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Evidence for the Presence of a High-Affinity Laminin Receptor-Like Molecule on the Surface of *Candida albicans* Yeast Cells

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Two polypeptides of 37 and 67 kDa that bind laminin were detected in cell wall extracts of *Candida albicans* blastoconidia. The 37-kDa species, found only in yeast cell wall extracts, cross-reacted with a rabbit polyclonal antibody (PAb 4160) directed towards the carboxyl-terminal laminin-binding domain present in the human 67-kDa high-affinity laminin receptor (67LR) and its 37-kDa precursor (37LRP), whereas another antibody (PAb 4056), directed against internal domains of 67LR and 37LRP, recognized a 37-kDa species in wall extracts from both blastoconidia and germinated blastoconidia. Indirect immunofluorescence with PAb 4160 showed a patchy binding pattern only on yeast cells that represented about 10% of the entire blastoconidia population.

Adhesion of the opportunistic pathogen *Candida albicans* to host tissues seems to be an essential factor for both commensal colonization and establishment of disseminated candidiasis, which is the gravest form of disease caused by this organism. Attachment of the fungus, mediated by the interaction between complementary molecules on both parasite and host surfaces (i.e., endothelium and/or subendothelial extracellular matrix), is thought to play an important role in the dissemination of *C. albicans* cells in vivo and the establishment of metastatic sites of infection throughout the body (15). In this regard, a receptor for fibronectin, which is believed to be one of the animal host ligands for *Candida* adhesion (4), on *C. albicans* cells has recently been identified (14). This receptor also appears to mediate the adherence of fungal cells to other protein components of the extracellular matrix, such as laminin and collagen types I and IV (14, 15). Additionally, candidal receptors for other mammalian serum proteins, such as C3d (3), iC3b (13), and fibrinogen (7), have been characterized (for a review on adherence and receptor relationships in *C. albicans*, see reference 2).

Laminin, a major component of the basement membrane (31), is a large multidomain glycoprotein which seems to play a critical role not only in normal cell adhesion but also during tissue invasion and metastasis by tumor cells and microorganisms. In this context, specific laminin receptors have been identified on tumor cells and pathogenic bacterial species (17, 25, 30, 35); recently, receptors for laminin have also been detected on *C. albicans* cells (1). Some of the laminin-binding proteins already identified from animal cells belong to the integrin family, whereas others belong to the so-called nonintegrin receptors (21, 27). Among the latter, a 67-kDa laminin receptor (67LR) and its 37-kDa precursor (37LRP), both of which bind to laminin with high affinity, from human cancer and normal cells have been extensively characterized (8, 9, 26).

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C. albicans ATCC 26555 (serotype A) was propagated as blastoconidia or blastoconidia-bearing germ tubes basically by the procedure reported previously (5, 18), with the medium described by Lee et al. (16). β-Mercaptoethanol (βME) extracts containing protein and glycoprotein cell wall components and protoplast homogenates from both cell growth forms were obtained as already reported (6, 7). Sulfhydryl compounds such as dithiothreitol and βME are believed to solubilize protein and mannoprotein moieties that appear to be present in the outermost layers of *C. albicans* cell walls and that are mostly related to adherence, receptor, and enzymatic activities (2, 7, 18). The total sugar and protein contents in the different samples were determined colorimetrically (11, 19), with glucose and bovine serum albumin, respectively, as the standards.

The βME extracts and the protoplast homogenates were analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) under reducing conditions on slab gradient gels (5 to 15% polyacrylamide) and electrophoretically transferred to nitrocellulose paper for Western blotting (immunoblotting) as described elsewhere (5). When polyacrylamide gels were stained with Coomassie blue, βME extracts from blastoconidia (Fig. 1A, lane 1) and germinated blastoconidia (Fig. 1A, lane 2) were found to contain about 17 polypeptide chains with molecular masses ranging from 70 to 17 kDa, whereas total cell homogenates of blastoconidia (Fig. 1A, lane 3) and germinated blastoconidia (Fig. 1A, lane 4) protoplasts exhibited a very complex array of medium- to low-molecular-mass protein species.

