects while others are clinically normal. Surviving calves infected before the onset of immune competence at 120 to 140 days gestation are persistently infected with, and apparently immuno-tolerant to, their virus.

The mechanisms involved in immune tolerance are poorly understood but the practical consequences are that when maternal immunity has waned affected animals are viraemic, are free of, or have very low levels of neutralising antibodies to BVDV and constantly shed infectious virus. Depending on the severity of their infection they can remain healthy for years but for reasons as yet unknown they may succumb at any time to severe, usually fatal, acute or chronic mucosal disease (MD). Recognition of the existence of such animals, which may comprise one to three per cent of the total cattle population has helped to explain the sporadic cases of BVDV infection originally described as mucosal disease (MD) which has a high mortality but low morbidity. Animals with MD have severe clinical symptoms relating to the alimentary tract and enteric lesions are observed at necropsy. The other recognised enteric disease caused by BVDV is the epidemic form as described in the first recorded case of bovine virus diarrhoea. It has a high morbidity and low mortality, and manifests itself with mild clinical symptoms and pathological lesions. It can be reproduced readily in normal susceptible animals. Experimental reproduction of severe fatal MD, however, has only recently been achieved by superimposing BVDV infection on already persistently infected animals.

As well as inducing immuno-tolerance following early fetal infection BVDV can cause other dysfunctions of the immune system due to its ability to infect lymphoid cells, and such infection of lymphoid tissue could account for some of the chronically affected calves which result from BVDV infection *in utero*. Infection of lymphoid tissue can also result in temporary immunosuppression during post-natal infections of healthy calves which can

predispose them to disease caused by other microorganisms. BVDV may thus contribute to syndromes of multi-factorial aetiology such as calf respiratory disease and neonatal diarrhoea.

BVDV can infect sheep, pigs, goats and deer and there is serological evidence that it occurs in a variety of wild ruminants. While possible transmission from these species cannot be ignored, cattle are the principal hosts. The virus is easily transmitted by contact and circulates freely among normal calves no longer protected by maternal immunity. These calves excrete virus for up to 21 days and thereafter develop long lasting immunity to that strain. BVDV isolates exhibit a range of antigenicity but natural infection with one probably confers some immunity against other field strains. Calf-to-calf transmission between normal animals will account for many infections but it is the calves infected in early gestation, which become persistently infected, that ensure the maintenance of high levels of virus in the environment. If such calves reach sexual maturity they tend to have reduced fertility but may produce, by vertical transmission, further persistently infected calves.

Control of the diseases due to BVDV must be aimed at preventing the production of congenitally infected calves which are seeded out into the general cattle population as remarkably efficient spreaders of virus.

ACKNOWLEDGEMENTS

We thank all the members of the Scottish V.I. Service whose collaboration over the past 6 years has helped to generate the information reported in this paper.

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CONTROL OF THE BOVINE PESTIVIRUS SYNDROME IN CATTLE: A CASE FOR SOCIAL COST-BENEFIT ANALYSIS?

R.M. BENNETT * AND J.T. DONE *

The basic principles of Social Cost-Benefit Analysis (SCBA) have been applied, to varying degrees, to appraise the desirability of different public investment decisions since the 1930s, starting with the appraisal of water resource projects in the United States and used in relation to other social projects such as public health since the 1950s. However, it was not until the 1960s that the concept of SCBA became generally acknowledged in the UK, with reference to its uses in a Government White Paper in 1967.

One of the first applications of SCBA to animal health in the UK was by Power and Harris in 1968 in an evaluation of two alternative methods for controlling foot-and-mouth disease in Great Britain. Since that time cost-benefit analysis has been used to appraise various animal diseases and programmes for disease control (see Beal, 1980; Ellis, 1982), although many are little more than financial appraisals with little attempt at considering costs and benefits to society in a wider context (Grindle, 1986). The majority of these studies also appear to have considered only a very limited range of alternative strategies or options toward disease control, which seems to imply that a decision has already been taken concerning the most likely or desirable strategies, with the criteria of selection usually left unstated.

The objective of this paper is to discuss the methodology of the process of economic appraisal of animal diseases and of disease control strategies, and to consider the extent to which the techniques and philosophy of SCBA can help in such an appraisal.

THE PRINCIPLES OF ECONOMIC APPRAISAL AND SCBA

It may be asked why an economic appraisal of animal diseases and of possible control strategies is necessary.

Economics is concerned not merely with money or financial matters but with the allocation of scarce resources to alternative possible uses within society. Given that resources are almost always not infinite in supply, the decision to employ resources in one area will necessarily deprive another area of their use. For example, resources committed to reducing disease effects

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by a policy of slaughtering disease carriers may mean that less resources can be used for the development of diagnostics, vaccines, prophylactics and therapeutics. It is important therefore that such resource allocation decisions are made with information concerning the relative advantages and disadvantages (benefits and costs) of alternative uses, before (and also, periodically, after) resources are committed. It is one of the functions of economists using cost-benefit analysis to provide this information for decision makers.

Social Cost-Benefit Analysis attempts to evaluate all the important costs and benefits associated with an allocation of resources and to determine on which people in society such costs and benefits are likely to fall. Thus SCBA considers not just the direct costs and benefits of a policy but also the consequential or external effects outside of the strict confines of the area of resource use.

For example, a national slaughter policy of disease carriers might involve direct costs of the loss in value of livestock slaughtered to farmers and disruption to farm production, together with costs to the Government of administration of the policy and of compensation payable to farmers. The latter is, in effect, a transfer payment from Government to producers, transferring a large amount of the costs of livestock slaughter from farmers to the Government and indirectly to taxpayers. However, external to this slaughter policy, there may be more wide-ranging effects. First, if significant numbers of animals are slaughtered the domestic supply situation of products from these animals will be changed both in the short-run and the long-run. In the long-run and in the absence of increased imports, prices received by producers and paid by consumers for the lower level of output are likely to increase (assuming no Government intervention), involving a transfer of at least some of the costs of the policy to consumers from producers. Disruption to supply caused by a policy may in fact have an impact both 'upstream' to marketing processes through to consumers, and 'downstream' to the suppliers of inputs to the production process. This will involve absolute costs to marketing and other processes caused by inefficiencies in resource allocation as a result of significant supply changes, and also a transfer of costs and benefits between various sectors within society.

It is evident that such external or third party effects can become very complex and there is a danger of 'double-counting' costs and benefits. However, it is perhaps the prime role of SCBA to make the analyst and decision maker aware of such effects and to look at the impact of resource allocations in the wider context of society.

The practical application of SCBA involves several difficulties. The valuation of costs and benefits such that they truly reflect their value to society is one of the most difficult aspects of SCBA. Resources and goods are valued according to their 'opportunity cost' and according to consumers 'willingness to pay'. Market prices, due to various market imperfections or distortions, may not provide a reasonable guide as to the true value of an item to society and may therefore have to be adjusted. Market prices may not exist for certain non-traded items nor for 'intangible' items such as the degree of animal suffering. Many costs and benefits may not be quantifiable, especially in money terms, and must therefore remain as qualitative assessments, but which are still of importance. A time factor must also be included in a SCBA, whereby future benefits and costs are discounted to a present value using an appropriate discount rate. The choice of discount rate is often of

some difficulty when considering public or national projects.

In the past, SCBA has perhaps come somewhat into disrepute for assuming that everything can be reduced to a monetary value, in that many studies have attempted to place a financial value on costs and benefits where it was highly debatable whether they could be measured in money terms (for example human life), and by assigning questionable values to non-market items or by dubiously adapting market prices to take account of distortions.

Future SCBA studies should concentrate on valuing only those costs and benefits that can reasonably be quantified by the measuring rod of money and adjusting market prices only where significant distortions exist and where more appropriate valuations can be constructed.

The principle of SCBA of trying to consider all costs and benefits both direct and indirect can also be applied at the individual producer or farm level. In fact, many of the difficulties of applying SCBA to national policies disappear at the farm level. Thus when considering animal health programmes on the farm, producers can take account not only of the direct consequencies of each policy on the costs and returns from their livestock but also the indirect effects on other enterprises and on the farm business as a whole. For example, elements ranging from an improvement in working conditions to the reduction of risk and uncertainty as a result of an animal health policy on the farm might be taken into account, either quantitatively or qualitatively.

APPLICATION OF SCBA TO ANIMAL HEALTH AND TO STRATEGIES FOR DISEASE CONTROL

Figure 1 shows the necessary stages involved in any economic analysis of disease effects or of possible strategies for control. There are of course, many interrelationships between stages that are not shown in this simplified diagram.

First, information concerning the epidemiology of a disease is necessary, both descriptive and statistical. This is used to develop an epidemiology model of the disease. Such a model may be in several parts and will contain information on disease pathways, incidence rates and effects.

The epidemiology model of a disease is then combined with a model of the production systems likely to be affected by the disease in order to identify the impact of the disease or any strategies for control on the production process. This knowledge together with other information (such as whether a vaccine exists to combat the disease) can then be used to formulate and investigate technically feasible alternative strategies for disease control or abatement.

The economic model provides information on the areas of impact of the disease or control strategies other than the production processes directly affected and, together with the techniques of SCBA it gives an identification and evaluation of the important costs and benefits associated with the disease and with each possible strategy for control (both in terms of the direct effects on the production process and in a wider context). The process of SCBA is then completed by a rationalisation of costs and benefits to enable the comparison of alternative strategies and to provide a measure of the economic effects or impact of the disease. Such a rationalisation will result in a Net Present Value or benefit-cost ratio attached to each strategy. Other qualitative elements may also exist which cannot be

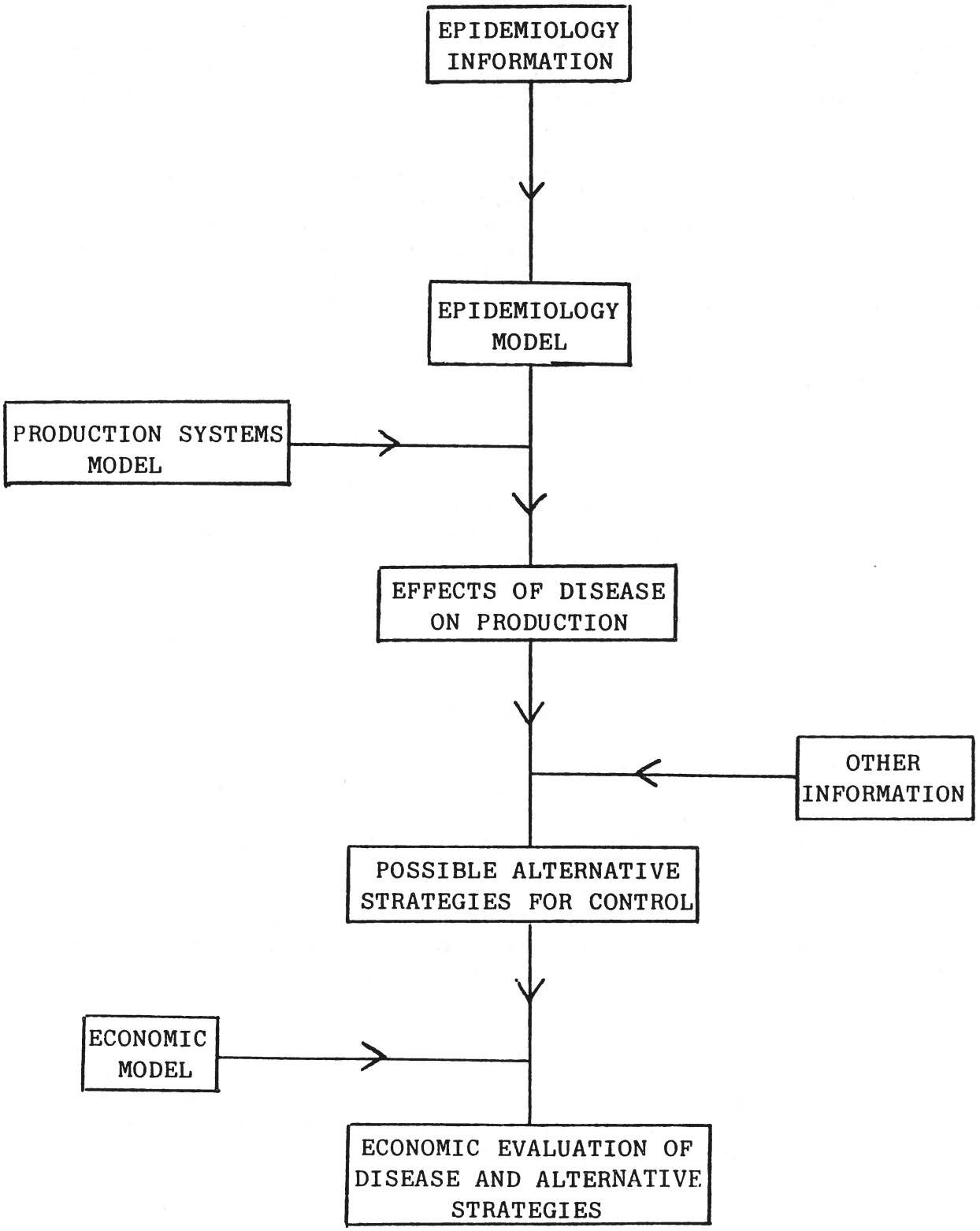

Fig. 1. Stages necessary to enable economic evaluation.

aggregated with others into a single measure, and care must be taken to ensure that such elements are properly regarded as part of the evaluation.

It is important that all the possible options for disease control are considered, however briefly, before being dismissed. The exploration and formulation of possible strategies for disease control must be carried out systematically using the epidemiology and production system models, and not merely an investigation of previous strategies used on other disease complexes.

The final choice of strategy is up to the decision maker, with SCBA providing a more informed basis for such a decision. The process of SCBA is often of more value than any end results of the analysis in the form of Net Present Values or benefit-cost ratios, since it forces the analyst to think carefully and systematically about the effects of a disease on society and of the feasible options available for its control.

ECONOMIC APPRAISAL OF THE BOVINE PESTIVIRUS SYNDROME

Epidemiology

A simple diagrammatic, descriptive model for the Bovine Pestivirus Syndrome (BPS) is shown in Figure 2. Previous 'guestimates' of disease incidence/ prevalence used by Spedding, Bennett and Done (1986), have been adapted to take account of better and more accurate epidemiological information made available to the authors since that paper was presented. However, more suitable and accurate data in the form of a cohort study of disease incidence is still required to enable a more reliable economic appraisal.

Components of the Bovine Pestivirus Syndrome

As shown diagrammatically in Figure 2, the pathological (and direct economic) effects of BVDV[1] infection of cattle can conveniently be grouped into four main categories related to the age and immune status of the host, but also mediated by the weight and duration of the insult, viz:

-A: in the post-natal immune-competent, but non-immune animal: inapparent or mild clinical disease (= bovine virus diarrhoea), usually with no mortality; normally followed by sero-conversion and active immunity. In the pregnant non-immune female, infection of the dam will usually result in infection of the conceptus (cf B1 and B2).

-B1: in the embryo/foetus infected before the onset of immunocompetence (ie before about 100-120 days gestation); death of the conceptus, with or without abortion, if infected early in gestation; developmental defects in survivors; immune paralysis or immune-tolerance with persistent postnatal excretion of BVDV; and, in the case of females, liability to infect their own progeny in utero.

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1 Bovine Virus Diarrhoea Virus.

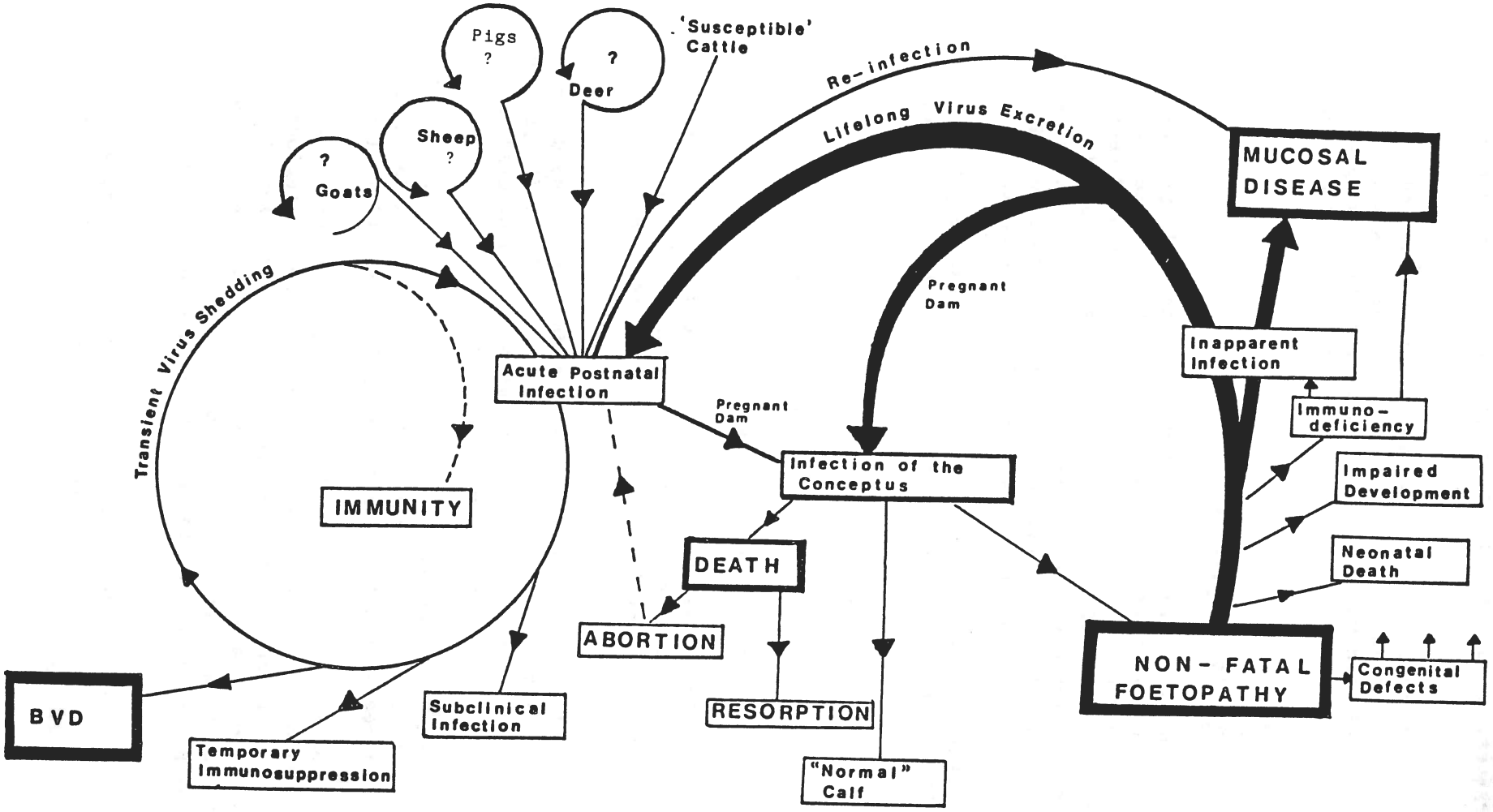

Fig. 2. BPS : Patterns of infection and disease

(Modified from Spedding et al. 1986)

-B2: in the foetus infected after the onset of immune-competence; variable teratogenesis of limited duration; prenatal seroconversion and active immunity.

-C: in surviving animals of group B1 which subsequently become infected with another strain of BVDV; classical mucosal disease (cf Roeder and Drew, 1984; Duffell and Harkness, 1985) with variable, but generally high, mortality; normally no sero-conversion or immunity, but continuing excretion of BVDV.

Production systems and disease effects

The two main production systems affected by BPS are milk production and beef production. These systems are related in that both may be carried out on the same farm, and also because a considerable proportion of beef animals come from dairy herds. The effects of BPS on these systems are in the form of (i) loss of livestock through death (ii) reduced or lost milk output (iii) reduced or lost beef output (iv) disruption to production and waste or under-utilisation of resources such as land, labour or capital.

Figure 3 shows a breakdown of BPS into four main disease pathways or components together with their economic effects on the production system.

Possible strategies for control

There are four major policy options for dealing with BPS, viz:

-A) 'Laissez-faire'; ie a purely passive policy.

-B) 'Control' in the sense of "...purposeful reduction of specific disease prevalence of relatively low levels of occurrence" (Andrews and Langmuir, 1963); though perhaps 'disease abatement' might be a preferable term (Done, 1985).

-C) 'Elimination', interpreted either as a reduction of the disease prevalence to a lower level than is usually aimed at in 'control' programmes (Payne, 1963 a & b), or as elimination of the disease without eradication of the causal pathogen (Spinu and Biberi-Moroianu, 1969).

-D) 'Eradication', defined as "...the purposeful reduction of specific disease preval nce to the point of continued absence of transmission within a specified area by means of a time-limited campaign" (Yekutiel, 1980).

Within these categories a variety of tactics may be combined/permutated to generate a wide range of alternative strategies at local, regional or global levels. In this paper we have concerned ourselves primarily with policies as they might be applied at national level in the UK. At individual enterprise level, costs and benefits would obviously vary tremendously, generating social/political pressures which would also need to be taken into account.

As examples, we have considered three different strategies; a 'do nothing/ laissez-faire' system, a 'test and eliminate' system and a 'vaccinate, test and eliminate' system.

BPS Disease Components	Economic Consequences: Loss of output	Loss of livestock value	Cost of medication	Disruption to production and markets
MD	Milk Beef	All cattle	Symptom reduction	Milk Beef
Fatal foetopathy	Milk	Embryo foetus	-	Milk Suckler beef
Non-fatal foetopathy	Beef	Calf	-	Beef
BVD	Milk Beef	-	Symptom reduction	Milk Beef

Fig. 3. Economic consequences of BPS disease effects

The first involves evaluating the present economic importance of the effects of BPS. Estimates of the likely incidence and magnitude of effects of BPS are used and related to the national (UK) herd of both dairy and beef cattle. The direct economic effects are valued at estimated market prices and are presented on an annual basis.

The second strategy ('Test and Eliminate') consists of testing for the presence of virus in all cattle and eliminating the virus-positive animals. This heroic operation is assumed to take one year, with the benefits not realised until year two onwards. Benefits are assumed to be a disease-free national herd for 5 years with all direct costs borne in year one. These are discounted to give a Net Present Value.

The 'Vaccinate, Test and Eliminate' strategy is based on protecting the conceptus from transplacental infection by giving breeding females sufficient immunity to frustrate infection during pregnancy. The strategy consists of two parts. First, the vaccination of all females before pregnancy with an inactivated polyvalent viral or subcomponent vaccine and then testing for sero-conversion. Those females that do not sero-convert are culled before mating. It is assumed that vaccination will be required before each subsequent pregnancy and therefore entails costs of vaccination and culling each year. Benefits depend largely on the efficacy of the vaccine and are in the form of reduced disease costs compared to the 'Do Nothing' situation.

It must be stressed that the active strategies identified here are merely examples arbitrarily chosen to illustrate the application of SCBA. We have deliberately not attempted to choose a "best" strategy, since our main aim is to advocate an approach rather than to recommend a solution.

Economic model/framework and analysis

The detail of the economic framework and analysis of the economic appraisal of the basic BPS situation and of the two arbitrarily selected strategies for control are not contained here, but are available from the authors as an Appendix to this paper. The analysis takes into account only the direct costs and benefits in terms of which costs and benefits are included and how they have been valued. Estimations of the economic consequences of certain BPS disease effects, such as reduced resistance of infected livestock to other diseases, have not been possible due to a lack of information.

No evaluation of the indirect or consequential costs and benefits necessary for a full SCBA has yet been carried out; nor have the welfare or equity implications of these been assessed. Such analysis in detail can be very time consuming and complex; and, unless significant levels of costs and benefits are involved, may not be worthwhile. However, a few points are worthy of consideration.

The disease effects of BPS result in reduced output of beef and milk or a waste of resources (compared with a disease-free situation) in the long-run,which appear to be costs borne by producers. Taking beef production as an example, reduced supplies (or higher production costs for a given level of output) result in higher beef prices received by farmers and paid by consumers, so that at least part of the disease cost is passed on to consumers. If by a national disease control strategy for BPS beef output is increased, market prices will tend to fall, with a subsequent transfer of some of the benefit of disease control from producers to consumers.

However, with UK Government and EEC intervention, prices to producers may largely be maintained by (i) payment of the beef variable premium (ii) intervention buying of beef. Thus producers appear to receive most of the benefit of increased output, consumers to remain largely unaffected (assuming market prices are maintained at previous levels) but the UK Government and FEOGA (the EEC common fund) experience a cost, which is ultimately borne by UK taxpayers to a large extent. Other costs and benefits are also involved, but this gives an example of how effects on supply may impact on society. In order to appreciate the magnitudes and importance of such costs and benefits and welfare changes, some quantitative analysis would be required.

LIMITATIONS OF THE ANALYSIS

We are well aware of the many shortcomings of this exercise; of the paucity of reliable epidemiological data; of the crudity of our attempts to quantify disease costs (cf Howe, 1985); of the need for better models for predicting the direct effects of disease control programmes (cf Beal, 1980).

We have ignored/disregarded the fact that no licensed BVDV vaccine of high potency is currently available in the UK. Alternative options for dealing with this situation will include licensing existing vaccines, developing new vaccine(s), using carrier animals to spread infection, practising "feed-back" and soldiering on.

Likewise, we have assumed that the testing required for the active control strategies would all be carried out at static prices with neutral effect on laboratory resources; and we have not attempted any evaluation of investment in R & D to produce cheaper and/or better tests.

These and other decision analyses are, of course, complementary to the aspects of BPS control that we have looked at and indeed they are essential to proper/effective SCBA in this field.

RESULTS OF ANALYSIS

The direct costs due to the "disease" effects of BPS in the UK are estimated by this analysis at over £47 million per annum.

The 'Test and Eliminate' strategy for disease control is estimated to result in a Net Present Value (NPV) of nearly -£8 million and thus represents a net cost to society.

The 'Vaccinate, Test and Eliminate' strategy for control results in a net cost of nearly £37 million and therefore also appears to be an economically unattractive strategy at the national (UK) level.

CONCLUSIONS AND COMMENTS

The results of this analysis appear to show that a national programme of an eradication nature (our 'test and eliminate' strategy) and the vaccination policy considered are both too costly in relation to the benefits derived from them. However, the benefit-cost ratio could be greatly improved by more selective strategies. For example, the vaccinate, test and

eliminate strategy could be confined to those UK herds which experience a significantly greater than average loss from BPS and thus a greater level of disease costs as a result. The break-even point in our calculations for such a strategy, would be for that percentage of herds which overall experience an average BPS incidence rate/disease cost of just over twice (2.1) the average national incidence/disease effects. This calculation depends on a number of assumptions, including the ability to monitor disease incidence in herds. However, the general principle remains, that limited policies directed towards 'problem' herds in the UK cattle population may result in positive net benefits for society, where more blanket policies do not. Such limited programmes are likely to have a much higher benefit-cost ratio, would also benefit those individual producers experiencing the greatest risk from BPS disease effects and should therefore be worth further investigation.

In conclusion, to the enlightened, these comments may seem like a blinding revelation of the obvious and the familiar (cf Ellis, 1982); but they may also give some of us cause to ponder why, in this country, we still continue to rely so heavily on subjective value judgements alone in choosing animal disease control policies.

Clearly, further work needs to be carried out on the economic consequences of the Bovine Pestivirus Syndrome and on possible strategies toward this disease complex. In particular, and as a matter of urgency, more reliable and detailed epidemiological information is required in a usable form. This requires an inter-disciplinary approach, whereby epidemiologists and agricultural economists can cooperate to produce adequately accurate estimates of the effects of disease or of disease control strategies on the production process.

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CONTROLLED RELEASE GLASS FOR DRUG DELIVERY IN BEEF PRODUCTION SYSTEMS

W. M. ALLEN* and C. F. DRAKE§

To ensure that veterinary medicines, such as parasiticides, vaccines and growth promoters and essential micronutrients are administered to domestic animals in the most economic and productive way, it is necessary to consider such factors as their route and frequency of administration, the duration of their biological effect, and their potentially toxic side-effects. This paper concentrates upon the development of one system, for administering drugs to beef cattle, which can overcome some of the disadvantages of conventional methods. These disadvantages are illustrated in Table 1 by comparing the properties of a conventional single-dose system for delivering a drug or nutrient with the properties of an "ideal" system.

An ideal delivery system would provide an effective concentration of the active substance for a sufficient period and would avoid excessive concentrations, it would avoid premature inactivation and excretion of the drug and it would target it to specific sites or specific receptor cells.

Table 1. Properties of conventional single-dose delivery systems contrasted with the properties of an "ideal" delivery system

"Conventional"	"Ideal"
Short duration of action	Lasting duration of action
High peak concentration	Lower peak concentration
Much of dose wasted through excretion	Little of dose wasted through excretion
Not targeted to specific sites	Targeted to specific sites
Expensive	Cheap

The disadvantages associated with conventional systems of administering drugs, usually as a single dose, are a result of the kinetics of absorption and dispersal of the drug after its administration.

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For example, a drug administered subcutaneously will be released from the site of administration at a rate which depends initially upon the quantity administered, its physico-chemical characteristics, its solubility in tissue fluid, and the nature of the site. Its rate of release will follow an approximately exponential pattern (see Fig. 1a) when the drug is released at a rate proportional to the amount remaining at the site. In these circumstances the concentration of the drug in the blood rises to a peak shortly after administration and may even reach a toxic level. It is transported in solution in the blood, either "free" or "bound" to the plasma proteins, to its site of action; in the case of antibacterial drugs, to the site of infection and, in the case of hormones, to the specific receptor sites in the hormonally-dependent tissues. Simultaneously, however, the drug will either be being metabolised, especially in the liver, or excreted through the kidney. Metabolism usually inactivates the agent, and excretion removes it from the circulation. The rates of inactivation and excretion will determine the effective biological life of the agent. Similar considerations apply to oral and topical routes of administration.

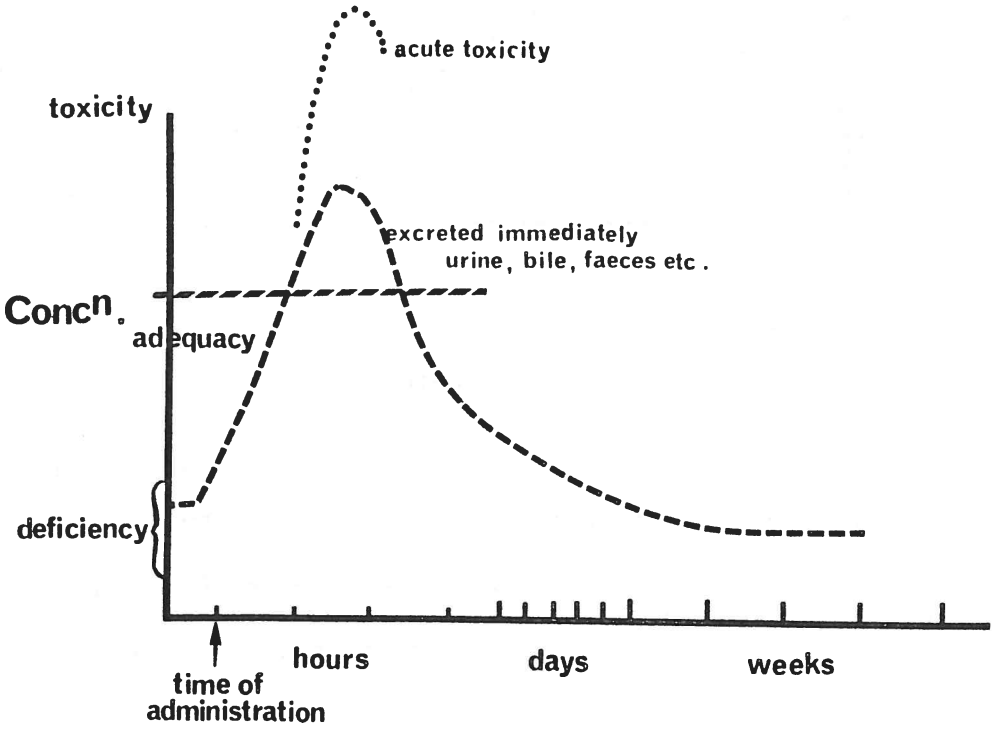

Figure 1a. Uptake of drug into blood following conventional therapy

y axis concentration in blood x axis time

Other factors which influence the absorption and efficacy of drugs after intramuscular, subcutaneous or intramammary administration have been summarised in a review by McDiarmid (1983). These factors include the relative solubilities of the drug in water and lipid, which can be influenced by the solvent or carrier used, the surface area of the substance (which depends upon its particle size), its diffusion properties through tissues and the inclusion of agents which affect local physiological activity (e.g. vasoconstrictors).

The objective of controlled release systems is to modify the rate of release of a drug so that it becomes either constant for periods of days, months or even years (see Fig. 1b), or pulsatile at predetermined intervals, or variable to suit particular requirements. As a result therapy can be provided virtually continuously without the need for repeated administrations which are difficult in practice for animals under extensive husbandry conditions.

