M. D. de Jong and P. C. Scheepens
Centre for Agrobiological Research (CABO), P.O. Box 14, 6700 AA Wageningen, Netherlands

J. C. Zadoks
Department of Phytopathology, P.O. Box 8025, 6700 EE Wageningen, Netherlands

Risk Analysis for Biological Control: A Dutch Case Study in Biocontrol of *Prunus serotina* by the Fungus *Chondrostereum purpureum*

Prunus serotina Ehrh. was introduced into the Netherlands from North America around 1920 to improve the understory and litter in planted pine forests. Instead, *P. serotina*, having opportunistic traits (1), developed into a forest pest, spreading fast, colonizing cleared areas, suppressing young pines in plantations (Fig. 1A), and crowding out the native understory species. Mechanical control is expensive and ineffective. Chemical control is possible but undesirable because herbicides affect nontarget species. Therefore, biological control was considered.

Chondrostereum purpureum (Pers.:Fr.) Pouzar is a widespread saprophyte in wood of deciduous trees (11). It is parasitic to *P. serotina* and to cultivated plum (*P. domestica* L.) and cherry (*P. avium* L.) and various ornamentals of the *Prunus* group. *P. serotina* can be controlled by treating its stumps with mycelium of *C. purpureum* (12). Before developing *C. purpureum* as a mycoherbicide (15), it was necessary to assess the hazard posed by an artificially increased population of the fungus to nontarget plants.

The risk-assessment study consisted of field experiments, surveys, and simulations, using models current in epidemiology, micrometeorology, and air pollution theory. This paper gives an overview, foregoing many aspects published elsewhere (2).

Development and Testing of the Simulation Model

Elements of epidemiological and air pollution theories were utilized to develop a conceptual model (Fig. 2) and later a simulation model for biological control of *P. serotina* by *C. purpureum*. Epidemiological theory is based on the concept of the infection cycle. Air pollution theory uses the terms "emission," "transmission," and "immission." A spore cloud is emitted from a source area, transmitted by wind, and immitted into a target area. The source area may be a forest with naturally or artificially infected trees on which the fungus sporulates. The target area, where the spore cloud touches the vegetation, may happen to be a plum orchard and *C. purpureum* could cause unwanted disease (plum trees are nontarget trees from the viewpoint of biocontrol). A combination of elements from the two theories led to a conceptual model and hence to a simulation model, which was then tested in parts.

The potential hazard of biological control for nontarget plants within a forest ("control area") was estimated by testing the susceptibility of wild *Prunus* species through inoculation. The hazard to nontarget plants outside the control area (especially fruit trees) was tested by calculating hourly emission and transmission data in a time-series analysis (9). Cumulative frequency diagrams summarizing daily means of hourly values were used for the time-series analysis. The relative risk to nontarget plants was calculated as the ratio of added immission, following biological control, to natural immission (spores from natural sources), using long-term mean values. Calculations of spore immission were complemented with inoculation experiments.

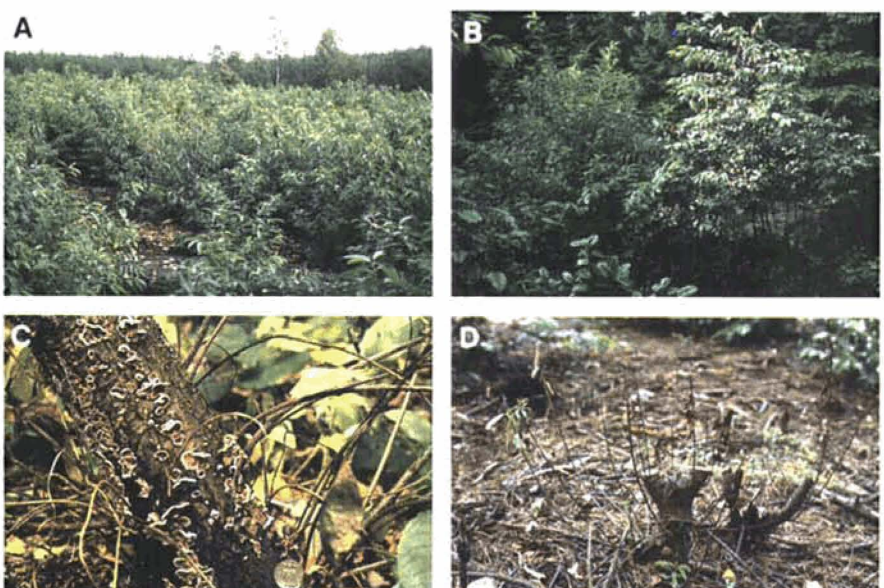

Fig. 1. Biological control of *Prunus serotina* with the fungus *Chondrostereum pupureum*: (A) Heavy infestation of a young pine planting by *P. serotina*. (B) Healthy shrub of *P. serotina* (left) compared with (right) diseased (silverleaf) shrub after inoculation with *C. purpureum*. (C) Basidiocarps of *C. purpureum* on stump of *P. serotina*. (D) Diseased sprouts on stump of *P. serotina* that had been cut and inoculated with *C. purpureum*.

