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Bud necrosis, a storage disease of tulips. I. Symptoms and the influence of storage conditions.

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Abstract

The symptoms of bud necrosis in tulip bulbs are described. This disorder is neither contagious nor hereditary. It is found more often and more severely in certain cultivars (viz 'Red Champion' and 'White Sail') than in others (viz 'Rose Copland') in which it appears less frequently or not at all. The disease originates in the stamens of the flower-producing main bud during dry storage between lifting and planting of the bulbs. The occurrence of the disease in sensitive cultivars is promoted by storage of the bulbs under conditions of poor ventilation (viz packed in cardboard boxes) and at higher temperatures later in the storage period (after September 1).

The experimental results suggest that there are several pathogenic factors and that the combined action of these factors can lead to bud necrosis; the primary factor is probably of physiological nature.

Introduction

Bud necrosis in tulips has long been known but its cause was identified only recently (De Munk, 1971). The literature does not contain a satisfactory description of the symptoms. They will be described in the present paper. Two groups of symptoms can be distinguished: those in mature plants and those of the buds occurring within the bulbs before planting, when the vegetative and generative parts are still in an embryonic stage.

Symptoms of the buds before planting

During dry storage, externally healthy looking tulip bulbs may show a number of necrotic phenomena in the bud from which the main (flower-producing) shoot will develop. In light cases only the stamen primordia are necrotic; in severe cases the whole bud and the inner bulb scales are necrotic. On the basis of the degree to which the buds are affected we distinguish between stamen necrosis, flower necrosis, shoot necrosis, heart necrosis, and bulb necrosis.

In stamen necrosis one or more of the stamens are wet, glassy, and slightly discoloured, due to infiltration of the tissue. The stamens show decay in one or more anthers, a process which starts at the tip of the stamen and is accompanied by a discolouration to black (Fig. 1A). Sometimes, however, the necrosis is not combined with infiltration. The deflection then takes the form of brown spots, especially at the tips of the stamens (Fig. 2).

In flower necrosis, all stamens, the pistil and perianth leaves become necrotic.

Fig. 1. Dissected buds (tulip cv. 'Red Champion'), showing different degrees of bud necrosisA: foliar and perianth primordia removed; onset of decay at the tips of the stamens, which are partly collapsed (stamen necrosis). B: foliar primordia removed; all flower parts are decaying (flower necrosis). C: necrosis has reached the outer foliar primordium (shoot necrosis). D: the necrotic parts partially eaten by mites (heart necrosis).

