CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

1.1. INTRODUCTION

Diseases of wombats and most other Australian marsupials have been little studied. Recently there has been an increased interest in diseases of free-living animals and the ecological role of diseases in populations, particularly their ability to regulate animal abundance (Scott, 1988; Thorne and Williams, 1988; Lyles and Dobson, 1993; Spalding and Forrester, 1993; Robinson, 1996; Tompkins and Wilson, 1998; Daszak et al., 2000). There is also an awareness that free-living species may act as reservoirs of disease of man and domestic animals (Robinson, 1996; Daszak et al., 2000). Therefore it is an opportune time to investigate an important disease of wombats, sarcoptic mange, which may regulate wombat abundance. This is particularly relevant to small isolated populations of wombats threatened with extinction (Wells and Pridmore, 1998). In addition, sarcoptic mange is also a disease of man and domestic animals (Doube, 1982).

Sarcoptic mange is a cosmopolitan disease of mammals caused by the mite Sarcoptes scabiei. There have been numerous studies of sarcoptic mange in domestic animals and in humans where the disease is referred to as scabies. In contrast there have been few studies in free-living mammals (Arlian, 1989; Burgess, 1994). However, extrapolating knowledge of the disease in other mammals to wombats may
be invalid since the wombat is unique as a fossorial marsupial herbivore (Wells and Pridmore, 1998). Therefore, it is necessary to assess whether sarcoptic mange in wombats is similar to sarcoptic mange in other animals and to identify any aspects of the disease peculiar to wombats.

1.2. LITERATURE REVIEW

1.2.1. SARCOPTIC MANGE IN WOMBATS

Of the Australian marsupials, sarcoptic mange has only been reported in wombats and the koala (*Phascolarctos cinereus*) (Vombatiformes) and the common ringtail possum (*Pseudocheirus peregrinus*) (Pseudocheiridae) (Wells, 1971; Southcott, 1976; Domrow, 1992; Skerratt, 1998). However, sarcoptic mange rarely occurs in free-living koalas and common ringtail possums whereas it is common in wombats (Domrow, 1992; Martin et al. 1998; Martin and Handasyde, 1999). There are three extant species of wombats. The northern hairy-nosed wombat (*Lasiorhinus krefftii*) is now confined to a single locality north-west of Rockhampton in Queensland (Qld) (Johnson and Gordon, 1995). The southern hairy-nosed wombat (*Lasiorhinus latifrons*) occurs in South Australia (SA) and south, eastern Western Australia (WA) (Wells, 1995). The common wombat (*Vombatus ursinus*) occurs in Victoria (Vic), New South Wales (NSW), the Australian Capital Territory (ACT), Tasmania (Tas) and Flinders Island, the south-east of SA and south-east Qld (McIlroy, 1995). All are medium-sized (up to 40 kg) fossorial marsupials. However, relatively little is known about their population biology or the effect of disease on wild populations, including the major infectious disease affecting wombats, sarcoptic
Sarcoptic mange is predominantly found in one of the three species of wombat, the common wombat (*Vombatus ursinus*) (Gray, 1937; Wells, 1971; Doube, 1982; Presidente, 1982; Skerratt, 1998). In this species, sarcoptic mange can be debilitating. Several reports of severe sarcoptic mange in common wombats describe animals as emaciated and lacking hair, with a thick dry crust, composed of keratin, many mites and their debris, bacteria and neutrophilic debris adherent to skin (Sweatman, 1971; Presidente, 1982; Skerratt, 1998). When crust is present, the disease is called hyperkeratotic, crusted or sometimes parakeratotic sarcoptic mange. In humans it is referred to as “Norwegian” scabies since the condition was first recognised in Norway in leprosy patients by Boeck and Danielssen in the 19th century (Burgess, 1994). Here, the term parakeratotic is preferred because it describes the disturbance to the process of keratinisation caused by the tunnelling of mites in the skin (Van Neste and Lachapelle, 1981; Van Neste and Staquet, 1986). Parakeratotic sarcoptic mange in wombats is similar to that found in domestic animals and humans (Skerratt, 1998). In addition, the thick crust may be fissured by the movement of the wombat (Skerratt, 1998). The underlying skin may also crack, resulting in haemorrhage, pyoderma and sometimes cutaneous myiasis (Skerratt, 1998). Sarcoptic mange may encompass the entire body of the wombat with the head, neck, shoulders and limbs most commonly affected (Skerratt, 1998). Typically, the epidermis is thickened and there is a mild, predominantly mononuclear, inflammatory infiltrate in the dermis (Skerratt, 1998). In some wombats, hypersensitivity reactions to *Sarcoptes*, with few mites present in the skin, occur (Munday, 1978). Intense pruritus, a characteristic sign of hypersensitivity reactions to *Sarcoptes* in humans and animals
(Arlian, 1989; Burgess, 1994), has been reported in wombats with sarcoptic mange (Perry, 1983). Only occasional outbreaks of sarcoptic mange have been recorded in the southern hairy-nosed wombat (Lasiorhinus latifrons) (Wells, 1971) and the disease has not been reported in the northern hairy-nosed wombat (Lasiorhinus kreffii).

The first report of sarcoptic mange in a common wombat was by Latreille (1818). His report contains a description of two species of mite that were collected from a mangy wombat held live at the Muséum nationale d’Histoire naturelle, Paris in the early years of the 19th century. From this description one species appears to be the mange mite, *S. scabiei*. The other species appears to belong to the genus *Acaroptes* Womersley, 1953, a genus that is commonly found on the surface of the skin of apparently healthy wombats (Womersley, 1953; Skerratt, 1998). The wombat with mange subsequently died and its preserved skin is almost certainly the one in which Duméril found mites that were considered by Fournier to be identical to *S. scabiei* found on man (Railliet, 1895). People who were in close contact with this wombat developed signs of scabies (Latreille, 1818; Railliet, 1895). They were treated with sulphur and the signs resolved (Railliet, 1895). Organic or metabolised products of sulphur appear to have acaricidal activity and for many years sulphur was the drug of choice when treating scabies (Burgess, 1994).

The wombat from which Latreille collected the mange mites was one of three common wombats brought back alive to France on the “Naturaliste”, a ship from Nicholas Baudin’s expedition to Australia between 1800 and 1803. They were taken to the zoological park of the Muséum national d’Histoire naturelle in Paris (Geoffroy,
1803; De Beaufort, 1966; Horner, 1987). The skins of the common wombats collected by Baudin are still in the Muséum (De Beaufort, 1966) and one of the mounted animals has lesions consistent with mange, although no *S. scabiei* mites were detected in a recent examination (I. Beveridge, unpublished observations). The two mange-free wombats became type specimens of the now extinct King Island subspecies. The mangy wombat was possibly the ‘extraordinarily large wombat’ given to Baudin on board his ship the “Géographe” by the Captain of an English schooner returning from sealing whom Baudin had met two days out from Port Jackson (De Beaufort, 1966; Cornell, 1974; Horner, 1987). This wombat was transferred to the other ship commanded by Baudin, the “Naturaliste”, at Sea Elephant Bay, King Island, before the “Naturaliste” sailed for France (Horner, 1987). Womersley suggested that this wombat may have come from Tasmania (Womersley, 1953).

The fact that a common wombat had sarcoptic mange in the early years of the 19th century raises the possibility that *S. scabiei* var. *wombati* is an endemic parasite of common wombats, possibly introduced to Australia by aboriginals, but it is also possible that Baudin’s afflicted wombat contracted *S. scabiei* from a person or one of the variety of animals (including dogs) held on the Naturaliste during its long voyage back to France (Horner, 1987). It is likely that the wombat did not have signs of mange when the Naturaliste departed from Sea Elephant Bay since all animals on board appeared healthy at the time (Cornell, 1974; Horner, 1987). *S. scabiei* may have been brought to Australia by the dingo (*Canis familiaris dingo*) several millennia ago (Corbett, 1995), since dingoes have been reported with sarcoptic mange (McCarthy, 1960a; McCarthy, 1960b; Hoyte and Mason, 1961). If *S. scabiei* was not present in Australia prior to the first white settlers arriving in 1788, then it is possible that there
have been several recent introductions of the parasite by immigrants and their animals. Dogs, foxes, pigs, horses and camels are hosts of *S. scabiei* in Australia (Domrow, 1992) and all have been introduced within the last 200 years (Strahan, 1995). Of these animals the one most likely to transmit *S. scabiei* to wombats is the European red fox (*Vulpes vulpes*). It occasionally utilises wombat burrows, where conditions of high humidity and low, relatively stable temperature (Brown, 1964; Triggs, 1996) appear ideal for the survival and transmission of mites (Arlian, 1989). The fox was introduced to Australia around 1850 (Rolls, 1984). If *S. scabiei* has been recently introduced into wombat populations then the host-parasite relationship could be a rapidly evolving one as has occurred with myxovirus in rabbits in Australia and Britain (Ross, 1982).

The variety of *S. scabiei* found on wombats, named *wombati* by Railliet (1895), was formally described by Womersley (1953) (Fig. 1.1). Fain (1968) concluded that female *Sarcoptes* from wombats were morphologically indistinguishable from those found on many animals such as goats, sheep, llamas, horses, dogs, ferrets, rabbits and foxes. However, they could be differentiated from female *Sarcoptes* found on humans and some animals such as camels, cows and pigs based on the lack of a clear area within the field of dorsal scales and the inconsistent presence of ventral scales. The host specificity of *S. scabiei* var. *wombati* is unknown, although humans, dogs and koalas have developed *S. scabiei* infestations after contact with common wombats which were, or appeared to be, infected with *S. scabiei* var. *wombati* (Latreille, 1818; Railliet, 1895; Gray, 1937; Barker, 1974; Arundel et al., 1977; Arlian, 1989).
The earliest report of an epizootic of sarcotic mange in free-living common wombats occurred in 1937 in southern New South Wales and resulted in a decline in wombat numbers from thousands to a few (Gray, 1937). Outbreaks of sarcotic mange in southern hairy-nosed wombats were first reported in 1971 (Wells, 1971), and *S. scabiei* were first collected from southern hairy-nosed wombats in 1976 (Southcott, 1976). Despite reports of many recent declines in the local abundance of common wombats and the fact that the distribution of all three species of wombats has contracted markedly since European arrival (Johnson and Gordon, 1995; McIlroy, 1995; Wells, 1995), little is known about sarcotic mange in wombats.

### 1.2.2. BIOLOGY OF *SARCOPTES SCABIEI*

The history of *S. scabiei* predates modern times. A probable early reference (ca. 1200 BC) occurs in Leviticus 13: 1-8 when diseases causing scale are mentioned. Aristotle (384-322 BC) may have first recognised *S. scabiei* when he recorded the presence of a parasite, which he described as a louse, living in the skin of people (Roncalli, 1987). Several Roman statesmen described various treatments for mange in animals (Roncalli, 1987). However advances in understanding the aetiology of scabies were not made until the microscope invented by Jansen was available to Giovanni Cosimo Bonomo (1663-1696 AD) to study the mite (Friedman, 1948; Roncalli, 1987). Latreille (1802) is credited with the name *Sarcoptes scabiei* but Linnaeus (1758) first named the mite *Acarus siro var. scabiei* (Fain, 1968). A number of studies in the following two hundred years significantly improved the understanding of the morphology and life-cycle of *S. scabiei* (Friedman, 1948). However, it was not until the second world war when serial experimental infections on conscientious objectors
to military service significantly advanced the understanding of the symptoms, parasitic infection and immune response of scabies (Mellanby, 1944; Mellanby, 1985b). In the last 30 years that there have been studies on a number of different aspects of the biology of *S. scabiei* such as morphological variability, host preference, life-cycle, associated pathology, immunogenicity, transmission, epidemiology and treatment (Arlian, 1989; Burgess, 1994; Burgess, 1999).

*S. scabiei* is a cosmopolitan parasitic mite infecting at least 47 different mammalian host species belonging to 21 families and 8 orders, the Artiodactyla, Perissodactyla, Carnivora, Primates, Insectivora, Lagomorpha, Rodentia and Diprotodonta (Sweatman, 1971; Southcott, 1976; Fain, 1978; Andrews, 1983; Klompen, 1992; Domrow, 1992). Generally, mites from different host species cannot be differentiated morphologically from one another (Fain, 1968; Kutzer, 1970; Pence et al., 1975; Fain, 1978; Zahler et al., 1999). This is because the degree of morphological variability is similar within and among mites on different species of hosts (Fain, 1968; Pence et al., 1975; Fain, 1978; Zahler et al., 1999). However, populations of mites from humans and dogs have been shown to be genetically distinct using allele frequencies (Walton et al., 1999a). Phenotypic differences such as physiological differences among mites from different host species have also been observed (Arlian, 1989). Mites may exhibit reduced ability to establish infection on another host species, are preferentially attracted to the host odour of the species they normally infect and exhibit some unique immunogenic antigens (McCarthy, 1960b; Stone et al., 1972; Barker, 1974; Fain, 1978; Samuel, 1981; Arlian et al., 1984b; Abu-Samra et al., 1985; Ibrahim and Abu-Samra, 1987; Arlian et al., 1988c; Arlian, 1989; Arlian et al., 1996b). It is thought that these phenotypic differences arise due to mites
adapting to the host species they infect (Fain, 1978; Arlian, 1989; Burgess, 1994). Interestingly, some mites will readily infect and maintain an infection on a different host species (McCarthy, 1960b; Stone et al., 1972; Barker, 1974; Samuel, 1981; Abu-Samra et al., 1985; Ibrahim and Abu-Samra, 1987). For these reasons populations of *S. scabiei* on different host species are generally regarded as different varieties although some varieties have been described as distinct species (Fain, 1968; Kutzer, 1970; Pence et al., 1975, Zahler et al., 1999; Walton et al., 1999a). The mite on red foxes, *Vulpes vulpes*, is regarded as belonging to the variety “canis” because of its ability to readily infect and maintain infection in other canids (Stone et al., 1972). However, numerous different varieties and species of *S. scabiei* have been described based largely on the species of host they infect (Fain, 1968; Kutzer, 1970; Fain, 1978). The taxonomy of varieties based on host origin has only been validated genetically for var. *canis* and var. *hominis* (Walton et al., 1999a).

It is thought that the ancestor of *S. scabiei* was a parasite of primates (Fain, 1968; Andrews, 1983). Fain (1968) suggests that it is derived from the genera parasitising non-human primates. Andrews (1983) suggests that *Sarcoptes* evolved as a separate lineage from other species within the Sarcoptinae some time after the cercopithecoid and hominoid hosts diverged, based on cospeciation of these parasites with their hosts. In Klompen’s (1992) phylogenetic analysis of the Sarcoptidae (length 258, CI = 0.520), the Sarcoptinae parasitising primates formed a clade. *Kutzerocoptes*, a parasite of monkeys (Cebidae), was basal to *Prosarcoptes*, parasitic on monkeys (Cercopithecidae) and *Sarcoptes* (Klompen, 1992). *Trixacarus*, a genus that infects rodents, formed a sister group within the Sarcoptinae (Klompen, 1992). However, Klompen (1992) warned that only two additional steps (260 versus 258) were required
for the Sarcoptinae associated with primates to form a sister group with the Teinocoptinae, a monophyletic group associated ancestrally with bats, although some have transferred to other small mammals (Klompen, 1992). Interestingly, apart from *Sarcoptes scabiei*, the Sarcoptidae parasitising marsupials, *Diabolicoptes sarcophilus*, *Satanicoptes phascogale* and *Satanicoptes armatus*, form a monophyletic group, the Diabolicoptinae, that is basal to the Sarcoptinae and the Teinocoptinae (Klompen, 1992). The Sarcoptidae is one of 14 families within the Sarcoptoidea, a superfamily that exclusively parasitises mammals (O’Connor, 1982). O’Connor (1982) assigned it to the cohort Psoroptidia along with three other superfamilies, which are mostly parasites of birds, Pterolichoidea, Analgoidea and Pyroglyphoidea, in the sub-order Astigmata.

*Sarcoptes* is thought to rely mainly on close contact for transfer between hosts although fomites are also important (Mellanby, 1985b). This is because the mite relies on the environment supplied by its host for survival. Once off the host, the mite is liable to starve and desiccate. It can survive for approximately 3 weeks under favourable conditions of low temperature, 10° C, and high humidity, 98 % (Arlian et al., 1989). In addition, mites can not actively seek a host when the temperature is below 20° C (Epstein and Orkin, 1985). However, *Sarcoptes* can be readily transmitted between hosts that share burrows because of the stable, generally favourable environment provided by burrows that enables mites to survive for comparatively long periods (Gerasimov, 1953). Phoresy of mites on flies may also enable transfer of mites between hosts (Gerasimov, 1953). Due to these constraints, Fain (1968, 1978) and Andrews (1983) proposed that the spread of *Sarcoptes* from man to animals was due to domestication of animals. Andrews (1983) suggested that
dogs were first infected due to their early domestication, 14,000 years ago. Dogs then spread infection to other wild carnivores. Ungulates were infected when they were domesticated, 4,000 – 11,000 years ago (Andrews, 1983). The less likely modes of transmission such as by fomites, burrow-sharing or phoresy may explain transmission of *Sarcoptes* that would be unlikely to occur by direct contact (Gerasimov, 1953; Andrews, 1983). Andrews (1983) and Fain (1968, 1978) suggest that a reduced immune response in a recipient host would facilitate transfer of mites between species. They also argued that the morphological variability in *Sarcoptes* populations ensures the presence of individual mites capable of infecting a different host species. Andrews (1983) suggested that variation within the skin of an individual host species maintains this morphological variability.

*S. scabiei* is minute, approximately 100 to 500 µm in length, and barely visible to the naked eye (Fain, 1968). Generally, it is ovoid, flattened dorso-ventrally and appears white, with brown legs and epimeres, structures within the body that support the legs. It tunnels into the superficial layers of the epidermis of mammalian skin and is found within the stratum corneum and Malpighian layers (Van Neste and Lachapelle, 1981; Van Neste and Staquet, 1986). The life-cycle of the mite consists of five stages: egg, larva, protonymph, tritonymph and adult, with ecdysis occurring within tunnels (Heilesen, 1946; Fain, 1968). The deutonymphal life-cycle stage has been lost in the Psoroptidia (O'Connor, 1982). Sexual dimorphism is apparent by the tritonymph stage (Fain, 1968; Arlian and Vyszenski-Moher, 1988). Males appear to seek out females to mate and then eggs are laid by females within tunnels at a rate of 2 to 3 per day (Heilesen, 1946; Mellanby, 1985a). The life-cycle is completed within
10 to 14 days although females may survive for 4 to 5 weeks (Mellanby, 1944; Heilesen, 1946; Friedman, 1948; Arlian and Vyszenski-Moher, 1988).

The clinical signs of *S. scabiei* infection usually become apparent 2 to 4 weeks after infection. This is regarded as the sensitisation phase of the infection (Mellanby, 1944; Davis and Moon, 1990b; Little et al., 1998). In man, a number of different lesions due to *S. scabiei* infection have been identified such as: serpiginous burrows containing mites, vesicles, erythematous papules, nodules and body rashes (Epstein and Orkin, 1985). Secondary complications also occur due to the pruritus associated with scabies such as excoriation and bacterial infection (Epstein and Orkin, 1985). Primary immune responses generally limit the number of mites in people and domestic animals (Mellanby, 1944; Davis and Moon, 1990c). Parakeratotic or crusted scabies, which arises due to high intensities of mite infection, appears to be rare in people and some domestic animals such as the dog. It usually occurs when there is an underlying illness or condition, which alters the normal immune response to *S. scabiei* (Anderson, 1981; Burgess, 1994; Paterson et al., 1995). Treatment of the host with immunosuppressive drugs may also lead to high intensities of mite infection (Davis and Moon, 1990c). The onset of an immune reaction is much more rapid with reinfection and may eliminate mite infection in domestic animals (Arlian, 1996; Arlian et al., 1996a). In contrast, high intensities of infection are relatively common in several species of wild mammals (Sweatman, 1971; Mörner, 1992; León-Vizcaíno et al., 1999) including wombats (Sweatman, 1971; Skerratt, 1998).

The epidemiology of *S. scabiei* is not well understood, with epidemics occurring periodically (Arlian, 1989; Burgess, 1999). The development and then
waning of immunity within populations has been suggested as the major reason for cyclical epidemics of scabies (Burgess, 1999). Supporting this is the fact that immunity has been shown to be important in reducing mite burdens (Mellanby, 1944; Arlian, 1996; Arlian et al., 1996a). Close bodily contact and high intensities of infection are important in promoting epidemics (Mellanby, 1985b). In foxes in Sweden, an outbreak of mange from 1975-1986 had its greatest impact on populations in mixed agricultural and forest areas compared with populations in boreal forest (Lindström, 1991). Seasonal changes in prevalence of sarcoptic mange and scabies have been attributed to effects of climate on the survival and reproduction of *S. scabiei* and the health of the host (Yeruham et al., 1996; Burgess, 1999). Concomitant disease and food shortage are factors that predispose individuals and therefore populations to sarcoptic mange and scabies (Mörner and Christensson, 1984; Skerratt, 1998).

At present, there are a number of acaricides derived from the macrocyclic lactones, pyrethrins and organophosphorus compounds that effectively treat sarcoptic mange (Havas Medimedia, 2001). Since the arrival of the avermectins into the pharmaceutical market in the 1980’s (Campbell, 1989) there have been numerous studies reporting the efficacy of various macrocyclic lactones against *S. scabiei* in a number of domestic species (Martineau et al., 1984; Logan et al., 1993; Losson and Lonneux, 1993; Arends et al., 1999). These acaricides are relatively easy to apply and have been used successfully to treat sarcoptic mange in wombats (Booth, 1999).

1.2.3. BIOLOGY OF THE COMMON WOMBAT
The common wombat (*Vombatus ursinus*) occurs mainly in forested areas along the Great Dividing Range from Melbourne to just north of Sydney, in coastal areas east of the Great Dividing Range in southern New South Wales and eastern Victoria and in Tasmania (Fig. 1.3) (McIlroy, 1973). Isolated populations occur in south-eastern South Australia, south-western Victoria, north-eastern New South Wales, Flinders Island and in the Wyberba area in Queensland near the New South Wales border (Fig. 1.3) (McIlroy, 1973). The distribution of the species in northern New South Wales and Queensland is limited to elevations above 600 m, which is thought to be due to the common wombat’s dependance on a mesic environment and its inability to withstand ambient temperatures above 25°C (McIlroy, 1973; Brown, 1984). In South Australia, Victoria and Tasmania common wombats occur in coastal scrub, woodland and heathland as well as forests (McIlroy, 1973). The species is protected in all states although it is unprotected in 193 parishes in eastern Victoria where it is still regarded as a major agricultural pest (Temby, 1998). The species is regarded as secure, although in South Australia common wombat populations are regarded as vulnerable (Temby, 1998).

The southern hairy-nosed wombat (*Lasiorhinus latifrons*) occurs mainly as four large separate populations in southern South Australia and south-eastern Western Australia (Fig. 1.3) (Wells, 1995). It is a protected species, although permits are issued for culling of wombats that interfere with agricultural practices and Aborigines may hunt wombats as part of traditional land practices (St John, 1998). In contrast to the secure status of southern hairy-nosed wombats, the northern hairy-nosed wombat (*Lasiorhinus kreftii*) is critically endangered and occurs as a single isolated population of approximately 70 individuals at Epping Forest in central Queensland.
(Fig. 1.3) (Johnson and Gordon, 1995; Horsup, 1998). The distribution of all three species of wombat has contracted since European settlement (Johnson and Gordon, 1995; McIlroy, 1995; Wells, 1995).

Despite the common wombat being one of Australia’s largest, most charismatic mammals, its biology is not well understood (Wells, 1989; McIlroy, 1995; Triggs, 1996; Wells and Pridmore, 1998). This is significant since in order to understand the relationship between *S. scabiei* and common wombats it is necessary to understand the biology of both. The aspects of the biology of the common wombat that relate to the transmission and epidemiology of sarcoptic mange in wombat populations are particularly important. Common wombats are generally solitary animals when foraging above ground and it is unlikely that mites would be readily transmitted between individuals (McIlroy, 1973; Taylor, 1993; Triggs, 1996; Buchan and Goldney, 1998). However, wombats will share a number of burrows for diurnal shelter where transmission of mites might occur, as has been demonstrated in fox dens (Gerasimov, 1953). Common wombats in the sclerophyll forest of Bucccleuch State Forest in NSW will use up to 11 burrows over several months (McIlroy, 1976). In the few wombat populations in which the extent of burrow sharing has been determined, wombats will generally share a burrow by occupying it at different times but occasionally will occupy it together (McIlroy, 1973; Taylor, 1993). At Bucccleuch State Forest, up to five wombats shared the same burrow and no more than two wombats occurred in the same burrow at the same time (McIlroy, 1973). However, the frequency of burrow use depended on the density of wombats (McIlroy, 1973). Variation in use of burrows between common wombat populations is demonstrated by comparing McIlroy's findings with the observation made by Douglas (1960) that up to
eight common wombats may be found in a single burrow. If *S. scabiei* has been recently introduced into wombat populations, then it is probably evolving to maximise its chances of being transmitted under the conditions of burrow use present in each population.

As pointed out by Andrews (1983), the mammalian skin is a relatively conservative organ however other aspects of the biology of the common wombat such as its physiology and grooming behaviour may affect its relationship with *S. scabiei*. Wombats have low metabolic rates and very low nutrient requirements compared with eutherian and even metatherian mammals of similar size (Dawson et al., 1989; Hume and Barboza, 1998; Johnson, 1998). It is possible that wombats are less susceptible to the energetic demands of *S. scabiei* infection compared with other mammals due to their low energy requirements. Wombats are dissimilar to most other animals affected by sarcoptic mange in that they do not groom themselves with their teeth or mouth but predominantly use theirs claws or various objects that they can rub against (Triggs, 1996). This unique behaviour may also affect the spread and predilection sites of the mite on the wombat’s body.
1.3. CONCLUSIONS

A study of sarcoptic mange in the common wombat, like many studies to date on the common wombat (McIlroy, 1973) would be the first of its kind in relation to a particular aspect of the biology of common wombats. McIlroy (1973) studied the ecology of common wombats, whereas the investigation of a specific disease, sarcoptic mange, is proposed here. The aim of this study is to provide knowledge that will enable better management of sarcoptic mange in wombat populations. In particular, this knowledge may improve the conservation of wombats, such as by helping to preserve small isolated populations that are threatened with extinction by various possible factors including sarcoptic mange. Whilst this study predominantly aims to advance our understanding, it may also lead to the development of a number of hypotheses to be tested in the future. In addition, due to the lack of studies of sarcoptic mange in free-living animals an investigation into sarcoptic mange in the common wombat will also advance the general understanding of this important cosmopolitan disease.

The research approach that was undertaken is outlined in the following pages.
1.4. RESEARCH APPROACH

For the reasons stated previously and because there is a lack of basic knowledge of sarcoptic mange in wombats the investigation was broad in scope. However, the lack of methodologies and resources in some areas limited their study. In order to confirm that sarcoptic mange is an important disease of wombats that predominantly affects the common wombat, a questionnaire survey to determine the distribution and prevalence of sarcoptic mange in wombat populations was undertaken. Whilst the results of a questionnaire survey should be interpreted cautiously, it can provide information that would otherwise be extremely laborious to collect (beyond the capacities of a PhD student). The results of a questionnaire survey are described in Chapter 2. In order to facilitate diagnosis of sarcoptic mange and understand the pathogenesis of sarcoptic mange in wombats the distribution of *S. scabiei* on wombats and the effects of sarcoptic mange on the health of wombats were determined. Since common wombats with severe parakeratotic sarcoptic mange occur commonly in the wild and are relatively easy to collect they were studied initially (Chapter 3).

Experimental infections of captive common wombats with *S. scabiei* were undertaken (Chapter 4). This was done in order to determine the sequence of development of clinical signs of sarcoptic mange in wombats to further improve recognition of early stages of the disease. The effects of mild to severe localised sarcoptic mange on the health of wombats and therefore the pathogenesis of less severe forms of the disease were also examined. The collection of free-living common wombats with severe sarcoptic mange and the experimental infection of captive
common wombats with *S. scabiei* enabled the examination of the cellular immune response in the dermis of wombats to *S. scabiei* and the study of the population dynamics of *S. scabiei* (Chapters 5 and 6, respectively).

During the course of the above studies several zoonotic infections occurred and opportunistic observations on them were published in the Australian Veterinary Journal (in Appendices). Because of the difficulty in differentiating between varieties of *S. scabiei*, a study examined a specific mitochondrial genomic region in *S. scabiei* to see if *S. scabiei* from different hosts could be readily identified (Chapter 7). Finally, ecological aspects of a free-living common wombat population that occurred at high density were studied to determine the most likely method of transmission of *S. scabiei* between wombats. Armed with the knowledge and experience gained from all of the above studies, *S. scabiei* was introduced into the population to examine the effects of sarcoptic mange on the behaviour of wombats and how this might affect the spread of the disease within the population. This experimental introduction of *S. scabiei* into a wombat population also provided additional information on the likely mode of transmission of *S. scabiei* between wombats and whether control of the spread of the disease is possible (Chapter 8). Discussion of the ways in which the management of sarcoptic mange in wombat populations might be improved are based on the findings of the research undertaken (Chapter 9).

In the text, for the sake of brevity, the variety of *S. scabiei* is only stated when relevant.
Figure 1.1. Scanning electron micrograph of an adult female, *Sarcoptes scabiei var. wombati* (length of body 468 µm). Note the lack of a clearing in the field of dorsal scales.

Figure 1.2. Common wombat (*Vombatus ursinus*) with severe sarcoptic mange above ground at noon in the Wombat State Forest, Bullengaroo, Victoria in 1998.
Figure 1.3. Distribution of extant wombats, the common wombat (CW) (*Vombatus ursinus*), the southern hairy-nosed wombat (SHW) (*Lasiorhinus latifrons*) and the northern hairy-nosed wombat (NHW) (*Lasiorhinus kreffiti*) (Strahan, 1995). Current distribution in black and previous distribution in grey.
CHAPTER 2

CURRENT DISTRIBUTION OF SARCOPTIC MANGE IN WOMBATS

2.1. INTRODUCTION

At the National Conference ‘Wombats in Australia’ (Adelaide, September 1994) delegates from across Australia expressed concern about the prevalence of sarcoptic mange in wild populations of the common wombat. In order to provide some initial information on the present extent of sarcoptic mange in both common and hairy-nosed wombats, a survey, based on a questionnaire, was conducted. The significance of sarcoptic mange in the conservation and management of wombats, particularly the common wombat is discussed.

2.2. MATERIALS AND METHODS

The questionnaire consisted of two parts. The first (Appendix 2.1) aimed to determine the present extent of mange across the range of all three wombat species and the second (Appendix 2.2) invited people to collect data on the prevalence of mange in wombats over a 3 month period (November 95 to January 96). People were also asked to provide any relevant observations not covered by the questionnaire. Both parts were distributed to biologists, rangers, animal carers and naturalists. Respondents were asked to use the 1:100,000 series map sheet names for the name of the locality of their wombats. Each locality (map sheet) represents an area of about
2.3. RESULTS

There were 103 returns to part 1 and 4 returns to part 2 of the questionnaire. The returns contained information from 66 localities.

Ninety-three returns to part 1 and four returns to part 2 provided information from 60 localities for common wombats (see Table 2.1). With the exception of Qld, returns were received from all states in which this species occurs (see Fig. 2.1, Table 2.1). 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 (Skerratt, 1998) were reported from 56 (93.3%) of localities and not observed at 4 (Table 2.1). Mange was observed in both juvenile and adult animals. The mite *S. scabiei* was identified in skin scrapings from common wombats at 31 (51.7%) localities in SA, Vic, NSW, ACT, Tas and Flinders Island (Table 2.1).

Prevalence estimates were obtained at four localities, three in southern Vic and one in northern NSW. The percentage of animals at the Victorian sites showing obvious signs of sarcoptic mange ranged from 0 to 22.2%. At the single NSW site from which a report was received, 15% showed obvious signs of mange (Table 2.2).

Eight returns (Part 1), covering five localities, dealt with southern hairy-nosed wombats. One of these reported clinical signs of mange (see Table 2.3) but the presence of *S. scabiei* was not determined. There were two returns (Part 1) for
northern hairy-nosed wombats from Epping Forest, Qld (the only remaining population of this species). Sarcoptic mange was not observed in this species.

Dogs and foxes were reported to be present at all mainland sites at which sarcoptic mange was observed in wombats.

The following observations and opinions on free-living common wombat populations were also provided by some of the respondents:

• In populations monitored over several years there were always a few common wombats with mange.
• Some common wombats recovered from mange.
• The prevalence of mange varied over time with epizootics occurring sporadically.
• Epizootics of mange correlated with a decline in the local abundance of common wombats.
• Mange was the major cause of mortality in common wombats.
• The prevalence of mange increased during drought or winter, when there was little feed available, or after habitat degradation had occurred.
• Mange was more prevalent when there was a high density of common wombats.
• In an area near Robe, SA where common wombats have been locally extinct for over 20 years, the last common wombat seen by the respondent had mange.
• There has been a long history of mange in some areas while it has only recently arrived in others. For example, mange had not been observed in a common wombat population under close observation for over 20 years in coastal East Gippsland, Vic.
A mange outbreak, which started in 1993, substantially reduced the local abundance of wombats.

- Mange was not observed in common wombats around Narooma, NSW but was frequently observed in foxes and dogs.

2.4. DISCUSSION

Whilst the data from questionnaire surveys must be interpreted cautiously, they provide ready information on topics, such as the distribution of a disease, that would be an enormous task to obtain otherwise.

Common wombats

*Occurrence.* The results of this survey suggest that sarcoptic mange occurs throughout most of the range of the common wombat (Fig. 2.1) and that it is enzootic in some populations. Mange was observed at 93.3% of localities and the mite *S. scabiei* was positively identified from 51.7% of localities from which returns were received. At several localities, a significant proportion of the population (22.2 and 21.7% at two Victorian localities and 15% at one in NSW) had sarcoptic mange. Based on small samples of road-killed animals, the prevalence of sarcoptic mange in common wombats at two locations in Victoria, Moe (1982-1984) and Healesville (1992) was estimated at 14% (n = 7) and 5% (n = 22) respectively (Smales, 1987; Skerratt, 1998). A similarly high prevalence of sarcoptic mange has been reported in human populations in several countries, in red foxes (*Vulpes vulpes* = *V. fulva*) in central New York, in red foxes (*Vulpes vulpes*) in Sweden, and in coyotes (*Canis latrans*) in Texas (Fain, 1978; Tullar and Berchielle, 1981; Lindström and Mörner,
While the evidence is far from complete, the observations of a number of respondents suggested that epizootics of sarcoptic mange occur sporadically in common wombat populations. A detailed study of sarcoptic mange in coyote populations in southern Texas found that mange in coyotes was enzootic for a 45 year period between two epizootics, one which ended in 1930 and the other which started in 1975 (Pence and Windberg, 1994). The prevalence of sarcoptic mange during these epizootics differed between adjacent populations of similar density, age distribution and social composition. This was attributed to differences in resistance to sarcoptic mange between the study populations. The apparent pattern of epizootics in wombat populations could similarly be caused by differences in resistance.

The suggestions by a number of respondents that the prevalence of mange in common wombats increased at higher population densities and in times of malnutrition such as drought or during winter, although subjective, are also supported by studies on other mammals. Sarcoptic mange was found to be more prevalent in denser populations of coyotes (Pence and Windberg, 1994) and severe sarcoptic mange is frequently associated with malnutrition in winter in temperate climates or in the dry season in tropical climates in a variety of species (Sweatman, 1971). However, researchers studying water buffaloes, pigs and wild ruminants suggest that the increased longevity of *S. scabiei* off the host in winter may enhance its transmission and could account for the greater prevalence of mange during winter (Davies et al., 1991; Hayat et al., 1996; Yeruham et al., 1996).
Effects of mange. As with coyotes, common wombats were observed by respondents to either recover or die from sarcoptic mange (Pence and Windberg, 1994). Respondents also observed that epizootics of sarcoptic mange reduced the abundance of wombats. Similar findings were reported in NSW in wombats (Gray, 1937) and in foxes in Australia, Finland and Sweden (Ratcliffe, 1956; Mörner, 1984; Mörner and Christensson, 1984; Danell and Hörfeldt, 1987; Lindström, 1991; Lindström et al., 1994). This is a significant observation as there are now many small populations of common wombats confined to remnant habitat, particularly at the extremes of its range (Fig. 2.1). The fate of a small population may be governed by the specific fortunes of each of its few individuals (Caughley, 1994). Mange may therefore pose a threat to some wombat populations simply because they are small.

There were a number of local extinctions of remnant populations of common wombats in the south-east of South Australia in the early 1900s (Cooke, 1998). These have been attributed to a failure of recruitment brought about by malnutrition resulting from a shortage of preferred perennial grasses, this shortage being primarily caused by drought but exacerbated by competition from rabbits (Mallet and Cooke, 1986; Cooke, 1998). The potential role of disease in these extinctions was not considered and our survey suggests that sarcoptic mange can be prevalent in common wombats during periods of nutritional stress such as would occur during drought. It may be significant that one respondent reported mange in the last wombat seen in an area where a local extinction subsequently occurred (near Robe, SA).

The role of canids in the epizootiology of sarcoptic mange in wombats is unknown, but the absence of foxes from Tasmania and Flinders Island (Coman, 1995)
indicates that foxes do not need to be present for sarcoptic mange to be prevalent in free-living wombats. In addition, as was reported by one respondent from Narooma, NSW, mange does not necessarily occur in wombats when the disease is prevalent in foxes and dogs in the same area. Dogs have been reported to develop *Sarcoptes* infestations after hunting wombats (Gray, 1937), and foxes occasionally utilise wombat burrows where conditions of high humidity and low, relatively stable temperature (Brown, 1964; Triggs, 1996) appear ideal for the survival and transmission of mites (Gerasimov, 1953; Arlian, 1989).

Alopecia in hairy-nosed wombats

The clinical signs of severe sarcoptic mange seen in common wombats were not reported for hairy-nosed wombats in this survey. Similarly, the literature on sarcoptic mange in wombats indicates that common wombats rather than hairy-nosed wombats are mainly affected by sarcoptic mange (Gray, 1937; Wells, 1971; Doube, 1982; Skerratt et al., 1998). Respondents who had examined both northern and southern hairy-nosed wombats did report a flaky skin condition involving some alopecia but the condition was only seasonally apparent with individuals monitored over several years recovering fully from it. The condition may result from infestation with a less pathogenic ectoparasite but awaits confirmation.

In conclusion, sarcoptic mange was not reported in hairy-nosed wombats, however, it occurred in common wombat populations throughout their range including smaller isolated populations in eastern South Australia, western Victoria, northern New South Wales and Flinders Island.
Table 2.1. Number of localities at which clinical signs of mange were observed and *Sarcoptes scabiei* var. *wombati* was present on common wombats.

