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Rose bud proliferation, a disorder of still unknown etiology

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Abstract

A new, peculiar and often harmful budding disorder in roses, known to occur in the Netherlands since about 1954 and presumably in some other countries, is described. It may lead to a failure of up to 90% of the plants in a nursery field.

Affected buds, having normally attached after budding in summer but remaining dormant until spring, then develop into proliferating tiny witches' brooms or curly heads ('kroeskoppen') and later succumb or recover. Full-grown plants never show any abnormality.

Entomological, bacteriological, mycological and virological studies could not reveal the cause of the disease. Bud-graft experiments did not unequivocally demonstrate its infectious nature. Many field observations and data gathered by the Plant Protection Service through a questionnaire did not show or suggest any correlation between disease incidence and special conditions or cultural practices.

The disease is now supposed to be due to either a hormonal imbalance brought about by the act of budding, or by a pathogenic micro-organism occurring in low concentration in affected tissue or disappearing from it after the onset of the pathological process.

Introduction

In the Netherlands garden roses are propagated by budding onto rootstocks. These are seedlings of *Rosa dumetorum* 'Laxa', *R. multiflora*, *R. rubiginosa*, and special selections of *R. canina*, all produced on special holdings. After planting in the nurseries in spring, they are budded on the root neck during July and August. The inserted dormant buds normally develop next spring, when the rootstock is cut back just above the inserted bud, thus forcing the latter to further develop.

In several Dutch rose nurseries since about 1954 a peculiar and often harmful growth aberration of these developing buds is known to occur. Such buds proliferate into curly heads (Dutch: 'kroeskoppen') which usually die prematurely. Losses can amount to 90%, but vary considerably.

The disease may resemble 'rose stunt' or 'bud proliferation', incompletely described in England (Hutton, 1970; Ikin and Frost, 1974). 'Pinch off', superficially described in Oregon, USA (Roberts, 1962) has symptoms (Wagnon and Traylor, Sacramento Calif., USA, personal communication, 1973) also suggestive of rose bud proliferation.

Preliminary research in the Netherlands could not reveal the etiology of the disease, as is the case with rose stunt disease in England (Ikin and Frost, 1974) and with pinch off in California (Wagnon and Traylor, personal communication, 1973).

The present paper describes the disease and its incidence, summarizes the research performed in the Netherlands, and discusses its possible nature.

Fig. 1. Symptoms of bud proliferation in *Rosa* 'Elmshorn' on *R. rubiginosa*. Bar represents 1 cm.

