Five basidiomycetes in living stems of *Picea abies*, associated with 7-25 year-old wounds

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A total of 37 *Picea abies* (L.) Karst. stems with decay developing from 7-25 year-old wounds was examined. The average length and lateral area of decay columns was, respectively, 448±167 cm and 97±56 cm^2 in 29 stems infected by *Stereum sanguinolentum* (Alb. & Schw.: Fr.) Fr., 264±56 cm and 93±66 cm^2 in 4 stems infected *Sistotrema brinkmannii* (Bres.) J. Erikss., 115±4 cm and 76±5 cm^2 in 2 stems infected by *Resinicium bicolor* (Alb. & Schw.: Fr.) Parm., 514 cm and176 cm^2 in stem infected by *Postia stiptica* (Pers.: Fr.) Jül., and 363 cm and 248 cm^2 in stem infected by *Coniophora arida* (Fr.) Karst. Results of the study provide additional evidence on *S. sanguinolentum* as most widespread and harmful wound pathogen in *P. abies* trees. Among fungal species investigated *R. bicolor* showed the slowest development in stems and was the least harmful wound invader on *P. abies*.

Key words: *Stereum sanguinolentum, Sistotrema brinkmannii, Resinicium bicolor, Postia stiptica, Coniophora arida*, decay, wounds, *Picea abies*.

Introduction

Following mechanical injury when the bark is removed from the living stems of Norway spruce (*Picea abies* (L.) Karst.), wound surfaces are usually colonized by a number of wood inhabiting fungi. Among them *Stereum sanguinolentum* (Alb. & Schw.: Fr.) Fr. is a very common decay causing species that invades 15-40% of injuries (Ekbom 1928; Pechmann and Aufsess 1971; Muravjova 1971; Isomäki and Kallio 1974; Schönhar 1975, 1979; Huse 1978; Norokorpi 1979; Roll-Hansen and Roll-Hansen 1980a, 1981; Hallaksela 1984a, b; Solheim and Selås 1986; Vasiliauskas *et al.* 1996; Vasiliauskas and Stenlid 1998a). In some stands this fungus was found to infect even 51.9-88.2 % of wounded spruce trees (Domanski 1966; Aufsess 1978; El Atta and Hayes 1987; Igolkina 1990). *S. sanguinolentum* in wood of *P. abies* is commonly found both as pioneer species and, in advanced decay, as combative decayer (Hallaksela 1994), and in wounded *P. abies* the fungus is usually responsible for an extensive heartrot reaching length of 1-1.75 m during 4-8 years after wounding (Pawsey and Stankovicova 1974; El Atta and Hayes 1987). However, the development of *S. sanguinolentum* decay in living *P. abies* stems with older injuries has not been yet documented, except for early study of Ekbom (1928) who reported 3-4 m long decay columns in spruces with 15-year-old wounds.

Among the other basidiomycetes colonizing wound surfaces, *Sistotrema brinkmannii* (Bres.) J. Erikss. in a number of studies was reported to be an occasional invader of injuries on spruce (Shea 1960; Pechmann and Aufsess 1971; Pechmann *et al.* 1973; Bonnemann 1979; Norokorpi 1979; Aufsess 1980; Roll-Hansen and Roll-Hansen 1980a; Gregory 1984; Hallaksela 1984a; Vasiliauskas *et al.* 1996; Vasiliauskas and Stenlid 1998a). According to Rizzo and Harrington (1988), pointed out that *S. brinkmannii* was a common cause of the brown butt rot in fir stems damaged by wind. However, many authors have doubted the ability of the fungus to cause appreciable decay in standing trees (Shea 1960; Esslyn 1962; Pechmann and Aufsess 1971; Bonnemann 1979; Aufsess 1980; Roll-Hansen and Roll-Hansen 1980a; Gregory 1984), although their observations were not based on long term experimental data.

