

SKIN TEST AND RADIOALLERGOSORBENT TEST CHARACTERISTICS OF SCABIETIC PATIENTS

MARJORIE S. MORGAN, LARRY G. ARLIAN, AND STEPHEN A. ESTES

Department of Biological Sciences, Wright State University, Dayton, Ohio

Abstract. The scabies mite *Sarcoptes scabiei* and the *Dermatophagoides* house dust mites (HDM) are related phylogenetically and are the sources of several cross-reactive antigens. The purpose of this study was to investigate the immune response to *S. scabiei* and HDM in scabietic patients. Skin test sensitivity and serum IgE to both *S. scabiei* and HDM were determined for patients who had or previously had confirmed ordinary scabies. A retrospective group included nine subjects who had received successful treatment three weeks to one year prior to the study. A prospective group included 16 subjects with active scabies. Allergic histories were obtained, serum was collected, and skin prick tests (SPTs) were performed at enrollment for all and periodically over the next 12 months for the prospective patients. None of the individuals in either group reported a known sensitivity to HDM. Six of the nine retrospective patients were SPT positive to both *S. scabiei* and HDM and two of these showed circulating IgE specific for these antigens. At diagnosis, 13 of 16 patients with active scabies were SPT positive to *S. scabiei* and 12 of these were also SPT and/or radioallergosorbent test positive to HDM. Six patients had circulating IgE directed at both *S. scabiei* and HDM antigens while one subject had IgE to *S. scabiei* only and another had IgE directed at HDM only. Twelve of the 15 subjects tested also showed IgE and/or IgG binding to one or more bands on Western blots of an *S. scabiei*-specific protein fraction. This study indicated that approximately half of the patients with active scabies had *S. scabiei*- and HDM-specific circulating IgE while most patients cured of scabies lacked *S. scabiei*- and HDM-specific serum IgE. The data also suggested that antibodies to *S. scabiei* in scabietic patients also recognize HDM; however, some antibodies were directed at scabies-specific antigens.

Scabies is a parasitic skin disease caused by the mite *Sarcoptes scabiei*. Although estimates suggest that more than one million Americans have the disease at any given time, it remains difficult to diagnose since patients often present with a variety of pruritic rashes and other nonspecific symptoms.¹ A proven diagnosis requires the recovery of a scabies mite or its products and the specimens may be difficult to obtain because the technique requires experience. Improved testing methods are clearly desirable.

The development of a suitable diagnostic test has been hampered by the unavailability of a sufficient quantity of mite specimens from which to prepare suitable reagents and by the reports of cross-sensitivity and cross-reactivity with the phylogenetically related *Dermatophagoides* house dust mites *D. farinae* and *D. pteronyssinus*.^{2–5} The use of *S. scabiei* cultivated on suitable animal hosts has resulted in the production of larger quantities of useful antigen extracts.⁶ Less progress has been made, however, in elucidating the sources of the house dust mite cross-sensitivity.

The purpose of this study was to investigate the immune response to scabies in patients whose house dust mite sensitivities and allergic histories were also clearly defined to show that scabies mites possess species-specific antigens that may be used in a diagnostic test for scabies.

SUBJECTS, MATERIALS, AND METHODS

Study subjects. All subjects in this study had ordinary scabies that was confirmed by the recovery of *S. scabiei* mites by skin scrapes. At enrollment, all subjects provided informed consent and completed a questionnaire to assess the time since the onset of symptoms, allergic histories of themselves and their family members, and to identify any medications that might interfere with the experimental protocol. The protocol and questionnaires used in this study were reviewed and approved by the Human Subjects Com-

mittee of the Wright State University Institutional Review Board.

The study subjects were divided into two groups so that differences between the immune responses of those recently cured of scabies and those with active disease could be identified. The retrospective group was composed of nine subjects (four females and five males) 24–61 years of age who had reported exhibiting scabietic symptoms for two weeks to three months prior to diagnosis and who had received successful treatment for their disease within the previous year. These subjects provided a blood sample and were given skin prick tests (SPTs) as outlined below. The prospective group was composed of 16 patients (six females and 10 males) 20–72 years of age who had active scabies at the time of enrollment and who reported having symptoms for 0–13 months prior to diagnosis. At their initial visit, these patients were treated for scabies in addition to providing a blood sample and agreeing to an SPT. Six to 10 weeks later, these patients returned to confirm that their disease had been cured and to undergo a second blood sampling and SPT. Blood was also drawn from these patients at six and 12 months after treatment.

