The LlpA protein from *Pseudomonas putida* represents the prototype of a new family of antimicrobial agents with bacteriocin-like properties. Members are found in Gamma- and Beta-Proteobacteria, including plant and human pathogens, and mediate strain-specific intra-genus killing across species borders but without the need for a cognate immunity protein.

On the way to elucidate its mode of action, the 3D-structure of LlpA was determined, enabling the identification of structural determinants that contribute to activity and specificity. LlpA is built from two tightly interacting β-prism-folded MMBL domains stabilized by a β-hairpin. The MMBL (monocot mannose-binding lectin) domain is commonly found in eukaryotic lectins, being particularly widespread among plants but also recently identified in several fish and fungal species. Each MMBL domain in LlpA contains three putative mannose-binding motifs, exhibiting different levels of sequence degeneracy. Of the six sites, only three retain a potential carbohydrate-binding architecture. Soaking of crystals with different sugars demonstrated the binding of methyl-D-α-mannopyranoside and oligomannosides to only one of these sites in the carboxyterminal domain. Binding of methylmannose to LlpA with millimolar affinity was confirmed by isothermal titration calorimetry. LlpA mutant proteins with a sterically occluded binding site displayed reduced bacteriocin activity. Differential activity of engineered *P. putida/P. fluorescens* LlpA chimers revealed that target specificity is mainly determined by the aminoterminal domain. The latter is structurally more divergent from the plant lectin domains compared to the sugar-binding carboxyterminal domain.