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Jamun (*Syzygium cumini* (L.)): A Review of Its Food and Medicinal Uses

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ABSTRACT

Eugenia jambolana Lam., commonly known as black plum or "jamun" is an important medicinal plant in various traditional systems of medicine. It is effective in the treatment of diabetes mellitus, inflammation, ulcers and diarrhea and preclinical studies have also shown it to possess chemopreventive, radioprotective and antineoplastic properties. The plant is rich in compounds containing anthocyanins, glucoside, ellagic acid, isoquercetin, kaemferol and myrecetin. The seeds are claimed to contain alkaloid, jambosine, and glycoside jambolin or antimellin, which halts the diastatic conversion of starch into sugar. The present review has been primed to describe the existing data on the information on traditional and medicinal use.

Keywords: Jamun-*Syzygium cumini*; Chemopreventive; Radioprotective; Antineoplastic Activities

1. Introduction

Syzygium cumini (Family Myrtaceae) is also known as *Syzygium jambolanum* and *Eugenia cumini*. Other common names are Jambul, Black Plum, Java Plum, Indian Blackberry, Jamblang, Jamun etc. Today these trees are found growing throughout the Asian subcontinent, Eastern Africa, South America, Madagascar and have also naturalized to Florida and Hawaii in the United States of America [1]. The tree fruits once in a year and the berries are sweetish sour to taste. The ripe fruits are used for health drinks, making preserves, squashes, jellies and wine [1]. In association to its dietary use, all parts of the tree and, importantly the seeds are used to treat a range of ailments, the most important being diabetes mellitus [2]. Different parts of the jambolan were also reported for its antioxidant, anti-inflammatory, neuropsycho-pharmacological, anti-microbial, anti-bacterial, anti-HIV, antileishmanial and antifugal, nitric oxide scavenging, free radical scavenging, anti-diarrheal, antifertility, anorexigenic, gastroprotective and anti-ulcerogenic and redioprotective activities [2]. (**Figure 1**) shows the Jamun fruit.

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2. Composition of Fruit

Analyses of the fruit in the Philippines were reported in 1924 as follows: Waste, 25%; edible portion: water, 80.80%; ash, 0.70; protein, 0.81; sugar, 12.70 (fructose and glucose; no sucrose); acidity (as sulphuric), 0.63%; (as malic) 0.88% [3]. The following composition per 100 grams of edible portion was reported for fruits freshly picked at the Lancetilla Experimental Garden, Honduras, in 1948: Moisture, 85.8 gm; ether extract, 0.15 gm; crude fiber, 0.3 gm; nitrogen, 0.129 gm; ash, 0.32 gm; calcium, 8.3 mg; phosphorus, 16.2 mg; iron, 1.62 mg; carotene, 0.004 mg; thiamine, 0.008 mg; riboflavin, 0.009 mg; niacin, 0.290 mg; total ascorbic acid, 5.7 mg [4]. Virmani gives the following analysis: specific gravity, 1.0184; total acidity (as acetic acid), 5.33 per 100 cc; volatile acidity (as acetic acid), 5.072 per 100 cc; fixed acidity, 0.275% as citric; total solids, 4.12 per 100 cc; ash, 0.42; alkalinity of ash, 32.5 (N/10 alkali); nitrogen, 0.66131; total sugars, 0.995; reducing sugars, 0.995; non-volatile reducing sugars, 0.995; alcohol, 0.159% by weight; oxidation value ($KMnO_4$, 186.4); iodine value, 183.7; ester value, 40.42. Other reported constituents of the seeds are: protein (6.3 to 8.5%), fat (1.18%), crude fiber (16.9%),

Figure 1. Jamun fruit in a bunch.

ash (21.72%), calcium (0.41%), phosphorus (0.17%), fatty acids (palmitic, stearic, oleic and linoleic), starch (41%), dextrin (6.1%), a trace of phytosterol, and 6 to 19% tannin [5]. The fruits are avidly eaten by birds and four footed animals (jackals and civets in India). In Australia, they are a favorite food of the large bat called "flying fox". Analyses of the leaves show: crude protein (9.1%), fat (4.3%), crude fiber (17.0%), ash (6.0%), calcium (1.3%), phosphorus (0.19%) [6]. It consists mainly of mono- or sesqui-terpene hydrocarbons which are "very common in essential oils." Constituents of *Syzgium cumini* seeds are fatty oils (30 g/kg), including lauric (2.8%), myristic (31.7%), palmitic (4.7%), stearic (6.5%), oleic (32.2%), linoleic (16.1%), malvalic (1.2%), sterculic (1.8%) and vernolic acid (3%) and phytosterols such as β-sitosterol. Further constituents are tannins (6%), predominantly corilagin, ellagitannins, ellagic acid, galloyl-galactoside and gallic acid [7]. The leaf oil consists of 16.91% octadecane, 9.98% nonacosane, 9.38% triacontane, 7.38% octacosane, 4.86% Heptacosane, 4.25% hexadecanoic acid and 4.02% eicosane. The seed oil consists of 33.2% 1-chlorooctadecane, 9.24% tetratetracontane, 8.02% decahydro-8a-ethyl-1,1, 4a,6-tetramethylnapahthalene, 5.29% 4-(2-2-dimethyl-6-6-methylenecyclohexyl) butanol, 5.15% Octadecane, 3.97% octacosane, 1.72% heptacosane and 1.71% eicosane. [8]. Java Plum consist of Energy 251 kJ (60 kcal), Carbohydrates 15.56 g, fat 0.23 g, Protein 0.72 g, water 83.13 g, Vitamin A 3IU, Thiamine (vit B1) 0.006 mg (1%), Riboflavin (vit. B_2) 0.012 mg (1%), 0.260 mg (2%) Niacin (vit. B_3), 0.160 mg (3%) Pantothenic acid (B_5), 0.038 mg (3%) Vitamin $B_6$0.038 mg (3%), 14.3 mg (17%) Vitamin C, 19 mg (2%) Calcium, 0.19 mg (1%) Iron, 15 mg (4%) Magnesium, 17 mg (2%) Phosphorus, 79 mg (2%) Potassium, 14 mg (1%) Sodium [9]. The Fruit Contain 83.70 - 85.80 g moisture, 0.70 - 0.13 g protein, 0.15 - 0.30 g fat, 0.30 - 0.90 g crude fibre, 14.00 g carbohydrate, 0.32 - 0.40 h ash, 8.30 - 15.00 mg calcium, 35.00 mg magnesium, 15.00 - 16.20 mg phosphorus, 1.20 - 1.62 mg iron, 26.20 mg sodium, 55.00 mg potassium, 0.23 mg copper, 13.00 mg sulfur, 8.00 mh chlorine, 8. I.U vitamin A, 0.01 - 0.03 mg thiamine, 0.009 - 0.01 mg riboflavin, 0.20 - 0.29 mg niacin, 5.70 - 18.00 mg ascorbic acid, 7.00 mg chlorine and 3.00 mcg folic acid per 100 g of edible portion [10].

