Occurrence and Characteristics of a Seed-Transmitted Potyvirus from Indian, African, and North American Guar

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ABSTRACT

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Fifty seed lots of guar (*Cyamopsis tetragonoloba*) from India, Pakistan, the former Belgian Congo, and the USA contained a virus which induced characteristic red lesions on *Chenopodium amaranticolor*. Three isolates from India, Africa, and the USA showed similar flexuous-rod morphology with a normal length of 761 nm and similar pinwheel and scroll-type inclusion structures in the electron microscope. Physical properties and herbaceous host range also were similar for these isolates. The virus belongs to the potato virus Y (potyvirus) group, and can be differentiated from most other seed-transmitted legume viruses. Symptoms on *Chenopodium amaranticolor* and the host range differ from those of the other three legume potyviruses with identical inclusion bodies and indicate that the guar virus is different from them. Low virus concentration in all hosts and continuous loss during purification prevented serological determination of relationships. The virus presumably has been imported into North America with its guar host. Since naturally infected and inoculated guar plants remained symptomless, it tentatively has been named "guar symptomless virus" (GSV).

Guar [*Cyamopsis tetragonoloba* (L.) Taub.] (syn. *C. psoraloides* DC) originates in India (29) where it is cultivated mostly as a vegetable and fodder plant. It is grown in Africa for similar purposes but on a smaller scale. During the 1800's it was introduced into the southern USA where today approximately 200,000 hectares (ha) are grown annually. Most of the North American crop is used as a base for gum and lacquer production. At least three naturally-occurring guar viruses are included in a bibliography of guar diseases (20).

Guar is used as an indicator for viruses such as soybean mosaic (28), potato virus S (43), sunn-hemp (30, 37), and others (32, 36). When we used guar as a potential local-lesion indicator for cherry rasp leaf virus (14), we incidentally encountered a seed-transmitted virus in several seed lots from the USA.

Initial tests (13) showed that the properties of this virus were different from those of other guar and legume viruses. The present study was therefore undertaken to determine the natural occurrence of the guar virus in seed lots from different continents, to establish its characteristics, and to detect possible relationships to other viruses.

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MATERIALS AND METHODS

Inoculations from guar plants and seeds routinely were carried out by grinding tissue in five parts (w/v) of 0.01 M phosphate buffer, or in 1% nicotine (alkaloid), and applying the resulting suspension to four fully expanded Carborundum-dusted leaves of test plants. *Chenopodium amaranticolor* Coste et Reyn. reacted with characteristic red lesions and therefore was used as a local-lesion indicator for all routine assays and attempted reisolations of virus from inoculated plants. For general seed transmission tests, 10 germinating seeds per cultivar were ground in five parts (w/v) nicotine buffer and indexed together. For the determination of approximate seed transmission percentages, 25 seeds of three cultivars were tested individually by the same method. Tests were conducted with 50 random samples from three different sources: (i) six commercial cultivars from Texas and Oklahoma, USA; (ii) twenty plant introductions from India, Pakistan, the former Belgian Congo, and the USA which had been maintained for at least 7 yr under P.I. numbers at the U.S. Department of Agriculture Plant Introduction Collection at Experiment, Georgia; and (iii) twenty-four cultivars and breeding lines obtained directly from India. Single-lesion virus isolates from cultivars CP 31 (India), P.I. 263406 (Plant Introduction from the former Belgian Congo) and Brooks (USA) were selected for a determination of the thermal inactivation point (10

min), dilution end-point, and seed transmission percentage. Thermal inactivation and dilution end-point were determined by using inoculated leaves of *C. amaranticolor* and *Chenopodium quinoa* Willd. as virus sources. Immediately after the treatment, infectivity was assayed on four fully-expanded leaves of *C. amaranticolor*. Each test was repeated three times. Host-range studies were conducted with the same three isolates in insect-proof greenhouses during spring, summer, and fall. All plants were grown in sterilized soil and temperatures were maintained between 18 C and 23 C. Groups of four plants were inoculated at the four-leaf stage with infective crude juice from *C. amaranticolor* and symptoms were recorded at 4-day intervals. Inoculated and noninoculated leaves were back-indexed separately on *C. amaranticolor*. Tests giving negative results were repeated at least once.

