WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Comparision of *Invitro* Antioxidant Activity of *Fagonia Cretica* and *Huperzia Serrata*

Souvik Dutta[1], Dr. G Tulja Rani[2], K Swarna Priya[3], K Tejaswini[4], P Lavanya[5]

[1]Assistant Professor, Department of Pharmacology, Malla Reddy Pharmacy College, Hyderabad
[2]Principal and Professor, Department of Pharmaceutical analysis, Malla Reddy Pharmacy College, Hyderabad
[3,4,5]Student, Malla Reddy Pharmacy College, Hyderabad

ABSTRACT

The present study compares the in vitro antioxidant activity of *Fagonia cretica* and *Huperzia serrata* using standard assays such as Hydrogen Peroxide (H_2O_2) scavenging, Ferric Reducing Antioxidant Power (FRAP), and Total Phenolic Content (TPC). Both plants are traditionally known for their medicinal properties, particularly in oxidative stress-related conditions. Extracts were prepared using methanol, and ascorbic acid was used as the standard reference. Results indicated that *Fagonia cretica* exhibited higher H_2O_2 scavenging and FRAP values, while *Huperzia serrata* showed greater phenolic content. The variation in antioxidant potential is attributed to the differences in phytochemical composition. This comparative analysis highlights the therapeutic potential of both species, suggesting *Fagonia cretica* as more potent in reactive oxygen species neutralization.

Keywords: *Fagoniacretica*, *Huperziaserrata*, antioxidant activity, invitro assays, FRAP, H_2O_2scavenging, total phenolic content, medicinal plants, oxidative stress, phytochemicals.

INTRODUCTION

Antioxidants are groups of compounds that neutralize free radicals and reactive oxygen species (ROS) in the cell [1]. Antioxidant activity in food and beverages has become one of the most interesting features in the science community. These antioxidants provide protection against damage caused by free radicals played important roles in the devolopment of many chronic disease including cardiovasculer diseases, aging, heart disease, cancer, inflammation [2]

A free radical can be defined as any molecular species capable of independent existence that containsanunpairedelectroninanatomicorbital. Thepresenceofanunpairedelectronresults in certain common properties that are shared by most radicals. Many radicals are unstable and highlyreactive. Theycaneither donateanelectronto or accept anelectronfromother molecules, therefore behaving as oxidants or reductants.[3]

The most important oxygen-containing free radicals in many disease states are hydroxyl radical, superoxide anion radical, hydrogen peroxide, oxygen singlet, hypochlorite, nitric oxide radical, and peroxynitrite radical. These are highly reactive species, capable in the nucleus, and in the membranes of cells of damaging biologically relevant molecules such as DNA, proteins, carbohydrates, and lipids.[4]

Free radicals attack important macromolecules leading to cell damage and homeostatic disruption. Targets of free radicals include all kinds of molecules in the body. Among them, lipids, nucleic acids, and proteins are the major targets.

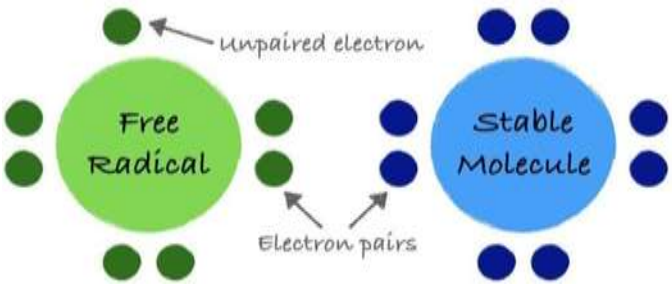


Fig.No.01FreeRadical Production of free radicals in the human body

Either the body's natural, vital metabolic activities or external factors including exposure to X- rays, ozone, cigarettes, air pollution, and industrial chemicals are the source of free radicals and other ROS..[5] Both enzymatic and nonenzymatic processes in cells result in the constant production offree radicals. The respiratorychain, phagocytosis, prostaglandin synthesis, and the cytochrome P-450systemare examples ofenzymatic reactions that canproduce free radicals..[6] In addition to ionizing reactions, nonenzymatic interactions of oxygen with organic molecules can also produce free radicals**.**Some internally generated sources of free radicals are [7]

- Mitochondria
- Xanthineoxidase
- Peroxisomes
- Inflammation
- Phagocytosis
- Exercise
- Ischemia/reperfusioninjury
- Cigarette smoke
- Environmentalpollutants

Free radicalreactions are expected to produce progressive adverse changes that accumulate with age throughout the body. Such “normal” changes with age are relatively common to all. These are manifested as diseases at certain ages determined by genetic and environmental factors. Cancer andatherosclerosis, two major causesofdeath, aresalient “freeradical”diseases. Studies on atherosclerosis reveal the probability that the disease may be due to free radical reactions involving diet-derived lipids in the arterial wall and serum to yield peroxides and other substances. These compounds induce endothelial cell injury and produce changes in the arterial walls.[8]

