Pravara Med Rev; March 2025, 17 (01), 59 - 67
DOI: 10.36848/PMR/2025/19800.40196

Original article

Study of Erastin Induced ferroptosis by Medicinal Plants

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Abstract

Ferroptosis is a recently discovered programmed cell death triggered by accumulation of iron-induced toxic lipid peroxides. Ferroptosis was first recognized in year 2012 during screening of anticancer drugs that selectively killed RAS-mutated cancer cells. Erastin is a chemical inhibitor of system Xc^- that depletes cellular glutathione (GSH) and facilitates iron induced lipid peroxidation leading cell death. Plants are richest source of medicinal drugs and currently majority of FDA-approved drugs are derived from plant source. This prompted us to screen medicinal plants for discovery of novel ferroptosis inhibitor. In the present study, we collected 15 medicinal plants and processed their leaves for preparation of aqueous extracts or soxhlet solvent extracts. In total, we had 34 extract powders that included 20 aqueous extracts and 14 solvent extracts for screening studies. Our primary screening resulted in identification of 12 lead candidates, with 5 showing significant inhibition in secondary screening, which showed significant inhibition of erastin-induced ferroptosis. Ocimum tenuiflorum holy basil showed the most significant inhibition at a low concentration. Further studies are undergoing to identify the bioactive compound in the medicinal plants leave extracts.

Key Words: *Ferroptosis, Erastin, Medicinal plants, MTT assay, Ferroptosis Inhibitors.*

Introduction

Tight regulation of cell death is vital during development, normal homeostasis as well as pathogenesis of diseases. Several acute and chronic Diseases are associated with excessive cell death resulting tissue damage and organ failure.[1] Caspase dependent apoptosis is well studied programmed cell death which is implicated in normal and disease pathogenesis. [2] Ferroptosis is representing a pathogenic programmed cell death characterized by iron mediated production of toxic lipid hydroperoxides. [3] Ferroptosis is not inhibited by caspase inhibitors and therefore represent a unique form cells death which is widely implicated in several acute and chronic diseases such as asthma, COPD, Ischemic heart diseases, brain trauma, neurological diseases, and renal failure.[4]

Since Ferroptosis involves accumulation of toxic lipid peroxides, enzymes involved in generation and or detoxification of lipid peroxides modulate ferroptosis.[5] Oxidative stress and activation of lipoxygenase (oxygenate polyunsaturated fatty acid (PUFA)-phospholipids) are key inducers of Ferroptosis.[6] On the other hand Glutathione and Glutathione peroxidase 4 plays critical role in detoxification of phospholipid hydroperoxides and protect from

ferroptosis.[7] Pharmacological depletion of GSH or inhibition of GPX4 activity is shown to induce ferroptosis.[8] In contrast, agents that increase GSH or GPX4 activity are shown to inhibit ferroptosis. Because ferroptosis is implicated in several acute and chronic diseases, there is a urgent need for developing pharmacological inhibitors of ferroptosis for treatment of diseases.[9]

Medicinal plants are commonly used for the treatment of various diseases, as they are considered to have advantages over the conventionally used drugs that are much expensive and known to have harmful side effects.[10] Oxidative stress and inflammation have been mainly implicated in many of the disease pathology.[11] Therefore, considerable attention has been focused among phyto-therapy researchers in identifying dietary and medicinal phytochemicals with antioxidant and anti-inflammatory activity that can inhibit, retard or reverse the multi-stage pathophysiological events underlying the disease pathology.

