ABSTRACT
Peptic ulcer represents a serious medical condition. Approximately 500,000 new cases are reported each year, with 5 million people alone affected in United States alone. The highest risk of contracting peptic ulcer disease are those generations born around the middle of the 20th century. Ulcer disease has become a disease predominantly affecting the older population, with the peak incidence occurring between 55 and 65 years of age. Patients with peptic ulcer disease may present with a range of symptoms, from mild abdominal discomfort to catastrophic perforation and bleeding. Most patients with peptic ulcer disease present with abdominal discomfort, pain, or nausea. The various herbal plants are claimed to possess anti-ulcer. Preclinical experiments are done to screen the anti-ulcer activity in various herbal drugs using various animal models discussed in screening assays.

KEYWORDS: Peptic Ulcer, Symptoms, Herbal Plants.

INTRODUCTION
Peptic ulcer indicates an interruption in the continuity of the intestinal mucosa as a result of the action of acids and pepsin. The ulceration can occur in the stomach, duodenum and sometimes in the jejunum. Ulcers may range in size from several millimeters to several centimeters. Ulcers are delineated from erosions by the depth of penetration (erosions are more superficial and do not involve the muscularis mucosae). Gastritis indicates inflammation of the gastric mucosa, without ulceration. Gastritis is usually a precursor of ulceration, but either condition can occur in isolation.\(^3\)
Most common type of ulcers

Peptic ulcer
Any ulcer that is exposed to pepsin is referred to as peptic ulcers. Peptic ulcers are found in the lining of stomach or duodenum. Pepsin is normally present along with hydrochloric acid in the stomach lining.[3,9]

Duodenal Ulcer
When a peptic ulcer is in duodenum, it is called a duodenum ulcer. This type of peptic develops in the first part of the small intestine. Some of the symptoms of a duodenal ulcer are interestingly quite opposite to those of gastric ulcers. Duodenal ulcers are the most common ulcers found in western world.[5,6]

Gastric ulcer
When a peptic ulcer is in stomach, it is called a gastric ulcer. The symptoms of gastric ulcers are more specific than peptic ulcer symptoms.

Esophageal ulcer
This type of ulcer occurs in the lower end of your esophagus. Esophageal ulcers are often associated with a bad case of acid reflux, or GERD as it is commonly called (short for Gastro Esophageal Reflux Disease).

Bleeding ulcer
Internal bleeding is caused by a peptic ulcer which has been left untreated. When this happens, it is now referred to as a bleeding ulcer-this is the most dangerous type of ulcer. See your doctor immediately if you are showing symptoms.[3]

Refractory ulcer
Refractory ulcers are simply peptic ulcers that have not healed after at least 3 months of treatment.

Stress ulcer
Stress ulcers are a group of lesions found in the esophagus, stomach or duodenum. These are normally only found in critically ill or severely stressed patients.
Epidemiology of peptic ulcer disease

Ulcers can occur at any age, including infancy and childhood, but are most common in middle-aged adults. Peptic ulcer (gastric and duodenal) occurs most commonly in patients aged 30 to 50 years, although patients over the age of 60 years account for 80% of deaths even though they only account for 15% of cases. Prevalence has shifted from predominance in males to similar occurrences in both sexes. Lifetime prevalence is approximately 11% to 14% for men and 8% to 11% for women.\(^{[4-6]}\)

Aetiology/pathophysiology

H pylori and NSAIDs disrupt the normal mucosal defense and repair, making the mucosa more susceptible to acid. H pylori infection is present in 80% to 90% of patients with duodenal ulcers and 70% to 90% of patients with gastric ulcers. If H pylori is eradicated, only 10% to 20% of patients have recurrence of peptic ulcer disease, compared with 70% recurrence in patients treated with acid suppression alone. Although the cause of peptic ulceration in some patients is apparent (such as aspirin usage), in most cases the pathogenesis is unknown.

There are, however, a number of factors which have been identified as possibly leading to peptic ulceration, namely.

- Helicobacter pylori infection: As already mentioned, H pylori is present in the mucosa of 80% of patients with peptic ulceration and gastritis, while it is only present in 20% of the normal healthy population.
• Genetic tendency: A genetic tendency occurs especially in the case of duodenal ulceration.

• Furthermore, a family history exists in 50% to 60% of children with duodenal ulcer.