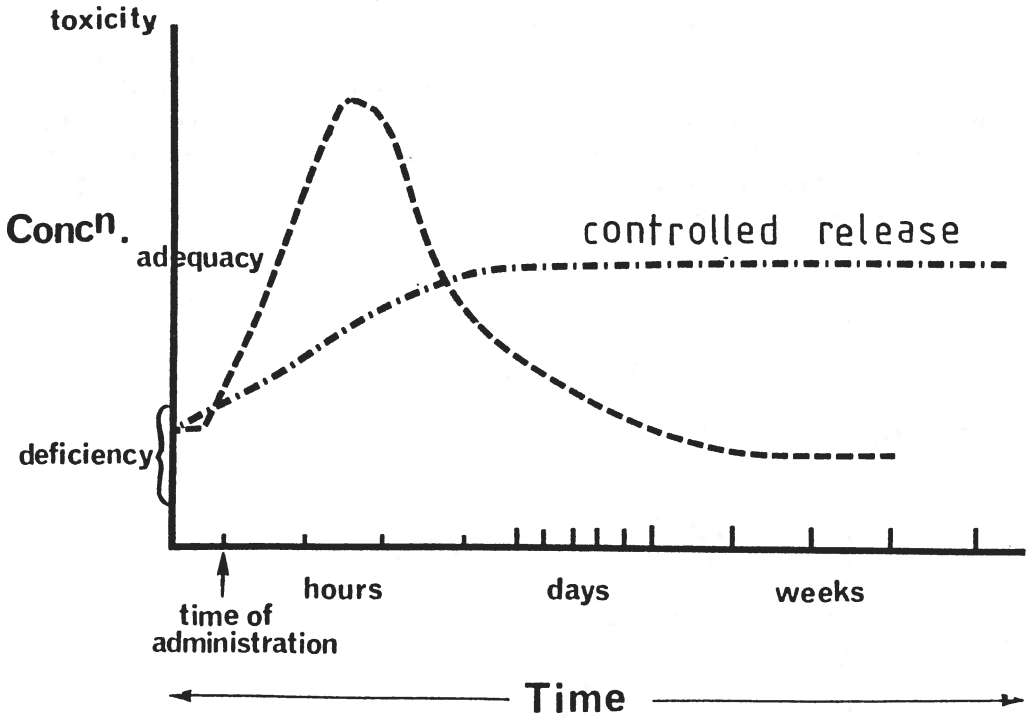

Fig. 1b. Uptake of drug into blood using controlled release techniques and in comparison with conventional techniques

Controlled release systems have already been successfully applied to a number of agents used in veterinary medicine, for either topical, oral or parenteral use, and some of them can be adapted to provide a pulsatile release.

Medication of the drinking water through an automatic dispensing unit has also been described as a means for the continuous controlled delivery of substances including trace elements and anthelmintics (Macpherson, 1983; Downey & O'Shea, 1985) but this method can be used successfully only when drinking troughs provide the only water supply; it will not be discussed further in this paper.

CONTROLLED RELEASE SYSTEMS SUITABLE FOR USE IN BEEF ANIMALS

A. External route of administration

In some areas of Britain the headfly, Hydrotea irritans, is a common ectoparasite of sheep. There has been partial success in protecting sheep from this fly by spraying them with a synthetic pyrethroid, 'Permethrin', but unfortunately the protection is short-lived and repeated treatments are necessary.

Appleyard (1982) and Appleyard et al. (1984) fitted sheep with polyvinyl chloride (PVC) tags which were impregnated with either 8.5% cypermethrin w/w (Shell Chemicals), or 10% w/w permethrin (Coopers Animal Health), or 8.5% w/w fenvalerate. The tags were tied to the base of the horns with twine so that they lay on the poll of the sheep.

Treatment with either cypermethrin or permethrin tags protected the sheep against headfly damage for several weeks longer than a single spraying and the benefit was also conferred on lambs born to the treated ewes. Fenvalerate did not protect the sheep.

The activity of the tags depends upon the insecticide diffusing slowly outwards and coming into direct contact with the headfly. After the surface layer of insecticide has been released, more has to diffuse from the interior of the tag and, as a result, the initial rapid rate of release is followed by a gradually declining rate.

The same principle has been applied successfully to the control of Gulf Coast ticks, hornflies and other face flies on beef cattle in the USA and elsewhere.

B. Oral route of administration

The ruminant's characteristic rumen and diverticular pouch, the reticulum, has proved to be a perfect "home" for several types of controlled release devices.

Boluses: More than 20 years ago it was discovered that boluses, or bullets, containing cobalt or made of a magnesium alloy with a density greater than 3.0, were retained in the reticulo-rumen of sheep and cattle. The cobalt bullets contained 30% CoO and 70% iron grit, the magnesium bullets were composed of a Mg/Al/Cu alloy (86, 12 and 2 by weight respectively), and were weighted with iron shot. The dimensions of the bullets for cattle were approximately 2.5 cm diameter x 7.6 cm long, and for sheep 1.9 cm diameter x 4.6 cm long (Coopers Animal Health and Agrimin Ltd.). In the rumen they slowly dissolved and released useful amounts of Co or Mg into the gastrointestinal tract. Similar boluses containing metallic selenium have also been fabricated.

The rate of solution of all these boluses generally decreases rapidly. Nevertheless they protect sheep against the development of cobalt and selenium deficiency for several months. They are rarely adequate for cattle (see Fig. 2).

The rate of solution of the boluses may be reduced either by the accretion of calcium salts onto the surface or by the formation of other insoluble salts on the surface (e.g. FeS in the case of the selenium bolus).

The magnesium boluses were designed to protect cattle for short periods during the spring and autumn when the risk of hypomagnesaemia is greatest. The requirements of sheep and cattle for cobalt and selenium are only a few mg daily, but the requirement for magnesium is more than a thousand times greater ($\geq$3 g/day). It is therefore not surprising that the magnesium bolus rarely provides adequate protection against the onset of hypomagnesaemia. In one experiment a Mg bolus weighing 240 g released less than 300 mg of magnesium during the first 30 days after administration (Allen, unpublished).

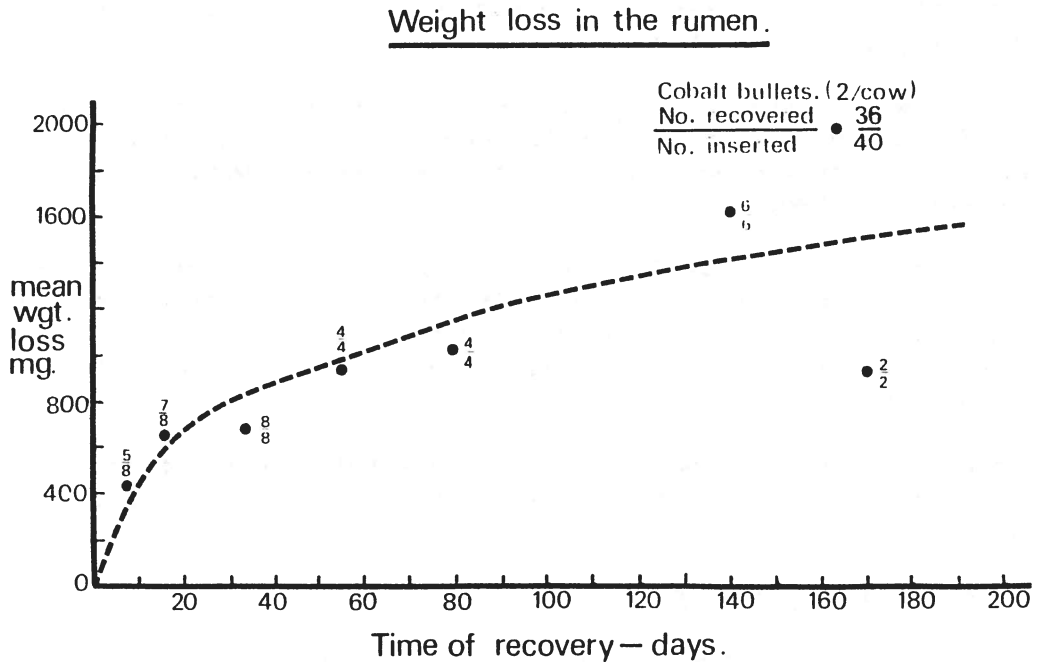

Fig. 2. The dissolution rate of cobalt boluses in cattle

y axis weight loss x axis time after insertion

Needles: More recently small dense needles of copper oxide (Copporal, containing 4 g Cu for sheep, Beecham Animal Health) approximatley 4 mm in length, have been administered to sheep and cattle. A high proportion of the needles lodge in the stomachs - mainly in the abomasum - and they slowly dissolve and release copper for 30-40 days after dosing. The copper can be absorbed and stored in the liver, and protects the animals against copper deficiency for several months (Macpherson, 1983). The dose recommended for growing cattle is 20 g of the needles.

Boluses fo control of endoparasites: A controlled release system was developed during the late 1970's to deliver morantel tartrate directly into the gastro-intestinal tract of cattle (Jones, 1983). A cylindrical metal bolus (Paratect bolus, Pfizer Ltd.), approximatley 10 cm long x 3 cm diameter is administered by balling gun into the reticulo-rumen. The bolus is loaded with 13.5 g of morantel tartrate in polyethylene glycol. The morantel tartrate escapes across a cellulose acetate hydrogel membrane at a constant rate of approximately 90 mg/day for the duration of the grazing season. Many field trials have demonstrated that this bolus protects cattle against the build-up of a significant worm burden.

One disadvantage of the Paratect bolus is that the metal container remains in the rumen for the animal's lifetime. A further possible disadvantage is that parasites which have been exposed to low concentrations of the parasiticide for long periods may develop resistance. However, there is no evidence that resistance has developed.

Mechanical systems for delivery of rumen-active growth promoters: Watson & Laby (1978) described novel boluses for supplying monensin (Elanco Products Ltd.) continuously to grazing ruminants for 6 weeks. The boluses consisted of a metal cylinder containing a spring loaded plunger which held the active substance against a fatty acid plug which was gradually eroded by the action of the rumen bacteria. The system has not been applied commercially.

Delivery of essential trace elements from glasses and cements: More recently new types of bolus have been developed for the controlled release of trace elements to ruminants. They are based on either water soluble glasses (CRG; Drake, 1985) or water soluble cements (Manston et al., 1985; Manston & Gleed, 1985). The cements will not be discussed further.

The soluble glasses were developed in the Standard Telecommunications Laboratories from the early 1970's initially as controlled sources of inorganic ions (Drake & Graham, 1976). They are phosphate glasses in which the cations, which can be any electropositive element, may constitute up to about 65 mole % of the glass.

PROPERTIES OF CRG

All the glasses consist of a three-dimensionally linked, polyanionic framework within the interstices of which the cations are distributed. The anionic constituent is the phosphate polyanion and the cations can be any of the elements of Groups I and II of the Periodic Table, in particular Na, K, Mg, Ca and Zn, and the transition elements.

The glasses are prepared by melting the oxides of the constituent elements at about 1100°C, casting the molten glass, and annealing the product. They dissolve completely in aqueous solutions, the rate-limiting step being the rupture of the cross-linking bonds of the phosphate framework. As these bonds break polyphosphate anions together with their associated cations are transferred to the solution.

The consequences of this mechanism of solution are as follows:-

1. The rate of solution is proportional to the surface area exposed and the cations can therefore be released at an almost uniform rate for long periods.

2. In glasses composed of a monovalent alkali metal oxide, a divalent metal oxide and phosphorus pentoxide, the rate of solution is reduced as the concentration of the divalent metal oxide increases. Trivalent metal oxides produce an even greater reduction in the rate of solution.

3. The rate of solution varies with the pH of the aqueous phase and CRG's can be formulated whose rate of solution depends upon pH in a predetermined way.

4. The rate of solution approximately doubles for each 10°C rise in temperature.

5. The constituents of CRG are commonly present in normal living tissue and should not cause adverse reactions. Evidence to support this view has been reported by Allen et al. (1978) and Burnie et al. (1981). The latter implanted

monolithic and sintered CRG into soft tissue and into cavities in bone. The absence of significant reactions at the sites of implantation and the healthy character of the new tissue which replaced the CRG demonstrated that they are an acceptable basis for release systems for parenteral use.

Phosphate based glasses have been made with a wide range of solubilities so that the constituent elements can be released at a rate appropriate to meet a particular animal's requirements. The glass is completely soluble so that there is no core or residue left after treatment. The trace elements are released at an almost steady rate for periods up to one year (see Fig. 3).

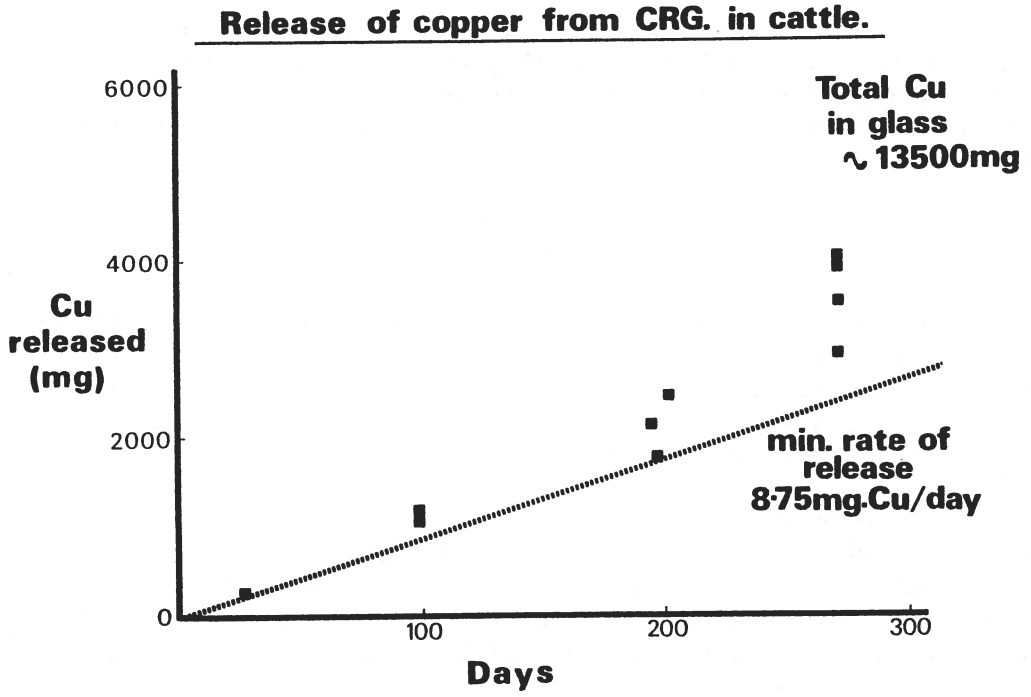

Fig. 3. Release of copper from CRG into the rumen of cattle

y axis amount of copper released x axis time

In a series of trials Allen et al. (1984, 1985) demonstrated that a single bolus weighing approximately 75 g or 15 g can supply the needs of cattle and sheep respectively for Cu and Co.

The Cu-containing CRG bolus prepared for cattle contained 18% by weight of copper and was 2.5 cm in diameter x 5 cm long. Its density was 2.8. The Co-containing CRG bolus contained 1.8% cobalt by weight and its dimensions were similar to those of the copper bolus. Its density was approximately 2.6. The boluses were administered by balling gun, and control animals were left untreated. The solubility of the boluses was adequate to supply a large proportion of the recommended daily dietary allowances of the elements (Agricultural Research Council, 1980). The cobalt boluses released a minimum of 0.85 mg cobalt/day and the copper boluses released between 9.0 mg and 14.5 mg copper/day (Fig. 3).

The cattle were slaughtered at intervals after dosing and the boluses were recovered from the forestomachs, washed, dried and weighed. Blood and liver samples were collected at slaughter and the concentrations of copper or vitamin B12 were estimated.

All the boluses were recovered from the 26 cattle dosed with the Co-CRG; after the administration of Cu-CRG boluses to 28 cattle 26 were recovered up to 276 days after dosing. In all the experiments in which it has been possible to search the reticulo-rumen at slaughter more than 90% of the administered boluses have been recovered up to 1 year after dosing.

In a series of experiments on a farm where a primary copper deficiency had been previously demonstrated (Gleed et al., 1983), and where copper therapy had produced a growth response (Fig. 4), the administration of Cu-CRG significantly increased the concentration of copper in plasma and liver (control group, plasma 12.7, s.d. 3.65 micromoles/l, liver 4.78, s.d. 1.35 mg/kg fresh weight, n = 8; treated group, plasma 18.1, s.d. 3.76 micromoles/l, liver 17.4, s.d. 12.3 mg/kg fresh weight, n = 12, $p<0.01$).

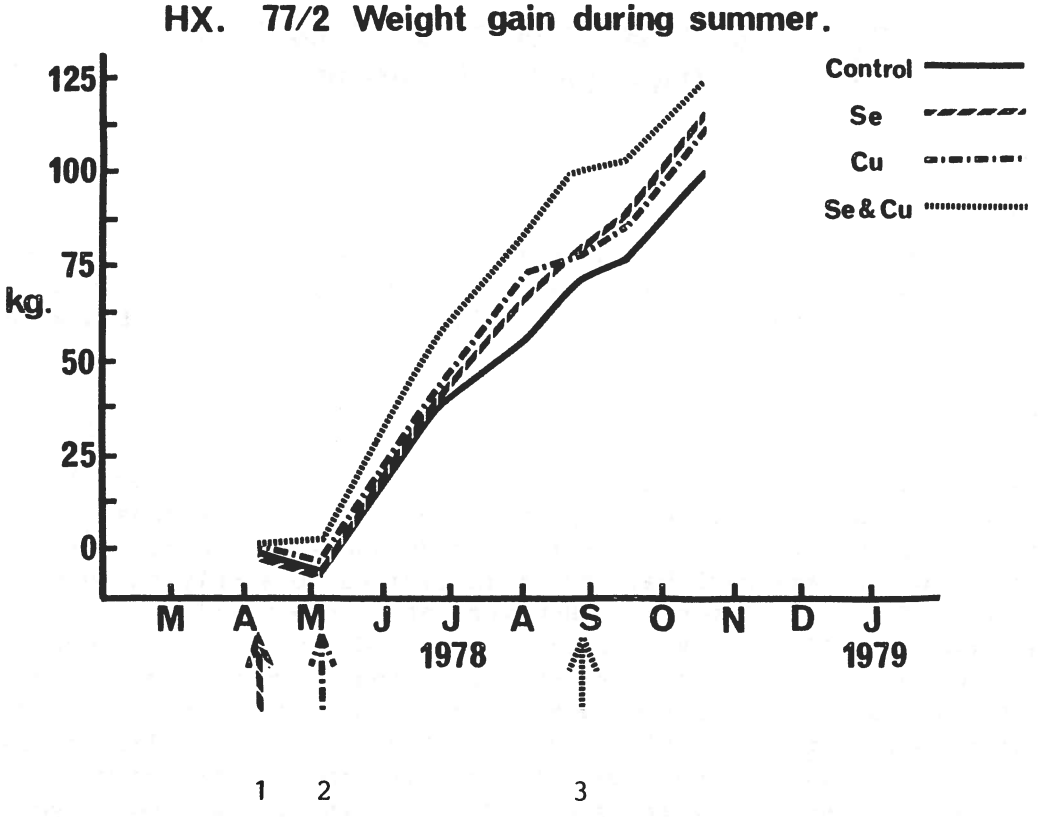

Fig. 4. Weight gain of beef cattle supplemented with Cu and Se

y axis weight gain x axis time, months March 1978-June 1979

1. Se supplement. 2. Cu supplement. 3. Cu and Se supplement.

In previous years it had been necessary to administer parenteral doses of copper at intervals of 3.5-4 months to maintain adequate blood copper concentrations. The single bolus of controlled release glass provided enough copper for at least a year, avoided the need for repeated administrations and avoided the risk of toxicity associated with large single doses of Cu administered parenterally (up to 200 mg Cu as a single dose; Fig. 5).

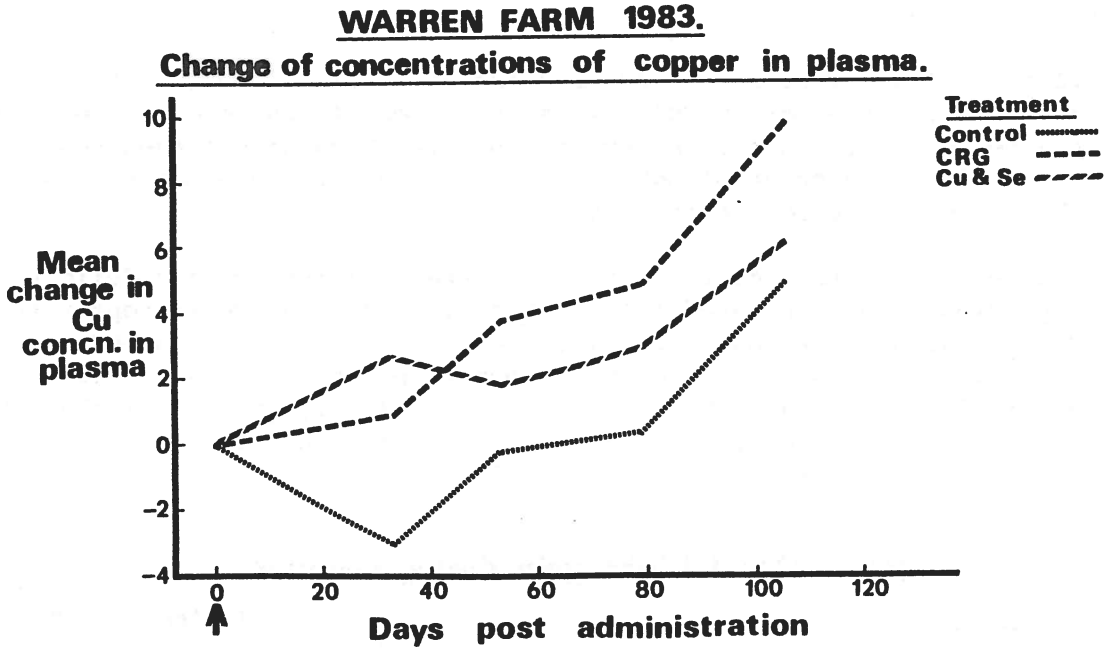

Fig. 5. Changes in the concentration of Cu in plasma after treatment with either copper CRG (1-75 g bolus) or a single parenteral dose of 200 mg copper (as the acetate) plus sodium selenate (0.15 mg Se/kg bodyweight)

CONCLUSIONS

Controlled release glasses are cheap to produce and they are likely to be developed rapidly worldwide. A phosphate based controlled release glass which contains cobalt, selenium and copper is already available commercially (Cosecure, Cooper Animal Health), and it is claimed to supply these three trace elements for several months. However, after one year's use in the field it appears to be failing, on some farms, to provide sufficient copper to prevent copper deficiency. This is not surprising since the published figures suggest that in cattle it releases a maximum of 13 mg of copper/day. Our observations (Allen et al., 1984) suggest that although up to 14.5 mg Cu/day can protect cattle against the onset of a simple copper deficiency it is unlikely to protect animals grazing pastures rich in sulphur and molybdenum. Nevertheless it should not be difficult to fabricate glass boluses which release enough copper to protect against such severe secondary copper deficiency.

THE FUTURE

The application of controlled release delivery techniques to problems related to animal health and production could advance significantly in the next decade. New developments can be expected for the delivery of agents such as growth promoters, the trophic peptide hormones and immuno-stimulants and other immuno-modulants. Their application for the administration of parasiticides

is also likely to expand. Morantel has already been used successfully but the Avermectins probably have an even greater potential because of their potency at low concentrations.

Controlled release techniques provide new opportunities for improving the methods for the treatment and prevention of disease and they can help to increase the efficiency of animal production. They will complement and encourage the development of the new products of bio-engineering, particularly the peptides, vaccines and hormones. They provide excellent prospects for improving the health and productivity of the world's animal population.

ACKNOWLEDGEMENTS

This report describes projects completed in collaboration with the Milk Marketing Board, the Institute for Research on Animal Diseases, Compton and the Standard Telecommunications Laboratories, Harlow.

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HILL AND UPLAND BEEF CATTLE PRODUCTION : SOME NUTRITIONAL CONSIDERATIONS

A.J.F. RUSSEL*

Most of the beef produced in this country comes from the dairy herd with only about one-third being produced from the national beef herd. The imposition of milk quotas and the introduction of incentives to milk producers to leave the industry will, in the long term, reduce the number of cull cows and dairy-bred calves available for slaughter. This is likely to lead to a renewed importance of purpose-bred beef animals, the majority of which have their origins in hill and upland farms, even if in many cases the final products are sold for slaughter from lowground enterprises.

A decrease in the supply of beef from the dairy herd is not, however, enough in itself to ensure the economic viability of beef enterprises on hill and upland farms. Beef production is generally less profitable than sheep production, partly because of differences in the rate at which returns from sheep and beef have increased in recent years, and partly because of the high capital and recurrent costs associated with keeping suckler cows. Feeding costs generally represent more than 80% of the total variable costs of producing a weaned suckled calf. It is therefore pertinent to consider the nutritional management of beef cows and calves on hill and upland farms and particularly as regards opportunities for reducing feeding costs without unduly penalizing production.

HILL FARMS

Beef cattle enterprises on hill farms are usually of secondary importance to the hill sheep flock and consist almost entirely of suckler herds producing weaned calves for sale. Cow numbers are in most cases small, being limited by the amount of fodder which can be conserved for winter feeding from the small proportion of cultivatable land. This varies widely according to land and vegetation types but is seldom more than 10% of the total acreage and is often only 1-2%. At one time hill suckler herds were predominantly spring-calving. Limited supplies of winter feeding decreed that the calf crop was sold in the autumn and all too often the sale of these young and light-weight animals failed to realise a price which adequately recompensed the breeder. In an attempt to improve the quality and increase the weight of the calves at the autumn sales the date of calving would be advanced, often without a proper appreciation of the increased costs of feeding the lactating cow throughout the winter and early spring. Now, Meat and Livestock Commission recorded hill herds contain fewer spring (March-May) than autumn (September-November) calving herds, although in the majority of cases calving is spread over more than one season (MLC, 1985).

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UPLAND FARMS

On upland farms where usually at least 50% of the land is enclosed and comprises sown or permanent pasture, cattle enterprises more often exist in their own right and not as secondary to sheep flocks. Many such farms have associated areas of rough grazing which are utilised by cattle at certain times of the year. In general, these cattle enterprises make a greater contribution to the overall economy of the farm business than in the true hill areas. There are greater opportunities on upland farms for more varied types of cattle enterprises, including the rearing of purchased calves and the finishing of stores, but the sale of calves from the suckler herd again represents the most common form of production.

The size of the suckler herd on upland farms is invariably larger than on hill farms and returns per cow are generally greater. Costs, however, also tend to be higher with the result that the difference in gross margin per head is not great (MLC, 1985). Spring and autumn calving systems are represented in equal proportions in MLC recorded herds. Gross margins per cow are, in general, greater from autumn calving systems where calves are retained for longer periods, but on a per hectare basis gross margin is little affected by season of calving.

WINTER NUTRITION

The use of body reserves

Most systems of weaned suckled calf production seek to use the cows ability to draw on body reserves of fat and protein to maintain production during winter when feeding is in limited supply and expensive. There is ample evidence, reviewed by Russel and Broadbent (1985), that low levels of winter feeding, whether to pregnant or lactating cows, need not incur any biologically significant production penalties, provided always that body reserves are adequate at the outset, that the severity of undernutrition is regularly monitored, and that the system of management allows the animals opportunity to replenish in full during the subsequent grazing season the body reserves utilised during the winter.

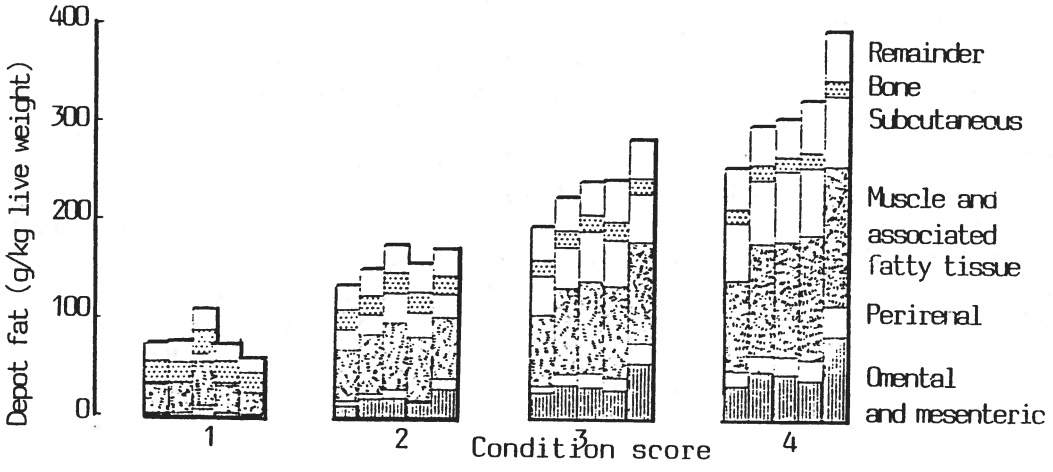

Fig. 1. The partition of fat between depots and total fat as a proportion of live weight in Hereford x Friesian (HF), Galloway (G), Blue Grey (BG), Luing (L) and British Friesian (F) cows at different condition scores (from Wright & Russel, 1984).

The adequacy of reserves of body fat and protein are most commonly judged from subjective assessments of cow body condition (Lowman, Scott & Sommerville, 1976) and work by Wright and Russel (1984) has confirmed that condition score can be used as an index to predict body composition with a degree of precision acceptable for most practical purposes. As shown in Figure 1, there are important differences between genotypes in the total quantity and distribution of body fat at any particular condition score. There is, however, a sufficient similarity between the suckler cow genotypes studied to allow some general statements to be made.

Suggested patterns of change in body condition throughout the year for spring and autumn-calving suckler cows on upland and hill farms are illustrated in Figure 2. On upland farms cows calving in, say, March, and in condition score 3.5 at the beginning of winter can safely lose one unit of body condition during late pregnancy and a further half condition score between calving and turnout. On many hill farms the rate of replenishment of body reserves which can be realistically achieved from the relatively poorer summer grazings necessarily constrains the extent of use of these reserves during winter. In these situations an amplitude of around one unit of body condition, from 3.0 to 2.0, would be more appropriate.

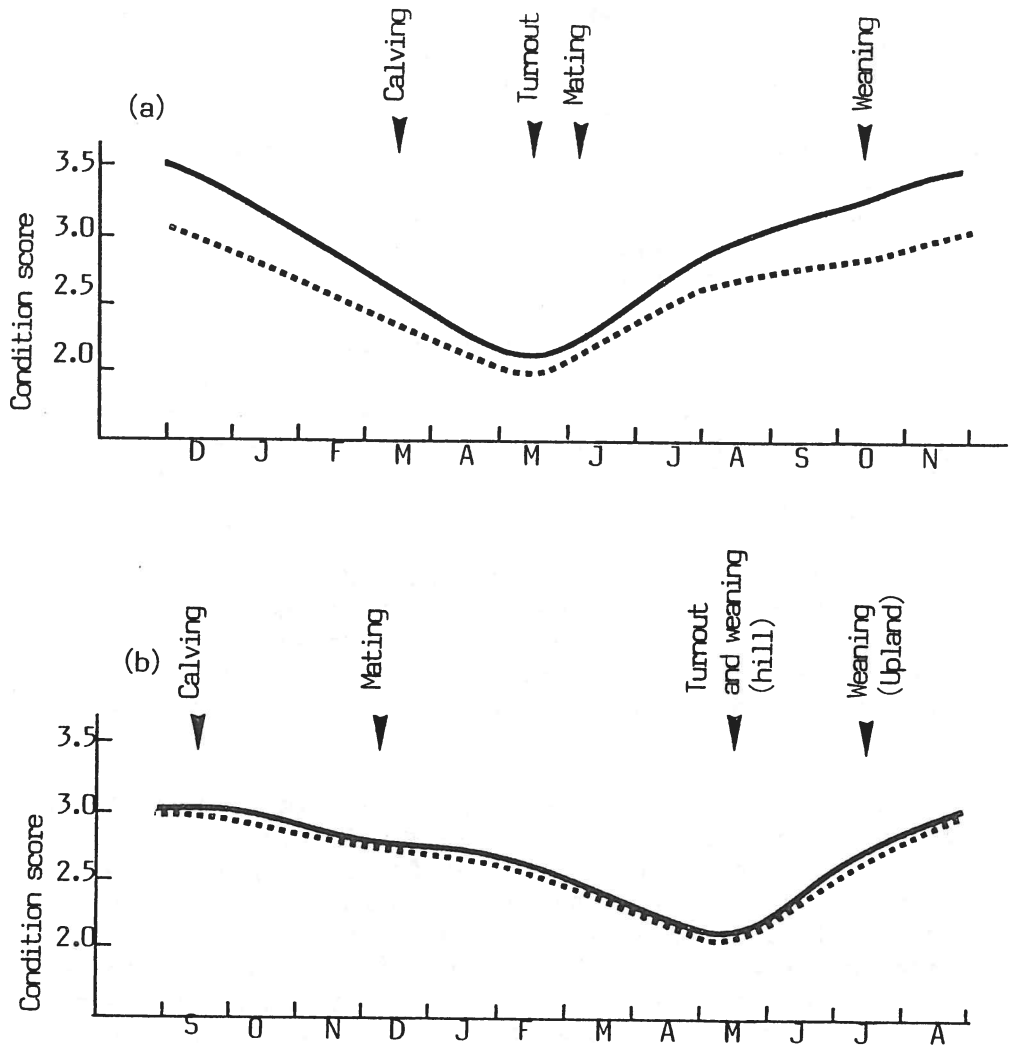

Fig. 2. Suggested patterns of change in body condition score throughout the year in (a) spring-calving and (b) autumn-calving suckler cows on upland (——) and hill (– – –) farms (from Russel & Broadbent, 1985).

The constraints on the use of body reserves in autumn-calving cows are, firstly, that to avoid calving difficulties cows should not be in a body condition score of more than 3.0 at parturition, and second, that the degree of undernutrition during the periods of rebreeding and implantation should be minimised. Ideally cows should be fed to meet in full the requirements for maintenance and the contemporary level of milk production from before the beginning of mating until one month after rebreeding is complete. In practice, however, a somewhat lower level of feeding resulting in a small and controlled loss of condition is commonly regarded as acceptable. The transition to a lower level of feeding and greater dependence on body reserves thereafter must be made gradually. As with spring-calving cows, a condition score of 2.0 at turnout is appropriate for both hill and upland autumn-calving cows.

Suggested levels of feeding

The calculation of levels of energy intake which will result in the patterns of condition-score change suggested above require a knowledge of the amount of energy contributed by the utilisation of body tissue and, since tissue energy is used more efficiently than dietary energy, of the quantities of feed which tissue mobilisation can replace. As an example, consider spring-calving cows in late pregnancy, condition score 3.5, weighing 590 kg, with a maintenance requirement of 54 MJ ME/day (Russel & Wright, 1983a) and a mean daily requirement for foetal growth over the final 150 days of pregnancy of 12 MJ ME (based on ARC, 1980). This gives a total daily energy requirement of 66 MJ ME. If, during this period, such cows lose one unit of body condition, i.e. condition score drops from 3.5 to 2.5, Wright & Russel (1984) have calculated that this is associated with a change in body energy of some 2240 ME. Since body energy is used for foetal growth more efficiently than dietary energy this will supply the equivalent of 3000 MJ dietary ME, i.e. an average of 20 MJ ME/day.

