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Sub-Clinical Ovine Psoroptic Mange and it's Role in the Epidemiology of Disease.

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Introduction

Psoroptes ovis is an obligate ectoparasitic mite causing a debilitating dermatitis involving hair or wool loss and a pruritic scab formation. The parasite occurs in all the sheep rearing countries of the world, although it was eradicated from Australia and New Zealand towards the end of the nineteenth century. Ovine psoroptic mange (sheep scab) can be a cause of considerable suffering and even mortality within infested flocks. Flock productivity can be severely affected, either directly through reduced lamb crops or downgraded wool or leather or indirectly through the use of expensive chemical control programmes with their associated withholding periods for meat, milk or fleece.

Although the mite possesses long, sharp, barbed chelicerae, capable of piercing and scraping the skin, and fused palps forming a sponge like "lapping" organ, the pseudorutella (Rafferty and Gray, 1987), the clinical signs of sheep scab are caused only partly, if at all, by the direct action of mite feeding. Scab is a form of allergic dermatitis, initiated by allergens contained in the mite faecal pellets, which in *P.ovis* are enveloped in

part of the mite's own mid-gut (the peritrophic membrane or PM). *P.ovis* exploits this allergic reaction: the heat and humidity produced by the inflammation forming the micro-climate needed for mite survival and the leakage of serous exudates, forming the basis of the mites nutrition. In short the mite cannot survive without the inflammation.

Preliminary results from an ongoing survey (at present involving eight flocks with a total of 1616 sheep) have shown the prevalence of sheep presenting scab lesions within infested flocks to be between 7.8% and 60.0%. These results also highlighted the prevalence of sub-clinical lesions (ie. lesion areas below 100 cm^2 or 2.5% body cover) within infested flocks to e between 10.0% and 90.6%. Sub-clinical disease is generally asymptomatic, symptoms if they do occur include occasional episodes of restlessness, rubbing against fence posts etc, soiled and stained areas of wool (particularly on the shoulders), head tossing and deranged or tagged fleece. Differential diagnosis can be problematic as these symptoms are also indicators of the presence of other ectoparasites (eg. chorioptic mange (*Chorioptes bovis*), chewing lice *(Bovicola ovis*), blowfly strike (*Lucilia* spp), keds (*Melophagus ovinus*), biting flies, or even scrapie! It is of paramount importance that the cause(s) of flock irritation are identified. Administration of an inappropriate control strategy may select for acaricide resistance. Sheep with sub-clinical scab can look perfectly normal and can unknowingly be introduced to other flocks and remain undetected for considerable periods of time, or they may be introduced to flocks, where the factors that constrained the original sub-clinical infestation may not be in operation, allowing disease to spread rapidly within the contact flock. This paper outlines a series of studies carried out at the VLA (Weybridge) to investigate the nature of sub-clinical *P.ovis* infestations and the factors that may maintain sub-clinical infestations in the United Kingdom.

The Phases of Sheep Scab

In many host-parasite relationships there is a period of parasite increase, followed by stability, then by decline (Matthyse *et al.,* 1974). Six such phases have been observed in the temporal progression of sheep scab, as populations of *P. ovis* adapt to the changing ecological conditions confronting them (Bates, 1997a, 1999c). These phases consist of:-

1. The sub-clinical (colonisation, lag, pre-patent or sensitisation) phase.
2. The rapid growth phase.
3. The peak (plateau, maturity, climax) phase.
4. The decline (death or decay) phase.
5. The regressive phase
6. The cryptic phase.

These phases can be deduced by recording the temporal changes in, a) the numbers of live adult female *P.ovis in situ* around the periphery of the lesion, b) the area of the scab lesion itself and c) the anti-*P.ovis* IgG titre, deduced by ELISA (Bates, 1997a, 1999c).

