Blocking of *Pseudomonas aeruginosa* and *Ralstonia solanacearum* Lectins by Plant and Microbial Branched Polysaccharides Used as Food Additives

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Pseudomonas aeruginosa antibiotic resistance prompted the search for glycodecoys that would block its lectin-dependent adhesion to human cells. We have used the lectins of this pathogen, PA-IL (galactophilic LecA) and PA-IIL (fucophilic LecB), and two additional pathogenic bacterial lectins, CV-IIL (fucophilic, of *Chromobacterium violaceum*) and RS-IIL (mannophilic, of *Ralstonia solanacearum*), for assaying the pathogenic lectin-blocking abilities of some plant and microbial polysaccharidic food additives, adding the mannophilic plant lectin Con A as a reference. Locust-bean and guar galactomannans and acacia gum very strongly inhibited PA-IL. The other lectins, excluding CV-IIL, were very strongly inhibited by yeast mannan. Xanthan and inulin were weak inhibitors. The differential blocking of these lectins by galactosylated branches of plant polysaccharides and by mannan matched their inhibition by avian egg whites, human milk, and royal jelly (protecting animal embryos and neonates from infections). The nondigestability and nontoxicity of the food additives are advantageous for curing gastrointestinal and external infections.

KEYWORDS: Antiadhesion polysaccharides; bacterial lectins; *Chromobacterium violaceum*; food additives; *Pseudomonas aeruginosa*; *Ralstonia solanacearum*

INTRODUCTION

The worldwide-distributed *Pseudomonas aeruginosa* (*P. aeruginosa*) and the tropical and subtropical *Chromobacterium violaceum* (*C. violaceum*) and *Ralstonia solanacearum* (*R. solanacearum*) are soil saprophytic bacteria that are occasionally transformed into dangerous, opportunistic, aggressive pathogens. The first two are mainly animal (including human) pathogens, and the third is a plant pathogen. Among their virulence factors, they produce adhesins, including hemagglutinating carbohydrate-specific lectins (*1*−*3*), enabling their autoaggregation to biofilm formation and adhesion to heterologous host cells that bear the competent saccharides on their surface receptors (*4*). The lectin-mediated bacterial anchoring to the target cell surfaces enables maximal cell destruction by the bacterial virulence factors, whose production is coregulated with that of the lectins involving "quorum-sensing" signals (*4*).

P. aeruginosa produces galactophilic PA-IL (LecA) and fucophilic (mannophilic) PA-IIL (LecB) lectins (*1*). Their levels are highest in the most virulent strains (*4*), and their binding profiles to animal tissues are in accord with the intact bacterium preferential cell adhesion profiles (*4*,*5*). The lectins themselves affect the target cells, but their cofunction with the other virulence factors causes the major infection-associated host cell damage (*4*).

Both *R. solanacearum* and *C. violaceum* produce lectins that are structurally homologous to PA-IIL: *R. solanacearum* second lectin (RS-IIL) [mannophilic (fucophilic)] (*2*) and *C. violaceum*

lectin (CV-IIL) [fucophilic (mannophilic)] (*3*) (**Table 1**). Although CV-IIL resembles PA-IIL in being more fucophilic than mannophilic, it differs from it in H antigen (Fucα1-2Gal-) preference, while PA-IIL prefers the Le^a epitope [Fucα1-4(Galβ1-3)GlcNAcβ1-3Galβ1-4Glc] (*6*, *7*). RS-IIL, like the mannophilic plant lectin concanavalin A (Con A), does not differentiate between these two antigens (*2*). These specificity differences might contribute to *P. aeruginosa* and *C. violaceum* involvement in nosocomial human infections, endangering the lives of patients who suffer from cystic fibrosis, burns, and immunodeficiency, while *R. solanacearum* is mainly an aggressive phytopathogen that causes great agricultural losses by wilting crops of over 200 diverse plant species (*8*).

The information on the human cell receptors that bind the two *P. aeruginosa* lectins (*4*, *9*) and the discovery of these lectin genes (*10*) enabled insight into the molecular basis of the lectin crystal three-dimensional (3D) structure interactions with their ligands (*5*, *6*, *11*). Furthermore, the growing antibiotic resistance of *P. aeruginosa* aggressive infections, combined with a shortage of new effective drugs, provoked efforts to find new strategies to abrogate *P. aeruginosa* lectin-dependent adhesion. This goal could be achieved by three main manipulations: vaccination, prevention of lectin production, and competitive blocking of the lectin binding to target cell surface receptors by "receptor-mimicking" glycodecoys (RMGDs).