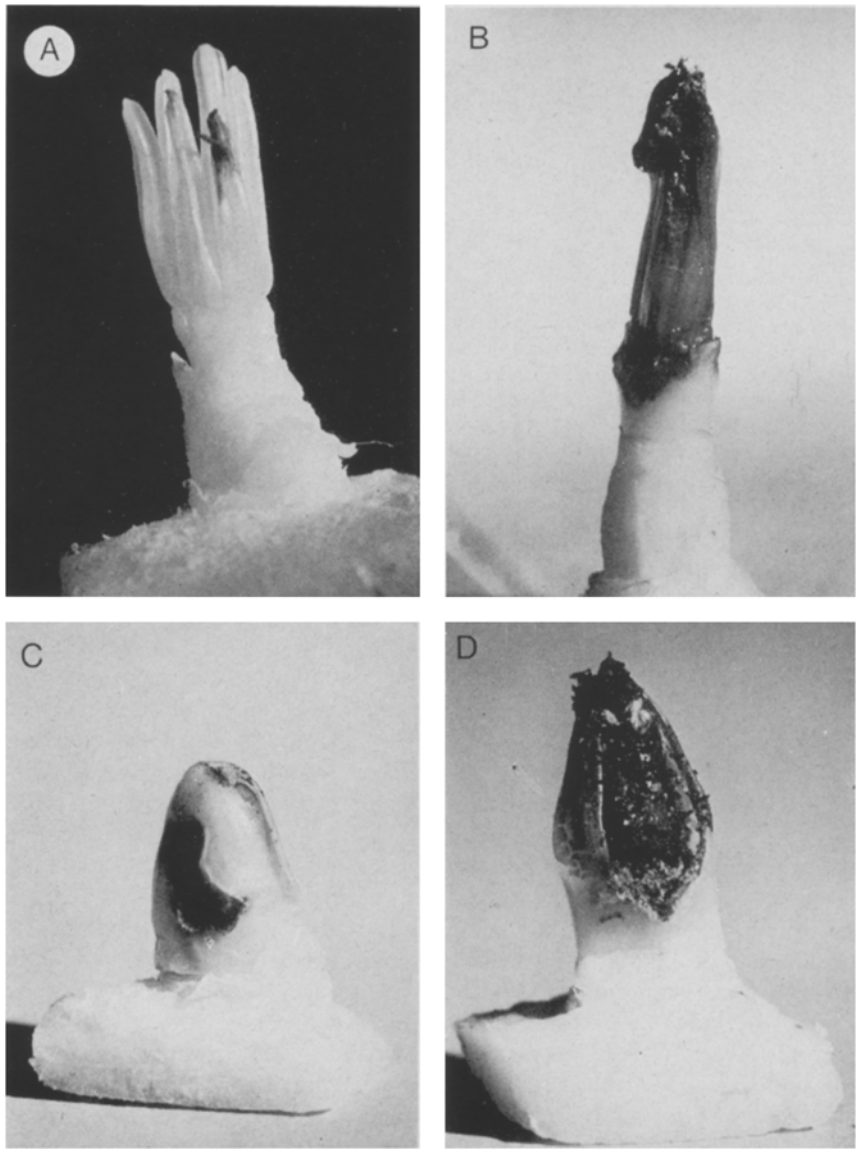

Fig. 1. Uitgeprepareerde knoppen (cv. 'Red Champion') die in verschillende mate kernrot vertonen. A: (loof- en bloembladprimordia weggesneden) het begin van verrotting aan de toppen van de gedeeltelijk vervallen meeldraden (meeldraadnecrose). B: loofbladprimordia weggesneden; alle bloemdelen rotten (bloemnecrose). C: de necrose wordt zichtbaar op het buitenste loofbladprimordium (spruitnecrose). D: de necrotische delen zijn gedeeltelijk door mijten weggevreten (kernnecrose).

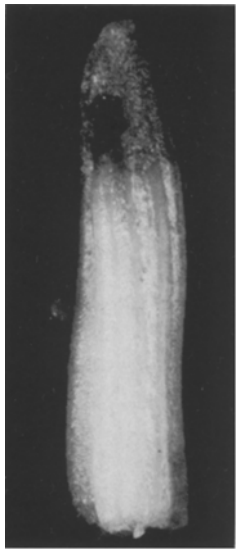

Fig. 2. Dissected stamen (cv. 'White Sail') showing a dark brown spot at the tip. (Stamen necrosis without tissue infiltration).

Fig. 2. Uitgeprepareerde meeldraad (cv. 'White Sail'), welke een donkerbruine vlek vertoont aan de top. (Meeldraadnecrose zonder weefselinfiltratie).

The affected parts turn brownish-black and are sharply delimited against the unaffected tissue (Fig. 1B).

In shoot necrosis, not only the flower parts but also some foliar primordia are partly or totally affected and become brownish-black (Fig. 1C). In heart necrosis, all foliar primordia are necrotic. Only the first internode of the stem remains healthy (Fig. 1D).

In bulb necrosis, the inner bulb scales as well as the bud are affected. Here too, the characteristic brownish-black discolouration of the necrotic tissue can be observed.

The phenomena of the disease are usually associated with reduced growth of the

Fig. 3. Dissected buds (cv. 'White Sail'). On the left, a normal, closed bud; on the right an abnormal, open bud showing the reduced growth of the foliar primordia as a consequence of which the stamens project beyond the bud.

