Seasonal Changes in Northern Red Oak Susceptibility to *Phytophthora cinnamomi*

C. ROBIN, F. DUPUIS, and M. L. DESPREZ-LOUSTAU, Station de Pathologie Végétale, Institut National de la Recherche Agronomique, Centre de Bordeaux, BP 81, 33883 Villenave D'Ornon, France

ABSTRACT

Robin, C., Dupuis, F., and Desprez-Loustau, M. L. 1994. Seasonal changes in northern red oak susceptibility to *Phytophthora cinnamomi.* Plant Dis. 78:369-374.

Seasonal changes in susceptibilty to ink disease, caused by *Phytophthora cinnamomi,* were evaluated in northern red oaks (*Quercus rubra*). At monthly intervals, direct trunk inoculations were performed in mature trees, and disease severity was assessed 1 mo later. Concurrently, bark strips were removed from the trunk of each tree, inoculated with *P. cinnamomi,* and incubated in standardized conditions. Analysis of variance showed that, both on unexcised and excised tissues, lesion development significantly changed depending on the date of inoculation. An effect of temperature on lesion development in situ was observed; no lesions were induced from December to February, and the greatest susceptibility of red oaks was observed in June. This seasonal pattern of susceptibility could not be accounted for simply by a climatic effect on the fungus development, because the lesion development in excised bark tissues was least from October to January and increased until May. Throughout the experiment, the relative water content (RWC) of the bark was measured. Collar bark tissues were more resistant to *P. cinnamomi* and more moist than tissues removed at a 2-m height. The RWC also varied during the year. These changes could not be related to climatological data, but the lesion linear extension in vitro was significantly related to this parameter, which could reflect physiological changes in the living bark. Analysis of variance also demonstrated tree-to-tree variability of red oak susceptibility. This could be at least partly explained by differences in tree phenology, because the correlation between phenology and bark susceptibility in early spring was significant and positive.

Additional keywords: cortical cankers

Northern red oak (*Quercus rubra* L.) has been an ornamental tree in France for two centuries. It is now the principal hardwood species used for afforestation. However, red oak is highly susceptible to ink disease, caused by *Phytophthora cinnamomi* Rands (15). In plantations in southwestern France, this fungus is the principal threat to this forest species. Disease impact can be high—up to 67% of trees are infected in some plantations (10). The fungus does not cause tree decline, but it leads to important timber yield losses because of cankers which develop several tens of centimeters up the trunk.

Although infections by *P. cinnamomi* occur in roots, the most serious damage results when the pathogen colonizes the trunk cortical tissues, especially the secondary phloem and the cambium. In response to infection, some xylem callus-curls appear in the margins of the necrotic cambium, and phenolic compounds ooze out of the cankers (15). This latter response, common in oaks after any bark injury, is observed in ink-diseased trees only in early spring, which suggests that the most detrimental damage to the tree starts at this time. Dendrochronological studies of red oak trunk cankers have shown that lesions caused by *P. cinnamomi* principally develop before or during the annual formation of spring vessels (20,21). Delatour (4) described a seasonal fluctuation in lesion development in inoculated stems of 2-yr-old red oaks. This author also reported a lag in lesion development in the plants inoculated and stored outside compared to those located in a glasshouse. In natural conditions, environmental factors also affect the development of lesions caused by *P. cinnamomi* in red oak trunks (10,21). These observations and results suggest a seasonal pattern of susceptibility to ink disease in red oaks.

Host resistance is presently the most promising means of control of ink disease. Breeding for resistance in red oaks has been investigated, and techniques were developed for screening tests (18). In the future, chemical control may be an additional approach, since injections or sprays of phosphorous acid reduce the severity of several tree diseases caused by *P. cinnamomi* and other *Phytophthora* spp., such as avocado root rot or crown rot of almond and cherry trees (30,31). To further exploit these strategies, research is needed concerning environmental and host factors which might influence the development of the trunk lesions. An accurate knowledge of the greatest susceptibility period for trees is necessary to allow effective timing of inoculations in breeding tests and of fungicide applications. Our study investigated the seasonal variation in susceptibilty to ink disease in *Q. rubra.* This paper reports the results of in situ and in vitro monthly inoculations of red oaks by *P. cinnamomi.*

Accepted for publication 1 November 1993.

