WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 15, 1685-1710. Research Article ISSN 2277–7105

# EXPLORING QUINOLINE SCAFFOLDS THROUGH INSILICO TECHNIQUE FOR THE DRUG DEVELOPMENT

**Rachel Mathew[1], Vani V.[2], Snuja Maria Saji*[3], Anchu Raj[4]**

[1,2,*3,4]Department of Pharmaceutical Chemistry, Mar Dioscorus College of Pharmacy, Hermongiri Vidyapeetam, Alathara, Sreekariyam, Thiruvananthapuram.

Article Received on
21 June 2025,

Revised on 10 July 2025,
Accepted on 30 July 2025,

DOI: 10.20959/wjpr202515-37856

***Corresponding Author**
**Snuja Maria Saji**
Department of
Pharmaceutical Chemistry,
Mar Dioscorus College of
Pharmacy, Hermongiri
Vidyapeetam, Alathara,
Sreekariyam,
Thiruvananthapuram.

## ABSTRACT

Quinoline and its derivatives exhibit potent pharmacological properties, including antimalarial, antimicrobial, anticancer, and anti-inflammatory activities. This review outlines their chemistry, biosynthesis, and identification—highlighting quinine from *Cinchona* bark. It discusses therapeutic applications, resistance challenges, and drug design innovations, underscoring quinoline's pivotal role in modern medicinal chemistry. In this study, ten quinoline analogues were designed using ChemSketch, and evaluated for drug‑likeness using Molinspiration, biological activity via PASS, and pharmacokinetic/toxicity profiles through ADMET‑AI. Molinspiration analysis indicated that most analogues meet Lipinski's criteria, with QA5 and QA10 showing moderate deviations in lipophilicity. PASS predictions for gluconate 2‑dehydrogenase inhibition identified QA1, QA2, and QA7 as having very high activity ($Pa > 0.9$), with QA2 being the most potent ($Pa = 0.975$). ADMET‑AI predicted excellent intestinal absorption and high bioavailability for QA1, QA2, QA7, QA8, and QA10; however, QA4, QA5, and QA6 exhibited lower oral bioavailability. Distribution profiles suggested strong blood–brain barrier penetration and high tissue distribution for QA1, QA2, QA5, QA7, and QA8. Metabolic analysis flagged potential CYP1A2 inhibition for QA1, QA3, QA4, QA5, and QA7. Toxicity concerns were highest for QA5 and QA9 (mutagenicity, hepatotoxicity, carcinogenicity), whereas QA2, QA8, and QA10 demonstrated comparatively safer profiles. Overall, QA2 emerges as the most promising candidate, balanced by high predicted efficacy and favorable ADMET/toxicity.

**KEYWORDS**: Natural products, Quinoline Alkaloids, Medicine, Cinchona.

## NATURAL PRODUCTS

## 1. INTRODUCTION

Natural products include chemical substances synthesized by plants or animals, or chemical substances found in nature with particular pharmacological effects. They are classified into three categories: primary metabolites, secondary metabolites, and polymeric molecules. Primary metabolites are intermediates of catabolic and anabolic pathways that occur in plants, and are required for plant survival. Plants medicinal value is conferred by secondary metabolites such as alkaloids, tannins, saponins, flavonoids, anthraquinones, glycosides, terpenes, essential oils, and resins. They form salts with acids and precipitate with phosphor tungstic acid, phosphor molybdic acid, picric acid, and mercury. Recently, the science of ethnobotany, which is the scientific study of the relationships between humans and plants, has been employed to understand the cultural concepts surrounding the perception of the chemistry of plants and natural products. Although, natural products are chemical compounds synthesized by living organisms, they are more commonly referred to as natural chemicals with medicinal properties. Natural products can be extracted from the tissues of terrestrial plants, marine organisms, or microorganisms. Leaves, flowers, twigs, barks, rhizomes, roots, seeds, and fruits are some of the plant parts that are commonly used to extract natural substances.

Natural products and their structural analogues have historically made a major contribution to pharmacotherapy, especially for cancer and infectious diseases. Nevertheless, natural products also present challenges for drug discovery, such as technical barriers to screening, isolation, characterization and optimization, which contributed to a decline in their pursuit by the pharmaceutical industry from the 1990s onwards. In recent years, several technological and scientific developments — including improved analytical tools, genome mining and engineering strategies, and microbial culturing advances — are addressed such challenges and opened up new opportunities. Here, we summarize recent technological developments that are enabling natural product-based drug discovery, highlight selected applications.[1]

## PROBLEMS WITH SYNTHETIC DRUGS

Health Risks, Lack of regulation, Environmental and Social Impact, Psycological and Social Impact.

## 2. CLASSIFICATION OF NATURAL PRODUCTS

### PRIMARY AND SECONDARY METABOLITES

Plants contain a wide variety of natural products that can be broadly categorized into primary and secondary metabolites.

Essential for the growth and development of all plant species, primary metabolites such as nucleotides, amino acids, and acyl lipids are omnipresent across the plant domain.[1] In sharp contrast, secondary metabolites are distinctive to particular taxonomic plant groups, fulfilling pivotal roles in growth and development, stress responses, and ecological adaptation.[2,3] More importantly, these secondary metabolites, also esteemed as important or valuable natural products, constitute a prolific source of medicines, nutraceuticals, flavors, cosmetics, and industrial raw materials, which have significant social and economic value and have attracted increasing research interest in recent years.[1] When it comes to the category of phytohormones, the opinions are controversial. On one hand, phytohormones are almost indispensable for every plant. On the other hand, several phytohormones are structurally related to other secondary metabolites and even share the same partial biosynthetic pathways.[4] We consider phytohormones as a unique group of small molecules, independent of primary and secondary metabolites.[2] [5] [8]

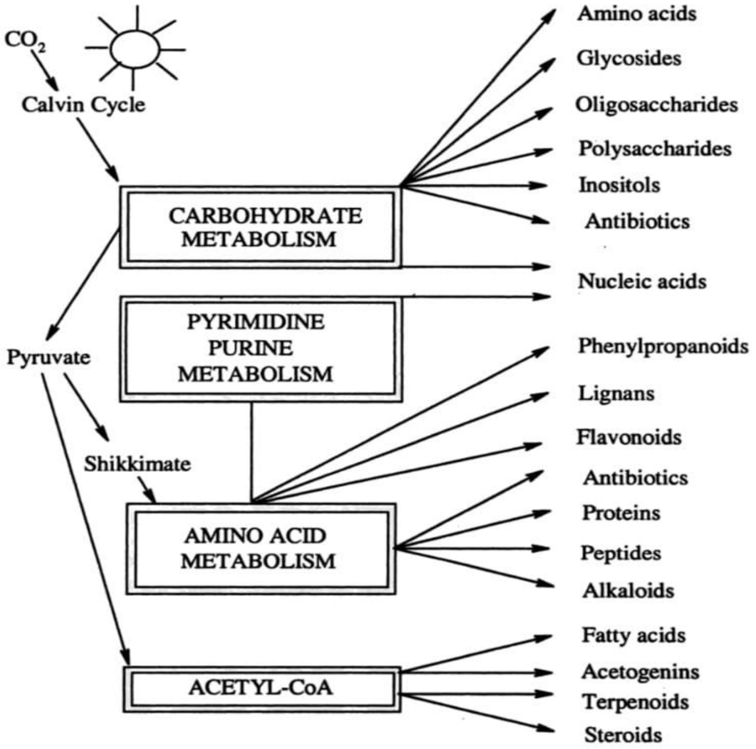


**Figure GI.1** Outline of biogenesis of natural products

## ALKALOIDS

Alkaloids are classified as being plant derived, pharmacologically active, basic compounds derived from amino acids that contain one or more hetrocyclic nitrogen atom. Alkaloids are a huge group of naturally occurring organic compounds which contain nitrogen atom or atoms (amino or amido in some cases) in their structures. These nitrogen atoms cause alkalinity of these compounds. These nitrogen atoms are usually situated in some ring (cyclic) system. For example, indole alkaloids are those that contain nitrogen atom in indole ring system. Generally based on structures, alkaloids can be divided into classes like indoles, quinolines, isoquinolines, pyrrolidines, pyridines, pyrrolizidines, tropanes, and terpenoids and steroids. Other classification system is connected with a family of plant species that they occur. Alkaloids in pure form are usually colorless, odorless, crystalline solids, but sometimes they can be yellowish liquids. Quite often, they have a bitter taste. These compounds are generally produced by many plant species, mainly by flowering plants, and also by some animals. Plants produce and store many organic compounds like amino acids, proteins, carbohydrates, fats, and alkaloids, which are usually treated as secondary metabolites. They are stored in each part of the plant—leaves, stem, root, and fruits of plants—but in different amounts.

