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CHEMICAL PROFILING, ADME PREDICTION OF *BOERHAVIA DIFFUSA* LINN. AND *CRATEVA NURVALA* BUCH. HAM IN THE MANAGEMENT OF UROLITHIASIS

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Keywords:

DPPH Assay, ADME, UV-VIS, FTIR, GC-MS analysis, *Boerhavia diffusa, Crateva nurvala*

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ABSTRACT: Background: This study was carried out to investigate qualitative and quantitative phytochemical analysis and free radical scavenging activity of *Boerhavia diffusa* (root) and *Crateva nurvala* (bark) along with certain computational ADME (Absorption, Distribution, Metabolism, Excretion) parameters of compounds analyzed from *Boerhavia diffusa* (root) and *Crateva nurvala* (bark). **Methods:** The dried powder of plants were extracted with increasing polarity of solvent namely ethyl acetate, acetone, ethanol and distilled water by a three-step sequential extraction procedure. The chemical compositions of extracts were investigated using UV-Vis, FTIR and PerkinElmer Gas Chromatography-Mass Spectrometry. Ethanolic extract of *Boerhavia diffusa* and *Crateva nurvala* was evaluated *in vitro* for its ability to inhibit the major enzyme activities of GOX and LDH through spectrophotometrically and mode of inhibition were evaluated using Lineweaver-Burk plots while ADME parameters were derived from ADMET server admet SAR. **Results:** Among all extracts, ethanolic extracts showed the highest phenol, flavonoid, proanthocyanidins, and coumarin content. High DPPH free radical quenching activity was observed in ethanolic, ethyl acetate and acetone extracts of the plant which was significantly comparable with ascorbic acid. Also, plant extracts, analyzed by UV-FTIR and GC-MS which showed promising Glycolate oxidase and Lactate dehydrogenase inhibition and found to contain phenolic, glycosides and flavonoids compounds. **Conclusion:** The FTIR and GC-MS spectrum profile of the medicinally important plant extract having various bioactive compounds and interestingly from the absorption, distribution, metabolism, and excretion (ADME)-Toxicity analysis the compound have enhanced pharmacological properties and can be used as an herbal alternative for the synthesis of antiurolithiac agents.

INTRODUCTION: Urolithiasis is a complex process that results from several physicochemical events including crystal nucleation, aggregation, and growth of insoluble particles in the kidney [1].

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Calculogenesis is influenced by the interplay of critical factors, *viz.* stone inhibitors, complexing agents and stone promoters. The sequence of events in the formation of any urinary stone can be:

Urinary saturation ⇨ super saturation ⇨ nucleation ⇨ crystal ⇨ growth ⇨ crystal ⇨ aggregation ⇨ crystal retention ⇨ stone formation.

Urolithiasis affects 10-12% of the world population, especially in the industrialized countries. It has a recurrence rate of 50% [2].

In spite of substantial progress in the study of the biological and physical manifestations of kidney stones, there is no satisfactory drug to use in clinical therapy [3]. Therefore, it is worthwhile to look for an alternative for the management of urolithiasis. Herbal drugs are essential components of traditional medicine in several countries including India. *Boerhavia diffusa* and *Crateva nurvala* are important medicinal plants much used in Ayurveda and Unani medicines and other traditional medicines in many parts of the world. *Crataeva nurvala* Buch.-Ham. belongs to the family Capparaceae. The stem bark is being widely used as a single drug or in compound formulations for the treatment of urinary disorders including urolithiasis, prostatic hypertrophy, blood purifier, skin infections, neurogenic bladder and chronic urinary infections [4]. *Boerhaavia diffusa* Linn. belongs to the family Nyctaginaceae and is commonly known as, punarnava. Traditionally, this plant has been extensively used in the treatment of dyspepsia, jaundice enlargement of liver, abdominal pain and as an antistress agent. It also has diuretic, anti-inflammatory, antidiabetic, antibacterial and anticancer properties [5, 6].

According to the World Health Organization (WHO) in 2008, more than 80% of the world's population relies on traditional medicine for their primary healthcare needs [7]. Phenolics and flavonoids are polyphenols, an important class of secondary plant metabolites possessing an impressive array of pharmacological activity which includes free radical scavenging, inhibition of hydrolytic and oxidative enzymes and anti-inflammatory action, *etc.* [8] Oxidation reaction can produce free radicals, which can damage cells and tissues. Recently, there has been an upsurge of interest in the therapeutic potential of medicinal plants as antioxidants in reducing such free radical-induced tissue injury.

As a result, antioxidants are often reducing agents such as phenolic compounds (flavonoids, phenolic acids), sugars, vitamins, saponins, ethereal oils, polyunsaturated fatty acids, phospholipids, enzymes, amino acids, thiols, *etc*. As every plant has its metabolite fingerprinting, profiling by some platform technology such as gas chromatography-mass spectrometer (GC-MS), liquid chromatography-mass spectrometer and nuclear magnetic resonance, it is crucial to have a holistic overview of all metabolites present in the extracts [9]. Spectroscopic (UV-Vis, FTIR) methods are a simple, cost-effective and rapid test for detecting phytocomponents. Hence, the present study is designed to evaluate the phytochemical profile of *Boerhavia diffusa* and *Crateva nurvala* with the aid of GC-MS, UV-VIS, and FTIR Techniques and computer analysis using ADME to ascertain the rationale for its use in traditional medicine.

MATERIALS AND METHODS:

Drugs: *Boerhavia diffusa* (root) and *Crateva nurvala* (bark) were procured from Yogesh pharma Pvt. Ltd., Nanded (MS), India. The barks and roots were washed thoroughly with water to remove dust and dried under the shade at room temperature for 5 days. The dried parts were ground using a blender to obtain the coarse powder and kept in an airtight container until further use. Powdered plant material (150 g) were successively extracted with Soxhlet apparatus using the solvents in order of increasing polarity *viz*., C), ethyl acetate, acetone, ethanol, and water. Each time the marc was dried and later extracted with other solvents. All the extracts were concentrated by distilling the solvent in a rotary vacuum evaporator.

Phytochemical Screening of Bark Extract: The extracts were subjected to phytochemical tests to investigate the presence or absence of active secondary metabolites using standard procedures [10].

Quantitative Estimation of Phytoconstituents:

Quantitative Estimation of Total Phenols: Folin-Ciocalteu reagent was used to determine the total phenolic content (TPC) of the various organic crude extracts with slight modification [11]. 1.5 ml Folin-Ciocalteu reagent (diluted 1:10 with de-ionized water) was added to the 0.5 mL of the plant extract sample, and was vortexed for 5 min, followed by addition of 3 mL of sodium carbonate solution (7.5%). This reaction mixture was incubated for 30 min at room temperature in the dark. The absorbance of the resulting blue color was measured by using double beam UV-Vis spectrophotometer (UV Shimadzu- 1800) at a fixed wavelength of 760 nm. The calibration curve was prepared by employing gallic acid at concentrations of 10 to 100 µg/ml **Fig. 1**.

The TPCs were determined using a linear regression equation obtained from the standard plot of gallic acid. The content of total phenolic compounds was calculated as mean ± SD (*n*=3) and expressed as mg/g gallic acid equivalent (GAE) of dry extract.

Quantitative Estimation of Total Flavonoid: Aluminum chloride colorimetric method was used for flavonoids determination as described by [11]. 0.5 mL of each plant extract was mixed with 1.5 mL of ethanol, 0.1 mL of 10% aluminum chloride, 0.1 mL of 1 M potassium acetate and 2.8 mL of distilled water. The reaction mixture was allowed to stand at room temperature for 30 min, and the absorbance of the reaction mixture was measured at 415 nm.

The calibration curve was prepared by using rutin at concentrations of 10 to100 µg/ml in ethanol **Fig. 1**. The amount of flavonoid was calculated from the linear regression equation obtained from the rutin calibration curve. The flavonoid content was calculated as mean ± SD (*n*=3) and expressed as mg/g of rutin equivalent (RE) of dry extract.

Quantitative Estimation of total Proanthocyanidins: The total proanthocyanidin was determined using the procedure reported by Sun *et al*. [12] A volume of 0.5 mL of 0.1 mg/mL of extract solution was mixed with 3.0 mL of 4% vanillin-methanol solution and 1.5 mL hydrochloric acid, the mixture was allowed to stand for 15 min at room temperature, the absorbance was measured at 500 nm. Total proanthocyanidin contents were expressed as catechin (mg/g) using the following equation of the curve: Y= 0.69x + 0.076, R2 = 0.970, where x is the absorbance and Y is the catechin equivalent **Fig. 1**.

Quantitative Estimation of Total Coumarin: Coumarin content was determined with slight modification in procedure reported by Bruna Medeiros-Neves *et al.* [13] A volume of 0.5 mL of the extract was mixed with 2 ml of distilled water and 0.5 mL of lead acetate solution. The sample was shaken, and then 7 mL of distilled water was added before transferring 2 mL of this solution to a new test tube and added 8 mL of hydrochloric acid solution. The sample was incubated at room temperature for 30 min, and the absorbance was measured at 500 nm. Total coumarin contents were expressed as coumarin (mg/g) using the following equation of the curve: Y= 0.050x + 0.022, R2 = 0.992, where x is the absorbance and Y is the coumarin equivalent **Fig. 1**.

Free Radical Scavenging Activity by DPPH Assay Method: The capacity of the extracts and ascorbic acid to scavenge the stable 1, 1-diphenyl-2-picrylhydrazyl (DPPH) free radical was measured as per reported method [14]. 0.1 mL of extracts and ascorbic acid of different concentrations (10 to 200 µg/ml) was mixed with 2.9 mL of 0.1mM DPPH solution. The solution was rapidly mixed and allowed to stand in the dark at room temperature for 30 min. The blank was prepared similarly without extract or ascorbic acid. The decrease in absorbance of each solution was measured at 517 nm using UV-Vis spectrophotometer.

The negative control was prepared by mixing 0.1 mL of ethanol with 2.9 mL of DPPH solution. The percentage of radical scavenging activity of tested extracts and positive control ascorbic acid was calculated by using the following formula:

$$\text{Free radical scavenging activity (\%)} = [Ac - As] \times 100 / Ac$$

Where Ac=Absorbance of control at 517 nm and As=Absorbance of the sample.

The concentration of the sample required to scavenge 50% of DPPH free radical (IC_{50}) was determined from the curve of percent inhibitions plotted against the respective concentration.

UV-Vis and FTIR Spectroscopic Analysis: The ethanolic extracts were examined under visible and UV light for proximate analysis. For UV-VIS and FTIR spectrophotometer analysis, the extracts were centrifuged at 5000 rpm for 15 min and filtered through Whatman no. 1 filter paper. The sample is diluted to 1:10 with the same solvent. The extracts were scanned in the wavelength ranging from 200-1100 nm using UV-1800 Shimadzu UV Spectrophotometer, and the characteristic peaks were detected.

FTIR analysis was performed using Thermofisher Scientific, model- Nicolet 6700 FTIR system in a scan range of 400-4000 cm^{-1} and characteristic peaks and their functional groups were detected.

Gas Chromatography-Mass Spectrometry Analysis: The GC-MS analysis of the ethanolic extracts were carried out using a Agilent 7890 A gas chromatogram equipped and coupled to a mass detector 5975 MSD spectrometer with DB 5 MS and 30 m × 0.25 µm DF of the capillary column. Ultra-high purity helium (99.99%) was used as carrier gas at a constant flow rate of 1.0 mL/min. The injection, transfer line and ion source temperatures were at all 290 °C. The ionizing energy was 70 eV. Electron multiplier voltage was obtained from autotune. The oven temperature was programmed from 60 °C (hold for 2 min) to 320 °C at a rate of 3 °C/min. The crude sample was diluted with an appropriate solvent (1/10, v/v) and filtered.

