Ringworm - occurrence, control and prevention in young cattle

Thesis

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Ringworm - Occurrence, control and prevention in young cattle

By

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DECLARATION

I hereby declare that this thesis has been composed by myself, that it has not been submitted previously for a higher degree, that the work of which it is a record has been done by myself or, where jointly with other workers, is accompanied by a signed declaration by these workers, and that all quotations are distinguished by quotation marks and the sources of information specifically acknowledged.

John Edwardson

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ABSTRACT

Regular weekly observations of naturally occurring outbreaks of ringworm in young cattle over many years have provided detailed information on facets of the epizoology of the disease, control by therapy and preventive measures.

The epizoology or distribution of the disease demonstrated an average incubation period of 28 days.

Sites of lesions aided detection of early disease with the predominant site for first lesions being around the eyes. In yearlings unusual sites for lesions included the perianal area and under the jaw. All outbreaks were found to resolve with / to spontaneous recovery and in these studies the mean duration of lesions was 4 weeks, duration in animals was 8 weeks, and in outbreaks 18 weeks in untreated disease of naturally occurring outbreaks. These figures demonstrated the importance of determining spontaneous recovery in assessing the value of therapy in drug trials.

Control by therapy was found to be variable and difficult if not supported by preventive measures.

Controlled drug trials by strict regime have shown the effectiveness of Griseofulvin. However the use of Natamycin or Enilconazole was not so effective if treatment was delayed.

Early therapy with copper naphthenate and cod liver oil was effective when supported by preventive measures. The major obstacle to

control on calf and beef units appears to be the frequent introduction of infection via purchases of calves.

Fifteen outbreaks were considered to be introduced through 45 group purchases in 3 years.

GENERAL INTRODUCTION

Although ringworm is a common disease in cattle there is a paucity of information about naturally occurring outbreaks. There is a need for information to clarify the epizoology of the disease in naturally occurring disease in modern intensive beef production, rather than in experimental work prompted the detailed study to provide the facts and problems relating to control by therapy and prevention.

LITERATURE REVIEW

Ringworm - The Disease

Various descriptions have been used for the condition of ringworm. Ainsworth and Austwick (1973) produced a working definition of ringworm being "the common name given to the disease. Superficial infections of keratinized layers of the skin and its appendages (hair, feathers, horns) of farm, domestic and wild animals and birds (also man) caused by a group of closely related fungi known as the dermatophytes." The common term for the disease - ringworm - appears to arise from various definitions resulting from 'rounded lesions' produced by 'worm-like' organisms affecting skin 'dermis' and hair 'trichos'. The mycotic origin of ringworm was first discovered by Groby 1843 (cited by Mortimer 1955).

In cattle the dermatophyte Trichophyton verrucosum appears to be the only fungus causing ringworm worldwide (Ainsworth and Austwick;

(1959), and is the most common cause of the disease in the USA and Canada (Georg, 1956). However, Trichophyton verrucosum may affect all farm animals (Jensen and Mackey, 1971; Ainsworth and Austwick, 1973). Various synonyms have been used for the organism including T. album, T. ochraceum, T. discoides and T. faviforme.

Other dermatophytes have been recovered from cattle with ringworm and those have included T. rubrum, T. violaceum, T. mentagrophytes, Microsporum gypseum and M. canis (Carlton and Armstrong, 1980; Davison, 1968; Jungeman and Schwartzman, 1972). Other isolations have been made including T. villosum in China (Ainsworth and Austwick, 1959), T. papillosum in Syria and Morocco (Le Basque, 1933, 1934), Grubyella camerunensis in the Cameroons (Ota and Gaillard, 1926) and T. megnini in Brazil (Salles Gomes, Freitas and Cunha, 1954) and Germany (Blank, 1955).

Symptomatology

The lesions are discrete and vary in size from circular patches, 1 cm or less in diameter, to extensive areas. Sometimes there is only scaliness and loss of hair. More usually the lesions, at first white, develop thick, yellowish-brown, asbestos-like crusts (Ainsworth and Austwick, 1959) and may be dry and circular with extensive areas of alopecia (Blank, 1953), the extent depending on the dose of fungus (Lepper, 1969, 1972). The numbers of lesions in naturally occurring outbreaks are rarely given (Ford, 1956; Edwardson and Andrews, 1979). However Misra (1973) reported between 5 and 23

lesions per animal, mainly occupying sites on the head. It has been suggested that lesions on housed animals were more likely to attract attention than the same disease at pasture (Pepin and Austwick, 1968).

Workers have variously described their findings regarding location of lesions. Thus Ford (1956) and Ainsworth and Austwick (1973) stated that lesions occurred most frequently on the head and particularly the neck. However, Jensen and Mackey (1971) wrote that the face, especially around the eyes, and the neck, were the usual sites. Gentles and O'Sullivan (1957) gave more precise information of distribution for 77 cattle namely:-

	Head	Neck	Shoulders	Back	Lumbar region	Tail
Animals	61	41	12	18	12	7
Percentage	79	53	15	23.5	15.5	9

Other descriptions of siting include 'head', 'around eyes', 'above the muzzle' and 'bases of ears' (La Touche, 1952), whilst Anderson and Campbell (1964) state that head, neck and perianal areas were noted as frequently affected 'regions'. Muende and Webb (1937) reported lesions as being sited in the moist areas anterior to the shoulders, around the eyes, nostrils and mouth.

The predilection for sites indicated contact with objects likely to cause trauma (Klobusicky and Buchvald, 1974). These authors indicated that head, neck and tail were the first, second and third most common sites respectively of several sites examined in many animals.

The least often affected places included abdomen, dewlap, sternum, inguinal region, udder and claws of fore and hind legs. Continuous trauma of the neck by the tethering chain explained the frequent localisation of lesions in that region.

Pandey (1979a) described the distribution of lesions as "the site of ringworm lesions is dependent on the sex and age of animals. Sex is the key factor for animals older than 12 months, with lesions characteristically occurring on the thorax and limbs in cows and heifers, and on the dewlap and the intermaxillary space in bulls".

The first and most common lesions in a naturally occurring outbreak in calves were usually around the eyes (Edwardson and Andrews, 1979). Other sites in descending order of frequency were the face, ears, neck, perianal area, hind legs, trunk and shoulders.

Duration of lesions

The duration of individual lesions has been rarely reported except for some experimental infection (Sellers et al., 1956; Lepper, 1972) and a case of field disease (Edwardson and Andrews, 1979).

Table 1.1 - Duration of lesions in natural and experimentally induced ringworm

Reference	Experimental disease Natural disease	Duration of lesions
Sellers et al. (1956)	Experimental	Within 4 M of first injection
Lepper (1972)	Experimental	42 - 189 D young animals 6 - 27 W young animals
	Experimental	21 - 88 D older animals 3 - 12½ W older animals
Edwardson & Andrews (1979)	Natural	1 - 11 W range 8 W mean

Key: D = days; W = weeks; M = months

Duration of the disease in animals

Duration of disease in individual animals (Table 1.1) varies according to the extent of lesions and the dose of fungus (Lepper, 1972) and is shorter in older animals than younger ones with regression at 19 - 36 days in all animals. The period might be shortened by licking and grooming activities (Lepper, 1972; Edwardson and Andrews, 1979). In naturally occurring disease the duration in animals from first signs to complete healing, including regrowth of hairs, averaged 8 weeks in individual calves and ranged between 3 and 12 weeks (Table 1.2).

Ford (1956) reported an average period of 17 weeks with a range of 2 - 26 weeks. Obviously the duration of outbreaks was longer (Table 1.3).

Table 1.2 - Duration of the disease caused by natural or experimentally induced ringworm on animals

Reference	Experimental disease Natural disease	Duration in animals	Comments
Lepper (1972)	Experimental	7 - 13 weeks	Daily inspection New hair growth
Lepper (1972)	Experimental	8 - 27 weeks	Complete healing
Rook et al. (1954)	Experimental	6 - 12 weeks	
Edwardson & Andrews (1979)	Natural	3 - 12 weeks	Weekly inspections
Ford (1956)	Natural	2 - 26 weeks	

Duration of outbreaks

Table 1.3 - Duration of outbreaks in naturally occurring ringworm

Reference	Experimental disease Natural disease	Duration of outbreak	Comments
Ford (1956)	Natural Natural	10 W - 17 W 30 W	Group outbreak Farm outbreak with inter-mixing
Edwardson & Andrews (1979)	Natural	20 W - 33 W	Farm outbreak with inter-mixing
McPherson (1959b)	Natural	11 W 13 W 14 W	
Key: W = weeks			

Spontaneous recovery

All artificially produced disease has been found to recover spontaneously without treatment within one to five months in all animals (Hoerlein, 1945; Kielstein, 1967). A similar view was expressed in other studies with spontaneous recovery occurring 150 days after infection was first noted and 90 days after treatment (O'Brien and Sellers, 1958). Spontaneous recovery (Table 1.5) has been suggested to occur when animals were turned out in the spring but McPherson (1957) contended this opinion.

Table 1.4 - Spontaneous recovery in natural and experimentally induced disease

Reference	Experimental disease Natural disease	Recovery period	Comments
Hoerlein (1946)	Experimental	1 - 5 M	
Keilstein (1967)	Experimental	1 - 5 M	
O'Brien and Sellers (1958)	Natural	150 D 90 D	After 1st signs After treatment

Immunology

It is stated that there is some natural immunity against ringworm in cattle (Ainsworth and Austwick, 1959). However, there is little precise data regarding acquired immunity (Ford, 1956; Sellers et al., 1956), although Lepper (1972) considered there was lasting acquired immunity by ringworm.

During a period of 11 years (1955 - 66) samples from cattle on 438 premises were examined and 223 were found to be infected with T. verrucosum isolated from 199 of them (Pepin and Austwick, 1968). They stated that no dermatophyte could be grown from the other 24 samples, but there was little to suggest that these were infected by species other than T. verrucosum.

English (1970) and Pepin (1984) suggested that direct microscopy of a dermatological specimen in potassium hydroxide (which can be done within half an hour) is all the practitioner requires to decide on therapy. However, the fungus was sometimes missed when using microscopy and culture was necessary to confirm disease where samples were negative or doubtful. This author contended that petri-dish cultures allowed direct examination and speeded the obtaining of cultural findings because the colony is easily recognised as T. verrucosum showed short thick hyphae, many ending in swollen vesicles whilst the same method allowed removal of fast growing contaminants. Lepper (1972) stated that T. verrucosum could not be isolated until 21 days after inoculation and could only be isolated for half the period that lesions were present.

Carrol (1974) indicated the importance of colour changes in evaluating dermatophyte test media for diagnosis of dermatophytes. Undertaking the determination of whether a culture was a dermatophyte or a saprophyte, they stated that it was necessary to consider colony, pigmentation and time of colour change, in addition to DTM colour change. Two observations were presented:-

1. Colony Colour: Dermatophytes were light, white to apricot in colour. Saprophytes were usually coloured brown, grey, green or yellow.

2. Time of Colour Change: Dermatophytes changed the colour of the medium before or at the time of first visible growth. Saprophytes had a well established growth before inducing a colour change.

A rapid method for the identification of the slow growing T. verrucosum was suggested by Lamport et al. (1984). They considered Sabouraud's medium to be doubtful for the initial isolation because of the strong probability of fungal and/or bacterial contamination and there appeared to be no benefit from the inclusion of yeast with mycobiotic medium. Mycobiotic medium alone was found to be the most satisfactory for culture purposes at an incubation temperature of 37°C. However, Pepin (1984) contended that 0.5% yeast improves the culture medium.

Ellis (1982) considered the success of the culture technique for isolation of T. verrucosum was probably due to:-

1. The material selected used for inoculation of plates.

2. Medium containing cycloheximide and chloramphenicol as inhibitors of fungi and bacterial contaminants.

3. Medium used in sufficient depth in plates.

4. Incubation at 37°C for the first week to encourage rapid growth of T. verrucosum and discourage growth

of contaminants, and then reducing to 25°C to prevent drying out of medium.

5. Plates examined regularly for early diagnosis of a positive culture before being overgrown and to allow sub-culturing, the identification of the fungus being confirmed by examination of the colonial growth in cotton blue lactophenol.

Ellis (1982) further stated that glucose-peptone agar (Sabourauds glucose agar) containing chloramphenicol and cycloheximide as saprophytic bacterial and fungal inhibitors is frequently used as a culture medium in petri-dishes or as shapes in McCarthey bottles.

Differential diagnosis

Georg (1954) deplored the failure of veterinary surgeons to carry out laboratory examinations, alleging that ringworm might be confused in man or beast. This has led to much expensive, persistent and succesful 'ringworm treatment' for cases of seborrhoea, scabies, eczema, vitamin deficiency and other dermatological conditions.

Mycotic dermatomycosis (Dermatophilus congolensis infection) is registered as having similarities to ringworm but the incidence in Great Britain does not appear to be high in cattle, nor should it or its lesions cause problems in differentiation (Reid, 1921; Albiston, 1933; Shoultz, 1955; Chodnik, 1956; Brooks, 1960; Egerton, 1964 and Munday, 1967).

Infectious foci were thought to occur independently of one another on the animal and penetration ceased at a time when a focus reached a certain size. After one to five months spontaneous healing set in, inferring that resistance and immunity had developed (Kielstein, 1967). The same author (quoted by Lepper, 1969) stated that black-coated animals, due to copper and iron pigment, were better able to resist ringworm than others.

Recrudescence

No recrudescence has been observed after initial recovery in several naturally occurring outbreaks which were untreated (Edwardson, 1980, unpublished data). Otherwise the phenomenon does appear to have received little comment. Recurrence was recorded by Pandey (1979a) in a self contained herd with no recent intakes. This could not be interpreted as recrudescence but more probably infection from the environment for animals not previously affected.

The use of drugs may in certain circumstances influence the acquisition of immunity, and be followed by a second infestation or recrudescence. The danger of re-infection after treatment is noted on data sheets for some therapeutic agents (Fulcin 87/88, Grisovin 87/88, Mycophyt 87/88).

Diagnosis

Clinical diagnosis of ringworm is held by some authors to be significant and basic (Jensen and Mackey, 1971) but others state laboratory diagnosis is important for differential diagnosis and the application of

rational therapy (Ainsworth and Austwick, 1955). Pepin and Austwick (1968) declare, however, that 75% of laboratory samples are negative for fungal diseases. Thus it appears that the symptomatology and siting of lesions aid clinical diagnosis, whilst the pathology of the disease and finding of the causal organism confirm a diagnosis. The lesions of bovine ringworm are sufficiently distinctive and characteristic for accurate diagnosis - the single or multiple circular, dry, scaly, grey, depilated crust being a significant sign (Jensen and Mackey, 1971).

Pathology

When diagnosis is being performed in the laboratory Georg (1954) noted that T. verrucosum was not detected by Woods lamp. Menges and Georg (1957) considered that culture was important since infected hairs could be overlooked in direct examination. Clinical diagnosis of ringworm can be confirmed by mounting hairs from lesions in caustic potash when infected hairs are seen to be sheathed by spores 2 - 8 μ in diameter which, in early infections, are arranged to be seen in linear series (Ainsworth and Austwick, 1959). For specific identification cultures have to be made and the isolation of T. verrucosum in pure culture is often difficult because this species is slow growing and has rather exacting nutritional requirements (Ainsworth and Austwick (1959) quoting Georg (1950)) and because the skin of cattle is usually heavily contaminated with the spores of saprophytic moulds as well as bacteria.

Epizoology

Present knowledge of the epizoology of ringworm has been mainly retrieved from surveys undertaken in various countries (McPherson, 1957; Mortimer, 1955; Sellers, 1956). Calf purchases may be a major source of infection on non-breeding premises (Horrox, 1976) and it was advised that buying-in infection should be avoided. Infected transport and interim premises might be involved in some cases (Edwardson and Andrews, 1979). In buildings infection could be contracted from diseased animals, contaminated walls, partitions, woodwork and equipment (Ainsworth and Austwick, 1959). Dust could also be hazardous on farms (Ainsworth and Austwick, 1959). Major importance must be attached to the findings of McPherson (1957), who pointed out that fungi and their spores could live several months outdoors in northern Britain through summer months whereas indoors at room temperature the durability could be four to five years. Ultra violet light could kill spores in a few minutes *in vitro*. Kachmic and Tracik (1969) also found that the organism survived for five months in cattle hair and crusts buried 5 cm within animals bedding, and could be cultured from an infected hair which had been in a cow's stomach for 72 hours.

Kachmic *et al.* (1967) found that *T. verrucosum* survived four years and three months in the laboratory and also proved survival under straw bedding. These survival factors explained the incidence of disease by contact with contaminated environments and in the absence of infected animals, Ozegovic *et al.* (1967) stated that the fungus was not always found in bedding and straw but was sometimes found in fomites in brushes and from wooden barriers. Chatterjee *et al.* (1983), quoting Walker (1955), noted that the fungus could not be

found in soil on infected premises although many sheds contained affected animals, but it was isolated from wooden gates and contaminated walls. Ainsworth and Austwick (1959) supported the findings that the fungus was not always in dung substrate or soil.

Table 1.5 - Incubation period of naturally and experimentally induced ringworm disease

Reference	Natural or experimental disease	Incubation	Frequency of observation
Hoerlein (1945)	Experimental	18 - 24 D	Daily
Ford (1956)	Natural	4 - 6 W	Weekly
O'Sullivan (1959)	Experimental	5 W	Daily
Lepper (1972)	Experimental	7 - 35 D	Daily
Edwardson and Andrews (1979)	Natural	29 D	Weekly
Andrews and Edwardson (1981)	Natural	28 D	Weekly

Key: D = days; W = weeks

Whilst the incubation periods (Table 1.5) vary, assessment cannot always be made as to the starting date of risk with infection. Variation was partly due to dose of fungus and age of the animals (Lepper, 1972) but when disease occurs after specific incidents not only should the incubation period be calculated but the source of infection should also be indicated. This latter factor was of importance in planning control measures. In some outbreaks (Andrews and Edwardson, 1981; Edwardson and Andrews, 1979) cognisance was taken of relative movements and contacts in the period prior to manifestation

of disease. Lepper (1972) reported the period as variable for dose of fungus.

Incidence

Ringworm in cattle is world wide in distribution (Ainsworth and Austwick, 1959). It has involved investigations in the USA, Europe, South Africa, Russia and Australia (Jensen and Mackey, 1971). Blank (1955) provides a formidable list of countries suffering trichophytosis in cattle. Disease in Brazil was suggested to be imported from USA and Holland (Londero et al., 1963). The incidence was regarded as high throughout Great Britain and 15% of animals were found affected in a survey in East Anglia (Mortimer, 1955) while a study in northern Britain found 2.89% of animals and 25.7% of herds affected (McPherson, 1957a). Sellers (1956) found 28 affected premises when surveying 43 farms and reported hygiene low in affected places, with calves being the main age group affected. The incidence of infection was low in dairy herds but very prevalent in calf rearing and beef type establishments (Mortimer, 1955). Others have also stated that the disease was common in beef rearing herds (Sellers et al., 1956; McPherson, 1957a and later Ozegovic, 1967) commented on a high incidence on large farms. It was suggested that a marked increase in ringworm incidence occurred in Europe during the years 1961 - 1967 (Kaben, 1967) and this observation was supported by Chmel (1967) who considered that concentrating agricultural production, often under unfavourable conditions, had produced the increase. Human infection with cattle ringworm tended to be at its highest level in January, February and March when cattle were housed and during

June when pens were being cleaned (Gentles and O'Sullivan, 1957).

The spread of the disease was believed to be enhanced by gathering, crowding, crating for handling and blood sampling (Ford, 1979) whilst crowding of cattle in autumn housing can lead to an explosive incidence of ringworm (Chmel et al., 1967). Disease levels were higher in the winter when animals were housed and in close contact than in summer (Ainsworth and Austwick, 1959) but concentrations of animals, natural healing, grooming, dietary inadequacies and resistance might also play a part in epizoology (Sellers et al., 1956).

Little has been reported about ringworm outbreaks at pasture, although one was described by Ford (1956) who went on to state a general opinion that recovery was spontaneous at pasture. This was supported by Ainsworth and Austwick (1955) who stated most workers believed disease cleared in the spring. However, McPherson (1957a) claimed there was no support for the notion that disease disappeared with turnout in the spring. He (1957b) contended that summer sunshine did not penetrate scabs and crusts of ringworm lesions to kill the spores. However, in some countries, ultra violet light has been considered valuable in shortening the duration of cattle ringworm, making isolation of fungus difficult and presumably limiting the spread of disease (Abu et al., 1976). In addition, McPherson (1957b) affirmed that ultra-violet light killed fungus in vitro. However, conversely a survey showed that dark buildings and ringworm incidence were not related (McPherson, 1957a).

