J Tissue Sci Eng 2018, Volume 9
DOI: 10.4172/2157-7552-C3-052

Characterization of the multiple molecular mechanisms underlying RsaL control of phenazine-1-carboxylic acid biosynthesis in the rhizosphere bacterium *Pseudomonas aeruginosa* PA1201

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Phenazines are important secondary metabolites that have been found to affect a broad spectrum of organisms. These compounds exhibit wide-spectrum antimicrobial activity and their roles in the biological control of plant pathogens have been studied extensively. In addition, phenazines have also been used as lead molecules in biotechnological applications including environmental sensing, microbial fuel cell production and anticancer therapy. Therefore, researchers in disparate disciplines have long been interested in elucidating the mechanisms that control phenazines activity and biosynthesis. Due to rising concerns regarding the use of chemical pesticides in the 1980's, researchers became very interested in the antifungal properties of the phenazines produced by soil *Pseudomonas* and their potential use in the control of fungal diseases of plants. A 1% Shenqinmycin suspension which contains the phenazine PCA as active ingredient has recently been registered in China as a green biopesticide. Two almost identical gene clusters *Phz1* and *Phz2* are responsible for phenazines biosynthesis in the rhizobacterium *Pseudomonas aeruginosa* PA1201. The transcriptional regulator RsaL is a potent repressor of Phenazine-1-Carboxylic Acid (PCA) biosynthesis. RsaL negatively regulates *Phz1* expression and positively regulates *Phz2* expression via multiple mechanisms. First, RsaL binds to a 25 bp DNA region within the Phz1 promoter to directly repress *Phz1* expression. Second, RsaL indirectly regulates the expression of both *Phz* clusters by decreasing the activity of the las and pqs Quorum Sensing (QS) systems, and by promoting the rhl QS system. Finally, RsaL represses *Phz1* expression through the downstream transcriptional regulator CdpR. RsaL directly binds to the promoter region of CdpR to positively regulate its expression and subsequently CdpR regulates *Phz1* expression in a negative manner. We also show that RsaL represents a new mechanism for the turnover of the QS signal molecule N-3-oxododecanoyl-homoserine lactone (3-oxo-C12-HSL).

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