Spread of *Stereum sanguinolentum* Vegetative Compatibility Groups within a Stand and within Stems of *Picea abies*

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A total of 57 naturally established *Stereum sanguinolentum* isolates was obtained from artificially wounded *Picea abies* stems in a forest area of 2 ha in Lithuania. Somatic incompatibility tests revealed 27 vegetative compatibility groups (VCGs) that contained 1–10 isolates. There was no spatial clustering of *S. sanguinolentum* VCGs within the forest area. The extent of *S. sanguinolentum* decay was analysed in 48 *P. abies* stems, 9–26 cm in diameter at breast height. Within 7 years of wounding, the length of *S. sanguinolentum* decay column in stems was 107–415 cm (291.5±77.3 cm on average), lateral spread of the fungus at the butt was 38–307 cm^2 (142.3±66.8 cm^2) and decayed proportion of the stem cross-section at the wound site (the butt) was 3–84 % (36.8±19.7 %). In average, *S. sanguinolentum* VCG that infected 10 trees exhibited more slow growth inside the stem than VCGs that infected only one tree, and vertical growth varied to a greater extent within this VCG than among different VCGs. Correlation between stem diameter and vertical spread of *S. sanguinolentum* was not significant (r = –0.103). Despite uniformity of debarked area on all stems 7 years ago (300 cm^2), open wound sizes on individual trees at the time of study were between 97–355 cm^2 (215.1±59.2 cm^2) indicating large differences in wound healing capacity.

1 Introduction

Stereum sanguinolentum (Alb. & Schw.: Fr.) Fr. is a common basidiomycete throughout the boreal and temperate forests that causes white rot in newly dead conifer logs and stumps (Björkman 1958, Jahn 1979, Eriksson et al. 1984, Breitenbach and Kränzlin 1986, Niemelä et al. 1995). It is also an important pathogen of Norway spruce (*Picea abies* (L.) Karst.), able to cause decay in living trees after infecting stems through open wounds (Isomäki and Kallio 1974, Pawsey and Stankovicova 1974a, El Atta and Hayes 1987). In nature, *S. sanguinolentum* is spread by airborne basidiospores that often are produced parthenogenetically (Robak 1942). This may lead to formation of non-outcrossing sub-populations of the fungus that are genetically isolated from one another (Rayner and Turton 1982, Ainsworth 1987). In Northwestern Europe over 30 distinct clonal or near-clonal subpopulations of *S. sanguinolentum* have so far been detected (Rayner and Boddy 1988). Some of these seemed to be highly local whereas others were more widespread.

A self- non-self rejection mechanism known as somatic or vegetative incompatibility operates to delimit individual genotypes from one another in many fungal species (Rayner et al. 1984). On this basis isolates can be assigned to vegetative compatibility groups (VCGs) that are likely to represent groups of closely related mycelia or even single clones or genets (Anderson and Kohn 1995). In non-outcrossing populations of *S. sanguinolentum*, interactions of all sibling and some non-sibling primary mycelia result in intermingling whereas other non-sibling interactions result in rejection response (Ainsworth 1987). During preceding work several VCGs of *S. sanguinolentum* were detected in Baltic sea region, distributed mainly in local forest stands, although the distribution of some VCGs was extended on a large geographical scale and compatible isolates of the fungus were found at several sites in Sweden, Finland and Lithuania (Vasiliauskas and Stenlid 1998a).

Detection of VCGs provide information concerning scale and spatial distribution of genetic variation in natural populations. Furthermore, genetically distinct individuals of the fungus (rep-resentatives from different VCGs) may exhibit different ability of spread inside the tree. From the forester's point of view, size of each *S. sanguinolentum* genet within a living stem is closely associated with potential decay losses in a forest stand. The aim of the present investigation was to study the spatial distribution of *S. sanguinolentum* VCGs in a forest stand and their spread inside living stems of *P. abies* following natural infections.