• Medicine: Medicine such as aspirin, NSAIDs and corticosteroids can cause peptic ulceration.

• Alcohol: Chronic drinkers of alcohol develop ulceration, while the occasional drinker normally only develops gastritis. Although alcohol is identified as a strong promoter of acid secretion, no definite data link moderate amounts of alcohol to the development or delayed healing of ulcers.[1,3,6]

• Cigarette smoking: It is a risk factor for the development of ulcers and their complications. In addition, smoking impairs ulcer healing and increases the incidence of recurrence. Risk correlates with the number of cigarettes smoked per day.

• Stress: Severe physiologic stress can cause peptic ulcer disease, for example burns, central nervous system trauma, surgery and severe medical illness.

• Bile salts and pancreatic enzymes: They can cause ulceration when they leak back into the stomach on account of an inefficient pyloric sphincter, or when stasis of the intestinal bolus occurs as a result of partial obstruction.

• Toxins secreted by micro-organisms: e.g. toxins secreted in chronic gastroenteritis.

• Hypersecretory states. This is an uncommon cause. Examples include gastrinoma (Zollinger- Ellison syndrome), multiple endocrine neoplasia (MEN-I), antral G cell hyperplasia, systemic mastocytosis and basophilic leukemias. Very few patients have hypersecretion of gastrin (Zollinger-Ellison syndrome).

• Chronic conditions: Diseases associated with an increased risk of peptic ulcer disease include cirrhosis, chronic obstructive pulmonary disease, renal failure and organ transplantation.

• Rare conditions: Other rare, miscellaneous causes include radiation-induced or chemotherapy-induced ulcers, vascular insufficiency and duodenal obstruction. These
factors weaken the normal protective barrier of the mucous membrane of the stomach and small intestine and may cause increased secretion of acid and pepsin, with resulting inflammation and subsequent ulceration.\textsuperscript{[1,3,6]}

**Figure 2 PATHOGENESIS OF PEPTIC ULCER**

**Figure 3 PATHOGENESIS OF PEPTIC ULCER**

**Symptoms**
Most patients with peptic ulcer disease present with abdominal discomfort, pain or nausea. The pain is located in the epigastrium and usually does not radiate. However, these symptoms are neither sensitive nor specific. Pain radiating to the back may suggest that an ulcer has penetrated posteriorly, or the pain may be pancreatic in origin. Pain radiating to the right upper quadrant may suggest disease of the gallbladder or bile ducts. Patients may describe the
pain of peptic ulcer as burning or gnawing, or as hunger pains slowly building up for 1–2 hours, then gradually decreasing. Use of antacids may provide temporary relief. Classically, gastric ulcer pain is aggravated by meals, whereas the pain of duodenal ulcers is relieved by meals. Hence, patients with gastric ulcers tend to avoid food and present with weight loss, while those with duodenal ulcers do not lose weight.\cite{9,2}

**Diagnosis of peptic ulcer disease**

Symptoms depend on ulcer location and patient age. Many patients, particularly elderly patients, have few or no symptoms. Pain is however the most common symptom, often localised to the epigastrium or mid-epigastrium and relieved by food or antacids. The pain is described as burning, gnawing, constant or annoying, or sometimes a sensation of hunger. The course is usually chronic and recurrent. Only about 50% of patients present with the characteristic pattern of symptoms.\cite{1,4}

Gastric ulcer symptoms often do not follow a consistent pattern (for example, eating sometimes exacerbates rather than relieves, pain). This is especially true for pyloric channel ulcers, which are often associated with symptoms of obstruction (for example bloating, nausea and vomiting) caused by oedema and scarring. In general, however, in gastric ulcers, pain typically starts whenever the stomach is empty (usually approximately an hour after eating), and is generally relieved by antacids or food but aggravated by alcohol and caffeine. Weight loss and gastrointestinal bleeding occur more frequently with gastric ulcers. Patients can experience weight loss of 5 kg to 10 kg and although this could indicate carcinoma, especially in people over 40 years, on investigation a benign gastric ulcer is found most of the time.\cite{1,4,5}

