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THE DUTCH ELM DISEASE IN EUROPE AROSE EARLIER THAN WAS THOUGHT

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Abstract. In January 1922, Dina Spierenburg published a second article on Dutch elm disease (DED) but with a title identical to the first: "Een onbekende ziekte in de iepen." Here she told of isolating a "*Cephalosporium-Graphium*" mixture from discolored wood in the growth layers of 1912 and 1913 of several small DED-infected, street-side elms in Renkum, The Netherlands, and in the layers of 1912-1915 of a large elm in Rotterdam. Her photos show that she had the same fungus as the one that Schwarz the same year was naming *Graphium ulmi*. Spierenburg's distribution map for 1921 showed that DED was then already in all provinces of The Netherlands. If one accepts Belgian and French DED reports (not based on cultures), the epiphytotic center appears to have been near Antwerp. In view of later experiences with rates of DED spread, the European infestation appears to have begun about 1900 to 1905. World War I events therefore are quite unrelated to DED origin.

En janvier 1922, Dina Spierenburg publiait un second article sur la maladie hollandaise de l'orme (M.H.O.--DED) mais avec un titre identique au premier: "Een onbekende ziekte in de iepen". Elle y traitait de l'isolation d'un mélange de "*Cephalosporium-Graphium*" provenant du bois décoloré des cerneaux annuels de 1912 et de 1913 de plusieurs petits ormes de rues en alignement infestés par la M.H.O. à Renkum, Pays-Bas, et des cerneaux annuels de 1912 à 1915 d'un grand orme de Rotterdam. Ses photos montrent qu'elle avait le même champignon que celui que Schwarz avait nommé la même année *Graphium ulmi*. La carte de

distribution de Spierenburg en 1921 montrait que la M.H.O. était d'ores et déjà dans toutes les provinces des Pays-Bas. Si on accepte les rapports belges et français sur la M.H.O. (non basés sur la culture), l'épicentre phytologique apparaît avoir été près d'Antwerp. En regard des expériences suivantes avec les niveaux d'étendues de la M.H.O., l'infestation européenne apparaît avoir débuté vers 1900 à 1905. Les événements de la première guerre mondiale n'ont par conséquent aucune relation avec les origines de la M.H.O.

It's widely accepted that Dina Spierenburg, pathologist of the Netherlands' Plantenziektenkundige Dienst (PD) (Plant Protection Service), published the first announcement¹ to the world, of the disease of elms that later came to be called the Dutch elm disease (DED). Her description of events of the year 1920 is dated January 1921 (29).

Far less well known is that only one year later Spierenburg published a *second* article which appears at first glance to have exactly the same title! This report, describing DED events of 1921, is dated January 1922 (30). In the table of contents

This article was begun in 1984 when the author was guest of Rijksinstituut voor Onderzoek in Bos- en Landschapbouw "De Dorschkamp" (Dutch National Research Institute for Forestry & Landscape Planning) and of Vakgroep Fytopathologie, Landbouwhogeschool (Dept. Phytopathology, Dutch Agricultural University) in Wageningen, The Netherlands.

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¹Guyot (16) is sometimes cited as first. But a) Guyot's report appears in an Oct-Dec 1921 issue which was "published Feb. 1, 1922"; b) after describing symptoms he had observed in 1918 on elms in Picardy, in Bussy by the Somme River, Guyot stated that the trouble did not recur in 1919, 1920, or 1921, which is hardly typical of DED, while the Dutch reported a steadily increasing number of afflicted elms and include "a few" 1917 cases; c) Guyot isolated no pathogen; and d) Guyot stressed that there was no epidemic, while Spierenburg described isolating the correct DED pathogen from ever more cases.