It can therefore be estimated that the dietary energy input required to achieve such a prescribed change in condition is some 46 MJ ME/day or about 70% of the amount which would be required to prevent any loss of body condition. The economy in feeding stuffs achieved from the use of body reserves is thus of the order of 30% or the equivalent of about 0.4 t of hay, 1.3 t of silage or some 270 kg of barley.

Cows calving in condition score 2.5 can safely be fed to lose a further 0.5 unit of body condition over the eight weeks or so to turnout. It has been calculated (I.A. Wright, personal communication) that an energy intake of some 70 MJ ME/day will result in a reduction in condition score from 2.5 to 2.0 over the first eight weeks of lactation in a cow producing 9-10 kg milk per day.

These types of calculation have been used to produce suggested levels of energy intake during periods of dependence on conserved fodder, as illustrated in Figure 3. These suggested levels of feeding apply to the 'average' cow, but in practice there will be variation in body condition within any group of cows. To achieve a degree of uniformity in target condition score at, say, calving or turnout cows should be condition scored regularly and allocated between at least two feeding groups on the basis of body condition. Levels of feeding to groups above and below the appropriate mean condition score can be adjusted on the basis that a change in intake of

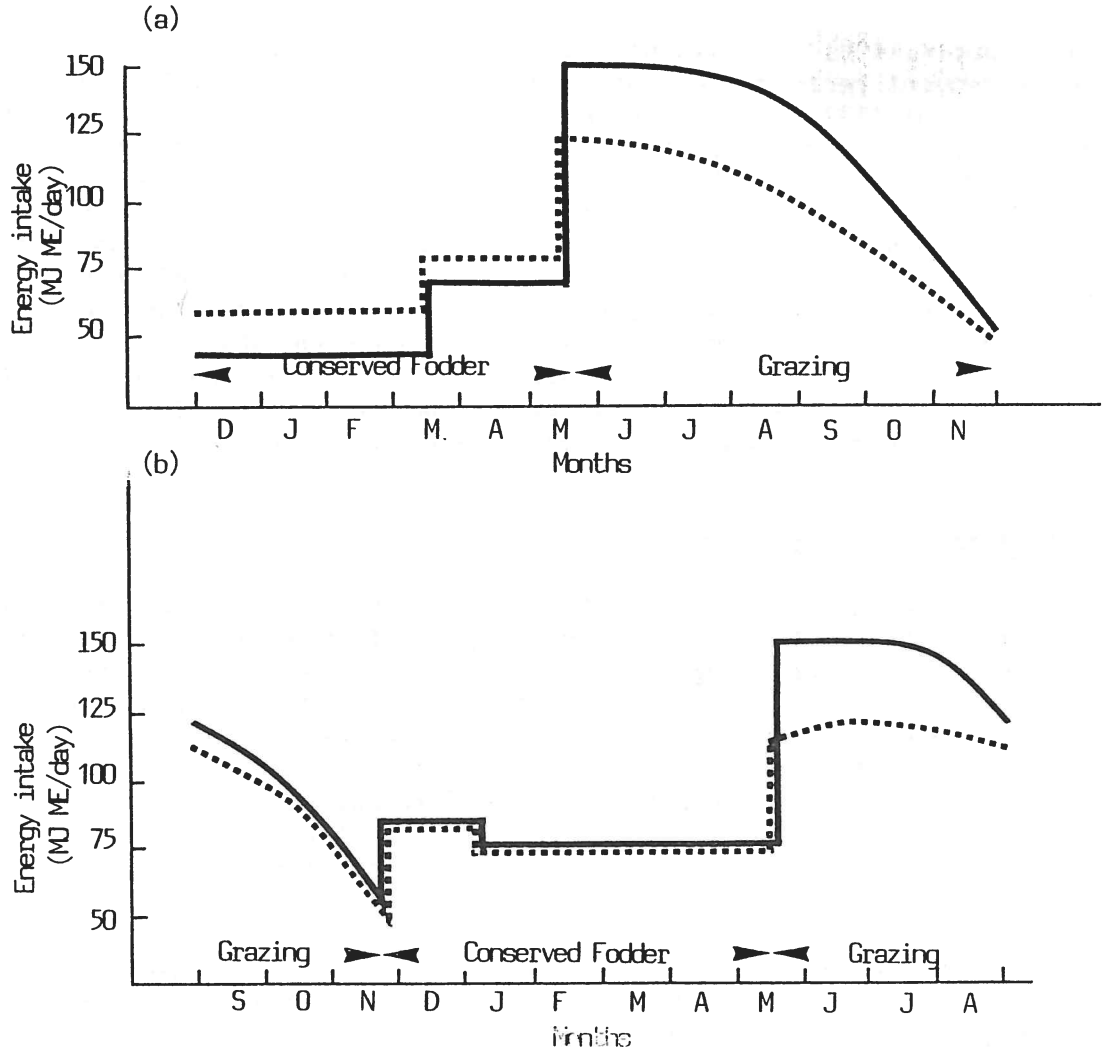

Fig. 3. Stylised patterns of energy intake throughout the year of (a) spring-calving and (b) autumn calving suckler cows on upland (———) and hill (- - - - -) farms (from Russel & Broadbent, 1985).

10 MJ ME/day will result in a difference in condition score of about 0.15 over a 100 day period (calculated from Russel & Wright (1983).

Consequences to production

It can be argued that any nutritional limitation must necessarily result in a production penalty. What is important in practice, however, is to have a quantitative knowledge of the relationships between nutritional adequacy and production, be that measured in terms of calf birthweight, level of milk production or calf growth rate. Such knowledge allows decisions to be made whereby the cost of the reduction in, say, level of milk production and therefore also in calf growth rate, can be balanced against the cost of the additional feeding which would be required to prevent that loss of production.

The suckler cow has a considerable ability to buffer production by utilising body reserves during periods of underfeeding. Production penalties arising from even very low levels of energy intake are generally small and often of little biological significance. For example, levels of energy intake during late pregnancy which are less than the maintenance requirements of non-pregnant, non-lactating cows have been shown by Powell and Matravers

(1975) and Chapple (1981) to have had no detectable effect on calf birth weight of subsequent performance. Other workers, e.g. Hight (1966, 1968a & b) and Russel et al (1979) have noted reductions in calf birthweights of only 8-10% from levels of energy intake as low as 30 MJ ME/day during the final 12 weeks of pregnancy. Indeed, the somewhat reduced calf birth weights and moderate body condition at calving of cows which have been underfed in late pregnancy are likely to be beneficial in reducing the incidence of assisted calvings and consequent calf mortality. Similarly, the levels of feeding suggested in Figure 3 as appropriate for lactating suckler cows are lower than those required to meet the full costs of maintenance plus milk production and will inevitably depress milk production and consequently calf growth rate at the time. As discussed below, however, such production penalties can be minimised and in some cases completely overcome by subsequent nutritional management.

Assessment of the adequacy of nutrition

No matter how good the guidelines for the winter feeding of suckler cows before or after calving, there is always a degree of uncertainty in practice as to whether the cows are being fed as intended. Differences between animals in size, level of production and stage of pregnancy or lactation can all contribute to errors in estimating food requirements. Inaccuracies in estimating the quantities and nutritional value of the rations fed and differences in food consumption between individual animals contribute to doubts about levels of energy intake. There is therefore a need to monitor the adequacy of nutrition to ascertain the extent to which the particular nutritional objectives are being achieved.

In theory the simplest objective measure of nutritional adequacy is live-weight change. In practice, however, increases in the weight of the gravid uterus in pregnant cows, and in gut-fill in cows in early lactation, make changes in gross live weight difficult to interpret. The regular monitoring of body condition score, although subjective, is in practice a better guide on which to base nutritional management. As suggested above, cows should be grouped for feeding according to whether they are above or below the appropriate body condition and should be condition scored of regular intervals of 2-3 weeks to ensure that the desired rate of condition-score change is being achieved. By moving cows between groups and altering levels of feeding as indicated above it should be possible to achieve the predetermined target condition scores at strategic points such as calving and turnout, and to do so with a high degree of uniformity between animals.

The adequacy of nutrition can also be monitored by estimating the magnitude of the energy deficit from concentrations of circulating metabolites. It has been shown by Russel and Wright (1983b) that plasma 3-hydroxybutyrate and free fatty acid concentrations can serve as useful indices of energy status in suckler cows. Of these two variables, 3-OHB concentration is perhaps the more useful in cows in which there is a relative high glucose demand as in late pregnancy and during lactation. The relationship between plasma 3-OHB concentration and energy status, illustrated in Figure 4, is given by the equation:

$$y = -0.693 + 0.020\ (\pm 0.028)x + 0.001\ (\pm 0.0003)x^2 \quad (R^2 = 0.92)$$

where y = 3-hydroxybutyrate concentration (mmol/l)
x = energy status (MJ ME/day)

This relationship enables the magnitude of the energy deficit to be estimated

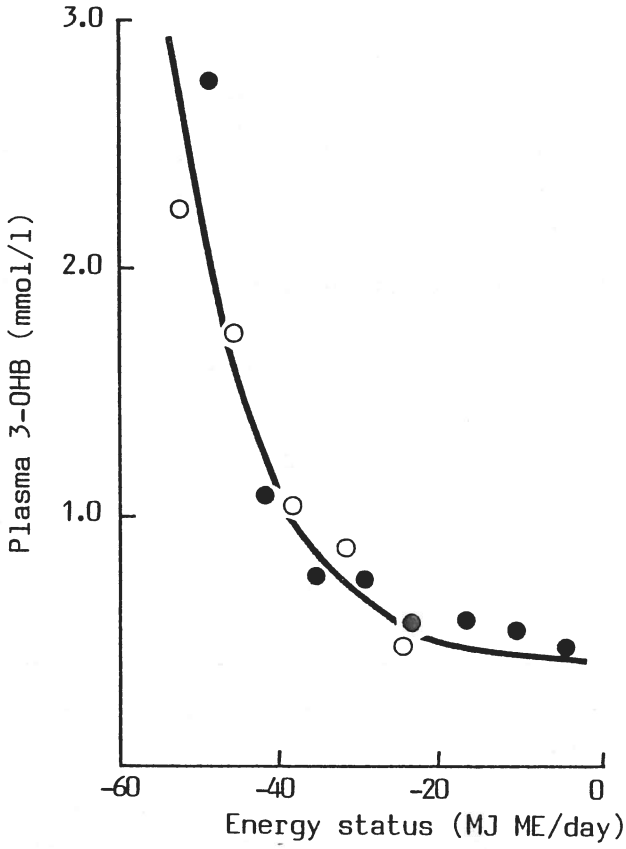

Fig. 4. Relationship of plasma 3-hydroxybutyrate concentration to energy status in housed cattle during late pregnancy; (year 1 (•), year 2 (O) (from Russel & Wright, 1983b).

relatively quickly, whereas changes in condition score take some time to become manifest.

NUTRITION DURING THE GRAZING SEASON

The performance of cows and their calves during the grazing season is determined by the quality and amount of herbage available, and to a lesser extent by the nutritional regime imposed during the winter prior to turnout. These factors all influence the nutrient intake of both cows and calves. The sward factors of herbage quality and quantity affect intake directly; the higher the digestibility the greater the weight of herbage ingested and, because each kilogram contains more nutrients, the effect of increasing quality on the intake of, say, energy is considerable. Similarly, and within wide limits, the greater the quantity of herbage available, the higher the intake. The effect of previous nutritional regime can be classified as an 'animal factor' and is considered to operated through effects of body composition on voluntary intake (Russel and Wright, unpublished), lean animals having higher intakes than animals in higher body condition.

The concept of managing the grazing of sown pastures by sward surface height is comparatively new. Responses in spring-calving cows in milk yield and cow and calf live-weight change to differences in sward height, as measured in a recent experiment (Wright & Russel, 1986), are presented in Table 1. These results indicate that the highest levels of individual animal performance are obtained on the tallest swards. However, when stocking rate is taken into account greatest output in terms of calf gain per hectare is

observed on the shortest swards. In the latter case, however, cow live-weight changes are unacceptably low and do not permit adequate recovery of body reserves. Consideration of both individual animal performance and output per hectare indicates that swards maintained at around 8 cm during the grazing season will support high levels of production from suckler cows and calves and ensure the full replenishment of body reserves necessary for viable year-round systems of management.

Table 1. Cow and calf performance in relation to sward height

Mean sward height (cm)	4.4	6.0	7.0	9.1	11.0	s.e.
Cow live-weight gain (kg/day)	-0.52	-0.05	0.32	0.75	0.40	0.118
Calf live-weight gain(kg/day)	0.88	0.91	0.98	1.04	1.06	0.048

Suckler cows have a considerable ability to increase milk production in response to the improved nutrition which invariably follows turnout, and to do so even in late lactation. The data in Table 2 show similar levels of increase of about 4 kg/day in milk production from cows calving in November-December and March-April when turned out to a fertilised ryegrass pasture in

Table 2. The effect of season of calving on increase in milk production following turnout

Season of calving	Nov-Dec.	March-April	s.e.d.
Milk production (kg/day)			
Prior to turnout	5.18	7.15	0.477
Following turnout	9.37	11.05	0.678
Increase	4.19	3.30	0.493

mid-May (Russel et al, 1984). Although the importance of milk production as a factor determining calf growth rate decreases with age, the increased production of the earlier calving cows still has a significant and beneficial effect on calf performance (Russel et al, 1979).

The levels of milk production illustrated in Figure 5 provide a further example of the increases following turnout and shows the effects of previous nutritional treatment (Hodgson et al, 1980). Cows wintered on a low nutritional regime and consequently in only moderate body condition at turnout show a greater increase in milk production subsequent to turnout than that measured in cows more generously fed during winter and therefore in higher body condition. Not only is the increase in milk production of the previously poorly fed cows greater, but the absolute level of production is maintained at a higher level over the duration of the grazing season. This

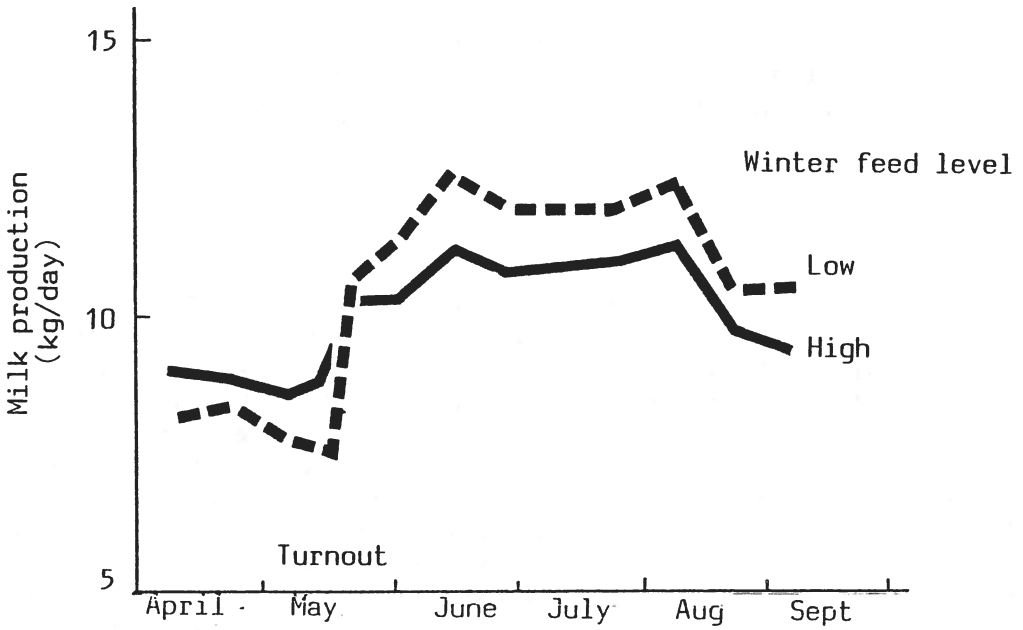

Fig. 5. Milk production of cows fed high or low levels of nutrition in early lactation (from Hodgson et al, 1985).

difference in production is reflected in calf growth rate; although the calves from cows on the low level of winter feeding are lighter at turnout, their superior rate of gain thereafter enables them to make good this early penalty by weaning. At the same time rates of cow live-weight gain are also greater and by September they attain the same live weight and body condition as animals better fed during the previous winter. It is postulated that these effects operate through an effect of body composition on voluntary intake, lean cows eating more than fatter cows and partitioning their 'marginal' intake between body tissue replenishment and milk production.

The herbage production of and nutrient intake from a range of hill plant communities grazed by cattle and sheep have been reviewed by Hodgson and Grant (1981) and more recent information on the same topics have been presented by Hodgson and Eadie (1984) and Grant et al (1985). Briefly, work to date indicates that the quality of the diets selected by cattle from Calluna and blanket bog (Eriophorum/Tricophorum/Calluna) communities is such that intakes are likely to be insufficient to meet maintenance requirements, let alone the needs of a lactating suckler cow. Cattle graze these communties less selectively than do sheep and ingest more dead material. Such grazing by cattle might be thought to improve the quality of the herbage for sheep, but Hodgson and Eadie (1984) conclude that on these particular communities cattle can cause significant damage by treading and by breaking and uprooting plants.

In Agrostis/Festuca, Nardus and Molinia communities the quality of the diet selected by cattle is also less than that ingested by sheep, but they are nevertheless able to consume during the summer months a quantity of material of reasonable quality (OMD c. 0.7) which Hodgson and Eadie (1984) calculate would support a level of milk production of 10 kg/day while also permitting some modest increase in live weight and condition. Hill suckler cows are not infrequently grazed on these communities with the express purpose of improving the quality of the pasture for sheep. The available evidence indicates that there is some substance in this contention with respect to Nardus communities, that cattle can perform a useful role and at

the same time obtain a reasonable quality diet from Molinia communities in the early summer, but that on Agrostis/Festuca areas they will not necessarily bring about improvement which could not be achieved through heavier stocking with sheep (Hodgson & Eadie, 1984).

COMPENSATORY GROWTH

The superior performance at pasture of lactating cows and their calves following a low level of nutrition during winter may be considered as analogous to the compensatory growth more often noted in growing and finishing cattle. Although the mechanisms have not been fully elucidated they are in all probability similar. Recent studies (Wright et al, 1983 & 1984) have examined the effects of varying levels of winter feeding of different ages and weights of weaned calves on performance on sown pasture during the subsequent grazing season. Calves weighing on average 275 kg were fed diets of silage and barley to provide energy intakes ranging from some 40-70 MJ ME/day over winter. These treatments resulted in growth rates ranging from 0.3 to 0.9 kg/day. Performance at pasture during the following summer was inversely related to rate of winter live-weight gain, as shown in Table 3. These data show that herbage mass also had a marked effect on summer performance and that the effects of winter treatment on summer

Table 3. The effect of low, medium and high levels of winter feeding and of sward height on performance at pasture of growing and finishing cattle

	Short sward			Tall sward			s.e.
Winter feed level	L	M	H	L	M	H	
Live-weight gain (kg/day)							
Year 1	1.10	1.02	0.87	1.35	1.23	1.19	0.060
Year 2	0.86	0.66	0.51	1.26	1.18	0.91	0.090

performance were evident at both sward heights. Measurements made in these investigations indicate that the nutritional treatments imposed during winter result in differences in body composition at turnout which influence both herbage intake and the composition of tissue gained during the grazing season (Wright & Russel, 1984; Wright et al, 1983 & 1984).

The evidence to date indicates that weaned suckled calves fed inexpensively during the winter to gain perhaps 0.3 to 0.5 kg/day will have significantly higher growth rates at pasture in the following summer than animals fed more generously and gaining weight more rapidly during winter. The compensation, however, is not likely to be complete and the animals fed better during winter will still be heavier at the end of the grazing season. Low levels of winter nutrition extend the time required to achieve a particular degree of finish and also the weight at which that is attained. Thus, the optimum level of winter nutrition for any particular age and weight of weaned suckled calf is likely to be determined by factors such as the availability and quality of the summer grazing and date of finishing which best suits the management and resources of the particular farming enterprise.

PREGNANCY DIAGNOSIS AND DETERMINATION OF FOETAL AGE

The early diagnosis of pregnancy in suckler cows enables the identification of fertility problems at both the individual animal and herd level and is an aid to the achievement of compact seasons of calving and short calving intervals. The accurate determination of foetal age is a valuable aid to management, allowing cows to be grouped for feeding according to expected date of calving.

Real-time ultrasonic scanning, which is becoming increasingly widely used to determine foetal numbers in sheep, can be used to diagnose pregnancy and estimate foetal age in cattle. Examinations are made rectally using a 3.5 MHz transducer. Echoes returning from tissue boundaries within the path of the beam are displayed as two-dimensional images on a small screen. The distinguishing features of pregnancy as diagnosed by scanning are the recognition the the fluid-filled uterus and the presence of the distinctive cotyledons and of the foetus.

Pregnancy can be diagnosed with a high degree of accuracy from 30 days post-conception by the imaging of the gravid uterus. At that stage the cotyledons are not developed and it can, on occasion, be difficult to locate the embryo. By 45-50 days the foetus, with a distinctive head and trunk with tail and limb buds, is readily recognised, and by 70-80 days, the cotyledons have developed their characteristic shape and appearance. After about 130-140 days of gestation the foetus may lie beyond the 20-25 cm depth of the ultrasound beam, but pregnancy can still be accurately and confidently diagnosed from the fluid-filled part of the uterus and the cotyledons which are still visible. At this stage and later it is also possible to diagnose pregnancy by external examination using the same instrument, placing the transducer towards the lower surface of the abdomen immediately in front of the udder and to the right side of the mid-line.

In one recent trial (White et al, 1985) 179 beef cows were examined rectally and 176 of the diagnoses agreed with the calving results. One cow which subsequently calved was diagnosed as non-pregnant and this was clearly an error on the part of the operator. Two cows which were diagnosed as pregnant failed to calve, but since predictions of stage of gestation were made in these cases it is probable that the diagnoses were correct at the time at that these foetuses subsequently died and were lost. The overall level of accuracy was therefore 98.3%. In another smaller trial with 98 cows the accuracy was 99%.

Foetal age can be estimated from measurements of foetal parts made either directly on 'frozen' images on the scanner screens of those instruments which have integral measurement devices or from photographs of images. Age is estimated by the use of specially developed equations. The frequency with which particular estimates can be made depends on the age and position of the foetus. In trials it was found that trunk diameter was the measurement made more often than any other and that crown-rump length gave the most accurate estimate of age ($\pm$ 3.1 days). In a trial with 96 cows the difference between the mean predicted and actual dates of calving was 0.3 days; 58% of cows calved within 3 days of the predicted date and 90% within 10 days.

Research work is currently in progress to increase the incidence of twinning in cattle. When this becomes a practicality it will be important to identify those cows carrying twin foetuses so that they may be fed and managed appropriately during late pregnancy. Preliminary studies have shown

that scanning can be used to detect twin-bearing cows with a high degree of accuracy.

CONCLUSIONS

In theory the feeding of suckler cows and growing beef cattle should present no problems. Nutrient requirements for different classes of stock and a wide range of levels of production are well documented (see e.g. ARC, 1980). Feeding cows or growing stock strictly according to requirements throughout the annual cycle will undoubtedly maximise biological efficiency as measured in terms of the inputs and outputs of energy and other nutrients. The principal objective of systems of cattle production, however, is to maximise economic efficiency and, in practice, this demands levels and patterns of feed inputs at different stages of the annual cycle which depart quite markedly from those designed to meet the requirements for maintenance plus current production. In practice this means feeding animals at levels less than full requirements during periods when fodder is scarce or expensive and at levels in excess of requirements at times when fodder is plentiful and less expensive.

The suckler cow has a considerable ability to draw on its body reserves of fat and protein to maintain production during periods of undernutrition and also to respond to increases in nutrient provision by increasing milk production and replenishing depleted body reserves. To capitalise on this capacity to buffer production at the expense of body tissue it is necessary to ensure that there is adequate provision within the systems of management for full replenishment of body fat and protein.

As a generalisation, it can be stated that the higher the standard of grassland management in the grazing season the greater the opportunity for economies in winter feeding. In practical terms this implies that on uplands farms with sown swards there is likely to be more scope for systems with large amplitudes of change in cow weight and condition throughout the year than on hill farms where the quality of summer grazing generally constrains the extent of tissue replenishment. This should not, however, preclude attempts to improve the quality of summer grazings for cattle on hill farms.

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ANIMAL WELFARE

ANIMAL WELFARE: IS AN EPIDEMIOLOGICAL APPROACH POSSIBLE?

R. EWBANK*

If the term animal welfare could be suitably defined it might then be possible to start quantifying welfare within animal populations, correlating it with aspects of the environment and studying its patterns of occurrence i.e. an epidemiological approach to animal welfare might be feasible.

There as yet seems to be no agreed definition of the term. My own working approach is to replace the word 'welfare' by the phrase 'health and well-being'. Both health and well-being however have positive and negative components. Health is more than the absence of disease and well-being is more than the absence of distress. The stockman recognises and delights in animals which are contented, productive and thriving. There is a pattern of signs, not easy to define but readily recognisable by the competent observer, which indicates a state of health and well-being. The position is complicated by the tendency of some people to make welfare assessments solely on aesthetic and moral grounds and for others to use a mixture of moral, factual and scientific evidence.

It is perhaps easier to identify and quantify the negative aspects of animal welfare than to struggle with the somewhat vague concepts of health and well-being. Ill-treatment of animals basically covers the actions (or inactions) by humans which cause animals to suffer. Ill-treatment can be divided into three main forms: abuse, neglect and deprivation (see Table 1).

Table 1. Results of ill-treatment of animals

Type	Symptoms	Effect
Abuse (deliberate)	Fear, injury, pain, distress etc. (suffering)	Biological inefficiency and economic loss
Neglect (through ignorance, idleness or overwork)	Malnutrition, poor growth, disease, distress, etc. (suffering)	Biological inefficiency and economic loss
Deprivation (built into some husbandry systems)	Changes in behaviour, occasional abnormal behaviour (suffering?)	? ? ?

Modified from Ewbank (1981a and 1981b)

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Information from implementation of the legislation

In England and Wales abuse and deliberate neglect are prosecutable under the 1911 Protection of Animals Act (under the 1912 Act in Scotland) and, in the particular case of farm animals on agricultural land, under the Agricultural (Miscellaneous Provisions) Act 1968. It would thus seem feasible for epidemiologically useful data to be obtained from court records. In the UK, however, most cases get no further than the magistrates court (or its equivalent) and unfortunately the statistics relating to this level of court activity are incompletely tabulated and poorly correlated.

Since the passing of the Agriculture (Miscellaneous Provisions) Act in 1968, veterinary officers of the Ministry of Agriculture (and its equivalents) have made farm welfare inspections. Some statistical details of these inspections have been published annually in the Reports of the Chief Veterinary Officer (see Table 2).

Table 2. Number of inspections etc. made in Great Britain from 1977 to 1984 under the Agriculture (Misc. Prov.) Act, 1968. (Source: MAFF, Annual Reports of Chief Veterinary Officer 1977-1984)

Year	Number of holdings inspected	Cases of neglect		Approx. number of resulting prosecutions
		Number	% of inspections	
1977	1732	28	1.62	0
1978	1852	29	1.57	1
1979	2736	62	2.27	-
1980	2252	61	2.71	0
1981	3502	60	1.67	4
1982	3987	78	1.96	6
1983	5498	69	1.26	12?
1984	7108	59	0.83	12?

It would seem from these figures that both the number and possibly the severity of the inspections has increased over the years. It is only for the year 1977, however, that any attempt can be made to work out the total number of holdings liable for inspection - and then only for England and Wales. It can be estimated from farm classification figures (MAFF, 1979) that in that year there were probably about 160,000 holdings in these two countries with farm animals on them. Of the total of 1732 inspections in 1977, some 1196 were carried out in England and Wales i.e. it seems that possibly 0.8% of the farms 'at risk' in those countries were visited for welfare inspections. It is probable that this percentage figure has considerably increased since then, as it is certainly known that the number of inspections has gone up, (see Table 2) and it is likely that the general trend over the years for farm units to increase in size has probably resulted in a reduction in the total number of holdings with farm animals.

In the last two recorded years i.e. 1983 and 1984 an attempt has been made to classify (see Table 3) the cases of neglect. If this exercise

is continued it should produce material suitable for epidemiological analysis.

Table 3. Classification of the kinds of neglect detected on MAFF welfare inspections in 1983 and 1984 (Source: MAFF, Annual Reports of Chief Veterinary Officer)

	1983	1984
Failure to note that a minor specific welfare problem had arisen	12	9
Inadequacy of the stock keeper	15	10
Unforeseeable accident with equipment in intensive units	2	1
'Straightforward' neglect	40	37
Incorrectly carried out minor surgical operations	-	2
	69	59

Information from disease surveys etc.

Most diseases and certainly most clinical disease states reduce the well-being of the affected animals and, at times, are the cause of discomfort, pain and distress. In many cases disease surveys are also welfare surveys and, other things being equal, low levels of disease in an animal population can often be equated with that population's high welfare status.

Abuse and neglect of animals usually result in injury and disease - conditions which will be detected on clinical examination and thus are capable of being handled in traditional disease type surveys. The recording of bruising on the slaughter line and the employment of analytical techniques to sort out the probable causes and then the tracing back to the particular transporters and/or to the farm of origin would seem to be a good example where an epidemiological approach to a problem could in the end result in both a reduction in injuries and a reduction in suffering.

In some of the intensive husbandry systems the animals are kept in, what some people consider as barren environments, i.e. the animals do not, probably, have the physical facilities for fulfilling all their behavioural needs. It is believed that this can result in the development of obvious abnormal behaviours. See Table 4 for a clinical classification.

Table 4. Clinical classification of obvious abnormal behaviours

(Source: Ewbank,1985)

Easily seen 'self-evident' abnormal behaviour
- Pathological changes, i.e. signs of overstress and/or distress
- Decrease in biological production, e.g. tail-biting in pigs

Easily seen 'minor' abnormal behaviour
- Little or no coexisting pathological changes, i.e. few signs of overstress and/or distress
- Probably not resulting in loss of biological production, e.g. bar-gnawing in stall-confined sows

Quantitive and/or qualitative changes in otherwise normal behaviour
- No signs of overstress or distress
- Unknown effect on biological production, e.g. increase in normal aggression (apparently needed to maintain social order) seen in some highly stocked pigpens

Some of these abnormal behaviours may be linked with pathological changes, i.e. tail-biting in pigs and thus handleable in the traditional disease type surveys. Others, e.g. bar-gnawing in stalled-sows are not usually associated with obvious injury or disease and are therefore not likely to be included in 'disease' surveys. Yet from the welfare point of view it is in the behavioural area that there is the most need for work. The only recent UK survey which attempts to correlate behaviour and husbandry is the calf study carried out by a joint University of Bristol/Ministry of Agriculture team (Webster et al, 1985).

If the so-called abnormal behaviours are an indication of distress in animals then epidemiological studies which link their occurrence with particular husbandry systems or with particular components of rearing methods, could give most useful clues as to the possible aetiology and significance of the conditions.

CONCLUSIONS

An epidemiological approach to animal welfare is possible and is indeed desirable.

Most of the current survey and epidemiological work done on animal disease has a strong welfare component.

For further progress to be made three obvious difficulties will have to be overcome:-

1) the problem of the definition of welfare
2) the incomplete recording of animal welfare prosecutions in the lower courts
3) the difficulty of identifying and recording abnormal behaviours under field survey conditions.

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THE MINISTRY'S ROLE IN ANIMAL WELFARE

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Historically, this country has had some welfare legislation for well over 150 years. The first in 1822 - The Cruel Treatment of Cattle Act - was followed in 1835 by the Cruelty to Animals Act. The latter Act covered all domestic animals and other animals in captivity. In the intervening period the RSPCA was formed, with the Scottish Society being founded in 1839.

In 1911 (1912 in Scotland) the Protection of Animals Act was passed and this is still in force today. This Act, as amended, uses a form of words which makes it an offence to "cruelly beat, kick, over-ride, over-drive, over-load, torture, infuriate or cause unnecessary suffering to any animal". It is not my intention to recite all the provisions - read the Act for yourselves - but merely to mention the value of certain aspects of the legislation. For instance, under this Act, Courts can ban people from keeping animals only after a second conviction and a police constable can authorise a veterinary surgeon to destroy certain animals where it would be cruel to keep them alive. Most of you will be aware of the Cruelty to Animals Act 1876 - still in force - but covering laboratory animals which are not part of my remit.

For the next fifty years or so the 1911 Act was to serve to protect the welfare of animals. However, in 1964 a book called "Animal Machines" by Ruth Harrison stimulated public concern about the welfare of livestock. This was followed by the so-called Brambell Committee which published a report in December of the same year. As a result new legislation was introduced. The passing of the Agriculture (Miscellaneous Provisions) Act 1968 was a major milestone in the field of animal welfare. It made the Minister of Agriculture responsible for the welfare of livestock on farms. It provided the power to give free advice on animal welfare; to make regulations on a variety of topics related to animal welfare; and to issue Codes containing recommendations covering the welfare of farm livestock.

The Act of 1968 made it an offence for any person to cause unnecessary pain or unnecessary distress to livestock on agricultural land and, in addition, gave Ministry officers the powers of entry to ascertain whether an offence has been committed.

In order to assist the Minister in his new role an independent advisory body, The Farm Animal Welfare Advisory Committee, was set up. This latter body was replaced in 1979 by the Farm Animal Welfare Council (FAWC) which had a slightly different remit, although it advises Ministers on farm animal welfare and puts forward proposals for regulations and advisory Codes, as previously. Any of the proposed regulations and Codes must be the subject of consultation with interested parties and approval from Parliament. The Codes, covering most farm livestock, are not mandatory but can be used in evidence to establish that an offence has been committed. These Codes provide the basis for the advice given by the State Veterinary Service.