© 1994 The American Phytopathological Society

MATERIALS AND METHODS

Experimental design. Experiments were performed in a 45-yr-old northern red oak plantation located in Doat (Gers, France) with 32% trunk-infected trees (10). The trees were dominant or codominant, with no lesions visible on trunks. They were marked for felling, and so available for destructive trials. At monthly intervals from 10 April 1990 to 10 May 1991, inoculations were conducted on 10 trees, and bark strips were collected from the same trees for inoculations in the laboratory. Daily rainfall and maximal (Tx) and minimal (Tn) temperatures were obtained from the nearest weather station, which was located in Salles d'Armagnac (15 km away) and operated by the Meteorologie Nationale. Averages of these data, recorded between two consecutive inoculation dates, were calculated (Fig. 1A). The number of frost days ($Tn < 0$ C) and of high-temperature days ($Tx > 25$ C) which occurred between the same periods are illustrated in Fig. 1B.

Inoculations. Trunks were inoculated as previously described with an isolate of *P. cinnamomi* from red oak (19). The fungus was grown on V8 agar medium (containing 20% v/v of V8 juice, 2 g/L of $CaCO_3$, and 20 g/L of agar). Disks (1 cm diameter) of 5-day-old cultures were inserted into wounds of the same size in the phloem, 1.50 m above ground level. The wounds were covered by the removed bark disk, wet sterilized cotton, aluminum foil, and plastic wrap. After 1 mo, the cortical tissues were peeled off, the half-lesions that developed in cambium above and below the inoculation point were measured, and the average half-lesion length was calculated for each tree.

At the time of each trunk inoculation, three cortical tissue strips (4.5 cm long × 1.5 cm wide) were cut from the bark of the same trees, protected from desiccation with a plastic film, and stored in an icebox until their inoculation later in the day. The removed strips comprised all the tissues between the dead outer rhytidome and the xylem, although it is likely that the cambium was severely damaged by removal from the tree. In

the laboratory, the bark strips were wounded on the inner face and at one side by removing a 5-mm-diameter disk of tissues with a cork borer. A similarly sized culture disk of *P. cinnamomi* as previously described was placed in this wound and covered by the bark disk. The bark strips were placed on a wet sponge sheet in a plastic box in which a high level of humidity was maintained. A benomyl solution (10 mg/L) was then sprayed into this moist chamber. After 3 days at 25 C in the dark, the lesions that developed in the phloem were measured. In previous experiments, *P. cinnamomi* was reisolated in all lesion tissues from both the inoculated trunks and the excised barks (18). In both experiments the linear lesion extension was calculated.

Bark strips could not be removed at the same height as tree inoculations, as this would cause tree girdling. Consequently, in April and May 1990, the strips were excised at the ground level. To determine whether the height of collection influenced the development of lesions in bark strips, 0-, 1-, and 2-m height collections were compared in June 1990. The following months, all strips were removed at 2-m height.

Monitoring of bark relative water content and spring flush of the red oaks. A fourth bark strip was similarly removed from each tree at the time of inoculation and used to determine the relative water content (RWC) of the bark. The fresh weight (FW) of the samples was measured in the laboratory. Samples were incubated overnight in water, blotted dry, weighed to determine turgid weight (TW), dried at 70 C, and reweighed to determine dry weight (DW). RWC was calculated according to the formula $RWC = [(FW - DW)/(TW - DW)] \times 100$.

Oak phenology was observed from March to April 1991. Each tree within the experimental plot was examined with binoculars. The growth stages of the top, middle, and lower parts of the canopy were separately assessed by estimating the percentage of tips carrying dormant, turgid, or green buds or fully expanded leaves. These growth stages were assigned the values 0, 1, 3, and 5, respectively. Finally, the phenologic stage of each part was determined as the sum of each growth value multiplied by the percentage of each growth stage.

Statistical analysis. Linear lesion extensions in trunks and excised bark, as well as RWC, were subjected to analysis of variance. Data were processed by the SAS general linear model procedure (22). Since three replicates per tree were performed during the bark tests, the month × tree interaction could be tested by a repeated measures analysis of variance (16). Relationships between variables were investigated by calculating Spearman's coefficient of correlation (26).

RESULTS

Trunk inoculations. Every month, except from December 1990 to February 1991, all tree inoculations resulted in cortical lesions. The largest lesions, on average, resulted from inoculations performed in June 1990 (Fig. 2A). Eight of the 10 trees exhibited maximal susceptibility in June, two in July. The linear lesion extension varied significantly with the time of inoculation ($P < 0.001$) and the individual tree ($P = 0.001$), even when only the months during which lesions occurred in situ were considered. On the basis of the standard errors, differences in tree susceptibility were the least in May 1990 (Fig. 2A).