We purified virus from inoculated leaves of *C. quinoa*, injected the virus into rabbits, and tested the resulting sera according to the method of Wetter (40), which had been used with good success for potato virus Y and other viruses of that group. The method essentially consisted of butanol-chloroform clarification, concentration by centrifugation and sucrose density gradients, dialysis, two intramuscular injections of 3 ml each with virus and incomplete Freund's adjuvant, 10 days apart, into each of two rabbits, and tests of the resulting sera on glass slides. We deviated from Wetter's method in grinding the infected leaves with a meat grinder instead of a fruit press, and in assaying after each purification step on *C. amaranticolor*, instead of serologically.

The electron microscopical detection of virus particles was done with crude *C. amaranticolor* and guar sap negatively stained with 2% neutral sodium phosphotungstate. For measurements of particle dimensions, the negatively-stained preparations were mixed with crude sap from tobacco mosaic virus (TMV)-infected *Nicotiana tabacum* L. 'Samsun'. The length of the TMV particles which served as length standard was assumed to be 300 nm. The method of Govier and Woods (12) was used to determine the effect of Mg^{++} ions on particle length. Ultra-thin sections were made from pieces of leaves of infected and healthy guar and of *C. amaranticolor* after embedding in Epon [for details, see (23)].

RESULTS

Detection, occurrence, and seed transmission.—On *C. amaranticolor*, all 50 seed lots tested consistently induced lesions which appeared on the 4th day after inoculation as chlorotic spots, and turned bright red on approximately the 10th day. Uniformity and specificity of lesion development served to distinguish the virus under study from other viruses occasionally encountered in guar seeds. The percentage of seed transmission varied from 12% in cultivar Brooks to 28% in P.I. 263406. Twenty-two plants of cultivar Brooks and 24 plants of P.I. 263406 grown from these partially-infected batches to flowering stage under greenhouse conditions remained uniformly symptomless. Similarly, when 14 indexed healthy guar plants of three cultivars were inoculated with the Brooks isolate, none developed any symptoms, but bioassays on *C. amaranticolor* showed that 11 were systemically infected. Mottling or upward leaf rolling observed in a few individual plants of other cultivars could not be linked with infection by the virus under study.

Host range and physical properties.—Stymptoms and host range were identical for the three isolates studied in detail and corresponded to those obtained previously (13) with three USA isolates. The infection remained local on most hosts. Lesions on *C. amaranticolor* began to appear on the 4th day as light-green rings, turning red on approximately the 10th day. Occasionally, they were followed by a partial systemic mottle. *Chenopodium album* L., *C. murale* L., *C. quinoa* (three cultivars), *Cyamopsis tetragonoloba*, *Vigna sinensis* L., and *Phaseolus vulgaris* L. (three cultivars) generally remained symptomless, except for occasional development of faint green lesions on *C. album* and *C. quinoa*, as well as systemic mottle on *C. quinoa*. Bioassays from inoculated leaves and from leaves with systemic mottle to *C. amaranticolor* confirmed that infection had taken place. The following plant species could not be infected with any of the three isolates: *Chenopodium foetidum* Schrad., *Cucumis sativus* L., *Cucurbita maxima* Duchesne,