Infection with T. verrucosum occurs in other species such as dogs, horses, donkeys, sheep, goats and humans in spread from cattle. In the 1955 - 1966 period Austwick and Pepin (1967) found T. verrucosum in samples submitted to a major laboratory from the following animals:-

Horses - 19; Cattle - 199; Dog - 1; Pig - 1; Sheep - 22

Ainsworth and Austwick (1959) stated that spread occurs mainly from animal to animal, and from walls and woodwork. Dust in farm buildings may also aggravate the spread of disease (Ainsworth and Austwick, 1955) but lice were not considered a major influence in the spread of disease (McPherson, 1957a). In a thesis devoted solely to pediculosis in cattle there was no mention of any relationship with ringworm incidence (Oormazdi, 1976).

Control of ringworm by therapy

The older substances used in therapy against ringworm included brimstone (sulphur) and oil, petrol, paraffin and neat iodine (Horrox, 1976). Therapy has seen radical changes in the last 30 years with the discarding of old and introduction of new remedies. Systemic treatments have been evolved based mainly on the product Griseofulvin. Topical applications have also been developed in the form of sprays and shampoos and involve a multiplicity of agents. Those dealt with in this section are natamycin, enilconazole and copper napthenate in cod liver oil.

Griseofulvin

Griseofulvin (Fulcin, ICI, Grisovin, Glaxovet) was reported to be safe and effective when given orally, thereby reducing costs, avoiding handling and an easily administered premix (Hiddleston, 1970, 1973). However, although it was found to be effective, it was considered to be expensive and difficult to administer in some cases (Horrox, 1976; Pearson and Rankin, 1962). The drugs expense was also commented upon in a controlled trial using a fine particle mix with dispensing agent and where photography was used to assess the results of the treatment (Cobb et al., 1963). In a naturally occurring outbreak when efficacy was again assessed by photography but with no comprehensive details and figures of activity (Edgson, 1970), the mix was also considered to be expensive for large groups.

Uvarov (1961) and Fulcin Data Sheet (1977) stated that the drug was absorbed into the keratin layers of the skin. During treatment of all animals in a group the latter advised treatment of buildings with washing soda and disinfectant. Griseofulvin did not kill the fungus, but was stable and protected cells from infection. In guinea pigs the antibiotic was effective in vitro but had poor in vivo activity against the fungus because of non-penetration of keratin cells (Gentles, 1968). However, O'Sullivan (1959) found griseofulvin valuable in treatment of experimental disease. The danger of re-infection after treatment especially via spores surviving in long coats has been noted (Grisovin Data sheet, 1979). Small particle size was important in aiding the absorption of griseofulvin (Atkinson et al., 1962).

Gotz (1967) stated "In no circumstances do we give up local

antimycotic treatment for, as is well known, griseofulvin does not destroy the pathogen. Fungal elements scattered in the adjacent areas thus retain their vitality even though griseofulvin is given internally". In a griseofulvin drug trial (Andrews and Edwardson, 1981) all lesions were accurately recorded with cross infection between two groups possible and the trial was considered comprehensive and well designed to show the efficacy of griseofulvin (Ellis, 1982).

Natamycin

Natamycin (Mycophyt, Mycofarm) is an antibiotic commercially prepared shampoo or spray for horses, cattle and buildings. No precautions were required when applying around the eyes (Mycophyt Data Sheet, 1979). No contraindications were quoted for this non-toxic broad spectrum antimycotic (Spanoghe and Oldenkamp quoting Raab, 1977). The substance is fungicidal (Oldenkamp, 1979) and could be used on buildings as well as animals and is sporicidal.

Spanoghe and Oldenkamp (1977) conducted a trial using natamycin on 41 test animals with 10 untreated controls. Inspections were at three weekly intervals and results based mainly on mycological evidence. The period of observation was twenty weeks when 80% recovery was observed in the test animals and no recovery in the control group. However, the age of disease at commencement of trial was not given for either group and no comment made regarding spontaneous recovery. Oldenkamp (1982) found there was no prophylactic value in natamycin therapy.

Enilconazole

Enilconazole (Imazalil, Imaverol, Janssen, Crown Chemicals Co.) is a relatively new antimycotic agent for topical therapy which has been reported upon by several authors (Keyser, 1979; De Smet et al., 1979; Heykants et al., 1981; Crown Chemical company, 1981, 1982a, 1982b; Imaverol (Janssen) Data Sheet, 1981). Imaverol Data Sheet (1987/88) states Imaverol is a synthetic antimycotic and was indicated for the treatment of dermatomycosis or ringworm in cattle, horses and dogs. It is recommended that since dermatophytes will extend into the hair follicles, possible crusts must be removed with a hand brush which has to be soaked in diluted Imaverol solution. It is then highly recommended that the animals be sprayed entirely at the first treatment so as to reach the sub-clinical lesions. It is provided for external use only.

Copper naphthenate

Copper naphthenate is presented as a green solution with cod liver oil (Kopertox, Crown Chemical Co.).

It is used as a topical spray and it is claimed the product will penetrate lesions until infection and kill spores without affecting ringworm immunity. In consequence the Kopertox Data Sheet (1987/88) writes "do not scrape, brush or remove ringworm lesions".

General aspects of therapy

Ainsworth and Austwick (1959 and McPherson (1959b) asserted that

topical drugs had little effect on the natural course of disease. McPherson (1959b) also asserted that none of the antimycotic agents tested by him influenced the duration of the experimental lesions, and went on to write that from the practical point of view it could be assumed that if treatment of a group of animals was not instituted until infection was widespread, as assessed by visual inspection, and has been present long enough to stimulate the average owner to action, any agent may well be credited with merit. The best that could be said is that surface dressings may curtail the extension of recent lesions and limit the dissemination of infective material.

Fourteen years later, Ainsworth and Austwick (1973) still stated there was no satisfactory treatment for ringworm and infection was self limiting.

Other workers queried the penetration of drugs to effective levels. Gentles (1958) asserted that many drugs of high antifungal activity *in vitro* produce clinically disappointing results. This was probably due to inability to penetrate the keratin of the skin, hair or nails to the site of dermatophyte activity. This was confirmed by Ellis (1982) in her findings on a controlled drug trial. In 1963 the multiplicity of topical remedies both home-made and proprietary suggested to Cobb *et al.* (1963) that none was successful and the only report of a controlled clinical trial of topical remedies (McPherson, 1959) showed all to be without effect. It would indeed be surprising if any local treatment was effective in this condition, because growth of the *Trichophyton* fungus occurs within the base of the hair, some millimetres beneath the skin surface where it cannot be reached by superficial

applications. It was probable that the great variation in the duration of untreated lesions gives rise to impressions of cure where no effective controls were kept. However, Horrox (1976) suggested full shampoo with modern drugs to stop spread from first lesions, and gave a list of older drugs which were thought to be dangerous.

Treatment of buildings and equipment required essential detergents, lysol, sodium hypochlorite, washing soda and creosote as basic materials (Horrox, 1976; ICI Fulcin Data Sheet, 1977). Ellis (1982) quoting Blood et al. (1979) wrote "Topical treatments are of greatest value in initial stages of an outbreak, to curtail extension of lesions and limit dissemination of infective material".

Drug tests and trials

Variations in the duration of lesions in outbreaks of disease probably reflect immunological factors. This variant together with the unknown age of lesions in most clinical trials, renders assessment of the efficacy of antimycotic agents hazardous by this means. Thus experimental lesions were considered to provide superior biological testing facilities (McPherson, 1959b; O'Brien and Sellers, 1958).

Ellis (1982) noted common failings in trials included failure to record all lesions regularly and accurately except for one (Andrews and Edwardson, 1981) and further stated that until 1979 the epizoology of ringworm had received little attention. Many therapeutic agents had been recommended but few had received properly controlled trials. It was now known that, in assessing an agent, factors which had not previously been appreciated should be taken into account.

Drug trials reported in the literature are listed in Appendix 2 of this work and show that full life history of disease in animals under trials is not provided in most reports. In consequence, according to McPherson (1959a), drugs applied in advanced disease may gain the merit or kudos for spontaneous recovery. Likewise, Ellis (1982) refers to the comprehensive study by Edwardson and Andrews (1979) as significant when considering therapeutic efficacy as it described individual variations and spontaneous recovery. The incubation periods and duration of lesions were recorded, lesions were found to develop whilst others receded. Ellis reports that all animals should be inspected at weekly intervals in drug trials. It was also considered by the author that all lesions should be identified and recorded by site and size, using a list of sites, with abbreviations for each, and indicating right or left side of animal. A new lesion was defined as one which had appeared since the previous week and therefore could be up to 7 days old. Lesions were recorded until there was total hair regrowth over the area, and this was the definition of recovery of a lesion. An animal was defined as clinically cured when all lesions had disappeared.

Factors which one considered relevant in assessing drug effectiveness by trial are listed by Ellis (1982):-

1. Sufficient numbers of animals.
2. Sufficient numbers of similar untreated control animals for comparison, kept in similar conditions.
3. Random division of animals into the two groups.
4. Mycological confirmation of disease.

5. No variations in mode of therapy.

6. Age of lesions prior to therapy and previous history of disease should be known, to take natural resolution and specific acquired immunity into account.

7. Lesions regularly recorded for size, site and duration.

8. Definition of recovery should be given.

9. Observation of recrudescence of disease.

10. Statistical analysis of data.

The aim of the above factors is to exclude as many variables as possible which might influence the result, but this is not always possible especially in field trials with naturally occurring disease (Ellis, 1982). It was concluded that in the trial undertaken by her not all desirable criteria were met but on the average farm and under normal farm practice, the conditions and findings were of practical value.

Control of ringworm by preventive measures

This section is a summary covering aid in control by direct prevention, husbandry, hygiene, disinfection and vaccination.

There is a paucity of advice regarding the very significant factor of preventing the introduction of infection by calves purchased for rearing herds and where such animals show high incidence of the disease. However, Horrox (1976) advised the most important thing was not to buy in infection. He was surprised how many farmers did not think twice about purchasing a calf with one small lesion which could cause

trouble throughout a complete herd. La Touche (1955) gave somewhat similar advice that some degree of effective control could certainly be introduced by careful inspection of cattle for sale at local cattle fairs, with a view to preventing the movement of those with ringworm. He further states that the eradication of cattle ringworm by means of prophylatic measures in combination with hygiene and therapeutic practice must be laborious, and unrewarding for a long period for practical and economic reasons, but that no progress can be made without a start.

Eradication is impracticable but control can be successful by understanding factors influencing the spread of the disease (Austwick and Pepin, 1967). In this context a fundamental factor is the information regarding viability and longevity of fungi given by McPherson (1975b) who found that spores survived in buildings between 4 and 5 years and outdoors, several months. Thus, likely sources of infection are important when planning husbandry practices. Ford (1956) indicated spread through the common meeting place of a cattle crush/crate where animals were held for bleeding - when the rubbing on the sides of the crate aided the production of many neck lesions. Trauma in tethering chains also produced multiple neck lesions in outbreaks reported by Klobusicky et al. (1974). In recent years several workers on the disease have reported increased incidence relating to intensivism and high concentration of animals.

Zrunek (1967) stated that in large concentrations prevention was rendered even more difficult and recommended paracetic acid spray for cattle and cowshed installations at 6 weekly intervals to reduce weight

of infection. Chmel (1967) thought that concentrating agricultural production, often under unfavourable conditions as far as prevention is concerned, has been mainly responsible for the fact that spread of infection has today become a problem not only for central and north-west Europe but also for other European countries. Even on other continents an increased incidence rate has been noted where disease was rarein the past with the highest incidence being reported on highly mechanised and heavily stocked farms (Thelin, 1980).

It is generally considered that ringworm infection seems to be contracted by contact of healthy and diseased animals with contaminated walls and woodwork of buildings. Calves are commonly kept confined so that there are many opportunities for spread of infection and for constant re-infection within a group of animals, and for an infected group of animals to contaminate the environment (Ainsworth and Austwick, 1959). These authors (quoting McPherson (1957b)) write that the spread of T. verrucosum was unlikely to be influenced by summer sunshine, but the few contacts between one animal and another in the field might well account for the few records of epizootic outbreaks while the animals are at grass. Ozegovic and Grin (1967) reported the difficulty of controlling disease by treating only affected animals especially with much intermixing and changing of pens.

Fraser (1974) and Hafez (1952) dealing with animal behaviour record the licking, rubbing and sucking features in grooming exercised between animals. These exercises indicate a mode of spread of disease which could be heeded in husbandry practices to minimise spread of

disease, especially in young calves in single pens not segregated.

Various husbandry practices hereto recorded indicate the emphasis which must be placed on hygiene to minimise and prevent build-up of weight of infection.

Refai and Milgy (1968) stated that uncrowded buildings with the application of general hygiene measures minimise the incidence of infection. The viability and longevity of spores in the environment has been recorded (McPherson, 1957b) and this was further emphasised by Kachmic et al. (1967) who found the fungus was viable for 4 years 3 months under cattle straw bedding. This evidence inevitably forecasts build-up of infection in premises unless hygiene measures are strongly exercised. As previously stated, Ozegović (1967) found fungus in the fomites of brushes used for grooming. The importance of dust should not ignored since this may harbour spores on ledges, in crevices and generally in the animal environment and thus be hazardous without rigid hygiene (Ainsworth and Austwick, 1955).

Ainsworth and Austwick (1959) also emphasise the spread of disease not only from infected animals but also from contaminated walls and woodwork of the buildings. Because of the long survival of spores in dung it should be removed from animal sheds 200 - 300 m and allowed to ferment completely before use as manure (Ryabova, 1955). Rook et al. (1954) recommended a high standard of hygiene scouring of old woodwork and disinfection to reduce incidence. The practice of extensive hygiene is underlined by these authors to minimise

build-up of infection in cattle environment, where disinfection may not be practised in occupied pens, or in preparing for disinfection in vacated premises or equipment for disinfection. Hygiene presumably will additionally reduce the expense of disinfection and therapy.

A major contribution on the disinfection of buildings and equipment has been made by McPherson (1959a) who undertook the testing of many substances in a search for fungicides. His conclusions were that lysol, phenol based disinfectants, sodium hypochlorite and ammonia were the most efficient agents. Horrox (1976) advised that whichever medical method is used to combat ringworm, it is vitally important to take active steps to reduce infection within the buildings. For metal and brick-work, blow lamp sterilisation was proposed, with hot water/washing soda solution as next best application and for woodwork creosote was recommended. Ainsworth and Austwick (1959) said much can be done to reduce the chances of infection by the thorough cleaning of cowsheds, and success has been claimed in breaking a succession of epizootics among calves kept in a particular shed by spraying both walls and woodwork with Bordeaux mixture or other copper-containing fungicide.

Calton and Armstrong (1980) referred to many reports in overseas literature about the use of vaccine prepared from T. verrucosum for the treatment and prevention of bovine dermatomycosis. The reports indicated that the vaccine TF 130 was highly effective and approved by the USSR Ministry of Agriculture. Ellis (1982) quoted Sarkisov (1978) that the use of 'TF 130' vaccine had been reviewed and shown that an 80% reduction in bovine ringworm in cattle in USSR followed

vaccination of 130 million cattle.

Naess and Sandvik (1981) and Aamodt et al. (1982) reported on the testing and use of the vaccine with official approval in Norway. Others have reported on vaccination particularly in Europe, including the Netherlands (Brethouwer, 1982), Norway (Liven et al., 1985; Gudding, 1986), USSR (Heifits, 1985). The vaccine used in these countries was produced from T. verrucosum in the Soviet Union (Liven et al., 1985). Naess and Sandvik (1981) raise attention to the need for very early (when only days old) protection of calves moved from infected herds.

Carlton et al. (1980) stated that the vaccine was not currently available in the USA and also pointed out that in Norway ringworm is a notifiable disease where regulations forbid animals to be moved from infected herds to markets and public pastures when infected.

CHAPTER 2

Some observations on an outbreak of ringworm in a group of young cattle reared indoors

INTRODUCTION

An outbreak of ringworm was studied in a group of 32 Friesian bull calves being reared intensively for beef. Weekly observations were made following their arrival at the farm at about one week old until slaughter at $10\frac{1}{2}$ to 12 months of age.

MATERIALS AND METHODS

Thirty two Friesian male calves originating from 31 different premises entered rearing accommodation on 31/1/77. Most had been held at the dealer's premises prior to delivery to the unit. Their ages were estimated to be one week old upwards at the first inspection on 3/2/77 (Week 1). During the investigation the calves occupied three different sets of buildings. First, they were in single pens, next in open sheds as four groups and finally as two groups in other open-fronted sheds.

The animals were inspected at weekly intervals until slaughter was completed on 20/3/78 (Week 53). Additional identification to facilitate observations was by large-number ear tags.

Each animal was inspected weekly for general health and skin lesions. The latter were recorded on foolscap-size sketch cards. Trichophyton verrucosum was found by culture in skin scrapings from several animals.

RESULTS

Spread of infection

Thirty of the thirty two calves developed ringworm (see Table 2.2). The first animal (No. 104) showed the disease 10 days after arrival at the unit, with a second calf (No. 87) developing lesions 24 days after arrival. At 15 days after weaning and movement to the hardening-off area, four other calves showed signs, two formerly in contact with No. 104, one in contact with No. 87 and one formerly opposite No. 104.

Sites and distribution of lesions

Site of first lesion

In the thirty affected animals the first lesion was seen around the eyes (10 cases), on the ears (10 cases), face including forehead (4), neck and jaw (3) and hind legs (3).

Number of lesions

The total number of lesions recorded for the thirty affected animals was 316 (mean 10.5 ± 8.95 SD lesions per animal). The number of lesions on individual animals ranged from 1 to 40 (Table 2.1).

Sites of lesions

The most usual sites were eyebrows and adjacent areas (100), followed by face, including poll area (85), ears (58) and neck including jaw area (32). Distribution of lesions is shown in Table 2.2.

Shape and area of lesions

Some 286 lesions were circular in shape and varied in size from 0.4 cm to 10 cm in diameter. There were 30 angular lesions which varied in size between 0.7 cm x 1.3 cm and 7.5 cm x 10 cm. The average infected area in each animal was 20.7 ± 16.61 SD sq cm with a range of 8.6 sq cm to 68 sq cm.

Duration of the lesions

Duration of ringworm in individual animals was considered to be the period between the first sign of hair loss to the covering of the lesion with new hair growth. The mean period of disease in the thirty animals was 8.0 ± 2.67 SD weeks with a range of 1 to 11 weeks. The mean duration of individual lesions was 3.6 ± 2.17 SD weeks and varied from 1 to 11 weeks (Table 2.3).

The outbreak continued in the study batch for 28 weeks from the first sign in the animal No. 104 to the first sign in the last-affected animals, Nos. 89 and 92. The last sign of disease was on animal No. 92, 33 weeks after the first sign on No. 104.

Therapy was not applied in this outbreak of ringworm.

DISCUSSION

Origin of infection

The source of infection of the first-affected calf (No. 104) was most

probably the premises of origin of the calf, but it could have been contracted in transit as the lesions appeared 10 days after arrival of the calf. However, as average incubation appeared to be 4 weeks, the farm of origin was more likely to be the source rather than the lorry used in transit, interim premises or the rearing premises.

The origin of infection in the second animal (No. 87) was harder to determine, as lesions developed 24 days after entry and this calf was from a different source from No. 104, but again infection was probably contracted on farm of origin or in transit.

Spread of infection

The two first-affected calves appeared to introduce infection to the premises of trial. Hygiene, cleaning and disinfection were practised between batches in the initial accommodation.

The following agents and factors were considered in assessing and exploring the spread of infection:-

1. Tongues: licking, sucking and grooming
2. Direct contacts; infected animals - rubbing, riding, butting
3. Rubbing posts and places; buildings, pens, feed hoppers, water bowls, partition bars, catching crates, weigh machines, common meeting places and tools
4. Indirect contacts; handling, dehorning, operations, litter vermin.

In the year of observation factors 1. and 2. were considered the most

important.

