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CODEN: IAASCA **REVIEW ARTICLE**

Overview On Anti-Ulcer Activity *Of Basella Alba :* A Therapeutic Herb

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ABSTRACT
In Ayurveda, groups of plant known as Rasayanas have been extensively used as rejuvenating for arresting the progression of aging, to provide resistance against disease as well as those induced by emotional perturbation and to promote general welfare of the individual. It is, thus, obvious that long before the concept of adaptogen was involved in the middle part of this century, an extremely similar theory has been propounded centuries ago in Ayurveda. Gastric H^+-K^+ ATPase of the parietal cell is the H^+ ion pump responsible for acid secretion in the stomach and has been identified as a pharmacological target for the development of a drug to treat ulcers. The histamine H_2-receptor antagonist cimetidine, ranitidine and famotidine act as potent inhibitors (70-80%) of secretion. Basella alba L. herb is used traditionally in Thailand for anti-inflammatory, cytotoxicity and antioxidant activities of anti-inflammatory remedies. This allowed the selection of lead extracts for various ethno pharmacological researches. The active constituents of Basella alba is Basellasaponin (A, B, C and D), Kaempherol, Betalain, which are therapeutically used for antifungal, anticonvulsant, analgesic, anti-inflammatory, androgenic activities and for the treatment of anemia. It also used for the hemorrhages, skin diseases, sexual weakness, ulcers and as laxative in children and pregnant women.
KEYWORDS: *Emotional perturbation, H_2-receptor antagonist, cytotoxicity, androgenic*

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INTRODUCTION

Gastric hyperacidity and ulcer are very common causing human suffering today. It is an imbalance between damaging factors within the lumen and protective mechanisms within the gastro duodenal mucosa. Although prolonged anxiety, emotional stress, hemorrhagic surgical shock, burns and trauma are known to cause severe gastric irritation, the mechanism is still very poorly understood (Rao *et al*., 2003).

Peptic ulcer represents a major health problem, both in terms of morbidity and mortality. The rise in gastric acidity and peptic activity are usually a manifestation of a physiological disturbance affecting one or more mechanisms which normally regulate gastric secretion. Neurotransmitters or hormones that directly stimulate secretion of hydrochloric acid and pepsin by the gastric glands are acetylcholine, gastrin and histamine. In addition there are other factors which play an important role in the manifestation of peptic ulcers. The activity of the gastric secretory cells has been found to be stimulated by caffeine, alcohol, hydro alcoholic acid, sodium chloride, nonsteroidal anti-inflammatory drugs (NSAIDs) and stress. There are two major components to the ulcerogenic effects of NSAIDs in the stomach, namely their topical irritant effects on the epithelium and their ability to suppress prostaglandin synthesis. The ability of NSAIDs to cause gastric damage correlates well with the ability to suppress gastric prostaglandin synthesis. There is also a time and dose dependency of both suppuration of gastric prostaglandin synthesis and ulcerogenic activity.

The usual medical treatment for peptic ulcer is either by the inhibition of acid secretion or by neutralization of the acid. The neutralization of gastric acid can be done by antacid administration but their effectiveness is only for a brief period. Muscarinic antagonists such as atropine or pirenzipine are effective inhibitors of acid production.

ANATOMY OF AN ULCER

An ulcer can be thought of as a lesion or sore that forms along the stomach or intestinal wall where it can corrode muscle and blood vessels and cause bleeding, evidenced blood in the stool. If this process is allowed to continue, bacteria and partially digested material can eventually leak into the abdominal cavity causing inflammation and severe pain. A perforated ulcer of this type usually requires immediate

surgery. Ulcers can also occur in the duodenum and can restrict or block the intestinal opening, a condition that also demands immediate attention.

Ulcers do not always produce symptoms. But, the most common symptom reported is burning pain between the breastbone and navel, usually occurring shortly after a meal or when the stomach is empty. An accurate diagnosis may involve an upper GI series or x-rays of the esophagus, stomach and duodenum after drinking a barium cocktail. Other measures include blood and stomach tissue tests to determine if H. Pylori is present.