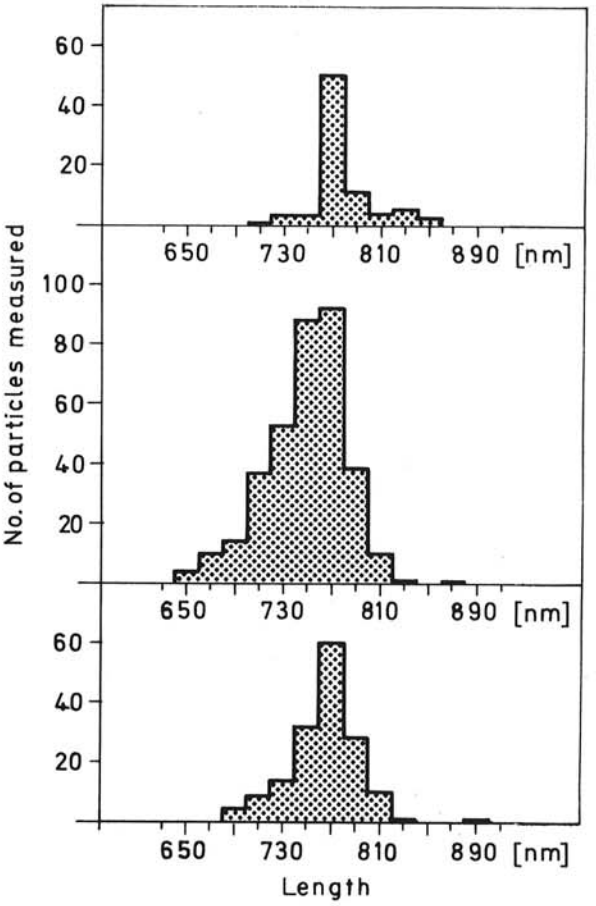


Fig. 1. Histograms of particle-length distributions in crude sap of three different sources of guar symptomless virus (GSV): upper: American guar cultivar Brooks; middle: Indian guar CP 37; lower: Indian guar supplied by V. S. Verma.

Lycopersicon esculentum L., *Nicotiana tabacum* L., *N. clevelandii* Gray, *Pisum sativum* L., and *Vicia faba* L.

In the heat inactivation experiments, the average number of lesions produced on *C. amaranticolor* remained constant at approximately 18 (cultivar Brooks and P.I. 263406 isolates) and 15 (CP 31 isolate) up to 52 C. At 54 C, corresponding numbers of lesions were 2.2, 1.2, and 1.5. At 56 C, the 'Brooks' isolate induced 0.75 lesions, the CP 31 isolate induced 1.0 lesion, and no lesions were induced by the P.I. 263406 isolate. No lesions were observed at 58 C with any of the isolates. The dilution end-point varied with concentration of the inoculum and generally reached 10^{-2} to 5×10^{-2}. In one case, that of the Brooks isolate reached 10^{-3}.

Repeated attempts to increase the virus concentration in test plants by means of weekly transfers from inoculated leaves failed. Maximum number of lesions per inoculated leaf of *C. amaranticolor* rarely exceeded 25, and only slightly higher numbers of lesions were obtained when germinating guar seeds or inoculated leaves of *C. quinoa* were used as inoculum.

Continued bioassay monitoring of virus during purification attempts indicated that concentration increased only two- or threefold compared to crude infective juice. In the density gradients, a 2-mm band with approximately threefold the infectivity of crude juice was detected by assay on *C. amaranticolor*. This band was used for the injection of the two rabbits. However, none of the final sera displayed any serological activity against the virus.

Particle morphology and structure of inclusions in host cells.—Negatively stained crude sap from infected guar contained elongated flexuous particles with a normal length of 761 nm and a width of about 12 nm. This value was calculated from measurements of 660 particles obtained in 14 separate preparations taken from six different guar plants. Figure 1 illustrates the length distribution of three different guar virus sources. All three have indistinguishable particle length. Particle-length averaged 775 nm after addition of Mg^{++} ions to the crude sap, whereas 748 nm were measured after addition of EDTA to the crude sap. The Mg^{++} effect, if significant at all, thus was much smaller than that reported for some other potyviruses (12, 19). Crude sap from lesions of *C. amaranticolor* contained particles indistinguishable from those in guar.

Ultra-thin sections from infected guar and *C. amaranticolor* always revealed some cells with cylindrical inclusion bodies, which are considered to be characteristic of the potyvirus group (7). The inclusion bodies contained only pinwheel and scroll elements (Fig. 2, 3); inclusion elements of the laminated type were not detected. A further conspicuous component of the inclusion bodies was small vesicles containing fibrous material. These were derived from dictyosomes which often were present in an apparently active state in the region of the inclusion bodies (Fig. 2). An aggregate of fibrillous material that might represent aggregated virus particles was found in an infected cell on one occasion (Fig. 3). In infected tissues of *C. amaranticolor* and of guar, abnormally large mitochondria occasionally were observed and in infected guar unusual cell wall proliferations were found (Fig. 4, 5, 6). Ultra-thin sections from indexed virus-free guar and from noninoculated *C. amaranticolor* contained no inclusion bodies, enlarged mitochondria, or cell wall proliferations.

