Bovine Salmonellosis

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Summary

The clinical signs and post morten lesions in bovine salmonellosis are described and the diagnostic procedures for confirmation of a clinical diagnosis are outlined. Epidemiological features of the disease, particularly in relation to *S. dublin* and *S. typhimurium* infection are discussed and treatment and control measures are considered.

Introduction

Salmonellosis in farm livestock and its association with human infection has attracted a great deal of attention, particularly in recent years. The appearance of a chloramphenicol resistant strain of *Salmonella typhimurium phage type DT204* in calves in Great Britain[1] highlighted the potential public health risks and since then chloramphenicol resistant strains of the same organism, thought to have in some cases been derived from calves, have been isolated from sick humans.[2] More recently, chloramphenicol resistance has been demonstrated in other phage types of *S. typhimurium* and *Salmonella dublin* isolated from calves and other animals.[3]

It is now generally accepted that the incidence of salmonella infection in farm livestock is clearly related to husbandry methods and practices and there is a great deal of evidence to indicate that extensive systems in particular favour the spread of infection and a subsequent increase in the level of clinical disease. A number of comprehensive reviews of bovine salmonellosis have been published over the last two decades or so.[4,5,6,7,8] The main purpose of this paper, however, is to review for practitioners the more relevant features of the disease, derived from the published reviews and from the author's experience of bovine salmonellosis in Great Britain.

Salmonella Dublin Infection

S. dublin is regarded as the most specific sero-type in cattle, although it has been recovered from other animals and occasionally birds. Until recently, *S. dublin* was the commonest sero-type isolated from cattle in Great Britain, but the reasons for the decline in prevalence have not been fully explained.

Clinical Findings

Adults - Adult cattle of all ages and breeds may be affected with an acute or sub-acute form of illness. The vast majority of outbreaks involve one or two animals but when the infection has been recently introduced into a herd a number of animals may succumb.

Acute salmonellosis is associated with a septicaemia and the onset is sudden, characterised by a fever, dullness, anorexia and a marked drop in milk yield. After a period of about 24 hours, a severe diarrhoea develops, with fetid faeces which may contain blood, mucus and shreds of bowel mucosa. Animals that are pregnant may abort. Illness may last for about five to seven days and in the author's experience about 70 per cent of untreated animals may die, whilst in treated animals the mortality rate is reduced to between 10 and 15 per cent. Complete recovery may take up to two months, but many recovered animals will from time to time develop a transient diarrhoea, which does not require treatment.

Sub-acute salmonellosis produces a much milder clinical picture of diarrhoea, drop in milk yield, temporary anorexia with no fever or only a slightly elevated temperature. The vast majority of these cases require no treatment and mortality is very low.

As indicated earlier, abortion may occur in pregnant animals which are acutely affected, but work by one of the author's former colleagues[9] revealed that abortion produced by *S. dublin* may occur without any other clinical signs. In 111 cases of abortion associated with *S. dublin* infection, abortion was the only clinical sign in 86. In contrast to this, however, other workers[10] demonstrated in field studies that abortion was preceded by a fever which lasted for a period of up to eight days.

Calves - Calves between three and six weeks of age are more commonly affected and illness is uncommon in calves during the first week of life. Frequently up to 10 per cent of calves may be affected, and under adverse environmental conditions the morbidity rate may reach 50 per cent or more, with a similar mortality rate.

There is a very wide variation in the clinical picture in calves, but the enteric form of the disease is by far the commonest. Fever, dullness and anorexia, followed by diarrhoea and dysentery are the typical sign with the calves becoming rapidly dehydrated and weak.

Calves which develop the septicaemic form of the disease may exhibit a variety of clinical signs. They may suddenly collapse and die with no evidence of diarrhoea, or they may be fevered and depressed with a very obvious jaundice. Others may exhibit nervous signs only when the infection has localized in the brain. Respiratory signs may be evident when there is lung involvement, but the salmonella infection is considered to be secondary under these circumstances. There may be bone and joint involvement which are manifested as an osteitis and poly-arthritis. Gangrenous lesions of the lower limbs, sloughing of ear tips and tail have been encountered by the author and others, [11,12] which are thought to be due to *S. dublin* infection.