It was noted, however, that the calves licked the woodwork and they especially licked one another, particularly around the head, throughout the period of observation. There was marked tongue action, with sucking, between young unweaned calves before and after feeding in the early morning. It is postulated that in some outbreaks, the mode of transmission might be as follows:- licking and sucking of an infected animal or lesion might lift infection in scales, scabs and hairs. The tongue papillae could hold or harbour the hairs with spores; the individual animal then licking another area on itself, or another animal, could deposit infection in a slightly scarified area, moisture then aiding the anchorage and attachment of spores to the hairs and skin. Although the stress of re-grouping, intermixing of animals, changing of accommodation at different age points and the formation of new social orders may have aided spread of infection, it would appear probable that in this study contact with infected animals was the most important factor. Upsurge in numbers of new lesions occurred 1 to 6 weeks after each new intermixing.

Susceptibility

Farmers commonly hold the view that sick, unhealthy, and undernourished animals are more susceptible to ringworm and suffer more severely than healthy robust animals (Sellers et al., 1956). This was not the case in the present study, when two animals not affected in the batch suffered other disease. The only animal to survive, and not be affected, had severe enteritis and dysentery and was the lightest calf in weight for a long time. The other animal died after

pneumonia 48 days after arrival, but might have suffered ringworm had it survived.

CONCLUSIONS

The study revealed and demonstrated spontaneous recovery and provided information on duration of disease in lesions, individual animals and in a farm outbreak.

Table 2.1 - Number of ringworm lesions in individual calves

Number of lesions	Number of animals affected
1 - 5	12
6 - 10	6
11 - 15	5
16 - 20	3
21 - 25	2
over 25	2

Table 2.2 - Distribution of ringworm lesions and number of animals affected at each site

	Around Eyes	Ears	Muzzle Nostrils	Forehead Face	Cheeks Jaw	Poll	Dewlap Neck	Shoulders	Withers	Body Trunk	Legs	Perianal Tail area	Total
No. of lesions	100	56	24	49	24	5	19	5	-	7	11	16	316
Percentage	61	18	8	16	8	2	6	2	-	2	4	5	
No. of animals	26	23	15	10	14	3	7	5	-	5	7	13	30
Percentage	87	71	50	35	47	10	23	17	-	17	23	43	

Table 2.3 - Duration of individual ringworm lesions

Number of lesions	Number of weeks duration
61	1
54	2
62	3
47	4
34	5
20	6
19	7
9	8
8	9
1	10
1	11
-	12

CHAPTER 3

Ringworm in young Friesian cattle reared indoors and outdoors

INTRODUCTION

The epizoology of ringworm in 44 Friesan male calves was studied on a beef unit, where the animals - purchased at a week old - spent their first winter indoors and the following summer outdoors at grass.

MATERIALS AND METHODS

Forty-four male calves were purchased from a dealer on 14/9/77 and they were housed in single pens in a controlled environment. Weeks 1 to 6 were spent in this accommodation where dehorning was performed in Week 4 and castration in Week 6.

Preventive measures undertaken resulted in all manure and litter being removed. Additionally all fittings, equipment, walls and floors were hosed with phenol-type disinfectant, and steam under pressure, prior to entry of cattle into single-pen accommodation and hardening-off areas.

However, the accommodation occupied in Week 15 and subsequently, were not so treated. There was no application of antimycotic therapy in the outbreak. Antilice powder was applied to all animals in Week 5 and a regular weekly inspection of the calves was made throughout their lives from Week 1 to Week 91.

Subsequently between Week 7 and Week 14 the animals were separated into two groups and held in hardening-off accommodation.

Week 15 to Week 24 were spent in beef yards and Week 24 to Week 25 in similar accommodation. In Week 36 they were moved to Pasture A, in Week 45 to Pasture B, Week 47 back to Pasture A1 and in Week 50 to Pasture C.

RESULTS

Ringworm lesions appeared on 16/2/78 (Week 23) in three animals, two in one pen and one in another, in a covered yard house. Each group had contact with the other through and between horizontal bars and each group fed from troughs guarded by sloping bars. One animal died from ruminal tympany on 9/2/78 (Week 22) and 39 of the remaining 43 animals subsequently became affected with ringworm; 34 were affected inside and 5 became affected whilst at pasture.

There was no re-appearance of disease after the initial duration of 28 weeks, ending on 23/8/78 (Week 50). The test batch (43 animals), newly recovered from ringworm, were intermixed with 38 fat Hereford cattle, showing ringworm on 11 animals, between 28/8/78 (Week 50) and 12/9/78 (Week 53) but no further incidence of disease occurred in the former batch during the following 38 weeks.

There were 435 lesions on 39 animals (Table 3.1). The first lesion of the outbreak was observed on 16/2/78. The mean duration of individual lesions was 3.6 weeks and the range 1 to 15 weeks (Table 3.3). The mean duration of disease in individual animals was 7.5 weeks and the range 1 to 19 weeks. The outbreak lasted 28 weeks between the

first sign of disease and the last sign of disease. The period between the first sign of disease in the first animal affected, and the first sign in the last-affected animals was 16 weeks. The last signs of the outbreak were seen in Week 50, 14 weeks after commencement of grazing.

Some disturbance of general health occurred as there were four cases of enteritis in Week 2 and eight of respiratory disease in Weeks 5 and 6.

Six hundred and seventy nine warts developed on 32 animals between Week 40 and Week 90. However, 677 of these were on 31 animals between Weeks 56 and 90.

DISCUSSION

Origin of infection

The first signs of disease occurred 22 weeks after entry to the premises, when 3 animals showed lesions 7 weeks after moving to Accommodation 3. The origin of infection was not proved, but there was the strong possibility of residual infection in a building (Accommodation 3). Manure and litter from a previous batch were not removed before entry of this batch of calves.

Incubation periods

These could not be assessed in this outbreak because origin of infection and timing of actual contact, or exposure, could not be proven.

Spread of infection

First signs of disease occurred when cattle were in two groups of 22 and 21 animals. However, there was a suggestion of earlier relationships influencing spread of disease. Thus during weekly inspections it was noticed that animals previously penned alongside each other in single pens tended to groom one another, to graze together, and range or lie together.

It is postulated that most infection was spread by the tongue of one animal licking another in the process of grooming, as evidence by the number of lesions around the eyes, inside and around the ears, and nostrils but much infection was acquired by handling, crating and rubbing susceptible sites between partition bars.

Lesions

The most significant observation of the outbreak, and of the previously mentioned outbreak (Edwardson and Andrews, 1979), was the presence of most lesions around the eyes of the affected animals (Table 3.2). this does not agree with the work of Ainsworth and Austwick (1959) who stated that lesions occurred most frequently on the head, although they did not specify the ocular region, or with others, who stated lesions occurred particularly on the neck (Ford, 1956; Gentles and O'Sullivan, 1957) but the flanks, rump and limbs might be involved.

In this outbreak, as in a previous outbreak on the same premises (Edwardson and Andrews, 1979), there were few lesions on the neck. Ford (personal communication, 1979) has commented that although the

test animals in his case were at grass, they were collected at intervals for bleeding in a metal-framed crush, with wooden sides, in which they were held by the neck. This exercise may have contributed to spread of disease, particularly on the neck.

CONCLUSIONS

Grooming may be more frequent and thorough among young animals indoors and that, as well as aiding spread, possibly aids healing by removal of scabs and hairs. On the other hand, this facility would be less outdoors in older animals where greater opportunity for rubbing would aid spread of infection and lesions on the same animal, with less fraternisation and grooming from neighbours.

Animal behaviour habits appeared to be influential in the spread of disease especially in youth.

Table 3.1 - Number of ringworm lesions in individual calves

Number of lesions	Number of animals affected
1 - 5	17
6 - 10	7
11 - 15	5
16 - 20	5
21 - 25	2
over 25	3
	39
0	4 (1 casualty) T - 44

T = total

Table 3.2 - Distribution of ringworm lesions and number of animals affected at each site

	Around eyes	Ears	Muzzle Nostrils	Forehead Face	Cheeks Jaws	Poll	Dewlap Neck	Shoulders	Withers	Body Trunk	Legs	Perianal Tail area	Total
No. of lesions	193	42	44	63	16	5	46	10	4	1	1	10	435
Percentage	44	10	10	15	4	1	11	2	1	-	-	2	
No. of animals	32	18	18	16	15	4	16	7	3	1	1	5	39
Percentage	83	46	46	41	33	10	41	18	8	3	3	13	

Table 3.3 - Duration of individual ringworm lesions

Number of lesions	Number of weeks duration
109	1
62	2
86	3
56	4
36	5
24	6
36	7
7	8
4	9
8	10
3	11
-	12
3	13
-	14
1	15

CHAPTER 4

An outbreak of ringworm in Hereford cross Friesian cattle reared indoors and outdoors

INTRODUCTION

Almost concurrently with the outbreak described in chapter 3, a similar outbreak of disease and epizoology occurred in near-white faced cattle, affording some comparisons between two distinctly separate groups of animals. Both outbreaks occurred in cattle wintering indoors and grazing in the summer-autumn period, with disease arising indoors for a similar period.

MATERIALS AND METHODS

The test animals were 40 one-week old Hereford cross Friesian male calves purchased from a dealer.

In Week 7, after single-pen accommodation, they were separated into two groups of 20 animals each, with no contact between groups. In Week 17 they went to pasture together, and in the following seven months occupied seven different pastures. Weekly inspections were made from Week 1 until Week 106.

RESULTS

The first instance of infection occurred in Week 4 and this animal (No. 26) became the most heavily infected with 76 lesions. In Week 12 there were 4 affected animals in Group 1 and 12 in Group 2 (including animal No. 26). In week 17, at turnout to pasture, there were

15 affected in Group 1 and 17 in Group 2. Three other animals became affected at pasture.

Three hundred and seventy lesions were observed on 35 affected animals. The range was 1 to 76 and the mean 10.6. Four animals were excluded from records as casualties. One other animal remained unaffected (Table 4.1).

Lesions were distributed on different sites as shown in Table 4.2. the maximum estimated size of individual lesions varied between 0.6 cm diameter and 15 x 10 cm. Most were 0.5 - 3 cm in diameter. The majority of lesions were annular in shape, few coalesced and few were of irregular shape. The total area of lesions in individual animals varied between 0.6 cm^2 and 364 cm^2.

The duration of individual lesions varied between 1 and 18 weeks with a mean of 4.2 weeks (see Table 4.3) whilst the mean duration of disease was 8 weeks (range 2 - 21 weeks).

The outbreak lasted for 26 weeks from Week 4 until Week 29; from 14 weeks before turnout and 12 weeks at pasture. The first animal (No. 26) was affected in Week 4. The last animals affected were Nos. 1 and 6 in Week 19.

The majority of lesions were depilated, grey, scaly or flaky annular areas of skin. Grey, crusty scabs were noted in 54 lesions involving 19 infected animals. The animals were seen weekly until slaughter in batches between Week 89 and Week 106. Although intermixing with other animals occurred at Week 67, and with other affected animals

between Weeks 17 and 82, there was no further sign of the disease in the test batch.

DISCUSSION

Disease was first recognised in one animal in Week 4 and in a second animal in week 6. The two animals were unrelated and in very separate parts of the accommodation. The origin of disease was probably the separate premises of origin of these calves. The possibility of disease from transport or destination premises cannot be excluded, but the latter were scrupulously cleansed and disinfected before reception.

Incubation periods were estimated as between 3 and 6 weeks. All animals incubated disease indoors, 2 in single-pen accommodation and 33 when the batch was in two groups in open-fronted sheds. There was no evidence of accelerated resolution or suppression of disease when animals were turned out to pasture, as seen in the undermentioned figures.

	Indoors only	Indoors and outdoors	Outdoors only
Number of animals affected and recovered	7	25	3
Number of lesions appearing	160	113	97
Mean duration of lesions from origin (weeks)	3.2	7.2	2.5

Lesions 'indoors' arose and healed indoors. Lesions 'indoors and outdoors' aroseindoors and healed outdoors. Lesions 'outdoors only' arose and healed outdoors.

CONCLUSIONS

No significant differences were revealed in disease manifestation between the white-faced breed of this investigation and the mainly black-faced Friesians of another outbreak concurrent on the same farm. Comparisons relating to siting of lesions and number of lesions are given in Appendix 3.

Spontaneous recovery occurred in this outbreak and there was no recrudescence.

Table 4.1 - Number of ringworm lesions in individual calves

Number of lesions	Number of animals affected
1 - 5	16
6 - 10	8
11 - 15	6
16 - 20	0
21 - 25	2
over 25	3

Table 4.2 - Distribution of ringworm lesions and number of animals affected at each site

	Around eyes	Ears	Muzzle Nostrils	Forehead Face	Cheeks Jaws	Poll	Dewlap Neck	Shoulders	Withers	Body Trunk	Legs	Perianal Tail area	Total
No. of lesions	104	53	16	59	19	13	67	6	6	4	6	17	370
Percentage	28	14	4	16	5	3	18	2	2	1	2	5	
No. of animals	29	20	8	13	11	3	18	5	2	4	2	8	35
Percentage	83	57	23	37	31	9	51	14	6	11	6	23	

Table 4.3 - Duration of individual ringworm lesions

Number of lesions	Number of weeks duration
116	1
36	2
40	3
47	4
19	5
27	6
10	7
34	8
18	9
4	10
4	11
5	12
3	13
1	14
3	15
2	16
1	17
1	18

CHAPTER 5

An outbreak of ringworm in young cattle at pasture

INTRODUCTION

Here an outbreak is described in batch of young Hereford cross Friesian steers reared for beef in 1979, The incidence arose at pasture and subsided before the animals went indoors in the autumn.

MATERIALS AND METHODS

The test animals were initially 40 one-week old Hereford cross Friesian calves, purchased from a dealer. . . Dehorned and castrated by Week 5.

The animals were first accommodated in single pens in a controlled environment house. After castration, dehorning and weaning they were placed in five equal groups in open-fronted sheds.

They were turned out to pasture 19 weeks after purchase. Pasture fields were surrounded by thorn hedges fronted by barbed wire fixed to interval wooden posts. There were two large trees, which because of their shape at base, afforded little or no rubbing place, or area. Test animals were penned away by electrified fences from two other groups of cattle using the same field. The predominant weather, during the outbreak, was cloudy, dull and wet. No antimycotic treatment was applied during the outbreak. No other treatment was administered.

The animals were seen weekly commencing in the week of purchase, 5 days after arrival and weekly recording of general health and incidence from Week 5.

RESULTS

Disease was observed in 8 animals in Week 28, 9 weeks after turnout to pasture. Thirty eight animals were in the batch (2 animals had died before turnout) and 35 became infected.

The initial manifestation indicated a single lesion in 6 animals, 3 on another and 4 on the eighth animal. In the following 3 weeks, 26 other animals became affected, making a total of 34 out of the 38 animals. After a further 5 weeks another animal showed a single lesion. Three other animals did not show any clinical evidence of disease.

The lesions totalled 256 and were produced by 35 affected animals with a mean number of 7.3. The range was 1 to 23 lesions.

Lesions were sited in places previously associated with disease. Twenty nine animals displayed 105 lesions around the eyes; 27 animals showed 61 lesions on the neck. They were discrete, annular, grey and scaly. Many were scabbed and crusty. The lesions persisted between 1 and 14 weeks (mean of 2.7 weeks) (Table 5.3).

The duration of the disease in individual animals ranged from 1 to 15 weeks with a mean of 6.5 weeks.

The whole outbreak lasted 17 weeks from first sign of disease to complete healing with new growth of hair. There was no major disturbance of general health. However, an outbreak of cutaneous papillomas (warts) affected 17 animals between Week 51 and Week 77 and these animals had all previously contracted ringworm.

DISCUSSION

Origin of infection

In early July 1979 there was a collapse of fencing, and failure of electrified fences, with consequent intermixing of the three groups of animals, and despite repairs to fencing, and subsequent separations into original groups, further failures of fencing led to further intermixings. Direct contacts by the test animals with the other groups occurred on or about 24 weeks after the purchase, about 4 weeks before lesions were visible.

The sudden outbreak in 8 animals suggests an exposure to concentrated infection not previously available. This source was possible surviving and residual infection on the 35 Hereford cross Friesian animals which had recovered from disease in 1978 and which were 19 months old at the time of contact.

Manifestation of disease 4 weeks after intermixing with other groups of animals suggested infection as contracted in fraternization, but the possibility of spores on fencing posts could not be discounted. Thus an incubation period of about 4 weeks appeared to be similar to that

of previous ringworm infections on the farm.

The disease was seen to spread rapidly to involve 34 of 35 infected animals in 4 weeks:-

Week	28	-	8	cases	8
Week	29	-	5	cases	13
Week	30	-	18	cases	31
Week	31	-	3	cases	34
Week	36	-	1	case	35

Grooming (licking) and rubbing (scratching) were not seen during weekly inspections though all groups, and members of them, spent time huddled together; but stockmen reported seeing grooming (licking) during periods of intermixing. These activities may explain rapid spread of disease. Three animals remained without visible lesions.

Initial and subsequent lesions were more common on the necks of infected animals than seen in indoor incidence - presumably contracted from rubbing posts. Neck lesions were predominant and numerous in the pasture incidence as described by Ford (1956). It is probable that rubbing and scratching are facilitated on fencing posts, outdoors injury being caused by splinters which may cause the introduction of hairs and spores - thus suggesting that outdoor infection is more likely than that contracted indoors where only flat walls surround the pen.

Durations of lesions ranged from 1 to 4 weeks with a mean of 2.7 weeks. Durations of disease in individual animals ranged from 1 to 15 weeks with a mean of 6.5 weeks.

There was no quick recovery in this outbreak as suggested in general in the literature relating to incidence at pasture. The duration was 17 weeks, similar to the period of 17 weeks recorded by Ford (1956). The period from first affected animal (Week 28) to last affected animal was 9 weeks, but healing and recovery were not completed until 17 weeks (Week 44).

CONCLUSIONS

The appearance of disease 9 weeks after entering pasture leaves no doubt that infection was contracted outdoors. The origin of infection is less clear but appeared to be from recovered animals, with which test animals had contact during straying. The predominant sites of the ringworm were about the eyes and the necks of these animals.

Spontaneous recovery was again a feature of the outbreak with no recurrence or recrudescence after intermixing later. Duration of outbreak was shorter with affected animals than in outbreaks where intermixing occurred after initial appearance of disease.

Table 5.1 - Number of ringworm lesions in individual calves

Number of lesions	Number of animals affected
1 - 5	17
6 - 10	10
11 - 15	4
16 - 20	3
21 - 25	1
over 25	0
	35
none	3 + 2 casualties

Table 5.2 - Distribution of ringworm lesions and number of animals affected at each site

	Around eyes	Ears	Muzzle Nostrils	Forehead Face	Cheeks Jaws	Poll	Dewlap Neck	Shoulders	Withers	Body Trunk	Legs	Perianal Tail area	Total
No. of lesions	105	36	5	13	14	3	61	4	-	2	5	8	256
Percentage	41	14	2	5	5	1	24	2	-	1	2	3	
No. of animals	29	20	3	8	11	3	27	3	-	2	4	4	35
Percentage	83	57	9	23	31	9	77	9	-	6	11	11	

Table 5.3 - Duration of individual ringworm lesions

Number of lesions	Number of weeks duration
130	1
26	2
26	3
26	4
15	5
13	6
7	7
6	8
2	9
-	10
1	11
-	12
1	13
3	14

CHAPTER 6

Epizoology discussion

The epizoology and aetiology of ringworm disease

In this study the epizoology, or incidence and distribution of ringworm in young cattle is recorded from naturally occurring outbreaks. The first incidence occurred in animals housed entirely indoors (Chapter 2). The second and third outbreaks involved animals kept indoors during the autumn and winter but grazing in spring and summer (Chapters 3 and 4). The fourth outbreak took place entirely outside during the summer months (Chapter 5).

Detailed observation and recording of ringworm outbreaks was undertaken at regular weekly intervals during all seasons and over twelve years. Records included the site, size, number and duration of each lesion.

The probable source of infection in the series of four outbreaks was determined in each outbreak. In the trial described in Chapter 2, introduction of infection appeared to be via purchased calves. In another (Chapter 3) the most likely origin was infected buildings as disease arose when there was no direct contact with other animals. In the third outbreak (Chapter 4) infection was very probably from a vendor's herd or dealer's premises. In the fourth case, there was evidence that infection was transmitted from recovered animals to susceptible animals in accidental intermixing outdoors (Chapter 5).

Observations on the calf and beef unit in the period 1977 - 1980 suggested the following sources of infection in 25 outbreaks:-

Purchases from dealers	3 outbreaks
Other animals on the farm	9 outbreaks
Infected buildings and equipment	13 outbreaks

The most obvious source of infection was other infected animals but in the majority of outbreaks, on the major unit, the disease occurred in the fattening and finishing end of the line of accommodation, where cleansing and disinfection were less rigorous and where trips to and from catching crates and weighing machines were more freuqent than elsewhere on the farm - and thereby provided common indirect meeting places for many groups.

