Reviews

Ectoparasites: Direct Impact on Host Fitness

T. Lehmann

Despite being restricted to the host's first line of defence (the integument, away from vital organs), ectoparasite damage has a pronounced impact on host fitness. This generalization can be explained by the reduced dependence of most ectoparasites on their individual host, which minimizes the fitness loss linked to host death. This explanation implies that permanent ectoparasites evolve less 'aggressively' than do either nest or field ectoparasites. This, and other determinants of ectoparasitic virulence are discussed here by Tovi Lehmann.

Weight loss, the reduced production of milk, eggs, meat, hide and wool, fetal abortions and death are extensively documented examples of direct (excluding pathogen transmission) consequences of ectoparasitism in domestic animals[1,2]. The prediction of the 'conventional wisdom' that parasites evolve to be harmless to their hosts is implicit in most explanations of these damages, ie. the confinement of crowded hosts, the provision of shelters for hosts that also protect ectoparasites, the exposure of hosts to unfamiliar ectoparasites, and artificial selection for host traits other than ectoparasite resistance. These explanations imply unusually high ectoparasite loads. Likewise, summaries of ectoparasite–host relationships generalize that ectoparasites, apart from their role in the transmission of pathogens, affect their hosts negligibly in the wild[2,3]. This distinction between direct and indirect impact of ectoparasites has recently become less apparent. This review examines the hypothesis of negligible ectoparasite impact upon components of host fitness (reproduction and survival) in the light of recent studies and analyzes ectoparasitism with respect to parasite pathogenicity.

Ectoparasites primarily share the exploitation of their host's integument. The large diversity among ectoparasites relates to the independent origin of many taxa (eg. ectoparasitism evolved at least seven times in insects[4]), which include protozoa (eg. *Ichtyobodo necator* on octopuses[5]), nematodes (eg. *Noctuidonema guyanense* on moths[6]) and mollusks (eg. *Boonea impressa* on oysters[7]), in addition to the more familiar monogenean helminths, leeches, crustaceans, acari and insects. Ectoparasites are found on a wide range of animal hosts. Because of the diversity of associated organisms and environmental settings, generalizations would be subject to many exceptions.

Even though I will consider ectoparasitism from a broad perspective, my familiarity with mammal ectoparasites biases this review toward insects and acari. These were chosen narrowly (excluding mosquitoes, bot flies, etc.) to comply with all the definitions of ectoparasites.

Tovi Lehmann is at the Department of Entomology, Forbes 410, College of Agriculture, University of Arizona, Tucson, AZ 85721, USA.

Empirical studies

Problems in interpretation of available data. To establish parasite impact on host fitness, it is necessary to manipulate parasite load deliberately (without exceeding too much the maximum natural infestation level) under natural conditions. In parallel, other pathogens that are suspected of being linked with ectoparasite load have to be monitored. Negative correlation of parasite load and a component(s) of host fitness cannot rule out alternative hypotheses that both parasite load and host fitness are affected by another factor. However, negative correlations support the supposition of parasite impact most strongly when a parasite-specific pathology is evident, as with tick paralysis[8], or with epidermal changes in fish leading to the disruption of the osmotic impermeability of the epidermis, resulting in tubular degeneration and kidney necrosis[9]. Laboratory studies should be considered with caution because their findings may not represent the situation in nature.

Results that do not demonstrate parasite impact are meaningful despite the inability to conclude that the parasite has no impact on host fitness. Such results can indicate negligible impact, but they can also reflect insufficient data or even a pathogenic parasite that kills highly infested hosts before they can be sampled, thus only allowing low infestation rates to be sampled. However, this problem can be alleviated to some extent by a frequent sampling design. Several field studies have suggested that interactions between infestation load and factors such as climate, food shortage and predators are important (see below). Thus, laboratory findings showing 'no effect' are ambiguous because laboratory studies do not include such interactions.

Examination of the hypothesis that commensalism is the natural end point of parasitism should not rely on information from recently established relationships (usually related to human activity, eg. species introduction, habitat changes). Thus, references to such systems have been excluded for the most part from this review. Although the time scale associated with the term 'long-established relationship' has not been defined, this category excludes relationships with time measured in hundreds of years.

