

# Spread of *Amylostereum areolatum* and *A. chailletii* decay in living stems of *Picea abies*

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## Summary

Fifteen injured *Picea abies* stems infected with *Amylostereum areolatum* and 14 stems with *A. chailletii* were cut and dissected. There were no significant differences between the spread of *A. areolatum* and that of *A. chailletii* within the stems. During the first 10 years after injury, average vertical spread for both species was 2.8 m, and spread over stem cross section constituted approximately 130–160 cm<sup>2</sup>, thus affecting 30–40 per cent of the total cross section area. Tunnels made by woodwasp larvae were encountered in every analysed stem, and in all cases they were situated within the columns of decayed wood. Correlation analyses showed positive relationships between wound size and length of decay, between tree diameter at breast height (d.b.h.) and spread of decay over stem cross section, between tree d.b.h. and length of decay, and between width of annual growth rings and decay cross section area.

## Introduction

*Amylostereum areolatum* (Fr.) Boid. and *A. chailletii* (Pers.: Fr.) Boid. are decay fungi occurring saprotrophically on fallen trunks and on stumps of *Picea* spp. and *Abies* spp. (Eriksson and Ryvarden, 1973; Eriksson *et al.*, 1978; Breitenbach and Kränzlin, 1986). In central and northern Europe *A. areolatum* is also known as the cause of wound decay in *Picea abies* (L.) Karst., usually infecting 3.5–20 per cent of open bark wounds on living trees (Pechmann and Aufsess, 1971; Schönhar, 1975; Koch and Thongjiem, 1989; Vasiliauskas *et al.*, 1996; Vasiliauskas and Stenlid, 1998a). In Byelorussia, *A. areolatum* is reported from 25–83 per cent of mechanically injured *P. abies* stems (Kovbasa, 1996). Also *A. chailletii* occasionally

invades living *P. abies* stems with various types of bark injury (Vasiliauskas *et al.*, 1996; Vasiliauskas and Stenlid, 1998a), although this fungus is more common in injured trees from other conifer species in the British Isles (Pawsey and Gladman, 1965; Pawsey, 1971; Redfern *et al.*, 1987) and North America (Parker and Johnson, 1960; Hunt and Krueger, 1962; Etheridge and Morin, 1963; Wallis and Morrison, 1975; Aho, 1977; Hinds *et al.*, 1983; Aho *et al.*, 1987; Rizzo and Harrington, 1988).

Both *A. areolatum* and *A. chailletii* are associated with woodwasps from the genera *Sirex* and *Urocerus* which are able to introduce the fungi into living trees (Stillwell, 1966; Coutts and Dolezal, 1969; Thomsen, 1996). During the 1940s and 1950s, combined attacks by *Sirex noctilio* F. and *A.*

*areolatum* devastated *Pinus radiata* D. Don plantations on hundreds of thousands of hectares in New Zealand, Tasmania and Australia (Talbot, 1977). Injured trees exuding resin are particularly attractive to woodwasps (Rawlings, 1948; Gilmour, 1965; Titze and Mucha, 1965), and recent studies have indicated that either fungus, but especially *A. areolatum*, may often be transmitted to wounded *P. abies* by woodwasps (Thomsen, 1996; Vasiliauskas *et al.* 1998).

The fungus in the *Amylostereum*-woodwasp symbiosis provides food resources for the larvae in the form of mycelium (Francke-Grossmann, 1939; Parkin, 1942) and reduces moisture content of wood so that the eggs hatch and the larvae bore in relatively dry wood (Coutts and Dolezal, 1965). The life cycle of the insects is completed in 3-4 years (Schwerdtfeger, 1957).

The authors' earlier study indicated that fungal successions may occur in wounds on stems of *P. abies*: some species of decay fungi are more common in fresh wounds, while others are more frequent in older wounds (Vasiliauskas *et al.*, 1996). According to Stillwell and Kelly (1964), *A. chailletii* causes incipient decay in balsam fir (*Abies balsamea* (L.) Mill.), but is replaced after one year by *Trichaptum abietinum* (Fr.) Ryv. The following questions may therefore be asked. Do the *Amylostereum* fungi disappear from the wood of wounded *P. abies* stems after the life cycle of the woodwasps is completed, or do they continue to develop and spread within stems, thus causing decay? What are the losses in the latter case? Do tree and/or wound parameters affect subsequent development of the fungi? The aim of the present study was to provide answers to these questions.