Duodenal ulcers tend to produce more consistent pain. Pain is absent when the patient awakens but appears midmorning, is relieved by food, but recurs two to three hours after a meal. Pain that awakens a person at night, a few hours after falling asleep, is also common and is highly suggestive of duodenal ulcer. The pain then usually subsides by morning and is often relieved after eating. This is not commonly noticed in gastric ulceration. In neonates, perforation or haemorrhage may be the first manifestation of duodenal ulcer. Haemorrhage may also be the first recognized sign in later infancy and early childhood, although repeated vomiting or evidence of abdominal pain may be a clue. Diagnosis of peptic ulcer is by patient history, and confirmed by endoscopy and testing for H pylori.\cite{1,4-8}
Carbon-13 urea breath tests detect active H pylori infection by testing for the enzymatic activity of bacterial urease. In the presence of urease produced by H pylori, labeled carbon dioxide is produced in the stomach, absorbed into the bloodstream, diffused into the lungs and exhaled.

Stool or faecal antigen testing identifies active H pylori infection by detecting the presence of H pylori antigens in stools.

Serology, which is immunoglobulin G (IgG) based, can be measured in serum, plasma or whole blood. It will, however, not distinguish between a previous or a current infection.

Biopsy-based urease tests, which are invasive and can only be done at gastroscopy or in the acute hospital setting. There are two methods for this test. In the CLO test (“Campylobacter-like organisms” test, the rapid urease test) a fragment of mucosal membrane is placed into a special jelly which undergoes a colour change in 10 to 20 minutes, or the specimen is sent for histology which may take up to 24 hours to obtain the result.

Endoscopy (EGD)
An endoscope is a thin, flexible tube with a tiny camera at the end. The patient is given a mild sedative, and then the tube is passed through mouth in to the lining of the stomach to diagnose a peptic ulcer. Tiny samples of the tissue will be taken (biopsy), which are examined under a microscope.

If a diagnostic imaging test reveals an ulcer, the patient will most likely have a test to see if H pylori bacteria are present.\[3\]
CLASSIFICATION OF PEPTIC ULCER DRUGS[9]

1. Gastric acid secretion inhibitors
   (a) *H₂* antihistamines: Cimetidine, Ranitidine, Famotidine, Roxatidine.
   (b) *Proton pump inhibitors*: Omeprazole, Esomeprazole, Lansoprazole, Pantoprazole, S (-) Pantoprazole, Rabeprazole, Dexrabeprazole.
   (c) *Anticholinergics*: Pirenzepine, Propantheline, Oxyphenonium.
   (d) *Prostaglandin analogues*: Misoprostol.

2. Gastric acid neutralizers (Antacids)
   (a) *Systemic*: Sodium bicarbonate, Sod. Citrate.
   (b) *Nonsystemic*: Magnesium hydroxide Mag. trisilicate, Aluminium hydroxide gel, Magaldrate, Calcium carbonate.

3. Ulcer protectives
   Sucralfate, Colloidal bismuth subcitrate (CBS).

4. Anti-*H. pylori* drugs
   Amoxicillin, Clarithromycin, Metronidazole, Tinidazole, Tetracycline.

**Branded names of drug**
* Cimetidine: Cimetin, Tagamet, Aciloc, Azylec.
* Famotidine: Pepcid
* Nizatidine: Axid
* Lansoprazole: Prevacid
* Rabeprazole: Aciplex
* Pantoprazole: Protonix

**Herbal drugs**
* Alsarex (Himalaya charak product)
* Herbolax
* Boswellia
* Vara churna
* Triphala[11]
Animal models used in the screening of anti ulcer activity\[^{[47]}\]

Various screening models are used for the screening of the anti ulcer activity .it helps to understanding the aetiology of the ulcer and screening of anti ulcer agents.

- Aspirin induced ulcers
- Ethanol induced ulcers
- Pylorus ligation induced ulcers
- Water immersion stress induced ulcers
- Indomethacin induced ulcers
- Histamine induced ulcers
- Reserpine induced ulcers
- Serotonin induced ulcers
- Acetic acid induced ulcers
- Hydrochloric acid induced ulcers

Some of the detailed procedures carried out are:

Aspirin induced ulcers

Principle

Aspirin is a NSAID which inhibit the synthesis of prostaglandins. Prostaglandins protect the gastric mucosa by producing leukotrienes and bicarbonate ions. Aspirin also inhibit the gastric peroxidase and may increase mucosal hydrogen peroxide and hydroxyl ions level to cause oxidative mucosal damage.