<table>
<thead>
<tr>
<th>State or territory</th>
<th>Number of returns to questionnaire part 1</th>
<th>Number of localities(^a) at which wombats observed</th>
<th>Number of localities at which clinical signs of mange observed</th>
<th>Number of localities at which <em>S. scabiei</em> was present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tasmania &amp; Flinders Is.</td>
<td>6</td>
<td>4</td>
<td>3 (75%)</td>
<td>3 (75%)</td>
</tr>
<tr>
<td>Victoria</td>
<td>38</td>
<td>21</td>
<td>21 (100%)</td>
<td>12 (57.1%)</td>
</tr>
<tr>
<td>South Australia</td>
<td>8</td>
<td>6</td>
<td>5 (83.3%)</td>
<td>4 (66.7%)</td>
</tr>
<tr>
<td>Australian Capital Territory</td>
<td>3</td>
<td>1</td>
<td>1 (100%)</td>
<td>1 (100%)</td>
</tr>
<tr>
<td>New South Wales</td>
<td>38</td>
<td>28</td>
<td>26 (92.9%)</td>
<td>11 (39.3%)</td>
</tr>
<tr>
<td>Total</td>
<td>93</td>
<td>60</td>
<td>56 (93.3%)</td>
<td>31 (51.7%)</td>
</tr>
</tbody>
</table>

\(^a\)A locality is defined as the area encompassed by the appropriate map sheet from the 1:100,000 series map. See Appendix 2.3 for a complete list of localities for each state.
Table 2.2. Prevalence of clinical signs of mange and presence of *Sarcoptes scabiei* var. *wombati* on common wombats during a three month period (November 1995 to January 1996).

<table>
<thead>
<tr>
<th>Locality</th>
<th>Number of wombats observed</th>
<th>Number of wombats with clinical signs of mange</th>
<th><em>S. scabiei</em> present on wombats with mange</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foster, Vic</td>
<td>23</td>
<td>5 (21.7%)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>+</td>
</tr>
<tr>
<td>Warrigal, Vic</td>
<td>16</td>
<td>0 (0%)</td>
<td>ND</td>
</tr>
<tr>
<td>Woodend, Vic</td>
<td>9</td>
<td>2 (22.2%)</td>
<td>+</td>
</tr>
<tr>
<td>Yarrowitch, NSW</td>
<td>NS</td>
<td>About 15%&lt;sup&gt;b&lt;/sup&gt;</td>
<td>ND</td>
</tr>
</tbody>
</table>

<sup>a</sup>Locality names are the map sheet names from the 1:100,000 map series.

ND = Not determined.

NS = Numbers not supplied by respondent.

<sup>b</sup>Prevalence estimate supplied by respondent.
Table 2.3. Presence of clinical signs of mange in southern hairy-nosed wombats at various localities in South Australia.

<table>
<thead>
<tr>
<th>Locality</th>
<th>Number of returns</th>
<th>Wombats with clinical signs of mange&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eudunda</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Lincoln</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Mannum</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Streaky Bay</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Swan Reach</td>
<td>4</td>
<td>+</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>8</strong></td>
<td><strong>1</strong></td>
</tr>
</tbody>
</table>

<sup>a</sup>Locality names are the map sheet names from the 1:100,000 map series.

<sup>b</sup>Presence of *S. scabiei* on wombats not determined.
Figure 2.1. Occurrence of mange in relation to the current distribution of the common wombat (*Vombatus ursinus*).

Black triangles depict locations at which mange was reported. The stippled area indicates the current range of the common wombat according to Triggs (1996).
Figure 2.2. Occurrence of mange in relation to the current distribution of the southern hairy-nosed wombat (*Lasiorhinus latifrons*).

Black triangles depict locations at which mange was reported. The stippled area indicates the current range of the southern hairy-nosed wombat according to Wells (1995).
CHAPTER 3

DISTRIBUTION OF LIFE CYCLE STAGES OF \textit{Sarcoptes scabiei} var. \textit{Wombati} AND CLINICOPATHOLOGICAL EFFECTS OF SEVERE MANGE ON COMMON WOMBATS (\textit{Vombatus ursinus}) IN VICTORIA

3.1. INTRODUCTION

The clinical, pathological and histopathological changes induced by severe sarcoptic mange have been investigated in coyotes (\textit{Canis latrans}), rabbits (\textit{Oryctolagus cuniculus}), domestic dogs, and foxes (\textit{Vulpes vulpes}) (Pence et al., 1983; Arlian et al., 1988a; Arlian et al., 1990). The distribution and number of some life cycle stages of \textit{Sarcoptes scabiei} have been determined in pigs and humans with slight to moderate sarcoptic mange (Johnson and Mellanby, 1942; Bartley and Mellanby, 1944; Davis and Moon, 1990a) but there have been no studies on distribution and number of \textit{S. scabiei} on hosts with severe parakeratotic sarcoptic mange, nor have mite numbers been related to clinical signs (Arlian, 1989; Burgess, 1994).

The pathogenesis of severe sarcoptic mange in common wombats (\textit{Vombatus ursinus}) is not well understood despite being widespread in wombat populations and considered to affect the abundance of wombats (Martin et al., 1998; Skerratt et al., 1998; Chapters 1 and 2). The histopathological changes in the skin and internal organs of common wombats with severe parakeratotic sarcoptic mange have been described
(Skerratt, 1998). However, the clinical pathological changes have not. Here, the systemic effects of severe parakeratotic sarcoptic mange on common wombats have been investigated, including clinical pathology, the distribution and number of each life cycle stage of *S. scabiei var. wombati* and how mite numbers relate to clinical signs of mange.

3.2. MATERIALS AND METHODS

Ten wombats with severe parakeratotic sarcoptic mange were collected from forested areas of the state of Victoria, Australia, over 10 consecutive months from 10 April 1997 to 22 February 1998. Seven female and three male common wombats, six of them adults and four subadults, were collected from Bullengarook (37°31’ S, 144°29’ E), Eden Park (37°30’ S, 145°04’ E), Christmas Hills (37°39’ S, 145°19’ E), Chum Creek (37°30’ S, 145°29’ E), Toolangi (37°32’ S, 145°29’ E) and Longwarry North (38°05’ S, 145°47’ E) in Victoria. Wombats were found grazing during the day and could be readily approached. They were either caught by hand or tranquillised with a blowdart containing tiletamine and zolazepam (3 mg/kg Zoletil; Virbac, Peakhurst, New South Wales, Australia). Two wombats were moribund and died soon after capture and the remaining eight were euthanased with pentobarbitone injected into the abdomen (325 mg/kg Lethabarb; Virbac). Blood was collected by cardiac puncture at the time of euthanasia for haematology and serum biochemistry and clotted blood was collected for serum biochemistry from wombats that died soon after capture. It is possible that some of these wombats were dehydrated when blood was collected and their hydration status affected haematological and biochemical values. The weight of the wombat and the dorsal length from the upper lip to the end of the
tail were measured. The sex, maturity and body condition based on subcutaneous fat deposits, muscle mass, and the prominence of bones were recorded. Both lateral aspects of the wombat were photographed and the degree of crusting and alopecia and whether fissuring occurred were visually assessed at 35 sites on the skin (Fig. 3.1). The degree of scale crust thickness and alopecia at a site were assigned to one of four categories. For scale crust thickness these were: (1) no scale, (2) some thin scale, (3) thin crust (1-5mm thick) and (4) thick crust (>5mm thick). For degree of alopecia these were: (1) no hair loss, (2) slight hair loss < 25%, (3) moderate hair loss 25-75% and (4) severe hair loss >75%.

The entire skin of each wombat was removed and a 7 mm diameter skin biopsy punch attached to a drill was used to remove cores of skin from the 35 sites. The inner and outer surfaces of the pinnae were also sampled. The specimens were macerated in 10% potassium hydroxide in test tubes placed in boiling water until the skin had dissolved. To determine the number of each life cycle stage of *S. scabiei var. wombati* the solution was made up to 10 ml with water and an aliquot of between 1-5 ml, depending on the density of mites and the solution’s opacity, was examined under a stereomicroscope (Clark et al., 1971). If the density of mites was very high then a 1 ml aliquot was added to 9 ml of water and an aliquot of between 1-5 ml of this solution was examined depending on the density of mites. Life cycle stages of *S. scabiei var. wombati* were identified according to Fain (1968). Males, larvae, eggs, and egg shells could be easily identified based on their morphology. Females were differentiated from other life cycle stages based on their larger size (length > 350 µm), measured with an eyepiece micrometer in a stereomicroscope and occasionally on the presence of an egg in utero. Female mites were examined with a compound
microscope to verify their identity as *S. scabiei var. wombatii* (Fain, 1968).

Tritonymphs could not be differentiated from protonymphs under the stereomicroscope and all were simply classed as nymphs. Ecdyses were not counted. The distribution of life cycle stages in 10 ml of potassium hydroxide solution was assessed for normality by dividing the solution into ten 1 ml aliquots. A one-sample Kolmogorov-Smirnov exact test was performed with the null hypothesis being that the differences between the observed frequencies and the mean were normally distributed (Sokal and Rohlf, 1997). The distribution of mites in a region of skin with similar clinical signs was assessed for normality by taking five biopsies. A one-sample Kolmogorov-Smirnov exact test was again performed with the null hypothesis being that the differences between the observed frequencies and the mean were normally distributed (Sokal and Rohlf, 1997). Differences between sites were assessed for significance using a randomised block design with the blocking variable being wombats and the treatment variable being sites (Sokal and Rohlf, 1997).

Biopsies were fixed in Bouins fixative from 20 of the above 35 sites to measure the thickness of crust and skin. Thicknesses were compared for significant differences using Wilcoxon’s matched-pairs signed-ranks test (Sokal and Rohlf, 1997). Correlations between clinical signs and mite numbers were performed using either Spearman’s coefficient of rank correlation or Pearson’s coefficient of product moment correlation (Sokal and Rohlf, 1997).

Wombats were necropsied and samples of liver, lung, kidney, myocardium, brain, adrenal gland, thyroid gland, prescapular lymph node, mesenteric lymph node, spleen, stomach, duodenum, jejunum, ileum, colon, uterus, and gonads were fixed in
10% formalin and processed for histological examination. Culturing and identification of bacteria was attempted from internal tissues suspected of being infected. The colonic contents were examined for nematodes and ticks were removed from skin for identification. The proportion of white pulp in histological sections of spleens of wombats with sarcoptic mange were measured using a Sigma Scan Scientific Measurement System 1.10 (Jandel Scientific, Corte Madera, California, USA) and compared with proportions from three normal wombats using the Student’s two sample t-test (two tailed) assuming equal variances (Sokal and Rohlf, 1997).

Haematological parameters were measured using a Coulter Counter, Model S-Plus 4, impedance haematology analyser, calibrated with human blood (Coulter Electronics, Hialeah, Florida, USA), and by examination with a compound microscope of a blood smear stained by the May-Gruenwald-Giemsa method (Strumia, 1963). Electrolyte concentrations were determined by the ion-specific potentiometry method (Baker and Silverton, 1985) using a Vet Lyte electrolyte analyser (Idexx Laboratories, Zetland, NSW, Australia). Calcium, phosphate, urea, creatinine, glutamate dehydrogenase, alkaline phosphatase, aspartate aminotransferase and creatine kinase concentrations were determined colorimetrically with a Cobas Mira spectrophotometer (Roche Diagnostics, Branchburg, New Jersey, USA) using reagents and methods from Trace Scientific (Baulkham Hills, New South Wales, Australia), and albumin and total protein concentrations were determined colorimetrically using methods and reagents from Randox Laboratories (Crumlin, Antrim County, UK). Values from five captive wombats collected from Glenburn (37°26′ S, 145°25′ E) (Victoria, Australia) and held in captivity for two months and those reported by Presidente (1982) for captive wombats from Boolarra (38°23′ S, 146°25′ E) (Victoria, Australia) were compared using the Student’s t-test (two tailed) assuming equal variances (Sokal and Rohlf, 1997).
146°17′ E) (Victoria, Australia) were used as a comparison of clinically healthy wombats. Differences were assessed for significance using the Student’s two sample t-test (two tailed) assuming either equal or unequal variances (Sokal and Rohlf, 1997). Electrophoretograms were carried out on Gelman super sepraphore mylar supported cellulose acetate strips in a Gelman semi-micro electrophoresis chamber. The strips were stained with Ponceau S and scanned with a Gelman ACD-18 densitometer (Gelman Sciences, Ann Arbor, Michigan, USA). All statistical calculations were performed by hand calculator or SPSS® for Windows 8.0 (SPSS Inc., Chicago, Illinois, USA). Differences were regarded as significant when $P < 0.05$.

3.3. RESULTS

Subcutaneous fat was not seen in any of the wombats and all were in poor body condition. The ranges and means ± standard errors of the weights and lengths were 10-28 (21 ± 2) kg and 76-114 (100 ± 3) cm, respectively. All wombats had at least 30% of the skin surface affected with severe parakeratotic sarcoptic mange.

There was no evidence that life-cycle stages were not normally distributed in the potassium hydroxide solution ($P$ exact two-tailed = 0.826). There was no evidence that mites were not distributed normally in a region of skin with similar clinical signs ($P$ exact two-tailed = 0.949). When considering the mean or median number of each life cycle stage of *S. scabiei* for the 10 wombats, larvae were the most prevalent life-cycle stage followed by eggs, nymphs, females, and males (Table 3.1). The anterolateral part of the body was most heavily parasitised followed by the posterolateral, the dorsal region between the ears, the ears, ventral abdomen, medial
aspect of the legs, axillary and inguinal areas, and the dorsal midline (Fig. 3.1, Table 3.1). More than 1,000 mites per cm² were found at all sites on the lateral sides of the body except those near the rump, which had fewer than 400 mites per cm². The ears had 1,000 mites per cm² on each side of the pinna. Sites along the dorsal midline varied greatly in intensity of infection. The site between the ears was heavily parasitised (2,140 per cm²) whereas the site on the rump was the least infested site on the body (60 mites per cm²). Mite numbers were just below 1,000 per cm² on the ventral abdomen. Mite numbers were low, below 400 per cm², on the medial aspect of legs and in the axillary and inguinal sites. The site on the dorsal midline between the ears and those on the ears and lateral surfaces, apart from those high on the hindlimb near the rump, were 5 to 100 times more heavily infested than sites on ventral surfaces and the dorsal midline ($P < 0.05$). Mite numbers at any site did not differ significantly from those at the symmetrical site on the other side of the body ($P > 0.05$).

Scale crust thickness and severity of alopecia were correlated with mite numbers (Table 3.2). The correlation coefficients ($r$) for qualitative crust thickness, qualitative alopecia, and quantitative crust thickness versus numbers of mites were 0.69, 0.71 and 0.61, respectively ($P < 0.001$). Crust thickness was also highly correlated with severity of alopecia, $r = 0.75$ ($P < 0.001$) (Table 3.2). Fissuring in crust and skin occurred only in approximately half the wombats when scale crust was present (Table 3.2). There were no significant differences in scale crust thickness and degree of alopecia between sites and their symmetrical sites on the other side of the body ($P > 0.05$). Hence, the amount of scale crust and the degree of alopecia on each
side of the body were symmetrical. Crust thickness was not correlated with skin thickness ($P = 0.247$).

Three wombats with low albumin concentrations had ascites. Another wombat had more than 10 ml of pink fluid in its left pleural cavity, a roughened left pleural surface and left thoracic wall and adhesions between the pleura and thoracic wall. The mitral valve had irregular soft red material attached to its caudal margin. A light growth of gram negative organisms was obtained from fluid in the pleural cavity, although few leucocytes and no bacteria were seen in a smear of the fluid. Moderate, multifocal, mononuclear inflammation occurred in the myocardium and mild, interstitial, mixed inflammatory infiltrates were seen in the renal cortices. The right lung and the extremities of the left lung of one wombat were dark red and firm and oozed blood when cut. This wombat and another had focal areas of mixed, but predominantly mononuclear, inflammatory infiltrate within lungs. One wombat had abscesses containing gram negative coccoid bacteria in submandibular lymph nodes and fibrous lesions with mononuclear inflammation in the kidney consistent with chronic infarction. Another wombat had an abscess (5 mm diameter) containing gram negative cocci in a prescapular lymph node and a small, multifocal, mixed, but predominantly mononuclear inflammatory infiltrate, in the myocardium. One wombat had many small foci of mononuclear inflammation in the myocardium. One wombat had a cataract in the right eye and the mucosa in the first 10 cm of the colon was thickened with gas and congested. Two wombats had focal, endogenous, lipid pneumonia at the periphery of lobes.
Seven wombats had vacuolation of hepatocytes consistent with fatty change of the liver. Eight wombats had multifocal areas of inflammation and necrosis of the liver, consistent with bacteria entering the body, possibly from breaks in the integrity of the skin due to mange, and causing reactive hepatitis. Testes contained little or no sperm and ovaries had few or no follicles. One female wombat had old corpora lutea only. Prescapular lymph nodes, mesenteric lymph nodes and spleens from wombats with mange \((n = 10)\) were similar to those from free living wombats without mange \((n = 3)\) in terms of the frequency, size and cellular density of germinal centres and the density of parafollicular tissue. There was no significant difference in the proportion of white pulp in histological sections of spleens of wombats with mange compared with normal wombats, 12.2 % and 11.5 %, respectively \((P = 0.845)\). There appeared to be more nuclear debris in germinal centres of the prescapular lymph nodes from wombats with mange, consistent with lysis of lymphocytes and compatible with an effect of stress hormones (glucocorticoids). The histopathological changes present in the skin were similar to that described by Skerratt (1998) for wombats with parakeratotic sarcoptic mange. Histopathological lesions were not seen in the gastrointestinal tract, brain, spleen, thyroid, reproductive tract and gonads of wombats.

The mean corpuscular volume, mean corpuscular haemoglobin and concentrations of lymphocytes, calcium, creatinine, total solids, total protein and albumin, determined electrophoretically and by the bromocresol method, of wombats with mange were significantly lower and concentrations of neutrophils, monocytes, phosphorus, urea, glutamate dehydrogenase, aspartate aminotransferase and creatine kinase were significantly higher than those of captive wombats without mange in the
present study \((P < 0.05)\) (Tables 3.3, 3.4). Haematocrit and concentrations of haemoglobin, erythrocytes, mean corpuscular haemoglobin, leucocytes, band neutrophils, eosinophils, nucleated erythrocytes, sodium, potassium, chloride, glucose, total bilirubin, alkaline phosphatase, gamma glutamyltransferase, and electrophoretically determined alpha 1 and 2, beta and gamma globulins of wombats with mange were not significantly different from those of captive wombats in the present study \((P > 0.05)\) (Tables 3.3, 3.4). Corpuscular haemoglobin, haematocrit and concentrations of haemoglobin, lymphocytes, calcium, glucose, total protein and electrophoretically determined albumin and globulins of wombats with mange were significantly lower and concentrations of neutrophils, phosphorus and aspartate aminotransferase were significantly higher than those reported by Presidente (1982) \((P < 0.05)\) (Tables 3.3, 3.4). Mean corpuscular volume and concentrations of erythrocytes, mean corpuscular haemoglobin, leucocytes, monocytes, eosinophils, sodium, potassium and chloride were not significantly different from those reported by Presidente (1982) \((P > 0.05)\) (Tables 3.3, 3.4).

Seven of eight wombats had some haematological values outside the range of values reported by Presidente (1982). Two had lower concentrations of haemoglobin, two had lower haematocrits, three had lower mean red blood cell volumes with one also having a lower level of mean red blood cell haemoglobin and one had a lower mean red blood cell haemoglobin concentration. Two wombats had 1 to 3 nucleated red blood cells per 100 white blood cells. Three wombats had slight anisocytosis of red blood cells, three had polychromasia of a small to moderate number of red blood cells, one had hypochromasia of red blood cells and one had stomatocytes. Six of eight wombats had blood leucocyte concentrations outside the range reported by Presidente
Two wombats, one with pneumonia and another with abscesses in submandibular lymph nodes, had a neutrophilia and one also had a lymphopenia and the other had activated lymphocytes (increased amounts of basophilic intensity of cytoplasm). One wombat had a monocytosis, increased concentrations of band neutrophils and activated lymphocytes. This wombat had a small abscess in its axillary lymph node. Two wombats had either activated or very large lymphocytes; one of these had pneumonia and the other had endogenous lipid pneumonia and some renal tubular necrosis. One wombat had slight basophilic granulation of the cytoplasm of neutrophils indicating “toxic” change. This wombat had bacterial infection of the pleural cavity and inflammation of myocardium and kidneys. Three of six wombats with abnormal blood leucocyte values had bacterial infections in internal organs and all six had inflammation in organs other than skin. The two wombats with normal blood leucocyte values had no visible lesions other than sarcoptic mange.

All 10 wombats had some biochemical values outside the ranges reported by Presidente (1982) (Table 3.5). One wombat had an elevated sodium concentration, four wombats had higher potassium concentrations, four wombats had lower potassium concentrations, three wombats had lower calcium concentrations, four wombats had higher phosphate concentrations, nine wombats had higher concentrations of urea, two wombats had lower concentrations of glucose, one wombat had a higher concentration of glucose, eight wombats had higher concentrations of glutamate dehydrogenase, eight wombats had higher concentrations of aspartate aminotransferase, five wombats had lower concentrations of total protein, and three wombats had lower concentrations of albumin. On electrophoresis, five and three of ten wombats had lower concentrations of albumin and globulins, respectively,

Seven of the 10 wombats had between one and 20 ticks attached to ventral areas unaffected by severe mange. These were *Aponomma auruginans*, *Ixodes tasmani*, and *I. cornuatus*. *Strongyloides spearei* was seen in histological sections of the duodenum and jejunum of six wombats. *Eimeria arundeli* was seen in histological sections of the jejunum and ileum from five wombats. Ciliates were seen in histological sections of the colon from eight wombats. Seven of the ten wombats had nematodes, *Phascolostrongylus turleyi* and *Oesophagostomoides* spp. (Strongylidae) in their colon. A minimal to moderate mixed inflammatory infiltrate was present in the small intestines and colons of wombats. No parasites were seen in stomachs nor was there evidence of infection with *Fasciola hepatica* in livers.

3.4. DISCUSSION

Wombats with severe sarcoptic mange, which were found grazing during the day, were used in this study. Wombats are generally nocturnal hence our selection of mange affected wombats was biased towards animals where the severity of mange led to abnormal behaviour. Despite this biased selection of wombats, they provided valuable information.

*Stages*
The ratio of female to male *S. scabiei* var. *wombati* (2 : 1) suggests that males may mate with several females or that females live longer than males. Heilesen (1946) found that *S. scabiei* males seek females and Arlian et al. (1984a; 1989) found that females survive longer than males off the host. The ratio of larvae and eggs to females (11 : 6 : 2) is lower than expected since *S. scabiei* var. *hominis* females lay 2 to 4 eggs per day and *S. scabiei* var. *canis* larvae and eggs last for 3 to 4 and 2 to 3 days, respectively (Van Neste and Lachapelle, 1981; Arlian and Vyszenski-Moher, 1988). The ratio of nymphs to females (3 : 2) is much lower than expected given that nymphs survive for 5 to 7 days according to Arlian and Vyszenski-Moher (1988). This suggests that there is either a high mortality of nymphs and moulting larvae or that nymphs leave the host in search of new hosts. Alternatively, the nymphs of *S. scabiei* var. *wombati* could have a shorter life span than other varieties of *S. scabiei*. Misclassification of nymphs as females whilst counting was unlikely to account for such a large discrepancy between the expected ratio of nymphs to females and the ratio reported here, given the life cycle of the mite.

**Symmetry**

The consistent difference and symmetry in the degree of parasitism between sites on the body suggests that there is site predilection for the mite, namely the anterolateral surfaces of the wombat’s body. Site predilection has been reported for *S. scabiei* in pigs and humans (Davis and Moon, 1990a; Johnson and Mellanby, 1942; Bartley and Mellanby, 1944). Davis and Moon (1990a) suggest that the faces and ears of pigs are more readily infected because pigs explore with their noses. They also suggest that the face and ears provide a more favourable environment because the creases in skin and coarse hair may prevent removal of mites by abrasion. In humans,
the hands and feet are more frequently infected than other parts of the body (Johnson and Mellanby, 1942; Bartley and Mellanby, 1944). Johnson and Mellanby (1942) stated that the reaction to mites in these areas was comparatively slight. The factors affecting *S. scabiei* site predilection in wombats were not apparent. Skin thickness was not inversely correlated with parasitism although the thickest skin, the rump, had the lowest intensity. Severe parakeratotic *S. scabiei* infection should always be visible, even from a distance of a hundred metres given that clinical signs of mange, namely thickness of scale crust and severity of alopecia, were highly correlated with each other and with the number of *S. scabiei*. Skerratt (1998) found that hair follicles were atrophic in common wombats with severe parakeratotic sarcoptic mange suggesting that the thick scale crust prevents hair regrowth. A view of one side of a wombat is sufficient to assess the severity of mange since clinical signs of severe sarcoptic mange were symmetrical. These findings are relevant to the interpretation of observations made in the field on wild wombats.

*Other infections*

In contrast to this study, Skerratt (1998) did not report bacterial infections in nine common wombats with severe parakeratotic sarcoptic mange from the shire of Healesville in 1992 although one had *Pneumocystis carinii* infection concurrent with lymphosarcoma. It is probable that bacterial infections in the dermis, which occur following fissuring associated with scale crust, allow bacteria to infect internal organs. Secondary bacterial infections have been reported in rabbits and humans with scabies (Arlian et al., 1990; Burgess, 1994). While the immune response in the dermis towards severe *S. scabiei* infection in wombats is slight (Skerratt, 1998), there was no
evidence of lymphoid depletion in lymph nodes or spleens in the wombats studied here. Lymphoid depletion has been reported in several species of marsupials, occurring seasonally in rainforest possums (Speare et al., 1984), or following breeding in male antechinus (Barker et al., 1978) and in possums, in poor condition, held in captivity (Presidente and Correa, 1981). The greater quantity of nuclear debris in the prescapular lymph nodes is probably due to stress hormones (glucocorticoids) causing lysis of lymphocytes or possibly a consequence of the immune response in the skin.

Several pathological changes suggest that parasitised wombats were utilising energy stores to meet the imbalance between the energy requirements associated with severe parakeratotic sarcoptic mange and their nutritional supply. Parasitised wombats were in poor body condition, lacked subcutaneous fat, had low concentrations of protein, albumin and globulins in blood and had low concentrations of creatinine indicating a low muscle mass (Duncan et al., 1994). The high concentrations of urea in blood suggest increased protein catabolism and fatty change in the liver is consistent with wombats mobilising their peripheral fat stores but lacking sufficient protein synthesis to generate lipoprotein (Jubb et al., 1993). The wombats with severe parakeratotic sarcoptic mange reported by Skerratt (1998) were generally emaciated. Similar changes were seen in coyotes and rabbits with severe parakeratotic sarcoptic mange, >50% and 10 to 30% of body affected, respectively, except that rabbits had low blood urea nitrogen and high total serum protein while coyotes had normal total serum protein concentrations (Pence et al., 1983; Arlian et al., 1988a). The absence of these changes in dogs and foxes with short term advanced sarcoptic mange is attributed to the brief duration of the mange (Arlian et al., 1995, Little, 1998)(Arlian et al., 1995). The low concentrations of blood glucose in wombats have also been seen

Arlian et al. (1988a) demonstrated that severe mange can increase the energy demands of rabbits leading to weight loss but did not attribute these energy demands to the consumption of body tissue by mites. However, the energy demands of scratching, scale crust production and exudation from fissures in skin may be large. Wombats are normally nocturnal, but those with severe mange are diurnal (Skerratt, 1998) possibly because of energy demands or for thermoregulatory reasons. They may also be unable to compete with healthy wombats for resources at night. Presidente (1982) and Doube (1982) suggested that wombats with severe sarcoptic mange may have impaired powers of mastication and may starve. Wombats with severe sarcoptic mange may eat less.

Wombats with severe mange when grazing, take little heed of their surroundings, and therefore can be more readily approached (unpublished observations). Alados et al. (1996) found that the complexity of exploratory behaviour was reduced in Spanish ibex (Capra pyrenaica) with S. scabiei infection. Vision and hearing may also be reduced in wombats with severe sarcoptic mange (Skerratt 1998). Gonads of mature wombats were not active or had minimal activity suggesting that wombats with severe mange are unlikely to reproduce. It is unlikely that wombats were collected exclusively during the non-breeding season given its long duration (McIlroy, 1973) and the 10 month period of collection of wombats.
Wombats with severe sarcoptic mange had a lower haematocrit, mean corpuscular volume, mean corpuscular haemoglobin and concentrations of haemoglobin than captive wombats. This may have been due to sarcoptic mange, as rabbits and dogs with severe and short term advanced sarcoptic mange respectively have developed anaemia, and chronic disease can cause anaemia (Arlian et al., 1988a, 1995; Duncan et al., 1994). However, the low mean corpuscular volume in some wombats suggests that the anaemia may have been due to dietary insufficiency (Duncan et al., 1994). Arlian et al. (1988a) suggested that iron deficiency may be involved in some way. The presence of nucleated and polychromatic erythrocytes in the blood of a few wombats indicates that a regenerative response was possibly occurring in bone marrow (Duncan et al., 1994).

The immune system of wombats with severe mange was responding to either the bacterial infections in internal organs and skin or the *S. scabiei* infestation given their higher concentration of neutrophils and monocytes, the presence of band neutrophils, transformed lymphocytes and neutrophils, and the lower concentration of lymphocytes in the blood compared with captive wombats (Skerratt, 1998; Duncan et al., 1994). Endogenous corticosteroid production and release by adrenals could also be contributing to these changes in the blood (Duncan et al., 1994). Similar leucocyte changes occurred in rabbits and dogs with severe and short term advanced sarcoptic mange but not in coyotes with severe mange (Arlian et al., 1988a, 1995; Pence et al., 1983). Foxes with short term, severe parakeratotic sarcoptic mange had a neutrophilia and eosinophilia (Little et al., 1998). Total globulins were low in wombats with severe mange although concentrations of each type of globulin, alpha 1 and 2, beta and
gamma, were normal. Total globulins were high in rabbits and normal in coyotes, dogs and foxes with mange (Arlian et al., 1988a, 1995; Pence et al., 1983). Although globulin levels remained normal in coyotes, gamma globulins increased with severity of mange and alpha globulins decreased (Pence et al., 1983).

The hyperkalaemia in some wombats was most likely an artefact due to the delay between sample collection and measuring potassium concentrations as prolonged contact between serum and the blood clot can lead to mildly elevated potassium concentrations (Duncan et al., 1994). The hypokalaemia in other wombats may have been due to anorexia (Duncan et al., 1994). Potassium concentrations did not deviate from normal in rabbits, dogs and foxes with sarcoptic mange (Arlian et al., 1988a, 1995; Little et al., 1998). The low calcium concentrations were sometimes associated with low albumin and protein concentrations and were probably partly due to less protein bound calcium (Duncan et al., 1994). Similar low calcium concentrations were reported in rabbits and coyotes with severe sarcoptic mange but not in dogs or foxes with short term advanced sarcoptic mange (Pence et al., 1983; Arlian et al., 1988a, 1995; Little et al., 1998). Hypocalcaemia was also often associated with hyperphosphataemia and may be partially due to hypoparathyroidism (Duncan et al., 1994). The higher concentrations of glutamate dehydrogenase were probably not due to the reactive hepatitis or fatty change seen in livers since the relative risk of elevated concentrations due to reactive hepatitis and fatty change or both were 0.75, 0.71 and 0.60, respectively (Table 3.5). Elevated concentrations of liver enzymes suggestive of liver damage occurred in rabbits with severe parakeratotic sarcoptic mange but not in dogs or foxes with short term advanced sarcoptic mange (Arlian et al., 1988a, 1995; Little et al., 1998). Arlian et al. (1990) found that rabbits
with severe parakeratotic sarcoptic mange had amyloidosis, hepatocellular swelling and necrosis, and cholangitis of livers. Elevated lactate dehydrogenase concentrations in coyotes were attributed to skin and not liver damage (Pence et al., 1983). The high concentrations of aspartate aminotransferase in the blood of wombats may have been due to liver damage but also may have been associated with muscle damage indicated by the high creatine kinase concentrations (Duncan et al., 1994). Since healthy wombats which were captured and restrained had high concentrations of aspartate aminotransferase and creatine kinase (unpublished observations), the muscle damage may have been due to the capture, restraint and transport of wombats. Erythrocyte, mean corpuscular haemoglobin, sodium and alkaline phosphatase concentrations were similar in wombats whether or not they had sarcoptic mange, as is the case in rabbits, dogs and foxes (Arlian et al., 1988a, 1995; Little et al., 1998). Wombats with severe parakeratotic sarcoptic mange, reported by Skerratt (1998), had similar internal and external parasites to those reported here, although prevalences and intensities of infection differed. Skerratt (1998) found that the number of species of internal parasites and intensity of infection did not differ between wombats with severe sarcoptic mange and healthy wombats. However, he did find that wombats with mange had fewer species of ectoparasites than healthy wombats.

In summary, *S. scabiei* is more abundant in anterolateral areas of the wombat’s skin, numbers are highly correlated with thickness of scale crust and degree of alopecia, cause emaciation and haematological and biochemical changes consistent with starvation and anaemia when numerous, predispose wombats to bacterial infection and result in a leucogram indicative of inflammation.
Table 3.1. Mean numbers of *Sarcoptes scabiei* var. *wombati* per square centimetre at 35 sites on 10 wombats with severe parakeratotic sarcoptic mange from Victoria, Australia.

(See Fig. 3.1 for position of site on body)

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<th>Larvae</th>
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*a* Mean numbers at sites 3 and 6 are the number of mites on the inner and outer surfaces of the pinnae.
Table 3.2. Percentage of wombats (n = 10) from Victoria, Australia with crust, alopecia and fissuring at 35 sites on the body.

(See Fig. 3.1 for position of site).

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<td>14</td>
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<tr>
<td>15</td>
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<tr>
<td>16</td>
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<td>17</td>
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<td>34</td>
<td>70</td>
</tr>
<tr>
<td>35</td>
<td>70</td>
</tr>
</tbody>
</table>

<sup>a</sup>A = no crust, B = some thin scale, C = thin to moderate (1-5 mm) crust, D = moderate to thick (>5mm) crust.