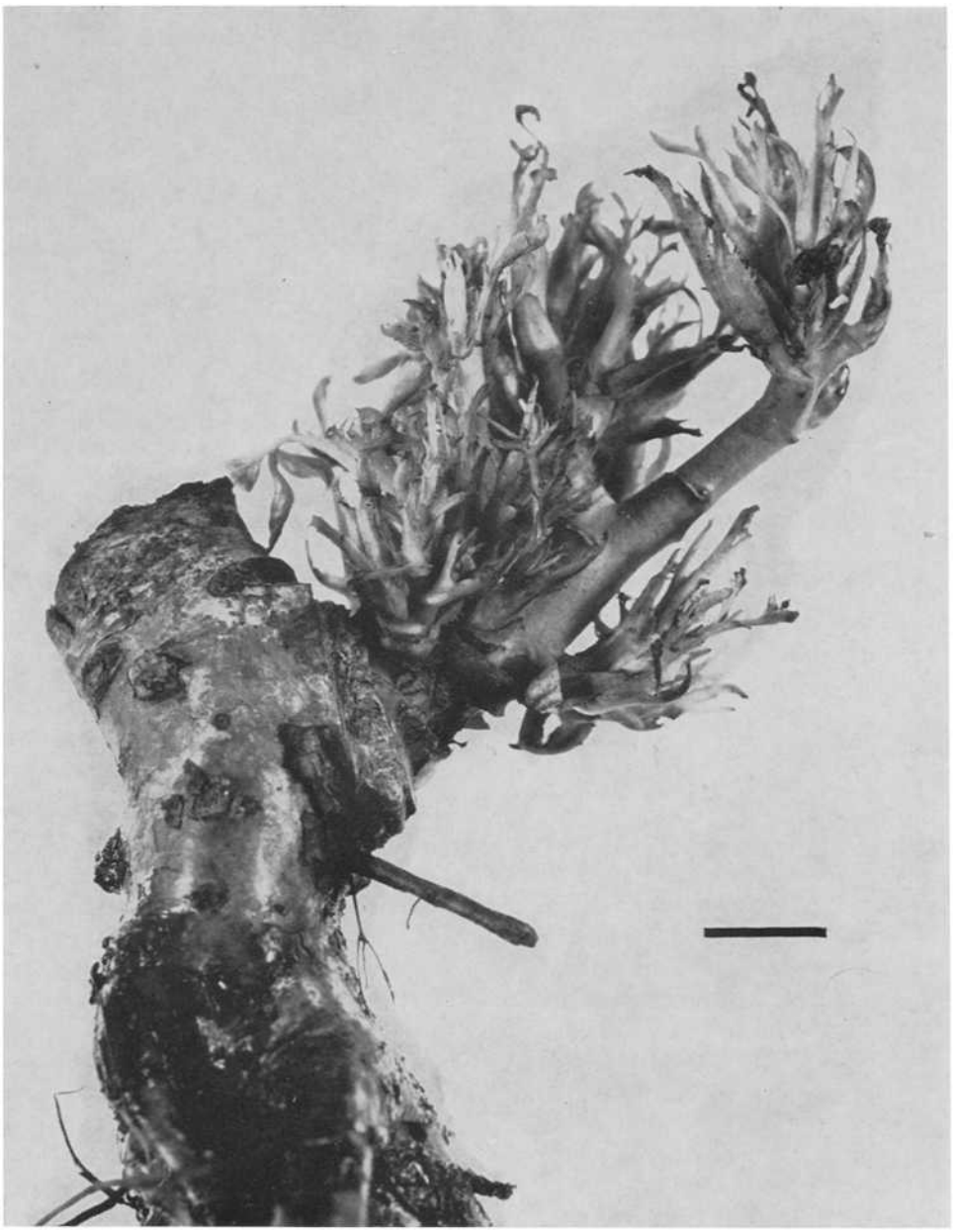

Fig. 1. Symptomen van kroeskopziekte in Rosa 'Elmshorn' op R. rubiginosa. Staaf geeft 1 cm aan.

Fig. 2. Symptoms of varying severity of bud proliferation in 'Orange Morsdag' on *R. rubiginosa*; Plant at the left tends to recover. Bar represents 1 cm.

Fig. 2. Symptomen van uiteenlopende ernst van kroeskopziekte in 'Orange Morsdag' op R. rubiginosa; linker plant vertoont neiging tot herstel. Staaf geeft 1 cm aan.

Disease symptoms

In case of bud proliferation, the bud, which has attached and remained dormant after budding last summer as normal, shows abnormal development in spring (Fig. 1 and 2) and growth retardation. The first leaves consist of a sheath only. Further leaves develop poorly, hardly producing blades. Leaflets remain tiny and weedy, and shoot development often comes to a complete standstill. At the base of the original shoots various adventitious buds develop and, often axillary buds start to develop on the stunted shoots, but they fail to elongate. This leads to a proliferation of shoots into a tiny witches' broom of a few centimeters high, often not exceeding 1 cm. Infrequently, miniature flowerbuds are formed (Fig. 2). The bushy sprouting led to the Dutch name 'kroeskopziekte" (= curly head disease). Sometimes the original bud may already develop in fall, not showing any abnormality in that season, but producing aberrant branching when cut back next spring.

Initially no necrosis or die-back is observed, but mostly the affected plants gradually deteriorate or desiccate and may already have succumbed by the end of May. Some-

Fig. 3. Bud proliferation in 'Sympathie' on *R. multiflora;* right, healthy plant; affected plant left is recovering.