### Materials and methods

The studied forest area was located in central Lithuania, 10 km east of Kaunas (54° 55' N, 24° 02' E). During preceding work, 540 wounded *P. abies* stems were investigated: 19 (3.5 per cent) were found to contain *A. areolatum*, whereas 14 (2.6 per cent) were infected by *A. chailletii* (Vasiliauskas and Stenlid, 1998a). Sampling of stems and isolation of fungi were carried out as described by Vasiliauskas *et al.* (1996). 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. Fungal colonies were subcultured after 10-15 days of growth, and species were identified in pure culture according to descriptions by Nobles (1965) and Stalpers (1978). All sampled trees were consecutively numbered and their diameter at breast height (d.b.h.) was measured. Wound sizes on each stem were estimated as described by Vasiliauskas *et al.* (1996).

Fifteen *P. abies* stems with *A. areolatum* and 14 stems with *A. chailletii* were included in further analyses. Selected trees were cut, dissected and the following parameters measured: age of tree, age of wound, radial increment of tree at the wound cross section during the 10 years before injury, extent of decay within stem. The age of each 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 the width of ten annual growth rings formed during the years before wounding. The vertical extent of decay was estimated by cutting the stems into sections 1 m long. The last section after which absence of decay was noted, was sliced into 5 cm discs up to the end of the decayed wood, and the total length of the decay column was then recorded. The lateral distribution of decay over stem cross section was measured at the site of maximal wound width by marking the total stem cross section area and the 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<sup>2</sup> of the same plastic. The same principle was applied previously for measuring wound sizes (Vasiliauskas *et al.*, 1996).

Correlations between tree and *Amylostereum* decay parameters were calculated, and the extent of *A. areolatum* and *A. chailletii* decay in stems was compared using the *t*-statistic (Clarke, 1989).

### Results

*Picea abies* trees included in the analyses were 50-60 years of age, 7-28 cm in d.b.h. bearing wounds 17-1414 cm<sup>2</sup> in size that were made 7-24 years ago. All selected stems had woodwasp emergence holes on the wound surfaces (Figure 1a).

