Snapmelon (Cucumis melo L. Momordica Group), an indigenous cucurbit from India with immense value for breeding

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Snapmelon (*Cucumis melo* L. Momordica Group), an indigenous cucurbit from India with immense value for breeding

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Abstract

Snapmelon(*Cucumis melo* L. Momordica Group; 2*n* = 2*x* = 24) is native to India, where it is widely cultivated and is commonly called 'phut,' which means to split. Immature fruits are cooked or eaten raw. In this paper we review the wealth of genetic resources in Indian snapmelon landraces for resistance to fungal and viral diseases, nematodes, and insects, and tolerance to drought, soil salinity, and high temperature. Global melon breeding programs have transferred many of these qualities into open-pollinated and hybrid varieties of sweet melon cultivated in Africa, Asia, Australia, Europe, and the Americas. Snapmelons are sources of high fruit acidity, a trait that has been utilized to breed uniquely flavored melon cultivars. High frequencies of unique alleles have been identified in snapmelon collections from various parts of India. Snapmelon can serve as a source of new resistance genes to combat pathogens and pests, and to strengthen crop resilience against climate change. More effort is needed to collect, characterize, evaluate and preserve snapmelon diversity in genebanks.

INTRODUCTION

Snapmelon is native to India, which is considered the center of domestication of melon by some researchers with the earliest melon remains at the Indus Valley site of Harappa dated between 2300 and 1600 BC (Vishnu-Mittre, 1974). It is widely cultivated in various Indian states such as Rajasthan, Gujarat, Punjab, Haryana, Uttar Pradesh, West Bengal and some other northeastern states. It is also cultivated in other countries of South-East Asia, for instance Myanmar (Yi et al., 2009) and Vietnam (Phan et al., 2010). It is commonly called 'phut,' which means to split. Fruit cracking is either longitudinal or random starting in the middle of fruit, though in some instances only skin peeling (longitudinal or random) occurs (Dhillon et al., 2007). It is also known by other names such as 'phoot kakari' or 'kakadia.' Several types of fruit shape are found in snapmelon viz. round, acorn, oblate, ovate, elongated, elliptical and pyriform (Dhillon et al., 2007). Fruit flesh color varies from cream and yellow to orange. Vines are monoecious. Immature fruits may be eaten raw or cooked, or pickled or dehydrated for off-season use. After removing the coat, seeds are used in bakery products and the traditional drink 'thandai'. Fruits are sources of vitamin C, iron and calcium (Goyal and Sharma, 2009). We review the wealth of genetic resources in Indian snapmelon landraces for resistances to fungal and viral diseases, nematodes and insects, tolerance to drought and salinity, genes for unique flavors, and the present status of genetic diversity of snapmelons in different parts of India.

Fungal disease resistance

Powdery mildew is a serious foliar disease of melon worldwide (Sitterly, 1978). It affects the plant canopy and subsequently the fruit yield and quality. Cucurbit powdery

mildew is mainly incited by two fungal species: *Podosphaera xanthii* (syn. *Sphaerotheca fuliginea* auct.p.p.) and *Golovinomyces cichoracearum* (syn. *Erysiphe cichoracearum* auct.p.p.), the first species being the most frequent on melon. More than 30 races of each pathogen have been identified on melon (Lebeda and Sedláková, 2004; McCreight et al., 2012). Powdery mildew was first noted on an epidemic scale in 1925 in the melon production area in the Imperial Valley of California where it remained a serious problem for several successive years (Jagger, 1926; Jagger and Scott, 1937). A source of genetic resistance was identified in accession Calif. 525, which was a self-pollinated increase of 'Big Round' that was brought to the United States by D.N. Mehta (Second Economic Botanist, Nagpur Provinces, India) an Indian student of J.T. Rosa (Swarup, 2000; Kathleen R. Reitsma, personal communication; I.C. Jagger, unpublished pedigree note). This germplasm was collected from Kathiawar region of Gujarat state in India. Powdery mildew-susceptible, orange flesh 'Hale's Best' melon (*C. melo* Reticulatus group) was crossed with Calif. 525 and the F_1 was resistant to the local strain of powdery mildew. A resistant F_2 selection that produced large elongated fruits was backcrossed to 'Hale's Best' to recombine powdery mildew resistance with superior horticultural qualities of 'Hale's Best'. Seven generations of inbreeding and selection led to the development and release of 'Powdery Mildew Resistant Cantaloupe No. 45' ('PMR 45') to the western United States melon industry in 1935 (Jagger and Scott, 1937).