The particle-free diluted crude extracts (1 µL) were taken in a syringe and injected into injector with a split ratio of 30:1. All data were obtained by collecting the full-scan mass spectra within the scan range 30-600 amu. The percentage composition of the crude extract constituents was expressed as a percentage by peak area. The identification and characterization of chemical compounds in ethanolic crude extract were based on GC retention time. AMDIS and NIST Version-Year 2011 was used MS data library and comparing the spectrum obtained through GC-MS compounds present in the plant's sample was identified.

Lactate Dehydrogenase Enzyme Inhibition Assay: [15] Assay mixture contained 0.3mM NADH, 2.0 mM pyruvate and 100 µL enzyme in a volume of 3.0 ml. The reaction was started by addition of enzyme. Lactate dehydrogenase inhibitory activity of test plant extracts was monitored spectrophotometrically following the absorbance at 340 nm under aerobic condition. The reaction mixture containing 0.3 mM NADH, 100 µL enzymes in the volume of 3.0 mL and a solution of test plant extracts in DMSO was incubated at room temperature for 15 min. The reaction was started by addition of 2 mM pyruvate, and l-lactate formation was then followed by a measure of decreasing absorbance at 340 nm. The inhibitory activity of each test compound was indicated by their IC_{50} values calculated using linear regression curve.

The percent inhibition of enzyme activity was calculated using standard formula [16].

Glycolate Oxidase Enzyme Inhibition Assay: [15] Each assay contained 200 µM potassium phosphate (pH 7.0), 1 mg of bovine serum albumin, 3 µM EDTA, 0.1 µM DCIP, enzyme and water to a volume of 3 ml and a solution of test plant extracts in DMSO was incubated at room temperature for 15 min.

The reaction was started by addition of 2 µM Sodium glycolate was then followed by a measure of the decrease in absorbance at 600 nm. The inhibitory activity of each test compound was indicated by their IC_{50} values calculated using linear regression curve. The percent inhibition of enzyme activity was calculated using standard formula [16].

Lineweaver-Burk Plots: This kinetics study was carried out in the absence and presence of active compounds with varying concentrations of substrate. The initial velocity was expressed as the absorbance decrease at 340 nm for lactate dehydrogenase and 600 nm for glycolate oxidase per 10 s in the assay and Lineweaver–Burk plot analysis was performed [17].

ADMET Predictions: ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) analyses constitutes the pharmacokinetics of a drug molecule [18]. In this study, prediction and significant descriptors of drug-likeness such as mutagenicity, toxicological dosage level and pharmacologically relevant properties of the compounds were predicted using Swissadme (http://www. swissadme.ch) and admetSAR (lmmd.ecust.edu. cn:8000) servers.

Data Analysis: The results are expressed as mean ± SD. Student's *t*-test and one way ANOVA were applicable and used to analyze the level of statistical significance between groups. $P<0.05$ were considered statistically significant. Linear regression analysis was used to calculate the IC_{50} values.

RESULTS:

Preliminary Phytochemical Screening: Preliminary phytochemical analysis showed the presence of major classes of secondary metabolites such as Alkaloids, tannins, saponins, flavonoids, cardiac glycosides, *etc*. in extracts **Table 1a** and **1b**.

TABLE 1A: PRELIMINARY PHYTOCHEMICAL SCREENINGS OF *BOERHAVIA DIFFUSA* (PUNARNAVA) ROOT EXTRACT

Phytoconstituents	Test/ Reagents	Ethyl acetate extract	Acetone extract	Ethanolic extract	Aqueous extract
Alkoids	Dragendorff's Test	+	+	+	+
	Hager's Test	+	+	+	+
	Wagner's Test	+	+	+	+
Proteins	Biuret Test	-	+	+	+
	Ninhydrin's Test	-	+	+	+
	Millon's Test	-	+	+	+
Tannins	Lead acetate	+	+	+	+
	Ferric chloride	+	+	+	+
Steroids	Salkowski test	-	-	-	-
Tarpenoids	Salkowski test	-	+	+	+
Carbohydrate	Fehling's Test	-	+	+	+
	Benedict's Test	-	+	+	+
Flavonoids	Shinoda test	+	+	+	-
	Lead Acetate Test	+	+	+	-
	Sodium Hydroxide Test	-	+	+	-
Glycoside	Keller-killani Test	-	-	-	-
Saponins	Foam Test	+	+	-	-

TABLE 1B: PRELIMINARY PHYTOCHEMICAL SCREENINGS OF *CRATAEVA NURVALA* (VARUNA) BARK EXTRACT

Phytoconstituents	Test/ Reagents	Ethyl acetate extract	Acetone extract	Ethanolic extract	Aqueous extract
Alkoids	Dragendorff's Test	+	+	+	+
	Hager's Test	+	+	+	+
	Wagner's Test	+	+	+	+
Proteins	Biuret Test	-	-	-	-
	Ninhydrin's Test	-	-	-	-
	Millon's Test	-	-	-	-
Tannins	Lead acetate	+	+	+	+
	Ferric chloride	+	+	+	+
Steroids	Salkowski test	+	+	+	+
Terpenoids	Salkowski test	-	-	-	-
Carbohydrate	Fehling's Test	-	+	+	+
	Benedict's Test	-	+	+	+
Flavonoids	Shinoda test	+	+	+	+
	Lead Acetate Test	+	+	+	+
	Sodium Hydroxide Test	+	+	+	+
Glycoside	Keller-killani Test	-	+	+	+
Saponins	Foam Test	-	+	+	+

Quantitative Estimation of phytoconstituents and Free Radical Scavenging Activity: The present study revealed a relatively good amount of phenols, flavonoids, coumarin and proantho-cyanidins contents of ethanolic extract of *Boerhaavia diffusa and Crataeva nurvala* as shown in **Table 2**. The correlation coefficient of total phenols, total flavonoids, coumarin, and total proanthocyanidins were (r =0.993, 0.984, 0.992 and 0.970) respectively **Fig. 1**.

TABLE 2: QUANTITATIVE ESTIMATION OF TOTAL PHENOLICS, FLAVONOIDS, FLAVONOLS, PROANTHOCYANIDINS AND COUMARIN (n=3)

Plants	Extracts	Total phenolics (GAE mg/g)	Total flavonoids (RE mg/g)	Total Proanthocyanidins (Catechin mg/g)	Total coumarin (coumarin mg/g)
Boerhaavia diffusa	Ethanolic	155.35 ± 2.50	75.19 ± 0.19	0.17 ± 0.25	0.527 ± 0.2
	Acetone	131.291 ± 1.5	56.91 ± 0.28	ND	0.453 ± 0.11
	Ethyl acetate	118.26 ± 1.45	32.77 ± 1.64	ND	ND
	Aqueous	85 ± 1.30	ND	ND	ND
Crataeva nurvala	Ethanolic	235.3 ± 1.94	11.38 ± 0.13	0.06 ± 0.11	0.48 ± 0.1
	Acetone	47.4 ± 0.73	7.09 ± 0.19	ND	0.44 ± 0.11
	Ethyl acetate	43.6 ± 0.87	3.7 ± 0.74	ND	0.32 ± 0.24
	Aqueous	45.8 ± 0.94	6.5 ± 0.76	ND	ND

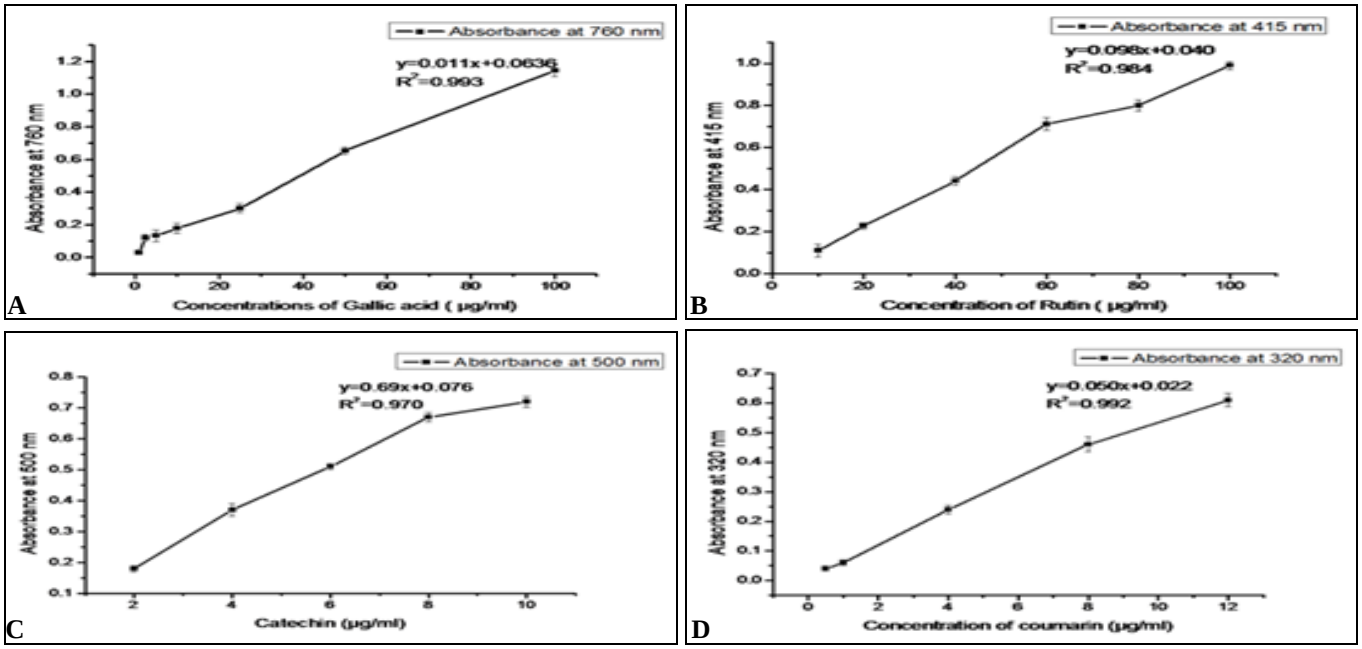


FIG. 1: STANDARD CALIBRATION CURVE FOR TOTAL STANDARD CALIBRATION CURVE FOR TOTAL PHENOLIC CONTENT (A), TOTAL FLAVONOIDS CONTENT FOR STANDARD RUTIN (B), TOTAL PROANTHOCYANIDINS CONTENT FOR STANDARD CATECHIN (C) AND TOTAL COUMARIN FOR STANDARD COUMARIN (D).

The antioxidant activity of *Boerhavia diffusa* and *Crataeva nurvala* extracts in solvents of varying polarity were measured regarding hydrogen donating or radical scavenging ability. The method is based on the reduction of alcoholic DPPH solutions in the presence of a hydrogen donating antioxidant. DPPH solutions show a strong absorption band at 517 nm appearing as a deep violet color. The absorption vanishes and the resulting decolorization is stoichiometric concerning the degree of reduction. The results of the free radical scavenging activity (%) of the *Boerhavia diffusa* and *Crataeva nurvala* extracts assessed by DPPH assay and amount of the sample needed for 50% inhibition of free radical activity, IC_{50} values were summarized in **Fig. 2** and **Table 3**.