2 Material and Methods

The study site was located in central Lithuania, 10 km east of Kaunas in Dubrava forest area (54°55' N, 24°02' E) and contained a *P. abies* stand consisting of trees approx. 50 years old. The experimental wounding, sampling and isolation of fungal cultures was aimed to study fungal colonization of open wounds on spruce, and has been already described in detail by Vasiliauskas and Stenlid (1998b). Briefly, a set of 180 sound looking *P. abies* stems was selected, all trees then numbered and mapped. On each of them an artificial injury 300 cm^2 in size (15×20 cm) was inflicted in January 1990 at the butt (not exceeding 0.4 m height from the ground) by tearing the bark off with an axe. Six years later, in January 1996, 156 of the remaining numbered stems were sampled (in the meantime 24 of the initially selected trees had died). Each 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 samples were surface sterilized by flaming and placed on Petri dishes containing Hagem agar (HA) medium (Stenlid 1985). Fungal colonies were subcultured after 10–15 days of growth. Samples obtained from 45 stems (28.8 %) gave growth to *S. sanguinolentum* (Vasiliauskas and Stenlid 1998b). In February 1997 (7 years since wounding and 1 year since sampling), wound sizes of the trees were measured and they were cut. This revealed the presence of *S. sanguinolentum* decay in twelve more stems. The decayed wood was then sampled and yielded cultures of the fungus. Therefore a total of 57 isolates of *S. sanguinolentum* were obtained in the
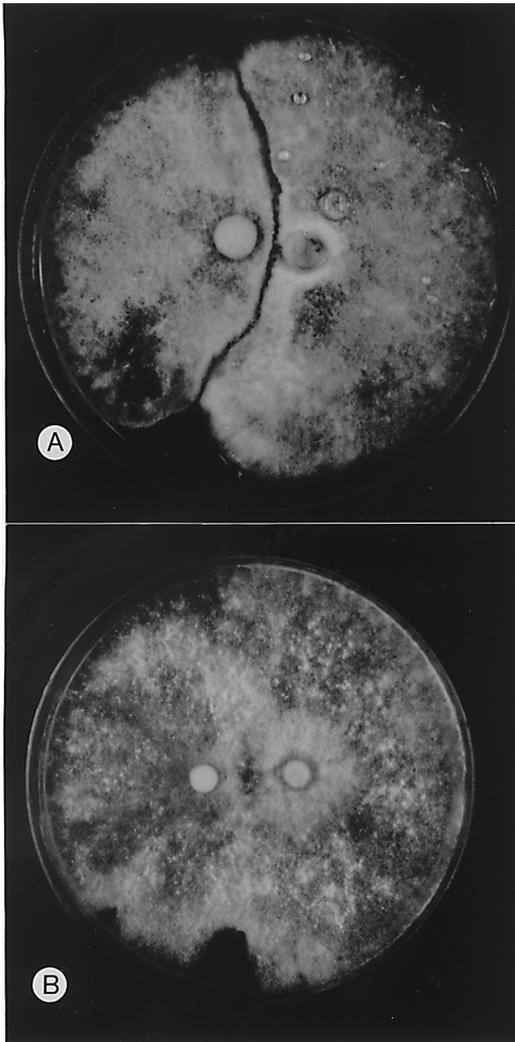
Fig. 1. Somatic incompatible (A) and somatic compatible (B) mycelial reactions in pairings between different isolates of *Stereum sanguinolentum*.

study site. Distances between every isolation of the fungus within the plot were estimated from the map.

Among 57 stems infected by *S. sanguinolentum* nine contained rot caused by another fungus, *Heterobasidion annosum* (Fr.) Bref. Therefore these 9 stems were excluded from further analyses regarding fungal spread within the trees. As a result the extent of *S. sanguinolentum* de-

cay was analysed in 48 *P. abies* stems that bore butt wounds of identical age (7 years) and identical initial size (300 cm²). Average diameter of the trees at the breast height (DBH) was 18.1±4.4 cm (varied between 9–26 cm). 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 crossection at the stump was measured marking total crossection 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 cm² of the same plastic. The same principle was applied for measuring wound sizes (Vasiliauskas et al. 1996). Prior-injury radial growth was estimated as width of 10 annual growth rings formed during the 10 years before wounding.