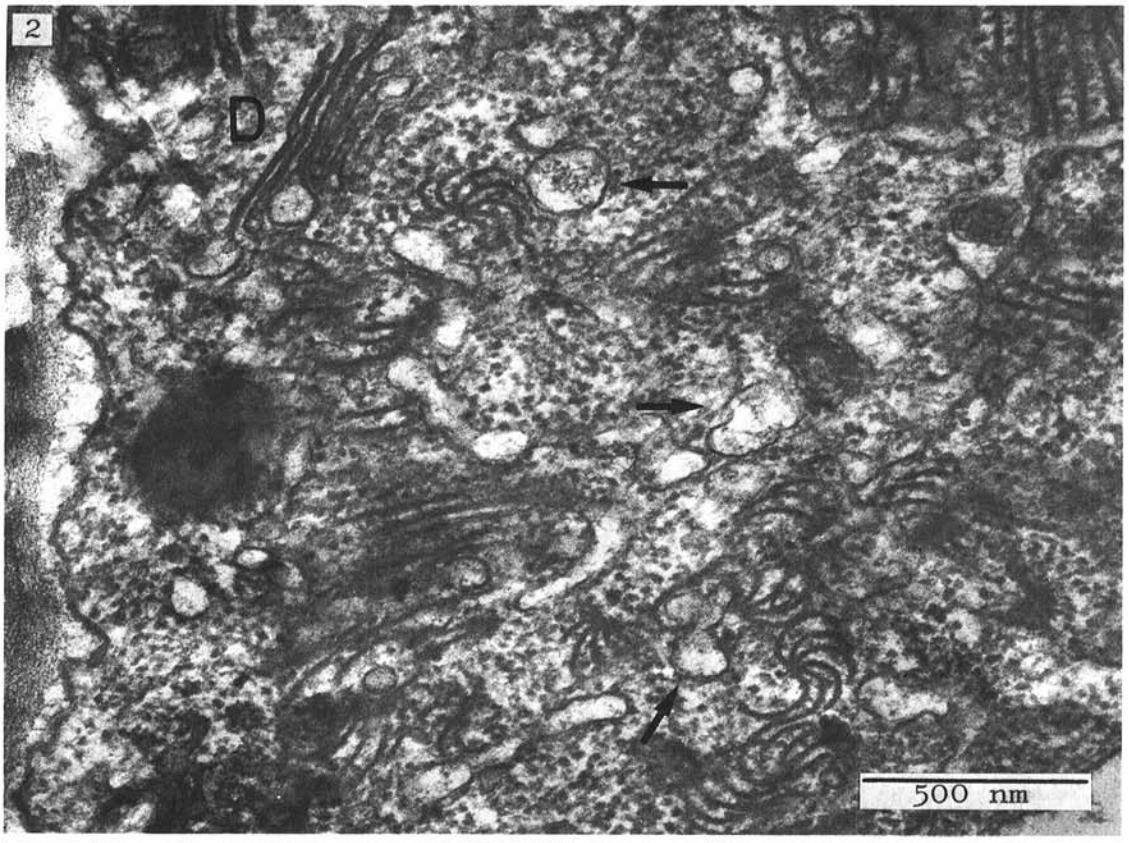
DISCUSSION

The data obtained show that a virus could be isolated from all 50 guar seed samples. All isolates induced red lesions on the inoculated leaves of *C. amaranticolor*. Since Hollings (16) has proven the usefulness of this host for identifying plant viruses, and since the red lesions were specific, uniform, and unlike those induced by other viruses on *C. amaranticolor*, we concluded that all isolates are of the same virus. This conclusion was corroborated by the finding that the three extensively studied isolates from India, Africa, and the USA had identical host ranges as well as similar physical properties, and that infected plants always contained flexuous rod-like particles of identical length and pinwheel and scroll-type inclusions.