The Sub-Clinical Phase

The sub-clinical phase is characterised by low mite numbers and small lesions (below 2.5 % body cover). Initial observations of lesions in this phase were similar to those recorded by Downing (1936) and Spence (1949), with mites feeding within hours of contact and forming a small raised vesicle filled with fluid. Excess serous fluid, leaking from the vesicle drying to form a yellow scab (approximately 1.0 cm^2), often with a moist, faintly green, periphery. Pruett *et al.,* (1986) postulated that the resulting seepage of serous fluid could enhance the feeding environment of the mite. One of the obvious characteristics of the sub-clinical phase is that lesions are virtually undetectable. In laboratory infestations, using the 25 adult female *P.ovis* of the VLA Reference isolate, no obvious clinical signs of infestation were observed until 48 days post challenge, where a noticeable discolouration of the wool was observed over the site of challenge. A humoral immune response is known to be elicited against *P.ovis*, but circulating antibodies were not detected until the lesion itself covered 1.27% of the body and the infestation entered the rapid growth phase (Bates, *unpublished observations*). Vishnyakov (1998) demonstrated that there was also a change in the immune status at the T-lymphocyte sub-population level within 30 to 40 days after artificial infestation.

In laboratory studies the sub-clinical phase can last for 14 to 40 days, during which the mite adjusts to the new host and the host responds immunologically to the mite. If the sheep is unable to mount an allergic response the micro-climate and feeding environment

is not initiated and the mites cannot colonise. If on the other hand, the sheep is immunologically responsive (susceptible), an active lesion is produced. These clinical observations supported those of Stockman (1910), who showed that experimental infestations remain undetectable until between 25 to 30 days post challenge, even with a challenge higher than expected in the field, but the time period between contact to presentation of clinical signs in the field, however, can be 60 to over 240 days. Progression to the rapid growth phase may be slow on sheep of low scab susceptibility but considerably shorter on sheep susceptible to infestation.

Following the sub-clinical phase the disease may then enter the "Rapid Growth Phase," characterised by a rapid increase in mite numbers and lesion spread, an increase in circulating IgG, and a definite increase in clinical signs of infestation. Body heat will dry the serous exudate to form the scab. *P.ovis* (being semi-liquid pool feeders) cannot feed on the hardened scab, and together with the hyper-keratinised skin and build up of toxic faecal material, are forced to aggregate at the periphery of the expanding lesion. The lesion gradually spreads outwards as the mite population increases. Rubbing and head tossing become more excessive, areas of wool loss may appear, together with open, bleeding wounds. Sheep rapidly lose condition and epileptiform seizures may be evident (Bygrave *et al*, 1993). In the later stages mites begin to migrate to the "cryptic sites" (the pinnae, infra-orbital fossae (IOF), inguinal fossae and the perineum) and the external auditory canal (EAC). Spence (1949) observed that colonisation of the cryptic sites occurred 45 to 60 days after the onset of the rapid growth phase. High populations of mites during this phase render it more likely for them to pass to other sheep (Berriatua *et al.*, 1999). Antibody titres continue to increase, but eventually the lesion growth slows down or stops completely and the mite population decrease rapidly as the disease enters the "Decline Phase."

The general appearance of the lesion changes, the active moist edge becomes indistinct and scaly. Aural haematoma and secondary bacterial infections may also occur (Bates, 1999c). Mite populations decrease, partly due to the lack of feeding sites, but also due to the immune response elicited by the host. Immunoglobulin has been shown to be secreted onto the skin in concentrations comparable to circulating levels (Watson *et al.*, 1992), and this may mediate immunological protection. Dermal immunoglobulin may affect mite

fecundity and survival, by attacking the mid-gut cells of the mite (which also forms the PM of *P.ovis*), inhibiting nutrient absorption and ultimately egg production (Stromberg and Fisher, 1986). The lack of feeding sites and the host immune response may also force the mites to disperse at random over the entire body and many continue to reach the cryptic sites. Eventually new wool growth begins in previously denuded areas and the scab continues to lift away from the skin as the wool grows (the "Regressive Phase").

Sheep appear to recover completely, but they may still be harbouring small populations of mites, under dry scabs or in the cryptic sites, waiting to re-infest the sheep once normal skin conditions are restored ("pseudorecovery"). It is not unusual for the mite population to disappear completely and an animal to make a full natural recovery without acaricidal treatment. In this "Cryptic Phase", previously infested sheep may appear clinically normal, but mites can still be found in the EAC or IOF.