Bark tests. From March to September 1990 and from February to April 1991, more than 85% of all infected strips exhibited one lesion (Table 1). Length of necrotic tissues could be easily measured as a dark discoloration developed in the phloem below the inoculation point. The mean linear lesion extension was more than 5 mm/day (Fig. 2B). The largest lesions, on average, were observed in May 1990 (0-m height) and in 1991 (2-m height). From October 1990 to January 1991, lesions were not systematically induced. They were diffuse, and phloem discoloration was not intense enough to enable an accurate measure. This resulted in high standard deviations and low average lesion extension. Consequently, although all monthly average extensions were given in Figure 2B, these data were not included in the statistical analysis. A repeated measures analysis of variance was performed on data from June to September in 1990 (Table 2) and from February to May in 1991. Results of both analyses were similar: the three dependent variables, i.e., the bark test date, the tree, and the interaction date × tree, had a significant effect on the daily lesion extension in excised bark. Differences among trees were highly sig-

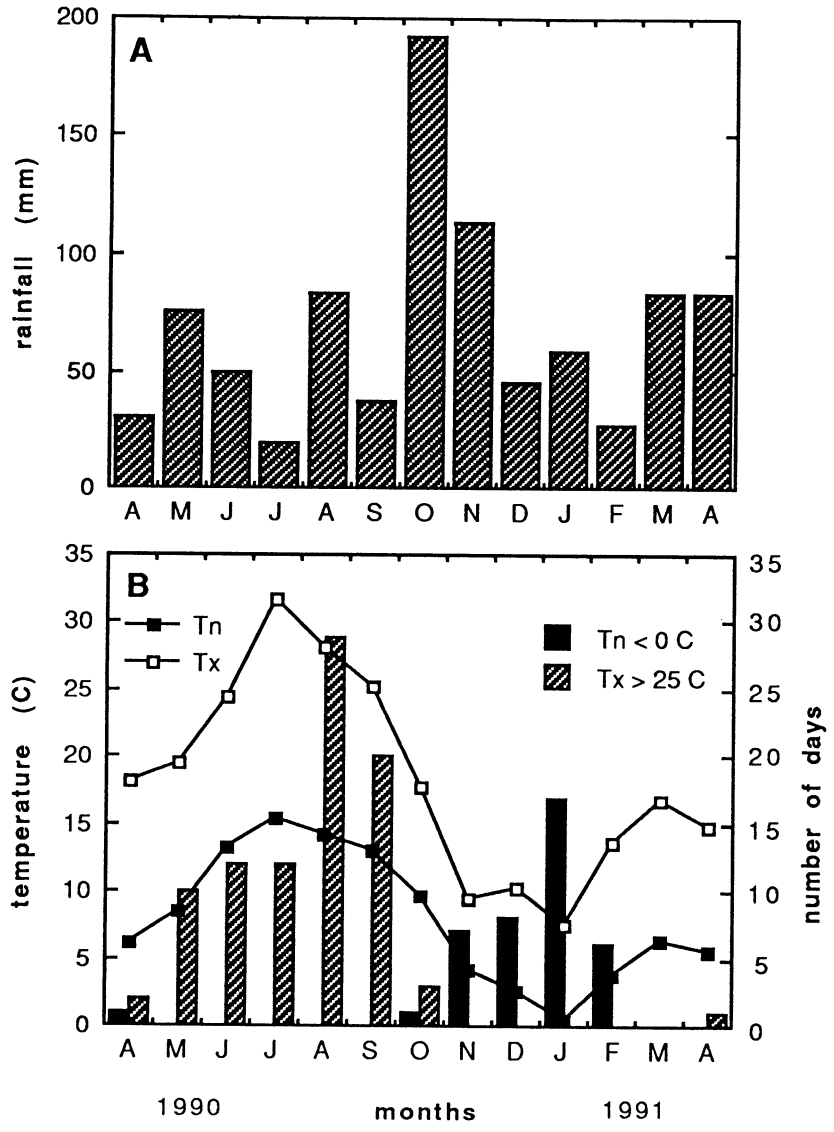

Fig. 1. **(A)** Monthly rainfall and **(B)** average monthly temperature and number of frost days and of days with maximal temperature >25 C recorded at the weather station nearest the red oak stand (Doat, Gers, France) during the experiment.

nificant ($P < 0.01$) in March 1991, April 1990, and August and September 1990 (Table 1).

The tree and height effects were significant on the lesion extension and on the RWC. The collar tissues appeared significantly more resistant to invasion by *P. cinnamomi* and more moist than the trunk tissues (Table 3). These results have been reproduced twice with trees from different origins (*unpublished*).