OXIDATIVESTRESS

Thephrasereferstothestateofoxidativedamage that ariseswhenthere isanimbalance between the production of free radicals and antioxidant defenses. [9] Damage to a variety of molecular species, such as lipids, proteins, and nucleic acids, is linked to oxidative stress, which results froman imbalance betweenthe generationoffree radicalsand antioxidant defenses. [10]Tissues damagedbytrauma,infection,heatinjury,hypertoxia,toxins,andexcessiveexercisemay experience short-term oxidative stress. Increased levels of the enzymes that make ROS (such as xanthine oxidase, lipogenase, and cyclooxygenase), phagocyte activation, the release offree iron and copper ions, orabreakdownoftheelectrontransport chainsofoxidativephosphorylationare allproduced by these wounded tissues.The imbalance between ROS and the antioxidant defense systemhas been implicated in the development, promotion, and progression of cancer as well as the adverse effects ofradiation and chemotherapy. Age-related eye illness, neurologicaldiseases including Parkinson's disease, and diabetes mellitus have all been linked to ROS induction and consequences. [11]

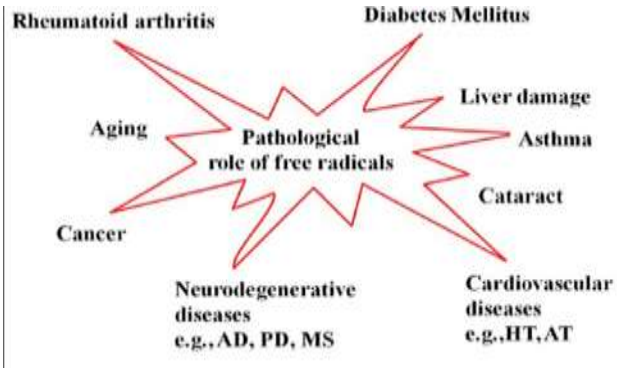


Fig.No.02RoleofFreeRadicals

ANTIOXIDANTS

Anantioxidant is a molecule stable enough to donate an electron to a rampaging free radical and neutralize it, thus reducing its capacity to damage. These antioxidants delay or inhibit cellular damage mainly through their free radical scavenging property.[12] These low-molecular-weight antioxidants can safely interact with free radicals and terminate the chain reaction before vital molecules are damaged. Some of such antioxidants, including glutathione, ubiquinol, and uric acid, are produced during normal metabolism in the body.[13] Other lighter antioxidants are found in the diet. Although there are severalenzymes systemwithin the bodythat scavenge free radicals, the principle micronutrient (vitamins) antioxidants are vitamin E (α-tocopherol),vitamin C (ascorbic acid), and B-carotene.[14] The body cannot manufacture these micronutrients, so they must be supplied in the diet.

Antioxidants act as radical scavenger, hydrogen donor, electron donor, peroxide decomposer, singlet oxygen quencher, enzyme inhibitor, synergist, and metal-chelating agents. Both enzymatic and nonenzymatic antioxidantsexist in the intracellular and extracellular environment to detoxify ROS.[15]

