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Adherence of Human Vaginal Lactobacilli to Vaginal Epithelial Cells and Interaction with Uropathogens

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Three strains of *Lactobacillus*, identified as *Lactobacillus acidophilus*, *Lactobacillus gasseri*, and *Lactobacillus jensenii*, were selected from among 70 isolates from the vaginas of healthy premenopausal women for properties relevant to mucosal colonization or antagonism. All three self-aggregated and adhered to epithelial vaginal cells, displacing well-known vaginal pathogens, such as *G. vaginalis*, and inhibiting the growth in vitro of *Escherichia coli* and *Streptococcus agalactiae*. The surface components involved in self-aggregation appeared to be proteins for *L. gasseri* and lipoproteins for *L. acidophilus* and *L. jensenii*, as judged by susceptibility to treatment with appropriate degrading enzymes. The factors responsible for adherence to epithelial vaginal cells seemed to be glycoproteins (*L. acidophilus* and *L. gasseri*) and carbohydrate (*L. jensenii*). The receptors of the vaginal cells were glycolipids, which presumably were the targets of the competition observed between the lactobacilli and the pathogenic microbes.

The vaginal ecosystem harbors a microbiota that is being increasingly recognized as protecting it from invading pathogens, including those that cause urinary tract infections and sexually transmitted diseases. Lactobacilli are dominant in this habitat, at 10^7 to 10^8 CFU/g of vaginal fluid in healthy premenopausal women (18). Among them, those belonging to the *Lactobacillus acidophilus* group and *L. fermentum* are most frequently isolated, although others, such as *L. plantarum*, *L. brevis*, *L. jensenii*, *L. casei*, *L. delbrueckii*, and *L. salivarius*, are isolated as well (14).

Lactobacilli are believed to interfere with pathogens by different mechanisms. The first is competitive exclusion of genitourinary pathogens from receptors present on the surface of the genitourinary epithelium (5, 21). Second, lactobacilli coaggregate with some uropathogenic bacteria (14), a process that, when linked to the production of antimicrobial compounds, such as lactic acid, hydrogen peroxide, bacteriocin-like substances (12, 15), and possibly biosurfactants (21), would result in inhibition of the growth of the pathogen.

Adherence of bacteria to epithelial cells has been shown to be an important factor in the colonization of mucous membranes. However, little is known about the mechanisms by which lactobacilli from the vaginas of healthy young women adhere to vaginal epithelial cells, although the variety of surface structures in these bacteria implies that a spectrum of adherence mechanisms may exist. Furthermore, self-aggregation may substantially increase the colonization potential of lactobacilli in environments with short residence times.

In this study, we report on the mechanisms of self-aggregation and adherence to epithelial vaginal cells of three vaginal *Lactobacillus* isolates. Furthermore, we analyzed how these properties may interfere with pathogenic colonization, both through cell surface receptor competition and growth inhibition.

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MATERIALS AND METHODS

Strains and culture conditions. Lactobacilli were incubated on LAPTg agar or broth (13) at 37°C. Initial isolations from vaginal samples were done under a 5% CO_2 atmosphere. The isolates were subsequently incubated by aerobiosis. *Gardnerella vaginalis* was grown in brain heart infusion (Biokar) under a 5% CO_2 atmosphere, *Escherichia coli* was propagated in eosin-methylene blue (Pronadisa), and *Streptococcus agalactiae* and *Candida albicans* were grown in LAPTg at 37°C by aerobiosis. All strains were ambulatory or clinical specimens obtained at the Hospital Monte Naranco.

Aggregation tests. Determination of the self-aggregation ability of lactobacilli and biochemical treatments of the cells to determine the nature of the aggregation factor(s) were performed as described previously (3).