Factors Affecting the Growth of Scab Lesions

The Effects of Age, Sex and Breed.

O'Brien (1996) and Bates (1999c) observed no differences in susceptibility between male and female sheep. The existence of patent infestations on young lambs is equivocal. Downing (1936) saw lesions on young lambs four and seven days old, but recent clinical studies have shown lambs under one month old to only present circumscribed areas of clean tagged wool ("leopard lambs"), but with no definite lesions observed on closer examination (Bates, *unpublished observations*). Sheep scab is primarily a winter disease although a significant number of cases do occur during the summer (Bates, 1991). Mites infesting sheep with short wool are exposed to sunlight, low humidity and extremes of heat and thus may be very slow to colonise and exhibit a very long sub-clinical phase with the rapid growth phase only occurring when the fleece length is long enough to matt together and form a suitable microclimate (usually in the Autumn).

The breed of the host can have a profound affect on scab establishment and progress. Fourie *et al.,* (1997) demonstrated that the progress of sheep scab lesions was almost five times greater in the South African Merino, compared to the local Dorper breed and suggested that Dorper sheep may play an important role in the spread of disease. In the

UK there are over 50 major breeds of sheep, over 13 minor breeds, eight rare breeds, eight recognised cross breeds and over ten introduced European breeds. In a recent study at Weybridge three distinct major breeds (Polled Dorset, Swaledale and North of England Mule) were assessed regarding their susceptibility to scab. The Polled Dorset is a hornless strain of the Dorset Horn, both lowland, short-wool breeds producing fine densely grown wool, with a mean staple length of 8.1cm and a total fleece weight of 2.25 to 3.0 kg. The Swaledale is a hardy breed of hill sheep, ideally suited to endure exposure on high lying moorland of Northern England. The breed is horned in both sexes, with a mean staple length of 10.2cm and a total fleece weight of 1.5 to 3.0 kg, mostly of course quality destined for carpet yarn. The North of England Mule is a result of a Bluefaced Leicester ram on a Swaledale (or Scottish Blackface) ewe. The North of England Mule is currently the most popular commercial breed of sheep throughout the UK, accounting for 14.5% of the British wool clip (used in the manufacture of both knitware and carpets). It is a hornless breed, with a mean staple length of 10.25 cm and a total fleece weight of 2.5 to 3.5 kg.

The Polled Dorset's presented thick, crusty lesions with well defined peripheries, with a mean percent body cover of 46.08% after 60 days. The Mules presented similar, if not faster growing lesions (67.12% body cover) of the same period of time. One characteristic of infestation on the Mules was that the fleece was matted tightly over the lesion making it extremely difficult to part. Although a number of Swaledales did present clinical disease over the 60 days of the study, most were very effective in grooming the challenge area, either by biting or chewing the fleece directly over the challenge site or scratching it with their horns. The results being extensive areas of close cropped wool over the challenge site, restricting lesion growth (25.02% body cover over 60 days) or totally preventing lesion establishment. The mean rate of lesion growth was significantly slower on Swaledales (37.08 cm^2 per day) compared to the Polled Dorsets and the Mules (57.5 and 84.06 cm^2 per day respectively). Breed differences were also observed regarding the numbers of adult female *P.ovis* with time. The finer wooled breeds (Polled Dorset and Mule) presented comparatively high mean mite populations 60 days after challenge (264.4 and 182.3 mites per sheep, respectively). The courser wooled Swaledales, only presented mean populations of 24.7 mites per sheep over the same time period.

Natural Resistance to Infestation.