CAUSES OF ULCER

Helicobacter pylori gastritis

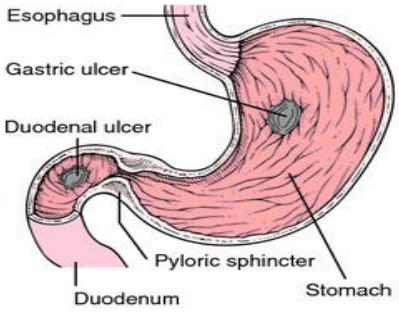


Fig.1. Peptic Ulcer disease

About 15-20% cases infected with H. Pylori never develop a duodenal ulcer in their lifetime while gastric colonization by H. Pylori never develops ulceration and remain asymptomatic. H. Pylori can be identified in mucosal samples by histologic examination, culture, increased activity, and serology (IgG and IgA antibodies to H. Pylori).

Acid-pepsin secretion

There is conclusive evidence that some level of acid-pepsin secretion is essential for the development of duodenal as well as gastric ulcer. Peptic ulcer never occurs in association with pernicious anemia in which there are no acid and pepsin-secreting parietal and chief cells respectively.

Mucus secretions

Any condition that decreases the quantity or quality of normal protective mucus 'barrier' predisposes to the development of ulcers.

Gastritis

Some degree of gastritis has been always present in the region of gastric ulcer, though it is not clear whether it is the cause or the effect of ulcer. Besides, the population distribution pattern of gastric ulcer is similar to that of chronic gastritis.

Local irritants

Pyloric antrum and lesser curvature of the stomach is the site most exposed for longer periods to local irritants and thus are the common sites of occurrence of gastric ulcers. Some of the local irritating substances implicated in the etiology of peptic ulcers are heavily spiced foods, alcohol, cigarette smoking, unbuffered aspirin, non-steroidal anti-inflammatory drugs etc.

Dietary factors

Nutritional deficiencies have been regarded as an etiologic factor in peptic ulcer e.g. occurrence of gastric ulcer in poor socioeconomic strata, higher incidence of duodenal ulcer in part of South India.

Psychological factors

Psychological stress, anxiety, fatigue and ulcer-type personality may exacerbate as well as predisposing to peptic ulcer disease.

Genetic factors

People with blood group 'O' appear to be more prone to develop peptic ulcers than those with other blood groups. Genetic influences appear to have greater role in duodenal ulcers as evidenced by their occurrence in families, monozygotic twins and association with HLA-B5 antigen.

Hormonal factors

Secretion of certain hormones by tumors is associated with peptic ulceration e.g. elaboration of gastrin by islet-cell tumor in Zollinger-Ellison syndrome, endocrine secretion in hyperplasia and adenomas of parathyroid glands, adrenal cortex and anterior pituitary.

Miscellaneous

Duodenal ulcers have been observed to occur in association with various other conditions such as alcoholic cirrhosis, chronic renal failure, hyperparathyroidism, chronic obstructive pulmonary disease, and chronic pancreatitis.

Endogenous mediators

Several endogenous mediators or substance has been identified and reported to be involved in the induction of gastrointestinal lesions. These have been found to be included lipid metabolites, neuropeptides, biogenic amines, reactive oxygen species and free radicals (Elasbach and Weiss., 1988).

Platelet-activating factor (PAF)

PAF one of the most potent ulcerogen (Rosam *et al*., 1986). The mechanism involved, in PAF includes ulceration is due to the sequestration of neutrophil aggregates in the stomach, vasoconstriction, generation of free radicals and release of lysosomal enzymes (Mcmanus *et al*., 1980; Wallace and Whittle, 1986).

Thromboxane A2 (TXA2) and Leukotriens (LTC4/D4)

They are derived from arachidonic acid through the action of the enzyme cyclooxygenase (TXA_2) and lipooxygenase (LTC_4/D_4). Vasoconstriction may be a causative factor in the TXA_2 mediated gastric mucosal ulceration which predisposes the mucosa to disruption by local irritants. Leukotriens induce vasoconstriction in the vascular bed in the rat sub mucosa that leads to tissue necrosis in the stomach (Wallace and Whittle, 1985). Leukotriens also stimulate pepsinogen secretion from gastric from gastric chief cell (Fiorucci *et al*., 1995). Involvement of Leukotriens in stress induced (Ogle and Chou, 1989) and ethanol induced (Wallace *et al*., 1989) ulcer by a decrease in blood flow and increase in reactive oxygen species (Peskar *et al*, 1991; Vaananen *et al*., 1992) has been reported.

