Acarologia is proudly non-profit, with no page charges and free open access

Please help us maintain this system by encouraging your institutes to subscribe to the print version of the journal and by sending us your high quality research on the Acari.

Subscriptions: Year 2020 (Volume 60): 450 €
http://www1.montpellier.inra.fr/CBGP/acarologia/subscribe.php
Previous volumes (2010-2018): 250 € / year (4 issues)
Acarologia, CBGP, CS 30016, 34988 MONTFERRIER-sur-LEZ Cedex, France
ISSN 0044-586X (print), ISSN 2107-7207 (electronic)

The digitalization of Acarologia papers prior to 2000 was supported by Agropolis Fondation under the reference ID 1500-024 through the « Investissements d’avenir » programme (Labex Agro: ANR-10-LABX-0001-01)

Acarologia is under free license and distributed under the terms of the Creative Commons-BY-NC-ND which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original author and source are credited.
THE ORIGIN AND EVOLUTION
OF HOST ASSOCIATIONS OF SARCOPTES SCABIEI
AND THE SUBFAMILY SARCOPTINAE MURRAY

BY J. R. H. ANDREWS *

HOST-PARASITE
CO-EVOLUTION

ABSTRACT : FAIN's hypotheses on the origins and spread of Sarcoptes scabiei
are re-examined in the context of transmission mechanisms, host-parasite co-evo-
lution, and the history of animal domestication. While the broad outlines of the
hypotheses are still found acceptable, changes in detail and alternative hypotheses
have been suggested.

CO-EVOLUTION
DES HÔTES
ET DE LEURS
PARASITES

RÉSUMÉ : L’hypothèses de FAIN sur l’origine et l’expansion de Sarcoptes scabiei
est de nouveau examinée dans le contexte des mécanismes de transmission,
de l’évolution des hôtes et de l’histoire de la domestication des animaux. Pen-
dant que les grandes lignes de ces hypothèses sont toujours encore acceptables,
un changement de détail et des hypothèses alternatives étaient proposés.

Sarcoptes scabiei (L.) (Acariformes : Sarcopti-
dae) is the agent of scabies in humans and sarcoptic mange in other mammals. In a review that
gave special emphasis to this species, FAIN (1968)
concluded that the subfamily Sarcoptinae Murray
contained the highly variable (monotypic) genus Sarcoptes and four other (monotypic) genera,
summarised with their host records in Table 1 (see Table 1, page 91). Contrasting views of Sarco-
ptes, yet to find support, are held by KUTZER
(1970). His separation of the genus into 9 species
has been commented upon by PENCE, CASTO
and SAMUEL (1975).

In spite of a substantial literature on S. scabiei
and its various disease manifestations, and a
curiously wide host range, there has been little
speculation on the origins and evolution of the
mite related to that of its hosts. However FAIN
(1968, 1978) presented the outlines of an attractive
hypothesis on Sarcoptes origins and spread, the
essence of which was :

1. That Sarcoptes had primate host origins,
arising from one of three genera parasitising
monkeys.
2. It spread from humans to domestic animals
and thence to wild carnivores and wild bovids.
3. Mechanisms that aided this spread were :
   Host factors — lowered host immunity through
domestication and susceptibility through changes
in nutrition.
   Mite factors — interbreeding of mites from
distantly related mammals providing new char-
acters and encouraging adaptability. The
variation found in a given Sarcoptes population
provides a reservoir of adaptive characters.

In presenting his hypothesis FAIN did not speci-
ically exclude alternative possibilities or give
detailed consideration to the evolution of the pri-
mate hosts. The purpose of the present paper is
to examine his proposals in detail, consider some

* Zoology Department, Victoria University of Wellington, New-Zealand.

Acarologia, t. XXIV, fasc. 1, 1983.
alternatives, and to incorporate some new information that has appeared since his work was published. The order of the discussion will aim to follow the above summary of the hypothesis, but for greater clarity, the mechanisms of *Sarcoptes* transmission first need explanation.