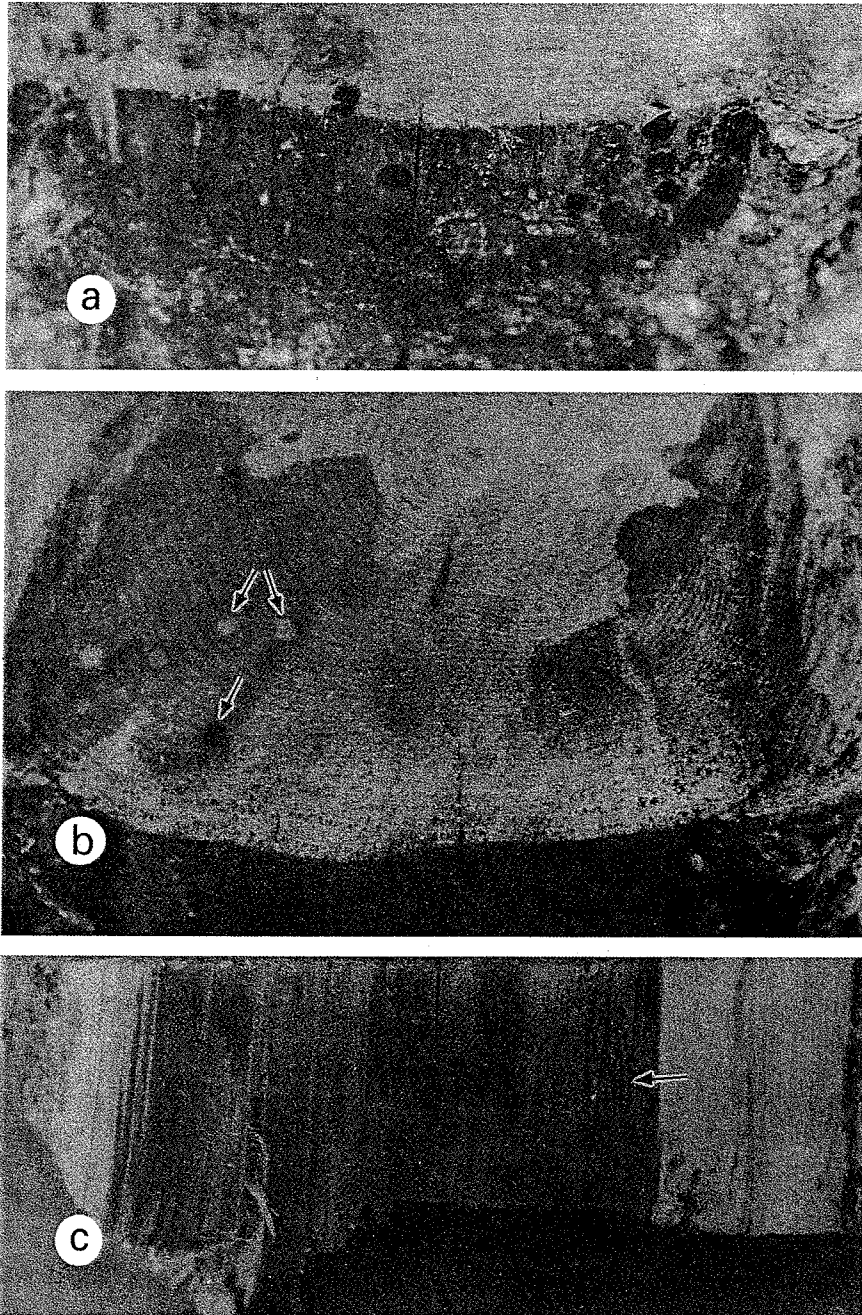


Figure 1. Patterns of woodwasp-*Amylostereum* attack in wounded stems of *Picea abies*: (a) surface of 9-year-old wound showing woodwasp emergence holes, (b) cross section of 7-year-old wound showing *Urocerus gigas* larvae tunnels (arrows) within wood decayed by *Amylostereum chailletii*, (c) longitudinal section of *Amylostereum areolatum* decay column above the 10-year-old wound; tunnels of *Sirex juvencus* larvae indicated by the arrow. Diameter at breast height of the stems was 18–22 cm, and diameter of woodwasp tunnels was approximately 4 mm

Data reflecting main stem, wound and decay parameters of analysed trees are summarized in Table 1. They indicate that, first, there were no significant differences between the two sets of stems, neither regarding their size, prior-injury growth rate, age nor extent of wounding. Second, there were no significant differences between the spread of *A. areolatum* and that of *A. chailletii* within injured stems of *P. abies*. During the first 10 years after injury the average vertical spread for both species was 2.8 m, and the spread over stem cross sections constituted approximately 130–160 cm<sup>2</sup>, thus affecting 30–40 per cent of total cross section area (Table 1). Maximal decay length of *A. areolatum* was 430 cm, and that of *A. chailletii* 442 cm. Maximal decay expansion over stem cross section was 58 per cent for *A. areolatum* and 79 per cent for *A. chailletii*. Tunnels made by woodwasp larvae were encountered in every analysed stem, and in all cases they were situated within the columns of decayed wood (Figure 1b, c). Decay columns developed in wood formed before wounding and, with time, tended to form a heartrot (Figure 1c). Although the insects were not specially looked for during the present study, in one of the stems infected by *A. areolatum* an emerging adult of *Sirex juvencus* was found, and in one of the stems infected by *A. chailletii*, pupae of *Urocerus gigas* were found. Both of these stems were wounded 7 years ago.

Correlation analyses showed positive relationships between wound size and length of decay and between tree d.b.h. and spread of decay over stem cross section; correlation between decay

cross section area and length of decay was statistically not significant (Figure 2). However, due to the limited number of trees examined, in some cases correlations were significant only when stems with both *Amylostereum* species together were analysed. The same was noted for positive correlations between tree d.b.h. and length of decay ( $r = 0.390$ ;  $P < 0.05$ ), and between width of annual growth rings and decay cross section area ( $r = 0.476$ ;  $P < 0.01$ ). These observations, including data in Figure 2b, indicate that following infection by *Amylostereum* fungi, bigger decay columns tend to develop in dominating stems than those in less vigorous stems of *P. abies*. Age of wounds (7–24 years) seemed to have no influence on vertical and lateral spread of decay, and size of wounds had no influence on lateral spread of decay over stem cross section.

## Discussion

Previous data regarding the spread of *Amylostereum* fungi within stems of living trees are rather few and limited. Siepmann (1971) reported that *A. areolatum* can cause an active rot within living stems of *P. abies*. According to Vaartaja and King (1964), *A. areolatum* has a growth rate in wood of living *P. radiata* of 50 mm in 4 months. Koch and Thongjiem (1989) examined five *P. abies* trees with 2½ year-old wounds infected with *A. areolatum* and found that columns of discoloured wood in stems were 40–190 cm long. It was noted that also *A. chailletii* is an active

Table 1: Means of parameters ( $\pm$  standard deviations) of analysed *Picea abies* trees, wounds and *Amylostereum* decay columns compared by *t*-test