<sup>b</sup>A = no alopecia, B = slight hair loss, C = moderate hair loss, D = severe to complete hair loss.
Table 3.3. Haematological values for healthy captive wombats together with those of Presidente (1982), compared with wombats exhibiting severe parakeratotic sarcoptic mange.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Captive&lt;sup&gt;a&lt;/sup&gt; (n = 5)</th>
<th>Captive (Presidente, 1982)&lt;sup&gt;b&lt;/sup&gt; (n = 8 – 12)</th>
<th>Sarcoptic Mange&lt;sup&gt;c&lt;/sup&gt; (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SE</td>
<td>Range</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>11.9&lt;sup&gt;d,e&lt;/sup&gt; ± 0.7</td>
<td>10.1 – 14.2</td>
<td>13.7&lt;sup&gt;d&lt;/sup&gt; ± 0.3 (8)&lt;sup&gt;f&lt;/sup&gt;</td>
</tr>
<tr>
<td>Haematocrit (%)</td>
<td>35.2&lt;sup&gt;d,e&lt;/sup&gt; ± 1.0</td>
<td>32.0 – 37.0</td>
<td>38.1&lt;sup&gt;d&lt;/sup&gt; ± 0.9 (8)</td>
</tr>
<tr>
<td>Erythrocytes (x10&lt;sup&gt;12&lt;/sup&gt;/l)</td>
<td>5.1&lt;sup&gt;d&lt;/sup&gt; ± 0.3</td>
<td>4.4 – 6.0</td>
<td>5.7&lt;sup&gt;d&lt;/sup&gt; ± 0.2 (8)</td>
</tr>
<tr>
<td>MCV&lt;sup&gt;g&lt;/sup&gt; (fl)</td>
<td>68.8&lt;sup&gt;d&lt;/sup&gt; ± 2.1</td>
<td>62.0 – 73.0</td>
<td>66.0&lt;sup&gt;d,e&lt;/sup&gt; ± 1.0 (12)</td>
</tr>
<tr>
<td>MCH&lt;sup&gt;h&lt;/sup&gt; (pg)</td>
<td>23.2&lt;sup&gt;d&lt;/sup&gt; ± 0.2</td>
<td>23.0 – 24.0</td>
<td>22.5&lt;sup&gt;d&lt;/sup&gt; ± 0.5 (12)</td>
</tr>
<tr>
<td>MCHC&lt;sup&gt;i&lt;/sup&gt; (g/dl)</td>
<td>33.6&lt;sup&gt;d&lt;/sup&gt; ± 1.2</td>
<td>32.0 – 38.0</td>
<td>34.1&lt;sup&gt;d&lt;/sup&gt; ± 0.5 (12)</td>
</tr>
<tr>
<td>Leucocytes (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>12.5&lt;sup&gt;d&lt;/sup&gt; ± 0.7</td>
<td>10.4 – 14.0</td>
<td>12.9&lt;sup&gt;d&lt;/sup&gt; ± 1.2 (12)</td>
</tr>
<tr>
<td>Bands (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0.0&lt;sup&gt;d&lt;/sup&gt; ± 0.0</td>
<td>0.0 – 0.0</td>
<td>-</td>
</tr>
<tr>
<td>Neutrophils (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>4.0&lt;sup&gt;d&lt;/sup&gt; ± 0.5</td>
<td>3.1 – 5.5</td>
<td>5.3&lt;sup&gt;d&lt;/sup&gt; ± 0.8 (12)</td>
</tr>
<tr>
<td>Lymphocytes (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>8.3&lt;sup&gt;d&lt;/sup&gt; ± 0.7</td>
<td>6.5 – 10.5</td>
<td>6.5&lt;sup&gt;d&lt;/sup&gt; ± 0.9 (12)</td>
</tr>
<tr>
<td>Monocytes (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0.0&lt;sup&gt;d&lt;/sup&gt; ± 0.0</td>
<td>0.0 – 0.0</td>
<td>0.6&lt;sup&gt;e&lt;/sup&gt; ± 0.1 (12)</td>
</tr>
<tr>
<td>Eosinophils (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0.2&lt;sup&gt;d&lt;/sup&gt; ± 0.1</td>
<td>0.0 – 0.5</td>
<td>0.4&lt;sup&gt;d&lt;/sup&gt; ± 0.2 (12)</td>
</tr>
<tr>
<td>NRC (/100 Leucocytes)&lt;sup&gt;j&lt;/sup&gt;</td>
<td>0.2&lt;sup&gt;d&lt;/sup&gt; ± 0.2</td>
<td>0.0 – 1.0</td>
<td>-</td>
</tr>
<tr>
<td>Total Solids (g/l)</td>
<td>69&lt;sup&gt;d&lt;/sup&gt; ± 2</td>
<td>64 – 76</td>
<td>-</td>
</tr>
</tbody>
</table>

<sup>a</sup> Four female, one male, three mature and two immature wombats.
<sup>b</sup> When (n = 8) eight female, five mature and three immature wombats. When (n = 12) eight female, four male, seven mature and five immature wombats.
<sup>c</sup> Six female, two male, four mature and four immature wombats.
<sup>d</sup> – <sup>g</sup> Means in a row not sharing an identical superscript letter were significantly different from each other (P < 0.05).
<sup>h</sup> Number of wombats in parentheses.
<sup>i</sup> MCV = mean corpuscular volume.
<sup>j</sup> MCH = mean corpuscular haemoglobin.
<sup>k</sup> MCHC = mean corpuscular haemoglobin concentration.
<sup>l</sup> NRC = nucleated red cell.
Table 3.4. Biochemical values for healthy captive wombats together with those of Presidente (1982), compared with wombats exhibiting severe parakeratotic sarcoptic mange.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Captive&lt;sup&gt;a&lt;/sup&gt; (n = 5)</th>
<th>Captive (Presidente, 1982)&lt;sup&gt;b&lt;/sup&gt; (n = 5–9)</th>
<th>Sarcoptic Mange&lt;sup&gt;c&lt;/sup&gt; (n = 5–10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SE</td>
<td>Range</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>145 ± 3</td>
<td>141–155</td>
<td>139 ± 2</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>5.3 ± 0.3</td>
<td>4.7–6.1</td>
<td>4.5 ± 0.1</td>
</tr>
<tr>
<td>Chloride (mmol/l)</td>
<td>103 ± 2</td>
<td>98–110</td>
<td>96 ± 3</td>
</tr>
<tr>
<td>Calcium (mmol/l)</td>
<td>2.7 ± 0.1</td>
<td>2.5–3.0</td>
<td>2.6 ± 0.1</td>
</tr>
<tr>
<td>Phosphate (mmol/l)</td>
<td>1.0 ± 0.2</td>
<td>0.6–1.4</td>
<td>1.7 ± 0.2</td>
</tr>
<tr>
<td>Urea (mmol/l)</td>
<td>8.6 ± 0.8</td>
<td>6.9–11.8</td>
<td>5.2 ± 0.7</td>
</tr>
<tr>
<td>Creatinine (mmol/l)</td>
<td>0.07 ± 0.00</td>
<td>0.06–0.09</td>
<td>-</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>3.8 ± 0.1</td>
<td>3.5–4.1</td>
<td>5.1 ± 0.4</td>
</tr>
<tr>
<td>Total Bilirubin (µmol/l)</td>
<td>3.6 ± 1.1</td>
<td>1.0–7.0</td>
<td>-</td>
</tr>
<tr>
<td>Glutamate Dehydrogenase (U/l)</td>
<td>11 ± 2</td>
<td>7–17</td>
<td>-</td>
</tr>
<tr>
<td>Alkaline Phosphatase (U/l)</td>
<td>227 ± 37</td>
<td>142–320</td>
<td>-</td>
</tr>
<tr>
<td>γ-glutamyltransferase (U/l)</td>
<td>8.4 ± 0.9</td>
<td>7.0–11.0</td>
<td>-</td>
</tr>
<tr>
<td>Aspartate Aminotransferase (U/l)</td>
<td>34 ± 5</td>
<td>22–47</td>
<td>55 ± 13</td>
</tr>
<tr>
<td>Creatine Kinase (U/l)</td>
<td>83 ± 23</td>
<td>31–136</td>
<td>-</td>
</tr>
<tr>
<td>Total Protein (g/l) Biuret&lt;sup&gt;h&lt;/sup&gt;</td>
<td>68 ± 2</td>
<td>62–72</td>
<td>71 ± 2</td>
</tr>
<tr>
<td>Albumin (g/l) Bromocresol&lt;sup&gt;i&lt;/sup&gt;</td>
<td>32 ± 1</td>
<td>28–35</td>
<td>-</td>
</tr>
<tr>
<td>Electrophoretic&lt;sup&gt;j&lt;/sup&gt;</td>
<td>31 ± 0.5</td>
<td>29–32</td>
<td>29 ± 1</td>
</tr>
<tr>
<td>Total Globulins (g/l)</td>
<td>37 ± 2</td>
<td>30–41</td>
<td>42 ± 2</td>
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<tr>
<td>Alpha 1 (g/l)</td>
<td>2.4 ± 0.2</td>
<td>1.7–2.9</td>
<td>-</td>
</tr>
<tr>
<td>Alpha 2 (g/l)</td>
<td>7.7 ± 0.7</td>
<td>6.4–10</td>
<td>-</td>
</tr>
<tr>
<td>Beta (g/l)</td>
<td>23 ± 1</td>
<td>21–28</td>
<td>-</td>
</tr>
<tr>
<td>Gamma (g/l)</td>
<td>3.4 ± 0.7</td>
<td>1.7–5.3</td>
<td>-</td>
</tr>
</tbody>
</table>

<sup>a</sup> Four female, one male, three mature and two immature wombats.
<sup>b</sup> When (n = 9) five female and four male wombats. When (n = 5) three female and two male wombats.
<sup>c</sup> When (n = 10) seven female, three male, six mature and four immature wombats. When (n = 5) three mature female and two mature males.
<sup>d</sup> Means in a row sharing an identical superscript letter were not significantly different from each other (P > 0.05).
<sup>e</sup> Number of wombats in parentheses.
<sup>f</sup> Determined by the biuret method.
<sup>g</sup> Number of wombats in parentheses.
<sup>h</sup> Determined by the bromocresol green method.
<sup>i</sup> Determined electrophoretically.
Table 3.5. Numbers of mangy wombats with elevated glutamate dehydrogenase concentrations, reactive hepatitis and fatty change in the liver.

<table>
<thead>
<tr>
<th></th>
<th>Elevated Glutamate Dehydrogenase Concentrations</th>
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<tr>
<td></td>
<td>Yes</td>
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<tr>
<td><strong>Reactive Hepatitis</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6</td>
</tr>
<tr>
<td>No</td>
<td>2</td>
</tr>
<tr>
<td><strong>Fatty Change</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>5</td>
</tr>
<tr>
<td>No</td>
<td>3</td>
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<tr>
<td><strong>Reactive Hepatitis and Fatty Change</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3</td>
</tr>
<tr>
<td>No</td>
<td>5</td>
</tr>
</tbody>
</table>
Figure 3.1. Mean densities of mites, *S. scabiei* var. *wombati*, in 10 wombats with severe parakeratotic sarcoptic mange, at sites on lateral surfaces, along the dorsal midline and on the ventral surface of the body. The number of a site is underlined and corresponds with the number in Table 3.1. Mean densities for sites 3 and 6 on the ears are the density of mites on both the inner and outer surfaces of the pinnae.
Figure 3.2. Adult, male common wombat (*Vombatus ursinus*), from Christmas Hills, Victoria with severe parakeratotic sarcoptic mange showing typical distribution of parakeratotic scale, hair loss and excoriation.

Figure 3.3. Adult, male common wombat (*Vombatus ursinus*), from Christmas Hills, Victoria with severe parakeratotic sarcoptic mange showing parakeratotic scale adherent to the skin.
CHAPTER 4

EXPERIMENTAL INFECTION OF CAPTIVE COMMON WOMBATS (*Vombatus ursinus*) WITH *Sarcoptes scabiei* var. *Wombati*

4.1. INTRODUCTION

The histopathological changes in the skin and internal organs of common wombats with severe parakeratotic sarcoptic mange have been investigated (Skerratt, 1998). The histopathological response in wombat skin is similar to that found in other animals and humans apart from the consistently mild inflammatory infiltrate seen in the dermis of wombats compared with the variable response seen in other animals and humans (Skerratt, 1998). In addition, concurrent disease that is a risk factor for severe parakeratotic sarcoptic mange in other animals and humans was rare in free-living wombats (Skerratt, 1998). Clinical signs, intensity of mite infection and clinical pathology in free-living wombats with severe hyperkeratotic sarcoptic mange has been investigated recently (Skerratt et al., 1999; Chapter 3). Clinical signs of parakeratosis and alopecia were correlated with intensity of infection and several clinical pathological changes indicative of anaemia, inflammation and starvation were seen (Skerratt et al., 1999). However, the published data provides no information on the temporal development of clinical signs and clinical pathological changes in wombats experimentally infected with *S. scabiei* have not been investigated. The purpose of this study was to examine the clinical and clinical pathological responses of wombats infected and reinfected with varying numbers of mites.
4.2. MATERIALS AND METHODS

Collection and preparation of animals

Free-living adult and sub-adult common wombats were trapped at Glenburn, Victoria, Australia (37°26′ S, 145°25′ E) by placing cage traps over active burrow entrances (McIlroy, 1976). Wombats (with no signs of sarcoptic mange) (Skerratt et al., 1999) were transported to the Veterinary Clinical Centre, University of Melbourne, Werribee, Victoria and housed in 20 m² concrete based cattle pens with 3 concrete walls and vertical steel bars at the front inside a shed. Each pen had 2 wombats with each pair consisting of a male and female or two females. Each pen contained 2 kennels and a bed of straw. Wombats were fed lucerne and clover hay, fresh oats, carrots, and straw and were given fresh water daily. Wombats appeared to adjust well to captivity within a few days. A 10 cm² skin scraping was taken from the shoulder region of wombats brought into captivity. Skin scrapings were examined under a stereomicroscope for the presence of mites. *S. scabiei* were not found, however five wombats were infected with the endemic mite *Acaroptes vombatus* which is regularly present on wombats and is usually not associated with skin disease (Skerratt, 1998). Therefore, all wombats were given two subcutaneous treatments of ivermectin at 300 ug/kg (Ivomec, injectable for cattle, Merck & Co) 10 days apart to eradicate *A. vombatus*. All wombats were infested with ticks, mostly *Aponomma auruginans*, the common wombat tick, but some wombats were infested with *Ixodes tasmani*. Ticks were removed manually if still attached following ivermectin treatment.
Collection and preparation of S. scabiei

Two free-living common wombats with severe mange were collected from Kinglake (37°32’ S, 145°22’ E) and Healesville (37°41’ S, 145°32’ E), Victoria. The mangy wombats were euthanased with pentobarbitone sodium, 162.5mg/kg, (Lethabarb, Virbac) and mites were collected from skin and parakeratotic crust placed in petri dishes in a metal tray which was vibrated and warmed by a magnetic stirrer hot plate. This stimulates mites to move out of skin and parakeratotic crust (Sheahan and Hatch, 1975). Mites in petri dishes were counted using a stereo microscope. The wombat from which mites were collected for infections in the first experiment (see iii) had been dead 24 hours before mites were harvested. For the second experiment, mites were harvested immediately after the wombat was euthanased and were either kept at room temperature or kept at 10°C overnight before being transferred to experimental wombats.

Study design

The experimental study consisted of two experiments (Fig. 4.1).

**Experiment 1.** This was a pilot study to see whether wombats would develop mange after being infected with approximately 1000 S. scabiei. Four healthy wombats were held in captivity for 35 days before infection. Two wombats were infected simultaneously and two remained as uninfected controls and were kept separate from the infected wombats. All wombats were treated with the acaricide ivermectin 11 weeks after infection for welfare reasons and to test the effectiveness of ivermectin.

**Experiment 2.** Five months after wombats were treated for mange in experiment 1, 3 further wombats which had been in captivity for 90 days were infected
simultaneously with approximately 5000 mites. The 4 wombats from the first experiment were also infected with 5000 mites which was the second time that two of these animals had been infected. These two wombats were infected on the opposite side, right hand shoulder, to the side infected in the first experiment, left hand shoulder. Two animals that had been in captivity for 9 months were kept separate as unchallenged control wombats. Wombats were again treated with ivermectin 11 weeks after infection.

Wombats were anaesthetised with tiletamine/zolazepam at 4mg/kg (Zoletil, Virbac) in order to brush mites from petri dishes onto an area of approximately 500 cm² on their left shoulder. Wombats were subsequently reanaesthetised once a week for five weeks and then once a fortnight for six weeks in order to take 7 mm diameter skin biopsies to examine the cellular response to mite infection. In addition to tiletamine/zolazepam wombats were given local anaesthetic, lignocaine hydrochloride 20-40 mg (2% Lignocaine, Troy Laboratories), at the site for biopsy. Biopsy wounds were sutured with 4 mm thick nylon suture (Vetafil, Bengen). Blood was also taken from the cephalic vein for haematology and serum biochemistry and the skin of wombats was assessed visually to monitor any change in clinical signs of sarcoptic mange. Wombats were weighed at the start, during and at the end of each part of the experiment.

Skin scrapings (10 cm²) or 7mm diameter skin biopsies were also taken during and at the end of experiments from shoulders infected with mites to determine the intensity of mite infection (mites/cm²). These were macerated in 10% potassium hydroxide in test tubes placed in boiling water until the skin had dissolved. To determine the number of mites, the solution was made up to 10 ml with water and an
aliquot of between 1-5 ml, depending on the density of mites and the solution’s opacity, was examined under a stereomicroscope. If the density of mites was very high, then a 1 ml aliquot was added to 9 ml of water and an aliquot of between 1-5 ml of this solution was taken depending on the density of mites (Skerratt et al., 1999). On day 77 of infection, each mangy and control wombat was monitored for 10 minutes when not sleeping, eating or drinking to determine the proportion of time spent scratching.

Haematological parameters were measured using a Coulter Counter, Model S-Plus 4, impedance haematology analyser, calibrated with human blood (Coulter Electronics, Hialeah, Florida, USA), and by examination with a compound microscope of a blood smear stained by the May-Gruenwald-Giemsa method (Strumia, 1963). Electrolyte concentrations were determined by the ion-specific potentiometry method (Baker and Silverton, 1985) using a Vet Lyte electrolyte analyser (Idexx Laboratories, Zetland, NSW, Australia). Calcium, phosphate, urea nitrogen, creatinine, glutamate dehydrogenase, alkaline phosphatase, aspartate aminotransferase and creatine kinase concentrations were determined colorimetrically with a Cobas Mira spectrophotometer (Roche Diagnostics, Branchburg, New Jersey, USA) using reagents and methods from Trace Scientific (Baulkham Hills, New South Wales, Australia). Albumin and total protein concentrations were determined colorimetrically using methods and reagents from Randox Laboratories (Crumlin, Antrim County, UK). Electrophoretograms were carried out on Gelman super sepraphore mylar supported cellulose acetate strips in a Gelman semi-micro electrophoresis chamber. The strips were stained with Ponceau S and scanned with a Gelman ACD-18 densitometer (Gelman Sciences, Ann Arbor, Michigan, USA). Eleven weeks following challenge, wombats were treated with three doses of ivermectin at 300 ug/kg, each dose given 10 days apart. Wombats were also
given long acting penicillin injections, procaine and benzathine penicillin, 15 mg/kg and
11 mg/kg, respectively (Norocillin L.A., Heriot).

Data analysis

The changes in weight, haematological and serum biochemical parameters
between the start and the end of each part of the experiment were compared for infected
and control wombats. Differences were assessed for significance using the Student’s
two tailed t test, for paired and independent samples assuming either equal or unequal
variances or the Mann-Whitney U test (Sokal & Rohlf, 1997). When differences were
significant, a repeated measures general linear model was used to test whether the
change in the parameter during the course of the experiment, measurements taken on 0,
19, 34 and 77 DAI, was significantly different between infected and control wombats
(Sokal & Rohlf, 1997). The coefficient of rank (Spearman’s) correlation, one tailed, was
used to determine the correlation between two continuous variables (Sokal & Rohlf,
1997). All statistical calculations were performed either using a hand calculator or
SPSS® for Windows 8.0 (SPSS Inc., Chicago, Illinois, USA). Differences were
regarded as significant when $P < 0.05$. Two infected wombats, No. 1 and No. 7, were
excluded from statistical tests since one (1) did not develop clinical signs of mange
apart from erythema and excoriation and another (7) died during the course of the
experiment.

4.3. RESULTS

Clinical observations
All infections led to the development of sarcoptic mange whether wombats had 1000 (n = 2) or 5000 (n = 7) *S. scabiei* placed on their shoulder or whether wombats were infected for the first time (n = 7) or reininfected (n = 2) (Table 4.1). Seven infections resulted in erythema, parakeratosis, alopecia, excoriation and fissuring of parakeratotic scale and skin. These occurred in one wombat (No.2) given 1000 mites, 4 (Nos. 3-6) given 5000 mites and two wombats (Nos. 1 & 2) that were reininfected with 5000 mites (Table 4.1). In contrast, two infections led to only some of the above clinical signs, erythema and excoriation in a wombat (No. 1) given 1000 mites and parakeratosis and fissuring of parakeratotic scale and skin in a wombat (No. 7) given 5000 mites (Table 4.1). Although the rate of development of lesions varied among wombats given a similar number of mites (Table 4.1), clinical signs of mange developed earlier and lesions progressed more rapidly on wombats infected with 5000 mites compared with wombats infected with 1000 mites. In addition, clinical signs were seen earlier but lesions developed at a slower rate in a reininfected wombat that had previously developed mild parakeratotic sarcoptic mange (Table 4.1). The four control wombats did not develop lesions.

The sequence of development of clinical signs of sarcoptic mange was erythema, shortly followed by parakeratotic scale, which was adherent to the skin and then several weeks later alopecia, excoriation and fissuring of parakeratotic scale and skin (Table 4.1). In wombats infected for the first time, erythema developed within 24 hours in a wombat (No. 5) given 5000 mites, within 3 to 14 days after infection (DAI) in 4 wombats given 1000 (No. 2) and 5000 mites (Nos. 3, 4 & 6), and by 49 DAI in a wombat (No. 1) given 1000 mites. This last animal did not develop other signs of
sarcoptic mange apart from excoriation by the end of the experiment (77 DAI). In reininfected wombats, erythema developed within 24 hours in one wombat (No. 2) given 5000 mites that had previously developed mild parakeratotic sarcoptic mange and within 3 to 14 DAI in another wombat (No. 1) given 5000 mites that had previously developed clinical signs of erythema and excoriation.

Adherent parakeratotic scale developed shortly after erythema appeared, within 7 to 14 days in 4 of 5 wombats infected for the first time, one (No. 2) given 1000 and three (Nos. 4-6) given 5000 mites, respectively and was apparent at the same time as erythema in the remaining wombat (No. 3) given 5000 mites. In reininfected wombats given 5000 mites, parakeratotic scale developed between 7 and 14 days after erythema in one wombat (No. 2) that had previously exhibited mild parakeratotic sarcoptic mange and at the same time as erythema in another wombat which had not developed sarcoptic mange previously apart from signs of erythema and excoriation. Overall, parakeratosis was apparent within 14 DAI in 5 of 8 wombats and within 21 DAI in the remaining 3 wombats (Table 4.1).

Alopecia became apparent within 14 to 42 days after erythema had developed; within 14 days in one wombat (No. 6) given 5000 mites, 21 days in three wombats (Nos. 3 & 4) given 5000 and (No. 1) reinfected with 5000 mites and in the three remaining wombats 28, 35 and 42 days, (Nos. 2, 2 & 5) given 1000, reinfected with 5000 and given 5000 mites, respectively (Table 4.1). Lifting of parakeratotic scale correlated with initial signs of alopecia. However, once parakeratotic scale started to build up and form a crust it was less likely to lift. Signs of excoriation were evident in six wombats (Nos. 3-6 and 1 & 2 upon reinfection) by 49 DAI and one wombat (No. 2)
by 77 DAI. Fissuring of skin and parakeratotic crust became apparent by 49 DAI and was present in all wombats that were infected for the first time with 5000 mites by 77 DAI.

Lesion distribution

During the course of infection, erythema and parakeratosis spread from the infection site on the left shoulder onto the face and along the flanks. The parakeratosis also thickened to form a parakeratotic crust, several millimetres thick in the shoulder region in six wombats (Table 4.2). Erythema spread to the ventral surfaces of the thorax and abdomen and to the medial surface of the legs. Alopecia was confined mainly to the shoulder region but its severity increased during the experiment. Excoriation was evident on shoulders, flanks and ventral surfaces. Fissuring of parakeratotic crust and skin was confined to the shoulders and flanks. The right shoulder, opposite the one infected, developed erythema and some parakeratosis by 77 DAI in four wombats that developed severe parakeratotic sarcoptic mange on their left shoulder. In three wombats that developed mild to moderate parakeratotic sarcoptic mange on their left shoulder, the right shoulder showed little to no signs of sarcoptic mange.

Mite population

S. scabiei were recovered from 8 of 9 clinically infected wombats at the end of the experiments, 6 of 7 wombats infected for the first time and 2 of 2 reinfected wombats (Table 4.2). Intensity of infection increased dramatically in wombats that were initially infected with 5000 mites (Table 4.2). Intensity of infection did not appear to
increase greatly in one wombat (No. 2) infected with 1000 mites and mites were not found on another wombat (No. 1) given 1000 mites that only developed erythema by 49 DAI. For wombats given 5000 mites, the lowest intensity of infection by a factor of at least 5, 77 DAI, occurred in a reinfected wombat given 5000 mites (Table 4.2). The highest intensity of infection by a factor of at least 5, 49 DAI, occurred in a wombat given 5000 mites that lost 12% of its body weight prior to infection (Table 4.2). *S. scabiei* were never found on control wombats. The density of mites was correlated with the thickness of parakeratotic crust on the shoulder for wombats given 5000 mites, $r = 0.77 \ (P < 0.05, \ n = 6)$, but not time spent scratching ($P = 0.1, \ n = 6$) (Table 4.2).

Wombats with mange scratched significantly more than control wombats ($P < 0.05, \ n=8$) using the Mann-Whitney U test or the Student’s two tailed t test, for independent samples assuming unequal variances (Table 4.2).

**Response to ivermectin**

Clinical signs resolved in all wombats treated with three subcutaneous injections of ivermectin at 300 µm/kg with each injection given 10 days apart. However, a recrudescence of mild sarcoptic mange occurred in 3 wombats two months after the last injection of ivermectin. Wombats were retreated with three injections of ivermectin and mites were successfully eliminated with wombats showing no signs of mange four months after the last injection of ivermectin in the second treatment.

After treatment with ivermectin, parakeratotic crust started to detach from skin within 10 days and large amounts detached after 20 days. Fissuring of skin disappeared within 20 days. Within 28 days, parakeratotic crust had detached from the skin with
most of it having been removed by the wombat. Erythema and excoriation were reduced but did not disappear until 63 days after treatment commenced (DAT). Alopecia increased substantially after treatment and extended until all parakeratotic crust had been removed 63 DAT. Hair regrowth became evident once scale crust had detached at 28 DAT but was not complete until 84 DAT. Clinical signs of erythema and parakeratosis reappeared several weeks after clinical signs had resolved in three wombats that had the greatest intensity of infection prior to treatment. *S. scabiei* were recovered from skin scrapings from these wombats.

**Clinicopathological findings**

The seven infected wombats exhibited significant changes, 77 DAI, in the concentration of neutrophils, monocytes, sodium, phosphate, creatinine, total bilirubin and gamma globulins (Student’s t test for paired samples, *P* < 0.05) (Table 4.3 & 4.4). However, only the concentration of leucocytes and neutrophils increased significantly after 77 days in wombats with mange compared with control wombats (Student’s t test, for independent samples, unequal variances assumed, *P* < 0.05; repeated measures general linear model, *P* > 0.05) (Table 4.3 & 4.4). The change in neutrophil concentration was correlated with intensity of infection for wombats given 5000 mites, *r* = 0.89 (*P* < 0.05, *n* = 6) (Table 4.2). The rise in concentration of leucocytes and neutrophils occurred steadily during the 77 days of infection. Wombats lost weight during the development of clinical signs and this decline was significant when compared to the change in weight of control wombats, -1.3 kg ± 0.6 standard error (SE) versus 2.0 kg ± 0.9 SE (Student’s t test, independent samples, equal variances assumed,
Prior to the first experiment, one control wombat and one wombat to be infected gained weight and the other control wombat and wombat to be infected lost weight in captivity. All wombats had either gained or maintained their weight in the months prior to the commencement of the second experiment apart from one female wombat that had lost 3 kg, 12% of its bodyweight attributed to harassment by its male pen mate. It was moved to an enclosure on its own for the second experiment. This wombat later died through misadventure 49 days after the start of experiment 2.

4.4. DISCUSSION

Erythema, which is the first clinical sign of sarcoptic mange in wombats, usually occurs within 14 days of infection with 5000 mites. Humans, pigs and foxes develop delayed hypersensitivity reactions, of which erythema is a clinical sign, within 14 to 28 days of infection with *S. scabiei* (Mellanby, 1944; Davis & Moon, 1990a; Little et al., 1998). Parakeratosis follows the development of erythema within 14 days and arises from the actions of mites burrowing in the Malpighian layer of the epidermis leading to disturbance of the terminal differentiation of squamous epithelial cells (Van Neste and Lachapelle, 1981; Van Neste and Staquet, 1986). Parakeratotic scale does not readily detach from skin and so forms a crust. The production of parakeratosis is correlated with the density of mites burrowing in the epidermis (Skerratt et al., 1999) and in this study the thickness of parakeratotic crust was also correlated with the intensity of infection. Alopecia and excoriation did not become evident until several weeks after erythema and parakeratosis had developed. A similar progression of dermatological changes has been reported in coyotes (*Canis latrans*), dogs (*Canis familiaris*) and foxes
Alopexia and excoriation were the consequence of scratching, which occurred more frequently in infected, compared with control, wombats. Although correlated with alopecia (Skerratt et al., 1999), intensity of infection may not be causally related to hair loss. Intensity of infection was not correlated with proportion of time spent scratching in this study. Rather, the degree of pruritus and therefore scratching probably reflects the severity of an underlying dermatological immune response (Davis & Moon, 1990b) although there may be a pruritic sensation associated directly with large numbers of mites burrowing in the skin.

The slower rate of development of clinical signs of mange in wombats given 1000 mites compared with those given 5000 mites could be directly related to the infecting dose. For example it is known that the rate of development of signs of cutaneous hypersensitivity in pigs is dependent on the number of *S. scabiei* present (Davis and Moon, 1990a). The variation in the rate of development of clinical signs of mange in wombats given approximately the same number of mites could also be due to minor variations in the infective dose. However, the immune response of the wombat to the mite and individual skin characteristics such as degree of soiling of the skin may also affect the rate of development of clinical signs. Rapid induction phases have been reported for humans reinfected with *S. scabiei* (Mellanby, 1944) and it is noteworthy that one wombat infected for the first time, developed erythema 1 DAI (No. 5). This animal also developed less severe mange and a lower intensity of infection than other wombats infected for the first time with approximately the same number of mites, suggesting that its early response to mite infection may have retarded the development of mange. The reason for this relatively early response is not known but it is possible
that this wombat had previously had mange in the wild and that the early response was anamnestic. A secondary immune response may explain why one reinfected wombat (No. 2) developed early erythema and subsequently only mild mange and the lowest intensity of infection. Such a response has been reported to affect mite survival in humans, rabbits and dogs but not in foxes (Mellanby, 1944; Arlian et al., 1994, 1996a; Little et al., 1998). Loss of weight prior to the experiment may explain why one wombat (No. 7) developed little erythema, hair loss or excoriation, which might be a manifestation of a suppressed immune response. It might also explain why this wombat had the thickest parakeratotic crust and a greater intensity of infection. An incompetent immune response is recognised as promoting the development of parakeratotic sarcoptic mange in wombats (Skerratt, 1998).

One wombat (No. 1) that was infected with 1000 mites and held in contact with a second wombat (No. 2) that developed mange after being infected with 1000 mites, did not develop clinical signs of mange apart from erythema and slight excoriation. Mites were not found on this wombat at the end of the first experiment. It is possible that the mites used to inoculate the wombat were affected by the 24 hr delay in harvesting them and that they did not readily establish on their new host. In addition, this wombat was a male with dirty and often wet skin, which may have affected the ability of mites to infect it. It is possible that the threshold dose of *S. scabiei* required before mange will develop varies between individuals since this wombat developed mange when it was given a higher dose of mites. Failure of *S. scabiei* to establish on a new host has occurred in an infection experiment with coyotes where a similar number of mites were transferred, 500-1500 (Samuel, 1981).
Mange did not develop to any extent on the shoulder opposite the one infected. Rather, mange occurred at the site of inoculation and then adjacent areas. Spread of infection from the inoculation site has also been reported for foxes. However, in this species, most of the body became involved within 4 weeks after an initial dose of 5500 mites (Little et al., 1998) reflecting a more rapid spread of mites than occurred in wombats. Mites also spread rapidly, within 5 weeks, from the back to body extremities in dogs infected with *S. scabiei* from a red fox (Bornstein, 1991). Mites spread more slowly in red foxes infected on their midback with few mites, < 200, with most body extremities infected within 4 months (Bornstein et al., 1995). Rapid migration of mites from the body to the head in foxes is thought to be due to foxes gnawing at infection sites (Gerasimov, 1953). The slower migration of mites in wombats may be due to the fact that they groom themselves differently to foxes and dogs, using only their claws and not their teeth, and do not sequentially groom their body (Triggs, 1996). Because wombats do not use their teeth they may not readily transfer mites from one side of the body to the other. This suggests that the symmetrical, severe mange that is seen in free-living wombats (Skerratt et al., 1999) is due to mites infecting both sides of the wombat simultaneously. However, the significant difference in intensity of infection of *S. scabiei* between adjacent sites on the body suggests that there is some degree of site selection by the mite (Skerratt et al., 1999).

The only significant haematological change associated with development of sarcoptic mange in common wombats in this study, neutrophilia without a left shift, is indicative of a continuing acute but not overwhelming inflammatory response (Duncan et al., 1994). Neutrophilia may be largely due to the secondary effects of *S. scabiei* infection, such as excoriation with traumatic injury to the skin and intra-dermal
abscesses, although the change in neutrophil concentration was correlated with intensity of infection. Skerratt (1998) reported that neutrophilic exocytosis in the skin of common wombats with severe hyperkeratotic sarcoptic mange appeared to be associated more commonly with bacteria than mites. Free-living common wombats with severe hyperkeratotic sarcoptic mange exhibited a neutrophilia. However they also had other haematological and biochemical changes indicative of anaemia, chronic inflammatory disease and starvation (Skerratt et al., 1999), most likely reflecting a longer duration of clinical disease. *S. scabiei* infection in other animals causes neutrophilia along with other haematological and biochemical changes (Pence et al., 1983; Arlian et al., 1988b, 1995; Dalapati et al., 1996; Little et al., 1998). The difference in haematological and biochemical changes between studies is possibly due to a difference in the severity, extent and duration of mange rather than a host difference. The changes seen in this study are most similar to the changes seen in short term infection studies, 7 – 8 weeks, of dogs and foxes (Arlian et al., 1995; Little et al., 1998). In addition to a significant rise in neutrophils, dogs exhibited a significant decrease in haemoglobin concentration and haematocrit and foxes exhibited a rise in eosinophils (Arlian et al., 1995; Little et al., 1998). Although animals developed severe mange in the current study and the studies of Arlian et al. (1995) and Little et al. (1998), haematological and biochemical changes were few. It appears that mange must be chronic or occur in free-living animals before severe haematological and biochemical changes occur (Arlian et al., 1988b; Pence et al., 1983; Dalapati et al., 1996; Skerratt et al., 1999).

The decline in weight of mangy wombats in this experimental study has also been seen in free-living wombats and is thought to be due to the “energy demands” of mange (Skerratt et al., 1999). The demand is not accounted for by the energy
consumption of mites (Arlian et al., 1988a) but is likely to be due to production of parakeratotic scale and scratching and possibly also due to reduced food intake due to the systemic effects of severe inflammatory disease.

The recrudescence of sarcoptic mange in wombats after treatment and recovery from mange could have been due to reinfection by mites persisting in the environment, such as in scale crust that had detached from skin. In cattle, the treatment regime used would have resulted in therapeutic levels of the drug being maintained for at least a month (Campbell, 1989), but similar data are not available for wombats. It is known that *S. scabiei* survive at least 3 weeks in the laboratory under favourable conditions of high humidity (97%) and low temperature (10°C) (Arlian et al., 1989). Although ivermectin is an effective acaricide in a variety of mammals (Campbell, 1989), the failure of ivermectin to completely eliminate *S. scabiei* in wombats with severe hyperkeratotic sarcoptic mange is not unusual (C. Davis, pers. comm.). Treatment failure with 3 doses of ivermectin given 14 days apart has been reported in Australian aboriginals with crusted scabies (Walton et al., 1999b). In the case of wombats successful elimination of mites is achieved by concurrently treating with another acaricide topically and manually removing scale crust (C. Davis, pers. comm.). Similar treatment regimes have been used in horses and humans with hyperkeratotic scabies (Christensson et al., 1984; Meinking et al., 1995). Higher doses of drug may also have been more effective, since psoroptic mites have been eliminated more rapidly in rabbits given higher doses of ivermectin, 800 µg/kg (Harikrishnan et al., 1996). However there is potential for drug toxicity especially in animals severely debilitated by sarcoptic mange because of an alteration in the pharmacokinetic properties of drugs.
In conclusion, the sequence of clinical signs of sarcoptic mange observed in wombats was generally consistent between animals and was independent of initial dose of mites or of previous, recent exposure to *S. scabiei*. Erythema occurred initially, and was closely followed by parakeratotic scale, and subsequently by alopecia and fissuring. The extent and severity of clinical signs such as the thickness of parakeratotic crust and degree of alopecia increased with time and spread slowly from the infection site. The rate of development of clinical signs was dependent on the initial dose of mites and probably on factors such as naivety of the immune system and body condition. The rate of increase in intensity of infection was also probably dependent on similar factors. Neutrophils steadily rose in concentration in blood, weight was gradually lost and pruritus significantly increased as sarcoptic mange developed in wombats. Treatment with 3 injections of ivermectin given 10 days apart led to complete resolution of clinical signs but not all mites were eliminated and there was a recrudescence of sarcoptic mange. Mites were successfully eliminated after a second treatment with 3 injections of ivermectin.
Table 4.1. Temporal development of clinical signs of sarcoptic mange in common wombats infected and reinfected with 1000 and 5000 mites. Stars (*) indicate presence, severity and extent of clinical sign.

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<td></td>
</tr>
<tr>
<td>2 (Reinfected)</td>
<td>5000</td>
<td>Erythema</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>***</td>
<td>****</td>
<td>*****</td>
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<tr>
<td></td>
<td></td>
<td>Scale</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>***</td>
<td>****</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hair loss</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>***</td>
<td>****</td>
<td>*****</td>
<td>****</td>
<td>***</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controls (n=4)</td>
<td>0</td>
<td>Erythema</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Scale</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hair loss</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*a Erythema * represents erythema on the infected shoulder, each additional star (*) represents erythema in an additional region, these being the flank, face, abdomen and chest.

*b Scale * represents parakeratotic scale developing on the infected shoulder, ** represents an increase in thickness of scale to 1-2 mm and the formation of continuous sheets of scale, additional stars (*) represent the spread of parakeratosis to the flank and increasing thickness of scale with 5 stars indicating a scale thickness of approximately 10 mm thick on the shoulder.

*c Hair loss * represents the first signs of hair loss on the infected shoulder, additional stars (*) represent increasing hair loss on the shoulder region and hair loss on the flank, with 5 stars indicating complete hair loss on the shoulder.
Table 4.2. Number of mites/cm² and thickness of scale crust on shoulders, percentage of time spent scratching determined by 10 minute observation and change in concentration of circulating neutrophils for wombats at 46 and 77 days after infection with 1000 or 5000 mites on their shoulders.

<table>
<thead>
<tr>
<th>Wombat No.</th>
<th>Mites</th>
<th>Mites on Shoulder (/cm²)</th>
<th>Crust (mm)</th>
<th>Scratch (% 10min)</th>
<th>Neutrophil Increase (x10⁹/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td>1 (Infected)</td>
<td>1000</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2 (Infected)</td>
<td>1000</td>
<td>0</td>
<td>0</td>
<td>0.3</td>
<td>0</td>
</tr>
<tr>
<td>3 (Infected)</td>
<td>5000</td>
<td>0</td>
<td>66</td>
<td>9</td>
<td>6200</td>
</tr>
<tr>
<td>4 (Infected)</td>
<td>5000</td>
<td>0</td>
<td>320</td>
<td>3</td>
<td>11600</td>
</tr>
<tr>
<td>5 (Infected)</td>
<td>5000</td>
<td>0</td>
<td>72</td>
<td>0</td>
<td>1200</td>
</tr>
<tr>
<td>6 (Infected)</td>
<td>5000</td>
<td>0</td>
<td>144</td>
<td>35</td>
<td>10800</td>
</tr>
<tr>
<td>7 (Infected)</td>
<td>5000</td>
<td>0</td>
<td>2480</td>
<td>1 a</td>
<td>-</td>
</tr>
<tr>
<td>1 (Reinfected)</td>
<td>5000</td>
<td>0</td>
<td>20</td>
<td>8</td>
<td>4800</td>
</tr>
<tr>
<td>2 (Reinfected)</td>
<td>5000</td>
<td>0</td>
<td>12</td>
<td>0</td>
<td>220</td>
</tr>
<tr>
<td>3 (Control)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4 (Control)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>8 (Control)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>9 (Control)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

ND = Not determined.

aWombat 7 died on day 49 through misadventure.
Table 4.3. Haematological values for wombats infected with *S. scabiei* and control wombats prior to and 11 weeks after experimental infection.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Infected&lt;sup&gt;a&lt;/sup&gt; (n = 7)</th>
<th>Control&lt;sup&gt;b&lt;/sup&gt; (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 0</td>
<td>Day 77</td>
</tr>
<tr>
<td><strong>Mean ± SE</strong></td>
<td><strong>Mean ± SE</strong></td>
<td><strong>Mean ± SE</strong></td>
</tr>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>11.0 ± 0.4</td>
<td>11.5 ± 0.4</td>
</tr>
<tr>
<td>Haematocrit (%)</td>
<td>33.9 ± 1.3</td>
<td>34.4 ± 1.1</td>
</tr>
<tr>
<td>MCHC&lt;sup&gt;e&lt;/sup&gt; (g/dl)</td>
<td>32.4 ± 0.6</td>
<td>33.3 ± 0.3</td>
</tr>
<tr>
<td>Leucocytes (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>8.3 ± 0.8</td>
<td>12.5 ± 2.1</td>
</tr>
<tr>
<td>Bands (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neutrophils (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>2.9 ± 0.4</td>
<td>7.5 ± 1.7</td>
</tr>
<tr>
<td>Lymphocytes (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>5.1 ± 0.5</td>
<td>4.2 ± 0.6</td>
</tr>
<tr>
<td>Monocytes (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0.04 ± 0.03</td>
<td>0.23 ± 0.05</td>
</tr>
<tr>
<td>Eosinophils (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0.26 ± 0.09</td>
<td>0.49 ± 0.12</td>
</tr>
<tr>
<td>Basophils (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>0</td>
<td>0.08 ± 0.04</td>
</tr>
<tr>
<td>NRC&lt;sup&gt;f&lt;/sup&gt; (/100 Leucocytes)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Platelets (x10&lt;sup&gt;9&lt;/sup&gt;/l)</td>
<td>449 ± 59</td>
<td>404 ± 50</td>
</tr>
<tr>
<td>Total Solids (g/l)</td>
<td>65.6 ± 1.8</td>
<td>69.1 ± 1.4</td>
</tr>
</tbody>
</table>

<sup>a</sup> Five female, two male, adult wombats.