**TRANSMISSION MECHANISMS**

Transmission is a function of the host's ecology, behaviour and susceptibility as well as the adaptability of the transmitted mites, and these factors will be examined under the following headings:

- **Host contact**:

  Transmission of *Sarcoptes* between hosts is normally achieved by close bodily contact, although an experiment conducted by Gerasimoff, 1953, showed that the mite can be transmitted to uninfected animals occupying the empty dens of mangy hosts, and there is the further possibility that flesh-flies feeding on the carcases of dead animals can act as phoretic (i.e. transport) hosts — Gerasimoff observed larval mites on the ovipositors of flies for about 24 hours after contact between flies and a dead host. From the present author's observations of advanced sarcoptic mange in red foxes it was noted that the mites laid eggs in the fissures and chambers of the crusted hyperkeratotic layers of the skin, rather than in the more conventional burrow seen in many human infestations. Therefore it is possible that eggs may become detached from the host, drop on to some substrate, and hatch when the appropriate chemotactic and thermotactic stimuli of a new host present themselves (and given that environmental conditions did not adversely affect viability e.g. dessication). The limits for viability for such eggs would probably be within those quoted by Heilesen (1946). Gerasimoff (loc. cit.) noted that all mite stages could be infective to the fox hosts — (a finding for which there are contrary views, e.g. Heilesen, 1946) and as mites will often leave a dead host, it is unclear from Gerasimoff's experiment whether it was wandering mites or hatching eggs that were found in his infective dens.

More work is required on *Sarcoptes* transmission, particularly between wild hosts, but the possibility of phoretic transmission clearly has significance for the discussion of the evolution of host relationships of the mite. It would obviate the need for close ecological and other relationships between different host species (excepting the indirect link provided by the fly) prerequisite to the transmission process. The foregoing suggests that we work on the assumption that there are three possible means of *Sarcoptes* transmission — direct contact, phoretic, and indirect (via disseminated stages) transmission.

- **Contact opportunities**:

  These can be divided into ecological, taxonomic, and domestic animal relationships:

  a. Ecological relationships: include predator-prey relationships and the sharing or temporary occupation of another's habitat (particularly a den or burrow). The type of relationship will determine the direction in which the transmission will normally proceed i.e. which is the donor and which is the recipient host. For example predator-prey relationships will result in the mite transmitting in the direction from prey to predator. Habitat transmissions will tend to have transmissions flowing from the dominant user, although there may be two-way exchanges;

  b. Taxonomic relationships: exist where hosts of different species or subspecies (but having, say, generic relationships) from time to time interbreed or associate. In such cases transmission could proceed in either direction. Examples are seen in the family Canidae;

  c. Domestic animals relationships: these involve contacts between humans and an assortment of domestic animals — predominantly ungulates, dogs and cats. There may also be contacts between species of domestic animals — e.g. between working dogs and sheep or cattle. Human-domestic animal contact (e.g. riding, milking, mustering, shearing, the handling of slaughtered animals and of pets) is considerable, often prolonged, and frequently results in scabies transmissions — usually of the self-limiting kind.

- **Host susceptibility — mite adaptability**:

  We also need to establish susceptibility because infestations do not always establish on new host
species and eventually die out. For a successful cross-transmission to take place there must be a predisposing host condition and/or adaptability of the mite.

Kutzler and Onderscheke (1966) and Onderscheke, Kutzler and Richter (1968) established a relationship between host nutrition and sarcoptic mange in chamois and regarded Vitamin A as particularly important for mange prevention. Fain (1968) suggested that nutritional factors may be involved and also that the process of domestication affected mammalian immunity. Also, it seems likely that early human-domestic mammal contacts were with young, sickly or otherwise stressed animals. One or some combination of these factors might have predisposed a host to infestation. There also exists a kind of predisposition that has been well described from humans and very likely has an analogue in populations of wild and domestic animals. Its manifestation is called crusted or Norwegian scabies, and it is typically recorded from the elderly, some of those with genetic diseases, those who are otherwise physically debilitated, as well as from the artificially immunosuppressed. Although this condition is typically regarded as a result of some malfunction of the immune system, the cause is not clear and more than one phenomenon may be involved. For example, in some cases it may involve lowered host resistance rather than some inadequacy of the host immune response, and nutritional factors described above may also be included. Whatever the causes, the results are rather similar: increased susceptibility, vastly greater numbers of mites per host, and a greatly increased capacity for infesting new hosts.

