

WOMBAT PROTECTION SOCIETY OF AUSTRALIA INTERIM REPORT ON ERADICATING MANGE IN WILD WOMBATS FEBRUARY 2007. Ed. A.Cox

WHERE TO BEGIN

Imagine you are standing in a round room with many doors. You are in the mange library and each door opens into a room filled with information. The first door might open into the amazing world of parasites and you could spend hours in there learning about *Sarcoptes scabiei*, the mite that causes mange. The next door leads to a room filled with photographs and stories about mangey wombats, and you could spend hours learning about how this mite effects their skin, their body and their behaviour. The next door leads to a room filled with books and journal articles about soil science and you could spend hours investigating how Australian soils are depleted of vital minerals through human activity. The next door leads to a room that is filled with material on animal health and nutrition and you may spend hours reading about physiological impacts of dietary deficiencies on animals. Yet another door leads to a room showing aerial photographs of Australia showing the ongoing clearing of land and the gradual death of once mighty rivers as they crawl to a halt, filled with sediment and dams. There are many other doors as well, doors labeled Veterinary Interventions for Mange, doors labeled Chemical Treatments for Mange and doors labeled Natural Cures for Mange amongst them.

Which of these doors pulls to you will depend on many things. It will depend on when you were born and the circumstances into which you were. Had you been a farmer in the early part of last century you would be bemused that anyone would want you to walk through any of these doors for the sake of a wombat. And in fact, many farmers of today would be bemused. It will depend on your education and where and when you were educated and in what field. Had you be trained some decades back, you wouldn't have been taught anything about wombats or native animal populations. If you were trained more recently you may have been taught animal management paradigms(ways of thinking) dictated by economic rationalism.(the dominant paradigm or ideology in Australia today). In this paradigm, value is placed only on the rare; where there seems to be enough of a particular animal, its value and its worthiness is considered lesser and this impacts on whether any assistance is provided to problems it may face. In this way of thinking, the Northern Hairy Nose Wombat with only 130 members left is worthy of intervention, the more common but fewer Southern Hairy Nose Wombat is less valued and Common wombat whose "commonality" is announced in her very name is worthless. How we think, the dominant paradigms that restrict or direct that thinking, determine where we go and what we do. This report attempts to give voice to a diverse group of paradigms from a wide array of people. In doing this we try to understand the thoughts of those who believe that all sentient beings are intrinsically worthy and deserve not to suffer through to those who hold this sentiment only for other specific species, upon which they put value or otherwise, to those who hold that belief only for members of their own species.

THE LANGUAGE USED TO DISCUSS THE PROBLEM

In this report we have chosen to use the equivalent terms used for humans to describe what is happening to wombats. We do this because we believe that an impediment to people understanding the issues is often the confusion in the language used. We note that when people write about mange in humans they use the word scabies, this makes other people think there is some difference between mange and scabies. That is wrong. Mange and scabies are different names for an infestation by the same creature.

We note that both scabies and mange are often called “infections”. That is wrong. Scabies-mange is a parasitic infestation. The mite that causes it is a parasite. An infestation can lead to infection, but an infection is caused by bacteria entering open wounds. Where we quote someone who makes this error you will see (sic) written after the word infection.

An additional problem occurs when authors use the word “infected” to refer to *their* behavior in placing mites on wombats. That is wrong. They have infested wombats with *S.scabiei* mites.

We have chosen to use the word “INFESTED” to remind readers we are not discussing a disease, we are referring to a parasitic infestation. Parasites feed off another animal, and that animal is called the host. In the context of this report, the wombat is the host to the mite. The adult mite has eight legs, so is classed in with spiders rather than insects that have only six legs. Spiders are arachnids. This is where the word acaracide, a product which kills (cide) arachnids comes from.

We also use the terms eradicate or fix rather than cure to again remind readers this is a parasitic infestation not a disease.

Where we believe terms can be misinterpreted or prejudice an appropriate response we use either both the scientific and common understanding term together or the later alone. So we will use the word epidemic rather than epizootic to refer to unusual increases in infestations and endemic rather than enzootic to refer to usual infestations. An epidemic or an epizootic are abnormal infestations while endemic or enzootic refers to something that is normal to the population. There is a mite found on healthy wombats (*Acaroptes*) that causes no problem to them, these mites are enzootic or endemic mites while the scabies (*S.scabiei*) mite is abnormal and causes problems and is epidemic or epizootic.

We use the term naturally raised wombat to refer to a wombat that was reared in the wild by another wombat. We use the term hand reared to refer to wombats raised by humans. We use the term free wombat to describe one that lives naturally, uncaged and unfenced and captive wombat to refer to one being held in an unnatural, human controlled environment such as a zoo, laboratory or other confined environment. There can then be four descriptive terms, free hand reared, captive hand reared, free naturally reared and captive naturally reared. These terms become important when we look at studies done on wombats. While there isn't a great deal around, much of what has been done can be

confusing and incorrect conclusions may be made because these four classes aren't differentiated. To give one simple example of the importance of this, if studies are based on free or captive handreared animals, given immunity may be passed to the young wombat via its mother's milk these animals may have different immunological responses to naturally reared animals. Similarly if studies are done on naturally reared captive wombats issues such as stress or lack of access to naturally occurring acaracides may have an impact on the results of such studies.

To make the information more readable, we have offered "extensions" to certain sections or discussions in this paper. These extensions are to be found at the end of the main body of the work and provide either a more in depth discussion of something mentioned, or a better explanation of a process referred to. For example, when immune system effects are discussed an extension section gives further details about immune responses.

WHAT THIS PROJECT AIMS TO ACHIEVE

The Wombat Protection Society of Australia has set as a five year goal the eradication of mange in the free wombat population. We state and make clear that we recognize this aim is underpinned by a certain way of thinking and therefore dictates a certain way of responding. We make no apologies for this and our society Bulletins have outlined our philosophy. (Bulletins 1-3,2006;4-5,2007) .

Our goal has met with some interesting reactions, and we recognize that each person's dominant paradigm dictates that reaction. It also dictates which of the mange library rooms people spend more time in because they are more comfortable and familiar with the information in that particular room. So we have people who say that a free naturally raised wombat 10 kilos or more with scabies/mange infestations should be euthanised. This is in fact what many animal welfare groups do with wombats with *S.scabiei* infestations. We have people who say that scabies-mange occurs in sporadic outbursts and these have happened throughout history and the wombat population lives on, so there is no problem. We have people who say that wombats aren't a threatened species so there is no need to intervene. Others who say that any treatment would be worse than the infestation... and strangely enough, whether commercial chemical or other eradication methods are discussed, the proponents of the "other side" make the same comment. Still others believe something should be done, but know not what to do and others want to spend more time in the research rooms saying until we know what to do we should just read more. As well as all these views, the "sporadic outburst phenomena" theory favoured by government departments , and its underlying hypothesis that it is alright to do nothing about serious health issues in native animal populations unless the animal is almost extinct, exists and fits into the current paradigm of economic rationalism. Some of these views favour apathy and inaction. In answer to this, and irrespective of which room you feel most comfortable in, we invite everyone to start in the library foyer and learn about what it is like to be a wombat infested with *S.scabiei*.

INFESTED WOMBATS



A wombat showing clinical signs of scabies/mange

Scabies/Mange progresses from redness of the skin due to congested capillaries (erythema) caused by female mites burrowing into the skin and leads to disturbances in the process of keratinisation . Keratin is the basis of hair and nails and horny tissues and keratinisation leads to an abnormal thickening of skin (parakeratosis). Next comes hair loss (alopecia), followed by open scratches on the skin (excoriation) and then the bald, thickened skin cracks open (fissuring of parakerotic crust).Starvation and secondary bacterial infections lead to death.