In later incidences records revealed that disease occurred in 15 of 45 groups of calves purchased from a dealer in the period 1985 (2), 1986 (5), 1987 (8). This suggests that infection may be contracted not only on vendor's premises but in markets, dealer's premises and dealer's transport. Obviously repeated introductions of infection via purchases thwart control and prevention and make therapy, labour and effort expensive.

The occurrence of ringworm in cattle on the farm under review has been perpetual and judging by purchases via markets and age-old surveys, the disease is nationally widespread. Widespread incidences, morbidity and susceptibility are inter-related factors and depend on several factors including the source of infection, weight of infection which according to Lepper (1972) influences not only the initial incidence but also the extent of disease, and general immunity or resistance, natural or acquired.

The farm outbreaks recorded in earlier pages demonstrated multiple incidence and occurrence which was not influenced by season or surroundings.

Incubation periods are difficult to determine in naturally occurring outbreaks. Dates of exposure to known infected contacts may aid assessment of the period. General experience and observation have indicated 28 days as a common incubation period in naturally occurring outbreaks. Thus consideration in retrospect makes this period valuable in identifying probable sources of infection, as indicated in the section on aetiology.

The morbidity or distribution and occurrence of disease in the groups surveyed in earlier pages reached 95%+ in each case. This appeared to reflect level of infection and the absence of therapy and neglect of other control or preventive measures. The figures are provided in Appendix 1.

Susceptibility to ringworm in cattle may appear to be greater in calves and young stock than in adults. But the latter may often include recovered animals or those with acquired resistance. The susceptibility of the adults is no less than that of the youngsters, according to the literature.

Sick and unthrifty animals are usually considered more susceptible to disease than healthy, thriving ones, but this did not always appear to be the case, and McPherson (1957a) supported this view. Conversely, several workers believed ringworm produced unthriftiness (Pearson and Rankin, 1962; Horrox, 1976). Ford (1956) suggested emaciation might occur in calves where lesions were extensive and Jensen and

Mackey (1971) that growth rates and unthriftiness might be affected by ringworm.

In early outbreaks it was noticeable that sick animals were semi-isolated from neighbours, were not groomed by them and did not partake in rubbing activities - they were also less affected than other members of their group. In one outbreak of ringworm two unaffected animals suffered other diseases.

Although bare patches resulting from pediculosis were found in some animals, there was no evidence that such areas were always, or even often, affected with superimposed ringworm and this confirmed the findings of McPherson (1957a), that lice were not a major influence in the spread of the disease.

In Chapter 3 the outbreak of ringworm was followed by a high level of wart infection (papilloma) on sites of the earlier disease. This was a coincidental occurrence of another infection since there was no statistical correlation. However it is possible that the two infections might have similar predilection sites (Jarrett, 1981; Edwardson, unpublished findings, 1980) or that one predisposes to the other.

Spread of the disease was accentuated by different activities in group fraternisation when animals were grooming one another by licking or tonguing, then the hard papillae of the tongue might bruise the thin skin around the eyes - the commonest site for first lesions.

More vigorous activities could also be responsible for spread by rubbing, scratching, riding and butting. Some semi-enforced rubbing

and scratching may occur in handling animals for various operations in catching crates and weighing machines. Likewise feeding from troughs out-with accommodation pens, between bars, especially bars sloping or positioned at different angles, could allow mild trauma and inlet of infection. Similarly there appeared to be more lesions on the necks of animals feeding between bars and animals outdoors having rubbing posts such as fencing posts and trees, than animals indoors in pens having few projections and mainly flat walls (Appendix 3).

Additionally, spread may be enhanced by crowding, intermixing of groups, large groups and by mixing susceptible and recovered groups - such exercises probably led to the build-up of level of infection providing more infective doses, and a greater contamination of the environment.

Lesions have been observed on animals following the lines of a grooming tool, thus equipment might provide the necessary bruising for establishment of spores. These cases probably received infection from dust removed in preparing premises and animals for exhibition. Also emphasised was the probability of disease being introduced by splinters of wood or metal from fencing posts, trees and equipment. Khan (1980 - personal communication) quoted a case of tetanus contracted via a splinter from the rails of a race-course, thus underlining the importance of trauma in the introduction of infections. Jillson and Buckley (1951) confirmed that injuries predisposed to disease and that initial lesions were at the site of trauma.

Fraser (1972) indicated that young cattle spent 52 minutes each day in grooming by rubbing and scratching and Hafez (1962) stated the

head, neck and shoulders were the main areas licked in grooming, and these were the commonest sites for ringworm.

Spread of disease was possibly enhanced, aggravated and prolonged by modern husbandry including infrequent removal of litter and manure between batches, minimal hygiene and absence of disinfection procedures. Open partitions, crowding, intermixing of clean and affected groups spread disease all add to the weight of infection.

The following agents and factors were summarised in Chapter 2 and considered in assessing and exploring spread of infection:-

1. Tongues; licking, sucking and grooming.
2. Direct contacts; infected animals, rubbing, riding, butting.
3. Rubbing posts and places; buildings, pens, food hoppers, water bowls, partition bars, catching crates and tools.
4. Indirect contacts; handling, dehorning operations, litter and vermin, plus sponging, brushing etc. in connection with use of therapeutic agents.

In that year of observation (1977), factors 1 and 2 were considered the most important.

There are other factors which may be considered such as straying of animals about a unit, contact 'over the hedge' with cattle of another owner and contact with other species. The sites of infection frequently indicate the major mode of spread and thus contribute to any plans of prevention which may be in hand or mind. Relevant charts and

tables are appended to illustrate the points above (Appendices 1-5). The most puzzling site of spread is the perianal area and tail, not uncommon in yearlings.

Duration and disease

Duration of each and every lesion has been reported in all recorded outbreaks and has shown that many lesions were short-lived, being present for only 1 to 2 weeks. The mean duration as 3.7 weeks (4) with a range of 1 to 18 weeks. Some lesions healed before others appeared and all seemed to have individual existences, expanding peripherally until presumably resistance or acquired immunity overcame the advances.

Duration of the disease in individual animals averaged 8.2 weeks in many outbreaks and was important when considering control measures.

The duration of disease in outbreaks might vary due to many influences such as intermixing of groups of animals and multiple movements or changes of accommodation, but in uninterrupted straight cases the mean was 18 weeks. In mixed batch outbreaks the period lasted 28 to 33 weeks. Appendix 1 demonstrates all facets of duration of ringworm in young cattle.

Infection recurrences and immunity

This subject includes some of the most important findings and inferences of the investigations reported in this thesis. The orthodox definition of spontaneous recovery from ringworm is the unaided resolution

of the disease and the natural return to health (without external influence).

In the literature (Ford, 1956; Ainsworth and Austwick, 1955) the assumptions and inferences are that spontaneous recovery occurred principally when cattle were turned out to pasture and that recovery was then a rapid process. The current investigations have shown that spontaneous recovery occurred in cattle indoors and outdoors and that turning-out had no very obvious influence on recovery.

Figures in Appendix 1 show a general average for duration of disease in lesions, animals and outbreaks. The spontaneous recovery factor appears to have been ignored in many trials and tests of drugs used in therapy. O'Brien and Sellers (1958), however, appear among the few, stating that in their trials spontaneous healing of treated and untreated animals occurred 150 days after the infection was first noted and 90 days after treatments were applied.

Spontaneous recovery is not camouflaged by inherent immunity because current observations have shown that up to 95% of animals develop clinical infection when exposed. Spontaneous recovery indicates the acquisition of immunity as disease progresses. From the observations in this work, immunity lasts at least one year after an outbreak. Thus spontaneous recovery is a very important factor to be considered in the therapy and control of disease. The treatment of ringworm in cattle is often initiated when the disease is well established and peaking or waning. Treatment is then credited with success when really it is due to spontaneous recovery. Thus the efficacy of a drug cannot be

measured unless age of disease is known when the treatment is initiated and comparable controls are used in each trial. A suggested code of practice for the testing of drugs is given in Chapter 11. Appendix 2 demonstrates the findings in many drug trials reported in the literature where age of disease prior to treatment is not given and where there are inadequate fair or equal comparisons between control and trial groups of animals.

Inherent immunity to ringworm in young cattle appears to be minimal and would rarely appear to rise above 5% in any outbreak. It is possible in some cases of low and negligible incidence of disease that low level of infection, or effective early therapy, may suggest immunity.

CHAPTER 7

Observations on the treatment of ringworm with Griseofulvin

INTRODUCTION

The efficacy of griseofulvin therapy in an outbreak of ringworm in calves in 1980 was tested on a farm not previously studied. The outbreak had resulted in infection of the farm manager.

MATERIALS AND METHODS

Test animals

The test animals were Hereford cross Friesian male calves purchased in markets at one-week old on Week 1 and Week 3. Transport from market to farm was by the owner's horse box.

Three or 4 calves were accommodated in each of several loose boxes near to the farm manager's house, where they were fed milk substitute, hay and coarse mix. They were weaned or moved to a barn or covered yard in Week 15 when aged 12 to 15 weeks. The barn accommodated four groups of animals on each side of a central concrete passageway. The earth floors of the pens were below ground level and the sides of the building were open above $2\frac{1}{2}$ metres. The pens were open-fronted and open-sided with spaces measuring 37 cm between the horizontal metal bars of the partitions.

Feed after weaning was hay ad libitum and 2 kg of concentrates daily, with added mineral mixture. Castration and dehorning took place in Week 22. The weigh machine and a metal catching crate were used

commonly on different farms in the same occupation.

The 10 treated animals occupied Pen L3 (Group 1) (Fig. 7.1). The 9 control/untreated animals occupied Pen L4 (Group 2). The animals of both groups did not go to pasture before recovery from ringworm. Natural light was abundant and ventilation was natural and unrestricted in the barn-type accommodation. During the trial no major movements took place involving the test and control animals and pens. Two animals (B16 and 1095) were added to the control group in Week 20, 10 days before the trial commenced and they were removed in Week 28 to other premises in the same occupation, but it was possible to complete the necessary inspections of these.

No treatment had been applied to the trial animals prior to the test under report. Treatment commenced in Week 21 in the test group of 10 animals with oral application of the first dose at the rate of 7.5 mg/kg body weight (BW). Individual doses were based on girth circumference weights. Thereafter, for each of 6 days, a similar dose was incorporated in concentrate feed for each of the 10 animals. The manager reported that the mix was eaten evenly by all animals.

No special preventive measures were adopted on the premises. Manure and litter were removed annually from the floor. Phenol-based disinfectant was sprayed annually.

The animals were routinely inspected and consequently were seen on Week 20 and Week 21 when evidence of ringworm was noted in test and control groups. The manager reported seeing lesions first in Week 19. Test and treatment commenced on Week 21 and inspections continued weekly

until Week 35, following recovery from disease in both groups (see Table 7.1). All animals were examined individually in a crate. All lesions were recorded. Samples of hair and skin scrapings were taken from lesions of several animals and submitted for laboratory examination.

The lesions were described as new, healing, dead and healed lesions based on the following description. The early sign of a ringworm lesion was thinning of hair, followed by grey scaling of the underlying skin. Shedding of the remaining hair from the lesion followed within a week and its outline was then revealed. Thereafter there was peripheral expansion, with retention of shape in most cases. Coalescence was relatively rare (but was recorded in 4 infected animals). Grey scaling areas were the general feature rather than distinctly crusty lesions. Swelling and hardening of infected areas were rare (but were observed in 5 animals).

During the healing of lesions there was a halt to peripheral growth of the lesion. The area took on a dull appearance followed by disappearing of scales and the onset of pink and white colouration. Hair growth usually commenced at the centre of a lesion and usually thereafter quickly covered the area. Hair growth was not usually sufficient to mask a lesion until at least a week after appearance of new hair. Black hair grew in the previously black areas, with no grey hair or bare patches persisting following healing.

RESULTS

Lesions were seen in both groups in Week 19 by the farm manager. When the test began in Week 21 all animals (10) in the treated group were affected but only 7 in the control group were affected. The remaining 2 became affected in Week 26 and Week 27. Trichophyton verrucosum was cultured from hair and skin samples. The number of lesions on each animal and new lesions are shown in Table 7.2. The commonest site of lesions was around the eyes and other common sites were ears and shoulders. In one control animal numerous lesions were seen in the perianal region and the tail area (Appendix 3). The lesions were thick and scabby. Healing lesions on treated animals showed disintegration and flaking, rather than peeling-off of whole scabs or crusts.

The duration of disease in individual lesions varied between 2 and 8 weeks, with a mean of 3.4 in Group 1 and between 1 and 14 weeks with a mean of 5 weeks in Group 2.

In individual animals the duration of disease was between 3 and 9 weeks in the test group (mean 7 weeks) and between 5 and 16 weeks in the control group (mean 10 weeks) (Table 7.3).

The duration of the disease in the test group was 9 weeks, namely from Week 19 to Week 27. The duration of disease in the control group was 16 weeks from Week 19 to Week 34.

DISCUSSION

The origin of infection appeared to be in the buildings and equipment and not from the premises of origin of purchased calves. Several animals developed signs at about the same time. The first signs of disease were observed in Week 19, 4 weeks after changing accommodation. There had been no sign of infection in the rearing boxes, or in contact pens in the barn, at the material time.

Both groups were moved into the barn on Week 15 and if the source of infection was in the building then the incubation period was about 29 days.

No evidence of pruritus was observed during inspection periods and it is therefore assumed that spread of infection, and extension of lesions, came about by direct contact with infected animals during grooming, when rubbing or butting one another, or when rubbing on dividing metal bars or partitions.

It was noted that 2 control group animals did not become visibly affected until Week 26 and Week 27 respectively, but the duration of disease in these was comparable with that of animals affected at an earlier period. On one animal in the control group there were many lesions in the perianal area, on and under the tail and downwards, on the posterior aspect of the legs. Lesions were seen on or under the tail and around the anus in 6 animals (2 test animals and 4 control animals). Lesions were seen on the legs - mainly on the fetlocks of the hind legs - in 9 animals (3 in test group and 6 in control group).

CONCLUSIONS

The drug used in this trial was effective in reducing the duration of disease in the treated group to 9 weeks, whilst the disease lasted for 16 weeks in the control animals. There were fewer lesions in the test group - 91 (10 animals) than in the control animals - 120 (9 animals). Duration of disease in individual animals averages 7 weeks, with a range of 3 to 9 weeks in the test animals. In the control group there was an average duration of 9.9 weeks, with a range of 5 to 16 weeks (Tables 7.2 and 7.3).

Figure 7.1 Griseofulvin trial.

Incidence and morbidity in treated (□)

and control (●) groups

Figure 7.1

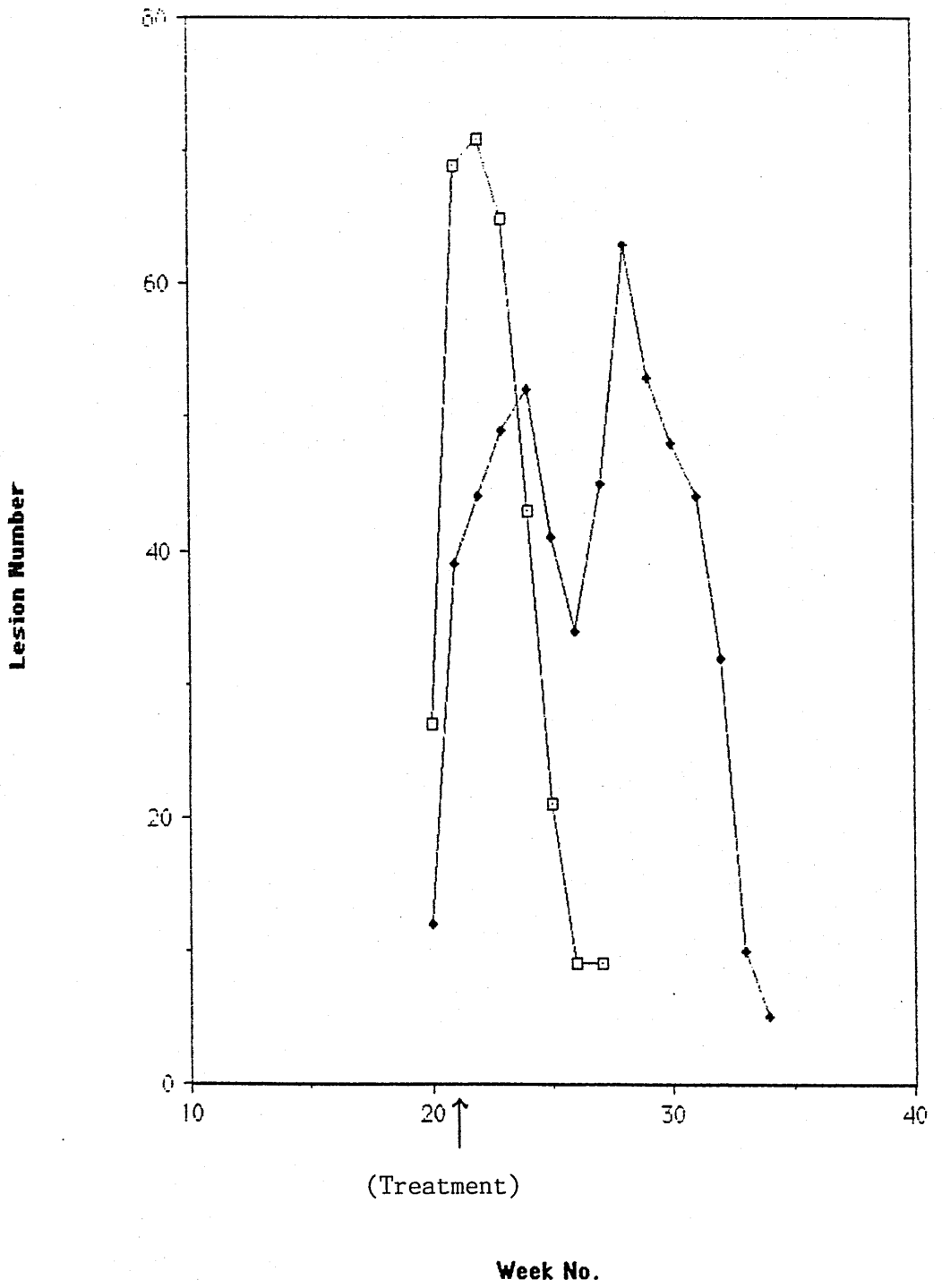

Table 7.1 - History of animals and outbreak

Week No.	Date	Animals numbers T	Animals numbers C	Activity	Movement (origin)	Movement (destination)	Disease incidence affected animals T	Disease incidence affected animals C
1	13/08/79	6 week-old calves		purchase 14/08	market	loose boxes		
2	20/08/79	7 week-old calves		purchase 15/08	market	loose boxes		
3	27/08/79	7 week-old calves		purchase 29/08	market	loose boxes		
4	03/09/79							
5	10/09/79							
6	17/09/79							
7	24/09/79							
8	01/10/79							
9	08/10/79							
10	15/10/79							
11	22/10/79							
12	29/10/79							
13	05/10/79							
14	12/11/79							
15	19/11/79				loose boxes 16/11	barn		
16	26/11/79							
17	03/12/79							
18	10/12/79							
19	17/12/79	10	7	first signs RW both groups 15/12			+	+
20	24/12/79	10	9	2 animals added to control group (B16 and 1095)			8	5
21	31/12/79	10	9	Treatment			10	7
22	07/01/80						10	7
23	14/01/80						10	7
24	21/01/80						8	7
25	28/01/80						8	7
26	04/02/80						5	6
27	11/02/80			last signs T group			2	6
28	18/02/80			2 animals removed from control group (B16 & 1095)				5
29	25/02/80							5
30	03/03/80							5
31	10/03/80							5
32	17/03/80							5
33	24/03/80							4
34	31/03/80			last signs C group				
35	07/04/80							
36	14/04/80							
37	21/04/80							
38	28/04/80							

Period of observation 20/12/79 - 28/04/80 = 19 weeks

Period of treatment 31/12/79 - 06/01/80

T = treated animals

C = control animals

Table 7.2 - Animals and disease incidence

Week no.	Date	Affected (actual)		Affected (cumulative)		Active		New (actual)		New (cumulative)	
		T	C	T	C	T	C	T	C	T	C
17	03/12/79										
18	10/12/79										
19	17/12/79	+	+	+	+	+	+	+	+	+	+
20	24/12/79	8	5	8	5	27	12	27	13	27	13
21	31/12/79	10	7	10	7	69	39	42	27	69	40
22	07/01/80	10	7			71	44	14	13	83	53
23	14/01/80	10	7			65	49	7	7	93	60
24	21/01/80	8	7			43	52	1	7	91	67
25	28/01/80	8	7			21	41		3		70
26	04/02/80	5	6		8	9	34		4		74
27	11/02/80	2	6		9	9	45		17		91
28	19/02/80		6				63		28		119
29	25/02/80		5				53		1		120
30	03/03/80		5				48				
31	10/03/80		5				44				
32	17/03/80		5				32				
33	24/03/80		4				10				
34	31/03/80		2				5				
35	07/04/80										
36	14/04/80										
37	21/04/80										
38	28/04/80										

	Test	Control
Total animals	10	9
Total animals affected	10	9
Total animals non-affected	0	0
Total lesions	91	120

T = test group
C = control group

Table 7.3 - Duration of disease

Animal number	Duration of disease (weeks)	Lesions	Duration of lesions - weeks 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
TEST GROUP																				
1	9	6				3	1		1	1										
2	3	2	1		1															
3	7	6		1		3		2												
4	9	6			1	1	3	1												
5	8	2					1	1												
6	5	5	3		2															
7	7	21	5	8	3	3	2													
8	6	10	3	4	1		1	1												
9	8	18	1	1	5	7	2	2												
10	8	15	1	4	2	5		1	2											
Total		91	14	18	15	22	10	8	3	1										
Range	3-9	2-21	1-8																	
Mean	7.0	9.1	3.4																	
CONTROL GROUP																				
1	5	4	2		1		1													
2	7	4				1		3												
3	13	17				3	4	1	3	1	1	1	2	1						
4	16	4								1			1	1		1				
5	8	12	3	2	1	2	2	1	1											
6	7	8				3	5													
7	14	13				1	5	4	1		1	1								
8	9	40	2	9		7	5	15	2											
9	10	18	3	1	4	4	3	2		1										
Total		120	10	12	6	21	25	26	7	3	2	2	3	2		1				
Range	5-16	4-40	1-14																	
Mean	9.9	13.3	5.0																	

CHAPTER 8

Topical Natamycin in the treatment of ringworm in young cattle

INTRODUCTION

The use of natamycin in treatment of cattle with ringworm has been reported by Spanoghe and Oldenkamp (1977). However the trials did not record in detail disease in individual animals, especially prior to treatment date. The present study was undertaken to examine critically and clinically an incidence of naturally occurring ringworm in calves using natamycin as a topical treatment.