Alkaloids are the most prevalent category of nitrogen-containing compounds in plants, with over 12,000 alkaloids identified, which have been extensively studied.[13] These bioactive compounds are pivotal as secondary metabolites, providing plants with a crucial defense mechanism against herbivores and pathogens. Additionally, alkaloids contain various nitrogenous heterocycles, which stand as the primary active constituents in numerous medicinal plants.[2]

## CLASSIFICATION OF ALKALOIDS

1. Taxonomical classification: This classification is based on the distribution of alkaloids in various plant families, like solanaceous or papilionaceous alkaloids. Some times they are grouped as per the name of grouped genus in which they occur, e.g. ephedra, cinchona, etc.
2. Biosynthetic classification: This method gives significance to the precursor from which the alkaloids are biosynthesized in the plant. Hence the variety of alkaloids with different taxonomic distribution and physiological activities can be brought under the same group, if they are derived from the same precursor, e.g. all indole alkaloids from tryptophan are

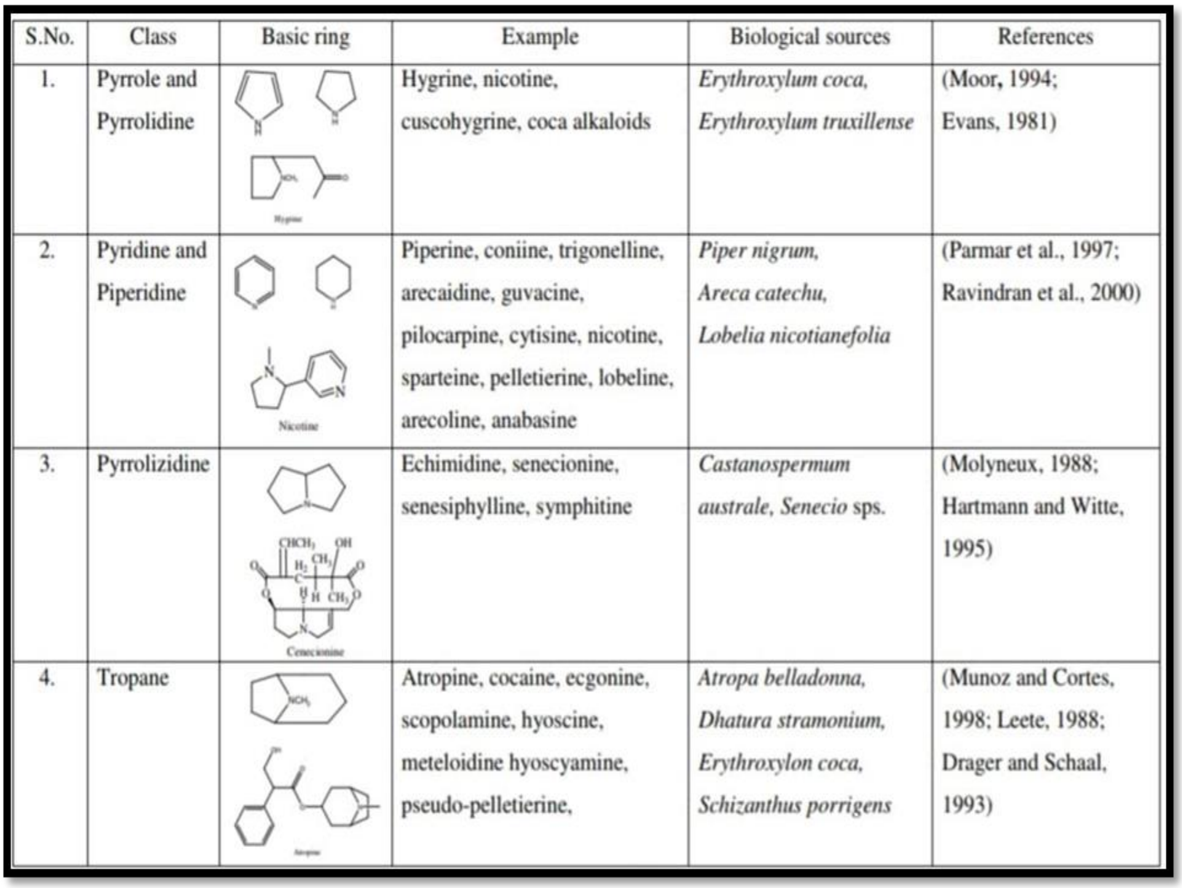
grouped together. Alkaloids derived from amino acid precursors are grouped in the same class, such as ornithine, lysine, tyrosine, phenylalanine, tryptophan, etc.

3. Pharmacological classification: This classification is based on the physiological action or biological activity of alkaloids on animals like CNS stimulants or depressants, sympathomimetics, analgesics, purgatives, etc.
4. Chemical nature of alkaloids. Within the same chemical structure the alkaloids can exhibits more than one physiological action e.g. morphine is narcotic-analgesic, while quinidine is cardiac depressant.
5. Chemical classification: this classification is most accepted way to specify the alkaloids. The alkaloids are categorised into three divisions.

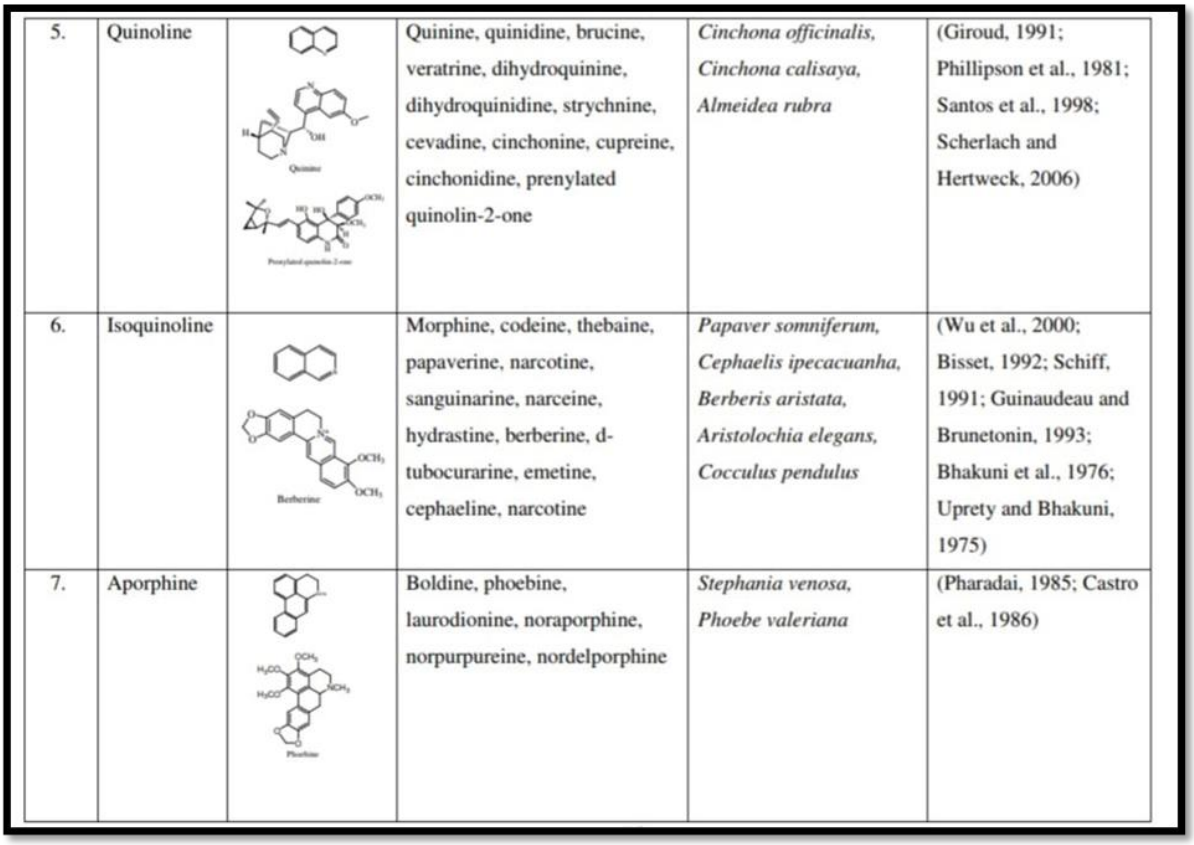
a. True alkaloids: These have a heterocyclic ring with nitrogen and are derived from amino acids.eg. Morphine, quinine.

b. Proto alkaloids: These does not have heterocyclic ring with nitrogen and derive from amino acids, e.g. colchicine.

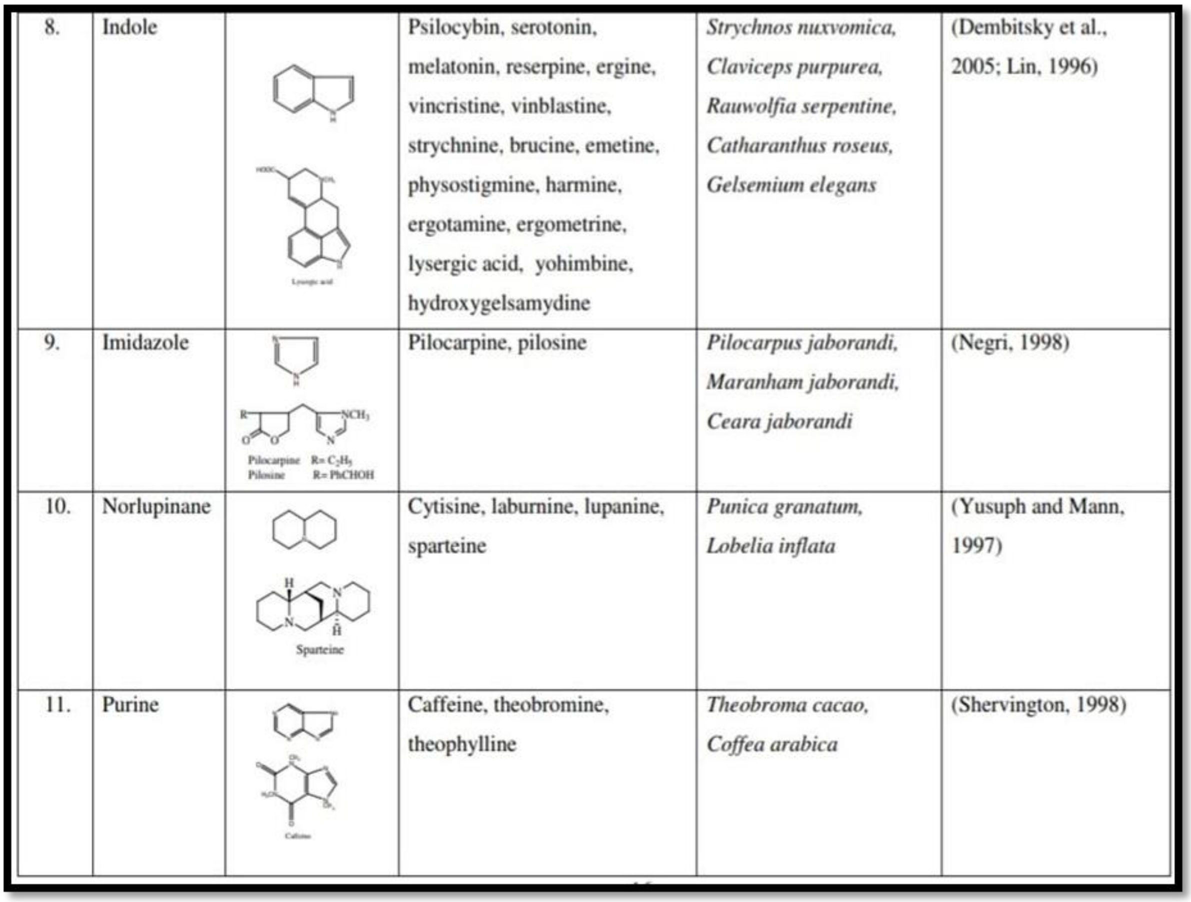
c. Pseudo alkaloids: These have heterocyclic ring with nitrogen and derived from terpenoids or purines but not derived from amino acids. eg. ephedrine, caffeine.[3]



