RESISTANCE TO VIRUS DISEASES OF SOME F_1 CLONES DESCENDED FROM THE SPECIES HYBRIDIZATION *SOLANUM DEMISSUM* x *S. TUBEROSUM*

ONNI POHJAKALLIO and LAURA KARHUVAARA

Department of Plant Pathology, University of Helsinki

Received January 11, 1960

The species hybridization *Solanum demissum* Lindl. x *S. tuberosum* L. (Rosafolia) carried out in 1947 (7) gave rise to a potato plant forming tubers with a purple skin. Most of the progeny of this plant continued to form purple tubers, but some, as the result of bud mutations, formed tubers with a white skin. Bud mutations also lead to changes of other kinds in the properties of plants (3). One of the clones forming purple tubers appeared to be earlier in its development than the others. Some other symptoms suggested that the peculiar character of this clone was solely due to a virus infection (3, 10). Later, the early maturing character also emerged in many of the other F_1 clones forming purple tubers, while all the potato plants forming white tubers remained true to their type of late maturation. Consequently, it looked as though as a result of bud mutations, progenies resistant to a virus had been raised from susceptible potato plants.

Material

All the potato plants from the species hybridization *Solanum demissum* x *S. tuberosum* (Rosafolia) examined in the following are the vegetative progeny of one potato seedling grown in the summer of 1948. The skin of the tubers of this potato plant was purple; the whole of its appearance bore evidence of its hybrid origin. It and its vegetative progeny were frost-hardy (7, 11), resistant to potato blight (7, 8), and formed tubers later than the *S. tuberosum* varieties (10).

In 1949, this potato clone seemed to retain its original properties, but in 1950 some of the plants formed white tubers (3). The haulms of these colour mutants were very luxuriant at the end of the growing season (10), thus resembling the bolter type (cf. 5, 13). Only the tubers which were attached to the potato plant

were reserved for setting. The tuber yield of each plant was put into a separate paper bag, in which it was preserved over the winter. In the field experiments the tubers were planted at distances of 1.2 metres. Consequently, the tubers of the different potato plants were not mixed with each other.

The potato plants forming tubers with a white skin, which were evidently bud mutants of the original potato plant (cf. 3), also formed white tubers in all subsequent vegetative generations. Since the year 1950, however, none of the potato plants of the original type has given rise to further white tubers. On the other hand, many of the plants forming purple tubers changed with regard to the rate of their development becoming earlier. Because this phenomenon was assumed to be due to some virus disease, infection experiments and serological investigations were performed. For the serological investigations Prof. Dr. van Slogtern (Laboratorium voor Bloembollenonderzoek, Lisse, Holland) kindly sent normal sera as well as antisera against the potato viruses X, S, and Y. Attention was also paid to the symptoms of the disease. In addition, the length of the flowering time and the productivity of the potato clones were investigated.

Results

The potato plants forming tubers with a purple skin. As the result of bud mutations different kinds of clones were found among the F_1 material investigated, the plants of which developed purple tubers (10). The differences between the properties of these clones, however, were relatively slight, and therefore in what follows this material has been treated as a unit. A characteristic of all these clones was their tendency to be converted into early maturing types. To solve the question of whether this phenomenon also arose from a bud mutation or was due to a virus infection, core graftings were made. A piece of healthy tuber was removed with a cork borer, and into the hole was inserted a piece taken from a tuber of a plant of the early maturing type by means of a slightly larger cork borer. Graftings were carried out late in the winters of 1956 and 1959 and in autumn 1957. In the following summers grafted as well as ungrafted tubers were planted in field experiments. In 1956 and 1957 the field experiments were placed near the other potato stands. Consequently spontaneous virus infections were possible. In 1959, the potato plants were set in specially, isolated plots located at distances of ca. 50 metres from any other potato stands. The setting tubers for these latter experiments had also been cultivated in special isolation (in 1958). In spite of these precautions, nearly a fourth of the plants had spontaneously acquired the character of early maturity in 1959. However, in all the 3 field experiments the corresponding transformation appeared in larger numbers among the grafted than among the ungrafted plants (Table 1); the result, which appeared to be statistically significant, did not depend on whether the piece inserted at grafting carried a bud or not. Consequently, the phenomenon in question seemed to be an infectious disease. This opinion was supported by the facts that in field experiments in which the original and early maturing