Fig. 3. Uitgeprepareerde knoppen (cv. 'White Sail'). Links een normale gesloten knop; rechts een abnormale open knop waarbij als gevolg van geremde groei van de loofbladprimordia de meeldraden naar buiten steken.

buds. The foliar primordia are, however, relatively more inhibited than the stamens, as a consequence of which the stamens project beyond the sheath of the foliar primordia (Fig. 3).

Probably depending on the nature of the organisms present, the necrotic parts are either dry or wet. The organisms found on the decaying buds include a number of fungi (e.g. *Penicillium* spp.) and bacteria known as non-parisitic and also mites (*Rhizoglyphus echinopus* and *Tyrophagus* sp.). Ultimately, the necrotic parts may be totally eaten up by these mites.

Symptoms during flowering in the field

When bulbs with necrosis in the buds are planted, very evident abnormalities occur in the period of flowering. In the same way as for buds, the degree of deformation can be classified as stamen, flower, shoot, heart, and bulb necrosis.

In stamen necrosis, some or all of the stamens are decayed. The brownish-black, often mouldy remnants of the stamens are present in the otherwise healthy flower. When the flowers open, these remnants can be found adhering to the inner surface of the perianth leaves.

In flower necrosis, the whole flower is decayed and the last stem internode above the uppermost leaf is either not developed or decayed like the flower. The leaves and the remaining part of the stem are normal. The remnant of the flower bud can be observed as a brownish-black stump between the upper leaves (Fig. 4A).

In shoot necrosis, one or more leaves are lacking as well as the flower, and the stem has hardly developed. In most cases the remaining leaves are deformed by partial decay (Fig. 4B). The green of the foliar is more intense than normal due to the lack of the wax layer and stained by contiguous decayed parts.

In heart necrosis, no aerial parts of the main shoot develop. In the mother bulb the remnant of the main bud can be seen as a black stump (Fig. 4C). Under these conditions and sometimes also with shoot and flower necrosis, completely intact shoots may develop from axillary buds. These shoots differ from the normal main shoot by a weaker habit and smaller flowers. In bulb necrosis, finally, the whole inner part, including the main and axillary buds and the inner bulb scales, is decayed. Inside the still intact outer bulb scales, a black decaying mass will be found. At this degree of bud necrosis, no development of axillary buds will take place (Fig. 4D).

Sometimes aberrations are found that can be confused with bud necrosis, viz stamen anomalies originating from infection by *Botrytis tulipae*, frost damage, or 'heating in transit' (Van Slogteren, 1937). Furthermore, the last-mentioned disorder resembles bud necrosis because not only the stamens but also the whole flower and some of the leaves can be affected. These disorders, however, can always be distinguished from bud necrosis because the typical brownish-black discolouration occurring during decay does not appear. In 'heating in transit', the necrotic parts are papery, dry, and light yellow, and total decay does not occur.

It is obvious that the planting of bulbs with affected buds will result in deformed aerial parts or even in their absence. Because 5 to 6 months elapse between the presence of symptoms before planting and the observation of symptoms during flowering in the field, it could be suggested that the symptoms on fully-grown

Fig. 4. Field symptoms of bud necrosis. A: remnants of a decayed flower bud between the upper leaves (flower necrosis). B: tulip plants, showing the absence of flowers and stunted leaf growth; some shoots have developed from axillary buds (shoot necrosis). C: tulip bulb, lifted in spring and cut longitudinally showing the remnant of the main bud as a black stump; the axillary bud of the inner bulb scale is forming a new shoot (heart necrosis). D: tulip bulb, lifted in spring and cut longitudinally; the whole inner part is necrotic, viz the main bud, the inner bulb scale and part of the disc; outer scales and root system are still intact (bulb necrosis).