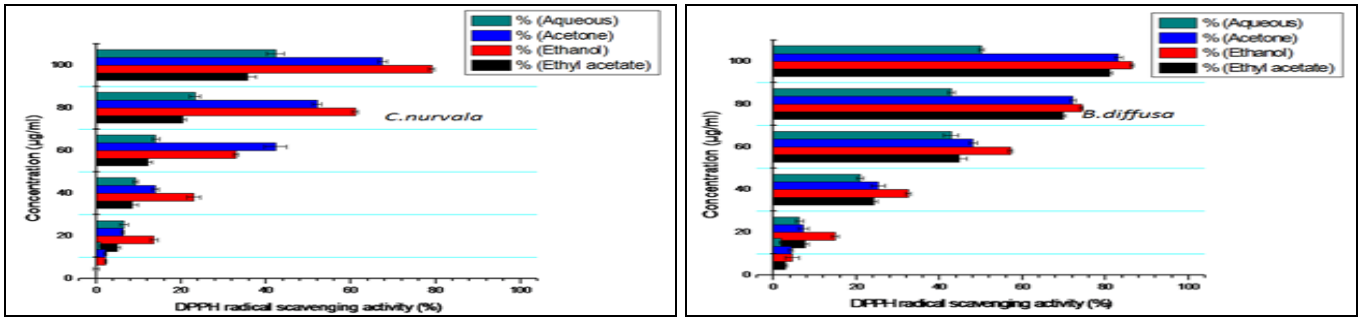


FIG. 2: FREE RADICAL SCAVENGING ACTIVITY (%) OF *BOERHAVIA DIFFUSA* AND *CRATAEVA NURVALA*

TABLE3. IC_{50} VALUE OF FREE RADICAL SCAVENGING ACTIVITY BY DPPH METHOD

Plants	IC_{50} of DPPH inhibition in µg/ml			
	Ethanolic extract	Acetone extract	Ethyl acetate extract	Aqueous extract
Boerhaavia diffusa	57.07 ± 0.34	63.27 ± 0.21	65.78 ± 0.24	91.18 ± 0.33
Crataeva nurvala	77.54 ± 0.29	69.31 ± 0.8	156.24 ± 1.7	134.96 ± 0.54
Ascorbic acid (Control)	14.82 ± 0.27			

Values are expressed in mean ± SD; *: $P<0.05$

Boerhaavia diffusa ethonolic extract was having phenolic (23.9 ± 0.25 mg/g of gallic acid equivalent), flavonoids (13.8 ± 0.13 mg/g of rutin equivalents), coumarin (1.48 ± 0.1 mg/g coumarin equivalent) and proanthocyanidins contents (1.6 ± 0.11 mg/g equivalent of catechin). The *Crataeva nurvala* extract showed the least concentration of phenol (18.2 ± 0.50 mg/g of gallic acid equivalent),

flavonoids (11.9 ± 0.19 mg/g of rutin equivalents), coumarin (1.527 ± 0.2 mg/g coumarin equivalent) and proanthocyanidins (1.7 ± 0.25 mg/g equivalent of catechin) contents.

Spectrophotometric Analysis: The qualitative UV-Vis and FTIR spectrum profile of *Boerhavia diffusa* and *Crataeva nurvala* ethanolic extracts were selected at a wavelength from 200 to 1000 nm due to the sharpness of the peaks and proper baseline. The profile showed the peaks with the absorption mentioned in **Fig. 3a** and **3b**. The results of UV-Vis and FTIR peak values and functional groups were represented in **Table 4** and **5**. The FTIR spectrum confirmed the presence of alcohols, phenols, alkanes, alkynes, alkyl halides, aldehydes, carboxylic acids, aromatics, nitro compounds and amines in different plant extracts.

Chemical Composition of Extracts by GC-MS Analysis: GC-MS chromatogram of the ethanolic extract of *Boerhaavia diffusa and Crataeva nurvala* **Fig. 4** showed peaks indicating the presence of phytochemical constituents. All the constituents were characterized and identified by comparison of the mass spectra of the constituents with the AMDIS and NIST Version-Year 2011 library. The active principles with their retention time (RT), molecular formula, molecular weight (MW) and concentration (%) are presented in **Table 6**.

TABLE 4: UV-VIS PEAK VALUES OF ETHANOLIC EXTRACTS OF *BOERHAVIA DIFFUSA* AND *CRATAEVA NURVALA*

Boerhavia diffusa.	Wavelength (nm)	Abs.
	1029	0.0779
	664	0.5630
	606	0.2517
	388	3.1294
	925	0.0114
	622	0.2335
	593	0.2437
	367	2.8693
Crataeva nurvala.	**Wavelength (nm)**	**Abs.**
	1029	0.0877
	338	3.7931

TABLE 5: FTIR PEAK VALUES AND FUNCTIONAL GROUPS OF *BOERHAVIA DIFFUSA* AND *CRATAEVA NURVALA*

Boerhavia diffusa	Peak values	Functional group
	3850.92	Unknown
	3835.69	Unknown
	3813.92	Unknown
	3708.51	Phenols
	3742.49	Phenols
	3390.83	Alcohols/Phenols
	2925.54	Alkanes
	2354.54	Unknown
	1651.41	Alkenes, Primary amines
	1050.53	Carboxylic acid
	1024.04	Aliphatic Amines
Crataeva nurvala	**Peak values**	**Functional group**
	3418.94	Alcohol (including phenol)
	2925.59	Alkanes
	2849.06	Carboxylic acids
	1621.59	Non-acid carbonyl
	1038.76	Alcohol

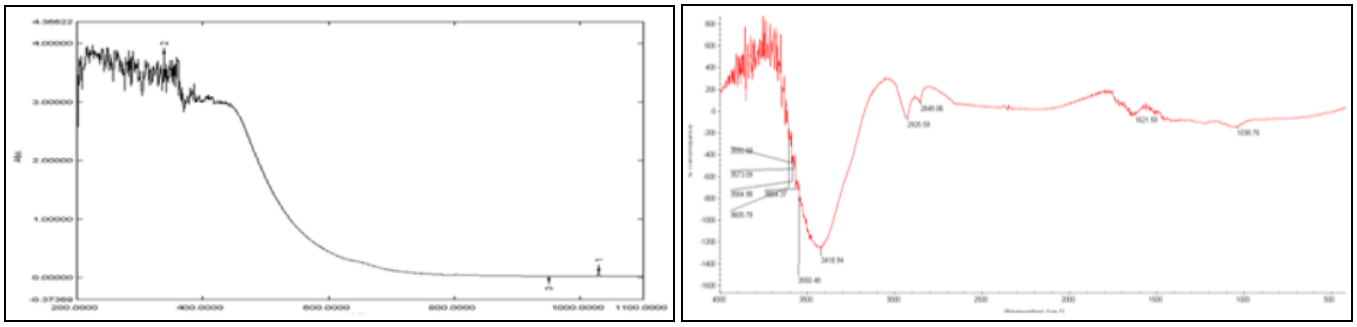


FIG. 3A: UV-VISIBLE AND FTIR SPECTRUM OF *CRATEVA NURVALA* (ETHANOLIC EXTRACT)

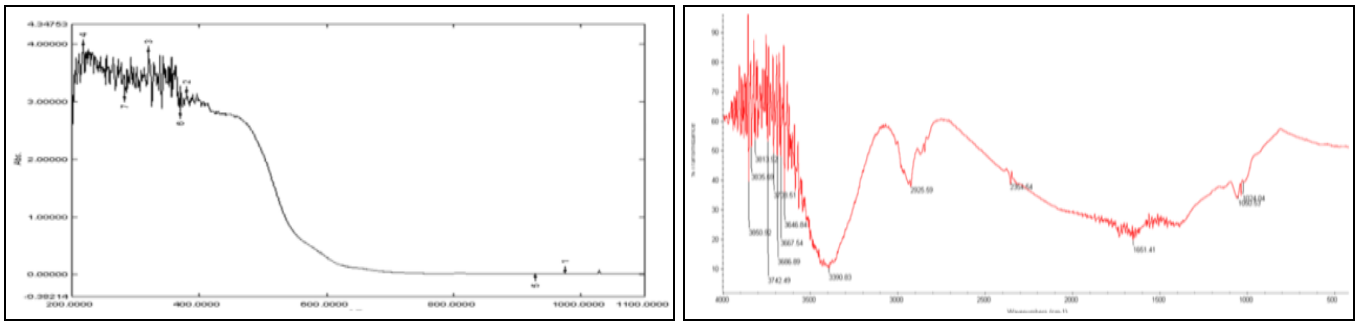


FIG. 3B: UV-VISIBLE AND FTIR SPECTRUM OF *BOERHAVIA.DIFFUSA* (ETHANOLIC EXTRACT)

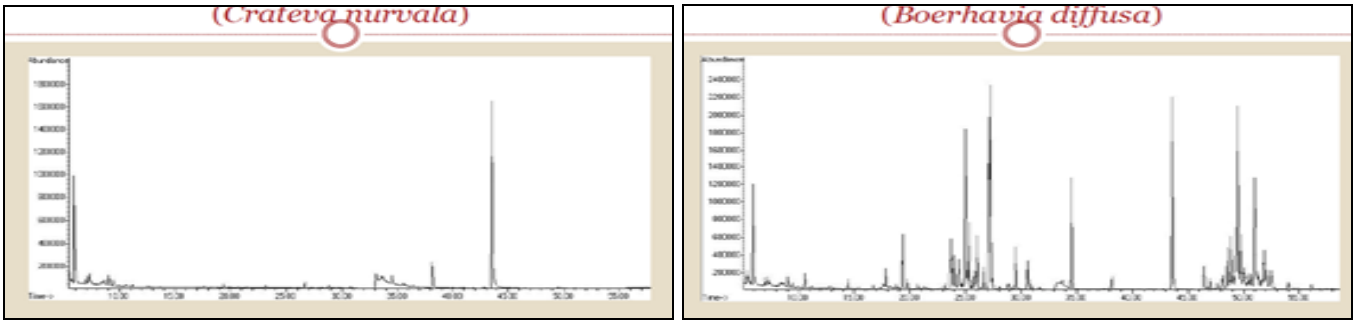


FIG. 4: TOTAL ION CHROMATOGRAM (GC-MS) OF PLANTS (ETHANOLIC EXTRACTS)

TABLE 6: BIOLOGICALLY ACTIVE CHEMICAL COMPOUNDS OF ETHANOLIC EXTRACT OF *BOERHAVIA DIFUSA* L. AND *CRATEVA NURVALA*. A: *BOERHAVIA DIFUSA L.* (ETHANOLIC EXTRACT)

