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Review on: Molecular Docking-Based Screening of Diclofenac and its Derivatives for Cox-2 Inhibitors

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Abstract: *Cyclooxygenase-2 (COX-2), also known as prostaglandin-endoperoxide synthase 2, catalyses the conversion of arachidonic acid to prostanoid precursors and is an important pharmacological target for pain, fever and inflammation. Diclofenac is a widely used phenylacetic-acid derivative non-steroidal anti-inflammatory drug (NSAID) that inhibits both COX isoforms, with a preferential COX-2 profile reported under some assay conditions. Its therapeutic use is limited by gastrointestinal, renal, hepatic and cardiovascular safety concerns. Structure-based molecular docking provides a means of examining ligand orientation in the COX-2 active-site channel, mapping residue contacts and prioritising related compounds for further study. The diclofenac–COX-2 crystal complex (PDB 1PXX) provides an experimentally observed structural reference. Published structural analyses describe polar contacts involving the diclofenac carboxylate and Tyr385/Ser530, together with hydrophobic contacts in the central and proximal regions of the channel. Studies of designed diclofenac derivatives, including work using human COX-*

2 structure PDB 5F19, have reported more favourable predicted docking scores than the parent drug. Other computational investigations combine docking with molecular dynamics (MD), MM/GBSA calculations and ADMET prediction. These approaches can strengthen early-stage prioritisation but do not establish enzyme inhibition, isoform selectivity or clinical safety. This review summarises COX-2 structure and pharmacology, diclofenac binding, docking methodology, reported derivative studies, validation, complementary computational methods, ADMET considerations and major limitations. A reproducible workflow based on appropriate receptor selection, consistent ligand preparation, redocking validation and cautious interpretation is proposed. Experimental COX-2 and COX-1 assays remain essential to confirm activity and selectivity.

Keywords: Diclofenac; cyclooxygenase-2; COX-2; non-steroidal anti-inflammatory drugs; molecular docking; AutoDock Vina; redocking; molecular dynamics; MM/GBSA; ADMET; virtual screening.

I. INTRODUCTION

Non-steroidal anti-inflammatory drugs are widely used to relieve pain, fever and inflammation. Their principal mechanism involves inhibition of cyclooxygenase enzymes, which convert arachidonic acid into prostaglandin H2 (PGH2), the precursor of prostaglandins, prostacyclin and thromboxane. These prostanoids participate in inflammatory responses as well as normal physiological processes. COX-1 is commonly associated with constitutive functions such as gastric mucosal protection and platelet activity, whereas COX-2 is inducible in many inflammatory settings. This distinction is not absolute: COX-2 also has physiological roles in selected tissues, and COX-1 may contribute to inflammation [1–3].

Diclofenac is a phenylacetic-acid derivative containing two aromatic rings, chlorine substituents and a carboxylic acid group. It is generally classified as a non-selective or preferential COX inhibitor rather than a highly selective coxib. Its relative COX-1/COX-2 activity depends on assay conditions and biological context. Although diclofenac is clinically

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useful, gastrointestinal ulceration and bleeding, renal effects, hepatic injury and cardiovascular risk are important safety considerations, particularly with systemic exposure and prolonged use [2,4,5]. These concerns have encouraged research into diclofenac-based analogues that may retain anti-inflammatory activity while improving physicochemical or predicted safety properties.

Molecular docking is a structure-based computational method used to sample possible ligand orientations and conformations within a protein binding site and rank the resulting poses using a scoring function. It can support virtual screening and structure optimisation at relatively low cost. However, a docking score is not a measured binding free energy, IC50 or Ki. A credible study therefore requires structural validation, consistent preparation and interpretation alongside other computational and experimental evidence [6–9]. This review integrates two supplied review documents into a concise synthesis focused on diclofenac and its derivatives as candidate COX-2 inhibitors.

