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Review

EPIDEMIOLOGICAL PROBLEMS OF SCABIES

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The information obtained from 86 American and 73 foreign dermatologists and from a review of the world literature is summarized: "Since 1963 and 1964 there has been a progressive increase in scabies in many parts of the world, to epidemic proportions in some areas. The epidemic has not yet involved the United States or Canada except for sporadic outbreaks and small foci (mainly so-called hippies)."[1]

Since 1971, the situation has become worse and several new countries have been faced with the problem of scabies. In the United States, the disease has increased noticeably and an epidemic was reported in Maine in 1971 involving one fifth of a community of 15,000.[2]

At the same time that the number of cases of scabies has increased, the disease has tended to lose its classic aspect and to occur in atypical forms which are sometimes more difficult to recognize. One of the most frequent of these forms is the "scabies of the cultivated," which is observed in clean persons. It is characterized by a minimum of symptoms and usually by the absence of burrows. In spite of its mild aspect, this form of scabies is readily contagious. Nodular scabies is another atypical form in which lesions consisting of reddish-brown pruriginous nodules occur on covered parts of the body. These nodules may persist for several months despite antiscabies therapy. Mites are occasionally found in the nodules.[2]

The resurgence of scabies is difficult to explain. One may ask if the current upheaval of the social and moral aspects of our society has not contributed to this situation, at least in some countries. The mass gatherings of certain groups of young people and the gradual relaxation of cultural deterrents have created a degree of promiscuity unknown heretofore. The increase in scabies runs parallel to the increase in other external arthropod parasites, especially the lice *Pediculus humanus* and *Phthirus pubis*.[3] This indicates that the increase in mange is due less to the parasite than to the relaxation of standards of personal hygiene.

There are still many other causes that may have contributed to the current resurgence of scabies in the world: increased travel, importation of itinerant workers (important in Germany, France and Belgium), adoption of foreign-born children, transmission to man of canine scabies (frequent in United States), and periodic loss of immunity in a population.[1] Parish[4] believes that the removal of hexachlorophene from most popular soaps has probably played a role in the resurgence of scabies in the U. S.

Resistance to modern scabicides has been suggested from the United States,[5] but not substantiated. Should this new condition be confirmed, scabies could become even more widespread.

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Terminology: Skin Conditions Caused by Mites

According to Roberts,[6] the US Department of Agriculture uses the term scabies not only for sarcoptic, but for psoroptic, chorioptic and psorergatic acariases in livestock. All other mite infestations, such as those produced by *Demodex*, "chiggers," and *Raillietia*, are referred to as mange.

Zumpt[7] proposes to restrict the term scabies to the infestation with *Sarcoptes scabiei* in man, and to use the terms sarcoptic mange for these infestations in animals. He also proposes the terms demodicidosis for the syndrome in man connected with *Demodex folliculorum*, whereas the lesions from *Demodex* in animals are termed demodectic mange. All types of dermatitis in man produced by mites other than *Sarcoptes scabiei* or *Demodex* are classified as acarodermatitis.

In this paper I will use the term scabies for the disease produced by *Sarcoptes scabiei* in man and animals, and the term dermatitis for the infestations of the skin of man by mites other than *Sarcoptes scabiei* and *Demodex*.

Scabies and Dermatitis Produced by Mites

It is important not to confuse scabies caused by *Sarcoptes scabiei* with pruriginous dermatitis produced by a variety of other generally nonpathogenic mites. Scabies is a chronic disease of the skin characterized essentially by a pruriginous eruption produced by mites, which live and reproduce in the superficial layers of the epidermis. In man, 2 species of mites, *Sarcoptes scabiei* and *Demodex folliculorum* are capable of colonizing the skin. Both are strictly parasites of the skin and cannot live in other habitats.

The mechanism responsible for the pruritus in scabies is still a controversial subject. Apparently, the mechanical movements of the rostrum and the saliva of the mite do not play an important role. Mellanby[8, 9] has shown that itching begins only 4 weeks after a primary infestation and during this early period symptoms are very mild or absent. Therefore, the pruritus is thought to be due to the development of hypersensitivity. In reinfected persons itching is more intense and appears sooner (24 hours after infestation), and the average number of mites remains much smaller than in primary infection. Mellanby states that scratching, which removes the mite mechanically, and the stronger reaction of the skin limit the size of the mite population. Skin tests with mite extracts give positive reactions in 6 out of 7 patients with scabies.

