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Combination of QSAR and structure-based drug design approaches to study the inhibition of Fyn kinase signaling pathway

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The CD36 receptor plays a significant role in different stages of atherosclerosis pathology. The binding of β-amyloid on this receptor induces the activation of a signaling cascade responsible to the inflammatory response stimulating and the development of atherosclerotic plaque. This signal transduction pathway is mediated by two tyrosine kinase members of the Src family, Fyn and Lyn also MAP kinase and p44/42 thus the interruption of this signaling cascade by chemical inhibitors leading to the inhibition of the inflammatory response of macrophages to the β-amyloid and therfore the treatment of atherothrombotic disorders. High-performance bioinformatics and chemo-informatics tools were used in this work to study the inhibition of Fyn kinase in terms of structure-activity and structure-selectivity relationships. The present work consists of two parts; the first part is devoted to the Quantitative Structure-Activity Relationship (QSAR) study of Fyn kinase inhibitors. This study involves three main steps: The calculation of the descriptors, the construction of a QSAR model and the model validation. In the second part, we performed molecular docking to analyze the interaction between Fyn kinase in DFG-in and DFG-out conformations and their inhibitors. The obtained results have helped us to suggest an efficient scaffold of Fyn kinase inhibitor.

Biography

Zineb Tarhda is currently pursuing PhD in Mohammed V University, Morocco.

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