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Nutraceuticals in Diabetes

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ABSTRACT

Diabetes mellitus (DM) is a heterogeneous metabolic disease that shares the characteristic of persistent hyperglycemia along with abnormalities in the metabolism of proteins, fats, and carbohydrates. Syzygium jambolanum commonly known as jamblang or jamun, java plum is a most important plant into the various traditional systems of medicine. Syzygium jambolanum is very effective in the treatment of diabetes mellitus, which is beneficial in abdominal diseases such as loss of appetite, abdominal pain, dysentery and irritable, bowl, syndrome. Sometimes, it is also used for indigestion. Diabetes, sysentery, and diarrhea are treated with jamun seed powder. Jamun leages are prescribed for nousea,vomiting,bleeding disorder and metrrhagia.

INTRODUCTION

The diabetes mellitus (DM) is a metabolic disorder characterized by hyperglycemia in the fasting plasma glucose 126 mg/dL and or 200 mg/dL is 2 hours after 75 g oral glucose. It is glycosuria, hyperlipidaemia, negative nitrogen balance and sometimes is ketonaemia. The widespread pathological change is the thickening of the capillary is basement the membrane, increase in vessel wall matrix and cellular proliferation resulting in vascular complications like lumen narrowing.[3]

1. Type-1 (IDDM) Insulin Dependent Diabetes Mellitus
2. Type-2 (NIDDM) Non-Insulin Dependent Diabetes Mellitus

Jamun is also called Syzygium Jambolanum and Eugenia cumini. Other name is also known as Black Plum, Indian Blackberry etc and that taste are sweetish sour. Its belong to the family Myrtles “Myrtaceae”. Syzygium fruit is the oval in shape. The changes color to the crimson black with ripening Jamun have different activity like as antioxidant, anti-inflammatory, neuropsycho-pharmacological, anti-microbial, anti-bacterial, anti-HIV, antileishmanial and antifungal etc.[4]

Jamun fruit in a bunch.

Plant Profile

The seasonal perishable jamun berry, also known as java plum (in English) and Syzygium cumini (linn) Skeels in botany, is what gives jamun, also known as Syzygium cumini (linn) Skeeels, its name jambul. It is a member of the Myrtle (Myrtaceae)family. The jamun tree is an evergreen that can reach a height of 30 meters. And measures about 3.6 meters in width.

Jamun trees are found in India's topical and subtropical regions can be found in subcontinent of India. The jamun tree is widespread.[5]

SYNONYMS & VERNACULAR NAMES

Hindi Name: Jamun, Jambul

English Name: Java Plum (sometimes Malabar plum), Indian blackberry

Sanskrit Name: Jamboo, Raj jamboo

Bengali Name: Kalajaam

Unani Name: Jaman

Other Name: Jamblang, Jambolan, Duhat, Jambolan Plum, Kavika, Mesegerak

Botanical Synonyms: Eugenia cumini & Eugenia jambolana

Latin: Syzygium cumini (linn) Skeeels

Ayurvedic name: Jambu, Mahaphala[6]

BOTANTICAL CLASSIFICATION

Kingdom- plantae

Sub-Kingdom- Viridiplantae

Division- Tracheophyta (Tracheophytes or Vascular Plants)

Sub-devision- Spermatophyta (Spermatophytes or Seed Plants)

Class- Magnoliopsida

Superorder- Rosanae

Order- Myrtales

Family- Myrtaceae

Genus- Syzygium

Species- Syzygium cumini

The Jamun plant was first planted on the Indian subcontinent, and it is now grown in a variety of south Asian countries and territories, including India, Bangladesh, Nepal, Pakistan, Burma, and Indonesia.

Sri Lanka, too. The jamun trees is revered by Buddhists in Southern Asia, and because it is devoted to Lord Krishna, it is frequently grown close to Hindu temples.[7]

MEDICINAL PARTS

- Anti-diabetes
- Seed
- Stem bark
- Kernel
- Leaves
- Fruit pulp

CHEMICAL CONSTITUENT

- Glucoside
- Jambosine
- Ellagic acid
- Anthocyanins
- Glycoside jambolana or antimellin

MEDICINAL PROPERTIES

- Anti-inflammatory
- Liver stimulant
- Digestive stimulant
- Mild astringent

PHARMACOLOGICAL ACTION

All parts of the jamun tree are generally astringent, anti-diarrheal, anti-microbial, anti-inflammatory.[8]