MATERIALS AND METHODS

The calves in the study were male Friesian animals purchased as one-week old, on 26th November 1980 (Week 1) and 3rd December 1980 (Week 2). They were contract reared away from the trial premises on a farm where ringworm first appeared on 14th January 1981 (Week 8) in 2 animals (Nos. 11 and 21). All were weaned and moved to hardening-off accommodation on the finishing farm on 30th January 1981 (Week 11), where they were divided into two groups.

The calves were first seen by the author in Week 11 when animal No. 21 was affected in Group 1 (treated) and animal No. 11 was affected in Group 2 (control) (Table 8.1). In Week 14 both groups were moved to adjacent fattening area accommodation - in open-fronted sheds and penned back from small open yards. The two groups were separated by a partition to prevent physical contact and there was no contact with other cattle.

Application of the drug natamycin was by two sprayings of the head, neck and back of each animal in the test group (Fig. 8.1). Particular note was made of age of lesions. Natamycin (Mycofarm) was prepared in suspension - 1 g natamycin in 10 g of powder suspended in 10 l of water, sufficient for 15 calves 6 to 9 months old - and was applied only to Group 1 animals, strictly in accordance with the data sheet and veterinary advice. The farm was in a hard water area and thus distilled water was used for the first suspension of the drug, and filtered clean rain water for the second operation. The first spraying was in Week 16.

The test animals were under cover when they were treated in Week 16 and were separated 2 at a time for spraying between two gates in the open yard in front of their pen. The outside exposure was about 2 min for each animal. The second treatment was 5 days layer. On the latter occasion the inside of the test animals' accommodation was also sprayed with the drug suspension. All apparatus and equipment used in the operations was either new, or thoroughly cleansed before use. Care was taken to spray thoroughly all lesions and all vulnerable sites - head, neck and back as well as inside ear lobes, under jaws and around eyes. The procedure was watched by a veterinary surgeon from Mycofarm and further spraying was undertaken if required. Two sprayings were considered by the company veterinarian to be adequate for the trial. All animals were identified by large-number ear tags.

Definitions of new lesions, healing, dead lesions and healed lesions are based on the criteria used previously (Chapter 7). The number,

age, size and site of lesions were recorded weekly from the onset of disease until complete recovery, as well as prior to each drug application. There was no inter-current disease and no other therapy was applied. All the animals were kept intensively and no pasture was involved. All animals were weighed in Weeks 12, 17, 23, 30 and 40.

RESULTS

All animals in each group showed clinical evidence of disease by Week 18. The duration of the outbreak in the treated cattle was 22 weeks - 8 weeks before treatment and 14 weeks after treatment. In the control group disease duration was 21 weeks - 8 weeks before treatment date (14th January, 1981) and 13 weeks after treatment date (10th June, 1981) (see Table 8.1).

First lesions were seen by a stockman in Week 8, with 3 lesions on animal No. 21 and 1 on animal No. 11. In Group 1, 12 animals were visibly affected when treatment began in Week 16. One animal, No. 21 had recovered and 5 remaining animals became visibly affected in the 2 weeks following treatment. In Group 2, 12 animals were affected on the treatment date and 6 others showed lesions in the ensuing 2 weeks.

Details of incidence and spread are shown in Table 8.2. The spread of disease in both groups was similar but, especially in the treated group, appears to have been aggravated by certain factors. Handling and crating of animals for treatment and sampling and weighing may have contributed to the high infection level. Both groups made 5

visits to the weighing machine. Environmental infection of long standing may also have been responsible for re-infections in the treated group.

The duration of disease in individual animals in the test group ranged between 4 and 13 weeks with a mean of 9 weeks. In the control group, disease in individual animals ranged between 3 and 14 weeks with a mean of 8 weeks (see Table 8.3). Duration and ages of lesions for each group are given in Tables 8.4 and 8.5.

The sites of lesions in the two groups of animals are give in Appendix 3. The number of lesions in Group 1 was 340 (range 3 to 72), with a mean of 19. In Group 2 240 lesions occurred, ranging from 1 to 50 with a mean of 13. New lesions occurring week by week in each group are given in Table 8.2

General good health remained in all but 1 animal, which became unthrifty, possibly from copper deficiency.

DISCUSSION

The appearance of disease in the overall outbreak was 8 weeks after purchase on contract-rearing premises. The initial origin of infection was, therefore, probably from the farm itself. The incubation period could not be assessed on the available evidence.

In the trial by Spanoghe and Oldenkamp (1977) 95% of animals showed recovery or marked improvement in 11 to 12 weeks but this evidence of duration of disease is incomplete - the period of clinical manifestation is not given from first signs to complete healing.

In the present study disease existed for 10 weeks before treatment and 12 weeks afterwards in the treated group. In the controls there was disease for 10 weeks before the treatment date and for 11 weeks after that date.

CONCLUSIONS

Treatment with natamycin of 18 test animals did not shorten the duration of clinical ringworm when compared with the duration in 18 control animals (Table 8.1). Close inspection of the various comparable details of incidence in test and control animals revealed no evidence in favour of therapy - involving number of lesions, duration of disease in lesions, animals and groups.

Table 8.1 - The number and duration of ringworm lesions following use of natamycin

	Test animals	Control animals
Number of lesions	340	240
Mean	18.9	13.3
Range	3 - 72	1 - 50
Duration of lesions		
Mean	3.9 weeks	3.6 weeks
Range	1 - 13 weeks	1 - 11 weeks
Duration in animals		
Mean	8.9 weeks	8.0 weeks
Range	4 - 13 weeks	3 - 14 weeks
Duration of outbreak	22 weeks	21 weeks

(see also Tables 8.2, 8.3 and 8.4).

More detailed work on different premises would appear desirable to assess further the clinical value of the drug. However it is important to ensure false credit is not attributed to drugs when it is due to spontaneous recovery (McPherson, 1959b).

Figure 8.1 Natamycin trial

Incidence and morbidity in treated (□)

and control (●) groups

Figure 8.1

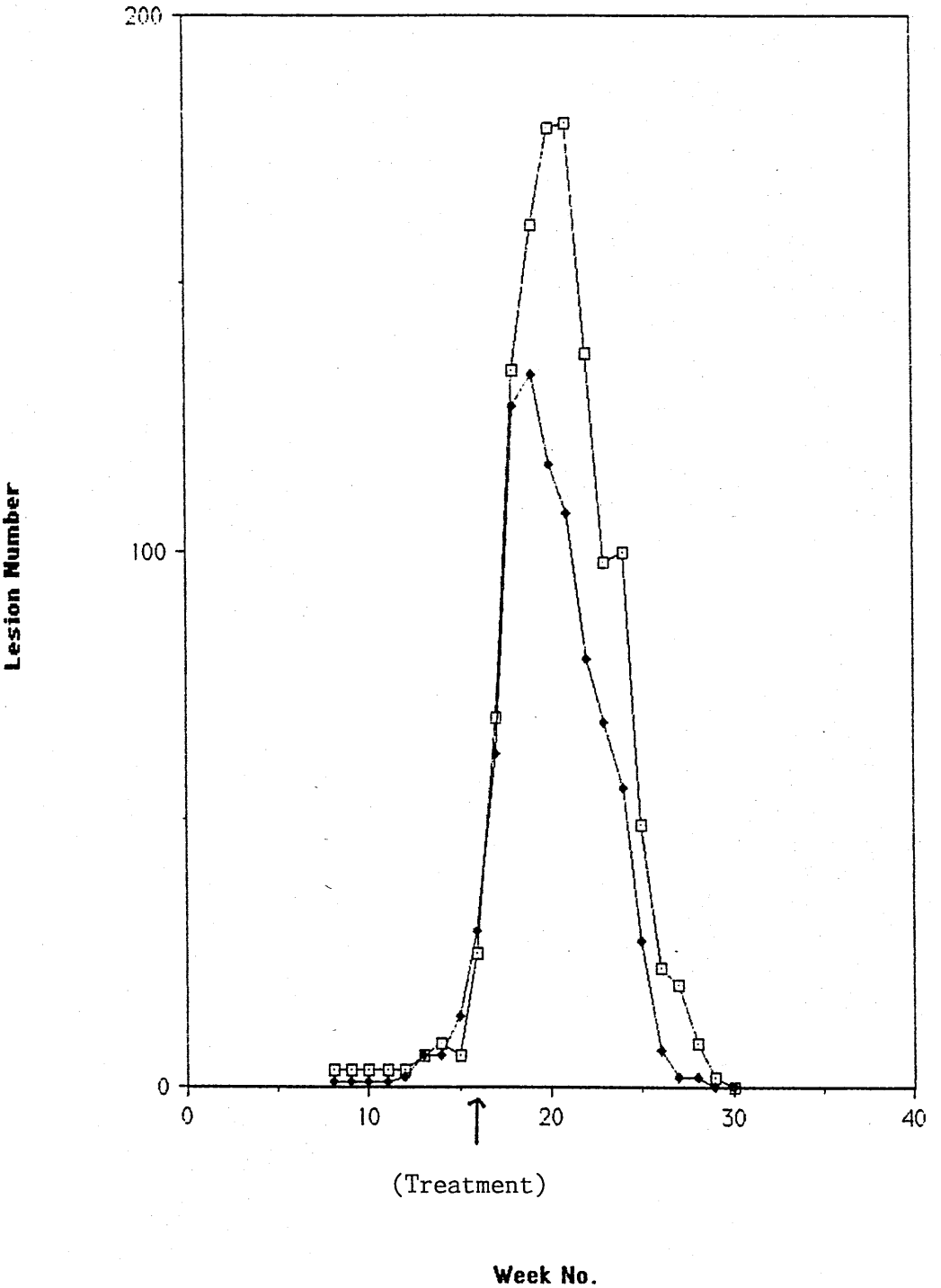

Table 8.2 - History of animals and outbreak

Week no.	Date	Animals numbers T	Animals numbers C	Activity	Movement (origin)	Movement (destination)	Disease incidence affected animals T	Disease incidence affected animals C
1	26/11/80	20 calves		purchase	dealer intermixed	rearer		
2	03/12/80	20 calves		purchase	dealer intermixed	rearer		
3	10/12/80							
4	17/12/80							
5	24/12/80							
6	31/12/80							
7	07/01/81							
8	14/01/81			first signs ringworm			1	1
9	21/01/81						1	1
10	28/01/81	36 calves (intermixed)			rearer	hardening-off yards on trial grounds	1	1
					random division into two equal groups			
11	04/02/81						1	1
12	11/02/81						1	2
13	18/02/81						2	3
14	25/02/81				H O yards	fattening pens	4	3
15	04/03/81						8	7
16	11/03/81			Treatment 11/03			12	12
				Treatment 16/03				
17	18/03/81						14	16
18	25/03/81						17	18
19	01/03/81						16	16
20	08/03/81						14	14
21	15/04/81						13	12
22	22/04/81						13	11
23	29/04/81						12	8
24	06/05/81						7	7
25	13/05/81						7	3
26	20/05/81						5	2
27	27/05/81						4	2
28	03/06/81			last signs ringworm C group				
29	10/06/81			last signs ringworm T group			2	
30	17/06/81							

Period of observation 04/02/81 - 28/01/81 - 08/07/81 = 22+ weeks

Period of treatment 11/03/81 - 16/03/81

T = treated animals

C = control animals

Table 8.3 Animals and Disease Incidence

Week No.	Date	Affected Animals (Actual)		Affected Animals (Cumulative)		Active Lesions		New Lesions (Actual)		New Lesions (Cumulative)	
		T	C	T	C	T	C	T	C	T	C
1	26/11/80										
2	03/12/80										
3	10/12/80										
4	17/12/80										
5	24/12/80										
6	31/12/80										
7	07/01/81										
8	14/01/81	1	1	1	1	3	1	3	1	3	1
9	21/01/81	1	1			3	1				
10	28/01/81	1	1			3	1				
11	04/02/81	1	1			3	1				
12	11/02/81	1	2		2	3	2		1		2
13	18/02/81	2	3	2	3	6	6	3	4	6	6
14	25/02/81	4	3	4		8	6	3	0	9	6
15	04/03/81	8	7	9	7	6	13	10	7	19	13
16	11/03/81	12	12	13	14	25	29	12	19	31	32
17	18/03/81	14	16	15	17	69	62	44	34	75	66
18	25/03/81	17	18	18	18	134	127	67	65	142	131
19	01/04/81	16	16			161	133	46	32	188	163
20	08/04/81	14	14			179	116	58	28	246	191
21	15/04/81	13	12			180	107	41	24	287	215
22	22/04/81	13	11			137	80	10	9	297	224
23	29/04/81	12	8			98	68	8	7	305	231
24	06/05/81	7	7			100	56	22	8	327	239
25	13/05/81	7	3			49	27	4	1	331	240
26	20/05/81	5	2			22	7	4	0	335	
27	27/05/81	4	2			19	2	3	0	338	
28	03/06/81	3	2			8	2	2	0	340	
29	10/06/81	2	0			2	0	0	0		
30	17/06/81	0	0			0	0	0	0		

*Casualties excluded.

	Test	Control
Total Animals	18	18
Total Animals Affected	18	18
Total Animals Non-Affected	0	0
Total Lesions	340	240

T = Treated Animals
C = Control Animals

Table 8.4 - Duration of disease in test group

Animal number	Duration of disease (weeks)	Lesions	Duration of lesions - weeks 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
TEST GROUP																				
1	7	32	2	5	10	8	6	1												
2	10	24	7	4	2	4	3		2		1	1								
3	9	3	2										1							
4	4	4		2	1	1														
5	11	5	1	1						1	1		1							
6	12	40	16	4	2	101		2	2	3										
7	13	45	9	9	3	5	1	4	8	3	2				1					
8	4	5			1	4														
9	5	5		1			1	3												
10	7	3			1		1			1										
11	12	11	3	2		2	2	2												
12	11	33	3	7	3	1	9	7	3											
13	10	72	5	8	11	18	9	10	9	1	1									
14	12	39	20	3		4	2	4		1	3	1	1							
15	9	4	1		1		1		1											
16	7	4	1		1		2													
17	9	7	2	2			1	1		1										
18	7	4	1	1		1	1													
Total		340	73	49	36	58	40	34	25	11	8	2	3		1					
Range	4-13	3-72	1-13																	
Mean	8.9	18.9	3.9																	

Table 8.5 - Duration of disease in control group

Animal number	Duration of disease (weeks)	Lesions	Duration of lesions - weeks 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
CONTROL GROUP																				
1	6	2			1			1												
2	7	6	2			2		1	1											
3	4	10		1	7	2														
4	8	18	2	2	1		4	7	2											
5	8	28	7	8	8	3			1	1										
6	10	13		2	3	2	2	1	1	1	1									
7	11	2			1					1										
8	14	18	4	2	2	3	3	1				1	1							
9	5	10	2	1	3	4														
10	8	15	6	3	2	3	1													
11	12	50	5	7	9	9	3	13	2	1	1									
12	3	1			1															
13	9	11	5	1		1	2		1		1									
14	7	6	3				1		2											
15	9	24	11	3	4			4		1	1									
16	6	10	2	1	6			1												
17	8	14	2	4	3	2	2	1												
18	8	2			1				1											
Total		240	51	35	52	31	18	30	11	5	4	1	2							
Range	3-14	1-50	1-11																	
Mean	8.0	13.3	3.6																	

CHAPTER 9

The controlled testing of topically applied Enilconazole for the treatment of ringworm in young cattle

INTRODUCTION

Enilconazole is known to be effective against fungal infections including ringworm and was licensed in 1982 for use in cattle. Prior to its introduction, a trial was undertaken at NAC Agricultural Unit to determine its efficacy.

MATERIALS AND METHODS

The test and control groups consisted of 37 animals, divided into two similar size groups housed in adjoining pens of the same building. Disease in the animals was recorded at weekly visits (Tables 9.1 and 9.2).

The animals of each group were all purchased together as week-old calves in September (Week 1). All cattle were Friesian bulls and reared for cereal bull beef purposes. There were 19 animals in the test group and 18 animals in the control group.

Treatment commenced on 1st March, 1982 (Week 23) when affected animals in the test group were sponged on affected areas with 0.2% solution of concentrate enilconazole in warm water in accordance with data sheet instructions. All the animals in the treated group were given a whole-body spraying with the prescribed mixture on 1st March. Infected controlswere again sponged with 0.2% enilconazole consecutively on 5th March, 8th March and 12th March 1982. A second treatment of the whole group was performed on 15th March.

The treatment regime was observed and approved by the company due to market the product.

Scrapings were taken from lesions before the commencement of treatment, on 26th February (Week 22) and during treatment on 5th March (Week 23) and 12th March (Week 24), and subjected to microscopical and cultural examinations.

RESULTS

Disease was first observed (Fig. 9.1) on 15th February (Week 21) and could have been up to 14 days old as no ringworm was observed at the previous inspection on 1st February (Week 19). At that time 3 animals were infected in each group. The following week (Week 22) another animal was infected in the treatment group and 2 in the control. At the time of treatment (Week 23) 5 animals were infected in each group. Infection in 2 treated calves cleared up during the application of enilconazole but 3 controls also became clear of infection,

Subsequently infection did follow a similar pattern in each group. However the number of lesions in each group was variable (Tables 9.3,9.4). The treatment group totalled 684 (mean 40.2) and the control group developed 205 (mean 14)(Table 9.2). All areas tended to show more lesions and this was particularly apparent around the eyes, ears, muzzle and nostril, forehead and face, cheeks and jaws, dewlap and neck, shoulders and perianal area. Duration of disease varied from 1 to 16 weeks in the enilconazole group and 1 to 15 weeks in the

controls. However, the duration on individual animals was greater in the treated group, 9.1 weeks v. 8.2 weeks. The outbreak lasted longer (19 weeks) in those treated than the controls (17 weeks).

Culture of the samples from enilconazole treated and control animals showed Trichophyton verrucosum to be present before and during treatment. This suggested the infection was still viable.