Skin prick test and radioallergosorbent (RAST) test. Extracts of *D. farinae* (30,000 AU/ml), *D. pteronyssinus* (30,000 AU/ml), and rabbit epithelium (Rb Ep; 1:10), as well as positive (histamine; 1.0 mg/ml) and negative controls were obtained from Bayer Pharmaceuticals (Spokane, WA). A sterile extract of *S. scabiei* var. *canis* was prepared as described below and was diluted to 0.5 mg of protein/ml using sterile glycerol diluent. The SPTs were performed using DermaPiks® (Greer Laboratories, Lenoir, NC), read at 20 min, and scored using the method of Aas and Belin.⁷ Using this method, the area of the wheal produced by the negative control solution is –; the wheal area of the positive histamine control is +++; + is 25% of the positive control area; ++ is 50%; and ++++ is 200% of the control.

All serum samples were commercially tested for total IgE and a RAST was performed for specific IgE against *D. farinae*, *D. pteronyssinus*, and Rb Ep (Clinical Immunology and Allergy, Liberty, MO). Total IgE levels ≥ 100 U/ml were considered elevated and RAST scores were reported using the modified RAST scoring system, which ranges from 0 (no allergen-specific IgE present in the serum) to 6 (high levels of specific IgE).⁸

Mite extracts and rabbit antisera. An extract of *S. scabiei* var. *canis* was prepared from scabies mites harvested from the ears of scabies-infested rabbits as previously described.^{6,9} A mixed *D. farinae/D. pteronyssinus* extract was also prepared according to published methods.⁹ Protein contents of the extracts were determined using the method of Bradford with bovine plasma albumin as a standard.¹⁰

The pool of serum from scabies-infested rabbits used in this study has previously been characterized.¹¹ A pool of serum from *D. farinae/D. pteronyssinus*-immunized rabbits was prepared from 16 individual serum samples.⁹ Sera included in each antiserum pool were selected based on their individual crossed immunoelectrophoresis profiles.¹¹

Crossed immunoelectrophoresis (CIE) and crossed radioimmunoelectrophoresis (CRIE). Human scabies sera were screened for circulating IgE directed against *S. scabiei* and *D. farinae/D. pteronyssinus* allergens using CIE and CRIE.⁴⁻⁶ Ten microliters of mite extract (25 μ g of *S. scabiei* and 39 μ g of *D. farinae/D. pteronyssinus* protein) were electrophoresed in the first dimension 1% agarose gel for 30 min at 200 V. Antigen-containing lanes were excised and placed transversely on a sheet of GelBond® (FMC BioProducts, Rockland, ME). An upper gel (anodic) containing 250 μ l of rabbit antiserum in 3 ml of 1% agarose and a lower gel (cathodic) with 50 μ l of serum in 1 ml of agarose were poured and electrophoresed overnight at 50 V. Gel plates were washed, pressed, dried, and representative gels were stained with Coomassie Brilliant Blue R-250.

The presence of IgE directed against *S. scabiei* and/or *D. farinae/D. pteronyssinus* allergens circulating in the patients' sera was visualized by CRIE using unstained gels as previously described.⁴⁻⁶ Briefly, CIE gels were incubated overnight in human serum (0.5 ml of serum/gel) diluted with 20 ml 0.1 M phosphate buffer, pH 7.5, with 2 mg/ml of bovine serum albumin (BSA) and 1% Tween 20 (RAST buffer). The IgE-binding allergens were visualized by incubating gels with 1 million cpm of ¹²⁵I-labeled anti-human-IgE (Kallestad® Allercoat® RAST isotope reagent; Sanofi Diagnostics Pasteur, Chaska, MN) diluted in 20 ml of RAST buffer. Gels were washed, dried, and placed in cassettes with tungsten-enhancement screens and X-OMAT LS film (Eastman Kodak, Rochester, NY) at -80°C . Film was exposed for three, 12, or 28 days. To standardize and to make comparisons, all gels were processed identically in one batch with one lot of reagents.

Immunoisolation of *S. scabiei*-specific and *D. farinae/D. pteronyssinus* cross-reactive proteins. An anti-*D. farinae/D. pteronyssinus* immunoaffinity column was prepared to remove *D. farinae/D. pteronyssinus* cross-reactive proteins from an *S. scabiei* extract using an Immunopure® Protein A IgG Orientation Kit (Pierce Chemical Co., Rockford, IL). A 10-ml column was prepared using 23 ml of rabbit anti-*D. farinae/D. pteronyssinus* serum. All proce-

dures were performed according to the manufacturer's recommendations.

An aliquot of *S. scabiei* extract was diluted to 10 mg of protein/10 ml with 10 mM Tris, pH 7.5, and was loaded onto the column. Unbound proteins were collected and the column was washed with Tris loading buffer. Bound antigens were eluted with 0.1 M glycine, pH 2.8, and the eluate was immediately neutralized. The presence of *S. scabiei*-specific and *D. farinae/D. pteronyssinus* cross-reactive antigens in the unbound and bound protein fractions, respectively, was monitored by CIE. The unbound fraction was reappplied to the column and the process was repeated until CIEs showed that no anti-*D. farinae/D. pteronyssinus*-precipitable antigens were present in the unbound fraction (i.e., the fraction contained only *S. scabiei*-specific antigens).

Sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) and immunoblotting. These two procedures were performed to screen selected prospective subjects' sera against *S. scabiei*-specific and *D. farinae/D. pteronyssinus* cross-reactive proteins and to determine the molecular weights of IgE- and/or IgG-binding allergens/antigens contained in the two immunoaffinity column fractions. Electrophoresis was carried out on 12% acrylamide gels under nonreducing (no reducing agent added to the sample buffer) conditions using the method of Laemmli.¹² Each gel contained a single sample trough that was loaded with 150 μ g of protein and a small well that contained Kaleidoscope® prestained standards (Bio-Rad, Hercules, CA) to provide molecular weight references. The SDS-PAGE-resolved proteins were electrophoretically transferred to Pro-Blott® polyvinylidene difluoride membrane (Applied Biosystems, Foster City, CA) in 10 mM 3-(cyclohexylamino)-1-propanesulfonic acid, 10% methanol, pH 11, according to the manufacturer's recommendations. Blots were blocked overnight with a solution composed of 8 M urea, 0.1 M 2-mercaptoethanol, and 5 mg/ml of BSA.^{4,6} Membranes were placed in a Fast Blot Developer (Pierce Chemical Co.) and each slot was loaded with 0.2 ml of serum in 3 ml of RAST buffer for overnight incubation. Following extensive washing, the intact blot was removed from the apparatus and placed in a dish containing 2 million cpm of ¹²⁵I-labeled anti-human-IgE. Blots were washed, dried, placed in cassettes, and subjected to autoradiography as described above for CRIEs.

Following autoradiography, IgG antibody binding to the blots was detected using peroxidase-labeled goat anti-human-IgG (Fisher Scientific, Pittsburgh, PA) at a dilution of 1:2,000. All blots were developed for 5 min using the CND (4-chloro-1-naphthol/3,3'-diaminobenzidine/H₂O₂) chromogenic substrate of Young.¹³

RESULTS

Although 67% (6 of 9) of the retrospective subjects and 44% (7 of 16) of those in the prospective group reported previous allergic histories (predominantly hayfever, asthma, and atopic dermatitis), none of the 25 scabietic individuals in the study reported any history of house dust mite sensitivity or allergy.

All of the nine retrospective subjects that had been cured of scabies in the previous year had total IgE levels within

TABLE 1
Skin prick test (SPT) and radioallergosorbent test (RAST) data for retrospective subjects*

Subject no.	Total IgE	SS SPT	Rb Ep		DF		DP	
			SPT	RAST	SPT	RAST	SPT	RAST
301	99	+	+	0	++	0	-	1/0
302	34	++	+	0	-	0	+	0
303	6	-	-	0	-	0	-	0
304	5	+++	++	0	+	0	++	0
305	12	+	-	0	-	0	++	0
306	93	-	+	0	+++	5	++++	5
307	38	++	++	0	++	0	++	0
308	26	++	++	0	++	1	++	0
309	52	-	+	0	+	0	-	0

* SS = *Sarcoptes scabiei*; SPT = skin prick test; Rb Ep = rabbit epithelium; DF = *Dermatophagoides farinae*; DP = *D. pteronyssinus*. - = wheal area equal to that of the negative control; + = 25% positive control wheal area; ++ = 50% positive control; +++ = 100% positive control; ++++ = 200% positive control.

the normal range (≤ 100 U/ml; Table 1). Six (67%) of these subjects were SPT positive to *S. scabiei* extract and all except one (305) also produced a positive reaction to Rb Ep, a control from the host on which the scabies mites used to make the extract were grown. All of these persons were also SPT positive to *D. farinae* and/or *D. pteronyssinus*. Patient 303 had negative SPT and RAST results to all extracts. Two subjects (306 and 308) were both RAST and SPT positive to *D. farinae* and/or *D. pteronyssinus*. Two (306 and 308) of the nine sera had circulating IgE directed against *S. scabiei* or *D. farinae/D. pteronyssinus* allergens when probed by CRIE, and the IgE binding to the *S. scabiei* allergens was only faintly visible by autoradiography (Figure 1).

Half (8 of 16) of the prospective subjects had elevated total IgE levels at the time of diagnosis (Table 2), but there was no correlation between increased circulating IgE and reported allergic symptoms. All except one patient returned for follow-up testing, but only seven individuals completed the 12-month experimental protocol. Only subjects 401 and 406 showed a marked decrease (141 to 46 U/ml at 12 months for 401 and 248 to 178 U/ml at 10 weeks for 406) in total IgE following treatment for scabies, while the levels in most patients remained fairly constant (Table 2).