Race 2 of *P. xanthii* overcoming the resistance of 'PMR 45' appeared in Imperial Valley in 1938 (Jagger et al., 1938). Resistance to this new race was identified in 'Piria', that was donated by D. Mehta of Nagpur, Madhya Pradesh, India to the United States in 1929 and

designated PI 79376 (Pryor et al., 1946; USDA, ARS, 2014). Resistance to race 2 was combined with resistance to race 1 in melon cultivars ‘PMR 5’ and ‘PMR 6’, released in 1942, and ‘Campo’ and ‘Jacumba’ released in 1964 in the United States (Bohn et al., 1965). PI 124111 (MR-1)from the Bihar state of India, was resistant to 26 races of *P. xanthii* (McCreight et al., 2012). This accession was also resistant to several races of *G. cichoracearum* (Pitrat et al., 1998; Lebeda et al., 2012).

Powdery mildew resistance genes identified in Calif. 525 and PI 79376 were introgressed in many melon breeding lines and cultivars (Harwood and Markarian, 1968) and are still prevalent in modern melon releases in United States: ‘Georgia 47’ (Anon. 1954), ‘Home Garden’ (Ivanoff, 1957), ‘Gulfstream’ and ‘Planter Jumbo’ (Nugent, 1994), ‘Mainstream’ and ‘Edisto 47’(Nugent et al., 1979), and recently released ‘Chujuc’ and ‘Pacal’ (Crosby et al., 2007, 2008). Moderately powdery mildew-resistant melon cultivar ‘Punjab Sunehri’, released in 1975 in Punjab state of India (Nandpuri et al., 1975; Waraitch et al., 1977), has ‘Edisto’ in its pedigree. This orange flesh powdery mildew-resistant cultivar remained popular with Indian growers and consumers for two decades. Race 1 and race 2 powdery mildew-resistant Reticulatus Group cultivars PMR 5, Dulce, Gulfstream and Jacumba were resistant to a race-unknown population of powdery mildew in India in a controlled inoculation experiment (Waraitch et al., 1977). PI 414723 was also resistant to many races of *P. xanthii* (McCreight et al., 2012). Several race-specific genes have been described in these accessions (Dogimont, 2010-2011). Fergany et al. (2011) reported two additional snapmelon accessions resistant to powdery mildew: AM 22 to races 1 (strain Sm3) and 3 (strain 00Sm39) and AM 86 resistant to races 1 and 5 (strain 98Sm65). A large number of powdery

mildew resistance genes have been described (Dogimont 2010-2011), most of them are likely allelic. Several QTLs conferring resistance to different races have been mappind in melon Linkage Groups (LG) II, V and XII (Perchepied et al., 2005; Fukino et al., 2008; Zhang et al., 2013)

Downy mildew caused by *Pseudoperonospora cubensis* (Berk. & Curtis) Rostov. is a common foliar disease of melons in humid production areas of the world. Six pathotypes have been identified: 1 and 2 in Japan, 3 and 6 in Israel, and 4 and 5 in the United States (Cohen et al., 2003). These pathotypes do not colonize *Luffa* ssp. (Thomas et al., 1987), whereas the Indian and Chinese isolates of cucurbit downy mildew are able to colonize *Luffa* ssp. and are considered as distinct races. Shetty et al. (2002) confirmed that the downy mildew races in U.S. are distinct from the race in Asia, whereas the race in Poland is similar to the races in the U.S. Four partially dominant resistance genes have been identified in three accessions of snapmelon: PI 124111 (*Pc-1, Pc-2*), PI 414723 (*Pc-3*), and 5-4-2-1 (*Pc-5*) (Dogimont, 2010-2011). Resistance to *P. cubensis* races 3 and 6 in Israel has been found in PI 124111F controlled by two R genes, *At1* and *At2* (Taler et al., 2004). Interestingly, PI 124111F, reported resistant to six pathotypes of *P. cubensis*, was susceptible to an Indian isolate of *P. cubensis* (More, 2002), but IC 267353, IC 274029, KP7, and B-159 were resistant to this Indian isolate (Dhillon et al., 2007; Pandey et al., 2008). It will be interesting to test the reaction of these genotypes to the six pathotypes of *P. cubensis* available in the other parts of the world.

