**Literature survey:**

1) **Taleley J.J. et al** \(^{13}\) reported synthesis of a series of substituted pyrazolyl benzenesulfonamides for the use of inflammation and inflammation related disorders.

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\text{R}^1=\text{SO}_2\text{NH}_2, \text{R}^2=\text{CF}_3, \text{R}^3=\text{H}, \text{R}^4=\text{Cl Ph} \\
\text{R}^2=\text{CF}_2\text{H}, \text{R}^3=\text{H}, \text{R}^4=\text{Cyclopentyl} \\
\text{R}^2=\text{CF}_2\text{CF}_3, \text{R}^3=\text{H}, \text{R}^4=\text{4-OCH}_3-2\text{-Napthyl}
\]

2) **Penning T.D. et al** \(^{14}\) reported the discovery of a series of sulfonamide-containing 1,5-diarylpyrazole and evaluated for their ability to inhibit cyclooxygenase-2 in-vitro and in-vivo.

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\text{R}^1=\text{H}, \text{R}^2=\text{-CF}_3, \text{R}^3=\text{-4-F}, \text{R}^2=\text{-CF}_3, \text{R}^1=\text{-4-CO\text{NH}_2}, \text{R}^2=\text{-CHF}_2, \\
\text{R}^1=\text{-4-OCH}_3, \text{R}^2=\text{-CHF}_2
\]

3) **Habeeb A.G. et al** \(^{15}\) reported design and synthesis of Celecoxib and Rofecoxib analogues as selective cyclooxygenase-2 inhibitors in which the respective SO \(2\) NH \(2\) and SO \(2\) CH \(3\) hydrogen-bonding pharmacophores were replaced by a dipolar azido bioisosteric substituent.
4) Shrikhande, A.A. et al\textsuperscript{16} reported heterocyclic compounds, diaryl pyrazoles, for their COX-2 inhibition.

5) Liu, Hong et al\textsuperscript{17} reported inhibitory mode of 1,5-diaryl pyrazole derivatives against cyclooxygenase-2 and cyclooxygenase-1 using molecular docking and 3D QSAR analyses. Structure based investigations and 3D QSAR provided possible guidelines and accurate activity predictions for novel inhibitor design.

6) Sarathy, K.P. et al\textsuperscript{18} reported QSAR study by Fujita-Ban model of some substituted α, β-Diaryl five-membered heterocycles as COX-1/COX-2 inhibitors. The results are indicative of selective inhibition of COX-2 by diarylimidazoles than diaryloxazolones and diarylpyrazoles.

7) Md. Jashim Uddin et al\textsuperscript{19} reported the a group of Celecoxib analogues in which the para-SO\textsubscript{2}NH\textsubscript{2} substituent on the N1-phenyl ring was replaced by a para-sulfonylazido (-SO\textsubscript{2}N\textsubscript{3}) or a meta-SO\textsubscript{2}N\textsubscript{3}, substituent were designed for evaluation as selective cyclooxygenase-2 (COX-2) inhibitors. A molecular modeling (docking) study showed that the SO\textsubscript{2}N\textsubscript{3} group inserts deep inside the secondary pocket of the COX-2 bindingsite. The SO\textsubscript{2}N\textsubscript{3} moiety undergoes a dual H-bonding interaction via one of its -SO\textsubscript{2} oxygen-atoms, and an electrostatic (ion-ion) interaction via the terminal azido (N\textsubscript{3}) nitrogen-atom, to the guanidino NH\textsubscript{2} of Arg513 in the secondary pocket of COX-2. These observations indicate that an appropriately positioned -SO\textsubscript{2}N\textsubscript{3} moiety is a novel alternative bioisostere to the traditional -SO\textsubscript{2}NH\textsubscript{2} and -SO\textsubscript{2}Me pharmacophores present in selective COX-2 inhibitors that are only capable of H-bonding interactions with the COX-2 isozyme.
8) Talley, J.J. et al\textsuperscript{20} reported synthesis of a potent and selective inhibitor of COX-2, 4-[5-methyl-3-phenylisoxazol-4-yl]benzene sulfonamide, Valdecoxib

Valdecoxib is a highly selective and potent inhibitor of COX-2 in human whole blood and against recombinant human enzyme. An active metabolite was also found to be a COX-2 selective inhibitor.

9. Vane, J.R. et al\textsuperscript{21} reported physiological and pathological functions of COX-1 and COX-2 on kidney.

Maintenance of normal kidney function is dependent of PGs both in animal models of disease states and in patients with congestive heart failure, liver cirrhosis, or renal insufficiency.