Table 1. Emission of spores of *Chondrostereum purpureum* from a model larch forest at different wind speeds[a]

Wind speed ($m\ s^{-1}$)	Proportion of spores		
	Emitted horizontally	Emitted vertically	Deposited
0.5–2.5	0.19	0.07	0.74
2.5–5.5	0.36	0.17	0.47
>5.5	0.47	0.23	0.30

[a] From de Jong (2). Entries are proportions of spores escaping in different directions, according to the wind speed above the forest. Data were generated by simulation, using a multilayer diffusion model.

The hazard of deploying *C. purpureum* for control of *P. serotina* throughout the Netherlands was estimated by registering the simultaneous occurrence of forests with *P. serotina* and fruit-growing areas in grid cells of 5 × 5 km on a map of the country.

Fructification and sporulation. The fungus forms basidiocarps on diseased stems, branches, and stumps (Fig. 1C), usually beginning in mid-September and continuing until mid-December. The basidiocarps shrivel during dry weather and resume growth after thorough wetting. They are killed by moderate frost (2). Basidiospore production begins a few days after the onset of fructification and continues throughout autumn when temperature and humidity permit (2). Conditions favorable for sporulation were determined in indoor and field experiments by measuring environmental variables and coincident spore production (2). Spore production rate, defined as the number of spores formed per second per square meter of forest soil surface, was determined by multiplying the number of *P. serotina* stumps per square meter by the basidiocarp surface area per stump in square meters and the number of spores produced per square meter of basidiocarp per second. Mean basidiocarp surface area was determined by counting basidiocarps on stumps and using a standard key to assess basidiocarp surface areas (2). Basidiospore production in spores per second per square centimeter of basidiocarp was estimated from field and indoor experiments (2,6,13).

Emission, transmission, and immission. At the control area, the ambient air is charged with basidiospores produced by basidiocarps and discharged by the processes of sedimentation, impaction, and emission. Model calculations assuming a steady state of spore production and spore loss permit the partitioning of spores among sedimentation, impaction, and emission. Emission takes place in two directions, vertical (upward) and horizontal (downwind) (Table 1).

The vertical distribution of spores in the forest air depends on the temperature profile, stability of the air, wind speed, and turbulence, calculated using micrometeorological models (4) and a model of spore dispersal (8). Emission of spores from the control area was calculated as the product of fructification, sporulation rate, and escape fraction. Spore density in the forest air was determined experimentally by means of a volumetric spore trap.

A model forest measuring 250 × 250 m was used for transmission calculations. The model forest was considered as a point source, so that Gaussian plume models (7) could be applied in a time-series analysis of emission and transmission. The Gaussian plume model for long-term mean concentrations (7) was used to calculate theoretical spore concentrations in the immission area. Immission and spore landing were calculated by means of the Gaussian plume model, complemented by frequency analysis (3). At chosen immission points downwind from the model forest, the maximum daily immission (spores per cubic meter) was calculated at chosen probability levels. The immission was then translated into deposition (spores per square meter) on fresh wounds of nontarget trees, using an estimated deposition velocity, and wound size (square centimeters) and wound frequency (wounds per tree) data obtained in a normally pruned plum orchard. The deposition rate was taken to be 0.01 m per second (5).

Infection and disease. Infection and disease may follow deposition of spores on wounds. The probability of disease after inoculation was determined by inoculating wounds (0.5–1.0 cm in diameter) of the susceptible *P. insititia* Jusl. 'St Julien' with droplets containing known numbers of spores.

The experimental site was a 45-year-old stand of larch (*Larix leptolepis* (Sieb. & Zucc.) Gord.), 17 m tall, in the municipality of Ede, heavily infested by *P. serotina*. Weather data were recorded at the site and at a standard weather station in the vicinity of Wageningen, 15 km distant. For inoculation, shrubs 2–3 m tall were cut or sawed at a height of 0.25 m and agar pieces with mycelium from various isolates of *C. purpureum* were applied to the fresh wounds. The incidence of stumps with basidocarps, the number of basidiocarps per stump, and the mean size of the basidiocarps were assessed in the two autumns following inoculation (2).