Heilesen[10] has repeated the experiments of Mellanby. He agrees that itching generally occurs very early in reinfestations but he claims that it is not a constant feature and that it may also appear at an early stage in some primary infestations. He failed to demonstrate significant hypersensitivity by experiments with mites. He could not find any differences in the number of mites between primary and reinfested persons.

Dermatitis is produced by a variety of mites other than *S. scabiei* or *Demodex* and these too are manifested by both a pruritus and an eruption which can develop either acutely or chronically. The mites are accidental and temporary invaders of the human skin, and they are incapable of reproducing either on or in the skin. These species of acarines capable of producing dermatitis are numerous and belong to diverse families.[11] Most of them are free-living and normally infest stored food, but they produce a dermatitis in persons working with these contaminated products. Those most frequently encountered are "copra itch"

produced by *Tyrophagus* spp., "baker's itch" and cheese mite dermatitis produced by *Acarus siro*, and "grocer's itch" by *Glycyphagus domesticus*.[12]

Among the mites producing dermatitis in man, one species deserves a special mention: it is *Cheyletiella yasguri*, a parasite producing mange in dogs. This species was first described from the US and recently has been found on several occasions in cases of contact dermatitis in the US and Europe. We have observed one such case in Holland: it involved 3 members of the same family who had suffered from stubborn cases of dermatitis for a period of about 9 months, but no mite was demonstrable on their skin. By chance, the dog in the household was examined and was found to have lesions which harbored numerous specimens of *Ch. yasguri*. All symptoms disappeared quickly in both the family and the dog after treatment with an acaricide.[13]

The mechanism for the production of dermatitis has not been explained. It is generally agreed that allergy plays the main role, especially in contact dermatitis produced by stored food mites. However, in patients in whom the mites are attached to the skin by means of the rostrum (chiggers), the primary reactions (mechanical penetration by the rostrum and injection of saliva) are probably more important than allergy.

Crusted Scabies

Crusted scabies (Norwegian scabies or *scabies crustosa*)[33] is a rare form of scabies first described by Danielssen and Boeck[14] in lepers in Norway. As of this writing, about 200 reports of crusted scabies have appeared from various parts of the world.[7] This form differs from classic scabies by the hyperkeratotic, scaly and crusted aspect of the lesions. In some places there are deep fissures in the horny crust. The extent of lesions varies but the scaling, infiltration and redness are often widespread. The most characteristic lesions are seen at the extremities, where keratotic excrescences are often produced. The neck, the face and the scalp may also be involved by the scaling, and alopecia may occur. Burrows are generally present, but Wells[15] observed a patient in whom burrows, vesicles and pustules were completely absent. The pruritus is often mild or absent.[16] The disease requires at least a year for development and may last for more than 21 years.[17]

The mites are always extremely abundant in the lesions and there may be upward of 2 million adult mites in the crusts.[18] In a patient from Finland observed by Pirilä et al.,[19] there were about 200 mites per sq cm in and beneath the crusted lesions.

Cases of crusted scabies are highly contagious and they have often been the source of hospital epidemics. Wells[15] reported that 67 cases of scabies occurred among patients and nursing staff infested by a single patient with crusted scabies. In none of these secondary cases did the disease develop into crusted scabies. Intensely itchy papules were present in all the patients. In most of the cases the symptomatology was atypical, in that the burrows were very rare and the lesions were generally not located in the classic sites but appeared in places which had been in contact with the source of infestation.

Crusted scabies is often mistaken for atypical psoriasis, seborrheic dermatitis or erythrodermia, and in many cases the patient has spread the disease before the correct diagnosis has been made.

We have studied the mites collected in 3 cases of crusted scabies. In one of these patients (the patient of Pirilä et al),[19] the mites were identical to those from ordinary scabies. In the 2 others

(patients from England), the female specimens differed from those from ordinary scabies by the larger size of the bare area in the dorsal field of scales.[20] The bare area was particularly large in females of one of these 2 series. These mites had been collected by Wells[15] in a patient who had no burrows. This observation suggests that the development of scales on the dorsum of the females is probably in relation to the presence or the absence of burrows in the host. The scales probably function in stabilizing the mite in the burrow during the process of boring and locomotion. The mites with numerous scales on the dorsum would presumably be better adapted to live in burrows than those with fewer scales.