10. Purthi, R.S. et al\textsuperscript{22} reported the role of COX-2 activity and the potential clinical usefulness of COX-2 specific inhibitors to urological oncology and discussed the outcomes of the molecular mechanisms and clinical effects of COX-2 function and suggested that COX-2 specific inhibitors may serve as antitumor drugs with therapeutic and chemo preventive roles for urological cancers.

11. Emery, P.\textsuperscript{23} reported how COX-2 changes function of gastrointestinal system. Cyclooxygenase enzyme is reported to be involved in the reduction of gastric acid production, stimulate gastric fluid secretion, increase secretion of viscous mucosa and exert a direct vasodilator action on gastric mucosa.

12. Kunz, T. et al\textsuperscript{24} reported relation between COX-2 and Alzheimer’s disease. Cyclooxygenase-2 enzyme is reported to be involved in various nervous system disorders which include Alzheimer’s disease.

13. Kyrkandies, S. et al\textsuperscript{25} reported relation between COX-2 and Alzheimer’s disease. Cyclooxygenase-2 enzyme is reported to be involved in various nervous system disorders which include Alzheimer’s disease.
14. Aisen, P.S.\textsuperscript{26} reported relation between COX-2 and Alzheimer’s disease. Cyclooxygenase-2 enzyme is reported to be involved in various nervous system disorders which include Alzheimer’s disease.

15. Guo, J.S.\textit{et al}\textsuperscript{27} reported physiological and pathophysiological function of Cyclooxygenase on Blood Vessel’s. Cyclooxygenase enzymes are also expressed in blood vessels. For e.g. Protein expression of angiogenic factor during gastric ulcer healing.

16. Doret, M.\textit{et al}\textsuperscript{28} reported action of Cyclooxygenase-2 on Smooth Muscles. Enhanced expression of cyclooxygenase-2 enzyme is thought to be involved in the relaxation of smooth muscles of bronchial tissue.

17. Singh, P.\textsuperscript{29} reported Cyclooxygenase and Cancer interconnected to each other. Cox-2 have been detected in gastric and breast tumors.

18. Hull, M.A.\textsuperscript{30} reported Cyclooxygenase-2 as a target for cancer chemoprevention. COX-2 inhibitors are considered as attractive candidates for the chemoprevention.

19. Singh, G.\textit{et al}\textsuperscript{31} reported the role of cyclooxygenase-2 in breast cancer. COX-2 enzymes are also expressed in the breast cancer; that is they are involved in the pathogenesis of breast cancer.

20. Basler, J.W.\textit{et al}\textsuperscript{32} reported Cyclooxygenase-2 Selactive Inhibitors for Prostate Cancer Chemoprevention. COX-2 inhibitors are considered as attractive candidates for the chemoprevention.

21. Chell, S.\textit{et al}\textsuperscript{33} worked on COX-2 represent potential for the prevention/treatment of colorectal cancer. COX-2 inhibitors also play a promising role in the prevention of colorectal cancer.

22. Kawai, N.\textit{et al}\textsuperscript{34} studied Cyclooxygenase and colon cancer. It is also reported that COX-2 expression in intestinal epithelial cells increases resistance to apoptosis, promotes tumor angiogenesis, and enhances invasion and metastasis.

24. Bolli, R. et al and Mahadevan, U. et al\textsuperscript{36-37} reported COX-2 inhibitors (Coxibs) and cardiovascular actions. COX-2 inhibitors lack anti-platelet activity, coxibs are suited for the provision of cardiovascular prophylaxis and in patients at risk of myocardial infarction.

25. Catella, F. et al\textsuperscript{38} reported Cyclooxygenase inhibition and thrombogenicity. COX-2 inhibitor-treated patients with diseases that predispose to thrombosis should be monitored carefully for such type of complications.

26. Karim, S. et al\textsuperscript{39} reported physiological and pathological function of Endothelial cell and COX-2. Cyclooxygenase-2 has been demonstrated in human umbilical vein endothelial cells after induction by IL-1\textalpha and phorbol ester. During inflammation, the increased permeability of the vascular endothelium is caused by retraction of endothelial cells leading to exudation and migration of phagocytic cells.

27. Sengupta, S. et al\textsuperscript{40} reported COX-2 and gestation and parturiation. Both COX-1 and COX-2 are expressed in the uterine epithelium at different times and may be involved in implantation of the ovum and angiogenic processes of placenta formation.

28. Cai, Q. et al\textsuperscript{41} reported COX-2 alter the function of Vascular smooth muscle. Induction of COX-2 has been demonstrated in vitro in arterial smooth muscle cells treated with platelet-derived growth factor.