The guar virus differs in particle morphology from tobacco ringspot virus which causes the guar top necrosis disease in the USA (5, 22, 39). On the basis of the sparse published data, it is impossible to say whether the guar virus may be related to one of the agents causing the mosaic and streak diseases (3, 31, 38) occasionally reported from the USA. The same holds true for possible relationships to several seed-transmitted legume viruses from India (6, 26, 36), which are incompletely studied and which have not been reported to occur naturally in guar.

Particle morphology and induction of pinwheel and scroll-type inclusions place the guar isolates into the potyvirus group (7). Although pinwheels and scrolls are reported to be induced in some allegedly virus-free callus cultures by medium modification (41, 42), they nonetheless generally are accepted as virus-specific taxonomic criteria when they occur in differentiated plants infected with potyviruses (2, 7, 11). Classification of and relationship between legume viruses within the potyvirus group are somewhat uncertain (2, 7, 9, 10, 15, 18, 27, 33, 34). Host reactions, physical properties, serology, and particle properties have been employed, but often have given variable or contradictory results. Because of this variation, and because most legume potyviruses occur naturally in a number of strains, their classification by any of these characteristics has little or no bearing on their pathogenic properties (2, 18).

With these reservations in mind, we have compared the inclusion bodies, host responses, and other characteristics of the guar virus with those of other potyviruses. Table 1 illustrates some of the differences. Since it has been repeatedly demonstrated by Edwardson (7) and others (4, 10, 17, 21) that the structure of the inclusion bodies permits differentiation of potyviruses, we have used this criterion to distinguish the guar virus from most other seed-transmitted legume potyviruses. The characteristic pinwheel and scroll inclusions differentiated the guar virus from pea mosaic, pea seed-borne mosaic, soybean mosaic, and western bean mosaic virus. This differentiation is corroborated by differences in the host range and symptom expression on herbaceous hosts (8, 25, 28, 35). However, inclusion body characteristics did not allow differentiation within Edwardson's subdivision I, which also contains bean common mosaic, cowpea aphid-borne mosaic, desmodium mosaic, and azuki bean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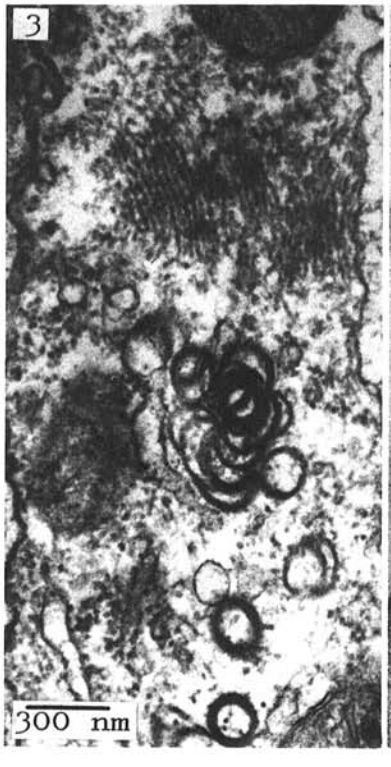
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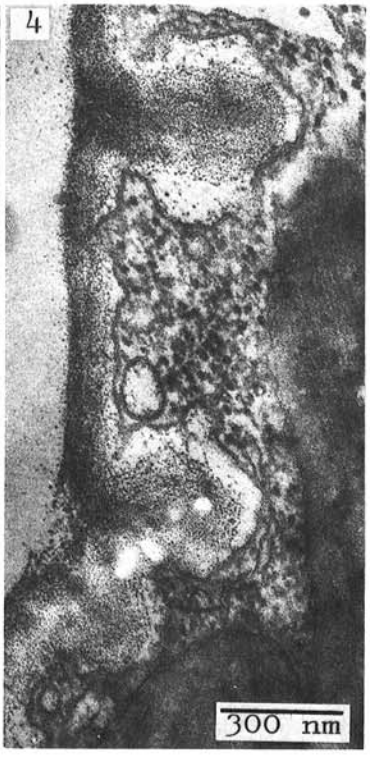
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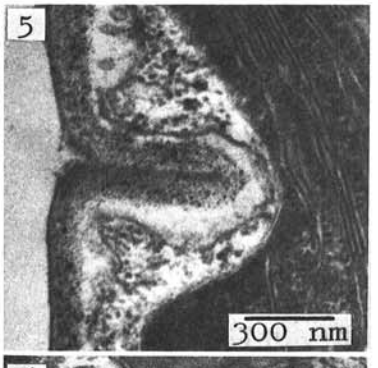
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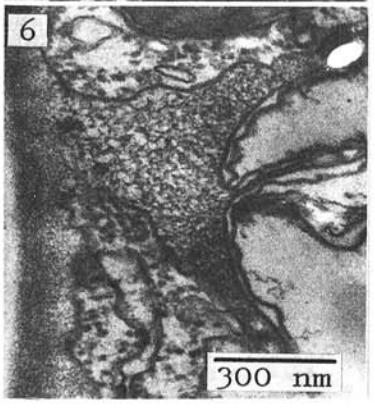
6
300 nm

