

Proceedings of the Iowa Academy of Science

Volume 46 | Annual Issue

Article 14

1939

Diseases of Cultivated Lupines

C.J. Gould Jr.
Iowa State College

Copyright © Copyright 1939 by the Iowa Academy of Science, Inc.
Follow this and additional works at: <https://scholarworks.uni.edu/pias>

Recommended Citation

Gould, C.J. Jr. (1939) "Diseases of Cultivated Lupines," *Proceedings of the Iowa Academy of Science*: Vol. 46: No. 1 , Article 14.
Available at: <https://scholarworks.uni.edu/pias/vol46/iss1/14>

This Research is brought to you for free and open access by UNI ScholarWorks. It has been accepted for inclusion in Proceedings of the Iowa Academy of Science by an authorized editor of UNI ScholarWorks. For more information, please contact scholarworks@uni.edu.

DISEASES OF CULTIVATED LUPINES

C. J. GOULD, JR.¹

The cultivation of lupines has been practiced in Europe for over a thousand years. Impoverished Greeks and Romans used lupine meal in their bread and farmers have long grown the plants for forage and green manure. Extensive cultivation of these plants would be expected because the seed, which consists of from 30 to 40 per cent protein, is high in food value and because relatively high yields can be obtained even under unfavorable conditions. Stock poisoning, however, has frequently resulted from consumption of these plants owing to the presence of harmful alkaloids. Such substances could be removed from the seeds, but only by soaking in water. Since this procedure required considerable time and energy and since the other parts of the plant could not be treated in this way, lupines have not been grown extensively in the past.

Within the last fifteen years German and Russian workers, by selecting from millions of plants, have obtained alkaloid-free (sweet) varieties of white (*Lupinus albus* L.), blue (*L. angustifolius* L.) and yellow (*L. luteus* L.) lupines. Since the development of these new varieties, the cultivation of lupines has been increasing rapidly in Germany, Russia and neighboring countries (18).²

Lupines, except as ornamentals, have not found particular favor in the United States for two reasons: (1) the plants contain poisonous alkaloids, and (2) they often grow poorly. Now that alkaloid-free lupines are available, interest in their cultivation is rapidly increasing in this country.

In personal and written communications to the writer, the statement has been made frequently that stands are often very uneven in growth and that on many plants the central leaves become yellow and drop. Such a condition appears to be widespread in the United States on greenhouse-, nursery-, garden-, and field-grown lupines. In nurseries, entire stands of the flowering lupine, *L. polyphyllus* Lindl., frequently succumb. These conditions have led to a survey of the literature relating to diseases of lupines.

Brief accounts of the economic importance, range and symptoms

¹ The author is grateful to Dr. I. E. Melhus for his encouragement and constructive criticism throughout the preparation of this article.

² Numbers within parentheses refer to articles listed in the bibliography.

are given for the more important diseases, but only essentially significant points for the less important ones. Control measures for the various diseases are summarized at the end of the article.

MAJOR DISEASES OF LUPINES

Leaf Spot caused by *Ceratophorum setosum* Kirchn.

Leaf spot caused by *Ceratophorum setosum* (L³)³ is commonly found on a number of lupine species in Europe and in India, but it has not yet been reported in the United States. An attack of the fungus usually spreads rapidly throughout a planting, starting on the basal leaves and spreading to the developing terminal leaves. The first symptoms appearing on leaves and pods are small spots which rapidly enlarge, and become circular or irregular with a zonate appearance, and may vary in color from reddish-brown to dark brown. Coalescence of the lesions usually occurs on the leaves which soon shrivel and fall. Plants on which leaf attack is severe are usually stunted.

Fungus penetration of seeds followed by the formation of necrotic lesions occurs in young pods but not in mature pods. Infected seed lots germinate poorly (4, 7, 11, and 13).

Stem Necrosis caused by *Ascochyta* species

Several *Ascochyta* species have been reported attacking lupines in the United States and other parts of the world, but the most common species is apparently *Ascochyta pisi* Lib. (L¹). Primary attacks by this organism generally are manifested by the appearance at the crown of lesions which are variable in size, sunken and brown and which soon become speckled with numerous dark brown pycnidia. Stem and branch infections usually result in a gradual wilting of the upper parts of the plant. The leaves lose their green color, become yellow and eventually drop, leaving the crown of the plant bare. Plants vigorous at the time of the attack will remain erect until death, but weak plants may collapse at the point of infection (2 and 7).