White rotting basidiomycete *Resinicium bicolor* (Alb. & Schw.: Fr.) Parm. was isolated from wound or butt rot columns in standing spruces and firs during many studies (Whitney 1961; Pawsey and Gladman 1965; Pawsey 1971; Pechmann and Aufsess 1971; Siepmann 1971; Pechmann *et al.* 1973; Kallio and Tamminen 1974; Norokorpi 1979; Schönhar 1979; Hinds *et al.* 1983; Hallaksela 1984b; Rizzo and Harrington 1988; Vasiliauskas and Stenlid 1998a). *R. bicolor* does not always enter trees via wounds, probably because it is cord-forming fungus and therefore can grow into roots directly from the ground (Hinds *et al.* 1983; Kirby *et al.* 1990; Holmer and Stenlid 1997). In several studies the fungus was reported as an important cause of root and butt rot (Siepmann 1971;

Pechmann *et al.* 1973; Rizzo and Harrington 1988). However, after invasion to living spruces through open wounds, the rate of extension of *R. bicolor* was low, the average being about 8 cm (3 inches) a year (Pawsey 1971). According to Pechmann and Aufsess (1971), pointed out that decay caused by *R. bicolor* in *P. abies* stems seldom exceeded the length of 2 m. Experimental evidence is available, suggesting that *R. bicolor* may act also as a weak pathogen that in some cases spreads slowly in living woody tissues of spruce, fir and pine (Siepmann 1981a, b; Harrington *et al.* 1989; Holmer and Stenlid 1997).

Previous investigations in Lithuania had revealed that fruiting bodies of decay fungus *Postia stiptica* (Pers.: Fr.) Jül. and its relative *Postia caesia* (Schrad.: Fr.) Karst. frequently appear on injured *P. abies*, comprising 44.4 % among all fruiting bodies of basidiomycetes growing on wounds (Vasiliauskas 1993). Both these fungi were fruiting commonly on wounds of *P. abies* also in former Czechoslovakia and consequently were regarded to be important spruce pathogens in the region (Prihoda 1957; Hašek 1965; Cervinkova 1980; Soukup 1985; Cerny 1989). *P. stiptica* was presumed to be responsible for about 23-70 % of the total wound decay damage in several spruce stands in Great Britain (Pawsey 1971) and it was found to be present in 62 % of wounded *P. abies* in Byelorussia (Kovbasa 1996). This fungus is the cause of brown butt rot in wounded spruces that spreads 10-35 cm per year, and as a consequence total length of decay in old wounds can reach 5-7 m (Pawsey 1971; Soukup 1985; Cerny 1989).

Another two close related fungi that are able to invade living spruces through wounds and cause brown cubical butt rot are *Coniophora arida* (Fr.) Karst. and *Coniophora puteana* (Schum.: Fr.) Karst. (Etheridge 1956; Parker and Johnson 1960; Whitney 1961; Pawsey 1971; Pechmann and Aufsess 1971; Pechmann et al. 1973; Norokorpi 1979; Schönhar 1979; Hinds *et al.* 1983; Rizzo and Harrington 1988; Vasiliauskas 1993; Vasiliauskas and Stenlid 1998a). Fruitbodies of the latter species were commonly found also on stem wounds of hardwoods *Quercus robur* L. and *Fraxinus excelsior* L. (Vasiliauskas 1998a; Vasiliauskas and Stenlid 1998b). Data regarding spread of these fungi within stems is very limited. Pawsey (1971) reported 2.75 m (9 feet) long decay column caused by *C. puteana* in *P. abies* stem with 16-year-old scar. According to Cerny (1989), the extent of decay caused by *C. arida* in wounded *P. abies* stems may reach 2-3 m in length.

The aim of the present study was to investigate the extent of decay columns caused by *S. sanguinolentum*, *S. brinkmannii*, *R. bicolor*, *P. stiptica* and *C. arida* within wounded stems of *P. abies*.

