



Molecular Docking and ADME Guided Computational Identification of PKS-13 Inhibitors for Tuberculosis

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ABSTRACT,

Tuberculosis, one of the leading causes of mortality across the globe remains a prominent challenge and continues to affect the quality of lives of millions of people. Long treatment duration, improper prescription practices and premature discontinuation of therapy have led to the emergence of multi-drug resistant (MDR) and extensively drug resistant (XDR) Tuberculosis. In the present study, polyketide synthase-13 (PKS-13), a protein in the biosynthetic pathway of mycolic acids which is responsible for the final condensation step in the cell wall synthesis of the mycobacterium was targeted to identify potential inhibitors for tuberculosis using computational studies. 6H-[1]-benzofuro[3,2,c]-[1]benzopyran-6-one (coumestan) scaffold was subjected to similarity screening using SwissSimilarity tool. The resultant scaffolds were subjected to virtual screening against PKS-13 and prioritized based on docking scores. The docking scores of the coumestan analogues were in the range of -7.5 to -8.9 kcal/mol. The compounds were further screened for *in silico* ADME properties using SwissADME to eliminate potentially toxic molecules from further studies. The molecules with good docking scores and acceptable ADME properties are reported in this article.

Keywords: Tuberculosis, Polyketide synthase 13(PKS-13), mycolic acid synthesis, Similarity screening, SwissADME, PyRx

INTRODUCTION

Tuberculosis (TB) remains one of the most formidable infectious diseases affecting global public health, caused by the pathogen *Mycobacterium tuberculosis* (Mtb). Despite being a preventable and curable disease, TB continues to exact a devastating toll on human life, particularly in low- and middle-income countries. According to the World Health Organization (WHO), an estimated 10.8 million people fell ill with TB in 2023, with approximately 1.25 million deaths, making it one of the leading causes of mortality from a single infectious agent worldwide¹.

India accounts for approximately 27% of the global TB incidence, the highest of any country. In 2023, India reported an estimated 2.8 million new TB cases and over 3.2 million TB-related deaths². Current first-line treatment for drug-susceptible TB involves a six-month regimen comprising isoniazid, rifampicin, pyrazinamide, and ethambutol. While effective when adhered to properly, this prolonged therapy is associated with significant drawbacks including hepatotoxicity, peripheral neuropathy, and gastrointestinal disturbances³. The emergence of multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB) has further complicated treatment, necessitating second-line drugs such as bedaquiline, delamanid, linezolid, and fluoroquinolones. These regimens are longer (up to 18–24 months), more toxic, considerably more expensive, and achieve lower cure rates⁴⁻⁶. Consequently, there is an urgent need to identify novel therapeutic targets and develop novel anti-tubercular agents with improved efficacy and safety profiles.

The mycobacterial cell wall represents an attractive target due to its essential role in bacterial survival and pathogenicity. A critical component of this cell wall is mycolic acid, a long-chain fatty acid unique to

mycobacteria that confers intrinsic resistance to many antibiotics and host immune mechanisms⁷⁻⁹. The biosynthesis of mycolic acids involves the type I fatty acid synthase (FAS-I) and type II fatty acid synthase (FAS-II) systems, culminating in the final condensation step catalyzed by polyketide synthase-13 (PKS-13).^{10,11}

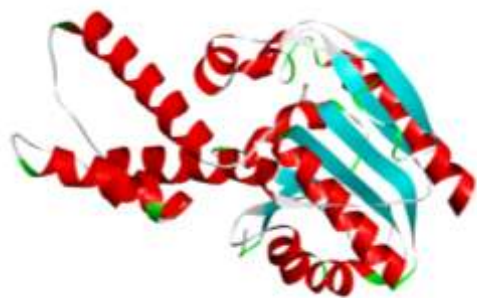


Fig 1: 3D representation of PKS-13 protein (PDB ID: 5V3W)

