Synthesis, Characterization, Superoxide Dismutate Scavenging and Cytotoxic Activities of Cu(II) and VO(II) Complexes Incorporating Water Soluble Pyridine and Benzimidazole Ligands

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The modeling studies that mimic the active sites of metalloenzymes are important for understanding the reaction mechanism of the metalloproteins and for developing small molecular weight biomimetic catalysts as superoxide dismutase (SOD) mimics [1]. Examples of such complexes include several mononuclear derivatives with different types of pharmacological activity [2,3]. Although the mechanistic aspects of O$_2^-$ dismutation by metal ions and complexes is very limited, it has been established that the metal ion must be capable of being both oxidized and reduced by superoxide [4]. As a part of a research project devoted to the synthesis and characterization of copper(II) complexes with pharmacological activity [3], we have now prepared a new water soluble ligand, namely bis(2-benzimidazolylmethyl-6-sulfo)amine $L_2$ based on the water insoluble bis(2-benzimidazolylmethyl)amine $L_1$ [5]. This ligand was used for the synthesis of two Cu(II) and VO(II) complexes [L$_2$Cu(H$_2$O)$_2$] 1 and [L$_2$VO(H$_2$O)$_2$] 2, in which their general physicochemical properties were investigated. These model complexes represent suitable functional model of the enzyme SOD. Their nuclease–like catalytic activity were also investigated under aerobic conditions at room temperature and in the absence of any external additives.

The obtained experimental observations demonstrated that the copper complex 1 has promising ability towards the cleavage of the genomic DNA. To further elucidate the anticancer ability of the reported copper(II) complex 1, the cytotoxicity assay was performed using Caco-2 colon cancer cell line. The obtained results indicate that copper(II) complex 1 has good anti-cancer activity. Its easy preparation and the good solubility in water make it a promising anticancer drug. Further investigations of the in vivo studies are in need to clarify the real antiproliferative mechanism of these biocatalytic systems.

REFERENCES