Recently non-apoptotic cell death pathways have gained special attention particularly due to their role in inflammation and as potential therapeutic target in cancer.[12] Ferroptosis, a newly discovered regulated form of cell death leading to glutathione depletion resulting from inhibition of cysteine uptake or inactivation of the lipid repair enzyme glutathione peroxidase (GPX4) causes iron-dependent accumulation of ROS and lipid-based ROS, particularly lipid hydroperoxides.[13] A selenoprotein, is the only reduced glutathione peroxidases which handles lipid hydroperoxides and uses GSH to convert potentially toxic lipid hydroperoxides to non-toxic lipid alcohols.[14] GSH, a tripeptide of glutamate cysteine and glycine, is a well-known intracellular antioxidant protecting cells from oxidative or other forms of stress and serves as a cofactor for glutathione peroxidase and S-trasnferase enzyme families.[15]

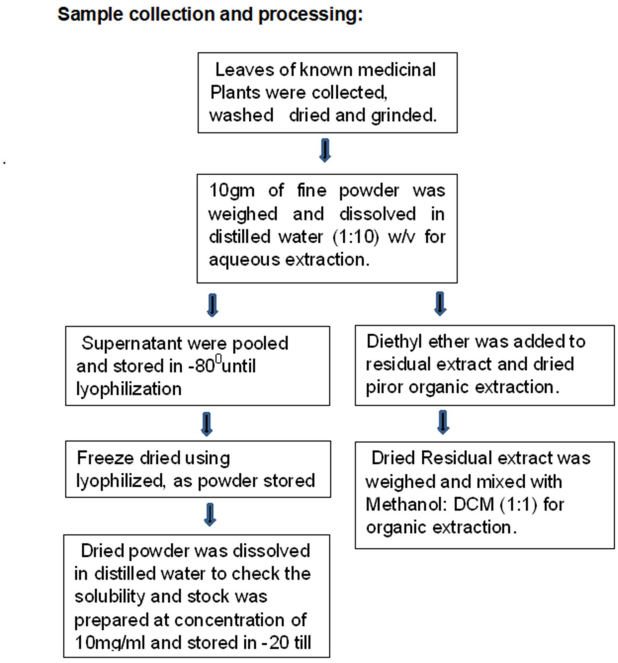
Ferroptosis induced cell death can be suppressed exclusively by iron chelators, lipophilic antioxidants and inhibitors of lipid peroxidation. Several studies identified lipophilic antioxidants (α-tocopherol, Butylated hydroxytoluene, and β-carotene) as strong suppressors of erastin-induced cell death.[16] Vitamin E family and flavonoids can inhibit lipoxygenases activity in some contexts.[17] Inhibitor of iron metabolism and iron chelators (deferoxamine and ciclopirox) suppresses ferroptosis by reducing availability of iron.[18] Similarly, ferrostatins and liproxstatins inhibit lipid peroxidation, possibly by acting as radical trapping antioxidants.[19] similarly to the lipophilic antioxidants BHT, BHA, and vitamin E. In this study, organic and aqueous extracts from 20 different medicinal plants were used to screen and identify the inhibitors of ferroptosis induced by erastin in human lung cell line.

Methodology

Sample collection and processing:

Leaves of medicinal plants *Piper betel(BT), Tridax procumbens(TD), Ziziplus xylopyrus, Cardiospermum halicacabum, Ixora coccinea(IC), Antigonon leptopus, Mirabilis jalapa, Thespesia populnea, Gymnema sylvestre, Clerodendrum serratum (CS), Michelia champaca, Plectranthus amboinicus* (Mexican mint)**,** *Asparagaceae racemosus* (Satavari)*, Ocimum tenciflorum* (Tulsi)*, Pongamia pinnata, Rawolfia surpentina* (Sarpagandhi)*,* Mimosa pudica*, Lantana camara, Callistemon viminalis(Bottle brush), Swertia chirata, Caesalpinia sappan(* Pathanga), Andrographis paniculata (Green chirata) were gathered according to their potential medical applications. It has been recognized as a herbarium voucher at JSS University, Mysuru (Voucher Number: 145/2018), After three rounds of washing with tap water, the collected leaves were left to dry in the shade. After 35–45 seconds of grinding dried leaves at low, medium, and high speeds, the consistency of the crushed powder was examined. A sieve (42 microns) is used to divide the ground powder into fine and coarse powder. Falcon tubes are used to contain fine powder, which is then wrapped in aluminum foil. As per study flowchart (Fig.1) organic (Soxhlate extraction) and aqueous extracts from 20 plants were collected.