PKS-13 is an essential, multidomain enzyme that catalyzes the Claisen-type condensation of two fatty acyl chains, a meromycolyl chain and a carboxylated α -branch, to form α -alkyl β -ketoacyl products, the immediate precursors of mycolic acids. The 3D representation of PKS-13 protein used in the present study is shown in Figure 1. The enzyme possesses multiple functional domains including acyltransferase (AT), ketosynthase (KS), and thioesterase (TE) domains, each presenting potential druggable sites. Genetic studies have demonstrated that PKS-13 is essential for *M. tuberculosis* viability, and its inhibition leads to rapid bactericidal activity both *in vitro* and *in vivo*. The pathway and the condensation step of mycolic acid synthesis involving the role of PKS-13 is shown in Fig 2 and 3. The thioesterase domain, in particular, has emerged as a promising target, with several small-molecule inhibitors demonstrating potent anti-mycobacterial activity in preclinical studies.¹²⁻¹⁸

The essential nature of PKS-13 in mycolic acid biosynthesis, the absence of human homologs, and the availability of crystal structure, enables structure-based drug design and targeting PKS-13 represents a compelling strategy for developing novel anti-TB leads. Computer-aided drug design approaches such as molecular docking and *in silico* ADME profiling, are powerful tools for the identification of potent, drug-like PKS-13 inhibitors with favourable pharmacokinetic properties.

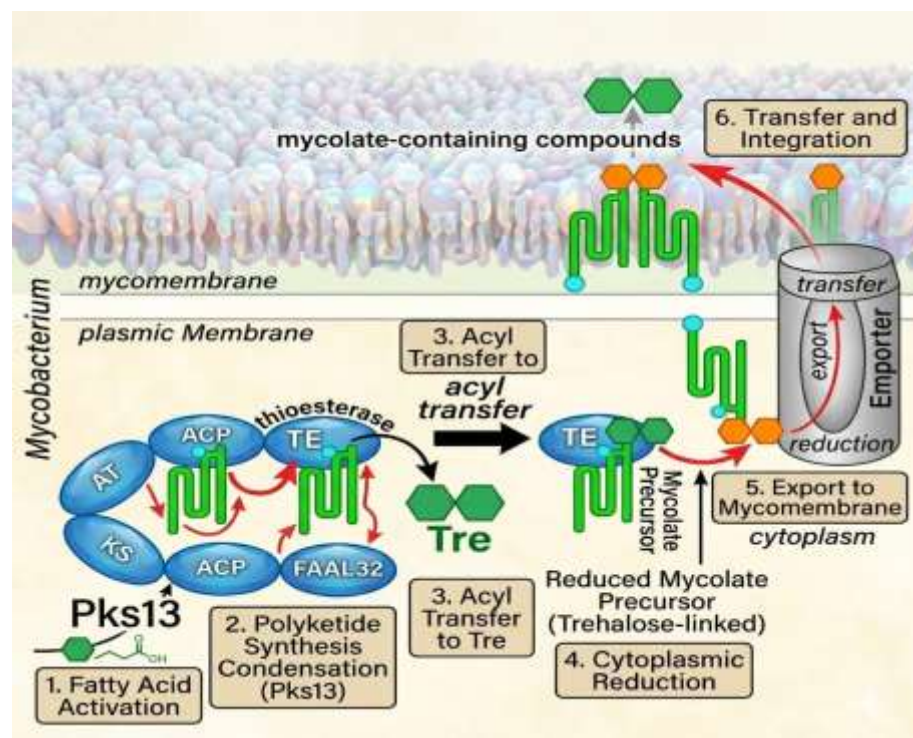


Fig 2: Pathway of PKS-13 protein showing the various domains of the PKS-13 protein essential for the synthesis of mycobacterium cell wall