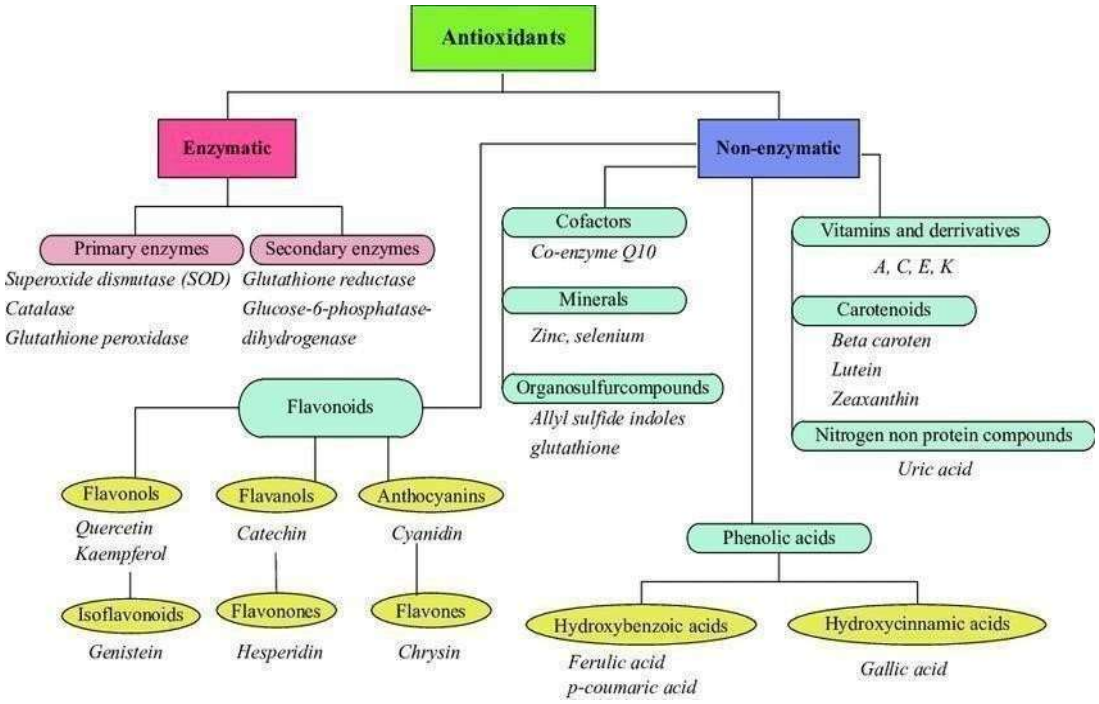


Fig.no.03:Classificationofantioxidants

The **sourcesofnaturalantioxidants** are mainlyplants, i.e., edible vegetables, fruits, spices, and herbs, which are rich in vitamins, phenolic compounds, carotenoids, and microelements [16,17,18,19]. However, it should be emphasized that the antioxidant activity is different for different varieties and morphological parts of natural resources. In addition, the activity ofnatural products is influenced by many other factors, such as climatic and soil conditions or harvest time. They hinder the standardization of natural products to a large extent.

MECHANISMOFACTIONOFANTIOXIDANTS

Two principle mechanisms of action have been proposed for antioxidants.[20] The first is a chain- breaking mechanism by which the primary antioxidant donates an electron to the free radical present in the systems. The second mechanism involves removal of ROS/reactivenitrogen species initiators (secondary antioxidants) by quenching chain-initiating catalyst. Antioxidants may exert their effect on biological systems by different mechanisms including electron donation, metal ion chelation, co-antioxidants, or by gene expression regulation.[21]

LITERATUREREVIEWONFAGONIACRETICA

S.no	Study/Author	Year	Activities	Findings
1	Rehmanetal.	2024	Anti-oxidant activity	RecentstudieshaveconfirmedFagonia cretica's strong antioxidant properties that may help in reducing oxidative stress and improving overall health.
2	Khanetal.	2023	Toxicological Assessment	Conducted a safety study on the toxicologicalprofileofFagonia*cretica*. Found no significant toxicity in animal models at therapeutic doses.
3	Javedetal.	2023	Antioxidantand Anticancer Properties	Exploredthepotentialanticanceractivity of *Fagonia cretica* extracts. Found significant antioxidant activity and inhibition of cancer cell proliferation.
4	Khan,M.,etal	2022	Antioxidantand antimicrobial properties of Fagonia cretica	FoundthatFagoniacreticaexhibited strongantioxidantandantimicrobial properties,supportingitstherapeutic uses in traditional medicine.

LITERATUREREVIEWONHUPERZIASERRATA

S.no	Study/Author	Year	Activities	Findings
1	Liuetal.	2024	Neuroprotective andAntioxidant Effects	Foundthatit reducedoxidativestressand improvedcognitivefunctionbyenhancing acetylcholine levels.
2	Jiangetal.	2024	Lycopodium alkaloids and cholinesterase inhibition	Isolated16lycopodiumalkaloids, including 10 new compounds. Notably,theyexhibitedpotentAChE inhibitionsuggestingitspotentialfor Alzheimer's treatment.
3	Guoet al.	2024	Endophytic fungusproducing Huperzine A	Identified a new endophytic fungus (Staphylotrichum) in *H. serrata* that producesHuperzineAatayieldof5.20 µg/g, offering a sustainable source for this compound.
4	Zhao etal.	2024	Total alkaloid extract from *Huperziaserrata* (HsAE)	Showedgoodantioxidantactivityand free radical scavenging.

PLANTPROFILE

I. FAGONIACRETICA

ScientificName:*Fagoniacretica* L.

Family: Zygophyllaceae

CommonNames: Dhamasa,Virgin'sMantle,Fagonia Arabica

Morphology&Habitat:

Fagonia cretica is a small, thorny, perennial herb or subshrub found predominantly in arid and semi-aridregions, including parts of Africa, theMiddleEast,andSouthAsia.Theplant typically grows low to the ground, forming spreading mats with small, trifoliate leaves and purple or pinkish flowers. It thrives in rockyor sandy soil and survives well under drought conditions.

TraditionalUses:

Traditionallyused in Unani and Ayurvedic medicine, *Fagonia cretica* is known for treating fever, inflammation, skindisorders,andliverailments.It iswidelyconsumedasadecoctionfor general detoxification and to boost immunity.

PhytochemicalConstituents:

The plant is rich in bioactive compounds such as flavonoids (quercetin, apigenin), saponins, alkaloids, tannins,terpenoids ,andglycosides. Thesecompoundsarelargelyresponsibleforits therapeutic effects.

PharmacologicalActivities:

- **Antioxidant:**Duetoitshighflavonoidandphenoliccontent, *Fagoniacretica*exhibits strong free radical scavenging activity.
- **Anticancer:**Severalstudieshave indicatedcytotoxiceffectsagainstcancercelllines.