Materials and methods

The studied forest area was located in central Lithuania, 10 km east of Kaunas (Dubrava Forest). During the preceding studies several hundreds of living *P. abies* stems with logging and bark stripping wounds were investigated and a number of them was found to contain decay columns caused by *S. sanguinolentum*, *S. brinkmannii*, *R. bicolor*, *P. stiptica* and *C. arida* (Vasiliauskas 1993; Vasiliauskas and Stenlid 1998a). All such trees were numbered in the investigated stands. Later on, sampling of the numbered stems and isolation of fungi were carried out as described by Vasiliauskas *et al.* (1996). Briefly, each wounded tree was sampled by inserting an increment borer 6-8 cm deep into the stem 1-3 cm away from the wound edge. Bore cores were brought to the laboratory in sterilized glass tubes. Within 5 h of collection all woody pieces were surface sterilized by flaming and placed on Petri dishes containing Hagem agar (HA) medium: 5 g glucose, 0.5 g NH_4NO_3, 0.5 g KH_2PO_4, 0.5 g $MgSO_4 \cdot 7H_2O$, 5 g malt extract, 20 g agar, 1000 ml H_2O, pH 5.5. Fungal colonies were subcultured after 10-15 days of growth and species in pure culture were identified according to descriptions by Nobles (1965) and Stalpers (1976). Diameter at breast height (DBH) of all sampled trees was measured and wound sizes (debarked areas in cm^2) on each stem were estimated as described by Vasiliauskas *et al.* (1996).

A total of 37 *P. abies* stems was analysed during the present study, including twenty nine stems with *S. sanguinolentum*, four stems with *S. brinkmannii*, two stems with *R. bicolor*, one stem with *P. stiptica* and one with *C. arida*. Among them, 12 bore stem wounds made by moose or red deer 1-2 m high (all those were infected only by *S. sanguinolentum*), and 25 bore butt wounds made during logging and extraction 0-0.5 m high above ground. Selected trees were cut, dissected and the following parameters measured: age of a wound, radial increment of a tree at the wound cross section during 10 years before the injury, extent of decay within stem. Age of the wound was estimated from the number of growth rings formed in stems during the years since wounding occurred. Prior-injury radial growth was estimated as width of ten annual growth rings formed during the years before wounding. Vertical spread of decay was estimated by cutting the stems into sections of 1 m length. Last section after which absence of decay was noted, was sliced into 5 cm discs until the end of decayed wood and total length of decay column was then recorded. Lateral spread of decay over stem cross section was measured at the site of maximal wound width by marking total stem cross section area and macroscopically visible decay border directly onto transparent plastic sheets. Marked areas were later cut out of the sheets, weighed and their dimensions calculated according to the mass of 100 cm^2 of the same plastic. The same principle was applied during previous measuring of wound sizes (Vasiliauskas *et al.* 1996).

Results and discussion

P. abies trees included into the analyses were between 50-60 years of age, 8-27 cm in DBH, bearing wounds 28-2114 cm^2 in size that were inflicted 7-25 years ago. Main parameters of investigated stems, wounds and decay columns are presented in Table 1. Data show that all investigated fungi, probably with the exception of *R. bicolor*, during 1-2 decades are able to cause considerable decay losses in living *P. abies* stems. It must be remembered that *P. abies* trees in forest stands are most extensively damaged, both due to logging and bark stripping, when being 30-50 years of age (Vasiliauskas 1993), so another 50-30 years must pass until wounded trees will reach maturity age suitable for final harvesting. Therefore, the extent of decay noted in the studied stems with 7-25 year-old wounds during the present work can not be regarded as a final dimension for trees at the clear cut age. It is very likely that wood losses caused by decay in such stems would increase during the future years.

If presumed that all infections took place during the first year after injury, mean annual spread calculated from the data in Table 1, was about 37 cm/year for *S. sanguinolentum*, 14 cm/year for *S. brinkmannii*, 7 cm/year for *R. bicolor*, 21 cm/year for *P. stiptica*, and 30 cm/year for *C. arida*. In addition, *S. sanguinolentum*, *P. stiptica* and *C. arida* exhibited intense lateral spread over stem cross section, thus decaying approximately 44-48 % of total cross section area (Table 1). When wounds of identical age (7 years) and identical initial size (1520 cm) were examined on *P. abies* stems during the previous study, the average length of *S. sanguinolentum* decay columns was 291.5 cm, therefore mean annual spread of the fungus consisted of 42 cm/year during 7 years (Vasiliauskas and Stenlid 1998c), thus corresponding well with the results of the present work and the early Swedish study (Ekbom 1928). However, *S. sanguinolentum* growth rates noted above are slightly higher than reported by other authors. Following natural infections upward yearly extension of *S. sanguinolentum* varied between 10-40 cm within the period of 1-4 years (Pawsey and Stankovicova 1974; Kallio 1976; Roll-Hansen and Roll-Hansen 1980a; Solheim and Selås 1986). In *P. abies* stems with 4-8 years old extraction wounds length of *S. sanguinolentum* decay column fell into the range of 1-1.75 m (Pawsey and Stankovicova 1974; El Atta and Hayes 1987). Other decay fungi in *P. abies* associated with stem injury, *Amylostereum areolatum* (Fr.) Boid. and *Amylostereum chailletii* (Fr.) Boid., exhibited mean annual spread of about 28 cm/year, and length of *Amylostereum* decay after 7-24 years in most cases fell into the range of 1-4 m (Vasiliauskas 1998b).