DISCUSSION

The reason for this outbreak of ringworm is difficult to determine. It was obviously not due to calves bringing in latent infection. However it did occur 3 weeks after movement from one accommodation to the other. It would seem both groups had a common source of infection as it broke out almost simultaneously. Once disease occurred it was probably spread by contact as well as routine handling of the animals for weighing etc.

According to the records in Table 9.2, the spread of disease was the same in each group until 3 weeks after commencement of treatment when the incidence soared and the number of lesions, particularly in the test group, made a steep rise. The peak of incidence occurred in Week 29 in both groups.

In this particular trial the use of enilconazole did not have an effect on either the duration of lesions or infection of the animals. In addition, it did not stop spread of ringworm in the test group. In fact significantly more lesions occurred in the treated group as

opposed to those not receiving therapy. It would appear that most new lesions occurred at Week 26 and Week 29. It would seem to be more than coincidence that in the first case this was 3 weeks after all the cattle had been initially sprayed with 0.2% enilconazole and in the second case it was 4 weeks after the second spray. The degree of infective challenge cannot be estimated but it would be a working hypothesis to suggest it was similar in each group. It thus seems likely that disease was spread by the treatment regime.

The sponging of the lesions followed by spraying might well have helped to spread the infection over the animal's body and thereby also made it more likely to be contracted by other cattle.

In the initial phase of treatment the lesions in some cattle resolved. This might have been credited to the efficacy of the compound. However, similar recovery, in this case spontaneously, was also seen in some of the control animals. Thus it seemed that topical enilconazole did not prevent latent infection emerging and this could possibly have been due to the lack of penetration of the hair and skin.

It could perhaps be argued that one reason enilconazole was not effective in this trial was because treatment was delayed for up to 2 weeks as 1 weeks's observation was missed. This could have allowed the build-up of infection on the animals and its transfer to others within the group. It is thus possible, if each lesion were treated as it arose, that a different result might have been obtained.

The need for strict practice of hygiene when treating lesions topically by sponging might also be a factor influencing spread. In

this case the same sponge was used to treat each lesion on the infected animals. Topical spraying, provided it can penetrate the lesions, might be more conducive to stopping spread of ringworm once present on an animal.

CONCLUSION

Although other factors would have aided spread of infection in this trial when using enilconazole, the application of the drug via the physical removal of scabs and crusts was probably the most important factor in the spread and upsurge of disease in the treated animals.

Figure 9.1 Enilconazole trial

Incidence and morbidity in treated (□)

and control (●) groups

Figure 9.1

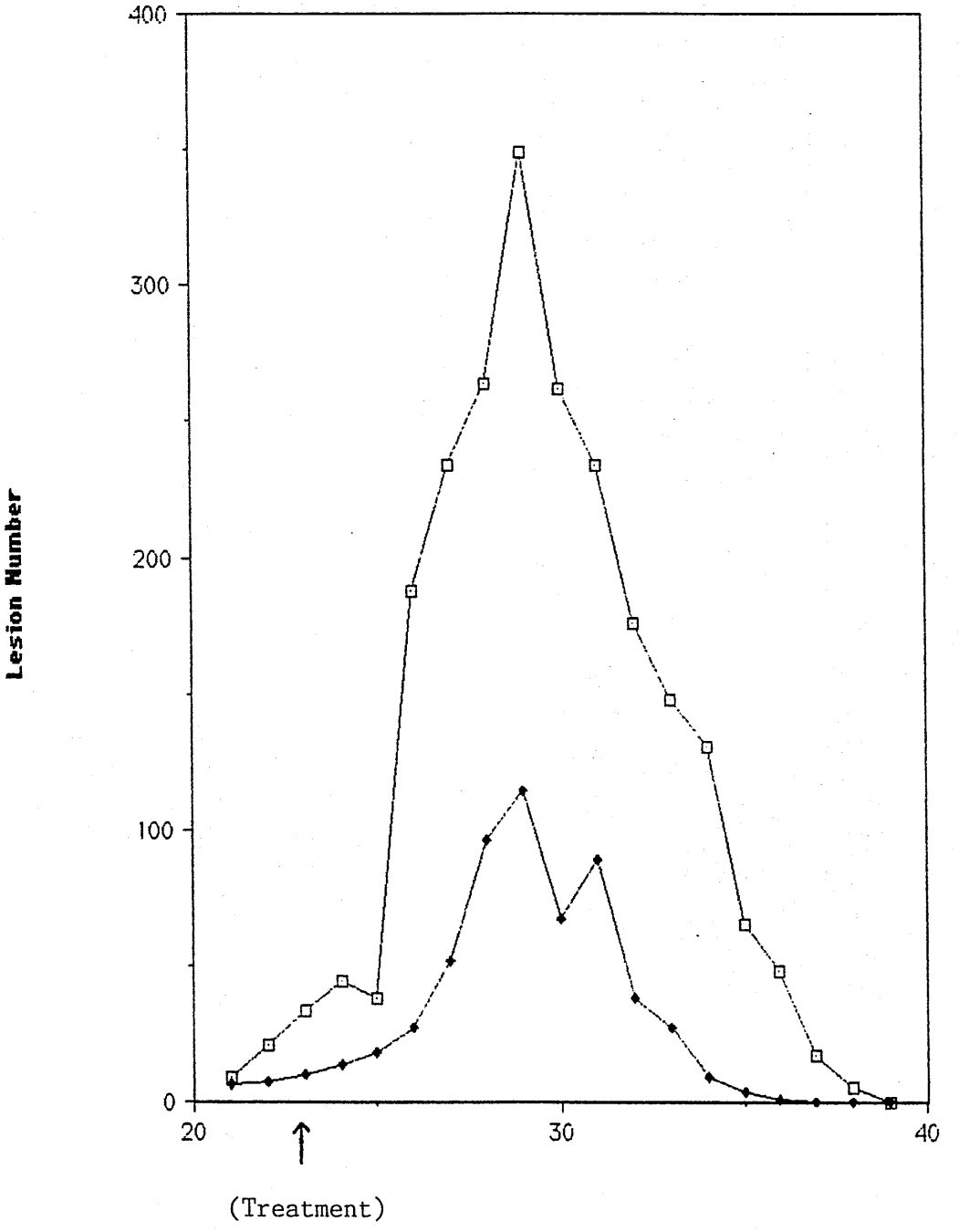

Table 9.1 - History of animals and outbreaks

Week no.	Date	Animals numbers	Activity	Movement (origin)	Movement (destination)	Disease incidence affected animals T	C
1	28/09/81	37 calves	purchase	dealer	single pens		
2	05/10/81						
3	12/10/81						
4	19/10/81						
5	26/10/81						
6	02/11/81	5 groups	weaned	single pens	hardening-off pens 1 - 5		
7	09/11/81						
8	16/11/81						
9	23/11/81						
10	30/11/81						
11	07/12/81						
12	14/12/81						
13	21/12/81						
14	28/12/81						
15	04/01/82						
16	11/01/82						
17	18/01/82						
18	25/01/82	18 19	re-group-ing	H O pens	beef yards 5+6		
19	01/02/82		inspection 03/02				
20	08/02/82	19 18	no inspection 08/02				
21	15/03/82		first signs ringworm			3	3
22	22/03/82					4	5
23	01/03/82		Treatment 01/03 05/03			5	5
24	08/03/82		08/03			5	6
25	15/03/82		Treatment 15/03			4	6
26	22/03/82					10	8
27	29/03/82					10	14
28	05/04/82					13	15
29	14/04/82					13	14
30	19/04/82					13	15
31	26/04/82					15	15
32	03/05/82					11	10
33	10/05/82					11	7
34	17/05/82					12	5
35	24/05/82					4	3
36	31/05/82					5	2
37	07/06/82					3	0
38	14/06/82					2	0
39	23/06/82					0	0
40	28/06/82						
41	05/07/82						
42	12/07/82						
43	21/07/82						
44	26/07/82						

Table 9.2 Animals and Disease Incidence

Week No.	Date	Affected Animals (Actual)		Affected Animals (Cumulative)		Active Lesions		New Lesions (Actual)		New * Lesions (Cumulative)	
		T	C	T	C	T	C	T	C	T	C
19	01/02/82										
20	08/02/82										
21	15/02/82	3	3	3	3	9	6	9	6	9	6
22	22/02/82	4	5	4	5	21	7	14	4	23	10
23	01/03/82	5	5	5	5	34	10	18	3	41	13
24	08/03/82	5	6	5	7	44	14	21	4	62	17
25	15/03/82	4	6	6	9	38	18	6	7	68	24
26	22/03/82	10	8	11	11	188	27	158	9	226	33
27	29/03/82	10	14	11	16	238	52	85	27	311	60
28	05/04/82	13	15	15	17	264	96	80	58	391	118
29	12/04/82	14	15	15	17	349	114	131	45	522	163
30	19/04/82	13	14	15	17	262	67	37	6	559	169
31	26/04/82	14	15	17	17	234	89	36	29	595	198
32	03/05/82	10	10	17	17	176	38	46	4	641	202
33	10/05/82	12	7	17	17	148	27	16	2	657	204
34	17/05/82	11	4	17	17	131	9	12		669	204
35	24/05/82	4	2	17	18	65	4	5	1	674	205
36	31/05/82	5	1	17	18	48	1	9	0	683	205
37	07/06/82	3	0	17	18	17	0		0	683	205
38	14/06/82	2		17	18	5	0	1	0	684	205
39	21/06/82	0		17	18	0	0	0	0	684	205
40	28/06/82										
41	05/07/82										
42	12/07/82										
43	19/07/82										
44	26/07/82										
45	02/08/82										
46	09/08/82										
47	16/08/82										
48	23/08/82										
	30/08/82										

*Casualties excluded.

	Test	Control
Total Animals	19	18
Total Animals Affected	17	18
Total Animals Non-Affected	2	0
Total Lesions	684	205

T = Treated Animals
C = Control Animals

Table 9.3 - Duration of lesions in test group

Animal number	Duration of disease (weeks)	Lesions	Duration of lesions - weeks 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
TEST GROUP																				
1	11	25	9	2	4	3	4	2		1										
2	12	50	18	10	4	1	2	3	2	2	6	2								
3	2	1		1																
4																				
5																				
6	1	1	1																	
7	11	42	12		5	10	2	4	6	3										
8	11	70	9	2		12	18	3	3	17	5	-	1							
9	16	27	8	3	4	1	3	6	2											
10	9	67	19	11	8	7	8	4	5	3	2									
11	14	80	11	4	13	20	22	7		2			1							
12	8	10	6			1			2	1										
13	11	84	12	1	10	14	4	14	10	6	8	5								
14	7	17	7	7	1		1		1											
15	10	80	16	2	17	10	14	8	6	6		1								
16	8	35	19	4	4	2	2	1	1	2										
17	9	47	7	19	11	1	4		2	3										
18	7	35	8	8	4	7	8													
19	7	13	2	7	2	1	1													
Total		684	164	81	87	90	93	52	40	46	21	8	2							
Range	1-16	1-84	1-11																	
Mean	9.1	40.2	3.9																	

Table 9.4 - Duration of lesions in control group

Animal number	Duration of disease (weeks)	Lesions	Duration of lesions - weeks 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
CONTROL GROUP																				
1	13	12	5		1	2	2		1	1										
2	7	13	3		3	6			1											
3	8	6	3			1	1			1										
4	8	18	5	1	2	3		4	3											
5	11	8		3	3	1				1										
6	7	19	5		2	8	4													
7	5	17	10	2	2	2	1													
8	6	15	5	2	3	2	3													
9	11	14	10	1		1	1		1											
10	1	1	1																	
11	8	2	1				1													
12	5	4	2	1	1															
13	15	9	4	1	1		1	1		1										
14	9	41	11	11	7	3	4	3	2											
15	5	7	4	1			2													
16	2	1		1																
17	14	5	1		1			1	1		1									
18	12	13	2			3	1		1	1		1								
Total		205	72	24	26	32	21	9	19	5	5	1								
Range	1-15	1-41	1-10																	
Mean	8.2	11.4	3.2																	

CHAPTER 10

Observations on an outbreak of ringworm in young cattle treated in part with two types of topical therapy

INTRODUCTION

Topical therapy of ringworm has been a major method of application for many years. Several compounds have been used by the agricultural industry including copper naphthenate and cod liver oil (Kopertox, Crown) which have been available for over 20 years. More recently, enilconazole (Imaverol, Crown) has been introduced and was the subject of a previous chapter (Chapter 9). It was thought advisable to test the efficacy of one of the most long-standing and widely used topical applications. However, the use of untreated controls was resisted by the cattle owners and so Imaverol was used as a control comparison.

MATERIALS AND METHODS

Two groups of calves consisting of 18 Belgian Blue x Friesian (BB) and 18 Gelbvieh x Friesian (GV) were purchased from a dealer and delivered on 20th October 1987 when about a week old. Initially they were housed in a new calf house which had only previously been used for one batch of calves. No calves developed ringworm in this batch although the house was cleaned but not disinfected prior to the study group's entry. The calves were disbudded and vaccinated against Clostridium chauvoei on 17th November. A week later (24th November) they were weighed and moved to pens in the hardening-off accommodation.

This second housing had been cleansed, disinfected and left/unoccupied

for 8 to 10 weeks. Sampling of this accommodation was undertaken on 9th September prior to the cleaning procedures. The 36 calves were divided equally according to breed into six groups in this accommodation. The pens were separated by a solid concrete wall 1½ m (5 ft) high. The calves were moved to a third building (the Dales) on 13th January 1988 where they were kept in the two breed groups. After 2 weeks (27th January) they were moved in breed groups to a fourth premises (monopitch building; pens 1 and 2) and the treatment trial was discontinued although weekly observation was still undertaken. No cleaning or disinfection occurred in the fourth unit prior to changes in the penning of calves. They remained in the monopitch building until slaughtered in September 1988. During their time in the fourth premises they moved pens (pen 3 and 4) again in breed groups on 9th March. Subsequently they had a final move within the monopitch building on 22nd June (pens 5 and 6).

Weekly observations were performed during the life of these animals except for 2 weeks (3/2/88 and 10/2/88) using the previous recording system. As infection occurred individually infected animals were treated with either Kopertox or enilconazole. This was applied as a spray and began as soon as the first lesion was observed. The first animal infected was sampled to confirm the diagnosis of ringworm. Kopertox was sprayed on day 1, 5, 9 and 13 (i.e. every 4th day) as indicated in the data sheets (i.e. twice weekly). Imaverol was used on day 1, 3, 7, 9, 11 as routinely undertaken on this unit although the data sheets suggested three to four times at 3-day intervals.

RESULTS

During their stay at the unit there appeared to be several cases of ringworm (17 animals) and these could conveniently be divided into six outbreaks although four occurred during the therapy trial - involving 17 of 36 animals initially in six groups of 6 animals each. Outbreaks 5 and 6 involved 7 animals not previously affected some of each group.

Outbreak No. 1

The first case of disease involved 2 lesions on BB calf 367 in the second housing on 16th December 1987. These were confirmed to be due to Trichophyton verrucosum. Treatment was undertaken with Kopertox. A third lesion was found in the fourth week of the outbreak and the outbreak resolved 7 to 9 weeks after first seen. No other cases occurred in in-contact animals and no recrudescence of infection occurred in animal 367.

Outbreak No. 2

This involved GV calves in pen 5 and began on 6th January. Four calves (283, 284, 285, 296) were infected at that time. Calf 285 had 3 lesions and the other 3 had 1 each. Calf 283 subsequently developed another lesion on the third week of infection. Calves 283 and 296 received Kopertox and 254 and 285 were given Imaverol. Calf 280 showed infection 1 week after the other 4 (13th January) when in the Dales building and was untreated.

Lesions treated with Kopertox remained on one animal for 3 weeks and on the other from 4 to 6 weeks. On those treated with Imaverol lesions lasted 4 to 6 weeks and on the untreated control (280) remained for 7 weeks. The total outbreak lasted 8 weeks. There was no recrudescence of infection in any of these animals and calf 281 did not contract ringworm.

Outbreak No. 3

This next problem involved 2 calves (365, 366) following movement to the Dales building on 27th January. These may have been infected by calf 367 or perhaps following handling (see later). A single lesion was present in each animal. Calf 365 was treated with Imaverol and the lesion lasted 6 weeks. Calf 366 received Kopertox and its lesion, of similar size, lasted 4 weeks. Two other calves (368 and No Tag) developed ringworm. One (368) occurred in a non-observation period and was not treated; it lasted 4 weeks. The other calf (No Tag) had a single lesion for 1 week.

Outbreak No. 4

This was again in BB calves in the monopitch building and was in pen 2 which previously had not shown infection. It is considered a separate outbreak because of its likely origin (see later). Three animals were involved, 2 developing one lesion on the head and Calf 360 had 3 sites of infection on 27th January. Calves 360 and 361 received Imaverol and 362 was treated with Kopertox. The lesions on all 3 animals resolved within 3 weeks.

Outbreak No. 5

This occurred on 6th April 1988, 4 weeks after a change of penning in the monopitch building and was considered to involve only BB animals. Five animals were involved, 2 of these (360, 366) had 2 lesions lasting one week. Three other calves (356, 364, No Tag) were also infected. Calf 356 showed up to 16 lesions beginning on 6th April. No Tag calf showed 1 lesion which remained for 7 weeks and calf 360 (previously infected) also had 1 lesion at the start of the outbreak. Calf 364 showed a lesion for 1 week on 27th April and on the 4th May calf 366 had 2 lesions again for 1 week.

Outbreak No. 6

This occurred at the same period in GV animals in pen 3 of the monopitch building. Four animals (292, 297, 298, 299) were involved, the first (298) on 6th April had 5 - 10 small lesions. Calf 299 had 4 lesions on the same day. Calf 297 was first observed on 13th April with over 12 lesions and 292 had 5 - 10 lesions on 20th April. All infected cattle were treated on 4th May and then 6th, 8th and 10th May. Lesions lasted 1 to 8 weeks and were all resolved 4 weeks after the use of Imaverol.

Samples of hair and debris from the environment taken prior to the entry of the cattle (9th October 1987) did not produce T. verrucosum. However the fungus was isolated from samples from selected cases.

DISCUSSION

The origin of each outbreak is not clear. However, as infection was not detected when the premises were sampled, it did suggest that each outbreak could be considered a separate incident.

Origin of outbreaks

Outbreak No. 1

In the initial outbreak the group of BB calves was dehorned and vaccinated against Clostridium chauvoei on 17th November 1989. The animals were weighed and moved from apparently clean and non-infected new accommodation to cleansed and disinfected pens 1 week later on 24th November 1987. At the time of operations a female student working on the unit contracted ringworm on a forearm and since she was reported to be reluctant to cover the large lesion with her sleeve it is probable that when holding the calves for operations the affected arm rubbed against the sites causing lesions on animal 367. The incubation period is usually about 28 days and so the time of infection could well have been the date of disbudding, 29 days before the first sign. Alternatively, the tools and weighing machine may have transmitted infection.

Outbreak No. 2

The first signs of ringworm in the second outbreak were observed on 6th January 1988 in GV calves in pen L5 in the follow-on area. Four of the 6 animals - 296, 283, 284, 285 - revealed head lesions 26 days after catching, handling and vaccinations on 11th December 1987.

This perhaps suggests dissemination of infection at that time.

Outbreak No. 3

Disease appeared in 2 BB animals on 27th January in a monopitch pen and in a group formerly in pen L3, the site of the initial outbreak. Catching, handling and vaccinations on 28th December 1987 probably conveyed infection in this case. The incubation period would have been 30 days.

Outbreak No. 4

This incidence followed a pattern similar to outbreak no. 3 in follow-up pen L2 and also occurred 30 days after catching, handling and vaccinations in 3 BB animals (360, 361, 362) on 28th December 1987. the first signs of disease were on 27th January. Segregation of animals in the six follow-on pens was more or less complete and no other activities were considered to be related to the incidence of disease.

Outbreak No. 5

This incidence became evident on 6th April 1988, some 10 weeks after outbreak no. 4 and 4 weeks after movement of BB animals from pen 2 to pen 4 in the monopitch accommodation. The pens were dirty with no removal of litter, nor disinfection. There was also handling, weighing and visits to common meeting places. However the possibility of spread from the previously clinically affected animals could not be ruled out.

Outbreak No. 6

The incidence in outbreak no. 6 followed the same pattern as outbreak no. 5. Eighteen GV cattle were moved from monopitch pen 1 to monpitch pen 3 on 9th March 1988 and 28 days later, on 6th April, ringworm lesions were observed.

As infection so often followed movement, the various collecting areas and handling equipment did seem to be a possible source of infection. However another explanation could be that the stress of changed routine allowed dormant spores on the cattle to germinate and cause infection.