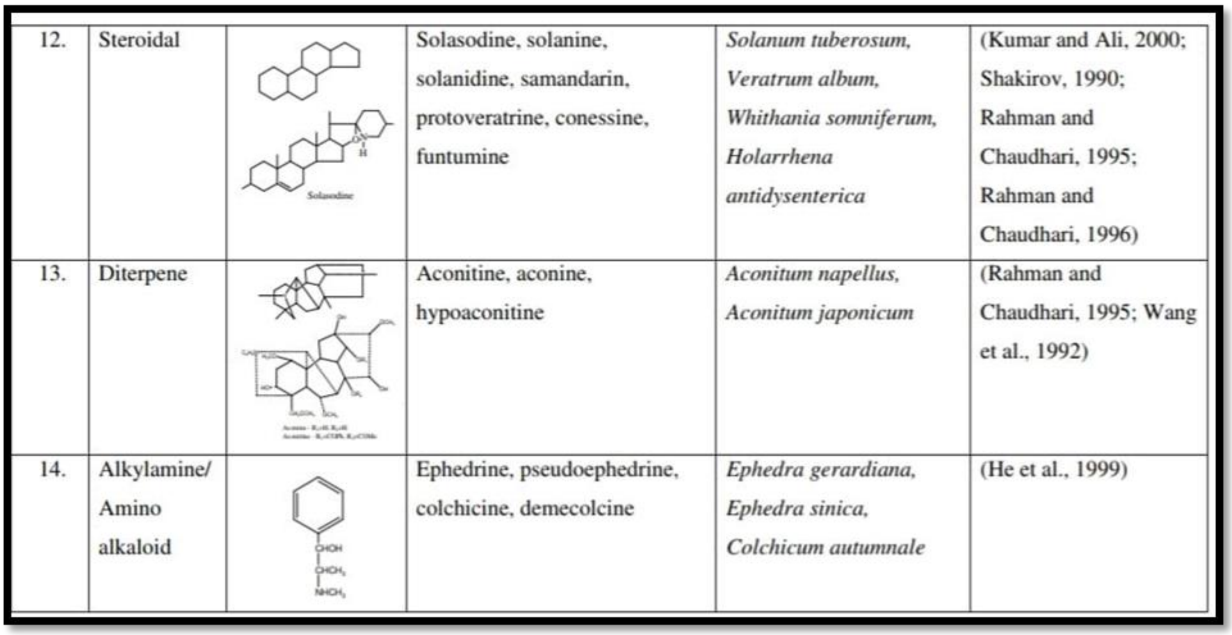
| S.No. | Class | Basic ring | Example | Biological sources | References |
|---|---|---|---|---|---|
| 1. | Pyrrole and Pyrrolidine | | Hygrine, nicotine, cuscohygrine, coca alkaloids | *Erythroxylum coca, Erythroxylum truxillense* | (Moor, 1994; Evans, 1981) |
| 2. | Pyridine and Piperidine | Nicotine | Piperine, coniine, trigonelline, arecaidine, guvacine, pilocarpine, cytisine, nicotine, sparteine, pelletierine, lobeline, arecoline, anabasine | *Piper nigrum, Areca catechu, Lobelia nicotianefolia* | (Parmar et al., 1997; Ravindran et al., 2000) |
| 3. | Pyrrolizidine | Cenecionine | Echimidine, senecionine, senesiphylline, symphitine | *Castanospermum australe, Senecio* sps. | (Molyneux, 1988; Hartmann and Witte, 1995) |
| 4. | Tropane | | Atropine, cocaine, ecgonine, scopolamine, hyoscine, meteloidine hyoscyamine, pseudo-pelletierine, | *Atropa belladonna, Dhatura stramonium, Erythroxylon coca, Schizanthus porrigens* | (Munoz and Cortes, 1998; Leete, 1988; Drager and Schaal, 1993) |



| | | | | | |
|---|---|---|---|---|---|
| 5. | Quinoline | Quinine<br>Prenylated quinolin-2-one | Quinine, quinidine, brucine, veratrine, dihydroquinine, dihydroquinidine, strychnine, cevadine, cinchonine, cupreine, cinchonidine, prenylated quinolin-2-one | *Cinchona officinalis, Cinchona calisaya, Almeidea rubra* | (Giroud, 1991; Phillipson et al., 1981; Santos et al., 1998; Scherlach and Hertweck, 2006) |
| 6. | Isoquinoline | Berberine | Morphine, codeine, thebaine, papaverine, narcotine, sanguinarine, narceine, hydrastine, berberine, d-tubocurarine, emetine, cephaeline, narcotine | *Papaver somniferum, Cephaelis ipecacuanha, Berberis aristata, Aristolochia elegans, Cocculus pendulus* | (Wu et al., 2000; Bisset, 1992; Schiff, 1991; Guinaudeau and Brunetonin, 1993; Bhakuni et al., 1976; Uprety and Bhakuni, 1975) |
| 7. | Aporphine | Phoebine | Boldine, phoebine, laurodionine, noraporphine, norpurpureine, nordelporphine | *Stephania venosa, Phoebe valeriana* | (Pharadai, 1985; Castro et al., 1986) |



| | | | | | |
|---|---|---|---|---|---|
| 8. | Indole | Lysergic acid | Psilocybin, serotonin, melatonin, reserpine, ergine, vincristine, vinblastine, strychnine, brucine, emetine, physostigmine, harmine, ergotamine, ergometrine, lysergic acid, yohimbine, hydroxygelsamydine | *Strychnos nuxvomica, Claviceps purpurea, Rauwolfia serpentine, Catharanthus roseus, Gelsemium elegans* | (Dembitsky et al., 2005; Lin, 1996) |
| 9. | Imidazole | Pilocarpine R= $C_2H_5$<br>Pilosine R= PhCHOH | Pilocarpine, pilosine | *Pilocarpus jaborandi, Maranham jaborandi, Ceara jaborandi* | (Negri, 1998) |
| 10. | Norlupinane | Sparteine | Cytisine, laburnine, lupanine, sparteine | *Punica granatum, Lobelia inflata* | (Yusuph and Mann, 1997) |
| 11. | Purine | Caffeine | Caffeine, theobromine, theophylline | *Theobroma cacao, Coffea arabica* | (Shervington, 1998) |



| | | | | | |
|---|---|---|---|---|---|
| 12. | Steroidal | | Solasodine, solanine, solanidine, samandarin, protoveratrine, conessine, funtumine | *Solanum tuberosum, Veratrum album, Whithania somniferum, Holarrhena antidysenterica* | (Kumar and Ali, 2000; Shakirov, 1990; Rahman and Chaudhari, 1995; Rahman and Chaudhari, 1996) |
| 13. | Diterpene | | Aconitine, aconine, hypoaconitine | *Aconitum napellus, Aconitum japonicum* | (Rahman and Chaudhari, 1995; Wang et al., 1992) |
| 14. | Alkylamine/ Amino alkaloid | | Ephedrine, pseudoephedrine, colchicine, demecolcine | *Ephedra gerardiana, Ephedra sinica, Colchicum autumnale* | (He et al., 1999) |

## QUINOLINE ALKALOIDS

Quinoline[1] or 1-aza-napthalene or benzo[b]pyridine is nitrogen containing heterocyclic aromatic compound. It has a molecular formula of C9H7N and its molecular weight is 129.16. The log P value is 2.04 and has an acidic pKb of 4.85and a basic pKa of 9.5. Quinoline is a weak tertiary base. It can form salt with acids and displays reactions similar to those of pyridine and benzene. It shows both electrophilic and nucleophilic substitution reactions. It is nontoxic to humans on oral absorption and inhalation.

Quinoline nucleus occurs in several natural compounds (Cinchona Alkaloids) and pharmacologically active substances displaying a broad range of biological activity. Quinoline has been found to possess antimalarial, anti-bacterial, antifungal, anthelmintic, cardiotonic, anticonvulsant, anti-inflammatory, and analgesic activity.

## IDENTIFICATION TEST OF QUININE

1. Thalleioquin Test (Green Fluorescence Test)

Procedure: Dissolve a small amount of quinine sulfate in dilute acetic acid.

Add a few drops of bromine water (or chlorine water).Then add excess ammonia solution.

Observation: A green fluorescence or emerald-green color appears.

Mechanism: The test is based on the oxidation of quinine, forming thalleioquin, which fluoresces green under ammonia.

2. Fluorescence Test (Under UV Light)

Procedure: Dissolve quinine in water or dilute sulfuric acid .Expose the solution to UV light (365 nm).

Observation: A strong blue fluorescence is observed due to the quinoline ring structure.

Note: This property is used in tonic water, which glows blue under UV light.

3. Acid-Sulfate Test

Procedure : Dissolve quinine in dilute sulfuric acid. Observe the solution under UV light.

Observation: Intense blue fluorescence.

Confirmation: The fluorescence disappears upon adding hydrochloric acid but reappears when neutralized with ammonia.

4. Mayer's Test (General Alkaloid Test)

Procedure: Add Mayer's reagent (potassium mercuric iodide solution) to a quinine solution.

Observation: A white or creamy precipitate forms, indicating the presence of an alkaloid.

