OVINE MASTITIS, WITH SPECIAL REFERENCE TO MASTITIS CAUSED BY *PASTEURELLA MASTITIDIS*.

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SUMMARY.

Six outbreaks of mastitis of sheep are described. Two outbreaks were caused by Pasteurella mastitidis **and one by** Staphylococcus aureus. **In the remaining three outbreaks,** Past. mastitidis **was associated with** Corynebacterium pyogenes **in one,** Staph. aureus **in another and** Staph. aureus **and streptococci in the third.**

INTRODUCTION.

Ovine mastitis has been reported from Europe (Lesbouyries, Berthelon and Macrides 1935), Russia (Milovzorov and Tchasnovnikov 1933), Great Britain (Leyshon 1929) and America (Marsh 1932; Smith and Harnden 1943; Tunnicliff 1949).

Many authors (Haupt 1932; Miessner and Schoop 1932; Marsh 1932; Milovzorov and Tchasnovnikov 1933; Macrides 1936; Smith and Harnden 1943; Tunnicliff 1949) have reported that *Pasteurella mastitidis* is the causative organism. Other organisms that have been incriminated are *Micrococcus ovis* (Haupt 1932), *Clostridium perfringens* (Weinberg, Forgeot and Moureu 1937) and *Corynebacterium ovis* (Rosati and Bertolino 1938). *Staphylococcus aureus* was isolated from the majority of cases of ovine mastitis in Tasmania (Dumaresq, personal communication 1948) and also from cases in New Zealand (Gilruth 1910), England (Leyshon 1929) and Italy (Boi 1940).

During the last five years material from six outbreaks has been examined at this Station. Information on these outbreaks is recorded.

HISTORY OF OUTBREAKS.

Outbreak 1.

An Inspector of Stock in south-western Queensland reported on an outbreak in 1948 as follows:—

> Seventy-two in a flock of 2,400 ewes, of mixed ages, had died and six had recovered. The incidence was highest in ewes suckling lambs $2\frac{1}{2}$–4 months of age. Symptoms were swelling of the udder, stiffness of the hind legs, anorexia, fever and trembling. On autopsy the udder of one ewe was swollen, firm and showed evidence of gangrene. The supramammary lymph nodes were swollen. No other abnormality was noted.

Past. mastitidis was isolated from one of two milk samples submitted to this Station. Three millilitres of a broth culture of this organism was inoculated into the teat canal of the right half of the udder of a lactating ewe. After 48 hours the milk was thickened and showed a slight greenish tinge, but the organism was not recovered.

Outbreak 2.

This outbreak occurred in 1948 on another property in south-western Queensland. The Sheep and Wool Adviser reported as follows:—

> The lambs were marked and vaccinated against contagious ecthyma between 14th October and 6th November. Two days after the completion of these operations, some ewes would not allow the lambs to suckle, and on examination scabs were found on the teats. About 70 ewes died, and all of these animals had discoloured udders. The mortality was investigated on 24th November. Examination of one ewe, which was dull and standing in the shade, showed that the left half of the udder was black on the ventral portion and the remainder was discoloured. The medial portion of the right half was purple, and the affected parts were firm to the touch. On autopsy the internal organs were normal.

Pipettes of the liver, mesenteric lymph node and udder yielded *Staph. aureus*. Histopathological examination of the mammary gland showed an acute mastitis with hyperaemia, oedema and haemorrhage. There was massive necrosis, marked accumulations of neutrophils, and plugging of the alveoli with exudate and cellular debris.

Outbreak 3.

This outbreak occurred in 1952 and involved 6- and 7-year-old ewes yarded for shearing. They were in fair condition and had 4-month-old lambs at foot.

About 40 ewes and a number of lambs died. The Sheep and Wool Adviser reported the following observations on one ewe:—

> Symptoms were listlessness, laboured breathing and moist discharge from both nostrils. The left half of the mammary gland was bluish red, and there was a black scab on the end of the teat. The medial portion of the right half of the gland was dark pink. A swelling 8 inches long and 5 inches wide was present anterior to the udder.

Corynebacterium pyogenes was isolated from the left half of the udder and *Past. mastitidis* from the right half.

Outbreak 4.