Table 1. Average stem, wound and decay parameters (means ± standard deviations) of analyzed *Picea abies* trees

Fungus	No of trees	Stem DBH, cm	Wound age, yrs	Wound size, cm^2	Decay length, cm	Lateral spread of decay, cm^2	Decayed stem area, %
Stereum sanguinolentum (Alb. & Schw.: Fr.) Fr.	29	16±5	12.1±3.4	359±501	448±167	97±56	48±19
Sistotrema brinkmannii (Bres.) J. Erikss.	4	18±7	18.3±7.4	240±117	264±56	93±66	22±10
Resinicium bicolor (Alb. & Schw.: Fr.) Parm.	2	24±3	15.5±3.5	187±16	115±4	76±5	13±5
Postia stiptica (Pers.: Fr.) Jül.	1	20	24	355	514	176	44
Coniophora arida (Fr.) Karst.	1	22	12	579	363	248	45

Previous study on *S. sanguinolentum* in living *P. abies* demonstrated positive correlations between surface area of wounds and vertical extension of decay, stem DBH and decay extension, width of annual growthrings and lateral penetration of decay (Ekbom 1928; El Atta and Hayes 1987). Apart of *S. sanguinolentum*, some evidence was already provided also for other decay fungi indicating that their development in living *P. abies* stems may also be favoured by good radial increment and tree DBH, as in cases of *Heterobasidion annosum* (Fr.) Bref. (Curtois 1970; Isomäki and Kallio 1974; Dimitri and Schumann 1989), or *Amylostereum* spp. (Vasiliauskas 1998b). Also wound size was shown to influence positively development of different species of basidiomycetes in *P. abies* stems (Isomäki and Kallio 1974; Roll-Hansen and Roll-Hansen 1980b; Vasiliauskas 1993; Vasiliauskas 1998b). However, our preceeding work failed to reveal any relationships between tree or wound parameters and extent of *S. sanguinolentum* decay, except for weak positive correlation between stem DBH and extent of decay at the stem cross section ($r=0.316$; $p<0.05$) (Vasiliauskas and Stenlid 1998c). Results of the present study confirmed positive relationship between stem DBH and lateral spread of *S. sanguinolentum* over stem cross section ($r=0.663$; $p<0.001$). Contrary to the expectations, only weak and statistically not significant correlations were noted between the age of the injury and the length of decay ($r=0.345$; $p>0.05$) (Fig.1), between the size of the wound and the length of decay ($r=0.312$; $p>0.05$) (Fig. 2), and between the width of 10 prior-injury growth rings and lateral extent of decay ($r=0.333$; $p>0.05$). Also in previous work no correlation was found between the age of wound and spread of *Amylostereum* decay in injured *P.abies* (Vasiliauskas 1998b). The most probable explanation could be that the spread of decay fungi infecting wounds may be more affected by wound parameters during the first post-injury years, while majority of wounds examined in both studies were more than 10-years-old. Several authors had reported decreasing advance of decay in conifer stems with older wounds (Ekbom 1928; Parker and Johnson 1960; Isomäki and Kallio 1974; Vasiliauskas 1993). According to El Atta and Hayes (1987), mean vertical extension of *S. sanguinolentum* decay from 4-year-old and 8-year-old extraction wounds was 136.6 cm and 150.5 cm, respectively, and the difference was statistically not significant. Despite rather broad spectrum of wound ages analysed, the present study on its own too, failed to reveal significant relationship between the wound age and extent of *S. sanguinolentum* decay within stems of *P. abies* (Fig. 1). However, a comparison of data sets from our two independent investigations provides stronger evidence that further spread of the fungus within stems takes place in the course of time. In preceeding work, average length of *S. sanguinolentum* decay associated with 7-year-old injuries was 291.5±77.2 cm (Vasiliauskas and Stenlid 1998c), when during the present study it was 447.7±167.1 cm in approximately 12-year-old wounds (Table 1). Difference between these two mean values, when compared by the Student's t-test for comparison of means, proved to be highly statistically significant ($p<0.0001$).