Graffin (15) also has been cited as the first to point out that elms were dying, in a letter read to the June 1920 session of the French Academy of Agriculture. But he merely reported the death of a good many elms in the Argonne, which he attributed to the direct toxic influence of war gases used where the trees had been growing. It is indeed possible that DED was in France in 1918, and throughout Belgium as well. Unfortunately, in general reports like this, of dying elms outside The Netherlands in this era, weren't based on isolation of the fungus in culture.

its title ends "II." But that numeral is missing at the top of her actual article, so it doesn't appear in parts that nowadays are photocopied. Here it merely says again, "Een onbekende ziekte in de iepen" [An unknown disease in the elms].

Perhaps because of this identical title, few people ever mention Spierenburg's second article. Hence some vital information came to be overlooked (14).

Cause of DED

Among many possible DED causes suggested in the 1920's, the true cause was quickly found: a fungus. This fungus was described and named (*Graphium ulmi* n.sp.) in 1922 in the doctor's dissertation of Marie Beatrice Schwarz (27). After a storm of criticism, Schwarz's diagnosis was confirmed: Dr. Christine Buisman proved in 1928 that *G. ulmi* indeed caused DED (6, 7).

Spierenburg had reported isolating this same fungus at the PD from dying elms in 1920 and 1921. But she thought that she had here a mixture of two fungi, which she was unable to separate. Based on the two Imperfect-stage (conidial) genera that she saw in her culture plates, she called her mixture "*Cephalosporium-Graphium*" (29, 30).

Spierenburg's text shows that by 1922 she was confident that any tree from which this fungus "mixture" was isolated—from certain brown streaks in the xylem—was afflicted by what she called "**the elm disease**" and that any other dying elm didn't have that disease (30). Clearly she'd learned how to tell DED apart from anything else. In culture, she said, *Cephalosporium* always accompanied *Graphium* but *Graphium* didn't always appear with *Cephalosporium*. She even published macroscopic and microscopic photographs of

typical cultures of *G. ulmi* (30, plates II & III). We now know these are two conidial stages of the **one** DED fungus, and our isolating experiences today are the same as hers were nearly 70 years ago.

But Spierenburg didn't claim she'd proved this fungus caused DED. Inoculations made with her "mixture" gave her only brown streaking in the wood...without wilt in the foliage (30). Indeed in 1930 Spierenburg herself acknowledged that Dr. Schwarz had been the discoverer of the fact that *G. ulmi* was the true cause of DED (31).

Only two years later (1932) Dr. Christine Buisman reported finding the sexual (ascospore) stage of this transferred to *Ceratostomella ulmi* (9). (Later this was transferred to *Ceratocystis ulmi* (Buisman) C. Moreau and to *Ophiostoma ulmi* (Buisman) Nannfeldt.) However, during the first 13 years of DED research, only the conidial stages were known.

Not From China?

Many theories have been advanced to account for the death of vast numbers of elms in northwestern Europe and later on other continents, and for the sudden appearance of *Graphium ulmi*. The cases examined by the PD in 1920 involved the sapwood layers (annual rings) of 1920, 1919, 1918 "and rarely 1917." Some of these trees were said to have been dead several years when sampled in 1920. The event looming largest in every mind at that time, of course, was World War I. Naturally, theories advanced to account for this fungus included several based on events of that war.²

In 1938 Boyce, in his new textbook (5), noted that both Chinese elm, *Ulmus parvifolia*, and Siberian elm, *U. pumila*, showed high resistance

²Among the many other possible causes of elm death (mostly suggested by lay people and largely summarized by Spierenburg (29, 30, 31): {1} direct toxicity of the poison gas clouds, {2} flashing of the battlefield searchlights (both suggested in France), {3} drought, {4} earth fill, {5} sinking of the ground in the vicinity of mines (suggested in Limburg Province, The Netherlands), {6} bacterial infection, {7} heavy bloom of elms the previous spring (both suggested in Germany), {8} fumes from the wartime burning of soft or brown coal for fuel, {9} incompletely burned gases from many motors, {10} higher water table, {11} lower water table, {12} breakage of buried electric cables that let electricity spread into the ground, {13} use for road construction of sand from the Maas River whose drainage basin included the great battlefields of the War, {14} dumping of salt water ["pekkel"] from ice wagons, {15} pouring of salty potato-water onto the elm planting sites, {16} leakage of illuminating gas from underground pipes, and so forth.