Electron microscopy. Lactobacilli from overnight cultures in LAPTg broth were washed with distilled water and resuspended in the liquid that remained in the pellets, and 5-μl aliquots were allowed to stand on copper grids coated with Formvar (Merck). The excess liquid was removed, 5 μl of 2% (wt/vol) uranyl acetate solution was added, and the mixture was allowed to stand for 2 min. The negatively stained cells were examined in a JEOL 2000 EXII transmission electron microscope at 120 kV.

Hydrophobicity determination. The surface hydrophobicity of the lactobacilli was determined by measuring the affinity of cells cultured overnight for xylene in a two-phase system (water-xylene) (17).

Adherence assays. Vaginal epithelial cells were collected from healthy premenopausal women and treated as described previously (23). Overnight cultures of the lactobacilli to be tested were suspended to 10^8 cells/ml in Eagle's minimal essential medium (Flow Laboratories). Equal volumes of the bacterial suspensions and of vaginal cells were mixed and incubated at 37°C with orbital shaking (100 rpm/min) for 30 min. Afterward, the suspensions were passed through 8-μm-pore-size Millipore filters and washed with 1 volume of Eagle's medium. The cells retained on the filter were placed on albumin-coated microscope slides, fixed with ethanol, and Gram stained. The assays were started within 1 h of the collection of the epithelial cells, and each determination was performed in duplicate. As a negative control for adherence, *L. plantarum* LL 441 isolated from cheese whey was used (10).

The nature of the bacterial and eukaryotic factors involved in adherence was determined through treatment of the cells with proteinase K, lipase, and sodium metaperiodate as described before (2, 20). The sensitivity of adherence to temperature was assayed by heating lactobacillus suspensions to 100°C for 10 min in phosphate-buffered saline. The reversibility of adherence was tested by repeatedly washing the mixed lactobacilli and epithelial cells with 20 mM EDTA or EGTA.

Interference assays. Interference experiments were performed with *G. vaginalis* and *C. albicans*, since they were the only potential genitourinary pathogens used in this work that showed a significant capacity to adhere to vaginal cells (see below). The procedures described by Spencer and Chesson (19) were used, with some modifications. For exclusion tests, lactobacilli and vaginal epithelial cells were incubated together for 30 min; afterward, *C. albicans* or *G. vaginalis* cells were added, and incubation was continued for a further 30 min. For competition tests, lactobacilli, any of the pathogens, and vaginal epithelial cells were mixed

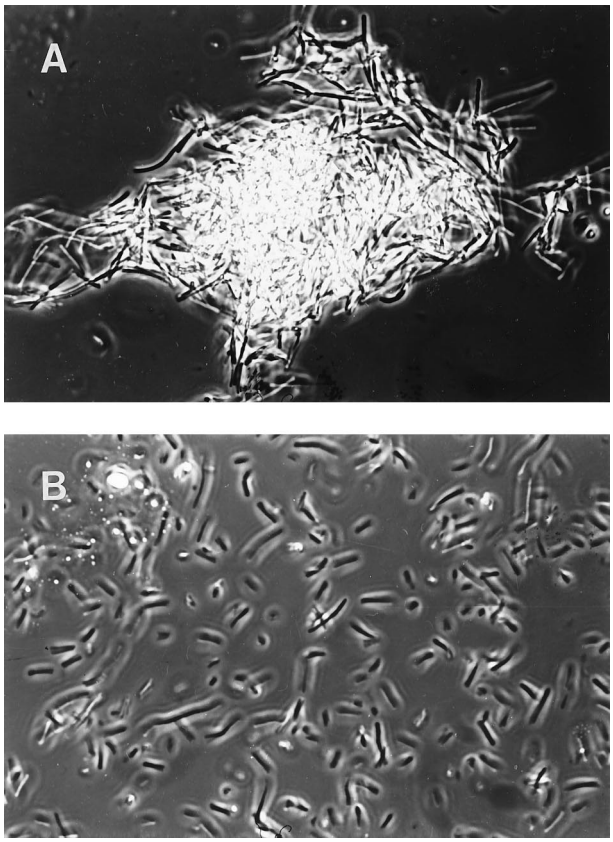

FIG. 1. Microscopic observations of autoaggregating *L. acidophilus*. (A) Control. (B) Cells treated with proteinase K or lipase.

and incubated for 30 min. For displacement tests, *C. albicans* or *G. vaginalis* and vaginal epithelial cells were incubated together for 30 min, lactobacilli were added, and incubation was continued for a further 30 min. The resulting suspensions were filtered, and cell observation was performed as indicated above.