Scabies/mange is caused by a mite , *S.scabiei* that is related to spiders not insects. In general this mite cannot be distinguished on its host species by its physical features (morphologically) .It is believed that the mite originated on primates and has adapted to prefer certain host species. Lots of animals and humans all get infestations from this mite.

Lee Skerratt, author of the only systemic study on mange in wombats states that “sarcoptic mange in wombats is similar to that found in domestic animals and humans” (2001,p.31.) Irrespective of the intensity of the initial infestation, though not inevitable, (Ibid) clinical signs develop and progress the same way. The wombat begins to feel an irritable urge to scratch (pruritis) then his or her skin reddens due to congested capillaries (erythema) and the skin begins to thicken (parakeratosis) and hair falls out, (alopecia), leading to abrasions from scratching on the skin surface (excoriation) .The thickened, bald skin gets cracks and craters, much the same as what happens if a human has a thick scab on a wound and then stretches the scab before the underlying skin has healed. These cracks are called fissures. Once the fissuring of parakerotic crust occurs, opportunistic bacterial infections can now enter the wombat through these open wounds and they frequently become flyblown (myiasis). Often the skin crusts invade the skin

around the eyes and blind the wombat, however the wombat would not be blind if the infestation was eradicated. When the thickened crust is present the infestation and its impact on the skin is called hyperkeratotic or crusted or parakeratotic mange or scabies or sarcoptic mange and in humans is sometimes called crusted or Norwegian Scabies. The hyper and para keratotic definitions refer to changes in the process of keratinisation, the means by which keratin, the important component of hair and nails and horny tissue is made. Hyper / para refers to this process being disrupted and the disruption leads to the thickening and the abnormal skin condition. "The thick crust may crack resulting in haemorrhage, pyoderma and sometimes cutaneous myiasis"(Skerratt,2001,p.31). Haemorrhage means bleeding, pyoderma purulent/pus affected skin, and cutaneous myiasis means flyblown skin. The wombat is now "emaciated and lacking hair, with a thick dry crust, composed of keratin, many mites and their debris, bacteria and neutrophilic debris adherent to skin"(Ibid). A neutrophil is a granular leucocyte or blood cell.

The processes are caused by the female mite burrowing deeply into the underlying skin tissues. The reaction to her faecal waste and other mite debris, causes the response that leads to the scratching which in turn leads to the other clinical signs. The female mite burrows into the skin to make places for her eggs. The female mite lays eggs at the rate of approximately three per day. She can tunnel quite extensively, up to 0.5mm each day . The fissured crusts are very resistant to moving, unlike normal scabs, which cover underlying healing tissues and lift as the skin heals, these crusts become thicker as the infestation develops.

The mite needs a host upon which to live and dies without a host. Optimal conditions for mite survival off a host is a temperature less than 10 degrees celsius and humidity of 98%. (Skerratt,2001,p.38). It is these conditions that have led people to concluding that mites may be translocated (transferred) in burrows. In such conditions only, can the mite live off a host for up to three weeks. If it doesn't find a host in that time, or if those conditions aren't maintained, it dies. In addition "the mite cannot actively seek a host when the temperature is below 20 (degrees)C" and "once off the host it is likely to starve and desiccate" (break up) (Ibid.).

The wombat's behaviour begins to change when the clinical signs progress to crusted scabies. He or she becomes unable to roam far enough to get sufficient food or water and gradually dies from a combination of starvation and infection, with lung (pleural) changes most frequently noted. The wombat is often found out in the open at this stage and abnormally found out and about in daytime. At this stage he or she is so sick they are relatively easy to catch. Left alone they will die. It is not a pleasant death and it is not a quick death. Wombats with parakeratotic skin fissures may get flyblown in the deep fissures which means live maggots, the fly's larvae, wriggle in and eat skin tissue causing extreme discomfort. Infection may also enter the wombat's body through these fissures so other bacterial infections affect the animal. The skin around the eyes is often affected and when it crusts sufficiently the animal is blinded. Triggs, writes "in severe cases the eyes may be virtually closed by the lesions"(2002,p.116). Untreated, the wombat, blinded by and exhausted from the parasitic load, either dies from a secondary infection

or starves or dies from thirst as they become unable to wander and chew (masticate) properly. The mange “may encompass the entire body of the wombat with head, neck, shoulder and limbs most commonly affected” (Skerratt,2001.p.31).

A number of references have been made to the behaviour of severely infested wombats staying around one burrow/area. An unlucky wombat will live this way for many months. The same unlucky wombat may inadvertently share its mites with another wombat that comes into near contact or uses a burrow the infested wombat used, though this is still an hypothesis (theory) for mite translocation and has yet to be proven.

We believe it is more likely, that when the wombat dies and its burrow is shared, and temperatures are above 20 degrees celsius, only then are all the conditions present to allow the mites to move off their host and seek a new one.

Skerratt’s (2001) field studies failed to prove that wombat-wombat or burrow sharing is the mode of transmission, yet most people believe this is the most likely means of mite translocation. He infested free wombats at Padilpa in Victoria with high mite densities (p.171), confirmed they developed mange (p.181) and shared burrows (p.177), however “in the 100days following infection(sic) of 5 wombats, 34 wombats that had not been infected(sic) but shared burrows with infected(sic) wombats were trapped. They did not show clinical signs of sarcoptic mange nor was s.scabiei found in skin scrapings”(p.182) .

This suggests that some other ameliorating factor may occur in the free population in the right conditions. Skerratt’s experimentally infested captive wombats developed clinical signs of mange faster than those free wombats experimentally infested at Padilpa. It is also interesting to note that one of his captive, experimentally infested wombats did not develop mange when 1000 mites were placed on it, despite housing this wombat with another infested with the same amount. Later, the wombat did develop mange when infested with 5,000 mites. This suggests that there is variability within the wombat population in individual responses to certain mite loads.

Some people believe that the fox is a vector (transmitter) of the mite and this also has yet to be proven, and genetic evidence tends to suggest that this may not be true. In 2006 , Sue Jaquinot the Deputy Secretary for Victorian Resources and Regional Services replied to a letter asking what the Victorian government was doing to combat mange writing;“The red fox may be a major vector for mange in wombats, through the sharing of burrows” and went on to explain existing fox control programs as a contribution to limiting the spread of mange in wombats in Victoria. Triggs writes “Whether the sarcoptes mite came to this country with the dingo about 4000 years ago, or with domestic dogs and foxes in the last 200 years is not known- probably both as it infects wolves (the dingo is an Asiatic wolf) as well as dogs and foxes”(2002,p.116). Were foxes or dingos the only translocators of mites, mange should not have been seen in Tasmania or on Flinders Island, and in both areas wombats have been reported with mange. These observations have been made previously and are not explained by the recent illegal release of foxes in Tasmania. (Fox DNA project University of W.A. 2007). The theories of wombat to wombat and fox translocation of the mites through burrow sharing will be

elaborated upon in the extension section of this paper. The theory of dogs translocating the mites, is now understood through D.N.A. analysis to be unlikely because “mites from humans and dogs have been shown to be genetically distinct using allele frequencies” (Skerratt,2001,p.36). This will also be discussed in the Where did it come from? extension.