- **Anti-inflammatory&Antimicrobial:**Ithasshownsignificantactivityagainst microbial strains and inflammation in various experimental models.

ExtractionMethods:

Forphytochemicalandantioxidant studies,extractionistypicallydoneusingsolventslike methanol, ethanol, or water through maceration, Soxhlet, or cold infusion.

II. HUPERZIASERRATA

ScientificName: *Huperziaserrata*(Thunb.exMurray)Trevis.

Family: Lycopodiaceae

CommonNames: Chineseclubmoss,toothed clubmoss

Morphology&Habitat:

Huperziaserrata isaslow-growing,perennial,spore-bearingplant foundprimarilyinEast Asia, especially in China, Japan, and Korea. It typically grows in shaded, mountainous, and forested areas. The plant has erect or creeping stems with small, serrated, scale-like leaves arranged spirally.

TraditionalUses:

In traditional Chinese medicine, it has been used for centuries to treat fever, muscle strain, swelling,andmemory-relateddisorders. Itisalsousedasageneral tonic for improving blood circulation and supporting neurological health.

PhytochemicalConstituents:

Theplant isarichsourceofalkaloids,particularly **huperzineA**,whichisthe most studied compound. Other constituents include flavonoids, triterpenes, and phenolic acids.

PharmacologicalActivities:

- **Neuroprotective:**HuperzineAisapotent,reversibleacetylcholinesteraseinhibitor, which enhances memory and cognitive function.
- **Antioxidant:**Showsstrongantioxidantactivitybyneutralizingfreeradicalsandreducing oxidative stress.
- **Anti-inflammatory&Anti-convulsant:**Exhibitsprotectiveeffectsinneurologicaland inflammatory conditions.

ExtractionMethods:

Alkaloids,especiallyhuperzineA,arecommonlyextractedusingethanolormethanolby maceration, Soxhlet extraction, or ultrasonication, followed by purification steps.

MATERIALSANDMETHODOLOGY

ApparatusRequired: Testtubes/Microplates(96-well),Pipettesandmicropipetteswithtips, Beakers, conical flasks, measuring cylinders, Water bath / Incubator, UV-Visible Spectrophotometer, Analytical balance, Distilled or deionized water

ChemicalsRequired: HydrogenPeroxide,PhosphateBuffer,AscorbicAcid,Ethanol, Folin– Ciocalteureagent, Sodiumcarbonate(Na_2CO_3),Ferricchloride,GallicAcid,Trichloro Acetic acid, Potassium Ferri cyanide.

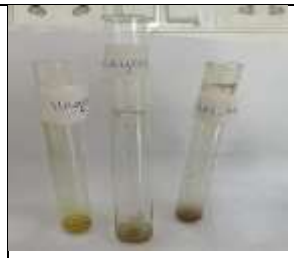
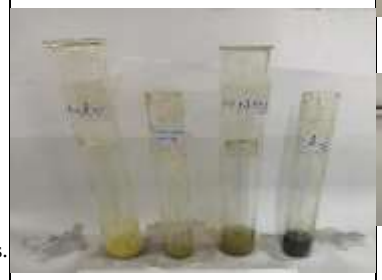
EXTRACTIONPROCESS

- **Weighedthepowderedplantmaterial**andplaceditintoacleancontainer.
- **Added a suitable solvent** (e.g., ethanol) to completely immerse the plantmaterial.

- **Sealedthecontainer**topreventsolventevaporation.
- **Allowed the mixture to stand**at room temperature for24 to72 hours (or longer), occasionally stirring or shaking to enhance extraction.
- **Maceration Process**: Transferred the weighed powder to a clean **stoppered or closed glass container.** Added the solvent, making sure the powder is fully submerged. Allowed to stand for **7 -14 days** at **room temperature**, away from light. **Shake or stir** occasionally (2–3 times per day) to improve contact between solvent and plant material.
- **Filtered the mixture** through filter paper or muslin cloth to separate the liquid extract from the plant residue.
- **Collectedthefiltrate**forfurtheruseor concentration.
- **Concentration ofExtract:** Concentratedthe filtrateunder reduced pressure using a rotary evaporator or by gentle heating to remove the solvent and obtain a crude extract. For aqueous extracts, freeze-drying or spray drying may be used. After filtration, the collected liquid extract is subjected to **concentration** to remove the solvent and obtain the crude extract. This is typically done using a **rotary evaporator**under **reduced pressure** and **controlled temperature** (usually below 40°C) to prevent degradation of heat-sensitive compounds. In the case of water- based extracts, **freeze-drying (lyophilization)** may be used instead to preserve compound integrity. The concentration continues until most of the solvent is evaporated,leavingbehindasemi-solidorsolidcrudeextractreadyforfurtheruse or analysis.
- **Storage of Extract:** Stored the concentrated extract in airtight containers, preferably in a cool, dark place to prevent degradation.