THERAPEUTIC SIGN

- Splenopathy
- Poor appetite
- Indigestion
- Cancer
- Liver disorder
- Chronic colitis
- Rectal Bleeding (Bleeding diarrhea)
- Pharyngitis
- Urethrorrhea

DOSAGES AND ADMINISTRATION

- Juice-10-20mL
- Powder-3-6g

MEDICINAL USES OF JAMUN

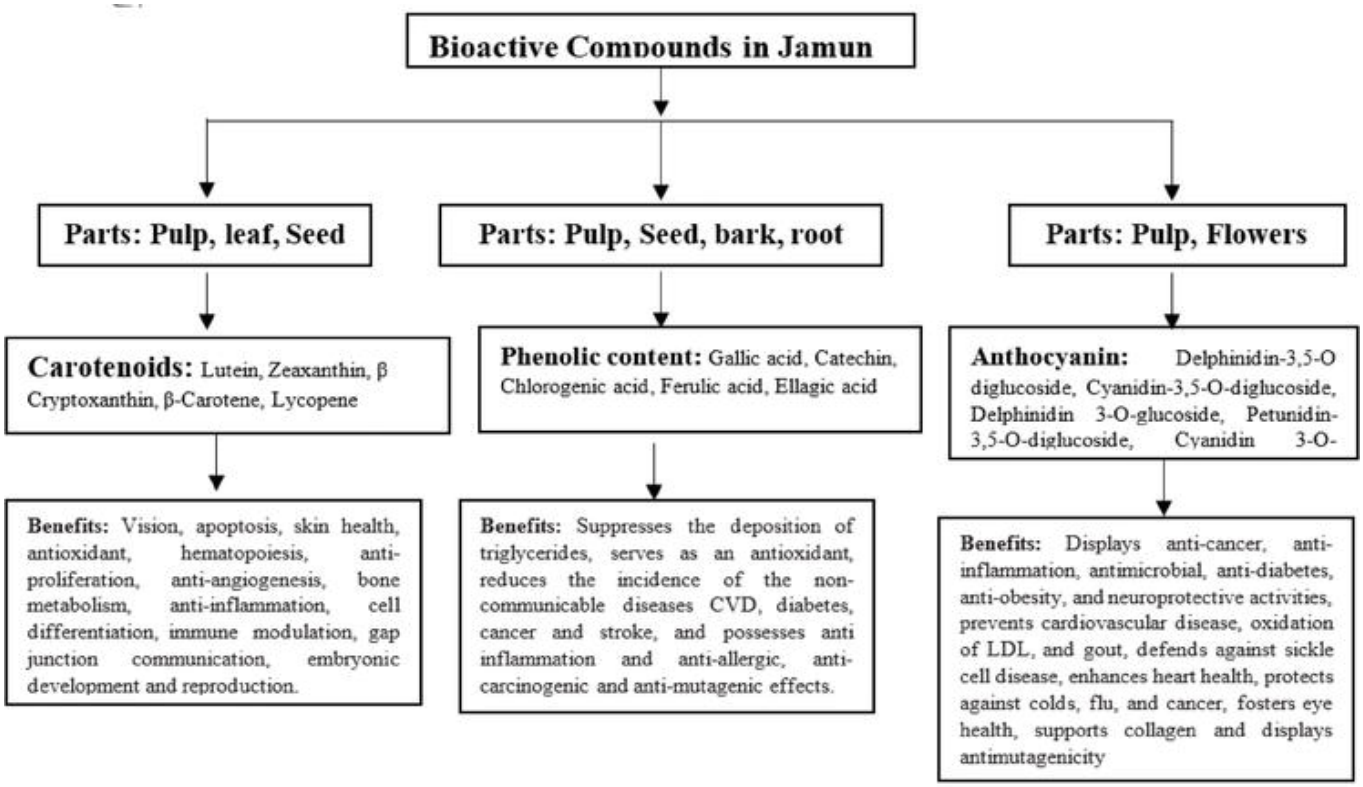
- Stomach pain
- Arthritis
- Heart issues
- Flatulence
- Asthma
- Dysentery
- Stomach spasm

