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# Diversity in structural consequences of MexZ mutations in *Pseudomonas aeruginosa*

**Samad Jahandideh**
Sanford-Burnham Medical Research Institute, USA

*Pseudomonas aeruginosa* is the major cause of morbidity and mortality in patients with cystic fibrosis. The high level of intrinsic and acquired antibiotic resistance of *P. aeruginosa* is attributable to the low permeability of its outer membrane in combination with the expression of several multidrug resistance efflux systems. MexZ, the negative regulator of the MexXY efflux pump, is found to be the most frequently mutated gene in *P. aeruginosa* isolated from cystic fibrosis patient lungs, confirming its importance in multidrug resistance. Structural consequences of four MexZ mutations, including L25P, G46V, P151L, and S202F, have been explored based on the known structure of MexZ using both molecular modeling and molecular dynamics methods. According to obtained results, G46V mutation, which completely abolishes the ability of MexZ binding to DNA, occurs in a specific evolutionary conserved region of MexZ. In addition, the most fluctuation values occur in DNA-binding domain and Helix4. The obtained results explore details of diversity in structural consequences of MexZ muta- tions in *P. aeruginosa*.

## Biography

Samad Jahandideh received his PhD in Biophysics from Tarbiat Modares University, Tehran, Iran in 2010. He then took his postdoctoral training in computational biology at the University of Alabama at Birmingham and Sanford-Burnham Medical Research Institute, La Jolla, California. Before moving to USA, he worked as an assistant professor and Vice-Chancellor for Research in the Department of Medical Physics at Shiraz University of Medical Sciences, Shiraz, Iran. He has published more than 25 papers in reputed journals and reviews research manuscripts at number of journals, including Bioinformatics, BMC Bioinformatics, and BMC Genomics.

sjahandideh@uab.edu