PRELIMINARYPHYTOCHEMICALSCREENING

TESTS	FAGONIACRETICA	HUPERZIASERRATA
TESTFORCARBOHYDRATES **Molish's test:**To 2-3ml of extract add few drops of alpha-naphtol solutioninalcohol shake and conc. H2SO4from the side of test tubes. Violet ring is formed at the junction of two liquids. **Benedict's Test:**Mix equal parts of sample and Benedict's reagent,heat for2–5minutes,andobservecolor change.Colorchangesfrom blueto green, yellow, orange, or brick-red. **Fehling'sTest:**Mixequalvolumes ofFehling's A and Fehling's B. Add themixturetothesampleandheat. Red ppt indicate reducing sugar.	  	  
TESTFORPROTEINS: **Biuret test:** To 3ml of test solution added 4% NaOH and few drops of 1%CuSO4solution.Aviolettopink colourindicatespresenceofproteins.		
TESTFORALKALOIDS: **Mayer'stest:**2-3mlfiltratewithfew drops of Mayer's reagent gives ppt.		

Wagner's test: Added Wagner's reagent (iodine in potassium mercuric iodide) to the sample. A brown/red ppt indicates the presence of alkaloids**.** **Hager's Test:**Added Hager's reagent (picric acid solution) to the test sample.A yellow ppt indicates the presence of alkaloids.		
TESTFORFLAVONOIDS: **Shinoda test:**To dry powder added 5 ml of 95% ethanol, few drops of conc. HCLand 0.5gmofmagnesium turnings. Result: A pink, red, or orange color appeared, indicating the presence of flavonoids.		
TEST FOR TANNINS AND PHENOLIC COMPOUNDS **LeadAcetateTest:**Addedlead acetate, black ppt indicated phenolics. **FerricChlorideTest:**Added$FeCl_3$, blue/green/purple color indicated phenolics. **Nitric acid Test:**Added concentrated HNO_3 to the sample. A yellow ororange color indicatedthe presence of phenolic compounds. **Bromine Water Test:**Added bromine water, decolorization/white ppt indicates presence of phenolics.		   

METHODS

1. **Hydrogenperoxidescavengingactivity**

Hydrogenperoxidesolution(2 mM) wasprepared in50 mMphosphatebuffer (pH7.4). Aliquots (0.1 ml) ofdifferent fractions was transferred intothetesttubes and their volumes were made up to 0.4 ml with 50 mM phosphate buffer (pH 7.4) After addition of 0.6 ml hydrogen peroxide solution, tubes were vortexed and absorbance of the hydrogen peroxide at 230 nm was determined after 10 min, against a blank [23].

2. **Estimationoftotalphenolic content**

The total phenolic content was determined by the spectrophotometric method [27]. In brief, a 1 ml of sample (1 mg/ml) was mixed with 1 ml of Folin-Ciocalteu's phenol reagent. After 5 min, 10 ml of a 7% Na2CO3 solution was added to the mixture followed by the addition of 13 ml of deionized distilled water and mixed thoroughly. The mixture was kept in the dark for 90 min at 23°C, after which the absorbance was read at 750 nm. The TPC was determined from extrapolation of calibration curve which was made by preparing gallic acid solution. The estimation of the phenolic compounds was carried out in triplicate. The TPC was expressed as milligrams of gallic acid equivalents (GAE) per g of dried sample.

3. **Ferricreducingantioxidantcapacity**

The reducing powers ofthe extracts and the positive standard controls were determined by using thepotassium ferricyanide reduction method (Oyaizu, 1986). Extracts (0.5 mL) of the different concentrations (i.e., 10–1000 µg/mL^{-1}) that were made by reconstituting in respective solvents were mixed with 2.5 mL of (0.2 M) sodium phosphate buffer (pH 6.6) and 2.5 mL of potassium ferricyanide [K3Fe (CN)6] (1%) solution and vortexed. After incubation at 50 °C in oven for 20 min, 2.5 mL of Trichloroacetic acid (TCA) (10%, w/v) was added to all the tubes andcentrifuged at 3,000 x*g*for 10 min. Afterwards, upper layer of the solution (or supernatant) (5 mL) wastakenand mixed withdeionized water (5mL). To thissolution, 1mLofFeCl3(1%) was added in each test tube and incubated at 35 °C for 10 min. The formation ofPerls Prussian color was measured at 700 nm in a UV-Vis spectrophotometer (Shimadzu UV-2401PC). Increased absorbance of the reaction mixture indicates increased reducing power.