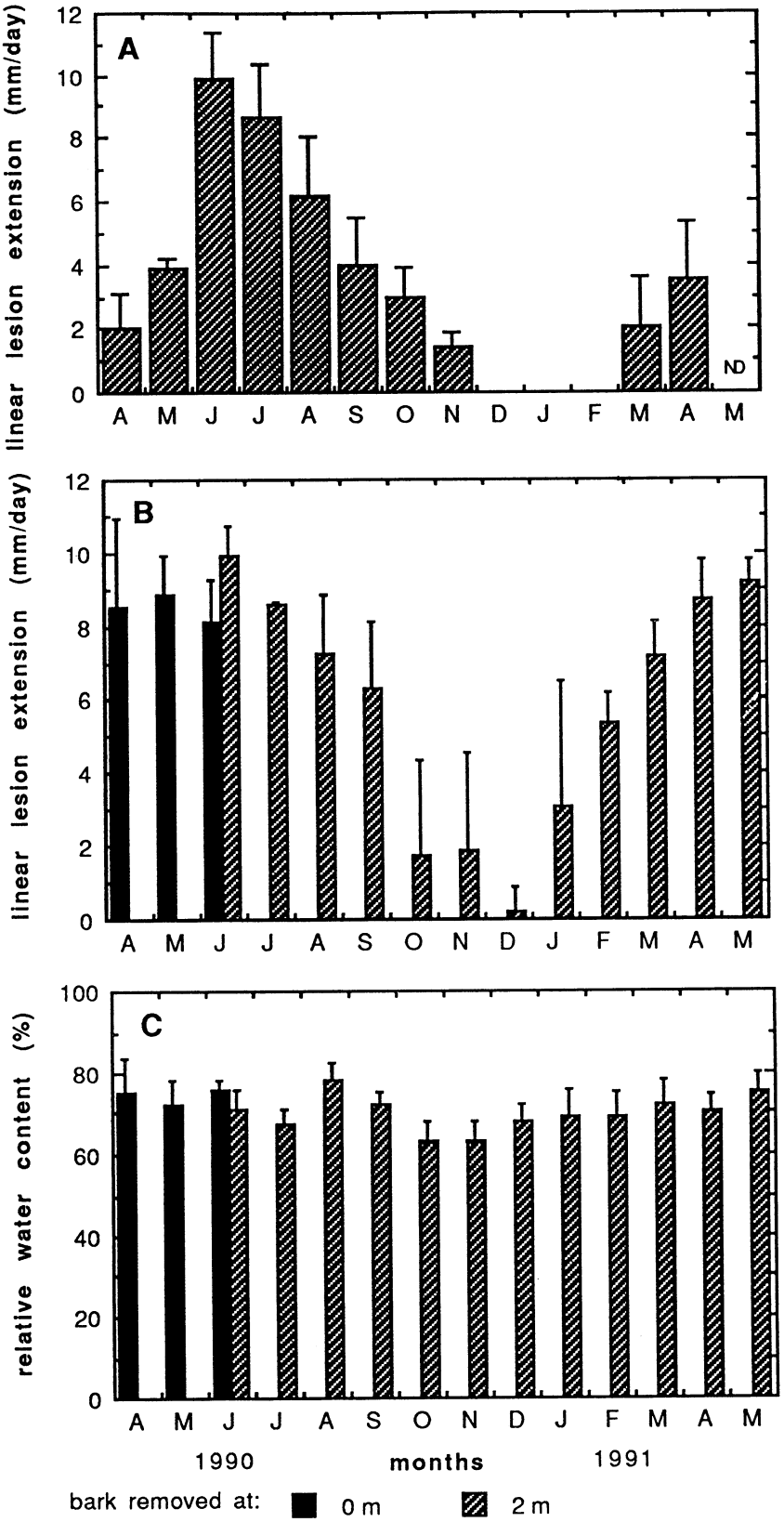

Fig. 2. Temporal changes in linear lesion extension rate caused by *Phytophthora cinnamomi* in **(A)** inoculated red oak trunks, **(B)** excised bark tissues, and **(C)** relative water content (RWC) of excised bark examined monthly. Bark was removed at ground level or 2 m above the ground. Each value is the average of 10 trees (one trunk inoculation, three excised bark inoculations, and one measure of the bark RWC). Vertical bars represent standard errors.

Relationships between seasonal changes in relative water content, climatic factors, and susceptibility of red oaks. The RWC of red oak bark varied significantly during the study ($P < 0.001$) but without any significant relationship to rainfall or maximal and minimal temperatures (Table 4). The maximal and minimal RWC were observed in August 1990 and October 1990, respectively (Fig. 2C).

Average lesion lengths measured in trees were correlated with the lesion length measured in excised bark strips the day of trunk inoculations assessments and also highly correlated with the temperature data (average temperatures, number of frost and hot days) but not with rainfall or RWC of the bark (Table 4). The correlation coefficients between lesion development in excised bark tissues and temperature data were much less, and significant only for maximum temperature and number of frost days. In addition, a positive correlation between lesion development in vitro and RWC of bark was observed.