Coaggregation assays. Coaggregation assays were designed based on previously reported methods (16). Microorganism suspensions were adjusted to an A_{600} of 0.6. Aliquots of 500 μl of the three *Lactobacillus* strains were mixed with 500 μl of each of the four pathogens and incubated at 37°C in an orbital shaker at 100 rpm for 4 h. The suspensions were then macroscopically scored for coaggregation according to a scale described elsewhere (16). In addition, they were observed under a phase-contrast microscope after Gram staining.

Statistical analysis. All measurements were made with a minimum of duplicate samples per variable for each experiment. Data are expressed as mean ± standard deviation for representative experiments. Comparisons were analyzed by Student's *t* test.

RESULTS

Selection of adherent lactobacilli. Vaginal exudates were swabbed onto selective media for sexually transmissible pathogens and on chocolate agar. Incubation was done for 72 h at 37°C under a 5 to 10% CO_2 atmosphere with daily inspections for growth. From the first series of media, the potential genitourinary pathogens indicated in the Materials and Methods section were obtained. From the chocolate agar plates, white colonies, consisting of gram-positive bacilli unable to grow under complete aerobiosis, were isolated, restreaked onto LAPTg agar, and incubated under the same conditions. In this way, 70 vaginal *Lactobacillus* isolates were obtained. Three of them were selected for their autoaggregating ability and adherence to vaginal epithelial cells. They were classified as *L. acidophilus*, *L. gasseri*, and *L. jensenii* with the API 50 CHL system (BioMerieux).

Aggregation studies. All three strains self-aggregated, producing macroscopic granules; the effect was also observed under the light microscope (Fig. 1A). Aggregation was abolished by treatment of the cells with proteinase K and, for *L. acidophilus* and *L. jensenii*, also after incubation with lipase (Fig. 1B). This result indicates that the aggregation-promoting factor is a protein for *L. gasseri* and a lipoprotein for the other organisms (or separated lipids and proteins, both of which would be necessary for aggregation to occur). Since no effect was seen after phenol extraction or sodium metaperiodate treatment of the cells, lipoteichoic acids or carbohydrates are probably not involved in the aggregation of these strains (8).

Irrespective of the nature of the factors, all three strains showed extremely high surface hydrophobicity, with values in excess of 80% of the cells migrating into the xylene phase in the two-phase Rosenberg test (17); nonadherent lactobacilli were much more hydrophilic, showing values of 40% or less.