Field surveys. Surveys for the natural occurrence of silverleaf symptoms on wild *Prunus* were performed in various parts of the Netherlands. Natural occurrence of basidiocarps of *C. purpureum* were surveyed in several locations. Basidiocarps were rather common on stumps and logs of recently felled hardwood trees, but incidence varied according to tree species and vegetation type (2,11).

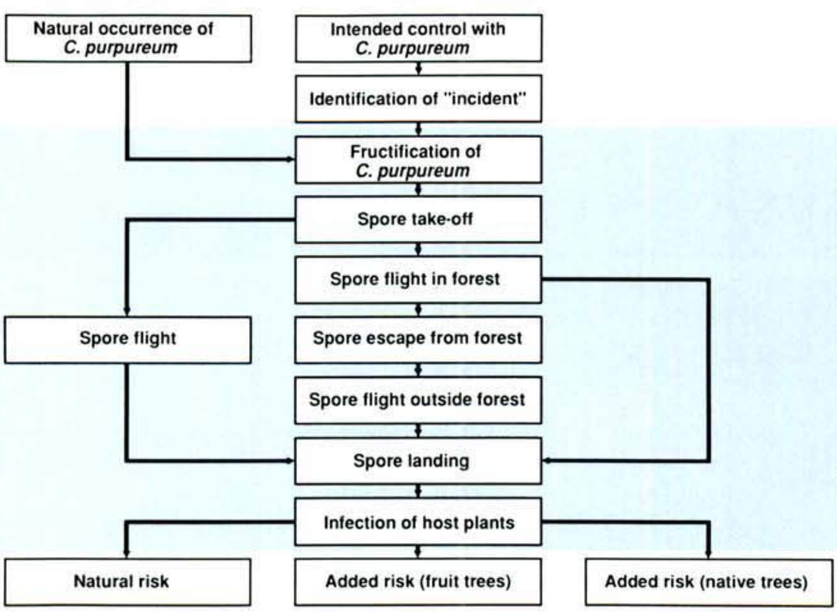

Fig. 2. Infection cycle of *Chondrostereum purpureum* with natural and added infection risks. An "incident" is an undesirable side effect, such as the infection of a nontarget tree, of a desirable action, such as biological control.

Acquisition of Data, Estimation of Risk

Fructification and sporulation. The experimental forest was heavily infested by *P. serotina*, with 0.75 shrubs per square meter. In autumn, following cutting and inoculation of the stumps, an average of about 6 cm^2 of basidiocarp per stump was found. Sporulation (Fig. 3) was related to rainfall, temperature, and relative humidity. It stopped when relative humidity decreased below 90%, when temperature fell below 0 C or rose above approximately 25 C. The spore density (spores per cubic meter of air) near the stumps showed a modest daily rhythmicity (Fig. 4).

Spore flight: Emission. In the experimental forest, the hourly spore density in the air at 0.5 m height was measured throughout autumn in 1982 and 1983 (Fig. 4). The spore trap was placed in an area with a high density of fructifications, thus ensuring a predominance of *C. purpureum* basidiospores. Though basidiospores of many fungi appear similar, those of *C. purpureum* can be differentiated by their size and characteristic shape.

Spore flight: Transmission and immission. The model forest was considered as a point source, to which a Gaussian plume model could be applied. A short-term version of the model (9) was used to calculate hourly values for immission points at 500 and 5,000 m downwind of the experimental forest, using real weather data during a measuring period of 80 days in the autumn of 1983. Daily mean values of emission were calculated, and these were represented as cumulative frequencies (Fig. 5). The values of the 90th percentile were taken as characteristic immission values, 160 and four spores per cubic meter of air for the immission points 500 and 5,000 m downwind, respectively.

Spore landing: Infection and disease. The surface areas of pruning wounds were measured in a bush tree plum orchard; wounds measured about 0.001 m^2 per tree. The mean deposition rate was estimated to be about 0.01 m sec^{-1}. Thus, the number of spores deposited daily on wounds of a freshly pruned tree was estimated to be 144 at 500 m and four at 5,000 m downwind of the model forest, using the 90th percentile of immission (2). Inoculation experiments indicated that droplets containing about 10 spores each could infect wounds up to at least 10 days old at about 20% probability (Table 2).

The risk to nontarget trees is high 500 m distant from a control area but negligible 5,000 m distant. Inevitably, a conclusion differentiating "high" and "negligible" is a subjective one. It is, however, on the safe side because the calculations refer to nontarget trees downwind of the control area. In any other direction the hazard to nontarget trees will be less than indicated.

Survey data. Several areas were surveyed to study naturally occurring infection by *C. purpureum*. At three randomly chosen sample sites of 1,000 m^2, an average basidiocarp area of 0.025 m^2 per site was found, but none was found in 28 similar sites. Some 55 roadside vegetations were sampled, of which one had 0.017 m^2 of basidiocarps. At 10 sites, where lane trees (mainly poplars) had been cut in the preceding year, an average of 0.0135 m^2 of basidiocarps per site was found. It was assumed that in orchards, tree nurseries, and windbreaks, basidiocarps were absent because of hygienic measures.