RESULTS

1. HYDROGENPEROXIDESCAVENGINGACTIVITY

The hydrogen peroxide (H_2O_2) scavenging activity of the extracts was evaluated by measuring the decrease in absorbance at 230nm in the presence of the sample, compared to the control (H_2O_2 solution without extract). The percentage inhibition was calculated relative to thecontrol, and results were expressed as ascorbic acid equivalents (mg AAE/g of dry weight) using a standard calibration curve prepared with ascorbic acid. [Control value is:**0.50]**

Formulatocalculate**H_2O_2scavengingactivity is:**

H_2O_2Scavenging activity(%)=$(Acontrol - Asample)$

$Asample$

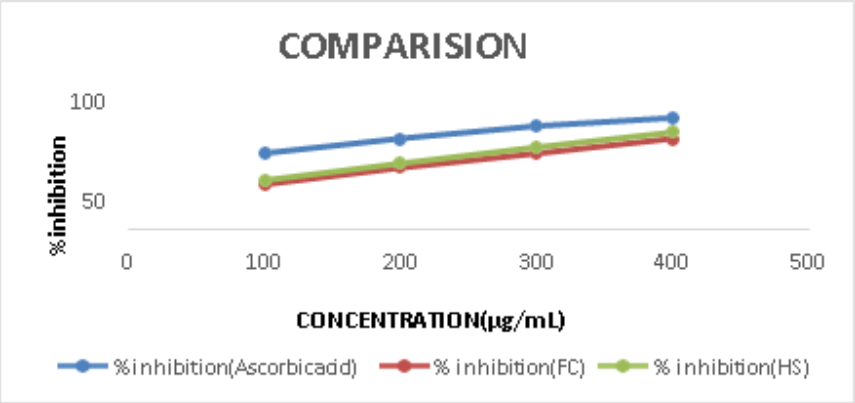
Where,

A_0=Absorbanceofcontrol(noantioxidantadded—maximumreactionorradical concentration)

A_1 = Absorbance of sample (with antioxidant — shows how much the radical concentration has decreased)

TableNo.02HYDROGENPEROXIDESCAVENGINGACTIVITY

Concentration (µg/mL)	Absorbance of ascorbic acid	%inhibiting activity of ascorbic acid	Absorbance of Fagonia Cretica	%inhibiting activity of Fagonia cretica	Absorbance ofHuperzia Serrata	%inhibiting activity of Huperzia serrata
100	0.30	40	0.42	16	0.44	12
200	0.24	52	0.36	28	0.38	24
300	0.18	64	0.30	40	0.32	36
400	0.12	76	0.24	52	0.26	48



GraphNo.01Comparision of%inhibitingactivityofextractswithstandard

2. FERRICREDUCINGANTIOXIDANTPOWERASSAY[FRAP]

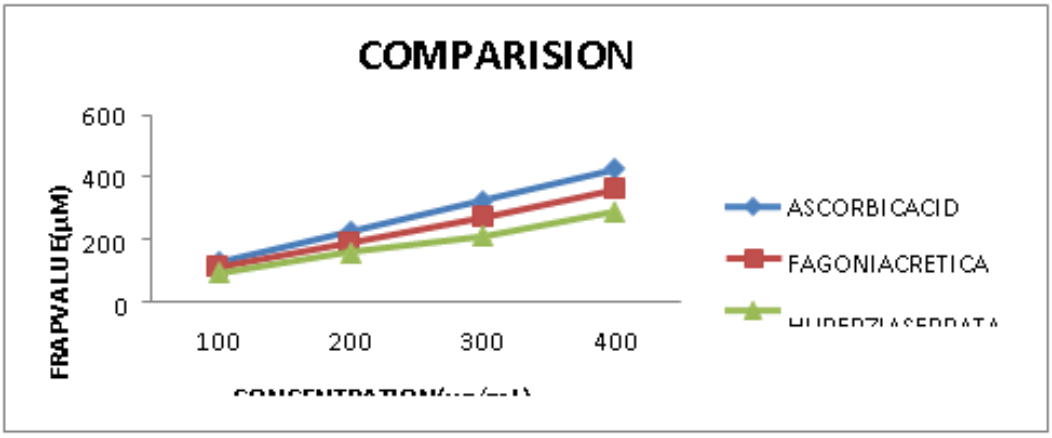
The antioxidant potential of the extracts was evaluated using the Ferric Reducing Antioxidant Power (FRAP) assay. Absorbance was measured at 593nm, and the results were expressed as ascorbic acid equivalents (mg AAE/g ofdryweight) based onthe standard curve prepared using ascorbic acid.

Formulatocalculate **FRAPvalueis:**

FRAPValue(µM) = $\frac{Absorbance}{Slope\ of\ standard cure}$

TableNo.02**FERRICREDUCINGANTIOXIDANTPOWERASSAY**

Concentration (µg/mL)	**Absorbance of ascorbic acid**	**FRAP value**	**Absorbance of Fagonia Cretica**	**FRAPvalue**	**Absorbance ofHuperzia Serrata**	**FRAPvalue**
50	0.25	59.5	0.20	47.6	0.18	42.8
100	0.45	107.1	0.35	83.3	0.30	71.4
150	0.68	161.9	0.55	130.9	0.48	114.2
200	0.88	209.5	0.72	171.4	0.62	147.6



GraphNo.02 Comparisionof FRAPValuesofextractswithstandard

3. TOTALPHENOLICCONTENT[TPC]

The total phenolic content was determined using the spectroscopic method and the absorbance wasrecordedat760nm usingascorbicacidasstandard.TheTPCwasexpressedasAscorbicacid equivalent (mg AAE) per gramofthedried samples.