After electrophoresis, proteins were electroblotted to nitrocellulose paper (Bio-Rad) and assayed for laminin binding as follows. The nitrocellulose membranes were blocked with 4% bovine serum albumin in 10 mM Tris-hydrochloride buffer (pH 7.4) containing 0.9% NaCl (TBS buffer) for 2 h at 37°C. The nitrocellulose sheet was washed once with TBS containing 0.05% Tween 20 (TBST) and incubated for 30 min at room

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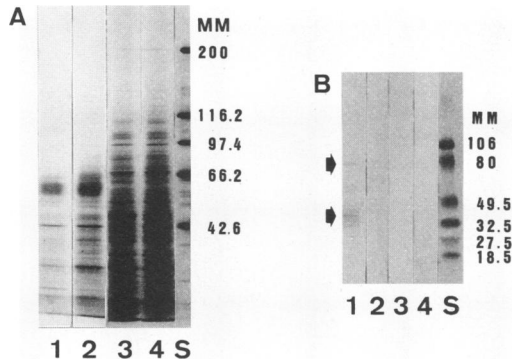

FIG. 1. Binding of human laminin to polypeptide species released from *C. albicans* cell walls. βME extracts (lanes 1 and 2; samples applied to each well contained ca. 300 μg of material, expressed as total sugar content) and protoplast homogenates (lanes 3 and 4; 40 μg of protein per well) from blastoconidia (lanes 1 and 3) and germinated blastoconidia (lanes 2 and 4) were separated by SDS-PAGE on slab 5 to 15% gradient gels and stained with Coomassie blue (A) or blotted to nitrocellulose (B). Arrows point to a 37-kDa and a 67-kDa species present exclusively in βME extracts of blastoconidia (lane 1) that exhibited reactivity after detection with the antilaminin antibody after incubation of the nitrocellulose sheet in purified human laminin (B). The molecular masses (MM) (in kilodaltons) of standard proteins are indicated to the right of each panel. Prestained molecular size standard proteins were run in parallel in lane S of panel B.

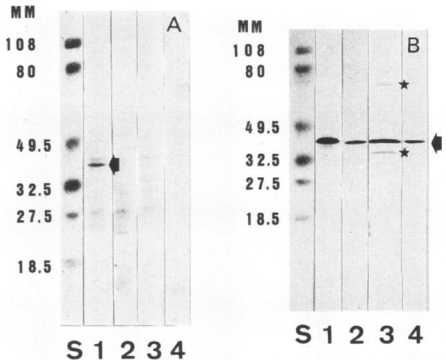

FIG. 2. Western immunoblot analysis of βME extracts (lanes 1 and 2) and protoplast homogenates (lanes 3 and 4) from blastoconidia (lanes 1 and 3) and germinated blastoconidia (lanes 2 and 4) reacted with PAb 4160 (A) and PAb 4056 (B). The amount of sample applied to each well is described in the legend to Fig. 1. Arrows in panels A and B point to a 37-kDa species that exhibited reactivity after detection with the two antibodies. Stars in panel B indicate other polypeptides present in protoplast homogenates from yeast cells that exhibited cross-reactivity towards PAb 4056. The molecular masses (MM) (in kilodaltons) of prestained standard proteins run in parallel in lane S are indicated to the left of each panel.

temperature with agitation in 10 mM phosphate buffer (pH 7.4) containing 1% bovine serum albumin, 0.9% NaCl, 1 mM $CaCl_2$, 1 mM $MgCl_2$, and laminin (50 μg/ml; Sigma). After being washed four times (10 min per wash) with TBST (the NaCl concentration in the buffer solution was increased to 1 M for the second wash), the blots were incubated with rabbit antilaminin antiserum (Sigma) at a 1:500 dilution in TBST plus 1% bovine serum albumin (TBSTB), washed as described above, and incubated again with peroxidase-labelled goat anti-rabbit immunoglobulin G (IgG) antiserum (Bio-Rad) at a 1:2,000 dilution in TBSTB. After agitation for 1 h at room temperature, the nitrocellulose sheets were washed again as described above, and reactive bands were developed with hydrogen peroxide and 4-chloro-1-naphthol as the chromogenic reagent.

Western blot analysis with the laminin-antilaminin antibody assay described above allowed detection of two species with apparent molecular masses of 37 and 67 kDa from βME extracts of *C. albicans* blastoconidia only (Fig. 1B, lane 1, arrows). No reactive bands were observed when the nitrocellulose sheet containing all the different samples was incubated with the antilaminin conjugate alone, indicating that the reaction was dependent on the previous interaction of the blotted polypeptides with laminin. The 67-kDa species seems to be mannoprotein, as determined from its reactivity with concanavalin A, whereas the 37-kDa laminin-binding component showed no reactivity towards the lectin (6).