All 57 *S. sanguinolentum* isolates were included in somatic incompatibility tests that were performed as described earlier (Vasiliauskas and Stenlid 1998a). Briefly, the isolates were paired in all possible combinations (1596) and in self-pairing controls (57). In all tests 4 mm mycelium + HA inoculum discs were cut from the margin of actively growing colonies and placed 1.5–2.0 cm apart in the centre of 9 cm Petri dishes containing approx. 20 ml HA. These were incubated up to 60 days at room temperature and examined periodically. No more than two inoculum discs were confronted in one plate. Interactions between two mycelia were regarded as compatible when a continuous mycelial mat was formed between isolates, corresponding well to that of self-pairing controls, and all other types of mycelial interaction following contact were classed as incompatible (Fig. 1).

Correlations and regression equations between tree, wound and decay parameters were calculated, and the extent of decay caused by isolates from different VCGs was compared using t-statistic for comparison of means (Mead and Curnow 1983).

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3 Results

Somatic incompatibility tests with 57 of *S. sanguinolentum* isolates revealed 27 VCGs that contained 1–10 isolates. Distribution of VCGs in relation to the number of infected stems is shown in Fig. 2 and their spatial distribution in the forest stand is presented in Fig. 3. There was no spatial clustering of *S. sanguinolentum* VCGs within the forest area studied (Figs. 3 and 4).

S. sanguinolentum VCGs that tended to infect more trees exhibited slightly slower growth rates inside the stems (Fig. 5). This is shown also by comparison of average spread of decay columns caused by the isolates from the largest VCG (10 stems infected) and the single infection isolates

(Table 1). Vertical growth of fungus in stems varied to a greater extent within this VCG than among different VCGs, as this is indicated by almost twice higher values of standard deviation (Table 1).

Length of *S. sanguinolentum* decay column in stems varied between 107–415 cm (291.5±77.3 cm on average). Lateral spread of the fungus at the stump (wound cross-section) varied between 38–307 cm^2 (142.3±66.8 cm^2 on average) and as a result decayed proportion of the stem cross-section comprised 3–84 % (36.8±19.7 % on average). The length of decay column correlated positively with lateral spread of the fungus at the stump (r = 0.474, *P* < 0.001) and with the decayed proportion of the stump cross-section (r =
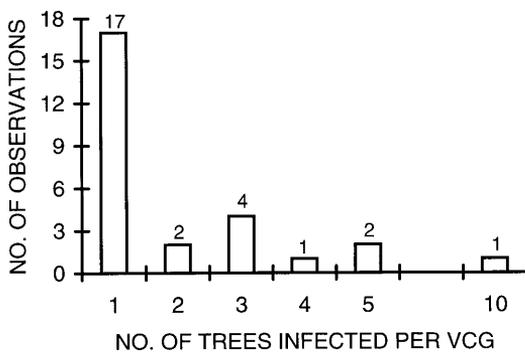

Fig. 2. Distribution of *Stereum sanguinolentum* vegetative compatibility groups (VCGs) according to the number of infected trees.
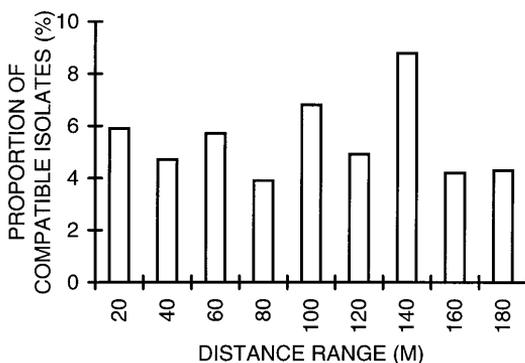

