

Cross-Antigenicity Between *Sarcoptes scabiei* and the House Dust Mite, *Dermatophagoides farinae* (Acari: Sarcoptidae and Pyroglyphidae)

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ABSTRACT *Sarcoptes scabiei* var. *canis* (L.) (SS) was shown to share a high degree of cross-reactivity with the house dust mite, *Dermatophagoides farinae* Hughes (DF). Sera from scabies-infested rabbits contained antibodies that recognized at least six antigens in extracts produced from DF body material and three from DF fecal material. Likewise, rabbit antiserum to DF body and DF fecal extracts contained antibodies that recognized SS antigens. Sera from control rabbits reacted with DF body and SS extracts did not show antibodies to antigens in either extract. Six of nine SS antigens were cross-reactive with DF antigens. Sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) fraction profiles of SS exhibited at least 30 protein and peptide bands. Fifteen SS bands bound DF-specific immunoglobulin E (IgE) following incubation in sera from nine DF skin-test-positive patients with no history of scabies. The IgE binding varied within and among these patients. The sera of these patients also exhibited significant IgE levels to multiple DF antigens, as demonstrated by crossed radioimmunoelectrophoresis (CRIE) and immunoblotting. Control sera from nonallergic control patients who were skin test negative to DF and without scabies exhibited no IgE binding to any SS band.

KEY WORDS Arachnida, scabies, antigenicity, immunity

IT IS CLEAR that *Sarcoptes scabiei* (L.) stimulates a cellular and humoral immune response in the host (Hancock et al. 1974; Van Neste & Salmon 1978, 1980; Falk 1980; Falk & Bolle 1980; Van Neste 1981; Van Neste & Lachapelle 1981; Falk & Matre 1982; Dahl 1983; Arlian et al. 1985; Arlian in press). However, neither the source nor the characteristics of the mite antigens that trigger the host immune reactions are clear, and our understanding of the immune response is incomplete. The lack of adequate quantities of pure *S. scabiei* antigenic material and high-titer mite-specific antisera had hindered investigations designed to isolate and characterize the mite antigens, to characterize the hosts' humoral immune responses, and to determine their relationship to the disease. Such immunological studies are now possible with the discovery that *S. scabiei* var. *canis* from dogs can be transferred to rabbits, and chronically infested rabbits are a good source of large quantities of mites and mite-specific antiserum (Arlian et al. 1984c, 1985).

Arlian et al. (1985) used mites and antisera from parasitized rabbits to show that extracts of *S. scabiei* var. *canis* are the source of at least nine antigens. However, the character of antigens of other scabies strains and the relationship between the antigens of var. *canis* and the antigens of scabies strains from other hosts, such as var. *hominis*, are un-

known. Likewise, the relationship between scabies antigens and the antigens of other medically important mites, such as *Dermatophagoides farinae* Hughes (the common house dust mite), has not been investigated directly. Indirect evidence suggests that there may be a cross-immunological relationship between scabies and allergy to house dust mites and that the relationship may be important to understanding human susceptibility to scabies and the clinical manifestations of the disease (Falk & Bolle 1980, Falk 1981, Falk et al. 1981, Arlian et al. 1985). This interrelationship is particularly important because house dust mites are cosmopolitan inhabitants of homes throughout the world, and scabies infestations and allergies to house dust mites are common (Van Bronswijk & Sinha 1971; Wharton 1976; Mellanby 1977; Van Bronswijk 1978; Orkin & Maibach 1978; Arlian et al. 1982, 1983; Orkin & Maibach 1985). There is little direct evidence to verify the cross-reactivity of scabies mites and house dust mites. Because of the importance of cross-reactivity to the epidemiology of scabies, the purpose of this study was to determine directly the cross-antigenicity between scabies mites and the house dust mite.

Materials and Methods

Mite Source and Extract Preparation. Heavily infested crusts were removed from New Zealand white rabbits that were chronically infested with *S. scabiei* var. *canis* (SS). Fresh crusts were placed

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around a beam of light (900 ft candle intensity, 1 by 3 cm area) directed through a Petri dish. All life stages of mites were attracted to the light beam; they crawled from the crusts and gathered en masse in the light beam. Every 1–3 h for approximately 6–8 h, the mites that gathered in the beam were collected by aspiration, killed by freezing, and then dried over Drierite (W. A. Hammond Drierite, Xenia, Ohio) under vacuum.