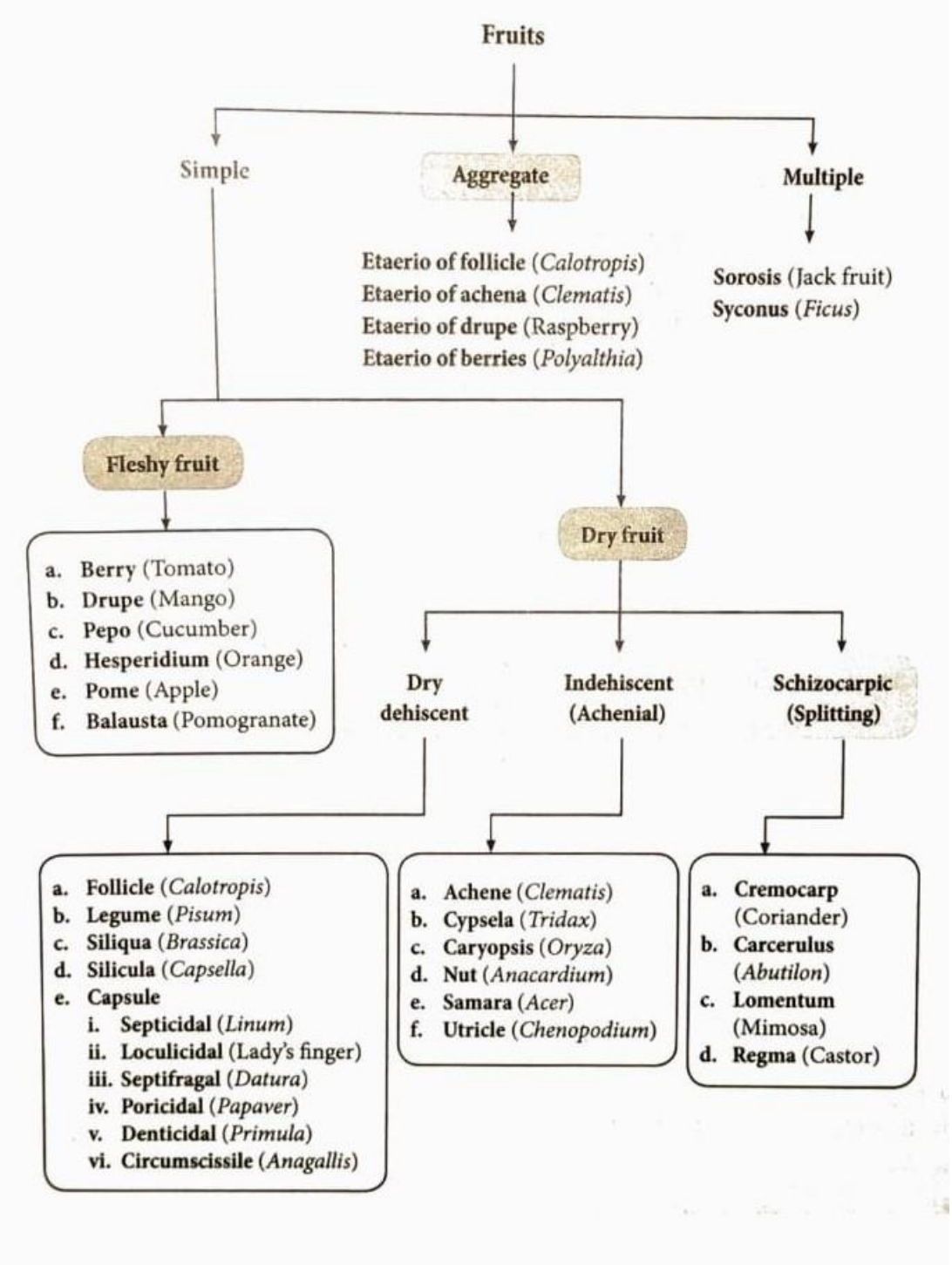
NUTRITION IN JAMUN

The place and environment where jamun fruit is grown have a significant impact on the composition of the fruit. In a certain area in contrast to vitamin composition, mineral composition is greatly influenced by climate.