Procedure

Albino rats of either sex weighing between 150-200gms are divided into five groups of six animals in group. The animals are fasted for 24hours. The test drug in varying concentrations based on the design of the experiment is administered orally in 2% gum acacia solution 30 minute prior to aspirin at dose of 200mg/kg. 4 hours later the rats are sacrificed by using anaesthetic ether and their stomachs dissected and they were opened along greater curvature for the determination of gastric lesions. Ulcer index calculated by noting the number of ulcers per animal and severity scored by observing the ulcers microscopically with the help of 10x lens and scoring is done.
Ethanol induced ulcers

Principle
Alcohol causes secretion of gastric juice and decrease mucosal resistance due to which protein content of gastric juice is significantly increased by ethanol. This could be leakage because of plasma protein in the gastric juice with weakening of mucosal resistance barrier of gastric mucosa, this leading to peptic ulcer.

Procedure
Albino rats of either sex weighing between (150-200gms) are divided into group. The animals are fasted for 24 hours with free access water. Animals are given test drugs or standard drug. 1 hour later 1ml/200gm of 99.80% alcohol is administered orally to each animal. The animals were anaesthetized 1 h latter with ether and stomach was incised along the greater curvature and ulceration was scored. The number of ulcers and the length of each ulcer were measured. Ulcer index was calculated using severity scores and avg no of ulcers per animal

Pylorus ligation induced ulcers

Principle
In Pylorus ligation pylorus part of the stomach was ligated it helpful to produce the ulcers in rats by stopping passage of gastric contents from stomach it creates the acidic medium in stomach for longer time and produces the ulcer.

Procedure
Albino Wistar rats of either sex weighing between (150-200 gms) are divided into groups of a animal. sIn this method albino rats are fasted in individual cages for 24 hours. Test drug or standard drug or control vehicle is administered 30 minute prior to pyloric ligation. Under light ether anaesthesia, the abdomen is opened and the pylorus was ligated. The abdomen is then sutured. At the end of 4 hours after ligation the animals are sacrificed with excess of anaesthetic ether, and the stomach is dissected out gastric juice is collected were drained into tubes and were centrifuged at 1000 rpm for 10 minutes and the volume is noted. The pH of gastric juice is recorded by pH meter. Then the contents are subjected to analysis for free and total acidity. The stomachs are then washed with running water to see for ulcers in the glandular portion of the stomach. The numbers of ulcers per stomach are noted and severity of the ulcers scored microscopically with the help of 10x lens.
Histopathological studies were conducted by fixing stomach tissues in 10% formalin for 24 h. The formalin fixed specimens are embedded in paraffin and section (3-5μm) and stained with haematoxylin and eosin dye. The histochemical sections are evaluated by light microscopy.

**Acetic acid induced ulcers**

**Principle**

Acetic acid enhances the ulceration in stomach by increasing the acidity of stomach contents and acetic acid also causes gastric obstruction leads to the ulceration.

**Procedure**

Rats were anaesthetized with pentobarbitone (35 mg/kg, ip). The abdomen was opened and the stomach was visualized. A cylindrical glass tube (6 mm in diameter) was tightly placed upon the anterior serosal surface of the glandular portion of stomach 1 cm away from the pyloric end. 50% acetic acid (0.06 ml/animal) was instilled into the tube and allowed to remain for 60 sec on the gastric wall. After removal of acid solution, the abdomen was closed in two layers and animals were caged and fed normally. Standard and test drug administered orally 4 h after the application of acetic acid and continued up to 9 days after induction of ulcer. The animals were then sacrificed after 18 h of the last dose of drug on 10th day of experiment to assess the ulcer size and healing. Ulcer index was calculated based upon the product of length and width (mm2/ rat) of ulcers.

