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Pseudomonas chlororaphis (PA-23) and its transposon derived mutants differentially displayed antagonism against *Rhizoctonia solani*

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Biological control provides alternatives to the extensive use of fungicides in plant protection Bacteria have proved to be excellent sources of antagonists, owing to their multiple mechanisms of disease control. *Pseudomonas* spp. mediate crop protection by exerting mechanisms of inhibitory activity such as the production of extracellular enzymes, competition, induced systemic resistance and antibiosis. Direct antagonism of the pathogen through antibiosis is one of the mechanisms by which disease-suppressive bacteria achieve disease control. Antibiosis is mediated through the production of a chemically heterogeneous group of organic, low-molecular weight compounds which, at low concentrations, are deleterious to the growth or metabolic activities of other microorganisms. *Pseudomonas chlororaphis* strain PA23 is a strong antagonist of several fungal pathogens. This bacterium was previously reported to produce phenazines and pyrrolnitrin as main antibiotics responsible for its antagonistic capacity. Several studies suggested that phenazines and pyrrolnitrin produced by pseudomonads are important for biocontrol of plant diseases. *Rhizoctonia solani* is one of the main soil-borne pathogens in Sri Lanka affecting many different crops. In Petri plate assay *P. cholororaphis* PA-23 antagonizes *R. solani* and prevents formation of sclerotia. Therefore, the main objective of the present study was to determine which antibiotic(s) is responsible for the inhibition of *R. solani in vitro*. To achieve this, transposon-derived mutant strains of *P chlororaphis* PA-23 which lacks biosynthesis of either phenazines (PA-23-63), pyrrolnitrin (PA23-8) or both antibiotics (PA23-63-1) were used. It was revealed that phenazine-minus mutant (PA23-63) lost antifungal activity completely while pyrrolnitrin-minus mutant (PA23-8) constitutively antagonized *R. solani* to the same extent as the wild type (PA-23). This indicates clearly that phenazine is responsible for antagonism for *R. solani* but there is no apparent role of pyrrolnitrin in antagonism. Furthermore, the double mutant (PA23-63-1), which lacks the ability of biosynthesis of both antibiotics, also lost antifungal activity. Effects of phenazine on the formation of sclerotia and degradation of fungal cell wall are under investigation.