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Review Article

Strategies for the Development of Resistance against Banana Bunchy Top Disease

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ABSTRACT

Banana bunchy top disease (BBTD) caused by the banana bunchy top virus (BBTV) is one of the serious diseases of concern. It is mainly transmitted by banana aphids. This review focuses on information related to the problem and biotechnological solutions, including pathogen-derived resistance (PDR) and gene silencing. Various strategies adopted for the development of transgenic varieties viz., Pathogen-derived resistance (PDR), Replicase-mediated resistance, Coat Protein (CP)-mediated protection, Movement Protein-mediated protection (MP-MP), Antisense technology, RNA interference (RNAi)-mediated resistance, Virus-induced cell death, DNA binding proteins, etc. Recently, BBTV-resistant banana clones have been developed through RNAi technology. More emphasis is required to create research and development in the banana growing belt of the world. Today's demand is to adopt biotechnological tools and their output for the sustainability and eco-friendly cultivation of bananas. These techniques are also useful for the promotion of organic cultivation and the conservation of our natural resources by reducing the use of pesticides. Adoptions of transgenic resistance crop varieties in bananas, as well as other crops, are required to fight against diseases of similar nature.

Keywords- *Banana bunchy top virus (BBTV), Antisense technology, transgenic resistance, Gene silencing, Coat protein, RNA interference, RNAi technology*

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INTRODUCTION

Banana (Musa paradisiaca) is the most popular commercial fruit crop grown worldwide and serves as a staple food in many countries [1]. Banana production and export worldwide are affected by many viral diseases such as banana bunchy top disease (BBTD) which is caused by banana bunchy top virus (BBTV) [2]. BBTV is one of the most serious diseases of bananas in Asia, Australia, and the South Pacific. It can contribute up to 100 % yield reduction and is responsible for a dramatic reduction in cropping areas in the old world. The disease received its name from the bunched appearance of leaves at the top of infected plants. It is a Nano virus, a single-stranded DNA virus with isometric virions 18 - 20 nm in diameter [3, 4]. It belongs to the family Nanoviridae, a small family of plant-infecting, circular single-stranded DNA (ssDNA) viruses. Nanoviridae has two genera, Nanovirus and Babuvirus, and a total of nine virus species [4, 5]. Genus Babuvirus initially included only BBTV [6], but later Abaca bunchy top virus (ABTV) and Cardamom bushy dwarf virus (CBDV) were reported as new virus species under this genus [7, 8]. BBTV infects most banana cultivars, retards the growth of infected plants, and causes economic losses to banana production. BBTV is transmitted by vegetative propagation and the aphid vector, Pentalonia nigronervosa [9]. Due to the high destructive potential of the disease, the Invasive Species Specialist Group (ISSG) of the International Union for the Conservation of Nature (IUCN) listed BBTV as one of the World's 100 Worst Invasive Alien Species [10]. This review aims to study the banana bunchy top virus distribution, symptoms, transmission, host range, genome organization, and biotechnological intervention to deal with the banana bunchy top disease.

HISTORY AND SPREAD OF THE VIRUS

Banana bunchy top disease has been reported in many countries. It was first reported from Fiji in 1889 and caused tremendous losses that threatened Fiji's banana export industry [11, 12, 13].

Subsequently, this virus spread to Australia and threatens the banana industry there because of the importation of infected banana suckers from Fiji [11, 14]. In the year 1913, it was introduced in Sri Lanka and later into Southern India in the 1940s, where the virus spread to all banana-growing parts and

covered the entire area of the country during the 1970s [15]. The hill bananas (Virupakshi AAB and Sirumalai AAB) variety grown in Tamil Nadu under a multitier system was highly susceptible to BBTV. The virus causes a drastic reduction in hill banana cultivation from 18,000 ha in the 1970s to 2,000 ha at present and showed an incidence of 14-74% percentage [16, 17, 18]. BBTD incidence occurred between 20-30% from 2006 to 2007 in India's northern zones viz., Lucknow, Barabanki, Bahraich, Kanpur, and Etawah districts of Uttar Pradesh [19]. According to a recent report, there was a newly isolated BBTV complete genome that was identified in Meghalaya, India, and the Pacific India Ocean group [20].