3. Food Uses

Good quality jambolan juice is excellent for sherbet [11, 12], syrup and "squash". In India the latter is a bottled drink prepared by cooking the crushed fruits for 5 to 10 minutes at 140˚F, pressing out the juice, combining it with sugar and water and adding citric acid and sodium benzoate as a preservative [13]. Jambolans of good size and quality, having a sweet or sub acid flavor and a minimum of astringency, are enjoyable raw and may be made into tarts [14], sauces and jam. Astringent fruits are improved in palatability by soaking them in salt water [14] or pricking them, rubbing them with a little salt, and letting them stand for an hour [15]. All but decidedly in ferior fruits can be utilized for juice which is often comparable to grape juice [16]. When extracting juice from cooked jambolans, it is recommended that it be allowed to drain out without squeezing the fruit and it will thus be less as tringent. The white-fleshed jambolan has adequate pectin and makes a very stiff jelly unless cooking is brief [17]. The more common purplefleshed yields richly colored jelly [18] but is deficient in pectin and requires the addition of a commercial jelling agent or must be combined with pectin-rich fruits such as unripe or sour guavas, or ketembillas [18]. In Goa and the Philippines [19], jambolans are an important source of wine, resembling Port [20]. Brandy and a distilled liquor called "jambava" have also been made from the fermented fruit. Jambolan vinegar, extensively made throughout India, is an attractive, clear purple, with a pleasant aroma and mild flavor.

4. Uses in Traditional Medicine

Traditionally the jambul fruits, leaves, seeds, and bark are all used in ayurvedic medicine. The bark contains tannins and carbohydrates, accounting for its long-term use as an astringent to combat ailments like dysentery [21]. A glycoside in the seed, jamboline, is considered to have antidiabetic properties [22]. Older French research shows that the seeds have a significant hypoglycemic effect in diabetic rabbits [23]. The seeds have also shown anti-inflammatory effects in rats and antioxidant proper-

ties in diabetic rat [24]. Older reports from Indian medical journals suggest jambul seed and bark can be beneficial in humans with diabetes [25]. Jamun fruit seeds and pulp have been reported to serve various purposes in diabetic patients, such as lowering blood glucose levels and delaying diabetic complications including neuropathy and cataracts [2,26]. Jamun is most often recognized as an adjuvant therapy in type-2 diabetes. This has been traced not only to its anthocyanin-rich, dark-purple fleshy pulp, but also to its seeds, which have been most studied for their antidiabetic principles. Jamun seeds are reported to be a rich source of ellagitannins (ETs), including corilagin, 3,6-hexa hydroxyl diphenoyl glucose and its isomer 4,6-hexahydroxy diphenoyl glucose, 1-galloylglucose, 3-galloylglucose, gallic acid, and ellagic acid (EA) [26]. This marker compound has anti-diabetic activity. When alloxan induced diabetic rats were fed with Jamun seed extract, the blood glucose, blood urea, serum cholesterol and serum triglyceride levels were found to decrease significantly [27]. Jamun fruit reduces the sugar in the blood and is very good in the control of diabetes. Its seeds contain Glucoside, Jamboline and Ellagic acid, which are reported to have the ability to check the conversion of starch into sugar in case of excess production of glucose [27]. All parts of the jambolan can be used medicinally and it has a long tradition in alternative medicine. The plant has been viewed as an antidiabetic plant since it became commercially available several decades ago.

From all over the word, the fruits have been used for a wide variety of ailments, including cough, diabetes, dysentery, inflammation and ringworm [28]. It is also an ancient medicinal plant with an illustrious medical history and has been the subject of classical reviews for over 100 years. It is widely distributed throughout India and Ayurvedic medicine (Indian folk medicine) mentions its use for the treatment of diabetic mellitus. Various traditional practitioners in India use the different parts of the plant in the treatment of diabetes, blisters in mouth, cancer, colic, and diarrhea, digestive complaints, dysentery, piles, pimples and stomachache [29]. During last four decades, numerous folk medicinal reports on the antidiabetic effects of this plant have been cited in the literature. In Union medicine various parts of Jambolan acts as liver tonic, enrich blood, strengthen teeth and gums and form Good lotion for removing ringworm infection of the head [30].

E. jambolana leaf extract showed hypoglycemic action in diabetic rats [30]. The seed powder of *E. jambolana* is reported to have hypoglycemic action in streptozotocin-diabetic rats [31,32]. Its effect may be persistent, as in one study, homeostasis was maintained in the rats for two weeks after the cessation of treatment [32]. In alloxan-diabetic rabbits the water extract of *E. jambolana* fruit pulp was more effective than the ethanol extract at reducing fasting blood glucose and improving blood glucose levels in the glucose tolerance test. *E. jambolana* also increased blood insulin levels in both diabetic and severely diabetic rabbits [33,34]. The inhibition of insulinase activity from liver and kidney by extract of *Eugenia jambolana* also has been reported, which points out to its extra-pancreatic mechanism [34]. Another study also found that *E. jambolana* seed extract reduced blood glucose, glycosylated hemoglobin, and increased plasma insulin [35]. *E. jambolana* fruit combined with bitter melon decreased insulin levels that were raised in diabetic rats fed a fructose diet [36].

Jamun is most often recognized as an adjuvant therapy in type-2 diabetes. This has been traced not only to its anthocyanin-rich, dark-purple fleshy pulp, but also to its seeds, which have been most studied for their antidiabetic principles. Other reports from Indian medical journals suggest jambul seed and bark can be beneficial in humans with diabetes [37]. When alloxan induced diabetic rats were fed with Jamun seed extract, the blood glucose, blood urea, serum cholesterol and serum triglyceride levels were found to decrease significantly [27]. Jamun fruit reduces the sugar in the blood and is very good in the control of diabetes.

Ayurvedic texts suggest that 1 - 3 g of seed powder per day is an average dose 44 additionally, Juice of ripe fruits in the amount of 0.5 - 2 tsp (2.5 - 10 ml) at least three times daily have been recommended for the treatment of diabetes. Administration of 100 and 200 mg/kg body weight of aqueous extract of *Syzygium cumini pulp* significantly decreased the blood glucose level in the experimental rats suggesting that it has hypoglycemic properties. The decreased body weight in diabetic rats is due to excessive breakdown of tissue proteins. Treatment with *Syzygium cumini* improved body weight significantly in a dose dependent manner, indicating prevention of muscle wasting due to hyperglycemic condition.