Histamine

Histamine is present in large quantities, about 40 micrograms per wet weight, in the oxyntic mucosa of humans and other mammals (Reite, 1972, Trodl *et al*., 1975). Histamine has been found in the gastric wall and it is a powerful stimulant for gastric secretion (Black *et al*., 1972). However, excessive release of histamine by histamine release or by injection of an aqueous solution of histamine produces gastric and duodenal ulcer (Shayer, 1974). Histamine is involved in the other type of ulceration because blocking of histamine receptors prevents reserpine, steroid and NSAIDs induced gastric ulcer in human and experimental animals (Lau and Ogle, 1981). Histamine blockers such as Ranitidine have been reported to prevent psychological stress-induced gastric ulceration (Chou and Ogle, 1978).

Serotonin (5-hydroxytryptamine, 5HT)

Over 90% endogenous 5-hydroxytriptamine is found in the gastrointestinal tract. 5-HT is stored in endocrine cells and enteric neurons in the gut. It is established that 5-HT possesses ulcer producing properties. Serotonin has been shown to be involved in the ethanol and reserpine induces ulceration.

Free Radicals

Free radicals are defined as chemical species possessing unpaired electrons in their outer orbit which are generally very reactive. The most important reactant in free radical biochemistry in the aerobic cells is oxygen and its radical derivative O^{-}_{2} and OH, H_2O_2 and transition metals (Fe^{2+}, Cu^{+}) (Cheeseman and Slater, 1993).

The hydroxyl radicals are capable of reversibly or irreversibly damaging compounds of all biochemical classes, including nucleic acid, protein, and free amino acids, lipids, lipoprotein, carbohydrates and connective tissue macromolecule.

The role of oxygen derived free radicals has been demonstrated in acute and chronic ulceration (Del Saldato *et al*., 1985; Bast *et al*., 1991; Freeman and Crapo., 1982). Involvement of a neutrophil in ulcer has been implicated in different models of gastrointestinal mucosal injury such as colitis, ischemia (Perry *et al*., 1986), reperfusion (Grisham *et al*,. 1990) stress (Das *et al*., 1997) and ethanol (Mizui and Doteuchi., 1986) induced ischemia/reperfusion and ethanol induced injury to the gastric and intestinal mucosa are substantially ameliorated in neutropenic animals (Tepperman and Soper, 1994).

GASTRIC MUCOSAL DEFENSE MECHANISM

Mucous bicarbonate barrier

Peptic ulcers are not solely induced by the offensive factor of the acid and pepsin but the breakdown of mucosal resistance is also considered as an important factor making the stomach susceptible for ulcer (Goel and Bhattacharya, 1991). The entire surface of the gastric mucosa is covered by a continuous layer of mucus gel, which has a variable thickness of less than 500 μm (Allen *et al*., 1993). Both the surface mucus cells and the mucous neck cells in the upper part of the gland secrete mucus. Mucus is released predominantly by the process of exocytosis. Mucus consist of about 71% by weight salt and other daily

soluble components, 0.5-1% free protein and a similar quantum of carbohydrate rich glycoprotein and 95% or more water.