Table 1: Reports of banana bunchy top disease from different parts of the world

Sl. No.	Continents	Country – year	Reference
1	Africa	Egypt – 1901	[11]
		Congo – 1958	[21]
		Gabon – 1960	[22]
		Zaire – 1960	[22]
		Burundi - 1960-80	[23]
		Central African Republic - 1960-80	[24]
		Rwanda - 1960-80	[25]
		Malawi – 1990	[26]
		Angola – 2009	[27]
		Cameroon – 2009	[28]
2	Asia	Sri Lanka – 1913	[29]
		Philippines – 1924	[30]
		India – 1940	[15,31]
		Taiwan – 1961	[30]
		Malaysia – 1969	[31]
		Myanmar – 1969	[32]
		China – 1979	[33]
3	Pacific regions	Australia – 1913	[11, 14]
		Vietnam – 1969	[32]
		Fiji – 1889	[11, 30]
		Hawaii – 1989	[24]

SYMPTOMS OF BANANA BUNCHY TOP DISEASE

Banana bunchy top disease symptoms are evident approximately 25 days in plants following the transmission of the virus, although this varies depending on the temperature and age of plants [34]. The typical symptom of a bunchy top shows dark green streaks appearing in the veins of the lower portion of the leaf midrib and the leaf veins, midribs, petioles and pseudo stem with the group of leaves clustered on the top of the plant gives the bunchy appearance to the plant. As the disease progress in the advanced stage, the apex leaves of the plant become short and narrow with chlorotic and curling towards the upward side and become brittle. Plants infected in a later stage or in severe infection, produced small distorted fruits and the tip of the male bud are look like the shape of a bird's mouth [35] (Figure: 1). Although the symptoms are distinctive in all Musa species reported some symptomless BBTD from Taiwan [24]. They also reported attenuated symptoms in bananas from Fiji followed by an initial severe outbreak. However, it was reported that no natural resistance has been found in any banana cultivars [36].

Figure 1: Symptoms of Banana Bunchy Top disease

TRANSMISSION OF THE BANANA BUNCHY TOP VIRUS

The bunchy top disease is transferred mainly by banana aphids (Pentalonia nigronervosa). Over long distances, it is transmitted by vegetative propagation [11]. It is not transmitted by mechanical means [37]. It was reported that the manner of aphid transmission is persistent and non-replicative and the efficiency of transmission ranges between 46 and 67% [9]. He also reported that the acquisition feeding period is a minimum of four hours and inoculation feeding of a minimum of fifteen minutes after removal from the virus source Retention of infectivity in the aphid has been reported up to 20-23 days.

Host range of the virus

Banana bunchy top virus has a wide range of host plants from the families including Araceae, Commelinaceae, Musaceae, and Zingiberaceae and alternative hosts are Canna indica and Hedychium coronarium were reported to be the host for BBTV in Taiwan [38, 39].

GENOME ORGANIZATION OF BBTV

Banana bunchy top virus is isometric in shape with a diameter of 18-20 nm and belongs to the genus Babuvirus from the family Nanoviridae [4, 6]. It has a multicomponent genome comprising at least six transcriptionally active circular single-stranded DNA (DNA-R, -U3, -S,-M, -C, and -N) components, each approximately 1 kbp in size [4, 6, 40]. Individually each genomic component is encapsulated in the isometric virion particles of 18–20 nm diameters. All components have a few common genome organizations viz. common region-stem loop (CR-SL), common region–major (CR-M), open reading frame (ORF), TATA box, and Polyadenylation signal associated gene (Figure: 2). Each icosahedron contains 20 triangular faces and 30 edges. Some isolates of BBTV contain additional Rep-encoding DNAs that are capable only of self-replication and behave like satellite molecules. The genomic components are composed of two parts one is an intergenic region (IR) and at least one open reading frame (ORF) which is transcribed in the virion sense [40].