Fig. 4. Occurrence of somatic compatible *Stereum sanguinolentum* isolates in relation to the distance between infected trees within the forest stand (r = –0.007). Data based on 1596 distance measurements between the pairs of isolates.
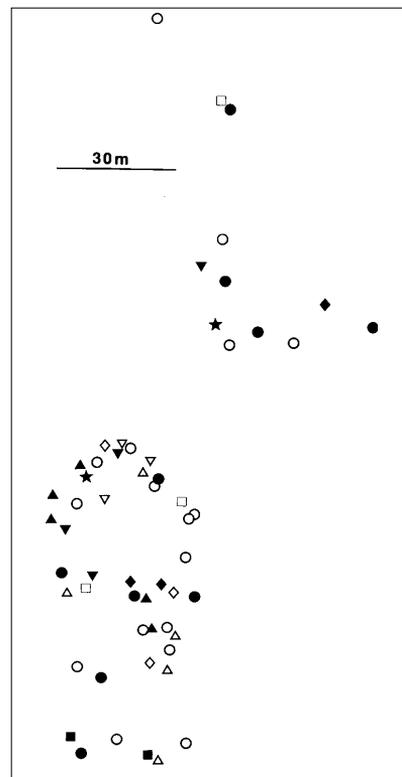

Fig. 3. Spatial distribution of *Stereum sanguinolentum* vegetative compatibility groups (VCGs) within studied forest stand. Open circles show trees from which single representatives of different VCGs were isolated. Each other symbol indicates affiliation to a separate VCG consisting at least of two compatible isolates.

Table 1. Average spread of *Stereum sanguinolentum* vegetative compatibility groups (VCGs) in stems of *Picea abies*.

Parameters of decay column	Mean ± standard deviation		Difference significant at
	within VCG that infected 10 trees	among VCGs that infected one tree	
Length (cm)	250.4±80.9	325.1±47.1	$P = 0.012$
Lateral spread (cm^2)	126.9±74.4	163.9±74.8	$P = 0.277$*
Decayed stem cross-section area (%)	22.5±16.6	38.9±17.0	$P = 0.040$
Number of analyzed stems	8	14	

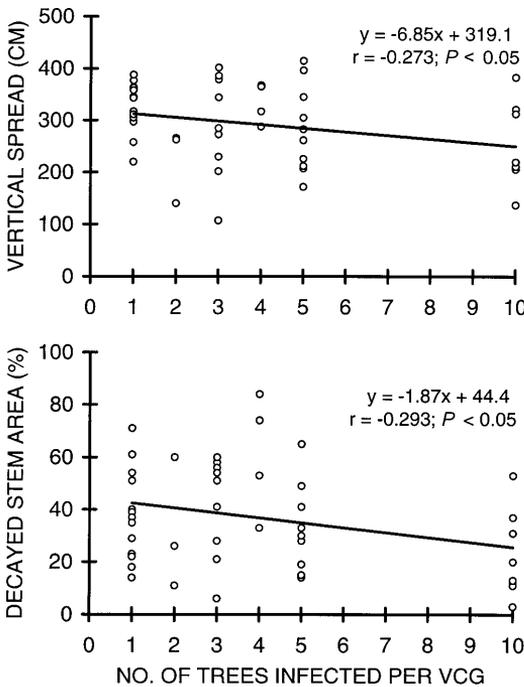* = difference statistically not significant.

Fig. 5. Extent of decay in *Picea abies* stems caused by *Stereum sanguinolentum* vegetative compatibility groups (VCGs) in relation to the number of trees infected per one VCG.

Fig. 6. Relationship between prior-injury radial growth of tree and wound surface area following 7 years after artificial injuries 300 cm^2 in size (15×20 cm) were inflicted on *Picea abies* stems. Correlation statistically not significant (r = –0.206).

