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# Production, Properties and Specificity of a New Bacterial L-Fucose- and D-Arabinose-Binding Lectin of the Plant Aggressive Pathogen *Ralstonia solanacearum*, and Its Comparison to Related Plant and Microbial Lectins[1]

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**The worldwide distributed plant aggressive pathogen *Ralstonia solanacearum*, which causes lethal wilt in many agricultural crops, produces a potent L-fucose-binding lectin (RSL) exhibiting sugar specificity similar to that of PA-IIL of the human aggressive opportunistic pathogen *Pseudomonas aeruginosa*. Both lectins show L-fucose > L-galactose > D-arabinose > D-mannose specificity, but the affinities of RSL to these sugars are substantially lower. Unlike *Ulex europaeus* anti-H lectin, but like PA-IIL and *Aleuria aurantia* lectin (AAL), RSL agglutinates H-positive human erythrocytes regardless of their type, O, A, B, or AB, and animal erythrocytes (papain-treated ones more strongly than untreated ones). It also interacts with H and Lewis chains in the saliva of "secretors" and "nonsecretors." RSL purification is easier than that of PA-IIL since *R. solanacearum* extracts do not contain a galactophilic PA-IL-like activity. Mass spectrometry and 35 N-terminal amino acid sequencing enabled identification of the RSL protein (subunit ~9.9 kDa, ~90 amino acids) in the complete genome sequence of this bacterium. Despite the greater phylogenetic proximity of *R. solanacearum* to *P. aeruginosa*, and the presence of a PA-IIL-like gene in its genome, the RSL structure is not related to that of PA-IIL, but to that of the fucose-binding lectin of the mushroom (fungus) *Aleuria aurantia*, which like the two bacteria is a soil inhabitant.**

**Key words: bacterial lectin, blood groups, L-fucose, plant pathogen, *Ralstonia solanacearum*.**

Fucose-binding lectins in various organisms, including plants, animals, and microorganisms, have been described. Some, like UEA-I of the plant *Ulex europaeus*, exhibit strict anti-H (type-2) specificity (*1*). Others, like the plant *Griffonia simplicifolia* GS-IV (*2*) and *Ulva lactuca* ULL (*3*), as well as the bacterial *Pseudomonas aeruginosa* PA-IIL (*4–6*) and mushroom *Aleuria aurantia* AAL (*7, 8*), react with Lewis in addition to the H epitopes and exhibit a wider hemagglutinating spectrum.

Owing to their stability and special sugar specificity, AAL and PA-IIL have been used for carbohydrate probing, fractionation and study (*9–11*). Studies on the effects of PA-IIL on animal cells were also important for understanding its role in *P. aeruginosa* pathogenicity (*10, 12*). Following identification of a PA-IIL gene and protein sequences (*13*), we have revealed that there is a PA-IIL-like gene, encoding a putative protein similar to PA-IIL in subunit molecular mass and exhibiting 70% identity with its amino acid sequence, in the aggressive phytopathogenic bacterium *Ralstonia solanacearum* (*14*). This finding led to the discovery of a fucose-binding lectin, RSL, in extracts of this bacterium (*14*). *R. solanacearum* is one of the world's most important phytopathogens (*15, 16*), causing lethal wilt in hundreds of plant species, including potato, tomato, peanut, tobacco, eggplant, and banana (*17*). Although it is most troublesome in the tropics and subtropics, *R. solanacearum* also threatens cooler climate crops, especially potatoes and tomatoes. Because of its economical importance, the complete genome sequence of this bacterium was determined (*18*), and there is increasing interest in identifying the factors which contribute to this soil-located bacterium's ability to attach to, invade and colonize plant roots.

The present paper describes the production and properties of this new fucose-binding lectin, RSL, and its sugar specificity and affinity, as well as its hemagglutinating activity towards diverse erythrocyte types and interactions with blood group substances in saliva. The latter were compared to those of *P. aeruginosa* lectins and plant lectins UEA-I and Con A, which are related to RSL in fucose and mannose binding. It ends with a study of its amino acid sequence and its surprising partial homology to AAL.