Figure 1: Extraction procedures



Cell culture: We used normal human bronchial epithelial cells (Beas2B) for the entire cell based assay. Beas2B cells were cultured in DMEM:HAMF12(Dulbecco modified eagles media) and supplemented with 10% v/v foetal bovine serum (FBS), 1% w/v penicillin and 1% w/v L-glutamine (200 mM). All these reagents were obtained from Sigma–Aldrich.

Assessment of Cytotoxicity by MTT assay:

To screen and identify standardized extracts for inhibiting erastin-induced ferroptosis in normal Beas2b cell line and to know toxic doses of plant extract we performed MTT assay.

MTT assay is a colorimetric assay that measures the reduction of yellow 3-(4, 5-dimethythiazol- 2-yl)-2, 5-diphenyl tetrazolium bromide (MTT) by mitochondrial succinate dehydrogenase NADPH dependent.The dye enters the cells and passes into the mitochondria where it is reduced to an insoluble, coloured (dark purple) formazan product. The formazan product is solubilised with 100% DMSO 10% SDS and 0.1N Hcl and absorbance was measured at 570nm. Only live cells will reduce MTT and form formazan product.

Bioassay for discovering ferroptosis inhibitors:

For screening for ferroptosis inhibitors, we used erastin-induced ferroptosis in vitro model system. Normal human lung epithelial cells (Beas2B) cells were treated with increasing concentrations (2.5, 5.0 and 10 µM) of erastin. After 24h, the cell death was assessed by MTT assay. The data was expressed percentage cell death as compared to vehicle control. The cells were treated with test agent 24h prior to erastin treatment to identify identified ferroptosis inhibitor.

Screening approach: We divided our screening approach into Phase I primary screening and Phase II secondary screening.

Phase I primary screening studies:

Cytotoxic profile of test agents: Beas2B cells were treated with 25 or 100 µg/mL concentrations of 31 test agents for 24h and subsequently, cell death was assessed by MTT assay.

Bioassay: Beas2B cells were first treated with 25 or 100 µg/mL concentrations of 31 test agents for 24h and subsequently, the cells were incubated for additional 24h with erastin (5 or 10µM). At the end of erastin treatment, the cell death was assessed by MTT assay. Test agents that inhibited erastin-induced cell death by at least 25% were selected as lead candidates for phase II screening studies.

Phase II secondary screening studies: To validated the bioactivity of the identified lead candidates from phase I studies, Beas2B cells were pre-treated with lead candidates at different concentrations for 24 h and subsequently treated with erastin for additional 24h. The test agent that showed good dose- response was selected as best lead candidate for further bioactivity characterization studies.

Results

Test Agents: In total, we had 34 test agents in dried-powder form, which included 15 aqueous extract powder and 14 organic extract powder (Figure 2A & 1B). The aqueous extracts were solubilized in distilled water whereas organic solvent extract powders were solubilized in DMSO. The stocks solutions were prepared as 10mg/mL and stored at -80^{o}C.

Cytotoxicity of test agents: To determine non-lethal dose of test agent for screening studies, Beas2B cell were exposed to test agents at a concentration of 20 and 100µg/mL and cell death was measured by MTT assay. Figure 1A & 1B shows the cytotoxicity profile of aqueous extracts-test agent (superscript AQ refers to aqueous extraction) and solvent extract-test agents (superscript SX refers to solvent extraction). The concentration of test agents that showed less than 80% viability of cells was considered as lethal dose. None of the aqueous extracts-test agents showed toxicity at indicated concentrations. Except LC^{AQ}, BB^{AQ} & CS^{AQ}, solvent extracts-test agents also showed no toxicity at the indicated concentrations. Test agents: LC^{AQ}, BB^{AQ}, CS^{AQ} were cytotoxic at 100µg/ml.

