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Development of a dry, stable and inhalable acyl-homoserine-lactone-acylase powder formulation for the treatment of pulmonary *Pseudomonas aeruginosa* infections

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MENGESAHKAN
Saltanan/temuan sejati dengar
Surabaya...

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Dekan.

Dr. rer. nat. Maria Garetti M. Purwanto

1. Introduction

*Pseudomonas aeruginosa* is an opportunistic pathogen to humans, that becomes virulent in many hospital-acquired contaminations, such as in urinary tract, surgical wound, pneumonia and bloodstream infections. Patients with immunosuppression, cystic fibrosis (CF), chemotherapy and trauma have an increased risk for the infection (Jones et al., 2010; Lai et al., 2003).

Cell-to-cell signaling is an essential prerequisite for the establishment of *P. aeruginosa* infections (Donahedian, 2003; Van Delden and Iglewski, 1998). During invasion and infection, this bacterium switches on a subset of genes important for virulence to its host cells. Many of these virulence factors, such as rhamnolipid (Jeessen et al., 2007), are produced under the control of quorum sensing (QS) signaling molecules (Bjarnsholt et al., 2010; Defoirdt et al., 2010; Nadal Jimenez et al., 2012; Van Delden and Iglewski, 1998). *P. aeruginosa* possesses a complex QS system with at least three signal molecules, N-3-oxododecanoyl-homoserine lactone (3-oxo-C12-HSL), N-butyryl-homoserine lactone (C4-HSL) and 2-heptyl-3-hydroxy-4-quinolone (PQS) (Williams and Camara, 2009). Several studies reported that these signal molecules (Ericsson et al., 2002; Favre-Bonte et al., 2002; Singh et al., 2000) and also mRNA of the auto-inducer synthase gene luxS (Ericsson et al., 2002) can be detected at elevated levels in sputum samples of CF patients’ lungs. The signal molecules not only induce virulence, but they can also cause themselves inflammatory responses (Mayer et al., 2011; Zhu et al., 2008). These facts suggest that a suppression of the *P. aeruginosa* QS system might reduce expression of the virulence factors in the lung tissue of CF patients.

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