II. COX-2 STRUCTURE AND THE DICLOFENAC-BINDING SITE

COX enzymes are membrane-associated heme proteins with a cyclooxygenase channel and a peroxidase site. NSAIDs inhibit the cyclooxygenase function by occupying the substrate/inhibitor channel and interfering with productive access or positioning of arachidonic acid. The channel is elongated and largely hydrophobic, with a constriction region near its entrance and deeper pockets that accommodate different inhibitor scaffolds. Residues frequently discussed in NSAID recognition include Arg120 and Tyr355 near the channel entrance, and Tyr385, Ser530, Phe518, Leu352, Val349, Trp387 and Met522 in deeper regions [1,2].

COX-1 and COX-2 share a similar overall fold but differ at selected amino-acid positions that influence pocket geometry. In particular, the COX-2 side pocket is associated with Arg513 and a smaller residue at position 523 (Val523 in COX-2 compared with Ile523 in COX-1). This structural feature can be exploited by some selective COX-2 inhibitors, including coxibs. Nevertheless, occupancy of the COX-2 channel or side pocket alone does not prove selectivity; a comparative COX-1 assessment is needed [2,6].

The diclofenac–COX-2 crystal structure, PDB 1PXX, is a useful structural anchor. Published analyses describe a binding orientation in which the carboxylate group is directed toward Tyr385 and Ser530, while the aromatic rings occupy hydrophobic regions of the channel. Reported central-pocket contacts include Leu352, Leu384, Tyr385, Trp387, Met522 and Gly526; contacts around the chlorine-bearing ring include Val349, Val523, Ala527, Ser530 and Leu531 [1,2]. These residues are useful landmarks for checking docked poses, but the distances and interaction types should be recalculated from the actual prepared structure used in each study.

A contact diagram should not be interpreted as proof of energetic importance. Hydrogen-bond and hydrophobic-contact labels depend on geometric cut-offs and software settings. Reports should identify the analysis program, distance criteria, ligand functional group and residue involved, and whether an interaction is present in a single pose or persists during simulation.

III. CHEMICAL FEATURES AND PHARMACOLOGICAL CONTEXT OF DICLOFENAC

Diclofenac contains an acidic carboxyl group, two aromatic rings and chlorine substituents. The carboxylate can contribute to polar or ionic recognition, while the aromatic rings and chlorine atoms influence hydrophobic fit and shape complementarity within the COX channel. At physiological pH, the carboxyl group is expected to be predominantly deprotonated; therefore, ligand preparation should state whether the neutral acid or ionised carboxylate was modelled. These representations should not be mixed without justification [2,10].

The distinctive diclofenac pose differs from the common constriction-anchored binding mode described for several other NSAIDs. This makes the native 1PXX pose especially useful for checking whether a docking protocol can reproduce a known diclofenac orientation. For comparative screening, diclofenac may be assessed alongside a non-selective NSAID such as ibuprofen or indomethacin and a selective COX-2 inhibitor such as celecoxib or etoricoxib. Comparisons are meaningful only when receptor preparation, ligand treatment, search space and scoring protocol are kept consistent [2,6–8].

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IV. MOLECULAR DOCKING METHODOLOGY

A typical docking workflow begins with selection and inspection of a suitable COX-2 structure, followed by receptor preparation, ligand preparation, binding-site definition, docking, pose analysis and validation. Human COX-2 is preferred when the intended interpretation concerns human pharmacology. PDB selection should consider species, isoform, resolution, completeness, chain, biological assembly, co-crystallised ligand, heme status, missing residues and active-site geometry. PDB 1PXX is particularly relevant to diclofenac because it contains the drug in a COX-2 complex; PDB 5F19 has been used in a representative diclofenac-derivative design study [2,11,12].

4.1. Receptor preparation:

The selected structure should be inspected before removing waters, ions, cofactors or co-crystallised ligands. COX-2 is a heme-associated enzyme, so the treatment of heme and other heteroatoms should be explicitly justified. The biologically relevant chain should be selected, local structural gaps examined, and hydrogens and charges assigned using a documented procedure. The original PDB should be retained as an unmodified audit copy [2,11].

4.2. Ligand preparation:

Diclofenac and comparator structures can be obtained from reliable chemical databases such as PubChem. Compound identity, bond orders, formal charges, stereochemistry where relevant, tautomeric state and protonation should be checked. Three-dimensional conformers should be generated and geometry-optimised using a stated method. The chosen diclofenac form—neutral acid or carboxylate—must be reported [10].