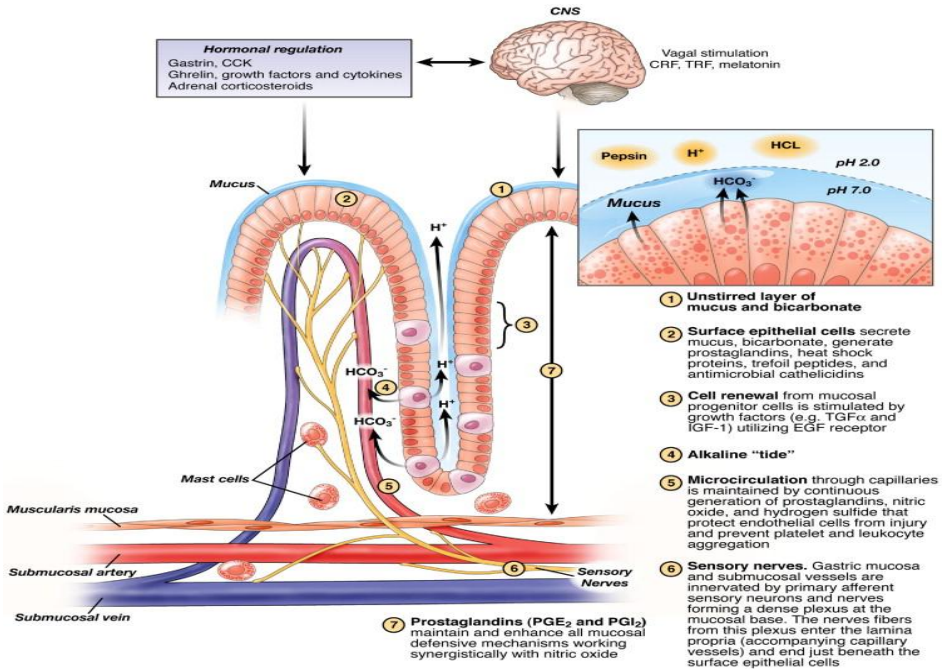


Fig. 2. Gastric mucosal defense mechanism

Mucus and bicarbonate secretion (Allen & Garner, 1980) complement each other and little protection can be afforded by either if functioning in isolation. Secretion of alkali from surface epithelial cells into the unstirred mucus layer thereby forming a pH gradient offers better protection than alone (Williams and Turnberg, 1981). Recent studies suggest that vagal cholinergic stimulation and luminal acid control gastric bicarbonate secretion (Fandriks and Jonson, 1990). The protective zone produced by bicarbonate transport into the mucus gel is termed as 'mucus-bicarbonate barrier' offering gastro protection against damaging factors (Wallace, 1992).

Gastric mucosal renewal and restitution

The rapid proliferation of gastric mucosa plays an important role in mucosal protection during normal state and following mucosal damage. After mucosal damage, the undifferentiated neck cells proliferate, towards the lumen and differentiate into surface epithelial cells. The proliferation of gastric mucosal cell is the intrinsic property to fight against any damage. Following extensive damage of the surface epithelial cells, repair occurs within a few hours, through a process called restitution which is not due to cell proliferation. This restitution process is augmented by high bicarbonate concentration and PGs do not appear to be involved in restitution (Goel and Bhattacharya, 1991).

Epidermal growth factors (EGF)

EGF is the most studied growth factor found in abundance in human salivary glands, bruneers glands and pancreas. Considerable evidence has revealed the secretion of various growth factors into the gastrointestinal lumen (Konturek *et al*., 1984). The presence of EGF like immune reactivity in human gastric secretion has been recently demonstrated (Konturek *et al*., 1981).

Mucosal and sub mucosal blood flow

Mucosal blood flow constituents an important line of mucosal defense. It plays a vital role in protecting the mucosa by delivering oxygen, nutrients and bicarbonate to the cells and removing the hydrogen ion that has penetrated the mucus-bicarbonate and epithelial barrier. Prostaglandins appear to be important in regulating mucosal blood flow because in NSAIDs induced ulcer decrease in gastric mucosal blood flow has been observed (Kitahora and Guth, 1987; Goel and Bhattacharya, 1991).

Endogenous Prostaglandins

Prostaglandins (PGs) are synthesized in large amounts by the gastric and intestinal mucosa. The PGE and PGI series of PGs have been shown to protect the deeper mucosal cells from the experimental necrotic damage (Miller, 1983) and some studies also suggest that a deficiency in prostaglandin production may contribute to ulcer formation (Dajani, 1986)

PGs increase gastric mucosal blood flow, mucus and bicarbonate secretion strengthens the mucus bicarbonate barrier. Since PGs protects the gastric mucosa by influencing all aspects of cytoprotective

mechanism. Any drug (NSAIDs) or abnormal physiological (Stress) or pathological (*H. pylori*) condition which inhibits prostaglandin biosynthesis, is expected to cause gastric damage (Goel and Bhattacharya, 1991).

Sulphydryl compounds (SC)

Non protein sulphydryl compounds are present in high concentration in the gastric epithelium. The major component of the SC is reduced glutathione (Karmeli *et al*., 1996) which is capable of binding to reactive free radicals which are generated during tissue ischemia and injury induced by noxious agents like ethanol (Avila *et al*., 1996). The precise role of SC in gastroduodenal cytoprotection remains unclear, but they appear to be involved that the bloking agent can reduce the cytoprotection effect of PGs in stomach (Karmeli *et al*., 1996).