The mineral of present in jamun pulp seeds includes example:

- Sodium
- Potassium
- Magnesium


Bioactive Compounds in Jamun
Parts: Pulp, leaf, Seed
Parts: Pulp, Seed, bark, root
Parts: Pulp, Flowers
Carotenoids: Lutein, Zeaxanthin, β Cryptoxanthin, β-Carotene, Lycopene
Phenolic content: Gallic acid, Catechin, Chlorogenic acid, Ferulic acid, Ellagic acid
Anthocyanin: Delphinidin-3,5-O diglucoside, Cyanidin-3,5-O-diglucoside, Delphinidin 3-O-glucoside, Petunidin-3,5-O-diglucoside, Cyanidin 3-O-
Benefits: Vision, apoptosis, skin health, antioxidant, hematopoiesis, anti-proliferation, anti-angiogenesis, bone metabolism, anti-inflammation, cell differentiation, immune modulation, gap junction communication, embryonic development and reproduction.
Benefits: Suppresses the deposition of triglycerides, serves as an antioxidant, reduces the incidence of the non-communicable diseases CVD, diabetes, cancer and stroke, and possesses anti inflammation and anti-allergic, anti-carcinogenic and anti-mutagenic effects.
Benefits: Displays anti-cancer, anti-inflammation, antimicrobial, anti-diabetes, anti-obesity, and neuroprotective activities, prevents cardiovascular disease, oxidation of LDL, and gout, defends against sickle cell disease, enhances heart health, protects against colds, flu, and cancer, fosters eye health, supports collagen and displays antimutagenicity



CHEMOPREVENTIVE EFFECTS

The pharmaceutical industry faces significant obstacles in increasing the effectiveness of their R&D, despite the strict application of well-established drug-likeness guidelines in drug development. The number of new drugs approved by the FDA for every $1 billion US spent on R&D alone is known as R&D efficiency. Since then, the price of finding and creating a drug has increased, from US$ 800 million in 2001 to an estimated US$ 3 billion today. The

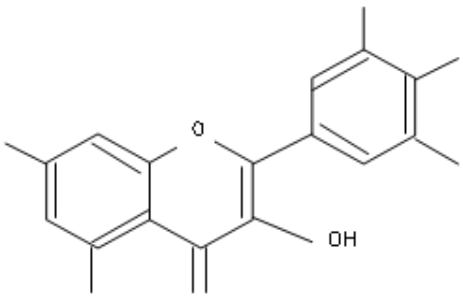
total cost of failure is included in the cost of drug development, so the estimated cost is an average estimate for the introduction of a new drug into clinical practice [17].[9] It is concerning that fewer new drugs are being approved for every $1 billion spent on research and development. Consequently, Scannell et al. methodically It has been reported recently [71] that jamun has cancer chemopreventive properties in Swiss albino mice with DMBA-induced croton oil promoted two-stage skin carcinogenesis. When comparing the experimental group (carcinogen alone) to the perinitiation (i.e., 7 days before and 7 days after the application of DMBA) or post-initiation (i.e., from the day of start of croton oil treatment and continued till the end of the experiment) phases, feeding 125 mg/kg body weight/animal/day of the extract reduced the cumulative numbers of papillomas, the tumor incidence, and increased the average latency period [71].[10] The cumulative number of gastric carcinomas as well as the tumor burden and incidence were all decreased by jamun. Additionally, reports indicate that anthocyanins, gallic acid, ellagic acid, and flavonoids Furthermore, current findings indicate that ellagitannin, a component of jamun and its colonic.[11]

Phytochemicals of Jamun with reported chemo preventive effects

Sr. No	**Agent**	**Chemopreventive effects and the mechanisms operating**
1	Oleanolic acid	1) Inhibits tumor promotion in mouse skin [72]; 2) Inhibits azoxymethane (AOM)-induced colonic aberrant crypt foci and multi-crypt aberrant crypt/foci in a dose dependent manner
2	Ellagic acid	1) Inhibitor of benzo[a]pyrene-induced pulmonary adenoma and 7,12-dimethyl benz[a]anthracene-induced skin tumorigenesis in Swiss mice [75]; 2) Topical application [76] as well as oral feeding of ellagic acid [76] rendered protection against 3-methylcholanthrene-induced skin tumorigenesis in mice and decreased tumor incidence, number of tumors, tumors per mouse and tumors per tumor bearing animal [76,77]; 3) Topical application of ellagic acid and oral before a tumor-initiating by B[a]P 7,8-diol-9,10-epoxide-2 and promotion with 12-O-tetradecanoylphorbol-13-acetate inhibited the number of skin tumors per mouse [78]; 4) Ellagic acid applied topically to female CF-1 mice the two-step initiation-promotion protocol [79]; 5) Topical application of ellagic acid