As wombats have a large home range, moving usually 1-2 kilometres each night and up to 4 kilometres (Triggs,2002,116) it is very likely they will encounter another wombat with mange and in many areas there are no places where wombats are mange free. Wombats will also travel beyond their home range, perhaps hunted out of it through territorial disputes. Member number 694321 retrieved a hand reared free wombat some twenty kilometers from her release point. If wombat to wombat is the mode of translocation of mites, then, clearly all wombats are at risk. According to the Minister for Environment and Conservation of South Australia, Gail Gago, 75% of Southern Hairy nose wombats caught or observed by a research group from the Adelaide Zoo in January 2005 were showing signs of mange. The South Australian Department of Environment and Heritage confirmed what they term “an outbreak” of sarcoptic mange in a population of Southern Hairy Nose Wombats in the Murraylands in November 2004. According to Gail “The disease had not previously been documented in the species.”, however, this is not true. According to Wells, writing in 1971, and quoted in Skerratt (2001,p.32) “there have been occasional outbreaks in the Southern Hairy Nose Wombat but not the Northern Hairy Nose Wombat”. There is no state in Australia that does not have mange within its wombat populations, however so far there does not appear to be mange present in the remaining 130 *Lasiorninus Krefftii* (Northern Hairy nose Wombats) located in Epping State Forest in Queensland. These animals are highly vulnerable as they represent the last of their species. This leads to the conclusion that mange is now widespread within the free wombat population.

Mange is totally fixable. We use “fixable” because it is not a disease. It is a parasitic infestation. Humans get scabies/mange and very quickly get rid of scabies/mange. Humans would not accept “sporadic outbursts” that “don’t impact on overall population numbers.” Watch what happens when one child in a school gets head lice. Action is taken very quickly because humans are well aware of the exponential breeding capacity of lice and mites. Exactly the same thing happens with a scabies outbreak in a hospital or nursing home. Where necessary, barrier nursing of crusted scabies occurs, but generally it is simple over the counter preparations that when properly applied “cure” this supposedly “incurable disease”.

From a wombat captured in the early days of settlement “people in contact with this wombat developed signs of scabies”. “They were treated with sulphur and the signs resolved.” **“Organic or metabolized products of sulphur appear to have acaricidal activity and for many years sulphur was the drug of choice when treating scabies”** (Skerratt,2001,p.32) . In a modern day medical encyclopedia, with reference to scabies, “scabies treatment consists of topical permethrin cream (Elimite), lindanne or crotamiton (Eurax). **For infants, 5 to 6 per cent precipitated sulphur in petrolatum (petroleum**

jelly), applied twice daily for a week, is usually adequate.” (Miller-Keane,1997,p.1444)

Mange is not an incurable disease as some would suggest. Nor are its effects irreversible, though some believe that after a mange infestation, overall immunity can be affected, (though it seems equal numbers believe that immunity increases as believe immunity decreases.) Skerratt’s work shows that once the parakerotic crusts are removed and the mite is removed, the wombat stops scratching incessantly and its skin heals. It makes a full recover and is not more susceptible to recurrences of the mite.

The Society wrote to all State Premiers asking what their state was doing to combat mange. The outcome of these letters is pitiful. One researcher, David Taggart, (not a “task force” as the S.A. Government responded) (Letter,S.A.Gov.2006) is monitoring the affected Southern Hairy nose Wombats. That is it. Tasmania thinks it has no problem, telling us “whatever the fate of the Common wombat on mainland Australia, this species remains secure Tasmania.” (Letter,Tas.Gov.2006). Victoria hopes their fox reduction program will help and N.S.W. admits doing nothing.

If mange is totally fixable and if it causes such suffering, why isn’t anything being done to get rid of it?. There are many impediments to taking decisive action to eradicate scabies/mange in free wombats and understanding some of these may help lead to a proactive management plan.

IMPEDIMENTS TO ERADICATING MANGE

Attitudes to Mange

Apparently a joke amongst farmers who illegally shoot wombats is that you never shoot a mangey one, because it is going to kill off all the rest. Australian Law allows a native animals to suffer mange where under the same law any individual who kept a pet in such a condition would be charged with animal cruelty. Presumably this anomaly arises because native animals are the property of the Crown and the Crown and its representatives do not have to meet the same standards of duty of care as the rest of the population.

The “Sporadic Outburst” Impediment

There are a number of people who believe that mange-scabies occurs in sporadic outbreaks, that wombat population numbers don’t get effected and that these outbreaks have no more than a local effect. This is wrong. The Victorian Department of Sustainability and Environment states “periodic episodes of severe mange in wombats have been reported previously. Wombat populations do recover from these outbreaks and it seems unlikely that mange is a serious threat to their long term survival” (Letter,Dec.2006). David Llyewellyn, Minister for Primary Industries and Water in Tasmania writes “while I understand that mange is present in wombat populations in Tasmania, wombats remain widespread and locally abundant here” and this suggests “that mange is having no more than a very local impact on wombat populations”

(December 2006.Letter.Tas.Gov.) Lee Skerratt wrote in his doctoral thesis written in 2001 that the common wombat upon whom his study is based, is affected by sarcoptic mange throughout its range and claimed “prevalence of the disease is generally low, with less than 5% of wombats in a population affected. However, epizootics (humans equivalent epidemics) occur sporadically”(2001,p.3). This figure is not consistent with Skerratt’s own survey results covered in chapter two of his thesis 2001.

In Skerratt’s 2001 survey, taken between November 1995 and January 1996 (p.50) “Mange was reported to be widespread and to occur through most of the range of the common wombat. Alopecia and thick crusts on the skin, which are clinical signs of mange were reported from 93.3% of localities” (p.51) and identified positively from skin scrapings from common wombats at 51.7% of localities in South Australia, Victoria, New South Wales, Australian Capital Territory, Tasmania and Flinders Island. (Ibid.p51). Of 48 common wombats in this survey, all from Victoria, 7, or 14% had clinical signs of mange. An estimate from N.S.W. of 15% is not substantiated by any figures. Of 8 Southern Hairy Nose wombats observed, 14% had clinical signs of mange.(see Table 2.2 p.58 and table 2.3 p.59 in Skerratt,2001).

We are not sure where the 5% figure came from and this is not what is being experienced on the ground in wildlife organizations’ reports about mange. We believe that this may be a figure estimating mange at Healesville, Victoria in 1992. There, an estimate of wombats affected by mange was made of 5%, however why 5% is quoted when a similar estimation made at Moe at the same time of 14% is unknown. Furthermore it would be an extreme extrapolation of these very limited figures, both from Victoria, to apply it to common wombats throughout their range as is done on page three of Skerratt’s work.

It also, unfortunately is not what is being seen in the epidemic in the Murraylands Southern Hairy Nose Wombat where from the first report of sarcoptic mange in 2001 (Skerratt,p.35) in that group to January 2005 where “75% of animals in a recent survey had clinical signs of mange”. (Letter, SA Gov.2007). That is an exponential increase. To have so many animals effected simultaneously, particularly given the more social and burrow sharing behaviour of the southern Hairy Nose has the potential to wipe this species out as well. Both complete extinctions and local extinctions are recorded. Many isolated populations have disappeared completely, the King Island population which was here at time of settlement is now extinct (Skerratt,2001,p.33) and in Mumbulla State Forest in N.S.W. wombats, once common in 1988 (Lunney and O’Connell ,1988.p.695) are thought not to be there by people living in the forest. (communication, member 694321) . A further complete population extinction occurred in Robe, South Australia where wombats have not been seen for 20 years and the last one seen had mange. (Skerratt,2001,p.52).