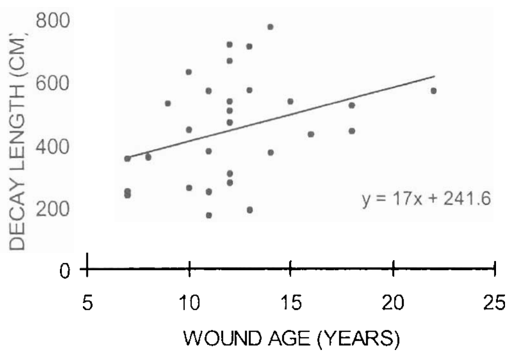

Fig. 1. Relationship between wound age and length of Stereum sanguinolentum decay in Picea abies stems ($r=0.345$; $p>0.05$).

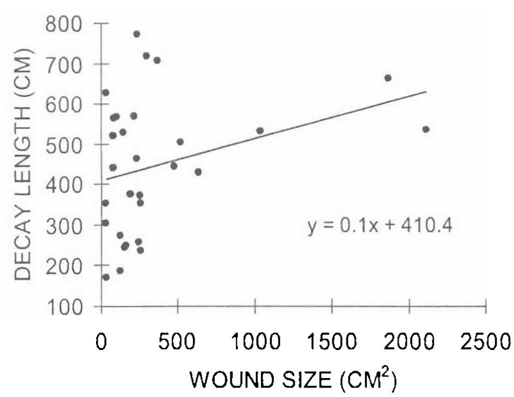

Fig. 2. Relationship between wound size and length of Stereum sanguinolentum decay in Picea abies stems ($r=0.312$; $p>0.05$).

Patterns of wound decay caused in *P. abies* stems by *S. brinkmannii*, *R. bicolor*, and *C. arida* are shown in Figures 3, 4 and 5, respectively. Decay columns of several butt rotting fungi in *P. abies* were found to extend to a height about 20-25 times exceeding their diameter at the stump, as it was shown for *H. annosum* (Zycha *et al.* 1970; Kallio and Tamminen 1974; Swedjemark and Stenlid 1993; Vasiliauskas and Stenlid 1998d), *S. sanguinolentum* (Vasiliauskas and Stenlid 1998c), *A. areolatum* and *A. chailletii* (Vasiliauskas 1998b). Taken that decay caused by *S. brinkmanni*, *R. bicolor*, *P. stiptica* and *C. arida* at the stump cross sections approximate circles, the average diameter of the decay column at the stump level, calculated according to mean decay area (92.5, 75.5, 176 and 248 cm^2, respectively; Table 1), approximates to 10.9 cm for *S. brinkmannii*, 9.8 cm for *R. bicolor*, 15.0 cm for *P. stiptica* and 17.8 cm for *C. arida*. Therefore, length v.s. diameter ratio of the

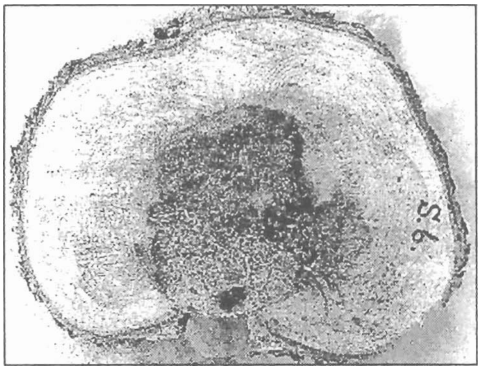

Fig. 3. Decay caused by *Sistotrema brinkmannii* at the stem cross section of *Picea abies* wounded 18 years ago.