Table 1. The results of core grafting infections

Year	Clone (colour of tuber)	Piece inserted	No. of tubers grafted	Percentage of plants infected	Distance from virus-diseased potato plants m.
1956	purple	—	15	20	10
—»—	—»—	carrying a bud	20	65	10
—»—	—»—	no bud	20	65	10
1957	—»—	—	33	36	10
—»—	—»—	carrying a bud	22	59	10
—»—	—»—	no bud	13	62	10
1959	—»—	—	50 [1]	24	50
—»—	—»—	no bud	49 [1]	100	50
1956	white	—	10	0	10
—»—	—»—	carrying a bud	20	0	10
—»—	—»—	no bud	20	0	10
1957	—»—	—	21	0	10
—»—	—»—	carrying a bud	20	0	10
—»—	—»—	no bud	21	0	10

1) For setting, the tubers were cultivated in specially isolated plots in 1958, when symptoms of early maturity were observed in one of the 50 plants; this plant was eliminated on August 4.

types of potato plants were cultivated at distances of only 1.2 metres from each other, transformation into an earlier maturing type of plant was more frequent than when the plants of the original type were cultivated in special isolated plots. In 1958, only one of the 50 potato plants of the special plot was transformed into an early maturing type, while the corresponding percentage in the mixed plot with both types was 58. In 1959, when the weather was exceptionally warm and rainless, the corresponding numbers calculated in percentages were 24 and 91.

The disease reported above was first observed in 1952. In 1952 and 1953 only a few diseased plants were found. In the years 1954 to 1959 the rates of new infections were as follows: 21 % in 1954, 49 % in 1955, 51 % in 1956, 57 % in 1957, 58 % in 1958, and 91 % in 1959. Because the disease appeared to be infectious, and without a single exception transmittable in the tubers to the following vegetative generations of the potato plant (7 generations investigated) the disease seemed to be caused by some virus.

Serological investigations showed that the viruses X and S were not present in the diseased potato plants. By contrast an agglutination test with antiserum against virus Y gave a positive result. It is true that in some cases the normal serum used also reacted positively with the sap of that plants.Thus the result cannot be

Table 2. The earliness of flowering of the F1 potato plants forming tubers with a purple skin.

Year	Date of setting	Date of lifting	Date of onset of flowering		Date of end of flowering	
			Diseased plants [1])	Healthy plants	Diseased plants	Healthy plants
1954	29/5	1/10	8/7	7/7	21/7	6/9
1955	7/6	26/9	13/7	13/7	1/8	20/8
1956	5/6	26/9	19/7	18/7	1/8	27/8
1957	29/5	20/9	9/7	10/7	28/7	20/9
1958	5/6	18/9	18/7	19/7	1/8	19/8
1959	15/5	19/9	5/7	6/7	14/7	12/8

[1]) Many plants did not flower at all. The haulms died early; the haulms of 93 % of the diseased plants died before the beginning of September, when the haulms of all the healthy plants were still alive.

Table 3. The effect of the date of lifting on the tuber yield of F_1 potato plants forming tubers with a purple skin

Condition of the plants	Yields of 6 potato plants (g)					
	1954		1955		1956	
	31/7	6/10	1/8	15/9	1/8	10/9
Healthy	79	2487	12	1163	7	517
Virus diseased	273	1585	21	590	43	815

Table 4. The relation between the number of vegetative generations during which the potato plants had had virus disease and the tuber yield (lifted late in autumn).