Relationships between tree-to-tree variability in phenology and susceptibility to *P. cinnamomi*. In 1991, the flush process began between 12 March and 9 April. At the end of this period, the most advanced growth stage observed was green bud, in only two trees within the experimental plot. On 18 April, differences among trees were more obvious. Estimations of tree phenology at this date for the top, middle, and bottom of the canopy are given in Table 5.

No statistically significant correlation was found between susceptibility in situ and phenology. However, in March and May 1991, growth of *P. cinnamomi* in

Table 1. Percent strips developing lesions for each bark test date each month from April 1990 to April 1991 following inoculation with *Phytophthora cinnamomi*

Month	Infected strips showing lesion[x] (%)	$P > F$ tree[y]
April	97	<0.001
May	100	0.037
June, 0 m	100	0.060
June, 2 m	100	0.180
July	100	0.037
August	100	<0.001
September	80	<0.001
October	30	ND[z]
November	30	ND
December	7	ND
January	57	ND
February	85	0.120
March	100	0.004
April	97	0.070

[x] Three strips per tree were inoculated, and lesion development was assessed 2 days after inoculation.
[y] Analysis of variance was performed each month to test the tree effect on the lesion length (29 degrees of freedom).
[z] Not determined.

excised bark was significantly correlated with the more advanced phenological stage assessed in the two lowest parts of the canopy (Table 5).

DISCUSSION

To minimize individual tree variability while investigating the effects of inoculation time, trunk inoculations and excised bark tests were performed on the same trees throughout this study. This necessitated concluding the experiment after 14 mo, when the trees were girdled by monthly inoculations. The data obtained in April and May 1990 are in good accordance with those obtained in 1991 during the same period, and no difference in red oak susceptibility could be detected between the 2 yr, showing that repeated inoculations did not alter tree susceptibility to *P. cinnamomi.* Linear lesion extensions were of the same order after *P. cinnamomi* inoculations in both excised and unexcised cortical tissues. Therefore, detachment of tissues apparently did not cause physiological changes resulting in extensive colonization of the bark by the pathogen, and results from tests on excised bark could be compared to those from direct inoculations. The difference of susceptibility of collar and trunk tissues reported here are in good agreement with previous observations of naturally infected red oaks (20).

Our results indicate seasonal changes in the rate of expansion of lesions caused by *P. cinnamomi* in cortical tissues after inoculations both in situ and in vitro. During winter, from October to February, trunk lesion length decreased to zero. It peaked in spring or early summer. It is most likely that temperature is the limiting climatological factor in forest stands, as suggested by the significant positive correlations observed between the temperature data and lesion development in trunks. Trunk and stem inoculations of *Eucalyptus* spp. and *Banksia grandis* with *P. cinnamomi* have shown that fungal growth rate in trees increases in summer (24,29). In secondary tissues of *E. marginata* and *B. grandis,* temperature–growth relationships of *P. cinnamomi* have been studied; daily growth rate of the fungus increased with temperatures between 10 and 30 C, and decreased below this range (25). Moreover, the lack of cold-hardiness of *P. cinnamomi* at temperatures below zero has been demonstrated (1,10). In December 1990 and January and February 1991, average maximal temperature was less than or equal to 10 C, and more than six frost days occurred each month. *P. cinnamomi* growth and survival under these conditions were not possible, which explains the absence of lesions in trunks.

The effect of temperature on pathogen growth cannot entirely account for the low disease incidence in winter. The bark test technique enabled us to estimate the inherent and immediate receptivity of the phloem because it was performed in standardized conditions and assessed after 3 days. Periods of greatest susceptibility (from April to June) occurred during active shoot extension. The bark receptivity to *P. cinnamomi* decreased with leaf senescence and growth cessation, and increased in February, 2 mo before the breaking of dormancy. In *Quercus robur,* cambium reactivation occurs in 30-yr-old trees 3 wk before the first spring flush, throughout the bole and in branches (9). If such a timing and growth pattern occur in *Q. rubra,* it would mean that the receptivity of the bark tissues to *P. cinnamomi* changes before the reactivation of the cambium. The variation of red oak

Table 2. Linear extension of lesions caused by *Phytophthora cinnamomi* in excised cortical tissues of red oaks from June to September 1991: repeated measures analysis of variance, effects of month of inoculation and tree

Source of vacation	df	Wilks' lambda[z]	MS	*F*	*P*
Month	3	0.095		56.94	0.0001
Month × tree	24	0.034		4.00	0.0001
Tree	9		945.58	5.30	0.0009
Error	20		396.67	$R^2 = 84\%$	

[z] Wilk's lambda is the multivariate test for the repeated measures, whereas mean squares (MS) are from the univariate decomposition of the multivariate tests.