4.3. Binding-site and grid defination:

The docking grid should cover the known inhibitor channel and be centred using the co-crystallised ligand or a clearly justified active-site definition. The grid centre coordinates and box dimensions should be reported. A grid that is too small may exclude valid poses, while an unnecessarily large grid can reduce search efficiency. The same grid and search parameters should normally be used for compounds in a direct comparison [7–9].

4.4. Docking and scoring:

Commonly used programs include AutoDock4, AutoDock Vina, Glide and GOLD. AutoDock4 uses a force-field-based scoring approach, whereas Vina uses an empirical scoring function and efficient conformational search [7,8]. Scores from different programs are not directly interchangeable. Within-study comparisons are more defensible when compounds are prepared and docked using the same receptor, software version and parameters. A favourable score should be considered together with pose plausibility, interactions, ligand strain and predicted ADMET properties—not as a stand-alone measure of potency [6–9].

V. EVIDENCE FROM DICLOFENAC-DERIVATIVE STUDIES

A representative structure-based design study published in 2021 used human COX-2 (PDB 5F19; reported resolution 2.24 Å) to compare diclofenac with six designed derivatives. The supplied review reports use of PyRx 0.8 and a Lamarckian Genetic Algorithm workflow, with ligand energy minimisation and selection of the best-ranked conformation. The reported docking scores were −7.2 kcal/mol for diclofenac and −7.8, −8.1, −8.3, −8.4, −8.5 and −8.6 kcal/mol for derivatives D-1 through D-6, respectively. D-6 was the best-ranked member of that series and was reported to form hydrogen-bond and hydrophobic contacts involving residues including Arg120, Tyr355 and Ser530 [13]. These are computational results from one study and should not be interpreted as experimentally measured inhibition or clinical benefits. Other work described in the supplied review documents explores several strategies for modifying the diclofenac scaffold. A 2024 computational study combined docking, molecular dynamics, MM/GBSA and pharmacokinetic prediction for novel diclofenac-derived compounds. Another reported set of derivatives docked against COX-2 PDB 5IKR with Glide scores in the approximate range of −10.07 to −10.66 kcal/mol, compared with approximately −9.3 kcal/mol for diclofenac in that study. Schiff-base, thiadiazole-containing and phenylalanine-containing derivatives have also been investigated using docking and, in some cases, synthesis or biological evaluation [14–17].

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The reported score ranges across studies cannot be pooled into a single ranking because different receptor structures, ligand preparations, software packages, scoring functions and protocols were used. A more negative score within one consistent study may support prioritisation, but it does not demonstrate greater enzyme inhibition. Similarly, predicted improvements in ADMET properties do not establish reduced adverse effects in humans. The available evidence supports the value of combining docking with complementary methods and experimental testing, rather than relying on score comparisons across unrelated studies.

VI. KEY PROTEIN–LIGAND INTERACTIONS

The literature identifies several recurring COX-2 residues relevant to NSAID recognition. Arg120 and Tyr355 lie near the channel entrance and can contribute to ligand anchoring or orientation for some NSAID scaffolds. Tyr385 and Ser530 are frequently discussed in relation to the distinctive diclofenac binding orientation and polar contacts with its carboxylate. Hydrophobic residues such as Leu352, Val349, Val523, Ala527, Trp387 and Phe518 contribute to packing of aromatic portions of ligands. The exact contacts depend on ligand structure, orientation, receptor conformation and the analysis criteria [1,2,13].

For diclofenac specifically, the reported crystallographic pose places its carboxylate toward Tyr385 and Ser530, with aromatic portions occupying central and proximal hydrophobic regions. Arg120 and Tyr355 should not be assumed to form obligatory interactions in every diclofenac pose. Likewise, the presence of contacts with the COX-2 side pocket does not, by itself, establish isoform selectivity. A rigorous interaction analysis should report distances, angles where relevant, residue numbering, software and cut-offs, and should distinguish crystallographic observations from newly predicted contacts.