The latter strategy has diverged into three streams: (a) Basic research of the detailed interactions of the bacterial lectins with well-defined natural complex glycosylated compounds (*12*), occasionally including blood group-specific ligands. This approach

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Table 1. Simplified Comparison of the Basic Specificities of the Examined Lectins *P. aeruginosa* PA-IL and PA-IIL, *C. violaceum* CV-IIL, *R. solanacearum* RS-IIL, and Con A to the Monosaccharides L-Fucose (Fuc), D-Mannose (Man) and D-Fructose (Fru), D-Glucose (Glc), D-Galactose (Gal), and Human Milk GPs (HMGPs) Graded on a Relative Scale of − to +++++

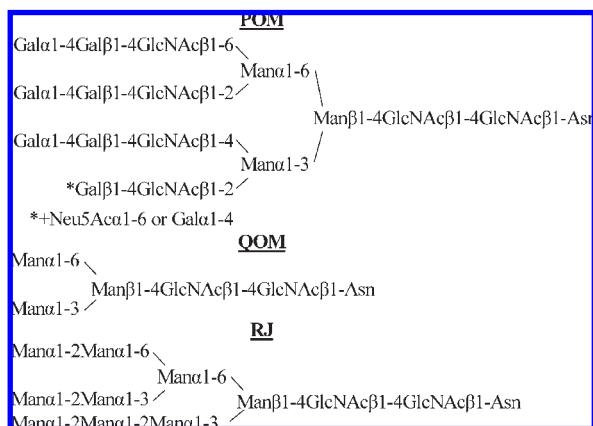| lectin/sugar	Fuc	Man and Fru	Glc	Gal	HMGPs
PA-IL	−	−	−	++++	+++
PA-IIL	+++++	++	−	−	+++++
CV-IIL	+++++	++	−	−	+++
RS-IIL	+++	+++++	−	−	++
Con A	−	++++	+++	−	++

Figure 1. Simplified oligosaccharide structures of the avian egg white ovomucoids (which constitute around 55% of each egg white protein) of pigeon [POM (*16*)] and quail [QOM (*17*)] and of the major RJ GPs (*19*).

Figure 2. Simplified structures of locust-bean and guar (*28*), inulin (*30, 31*), mannan (*33, 34*), and xanthan (*32*) polysaccharides.

has led to disclosure of PA-IL preference order Galα1-6- > Galα1-4- > Galα1-3- derivatives, which are present in the disaccharide melibiose, trisaccharide raffinose, and tetrasaccharide stachyose (*12*), and of PA-IIL highest affinity to oligomeric Le[a] (*6, 7, 13*). (b) Discovery of natural RMGDs that protect sensitive plants and animals, especially their embryos and neonates (newly born or hatched), against infections. Human milk glycans, which protect newborns from bacterial infections by preventing homing of bacteria and bacterial toxins onto their target cells (*14*), block PA-IL by its galactosylated components and PA-IIL by Le[a]-bearing oligomers and glycoproteins (GPs) (*15*). Pigeon egg white, which was shown to protect mice against infections by P-fimbriated uropathogenic *Escherichia coli* [due to its galactosylated GPs displaying blood group P[K] and P[1] epitopes (**Figure 1**) (*16*)], also strongly block PA-IL (*17*). The highly mannosylated GPs of both quail egg white (**Figure 1**) and beehive products (**Figure 1**) [royal jelly (RJ) and honey, which are used for human infection therapy (*18*)], also strongly block PA-IIL (*17, 19*). Similarly, plant seed Galα1-6-bearing galactomannans (**Figure 2**) neutralize PA-IL (*20*). The herein-described natural RMGDs were detected by PA-IL and PA-IIL using hemagglutination inhibition and Western blot assays (*15, 17, 19*). Despite the saccharide diversity of these RMGDs, they similarly inhibited each lectin interaction with various cell surface receptors due to cross-interactions with its binding site. These bacterial lectins have been recently recommended for usage as nonglycosylated recombinant lectins in microarrays (*21*). (c) Screening of thousands of synthetic multivalent lectin-blocking polyglycodendrimers bearing galactosides, mannosides, and fucosides linked to multimeric peptides, saccharides, or other scaffolds varying in branching length and multivalency. Most of them were tailored by wide-scale organic chemical manipulations according to combinatory
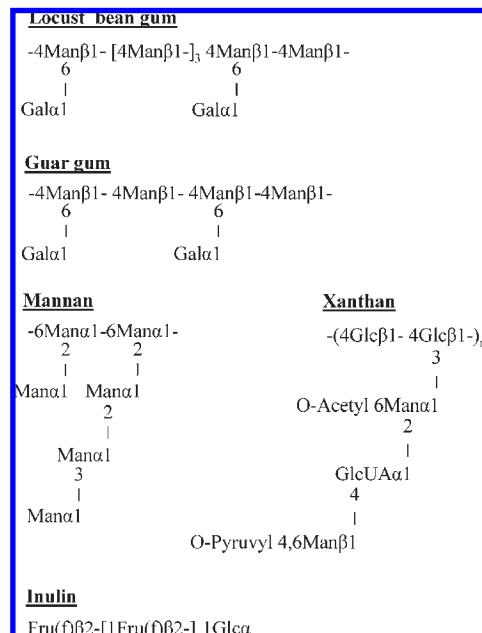
computer modeling (*22−27*). Their investigations demonstrated the high (subnanomolar) affinities of α-mannosylated multiantennary clusters to uropathogenic *E. coli* K12 type 1 (FimH) adhesins (*27*) and of fucosyl-peptide dendrimers to PA-IIL (*23−26*). Imberty et al. (*22*) have recently reviewed that subject, providing the crystal 3D structural basis for the high affinities of synthetic glycodendrimers to microbial adhesins.