Apparently, there is a certain level of naturally occurring opportunistic infection, but natural infection of wild *Prunus* species was negligible. No disease was found on indigenous wild *Prunus* species among more than 1,200 plants examined. Incidence on *P. serotina* was 0.6% in more than 3,000 plants, even though most plants had been cut at least once. Inoculation of naturally occurring *Prunus* species by placing mycelial mats on fresh wounds led to some disease but little mortality (Table 3), corroborating the survey experience. The risk to wild *Prunus* species within the control area appears to be negligible. Natural infection of plum trees, a serious problem in the past, is of minor importance at present.

Efficacy of biocontrol. Results of biocontrol (Fig. 1B and D) were highly satisfactory. In one 1986 experiment near Heerlen, 61% of 321 trees inoculated with *C. purpureum* died within 2 years, whereas 56% of 101 glyphosate-treated trees and 1% of 142 control trees died within those 2 years (12).

Hazard maps. Maps of the Netherlands were available for the distribution of pine forests, *P. serotina*, and fruit-growing localities. Geographical data were projected on a map of the country

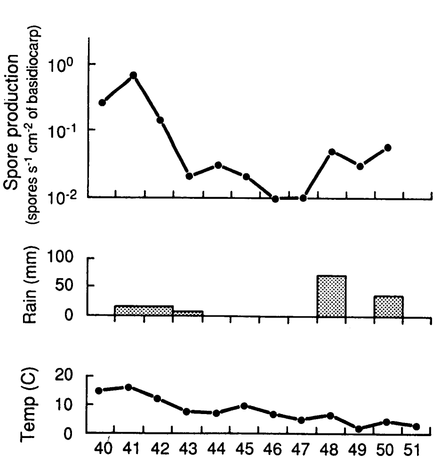

Fig. 3. **Variation of spore production by basidiocarps of *Chondrostereum purpureum* (in spores per square centimeter per second) with temperature (2) in the experimental forest, 1983. Precipitation is expressed as weekly totals and other data as weekly averages.**

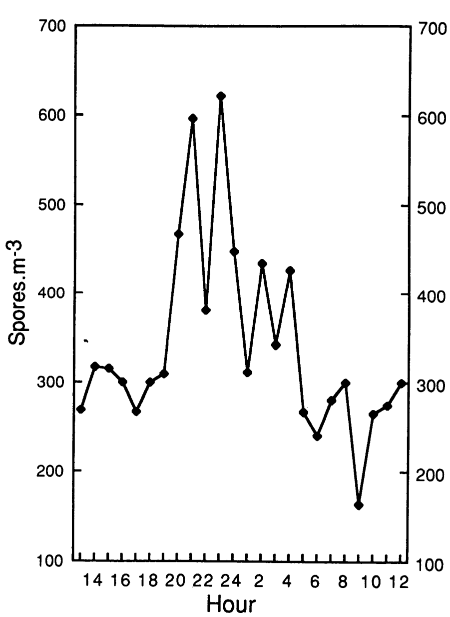

Fig. 4. Hourly spore density in spores per cubic meter of air in a larch forest, at 0.5 m, determined with a Burkard spore trap (2). Entries are averages for 1982 (25 October through 17 December) and 1983 (21 September through 4 December). Variation is considerable, from zero to multiples of graph values. On average, spore density reflects a modest daily rhythmicity.

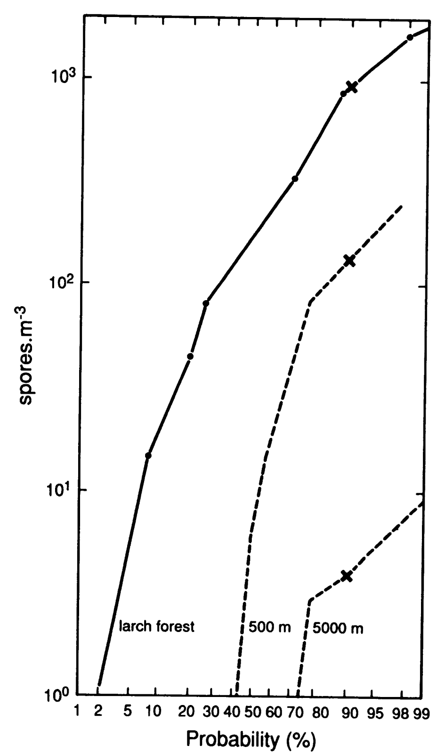

Fig. 5. Cumulative frequency distribution (daily means of hourly values) of densities of basidiospores of *C. purpureum* in the air of the forest and at immission points 500 and 5,000 m downwind of a model forest (2). Probabilities are plotted on a normal probability scale. The solid line represents experimental data, and the broken lines represent calculated values using observed spore densities and weather data. The x in each curve indicates the point where spore immission is equal to or less than the corresponding frequency value in 90% of the days.