<sup>b</sup> Four female, one sub-adult and three adult wombats.

<sup>cd</sup> Means in a row sharing an identical superscript letter were significantly different from each other (*P* < 0.05).

<sup>e</sup> MCHC = mean corpuscular haemoglobin concentration.

<sup>f</sup> NRC = nucleated red cell.
Table 4.4. Biochemical values for wombats infected with *S. scabiei* infected and control wombats prior to and 11 weeks after experimental infection.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Infected ($n = 7$)</th>
<th>Change</th>
<th>Control ($n = 4$)</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 0</td>
<td>Day 77</td>
<td>Day 0</td>
<td>Day 77</td>
</tr>
<tr>
<td></td>
<td>Mean ± SE</td>
<td>Mean ± SE</td>
<td>Mean ± SE</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>$142c ± 1$</td>
<td>$150c ± 3$</td>
<td>$8 ± 2$</td>
<td>$147 ± 2$</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>$5.3 ± 0.2$</td>
<td>$5.1 ± 0.1$</td>
<td>$-0.3 ± 0.2$</td>
<td>$5.2 ± 0.2$</td>
</tr>
<tr>
<td>Chloride (mmol/l)</td>
<td>$101 ± 1$</td>
<td>$105 ± 2$</td>
<td>$4 ± 1$</td>
<td>$103 ± 0$</td>
</tr>
<tr>
<td>Calcium (mmol/l)</td>
<td>$2.5 ± 0.0$</td>
<td>$2.5 ± 0.1$</td>
<td>$0.1 ± 0.1$</td>
<td>$2.2 ± 0.2$</td>
</tr>
<tr>
<td>Phosphate (mmol/l)</td>
<td>$1.5c ± 0.1$</td>
<td>$1.2c ± 0.1$</td>
<td>$-0.3 ± 0.1$</td>
<td>$1.2 ± 0.2$</td>
</tr>
<tr>
<td>Urea (mmol/l)</td>
<td>$5.9 ± 0.4$</td>
<td>$6.1 ± 0.5$</td>
<td>$0.2 ± 0.3$</td>
<td>$5.8 ± 0.4$</td>
</tr>
<tr>
<td>Creatinine (mmol/l)</td>
<td>$0.10c ± 0.00$</td>
<td>$0.07c ± 0.00$</td>
<td>$-0.02 ± 0.00$</td>
<td>$0.08 ± 0.00$</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>$3.7 ± 0.3$</td>
<td>$4.4 ± 0.2$</td>
<td>$0.7 ± 0.2$</td>
<td>$4.3 ± 0.6$</td>
</tr>
<tr>
<td>Total Bilirubin (µmol/l)</td>
<td>$2.7c ± 0.3$</td>
<td>$1.3c ± 0.5$</td>
<td>$-1.4 ± 0.6$</td>
<td>$2.3 ± 0.5$</td>
</tr>
<tr>
<td>Glutamate Dehydrogenase (U/l)</td>
<td>$10.2 ± 1.5$</td>
<td>$10.8 ± 1.1$</td>
<td>$0.7 ± 1.0$</td>
<td>$14.5 ± 3.5$</td>
</tr>
<tr>
<td>Alkaline Phosphatase (U/l)</td>
<td>$335 ± 66$</td>
<td>$186 ± 27$</td>
<td>$-149 ± 44$</td>
<td>$410 ± 171$</td>
</tr>
<tr>
<td>γ-glutamyl transferase (U/l)</td>
<td>$8.0c ± 0.7$</td>
<td>$4.9c ± 0.6$</td>
<td>$-3.1 ± 0.5$</td>
<td>$6.0 ± 2.0$</td>
</tr>
<tr>
<td>Aspartate Aminotransferase (U/l)</td>
<td>$45 ± 5$</td>
<td>$40 ± 2$</td>
<td>$-4 ± 4$</td>
<td>$80 ± 27$</td>
</tr>
<tr>
<td>Creatine Kinase (U/l)</td>
<td>$73 ± 23$</td>
<td>$63 ± 5$</td>
<td>$-9 ± 22$</td>
<td>$1183 ± 982$</td>
</tr>
<tr>
<td>Total Protein (g/l) Biuret$^d$</td>
<td>$69 ± 2$</td>
<td>$69 ± 1$</td>
<td>$0 ± 1$</td>
<td>$63 ± 3$</td>
</tr>
<tr>
<td>Albumin (g/l) Bromocresol$^e$</td>
<td>$31 ± 1$</td>
<td>$31 ± 0$</td>
<td>$0 ± 1$</td>
<td>$29 ± 2$</td>
</tr>
<tr>
<td>Electrophoretic$^f$</td>
<td>$31 ± 1$</td>
<td>$31 ± 1$</td>
<td>$0 ± 1$</td>
<td>$31 ± 1$</td>
</tr>
<tr>
<td>Total Globulins (g/l)</td>
<td>$38 ± 1$</td>
<td>$38 ± 2$</td>
<td>$0 ± 1$</td>
<td>$32 ± 2$</td>
</tr>
<tr>
<td>Alpha 1 (g/l)</td>
<td>$2.5c ± 0.1$</td>
<td>$2.8 ± 0.2$</td>
<td>$0.2 ± 0.3$</td>
<td>$2.6 ± 0.5$</td>
</tr>
<tr>
<td>Alpha 2 (g/l)</td>
<td>$6.5c ± 0.5$</td>
<td>$6.6 ± 0.4$</td>
<td>$0.1 ± 0.6$</td>
<td>$6.5 ± 0.4$</td>
</tr>
<tr>
<td>Beta (g/l)</td>
<td>$26 ± 1$</td>
<td>$26 ± 2$</td>
<td>$0 ± 1$</td>
<td>$21 ± 1$</td>
</tr>
<tr>
<td>Gamma (g/l)</td>
<td>$2.7c ± 0.3$</td>
<td>$1.5c ± 0.3$</td>
<td>$-1.2 ± 0.4$</td>
<td>$1.9 ± 0.2$</td>
</tr>
</tbody>
</table>

$^a$ Five female, two male, adult wombats.

$^b$ Four female, one sub-adult and three adult wombats.

$^c$ Means in a row sharing an identical superscript letter were significantly different from each other ($P < 0.05$).

$^d$ Determined by the biuret method.

$^e$ Determined by the bromocresol green method.

$^f$ Determined electrophoretically.
Figure 4.1 Schematic representation of experimental infection of common wombats, *Vombatus ursinus*, with the mange mite, *Sarcoptes scabiei var. wombati*.

In the first part of the experiment, 2 wombats were infected with 1000 mites and there were 2 control wombats. Wombats were treated with ivermectin after 3 months. At 8 months, 2 wombats were reinfected and 5 were infected for the first time with 5000 mites and there were 2 control wombats. Again wombats were treated with ivermectin 3 months later.
Figure 4.2 Mean concentration of neutrophils in control wombats and mange affected wombats at 0, 19, 34 and 77 days after infection with *S. scabiei* var. *wombati*.

Error bars represent the standard error. Control wombats (n=4), mangy wombats (n=7).
Figure 4.3. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 2) with sarcoptic mange, 77 days after infection of her left shoulder with 1000 *Sarcoptes scabiei* var. *wombati*.

Figure 4.4. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 2) with parakeratotic scale, hair loss, erythema and excoriation on the left shoulder, 77 days after infection with 1000 *Sarcoptes scabiei* var. *wombati*. 
Figure 4.5. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 3) with sarcoptic mange, 77 days after infection of her right shoulder with 5000 *Sarcoptes scabiei* var. *wombati*.

Figure 4.6. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 3) with parakeratotic scale crust, hair loss, fissuring and excoriation on the right shoulder, 77 days after infection with 5000 *Sarcoptes scabiei* var. *wombati*. 
Figure 4.7. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 4) with sarcoptic mange, 77 days after infection of her right shoulder with 5000 *Sarcoptes scabiei* var. *wombati*.

![Image of adult wombat with sarcoptic mange](image1)

Figure 4.8. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 4) with parakeratotic scale crust, hair loss and fissuring on the right shoulder, 77 days after infection with 5000 *Sarcoptes scabiei* var. *wombati*.

![Image of adult wombat with parakeratotic scale crust](image2)
Figure 4.9. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 5) with sarcoptic mange, 77 days after infection of her right shoulder with 5000 *Sarcoptes scabiei* var. *wombati*.

Figure 4.10. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 5) with parakeratotic scale and hair loss on the right shoulder, 77 days after infection with 5000 *Sarcoptes scabiei* var. *wombati*. 
Figure 4.11. Adult, male, captive common wombat (*Vombatus ursinus*) (Infected 6) with sarcoptic mange, 77 days after infection of his right shoulder with 5000 *Sarcoptes scabiei* var. *wombati*.

Figure 4.12. Adult, male, captive common wombat (*Vombatus ursinus*) (Infected 6) with parakeratotic scale crust, hair loss and fissuring on the right shoulder, 77 days after infection with 5000 *Sarcoptes scabiei* var. *wombati*. 
Figure 4.13. Adult, male, captive common wombat (*Vombatus ursinus*) (Reinfected 1) with sarcoptic mange, 77 days after infection of his right shoulder with 5000 *Sarcoptes scabiei* var. *wombati*.

Figure 4.14. Adult, male, captive common wombat (*Vombatus ursinus*) (Reinfected 1) with parakeratotic scale crust, hair loss and fissuring on the right shoulder, 77 days after infection with 5000 *Sarcoptes scabiei* var. *wombati*. 
Figure 4.15. Adult, female, captive common wombat (*Vombatus ursinus*) (Reinfected 2) with sarcoptic mange, 77 days after infection of her right shoulder with 5000 *Sarcoptes scabiei* var. *wombati*.

Figure 4.16. Adult, female, captive common wombat (*Vombatus ursinus*) (Reinfected 2) with parakeratotic scale and hair loss on the right shoulder, 77 days after infection with 5000 *Sarcoptes scabiei* var. *wombati*. 
Figure 4.17. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 7) with sarcoptic mange, 41 days after infection of her right shoulder with 5000 *Sarcopes scabiei* var. *wombati*. The fur was clipped so that parakeratotic scale was visible.

Figure 4.18. Adult, female, captive common wombat (*Vombatus ursinus*) (Infected 3) 44 days after treatment for severe sarcoptic mange with three consecutive injections of ivermectin at 300 µg/kg.
CHAPTER 5

INFLAMMATORY RESPONSE IN THE DERMIS OF COMMON WOMBATS INFECTED WITH SARCOPTES SCABIEI VAR. WOMBATI

5.1. INTRODUCTION

The opportunity to examine various aspects of the relationship between *S. scabiei* and wombats presented itself following the collection of material from free-living common wombats with sarcoptic mange and from captive common wombats experimentally infected with *S. scabiei*. Here the dermal cellular immune responses of common wombats to infection with *S. scabiei* have been investigated. In Chapter 6 the population dynamics of *S. scabiei* var. *wombati* have been examined.

The dermal cellular immune response of the host towards *S. scabiei* appears to regulate the intensity of infection (Davis and Moon, 1990c; Arlian et al., 1994; Arlian, 1996; Arlian et al., 1996a; Arlian et al., 1997). The dermal cellular immune response to *S. scabiei* infection has been examined in humans and several animals including rabbits, dogs, foxes, chamois and pigs (Sheahan, 1975; Ackerman, 1985; Van Neste, 1986; Morsy and Gaafar, 1989; Arlian et al., 1994; Arlian, 1996; Arlian et al., 1996a; Arlian et al., 1997; Little et al., 1998; Rode et al., 1998). Whilst marsupials have an immune system similar to that of eutherian mammals (Croix et al., 1989) differences have been identified such as slower antibody responses in metatherians (Johnson et al., 1989; Wilkinson et al., 1992; Skerratt et al., 1997), suggesting that differences may exist in the immune response of wombats to *S. scabiei* that affect its ability to
regulate the abundance of mites. In this study, conventional staining methods as well as immunohistochemistry using antibodies raised against conserved antigens were used to identify cell types in the dermis of common wombats infected with *S. scabiei*. The immunohistochemical staining of T and B lymphocytes of Australian marsupials such as the koala (*Phascolarctos cinereus*), brushtail possum (*Trichosurus vulpecula*), tammar wallaby (*Macropus eugenii*) and ringtail possum (*Pseudocheirus peregrinus*) has been achieved using antibodies raised against intracytoplasmic regions of receptors such as CD3 and CD79b of human T and B lymphocytes (Hemsley et al., 1995; Wilkinson et al., 1995), suggesting that these antibodies may also identify T and B lymphocytes of wombats. Immunohistochemistry using the avidin-biotin-peroxidase method has been employed successfully in studies on tissues of the southern hairy-nosed wombat (*Lasiorhinus latifrons*) with anti-human tissue antibodies, such as antibodies raised against desmin, myoglobin, vimentin, glial fibrillary acidic protein, S100 protein and keratin (Byard et al., 1998). However, anti-human cell antibodies, such as anti-human leucocyte common antigen did not stain wombat leucocytes (Byard et al., 1998).

5.2. MATERIALS AND METHODS

*Animals/tissues*

**Free-living wombats with severe sarcoptic mange**

Ten free-living wombats with severe parakeratotic sarcoptic mange were collected and euthanased (see Chapter 3.2). Three free-living wombats without sarcoptic mange that had been hit on a road by a vehicle or shot by landowners were also collected for comparison. The entire skin of each wombat was removed and a 7
mm diameter skin biopsy punch attached to a drill was used to remove cores of skin from 6 sites, (Nos. 7 – 12, Fig. 3.1). Biopsies were fixed in Bouin’s fixative and then transferred to 70 % alcohol prior to histological sectioning and staining. Two samples of each of the following tissues were also collected; spleen, peripheral lymph node (PLN) and mesenteric lymph node (MLN). One sample of each tissue was fixed in Bouin’s fixative. The other was frozen in OCT medium (Tissue-Tek, Indianapolis) in cryomolds (Tissue-Tek) in liquid nitrogen in case fixation of tissues in Bouin’s fixative adversely affected immunohistochemical staining. Frozen tissues were stored at -70°C prior to histological sectioning on a cryostat and staining.

Captive wombats experimentally infected with *S. scabiei*

Seven captive common wombats were experimentally infected with 1000 – 5000 *S. scabiei* var. *wombati* on their shoulders. The mean intensities of infection at 0 DAI and 77 DAI on the shoulder were 9 ± 1 and 1244 ± 458 mites/cm² (± standard error, n = 7), respectively. Four captive wombats were not infected and acted as controls (see Chapter 4.2). Skin biopsies, 7mm diameter, were taken during the course of the experiment (0, 1, 4, 12, 19, 34 and 77 DAI) from shoulders infected with mites (corresponding to sites 10 and 14 in Fig. 3.1) and fixed in Bouin’s fixative prior to histological sectioning and staining. Biopsy wounds were sutured with nylon suture material (Vetafil, Bengen, Germany).

*Staining of histological sections*

Histological sections were cut at a thickness of 4 µm from fixed tissues embedded in paraffin or from frozen tissues embedded in OCT medium. Histological sections of fixed skin biopsies were stained with haematoxylin and eosin to examine
the total number of cells and the percentage of polymorphonuclear cells in the dermal infiltrate. Histological sections of fixed skin biopsies were also stained with i) alcian blue, Tetrakis or toluidine blue to examine the number of mast cells in the dermal infiltrate (differentially stained cytoplasm of mast cells blue or purple, respectively) (Henwood, 1994; Lilee and Fulmer, 1976), ii) chromotrope 2R to examine the number of eosinophils (stained eosinophil granules bright red) (Appendix 5.1) and iii) methyl green - pyronin to determine the number of plasma cells (stained RNA in plasma cells pink) (Sheehan and Hrapchak, 1980). Positive tissue controls for these stains were, respectively, a mast cell tumour in a dog, eosinophilic dermatitis in a horse and an eosinophilic granuloma in a cat, and plasmacytic enteritis and chronic dermatitis in a dog. The negative tissue control was blood from captive common wombats in which no cells were stained apart from the occasional eosinophil.

Immunohistochemistry was used to examine the number of T and B lymphocytes in the dermal response. A number of primary antibodies, some of which have been shown to have interspecies reactivity, were tested initially to determine the most sensitive and specific.

**Immunohistochemistry**

Tissue sections on glass slides were labelled and circumscribed with a diamond edged marker to demarcate the area to be used for immunohistochemical study and to localise reagents to the defined area. Histological sections of paraffin embedded tissues on glass slides were then loaded into racks and the paraffin removed by immersion in xylene. The sections were rehydrated gradually using graded alcohols and “quenched” for endogenous peroxidase activity with 0.3 % hydrogen peroxide in phosphate buffered saline (PBS) for 10 minutes. Histological sections of
frozen tissues were fixed in acetone at 4 °C, and “quenched” for endogenous peroxidase activity with 0.3 % hydrogen peroxide for 10 minutes. Both fixed and frozen histological sections were then incubated with normal sheep serum at a 1:20 dilution with PBS to block non-specific binding sites of primary antibodies. Excess sheep serum was drained from the slide prior to application of the primary antibody. Polyclonal or monoclonal antibodies raised against intracytoplasmic portions of T and B cell receptors were used as primary antibodies for the identification of T and B cells in tissues (Table 5.1). Serial dilutions of these antibodies were used in order to determine the dilution that minimised background staining but differentially stained target cells (Table 5.1). Both the blocking agent and the primary antibody were applied with a 100 µl pipette until the entire tissue section was covered. The Dako Envision System (Dako, Carpinteria, California, USA) was then used to visualise cells to which primary antibodies had adhered (the cytoplasmic margins of immunopositive cells were brown). In the first step, horseradish peroxidase conjugates with a secondary antibody. The second step involves the application of 3,3′-diaminobenzidine (DAB) chromogen. Incubation times for the blocking agent and the primary and secondary antibodies were either 30 or 60 minutes at 24 °C and this was carried out at high humidity to prevent tissue sections from drying out. Tissue sections were washed twice in PBS for 5 minutes after primary and secondary antibody incubations. Rabbit immunoglobulin (Dako, Glostrup, Denmark) was used as a negative control. Sections were counterstained with haematoxylin in order to highlight the nuclei of cells.

*Cell counts*
Cells in the dermis immediately below the epidermal-dermal junction were counted with an ocular 10 x 10 square grid, 0.023 mm², at 400 x magnification using a compound microscope (Olympus, Tokyo, Japan). For each histological section the number of stained cells in a grid was counted. To increase the accuracy of the estimate of cellular density, if few stained cells were present, additional grids were counted until at least 10 stained cells had been counted in total. The thickness of the epidermis was also measured with the grid.

5.3. RESULTS

*Sensitivity and specificity of antibodies used in immunohistochemical staining*

Assuming that the histological distribution of lymphocytes in lymphoid tissue in wombats is typical of other marsupials, then excellent consistent differential staining of T lymphocytes occurred in histological sections of fixed spleen, PLN, MLN and skin with polyclonal antibodies anti-CD3 up to 1:1000 dilution and monoclonal antibodies anti-CD5 up to 1:500 dilution (Table 5.1). Monoclonal anti-CD3 also differentially stained T lymphocytes but less strongly and consistently at a 1:100 dilution. Consistent differential staining of B lymphocytes occurred with the monoclonal antibody anti-CD79b at a 1:10 dilution whereas poor differential staining of B lymphocytes occurred with the monoclonal antibody anti-HLA at a 1:10 dilution. Monoclonal anti-CD79b at a 1:25 dilution faintly stained cells in skin which had the appearance of macrophages or mast cells rather than B cells. Monoclonal antibodies anti-CD79a, anti-CD8, anti-CD68 and anti-BCL-2 and polyclonal anti-koala IgG did not differentially stain any cells in histological sections of fixed spleen, PLN, MLN and skin. The antibodies were also tested on histological sections of frozen tissues.
Polyclonal ant-CD3 stained T lymphocytes but less strongly than fixed tissues; monoclonal anti-HLA differentially stained B lymphocytes at a 1:100 dilution but the remainder of the monoclonal antibodies anti-CD5, anti-CD79a, anti-CD79b, anti-CD8, anti-CD68 and anti-BCL-2 and polyclonal antibodies anti-koala IgG did not differentially stain cells. However these antibodies were only tested on frozen tissues once. Rabbit immunoglobulin did not stain cells in fixed or frozen histological sections of spleen, PLN, MLN and skin. There was some background staining of connective tissues noted in most tissue sections but this did not affect the interpretation of staining of cells.

Based on the sensitivity and specificity of the polyclonal antibodies, anti-CD3 was used to stain T lymphocytes in the dermis of wombats with sarcoptic mange with rabbit immunoglobulin as a negative control. Monoclonal anti-CD79b was used to stain B lymphocytes in the dermis of wombats with sarcoptic mange with monoclonal anti-CD79a as a negative control.

Inflammatory response of wombats to S. scabiei

Free-living wombats with severe sarcoptic mange exhibited a significant inflammatory cellular response in their upper dermis when compared with normal wombats (Table 5.2). There were significantly more neutrophils, eosinophils, T lymphocytes, mast cells and total cells in the dermis of free-living wombats with severe sarcoptic mange compared with normal wombats (Table 5.2). A moderate percentage (29.7 ± 1.8 %) of the cellular immune response in the dermis was made up of mast cells, T lymphocytes, eosinophils and neutrophils with mast cells and T lymphocytes comprising 14.2 ± 1.2 % and 9.5 ± 0.9 % of the cells in the dermis,
respectively. Eosinophils and neutrophils were present at much lower percentages, 3.2 ± 0.8 % and 2.8 ± 0.6 %, respectively. Occasionally a plasma cell or B cell was present. The vast majority of cells in the response were not identified using differential stains but appeared to be cells of connective tissue such as fibroblasts. There were significant correlations between the number of mites in the epidermis and the total number of cells in the dermis (r = 0.70, n = 58), the number of T lymphocytes (r = 0.69, n = 58) and the number of eosinophils (r = 0.55, n = 58).

Captive wombats experimentally infected with *S. scabiei* also developed a significant inflammatory cellular response in the dermis compared with control wombats 77 DAI (Table 5.3). There were significant increases in the number of T lymphocytes, plasma cells and the total number of cells in the dermis of infected wombats compared with control wombats (Table 5.3). There was a significant increase in the number of mast cells in infected wombats but this change was not significant when compared with control wombats (Table 5.3). There was an increase in the number of neutrophils in infected wombats but this was not significant nor was the change significant when compared with the change in control wombats (Table 5.3). There was a slight increase in the number of eosinophils in infected wombats but it was not significant (Table 5.3). T lymphocytes, plasma cells, mast cells and neutrophils did not start to increase within the dermis until 12 DAI whereas the epidermis started to increase in thickness immediately (Fig. 5.1). Almost half (44.5 ± 2.0 %) of the inflammatory cellular response in captive wombats 77 DAI was composed of T lymphocytes (18.9 ± 2.2 %), mast cells (9.9 ± 1.9 %), plasma cells (8.9 ± 1.3 %) and neutrophils (6.8 ± 2.7 %). Eosinophils were absent. Again, the
majority of cells were not identified using differential stains but they were probably cells of connective tissue.

5.4. DISCUSSION

Immunohistochemical staining of tissues of the common wombat (V. ursinus) is similar to that of the koala, its closest relative, and other marsupials, the ringtail possum and the tammar wallaby, with monoclonal antibodies anti-CD5, anti-CD3 and anti-CD79b differentially staining cells (Hemsley et al., 1995). Similarly, the monoclonal antibody anti-CD79a did not stain cells and the polyclonal antibody anti-CD3 stained cells more strongly than monoclonal anti-CD3 (Hemsley et al., 1995). However, anti-koala IgG antibodies did not differentially stain wombat plasma cells although they stained plasma cells from koala, brushtail and ringtail possums and tammar wallabies. Although it is possible that there was some false negative and positive staining, these results support the conclusion that intracytoplasmic regions of some receptors are highly conserved among species (Jones et al., 1993; Wilkinson et al., 1995). Antibodies raised against external receptors such as anti-koala IgG and anti-HLA were less cross-reactive. Similarly, anti-human leucocyte common antigen did not stain lymphocytes of the southern hairy-nosed wombat (Byard et al., 1998).

The cellular immune response in the dermis of wombats exhibited typical aspects of an immune response to S. scabiei (Davis and Moon, 1990c). There was an induction phase for wombats experimentally infected with S. scabiei represented by the absence of a dermal inflammatory infiltrate for at least 12 DAI. The speed of development of an immune response is proportional to the density of mites (Davis and
Moon, 1990b). In contrast, the epidermis started to thicken the day after mites burrowed into it, suggesting that the burrowing of mites is responsible for epidermal stimulation and the increased production of keratinocytes. This has also been reported in rabbits (Arlian et al., 1994). T lymphocytes were present as a relatively large proportion of the dermal inflammatory response in both free-living wombats with severe sarcoptic mange and wombats experimentally infected with *S. scabiei*. They are a consistent feature of the dermal inflammatory response to *S. scabiei* and generally reflect the development of a type IV (delayed) hypersensitivity reaction (Arlian, 1996). The numbers of T lymphocytes in the dermis of wombats were correlated with the intensity of mites, suggesting that T lymphocytes responded to the numbers of mites or products of mites. Similarly, densities of lymphocytes were greater in older versus younger lesions in humans with scabies (Falk and Matre, 1982).

Mast cells were also present in relatively large numbers in wombats and have been reported to migrate to the dermis in pigs with crusted sarcoptic mange (Sheahan, 1975). In experimentally infected wombats, this may represent the beginning of a type I (immediate) hypersensitivity response, although eosinophils, another feature of type I hypersensitivity, were not present (Tizard, 2000). In free-living wombats, which had suffered from sarcoptic mange for an extended period, eosinophils were present together with mast cells in the dermal inflammatory response. The development of type I hypersensitivity follows that of type IV hypersensitivity in other animals (Davis and Moon, 1990c). In free-living wombats, the numbers of eosinophils in the dermis were correlated with the densities of mites suggesting that the numbers of eosinophils responded to the presence of mites or products of mites.
Neutrophils were present in the dermal inflammatory response of both free-living wombats with sarcoptic mange and wombats experimentally infected with *S. scabiei*. However, the increase in numbers of neutrophils in experimentally infected wombats was not significant due to the extensive variation in the number of neutrophils present in the dermis between individual wombats. This may be because there are other factors besides the presence of mites which determine the magnitude of the neutrophilic response such as the exposure of the dermis to bacteria due to excoriation (Skerratt, 1998). In free-living wombats, the numbers of neutrophils were not correlated with the intensity of mites. In addition, biopsy of the skin led to a relatively slight neutrophilic response in control wombats. Neutrophils constitute a greater proportion of the inflammatory response in secondary infections of *S. scabiei*, suggesting that there are factors other than the number of mites that control the response by neutrophils (Arlian, 1996). Arlian (1996) suggested that their oxidative burst is important in eliminating mites.

Plasma cells were present in experimentally infected wombats. However, they were not detected in free-living wombats with severe sarcoptic mange. B lymphocytes were also not found. This may represent the development of some tolerance to infection with *S. scabiei* in free-living wombats and a reduction in the humoral immune response (Davis and Moon, 1990c; Tizard, 2000). Desensitisation is the last in the sequence of phases that develop during the immune response to arthropod antigens of which exhaustion or suppression of antibody-producing cells is a feature (Davis and Moon, 1990c).
Comparison between intensities of the cellular infiltrates in different host species can not be made unless the extent of exposure of the hosts to mites is known. Therefore, studies should quantify the density of mites and duration of infection in the host to determine the extent of exposure (Davis and Moon, 1990b). The differences between varieties of *S. scabiei* in the type of immune response that they elicit must also be considered (Arlian et al., 1996b).

In conclusion, the immune response of wombats experimentally infected with *S. scabiei* is dominated by T lymphocytes, plasma cells, mast cells and neutrophils and appears to be similar to that reported for dogs (Arlian et al., 1996a; Arlian et al., 1997). This is a significant finding since resistance to secondary infections of *S. scabiei* has been identified in dogs and suggests that vaccination with an appropriate antigen from *S. scabiei* may provide some protection against infection with *S. scabiei* (Arlian et al., 1996a). It is different to the immediate hypersensitivity response which occurs initially in foxes and rabbits (Little et al., 1998; Arlian et al., 1994). Pigs also differ in that eosinophils are initially present in their inflammatory infiltrate in the dermis (Davis and Moon, 1990c). The immune response of free-living wombats with severe sarcoptic mange is typical of an immune system that has become desensitised to infection with *S. scabiei* (Davis and Moon, 1990c). However, desensitisation may also occur even though aspects of a hypersensitivity response are present (Davis and Moon, 1990c). It appears that once parakeratotic scale has built up sufficiently to prevent mites being removed by scratching the immune response is less effective at limiting the number of mites. The number of mites then increases dramatically and provides the immune system with a vast amount of antigen that probably results in desensitisation.
Table 5.1. Type, source, specificity and dilution of primary antibodies used in immunohistochemical staining of wombat tissues and whether antibodies differentially stained cells.

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Specificity</th>
<th>Dilutions&lt;sup&gt;d&lt;/sup&gt;</th>
<th>Immunopositive cells</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Polyclonal</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rabbit anti-human T cell, CD3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>T lymphocytes. Reacts with intracytoplasmic domain of CD3 epsilon chain (Mason et al., 1989).</td>
<td>1:10, 1:50, 1:100, <strong>1:500</strong>, 1:1000</td>
<td>+</td>
</tr>
<tr>
<td>Rabbit anti-koala IgG&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Plasma cells (Wilkinson et al., 1991).</td>
<td>1: 1000, 1:2000, 1:5000, 1:10,000, 1:20,000</td>
<td>-</td>
</tr>
<tr>
<td><strong>Monoclonal</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouse anti-human B cell, CD79a&lt;sup&gt;c&lt;/sup&gt;</td>
<td>B lymphocytes and plasma cells. Reacts with intracytoplasmic C-terminal part of mb-1 protein (Mason et al., 1992).</td>
<td>1:10, 1:50, 1:100</td>
<td>-</td>
</tr>
<tr>
<td>Mouse anti-human B cell, CD79b&lt;sup&gt;c&lt;/sup&gt;</td>
<td>B lymphocytes. Reacts with intracytoplasmic part of B29 protein (Mason et al., 1992).</td>
<td><strong>1:10</strong>, 1:50, 1:100</td>
<td>+</td>
</tr>
<tr>
<td>Mouse anti-human HLA-DP, DQ, DR&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Principally B lymphocytes, activated T lymphocytes and a variety of other cells. Reacts with beta chain of products of gene subregions DP, DQ and DR (Ghosh et al., 1984).</td>
<td>1:10, 1:50, <strong>1:100</strong></td>
<td>+</td>
</tr>
<tr>
<td>Mouse anti-human T cell, CD3&lt;sup&gt;c&lt;/sup&gt;</td>
<td>T lymphocytes. Reacts with intracytoplasmic domain of CD3 epsilon chain (Mason et al., 1989).</td>
<td><strong>1:10</strong>, 1:50, 1:100, 1:500</td>
<td>+</td>
</tr>
<tr>
<td>Mouse anti-human T cell, CD5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Principally T lymphocytes and small proportion of tissue B lymphocytes (Kroese et al., 1992).</td>
<td>1:10, 1:50, 1:100, <strong>1:500</strong></td>
<td>+</td>
</tr>
<tr>
<td>Mouse anti-human T cell, CD8&lt;sup&gt;c&lt;/sup&gt;</td>
<td>T lymphocytes.</td>
<td>1:10, 1:50, 1:100</td>
<td>-</td>
</tr>
<tr>
<td>Mouse anti-human CD68&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Macrophages.</td>
<td>1:10, 1:50, 1:100</td>
<td>-</td>
</tr>
<tr>
<td>Mouse anti-human BCL-2&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Lymphocytes. Reacts with protein produced by BCL-2 gene.</td>
<td>1:10, 1:50, 1:100</td>
<td>-</td>
</tr>
</tbody>
</table>

<sup>a</sup>Produced by Dako, Glostrup, Denmark.  
<sup>b</sup>Produced by Central Veterinary Laboratories, Department of Agriculture, Adelaide, South Australia, Australia.  
<sup>c</sup>Produced by LRF Immunodiagnosics Unit, Department of Clinical Biochemistry and Cellular Science, University of Oxford, Oxford, UK.  
<sup>d</sup>Serum dilutions were used in order to determine the dilution that minimised background staining but differentially stained cells (optimal dilution in bold).
Table 5.2. Mean numbers of mites and types of cells in the upper dermis of naturally infected free-living wombats with sarcoptic mange at sites Nos. 7-12 in Fig. 3.1 compared with normal wombats.

<table>
<thead>
<tr>
<th>Wombats</th>
<th>Mangy (n=10)</th>
<th>Normal (n=3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mites (/cm²)</td>
<td>1540* ± 270</td>
<td>0</td>
</tr>
<tr>
<td>Cells (/mm²)</td>
<td>5230* ± 270</td>
<td>2450 ± 160</td>
</tr>
<tr>
<td>Neutrophils (/mm³)</td>
<td>170*± 43</td>
<td>5 ± 5</td>
</tr>
<tr>
<td>Eosinophils (/mm³)</td>
<td>239* ± 65</td>
<td>0</td>
</tr>
<tr>
<td>T cells (/mm³)</td>
<td>604* ± 75</td>
<td>291 ± 69</td>
</tr>
<tr>
<td>Mast cells (/mm³)</td>
<td>640* ± 63</td>
<td>338 ± 28</td>
</tr>
</tbody>
</table>

* Indicates significant difference ($P<0.05$, Student t-test).

Plasma cells were not found in the dermis.
Table 5.3. Numbers of various cell types per mm$^2$ in the upper dermis and thickness of the epidermis (µm) in wombats experimentally infected with *Sarcoptes scabiei* var. *wombati* and in control wombats on days 0 and 77 after infection.

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Infected (n=7)</th>
<th>Controls (n=4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 0</td>
<td>Day 77</td>
</tr>
<tr>
<td>Neutrophils (/mm$^2$)</td>
<td>0</td>
<td>742 ± 439</td>
</tr>
<tr>
<td>Eosinophils (/mm$^2$)</td>
<td>0</td>
<td>6 ± 6</td>
</tr>
<tr>
<td>T Lymphocytes (/mm$^2$)</td>
<td>120$^a$ ± 30</td>
<td>1080$^a$ ± 251</td>
</tr>
<tr>
<td>Mast Cells (/mm$^2$)</td>
<td>216$^a$ ± 23</td>
<td>502$^a$ ± 95</td>
</tr>
<tr>
<td>Plasma Cells(/mm$^2$)</td>
<td>0$^a$</td>
<td>400$^a$ ± 50</td>
</tr>
<tr>
<td>Total Cells (/mm$^2$)</td>
<td>1619$^a$ ± 127</td>
<td>7149$^a$ ± 1660</td>
</tr>
<tr>
<td>Thickness of Epidermis (µm)</td>
<td>34$^a$ ± 3</td>
<td>321$^a$ ± 53</td>
</tr>
</tbody>
</table>

The numbers of cells at day 0 and day 77 were compared for both infected and control wombats. The change in the number of cells between day 0 and day 77 for infected and control wombats were compared. Numbers sharing a superscript letter in the same row were significantly different ($P<0.05$).
Figure 5.1. Changes in the percentage of cell types in the cellular infiltrate, which occurred in the upper dermis of wombats experimentally infected with *Sarcoptes scabiei* var. *wombati*, 77 days after infection.
Figure 5.2. Change in epidermal thickness (µm) with time in wombats (n=7) experimentally infected with 1000 to 5000 *Sarcoptes scabiei* var. *wombati*.

Epidermal thickness did not change in control wombats (Table 5.3). Error bars show 95.0% confidence interval of means.
CHAPTER 6

POPULATION DYNAMICS OF SARCOPTES SCABIEI VAR. WOMBATI

6.1. INTRODUCTION

The dynamics of animal populations including mites has been extensively researched. However little has been done to understand the dynamics of parasitic mites on mammals. Preliminary work on the sheep itch mite (*Psoroptes ovis*) used a simple model, the Leslie Matrix Model (also referred to as the Bernardelli-Leslie-Lewis Model), to predict the change in structure of populations with time (Manly, 1990; Wall et al., 1999). The model is composed of a column vector that describes the initial population structure, such as the number of each life-cycle stage, and a matrix that describes the population dynamics of the different life-cycle stages. Multiplication of the vector and the matrix determines population growth. The model’s accuracy was tested with experimental infections (Wall et al., 1999). A similar approach is used here with *S. scabiei* var. *wombati*.

6.2. MATERIALS, METHODS AND RESULTS

Population Coefficients from Steady State Data.

1. Introduction

A complete dynamic population model can only be obtained from time series data (Manly, 1990). The information that can be obtained if only static data is
available is considered here. Using the life-cycle of *Sarcoptes scabiei*, consider the relationship between the number of eggs at time interval (n-1), $E_{n-1}$, and the resulting larva at the next time interval n, $L_n$. One of two following difference equations may be applicable depending on the duration of the time interval.

(1) \[ L_n = \beta E_{n-1} \]

(2) \[ L_n = \alpha L_{n-1} + \beta E_{n-1} \]

where $\beta$ = survival rate

$\alpha$ = proportion of previous generation that still exist as larvae

The first equation (1) assumes that the only surviving larva have hatched within the time interval with survival rate $\beta$ whereas the second equation assumes that $\alpha$ of the previous generation still exist as larvae.

If the populations of eggs and larvae are assumed to have converged to a steady-state equilibrium, $E_{n-1} \rightarrow E_n \rightarrow E^*$ and $L_{n-1} \rightarrow L_n \rightarrow L^*$ then measurement of these populations will enable the following calculations:

If (1) is applicable then

(3) \[ \beta = \frac{L^*}{E^*} \]

and population counts of larvae can be regressed against population counts of eggs to determine $\beta$. If, however, (2) is applicable then

(4) \[ L^* = \alpha L^* + \beta E^* \]

:. \[ L^* = \frac{\beta}{1-\alpha} E^* \]

\[ \frac{L^*}{E^*} = \frac{\beta}{1-\alpha} \]

and thus regression will only estimate the combination of model parameters, $\beta/(1-\alpha)$. Additional information is required to identify the two population parameters.
Biological constraints and the ratio of the populations will sometimes indicate which of the two models is appropriate.