The goal of the present research was to assay the blocking of the above-described *P. aeruginosa*, *C. violaceum*, and *R. solanacearum* lectins, PA-IL, PA-IIL, CV-IIL, and RS-IIL, by natural plant and microbial polysaccharides, including the widely used commercial food additives (thickening, stabilizing, and foaming agents). The examined natural polysaccharides included two from leguminous plant seeds, locust (carob)-bean gum (E-410, HL-350/450) and guar gum (guaran, E-412) galactomannans (*28*); two from plant stems, acacia (arabic) gum (E-414) (*29*) and inulin (from chicory) (*30, 31*); bacterial xanthan gum (E-415) (*32*); and yeast mannan (*33, 34*) (**Figure 2**). These polysaccharides were chosen for several reasons: (a) their presence in embryo-protecting plant seeds (of carob and guar) and plant stem wounds (acacia bark exudate) that might be associated with in vivo anti-infection function; (b) the preliminary observation of PA-IL binding by locust-bean and guar-gum galactomannans (*20*); (c) their resistance to digestion (*30*), ensuring the endurance of their effect in the gastrointestinal tract; (d) their wide usage in the food industry, being added even to baby's milk formulas, indicating their low or nontoxicity; and (e) the documented improvement of gastrointestinal physiological functions and of microflora composition [including prebiotic effects (*28, 30, 31, 35*)], even in the babies receiving them in enriched milk formulas (*28, 31*), in animals (*34*), and in adult human beings (*30, 36*). The antiadhesion potentials of the examined polysaccharides, assayed by their ability to inhibit the hemagglutinating activities (binding to human erythrocytes) of the four bacterial lectins (PA-IL, PA-IIL, CV-IIL, and RS-IIL) and the plant reference mannophilic without fucophylicity lectin Con A, were compared to those of human and cow milks, pigeon and quail egg whites, and beehive products (RJ and honey), which function as highly effective antiadhesion RMGDs (*15, 17, 19*).
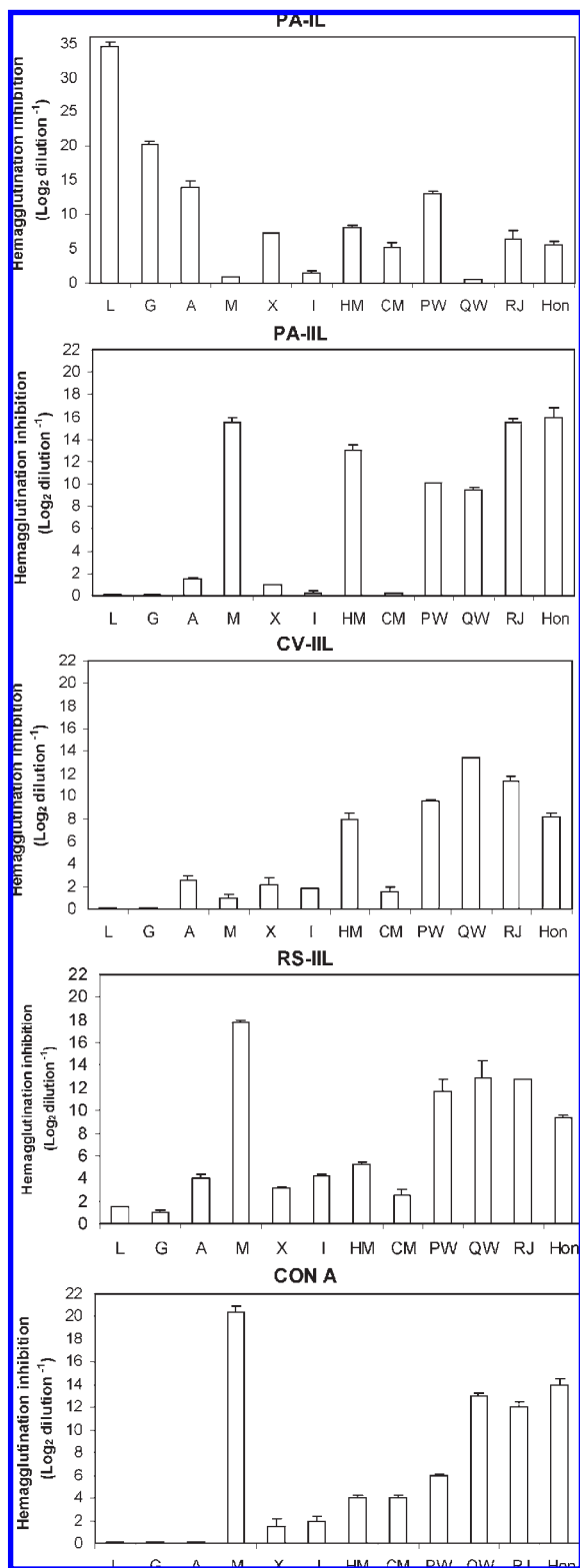
Figure 3. Inhibitions of the hemagglutinating activities of the four bacterial lectins *P. aeruginosa* PA-IL and PA-IIL, *C. violaceum* CV-IIL, and *R. solanacearum* RS-IIL and of the plant lectin Con A by 2-fold dilution series of locust-bean (L), guar (G) and acacia arabic (A) gums, yeast mannan (M), xanthan gum (X), and inulin (I), all of them beginning at 5 mg/mL concentration—as compared to their inhibitions by dialyzed human and cow milks (HM and CM) and egg whites of pigeon and quail (PW and QW) GPs and by RJ and honey (Hon). The data presented in this figure represent means ± SEMs of at least five experimental results for each lectin. The inhibiting polysaccharide concentrations can be easily