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Abbreviations: AAL, *Aleuria aurantia* lectin; PA-IL, *Pseudomonas aerugionsa* galactophilic lectin; PA-IIL, *P. aeruginosa* fucose-binding lectin; RSL, *Ralstonia solanacearum* lectin; UEA, *Ulex europaeus* agglutinin.

## MATERIALS AND METHODS

*Bacterial Cultures and Extract Preparation*—P. *aeruginosa* ATCC 33347 and *R. solanacearum* ATCC 11696 were grown for 3 days (at 28°C with vigorous shaking) in Nutrient Broth (N.B.) and Eagon-Grelet (E.G.) media with and without choline, as previously described (*6*).

The harvested cells were disintegrated by ultrasonic vibration and the separated cell-free extracts were collected.

*Erythrocytes*—Human O, A, B, and AB as well as sheep, rabbit, rat and mouse erythrocytes were washed three times with a phosphate-buffered (0.025 M, pH 7.2) isotonic NaCl solution (PBS). Papain-treated cells were obtained by incubating a 5% v/v washed cell suspension in PBS with a 0.1% papain (crude preparation, Sigma) and a 0.01% cysteine solution, with shaking, at 37°C for 30 min. The enzyme-treated cells were washed three times in PBS and then resuspended in it to a concentration of 5%.

*Hemagglutination and Inhibition Tests*—0.05 ml of each bacterial lectin preparation examined was serially diluted with 0.05 ml of saline to produce two-fold dilutions. 0.05 ml of the erythrocyte suspension was added to each tube. After 30 min at room temperature, the tubes were centrifuged for 30 s (1,000 ×*g*) and the hemagglutination in each of them was graded on a scale of "0–10." The hemagglutinating score was calculated by summing up the values obtained for the positive tubes.

In the hemagglutination-inhibition test, each examined sugar solution (0.3 M concentration) was serially diluted in 0.05 ml volume and then 0.05 ml of the lectin solution (giving a titre of 1/16–1/32 in the above-described hemagglutination test, beginning with maximal agglutination–"10") was added to each tube. After 30 min at room temperature, 0.05 ml of the 5% erythrocyte suspension was added to each tube, and after another 30 min, hemagglutination was examined as above. The inhibition intensity was calculated by subtracting the hemagglutination score obtained for *n* tubes of the series containing inhibitor dilutions from *n*-fold "10."

*Purification of Microbial Lectins*—RSL and PA-IIL were purified essentially as described before (*6*), by heating to 70°C for 15 min, precipitation by 70% neutralized ammonium sulfate saturation, and then affinity chromatography on a Sepharose-mannose column. The only difference being that PA-IL was removed from the PA-IIL preparation before the Sepharose-mannose column step. Such a step was not required for RSL, which was not accompanied by a PA-IL–like activity.

*SDS-PAGE*—The purified RSL and PA-IIL preparations examined, at a protein concentration of 0.5 mg/ml, were mixed with an equal volume of sample buffer [0.1 M Tris-HCl, pH 6.8, containing 4% (w/v) SDS (sodium dodecyl sulfate), 20% v/v glycerol, 0.2 M dithiothreitol (DTT), and 0.2% w/v bromophenol blue] and boiled for 3 min. Fifteen microliters of each and 10 μl of low range precision protein standards (Bio-Rad) were subjected to 15% SDS-PAGE, according to Laemmli (*19*). The gels were stained with Coomassie Brilliant Blue (CBB).

*Mass Spectrometry Analysis*—Mass spectrometry of the purified lectin preparation was performed at the Technion Protein Research Center (Haifa, Israel) using a matrix-assisted laser desorption ionization time of flight (MALDI-TOF) mass spectrometer (MALDI-TOF 2E; Micromass, Manchester, UK).

*N-terminal Amino Acid Sequence*—The lectin's N-terminal amino acid sequence was determined by Edman degradation performed both at the Technion Protein Research Center, Haifa, and independently by Dr. Jean Gagnon at IBS Grenoble, using a Precise Sequence model 492 (Applied Biosystems). Phenylthiohydantoin amino acid derivatives were identified and quantitated by HPLC analysis online as recommended by the manufacturer.