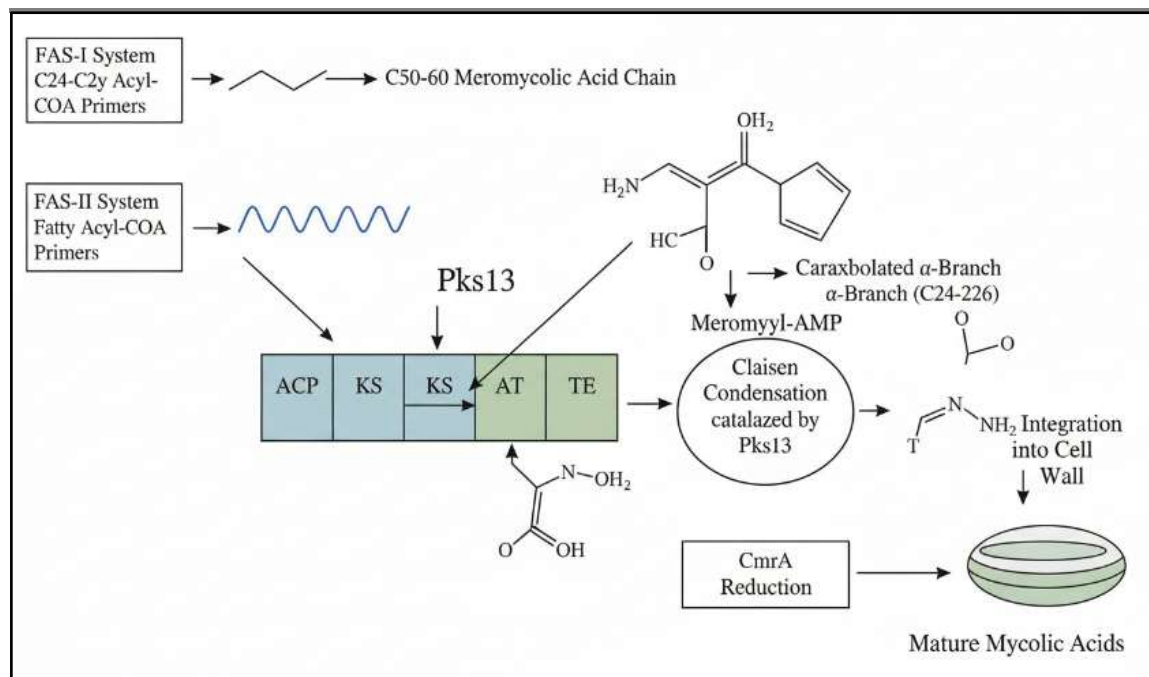


Fig 3: Condensation step catalysed by PKS-13 protein in mycolic acid biosynthesis pathway leading to the synthesis of mycolic acids

METHODOLOGY

The present study was designed for the identification of potential Polyketide synthase-13 (PKS-13) inhibitors, using computational techniques such as similarity screening, molecular docking, and *in silico* ADME profiling. Our objective was to computationally screen potential inhibitory compounds, evaluate their binding affinities to the PKS-13 protein, and predict their drug-likeness properties. The PKS-13 protein with pdb ID – 5V3W, was downloaded from the macromolecular database, RCSB Protein Data Bank.¹⁹ The protein, 5V3W has a resolution of 1.72Å and two chains A and B with 286 amino acid residues. In the present study, chain A was retained for the structure-based drug design study.

Active Site Identification

The identification of active site in protein is crucial for understanding their function. CASTp (Computer Atlas of Surface Topography of proteins) is a widely used tool for identifying and analyzing the geometric and topological properties of protein structures, including surface pockets and cavities, which are often indicative of active sites. In the present study, the active site of the PKS-13 protein (pdb ID- 5V3W) was identified using CastP.²⁰

Ligand Identification Using Swiss Similarity

Novel Coumestan-based derivatives have been identified by Shichun Lun *et al.*, as one of promising chemotype, exhibiting potent antitubercular activity through the selective inhibition of the PKS-13 enzyme.²¹ These molecules demonstrated strong efficacy against both drug susceptible and drug-resistant strains of *Mycobacterium tuberculosis* while maintaining minimal cytotoxicity toward healthy mammalian cells. Moreover, no cross resistance was observed between the Coumestan analogues and those currently available first-line antitubercular agents. Therefore, designing molecules using coumestan scaffold is an effective strategy for structure-based drug design.

Swiss Similarity is a powerful computational tool for virtual screening, enabling rapid identification of bioactive compounds having structural and pharmacophoric similarities with known active molecules. The tool integrates multiple similarity metrics such as electroshape, spectrophores, and molecular fingerprints, providing comprehensive scaffold comparison capabilities.²² It helps in early-stage drug discovery by efficiently narrowing vast chemical libraries to manageable candidate sets without requiring target structural information.

SwissSimilarity facilitates scaffold hopping, identifying chemically diverse compounds with conserved bioactivity potential. The similarity tool's accessibility, speed, and integration with curated databases including DrugBank²³ and ChEMBL²⁴ make it a valuable tool for computational medicinal chemists seeking novel therapeutic candidates with optimized pharmacological profiles.