Melon Fusarium wilt (MFW) is caused by the soil-borne fungal pathogen *Fusarium oxysporum* Schlechtend:Fr.f.sp.*melonis* (H.N. Hansen) W.C. Snyder & H.N. Hans (*Fom*).

127 The pathogen survives in the soil as chlamydospores and is able to colonize crop residues and
128 roots of most of crops cultivated in rotation with melon (Gordon et al., 1989), thus rendering
129 crop rotation as a limited tool to manage this disease. Soil solarization can reduce soil
130 inoculum but is limited by local climate factors, i.e., temperature and relative humidity
131 (Tamietti and Valentino, 2006) and it is not suitable for intensive vegetable farming systems
132 where there is insufficient time for effective soil solarization. Grafting of susceptible melon
133 scions onto Fusarium wilt-resistant rootstocks is an effective control strategy for MFW, but
134 the additional cost limits this approach to very high value melon cultivars. Use of resistant
135 varieties is regarded as the most effective strategy to control this disease. MFW isolates have
136 been designated into four physiological races: 0, 1, 2, and 1.2. Two dominant resistance
137 genes, *Fom-1* and *Fom-2*, control resistance to races 0 and 2, and 0 and 1, respectively and
138 were identified in PI 124111F and its derivative MR-1 (Cohen and Eyal, 1987; Zink and
139 Thomas, 1990). *Fom-2* has also been reported in PI 414723. Using MR-1 and PI 414723,
140 these two genes have been cloned using chromosome walking strategies and belong to the
141 NB-LRR family (TIR subfamily for *Fom-1* and non-TIR for *Fom-2*) (Joobeur et al., 2004;
142 Brotman et al., 2013). These *Fom* genes along with cucurbit powdery mildew resistance
143 genes are routinely deployed in modern melon commercial hybrids. Accession AM 27
144 exhibited uniform resistance to race 2 and segregated for resistance to race 1 (Fergany et al.,
145 2011). Resistance to race 1.2 seems to have a complex genetic control what is hampering the
146 development of reliable molecular markers and subsequent cloning (Oumouloud et al., 2013)

147 MFW and leafminer (*Liriomyza* spp) are the most devastating disease and insect pests
148 of melon in India. The prevailing melon cultivars grown by Indian farmers (NS 7475, Punjab

Sunehri, Punjab Hybrid 1, Durgapur Madhu, Kashi Madhu, Pusa Madhurus, Arka Jeet, Arka Rajhans) are susceptible to MFW. Existing global melon genetic resources, including recent releases of seed companies, have been found susceptible in Indian field conditions (N.P.S. Dhillon, unpublished data; Arvind Kapur, personal communication).

There was a much lower incidence of MFW or Monosporascus sudden wilt, which is incited by *Monosporascus cannonballus* (Pollack & Uecker), exhibited by snapmelon germplasm compared with 100% loss of muskmelon (Reticulatus Group) landraces and varieties during melon germplasm collection expeditions in farmer's fields in the arid and semi-arid areas of Rajasthan and southern Punjab in India (N.P.S. Dhillon, unpublished data). Snapmelon accessions may have additional genes for resistance to MFW and *M. cannonballus*.

Alternaria leaf blight of melons caused by *Alternaria cucumerina* (Ellis &Everh.) is widespread in wet and warm conditions (20 to 30°C) areas having sandy soil such as southern India and southeastern United States (Thomas, 1996). Resistance to this fungal pathogen is controlled by the single dominant gene *Ac* in MR-1, which was derived from PI 124111 (Thomas et al., 1990).

Viral disease resistance

Numerous viruses affect melons worldwide. Three kinds of virus symptoms generally appear on the vines: 1) mosaic on leaves associated with leaf and fruit discolorations and deformation, 2) yellowing of leaves coupled with leaf thickening, and 3) necrotic spots or

progressive necrosis resulting in vine death (Lecoq et al., 1998). Melon fields may be infected with more than one virus.