Crusted scabies is more frequently observed in patients 65 or older than in young people; however, Hubler and Clabaugh[21] reported an epidemic involving 22 patients ranging in age from 12 to 20, all physically and mentally handicapped (mostly Down's syndrome). The reason that the scabies evolves into the crusted type in some persons is not completely understood. As we have already assessed, it is thought that in ordinary scabies the itching results from sensitization of the skin toward the antigen of the mite. This sensitization results in the production of an antibody which is responsible not only for the pruritus but also for the destruction of many of the mites. In persons who have a deficiency in their immunologic reaction, the skin is not able to produce this specific antibody. Pruritus, resulting in scratching and in mechanical removal of mites, is probably an important factor in limiting the mite population. In the absence of scratching, there is an unrestricted multiplication of the mites in the corneous layer of the skin.

Crusted scabies is generally observed in persons with some condition of stress which probably has lowered their immunologic reaction, e.g., malnutrition, avitaminosis, leprosy, severe infections, diabetes, leukemia, mental retardation. That immunologic factors are important in the production of crusted scabies is substantiated by the fact that the disease may appear during immunosuppressive therapy with corticosteroids or azathioprine.[22–23]

Crusted scabies is also observed in some nervous diseases which produce analgesia, such as syringomyelia in which a lowering of immunity is less evident. In this particular case, the absence of pruritus is probably sufficient to produce this syndrome. Prakken and van Vloten[24] were able to demonstrate the presence of circulating antibodies to scabies in a patient with crusted scabies, with a Prausnitz-Küstner passive transfer test. That proves that a diminution of immunity is not always the only factor responsible for the disease.

Crusted scabies is not a condition restricted to man. Severe clinical manifestations resembling those of crusted scabies are also known in domestic or wild animals.[25, 26]

Human Infestations with Animal Strains of Sarcoptes

Human infestations with *Sarcoptes* of animal origin are common. These infestations are generally considered as self-limiting but in some cases the lesions may continue for several weeks and even persist until treatment is begun.[27] The nonhuman strain of *Sarcoptes* which infest man most frequently is that from the dog but strains from the camel, horse, pig, goat, sheep, chamois, ferret, fox and the llama have also been reported on various occasions.

Infestation of man with canine scabies often occurs in small epidemics.[27, 28] The symptoms appear suddenly or after a

very short incubation period. The eruption is generally papular, more rarely vesicular, and the pruritus is intense. When the scabies is contracted from dog pets, the eruption is located on areas which have been in contact with the pet. Burrows are not found and mites are generally very rare; however, in the 2 cases described by Kutzer and Grünberg,[29] mites were found on 1 person for 4 days and on the other for 7 days. Often the scabies affects several or all the members of the family. The severity and the duration of the disease are variable; it may persist for several weeks until treatment is initiated or may be self-limiting.[30]

Human scabies of canine origin should not be confused with dermatitis produced by *Cheyletiella yasguri*, a parasitic mite of dog described from the US.[31] This mite is a producer of dermatitis in man in this country as well as in Europe.[13] Therefore, in humans who have skin lesions with itching and in whom *Sarcoptes* cannot be demonstrated, *Ch. yasguri* should be suspected.

The identification of the *Sarcoptes* strains from the dog is based mainly on the distribution of the cuticular scales on the body of the female mite.[20]

