INFECTION AND IMMUNITY, Nov. 1990, p. 3765–3769
0019-9567/90/113765-05$02.00/0

Vol. 58, No. 11

Recognition of Binding Sites on *Candida albicans* by Monoclonal Antibodies to Human Leukocyte Antigens

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Received 2 May 1990/Accepted 13 August 1990

Candida albicans **exhibits examples of human molecular mimicry, including receptors resembling human steroid receptors and human complement receptor (CR)-like receptors that bind the complement fragments C3d and iC3b. To further characterize the epitopes of the *Candida* human CR-like molecules, a panel of monoclonal antibodies (MAbs) against epitopes within the human CR3 was used. MAbs Mol, M1/70HL, and 7C3 bound to the germ tube, as assessed by immunofluorescence. MAbs Leu15, 60.1, and 95G8 did not bind. Miscellaneous MAbs against other antigens on human leukocytes (B2, 3D9, and OKT4) did not bind. However, MY9, which recognizes a monocyte antigen, bound extensively to the germ tube. The binding of certain anti-CR3 MAbs suggested limited identity between the *Candida* CR3-like receptor and the human CR3. The binding of MY9 identified an antigen recognized by a MAb to a human cell antigen not previously known to exist on *C. albicans*.**

In 1985, Heidenreich and Dierich first described the binding of erythrocytes coated with human iC3b and C3d to the germ tubes of *Candida albicans* and *Candida stellatoidea* (17). They concluded that the germ tubes of these *Candida* species had surface receptors for bound complement components that functioned similarly to the human complement receptor 2 (CR2) and the human CR3 of host defense cells in their recognition of C3d and iC3b. In 1986, Edwards et al. (8) confirmed these findings and showed also that monoclonal antibody Mol (throughout this report, Mol refers to the monoclonal antibody directed against the Mol antigen), a monoclonal antibody directed to the alpha chain of the human CR3 receptor (CD11b), recognized antigenic constituents on the germ tube. Additionally, with high concentrations of antibody and erythrocyte targets prepared with small numbers of complement molecules, they showed blocking of the attachment of C3d-coated erythrocytes by monoclonal and polyclonal antibodies to the human CR2 receptor. Gilmore et al. reported the recognition of *Candida* cell wall antigens by OKM1 (14) and confirmed the recognition of surface molecules of *Candida* spp. by Mol. Later, they also showed that OKM1 and M1/70, two monoclonal antibodies that recognize CD11b, recognized *Candida* cell wall antigens (15). The findings of molecules on *Candida* spp. with characteristics of the human complement receptors, coupled with the discovery of hormonal receptors on the organism, represent exciting examples of molecular mimicry that may have significance for pathogenicity.

To further define this mimicry by *C. albicans* of human epitopes on the surface of the organism, we conducted studies with recognition by monoclonal antibodies to human antigens. We included antibodies to human complement receptors to establish similarities or differences between receptors and receptorlike molecules as well as antibodies to miscellaneous human antigens. During this process, we found binding of a *Candida* surface antigen by a monoclonal antibody, MY9, which recognizes a class of antigens not previously reported to bind to *Candida* cells. MY9 binds to a differentiation/maturity marker on human phagocytic cells (CD33) and is not known to recognize human complement receptors. This antigen may represent another example of human molecular mimicry by *Candida* spp.

MATERIALS AND METHODS

Organisms. Seven strains of *C. albicans* (ATCC 36082, 36086, and 36802 as well as B311, 4918, 4918-10, and SC5314) and isolates of *C. tropicalis* and *C. parapsilosis* from patients were used. They were maintained at 4°C on YDK agar slants (yeast extract [Difco, Detroit, Mich.], dextrose, and potassium phosphate). Organisms were inoculated into yeast nitrogen base broth (Difco) supplemented with 1% dextrose (J. T. Baker, Phillipsburg, N.J.) and 0.15% L-asparagine (Calbiochem, San Diego, Calif.) and grown overnight at 27°C on a rotator.