MATERIALS AND METHODS

Lectin Preparations. The bacterial lectins PA-IL, PA-IIL, CV-IIL, and RS-IIL were isolated from cell extracts of *P. aeruginosa* ATCC 33347, *C. violaceum* (Bergonzini) ATCC 12472, and *R. solanacearum* ATCC 11696, which were purchased from the American Type Culture Collection (ATCC) (Manassas, VA). They were purified by affinity chromatography, and their quality was controlled by sodium dodecyl sulfate−polyacrylamide gel electrophoresis with Coomassie brilliant blue staining, as earlier described (*1−3*). Con A was purchased from Sigma-Aldrich Co. (St. Louis, MO).

Examined Preparations. *Polysaccharides.* The six polysaccharides used—locust-bean (carob) gum, guar gum (from *Cyamopsis tetragonoloba* seeds), acacia gum (from *Acacia senegal* tree bark), mannan (from *Saccharomyces cerevisiae*), xanthan (from *Xanthomonas campestris*), and inulin (from chicory roots)—were purchased from Sigma-Aldrich Co. Each one of them was the only type available from that company. The interactions of these preparations with the examined lectins accorded their reported sugar composition, without apparent nonspecific reactions.

Milks. Human milks were obtained from healthy mothers (volunteers), and cow milks were purchased from food markets. They were centrifuged (10000*g*) for 10 min, and the intermediate phase was carefully separated, dialyzed overnight against saline (0.85% NaCl), and stored at −20 °C.