5. Dragendorff's Test (General Alkaloid Test)

Procedure: Add Dragendorff's reagent (potassium bismuth iodide) to a quinine solution.

Observation: An orange-brown precipitate forms, confirming alkaloid presence.

6. Erythroquinine Test (Red Color Formation)

Procedure: Treat quinine with glacial acetic acid and concentrated sulfuric acid.

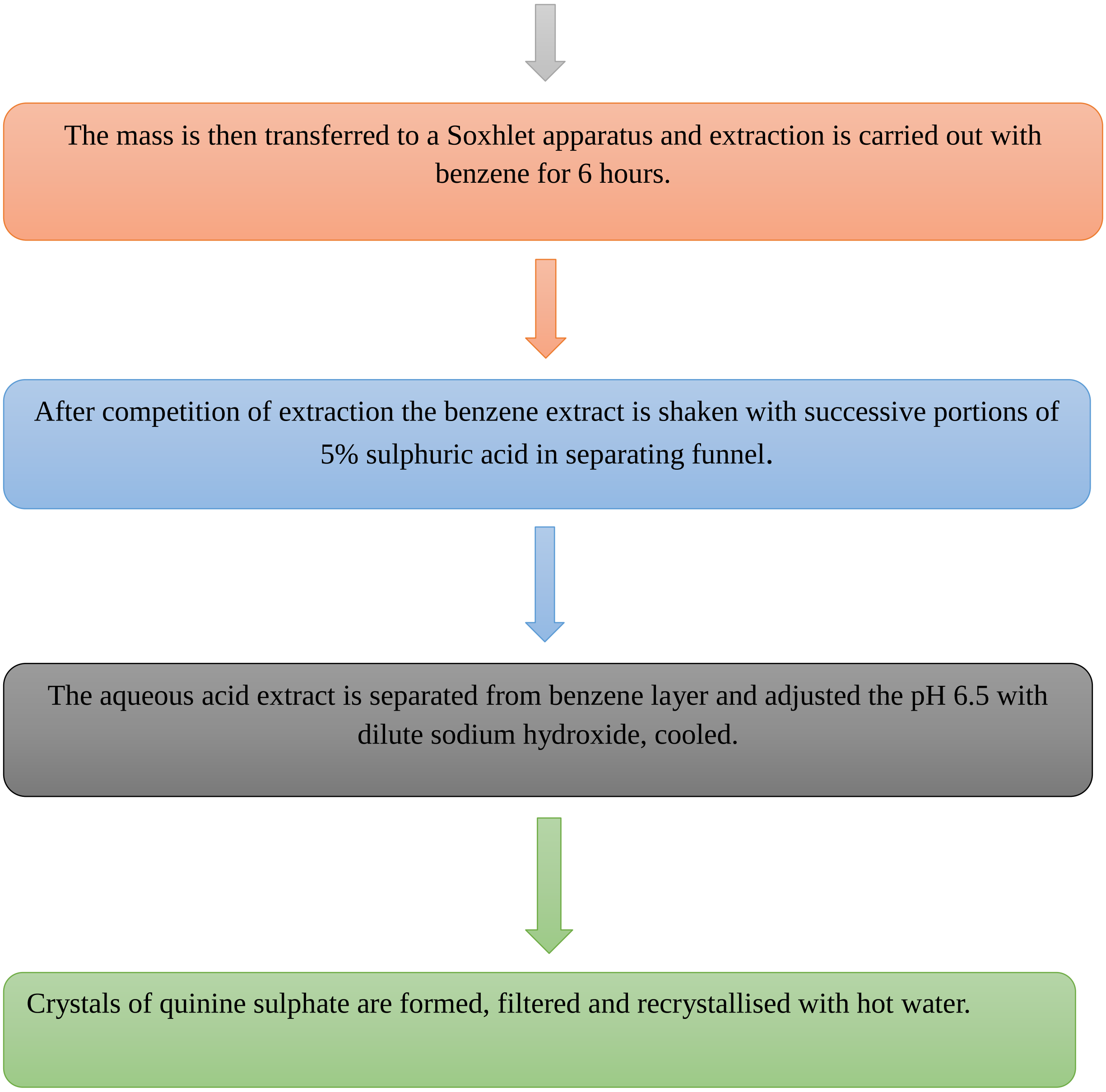
Observation: A red color develops (due to the formation of erythroquinine).[1] [2] [8]

## ISOLATION OF QUININE

Required quantity of dry powder bark material is first well mixed with about 30% of its weigh alcoholic calcium hydroxide or calcium oxide (20%) and sufficient quantity of Sodium hydroxide solution (5%) to make a paste.

↓

It is allowed to stand for few hours so that alkali can convert cinchona alkaloids to free bases.



The mass is then transferred to a Soxhlet apparatus and extraction is carried out with benzene for 6 hours.

After competition of extraction the benzene extract is shaken with successive portions of 5% sulphuric acid in separating funnel.

The aqueous acid extract is separated from benzene layer and adjusted the pH 6.5 with dilute sodium hydroxide, cooled.

Crystals of quinine sulphate are formed, filtered and recrystallised with hot water.

## USES

- Quinoline is used in the manufacture of dyes, the preparation of hydroxyquinoline sulphate and niacin.
- It is also used as a solvent for resins and terpenes.
- Quinoline is used as a solvent and reagent in organic synthesis
- Several anti-malarial drugs contain quinoline substituents. These include quinine, chloroquine, amodiaquine, and primaquine.
- Its principal use is as a precursor to 8-hydroxyquinolinewhich is a versatile chelating agent and precursor to pesticides.

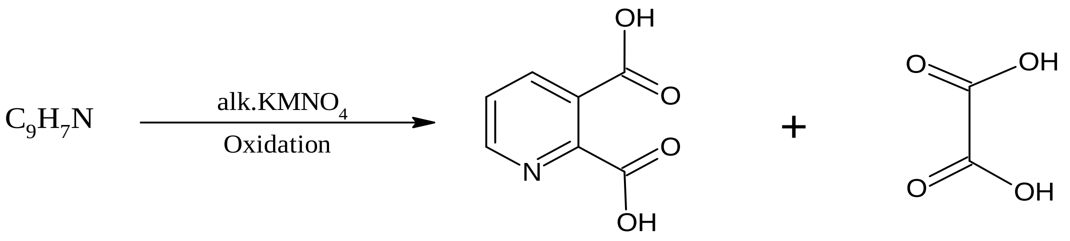
## LITERATURE REVIEW

1. Quinoline and its hybrids have emerged as powerful scaffolds in medicinal chemistry, showing potent activity against cancer, infectious diseases, inflammation, and neurological disorders. Strategic substitutions and molecular hybridizations significantly enhance their biological profiles, with many compounds outperforming standard drugs in preclinical studies. This review emphasizes quinoline's continued potential in innovative drug design. [5]
2. Quinoline derivatives exhibit promising antioxidant and neuroprotective properties, making them strong candidates for combating oxidative stress linked to neurodegenerative diseases like Alzheimer’s and Parkinson’s. Through strategic computational design and filtering based on bioavailability, toxicity, and synthesis, several derivatives were identified as more effective than common antioxidants like Trolox and capable of inhibiting key neurological enzymes. Their multifunctionality suggests potential as future therapeutic agents. [14]
3. Quinoline derivatives, particularly polyquinolines, show promising dual activity as both antiprion and antimalarial agents. Several compounds demonstrated nanomolar-range efficacy and improved selectivity compared to existing treatments like chloroquine and quinacrine. The study highlights structural similarities in their mechanism of action, suggesting overlapping molecular targets and opening new possibilities for drug repurposing and development.[6]
4. These include antimalarial, anticancer, antioxidant, antidiabetic, antimicrobial, antifungal, antiobesity, antiviral, and hair growth–stimulating effects. Their broad therapeutic potential makes them valuable candidates for both drug and cosmetic development, although enhancing delivery systems and conducting further research are crucial for maximizing their efficacy and safety.[9]
5. New quinoline derivatives have shown promising bioactivities against malaria, leishmaniasis, cancer, and microbial infections. Their integration into drug delivery systems further enhances their pharmaceutical value. Altogether, this review underscores how the fusion of synthetic innovation with environmental responsibility is redefining the future of quinoline-based drug development.
6. New 8-aminoquinoline analogs like tafenoquine and bulaquine show enhanced efficacy and reduced toxicity for treating protozoal infections. Simultaneously, synthetic quinolines are exhibiting potent antimicrobial activity, especially against resistant strains

like MRSA. These trends highlight the quinoline scaffold as a dynamic and adaptable core for developing future therapeutics across a broad spectrum of diseases.[10]

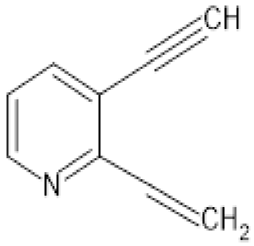
## STRUCTURAL ELUCIDATION OF QUINOLINE

1. Molecular formula is C9H7N deduced from analytical and molecular weight determinations
2. It undergoes electrophilic aromatic substitution reactions like nitration, sulphonation, etc. but does not give Friedel-Craft reaction
3. It is resistant to addition reactions.
4. On vigourous oxidation with alk. KMnO4, it gives pyridine-2,3-dicarboxylic acid (quinolic acid) and oxalic acids.

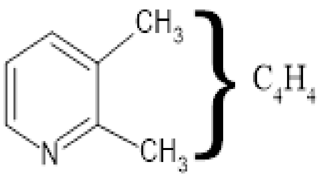


Pyridine-2,3-dicarboxylic acid Oxalic acid

From the reactions, the two possible structures for quinolone are proposed as A or B.