Table 2. Incidence of disease (silverleaf symptoms) and mortality in young cultivated plum trees (*Prunus insititia*) in relation to method and severity of inoculation with *Chondrostereum purpureum*[a]

Inoculation method	Number of trees				Proportion diseased + dead
	Inoculated	Healthy[b]	Diseased[b]	Dead[b]	
Mycelium	180	89	53	38	0.50[c]
10 spores/wound	180	142	23	15	0.21
100 spores/wound	180	150	22	8	0.17
1,000 spores/wound	180	146	29	5	0.19
None	60	56	2	2	0.07[d]

[a]From de Jong (2). Inoculations were performed in the field by applying droplets with basidiospores to pruning wounds.
[b]Two years after inoculation.
[c]Difference between mycelium and spores significant, $P<0.05$.
[d]Difference between control and spores significant, $P<0.01$.

Table 3. Incidence of disease (silverleaf symptoms) and mortality in naturally occurring wild *Prunus* species inoculated with *Chondrostereum purpureum*[a]

Species	Number of trees inoculated	Percentage of trees 2 yr after inoculation		
		Healthy	Diseased	Dead
P. avium	6	100	0	0
P. padus[b]	59	76	9	15
P. spinosa	54	67	16	17
P. domestica	26	50	13	37

[a]From de Jong (2). Inoculations were performed in situ by applying mycelium to artificial wounds.
[b]Silverleaf symptoms are difficult to recognize with certainty.

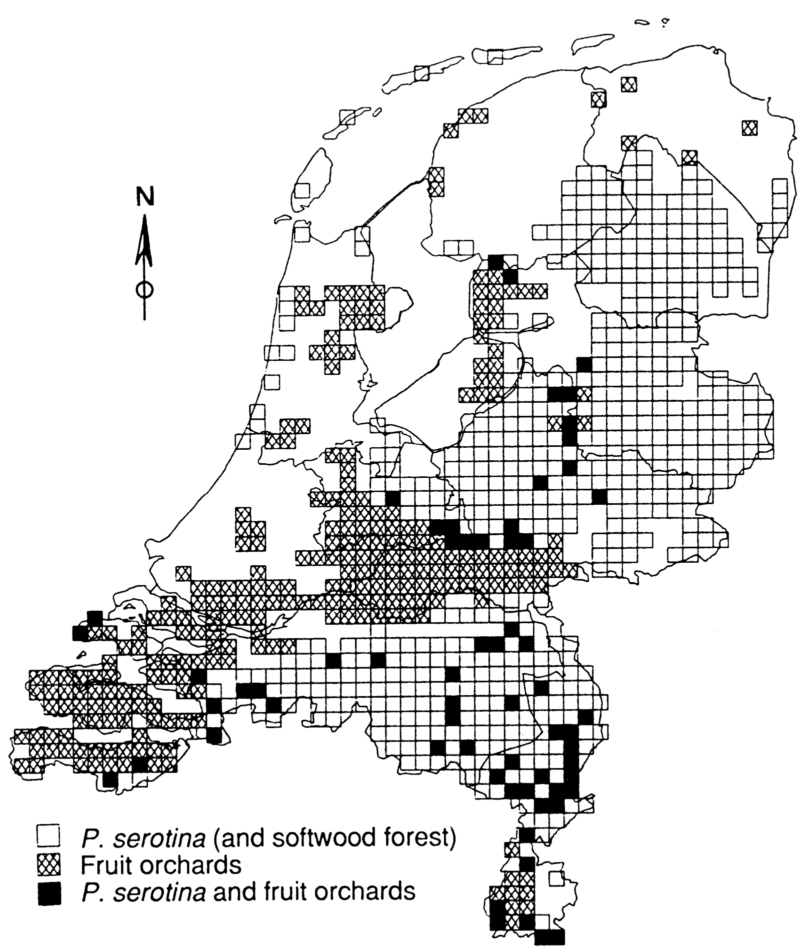

Fig. 6. Map of the Netherlands with the distribution of *Prunus serotina*, fruit orchards, or both, over 5 × 5 km grid cells (2).

overlain by a 5 × 5 km grid (Fig. 6). Grid cells with both *P. serotina* and fruit production are rare, about 4.5%.