VII. VALIDATION AND INTEGRATION WITH COMPLEMENTARY METHODS

Redocking is a central validation step. It involves removing the co-crystallised ligand, preparing it for docking and attempting to reproduce its experimentally observed pose in the same receptor structure. The predicted and crystallographic poses are superimposed and heavy-atom root-mean-square deviation (RMSD) is calculated after appropriate atom mapping. An RMSD around or below 2 Å is commonly used as a practical pose-reproduction benchmark, although ligand symmetry, atom correspondence and protocol details affect interpretation [6,13,18]. A successful redocking result shows that the method can recover one known pose under the chosen conditions; it does not prove accurate ranking of unrelated compounds, potency prediction or COX-2 selectivity.

A robust validation plan may also include known active/reference compounds, cross-docking into another suitable COX-2 structure, modest grid-sensitivity checks, alternative plausible protonation states and, where justified, comparison with experimental inhibition data. The study should report software and version, receptor and ligand preparation, grid coordinates and dimensions, exhaustiveness or search settings, number of poses, random seed where available, atom mapping and RMSD method. Negative scores alone are not evidence of successful validation [6–9].

Molecular dynamics simulations can provide information about time-dependent stability of a selected protein–ligand complex. Common analyses include protein and ligand RMSD, residue RMSF, hydrogen-bond persistence and key contact behaviour. MM/GBSA or MM/PBSA calculations may provide an additional approximate estimate of relative binding free energy and can be used to re-rank selected complexes [14,18]. These methods remain model-dependent and do not replace experimental binding or enzyme assays. MD should be applied to a justified shortlist rather than indiscriminately to every screened compound.

VIII. ADMET AND SAFETY CONSIDERATIONS

ADMET describes absorption, distribution, metabolism, excretion and toxicity. Tools such as SwissADME, pkCSM and ADMETlab can provide early estimates of solubility, intestinal absorption, permeability, plasma-protein binding, distribution, metabolic liabilities, clearance and selected toxicity alerts. Drug-likeness descriptors such as molecular weight, lipophilicity and topological polar surface area may also help prioritise compounds [10,14–17].

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These predictions are screening aids, not measured pharmacokinetic or toxicological results. A predicted CYP interaction is not proof of a clinical drug interaction, and the absence of a toxicity alert does not establish safety. Diclofenac has recognised gastrointestinal, renal, hepatic and cardiovascular risks; docking to COX-2 alone cannot predict or eliminate these outcomes. Claims that a derivative is "safer" should therefore be avoided unless supported by appropriate experimental evidence. The most defensible description is that a compound has a favourable *predicted* profile that warrants further evaluation.

IX. LIMITATIONS AND FUTURE PERSPECTIVES

Molecular docking uses simplified scoring functions and finite conformational sampling. Common workflows treat the receptor as rigid or only partially flexible, and may incompletely represent protein dynamics, solvent rearrangement, entropic contributions and coupled protonation effects. Different software packages can produce different poses and scores for the same ligand–receptor pair [6–9]. COX-2 presents additional challenges because the channel and side-pocket geometry can vary with ligand scaffold and receptor conformation. Heme and local water handling may influence the prepared system. A visually plausible pose or stable MD trajectory from a selected starting pose does not prove a correct binding mechanism [1,2].

Future work may benefit from ensemble docking using multiple receptor conformations, more systematic protonation and receptor-preparation sensitivity analysis, and multi-objective optimisation that considers affinity, selectivity and ADMET together. Machine-learning-assisted screening and advanced free-energy methods may improve prioritisation, but their value depends on appropriate training data, validation and experimental feedback. If selectivity is a stated objective, matched docking or experimental assessment against COX-1 and COX-2 is necessary. Ultimately, enzyme inhibition assays, cellular anti-inflammatory models and appropriate safety studies are required before therapeutic claims can be made.

X. RECOMMENDED REPRODUCIBLE WORKFLOW

For a diclofenac-focused B.Pharm or research screening project, the following sequence provides a transparent framework: (1) select a human COX-2 structure appropriate to the question and document PDB ID, chain and resolution; (2) inspect the native ligand, heme, waters, missing residues and active-site geometry; (3) prepare the receptor while recording all heteroatom decisions; (4) prepare diclofenac and comparator ligands with justified protonation states and optimised geometry; (5) define the grid from the native ligand or a documented active-site definition; (6) redock the native ligand and report RMSD with atom mapping and pose superposition; (7) dock diclofenac and selected reference compounds using consistent parameters; (8) inspect poses and report residue-level contacts with analysis criteria; (9) apply ADMET prediction and, if justified, MD/MM-GBSA to a small shortlist; and (10) state limitations and propose experimental COX-2 and COX-1 assays.