The aggregation-promoting molecules seemed to form a

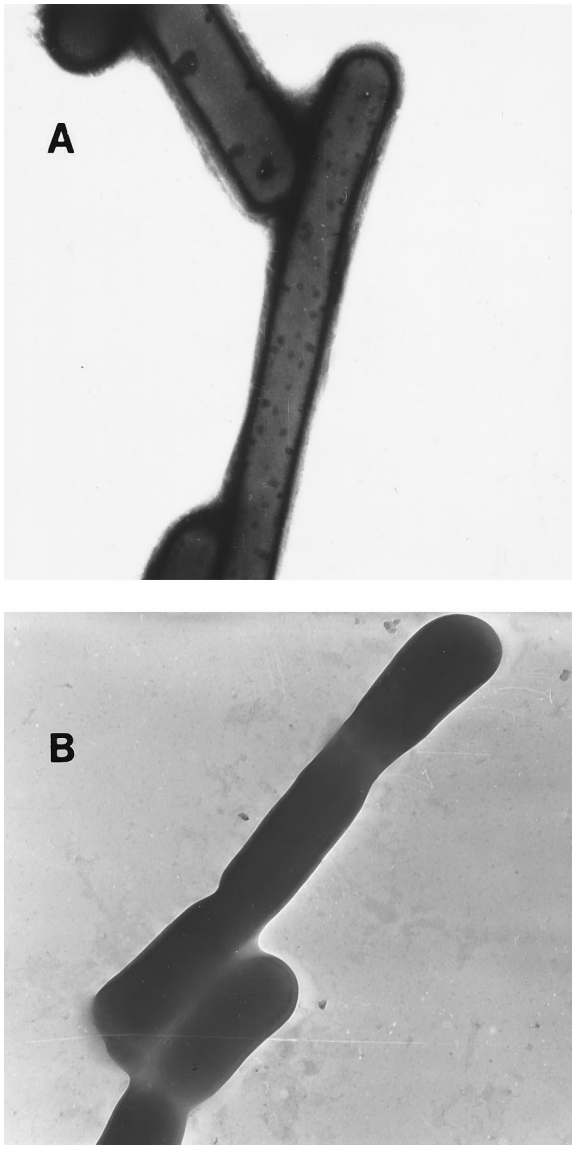

FIG. 2. Electron micrographs of negatively stained *L. acidophilus*. (A) Control. (B) Cells treated with proteinase K or lipase.

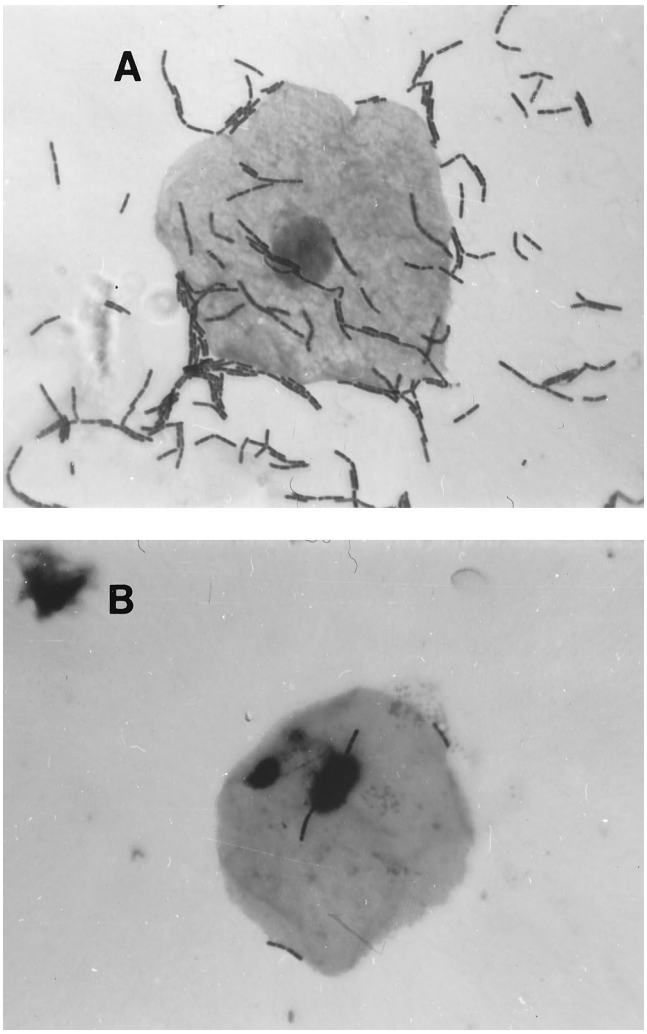

FIG. 3. Adherence of lactobacilli to vaginal epithelial cells. (A) Gram-stained preparation of adherent *L. acidophilus* isolated from human vaginal cells. (B) As a negative control, *L. plantarum* LL 441, isolated from dairy products, is shown.

continuous layer around the cells, as observed by negative staining before (Fig. 2A) and after (Fig. 2B) treatment with proteinase K or lipase.