*Genetic Analysis*—The 35 N-terminal amino acid sequence of the purified RSL was used together with the PATTERN program (*20*) to search a non-redundant databank of sequences available on the Infobiogen internet site (http://www.infobiogen.fr).

*Plant Lectins*—UEA-I from *Ulex europaeus* and Con A were purchased from Sigma.

## RESULTS

The lectin-dependent hemagglutinating activities in extracts of *R. solanacearum* and *P. aeruginosa* grown in different media were found to be greatly affected by the culture medium composition (Figs. 1 and 2). Choline addition to the cultures stimulated *P. aeruginosa* lectin production but had no positive effect on the *R. solanacearum* lectin level. E.G. medium, which stimulated the galactophilic lectin PA-IL production on account of PA-IIL in the *P. aerugi-*

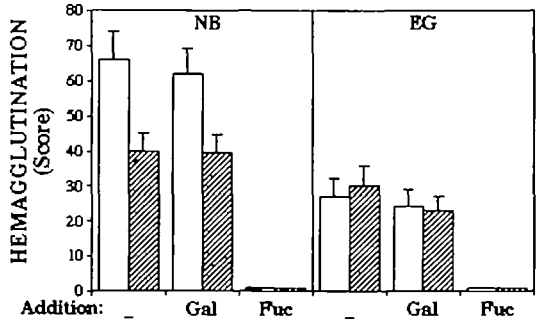


Fig. 1. **Hemagglutinating activities in cell-free extracts of *R. solanacearum* grown in N.B. and E.G. media without (□) and with (▨) choline addition.** The activities were examined in the absence and presence of 0.3 M galactose or fucose.

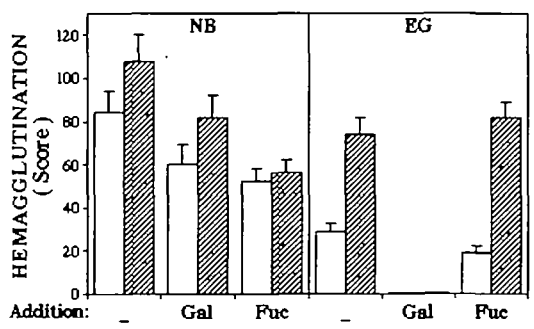


Fig. 2. **Hemagglutinating activities in cell-free extracts of *P. aeruginosa* grown in N.B. and E.G. media without (□) and with (▨) choline addition.** The activities were examined in the absence and presence of 0.3 M galactose or fucose.

*nosa* cells, repressed *R. solanacearum* RSL production. Therefore, N.B. was chosen for both RSL and PA-IIL production.

The four examined lectins (RSL, PA-IIL, Con A, and UEA-I) agglutinated all the papain-treated erythocytes much more strongly than the respective untreated ones. Therefore, the treated cells were used.

The agglutination of human and different animal erythrocytes by the examined lectins showed that RSL, PA-IIL, and Con A, as opposed to UEA-I, agglutinated all the animal erythrocytes examined (Fig. 3). The agglutination of the animal erythrocytes by RSL and Con A was stronger, while that by PA-IIL, and UEA-I was weaker than that of the human erythrocytes. UEA-I was almost strictly selective for the latter.

Figure 4 shows that agglutination of the H-positive human erythrocytes by RSL, PA-IIL and Con A occurred irrespective of the ABO blood type, while UEA-I exhibited significant anti-O(H) preference. The much weaker agglutination of B and A erythrocytes by UEA-I was due to interference by the presence of adjacent galactose or N-acetylgalactosamine residues (respectively) with its interaction with