Dermatophagoides farinae (DF) cultures were maintained on a 1:1 mixture of Purina Laboratory Show (Ralston Purina, St. Louis) and Fleischmann's granulated dry yeast (Standard Brands, New York) at room temperature and 75% relative humidity. Culture-medium-free live DF of all life stages were aspirated from culture container rims as they migrated from the medium; they were frozen, desiccated, and stored over Drierite. DF feces material (96% fecal pellets) was collected by sieving well-established DF cultures in which the food supply had been exhausted. Extract preparations were identical to those described previously (Arlian et al. 1984a,b; Arlian et al. 1987a,b,c).

Dried SS material was ground to a fine powder, defatted in anhydrous ethyl ether, and then extracted for 96 h at 4°C in the appropriate volume of distilled water to provide a 1:20 wt/vol concentration. Extracts were centrifuged and the supernatant was drawn off for use in immunoassays.

Mite Antisera. Thirteen New Zealand white rabbits infested with SS were used as the sources of SS antisera. Antisera to DF body and DF feces extracts were raised by subcutaneous immunization of New Zealand white rabbits. The immunization procedure and evaluation of antisera were performed as described previously (Arlian et al. 1984a,b; Arlian et al. 1987a,b,c).

CIE and CRIE. The cross-reactivity of SS and DF (body and feces) extracts was examined by heterologous two-dimensional crossed immunoelectrophoresis (CIE). We used procedures and reagents identical to those reported in earlier studies (Arlian et al. 1984a,b; Arlian et al. 1987a,b,c). First-dimensional electrophoresis was for 35 and 20 min for SS and DF body extracts, respectively. SS extract was reacted with the various DF antisera, and the selected DF extracts were reacted with SS antisera. Fifteen microliters of extract (1:20 wt/vol SS; 1:10 wt/vol DF) was separated in the first-dimension gel and then reacted with specific quantities of the test antisera in the second dimension. Second-dimension anodic and cathodic gels contained 500 μ l and 100 μ l of SS antisera or 100 μ l (normal antisera concentrated 5 $\times$) and 15 μ l (concentrated 5 $\times$) of DF antisera for the top and bottom gels, respectively. As a negative control, all test extracts (SS, DF body, and DF feces) were reacted with normal rabbit serum. Precipitin lines on individual gels resulting from the various reactions were made visible by Coomassie Brilliant Blue R-250 stain (Sigma Chemical, St. Louis). For crossed radioimmunoelectrophoresis (CRIE), unstained CIE re-

actions of the DF extracts and their corresponding antisera were incubated overnight with gentle shaking (on a Junior Orbit Shaker [Lab-line Instruments, Melrose Park, Ill.]) in sera (700 μ l) from the DF-sensitive patients and RAST A buffer (19.2 ml). They then were washed three times with RAST B buffer (Arlian et al. 1984a). The gels were incubated overnight with ^{125} I-labeled (1,000,000 counts/min/gel) antihuman IgE and RAST C buffer (20 ml), washed, pressed, and air dried. Autoradiography was then performed for 1–5 d. RAST A, B, and C buffer were compositions prepared fresh in the lab prior to each use and were as described previously by Arlian et al. (1984b).

SDS-PAGE (Polyacrylamide Gel Electrophoresis). Extract proteins of SS and DF were separated by standard sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) using a discontinuous buffer system; 12% polyacrylamide resolving gel containing 0.1% SDS in 0.375 M Tris-HCl, pH 8.8; and a stacking gel containing 5% acrylamide and 0.1% SDS in 0.125 M Tris-HCl, pH 6.8. The running buffer contained 0.025 M Tris, 0.19 M glycine, and 0.1% SDS, pH 8.3. Twenty-five μ l of mite extract in an equal volume of sample buffer (0.125 M Tris, 4% SDS, 20% glycerol, and 0.002% bromophenol blue, adjusted to pH 6.8 with HCl) were electrophoresed vertically in a 140-by 120-by 3-mm gel at 140 V (volts, constant voltage) until the bromophenol marker reached the end of the gel. A solution of low-molecular-weight protein standards (BSA [bovine serum albumin], 66,000; ovalbumin, 45,000; trypsinogen A, 24,000; lysozyme, 14,300) was also electrophoresed to provide molecular weight markers for use in assessing the approximate sizes of SS proteins and peptides. Reference gels were stained with silver nitrate to show the protein-peptide bands.