Cucumber mosaic virus (CMV) causes economic losses in melon worldwide. Resistance to CMV was first reported in accessions belonging to the Conomon Group from East Asia and is controlled by recessive oligogenes (Karchi et al., 1975; Dogimont et al., 2000; Essafi et al., 2008; Guiu-Aragonés et al., 2014). This resistance is effective against some CMV strains and is, thus, not easy to use for the development of commercial F_1 hybrids. Resistance to a broad spectrum of CMV strains likely will need the combination of genes from different CMV resistance sources.

Snapmelon accessions AM 25, AM 82, IC 274014, SM 67, SM 72, SM 73, SM 82, MM 3974, MM 3982 and MM 3994 were highly resistant to CMV (Dhillon et al., 2007, 2009; Fergany et al., 2011; Malik et al., 2014). These accessions may contribute to a broad-based resistance against different strains of CMV prevailing in different parts of the world.

Zucchini yellow mosaic virus (ZYMV) is a serious virus of cucurbits worldwide (Desbiez and Lecoq, 1997). Three complementary, dominant genes in PI 414723 (*Zym-1*, *Zym-2*, and *Zym-3*) impart resistance to ZYMV (Pitrat and Lecoq, 1984; Danin-Poleg et al., 1997). Accessions IC 274007, IC 274014, and PI 179905 are potentially useful sources of resistance to ZYMV (Dhillon et al., 2007).

Papaya ringspot virus watermelon strain (PRSV-W), formerly *Watermelon mosaic virus 1*, is very common in the tropics (Lecoq et al., 1980). Two alleles, Prv^1 and Prv^2, found in the weedy type melons PI 180280 and PI 180283, respectively, condition resistance to

PRSV-W (Kaan, 1973; Webb, 1979; Pitrat and Lecoq, 1983). PI 414723 has the *Prv*^{2} allele (M. Pitrat, unpublished data) and this gene has been recently isolated from the Indian accession PI 414723, encoding for a NBS-LRR type protein (Brotman et al., 2013). Nine accessions from northern India segregated for resistance to PRSV-W: IC 267360, IC 267363, IC 267374, IC 267384, IC 274006, IC 274007, IC 274010, IC 274011 and IC 274013 (Dhillon et al., 2007). The genetic relationships between genes in these accessions and *Prv* have not been established. Twenty-nine landraces from Kerala and Tamil Nadu states in southern India, exhibited necrotic symptoms in response to artificial inoculation with the potyvirus *Moroccan watermelon mosaic virus* (MWMV) (Fergany et al., 2011).

Watermelon mosaic virus (WMV), formerly *Watermelon mosaic virus 2*, is another widespread potyvirus of melons. Genetic resistance to WMV was reported in PI 414723 (Munger, 1991) and is controlled by a single dominant gene, *Wmr* (Gilbert et al., 1994).

Cucurbit aphid-borne yellow virus (CABYV) is a worldwide important polerovirus of melons transmitted by aphids. 'Faizabadi phoont', and PI 414723 were reported resistant to CABYV (Dogimont et al., 1997).

Watermelon chlorotic stunt virus (WmCSV) is economically important in Yemen, Sudan, and Iran (Yousif et al., 2007). PI 414723 provided resistance during graft inoculation experiments and multiple field trials in Sudan (Yousif et al., 2007).

Cucurbit leaf crumple virus (CuLCrV) is a sweetpotato whitefly-transmitted begomoviruses of melon that have appeared in commercial melon fields in the southwestern United States, western Mexico, and Central America since 1977. PI124111, PI 179901, and

PI 414723 exhibited partial resistance to CuLCrV in naturally-infected field and controlled inoculation greenhouse tests. Resistance in PI 313970 (Acidulus Group) was conditioned by a single recessive gene and appeared allelic to that in the snapmelon accessions (McCreight et al., 2008).

Cucumber green mottle mosaic virus (CGMMV) is economically significant in greenhouse production (Hollings et al., 1975) and has been reported in Europe and Asia. A biological vector of this melon virus is unknown, but CGMMV is transmitted mechanically and through growing media (Lecoq et al., 1998). In early 1980s, CGMMV affected 70% to 80% of plants in peri-urban cucurbit fields of Delhi, India (Raychaudhury and Varma, 1978). Identification of resistance to CGMMV in 'Phoot' led to the development of five Indian lines (VRM 5-10, VRM 29-1, VRM 31-1-2, VRM 42-4, and VRM 43-6) that had high-level resistance to CGMMV along with improved yield and sweetness (More et al., 1993).