If it were true, as some would have it that mange occurs in isolated “sporadic outbursts” in the naturally reared free population and population numbers do not get affected, then we should be taking action to ensure that when these “sporadic outbursts” occur, animals are treated, from a humane perspective if for no other reason. After all, if we are only

looking at “sporadic outbursts” of mange- what would be the difficulty in dealing with what should be small numbers of “sporadically infested” wombats?. Yet the “sporadic outburst” hypothesis is used to justify taking no action, when, if animal welfare was the point of evaluation, it would suggest a more immediate and expedient response. It would also suggest an easier and more targeted response would be possible.

The sporadic outburst theory also stands in stark contradiction to what we know. An epizootic/epidemic of scabies/mange occurred in N.S.W. in 1937 and was reported by Grey. This “outbreak” “resulted in a decline in wombat numbers from thousands to a few”. (Skerratt,2001,p.35). Despite Gail Gago’s assertion, following confirmation of scabies/mange in the Murraylands population of Southern Hairy Nose wombats, that “the disease(sic) had not been previously documented in the species”, “*S.scabiei* were first collected from Southern Hairy Nosed wombats in 1976 by Southcott and were first reported by Wells in 1971; and despite Victoria’s Department of Sustainability and Environments’ claim that “periodic episodes of severe mange in wombats have been reported previously” and “Wombat populations do recover from these outbreaks and it seems unlikely mange is a serious threat to their long term survival” (Letter,2006) and Tasmania’s assertion “that mange is having no more than a very local impact on wombat populations (Letter,2006) ; following a literature review and survey conducted on the prevalence of mange Skerratt (2001,p.35) concludes “despite reports of many recent declines in the local abundance of common wombats...little is known about sarcoptic mange in wombats” and that “the distribution of all three species of wombats has contracted markedly since European arrival” and that “small, isolated populations (that) are threatened with extinction by various factors including sarcoptic mange.” It is also true that many populations have disappeared completely and so few animals remain in some areas that people often claim there are no wombats / never were any wombats here. Member 694321(2007,communication) reports people in Mumbulla State Forest in N.S.W. claim no wombats ever lived there because in their life time they haven’t seen wombats there, yet we know from Lunney and O’Connell that “the wombat was locally common but rarely seen” in the late eighties.(1988,p.695) Because population surveys were not done to map wombat’s original range, the sporadic outburst theory gets credence, because there is no information to indicate what has happened to their numbers in general. Yet we do know that overall, their numbers have dwindled, the remaining populations have become more isolated and many local populations are now extinct. One species is on the very brink of extinction and another vulnerable. We know these things even in the absence of any historical mapping of their populations and common sense suggests if numbers are declining and population distribution contracting, there is a major problem occurring.

The “Over 10 Kilos Do No Treat” Impediment

Animal welfare groups often despair when it comes to treating wombats with mange and different groups have established different rules. The “over ten kilos” shoot it/ euthanise it rule is one such rule. There is no doubt, that often this decision has been reached with an enormous amount of care and concern and involves consideration of many factors. Rescuing and trying to treat a still healthy 30-40 kilo wombat with mange is by no means

an easy task- especially if you are looking at popularized post 1980s treatments that may require up to six injections and/or involve cage captures, even to use a back strap application. Alternative treatments may require that the wombat be kept warm and either quarantined from others with scabies/mange or if kept with others re-treated due to prevent reinfestation. The lack of facilities and concern about holding wombats in captivity all factor into this impediment. The idea of giving the littler ones a go has more to do with ease of handling and quarantining and the hope they can adjust better to temporary captivity than it has to do with any greater or lesser chance of survival were the same treatment given to the over 10 kilo animal. However, we need to take care that we don't get blindsided by this type of rule and forget why it was created and the limitations that led to it. These "rules" have a tendency to become divorced from their roots and then applied in changed or different circumstances. Because at some point in time, referring to some specific wombats, an agency or person using some specific treatment made a decision to euthanise all wombats with mange over 10 kilos" is NOT the same thing as saying if wombats are over 10 kilos you cannot eradicate their infestation. It has been shown that perfectly healthy , naturally reared , free wombats taken into captivity will get mange when infested with enough mites. It has been shown those same wombats can have those mites eradicated and make a complete recovery from the effects of mange"; see Skerratt, 2001 (Chapter 4 pp.88-99), where captive wombats were infested with scabies mites and developed severe mange, then had all mites eradicated from them and (Chapter 8 pp. 161-185), where naturally reared free wombats were infested with mites and released into a naturally reared free population experimentally. In the later, no other free wombat became infested and the experimentally infested wombats were later recaptured and successfully treated and all mites removed.

Despite Skerratt's work, proving otherwise, the "can't treat certain types of wombat" myth continues. Sue Jaquinot , responding to the query about what Victoria was doing to combat mange, writes; "While it may seem desirable to treat affected animals with long lasting acaricides to kill the mites causing the disease, in practice this is not feasible" (Letter,2006.) Age and size of a wombat is only an impediment to the ease with which people find currently promoted chemical treatments (long acting acaricides) able to be applied. You can understand that attitude arising when one long lasting acaricide, arguably the most used since Skerratt's 2001 work, Ivermectin, requires six injections to produce efficacy against recurrences and to remove the immediate infestation. However, "the can't treat impediment" falls into the trap of making evaluations about what is available at a current period of time. It has nothing to do with whether the animal is treatable, will make a full recovery and nor should it have any basis in the decision making processes regarding whether we should take action to eradicate mange in the free population or not. Mange, at all levels of severity, can be eradicated on any wombat, Skerratt has clearly demonstrated that. Whether commercial chemical acaricides are "feasible" as the means by which this is done is a different story all together. We forget humans and animals managed mange and got rid of it quite successfully long before the advent of commercial chemical products.

The “Can’t Treat Wild Wombats, they get Reinfected ” Impediment

This attitude is not intentionally cruel or stupid, in fact it is mostly opinioned by those governed by compassion for the wombat. To read how one wombat carer came to have this attitude and all the heartbreak that entailed see “Wombats That I won’t Treat” <http://www.wombat.echidna.id.au> It comes from people who have seen failures with treated wombats either getting reinfested or from people not knowing what to do with a big animal that has severe mange. It also seems that the risk of reinfestation is used as a rationale for not treating wombats without having been thought through sufficiently well. The Society wrote letters to all Australian State Premiers asking what they were doing in their State to combat mange in wombats. The “can’t do anything because of ‘reinfection’ (sic) argument was actually put in a reply to one of these letters and used as a justification for doing nothing. “Treatment of a wombat to destroy the mange mites does not provide immunity from future infestation by mite” (Letter, Vic. 2006) . This is the equivalent of saying let’s not treat any bacterial infections in people or animals because treatment doesn’t stop them catching the disease again. Of course treatment doesn’t guarantee anything regarding reinfestation... that’s the point. Wombats will get infested and wombats will get reinfested until mange is better controlled. If we read how reinfestations are dealt with and managed in humans the absolute opposite of the “do nothing” approach is recommended because of the concern with crusted scabies that “this advanced state of infection can act as a source for local epidemics” (Syd.Uni ,2007). It makes no sense to NOT treat carriers of an epidemic and the point of an epidemic is reinfestation. It ceases to be an epidemic when reinfestation stops.