		simultaneously, 12-dimethyl-benz[a]anthracene-induced skin tumors in mice [80]; 6) The levels of aryl hydrocarbon hydroxylase (AHH) activity in skin and liver and the extent of 3H-BP-binding to skin, liver and lung DNA were decreased [76];7) binding to DNA in cultured BALB/C mouse keratinocytes [81]; 8) Inhibited the epidermal microsomal aryl hydrocarbon hydroxylase (AHH) activity and of benzo[a]pyrene (BP)-binding to both calf thymus DNA *in vitro* and to epidermal DNA *in vivo* [82].
3	Gallic acid	1) Inhibits the TPA-induced inductions of epidermal ornithine decarboxylase activity, hydroperoxide production and DNA synthesis, and also inhibits the promotion of skin papillomas and carcinomas in the two-step initiationpromotion protocol [79]; 2) Administering (0.3% to 1%) for twenty consecutive weeks from four weeks of age to the male TRAMP
4	Quercetin	It reduced the polyamine levels and the proliferation [84]; 2) Prevents N-nitroso diethylamine induced lung tumorigenesis in mice [85]; 3) Prevents 20-methyl cholanthrene-induced cervical neoplasia in virgin Swiss albino mice by increasing the antioxidant enzymes, decreasing DNA damage and he lipid peroxidation [86,87]; 4) Decreases DMBA-induced DNA damage [88]; 5) In a bioengineered human gingival epithelial tissue, quercetin was observed to
5	Myricetin	1) Inhibits epidermal growth factor (EGF)-activated cell transformation of JB6 cells by modulating DNA binding and transcriptional activity of STAT3 [91,92], and mitogen-activated protein kinase (MEK) [93] and inhibitor of of neoplastic cell transformation and MEK1 [94]; 2) Prevents TPA-induced transformation, PKC activation, and c-jun expression in mouse fibroblast cells [95]; 3) Suppresses UVB-induced skin cancer by targeting Fyn in JB6 cells [96]. Inhibits Akt survival signaling and induces Bad-mediated apoptosis in immortalized human keratinocytes (HaCaT cells) [97]; 4) Inhibits matrix metalloproteinase 2 protein expression and enzyme activity in colorectal carcinoma cells [98] and also down-regulates phorbol ester-induced cyclooxygenase-2 expression in mouse epidermal cells by blocking activation of nuclear factor kappa B [94]; 5) Inhibits polycyclic aromatic hydrocarbon-DNA adduct formation in epidermis and lungs of SENCAR mice [99].

6	Kaempferol	1) Have phosphatidylinositol 3-kinase inhibitory effects and prevent neoplastic transformation [100].
7	Betulinic acid	1) Topical application of betulinic acid inhibited the TPA-induced inflammation and decreased the levels of ornithine decarboxylase [101]; 2) Markedly inhibited the 7, 12-dimethylbenz[a]anthracene and TPA promoted skin tumor formation in mice [101].
8	β- sitosterol	1) Topical application of *β*-sitosterol inhibited the TPA-induced inflammation [101]; 2) Induces dose-dependent growth inhibition, induces apoptosis, suppresses the expression of *β*-catenin and PCNA antigens in human colon cancer cells (COLO 320 DM cells) [102]; 3) *β*-sitosterol supplementation reduced the number of aberrant crypt and crypt multiplicity in DMH-initiated rats in a dose-dependent manner with no toxic effects [102].
9	Delphinidin	1) Suppresses 12-O-tetradecanoylphorbol-13-acetate (TPA)-induced cell transformation and activator protein-1 transactivation in the JB6 cells by blocking the phosphorylation of protein kinases in the extracellular signalregulated protein kinase (ERK) and the c-Jun N-terminal kinase (JNK) signaling pathways [103]; 2) Possess chemopreventive effects against prostate carcinogenesis in both *in vitro* and vivo study models [104]; 3) Suppresses ultraviolet B-induced cyclooxygenases-2 expression through inhibition of MAPKK4 and PI-3 kinase [105].