**Literature Review**

Mr. G. Venthapooshan et al 2010 had studied about antiulcer activity Of Mimosa Pudica leaves in rats. He had done preliminary phytochemical analysis and toxicity studies. He had performed various antiulcer screening methods like Where he found out that NSAIDS like aspirin cause gastric mucosal damage by decreasing PG levels through the inhibition of PG synthesis. The antiulcer activity of Mimosa Pudica extract in pylorlous ligation model is resulting in to the significant reduction of gastric volume, total acidity, free acidity, gastric Juice. The preliminary phytochemical studies revealed the presence of flavanoids in the methanolic extract of the plant Mimosa Pudica has antiulcerogenic activity. From the result it can be concluded that antulcer activity of Mimosa Pudica could be due to modification of defective factor and improved gastric cytoprotective.[10-16]

Mr. Vinay Kumar Varma et al 2012 reported about anti ulcer activity of Moringa Oleifera leaves against aspirin and ethanol induced gastric ulcer in rats. The activity was tested in
Wistar albino rats. At dose level of 125,250, and 500 mg/kg orally and compared with ranitidine as standard during ulcer condition there was increase in gastric mucosal SOD and LPO levels. The alcoholic leaves extract of Moringo Oleifera has shown ulcer protective effect.. The extract decrease ulcer and acid pepsin secretion a change was also seen in SOD, CAT, and LPO levels. In rat gastric mucosa due to antioxidant property of Moringa leaves. From the result it can be concluded that anti ulcer activity of Moringa Oleifera may be due to auto digestion of gastric juice and decrease mucosal blood flow and break down of mucosal barrier.\[17-21\]

Kamal Kumar et al 2013 discussed about anti ulcer activity of ethanol extract of the stem bark of Carreya Arborea Roxb. He had done preliminary phytochemical analysis and toxicity studies. He had performed various anti ulcer screening methods. The preliminary phytochemical analysis of EECA showed the presence of carbohydrate, glycosides, phytosterols, phenolic compa, tannin and saponin. Phytol constituents like tannins and saponins may be responsible for anti ulcer activity of EFCA he concluded that ethanol extract of stem bark of Carreya Arborea Roxb have shown significant effect on the ulcer induced by three models such as ethanol induced, cold stress induced and pyloric ligation model. The maximum ulcer protection EECA has been shown in the ethanol induced and cold stress induced models and significant effect was found at both 300 mg/kg and 600 mg/kg dose levels.\[22-25\]

Geham salah eldin moran et al 2016 studied anti ulcer activity of Red Pepper and Garlic Bulb against indomethacin induced gastric ulcer in rats. He had done bio chemical and histopathological study. For this he used chemicals indomethacin and ranitidine and divide the animals into five groups. He involves various methodsand parameters for the determination of ulcer. The oral administration of RCP and GA juice as pre treatment showed a significant reduction in lesion index. The mechanism of gastro protective maybe attributed to significant reduction. In volume gastric juice total acidity of gastric juice gastric ulcer index, serum interleukin-1, gastric cyclooxygenase activity compared with ulcerated control group and he conclude that of gastric affronts of indomethacin by administration of fresh juice of red pepper and garlic bulb regimen is indicative of their excellent gastro protective and antioxidant potential in rats.\[26-30\]

V.I Borikar et al., 2009 studied about anti ulcer activity of Bauhinia racemosa in rats. He conducted the experiment by dividing the Wistar rats into 5 groups T₁, T₂, T₃, T₄ and T₅
were given 0.5 ml normal saline aqueous extract in the dose ratio of 100 mg/kg body weight alcoholic extract can also be administrated after one hour paracetamol is administrated and the number of ulcers, ulcer scone percent incidence, ulcer index and healing index was recorded and he concluded that there was a decrease in percent of ulcer and ulcer index and increase in healing index. This may be due to the presence of flavonoids which may reduce Bauhnia racemosa has potent anti ulcer activity.\textsuperscript{31-38}

S. Gopinathan et al, 2014 researched about anti ulcer activity of Aloe Vera juice and Aloe Vera and Amla fruit combined in ethanol induced ulcerated Rats. He conducted the experiment by comparing the efficacy of both plant based juice with standard reference anti ulcer drug ranitidine. The administration of plant juice decrease the offensive factors like ulcer index and acid secretion and also reduced the amount of protein and carbohydrate in the stomach fluid further plant juice, Increased the defensive factors like activity of oxidase enzyme such as superoxide dismutase and reduced glutathione activity of alkaline phosphate and lipid peroxidase was higher in diseased condition of hemoglobin, RBC WBC count also normal after treatment with plant juice and he concluded that the plant juice having efficiency in gastro protective activity.\textsuperscript{39-42}