Adherence to vaginal epithelial cells. The three human *Lactobacillus* strains were adherent, while *L. plantarum* LL 441, a strain isolated from dairy products and used as a negative control, was not (Fig. 3). The adherence ability was lost upon treatment of the cells with sodium metaperiodate and, for *L. acidophilus* and *L. gasseri*, with proteinase K. However, lipase did not exert any significant effect on adherence. Finally, it seemed that cations were necessary for *L. gasseri* and *L. jensenii* adherence, while *L. acidophilus* adherence was heat sensitive (Table 1). These data indicate that *L. acidophilus* receptors are heat-sensitive glycoproteins, those of *L. gasseri* are cation-requiring glycoproteins, and those of *L. jensenii* are cation-requiring carbohydrates.

The receptors of the vaginal epithelial cells for the three *Lactobacillus* strains appeared to be glycolipids, as deduced by the large decrease in the adherence capacity resulting from treatment of the vaginal cells with lipase and metaperiodate (Table 2).

TABLE 1. Ability of *Lactobacillus* isolates to adhere to vaginal epithelial cells

Treatment	No. of adherent cells (mean ± SD) of:		
	L. acidophilus	*L. gasseri*	*L. jensenii*
None (control)	46.1 ± 13.7	38.6 ± 15.4	24.3 ± 12.5
Proteinase K (1 mg/ml)	2.6 ± 2.3[a]	2.3 ± 2.1[b]	20.5 ± 6.9[c]
Lipase (1 mg/ml)	45.9 ± 13.4[a]	33.2 ± 14.2[b]	24.9 ± 12.4[c]
Sodium metaperiodate (10 mg/ml)	1.47 ± 1.44[a]	2.3 ± 2.2[b]	4.5 ± 4.1[c]
Heat (100°C)	2.81 ± 2.7[a]	32.2 ± 17.8[b]	20.6 ± 9.5[c]
EDTA (20 mM)	45.3 ± 22.02[a]	4.3 ± 3.03[b]	3.5 ± 3.2[c]
EGTA (20 mM)	42.5 ± 20.2[a]	2.9 ± 2.5[b]	3.9 ± 2.05[c]

[a] $P < 0.05$ compared with the *L. acidophilus* control.
[b] $P < 0.001$ compared with the *L. gasseri* control.
[c] $P < 0.05$ compared with the *L. jensenii* control.

Adhesion interference of urogenital pathogens. Strains of four potential genitourinary pathogens were obtained from the same vaginal exudates that rendered the lactobacilli used in this work. In this way, we attempted to compare the behavior of strains that presumptively were competing for the same biotope. No adhesion to epithelial vaginal cells was observed for *E. coli* and *S. agalactiae*. However, *C. albicans* and *G. vaginalis* were adherent. The adhesion interference experiments were then restricted to strains of these two species, with the competing strain being *L. acidophilus*. A large reduction in adherence was observed when *C. albicans* cells were added together with *L. acidophilus* cells to the vaginal cells. The same was found for *G. vaginalis* which, in addition, was displaced by the lactobacilli when attached to the vaginal cells (Table 3).

Coaggregation experiments. Mixed cultures of all three *Lactobacillus* strains with any of the four potential pathogens showed coaggregation with *E. coli*, *C. albicans*, and *G. vaginalis* but not with *S. agalactiae* (Table 4). As an example, Fig. 4A shows the microscopic appearance of the aggregates formed between *C. albicans* and *L. acidophilus*, which contrasts with the presence of isolated *S. agalactiae* cells surrounding aggregates of the same lactobacilli (Fig. 4B).

DISCUSSION

In recent years, there has been an increasing recognition of the role of lactobacilli in the maintenance of the homeostasis within dynamic ecosystems such as the vagina and in the prevention of colonization and infection caused by pathogenic organisms (11).