At presentation, 13 of 16 patients (81%) with active scabies exhibited skin test reactivity to *S. scabiei* and eight of them did so in the absence of any reaction against Rb Ep (Table 2). Twelve of these 13 patients (all except subject 407) were also SPT and/or RAST positive to *D. farinae* and/or *D. pteronyssinus*. Several persons were skin test sensitive to one or more extracts six weeks after having tested negative at the initial visit. All subjects were SPT reactive to *D. farinae* and/or *D. pteronyssinus* by the second visit and only subject 404 remained unreactive to *S. scabiei* extract by this time.

The CRIE was performed on all sera to further characterize the circulating IgE of these scabietic patients and to determine if any changes occurred in the 12 months following cure of the disease (Table 3). Seven of 16 subjects (44%) had circulating IgE that bound to the *S. scabiei*-homologous CRIE gels and all but one of these (subject 401) also had IgE that bound to the *D. farinae/D. pteronyssinus* CRIEs. These persons were also RAST reactive to *D. farinae* and/or *D. pteronyssinus* (Table 2). One patient (401) had IgE that bound to the *S. scabiei* allergens but not to the *D. farinae/D. pteronyssinus* allergens while another (412) exhibited bind-

ing to the *D. farinae/D. pteronyssinus* gels but not to the *S. scabiei* proteins. Additionally, the degree of IgE binding did not appear to diminish over the 12 months following treatment for scabies.

To determine if the IgE binding to *S. scabiei* proteins in the CRIE experiment was the result of binding to scabies-specific or *D. farinae/D. pteronyssinus* cross-reactive allergens, an anti-*D. farinae/D. pteronyssinus* immunoaffinity column was used to partition scabies extract into *S. scabiei*-specific (those antigens that did not bind to the column) and *D. farinae/D. pteronyssinus* cross-reactive (those antigens that bound to and were eluted from the column) fractions. The IgE and IgG binding to the proteins in these fractions was assessed by SDS-PAGE/immunoblotting (Table 3 and Figure 2). Twelve of the 15 persons (80%) tested showed IgE and/or IgG binding to one or more proteins in the *S. scabiei*-specific antigen fraction. Interestingly, all but one of the persons who had exhibited IgE binding to both *S. scabiei* and *D. farinae/D. pteronyssinus* CRIE gels also had IgE that bound to both *S. scabiei* extract fractions.

The patterns of both IgE and IgG binding to the *S. scabiei*-specific antigen blots were examined to determine if a characteristic protein was recognized by all the scabietic patients (Figure 2 and Table 4). Although 12 of the 15 individuals tested had IgE and/or IgG that bound to one or more bands on the *S. scabiei*-specific antigen blot, no more than two individuals had IgE that bound to any given protein and most of the IgE binding was only faintly visible. Five of the 15 subjects (33%) had IgG that bound to a 36.5-kD antigen. As in the CRIE experiment, the immunoblot profiles were the same at diagnosis and 12 months after treatment.

DISCUSSION

The results of this experiment clearly indicate that humans present a very heterogeneous immunologic response to infestation by *S. scabiei*. Less than half (7 of 16) of the patients with active scabies (prospective) and only two of nine cured subjects (retrospective) had *S. scabiei*-reactive circulating IgE and all but one of these individuals also had *D. farinae/D. pteronyssinus*-directed IgE.

Previous animal studies have suggested that dogs,^{14,15} rabbits,¹⁶ and pigs¹⁷ develop detectable circulating antibody titers to scabies antigens within 2-5 weeks of being experimentally infested. The majority of patients in this study had