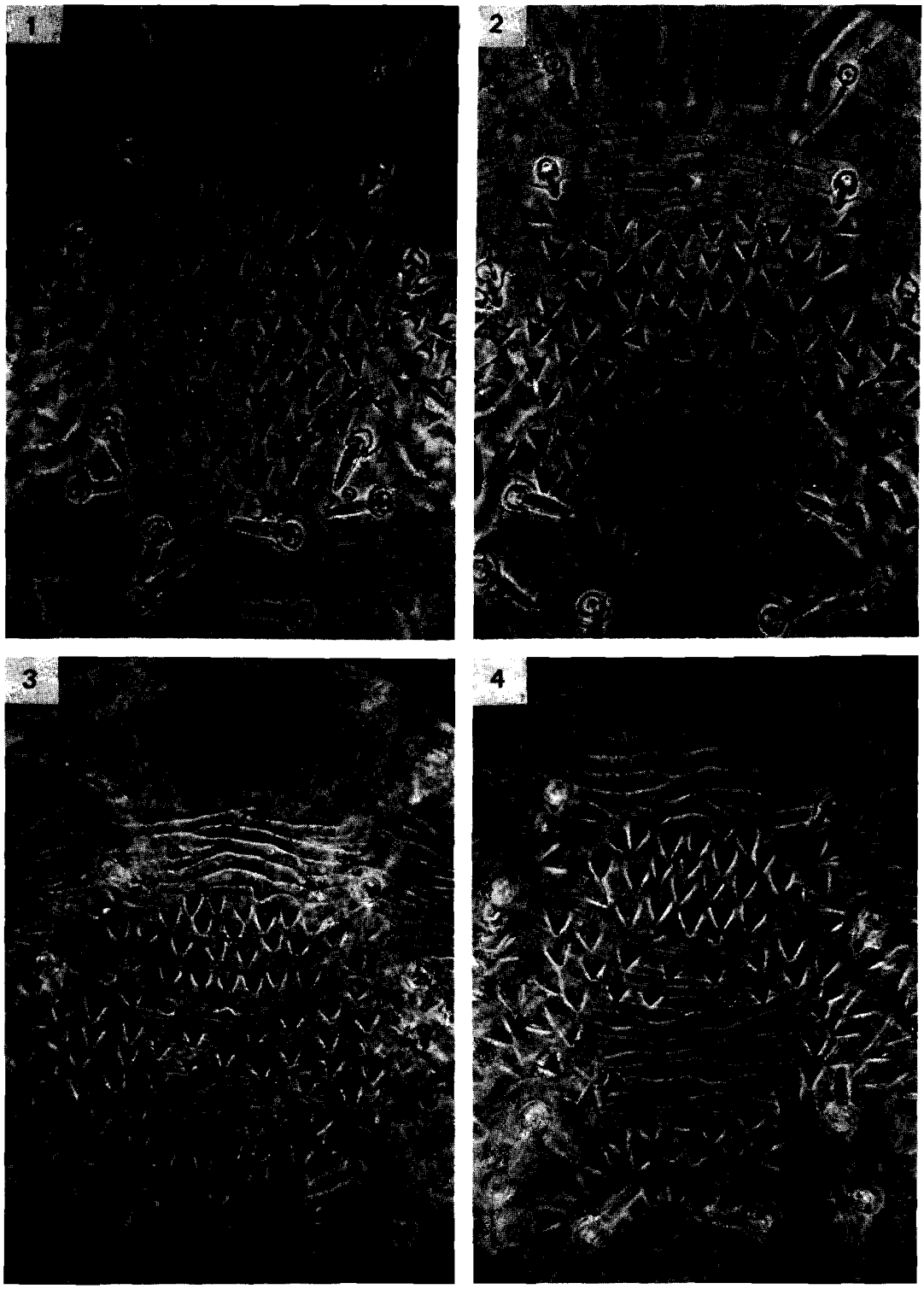
Variability of Sarcoptes Scabiei

Until now, more than 30 species and 15 varieties have been described in the genus *Sarcoptes*. All these "species" are based on variable morphologic characteristics without taxonomic value. From 1962 to 1968, I had the opportunity to examine series of specimens of almost all these described "species." The result is that I am convinced that the genus *Sarcoptes* contains only 1 valid but variable species. Variability becomes evident not only if one compares the strains obtained from 2 different host species (man and dog = host variability) but also within a single strain infesting an individual animal (= individual variability). A third kind of variability is observed occasionally when strains from the same host are compared, but in geographically separated countries (= geographical variability.)[20]