Studies at the VLA (Weybridge) have demonstrated a marked gradation in the severity of disease with time in sheep challenged with the same number of adult female mites, ranging from no lesion formation to extensive scab cover in a matter of weeks. It was also shown that animals that elicit high antibody titres demonstrate the more extensive lesions (Bates, *in press*). Data obtained from the breed susceptibility study supported these findings, with individual Swaledale ewes maintaining sub-clinical infestations (ie. below 2.5% body cover) for well over 60 days, compared to others presenting 83% lesion cover, over the same time period. The length of the sub-clinical phase will thus depend on the immune-competence of the individual sheep. If an individual is unable to mount an allergic response to the mite, the lesion will not be permanent, and disease will not progress past the sub-clinical phase. If on the other hand, an individual is immunologically responsive, the infestation will establish and the lesion will enter the rapid growth phase. Rafferty and Gray (1987) showed that the degree of susceptibility of individual rabbits to *P.cuniculi* infestations differed due to the host's natural resistance to mites. Studies in the USA have shown that the pathogenesis of psoroptic mange of cattle is related to the hypersensitivity reaction of the host to the mite antigen: the density of mites within a given area of lesion is dependent on the susceptibility of an individual animal and that the size of the lesion was not indicative of mite density. The latter has also been observed on sheep at the VLA (Weybridge), with some sheep presenting small lesions with large numbers of mites and others large, expanding lesions with only one or two mites detectable (Bates, *in press*).

Concomitant Infections/Infestations

The sheep chewing louse (*Bovicola ovis*) is a common parasite of sheep on common grazing uplands of the UK. Observations at Weybridge have shown that sheep with pre-disposing infestation of chewing lice will not accept challenges of sheep scab mites, whereas sheep with active scab can be colonised by lice following natural exposure. The exact nature of this inter-species exclusion is unknown, but the skin changes initiated by lice feeding/excretion may render it unfavourable for mite colonisation. Lice, on the other hand, may actively feed on the scab lesion (Bates, 1999). Similarly any disease or treatment that suppresses the immune system may also affect the progress of infestations.

Sheep Previously Exposed to *P.ovis*.

The phases of scab appear to be significantly altered during re-infestation. Bates (2000) demonstrated that acquired resistance after a year was manifested by the lesion and mite burden remaining sub-clinical for over 50 days and thus supporting the observations of Spence (1949). Colonies eventually established and clinical sheep scab was observed, although mite populations remained extremely low. Similar observations have been reported in *Psoroptes* spp. infestations of other hosts (Stromberg *et al*., 1986; Guillot and Stromberg, 1987; Stromberg and Guillot, 1989; Urlir, 1991). This apparent acquired resistance may not be entirely immunologically modulated, indeed if at all. Changes in the host skin character and the increased age of sheep may have also have influenced the pathology of re-infestation. It has been reported that sheep scab can have a significant effect on the quality of processed leather (Pearson, 1996), suggesting significant changes in the character of the sheep's skin following *Psoroptes* infestation. These skin changes may also make it difficult for mites to feed following re-infestation.

Challenge Dose and Challenge Site

P.ovis can be contracted indirectly via live mites in tags of wool or scab attached to brambles, fencing, farm machinery, animal housing etc, but is usually contracted directly through forced sheep to sheep contact at market, in livestock lorries, at feeding troughs or any time that sheep are gathered and kept tightly packed together. An infestation can be initiated by only one egg laying female or hundreds of mites, depending on the mite burdens on other infested sheep or on the numbers of mites in the environment, together with the relative period of contact. For a low challenging dose, the growth of the lesion will vary accordingly: ie. on sheep of low scab susceptibility the disease may be several months in the sub-clinical phase.

All the data presented in this paper describes the results of one artificial challenge on the withers of experimental sheep. In the field a sheep may have several challenges with several discrete lesions or in the later stages many lesions coalescing. Certain areas of the sheep are unfavourable for initial colonisation by *P.ovis*, although they will be colonised as the lesion spreads towards them, as mite populations adapt to the environmental changes presented by the host (Bates, 1999c). Examination of naturally