2. Leslie Projection Matrix, M

Given a vector of the population of the mite stages (nymphs includes the mite stages protonymphs and tritonymphs and adult males are not considered)

\[
\begin{align*}
\nu &= \begin{bmatrix}
\text{eggs}(E) \\
\text{larva}(L) \\
\text{nymphs}(N) \\
\text{females}(F)
\end{bmatrix} \\
M &= \begin{bmatrix}
0 & 0 & 0 & \beta_{ef} \\
\beta_{le} & 0 & 0 & 0 \\
0 & \beta_{nl} & 0 & 0 \\
0 & 0 & \beta_{jn} & 0
\end{bmatrix}
\end{align*}
\]

A Leslie projection matrix M can be defined to generate the population number trajectory for the assumed time interval duration

\[
\nu_n = M\nu_{n-1}
\]

The simplest Leslie matrix is obtained by using (1) to define all transition dynamics. Hence

\[
\lambda^\nu \nu^* (\lambda \text{ is a scalar defined below}) \text{ which satisfies the equation }
\]

\[
M\nu^* = \lambda \nu^*
\]

where \(\lambda\) may be interpreted as the asymptotic rate of increase per time interval. The relationship of population growth to the magnitude of \(\lambda\) is given as follows.
Replacing $v^*$ in (8) by the measured population values as in (5) and assuming the population to have converged to an asymptotic trajectory, the elements of $M$ can be solved from (8) thus

\[ \beta_{cf} = \lambda \frac{E_*}{F_*}; \quad \beta_{tc} = \lambda \frac{L_*}{E_*}; \quad \beta_{al} = \lambda \frac{N_*}{L_*}; \quad \beta_{fn} = \lambda \frac{F_*}{N_*} \]

Note that these constants are not independent. In the steady state they are constrained by the consistancy relationship

\[ \beta_{cf} \beta_{tc} \beta_{al} \beta_{fn} = \lambda^4 \]

The ratios in (9) were evaluated by regression analysis on data taken from the 10 wombats with severe sarcoptic mange described in Chapter 3. The populations of mites on these wombats were assumed to have reached a steady state since wombats appeared to have been infected for a long duration, at least 3 to 6 months. The unconstrained and constrained regression coefficients are given in Fig. 6.1 (see Appendix 6.1 for their method of calculation). The fact that the constrained coefficients are close to the unconstrained coefficients (within the 0.7 significance bounds as indicated by the variance of the estimates) indicate that the asymptotic assumption is valid (Fig 6.1). The program Matlab for Windows, version 4.2c.1 (The Mathworks, Inc.), was used to run matrix models.
The simple Leslie model can be rejected for two reasons:

1. The structure of the matrix results in violent oscillations of the trajectory.
   (the eigenvalues are of the form $\lambda[1, -j, +j, -1]$)

2. The regression coefficient $\beta_{le}=2.28$. This defines the implausible relationship
   for $\lambda=1$ (steady state population)

$$L_n=2.28E_{n-1}$$  \hspace{1cm} (11)

Equation 2 has therefore been used to modify the second row of the Leslie matrix.

$$M = \begin{bmatrix} 0 & 0 & 0 & \beta_{ef} \\ \beta_{le} & \alpha & 0 & 0 \\ 0 & \beta_{ml} & 0 & 0 \\ 0 & 0 & \beta_{pe} & 0 \end{bmatrix}$$  \hspace{1cm} (12)

Using (8) as before to evaluate the matrix constants, the $\beta$ element in the second row now becomes

$$\beta_{le} = (\lambda - \alpha) \frac{L^*}{E^*}$$  \hspace{1cm} (13)

where both the rate of increase, $\lambda$, and $\alpha_l$ must be specified.

$\alpha$ estimates the proportion of each life-cycle stage that remain, i.e. that do not
hatch nor moult to the next stage or die in the case of adult females, for a given time
interval. It reflects the innate biology of the parasite. $\beta$ estimates the number of each
life-cycle stage that actually survive to the next stage for a given time interval and is a
measure of survivorship. It does not include migration and mites that emigrate are
regarded as dead. In the case of $\beta_{ef}$, it estimates the rate of egg production by females.

$\alpha_l$ can be determined from the duration of the larval life-cycle stage, which has been
determined for a few varieties of $S. scabiei$ (Arlian, 1989; Burgess, 1994). Therefore
the value of $\alpha$ for $S. scabiei$ var. wombat can be estimated ($\alpha = 1 - \text{time interval}$)
duration of life-cycle stage) assuming that the duration of the larval life-cycle stage of var. *wombati* is similar to var. *canis*, a variety that is morphologically and genetically similar (Chapter 6). The mean duration for the larval life-cycle stage of var. *canis* is 3.7 days (Arlian and Vyszenski-Moher, 1988). Therefore, for a 2 day time interval, only approximately half of the larvae will mature to nymphs and $\alpha_t = 0.54$. The $\alpha$ values for other life-cycle stages can also be estimated providing a new Leslie Matrix.

\[
M = \begin{bmatrix}
\alpha & 0 & 0 & \beta_{ef} \\
\beta_{le} & \alpha & 0 & 0 \\
0 & \beta_{nl} & \alpha_n & 0 \\
0 & 0 & \beta_{fn} & \alpha_f 
\end{bmatrix}
\]

Given that the duration of egg and nymphal stages of *S. scabiei* var. *canis* are 52 hr and 5.6 days, respectively (Arlian and Vyszenski-Moher, 1988), then for a 2 day interval $\alpha_e = 0.08$ and $\alpha_n = 0.64$. The duration of the adult, female life-cycle stage was not determined for *S. scabiei* var. *canis* but the adult females of *S. scabiei* var. *hominis* may survive for 4 to 6 weeks (Burgess, 1994). The estimate of 40 days provided by Mellanby (1944) is used here, hence $\alpha_f = 0.95$.

The value of $\beta$ for a 2 day time interval can now be recalculated for each life-cycle stage using the formulae 13 and 4, $\lambda=1$.

\[
\begin{align*}
\beta_{le} &= 1.00, \\
\beta_{nl} &= 0.134, \\
\beta_{fn} &= 0.031, \\
\beta_{ef} &= 1.86.
\end{align*}
\]

The new Leslie Matrix Model can be tested against *S. scabiei* population growth that occurred in experimental infections of captive and free-living wombats.
The numbers of mites on experimentally infected wombats were estimated by counting a proportion of mites obtained from skin scrapings and biopsies (see Chapters 4 and 7 for methods). Figures 6.2 to 6.4 show population trajectories of mites using the Leslie Matrix Model and the number of mites used to infect each wombat in experimental infections. Values of $\lambda$ were adjusted so that population growth in the trajectories was similar to that of larvae in experimental infections since larvae were the most numerous life-cycle stage and their estimate was more likely to be accurate (Figs 6.2 to 6.4) (Clark et al., 1971). The Leslie Matrix Model gave a reasonably accurate prediction of the average number of each life-cycle stage, apart from nymphs, of S. scabiei present on four wombats, Infected 3, 4, 6 and Reinfected 1, at 78 days after infection (DAI) when $\lambda=1.2$ (Fig. 6.2). Nymphs were over-estimated by a factor of 2. The model was less accurate at predicting the average number of each life-cycle stage when $\lambda=1.1$ at 78 DAI for two wombats, Infected 5 and Reinfected 2, (Fig. 6.3). Eggs, nymphs and females were under-estimated by factors of 6, 2 and 2, respectively. The model was also less accurate at predicting the average number of each life-cycle stage after a shorter period of population growth, at 48 DAI when $\lambda=1.2$ for infected wombats 3, 4, 6 and Reinfected 1 (Fig. 6.2) and at 40 DAI when $\lambda=1.35$ for one wombat, Infected 7 (Fig. 6.4). However, the model gave a reasonably accurate prediction of the average number of each life-cycle stage, apart from larvae and females, at 48 DAI when $\lambda=1.1$ for infected wombats 5 and Reinfected 2 (Fig. 6.3).

6.3. DISCUSSION
Although the Leslie Matrix Model of population dynamics of *S. scabiei* var. *wombati* is simplistic (Manly, 1990) it gave reasonable predictions of population dynamics if growth rates were relatively high, $\lambda \geq 1.2$, and duration of infection was relatively long, $\geq 78$ days. The model may give less accurate predictions when growth rates are lower or duration of infection is shorter. The imprecision of estimates of the number of each life cycle stage based on counting a proportion of the population must also be considered when assessing the accuracy of predictions. This imprecision was not determined in the present study but was kept to a minimum by counting at least 10 mites before calculating an estimate (Clark et al., 1971). Estimates for the life-cycle stages “adult female” and “nymphs” were relatively imprecise because sometimes less than 5 of each were counted to determine an estimate. Manly (1990) suggested incorporating an error term into the Leslie Matrix Model that accounted for both stochastic population variation and sampling errors.

$\beta$ values in the Leslie Matrix Model of the population dynamics of *S. scabiei* var. *wombati* suggest that birth rates are less than 1 egg per female per day. This is less than the 1 to 2 eggs per day per female reported for other varieties of *S. scabiei* (Arlian 1989; Burgess 1994) and is probably because the $\beta_{ef}$ value in the Leslie Matrix Model includes all females even females that were reproductively immature. Nymphs were the life-cycle stage least likely to survive to the next stage even allowing for the fact that this stage has a longer duration than the larval and egg stages and that the $\beta_{fn}$ value does not include nymphs that survive to become adult males (survivorship of nymphs for a two day interval = $4.2\beta_{fn}$, given that the nympha-stage is 5.6 days and 0.33 become males) (Chapter 3). The poor survivorship of nymphs is probably due to the two molts that occur in this stage, protonymph to tritonymph to
adult. The egg was most likely to survive to the next stage. The $\beta_{le}$ value suggested that all eggs hatched within 2 days whereas the mean time for var. *canis* eggs hatching was 52 hours. The $\beta_{le}$ appears to be an overestimate given that it is unlikely that all eggs would hatch. It is possible that this overestimate could be due to immigration of larvae from areas that were not sampled to areas that were sampled.
Figure 6.1. Constrained regressions of the life-cycle stages of *Sarcoptes scabiei* var. *wombati* (blue line excludes outliers).

Constrained and unconstrained regression coefficients of life-cycle stages of *Sarcoptes scabiei* var. *wombati* listed below:

<table>
<thead>
<tr>
<th></th>
<th>eggs/females</th>
<th>larva/eggs</th>
<th>nymphs/larva</th>
<th>females/nymphs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constrained coefficients</td>
<td>2.02</td>
<td>2.28</td>
<td>0.372</td>
<td>0.612</td>
</tr>
<tr>
<td>Unconstrained coefficients</td>
<td>2.4</td>
<td>2.11</td>
<td>0.175</td>
<td>0.78</td>
</tr>
<tr>
<td>Variances on unconstrained coefficients</td>
<td>0.223</td>
<td>0.16</td>
<td>0.034</td>
<td>0.144</td>
</tr>
</tbody>
</table>
Figure 6.2. Population trajectory for density of *Sarcoptes scabiei* on wombats when 6 larvae, 2 nymphs and a female occurred per cm² on 0 days after infection and $\lambda=1.2$ for a 2 day time interval.

Below is a comparison of the model’s prediction and the density of each life-cycle stage excluding males on captive wombats, “Infected 3, 4 and 6 and Reinfected 1”, experimentally infected with *Sarcoptes scabiei* var. *wombati*.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>DAI</th>
<th>Larvae</th>
<th>Eggs</th>
<th>Nymphs</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>I3, I4, I6, RI1</td>
<td>0</td>
<td>6</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>I3</td>
<td>49</td>
<td>42</td>
<td>60</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>I4</td>
<td>49</td>
<td>220</td>
<td>150</td>
<td>70</td>
<td>30</td>
</tr>
<tr>
<td>I6</td>
<td>49</td>
<td>72</td>
<td>24</td>
<td>72</td>
<td>0</td>
</tr>
<tr>
<td>RI1</td>
<td>49</td>
<td>12</td>
<td>36</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Mean of I3, I4, I6, RI1</td>
<td>49</td>
<td>87</td>
<td>68</td>
<td>42</td>
<td>9</td>
</tr>
<tr>
<td>Model’s prediction</td>
<td>48</td>
<td>352</td>
<td>162</td>
<td>131</td>
<td>80</td>
</tr>
<tr>
<td>I3</td>
<td>77</td>
<td>4400</td>
<td>2000</td>
<td>400</td>
<td>800</td>
</tr>
<tr>
<td>I4</td>
<td>77</td>
<td>9200</td>
<td>4800</td>
<td>400</td>
<td>1600</td>
</tr>
<tr>
<td>I6</td>
<td>77</td>
<td>7600</td>
<td>2000</td>
<td>2000</td>
<td>800</td>
</tr>
<tr>
<td>RI1</td>
<td>77</td>
<td>2600</td>
<td>1800</td>
<td>1400</td>
<td>400</td>
</tr>
<tr>
<td>Mean of I3, I4, I6, RI1</td>
<td>77</td>
<td>5950</td>
<td>2650</td>
<td>1050</td>
<td>900</td>
</tr>
<tr>
<td>Model’s prediction</td>
<td>78</td>
<td>5421</td>
<td>2489</td>
<td>2016</td>
<td>1233</td>
</tr>
</tbody>
</table>

I Infected, RI reinfected (see Chapter 4).

DAI Days after infection.
Figure 6.3. Population trajectory for density of *Sarcoptes scabiei* on wombats when 6 larvae, 2 nymphs and a female occurred per cm$^2$ on 0 days after infection and $\lambda=1.1$ for a 2 day time interval.

Below is a comparison of the model’s prediction and the density of each life-cycle stage excluding males for captive wombats, “Infected 5 and Reinfected 2”, experimentally infected with *Sarcoptes scabiei* var. *wombati*.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>DAI</th>
<th>Density (/cm$^2$) of life-cycle stages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Larvae</td>
</tr>
<tr>
<td>I5, RI2</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>I5</td>
<td>49</td>
<td>32</td>
</tr>
<tr>
<td>RI2</td>
<td>49</td>
<td>8</td>
</tr>
<tr>
<td>Mean of I5, RI2</td>
<td>49</td>
<td>20</td>
</tr>
<tr>
<td>Model’s prediction</td>
<td>48</td>
<td>44</td>
</tr>
<tr>
<td>I5</td>
<td>77</td>
<td>100</td>
</tr>
<tr>
<td>RI2</td>
<td>77</td>
<td>240</td>
</tr>
<tr>
<td>Mean of I5, RI2</td>
<td>77</td>
<td>170</td>
</tr>
<tr>
<td>Model’s prediction</td>
<td>78</td>
<td>183</td>
</tr>
</tbody>
</table>

† Infected, RI reinfected (see Chapter 4).

DAI Days after infection.
Figure 6.4. Population trajectory for density of *Sarcoptes scabiei* on wombats when 6 larvae, 2 nymphs and a female occurred per cm$^2$ on 0 days after infection and $\lambda=1.35$ for a 2 day time interval.

Below is a comparison of the model’s prediction and the density of each life-cycle stage excluding males for a captive wombat, “Infected 7”, experimentally infected with *Sarcoptes scabiei* var. *wombati*.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>DAI</th>
<th>Larvae</th>
<th>Eggs</th>
<th>Nymphs</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>I7</td>
<td>0</td>
<td>6</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>I7</td>
<td>41</td>
<td>1920</td>
<td>480</td>
<td>400</td>
<td>80</td>
</tr>
<tr>
<td>Model’s prediction</td>
<td>40</td>
<td>1770</td>
<td>813</td>
<td>658</td>
<td>403</td>
</tr>
</tbody>
</table>

I Infected (see Chapter 4).  
DAI Days after infection.
CHAPTER 7

VARIATION IN THE MITOCHONDRIAL SMALL SUBUNIT 12S RIBOSOMAL RNA GENE OF SARCOPTES SCABIEI FROM WOMBAT, DOG AND HUMAN

7.1. INTRODUCTION

During the course of this study scabies occurred in three people who handled dead common wombats affected with severe hyperkeratotic sarcoptic mange. Transmission of scabies originally acquired from a common wombat from person-to-person also occurred. However, anecdotal evidence from these infections suggested that Sarcoptes scabiei var. wombati behaves differently in humans compared to S. scabiei var. hominis in that infections appear to be self-limiting, mites are difficult to recover and clinical signs differ (Observations on these zoonotic infections were published in the Australian Veterinary Journal (Skerratt and Beveridge, 1999) and a copy of the paper is included in the Appendices). There are also morphological differences that exist between the taxa (Fain, 1968; Zahler et al., 1998). However there is little genetic evidence for the separation of these taxa (Walton et al., 1999a). More importantly there is little genetic evidence for the differentiation of var. wombati from var. canis. This has important consequences for understanding the epidemiology of sarcoptic mange in sympatric wombat, dog and fox populations. Since wombats are sedentary, it is thought that dogs and foxes, which are more mobile and often enter wombat burrows, may be important in the introduction of sarcoptic mange into wombat populations (Martin et al., 1998; Skerratt et al., 1998;
Chapter 1; Chapter 2). However this hypothesis can not be tested until there is some way of separating the taxa. In contrast to human mites, mites derived from wombats cannot be distinguished morphologically from mites from dogs and foxes and there is no evidence that the taxa behave differently (Fain, 1968).

Recently, DNA fingerprinting has allowed the differentiation of a population of *S. scabiei* from a wombat from those originating from dogs and humans (Walton et al., 1999a). However, sequencing of the second internal transcribed spacer of ribosomal RNA (rRNA) did not differentiate mites of different host origin or geographical location (Zahler et al., 1999). Mitochondrial DNA has provided useful population markers for a range of species of Acari such as tetranychid and orbátid mites and ixodid ticks, and could also be applicable to *S. scabiei* (Navajas et al., 1994; Salomone et al., 1996; Norris et al., 1997; Crosbie et al., 1998; Navajas et al., 1998). In the present study, a portion of the mitochondrial small subunit 12S rRNA gene sequence for *S. scabiei* was characterised from wombat, dog and human hosts and its usefulness in detecting differentiation among populations of *S. scabiei* was demonstrated.

7.2. MATERIALS AND METHODS

Mites were collected from humans in Darwin and a dog in an Aboriginal household in Maningrida in Arnhem Land in the Northern Territory, Australia. Mites were collected from common wombats (*Vombatus ursinus*) from five localities in Victoria, Australia: Bullengarook (37°31′ S, 144°29′ E), Eden Park (37°30′ S, 145°04′ E), Christmas Hills (37°39′ S, 145°19′ E), Chum Creek (37°36′ S, 145°29′ E),
Toolangi (37°32′ S, 145°29′ E) and Longwarry North (38°05′ S, 145°47′ E). To obtain mites from wombats, skin scrapings from hosts were placed in Petri dishes in a metal tray on a magnetic stirrer. The vibrations stimulated mites to move away from skin debris (Sheahan and Hatch, 1975). Petri dishes were examined with a stereomicroscope and mites were picked up with a metal pointer and were stored individually in 1.5 ml tubes at –70°C. Mites from humans and dogs were collected as previously described (Walton et al., 1999a).

DNA was extracted from mites by adding 10 µl of water to the tubes, centrifuging the tubes to ensure mites were at the bottom, briefly placing the tube into liquid nitrogen to freeze the mite and water, and then crushing the mite and ice with a pestle. Then 100 µl of water containing 5% chelex beads (Biorad) were added to each tube. DNA was eluted by heating the chelex solution to 97°C for 10 min. A DNA fragment corresponding to most of the third domain of the mitochondrial small subunit 12S rRNA gene was amplified by polymerase chain reaction (PCR) with the primers SR-J-14199 (forward: 5’-TACTATGTTACGACTTAT-3’) and SR-N-14594 (reverse: 5’-AAACTAGGATTAGATACCA-3’). The names of the primers refer to the site at which they anneal by gene, strand and 3’ base number relative to the Drosophila yakuba sequence (Simon et al., 1994). Elongase (GIBCO: BRL) was used in PCR, as recommended by the manufacturer, with the following cycle conditions: 94°C for 1 min; 35 cycles of 30 s at 92°C, 30 s at 40°C and 1 min at 68°C plus an additional 7 min at 68°C. Control samples without DNA were included in each PCR run. Three microlitres of each PCR product were examined on ethidium bromide-stained 1% agarose-TAE gels. If insufficient PCR product was produced for nucleotide sequencing, a nested PCR reaction was performed using 1 µl of PCR
product and the internal primers SR-J-14233 (forward: 5’-
AAGAGCGACGGGCGATGTGT-3’) and SR-N-14588 (reverse: 5’-
AAACTAGGATTAGATCCCTATTAT-3’) again designed from the 12S sequence
for *D. yakuba*. Both strands were sequenced directly from the PCR products with the
same primers as used for amplification. PCR fragments were purified with Qiaquick
columns (QIAGEN); 100-200 ng of template was used in each cycle-sequencing
reaction (DyeDeoxy terminator; PE – Applied Biosystems). An automatic sequencer
(ABI 373A) resolved the sequencing fragments, and sequences were aligned by eye.
To check that a DNA fragment corresponding to the third domain of the
mitochondrial 12S rRNA gene of *S. scabiei* was being amplified by PCR, sequences
were searched for conserved motifs that are found in this region of animal 12S rRNA
genes (see Hickson et al., 1996, 2000).

7.3. RESULTS

Sequences were readily determined and there was no evidence of
polymorphism within individual sequences suggesting that they were not derived
from pseudogenes (Zhang and Hewitt, 1996). Eight conserved motifs were found
corresponding to the reverse complement of motifs Nos. 2 - 9 reported by Hickson et
al. (2000). Four motifs matched exactly those of Hickson et al. (2000), four had a
different nucleotide at one base position and one had different nucleotides at two base
positions. In total, 51 of 57 base positions in motifs matched those reported by
Hickson et al. (2000). Motifs were in the same order and with a similar number of
nucleotides between motifs as reported by Hickson et al. (2000), with a mean
minimum difference of four, and a range of 0 – eight nucleotides. This suggests that
the sequences were from the 12S region (Hickson et al., 1996). Genbank was searched for sequence similarities. The only significant sequence alignments in arthropods were with the 12S small subunit rRNA gene of the beetles *Echoma anaglypta* and *Eugenysa coscaroni*. There were no sequences from mites in Genbank for the 12S small subunit rRNA gene region. Significant alignments occurred in organisms other than arthropods such as *Drosophila* sp., but not in the 12S genomic region. However, the sequence alignment programs used in Genbank do not consider secondary-structure features and conserved motifs and hence may produce poor alignments (Hickson et al., 2000). Control samples containing no DNA and host DNA did not produce a DNA fragment when amplified by PCR. The above findings, together with the fact that the fragment amplified was of the expected size (380bp), appears to be strong evidence in favour of the assumption that the fragment amplified was the portion of the 12S gene targeted.

Mitochondrial 12S rRNA gene sequences were obtained from 23 *S. scabiei* individuals taken from eight wombats, a dog and three humans. Sequences were 326 base pairs in length with 82% A + T content. Nine distinct 12S sequences (haplotypes) were defined among the 23 mites examined (Table 7.1). The most common sequence (10/23; Haplotype 1, Table 7.1) was found in 10 mites taken from seven wombats from four separate localities in Victoria. The next most common sequence (5/23; type 4, Table 7.1) was found in three mites from a dog from Maningrida in the Northern Territory and two mites from a human from Darwin in the Northern Territory. Six other haplotypes (types 2, 3, 6-9, Table 7.1) were only found in individual mites: two from wombats from different localities in Victoria and four from two humans from Darwin in the Northern Territory. One other distinct sequence
(Haplotype 5, Table 7.1) was present in two mites from a dog from Maningrida in the Northern Territory. All haplotypes were similar with only 1 – 8 bp (0.3 – 2.7 %) difference upon pairwise comparison. The most common sequence (Haplotype 1), found only on wombats in Victoria, was only one base pair different from the second most common sequence (Haplotype 4) found on both dogs and humans from the Northern Territory. The largest sequence difference, 8 bp, occurred between a mite from a wombat from Victoria (Haplotype 2) and a mite from a human from the Northern Territory (Haplotype 6). Comparison among all sequences revealed 12 variable positions. Transitions occurred at two of these positions, transversions at seven of the positions and base insertion or deletion at the remaining three positions.

The relationships among the 9 haplotypes were analysed using a Maximum Parsimony (MP) approach in PAUP* (ver 4.0b4a; Swofford, 2000) using a Branch and Bound search based on all differences (including indels). This search is guaranteed to find all the most parsimonious trees. We assessed the robustness of the relationships inferred by 1000 bootstrap replicates (Felsenstein, 1985). We also tested for heterogeneity in the frequencies of the 12S haplotypes among the 3 host groups using Roff and Bentzen's (1989) procedure, as employed in the program REAP (McElroy et al., 1992).

Ten equally most parsimonious trees were identified, one of which is presented as an unrooted network in Fig. 7.1. The only branch present in all MP trees was also the only one to receive majority bootstrap support (82%). This branch divided the mites into two groups: one comprised mites from wombats, dogs and humans and the other comprised mites from dogs and humans. Thus there is no significant phylogenetic divergence among mites from different hosts or localities.
There is, however, a striking pattern in terms of the distribution of the 12S haplotypes among the 3 host groups: while some mites from dogs (3) and humans (2) from the NT share one of the distinct 12S haplotypes (Haplotype 4), mites from wombats from Vic. (n = 13) do not share any haplotypes with the other two groups (n = 10). This pattern implies highly significant genetic differentiation among these populations ($P < 0.0001$).

7.4. DISCUSSION

Fixed genetic differences did not occur between the 12S small subunit rRNA genes of *S. scabiei* from different hosts or localities. The lack of morphological or fixed genetic sequence differences among individual *S. scabiei* from different hosts has been reported previously (Fain, 1968; Pence, 1975; Zahler et al., 1999; Walton et al., 1999a). It therefore appears that populations of mites from different hosts are closely related and is consistent with the view that the mites from wombats have been introduced recently to Australia (Skerratt et al., 1998). This is important with regard to conservation of wombats, since it may be that the introduction of *S. scabiei* into naïve populations has resulted in epizootics with resultant reduction or extinction of local populations (Gray, 1937; Martin et al., 1998; Skerratt et al., 1998). Similar declines have occurred in other animals including foxes (*Vulpes vulpes*) in Scandinavia and Spanish ibex (*Capra pyrenaica*) in Spain (Mörner, 1992; Lindström, 1991; León-Vizcaíno et al., 1999).

The variation in 12S haplotypes enabled all wombat mites to be differentiated from dog and human mites (Fig. 7.1). Since mites were sampled from few hosts it is
possible that the differences that were observed between mites from different hosts were due to sampling bias. However, microsatellite data also differentiated wombat mites from both dog and human mites (Walton et al., 1999a). Although the wombat mites originated from a different locality to the dog and human mites, Walton et al. (1999a) found that origin was less important than host as a determinant of genetic identity. Validating this for wombat mites has not been possible due to the difficulty in collecting mites from both wombats and species sympatric with wombats such as foxes. Irrespective of this obstacle, the mitochondrial 12S rRNA gene may be useful as a population genetic marker. Further comparison between this study and that of Walton et al. (1999a) suggests that microsatellites may be preferable to the mitochondrial 12S rRNA gene in differentiating between mites from different host species. Sympatric populations of dog and human mites have been shown to be genetically distinct using microsatellites (Walton et al., 1999a) but could not be differentiated based on 12S rRNA sequence variation or haplotype frequencies. Further sequence analysis such as full sequencing of the 12S gene and larger sample sizes may enable their differentiation. A larger study consisting of mites from a number of individuals from different host species from allopatric and sympatric populations would be informative but was not immediately possible due to the lack of readily available live or frozen *S. scabiei* from different hosts and localities.
Table 7.1. Variable sites in the coding region of the mitochondrial 12S rRNA gene of *Sarcoptes scabiei*.

Sites are numbered according to their position in the 326 bp alignment of sequences. A dot indicates identity with the reference sequence in row 1 (Haplotype 1); a dash indicates an alignment gap; and a letter represents a base substitution. All isolates from Australia.

<table>
<thead>
<tr>
<th>Mite</th>
<th>Host</th>
<th>Location</th>
<th>Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>W1.1</td>
<td>Wombat 1</td>
<td>Toolangi, Victoria</td>
<td>13</td>
</tr>
<tr>
<td>W1.2</td>
<td>Wombat 1</td>
<td>Toolangi, Victoria</td>
<td>16</td>
</tr>
<tr>
<td>W1.3</td>
<td>Wombat 1</td>
<td>Toolangi, Victoria</td>
<td>19</td>
</tr>
<tr>
<td>W1.4</td>
<td>Wombat 1</td>
<td>Toolangi, Victoria</td>
<td>47</td>
</tr>
<tr>
<td>W1.5</td>
<td>Wombat 1</td>
<td>Toolangi, Victoria</td>
<td>59</td>
</tr>
<tr>
<td>W2.1</td>
<td>Wombat 2</td>
<td>Chum Creek, Victoria</td>
<td>106</td>
</tr>
<tr>
<td>W2.2</td>
<td>Wombat 2</td>
<td>Chum Creek, Victoria</td>
<td>123</td>
</tr>
<tr>
<td>W3.1</td>
<td>Wombat 3</td>
<td>Chum Creek, Victoria</td>
<td>171</td>
</tr>
<tr>
<td>W4.1</td>
<td>Wombat 4</td>
<td>Longwarry Nth, Victoria</td>
<td>196</td>
</tr>
<tr>
<td>W5.1</td>
<td>Wombat 5</td>
<td>Longwarry Nth, Victoria</td>
<td>210</td>
</tr>
<tr>
<td>W6.1</td>
<td>Wombat 6</td>
<td>Longwarry Nth, Victoria</td>
<td>229</td>
</tr>
<tr>
<td>W7.1</td>
<td>Wombat 7</td>
<td>Eden Park, Victoria</td>
<td>292</td>
</tr>
<tr>
<td>W8.1</td>
<td>Wombat 8</td>
<td>Bullengarook, Victoria</td>
<td>13</td>
</tr>
<tr>
<td>D1.1</td>
<td>Dog 1</td>
<td>Maningrida, NT</td>
<td>16</td>
</tr>
<tr>
<td>D1.2</td>
<td>Dog 1</td>
<td>Maningrida, NT</td>
<td>19</td>
</tr>
<tr>
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1*GenBank accession number AF372989 for complete sequence

NT Northern Territory.
Figure 7.1. One of 10 maximum parsimony trees of relationships among the 9 distinct 12S haplotypes (haplo) from *Sarcoptes scabiei* presented as an unrooted network. The number on the branch between haplotypes 1 and 5 is the percent of bootstrap replicates in which this branch appears. The host and locality details are also shown for each haplotype. See Table 7.1 for host and locality of mite.
Figure 7.2. Consensus nucleotide sequence data for a fragment (326 bp) of the coding region of the mitochondrial 12S rRNA gene of *Sarcoptes scabiei* obtained from 23 mites.

Primers SR-J-14199 (forward: 5’-TACTATGGTTACGACTTAT-3’) and SR-N-14594 (reverse: 5’-AAACTTAGGATTAGATACCA-3’) were used. The red nucleotides are sites within the consensus sequence of haplotypes that vary in the type of nucleotide. Halotype 1 (Table 7.1) is shown here.

ACTTTTTCTAGGAATATCAAAAATTTATTTTTATTTAAATTTTTACGTTTCA CATCCATTTTAAAAATTTATGAAAAATTTTTCTAATTTTTGTAACTCAT ATAAATCCTTTTTATTATCTTCACATTGATCTGAATTAAAAATATTTTATT TTGAGAAAAAAA-TAAAATTTTTATCTAAACACGTAC- AAAAAAAAAAGTTAAAATTTAAAAGTGGGTATCTTTTTAATTATAC AAGTTTCTGTGAATATATAGAAAAACCGCCAATTTTTGTTATT ATAAATTTACTACTTTATAACACTAAAAA
CHAPTER 8

THE EFFECTS OF SARCOPTIC MANGE ON BEHAVIOUR AND ECOLOGY OF COMMON WOMBATS IN A POPULATION AT HIGH DENSITY

8.1. INTRODUCTION

Sarcoptic mange, which is caused by the mite, *S. scabiei* var. *wombati*, is widespread in common wombat populations and has the potential to affect the abundance of wombats. However, the method of transmission of the mite between wombats is not well understood (Skerratt et al., 1998; Martin et al., 1998; Chapter 1; Chapter 2). In humans, transmission of *S. scabiei* relies mainly on close bodily contact (Mellanby, 1944). Since common wombats are generally solitary (McIlroy, 1973; Taylor, 1993), mites are most likely to be transferred between wombats overlapping in burrow use (Skerratt et al., 1998). Similarly, overlap in den usage is thought to be important in the transmission of *S. scabiei* between foxes (Gerasimov, 1953). McIlroy (1973) and Taylor (1993) both reported that common wombats overlapped in their burrow usage but rarely occurred in the same burrow together. They did not determine the extent of burrow use overlap among common wombats.

Sarcoptic mange may affect the behaviour of wombats and hence disease transmission. Behavioural changes such as reduced exploratory behaviour have been reported in free-living red foxes (*Vulpes vulpes*) and Spanish ibex (*Capra pyrenaica hispanica*) with sarcoptic mange (Overskaug, 1994; Alados et al., 1996). Wombats with severe parakeratotic sarcoptic mange show behavioural changes as they may be
more active diurnally and may be readily approached (Skerratt et al., 1999; Chapter 3). The effects of mild to moderate sarcoptic mange on the behaviour of common wombats and whether a change in behaviour affects the transmission of mites has not been determined.

Epizootics of sarcoptic mange are thought to be more likely in wombat populations at high densities (Martin et al., 1998). However, common wombat populations at high density have not been studied in detail, with only a single general study of the ecology of mange-free wombats in agricultural land in north-eastern Tasmania (Taylor, 1993). Populations at lower density, confined to continuous sclerophyll forest, have been better studied, but their ecology may differ from that of populations occurring at high densities (McIlroy, 1973; Buchan and Goldney, 1998).

Therefore the aims of this study were: 1. to determine the extent of burrow sharing among common wombats, 2. to examine the effects of mild to moderate sarcoptic mange on the behaviour of common wombats and 3. to study a wombat population at high density and investigate the effects of high density on transmission of sarcoptic mange.

8.2. MATERIALS AND METHODS

Study site

A common wombat population of high density that occurred in riparian vegetation and adjacent pasture along the Yea River at Padilpa (37°22′6″ S, 145°28′30″ E), elevation 200m, near Glenburn (37°26′ S, 145°25′ E), Victoria, was
studied (Figs 8.1, 8.2). Landholders had reported seeing mange in wombats in that area prior to the study. The upper storey of vegetation was dominated by manna gum (*Eucalyptus viminalis*), silver wattle (*Acacia dealbata*) and the shrub *Hymenanthera dentata*. The understorey consisted mainly of native and introduced grasses, native sedges and reeds and introduced weeds such as blackberry (*Rubus fruticosus*). Meteorological data were recorded at weather stations near to Padilpa, precipitation (mm) at Glenburn (37°22'53"S, 145°28'20"E), elevation 250 m, and average maximum air temperatures (°C) at Eildon Fire Tower (37°12'39"S, 145°50'27"E), elevation 638 m, for each month from August 1999 to November 2000. Minimum and maximum temperatures were also recorded at Padilpa with a thermometer.

**Animal capture**

Initially, an attempt was made to net wombats (Taylor, 1993), but this was abandoned because it disturbed the normal nocturnal behaviour of wombats and was difficult to perform due to the presence of an electric fence between the remnant riparian vegetation and pasture. Wombats were then caught in weld mesh cage traps (800 x 450 x 450 mm³) placed over burrow entrances (McIlroy, 1976) (Fig. 8.3). Traps were set by holding a swinging gate open with a stick and tying a locking bar up loosely with thin wire (wire normally used to tie plastic bags) (traps designed by Murray Evans) (Fig.8.4). A wombat would knock the stick over when entering the trap, the swinging gate would close behind it and the movement of the wombat in the trap would cause the locking bar to fall (Fig. 8.5). Burrow entrances were excavated so that the entrance of the trap could be pushed into the burrow. Logs and sticks were placed on either side of and above the trap to deter wombats from digging past the trap or pushing the trap out of the way. Traps, which could not be securely jammed
into the burrow, were pegged. At the start of the study, wombats were followed at
night until they entered a burrow and then the burrow was “trapped”. Although this
method ensured that traps were set on a burrow that was occupied, it was more
difficult to set traps at night and it disturbed the normal nocturnal behaviour of the
wombats. Therefore, to improve the probability of trapping an occupied burrow
during the day, several methods employed by McIlroy (1973, 1976) were used. The
location of burrows was identified and burrows were classified into three types,
“major”, “medium” and “minor” according to the criteria of McIlroy (1973): length,
entrance area and the size of the mound of soil at the entrance. Burrows were marked
with tape and a number painted on a nearby tree. “Major” burrows were trapped since
they are more likely to be occupied than “medium” or “minor” burrows. Prior to
trapping, sticks were placed upright at burrow entrances and soil was raked around
burrow entrances. Footprints in the raked soil and the direction of sticks, which had
been knocked over, were used to determine the movements of wombats and whether a
burrow was likely to be occupied (McIlroy, 1973, 1976). Occasionally, traps were
placed because wombats were seen entering a burrow in the daytime or noises typical
of wombats moving or grooming inside the burrow were heard (McIlroy, 1976).

Population estimates

The estimate of the population density at Padilpa was determined by mark-
recapture methods (McIlroy, 1977). Another estimate of population density was made
during the course of this study in a different area, a property at Boho South (36°48’ S,
145°45’ E), elevation 600 m, which provided a comparison for Padilpa. The estimate
at Boho South was made using the direct estimate procedure of McIlroy (1977) where
the burrows most likely to contain wombats were trapped for four consecutive nights,
although McIlroy (1977) trapped for only three consecutive nights. The remaining
burrows had indicator sticks placed over their entrances. This method relies on the
assumption that wombats are unlikely to remain inside a burrow for more than four
nights.

Animal handling, sampling and marking

Wombats were anaesthetised with tiletamine/zolazepam at 4 mg/kg (Zoletil,
Virbac) to facilitate handling and collection of morphometric data. They were
weighed using Salter spring scales (0 – 50 kg at 0.2 kg divisions). Vernier callipers
(Mitutoyo, Japan) were used to measure the length of a manus and a pes, the length of
a testis and epididymis and width of a testis. Spring callipers were used to measure the
length of the cranium from the occipital crest to the anterior margin of the rhinarium.
The body length of wombats was determined by placing the animal in dorso-ventral
recumbency and measuring the distance along the ventral body surface between the
cloaca and the anterior margin of the rhinarium with a tape measure. Pouches of
females were inspected for size, moisture content, pouch young, elongated enlarged
teats and swollen mammary glands. Elongated teats were stripped to see if they would
express milk. Pouch young were measured similarly to adults although body length
was not measured because pouch young could not be easily held in dorso-ventral
recumbency (Fig. 8.6). Testes of pouch young were not measured. All animals were
examined for ectoparasites (Fig. 8.7) and signs of sarcoptic mange such as erythema,
parakeratosis and alopecia.

For each animal, the sex and pelage colour and unusual characteristics such as
hair loss, scars, damaged ears, markings and wounds were recorded (Fig. 8.8). Ear
biopsies were taken for genetic analysis from the middle of the pinna using either a 4 or 7 mm diameter biopsy punch. Swivel disc tags used for marking sheep, cut to a tear drop shape to fit the shape of the pinna, were attached to the ear using the hole in the pinna created by the biopsy punch. Animals were also tattooed on their inner thigh with consecutive numbers using a battery-powered tattoo gun and Indian ink. Wombats were allowed to recover fully from the anaesthetic before being released at the site of capture.