Results in 1956			Results in 1957			Results in 1958			Results in 1959		
Year of infection	No. of plants	Tubers g/plant	Year of infection	No. of plants	Tubers g/plant	Year of infection	No. of plants	Tubers g/plant	Year of infection	No. of plants	Tubers g/plant
1952	3	87	1952	3	145	1952	3	119	1952	3	59
1953	3	83	1953	2	256	1953	3	92	1953	3	46
1954	12	170	1954	3	202	1954	3	257	1954	3	86
1955	6	57	1955	7	75	1955	2	66	1955	3	11
1956	5	124	1956	6	257	1956	3	106	1956	5	39
Healthy	28	330	1957	13	536	1957	5	75	1957	9	30
plants			Healthy	10	526	1958	16	120	1958	5	55
			plants			Healthy	11	218	1959	31	247
						plants			Healthy	3	258
									plants		

considered quite conclusive. However, in addition to the results of the agglutination test the symptoms occurring in the diseased plants (leaflets a little wrinkled, and small, dark brown necrotic streaks on the under side of the leaflet) also pointed to the presence of virus Y. Consequently, in the following the disease has been considered to be the potato rugose.

The earliness of flowering of the potato plants was not affected by the virus disease (Table 2). By contrast, the length of the flowering time of the diseased potato plants was decreased relative to that of the healthy plants; besides, the haulms of the diseased plants turned yellow and died relatively early. When lifted early the tuber yield of the diseased potato plants appeared to be heavier than that of the healthy plants (Table 3). But at the time of late lifting the largest amount of tubers was yielded by the healthy potato plants.

The effect of the virus disease on the tuber yield did not always appear in the same summer in which the potato plants had contracted the virus infection (Table 4). This, perhaps, arose from the fact that the virus hastened the rate of development of the potato plants and in that manner partly compensated for the loss caused by the disease. In these experiments lifting was carried out late in autumn (cf. Table 2). Consequently, the tuber yields of the systemically diseased plants were generally much lower than those of the healthy plants. On the other hand the tuber yield appeared to be independent of the number of vegetative generations during which the potato plants had had the virus disease. The variations in the yields of the plants infected in the different years depend, in part at least, on the fact that the potato material included different clones the properties of which varied somewhat on account of bud mutations. In 1959, the material investigated included 21 potato clones forming purple tubers. All these clones appeared to be susceptible to the virus infection.

The potato plants forming tubers with a white skin. From the potato plants forming white tubers descended from the bud mutation, 4 clones were chosen in 1950. One of these was cultivated for a few years only. Later, in 1954 and 1955, however, 2 new clones were separated from the original clones. Consequently, altogether 5 clones forming white tubers were examined:

Table 5. The yields (g/plant) of the bud mutant clones forming tubers with a white skin, 1951 to 1959

Year	W 1	W 2	W 3	W 4	W 5
1951	841	233	1221		
1952	932	53	953		
1953	1773	556	1880		
1954	682	541	2283		
1955	975	222	1750	615	
1956	775	100	787	968	47
1957	1189	56	1036	770	55
1958	1115	260	1545	1225	13
1959	1388	1054	1751	845	332

W 1. Flowers dark blue.
W 2. Flowers dark blue; maturing later than clone W 1.
W 3. Flowers light blue.
W 4. Flowers light blue; maturing later than clone W 3.
W 5. Flowers light blue; maturing still later than clone 4.

Generally, the heaviest yields were obtained from the clones developing earliest. Especially in years when the temperature was relatively low (1956 to 1958) and the length of the growing season decreased, the slow developing clones yielded very little (Table 5). In the experiments in which the potato plants were set at distances of 1.2 metres from each other, the numbers of plants investigated in different years were as follows:

Year	1952	1953	1954	1955	1956	1957	1958	1959
No. of plants	79	47	36	37	30	24	24	20

In none of these plants were signs of the virus disease visible to the naked eye. Besides, the results of the core grafting experiments showed that these clones were resistant to the virus (Y) which caused the early maturity in the clones forming purple tubers (Table 1). There was no evidence of virus Y or X by means of the serological tests either. The antiserum of virus S on the contrary produced agglutination in the sap of some of the plants forming white tubers. Corresponding results were obtained with the sap of *Solanum demissum*. However, at no time during the whole period from year 1945 (cf. 12), when the *S. demissum* material was obtained for our experiments, were visible symptoms of a virus disease discerned in this species.