5. Medicinal Properties

The jambolan has received far more recognition in folk medicine and in the pharmaceutical trade than in any other field. Medicinally, the fruit is stated to be astringent, stomachic, carminative, antiscorbutic and diuretic [37]. Additionally, a fruit extract showed antimicrobial and cytotoxic activities and may potentially be used on topical antimicrobial products. In comparison to other non-traditional fruits jambolao showed considerable high antioxidant activity, which can constituent such as anthocyanins, tannins and flavonols [38]. The anthocyanin composition was characterised by the presence of 3,5-diglucosides of five out of six aglycones commonly found in foods [39]. Fruits contain many different kinds of

anti-oxidant compounds, including flavonoids, phenolics, carotenoids and vitamins, which are all considered beneficial to human health, for decreasing the risk of degenerative diseases by reduction of oxidative stress, and for the inhibition of macromolecular oxidation [40]. There is a very high anthocyanin content in *S. cumini* fruits which attributes to its antioxidant and free radical scavenging activity. These pigments can be a good source of natural food colourants for the food processing industries [41].

Fruit bark decoction for antiplasmodial activity was performed, leading to the isolation of three known ellagic acid derivatives (ellagic acid, ellagic acid 4-O-alpha-L-2"-acetylrhamnopyranoside, 3-O-methylellagic acid 3'-O-alpha-L-rhamnopyranoside), as well as the new derivative 3-O-methylellagic acid 3'-O-beta-D-glucopyranoside [42]. 3-hydroxy androstane [16,17-C] (6'methyl, 2'-1-hydroxyl-isopropene-1-yl) 4,5,6 H pyran present in *Syzygium cumini* seed is one of the important marker compound [43]. Phytochemical investigation of the stem bark of *Syzygium cumini* (L.) Skeels (Myrtaceae) yielded four new lignan derivatives characterised as (7*α*,8*α*,2'*α*)-3,4,5-trimethoxy-7,3',1',9'-diepoxylignan (cuminiresinol), (7*α*,7'*α*,8*α*,8'*α*)-3,4-dioxymethylene-3',4'-dimethoxy-7,9',7',9-diepoxylignan-5'-ol (5'-hydroxy-methyl-piperitol), (7*α*,7'*α*,8*α*,8'*α*)-3'-methoxy-9-oxo-7,9',7',9-diepoxylignan-3,4,4'-triol or 3-demethyl-9-oxo-pinoresinol (syzygiresinol A), (7*α*,7'*α*,8*α*,8'*α*)-9-oxo-7,9',7',9-diepoxylignan-3,4,3',4',5'-pentaol or 3,3'-didemethyl-9-oxo-pinoresinol along with the known lignans di-demethyl-5-hydroxypinoresinol, dimethylpinoresinol, didemethoxypinoresinol, pinoresinol and 4'-methyl-5'-hydroxypinoresinol [44]. The anthocyanins occur as 3,5-, but not 3-diglucosides, of delphinidin, cyanidin, petunidin, peonidin, and malvidin. This is the report to use a combination of spectrometric and spectroscopic methods to identify unequivocally the structures of *E. jambolana* fruit anthocyanins [45]. For instance, flavonoids have been referred to as nature's biological response modifiers, because of their inherent ability to modify the body's reaction to allergies and virus and they showed their anti-allergic, anti-inflammatory, anti-microbial and anti-cancer activities. Plant steroids are known to be important for their cardiotonic activities and also possess insecticidal and antimicrobial properties. They are also used in nutrition, herbal medicine and cosmetics [46]. Seed extracts of *S. cumini*, the part most often used in Ayurvedic medicine, were previously shown to have high levels of total phenolics and good activity in the trolox equivalent antioxidant capacity (TEAC) and ferric reducing antioxidant power (FRAP) antioxidant assays [47].

The juice of the ripe fruit, or a decoction of the fruit, or jambolan vinegar, may be administered in India in cases of enlargement of the spleen, chronic diarrhea and urine retention [16,38]. Water-diluted juice is used as a gargle for sore throat and as a lotion for ringworm of the scalp [16,38]. Jambolan juice and mango juice, half and half, quench thirst in diabetics [38]. The seeds (marketed in-inch lengths) and the bark are official in the Dutch [16]. They are much used in tropical medicine and are shipped from India, Malaya and Polynesia, and to a small extent from the West Indies [48], to pharmaceutical supply houses in Europe and England [49].

Studies in the past one decade have also shown that Jamun possess antineoplastic [50]. Radioprotective [51-54] and chemopreventive effects [55] all of which are useful in the prevention and treatment of cancer. The reasons for the myriad pharmacological effects are due to the presence of diverse phytochemicals like flavonoids, anthocyanins, terpenes [2] and are enlisted in **Table 1**.

Extracts of both, but especially the seeds, in liquid or powdered form [61], are freely given orally, two or three times a day to patients with diabetes mellitus or glycosuria [38]. In many cases, the blood sugar level reportedly is quickly reduced and there are no ill effects [38]. Fresh seeds are considered superior to dried seeds [62]. Reduction of blood sugar was obtained in alloxan diabetes in rabbits [62]. In experiments at the Central Drug Research Institute, Lucknow, the dried alcoholic extract

Table 1. Phytochemicals present in the jamun plant.

Sr. No	Plant part	Chemicals present
1	Seeds	Jambosine, gallic acid, ellagic acid, corilagin, 3,6-hexahydroxy diphenoylglucose, 1-galloylglucose, 3-galloylglucose, quercetin, *β*-sitoterol, 4,6 hexahydroxydiphenoylglucose, [2,56].
2	Stem bark	Friedelin, friedelan-3-*α*-ol, betulinic acid, *β*-sitosterol, kaempferol, *β*-sitosterol-Dglucoside, gallic acid, ellagic acid, gallotannin and ellagitannin and myricetine [2,56].
3	Flowers	Oleanolic acid, ellagic acids, isoquercetin, quercetin, kampferol and myricetin [2].
4	Fruit pulp	Anthocyanins, delphinidin, petunidin, malvidin-diglucosides [2,57,58].
5	Leaves	*β*-sitosterol, betulinic acid, mycaminose, crategolic (maslinic) acid, n-hepatcosane, n-nonacosane, n-hentriacontane, noctacosanol, n-triacontanol, n-dotricontanol, quercetin, myricetin, myricitrin and the flavonol glycosides myricetin 3-O-(4"-acetyl)-*α* Lrhamnopyranosides [2,59].
6	Essential oils	*α*-terpeneol, myrtenol, eucarvone, muurolol, *α*-myrtenal, 1, 8-cineole, geranyl acetone, *α*-cadinol and pinocarvone [60].

