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RESEARCH ARTICLE

Synthesis, Structural Characterization, Catalytic, Biological and α -Glucosidase Inhibitory Studies of Metal Complexes with Flavone Derivatives

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ABSTRACT

Heterocyclic compounds, in particular oxygen-containing heterocyclic compounds, are of special interest to medicinal chemists because of their unusual biological properties. In the present study, the highly conjugated nitrogen heterocyclic scaffold comprised of flavone derivative with metal acetates to form metal chelates of the type $[M^{II}L(OAc)_2]$, flavone analogues (L); $M=Co^{2+}$, Zn^{2+} , Cu^{2+} and Ni^{2+} . The above title compounds were characterized using composition analysis of spectroscopic techniques. Based on spectroscopic and measurements confirmed that square planar arrangements for the Co²⁺, and Ni²⁺ complexes. Antimicrobial efficacy of prepared complexes were assessed against A.flavus, A.niger, B.subtilis, E. coli, C. albicans and S.aureus. The antimycobacterial (H₃₇Rv) efficacy of flavone analogues and its complexes screened MABA approach compared with using and standard. The acetylcholinesterase (AChE) inhibitory effect of the ligand was examined to therapeutic efficiency of compound in the neurodegenerative disorders. The synthesized ligand exhibited selective inhibition (AChE & BuChe) values (IC₅₀ : 0.20 (flavone analogue), 2.41 (Rivastigmine) and (Galantamine), respectively. Further, the in vitro anti-inflammatory efficiency of metal chelates were performed with the help of egg albumin method. The α -glucosidase inhibition activity was also carried out prepared metal complexes.

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Introduction

emergence of multidrug resistant pathogens novel antimicrobial exposed the emerging need for multi interactions. The agents with target acetylcholine (neurotransmitter) is essential for the treatment of memory and learning in neurodegenerative and the lower level of patients Alzheimer's disease (AD).

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Acetylcholinesterase (AChE) as a neurotransmitter which modulates the acetylcholine and other esters of choline and terminates essential brain functions [Cheung J, Rudolph MJ, Burshteyn F, 2012]. It plays an imperative action in the formation of fibrils through the aggregation of amyloids [Sonmez F, Zengin Kurt B, Gazioglu I, 2017; Tripathi RKP, M Sasi V, Gupta SK, 2018; Soyer Z, Uysal S, Parlar S, 2017; Ali AE, Elasala GS and Ibrahim RS, 2019]. The primary therapeutic strategies for anti-AD to reduce the speed of denaturation of