Blot Transfer and Autoradiography. Proteins were transferred from the separating gel to a nitrocellulose membrane using a Bio-Rad Trans-Blot cell system (Bio-Rad Laboratories, Rockville Centre, N.Y.). A strip of the SDS-PAGE gel was removed and stained to serve as a reference. The remaining unstained gel was placed in direct contact with a sheet of wetted nitrocellulose membrane and then sandwiched between two pieces of blotting filter paper (Bio-Rad). These were covered by two fiber pads and finally secured between two rigid perforated hinged plates. The membrane was positioned anodically in the Trans-Blot cell and the proteins were transferred at 280 mA (milliampere) constant current for 2 h. The transfer buffer contained 50 mM Tris, 192 mM glycine, 0.1% SDS buffer with 20% methanol, pH 8.2. After transfer, the SDS-PAGE gel was silver stained to verify the transfer. The membrane was soaked overnight in an 8 M urea solution containing 0.1 M 2-mercaptoethanol and 5 mg/ml BSA to block binding sites, then washed in a 50 mM Tris-HCl-saline solution. The membrane was then cut into strips to separate each lane. Each strip was treated identically to the

Table 1. Antibody response of sera from SS-infested rabbits to DF body or DF feces antigen

Scabietic rabbit serum	Cross-reacting antigen								
	DF body antigen no.						DF feces antigen no.		
	1	2	3	4	5	6	1	2	3
A	X ^a	X	X	X	X	X	X	X	X
B		X		X	X	X	X	X	X
C	X	X	X	X		X	X	X	X
D		X		X		X	X	X	X
E	X	X		X		X	X	X	X
F		X				X	X	X	X
G	X	X		X		X	X	X	X
H		X	X					X	
I		X		X		X	X		X
J				X		X	X	X	
K		X		X	X	X	X		X
L		X		X	X	X	X	X	X
M	X	X	X	X	X	X	X	X	X

^a X denotes an antigen-antibody precipitate formed when the scabietic rabbit serum is reacted with DF body or DF feces extract by CIE.

CRIE gels, except that each was washed three more times with Tris-saline solution following the last RAST B buffer wash (after incubation in RAST C buffer). Each was incubated briefly in patient sera and ¹²⁵I-labeled antihuman IgE, and autoradiography was performed.

Human Sera. Sera from nine DF-sensitive individuals with positive prick (≥ 3 mm wheal and flare at 10 mg/ml wt/vol) and no known history of SS infestation were used to perform autoradiography for detection of cross-reacting antigens and allergens. Three sera from patients who were skin test negative to DF and had no history of SS served as negative controls.

Results

Cross-Reactivity of DF and SS Antigens. Heterologous CIE gels of DF body extract reacted with individual sera from 13 SS-infested rabbits exhibited up to six antigen-antibody precipitin lines, all on the anodal side of the gel (Table 1, Fig. 1). The rabbits exhibited circulating antibodies that recognized at least six DF body antigens. The number of cross-reacting antigens recognized by an individual serum varied from two to six (Table 1). Sixty-two percent of the rabbits exhibited circulating antibodies to at least four DF body antigens. Fifteen percent and 23% had antibodies directed at three and two DF body antigens, respectively. Seventy-seven percent of the rabbits had antibodies directed at cross-reacting antigen nos. 2, 4, and 6. The reciprocal heterologous CIE cross of SS extract reacted with antiserum built to DF body extract demonstrated four antigen-antibody precipitin lines. Sera from control rabbits reacted with DF body and SS extracts demonstrated no antibodies to antigens in either extract.

Crossed immunoelectrophoresis reactions of DF feces extract with individual sera from the 13 SS-

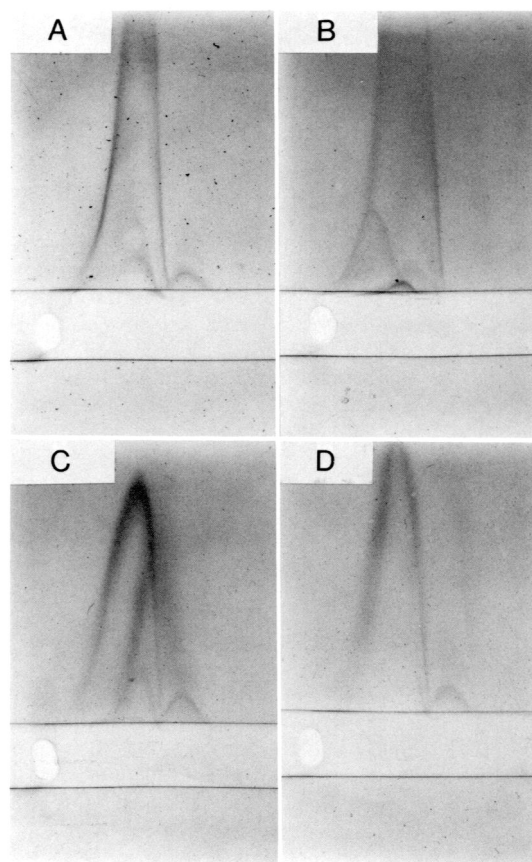


Fig. 1. Representative CIE patterns of DF body extract reacted with sera from SS-infested rabbits.