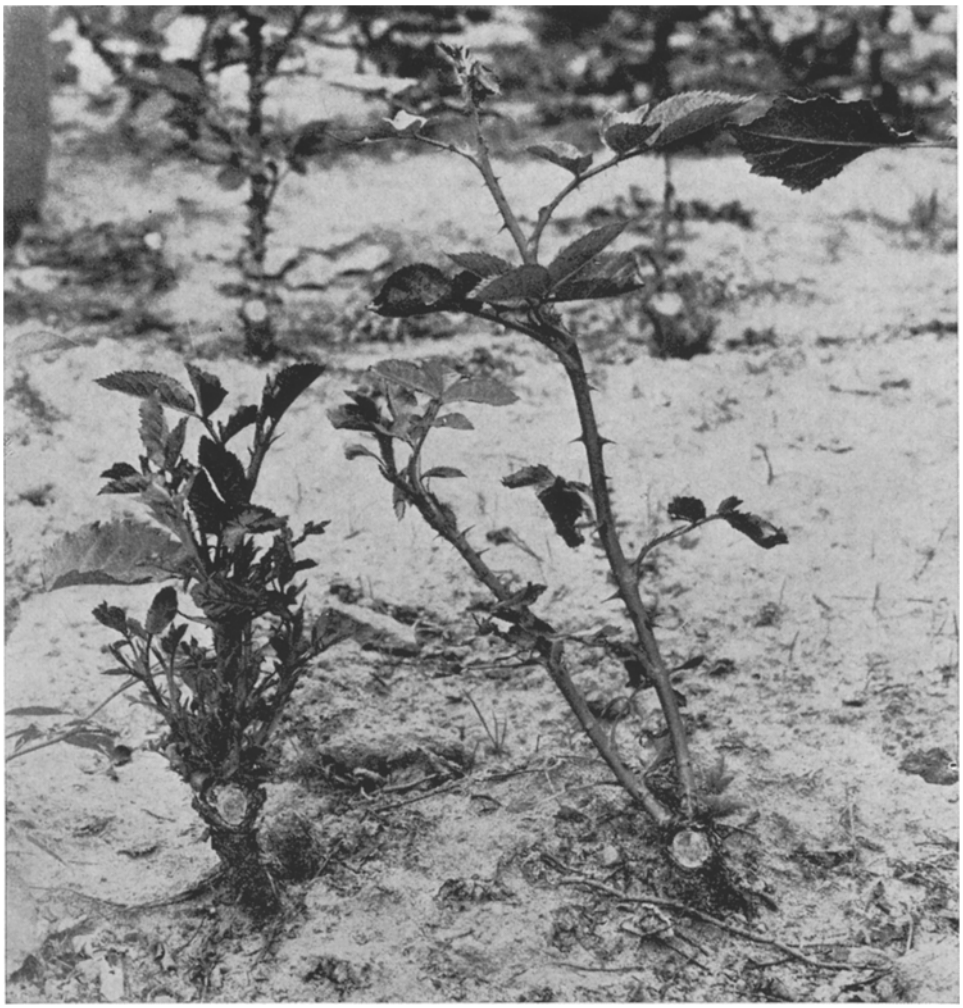

Fig. 3. Kroeskopziekte in 'Sympathie' op R. multiflora; rechts, gezonde plant; de aangetaste linker plant begint zich te herstellen.

times brooms of some 10 cm may develop which are easy to detach from the shield, which itself remains attached to the rootstock.

Occasionally plants recover by producing one or a few at first slender branches (Fig. 3) mostly with more or less elongated internodes and later looking completely normal, thus yielding a normal, perhaps initially slightly smaller rose bush. Such plants never show a recurrence of any of the previous abnormalities. The slightly more numerous branchlets at the base remain for some time as a reminder of the original curly head. Diseased plants transferred to the greenhouse soon recuperate also, remaining healthy afterwards.

Fig. 4. Patchwise occurrence of bud proliferation in 'New Dawn' on *R. dumetorum* 'Laxa'.

Fig. 4. Pleksgewijs voorkomen van kroeskopziekte in 'New Dawn' op R. dumetorum 'Laxa'.

Wild sucker shoots developing from stocks with a proliferating bud, or from recovered plants, have never been observed to produce any abnormal growth.

Distribution of the disease and conditions

In the Netherlands the disorder mainly occurs in Eastern Brabant and Northern Limburg, where about 80% of the culture is situated, but it has also been observed in rose nurseries elsewhere. Its distribution in the field is erratic and completely unpredictable. Often a few affected plants can be found, occurring singly or some together in the same row. Frequently, however, such plants are separated by one or a few normal ones. Aberrant plants may also occur in patches in fields otherwise normal (Fig. 4). Sometimes nearly all plants are affected, which leads to severe losses and makes the disease of economic importance.