These stationary-phase organisms were sonicated (Branson Sonic Power 350) for 3 s (setting 4), yielding greater than 95% singlet blastospores (viability, determined by trypan blue exclusion, was unaltered). This suspension was centrifuged at 700 × *g* for 7 min and washed twice in 0.85% NaCl. The organisms were suspended in Dulbecco phosphate-buffered saline (DPBS), sonicated for an additional 3 s, counted by hemacytometer, and adjusted to the desired concentrations for adsorption (see below) or for use within the monoclonal antibody binding assay, as either treated or untreated organisms (see below).

Monoclonal test antibodies. Many antibodies tested for binding to *C. albicans* germ tubes were anti-human CR3 antibodies. The antibody name, epitope recognized, and immunoglobulin class are listed in Table 1. All antibodies were tested against strain ATCC 36082 at a final concentration of 400 μg/ml. The immunoglobulin M (IgM) anti-CR3 monoclonal antibodies were also tested with strain B311, and monoclonal antibody MY9 was tested with all nine *Candida* strains. Multiple additional strains of *C. albicans*

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TABLE 1. Antibodies used in immunofluorescence assay

Antibody	Epitope recognized or function	Class[a]	Supplier
Mol	CR3 α-chain (CD11b)	IgM	Coulter Immunology
M1/70HL	CR3 α-chain	IgM	Hybritech
60.1	CR3 α-chain	IgG	Gift (P. G. Beatty)
Leu15	CR3 α-chain	IgG	Becton Dickinson
95G8	CR3 β-chain (CD18)	IgM	P.I.'s stock[b]
7C3	CD15	IgG	P.I.'s stock
3D9	CR1	IgG	P.I.'s stock
H9	Agglutinating	IgM	Gift (J. E. Cutler)
C6	Agglutinating	IgM	Gift (J. E. Cutler)
OKT4	T-cell antigen (CD4)	IgG	OrthoImmune
MY9	Monocyte antigen (CD33)	IgG	Coutler
B2	B-cell antigen (CD21)	IgM	Coulter
MsIgG	—[c]	IgG	Coulter
MsIgM	—	IgM	Coulter

[a] All were of mouse origin except M1/70HL (rat origin).
[b] P.I., Principal investigator.
[c] —, Not directed at specific epitope.

TABLE 2. Relative anti-CR3 and miscellaneous antibody binding to *C. albicans* ATCC 36082

Antibody type	*C. albicans* ATCC 36082				
	Positive			Negative	
	Antibody	Epitope or function	Immunofluorescence score[b]	Antibody	Epitope or function
Anti-CR3	M1/70HL	CD11b	2.5	95G8	CD18
	Mol	CD11b	3	60.1	CD11b
	7C3	CD15	2.5	Leu15	CD11b
Miscellaneous	MsIgM	—[c]	2	B2	CD21
	MY9	CD33	4	3D9	CR1
	MsIgG	—	4	OKT4	CD4
	C6	Agglutinating	4[d]	Mouse ascites	—
	H9	Agglutinating	4[d]		

[a] Except where noted, all binding was to the germ tubes only.
[b] Graded 1 to 4 on a visual scale (see text).
[c] —, Not directed at specific epitope.
[d] blastospores only stained, germ tubes did not stain.

were not evaluated with the other monoclonal antibodies because of prohibitive costs. Negative-control immunoglobulin class antibodies were B2 as an IgM control and 3D9 as the IgG control.

Adsorption of second antibody. Overnight cultures were prepared and inoculated into 200 ml of germination medium (0.075% gelatin [Difco], 0.15% sucrose [J. T. Baker]) at 5×10^6 blastospores per ml. These organisms were incubated at 37°C with slow stirring (36 rpm) for 2 h to induce germination. The germ tubes were centrifuged at 1,000 × *g* for 10 min and washed once in 0.85% NaCl. The pellet was suspended in 3 ml of PBS and divided into three 1.5-ml microfuge tubes. These tubes were spun for 2 min at 9,000 × *g*, 4°C, and the supernatant was discarded.