In 1952 a 4-tooth ewe in a flock of 350 Merino ewes with 1–2-month-old lambs at foot was noticed lame. Three days later this animal was examined by a Sheep and Wool Adviser, who reported as follows:—

> The ewe had anorexia and was dull. The left side of the udder was hot, red and swollen, and the swelling extended 6 inches along the abdomen. The other side of the udder contained no milk, but was normal on palpation. This ewe responded to two subcutaneous injections at twelve-hour intervals of sodium sulphamezathine ($33\frac{1}{3}$ per cent. solution) at the rate of 1 ml. per 5 lb. body weight. Two further cases of mastitis occurred in this flock, but no treatment was given, and one ewe died.

Past. mastitidis, Staph. aureus and streptococci were recovered from the milk sample submitted to the laboratory.

Outbreak 5.

A ewe from the experimental flock at this Station was culled in 1953 and autopsied because of old age, faulty teeth and emaciation. One half of the udder was distorted and firm and contained irregular cavities filled with greenish-yellow semi-fluid pus. Fibrous tissue had replaced almost all of the remaining mammary tissue. *Past. mastitidis* was recovered from the pus.

Another ewe in good condition was found dead. On autopsy both halves of the udder were enlarged and firm, the cut surface showing mottling with small areas of fibrosis. Brownish fluid was present. *Past. mastitidis* and *Staph. aureus* were isolated from the diseased tissues.

Outbreak 6.

This occurred in 1953 in a flock of 100 stud ewes, 2–8 years old with 2–4-week-old lambs at foot. The first symptoms noted were lameness and swelling of one half of the udder, the skin being bluish-black. Of seven cases seen, six died. There was a history of cases having been seen in previous years on this property. When recovery occurred there was sloughing of the affected half of the udder.

Past. mastitidis was isolated from milk samples from each of three ewes. The milk was green, contained many clots and films showed numerous leucocytes, lymphocytes predominating. A 24-hour broth culture injected into the left teat canal produced an acute mastitis with later sloughing of that half.

BACTERIOLOGICAL RESULTS.

The bacteria isolated from the six outbreaks are listed in Table 1.

Table 1.

BACTERIA ISOLATED FROM SIX OUTBREAKS OF OVINE MASTITIS.

Outbreak.	Bacteria.
No. 1	*Past. mastitidis*
No. 2	*Staph. aureus*
No. 3	*Past. mastitidis, C. pyogenes*
No. 4	*Past. mastitidis, Staph. aureus,* streptococci
No. 5	*Past. mastitidis, Staph. aureus.*
No. 6	*Past. mastitidis*

The strains of *Staph. aureus* were Gram positive cocci and produced typical colonies and haemolysis on sheep's blood agar.

The *C. pyogenes* strains were Gram positive diphtheroid bacilli, which produced acid in glucose, maltose, sucrose, mannitol and lactose, liquefied Loeffler's inspissated serum and produced haemolysis on sheep's blood agar. The single strain of streptococci was not identified.

The strains of *Past. mastitidis* were all Gram negative, non-motile, non-sporing bacilli. After 24 hours' incubation at 37°C., colonies on 10% sheep's blood agar were 2–3 mm. in diameter, greyish, round, convex and entire. A zone of haemolysis usually occurred beneath the colony. The biochemical characteristics are given in Table 2. As cultures were not retained, only those characteristics tested at the time of isolation are given. Acid production was often slow, particularly with lactose medium. Andrade's indicator was used in carbohydrate media, and incubation was for at least seven days at 37°C.

The strain from outbreak 6 was non-pathogenic for mice, guinea pigs and rabbits when inoculated intraperitoneally. One rabbit inoculated with 1 ml. culture intravenously was not affected.

DISCUSSION.

Miessner and Schoop (1932) differentiated two clinical forms of this disease—*mastitis infectiosa* caused by *Past. mastitidis* and *mastitis gangraenosa* produced by *Past. mastitidis* plus staphylococci or by staphylococci alone. However, other species of bacteria rapidly become established in the damaged mammary tissue, particularly in mastitis caused by *Past. mastitidis* (Lesbouyries, Berthelon and Macrides 1935; Tunnicliff 1949). *C. pyogenes* in particular is often a secondary invader and causes severe damage of the udder tissue. Hence, specimens for bacteriological examination should be taken from early cases.

The economic loss caused by ovine mastitis, particularly in Australia, is difficult to assess, but the absence of reports suggests that it is relatively rare. Tunnicliff (1949), in an excellent review of pasteurella mastitis in ewes in the United States, stated that his records showed that over a period of 17 years, 2–3% of 19,550 lambing ewes developed mastitis. The mortality in these ewes was 21%.