It’s a Wild Animal and Won’t Cope Impediment

As mentioned in the opening, this attitude often stems from what people have seen done to wombats in the course of treating mange. Capture cages and other stressful devices have been used in experimental work with wombats and often people believe the only way is to capture a wild wombat to effectively treat him or her. Lee Skerratt undertook research using wombats where mites were intentionally put on these animals and they were regularly sedated for various reasons (p.165) and at the end of maximum infestation, the infestation was removed using six ivermectin injections and antibiotics to guard against any lasting secondary infections. This serves as the only systemic study of “inducing” and “curing” mange in wombats and Lee’s studies are widely reported. It appears to be based on Lee’s study that many people believe that these sort of practices, imposed on naturally reared free wombats, would not be practical or reasonable.

We have to take care that we don’t see what has been done experimentally as the benchmark for what can be done practically. An experimental study into parasitology using wombats such as Lee Skerratt’s is not the same thing as research to show how to treat parasitic infestations on wombats in the wild. While we can learn from Lee’s work, he was using only one method to remove the mite and it was not part of the scope of his study to look at any other alternatives. Veterinarians and animal welfare groups and government departments use Skerratt’s research without identifying its limitations. It was never intended to be, nor has Lee ever suggested that it was a study into acaracides. It was a study into mites and the use of one acaracide as proof that you can induce severe infestations of mange into naturally reared captive (Chapter 4 pp.89 –99) and free

(Chapter 8,pp. 161-185) wombats and that there is at least one acaricide, Ivermectin, that will eradicate the mange in both.(pp.97-98 and pp.172-173) Too many people who have not read Lee's work now claim six injections of Ivermectin is the only way to eradicate mange. That is a total nonsense, there are many acaricides available.

There are other issues involved in any treatment that may be recommended for naturally reared free wombats . Member no.694330 reports that South Australian Law requires an animal taken from the wild not be released back to the wild. So whether good, bad or indifferent, in one State at least, treatment for wild wombats would require mass declaration of sanctuaries to stay within the law were any form of removal from the wild to treat contemplated.

Most animal rescue groups simply don't have the person power to even contemplate the types of activity Lee Skerratt undertook.

There is real concern amongst these groups that any type of captive treatment is impossible both because no facilities exist as well as concerns that the wombat wouldn't adjust to either temporary confinement or later release. It is strange though how Lee Skerratt's work is well quoted to say how difficult it would be to treat wombats, but isn't quoted when generally held beliefs are laid asunder by it. Despite the argument that the removal of wombats from their environment into captivity would be cruel, Lee's work provides evidence that they settled in quite quickly to a captive situation and even major dietary changes, within a matter of days. (2001,p.89) Lee Skerratt's work suggests this was the case even though he added an additional stress by housing otherwise independent and solitary animals together. Member number 694324 regularly houses groups of wombats together to treat mange and only occasionally has to isolate one because of ongoing and dangerous aggression to the others. While we are not promoting any of these methods, what we are saying is much of what is claimed about what may happen if wombats are treated, has not been substantiated nor is it supported by what little has.

Animal welfare concerns are valid and should be used to determine and shape the type of policy for treatment. Not to discourage or validate a policy of non treatment. While it might be possible to trap every wild wombat six times and give it injections, the person power needed, the stress on the wombat, let alone the practicality of the exercise or concerns about long term implications from product use, would rule out such a plan. That may be the option of choice for some severely manged wombats needing antibiotics but certainly isn't implicated for those showing mild to moderate infestations. Also, it doesn't mean it is not possible to come up with a treatment that can be applied to wombats in the wild. Worries about capture, practicalities of locating wombats and laws enforcing their non release, should just inform the type of treatment we are looking for. It would seem that a treatment that can be used on wild wombats without the need to capture them would be ideal. We need to think outside the square.

The "Their Immunity is compromised" impediment.

Once again people opinioning this attitude have very good intentions. They confuse an infestation with a disease and see its recurrence as some type of proof that the wombat is

compromised and then conclude that there is no point in treating wombats. Some people believe that the wombat's immune system becomes impaired by the mite and that is why secondary infections can occur. They believe that wombats that have had mange once are likely to get it again. While true, these are not causal. From this they feel to treat the animal and put "it through" treatment is unfair as the animal will get more stress and may die from that alone or, if treated, because immunologically impaired, some other infection will kill it, or failing those two taking the animal out, it will get infested again. The Victorian Government when asked about their management of mange wrote "Treatment of a wombat to destroy the mange mites does not provide immunity from future infestation by mites" (Letter, 2006).

We can probably respond best to these concerns by paralleling what happens in human epidemics of mange. The "does immunity increase/decrease/ therefore do we treat/not treat" argument apparently, only applies to wombats. To humans it is irrelevant because ALL are treated, whether they are likely to get reinfested or not. In fact it is known that immunologically impaired people are MORE likely to get reinfested but fortunately, in people, that doesn't lead to a policy of NOT treating them because they are more likely to get scabies/mange back. Medically, a "don't treat the more severely infested" argument is ridiculous because it will result in an epidemic. The "severely infested" are the very ones you must treat to stop the epidemic. It is because wombats aren't being treated that they are getting constantly reinfested. It is the infestation that causes damage, it is the infestation that causes immunological problems. Once the infestation is gone and any secondary bacterial infections treated, the immune system is not compromised permanently. Skerratt (2001, pp 88-99) showed quite definitively that you can take healthy naturally reared free wombats, induce severe sarcoptic mange in them by infesting them with enough mites, see the immunological changes (p.80) and by treating the infestation and secondary bacterial infections (p.92) clinically resolve the whole process. (p.94) He also showed that when reintroduced, those animals previously infested, react faster, (p.94) so some longer lasting immune response is present, "clinical signs were seen earlier but lesions developed at a slower rate in a reinfested (sic) wombat that had previously developed mild parakeratotic sarcoptic mange" (p.94). As with humans who experience an allergic reaction wombats that are reinfested REACT by scratching (pruritis) faster the second time round. Rather than this being an indicator of a failing system, as with allergies, it is the additionally sensitized response, which may in fact help the animal shed more mites than when initially infested. The infestation causes all the problems and reactions. To apply the counter argument and say because their immune system is effected and they will get reinfested so there is no point in treating them leads to perpetuation of the mange epidemic in wombat populations. It also makes people believe there is a necessary connection between the two. There is not.

A treated wombat that does not come back into contact with the mite won't get mange. It is also possible that wombats previously exposed to mange may be more resistant to developing mange a second time because they have a faster reaction to the mite and may manage to scratch more off before they set up a high density. Lee Skerratt introduced five wombats that he had heavily infested with mange mites into the naturally reared free population of wombats at Padilpa in Victoria. He documented their sharing of burrows

with some 34 other Padilpa wombats over a period of 100 days. He checked the thirty four companions and found NO MITES and NO MANGE on these animals. Furthermore, he found that the five infested naturally reared free living wombats developed mange more slowly than his captive wombats after an equivalent period. He rated their mange as mild to moderate, where in the same period his captive wombats had developed (with one exception) what he termed severe mange.