The microfuge tubes were placed in ice, and the second antibody, fluorescein isothiocyanate-conjugated goat (FITC-GT) anti-mouse IgG or FITC-GT anti-rat IgG (Cappel, West Chester, Pa.) diluted to 1:150 (a maximum volume of 450 μl), was sequentially adsorbed with a total of 10^9 germ tubes (3×10^8 germ tubes per microfuge tube). This suspension was incubated on ice for 30 min with intermittent vortexing and then spun at 9,000 × *g* for 2 min at 4°C for each adsorption. Following the three adsorptions, the supernatant was transferred to a clean tube and centrifuged as above. The supernatant was collected and stored at 4°C in the dark overnight.

Immunofluorescent antibody binding assay. Fresh overnight cultures of *C. albicans* were prepared and adjusted to 5×10^5 blastospores per ml in germination medium. To each well of an eight-chamber Lab-Tek (Miles Scientific, Naperville, Ill.) slide, 200 μl of organisms was added and allowed to germinate at 37°C for 90 min. The wells were rinsed twice in DPBS and blocked with 200 μl of 1% bovine serum albumin (BSA; Sigma, St. Louis, Mo.) and 0.1% Tween 20 (Sigma) for 30 min at 4°C. The blocking BSA solution was replaced with 200 μl (from the 400-μg/ml dilutions) of the antibody to be tested (80 μg per well) and incubated for 60 min at 4°C. The wells were then rinsed three times with PBS, and 150 μl of adsorbed FITC-GT anti-mouse or FITC-GT anti-rat IgG was added. The slide was then incubated at 4°C in the dark for 30 min. The wells were washed three times in PBS. The Lab-Tek superstructure and gasket were removed, and an antiquencher of 90% glycerol (Sigma)–10% *p*-phenylenediamine (Sigma) was added. A cover slip was applied, and the slide was stored at 4°C in the dark until viewed with a Zeiss phase-contrast epifluorescence microscope. The amount of immunofluorescence was scored on a scale of 1 to 4 as follows: 1, barely detectable; 2, definite immunofluorescence but minimal in amount; 3, moderate immunofluorescence; 4, heavy staining (Fig. 1 shows grade 4 immunofluorescence). Each slide was read by two observers.

MY9 antigen characterization. *C. albicans* ATCC 36082 and SC5314 germ tubes were treated with heat, pronase, or chymotrypsin by modifications of the method of Sundstrom and Kenny (33). Organisms were adjusted to 5×10^6/ml in germination medium, and 200-μl samples were added to Lab-Tek 8 slides. Following germination, wells were rinsed twice in calcium- and magnesium-free PBS (PBS-CMF). Heat treatment consisted of placing a slide at 100°C for 30 min. Pronase (Calbiochem, San Diego, Calif.) was added at 2.5 mg/ml in PBS-CMF to a slide at 37°C for 10 min. Chymotrypsin (Sigma Chemical Co., St. Louis, Mo.) was added at 2.5 mg/ml in PBS-CMF to a slide at 37°C for 2 h. At the end of each treatment, wells were rinsed four times with PBS-CMF and placed at 4°C with BSA-Tween 20 for 30 min. The immunofluorescent binding assay then was continued as above.

RESULTS

Fluorescence-activated cell sorter analysis was performed in our preliminary studies, but greater than 50% of the organisms of each sample adhered to the internal components of the instrument and were irretrievable. Since such a high rate of loss would likely be associated with measuring a nonrepresentative cell population, we used standard light microscopic immunofluorescence techniques. Additionally, we were interested in surface expression of antigens and used whole (nonsolubilized), live (non-fixed) organisms. The results of the immunofluorescence assay are in Table 2, showing that certain anti-human CR3 monoclonal antibodies recognized the *Candida* germ tube antigens while others did not. In our panel, the anti-human CR3 alpha-chain monoclonal antibodies, Mol and M1/70, bound to the *Candida* germ tubes; leu15 and 60.1 did not. An anti-human CR3 beta-chain antibody (21), 7C3, bound to the *Candida* germ tubes, while