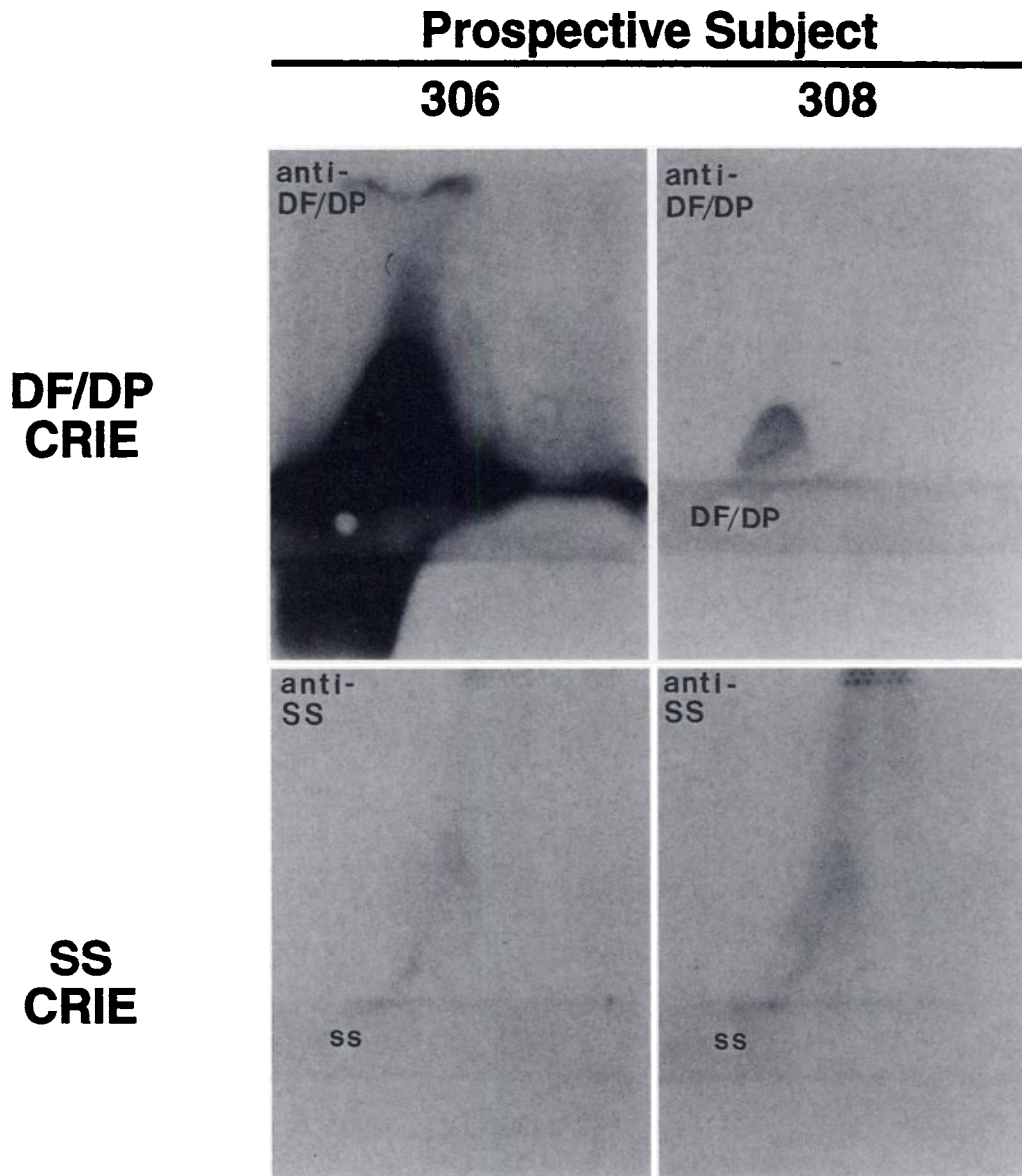


FIGURE 1. IgE-binding allergens on *Dermatophagoides farinae/D. pteronyssinus* (DF/DP) and *Sarcoptes scabiei* (SS) homologous crossed radioimmuno-electrophoresis (CIE) probed with the serum of subjects 306 and 308.

been infested with mites for at least that long. However, the typical mite burden in ordinary scabies has been estimated to be approximately 10–15 adult female mites,^{18, 19} while the animal studies reported above included experimental inoculations of > 1,000 mites. It is clear however, that chronic, untreated ordinary scabies can elicit in humans both *S. scabiei*-directed IgE and IgG production similar to that observed in other animal hosts.⁶

Our results are consistent with those of earlier studies of human scabies subjects that reported that less than half of scabies patients had elevated levels of total IgE.^{2, 3, 20–22} Interestingly, half of our subjects had very low IgE levels and there was no apparent correlation between IgE level and reported symptoms or immunologic response to scabies or house dust mites as measured by SPT, RAST, CIE, or immunoblotting. Additionally, the proportion of subjects with

circulating scabies-specific IgE (9 of 25 = 36% by CIE) is similar to that reported earlier.^{21, 22}

The positive immunologic responses to house dust mites in scabetic patients are a continuing concern. Although none of the subjects reported any known sensitivity to house dust mites, all lived in the vicinity of Cincinnati, Ohio, an area where most homes are inhabited by *D. farinae* and *D. pteronyssinus*.²³ Additionally, *S. scabiei* extracts are known to contain proteins that cross-react with both *D. farinae* and *D. pteronyssinus*.^{4, 5} Using the anti-*D. farinae/D. pteronyssinus* immunoaffinity column, we were able to separate *S. scabiei* proteins with *D. farinae/D. pteronyssinus* cross-reacting epitopes from those *S. scabiei*-specific proteins. Significantly, all of the subjects that had shown IgE binding to both *D. farinae/D. pteronyssinus* and *S. scabiei* CIE gels also showed IgE binding to both the blots containing *D.*

