Criteria 3- Research, Innovations and Extension (110)

Key Indicator 3.3- Research Publication and Awards (25)

3.3.1 Number of research papers published per teacher in the Journalsnotified on UGC care list during the last five years

3.3.1.1. Number of research papers in the Journals notified on UGCCARE year wise during the last five years

Year	2017-18	2018-19	2019-20	2020-21	2021-22
Number	00	00	02	01	00

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RESEARCH ARTICLE

Design and Molecular Docking Studies of Some 2, 3 DI-Substituted Quinazolin-4-One Analogues Against Staphylococcus Aureus UDG

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Abstract: ***Background*:** In this present investigation, some 2, 3 disubstituted-quinazolin-4-one derivatives are designed and docked against chain A and chain B of (3WDF) receptor.

***Methods*:** The heterocyclic fused rings quinazolinone have drawn a great attention owing to their expanded applications in the field of pharmaceutical chemistry. The diverse range of molecules with quinazoline/quinazolinone moieties have been reported to exhibit a broad spectrum of biological activities.

***Results and Conclusion*:** The results designate that the quinazolinone ring forms hydrophobic and hydrogen bond contacts with ASN 127 A, ALA 126 A, and SER 83 B, SER 183 B amino acid residue.

Keywords: Docking, dock score, conformer, protein, ligand, quinazolinone.

1. INTRODUCTION

Quinazoline and quinazolinone scaffolds represent a very important category of biologically active nitrogen heterocyclic compounds. The spread of marketed medicine has supported these moieties. A diverse range of molecules with quinazoline/quinazolinone moieties are reportable to exhibit a broad spectrum of biological activities. Their easy synthetic accessibility and flexibility in structural modifications and fictionalization add more to their appeal in medicinal chemistry [1-9].

1.1. Staphylococcus Aureus UDG (3WDF)

Staphylococcus aureus is the protein that acts as an uracil DNA glycosylase inhibitor, also named as SAUGI. The SAUGI has a high binding affinity towards both s.aureus and human UDG. The SAUDG has a functional role in DNA repair and host defence [45].

2. MOLECULAR DOCKING

Molecular docking is the method that involves inserting molecules in appropriate configurations to interact with a receptor. In molecular modeling, the term "molecular docking" refers to the study of two or more molecular structures fit together. The molecular docking helps in the identification of the Ligands correct Binding geometry (Pose) within the binding site (Binding mode) and Prediction of the binding affinity (Scoring Function). The two ways available for docking are rigid docking. The internal geometry of each of the receptor-associated ligand are treated as rigid and flexible docking wherever an enumeration on the rotation of one of the molecule (usually smaller one) is performed. For each rotation the energy is calculated and the latter the foremost optimum pose is selected [38, 39, 42].

3. MATERIALS AND METHODS

3.1. Hardware and Software

All Molecular docking studies were performed using the Molecular design Suit (VLife MDS software, a demo version from VLife Sciences, Pune, India).

3.2. Structure Conformer Generation

Structure of the compounds was drawn by using the 2D structure application VLife2D draw and 2D structures were regenerated into 3D structures. All the 3D structures are optimized to reduce the energy of molecules. Conformers for every structure were generated by applying systemic force field methodology and also the least energy conformer is chosen for more studies [38-44] Figs. (1-4).

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RESEARCH ARTICLE

PHARMACOLOGICAL EVALUATION OF NEURO PROTECTIVE EFFECT OF FERULIC ACID IN ANIMAL MODEL OF NEUROPATHY

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ABSTRACT

Aim: Pharmacological evaluation of neuro protective effect of Ferulic acid in animal model of neuropathy. ***Materials and Methods:*** Diabetes was induced in rats by intraperitoneal injection of single dose of STZ (60 mg/kg). Neuropathic pain was assessed in diabetic rats by pin prick method, cold allodynia and hot plate method. Pain was developed at 4th week. At the end of experiment animals were scarified and biochemical changes (Lipid peroxidation, SOD, reduced glutathione and CAT content) in sciatic nerve were evaluated. Animals were treated with Ferulic acid doses (50, 100 and 150 mg/kg i.p) for 4th week. ***Results:*** Treatment with Ferulic acid at doses of 50,100 and 150 mg/kg significantly restored the reduced body weight, food, water and elevated blood sugar level. Further the drug Ferulic acid showed dose dependent reduction in pain threshold tested by mechanical, cold and thermal hyperalgesia. The level of lipid peroxidation, reduced glutathione, SOD and CAT content was significantly prevented. The blood serum level on sodium, potassium, urea, uric acid and creatinine content was significant prevented. ***Conclusion:*** The result of present study suggests the antidiabetic, antioxidant and neuroprotective property of Ferulic acid in laboratory animals.

