ISSN: 0976-3376

Available Online at http://www.journalajst.com

ASIAN JOURNAL OF SCIENCE AND TECHNOLOGY

Asian Journal of Science and Technology
Vol. 07, Issue, 07, pp.3143-3144, July, 2016

RESEARCH ARTICLE

STUDIES ON THE ANTI-INFLAMMATORY ACTIVITY OF SCHIFF BASE DERIVED COMPOUNDS

[1]Mandalogu Shashikala *[2]Arunadevi, P., [2]Sarangapani, M. and [1]Prasad, M.S.K

[1]Department of Biochemistry, Kakatiya University, Telangana, Warangal
[2]University College of Pharmaceutical Sciences, Kakatiya University, Telangana, Warangal

ARTICLE INFO

Article History:
Received 15th April, 2016
Received in revised form
26th May, 2016
Accepted 17th June, 2016
Published online 30th July, 2016

Key words:
Schiffs bases,
Anti-inflammatory,
Carragenean,
Ruthenium, Copper.

ABSTRACT

Two Schiff bases were synthesized from raceacetophenone: 1) ADS1: 4-ethyl-6-{(E)-1-[(3-nitrophenyl) imino] ethyl} benzene-1, 3-diol and 2) ADS3: 4-ethyl- 6-{(E)-1-[(2-nitrophenyl) imino] ethyl} benzene-1, 3-diol. Then their metal complexes were formed. The metals ions selected for the synthesis of new complexes were Ruthenium and Copper. These complexed Schiff based derived compounds were evaluated for antiinflammatory activity by carragenean induced ratpaw odema method in rats.

INTRODUCTION

The inflammatory process is the response to an injurious stimulus. It can be evoked by a wide variety of noxious agents (*e.g.,* infections, antibodies, or physical injuries). The ability to mount an inflammatory response is essential for survival in the face of environmental pathogens and injury; in some situations and diseases, the inflammatory response may be exaggerated and sustained without apparent benefit and even with severe adverse consequences. No matter what the initiating stimulus, the classic inflammatory response includes calor (warmth), dolor (pain), rubor (redness), and tumor swelling). Inflammatory responses occur in three distinct temporal phases, each apparently mediated by different mechanisms: (1) an acute phase characterized by transient local vasodilation and increased capillary permeability; (2) a delayed, subacute phase characterized by infiltration of leukocytes and phagocytic cells; and (3) a chronic proliferative phase, in which tissue degeneration and fibrosis occur. Many mechanisms are involved in the promotion and resolution of the inflammatory process. Although earlier studies emphasized the promotion of migration of cells out of the microvasculature, recent work has focused on adhesive interactions, including the E-, P-, and L-selectins, intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), and leukocyte integrins, in the adhesion of leukocytes and platelets to endothelium at sites of inflammation. Activated endothelial cells play a key role in "targeting" circulating cells to inflammatory sites. Expression of the adhesion molecules varies among cell types involved in the inflammatory response. Cell adhesion occurs by recognition of cell-surface glycoproteins and carbohydrates on circulating cells due to the augmented expression of adhesion molecules on resident cells. Thus, endothelial activation results in leukocyte adhesion as the leukocytes recognize newly expressed L-selectin and P-selectin; other important interactions include those of endothelial-expressed E-selectin with sialylated Lewis X and other glycoproteins on the leukocyte surface and endothelial ICAM-1 with leukocyte integrins.

It has been proposed that some, but not all, tNSAIDs may interfere with adhesion by inhibiting expression or activity of certain of these cell-adhesion molecules. Novel classes of anti-inflammatory drugs directed against cell-adhesion molecules are under active development but have not yet entered the clinical arena. In addition to the cell-adhesion molecules outlined above, the recruitment of inflammatory cells to sites of injury involves the concerted interactions of several types of soluble mediators. These include the complement factor C5a, platelet-activating factor, and the eicosanoid LTB_4. All can act as chemotactic agonists. Several cytokines also play essential

Corresponding author: Arunadevi, P.,
University College of Pharmaceutical Sciences, Kakatiya University, Telangana, Warangal

roles in orchestrating the inflammatory process, especially interleukin-1 (IL-1) and tumor necrosis factor (TNF), IL-1 and TNF are considered principal mediators of the biological responses to bacterial lipopolysaccharide (LPS, also called endotoxin). They are secreted by monocytes and macrophages, adipocytes, and other cells. Working in concert with each other and various cytokines and growth factors (including IL-8 and granulocyte-macrophage colony-stimulating factor; they induce gene expression and protein synthesis in a variety of cells to mediate and promote inflammation.