infested rabbits demonstrated from one to three antigen-antibody precipitin lines (Table 1, Fig. 2). Sixty-nine percent of the 13 rabbits had circulating antibodies directed against three DF feces antigens, whereas 23 and 8% had circulating antibodies that recognized two and one DF feces antigens, respectively.

IgE Antibody Binding to SS and DF Antigens Resolved by SDS-PAGE. Fractionation of SS extract by SDS-PAGE resulted in the visualization of more than 30 protein and peptide bands by silver staining (Fig. 3). At least 20 bands were less than 66,000 daltons, and 8 of these gave dense silver staining. Likewise, SDS-PAGE fractionation of DF body and DF feces extracts demonstrated 25 and 18 silver-stained bands, respectively (Fig. 3).

After blot transfer of the protein tracks onto nitrocellulose membranes and incubation of individual tracks of SS in sera of nine DF-sensitive patients, and then ¹²⁵I-labeled antihuman IgE, autoradiograms revealed multiple SS bands bound IgE in the sera from the DF-sensitive patients (Table 2, Fig. 4). A total of 15 SS bands bound DF-specific IgE. Eight of nine DF-sensitive patients exhibited IgE binding to at least one SS antigenic band, and the number of bands that bound IgE

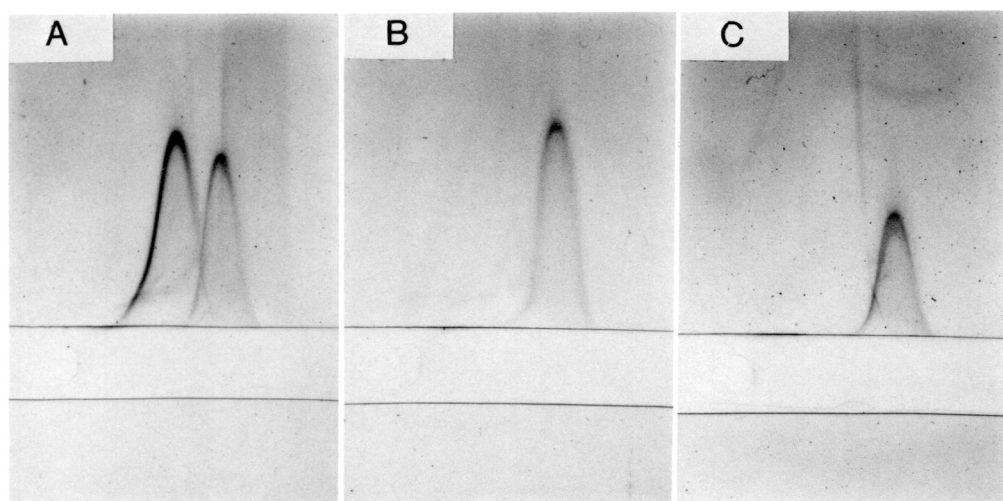


Fig. 2. Representative CIE of DF feces extract reacted with sera from SS-intested rabbits.

varied between patient sera (Table 2). Likewise, quantitative IgE binding to specific bands as measured by the intensity of radiostaining varied within and among sera. The diverse IgE-binding patterns and differing intensities indicated variation in sera-specific IgE antibody levels and/or binding affinities to the various cross-reacting antigens or antigenic determinants. Four protein bands gave

strong IgE binding (intense radiostaining), six exhibited moderate IgE binding, and the remainder exhibited weak binding among the nine sera from the DF-sensitive patients. Control sera from two nonallergic control patients, who were skin test negative to DF and free of scabies, exhibited no IgE binding to any SS band.

Following the blot transfer of SDS-PAGE-separated proteins and peptides and autoradiography, the sera from the same DF-sensitive patients also exhibited IgE binding to DF body and DF feces protein bands (Table 2, Fig. 5). Likewise, IgE binding to the different protein bands varied within and among patients, with each serum exhibiting circulating IgE that recognized from two to seven bands as allergens. In all but one serum, IgE was directed to a greater number of *D. farinae* antigens than *S. scabiei* antigens.