FormulatocalculateTPCis:

TotalPhenolicContent(mgAAE/g) $= \frac{(A sample - A blank) \times DF}{Slope}$

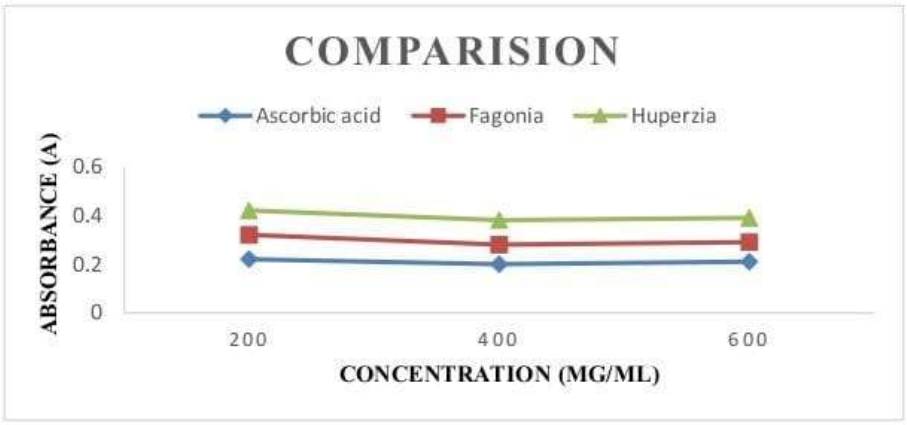
Where,

Asample=Absorbanceofthetestsample Ablank =Absorbance of the blank

Slope =Fromascorbicacidstandardcurve(μM^{-1}) DF =Dilution factor

TableNo.04**TOTALPHENOLICCONTENT**

Concentration (µg/mL)	**Absorbance of ascorbic acid**	**TPC of Ascorbicacid**	**Absorbance of Fagonia Cretica**	**TPCoffagonia cretica**	**Absorbance ofHuperzia Serrata**	**TPC of huperzia serrata**
200	0.22	4762.2	0.20	4762.0	0.21	4762.1
400	0.32	7142.9	0.28	6190.5	0.29	6428.6
600	0.42	9523.8	0.38	9047.6	0.38	7619.0



Graph No. 03 Comparision of TPC Values of extracts with standard

CONCLUSION:

The present study aimed to compare the in vitro antioxidant activities of *Fagonia cretica* and *Huperziaserrata*usingwell-establishedassays, includingH2O2radicalscavenging,FRAP(ferric reducing antioxidant power), and hydrogen peroxide scavenging methods. Additionally, total phenolic content (TPC) was estimated to evaluate the contribution ofphenolic compounds to the antioxidant capacity of the plant extracts.

Both *Fagonia cretica* and *Huperzia serrata* extracts demonstrated considerable antioxidant activity in a concentration-dependent manner across all assays. However, *Fagonia cretica* consistently showed higher efficacy, with greater higher FRAP values, and better hydrogen peroxide neutralization, indicating a stronger antioxidant potential.

The estimationofTotalPhenolic Content revealed that *Fagonia cretica* contained a significantly higher amount of phenolic compounds compared to *Huperzia serrata*. Since phenolics areknown to contribute directly to antioxidant activity by donating electrons or hydrogen atoms to free radicals, this higher TPC strongly correlates with the superior antioxidant performance observed in *Fagonia cretica*. *Huperzia serrata*, although slightly lower in phenolic content and antioxidant capacity, still exhibited noteworthy activity. Its bioactive components such as saponins, glycosides, and flavonoids contributeto its effectiveness and support its traditionaluse in managing liver disorders, inflammation, and tumors.

Inconclusion, both*Fagonia cretica* and *Huperzia serrata* possesssignificant invitroantioxidant activity, with *Fagonia cretica* demonstrating superior performance. The higher total phenolic content in *Fagonia cretica* likelyplays a major role in its strong antioxidant capacity. This study supports their continued exploration as natural antioxidant sources and contributes to thegrowing evidence favoring plant-based solutions to combat oxidative stress and related diseases.