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Certain regulations have been formulated under the 1968 Act:

a) Welfare of Livestock (Intensive Units) Regulations 1978

These define intensive farming units and require the regular, and at least daily, inspection of livestock and automatic equipment. Action must be taken to correct any defects and safeguard the livestock from unnecessary pain or distress.

b) Welfare of Livestock (Prohibited Operations) Regulations 1982

These regulations consolidate earlier welfare regulations and prohibit certain additional operations on livestock on agricultural land:

i) Penis amputation and other penile operations;

ii) Freeze dagging of sheep;

iii) Short tail docking of sheep, requiring sufficient tail to cover the vulva of female sheep and the anus in males;

iv) Tongue amputation in calves;

v) Hot branding of cattle;

vi) Tail docking of cattle;

vii) Devoicing of cockerels;

viii) Surgical castration of a male bird;

ix) Any operation designed to impede the flight of bird, other than feather clipping;

x) Any appliance for limiting vision in a bird which involves penetration/mutilation of the nasal septum;

xi) Tail docking of a pig unless performed by quick and complete severance and the pig is less than 8 days old;

xii) Removal of any part of antlers of a deer before the velvet is frayed and the greater part shed.

There are exemptions for certain operations:

a) Cruelty to Animals Act 1876;

b) First aid

c) Veterinary surgeon's treatment for injury or disease.

There is of course, other legislation designed to protect the welfare of livestock on the farm:

- the Veterinary Surgeons Act 1966
- the Protection of Animals (Anaesthetic) Act 1964 and various others.

The Docking & Nicking of Horses Act 1949 has been with us for so long that one would think that everyone would know about it. However, during the last 12 months, docked horses have been discovered and prosecutions taken. It is as well to be aware that it still happens and act accordingly where offences are found.

So far I have spoken only about protection of animals on the farm. There is yet further legislation protecting animals in transit:-

- Transit of Animals (General) Order 1973

- Transit of Animals (Road and Rail) Order 1975

- Conveyance of Live Poultry Order 1919

plus protection for those undergoing sea transport:-

- Transport Animals Order 1927

- Animals (Sea Transport) Order 1930

and the Export of Animals (Protection) Order 1981 which provides general cover for animals passing from our shores.

One final piece of legislation to mention is the Markets (Protection of Animals) Order 1964 as amended.

It is not possible for me to provide an exhaustive list of the measures which protect the welfare of animals or even to provide a coherent summary of those measures I have mentioned. I would suggest that the foregoing provides adequate references to follow-up. Some will say that I have neglected to cover the regulations which protect animals at the time of slaughter. They are extremely important but are the responsibility of my colleagues in the Meat Hygiene Section.

It must be obvious to all from the brief details that I have provided that a considerable body of legislation exists to protect livestock from birth to slaughter. However, for it to be effectively operated those involved must have an intimate knowledge of what it says. We spend considerable time and effort ensuring that the State Veterinary Service staff are conversant with the law and seek to encourage them to support Local Authorities who are responsible for much of the implementation of the law. These Authorities do not have a universal responsibility - for instance they do not have to prosecute under the Protection of Animals Act 1911 (anyone can use this legislation) and we have the responsibility for the Miscellaneous Provisions Act 1968 - but are involved in virtually all else.

Where does the State Veterinary Service fit into all this? We have a structure which starts at Head Office, spreads to Regions and then onto Divisional Offices.

I have in the welfare section at Tolworth 3 Senior Veterinary Officers (SVO) (the equivalent of Divisional Veterinary Officers) and a Marine Superintendent plus his outstationed deputy.

One SVO spends most of the time in servicing the Farm Animal Welfare Council by the provision of specially commissioned papers and literature searches. In addition, attendance at many of their sub-committees to assist in the deliberations is a requirement of the post.

One SVO is responsible for all aspects of on-farm welfare - a considerable job as you might imagine - and the other covers all aspects of transport welfare. The Marine Superintendent and deputy are qualified mariners and provide advice on all aspects of livestock transported by sea. They inspect livestock carrying vessels regularly and make accompanied voyages. Their experience is valuable to our service and is much appreciated by overseas authorities. The SVOs, and VOs in the field, are often sent to accompany livestock by sea, or air to monitor welfare and their reports and experience provide the basis for our advice to the industry.

Whenever any of our staff visit farms, markets or slaughterhouses they are alert to any indications of welfare problems. Visits to farms will follow any suggestion that problems exist and standards will be measured against the welfare codes.

Random visits are made to all types of farming enterprise and each veterinary officer is expected to meet a target of two specific welfare visits each month - although this might depend on staffing levels and workload.

Members of the public will sometimes report suspicions that animals are being neglected or ill-treated and all such complaints are quickly investigated. I think it true to say that rarely is deliberate cruelty encountered. By far the greatest number of cases fall into the category of neglect due to physical or mental incapability and, sometimes, inability to meet debts.

Prosecution is never taken lightly. We would always wish to solve the problems using advice and persuasion. Where specialist nutritional, or indeed, other advice is required, we are empowered to take such advisers with us to visit a farm. Prosecution does not necessarily improve the lot of the neglected animals and fines could make matters worse.

To carry out their duties in a satisfactory manner it is important that staff receive adequate training and we spend much time in trying to give them a strong background knowledge. Part of the 'New Entrants' course introduces them to welfare in all its facets. This is followed up by a specialist welfare course in which matters can be discussed at greater depth. With some experience before officers attend we find that this course engenders a great deal of enthusiasm and staff have a greater degree of confidence in carrying out their welfare duties.

Some staff in each region have the opportunity to attend a specially organised animal behaviour course at Edinburgh, designed to give them a greater ability to interpret what they see on farms. We can't hope to get everyone on such a course but those officers fortunate enough to attend are expected to pass on what they learn to their colleagues. With a greater awareness of welfare it is, perhaps, a topic which ought to be given more time at veterinary schools, although (before anyone hastens to their defence) I am well aware of competing demands at such places.

Where we feel that a need for training in depth is necessary special seminars can be organised. Recently, for instance, we have put considerable thought into raising the level of understanding of transit legislation by the use of 'case studies' in sessions involving ten or twelve staff. Once again, we expect them to pass back the knowledge they gain to their colleagues.

Much can be gained by observation on farms and it is the considered opinion of the welfare section that close observation must precede any experimentation - and I use this word in its widest sense. A study has already been conducted into calf behaviour in collaboration with colleagues in university departments and we are, at present, actively considering extending these to include poultry, pigs and sheep. Obviously these have to be very carefully organised as they could take up a considerable amount of staff time. However, as welfare is such an important and emotive subject we feel that the time is well spent as long as the results can be used to improve the overall standards of animal management and care.

I am well aware of the considerable amount of welfare orientated research which takes place. We try to keep track of it all by frequent discussions with those involved and draw relevant papers to the attention of colleagues in the service. They, in their turn can provide information in the other direction by reporting on problems that require examination and also new developments in animal husbandry at the farm level. Funds available for research are shrinking annually and we must be certain that what is available is used to the best effect.

Not all research takes place in this country. We, therefore, maintain contacts with workers in a great many countries, which seems a sensible way of avoiding duplication of effort. This can involve attending overseas conferences or meetings of EC groups in Brussels or Council of Europe in Strasbourg or correspondence at a personal level. Much valuable information can be gained from informal discussion at any of the above gatherings. The Chief Scientists Group of MAFF is another valuable source of information and we maintain close contact with them.

In between Tolworth and veterinary officers in the field come the veterinary staff in the Regions. They have always been responsible for co-ordinating welfare activities in their respective Regions but have more recently taken on an extra role. Where prosecutions are taken the clearance of statements and liaison with Legal Branch has been devolved to them with the aim of speeding up matters and ensuring that cases come to court in as short a time as possible. Once again, our aim is to improve the standard of welfare of farm livestock. Where successful prosecutions are taken, publicity can add to the benefit by providing a suitable warning to others whose standards of stockmanship may not be all that it ought.

I have spoken mostly about field veterinary staff but this does not mean that those in the Veterinary Investigation Service abdicate responsibility for animal welfare. In fact, many cases are brought to light during their investigations and are, in the main, solved by close collaboration with all concerned. I venture to suggest that the practising veterinary surgeon has also a major role to play in welfare - to retain credibility, the profession must seek to maintain the standards of care and husbandry of the animals under their care. I know that they can find themselves in difficult circumstances but they must not shrink from reporting clients whose welfare standards are low and are not amenable to advice.

So far I have not spoken about the State Veterinary Service and its relationship with the Farm Animal Welfare Council - that independent body I spoke of earlier. Its secretary and his staff are members of MAFF who work full time on servicing the Council. In addition there is an administrative assessor who provides guidance on political matters and I, as technical assessor, am responsible, with colleagues in my Section, for seeking out and supplying the scientific evidence the Council may require in its studies.

You may be aware that the Council has issued reports on mutilations - on which the regulations discussed earlier were based - and on the welfare of poultry and red meat animals at the time of slaughter. It recently concluded its study of slaughter welfare with a report on slaughter by religious methods. Each of these reports includes a great many recommendations for Ministers to consider. A report on the welfare of farmed deer has not long been published and we are shortly to see a report on markets.

The Council has just started a study on the transport of farm animals and horses and is in the process of updating the Welfare Codes for Sheep and writing Welfare Codes for Goats. It is also active in many other ways but those I have mentioned will suffice to show as examples of its wide-ranging interests.

Before I finish perhaps I can venture a small excursion into the future. Crystal ball gazing is never an exact science and at the present time the view can best be described as cloudy. I foresee welfare remaining, or increasing, as a topic of importance for the veterinary profession. The general public's concern will not diminish and the committed welfarists will ensure that it remains in the forefront of people's minds. Alternatives to the intensive systems of management will continue to be sought but it would be naive to believe that they can be isolated from the economics of farming. There will always be those who are prepared to pay a little more for their produce, but many more, by force of circumstance, will choose the cheapest.

One way forward could be within the context of the EEC. I accept that any attempt to obtain agreement between 12 nations, as it now is, can be fraught with difficulty. However, we must persevere if the standard of livestock care is to be improved. There must be a united step forward as there is little point in improving our standards if it makes livestock products expensive to produce and opens the door to cheaper products from other countries with poorer welfare standards.

Work will, I am sure, continue in the Council of Europe. 'The European Convention for the Protection of Animals Kept for Farming Purposes' was but the first step. At present, standards for pig husbandry and horse transport are being worked on but I am sure that other topics will follow.

Finally, we, in this country will continue to monitor and observe the standards of husbandry and animal behaviour. We must use our findings to improve the lot of animals on our farm. While I accept that the standards in general are high we must continue to strive to improve those of questionable status. More and more people will question intensive methods and we must have viable alternatives to offer the farming community. As I see it we still have much work ahead in the field of animal welfare.

I hope it is evident from what I have said that the State Veterinary Service has a considerable commitment to animal welfare. I have sketched out its involvement but am only too aware that I may have left many questions in your mind. Within reason, I am prepared to answer them in the time remaining but am always to be reached by letter or telephone if I can help.

SOME ECONOMIC CONSEQUENCES OF INTRODUCING MINIMUM SPACE REQUIREMENTS FOR LAYING HENS

FRANCES SANDIFORD*

The introduction of statutory restrictions on particular animal production systems may have wide-ranging economic impacts, potentially arising from the effects of the legislation on

(i) the direct costs of production;
(ii) the consumer price level and consumption of commodities affected;
(iii) the structure of the farming industry, where 'structure' includes the combination and use of inputs on the individual farm (the 'internal structure'), and the number and size of holdings in the industry (the 'external structure');
(iv) the adjustment costs incurred in complying with the legislation;
(v) trade in the commodities affected;
(vi) the supply of output.

The focus of this paper is the economic consequences of one set of statutory restrictions: the introduction of minimum space requirements (MSRs) for laying hens to be effective from either 1/1/1990 or 1/1/1995.

DIRECT COSTS OF EGG PRODUCTION

The direct costs of producing eggs depend on the quantities of the different inputs used in the production process and the prices that the producer pays for these inputs. Data on individual flocks in the Poultry Laying Flock Recording Scheme (PLFRS) are analysed using multiple regression techniques, firstly to estimate the relationship between bird space and feed consumption. This is because increasing bird space is thought to increase feed consumption per bird through the lowering of house temperatures; more feed is required by the bird to maintain body temperature. These results are presented in Table 1. Secondly, the average relationship between costs per dozen eggs and input quantities and prices is estimated; the effects of MSRs on feed consumption are then used in this relationship so that we can quantify the expected changes in the direct costs of egg production following the introduction of MSRs. Within the limitations of our data and methodology, lower and upper limits on the magnitudes of the likely cost changes for Great Britain are shown in Table 2.

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Table 1. The effects of increased bird space on feed consumption

Stocking density sq cm	% increase	Feed consumption g/bird/day	% increase
473		118.5	
500	5.6	120	1.3
550	16.3	123	3.8
600	26.8	126	6.3
650	37.4	129	8.8
750	58.6	135	13.8

Table 2. The effects of MSRs on egg production costs

Stocking density sq cm	Production cost p/doz		Percentage cost increase	
	Lower	Upper	Lower	Upper
Baseline	40.0			
500 sq cm	40.1	40.2	0.3	0.6
550 sq cm	40.3	40.7	0.8	1.6
600 sq cm	40.5	41.1	1.3	2.6
650 sq cm	40.8	41.5	1.8	3.6
750 sq cm	41.2	42.2	2.9	5.5

Great care is needed in the interpretation of these results: they characterise the average relationships that are found across the farms in the sample, and, it is to be hoped, by extension, the industry. They do not provide any information about what would happen to an individual farmer if he were to change his inputs. By characterising average cross-farm relationships, the results provide descriptive information about the average unit in the industry, not prescriptive information for the individual. Broadly speaking, the percentage cost changes are what we should expect to see on average for the industry as a whole, after any adjustment period, all other things remaining the same. It can be seen that an MSR of 500 sq cm per bird would be expected to increase production costs by between 0.3% and 0.6%. The reason why this is so low is that the mean stocking density now is 473 sq cm per bird and so the change required is very small; this should be contrasted with the modal or 'typical' stocking density in Great Britain, which is 450 sq cm. Changing to 550 sq cm would probably increase costs by 0.8%-1.6%, with 600 sq cm leading to increases of between 1.3% and 2.6%, and 650 sq cm leading to increases ranging from 1.8% to 3.6%. A very strong note of caution must be sounded with regard to the results for an MSR of 750 sq cm. In this case, and only in this case, we are extrapolating our results beyond the range of our observations. The highest bird space in our sample is 668 sq cm. The validity of extrapolating to account for the effect of an MSR of 750 sq cm depends crucially on whether the underlying feed consumption - bird space relationship that we have estimated holds true for those levels, and whether the estimated cost relationship does too; also, the capital cost figure is assumed to be applicable. If these relationships do hold at higher bird spaces, then our

estimated range of cost increases of 2.9%-5.5% is acceptable. Even if capital costs were 20% higher, the range of estimated cost increases would only be 4.1%-6.7%.

We should not be surprised by the magnitude of the cost changes discussed here. The fact that about 40% of producers in our sample are using stocking densities between 475 and 668 sq cm per bird suggests that the cost disadvantages of relatively high bird spaces are not sufficient to prevent these producers from competing successfully in what is an extremely competitive market.

RETAIL EGG PRICES AND THE CONSUMPTION OF EGGS

How changes in the direct costs of egg production consequent upon the introduction of MSRs would affect retail egg prices depends on the structure of marketing margins - the relationship between prices at different market levels - in the shell egg market. The results of our analysis suggest that marketing margins for shell eggs consist of an absolute mark-up with no percentage element, i.e. retail prices appear to be a straight mark-up over farm prices, in the longer term, with no element that is proportional to farm price. This is true for the overall farm - retail margin and for the producer - packer and packer - retail margins in the long run. There is no evidence to support the view that increases in costs of production, assuming that they are exactly reflected in the farm-gate price in the long run, will result in consumer price increases of more than the cost of production increases. This means that any given percentage increase in prices at the farm level will ultimately lead to smaller percentage increases in retail prices. The effects of the production cost increases shown in Table 2 on retail egg prices are presented in Table 3. It can be seen that the ranges of increases in consumer prices for MSRs of 500, 550, 600, 650 and 750 sq cm are: 0.1% to 0.3%, 0.4% to 1.0%, 0.7% to 1.6%, 1.1% to 2.1%, and 1.7% to 3.1% respectively. The caveats about the results for an MSR of 750 sq cm discussed above are equally applicable here.

Table 3. The effects of MSRs on the retail price of eggs

Stocking density	Percentage retail price increase	
	Lower	Upper
500 sq cm	0.1	0.3
550 sq cm	0.4	1.0
600 sq cm	0.7	1.6
650 sq cm	1.1	2.1
750 sq cm	1.7	3.1

What of the effect of these retail price changes on the quantity of eggs demanded? This depends on consumers' price elasticity of demand for eggs: in recent years, this has not been found to be significantly different from zero. The question is whether the retail price changes we have estimated here are sufficiently marginal for the estimate of zero price elasticity of demand to be applicable. The degree of price variation underlying the zero price elasticity of demand estimates is substantial, even allowing for seasonal

effects. This suggests that retail price increases of 3.1% - which is the highest result of our estimated increases in production costs - would be unlikely, of themselves, to affect egg consumption. It is often said that very few consumers would be willing to pay more for 'welfare' eggs. This should not be confused with the point at issue here. It may well be true that very few consumers would be prepared to pay more for eggs if they were offered a choice between higher-priced 'welfare' eggs and lower-priced ordinary eggs. This is quite consistent with a zero price elasticity of demand for eggs since this relates to a homogeneous product. The option of paying more for a perceived quality difference is not there. Our conclusions are that for the range of likely price increases for eggs, egg consumption will be unaffected.

THE CHANGING STRUCTURE OF THE UK EGG INDUSTRY

The UK egg industry to which these welfare regulations would apply has been undergoing substantial structural change over the last twenty years. There have been pressures on the industry to concentrate and contract, the latter including the declining demand for eggs since 1970 and two particular changes in the internal structure of the industry. Firstly, the average egg yield per laying fowl has been increasing steadily since the early 1960s as a result of technical and genetic improvements; regression-based estimates of these yield increases using data from the MAFF Egg Yield Survey are that the greatest increase has been for layers in cages - 2.2 eggs per bird per annum (p.b.p.a.) - followed by those on deep-litter - 2.0 eggs p.b.p.a. - and on range - 1.8 eggs p.b.p.a. Secondly, there was a rapid movement during the 1960s away from free range and deep-litter egg production into battery cage production which has been associated with a higher average egg yield per bird than the other production systems. This movement continued through the 1970s, albeit at a reduced rate. According to the MAFF Egg Yield Survey, over 96% of the national laying flock is now housed in battery cages, with just under 2% on each of deep-litter (and miscellaneous) and free range; this compares with 19.3% in battery cages, 49.8% on deep-litter and 30.9% on range in 1960/61.

These pressures for contraction have led to a national laying flock that is perhaps 20% smaller now than it was in 1967. The total number of holdings has also fallen throughout this period, but whereas the decline has been continuous and rapid amongst those with fewer than 5,000 birds, the number of larger holdings has only been falling since 1973. The indications are that, even in the absence of welfare legislation, the underlying demand and supply conditions of the egg market will lead to further changes.

THE FUTURE INDUSTRY STRUCTURE

The type of structure that the industry will need to have depends on the underlying demand and supply conditions, the most important components of which are likely to be (i) the projected continuation of the decline in the demand for eggs, (ii) technological change in the form of increased egg yields per bird, and (iii) legislation governing the welfare of laying hens.

The demand for eggs

Household demand is the main component of total shell egg demand; the determinants of this demand are thought to be household structure and composition, and tastes. Economic variables, such as the price of eggs and

income, appear to have no significant effect on demand. Three forecast options for household egg demand are used to reflect the likely future changes in tastes; each is combined with the forecast effects of household structure and composition on egg demand to yield forecasts of total household shell egg consumption. The total demand for shell eggs is a weighted sum of three components: household, catering and institutional demand. The three forecast options, which are presented in Table 4, are calculated as index numbers, at constant prices and with 1983 as the base year.

Table 4. Forecasts of total shell egg demand for the UK

Forecast basis	Total shell egg demand Index No. (1983 = 100)	
	1990	1995
Option 1 - High	99.7	99.7
Option 2 - Medium	94.2	92.6
Option 3 - Low	86.2	76.8

Output per bird

It is thought that egg yields will continue to increase, although at a slower rate than was the case in the 1960s and 1970s. (Some levelling off of yields has been observed over the last four years.) A base-line of 259.5 eggs p.b.p.a. in 1984 is used for flocks with 500+ birds housed in battery cages; for non-cage birds, the 1984 base-year yield is taken to be 219.0 eggs p.b.p.a. An average annual yield increase of 1 egg p.b.p.a. is assumed for the forecast period. For the base year, it is assumed that the higher yield figure of 259.5 eggs p.b.p.a. applies to the 97% of the UK flock on holdings with 500+ birds, and the lower figure of 219.0 eggs p.b.p.a. applies to the other 3% of the national flock on holdings with <500 birds. For the forecast years, the lower yield forecasts are applied to 5% of birds, and the higher yield forecasts to 95% of birds, giving projected average egg yields for 1990 and 1995 of 263.5 and 268.5 eggs per bird.

The future industry structure in the absence of MSRs

The combined effect of yield increases and demand decreases on commercial laying flock requirements are shown in Table 5.

Table 5. Forecasts of required UK commercial laying flock, 1990 and 1995

Forecast basis	Baseline (83/84)	1990	1995
Average Egg Yield (Eggs p.b.p.a.)	257.9	263.5	268.5
Commercial Flock Size (Millions of Birds):			
Option 1 - High Demand	43.46	42.46	41.67
Option 2 - Medium Demand	43.46	40.21	38.82
Option 3 - Low Demand	43.46	36.93	32.46

It can be seen that all of these forecasts involve a reduction in the size of the UK laying flock. The issue here is how this reduction is likely to be effected. If the trends since 1967 were to continue, there would be a relatively rapid decline in the number of smaller holdings, where 'smaller' here refers not only to those with <5,000 birds, but also to those with 5,000-9,999 birds. The number of holdings in the 10,000-19,999 bird range would also be expected to decline, although at a slower rate. Those least affected by the pressures to reduce the size of the national laying flock would be in the 20,000+ bird group, which might show a slight fall in numbers that could be largely off-set by small increases in the average flock size. The proportion of the national flock on these larger holdings would therefore increase over time from its 1983 level of 63%. In short, continuing pressure to reduce bird numbers seems bound to increase concentration in the number of operating units.

In terms of the internal structure of the industry, it is quite possible that we shall see relatively little change in the absence of welfare legislation compared to the major changes that occurred during the 1960s. The proportion of birds housed in battery cages is already so high that little further change can be expected in that direction. On the contrary, perhaps, the pressures for continuing industry contraction from falling real egg prices might lead some producers to switch to more extensive systems of production so that they can take advantage of the premium prices paid for free-range eggs. How large this market might be is very difficult to assess. Given a reduction in premium as more suppliers enter the market, and the limited number of consumers who are prepared to pay a premium, it seems intuitively unlikely that the non-cage sector would, in the absence of welfare legislation, account for as much as 10% of the retail market.

The future industry structure in the presence of laying hen welfare legislation

A problem with trying to assess the effects of laying hen welfare legislation on industry structure is our present uncertainty about the performance of alternative technologies. The predominance of battery cages in the UK means that there are few large-scale commercial examples of other production systems. Percheries, aviaries, deep-litter, straw-yards and free-range are all possible options.

For the legislation options that involve comparatively small increases in cage space per laying bird - say, up to an MSR of 550 sq cm per bird - the predominant production system may well remain the battery cage. On our evidence about capital costs, these options would not necessarily require a higher capital input per bird. They might require slightly higher labour inputs, but the most likely change in internal industry structure is an increases feed usage as a result of lower house stocking densities. The stocking density of the house is a critical factor in determining temperature and therefore feed intake. However, different cage formations could perhaps be employed to keep house stocking densities high enough to avoid a fall in temperature and a consequent rise in feed consumption. There is no obvious reason why these legislative options should lead to changes in the external structure of the industry.

The question becomes more problematic when a change to different production systems is being considered - either because battery cages are banned or because the introduction of higher MSRs (say, 600 or 750 sq cm per bird) makes other systems economically competitive. For deep-litter,

straw-yards and free-range, all input usage with the exception of energy would be expected to increase for a given level of output: labour, feed, numbers of birds, land and capital - although the capital mix would differ according to the system. For multi-tier houses and aviaries, experimental results suggest that feed, labour and capital inputs would be higher than for battery cages, but not as high as deep-litter and straw-yards. Unless the alternative production system(s) that replaced battery cages exhibited diseconomies of size, the external structure of the industry need not change as a result of laying hen welfare legislation.

ADJUSTMENT COSTS

Costs of adjustment can arise in two ways. Firstly, there may be accelerated depreciation of existing capital. This refers to that part of the capital stock that becomes redundant before it would be expected to in the normal course of events because the welfare legislation renders it obsolete. In the case of legislation that can be complied with using the same basic technology - such as the introduction of MSRs for laying hens in battery cages - the capital stock need not be rendered technologically obsolete; keeping it in use to avoid accelerated depreciation might, however, involve the incurrence of higher production costs. To see this, consider the effect of target MSRs of 500 and 600 sq cm on a producer who is currently using 2250 sq cm cages with 5 birds to the cage, i.e. 450 sq cm per bird. To comply with the legislation, he could either buy new cages that would enable him to stock at exactly 500 or 600 sq cm per bird (and in the long run, this is perhaps the most likely response), or he could continue to use his existing cages while stocking them with fewer birds; 4 or 3 birds to the cage would achieve the target MSRs of 500 or 600 sq cm per bird. In fact, the targets would be substantially over-achieved since the de facto stocking densities would be 563 or 750 sq cm per bird. Therefore, if this producer chose not to incur accelerated depreciation costs, he would have to stock at a density of either 563 or 750 sq cm per bird. In so doing, he would incur direct production costs higher than those associated with a stocking density of 500 or 600 sq cm per bird - which is all that would be required by law. The increase in production costs over that which is strictly necessary to meet the targets, and which is owing to the economic, rather than the technological, obsolescence of the capital stock, is the other way in which adjustment costs might arise.

Who would be affected by the introduction of MSRs in GB?

The model stocking density class for laying hens in GB is 450-474 sq cm, and the mean stocking density is 473 sq cm. If MSRs are introduced, then those producers who are affected by them have the option of complying by reducing the numbers of birds per cage, or by investing in new cages. For each policy option, the numbers affected are assessed, and the consequences of compliance using existing cages are shown in Table 6.

Adjustment costs (i): changes in production costs in GB

It is clear from Table 6 that the changes in the internal structure of production are substantial when the existing cages are used to comply with any of the proposed new welfare standards. There is also such a diversity of cage sizes and stocking densities that no target MSR could avoid this. For all the options, continued use of existing cages means that about two-thirds of flocks would have to be housed at stocking densities of more than 50 sq cm above the

MSR. It is possible to obtain rough estimates of the extra production costs that could be incurred because of the continued use of existing laying equipment, over and above those which would necessarily be incurred to meet the target MSRs. This cost difference is properly an adjustment cost.

Table 6. The consequences for stocking densities of compliance with welfare standards using existing cages in GB

Policy option	Baseline	500 sq cm	550 sq cm	600 sq cm	750 sq cm
Flocks affected		71.5%	90.5%	95.2%	99.5%
Stocking density:					
modal class sq cm/bird	450-474	550-574	550-574	600-624	750-774
mean " "	473	568	632	696	879
maximum " "	750	750	824	882	1394
Percentage in modal class	39.7%	23.3%	23.3%	18.5%	18.5%

Capital costs: The mean number of birds per cage in the sample, i.e. the baseline mean, is 4.79. MSRs of 500, 550, 600 and 750 sq cm per bird imply reductions in the mean numbers of birds per cage of 16%, 24%, 31% and 45%, respectively; i.e. the capital costs during the adjustment period have to be spread over fewer birds.

Non-capital costs: Because the mean bird spaces would be higher than those required by the welfare standards, feed consumption per bird would be higher than is strictly necessary. Other costs might also be expected to change, but it is not possible to say by how much.

The capital and non-capital cost changes can be combined and compared with those which we should expect to see in the long run after all adjustment has been made to the new welfare standards. This gives us an approximation of the difference in production costs that could arise from the use of economically obsolete capital (see Table 7).

Table 7. Adjustment costs (i) - An assessment of the extra costs of egg production incurred through compliance with MSRs using existing cages in GB

Policy option	Baseline	500 sq cm	550 sq cm	600 sq cm	750 sq cm
Long-run direct costs pence/doz eggs	40.0	40.2	40.7	41.1	42.2
'Adjustment' production costs, pence/doz eggs		41.3	41.9	42.6	44.4
Difference between long-run and 'adjustment' production costs, pence/doz eggs		1.1	1.2	1.4	2.2
Percentage increase in production costs attributable to adjustment costs		2.8%	3.0%	3.5%	5.5%

Adjustment Costs (ii): Accelerated capital depreciation in GB

The two major capital components in battery-cage egg production are the house structure and the laying equipment. The introduction of MSRs for laying hens kept in battery cages would not entail accelerated depreciation of the house because it could be re-equipped with new battery cages. Insofar as cages are scrapped before they otherwise would be because of the MSR legislation, their reduced life constitutes a cost of adjustment. Our estimate is that 15% of the existing capital stock of laying equipment would be lost through accelerated depreciation if MSRs were introduced from January 1 1990. The assumptions that have been used to produce this estimate can be found in Sandiford (1985). It is important to note that this loss applies to the investment that has already been made, not to the investment in new cages to meet the welfare standards.

At 1983 values, the average cage replacement price is estimated to be £2.78 per bird. It is estimated that the size of the GB flock in battery cages is 38.3 m birds, giving a total value of laying equipment for these birds of £106.5 m. The maximum loss to the GB industry through accelerated depreciation is therefore 15% of £106.5 m, i.e. £16.0 m. In the absence of welfare legislation, the GB flock kept in battery cages in 1990 is expected to fall by 5.2%, 10.2% or 17.8%, depending on the demand forecast used. This means that, in the normal course of events, we should expect a percentage of the capital stock to be made redundant, the exact percentage depending on the level of demand and the technical improvements in bird performance. Given this, it could be argued that the effective accelerated depreciation of laying equipment as a result of the introduction of MSRs should take account of the expected 'normal' depreciation for this declining industry. Therefore, our estimated maximum figure of £16.0 m can be compared with the likely 'normal' capital redundancy, which would include the redundancy of buildings as well as laying equipment, i.e. £11.5 m, £22.6 m, or £39.4 m. These results can be seen in Table 8.

Table 8. Adjustment costs (ii) - estimates of accelerated capital depreciation following the introduction of MSRs for laying hens in GB from 1/1/1990

	Baseline	Demand Option 1 High	Demand Option 2 Medium	Demand Option 3 Low
Millions of birds in battery cages in GB	38.3	36.3	34.3	31.5
Value of laying equipment, £ m	106.5	100.9	95.6	87.6
Value of buildings, £ m	114.9	108.9	102.9	94.5
Expected normal capital redundancy, £ m		11.5	22.6	39.4
Accelerated capital depreciation of laying equipment, £ m		16.0	16.0	16.0

A comparison of the two types of adjustment cost

The two types of adjustment cost are mutually exclusive: no producer can simultaneously incur both for the same flock. It is interesting, therefore, to try to compare the two in aggregate. Those for accelerated capital depreciation have already been estimated and it should be noted that they are invariant with respect to the level of the target MSR, as long as the producer does not switch to another production system in consequence of the cage welfare standards. The detailed assumptions underlying the aggregate estimates of extra production costs can be found in Sandiford (op.cit.), but of immediate relevance is the assumption that cages have a ten year life. With a five year derogation period, this implies that some existing cages would be used for egg production until the end of 1994. The quantity of eggs being produced with such cages would fall both as a result of the adjustment process and because of the reduced laying fowl requirements. Consequently, the extra production costs would fall in each of the five years; these costs are discounted at 10% for each of the years 1991 to 1994 to provide a total figure at 1990 prices, on the assumption that yields remain at current levels. The results are given in Table 9. However, the fact that total adjustment costs could apparently be minimised by scrapping all the existing cages by 1990 does not mean that it would be economically optimal for each individual producer to do so.

Table 9. A guide to the total adjustment costs incurred through compliance with welfare standards using existing cages in GB, 1990-94

		Demand Option 1 High	Demand Option 2 Medium	Demand Option 3 Low
Eggs produced at 'adjustment' cost levels, m doz:	1990	398	378	347
	1991	319	301	272
	1992	239	225	200
	1993	159	150	130
	1994	80	74	64
Total extra production costs attributable to adjustment costs, £ m at 1990 prices:				
	MSR=500	11.6	11.0	9.9
	MSR=550	12.7	12.0	10.9
	MSR=600	14.8	14.1	12.7
	MSR=750	24.3	23.0	20.6

TRADE AND THE SUPPLY OF EGGS

An increase in the direct costs of producing eggs in the UK as a result of the introduction of MSRs might adversely affect the international competitiveness of the UK egg industry. The question then is what might happen to trading patterns and the supply of eggs?

Let us consider first the imposition of such legislation at the EC level. The Community as a whole is a net exporter of shell eggs, and is estimated to

be about 102% self-sufficient in egg production. The majority of EEC trade in shell eggs takes place within the Community. However, there is an EEC export trade to third countries of between 5,000 m and 6,000 m eggs per year, mostly to the Middle East and North Africa, but the support mechanism for internal EEC market prices means that trade with third countries takes place at world prices. There should therefore be no reduction in the supplies of imports from, or the demand for exports by, third countries because of unfavourable price movements. With the majority of shell egg trade being intra-EEC, and with a Community capacity for egg production that exceeds Community demand, it is hard to visualise EEC supplies of eggs being affected by the introduction - after a 5-10 year derogation period - of MSRs for laying hens. Additionally, the possibility of larger export markets in countries such as Switzerland, which is introducing more stringent welfare legislation of its own, should not be discounted. Third country supplies conforming to the EEC welfare standards would probably not be forthcoming in the light of the large and extremely competitive EEC egg industry, but internal supply conditions are such that this would not be a problem.

In the absence of Community agreement on laying hen welfare legislation, the question of the feasibility of unilateral action by the UK remains. UK exports of shell eggs have generally exceeded imports since 1976 - the figures for 1980-4 are shown in Table 10 - and self-sufficiency in egg supplies including egg products (with the exception of albumen) has been 99%-100% or more for some years according to the Annual Review of Agriculture. The ability of the UK to produce its own egg requirements without disrupting supplies to the market is not in doubt. However, since prices in the UK would probably rise as a result of the welfare legislation, there is the problem of increased imports from other EEC countries if the UK were unable to exclude them on welfare grounds. There has been substantial expansion of egg production capacity in the Netherlands, increasing the Dutch share of EEC output from 14.7% in 1982 to 16.1% in 1984, and it was only in late 1984 that a ban was proposed on any further expansion. The UK became France's biggest egg export market in 1984, at the same time as third country markets were declining. The UK is therefore seen as an attractive market now by both France and the Netherlands; allegations have long abounded about eggs from these countries being dumped on the British market and it is certain that higher UK prices would lead to increased imports from the rest of the Community. Just how great this increase might be is hard to say, but UK price increases of, say, 2.5% following the introduction of welfare legislation might result in increases in imports of 5%.