Kyuri green mottle mosaic virus (KGMMV) is economically significant in Japan, Korea, and Indonesia (Daryono et al., 2005). It is mechanically transmitted and seed-borne. PI 414723 is resistant to KGMMV (Daryono et al., 2005).

Spring (dry season) melons in the trans-Gangetic plains of India are threatened by CMV, CGMMV, SqMV, PRSV, and ZYMV, whereas whitefly transmitted begomoviruses predominate during the rainy season in this region (Sharma et al., 2007). Landrace IC 274014 is an asymptomatic host of CMV that also exhibited field resistance to an unidentified begomovirus (Sharma and Kang, 2009).

Root-knot nematode and insect resistance

Root-knot nematode (RKN), *Meloidogyne* spp, is found in melon fields worldwide, particularly in sandy soils. Its impact on melon yield depends upon the RKN population density in the field. Current melon cultivars are susceptible to RKN. High-level resistance to *M. incognita* has been identified in landrace IC 274023 (Dhillon et al., 2007) which should be exploited to develop the first RKN-resistant melon variety.

Melon aphid, also called cotton melon aphid (CMA), *Aphis gossypii* Glover, is found throughout most of the temperate, subtropic and tropic regions of the world. Younger plants are more susceptible to feeding. CMA is also an efficient vector of viruses including CMV and potyviruses. Strong resistance to CMA biotype D (McCreight et al., 1992) available in PI 414723 was used in conventional breeding to develop orange flesh melon breeding lines AR Topmark, AR-5, and AR Hale's Best Jumbo (McCreight et al., 1984). These breeding lines exhibited different levels of resistance to virus transmission by CMA (Kishaba et al., 1992). PI 414723 has three components of resistance to CMA: antibiosis, antixenosis, and tolerance (Bohn et al., 1972). Tolerance to CMA in PI 414723 is expressed as freedom from curling of leaves and is governed by the single dominant gene *Ag* (Bohn et al., 1973). Snapmelon landraces IC 267353, IC 267384, and IC 274010 have resistance to virus transmission by CMA (Dhillon et al., 2007).

Cucumber beetles (CB) infest melon seedlings and fruit (Kishaba et al., 1998). Seedling and fruit resistance to western striped CB [*Acalymma trivittata* (Mannerheim)] and spotted CB [*Diabrotica undecimpunctata* (Mannerheim)] has been identified in PI 414723 but genetic basis of resistance not determined.

253 Sweetpotato whitefly (SPWF), *Bemisia tabaci* Gennadius, is another economically
254 important insect pest in the desert southwestern United States. It has several biotypes. SPWF-
255 A is a vector of *Lettuce infectious yellow virus* (LIYV) whereas SPWF-B is a vector of
256 *Cucurbit yellow stunting disorder virus* (CYSDV) and *Cucurbit leaf crumple virus*
257 (CuLCrV). Resistance to SPWF-B was reported in PI 414723 (Boissot et al., 2003).

258 **Melon flavor enrichment**

259 Accumulation of sugar and acid in melon fruit imparts unique taste and flavor.
260 Titrable acidity of Indian commercial melons ranges 0.12% to 0.2% (N.P.S. Dhillon,
261 unpublished data). High acidity sources have been reported in snapmelon landraces IC
262 274021 (0.61%) and IC 267360 (0.57%) (Dhillon et al., 2007). The low-pH gene derived
263 from snapmelon accession IND-35 was exploited using marker-assisted selection by
264 Syngenta in 2008 to develop the pleasant-tasting F_1 hybrid melon 'GWANIPA' that was
265 commercialized in UK, Germany and Holland by Kernel Export (Jordi Garcia-Mas, personal
266 communication). This melon has a lemon flavor and contains 700-800 mg citric acid per 100
267 g fruit fresh weight (FW) with a pH level of 4.5 (patent no. EP 1587933 B1) (Casanueva et
268 al., 2010).