Lactobacilli are important components of the normal vaginal microbiota. They help to repel invading pathogens and may also prevent urinary tract infections by interfering with the

TABLE 2. Nature of the receptors of the vaginal epithelial cells for the *Lactobacillus* strains

Vaginal cell treatment	No. of adherent cells (mean ± SD) of:		
	L. acidophilus	*L. gasseri*	*L. jensenii*
None (control)	46.1 ± 13.7	38.6 ± 15.4	24.3 ± 12.5
Proteinase K (1 mg/ml)	39.3 ± 15.05[a]	24.8 ± 13.6[b]	30.7 ± 9.4[c]
Lipase (1 mg/ml)	1.6 ± 1.4[a]	5.9 ± 5.5[b]	5 ± 1.7[c]
Sodium metaperiodate (10 mg/ml)	1.1 ± 0.9[a]	10.7 ± 6.7[b]	2.9 ± 2.5[c]

[a] $P < 0.01$ compared with the *L. acidophilus* control.
[b] $P < 0.001$ compared with the *L. gasseri* control.
[c] $P < 0.05$ compared with the *L. jensenii* control.

TABLE 3. Effect of *L. acidophilus* on the attachment of *C. albicans* and *G. vaginalis* to vaginal epithelial cells under conditions of exclusion, competition, and displacement

Strain	No. of adherent cells (mean ± SD) under the following conditions:			
	Control	Competition	Exclusion	Displacement
C. albicans	22.5 ± 5	5.5 ± 2.8[a]	23 ± 10.6[a]	18.2 ± 7.4[a]
G. vaginalis	34.5 ± 9.8	8 ± 3.1[b]	37.5 ± 6.5[b]	5.3 ± 2.8[b]

[a] $P < 0.05$ compared with the *C. albicans* control.
[b] $P < 0.05$ compared with the *G. vaginalis* control.

colonization of the periurethral epithelium by uropathogens such as *E. coli*. Thus, a loss of vaginal lactobacilli may predispose women to the acquisition of genitourinary infections.

For this reason, the prophylactic use of selected *Lactobacillus* strains may be an effective means of restoring the normal microbial flora in the vagina (9, 11), thus preventing infections. The characteristics needed for a *Lactobacillus* strain to serve effectively as a prophylactic agent include avid adherence to vaginal epithelial cells, interference with the adherence of other pathogens, and the production of H_2O_2 and/or other molecules capable of inhibiting the growth of pathogens (1).

At present, the molecular mechanisms by which lactobacilli adhere to epithelial cells remain unknown. Several studies have suggested that *Lactobacillus* adherence is mediated by proteins (6, 8, 22), while others have suggested a role for lipoteichoic acid (5) and carbohydrate (4, 7).

The three vaginal isolates selected in this work were able to self-aggregate in a process mediated by surface proteins or lipoproteins. In addition, the three strains strongly adhered to vaginal epithelial cells, whereas lactobacilli recovered from other sources, such as dairy products, adhered in significantly lower numbers, indicating that adherence is an idiosyncratic property of vaginal lactobacilli.

Both self-aggregation and adhesion may favor the colonization of the vaginal epithelium through the formation of a bacterial film that may contribute to the exclusion of pathogens from the vaginal mucosa.

Multiple components of the bacterial cell surface seem to participate in the adherence of the strains to vaginal epithelial cells. In *L. acidophilus* and *L. gasseri*, adherence involved proteins and carbohydrate (possibly a glycoprotein), while *L. jensenii* adherence seemed to depend exclusively on carbohydrates. In *L. gasseri* and *L. jensenii*, divalent cations, probably Ca^{2+}, were also involved in adherence, as judged by sensitivity to EDTA and EGTA. This diversity of adherence requirements was reported before, although for digestive epithelium. Thus, *L. fermentum* adherence to mouse squamous epithelium was sensitive to chelating agents (6), while colonization of chicken tissue by *L. acidophilus* was not (7). However, the adherence factors seem to be different from those that mediate the self-aggregation of the strains; for example, *L. jensenii* self-aggregation depends on lipoproteins, while adherence to vaginal cells relies on carbohydrates.