The analysis could be continued at a higher level of resolution. The survey yielded mean infection data for various classes of vegetation. Using 1:25,000 Ordnance Survey maps, any grid cell could be subdivided in blocks of 250 × 250 m. The vegetation of each block was characterized as belonging primarily to one of six possible classes. A high-resolution map was then produced in which each block had a specific emission factor related to its vegetation class. Table 4 shows the result for the map named for the village of Oosterbeek. The emission factors of different blocks can be added together for any area as natural sources of infection plus additional sources of infection due to biological control. Such data are shown in Table 5, where selected rural areas are represented by cells in which *P. serotina* and orchards coexist. Table 5 indicates that total emission values for additional and natural sources are of the same order of magnitude. The quotient obtained from the ratio of additional to natural emission (Table 5) is termed "relative risk." The technical advantage in the use of this quotient is that it tends to cancel the scaling errors. These errors must be approximately equal.

In Figure 7A, a high-resolution map represents a 10 × 10 km area near the village of Oosterbeek. Figure 7B translates that map into the logarithm (base 10) of the relative risk values. The part north of the river Rhine is a hill country with forests, where the log values are neutral or slightly positive. The area south of the Rhine consists of arable land and orchards and shows only negative log values. Obviously, the geographic separation of positive and negative areas reduces the risk of infection of nontarget trees after biological control.

Risk Analysis

Terminology for risk analysis proposed by Rowe (10) is followed here. His term "risk assessment" consists of two components, "risk determination" and "risk evaluation." Risk determination consists of "risk identification" and "risk estimation." Risk evaluation consists of "risk aversion" and "risk acceptance." Risk, R, is usually considered to be a function of the probability, P, of an "undesirable event" and its "magnitude," M, $R = f(P, M)$.

Risk identification and estimation. Biological control of *P. serotina* by means of *C. purpureum*, though effective in itself, might have an undesirable side effect: disease in nontarget trees. The risk identification reported here, incomplete as it may be, shows that wild or semiwild trees of the *Prunus* family are not at risk, but some ornamentals and cultivated species might be.

Risk estimation is concerned with the probability of an undesirable event and the magnitude of its consequences. The magnitude of the consequences was not studied; it will usually be limited to the loss of individual fruit or ornamental trees. The economic value of such loss can be calculated but is not relevant here. The present study concentrates on the probability of the "undesirable event," which is expressed as "relative risk" or the proportion of expected additional infection over existing natural infection. This proportion makes sense in a risk evaluation and appeals to the public and the decisionmakers.

Risk aversion and risk acceptance. Risk evaluation implies both risk aversion and risk acceptance. Risk aversion depends on time, methods, and money available to reduce or avoid risk. For biocontrol with *C. purpureum*, the ideal risk aversion is to create a highly pathogenic but asporogenic strain of the fungus. The method seems technically feasible but difficult. The pathogenic monospore cultures tested so far do not show reduced fructification, in line with results from other Basidiomycotina (14).

Risk aversion by growers seems fairly easy. If growers near areas to be treated are warned in time, e.g., by way of the local press, they could refrain from pruning in the autumn following treatment or could treat pruning wounds with a fungicide. Pruning susceptible trees in summer is currently advised to prevent infection from natural sources. The major difficulty would be the expense and inconvenience for growers who are unable or unwilling to prune in summer.

Risk acceptance is a subjective matter that may become a political issue. Risk acceptance is solicited by establishing risk references (here "relative risk") and indicating risk referees. Risk referees are growers, individually and collectively, with trees at risk, or some state authority acting on behalf of them. The state authority will be subject to various pressures from forest managers, chemical industry, tree growers feeling uncomfortable, antipollution activists, and nature conservationists.

The present study, providing a fairly objective relative risk measure, paved the way for the approval of biocontrol of *P. serotina* with *C. purpureum* in the sense of "Yes, if the various political concerns can be satisfied."

Reality and the Model

Risk analyses often treat events that might happen but have not yet happened. They are studies of model behavior and not of reality. The present overall risk model combines several component models that are currently accepted, or at least not refuted, by the scientific community. Empirical confirmation was sought where possible to validate elements of the model. Validation of the

Table 4. Emission values of various vegetation types with or without biological control of *Prunus serotina* by *Chondrostereum purpureum*[a]

Vegetation type	Source of spores	Relative frequency of vegetation type	Emission values[b]
No woody vegetation	None	0.583	0
Pine forest	Additional[c]	0.074	37,000
Mixed forest	Natural	0.171	10,000
Deciduous forest	Natural	0.063	20,000
Small woods	Natural	0.027	10,000
Roadside plantings	Natural	0.050	1,000
Orchards	None	0.029	0
Tree nurseries	None	0.003	0

[a] From de Jong (2). Results of high resolution mapping using a 10 × 10 km section of the 1:25,000 Ordnance Survey map named for the village of Oosterbeek are summarized. Vegetation types were determined for each of 1,600 blocks.
[b] Emission values represent mean numbers of spores emitted per second per grid cell (250 × 250 m), calculated for each vegetation type by means of a long-term emission model.
[c] Spores resulting from biological control with *C. purpureum*.