However, on occasions the clinical signs may be so mild and transient that they virtually pass unnoticed.

Post Mortem Findings

The post mortem lesions in the typical adult case in no way can be considered to be pathognomonic. The carcase and parenchymatous organs are usually congested and there is a mucoid/necrotic enteritis. A bacteriological examination is essential for confirmation of a clinical diagnosis.

The main post mortem findings in calves include congestion and dehydration of the carcase, enlargement of the spleen, jaundice, apical pneumonia, small necrotic foci in the liver and kidneys, enlargement of the mesenteric lymph nodes and a mucoid or diphtheritic enteritis. Brain lesions usually manifest themselves as a fibrinous meningo-encephalitis often with fibrinous deposits at the base of the brain and over the fourth ventricle. When joints are affected, the joint cavities and the adjacent tendon sheaths contain a sero fibrinous fluid. Epiphyseal separation, osteo-periostitis and rarefying osteomyelitis of the limb bones have also been described in chronic *S. dublin* infection in calves.[13] As in adult cattle, none of the lesions are specific for *S. dublin* infection and confirmation is dependent upon a bacteriological examination.

Epidemiological Features

S. dublin demonstrates a high degree of host specifity and appears to be particularly well adapted to cattle. Any outbreak of disease in cattle is most likely to originate from other cattle, either directly or indirectly.

Adult cattle which have recovered from clinical disease caused by *S. dublin* infection are likely to excrete the organism in their faeces for years — if not for life. It has been suggested that the successful treatment of clinical salmonellosis in adult cattle with antimicrobial substances may have resulted in the survival of a large number of animals which would have otherwise died, and that these survivors would continue to excrete because although they had been successfully treated, this had not produced a bacteriological cure.[14] The same authors also pointed out that some adult cattle which become infected with *S. dublin* do so without developing clinical disease, but become persistent or intermittent excretors.

For a number of years it was suggested that there was a link between clinical salmonellosis due to *S. dublin* infection and concurrent *Fasciola hepatica* infection.[15,16] This link has now been established, and it has been clearly established that infection with *Fasciola hepatica* increases the susceptibility of cattle to *S. dublin* and predisposes to the carrier state.[17] As carriers will excrete, either continuously or intermittently, it follows therefore that *Fasciola hepatica* infection will not only increase the susceptibility of cattle to *S. dublin* infection, but also influence the excretor rate. It is now recognized that the marked reduction in the incidence of *S. dublin* infection in cattle which has occurred in Great Britain over the last 9 years is in part due to climatic factors which have been unfavorable for the life cycle of *Fasciola hepatica* and therefore reduced its prevalence.

It has been pointed out that epidemiological investigations into *S. dublin* infection in cattle has largely been confined to the study of clinical cases and/or localization in the gall bladder of recovered or excretor animals.[18] Investigation of a herd in North West England revealed that 14 of 59 cows with negative rectal swabs were found to be infected at post mortem examination.[16] Such animals would be classified as latent carriers and these animals would only excrete when stressed by intercurrent disease or some other factor. In a dairy herd in Scotland, 2 carrier animals were detected, one in which *S. dublin* was isolated from the tonsil, and the other in which the organism was demonstrated in the gall bladder.[18] Thus the role of the latent carrier must not be forgotten when the examination of a known infected herd by rectal swabbing fails to detect excretor animals.

When abortion due to *S. dublin* occurs, the organism can readily be isolated from the genital discharges, the foetus and placenta, but in a proportion of cases not in the faeces of cows which abort with no other clinical signs. The vaginal mucus may yield *S. dublin* on culture for a period of four to six weeks after abortion and veterinarians carrying out vaginal examinations during this period should take great care as it is likely that they will develop skin lesions resembling the well-known "brucella rash".