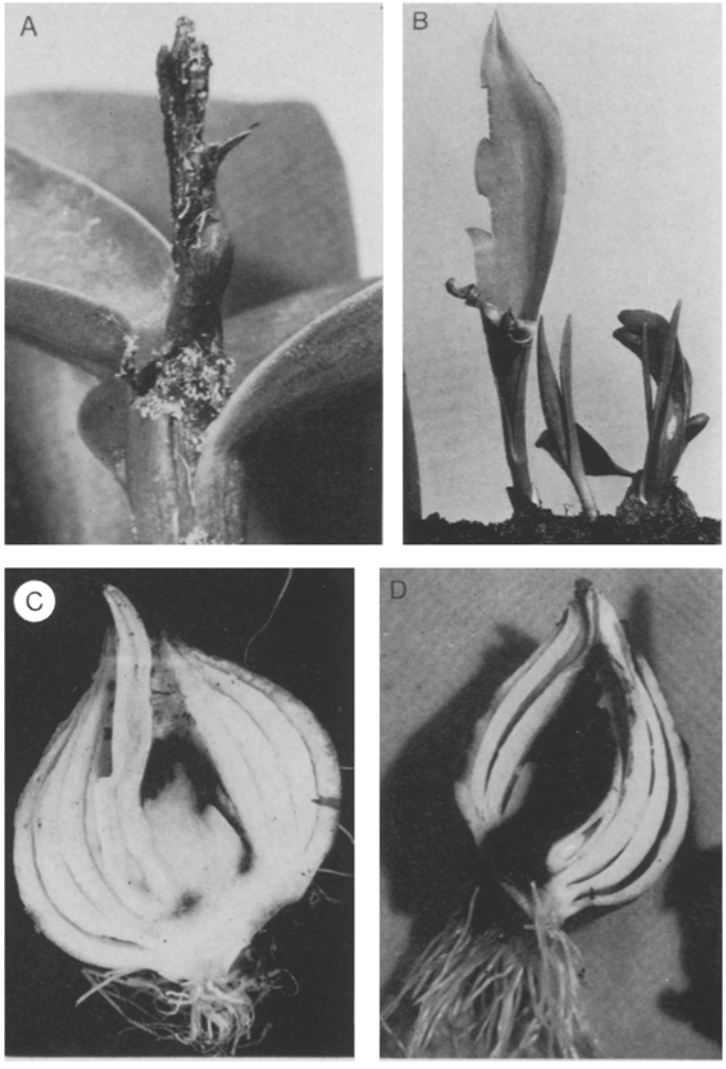

Fig. 4. Veldsymptomen van kernrot. A: resten van een verrotte bloemknop tussen de bovenste loofbladen (bloemnecrose). B: tulpeplanten waarbij de bloemen ontbreken en de bladeren misvormd zijn; uit okselknoppen hebben zich enkele spruiten ontwikkeld (spruitnecrose). C: in het voorjaar opgegraven en doorgesneden tulpebol met een zwarte stomp, het restant van de hoofdknop; de okselknop van de binnenste bolrok vormt een nieuwe spruit (kernnecrose). D: in het voorjaar opgegraven en doorgesneden tulpebol; het hele binnengedeelte van de bol, bestaande uit hoofdknop, binnenste bolrok en een gedeelte van de schijf, is necrotisch; de buitenste bolrokken en het wortelstelsel zijn nog gaaf (bolnecrose).

Table 1. Percentages of affected bulbs in 3 samples of bulbs 'Red Champion', determined before planting (15/11/1964, N = 50) and during flowering in the field (17/5/1965, N = 80).

Sample	Before planting	During flowering
a	4	12
b	66	64
c	93	91

Tabel 1. Percentages aangetaste bollen in 3 partijen van 'Red Champion', bepaald op een tijdstip voor het planten (15/11/1964, N = 50) en tijdens bloei op het veld (17/5/1965, N = 80).

plants are the result of other causes and not of bud necrosis originating during the storage period.

The experimental evidence, however, indicates that necrotic phenomena in the buds are indeed responsible for the described field symptoms. A very close correlation was found between percentage of diseased bulbs at the end of the storage period and the percentage of diseased plants in the field (Table 1).

Occurrence of the disorder

Although many fungi and bacteria could be isolated from affected buds, these organisms cannot be regarded as the primary cause of the disorder, because it was impossible to infect healthy buds with these organisms. The disease is not contagious, because healthy bulbs surrounding affected ones during storage or in the field remain healthy. The offspring of affected bulbs can also be completely normal (Table 2).