Since the laminin-binding species observed in the βME extracts from yeast cells had apparent molecular masses very similar to those described for the human 67LR and 37LRP, we next investigated the reactivity of blotted polypeptides with affinity-purified rabbit polyclonal antibodies (PAb) raised to synthetic peptides deduced from the cDNA sequence of the 67LR species. These antibodies recognized different topologic domains of both 67LR and 37LRP: PAb 4160 is directed towards an extracellular carboxyl-terminal domain, located near the putative laminin-binding site (9), and PAb 4056 recognizes a putative internal domain (8). Immunodetection of blotted polypeptides was done as described previously (5), using the specific antibodies diluted 1:100 in TBSTB and peroxidase-labelled goat anti-rabbit IgG (Bio-Rad) as the indicator antibody (1:2,000 dilution in TBSTB). Reactivity with a 37-kDa species present exclusively in βME extracts from blastoconidia was observed when PAb 4160 was used as the probe (Fig. 2A, lane 1, arrow), whereas PAb 4056 cross-reacted with a major 37-kDa band present in all samples tested (Fig. 2B, lanes 1 to 4, arrow) and also with two other components of 67 and 34 kDa that exhibited a minor reaction towards the antibody. The latter proteins were detected only in the protoplast homogenates from yeast cells (Fig. 2B, lane 3, stars).

The surface expression of laminin receptor-like molecules in *C. albicans* cells was investigated by indirect immunofluorescence (IIF) with PAb 4160 as the probe. Specimens of blastoconidia and germinated blastoconidia were prepared for IIF analysis in a Cytospin 2 centrifuge (Shandon) at 1,000 rpm for 10 min. After centrifugation, cells were fixed with cold acetone for 5 min and reacted for 1 h at room temperature with PAb 4160 diluted 1:50 in 10 mM phosphate (pH 7.4)–0.15 M NaCl. After several washes, cytoslides were reacted with fluorescein isothiocyanate-conjugated pig anti-rabbit IgG (Dako). Slides were examined and photographed with a Leitz Labolux II photomicroscope.

Reactivity with PAb 4160 was found exclusively on the cell surface of blastoconidia (Fig. 3B and C), particularly on yeast cells that appeared to have a larger average size than the other cells in the preparations (Fig. 3E, arrows). These cells represented roughly 10% of the entire blastoconidia population and exhibited a patched fluorescence pattern on their surfaces (Fig. 3B and C). Reactivity appeared to depend on the previous interaction of the specific antibody (PAb 4160) with cells, since fluorescence was not observed when the cells were reacted with the second fluorescein isothiocyanate-labelled marker antibody alone. Cell-bound fluorescence was not observed on mycelial filaments or on blastoconidia from which germ tubes emanate with PAb 4160 (Fig. 3). A very faint fluorescence was observed on both yeast cells and mycelial filaments when