Calves which have recovered from the clinical disease usually excrete the organism for a few weeks after clinical recovery. Occasionally, however, a calf may become a carrier and it has been reported that under experimental conditions calves can become latent *S. dublin* carriers. It has also been shown that once calves become infected within a calf house, the infection may establish itself and persist for long periods without the introduction of further infection.[5]

It is now well recognized that the introduction of *S. dublin* infection into salmonella-free herds is largely through the purchase of infected animals. Such animals may have acquired infection on their home premises, in markets, or in transit. Other domestic animals and free-living animals may

also introduce infection, but the available evidence indicates that they do not play a major role in the spread of disease in cattle. Rats and mice may prolong the persistence of infection on the farm but this is almost certainly of limited duration.

The development of intensive systems of husbandry involving the loose housing of adult cattle has undoubtedly facilitated the spread of salmonella infection, particularly *S. dublin* [16] Under intensive grazing conditions a high percentage of animals may become passive carriers and excrete the organism, but when these animals are housed and tethered in stalls they cease to excrete within a period of about two weeks. This does not happen when cows are loose housed — the infection continues to spread under these circumstances.

The appearance of *S. dublin* infection in home bred calves is almost invariably associated with infection in the adult section of the herd. Carrier animals may give birth to healthy calves which acquire infection soon after birth through contact with such animals, or an infected environment. However, it has been demonstrated that latent adult carriers of *S. dublin* may produce congenitally infected calves which in turn transmit infection to other calves. On a few occasions the author has encountered salmonellosis in calves following the ingestion of milk from a cow with *S. dublin* mastitis.

Environmental factors must also be briefly reviewed. It has been reported that on occasions *S. dublin* infection may be introduced into a herd by means other than through the purchase or movement of animals.[21] For instance, in certain parts of Wales it is thought that the spread of *S. dublin* infection was largely due to the contamination of water courses by grazing animals and by farm effluent.[14] In the same report reference is made to the fact that polluted water may contaminate pastures through flooding and outbreaks of clinical disease may occur in cattle grazing such pastures. The importance of contaminated pastures is illustrated by the fact that there is a well-marked seasonal incidence of *S. dublin* infection in cattle in Great Britain. The majority of incidents in adult cattle are recorded during the summer and autumn reaching a peak towards the end of grazing season, but after the cattle are housed there is a rapid decline particularly in those herds that are stalled. **It is very difficult to assess the importance of feeding stuffs as a source of *S. dublin,* but in Great Britain up to the present time there is no firm evidence to indicate that feed has played a major role in the dissemination of infection.**

A number of physiological, managemental and nutritional factors, as well as intercurrent infections, are important in increasing the susceptibility of cattle to infection and precipitating clinical disease. Pregnancy and a reduction in the plane of nutrition are two such factors, and on at least two occasions the author was convinced that water deprivation during hot weather precipitated clinical disease in dairy herds. As well as *Fasciola hepatica* infection, *babesiosis* and *rickettsiosis* have often been known to precipitate clinical disease in cattle in Wales.

Salmonella Typhimurium Infection

Salmonella typhimurium, in contrast to *S. dublin* cannot be regarded as a host specific sero-type for cattle; nevertheless it is an important pathogen of this species. The organism has been isolated from a variety of animal species including man, poultry and wildlife. At the present time, *S. typhimurium* is the commonest sero-type isolated from cattle in Great Britain.

Clinical Findings

Adults — As in outbreaks of *S. dublin* infection, cattle of all ages and breeds are susceptible. In the main, the disease manifests itself in an acute form with the majority of outbreaks involving more than one or two animals.