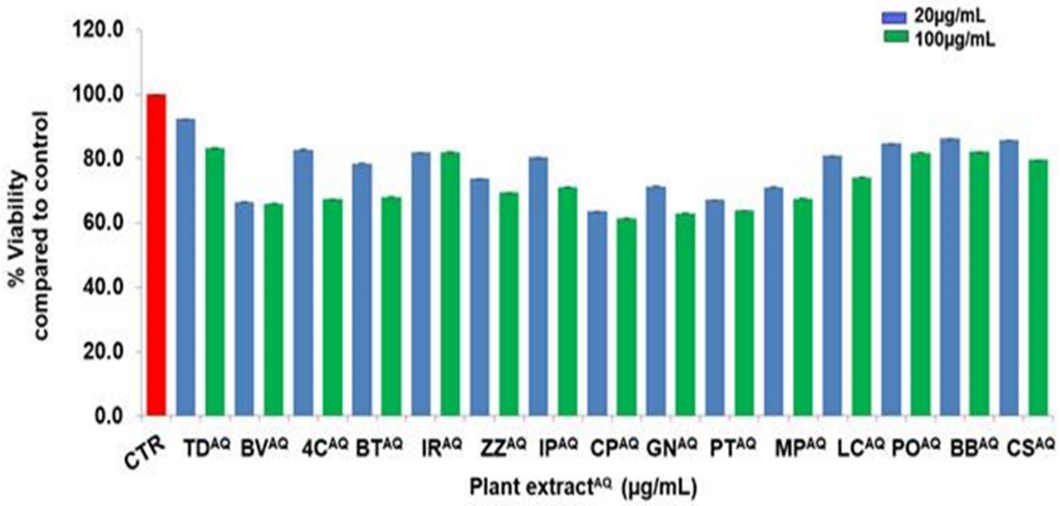


Figure 2A: Cytotoxicity profile of aqueous extract-test agents.

Beas2B cell were treated with test agents at indicated concentrations for 24h and cell death was assessed by MTT assay (superscript ^{AQ}, refers to aqueous extraction)

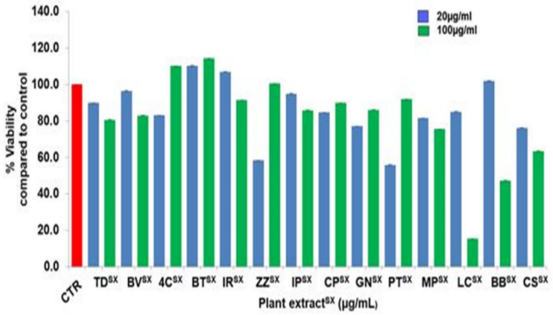
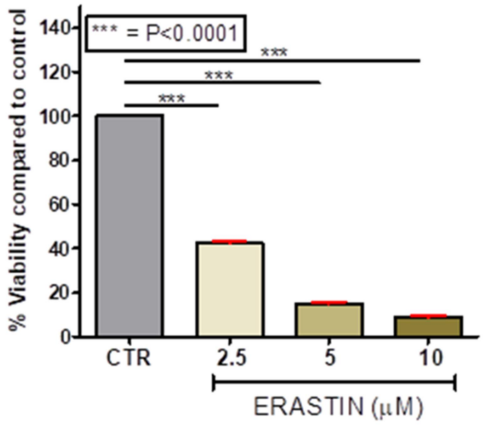


Figure 2B: Cytotoxicity of profile of solvent extract-test agents. *Beas2B cell were treated with test agents at indicated concentrations for 24h and cell death was assessed by MTT assay (superscript ^{SX} refers to solvent extraction).*

Erastin-induced ferroptosis in normal bronchial epithelial cells: Erastin is a potent inducer of ferroptosis and has been widely used for discovering ferroptosis inhibitors. To select for optimal dose of erastin for screening studies, we treated Beas2B cells with increasing doses of erastin. Erastin-induced concentration dependent increase in cell death and the cell death was over 80% at concentrations 2.5, 5.0 and 10 µM as compared to vehicle control (Figure 2). Ferrostatin 1 is a potent inhibitor of erastin-induced ferroptosis.