*Surgical implantation of radiotranmitters*

To enable specific individuals to be studied over time, radio transmitters were implanted. Although collars with radio transmitters attached have been used successfully in wombats previously (Evans, 1998), they were not considered to be suitable because of the irritation and damage that they may cause to wombats with sarcoptic mange. Initially, subcutaneous placement in the ventral neck region was chosen because of the reported transmural movement of transmitters placed in the left paralumbar fossa across the abdominal and intestinal wall causing obstruction of the gut of southern hairy nosed wombats (*Lasiorhinus latifrons*) (Maver, 1998) (Figs 8.9, 8.10). However, due to the loss of 8 of 12 radio transmitters from the ventral neck region, 6 transmitters were subsequently placed intra-abdominally into the same wombats.

Cylindrical radio transmitters, weighing 14 g, measuring 16 mm in diameter and 55 mm in length, emitting a distinct frequency in the range of 150000 to 150150 kilohertz, with a battery life of 18 months, were used (Biotrack, Dorset, UK). They were triple coated in an implant grade high strength RTV silicone elastomer
dispersion in xylene (Applied Silicone, California, USA), and allowed to cure for a
week prior to surgical implantation. Radio transmitters were sterilised by immersion
overnight in either 10% povidone iodine or 2.5% glutaraldehyde (Cidex, Johnson and
Johnson, North Ryde, New South Wales) prior to surgery and then washed thoroughly
with sterile saline.

Wombats were anaesthetised with tiletamine/zolazepam at 10-15 mg/kg given
intramuscularly. Several millilitres of local anaesthetic, lignocaine hydrochloride 20-
40 mg (2% Lignocaine, Troy Laboratories) were injected subcutaneously around the
surgical site to provide additional analgesia. The fur in the ventral neck region was
clipped and the skin was then cleaned with 0.05% chlorhexidine acetate and 0.5%
cetrimide (Baxter Healthcare, Toongabbie, New South Wales) until no dirt could be
detected on the cotton wool swab. It was then cleaned with 70% alcohol and the
swabs were again checked for dirt. Finally, the skin was cleaned with 10% povidone-
iodine (Betadine, Faulding Pharmaceuticals, Salisbury, South Australia). Sterile
drapes were then used to delimit the surgical site. A 3-4 cm incision was made in the
skin of the ventral neck with a scalpel blade and a pocket large enough for the
transmitter to fit was created posterior to the incision under the skin by blunt
dissection (Fig. 8.9). The incision was then closed with a continuous subcutaneous
suture using 2.0 mm diameter polydioxanone (PDS) material (Ethicon, Johnson and
Johnson, North Ryde, New South Wales) and single interrupted skin sutures using 4
mm thick nylon (Vetafil, Bengen, Germany) (Fig. 8.10). The sutured surgical wound
was sprayed with an antibacterial insect repellent (Cetrigen, Virbac, Peakhurst, New
South Wales). The wombat was given a long acting penicillin injection, which
contains a combination of penicillins given at different dose rates, procaine and
benzathine penicillin, 15 mg/kg and 11 mg/kg, respectively (Norocillin L.A., Heriot Agvet, Rowville, Vic). Surgical equipment was sterilised prior to surgery by autoclave. Equipment that was reused in the field was sterilised by steaming it in a pressure cooker for 30 minutes. Sterile surgical gloves were worn during the surgery and a surgical scrub of hands and arms with 10% povidone iodine was undertaken prior to surgery.

The surgical technique for implantation of intra-abdominal radio transmitters was similar to that mentioned above except that the ventral abdominal midline was used as the surgical site for access into the peritoneal cavity and a sterile plastic film (OpSite, Smith and Nephew, England) was placed on the skin prior to incision to reduce the chances of wound contamination. An incision was made through the skin, the abdominal muscle and the linea alba. Transmitters were attached to the abdominal wall using a technique suggested by Andrew Rissman (Monarto Zoological Park, South Australia, in litt.). Non-absorbable suture material polypropylene (3.5 mm diameter) was used to suture a small loop of stainless steel suture material 3.5 mm in diameter (Ethicon, Johnson and Johnson) attached to the transmitter by medical grade silastic adhesive (Dow Chemical Company, USA) to the abdominal wall. The linea alba and abdominal muscle were closed with simple interrupted sutures using absorbable polydioxanone (3.5 mm diameter) suture material. The subcutaneous and skin layers were closed as above.

Animals which may have been dehydrated prior to surgery due to staying down a burrow for several days before being trapped were given 500 ml of intravenous, isotonic saline fluids (0.9% NaCl) during the operation. If wombats
started to recover from the anaesthetic during the operation they were given a third of the original dose of anaesthetic, tiletamine/zolazepam, intravenously or half of the original dose intramuscularly. Subcutaneous and intra-abdominal transmitters were recovered from wombats at the end of the study using the same sterile procedures described above. Wombats with intra-abdominal transmitters were placed on a rebreathing anaesthetic machine and halothane inhalation anaesthetic (Veterinary Companies of Australia, Artamon, New South Wales) after induction of anaesthesia with tiletamine/zolazepam due to the longer time it took to undertake the operation. The skin layer was closed with an intracutaneous continuous suture using absorbable PDS (2.0 mm diameter) suture material because wombats would not be recaptured to remove stitches from skin. Towards the end of this study, lightweight radio transmitters (Titley Electronics, Ballina, New South Wales) became available. These transmitters weighing about 1 - 2 g and were glued with superglue to ear tags (Fig. 8.11). The total weight of ear tag and transmitter was approximately 5 g.

Recovery of transmitters that were lost by wombats
Radio transmitters, which were placed subcutaneously, were invariably lost from wombats when in a burrow. Transmitters were recovered from burrows by digging with a shovel or a backhoe equipped with a 50 cm wide bucket at the point of the strongest signal above ground until the burrow was reached (Figs 8.12, 8.13).

Radio location and “spooling” of wombats
Radio transmitters enabled location of wombats within specific burrows during the day. The number of days and consecutive days that wombats were radio located during the period August 1999 to November 2000 varied among individuals,
17 – 50 days and 10 – 30 consecutive days, respectively. Radio transmitters were not used to follow the movements of individuals at night because the observer frequently disturbed other wombats whilst following an individual. The observer also regularly needed to use a spotlight in order to identify the individual being followed from other wombats near by which again disturbed wombats. Therefore the movements of wombats were determined by “spooling”, a technique which involves attaching a spool to a wombat which unwinds as the animal moves about, leaving a thread line behind. Spools contained centrally wound nylon No. 10 bobbins, 248 m in length, (Danfield Bobbins, Penguin Threads, Prahran), which unwind from the centre. In each spool, 4 or 5 bobbins were tied together in series enclosed in shrink-wrap and then elastoplast (10 cm width). Spools weighed between 30 and 40 g and were glued to the fur on the back of wombats with epoxy resin that cured in 5 minutes (Fig. 8.14). Bobbins were dyed one of four different colours (Gold Cross Fabric Dye, Dye Manufacturers of Australia, Brisbane, Queensland) and two different coloured bobbins were placed alternately in each spool. Bobbins were placed in boiling dye solution for 30 minutes in order to dye them. The thread line from the spool was tied to a tree root at the burrow entrance where the wombat was released. The next day the thread line was followed to determine the use of space and burrows by wombats (Figs 8.15, 8.16). The movement of the wombat was mapped by taking an Australian Map Grid position (Australian Geodetic Datum 66) every 20 m using a global positioning satellite system (GPS, Garmin). Twenty metre intervals were initially determined by a chain and then were estimated by pacing.

*Experimental infection with S. scabiei*
Since wombats with sarcoptic mange were not found at Padilpa, five wombats without sarcoptic mange were deliberately infected with *S. scabiei* var. *wombati* to determine the effects of mild to moderate forms of sarcoptic mange on wombat behaviour. *Sarcoptes scabiei* were obtained from parakeratotic crusts from a common wombat with sarcoptic mange from Tonimbuk, Victoria and an experimentally infected captive wombat held at the Veterinary Clinical Centre, Werribee, Victoria which was originally infected with mites from a wombat from Healesville (37°41′ S, 145°32′ E). Mites were obtained from parakeratotic crusts by the method of Sheahan and Hatch (1975) by placing them in Petri dishes on a metal tray on a magnetic stirrer plate. The vibrations from the magnetic stirrer plate stimulated mites to move out of crusts. Three adult female wombats, two with pouch young (Female 1 and 3) and the third (Female 2) suckling a young at heel, and two adult males (Male 1 and 2) were infected with between 4000 and 8000 mites each (Table 7.8). Wombats were infected by brushing mites onto the shoulders, flanks and legs. These sites on the body were chosen because they have a significantly higher intensity of infestation than other areas in free-living wombats with sarcoptic mange and therefore are the most likely to allow the establishment of mite infection (Chapter 3, Skerratt et al., 1999).

In order to quantify the severity of clinical signs of sarcoptic mange in the infected wombats, the body surface was divided up into 13 sections of equal value (Fig 8.17). There were four sections which were duplicated on each side of the body, face, foreleg, flank and hindleg, totalling eight sections, and five sections that included both sides of the body, forehead, back, rump, abdomen and between the shoulders. These areas were chosen since the severity of clinical signs differed between them but was similar within them in the study of free-living common
wombats with severe sarcoptic mange (Skerratt et al., 1999; Chapter 3). The clinical 
signs of sarcoptic mange that were identified in free-living common wombats and 
captive, experimentally infected common wombats, erythema, parakeratosis (scale 
crust) and hair loss were quantified in each section (Chapter 3 & 4; Skerratt et al., 
1999). The average thickness of parakeratotic scale was categorised subjectively as <1 
mm, 1-5 mm or >5 mm. The degree of hair loss for each section was categorised 
subjectively into the following <25%, 25-50%, 50-75% or 75-100%. Erythema was 
regarded as being either present or absent (100 or 0%). The median value for 
parakeratotic scale and hair loss for all sections was then determined as a measure of 
the severity of each clinical sign. The severity of erythema was described as the 
percentage of body sections with erythema. Three body sections, the rump, back and 
forehead were not included when determining the severity of each clinical sign since 
they were not affected.

Wombats were caught 50 and 100 days after infection (DAI) to assess the 
severity of sarcoptic mange, determine the intensity of infection and to examine their 
behaviour by the technique of “spooling” (explained above). They were treated to 
eliminate *S. scabiei* infection 100 DAI with ivermectin at 400 µg/kg (Ivomec, 
injectable for cattle, Merck & Co, South Granville, New South Wales). Five healthy 
wombats, which were also fitted with spools at this time, were also treated with 
ivermectin in order to act as controls and to prevent them becoming infected with *S. 
scabiei*. Wombats were re-caught 128 DAI to retreat with ivermectin at 800 µg/kg and 
to wash in a topical acaricide, amitraz at 250 µg/ml concentration (Demadex, Delta 
Laboratories, Hornsby, New South Wales) to ensure that all mites were killed. Radio 
transmitters were also removed at this time. One wombat (male 1) with a significantly
higher intensity of infection was treated twice more with each treatment given 10 days apart. Another wombat (Female 2), with the second highest intensity of infection was recaptured 2.5 months later to ensure that a recrudescence of sarcoptic mange had not occurred in that animal.

Statistical analyses

The mean and the standard error of means together with the range and the number of samples collected are provided for descriptive statistics. To determine whether statistically significant differences occurred between treatment groups, such as mangy versus not mangy, or male versus female, Student t-test (Excel 97, Microsoft, USA) was used. When more than two groups were compared, an ANOVA, was used. A general linear model in the program SPSS® for Windows 8.0 (SPSS Inc., Chicago, Illinois, USA) was used to test whether the ratios of weight to head length for the sexes were significantly different (see Sokal and Rolfe, 1997 for an explanation of statistical tests). The program MapInfo version 6.0 (MapInfo Corporation, USA) was used to map the movements of wombats. The program Ranges Version 5.0 (Natural Environment Research Council, UK) was used to determine the area of convex polygons, which summarised the area in which wombats moved.

8.3. RESULTS

Success of trapping and marking of wombats

The majority of wombats (64%) was caught on the first night a trap was placed over a burrow entrance (Table 8.1). However, approximately 24% of wombats
did not enter the trap on the first night and were caught on subsequent nights. Therefore one in four wombats remained in the burrow on the first trap night. The remainder of wombats that were caught, 12%, cohabited a burrow and were forced to remain in burrows for more than one night due to another wombat already being caught in the trap. Generally, wombats entered a trap after midnight but before dawn. One young at heel joey, a 2.9 kg female, entered a trap during the day (Fig. 8.5). The proportion of wombats caught on the first night a trap was placed over a burrow did not change during the course of the study (Fig. 8.18) even though the majority of wombats trapped in the latter half of the study were recaptures (Fig. 8.19). Therefore, there was no evidence that the experience of being trapped affected the subsequent behaviour of wombats towards traps. It is possible that wombats were more likely to dig past a trap after having been previously trapped. One wombat escaped from a burrow, by digging past a trap, for every five wombats caught (Table 8.1). Very rarely, wombats escaped due to another wombat digging into the burrow past the trap. Occasionally, wombats escaped back down a burrow by pushing the swinging gate past the angle-iron stops.

There were some undesirable side effects to trapping and marking animals. Some wombats broke nails on their manus and chipped teeth whilst trying to get out of a trap. However within a few months, both nails and teeth regrew back to normal. Some animals eroded the skin of their noses by repeatedly lifting the locking bar in the trap with their head and some eroded the skin of their manus by digging at the floor of the trap. A few wombats pulled ear tags out, splitting the pinna and some ears became infected and, very rarely, provided sites for myiasis to develop. However, the majority of ear tags caused no secondary problems. They often became covered with
dirt and although they were difficult to identify at night from a distance, they could be identified when the wombat was in a trap. Tattoos were not associated with any side effects and did not fade during the course of the study.

Reproductive status

A large proportion (84%) of adult females captured (n=25) at Padilpa from August 1999 until January 2001 showed signs that they were reproducing. Females either had pouch young or, if their pouch was empty, showed signs of lactation such as swollen teats from which milk could not be expressed but which were probably in the process of regression after young had been weaned. There was also a high proportion of young animals in the population (45%) (Table 8.2). Only four females did not show signs of reproduction. Two of these were large females weighing 29 kg each, caught in August and October, respectively and may have been about to reproduce. The remaining two were small females weighing 22 and 24 kg, caught in August and April, respectively and may have been nulliparous. Small pouch young weighing less than 200 g were found in pouches from March to May in 2000 although two pouch young weighing less than 100 g occurred in August and November of 1999 (Fig. 8.20). Females that weighed less than 22 kg did not show any signs of reproduction. The lightest female to reproduce weighed 22.3 kg however this was after it had been lactating for 10 months since it weighed 25 kg when it was captured just prior to parturition 10 months earlier.

Pelage/wounds

Wombats varied in pelage colour. Forty were brown, 17 were grey and 8 were black. About one third (38%) of the adult males (n=16) had hair loss on their forehead
and 31% had evidence of fighting. Bite wounds and scars on the back, neck and head were the most common injuries observed followed by torn pinnae. One adult male had a torn cloaca as well as bite wounds on the neck. Only one adult female (4%) showed signs of fighting, indicated by a torn pinna. One adult female had hair loss on its forehead and rump and another had hair loss on its rump. Hair loss on the rump was also seen in an adult male and a female joey at heel. Immature wombats did not have wounds or scars.

Weight, size and growth

Adult males and females were similar in weight and size such as head length, pes and manus length and body length (Table 8.3). Adult males had a significantly higher head length to weight ratio than adult females (Fig. 8.21). Weight in adults can be explained by the following regression,

\[ \text{weight} = 0.33 \times \text{head length} + 29.88 \times \text{sex} - 0.15 \times \text{sex} \times \text{head length} - 41.77 \]

\( \text{SE}=0.06, P=0.00 \quad \text{SE}=15.61, P=0.04 \quad \text{SE}=0.07, P=0.04 \quad \text{SE}=12.78, P=0.00 \)

(sex: 1=male, 0=female, weight (kg) and head length (mm), n=42, \( r^2=0.60, P=0.00 \)).

Pouch young grew slowly until they reached 1 kg in weight after which there was an exponential increase in growth rate until joeys reached about 13 kg and were weaned (Fig. 8.22). Pouch young took between 6 and 7 months to reach 1 kg (Fig. 8.22).

Density of wombats/ burrow use

The direct estimate of the density of wombats at Padilpa based on mark-recapture methods suggested that there were 58 wombats, excluding pouch young, in the 3 ha of remnant vegetation along the Yea River giving a density of 19 wombats/
Wombats were not distributed evenly but often occurred together in burrows. Most occupied burrows contained more than one wombat (71%) with the number of wombats ranging from two to five, excluding pouch young (Table 8.5). Wombats occupying the same burrow could be any combination of age, sex and stage of reproduction (Table 8.6). However, opportunistic observations on wombats, which were seeking burrows for diurnal shelter, revealed that they would not enter some burrows after pausing at the burrow entrance to inspect it. On one occasion, an adult male was seen entering a burrow from which he then emerged, chased by a larger male. The larger male then stopped above the burrow entrance and made repeated vocalisations, similar to the noise one can make by sucking and blowing air through a partially closed mouth and allowing ones cheeks to move in and out. The male also raised his head while the intruder ran away. Joeys that had recently emerged from the pouch, between 2.5 and 3.0 kg in weight, showed strong burrow preferences. A single bobbin was stuck to a 2.9 kg female joey that was caught in a trap to see where it would roam without its mother (Fig. 8.5). The mother had a radio transmitter attached and was not nearby when the joey was “trapped”. The joey did not venture more than 20 m from the burrow entrance whilst the mother was away for 24 hours. A 2.5 kg joey was released at a burrow entrance that was 10 m from the one in which it had been trapped. However, it would not enter the burrow and returned to the burrow in which it had been trapped. The total number of burrows used by Females 1 and 2 and Males 1 and 2 were 11, 8, 10 and 12, respectively between August 1999 and November 2000 (Table 8.7). Female 3 used 6 burrows after being located in a burrow on 17 occasions between April and November 2000 (Table 8.7). Many burrows were used for only a few nights while others were used regularly.
Ranging behaviour and climate

Adult and sub-adult wombats of both sexes usually walked past a number of burrow entrances, 2.1 ± 0.3 (0 – 12), during their nightly activities (No. of nights monitored=55). However they generally did not enter a burrow, 0.6 ± 0.1 (0 – 6) burrows entered per night, (No. of nights monitored=55). These figures do not include the burrows used for diurnal shelter unless a wombat entered then emerged and re-entered a burrow used for diurnal shelter. Almost half of the burrows (43%) appeared to be unoccupied at any one time (Table 8.5). There was a concentration of burrows in the large patch of remnant vegetation at the northern end of Padilpa (Fig. 8.23). Wombats used a few of the burrows that occurred nearby but outside the remnant vegetation on Padilpa for diurnal shelter (Fig. 8.23). Most of these burrows occurred near the river. The entrances of a few burrows that were located along a fence perpendicular to the river at the northern end of Padilpa were visited regularly by two of the large adult males (Males 2 & 3) but were not used for diurnal shelter by these wombats (Figs 8.23, 8.24). Very few new burrows were dug from August 1999 to November 2000 but renovations to existing burrows occurred after heavy rains and flooding in spring of both years.

Most rainfall at Padilpa in 2000 occurred in late autumn, winter and spring (Table 8.8). The distances travelled and areas covered per night by wombats without sarcoptic mange at Padilpa in 2000 decreased after the “autumn break”, which is regarded as the first significant rainfall after a dry summer. They continued to decrease significantly in late winter and mid spring (Table 8.9). Healthy adult males travelled significantly further each night than healthy adult females, 833 ± 87, 120 -
>1567 m (nights=24) versus 512 ± 43, 140 - >1220 m (nights=35), respectively (Student t-test, \(P<0.05\)) (see also Table 8.10). Adult males also covered a larger total area than adult females despite a similar number of fixes of location, 7.3 ± 0.6, 6.1 – 8.3 ha (n=3) versus 3.8 ± 0.5, 2.4 – 5.0 ha (n=4), respectively (Student t-test, \(P<0.05\)) (see also Table 8.10). The determination of area used by wombats was limited to between 3 and 9 nights of spooling for each wombat between December 1999 and October 2000 (Fig. 8.25). One sub-adult male travelled similar distances each night and covered a similar total area to females (Table 8.10). Large adult males appeared to travel the furthest distance each night, although adult and sub-adult females occasionally travelled long distances. A sub-adult female travelled from her burrow on the river for over a kilometre across Padilpa, south along Steuarts Road and then up Break O’Day Creek before the thread line ran out, 1040 m from the release site. She was caught a few days later back in the same burrow. The movements of adult males were generally perpendicular to the river whereas the movements of adult females were parallel to the river (Figs 8.24, 8.26, 8.27, 8.28). Wombats generally emerged from their burrows at dusk or in the first few hours after dusk in winter and summer despite a large difference in ambient temperatures at this time, 8 – 14 °C, versus 19 – 25 °C, respectively. There was no significant difference between individuals in time of emergence, Table 8.11, ANOVA single factor \(P>0.05\). One female with young at heel (Female 6) consistently emerged from her burrow after 10.00 p.m. (data not shown). Large adult males (>29 kg) emerged before adult females in the summer of 1999/2000, 8.56 ± 0.06 p.m. (nights = 10) versus 9.26 ± 0.10 p.m. (nights = 20), Student t-test \(P<0.05\) (see also Table 8.11). Occasionally, wombats would not emerge from their burrow to feed at night and sometimes they were seen wandering around during the day.
**Territorial behaviour**

“Spooling” of wombats revealed that they distributed their scats both near and far away from burrows during their nocturnal wanderings. Wombats also regularly visited and may have drunk from water bodies at night such as the river and surrounding dams. Marking of territory occurred occasionally during the wombat’s nocturnal activities denoted by removal of grass by scratching to expose a small patch of soil approximately 400 cm² in area. Scats were also sometimes present (Fig. 8.16).

**Ectoparasites**

Ticks, fleas, lice and mites were prevalent on common wombats at Padilpa (Table 8.12). The most prevalent ectoparasite was the common wombat tick, *Aponomma auruginans*, occurring on 93% of wombats at intensities ranging between 10 and greater than 100 ticks per wombat. Ticks were easily seen on ventral areas but also occurred on more densely furred parts of the body such as the head and chest (Fig. 8.7). The tick *Ixodes tasmani* was less prevalent, being found on 12% of wombats. It occurred at a much lower intensity with just a few ticks seen on each wombat. Female *I. tasmani* usually occurred around the cloaca while nymphs were seen on the margins of the pinnae. Fleas and lice were occasionally seen wandering on ventral surfaces but were usually seen by parting the dense fur on the lateral surfaces and along the back. Sometimes fleas and lice would only be found after searching the fur for 10 to 15 minutes. The mite *Acaroptes vombatus* was present on wombats from Padilpa but *S. scabiei* was not found. Signs of sarcoptic mange such as erythema, scale crust adherent to the skin and hair loss were also not seen in wombats from Padilpa. Hair loss was occasionally seen but was not associated with mite infection.
Hyperkeratosis (dandruff) was also seen but it was not adherent to the skin like the scale crust that is seen with infection with *S. scabiei*.

*Infection with S. scabiei*

All wombats infected experimentally with *S. scabiei* developed sarcoptic mange. The intensity of infection was low in all wombats 50 DAI (Table 8.13). Female 3 was not trapped at this time as it had lost its transmitter. Intensity of infection remained low in three wombats at 100 DAI and had decreased since 50 DAI in two wombats (Table 8.13). Extent of erythema and thickness of parakeratotic scale did not change in these wombats between 50 and 100 DAI. However, there was significant hair loss in this period (Table 8.14). Using the classification system in Chapters 3 and 4, these three wombats exhibited mild sarcoptic mange (Figs 8.29, 8.30, 8.31). After treatment with ivermectin at 400 µg/kg 100 DAI, mites were not found in skin scrapings 128 DAI (Table 8.13). There was a large increase in intensity of infection in two wombats, Female 2 and Male 1 between 50 and 100 DAI. These two wombats had thicker parakeratotic scale on some sections of the body, more extensive erythema and greater hair loss (Table 8.14). They also had some fissuring of thick parakeratotic scale and skin, and these were classified as mild to moderate cases of sarcoptic mange (Figs 8.32, 8.33). Treatment with ivermectin at 400 µg/kg 100 DAI had not killed all the mites on these two animals 128 DAI, although intensity of infection was lower than that 100 DAI. Hence, both wombats were re-treated with ivermectin as well as a topical acaricide, amitraz. The wombat with the greater intensity of infection (Male 1) was treated 2 more times, 10 days apart, and the other wombat (Female 2) was checked several months later for signs of *S. scabiei* infection.
Both recovered completely showing no signs of sarcoptic mange and mites were not found in skin scrapings.

It appears that mites were not transferred from wombats infected with *S. scabiei* to other wombats. In the 100 days following the infection of 5 wombats, 34 wombats that had not been infected but shared burrows with infected wombats were trapped. They did not show clinical signs of sarcoptic mange nor was *S. scabiei* found in skin scrapings.

Adult wombats without sarcoptic mange lost weight in autumn and early winter (Table 8.15). The weight loss was significant in early autumn (Table 8.15). These same wombats were then infected with *S. scabiei var. wombati* in winter and gained weight in late winter despite just being infected with *S. scabiei*. However, in early spring, between 50 and 100 DAI, wombats with sarcoptic mange lost weight. This weight loss was significant. They started to increase in weight again in spring after treatment for sarcoptic mange with ivermectin. However the two wombats with the highest intensity of infection which were still infected with mites 128 DAI did not increase in weight (Tables 8.13 and 8.15).

Wombats with sarcoptic mange travelled significantly further and covered a larger area than wombats without mange in mid-spring, covering similar distances and areas to wombats without sarcoptic mange in early autumn (Table 8.9). The distance travelled and area covered by mangy wombats was greater in mid-spring (100 DAI) compared with late winter (50 DAI).
Wombats with sarcoptic mange used fewer burrows and were less likely to change burrows than wombats without sarcoptic mange (Table 8.16). The difference between the two groups was not significant. However the $P$ value approached 0.05 when the number of consecutive nights that a wombat used a burrow was compared between wombats with sarcoptic mange and those without (Student t-test, $P>0.05$). Mangy wombats were also less likely to enter a burrow as they wandered around at night than wombats without mange, $0.3 \pm 0.2$ burrows entered per night (nights=15) versus $0.9 \pm 0.4$ (nights=18), respectively. Again this difference was not significant (Student t-test, $P>0.05$). The number of burrow entrances that wombats walked past each night was similar for mangy and non-mangy wombats, $3.0 \pm 0.6$ (n=15) and $3.1 \pm 0.7$ (n=18), respectively.

**Genetics**

The methods and results of genetic analysis of the wombats in this study are published elsewhere (Banks et al., 2001).

**Success of radio transmitters**

Subcutaneous and intraperitoneal transmitters as well as transmitters attached to ear tags could be detected from at least 100m when the wombat was underground and from several kilometres when the wombat was in a direct line of sight above ground. The placement of subcutaneous radio transmitters was relatively unsuccessful with the majority being lost from the implanted site after variable periods, but usually within a few months (Table 8.17). One transmitter remained under the skin of the ventral neck region for 10 months before it was lost. Transmitters in the process of being rejected were surrounded by purulent material, which drained from a wound.
near the anterior end of the transmitter. Transmitters that were not rejected but which were removed from the ventral neck region after variable periods were surrounded by a fibrous capsule. Intra-abdominal transmitters were also relatively unsuccessful since half (3 of 6) were lost after a few months (Table 8.17). One intra-abdominal transmitter crossed the abdominal wall and came out through the skin near the surgical site. The other two may have crossed the intestinal wall into the lumen of the gut and been passed out in faeces since there was no sign that they come out through the skin. The intra-abdominal transmitters that had not been rejected but which were removed from wombats had broken free from their attachment to the abdominal wall. Two were found enclosed in omentum, one surrounded by a fibrous capsule and the other by 50 ml of clear fluid. The third transmitter had lodged in the pelvis of a male between the bladder and rectum but there was no reaction to the transmitter, which had been in place for 8 months, and it did not affect the wombat’s ability to defaecate or urinate. Seven of the twelve implanted transmitters also stopped emitting a signal within 2 to 5 months due to battery failure which limited the number of animals that could be studied. Transmitters attached to ear tags were reasonably successful although they showed signs of being too heavy for the ear and some appeared to be pulling the ear tag out of the pinna. One transmitter had its aerial broken at the base.

**Burrow dimensions**

Recovery of some transmitters from burrows by digging with a shovel or backhoe enabled the measurement of burrow dimensions. The rooves of burrows 7 to 15 m from the burrow entrance were 2.4 ± 0.1 m (1.8 – 2.8 m, n=7) below the surface. Burrows 7 to 15 m from the entrance were wider than they were high, 0.36 ± 0.02 m (0.30 – 0.41 m, n=5) versus 0.26 ± 0.02 m (0.20 – 0.31 m, n=7), respectively. The
cross-section of the burrow was roughly elliptical in shape with respect to the horizontal axis. Burrows divided underground, which created a network of tunnels that enabled burrow entrances to be connected. Usually only two burrow entrances were connected but in one case three burrow entrances were connected within 20 m of each other. A sleeping chamber was also found 10 m from the entrance to a burrow and 2.5 m from the end of the burrow. It was 1.0 m wide by 0.4 m high by 0.8 m long and much larger than the volume of an adult wombat.

**Other animals**

Several other species of native and introduced mammals were present at Padilpa. The common brushtail possum (*Trichosurus vulpecula*), the mountain brushtail possum (*Trichosurus caninus*), the common ringtail possum (*Pseudocheirus peregrinus*), the sugar glider (*Petaurus breviceps*), rabbits (*Oryctolagus cuniculus*) and mice (*Mus musculus*) were plentiful whereas koalas (*Phascolarctos cinereus*), water rats (*Hydromys chrysogaster*), platypus (*Ornithorynchus anatinus*) and foxes (*Vulpes vulpes*) were rarely seen. Only rabbits and foxes were seen inhabiting wombat burrows along with tiger snakes (*Notechis scutatus*) and frogs (*Limnodynastes tasmaniensis*). Spotted pardalotes (*Pardalotus punctatus*) were also seen nesting in wombat burrows.

### 8.4. DISCUSSION

**Effects of sarcoptic mange on wombats**

Sarcoptic mange affected behaviour. Wombats with mild to moderate sarcoptic mange travelled significantly further and covered a larger area than wombats
without mange in mid-spring, covering similar distances and areas to wombats without sarcoptic mange in early autumn. This is possibly due to the energy drain of sarcoptic mange on wombats (Chapter 3 & 4, Skerratt et al., 1999). Captive wombats lost weight while developing localised moderate sarcoptic mange and continued to lose weight as mange became more severe (Chapter 4), showing that localised moderate and severe sarcoptic mange had an energy drain on wombats. Since the pasture was a mosaic and the species of grasses and their new growth upon which wombats probably prefer to feed were widely dispersed within the pasture, wombats could have been travelling further in order to eat more. The distance travelled and area covered by mangy wombats was greater in mid-spring (100 DAI) compared with late winter (50 DAI) despite the presence of more grass in mid-spring. This could be due to *S. scabiei* infection having a greater energy drain on wombats in spring, which is possible since wombats were infected with *S. scabiei* in mid winter and there is an induction phase during which there is no immune reaction by the host to mites. The host immune reaction is responsible for an energy drain by inducing severe pruritus (Chapter 4). In accordance with the above conclusions, wombats had no discernible hair loss at 50 DAI but obvious hair loss by 100 DAI. In addition, wombats lost weight between 50 and 100 DAI but gained weight prior to 50 DAI and after treatment for sarcoptic mange at 100 DAI. If the *S. scabiei* infections in wombats had been allowed to progress further then the behaviour of wombats may have changed again since moderate to severe sarcoptic mange causes debilitation. Foxes with severe sarcoptic mange are less active, use smaller areas, are more active during the day and probably eat less (Overskaug, 1994). Similarly wombats with severe sarcoptic mange are more active during the day and possibly eat less (Chapter 3, Skerratt et al., 1999). It is unlikely that lactation had a significantly greater effect on females during the
course of *S. scabiei* infection compared with prior to or after infection since two of the pouch young were small (<0.5 kg), requiring little milk, and the third was being weaned when wombats were infected. The young of Females 1, 2 and 3 weighed <100 g, 6.2 kg and 100 g on the 31st of March 2000 and 1.2 kg, 13 kg and between 2-3 kg on the 7th of October, respectively.

The extent and severity of clinical signs of sarcoptic mange, erythema, parakeratotic scale and hair loss, was greatest in the two wombats with the highest intensity of *S. scabiei* infection. Clinical signs have been correlated with intensity of infection in free-living wombats with severe sarcoptic mange and captive wombats experimentally infected with *S. scabiei* var. *wombati* (Chapter 3 & 4, Skerratt et al., 1999). The intensity of infection decreased in two wombats from 50 to 100 DAI. During this period, the extent and severity of clinical signs of erythema and parakeratosis did not change in these two wombats. However, there was an obvious increase in the amount of hair loss suggesting that these wombats scratched more in this period. Mellanby (1944) and Davis and Moon (1990c) suggested that scratching reduces the intensity of infection by mechanical removal of mites. It is also possible that the delayed hypersensitivity phase that occurs after the induction phase in the standard immune response to *S. scabiei* occurred during this period and made the skin less hospitable for mites (Davis and Moon, 1990c). Martin et al. (1998) reported that some wombats may recover from sarcoptic mange and it is possible that the three wombats with low intensities of infection 100 DAI may have eventually eliminated mite infection without treatment. The back, rump and forehead were not affected in the present study and were the least affected parts of the body in free-living common wombats with severe sarcoptic mange (Chapter 3, Skerratt et al., 1999). This could be
due to mites not being brushed onto these areas when wombats were infected with \textit{S. scabiei} in this study.

The Leslie Matrix Model of population dynamics of \textit{S. scabiei var. wombati} can be used to determine the population growth of \textit{S. scabiei} on wombats. In two groups (n=2, 3) of free-living wombats infected with \textit{S. scabiei var. wombati} $\lambda=1.05$ and 0.97, respectively, for a two day time interval (Fig. 8.35, Table 8.18) whereas in three groups (n= 4, 2, 1) of captive wombats $\lambda=1.2$, 1.1 and 1.35, respectively (Chapter 6). There are confounding factors that prevent ready comparison between free-living and captive wombats. However, it is possible that the lower growth rates of \textit{S. scabiei} in free-living wombats were due to density dependent effects. Although the initial numbers of mites used to infect free-living and captive wombats were similar, mites were spread evenly over lateral surfaces of free-living wombats to emulate natural infection whereas all mites were placed on the shoulders of captive wombats. Therefore, the density of mites on free-living wombats was 5 times lower than captive wombats at infection. If there is an inverse density-dependant effect on population growth then it could be due to decreased survivorship at lower population densities or decreased fecundity at lower densities (Manly, 1990). Either or both are theoretically possible since mites at low density do not create sheets of parakeratotic scale, which protect them from being scratched off by the host. They also may have difficulty finding a mate. Manly (1990) suggests that mortality rates may be inversely density dependent for small populations but density dependant for large populations.

Wombats without sarcoptic mange changed the burrow they were using for diurnal shelter more frequently and were more likely to enter a burrow during their
nocturnal activities than wombats with sarcoptic mange. It is possible that this
difference is due to season rather than to sarcoptic mange since burrow use by healthy
wombats was determined in the first half of 2000 and that of mangy wombats in the
latter half. Alternatively, wombats with sarcoptic mange are less likely to change
burrows or enter a burrow possibly due to the extra energy demand of sarcoptic
mange causing wombats to decrease their exploratory behaviour in deference to
increased feeding. A similar pattern has been observed in Spanish ibex with sarcoptic
mange (Alados et al., 1996).

Since *S. scabiei* did not appear to be readily transferred between wombats
when intensity of infection was low, then it is likely that wombats with parakeratotic
sarcoptic mange, which have high intensities of infection act as a major source of
infection for the rest of the population. People with parakeratotic scabies (Norwegian
scabies) are a major source of infection for nursing staff and residents of nursing
homes (Burgess, 1994). It may therefore be possible to halt the transmission of *S.
scabiei* within a wombat population by reducing the average intensity of infection to a
low level. This could be readily achieved by destruction or removal of moderately to
heavily parasitised wombats and treatment of the rest of the population with an
effective long-acting acaricide.

Estimates of the prevalence of sarcoptic mange in wombat populations should
take into account the behavioural changes that may occur in wombats with sarcoptic
mange and how these changes may affect the estimate. For example, wombats with
mild to moderate sarcoptic mange are likely to be overestimated in netting or shooting
operations given that they may travel further, cover a larger area and are less aware of threats than wombats without mange.

**Environmental conditions/ body condition**

The availability of feed for wombats in agricultural areas depends on both the seasonal rainfall and farm management since pastures are grazed by cattle and cut for hay. The pattern of rainfall at Padilpa in 2000 was typical for a temperate area with most rain falling in late autumn, winter and spring (Table 8.8). Higher temperatures in late autumn and spring result in a rapid growth of grass. Cattle grazed pasture until winter when they were excluded so that hay could be made from the spring growth. Hence there was little grass prior to the autumn break and it occurred in patches. Green growing grass was widely distributed after the autumn break but little was available for wombats due to competitive grazing by cattle, rabbits and horses. The amount of available grass then increased dramatically especially in spring. Both introduced and native grasses occurred in pasture along with introduced clovers and native sedges. Wombats regularly ate the sedge *Carex appressa* and a species of native reed, *Juncus*, which both grew in the drainage areas of pasture.

It is possible that adult wombats lost weight in autumn and winter due to a lack of feed (grass), from lack of rain and grazing competition. The demands of lactation would not have been great on Females 1 and 3 since their young were still very small during this time. However the young of Female 2 was growing rapidly and may have contributed to her loss of weight in autumn and early winter along with a lack of available feed. The wider distribution and availability of feed in late autumn, late winter and mid spring compared with early autumn may have been the reason for
wombats without sarcoptic mange decreasing the distances travelled and areas
covered per night after the autumn break at Padilpa in 2000. This supports McIlroy’s
(1973) suggestion that home ranges may change according to food supply.