0.435, $P < 0.01$). Sizes of the open wounds at the time of study varied between 97–355 cm^2 (215.1±59.2 cm^2 on average) and had no significant influence neither on longitudinal (r = 0.253, $P > 0.05$) nor on lateral extension (r = 0.076) of decay columns. Tree vigour prior to injury (expressed as the width of 10 prior 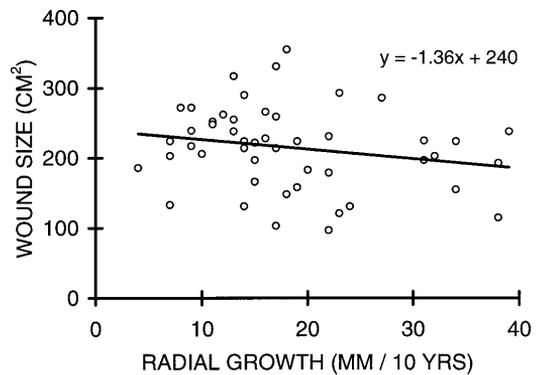injury growth-rings) had no significant influence neither on wound size (Fig. 6) nor on decay extension within a stem (r = –0.238 and r = 0.174, for longitudinal and lateral decay extension respectively). Decay cross-section area at the stump was larger in stems of bigger DBH (r = 0.316, $P < 0.05$), but the proportion of stem cross-section in thicker trees was comparatively less affected by the fungus (r = –0.719, $P < 0.001$). Correlation between stem diameter and vertical spread of *S. sanguinolentum* was not significant (r = –0.103).

4 Discussion

A previous study had shown that the wood of living spruce may be attacked by *S. sanguinolentum* already within the first year after the bark

is removed from the stem and that the number of injuries invaded by the fungus tends to increase with time (Vasiliauskas et al. 1996). Wounds exceeding 50 cm^2 in size make good entrance points for *S. sanguinolentum* (Schönhar 1975, Roll-Hansen and Roll-Hansen 1980, Vasiliauskas et al. 1996). Most suitable temperature range for infections of *S. sanguinolentum* lays between –8.3 and +5.0 °C (Kallio and Hallaksela 1979) and non-vegetation period wounds are more susceptible to establishment by the fungus as compared with vegetation period injuries (Etheridge 1969, Vasiliauskas and Stenlid 1998b).

It is known that VCGs of the fungus may be descendants from the same spore source (Ainsworth 1987) and, on the local scale, are composed from genetically identical or nearly identical individuals (Stenlid and Vasiliauskas 1998). However, no spatial clustering of the isolates from the same VCGs was observed in the stand, and a similar result has been reported in an earlier Swedish study (Vasiliauskas and Stenlid 1998a). The number of isolates within a VCG differed to a rather wide extent, from 1 till 10 (Fig. 2). It is very likely that VCGs of *S. sanguinolentum* consisting of more isolates, have certain advantages in colonizing wound surfaces of *P. abies*. Contrary to the expectations, isolates from the largest VCG exhibited generally slower growth rates within the stems and the variation of spread within this VCG was higher than among different VCGs (Fig. 5, Table 1). Its high infection frequency therefore may simply be due to higher concentration of spores in the air during infection period coming from closely situated or very active fruit bodies. Moreover, correlations in Fig. 5 are low and provide only weak support for the conclusion that *S. sanguinolentum* VCGs which infect more wounds generally grow more slowly within stems. Reason for high variation in growth among representatives from the largest VCG could be differences in resistance of individual *P. abies* trees to spread of decay fungi. Number of studies have shown wide variation in mycelial extent within stems of *P. abies* following artificial inoculations with *H. annosum* (Kaufmann et al. 1980, Ekman and Weissenberg 1981, Thibault-Balesdent and Delatour 1985, Dimitri and Schumann 1989, Hallaksela 1993, Huse and Venn 1994) and similar has

been reported for *S. sanguinolentum* (Pawsey and Stankovicova 1974b, Hallaksela 1993). In contrast to studies based on artificial inoculations, during our work natural infections of *S. sanguinolentum* were followed, and therefore the precise moment when the infections occurred is not known. Different isolates could have been entering the trees in our sample plot stepwise year after year, so the period of fungal growth inside stems could differ significantly for each individual, thus bringing in an additional source of variation.