Damping-off and Wilting caused by *Rhizoctonia solani* K.

Rhizoctonia solani (L¹) attacks on lupines have been reported in the United States, Europe and other localities. Infection of seedlings by this fungus usually results in damping-off while infection of older plants produces stunted individuals with light-yellowish foliage. Affected roots are generally dry-rotted, tough

³ Causal agents marked L¹ occur on lupines in the United States; those marked L² are found on plants other than lupines in this country; and those marked L³ occur on lupines only in foreign countries.

and dark brown, but may exhibit only local lesions. On such plants the leaflets eventually wilt, dry up and fall, leaving bare erect petioles. Young leaves may become flaccid and wither. The development of these symptoms may take from one to several days.

The perfect stage appears on the stem portions during moist weather. Inoculation tests have demonstrated the presence of biological races (10, 12, and 15).

Wilting caused by *Sclerotinia sclerotiorum* (Lib.) Mass.

Sclerotinia sclerotiorum (L¹) has been reported on lupines in the United States, Africa and also in Germany where it is apparently quite widespread. Following infection by this fungus, primary symptoms appear on the stem in the form of glassy discolored areas which later become white. Plant parts above this infection wilt and die. Lesions similar to those on stems appear on infected pods and if the seeds are immature, they too become infected. Black sclerotia may develop within the infected portions during dry weather and on their surfaces in moist weather (7).

Wilting caused by *Thielavia basicola* (B. and Br.) Zopf.

Injury to lupines by *Thielavia basicola* (L¹) has been reported in the United States, Holland and Italy, but the greatest damage apparently has occurred in Germany where in 1937 the organism was reported as becoming more destructive. Infection of seedlings by this fungus usually results in rotting of the crown, followed by collapse and decay of the plant. Infected roots of older cultivated plants usually become brown, shrunken, and fragile. Such plants may be stunted, or may wilt and decay.

Injury from attacks by this organism appears to increase in direct proportion to increased soil moisture and in inverse proportion to the organic content of the soil. Experiments by certain workers indicate the existence of biological strains of this fungus (7, 12, and 19).

Lime-induced Chlorosis

Foliage chlorosis (L²), caused by the presence of excessive amounts of lime in the soil, has resulted in considerable injury to lupines in Germany and Czechoslovakia and may be one cause of the difficulty of growing lupines in this country. The severity of the attack increases in direct proportion to a rising lime content of the soil.

The first symptoms of this disease are retarded growth of roots and shoots and rapid chlorosis of all parts of the young leaves

except the midribs. Later the midribs turn yellow, the growing shoot tip withers, and the plant dies.

The high calcium content in the soil decreases absorption and utilization of iron by the plant. Hence, lime-induced chlorosis is actually a result of iron deficiency (8 and 16).

Sore-shin caused by pea-virus No. 2

Sore-shin (L¹) of lupines, a virus disease, has been of considerable importance in several localities of New Zealand. The causal agent, pea-virus No. 2., sometimes causes the death of entire stands of lupines as well as of several other legumes in that country.

The first visible symptoms of this disease are a slight stunting of the plant associated with a curling of the growing point to one side and the appearance on that side of a light brown streak which extends the entire length of the stem. Leaf symptoms vary somewhat according to the species attacked, but in general, young leaves at first wilt, then turn black, while older leaves become tinged with purple, change to yellow and finally fall. Flowers frequently fail to develop on plants infected when immature while on older plants the young pods are black or spotted with black and droop instead of remaining oblique to the stem. Growth soon ceases after infection, the necrotic area spreads until the entire stem becomes brown, then black, the roots decay and the plant dies.

Experiments show that aphids may transmit the virus but no evidence has been found of transmission by seed. Although this virus attacks many lupine species, the common flowering type, *L. polyphyllus*, seems to possess a moderate degree of resistance (1, 5 and 14).

Browning caused by cucumber virus No. 1

The most destructive virus disease of lupines in Germany is that produced by the browning-virus (L²) which certain workers believe to be the same as that causing the New Zealand sore-shin disease. Symptoms on infected plants are very similar to those previously described for sore-shin (14 and 17).