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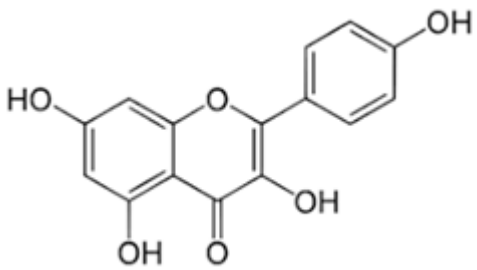

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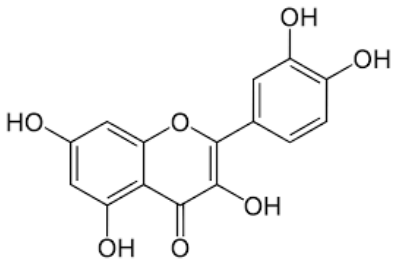

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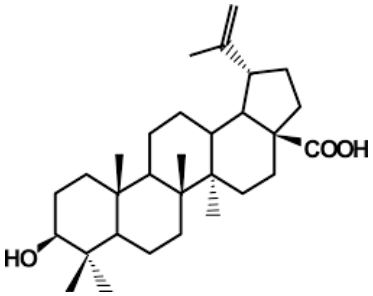

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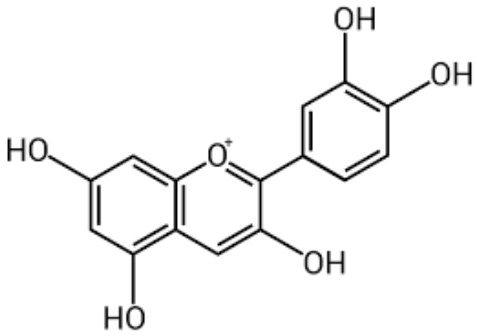

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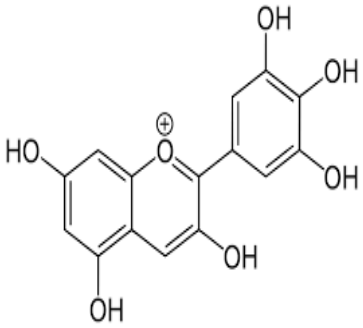

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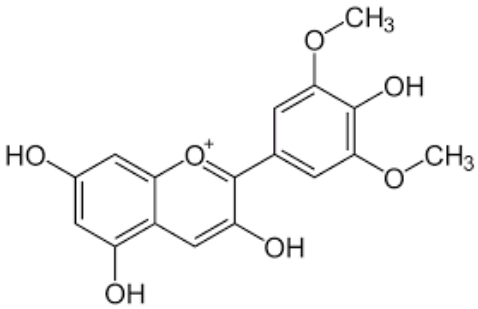

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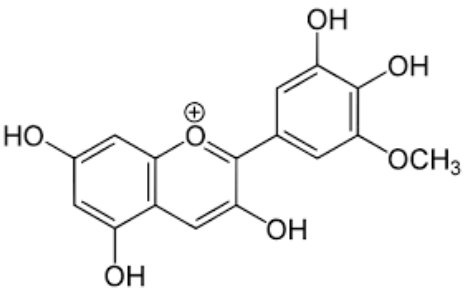

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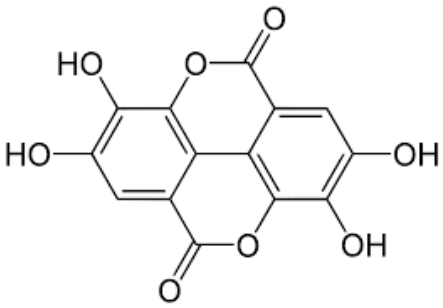
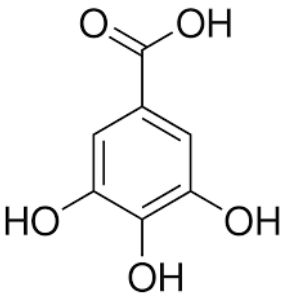


(i)



(j)

The reported structures of phytochemicals in jamun have potential applications in cancer treatment. (a) Myricetin; (b) Kaem- pherol; (c) Quercetin; (d) Betulinic; (e) Anthocyanin; (f) Delphinidin; (g) Malvidian; (h) Petunidin; (i) Ellagic Acid; (j) Gallic Acid.