Interestingly, the presence of elm bark beetles was recognized at once (29). And it was known that elm bark beetles had been present in vast numbers under the bark of many dying elms that presumably had been weakened by root injuries inflicted during the laying of the new gas pipes for city-street illumination plus smothering by the leakage of gas in London and Antwerp in the 1840's (31). But these beetles were still considered only secondary, merely attracted to already dying trees, until in 1929 Betrem (4) suggested that they might carry the DED fungus and in 1935 Fransen and Buisman (12) proved that this was indeed their role.

to DED. In countries where certain plant pathogens are long endemic, their diseases are often mild and host resistance is often high. Boyce therefore put forth the suggestion that the DED fungus MIGHT have been endemic in China and that Chinese labor units MIGHT have brought it with them to France during World War I. This idea was given wide attention, met with much credulity and still persisted as late as 1978 (19).

Two objections have been made to this Chinese theory. In the late 1930's and early 1940's, when Japan controlled large parts of China, a Japanese mycologist, Dr. Kiyowo Aoshima, traveled in the occupied areas, studying staining fungi in the genus *Ceratocystis*. In his reply to my inquiry, he wrote to me in 1959 that in China that he had never found either the DED fungus or DED's brown streaking in elm wood (1). In 1968 I reported this communication to the First International Conference on Dutch Elm Disease, in Ames, Iowa (18).

Secondly, Gibbs (13) rightly pointed out in 1980 that those Chinese workers (who first arrived in 1916) had remained in French factories, well back of the front lines. But DED broke out in what is now called the Benelux area northeast of the battle zone, and it was widespread as early as 1917. The Chinese labor-battalion theory didn't fit (although that fact doesn't necessarily exclude a Chinese origin for the fungus).

Not From Wartime Mustard Gas?

Still, might not the war zone itself have been the epicenter and the war have been the instigator? Direct poisoning of elms by war gas could occur only when and where the gas was in use. Guyot correctly objected to the poisoning theory because he had found that as early as 1918 many elm deaths were occurring far from the old areas of combat (16).

On the other hand, mustard gas, lavishly used as a World War I weapon, also is a well-known mutagen. The idea came into DED discussions, that a local fungus could have changed when it was exposed to mustard gas during some battle. For example, a mutant might produce a new sticky substance around its spores, allowing bark-beetle transport. Or a mutant might change or initiate toxin or enzyme production, resulting in a new ability

to cause disease.

The year 1912 preceded both World War I and the advent of mustard gas in battle. True, the German army tried out chlorine gas as early as January 31, 1915 (in Poland). And on the western front they again used chlorine as a poison gas on April 22, 1915 (at the Belgian town of Ypres, south of Oostende and near the French border). But the first war-time use of *mustard gas* came only in July 1917: by the German army at the Third Battle of Ypres (2, 26).

Of course mutations can occur any day, but they're much more common under the influence of such a mutagen. Who could say no? Well, in effect someone HAD ALREADY said "No" ... and long before the theory was propounded!

DED Cases in 1912

This "No" is implicit in Spierenburg's second article (30) where she reported on isolations from samples sent to the PD from many towns during 1921. In this January 1922 article she also told of her studies on DED in established street-trees that had been donated, for her research, by generous owners or community authorities.

By this time Spierenburg had grown very familiar with typical DED wood streaking (brown dots or brown ring or brown semi-circle in cross-section) and with the appearance of isolates of *Graphium ulmi*, although she still hadn't proved that it could cause the wilting.

In her 1922 article, Spierenburg described her experiments on 40 elm trees that formed two rows along the road to a paper factory in the Dutch town of Renkum (the "Ver. Kon. Papierfabrieken der firma Van Gelder Zonen"), whose directors, the Messers Beuker, had given PD permission to experiment on those elms. Renkum is the next town east of Wageningen, a few miles west of Arnhem on the north bank of the Rhine River.