The clinical signs in both acute and sub-acute form of the disease resemble those seen in *S. dublin* infection and the two infections cannot be differentiated on clinical grounds. Although abortions may occur with *S. typhimurium* infection, in contrast with *S. dublin* infection these are rare. Because the disease in adults is mainly of the acute type, mortality is higher than with *S. dublin* and unless affected animals are treated, 80 per cent or more may die. Treatment of clinically affected does not in the author's experience appear to be as successful as for *S. dublin.*

Calves — The clinical picture in calves is very similar to that seen in *S. dublin* infection and, as in adults, the two infections cannot be differentiated on clinical grounds. The majority of outbreaks in calves tend to be of the acute type with a high morbibity and frequently a mortality of up to 40 per cent or more, despite treatment. The author has not seen the gangrenous lesions of the limbs or sloughing of the ear tips and tail with *S. typhimurium* infection and only rarely has he encountered arthritis and meningo-encephalitis.

Post Mortem Findings

Pathognomonic lesions are not produced by *S. typhimurium* in adults or in calves. The post mortem lesions are similar to those described under *S. dublin* infection and confirmation is entirely dependant on the isolation and subsequent identification or the organism.

Epidemiological Features

Because *S. typhimurium* has such a wide host range and infection does not persist in recovered animals, it has certain epidemiological features which are different from *S. dublin* which must be emphasized.

In an epidemiological investigation of any outbreaks of disease in cattle, it may be necessary to carry out an examination of other farm animals, poultry, pet animals, rodents, wild birds and, indeed, farm personnel, as well as cattle. Despite the fact that the organism has such a wide host range, epidemiological evidence indicates that the

majority of infections in cattle are derived from other cattle.[7,14,23] The Zoonoses Order of 1975, which provides for the reporting and investigation of Salmonella outbreaks in farm animals in Great Britain, has enabled the State Veterinary Service to collect information which confirms this view. All isolates of *S. typhimurium* are subjected to phage typing and this allows for patterns and trends to be followed. Phage type DT 204 first appeared in cattle in 1975 and in 1976 represented about 12 per cent of the isolations from calves were of this sero-type. Within another eighteen months, however, about 50 per cent of the isolates were of this phage type, and during investigation of outbreaks it was apparent that the infection had been derived from other cattle. In adult cattle the picture is similar but rather less well defined.

Animals which recover from clinical disease due to *S. typhimurium* may continue to excrete the organism for some time, but in the vast majority of cases they do so for only a limited period of time and do not become permanent carriers.[6,7] Similarly, animals which develop mild clinical disease or sub-clinical infection also excrete the organism for a limited period of time only.

There is a very marked seasonal pattern in calf infection in Great Britain.[3] The peak period coincides with the autumn calf sales and this appears to be repeated each year. The importance of movement of calves cannot be over-emphasized in the maintenance and dissemination of infection in calves in Great Britain. In calf rearing and fattening units, clinical infection will die out fairly quickly if the movement of calves onto the premises is restricted.

Other possible sources of infection, however, must not be overlooked in any epidemiological investigation. Humans have on a number of occasions been the source of infection for calves. In incidents known to the author, calves become infected when taught to drink milk from a bucket by attendants who were infected. Leakages from cess pits and overflowing drains from dwellings have also been incriminated.

Environmental contamination and wildlife may also play a role in bovine *S. typhimurium* infection. Grazing cattle often obtain drinking water from streams and rivers which may receive effluent from sewage and meat processing plants. Again, data collected under the Zoonoses Order confirm that streams and rivers can be a source of infection. The role of wildlife in bovine salmonellosis is not clear. Birds, rats and mice are frequently found to be infected with salmonellae, particularly *S. typhimurium*. In the main it is thought that the infection in these species is a reflection of the contamination in their environment. However, the author investigated outbreaks of disease in cattle at pasture, which appeared to have been introduced by seagulls.[13] These birds had migrated onto the pastures when supplementary feeding in troughs had commenced and the feed — ground barley — was heavily contaminated with gull faeces. The same phage types of *S. typhimurium* were isolated from the gull faeces and cattle; and secondary outbreaks occurred when the gulls moved onto other farms. Other authors have incriminated birds and rodents in a similar manner.