CAS	Name of Compound	RT	Purity (%)	Model	Molecular weight	Molecular formula
17877428	>tromethamine (3,7-Dioxa-2,8-disilanonan-5-one, 2,2,8,8-tetramethyl-	10.5911	80%	73m/z	234.110	$C_9H_{22}O_3Si_2$
7449141	>Levoglucosan, tris(trimethylsilyl)-	23.1448	46%	333m/z	378.68	$C_{15}H_{34}O_5Si_3$
EPA-38016	>D-(-)-Fructopyranose, pentakis(trimethylsilyl) ether (isomer 1)	26.6323	57%	438m/z	541.06	$C_{21}H_{52}O_6Si_5$
EPA-38013	>D-Pinitol, pentakis(trimethylsilyl) ether	27.1282	52%	318m/z	555.08	$C_{22}H_{54}O_6Si_5$
6736943	>D-Galactose, 2,3,4,5,6-pentakis-O-(trimethylsilyl)-	28.0568	33%	TIC	570.10	$C_{22}H_{55}NO_6Si_5$
112390	>Hexadecanoic acid, methyl ester	28.7578	51%	185m/z	270.45	$C_{17}H_{34}O_2$
EPA-38017	>á-D-(+)-Talopyranose, pentakis(trimethylsilyl) ether	29.4953	65%	206m/z	541.06	$C_{21}H_{52}O_6Si_5$
EPA-38013	>á-D-(+)-Xylopyranose, tetrakis(trimethylsilyl) ether	29.5048	59%	204m/z	438.85	$C_{17}H_{42}O_5Si_4$
19126999	>Glucopyranose, 1,2,3,4,6-pentakis-O-(trimethylsilyl)-, D-	29.5184	48%	191m/z	541.06	$C_{21}H_{52}O_6Si_5$
18623228	>D-Xylose, tetrakis(trimethylsilyl)-	29.5286	44%	189m/z	467.89	$C_{18}H_{45}NO_5Si_4$
19126999	>Glucopyranose, 1,2,3,4,6-pentakis-O-(trimethylsilyl)-, D-	30.5667	51%	206m/z	541.06	$C_{21}H_{52}O_6Si_5$
18623228	>D-Xylose, tetrakis(trimethylsilyl)-	30.5796	55%	204m/z	467.89	$C_{18}H_{45}NO_5Si_4$
2582798	>Myo-Inositol, 1,2,3,4,5,6-hexakis-O-(trimethylsilyl)-	30.7273	52%	191m/z	613.24	$C_{24}H_{60}O_6Si_6$
19127152	>Mannose, 6-deoxy-2,3,4,5-tetrakis-O-(trimethylsilyl)-, L-	31.7783	41%	TIC	452.88	$C_{18}H_{44}O_5Si_4$
2582798	>Myo-Inositol, 1,2,3,4,5,6-hexakis-O-(trimethylsilyl)-	34.5787	56%	221m/z	613.24	$C_{24}H_{60}O_6Si_6$
19159252	>à-D-Glucopyranoside, 1,3,4,6-tetrakis-O-(trimethylsilyl)-á-D-fructofuranosyl 2,3,4,6-tetrakis-O-(trimethylsilyl)-	48.5498	45%	361m/z	919.74	$C_{36}H_{86}O_{11}Si_8$
EPA-38009	Sucrose, octakis(trimethylsilyl) ether	49.7474	42%	73m/z	361.00	$C_{36}H_{86}O_{11}Si_8$
19159252	>à-D-Glucopyranoside, 1,3,4,6-tetrakis-O-(trimethylsilyl)-á-D-fructofuranosyl 2,3,4,6-tetrakis-O-(trimethylsilyl)-	49.7573	40%	361m/z	919.74	$C_{36}H_{86}O_{11}Si_8$
EPA-38009	Sucrose, octakis(trimethylsilyl) ether	50.9886	39%	73m/z	361.00	$C_{36}H_{86}O_{11}Si_8$
2625469	ß-Sitosterol trimethylsilyl ether	59.6181	76%	129m/z	486.88	$C_{32}H_{58}OSi$
545471	>Lupeol	59.9548	55%	105m/z	426.71	$C_{30}H_{50}O$

B: *CRATEVA NURVALA* BUCH-HAM (ETHANOLIC EXTRACT)

CAS	Name of Compound	RT	Purity (%)	Model	Molecular weight	Molecular formula
74978260	>D-Altro-2-Heptulose, 1,3,4,5,6,7-hexakis-O-(trimethylsilyl)-	23.1447	51	205 m/z	643.269	$C_{25}H_{62}O_1Si_6$
EPA-380168	>D-(-)-Fructopyranose, pentakis(trimethylsilyl) ether (isomer 1)	26.6577	58	205 m/z	541.0615	$C_{21}H_{52}O_6Si_5$
112390	>Hexadecanoic acid, methyl ester	28.8189	43	TIC	270.45	$C_{17}H_{34}O_2$
EPA-366063	>N-methylene-n-octadecylamine	33.0643	44	182 m/z	283.53	$C_{19}H_{41}N$
2582798	>Myo-Inositol, 1,2,3,4,5,6-hexakis-O-(trimethylsilyl)-	34.5095	35	307 m/z	613.24	$C_{24}H_{60}O_6Si_6$
1617705	>Lup-20(29)-en-3-one	59.7533	85	109 m/z	424.70	$C_{30}H_{48}O$
545471	>Lupeol	59.9996	88	189 m/z	426.71	$C_{30}H_{50}O$

TABLE 7: BIOACTIVITY OF PHYTOCOMPONENTS IDENTIFIED IN THE ETHANOLIC EXTRACT OF *BOERHAVIA DIFUSA* L. AND *CRATEVA NURVALA*

***Boerhavia difusa* L.**	
Name of the compound	**Biological Activity****
>Hexadecanoic acid, methyl ester	Antioxidant, Hypocholesterolemic, Nematicide, Pesticide, Lubricant, Antiandrogenic, Hemolytic, 5-alpha-reductase inhibitor
>Myo-Inositol, 1,2,3,4,5,6-hexakis-O-(trimethylsilyl)-	Antidepression, liver problems, panic disorders, and diabetes
>D-Pinitol, pentakis(trimethylsilyl) ether	Smart drug, Anticancer, CNS-depressant, Decalcifier, Decongestant, coronary dialator, Decrease oxalate excretion, Decrease Lactate/pyruvate ratio, Dehydrogenase inhibitor, diuretic, provide vitamin D
>Glucopyranose, 1,2,3,4,6-pentakis-O-(trimethylsilyl)-, D-	Decrease oxalate excretion, Anticancer, Decalcifier, coronary dilator, Decrease Lactate/pyruvate ratio, diuretic, inhibit production of uric acid, oncostatic, oxidase inhibitor, xo inhibitor
>Mannose, 6-deoxy-2,3,4,5-tetrakis-O-(trimethylsilyl)-, L-	Low oxalate, Anti-LDL, Anticancer, Antitumor, Decrease Lactate/pyruvate ratio, laxative, litholytic, decrease oxalate excretion, and inhibit production of uric acid, xo inhibitor
β-Sitosterol trimethylsilyl ether	Antioxidant, analgesic, anti-inflammatory, hypocholesterolemic
>Lupeol	Anti-inflammatory, arthritis problem, diabetes, cardiovascular ailments, renal disorder, hepatic toxicity, antimicrobial, anti-leukemia
Sorbopyranose, 1,2,3,4,5-pentakis-O-(trimethylsilyl)-, L-	Larvicidal, Lactation, Anticancer, Laxative, Anti-leukemia, Litholytic, Lymphatic diseases, oxidase inhibitor
Crateva nurvala	
Name of the compound	**Biological Activity****
>Hexadecanoic acid, methyl ester	Antioxidant, Hypocholesterolemic, Nematicide, Pesticide, Lubricant, Antiandrogenic, Hemolytic, 5-alpha-reductase inhibitor
>Myo-Inositol, 1,2,3,4,5,6-hexakis-O-(trimethylsilyl)-	Antidepression, liver problems, panic disorders, and diabetes
>Lup-20(29)-en-3-one	Anti-leukamia, antibacterial, Antioxidant, Antitumor.
Sorbopyranose, 1,2,3,4,5-pentakis-O-(trimethylsilyl)-, L-	Larvicidal, Lactation, Anticancer, Laxative, Anti-leukemia, Litholytic, Lymphatic diseases, oxidase inhibitor
>Lupeol	Anti-inflammatory, arthritis problem, diabetes, cardiovascular ailments, renal disorder, hepatic toxicity, antimicrobial, anti-leukemia

(**Activity source: Dr. Duke's Phytochemical and Ethnobotanical Database)

Glycolate Oxidase and Lactate Dehydrogenase Inhibition and Mode of Inhibition: The experimental evidence indicates that all ethanolic extracts of *Boerhavia diffusa* and *Crateva nurvala* showed a good activity profile for inhibition of glycolate oxidase and lactate dehydrogenase as indicated by IC_{50} (µM) **Fig. 5** and **6**; **Table 8**. V_{max} and Km of *B. diffusa* and *C. nurvala* (ethanolic extracts) for lactate dehydrogenase (LDH) Inhibition and glycolate oxidase inhibition are mentioned in **Fig. 7** and **8**; **Table 9** and **10**.

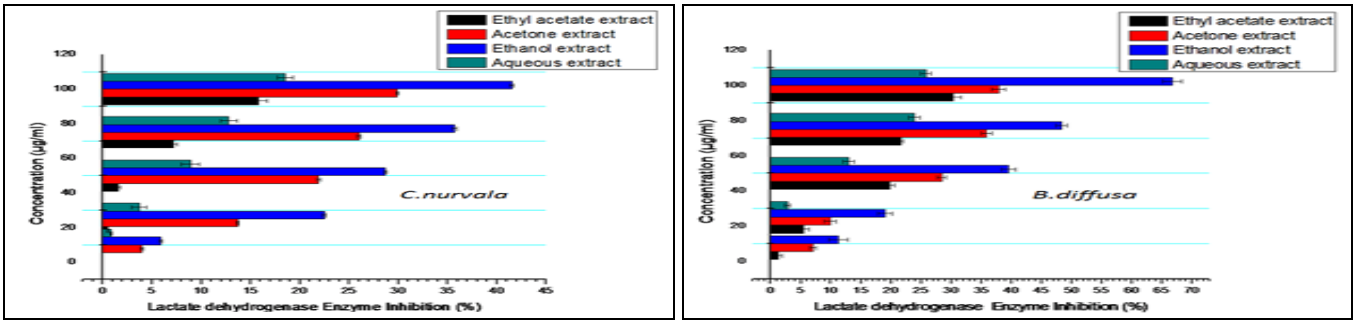


FIG. 5: LACTATE DEHYDROGENASE ENZYME INHIBITION ACTIVITY (%)

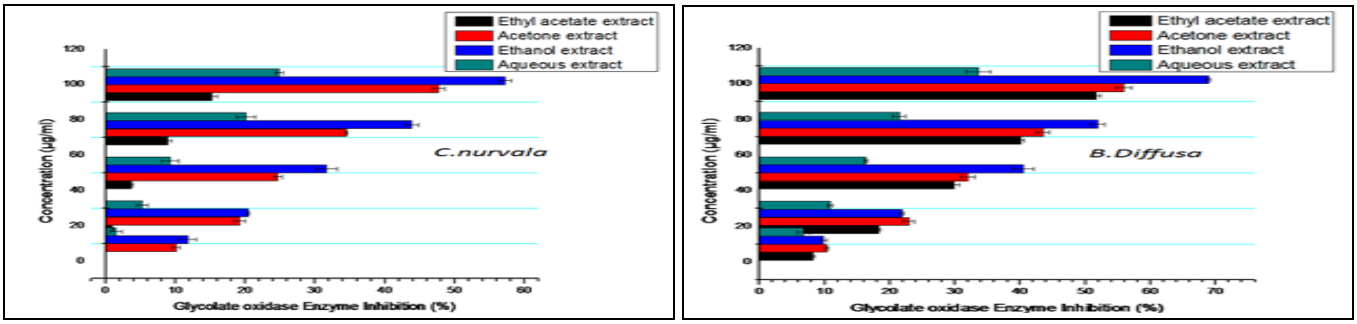


FIG. 6: GLYCOLATE OXIDASE ENZYME INHIBITION ACTIVITY (%)