Spierenburg sampled 39 trees (a 40th was cut down before she could study it) for typical brown streaks in the xylem rings. She made borings from the north, east, south and west sides of each trunk, thus taking 156 cores. Of the 39 trees, 31 showed DED streaking. In two elms this streak was in the 1913 wood layer, and in three more elms it was the 1912 wood (Fig. 1).

Spierenburg made separate isolations, culturing from these deeper wood layers. *In a tiny footnote* to her discussion of finding deeply buried discolored streaks in these 5 trees, she wrote, "Cultures from this wood sometimes yielded *Cephalosporium-Graphium*." *In other words, wood of 1912 and 1913 had yielded the DED fungus in her cultures!*

A large elm in Rotterdam, too, showed this typical streaking in its wood layers of 1912, 1913, 1914, and 1915. Spierenburg plated out wood chips from each of these four deeper layers. Alas, for this tree she only reported that "some of the cultures" yielded *Cephalosporium-Graphium*. Of this tree, then, we can be certain only that it became infected with DED no later than 1915. It could have been infected as early as 1912.

So it now seems obvious that World War I could have had nothing to do with calling DED into existence or bringing it to Europe. The disease presumably arose or arrived there soon after the turn of the century.

Can Rings Date Wilt Infections?

But does an early streak really date an infection? It's a common experience, studied for many years by Banfield (3) and the British (24) and thoroughly documented in Peace's DED bulletin (23), that elms of European species often recover repeatedly before a final DED infection kills them. Streaking remains in the sapwood of each year they'd been infected.

Jones, Krass & Sava in 1976 isolated *C. ulmi* from brown streaks as far back as the 1958 wood from *Ulmus procera* in California, 14 years before their "current" infection era (21). IN 1981 Campana, French and Locatelli found *G. ulmi* in deeply buried wood rings (back twenty years, to 1958) in trees of European elm species in southern California (11).

Any conclusion that such elms may have been infected with DED many years earlier was approached with great caution by Tidwell & Sava, who suggested in 1982 that buried brown rings might have resulted from crossing-over in roots or from deep wounding during the inoculation process (33). Root crossing-over to the following year had been shown to be possible in theory

(10). However, in the California case Campana considered that he'd eliminated questions of such transverse growth because he couldn't isolate *G. ulmi* from the 19 years worth of intervening, non-stained tissue layers.

Those of us who work in DED-resistance research have made many thousands of inoculations by pounding a chisel into elm trunks. We may judge for ourselves whether later we found deeply buried discolored rings in our experimental trees. I've never found this, nor has any other

TABEL 1. OVERZICHT VAN DE BOOMEN IN DE LAAN VOOR DE PAPIERFABRIEK TE RENKUM.