Table 10. UK trade in shell eggs, 1980 - 1984

Thousands of boxes		1980	1981	1982	1983	1984
Exports:	EEC	1157.0	1022.0[a]	867.5	894.0	634.6
	Third countries	55.3	41.0[a]	57.2	57.6	29.9
	Total	1212.3	1063.0[a]	924.7	951.6	664.5
Imports:	EEC	1077.0	926.4	259.3	770.0	1195.2
	Third Countries	3.2	12.2	0.0	0.8	0.0
	Total	1080.2	938.6	259.3	770.8	1195.2
Net Imports		-132.1	-124.4[a]	-665.4	-180.8	+530.7

[a]Includes unpublished estimates by H.M.Customs for missing months in 1981
Source: H.M.Customs

The effect of this on UK prices depends on the elasticity of total supply and the rates at which the demand and supply curves are shifting owing to falling demand and increasing output per bird, all of which are difficult to estimate with sufficient accuracy. Nevertheless, we can say that the downward pressure on prices caused by these demand and supply shifts would be exacerbated by increased imports. It would be extremely hard to justify such legislation to egg producers if they were not protected from the price effects of imports of eggs produced under conditions deemed to be unacceptable for hens housed in the UK. If restrictions on trade could not be enforced, it is hard to see how unilateral UK action could be made workable, particularly if, in the long run, domestic production were seriously undermined by lower-priced imports. Furthermore, in addition to the legal and economic problems of unilateral action on laying hen welfare by the UK, there is the moral dimension. If there could not be adequate policing of egg imports to ensure their compliance with UK welfare standards, whether it be for legal or practical reasons, or if a ban on imports were proved to be illegal, then unilateral British action on laying hen welfare would be open to the charge of having failed to achieve its moral objectives - and of having failed at the expense of UK egg producers.

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CONSUMER REACTION TO COST AND HEALTH CONSEQUENCES OF FREE RANGE SYSTEMS FOR VEAL AND POULTRY PRODUCTION

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Recognition of the behavioural and ethological needs of farm animals as an important element of their welfare has increased in recent years and is now a significant factor in the evaluation and practice of new animal husbandry methods and the marketing of animal products. The welfare of farm animals in the U.K. is governed by the Agriculture (Miscellaneous Provisions) Act 1968 which states, inter alia, that it shall be an offence to cause "unnecessary pain or unnecessary distress". Brambell (1965) evolved a positive philosophy of animal welfare for intensively kept stock. This proposed that animal husbandry procedures should comply with the basic behavioural needs of the animal and "disapproved of a degree of confinement of an animal which necessarily frustrates most of the major activities which make up its natural behaviour".

The European Convention for the Protection of Animals kept for Farming Purposes (1976) established that the principles of good husbandry should meet both the physiological and ethological needs of the animal. Ewbank (1980) broadened the definition of ill-treatment of animals to include behavioural deprivation. Paxman (1981) recommended in evidence to the House of Commons Select Committee investigation of Animal Welfare in Pig, Poultry and Veal Calf Production that the Act be broadened to recognise causing severe behavioural deprivation as an offence.

Against this background husbandry systems which allow animals to behave naturally to a very substantial extent are deemed preferable to methods which restrict behaviour. The former are generally recognised as "Alternative Methods" to the prevailing "Intensive Methods". Veal and table poultry are, for the most part, produced by intensive methods, but alternative methods for each are now well developed. Assessment of these methods raises scientific and commercial questions. What is the quantitative evidence of reduced pain, distress and behavioural deprivation, taking full account of pain and distress arising from ill-health? In the market place, what is the public perception of the end-products and what factors of commercial interest, politics, consumer preference and value influence the future balance of production between the two methods ?

Veal calves are reared under an intensive system in individual wooden crates, in controlled environment buildings on an exclusive diet of reconstituted milk. They are deprived of degrees of movement and grooming, rumination, exploration, play and other group inter-actions, and retreat.

The production of loose-housed veal calves has been described (Webster, [1981], [1985], Paxman [1981], [1985] and Volac [1982]). Loose-housed calves on the Quantock system are housed in groups of 40 in natural environment barns on straw bedding, some of which they eat. Rumination develops. They also feed at will on milk substitute from an automatic feeder. They play and explore and group behavioural patterns develop.

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The costs of the two methods of production are substantially similar although differing in detail. The capital and labour costs of the crated system are higher but the feed costs are lower because a degree of rationing results in a more efficient conversion of feed to carcass. The loose-housed calves are cheaper to care for, since less time is spent on feeding and cleaning, and to house, since environmental control is largely dispensed with, but feed costs are higher.

The available evidence indicates little difference in morbidity and mortality between the systems.

Fig. 1. Calf Mortality Rates in Loose-housed and Crated Veal, Beef Calves and National Herd

(Source: H.M.S.O. 1981, 38 - VIII, P.332)

Farm	Method	Period	Total Calves purchased	Total	Per cent-age
Quantock Veal	Crates	Feb.1979 to Aug. 1980	4,000	259	6.46
Quantock Veal	Loose-housed	Sept.1980 to Feb.1981	2,090	105	5.10
Wysing Grange	"	March 1978 March 1981	4,500	169	3,75
Irish Veal Farms	"	1980	3,351	97	2.89
West of Scotland College of Agriculture "Mortality in Groups of Purchased Friesian-Cross Calves" by Williams et al British Veterinary Journal 1980	Indiv-ual pens	1978-79	507	52	10.5
Kilkenny & Rutter "Perinatal Ill-health in Calves" 1975	"	1975	-	-	5.8
"Calf Wastage and Husbandry in Britain" (HMSO 1968)	All systems	1963	National herd	-	5.3

The majority of table poultry is produced in deep litter broiler houses. The method is well known and involves rearing birds from day old to approximately 7 weeks in controlled environment deep litter houses on a diet of high nutritional value containing significant amounts of animal proteins and growth promoters. The birds are very closely confined but can exercise fully. Subdued lighting depresses activity. Under normal lighting conditions, aggressive behaviour is likely to develop resulting in feather pecking and other vices.

In France a method of free-range table poultry production has developed which is specifically defined and regulated by Government Decree of 17th June 1983 and officially classified as the "Label Rouge" method (Ministere de l'Agriculture, Paris 1983). The Decree requires that poultry to be marketed under the Label Rouge brand shall conform to the following minimum standards, (SY.NA.LAF. 1983) :-

1. small rearing groups, maximum 500 birds.

2. diet containing not less than 70% cereals and no growth promoters or medication other than coccidiostat.

3. unlimited freedom to range.

4. minimum slaughter age of 82 days.

5. shall be slaughtered in an approved slaughterhouse and sold fresh within 6 days.

The majority of Label Rouge free-range poultry is produced in the pine forests that extend from Bordeaux to the Pyrenees, an area of over 1 million hectares (Les Fermiers Landais 1985). The sandy soil and mild climate allow simple home-made, uninsulated and mobile coops to be used. These are moved to new ground after each crop and each rearing site is used only once a year.

The diets used have an M.E. level of approximately 85% of conventional broiler diets and lower protein values. The feed is offered as a meal which reduces intake by approximately 20% as compared with crumbs and pellets.

The breed used is a slow-maturing traditional breed which has undergone some selection. The birds develop extensive ranging behaviour, and travel up to 200 metres from the coop. Birds from each coop develop flock behaviour patterns, returning each night to their own coop. They forage actively, dust bath, run and flap. Cockerels show aggressive behaviour but fighting and feather pecking do not occur, probably becayuse there are no spatial restraints.

The carcass produced has a dressed weight of 1.25 to 2 kgs, similar to the range in broiler birds half their age. Carcass conformation is characteristic for the breed, with long legs and relatively narrow breasts.

The Label Rouge production costs are approximately 20% higher than broiler costs, due to slower growth rates, less efficient food conversion, and higher labour input.

The mortality rate of the French birds is 2% on average although predation, mainly by buzzards, may increase this. Health is otherwise good and the practice is not to medicate. There are no significant differences in morbidity and mortality between Label Rouge and broiler poultry.

A modification of the Label Rouge system, to meet British environmental and market conditions is being evaluated. (Sainsbury, 1986).

To summarise, the alternative systems of veal and poultry production described have no apparent welfare disadvantages so far as morbidity and mortality are concerned. They have quantifiable advantages on behavioural and ethological grounds; there are measurably greater degrees of behavioural freedom and activity. The production costs of loose-housed veal is similar to crated veal, but of Label Rouge poultry, approximately 20% higher than broilers.

The factors that influence consumer buying trends and preferences are exceptionally complex and numerous. The underlying consumer desire is to purchase foods that represen perceived value, flavour, quality convenience, and purity, dietary benefits, and an acceptable provenance.

Imposed influences affect these intentions. Commercial interests are the most significant. Advertising has potent effects. Retailers tend to suppress product information. Labelling veal as free-range draws attention to the fact that most of the faster selling poultry and pork on display is intensively reared. Ingredient labelling highlights ingredients that consumers are learning to associate with ill-health. Conversely, pressure groups seek to influence buying trends through the media and political change, and animal rights groups have been a powerful force for changes in the law and heightened political sensitivity by all parties to animal welfare issues. Significant cultural trends include increased awareness of the interaction between diet and health, the practices of the food industry and recognition of animal rights.

Against this background, the marketabiity of free-range veal and poultry show remarkable differences. In France, Label Rouge poultry have progressively increased their share in a very substantial market sector and now account for 20% of sales at a price premium over broilers that is even larger. In Britain, by contrast, although free-range veal dominates the retail market with an 80% share, volume, which has been static, is currently declining.

I believe there are clear reasons for these differences which, to an extent, reflect the different attitudes and policies of the respective governments. In France, legislation was passed clearly defining free-range table poultry and requiring it to be categorically labelled. The consumer was presented with a product concept which was unequivocal and embodied desirable attributes - quality, purity, humanity, small traditional family farm origin, and reliability. The product qualities subsequently experienced of flavour and texture, reinforce the buying decision and justify the price premium.

In the U.K. it might be anticipated that free-range veal produced at no price disadvantage and with admirable antecedents might be expected to increase its sales. Three factors have militated against this: firstly veal is produced as a by-product from two dairy industry surplus commodities - bull calves and skim milk. The economic vis a tergo of the Continental industry requires the liquidation of these products, as veal, at any price and over-production has resulted in market prices below the cost of production. This has produced price competition quite unrelated to production costs. Even so, the British retail trade preferentially buys humanely reared veal as a self-protective measure, but at a modest premium to market prices for crated veal. What it fails to do is to countenance explicit product labelling which would jeopardise sales of intensively reared meat and poultry. The consumer therefore receives no compelling and persuasive message at the point of sale. The U.K. veal industry is unfortunately too small to rectify this by consumer advertising. Finally, veal is sold quite differently to poultry. The latter is mainly offered in whole carcass form directly to the consumer. Veal however is obviously portioned into many cuts, of which a substantial proportion are destined for the processing trade. This element is valued purely as a commodity and commands no price premium over similar cheap by-products from beef which have a substantially lower basic carcass value. In combination, these factors have reduced the profitability of the British industry and inhibited growth.

In conclusion, the ultimate widespread adoption of husbandry methods that more adequately meet the behavioural and ethological needs of farm animals will be dependent not on technical, but on market forces. These can be much influenced by Government attitudes and legislation in the field of food labelling.

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OPEN SESSION

THE IMPORTANCE OF THE RESPIRATORY TRACT IN TUBERCULIN REACTING CATTLE IN THE EPIDEMIOLOGY OF BOVINE TUBERCULOSIS

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Identifying the source of infection in tuberculosis breakdowns is very important with regard to the epidemiology of the disease and thus to subsequent control measures. Throughout Northern Ireland, the two principle sources of infection are the purchase of infected animals (approximately 30%) and spread from an infected contiguous herd (approximately 40%). A recent epidemiological survey of an area in Northern Ireland found that when infection became established, spread from contiguous herds accounted for 70% of breakdowns while movement of infected cattle was the principle method by which infection was introduced into the area (Report 1984a). Obviously for such spread to occur, excretion of M. bovis must take place.

Although it is generally recognised that tuberculous lesions are present in lymph nodes associated with the respiratory tract in over 90% of reactors with confirmed infection (Francis, 1971; Wilesmith, et. al., 1982; Report, 1984a), lung lesions are currently only found in 1-2% of such cases during abattoir examination in both Great Britain and Northern Ireland (Zuckerman, 1980; Report 1984a). When lung lesions are not detected it has been suggested that such cattle are non-excretors ("closed" cases) and thus unimportant in the epidemiology of the disease (Zuckerman, 1980). This may account for the small number of breakdowns attributed to spread from contiguous herds in Great Britain (3.4%) from 1972 to 1978 (Wilesmith, 1983) and more recently (3%) in the South West region of England (Report, 1984b).

The present study was undertaken to establish the frequency and relevance of tuberculous pulmonary lesions in tuberculin reacting cattle and thus clarify the role of the infected bovine in the epidemiology of the disease in Northern Ireland.

MATERIALS AND METHODS

During the 12 month period of the study, 1 or 2 reactors per week were examined. Only reactors identified at a tuberculin test in herds which were previously unrestricted, were used. Fifty-five such animals from 43 herds were submitted. Details of age, sex, tuberculin test result and previous testing history were obtained for each reactor and the following procedures carried out.

At the abattoir: A minimum of 2 ml of nasal mucus per animal was collected

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before slaughter using disposable sterile mucus samplers linked to a portable vacuum pump. The selected animals were subjected to the standard post-mortem examination for reactors. Extra care was taken during the examination to ensure that cross-contamination between lymph nodes did not occur. Left and right retropharyngeal, left and right bronchial, mediastinal and mesenteric lymph nodes were removed and placed in separate plastic bags. Other lymph nodes if found to have lesions were also included. The lungs were palpated but not incised and the entire pluck dissected from the carcase. Samples were stored at 4°C and delivered to the laboratory with 24 hours of slaughter.

At the laboratory: The lymph nodes were sectioned and samples submitted for bacteriological and histopathological examinations. A minimum of 2 ml of tracheal mucus was collected using the suction apparatus. The heart and diaphragm were carefully dissected from the lungs which were then inflated with 1.0 M saline solution. The inflated lungs were then dissected into individual lobes which were cut into sections 0.5 cm thick using a bacon slicer. Individual sections were examined over a light source and suspect lesions taken for bacteriological and histopathological examinations.

Histopathological examination

Lung and lymph node samples were fixed in 10% neutral formalin and processed by standard paraffin techniques. Sections of each were cut and stained with haematoxylin and eosin and also by the Ziehl-Neelsen method. Stained sections were examined for the presence of typical tuberculous lesions and acid-fast bacilli.

Bacteriological examination

Samples of lymph node tissues were processed individually. Lymph node and lung specimens were ground with sterile sand using a pestle and mortar. Decontamination was affected by oxalic acid (5% w/v), added during grinding. Suspended material was placed in a heating block for 20 minutes at 37°C, centrifuged at 3000 rpm for 15 minutes and the supernatant decanted. The deposit was inoculated using a cotton tipped swab onto 5 slants of Lowenstein-Jensen medium, 3 with pyruvate and 2 with glycerol. Inoculated slants were incubated at 37°C and examined after 28 days.

Nasal and tracheal mucus were processed as soon as possible after collection. Samples of mucus were digested and decontaminated using N-acetyl-L-cysteine/sodium hydroxide (Kubica et. al., 1964). The final suspensions were neutralised using 0.1 M HCL and phenol red indicator. Lowenstein-Jensen media were inoculated and incubated as above and in addition, guinea pigs were inoculated intramuscularly. After 28 days the guinea pigs were skin tested with avian and bovine-type PPDs, autopsies were carried out and selected lymph nodes cultured for mycobacteria. Smears prepared during the procedure were stained by Ziehl-Neelsen and Auramine-Rhodamine flurochrome methods and examined for mycobacteria.

RESULTS

Gross tuberculous lesions were detected during abattoir examination in 37 (67%) of the 55 reactors examined. Lesions were only detected in lymph nodes and tuberculosis infection was confirmed by laboratory examination in all suspected cases. Laboratory examination failed to detect evidence of infection in the remaining 18 reactors with no lymph node lesions visible.

Tuberculous lesions were identified in sections of lung tissue in 27 (73%) of the 37 reactors with lymph node infection, but no lesions were evident in lungs from the remaining 18 reactors. A total of 63 pulmonary lesions were found, giving an average of approximately 2 lesions per lung. The maximum number of lesions present was 9, but the majority of lungs (63%) had only one lesion present (Table 1). The lung lesions varied in size from 0.3 to 0.5 cm in diameter, but the majority (70%) were less than 1 cm and 32% were less than 0.5 cm (Table 2).

The lesions were evenly distributed between the left and right sides of the lungs (32 left, 31 right). Ninety per cent of all lesions were found in the diaphragmatic lobes, approximately half being located in the distal third. No lesions were detected in the intermediate lobe (Table 3). Only 2 lesions were in a sub-pleural location, all others being within the lung parenchyma.

A total of 72 lymph node lesions were found, giving an average of approximately 2 affected lymph nodes per reactor. Lymph nodes of the head and/or thorax were involved in all cases, an infected mesenteric lymph node being present in only one animal which also had pulmonary involvement. The distribution of affected lymph nodes was consistent with afferent drainage from lung lobes when pulmonary lesions were present, in all but 3 cases. Two of these had lesions confined to the retropharyngeal lymph nodes, while a third had a lesion present in the right bronchial lymph node but concomitant lung lesions were not detected in the ipsilateral apical and cardiac lobes.

Lymph node lesions consisted of typical granulomata with giant cells and multiple caseous foci, the majority of which were moderately calcified. Acid-fast bacilli were demonstrated in small numbers in almost all such lesions. Tuberculous granulomata present in lungs showed greater variation in nature with some consisting of highly cellular granulomata, while others had single or multiple caseous foci, several of which were mildly calcified. In general, calcification in lung lesions when present was not as pronounced as that in the corresponding respiratory lymph nodes. When multiple, necrotic or caseous foci were evident, smaller highly cellular granulomata without such foci were frequently present at the periphery of the lesion. In a few instances small granulomata of 1 mm or less in diameter were seen in lung sections distant from larger lesions which had been recognised grossly. Lung lesions involved alveolar, bronchiolar and/or bronchial tissue and some were evident extending into the lumina of the latter structures . Occasionally, lesions were also present in the alveolar septa and in the septal lymphatic vessels. Acid-fast bacilli were demonstrated in most of the larger lesions but were less frequently identified in the smaller granulomata. Some of the lesions observed on gross examination proved to be non-tuberculous in nature and consisted either of micro-abscesses, fibrotic tissue or parasitic-type granulomata.

M. bovis was isolated from 5 samples of nasal mucus and 4 samples of tracheal mucus taken from 7 reactors, all of which had lung lesions. Guinea pig biological tests were positive in the nine mucus samples while cultural procedures only detected 5. Although 3 of these reactors had multiple lung lesions, the remaining 4 had only single lesions of less than 1.5 cm in diameter (Table 4).

The interval from the last clear tuberculin test to the date of slaughter (testing interval) was calculated for each reactor, as a possible indication of the duration of infection. The interval for all reactors ranged from 5 to 15

Table 1. Distribution of Lungs by the Number of Lesions Present

Number of Lesions	Percentage of Lungs in each Category
One	63%
Two	15%
Three	7%
Four	7%
>Four	7%

Table 2. Distribution of Lesions by the Size of their Diameter

Diameter of the Lesion (cm)	Percentage of Lesions in each Category
0.0 to 0.5	32%
0.5 to 1.0	38%
1.0 to 1.5	6%
1.5 to 2.0	16%
>2.0	8%

Table 3. Distribution of Lesions by their Location

Lung Lobes Affected	Percentage of Lesions in each Lobe
Left Diaphragmatic	46%
Right Diaphragmatic	44%
Left Apical	3%
Right Apical	3%
Left Bronchial	2%
Right Bronchial	2%

Table 4. Details of reactors which were positive for *M. bovis* excretion

Testing Interval (Months)	Number, Size and Location of Lung Lesions	Lymph Nodes Affected
6	One (0.6 cm) R. diaph.	mediastinal
6	One (1.3 cm) R. diaph.	L & R retropharyngeal
9	One (0.8 cm) R. diaph.	L. bronchial & mediastinal
6	One (1.5 cm) L. diaph.	L. bronchial & mediastinal
12	One (1.2 cm) R. diaph. One (2.0 cm) L. diaph. One (5.0 cm) L. apical	L & R bronchial & mediastinal
12	Three (0.5 to 1.4 cm) L. diaph. Four (0.5 to 2.0 cm) R. diaph.	L & R retropharyngeal mediastinal.
12	Four (0.5 to 1.5 cm) L. diaph. Five (0.5 to 2.0 cm) R. diaph.	L. bronchial & mediastinal mesenteric

L = left; R = right; diaph. = diaphragmatic

Table 5. Tuberculin Test Results of Reactors

	Reactors with Confirmed Infection	Reactors without Confirmed Infection	Total
Reactors on Standard Interpretation	33	6	39
Reactors on Severe Interpretation	4	12	16
TOTAL	37	18	55

months with a mean value of 11.2 months (sd 2.77). No significant difference in this interval was found using Gossetts t test, between reactors in which infection was confirmed and those in which it was not. A similar level of non-significance was found between reactors with lymph node lesions only, and those with lung and lymph node lesions. However, 3 of the 7 reactors from which M. bovis was isolated in mucus had an interval of 6 months or less (Table 4) and the mean value for all 7 was 9.0 months (sd 3.0) which was significantly less ($p < 0.05$) than that recorded for the remaining 30 reactors with confirmed infection (mean 11.7, sd 2.9).

Thirty-nine (71%) of the 55 reactors were identified using standard interpretation at a tuberculin test. The remaining 16 (29%) were classified as reactors using severe interpretation, 6 had been inconclusive at a previous test while 10 were present in a herd test with reactors on standard interpretation. Only 4 of the reactors on severe interpretation had infection confirmed (Table 5).

DISCUSSION

In the present study 73% of reactors with confirmed lymph node infection also had detectible lung lesions. This compares with an abattoir detection rate of 1-2% in such animals and clearly indicates that abattoir examination alone cannot be used to classify reactors into excretors ("open cases") and non-excretors ("closed cases"). The practical difficulties experienced by meat inspectors under conditions of commercial slaughter will limit the detection of lung lesions to chance, unless they are large and/or superficial. Seventy per cent of lung lesions found in the study were less than 1 cm in diameter while 63% of lungs had only one lesion present. Further, most of the lesions were in the diaphragmatic lobes and in all but 2 cases were seated within the lung parenchyma. This corresponds favourably with previous work by Stamp (1948) who also found, using a similar detailed examination, what he considered to be primary lung foci in 89% of 100 tuberculous cattle. Again many lesions were less than 1 cm, present singly in lungs and located within the diaphragmatic lobes.

All lesions may not have been detected in the present study even by the meticulous techniques employed. Notably 32% of lung lesions had a diameter less than 0.5 cm, the thickness of the sections. Furthermore, histological examination revealed small granulomata of less than 1 mm in diameter distant from grossly observed lesions. Lung lesions similar to the above may well have been present in those reactors with lesions in respiratory lymph nodes only.

The presence of concomitant lung and lymph node lesions in pulmonary tuberculosis has previously been well accepted in the pathogenesis of the disease. Francis (1958) records that lesions in respiratory lymph nodes are secondary to those of the lung, while Jubb and Kennedy (1970) state that the combination of lesions in the initial focus and regional lymph nodes is known as a primary complex and is always present. These latter authors also suggest that the correspondence between the incidence of pulmonary lesions and lesions in the bronchial and mediastinal lymph nodes is largely a reflection on how diligently the lungs are searched, a view strongly supported by the current study. The failure to detect lesions in the bronchial and mediastinal lymph nodes in 2 of the reactors with lung lesions, may reflect a difficulty in completely dissecting these glands, especially in cattle with excess adipose tissue. However, Medlar (1940) found lung lesions in 10% of 200 reactor cattle which showed no visible lymph node involvement on careful macroscopic examination and suggests that failure to find visible glandular tuberculosis does not mean that lung lesions are absent.

Many previously established facts on the pathogenesis of bovine tuberculosis and the importance of tuberculous cattle in the epidemiology of the disease, have not been considered applicable to current eradication procedures. Thus it has been suggested that present day tuberculin testing regimes ensure that virtually all tuberculous cattle are detected and slaughtered at a very early stage of infection, before they can become excretors (Report 1978). However, the sample of reactors in the study was identified over a one year period at tuberculin tests throughout Northern Ireland and had a mean testing interval of less than one year. Northern Ireland has a similar testing regime to many other regions of the British Isles. Since 1982 all cattle are tested annually and in areas or herds which are considered at risk at least every 6 months. Thus 3 of the 7 reactors from which M. bovis was isolated in mucus had a testing interval of 6 months or less and would suggest that lung lesions and excretion can occur rapidly even with present day testing regimes. Further, excretion of the organism is probably common as M. bovis may well have been identified in many more animals by sequential mucus sampling. This technique is consistently recommended for the detection of tuberculosis in human cases where morning and 24 hour pooled mucus samples are taken over several days (Karsnow and Wayne, 1969). Also several of the lung lesions were evident, on histological examination, extending into the bronchial and/or bronchiolar lumina in reactors from which M. bovis was not isolated in mucus.

These findings reiterate the views of Stamp (1944) who postulated that the vast majority of tuberculous cattle are excreting tubercle bacilli directly from the lung, since pulmonary tuberculosis in its commonest form is an "open" lesion almost from its inception and that the majority of infected animals are a source of danger from the start of the disease process.

The study also corroborates the criterion adopted in Northern Ireland since the introduction of a compulsory eradication scheme in 1959 for deciding if spread from a neighbouring herd has occurred. This involves establishing that cattle with confirmed infection were present in neighbouring fields, independent of the detection of lung lesions during abattoir examination.

Cattle to cattle contact over boundary fences ("nosing") is known to occur and could easily facilitate the transmission of M. bovis from one respiratory tract to another. The known susceptibility by this route has been recorded by Francis (1947) who cites previous work by Chausse which suggests that respiratory tract infection can be established by 6 or less bacilli.

CONCLUSIONS

The results of this study confirm the continued importance of the infected bovine in the epidemiology and eradication of bovine tuberculosis in Northern Ireland. Tuberculous cattle can not be classified epidemiologically into "open" and "closed" cases by the detection of lung lesions during abattoir examination. Rather, all tuberculous cattle with lesions in respiratory lymph nodes should be regarded as possible excretors and thus an important source of infection for other cattle both within and between herds. The occurrence of both pulmonary lesions and excretion of M. bovis is not only common but appears to take place rapidly and is not prevented by the frequency of testing currently used to monitor herds.

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SURVEILLANCE OF INFECTIOUS DISEASES USING ABATTOIRS

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Monitoring of disease conditions readily observed at slaughter has been carried out for some time, through the medium of meat inspection by qualified veterinarians or other trained staff. This monitoring is essentially related to reasons for condemnation, and no action is taken other than the condemnation of the affected meat. The only exception in the UK is in surveillance of tuberculosis and EBL in cattle, where after laboratory confirmation affected animals are traced back to their herd of origin and the herd tested as part of the control of these notifiable diseases.

A natural extension of this practice is to collect blood or tissue samples regardless of any pathological signs and submit them to laboratory tests. We can still distinguish a monitoring activity, intended to give information about the prevalence of a response to a laboratory test in a slaughter population, from a surveillance activity where, by tracing positives, further action at the herd of origin will result in a reduction of risk to others.

This distinction is wider than that which the epidemiologist would normally make between monitoring and surveillance (Beal, 1983; Langmuir, 1976), but it is useful in this context because of its implications for the design of the investigation in question. A few thousand samples will often give a sufficiently accurate estimate of prevalence, but their effectiveness as part of a control scheme will depend on what contribution they make to the overall objective of reducing risk to other herds.

Davies et al (1976) specify four practical conditions for surveillance by test and trace-back to be possible. These are that the prevalence is low, that carcasses and offal are properly identified, that movements from farm to slaughter are fairly direct, and that tracing will normally result in action. It is the identification of animals and tracing of movements that seems to present the greatest practical difficulty.

Further difficulties lie with the interpretation of the results for monitoring purposes. On-farm surveillance, with visits to a sample of farms or even all farms at regular intervals, can be designed to produce results which can very readily be interpreted. The cost of farm visits however, is such that slaughterhouse surveillance can be the only practicable method of detecting cases which might be unreported and not ascertained by movement tracing or other connection with a known case. It is then important to make the best use possible of surveillance results, and to design the surveillance scheme so that some interpretation becomes possible.

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ABATTOIR SURVEILLANCE OF PIGS IN THE UK

In the 1970's a sequence of abattoir surveys was carried out with the main intention of monitoring, and detecting cases of, swine vesicular disease (SVD). The sixth survey, described by Hendrie et al (1978), showed that there was very little infection remaining but abattoir surveillance for SVD continued until the end of 1984. Samples collected in abattoirs were also tested for Aujeszky's disease, and with an eradication scheme beginning in March 1983 abattoir surveillance of culled sows and boars has become an important means of case detection.

TRACING BACK TO HERDS OF ORIGIN

Without any system of marking pigs in general use, tracing back to the herd of origin is dependent on records of each movement being kept. It is only practicable to follow these results up in the event of a positive test result, so without making special efforts, there is no information on the origin of pigs with negative results. On tracing back, it is often found that the pig has passed through the hands of a dealer who keeps a pool of culled sows, and although his movement records may be in good order, without identification of the pig a large number of origins are possible. On average it has been necessary to visit about four or five farms for every tracing exercise initiated.

Difficulties of tracing back are aggravated by the inevitable delays of laboratory testing. On occasions when a condition requiring tracing (such as a vesicle) is seen in a slaughterhouse, tracing is initiated immediately and memories are fresh enough to supplement written records.

SELECTION OF SLAUGHTERHOUSES AND SAMPLE SIZES

Where possible, it is preferable to specify the slaughterhouses to be used and the number of samples to be collected from each. This was not done in the earlier SVD surveys but in the sixth survey a scheme based on known abattoir sizes (potential throughputs) was used, and this gave some confidence of a fair spread of herds being included. Subsequently, sampling has been extended to include a substantial proportion of culled sows and boars, and the number of samples collected from each abattoir has depended on more practical considerations such as the co-operation of the management.

The intention of the design of the scheme should be to ensure that each pig slaughtered has an equal chance of being sampled. Without a randomisation scheme based on a sampling frame, or list of units to be sampled, this is impossible. Nevertheless, by giving a quota of samples to be collected in each slaughterhouse proportional to the known throughput of that slaughterhouse and by including all slaughterhouses with a substantial throughput together with some with a lower throughput, one hopes to produce a result which can be interpreted in terms of a random selection.

THE LIKELIHOOD OF A HERD BEING INCLUDED

A slaughterhouse survey will inevitably be biassed, in the sense of not giving every herd an equal chance of being included. The most obvious source of bias is the number of pigs slaughtered, which is directly related to herd size. The slaughterhouses used are another bias since they may be regularly taking pigs from some herds while other herds will go elsewhere. There is in particular a suspicion that smaller herds may use smaller slaughterhouses, and efforts need to be made to include some such slaughterhouses even though the yield in samples collected may be low.

The significance of the bias in the sample from a monitoring point of view is that any estimates of prevalence that result will be weighted towards herds favoured by the sampling scheme, particularly larger herds. The implication for surveillance is that unless supplementary measures are taken, disease in smaller herds will be missed. Whether this is significant will depend on other factors.

The herd size bias is significant only when herds rather than individual animals are the main units of interest, as is the case in a control scheme. A slaughterhouse survey should be able to give a fair estimate of prevalence in the pig population, which would be sufficient for an economic appraisal of disease loss.

INFERENCES FROM A SLAUGHTERHOUSE SURVEILLANCE SCHEME

There is no difficulty in estimating a prevalence in pigs, by dividing the number of positive samples by the total number of tests. Small herds do not make a substantial contribution to the total pig population so any failure of the scheme to include such herds is relatively unimportant. The main points to remember in interpretation are:-

(i) the prevalence is averaged over the period of the survey

(ii) the prevalence in slaughtered pigs may not be the same as in the those left on the farm

(iii) if large consignments of pigs from a single origin are included in sample, the variance of the estimate will be greater than binomial.

When trying to estimate a prevalence among herds, or the number of infected herds (an infected herd being defined as a herd containing one or more pigs positive to the test), then the sample is far from being a simple random sample so a simple estimate of herds found positive divided by herds sampled will be biassed. In any case, we do not know the number of herds sampled without tracing back all the negative samples! If, however, we can regard the sample as a random sample, then an unbiassed estimate of the number of infected herds is simply

$$\sum \frac{1}{P}$$

summed over all infected herds found by the survey, where P is the herd's probability of being found by the survey.

The principles involved in calculating the unbiassed estimate are the same as those in calculating an estimate from a stratified random sample (Woods, 1985, page 43). So all we need to do is to look at each of the herds found and try to assess its probability of being found without the benefit of hindsight. This can be done by discovering how many pigs they slaughter, what proportion go to slaughterhouses being monitored, the proportion of throughput in those slaughterhouses sampled, and the proportion of pigs on the farm that are positive to the test.

In the case of a stratified random sample the strata are deliberately introduced to reduce the variance of the estimate. In this case however, the variation in weights has the opposite effect. The variance of the estimator is

$$\sum \frac{(1-P)}{P}$$

summed over all infected herds. If there are infected herds with very low values of P (because they have a low chance of being detected) they will inflate the variance enormously.