of jambolan seeds, given orally, reduced blood sugar and glycosuria in patients [62]. Dr. Mukerji, in 1961, called the results promising, though the action of the seed extract is milder than that of the synthetic anti-diabetics. He holds that the bark extract affects the glycogenolysis and glycogen storage in animals [38]. On the other hand, Bhatnagar and co-workers, while screening the jambolan with 174 other popular Indian medicinal plants, found no physiological activity in the bark, which they collected in the month of September [63]. Other reported constituents of the seeds are: tannin, [16] to 19%; gallic acid, 1% to 2%; chlorophyll [55]; fatty acids (palmitic, stearic, oleic, and linoleic) [16]; starch, 41% [15], dextrin, 6.1%; protein, 6.3% [15]; and a trace of phytosterol [16].The seeds are claimed by some to contain a glycoside, jambolin [16,55] or antimellin [62], which halts the diastatic conversion of starch in to sugar [16]; also a resin yielding phenolic substances named jambidol [15,16] and ellagic acid [55], and an alkaloid, jambosine [61]. The bark contains 8% to 19% tannin [15,55], gallic acid, 1.67% [16], resin [64], small amounts of ellagic acid and myricetin [65]. A decoction of the bark is taken internally for dyspepsia, dysentery and diarrhea and also serves as an enema [16]. The dried and powdered seeds and root-bark are similarly employed [16]. Powdered bark mixed with curds is given in menorrhagia. Powdered jambolan and mango seeds, with curds, are used, like the fruit juice, in treating enlarged spleen and retained urine [38]. In India, the seed powder is administered as an antidote for strychnine poisoning [15]. The leaf juice is effective in the treatment of dysentery [16], either alone or in combination with the juice of mango or emblic leaves [38]. The leaves, steeped in alcohol, are prescribed in diabetes [55]. Jambolan leaves may be helpful as poultices for skin diseases [15,16]. The leaves yield 12% to 13% tannin (by dry weight) (IS), also an essential oil containing limeonene and dipentene (20% to 30%), about 40% of sesquiterpene (cadeninic type) and a little azulenic sesquiterpene [62]. Bark decoctions are taken for asthma and bron chitis [5] and are gargled or used as mouth wash for the astringent effect on mouth ulcerations, spongy gums [16] and for stomatitis [38]. Ashes of the bark, mixed with water, are spread over local inflamations; or, blended with oil, applied to burns [38].

In the year 2008, 12.7 million new cancer cases and 7.6 million cancer deaths occurred [66]. More worryingly, predictions are that by the year 2020, the global incidence of the cancer will increase by threefold, with a disproportionate rise in cases from the developing world countries that have limited resources to tackle the problem [67]. The conventional treatment modalities used in treating cancer, the surgery, radiotherapy, hormone therapy and chemotherapy remain prohibitively expensive to the large number of population in the developing countries. With an expected rise in cancer incidence, the mortality and associated morbidity will be enormous due to the compromised financial condition of the patients [66, 67]. Since the dawn of civilization, herbal drugs have been used in the ancient civilizations and their use in the treatment of cancer is on a rise especially in the developing and underdeveloped countries primarily due to its easy affordability, non toxic nature, easy acceptability, less toxic or no toxic effects and easy availability. Plants have been the main ingredients of various medications of the traditional Indian system of medicine, the Ayurveda and one such plant of immense importance is *Eugenia jambolana* Lam (Syn. *Syzygium cumini* Skeels or *Syzygium jambolana* Dc or *Eugenia cuminii* Druce) (**Figure 1**), colloquially known as Java plum, Portuguese plum, Malabar plum, black plum, Indian blackberry, jaman, jambu, jambul and jambool [68].

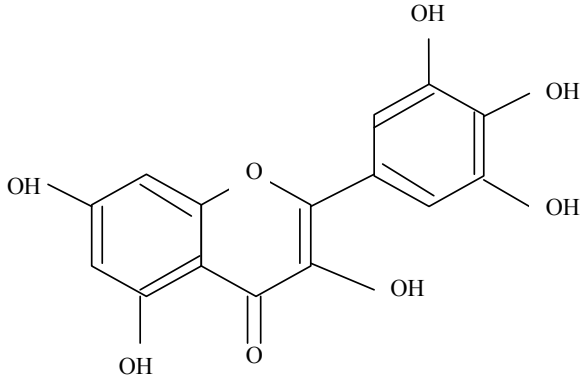
5.1. Chemopreventive Effects

Chemoprevention, a science that has emerged during the three last decade, presents an alternative approach to reducing mortality from cancer. Chemopreventive interventions may be applied at any time during carcinogenesis, from the initial molecular defect through the accumulated molecular, cellular and histopathologic aberrations that characterize disease progression before an invasive and potentially metastatic stage [69]. It aims at blocking, reversing, or delaying carcinogenesis before the development of invasive disease by targeting key molecular derangements using pharmacological or nutritional agents [69]. Very recently [70] have also observed that administration of the jamun extract (25 mg/kg body weight/day) was effective in preventing benzo-a-pyrene-induced forestomach carcinogenesis. Recently, [71] have reported that jamun possess cancer chemopreventive properties in the DMBA-induced croton oil promoted two stage skin carcinogenesis in Swiss albino mice. Feeding of 125 mg/kg body weight/animal/day of the extract either during 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 reduced the cumulative numbers of papillomas, the tumor incidence and increased the average latency period when compared with the control group (carcinogen alone) [71].

Jamun reduced the tumor incidence, tumor burden and cumulative number of gastric carcinomas. Reports also suggest that gallic acid, ellagic acid, flavonoids and anthocyanins (**Figure 2**) present in Jamun are reported to prevent experimental carcinogenesis in various organs (**Table 2**) and may have contributed to the anti-carcinogenesis. Additionally, recent observations also suggest that ellagitannin, a constituent of Jamun and its colonic