The important role of animal feeding stuffs in animal salmonellosis is now well recognized. However, it was only recently recognized that cattle feeding stuffs and feed ingredients could be a source of *S. typhimurium* as well as other sero-types. It has been demonstrated that unless special techniques are used, *S. typhimurium* may not be isolated from bone or bone meal.[25] The inclusion of poultry offal in cattle feeds and the feeding of dried poultry manure could well increase the risks of transmission of *S. typhimurium* unless these products are heat-treated. Ensiled poultry litter is now also fed to cattle and unless the ensiling process is satisfactory it may still contain viable pathogens such as *S. typhimurium* and other salmonellae when fed.

The spreading of farm waste and sewage onto grazing land is a possible source of *S. typhimurium* as well as other salmonellae. Outbreaks of *S. typhimurium* in cattle under such circumstances have been described.[26]

Other Sero-types

A large number of other sero-types have been recovered from cattle in most countries of the world. Data collected under the Zoonoses Order 1975 show that over 40 sero-types were isolated from cattle during 1978. The vast majority of these sero-types were introduced into Great Britain through the importation of feed ingredients such as meat and bone meal and fish meal, or through the importation of livestock. These so-called "exotic" sero-types are now established in livestock, environment and, indeed, in the human population and are now being recycled.

Clinical and Post Mortem Findings

The clinical signs produced by these sero-types are similar to those produced by *S. dublin* and *S. typhimurium*. In general, however, the disease is less severe in both calves and adults. Post mortem features again closely resemble those produced by *S. dublin* and *S. typhimurium*.

Epidemiological Features

The spread of sero-types other than *S. dublin* and *S. typhimurium* can occur through contact with other cattle or, indeed, other animals. In Great Britain, however, when infection by these sero-types occurs in a herd it is more likely to have been introduced in feeding stuffs than any other source.[27] In an outbreak in cattle in a dairy herd during 1977, 13 sero-types were isolated from sick animals and 17 from recently delivered concentrate feed. The inclusion of poultry offal, dried poultry manure and poultry litter also increases the risk of transmission of these sero-types to cattle. Sewage or sewage effluent are also possible sources. An outbreak of *S. paratyphi B* in cattle in Yorkshire was linked to contamination of a stream by sewage from a dwelling in

which a known carrier of the organism lived.[28] Cattle in South West England became infected with *S. aberdeen* when a sewer overflowed onto a pasture[29] Wildlife too may be important. *S. livingstone* was transmitted from a refuse tip to grazing cattle by gulls.[30]

It is important then, that when these incidents are investigated, all possible sources of infection should be considered and appropriate samples taken for laboratory investigation.

Animals which recover from clinical salmonellosis with these sero-types do not as a rule become carriers. A recent report of *Salmonella saintpaul,* however, indicated that some individual animals remain excretors,[31] and in this herd one cow was found to have an udder infection.

Laboratory Diagnosis

Cultural Examination

Confirmation of salmonellosis is achieved primarily by the isolation of the organism. In the live animal, cultural examination of faeces or rectal swabs is the most frequent method used. In adult cattle it has been shown however that the examination of freshly voided faeces is a more reliable method than the examination of rectal swabs.[32] The disadvantages of rectal swabs are that they may have little feacal material on them, and if they are in transit for a lengthy period may dry out and therefore not yield viable salmonellae. It should also be remembered that in the early febrile stage of the disease, before the onset of diarrhoea or dysentery, salmoneallae may not be detected in faeces or rectal swabs. In most clinical cases of salmonellosis in adult cattle, salmonellae can be isolated directly on selective media without pre-enrichment, during the diarrhoeic phase of the disease. Salmonellae may also be cultured from blood samples taken during the febrile stage.

Salmonellae may be readily isolated from the placenta, foetal stomach or vaginal mucus when abortion occurs. Care, however, must be taken when taking vaginal mucus, either by swabbing or suction tube, to prevent faecal contamination. Similar precautions must be taken with the placenta.