TABLE 2

Skin prick test (SPT) and radioallergosorbent test (RAST) data for prospective subjects at diagnosis and within 10 weeks of treatment for scabies*

Subject no.	Time	Total IgE	SS SPT	Rb Ep		DF		DP	
				SPT	RAST	SPT	RAST	SPT	RAST
401	Initial	141	++	++	1	++	1	++	1
	7 weeks	130	++	+	1	++	1	++	1
402	Initial	7	-	++	0	-	0	++	0
	8 weeks	7	+	+	0	++	0	++	0
403	Initial	274	++	-	1/0	+	1	++	1/0
	7 weeks	348	++	-	1	++	1	++	1
404	Initial	27	-	+++	0	-	0	+++	0
	7 weeks	24	-	-	0	++	0	++	0
405	Initial	QNS	++	++	QNS	++	QNS	-	QNS
	7 weeks	20	+++	++	0	-	0	+++	0
406	Initial	248	+	+	0	+	0	++	0
	10 weeks	178	++	+	0	++	1/0	++	0
407	Initial	6	+	-	0	-	0	-	0
	10 weeks	6	++	++	0	++	0	++	0
408	Initial	227	++	-	0	+++	4	+++++	4
	6 weeks	329	++	-	0	+++	4	+++	5
409	Initial	29	++	-	0	++	0	++	0
	6 weeks	24	++	-	0	++	0	++	1/0
410	Initial	8	++	-	0	+++	3	+++	3
	6 weeks	10	++	+	0	+++	3	+++	3
411	Initial	12	+++	++	0	++	0	++	0
	7 weeks	19	+	+	0	+	0	+	0
412	Initial	730	+	-	1/0	++	1	++	2
	8 weeks	717	++	-	1	++	1	++	1
413	Initial	100	+	-	0	+	1/0	+	0
414	Initial	>1,000	++	++	2	+++	4	+++	4
	8 weeks	>1,000	++	+	1	+++	3	+++	3
415	Initial	188	-	-	1/0	-	1	-	1
	6 weeks	174	++++	-	1/0	+++	1	+++	1
416	Initial	5	+	-	0	+	0	+	0
	6 weeks	5	+	-	0	+	0	+	0

* QNS = quantity not sufficient for analysis. See Table 1 for definitions of other abbreviations: - = wheal area equal to that of the negative control; + = 25% positive control wheal area; ++ = 50% positive control; +++ = 100% positive control; ++++ = 200% positive control; +++++ = 300% positive control.

TABLE 3

Crossed radioimmuno-electrophoresis (CRIE) and blot data for prospective subjects at diagnosis and 12 months after treatment for scabies*

Subject no.	Time	SS CRIE	SS-specific blot		DF/DP CRIE	SS × DF/DP blot	
			IgE	IgG		IgE	IgG
401	Initial	++	†	-	-	+	-
	12 months	+/-	†	-	-	+	-
402	Initial	-	-	†	-	-	-
	12 months	-	-	†	-	-	-
403	Initial	++	+	+++	+++	+++	-
	12 months	++	+	+++	+++	+++	-
404	Initial	-	-	+	-	-	-
405	Initial	ND	ND	ND	ND	ND	ND
	12 months	-	-	-	-	-	-
406	Initial	-	-	++	-	-	-
407	Initial	-	-	+++	-	-	-
408	Initial	+++	+	-	+++	++	-
409	Initial	-	-	+	-	-	-
	12 months	-	-	+	-	-	-
410	Initial	++	-	+	++	-	-
	12 months	++	-	+	++	-	-
411	Initial	-	-	-	-	-	-
	12 months	-	-	-	-	-	-
412	Initial	-	-	-	+++	+	-
413	Initial	-	-	+	-	-	-
414	Initial	+++	+++†	++†	+++	++	-
415	Initial	+++	+++†	-	++	++	-
416	Initial	+/-	ND	ND	+/-	ND	ND

* See Table 1 for definitions of abbreviations. CRIE scoring: +++ = first seen at 3 days; ++ = first seen at 12 days; + = first seen at 28 days; +/- = equivocal at 28 days. Blot scoring: one + per IgE- or IgG-binding band. ND = not determined.

† indicates poorly defined bands (shadow).