IgE Binding of Patient Sera to DF Body and DF Feces Antigens by CRIE. Homologous CIE of

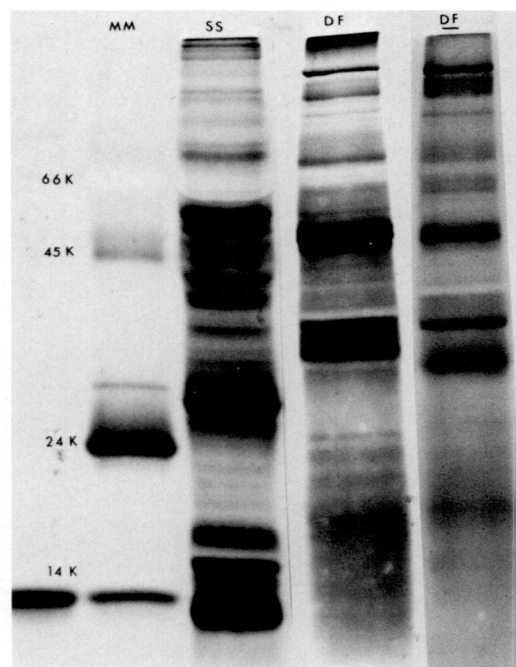


Fig. 3. Fractionation profile of *S. scabiei* (SS), *D. farinae* (DF) bodies, and *D. farinae* (DF) feces after SDS-PAGE. Molecular-weight markers (MM) were bovine serum albumin (66,000), ovalbumin (45,000), trypsinogen A (24,000), and lysozyme (14,300). Gels were stained with silver nitrate.

Table 2. Number of SDS-PAGE-resolved protein-peptide bands that bound IgE after incubation in individual sera from patients with positive skin test to *D. farinae*

Patient serum	No. protein bands exhibiting IgE binding ^a								
	SS			DF body			DF feces		
	S	M	W	S	M	W	S	M	W
1	0	0	1	0	0	4	1	0	1
2	2	3	0	0	1	4	1	0	1
3	2	3	0	3	1	3	1	1	3
4	0	1	2	0	1	4	1	0	2
5	0	0	3	3	0	2	1	1	2
6	1	3	0	5	1	0	3	0	2
7	1	0	2	3	2	1	0	2	3
8	0	0	0	2	0	0	0	1	2
9	1	0	4	5	0	2	2	0	2
Control ^b	0	0	0	0	1	0	0	1	0
Control ^b	0	0	0	0	0	0	0	0	1

^a S, strong; M, moderate; W, weak IgE binding.

^b Control, skin test negative to house dust mites.

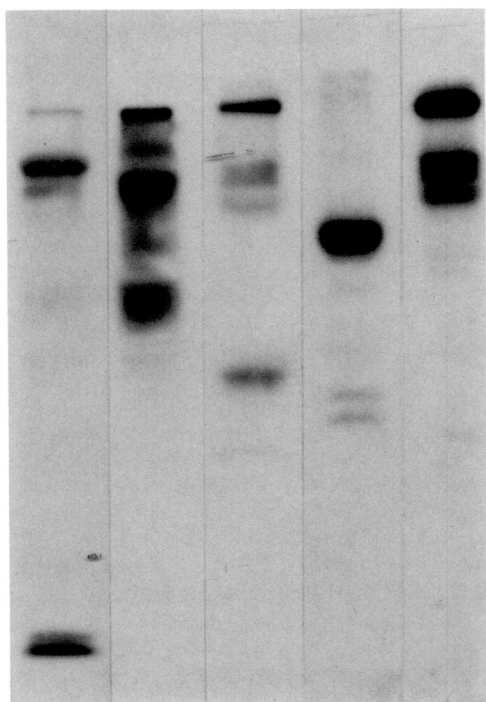


Fig. 4. Immunoblot analysis of antigens in SS extract recognized by antibodies from five representative sera of patients with DF-positive skin tests. Proteins of SS extract were separated by SDS-PAGE, transferred electrophoretically to a nitrocellulose membrane, and incubated in DF-sensitive sera and ^{125}I -labeled antihuman IgE. IgE binding was shown by autoradiography.