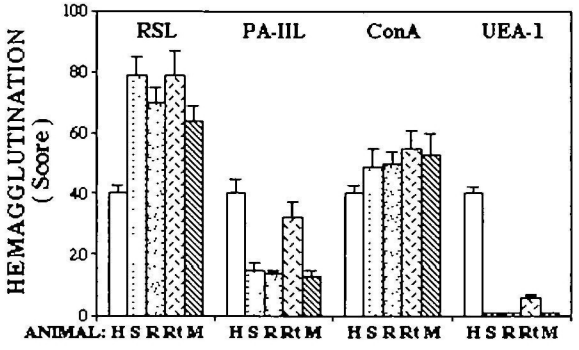


Fig. 3. **Hemagglutination of human (H), sheep (S), rabbit (R), rat (Rt), and mouse (M) papain-treated erythrocytes by RSL, PA-IIL, Con A, and UEA-I [using the dilution which gives similar activity towards human O(H) erythrocytes].**

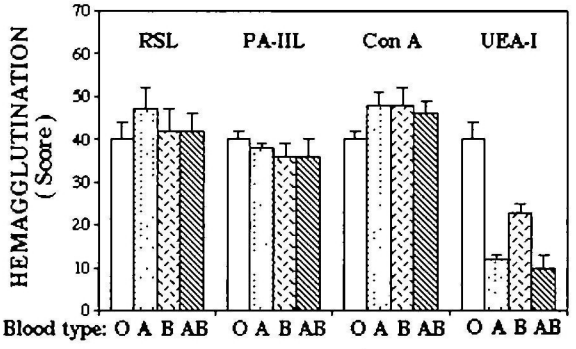


Fig. 4. **Hemagglutination of papain-treated human erythrocytes differing in the ABO blood type by RSL, PA-IIL, Con A, and UEA-I [using the dilution which gives similar activity towards human O(H) erythrocytes].**

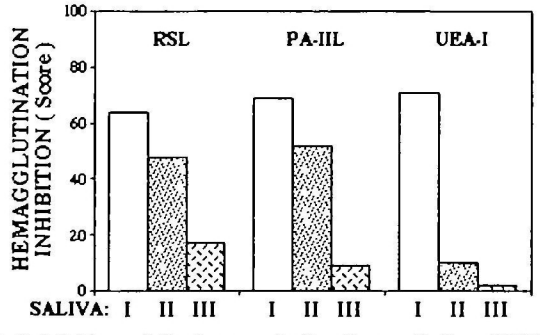


Fig. 5. **Inhibition of the hemagglutinating activity of RSL, PA-IIL, and UEA-I by saliva of Le$^b$ (I), Le$^a$ (II), and Le(a-b-) (III) donors.**

TABLE I. **Inhibition of RSL and PA-IIL hemagglutinating activities by the relevant sugars.**[a]

| | RSL | | PA-IIL | |
|---|---|---|---|---|
| | Concentration | rP | Concentration | rP |
| L-Fucose | $3.15 \times 10^4$ M$^{-1}$ | 100 | $5 \times 10^6$ M$^{-1}$ | 100 |
| L-Galactose | $6.3 \times 10^4$ M$^{-1}$ | 50 | $1 \times 10^6$ M$^{-1}$ | 50 |
| D-Arabinose | $5 \times 10^3$ M$^{-1}$ | 6.3 | $4 \times 10^5$ M$^{-1}$ | 12.5 |
| D-Mannose | $1 \times 10^2$ M$^{-1}$ | 3.15 | $3.15 \times 10^4$ M$^{-1}$ | 1.59 |
| D-Fructose | $1.2 \times 10^2$ M$^{-1}$ | 2.6 | $4 \times 10^4$ M$^{-1}$ | 1.25 |

[a]Minimal sugar concentration inhibiting each lectin hemagglutinating activity at the 30 hemagglutination score level. rP = relative inhibitory potential *versus* the inhibition by L-fucose = 100. This test is semiquantitative, allowing rough estimation of the relative inhibitory potential of the sugars. D-Ribose, L-arabinose, L-rhamnose, and D-xylose at the same concentration did not inhibit the hemagglutinating activities of these lectins.