To gather information on the possible rôle of certain environmental conditions, in 1965 a survey was made by the Plant Protection Service. Detailed data were assembled on cultivars affected, extent of attack, type, origin and pretreatment of rootstocks used, origin, quality and pretreatment of budding material, time and way of budding and binding, climatic and soil conditions, preceding crops, cultural practices as tilling, fertilizing, control of weeds, insects and other pests and diseases.

The results of the survey did not reveal any clue as to a possible pathogenic or essential cultural cause. They are in agreement with many field observations and farmers' interviews made by the present authors.

A few uncertain indications obtained from the questionnaire and field observations are worth mentioning. All cultivars of climbing and bush roses have been reported to show the phenomenon and to do so on all rootstocks used, although incidences usually are highest on *R. dumetorum* 'Laxa' and lowest on *R. canina* 'Schmid's Ideal'. The disease seems to be more prevalent in roses on bought rootstocks than on those grown in the nursery concerned, probably because of decrease in quality during transport and storage. Moreover, budding material from plants grown in the greenhouse may produce less curly heads than that from plants grown in the open. However, greenhouse material is budded about a month earlier, but there are indications that the time of budding is not of influence. The spotwise occurrence in the field is sometimes correlated with less favourable local conditions as frost, wind and lack or excess of water.

Research performed

Preliminary diagnostic work was first performed by the Plant Protection Service (PD), responsible for surveying Dutch crops for new and uncommon diseases and pests. These investigations with emphasis on fungi (Taconis, personal communication, 1966) did not reveal a pathogenic organism. The questionnaire mentioned above did not give any further clue.

Gradually a number of specialists, mainly of the Institute of Phytopathological Research (IPO), took over and tried to locate and isolate a possible incitant.

Originally a gall midge, the red bud borer (*Thomasiniana oculiperda*) was thought to be involved (De Vogel, Research Station for Arboriculture, Boskoop). Nijveldt (1966), however, could not establish its presence on the investigated fields neither in proliferated plants nor in the soil.

Repeated endeavours to isolate pathogenic bacteria also were unsuccessful (Maas Geesteranus, personal communication 1966, 1974 and 1975).

The incidental spotwise occurrence of the disease in the field led Van Hoof (unpublished report 1965 and personal communication 1974) to look for soil-borne viruses. Tobacco rattle virus and *Trichodorus* spp. were isolated from soil, but there was no correlation between these and the occurrence of symptoms. From diseased plants no virus could be transmitted mechanically to herbaceous test plants, even after the addition of nicotine to the extract. Later tests by the senior author further corroborated these negative results.

Witches' broom-like growth developing from abnormal buds brought about the suggestion that mycoplasmas might be involved. Although this seemed unlikely because of lack of systemic 'infection' in affected plants and lack of graft transmission (see below), in early June 1974 some bushy sprout tips and leaves of two affected plants were embedded in the conventional way, sectioned ultrathin and studied in the electron microscope. Phloem tissues appeared to be normal, at least totally void of the pleomorphic micro-organisms characteristic of mycoplasma-infected plants.

With help of the Research Station for Arboriculture at Boskoop (De Vogel) and the Experiment Garden for Arboriculture at Horst (Detz and Venhorst) several trials were made to possibly detect infectivity by budding. Two are described here.

In one experiment, started November 1966, scions from wild shoots, that had developed on rootstocks of 'Alain' rose plants originally showing the disease, were mist-

Table 1. Results of bud transmission experiment with *R.* 'Allain'.

Rootstocks	Single-budding with healthy buds only		Double-budding with diseased buds under healthy buds	
	number of abnormal plants over number of plants budded	percentage of abnormal plants	number of abnormal upper and lower buds over number of plants budded	percentage of plants with abnormal upper buds
R. canina 'Schmid's Ideal'	0/6	0	0+0/3	0
R. canina 'Superbe'	2/40	5	1+1/16	6
R. dumetorum 'Laxa'	1/14	7	14+8/38	37
R. multiflora	0/20	0	0+2/28	0
R. rubiginosa	4/12	33	3+2/7	43
Total	7/92	8	18+13/92	20+14

Tabel 1. Resultaten van de proeven tot overdracht door middel van oculatie met R. 'Alain'.

propagated in the greenhouse. The same was done with wild shoots from healthy plants. Half of the 200 possibly infected and of the 200 apparently healthy rootstocks thus obtained were then grafted in the greenhouse with scions from 'Alain' plants which had shown the disorder, and the other half with healthy scions. None of the plants resulting from the 400 combinations showed any sign of abnormality.