Avian Egg Whites. Pigeon and quail eggs were obtained from local bird breeders. The egg whites were separated, dialyzed overnight against saline, and stored at −20 °C

RJ and Honey. The RJ preparations were obtained from the beehive of Kfar Habad, Israel, and the honeys were purchased from food markets.

Hemagglutination and Its Inhibition Tests. Papain-treated human type O(H) red blood cells (erythrocytes, kindly obtained from the Magen David Adom National Blood Services in Israel) were used. They were prepared by three washings of the separated erythrocytes with PBS (phosphate-buffered saline, containing 0.025 M phosphate buffer at pH 7.2) and then treatment by 0.1% papain with 0.01% cysteine, as previously described (*1*). A 50 μL sample of each bacterial lectin preparation examined was serially diluted with 50 μL saline to produce 2-fold dilutions. Then, saline and 5% (V/V) erythrocyte suspension in saline (50 μL each) were added to each tube. After 30 min at room temperature, the tubes were centrifuged for 30 s (1000*g*), and the hemagglutinating activity was examined, as previously described (*1*).

In the hemagglutination inhibition test, each examined solution was serially 2-fold diluted in 50 μL of saline, and then, 50 μL of the lectin solution (at the highest dilution leading to agglutination of all of the erythrocytes in one large mass) was added to each tube. After 30 min at room temperature, 50 μL of the 5% papain-treated human O blood type erythrocyte suspension was added to each tube, and after another 30 min, hemagglutination was examined, as described above (*1*). The hemagglutination inhibition intensity was represented by the number of 2-fold dilutions (log $_2$ dilution^{-1}) without considerable hemagglutination preceding its reappearance.

Statistical Evaluation. The results of the hemagglutination and its inhibition tests were analyzed by Student's *t* test.

RESULTS

The inhibitions of the hemagglutinating activities of the five lectins (PA-IL, PA-IIL, CV-IIL, RS-IIL, and Con A) by the six polysaccharide series of 2-fold dilutions, beginning at a concentration of 5 mg/mL each, were compared to those of six animal products (human and cow milks, pigeon and quail egg whites, RJ, and honey) (**Figure 3**).

calculated from the *Y*-axis Log$_2$ dilution $^{-1}$ values of the 2-fold dilutions of the initial 5 mg/mL polysaccharide solution (2 represents 2^2 = 4-fold dilution, equaling a concentration of 1.25 mg/mL, and 10 is 2^{10} = 1024-fold dilution, equaling a concentration of approximately 4.9 μg/mL. Similarly, 20 is a 2^{20}-fold dilution, equivalent to approximately 4.77 ng/mL and 30 to about 4.66 pg/mL).

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P. aeruginosa galactophilic lectin PA-IL was most strongly inhibited by locust-bean gum (at the range of 1 pg/mL), followed by guaran (at the range of 1 ng/mL). The inhibiting activities of these two natural polydendritic galactomannans were far more potent than those of acacia arabic gum, which was also a fairly strong inhibitor, resembling the pigeon egg white GPs. Xanthan was a weaker PA-IL inhibitor, like the milk GPs and the beehive products (RJ and honey), whereas yeast mannan and inulin did not inhibit it at all, resembling the mannosylated quail egg white GPs (**Figure 3**).

P. aeruginosa fucophilic (mannophilic) lectin PA-IIL was not inhibited at all by the two galactomannans, neither was it sensitive to the acacia gum, xanthan, or inulin. However, it was very strongly inhibited by the branched yeast mannan, resembling its inhibitions by human milk GPs and the beehive products, which are stronger inhibitors of PA-IIL than the quail and pigeon egg white GPs (**Figure 3**).

C. violaceum fucophilic lectin CV-IIL resembled PA-IIL in displaying no sensitivity to the locust-bean and guar gums and low sensitivity to the acacia and xanthan gums and inulin, while exhibiting profound sensitivity to human milk, two avian egg whites, RJ, and honey. However, in profound contrast to PA-IIL, which was very strongly inhibited by yeast mannan, CV-IIL was not sensitive to this polysaccharide (**Figure 3**).

R. solanacearum mannophilic lectin RS-IIL, like PA-IIL, CV-IIL, and Con A, displayed very low sensitivity to the two galactomannans and low sensitivity to arabic gum, xanthan, and inulin, resembling its sensitivity to cow milk. However, unlike CV-IIL, it was most sensitive, even more than PA-IIL (closer to Con A), to inhibition by the yeast mannan, surpassing its sensitivities to the animal glycans examined (**Figure 3**).