Fulminating cases of sarcoptic mange are known from both domestic and wild animals. Kutzler (1970) described examples from pigs, cattle, sheep, and chamois; there have been numerous reports from red foxes (Vulpes fulva) and the author has been shown severe cases in porcupines. It appears that some groups of wild animals are naturally predisposed towards sarcoptic mange and it is possible that others will become predisposed as resistance is temporarily lowered, or some malfunction of the immune response appears. Naturally occurring epidemics of sarcoptic mange have been recorded from wild animals (Trainer & Hale, 1969) and there are several reports of captive wild animals suffering from severe sarcoptic mange (Fiennes, 1967; Fain, 1978). The generally lowered immunity resulting from domestication (Fain, 1968) has already been mentioned.

It seems likely that the susceptibility required for successful interspecific transmissions appeared on several occasions throughout history and allowed the passage of Sarcoptes from one mammalian order to another, to give the picture seen in current hosts records. Previously lacking has been convincing evidence that such cross-transmissions take place naturally. Sweatman (1971) quotes records of several cross-transmissions (mostly between domestic animals) but the circumstances and outcomes of a number of the cases were far from clear, and experimental transmissions (e.g. Kutzler, 1966; Kutzler and Grünberg, 1967; Stone et al., 1972) do not always meet with success. However, a relatively well documented example of a natural cross-transmission to a susceptible host was presented by Ruiz-Maldonado, Tamayo and Domínguez (1977) in which crusted scabies on a case of Turner's syndrome was believed to have Sarcoptes scabiei var. canis and a dog host as its source. Mites from the case were successfully transmitted back to dogs, but failed to establish on human hosts — the girl's family contacts acquiring a typical self-limiting zoonotic scabies that failed to establish. This case demonstrates the likelihood of a number of similar situations developing over the 15,000 or so years since the domestication of animals began.

The flow of Sarcoptes between species, utilizing the contact opportunities and mechanisms discussed above, is depicted in Fig. 1.

Although host susceptibility may be a prerequisite of cross-transmissions, the morphological variability of Sarcoptes (described by Fain, loc. cit. and Pence, Casto and Samuel, 1975) may present advantages when adaptation to a new host is required — a possibility suggested by Fain as part of his hypothesis. While such variability
may play a role, the capacity for physiological rather than morphological adaptability might more easily secure the mite a place on a new host species. The mammalian skin is a relatively conservative organ given to great variation in individual response to infestation, with resulting morphological changes in the skin itself. Thus the demands for morphological adaptability made on an invading mite may be at least as great within a host species as between host species.

In summary, what is suggested is that instances of susceptibility (arising from a variety of causes) have arisen periodically in certain host groups over the last 20,000 years or so, and that when such events coincided with ecological, domestic, related taxa, or phoretic contacts, successful transmissions took place. This process may have been aided by the variability of the mite, although possibly not to the degree indicated in the original hypothesis.
THE ORIGINS OF Sarcoptes

It may first useful to consider the implications of the concepts of ecological, physiological and phylogenetic specificity as discussed by INGLIS (1971). Ecological specificity is determined by host ecology or behavioural factors, and allows that a parasite can infect physiologically similar but possibly phylogenetically unrelated hosts. Physiological specificity has hosts that are neither phylogenetically related nor have they ecological aspects in common. Phylogenetic specificity has parasite evolution being determined by (not necessarily concurrent) host evolution and implies a long host-parasite association. These considerations suggest, according to INGLIS, that phylogenetic specificity is a consequence of either or both of ecological and physiological specificity. Some of the consequences of host specificity that lead to parallel host/parasite phylogenies are embodied in FAHRENHOLZ’S rule which states that the phylogeny of parasites tends to reflect that of their hosts (for discussion of this and other parasitophyletic rules see INGLIS, 1971; BROOKS, 1979). Parallel phyletic divergences of host and parasite are indicative of long associations so it is now pertinent to examine what we know of the host-parasite relationship of Sarcoptes and attempts to relate the divergences of the sarcop- tines to those of their hosts.

Ecological specificity is of some importance in the Sarcoptes-host relationship as transmission in most (although probably not all) instances relies heavily on host behaviour and associations, particularly in the case of transmission by body contact. This means of transmission may result in lines of mites more highly host specific than those transmitted by other means, e.g. phoretic transfer. It will be evident that transfer to "carrier hosts" such as flies will be highly dependent on the size of the donor mite population (itself dependent on host predisposition and response) and the success of the infestation will depend on the predisposition of the recipient hosts. It seems likely therefore that host response will influence the host specificity of the mite not only by deter-
have been recently acquired by their hosts. It can be argued (as in Inglis, 1971) that host specificity may be a feature of recent host-parasite associations.