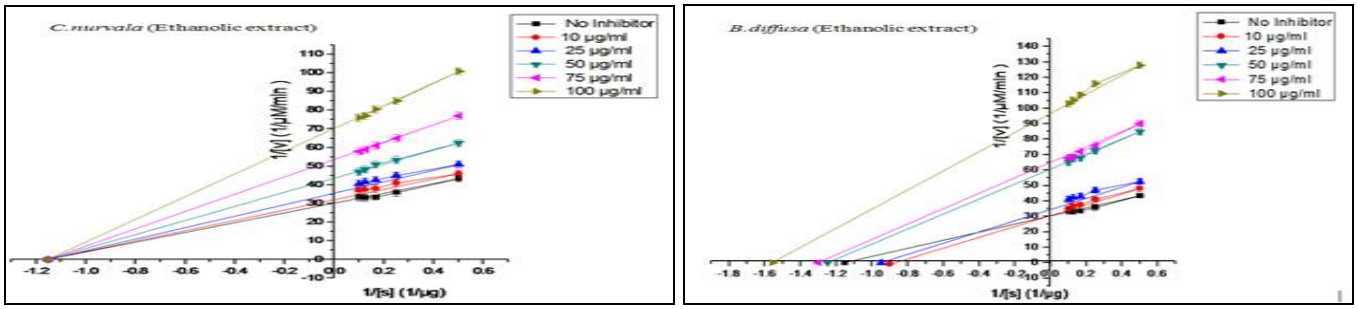


FIG. 7: LINEWEAVER-BURK PLOT FOR ENZYME GLYCOLATE OXIDASE

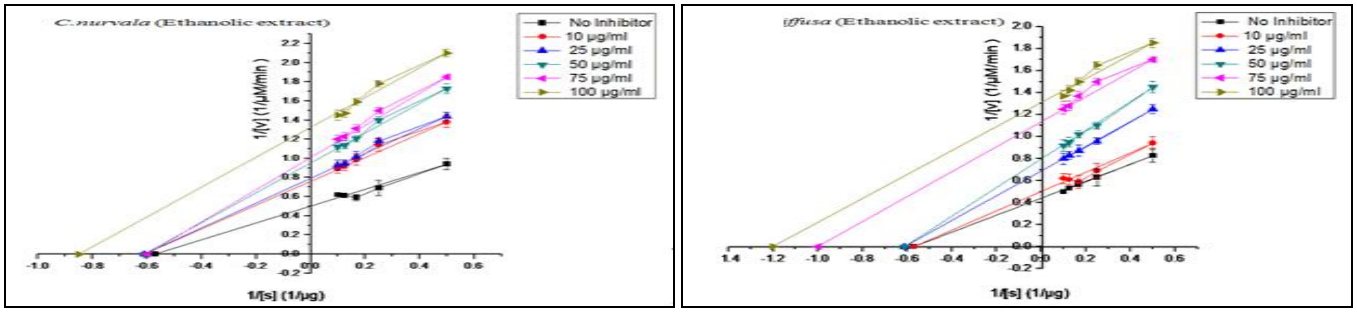


FIG. 8: LINEWEAVER-BURK PLOT FOR ENZYME LACTATE DEHYDROGENASE

TABLE 8: IC_{50} VALUE OF ENZYME GLYCOLATE OXIDASE AND LACTATE DEHYDROGENASE

I: GLYCOLATE OXIDASE

Plants	IC_{50} of glycolate oxidase (GOX) inhibition in µg/ml			
	Ethanolic extract	Acetone extract	Ethyl acetate extract	Aqueous extract
Boerhavia diffusa	69.53 ± 0.37	87.53 ± 0.96	95.50 ± 1.4	167.11 ± 0.21
Crataeva nurvala	86.50 ± 0.17	110.09 ± 0.59	313.84 ± 1.53	192.60 ± 1.35

II: LACTATE DEHYDROGENASE

Plants	IC_{50} of Lactate dehydrogenase (LDH) inhibition in µg/ml			
	Ethanolic extract	Acetone extract	Ethyl acetate extract	Aqueous extract
Boerhavia diffusa	73.67 ± 1.21	116.27 ± 1.1	158.52 ± 0.84	161.31 ± 1.52
Crataeva nurvala	149.50 ± 1.60	178.74 ± 0.80	318.56 ± 1.82	260.41 ± 0.98

Values are expressed in mean ± SD; *: $P<0.05$

TABLE 9: V_{max} AND Km OF *B. DIFFUSA* AND *C. NURVALA* (ETHANOLIC EXTRACTS) FOR LACTATE DEHYDROGENASE (LDH) INHIBITION

Samples	Concentrations (µg/ml)	Vmax (µg/min)	Km (µg/ml)	Type of Inhibition	IC_{50} value (µg/ml)
B. diffusa	10	2.38	1.754	Mixed type (	73.67
	25	1.42	1.639	between	
	50	1.25	1.639	Uncompetitive to	
	75	0.90	1	Non-competitive	
	100	0.76	0.833	Inhibition)	
C. nurvala	10	1.31	1.63	Non-competitive	149.50
	25	1.25	1.63		
	50	1.08	1.63		
	75	1	1.66		
	100	0.76	1.17		

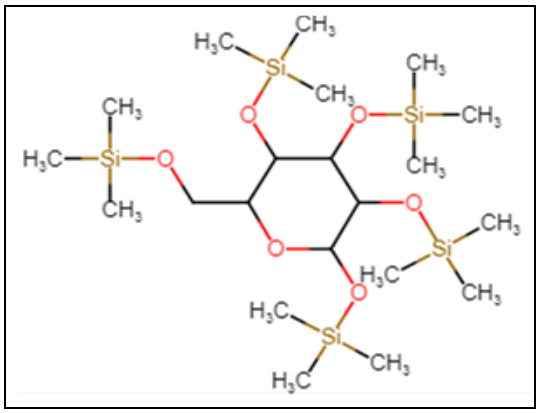
TABLE 10: V_{max} AND Km OF *B. DIFFUSA* AND *C. NURVALA* (ETHANOLIC EXTRACTS) FOR GLYCOLATE OXIDASE INHIBITION

Samples	Concentrations (µg/ml)	Vmax (µg/min)	Km (µg/ml)	Type of Inhibition	IC_{50} value (µg/ml)
B. diffusa	10	0.033	1.11	Uncompetitive	69.53
	25	0.028	1.05		
	50	0.016	0.8		
	75	0.0153	0.76		
	100	0.0105	0.64		
C. nurvala	10	0.031	0.86	Non-competitive	86.50
	25	0.028			
	50	0.022			
	75	0.018			
	100	0.0142			

ADMET Predictions: The potential ADME profiles of the compounds as predicted using an admetSAR server, while the distribution profile of the compounds as obtained from the admetSAR server is shown in **Table 11**. Computational study for the prediction of the relevant properties influencing bioactivity of the lead compounds was performed. The ADME properties of the compounds were evaluated, and the selected properties are linked to metabolism, cell permeation.

TABLE 11: ADMET PREDICTIONS

Compound 1: à-D-(+)-Talopyranose, pentakis (trimethylsilyl) ether



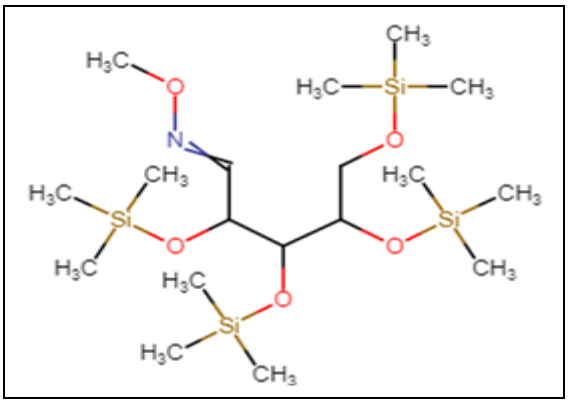
ADMET PREDICTED PROFILE - CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.8921
Human Intestinal Absorption	HIA-	0.8550
Caco-2 Permeability	Caco2+	0.5487
P-glycoprotein Substrate	Non-substrate	0.5765
P-glycoprotein Inhibitor	Inhibitor	0.5502
	Non-inhibitor	0.9673
Renal Organic Cation Transporter	Non-inhibitor	0.8459
	Distribution	
Subcellular localization	Mitochondria	0.6510
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8354
CYP450 2D6 Substrate	Non-substrate	0.8224
CYP450 3A4 Substrate	Substrate	0.5799
CYP450 1A2 Inhibitor	Non-inhibitor	0.8483
CYP450 2C9 Inhibitor	Non-inhibitor	0.8869
CYP450 2D6 Inhibitor	Non-inhibitor	0.9098
CYP450 2C19 Inhibitor	Non-inhibitor	0.7904
CYP450 3A4 Inhibitor	Non-inhibitor	0.9682
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9587
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9544
	Non-inhibitor	0.9550
AMES Toxicity	AMES toxic	0.5875
Carcinogens	Non-carcinogens	0.7804
Fish Toxicity	Low FHMT	0.9002
Tetrahymena Pyriformis Toxicity	Low TPT	0.5591
Honey Bee Toxicity	High HBT	0.7733
Biodegradation	Not ready biodegradable	0.6437
Acute Oral Toxicity	III	0.5012
Carcinogenicity (Three-class)	Non-required	0.6119

ADMET PREDICTED PROFILE - REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-1.9609	LogS
Caco-2 Permeability	0.7929	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.5012	LD_{50}, mol/kg
Fish Toxicity	1.4568	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.2406	$pIGC_{50}$, ug/L

Compound 2: Arabinopyranose, tetrakis-O-(trimethylsilyl)-, à-D-



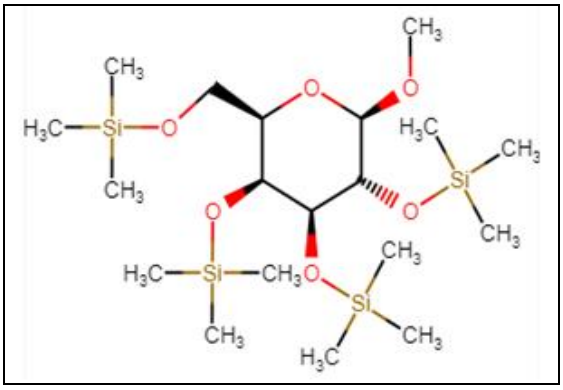
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.8827
Human Intestinal Absorption	HIA+	0.6426
Caco-2 Permeability	Caco2-	0.5405
P-glycoprotein Substrate	Non-substrate	0.6829
P-glycoprotein Inhibitor	Non-inhibitor	0.6470
	Non-inhibitor	0.8045
Renal Organic Cation Transporter	Non-inhibitor	0.8434
	Distribution	
Subcellular localization	Mitochondria	0.7063
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8123
CYP450 2D6 Substrate	Non-substrate	0.8160
CYP450 3A4 Substrate	Substrate	0.5652
CYP450 1A2 Inhibitor	Non-inhibitor	0.8670
CYP450 2C9 Inhibitor	Non-inhibitor	0.8627
CYP450 2D6 Inhibitor	Non-inhibitor	0.8911
CYP450 2C19 Inhibitor	Non-inhibitor	0.7965
CYP450 3A4 Inhibitor	Non-inhibitor	0.9008
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9624
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9649
	Non-inhibitor	0.8872
AMES Toxicity	Non-AMES toxic	0.5000
Carcinogens	Carcinogens	0.6667
Fish Toxicity	Low FHMT	0.8148
Tetrahymena pyriformis Toxicity	Low TPT	0.7104
Honey Bee Toxicity	High HBT	0.6987
Biodegradation	Not ready biodegradable	0.6385
Acute Oral Toxicity	III	0.5320
Carcinogenicity (Three-class)	Non-required	0.5694