Ectoparasites frequently harm their host. Recently, enough evidence has accumulated to implicate ectoparasites as being detrimental to their host in many host–ectoparasite associations (Table 1 and other references in the text). Several of these studies have established ectoparasite impact on components of host fitness (by field manipulation, as described above), but the majority did not rule out indirect impact; they only inferred that it is unlikely. However, data from these and other laboratory and field observational studies

strongly support direct ectoparasite impact. The frequencies and intensities of reduction of host survival and reproduction reported in 23 out of 26 ectoparasite–host relationships (Table 1) are higher than would be predicted by the hypothesis of negligible ectoparasite impact (even considering the tendency of workers not to publish 'negative' results).

Host pathology. Young hosts are often more affected by ectoparasites (Table 1), perhaps because they possess a higher ratio of accessible surface to body volume, and because their grooming behavior and other defence capabilities are often inefficient.

Hematophagous ectoparasites are known to induce anemia in vertebrates under natural conditions[10–13]. Hypoalbuminemia, hypophosphatemia, reduced serum calcium and other changes have also been reported[1,14]. Similarly, water mites, a group for which impact on the host has been studied extensively[15], are thought to drain substantial quantities of hemolymph from their host. Mite-infested damselflies may suffer desiccation because of integument injuries, especially in hot weather[16].

Weight loss can also be caused by ectoparasites (Table 1 and Ref. 1). Anorexia was shown in tick-infested cattle[1] and tick toxin was suggested to be involved, but intense irritation alone can result in reduced feeding. Cattle and sheep affected by psoroptic mange constantly rub, scratch, kick and bite themselves; they rub on posts, fences, trees and the ground, leaving the skin bare and bleeding[17].

Damage to feathers leading to poor insulation has been suggested as a cause of mortality in louse infestation of doves[18]. Similarly, tick-infested moose (*Alces alces*) show moderate to severe (>80%) hair loss during winter, a factor that was suggested to affect winter survival[14]. Psoroptic mange associated with hair loss and dermatitis is thought to result in winter mortalities due to heat loss in elk[19] and in bighorn sheep[20].

Tick paralysis is known mostly from unnatural hosts (man, dog and cattle) but paralysis has also been recorded in native hosts such as bandicoots in Australia[21] (primary hosts for *Ixodes holocyclus*) and birds infested with *I. brunneus*, a specific bird tick[8]. Tick paralysis has often led to death.

Tick saliva causes immunosuppression, as suggested by the destruction of inflammatory mediators (ie. histamine, bradykinin and anaphylatoxins), the inhibition of alternate-complement activation, reduced immunoglobulin M production, the repression of T-cell activation, and reduced lymphocyte response to mitogens[22,23]. It is noteworthy that studies showing resistance focused on unnatural hosts, but natural hosts usually do not possess strong immune responses conferring resistance to ticks[23].

Local damage in an infested organ might be very debilitating. Thus, acute inflammation following massive tick infestation of the mouth, nose, hands and feet of Chacma baboon infants was a major factor contributing to neonatal death of at least 43% of all young (nine out of 21) (Ref. 24). That these infants were unable to suckle suggested that inflammation of the infested parts was responsible.

Costs of host defence

Ectoparasites also have 'hidden' impacts on their hosts, that cannot be recognized as pathology, but that probably are expressed in reduced host fitness. These are costs associated with host defence. The most prominent example is that of birds deserting active nests (often with eggs or nestlings) in response to high ectoparasite density. Desertion in response to cliff swallow bug has been demonstrated experimentally in cliff swallows[25], and observationally in several bird–ectoparasite systems (eg. pelicans[26,27], cormorants[27], boobies[27] and martins[28]). This phenomenon is most intensive in late nesting due to the build up of high ectoparasite density[29].