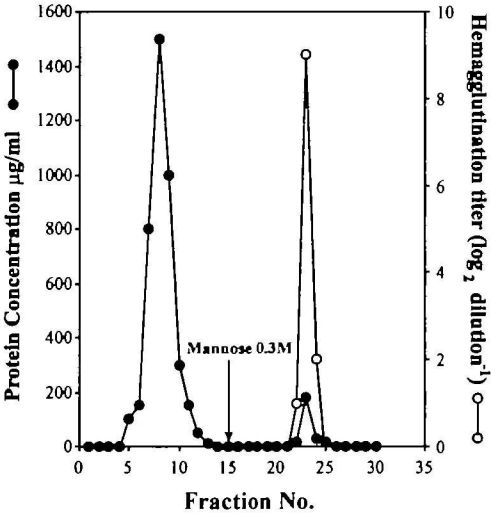


Fig. 6. **Elution profile on RSL Sepharose-mannose affinity chromatography with a 2 × 24 cm column and 10 ml fractions.**

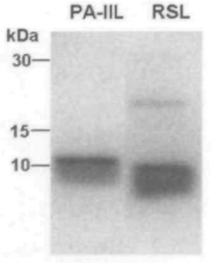


Fig. 7. **SDS-PAGE of the purified RSL preparation, compared to PA-IIL, stained with CBB.**

the H-specific fucose α2 residue.

Examination of RSL and PA-IIL inhibition by human saliva obtained from "secretors" $Le^b$, and non-secretors $Le^a$ as well as Le(a-b-) individuals revealed that both of them interacted with H and Lewis type chains (Fig. 5), while UEA-I specificity was restricted to the H chain.

Comparison of the inhibition of the two bacterial lectins by different sugars (Table I) showed that RSL and PA-IIL exhibit a similar inhibition profile: L-fucose > L-galactose > L-arabinose > D-mannose and fructose, but the RSL affinity to the respective sugars is much lower than the outstandingly high affinity of PA-IIL to them.

The lectin was efficiently purified by three steps: heating to 70°C (with no detectable lectin loss), ammonium sulfate (70% saturation) precipitation and Sepharose-mannose chromatography (Fig. 6).

SDS-PAGE analysis of RSL, compared to PA-IIL, (Fig. 7), revealed one or two very close subunit bands smaller (around 9.9 kDa) than that of PA-IIL (11,732 kDa). Since the difference between them was very small (180 Da), which might be equivalent to one or two residues, they were considered en-block as representing RSL. Another minor band was observed in the 20 kDa region. Its presence hampered quantitative amino acid analysis.

The MALDI-TOF mass spectrometry results indicated a major RSL peak at around 9.9 kDa, which was also divided into two sub peaks (9,810.15 and 9,996.52 Da, Fig. 8)—again considerably smaller than that of PA-IIL (11,732 kDa).

The RSL N-terminal amino acid sequence was found to be: SSVQTAATSWGTVPSIRVYTANNGKITERXWDGKG (where X probably stands for a cysteine residue), and indicated that an initiator methionine is not included in the RSL structure. This significant sequence information enabled a PATTERN search, which clearly indicated that this particular 35 amino acid sequence is present only at the N-terminal region of a putative protein translated from the complete genome sequence of *R. solanacearum* strain GMI1000 (Swiss Prot code Q8XXK6). This RSL protein, which consists of around 90 amino acids, may exhibit strain-dependent point divergence, as revealed by minute molecular weight variations.