The cultural examination of faeces/rectal swabs from calves is less satisfactory and it has been shown that up to 56 per cent of calves may be negative on culture.[33] It is suggested, therefore, that where there is illness in only one or two calves in a group, up to 6 calves should be sampled. A number of authors[33,34,35] have demonstrated that salmonellae may be excreted in the saliva, so that mouth swabs can also be submitted for cultural examination. It may be necessary to passage faeces or rectal swabs through enrichment media before plating on selective media.

As wide a selection as possible of specimens from carcases should be submitted and these should be sent in individual containers to prevent cross-contamination. Such a selection would be portions of lung — especially pneumonic areas — liver, gall bladder from adults, spleen, kidney, a portion of small intestine or a swab of the intestinal contents. If there are obvious lesions such as an arthritis, joint fluid or a joint, swab should be included. Similarly, if during life there were nervous signs exhibited, either the whole brain or a brain swab should also be submitted.

Serological Diagnosis

The most frequently used serological test for salmonellosis is the serum agglutination test (SAT). There has been a considerable amount of discussion about the value of the test, but it does seem at the present time that it had considerable merit and certain advantages over other tests. The test is carried out with somatic (O) and flaggellar antigens (H) of the salmonellae and therefore the identity of the particular sero-type involved must be established before the tests can be reliably applied.

Most of the work on the SAT has been carried out with *S. dublin.* It has been demonstrated that most healthy cattle under 12 months of age have either no agglutinins or agglutinins in low titre to salmonellae. In older cattle, agglutinins may develop, and it is not uncommon in parts of Wales to find healthy cattle with *S. dublin* "O" titres of up to 1:40 and "H" titres of 1:80 or 1:160. During the early stages of the disease the "O" and "H" titres may be low, but during the course of illness these rise and peak 2 - 4 weeks after the onset. In recovered clinical cases the *S. dublin* "O" titre may be in the range of 1:80 to 1:320, and the "H" titre 1:640 to 1:5120. The results with other sero-types are very similar.

Other tests which have been used include the complement fixation test (CFT) and indirect haemagglutination test. These do not appear to have any advantages over the SAT and are more laborious to perform.

In the author's opinion, serological tests are of limited value, certainly in the diagnosis of clinical salmonellosis. None of the tests are reliable in the early stages of the disease, and furthermore some recovered animals with demonstrable antibodies are no longer infected. Some animals which are infected — the latent carriers — may be serologically negative.

Antimicrobial Thereapy

A wide range of antimicrobial substances have been used in the treatment of clinical cases of bovine salmonellosis. The author has had a limited experience of treating affected animals in recent years and therefore most of the information has been gleaned from published reports and from colleagues in private practice.

In a survey of 300 veterinary practices in Great Britain the drugs most frequently used to treat and control salmonellosis in calves were, in order of frequency: — furazolidone, ampicillin, chloramphenicol, framomycin, neomycin, streptomycin, sulphonamide preparations and tetracyclines.[36] At that time the same range of drugs were

used in adult cattle. Since that survey was published in 1967 there has been little change except that since the introduction of trimethoprim/sulphadiazine compounds, these have largely replaced the sulphonamide preparations and tetracyclines are now more widely used. There is little firm evidence on the comparative efficiency of these drugs, but most practitioners believe that chloramphenicol, tetracycline and trimethoprim/sulphadiazine are most effective. Supportive treatment to counteract dehydration, intestinal demulcents and astringents, and careful nursing are equally important however if treatment is to prove successful.

The value of treatment, particularly of adult cattle infected with *S. dublin* has often been questioned. Most adult cattle infected with *S. dublin* remain carriers even after intensive treatment with antimicrobial drugs and therefore remain a source of infection to others. Therefore serious consideration should be given to the removal and/or slaughter of such animals, once they have recovered.