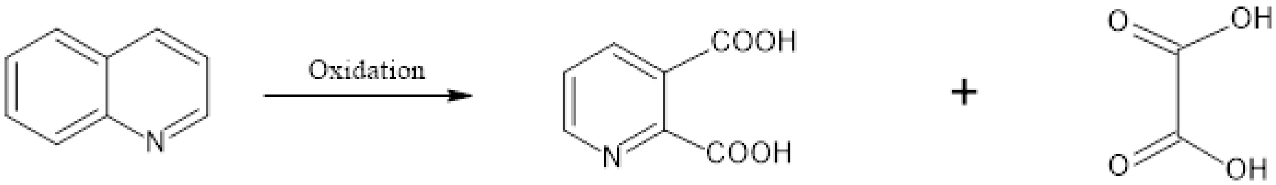


**A**



**B**

Quaternary ammonium salts are formed with alkyl halides. This suggests the presence of a tertiary N-atom in the ring. Hence, the pyridine ring should be fused with a benzene ring through a common carbon pair in quinolone.[16] [17]



Quinoline Pyridine-2,3-dicarboxylic acid (Quinolinic acid) Oxalic acid

## IN SILICO DESIGN OF ANALOGUES

## MATERIALS AND METHODS

| SI. No. | SOFTWARE USED | USAGE |
|---|---|---|
| 1 | CHEMSKETCH | It is used to draw 2D structures. |
| 2 | MOLINSPIRATION | It is used to calculate drug likeliness property |
| 3 | PASS | To predicts biological activity |
| 4 | ADMET AI | ADMET AI is used to predict the pharmacokinetic and toxicity profiles of drug candidates early in the development process, helping to optimize safety, efficacy, and reduce costly experimental failures. |

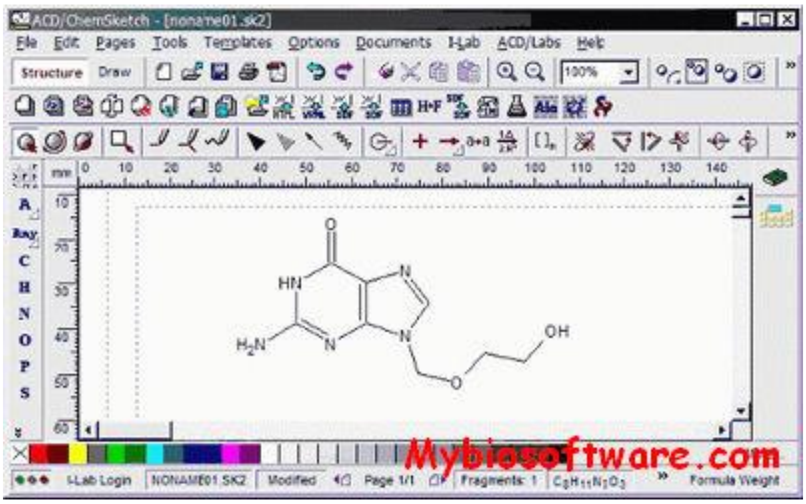
### Introduction to various software's

CHEMSKETCH

Chem Sketch is a chemical drawing software package from Advanced Chemistry Development, Inc., designed to be used alone or integrated with other applications. Chem Sketch is used to draw chemical structures, reactions, and schematic diagrams.

All of the 10 analogues of Quinoline were drawn using ChemSketch.

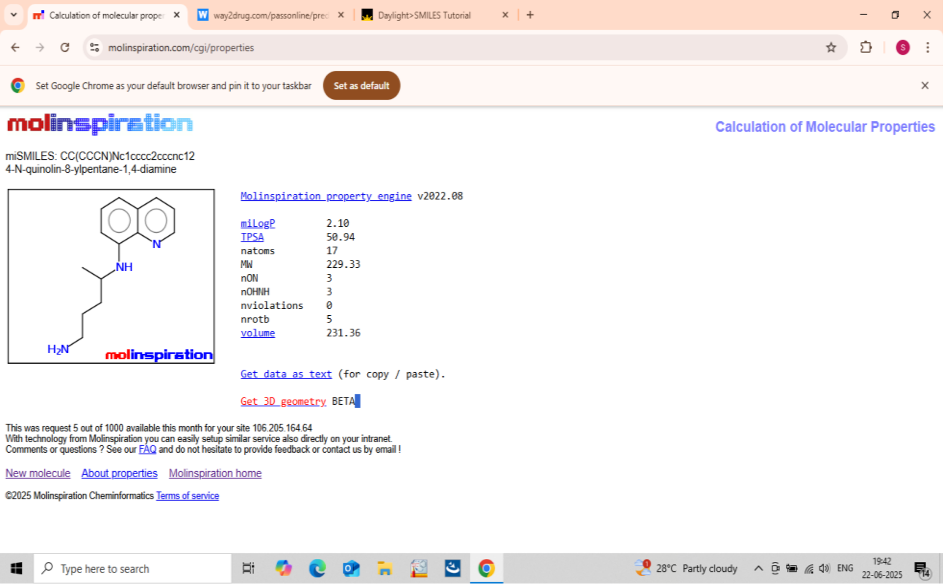
USE: Chem Sketch is a software used for creating and modifying chemical structures, including 2D and 3D molecular models. It also includes features such as calculation of molecular properties (e.g., molecular weight, density, molar refractivity etc.)[10] [11]



### MOLINSPIRATION

Molinspiration is a cheminformatics software company that provides tools for drug discovery and development, specifically focusing on molecular property prediction, bioactivity prediction, and virtual screening.

USE: The software helps researchers analyse and predict properties like lipophilicity, solubility, and bioavailability, aiding in the selection and optimization of drug candidates.[15]



miSMILES: CC(CCCN)Nc1cccc2cccnc12
4-N-quinolin-8-ylpentane-1,4-diamine

```
Molinspiration property engine v2022.08

miLogP         2.10
TPSA           50.94
natoms         17
MW             229.33
nON            3
nOHNH          3
nviolations    0
nrotb          5
volume         231.36

Get data as text (for copy / paste).

Get 3D geometry BETA
```

This was request 5 out of 1000 available this month for your site 106.205.164.64
With technology from Molinspiration you can easily setup similar service also directly on your intranet.
Comments or questions ? See our FAQ and do not hesitate to provide feedback or contact us by email !

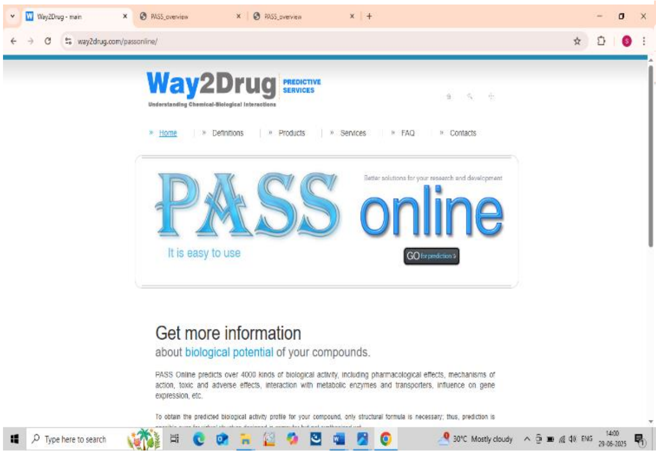
New molecule About properties Molinspiration home

©2025 Molinspiration Cheminformatics Terms of service

**PASS**

PASS (Prediction of Activity Spectra for Substances) is a software product designed as a tool for evaluating the general biological potential of an organic drug-like molecule. PASS provides simultaneous predictions of many types of biological activity based on the structure of organic compounds.

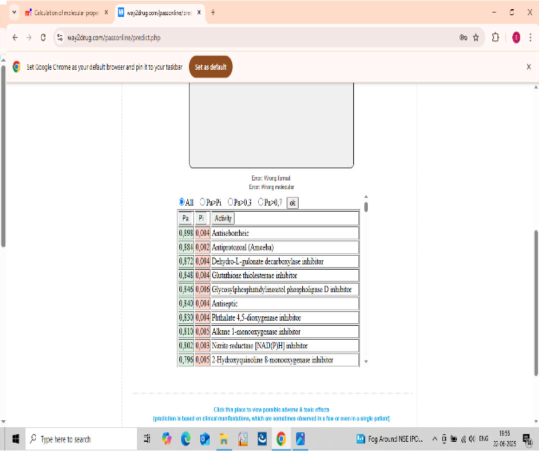
USE: It's a tool used in drug discovery and development to assess potential biological effects, including therapeutic effects, toxic effects, and interactions with enzymes and transporters.[13]



Get more information
about biological potential of your compounds.

PASS Online predicts over 4000 kinds of biological activity, including pharmacological effects, mechanisms of action, toxic and adverse effects, interaction with metabolic enzymes and transporters, influence on gene expression, etc.

To obtain the predicted biological activity profile for your compound, only structural formula is necessary; thus, prediction is



Error: Wrong format
Error: Wrong molecular

● All ○ Pa>Pi ○ Pa>0,3 ○ Pa>0,7 ok

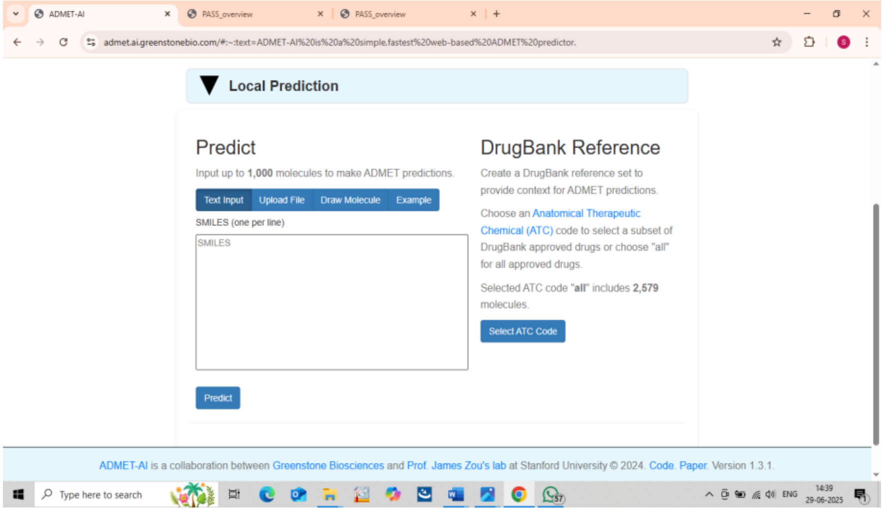
| Pa | Pi | Activity |
|---|---|---|
| 0,898 | 0,004 | Antiseborrheic |
| 0,884 | 0,002 | Antiprotozoal (Amoeba) |
| 0,872 | 0,004 | Dehydro-L-gulonate decarboxylase inhibitor |
| 0,848 | 0,004 | Glutathione thiolesterase inhibitor |
| 0,846 | 0,006 | Glycosylphosphatidylinositol phospholipase D inhibitor |
| 0,840 | 0,004 | Antiseptic |
| 0,830 | 0,004 | Phthalate 4,5-dioxygenase inhibitor |
| 0,810 | 0,005 | Alkane 1-monooxygenase inhibitor |
| 0,802 | 0,003 | Nitrite reductase [NAD(P)H] inhibitor |
| 0,796 | 0,005 | 2-Hydroxyquinoline 8-monooxygenase inhibitor |

Click this place to view possible adverse & toxic effects
(prediction is based on clinical manifestations, which are sometimes observed in a few or even in a single patient)

### ADMET AI

ADMET-AI is a machine learning platform focused on predicting the ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) properties of chemical compounds.