In 1973 a field experiment was started at Horst on August 10. 'Alain' buds of apparently healthy origin were grafted on five different rootstocks, 184 plants in total. Ten days later half of the plants were double-budded below the healthy bud with one of diseased origin. Final results were recorded May 16, 1974 (Table 1). The number of diseased upper buds in plants double-budded is higher than in the single-budded plants (18 versus 7; or in percentages, 20 versus 8). On *R. dumetorum* 'Laxa' rootstocks the effect was more pronounced (37 versus 7%). Moreover, in 10 out of the 13 double-budded plants with symptoms in the lower 'donor' bud, the upper bud was affected as well. This may suggest a transmission through budding. However, a physiological influence on the upper bud by double-budding cannot be excluded. Unfortunately, double-budding with healthy buds had been omitted. In addition, healthy buds did produce proliferation as well, although to a slightly lesser extent, but total numbers of plants involved were low. Moreover, the number of upper buds showing symptoms after double-budding still were low as compared to normal disease incidence in plants cultivated as usual (see also below).

As apparent from these experiments, an important problem is that the disease itself is not reliably reproducible. Material of diseased origin is able to produce healthy plants and supposedly healthy material can yield abnormal plants.

Meanwhile, some cultivar-rootstock combination trials were done to either locate healthy and diseased origins (sources of infection) or demonstrate varietal resistance and/or predisposition to the disorder.

In an experiment at Horst during 1968–1969 various combinations were exposed to natural occurrence of the disease. Six rootstock cultivars were budded with material from apparently healthy plants of nine cultivars. Results obtained during spring 1969

Table 2. Influence of cultivar-rootstock combinations on disease incidence (indicated in percentages of plants attacked) after natural 'infection'.

Rose cultivars	Rootstocks					
	R. canina 'Heinsohn's Rekord'	*R. canina* 'Inermis'	*R. canina* 'Pollmer'	*R. canina* 'Schmid's Ideal'	*R. dumetorum* 'Laxa'	*R. rubiginosa*
Alain	3	1	16	4	21	6
Fanal	6	16	23	7	6	2
Garnette	9	12	37	6	7	4
Morsdag	28	18	32	11	15	54
New Dawn	4	20	32	10	64	1
Orange Morsdag	21	20	36	15	19	59
Orange Triumph	2	2	16	6	8	5
Paul's Scarlet Climber	1	2	13	0	32	0
White Dick Koster	22	33	27	26	38	64
Average percentage	11	14	26	9	23	22

Tabel 2. Invloed van cultivar-onderstamcombinaties op de mate van voorkomen van de ziekte (aangegeven in procenten aangetaste planten) na natuurlijke 'infectie'.

are recorded in Table 2. *R. dumetorum* 'Laxa' and notably *R. canina* 'Pollmer' showed higher disease incidences than other rootstocks, especially *R. canina* 'Heinsohn's Rekord', 'Inermis' and 'Schmid's Ideal'. Introduction of a pathogen especially with the former stocks was unlikely because on e.g. 'Laxa' certain cultivars, like 'Fanal', 'Garnette' and 'Morsdag' did better than on e.g. 'Inermis'.

In a similar experiment, also carried out at Horst in 1972–1973 and with *R.* 'Alain' only and in triplicate, disease incidence was much higher. Percentages of attack again were highest on *R. dumetorum* 'Laxa'. Otherwise, results were highly erratic (see Table 3).

Since growers sometimes tend to ascribe the disorder to careless and hasty budding and thus to a more or less mechanical cause, Swinkels-Verpraet (unpublished report, 1969) made an anatomical study of normal and abnormal bud-unions harvested while showing the disease. She observed more necrotic tissue than normal between stock and bud and supposed this to obstruct the development of vascular connections. However, a causal relationship between the anatomical deviations studied and the macroscopic syndrome was not shown.