Table 5. Risk from dissemination of spores produced in biological control of *Prunus serotina* by *Chondrostereum purpureum*[a]

	Cumulated emission values of sources of infection[c]		
Area[b]	Natural × 10^6	Additional × 10^6	Relative risk[d]
Olst	2	0.4	0.2
Oosterbeek	5	4	0.8
Valkenburg	3	0	0
Wageningen	4	1	0.25
Weert	3	7	2.3

[a] From de Jong (2).
[b] Area names refer to Ordnance Survey maps (1:25,000) carrying the same names; areas measure 10 × 10 km.
[c] Spores per second accumulated over all blocks (vegetation types) in the area.
[d] Ratio of additional to natural emission.

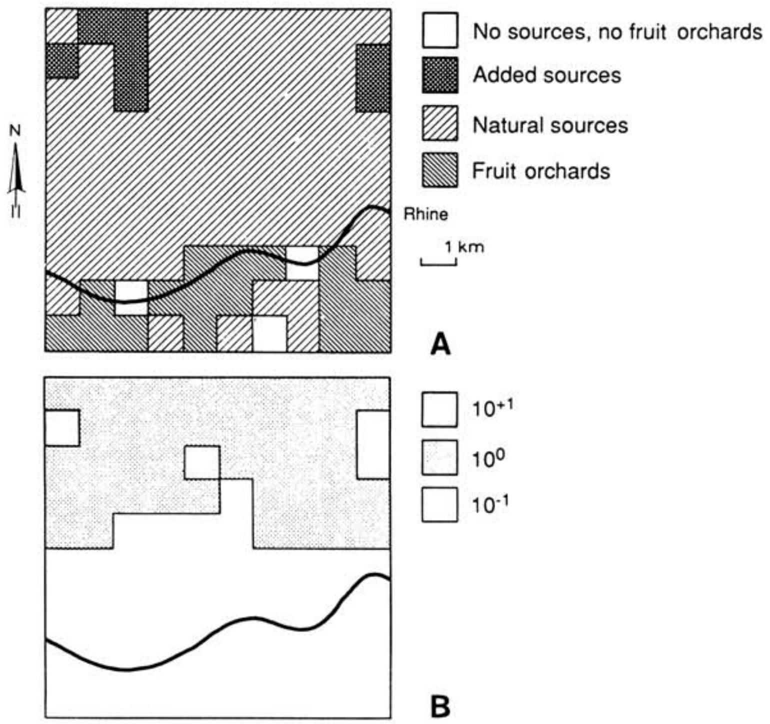

Fig. 7. High-resolution maps of a 10 × 10 km area near the village of Oosterbeek (2); the river Rhine separates a forest area in the north from a fruit-growing area in the south: (A) Position of spore sources and of fruit-growing area. (B) Relative immission (added over natural immission).

overall model was not feasible within the given constraints—and may be impossible. Insofar as figures are quoted, their order of magnitude matters more than their calculated values. Most estimates were conservative, e.g., choosing immission points downwind of the prevailing wind direction. The expression of the final results as the ratio of additional to natural immission, a ratio that supposedly represents the correct order of magnitude, allows the mapping of relative risk with a certain degree of realism.

The relative risk, the "signal to noise ratio" of biocontrol of *P. serotina*, was about 1.0 in most areas. In other words, the additional infection caused by biological control is of the same order of magnitude as the naturally occurring infection. Because the naturally occurring infection is generally considered to be below the nuisance level, the added infection also may be regarded as innocuous in most areas. Biological control of *P. serotina* by *C. purpureum* is usually safe, but in a few and easily identifiable areas some restrictions may be needed. Even in those areas, the spatial separation between pine forest and fruit-growing area usually will ensure safety of biological control. Only in rare and small areas where sour cherry (*P. cerasus* L.) is cultivated near a pine forest should biological control not be applied.

Models and their numeric results can serve only as means to support decision making. Whatever the accuracy of the calculations may be, any policy decision remains a subjective one. The Plant Protection Service of The Netherlands expressed the opinion that the risk due to *C. purpureum* as a mycoherbicide to control *P. serotina* is acceptable except within a safety distance of 500 m between a control area and an area of commercial fruit growing. A commercial company has shown an interest in producing the mycoherbicide.