Endogenous antioxidants

Antioxidants are defined as "any substance that even when present at low concentration compared with those of an oxidizable substrate, significantly delays or prevent oxidation of that substrate". Uncontrolled oxidation in aerobic organisms produces oxidative stress, cell damage and eventually cell death.

These free radical scavenging enzymes are the first line defense against oxidative injury within the cell and are known as preventive antioxidant. They remove the reactants involved in inhibition of the free chain reaction (Buettner, 1993).

Many plant secondary metabolites act as potent antioxidants. The natural antioxidant defense has shown that free radical scavenger/antioxidant such as SOD, Vitamin E, Vitamin C, Vitamin A, Glutathione reduces the mucosal injury induced by different mediators (Sharma and Gupta, 1997).

Nitric oxide (NO)

It is well reported that NO formed by constitutive enzyme plays an important role in the modulation of gastric mucosal integrity by interacting with sensory neuropeptides and endogenous prostaglandins (Tekeuchi *et al*., 1995).

Nitric oxide also inhibits the pentagastrin induced acid secretion in rats (Esplugues *et al*., 1993). Musin secretion by rat gastric cells has been found to be stimulated by nitric oxide and c-GMP.

TREATMENT OF ULCER

Antacids

Antacids are now prescribed mainly for symptomatic relief and widely accepted for self-medication. They are used to produce relief to the gastric pain associated with hyperchlorhydria. The majority of antacids is based on combination of calcium, aluminium and magnesium all of which cause side effects. At low dose antacid are ineffective in neutralizing acid in the stomach, extremely high doses of antacids are required to completely neutralize the excess acid in the stomach. The clinical status of antacid is in of flux- (Berstad, 1982; Ippoleti *et al*., 1983; Kumar *et al*., 1984). However, long term use of these antacids have been shown to produce significant mucosal protection (Goel and Bhattacharya, 1991).

Antacids are used in combination to give both immediate and sustained action, to minimize undesirable effects by using a lower dose of each component and to use one component to antagonize side effects of another (e.g. laxation versus constipation). The most common combination is that of Al $(OH)_3$ and Mg $(OH)_2$.

Histamine H_2-receptor Antagonist

H_2-receptor antagonists can competitively inhibit histamine action at all H_2-receptor but their main clinical use is as inhibitors of gastric acid secretion (Lund ell, 1975; Feldman and Burton, 1990). H_2-receptor antagonist also decreases gastric volume and pepsin concentration in the gastric content. H_2-receptor antagonist inhibits gastric acid secretion elicited by histamine or by gastrin in a dose dependent and competitive manner. These agents decrease both basal and food stimulated acid secretion by 90% or more. H_2-receptor antagonist also decreases intrinsic factor secretion from parietal cells. The most commonly used drugs are cimetidine (Shrees and Roberts, 1981) and ranitidine (Woodings *et al*., 1980; Shree and Roberts, 1981). Newer H_2-receptor antagonist, such as nizatidine, and famotidine (Orr *et al*., 1988) are also available. Recently roxatidine, roxatidine, has been shown to be devoid of such adverse effects and more potent and longer acting.

Muscarinic antagonists

Muscarinic antagonists reduce basal secretion of gastric acid by 40-50% however; stimulated secretion is inhibited to a lesser extent. Vagal stimulation produces an increased secretion of histamine and gastric acid that can be blocked by either nicotine or muscarinic antagonist (pirenzipine) (Del-Tacca *et al*., 1989).

Proton pump inhibitors

The ultimate mediators of acid secretion is the H^{+}-K^{+}ATPase enzyme (proton pump) found in the smooth membrane structure in the parietal cell called tubulo-vesicles as long as the cell is not secreting acid. (Sachs and Shin, 1995).

A strict relationship between inhibition of acid secretion and block of H^{+}-K^{+}ATPase by omeprazole has also been demonstrated. The inhibition is due to the irreversible interaction of the active compound with the SH group of H^{+}-K^{+}ATPase forming a disulphide bond (I'm *et al*., 1985). Similar to omeprazole other substituted benzimidazole such as timoprazole and picoprazole also inhibit acid secretion (Sewing, 1984).