Parameter	Stems with <i>Amylostereum</i> spp.		Difference significant at
	<i>A. areolatum</i> 15 trees	<i>A. chailletii</i> 14 trees	
Stem diameter (cm)	17.9 $\pm$ 6.1	17.6 $\pm$ 4.3	$P = 0.91$ n.s.
Wound size (cm <sup>2</sup> )	300.2 $\pm$ 215.9	361.2 $\pm$ 359.7	$P = 0.58$ n.s.
Wound age (years)	10.1 $\pm$ 4.5	9.9 $\pm$ 3.1	$P = 0.89$ n.s.
Prior-injury radial growth (cm10 <sup>-1</sup> )	2.0 $\pm$ 1.6	2.3 $\pm$ 1.6	$P = 0.63$ n.s.
Decay length in stem (cm)	277.3 $\pm$ 86.4	281.3 $\pm$ 108.3	$P = 0.91$ n.s.
Decay at wound cross section (cm <sup>2</sup> )	127.3 $\pm$ 117.2	157.9 $\pm$ 154.1	$P = 0.55$ n.s.
Decayed stem cross section area (%)	30 $\pm$ 15	38 $\pm$ 22	$P = 0.24$ n.s.

n.s., not significant

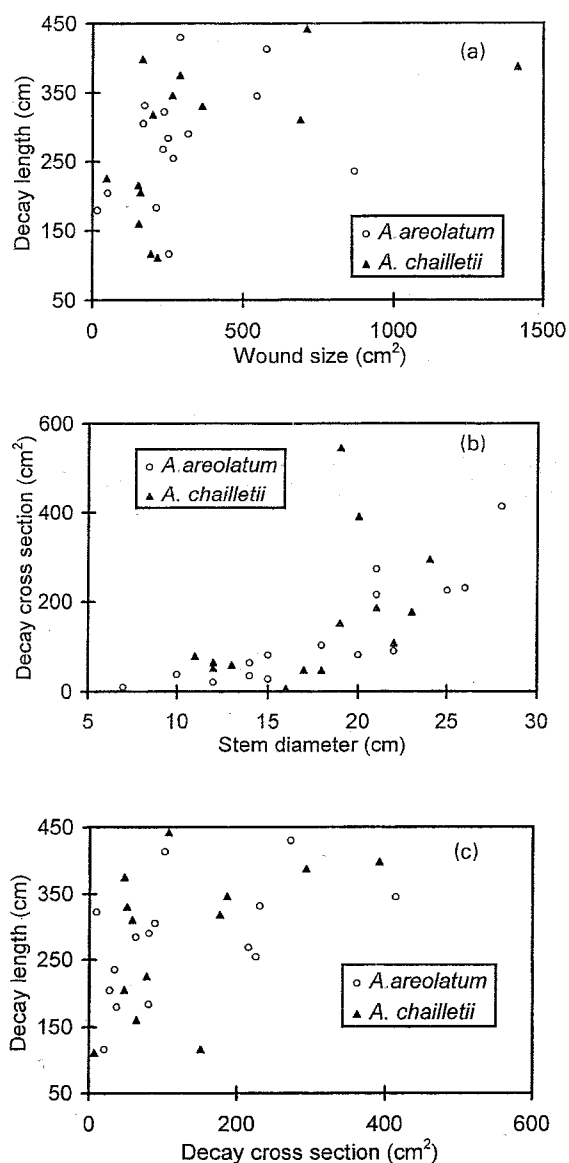


Figure 2. Spread of *Amylostereum* spp. in stems of *Picea abies*: (a) vertical spread of decay in relation to wound size (for both species  $r = 0.434$ ;  $P < 0.05$ ), (b) decay area at wound cross-section in relation to stem diameter (for both species  $r = 0.648$ ;  $P < 0.001$ ), (c) vertical spread of decay in relation to decay area at wound cross section (for both species  $r = 0.328$ ;  $P > 0.05$ , correlation not significant)

decayer in injured conifers (Etheridge and Morin, 1963; Redfern *et al.*, 1987), and its decay pattern resembles closely that of *Stereum sanguinolentum* (Alb. & Schw.: Fr.) Fr. (Pawsey, 1971; Hinds *et al.*, 1983). The rate of spread of 13 *A. chailletii* infections associated with 11–17-year-old

wounds on residual corkbark fir was approximately 11 cm year<sup>-1</sup> (Hinds *et al.*, 1983).

Results of the present study indicate that, after infection of *Amylostereum* fungi occurs in living stems of *P. abies*, the fungi continue to spread and develop there during the subsequent years,

resulting in rather extensive and persistent decay columns. Both species seem to remain active within the trees for many years. For example, in this work *A. areolatum* was isolated from 7–16-year-old wounds and *A. chailletii* from 7–24-year-old wounds.