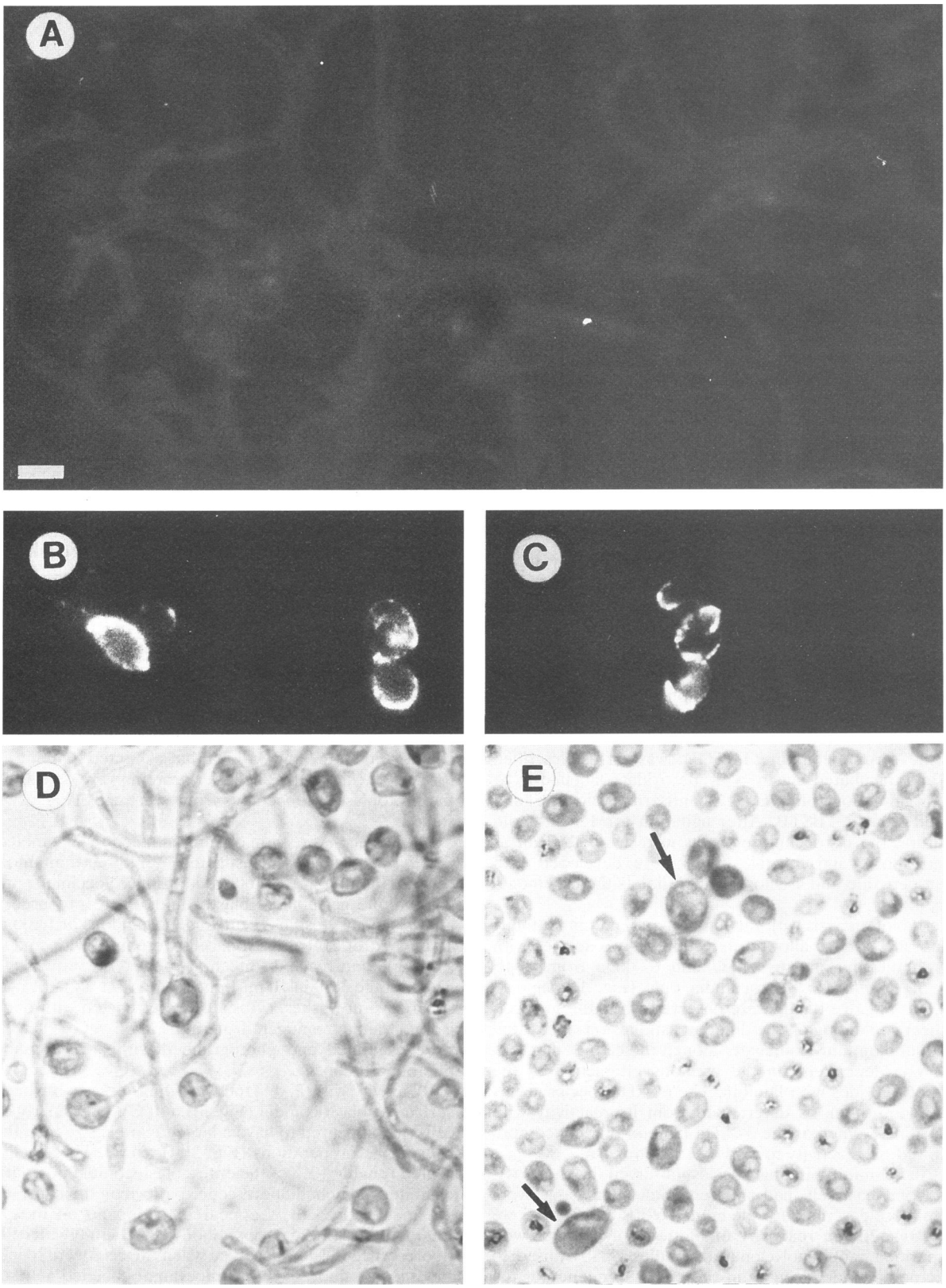

FIG. 3. Phase-contrast (D and E) and fluorescence (A through C) microscopy of cells incubated with PAb 4160 (1:50 dilution). *C. albicans* ATCC 26555 was incubated under conditions that induce germ tube formation (A and D) or budding growth (B, C, and E). In panel E, arrows point to yeast cells with a larger average size that were the only cells that exhibited fluorescent patches irregularly distributed on their surfaces. The bar marker in panel A represents 3 μm, and the magnification is the same for all panels.

PAb 4056 was used as the probe in the IIF assays (data not shown).

The candidal cell surface laminin receptor-like molecules identified in this work seem to be different from those described by Bouchara et al. (1). First, they identified laminin-binding sites on germ tubes but not in nongerminated blastoconidia, whereas the laminin receptor-like molecules reported in this article appear to be specific protein components of the walls of *C. albicans* yeast cells. In this context, our results add to other recent observations indicating that *C. albicans* blastoconidia are able to bind laminin (15). Second, while Bouchara et al. (1) identified germ tube-specific cell surface components (68, 62, and 60 kDa) with laminin-binding activity, similar in molecular mass to the 67LR molecule, these laminin receptors appear to belong to a family of *C. albicans* cell wall proteins and glycoproteins with multiple biological affinities for laminin, fibrinogen, and C3d (1, 3, 28, 32). However, the 37- and 67-kDa laminin-binding proteins described here did not bind other mammalian proteins, such as fibrinogen, fibronectin, and type IV collagen, under our experimental conditions. Also, the 37-kDa laminin-binding protein in the βME extract obtained from nongerminated blastoconidia (Fig. 1B, lane 1) as well as a 67-kDa species in the protoplast homogenates also from yeast cells (Fig. 2B, lane 3, upper star) cross-reacted with antibodies raised to different topologic and functional domains of the high-affinity human laminin receptor. This observation is not surprising, since cross-reactivity of antibodies to bacterial laminin-binding proteins with the putative laminin receptor from animal cells has already been reported (22). Also, a DNA sequence encoding a protein identical to the high-affinity human laminin receptor in *Saccharomyces cerevisiae* has recently been characterized (34).