Figures 3: Erastin-induced ferroptosis in Beas2B cells. *Cells were treated with increasing concentration of Erastin (2.5-10µm) and cell death was assessed by MTT assay*

Primary and secondary screening of test agents against Erastin-induced ferroptosis: In our primary screening, we identified 12 test agents as lead candidates out of total 34 test agents, which showed significant inhibition of erastin induced cell death (Figure 3A-B). Of the 12 lead candidates, 8 were aqueous extract-test agent and 4 were solvent extract-test agent. In the phase II secondary screening, we reevaluated the 12 lead candidates and the data was reproducible (Figure 4).

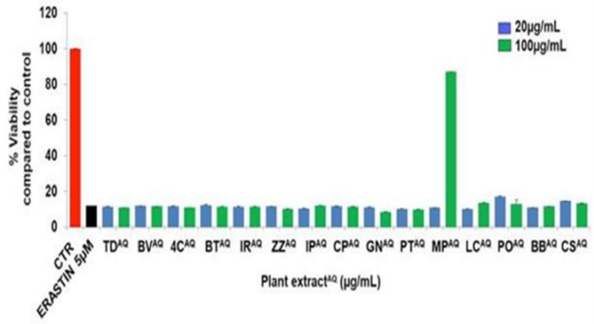


Figure- 4: Evaluation of aqueous extract-Test (A) and solvent extract-test agents (B) against Erastin-induced ferroptosis. Beas2B cells were treated with either aqueous extract-test agents or solvent extract-test agents for 24 h followed by erastin treatment for additional 24h. Cell death was assessed by MTT assay.

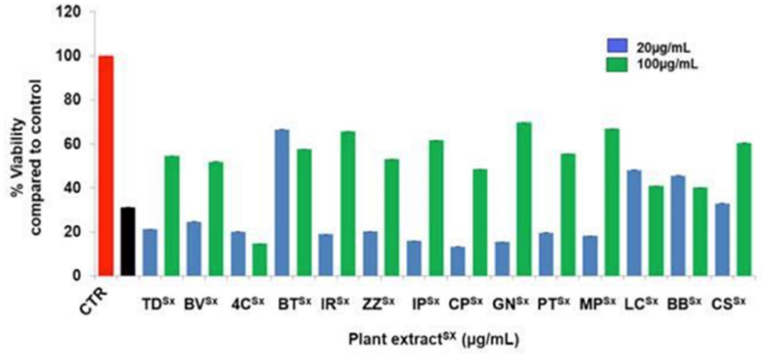


Figure-5: Secondary screening of 12 lead candidates. *The 12 lead candidates selected from phase I primary screening studies were reevaluated. Beas2B cells were treated with 12 lead candidates for 24h following by treatment with erastin for additional 24h. Cell death was assessed by MTT assay.*

These 12 test agents were Tridax procumbent (TD^{AQ}), (*Piper bete*) Beetle (BT^{SX}), Ixora coccinea (IC^{AQ}), (*Caesalpinia sappan)* Pathanga (PT^{SX}), Mimosa pudica (MP^{AQ}), (*Callistemon viminalis*) Bottle brush (BB^{SX}), Clerodendrum serratum (CS^{SX}), (*Asparagaceae racemosus)* Satavari (ST^{AQ}), (*Ocimum tenciflorum*) Tulsi (TU^{AQ}), Andrographis paniculata Green chirata (GC^{AQ}), (*Plectranthus amboinicus)* Mexican mint (MM^{AQ}), (*Rawolfia surpentina*) Sarpagandhi (SG^{AQ}). Among the 12 lead candidates 5 shows more than 25% inhibition of ferroptosis, solvent extract & aqeous extract of tulsi (TU^{AQ}) concentration of 20µg/ml, Mimosa pudica (MP^{AQ}), both concentration of 20µg/ml, 100µg/ml, (*Caesalpinia sappan)* Pathanga (PT^{SX}) concentration of 20µg/ml, (*Piper betel*) Beetle (BT^{SX}) both concentration of 20µg/ml & 100µg/ml, and (*Callistemon viminalis*) Bottle brush (BB^{SX}) concentration of 20µg/ml was most effective in inhibiting erastin-induced ferroptosis.