infested sheep in the field has shown that over 51% of lesions occur on the withers and mid back (Bates, *in preparation*). Artificial challenges on the withers, flanks and brisket have resulted in rapid establishment of mite colonies and extensive lesion growth. However, failure to establish or slower growth was recorded on the face, head, tail-head and belly (Bates, *in preparation*). Although it is traditional throughout the UK to dock the tails of sheep, in order to prevent fly strike, tails are often left intact in flocks grazing the moors and mountains of Wales. Infestations exclusively confined to these intact tails are not uncommon, with lesions terminating at the tailhead and the remainder of the sheep entirely free of scab lesions. In one Welsh flock investigated 41 out of 139 (29.5 %) of ram lambs presented scab lesions (with active mites) covering the intact tail, with little or no clinical signs. Temperature may play an important role in the selection of sites suitable for colonisation, the mean temperatures of the cryptic sites, body and tail being 34.2°C, 28.7°C and 20.5°C respectively (Bates, *in preparation*).

Mite Virulence.

Roberts and Meleney (1971) observed that distinct populations of *P.ovis* existed in the USA. These differences were based upon a) the success of certain isolates to withstand population reduction in the summer, b) these more aggressive isolates survived contact with organophosphate acaricides compared to less aggressive isolates, c) aggressive isolates survived longer on sheep in isolation and d) aggressive isolates of sheep origin were able to spread through herds of cattle more rapidly and present more obvious clinical responses than less pathogenic isolates.

Investigations have been carried out comparing 15 field isolates of *P.ovis* with the now 26 year old VLA (Weybridge) Reference Isolate, regarding their speed of lesion production, rate of mite population increase, irritation, potential for otoacariasis, efficacy of ivermectin and acaricide resistance (Bates, 1999b). All the geographical isolates of *P.ovis* produced a progressive lesion, characteristic of sheep scab, but the extent of the lesion produced with time varied considerably between the isolates. Twenty eight days post challenge "low virulence" isolates produced relatively small lesions (mean 0.6% body cover) compared to "high virulence" isolates, producing more extensive lesions (19.4% body cover) over the same time period. Considerable differences in mite burdens were also observed. A "low virulence" isolate presented a mean *P. ovis* burden of 12.8 mites per lesion (range 0.0 to 30.0 mites per lesion) compared to 139.3 mites per lesion (range 65.0 to 220.0 mites per lesion) for a "highly virulent" isolate. In comparison to the rate of lesion increase, a considerable variation in the rate of population increase between low, medium and high virulence isolates of *P. ovis* was also observed. Mite burdens produced by low virulence populations of *P. ovis* can theoretically remain sub-clinical for considerably long periods (well over 100 days) following infestation. In medium and high virulence populations this sub-clinical period can be as short as 21 or 14 days.

Single (200 μg kg^{-1} subcutaneous injections) of ivermectin fail to eradicate artificial infestations of a medium virulent population of *P.ovis* (the VLA, Reference isolate), reducing mite numbers by 52% within 24 hours, 90% within 10 days and 96% within 20 days but live mites were still detectable 86 days after treatment. The numbers of

surviving mites correlated directly (r = 0.96) to the mite burden at the time of treatment (Bates and Groves, 1991). Moulting (pharate) mites cannot feed, consequently may only ingest sub-lethal concentrations of acaricide once they are active. Potential for this evasive strategy is therefore increased the greater the mite population at the time of treatment (ie.in high virulence populations). Differences in the efficacy of single injections of ivermectin with respect to mite virulence were thus observed. Low virulence populations (characterised by low mite numbers) can be almost eradicated after a single injection, yet significant numbers of mites survive within high virulence populations (characterised by high mite populations). Double injections however eradicated all populations of sheep scab mite (Bates, 1994). Oral drenching with ivermectin produced a 48% drop in mite numbers within 24 hours of treatment, but there was little further decline and no relationship between the initial mite burden and the extent of control was observed (Bates and Groves, 1991). The apparent inefficacy of oral ivermectin may have significant effects on the epidemiology of sheep scab by extending the sub-clinical phase or selecting for resistance to other endectocides administered by injection (eg. doramectin, ivermectin or moxidectin). Similarly the use of synthetic pyrethroid (SP) pour-ons (not effective for scab control) may suppress scab infestations and select for resistance to SP plunge dip formulations (Bates, 1998).