Genome organization of BBTV reconstructed

Table 2: Roles of each component

Sl. No.	Components	Encodes to	Reference
1	DNA-R	Master replication initiation protein (Rep) - crucial for trans-replication of the BBTV genomic components (Horser et al., 2001 a).	[41]
2	DNA-S	Coat protein (CP)	[42]
3	DNA-M	Movement protein (MP)	[42]
4	DNA-N	Nuclear shuttle proteins (NSP)	[43]
5	DNA-C	Cell cycle link protein (Clink) – it has plant retinoblastoma-like binding ability to switch the host plant cells to S phase to make them more permissive for viral replication.	[43, 44]
6	DNA-U3	In pipeline	

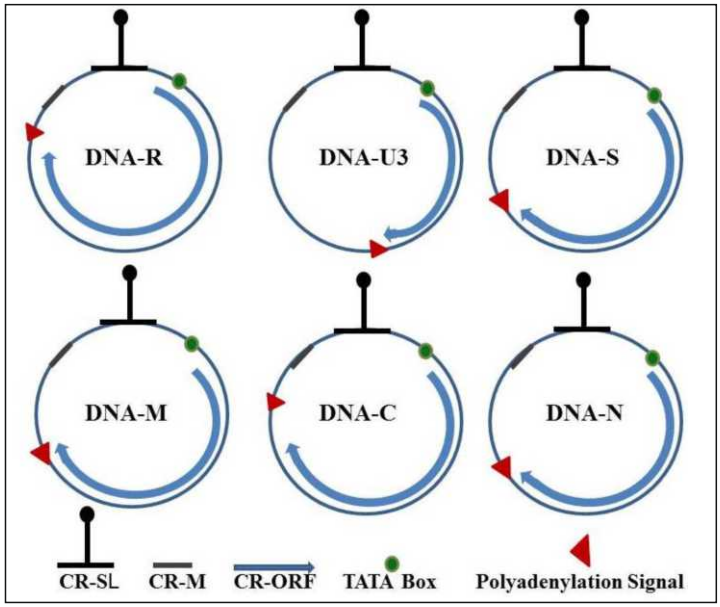

Figure 2: Multipartite component of BBTV

The exact role of DNA-U3 gene product and DNA-R encoded by the small internal ORF are unknown [40, 45], Multiple satellite DNAs known as deficient DNA-R components [46], encode non-essential Reps. which are capable of autonomous replication. However, it cannot trans-replicate any BBTV genomic component and depends on a helper virus to prepare cell conditions optimal for replication and movement within and between plants [47.]. The IR of each genomic component comprises a stem-loop common region (SL-CR), a major common region (CR-M), a TATA box and a polyadenylation signal [40] (62 % homology was found among all six genomic components in 69 bp SL-CR [40], and contains a stem-loop (SL) structure which contains the nano nucleotide loop sequence (5' TATTATTAC 3') and conserved among all components [40, 48] and iterative elements (iterons) that are potential Rep binding sites [49]. The CR-M varies in size from between 62 and 92 bp and shares at least 76% homology between all six genomic components [40]. The CRM contains three domains (domain I, II, and III), and short primer sequences that map to this region (5' of CR-M, domains I and II) have been isolated from BBTV virions [50], indicating its role in second strand synthesis of circular ssDNA genomic components.

STRATEGIES ADOPTED FOR THE DEVELOPMENT OF A TRANSGENIC VARIETY

Pathogen-derived resistance (PDR): It first postulated the concept of PDR in plants and suggested that the transgenic expression of pathogen sequences might interfere with the replication of the pathogen itself [50]. Substantiated the PDR concept and developed the theory of parasite or pathogen-derived protection using transgenic plants [51]. Pathogen-derived resistance mechanism deals with the expression of viral genes. The first practical concept of PDR against plant viruses was proposed and showed that the expression of tobacco mosaic virus (TMV) coat protein in tobacco plants has protected those plants against TMV [52]. For a successful generation of PDR following mechanisms are adopted viz., Full-length or truncated, wild-type or mutated viral sequences in sense, anti-sense or inverted repeats [53]. To generate PDR against geminiviruses, genes that encode the Rep, CP, MP, nuclear shuttle protein (NSP), and the replication enhancer protein (REnP) were used [54, 55]. Non-coding regions are also used sometimes for this purpose [21]. Present that basic molecular mechanisms deal with PDR based on protein-mediated resistance in which the expression of an unmodified or modified viral gene product interferes with the viral infection cycle and nucleic acid-based protection. PDR does not involve RNA-mediated resistance through the expression of protein products but resistance was conferred by modified viral transgenes which encoded untranslatable RNAs. This post-transcriptional mechanism operates at the RNA level and would therefore have the potential to suppress the accumulation of viral RNA that shares sequence identity with the silenced transgenes. However, it was reported that there was no correlation between the level of RNA accumulation and the degree of resistance [56].