Spread of disease producing a high level of incidence and morbidity was possibly not evident because the level of infection was relatively low in the environment due to cleansing and disinfection practices. Disease was also probably limited by early treatment when compared with other trials.

The morbidity level was low and lesions were few, small and short-lived. In the secondary outbreaks 5 and 6 between 6th April 1988 and 20th May 1988 there was delayed treatment and many more lesion developed. This demonstrated the importance of dealing with first lesions, controlling spread in animals and groups.

The drugs used in this trial were copper naphthenate and enilconazole. The copper compound was introduced around 1956. The data sheet advises 'Do not scrape, brush or remove ringworm crusts'. Enilconazole was introduced about 1981. The data sheet advises that the drug does not penetrate and that it is necessary to brush lesions to

remove scabs.

In the outbreaks under report, lesions were not scrubbed, scraped or brushed during therapy. In addition the copper compound was prepared as a liquid for spraying rather than used as an aerosol.

Comparison of the effects of the two drugs involved provides little evidence of differences (Table 10.1). Duration of lesions in one case of Kopertox therapy (7 - 9 weeks - outbreak no. 1) appeared to be slightly longer than in any treated with Imaverol but overall benefits appeared similar. Both therapies were aided by early application and before infected scabs and scales had dropped to the floor or been rubbed off on other animals. Both therapies were probably aided by the apparently low weight of infection in the environment. Later, in other outbreaks, delayed treatment meant spread had occurred on the animals and in the environment. Animals were affected in outbreaks nos. 5 and 6 possibly because earlier cases in the groups were resistent or immune following previous incidence. In outbreak no. 5 the disease occurred 2 weeks after outbreaks nos. 1 to 4 and with delayed treatment many lesions of varying size occurred. In 1 animal 16 such sites were counted on one occasion. In outbreak no. 6 again multiple lesions occurred in 4 animals.

An ealier (1981-1982) controlled drug trial using Imaverol showed more lesions in treated than in untreated control animals, possibly because sponging and removal of scabe caused spread of the fungus.

CONCLUSIONS

In all untreated naturally occurring outbreaks at this unit recorded by the author, morbidity has been 95 - 100%. In this trial the morbidity has been lower probably because of preventive measures reducing weight of infection and early therapy.

Lesions have been few, small and of short duration.

The findings in comparison with other controlled trials demonstrate that therapy alone is uneconomic, wasteful and ineffective on established disease.

It must also be concluded that physical removal of scabs advised by data sheets in the application of topical agents is dangerous.

There may also be advantage in applying oil based therapeutics to aid penetration and persistance of the fungicide.

Appendix 1 shows duration of naturally occurring outbreaks where no therapy has been applied. These figures serve to demonstrate the value of preventive measures and early therapy in outbreaks nos. 1 to 4.

Table 10.1 - Duration of disease - weeks

Outbreak Nu.	Animal no.	Lesions	Kopertox therapy Lesions	Animals	Outbreak	Imaverol therapy Lesions	Animals	Outbreak	No therapy Lesions	Animals	Outbreak
01 - 04 Numbers	12	20	8	5	4	7	5	3	5	2	2
Mean duration			4 - 5½	4 - 5	4½ - 6	3 - 4½	3 - 5	4 - 5	4	4	7
05						7+	7+	7+			
06						9+	9+	9+			
O 09	See Appendix 1					4	9	19			
O 10									3	8	17
O 7 x NOO's			See Appendix 1						4	8	18
O 3 x NOO's (3 different drugs)									4	8	17

CHAPTER 11

General aspects of therapy, control and prevention

General factors impeding efficient and economic therapy

A high incidence of ringworm on cattle rearing premises results from heavy levels of infection in the envrionment and on the animals. This was suggested in the early studies (see Chapters 2, 3, 4, 5). With such infection the morbidity in individual groups is usually very high. Frequently 95 - 100% of animals in individual groups are involved in such heavily infected premises. The longevity of the fungus, Trichophyton verrucosum, in the range of 4½ to 5 years (McPherson, 1957b), helps the build-up of infection in accommodation, weighing machines, common meeting places, tools and other equipment. Delayed therapy in such cases of high morbidity renders the control measures ineffective and wasteful.

Faulty application of drug preparations may also be counterproductive - even following data sheet advice in some cases can lead to spread of infection and thereby prevents control. Brushing, spraying, washing, scrubbing, scraping and removing scabs and crusts as requested by the directions for Imaverol and suggested for Defungit and Mycophyt appears to multiply the number of lesions very readily. This is demonstrated by the figures shown in the Imaverol trial, when there were three times more lesions in trial animals than in controls (see Chapter 9). The data sheet advising on application of Kopertox condemns such methods. The contraction of infection and production of lesions needs little rubbing or trauma for it to occur. Inter-animal grooming, licking, sucking and rubbing in crowded pens also facilitates spread (Chapters 1, 6).

In established disease not only will much infection already be deposited in the environment but also laid deep in the skin, hair follicles and under the coat where many drugs will fail to penetrate. In addition, persistence on the coat of many preparations is likely to be low. This makes repeated therapy necessary but probably uneconomical. Kopertox advisers emphasise the deep penetration of the preparation, whilst the Imaverol data sheet states that the preparation is not absorbed. Presumably other water-based solutions have limited persistence and penetration on treated animals.

Other important factors arise in the application of topical therapy. All the lesions may not be observed at the time of treatment. On many animals lesions have been found under the jaw and frequently under the tail and in the perianal region in older calves. Whilst lesions are rarely found on the abdomen and trunk it is all too easy to miss those on the rear of the animals. Application of systemic therapy overcomes this problem. However it may also be beset with difficulties where individual animals do not absorb or assimilate sufficient drug.

The continued infection in individual groups can also impede efficiency of drugs where there is no segregation of animals from other infected groups, recovered animals or access to contaminated equipment or environment. The fungus may often be difficult to find in the environment and so it is hard to identify a potentially infected building.

If therapy is delayed then often the degree of infection available for transmission is great. Infected material may also have dropped to the ground or lodged on walls or barriers. In some places it may not be

possible to disinfect the areas thoroughly either when the pen is occupied or after vacation.

Successful early therapy in clean surroundings may be unproductive in that where there is later exposure to infection, disease may still break out. However, with spontaneous recovery there is also acquired resistance or immunity. This prevents recrudescence or recurrence of infection. Thus animals recovering naturally from ringworm without therapy remain free from disease thereafter (see Chapters 1, 6).

Ideal factors producing efficient and economic control by therapy

First lesion therapy supported by prevention, husbandry, hygiene and disinfection measures appear to be significant factors in the control of ringworm in cattle (see Chapter 2). The results produced involve a saving of time and money in reducing incidence. This is especially so on premises where infection has previously occurred due to the absence of these precautions and where currently the risks of introducing infection are frequent through purchase of calves.

The value of early therapy has also been emphasised by Horrox (1976). It may be aided by vigilance in a 28-day incubation period for first signs of disease following moving or mixing calves. The predilection sites should be examined, especially the head and around the eyes etc. The first evidence is frequently a swelling in the suspicious area. Then the grey-white scales appear among the thinning hairs. Early segregation of calves will limit spread, especially when they are initially reared in single pens.

Therapy using a topical oil-based drug may be used early to stop further infection but it must be sprayed on and not sponged, washed or forcibly applied. Repeated drug applications may aid penetration and spread. Spraying with therapeutic agents of likely places where infection may have dropped in scales or scabs is feasible and helpful if animals are in limited accommodation. In all cases it is important to check probable sources of infection and segregate wherever practicable. In particular, metal bars between pens and feed troughs aggravate morbidity and spread and warrant cleansing and, if possible, disinfection.

During this present study a very marked fall in incidence and morbidity where husbandry, hygiene and disinfection measures have been implemented has been observed. This has led to further limitation of disease in individual animals treated early in such surroundings.

These findings may be checked against those occurring in spontaneous recovery. The efficacy of drugs may be monitored by a small number of affected animals in any treated group with few, small lesions and little or no spread of disease. Duration of lesions is less than the average 4 weeks, duration in the animals is minimal and less than the mean of 8 weeks and outbreaks last on average less than 18 weeks. These findings are the result of many years of study by naturally occurring outbreaks and represent spontaneous recovery figures for untreated groups of animals which have not been intermixed en route to maturity. Resistance and immunity appear to develop in these periods. Recurrence has not been observed in previously infected animals but spores may remain alive on the animals and in the environment.

Considering the efficacy and economics of therapy in the control of ringworm in cattle involves a multiplicity of factors. The cost of therapy is affected by the number of animals to be treated and the cost of time and labour employed in treatment. Reducing these factors in the manner portrayed in this discussion therefore aims for economy and efficiency of control measures in the interests of animal health and public health. The major factors in achieving these low levels of incidence and morbidity, extent of lesions and level of infection include prevention, husbandry, hygiene and early therapy of first lesions. This may seem repetitive but the points have been proven in drug trials and by observations of all aspects of the disease.

Therefore a suggested method for ascertaining ringworm infection levels is as follows:-

1. A control group must always be present to measure spontaneous recovery.
2. The cattle should have been monitored from birth.
3. The animals should be examined at short intervals, e.g. weekly.
4. The position, number and size of ringworm lesions should be noted.
5. The nature of the lesions should be recorded at each visit.
6. The duration of lesions should be recorded.
7. The duration of disease on animal should be recorded.
8. The animals should be monitored for recrudescence of infection.

9. Samples of infection should be used for identification of causal agents.

10. Details of all disease and its therapy should be recorded.

11. Likely sources of ringworm should be ascertained.

12. All movements and mixings of the animals should be recorded.

The control of ringworm in cattle involves knowledge of some aspects of the epizoology of the disease, general prevention measures, therapy and spontaneous recovery, as well as the possibility of nutritional deficiencies. However, many factors appeared to affect control of the disease. The longevity of spores in buildings seemed to be little known or understood and as spores havebeen stated to be viable for 4 to 5 yearsaway from the animal body in buildings, and several months outdoors (McPherson, 1957b), this must be an obstacle in control.

The level of infection build-up in buildings was a hidden hazard where hygiene is limited and disinfection not practised. Modern intensive and semi-intensive husbandry appeared to provide accommodation from which litter was not removed between different batches of animals, and where the feed system might not be suitable for the introduction of systemic drugs or their uptake on a controlled basis.

Drug testing and trials

In conducting tests and trials with drugs intended to control ringworm in cattle it is essential to heed the information and advice in the respective data sheets. It is also necessary when assessing the value of therapy to use a comprehensive observation code and keep records to facilitate comparison between drug test results and results of spontaneous recovery in untreated control animals.

During this present study the systemic drug griseofulvin was used in one trial while in the second, third and fourth trials topical dressings were applied.

Griseofulvin

The systemic drug griseofulvin (Fulcin, Coopers and Grisovin, Pitman Moore) has been used for 25 to 30 years. One advantage of the substance is that it may be administered in the food ration of the animals and handling is thus avoided. Also the affected batch may all be treated whether visibly affected or incubating disease. The drug does not kill spores but thwarts their activity in the layers of the skin. However, spores may remain alive in the coat, on the skin and in the environment to cause re-infection where precautions are not taken to protect animals against such recurrence.

A group of 10 test animals was employed with 9 control animals (Chapter 7). Disease in the test group was halted and duration in the group was 9 weeks, whilst in the control group disease persisted for 16 weeks.

The topical remedies are each based on different substances.

Natamycin (Mycophyt, Gist-brocades)

This preparation contains natamycin in a powder / suspension form to prepare. The data sheet states that 'the suspension can be applied by spraying or sponging. In mass treatment of cattle spraying may be preferable. For the individual treatment of horses and cattle, sponging down the affected area is advised. In mass treatment, the entire body surface of all animals (infected and non-infected) should be sprayed in order to prevent any spread from infected animals not yet showing visible

lesions'.

Chapter 8 records the testing of natamycin on 18 animals with 18 animals in the control group. No limitation of disease was recorded or observed and no significant difference in duration of disease was found between the test group (22 weeks) and control group (21 weeks). The last new lesions in the control group were 3 weeks earlier than in the treated group. The control group had fewer lesions (240) than the treated group (340 lesions) (see Table 8.1).

Enilconazole (Imaverol, Crown)

This preparation was launched about 1982 and is recommended for whole body spraying in cattle but it is recommended that crusts are removed by a hard brush. Cattle should be washed with the diluted emulsion. Further information states that Imaverol is not absorbed by the skin.

In Chapter 9, 19 test animals and 18 control animals were involved in the test of enilconazole. No beneficial effects were seen in the test group and the treated animals had more lesions, and disease persisted longer (19 weeks) than in the control group (17 weeks). There was no halting of disease in treated animals - if such had occurred it would have been noted on the lesions ladder of Table 9.1. Then also a gap would have occurred until new infection completed incubation (see Chapter 9).

The two topical drugs, natamycin and enilconazole, did not produce better results in the treated animals than spontaneous recovery in the contemporary controls.

Copper naphthenate and enilconazole

The object of this trial was to compare the value of copper naphthenate treatment against a previously tested therapy, enilconazole.

Copper naphthenate and cod liver oil (Kopertox, Crown)

The data sheet directions are forthright: contra-indications, warnings etc. - 'Do not scrape, brush or remove ringworm crusts. Kopertox will penetrate and kill infection (removal of crusts spreads infection).' further information - 'Kopertox penetrates deeply, destroys infectious spores and prevents the spread of infection.'

Although four distinct outbreaks of disease occurred in two groups of calves initially, in 3 pens of 6 animals and subsequently in two lots of 18 of each breed there was no obvious difference between the two treatment groups.

General aspects of therapy

Systemic drugs, mainly types of griseofulvin, have been used for 20 to 25 years/. There is a danger of re-infection after use if undertaken too early in disease. It is generally regarded as an expensive treatment/with the inference that resistance, or immunity, might be thwarted with the risk of re-infection of treated animals. Topical preparations have more variety.

Twenty nine drug trials reported by 20 combinations of authors are analysed in Appendix 2. Most of these display shortfalls in judging the value of therapy, including failure to make allowance for

spontaneous recovery. There was no record of precise age of disease at the commencement of treatment and no apparent synchronisation of factors which should be common to treated and control groups i.e. similar age of disease, similar age of animals, similar incidence of disease, similar exposure to infection and similar numbers of animals. Often 'regular' inspections were quoted without the interval being stated. An interval of 7 days appeared to be about the maximum acceptable as lesions appear and heal at short intervals throughout an outbreak, and so the true infection level must be recorded.

Records were considered suspect in trials where photographs were the only recorded evidence of incidence. These provided a limited survey of sites of infection. New lesions were not shown, as they arose, to give complete history, and quite important is full duration of disease in each animal before and after treatment. Above all, time must be devoted to inspect and observe to the fullest extent disease in each animal, in test and control groups. Any cursory inspection would fail to reveal the full extent of disease. There appeared to be limited value from therapy alone in controlling the diseases. Those drugs of limited value were also expensive.

The suggestion is made that the above-mentioned criteria should be embodied in a code of practice, or set of guidelines, for the controlled testing of drugs under field conditions to check efficacy and prevent unjustified credit being given to their use. A suggested method for ascertaining ringworm infection levels is outlined in Chapter 11.

These drug tests and trials have revealed many probable impeding and factors interfering in attempts to control ringworm by therapy. Among

these is the handling of animals in crating, weighing, taking of samples and intermixing. Additionally, contact with recovered animals, other affected animals within a group, and feeding through bars, especially sloping bars where rubbing of the neck region is a probable factor in spreading infection. Hidden lesions which may go untreated are to be remembered and thorough inspection made in application of therapy. Such sites may be in the ears, under the jaw, but importantly in the perianal area and on the tail. Penetration and persistence of drugs must also be considered regarding buried infection in the skin and in thick coats.

Factors even more important than the above are the dangers from contaminated premises where fungus may survive for years. Of even more importance is the danger of spreading disease on animals by removal of scabs and crusts in the application of drugs. Unfortunately this action is described in the data sheets of some drugs as a means of applying therapy where there may not be adequate penetration of the substance.

In intensive surroundings animal to animal behavioural habits - grooming one another by licking, sucking, rubbing and contact by butting and riding are factors to be considered.

Therapy of established disease must therefore be wasteful and uneconomic when it is unable to contend with the above impeding factors. Likewise if it is unable to compete with unaided, natural or spontaneous recovery then time will be best spent on prevention, husbandry, hygiene and disinfection to reduce the burden of infection and expense of drugs, labour and time. Additionally, successful early

therapy may lose its value if there arises recrudescence or recurrence through thwarted immunity/development due to lack of prevention, husbandry, hygiene or disinfection measures.

The value and importance of these latter measures have been proven and demonstrated on the trial premises and so sets a good example in the interests of public health, economics and healthy husbandry.

Control and prevention

Thus, the best control of ringworm in cattle is by therapy or allowing spontaneous recovery. Drug use is considered expensive when all animals in a group are treated. However, their value may be apparent when there is early treatment of first lesions, especially when combined with prior application of preventive measures for susceptible stock and premises. Therefore it is essential to understand and recognise the relevant factors in the epizoology of the disease.

The most important problem is that Trichophyton verrusocum can persist in buildings for between 4 and 5 years (McPherson, 1957b) and outdoors for several months. This means that in infected pens animals are exposed to infection before, during and after outbreaks, and that infection may drop to the floor and surroundings before, during and after any therapy.

The amount of fungus required to cause disease depends on the level of fungus in the source object (Lepper, 1969, 1972). Level of infection also appears to be very important in determining the number of animals which will become infected in a group. Thus in uncleansed and

and non-disinfected premises which have housed infected animals the incidence and morbidity in a group may be as high as 100% but averaging 95%. A major problem for calf rearers is the risk of animals incubating ringworm. Infection in a mixed batch may be the result of animals coming from many farms, from dealers' premises, transporters and market premises. Any one or more of these places may not have received adequate disinfection.

Sources of infection may include purchases, infected buildings (particularly woodwork), infected animals contacts, recovered animals, other farm animals, stray animals. The spread of disease may also be difficult to determine in some cases but since little rubbing is necessary to implant infection it appears highly probable that with natural animal-to-animal grooming, sucking and licking, infection is introduced in normal animal behaviour habits. Rubbing against equipment in common meeting places, against and through partition bars and food troughs also aids infection entry. Wooden splinters especially are involved. All these latter factors are aggravated by overcrowding where disease is prolonged and infection multiplied by intermixing of clean and infected groups.

The beneficial effects of drugs may be prevented by the presence of fungus in hair follicles, sebaceous glands and any deep layers of skin. It is very important when considering therapy with topical medication that drugs are not applied by sponging, brushing, wiping, scrubbing or rubbing to remove scales and scabs. Such activity would spread infection from animal to animal and scales and scabs would be deposited in the environment. thus the detection of the first lesion of any

outbreak can fundamentally aid control by therapy. Its prompt treatment prevents increase in infection.

When testing methods of therapy a strict code of practice must be observed to provide information regarding the efficacy of drugs which may be very efficient in vitro but inefficient in vivo because of inhibiting factors.

The NAC Calf and Beef Unit has endeavoured to set an international good example in the control of disease. The incidence of ringworm on the Unit has been reduced considerable in the last 3 years by cleansing, disinfection and limited therapy, but progress is repeatedly disrupted and almost ruined by new intakes of infected batches. While it is impossible to detect incubating disease, weaned calves have been admitted with clinical evidence of ringworm.

In the years 1985, 1986 and 1987 disease was introduced by 15 batches of 45 purchases (see Appendix V). It is therefore essential to find ways and means of preventing or at least reducing this input of infection. Initially it is suggested that vendors and dealers are advised that the NAC is endeavouring to set an international good example in controlling a disease of public health, economic and animal husbandry importance. Vendors and dealers should be asked to note the incidence of the disease on their farms and rounds, and invited to provide disease-free animals at all times. They should be asked to support the work of the Unit and seek the support of breeders.

The use of appropriate (fungicidal) disinfectants must be emphasised (McPherson, 1957a). The several risks in moving calves from breeding

farm to dealer's premises, through one or more markets, intermixing from several premises of origin and travelling in one or more transporters can multiply the risks of contracting infection. Direct delivery of calves from vendors to purchasers could help. On the Calf and Beef Unit purchased calves should be received into cleansed and disinfected accommodation.