Table 2. Phytochemicals of Jamun with reported chemopreventive 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 [73]; 3) Suppress preneoplastic lesions induced by 1, 2-dimethylhydrazine in rat colon [74].
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 20 min before each 12-O-tetradecanoylphorbol-13-acetate (TPA) treatment inhibit the inductions of epidermal ornithine decarboxylase activity, hydroperoxide production and DNA synthesis, and also inhibit the promotion of skin papillomas and carcinomas in the two-step initiation-promotion protocol [79]; 5) Topical application of ellagic acid simultaneously with phorbol-12-myristate-13-acetate (PMA) or mezerein resulted in significant protection against 7, 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) Is a potent inhibitor of benzo[a]pyrene metabolism and its subsequent glucuronidation, sulfation and covalent 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 inhibit 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 mice (a transgenic mice develops prostate tumor) caused a decrease tumors with decreasing the proliferative index with a concomitant increase in the apoptotic cells which were due to decrease in the expression of Cdc2, Cdk2, Cdk4, Cdk6, cyclin B1 and E [83].
4	Quercetin	1) Possesses chemopreventive effects against 4-nitroquinoline 1-oxide-induced and its administration during both initiation and post-initiation phases caused a significant reduction in the frequency of tongue carcinoma in rats. It reduced the polyamine levels and the proliferation [84]; 2) Prevents N-nitrosodiethylamineinduced 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 inhibit BaP-DNA binding, a precursor for mutagenesis and carcinogenesis [89]; 6) Quercetin supplementation prevents benzo(a)pyrene-induced carcinogenesis by modulating the antioxidants and decreasing lipid peroxidation, aryl hydrocarbon hydroxylase, gamma glutamyl transpeptidase, 5'-nucleotidase, lactate dehydrogenase and adenosine deaminase [90].
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) Possess inhibitory effects on phosphatidylinositol 3-kinase and inhibits the 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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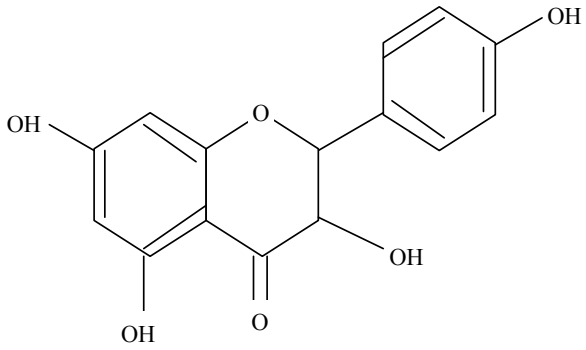

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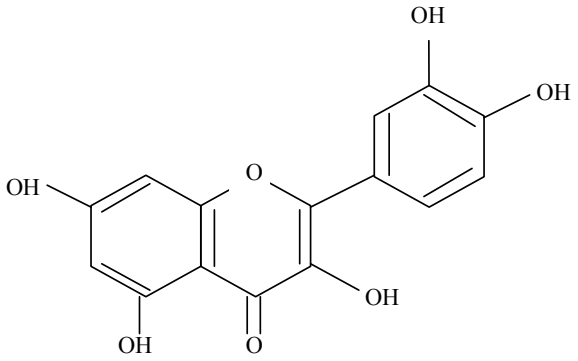

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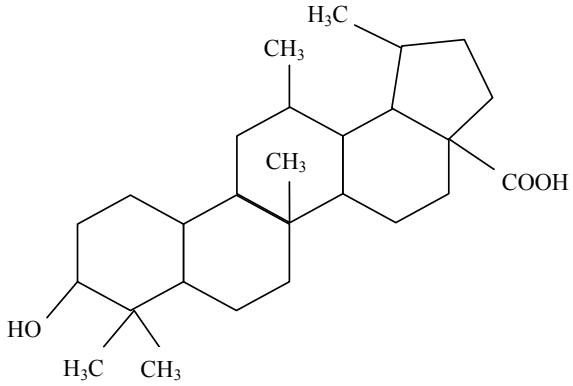

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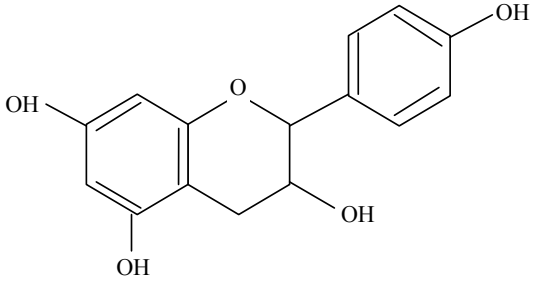

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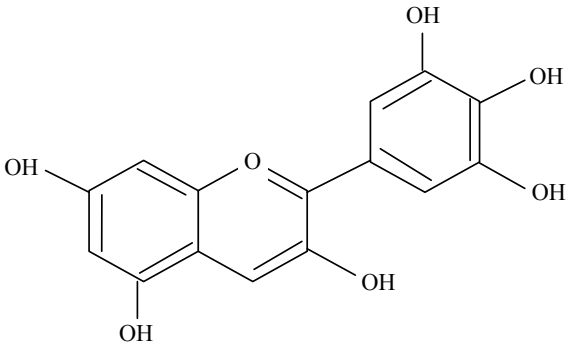

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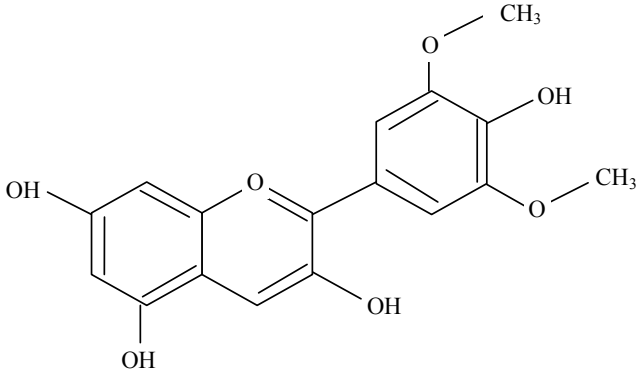

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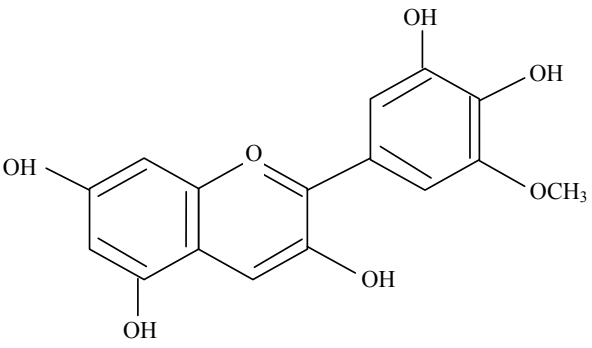