Carroll (1949) and Belschner (1950) described mastitis in Australian sheep but gave few details of the etiology of the disease.

That ovine mastitis may be a cause of economic loss is suggested by data collected by Moule (1954) during a survey of neo-natal mortality of lambs. He stated that 351 out of 1,587 ewes had abnormal udders. Although some of these were attributed to chronic infection, no information on the number of such cases or etiology was given. Miessner and Schoop (1932) considered that surviving ewes may not be suitable for breeding purposes because they may be unable to support a lamb. In outbreaks of pasteurella mastitis, pneumonia caused by the same organism often occurs in the lambs (Haupt 1932; Miessner and Schoop 1932; Lesbouyries, Berthelon and Macrides 1935).

There was no apparent common predisposing factor in the outbreaks described by us. Marsh (1932) suggested that bruising of the udder by large lambs may be a predisposing factor, while Miessner and Schoop (1932) noted that hungry lambs were often the cause of wounds through which infection could enter. The latter authors stated that infection occurred irrespective of the standard of hygiene maintained. In our outbreak 2 there was a possibility that the ewes had teat lesions caused by contagious ecthyma following vaccination of the lambs. These lesions would be favourable sites for the entry of the staphylococci. Dumaresq (1948) recorded sores and scabs on the tips of the teats in up to 60% of maiden Merino ewes, but the etiology of these lesions was not known.

Opinions differ on the value of immunisation for the prevention of pasteurella mastitis. Carefully killed cultures were used by Miessner and Schoop (1932) to vaccinate 800 ewes. Results indicated that a considerable degree of immunity was produced, particularly in ewes given three injections of vaccine. Also the disease was milder in vaccinated than in non-vaccinated animals. Lesbouyries, Berthelon and Macrides (1935) used Miessner and Schoop's method of vaccination but no definite conclusions could be drawn from their results. Tunnicliff (1949) found that formolised suspensions were not satisfactory as immunising agents, but that a living avirulent strain produced approximately 50% immunity against severe artificial infection. The viability of the living vaccine was not long enough to allow effective field application.

Formolised autogenous vaccines produced a favourable response in the treatment of pasteurella mastitis in sheep and goats but did not prevent the gland becoming functionless (Smith and Harnden 1943).

Table 2.

BIOCHEMICAL CHARACTERISTICS OF FIVE STRAINS OF *Past. mastitidis*.

	Strain.				
	1.	3.	4.	5.	6.
Arabinose ..	—	A		A (L)	A (L)
Xylose	A	A	A (S)	A	A
Rhamnose ..	—	—	—	—	—
Glucose	A	A	A	A	A
Fructose ..	A	A	—	A	A
Mannose ..	—	A	—	—	—
Galactose ..	A (S)	A	A	A	A
Sucrose	A	A	A	A	A
Maltose	A	A	A	A	A
Lactose	A	A	A (L)	A (L)	—
Trehalose ..		A		—	
Raffinose ..	A	A		A	A
Starch	—	—		—	A
Inulin	—	—		—	—
Dextrin		—		—	—
Glycogen ..		—		A (S)	—
Glycerol ..	A	A	—	A	A
Adonitol ..	—	—		—	—
Mannitol ..	A	A	A	A	A
Dulcitol	A (S)	—	—	—	—
Sorbitol	A	A	A	A	A
Salicin	A (S)	—	—	—	—
Inositol	—	A		A	A
Indole	—	—	—	—	—
H_2S		—		—	—

Key. — = negative; A = acid production; A (L) = late acid production; A (S) = slight acid production

As sheep are not usually watched closely for sickness under Australian conditions, the rapidly developing disease is often too advanced for treatment to be beneficial. Antiserum prepared in horses was not satisfactory in the treatment of ewes (Miessner and Schoop 1932). Acriflavine given intravenously did not cure one experimental or 13 natural cases (Marsh 1932). Sulphamezathine given orally and intravenously produced favourable response in experimental cases, but treatment was ineffective unless started within 24 hours of the appearance of symptoms (Tunnicliff 1949).

Measures for the control of mastitis due to *Past. mastitidis* should include the slaughter of survivors. Tunnicliff (1949) described a case where an experimentally infected ewe carried the organism for 10 months without any clinical evidence of infection until handling of the udder produced acute mastitis. Avoiding hunger and thirst of the lambs may prevent udder damage and so decrease the chance of infection (Miessner and Schoop 1932).

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