A wide range of antimicrobial substances have been fed prophylactically to calves to reduce "stress" and thereby prevent the onset of clinical salmonellosis. It has been difficult to assess the effectiveness of prophylactic therapy, but in the author's view it is of limited value and no substitute for a high standard of husbandry and management. There is no doubt however that the feeding of anitbiotics has influenced the emergence of resistant strains. During the period of 1961-1965 there was a marked increase in the prevalence of *S. typhimurium phage type 29* in cattle in Great Britain, and 99.7 per cent of these were resistant to one or more drugs.[23] It has also been reported that the feeding of tetracycline led to the emergence of salmonellae possessing transmissible drug resistance.[37]

The appearance of chloramphenicol resistance in certain strains of *S. typhimurium* was reported in Great Britain in 1978.[1] Initially, the resistant strain — phage type DT 204 — was isolated from calves on a dealer's premises, but subsequently the strain was isolated from a large number of premises throughout the country. There is no doubt that the appearance of the organism on other premises wa associated primarily with calf movements from the dealer's premises and subsequently through other dealers and through markets. The emergence of the chloramphenicol resistant strain has been explained.[2] It is thought that a strain resistant to sulphonamide and tetracycline acquired an R factor with a coding for resistance to chloramphenicol, streptomycin, sulphonamides and tetracyclines, through selective pressure exerted by chloramphenicol treatment of a sick animal. Other phage types of *S. typhimurium* have also acquired chloramphenicol resistance and a resistant strain of *S. dublin* appeared on one farm where previously a chloramphenicol resistant strain of *S. typhimurium* had been isolated.[3]

The appearance of chloramphenicol resistant strains of salmonellae has obviously serious public health implications. As far as the cattle population in Great Britain is concerned there has been no evidence that chloramphenicol strains have spread into adult cattle.

Chloramphenicol resistant strains do not appear to be more virulent for calves than chloramphenicol sensitive strains, and investigations on infected farms failed to identify persistent infection on any.[3]

The Control of Bovine Salmonellosis

The control measures for bovine salmonellosis have been well documented[8,14] and are therefore only outlined in this paper. In general, the basic measures are the same irrespective of the sero-type involved, but there may be differences in detail. *S. dublin* infection in adult cattle is likely to produce carriers and these must therefore be taken into consideration. *S. typhimurium* on the other hand has a wide host range and therefore other species of animals may have to be included in a control programme.

Clinical Disease in Calves and Adult Cattle

All animals which are clinically affected should as far as possible be immediately isolated or segregated so that contamination of the premises is reduced as much a possible. **It is also important that animals which have recovered should be retained in isolation for a period of at least two weeks after an apparent clinical recovery.** In the case of *S. dublin* infection the slaughter of clinical cases should be considered because they will probably remain carriers. Some owners may opt for slaughter of such animals after they recover and the carcases used for human consumption. Clinically affected animals should never be slaughtered at abattoirs nor the carcases passed for human consumption. The isolation accommodation and other buildings/yards exposed to infection should be thoroughly cleansed and disinfected and the faeces and effluent disposed of in such a manner as to reduce the risks to other livestock to a minimum. **Ideally no cattle should be removed from, or brought on to the premises until the cleansing and disinfection is complete.**

The source of infection in calves and, indeed, of *S. dublin* infection in older cattle is mainly adult "carriers" so that attempts should be made to detect permanent excretors by cultural examination of faeces or rectal swabs. This examination should be delayed until two or three weeks after the recovery or disposal of clinical cases. It has been suggested that adult cattle should not be regarded as active carriers unless salmonellae are isolated from individual animals on three successive tests carried out at intervals of about 14 days. Excretors identified in this manner should then be removed and slaughtered. The success of such an approach is completely dependent on the identification of all carrier animals, some of which may be intermittent or transient excretors. It may be necessary therefore to examine adult cattle as they calve, as excretion may occur only at or around calving. When this method fails to control *S. dublin* infection in a herd, there is frequently circumstantial evidence of infection being reintroduced into herds.