Host grooming and preening have been found to be effective against lice and mites infesting rodents[30], ticks infesting penguins[31], and lice infesting doves[18]. The use of plants containing active insecticides to control nest ectoparasites by birds has been demonstrated for the European starling and suggested as a general strategy in birds[11]. However, these behavioral defenses probably cost considerable time and energy. For example, tick-infested Rockhopper penguins spend about 20% of their time preening[31] (although grooming and preening have other roles beyond ectoparasite control).

Colony site selection is determined by ectoparasite density in cliff swallows[12,25,29]. Swallows re-use colony sites only if ectoparasite load is low, often resulting in annual intermittent occupation[12,29]. Nest selection within a colony is also affected by ectoparasite load in cliff swallows[32] and barn swallows[33]. The defense of a preferred nest (with few ectoparasites) against competitors has been observed in cliff swallows[32]. The presence of ectoparasitic mites in old nests increases the tendency to build new nests[25,33], thereby delaying reproduction. Nest selection and protection or building of new nests involves time and energy that could be invested elsewhere.

Lower survival rates of young birds following early fledging in response to nest ectoparasites has been demonstrated in martins[28], cliff swallows[12] and barn swallows[33]. Cliff swallow departure time from the nesting colony has been synchronized only in cliff colonies with high ectoparasite densities, with nests being deserted even prior to fledging[29]. No synchronization has been observed in colonies (from bridges) that support lower ectoparasite densities and with a one-week longer breeding period. Although other differences between bridge and cliff colonies have not been ruled out, these results suggest that ectoparasites may have been the reason for departure.

Reduced reproductive success has been correlated with male infestation in birds. Mated barn swallow males and males engaging in successful extra-pair copulations have been seen to carry lower loads of mites and lice than do unmated males and males not engaging in successful extra-pair copulations, respectively[34]. The pre-mating period was also longer among highly infested barn swallow males. Laboratory mate choice trials showed that female Rock Doves preferred males uninfested with lice[35].

Profile of a virulent parasite...ectoparasite?

Virulence as used here refers to a significant reduction in a component(s) of host fitness by an infection with a parasite species, and is associated with a cost to the parasite that determines an upper threshold for virulence beyond which the parasite experiences a net reduction in fitness. This cost is tied to the

Table 1. Ectoparasite impact on fitness components of hosts

Ectoparasite[a]	Host[a]	Method	Survival	Reproductive success	Ref.
Tropical fowl mite (*Ornithonyssus bursa*)	Barn swallow (*Hirundo rustica*)	Experimental (field)	No effect[b]	Reduced due to nestling mortality, delayed breeding, smaller second clutch size, reduced frequency of second clutches and lighter offspring	33
Swallow bug (*Oeciacus vicarius*)	Cliff swallow (*Hirundo pyrrhonota*)	Experimental (field)	No effect[b]	Reduced due to nestling mortality up to 50% as a result of reduced body mass and nest desertation	32
Swallow flea (*Ceratophullus celsus*)		Observed[c]	No effect[b]	No consistent effect and even increased nestling survival was observed	32
Swallow bug[d] (*Oeciacus vicarius*); Swallow tick[d] (*Argas cooleyi*); *Ornithodorus concanensis*[d]	Cliff swallow (*Hirundo pyrrhonota*)	Experimental (field)	No effect[b]	Reduced due to nestling mortality up to 65% as a result of anemia, reduced body mass and early fledging	12
Martin mite (*Dermanyssus prognephilus*)	Purple martin (*Progne subis*)	Experimental (field)	No effect[b]	Reduced due to nestling mortality as a result of reduced body mass, nest desertion, prematurely fledging and smaller brood size	28
Synoternus cleopatrae (Siphonaptera)	*Gerbillus andersoni allenbyi* (Cricetidae)	Experimental (field)	Reduced in all ages due to anemia, which also is caused by mesostigmatid mites and *Rhipicephalus sanguineus*	Not measured (but death usually occurs before onset of reproduction)	13
Haemaphysalis humerosa[d]; *Ixodes tasmani*[d]; *I. holocyclus*[d] (all Ixodidae)	Northern brown bandicoot (*Isoodon macrourus*)	Experimental (using enclosures)	Suggested to be reduced in juveniles due to lower growth rate, anemia and leucopenia	Not measured	43
Orntithodorus amblus (Argasidae)	Guanay cormorant (*Phalacrocorax bougainvillii*)	Observed[e]	No effect[b]	Reduced due to massive nest desertions	27
	Peruvian booby (*Sula variegata*)		No effect[b]	Reduced due to massive nest desertions	27
	Peruvian brown pelican (*Pelecanus occidentalis thagus*)		No effect[b]	Reduced due to massive nest desertions	27
Rhipicephalus gertrudae (Ixodidae)	Chacma baboon (*Papio ursinus*)	Observed	No effect	Reduced due to infant mortality up to 43%	24
Hen flea (*Ceratophyllus* sp.)	Great tit (*Parus major*)	Observed	No effect[b]	Reduced due to nestling mortality	44,45
	Blue tit (*P. caeruleus*)	Observed	No effect[b]	Reduced due to nestling mortality	44,45
	Coal tit (*P. ater*)	Observed	No effect[b]	Reduced due to nestling mortality	44,45
Haemaphysalis leporispalustris (Ixodidae)	Snowshoe hare (*Lepus americanus*)	Observed	No effect[b]	Reduced due to lower number of corpora lutea and embryos, increased rate of intra-uterine losses in heavily infested females	46
Ixodes dentatus *Haemaphysalis leposrispalustris*	Cottontail (*Sylvilagus floridanus alacer*)	Observed	Reduced mostly in adults due to anemia and lower degree secondary bacterial infections	No effect	10