Virtual Screening

Molecular docking is a powerful virtual screening tool, which helps in identifying potential leads. Molecular docking was carried out using AutoDock Vina module in PyRx virtual screening platform.²⁵ A grid box encompassing the active site of the PKS-13 thioesterase domain was defined with dimensions sufficient to accommodate ligand conformational flexibility. The exhaustiveness parameter was set to ensure adequate sampling of the conformational search space. The 157 coumestan derivatives were docked against the prepared protein structure at its active site. The co-crystal ligand 2Q5, ((2R)-2-[(2R)-2-[(2S)-2-[(2R)-2-oxidanyloxy]propoxy]propoxy]propan-1-ol) and the standard drugs used in TB treatment, Isoniazid, Ethionamide, Thiacetazone and Thiocarlide were also subjected to screening procedure in order to validate the docking protocol.

In Silico ADME Prediction

The absorption, distribution, metabolism and elimination are crucial properties for the clinical success of a drug candidate. Nearly 50% of the drugs fail because of poor ADME properties.^{26,27} Advances have been made in the *in silico* prediction of ADME properties of molecules, which resulted in a number of programs available for predicting several parameters/descriptors. Predicting ADME properties reduces animal testing, reduces cost and time and provides reliable toxicity prediction. In the present study, the ADME properties were predicted using Swiss ADME.²⁸

RESULTS AND DISCUSSION

The three-dimensional crystal structure of the *Mycobacterium tuberculosis* PKS-13 thioesterase domain (PDB ID: 5V3W), with resolution 1.70 Å was retrieved from RCSB Protein Data Bank. The protein structure was prepared using AutoDockTools (ADT) integrated within PyRx.²⁹ Preparation involved removal of co-crystallized water molecules, heteroatoms, and any pre-existing ligands. Polar hydrogen atoms were added, and Kollman charges were assigned to the protein structure. The prepared protein was subsequently converted to PDBQT format, which incorporates atomic charges and atom type definitions required for AutoDock Vina calculations.

The coumestan scaffold was submitted as a query molecule in SMILES format to the SwissSimilarity server. Compounds exhibiting similarity scores above 0.7 were retained, resulting in 157 coumestan derivatives with conserved pharmacophoric features. The structures were converted to 3D format (SDF) using Open Babel.³⁰

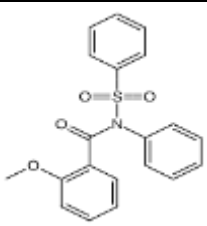
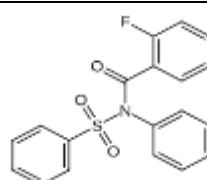
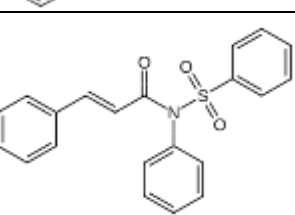
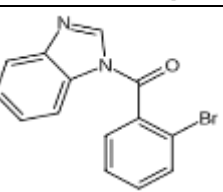
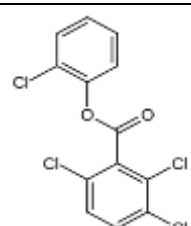
Virtual Screening

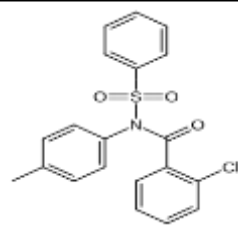
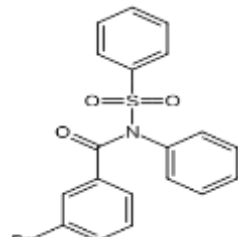
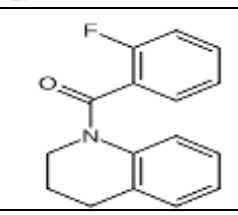
The 157 coumestan derivatives obtained from similarity screening were subjected to ligand preparation module in PyRx. Energy minimization was performed using the Universal Force Field (UFF) to optimize molecular geometries and achieve energetically favorable conformations.³¹ Steepest descent algorithm was used for optimisation of the ligands for 200 steps. All the ligands were converted to PDBQT format, ensuring proper assignment of rotatable bonds, Gasteiger charges, and atom types essential for accurate binding affinity predictions. For each ligand, 7–8 conformers were generated to account for structural flexibility during docking. The CastP predicted active site residues are SER1533, ASP1560, ALA1561, VAL1562, TYR1582, PHE1585, PHE1590, TYR1637, ASN1640, ILE1643, TYR1663, TYR1674 HIS1699 and PHE1670. The active site residue information was taken into consideration while generating the grid box around the protein during the docking protocol. The grid dimensions in angstroms are X-32.8109, Y-27.0559, Z-25.0000, with center at X-11.2101, Y-27.9723, Z-16.5511. The exhaustiveness is 8 and number of poses per ligand are 8. Autodock vina module in PyRx, version 0.8 was used for carrying out the docking experiment.