MINOR DISEASES OF LUPINES

Several other diseases of lupines have been reported but most of them have remained of minor importance. Among these are diseases caused by the following organisms:

Bacillus lathyri M. and T. Primary symptoms resulting from infection by *Bacillus lathyri* (L²) are rather similar to those of "sore-shin." Narrow necrotic lesions which range in color from brown to brownish-purple and are

sometimes longitudinally split appear on the stem. On the leaflets numerous small sunken black lesions appear and later coalesce. Death and abscission of leaflets may result from severe infection (6).

Bacillus caulivorus P. and D. and *B. phytophthorus* App. Infection by *Bacillus caulivorus* (L²) or *B. phytophthorus* (L²) results in the blackening and rotting of the roots, followed by a yellowing, wilting and withering of the shoots (7).

Botrytis cinerea Pers. In New Zealand blue lupines have suffered severely from attacks by *Botrytis cinerea* (L¹). Following infection by this organism primary symptoms develop on the stem, usually at the crown, in the form of slightly sunken lesions, varying from one-half to nine inches in length and from gray-green to brown in color. These lesions rapidly enlarge and usually girdle the stem in a short time. In moist air the fungus sporulates on the stem, forming a mouse-gray mycelial and sporogenous mass. Sclerotia later develop on the surface and produce conidia the following spring (2 and 7.).

Collybia velutipes Fr. and *Pleurotus ostreatus* Jacqu. *Collybia velutipes* and *Pleurotus ostreatus* (L¹) have been reported as very destructive wound parasites of *Lupinus arboreus* Sims (tree lupine, valuable as a soil retainer on sand dunes) in the vicinity of San Francisco. Infection occurs through mechanical wounds or insect tunnels and results in the death of part or all of the shrub, accompanied or followed by sporophore formation. Within the stem the wood becomes slightly discolored and rotted, later appearing light brown in color (9).

Cryptosporium leptostromiforme Kuhn. Infection by *Cryptosporium leptostromiforme* (L³) often results in premature death of lupines by the formation of stem lesions which at first appear bright, and later become brownish and often extensive. In these lesions the fungus fructifications appear later as elongated black pustules (7).

Erysiphe martii Lev. Attacks by *Erysiphe martii* (L¹) seldom cause severe injury to lupines. Fungus signs on the leaf surface appear as a typical powdery mildew, first whitish and tomentous and later speckled with black perithecia (7 and 12).

Fusarium vasinfectum Atk. Most recent reports of fusarial infections indicate the secondary nature of their attacks, but *Fusarium vasinfectum* (L²) apparently can infect primarily through root injuries. It destroys the cortex and grows into the vascular bundles of the stem, both of which tissues turn brown. Wilting follows the plugging of the xylem cells and the plant dies (7).

Macrosporium sarcinacforme Can. Attacks by *Macrosporium sarcinacforme* (L²) have caused considerable damage to lupines in Germany. Leaf infection is characterized by the formation of numerous lesions which are circular to oval in shape, one to four millimeters in diameter, bluish-gray to grayish-brown in color and sometimes surrounded by narrow pale or yellowish-green zones. Such lesions frequently coalesce. Later, similar lesions which are reddish-brown appear on the stem and pods, accompanied by a twisting of the growing tips. Severely infected plants usually die (11).

Pythium debaryanum Hesse. *Pythium debaryanum* (L¹), the causal agent of damping-off and root necrosis of many plants in the United States,

has been reported causing the development of similar symptoms in Texas bluebonnets (*Lupinus texensis* Hook.). Infected plants usually lose their normal green color, wilt and die within ten days (15).

Tomato-spotted virus. Tomato-spotted virus (L¹) has attacked lupines in the United States and in England. In warm regions, symptoms of the disease appear on the leaves as necrotic concentric rings or circular spots, accompanied by a slight amount of distortion and deformity. In cool regions severe necrosis in the form of dieback or streak may result in death of the plants (3 and 14).

CONTROL

In the reviewed literature several control measures were suggested for many of the above diseases, but only a few seemed to be based upon actual experimental work or extensive observations. Those that seemed justified on the above basis are given below. For the most part they refer to the use of resistant varieties.

In regard to attack by *Ceratophorum setosum*, observations by Green (4) and Siemaszko (13) indicate that *Lupinus angustifolius* and *L. luteus* are resistant; *L. mutabilis* Sweet, *L. polyphyllus* and *L. hirsultus* L. are of medium resistance, and *L. albus* and *L. cytisoides* Agardh are susceptible.

Under field conditions Roabe and Sengbusch (12) noticed that *Lupinus mutabilis* was characterized by considerable resistance to attack by *Thielavia basicola*, while *Lupinus luteus*, *L. angustifolius* and *L. albus* were more susceptible.