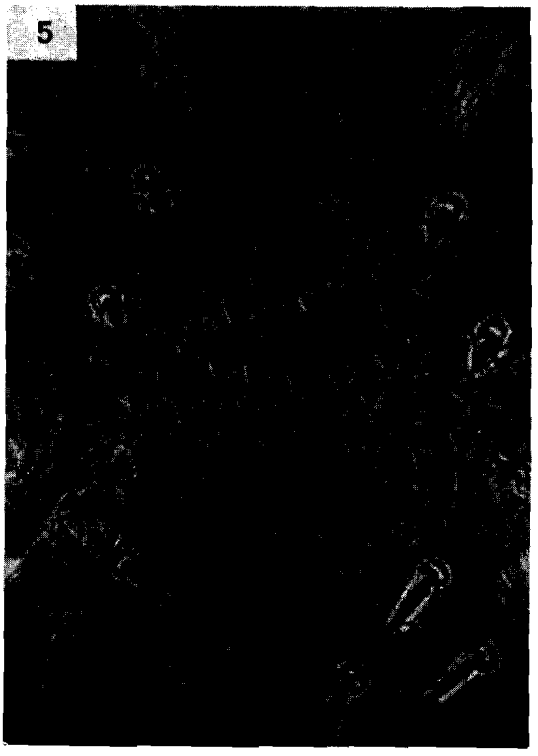
I summarize here the most important features resulting from this study and in relation to the variability of this species. Variability is much more marked in adult females than in adult males or immature mites. The most important characteristics involved with variability in female are as follows: the extent and the size of the cuticular scales on the dorsum and on the lateral regions of the venter, the shape and size of the anterodorsal shield, the size of the body and the length of the dorsal hairs. To obtain significant data in the comparison between different strains, it is necessary to examine a series of 10 to 20 specimens of each strain. The dorsal field of scales and the presence or absence of ventrolateral scales are the most important characteristics in the study of variability and in the identification of strains. The dorsal field of scales may either be complete without any bare area or may have a median bare area devoid of scales. According to the size of this bare area, one may distinguish 4 different types: in type I the bare area corresponds to an absence of 1 to 5 scales, in type II to an absence of 6 to 10 scales, in type III to an absence of 11 to 15 scales and in type IV to an absence of more than 15 scales (Figs. 1-6).

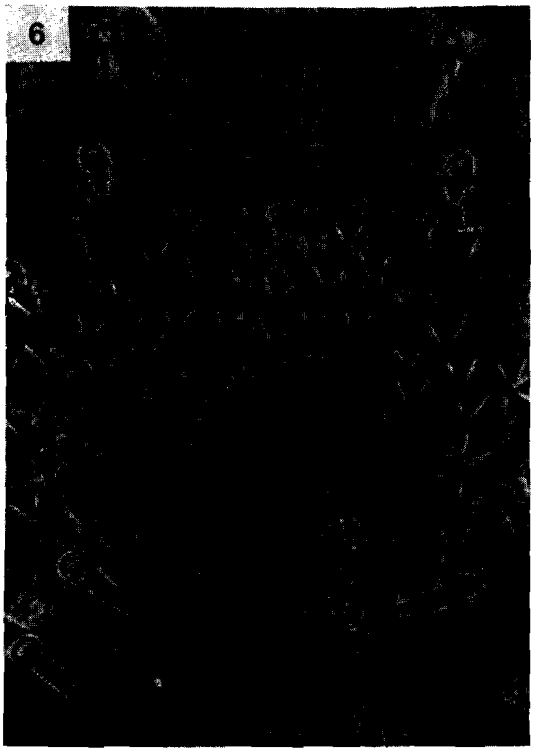
The strains of *S. scabiei* from various host groups such as bovines and carnivores are distinguished from each other by their different proportion of specimens with these variations.

In this respect, one may divide all the known strains of *S. scabiei* into 3 main groups according to the relative develop-



Figs. 1-4. Dorsum of females of *S. scabiei* from human scabies (phase contrast micrographs). 1-2, Case of ordinary scabies (patient from Finland): 1-absence of bare area; 2-bare area of type III. 3-4, Case of Norwegian scabies (patient from England): 3-bare area of type I; 4-bare area of type IV.





Figs. 5-6. Dorsum of females of *S. scabiei* (phase contrast microphotographs). 5, specimen from a ferret (absence of bare area); 6, specimen from crusted scabies (case of Wells) (bare area of type IV).

ment of the bare area in the dorsal field of scales:

1. *Strains with a bare area in most or in all the specimens.* This group contains strains completely devoid of ventrolateral scales (strains from man, camel, dromedary, peccary, gibbon, wild sheep, cabiai) and strains having ventral scales in all specimens (strains from domestic and wild pigs) or in some specimens (strains from a tapir from the Vienna zoo, a chimpanzee *Pan paniscus*, a goat from S. Africa, some African antilopes, horses from the US and S. Africa).
2. *Strains with most of the specimens devoid of a bare area.* This group contains strains completely devoid of ventrolateral scales (strains from cattle in Holland and Belgium) and strains with ventrolateral scales present in all the specimens (strains from dog, ferret, polecat, fox, llama, sheep and goat from Austria, chamois, red deer, mountain dog) or in almost all the specimens (strains from horses from Mayaguez and from Holland, wombat, chimpanzee *Pan troglodytes*).
3. *Intermediate forms.* This group contains strains with intermediate characteristics, for both the bare area and the ventrolateral scales, which prevent us from putting them in either of the 2 preceding groups. They are probably unstable strains still in the process of adaptation to a new host. We recognize here strains from a coatimundi, from a tapir from the Washington zoo and from rabbits in France.