PATHOGEN-DERIVED RESISTANCE WITH THE EXPRESSION OF VIRAL PROTEINS

Replicase-mediated resistance: Replicase protein-mediated resistance against a virus in the transgenic plant was first shown against Tomato mosaic tobamovirus (TMV) in tobacco plants containing a putative rep gene encoding 54 kDa replicase proteins [57]. The multifunctional replication-associated proteins (Reps) of viruses play an integral role in viral gene transcription regulation and the initiation and termination of virus replication. The Rep is thought to function as an oligomer and is possibly involved in the regulation of host gene expression, by interacting with host proteins involved in developmental and cell cycle regulation [58, 59]. Resistance developed for full-length, truncated, or mutated genes done by gene constructs of rep genes but this resistance is confined to a narrow spectrum of viruses. However, the resistance generated by the use of Rep sequences is very tight; a high dosage of input virus can be resisted easily by the transgenic plant. Similar resistance had been developed for several viruses' viz., Pea early browning virus (PEBV), Potato virus Y (PVY) [60], and Cucumber mosaic virus (CMV) [61]. In plants carrying a transgene derived from the replicase genes of Cowpea mosaic virus (CowMV) [62] and Pepper mild mottle tobamovirus (PMMV) [63]. It is evaluated that with the involvement of an RNA-mediated with homology-dependent resistance mechanisms.

Coat Protein (CP) mediated protection: In plant biotechnology use of viral CP as a transgene for producing virus-resistant plants was a great success. It was for the first time demonstrated virus-derived resistance in transgenic plants by using the cp gene of TMV [52]. He suggested that transgenic tobacco plants expressing a high level of TMV-CP were more resistant to TMV virions than to TMV-RNA. It was reported that CP-mediated protection against TMV was through the inhibition of virion disassembly in the initially infected cells. Hence, it was proposed that RNA inoculums could overcome the resistance because disassembly was not required to establish infection by naked RNA. The fabulous invention for the use of CP-mediated resistance to a virus is against papaya ringspot virus (PRSV). Transgenic papaya variety Sunset with cp gene was grown during the year 1991 to 1993 and found virus-free for 25 months. Subsequently, it was further crossed with other popular varieties such as Rainbow, which produced 11.2 t/ha marketable fruits compared to 5.6 t/ha from the non-transgenic lines [64].

Movement Protein-mediated protection (MP-MP): Movement proteins are required for long-distance systemic spread and have been used to develop resistance against various plant viruses. These proteins move from cell to cell and have been shown to modify the gating function of plasmodesmata and allowed virus particles or their nucleoprotein derivatives to spread to adjacent cells69. The main difference between CP-mediated protection and MP-MP is that the first one involves the expression of wild-type cp gene while the other is based on the transgenic expression of dominant negative mutant forms of viral genes. It was reported that transgenic expression of dysfunctional MP conferred resistance to TMV-MP which is conferred by transgenic expression of a dysfunctional TMV-MP is likely due to competition for the binding of plasmodesmata sites between the mutant MP and the wild-type MP of the inoculated virus [65]. MP-mediated protection deals with broad-spectrum resistance. The protection conferred by the mutant MP of TMV mediates resistance to viruses of the pots, cucumo, and tobraviral groups in addition to the targeted tobamoviruses [66]. This suggested that MPs of different viruses might interact with the plasmodesmata components [67]. Initially, transgenic plants expressing the defective movement protein were resistant to both Tobacco mosaic virus (TMV) and Cabbage leaf curl virus (CabLCV) especially since the proteins share 80% amino acid sequence similarities. In a similar experiment, tomato plants transformed with a mutated Bean dwarf mosaic virus (BDMV) mp gene showed resistance to tomato mottle virus (ToMoV) virus with a movement protein sharing 93% amino acid sequence identity with that of BDMV [68]. Hence, the use of MP Transgenes has a disadvantage in that they are often toxic when over-expressed in plant cells and are known pathogenicity determinants. Their uncontrolled expression has many undesirable effects on various aspects of plant development [68, 69].