The groups in contention as ancestral hosts of Sarcoptes are: primates (because of the presence of other sarcoptines in this group), ungulates and carnivores (both by virtue of their numerous host records of Sarcoptes). Consideration of the host records of other Sarcoptinae and a cladogram of primate phylogenetic relationships, allows the construction of a tentative phylogeny of the sarcoptine mites (Table 1, Figs. 3 and 4). In doing this several assumptions have been made and the qualifications are outlined below. Whatever the true host-sarcoptine species associations are, it is evident that there has been a radiation of sarcoptines among the cercopithecoids monkeys and from this one could conclude (assuming Fahrenholz's Rule) that a Sarcoptes ancestor diverged

![Fig. 3: Sarcoptine records related to Primate phylogeny. 1 = Pithesarcoptes. 2 = Prosarcoptes. 3 = Cosarcoptes. 4 = Sarcoptes. * Recorded from captive host. (Primate phylogeny partly after Goodman & Moore, 1971).](image)

![Fig. 4: A hypothetical phylogeny of Sarcoptines.](image)

and followed the hominoid line at some point following the separation of cercopithecoids and hominoids. From humans it spread to other mammalian orders via domestication, in ways described later. It appears that less weight in the above arguments attaches to the numerous ungulate and carnivore records of Sarcoptes, especially as the spread of the mite (in relatively undifferentiated form) among these hosts can be given other explanations.

Although he did not elaborate on the thinking that led to his conclusions, Fain (1978) suggested Sarcoptes arose from one of the three sarcoptine genera parasitising monkeys, but this seems unlikely if the above view of Sarcoptes origins is accepted. His earlier, and more general conclusion, that the ancestor of Sarcoptes was a parasite of monkeys, is more acceptable (Fain, 1968).
The above conclusions, and presumably Fain’s as well, were based largely on the current understanding of the requirements for Sarcoptes transmission. Chief of the was need for close bodily contact between hosts — which would place a degree of predictability on the pathways (linking different host species) along which transmission developed. However there are studies (see earlier discussion) that point clearly to other modes of transmission, such that quite unanticipated host species could have joined the ranks of those infested with Sarcoptes. If similar circumstances applied to the ancestral host and parasite, then other origins for Sarcoptes could be speculated upon. For example, phoretic transfer of sarcoptine mites from cercopithecoid hosts to a wild mammal group (e.g. artiodactyls) and later transfer to humans and domesticated mammals.

The direction of spread of Sarcoptes among its mammalian hosts is discussed later, but some explanation has to be found for the presence of Trixacarus on rodents. All records for this genus are from Europe on hosts of predominantly northern distributions (and presumably limited exposure to most primates except humans) so that the circumstances under which the progenitors of Trixacarus might have passed from primates to rodents are difficult to envisage. However there seems to be a parallel in the other sarcoptid subfamily, the Notoedrinae, which although largely from bats, has a few rodent and carnivore hosts. Therefore there may be something in the life styles of rodents that results in their acquiring infestations from other mammals. Also modes of transmission other than body contact may have resulted in an early acquisition of a sarcoptine mite by rodents.

There are several unsatisfactory features to the basis of the views expressed above. Many of the records of sarcoptines are from hosts in captivity and it is difficult to separate possible contaminations from natural records. The record of Prosarcoptes pitheci (see Table 1) from a Cebus, a new world monkey, I have regarded as a contamination. The circumstances and location (Austria) of the Cebus record further strengthens this interpretation. The platyrrhines diverged from the catarhrines some 60 million years ago and it is difficult to conceive that P. pitheci would survive undifferentiated on such widely separated host groups.

In a number of cases (e.g. in Fiennes, 1967) it is not easy to be certain of the classification of the mites involved — the term “scabies”, “sarcoptic mange” and “mange” are often broadly used and do not always signify accurate identification of the infesting agent. A number of sarcoptine records from primates are of pathogenic and sometimes fatal infestation — conditions not usually typical of lengthy host-parasite relationships, although the stress of captivity and other factors may have influenced the course of the disease.