The comparative study of the various strains of *S. scabiei* has lead us to several conclusions:

1. Each strain is a combination of "variants." The term variant is used here for specimens that vary slightly from each other by one or more of the variable characteristics.
2. The strains infecting the various hosts are distinguished from each other not by any qualitative character but only by a different proportion of these variants. For example, in the strains from carnivores the specimens with a bare area are very rare, while in strains from man most or all the specimens have a bare area. In strains from other animals we find an intermediate situation.
3. Each host group possesses a definite combination of these variants, which is morphologically adapted for these hosts.
4. Each strain of *S. scabiei* is probably able to infest any host group simply by modifying the proportion of its variants. Therefore, we may expect that when a strain from dog adapts to man, it will become morphologically similar to the human strain by a process of selection. This adaptation may require several generations of mites before it is completed and it could take weeks or months. Probably in most of the cases the mite is rejected before this adaptation occurs, either through scratching or by antibodies produced in the host. Adaptation might succeed more easily when a predisposing condition is present in the new host, such as decreased immunity by malnutrition or any other organic deficiency.

Now that we have access to powerful immunosuppressive drugs, it should not be difficult to perform such an experiment in animals and to verify if *Sarcoptes scabies* is really, as we believe, only a single variable and adaptable species.

Identification of Human and Canine Strains of S. Scabiei

It is beyond the scope of this paper to give the characteristics of all the strains which can be recognized by studying the variable characteristics, but owing to the frequency with which humans are infested by canine strains, it may be useful to list the most important features which would allow separation of the human from the canine strains.

In all the strains from human scabies, the ventrolateral scales are absent and the bare area in the center of the dorsal field is almost always present but variable in size. Among 3 strains from crusted scabies that we have examined, 2 had a larger bare area than in our 3 strains from ordinary scabies. In 18 females from a patient with ordinary scabies (a man in Finland), there was no bare area in 1 female but all the other specimens had a bare area which was of type I in 2 specimens, of type II in 9 specimens and of type III in 6 specimens (Figs. 1-2). In 9 specimens from a case of crusted scabies (Pirilä),[19] 1 had no bare area, 1 had a bare area of type I, 5 of type II and 2 of type III. Among 35 specimens from a second patient with crusted scabies (a man in England), a bare area was present in all the specimens and classified as follows: type I in 1 specimen, type II in 6 specimens, type III in 16 specimens and type IV in 12 specimens (Figs. 3-4). Among the 9 specimens from the third patient with crusted scabies (Wells),[15] 6 were of type III and 3 of type IV (Fig. 6). I have already mentioned that in the patient of

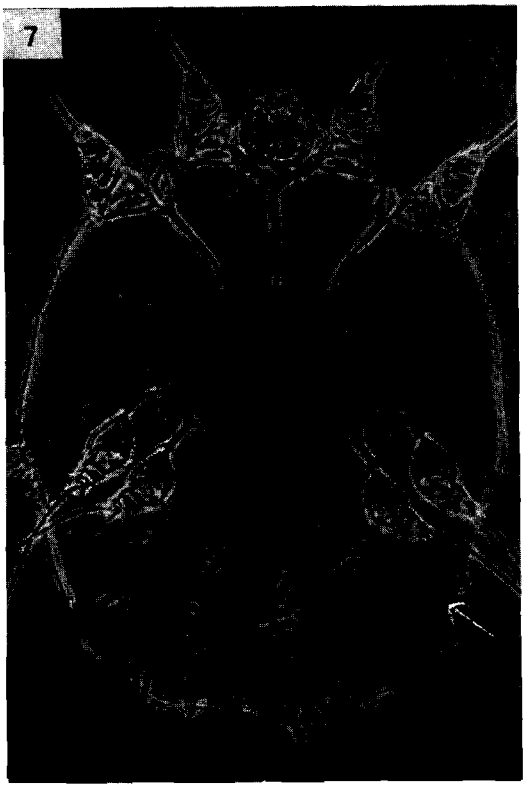


Fig. 7. Venter of a female of *S. scabiei* from a ferret (phase contrast microphotograph). The ventrolateral scales are indicated by an arrow.