Preventive measures in Self-Contained Herds

In self-contained herds, few animals are purchased and therefore infection may be introduced by means other than

through the introduction of livestock. When bulls or other animals are introduced they should whenever possible be isolated from the rest of the herd until they are shown to be free of infection. Drinking water should be from a piped supply and farm waste or sewage sludge should be applied to arable land or pasture which is to be used for conservation purposes. If farm waste or sewage sludge is applied to pasture, then it should have been stored for at least 4 weeks before application and there should be an interval of at least 4 weeks between application and grazing. Stock should not graze pastures which have been flooded if it is at all possible, and because of the relationship between *fascioliasis* and *S. dublin* infection, appropriate control measures for *fascioliasis* should be implemented. Stock should be maintained on an adequate plane of nutrition and feeding stuffs should ideally be protected against contamination by rats, mice and birds.

Preventive Measures for Purchased Calves

Under normal marketing conditions it is impossible to obtain salmonella "free" calves and preventive measures are usually aimed at preventing clinical disease. Calf rearers should therefore purchase strong healthy calves and rear them under good standards of husbandry and management. Whenever possible, calves should be bought in batches on an "all in, all out" basis and the calf houses thoroughly cleansed and disinfected between batches to prevent a build-up of infection. Routine preventive medication with antibiotics or antibacterial drugs has no part to play in the prevention of salmonellosis and is in fact contra-indicated. Vaccination of calves either before they leave the farm of origin or soon after arrival on the rearing farm is advocated where it is known that calves are likely to be exposed to infection.

Vaccination

Both live and dead vaccines have been used in the prevention of bovine salmonellosis, but it appears from the published reports that live vaccines appear to produce a greater degree of immunity than dead vaccines. Under field conditions, the author has no doubt that live vaccines are superior.

The live vaccine derived from an attenuated strain of *S. dublin* was shown to produce a good immunity against artificial challenge by the same organism,[39] and it also protects against *S. typhimurium infection. The vaccine has been available in Great Britain for over 10 years and has been widely used in calves. It has been difficult to assess its efficacy under field conditions because of the varying conditions under which it has been used. Most practicing veterinarians and farmers would agree that it is of considerable value in controlling S. dublin and S. typhimurium infections, despite the fact that in some farms the results have been rather disappointing and on others there have been adverse reactions.* Occasionally deaths have occurred within 48 hours of vaccination of 2-3 day old calves. The cause of death was an acute bacteraemia, and the organism responsible identified as the vaccinal strain of *S. dublin.* In all incidents the calves were shown not to have had any, or very little, colostrum and it is therefore important when recently-born calves are vaccinated that they should have received adequate colostrum. In older calves, reports of respiratory and enteric disease after vaccaination were made to the author. Many of these calves had however been ailing before they were vaccinated and they therefore should not have been vaccinated at that time. There was no indication that the vaccine had directly produced clinical disease but only exacerbated the clinical signs of existing viral infections. The vaccine is not generally recommended for use in adult cattle, but it has been used in the face of clinical disease in several herds known to the author. There were no adverse reactions recorded and although clinical disease disappeared shortly after vaccination, it is not known whether this was directly due to vaccination.

There is no doubt that vaccines have an important part to play in the control of bovine salmonellosis. However, they cannot be a substitute for good husbandry, management and hygiene.

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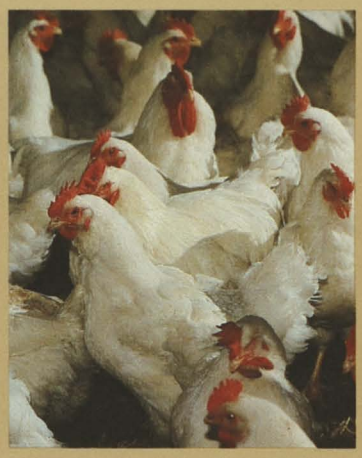

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