Table 3. *Phytophthora cinnamomi* lesion length and relative water content (RWC) in red oak bark strips removed at three heights above ground level in June 1990

	Height		
	0 m	1 m	2 m
Lesion length[x,z] (mm)	28.3 a	34.2 b	34.7 b
RWC[y,z] (%)	76.5 a	72.2 b	71.1 b

[x] Lesion lengths are measured 3 days after inoculations of bark strips with *P. cinnamomi* culture disks.
[y] RWC = (fresh water content/fully turgid water content) × 100.
[z] Across a row, values followed by the same letter are not significantly different at $P = 0.05$ according to Student-Newman-Keuls tests.

Table 4. Spearman's coefficients of correlation between monthly lesion lengths caused by *Phytophthora cinnamomi* in red oaks, relative water content (RWC) of red oak bark, and climatic data

	Lesion length in unexcised bark[x]	Lesion length in excised bark[y]	Relative water content[y]
Lesion length in trunks	1		
Lesion length in bark excised at 2-m height	0.72**[z]	1	
Relative water content	0.45	0.64*	1
Rainfall	−0.13	0.40	−0.46
Maximal temperature (Tx)	−0.85**	0.66*	0.56
Minimal temperature (Tn)	0.89**	0.59	0.48
No. hot days (Tx > 25C)	0.65*	0.43	0.76**
No. frost days (Tn < 0 C)	−0.63*	−0.61*	−0.34

[x] From April 1990 to April 1991.
[y] From June 1990 to May 1991.
[z] Coefficients of correlation significantly different from 0 at $P = 0.01$ and $P = 0.05$, indicated by * and **, respectively.

Table 5. Phenological assessments of red oaks on 18 April 1991 in Doat and correlations with their susceptibility to *Phytophthora cinnamomi,* evaluated by trunk inoculations and excised bark tests from March to May 1991

Part of canopy	Phenologic assessment[y]		Spearman's correlation coefficients				
			Trunk inoculations		Excised bark tests		
	Minimal	Maximal	March	April	March	April	May
Top	1.4	5	0.17	−0.27	−0.22	−0.02	−0.12
Middle	0.3	4	−0.33	−0.01	0.60*[z]	0.20	0.13
Lower	0	4	−0.13	−0.45	0.68**	0.56	0.60*

[y] Phenologic assessments: 0 = dormants buds, 1 = turgid buds, 3 = green buds, 5 = expanded leaves.
[z] Coefficients of correlation significantly different from 0 at $P = 0.05$ and $P = 0.01$, indicated by * and **, respectively.

susceptibility with growth stage was also demonstrated by the correlation of bark receptivity measured in March and the tree phenologic stage. These results are in accordance with those obtained in young red oaks, when three peaks of susceptibility, corresponding to three growth flushes, were observed (4). It is likely that the second and third flushes in mature trees involve less change in trunk cortical tissues at 1.5 or 2 m above ground level than in juvenile stems.

In our study, monthly average inner bark RWC at collection was positively correlated with the average length of lesions in excised bark tissues. This could indicate that *P. cinnamomi* growth in red oak bark increased with the RWC, as it has been established in *Eucalyptus marginata* (28). However, in the moist chamber, the RWC of the bark strips increased throughout the 3-day incubation. It is likely that the early process of bark colonization by the pathogen is influenced by the hydric status of the host at collection. However, as proposed by Bier (2), the RWC value could be used as a clinical index to indicate the turgor level of the cells and the physiological activity of the living bark, which may also account for the fluctuation of susceptibility to *P. cinnamomi.* This is supported by the lack of consistent monthly correlation between the individual lesion lengths in excised bark and RWC (*results not shown*). Moreover, no correlation has been observed between the average RWC of trees and the average linear lesion extension in trunks.