Discussion

So far, the research reported has not given any clue as to the cause of the phenomenon. These negative results are in agreement with those obtained in England with rose stunt disease or bud proliferation where plant pathologists, entomologists and horticulturists have not yet been able to disclose its cause (Hutton, 1970; Ikin and Frost, 1974). Graft transmissions failed (Hutton, 1970), and affected plants surviving transplanting to the greenhouse recovered from disease (Ikin and Frost, 1974).

Table 3. Disease incidence (recorded in percentages of plants attacked) in 'Alain' on various rootstocks after natural 'infection'.

Rootstocks	Experiment number			Average percentage
	1	2	3	
R. canina 'Brög's Stachellose'	19	10	3	9
R. canina 'Inermis'	19	3	0	8
R. canina 'Schmid's Ideal'	29	52	0	27
R. canina 'Superbe'	0	7	20	8
R. dumetorum 'Laxa'	90	70	70	77
R. multiflora	15	43	27	28
R. rubiginosa	11	15	0	9

Tabel 3. Mate van voorkomen van de ziekte (aangegeven in procenten van aangetaste planten) in 'Alain' op verschillende onderstammen na natuurlijke 'infectie'.

In its symptoms the abnormality reminds of rosetting reported from England and some other countries in the first year's growth of certain pear varieties budded on to quince stocks. In severe cases growth from the bud is extremely reduced, the total extension growth being less than 1 cm. Transmission tests were also negative and the disease could not be associated with damage by insects, mites, nematodes, fungi and bacteria, or with spray damage, nutritional factors, cultural practices, and abnormal weather conditions (Williams and Campbell, 1957; Luckwill, 1963).

It has been suggested by Ikin and Frost (1974) that the rose disorder may be similar to the effect of rose wilt virus on maiden plants as described by Fry and Hammett (1971) in New Zealand. That disease, however, is characterized by symptoms in established plants like leaf epinasty and curling, reduced apical dominance and progressive dieback of branches, ultimately leading to death of the whole plant. Infected *R. multiflora* rootstocks showed considerable reduction in growth vigour. Moreover, the disease could be easily transmitted by double-budding to several rose cultivars. It may also be confused with *Verticillium*-wilt (Fry and Hammett, 1971; Hammett, 1971), which is not infrequent in roses.

Wagnon and Traylor (personal communication, 1973) have compared rose pinch off in Oregon and California with rose wilt and found clear differences, in that pinch off is not sap and graft transmissible, not naturally spread, and has symptoms only visible in spring in budded plants with a proliferation of the bud.

Viruses are increasingly found to be involved in causing bud or graft union disorders either by disturbing the often labile balance between bud or graft and stock, causing direct graft incompatibility as in many woody crops, or by causing vascular necrosis below the union when a tolerant tree, well-established on a sensitive rootstock, later gets infected. With tristeza of citrus the phloem necrosis leads to increasing food depletion in, and deterioration of the root system, this in turn causing a slow or rapid decline of the above ground parts of the plant (Schneider, 1959; for a short description and references see Bos, 1970). Such diseases basically differ from rose proliferation. Moreover, viruses in any case should be graft transmissible.

Pear decline symptomatologically resembles citrus decline, but is caused by a phloem-limited mycoplasma. Some mycoplasmas like that of pear decline cause phloem deterioration (Batjer and Schneider, 1960). Many fail in doing so, but are

characterized by hormonal disturbances leading to excessive branching and floral abnormalities (Bos, 1970).

Bud proliferation of roses may suggest infection with a mycoplasma. Here again, these micro-organisms are translocated in the vascular tissue and the disorders concerned are at least graft transmissible, although the pathogen may be hard to detect as with apple proliferation (Seidl, 1965 and 1968). Symptoms of mycoplasma diseases usually develop under a wide variety of conditions and in many plant species. It is hard to understand why buds from infected rose plants would only proliferate after budding onto rootstocks and why infected rootstocks would only produce symptoms in buds inserted onto these and not in their own suckers.

If not infectious, then the possibility of a non-contagious, merely physiological disorder remains, but it is not understood how this is incited. There is no evident correlation between its occurrence and special cultural practices or conditions, except the act of budding and the associated artificial wounding itself and the cutting back of the rootstock later. However, the abnormality does not exclusively result from mechanical damage. There is some sort of continuing irritation, stimulating the plant to react.