Length of *Amylostereum* decay columns in most cases fell into a range between 1 and 4 m (Figure 2a, c). Very similar data were obtained for *S. sanguinolentum* in *P. abies* stems with 7-year-old wounds (Vasiliauskas and Stenlid, 1998b). However, considerably lower rates of *S. sanguinolentum* spread were reported in British studies, where length of decay columns varied between 1 and 2 m after 4–8 years (Pawsey and Stankovicova, 1974; El Atta and Hayes, 1987). Decay caused by *Heterobasidion annosum* (Fr.) Bref. in living *P. abies* trees usually spread up to a height of 1–6 m (Swedjemark and Stenlid, 1993). In another study, the average length of *H. annosum* decay columns was found to be 2.3–2.7 m (Perrin and Delatour, 1976).

Decay columns of *H. annosum* in living *P. abies* stems usually extend to a height about 20–22 times more than their diameter at the stump (Zycha *et al.*, 1970; Kallio and Tamminen, 1974; Swedjemark and Stenlid, 1993). For *S. sanguinolentum*, length vs. diameter ratio of decay columns is about 21.6 (Vasiliauskas and Stenlid, 1998b). In the present work, average diameter of *A. areolatum* and *A. chailletii* decay columns at the stump level calculated accordingly to mean decay area in Table 1 (127.3 and 157.9 cm<sup>2</sup>), would approximate to 12.7 and 14.2 cm, respectively. Therefore the length of decay columns of *A. areolatum* and *A. chailletii* is exceeding the decay diameter at the stump by a ratio of 21.8 and 19.8, respectively, and the ratio is very similar to that reported for *H. annosum* and *S. sanguinolentum*.

Results presented in Table 1 indicate that the mean annual spread of both *A. areolatum* and *A. chailletii* in living *P. abies* trees was at least 28 cm, taken that infections occurred during the first year after injury. However, this does not seem likely in all cases, because emerging adults as well as pupae were found in two trees with 7-year-old wounds, and the life cycle of woodwasps is 3–4 years long (Schwerdtfeger, 1957). No correlation was found during this study between age of wounds and spread of *Amylostereum* decay. The most probable explanation

is that the spread of the decay fungi that infect wounds is more pronounced during the first post-injury years, whereas wounds examined in the present work were aged between 7 and 24 years. When stems of *P. abies* were artificially injured and natural infections of fungi were investigated within a period of 1–4 years, yearly upward extension of *S. sanguinolentum* was found to vary between 10 and 40 cm (Pawsey and Stankovicova, 1974; Kallio, 1976; Roll-Hansen and Roll-Hansen, 1980a; Solheim and Selås, 1986; Vasiliauskas and Stenlid, 1998b). Maximal growth rates of *Cylindrobasidium evolvens* (Fr.) Jül. and *H. annosum* have been reported between 5–30 cm and 8–100 cm respectively (Roll-Hansen and Roll-Hansen, 1980a; Solheim and Selås, 1986). However, many authors have reported the decreasing advance of decay in conifer stems with older wounds (Ekbohm, 1928; Parker and Johnson, 1960; Isomäki and Kallio, 1974; Vasiliauskas, 1993).

The present work provides some evidence that the development of *Amylostereum* fungi in living *P. abies* stems is favoured by good radial increment and tree d.b.h. Studies on *H. annosum* have indicated that the spread of this fungus is also more intense *P. abies* with a faster radial growth (Curtois, 1970; Isomäki and Kallio, 1974; Dimitri and Schumann, 1989), and similar findings were reported for the wound-invading fungus, *S. sanguinolentum* (El Atta and Hayes, 1987). Apart from *Amylostereum* spp., wound size was also shown to enhance development of other wound-invading basidiomycetes in *P. abies* (Isomäki and Kallio, 1974; Roll-Hansen and Roll-Hansen, 1980b; El Atta and Hayes, 1987; Vasiliauskas, 1993).

This work has, therefore, shown that both *A. areolatum* and *A. chailletii* can, in some cases, be serious pathogens of *P. abies* and cause considerable decay in living stems. However, their infections in wounded trees in northern Europe are not as common as those of *S. sanguinolentum* (Kallio, 1976; Roll-Hansen and Roll-Hansen, 1980a; Vasiliauskas *et al.*, 1996; Vasiliauskas and Stenlid, 1998a) and, consequently, they are of less practical importance. However, towards the southern boundaries of the *P. abies* area, e.g. in Germany, Denmark and Byelorussia (Pechmann and Aufsess, 1971; Koch and Thongjiem, 1989; Kovbasa, 1996), *Amylostereum* decay may be an important cause of wood production losses in *P. abies* stands.

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