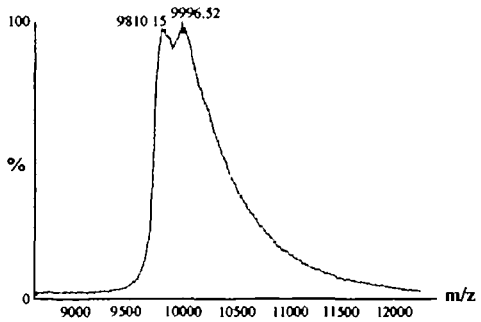


Fig. 8. **MALDI-TOF mass spectrum analysis of the purified RSL.**

The homology search performed with the RSL 90 amino acid sequence revealed the AAL from the mushroom *Aleuria aurantia* was the only protein exhibiting significant sequence identity with it (Fig. 9). As can be seen in this figure, alignment of the two repeating regions (43 and 47 amino acids) of RSL and six repeats of >45 amino acids of AAL show that each motif contains three reserved tryptophan residues (one being absolutely conserved) and two conserved glycine residues, as well as one basic (Arg) and one acidic (Glu) conserved residue. The pI of RSL was also found to be closer to the basic pI of AAL than to the very acidic one of PA-IIL (7.5 : 9.3 : 3.88, respectively).

## DISCUSSION

Despite the descriptions of numerous microbial lectins (*10*), there are only a few available in purified stable states that can be as widely used as plant or animal lectins. The reason is either a low level or instability. The *P. aeruginosa* and *Aleuria aurantia* fucose-binding lectins, PA-IIL and AAL, are among the exceptions. They have both been purified using conventional lectin purification procedures (*6, 8*), and the purified lectins are stable proteins like common plant lectins, being used like them in many experimental and diagnostic systems (*7–11*). Following identification of the PA-IIL gene (*13*), it has been revealed that the *R. solanacearum* genome contains an open reading frame encoding a putative protein that is shorter than PA-IIL by one amino acid, and its amino acid sequence exhibits 70% identity with that of PA-IIL. No PA-IL–like sequence was found in the *Ralstonia* genome. This information led us to examine *R. solanacearum* extracts for PA-IIL–like activity.

As can be seen in Fig. 1, *R. solanacearum* extracts obtained from cells grown in N.B. medium indeed exhibited high levels of L-fucose–binding lectin but no galactophilic activity. Under the same growth conditions, *P. aeruginosa* cells produced both PA-IL and PA-IIL (Fig. 2). An attempt to stimulate PA-IL–like lectin production by growing the cells in E.G. medium (*6*), which led to an increased PA-IL level on account of PA-IIL in *P. aeruginosa* cultures, did not induce similar galactophilic lectin production by *R. solan-*

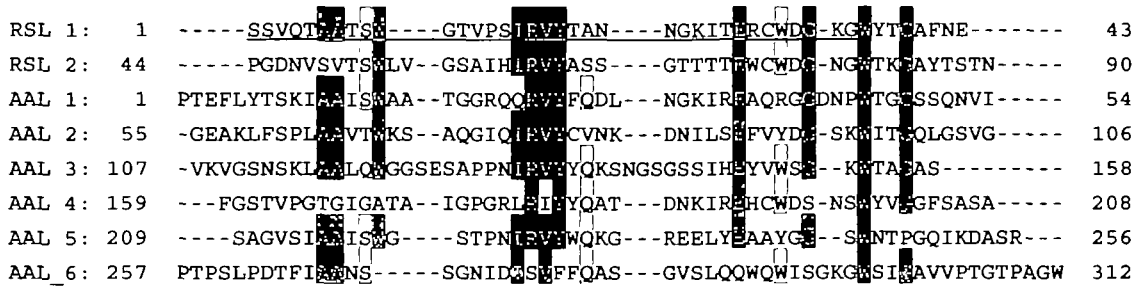


Fig. 9. **Amino acid homology of RSL within its molecule and with AAL repeats.** The experimentally determined N-terminal sequence is underlined.

*acearum*, while it repressed the RSL level. Furthermore, choline addition to the medium, which was shown to increase *P. aeruginosa* lectin level, was found not to be effective for elevating the RSL level. As in the case of the *Pseudomonas* lectins, most of the RSL activity was inside the cells, as opposed to the case of fimbrial hemagglutinins (*21*), which are present on the surface of *R. solanacearum* cells grown on stagnant medium under different conditions, but have not been shown to exhibit any sugar specificity.