This study is used by Lee and others to suggest that therefore only “severely manged wombats” pass on the mites . This seems to be where the euthanise/shoot/remove the severely manged wombat reaction derives and in fact is a conclusion Skerratt makes himself.(p.250); and where the ‘leave it alone it will kill off the others’ attitude (p.246) derives, as well as observations of this happening by those who see wombats as agricultural pests. The wombat with severe mange being the translocator of mites is however only an hypothesis and was not proven by Lee’s study. The finding that mild to moderately infested wombats as represented by their mild to moderate mange, DO NOT pass on the mange mite, should not lead to the conclusion that it must be the severely infested as represented by severe mange that DO. A number of other explanations are possible and were not canvassed by Lee in his analysis. One other explanation is that Padilpa wombats have immunity to low levels of mites (Lee infested the five wombats with 5000-8000 mites each) and although they shared burrows, their companions would not have been exposed to anywhere near this number of mites through sharing the burrows while these wombats were alive. In respect to this it is relevant to note what Lee himself noted that “landowners around Padilpa had seen mange in the population previously” (2001,p.163) It is just as likely, if not more so, that the Padilpa wombats, previously exposed to the mite had developed, if not immunity to them, at least a reaction which led to them shedding the mite rapidly. While this is possible, the fact that Lee inspected all of these animals and found NO MITES at all on any of the 34 burrow sharers really suggests that the mites were not leaving the infested animals. The study actually shows that wombat to wombat and burrow sharing are not means of transmission. To place the caveat on this of saying it must be only severe mange that fits those theories is to extrapolate this work into very shakey conclusions. Other explanations are just as likely, including some other vector is involved in the transmission of the mite. A further explanation of what Lee observed , but didn’t consider is that it is only when the wombat dies in a burrow it is sharing with another wombat that the mites leave the dead body and find a new host. Given the mite needs a living host, unlike maggots, for example, this is a much more likely explanation than mites living in burrows, wombat to wombat contact or any other vector being responsible for translocating mites from an mange infested wombat to an healthy one.

The study is used to recommend culling wombats with severe mange, despite the study also showing that severe mange is fixable. Removing the odd one wombat with mange from the population has not been proven to have an effect. If some other means of translocation of mites is occurring, phoresy,(flies) for example ,as has been suggested, we do not help wombats by culling ones with severe mange. What we should be doing is ensuring that ones with severe mange don’t crawl into burrows and die. If wombats get mange because they have no previous experience with it (naïve populations) from a dead

wombat and then don't get it as easily after they have, then a different course of action would be recommended. We would actively treat severely manged wombats to ensure they don't die, and treat those with moderate to milder infestations knowing that their experience with the mite now gives them some ability to resist becoming severely reinfested. By culling "manged wombats" we may be ending that wombat's suffering, but we should also consider that she well may be able to tolerate treatment and be mite free and recover, which would also end her suffering.

The "Its Global Warming" Can't Do Anything Impediment

There are many people who understand that Australian soils and our plants are in crisis. Not only do we live on a fragile ancient continent but we have used land clearing and agricultural practices such as the use of superphosphate which leads to decline in available minerals and nutrients in our already limited array of native grasses. Anyone who has watched a sick wombat selectively choose what to eat would understand that what should be a pharmacopia for wombats to self medicate has become limited and stripped of its medicines. Unwell wombats will walk kilometers to seek out one type of grass or bark or soil. They appear to know instinctively what they need to "cure" themselves. Wombats with severe diarrhoea for example, have been reported by member number 694301 to locate spider web and consume large quantities of this and to locate specific tree bark such as the rough barked angophora or brown stringy bark to balance their gut. While there is a cogent argument that animal disease is linked to environmental changes, that should not equal "doing nothing". Member number 694324 for example, works on growing a small plot of nutrient and mineral enriched grass in a "feedlot" in the hope that wild wombats accessing this improved feed will gain benefits from it. We know that certain elements like copper and sulphur have antiseptic and antibacterial properties and that some of these essential elements are now missing from common grasses. Were such nutritional deficiencies the cause of the wide spread of mange, again, they could be addressed by supplements and strips of nutrient enriched grasses being grown. It is probably more a problem of wombats being forced to reside in ever decreasing sized areas of land, away from their preferred forest gully habitats and being forced into contact with domestic animals. Reports seem to indicate that deep forest dwelling wombats are less likely to have mange than those living closer to human and domestic animals habitats.

Once again, even if these environmental factors are instrumental in the development of mange, this should not be used as an excuse to do nothing. We don't accept such changes impacting on human health or domestic animals' health, we should not accept them as an excuse to do nothing for wombats.

The "We must do something else first"

There are many things we could do to improve our environment and many people have expressed ideas about what could be done to help the health of wombats. The Society's goals, outlined in Bulletin 3 December 2006 set seeing the Riparian Zone fenced from stock and used as native animal corridors as one of its goals. It argues that with improved riparian zones available as wombat habitat, river flows will eventually increase, wombat burrows will be more regularly "flushed" and the nutrients available in river grasses will

improve. This improved healthy environment should see an improvement in wombat health, BUT the Society would like to have wombats still around to enjoy such environs.

Mange can and will lead to there being no wombats. Waiting until ultimate goals are achieved will be waiting too long.

Other people are concerned that until supposed vectors like foxes and dogs are controlled any positive steps to remove mange would be countered. We need to be cautious with our acceptance of any reason for not taking action now and while goals of what could be direct the way, not being there now should not stop the journey. There is often as much evidence against a particular theory that is used to justify inaction as for it. The dog/fox vector impediment does not explain why mange is found in Tasmania and Flinder's Island where these vectors are not present (the fox has recently, illegally been introduced into Tasmania but this temporally has nothing to do with wombat's there having mange). (Uni.W.A.FoxDNAProject2007) . It is also quite possible that the fox is not the vector, yet this theory and the burrow sharing theory are commonly held beliefs. The mange query response answered from Victoria includes; "The red fox may be a major vector for mange in wombats, through sharing of burrows. Control of foxes may assist with reducing the incidence of mange..." (2006.Letter.) The jury is out on the fox as vector. Mange mites have only been genetically differentiated for two groups of animals, humans and dogs. After this all the various "varieties" of mites are assumed. Thus var. wombatti has yet to be fully distinguished from that infesting foxes, which, because of the dog/fox relationship is termed var.canis. It still remains to be proven that the fox transmits mites and while there may be other reasons for taking action against foxes, dogs and dingos; the belief that wombat's mange can't be dealt with until foxes or any other supposed vector is managed is just another delaying tactic and one not substantiated by any evidence other than supposition.

The "Treatment is Worse than Disease Impediment"

There is no doubt that there are very mixed results from various treatments used and prescribed to treat wombats with mange. Most wombats with mange that get treated are either very young or so infested that they have been able to be captured. It should therefore be said, that those treating wombats with mange are working on the hard edge of treatment and it should not be surprising that many of these animals do not survive. Skerrat found that wombats showing what he termed severe mange had, in many cases developed secondary infections and that these bacterial infections were most likely caused through bacteria entering the skin via the open fissures. In the small number of wombats he studied he found "emaciation and haematological and biochemical changes consistent with starvation and anaemia" and that severe infestations "predispose wombats to bacterial infection and result in a leucogram indicative of inflammation" (2001,80). It is reasonable to conclude that the longer it takes to treat a wombat with mange, in general, the harder it will be. So it is not surprising many of the more severe cases don't make it.

Many people are concerned that some of the chemical treatments for mange have been linked in wombats and other animals to other problems such as tumours and growth

defects and they are concerned about returning wombats with a potentially “fatal genetic timebomb” (member no.694324) back into the population and creating a bigger problem than the one we started with. As mentioned previously, there are wombat advocates who would believe killing a wombat with mange is better than it having to be kept in captivity for any period of time, including the initial capture to treat. Others believe that releasing a cured or healthy wombat into any area where mange is seen will lead to that wombat dying and they would recommend sanctuaries and permanent captivity. Others are concerned that what they believe to be the only successful available treatments are both cruel and unsuccessful. So the “treatment is worse than the disease” attitude actually is quite diverse in its underpinnings are significantly different from person to person. That being said again we can apply the human model to the problem.