The possibility that the results presented here reflect a particular characteristic and/or physiological response of the *C. albicans* ATCC 26555 strain to the experimental conditions used in this work cannot be dismissed. In addition, heterogeneity in expression of surface immunodeterminants not only among different *C. albicans* strains but also among cells in a population (12, 24), and phenotypic colony-type switching, a phenomenon that is thought to modify the antigenic characteristics of the cell surface (20, 29), may also account for the appearance of the high-affinity laminin receptor-like molecules in the blastoconidia of ATCC 26555. We have reported that strain ATCC 26555 exhibited spontaneous high-frequency colony phenotypic switching (20), similar to the switching system initially described by David Soll for *C. albicans* 3153A (29). In this context, cells expressing the 37-kDa laminin receptor-like species, as revealed by IIF and phase-contrast microscopy (Fig. 3, arrows), exhibited a bean-shaped appearance and a size similar to those of the opaque phenotype cells in the white-opaque transition, which is one of the three distinct switching systems described for *C. albicans* (29); such cells could represent a particular switching phenotype within the population having specific cell surface components. Interestingly, significant differences have been reported in the adhesion characteristics as well as the cell surface properties (i.e., ultrastructure and antigenicity) of the so-called white and opaque cell phenotypes of *C. albicans* (29). Any or all of the possibilities discussed above could explain the differences between the results reported by Bouchara et al. (1) and our findings.

Along with observations from other authors (1, 15, 32), our data point to the possibility that different cell surface receptors for laminin may be constitutively or selectively expressed in *C. albicans* cells. Thus, a highly conserved homolog of the human 37LRP species, which in animal cells appears to be a multifunctional protein that may play different roles in the cytoplasm as well as on the cell surface as a laminin-binding component (9), is apparently present in *C. albicans* blastoconidia.

There are differences in the epitopes present in the 37-kDa laminin receptor-like protein expressed in the two growth forms of the fungus. Thus, although this molecule appears to be present in both morphological phases, as determined by the reactivity observed with PAb 4056, which is directed towards an internal domain of human 37LRP (Fig. 2B, lanes 1 to 4), the laminin-binding activity was detected exclusively in the 37-kDa species extracted by βME from *C. albicans* yeast cells, which cross-reacted only with PAb 4160 (Fig. 2A, lane 1). The absence of laminin-binding activity in protein extracts from germinated blastoconidia under our experimental conditions may thus be explained by sequence or posttranslational modifications affecting the carboxyl-terminal domain of the 37-kDa species present in germinated yeast cells.

Although germ tubes are believed to be the invasive form (23) of *C. albicans*, the existence of laminin receptors on yeast cells may allow such cells to disseminate in vivo, resulting in metastatic sites of infection throughout the host. In any case, as the germ tubes of *C. albicans* also contain cell surface laminin-binding components (1) apparently different from the ones described in this article, the existence of distinct families of laminin receptors selectively expressed in yeast cells and germ tubes may account for the different biological behavior of the two growth forms of *C. albicans* in human tissues.

Finally, it has been reported that PepTite-2000, a synthetic peptide including the RGD (arginine-glycine-aspartic acid) sequence which is recognized by the candidal fibronectin receptor, inhibits adherence of *C. albicans* yeast cells to the extracellular matrix and binding of protein components from the latter by more than 90% (15). In this context, another synthetic peptide (peptide G) of the deduced amino acid sequence from the carboxyl-terminal domain of 37LRP, which binds laminin with high affinity and represents a putative laminin-binding site of the 67LR species (9), has been shown to be of value in inhibiting tumor cell attachment to endothelial cells, a critical step in bloodborne metastasis (10). As the ability to bind to basement membrane seems to be an important mechanism by which pathogenic microorganisms adhere to and invade the host (2, 15, 33), and taking into account that laminin is a major component of the basement membrane that plays a fundamental role in cell adhesion and extravasation (31), it is tempting to suggest that peptide G may be of therapeutic value as an inhibitor of the dissemination of fungal cells needed for the establishment of systemic candidiasis.