DF body and DF feces extracts and their corresponding antisera detected at least 35 and 20 antigens in body and feces extracts, respectively (Fig. 6A and 7A). Sera from all nine DF-sensitive reference patients also were CRIE positive and demonstrated varied IgE binding to the different antigen-antibody precipitin lines exhibited on the stained CIE plates (Table 3, Fig. 6B,C; 7B,C). Among the nine sera, the number of antigens that bound IgE for an individual serum ranged from two to six for DF bodies and from four to seven for DF feces. Control sera from DF-sensitive individuals showed no IgE binding to the DF antigens, and those autoradiograms were blank.

Discussion

This study directly examined cross-reactivity between *S. scabiei* var. *canis* and *D. farinae* by using well-defined extracts of the two mites. The results clearly demonstrate that *S. scabiei* var. *canis* shares a high degree of cross-reactivity with *D. farinae*. Previous reports have shown that DF body and DF feces extracts are the source of multiple antigens (Arlian et al. 1987a,b,c). Fourteen and seven of these antigens in DF body and DF feces extracts, respectively, are allergens, and they stimulate IgE

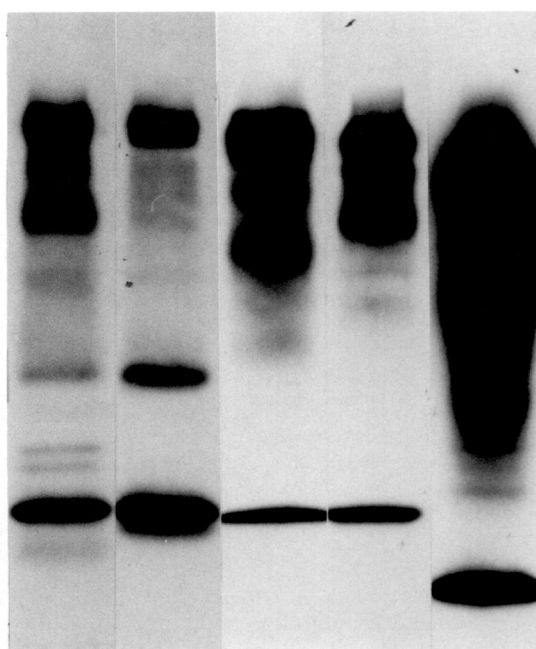


Fig. 5. Immunoblot analysis of antigens in DF recognized by antibodies from five representative sera of patients with DF-positive skin tests. SDS-PAGE gel-separated components of DF body extract were transferred to a nitrocellulose membrane; IgE binding was shown by autoradiography after incubation in DF-sensitive sera and ^{125}I -labeled antihuman IgE.

production in patients who are sensitive to *D. farinae*. Many of the antigens in DF bodies and DF feces are common, but some are specific to mite bodies. Likewise, it has been demonstrated that *S. scabiei* var. *canis* is the source of nine antigens (Arlian et al. 1985, Arlian in press).

The results of our investigation indicate that SS antigens are highly cross-reactive with some DF body and DF feces antigens. We have demonstrated that when DF body extract is reacted by CIE with sera from scabies-infested rabbits the sera contain antibodies directed against six SS antigens. Reciprocal reactions confirm the high level of cross-reactivity. Therefore, six of the nine SS antigens are cross-reactive with DF body antigens. Seven of the nine SS antigens are electronegative and two are electropositive (Arlian et al. 1985). All six of the SS antigens that cross-react with DF body antigens are electronegative. Likewise, the sera of scabies-infested rabbits also contain antibodies directed toward three electronegative DF feces antigens. Therefore, only one electronegative and two electropositive SS antigens share no antigenic determinants with DF antigens.

Given the high level of cross-reactivity between SS and DF and the large allergenic pool of DF, we expected that the cross-antigenicity between them would involve an IgE-type antibody response. IgE antibodies participate in the induction of allergic manifestations, and elevated serum IgE levels are