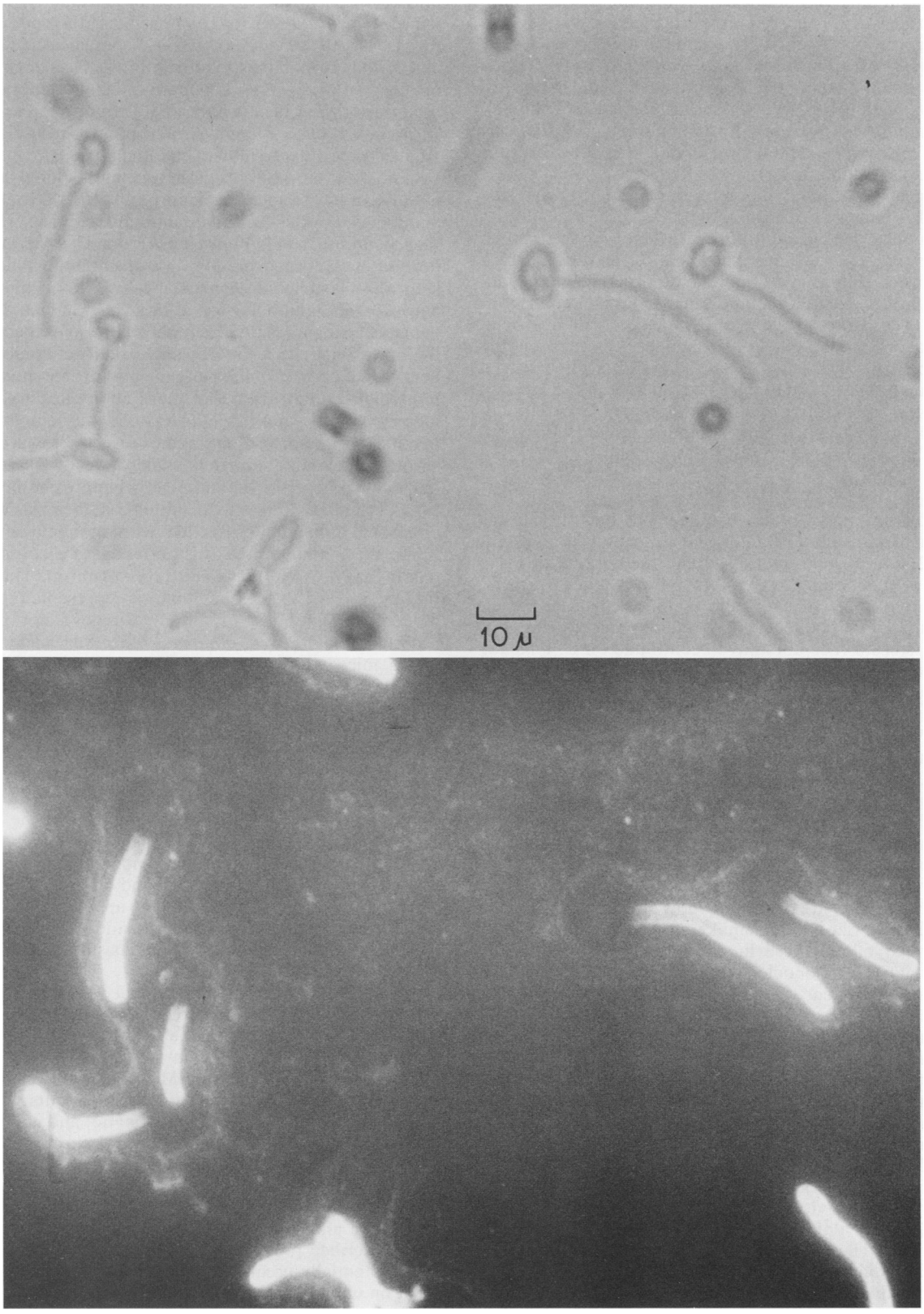

FIG. 1. (Top) Light field of *C. albicans* stained with MY9 monoclonal antibody. (Bottom) Same field under fluorescent lighting, showing MY9 adhering to the germ tube portion of the organism. Bar, 10 μm.