5.2. Impact of Radioprotection

The unfavorable effects force the doctors to stop treating patients or lower their dosage because the normal cells suffer irreversible damage. An agent that can provide a therapeutic distinction between cancerous and normal cells will be extremely helpful in these circumstances. Research has indicated that administering the hydroalcoholic extract of Jamun seed and the dichloromethane extract of Jamun leaf intraperitoneally can have a protective effect against radiation [107].[12] Chemical substances known as "radioprotectors" that may specifically shield normal cells from the harmful effects of radiation can produce therapeutic differences. Following [108]'s findings that the natural amino acid cysteine shielded mice from radiation-induced illness and death, numerous substances with varying pharmacological.[13] The highest dose of 80 mg/kg had the best results, but only when given intraperitoneally; in the oral route, only 22% of the animals survived, and in the radiation-only cohorts, none at all. It was also found that giving mice an intraperitoneal injection of the organic extract (dichloromethane-methane) of leaves (5, 10, 20, 30, 40, 50, 60, and 80 mg/kg b. wt.) five days prior to radiation treatment was effective in reducing radiation-related illness and mortality.[14] When compared to the concurrent irradiation control, histopathological investigations revealed that treating jamun leaves before radiation increased the height of the villus, the number of crypts, and the gob- let and dead cells. When compared to irradiation alone, the animals that received Jamun pretreatment recovered and regenerated more quickly. Additionally, jamun extracts shield DNA from radiation-induced damage (more on this later). Additionally, jamun contains the phytochemicals ellagic acid, gallic acid, quercetin, and oleanolic acid, which have radioprotective properties. [15]

5.3 Jamun's Antineoplastic Properties

For over fifty years, chemotherapy has played a significant role in the treatment of cancer and is a necessary treatment when metastasis has developed. Chemotherapy is used either alone or in conjunction with radiation and surgery, depending on the clinical stage and patient compliance [118]. Research indicates that approximately 47% of antineoplastic medications currently in use come from natural sources [119][16]. Regarding Jamun, numerous compounds

have positive effects. Whole Jamun extract has been demonstrated to have cytotoxic effects on cultured human cervical cancer cells, specifically HeLa (HPV-18 positive) and SiHa (HPV-16 positive), according to in vitro studies [177]. The extract resulted in concentration-dependent cell death, which was more noticeable in HeLa cells as opposed to SiHa cells [177].[17] Furthermore, when the pulp was cultured at an 80% concentration of the extracts, both the crude and the methanolic extracts of the pulp increased apoptosis in a time-dependent manner. In both cell lines, the crude extract was found to perform better than the methanolic extract [177]. Recent research by [57] has demonstrated that the standardized Jamun fruit extract has pro-apoptotic and antiproliferative properties in the cells, with broad therapeutic implications.[18]

Jamun phytochemicals with documented radioprotective properties

Sr. No	***Agent***	**Phytochemicals' radioprotective effects and the mechanisms operating**
1	Oleanolic acid	Reduces the development of ascitic tumors and speeds up the hematopoietic system's recovery in radiation-exposed mice.
2	Quercetin	1) Reduced DNA damage in yeast cells to protect them from γ-radiation damage Effectively shielded human peripheral blood lymphocytes from γ-radiation-induced DNA damage in vitro and plasmid DNA. The protective mechanisms were mediated by the antioxidant and inhibition of lipid peroxides Radiation-induced DNA damage in mouse WBC was prevented by intraperitoneal administration of quercetin 100 mg·kg/kg for three consecutive days prior to and/or following irradiation. When querecetin was given prior to radiation, there were noticeable effects
3	Gallic acid	Prevents lipid peroxidation and DNA damage brought on by radiation in both in vitro and in vivo settings.[19]

Phytochemicals in Jamun with reported antineoplastic activities.