DISCUSSION

As described in the Introduction, antibiotic resistance of pathogenic bacteria and a shortage of new drugs have stimulated the search for new strategies aimed to abrogate infections, including hampering pathogenic bacterial lectin-dependent adhesion to host cells. The discovery that the interactions of bacterial lectins (which are present on bacterial surface and toxins) with target cell receptors involve simultaneous polyvalent reactions with multiple complemetary receptors, which are stronger than the corresponding monovalent interactions (*25*), led to massive production of multivalent, competitive, lectin-inhibiting compounds. These compounds, which consist of repeating copies of polypeptidic or polysaccharide scaffolds that bear multiple dendritic glycosylated receptors, were shown to inhibit the binding of bacterial adhesins and toxins at subnanomolar inhibitory concentrations (*22, 23, 25, 27*). Sinclair et al. (*37*) have recently suggested an alternative prophylactic strategy against cholera toxin-induced global gastrointestinal disease based on the addition to food of oligosaccharides that functionally mimic the cholera toxin-binding target cell surface receptors as emulsifiers, stabilizers, or sweeteners. They pointed out the advantage of these indigestible oligosaccharides that pass into the small intestine, which is the site of cholera-toxin pathogenesis. Similar ideas have led us to examine the ability of several natural galactosylated and mannosylated plant and microbial polysaccharides, which are widely used in the food industry as emulsifiers and stabilizers, to block the galactophilic PA-IL, fucophilic PA-IIL and CV-IIL, and mannophilic RS-IIL produced by the pathogenic bacteria *P. aeruginosa*, *C. violaceum*, and *R. solanacearum*.

The locust (carob)-bean and guar bean gums (*20, 28*), which constitute approximately 35% of their seed endosperm mass, are widely used as economical food thickeners and stabilizers (E410 and E412). They are composed of Galα1-6 branches linked to Man

β(1-4)-linked backbone (scaffold) in Gal:Man ratios of 1:3.5−4 and 1:1.5−2, respectively (**Figure 2**). The first contains about 2000, and the second contains about 10000 residues.

The acacia gums (*29*) collected from exuded sap of bark (stems and branches) and fruit of the African leguminous *A. senegal* tree at sites of injury to the plant (sealing the injured area from invasion of microorganisms), are also widely used in the food industry as thickening, flavoring, and emulsion-stabilizing agents (E414). Their chemical composition is less defined, albeit unlike the neutral guar and locust-bean gums; they are slightly acid (due to D-glucuronic acid and several D-gluconic, D-galacturonic, and L-guluronic acid residues existing as mixed calcium, magnesium, and potassium salts) heteropolysaccharides (of MW around $250−286 \times 10^3$), containing a complex mixture of about 2% polypeptides and 88% highly branched arabic gum composed of D-galactose, L-arabinose, L-rhamnose, and D-glucuronic acid residues at an approximate molar ratio of 3:3:1:1). There are many Galβ1-3-linked side chains joined to the backbone main poly-Galβ1-3 chain by 1-6 linkages with β-D-glucopyranosyl and 4-*O*-methyl-β-D-glucopyranosyl, mostly as end units (*29*).

Xanthan gum (*32*), which is involved in the sticking of the plant pathogen *X. campestris* to the leaves of cabbagelike plants, is also an anionic polyelectrolyte with a cellulose-like Glcβ1-4 backbone and side chains containing terminal Manβ1-4 (with about 40% pyruvylation)-D-glucuronic acid α1-2 Man (mostly 6-acetylated) linked on alternating Glc residues (**Figure 2**). It is used in food (E415) as a thickener, stabilizer, emulsifier, and foaming agent and also in drug delivery industries.

Inulin, which is produced in diverse plants from sucrose, is composed of linear chains of β1-2-linked D-fructofuranosides, with glucose residues in their nonreducing end groups (**Figure 2**). It is also an undigestible, widely used food additive with documented positive effects on intestinal microflora and human (including infants) health (*30, 31*).