no binding occurred with 95G8. Recognition by monoclonal antibodies directed at epitopes other than human CR3 antigenic epitopes also occurred. Of particular interest was the strong recognition, distinctly restricted to the germ tubes, by MY9 (Fig. 1) on all seven of the *C. albicans* strains tested (see Discussion). Following heat, pronase, or chymotrypsin treatment, binding by MY9 was negative (data not shown). Also negative were 3-day-old hyphal cultures of *C. albicans* 36082, *C. parasilosis* (patient isolate), and *C. tropicalis* (patient isolate) (data not shown).

Binding of germ tube sites by mouse IgM (MsIgM) and MsIgG also occurred. These antibodies were originally included as class-specific controls, but when they were found to be positive, they were replaced by B2 and 3D9, which were negative. Intensity of immunofluorescence among antibodies varied, even though the same 400 μg/ml concentration was used. Both anti-human CR3 alpha-chain and beta-chain monoclonal antibodies recognized epitopes on the germ tube (see Table 1 for antibody descriptions), and recognition of germ tube antigens spanned both the IgG and IgM classes. In contrast to all the other monoclonal antibodies tested, both C6 and H9 (agglutinating antibodies) stained only the blastospore portion of the organism.

DISCUSSION

Molecular mimicry of human antigens by microorganisms has been of considerable interest in recent years (10–13, 26–28, 32, 34, 36), particularly within the context of these molecules functioning as microbial virulence factors. Knowledge of molecular mimicry within the field of mycology is limited at present but is rapidly increasing. For example, steroid receptors have been found on *C. albicans* by several investigators (6, 20, 23, 31; C. L. Frey, D. P. McDonnell, T. R. Butt, and D. J. Drutz, Program Abstr. 28th Intersci. Conf. Antimicrob. Agents Chemother., abstr. no. 28, 1988).

The more recent discovery of surface molecules that recognize sheep erythrocyte targets coated with iC3b (CR3) and C3d (CR2), the isolation and characterization of the CR2-like molecules on *Candida* spp., and the recognition of the surface of *Candida* spp. by monoclonal antibodies to human complement receptors (5, 8, 9, 17, 22, 25) have intensified interest in molecular mimicry by *Candida* spp.

The studies we are reporting now were conducted to further characterize the humanlike antigenic epitopes on *C. albicans* that mimic these human complement-like receptors. Our anti-CR3 monoclonal antibody results both extend and remain consistent with the findings of other groups (9, 14), in that some (particularly Mol and M1/70HL) but not all monoclonal antibodies specific for human CR3 recognized the *Candida* human CR3-like receptors. Further, we found 7C3 positive with a distribution on germ tubes similar to that of Mol and M1/70HL. Indirect evidence suggests that 7C3 may recognize an epitope on the beta-chain of CR3 (21). This monoclonal antibody recognizes the carbohydrate sequence of lacto-*N*-fucopentaose III, designated CD15 (2), which is present on the CR3 of neutrophils and in other neutrophil proteins. The positivity of 7C3 demonstrates recognition of both beta- and alpha-chain epitopes of human CR3 on *Candida* spp. These findings, coupled with the previously described binding of both iC3b- and C3d-coated erythrocytes to the germ tubes (8, 9, 17), strongly uphold the hypothesis that while the *Candida* human CR3-like molecule differs in molecular configurations from the human CR3 molecule, they have certain configurational characteristics of the human CR3 receptors as well as the human CR2.