Cryptic Sites

Two species of *Psoroptes* spp. have been recorded to infest sheep: *P. cuniculi* infesting the ears and *P. ovis* infesting the body (Sweatman, 1958). In Great Britain *P. cuniculi* has been recorded within tubes of scab situated within the last centimetre of the external auditory canal (EAC), next to the tympanic membrane, from sheep with no recent history of scab (Bates, 1996a, 1996b). *P.cuniculi* ear mites are morphologically identical to the sheep scab mite (*P.ovis*), but do not initiate clinical scab on transfer to the backs of scab naive sheep. Symptoms of psoroptic otoacariasis differed between lambs and adults. In adults the symptoms ranged from the asymptomatic to aural haematomae, violent head shaking and ear rubbing, leading to excoriation and wounding of the ear and ear base. In lambs symptoms included plaques of scab (often bloody) on the external ear cleft, excoriation of the ear base, ear scratching with the hind feet and inflammation of the external aspects of the horizontal canal. In all cases the internal pinnae were clear of the

typical psoroptic scabs characteristic mites in the cryptic phase. As for classical sheep scab, hypersensitivity is suspected to play a significant role, with infested animals exhibiting levels of pruritus ranging from negligable to extreme discomfort.

In a survey of over 200 sheep presenting active sheep scab, live *P.ovis* were observed in the external auditory canals (EACs) of 38.6% of infested sheep presenting lesion areas ranging between 20.9% to 100.0 % body cover (Bates, 1999c). Although the incidence of *P.ovis* otoacarisis increased the nearer the leading lesion edge approached the ears, 20.0% of sheep were infested in the EAC when the leading edge was as far away as the mid-back. In studies investigating the temporal progression of sheep scab it ws demonstrated that *P.ovis* migrated to the EAC as early as 28 days following artificial challenge, with the leading edge 28.0 cm from the base of the ears (Bates, 1999c). Unlike *P.cuniculi*, *P.ovis* isolated from the EAC can be infestive to the bodies of sheep. Acquired resistance to scab, as described earlier, may have a direct effect on the growth of sheep scab lesions originating from aural *P.ovis*, (or residual body mites in the regressed or cryptic phase of infestation) are to re-infest their previously infested host. Colonisation would be more successful on scab naïve hosts.

P.ovis and *P.cuniculi* are therefore synoxenous, occuring sympatrically on the same host and synotopic (sharing the same habitat, ie. the ear canal) and may therefore be expressions of different phenotypes within a single type species (Bates, 1999b, 1999c)).

Conclusions

The results of these studies indicate that sheep scab can remain sub-clinical for long periods and as sub-clinical disease is generally asymptomatic, can easily be unknowingly introduced to un-infested flocks.

The prevalence of sub-clinical scab may be high in the common grazing upland areas of the UK, where the disease has always been a recalcitrant problem. There is tentative evidence that populations of *P.ovis* originating from these upland grazings are generally of low virulence (Bates, 1999c) and that virulence may be influenced by environmental constraints. Infestations may be clinically suppressed by the repeated ineffective use of

licenced treatments (eg. plunge dipping or systemic endectocides), exacerbated by incomplete gathering and the consequent incomplete treatment of the whole flock. This may be coupled with the use of chemotherapeutic agents for the control of other ectoparasites endemic to hill grazings, that are only marginally effective against *P.ovis* (eg.SP pour-ons for the control of ticks (*Ixodes ricinus*) and chewing lice (*Bovicola ovis*)). Hill breeds of sheep are phenotypically less susceptible to disease, presenting small lesions with relatively low mite counts together with a well developed grooming behaviour, thus making the confirmation of disease difficult. Added to this the relatively low populations of mites on the majority of sheep and the extensive nature of hill farming renders the spread of disease considerably slower when compared to that encountered in intensively managed lowland flocks of close wooled sheep. The fact that upland areas are known foci of infestation also indicates the presence of significant numbers of animals that have previously been exposed to infestation and thus developed an acquired resistance manifested by sub-clinical infestations.

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