CHRONOLOGICAL STUDIES FOR EXPRESSION OF VIRAL PROTEINS

Table 3: Expression of viral proteins

Sl. No.	Source (Transgenic Sequence)	Transgene	Plant	Viruses under investigation	Reference
1	Tomato yellow leaf curl Sardinia virus	Truncated REP gene	*Solanum lycopersicum*	Tomato yellow leaf curl Sardinia virus (TYLCSV)	[70]
2	Bean golden mosaic Virus	rep gene mutants	*N. benthamiana*	Bean golden mosaic virus (BGMV)	[3]
3	African cassava mosaic virus	*rep* gene mutant	*N. benthamiana*	African cassava mosaic virus (ACMV)	[71]
4	Tomato leaf curl New Delhi virus	Truncated REP gene	*N. benthamiana*	Tomato leaf curl New Delhi virus (ToLCNDV); African cassava mosaic Virus *(ACMV);* Potato yellow and Mosaic virus *(*PYMV)	[72]

5	Tomato yellow leaf curl Sardinia virus Truncated REP gene	Truncated REP gene	*N. benthamiana*	Tomato yellow leaf curl Sardinia virus (TYLCSV) and Tomato yellow leaf curl virus (TYLCV)	[73]
6	Tomato yellow leaf curl virus	Truncated REP gene	*Solanum lycopersicum*	Tomato yellow leaf curl virus (TYLCV)	[74]
7	Mungbean yellow mosaic virus	Full-length and truncated REP genes	*N. benthamiana*	Mungbean yellow mosaic	[51]
8	Maize streak virus	*rep* gene mutants and truncated rep gene mutants	*Digitaria sanguinalis*	Maize streak virus (MSV)	[75]

PATHOGEN-DERIVED RESISTANCE WITHOUT PROTEIN EXPRESSION

Antisense technology: Development of conceptual models of post-transcriptional gene silencing (PTGS) and Pathogen derived resistance to viruses, suggested mechanisms for suppressing the accumulation of nucleus-derived RNA (gene silencing) and virus-derived RNAs with homology to the transgene. Such a mechanism would require a higher degree of sequence specificity because they are highly strain specific. The principle behind this is interaction leading to suppression of viral RNA may involve base pairing of the sense RNA transcript of the transgene and the negative strand of the viral RNA, which is produced as an intermediate during the replication cycle of most viral RNAs. RNA-dependent RNA polymerase (RdRP) is used for the production of antisense RNA encoded in the host genome using the transgene RNA as a template [76] and would have the potential to base pair with the transgenic and viral RNAs. The formation of duplex RNA can influence the accumulation of host and viral RNA to cause gene silencing and virus resistance in plants. The base-paired region may render the duplex RNA susceptible to degradation by RNases specific for double-stranded RNA [77]. It could also arrest the translation and consequently have an indirect effect on RNase susceptibility and translation could cause reduced accumulation of both nucleus and virus-derived RNAs. The response of homology-dependent resistance is due to the involvement of antisense RNA this strategy has been successfully exploited since 1991 to target and selectively suppress the expression of viral genes. The transcripts targeted have included those of tobacco mosaic virus (TMV) rep in tomato yellow leaf curl virus (TYLCV) rep in N. benthamiana and tomato [78, 79], tomato leaf curl virus (ToLCV) rep in tomato [78], tomato golden mosaic virus (TGMV) and beet curly top virus (BCTV) rep, TrAP and REn (the last of which encodes a replication enhancer protein in begomoviruses in tobacco [80], BGMV rep, TrAP, REn, and MP in beans [81] and ACMV REP, TrAP and REn in cassava [82]. The successful exploitation of this approach depends directly on base-pairing between target and antisense RNAs and will therefore work only against viruses closely related to the virus from which the transgene was derived. For broad-based resistance, multiple sequences might have to be targeted and this could be achieved using fused antisense RNA sequences to different parts of the viral genome [55].

Table 4: Antisense technology reported by different workers

Sl. No.	Source of transgenic Sequence	Transgene	Plant	Viruses under investigation	Reference
1	Tomato golden mosaic virus	REP	*N. tabacum*	Tomato golden mosaic virus (TGMV)	[83]
2	Tomato golden mosaic virus	REP, TrAP and REn	*N. tabacum*	Tomato golden mosaic virus (TGMV)	[80]
3	Tomato yellow leaf curl virus	REP	*N. benthamiana*	Tomato yellow leaf curl virus (TYLCV)	[78]
4	Bean golden mosaic Virus	REP, TrAP, REn and MP	*P. vulgaris*	Bean golden mosaic virus (BGMV)	[81]
5	Cotton leaf curl Kokhran virus	REP, TrAP and REn	*N. tabacum*	Cotton leaf curl Kokhran virus (CLCuKV)	[84]
6	Tomato yellow leaf curl virus	REP	*S. lycopersicum*	Tomato yellow leaf curl virus (TYLCV)	[39]
7	Tomato leaf curl Virus	REP	*S. lycopersicum*	Tomato leaf curl virus (TLCV)	[85]
8	African cassava mosaic virus	REP, TrAP and REn	*Manihot esculenta*	African cassava mosaic virus (ACMV)	[82]