USE: Chemical absorption, distribution, metabolism, excretion, and toxicity (ADMET), play key roles in drug discovery and development.[14]



Input up to **1,000** molecules to make ADMET predictions.

Create a DrugBank reference set to provide context for ADMET predictions.

Choose an Anatomical Therapeutic Chemical (ATC) code to select a subset of DrugBank approved drugs or choose "all" for all approved drugs.

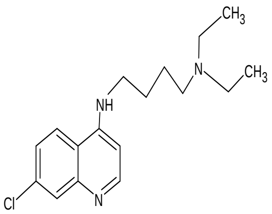
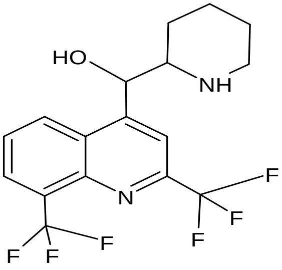
Selected ATC code "**all**" includes **2,579** molecules.

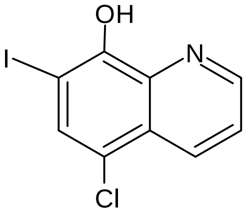
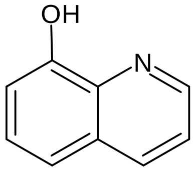
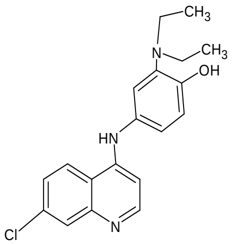
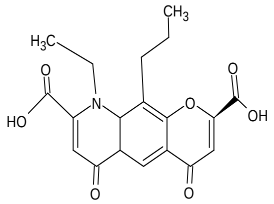
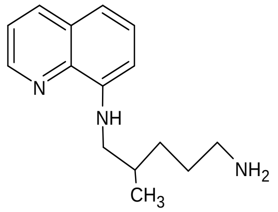
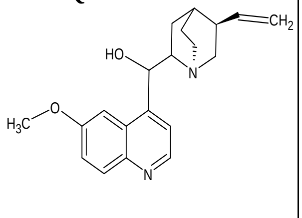
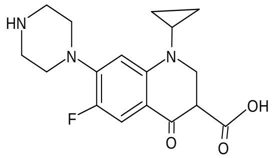
ADMET-AI is a collaboration between Greenstone Biosciences and Prof. James Zou's lab at Stanford University © 2024. Code. Paper. Version 1.3.1.

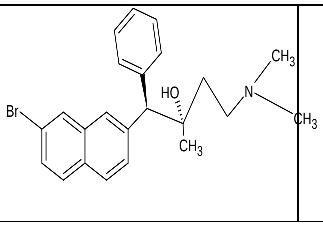
## RESULTS AND DISCUSSION

### CHEMSKETCH

ChemSketch is a chemical drawing software developed by ACD/Labs that allows users to create and visualize chemical structures, reactions, molecular models, and laboratory equipment setups for use in research, education, and publication.

| S.no | COMPOUND | COMPOUND CODE | SMILES | IUPAC NAME |
|---|---|---|---|---|
| 1. | CHLOROQUINE  | QA 1 | CCN(CC)CCCC(C)NC1=C2C=CC(=CC2=NC=C1)Cl | 4-N-(7-chloroquinolin-4-yl)-1-N,1-N-diethylpentane-1,4-diamine |
| 2. | MEFLOQUINE  | QA 2 | C1CCN[C@H](C1)[C@H](C2=CC(=NC3=C2C=CC=C3C(F)(F)F)C(F)(F)F)O | (S)-[2,8-bis(trifluoromethyl)quinolin-4-yl]-[(2R)-piperidin-2-yl]methano |

| | | | | |
|---|---|---|---|---|
| 3. | CLIOQUINOL<br> | QA 3 | C1=CC2=C(C(=C(C=C2Cl)I)O)N=C1 | 5-chloro-7-iodoquinolin-8-ol |
| 4. | OXYQUINOLINE<br> | QA 4 | C1=CC2=C(C(=C1)O)N=CC=C2 | quinolin-8-ol |
| 5. | AMODIAQUINE<br> | QA 5 | CCN(CC)CC1=C(C=CC(=C1)NC2=C3C=CC(=CC3=NC=C2)Cl)O | 4-[(7-chloroquinolin-4-yl)amino]-2-(diethylaminomethyl)phenol |
| 6. | NEDOCROMIL<br> | QA 6 | CCCC1=C2C(=CC3=C1OC(=CC3=O)C(=O)O)C(=O)C=C(N2CC)C(=O)O | 9-ethyl-4,6-dioxo-10-propylpyrano[3,2-g]quinoline-2,8-dicarboxylic acid |
| 7. | PRIMAQUINE<br> | QA 7 | CC(CCCN)NC1=C2C(=CC(=C1)OC)C=CC=N2 | 4-N-(6-methoxyquinolin-8-yl)pentane-1,4-diamine |
| 8. | QUINIDINE<br> | QA 8 | COC1=CC2=C(C=CN=C2C=C1)[C@@H]([C@H]3C[C@@H]4CCN3C[C@@H]4C=C)O | (S)-[(2R,4S,5R)-5-ethenyl-1-azabicyclo[2.2.2]octan-2-yl]-(6-methoxyquinolin-4-yl)methanol |
| 9. | CIPROFLOXACIN<br> | QA 9 | C1CC1N2C=C(C(=O)C3=CC(=C(C=C32)N4CCNCC4)F)C(=O)O | 1-cyclopropyl-6-fluoro-4-oxo-7-piperazin-1-ylquinoline-3-carboxylic acid |
| 10. | DIARYLQUINOLINE | QA 10 | CN(C)CC[C@@](C1=CC=CC2=CC=CC=C21)([C@H](C3=CC=CC= | (1R,2S)-1-(6-bromo-2-methoxyquinolin-3-yl)-4- |

| | | | | |
|---|---|---|---|---|
| |  | | C3)<br>C4=C(N=C5C=CC<br>(=CC5=C4)Br)OC)O | (dimethylamino)-2-naphthalen-1-yl-1-phenylbutan-2-ol |

All of the 10 analogues of Quinoline were selected using ChemSketch.

## MOLINSPIRATION

Calculate log P, molecular weight, hydrogen bond acceptors and donors, and also find any violations among the properties.

| Sl.No. | COMPOUND CODE | MOLECULAR WEIGHT | No. of H BOND ACCEPTORS | No. of H BOND DONORS | LOG P VALUE | No of VIOLATIONS |
|---|---|---|---|---|---|---|
| 1. | QA 1 | 305.85 | 3 | 1 | 4.01 | 0 |
| 2. | QA 2 | 378.32 | 3 | 2 | 4.24 | 0 |
| 3. | QA 3 | 305.50 | 2 | 1 | 3.62 | 0 |
| 4. | QA 4 | 145.16 | 2 | 1 | 1.68 | 0 |
| 5. | QA 5 | 341.84 | 4 | 2 | 5.38 | 1 |
| 6. | QA 6 | 371.35 | 8 | 2 | 2 | 0 |
| 7. | QA 7 | 229.33 | 3 | 3 | 2.10 | 0 |
| 8. | QA 8 | 324.42 | 4 | 1 | 3.06 | 0 |
| 9. | QA 9 | 333.36 | 6 | 2 | 0.82 | 0 |
| 10. | QA 10 | 412.37 | 1 | 1 | 5.57 | 1 |

## PASS(Prediction of Activity Spectra for Substances).

| COMPOUND CODE | GLUCONATE 2-DEHYDROGENASE INHIBITOR ACTIVITY | |
|---|---|---|
| | Pa | Pi |
| QA 1 | 0,966 | 0,001 |
| QA 2 | 0,975 | 0,001 |
| QA 3 | 0,643 | 0,092 |
| QA 4 | 0,807 | 0,015 |
| QA 5 | 0,850 | 0,008 |
| QA 6 | 0,496 | 0,205 |
| QA 7 | 0,942 | 0,002 |
| QA 8 | 0,440 | 0,255 |
| QA 9 | 0,717 | 0,047 |
| QA 10 | 0,464 | 0,232 |