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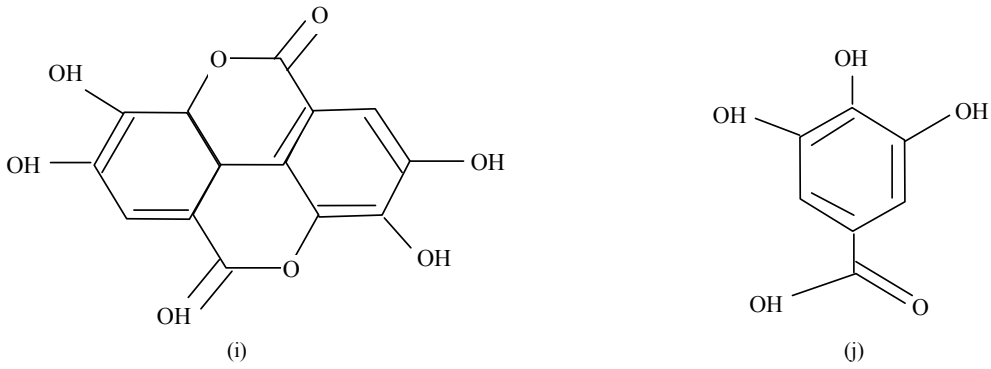

Figure 2. Structures of phytochemicals in Jamun reported to be of use in the treatment of Cancer. (a) Myricetin; (b) Kaempherol; (c) Quercetin; (d) Betulinic; (e) Anthocyanin; (f) Delphinidin; (g) Malvidian; (h) Petunidin; (i) Ellagic Acid; (j) Gallic Acid.

metabolite, urolithin A inhibit want signaling crucial in the process of colon carcinogenesis [106].

5.2. Radioprotective Effects

The affect felt by the normal cells are irreparable damage, leading to the untoward effects forcing the physicians to discontinue or reduce the treatment dose. In such situations, an agent that can render a therapeutic differential between the cancer and normal cell will be highly beneficial. Studies have shown that the intraperitoneal administration of the hydroalcoholic extract of the Jamun seed and the dichloromethane extract of Jamun leaf possess radioprotective effects [107]. Therapeutic differential may be achieved with chemical compounds that may selectively protect the normal cells from the deleterious effects of radiation termed as radio protectors. Since the observations of [108] that the natural amino acid cysteine protected mice against radiation-induced sickness and mortality, many compounds with varied pharmacological properties have been synthesized and evaluated for their radioprotective effects. Pretreatment with hydroalcoholic extract of jamun seeds (5 to 160 mg/kg body weight) for five consecutive days before exposure to supralethal dose of radiation (10 Gy) protected mice against the radiation-induced sickness and mortality. The best effect was observed at 80 mg/kg but only when administered through the intraperitoneal route as 50% of the animals survived when compare to 22% in the oral route and none in the radiation alone cohorts. The intraperitoneal administration of the organic extract (dichloromethane-methane) of leaves (5, 10, 20, 30, 40, 50, 60 and 80 mg/kg b. wt.) for five days before irradiation was also observed to be effective in preventing the radiation-induced sickness and mortality in mice. Histopathological investigations showed that Jamun leaf treatment before radiation elevated the villus height, the number of crypts and reduced the goblet and dead cells when compared with the concurrent irradiation control. The recovery and regeneration was faster in Jamun pretreated animals than the irradiation alone. Jamun extracts also provides protection to the DNA against the radiation-induced DNA damage (explained later). The phytochemicals ellagic acid, gallic acid, quercetin and oleanolic acid (**Figure 2**) present in Jamun also possess radioprotective effects (addressed in **Table 3**).

5.3. Antineoplastic Effects of Jamun

Chemotherapy has been an important modality in cancer treatment for more than five decades and is an obligatory treatment modality when metastasis has ensued. Depending on the clinical stage and the patient compliance, chemotherapy is used either alone or in combination with radiation and surgery [118]. Studies suggest that of all the antineoplastic drugs being used nearly 47% of the drugs are from natural sources [119]. With regard to Jamun many compounds exert beneficial influence (**Figure 2** and **Table 4**).

In vitro studies by [177] have shown that whole Jamun extract possess cytotoxic effects on the cultured human cervical cancer cells, the HeLa (HPV-18 positive) and SiHa (HPV-16 positive). The extract caused a concentration dependent cell death with the effect being more pronounced in the HeLa than SiHa cells [177]. Additionally, both crude as well as the methanolic extracts of the pulp caused a time dependent increase in apoptosis when cultured with 80% concentration of the extracts. The crude extract was observed to be better than the methanolic extract in both the cell lines [177]. In a study that has wide clinical implications, recent studies by [57] have shown that the standardized Jamun fruit extract possess antiproliferative and pro-apoptotic effects in the estrogen dependent aromatase positive (MCF-7aro), and estrogen independent (MDA-MB-231) breast cancer cells. The extract was highly effective against MCF-7aro and the IC50 was observed to be 27 μg/ml to that of 40 μg/ml in MDAMB-231. Most importantly, at equivalent

Table 3. Phytochemicals of Jamun with reported radioprotective activities.

Sr. No	Agent	Phytochemicals radioprotective effects and the mechanisms operating
1	Oleanolic acid	1) Inhibits the growth of ascitic tumors and enhances the recovery of hematopoietic system in irradiated mice [109].
2	Quercetin	1) Protected yeast cells from γ-radiation damage by reducing DNA damage [110]; 2) Effective inprotecting against γ-radiation-induced DNA damage to the human peripheral blood lymphocytes *in vitro* [104, and plasmid DNA [111]. The protective mechanisms were mediated by the antioxidant and inhibition of lipid peroxides [111]; 3) Intraperitoneal administration of quercetin 100 mg·kg/kg for 3 consecutive days before and/or after irradiation prevented radiation induced DNA damage in WBC of mice. Pronounced effects were when querecetin was administered before radiation [112,113]
3	Gallic acid	1) Inhibits radiation-induced damage to DNA and lipid peroxidation in both *in vitro* and *in vivo* conditions [114].
4	Ellagic acid	1) Protects yeast cells from γ-radiation-induced damage by reducing DNA damage [115]; 2) Inhibits γ-radiation induced lipid peroxidation in a concentration-dependent manner *in vitro* [116]; 3) Enhances the cytotoxic effects of radiation in neoplastic cells (Ehrlich ascites carcinoma and Hela) by inducing free radicals, reducing antioxidant enzymes and altering the mitochondrial potential, but protects the normal cells (splenic lymphocytes) of tumor-bearing mice against the radiation damage [117].

Table 4. Phytochemicals in Jamun with reported antineoplastic activities.