### Table 1.

Host records of the sub-family Sarcoptinae (partly after Fain, 1968)

<table>
<thead>
<tr>
<th>Species</th>
<th>Order &amp; Family</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prosarcoptes pitheci</td>
<td>O. Primates Cercopithecidae</td>
</tr>
<tr>
<td>Cercopithecus aethiops sabaeus</td>
<td>Cercopithecidae Cebidae</td>
</tr>
<tr>
<td>Papio papio papio</td>
<td>Cercopithecidae Cebidae</td>
</tr>
<tr>
<td>Cebus capucinus</td>
<td>Cercopithecidae Cebidae</td>
</tr>
<tr>
<td>Conarsacoptes scanloni</td>
<td>O. Primates Cercopithecidae</td>
</tr>
<tr>
<td>Macaca irus</td>
<td>Cercopithecidae Cercopithecidae</td>
</tr>
<tr>
<td>Pithearsacoptes talpoini</td>
<td>Cercopithecus talpoini</td>
</tr>
<tr>
<td>Sarcoptes scabiei</td>
<td>O. Primates Hominidae Pongidae</td>
</tr>
<tr>
<td>1 sp.</td>
<td>O. Carnivora Canidae</td>
</tr>
<tr>
<td>5 spp.</td>
<td>Mustelidae Procyonidae</td>
</tr>
<tr>
<td>1 sp.</td>
<td>Felidae Ursidae</td>
</tr>
<tr>
<td>2 spp.</td>
<td>O. Perissodactyla Equidae Tapiridae</td>
</tr>
<tr>
<td>1 sp.</td>
<td>O. Arniactyla Sulidae Tayassuidae</td>
</tr>
<tr>
<td>1 sp.</td>
<td>Canelidae Cervidae</td>
</tr>
<tr>
<td>2 sp.</td>
<td>Bovidae</td>
</tr>
<tr>
<td>2 spp.</td>
<td>O. Rodentia Muridae</td>
</tr>
<tr>
<td>4 spp.</td>
<td>Cricetidae Echidnidae</td>
</tr>
<tr>
<td>2 spp.</td>
<td>O. Lagomorpha Lagidae</td>
</tr>
<tr>
<td>8 spp.</td>
<td>O. Insectivora Erinaceidae</td>
</tr>
<tr>
<td>1 sp.</td>
<td>O. Diprotodontia Vombatidae Phascolarctidae</td>
</tr>
<tr>
<td>2 sp.</td>
<td>1 sp.</td>
</tr>
<tr>
<td>1 sp.</td>
<td>1 sp.</td>
</tr>
</tbody>
</table>
Catarrhine host records show some curious gaps and inconsistencies. The extent to which *Sarcoptes* occurs naturally on hominoids other than humans is not clear, although it has been recorded from captive hyllobatids and pongids. But why has there been no sarcoptine radiation in the hominoids as there has been in the cercopithecoids? There is an absence of any records (from captives or otherwise) from colobine monkeys. There is nothing readily apparent in the known ecology or social behaviour of catarrhines to explain this host distribution, so it is clear that further and closer examinations of the relevant host groups and their mites are required. Once better host records have been established it may be worthwhile considering those aspects of primate behaviour that might have led to the distribution of sarcoptines within this host group e.g. mixed species flocks and inter-primate predation.

### THE DIRECTION OF INFESTATION BY *Sarcoptes*

Following identification of possible ancestral hosts and the more likely means of transmission of *Sarcoptes* between the assorted host groups, it now remains to establish the order in which they become infested.

The order of infestation suggested by FAIN (1978) (humans-domestic animals-wild carnivores and wild bovids) was one that was (presumably) based on the assumption that bodily contact was required for transmission. The possibility of phoretic transmission, not considered at that time, provides for some exceptions to that order — indeed it permits explanation of the initial infestations of hosts unlikely to have become infested by any other means, e.g. porcupines and hedgehogs. Phoresy may have been relatively uncommon, or at best a contributor to more conventional transmission processes, which are less haphazard and are bound to impose some sort of order on the sequence of hosts infested.