RNAi (RNA interference) mediated resistance: Sequence-specific RNA degradation described as post-transcriptional gene silencing in plants, quelling in fungi, and RNA interference in nematodes. RNA silencing is an evolutionary mechanism protecting cells from pathogenic RNA and DNA, which is adopted as an adaptive immune system of plants against viruses [86]. It demonstrated viral protection through the use of untranslatable viral RNA for sequence-trigger-specific, post-transcriptional RNA degradation of the mRNA. PTGS is a high-level nuclear transcription determined from nuclear run-on experiments. By plotting with northern hybridization it was found that there were low and steady-state levels of mRNA accumulation in the cytoplasm which confers that the gene silencing system is triggered in the cytoplasm together with any viral RNA that has the same or similar sequence. Presently, there were many examples of functionally operated by PTGS which involve resistance based on the expression of viral genes such as antisense, coat-protein, replicase, sense co-suppression, satellite RNA, and ribosome. RNAi can be effectively used for geminiviruses, initially for Mungbean Yellow Mosaic Virus-Vigna (MYMV-Vig.) [87] and later for African Cassava Mosaic Virus (ACMV) [88]. It was produced in transgenic tobacco plants with constitutively expressing double-stranded (ds) RNA cognate to coding and non-coding regions of DNAb from Cotton Leaf Curl Virus (CLCuV) [89]. They reported that plants were resistant to CLCuV when challenged with agro-inoculation or white flies. PTGS used in the development of resistance against the viruses ACMV [55, 88, 90], Mungbean Yellow Mosaic Virus (MYMV) [87], Sri Lankan Cassava Mosaic Virus (SLCMV), East African Cassava Mosaic Virus (EACMV) [90], TYLCV [91, 92, 93], Bean Golden Mosaic Virus (BGMV) [94] and TLCV [95].

Recent BBTV-resistant banana clone development through RNAi technology: RNAi strategy has proved effective in the case of bananas to develop resistance to banana bunchy top disease. RNAi-based resistance was introduced into susceptible cultivar of banana (cv. Rasthali), wherein the master replication protein gene 'Rep' full coding sequence or 'Rep' partial coding sequence together with its 5' partial upstream regulatory region of the BBTV, was used to generate RNAi vector and successfully transformed into banana [8]. These plants were resistant to BBTV up to 6 months post-inoculation with viruliferous aphids. Moreover, RT-PCR failed to detect any BBTV-specific transcripts in these transgenic lines. A similar study was carried out using gene fragments from the four BBTV DNA segments namely DNA 1, DNA 3, DNA 4, and DNA 5 to generate RNAi vectors. The transgenic banana plants (cv. Grand Naine) generated showed delayed viral multiplication and development of symptoms [96]. It is very important to recognize a suitable target sequence and later to identify a transgenic line that expresses correct siRNA in proper amounts and is able to resist the virus. It was reported that some transformed clones of 'Dwarf Brazilian' (AAB, Pome subgroup) were resistant to BBTV under experimental conditions in Hawaii. Two Indian research groups have claimed success [97].