REFERENCES

1. AbuajahCI, Ogbonna AC, OsujiCM. Functionalcomponentsand medicinalpropertiesof food: a review. J FoodSciTechnol. 2015;52:2522–2529. doi: 10.1007/s13197-014-1396- 5.[DOI][PMCfreearticle][PubMed][GoogleScholar][Reflist]
2. Vaibhav DA, Arunkumar W, Abhijit MP, Arvind S. Antioxidants as an immunomodulator. İnt J Curr Pharm Res. 2011;1:8–10. [Google Scholar][Ref list]
3. Cheeseman KH, Slater TF. An introduction to free radicals chemistry. Br Med Bull. 1993;49:481–93. doi: 10.1093/oxfordjournals.bmb.a072625. [DOI] [PubMed] [Google Scholar][Ref list]
4. YoungIS, WoodsideJV.Antioxidantsinhealthanddisease.J ClinPathol.2001;54:176– 86. doi: 10.1136/jcp.54.3.176. [DOI] [PMC free article] [PubMed] [GoogleScholar][Ref list]
5. Bagchi K, Puri S. Free radicals and antioxidants in health and disease. EastMediterranean Health Jr. 1998;4:350–60. [Google Scholar][Ref list]
6. Bagchi K, Puri S. Free radicals and antioxidants in health and disease. EastMediterranean Health Jr. 1998;4:350–60. [Google Scholar][Ref list]
7. Liu T, Stern A, Roberts LJ. The isoprostanes: Novel prostanglandin-like products of the free radical catalyzed peroxidation of arachidonic acid. J Biomed Sci. 1999;6:226–35. doi: 10.1007/BF02253564. [DOI] [PubMed] [Google Scholar][Ref list]

8. EbadiM. Antioxidantsand freeradicalsin healthanddisease:Anintroductiontoreactive oxygen species, oxidative injury, neuronal cell death and therapy in neurodegenerative diseases. Arizona: Prominent Press; 2001. [Google Scholar][Ref list]

9. HarmanD.Roleoffreeradicalsinaginganddisease.AnnNYAcadSci.1992;673:126– 41. doi: 10.1111/j.1749-6632.1992.tb27444.x.[DOI] [PubMed] [Google Scholar][Reflist]

10. Rock CL, Jacob RA, Bowen PE. Update o biological characteristics of the antioxidant micronutrients- Vitamin C, Vitamin E and the carotenoids. J Am Diet Assoc. 1996;96:693–702.doi:10.1016/S0002-8223(96)00190-3.[DOI][PubMed][Google Scholar]

11. McCordJM.Theevolutionoffreeradicalsandoxidativestress.AmJMed. 2000;108:652–9.doi: 10.1016/s0002-9343(00)00412-5.[DOI] [PubMed] [Google Scholar]

12. RaoAL,BharaniM,PallaviV.Roleofantioxidantsandfreeradicalsinhealthand disease. Adv Pharmacol Toxicol. 2006;7:29–38. [Google Scholar]

13. Halliwell B. How to characterize an antioxidant- An update. Biochem Soc Symp. 1995;61:73–101. doi: 10.1042/bss0610073. [DOI] [PubMed] [Google Scholar]

14. ShiHL, NoguchiN, NikiN. Comparative studyondynamics ofantioxidative actionofα- tocopheryl hydroquinone, ubiquinol and α- tocopherol, against lipid peroxidation. Free RadicBiolMed.1999;27:334–46.doi:10.1016/s0891-5849(99)00053-2.[DOI] [PubMed][GoogleScholar]

15. Levine M, Ramsey SC, Daruwara R. Criteria and recommendation for Vitamin C intake. JAMA.1991;281:1415–23.doi:10.1001/jama.281.15.1415.[DOI][PubMed] [GoogleScholar]

16. Frie B, Stocker R, Ames BN. Antioxidant defences and lipid peroxidation in humanbloodplasma.ProcNatlAcadSci.1988;37:569–71.doi:10.1073/pnas.85.24.9748.[DOI] [PMC free article] [PubMed] [Google Scholar]

17. BansalS.,ChoudharyS.,SharmaM.,KumarS.S.,Lohan S.,BhardwajV.,Syan N.,Jyoti S.Tea:Anativesourceof antimicrobialagents.FoodRes.Int.2013;53:568–584.doi: 10.1016/j.foodres.2013.01.032. [DOI] [Google Scholar]

18. Jiang J., Xiong Y.L. Natural antioxidants as food and feed additives to promote health benefits and quality of meat products: A review. Meat Sci. 2016;120:107–117. doi: 10.1016/j.meatsci.2016.04.005. [DOI] [PubMed] [Google Scholar]

19. Lü J.-M., Lin P.H., Yao Q., Chen C. Chemical and molecular mechanisms of antioxidants: Experimental approaches and model systems. J. Cell. Mol. Med. 2009;14:840–860.doi:10.1111/j.1582-4934.2009.00897.x.[DOI][PMCfreearticle] [PubMed][GoogleScholar]

20. Rice-EvansCA,Diplock AT.Current statusofantioxidant therapy.FreeRadicBiolMed. 1993;15:77–96. doi: 10.1016/0891-5849(93)90127-g. [DOI] [PubMed] [Google Scholar]

21. Krinsky NI. Mechanism of action of biological antioxidants. Proc Soc Exp Biol Med. 1992;200:248–54. doi: 10.3181/00379727-200-43429. [DOI] [PubMed] [Google Scholar