Discussion

According to the investigations reported above, the rate of development of the potato plant was hastened considerably by infection with virus Y. It is true that the earliness of onset of flowering has not been determined by the virus infection, but the flowering manifested itself less abundantly and the flowering time was shorter in the diseased than in the healthy plants (Table 2). The virus disease hastened the yellowing and withering of the haulms. The onset of tuber formation also manifested itself earlier in the diseased than in the healthy plants. After the withering of the haulms, however, the tuber yield could no longer increase. Consequently, when not lifted until late autumn, the largest amount of tubers was yielded by the healthy plants (Tables 3 and 4). In the same way, exposure of the potato plants to a short photoperiod does not affect the onset of flowering, but decreases the number of the flowers, as well as the length of the flowering time, hastens the onset of tuber formation, the yellowing and withering of the haulms, and at the same time the increase in the tuber yield ceases relatively soon (cf. 9, 10). Consequently, a high degree of similarity has been found between the development of healthy potato plants exposed to short day, on the one hand, and of the virus-

diseased F_1 clones forming purple tubers, on the other hand. LIHNELL (4), too, has found that the potato rugose, especially, but also potato crinkle, lead to early maturation of the haulms of the potato plant. According to KENNEDY (1) potato leaf roll stimulates the onset of new tubers after dry periods.

In our experiments the virus did not bring about further degeneration of the potato plant in successive vegetative generations, but permanently altered its nature once and for all (Table 4).

The clones with white tubers arising from bud mutations appeared to be resistant to the virus Y investigated (Table 1). In these clones, a tendency to change to still later maturing types was observed (Table 5; 10). Thus the bud mutations have produced clones with properties even more closely resembling those of the mother plant (*Solanum demissum*), which forms white tubers, develops tubers extremely late in the longday conditions of Finland (12), and has appeared to be resistant to potato rugose in our experiments. The results of the reciprocal crosses indicate that the cytoplasms of *S. demissum* and of *S. tuberosum* are of different nature (7). These results invite the thought that the *S. tuberosum* genes, when in contact with *S. demissum* plasma, become labile and readily mutable (3; cf. also 2, 6), when their action declines and the properties of plants descended from this species hybridization readily change in the direction of those of the mother plant.

Conclusions

The following are the main conclusions to be drawn from the results of experiments in which the resistance of F_1 clones descended from the species hybridization *Solanum demissum* x *S. tuberosum* (Rosafolia) was investigated:

The clones forming purple tubers appeared to be very susceptible to the potato rugose. Bud mutants descended from the former, forming white tubers, appeared to be wholly resistant to this virus.

The virus accelerated the onset of tuber formation and the decline of the haulms of the susceptible F_1 plants.

The degeneration of the virus-diseased plants did not progress from one vegetative generation to the next; instead, the virus altered the rate of development of the potato plant to a new level.

REFERENCES

(1) KENNEDY, J. I. 1955. Some physiological aspects of virus degeneration of potatoes. Proceeding in the Ann. appl. biol. 43: 149—151.

(2) KOOPMANS, A. 1951. Cytogenetic studies on *Solanum tuberosum* L. and some of its relatives. Genet. 25: 193—337.

(3) LAURILA, KAIHO 1957. *Solanum tuberosum* L. ja *S. demissum* Lindl. -lajien välisten risteytysten F1-polvessa ilmenneestä kasvullisesta muuntelusta (Über vegetative Mutation in der Generation F1 bei Kreuzungen zwischen den Arten *Solanum tuberosum* L. und *S. demissum* Lindl.). Maatal. tiet. aikak. 29: 56—67.

(4) LIHNELL, D. 1943. De viktigaste potatisviroserna; symptom, spriding och betydelse. Handlingar till lantbruksveckan 1943: 303—309.

(5) MANNER, Rolf 1952. Erfarenheter rörande spontant uppträdande förändring av bestående natur i Early Puritan (Experiences from spontaneous changes in Early Puritan). Medd. Gullåkers växtförädl. anst. 1952: 240—247.

