Loss of cooperative traits affects strain competition in cystic fibrosis lung infection

Melanie Ghoul
University of Oxford, Oxford, United Kingdom

*Pseudomonas aeruginosa* is an opportunistic human pathogen that is the predominant cause of pulmonary exacerbation in patients with cystic fibrosis (CF), where most individuals succumb to infection at very early stages and subsequently suffer from prolonged chronic infections. *P. aeruginosa* harbours a suite of social and competitive traits. Among these traits is the production of bacteriocins, known as pyocins. An estimated 90% of *P. aeruginosa* strains produce pyocins and generally exhibit immunity to their own pyocin production. It has been demonstrated that social traits are lost with increasing colonization time over the course of a pulmonary infection when the bacteria become established, highly adapted and specialized to the CF lung. Pyocins are necessary to colonise the new environment of the CF lung, however, pyocigenicity and its associated competitive ability may be lost once an infection becomes established and chronic, in response to differential selective pressures within the lung. In this study I examine the diversity of pyocin production and resistance profiles of strains isolated from a non-lung environment and strains isolated at progressive stages from a pulmonary CF infection. Reduced pyocigenity and increased susceptibility to pyocins can have important implications in the treatment of *P. aeruginosa* pulmonary infections.