...continued

Ornithodoros capensis (Argasidae)	Texas brown pelican (*Pelecanus occidentalis*)	Observed	No effect[b]	Reduced due to nest desertion	26
Protocalliphora spp (Diptera)	Eastern bluebird (*Sialia sialis*)	Observed (in artificial nests)	No effect[b]	Reduced due to nestling mortality up to 24%	47
Protocalliphora hirundo (Diptera)	Barn swallow (*Hirundo rustica*)	Observed	No effect[b]	Reduced due to nestling mortality up to 30% as a result of reduced body mass	48
Protocalliphora sialia (Diptera)	Tree swallow (*Tachycineta bicolor*)	Observed	No effect[b]	No effect as can be measured by nestling body mass and fledgling rate	49
Mite (*Chrysomelobia labidomerae*)	Milkweed leaf beetle (*Labidomerae clivicollis*)	Experimental (laboratory)	Reduced, but not significantly	No effect	50
Water mite (*Tyrrellia circularis*)	*Dasyhelea mutabilis* (Ceratopogonidae)	Experimental (laboratory)	Reduced in adults	Not measured	51
Water mite (*Arrenurus* spp)	Damselfly (*Enallagma cyathigerum*)	Observed	Reduced in adults	Not measured (but substantial mortality occurs before onset of reproduction)	16
Water mite (*Limnochares aquatica*)	Water striders (*Gerris comatus, G. alacris, G. buenoi*)	Experimental (laboratory)	Reduced mostly in L1	Reduced due to prolonged duration of larval instars	15
Gyrodactylus salmonis (Monogenea)	Brook trout (*Salvelinus fontinalis*)	Experimental (laboratory)	Reduced	Not measured	9

[a] The Table summarizes studies that focused on whether ectoparasites affect host fitness (with or without manipulations) rather than commenting on it or providing preliminary data only.
[b] 'No effect' was conservatively adopted as an *ad hoc* conclusion because this study focused on reproductive success and was not intended to measure mortality. Thus only apparently heavy mortality could be detected.
[c] Fleas were not affected by the insecticide that was effective against the bugs.
[d] All ectoparasites (listed in decreasing abundance) were treated simultaneously with acaricides. Thus no specific effects could be attributed.
[e] Also provided circumstantial evidence against involvement of human disturbance, predation, disease (unrelated to the tick), heat and food shortage.

dependence of the parasite on its individual host. It probably acts as a selective force against parasite virulence, unless virulence is associated with a net increase in parasite fitness as a result of a linkage between virulence and transmission (see Refs 36–38 for a comprehensive discussion of this issue).