## ADMET AI

## ABSORPTION

| COMPOUND CODE | PROPERTY | PREDICTION | DRUG BANK PERCENTILE | EXPLANATION |
|---|---|---|---|---|
| QA 1 | Human intestinal absorption | 1.00 | 71.46% | Excellent absorption (score: 1.00), it is fully |

| | | | | |
|---|---|---|---|---|
| | | | | taken up through the gut. |
| | Oral bioavailability | 0.94 | 85.58% | High oral bioavailability |
| QA 2 | Human intestinal absorption | 1.00 | 85.89% | Excellent absorption (score: 1.00), it is fully taken up through the gut. |
| | Oral bioavailability | 0.83 | 56.69% | Good oral bioavailability |
| QA 3 | Human intestinal absorption | 1.00 | 96.80% | Excellent absorption (score: 1.00), it is fully taken up through the gut. |
| | Oral bioavailability | 0.82 | 53.66% | Good oral bioavailability |
| QA 4 | Human intestinal absorption | 1.00 | 90.93% | Excellent absorption (score: 1.00), it is fully taken up through the gut. |
| | Oral bioavailability | 0.61 | 26.95% | Low oral bioavailability |
| QA 5 | Human intestinal absorption | 1.00 | 56.42% | Excellent absorption (score: 1.00), it is fully taken up through the gut. |
| | Oral bioavailability | 0.77 | 46.06% | Low oral bioavailability |
| QA 6 | Human intestinal absorption | 0.51 | 17.22% | Poor absorption (score:0.51), therefore poor absorption through the gut. |
| | Oral bioavailability | 0.67 | 31.83% | Low oral bioavailability |
| QA 7 | Human intestinal absorption | 1.00 | 96.04% | Excellent absorption (score: 1.00), it is fully taken up through the gut. |
| | Oral bioavailability | 0.88 | 67.86% | Good oral bioavailability |
| QA 8 | Human intestinal absorption | 1.00 | 82.98% | Excellent absorption (score: 1.00), it is fully |

| | | | | |
|---|---|---|---|---|
| | | | | taken up through the gut. |
| | Oral bioavailability | 0.88 | 66.93% | Good oral bioavailability |
| QA 9 | Human intestinal absorption | 0.97 | 33.8% | Poor absorption (score:0.51), therefore poor absorption through the gut. |
| | Oral bioavailability | 0.93 | 82.55% | High oral bioavailability |
| QA 10 | Human intestinal absorption, | 1.00 | 98.02% | Excellent absorption (score: 1.00), it is fully taken up through the gut. |
| | Oral bioavailability | 0.88 | 67.27% | Good oral bioavailability |

**DISTRIBUTION**

| COMPOUND CODE | PROPERTY | PREDICTION | DRUG BANK PERCENTILE | EXPLANATION |
|---|---|---|---|---|
| QA 1 | Blood-Brain Barrier | 0.99 | 93.64% | A value of 0.99 is very high, suggesting strong likelihood of central nervous system (CNS) activity. |
| | Volume of Distribution at Steady State | 116.62 | 99.92% | the compound distributes extensively into body tissues rather than remaining in the plasma. |
| QA 2 | Blood-Brain Barrier | 0.97 | 82.16% | potential for central nervous system (CNS) activity, which might be relevant for drugs targeting the brain. |
| | Volume of Distribution at Steady State | 7.26 | 84.65% | above 0.6 L/kg usually indicates extensive distribution beyond just the blood compartment, so 7.26 is quite high |
| QA 3 | Blood-Brain Barrier | 0.59 | 36.87% | moderate chance that the compound can cross the blood-brain |

| | | | | |
|---|---|---|---|---|
| | | | | barrier (BBB). |
| | Volume of Distribution at Steady State | 0.00 | 36.10% | Indicates an extremely low predicted volume of distribution. |
| QA 4 | Blood-Brain Barrier | 0.60 | 37.53% | A value of 0.60 suggests a moderate chance of penetration. |
| | Volume of Distribution at Steady State | 0.00 | 6.05% | 0.00 implies negligible distribution beyond the plasma |
| QA 5 | Blood-Brain Barrier | 0.91 | 68.71% | A high probability that the molecule can cross the blood brain barrier. |
| | Volume of Distribution at Steady State | 28.27 | 98.91% | extensive distribution into body tissues |
| QA 6 | Blood-Brain Barrier | 0.24 | 14.73% | It is less likely to cross into the brain. |
| | Volume of Distribution at Steady State | 7.25 | 84.57% | The drug distributes extensively into body tissues. |
| QA 7 | Blood-Brain Barrier | 0.98 | 83.7% | A high probability that the molecule can cross the blood-brain barrier. |
| | Volume of Distribution at Steady State | 45.24 | 99.69% | The drug distributes extensively into body tissues |
| QA 8 | Blood-Brain Barrier | 0.93 | 72.24% | A value close to 1 (like 0.93) indicates a high likelihood of BBB penetration. |
| | Volume of Distribution at Steady State | 2.45 | 57.46% | Extensively, a drug is expected to distribute into body tissues relative to the bloodstream. |
| QA 9 | Blood-Brain Barrier | 0.29 | 17.72% | This score indicates a low probability that the molecule can cross the blood-brain barrier. |
| | Volume of Distribution at Steady State | 1.40 | 48.75% | This value suggests the compound has a moderate distribution |

| | | | | |
|---|---|---|---|---|
| | | | | throughout body tissues |
| QA10 | Blood-Brain Barrier | 0.87 | 63.47% | A value of 0.87 indicates a moderate to high that the molecule can penetrate the blood brain barrier. |
| | Volume of Distribution at Steady State | 7.27 | 84.72% | The drug distributes extensively into body tissues. |

### METABOLISM

| COMPOUND CODE | PROPERTY | PREDICTION | DRUG BANK PERCENTILE | EXPLANATION |
|---|---|---|---|---|
| QA 1 | CYP1A2 Inhibitor | 0.81 | 92.56% | the compound might interfere with drug metabolism, which is an important factor when evaluating drug safety and dosing. |
| | CYP2C9 Substrate | 0.15 | 51.53% | A score of 0.15 means the compound is unlikely to be metabolized by CYP2C9. |
| QA 2 | CYP1A2 Inhibitor | 0.30 | 75.88% | A value of 0.30 indicates a low-to-moderate chance of inhibition |
| | CYP2C9 Substrate | 0.14 | 49.28% | A low substrate probability (like 0.14) means CYP2C9 likely plays a minimal role in the drug's metabolism |
| QA 3 | CYP1A2 Inhibitor | 0.91 | 95.46% | A value of 0.83 indicates a high likelihood of inhibition |
| | CYP2C9 Substrate | 0.21 | 63.51% | A value of 0.24 suggests the compound has a low probability of being a substrate |
| QA 4 | CYP1A2 Inhibitor | 0.83 | 92.98% | A value of 0,83 indicates a very high likelihood that this molecule inhibits CYP1A2 |
| | CYP2C9 Substrate | 0.24 | 69.48% | The value indicates a 24% that is relatively low. |
| QA 5 | CYP1A2 Inhibitor | 0.90 | 95.35% | 90% probability inhibiting the CYP1A2 |

| | | | | |
|---|---|---|---|---|
| | | | | enzyme. |
| | CYP2C9 Substrate | 0.24 | 69.64% | 24% probability substrate of the CYP2C9 enzyme |
| QA 6 | CYP1A2 Inhibitor | 0.04 | 49.59% | . A prediction score of 0.04 tells us this molecule is very unlikely to inhibition |
| | CYP2C9 Substrate | 0.11 | 38.66% | This suggests the molecule is unlikely to be metabolized by CYP2C9, |
| QA 7 | CYP1A2 Inhibitor | 0.87 | 94.03% | 87% predicted probability that the molecule inhibits the CYP1A2 enzyme. |
| | CYP2C9 Substrate | 0.15 | 49.94% | 15% predicted probability that the compound is a substrate of the CYP2C9 enzyme |
| QA 8 | CYP1A2 Inhibitor | 0.46 | 82.01% | 46% predicted probability that the molecule inhibits the CYP1A2 enzyme |
| | CYP2C9 Substrate | 0.21 | 64.64% | 21% probability that the molecule is a substrate of the CYP2C9 enzyme |
| QA 9 | CYP1A2 Inhibitor | 0.01 | 30.98% | 0.01 This value indicates a *very low probability* that the compound inhibits the CYP1A2 enzyme |
| | CYP2C9 Substrate | 0.14 | 48.89% | A score of 0.14 means there's a low likelihood the molecule is metabolized by CYP2C9. |
| QA 10 | CYP1A2 Inhibitor | 0.57 | 84.99% | 57% predicted probability that the molecule inhibits the CYP1A2 enzyme. |
| | CYP2C9 Substrate | 0.30 | 78.60% | 30% probability that the molecule is a substrate of the CYP2C9 enzyme. |

**EXCRETION**

| COMPUND CODE | PROPERTY | PREDICTION | DRUG BANK PERCENTILE | EXPLANATION |
|---|---|---|---|---|
| QA 1 | Drug clearance (hepatocyte) | 49.08 | 61.46% | This represents how quickly liver cells (hepatocytes) can clear the drug from |

| | | | | |
|---|---|---|---|---|
| | | | | circulation. |
| QA 2 | Drug clearance (hepatocyte) | 0.00 | 10.20% | A zero value impliepossibly due to low uptake. |
| QA 3 | Drug clearance (hepatocyte) | 127.80 | 96.59% | Represents a very high hepatic clearance rate—how quickly liver cells metabolize and eliminate the drug. |
| QA 4 | Drug clearance (hepatocyte) | 131.66 | 97.32% | Represents a very high hepatic clearance rate—how quickly liver cells metabolize and eliminate the drug. |
| QA 5 | Drug clearance (hepatocyte) | 64.02 | 73.09% | moderately to rapidly cleared by liver cells. |
| QA 6 | Drug clearance (hepatocyte) | 21.30 | 33.97% | Lower end of hepatic clearance. |
| QA 7 | Drug clearance (hepatocyte) | 58.32 | 69.29% | moderately to rapidly cleared by liver cells. |
| QA 8 | Drug clearance (hepatocyte) | 36.76 | 50.60% | The compound is predicted to be cleared from liver cells at a rate of 36.76 µL/min |
| QA 9 | Drug clearance (hepatocyte) | 2.90 | 16.94% | Lower end of hepatic clearance. |
| QA 10 | Drug clearance (hepatocyte) | 80.95 | 82.86% | Represents a very high hepatic clearance rate—how quickly liver cells metabolize and eliminate the drug. |