If there are parts of the population that the survey does not cover, it is not surprising that it cannot produce an accurate description of the whole population. It can however produce a reasonably accurate estimate of numbers of infected herds in that part which it does cover. If a lower limit for P(or upper limit for 1/P) is taken we can estimate the number of infected herds below 1/P, and use that number of herds to estimate the variance of the estimate. For different limit values for P there will be different estimates (Fig. 1):

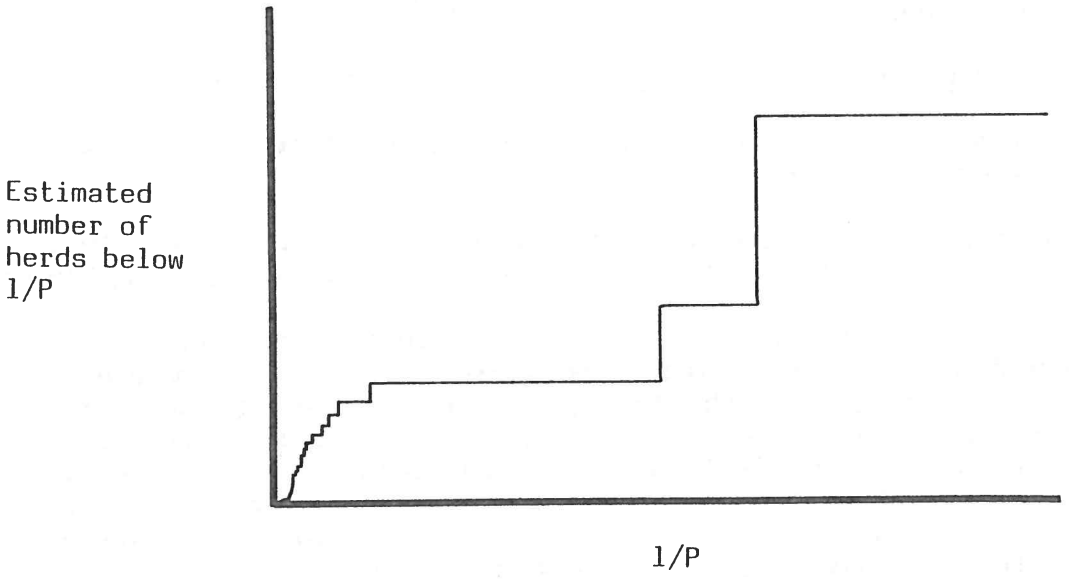

Fig. 1. Estimates of number of infected herds

If the survey has sufficiently large coverage, it may be possible to decide a value of 1/P above which most herds are not of interest (too small, or too low a within-herd prevalence). To the extent that this is possible the survey can give an estimate of numbers of "interesting" herds. The estimate is still subject to conditions i-iii above.

DETECTING CHANGES

If a slaughterhouse surveillance scheme is operated over a period of time, then what is even more important than estimating prevalence is to know whether the prevalence is increasing or decreasing. The first approach to this is to examine the rate of positives in the samples collected each month and decide whether there is evidence of a change in that rate. The 'cusum' plot is ideal for this purpose. By plotting the total number of positives found from the start of the scheme to the end of each month against the total number of samples collected, a rising graph is produced (Fig. 2):

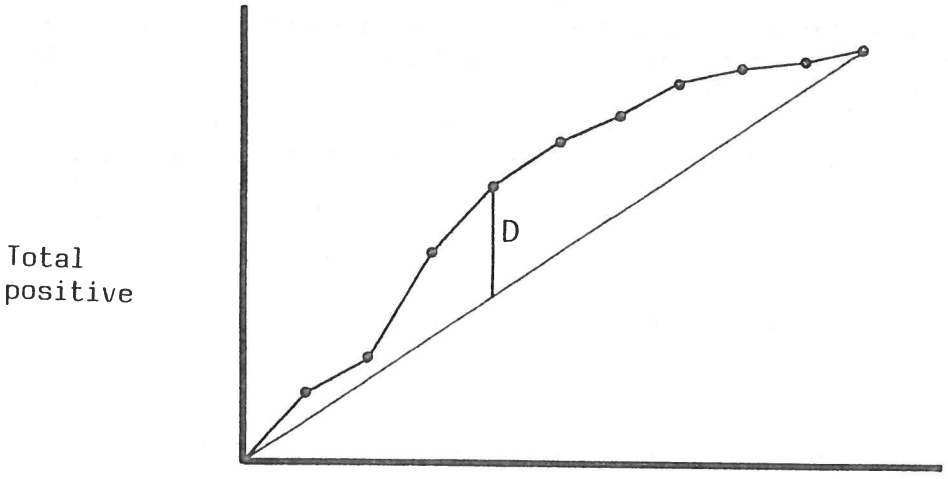

Fig. 2. Cusum chart of positive samples

If the rate of increase is falling off, then this provides evidence of a decrease in the rate of positives being found. It can be tested statistically by measuring the maximum difference D between the graph and a straight line joining the origin to the last point. The distribution of D under the null hypothesis is then found by simulating n random numbers between 1 and N (where n is the total number of positives and N the total number of samples). If these n numbers are the positions of the positives in the series of samples, a value of D can be calculated and this repeated several times.

This method of comparison over time successfully deals with the problem of varying numbers of samples being tested each month, but there are other potential sources of a spurious result. First, the problem of large consignments of pigs. When more than one positive is found among a batch of pigs with the same immediate origin, only one tracing exercise is initiated and so it is better to count tracings rather than actual positive samples. Secondly, the number of samples for each slaughterhouse will not be in exact proportion each month so if there are slaughterhouses with higher apparent rates of positives, they may be preferred in later

months so that an increase appears where there should be a decline in the overall rate. This can be corrected by calculating an "expected " number of positives for each slaughterhouse separately each month on the basis of its own results over the whole period, and plotting the cumulated total number of positives against the cumulated expected number. It is of course necessary to know where each positive was collected and how many samples were collected for each slaughterhouse each month. A third possibility is that there may be changes which are distorted by the bias of the survey, so that for example an apparent increase in rate of detection of positives could be the result of a small increase in prevalence among larger herds together with a large decrease in smaller herds. Apart from keeping a watch on the sizes of herds discovered each month, only a simulation of the dynamics of infection can shed any light on what is possible.

SIMULATING THE EFFECTS OF SLAUGHTERHOUSE SURVEILLANCE

If slaughterhouse surveillance is the main means of case-finding, then there is a suspicion that there could be an increase in numbers of small infected herds without this being apparent in the cases detected by the survey. To examine this possibility, a "worst-case" simulation can be carried out where it is assumed that slaughterhouse detection is the only means whereby an infected herd becomes uninfected; the chance of detection is proportional to herd size; and the chance of a herd becoming infected is proportional to the number of infected herds in existence, regardless of size.

This simulation depends on five parameters:

(i) the size distribution of the population

(ii) the size distribution of the initially infected herds

(iii) the probability of detection, per pig, per month

(iv) the number of initially infected herds

(v) the contact rate (probable number of newly infected herds, per infected herd, per month).

The first is available from national statistics, the second from records of previously discovered herds, and the third from national slaughtering rates. This leaves two "unknowns" and the number of cases detected by slaughterhouse survey can be used to get an approximate relationship between them (Fig.3):

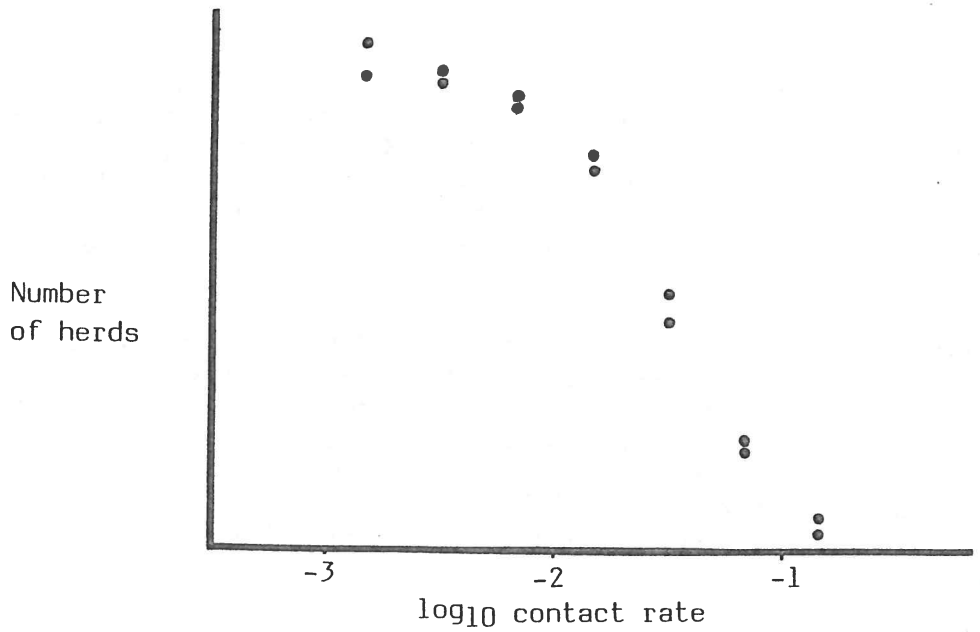

Fig. 3. The number of initially infected herds implied by one herd detected in months 13-24

This shows that quite a good estimate of the number of infected herds can be obtained by making assumptions about the nature of the spread of infection. Unfortunately, the results of the surveillance are not very sensitive for deciding what the contact rate ought to be. We might hope a high contact rate would result in a significant trend in numbers of cases detected each month over the year, but in this example simulation shows this is not necessarily the case (Fig. 4):

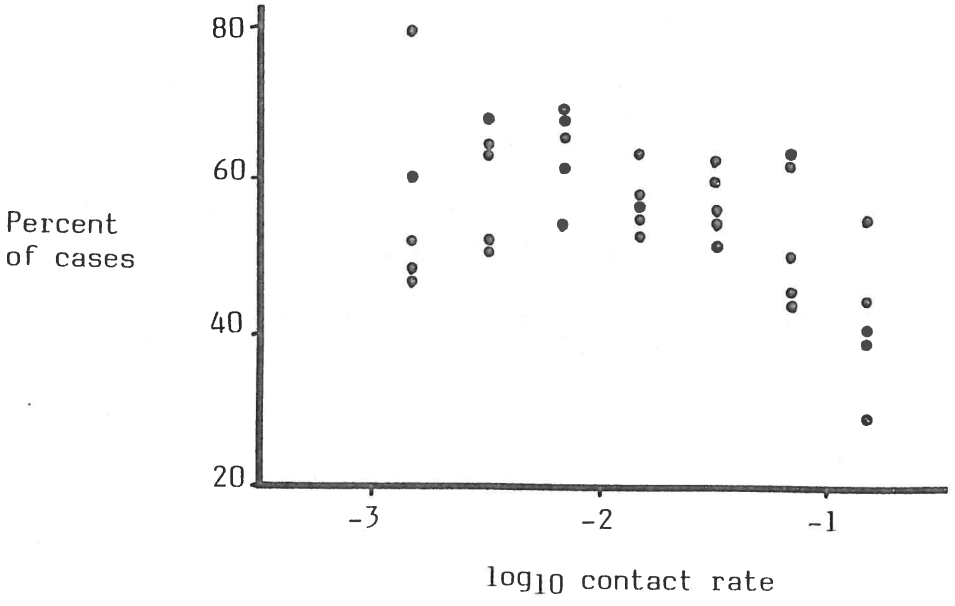

Fig. 4. The proportion of cases detected in the first half of the year

DISCUSSION

Despite all its drawbacks, slaughterhouse surveillance is the only way of getting information on disease that may be anywhere in a large population, without the massive expense of on-farm enquiries. It is important therefore to get the maximum possible information out of such a scheme, and also to realise its limitations and be prepared to supplement its results where necessary with special studies or other epidemiological knowledge.

In order to get the most out of a surveillance scheme, the importance of accurate information recording cannot be over-emphasised, and as happens so often it turns out that information which, at first glance, would not be thought particularly important turns out to play a vital part - in this case, it is the assessment of the chance that a herd that was detected, would have been detected in a hypothetical randomisation. This plays the role of the denominator which the epidemiologist is, as usual, seeking.

Simulation has a useful part to play in demonstrating the limitation of slaughterhouse surveillance. It shows clearly that very little can be inferred about the dynamics of infection from a moderate number of cases detected in slaughterhouses. It points out the dangers of relying entirely on slaughterhouses for case detection, and if better quantitative information on the effect of herd size on the probability of contracting or of spreading disease were available, it could provide more exact quantitative predictions.

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DATA RECORDING AND ANALYSIS IN DAIRY HERDS

THE DERIVATION OF PROGNOSTIC INDICATORS IN THE DOWNER COW SYNDROME: AN EPIDEMIOLOGICAL APPROACH TO A MULTIFACTORIAL CONDITION

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The downer cow condition was first described in 1955 (Hallgren) and although it has since been studied by several workers (Boyd 1964, Fenwick 1969, Jonsson & Pehrson 1969, Cox 1982 and Cox et al 1982), many aspects of the aetiology are still uncertain. Accurate figures for the incidence of the Downer Cow Syndrome are not available. However, an estimate may be made from the incidence of milk fever and the proportion of milk fever cases which later become downer cows. Using the data from Mullen (1975) and Cox (1982) an incidence rate of 0.47 cases per 100 dairy cows per year can be derived. With a national dairy herd of 2.8 million (MMB, 1984) this would result in approximately 13000 cases per year. As there are other causal factors in addition to milk fever (Andrews 1983), it can be appreciated that the downer cow is of considerable economic importance.

The downer cow presents a difficult clinical problem, often showing few clinical prognostic signs and requiring intensive nursing throughout an often protracted time course to maintain any chance of recovery. This difficulty has lead to the recent interest in the development of prognostic aids. In 1983 Andrews reported that changes in blood biochemistry levels might be used to predict the prognosis, and in 1983 the British Cattle Veterinary Association, in conjunction with Bristol University, set up a survey to investigate prognostic indicators for the downer cow. This paper presents the methods that were used to derive the prognostic indicators for the downer cow and then looks at ways in which the usefulness of the predictors may be assessed. As neither of the authors are statisticians by training, the theory and detail of the statistical procedures is not discussed.

In the survey the following definition of the Downer Cow, derived from that of Cox (1982), was used:-

> A cow that has been in sternal recumbency for more than 24 hours and which is not, at the time of examination, considered to be suffering from any specific metabolic or microbial infection, and has no clinically apparent structural injury which would definitely prevent her from rising.

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This is a clinically orientated definition applied early in the disease process and as such it is likely that cases of different aetiologies are included in the survey. The fact that the syndrome being considered is a poly-aetiological, multi-factorial condition must be borne in mind throughout the analysis, particularly when assessing the value of possible prognostic indicators.

METHODS AND MATERIALS

Approximately 50 members of the BCVA agreed to take part in the survey. They were asked to visit downer cows at twenty-four hour intervals and record the clinical signs and management of the case and collect blood samples. Between October 1983 and April 1985 64 downer cows and 8 normally calving control cows were included in the survey. The original aim was to take a blood sample 24 hours after the cow went down and then collect further samples at 24 hour intervals so that the changes in blood biochemistry could be investigated in a manner similar to that used by Andrews (1983). However, due to the practical constraints of general practice, this sampling protocol was not possible for most of the cases considered. The 64 cows sampled yielded 142 samples: the initial visit was at a variable time after the cow went down and a varying number of subsequent visits were made at varying time intervals thereafter. Rather than discard the major part of the data that did not conform to the original protocol, the samples were subdivided by sampling day for analysis. This created a problem because such sub-divisions contained a grouping of animals that was neither wholly dependent nor wholly independent of any other sampling day group. To allow valid statistical analysis to be performed, two assumptions have been made. Firstly, each sub-division is assumed to be representative of a population of downer cows sampled on that day, and secondly, that changes between sampling days are representative of daily changes within a population of downer cows.

Assessment of accuracy of prognostic indicators

There are various methods of interpreting test results as clinical indicators. As most tests are not pathognomonic for a clinical condition there will always be some error in the test interpretation; the various methods possible cope with such errors by various means. The classical method of clinical decision analysis assumes that the medical decision is dichotomous and so dichotomises the results of the test (Weinstein & Fineberg 1980), using a single cut-off point to determine the decision. The test's error is expressed as the level of false negative and false positive results. However, many predictive medical tests are not dichotomous but have three decision choices: positive, negative and inconclusive. An example is that of *per rectum* pregnancy diagnosis of cattle at six weeks *post coitum*. A dichotomous decision with a high level of false negatives and positives would be undesirable; a method using three decision options that has a higher level of accuracy of prediction with a proportion of cases that cannot be predicted at examination is preferable.

A more elegant method still, that can be used for test results of a continuous nature, is to divide the test score range into a number of sections and then determine the sensitivity and specificity for each section (Weinstein & Fineberg 1980). This allows a proportion of the cases to be predicted with a high degree of accuracy yet still predicts, to some degree of accuracy, all the cases considered. However when deriving a decision analysis method from practical data the more elegant the method, the more cases are needed to position the various cut-off levels and assess the accuracy of the predictions made. In the case of the downer cow it was decided that the dichotomous method was unsuitable as the prediction would be used to decide the animal's fate, and that a predictor using three decision choices should be used. The problem then arose of obtaining the optimal balance between the proportion of cases with an inconclusive test result and the accuracy of the conclusive results. This compromise could be resolved in an iterative manner using a computer but it was felt that there were insufficient cases available to do so. It was therefore decided that the conclusive predictions should be 100% accurate and that all the predictor error should be accommodated in the inconclusive prediction. The method of determining the cut-off points is described below.

Positioning of cut-off points: To determine the cut-off points and extent of the 'grey area' of uncertainty between the two points, the following method (illustrated in Fig. 1) was used. The predictor value for each case was plotted on a number line with some indication of the case outcome. Cases that are positioned closer to a case of the opposite outcome than a case with the same outcome are considered to be in the 'grey area' of uncertainty for which a prediction cannot be made. Having determined the approximate extent of the 'grey area', the cut-off points are positioned at each end such that they are equidistant between the point at the edge of the 'grey area' and that point closest to, but not in, the 'grey area'. The accuracy of the predictor was then calculated as:

$$\frac{\text{No. of cases for which a conclusive prediction made}}{\text{Total No. of cases considered by the predictor}} \times 100 \quad (1)$$

SINGLE FACTOR PROGNOSTIC INDICATORS

Duration of the case

There were 51 cases for which the time interval between the animal becoming recumbent and the case resolving were known. The average survival rate was 49%. The survival rate for animals recumbent for various times is plotted against the case duration in Fig. 2. Differences in survival rate between different case durations were tested for significance using the Chi-square test. Cases that resolved before day three had a significantly higher ($p < 0.05$) survival rate than cases that resolved later than day three. Cases that resolved three to five days after becoming recumbent had a poorer survival rate ($p < 0.05$) than cases that resolved later than five days. However there is no time at which it could be predicted that cases not yet resolved would have a certain outcome. Thus the length of time that an animal has been recumbent cannot be used as a prognostic indicator.

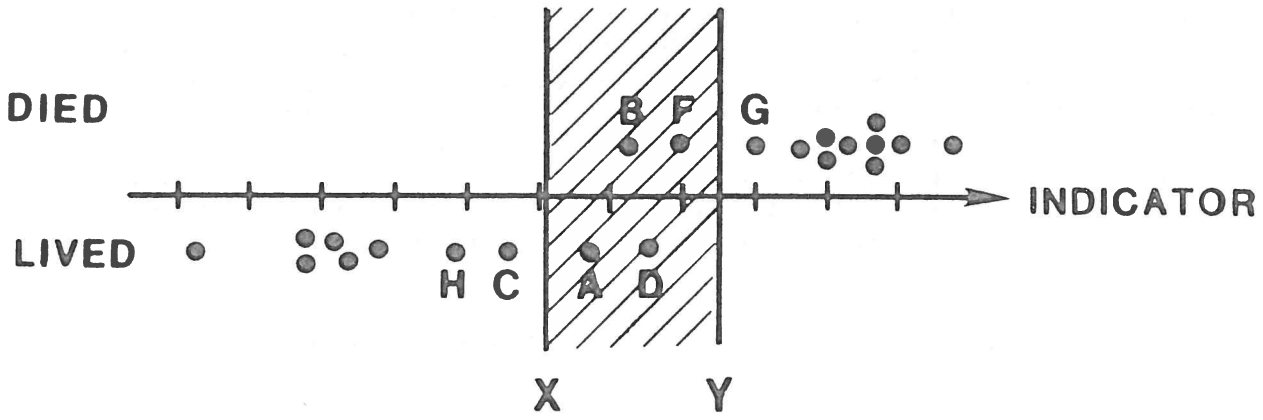

Fig. 1. Method of determining 'grey area' and cutoff levels for theoretical indicator number line. A to B less than A to C so A is in 'grey area'. F to D less than F to B so F is in 'grey area'. Cutoff line X equidistant between A and C. Cutoff line Y equidistant between F and G. 'Grey area' extends from X to Y.

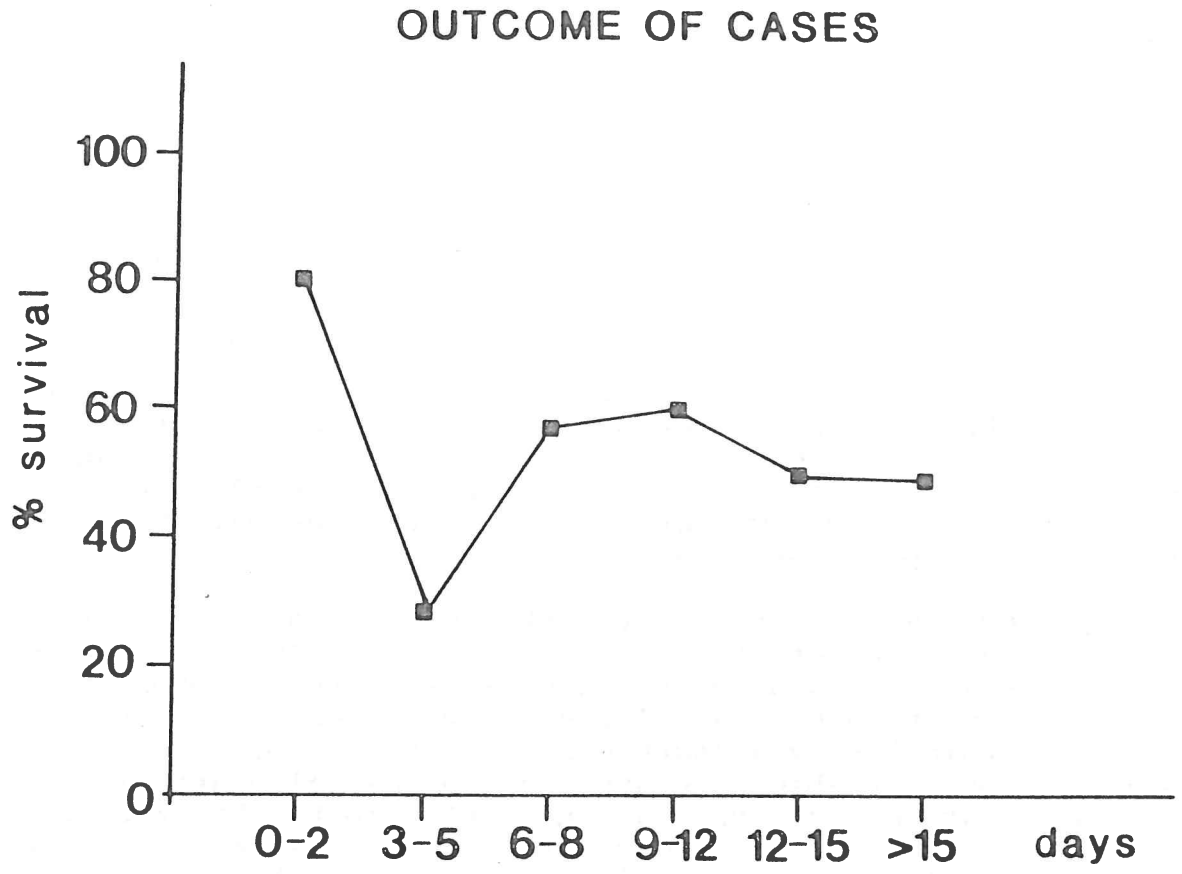

Fig. 2. Duration of cases. Time taken for case to resolve plotted against survival rate for the 51 cases where length of recumbency known, with the case duration subdivided by day.

Apparent cause of recumbency. Table 1

The survival rate of cases recumbent after hypocalcaemia is significantly higher ($p < 0.05$, Chi-square test) than the average survival rate. This suggests that a knowledge of the cause of recumbency may be a useful prognostic indicator. However in 36% of the cases considered experienced cattle practitioners could not determine the cause of recumbency, a difficulty previously noted by Jonsson and Pehrson (1969). Such difficulties are likely to limit the prognostic value of knowing the causal factor.

Table 1. Apparent Cause of Recumbency with Survival Rates for each Category

Factor	Cases	Survived
Dystokia / at Calving	24	33%
Down in Cubicles / Yard	6	33%
Milk Fever	5	80%
Post Calving	3	33%
Bulling Injury	2	50%
Not Known	22	-

Clinical signs, case management and blood biochemistry factors

Eleven aspects of the case management and clinical signs were extracted from the case proformae and the blood samples were analysed for a total of 16 serum components. In certain instances the case proformae were incomplete, or there was insufficient serum for the full biochemical analyses. Such cases have been omitted from the appropriate analyses.

Using procedures from the Minitab statistical package (Ryan et al, 1981) where appropriate the association between the outcome and each factor was calculated as follows: For the dichotomous factors the association was calculated using either the Chi-square test, or where this test was inappropriate, the Fisher Exact test. For the continuous variables the association was calculated from the Pearson product moment correlation co-efficient. The result- and 'p' values are given in Table 2 for those factors for which the result was available for the majority of cases and that had a 'p' value less than 0.3 at some stage. Several factors show a degree of association on some of the days considered with the quality of nursing and the Creatinine phospho-kinase (CPK) being significantly associated with the outcome on most days. This suggests that several of the factors may be worth considering as prognostic indicators. The predictive accuracy of the factors in Table 2 are given in Table 3. For the continuous factors the method used to

Table 2. Association of single factors with case outcome for day groups zero to greater or equal to five days.

Factor	Day 0	1	2	3	4	⩾5
Bedding Good or Bad	-	-	-	-	-	+
Bright or Dull	-	+	-	+	-	+
Attempting to Rise Yes or No	-	+	-	*	+	+
Eating Yes or No	-	+	-	+	+	-
Site Inside or Outside	-	+	+	-	-	-
Hoist Used or Not used	-	+	-	-	-	-
Nursing Good or Bad	*	***	**	+	***	***
Condition Score 1 to 5	-	-	**	-	-	+
Creatinine Phosphokinase log (u/l)	*	*	+	*	***	*
Amino-aspartate transferase log (u/l)	-	+	+	-	+	**
Urea log (mmol/l)	+	**	-	+	*	-
Calcium mmol/l	-	-	+	-	+	+
Magnesium mmol/l	-	+	+	+	-	*
Phosphorus mmol/l	-	+	-	+	-	*

(- = $p > 0.30$, += $p < 0.30$, *= $p<0.05$, **= $p<0.01$, ***= $p<0.001$)

Table 3. Percentage predictive accuracy of single factors considered for days zero to greater or equal to five days

Factor	Days 0	1	2	3	4	≥5
Bedding Good or Bad	50	41	57	38	58	57
Bright or Dull	65	55	46	56	46	56
Attempting to Rise Yes or No	47	58	54	81	69	68
Eating Yes or No	56	57	52	56	62	53
Site Inside or Outside	54	62	64	78	50	56
Hoist Used or Not used	50	60	53	64	44	48
Nursing Good or Bad	79	77	77	62	100	88
Condition Score Scored 1 to 5	0	0	12	0	8	36
Creatinine Phosphokinase log (u/l)	28	11	15	50	100	33
Amino-aspartate transferase log u/l)	21	14	36	0	0	37
Urea log (mmol/l)	6	31	0	0	54	0
Calcium mmol/l	10	6	12	12	33	25
Magnesium mmol/l	0	0	0	18	0	21
Phosphorus mmol/l	19	15	0	50	0	0

calculate the accuracy was as given above, for the dichotomous factors the accuracy was calculated as the percentage of cases for which the outcome was predicted correctly.

Although many of the factors considered were significantly correlated with the case outcome, only a few were able to predict the outcome of more than 75% of cases. As 75% accuracy is only a two-fold improvement on random prediction, and an animal may be slaughtered according to the prediction, such accuracy ought to be considered too low; an accuracy in excess of 90% should be sought. This reduces the useful indicator factors to the quality of nursing and CPK levels on day 4.

As previously stated, the downer cow syndrome is a poly-aetiological, multifactorial condition with numerous factors that can influence the case's development. Therefore it may be an over-simplification to attempt to predict the prognosis using an indicator based on only one factor as it will fail to consider those cases for which other factors are of greater importance. On this point it is interesting to note that one of the more useful factors considered, that of the quality of the nursing, is of a multifactorial nature as it requires the assessment of several aspects of the case management whereas the other factors are only examining one aspect of the case. Because of this multifactorial composition, the quality of nursing could be expected to be a more useful indicator and suggests that a truly miltifactorial indicator may give a more accurate and robust prediction.

MULTIFACTORIAL INDICATORS

Multifactorial indicators were derived for days one, two, three and four using a form of multivariate analysis called Canonical Variate Analysis (CVA) (Avery et al, 1983). This is a type of cluster analysis that looks at a set of cases that are subdivided into predefined groups and for which several factors are know. It calculates the linear combination of the factors considered that gives the best separation of the groups by maximising the ratio of the between-groups to the within-group variance. When, as here, only two groups are considered, the resultant combination of factors approximates to the best least-squares fit (Avery et al, 1983).

For each of days one, two, three and four all factors associated ($p < 0.3$) with the outcome (see Table 2) were considered in the initial analysis. Factors associated at the $p < 0.3$ level were used to reduce the chance of making a Type II statistical error; that of omitting a factor that was actually associated with the outcome (Thrusfield & Aitken 1985). Blood enzyme results were log-transformed where necessary to produce a more normal distribution and all the continuous variables were divided by the average value from the eight control cows to remove the effects of differing scales of measurement. The initial CVA was run using the transformed factors and the separation of the two outcome groups was tested for significance by examining the value of Mahalanobis's distance. If the separation was significant ($p < 0.05$) those factors for which the CVA derived loading was less than 0.01 were tentatively omitted and the analysis re-run. If the resultant

separation was still significant ($p < 0.05$) the factor was omitted permanently.

The final indicators derived for each day were:-

Day 1

Indicator =	-2.43	Quality of nursing	Good = 1 Poor = 0
	+2.44	log (Urea)	mmol/l
	+1.29	log (AST)	u/l
	-0.90	log (CPK)	u/l

Mahalanobis's Distance = 1.88 ($p < 0.05$)

AST = Aspartate Amino Transferase

Day 2

Indicator =	0.96	Condition Score	0 to 5
	+2.24	Quality of nursing	Good = 1 Poor = 0
	+1.13	Calcium	mmol/l
	-1.00	log (AST)	u/l
	+0.74	log (CPK)	u/l
	-1.02	Magnesium	mmol/l

Mahalanobis's Distance = 3.01 ($p < 0.01$)

Day 3

Indicator =	4.67	Attempting to rise	Yes = 1 No = 0
	+3.44	Magnesium	mmol/l
	-3.10	log (Urea)	u/l
	-0.11	log (CPK)	u/l

Mahalanobis's Distance = 3.01 ($p < 0.01$)

Day 4

Indicator =	2.71	log (CPK)	u/l
	-1.16	Attempting to rise	Yes = 1 No = 0

Mahalanobis's Distaice = 4.07 ($p < 0.05$)

Number line plots were produced for each factor using the method described and the 'grey area' and cut-off points were determined. From this the percentage accuracy was calculated. The cut-off points and accuracy for each day are:-

Day 1

Indicator	< -0.15	LIVE	Accuracy of 52% with
Indicator	> 2.00	DIE	29 cases considered

Day 2

Indicator	< 5.25	DIE	Accuracy of 81% with
Indicator	> 6.75	LIVE	21 cases considered

Day 3

Indicator	< 3.05	DIE	Accuracy of 83% with
Indicator	> 5.23	LIVE	12 cases considered

Day 4

Indicator	< 7.95	LIVE	Accuracy of 92% with
Indicator	> 9.14	DIE	12 cases considered.

For the predictor to be of clinical use a high degree of accuracy in excess of 80% is required. The predictor produced for day one with an accuracy of 52% will result in no prediction being made for approximately one in every two cases considered, making it of little practical use. Conversely, the predictors for days two, three and four with accuracies of 80-90% will result in few prediction errors. The predictors derived above suggest that it is possible to predict the outcome of downer cow cases on days two, three, and four after the cow becomes recumbent with an adequate degree of accuracy. The predictors are currently being used in a continuation of the BCVA investigation to predict the prognosis of cases for which the final outcome is later known. This is allowing the validity of these predictors to be assessed.

INTERPRETATION AND ASSESSMENT OF CANONICAL VARIATE ANALYSIS RESULTS

The second part of this paper is concerned with ways in which the merits of the multifactorial indicators may be assessed, using the previously produced predictors as examples where possible. There are few references in the literature regarding the evaluation and assessment of prognostic predictors, especially for veterinary applications, and so the methods described here are only an initial suggestion; discussion and comments would be welcomed.

One of the major limitations of Canonical Variate Analysis is that the resulting loadings are only mathematical artefacts (Avery et al, 1983). The set of loadings produced are only one of several possible sets that could be used to maximise the separation between clusters. If the factor values were to be re-ordered, transformed and rotated, it is probable that other, equally significant sets of loadings would be derived. It follows from this that neither the sign not magnitude of a factor loading can be taken as an indication of the relative importance of that factor. For example, the loading for CPK on day two appears to be of the inappropriate sign. A rise in CPK levels increases the predictor value and moves the case towards a prediction of 'Live'. However, previous analysis of CPK levels (Chamberlain 1986) has shown them to be higher in those animals that died. An example of an inappropriate magnitude for a loading is that attributed to CPK on day three. Previous analysis has shown that CPK is one of the factors most closely associated with the outcome on day three ($p=0.023$) (Chamberlain 1986) yet a hundredfold increase in CPK levels only alters the indicator value by 0.22 units, an increase that is unlikely to alter the predicted prognosis.

As the CVA derived loadings cannot be interpreted in such a manner and they are only one of several equally significant combinations possible, it would be desirable to have some other means of assessing how useful the indicator produced is as a clinical predictor, and to compare it with other alternative predictors.