Acknowledgments

We are indebted to the Ministry of Public Health and Environment and the Ministry of Education for funding; to the director of the Centre for Agrobiological Research for technical support; to J. de Flines, J. A. van Haasteren, P. van Halteren, and J. Goudriaan for advice; and to the feature editor and anonymous referees for constructive criticism.

Meindert D. de Jong **Piet C. Scheepens** **Jan C. Zadoks**

Dr. de Jong is a plant pathologist deeply interested in nonagricultural pathosystems. While a research scientist in the Weed Control Department of the Centre for Agrobiological Research (CABO) in Wageningen, Netherlands, he developed the risk assessment procedures reported here under the guidance of Dr. Scheepens. Risk assessment was the subject of his Ph.D. thesis, supervised by Dr. Zadoks. He obtained his degree from the Wageningen Agricultural University.

Dr. Scheepens is a plant pathologist from the Wageningen Agricultural University, where he obtained his Ph.D. degree. He spent 4 years as a research scientist at the Institute for Plant Pathology and Plant Protection in Göttingen, Federal Republic of Germany, to do his thesis research. At present he is a research scientist at the Centre for Agrobiological Research, where he is charged with the development of biological control of weeds.

Dr. Zadoks is a plant pathologist from the University of Amsterdam, where he obtained his Ph.D. degree. His thesis was based on research on stripe rust of wheat, done at the Research Institute for Plant Protection (IPO) in Wageningen. He became an assistant professor of plant pathology in 1961. At present he is professor of plant pathology and head of the Department of Phytopathology at Wageningen Agricultural University. He has specialized in epidemiology and crop loss, and his present interest is in focus formation and risk assessment.

Literature Cited

1. Auclair, A. N., and Cottam, G. 1971. Dynamics of black cherry (*Prunus serotina* Ehrh.) in southern Wisconsin oak forests. Ecol. Monogr. 41:153-177.
2. de Jong, M. D. 1988. Risico voor fruitbomen en inheemse bomen na bestrijding van amerikaanse vogelkers (*Prunus serotina*) met loodglansschimmel (*Chondrostereum purpureum*). (Risk to fruit trees and native trees due to control of *Prunus serotina* by means of the fungus *Chondrostereum purpureum*.) Ph.D. thesis. Wageningen Agricultural University, Netherlands. 138 pp.
3. Dorrestein, F. B. J. 1981. Frequentiemodellen voor luchtverontreiniging. Verslag V-94. Luchthygiene en -verontreiniging. Wageningen Agricultural University, Netherlands. 220 pp.
4. Goudriaan, J. 1977. Crop micrometeorology: A simulation study. PUDOC, Wageningen, Netherlands. 249 pp.
5. Gregory, P. H. 1973. Microbiology of the Atmosphere. 2nd ed. Leonard Hill, Aylesbury, UK. 377 pp.
6. Grosclaude, C. 1969. Le plomb des arbres fruitiers. VII. Observations sur les carpophores et les spores du *Stereum purpureum* Pers. Ann. Phytopathol. 1:75-85.
7. Kleine Commissie. 1976. Modellen voor de berekening van de verspreiding van luchtverontreiniging inclusief aanbevelingen voor de waarden van parameters in het lange-termijnmodel. Staatsuitgeverij, The Hague. 93 pp.
8. Legg, B. J., and Powell, F. A. 1979. Spore dispersal in a barley crop: A mathematical model. Agric. Meteorol. 20:47-67.
9. Nieuwstadt, F. T. M. 1980. Prediction of air pollution frequency distribution. Atmos. Environ. 14:259-265.
10. Rowe, W. D. 1980. Risk assessment approaches and methods. Pages 3-29 in: Society, Technology and Risk Assessment. G. Conran, ed. Academic Press, New York.
11. Runge, A. 1982. Pilzsukzession auf den Stuempfen verschiedener Holzarten. Pages 631-643 in: Struktur und Dynamik von Waeldern. H. Dierschke, ed. Cramer, Vaduz.
12. Scheepens, P. C., and Hoogerbrugge, A. 1988. Bestrijding van Amerikaanse vogelkers met loodglansschimmel (*Chondrostereum purpureum*). Gewasbescherming 19:141-147.
13. Spiers, A. G. 1985. Factors affecting basidiospore release by *Chondrostereum purpureum* in New Zealand. Eur. J. For. Pathol. 15:111-126.
14. Stahl, U., and Esser, K. 1976. Genetics of fruit body production in higher basidiomycetes. I. Monokaryotic fruiting and its correlation with dikaryotic fruiting in *Polyporus ciliatus*. Mol. Gen. Genet. 148:183-197.
15. TeBeest, D. O., and Templeton, G. E. 1985. Mycoherbicides: Progress in the biological control of weeds. Plant Dis. 69:6-10.