The resultant docked protein ligand complexes were ranked based on their docking scores, which reflect the predicted binding free energy. The protein-ligand complexes were then subjected to visual inspection of binding poses, focusing on the orientation, interactions within the binding pocket (e.g., hydrogen bonds, hydrophobic interactions), and potential key residues involved in ligand binding using Discovery Studio.³² The top 8 protein ligand complexes with the most favorable docking scores are given in table1. The residues TYR1582, PHE1585, GLY1633, TYR1637, PHE1670 and ARG1717 have been identified to have important binding interactions with the ligands. These residues interacting with the ligands are also corroborated with the predicted active site residues of CastP. The docking scores were found to be in the range of -7.3 to -8.9 kcal/mol. The molecule M3 exhibited highest docking score of -8.9 kcal/mol.

In order to validate our docking experiment, we have re-docked the protein 5v3w with the standard anti-TB drugs Isoniazid, Ethionamide, Thiacetazone and Thiocarlide. The docking scores obtained were -5.9, -5.5, -7.2 and -7.5 Kcal/mol respectively. Docking with co-crystal ligand 2Q5, ((2R)-2-[(2R)-2-[(2S)-2-[(2R)-2-oxidanylpropoxy]propoxy]propoxy]propan-1-ol) resulted in a binding score of -5.8 Kcal/mol. These results show that the coumestan derivatives reported in the present study have better binding affinity than the standard drugs which further support the hypothesis that they can be promisable leads for tuberculosis therapy. The only disadvantage here is that the standard drugs act on a different protein in the same mycolic acid synthesis pathway. Currently there are no clinical candidates available which act on PKS-13 protein in order to validate on the same protein. Therefore, we carried out the validation of docking experiment using the co-crystal of the protein and the currently available standard drugs used for tuberculosis treatment.

Table 1: Ligands showing docking score with intermolecular interactions and bond distance in Angstroms (Å)

S.No.	Structure	Docking score (ΔG-Kcal/Mol)	Intermolecular Interactions	Bond Distance(Å)
M1		-7.5	Pi-bonds M1:TYR1582 M1:PHE1670 M1:GLY1633	3.877 4.696 3.916
M2		-7.8	M2:TYR1637	4.725
M3		-8.9	M3:PHE1585	5.379
M4		-7.7	M4:TYR1582	3.908
M5		-7.6	M5:TYR1582	3.845

M6		-7.3	M6:ARG1717	2.216
M7		-8.4	M7:TYR1582 M7:GLU1633	3.890 3.882
M8		-8.0	M8:TYR1582	4.256

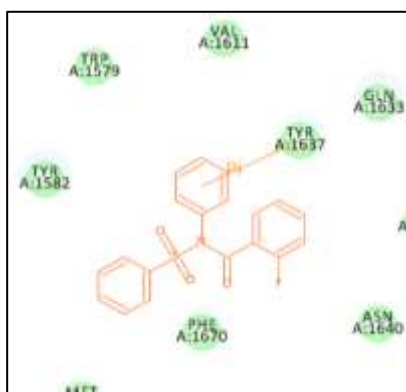
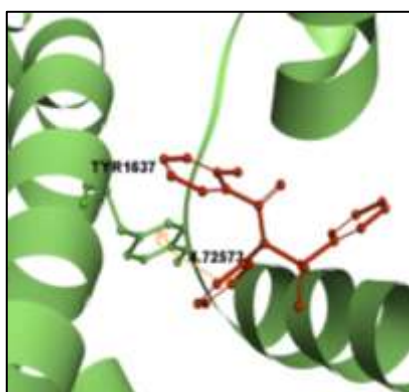
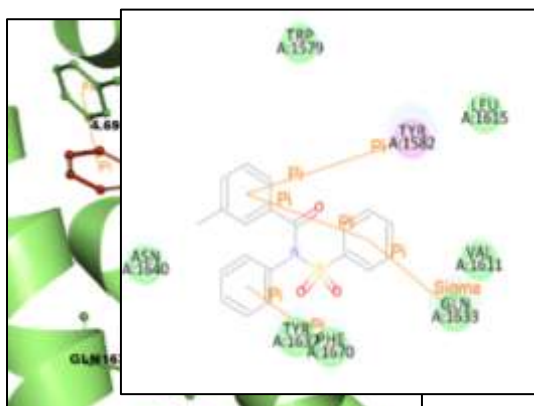