Based upon experimental work Parsche (8) found that *L. luteus* was most susceptible to a high soil-lime content, *L. perennis* L. was most resistant, and *L. albus* and *L. angustifolius* were intermediate in resistance.

Gardner and Whipple (3) obtained control of the tomato-spotted virus on lupines in commercial greenhouses by frequent fumigation and roguing.

Rhoads (9) observed that *L. chamissonis* Esch. was highly resistant to attack by *Collybia velutipes* and *Pleurotus ostreatus* whereas *Lupinus arboreus* was quite susceptible.

PERTINENT LITERATURE

1. CHAMBERLAIN, E. E. Pea mosaic. Host range and methods of transmission. N. Z. Journ. Sci. Tech. 18: 544-556. 1936.
2. CURTIS, K. M. Two fungal diseases of the blue lupin. N. Z. Journ. Agric. 26: 240-246. 1923.
3. GARDNER, M. W. AND O. C. WHIPPLE. Spotted wilt of tomatoes and its transmission by thrips. (Abs. in Phytopath. 24: 1136. 1934.)
4. GREEN, D. E. A lupin disease due to *Ceratophorum setosum* Kirchn., a fungus new to Great Britain. Journ. Roy. Hort. Soc. 58: 144-145. 1933.
5. NEILL, J. C., R. M. BRIEN AND E. E. CHAMBERLAIN. "Sore-Shin": a virus disease of blue lupins. N. Z. Journ. Agric. 49: 139-146. 1934.
6. PAINE, S. G. AND W. F. BEWLEY. Studies in bacteriosis. VIII. Further investigations of the 'stripe' disease of tomato. Ann. Appl. Biol. 10: 89-95. 1923.
7. PAPE, H. Krankheiten und Schädlinge der Lupine. Illus. Landw. Zeit. 47: 316-318. 1927.
8. PARSCHÉ, F. Über die Kalkchlorose der Lupine. Z. PflErnähr. Düng. A. 41: 282-312. 1935. (Abs. in Rev. Appl. Myc. 15: 298-299. 1936.)
9. RHOADS, A. S. The pathology of *Lupinus arboreus*, with special refer-

- ence to the decays caused by two wound-parasites-*Collybia velutipes* and *Pleurotus ostreatus*. *Phytopath.* 11: 389-404. 1921.
10. RICHTER, H. Fusskrankheit und Wurzelfäule der Lupine. *Zbl. Bakt. Abt. 2.* 94: 127-133. 1936.
 11. RICHTER, H. Blatt-, Stengel- und Hülsenflecken an Lupinen. *NachrBl. dtsh. PflSchDienst.* 17: 77-80. 1937. (Abs. in *Rev. Appl. Myc.* 17: 184-185. 1938.)
 12. ROABE, A. AND R. VON SENGBUSCH. Züchterish wishtige Beobachtungen an einigen Lupinenarten. *Züchter* 7: 244-248. 1935 (Abs. in *Rev. Appl. Myc.* 15: 100. 1936.)
 13. SIEMASZKO, W. Phytopathologische Beobachtungen in Polen. *Centralbl. für Bakt. Ab. 2.* 78: 113-116. 1929 (Abs. in *Rev. Appl. Myc.* 8: 630. 1929.)
 14. SMITH, K. M. A textbook of plant virus diseases. 1937.
 15. TAUBENHAUS, J. J. AND W. N. EZEKIEL. On a new damping-off disease of Texas bluebonnets. *Mycologia.* 24: 457-459. 1932.
 16. VAN GENNEP, V. C. De symptomen van physiologische ziekten van *Lupinus luteus* L. Thesis, Univ. of Utrecht, 107 pp. 1936 (English summary).
 17. VON KÖHLER, E. Weitere Untersuchungen über das Virus der Lupinenbräune. *Z. Pflkrankh.* 47: 87-97. 1937.
 18. VON SENGBUSCH, R. The breeding of sweet lupins. *Herbage Rev.* 6: 1-9. 1938.
 19. LUPINENWELKE UND IHRE BEKÄMPFUNG. *Dtsch. landw. Pr.* 64: 463. 1937. (Abs. in *Rev. Appl. Myc.* 17: 115-116. 1938.)

DEPARTMENT OF BOTANY,
IOWA STATE COLLEGE,
AMES, IOWA.