Boomnummer.	ZOMERWAARNEMINGEN.						WINTERWAARNEMINGEN.			
	Uiterlijk in zomer 1921.	Windstrijping, ziekte zich val. heeft uitgedrukt. Zomer 1921.	Op staan uitgebogen lopen.	Stamklover aantasting.	Kromming der takjes.	Inwendige ver. verkleuring.	Verkleuring in de laatste jaarringen.			
							N.	O.	Z.	W.
1	G	—	—	—	—	1912	⊙	■	⊙	—
2	1)	—	—	—	—	—	—	—	—	—
3	‡ ²⁾	—	—	■	■	■	■	■	■	■
4	G	—	—	■	—	■	⊙	■	—	—
5	■	N.	—	■ b†	—	1912	—	■	—	—
6	■	N.W.	—	■	—	■	⊙	—	—	—
7	■	—	—	■ b†	—	1913	—	⊙	—	■
8	‡	—	—	—	t■	1913	■	■	■	■
9	⊙	N.O.	—	—	⊙	—	—	⊙	—	—
10	G	—	—	—	⊙	1912	—	⊙	—	⊙
11	‡	—	—	—	t■	—	■	■	■	⊙
12	■	N.W.	—	■	■	■	■	⊙	■	■
13	■	N.W.	—	■	■	■	—	⊙	—	⊙
14	‡	Z.W. en N.	—	—	■	■	—	—	—	■
15	■	—	—	—	—	■	⊙	⊙	■	⊙
16	■	N.W.	—	—	⊙	■	—	⊙	—	—
17	■	N.O.	—	■	⊙	■	⊙	⊙	⊙	⊙
18	■	—	■	■ b†	■	—	■	■	■	■
19	■	N.O.	■	—	■	■	■	■	■	■
20	■	Z.W.	■	■	■	■	■	■	⊙	■
21	‡	Z.W.	■	—	⊙	■	⊙	⊙	⊙	⊙
22	■	Z.W.	—	—	■	■	■	■	⊙	■
23	‡	—	—	—	■	■	■	■	⊙	■
24	G	Z.	—	—	⊙	■	⊙	■	■	■
25	‡	—	—	—	⊙	■	⊙	■	⊙	■
26	G	—	—	—	⊙	■	—	—	—	—

1) Omgehakt, zie blz. 16 en tabel 2, blz. 18.
2) De breuken geven aan, welk gedeelte van de boom ziek was.

Figure 1. A small part of Spierenburg's Table 1 about her 1921 research in Renkum (30). The black diamond in an open square means "severely affected (discolored, etc.)." The "G" in column 2 means the tree looked healthy ("gezond") in 1921. A fraction tells how much of a tree looked sick. But the vital thing is those years: 1912, 1913!

DED scientist I've asked (unless such a tree was a survivor from a DED inoculation in the year of that earlier streak). Normally the recent ring is the only wholly functional one, and for a streak of any size to develop you'd need a rather fast sap movement. So it's up to any who disagree, to prove their point by making *G. ulmi* inoculations that DO consistently cause brown streaks in a single ring of many years earlier than the inoculation.

Other Early Reports

Peace (23) reported that brown stains were found in 1912, 1913 and 1914 wood in one elm in England. But he said the DED fungus hadn't been isolated from it. He didn't say whether culturing was actually tried. (*G. ulmi* was first confirmed in England in 1927.) Liese reported that xylem discoloration had been found as early as 1900 in elms in Germany (22).

But local steaking in elm wood can also be caused by bacteria, as Buisman (8) pointed out. Also, systemic vascular streaking in elm that is caused by *Verticillium* infection can't be told apart from DED streaking without culturing (8).

It's when we look in the Belgian literature that we see some faint traces of circumstantial confirmation. True, DED isn't mentioned in the thorough study of Huberty (1904-05) on elms and their culture in Belgium (20). But in 1985 Thill cited the opinion of a forest operator that the disease existed in the forests of Colfontaine, Belgium, in 1914 (32).

And as early as 1906 both Severin [in July (28)] and Quievy [in November (25)] reported scolytid beetle breeding attacks that led first to withering and falling of leaves, then to rapid drying out and dying of elms, in the area of Tournay, Belgium. The vivid description doesn't prove the presence of DED but it fits very well with DED's symptoms. We're left wondering, how could these beetles cause this much death without the aid of the fungus?

DED Spread is Slower

In retrospect, previously obscure matters often seem suddenly obvious. Now that we know DED likely existed as early as 1912 in Renkum, and possibly also that year in Rotterdam as well, we recall the slow, gradual way DED usually spreads

from town to town. The same disease took at least 15 years to cross Massachusetts: about 10 miles (15 km) a year, on the average. Apart from sudden leaps to a very few distant spots, like Colorado (1948) and California (1958), it needed a good half century to cross the USA by its usual bark-beetle transport. And this despite the fact that in USA it's known to have used automobile transport for parts of its journey (17).