Sr. No	Agent	Antineoplastic activity and the mechanisms operating
1	Oleanolic acid	1) Causes a dose and a time dependent cell kill of the human colon carcinoma cell line HCT15. Inhibits proliferation and arrested the cells in G0/G1 phase [120]; 2) Induces apoptosis in human leukemia cells HL60 through caspase activation [121]; 3) Selectively inhibits growth of ras oncogene-transformed R6 cells [122]; 4) Induces apoptosis in human liver cancer HepG2, Hep3B, Huh7 and HA22T cell lines [123]; 5) Inhibits growth of ascitic tumors in mice [108].
2	Quercetin	1) Causes dose-dependent cell kill, chromatin condensation in the colon cancer cells (Caco-2 and HT-29) [124]; 2) Potentiates inhibitory effect of a non-toxic dose of cisplatin, inhibits lung colonization of B16-BL6 colonies and in a dose-dependent manner [125]; 3) Inhibits the growth of the highly aggressive PC-3 prostate cancer cell line and the moderately aggressive DU-145 prostate cancer cell line, but ineffective on the poorly aggressive LNCaP prostate cancer cell line or the normal fibroblast cell line BG-9 [126]; 4) Inhibits expression of specific oncogenes and genes controlling G1, S, G2 and M phases of the cell cycle. It also up-regulated the expression of several tumor suppressor genes [126]; 5) Down regulates gelatinases A and B (matrixmetalloproteinases 2 and 9) in the human prostate cancer cells (PC-3) *in vitro* [127].
3	Kaempferol	1) Inhibits proliferation and induces cell death in human glioma cells through caspase-dependent mechanisms involving down-regulation of XIAP and surviving regulating by ERK and Akt [128]; 2) Mediates p53-dependent growth inhibition and induces apoptosis in human HCT116 colon cancer cell line by affecting Bcl-2 family proteins, PUMA and inducing ATM and H2AX phosphorylation [129]; 3) Induces apoptosis in various oral cancer cell lines (SCC-1483, SCC-25 and SCC-QLL1) through the caspase-3-dependent pathway [130]; 4) Induces apoptosis via endoplasmic reticulum stress and mitochondria dependent pathway in human osteosarcoma U-2 OS cells [131].
4	Myricetin	1) Induce apoptosis in 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 activation of 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-regulation of GADD45beta and 14-3-3 sigma and down-regulation of cyclin B1; and p53-independent mitochondrial-mediated apoptosis through up-regulation of PIG3 and cleavage of caspase-9 and 3 [134]; 4) Possess moderate proteasome inhibitory effects and induce apoptosis in the human leukemia cells Jurkat T cells [135].
5	Gallic acid	1) Induces apoptosis in human prostate LNCaP cells [136]; 2) Induces cytotoxic effects on DU145 prostate cancer cells, through generation of reactive oxygen species and mitochondria-mediated apoptosis [137]; 3) Blocks the growth of DU145 cells at G2/M phases by activating Chk1 and Chk2 and inhibiting Cdc25C and Cdc2 activities [137]; 4) Inactivates phosphorylation of cdc25A/cdc25Ccdc2 via ATM-Chk2 activation, leading to cell cycle arrest, and induces apoptosis in human prostate carcinoma DU145 cells [138]; 5) Possess anti-proliferative, pro-apoptotic and anti-tumorigenic effects against human prostate cells DU145 and 22Rv1 *in vitro* and in nude mice [139]; 6) Synergizes with doxorubicin to suppress the growth of DU145 cells [136]; 7) Induces apoptosis through both caspase-dependent and -independent pathways in the in A375.S2 human melanoma cells [140]; 8) Possesses *in vitro* anticancer effects against the human prostate cancer cells [141]
6	Betulinic acid	1) Is effective against a variety of cancer types but relatively safe to the normal cells and tissue at equal concentrations [141]; 2) Induces potent effect on growth inhibition, G2/M cell cycle arrest and triggers apoptosis in the human gastric adenocarcinoma AGS cells *in vitro*, possibly by the down-regulation of Hiwi and its downstream target Cyclin B1 expression [142]; 3) Causes a dose dependent cytotoxic effect on the rhabdomyosarcoma cell line RMS by inducing apoptosis through the intrinsic mitochondrial pathway. It also decreased GLI1, GLI2, PTCH1, and IGF2 expression as well as hedgehog-response *in vitro*. It also caused retarded the growth of RMS-13 xenografts by causing apoptosis and down-regulating GLI1 expression without affecting the microvascular density, cell proliferation, and myogenic differentiation unaffected [143]; 4) Induces apoptosis through the mitochondrial pathway and inducing cytochrome c