At minimum, the final report should include raw docking output, all selected poses, interaction diagrams, exact software versions and parameters, grid coordinates and dimensions, ligand identifiers and protonation states, validation results and the rationale for selecting compounds. Scores must be reported as outputs of the specified scoring function, not as experimental binding energies.

XI. CONCLUSION

The diclofenac–COX-2 crystal complex provides an experimentally observed structural benchmark for understanding diclofenac recognition and evaluating docking protocols. Published structural evidence describes polar contacts involving the diclofenac carboxylate and Tyr385/Ser530, alongside hydrophobic packing in the COX-2 channel. Computational studies of diclofenac derivatives report more favourable predicted docking scores for selected analogues and illustrate the value of integrating docking with molecular dynamics, free-energy estimation and ADMET prediction. However, differences in structures and computational protocols prevent direct numerical comparison across studies, and predicted scores or ADMET profiles cannot establish potency, selectivity or clinical safety. A reliable

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screening study should use an appropriate receptor, consistent ligand preparation, redocking validation, transparent interaction analysis and cautious interpretation. Experimental COX-2 inhibition testing, matched COX-1 assessment and subsequent pharmacological and safety evaluation remain essential steps toward identifying useful diclofenac-based anti-inflammatory candidates.

Table 1. Important COX-2 residues relevant to diclofenac and NSAID recognition

Residue / region	Reported relevance	Interpretation in docking
Arg120	Channel entrance; can anchor acidic groups in some NSAID poses	Check geometry; do not assume an obligatory salt bridge for diclofenac
Tyr355	Channel entrance and ligand orientation	Assess hydrogen-bond/contact geometry where present
Tyr385 / Ser530	Reported polar contacts with diclofenac carboxylate in 1PXX	Compare orientation and distances with the crystal pose
Leu352, Leu384, Trp387, Met522, Gly526	Central hydrophobic pocket contacts	Inspect aromatic packing and shape complementarity
Val349, Val523, Ala527, Leu531	Proximal pocket contacts around the chlorine-bearing ring	Inspect steric fit and ring orientation
COX-2 side pocket	Structural feature exploited by some coxibs	Pocket occupancy alone does not prove COX-2 selectivity

Table 2. Representative docking results reported for diclofenac and derivatives

Study / system	Reported result	Important caution
2021 derivative series; COX-2 PDB 5F19	Diclofenac: −7.2 kcal/mol; D-1 to D-6: −7.8 to −8.6 kcal/mol; D-6 ranked highest	One study-specific scoring protocol; not measured enzyme inhibition
2024 diclofenac-derivative modelling; COX-2 PDB 5IKR	Reported Glide scores for selected derivatives: approximately −10.07 to −10.66 kcal/mol; diclofenac approximately −9.3 kcal/mol	Different receptor and scoring workflow; do not compare directly with Vina/PyRx scores
Other derivative series	Schiff-base, thiadiazole- and phenylalanine-containing analogues have been explored	Synthesis and biological evidence vary by study; verify primary papers before making efficacy claims

Table 3. Minimum information for reproducible docking reports

Component	Information to report
Receptor	PDB ID, species, isoform, chain, resolution, missing residues, heme/water/ion treatment
Ligand	Name, database identifier, structure, protonation/charge, conformer and optimisation method
Docking	Software/version, scoring function, grid centre and dimensions, search settings, number of poses, random seed if available
Validation	Native ligand, atom mapping, RMSD method, pose overlay and reference-compound behaviour
Interaction analysis	Software, distance/angle criteria, residues and persistence across poses or MD frames
ADMET / MD	Tool and version, endpoints, force field, solvent, ions, equilibration and simulation duration
Reproducibility	Date, operating system, parameter files, commands/GUI steps and raw output files

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