**Population structure/ home ranges**

The male to female sex ratio at Padilpa was 0.9 (n=74) compared with 1.7
(n=53) at Buccleuch State Forest (McIlroy, 1973). If only adults are considered, then
the ratios were 0.6 at Padilpa (n=57) and 1.5 at Buccleuch State Forest (n=32),
respectively. McIlroy (1973) did not suggest why there were more males than females
at Buccleuch State Forest but considered that there was no significant difference from
unity in the sex ratio. The low ratio of adult males to females at Padilpa could be due
to the significantly larger territories used by males (7.3 ± 0.6, 6.1 – 8.3 ha, n=3)
compared with females (3.8 ± 0.5, 2.4 – 5.0 ha, n=4) and competition between male
wombats for territory (Table 8.10, Figs 8.24 - 8.28). Males at Buccleuch State Forest
also had larger home ranges (14.9 ± 3.4 ha, n=4) than females (7.5 ± 1.1 ha, n=2)
although the difference was not significant (McIlroy, 1973). McIlroy (1973) stated
that the size and shape of a home range depended primarily on the pattern of
distribution of burrows used and suitable feeding areas. It is difficult to compare the
home ranges of wombats which were defined using different techniques, spooling in
this study and radio tracking in others (McIlroy, 1973; Taylor, 1993; Buchan and
Goldney, 1998). In addition, many of the estimates of home range, especially in this
study, were limited to short periods of relatively intensive study and may therefore
represent underestimates. The plots of total area used by wombats versus the number
of fixes of location do not reach an asymptote for the adult male wombats and one of
the adult females (Fig. 8.25). However, it appears that home ranges of wombats at
Padilpa may have been smaller than the home ranges of wombats in the Buccleuch State Forest (McIlroy, 1973). This is not surprising despite Buccleuch State Forest receiving more rainfall than Padilpa given the abundant food resources, such as pasture, surrounding burrows at Padilpa compared with the patchy distribution of food in the Buccleuch State Forest, such as native grasses on the forest floor (McIlroy, 1973). It is possible that adult females at Padilpa were more reluctant than males to travel far from the remnant vegetation along the river due to them having young. This would explain the smaller areas used by adult females at Padilpa compared with adult males. It would also explain the movements of females, which were mostly parallel with the river as opposed to the movements of males, which were perpendicular to the river. There were more immature males than immature females at Padilpa and Buccleuch State Forest, with male to female ratios 1.4 (n=33) and 2.0 (n=21), respectively. It is possible that more males than females are born and this may reflect a higher mortality of males as they compete for burrows and territory. Males were more closely related to each other than females at Padilpa, Tonimbuk, Victoria and Comaum, South Australia, which suggests that males do not disperse as much as females and may compete for territory (Banks et al., 2001).

**Wombat population density**

The density of the wombat population at Padilpa is extremely high compared with the density of wombats in other areas (Table 8.4). This is mainly because population estimates in other areas have included areas that do not contain burrows such as pasture. The density of wombats rose from 0.1 to 0.3 wombats/ha in Buccleuch State Forest (Area E) if only forested areas were considered. If the pasture surrounding the remnant vegetation at Padilpa is included then the density of wombats
drops from 19 to about 2 wombats/ha. However, this is still a comparatively high
density of wombats, especially if it is compared with wombat populations in
continuous forest, which occur at densities of around 0.2 wombats/ha, despite having
similar climatic conditions to Padilpa (Table 8.4). In addition, the population estimate
at Padilpa was an underestimate, since half-way through the study (after 147 captures)
trapping was biased towards burrows that contained animals with radio transmitters.
An indirect estimate based on the average number of wombats per active burrow
(Table 8.5) multiplied by the number of active burrows (n=72) divided by the area of
remnant vegetation (3 ha) suggests that the wombat density was closer to 26
wombats/ha. This latter estimate would include more wombats that only partially
ranged onto Padilpa since there is a continuous wombat population extending along
the Yea River. This population estimate excludes pouch young which were also
excluded in estimates of population density reported by McIlroy (1973) and Taylor
(1993). There were no pouch young in the population at Yetholme studied by Buchan

McIlroy (1973, 1990) and Taylor (1993) also found that the density of
wombats was higher in remnant vegetation surrounded by abundant food resources
such as improved pasture. McIlroy (1973) found that a higher wombat density was
associated with a higher density of major burrows, burrows that are used for diurnal
shelter, and an increased frequency of burrow use. Similarly, the patches of remnant
scrub in this study and at Cape Portland, Tasmania, which were surrounded by
improved pasture were riddled with active burrows, 24 and 23 active burrows/ha,
respectively (Table 8.4; Taylor, 1993). However, the ratio of active burrows to
number of wombats is lower in areas of higher wombat density (Table 8.4). This can
be explained by greater multiple occupancy of burrows (Table 8.3). Multiple occupancy of burrows by common wombats has rarely been reported in previous studies (McIlroy, 1973; Taylor, 1993, Buchan and Goldney, 1998), although up to eight common wombats have been reported in the same burrow (Douglas, 1960). It appears that common wombats have adapted well to their environment at Padilpa. The small area of remnant vegetation for burrows demands that wombats share and occupy a limited number of burrows in order to utilise a large food resource. However wombats at Padilpa, Buccleuch State Forest and Cape Portland had aggressive disputes over use of burrows and wounds indicative of fighting (McIlroy, 1973; Taylor, 1993). It appears that there is a network of underground tunnels at Padilpa and probably numerous sleeping chambers enabling some degree of separation between wombats and therefore a reduction in disputation. In continuous forest, common wombats are generally regarded as territorial animals that maintain separate feeding areas (McIlroy, 1973). It is possible that common wombats are less territorial at Padilpa since the home ranges of wombats overlap entirely. If common wombats were to maintain their relatively solitary territorial nature at Padilpa then one would expect their density to be much lower. Wombats used multiple burrows over several months as reported by McIlroy (1976) and Taylor (1993). Some burrows were used preferentially for diurnal shelter, which was also reported by McIlroy (1976). Wombats frequently used the same burrow for several consecutive nights before changing to another. This was also true for the wombats at Cape Portland in the north-east of Tasmania (Taylor, 1993).

Reproduction
McIlroy (1973) considered that sexual maturity was reached around 22kg in weight. It is probable that females need to reach a greater weight than 22 kg before they can breed given that there was a weight loss of several kg associated with lactation in this study (Fig. 8.34). Large males appeared to sire most young in this study however a small male weighing 23 kg sired one of the young (Banks, unpublished observations). Reproduction at Padilpa is similar to most populations of common wombats in that births can occur at any time of the year (Triggs, 1996). However, most small pouch young weighing less than 200 g occurred between March and May at Padilpa and were probably born 3 months earlier. The wombats at Buccleuch State Forest are similar, with most births occurring from December to March (McIlroy, 1973). Adult males had a smaller weight to head length ratio than adult females. Body length was difficult to measure because wombats that were lightly anaesthetised did not have good muscle relaxation and would not lie in dorso-ventral recumbency.

Prevalences of ectoparasites, in particular S. scabiei

Ticks appear to be the most common ectoparasite of common wombats with the common wombat tick (Aponomma auruginans) being present on most wombats in this study, at Healesville, Morwell and Buccleuch State Forest (McIlroy, 1973; Smales, 1987; Skerratt, 1998). The tick Ixodes tasmani was much less prevalent occurring on approximately 10% of wombats and at much lower intensities with only a few individuals on one animal. A similar intensity and prevalence occurred on road-killed wombats around Healesville (Skerratt, 1998). Ixodes tasmani occurred on wombats at Buccleuch State Forest along with I. holocyclus (McIlroy, 1973). Ixodes cornuatus and I. victoriensis were also found in low numbers on wombats from
Healesville but were not present on wombats at Padilpa (Skerratt, 1998). Fleas, *Lycopsylla nova*, and lice, *Boopia tarsata*, were the next most common ectoparasites with prevalences higher at Padilpa than found on road-killed wombats around Healesville (Skerratt, 1998). This is possibly due to ectoparasites leaving cadavers, which were present on the sides of roads for variable periods in the study by Skerratt (1998). Similarly, the mite *Acaroptes vombatus*, which lives on the skin surface, was less prevalent on road-killed wombats from Healesville than wombats at Padilpa. The ears of wombats at Padilpa were not examined for mites but the ear mite *Rallietina australis* was common on road-killed wombats from Healesville (71% prevalence) (Skerratt, 1998). The prevalences of lice and fleas are underestimates since these parasites can be difficult to find in the fur of wombats and are usually only seen when intensities of infection are moderate to heavy unless an extensive examination is undertaken. Similarly, *Acaroptes* relies on examination of skin scrapings with a stereomicroscope and therefore only moderate to heavy infections are detected.

*Sarcoptes scabiei* was not found on the wombats at Padilpa although property owners in the area had reported seeing mangy wombats previously. If *S. scabiei* occurred at low prevalence at Padilpa then the mite could have easily escaped detection. The wombats at Padilpa are part of the continuous population of wombats that occur along the Great Dividing Range between Melbourne and Sydney in which sarcoptic mange is widespread (Martin et al. 1998). Sarcoptic mange appears to occur at low prevalence within common wombat populations, 4% (n=25) and 13% (n=7) in road-killed wombats from Healesville and Morwell, Victoria (Vic) respectively (Smales, 1987; Skerratt, 1998). Martin et al. (1998) reported slightly higher prevalences 22% (n=23) at Foster, Vic, 22% (n=9) Woodend, Vic, 15% (n was not
provided) Yarrowitch, New South Wales (NSW) and 0% (n=16) Warrigal, Vic. However the higher prevalences were reported by animal carers who may see a disproportionate number of sick animals (Martin, unpublished observations). A good example of bias in sampling occurred in 1992 when the veterinary department at Healesville Sanctuary collected 13 sick common wombats from the Healesville Shire, of which 62% had sarcoptic mange (Skerratt, 1998). In contrast only 4% (n=25) of road-killed wombats, which they collected that year, had sarcoptic mange (Skerratt, 1998). The estimates of 15% and 0% in Martin et al. (1998) were provided by biologists who had trapped free-living wombat populations and the estimates are less likely to be biased (Martin, unpublished observations). The prevalence of sarcoptic mange may change over time as demonstrated by a population at Tonimbuk, which exhibited a prevalence of 0% in 1996 and had a prevalence of 5% (n=40) in 2000 (Banks, unpublished observations). Buchan and Goldney (1998) reported that none of the wombats (n=9) at Yetholme showed signs of sarcoptic mange in the early 1990’s. At Buffalo River, Victoria, in March and May 2000, 4% (n=23) and 3% (n=32), respectively of common wombats shot had sarcoptic mange (Wolvekamp, unpublished observations). All reports of high prevalence of sarcoptic mange in free-living common wombats are anecdotal. Gray (1937) reported an epidemic of mange in wombats in NSW that severely reduced abundance. Martin et al. (1998) reported sporadic outbreaks of sarcoptic mange in common wombat populations associated with population declines. From 1997 to 2000, there were several reports of localised epidemics of mange in common wombats in NSW and Victoria, however, prevalences were not estimated (Skerratt, unpublished observations). There appeared to be a high prevalence of mange in wombats at Comaum, South Australia in 2000 (Banks, unpublished observations). Several factors have been suggested to cause a higher
prevalence of sarcoptic mange in free-living animals such as reduced resistance to infection, environmental stress such as drought or harsh winters, high host density and environmental conditions that favour mite survival and transmission (Martin et al. 1998). Resistance to infection has been shown to be important in domestic animals (Arlian et al., 1996a). The most important factor in wild populations appears to be a lack of resistance to infection since widespread epidemics of mange associated with severe population declines have been seen in naïve populations into which mange has been introduced (Mörner, 1984; Lindström and Mörner, 1985; Lindström, 1991; León-Vizcaino et al., 1999). Pence et al. (1983) also thought that waning resistance to infection was the reason for a second epidemic of sarcoptic mange in coyotes 45 years after the first. Cyclical scabies epidemics which occur in humans are thought to be due to changes in resistance (Burgess, 1994). Whilst epidemics of sarcoptic mange have been shown to affect host abundance, the effects of sarcoptic mange at low prevalence on population density are difficult to ascertain (Pence and Windberg, 1994; Gortázar et al., 1998). Studies on coyotes found that endemic sarcoptic mange had no discernible effect on population abundance (Pence and Windberg, 1994). It appeared that mange compensated for other mortality factors (Pence et al., 1983). In Spain, enzootic sarcoptic mange appears to currently affect the abundance of foxes (Gortázar et al., 1998). Enzootic sarcoptic mange may also have controlled the abundance of foxes in central New York in the 1970’s if they had not been extensively hunted (Tullar and Berchielle, 1981).

**Trapping**

The trapping success of common wombats in this study was one wombat caught for every 2.4 trap nights. The trapping success of common wombats at
Buccleuch State Forest and Braidwood in New South Wales (NSW) (McIlroy, 1977) was 3.7 trap nights/wombat and at Yetholme, NSW (Buchan and Goldney, 1998) it was 3.3 trap nights/wombat. However, the densities of wombats were lower by factors of 84 and 175, respectively (McIlroy, 1977; Buchan and Goldney, 1998). Wombats can be difficult to catch by placing cages over burrow entrances since a large proportion of burrows are empty at any one time. This is because common wombats use several burrows for diurnal shelter and even though wombats will share burrows at separate times or simultaneously, there are invariably more burrows than wombats (McIlroy, 1973; Taylor, 1993; Triggs, 1996; Buchan and Goldney, 1998). Another difficulty in trapping wombats is that they may be reluctant to enter a trap and will remain in a burrow for several nights before being caught or will dig under or around the trap. More trap escapes occurred in this study than at Buccleuch State Forest (McIlroy, 1973, 1976) possibly because the sandy alluvial soil was easier for digging than the red earth soils of Buccleuch State Forest. Direct population estimates rely on the assumption that most wombats will enter a trap rather than remain down a burrow. This appears to be a reasonable assumption given that most wombats were caught on the first night a trap was placed over their burrow in this study, at Buccleuch State Forest and at Yetholme with a decreasing proportion being caught on subsequent nights (McIlroy, 1976; Buchan and Goldney, 1998). Occasionally, wombats remained in burrows to avoid being trapped in both this study and at Buccleuch State Forest (McIlroy, 1976).

Radiotransmitters

It is possible that the behaviour of wombats was affected in the period immediately after the implantation of radio transmitters and the attachment of radio
transmitters to ear tags. However, any affect on behaviour was not apparent from the 
methods used to examine behaviour such as thread lines and radiolocation. There was 
an adjustment period to collars with radio-packages in northern hairy nosed wombats 
(*Lasiorhinus krefftii*) of 10 – 18 days (Woolnough et al., 1998). Trapping, anaesthesia 
and attachment of spools to wombats may have affected their behaviour but each 
wombat received the same treatment. It is possible that females reacted differently 
from males to the above interventions and this could explain the differences observed 
in their behaviour. Opportunistic observations on wombats revealed no obvious 
difference in behaviour between wombats that received the above treatments and 
those that did not. It is likely that the behaviour of wombats that rejected surgically 
implanted subcutaneous transmitters was affected but their behaviour was not 
examined. The rejection of surgically implanted subcutaneous transmitters has been 
reported previously in badgers (Agren et al., 2000). It was thought that the adherence 
of the skin to subcutaneous tissues resulted in pressure necrosis of tissues surrounding 
the transmitters. Similarly, wombats have skin that is tightly adherent to subcutaneous 
tissues. It may be possible to reduce the pressure on surrounding tissues by enlarging 
the subcutaneous pocket for the transmitter so that it is considerably larger than the 
transmitter. However, there could be an increased risk of infection due to the potential 
increased pooling of body fluids surrounding the transmitter because of the larger 
subcutaneous space. Intraperitoneal transmitters were tolerated well and were 
associated with minor pathological changes but complications occasionally occurred 
when transmitters lodged in vital areas in beavers (Guynn et al., 1987; Agren et al., 
2000). Intraperitoneal transmitters were prevented from moving around the peritoneal 
cavity of badgers for 3 months by suturing them to the abdominal wall with stainless 
steel. The stainless steel sutures holding intraperitoneal transmitters to the abdominal
walls of wombats broke, although they had been in place for six months compared with three months in badgers. The movement of the gut probably pushed the transmitter back and forth eventually breaking the sutures. Polypropylene would be a better suture material since it is less prone to cyclical stress than steel (Slatter, 1993). Attaching lightweight transmitters to ear tags has potential as a relatively easy and cheap method for short-term radio tracking of wombats.

In conclusion, common wombats that occur at high density may share burrows frequently and home ranges may overlap extensively facilitating the spread of sarcoptic mange. However, sarcoptic mange may not spread readily within a common wombat population at high density if *S. scabiei* var. *wombati* occurs at low densities. Common wombats that develop mild to moderate sarcoptic mange may increase the area that they use each night. However, they may enter fewer burrows at night and may move less frequently to another burrow for diurnal shelter thereby reducing the risk of spread of sarcoptic mange.
Table 8.1. Number of trap nights, number of wombats caught and the proportion of wombats caught on consecutive trap nights in this study at Padilpa, Victoria, compared with those of McIlroy (1976) at Buccleuch State Forest, New South Wales.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Trap nights</td>
<td>500</td>
<td>NA</td>
</tr>
<tr>
<td>No. of wombats caught</td>
<td>208</td>
<td>138</td>
</tr>
<tr>
<td>No. of wombats caught on 1\textsuperscript{st} night</td>
<td>133 (64%)</td>
<td>71%</td>
</tr>
<tr>
<td>No. of wombats caught on 2\textsuperscript{nd} night</td>
<td>54 (26%)</td>
<td>26%</td>
</tr>
<tr>
<td>No. of wombats caught on 3\textsuperscript{rd} night</td>
<td>14 (7%)</td>
<td>3%</td>
</tr>
<tr>
<td>No. of wombats caught on 4\textsuperscript{th} night</td>
<td>6 (3%)</td>
<td>1%</td>
</tr>
<tr>
<td>No. of wombats caught on 5\textsuperscript{th} night</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>No. of escapes</td>
<td>35</td>
<td>11</td>
</tr>
</tbody>
</table>

NA Not available.
Table 8.2. Age and sex of the common wombats (*Vombatus ursinus*) trapped at Padilpa, Victoria, in 1999/2000.

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pouch Young</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>Joey (&lt; 12kg)</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Juvenile (12-18kg)</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Sub-adult (18-22kg)</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Sub-total</td>
<td>19</td>
<td>14</td>
</tr>
<tr>
<td>Adult (&gt;22kg)</td>
<td>16</td>
<td>25</td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>39</td>
</tr>
</tbody>
</table>
Table. 8.3. Weight, head length, body length, length of manus and pes, length and width of testis and epididymis, age and sex classes of wombats (*Vombatus ursinus*) trapped at Padilpa, Victoria, from August 1999 to November 2000.

<table>
<thead>
<tr>
<th>Wombats*</th>
<th>Weight (kg)</th>
<th>Head Length (mm)</th>
<th>Manus Length (mm)</th>
<th>Pes Length (mm)</th>
<th>Body Length (mm)</th>
<th>Testis/Epididymis Length (mm)</th>
<th>Testis Width (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult Γ (n=17)</td>
<td>26.6 ± 0.6 (23.1 – 30.4)</td>
<td>215 ± 3 (197 – 238)</td>
<td>84.4 ± 1.0 (76.1 – 90.5)</td>
<td>98.3 ± 1.1 (88.2 – 105.1)</td>
<td>820 ± 10 (732 – 883)</td>
<td>46.8 ± 0.8 (41.0 – 55.0)</td>
<td>30.8 ± 0.4 (28.3 – 34.0)</td>
</tr>
<tr>
<td>Adult Ε (n=25)</td>
<td>28.3 ± 0.6 (22.0 – 34.6)</td>
<td>212 ± 1 (205 – 231)</td>
<td>83.7 ± 0.9 (77.0 – 94.7)</td>
<td>97.7 ± 0.9 (90.9 – 106.4)</td>
<td>836 ± 7 (765 – 915)</td>
<td>42.0 ± 0.8 (39.0 – 45.1)</td>
<td>28.1 ± 1.2 (23.5 – 32.0)</td>
</tr>
<tr>
<td>Sub-adults (7Γ, 4Ε)</td>
<td>19.8 ± 0.4 (18.2 – 21.8)</td>
<td>192 ± 3 (177 – 207)</td>
<td>78.8 ± 1.2 (70.0 – 84.5)</td>
<td>93.6 ± 1.2 (86.5 – 100.0)</td>
<td>740 ± 14 (700 – 790)</td>
<td>39.6 ± 1.6 (31.5 – 45.0)</td>
<td>27.2 ± 1.5 (23.0 – 37.0)</td>
</tr>
<tr>
<td>Juveniles (9Γ, 4Ε)</td>
<td>15.2 ± 0.5 (12.6 – 17.8)</td>
<td>180 ± 3 (166 – 198)</td>
<td>73.5 ± 1.2 (66.9 – 80.0)</td>
<td>86.6 ± 1.0 (79.4 – 94.0)</td>
<td>683 ± 9 (615 – 735)</td>
<td>39.6 ± 1.6 (31.5 – 45.0)</td>
<td>18.3 ± 1.8 (10.4 – 26.5)</td>
</tr>
<tr>
<td>Joeys (7Γ, 6Ε)</td>
<td>7.7 ± 1.0 (2.5 – 13.0)</td>
<td>147 ± 5 (116 – 166)</td>
<td>60.3 ± 2.8 (40.2 – 71.0)</td>
<td>71.1 ± 3.2 (49.4 – 85.5)</td>
<td>555 ± 28 (345 – 655)</td>
<td>26.2 ± 1.8 (17.7 – 33.4)</td>
<td>18.3 ± 1.8 (10.4 – 26.5)</td>
</tr>
<tr>
<td>PY (n=35)</td>
<td>0.7 ± 0.1 (0.0 – 2.8)</td>
<td>64.8 ± 5.0 (15.7 – 117.0)</td>
<td>25.2 ± 2.1 (6.0 – 51.0)</td>
<td>27.8 ± 2.5 (5.0 – 57.0)</td>
<td>NM</td>
<td>NM</td>
<td>NM</td>
</tr>
</tbody>
</table>

(means ± standard errors, range in parentheses)

*The measurements of wombats, which were recaptured and had grown significantly in the period between captures, were included.

The measurements of adult wombats, which were recaptured, were averaged. Several wombats, which were recaptured, had matured from one age class to another between captures.

PY Pouch young

NM Not measured
Table 8.4. Number of common wombats (*Vombatus ursinus*), study area, density of wombats and climate* at various locations in south-eastern Australia.

<table>
<thead>
<tr>
<th>Location</th>
<th>Wombats</th>
<th>Active Burrows</th>
<th>Area (ha)</th>
<th>Wombat Density (/ha)</th>
<th>Active Burrow Density (/ha)</th>
<th>Elevation (m)</th>
<th>Mean Annual Precipitation (mm)</th>
<th>Temperature Range (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Padilpa, Vic 2000</td>
<td>58</td>
<td>72</td>
<td>3</td>
<td>19.3(^a)</td>
<td>24</td>
<td>200 – 210</td>
<td>869</td>
<td>-1 – 38</td>
</tr>
<tr>
<td>Boho South, Vic 2000 &amp; 2001</td>
<td>3</td>
<td>16</td>
<td>60</td>
<td>0.1</td>
<td>0.3</td>
<td>570 – 730</td>
<td>986</td>
<td>-3 – 32</td>
</tr>
<tr>
<td>Cape Portland, Tas 1984 (Taylor, 1993)</td>
<td>IE, NA</td>
<td>NA</td>
<td>155</td>
<td>0.6 - 1.6</td>
<td>0.2</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Yetholme, NSW 1990’s (Buchan &amp; Goldney, 1998)</td>
<td>9</td>
<td>38</td>
<td>42</td>
<td>0.2</td>
<td>0.9</td>
<td>1105 – 1195</td>
<td>935</td>
<td>0 – 25</td>
</tr>
<tr>
<td>Buccleuch State Forest, NSW1970 Area B (McIlroy, 1973)</td>
<td>6</td>
<td>28</td>
<td>29</td>
<td>0.2</td>
<td>1.0</td>
<td>850 – 975</td>
<td>1170</td>
<td>-11 – 25</td>
</tr>
<tr>
<td>Buccleuch State Forest, NSW1970 Area E (McIlroy, 1973)</td>
<td>5</td>
<td>29</td>
<td>42</td>
<td>0.1</td>
<td>0.7</td>
<td>850 – 975</td>
<td>1170</td>
<td>-11 – 25</td>
</tr>
<tr>
<td>Braidwood, NSW1975 Area C (McIlroy, 1977)</td>
<td>10</td>
<td>121</td>
<td>71</td>
<td>0.1</td>
<td>1.7</td>
<td>630 – 670</td>
<td>676</td>
<td>NA</td>
</tr>
</tbody>
</table>

\(^a\)Climatic conditions were recorded at weather stations near to the localities at which wombats were studied: Glenburn and Eildon Fire Tower for Padilpa, Strathbogie for Boho South and Bondo for Buccleuch State Forest.

Pouch young excluded.

IE Indirect estimate.

NA Not available.

\(^a\)Density decreases by a factor of 10 to 1.9 wombats/ha when the pasture, which was grazed by wombats and surrounded the remnant vegetation, is included in estimate.

\(^b\)Range of burrow densities that occurred in different habitats.
Table 8.5. Numbers of burrows at Padilpa, Victoria that contained no wombats, one wombat or multiple wombats.*

<table>
<thead>
<tr>
<th>No. of wombats</th>
<th>Burrows (n=70)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>30 (43%)</td>
</tr>
<tr>
<td>1</td>
<td>11 (16%)</td>
</tr>
<tr>
<td>2</td>
<td>20 (29%)</td>
</tr>
<tr>
<td>3</td>
<td>7 (10%)</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>1 (1%)</td>
</tr>
</tbody>
</table>

*Pouch young excluded.
Table 8.6. Age, sex and reproductive status of common wombats (*Vombatus ursinus*) occupying the same burrow at Padilpa, Victoria.

<table>
<thead>
<tr>
<th>No. of Wombats</th>
<th>Age, Sex and Reproductive State</th>
<th>No. of Burrows</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Adult Γ, Adult Ε(PY)</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Adult Γ, Immature Γ</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>Adult Γ, Adult Γ</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Adult Γ, Adult Ε</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Adult Ε and Young at Heel Γ or Ε</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Immature Γ, Immature Ε</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Adult Γ, Adult Ε(Lactating)</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>Adult Ε(PY), Adult E</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>Adult Ε(PY), Immature Ε</td>
<td>1</td>
</tr>
<tr>
<td>2 + escape</td>
<td>Adult Γ, Immature Γ</td>
<td>1</td>
</tr>
<tr>
<td>2 + escape</td>
<td>Adult Γ, Immature Ε</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Adult Γ, Adult Ε and Young at Heel Γ</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Adult Γ, Adult Ε(PY), Adult Ε(PY)</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Adult Γ, Adult Γ, Adult Ε(PY)</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Adult Ε, Adult Ε(Lactating), Immature Γ</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Adult Ε, Adult Ε(PY), Immature Ε</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>Adult Γ, Adult Ε(PY), Adult Ε and Young at Heel Ε, Immature Ε</td>
<td>1</td>
</tr>
</tbody>
</table>

PY Pouch young.
Table 8.7. Number of times that a common wombat (Vombatus ursinus) was radio located in a burrow, number of different burrows used by a wombat and the range of the number of times that a wombat was radio located in each burrow at Padilpa, Victoria, from August 1999 to November 2000.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>No. of burrow fixes</th>
<th>No. of burrows</th>
<th>Range of fixes/burrow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female 1*</td>
<td>47</td>
<td>11</td>
<td>1 – 10</td>
</tr>
<tr>
<td>Female 2*</td>
<td>49</td>
<td>8</td>
<td>1 – 19</td>
</tr>
<tr>
<td>Female 3*</td>
<td>17</td>
<td>6</td>
<td>1 – 8</td>
</tr>
<tr>
<td>Male 1</td>
<td>41</td>
<td>10</td>
<td>1 – 17</td>
</tr>
<tr>
<td>Male 2</td>
<td>50</td>
<td>12</td>
<td>1 – 18</td>
</tr>
</tbody>
</table>

*With young.
Table 8.8. Precipitation (mm) at Glenburn (37°22′53″S, 145°28′20″E), elevation 250 m, and average maximum air temperatures (°C) at Eildon Fire Tower (37°12′39″S, 145°50′27″E), elevation 638 m, for each month from August 1999 to November 2000. Padilpa was close to both these sites at (37°22′6″S, 145°28′30″E), elevation 200 m.

<table>
<thead>
<tr>
<th>Year</th>
<th>Month</th>
<th>Precipitation (mm)</th>
<th>Mean Maximum Air Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999</td>
<td>August</td>
<td>139</td>
<td>11.1</td>
</tr>
<tr>
<td>1999</td>
<td>September</td>
<td>53.4</td>
<td>14.8</td>
</tr>
<tr>
<td>1999</td>
<td>October</td>
<td>83.6</td>
<td>16.7</td>
</tr>
<tr>
<td>1999</td>
<td>November</td>
<td>57.2</td>
<td>18.8</td>
</tr>
<tr>
<td>1999</td>
<td>December</td>
<td>85.8</td>
<td>22.4</td>
</tr>
<tr>
<td>2000</td>
<td>January</td>
<td>32.8</td>
<td>23.4</td>
</tr>
<tr>
<td>2000</td>
<td>February</td>
<td>29.6</td>
<td>29.1</td>
</tr>
<tr>
<td>2000</td>
<td>March</td>
<td>20.6</td>
<td>23.9</td>
</tr>
<tr>
<td>2000</td>
<td>April</td>
<td>52.6</td>
<td>18.6</td>
</tr>
<tr>
<td>2000</td>
<td>May</td>
<td>144.2</td>
<td>11.7</td>
</tr>
<tr>
<td>2000</td>
<td>June</td>
<td>74.3</td>
<td>8.9</td>
</tr>
<tr>
<td>2000</td>
<td>July</td>
<td>96</td>
<td>8.6</td>
</tr>
<tr>
<td>2000</td>
<td>August</td>
<td>116.8</td>
<td>10.4</td>
</tr>
<tr>
<td>2000</td>
<td>September</td>
<td>127</td>
<td>12.8</td>
</tr>
<tr>
<td>2000</td>
<td>October</td>
<td>103.6</td>
<td>14.7</td>
</tr>
<tr>
<td>2000</td>
<td>November</td>
<td>116.6</td>
<td>21.3</td>
</tr>
</tbody>
</table>
Table 8.9. Mean distance travelled and area used per night by common wombats (*Vombatus ursinus*) affected or not affected by sarcoptic mange at Padilpa, Victoria, in 2000.

<table>
<thead>
<tr>
<th>Month</th>
<th>No. of measurements</th>
<th>Not Mangy (No. of wombats used=15)</th>
<th>Mangy (No. of wombats used=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Distance (m)</td>
<td>Area* (Ha)</td>
</tr>
<tr>
<td>March</td>
<td>6</td>
<td>885 ± 111b</td>
<td>1.33 ± 0.27c</td>
</tr>
<tr>
<td>May</td>
<td>6</td>
<td>591 ± 168ab</td>
<td>1.55 ± 0.65d</td>
</tr>
<tr>
<td>July</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>August</td>
<td>6</td>
<td>510 ± 137a</td>
<td>0.34 ± 0.15e</td>
</tr>
<tr>
<td>October</td>
<td>6</td>
<td>533 ± 112a</td>
<td>0.53 ± 0.18c</td>
</tr>
</tbody>
</table>

abc Values sharing a letter were not significantly different.

* Minimum convex polygon used to determine area.
Table 8.10. Distances travelled and the total area used per night for male and female common wombats (*Vombatus ursinus*) at Padilpa, Victoria, in 2000.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Weight (Kg)</th>
<th>Lactating</th>
<th>Nights</th>
<th>Mean Distance (m)</th>
<th>Min.-Max. Distance (m)</th>
<th>Total Distance (m)</th>
<th>Area† (Ha)</th>
<th>Range Span (m)</th>
<th>No. of fixes of location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>Adult</td>
<td>25</td>
<td></td>
<td>8</td>
<td>811</td>
<td>200-1340</td>
<td>5980</td>
<td>6.13</td>
<td>372</td>
<td>286</td>
</tr>
<tr>
<td>Male</td>
<td>Adult</td>
<td>30</td>
<td></td>
<td>5</td>
<td>1315</td>
<td>1200-1440</td>
<td>5610</td>
<td>8.3</td>
<td>467</td>
<td>266</td>
</tr>
<tr>
<td>Male</td>
<td>Adult</td>
<td>30</td>
<td></td>
<td>3</td>
<td>919</td>
<td>439-1567</td>
<td>2756</td>
<td>7.33</td>
<td>651</td>
<td>121</td>
</tr>
<tr>
<td>Male</td>
<td>Sub-adult</td>
<td>19</td>
<td></td>
<td>5</td>
<td>530</td>
<td>181-988</td>
<td>2649</td>
<td>2</td>
<td>186</td>
<td>149</td>
</tr>
<tr>
<td>Female</td>
<td>Adult</td>
<td>27</td>
<td>Yes</td>
<td>6</td>
<td>684</td>
<td>280-1220</td>
<td>3420</td>
<td>4.16</td>
<td>417</td>
<td>184</td>
</tr>
<tr>
<td>Female</td>
<td>Adult</td>
<td>29</td>
<td>Yes</td>
<td>9</td>
<td>496</td>
<td>200-880</td>
<td>4155</td>
<td>2.38</td>
<td>225</td>
<td>213</td>
</tr>
<tr>
<td>Female</td>
<td>Adult</td>
<td>27</td>
<td>Yes</td>
<td>6</td>
<td>651</td>
<td>360-865</td>
<td>2846</td>
<td>3.7</td>
<td>325</td>
<td>146</td>
</tr>
<tr>
<td>Female</td>
<td>Adult</td>
<td>26</td>
<td>Yes</td>
<td>8</td>
<td>639</td>
<td>440-785</td>
<td>3833</td>
<td>4.95</td>
<td>307</td>
<td>234</td>
</tr>
</tbody>
</table>

Fixes of location were taken every 20 m along the thread line left by wombats to which spools were attached. The total distance travelled does not match the number of fixes multiplied by 20 because the number of fixes includes spooling events that only partially worked, i.e. when bobbins stopped unwinding or the thread line was lost, and fixes were not taken when there were breaks in the thread line and the path of the wombat was not obvious between the broken ends of the thread.

†Measured by convex polygon
Table 8.11. Time of emergence from a burrow for common wombats (*Vombatus ursinus*) at Padilpa, Victoria, during the summer of 1999/2000.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>Nights</th>
<th>Mean ± SE (hrs.mins)</th>
<th>Range (hrs.mins)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male 2</td>
<td>6</td>
<td>8.54 ± 0.07 p.m.</td>
<td>8.40 – 9.20 p.m.</td>
</tr>
<tr>
<td>Male 3</td>
<td>4</td>
<td>8.58 ± 0.11 p.m.</td>
<td>8.30 – 9.20 p.m.</td>
</tr>
<tr>
<td>Mean (Males)</td>
<td>10</td>
<td>8.56 ± 0.06* p.m.</td>
<td>8.30 – 9.20 p.m.</td>
</tr>
<tr>
<td>Female 1</td>
<td>9</td>
<td>9.35 ± 0.19 p.m.</td>
<td>8.50 – 11.35 p.m.</td>
</tr>
<tr>
<td>Female 2</td>
<td>6</td>
<td>9.08 ± 0.12 p.m.</td>
<td>8.40 – 10.00 p.m.</td>
</tr>
<tr>
<td>Female 4</td>
<td>5</td>
<td>9.31 ± 0.16 p.m.</td>
<td>9.00 – 10.30 p.m.</td>
</tr>
<tr>
<td>Mean (Females)</td>
<td>20</td>
<td>9.26 ± 0.10* p.m.</td>
<td>8.40 – 11.35 p.m.</td>
</tr>
</tbody>
</table>

*Means were significantly different (*P*<0.05, Student t-test).

Individuals did not differ significantly in their time of emergence (*P*>0.05, ANOVA).

Dusk occurred between 8.30 and 9.00 p.m.
Table 8.12. Prevalence of ectoparasites on common wombats at Padilpa, Victoria, compared with other sites in Victoria: Morwell, Gippsland (Smales, 1982) and Healesville (Skerratt, 1998), as well as Buccleuch State Forest, New South Wales (McIlroy, 1973).

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>TICKS (Ixodoidea)</td>
<td>97%</td>
<td></td>
<td>71%</td>
<td>100%</td>
<td>93%</td>
</tr>
<tr>
<td>Aponomma auruginans</td>
<td>NS</td>
<td></td>
<td>71%</td>
<td>100%</td>
<td>93%</td>
</tr>
<tr>
<td>Ixodes tasmani</td>
<td>NS</td>
<td></td>
<td>NS</td>
<td>13%</td>
<td>12%</td>
</tr>
<tr>
<td>FLEAS (Siphonaptera)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lycopsylla nova</td>
<td>NS</td>
<td></td>
<td>NS</td>
<td>13%</td>
<td>25%</td>
</tr>
<tr>
<td>LICE (Phthiraptera)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boopia tarsata</td>
<td>NS</td>
<td></td>
<td>NS</td>
<td>16%</td>
<td>32%</td>
</tr>
<tr>
<td>MITES (Astigmata)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acaroptes vombatus</td>
<td>NS</td>
<td></td>
<td>NS</td>
<td>13%</td>
<td>23%</td>
</tr>
<tr>
<td>Sarcoptes scabiei</td>
<td>5%</td>
<td></td>
<td>13%</td>
<td>4%</td>
<td>0%</td>
</tr>
</tbody>
</table>

NS Not stated.

Pouch young excluded.
Table 8.13. Numbers of *Sarcoptes scabiei* var. *wombati* used to infect common wombats (*Vombatus ursinus*) at Padilpa, Victoria, and the intensity of infection on the shoulders of infected wombats 50 and 100 days after infection and 28 days after treatment with an acaricide, ivermectin.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>No. of infecting mites</th>
<th>Mites on shoulder (/cm²)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DAI 0</td>
<td>DAI 50</td>
<td>DAI 100</td>
</tr>
<tr>
<td>Female 1*</td>
<td>6000</td>
<td>0.9</td>
<td>0.3</td>
</tr>
<tr>
<td>Female 2*</td>
<td>8000</td>
<td>2.3</td>
<td>18</td>
</tr>
<tr>
<td>Male 1</td>
<td>4000</td>
<td>1.0</td>
<td>19</td>
</tr>
<tr>
<td>Male 2</td>
<td>8000</td>
<td>0.4</td>
<td>0.2</td>
</tr>
<tr>
<td>Female 3*</td>
<td>8000</td>
<td>-</td>
<td>0.04</td>
</tr>
<tr>
<td>Mean</td>
<td>7000</td>
<td>1.2</td>
<td>7.5</td>
</tr>
</tbody>
</table>

*With young.

DAI Days after infection.

DAT Days after treatment.
Table 8.14. Average severity of clinical signs of sarcoptic mange, parakeratosis (scale crust), hair loss and erythema in common wombats (*Vombatus ursinus*) at Padilpa, Victoria, infected with *Sarcoptes scabiei* var. *wombati* before and after treatment.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>Erythema (%)</th>
<th>Parakeratosis (mm)</th>
<th>Hair loss (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>50 DAI</td>
<td>100 DAI (treatment)</td>
<td>28 DAT</td>
</tr>
<tr>
<td>Female 1*</td>
<td>60</td>
<td>60</td>
<td>50</td>
</tr>
<tr>
<td>Female 2*</td>
<td>40</td>
<td>80</td>
<td>80</td>
</tr>
<tr>
<td>Male 1</td>
<td>40</td>
<td>80</td>
<td>80</td>
</tr>
<tr>
<td>Male 2</td>
<td>60</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>Female 3*</td>
<td>DNT</td>
<td>50</td>
<td>60</td>
</tr>
<tr>
<td>Median</td>
<td>50</td>
<td>60</td>
<td>50</td>
</tr>
</tbody>
</table>

DAI Days after infection.