The existence in two phylogenetically closely related bacteria of lectins exhibiting similar sugar specificities, poses a very special challenge as to comparison of their interactions with diverse cells and different sugars, and their affinities to them. Examination of the agglutination of different human and animal erythrocytes by RSL and PA-IIL, compared to Con A and UEA-I (Figs. 3 and 4), showed that all these lectins agglutinated papain-treated erythrocytes much more strongly than untreated ones, and that RSL more intensely agglutinated animal *vs* human erythrocytes. In this respect, RSL was more like the mannose-binding Con A, as opposed to the fucose-specific PA-IIL and UEA-I, which showed higher and exclusive (respectively) avidity to human erythrocytes, preferentially of the O(H) blood type (Figs. 3 and 4). RSL, like PA-IIL, interacted with the H and Lewis chains, similarly agglutinating the H-positive A, B, AB, or O erythrocyte types, and interacting with the H and Le substances in human saliva (Fig. 5). These results prove that the addition of the B (galactose) and A (*N*-acetylgalactosamime)-specific sugars to the erythrocyte H chains interferes with UEA-I binding to them, but does not disturb PA-IIL and RSL interactions with them. Similar behavior has also been ascribed to AAL (*7, 8*).

As shown in Table I, RSL resembles PA-IIL in the sugar affinity order, but not in the magnitude, the order being similar to that of AAL [which also binds D-arabinose (*8*)], unlike the outstandingly high affinity magnitude of PA-IIL to these sugars.

RSL purification by the method used for PA-IIL (*6*) was found to be very efficient (Figs. 6 and 7) and much easier than PA-IIL purification, which requires an additional step to get rid of PA-IL.

Not withstanding our expectation, both N-terminal sequence and mass spectrum (Fig. 8) analyses of RSL revealed that it was not encoded by a *Ralstonia* PA-IIL–like gene. The RSL subunit is close to 9.9 kDa, while those of PA-IIL and the putative protein encoded by the *R. solanacearum* PA-IIL–like gene are both around 11.7 kDa. The latter protein (RS-IIL), which might be functionally related to PA-IIL (*22*), was not expressed in the examined *R. solanacearum* extracts but the conditions required for its production are now under investigation (at Bar-Ilan University). The most surprising finding was the similarity of the RSL 2 repeating segment (43 and 47 amino acids each) structures to the six repeating segments (>45 amino acids each structure) of AAL, which is produced by the mushroom *A. aurantia* (*23*), which is not so phylogenetically close to *R. solanacearum*, but is present with it in soil. The conserved tryptophans shown on alignment might be involved in either the sugar binding or the hydrophobic interactions between the repeats, as observed for Ricin (*24*), while the conserved acidic amino acids and arginine might be related to those present in the lectin loops that are associated with the sugar binding.

Overall, this paper describes a new fucose-binding lectin, RSL, which is closely related to PA-IIL and AAL in sugar specificity. The plant pathogen *R. solanacearum* which produces it lacks PA-IL–like galactophilic lectin such as that produced together with PA-IIL by the human pathogen *P. aeruginosa* (*4*). The availability of RSL, PA-IIL, and AAL, all of which bind fucose and arabinose to a higher degree and mannose to a lower one, showing different affinities, may be of great value for the study of lectin-carbohydrate interactions, and the factors which govern their sugar specificities and affinities. Knowledge of the above described affinities and particularly the difference in galactophilic lectin production may also contribute to understanding of the basis of the different host selectivities (*4*) of these two bacteria. The bacterial galactophilic lectin is more adapted to bind to galactose-bearing antigens (*4*), which are more abundant on mammalian cells, than ultimate fucose, arabinose or mannose residues (*25*). The relevance of fucose and arabinose-binding in soil microorganisms and plant pathogens might be related to the sugar composition of plant cell walls that contain these sugars. Fucose residues are present as part of xyloglucans, complex branched polysaccharides also called hemicelluloses, that are found in the primary cell walls of higher plants (*26*). More interestingly, fucose-containing xyloglucan oligomers have been shown to exert signalling effects on plant tissues (*27*). In this oligomer, the α-L-fucose is attached to position 2 of galactose, thereby creating a blood group H-type disaccharide, which has been shown to bind to the three discussed microbial lectins.

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