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Specific chemicals induce phenotype reversal and inhibit alginate production by mucoid *Pseudomonas aeruginosa*

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Bacterium *Pseudomonas aeruginosa* in the lung of cystic fibrosis patients often converts to a phenotype, mucoid, that produce excessive amount of alginate polymers. These alginate polymers form a thick mucus that causes difficulty in patient's breathing and contributes to both the morbidity and mortality of the patient. The cause and mechanism of this conversion of wild type to mucoid phenotype are still not clear. While there are commercial agents aim at washing and clearing the mucus, there are no drugs that control directly the mucus (alginate) production. Here, we demonstrate a specific class of synthetic molecules that can, in vitro, delay the production of alginate production, and inhibit the total amount of the alginate produced by mucoid phenotype. Our ongoing study of the mechanism of these chemical actions shows that one specific molecule can revert the mucoid phenotype back to wild-type *Pseudomonas aeruginosa* under conditions permit swarming. Structural variations of that specific chemical are NOT active for mucoid reversal and for alginate inhibition. This and other results suggest that the mode of action is likely a specific ligand-receptor binding event that triggers a cascade of chemical signaling events.

Biography

Yan-Yeung Luk has completed his PhD at The University of Chicago, and postdoctoral studies from University of Wisconsin-Madison. He has been faculty at the Chemistry Department of Syracuse University. He is the founder of LifeUnit LLC, a startup company focused on chemical control bacterial activities. He has published more than 30 papers in peer-reviewed journals and has been serving as a reviewer for NSF, and ACS journals. He is also a member on the scientific advisory broad of Orthobond INC.

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