The long term use of omeprazole results in complete inhibition of H^{+}-K^{+}ATPase enzyme. Prolonged treatment with in patients Omeprazole results in achlorhydria (blocked in acid secretion) and hypergastrenemia (Lamberts *et al*., 1993).

CYTOPROTECTIVE DRUGS

Misoprostol and Enprostil

PGE_2 and PGI_2, the predominant prostaglandins synthesized by the gastric mucosa, inhibit the secretion of acid and stimulate the secretion of mucus and bicarbonate. Misoprostol (analog of PGE_1) inhibit gastric acid secretion by inhibiting the histamine-mediated stimulation of the parietal cell. Currently misoprostol is used to prevent gastric ulceration in patients who use large doses of Aspiring-like drugs for the treatment of arthritis (Roth *et al*., 1989; Penney *et al*., 1994). Misoprostol and enprostil cause abdominal pain and diarrhea as well as bleeding in the first trimester of pregnancy (Levis *et al*., 1992).

Sucralfate

Sucralfate is a complex of aluminium hydroxide and sulfate sucrose which promote healing of ulcers. Sucralfate a non absorbable aluminium salt of sucrose octasulfate, served as reference compounds. The drug sucralfate is claimed to inhibit peptic activity. It has got a specific affinity for ulcer and protects the ulcer as natural mucus dose (Libeskind, 1982; Lichtenberger, 1983).

Tripotassium dicitrato bismuthate

It has been used clinically in duodenal ulcers refractory to cimetidine. Unfortunately it is uncertain how it works. Although it does adhere to the raw surface of an ulcer (Koo *et al*., 1982), this 'Band-Aid' action seems unlikely to protect from acid and peptic attack. Under the influence of bismuth, the microvilli of epithelial cell in the duodenal mucosa return to their normal height whereas cimetidine has no such action. It is a bismuth chelate which promotes the healing of peptic ulcers. It is possible that bismuth has a role in the maintenance of mucosal repair and a short course of treatment may provide a depot of bismuth, which gives some months of protection against relapse (Pounder, 1984). It may act by coating the ulcer and protecting the ulcer and protecting it in particular it absorbs pepsin. It also acts as bactericidal against *H. pylori.*

Deglycyrrhizinizes liquorice (DGL) and Carbenoxolone

DGL differs from pure liquorice-containing products and indeed, carbenoxolone in having a sufficiently low glycyrrhizinic acid content to render it free of the potential side-effects of carbenoxoline. The evidence for a beneficial effect of maintenance carbenoxolone is weak. In addition, in the latter study, hypertension and hypokalemia were found in 27% and 15% respectively in patients over the age of 60.

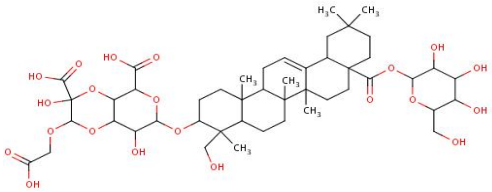
Table 1: Indian medicinal plants reported for its ulcer protective activity

S.No	Plant name (Family)	Plant Part used	Type extract	Experimental model	Reference
1.	Aegle marmelos (Rutaceae)	Unripe fruit	----	Hypothermic restraint stress, absolute ethanol, and indomethacin	Dhuley *et al.,* 2004
2.	*Anogeissus latifolia* (Combretaceae)	Bark	50% aqueous alcoholic	Aspirin, cold-resistant stress , pylorus ligated and ethanol	Govind Arajan *et al.,* 2006
3.	*Azadirachta indica* (Meliaceae)	Leaves	Ethanolic	-	Subapriya *et al.,* 2005
4.	*Bocopa monniera* (Scrophulariaceae)	Whole plant	Juice	Cold restraint stress, aspirin, ethanol, pylorus ligation	Rao *et al.,* 2000
5.	*Camellia sinensis* (Theaceae)	Seeds	Saponin fraction	Ethanol and indomethacin	Yoshikawa *et al.,* 2005
6.	*Centella asiatica* (Umbelliferae)	Whole plant	Juice	Cold restraint stress, indomethacin, ethanol, pylorus ligation, acetic acid	Sairam *et al.,* 2001
7.	*Curcuma longa* (Zingiberaceae)	----	Ethanol and ethyl acetate	Pylori-ligation	Kim *et al.,* 2005