269 **Vitamin and Mineral content**

270 Higher concentrations (up to 34.1 mg•100 g^{-1} FW) of vitamin C were detected in
271 snapmelon landraces of northern India compared to the germplasm from eastern India (upto
272 19.4 mg•100g^{-1} FW) and southern India (up to 9.0 mg•100g^{-1}FW) (Dhillon et al., 2007;
273 Fergany et al., 2011; Malik et al., 2014).

Iron and zinc deficiency is recognized as a nutritional problem worldwide (Uauy et al., 2006). Wide variation for P (2.6 to 21.4 mg•100g^{-1}FW), K (19.7 to 232.4 mg•100g^{-1}FW), Fe (0.5 to 0.89 mg•100g^{-1}FW) and Zn (0.12 to 0.68 mg•100 g^{-1}FW) have been identified in landraces (Fergany et al., 2011). This genetic variation for vitamins and minerals is important for breeding of new mineral- and vitamin-rich snapmelon varieties. Snapmelons are consumed by poor and middle-class consumers, and the fruit are available in the market for nearly five months of the spring and rainy season. Snapmelon fruits were used as food in the two Japanese islands (Hachijo and Fukue) during the two World Wars (Fujishita, 2004).

Snapmelon genetic diversity

Based on the variability at nine simple sequence repeat loci (160 alleles, polymorphism information content value 0.81), clear genetic differentiation was observed among gene pools of snapmelon germplasm from northern, southern and eastern region of India (Dhillon et al., 2013). Global melon reference populations were distinct from this germplasm; clearly snapmelon landraces possessed unique alleles when compared with international reference accessions. Snapmelon germplasm offers opportunity to widen the genetic base of melons inhabiting the secondary centers of diversity (eastern Asia, western Mediterranean area) and proximal parts (e.g., Turkey) of the primary center of diversity.

Snapmelons for developing climate-smart melons

To maintain and increase crop productivity in increasingly hostile environments, novel sources of genetic variation must be sought for adapting crops to unstable climate. Snapmelons were little explored gene pools and are readily available to the sweetmelon

genepools through conventional hybridization. Snapmelons are drought hardy, and are cultivated by small-scale farmers in arid and semiarid regions of India during the rainy season (Pareek and Samadia, 2002). Two highly drought and heat tolerant snapmelon selections, AHS 10 and AHS 82,were bred from local landraces from the arid region of Rajasthan, India (Pareek and Samadia, 2002). Snapmelon accession RSM 50 was highly drought tolerant compared to Reticulatus Group cultivars in a controlled (water deficit) irrigation field experiment (Dhillon et al., 2013). Accession Calif. 525 was acknowledged for contributing high-levels tolerances to salt and high temperature along with resistance topowdery mildew in 'PMR 45', the first modern western U.S. shipping-type melon (Jagger and Scott, 1937). Grafted melons are more tolerant to salinity than non-grafted controls (Orsini et al., 2013). Snapmelon landraces from the coastal, arid areas of India (Gujarat, Karnataka, Kerala, Andhra Pradesh, Tamil Nadu, Odisha) are potential sources of new salt, drought and heat tolerant rootstocks for melon grafting.

Conclusion

Snapmelons originated in India. This horticultural group has provided yeoman service to sweetmelon breeding programs worldwide. For example, PI 414723 was used in breeding as source of resistance to eight fungal and viral diseases and two insect pests. PI 124111 (MR-1) is resistant to powdery and downy mildew, Alternaria and Fusarium wilt. More than 1000 snapmelon accessions are maintained in four national genebanks in India (National Bureau of Plant Genetic Resources, New Delhi; Central Institute for Arid Horticulture, Bikaner; Central Horticultural Experiment Station, Bhubaneswar; Punjab Agricultural University, Ludhiana), but they have not been comprehensively evaluated against various

biotic and abiotic stresses. Erosion of snapmelon genetic diversity in India is a real threat because of rapid urbanization, and swift adoption and spread of commercial F_1hybrid sweetmelons across India. International, collaborative research efforts should be launched to collect, characterize and evaluate the snapmelon germplasm available in the many different agro-ecological regions of India. This will result in identification of unique and useful genes to broaden the narrow gene pool of sweetmelons, provide new sources for resistance to various diseases and insects of melons, and help in meeting the challenges of climate change to sustainable production of melon crop worldwide.

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