Salmah-Al-Radahe et al 2013 performed a work on antiulcer activity of swietenia mahagoni ethanol leaf extract against ethanol induced gastric ulcer. Where a treatment with either omeprazole or plant extract resulted in significantly protect of gastric mucosal injury and increase in mucous production flattening of gastric mucosal folds was also observed in rats pretreated with s. mahagoni leaf extract and he concluded that s. mahagoni has marked gastric protection along with reduction or obscene of oedema and leucocytes infiltration of submucosal layer.\textsuperscript{43-46}

Harsada pulcherrima etal 2011studied about te antiulcer activity of caesalpinia pulcherrima L.bark against aspirin induced ulcer in rats. He had done preliminary phytochemical screening and acute toxicity study. The current study shows that gastric juice in the group (cpi) and (cpA) recieved 200 and 300 mg/kg of the hydro alcoholic and aqueous extract of caesalpinia pulcherrima L respectively showed a significant increase in gastric juice pH, reduces the gastric volume, total acidity.\textsuperscript{47-50}

Raju.D et al., 2012 made study on the antiulcer activity of met. anolic extract of Terminalia chebula in wistar rats. He had done preliminary phytochemicals screening, macroscopical
and histopathological evaluation. He had performed various antiulcer screening methods like ethanol induced ulcer. METC at doses of 250,500mg/kg produce significant inhibition of gastric lesions induced by pylorous ligation induced ulcer and ethanol induced ulcer. The extract also show significant reduction in gastric volume, free acidity and ulcer index. From the result it can be concluded that methanolic extract of *Terminalia chebula* exhibit significant antiulcer activity experiment in rats. The extract posses antiulcerogenic as well as ulcer healing property. Which might be due to the presence of alkaloids, flavanoids, carbohydrates, glycosides, tannins, terpenoids, phenol and absence of fixed oil and steroids.[51-54]

**DISCUSSION AND CONCLUSION**

The anti-ulcer activity of the plant of *Mimosa pudica* was evaluated by employing aspirin, alcohol and pylorus ligation ulcer models. and suggested that *Mimosa pudica* extracts can suppress gastric damage induced by aggressive factors. Antiulcer activities of Carreya arborea were performed on Wister rats of either sex using ethanol induced, cold stress induced and pyloric ligation models. The 70% Ethanol extracts (300 and 600 mg/kg) showed significant antiulcer activity. The significant increase in the antiulcer activity of *Abutilon indicum* could be attributed to the presence of flavonoids (quercetin), alkaloids, tannins, saponin glycosides and phenolic compounds. Flavonoids are among the cytoprotective materials for which antiulcerogenic efficacy has been extensively confirmed. The *Swietenia mahagoni* leaf extract is capable of inhibiting gastric lesions formed by ethanol. *S. mahagoni* have been reported to contain flavonoids and it could be conceivable that the anti-ulcer activity of *S. mahagoni* leaf extract could be linked to the flavonoids since flavonoids are reported to protect the mucosa by preventing the formation of lesions by various necrotic agents (Saurez et al., 1996). The significant increase in the antiulcer activity of *Citrullus colocynthis* could be attributed to the presence of flavanoids, alkaloids, tannins, saponins glycosides and phenolic compounds. Flavanoids are among the cytoprotective materials for which antiulcerogenic efficacy has been extensively confirmed. It is suggested that, these active compounds would be able to stimulate mucus, bicarbonate and the prostaglandin secretion and counteract with the deteriorating effects of reactive oxidants in gastrointestinal lumen18. So the antiulcer activity of *Citrullus colocynthis* may be attributed to its flavanoids content.
Ulcer is an important gastrointestinal disease that caused by H.pylori and due to high intake of NSAIDS. The first objective of ulcer control programme is prevention of ulcer through identification of the causative organism and with the treatment of appropriate drug that also reduce the gastric acid secretion and there by heals ulcer. The studies were done on various experimental plants by the ethnolic and alcoholic extract of mimosa pudica, moringa olifera, carreya arborea, abutilion, swietenia mahagani, terminalia chebula, citrullus colosynthis against various antiulcer screening methods like aspirin induced ulcer, ethanol induced ulcer, pylorous ligation induced ulcer etc. they also reported that the antiulcer activity is due to the presence of phytochemical constituents like flavanoids, alkaloids, cardiac glycosides, carbohydrates, tannins, terpenoids. We acquired many information regarding ulcer trough this review.

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