Most ectoparasites pay a minimal price for killing their host. Ewald[39] suggested that parasites that move between hosts have extremely low costs associated with extensive use of host resources, and therefore should evolve toward extreme virulence. Similarly, many ectoparasites may increase their fitness by faster conversion of host tissue to progeny (ie. increased virulence, when boosted rate of host-tissue consumption affects host fitness), and may also minimize dependence on their individual host via one or more of the following: (1) shortening the duration of host exploitation in relation to their own life span; (2) utilizing a group of hosts; and (3) escaping from a dead host.

Minimized dependence through duration of exploitation. Shortening the time of individual host exploitation decreases the likelihood of host death prior to ectoparasite development or reproduction. This trend is manifested by 'field ectoparasites' that leave their host after each feeding and seek another host for the next meal. Usually, they require only a few large meals taken within a few days (eg. three meals for many hard ticks, one meal for water mites). Nest ectoparasites (dermapterans, fleas, many mites, etc.) spend a substantial part of their (parasitic) life in the host's dwelling rather than on the host body and thus may be protected from the host's defenses and also from being preyed upon while on the host (see below). Presumably, these ectoparasites have not reduced their consumption rate but only reduced the risk associated with remaining on the host.

Minimized dependence through exploitation of a group of hosts. While an endoparasite's longevity usually depends on one host, many ectoparasites (eg. soft ticks, cimicids and fleas) depend upon several hosts (nestlings in a nest, a colony of ground squirrels, etc.) that, as a group, are likely to survive longer than one host. Thus, individual host death may not be as costly to these ectoparasites.

Minimized dependence through escaping from dead hosts. Some hemipterans (eg. Triatomines[40]) and most ectoparasitic flies (79% of Streblidae and 76% of Hippoboscidae[2]) retain their flight capacity, even after host location (unlike Carnidae, which shed their wings when the host is reached[2]), and leave the host even

when gently handled[2]. Likewise, fleas and mites quickly abandon a rodent body when its skin is stretched by the grasp of a predator or captor. Fleas and mites that remain in their dead host's burrow or in the open field are likely to locate a new host[41].

Other determinants of ectoparasite virulence

Nest ectoparasites of hosts that use 'homes' for short periods probably pay a low price (in terms of fitness loss) for their pathogenicity, even when it results in host death, because the host will soon leave the nest in any case. When host availability is short relative to parasite longevity (eg. nestlings 3–4 weeks compared with several months or even years of many of the adult fleas, ticks, or hemipterans), the selection for rapid exploitation of the host is presumably stronger than to 'maintain' the host. This also applies for short-lived solitary hosts (eg. many small mammals live 3–5 months).

Because the duration of host availability may be affected by other parasites (ecto- and endoparasites), the existence of a common virulent parasite species that exerts its impact on the host independently of others may affect the evolution of virulence in the others. Such a process can exacerbate ectoparasitic virulence in vectors that transmit pathogenic endoparasites, due to the frequent co-occurrence of the vector and a disease that shortens host availability. A possible example is tularemia (caused by *Francisiella tularensis*), a bacterial agent transmitted by mites, ticks, fleas, flies and lice, and which is often fatal to various lagomorphs. This hypothesis can be tested when more data on ectoparasite virulence are available, enabling inter- and (particularly) intraspecific comparisons of virulence between ectoparasites that frequently infest sick hosts (in an enzootic area) and the same host–ectoparasite species in an area free of the disease.

Vectorial capacity of ectoparasites is usually linked with a high rate of transfer of an individual ectoparasite among several individual hosts. Taken together with sexual reproduction, which is the rule in ectoparasites[2], they generate high genetic heterogeneity of ectoparasite infrapopulations (on individual hosts). This may intensify intraspecific competition, which should select for virulence, and diminish kin selection, which should select for decreased virulence[37]. Recently, evidence has accumulated to suggest clonal reproduction in many protozoa and other endoparasites[42]. Because the genetic similarity in clonal organisms is much higher, the likelihood of reduced virulence through kin selection could be higher in endoparasites than in ectoparasites.