MATERIALS AND METHODS

Schiff bases are synthesized in the Department of Chemistry, Kakatiya University, Warangal were primarily tested earlier for their antiinflammatory activity. Antiinflammatory activity was carried out at University college of Pharmaceutical Sciences, Kakatiya University, Warangal, Telangana.

Experimental Method

Schiffbase were synthesised from the condensation of an amino acid with carbonyl compounds. These compounds and their metal complexes are very important as catalysts in various biological systems, polymers, dyes and medicinal and pharmaceutical fields. Some metal complexes were screened for anti-inflammatory activity by carragenean induced rat paw odema method.

Anti inflammatory test

The anti-inflammatory activity of the test compounds was evaluated in wistar rats weighing 120-140g employing the method of winter *et al*. (1963) and Diwan *et al*. (1989), male winstar rats were used for study. Animals were fasted overnight and divided into control, standard and different test groups 'each consisting of 3 animals. The different test compounds were administered to the animals in the test group at the dose of 100 mg/kg by oral route. The dose chosen is such that any activity is clearly noticeable in test compounds. Animals in the control group did not receive any drug. All results are related to control animal responses. Since the objective of this study is to develop secondary lead compounds based on diclofenac sodium. This drug was also administered in all groups for comparision of activity. Animals in the standard group received diclofenac sodium at the dose of 10 mg/kg by oral route. All these were administered as suspensions using sodium CMC as suspending agent. Control group of animals received a suspension of sodium CMC only. One hour after test drug administration, rats in all the groups were challenged with 0.1 ml of 1 % carragrenan in sub plantar region of left hind paw. A zero hour paw volume was measured for the rats using digital plethysmometer before the administration of carragrenan for all groups. Paw volumes were again measured at time interval of 3 hrs after the challenge of carragrenan. The percent inhibition of paw volume for each rat in treated groups was calculated by comparing with mean paw volume for each test group.

RESULTS AND DISCUSSION

The results obtained were presented in the table – 7. The activity of the compounds could be in the order of 3>4>5>6>1>2. All the compounds could show the activity at minimum dose tested 25mg/ug

Table 2. Anti-inflammatory screening data of the schiff base complexes

Compound	Dose (mg/kg)	Mean ±SEM
1	25	40.31 + 1.347
	50	30.5 + 1.3407
	100	25.5 + 0.3903
2	25	22.3+1.2172
	50	17.8+1.2172
	100	12.2 + 0.7233
3	. 2 5	30.0 + 0.527
	50	21.8 + 1.0386
	100	17.67 + 0.4513
4	25	24.3 ± 1.0453
	50	17.0 + 0.3333
	100	12.0 ±0.527
5	25	39.83 + 0.984
	50	39.83 + 0.984
	100	38.66+ 1.24
6	25	40.0 + 0.527
	50	40.33 ± 1.045
	100	39.5 + 0.391

Conclusion

The data suggest that compounds 3 & 4 even active. Compounds activity shown in the range o f 3 > 4 > 5 > 6 > 1 > 2.RuSp2 > $CuAL_7$> $CuAL_5$> $CuAL_6$>RuSPi> $RuSp_3$.

REFERENCES

Anant Prakash, Devjani Adhikari, 2011. *International Journal of Chem Tech. Research*, Vol.3, No.4, pp. 1891 – 1896, Oct – Dec, 2011.

Ashraf , M.A. Mahmood, K., Wajid, A. 2011. IP CBEE. 10, 1 – 7.

AshuChandhary, D.P. Jashli and R.V. Singh Metal Based Drugs, Vol. 8, No.6, 2002.

Fahim, N. and Singh, R.V. 1988. *Indian S.Chem*, 37A, 1126.

Fahmi, N., Saxena, C. and Sigh, R.V. 1996. *Bull. Chem. Soc. Jpn,* 69, 1.

RondlaRohini, KanneShanker, PuchakayalaMuralidhar Reddy, Yen – PengHo, VaddeRavinder, *European Journal of Medicinal Chemistry. Elsevier,* 3rd April, 2009, p. 1 – 10.

Samina Shahid, Muhammed Asam Raza and Shafiq-Ur-Rehman, 2009. *African Journal of Biotechnology* Vol.8 (19) pp. 5116 – 5121, 5th October.

Sekhon, B.S. and Leena Gandhi, 2006. Resonance April, 2006.

Sharma, K., Fahmi, N. and Singh, R.V., 2002. Main Group, Met, Chem, 25, 727.

Suslick, K.S., Reinest, T.J. 1988. *Chem – Edu, J.* 62, 1988, 974.

****