Continued

		release directly via PT Pore. The process is momentarily inhibited by the anti-apoptotic members of the Bcl-2 family, and is observed to be independent of Bax and Bak [144]; 5) Induces cancer cell death by apoptosis through the mitochondrial pathway and also sensitizes the anticancer effects of 5-fluorouracil (SNU-C5/5FU-R), irinotecan (SNU-C5/IRT-R) and oxaliplatin (SNU-C5/OXT-R) in chemoresistant colon cancer cell lines derived from the colon adenocarcinoma cell line
6	Betulinic acid	(SNU-C5/WT) (Jung *et al.*, 2007); 6) Effective against the androgen-refractory human prostate carcinoma PC-3 cells and this it achieves by inhibiting DNA binding, reduced nuclear levels of the NF-kappaB/p65, decreased IKK activity and phosphorylation of IkappaBalpha at serine 32/36 followed by its degradation [145]; 7) Inhibits the proliferation of Jurkat cells by regulating the cell cycle and arresting the cells at G0/G1 phase by down-regulating the expression of cyclin D3. It also induces apoptosis through the Bcl-xl [145].
7	1,8-Cineole	1) Induces apoptosis in human leukemia Molt 4B and HL-60 cells, but not in human stomach cancer KATO III cells [146].
8	*β*-Sitosterol	1) Inhibits growth of HT-29 human colon cancer cells by activating the sphingomyelin cycle [147]; 2) Activates the sphingomyelin cycle and induces apoptosis in LNCaP human prostate cancer cells; 3) Stimulates apoptosis in MDA-MB-231 human breast cancer cells *in vitro* and inhibits growth and metastasis of MDA-MB-231 in SCID mice [148-154]; 4) Inhibits growth and metastasis of human prostate cancer PC-3 cells, *in vitro* and in SCID mice [154]; 5) Induces apoptosis by stimulating Bax and activation of caspases in the HT116 human colon cancer cells [155]; 6) Induces apoptosis in MCA-102 murine fibrosarcoma cells by activation of ERK and the downregulation of Akt [156,157]; 7) Inhibits cell growth and induces apoptosis in SGC-7901 human stomach cancer cells *in vitro* [158]; 8) Induces significant dose-dependent growth inhibition, suppressed expression of beta-catenin and PCNA antigens in human colon cancer cells COLO 320 cells *in vitro*. Feeding beta-sitosterol also caused a dose dependent reduction in the number of aberrant crypt and crypt multiplicity in DMH-initiated rats with no toxic effects [102].
9	Delphinidin	1) Inhibits proliferation of human cancer cell lines MCF-7 (breast), SF-268 (central nervous system, CNS), HCT-116 (colon), and NCI-H460 (lung) [159]; 2) Induce cell cycle perturbations and apoptosis in human cell lines [160]; 3) Inhibits the growth and induced apoptosis in HL60 cells (Katsube *et al.*, 2003). Inhibited the growth of HCT116 cells [161]; 4) Preferentially inhibited the growth of the human vulva carcinoma cell line A431 by affecting the epidermal growth-factor receptor (EGFR), the tyrosine kinase activity and inhibited the activation of the GAL4-Elk-1 [162]; 5) Potent inducer of intracellular hydrogen peroxide and causes apoptosis in a time- and dose-dependent manner. Stimulates JNK pathway activation including JNK phosphorylation and c-jun gene expression, and activates caspase-3 and causes DNA fragmentation in HL-60 cells [163]; 6) Reduces cell growth, is potent EGFR- or PDE-inhibitor and the CAMP hydrolysis [164]; 7) Inhibits cell proliferation of human cancer cell lines, AGS (stomach), HCT-116 (colon), MCF-7 (breast), NCI H460 (lung), and SF-268 [165]; 8) Possess strong growth inhibitory effects against human hepatoma HepG(2), but were less effective against Hep3B, induced apoptotic cell death by up-regulation of Bax and down-regulation of Bcl-2 protein (Yeh *et al.*, 2005); 9) Induces apoptosis in HT-29 cells [166]; 10) Inhibits HGF-mediated membrane translocation of PKCalpha, decreases phosphorylation of STAT3. Repress HGF-activated NFkB transcription, phosphorylation of IKKalpha/beta and IkappaBalpha, and activation and nuclear translocation of NFkappaB/p65 [167]; 11) Suppress the phosphorylation of the epidermal growth factor receptor (EGFR) in human colon carcinoma cell line (HT29), human vulva carcinoma cell line A431 [168]; 12) Treatment to AU-565 cells, a EGFR in the positive breast cancer cells inhibited the phosphorylation of EGFR, activation of PI3K, phosphorylation of AKT and MAPK, inhibited EGF-induced autophosphorylation of EGFR, AKT and MAPK, activation of PI3K and cell invasion [169]; 13) Treatment of in human colon cancer HCT116 cells with delphinidin decrease cell viability; induces apoptosis; cleaves PARP; activates caspases-3, -8, and -9; increase Bax with a concomitant decrease in Bcl-2 protein; causes G2/M cell cycle arrest; inhibited IKKalpha, phosphorylation and degradation of Ikappa Balpha, phosphorylation of NF-kappaB/p65 at Ser(536), nuclear translocation of NF-kappaB/p65, NFkappaB/ p65 DNA binding activity, and transcriptional activation of NF-kappaB [170]; 14) Treatment to human PCa LNCaP, C4-2, 22Rnu1, and PC3 cells resulted in a dose-dependent inhibition of cell growth without having any substantial effect on normal human prostate epithelial cells. It caused a dose-dependent induction of apoptosis and arrest of cells in G2-M phase, decrease in phosphorylation of IkappaB kinase gamma, phosphorylation of nuclear factor-kappaB (NF-kappaB) inhibitory protein IkappaBalpha, phosphorylation of NF-kappaB/p65 at Ser(536) and NF-kappaB/p50 at Ser(529), NF-kappaB/p65 nuclear translocation, and NF-kappaB DNA binding activity. It also inhibited the tumor growth in athymic nude mice implanted with PC3 cells by causing decrease in the expression of NF-kappaB/p65, Bcl2, Ki67, and PCNA [171]; 15) Attenuates neoplastic transformation in JB6 Cl41 mouse epidermal cells by blocking Raf/mitogen-activated protein kinase kinase/extracellular signal-regulated kinase signaling [172]; 16) Possess antiproliferative, anti-invasive and apoptotic effects in human hepatoma Hep3B cells. It also caused concentration dependent increase in the sub-G1 fraction, mitochondrial dysfunction and reduction in antiapoptotic proteins (Bcl-2, xIAP, cIAP-1, and cIAP-2) [173]; 17) Selectively causes cytotoxic effects on the LoVo and LoVo/ADR, human colorectal cancer cell lines; while the non cancerous cells Caco-2 were unaffected [174]; 18) Inhibits receptor tyrosine kinases of the ErbB and VEGFR family [175].
10	Petunidin	1) Induces apoptosis in HT-29 cells [166]; 2) Inhibits the human breast cancer (MCF-7) cell growth [165].
11	Malvidin	1) Inhibits growth and induced apoptosis in HL60 cells [161]; 2) Induces cell cycle perturbations and apoptosis in human cell lines [160]; 3) Reduces cell growth, is potent EGFR- or PDE-inhibitors and effectively inhibited the CAMP hydrolysis [164]; 4) Malvidin inhibited AGS (stomach), HCT-116 (colon), MCF-7 (breast), NCI and H460 (lung) [165]; 5) Exhibits strong growth inhibitory effects against human hepatoma HepG(2), but were less effective against Hep3B [176]; 6) Induces apoptosis in HT-29 cells [166]; 7) Effective on metastatic colorectal cancer cell lines LoVo and LoVo/ADR [174]; 8) Possess antiproliferative, anti-invasive and apoptotic effects in human hepatoma Hep3B cells. It also caused concentration dependent increase in the sub-G1 fraction, mitochondrial dysfunction and reduction in antiapoptotic proteins (Bcl-2, xIAP, cIAP-1, and cIAP-2) [173]; 9) Possess good COX-1 and -2 inhibitory activities [159].

concentrations the extract was relatively non toxic as it did not induce cell death and apoptosis in the normal/ nontumorigenic (MCF-10A) breast cell line (IC50 > 100 µg/ml). Together these results clearly indicate that at supra dietary levels the fruit pulp extract possesses selective antineoplastic effects against breast cancer [57].

6. Conclusion

Jambolan is traditionally used for the treatment of various diseases especially diabetes and related complications. Most pharmacological works on diabetes were carried out with seeds but the pharmacological potential of the other parts of the plant is required to explore in detail. With regard to the antineoplastic activities studies suggest that Jamun is selective in its action in breast cancer cells. The effect of Jamun and its phytochemicals should also be investigated for its chemopreventive effects in other models of carcinogens, that includes chemical, radiation and viral carcinogenesis models. Mechanistic studies responsible for the chemopreventive and radioprotective effects are also lacking and need to be studied in detail. Based on these facts, this review highlights the role of jambolan in various treatments and recommend that further phytochemical and clinical research should be done on this traditional medicinal plant for the discovery of safer drugs. Studies should also be on understanding which of the phytochemicals are responsible for the observed beneficially effects and if effective, their mechanism of action.

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