Virulence may differ between ectoparasite categories. In light of the above discussion, it is not surprising that the ectoparasite list (Table 1) is comprised mostly of field ectoparasites and nest ectoparasites, and only rarely of permanent ectoparasites, such as lice or polyctenids (an hemipteran family of permanent ectoparasites), even though lice infest all these vertebrate hosts (and comprise the largest insect group in terms of species richness and host range). Lice are highly dependent on their individual host, have a lower mobility and lower tolerance to starvation, and cannot escape dead hosts; they are therefore more likely to evolve towards decreased virulence.

Although important, certain difficulties exist in interpreting the comparison of virulence across parasite and host species. However, 'within system' comparison minimizes most of these difficulties. Thus, ectoparasite species that infest the same host population can be compared regarding their impact on the host. For example, out of five taxa infesting *Gerbillus andersoni allenbyi*, anemia was caused by nest ectoparasites in the cases of a flea, *Synosternus cleopatrae*, a hard tick, *Rhipicephalus sanguineus*, and mesostigmatid mites, but not in the case of a louse, *Polyplax gerbilli*, and a permanent flea (rarely found in gerbil burrows), *Stenoponia tripectinata*[13].

Acknowledgements

I am grateful to José Ribeiro, who encouraged me to write this review and who gave valuable suggestions during its writing. Many thanks to Ed Cupp, Judith Bronstein, Greg Simmons and Martin Taylor, and especially to Frank Ramberg, for reading early versions of this manuscript and providing very helpful comments. I also wish to acknowledge the anonymous referees for their thorough and critical comments. During preparation of this work, I was supported by fellowships from the University of Arizona and from the Center of Insect Science.

References

1 Nelson, W.A. *et al.* (1977) *J. Med. Entomol.* 13, 389–428
2 Marshall, A.G. (1981) *The Ecology of Ectoparasitic Insects*, Academic Press
3 Kim, K.C. (1985) in *Coevolution of Parasitic Arthropods and Mammals* (Kim, C.K., ed.), pp 3–82, John Wiley & Sons
4 Waage, J.K. (1979) *Biol. J. Linn. Soc.* 12, 187–224
5 Forsythe, J.W. *et al.* (1991) *J. Fish Dis.* 14, 431–442
6 Marti, O.G. *et al.* (1990) *Ann. Entomol. Soc. Am.* 83, 956–960
7 Ward, J.E. and Langdon, C.J. (1986) *J. Exp. Mar. Biol. Ecol.* 99, 163–180
8 Mullen, G.R. and Hribar, L.J. (1991) *Highlights Agric. Res.* 38, 12
9 Cusack, R. and Cone, D.K. (1986) *J. Wildl. Dis.* 22, 209–213
10 Smith, R.H. and Cheatum, E.L. (1944) *J. Wildl. Manage.* 8, 311–317
11 Clark, L. and Mason, R.J. (1988) *Oecologia* 77, 174–180
12 Chapman, B.R. and George, J.E. (1991) in *Bird–Parasite Interactions* (Loye, J.E. and Zuk, M., eds), pp 69–92, Oxford University Press
13 Lehmann, T. (1992) *Parasitology* 104, 479–488
14 Glines, M.V. and Samuel, W.M. (1989) *Exp. Appl. Acarol.* 6, 197–213
15 Smith, B.P. (1988) *Can. J. Zool.* 67, 2238–2243
16 Arbo, A. (1990) *Odonatologica* 19, 223–233
17 Meleney, W.P. (1985) in *Parasites, Pests and Predators* (Gaafar, S.M., Howard, W.E. and Marsh, R.E., eds), pp 317–346, Elsevier
18 Clayton, D.H. (1991) in *Bird–Parasite Interactions* (Loye, J.E. and Zuk, M., eds), pp 258–289, Oxford University Press
19 Samuel, W.M., Welch, D.A. and Smith, B.L. (1991) *J. Wildl. Dis.* 27, 446–451
20 Muschenheim, A.L. *et al.* (1990) *J. Wildl. Dis.* 26, 554–557
21 Oxer, D.T. and Ricardo, C.L. (1942) *Aust. Vet. J.* 18, 194–199
22 Wikel, S.K. and Whelen, A.C. (1986) *Vet. Parasitol.* 20, 149–174
23 Ribeiro, J.M.C. (1989) *Exp. Appl. Acarol.* 7, 15–20
24 Brain, C. and Bohrmann, R. (1992) *J. Wildl. Dis.* 28, 188–191
25 Emlen, J.T. (1986) *Condor* 88, 110–111
26 King, K.A. (1977) *Wilson Bull.* 89, 157–158
27 Duffy, D.C. (1983) *Ecology* 64, 110–119
28 Moss, W.W. and Camin, J.H. (1970) *Science* 168, 1000–1003
29 Loye, J.E. and Carroll, S.P. (1991) in *Bird–Parasite Interactions* (Loye, J.E. and Zuk, M., eds), pp 222–241, Oxford University Press
30 Murray, M.D. (1990) in *Parasitism and Host Behaviour* (Barnard, C.J. and Behnke, J.M., eds), pp 290–315, Taylor & Francis
31 Brooke, M.L. (1985) *Auk* 102, 893–895
32 Brown, C.R. and Brown, M.B. (1986) *Ecology* 67, 1206–1218
33 Moller, A.P. (1990) *Ecology* 71, 2345–2357
34 Moller, A.P. (1991) in *Bird–Parasite Interactions* (Loye, J.E. and Zuk, M., eds), pp 328-343, Oxford University Press
35 Clayton, D.H. (1990) *Am. Zool.* 30, 251–262
36 May, R.M. and Anderson, R.M. (1983) in *Coevolution* (Futuyma, D.J. and Slatkin, M., eds), pp 186–206, Sinauer
37 Ewald, P.W. (1983) *Annu. Rev. Ecol. Syst.* 14, 465–485
38 Holmes, J.C. (1983) in *Coevolution* (Futuyma, D.J. and Slatkin, M., eds), pp 161–185, Sinauer
39 Ewald, P.W. (1987) *Ann. NY Acad. Sci.* 503, 295–306
40 Lent, H. and Wygodzinsky, P. (1979) *Bull. Am. Mus. Nat. Hist.* 163, 127–520
41 Mead-Briggs, D. (1964) *J. Anim. Ecol.* 33, 13–26