ADMET PREDICTED PROFILE - REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-1.6569	LogS
Caco-2 Permeability	0.7028	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.6942	LD_{50}, mol/kg
Fish Toxicity	1.4991	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.2513	$pIGC_{50}$, ug/L

Compound 3: Glucofuranoside, methyl 2,3,5,6-tetrakis-O-(trimethylsilyl)-, à-D-



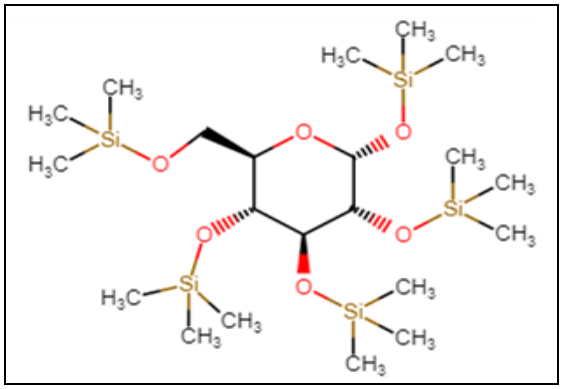
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.8921
Human Intestinal Absorption	HIA-	0.8550
Caco-2 Permeability	Caco2+	0.5487
P-glycoprotein Substrate	Non-substrate	0.5765
P-glycoprotein Inhibitor	Inhibitor	0.5502
	Non-inhibitor	0.9673
Renal Organic Cation Transporter	Non-inhibitor	0.8459
	Distribution	
Subcellular localization	Mitochondria	0.6510
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8354
CYP450 2D6 Substrate	Non-substrate	0.8224
CYP450 3A4 Substrate	Substrate	0.5799
CYP450 1A2 Inhibitor	Non-inhibitor	0.8483
CYP450 2C9 Inhibitor	Non-inhibitor	0.8869
CYP450 2D6 Inhibitor	Non-inhibitor	0.9098
CYP450 2C19 Inhibitor	Non-inhibitor	0.7904
CYP450 3A4 Inhibitor	Non-inhibitor	0.9682
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9587
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9544
	Non-inhibitor	0.9550
AMES Toxicity	AMES toxic	0.5875
Carcinogens	Non-carcinogens	0.7804
Fish Toxicity	Low FHMT	0.9002
Tetrahymena Pyriformis Toxicity	Low TPT	0.5591
Honey Bee Toxicity	High HBT	0.7733
Biodegradation	Not ready biodegradable	0.6437
Acute Oral Toxicity	III	0.5012
Carcinogenicity (Three-class)	Non-required	0.6119

ADMET PREDICTED PROFILE - REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-1.9609	LogS
Caco-2 Permeability	0.7929	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.5012	LD_{50}, mol/kg
Fish Toxicity	1.4568	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.2406	$pIGC_{50}$, ug/L

Compound 4: Glucopyranose, 1,2,3,4,6-pentakis-O-(trimethylsilyl)-, D-



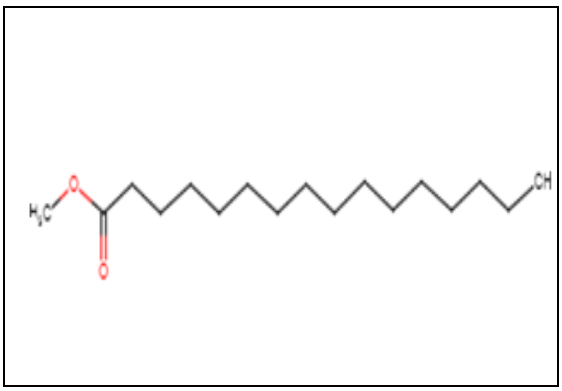
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.8921
Human Intestinal Absorption	HIA-	0.8550
Caco-2 Permeability	Caco2+	0.5487
P-glycoprotein Substrate	Non-substrate	0.5765
P-glycoprotein Inhibitor	Inhibitor	0.5502
	Non-inhibitor	0.9673
Renal Organic Cation Transporter	Non-inhibitor	0.8459
	Distribution	
Subcellular localization	Mitochondria	0.6510
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8354
CYP450 2D6 Substrate	Non-substrate	0.8224
CYP450 3A4 Substrate	Substrate	0.5799
CYP450 1A2 Inhibitor	Non-inhibitor	0.8483
CYP450 2C9 Inhibitor	Non-inhibitor	0.8869
CYP450 2D6 Inhibitor	Non-inhibitor	0.9098
CYP450 2C19 Inhibitor	Non-inhibitor	0.7904
CYP450 3A4 Inhibitor	Non-inhibitor	0.9682
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9587
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9544
	Non-inhibitor	0.9550
AMES Toxicity	AMES toxic	0.5875
Carcinogens	Non-carcinogens	0.7804
Fish Toxicity	Low FHMT	0.9002
Tetrahymena Pyriformis Toxicity	Low TPT	0.5591
Honey Bee Toxicity	High HBT	0.7733
Biodegradation	Not ready biodegradable	0.6437
Acute Oral Toxicity	III	0.5012
Carcinogenicity (Three-class)	Non-required	0.6119

ADMET PREDICTED PROFILE - REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-1.9609	LogS
Caco-2 Permeability	0.7929	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.5012	LD_{50}, mol/kg
Fish Toxicity	1.4568	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.2406	$pIGC_{50}$, ug/L

Compound 5: Hexadecanoic acid, methyl ester



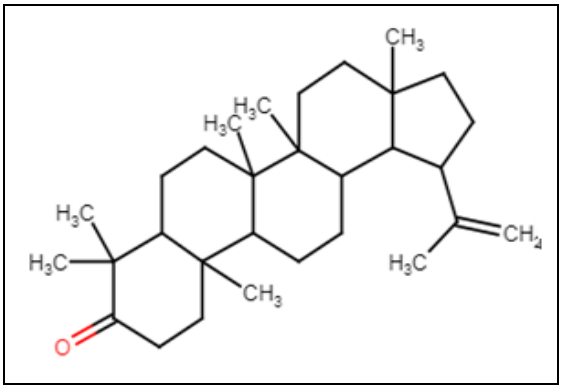
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9848
Human Intestinal Absorption	HIA+	0.9881
Caco-2 Permeability	Caco2+	0.8141
P-glycoprotein Substrate	Non-substrate	0.7061
P-glycoprotein Inhibitor	Non-inhibitor	0.8951
	Non-inhibitor	0.7988
Renal Organic Cation Transporter	Non-inhibitor	0.8908
	Distribution	
Subcellular localization	Mitochondria	0.4276
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8648
CYP450 2D6 Substrate	Non-substrate	0.8885
CYP450 3A4 Substrate	Non-substrate	0.6454
CYP450 1A2 Inhibitor	Non-inhibitor	0.5548
CYP450 2C9 Inhibitor	Non-inhibitor	0.9329
CYP450 2D6 Inhibitor	Non-inhibitor	0.9502
CYP450 2C19 Inhibitor	Non-inhibitor	0.9524
CYP450 3A4 Inhibitor	Non-inhibitor	0.9773
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9176
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9104
	Non-inhibitor	0.8787
AMES Toxicity	Non-AMES toxic	0.9765
Carcinogens	Carcinogens	0.5347
Fish Toxicity	High FHMT	0.8790
Tetrahymena Pyriformis Toxicity	High TPT	0.8990
Honey Bee Toxicity	High HBT	0.7623
Biodegradation	Ready biodegradable	0.8747
Acute Oral Toxicity	III	0.8589
Carcinogenicity (Three-class)	Non-required	0.7269

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-3.3987	LogS
Caco-2 Permeability	1.2386	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	1.4915	LD_{50}, mol/kg
Fish Toxicity	0.8236	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	0.6648	$pIGC_{50}$, ug/L

Compound 6: Lup-20(29)-en-3-one



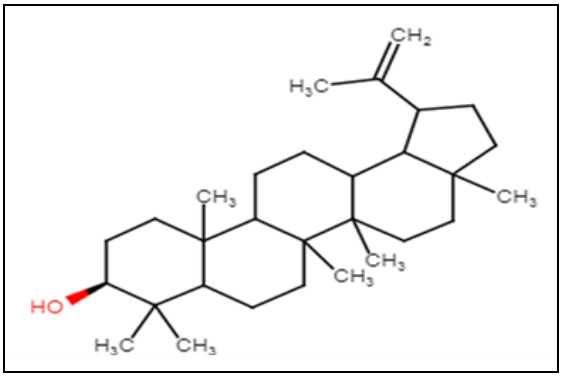
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9583
Human Intestinal Absorption	HIA+	1.0000
Caco-2 Permeability	Caco2+	0.8153
P-glycoprotein Substrate	Substrate	0.6211
P-glycoprotein Inhibitor	Inhibitor	0.8621
	Non-inhibitor	0.5218
Renal Organic Cation Transporter	Non-inhibitor	0.6970
	Distribution	
Subcellular localization	Mitochondria	0.4922
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8462
CYP450 2D6 Substrate	Non-substrate	0.8797
CYP450 3A4 Substrate	Substrate	0.7584
CYP450 1A2 Inhibitor	Non-inhibitor	0.8231
CYP450 2C9 Inhibitor	Non-inhibitor	0.8670
CYP450 2D6 Inhibitor	Non-inhibitor	0.9589
CYP450 2C19 Inhibitor	Non-inhibitor	0.5701
CYP450 3A4 Inhibitor	Non-inhibitor	0.8408
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.7721
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.8295
	Non-inhibitor	0.7390
AMES Toxicity	Non-AMES toxic	0.9620
Carcinogens	Non-carcinogens	0.8938
Fish Toxicity	High FHMT	0.9970
Tetrahymena Pyriformis Toxicity	High TPT	0.8992
Honey Bee Toxicity	High HBT	0.8614
Biodegradation	Not ready biodegradable	0.9776
Acute Oral Toxicity	III	0.6116
Carcinogenicity (Three-class)	Non-required	0.4936

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-4.0561	LogS
Caco-2 Permeability	1.8360	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.4398	LD_{50}, mol/kg
Fish Toxicity	-0.2851	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	1.0119	$pIGC_{50}$, ug/L

Compound 7: Lupeol



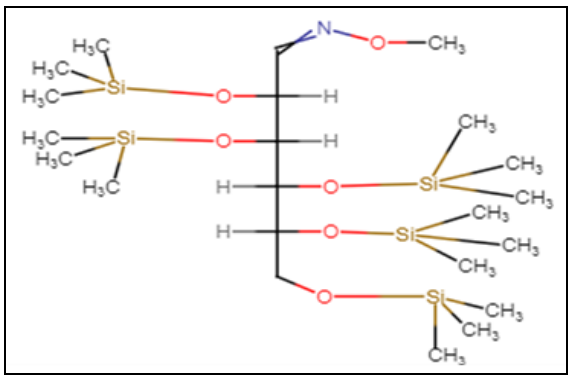
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9592
Human Intestinal Absorption	HIA+	0.9974
Caco-2 Permeability	Caco2+	0.8499
P-glycoprotein Substrate	Substrate	0.6969
P-glycoprotein Inhibitor	Inhibitor	0.6979
	Non-inhibitor	0.8203
Renal Organic Cation Transporter	Non-inhibitor	0.7710
	Distribution	
Subcellular localization	Lysosome	0.5245
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8184
CYP450 2D6 Substrate	Non-substrate	0.9047
CYP450 3A4 Substrate	Substrate	0.7687
CYP450 1A2 Inhibitor	Non-inhibitor	0.8619
CYP450 2C9 Inhibitor	Non-inhibitor	0.8200
CYP450 2D6 Inhibitor	Non-inhibitor	0.9506
CYP450 2C19 Inhibitor	Non-inhibitor	0.7320
CYP450 3A4 Inhibitor	Non-inhibitor	0.8441
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.7562
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.8599
	Non-inhibitor	0.7580
AMES Toxicity	Non-AMES toxic	0.9420
Carcinogens	Non-carcinogens	0.9188
Fish Toxicity	High FHMT	0.9972
Tetrahymena Pyriformis Toxicity	High TPT	0.9185
Honey Bee Toxicity	High HBT	0.8611
Biodegradation	Not ready biodegradable	0.9793
Acute Oral Toxicity	III	0.8578
Carcinogenicity (Three-class)	Non-required	0.5755