So we also suddenly realize that Spierenburg's 1921 map (30) shows *far too many* infested Dutch towns, over *far too large* an area of The Netherlands (Fig. 2), for any 3-year-old DED infestation. Spierenburg's Plate I shows that in 1921 the DED fungus already pervaded The Netherlands (although still a bit uncommon in the north). To this probably must be added the further large areas supposedly infested in neighboring Belgium, France, and Germany. If DED arose or arrived in 1917 at *one single spot* in western Europe, it's hard to conceive that it then should manage to be throughout this huge area only four years later.

Probably even 1912 wasn't the first year, either. It would be too much of a good thing, after all, if those few trees Spierenburg just happened to be given, for experiments, should chance also to have been the very first DED cases ever. Then again, there's that large elm in Rotterdam about 60 miles to the west (and upwind from Renkum).

We need raise no eyebrows at Renkum's paper industry; we needn't imagine mutagenic effects of any pollutants from their technology. It's likely that a tree along this street was inoculated during feeding by bark beetles that came there from DED-diseased elms elsewhere. Many elms had died in nearby Arnhem only a few years before.

Along this particular Renkum street root-grafting likely played a role in further DED spread. Spierenburg's Table 1 shows that the five trees with early infections are all near one another (Fig. 1).

Did DED Come From Elsewhere?

Another possible approach to the question of DED origin has had little attention. *Ulmus*, after all, isn't native anywhere in the southern hemisphere. A fungus from an area without wild elms (and with few planted elms) might have been brought to Europe about the turn of the century. Here it

would find abundant host trees in which it could cause a disease out of all proportion to its earlier, possibly saprophytic, custom.

In DED's inexorable march in the northern hemisphere, it's always been a newcomer. We may be fairly confident in assuming that either: a) certain strains of this pathogen—at least as we know it now—were indeed entirely new to the world when it first appeared in the Benelux area, or b) DED arose by the bringing together of a parasite, a vector insect, and a host plant which previously hadn't met, maybe because formerly they occurred only in different parts of the world.

At present we've no direct evidence whatever to support this second possibility. For present we've no direct evidence whatever to support this second possibility. For the fungus to have arrived from such an area, a port is an essential ingredient of course, and Antwerp fits this picture just as well as Rotterdam. But the fact that (until 1990 in New Zealand) there've been no reports as yet of DED cases among the imported elms used for street or ornamental plantings in southern-hemisphere countries argues against this hypothesis.

The French studies were without evidence from fungus cultures, but Guyot (16) pointed out there were brown rings in the wood of the dying trees! So it's reasonable for us to suppose that DED really was in France, too, as early as 1918. If we then work backward in space and time, the focus seems to lie somewhere between Picardy and Tilburg and the time seems to be considerably before 1912. *This exercise brings us to the general area of Antwerp and to the years 1900-1905.*

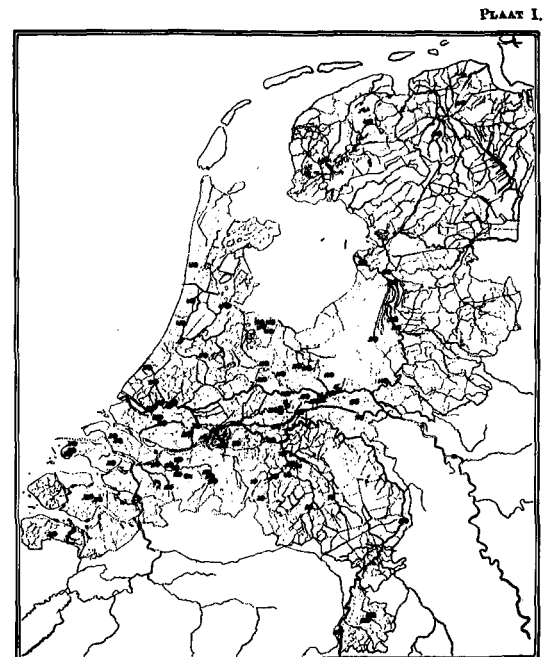
A Valid Effect of War

There's still a way that World War I might have been involved. The war distracted people's attention from all other matters, certainly in German-occupied Belgium and likely enough even in an uninvaded country like The Netherlands. DED would kill only a few elms at first. Under the circumstances dying elms might well be left standing. People were just too busy, too troubled.