Fig 4 2D and 3D view of docking interactions between the protein PKS-13 (PDB ID: 5V3W) with the ligands M1 and M2 obtained after virtual screening, prioritised based on docking score. The protein is represented as green colored flat ribbon model and the ligands are represented in red color ball and stick model. The hydrogen bonds are represented as black dotted lines and pi-pi interactions as orange color solid lines. The distances are in Angstrom units (Å). The left side figures represent 3D interactions and figures on the right side represent 2D interactions of the PKS-13 protein (PDB ID: 5V3W) with the ligands.

ADME Analysis

The evaluation of ADME properties is an essential part of drug discovery to minimize failures of drug candidates in clinical phases. The top-ranked compounds were subsequently subjected to *in silico* ADME profiling using SwissADME to assess their drug-likeness and pharmacokinetic properties. Predicted ADME results of the top scoring 8 molecules are given in Table 2. The various physicochemical properties such as molecular weight, donor & acceptor hydrogen bonds, TPSA, Lipinski's rule of five, etc., were found to be in acceptable range. The predicted ADME results of the molecules suggest that they can be optimized further and support their potential for experimental validation through biochemical and pharmacological studies.

Table 2: SwissADME Predicted ADME values of the top scoring docked molecules for PKS-13 protein

Mol.no.	iLog P	Mol wt. (g/mol)	H-bond Donor	H- bond Acceptor	TPSA	GI-absorption	Lipinski rule of 5	PAINS
M1	3.49	367.42	0	4	72.06	High	Yes0 violation	0 alert
M2	3.81	355.38	0	4	62.83	High	Yes0 violation	0 alert
M3	3.81	363.43	0	3	62.83	High	Yes0 violation	0 alert
M4	3.23	301.14	0	2	34.89	High	Yes0 violation	0 alert
M5	4.99	336	0	2	26.3	High	Yes0 violation	0 alert
M6	4.27	385.86	0	3	62.83	High	Yes0 violation	0 alert
M7	4.11	416.29	0	3	62.83	High	Yes0 violation	0 alert
M8	3.39	255.29	0	2	20.31	High	Yes0 violation	0 alert

CONCLUSION

The present study was designed for the identification of potential Polyketide synthase-13 (PKS-13) inhibitors, using computational techniques such as similarity screening, molecular docking, and *in silico* ADME profiling. Polyketide synthases, particularly PKS-13, is essential for the cell wall synthesis of Mycobacterium tuberculosis. Similarity screening helped in identifying potential leads for PKS-13 using coumestan scaffold as the template. The docking process resulted in identifying promising inhibitors for PKS-13, with scores ranging from -7.3 to -8.9 kcal/mol. The molecules exhibited good binding site interactions with the residues of the protein. The hits were further screened for their *in silico* ADME profiles. The ligands M3, M7 and M8 exhibited docking scores -8.9, -8.4 and -8.0 kcal/mol respectively and acceptable ADME properties which emphasise their role as promising inhibitors for PKS-13. The docking experiment was further validated using the standard drugs, Isoniazid and Ethionamide and the docking results showed that the coumestan derivatives used in the present study comparatively have better binding affinity than the standard drugs. Therefore, the results indicate the molecules reported in the present study can be promising leads for TB therapy. These molecules can be optimised further and carried forward for studies such as *in vitro* and *in vivo* pharmacological and toxicological studies to validate their activity and efficacy.

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Conflict of interest

The author report that there are no competing interests to declare.

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