Observations in practice indicate that bud necrosis occurs frequently and with high percentages in certain cultivars (e.g. 'White Sail' and 'Red Champion') as compared with others (e.g. 'Rose Copland') in which the disorder is rarely observed. These differences in sensitivity cannot be explained up till now.

On the basis of the above-mentioned observations, bud necrosis may be defined as a non-contagious storage disease occurring in susceptible cultivars with necrosis starting in the anthers. Infection by fungi and bacteria is probably secondary.

Influence of storage conditions of the bulbs

Because bud necrosis originates during the period of dry storage of the bulbs, it is important to know the influence of storage conditions on the origin of the disease. The most important factors during storage are temperature and ventilation.

Table 2. Percentages bud necrosis in samples of bulbs of the same lot ('Red Champion') in two succeeding years (N = 48).

Lot	1961/1962	1962/1963
a	30	0
b	73	0

Tabel 2. Percentages kernrot in bollen van dezelfde partij ('Red Champion') in twee opeenvolgende jaren (N = 48).

The influence of temperature was investigated in experiments in which bulbs of cultivars susceptible to bud necrosis ('White Sail' and 'Red Champion') were kept at a certain temperature throughout the storage period (from digging at the end of July to planting at the end of November) or one temperature was applied in the period from the end of July to 1 September and another temperature from 1 September to the end of November.

To investigate the influence of ventilation, bulbs of 'White Sail' and 'Red Champion' were packed in sealed cardboard boxes (100 bulbs, size 10/11 cm, per box measuring $23 \times 24 \times 30$ cm) for the entire storage period. The control bulbs were stored normally, i.e. in open boxes with wire-gauze bottoms.

After these treatments the bulbs were planted in the field. During flowering in the following spring, the percentages of plants showing bud necrosis were determined. The less specific symptoms of stamen necrosis were not taken into account.

The data of the temperature experiments (see Table 3) show that the bud-necrosis percentages in 'Red Champion' were generally higher than in 'White Sail' and that the occurrence of the disorder is more influenced in 'Red Champion'. In this cultivar the highest percentage of bud necrosis occurred after storage at 20 °C. For 'White Sail' no optimum temperature could be demonstrated. In both cultivars the occurrence of the disorder was promoted when the temperature was higher during the second period of the treatment than during the first period. Although the temperature during storage has an influence on the occurrence of bud necrosis, the large differences in the percentages in successive years show that this influence is not always equally strong.

The data in Table 4 show that in 'White Sail' the occurrence of bud necrosis in 1961-62 and 1962-63 was restricted almost entirely to bulbs stored in boxes. In 'Red Champion' bud necrosis was promoted considerably by this treatment. Furthermore, the percentages for the successive years showed wide variations, al-

Table 3. Percentages of bud necrosis observed in the field for 'Red Champion' and 'White Sail' in successive years (1959 to 1963) after various temperature treatments during storage before planting (N = 95).

Temperature treatment (°C)		Red Champion			White Sail	
first period: from lifting to 1/9	second period: from 1/9 to planting at the end of November	59/60	60/61	62/63	60/61	61/62
13	13	2	11	1	17	6
17	17	45	–	6	10	6
20	20	84	76	30	1	4
23	23	30	–	3	1	–
23	13	8	–	0	0.3	–
13	23	43	–	–	12	–
23	17	24	–	4	1	1
17	23	50	–	–	8	–

Table 3. Percentages kernrot te velde in 'Red Champion' en 'White Sail' in de jaren 1959 tot 1963 na verschillende temperatuurbehandelingen tijdens de bewaring voor het planten (N = 95).

Table 4. Percentages of bud necrosis in 'Red Champion' and 'White Sail' in successive years after storage in sealed cardboard boxes or in open boxes with gauze bottoms. Storage temperature 20 °C (N = 100).