42 Tibayrenc, M. and Ayala, F.J. (1991) *Parasitology Today* 7, 228–232
43 Gemmell, R.T. *et al.* (1991) *J. Wildl. Dis.* 27, 269–275
44 Winkel, W. (1975) *Die Vogelwelt* 96, 41–63
45 Winkel, W. (1975) *Die Vogelwelt* 96, 104–114
46 Keith, L.B. and Cary, J.R. (1990) *J. Wildl. Dis.* 26, 427–434
47 Pinkowski, B.C. (1977) *J. Wildl. Manage.* 41, 272–276
48 Shields, W.M. and Crook, J.R. (1987) *Ecology* 68, 1373–1386
49 Rogers, C.A., Robertson, R.J. and Stutchbury, B.J. (1991) in *Bird–Parasite Interactions* (Loye, J.E. and Zuk, M., eds), pp 123–139, Oxford University Press
50 Eickwort, R.C. and Eickwort, G.C. (1986) *Int. J. Acarol.* 12, 223–227
51 Lanciani, C.A. (1986) *J. Parasitol.* 72, 613–614

Host Resistance in Cattle Tick Control

J.J. de Castro and R.M. Newson

Cattle ticks are an important constraint on the livestock industry, particularly in tropical and subtropical areas, mainly because of the diseases they transmit and the costs of control. Conventional control is by means of acaricides; although there are still serious drawbacks, these can be minimized by a strategic approach. In this review Julio de Castro and Robin Newson look at alternatives. One is to make use of the host's innate abilities. In the simplest situation, where no control measures are applied, the animals develop their own protective immunity against ticks and tick-borne diseases (TBDs). Alternatively, breeds of cattle with naturally high resistance to ticks can be used, enhanced by selection. Methods of immunizing cattle against ticks are also under development. Ideally, ticks should be managed at an economically acceptable level by a combination of techniques, supported where necessary by vaccination against TBDs.