In humans, when a disease is only treatable by a method that either causes secondary problems or is painful/ uncomfortable, work is done to improve the method and treatment modality. If we get caught up in arguing why treatment CAN BE worse than the infestation itself we fall again into the trap of thinking of mange as an incurable disease, not a simple parasitic infection. If we only look to animal models for how to remedy mange, we will find an array of failures because that is precisely what the problem is. We have failed to find a humane, effective and successful method to treat wombats in the wild with mange. The energy debating why we shouldn't detracts from the energy needed to determine how we can. And, as with humans, whether we can meet every qualm or concern about our preferred treatment will be informed by those who have voiced their concerns about current treatments.

The “We Need to do more Research Impediment”

Scabies doesn't take rocket science to cure and for hundreds of years before profit making chemical companies became involved in veterinary products mange was managed in a variety of animal communities quite successfully. The entymology of the mite is well documented and what kills it is well known. While further DNA studies into the mite may explain its evolution and its predilections for certain hosts at certain times historically, and while this research may lead to better methods in the future, it is not necessary to know these things NOW to ACT. The mite, whatever its evolutionary history and its apparently changing DNA structure, is able to be killed off easily. It doesn't survive long without a host and whatever we find in future was its origins, that will yield little value if we wait until we have no wombats left . The behaviour of the wombat and how the mite spreads from one to another is also called into play as needing further research. Whether mites transfer through contact, burrows, scratching places or vectors isn't really relevant except in so far as these issues may inform how we most effectively treat the epidemic/ epizootic. If we knew for example that mites were only transferred through direct contact wombat to wombat, how would that change the need to treat and eradicate mange from as many wombats as possible? If we knew that mites were transferred from burrow sharing, how would that change the need to kill off the mites on animals so if they did burrow share they couldn't infest one another ? Given once the mite is on a wombat, it rapidly breeds up and then is available to infest other wombats, how does learning more about its rate of infestation help? Similarly how does knowing more about whether transfer is from burrows or scratching places help when we

already know if the mites are not removed from wombats the wombats will in most circumstances, be dead within a short period of time and through whatever means, could potentially transfer mites to another animal. In humans we would rapidly deal with the infestations and then ponder these issues. Why do we behave so differently because wombats are affected?

What We Do Know and Where That Suggests We Should Go

Our information review, suggests that we have enough information and enough research to set up a programme to eradicate mange. While knowing more about the mite's DNA, evolution, means of transfer may assist "fine tune" such a project, these things are not necessary to devise and begin taking action. They may be necessary to get funding or to get government involvement. That is not the same thing as saying we need more research to work out how to proceed. To this end the following KNOWN and EXISTING information does not need further research or replication. It needs INCORPORATION into a PROACTIVE plan for ERADICATION.

1. We know that sarcoptic mange is caused by a mite that can be easily eradicated using a variety of acaricides. These products exist commercially, eg; ivermectin, advocate, porcine ; in elemental form, eg; sulphur and they exist in nature ,eg. tea tree oil/eucalyptus oil, pyretherin.

2. We know that the intensity of mite infestation correlates with the severity of clinical signs seen in infested animals.

3. We know that if left, in general, sarcoptic mange progresses, mites increase incrementally and the affected wombat dies. We know this death is slow and painful and avoidable.

4. We suspect that the death of an heavily infested wombat in its burrow, if shared with another wombat, is the most likely means by which an intense load of mites will be transferred to another wombat, sufficient, irrespective of previous contact to the mite and possibility of immunity, to cause sarcoptic mange in that animal.

5. We know that mites do not live long off their host and burrows, even if infested via the death of a wombat from sarcoptic mange , will be mite free within three weeks. Leaving wombats with sarcoptic mange to die is likely to lead to more wombats dying from mange.

6. We know that treating wombats with early stages of clinical signs will lead to complete resolution of mange and the wombat will be healthy and not more likely to become reinfested, probably less so.

From what we know, we recommend;

1. We should take action to eradicate mange in wombats.
2. Wombats with clinical signs of mange should be treated with an acaricide.

3. Wombats with severe mange should be targeted for treatment and need, in addition, antibiotics to assist secondary infections preventing their deaths. Treatment of these animals is particularly important not only to prevent their suffering but to ensure no other wombat becomes infested.
4. Carers of hand reared wombats turning these animals from captive to free should avoid burrows where a wombat has died from sarcoptic mite infestation for at least three weeks.
5. Carers of wombats with severe mange should avoid allowing that animal to die and if it does should ensure no other wombat has contact with its bedding, housing or burrow for a period of at least three weeks.

The Next Step

In the next phase of this project, we will investigate the various treatment options. Taking into account various comments and concerns about wombat welfare, we believe options for treatment should be evaluated against certain criteria;

1. The degree of interference with the wombat's normal routine and lifestyle. The better treatment will not interfere, or limits the degree of interference with the normal routine and behaviour of the naturally raised free wombat.
2. The degree of likely impact on the longer term health of the animal. The better treatment has no new negative impacts on the long term well being of the wombat.
3. The degree of ease with which the treatment can be supplied to the wombat. The better treatment is simple, safe and able to be provided by a range of people.
4. The universality of treatment. The better treatment will be able to be used on wombats whatever their stage of mange development and should cause no harm to any other wombat that may come in contact with the treatment modality.
5. The unintended consequences of treatment. The better treatment should have no or as few unintended consequences to those using the treatment, other animals that may inadvertently come in contact with the treatment and the environment in general.

The next stage of our project will evaluate all known acaracides against these criteria.

EXTENSIONS TO PART ONE.

Mite Entomology -Understanding

This section is a summary of a paper on Scabies produced by the Department of Medical Entomology from Sydney University. It is available on line

<http://medent.usyd.edu.au/fact/scabies.html>

The “enemy” is a small mite, only just visible to the naked eye called *sarcoptes scabiei*. Enlarged, it looks a little like a tick with hairs hanging from it and the female of the species has short blunt spines which help her hold on in the tunnels she excavates in subcutaneous tissue. Like wombats, the mite has short thick legs. Immature larval scabies have six legs and this stage is followed by two nymphal stages both of which have eight legs onto the final adult stage where eight legs remain.

The female of the species is the main cause of problems for wombats. Within a very short period following mating, she digs a tunnel into the skin and lays 2-3 eggs there each day. The mite continues to burrow and lay eggs, eating the skin and tissue fluids which ooze from these tunnels and she progresses her tunnels into tissue at a rate of between 0.5mm and 5 mm PER DAY. Each tunnel contains only one female, her eggs and her faecal waste which is the irritant that causes most of the problems. Her eggs hatch in 48 hours and the larval mites dig their way to the skin surface where they begin their own deconstruction work. The larval mites dig smaller tunnels or hang onto hair follicles to complete their first moult and move to the next stage. They remain there eating body fluids from around hair follicles or make shallow burrows until the next stage which moults leading to adulthood. The newly moulted adults dig short tunnels/ burrows prior to mating. Once mated, the cycle that has taken 10-17 days to complete is finished. It is the newly fertilized females wandering around on the skin looking for a place to tunnel that can transfer to any other available host. Once they have dug in they rarely move and if dislodged by scratching they will dig back in. Mites have a high mortality with 90% of hatchlings dying and *mites removed from hosts die quickly*. “Quickly”, however can be as long as three weeks in optimal conditions.