When considering the values of a multifactorial indicator as a clinical prognostic predictor, three main aspects must be assessed:-

(1) The accuracy of the predictor.
(2) The robustness in terms of being able to accommodate both differing aetiologies and the expected data collection errors.
(3) The sensitivity of the predictor to respond to changes in factor levels likely to show a changed prognosis.

The accuracy of the predictor can be determined from a number line plot as described above. Although a number line plot also gives some indication of the predictor's robustness, this attribute and also the predictor's sensitivity are better investigated using the technique of sensitivity analysis.

Sensitivity analysis

Sensitivity analysis is a technique used in clinical decision analysis to test the stability of a decision analysis method to changes in various parameters (Weinstein & Fineberg 1980). In the context of a prognostic predictor the technique can be used to examine the robustness and sensitivity of the predictor and to pinpoint those factors that either degrade the robustness or that do not contribute to the sensitivity. Once indentified, attention can be paid to such factors to improve the overall predictor performance.

To assess the robustness of the predictor to data collection errors, the magnitude of the likely data collection error must first be estimated. With dichotomous variables the error would be a switch in recorded answer from 'Yes' to 'No' or vice versa. For continuous variables an error of $\pm$ 10% of the true value might be a realistic estimate. By looking at how the error in each factor affects the predictor it should be possible to get an indication of the robustness of the predictor to changes in each factor.

Changing a factor from a value typical of an animal in one outcome group to that of an animal in another group, whilst holding all other factors constant, allows the value of each factor, in terms of contributing to the responsiveness of the predictor, to be assessed. If such a change in a single factor causes the prediction to be reversed there is a risk of a prognostic error being made when considering cases with an aetiology in which this factor is not associated with the outcome. Conversely, if such a change in factor level has little effect on the predictor, the merit of including the factor in the predictor should be considered.

To construct a sensitivity analysis plot (Fig. 4) in a form that will allow comparison between predictors, the factor and predictor

values must be standardised. The factor values are standardised by e xpressing them in terms of standard deviations from the average value: standard normal deviates ('S.N.D.'). Changes in predictor value are expressed as percentages of the "Average" predictor value I_A - that is the predictor value obtained when all factors are set at the average for all the cases considered by the predictor.

To calculate the effect of variation in each factor, single factor values are altered whilst holding the other factors constant at their mean values. The new predictor value is plotted as a percentage of I_A against the change in factor value in terms of S.N.D. This produces a straight line plot for each factor considered (Fig. 4). The importance of a factor in the predictor is proportional to the gradient of the straight-line plot; the steeper the line the greater the influence.

To examine the effects of the expected data collection errors, as determined above, the expected errors are expressed in terms of S.N.D. and plotted on the appropriate straight-line plot. (Triangular symbols in Fig. 4). From Fig. 4 it can be seen that different types of variable affect the predictor to varying extents. Errors in recording logarithmically transformed variables have little effect on the predictor value, whereas linear discrete variables affect the predictor value slightly more and errors in recording dichotomous variables can alter the predictor value considerably and may even reverse the prediction made. Similarly, to examine the effect of a factor changing from that typical of one outcome group to that typical of another group (the predictor sensitivity), the average factor value for each outcome is expressed in terms of S.N.D. and plotted on the appropriate straight-line plot (square symbols on Fig. 4).

Predictor for Day 2 (Fig. 3): Most of the factors are little affected by recording errors but changes typical of a reversal in a case's prognosis produce an adequate change in predictor value. The one exception is the factor 'quality of nursing'. This factor has the steepest straight-line plot (gradient 15% I_A per S.N.D.). It follows that changes in this factor will have a considerable effect on the prediction. In addition, being a dichotomous variable, recording errors will also have a considerable effect; here reversing the prognosis. To reduce the effect of this factor it has since been divided into five sub-factors each of which is scored 1 to 5. If the behaviour of the 'quality of nursing' factor can be improved by such changes this predictor should be a useful and reliable clinical tool.

Predictor for Day 3 (Fig. 4): Of the three predictors considered this has the largest 'grey area' extending from 80% to 137% of I_A. Because of this, changes in individual factors do not in themselves reverse the prognosis. Three of the factors; log (Urea), 'attempting to rise' and magnesium have steep straight-line plot gradients. The 'attempting to rise' gradient is steepest (62% I_A per S.N.D.) and as it is a dichotomous variable recording errors have a considerable effect, changing the predictor from 33% to 155% I_A, a change that will invariably reverse the prognosis regardless of the values of the other factors. The assessment of whether a cow is attempting to rise or not will vary according to

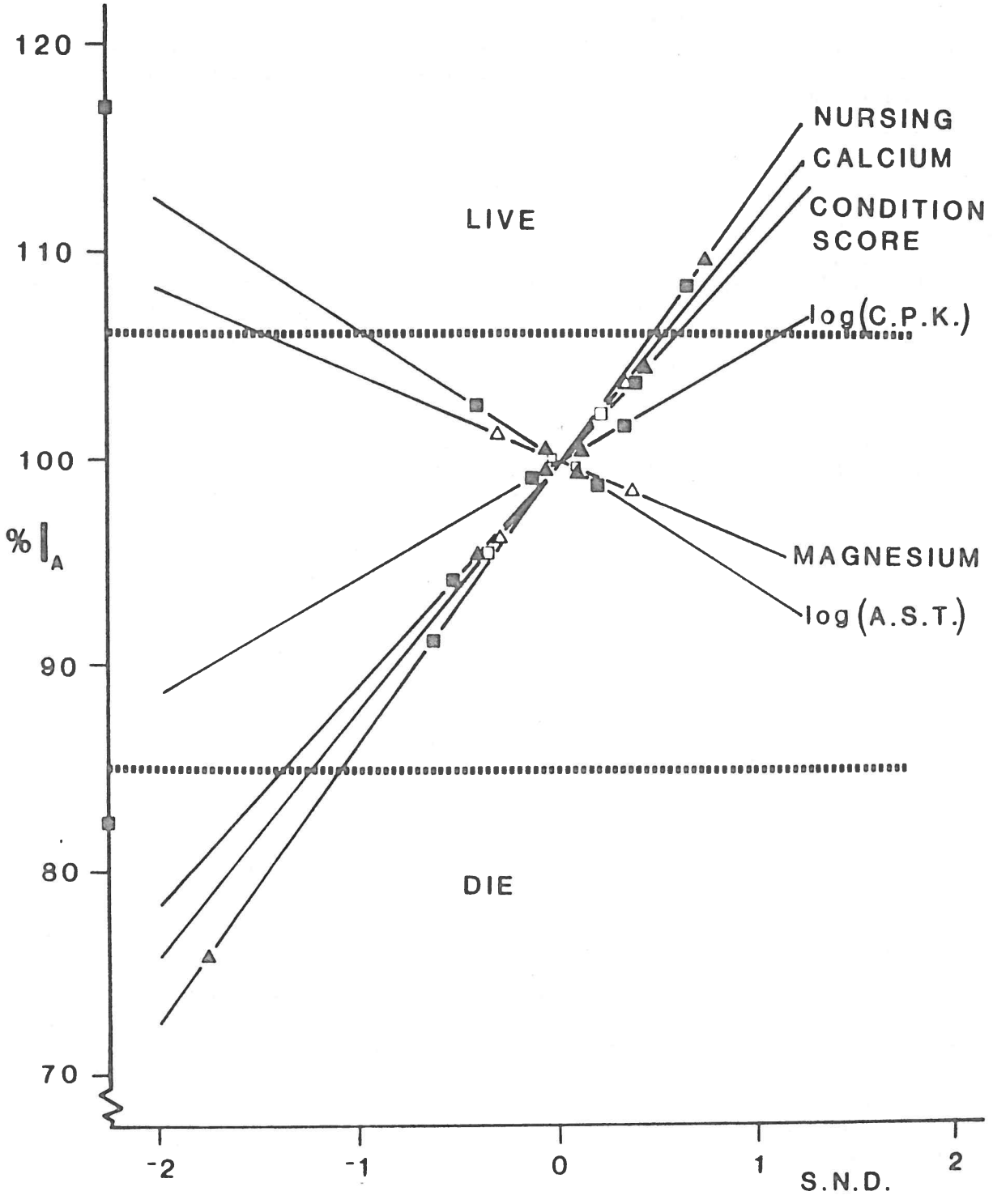

Fig. 3. Sensitivity analysis plot for day 2. Changes in individual factor values, expressed in standard normal deviates (S.N.D.) plotted against change in indicator value, expressed as percentage of indicator value obtained when all factors are at their mean value (I_A). ▲ - Effect of likely errors. ■ - Effect of changes in factors likely to be associated with a change in prognosis. n = 32.

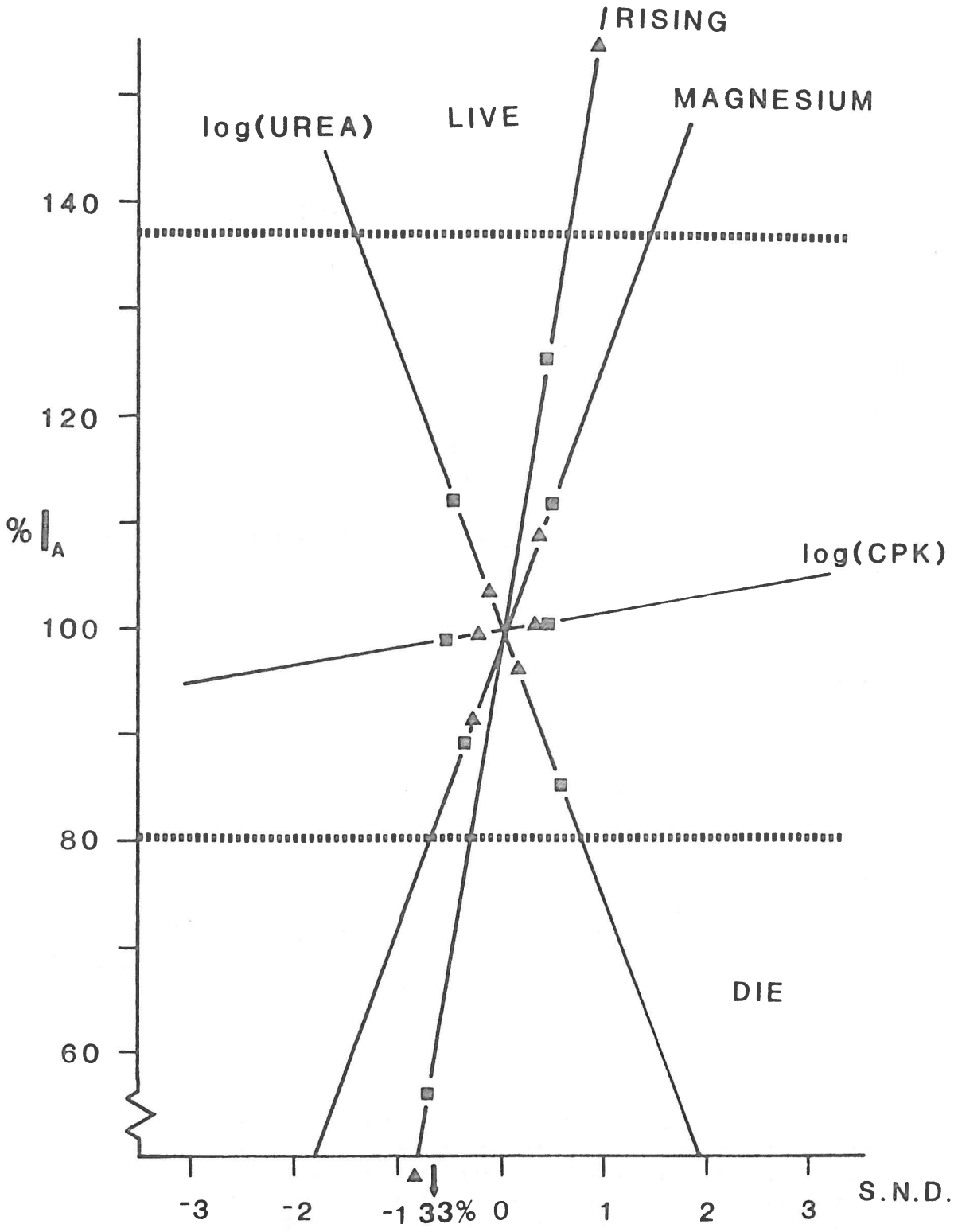

Fig. 4. Sensitivity analysis plot for day 3. Changes in individual factor values, expressed in standard normal deviates (S.N.D.) plotted against change in indicator value, expressed as percentage of indicator value obtained when all factors are at their mean value (I_A). ▲ - Effect of likely errors. ■ - Effect of changes in factors likely to be associated with a change in prognosis. n = 18.

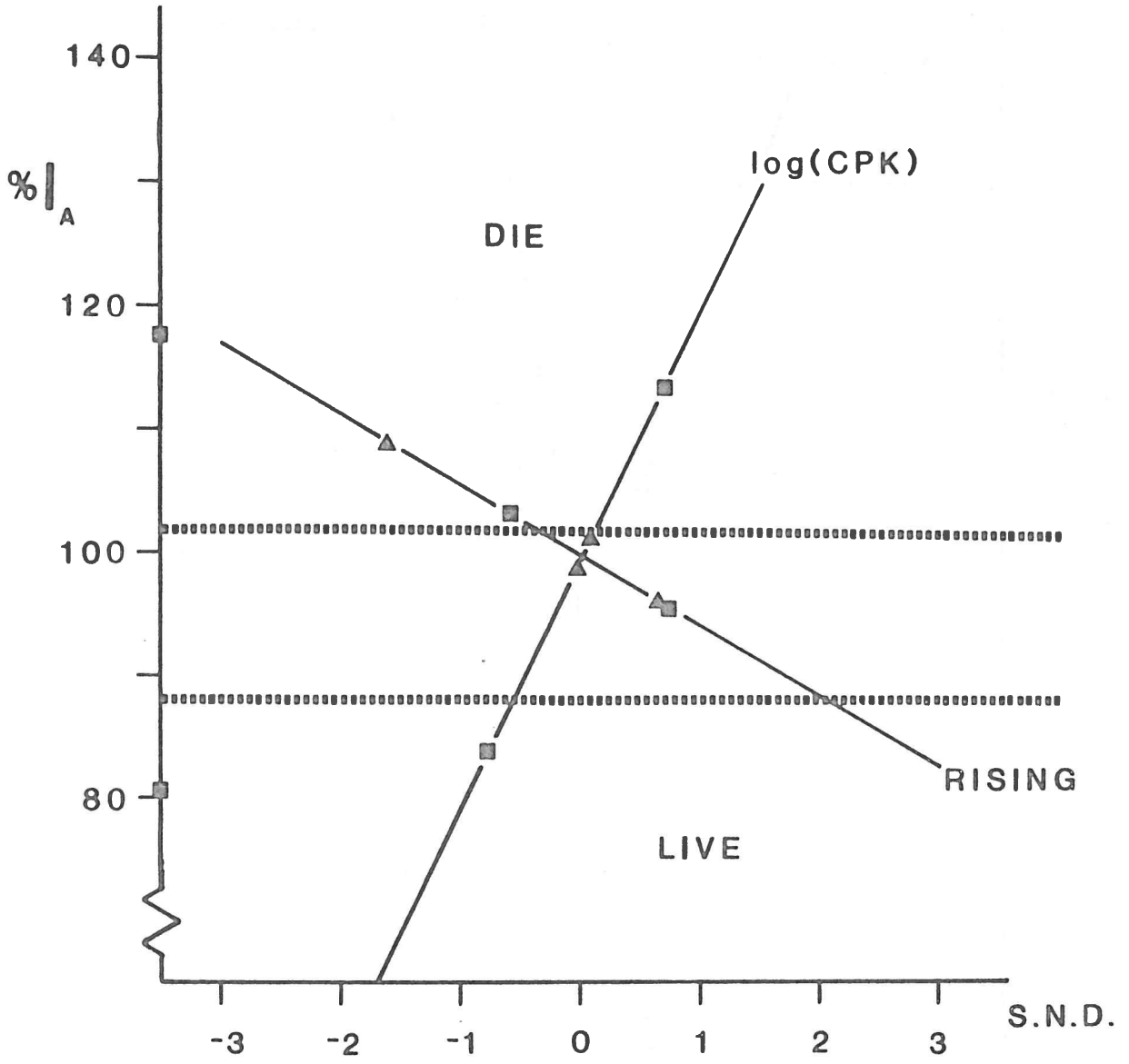

Fig. 5. Sensitivity analysis plot for day 4. Changes in individual factor value, expressed in standard normal deviates (S.N.D.) plotted against change in indicator value, expressed as percentage of indicator value obtained when all factors are at their mean value (I_A). ▲ - Effect of likely errors. ■ - Effect of changes in factor likely to be associated with a change in prognosis. n = 18.

how much the cow is stimulated when examined and hence recording errors are likely. For this reason the clinical usefulness of the predictor should be suspect unless the 'attempting to rise' factor can be altered to improve its stability.

The gradient of the straight-line plot for CPK is shallow (1.7% I_A per S.N.D.). Recording errors have little effect on the predictor but conversely changes likely to indicate a change in prognosis also have little effect. As such, although this factor does not degrade the predictors robustness it also adds little to its sensitivity and a new predictor omitting CPK should be investigated.

Predictor for Day 4 (Fig.5): The 'grey area' for the day four predictor is small, spanning from 88% to 102% of I_A. The majority of predictor variation is due to changes in the CPK factor (gradient 20% I_A per S.N.D.) with the 'attempting to rise' factor having a lesser effect (-5.5% per S.N.D.). Omission of the latter factor does not affect the predictor's accuracy but only reduces the 'grey area' size. Because of the dependence on the CPK factor this predictor may fail when considering cases with an aetiology where muscle damage is not important. Although all 18 cases considered here are correctly predicted by CPK alone until the aetiology of the downer cow syndrome is better understood predictors composed of only one factor should be avoided.

If, after consideration of a predictor's sensitivity and robustness, it is decided that the predictor is unsuitable, several further steps may be considered to improve it:-

(1) Adapt those factors that reduce the predictor's robustness to reduce their effect.

(a) Take logarithmic transformations wherever possible. This should be done whenever it makes the factor distribution more normal and it may justifiably be performed on all enzyme levels (Gaddum 1945).

(b) Reduce the effect of dichotomous variables by:-

i. Subdividing them. For instance, to reduce the effect of the nursing factor, it has been subdivided into five categories of 'Quality of bedding', 'Feeding', 'Turning', 'Muscle massage' and 'General tender loving care'.

ii. Increasing the reply options that can be made. For instance, the nursing subdivisions have been changed from a 'Yes/No' type answer to a score of one to five. However, systems that have too large a range of scores should be avoided as problems of subjective misclassification will start to affect the data.

(2) Factor Rotation

This consists of trying to find another combination of the factors that also give a significant separation of the outcome groupings. Computer algorithms exist for such investigations (Avery et al 1983)

but as the basic theory behind the algorithms is not fully understood, they should be used with extreme caution.

(3) Define new canonical axes

Methods exist for determining the centroids of the outcome clusters. If such centroids are joined to the origin, it might be expected that these connecting lines may form new axes that would give a better separation of the clusters.

(4) Limit the loading attributed to each factor

As the influence of a factor on the predictor is proportional to both the variance of the factor and the loading attributed to the factor, limitation of either could be expected to limit a factor's influence. Steps to control the factor variance are described above in (1). In theory limits could be put on the size of each factor weighting before an analysis was run, hopefully such controls would increase the predictor's robustness.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the contributions of the British Cattle Veterinary Association throughout the work: Mr D.A.K. Thornton, BCVA Clinical Research Officer, for instigating the research; for funding much of the work and to the individual members who submitted samples. We are also grateful to Mrs K.B. Bazeley and Dr. J.B. Day for analysing the samples during the first year, to Mr G. Talbot-Paine and Mr W. Millard for biochemical analyses, and Dr. S. Evans for advice on the statistical analyses

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SOME RELATIONSHIPS BETWEEN FERTILITY INDICIES IN DAIRY HERDS IN ENGLAND AND WALES 1984-5

A H POOLE* AND SUSAN J MABEY*

Checkmate is a herd fertility monitoring service based on information recorded on National Milk Records (NMR) operated by the Milk Marketing Board. The aims of Checkmate are:

To provide a regular review and analysis on a herd basis of overall and monthly fertility results.

To breakdown the calving interval into its components of interval from calving to first service, interservice intervals and service success.

To provide veterinary surgeons with information on which to base advice and treatments and the means of evaluating past treatments.

In the year ending May 1985 322 herds have subscribed to Checkmate. The whole sample has been analysed and also two sub-samples, the top and bottom 10 per cent, selected by the days from calving to assumed conception. As there is no pregnancy diagnosis information on NMR conception is assumed if no re-service date is recorded within 60 days of a service.

Table 1. Checkmate results 1984-5

	Whole sample	Top 10%	Bottom 10%
Number of herds	332	33	33
Herd size	136	129	111
Average interval:calving-assumed conception (days)	99	81	130
Average interval:calving-first service (days)	71	64	80
Services per assumed conception	1.82	1.65	2.22

The average interval between calving and assumed conception is 99 days, which with a 280 day pregnancy length, gives a calving interval of 379 days. The top group achieve a calving interval of 361 days some 49 days better than the bottom 10 per cent.

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The interval to first service mirrors this trend with the top group averaging 64 days, the whole sample 71 days and the bottom 10 per cent 80 days. The distribution of first service dates shows that the top group serve half the herd within 60 days and almost three quarters within 75 days compared to under a third in 60 days and just over half in 75 days for the bottom group.

Table 2. Distribution of 1st service dates (%)

Days from calving	Whole sample	Top 10%	Bottom 10%
1-45	13	17	9
46-60	26	33	20
61-75	25	23	25
76-90	18	16	18
91+	18	11	28

On average the whole sample took 1.82 services per assumed conception compared to 1.65 and 2.22 for the top and bottom 10 per cent sub-samples respectively.

This is largely a reflection of service success with the top group having over 60 per cent of each service being successful compared to under 50 per cent for the bottom group.

Obviously a cow will not hold to service if she is not in oestrus when served. The interval between services gives an indication of the effectiveness of heat detection.

Table 3. Distribution of interservice intervals (%)

Days	Whole sample	Top 10%	Bottom 10%
5-17	6	6	5
18-24	40	44	28
25-35	14	15	12
36-48	18	17	19
49-90	17	16	25
91+	5	2	11

Cows re-served in the 5-17 or 25-35 day periods probably indicates poor oestrus detection either at the first or repeat services. All groups have about a fifth of the repeats in these periods. Performance was unsatisfactory by this measure, which adds further weight to the opinion that poor heat detection is the largest single cause of poor fertility in herds.

As has been shown the main factors recorded by Checkmate which influence the interval from calving to assumed conception are the interval from calving to first service, the interval between services and service success. Regression analysis has been used to investigate any linear relationships between these factors for the herd.

Both the calving to first service interval and service success are directly related to the calving to assumed conception interval. However,

these two factors are naturally inversely related to each other. Herds with the shortest interval to first service needed more services per conception that those with longer intervals. The percentage of repeat services in the 18-24 day period was used as an indicator of the efficiency of oestrus detection. This was not significantly related to the other two factors. The culling rate was also considered but this was not related to any of the other fertility indices maybe as it was not possible to identify cows culled for fertility reasons.

Multiple linear regression showed the best fit between the factors as:

$$Z = 19.22 + 0.80Y + 19.92X - 0.33W$$

where Z = interval:calving to assumed conception (days)

Y = interval:calving to first service (days)

X = services per conception

W = returns to service within the 18-24 day period (%)

This gives a highly significant fit ($P<0.001$), multiple correlation coefficient $r = +0.77$. All regression coefficients were highly significant ($P<0.001$) their standard errors were 0.05 for Y, 1.23 for X and 0.04 for W.

Whilst Y and X are naturally inversely related, it is possible to improve both at the same time with the appropriate management of the herd.

Many factors affect the fertility of the herd. This analysis has only considered three, but they are the major factors of interval to first service, service success and oestrus detection and accounts for practically 60 per cent of the variation in the calving to assumed conception interval. They also have the advantage of being easily quantified and recorded by Checkmate.

POULTRY EPIDEMIOLOGY

A LONGITUDINAL SURVEY OF A CONTACT DERMATITIS IN BROILERS

S.G. McILROY*, E.A. GOODALL** AND C.H. McMURRAY***

Downgrading of broiler carcasses due to lesions of a contact dermatitis has been a major problem to commercial broiler production in Northern Ireland since 1978. The dermatitis was initially described by McFerran et al., (1983), while Greene et al., (1985) have described the clinical and pathological findings of the condition. These latter authors described the lesions as brown/black erosions and ulcers and note that the term 'burns' is commonly used by the local poultry industry when referring to the condition. The areas affected on broiler carcasses are the breast, hocks and feet, lesions on the former two sites resulting in downgrading of carcasses and/or necessitating further processing. Feet are removed during the normal processing of carcasses. It is suggested that the occurrence of the dermatitis is strongly associated with poor litter conditions.

Martland (1984) experimentally produced lesions on the breast, hocks and feet of turkeys when kept on wet litter. Martland (1985) also records the occurrence of ulcerative lesions on similar anatomical sites in broilers when subjected to wet litter conditions. The lesions on the broilers were considered to be identical to those reported from commercial units in Northern Ireland by Greene et al., (1985).

In view of the serious nature of this condition to profitable broiler production in Northern Ireland and the lack of both qualitative and quantitative information from commercial units, a longitudinal survey was initiated in December 1983 by the Veterinary Research Laboratories in co-operation with a major poultry organisation. This on-going survey covers the major aspects involved in commercial broiler production in an attempt to identify the important epidemiological factors involved in the occurrence of the condition.

MATERIALS AND METHODS

A special questionnaire was designed to cover the major aspects of commercial broiler production in all individual flocks utilized by the poultry organisation. Flock information was based on results from individual houses. In total, 31 production aspects were covered but only those pertinent to this paper are recorded here.

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1. Producer name, address, organisation code and individual house number.
2. Producer rating on a 1-5 scale (1 equalling the highest producer score) based on a subjective assessment of general husbandry ability.
3. Sex of birds categorized into cocks, pullets, as hatched and mixed sexes.
4. Dates house started and finally cleared.
5. Total number of birds placed in the house.
6. Average age (days) at removal. This was calculated from the percentage of the flock removed on successive occasions when more than one removal took place.
7. Stocking density calculated on the number of square feet per bird.
8. Meal manufacturer.
9. Type of litter used (straw, shavings and both).
10. Presence or absence of wet and/or sticky litter.
11. Grading information: percentage of carcasses with breast and hock lesions and also the number of grade A, grade B and reject birds. Both target and actual values.
12. Total flock mortality. Both target and actual values.
13. Average food conversion ratio for the entire flock. Both target and actual values.
14. Average weight per bird for the entire flock. Both target and actual values.
15. Average net income per 1000 birds. Both target and actual values.

Commercial targets were included for the latter five production aspects. These targets take into consideration other production variables which would be expected to have an effect on the achievable result. The difference between the target value and the actual value was used to analyse the independent effect which each of these production aspects had on the occurrence of the condition.

In addition extra information was obtained on the occurrence of incidences when litter deteriorated very rapidly (frequently overnight). In particular the time during production when such acute outbreaks of litter deterioration occurred, was recorded. Flocks with such incidences had been associated by the poultry organization with increased levels of lesions.

The questionnaire forms were completed by the accounting division and advisory staff of the poultry organization and submitted to the Veterinary Research Laboratories for validation on a monthly basis. The forms were subsequently logged onto a VAX 11-750 super mini computer and the data analysed using the software package DATATRIEVE, tailored to the special requirements of the survey. The set of data analysed here covers completely the period January 1984 to December 1985.

The data from flocks was grouped into 3 main categories, all flocks, flocks which experienced an acute outbreak of litter deterioration and flocks which did not. Data was analysed using a wide range of intervals of small strata size to identify trends and highlight all observations of possible interest. Subsequent analysis involved the pooling of strata into gradations meaningful to the industry. These latter strata were used in contingency tables and analysed statistically using the chi-squared test. Dependency relationships were investigated using standard linear regression techniques. Data on all relevent weather variables recorded on a daily basis at Aldergrove and computerized at the Meterological Office in Belfast was also

obtained and these files merged with the database constructed from the questionnaires.

The results presented here cover a small fraction of the total data collected and represent a summary of the main effects observed from the initial analysis.

RESULTS

Data from 986 flocks, containing 12,600,000 birds, was available for analysis. In all flocks, the average incidence of hock lesions was 21% while breast lesions occurred on 0.3% of carcasses. The average incidence of breast and hock lesions was much higher in flocks which recorded an acute outbreak of rapid litter deterioration. Fifteen percent of all flocks experienced such an acute outbreak and had approximately twice the level of hock lesions and ten times the level of breast lesions than flocks which did not. The incidence of breast lesions, although at a reduced level, was strongly correlated with the occurrence of hock lesions, a correlation coefficient of 0.92 ($p<0.001$) being obtained.

Predictably, wet and/or sticky litter was recorded in all instances when an acute outbreak had occurred. However, 31% of flocks in which no such outbreak took place, also recorded the presence of wet and/or sticky litter. These latter flocks had almost twice the level of hock and six times the level of breast lesions than flocks which did not record poor litter.

Sex: In general, male flocks had a higher incidence of the condition. Hock lesions were increased by one third while breast lesions were three times greater than female flocks. Fifteen percent of male flocks experienced an acute outbreak compared with 7% of female flocks. This difference was highly significant ($p<0.01$). The level of hock lesions recorded in both male and female flocks with acute outbreaks was similar. However, breast lesions were twice as frequent in such male flocks.

Litter: Twenty six percent of flocks used straw for litter while 73% used shavings. The remaining 1% recorded using a mixture of straw and shavings. Throughout the 2 year period under analysis there was little evidence of any difference in the occurrence of the condition when either straw or shavings had been used. Further, acute outbreaks occurred with equal frequency on litter of both straw and shavings. However, when an acute outbreak was recorded on straw, the average incidence of hock lesions was slightly greater than that recorded on shavings. Breast lesions were the same on both.

Meal manufacture: Ninety eight percent of flocks used 3 meal manufacturers, who each supplied approximately the same number of flocks. A lower incidence of hock and breast lesions was evident in flocks supplied by one meal manufacturer. This was principally due to a significantly ($p<0.01$) lower number of acute outbreaks occurring in flocks supplied by this manufacturer. Further, when acute outbreaks did occur, the resulting average incidence of the condition for both hock and breast lesions was less than that experienced when other meals were fed.

Stocking density: When the stocking density was increased, the incidence of both hock and breast lesions also increased. Subsequent analysis involved dividing flocks into high (0.48 square feet/bird or greater) and low

categories (0.49 square feet/bird or less). Forty six percent of flocks were stocked at the higher stocking density and recorded an increased incidence of both hock and breast lesions. Hock lesions were increased by 10% and breast lesions by 20% compared to the lower stocking density. Flocks at the higher density were significantly more likely to have an acute outbreak ($p<0.01$), although the outcome of such an event appeared to be little affected by the initial stocking density level.

Age of removal: In general, as the average age of removal increased, the level of both hock and breast lesions also increased. For further analysis flocks were divided into two categories, 47 days or more and less than 47 days at removal. Certainly, flocks which were kept longer had approximately one third more hock lesions while the level of breast lesions trebled. When acute outbreaks occurred and flocks were not removed, on average, before 47 days, hock and breast lesions again increased by similar amounts.

Food conversion ratio: Flocks which had a worse food conversion ratio than expected (i.e. greater than target value) had a higher level of both hock and breast lesions. In such flocks hock lesions increased by one-quarter while the incidence of breast lesions trebled, compared to flocks which attained their target food conversion ratios. No significant difference was found in flocks which failed to reach targets, between those which had an acute outbreak and those which did not.

Average weight per bird: Flocks which did not reach their target weight had an increased level of hock and breast lesions. Hock lesions were increased by one third in such flocks, while breast lesions quadrupled. Forty two per cent of flocks which experienced an acute outbreak did not reach their target weights and this was significantly more ($p<0.001$) than in flocks which attained or exceeded their target values.

Income per 1000 birds: Flocks which did not reach their target income per 1000 birds had a higher incidence of both hock and breast lesions. Hock lesions increased by 15% while breast lesions trebled. Sixteen percent of flocks which had an acute outbreak did not reach their target income and this was significantly more ($p<0.05$) than flocks with no such outbreak.

Season: In all flocks the incidence of lesions was greatest during the winter months (October to April inclusive). Fig. 1 shows the seasonal deviation from the overall mean level of hock lesions. A similar seasonal pattern was observed for breast lesions. Eighty three percent of acute outbreaks of litter deterioration occurred in the winter months which was significantly more ($p<0.001$) than in the summer months and greatly contributed to the overall seasonality of the condition (Fig. 2).

Weather: Multiple regression analysis of the average monthly incidence of hock lesions against the major weather factors was performed. The weather variable most strongly correlated with the average incidence of hock lesions (%) was relative humidity (%) where a correlation coefficient of 0.77 ($p<0.001$) was obtained. Assuming a dependence relationship between relative humidity (x) and hock lesions (y), a regression analysis was performed. The equation obtained was $y = -61.1 + 1.01x$. The standard error of the regression coefficient was 0.19. The relationship is demonstrated in Fig. 3. None of the other variables added significantly to the explanation of variation in the data.