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INTRODUCTION

One of the major concerns with uncontroled diabetes is the development of microvascular complications such as neuropathy, cardiovascular, nephropathy, retinopathy and erectile dysfunction. Among these complications, symptoms of diabetic neuropathy have been observed to emerge at the early stages. These symptoms include hyperalgesia (exaggerated response to non-noxious stimuli) and allodynia (low threshold pain stimuli) have commonly been reported in diabetic patients (Bril, 2012). Streptozotocin is one of the most common complications of diabetes affecting more than 50% patient with diabetes. Streptozotocin (STZ) induced diabetic rat model has been widely used to mimics insulin-dependent diabetes mellitus and a number of abnormalities (Thiticornpong, 2011). A single does of STZ leads to the development of hyperglycemia, after three weeks pain was developed in rats which are similar to those observed in patients with painful diabetic neuropathy. Treatment of DN is always a challenging and expensive task. It beings with optimizing glycemic control first and then associated pain. As oxidative stress is an ancillary player in DN, compounds with antioxidant property can be used as supplement with the conventional treatment. Based on the above assumption the present study was designed (Hosseini and Mahammad, 2013).

Ferulic acid1 (FA), or 4-hydroxy-3-methoxycinnamic acid, is one of the most abundant HCAs in Nature. It was first isolated in 1866 by Hlasiwetz and Barth, from the plant Ferula foetida (Apiaceae family). Nowadays, FA is a well-known and well-studied compound, with many applications in the industry and as a phytochemical. As stated previously, ingestion of secondary metabolites through dietary intake is one of the most significant forms of human consumption of these substances (Judy *et al.*, 2003). As such, it is important to consider the distribution of FA in products present in human nourishment. FA can be found throughout the plant kingdom as a ubiquitous component of plants' tissues, particularly as a constituent of their cells' walls. Therefore, it is only natural it is widely present in foodstuffs, namely grains, fruits, and vegetables, but it can also be found in beverages such as coffee and beer (Yamaguchi *et al.*, 2006).

MATERIALS AND METHODS

Drug and Chemicals: Streptozotocin (PubChem CID-29327) (Sigma-aldrich chemical Pvt. Ltd., USA.), acetone(PubChem CID-180) (Sigma), TBA (Pubchem CID-2723628) (Sigma), TCA (PubChem-6421) (Modern Science Apparatus Pvt. Ltd.), DTNB (PubChem CID-6254) (Modern Science.), Pet ether 60-

Effect of Vanillic Acid on Nerve Conduction Velocity in Chronic Constriction Injury Model of Neuropathy

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ABSTRACT

Background: Neuropathic Pain (NP) is less or symptomatically managed by presently available therapeutics. Therefore developing more effective drugs with minimum adverse effects is essential. Vanillic acid is phenolic secondary plant metabolite. Extensive research regarding phenolic acids with antioxidant, free radical scavenging and neuroprotective roles have been published. **Objectives:** The aim of this undertaken study was to evaluate the efficacy of vanillic acid (V.A.) to improve nerve conduction velocity in neuropathic pain induced by CCI (chronic constriction injury) and to evaluate its antioxidant potential. **Methods:** Rats were divided into 7 groups ($n=6$), as negative control, positive control (CCI), sham control, CCI+gabapentin (300 mg/kg, p.o.), V.A. (25 mg/kg, p.o.), V.A. (50 mg/kg, p.o.) and V.A. (100 mg/kg, p.o.). After surgery oxytetracycline (25 mg/kg, i.m.) was administered in animals to avoid any infection. Vanillic acid and gabapentin administered post-surgery from day 4th till 28th day. Velocity of nerve conduction and antioxidant and histopathological studies were conducted on 28th day. **Results:** Repeated oral administration of vanillic acid (50 mg/kg, 100 mg/kg) significantly improved MNCV. V.A. showed antioxidant property by significantly elevating level of GSH and also reversed histopathological changes induced by CCI. **Conclusion:** This study has suggested antioxidant and neuroprotective effect of vanillic acid in CCI induced peripheral neuropathy.

Key words: CCI, MNCV, Neuropathy, Gabapentin, Vanillic acid.

INTRODUCTION

Neuropathic Pain (NP) is initiated or caused by neuronal injury or functional disabilities in the nervous system.[1] NP is arising from damage to nerve due to tumors, diabetic neuropathy, herpes zoster, complex regional pain syndrome, AIDS, hypoxia.etc.[2] NP majorly affects quality of life of patients and has a great economic and social impact. It is reported by the institute of medicines that millions of American adults usually suffer from chronic pain and 17.9% suffer from neuropathic pain.[3] NP is multifactorial causing impairment in nerve function. The pathophysiology of pain is complex and involves central and peripheral pathways viz. neurotransmitter release, alteration in expression of ion channels and pain pathway.[4] It is known that both hyperalgesia and allodynia coexist in both, inflammatory and neuropathic pain.[5] Physiological stress caused by metabolic disorders, various inflammatory responses, viral infections, direct neuronal trauma, diseases like cancer or use of chemotherapeutic drugs and primary neurological diseases leads to neuronal functional disabilities and damage resulting into NP. Pain may be triggered by even any non-specific, small intensity stimulus, as neuronal injury changes neurophysiology to the long extent. These neuronal changes leads to over-expressions of ion channels and/or neuronal receptors generating abnormal action potentials and such synaptic transmission can result in

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