TABLE 4. Coaggregation between *L. acidophilus* and some genitourinary tract pathogens

Strain	Coaggregation score for:			
	C. albicans	*E. coli*	*S. agalactiae*	*G. vaginalis*
L. acidophilus	3	3	0	3
L. gasseri	4	4	0	4
L. jensenii	2	2	0	2

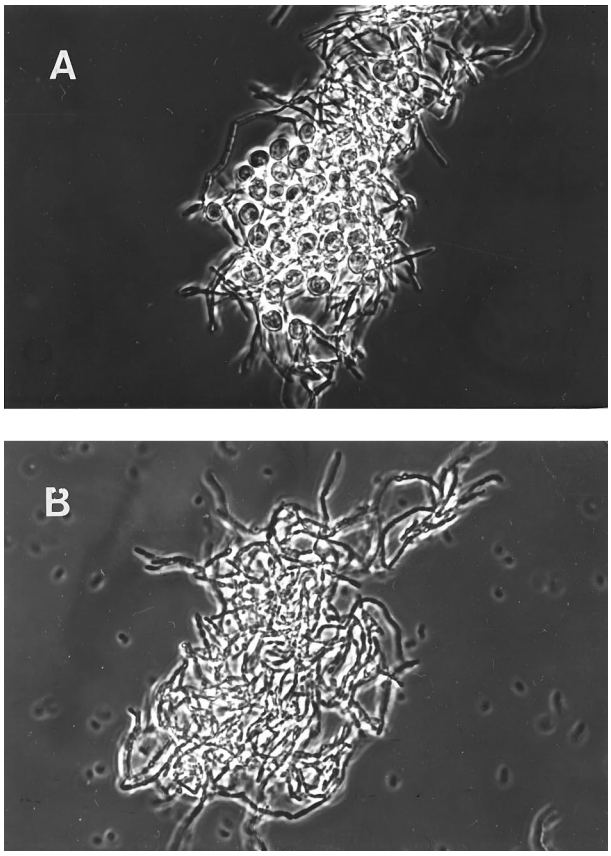

FIG. 4. Microscopic observations of coaggregation between *L. acidophilus* and *C. albicans* (A) and the lack of coaggregation between *L. acidophilus* and *S. agalactiae* (B).

The vaginal lactobacilli interfered with the adherence of genitourinary pathogens. In this respect, it is interesting to note first that *C. albicans* and *G. vaginalis* adhered to vaginal epithelial cells, while *E. coli* and *S. agalactiae* did not. Since the first two organisms produce pathology primarily at the vaginal level, while the others are just opportunistic pathogens, it may be deduced that adherence is an important virulence factor.

It is possible that *L. acidophilus* and *G. vaginalis* bind to the same receptors on the surfaces of vaginal epithelial cells. It appears that the affinity of *L. acidophilus* for those receptors is higher than that of *G. vaginalis*, as deduced by the displacement by *L. acidophilus* of adherent cells of *G. vaginalis.*

Finally, the vaginal lactobacilli coaggregated with all of the pathogens, with the exception of *S. agalactiae*, suggesting that this process is somewhat specific. The coaggregation may well impede the access of pathogens to tissue receptors and in fact may be an alternative explanation for the lack of adherence of *C. albicans* and *G. vaginalis* to vaginal epithelial cells in the presence of lactobacilli.

In conclusion, the lactobacilli used in this study may protect the vaginal epithelium through a series of barrier (self-aggregation, adherence) and interference (receptor binding interference, coaggregation with potential pathogens) mechanisms. Consequently, they may be excellent candidates for eventual use as prophylactic agents. Studies to further evaluate their feasibility as such are under way.

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