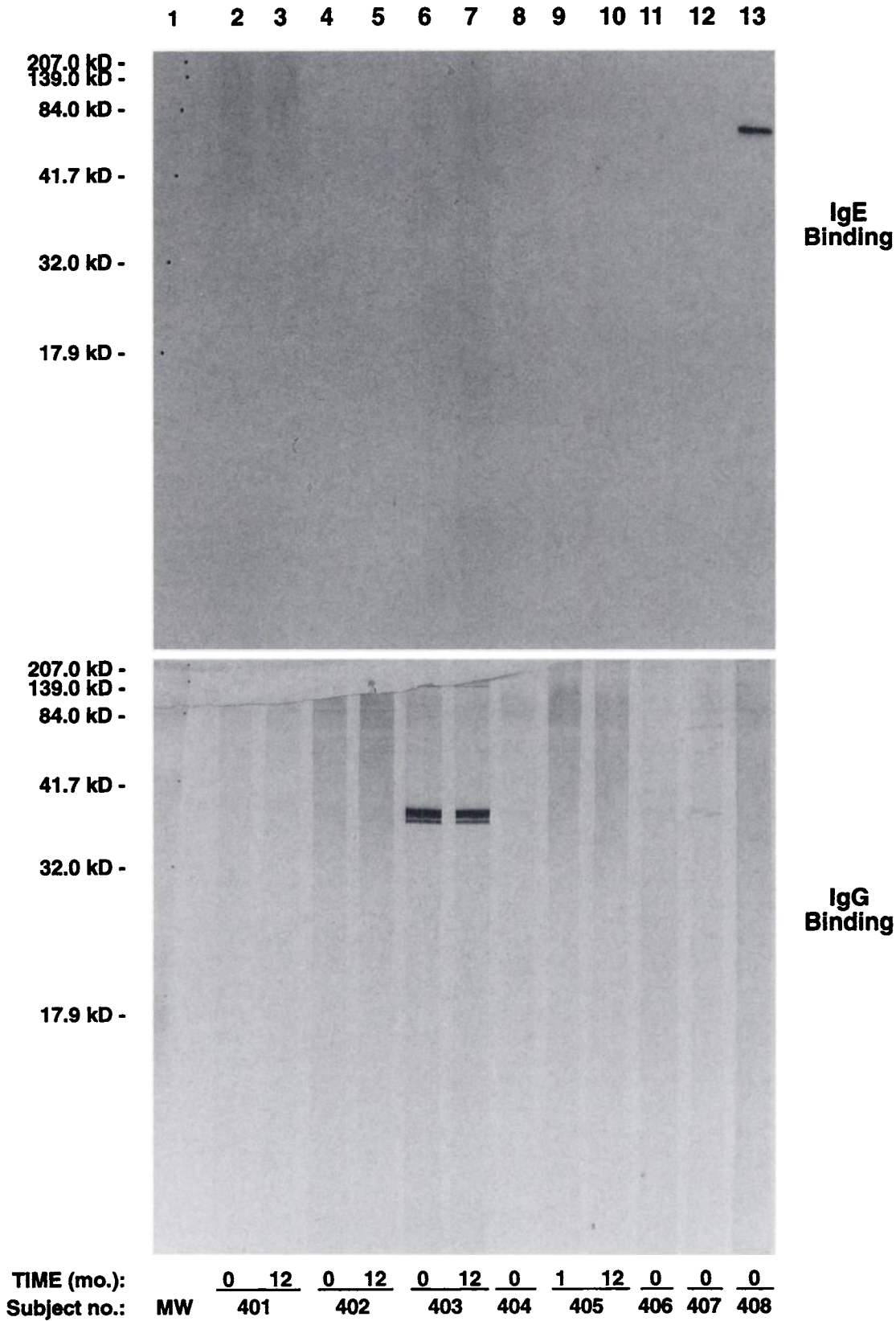


FIGURE 2. IgE (top) and IgG (bottom) binding to sodium dodecyl sulfate-polyacrylamide gel electrophoresis/immunoblots of *Sarcophyes scabiei*-specific proteins. Blots were probed with serum from representative prospective subjects collected at enrollment (time = 0) and 12 months after treatment for scabies. The molecular weights (MW) of reference markers are indicated in kilodaltons (kD).

TABLE 4

Scabies-specific proteins exhibiting IgE and IgG binding in the immunoblot experiment*

IgE-binding bands		IgG-binding bands	
MW (kD)	No. binding	MW (kD)	No. binding
220	2	120	1
69	1	83	1
43	2	73	1
38	1	58	2
18.5	1	43	1
		40	1
		36.5	5
		35.5	1

* Serum from 15 prospective patients was tested and the number of subjects exhibiting antibody binding is shown. MW = molecular weight; kD = kilodaltons.

farinae/D. pteronyssinus cross-reactive and *S. scabiei*-specific proteins, suggesting that the binding to the *D. farinae/D. pteronyssinus* CRIE gels may be due to circulating IgE specific for epitopes on scabies antigens that cross-react with epitopes also present on *D. farinae/D. pteronyssinus* proteins. When these interfering proteins were removed, 12 (80%) of the 15 scabietic subjects tested showed IgE and/or IgG binding to one or more proteins in the fraction containing only *S. scabiei*-specific proteins. This suggests that this set of antigens may prove useful in diagnosing scabies; however a more extensive screening of sera from potentially false-positive subjects (e.g., those with confirmed allergies to house dust mites and other atopsics) must be undertaken to confirm this possibility.