Proceeding on the assumption that humans were ancestral hosts, domestication presents itself as the most likely form of sustained contact necessary to ensure cross-transmission, and there is no dissent with FAIN on this point. In order to con-

The possible order in which various groups and species became infested is presented in Fig. 5. We can assume that the dog was the first domestic animal to acquire scabies from humans as more than 3000 years separate this from most other domestications (although REED, 1974, suggested that incipient reindeer herding began at about the same time). In the tenuous stages of early domestication, there were probably numerous occasions when wild animals were rejoined by partly domesticated relatives. Therefore *Sarcoptes* may have passed from the newly domesticated dog to wild canids well before the hoofed animals became infested. Once among the wild carnivores passage to ecological associates of these animals became possible. The first ungulates to become infested (via domestication) were probably sheep, followed by pigs, goats and cattle. The perissodactyls, including horse, camel and llama followed a little later.

Wild ungulates presumably became infested through contact with escaped domestic relatives, although some infestations e.g. antelopes and some cervids, are more difficult to explain as there were limited attempts at their domestication.
Perhaps phoretic transmission was responsible for these records. The last major group to be infested were a mixed bag of mammals taken captive (for zoos, laboratories, etc.) and subsequently infested by their captors.

Although the order outlined agrees with the general terms of Fain's proposal, there are differences in detail — particularly relating to the infestation of wild carnivores and other wild mammals before the majority of domestic animals.

Although the order outlined agrees with the general terms of Fain's proposal, there are differences in detail — particularly relating to the infestation of wild carnivores and other wild mammals before the majority of domestic animals.

![Diagram](image)

**FIG. 5 :** Hypothetical order and timing of Sarcoptes infestations based on assumption of humans as original source hosts.

(Note : the categories of host have been scaled in the diagram to indicate numbers of host species infected within each category).

### The Separation of Sarcoptes Varieties and Speciation of Sarcoptes

Attempts to divide Sarcoptes into three groups, based on the distribution of dorsal scales and the presence or absence of ventro-lateral scales were not particularly successful (Fain, 1968, 1978). However it was possible to distinguish specimens from carnivores from those of other hosts, particularly humans. This interpretation of Sarcoptes morphology is consistent with some of the proposals made above, as it is possible that Sarcoptes has been on dogs and at least some wild carnivores longer than any other non-human group.

In a situation where bodily contact between live hosts was the predominant (almost sole) mode of transmission, it could be assumed that host contact would have been largely intra-specific and a model of allopatric speciation would have to be applied to the parasite. This may have been the situation that Sarcoptes faced prior to animal domestication, and the potential widening of the host range of this mite. Given domestication, with close contacts between diverse species, and the increased use of other transmission options (phoresy and habitat sharing) there would be change towards a sympatric population model, where barriers may be physiological and ecological rather than geographical. Observations of Sarcoptes host-specificity suggest that within sympatric populations of Sarcoptes there are sufficient barriers to encourage host-specificity and perhaps (eventually?) speciation. However there are occasions (see earlier discussion) when it may be possible for these barriers to be breached.

### Conclusion

In large measure the proposals made by Fain and summarised at the beginning of this account are upheld, at least in their broad outline. However close examination and discussion of his propositions with the aid of little known or previously unavailable literature has allowed changes in some of the details of the hypothesis, summarised as follows:

1. Sarcoptes had primate host origins, possibly evolving as a line separate from other sarcoptines at some time after the cercopithecoid and hominoid hosts diverged.
2. It spread from humans to newly domesticated canines, and then subsequently to various wild...
mammals. Later other domestic animals, particularly ungulates, and more recently, captive mammals, were infested. As new groups were added, the cross-transmission of *Sarcoptes* between host species probably became even more complex.

3. Mechanisms that aided this spread were: periodic susceptibility (for a variety of reasons) of donor and recipient host species, combined with fortuitous transmission and host-contact features. Variability of the mite may have assisted adaptability.

In addition, from a wider discussion of transmission methods, it is possible to extract other, although perhaps less likely, hypotheses of *Sarcoptes* origins and spread.

ACKNOWLEDGEMENTS

I thank Professors J. A. F. GARRICK and Wm. B. NUTTING for reading the manuscript, also colleagues Drs. C. DESCH, W. STONE and A. HEATH for helpful discussion.

REFERENCES


*Paru en février 1983.*