The seasonal fluctuation in red oak susceptibility to *P. cinnamomi* described in this paper is in agreement with the seasonal changes in lesion development reported for several *Phytophthora* diseases of tree species. Inoculations of excised or unexcised stems and trunks have shown that the susceptibility of apple trees to *P. cactorum* (3,5,23), of walnut trees to *P. citricola* (14), and of citrus to *P. citrophthora* and *P. parasitica* (12) is related to the host growth rhythm, and peaks before and during the active shoot growth. In cinnamon trunks, Rands reported that daily rhythm in lesion extension caused by *P. cinnamomi* was linked to the physiological activity of the trees (17). In citrus, a recent study has shown that seasonal changes in root susceptibility to *P. citrophthora* and *P. parasitica* also increased during active root growth (13). All of these results support the hypothesis of Grainger, who postulated that the carbohydrate content of tissues affects their colonization by pathogens (6). Accumulation of fungistatic products in bark may also determine changes in host susceptibility, and the role of catechins and tannins in preformed resistance to *P. cinnamomi* in *Q. rubra* has been suggested (18). The physiology and chemistry of tree cortical tissues change during the year (8,27). This results in seasonal variation in bark nutritional properties and defense mechanisms. Pathogenic species which develop within the phloem, like several *Phytophthora* spp., are favored by active growth. However, differences in growth requirements exist among these species. For example, in apple tree tissues, the largest lesions caused by *P. megasperma* and *P. cryptogea* were observed not only in spring but also in winter (7). By contrast, cortical cankers caused by facultative parasites in willow and poplar proved to be more aggressive during tree dormancy than during tree growth (2). These weak pathogenic fungi are favored during winter when bark RWC is low and plant defenses less effective. Investigations of the histological and nutritional properties of bark removed at different times from red oaks are required to explain the seasonal fluctuation of susceptibility.

Important considerations in breeding for resistance can be deduced from our results. Screening tests should be performed using the bark strip technique, with optimal testing periods being the very beginning (March or April) and end (August to September) of the growing season. Differences among trees were the most obvious at these two dates. The significant tree × month interaction on lesion development, which resulted in different monthly rankings of trees for their susceptibility, could be accounted for by the differences in the receptivity period among trees. Nevertheless, only part of the variability in red oak susceptibility could be explained by the variability in phenology. Some trees showing a similar growth pattern did exhibit different levels of *P. cinnamomi* tolerance. It is likely that tree susceptibility could be explained by an intrinsic and biochemical component and by a phenologic and physiological component. In early spring, differences of susceptibility among red oaks could be mainly attributable to differences in tree precocity. Later in the season, when trees were in active growth, intrinsic properties would be more important. In apple trees, relationships between vigor and susceptibility to *P. cactorum* have been reported; susceptibility of rootstocks increases with vigor, which also affects the susceptibility of the grafted cultivar (3). In order to dissociate the intrinsic factor from the phenologic one, screening tests should be done at these two periods.

The seasonal variability of red oak susceptibility to the ink disease and tree phenology must also be taken into account to determine dates of possible chemical treatments. In *Leucadendron,* Marks and Smith (11) showed that metalaxyl and phosphonate inhibited *P. cinnamomi* development in the stem if applied 10 days before inoculation. It is likely that the effectiveness of prophylactic treatments of red oaks will depend on the timing of their application in relation to disease susceptibility. Fungicide application should be made in early spring in order to protect the trunks from the first development of the fungus in the active cortical tissues.

ACKNOWLEDGMENTS

We thank Sue Dugdale, Gianna Kalc-Wright, David Guest, and Gretna Weste for reviewing the manuscript, and François De Sabbathier for allowing us to use his property.