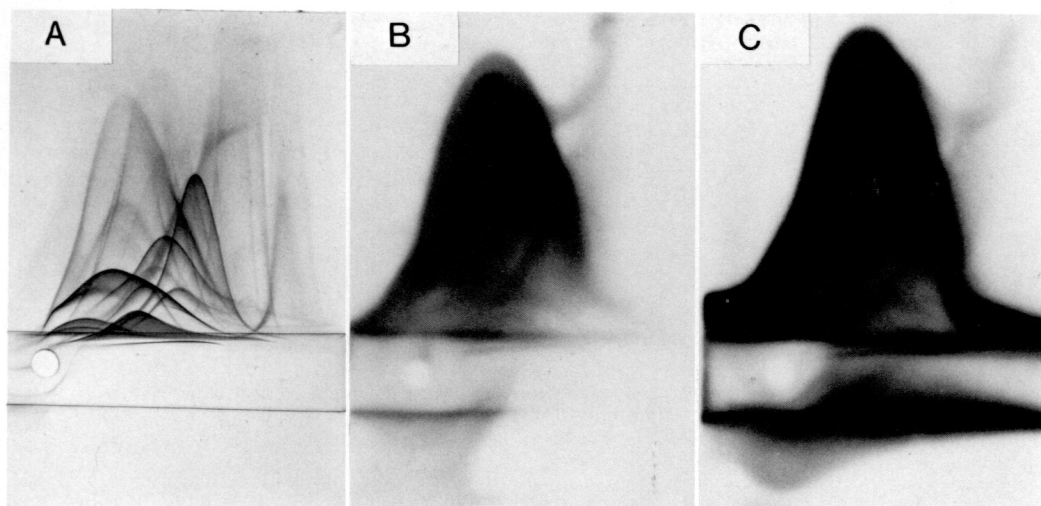


Fig. 6. (A) CIE pattern of DF body extract reacted with its corresponding antiserum. (B, C) Typical autoradiograms of different sera.

associated with allergic diseases and parasitic infections alike. This study shows that IgE-type antibodies directed against DF also recognize determinants on SS antigens. Fifteen SS bands separated by SDS-PAGE were found to bind IgE after being blotted following incubation in the sera of DF-sensitive patients. Furthermore, the fact that 15 bands bound IgE while only six electronegative cross-reacting antigens were identified by CIE indicates that at least some SS antigens exhibit multiple antigenic determinants. This high level of IgE binding was significant because the DF-sensitive patients had no history of scabies infestation. Non-allergic control sera, also from patients who had no history of *S. scabiei* infestation, showed no IgE binding to SS antigens.

Earlier studies have briefly reported that SS antigens cross-react with those from the house dust mites *D. farinae* and *D. pteronyssinus* (Falk et al. 1981, Arlian 1987), but no direct studies have documented that scabies patients produce elevated levels of specific IgE that is directed at *S. scabiei* and that cross-reacts with DF antigens as well. Indirect evidence suggested an allergenic relationship between scabies mites and house dust mites and elevated IgE levels directed at *S. scabiei*. Patients with scabies who also were sensitive to *D. pteronyssinus* have been reported to exhibit a more severe skin rash than do nonallergic persons (Falk & Bolle 1980). It also has been reported that some patients who have high levels of serum IgE and positive RAST test to *D. pteronyssinus* but no evi-

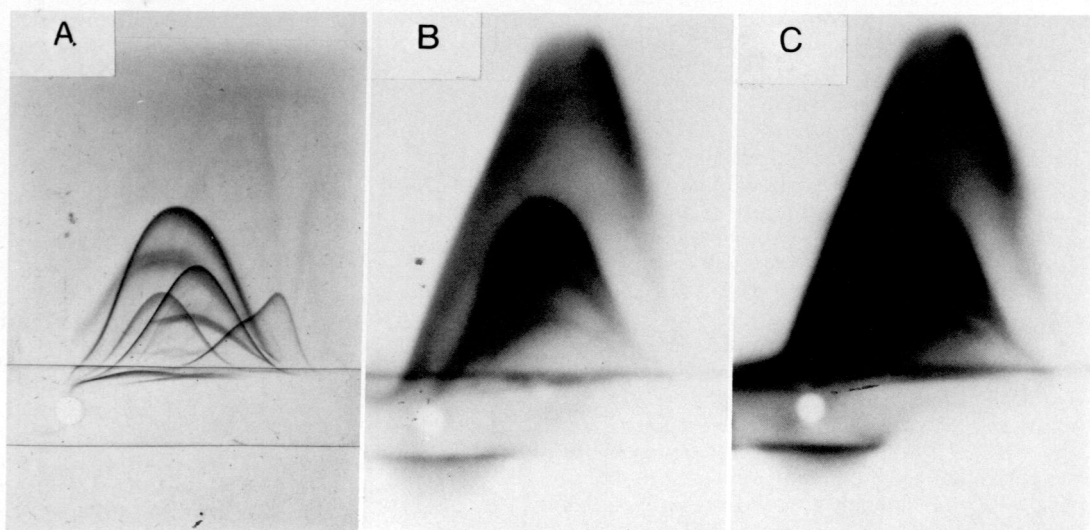


Fig. 7. (A) CIE pattern of DF feces extract reacted with its corresponding antiserum. (B, C) Typical autoradiograms of two sera.