8.	Desmodium gangeticum (Leguminosae)	----	Ethanolic	Ethanol, pylorus ligation, aspirin & cold-resistant stress	Dharmani *et al.,* 2005
9.	*Glycyrrhiza glabra* (Papilionaceae)	Root	Aqueous	Indomethacin	Aly *et al.,* 2005
10.	*Momordica charantia* (Cucurbitaceae)	----	----	Helicobacter pylori	Grover *et al.,* 2004
11.	*Moringa oleifera* (Moringaceae)	Seed	----	----	Richa *et al.,* 2005
12.	*Musa sapientum* (Musaceae)	Pulp	Methanolic extract	Cold restraint stress and anti-H. pylori activity	Goel *et al.,* 2001
13.	*Nigella sativa* L. (Ranunculaceae	----	Oil	Ethanol	Kanter *et al.,* 2005
14.	*Ocimum sanctum* L. (Lamiaceae)	Leaves	Methanolic extract	Ethanol, pylorus ligation, aspirin & cold-resistant stress	Goel *et al.,* 2005
15.	*Petrocarpus marsupium* **(**Papilionaceae)	Heart wood	Methanolic extract	Cold resistant to stress, aspirin, ethanol, pylorus ligation in NIDDM rats	Joshi *et al.,* 2004
16.	*Plantago major* L. (Plantaginacea)	Leaves	----	----	Eksp Klin *et al.,*2005
17.	*Petrocarpus Santalinus* (Leguminosae)	----	Ethanolic extract	Ibuprofen	Narayan *et al.,* 2005
18.	*Rauwolfia serpentine* (Apocynaceae)	----	----	Stress induced	Heloina de *et al.,* 2008
19.	*Rheum ribes* L. (Polygonaceae)	Leaves	Methanolic extract	Ethanol induced, pylorus ligation	Rakesh K Sindhu *et al.*, 2010
20.	*Solanum nigrum* (Solanaceae)	Fruit	Ethanolic extract	Cold resistant to stress, aspirin, ethanol, pylorus ligation	Jainu *et al.,* 2005

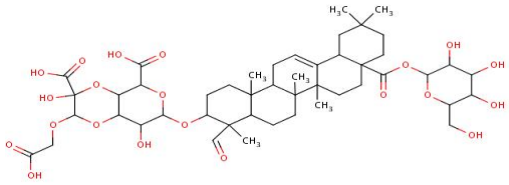
CHEMICAL REVIEW

Basellasaponin A: The first report on chemical investigation on *B. alba* showed the presence of Basellasaponin A. (Toshiyuki M *et al*., 2001).



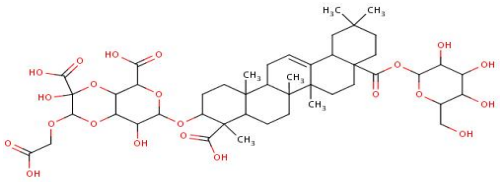
Molecular Formula: $C_{47}H_{70}O_{21}$, **Melting Point:** 228-230 ^{0}C, **Molecular Weight:** 970.44g. .mol^{-1}

Basellasaponin B: (Toshiyuki M *et al*., 2001).
Molecular Formula: $C_{47}H_{68}O_{21}$, **Melting Point:** 228-230 ^{0}C, **Molecular Weight:** 968.42g. .mol^{-1}



Basellasaponin B

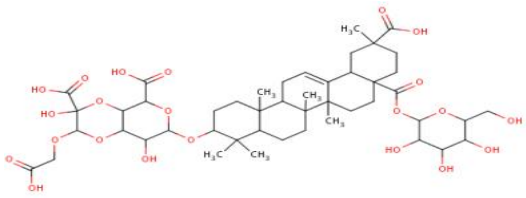
Basellasaponin C: (Toshiyuki M *et al*., 2001).
Molecular Formula: $C_{47}H_{68}O_{22}$, **Melting Point:** 226-228 ^{0}C, **Molecular Weight:** 984.42g. .mol^{-1}