Endeavour should be made to detect first lesions for prompt therapy. The earliest signs are frequently on the head, especially around the eyes and ears, with initial swelling, erythema, grey scales and depilation. Therapy should be by spraying, with no rubbing, sponging or brushing of lesions. Early segregation, isolation or removal will help to avoid spread. Feeding, routine operations or treatment of affected animals should be done after cleansing and disinfection of equipment including feed buckets and tools. The immediate environment of an affected calf should be sprayed with a suitable therapeutic agent to kill fungus deposited in scales and scabs on the woodwork and litter surrounding the animals.

Good husbandry is also an important factor in prevention. Segregation of affected animals from clean stock is beneficial. Segregation of purchases which may be incubating disease is of practical importance. The walls between pens sometimes allow contact between animals in two or three pens. Avoiding inter-pen contact may often be achieved readily by the use of different forms of mesh netting or material. This would avoid bars as partitions and not necessarily restrict light. There is certainly a practical importance in segregating and separating adjoining clean and affected groups of animals, and recognising the

point that recovered animals may carry live spores. Where possible the intermixing of groups of clean animals with affected animals should be avoided as this practice builds up weight of infection, prolongs incidence and encourages future spread of disease. Efforts to minimize rubbing places and escapes from open fronted pens will also prove helpful.

The occasional intermixing of groups on journeys from different pens to common meeting plaes - weighing machines, collecting yards or alleyways - can lead to spread of infection.

Hygiene must be practised in high degree in any control programme against ringworm. Litter, manure, dirt and dust should be removed whenever and wherever possible between batches. Cracks, crevices and woodwork may harbour spores for years (McPherson, 1957b) and should always receive thorough cleansing. Steam cleaning of the whole inside of a building or pen after each and every occupation should include roof, walls, floors, ledges, gutters, bars, partitions, food troughs and hoppers. Similar treatment should be provided for roadways, alleyways and floor gutters.

Common meeting places, races, catching crates, weighing machines and collecting yards should also be included in regular hygiene programmes. Cleansing of tractors, carts, forklifts and livestock transporters will also contribute to lowering weight of infection. Cleansing of other places should not be overlooked - isolation accommodation and the hands and boots of staff.

Disinfection of all the aforementioned places, equipment, vehicles and tools is essential to complement and supplement the cleansing processes. Again after much researching McPherson (1959a) has recommended the use of lysol, sodium hypochlorite, ammonia or phenol-based disinfectants/fungicides - whichever is least noxious for the interval between batches on a particular occasion. In some cases it may be necessary to rely on detergents.

Interim disinfection of partition bars may in some cases be advisable and helpful, especially when there are many visitors to a site or building. The cleansing and disinfection of protective clothing, especially footwear, should not be neglected. Footbaths at appropriate sites may serve a useful function in thwarting spread of disease to or from pens or buildings. The use of therapeutic agents may be beneficial and economical in small areas, especially small calf pens when infected scales and scabs are falling to the floor.

When action has been taken to prevent the introduction of infection and minimize spread of disease by segregation, cleansing and disinfection of housing and environment, then application of therapy will aid a practical and economic programme to control the disease. It would appear that the early use of griseofulvin or natamycin spraying and local topical therapy with copper naphthenate and cod liver oil may assist in treatment of ringworm.

CONCLUSION

The study of all aspects of the epizoology of ringworm in cattle, and

the conduct of several drug trials, using strict control and comparison measures, have contributed to the construction of a code of practice for the control of the disease.

Prevention, husbandry, hygiene and disinfection activities, followed by prompt early therapy for control of ringworm in young cattle is a regime shown to limit the incidence and morbidity of disease, and to aid the prevention of recrudescence or recurrence of disease in animals which are not naturally immune or which do not acquire resistance via infection.

Therapy via oil-based penetrating drugs applied topically by spraying alone will support this work. Incidence and morbidity figures over several years have shown the value of such practices.

GENERAL CONCLUSIONS

The average incubation period is 28 days. During this time vigilance to detect sources of infection and take action on therapy and preventive measures is important.

The establishment of time for spontaneous recovery also allows proper assessment of control measures.

Drug trials recorded in the literature have not heeded this fact.

Unless therapy is undertaken early and attention is paid to hygiene more lesions will occur.

Topical treatment must be by application spray and not sponging, scrubbing or removal of scabs.

On control measures emphasis must be laid on strict codes of practice for conduct of controlled drug trials but also for prevention practices.

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APPENDIX 1 Summary of ten outbreaks of ringworm in young cattle

APPENDIX 2 Drug trials on cattle rinworm reported in the literature

APPENDIX 3 Sites of lesions in ten outbreaks of ringworm in cattle

APPENDIX 4 Summary of ringworm incidence 1981 - 1988

APPENDIX 5 Sources of ringworm infection

APPENDIX 6 The therapeutic agents used, their commercial names and companies supplying them

Appendix 1 - Ten outbreaks of ringworm in young cattle

Outbreak	Epizoology 1 Indoors	2 Indoors & outdoors	3 Indoors & outdoors	4 Outdoors	Therapy 5 Grisovin T	5 Grisovin C	6 Mycophyt T	6 Mycophyt C	7 Imaverol T	7 Imaverol C	Totals Untreated animals	Totals Treated animals
Animals. Number	32	44	40	40	10	9	18	18	19	18	201	47
Animals affected Morbidity	30	39	35	35	10	9	18	18	17	18	184 94.6%	45 95.7%
Animals not affected Exclusions	2	5(4) 1D	5(1) 4D	5(3) 2D	0	0	0	0	2	0	10 7	2 0
Animals. Duratation. weeks												
Range	3/12	1/19	1/21	1/15	3/9	5/16	4/13	3/14	1/16	1/15	11/21 - 5/16	1/16 - 4/13
Mean	8.0	7.4	8.8	6.5	7.0	9.9	8.9	8.0	9.1	8.2	8.1	8.3
Outbreak. Duration. Weeks	33	28	26	17	9	16	22	21	19	17	18 = 4	17 = 3
Mixed groups = x	x	x	x									
Lesions. Number	316	435	370	256	91	120	340	240	684	205	1942 (277)	1115 (372)
Range	1/40	1/62	1/76	1/23	2/21	4/40	3/72	1/50	1/84	1/41	1/23 - 1/76	2/21 - 1/84
Mean	10.5	11.2	10.6	7.3	9.1	13.3	18.9	13.3	40.2	11.4	11	23
S.D.	9.0	12.2	13.2	5.4	6.3	10.8	19.6	11.6	27.2	9.1		
Lesions. Duration. Weeks												
Range	1/11	1/15	1/18	1/14	1/8	1.14	1/13	1/11	1/11	1.10	1/10 - 1/18	1/8 - 1/13
Mean	3.6	3.6	4.2	2.7	3.4	5.0	3.9	3.6	3.9	3.2	3.7	3.7
S.D.	2.2	2.5	3.2	2.6	1.7	2.5	2.4	2.2	2.5	2.4		
Animals affected before therapy date					10	7	13	12	5	5		
Animals affected after therapy date					0	2	5	6	12	13		
Duration of disease before therapy date - weeks					2	2	9	9	4	4		
Duration of disease after therapy date - weeks					7	14	13	12	15	13		

Appendix 2 - Drug trials on cattle ringworm reparted inthe literature

Author		Number of animals		Age of animals		Naturally-occurring (NOO) or experimental disease (ED)	Duration of disease before treatment		Drug Topical = T Systemic = S	
		Test	Control	Test	Control		Test	Control		
Anderson, P.C. & Campbell, J.R.	1964	NR	NR	NR	NR	NOO	NR	NR	Vitamin A	S
Andrews, A.H. & Edwardson, J.	1981	10	9	5m	5m	NOO	3wk	3wk	Grisovin	S
Becker, W. & Tiefenbach, B.	1969	142	-	NR	-	NOO	NR	-	Defungit	T
Bonsal, S.R., Gautam, O.P. & Monga, B.P.	1977	36	8	NR	NR	NOO	NR	NR	Jadit	T
Cobb, R.W., Martin, A.R. & Whalley, J.K.	1963	18	17	NR	NR	NOO	NR	NR	Fulcin	S
Edgson, F.A.	1970	26	14	1/8m	8/10m	NOO	NR	NR	Fulcin	S
El Abdin, Y.Z., Haza, S.M. & Mohamed, M.	1973	14	-	NR	_	NOO	NR	_	Fulcin	S
Hiddleston, W.A.	1970	116	64	NR	NR	NOO	NR	NR	Fulcin	S
Khanna, B.M., Datt, S.C., Monga, D.P. & Gera, K.C.	1974	50	9	2/12m	2/12m	NOO	NR	NR	Thiabendazole	T
Klatt, P.	1969	8	16	NR	NR	NOO	NR	NR	Defungit	T
Kriz, H. Jacobs, P. & Maryankova, K.	1971	70	-	NR	_	NOO	NR	_	Thiabendazole	T
Lauder, I.M. & O'Sullivan, J.G.	1958	2	1	3m	3m	ED	-	-	Grisovin	S
Misra, S.K.	1973	5	5	1yr	1yr	NOO	NR	NR	Grisovin	S
Monga, D.P., Satlia, K.C., Khanna, R.W.S. & Datt,S.C.	1974	20	12	5/8yr	5/8yr	NOO	NR	NR	Bordeau Mix	T
Monga, D.P., Khanna, B.M. & Saxena, A.K.	1975	30	5	12/18m	12/18m	NOO	NR	NR	Captan	T
Oldenkamp, E.P.	1979	258	NR	NR	NR	NOO	NR	NR	Natamycin	T
O'Sullivan, J.G.	1959	21	-	2m	-	ED	-	-	Grisovin	S
Pandey, V.S.	1979	30	30	NR	NR	NOO	NR	NR	Thiabendazole	T
		30		NR	NR	NOO	NR	NR		
Pearson, J.K.L. & Rankin, J.E.F.	1962	4	4	2/16m	2/16m	NOO	NR	NR	Grisovin	S
Satija, K.C. & Cautam, O.P.	1972	16	8	NR	NR	NOO	NR	NR	Iodine	T
		8		NR	NR	NOO	NR	NR	Acetic acid	T
		8		NR	NR	NOO	NR	NR	Acetica acid	T
		16		NR	NR	NOO	NR	NR	Alugin	T
		16		NR	NOO	NOO	NR	NR	Copper	T
Sevcik, B., Reichel, F. & Strakov, J.	1975	90	-	NR	-	NOO	NR	-	Aliphatic amines	T
Spanoghe, L. & Oldenkamp, E.P.	1977	41	10	NR	NR	NOO	NR	N	Natamycin	T

d = days; wk = weeks; m = months; yr = years

Appendix 3 - Sites of lesions in ten outbreaks of ringworm in cattle

			1 Around eyes	2 Ears	3 Muzzle Nostrils	4 Forehead + face	5 Cheeks + jaws	6 Poll	7 Dewlap + neck	8 Shoulders	9 Withers	10 Body (trunk)	11 Legs	12 Perianal Tail area	Total
1	1	Lesions	100	56	24	49	24	5	19	5	-	7	11	16	316
		%	32	18	8	16	8	2	6	2	-	2	4	5	
		Animals	26	23	15	10	14	3	7	5	-	5	7	13	30
		%	87	77	50	35	47	10	23	17	-	17	23	43	
2	3	Lesions	193	42	44	63	16	5	46	10	4	1	1	10	435
		%	44	10	10	15	4	1	11	2	1	-	-	2	
		Animals	32	18	18	16	13	4	16	7	3	1	1	5	39
		%	85	46	46	41	33	10	41	18	8	3	3	13	
3	4	Lesions	104	53	16	59	19	13	67	6	6	4	6	17	370
		%	28	14	4	16	5	3	18	2	2	1	2	5	
		Animals	29	20	8	13	11	3	18	5	2	4	2	8	35
		%	83	57	23	37	31	9	51	14	6	11	6	23	
4	5	Lesions	105	36	5	13	14	3	61	4	-	2	5	8	256
		%	41	14	2	5	5	1	24	2	-	1	2	3	
		Animals	29	20	3	8	11	3	27	3	-	2	4	4	35
		%	83	57	9	23	31	9	77	9	-	6	11	11	
5	6 T	Lesions	17	14	6	-	17	-	8	15	2	3	7	2	91
		&	19	15	7	-	19	-	9	16	2	3	8	2	
		Animals	8	6	3	-	3	-	2	8	2	1	4	2	10
		%	80	60	30	-	30	-	20	80	20	10	40	20	
6	6 C	Lesions	17	9	6	1	13	-	13	6	-	4	25	26	120
		%	14	8	5	1	11	-	11	5	-	3	21	22	
		Animals	8	4	4	1	3	-	4	5	-	3	6	4	9
		%	89	44	44	11	33	-	44	56	-	33	67	44	
7	7	Lesions	103	39	27	29	37	7	74	7	6	4	7	-	360
		%	31	12	8	9	11	2	22	22	2	1	2	-	
		Animals	17	12	8	7	11	4	11	5	4	3	3	-	18
		%	94	67	44	39	61	22	61	28	22	17	17	-	
8	7	Lesions	140	23	11	12	14	5	31	0	1	-	2	1	240
		%	60	10	5	5	6	2	13	-	1	-	1	1	
		Animals	17	12	6	6	8	3	10	-	1	-	2	1	18
		%	94	67	33	33	44	17	56	-	6	-	11	6	
9	8 T	Lesions	120	54	24	38	51	22	196	52	27	5	14	81	684
		%	18	8	4	6	7	3	29	8	4	1	2	12	
		Animals	15	14	11	12	12	5	14	10	8	4	7	14	17
		%	88	82	65	71	71	30	82	59	47	24	41	82	
10	8	Lesions	35	18	5	3	18	3	54	12	1	3	9	34	205
		%	17	4	2	1	9	6	26	6	1	1	4	17	
		Animals	13	10	3	2	10	7	14	7	1	1	5	10	18
		%	72	56	17	11	56	38	78	38	6	6	28	56	

T = treated animals; C = control animals

Appendix 4 - Summary of ringworm incidence 1981 - 1988

Incidence chart, embracing the following features and factors on calf and beef units.

1981 - 1988 Weekly records of incidence.

Fifty pens with 600 cattle normally accommodated.

Number of pens/groups showing disease incidence and number of animals affected are recorded.

Highest incidence in each year indicated by underline.

Rigid prevention, husbandry, hygiene, disinfection, early therapy measures not practised until mid 1985 save in preparation for the Royal Show.

P.H.H.D.T. blitz in 1985 provided much reduced incidence.

But incidence uplifted on several occasions via purchases of infected calves (see Appendix 5).

Group and pen incidence demonstrate that segregation of groups could reduce widespread incidence.

Appendix 4

Weeks		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52
1981	NGA	-	-	-	-	2	2	2	3	3	3	3	4	4	5	5	6	9	10	12	13	12	12	7	7	6	6	9	9	10	11	11	12	12	6	7	-	5	5	7	6	5	2	2	2	3							
	NAA	-	-	-	-	2	3	5	8	17	27	36	44	48	45	49	42	49	44	49	68	78	70	58	41	60	47	49	62	100	106	105	89	72	32	28	-	15	13	14	7	8	2	2	3	3							
1982	NGA	3	-	6	5	10	-	9	4	10	11	11	10	9	9	8	10	8	11	7	9	7	5	9	8	7	7	-	6	7	6	9	9	-	-	-	6	5	4	2	-	4	4	3	4	5	7	-	5	4	2	3	2
	NAA	19	-	29	38	38	-	60	29	58	75	75	46	67	77	72	80	68	69	43	45	39	43	45	56	48	45	-	39	31	24	30	34	-	-	-	29	25	19	6	-	12	8	5	5	9	9	-	6	11	7	15	9
1983	NGA	4	4	5	-	3	1	4	4	-	5	-	7	-	7	8	-	8	7	-	4	-	4	4	3	4	6	10	10	9	13	-	16	14	18	16	15	12	-	-	7	4	6	7	7	4	-	6	3	-	6	4	5
	NAA	13	19	23	-	8	1	7	8	-	27	-	43	-	42	50	-	47	51	-	25	-	27	25	17	25	28	25	22	23	33	-	33	49	59	49	53	53	-	-	27	12	32	32	27	17	-	25	15	-	24	19	17
1984	NGA	6	7	8	-	9	8	9	-	6	5	3	6	4	3	6	4	4	-	5	7	6	8	10	11	9	10	8	9	12	11	13	7	9	-	7	6	9	12	-	-	7	8	8	8	6	6	6	10	7	6	5	-
		16	17	33	-	36	24	23	-	20	14	9	15	16	10	18	15	16	-	20	20	20	14	22	24	28	42	42	32	34	38	37	24	39	-	37	24	36	49	-	-	18	22	22	19	11	14	20	30	30	31	32	-
1985	NGA	7	-	-	-	5	7	10	-	-	7	-	7	9	-	-	6	-	9	12	13	12	12	13	14	11	-	-	6	9	8	6	9	5	6	6	6	2	2	1	2	1	1	2	2	-	3	2	-	2	2	2	2
	NAA	24	-	-	-	20	25	56	-	-	57	-	64	67	-	-	43	-	47	75	86	101	97	79	95	60	-	-	21	28	24	13	23	9	7	8	9	2	3	3	2	1	1	3	2	-	9	8	-	12	8	11	9
1986	NGA	-	-	3	2	-	2	-	2	4	4	4	-	5	7	8	8	8	7	-	8	-	12	12	-	-	10	9	-	12	9	12	10	13	11	8	-	6	4	7	-	-	3	-	3	1	2	2	2	2	4	-	5
	NAA	-	-	5	3	-	3	-	4	13	16	15	-	14	17	23	39	47	39	-	60	-	67	62	-	-	54	39	-	36	18	33	25	24	25	18	-	10	12	35	-	-	3	-	4	1	3	3	4	7	9	-	9
1987	NGA	-	-	-	-	6	8	-	11	-	10	12	10	10	9	-	8	11	11	13	14	11	7	8	4	5	8	10	-	9	13	9	8	-	8	7	4	3	2	-	-	2	-	-	2	3	4	4	5	5	5	3	4
	NAA	-	-	-	-	20	31	-	40	-	27	37	33	38	42	-	36	40	34	29	37	22	10	11	10	11	13	23	-	19	28	16	24	-	24	18	15	7	6	-	-	6	-	-	7	6	6	10	5	5	8	6	5	6
1988	NGA	5	9	9	8	-	-	5	5	5	5	5	7	13	10	11	7	5	7	5	4	3	3	5	4	2	4	5	3	2	2	3	3	2	2	3	3	3	-	-	4	5	-	-	4	4	4	-	3	-	1	0	4
	NAA	5	16	19	13	-	-	11	11	21	14	13	16	25	24	21	13	13	18	10	7	6	9	9	8	6	11	16	5	2	2	3	3	2	2	3	3	4	-	-	13	10	-	-	8	9	8	-	7	-	1	0	7

NGA = NUMBER OF GROUPS AFFECTED
NAA = NUMBER OF ANIMALS AFFECTED

Appendix 5 - Sources of ringworm infection

R W	House where the ringworm occurred	Purchases	P	A	B	O
85						
Jan	Standring E	X		1 1		7
Feb		X	1	2		2
March	Standring E	X	1			1
April		X				
May		X			1	1
June		X			1	
July		X				
August		X				
September		X			1	
October						1
November		X				
December		X				
			2	2	3	12
86						
Jan	Barley Beef	X		1	1	3
Feb		X	1			
March		X		1		
April	Standring E	X	1			
May	Dales	X	1			1
June		X			1	4
July						2
August		X				3
September		X	1	1		
October		1				
November		X				
December		X	1			
			5	2	1	13
86						
Jan	Pole Barn	X		1 1		9
Feb		X	2			
March	Standring E		1			
April	Dales	X	1			
May	Standring W	X				
June		X				
July	Standring E	X	1			
August		X				
September		X				
October	Standring E	X	1			
November	Standring W	X	1			
December						
			8	-	-	9
	15	45	15	4	4	34

In the years 1985, 1986, 1987, 15 of 45 purchases of calves introduced ringworm to the calf and beef units. Origin of outbreaks in the same period is analysed in this table.

P = Purchases; A = animals; B = buildings; O = obscure

APPENDIX 6 - The therapeutic agents used, their commercial names and companies supplying them

Copper naphthenate and cod liver oil	Kopertox	Crown Products then Crown Animal Health (now Crown Veterinary Pharmaceuticals)
Enilconazole	Imaverol	Crown Products then Crown Animal Health (now Janssen Animal Health)
Griseofulvin	Grisevin	Glaxo Animal Health then Glaxovet Pitman-Moore (now Cooper Pitman-Moore)
Natamycin	Mycophyte	Mycofarm UK Ltd then Grist-Brocades (now Brocades)