Financial support: This work was supported by grant AI-17252 from the National Institutes of Allergy and Infectious Diseases, National Institutes of Health (Bethesda, MD).

Authors' address: Marjorie S. Morgan, Larry G. Arlian, and Stephen A. Estes, Department of Biological Sciences, Wright State University, Dayton, OH 45435.

Reprint requests: Larry G. Arlian, Department of Biological Sciences, Wright State University, Dayton, OH 45435.

REFERENCES

- Burgess I, 1994. *Sarcoptes scabiei* and scabies. *Adv Parasitol* 33: 235-292.
- Falk ES, Bolle R, 1980. IgE antibodies to house dust mite in patients with scabies. *Br J Dermatol* 102: 283-288.
- Falk ES, Bolle R, 1980. In vitro demonstration of specific immunological hypersensitivity to scabies mite. *Br J Dermatol* 103: 367-373.
- Arlian LG, Vyszynski-Moher DL, Gilmore AM, 1988. Cross-antigenicity between *Sarcoptes scabiei* and the house dust mite, *Dermatophagoides farinae* (Acari: Sarcoptidae and Pyroglyphidae). *J Med Entomol* 25: 240-247.
- Arlian LG, Vyszynski-Moher DL, Ahmed SG, Estes SA, 1991. Cross-antigenicity between the scabies mite, *Sarcoptes scabiei*, and the house dust mite, *Dermatophagoides pteronyssinus*. *J Invest Dermatol* 96: 349-354.
- Arlian LG, Morgan MS, Arends JJ, 1996. Immunologic cross-reactivity among various strains of *Sarcoptes scabiei*. *J Parasitol* 82: 66-72.
- Aas K, Belin L, 1974. Suggestions for biologic qualitative testing and standardization of allergen extracts. *Acta Allergol* 29: 238-240.
- Nalebuff DJ, Fadal RG, Ali M, 1981. An enthusiastic view of RAST in clinical allergy. *Immunol Allergy Prac* 3: 18-29.
- Arlian LG, Rapp CM, Morgan MS, 1995. Resistance and immune response in scabies-infested hosts immunized with *Dermatophagoides* mites. *Am J Trop Med Hyg* 52: 539-545.
- Bradford MM, 1976. A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. *Anal Biochem* 72: 248-254.
- Morgan MS, Arlian LG, 1994. Serum antibody profiles of *Sarcoptes scabiei* infested or immunized rabbits. *Folia Parasitol* 41: 223-227.
- Laemmli UK, 1970. Cleavage of structural proteins during the assembly of the head of bacteriophage T4. *Nature* 227: 680-685.
- Young PR, 1989. Enhancement of immunoblot staining using a mixed chromogenic substrate. *J Immunol Methods* 121: 295-296.
- Arlian LG, Morgan MS, Rapp CM, Vyszynski-Moher DL, 1996. The development of protective immunity in canine scabies. *Vet Parasitol* 62: 133-142.
- Bornstein S, Zakrisson G, 1993. Humoral antibody response to experimental *Sarcoptes scabiei* var. *vulpes* infection in the dog. *Vet Dermatol* 4: 107-110.
- Arlian LG, Morgan MS, Vyszynski-Moher DL, Stemmer BL, 1994. *Sarcoptes scabiei*: the circulating antibody response and induced immunity to scabies. *Exp Parasitol* 78: 37-50.
- Bornstein S, Zakrisson G, 1993. Clinical picture and antibody response in pigs infected by *Sarcoptes scabiei* var. *suis*. *Vet Dermatol* 4: 123-131.
- Mellanby K, 1942. Transmission and prevention of scabies. *Public Health* 55: 150-151.
- Mellanby K, 1944. The development of symptoms, parasitic infection and immunity in human scabies. *Parasitol* 35: 197-206.
- Falk E, 1980. Serum immunoglobulin values in patients with scabies. *Br J Dermatol* 102: 57-61.
- Rantanen T, Bjorksten F, Reunala T, Salo OP, 1981. Serum IgE antibodies to scabies mite. *Acta Dermatovener* 61: 358-360.
- Dahl JC, Schwartz B, Graudal C, Christophersen J, Henriksen SA, 1985. Serum IgE antibodies to the scabies mite. *Int J Dermatol* 24: 313-315.
- Arlian LG, Bernstein D, Bernstein IL, Friedman S, Grant A, Lieberman P, Lopez M, Metzger J, Platts-Mills T, Schatz M, Spector S, Wasserman SI, Zeiger RS, 1992. Prevalence of dust mites in the homes of people with asthma living in eight different geographic areas of the United States. *J Allergy Clin Immunol* 90: 292-300.