Sr. No	*Agent*	Antineoplastic activity and the mechanisms operating
1	Oleanolic acid	Causes the human colon carcinoma cell line HCT15 to die in a dose- and time-dependent manner. suppresses growth and stops cells in the G0/G1 phase [120];2) activates caspase to induce apoptosis in human leukemia cells HL60 [121];3) specifically suppresses the growth of ras oncogene-transformed R6 cells [122];4) induces apoptosis in human liver cancer HepG2, Hep3B, Huh7, and HA22T cell lines [123]; and5) suppresses the development of ascitic tumors in mice.[20]
2	Quercetin	1) Induces chromatin condensation and dose-dependent cell death in colon cancer cells (Caco-2 and HT-29) [124]; 2) Enhances the protective effect of a non-toxic cisplatin dose, preventing lung colonization of B16-BL6 colonies in a dose-dependent manner [125]; 3) Blocks the growth of the moderately aggressive and highly aggressive PC-3 prostate cancer cell lines, but not the poorly aggressive LNCaP prostate cancer cell line or the normal fibroblast cell line BG-9 [126]; 4) Blocks the expression of certain oncogenes and genes that regulate the G1, S, G2, and M phases of the cell cycle. Moreover, it increased the expression of multiple tumor suppressor genes. 5) In human prostate cancer cells, downregulates gelatinases A and B (matrix metalloproteinases 2 and 9).[21]
3	Kaempferol	inhibits the growth of human glioma cells and causes them to die through caspase-dependent mechanisms that include down-regulating XIAP and ERK and Akt regulation [128]; 2) mediates p53-dependent growth inhibition and causes apoptosis in the human HCT116 colon cancer cell line by influencing Bcl-2 family proteins, PUMA, and inducing ATM and H2AX phosphorylation [129]; 3) induces apoptosis in a variety of oral cancer cell lines (SCC-1483, SCC-25, and SCC-QLL1) via the caspase-3-dependent pathway [130]; 4) causes apoptosis in human osteosarcoma U-2 OS cells through endoplasmic reticulum stress and mitochondrial pathway[22]
4	Myricetin	1) Induce apoptosis in the following cell lines: HT-29 [132], Caco-2 cells [132], MCF7 (Rodgers and Grant, 1998), Jurkat T cells [133], OE33 [134], and HepG-2 [135]; 2) Inhibits proliferation, causes G2/M and S phase arrest, and induces mitochondria-mediated apoptosis by activating caspase 3, 9 of HepG-2 [135]; 3) Possess cytotoxic effects against the OE33 (human oesophageal adenocarcinoma cell line), causes G2/M cell cycle arrest by up-regulating GADD45beta and 14-3-3sigma and

		down-regulating cyclin B1; and p53-independent mitochondrial-mediated apoptosis through up-regulating PIG3 and cleaving caspase-9 and 3 [134]; 4) Possess moderate proteasome inhibitory effects and induce apoptosis in the human leukemia cells Jurkat T cells[23]
5	Gallic acid	causes human prostate LNCaP cells to undergo apoptosis 2) Through mitochondria-mediated apoptosis and the production of reactive oxygen species, induces cytotoxic effects on DU145 prostate cancer cells. 3) By activating Chk1 and Chk2 and inhibiting the activities of Cd25C and Cd2, it prevents the growth of DU145 cells during the G2/M phases. 4) The human prostate carcinoma DU145 cells undergo apoptosis and ATM-Chk2 activation-induced phosphorylation of cd25A is inactivated. In vitro and in nude mice, exhibit anti-proliferative, pro-apoptotic, and anti-tumorigenic properties against human prostate cells DU145 and 22Rv1. 6) Works in concert with doxorubicin to inhibit the proliferation of DU145 cells 7) Causes human melanoma cells to undergo apoptosis via both caspase-dependent and -independent mechanisms. 8) Has anticancer properties in vitro against human prostate cancer cells
6	Betulinic acid	At equal concentrations, it is relatively safe for normal cells and tissue while being effective against a range of cancer types [141]; 2) In vitro, it induces a strong effect on growth inhibition, G2/M cell cycle arrest, and apoptosis in human gastric adenocarcinoma AGS cells, possibly through the down-regulation of Hiwi and its downstream target Cyclin B1
7	1,8-Cineole	Causes human stomach cancer KATO III cells to not undergo apoptosis, but human leukemia Molt 4B and HL-60 cells do.

CONCLUSION

Traditionally, jambolan has been used to treat a wide range of illnesses, including diabetes and its complications. The majority of studies on diabetes pharmacology have focused on seeds; however, more research is needed to fully understand the pharmacological potential of the plant's other parts. Studies on Jamun's antitumor properties indicate that it acts specifically on breast cancer cells. It is important to look into the potential chemopreventive effects of jamun and its phytochemicals in other carcinogen models, such as those involving chemicals, radiation, and viruses. There are also insufficient mechanistic studies that explain the radioprotective and chemopreventive effects; these studies must be conducted in greater detail. These details allow this review to emphasize the function of jambolan in a range of treatments.[26]

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