Ticks are the most important ectoparasites of cattle in the tropics and subtropics, where they transmit such fatal diseases as babesiosis (vector *Boophilus* spp), theileriosis (*Hyalomma anatolicum*, *Rhipicephalus* spp and *Amblyomma* spp), anaplasmosis (*Boophilus* spp) and cowdriosis (*Amblyomma* spp). All these ticks take a single blood meal at each stage (larva, nymph and adult female); they are termed one-, two- or three-host species, depending on the number of times in the life cycle that they move on to a fresh host.

World losses caused by ticks have been estimated at US$7 billion annually[1], a figure that urgently needs to be updated. Although tick control is primarily aimed at TBD control, ticks themselves (Fig. 1) can be shown to cause significant reductions in liveweight gain, milk yield and calf production, plus obvious hide and udder damage. Tick bite lesions can also provide entry for myiasis-producing flies (screw-worm attack).

The objective of tick control should be to reduce tick populations below the economic threshold so that control costs are less than the anticipated benefits, not all of which can be priced in subsistence economies. Pastoralists, for instance, may depend heavily on their herds not only for their food supply but also for social standing and ceremonial purposes; peasant farmers may need cattle for ploughing, for milking and as an occasional source of cash income from sales.

Host resistance to ticks is the innate ability of a host, once primed, to mount an immune response (Box 1) to components of the saliva of feeding ticks, killing or debilitating them (Box 2). A rather different result is obtained by vaccination using purified tick-derived antigens.

Julio de Castro is at the GCP/ZAM/044/DEN Strategic Tick Control Project, c/o FAO Office, PO Box 30563, Lusaka, Zambia and Robin Newson is at Barn Cottage, Chapel Lane, Enstone, UK OX7 4NB.

Present tick control measures

The conventional method of control has long been the application of an aqueous suspension of acaricide by dipping or spraying. Recent developments in presentation now include acaricide-impregnated ear tags, slow-release rumen boluses, intramuscular injection and pour-ons. The two latter methods are proving popular and highly effective. Since the use of acaricides is under the control of the cattle owner, they give direct and visible results, but there are also the well-known disadvantages of rapidly increasing development costs, frequent emergence of resistance by ticks to acaricides, and the danger of residual contamination of meat and milk.

Despite these problems, chemical control still remains the method of choice for very intensive dairy and beef production systems in the developing world, particularly if they depend on cattle of pure *Bos taurus* breeds. These operations may be profitable enough to allow large bills for acaricides to be met.

However, the situation more frequently found in developing countries is one in which tick control, if any, is sporadic and ineffective. The livestock are of indigenous *Bos indicus* breeds, and may be infested with ticks of several species. The expected TBDs, though present, may not be obvious because all calves soon acquire mild infections of the prevailing TBDs and generally recover, leaving them with strong protective immunity that may

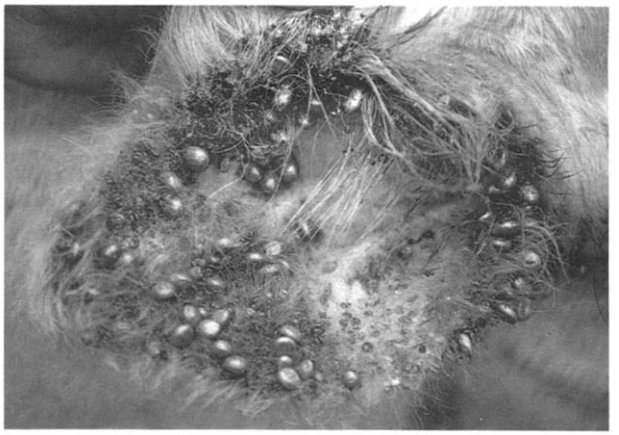

Fig. 1. Heavy infestation of Rhipicephalus appendiculatus *adults in the ear of a cross-bred steer.*