On the other hand, artificial inoculations often are unable to provide information regarding natural fungal behaviour in forest ecosystems. This was shown by the study of Hallaksela (1993), where the colonization of wood in living *P. abies* stems by inoculated *H. annosum* and *S. sanguinolentum* proceeded successfully during 2–3 growing seasons, which was followed by complete disappearance of the fungi after 5 years. In a similar study, Pawsey and Stankovicova (1974b) reported that after 9 months, growth of *S. sanguinolentum* was slower in inoculated and sealed wounds on *P. abies* stems as compared with open injuries. However, following natural infections this fungus can continue to cause decay in *P. abies* trees over a period of several decades (Isomäki and Kallio 1974, Hallaksela 1984, Vasiliauskas et al. 1996).

Average length of *S. sanguinolentum* decay columns found in the present study was 291.5 cm, therefore mean annual spread of the fungus consisted of 42 cm during 7 years, taken infections occurred during the first year after wounding. This is higher growth rate than previously reported. Following natural infections upward yearly extension of *S. sanguinolentum* varied between 10–40 cm within the period of 1–4 years (Pawsey and Stankovicova 1974a, Kallio 1976, Roll-Hansen and Roll-Hansen 1980, Solheim and Selås 1986). In *P. abies* stems with 4–8 years old extraction wounds length of *S. sanguinolentum* decay column most often fell into the range of 1–2 m (Pawsey and Stankovicova 1974a; El Atta and Hayes 1987). 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, as well as between radial growth

rate of tree and radial penetration of decay (El Atta and Hayes 1987). Except for correlations between stem DBH and decay cross-section area at the stump ($r = 0.316$, $P < 0.05$), and stem DBH and proportion of decayed stump area ($r = -0.719$, $P < 0.001$), other relationships between tree or wound parameters and extent of *S. sanguinolentum* decay revealed during the present work were not significant.

It is already known, that decay column of *H. annosum* usually extends to a height about 20–22 times exceeding its diameter at the stump (Zycha et al. 1970, Kallio and Tamminen 1974, Swedjemark and Stenlid 1993). Length of decay columns caused by *Amylostereum areolatum* (Fr.) Boid. and *Amylostereum chailletii* (Pers.: Fr.) Boid. in wounded spruce stems was exceeding the decay diameter at the stump to a ratio of 21.8 and 19.8, respectively (R. Vasiliauskas unpublished data). In the present work, average diameter of *S. sanguinolentum* decay column at the stump level calculated accordingly to mean decay area (142.3 cm^2), would approximate to 13.5 cm. Therefore for *S. sanguinolentum* length v.s. diameter ratio of decay columns approximates to 21.6, and is very similar to that reported for other decay fungi in *P. abies* stems.

Despite uniformity of initially debarked area on all stems (15×20 cm), wound sizes on individual trees showed high variation at the time of study (after 7 years) (Fig. 6). During the previous work considerable tree-to-tree variation in wound closure rates for *P. abies* was also recorded and average width of a wood layer covering the edge of debarked area each year was estimated as 1.5–2.0 mm (Vasiliauskas 1994). Both studies are in good agreement, since healing rate taken as 2 mm/year, average wound dimensions after 7 years should approximate 12.2 $\times$ 17.2 cm or 209.8 cm^2. Average wound size recorded during the present work was 215.1 cm^2. Note, that due to bark necrosis wounds on several stems had even increased from initial size of 300 cm^2 (Fig. 6). Positive correlations between wound closure rate and post-injury radial growth of spruce during 5 years ($r = 0.439$, $P < 0.01$) and 15 years ($r = 0.484$, $P < 0.001$) were recorded in the previous work (Vasiliauskas 1994). The present study showed insignificant relationship between prior-injury radial growth and

wound area that remained open after 7 years ($r = -0.206$, $P > 0.05$) (Fig. 6).

Acknowledgements

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