(6) MICHAELIS, P. 1935. Erhöhte Wachstumsintensität und Pilzresistenz durch Plasmavererbung, sowie über die Bedeutung des Plasmas bei Kreuzungsschwierigkeiten. Züchter 7: 74—77.

(7) POHJAKALLIO, O. 1951. Potatisens resistensfrågor. Nord. jordbr.forskn. 1951: 486—492.

(8) —»— 1954. Eräitä lisäselvityksiä vuoden 1953 perunarutto- [*Phytophthora infestans* (Mont.) de Bary] epidemian luonteesta (Some features of the *Phytophthora infestans* epidemic in the summer of 1953). Maatal. tiet. aikak. 26: 142—147.

(9) —»— & SALONEN, A. 1947. Der Einfluss der Tageslänge auf Entwicklung und Energiehaushalt einiger Kulturpflanzen. Acta agr. fenn. 67, 1.

(10) —»— SALONEN, A. & ANTILA, S. 1957. Analysis of Earliness in the Potato. Acta agr. scand. 7: 361—388.

(11) —»— VAARTAJA, O. & ANTILA, S. 1955. Frost resistance of potato tubers. Acta agr. fenn. 83: 42—62.

(12) VIIRILÄ, F. 1949. Päivän pituuden vaikutuksesta meksikolaisen luonnonvaraisen perunan, *Solanum demissum* Lindl., biologiaan (On the effect of day length on the biology of *Solanum demissum* Lindl.). Archivum soc. zool. bot. fenn. »Vanamo» 4: 1: 60—72.

(13) STEINECK, O. 1955. Die photoperiodische Reaktion von »Schosser» -Stauden der Sorte Erstling. Z. Pflanzenz. 35: 137—148.

SELOSTUS:

LAJIRISTEYKSESTÄ *SOLANUM DEMISSUM* × *S. TUBEROSUM* POLVEUTUVIEN ERÄIDEN Fi-KLOONIEN VIRUSTAUDINKESTÄVYYDESTÄ

ONNI POHJAKALLIO ja LAURA KARHUVAARA

Yliopiston kasvipatologian laitos, Helsinki

V. 1947 suoritetusta lajiristeytyksestä *Solanum demissum* Lindl. x *S. tuberosum* L. (Ruusulehti) saatiin siementaimi, joka syksyllä 1948 muodosti punakuorisia mukuloita. Mukuloista kasvatetut jälkeläiskasvit muodostivat myös v. 1949 punakuorisia mukuloita, mutta silmumutaatiosta johtuen osa kasveista v. 1950 muodosti mukuloita, joiden kuori oli valkea. Jatketuissa tutkimuksissa ilmeni, että punamukulaiset kloonit olivat viiruviroosinarkoja, valkomukulaiset sen sijaan virustaudinkestäviä; silmumutaatio oli siis aiheuttanut myös virustaudinkestävyyden.

Virustauti joudutti perunan mukulanmuodostumisen alkamista, josta johtuen sairas peruna antoi aikaisin korjattuna suuremman mukulasadon kuin terve. Virus ei vaikuttanut perunan kukinnan alkamisaikaan, mutta lyhensi tuntuvasti kukinta-aikaa ja vähensi kukkien määrää. Sitä paitsi virus joudutti varsiston tuleentumista ja kuolemista, josta johtuen myös mukulasadon suurentuminen tyrehtyi verraten aikaisin. Tästä johtuen terve peruna antoi paljon suuremman mukulasadon silloin kun sadon korjuu tapahtui vasta myöhään syksyllä.

Virustaudin vaikutus perunan mukulanmuodostukseen ilmeni yleensä osittain jo samana kesänä kuin peruna sai virustartunnan. Kaikki sairaan kasvin mukuloista kasvatetut kasvit osoittautuivat virustautisiksi. Viruksen vaikutus mukulasadon kehitykseen ilmeni jo saastuntaa seuraavana vuonna lopullisena. Sitä seuraavina vuosina ei virus enää enempää surkastuttanut perunaa, joten virus vain muutti perunan kehityksen toisenlaiseksi.