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-4.4139	LogS
Caco-2 Permeability	1.6517	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	3.3838	LD_{50}, mol/kg
Fish Toxicity	-0.0530	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	0.9639	$pIGC_{50}$, ug/L

Compound 8: Mannose, 2,3,4,5,6-pentakis-O-(trimethylsilyl)-, D-



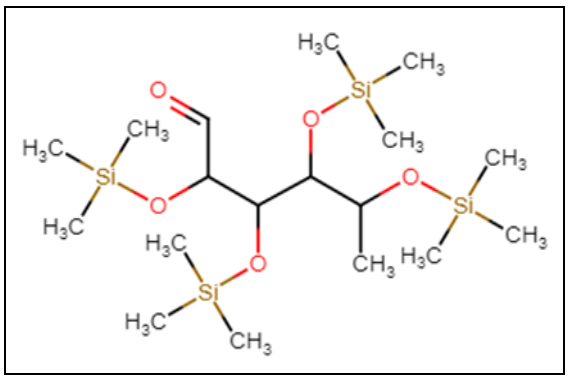
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.8827
Human Intestinal Absorption	HIA+	0.6426
Caco-2 Permeability	Caco2-	0.5405
P-glycoprotein Substrate	Non-substrate	0.6829
P-glycoprotein Inhibitor	Non-inhibitor	0.6470
	Non-inhibitor	0.8045
Renal Organic Cation Transporter	Non-inhibitor	0.8434
	Distribution	
Subcellular localization	Mitochondria	0.7063
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8123
CYP450 2D6 Substrate	Non-substrate	0.8160
CYP450 3A4 Substrate	Substrate	0.5652
CYP450 1A2 Inhibitor	Non-inhibitor	0.8670
CYP450 2C9 Inhibitor	Non-inhibitor	0.8627
CYP450 2D6 Inhibitor	Non-inhibitor	0.8911
CYP450 2C19 Inhibitor	Non-inhibitor	0.7965
CYP450 3A4 Inhibitor	Non-inhibitor	0.9008
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9624
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9649
	Non-inhibitor	0.8872
AMES Toxicity	Non-AMES toxic	0.5000
Carcinogens	Carcinogens	0.6667
Fish Toxicity	Low FHMT	0.8148
Tetrahymena Pyriformis Toxicity	Low TPT	0.7104
Honey Bee Toxicity	High HBT	0.6987
Biodegradation	Not ready biodegradable	0.6385
Acute Oral Toxicity	III	0.5320
Carcinogenicity (Three-class)	Non-required	0.5694

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-1.6569	LogS
Caco-2 Permeability	0.7028	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.6942	LD_{50}, mol/kg
Fish Toxicity	1.4991	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.2513	$pIGC_{50}$, ug/L

Compound 9: Mannose, 6-deoxy-2,3,4,5-tetrakis-O-(trimethylsilyl)



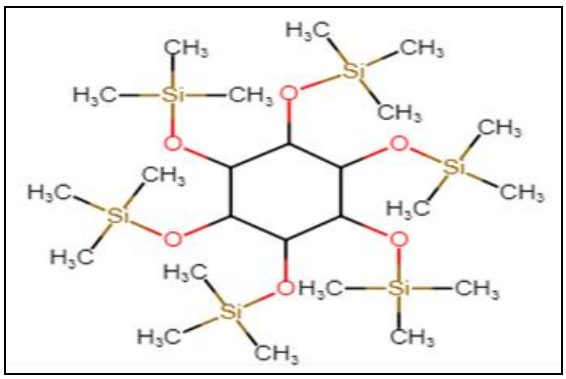
ADMET PREDICTED PROFILE - CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9473
Human Intestinal Absorption	HIA+	0.7642
Caco-2 Permeability	Caco2-	0.5238
P-glycoprotein Substrate	Non-substrate	0.7040
P-glycoprotein Inhibitor	Non-inhibitor	0.8254
	Non-inhibitor	0.9217
Renal Organic Cation Transporter	Non-inhibitor	0.9392
	Distribution	
Subcellular localization	Mitochondria	0.7589
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8449
CYP450 2D6 Substrate	Non-substrate	0.8613
CYP450 3A4 Substrate	Non-substrate	0.5215
CYP450 1A2 Inhibitor	Non-inhibitor	0.8736
CYP450 2C9 Inhibitor	Non-inhibitor	0.8713
CYP450 2D6 Inhibitor	Non-inhibitor	0.9349
CYP450 2C19 Inhibitor	Non-inhibitor	0.7864
CYP450 3A4 Inhibitor	Non-inhibitor	0.9446
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9441
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9871
	Non-inhibitor	0.9439
AMES Toxicity	Non-AMES toxic	0.6867
Carcinogens	Carcinogens	0.7110
Fish Toxicity	Low FHMT	0.6785
Tetrahymena Pyriformis Toxicity	Low TPT	0.9571
Honey Bee Toxicity	High HBT	0.8134
Biodegradation	Ready biodegradable	0.5187
Acute Oral Toxicity	III	0.4870
Carcinogenicity (Three-class)	Non-required	0.6385

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-0.6165	LogS
Caco-2 Permeability	0.8255	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.2175	LD_{50}, mol/kg
Fish Toxicity	2.3096	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-1.0982	$pIGC_{50}$, ug/L

Compound 10: Myo-Inositol, 1,2,3,4,5,6-hexakis-O-(trimethylsilyl)-



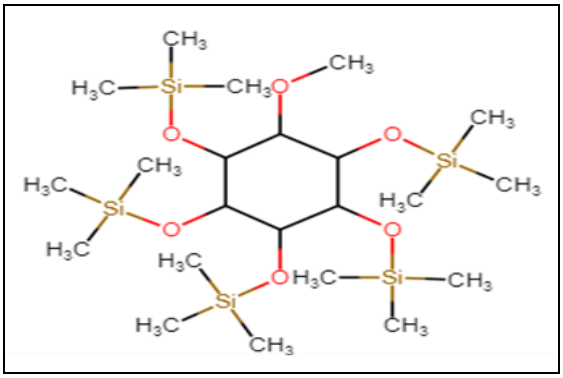
ADMET PREDICTED PROFILE – CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9112
Human Intestinal Absorption	HIA+	0.7965
Caco-2 Permeability	Caco2+	0.5800
P-glycoprotein Substrate	Non-substrate	0.6926
P-glycoprotein Inhibitor	Non-inhibitor	0.6467
	Non-inhibitor	0.9721
Renal Organic Cation Transporter	Non-inhibitor	0.9037
	Distribution	
Subcellular localization	Mitochondria	0.7017
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8327
CYP450 2D6 Substrate	Non-substrate	0.8141
CYP450 3A4 Substrate	Substrate	0.5518
CYP450 1A2 Inhibitor	Non-inhibitor	0.8293
CYP450 2C9 Inhibitor	Non-inhibitor	0.8767
CYP450 2D6 Inhibitor	Non-inhibitor	0.9374
CYP450 2C19 Inhibitor	Non-inhibitor	0.6998
CYP450 3A4 Inhibitor	Non-inhibitor	0.9147
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9436
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9590
	Non-inhibitor	0.9482
AMES Toxicity	Non-AMES toxic	0.7651
Carcinogens	Non-carcinogens	0.5987
Fish Toxicity	Low FHMT	0.7004
Tetrahymena Pyriformis Toxicity	Low TPT	0.9318
Honey Bee Toxicity	High HBT	0.8376
Biodegradation	Not ready biodegradable	0.7998
Acute Oral Toxicity	III	0.4080
Carcinogenicity (Three-class)	Non-required	0.6017

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-2.4271	LogS
Caco-2 Permeability	1.0786	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.2058	LD_{50}, mol/kg
Fish Toxicity	2.0719	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.5608	$pIGC_{50}$, ug/L

Compound 11: Pinotol, pentakis(trimethylsilyl) ether



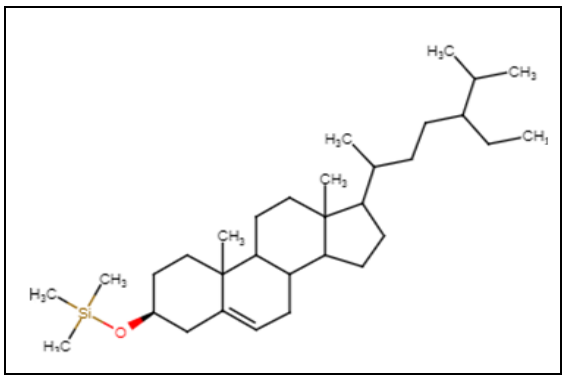
ADMET PREDICTED PROFILE - CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9112
Human Intestinal Absorption	HIA+	0.7965
Caco-2 Permeability	Caco2+	0.5800
P-glycoprotein Substrate	Non-substrate	0.6926
P-glycoprotein Inhibitor	Non-inhibitor	0.6467
	Non-inhibitor	0.9721
Renal Organic Cation Transporter	Non-inhibitor	0.9037
	Distribution	
Subcellular localization	Mitochondria	0.7017
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8327
CYP450 2D6 Substrate	Non-substrate	0.8141
CYP450 3A4 Substrate	Substrate	0.5518
CYP450 1A2 Inhibitor	Non-inhibitor	0.8293
CYP450 2C9 Inhibitor	Non-inhibitor	0.8767
CYP450 2D6 Inhibitor	Non-inhibitor	0.9374
CYP450 2C19 Inhibitor	Non-inhibitor	0.6998
CYP450 3A4 Inhibitor	Non-inhibitor	0.9147
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9436
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9590
	Non-inhibitor	0.9482
AMES Toxicity	Non-AMES toxic	0.7651
Carcinogens	Non-carcinogens	0.5987
Fish Toxicity	Low FHMT	0.7004
Tetrahymena Pyriformis Toxicity	Low TPT	0.9318
Honey Bee Toxicity	High HBT	0.8376
Biodegradation	Not ready biodegradable	0.7998
Acute Oral Toxicity	III	0.4080
Carcinogenicity (Three-class)	Non-required	0.6017

ADMET PREDICTED PROFILE – REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-2.4271	LogS
Caco-2 Permeability	1.0786	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.2058	LD_{50}, mol/kg
Fish Toxicity	2.0719	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.5608	$pIGC_{50}$, ug/L