## TOXICITY

| COMPOUND CODE | PROPERTY | PREDICTION | DRUG BANK PERCENTILE | EXPLANATION |
|---|---|---|---|---|
| QA1 | Mutagenicity | 0.64 | 89.69% | A value of 0.64 suggests a moderate-to-high risk of being mutagenic. |
| | Drug-Induced Liver Injury | 0.47 | 52.50% | A moderate probability score for causing liver toxicity**.** |
| | Carcinogenicity | 0.37 | 80.07% | This reflects the estimated probability of the compound being cancer-causing |
| QA2 | Mutagenicity | 0.12 | 36.95% | The compound has low mutagenicity risk, but not negligible. |
| | Drug-Induced | 0.24 | 36.91% | Suggests **a** low-to- |

| | | | | |
|---|---|---|---|---|
| | Liver Injury | | | moderate chance of causing liver toxicity. |
| | Carcinogenicity | 0.25 | 66.38% | A low probability of being cancer-causing. |
| QA3 | Mutagenicity | 0.30 | 66.03% | The value of 0.30 cause genetic mutation |
| | Drug-Induced Liver Injury | 0.88 | 79.95% | High risk of hepatotoxicity |
| | Carcinogenicity | 0.07 | 26.13% | Low risk of carcinogenicity. |
| QA4 | Mutagenicity | 0.46 | 79.95% | A value of 0.46 is moderate—not strongly mutagenic, but not entirely free of risk either |
| | Drug-Induced Liver Injury | 0.68 | 63.75% | moderate-to-high risk of hepatotoxicity. |
| | Carcinogenicity | 0.03 | 12.41% | This is a very low risk profile in terms of carcinogenicity. |
| QA5 | Mutagenicity | 0.62 | 88.76% | The value of 0.62 is relatively high mutagenic risk. |
| | Drug-Induced Liver Injury | 0.64 | 61.30% | Suggests a moderate risk of hepatotoxicity. |
| | Carcinogenicity | 0.42 | 83.99% | Very high risk profile in terms of carcinogenicity |
| QA6 | Mutagenicity | 0.44 | 78.60% | The value of 0.44 cause genetic mutation |
| | Drug-Induced Liver Injury | 0.96 | 90.81% | A very high probability (96%) that this compound could trigger liver damage. |
| | Carcinogenicity | 0.28 | 69.41% | Low risk profile in terms of carcinogenicity. |
| QA7 | Mutagenicity | 0.54 | 84.96% | The value of 0.54 causes genetic mutation |
| | Drug-Induced Liver Injury | 0.38 | 45.64% | A 38% probability of causing liver toxicity |
| | Carcinogenicity | 0.21 | 60.22% | Low risk profile in terms of carcinogenicity |
| QA8 | Mutagenicity | 0.62 | 88.95% | moderate-to-high probability (0.62) that the compound might cause genetic mutations. |
| | Drug-Induced Liver Injury | 0.24 | 35.79% | A low-to-moderate predicted risk of DILI (0.24). |
| | Carcinogenicity | 0.03 | 13.80% | Very low probability of |

| | | | | |
|---|---|---|---|---|
| | | | | being carcinogenic (0.03). |
| QA9 | Mutagenicity | 0.54 | 85.07% | This molecule has a moderate-to-high probability of being mutagenic |
| | Drug-Induced Liver Injury | 0.96 | 90.46% | Very high risk of hepatotoxicity |
| | Carcinogenicity | 0.18 | 55.14% | Low probability of being carcinogenicity. |
| QA10 | Mutagenicity | 0.05 | 19.85% | The molecule causes genetic mutations with low mutagenic potential. |
| | Drug-Induced Liver Injury | 0.38 | 46.30% | A 38% probability of causing liver toxicity. |
| | Carcinogenicity | 0.15 | 48.66% | Molecule has a relatively low but non-negligible risk of causing cancer. |

## CONCLUSION

The present study explored the potential of quinoline scaffolds as promising candidates for drug development using various in-silico approaches. Natural products, particularly alkaloids such as quinoline derivatives, have long been recognised for their diverse pharmacological activities. Through detailed structural analysis and computational tools, including ChemSketch, Molinspiration PASS, and ADMET AI, ten quinoline analogues were evaluated for drug likeness predicted bioactivity, pharmacokinetic, and toxicity profiles.

The results highlight that several analogues, particularly QA(chloroquine), QA2(mefloquine) and QA3(primaquine) exhibited favourable ADMET characteristics, high intestinal absorption and good oral bioavailability. Furthermore, PASS predictions demonstrated significant biological potential, including enzyme inhibition relevant to therapeutic applications. Although some analogues posed moderate risks of mutagenicity or hepatotoxicity, these insights serve as a foundation for further optimisation.

Finally this study underscores the effectiveness of combining natural product chemistry with modern computational methods to accelerate early-stage drug discovery. The quinoline scaffold supported by in silico validation remains a vital and aptable framework for the design of next-generation therapeutics targeting a wide range of diseases.

## ACKNOWLEDGEMENTS

We want to offer this endeavour to GOD ALMIGHTY for all the blessings showered on us during the course of this review. We take the privilege to acknowledge all those who helped in the completion of the review. At first, we express a deep sense of gratitude and indebtedness to the Department of Pharmaceutical Chemistry of Mar Dioscorus College of Pharmacy for helping in the completion of our review. We are extremely grateful to our Principal for her guidance and valuable suggestions, which helped to complete our work. We are deeply obliged to Mrs. Rachel Mathew, our guide as well as mentor, for her guidance, immense knowledge, insightful comments, constant support, and encouragement, which helped us complete our work within the time schedule. We express our sincere gratitude to Mrs.Rachel Mathew, our co-guide Mrs.Vani V, for sharing their expertise by giving constructive comments and suggestions upon reviewing our study.

## REFERENCES

1. de Souza MV, Pais KC, Kaiser CR, Peralta MA, de L Ferreira M, Lourenço MC. Synthesis and in vitro antitubercular activity of a series of quinoline derivatives. Bioorg Med Chem., 2009 Feb 15; 17(4): 1474-80. doi: 10.1016/j.bmc.2009.01.013. Epub 2009 Jan 15. PMID: 19188070.
2. Kaushik, Manish (2014-04-04). "A review of Innovative Chemical Drawing and Spectra Prediction Computer Software". Mediterranean Journal of Chemistry, 3(1): 759–766. doi:10.13171/mjc.3.1.2014.04.04.16. ISSN 2028-3997.
3. Medicinal natural products : a biosynthetic approach (3rdition ed.). Wiley. 380–381. ISBN 9780470742761.
4. Trenti F, Yamamoto K, Hong B, Paetz C, Nakamura Y, O’Connor SE. Early and late steps of quinine biosynthesis. Org Lett., 2021; 23(5): 1793–1797. doi:10.1021/acs.orglett.1c00206
5. O'Connor SE, Maresh JJ (August 2006). "Chemistry and biology of monoterpene indole alkaloid biosynthesis". Natural Product Reports., 23(4): 532–547. doi:10.1039/b512615k. PMID 16874388.
6. Bhat SV, Nagasampagi BA, Meenakshi Sivakumar. Chemistry of natural products. Berlin: Springer ; New Delhi Narosa, 2005; 1-2.
7. Cseke LJ, Kirakosyan A, Kaufman PB, Warber S, Duke JA, Brielmann HL. Natural Products from Plants. CRC Press, 2016; 30.

8. Finar IL. Organic Chemistry. Vol. 2: Stereochemistry and the Chemistry of Natural Products. 5th ed. New Delhi: Pearson Education India, 2002; 726-729.
9. Z. Li, H. Wan, Y. Shi, P. Ouyang, “Personal Experience with Four Kinds of Chemical Structure Drawing Software: Review on ChemDraw, ChemWindow, ISIS/Draw, and ChemSketch,” Journal of Chemical Information and Computer Sciences, 2004; 44(5): 1886–1890.ChemSketch is discussed as ACD/Labs’ molecular drawing software.
10. M. Kaushik, “A review of Innovative Chemical Drawing and Spectra Prediction Computer Software,” Mediterranean Journal of Chemistry, 2014; 3(1): DOI:10.13171/mjc.3.1.2014.04.04.16.ChemSketch is evaluated among tools like ChemDraw, MarvinSketch, and others.
11. Thangarasu P, Thamarai Selvi S, Manikandan A. Unveiling novel 2‑cyclopropyl‑3‑ethynyl‑4‑(4‑fluorophenyl) quinolines as GPCR ligands via PI3‑kinase/PAR‑1 antagonism and platelet aggregation evaluations: development of a new class of anticancer drugs with thrombolytic effects. Bioorganic Chemistry., 2018; 81: 468–480. DOI:10.1016/j.bioorg.2018.09.011
12. Molinspiration Cheminformatics Tools, Molinspiration™ (online), available at http://www.molinspiration.com (accessed [date of access]).
13. Lagunin A, Ivanov S, Rudik AV, Filimonov DA, Poroikov VV. PASS Targets: ligand-based multi-target computational system based on a public data and naïve Bayes approach. Bioinformatics., 2013; 29(16): 2062–2063. doi:10.1093/bioinformatics/btt322
14. Swanson K, Walther P, Leitz J, Mukherjee S, Wu JC, Shivnaraine RV, Zou J. ADMET-AI: a machine learning ADMET platform for evaluation of large-scale chemical libraries. Bioinformatics., 2024 Jul 1; 40(7): btae416. doi:10.1093/bioinformatics/btae416
15. Rani S, Mukhopadhyay N, Tiwari RS. Theoretical studies on the molecular properties, toxicity, and biological efficacy of 21 new chemical entities. ACS Omega., 2021; 6(43): [page numbers]. Molinspiration was applied to calculate molecular properties (log P, H-bond donors/acceptors, TPSA) and predict bioactivity across enzyme, GPCR, ion channel, kinase, and nuclear receptor classesHI
16. K.D Sharma and Y. R. Sharma, Kalyani Publishers, Unit IV.
17. University Chemistry, Vol-IV, Dr. U. N. Dash, Dr. K. K.Ojha, Himalaya Publishing house, Unit V.