The agglutinating monoclonal antibodies C6 and H9 bound only to the blastospore portion of the germinated organism in our system. However, the expression of the epitopes recognized by both these antibodies is strain and age dependent, and germ tube staining by both antibodies has been identified in other studies (3, 4). Having monoclonal antibodies that bind only to the blastospore and are not expressed on the germ tube in our system may be useful for future adherence studies (especially to human endothelial cells), when used in combination with the anti-germ tube monoclonal antibodies.

Early in our studies, we incorporated MsIgM and MsIgG antibodies as class-specific antibody controls. However, they bound at levels above background to the germinated portion of the organism. Why these antibodies did not also stain the blastospore (nonspecifically) is not known, since anti-*Candida* antibodies found in normal human serum recognize blastospores preferentially (18). We speculate that their positivity is a result of being produced in mice that had been exposed to *Candida* organisms in their normal flora. Regardless, recognizing that these antibodies are not satisfactory controls in a *Candida* system is essential for investigations of monoclonal antibodies directed toward *Candida* surface humanlike antigens. Therefore, we switched to monoclonal controls B2 and 3D9, which tested negative.

Of particular interest to us in these studies was the finding of strong immunofluorescence with monoclonal antibody MY9. We initially chose this antibody as a control, anticipating that it would not bind to the organism. This recognition by MY9 of a germ tube epitope represents binding by a monoclonal antibody of a new category not previously reported to bind to *C. albicans*. MY9 was made by immunizing mice with cells from a patient in blast crisis with chronic myelogenous leukemia (1, 7, 16, 24). The human antigen it recognizes is expressed by blasts, promyelocytes, and myelocytes in the marrow and by monocytes in the peripheral blood and gradually decreases during granulocyte differentiation. By immunoblotting, MY9 is known to recognize an antigen on human leukocytes with a molecular mass of 68 to 70 kDa, which has been designated the leukocyte differentiation antigen, CD33 (19, 29), and may be carbohydrate in structure. When immunoprecipitation was performed on the CD33, a discrete band on the sodium dodecyl sulfate-polyacrylamide gel did not appear (16). Additional evidence for carbohydrate determinant recognition by MY9 has been found from structural analysis of the CD33 antigen (30).

We considered the possibility that Mol and MY9 might cross-react. However, Mol recognizes the CD11b through the arginine-glycine-asparagine (RGD) ligand (35). The molecular mass of the CD11b is 160 kDa (19, 29). The only characteristics that MY9 and Mol apparently have in common is their presence on blast cells in patients with certain leukemias (myelomonocytic, monocytic, and acute granulocytic for Mol; acute myelogenous and chronic myeloid for MY9) and their presence on certain monocytes (peripheral blood monocytes for MY9 and adherent monocytes for Mol). Therefore, it is likely that MY9 is recognizing a humanlike antigenic epitope on *Candida* cells that has not been described previously.

Of potentially great importance is work by Simmons and Seed on cloning and sequencing the CD33 (30). The cDNA sequence they determined is predictive for a polypeptide of 40 kDa, a glycosylated membrane protein. They have been successful in inducing expression of the CD33 in genomic transfectants. Production of genomic transfectants may be-

come critical in studying the biological importance of CD33 expression on *Candida* spp. and perhaps other organisms.

What advantage, if any, the molecular mimicry of the human antigenic epitope recognized by MY9, or any other monoclonal antibodies recognizing human antigens, provides the organism is not known. However, current molecular biological techniques will probably enable transfection of nonpathogenic organisms for antigen expression and subsequent elucidation of its importance.

ACKNOWLEDGMENTS

We thank Patrick G. Beatty for supplying antibody 60.1, James E. Cutler for supplying antibodies C6 and H9, Richard A. Calderone for supplying *C. albicans* 4918 and 4918-10, Paula Sundstrom for supplying *C. albicans* SC5314, Stephen M. Puentes and Carrie L. Frey for their advice and assistance, and Holly Edwards for secretarial service. The IMT-2 Olympus phase-contrast microscope used in these studies was graciously donated by Toyota U.S.A.

Supported by Public Health Service grants AI-19990 and AI-15338 from the National Institutes of Health.

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