Table 5: Chronological success in Gene silencing

Sl. No.	Source of transgenic Sequence	Transgene	Plant	Viruses under investigation	Reference
1	African cassava mosaic virus	Chemically synthesized siRNA to REP mRNAa	*N. tabacum protoplasts*	African cassava mosaic virus(ACMV)	[88]
2	Mungbean yellow mosaic virus	DNA-A bidirectional Promoter	*Vigna mungo*	Mungo yellow mosaic virus (MYMV)	[87]
3	African cassava mosaic virus	REP gene (transgeneinduced PTGS)	*Manihot esculenta*	African cassava mosaic virus(ACMV), Sri Lankan cassava mosaic virus (SLCMV) and East African cassava mosaic virus (EACMV)	[90]
4	Tomato yellow leaf curl virus	Hairpin RNA derived from REP genea	*S. lycopersicum and N. benthamiana*	Tomato yellow leaf curl virus (TYLCV)	[91]
5	Tomato yellow leaf curl virus	Hairpin RNA Derived from REP gene	*S. lycopersicum*	Tomato yellow leaf curl virus (TYLCV)	[92]
6	African cassava mosaic virus	DNA-A bidirectional Promoter	*M. esculenta*	African cassava mosaic virus (ACMV)	[55]
7	Tomato yellow leaf curl virus	Hairpin RNA derived from COAT PROTEIN gene	*S. lycopersicum*	Tomato yellow leaf curl virus (TYLCV)	[93]
8	Bean golden mosaic Virus	Hairpin RNA derived from REP gene	*Phaseolus vulgaris*	Bean golden mosaic virus (BGMV)	[94]
9	Tomato yellow leaf curl virus	Hairpin RNA derived from REP and AC4 genes	*S. lycopersicum*	Tomato yellow leaf curl virus (TYLCV)	[95]
10	Banana Bunchy top virus	REP	*Banana (Musa spp)* cultivar.Dwarf Brazilian	Banana Bunchy top virus (BBTV)	[97]
11	Banana Bunchy top Virus	REP	*Banana (Musa spp)* cultivar. Rasthali (AAB)	Banana Bunchy top virus (BBTV)	[8]
12	Banana Bunchy top virus	Hairpin RNA derived from REP	Hill Banana (*Musa* spp) cultivar.Virupakshi (AAB)	Banana Bunchy top virus (BBTV)	[13]

RESISTANCE DUE TO THE EXPRESSION OF NONPATHOGENIC-DERIVED ANTIVIRAL AGENTS

Virus-induced cell death: Infected plants often have an innate defensive hypersensitive reaction that limits virus movement to the site of infection by inducing the death of infected cells and their neighbors. This type of reaction can be artificially induced to provide resistance against viruses in transgenic plants by the combined action of barnase and barstar proteins of Bacillus amyloliquefaciens [55, 82]. Barnase is a ribonuclease (RNase) and barstar is its inhibitor. In the absence of virus infection, the two transgenes expressed at similar levels resulted in the absence of RNase production. By placing barnase under the control of a viral virion-sense promoter that is activated during virus infection and barnstar under the control of a viral complementary sense promoter that is repressed during virus infection, an infected cell should over-express barnase relative to barnstar and die before the infecting virus proceeds for the further stage.

DNA binding proteins: The use of transgenically expressed DNA binding proteins to provide virus resistance relies on the identification of virus sequence-specific binding proteins that will not bind host DNA sequences. The virus Rep is a sequence-specific dsDNA binding protein 31 that recognizes and binds to direct repeats in the virion strand origin of replication (v-ori) where it initiates and terminates rolling circle replication [98, 99, 100]. This sequence-specific activity has been exploited by designing artificial zinc finger proteins with high affinity for the Rep-specific direct repeats in the v-ori of different geminiviruses [101], with the idea that the artificial zinc finger proteins will competitively block the binding of Rep due to the higher affinity of the artificial zinc finger protein–dsDNA interaction. This resulted from the inhabitation of viral replication. Successfully demonstrated this approach in Arabidopsis thaliana against beet severe curly top virus (BSCTV) [102].

CONCLUSION

Banana bunchy top virus is threatening banana growers so the time reaches to move forward with the adoption of modern biotechnological tools such as tissue culture technique and genetic transformation. Various methods of biotechnological tools help in creating resistant variety however further research needs in this direction to develop disease-free planting materials and create awareness about BBTV knowledge. There is a need to create awareness to popularize genetically modified plants/variety resistance to viral diseases such as bunchy top in bananas and other destructive diseases in various fruit plants. It is not only to reduce the cost of cultivation by minimizing/reducing plant protection costs but also a tool for eco-friendly management of disease control. It is helpful for the sustainable development of agriculture and horticulture as well as for the promotion of organic cultivation of bananas and other fruit crops with similar technological adoption in other crops. Now, time to come forward to adopt these new technologies for the betterment of human life and the increase of productivity and profitability of the farmer's income from limited farm resources.

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