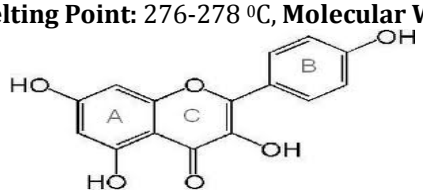
Basellasaponin C

Basellasaponin D: (Toshiyuki M *et al*., 2001).
Molecular Formula: $C_{47}H_{68}O_{22}$, **Melting Point:** 226-228 ^{0}C, **Molecular Weight:** 984.42g. .mol^{-1}

Basellasaponin D

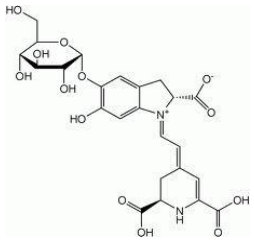


Kaempherol:
Molecular Formula: $C_{15}H_{10}O_6$, **Melting Point:** 276-278 ^{0}C, **Molecular Weight:** 286.23 g. mol^{-1}



Kaempherol

Betalain: Molecular Formula: $C_{24}H_{26}N_2O_{13}$, **Molecular Weight:** 550.46 g. mol^{-1}



Betanin
Betanin = Betacyanin = Betalain

PHARMACOLOGICAL REVIEW
In vitro pharmacology
Mucilaginous substances from *Basella alba* was studied for in vitro glucose entrapment and compared to glucomannan powder. The mucilage solution showed gel-forming characteristics and concentration response on glucose entrapment activity. (Roshan A *et al*., 2012).
Nutraceutical
In the present scenario, there is a rising demand for natural sources of food colorants with nutraceutical benefits with anthocyanins. Antioxidant assays were done by enzymatic assay methods like Super oxide dismutase, Catalase and Peroxidase, *Basella alba* showed higher activity.
Anti-Inflammatory, Cytotoxicity and Antioxidant activities
Crude aqueous extract possesses mild antioxidant and no tyrosinase inhibitory activity. *Basella alba* is used traditionally in Thailand for anti-inflammatory, cytotoxicity and antioxidant activities of anti-inflammatory remedies. This allowed the selection of lead extracts for various ethno pharmacological researches.
Antimicrobial activity
The methanolic extracts exhibited marked antimicrobial activity against gram positive and gram negative bacteria and fungi. *Basella alba* showed good inhibitory activity against Aspergillus niger. (Premakumari KB *et al* 2010).

Table 2: Plant profile and Botanical classification

PLANT PROFILE	
Botanical Name	*Basella alba L.*
Family	Basellaceae
Common Name	Indian Spinach
Synonyms	B.rubra, B.cordifolia. B. Lucida.
BOTANICAL CLASSIFICATION	
Kingdom	Plantae
Phylum	Magnoliophyta
Class	Magnoliopsida
Order	Caryophyllales
Family	Basellaceae
Genus	*Basella*
Species	*alba*

Distribution

B. alba is usually considered native of southern Asia (India).

Description

B. alba is a widely cultivated, cool season vegetable with climbing growth habit. It is a succulent, branched, smooth, twining herbaceous vine, several meters in length. Stem are Purplish or green. Leaves are fresher, ovate or heart-shaped, 5 to 12 cm long, stalked, tapering to a pointed tip. Spikes are auxiliary, solitary, 5-29 cm long, and purple when mature. Mainly leaves and stems are used for the medicinal purpose.

Fig.3. Plant of *Basella alba L.*

Medicinal property and uses:

Daily consumption of *Basella alba* has a positive effect on total-body vitamin A stores in men. (Haskell MJ *et al*., 2004).

B. alba leaves used for the treatment of hypertension by Nigerians in Lagos, and malaria in Cameroonian folk medicine. The plant has been reported for its antifungal, anticonvulsant, analgesic, anti-inflammatory and androgenic activities and for the treatment of anemia.

It is traditionally used in Ayurveda system of medicine to bring sound refreshing sleep when it is applied on head about half an hour before bathing (Anandaraja Gopal K *et al*., 2011).

It is used for hemorrhages, skin diseases, sexual weakness, ulcers and as laxative in children and pregnant women.

Basella mucilage has been used in Thai traditional medicine as a topical application for irritant, bruise, ringworm and laboring. Stem and leaves are used as mild laxative, diuretic and antipyretic.

B. alba leaves and stem for anticancer such as melanoma, leukemia and oral cancer.

B. alba is administered orally for the treatment of anal prolapsed or hernia.

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Ciation of this article

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