The fact that mites removed from hosts die quickly is of interest when techniques to control mites in wild wombat populations are considered. It suggests that fumigation of burrows would not be as useful a strategy, for example, as killing off the living mites on living wombats, however, recognizing that even if that is achieved, the concern that a wombat returning to its burrow may encounter still living mites and become reinfested needs to be considered. This was not validated in Lee Skerratt’s study, (2001,p.250) in fact the opposite was shown. Mild to moderate infestations were not passed onto other wombats sharing burrows despite Lee proving that the infested wombats did develop clinical signs of mange, did have high mite counts and did share burrows. (2001,pp161-185). Putting both these KNOWN FACTS together, would recommend some form of ongoing treatment that can be repeated until the life cycle of the mites is exhausted and no living mite can lay in wait in burrows.

In humans with scabies the initial infestation can remain undetected for a month or more. It is when the mite’s waste products, faeces, skin moults, saliva and moulting fluids move

into surrounding tissues that sensitization begins and the allergic reaction to these components causes severe itching. While the itching can be widespread and seems particularly to affect humans at night it does not necessarily relate to where mites are buried. In humans large areas of skin can be covered by a rash that only rarely coincides with areas of mite infestation. The rash areas can erupt into smaller itchier lesions. Because the mite buries in where it is least likely to be disturbed in humans this is often finger webbing and elbow and wrist creases. The scratching is what causes secondary infection and heavy infestations have led to anaemia . It is similar in wombats with the area between the ears, where wombats reach least successfully to scratch, often carrying the heaviest mite burden and apparently healthy looking wombats carrying increasing mite populations. It just depends at what point in the infestation process they are seen.

IDENTIFICATION OF THE MITE

Laboratory Diagnosis

Skin scrapings are examined with a compound microscope for the presence of mites, eggs or faeces. A glass slide is prepared using dilute potassium hydroxide or lactic acid to clear thick layers of skin cells. Clearing, using this process can take time and the reference article indicates hours to days. Information and Identification of scabies mites is provided through the Medical Entomology Department at ICPMR Westmead Hospital N.S.W. in humans.

IMMUNITY

The excerpt below from the document is included as it seems pertinent to observations made by various members that correlate with observations of mange in wombat *“untreated scabies infestations, especially in infants, immobilized geriatric patients AIDS and other immunologically compromised patients can support huge numbers of female mites. The patient’s skin may become crusted on the surface, with the underlying layers soft and honeycombed with tunnels, these infections are referred to as “Norwegian” or “crusted” scabies. Patients with this advanced state of infection can act as a source for local epidemics in health care facilities. Reinfected patients will develop an immediate itch when another scabies infection is initiated.”*

(<http://medent.usyd.edu.au/fact/scabies.html>, 2007)

This observation of the mite in humans has been reported in wombats and sometimes leads to the belief that their immune system is compromised because a reinfested wombat begins to scratch earlier than with a first infestation. In some wombats hypersensitivity reactions to *Sarcoptes* to only a few mites in the skin occurs” (Skerratt,2001,p32).

Reactivity is not the same as a compromised immune system. Some carers assess whether an animal has mange when no apparent signs exist by cuddling the animal and checking their own reactivity some hours later. Second infestations do cause earlier reactivity. The immune system is more reactive with a second infestation. This is a good indicator that even apparently healthy wombats should be proactively treated. Wombats love to scratch, whether they have mites or not so determining whether to treat a wombat by whether it

has observable signs of mange by scratching is counterproductive. Skerratt evaluated whether scratching was a good indicator of level of infestation and found that it was not. He suggested it was more likely to be an immune response and the early response to the mange mite might retard the development of mange (2001,p.100).

Skerratt found the presence of nucleated and polychromatic erythrocytes in the blood of some wombats and suggests that this may be indicative of a regenerative response to the mange, possibly occurring in the bone marrow (2001,p.78)

He also found that the dermal cellular immune response of the host toward the mite regulates the intensity of the infestation (2001.p.100) and despite their being a slower antibody response in metatherians (marsupials) (p.120) that “free living wombats with severe sarcoptic mange exhibited a significant inflammatory cellular response in the upper dermis(p.126) as did captive wombats experimentally infested (p.127) and that these responses were “typical of an immune response” (p.128) and that the “speed of the immune response was proportional to the mite density”(p.128). Eosinophils in the dermis correlate to mite density and this suggests they are a response to the products of the mite (p.129). From this Skerratt also suggests that a vaccine could possibly be made from an antigen of *S.scabiei* and this may, in the future provide protection.(p.101).

WHERE DID IT COME FROM?

There remains a lot of debate as to where the mite came from and this debate is not helped by the mite being classed on the basis of its host. Only two mites have been identified as differing from one another sufficiently to be classed separately. These are varieties *hominis* (the one affecting humans) and *canis* (the one affecting dogs). “Mites from humans and dogs have been shown to be genetically distinct using allele frequencies” (Skerratt,2001,p36) “The taxonomy of varieties based on host origin has only been validated genetically for *var.canis* and *var.hominis*”(Ibid.p.37). Phenotypic differences such as physiological differences amongst mites have been observed and may have reduced mites’ ability to establish on another host . Mites are preferentially attracted to host odours and the species they infest . Some mites exhibit unique immunogenic antigens. (Skerratt,2001,p.36). It is only ASSUMED at this stage that the mite affecting dingos (which are actually Asiatic wolves- see Triggs,2002,116) and foxes are the same. It is only an assumption made by observations of burrow sharing that the fox is implicated as a vector for wombats getting mites. There is some evidence to suggest that the fox can’t be the vector. Foxes were only introduced to Australia in the 1850s, yet mange was reported in the early 1800s. (Skerratt,2001,p34) The mite on wombats cannot be distinguished morphologically from mites found on goats, sheep, llamas, horses, dogs, ferrets, rabbits and foxes – however they can be distinguished morphologically from the mite found on humans and some animals such as camels, cows and pigs. (Skerratt,2001,p.34). Morphology refers to the form or structure of an organism or an organ, tissue or cell. Phenotypic (outwards, observable expression of hereditary) differences are believed to arise through the mite adapting to host species they

infest.(Skerratt,2001,p.37). There is also debate as to whether the mite came to Australia with the first explorers or earlier when the dingo was introduced.

There is also debate as to whether the mite changes to become host specific and possibly locality specific as well, and if this is occurring how long that process takes. What we do know is that at the current moment there are only two mites able to be genetically differentiated and those are *canis* and *hominis*.

Lee Skerratt for his Phd. from the University of Melbourne describes the scabies or mange mite used to infest wombats as *Sarcoptes scabiei* var.*wombatii* (Acari: Sarcoptidae) . He and another researcher also examined mites from dogs, humans and wombats from different locations. They concluded “ there were no fixed genetic differences between *S.scabiei* on different hosts or localities nor was there significant phylogenetic divergence among mites from different hosts or localities.” They found some differences between mites that don’t appear to be specific to host. This part of Lee’s study is interesting because we hear from member number 694324 who regularly treats mangey wombats that the mite from the wombats has given him mange on a number of occasions. In addition we heard about another wombat carer in N.S.W. who holds apparently unmanged wombats for a period of time and assesses by the itch they develop some hours later whether or not the animal is carrying mange mites. Lee also reports that two researchers handling dead mange infested animals developed mange. He also quotes one of the earliest reports of mange in the early 1800s and how people who handled that wombat also got mange. Now Lee purposely infested the wombats studied with a specific variety of mite and his work is done on its progress and spread. The study was quite artificial in terms of how contact between wombats is likely to occur with Lee putting 5000-8000 mites on each wombat. Wombats having casual contact with each other may distribute mange mites, but not in the proportions artificially engineered by Lee’s study.

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