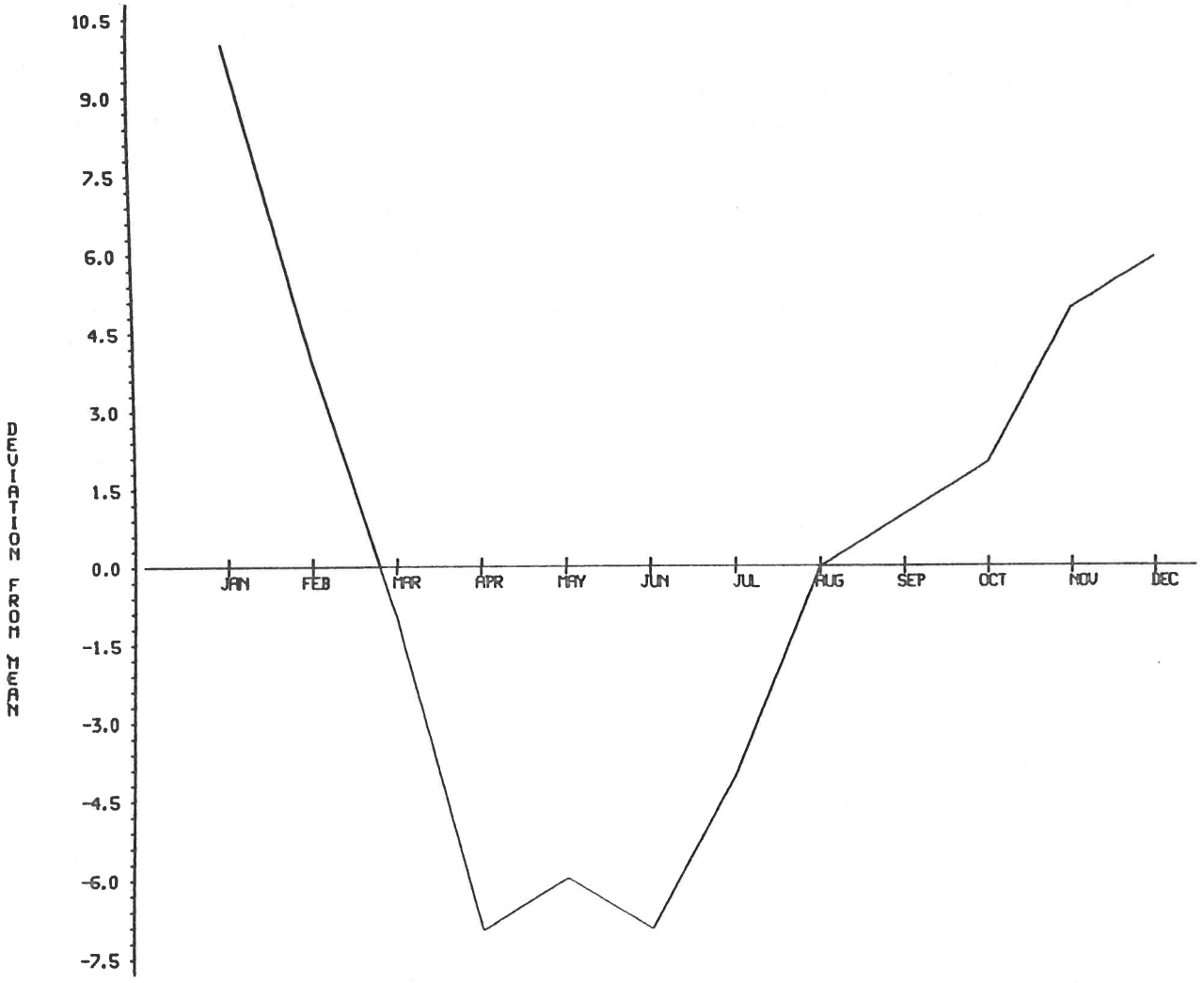

Figure 1. Seasonality pattern of hock burn.

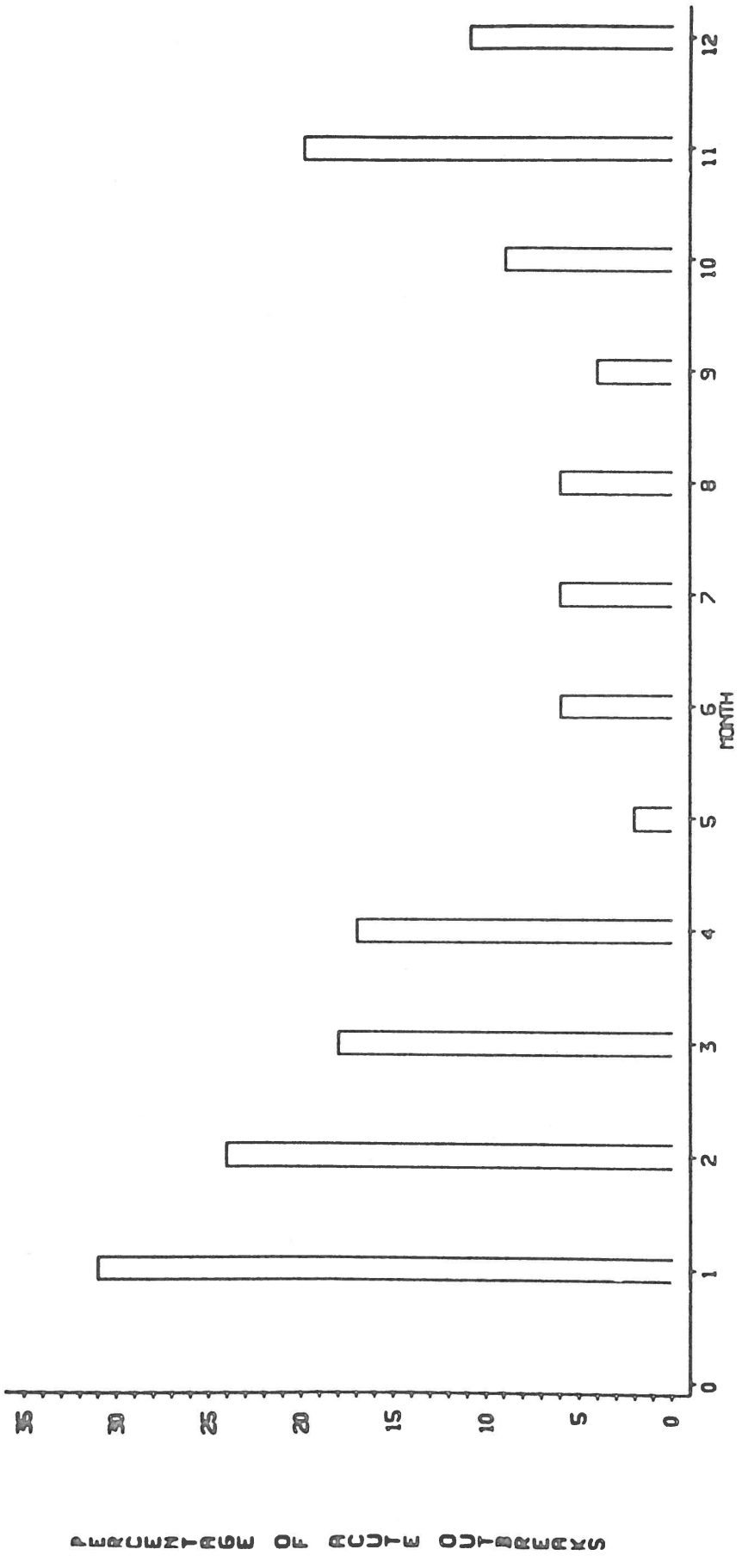

Figure 2. Acute outbreaks as a percentage of all flocks for each month.

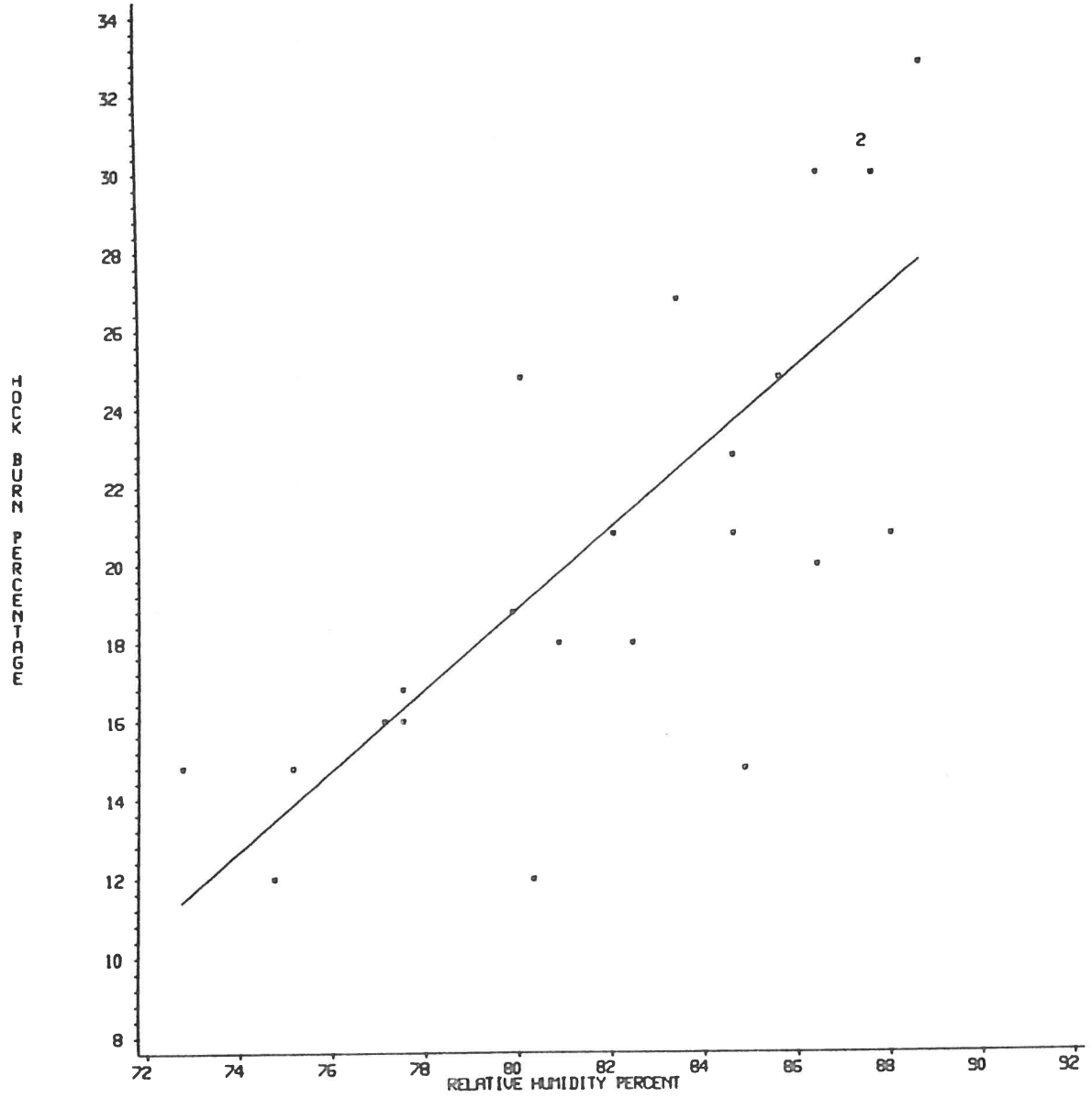

Figure 3. Relationship between relative humidity and hock burn.

DISCUSSION

Analysis of the data from such a large number of flocks, with a corresponding large number of birds, has enabled efficient quantitative estimates of the major factors associated with the condition. The average incidence of hock lesions over the 2 years was 21% and indicates that approximately 2.6 million birds were affected. Although the corresponding level of breast lesions was only 0.3%, this resulted in approximately 40,000 birds being affected. The latter condition is considered more serious as it invariably results in downgrading and frequently involves the loss of the breast area, even with further processing. Hock lesions may also result in downgrading or at least necessitate further processing, at considerable cost to the industry. When either breast or hock lesions occur at an unacceptably high level in a flock, serious management problems arise with regard to satisfying daily market demands for fresh whole poultry carcasses. Fresh, as opposed to frozen, is the major market for all broiler organisations in Northern Ireland and the levels of lesions recorded in this survey have serious implications in this highly competitive market area for the appearance and acceptability of poultry meat. Acute outbreaks of litter deterioration in flocks are associated with vastly increased levels of lesions and thus represent the greatest problem to the industry when presented for slaughter.

The survey results demonstrate unequivocally the clear relationship between the occurrence of lesions and litter conditions on commercial units. This is not only observed when acute outbreaks of litter deterioration occur, but also in flocks where litter conditions deteriorate over the production period. Wet, sticky litter has been observed adhering to the breast and hock areas of birds from flocks where litter deterioration had occurred and contact dermatitis lesions subsequently found on these sites (McIlroy. Personal communication). This may be an important mechanism in the pathogenesis of the condition. Certainly, the survey would indicate that the occurrence of lesions in flocks with seemingly normal litter is comparatively infrequent and of little or no concern to commercial broiler production.

The results of this survey also highlight the increased susceptibility of male birds to the condition. Martland (1985) observed no statistically significant difference between male and female birds kept on wet litter on a small experimental trial. However, field observations in Northern Ireland have suggested that male birds had a higher incidence of lesions (Greene et al., 1985) but this has not previously been quantified. The increased level of lesions may be due to the increased liveweight of male birds, leading possibly to a greater tendency towards adopting a sitting position and thus exposing hock and breast areas to more frequent, intimate litter contact. The inherent poor feathering of the breast in male birds may further increase the susceptibility of this area to contact with poor litter. It is apparent from our results that a serious downgrading due to breast lesions occurs most frequently in male flocks.

The use of either straw or shavings has no apparent effect on the occurrence of the condition and also on the occurrence of the acute outbreaks. The small increase in hock lesions seen in acute outbreaks when straw was used may be due to a reduced capability when exposed to extreme adverse conditions such as high moisture content. Otherwise, straw appears to be totally adequate for normal production.

The lower incidence of hock and breast lesions evident in the flocks supplied by one of the meal manufacturers is a new and interesting observation. The reason for this is unclear but obviously merits further investigation.

The use of a high stocking density has an adverse effect on the occurrence of lesions. This may be due to a failure of management to maintain adequate litter conditions when stocking levels are high, especially with regard to the occurrence of acute outbreaks. The commercial advantages obtained by high stocking density must be weighed against an increased occurrence of this important condition and subsequent economic loss.

Increasing the length of time birds are kept in a house leads to an increased incidence of both hock and breast lesions. This is probably due to the increased length of exposure to litter and is of special concern when litter conditions have deteriorated. This was especially noticeable with breast lesions, which frequently only occurred in flocks where the average age of removal was 47 days or more and may indicate that breast lesions occur after hock lesions. Obviously, the average weight per bird will also increase when birds are kept longer and may further increase the possible occurrence of lesions on contact areas. The survey results clearly suggest that when poor litter conditions are observed during production, all birds should be removed as soon as possible.

When the incidence of hock and breast lesions increases in flocks, food conversion ratio, average weight per bird and income deteriorate. This was especially true in flocks with acute outbreaks for the latter two commercially important factors. The effect of poor litter on general commercial broiler production would appear not to be wholly restricted to the occurrence of contact dermatitis lesions. Inherent husbandry practices which allow the deterioration of litter conditions, also have a marked effect on the major indices of profitable broiler production.

The seasonal occurrence of this condition is extremely interesting and the subsequent significantly high correlation found with relative humidity of special concern to Northern Ireland, a geographical area with high humidity especially in the winter months. Notably, the vast majority (83%) of acute outbreaks of litter deterioration occurred during the winter period. The maintenance of adequate ventilation, temperature and humidity in controlled environment poultry houses requires very careful monitoring especially when cold, damp climatic conditions prevail outside. Adequate ventilation is often wrongly constrained by the desire to conserve heat and frequently leads to a humid atmosphere with associated wet litter conditions, especially in peripheral areas remote from a heat source. This strong correlation found with monthly relative humidity warrants further investigation as there is some indication from the survey that individual producers and houses consistently recorded a low incidence of lesions and were free from acute outbreaks of litter deterioration, over the entire 2-year period. Identifying differences between these producers and those with a high incidence of lesions, including lesions associated with acute outbreaks, may be beneficial in determining cost effective methods by which satisfactory litter conditions can be maintained.

SUMMARY AND CONCLUSIONS

The survey results confirm the association between poor litter conditions on commercial units and the occurrence of contact dermatitis lesions. Further, as the survey contained data from approximately 1000 flocks and 12.6 million birds, any quantifiable differences in the incidence of the condition, however small, associated with specific production parameters are meaningful and important to the poultry industry. Sex, high stocking density, increased average age of removal and particular meal manufacturers have profound effects on the overall incidence of the condition and the occurrence of acute outbreaks of litter deterioration. This has prompted serious consideration by management. The associated reduction in the major indices of profitability recorded in flocks with increased levels of contact dermatitis lesions, has further reinforced the detrimental effects of poor litter conditions on commercial broiler production.

Reports are frequently sent to the poultry organization indicating current levels and trends in the incidence of the condition and also on many commercial production parameters not included in this paper. This ongoing survey has proved, and is continuing to prove, of great benefit to profitable commercial broiler production in Northern Ireland.

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ACKNOWLEDGEMENTS

The authors wish to thank John Rainey and Kevin McGlinchey for assistance with data processing. Also, Duncan Stewart and Gerry Partington for provision of meteorological data.

MORTALITY AND DISEASE PATTERNS OBSERVED IN NINE REPLACEMENT CHICKEN FLOCKS FROM 0-70 DAYS OF AGE

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Mortality that occurs in chicken flocks is known to vary considerably and although some surveys of mortality in adult chickens (Randall et al., 1977; Curtis et al., 1984) and broilers (Hemsley, 1965; Jackson et al., 1972; Reece & Bedmore, 1983) have been published, little has been published on losses during the early rearing period in replacement layers and breeders apart from reports from diagnostic laboratories concerned with specific disease problems and the records of poultry companies. It was therefore decided to examine all dead chicks on a rearing site during a winter and a summer rearing period to evaluate some of the recurrent problems that may occur and their interactions.

MATERIALS AND METHODS

The site was moderately sheltered and contained four fibre glass insulated deep litter houses with roof extraction fans thermostatically controlled, brooding heat being supplied by gas hover brooders.

Details of flock numbers and stocking density are shown in Tables 1 and 2. The parent breeder flock was a commercial brown egg strain and occupied house C in both winter and summer. House B held two flocks in winter but only one in summer. The flocks arrived on the site within 6 days of each other in winter (January) and 12 days in summer (July). Marek's disease vaccine was administered at the hatchery and infectious bronchitis vaccine (H120) at approximately 3 wks of age. Newcastle vaccine (HBI) was also given to the summer flock in water at approximately 3 wks and 7 wks of age. All chicks were beak trimmed.

An experienced and dedicated farm manager was responsible for the husbandry of the chicks.

An anti-coccidial, Pancoxin premix (Merck, Sharp and Dohme) was incorporated in the feed at a concentration of 400 grams per tonne. Carcasses were collected from the site each weekday, weighed and subjected to routine autopsy, the diagnosis recorded being that which was considered to be the most important condition contributing to death.

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Details were recorded using an Apple II Computer (48K) with a programme provided by Farm Plan Computer Systems Ltd. A limited amount of bacteriological, virological, parasitological and histopathological investigation was undertaken.

RESULTS

The mortality figures are summarised in Table 1 and figures for the more noteworthy disease conditions are given in Table 3 (major disease groups), Table 4 (oomphalitis and umbilical infection), Table 5 (respiratory disease group), Table 6 (starve-outs, runting syndrome and runts 0-21 days), Table 7 (coccidiosis).

Table 1. Mortality from 0-70 days of age

House Flock	Winter				Summer				Total		
	Chicks	Type	Deaths No.	Deaths %	Chicks		Deaths No.	Deaths %	Chicks	Deaths No.	Deaths %
A	5567	RP	207	3.72	6482	RP	189	2.91	12049	396	3.27
B 1	4352	RP	91	2.09	8333	RP	84	1.00	19323	230	1.19
2	6638	RP	55	0.83							
C	6787	BS	149	2.19	6842	BS	41	0.6	13629	190	1.39
D	7064	RP	83	1.17	8338	RP	78	0.93	15402	161	1.04
	30408		585	1.92	29995		392	1.3	60403	977	1.61

RP = replacement pullets
BS = breeding stock

Table 2. Stocking density and mortality 0-70 days of age

		Winter			Summer		
House	Area m^2	Chick/m^2	m^2/chick	Mortality %	Chick/m^2	m^2/chick	Mortality %
A	475	11.72	0.085	3.72	13.65	0.073	2.91
B	685	16.04	0.062	1.32	12.16	0.082	1.00
C	582	11.6	0.085	2.19	11.76	0.085	0.6
D	586	12.05	0.082	1.17	14.23	0.070	0.93

Table 3. Major groups of diseases causing mortality

	Winter		Summer			
	No.	%	No.	%	Total	
A. Oomphalitis/umbilical infection	86	0.28	59	0.2	145	0.24
B. Lung congestion, pericarditis, perihepatitis, colibacillosis	117	0.38	38	0.12	154	0.25
C. Nervous disease (3-8 days)	-	-	119	0.39	119	0.2
D. Starve-out, runting syndrome runts (0-21 days)	63	0.2	35	0.11	98	0.16
E. Coccidiosis, necrotic enteritis, peritonitis, torsion of bowel, dehydration (from 15 days)	123	0.4	34	0.11	157	0.26
Total	389	1.27	285	0.95	674	1.1
Other conditions	195	0.64	108	0.36	303	0.5
Total	584	1.91	393	1.31	977	1.61

Table 4. Oomphalitis and umbilical infection deaths

	A		B_1		B_2		C		D			
	No.	%	No.	%	No.	%	No.	%	No.	%	Total	%
Winter	29	0.52	17	0.39	8	0.12	22	0.32	10	0.14	86	0.28
Summer	18	0.28	16	0.19			9	0.13	16	0.19	59	0.19
Totals	47	0.4	41	0.21	(Total B)		31	0.22	26	0.17	145	0.24

Table 5. Lung congestion, pericarditis, peri-hepatitis "coli-bacillosis"

	A		B_1		B_2		C		D			
	No.	%	No.	%	No.	%	No.	%	No.	%	Total	%
Winter	83	1.5	20	0.45	3	-	6	.08	5	0.07	117	0.38
Summer	9	.03	7	0.02			9	0.13	12	0.14	37	0.12
Total	92	0.76	30	1.55			15	0.1	17	0.1	154	0.25

Table 6. Starve-outs, runting syndrome and runt, deaths, 0-21 days

1. Winter

Flock	A	B_1	B_2	C	D	Total	%
Starve-out	3	1	1	27	2	34	0.1
Runting syndrome	6	3	1	10	2	22	0.07
Runt	0	1	0	6	0	7	
Sub-total	9	5	2	43	4	63	0.2

2. Summer

	A	B	C	D	Total	%
Starve-out	1	0	3	5	9	0.03
Runting syndrome	9	3	3	7	22	0.07
Runt	2	0	1	1	4	
Sub-total	12	3	7	13	35	0.12
Total	21	10	50	17	98	0.16

Table 7. Coccidiosis death

(1) Winter

	A		B_1		B_2		C		D		Total	
	No	AR	No	AR	No	AR	No	AR	No	AR	No	%
Caecal coccidiosis only	20	26-31	10	32-35	11	23-42	1	36	16	25-43	58	0.19
Other coccidiosis	4	27-42	1	15	1	60	4	28-30	7	25-29	17	0.05
Total	24		11		12		5		23		75	0.24
%	0.43		0.13		0.18		0.06		0.32			

(2) Summer

	A		B		C		D		Total	
Caecal coccidiosis only	6	30-43	7	30-39	NA		2	36	4	41-47	19	0.06
Other coccidiosis	0		4	39-47	NA		2	44	4	42-45	10	0.03
Total	6		11		NA		4		8		29	0.1
%	0.09		0.13				0.05		0.01			

AR = age range in days

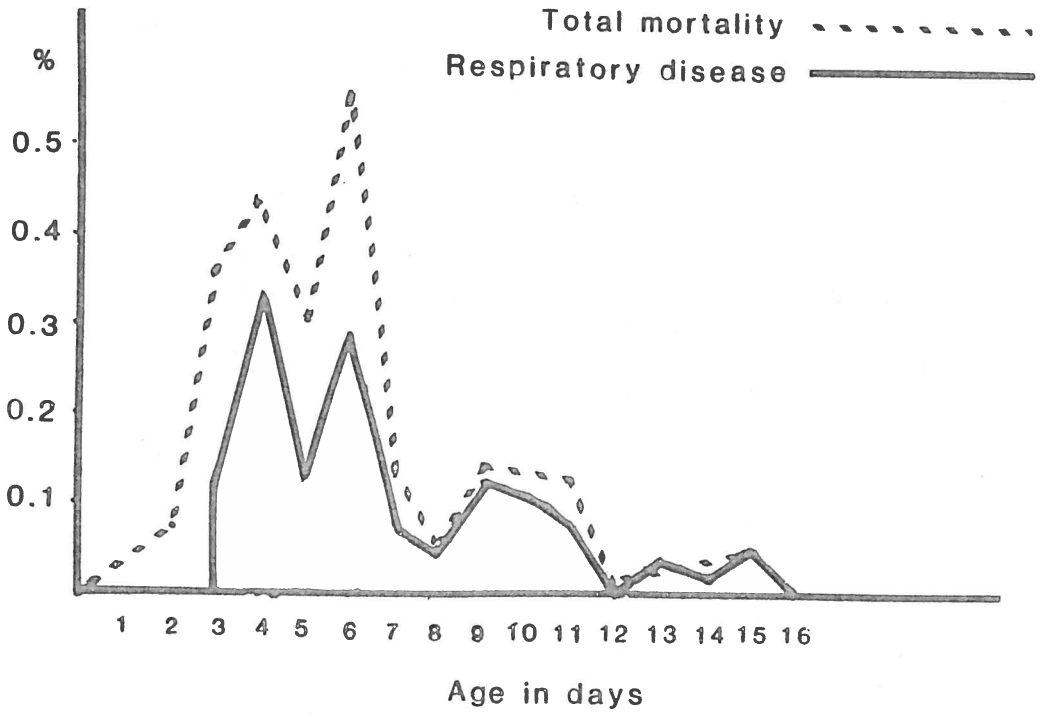

Fig. 1. Early mortality in Flock A (winter)

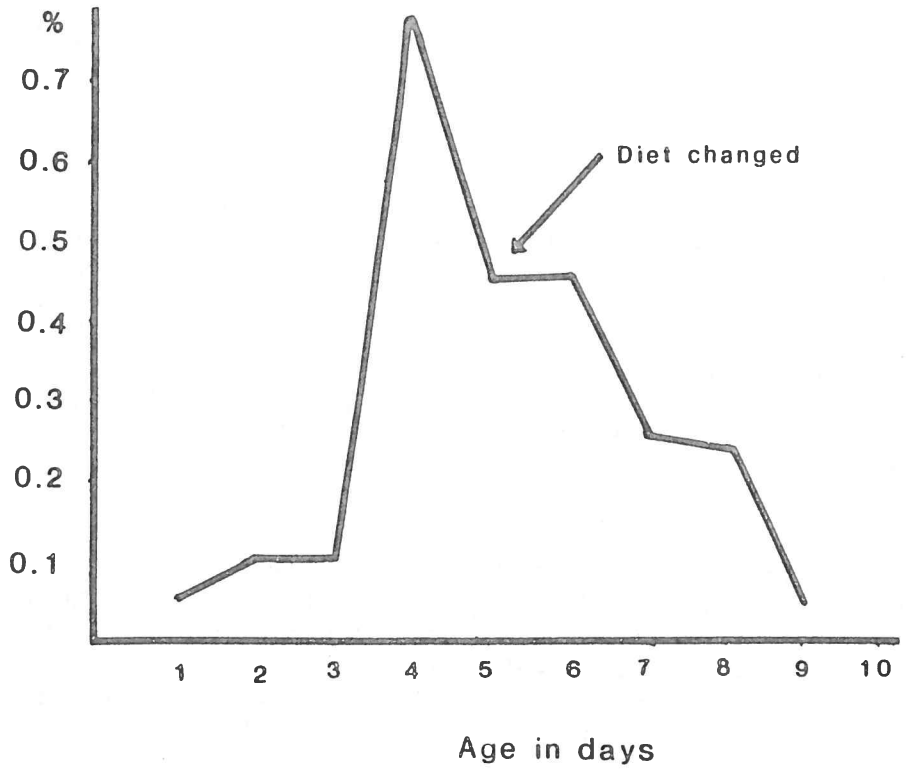

Fig. 2. Early mortality in Flock A (summer)

Limited bacteriological examination of a small number of typical oomphalitis cases was undertaken and environmental bacteria were detected including E. coli, staphylococci and Cl. histolyticum.

A potentially serious respiratory condition which affected flock A (winter) - see Table 5, was attributed from its first appearance to inadequate air intake in a very well insulated house. The condition began as acute lung congestion in 1-2 day old chicks and progressed to cases of pericarditis, perihepatitis and coli bacillosis. See Figure 1 Improvement of ventilation checked the problem and no other treatment was applied. Bacteriological examination indicated E. coli infection but it was not considered of primary importance.

The nervous disease (Table 3) Fig.2 affected only flock A in summer and was noticed in 4 day old chicks - symptoms of ataxia, opisthotonos and paddling movements of the legs were observed leading to death. The flock was fed from a bin shared with flock B which was 6 days older and unaffected. The food was replaced after 48 hours and the nervous disease was not observed after 8 days of age by which time 119 chicks (1.83%) of the flock were considered to have died from the condition. Attempts to isolate virus from the dead chicks by egg inoculatiomn or to transmit the condition to susceptible specific pathogen free chicks gave negative results. Histopathological examination of pancreas, proventriculus and central nervous system showed no evidence of epidemic tremor, but encephalomalacia was diagnosed in several specimens. Analysis of the food by a private analyst indicated an adequate amount of Vit. E to be present (73.4 mgm/kilo).

The occurrence of caecal coccidiosis in certain winter flocks led to a decision to medicate and Microquinox (sulphaquinoxaline 30 mgm/ml and pyremethamine 9 mgm/ml) was given in water for two three day periods three days apart from the following ages: A 23 days, B 20 days, B 24 days, C 28 days, D 26 days. No treatment was given to the summer flocks.

The other conditions totalling 303 chickens (0.5%) referred to in Table 3 comprised the following in the 0-21 day period (winter cases preceding summer): visceral gout 17, 11; deformed hocks 7, 2; cloacal obstruction 10, 9; accidental injury 11, 11; no diagnosis or decomposed 29, 3; bowel torsion, intussusception, obstruction (total) 7, 7; nephritis 4, 9; prolapse 4, 0; ascites 3, 0; necrotic hepatitis, peritonitis, air saculitis, anaemia, dehydration (each) 1, 0; crop impaction, enlarged heart, teratological defect (each) 0, 1; haemorrhagic lungs 8, 1; enteritis 4, 0, totalling 109/56.

Other conditions in the period 22-70 days were as follows - accidental injury 14,6; suffocation 15, 4; runts 7, 11; nephritis 3, 10; enlarged heart 4, 0; cannabilism 4, 1; deformed hocks 4, 0; no diagnosis or decomposed 8, 9; emaciation 9, 4; oomphalitis 3, 0; dermatitis 3, 0; ascites 2, 0; air sacculitis 2, 0; crop distension 2, 1; haemorrhage 2, 1; intestinal obstruction 2, 0; enteritis 1, 0; beak deformity 1, 0; urolithiasis 0, 3; leg fracture 0, 1; necrotic hepatitis 0, 1, totalling 86/52.

Additional details of diagnoses recorded in Table 3, Group E, as being associated with coccidiosis are: necrotic enteritis 5, 0; peritonitis 15, 2; torsion of bowel 8, 0; dehydration 20, 3.

DISCUSSION

This investigation indicates the complexity of the diseases which may affect a well managed poultry rearing operation and might be taken to justify the use of 'epidemiological diagnosis' (Schwabe 1984) as an essential adjunct to laboratory and clinical diagnosis. The fact that overall losses were kept to a low level (1.61%) is a tribute to the skill of the farm staff in managing the equipment and the livestock. Inevitably human error played a part in the causation of disease as in the respiratory disease syndrome in flock A (winter), and human error at the hatchery may have contributed to the amount of oomphalitis (through inadequate chick selection) and to the occurrence of smothering, accidental injury and other conditions. The cause of the encepholmalacia-associated nervous disease syndrome was not established but a human error factor could well be involved. Why only one flock was affected remains a mystery for flock B shared the same food.

The occurrence of caecal coccidiosis in all the flocks is of epidemiological interest and the fact that the condition tends to occur within certain time limits (see Table 7) may suggest that possibly continuous medication could be replaced by strategic therapy if and when the disease occurs. The possibility of drug resistance on the site has to be considered in view of the recurrence of caecal coccidiosis despite in-feed medication and (in winter) therapy. Although the losses from coccidiosis alone were not alarming, the secondary effects (Table 3E) must not be overlooked. Why particular individual chickens in the flock die with coccidiosis is not clear and it was noted that strong chicks were often those first affected. Defective husbandry did not appear to play any part in the onset of coccidiosis which would appear to be a site associated problem.

In Table 6 an indication of losses from starve-outs, acute runting syndrome and runt deaths (0-21 days) is given since it is considered necessary to be aware of these conditions even when they occur at a very low level in a flock for, on occasion, either of the three conditions, and particularly acute runting, can be responsible for spectacular losses. It is considered desirable to explain to stockmen why diseases occur as well as why they do not occur since such useful information encourages them to prevent the preventable and thus contribute to the welfare of the poultry and the industry.

Thirty-one % of the losses were due to a variety of 'other conditions' which are listed and include a great diversity of disease conditions any one of which may in another flock emerge as a much more significant problem for the farmer and any one of which, if it occurred in another domestic species, might attract more veterinary attention.

Mortality of chicks is a threat to the poultry industry if it rises to such levels that forward planning is seriously impeded and it represents an economic loss to the farmer. The welfare aspects of chick mortality have not attracted much attention, but it is clear that chicks affected with the major categories of disease (Table 3), especially where the condition is of the more prolonged and chronic type, will suffer from inability to take water and feed. Chicks that survive an acute condition and live on as under-developed weaklings will not easily survive the competitive life in a deep litter system and early culling is desirable. At the same time, chick mortality at a higher level than encountered in these intensively housed flocks is likely in any

alternative more traditional system.

The comparison between winter and summer losses indicates that many conditions are more lethal in winter but the differences are not so pronounced as might be expected. The major factors of human skill and human error which can determine a successful rearing have to be seen against a background of the many tasks to be undertaken by farm staff, and staff in supporting industries, often with limited time available and under a considerable pressure of events.

Acknowledgments

The assistance of the following at various times during the survey is acknowledged: Dr. J.R. Baker, Mr. I.R.D. Cameron, Dr. F.T.W. Jordan, Dr. M. Obwolo and Mr. P.C.W. Williams. Mrs. C. Taylor and Miss J. Hemsworth provided technical assistance. Mr. C.J. Randall examined brains from chicks with nervous disease. Farm staff were most helpful. Preparation of the manuscript by Mrs. C.J. Roberts.

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Printed at the University of Edinburgh