War itself undoubtedly killed many trees, including elms. So the elm bark beetle population would greatly increase in battle-devastated areas.

PLAATSEN OP DE KAART VOORKOMENDE.

39. Aarlanderveen.	74. Gorkum.	15. Oudend Bosch.
76. Ammersoelen.	30. 's-Gravendeel.	32. Oud-Beijerland.
42. Amsterdam.	38. 's-Gravenhage.	
57. Apeldoorn.		50. Peize.
	26. Haamstede.	33. Poortugaal.
49. Bedum.	3. Haaren.	
58. De Bildt.	41. Haarlem.	27. Renesse.
4. Boxtel.	61. Hardenbroek.	65. Renkum.
19. Breda.	9. Helmond.	36. Rotterdam.
71. Buren.	7. 's-Hertogenbosch.	17. Rozendaal.
25. Burg.	40. Hillegom.	
46. Bussum.	2. Hilvarenbeek.	60. Scherpenzeel.
	82. Hoensbroek.	35. Schiedam.
43. Castricum.	16. Hoeven.	5. St.-Michielsgestel.
	62. Houten.	48. Sneek.
73. Deil.	45. Huizen.	81. Staatsm. „Emma“.
37. Delft.		14. Standaardbuiten.
56. Deventer.	69. Ingen.	
55. Diepenveen.		51. Ten Boer.
64. Dieren.	52. Kampen.	77. Tiel.
13. Dinteloord.	23. Kapelle.	1. Tilburg.
28. Dirksland.	68. Kesteren.	
31. Dordrecht.	11. Klundert.	80. Venlo.
10. Dussen.	18. Kruisland.	34. Vlaardingen.
63. Ede.	47. Laren.	66. Wageningen.
8. Eindhoven.		54. Windesheim.
78. Elst. (O.-B.).	44. Naarden.	59. Woudestad.
	79. Nijmegen.	70. Wijk bij Duurstede.
12. Fijnaart.		
	83. Maastricht.	21. IJzendijke.
72. Geldermaken.	29. Melissant.	
6. Gemonde.		75. Zalt-Bommel.
20. Ginliken.	24. Oosterland.	53. Zwolle.
22. Goes.	67. Opheusden.	



Tekening: W. H. Rutsch, Wageningen.

Figure 2. Culture-confirmed distribution of DED in The Netherlands in 1921, as recorded by Spienburg in her Plate I (30).

Meanwhile the local population of elm bark beetles would expand also in the new DED areas. Soon this pre-war disease began to claim not just a few dozen victims each year, but thousands and then hundreds of thousands.

Final answers will require more evidence, but the Spierenburg data are significant. They establish an earlier date than has been generally realized for the presence of DED in Europe.

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ABSTRACT

ILES, JEFF. 1989. **The case against tree topping**. Grounds Maintenance 24(6):51, 74.

The practice of topping—also called heading, stubbing or dehorning—involves the drastic removal of large branches with little regard for location of the pruning cut. Professional arborists and other tree care practitioners now realize that the well-intentioned practice of topping can create a host of problems for trees and people who co-exist with them. Proper early training, selective branch thinning, or entire tree removal are more favorable alternatives. By removing a major portion of the tree canopy, the delicate balance between foliage and the rest of the tree is upset. These imbalances can lead directly to decline and death, or predispose trees to other problems, with death the inevitable result. Large branch stubs that result from topping are open invitations to insects and wood-rotting pathogens. Regrowth resulting from topping is also succulent and more susceptible to attack from insects and disease pathogens. Topping also disfigures the tree and ruins its aesthetic value in the landscape.