Table 3. Number of DF body or DF fecal antigens that bound IgE in serum from DF-sensitive patients after analysis by CRIE

Patient serum ^a	No. DF body antigens exhibiting IgE binding		No. DF feces antigens exhibiting IgE binding	
	Strong ^b	Weak ^c	Strong ^b	Weak ^c
1	3	2	0	5
2	0	2	2	2
3	2	4	3	2
4	0	2	2	2
5	3	2	2	4
6	2	3	4	3
7	2	3	3	2
8	0	3	3	2
9	2	4	3	2

^a Corresponding numbers on Table 2 and Table 3 are the same patients' sera.

^b Intense radiostaining after 1-2-d autoradiography.

^c Radiostaining after 1-2-d autoradiography, but less intense.

dence of atopy show both reduced circulating total serum IgE and specific antibodies to *D. pteronyssinus* after they have been treated for scabies infestation. This suggests that scabies mites induce IgE antibodies and that these are also directed against *D. pteronyssinus* (Falk 1981). In another study, Falk (1980) found that 39 of 94 patients with scabies at the time of presentation had increased levels of total serum IgE. It was suggested that atopic disease was a possible explanation. By contrast, Hancock et al. (1974) found that 99 of 100 scabies patients examined had normal total serum IgE levels. We previously reported in a preliminary study that three of six nursing home patients who were infested with scabies exhibited circulating IgE directed at *S. scabiei* var. *canis* antigens (Arlian in press). However, their sensitivity to house dust mites was not determined, and therefore, it was unknown whether this sensitivity was the result of cross-reactivity with *D. farinae* antibodies. We expect that the IgE antibody response was to scabies mites because the prevalence of house dust mites in the nursing home was low and the patients had been confined for longer than one year.

The antigenic relationship between *S. scabiei* from different host species—in particular between var. *canis* and var. *hominis* of humans—remains to be investigated. However, because sufficient quantities of *S. scabiei* var. *canis* are available for preparing extracts while adequate quantities of other strains are not yet available, and because of the close taxonomic relationship between the various strains and species of scabies mites, var. *canis* serves as a suitable model in investigating the antigenic properties of *S. scabiei* and the host immune response. Just as a high degree of cross-antigenicity is exhibited for the taxonomically related house dust mites, *D. farinae* and *D. pteronyssinus*, it can be expected that scabies strains also should exhibit similar cross-antigenicity and share common antigens. Indirect evidence supports a close antigenic

relationship between strains of *S. scabiei*. Falk et al. (1981) reported that rabbit antiserum to *D. farinae* recognized four *S. scabiei* var. *hominis* antigens. The difference between four cross-reacting antigens reported for var. *hominis* and the six we identified for var. *canis* probably can be attributed to differences between laboratories in methodology and in the quantity and quality of extracts and antisera used. However, if it is assumed that there are strain differences between var. *canis* and var. *hominis*, it appears that they differ only by two antigens. Real antigenic differences between strains, if they exist, remain to be investigated systematically.

In summary, it is highly probable that scabies-infested patients exhibit circulating antibodies built to *S. scabiei* that also recognize determinants on *D. farinae* antigens. Likewise, DF-sensitive patients with no history of *S. scabiei* probably exhibit circulating antibodies to *D. farinae* antigens that also recognize determinants on *S. scabiei* antigens. Although this study clearly establishes a direct immunological link and a high level of cross-reactivity between *S. scabiei* and *D. farinae*, the implications in terms of individual or host susceptibility to scabies or in terms of manifestation of the disease are still to be determined. It is known that some humans and other mammalian hosts are predisposed to scabies (Orkin 1971, Arlian et al. 1984, Arlian & Vyszanski-Moher 1987). Likewise, mammals are also predisposed to other related mite species (e.g., Psoroptes) (Sweetman 1958, Arlian & Vyszanski-Moher 1987). At this time, no explanation can be provided for either case. As previously mentioned, it has been suggested that DF-sensitive patients infested with scabies may exhibit more severe signs and symptoms and that atopy could predispose to scabies infestation (Falk & Bolle 1980). This may be the result of cross-reactivity between the two mites. Therefore, because of the cosmopolitan occurrence of house dust mites, the high prevalence of dust mite sensitivity, and the high degree of cross-sensitivity to scabies and house dust mites, studies concerning the human susceptibility to scabies, details of IgE and other humoral responses to scabies, and the epidemiology of the disease now must be conducted.

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