DAT Days after treatment.

DNT Did not trap.

*Had young.

The degree of erythema is described as the percentage of body sections (n=10) affected (Fig. 7.3).

The severity of parakeratosis is the median category thickness (0, <1, 1-5 or >5 mm) of parakeratotic scale for all body sections.

The severity of hair loss is the median category of hair loss (0, <25, 25-50 or >75 %) for all body sections.
Table 8.15. Change in weight of common wombats (*Vombatus ursinus*) prior to infection with *Sarcoptes scabiei* var. *wombati*, after infection and after treatment with ivermectin at 400-800 µg/kg at Padilpa, Victoria.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>Prior to infection</th>
<th>Weight (kg)</th>
<th>After infection</th>
<th>After treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>31st Mar</td>
<td>10th May</td>
<td>Change</td>
<td>1st July†</td>
</tr>
<tr>
<td>Female 1*</td>
<td>28</td>
<td>27.4</td>
<td>-0.6</td>
<td>26</td>
</tr>
<tr>
<td>Female 2*</td>
<td>28</td>
<td>27.9</td>
<td>-0.1</td>
<td>26.2</td>
</tr>
<tr>
<td>Male 1</td>
<td>25.5</td>
<td>23.5</td>
<td>-2</td>
<td>24.3</td>
</tr>
<tr>
<td>Male 2</td>
<td>31.3</td>
<td>29.8</td>
<td>-1.5</td>
<td>29</td>
</tr>
<tr>
<td>Female 3*</td>
<td>27.6</td>
<td>27.6</td>
<td>0</td>
<td>26.5</td>
</tr>
<tr>
<td>Mean</td>
<td>28</td>
<td>27.2</td>
<td>-0.8a</td>
<td>26.4</td>
</tr>
</tbody>
</table>

*With young.

*aSignificant change (*P*<0.05).

†Infected wombats with mites.

™Treated wombats with ivermectin.

NM Not measured.

ND Not determined.
Table 8.16. Ratio of the number of nights a common wombat (*Vombatus ursinus*) was located versus the number of different burrows used and the ratio of the number of consecutive nights a wombat was located versus the number of times the wombat changed burrows at Padilpa, Victoria.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>Nights/ burrows</th>
<th>Consecutive nights/change of burrow</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Uninfected</td>
<td>Infected</td>
</tr>
<tr>
<td>Female 1*</td>
<td>2.9 (29)&lt;sup&gt;†&lt;/sup&gt;</td>
<td>3.6 (18)</td>
</tr>
<tr>
<td>Female 2*</td>
<td>4.1 (29)</td>
<td>5.0 (20)</td>
</tr>
<tr>
<td>Male 1</td>
<td>2.7 (27)</td>
<td>3.5 (14)</td>
</tr>
<tr>
<td>Male 2</td>
<td>4.1 (29)</td>
<td>2.9 (21)</td>
</tr>
<tr>
<td>Mean</td>
<td>3.5 (114)</td>
<td>3.7 (73)</td>
</tr>
</tbody>
</table>

*With young.

†Number of nights in brackets.
Table 8.17. Location in body and persistence of radio transmitters in common wombats (*Vombatus ursinus*) at Padilpa, Victoria, and extent of acceptance or rejection by wombats.

<table>
<thead>
<tr>
<th>Location</th>
<th>Total</th>
<th>Accepted</th>
<th>Rejected</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Months</td>
<td>No.</td>
</tr>
<tr>
<td>Sub-cutaneous</td>
<td>12</td>
<td>4 (1-13)*</td>
<td>4</td>
</tr>
<tr>
<td>Intraperitoneal</td>
<td>6</td>
<td>4 (1-8)</td>
<td>3</td>
</tr>
<tr>
<td>Ear Tag</td>
<td>5</td>
<td>2 (1-3)</td>
<td>5</td>
</tr>
</tbody>
</table>

*Means, range in parentheses.*
Table 8.18. Number of *Sarcoptes scabiei* var. *wombati* in a 25 cm$^2$ area of the shoulder of free-living common wombats at Padilpa 0, 50 and 100 days after infection compared with a model’s (see Chapter 6 and Fig. 8.35) predictions of population growth of *S. scabiei* var. *wombati*.

<table>
<thead>
<tr>
<th>Wombat</th>
<th>Mites on shoulder (/25 cm$^2$)</th>
<th>DAI 0</th>
<th>DAI 50</th>
<th>DAI 100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female 2*</td>
<td></td>
<td>50</td>
<td>115</td>
<td>450</td>
</tr>
<tr>
<td>Male 1</td>
<td></td>
<td>50</td>
<td>25</td>
<td>475</td>
</tr>
<tr>
<td>Mean of Female 2, Male 1</td>
<td></td>
<td>50</td>
<td>70</td>
<td>468</td>
</tr>
<tr>
<td>Model’s Prediction (Fig. 8.35, $\lambda=1.05$)$^\dagger$</td>
<td></td>
<td>50</td>
<td>129</td>
<td>438</td>
</tr>
<tr>
<td>Female 1*</td>
<td></td>
<td>50</td>
<td>23</td>
<td>8</td>
</tr>
<tr>
<td>Male 2</td>
<td></td>
<td>50</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Female 3*</td>
<td></td>
<td>50</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Mean of Female 1 &amp; 3, Male 2</td>
<td></td>
<td>50</td>
<td>17</td>
<td>5</td>
</tr>
<tr>
<td>Model’s Prediction (Fig. 8.35, $\lambda=0.97$)$^\dagger$</td>
<td></td>
<td>50</td>
<td>17</td>
<td>8</td>
</tr>
</tbody>
</table>

*With young.

DAI Days after infection.

DAT Days after treatment.

$^\dagger$ Adult males of *S. scabiei* var. *wombati* have been included in the model.
Figure 8.1. Field site at Padilpa, Victoria, in spring 2000 showing strip of riparian vegetation and adjacent pasture.

Figure 8.2. Adult, male common wombat (*Vombatus ursinus*) (Male 2) sitting near burrow entrance during the day at Padilpa, Victoria.
Figure 8.3. Escape tunnel, dug around trap which was placed over the entrance to the burrow at Padilpa, Victoria.

Figure 8.4. Damaged trap caused by a common wombat (*Vombatus ursinus*) that escaped by breaking the welds of the iron mesh and pushing the swinging gate past the angle iron that was designed to stop the gate swinging out.
Figure 8.5. Joey, female common wombat (*Vombatus ursinus*), in trap at Padilpa, Victoria.

Figure 8.6. Earliest stage of development of a joey (*Vombatus ursinus*) seen at Padilpa, Victoria, in 2000.

![Engorged adult female ticks](image)

Figure 8.8. Adult, male common wombat (*Vombatus ursinus*) with a torn cloaca and bite wounds at Padilpa, Victoria, in 2000.

![Adult male common wombat](image)
Figures 8.9. Surgical wound that enabled implantation of a radio transmitter in the ventral neck region of an adult, male common wombat (*Vombatus ursinus*) (Male 2).

Figure 8.10. Bulge in ventral neck region due to a subcutaneous radio transmitter and surgical wound created during implantation, closed with simple interrupted stiches of nylon suture material in an adult, male common wombat (*Vombatus ursinus*) (Male 2).
Figures 8.11. Radio transmitter glued to a circular ear tag in the right ear of an adult, female common wombat (*Vombatus ursinus*) (Female 2).
Figure 8.12. Hole, 3 m deep, created when digging with a shovel to recover a radio transmitter lost in a wombat burrow 14 m from its entrance at Padilpa, Victoria.

Figure 8.13. Back hoe used to recover radio transmitters from wombat burrows at Padilpa, Victoria.
Figure 8.14. Spool containing cotton bobbins stuck with epoxy resin to the fur of an adult, male common wombat (*Vombatus ursinus*) (Male 1).

Figure 8.15. Thread line distributed by a common wombat (*Vombatus ursinus*) with a spool glued to its fur.
Figure 8.16. Scratchings and scats used by common wombats (*Vombatus ursinus*) to mark territory at Padilpa, Victoria.
Figure 8.17. Body surface of wombats, divided into 13 sections in order to quantify the extent of sarcoptic mange in wombats.

There were sections that were duplicated on each side of the body such as on the face (Sections 2 and 10), the foreleg (4 and 11), the flank (6 and 12) and the hindleg (9 and 13) and five sections that included both sides of the body, forehead (1), back (5), rump (8), abdomen (7) and between the shoulders (3). These areas were chosen since the severity of clinical signs differed between but was similar within them in the study of free-living common wombats with severe sarcoptic mange (Chapter 3, Skerratt et al., 1999).
Figure 8.18. Number of common wombats (*Vombatus ursinus*) captured along the Yea River at Padilpa, Victoria, compared with the number of wombats caught the first night a trap was placed over a burrow.
Figure 8.19. Number of common wombats (*Vombatus ursinus*) that were captured along the Yea River at Padilpa, Victoria, versus the number of wombats known to exist in the population.
Figure 8.20. Weight (kg) of pouch young of common wombats (*Vombatus ursinus*) trapped at Padilpa, Victoria, from August 1999 to November 2000.
Figure 8.21. Weight (kg) versus head length (mm) for adult male and female common wombats (*Vombatus ursinus*) trapped at Padilpa, Victoria, from August 1999 to November 2000.

The measurements for wombats, which were recaptured, were averaged. Female: Weight = 0.33 x Head Length – 42, $r^2=0.52$, $P<0.05$, n=25, Male: Weight = 0.18 x Head Length – 12, $r^2=0.66$, $P<0.05$, n=17.
Figure 8.22. Growth of joeys (kg) (*Vombatus ursinus*) trapped at Padilpa, Victoria, from August 1999 to November 2000.

Weight of joeys above 5 kg not shown but projected linear growth rates shown.
Figure 8.23. Map of field site at Padilpa, Victoria, showing location of common wombat (*Vombatus ursinus*) burrows along the Yea River.
Figure 8.24. Map of field site at Padilpa, Victoria, showing area used by three adult male common wombats (*Vombatus ursinus*).
Figure 8.25. The number of times that the location of common wombats (*Vombatus ursinus*) was determined (fixes) compared with the total area used by wombats at Padilpa, Victoria, between December 1999 and November 2000.

Fixes on location were taken at 20 m intervals along spooling thread lines. Each displayed point represents a night of “spooling”.

---

**Graph Description:**

- The x-axis represents the number of fixes (ranging from 0 to 300).
- The y-axis represents the area (in hectares) ranging from 0 to 9.
- Various lines and markers represent different sections of the Padilpa area, labeled as Γ1, Γ2, Γ3, Γ4, Ε1, Ε2, Ε3, E4.
Figure 8.26. Map of field site at Padilpa, Victoria showing area used by two adult female common wombats (*Vombatus ursinus*).
Figure 8.27. Map of field site at Padilpa, Victoria showing area used by an adult female common wombat (*Vombatus ursinus*).
Figure 8.28. Map of field site at Padilpa, Victoria showing area used by an adult female and sub-adult male common wombat (*Vombatus ursinus*).
Figure 8.29. Anaesthetised adult, male common wombat (*Vombatus ursinus*) (Male 1) with sarcoptic mange 100 days after infection with 4000 *Sarcoptes scabiei*.

Figure 8.30. Anaesthetised adult, male common wombat (*Vombatus ursinus*) (Male 2) with sarcoptic mange 100 days after infection with 8000 *Sarcoptes scabiei*. 
Figure 8.31. Anaesthetised adult, female common wombat (*Vombatus ursinus*) (Female 1) with sarcoptic mange 100 days after infection with 6000 *Sarcoptes scabiei*.

Figure 8.32. Anaesthetised adult, female common wombat (*Vombatus ursinus*) (Female 2) with sarcoptic mange 100 days after infection with 8000 *Sarcoptes scabiei*. 
Figure 8.33. Anaesthetised adult, female common wombat (*Vombatus ursinus*) (Female 3) with sarcoptic mange 100 days after infection with 8000 *Sarcoptes scabiei.*
Figure 8.34. Weight loss (kg) of lactating female common wombats (*Vombatus ursinus*) with young at heel at Padilpa, Victoria, between August 1999 and November 2000.
Figure 8.35. Population trajectories for density of *Sarcoptes scabiei* on free-living wombats when 30 larvae, 10 nymphs and 5 females occurred in a 25 cm$^2$ area on 0 days after infection on the shoulder and $\lambda=1.05$ and $\lambda=0.97$ for a 2 day time interval, respectively.
Sarcoptic mange represents a threat for wombat populations. The effects of enzootic sarcoptic mange are difficult to quantify, but epizootics of sarcoptic mange certainly have the ability to dramatically reduce the abundance of wombats (Martin et al., 1998, Chapter 2; Skerratt et al., 1998, Chapter 1). In agricultural areas where wombats are pests, epizootics of sarcoptic mange in wombats may be viewed by farmers as beneficial. However, epizootics of sarcoptic mange together with other concomitant causes of mortality, may lead to the extirpation of small, isolated wombat populations (Caughley and Gunn, 1996). This would generally be regarded as an undesirable outcome.

Although sarcoptic mange does not occur in the endangered northern hairy-nosed wombat (Lasiorhinus krefftii), there are concerns about its possible introduction (Martin et al., 1998; Skerratt et al., 1998; Gerhardt et al., 2000). Recently, consideration was given to erecting a boundary fence around the last population of northern hairy-nosed wombats at Epping Forest, Qld, in order to exclude predators such as dingoes and foxes but also to prevent the introduction of S. scabiei by these same predators (A. Horsup, in litt 2000).

Sarcoptic mange is uncommon in the southern hairy-nosed wombat (Lasiorhinus latifrons) but may be prevalent during droughts (Southcott, 1976; Martin
et al., 1998; Skerratt et al., 1998). Subsequent to the survey of the distribution of sarcoptic mange in wombat populations (Chapter 2), mange has been reported to occur in southern hairy-nosed wombats at two new localities, the Nullarbor Plain and the Gawler Ranges (I. Hough, personal communication; M. Heide, personal communication). If confirmed, these reports would substantially increase the known distribution of sarcoptic mange in southern hairy-nosed wombat populations. The only definitive report of isolation and identification of *S. scabiei* from a southern hairy-nosed wombat is that of Southcott (1976). The wombat came from Blanche Town, SA and had signs of sarcoptic mange in March of 1976 (Southcott, 1976). There is some doubt surrounding the report by R. Domrow of *S. scabiei* from a southern hairy-nosed wombat since the host does not occur at the locality cited, Robe, SA (D. Lee *in litt.* 1973) (Domrow, 1992). Rather the host is likely to have been *V. ursinus* since sarcoptic mange has been reported from common wombats at Robe (Martin et al., 1998). Wells (1971) reported occasional outbreaks of mange in southern hairy-nosed wombats but did not collect mites or record the localities of infected wombats.

Concern about the impact of sarcptic mange on common wombats comes from various members of the community including wildlife carers, naturalists and rural landowners (Skerratt, unpublished observations), who associate an outbreak of sarcptic mange with a decline in the abundance of their local common wombat population. A recent attempt to reduce the effects of sarcptic mange in common wombats (*Vombatus ursinus*) near Buckety, New South Wales (NSW) has utilised repeated treatments of wombats with mild to moderate sarcptic mange with acaricides and euthanasia and disposal of wombats with severe sarcptic mange (D. Breen, personal communication). A similar management regime was used to halt an
epizootic of notoedric mange in white-nosed coatis (*Nasua narica*) in western Mexico (Valenzuela et al., 2000). These circumstances are unusual and generally, wildlife carers and rural landowners do not have the resources or expertise to deal with sarcoptic mange in their local wombat population.

Most conservation authorities do not regard sarcoptic mange as a threat to the conservation of common wombats. Although sarcoptic mange is widespread in common wombats, this species is abundant in Victoria, New South Wales (NSW) and Tasmania and are regarded as pests in many agricultural areas of these states (Martin et al., 1998; Temby, 1998). Common wombats are rare in Queensland (Qld) and South Australia (SA) (Triggs, 1996; Temby, 1998) yet Queensland provides only limited resources to study the isolated population of wombats near its border with NSW at Stanthorpe (D. Hoolihan, *in litt* 1999). South Australia, a state in which common wombats are regarded as vulnerable (Temby, 1998), is the only state that has funded a survey of the distribution and prevalence of sarcoptic mange in wombats (Chapter 2, Martin et al., 1998).

At present, epizootic sarcoptic mange is regarded as having a greater impact on common wombat abundance than enzootic sarcoptic mange (Martin et al., 1998, Chapter 2; Skerratt et al., 1998, Chapter 1). A major risk factor for an epizootic is naivety of the host population (Chapter 7). Other factors likely to predispose a population to an epizootic are environmental stresses such as drought or habitat degradation, high population densities and climatic factors that favour mite survival such as relatively low temperatures and high humidity (Martin et al., 1998, Chapter 2). An epizootic could be prevented by recognition of the presence of risk factors in a
population and implementation of control measures for sarcoptic mange whilst the potential epizootic is at an early stage.

Clinical signs of sarcoptic mange are erythema, parakeratosis and hair loss and these are correlated highly with intensity of infection (Skerratt et al., 1999, Chapter 3, 4). Trauma from fighting and heavy parasitism with other mites such as Acarooptes vombatus can have similar clinical signs to sarcoptic mange such as alopecia (Fig. 9.1) (Skerratt, unpublished observations). The presence of sarcoptic mange in wombat populations usually becomes obvious when wombats with severe mange start feeding during the day (Skerratt et al., 1999, Chapter 3). This may be too late to enable effective control of an outbreak of sarcoptic mange. Spotlighting at night will detect early cases of sarcoptic mange since the clinical signs of hair loss and parakeratotic scale are highly visible as white patches on the anterior lateral areas of the wombat (Skerratt et al., 1999, Chapter 3, 4). Clinical signs are usually symmetrical so viewing one side of the wombat is sufficient to assess its mange status (Skerratt et al., 1999, Chapter 3). The location of the hair loss generally differentiates sarcoptic mange from trauma due to fighting which results in hair loss on the rump, back and forehead (Fig. 9.1) (Skerratt et al., 1999, Chapter 3, 4, 8). The diagnosis of sarcoptic mange should be confirmed by identification of the mite S. scabiei var. wombati from either skin scrapings or parakeratotic crusts. Mites occur in highest abundance in antero-lateral areas such as the shoulders, forelegs and chest (Skerratt et al., 1999, Chapter 3). At present the collections and records of S. scabiei var. wombati from wombats are few (Domrow, 1992).
In the past, techniques for managing sarcoptic mange in wombat populations have been confined to the culling of severely affected animals. These animals have been culled mainly for welfare reasons because of the obvious debilitating effects of severe sarcoptic mange (Skerratt, 1998). However, since severely affected animals are a major source of mites (Skerratt et al., 1999, Chapter 3) culling may also reduce the transmission rate of mites and hence reduce the spread of sarcoptic mange.

Any control measures in addition to culling of wombats with severe sarcoptic mange should consider the mechanism of transmission of mites between wombats and the conditions under which transmission is most likely to occur. It appears that mites are deposited in a burrow by a mangy wombat and then picked up by other wombats that use the burrow. There is no direct evidence for this, but wombats generally avoid close contact with one another when above ground, yet will readily share burrows with certain individuals (Chapter 8). Burrows enhance the survival of mites because they provide a relatively stable climate with low temperatures and high humidities (Skerratt et al., 1998, Chapter 1). However, it appears that intensity of infection must be relatively high before mites are actually deposited in a burrow and can infect another wombat (Chapter 8). Therefore, reducing the intensity of infection of *S. scabiei* in a wombat population may reduce transmission of the mite. Sarcoptic mange has been reported to be more prevalent in wombat populations in winter (Skerratt et al., 1998, Chapter 1). These observations were probably based on the number of wombats with severe sarcoptic mange. Severe infections may take three months to develop depending on a variety of factors such as initial infective dose of mites, body condition of the host and immunity of the host (Chapter 4, 5, 6). Therefore, a high prevalence of sarcoptic mange in winter is probably due to the spread of the disease in
autumn. The disease may spread in autumn due to better conditions for mite survival, lower temperatures and higher humidity, and because wombats may be in relatively poor body condition due to less feed being available over summer (Chapter 8). Any reduction in prevalence and intensity of infection in autumn will dramatically reduce the impact of the disease in winter. As occurs with nematode infections in sheep and cattle in south-eastern Australia, where rainfall mainly occurs in winter and spring, summer is the best time to reduce prevalence and intensity of infection (Cole, 1986). This is because *S. scabiei* will have its shortest survival in the burrow in summer when temperatures are higher and humidity lower (Chapter 8). Correspondingly, infection rates and intensities will be lower during autumn because of control measures implemented in summer (Cole, 1986).

It appears that mites infect the left and the right sides of the wombat simultaneously (Skerratt et al., 1999, Chapters 3, 4). This may be because wombats are infected by sharing sleeping chambers where they continually change their resting position (Triggs, 1996) enabling both sides of the body to be infected by mites on the floor of the sleeping chamber. Currently there is no method for treating burrows in order to eliminate mites. This is because burrows are long and tortuous, often 10 to 30 m in length (Nicholson, 1963; McIlroy, 1973; Steele and Temple-Smith, 1998; Chapter 8), and achieving concentrations of an acaricide which will kill mites throughout the burrow, especially in sleeping chambers, is impractical. In addition, there is the problem of ensuring that wombats do not suffer from associated chemical toxicities.
The most effective method for reducing mite abundance is to treat wombats with an acaricide. There are several acaricides which are easily applied topically such as ivermectin, moxidectin and selamectin (Havas Medimedia, 2001). However these topical applications may not be totally effective if the wombat has moderate to severe sarcoptic mange. This is because parakeratotic scale acts as a barrier to the acaricide, preventing it from reaching the superficial layers of the epidermis where most mites reside (Skerratt, unpublished observations; Barth and Preston, 1988). In addition, topical acaricides that are supposed to be absorbed systemically and then redistributed to other areas of the skin will fail if they are prevented from being absorbed by parakeratotic scale (Barth and Preston, 1988). Injection of acaricides is the preferred method of drug delivery. However, the possibility of drug toxicity in severely debilitated animals must be considered. Neurological signs have been seen in wombats with severe sarcoptic mange that have been treated with ivermectin or moxidectin at normal dose rates for domestic animals (200 µg/kg) (D. Breen, unpublished observations). It is also worth remembering that marsupials have lower resting metabolic rates than eutherian mammals (Dawson et al., 1989) and wombats have a lower metabolic rate than other marsupials of similar size such as macropods (Hume and Barboza, 1998; Johnson, 1998). They are therefore likely to metabolise and eliminate drugs more slowly and maintain higher concentrations of a drug for a longer time than most other mammals given the same dose of drug (Sedgwick, 1993).

Treating with two acaricides, one systemic and the other topical, appears to be very effective in reducing intensity of infection and eliminating infection (Chapter 4, 8). Again, care must be taken to avoid drug toxicity. Mechanical removal of parakeratotic scale and mites by washing the wombat with a keratolytic shampoo is
also effective in reducing the intensity of infection (Chapter 4, 8). This should not be attempted in debilitated free-living animals since they may die from such a procedure (Skerratt, unpublished observations). Debilitated free-living animals that are depressed or unresponsive to external stimulation should not be handled excessively. Even the initial stress of captivity may precipitate death in a debilitated wild animal. Treatment with long-acting antibiotics is also indicated in wombats with moderate to severe sarcoptic mange as they are predisposed to bacterial infections of internal organs (Skerratt et al., 1999, Chapter 3).

At present, it is impractical to attempt to eliminate *S. scabiei* from a free-ranging wombat population. To achieve this, all wombats in the population would need to be treated with an acaricide to eliminate mite infection. Mites would also need to be eliminated from burrows. Although there is no direct evidence, there is anecdotal evidence that mites persist in burrows and that burrow sharing is the method of transmission of mites between wombats. Since treatment of burrows is not practical then it is necessary to prevent wombats from becoming infected with mites until all mites die in burrows. *Sarcoptes scabiei* var. *suis* has been eradicated from swine herds by treating pigs and not treating the environment (Jacobson et al., 1999). *Sarcoptes scabiei* may survive off the host for a limited period only, but relies on the host for sustenance and to be able to reproduce (Arlian et al., 1989). Under favourable conditions in the laboratory, 100 % humidity and 10 °C, *S. scabiei* may survive for three weeks off the host (Arlian et al., 1989). It is not known how long mites, harboured in parakeratotic crusts that have detached from mangy wombats, can survive in wombat burrows but an acaricide would need to be continuously present at therapeutic concentrations in wombats for at least a month to ensure that mites had
died in burrows. The pharmacokinetics of acaricides in wombats are not known but most current formulations of acaricides such as Ivomec (ivermectin, Merck AgVet), Dectomax (doramectin, Pfizer Inc.) and Cydectin (moxidectin, Fort Dodge) at dose rates of 200 to 300 µg/kg have a limited duration of activity in other animals of 9, 18 and 25 days after treatment, respectively (Arends et al., 1999; Papadopoulos et al., 2000). Therefore wombats must be recaptured several times and retreated to ensure that effective acaricide concentrations are maintained. A recently available topical acaricide, selamectin (Revolution, Pfizer, West Ryde, New South Wales) also has a limited duration of efficacy of 2 weeks against ticks at a dose rate of 6 mg/kg in dogs with a single treatment (Jernigan et al., 2000).

Determining whether it is appropriate to treat a wombat population for sarcoptic mange will depend on estimates of the prevalence of the disease as well as recognition of the risk factors mentioned above. These estimates will be biased if they are based on spotlight counts or netting since wombats with mild to moderate sarcoptic mange may travel further and cover a larger area than healthy wombats (Chapter 8). In addition wombats with severe mange are less aware of their surroundings and may be readily approached (Skerratt et al., 1999, Chapter 3). Ideally, spotlight counts should be standardised by determining the prevalence of sarcoptic mange in a proportion of the population by trapping wombats in burrows. This will also provide an estimate of the number of wombats with early stages of the disease whose clinical signs are not visible from afar. Early signs of sarcoptic mange in wombats are erythema followed by parakeratotic scale and then hair loss (Chapter 4).
Future directions in management

Advances in the treatment and management of sarcoptic mange in wombat populations will probably be derived from studies on other species which are also afflicted by this cosmopolitan disease. Treatment advances for parasites including *S. scabiei* are likely to enable the eradication of *S. scabiei* from small isolated wombat populations and to make management of sarcoptic mange in large contiguous populations much easier. Already there is the possibility that a single injection of moxidectin (Proheart, Fort Dodge) used for annual control of nematode infections in dogs could be used as a long acting acaricide treatment in wombats. A long acting ivermectin treatment being developed for nematode control in sheep could also be used as a long acting acaricide treatment in wombats (Barber et al., 2000). This would greatly facilitate control of sarcoptic mange in wombat populations by reducing the number of times that wombats would need to be caught for acaricidal treatment. It is also possible that vaccination with a suitable antigen from *S. scabiei* may provide some protection against infection with *S. scabiei* (Chapter 5). In the interim, devices that automatically deliver topical acaricides to wombats as they enter or leave burrows could be devised.

As is the case with wombats, little work has been done on sarcoptic mange in foxes in Australia (Ratcliffe, 1956; Martin, 1995). Foxes are regarded as a pest to agriculture due to lamb predation and as a major threat to small mammal conservation again due to predation (Coman, 1995; Caughley and Gunn, 1996; Seemark, 1999). Enzootic sarcoptic mange has been reported to control fox abundance in North America and Spain and to have drastically reduced fox numbers after epidemics in Sweden, Norway, Britain and Australia (Ratcliffe, 1956; Tullar and Berchielle, 1981;
Holt and Berg, 1990; Mörner, 1992; Gortázar et al., 1998; Baker et al., 2000). Given that Australia has invested a large amount of money and time into biological control of its pests the lack of research on sarcoptic mange in foxes is surprising (Lugton, 1992; Seamark, 1999). Any future investigation of the effects of sarcoptic mange in foxes in Australia should also consider the degree to which mites are transmitted between foxes and wombats and how this affects the epidemiology of sarcoptic mange in these two host species. Phenotypic and preliminary genetic evidence suggests that the populations of *S. scabiei* on different host species are reproductively isolated from one another (Chapter 7). It is thought that mites adapt to the host species they infect and therefore do not readily survive on another host species, although, this does occasionally occur (Chapters 1, 7). If foxes play a minor role in the transmission of *S. scabiei* var. *wombati* between wombats then it is improbable that *S. scabiei* var. *wombati* will be introduced into the last remaining population of northern hairy-nosed wombats because there are no other wombat populations nearby. The most likely varieties of *S. scabiei* to be introduced into the population, var. *canis* or var. *hominis*, are less likely to survive on their new hosts than var. *wombati*. However, Mörner (1992) suggested that the epizootic of sarcoptic mange in foxes in Scandinavia in the late 1970’s and early 80’s led to the infection of several other species with sarcoptic mange including vulnerable populations of lynx (*Lynx lynx*) and blue (Arctic) fox (*Alopex lagopus*).

Hopefully future studies will test some of the assumptions presented above and provide a greater understanding of this important disease in wombats. Since *S. scabiei* var. *wombati* appears to be a recent parasite of wombats (Skerratt et al., 1998; Chapters 1, 7) the relationship between parasite and host is likely to be evolving as
occurred with myxomatosis and rabbits in Australia and Britain (Ross, 1982).

Procuring funding for studies on sarcoptic mange in wombats is difficult because conservation authorities direct funds towards areas that are currently regarded as more important. Philanthropy is likely to continue as the major source of funding unless conservation authorities consider the potential of sarcoptic mange to act as a key threatening process to wombat populations.
Figure 9.1. Emaciated, adult, male common wombat (*Vombatus ursinus*) from Steels Creek, Victoria, with hair loss and wounds on its rump and back in 1998.


Cooke, B. D. (1998). Did introduced European rabbits *Oryctolagus cuniculus* (L.)
displace common wombats *Vombatus ursinus* (Shaw) from part of their range
in South Australia? In Wombats. R. Wells and P. Pridmore (eds). Surrey
Beatty & Sons, Chipping Norton, New South Wales, 262-270.

R. Strahan (ed.). Australian Museum/Reed Books, Chatswood, New South
Wales, 696.

of the Corvettes Géographe and Naturaliste 1800-1803. Libraries Board of
South Australia, Adelaide, South Australia, 609 pp.

response of a marsupial (*Monodelphis domestica*) to sheep red blood cells.
Developmental and Comparative Immunology 13: 73-78.

Crosbie, P. R., Boyce, W. M. and Rodwell, T. C. (1998). DNA sequence variation in
*Dermacentor hunteri* and estimated phylogenies of *Dermacentor* spp. (Acari:

biochemical and pathomorphological changes of scabies in goats. Indian

and mountain hare during an outbreak of sarcoptic mange. Oecologia 73: 533-
536.


Disease reveals the predator: sarcoptic mange, red fox predation, and prey populations. Ecology 75: 1042-1049.


McCarthy, P. H. (1960b). The transmission of sarcoptic mange from the wild fox
\textit{(Vulpes vulpes)} to man and other species in central Queensland. Australian


\textit{ursinus} (Shaw, 1800). PhD thesis, Australian National University. Canberra,
286 pp.

\textit{ursinus} I. Capture, handling, marking and radio-tracking techniques.

\textit{ursinus} II. Methods for estimating population numbers. Australian Wildlife
Research 4: 223-228.

McIlroy, J. C. (1990). The common wombat strikes back. Wildlife Australia 27: 10-
11.

Australia. R. Strahan (ed.). Australian Museum/Reed Books, Chatswood, New
South Wales, 204-205.

treatment of scabies with ivermectin. The New England Journal of Medicine
333: 26-30.

Mellanby, K. (1944). The development of symptoms, parasitic infection and


APPENDICES

Appendix 2.1. The questions asked in the first phase of the national survey of mange in wombats

Q1. Locality (Please use the 1:100,000 National Topographic Map sheet names) e.g. Moe

Q2. Wombat species in area: (tick box) Common Wombat
    Southern Hairy Nosed Wombat
    Northern Hairy Nosed Wombat

Q3. Sarcoptic mange

a) Have you seen any wombats at this location with hair loss and/or thick crusting of the skin which you believe was mange? YES/NO

b) Has the mite *Sarcoptes scabiei* been identified from any of these animals? YES/NO

c) If present, how many years do you estimate that mange has been in this area?

d) Are there foxes, wild or domestic dogs at this location? YES/NO

e) If you have any other observations on mange which you think may be of relevance to this survey could you please add them here (include attachments if necessary).

Are you interested in participating in the second part of this survey - a three month observation period from 1st November 95 until 31st January 96? (See part 2 attached).
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Appendix 2.2. A copy of the questionnaire used in the second part of the survey

Survey of Prevalence of Sarcoptic Mange in Wombats

(Nov 95 - Jan 96)

Instructions: Please record your observations (allocating one row for each record) of all wombats examined between 1st November 1995 and 31st January 1996

<table>
<thead>
<tr>
<th>DATE</th>
<th>LOCATION (Map name)</th>
<th>MANGE PRESENT (yes or no)</th>
<th>CONFIRMATION OF MANGE (yes or no) (see Note 1)</th>
<th>METHOD OF DATA COLLECTION (e.g. road kill, live animal, day/night?)</th>
<th>SEX</th>
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Notes:

1. Scars and old wounds are sometimes confused with mange. Only answer yes to this question if sarcoptic mange has been confirmed by someone with expertise, preferably a veterinarian.
Appendix 2.3. Localities for which returns were received for common wombats. The number of returns for each locality followed by whether clinical signs of mange were observed and whether *Sarcoptes scabiei* was present are listed respectively after each locality.

**Tasmania and Furneaux Islands:** Flinders Island, 2, yes, yes; Georges Bay, 1, no, ND; Swan Island, 1, yes, yes; Tamar, 2, yes, yes.

**Victoria:** Bacchus Marsh, 1, yes, yes; Buffalo, 1, yes, ND; Euroa, 1, yes, ND; Foster, 4, yes, yes; Healesville, 3, yes, yes; Maffra, 3, yes, ND; Mallacoota, 1, yes, yes; Mansfield, 1, yes, ND; Matlock, 1, yes, yes; Moe, 2, yes, yes; Omeo, 1, yes, yes; Orbost, 2, yes, ND; Ringwood, 3, yes, yes; Stockyard, 1, yes, ND; Warragul, 6, yes, yes; Whitfield, 1, yes, ND; Wilson’s Promontory, 1, yes, ND; Woodend, 2, yes, yes; Wonthaggi, 1, yes, yes; Woolamai, 1, yes, yes; Yea, 1, yes, ND.

**South Australia:** Gambier, 1, yes, yes; Kingston, 1, yes, ND; Millicent, 3, yes, yes; Naracoorte, 1, yes, yes; Penola, 1, no, ND; Robe, 1, yes, yes.

**Australian Capital Territory:** Canberra, 3, yes, yes.

**New South Wales:** Bathurst, 1, yes, yes; Bega, 1, no, ND; Blayney, 1, yes, yes; Bombala, 1, yes, ND; Braidwood, 3, yes, ND; Brindabella, 1, yes, yes; Burragorang, 1, yes, yes; Cessnock, 2, yes, ND; Cobargo, 1, yes, ND; Cooma, 1, yes, ND; Eden, 1, yes, yes; Goulburn, 1, yes, ND; Howes valley, 1, yes, ND; Katoomba, 2, yes, yes; Kosciusko, 2, yes, yes; Merriwa, 1, yes, ND; Michelago, 3, yes, ND; Moss Vale, 1,
yes, ND; Mudgee, 1, yes, ND; Narooma, 1, no, ND; Nundle, 2, yes, yes; Oberon, 2, yes, yes; Tantangara, 1, yes, ND; Taralga, 1, yes, yes; Tumut, 1, yes, yes; Yarrangobilly, 2, yes, ND; Yarrowitch, 1, yes, ND; Yass, 1, yes, ND.

*Locality names are the map sheet names from the 1:100,000 map series.

ND = Not determined.
Appendix 5.1. Chromotrope 2R method for staining eosinophils.

Materials required:

<table>
<thead>
<tr>
<th>Material</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenol crystals</td>
<td>1 g</td>
</tr>
<tr>
<td>Chromotrope (C.I. 16570)</td>
<td>0.5 g</td>
</tr>
<tr>
<td>Soresen’s Phosphate Buffer 5.6 pH</td>
<td>100 ml</td>
</tr>
<tr>
<td>Methylene Blue</td>
<td>0.5% aqueous</td>
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</tbody>
</table>

To prepare Chromotrope solution:

1. Dissolve phenol in 5-10 ml of buffer.
2. Add Chromotrope and dissolve.
3. Add rest of buffer and mix well. (Keeps well and can be used repeatedly).

Method:

1. Bring histological sections to water.
2. Stain in Chromotrope solution for 20-30 min.
3. Rinse clean in water for 2 min.
5. Rinse in clean water.
6. Dehydrate, clear and mount sections.

Results:

Eosinophil granules – bright red
Background – blue

The elements of the Leslie matrix depend on the relationships

\[ e^* = c_{ef} f^*; \quad la^* = c_{le} e^*; \quad n^* = c_{nl} la^*; \quad f^* = c_{fn} n^* \]  

A1

These relationships were found by regression on the measured values of the number of members of the different life-cycle stages.

Given two vectors of measurements on different animals for females and eggs, \( e \) and \( f \), the least square error estimate of the relationship between the eggs and females, which minimises the cost function, \( J_{ef} = \|e - c_{ef} f\| \), given by

\[ c_{ef} = \frac{e' f'}{f' f} \]  

A2

Similarly for the other gain constants.

The correlation constants are, however, not independent as the system is cyclic. They should satisfy the constraint

\[ c_{ef} c_{le} c_{nl} c_{fn} = 1 \]  

A3

The constrained least squares estimate is obtained by minimizing the cost function

\[ J_{con} = \|e - c_{ef} f\| + \|a - c_{le} e\| + \|n - c_{nl} la\| + \|f - c_{fn} n\| + \mu (c_{ef} c_{le} c_{nl} c_{fn} - 1) \]  

A4

where \( \mu \) is the Lagrange multiplier, chosen such that the constraint is satisfied.
The value for \( c_{ef} \) minimizing the above cost function, is given by

\[
c_{ef} = \frac{u_{c_{ef}} + \sqrt{u_{c_{ef}}^2 - 4 \frac{\mu}{ff}}}{2}
\]

where \( u_{c} \) is the unconstrained least squares estimate given by (A2). The values for the other constrained estimates is similarly given. The value for the Lagrange multiplier, \( \mu \), which enables (A3) is easily found by numerical trial and error.
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Author/s: Skerratt, Lee Francis

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