The baker's yeast *S. cerevisiae* mannan (*33, 34*) consists of a polymeric Manα1-6-linked scaffold that bears branches of Manα1-2 and Manα1-3 residues (**Figure 2**). As seen in **Figure 3**, the galactophilic PA-IL, which preferentially reacts with Galα1-6 branches (*12*), precipitated the locust-bean and guar gums and was very strongly blocked by their Galα1-6 branches (at pg/mL and ng/mL concentrations, respectively). The inhibitory effects of these two galactomannans even surpassed that of the pigeon egg white GPs, which bear Galα1-4 branches (*16*) (**Figure 1**) that display blood group P-system epitopes that are PA-IL main receptors on human cell surfaces (*38*). PA-IL considerable inhibition by the acacia gum (**Figure 3**) might be due to Galβ1-3-linked dendrites, and its mild inhibition by xanthan might be attributed to occasional galactosylations that occur on this substance. Its resistance to inhibition by yeast mannan and inulin was anticipated based on that observed with the polymannosylated quail egg white GPs.

Unlike the galactophilic PA-IL, the fucophilic and mannophilic/fructophilic PA-IIL, RS-IIL, and Con A were indifferent to the galactomannans and to the linear inulin polyfructosan (**Figure 3**), despite their mannose- and fructose-rich chains (both presented in **Figure 2**). This might be attributed to their exclusive binding to these residues on branches, such as those of the yeast mannan Manα1-2 and Manα1-3 branches (**Figure 2**). The latter very strongly inhibited PA-IIL, RS-IIL, and Con A (at ng/mL concentrations) (**Figure 3**), exceeding their considerable inhibition by the animal mannosylated GPs. RS-IIL inhibition by mannan was found to be somewhat higher than that of PA-IIL (which displays Fuc over Man preference) and lower than that of Con A, which shares with RS-IIL lower sensitivity to human milk glycans while reacting with Man but not with Fuc.

As seen in **Figure 3**, CV-IIL, in profound contrast to PA-IIL, RS-IIL, and Con A, was not blocked by yeast mannan, despite sharing with them sensitivity to quail and pigeon egg white-, RJ-, and honey-mannosylated GPs (**Figure 3**). This difference might be associated with CV-IIL higher selectivity to the saccharide linkages, also exhibited in its much lower sensitivity to Le^a than that of PA-IIL and also as compared to its own preferential sensitivity to the H blood group antigen.

Comparison of the human and cow milk, pigeon and quail egg whites, as well as the beehive products to the plant seed and stem polysaccharide panel, originated from the recognition of the common denominator of these bioproducts that act as glycodecoys for coherent protection of those organisms, especially their new offspring, against infections. The results of this study are in accord with this concept, where Galα1-6 branches of the locust-bean and guar galactomannans (surrounding and protecting the leguminous embryos) are able, even more than the Galα1-4 branches of the pigeon egg white GPs, to block PA-IL binding to its target cell receptors on the human erythrocyte blood group P-system antigens that also bear Galα1-4 branches.

Taken together, the results of the present study indicate that the herein-examined branched polysaccharides, some of them keeping with their natural function of anti-infection protection, very strongly and selectively inhibit the pathogenic bacterial lectins by their multiple galactosylated and mannosylated branches. In virtue of their long-term broad commercial usage as common nontoxic food additives with documented indigestability and health-improving effects, even in infants with gastroesophageal reflux (*28*), the examined plant and bacterial polysaccharides might be superior to milk, avian egg, and RJ GPs for infection prevention. They might be added to dairy products for the prevention of *P. aeruginosa* infection transmission by dairy products originating from *P. aeruginosa*-infected goat, sheep, or cattle. Furthermore, they might be generally applied for the prevention and treatment of intestinal and external (skin) infections. However, it should be kept in mind that although lectin blocking has been shown to be useful for certain infection therapy (*39*, *40*), it might not abolish hydrophobic or other nonspecific bacterial adhesion to their target cells. The final treatment in those cases might require additional drugs combined with antibacterial elements.

ABBREVIATIONS USED

Con A, concanavalin A, *Canavalia ensiformis* lectin; *C. violaceum*, *Chromobacterium violaceum*; CV-IIL, *C. violaceum* lectin; Fuc, L-fucose; Fru, D-fructose; Gal, D-galactose; GP, glycoprotein; Le^a, Lewis (a); Man, D-mannose; *P. aeruginosa*, *Pseudomonas aeruginosa*; PA-IL, *P. aeruginosa* first (galactophilic) lectin (LecA); PA-IIL, *P. aeruginosa* second lectin (LecB); PBS, phosphate-buffered saline; RMGD, receptor-mimicking or -simulating lectin-blocking glycodecoy; *R. solanacearum*, *Ralstonia solanacearum*; RS-IIL, *R. solanacearum* second lectin

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