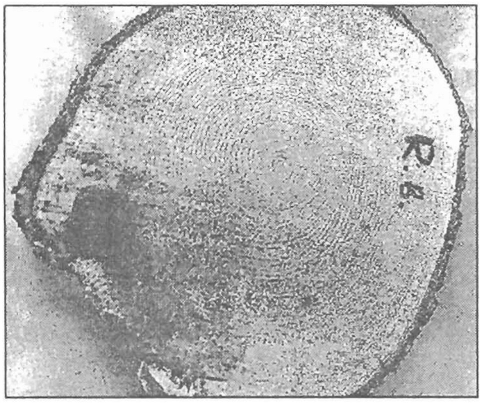

Fig. 4. Decay caused by *Resinicium bicolor* at the stem cross section of *Picea abies* wounded 13 years ago.

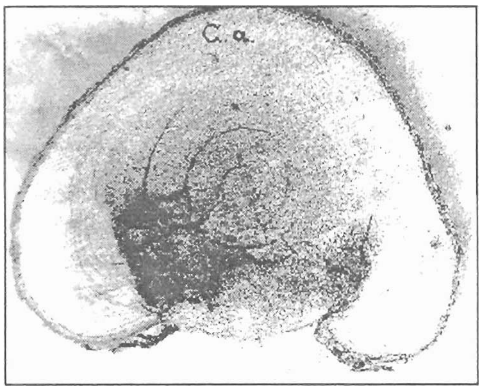

Fig. 5. Decay caused by *Coniophora arida* at the stem cross section of *Picea abies* wounded 12 years ago.

decay columns was 24 for *S. brinkmannii*, 12 for *R. bicolor*, 34 for *P. stiptica* and 20 for *C. arida*. Therefore for *S. brinkmanni* and *C. arida* length v.s. diameter ratio of decay columns was within the range that was already reported for other decay fungi in *P. abies* stems, when decay columns of *R. bicolor* appeared to be more "compressed", and that of *P. stiptica* was more "elongated". In the present work similar calculations were not performed for *S. sanguinolentum*, since infections of the fungus to almost half of analysed trees (12 out of 29) took place not via butt wounds but via 1-2 m high on a stem located bark stripping injuries, what leads to different formation of decay column due to additional fungal spread downwards towards the butt (Ekbom 1928; Vasiliauskas 1993).

Results of this study provide additional evidence on *S. sanguinolentum* as most widespread and harmful wound pathogen in *P. abies* trees. They also indicate that *P. stiptica* and *C. arida* may cause extensive decay losses in damaged *P. abies* stands, in cases when suitable environmental conditions for their more abundant infections are provided. Spread of *S. brinkmannii* in wounded spruces is slower, and, according to the literature, infections of this fungus are much more uncommon than above mentioned species. Among five fungal species investigated *R. bicolor* showed the slowest development in stems and probably is the least harmful wound invader on *P. abies*. However, to obtain more reliable data regarding the pathogenicity in spruce of *S. brinkmannii*, *R. bicolor*, *P. stiptica* and *C. puteana*, it would be desirable to analyse a bigger number of infected stems than it was performed in this work.

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РАЗВИТИЕ ПЯТИ ДЕРЕВОРАЗРУШАЮЩИХ ГРИБОВ В СТВОЛАХ ЕЛИ СО СТАРЫМИ ПОРАНЕНИЯМИ

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Резюме

Проанализировано 37 стволов ели (*Picea abies* (L.) Karst.), пораненных 7-25 лет назад. Средняя протяженность гнили в стволе, и ее площадь сечения в месте раны были, соответственно, 448±167 см и 97±56 см^2 в 29 стволах пораженных *Stereum sanguinolentum* (Alb. & Schw.: Fr.) Fr., 264±56 см и 93±66 см^2 в 4 стволах пораженных *Sistotrema brinkmannii* (Bres.) J. Erikss., 115±4 см и 76±5 см^2 в 2 стволах пораженных *Resinicium bicolor* (Alb. & Schw.: Fr.) Parm., 514 см и 176 см^2 в стволе пораженном *Postia stiptica* (Pers.: Fr.) J., 363 см и 248 см^2 в стволе пораженном *Coniophora arida* (Fr.) Karst. Результаты исследования показывают, что *S. sanguinolentum* является самым опасным раневым патогеном ели. *R. bicolor* в раненных деревьях распространялся наиболее медленно.

Ключевые слова: *Stereum sanguinolentum*, *Sistotrema brinkmannii*, *Resinicium bicolor*, *Postia stiptica*, *Coniophora arida*, раневая гниль, *Picea abies*.