Season	Red Champion		White Sail	
	storage in closed boxes	open storage	storage in closed boxes	open storage
1961/1961	73	30	81	3
1962/1963	39	15	91	1
1963/1964	82	58	1	0
1964/1965	a 91	10	–	–
	b 77	10		
	c 51	17		
1967/1968			a 8	0
	–	–	b 7	0
			c 42	0

Tabel 4. Percentages kernrot in 'Red Champion' en 'White Sail' in verschillende jaren na bewaring in gesloten kartonnen dozen of in open gaasbakken. Bewaartemperatuur 20 °C (N = 100).

though temperature treatments were the same, and almost as much variation was found between results of the experiments in triplicate in one season (1964-65 and 1967-68), although storage temperature and origin of the bulbs were the same. As for the storage-temperature experiments, we must conclude that storage of bulbs in closed boxes can promote the occurrence of bud necrosis considerably, but that influence of limited ventilation is not always equally strong.

Discussion

Although the same terminology is used for the symptoms in buds and mature plants this does not mean that a certain degree of disease in the buds will be manifested to the same degree in the mature plants. The degree of aberration reached in the full-grown plants is determined by the degree of decay of the bud at the time at which the process of decay is arrested, which usually occurs at the moment that the bulbs are planted at low temperatures (Beijer, unpubl.).

Storage of bulbs at higher temperatures later in the storage period (after 1 September) increased the percentage of bud necrosis. Such conditions are also known to promote the occurrence of the so-called 'heating in transit' (a kind of bud blasting) (Van Slogteren, 1937). In describing the symptoms we also mentioned the possible confusion between symptoms of bud necrosis and 'heating in transit'. The similarity between these anomalies suggest that after a two-temperature treatment physiological aberrations appear first and are followed secondarily by the process of decay.

The experimental results indicate differences between the response of 'Red Champion' and 'White Sail' to certain temperatures and poor ventilation. 'Red Champion' not only shows sensitivity to poor ventilation but is also highly sensitive to certain temperature treatments. 'White Sail' shows little sensitivity to temperature treatment but is especially sensitive to limited ventilation. These differences suggest that different mechanisms can lead to the same result.

The factors mentioned do not always have the same effect. In spite of the same treatment and origin of the tulip bulbs, we found a wide variation in percentage of bud necrosis from year to year and between duplicates in the same season. The present results do not permit conclusions concerning the prerequisites for the functioning of the mechanisms. A further analysis of the conditions having importance during the genesis of bud necrosis will be reported in another paper.

Samenvatting

Kernrot, een bewaarziekte in tulpen. I. Symptomen en de invloed van bewaaromstandigheden

Kernrot is een afwijking in tulpebollen die een bloeibare maat hebben. De ziekte ontstaat tijdens de droge bewaring van de bollen en begint met afwijkingen aan de meeldraden. Bij de beschrijving is onderscheid gemaakt tussen symptomen welke zich tijdens de droge bewaring voordoen aan de knoppen waaruit zich de bloeiende spruit zal ontwikkelen en symptomen welke zich voordoen aan de bollen tijdens de bloei te velde. Naar de mate waarin de knoppen en planten afwijken, wordt gesproken van meeldraadnecrose (Fig. 1A en 2), bloemnecrose (Fig. 1B en 4A), spruitnecrose (Fig. 1C en 4B), kernnecrose (Fig. 1D en 4C) en bolnecrose (Fig. 4D).

Het is gebleken, dat de ziekte bij bepaalde cultivars ('Red Champion' en 'White Sail') vaak voorkomt, bij andere minder vaak ('Rose Copland') of in het geheel niet. Er kon worden vastgesteld, dat de ziekte niet besmettelijk of erfelijk is.

In de proeven werd het ontstaan van de afwijking bij 'Red Champion' en 'White Sail' bevorderd door de bollen te verpakken in kartonnen dozen (ventilatiebeperking) en door de bollen later in het seizoen (na 1 september) te bewaren bij hogere temperaturen dan daarvoor. Op grond van de proefresultaten wordt aangenomen dat de primaire oorzaak van de afwijking van fysiologische aard is. De aanwezigheid van schimmels, bacteriën en mijten moet als tweede oorzaak worden beschouwd.

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