TABLE 1. Host response and physical properties of guar symptomless virus and other seed-transmitted legume viruses of the potyvirus group which induce pinwheel- and scroll-type inclusions

Characteristics[a]	Virus disease symptoms[b]				
	Guar symptomless	Azuki bean mosaic	Bean common mosaic	Cowpea aphid-borne mosaic	Desmodium mosaic
Host response					
Chenopodium album	0/+				
C. amaranticolor	r L		lg L to 0	lg or n L	0
C. quinoa	0/+		lg L	L	
Cyamopsis tetragonoloba	0/+		0 to M/+		M
Phaseolus vulgaris	0/+ to 0/− S: 0/−		M to n L	lg L to n L	0
Pisum sativum	0/−		0/+	0 +	
Vicia faba	0/−	r L		S: M or 0 +	
Vigna sinensis	0/+		L to M/+	S: M	
Thermal inactivation point (C)	54	55-60	56-58	57-60	
Dilution end point	5×10^{-2}	10^{-4}	10^{-3} 10^{-4}	10^{-3} 10^{-4}	
Particle length	761 nm	750 nm	750 nm	758 nm	775 nm

[a]Symptoms are those of inoculated leaves unless prefixed by S:
[b]Abbreviations: lg = light-green, r = red, n = necrotic, M = mottle, L = local lesions, O = no symptoms, S = systemically-infected leaf, /+ = backcheck-positive, and /− = backcheck-negative.

mosaic viruses, all of which seem to be related to each other and to the guar virus. The members of this subgroup can be distinguished from each other on the basis of host response: aphid-borne cowpea mosaic virus infects many species which are immune to the guar virus (24), and bean common mosaic virus and desmodium mosaic virus do not induce the red lesions of *C. amaranticolor* which are characteristic for the guar virus. A meaningful comparison with adzuki bean mosaic is not possible, since only few data have been published for this virus. The lack of a suitable purification method for the guar virus prevented a check of the serological relationships to the other members of this subgroup.

On the basis of the available information, we conclude that all our isolates are of one virus. Our data indicate that this virus may be undescribed, but further work would be needed to establish this beyond any doubt. For practical purposes, we suggest the designation "guar symptomless virus" (GSV) until serological or other relationships to other viruses may be established.

Although the 50 samples tested are not necessarily representative for all guar-growing areas of the world, it seems likely that GSV is common wherever guar is grown. It seems reasonable to assume that it has been introduced into the USA with infected seed from its country of origin, India. Since our data indicate that most guar seeds remained virus-free even though they came from commercially grown field stock, it should be relatively easy to produce completely virus-free stock. Whether this is economically desirable would have to be determined in areas where guar is grown commercially, and where field losses from infection and natural spread (presumably by aphids) can be determined. The advantages of using virus-free guar as local lesion hosts for other viruses are considerable, because transfers from infected plants would invariably cause contaminations such as those which led to the initiation of these studies.

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