LITERATURE CITED

1. Benson, D. M. 1982. Cold inactivation of *Phytophthora cinnamomi.* Phytopathology 72:560-563.
2. Bier, J. E. 1964. The relation of some bark factors to canker susceptibility. Phytopathology 54:250-253.
3. Dakwa, J. T., and Sewell G. W. F. 1981. Influence of rootstocks type and time of inoculation on the resistance of five apple scion cultivars to collar rot caused by *Phytophthora cactorum.* J. Hortic. Sci. 56:357-362.
4. Delatour, C. 1986. Le problème du *Phytophthora cinnamomi* sur le chêne rouge (*Quercus rubra*). Bull. OEPP 16:495-504.
5. Gates, J. E., and Millikan, D. F. 1972. Seasonal fluctuations in susceptibility of the inner bark tissues of apple to colonization by the collar rot fungus *Phytophthora cactorum.* Phytoprotection 53:76-81.
6. Grainger, J. 1962. The host plant as a habitat for fungal and bacterial parasites. Phytopathology 52:140-150.
7. Jeffers, S. N., and Aldwinckle, H. S. 1986. Seasonal variation in extent of colonization of two apple rootstocks by five species of *Phytophthora.* Plant Dis. 70:941-945.
8. Kramer, P. J., and Kozlowski, T. T. 1960. Physiology of Trees. McGraw-Hill, New York.
9. Lachaud, S., and Bonnemain, J. L. 1981. Xylogénèses chez les Dicotylédones arborescentes. I. modalités de la remise en activité du cambium chez les Hêtres et les Chênes âgés. Can. J. For. Res. 59:1222-1230.
10. Marcais, B. 1992. Influence de facteurs de l'environnement sur le développement de l'encre du chêne rouge, maladie causée par *Phytophthora cinnamomi.* Ph.D. thesis. University of Nancy.
11. Marks, G. C., and Smith, I. W. 1992. Metalaxyl and phosphonate as prophylactic and curative agents against stem infection of *Leucadendron* caused by *Phytophthora cinnamomi.* Aust. J. Exp. Agric. 32:255-259.
12. Matheron, M. E., and Matejka, J. C. 1989. Temporal changes in susceptibility of citrus phloem tissue to colonization by *Phytophthora citrophthora* and *P. parasitica.* Plant Dis. 73:408-411.
13. Matheron, M. E., and Matejka, J. C. 1993. Seasonal differences in susceptibility of three citrus rootstocks to root lesions caused by *Phytophthora citrophthora* and *P. parasitica.* Plant Dis. 77:729-732.
14. Matheron, M. E., and Mircetich, S. M. 1985. Seasonal variation in susceptibility of *Juglans hindsii* and Paradox rootstocks of English walnut trees to *Phytophthora citricola.* Phytopathology 75:970-972.
15. Moreau, C., and Moreau, M. 1952. Etude mycologique de la maladie de l'encre du chêne. Rev. Pathol. Veg. 31:201-231.
16. Moser, E. B., Saxton, A. M., and Pezeshki, S. R. 1989. Repeated measures analysis of variance: Application to tree research. Can. J. For. Res. 20:524-535.
17. Rands, R. D. 1922. Stripe canker of cinnamon, caused by *Phytophthora cinnamomi* n. sp. Mededeelingen van het Instituut voor Plantenziekten. Department van Landbouw, Nijverheid en Handel.
18. Robin, C. 1991. La maladie de l'encre du chêne rouge (*Quercus rubra* L.) causée par *Phytophthora cinnamomi* Rands: perspectives pour l'amélioration génétique de la résistance. Ph.D. thesis. University of Bordeaux.
19. Robin, C. 1992. Trunk inoculations of *Phytophthora cinnamomi* in red oaks. Eur. J. For. Pathol. 22:157-165.

20. Robin, C., Desprez-Loustau, M. L., and Delatour, C. 1992. Spatial and temporal enlargement of cankers of *Phytophthora cinnamomi* in red oak. Can J. For. Res. 22:362-366.
21. Robin, C., Desprez-Loustau, M. L., and Delatour, C. 1992. Factors influencing the enlargement of cankers of *Phytophthora cinnamomi* in red oak. Can. J. For. Res. 22:367-374.
22. SAS Institute. 1988. SAS/STATIM Procedure Guide. Release 6.03 ed. SAS Institute, Cary, NC.
23. Sewell, G. W. F., and Wilson, J. F. 1973. *Phytophthora* collar rot of apple: Seasonal effects on infection and disease development. Ann. Appl. Biol. 74:149-158.
24. Shearer, B. L., Michaelsen, B. J., and Somerford, P. J. 1988. Effects of isolate and time of inoculation on invasion of secondary phloem of *Eucalyptus* spp. and *Banksia grandis* by *Phytophthora* spp. Plant Dis. 72:121-126.
25. Shearer, B. L., Shea, S. R., and Deegan, P. M. 1987. Temperature-growth relationships of *Phytophthora cinnamomi* in the secondary phloem of roots of *Banksia grandis* and *Eucalyptus marginata.* Phytopathology 77:661-665.
26. Snedecor, G. W., and Cochran, W. G. 1957. Statistical Methods. 6th ed. Iowa State University, Ames.
27. Srivastava, L. M. 1964. Anatomy, chemistry and physiology of bark. Int. Rev. For. Res. 1:203-227.
28. Tippett, J. T., Crombie, D. S., and Hill, T. C. 1987. Effect of phloem water relations on the growth of *Phytophthora cinnamomi* in *Eucalyptus marginata.* Phytopathology 77:246-250.
29. Tippett, J. T., MacGrath, J. F., and Hill, T. C. 1989. Site and seasonal effects on susceptibility of *Eucalyptus marginata* to *Phytophthora cinnamomi.* Aust. J. Bot. 37:197-210.
30. Whiley, A. W., Pegg, K. G., Saranah, J. B., and Forsberg, L. I. 1986. The control of *Phytophthora* root rot of avocado with fungicides and the effect of this disease on water relations, yield and ring neck. Aust. J. Exp. Agric. 26:249-253.
31. Wicks, T., and Hall, B. 1989. Evaluation of phosphonic (phosphorous) acid for the control of *Phytophthora cambivora* on almond and cherry in South Australia. Aust. Plant Pathol. 19(4):132-133.