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REFERENCES

1. **Bouchara, J. P., G. Tronchin, V. Annaix, R. Robert, and J. M. Senet.** 1990. Laminin receptors on *Candida albicans* germ tubes. Infect. Immun. **58**:48–54.
2. **Calderone, R. A., and P. C. Braun.** 1991. Adherence and receptor relationships in *Candida albicans.* Microbiol. Rev. **55**:1–20.
3. **Calderone, R. A., L. Linehan, E. Wadsworth, and A. L. Sandberg.**

1988. Identification of C3d receptors in *Candida albicans.* Infect. Immun. **56:**252–258.
4. **Calderone, R. A., and W. M. Scheld.** 1987. Role of fibronectin in the pathogenesis of candidal infections. Rev. Infect. Dis. **9**(Suppl. 4):400–403.
5. **Casanova, M., M. L. Gil, L. Cardeñoso, J. P. Martínez, and R. Sentandreu.** 1989. Identification of wall-specific antigens synthesized during germ tube formation by *Candida albicans*. Infect. Immun. **57:**262–271.
6. **Casanova, M., J. L. López-Ribot, J. P. Martínez, and R. Sentandreu.** 1992. Characterization of cell wall proteins from yeast and mycelial cells of *Candida albicans* by labelling with biotin: comparison with other techniques. Infect. Immun. **60:**4898–4906.
7. **Casanova, M., J. L. López-Ribot, C. Monteagudo, A. Llombart-Bosch, R. Sentandreu, and J. P. Martínez.** 1992. Identification of a 58-kilodalton cell surface fibrinogen-binding mannoprotein from *Candida albicans*. Infect. Immun. **60:**4221–4229.
8. **Castronovo, V., C. Colin, A. P. Claysmith, P. H. S. Chen, E. Lifrange, R. Lambotte, H. Krutzsch, and M. E. Sobel.** 1990. Immunodetection of the metastasis-associated laminin receptor in human breast cancer cells obtained by fine-needle aspiration biopsy. Am. J. Pathol. **137:**1373–1381.
9. **Castronovo, V., G. Taraboletti, and M. E. Sobel.** 1991. Functional domains of the 67-kDa laminin receptor precursor. J. Biol. Chem. **266:**20440–20446.
10. **Castronovo, V., G. Taraboletti, and M. E. Sobel.** 1991. Laminin receptor complementary DNA-deduced synthetic peptide inhibits cancer cell attachment to endothelium. Cancer Res. **51:**5672–5678.
11. **Dubois, M., K. A. Gilles, J. K. Hamilton, P. A. Rebers, and F. Smith.** 1956. Colorimetric method for determination of sugars and related substances. Anal. Chem. **28:**350–356.
12. **Gil, M. L., M. Casanova, J. P. Martínez, and R. Sentandreu.** 1991. Antigenic cell wall mannoproteins in *Candida albicans* isolates and in other *Candida* species. J. Gen. Microbiol. **137:**1053–1061.
13. **Hostetter, M. K., J. S. Lorenz, L. Preus, and K. E. Kendrick.** 1990. The iC3b receptor on *Candida albicans*: subcellular localization and modulation of receptor expression by glucose. J. Infect. Dis. **161:**761–768.
14. **Klotz, S. A., and R. L. Smith.** 1991. A fibronectin receptor on *Candida albicans* mediates adherence of the fungus to extracellular matrix. J. Infect. Dis. **163:**604–610.
15. **Klotz, S. A., R. L. Smith, and B. W. Stewart.** 1992. Effect of an arginine-glycine-aspartic acid-containing peptide on hematogenous candidal infections in rabbits. Antimicrob. Agents Chemother. **36:**132–136.
16. **Lee, K. L., M. R. Buckley, and C. Campbell.** 1975. An amino acid liquid synthetic medium for development of mycelial and yeast forms of *Candida albicans*. Sabouraudia **13:**148–153.
17. **Lopes, J. D., M. Dos Reis, and R. R. Brentani.** 1985. Presence of laminin receptors in *Staphylococcus aureus*. Science **229:**275–277.
