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Title:	Analysing non-synonymous mutations in XDR and MDR tuberculosis drugs
Authors:	Jainarayanan, A.K. (/jspui/browse?type=author&value=Jainarayanan%2C+A.K.) Anand, N.M. (/jspui/browse?type=author&value=Anand%2C+N.M.)
Keywords:	Tuberculosis XDR MDR Non-synonymous mutations
Issue Date:	2019
Publisher:	Elsevier
Citation:	Scientific Reports, 9(1).
Abstract:	<p>Tuberculosis is a bacterial disease caused by <i>Mycobacterium tuberculosis</i>. It is known to be the second-largest cause of death and models a severe risk to public health throughout the world. Though it affects people of almost every age, individuals with weakened immune systems, (e.g., HIV infection) are more likely to get infected. The present study deals with analyzing non-synonymous mutations in anti-tuberculosis drugs, which may have a significant role in causing XDR and MDR tuberculosis drug resistance. Continued use of tuberculosis drugs, discontinuation of medicines and various other factors can promote drug resistance in the host's body. To understand the actual cause of resistance, we have identified some patterns of mutations which might be responsible for a change in the structure of the protein, ultimately causing drug resistance. Here, we aim to present some of the unique mutation patterns in the genes associated with the marketed drugs that might have a deleterious effect. In this study, we have used molecular docking approach for understanding the ligand binding affinity of the mutated drugs. The results are further validated by molecular dynamics studies.</p>
Description:	Only IISERM authors are available in the record.
URI:	<a href="https://www.sciencedirect.com/science/article/pii/S2405579419300440">https://www.sciencedirect.com/science/article/pii/S2405579419300440</a> ( <a href="https://www.sciencedirect.com/science/article/pii/S2405579419300440">https://www.sciencedirect.com/science/article/pii/S2405579419300440</a> ) <a href="http://hdl.handle.net/123456789/1692">http://hdl.handle.net/123456789/1692</a> ( <a href="http://hdl.handle.net/123456789/1692">http://hdl.handle.net/123456789/1692</a> )
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