Theoretically, this might result from a further hormonal imbalance ensuing from the production of wound hormone and depending on a range of conditions. This might explain the erratic occurrence of the disease.

The abnormality in certain ways resembles the neoplastic tumorous outgrowths which are not graft transmissible but ascribed to localized hormonal imbalances due to genetic factors as in certain *Nicotiana* hybrids. In vitro cultures from such tissues may produce organoid witches' broom-like structures and even recover (Kehr and Smith, 1954). However, with bud proliferation neoplastic growth is exclusively organoid in nature.

In spite of the failure of transmission tests and of experiments to isolate a pathogen, a bacterial cause still cannot be completely excluded. Several tumors in plants are known to be caused by e.g. *Agrobacterium tumefaciens*, but it is often hard or impossible to isolate that pathogen from abnormal tissue (e.g. Klement, 1974). Certain strains of this bacterium are known to produce organoid witches' broom-like structures and from these, normal sprouts may regenerate in tissue culture (Braun, 1954). The hypothesis of such micro-organisms being involved, occurring in low concentration or even disappearing once the process has started, seems tempting. However, a rôle of *A. tumefaciens* is improbable because its effect on greenhouse roses (Maas Geesteranus, personal communication 1975) differs completely from bud proliferation of outdoor roses. Moreover, *A. tumefaciens* is known to affect many other plant species in nature and such attacks are unknown or rare in the area where bud proliferation is prevalent in roses.

Samenvatting

Kroeskopziekte van roos, een afwijking met nog onbekende oorzaak

Een nieuwe, in 1954 voor het eerst vermelde, thans schadelijke oculatieziekte in rozenkwekerijen in het zuidoosten, maar ook voorkomend in andere delen van het land, wordt beschreven. Aantastingen variërend van enkele procenten tot 90% van de ocu-

laties op één perceel zijn waargenomen. De afwijking komt waarschijnlijk ook in Engeland en Amerika voor.

Aangetaste ogen ontwikkelen zich, nadat ze na oculatie in de vorige zomer normaal vergroeiden en slapende bleven, tot woekerende kleine heksenbezempjes of kroeskoppen, soms slecht één of enkele centimeters groot (Fig. 1 en 2). Later in het seizoen gaan deze scheutwoekeringen veelal te gronde, soms ook herstellen ze zich (Fig. 3) en er ontstaan dan geheel normale, hoogstens iets in groei achterblijvende of wat spichtige planten. Aan volgroeide planten is de afwijking nooit waargenomen. In een aanplant staan de aangetaste planten meestal grillig verspreid tussen de gezonde. Soms treedt pleksgewijs aantasting op (Fig. 4).

Door entomologisch, bacteriologisch, mycologisch en virologisch onderzoek kon geen ziekteoorzaak worden opgespoord. Een enquête gehouden door de Plantenziektenkundige Dienst onthulde geen duidelijke correlatie tussen de wijze en mate van voorkomen en speciale omstandigheden of bepaalde teeltmaatregelen. De verschijnselen werden in alle onderzochte cultivars van klim- en struikrozen en op alle gebruikte onderstammen, zij het niet in gelijke mate, waargenomen (Tabel 2 en 3). In entproeven in de kas kon het verschijnsel niet worden opgewekt, noch van zieke op gezonde planten worden overgebracht. In dubbeloculatieproeven buiten leek de ziekte van ogen van zieke herkomst over te gaan op er boven geöculeerde gezonde ogen (Tabel 1), maar daarbij bleven vele ogen van zieke herkomst gezond en werden ogen van gezonde herkomst ziek.

Verondersteld wordt tenslotte dat de afwijking te wijten is aan een door het oculeren en de daaruit resulterende callusvorming op gang gebrachte verstoring in de groeistofhuishouding, die van een samenstel van omstandigheden afhankelijk is. Het wordt echter niet geheel uitgesloten geacht, dat de verstoring toch op gang wordt gebracht door een micro-organisme, dat in afwijkend weefsel in lage concentratie voorkomt, of weer verdwijnt nadat het pathologische proces op gang is gebracht.

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