18. **López-Ribot, J. L., M. Casanova, J. P. Martínez, and R. Sentandreu.** 1991. Characterization of cell wall proteins of yeast and hydrophobic mycelial cells of *Candida albicans*. Infect. Immun. **59:**2324–2332.
19. **Lowry, O. H., N. J. Rosebrough, A. L. Farr, and R. J. Randall.** 1951. Protein measurement with the Folin phenol reagent. J. Biol. Chem. **193:**265–275.
20. **Martínez, J. P., M. L. Gil, M. Casanova, J. L. López-Ribot, J. García de Lomas, and R. Sentandreu.** 1990. Wall mannoproteins in cells from colonial phenotypic variants of *Candida albicans*. J. Gen. Microbiol. **136:**2421–2432.
21. **McDonald, J. A.** 1989. Receptors for extracellular matrix components. Am. J. Physiol. **257:**L331–L337.
22. **Motta, G. F., C. R. W. Carneiro, L. Gomes, and J. D. Lopes.** 1988. Monoclonal antibodies to *Staphylococcus aureus* laminin-binding proteins cross-react with mammalian cells. Infect. Immun. **56:** 1580–1584.
23. **Odds, F. C.** 1988. *Candida* and candidosis: a review and bibliography, p. 42–59. Bailliere Tindall, London.
24. **Poulain, D., V. Hopwood, and A. Vernes.** 1985. Antigenic variability of *Candida albicans*. Crit. Rev. Microbiol. **12:**223–270.
25. **Rao, C. N., S. H. Barsky, V. P. Terranova, and L. A. Liotta.** 1983. Isolation of a tumor cell laminin receptor. Biochem. Biophys. Res. Commun. **111:**804–808.
26. **Rao, C. N., V. Castronovo, M. C. Schmitt, U. M. Wewer, A. P. Claysmith, L. A. Liotta, and M. E. Sobel.** 1989. Evidence for a precursor of the high-affinity metastasis-associated murine laminin receptor. Biochemistry **28:**7476–7486.
27. **Ruoslahti, E.** 1991. Integrins. J. Clin. Invest. **87:**1–5.
28. **Saxena, A., and R. A. Calderone.** 1990. Purification and characterization of the extracellular C3d-binding protein of *Candida albicans*. Infect. Immun. **58:**309–314.
29. **Soll, D. R.** 1992. High-frequency switching in *Candida albicans*. Clin. Microbiol. Rev. **5:**183–203.
30. **Switalski, L. M., H. Murchinson, R. Timpl, R. Curtiss III, and M. Hook.** 1987. Binding of laminin to oral and endocarditis strains of viridans streptococci. J. Bacteriol. **169:**1095–1101.
31. **Timpl, R., H. Rohde, P. Gehron-Robey, S. I. Rennard, J. M. Foidart, and G. Martin.** 1979. Laminin—a glycoprotein from basement membranes. J. Biol. Chem. **254:**9933–9937.
32. **Tronchin, G., R. Robert, A. Bouali, and J. M. Senet.** 1987. Immunocytochemical localization of *in vitro* binding of human fibrinogen to *Candida albicans* germ tube and mycelium. Ann. Inst. Pasteur Microbiol. **138:**177–187.
33. **Valkonen, H. K., J. Veijola, B. Dagberg, and B. E. Uhlin.** 1991. Binding of basement-membrane laminin by *Escherichia coli*. Mol. Microbiol. **5:**2133–2141.
34. **Weinstock, K. G., M. D. Adams, C. Fields, and J. C. Venter.** 1993. Expressed sequence tags from *Saccharomyces cerevisiae*, abstr. B9, p. 23. ASM Conference on *Candida* and Candidiasis: Biology, Pathogenesis and Management, Baltimore, Md. American Society for Microbiology, Washington, D.C.
35. **Wewer, U. M., L. A. Liotta, M. Jaye, G. A. Ricca, W. N. Drohan, A. P. Claysmith, C. N. Rao, P. Wirth, J. E. Coligan, R. Albrechtsen, M. Mudryj, and M. E. Sobel.** 1986. Altered levels of laminin receptor mRNA in various human carcinoma cells that have different abilities to bind laminin. Proc. Natl. Acad. Sci. USA **83:**7137–7141.