Compound 12: *β*-Sitosterol trimethylsilyl ether



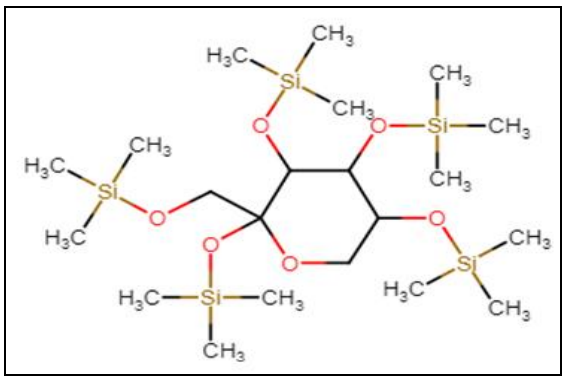
ADMET PREDICTED PROFILE - CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.9673
Human Intestinal Absorption	HIA+	0.9950
Caco-2 Permeability	Caco2+	0.6221
P-glycoprotein Substrate	Substrate	0.6710
P-glycoprotein Inhibitor	Inhibitor	0.8476
	Inhibitor	0.5000
Renal Organic Cation Transporter	Non-inhibitor	0.6922
	Distribution	
Subcellular localization	Lysosome	0.4124
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8778
CYP450 2D6 Substrate	Non-substrate	0.8112
CYP450 3A4 Substrate	Substrate	0.7701
CYP450 1A2 Inhibitor	Non-inhibitor	0.7685
CYP450 2C9 Inhibitor	Non-inhibitor	0.7888
CYP450 2D6 Inhibitor	Non-inhibitor	0.8759
CYP450 2C19 Inhibitor	Non-inhibitor	0.6383
CYP450 3A4 Inhibitor	Non-inhibitor	0.6911
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.5319
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Strong inhibitor	0.5310
	Non-inhibitor	0.6227
AMES Toxicity	Non-AMES toxic	0.7433
Carcinogens	Non-carcinogens	0.7878
Fish Toxicity	High FHMT	0.9943
Tetrahymena Pyriformis Toxicity	High TPT	0.9391
Honey Bee Toxicity	High HBT	0.8492
Biodegradation	Not ready biodegradable	0.9686
Acute Oral Toxicity	III	0.6021
Carcinogenicity (Three-class)	Non-required	0.5465

ADMET PREDICTED PROFILE - REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-4.6478	LogS
Caco-2 Permeability	1.2774	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.4351	LD_{50}, mol/kg
Fish Toxicity	0.2419	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	1.0064	$pIGC_{50}$, ug/L

Compound 13: Sorbopyranose, 1,2,3,4,5-pentakis-O-(trimethylsilyl)-, L-



ADMET PREDICTED PROFILE - CLASSIFICATION

Model	Result	Probability
	Absorption	
Blood-Brain Barrier	BBB+	0.8947
Human Intestinal Absorption	HIA-	0.9220
Caco-2 Permeability	Caco2+	0.5163
P-glycoprotein Substrate	Substrate	0.5421
P-glycoprotein Inhibitor	Inhibitor	0.5955
	Non-inhibitor	0.9550
Renal Organic Cation Transporter	Non-inhibitor	0.8222
	Distribution	
Subcellular localization	Mitochondria	0.6088
	Metabolism	
CYP450 2C9 Substrate	Non-substrate	0.8525
CYP450 2D6 Substrate	Non-substrate	0.8204
CYP450 3A4 Substrate	Substrate	0.5929
CYP450 1A2 Inhibitor	Non-inhibitor	0.8638
CYP450 2C9 Inhibitor	Non-inhibitor	0.8690
CYP450 2D6 Inhibitor	Non-inhibitor	0.9072
CYP450 2C19 Inhibitor	Non-inhibitor	0.7558
CYP450 3A4 Inhibitor	Non-inhibitor	0.9481
CYP Inhibitory Promiscuity	Low CYP Inhibitory Promiscuity	0.9583
	Excretion	
	Toxicity	
Human Ether-a-go-go-Related Gene Inhibition	Weak inhibitor	0.9242
	Non-inhibitor	0.8791
AMES Toxicity	Non-AMES toxic	0.5000
Carcinogens	Non-carcinogens	0.7529
Fish Toxicity	Low FHMT	0.8427
Tetrahymena Pyriformis Toxicity	Low TPT	0.6449
Honey Bee Toxicity	High HBT	0.7305
Biodegradation	Not ready biodegradable	0.8833
Acute Oral Toxicity	III	0.5684
Carcinogenicity (Three-class)	Non-required	0.5874

ADMET PREDICTED PROFILE - REGRESSION

Model	Value	Unit
	Absorption	
Aqueous solubility	-2.3267	LogS
Caco-2 Permeability	0.6844	LogPapp, cm/s
	Distribution	
	Metabolism	
	Excretion	
	Toxicity	
Rat Acute Toxicity	2.5394	LD_{50}, mol/kg
Fish Toxicity	1.8088	pLC_{50}, mg/L
Tetrahymena Pyriformis Toxicity	-0.1521	$pIGC_{50}$, ug/L

DISCUSSION: The concentrations of phenolic, flavonoids compounds were high in all extracts while proanthocyanidin and coumarin contents were low when compared with other compounds. The differences in polarity of the antioxidant components are noticeably the reason why phenolic compounds and antioxidant activity of the extract correlate. Polyphenolic compounds which influence the radical scavenging, inhibition of hydrolytic and oxidative enzymes and also act as anti-inflammatory agent [19]. The mechanisms of action of flavonoids are through scavenging or chelating process [20, 21.] Flavonoids are free radical scavengers, super antioxidants, and potent water soluble which prevent oxidative cell damage and have strong anti-cancer activity [22] and provide anti-inflammatory actions [23]. Cardiac glycosides are drugs and can be used in the treatment of congestive heart failure and cardiac arrhythmia. Tannins possess astringent, anti-inflammatory, antidiarrhoeal, antioxidant and antimicrobial activities [24].

Among all extracts, the phenolic content decreased in the order of ethanol>acetone>ethyl acetate> distilled water. Hence it was observed that ethanol is a good solvent for the extraction of phenols and these results are in agreement with the previously reported study [25, 26]. The interest in the phenols has increased outstandingly due to their prominent free radical scavenging activity. Polyphenols possess ideal structural chemistry for free radical scavenging activities which have shown to be more effective antioxidant *in-vitro* than vitamins E and C on a molar basis [27]. Phenol compounds could be classified as simple phenols, a single aromatic ring bearing at least one hydroxyl group and polyphenols with at least two phenol subunits like flavonoids or three and more phenol subunits called tannins [28].

The phenolic compounds of plant origin showed their anti-oxidative effect by various mechanisms, including their ability to scavenge free radicals or activate various antioxidant enzymes and inhibit oxidase [29]. Flavonoids are reported to be responsible for antimicrobial activity associated with some ethnomedicinal plants [30]. Flavonoids play an important role in effective antioxidant and show strong anticancer activities [31, 32]. Flavonoids are water-soluble antioxidants and possess pharmacological roles including anti-microbial, anti-inflammatory, cardioprotective, anti-allergic and anti-cancer activities [33]. The concentration of the flavonoids in plant extracts depends on the polarity of solvents used for the extracts which support the present results. The result showed a higher flavonoid content of ethyl acetate extract than of others extracts. It was expected that ethanol extract would have the higher antioxidant activity than that of other extracts, but surprisingly the acetone and ethyl acetate extracts have the good antioxidant activity in spite with less phenolic, flavonoids content **Table 3**. Since the natural antioxidant include many different compounds such as phenol, nitrous compounds, carotenoids and many unidentified compounds [34], compounds other than the phenolic compounds may be responsible for antioxidant activity in the water, ethanolic or acetone extract or sometimes the structure of flavonoids in ethyl acetate extract may decrease its antioxidant activity. Rice-Evans *et al*. showed that hydroxyl groups, the amount of conjugation and its kind, are two important factors in the antioxidant potential of phenolic compounds.

Stronger antioxidants usually are more conjugated and have more hydroxyl groups that make the antioxidant strong enough to scavenge the free radicals. The occurrence of peaks at 234-676 nm reveals the presence of phenolic and alkaloids.

The FTIR spectrum was used to identify the functional group of the active components based on the peak value in the region of infrared radiation. The biological activities listed **Table 7** are based on Dr.Duke's Phytochemical and Ethnobotanical Databases by Dr. Duke of the Agricultural Research Service/USDA [35]. Phytol was proven to exhibit antioxidant and antinociceptive effects [36, 37]. These sterols even blocked inflammatory cytokines and induced apoptosis [38-40]. Lupeol exhibited marked anti-inflammatory and anticancer properties [41]. Several investigations revealed that lupeol blocks tumorigenesis by affecting molecular growth pathways which are involved in cell proliferation and cell death [41].

In-vivo and *in-vitro* tests revealed the potent anti-mutagenic property of lupeol [42-44]. Identification of these compounds in the plant serves as the basis in determining the possible health benefits of the plant leading to further biologic and pharmacologic studies concerning urolithiasis and related renal disorders. In the case of glycolate oxidase inhibition, *Boerhavia diffusa* was found to be uncompetitive inhibitors as Km and V_{max} consequently decreased with increased inhibitor concentration. With *Boerhavia diffusa* there were Km values were constant while V_{max} consequently decreased with increased inhibitor concentration thus confirming a non-competitive type of inhibition by the Lineweaver–Burk equation.

In case of lactate dehydrogenase, *Crateva nurvala* was found to be non-competitive inhibitors as Km values were constant while V_{max} consequently decreased with increased inhibitor concentration, while *Boerhavia diffusa* showed the mixed type of inhibition (between uncompetitive to non-competitive Inhibition). Glycolate oxidase and lactate dehydrogenase are key enzymes for oxalate synthesis. These enzymes are present in liver peroxisome. If inhibition of these key enzymes takes place then ultimately oxalate synthesis get to stop, and calcium oxalate stone formation will not occur. Calcium oxalate stone is a major type of stone occurs in patients.

GC-MS results reveal compounds namely, D-Pinitol, pentakis (trimethylsilyl) ether; Mannose, 6-deoxy-2,3,4,5-tetrakis-O-(trimethylsilyl)-, L-; Arabinopyranose, tetrakis-O-(trimethylsilyl)-, à- D; Sorbopyranose, 1, 2, 3, 4, 5-pentakis-O-(trimethylsilyl)-, L-; >Lupeol;>Mannose, 6-deoxy-2, 3, 4, 5-tetrakis-O-(trimethylsilyl)-, L-; >Glucopyranose, 1, 2, 3, 4, 6-pentakis-O-(trimethylsilyl)-, D- clearly showed diuretic, litholytic, decrease oxalate excretion, decrease lactate/pyruvate ratio, dehydrogenase inhibitor, oxidase inhibitor and decalcifier nature. Inhibition of glycolate oxidase and lactate dehydrogenase may occur due to these phytoconstituents. This is the first report as per the author's knowledge.

CONCLUSION: Urolithiasis is a major problem worldwide, and significant advances in the treatment of urolithiasis have been made in the past few decades. However, the currently available treatment options are not completely satisfactory and associated with many side effects. In this study, we have collected the plants mentioned in literature on the ethnomedical use in the treatment of urolithiasis. These plants exhibit anti-urolithic effect by different mechanisms, such as Oxalate synthesizing enzyme (Glycolate oxidase and Lactate dehydrogenase) inhibition, crystallization inhibition mechanism of action, anti-inflammatory activity, and antioxidant activity.

Also, plant extract's analyzed constituents by UV-FTIR, GC-MS and ADMET profiling showed promising Glycolate oxidase and Lactate dehydrogenase inhibition and are found to be phenolic, glycosides and flavonoids. However, isolation of individual phytochemical constituents and subjecting it to biological activity (*in-vivo* study) will give fruitful results.

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