Discussion:

Ferroptosis, a type of programmed cell death, has garnered attention as a promising target for therapeutic intervention in various diseases, including cancer, neurodegeneration, and ischemia-reperfusion injury.[20] This specific pathway of cell death is characterized by the accumulation of lipid peroxides, which is initiated by the reduction or inactivation of glutathione peroxidase 4, a crucial regulator of cellular redox balance. According to research by Wei C et al 2024[21], ferroptosis has been recognized as a significant area of investigation. A well-known inducer of ferroptosis is the small molecule erastin, which functions by inhibiting the cystine/glutamate antiporter system xc-.[22] This leads to the depletion of glutathione and subsequent initiation of ferroptosis.

Experimental pharmacological inhibitors of ferroptosis are shown to protect from intracerebral hemorrhage driven stroke, renal injury, and ischemic-reperfusion injury and these exciting finding have underscored the importance of discovering new and safe ferroptotic inhibitors for therapeutics.[23] (Wang Y et al 2023). Numerous studies have demonstrated the antioxidant capabilities of medicinal plants, attributed largely to its high phenolic and flavonoid content. Günther M (2024) noted that the presence of flavonoid–phenolic acid hybrids compounds enables medicinal plants to scavenge free radicals and reduce oxidative stress, factors critical to ferroptosis.[24]

The data from the study on ferroptosis inhibitors revealed a significant impact of both solvent extract and aqueous extract on reducing ferroptosis in the Beas2B cell line. Tulsi (Ocimum tenuiflorum) (TU^{AQ}) and Pathanga (Caesalpinia sappan) (PT^{SX}) showed noteworthy inhibition at low concentrations, while Beetle (Piper betel) (BT^{SX}), Bottle brush (BB^{SX}), and Sarpagandhi (Rawolfia serpentina) (SG^{AQ}) exhibited moderate inhibition at both low (20µg/ml) and high (100µg/ml) concentrations. Of particular significance is the consistent display of potent ferroptosis inhibition by the aqueous extract of tulsi compared to the solvent extracts of the other four plant extracts. In a related study, Kose T et al. (2025) reported on the anti-ferroptosis activity and Fe-chelating properties of curcumin at specific doses in human pancreatic ductal adenocarcinoma cells.[25] Additionally, Zhang Y et al. (2024) documented curcumin's ability to inhibit chondrocyte ferroptosis by activating the Nrf2 signaling pathway.[26] Furthermore, Wenhan H et al. (2023) reported on resveratrol's protective effects on BEAS-2B cells from erastin-induced ferroptosis.[27]

A previous study revealed that plants are abundant in flavonoids and polyphenols, which have been found to aid in inhibiting lipid peroxidation and reducing levels of glutathione and also in preventing ROS-mediated depletion, thus reducing ferroptosis. Ocimum tenuiflorum, commonly known as holy basil, is rich in flavonoids such as luteolin, apigenin, quercetin, and kaempferol, as well as phenolic acids like Caffeic acid, Rosmarinic acid, Syringic acid, Ferulic acid, and Catechin.[28] These flavonoids and phenolic acids play a significant role in the therapeutic properties of the plant, potentially contributing to the inhibition of erastin-induced ferroptosis. Another study on Caesalpinia sappan, also known as Pathanga (PTSX), indicated the presence of brazilin, sappanchalcone, and quercetin, which are responsible for anti-ferroptosis properties.[29]

Conclusion

A study aimed to identify phytochemicals for inhibiting ferroptosis, a process linked to degenerative diseases. After screening 30 leaf extracts, 12 lead candidates were identified, with 5 showing significant inhibition. *Ocimum tenuiflorum* holy basil showed the most significant inhibition at a low concentration. This finding warrants further investigation to identify the bioactive compound of medicinal plants.

Declaration of interest:

The authors report no conflicts of interest.

Acknowledgment:

This work was supported by the Dean of Research, Department of Biochemistry, Meenakshi Academy of Higher Education & Research.

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