Wells burrows were completely absent and I suspect that there is a relationship between the size of the bare area and the presence of absence of burrows.

In strains of *S. scabiei* from carnivores (dog, ferret, fox), the scales are much more developed: the ventrolateral scales are almost always present (from 3 to 5 at each side) (Fig. 7) and the dorsal field of scales is generally complete, without bare area (Fig. 5). Rarely, some specimens may have a very small bare area which never exceeds the type I. In addition to the characteristic of the scales, the size of the body can be useful. The female specimens from human scabies are larger (length 392 to 500 μ, width 295 to 420 μ) than those from dogs (320 to 390 $\mu \times$ 250 to 300μ).

Scabies in Wild Animals

Scabies is very rare in wild animals living in their natural habitat, except in carnivores and bovines in which it is relatively frequent, especially in Africa south of Sahara.[26] The cases of scabies reported from other animals (wombat, cabiai, coatimundi, chimpanzees, gibbon) were probably all secondary infestations acquired in zoological gardens from other infested animals or from man. The captivity had probably reduced the immunologic reaction of these animals.

All these strains from wild animals show the same variability as those from man or from domestic animals.

Obtaining Antigens of Sarcoptes

So far, successful attempts to culture *Sarcoptes scabiei* in vitro have not been reported. The antigenic material necessary for immunologic investigations or diagnosis must therefore be obtained from lesions of infested animals. A simple method for isolating large numbers of *S. scabiei* from lesions in the ears of pigs has been described by Sheahan.[32]

We think that rabbits could probably be utilized as a source of antigenic material. These animals have been experimentally infested in France and it should not be difficult to infest them with human or animal strains. An important requirement for success is to preadapt the rabbits by maintaining them for a certain period before the infestation and during the experiment under conditions which would reduce their natural immunity, such as deficiency in proteins and vitamins (especially A) and in minerals, or by submitting them to immuno-

suppressive treatment (corticosteroids combined with azathioprine).

Another source of antigen which should be investigated for use in the diagnosis of scabies are the house dust mites (*Dermatophagoides pteronyssinus* and *D. farinae*), the common producers of allergic asthma in humans. These mites are free living yet belong to the same order as *Sarcoptes*. They feed on human corneous material as does *Sarcoptes*. Therefore, we may expect that they have some antigens in common. As these mites are easily cultured, they could be an abundant source of antigenic material.

Origin of Sarcoptes Scabiei

As we have pointed out, *Sarcoptes scabiei* possesses a mixture of both stable and variable morphologic characters. The great variability of some of these characteristics suggests that this species is not yet completely adapted to any of the present hosts but remains in a continuous adapting process in all of them. The causes of such instability are unknown but we may surmise that it relates to the great multiplicity of hosts that the species is capable of parasitizing. No other permanent parasitic mite has such a large variety of hosts as does *S. scabiei*. This mite produces scabies in 40 different hosts belonging to 17 families and 7 orders of mammals.

I think that this exceptional situation might be explained by the phylogeny of the mite. The family Sarcoptidae is at present composed of 4 subfamilies. The genus *Sarcoptes* belongs to the subfamily Sarcoptinae, which contains the four most evolved genera of the family.

I have suggested the hypothesis[20] that the genus *Sarcoptes* has derived from 1 of the 3 genera of Sarcoptinae parasitising monkeys (*Prosarcoptes, Cosarcoptes* and *Pithesarcoptes*). It seems that man is the original or primary host for *S. scabiei*, and that domestic animals acquired the mites from him. Later the mites infested wild carnivores and wild Bovidae. To adapt to these new hosts, the mite developed slight differences (morphologic and others) which have lessened its ability to reinfest man efficiently. However, these differences have never become sufficient to prevent occasional interbreeding with strains from man or other animals. One may surmise that the frequent interbreeding of the mite in zoologically remote mammals has on the one hand prevented speciation and on the other hand provided new genetic characters which have enhanced the adaptability of the mite to infest other hosts.

The variability of *S. scabiei* might therefore be considered to be the result of the continuous interbreeding in the strains infesting man and domestic animals.

Drug Name

azathioprine: Imuran

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