Management of *Pinus sylvestris* Stands Infected by *Gremmeniella abietina*

Aspects of Tree Survival, Growth and Regeneration after the Severe Outbreak in 2001

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Abstract

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The pathogen *Gremmeniella abietina* causes severe damage to native *Pinus sylvestris* and introduced *Pinus contorta* var. *latifolia* in Sweden. The recent *G. abietina* outbreak in 2001–2003, caused by the large tree type (LTT), affected at least 480,000 ha of middle-aged *P. sylvestris* stands and forced Swedish forest owners to sanitary clear-cut large areas of valuable forests. There was, however, little knowledge of survival and growth loss of infected trees and risks involved in replanting *P. sylvestris* in the *G. abietina*-infected slash.

In this thesis, *G. abietina* disease incidence on *P. sylvestris* seedlings planted after sanitation felling was studied, with and without removal of infected *P. sylvestris* slash. Furthermore, survival and vitality of *G. abietina* in the slash was studied by spore germination tests of pycnidia collected from infected slash at regular intervals. One year after planting, *G. abietina* pycnidia were found on 32% of the control seedlings and total infection, including stem cankers, reached 44%. Removal of infected slash reduced the number of infected seedlings by 50% and seedling mortality by 27%, one year after planting. The vitality of *G. abietina* in the slash was as high in pycnidia collected in 13- to 18-months-old slash as in pycnidia collected in fresh slash. It is recommended to wait at least two years after sanitary fellings before replanting with *P. sylvestris*.

To improve the accuracy of predictions of mortality and growth losses in infected, pole-sized *P. sylvestris* stands, mortality, diameter growth and insect colonisation were monitored and related to crown defoliation in four 40-year-old stands in 2001–2005. Of the killed trees, 84% were at least 90% defoliated the year before they died and trees with less than 90% defoliation were only killed in the initial phase of the outbreak. A majority of the trees were killed directly by *G. abietina* whereas less than one third died after colonisation by pine shoot beetle (*Tomicus piniperda*), a pest that was mainly found on dead trees or trees with at least 95% defoliation. A greater fraction of small trees died and generally did so more rapidly than larger trees. Regression analysis indicated that a mean defoliation of 2/3 of the crown resulted in an average loss of 50% in diameter increment. Based on this study, a defoliation limit of 75–80% is recommended for sanitary cutting of *P. sylvestris* trees in the initial phases of an outbreak, and a limit of *ca*. 90% for trees that survive the initial phases of an outbreak.

The interest in the productive *P. contorta* as an alternative to *P. sylvestris* is currently increasing in Sweden. To study the resistance of *P. contorta* to LTT *G. abietina*, seedlings of *P. contorta* and *P. sylvestris* were planted in gaps of an infected 40-year-old *P. sylvestris* stand in 2005. After two years, 45% of the *P. contorta* seedlings and 32% of the *P. sylvestris* seedlings were infected. However, mortality was lower and the mean length of infected tissue on surviving seedlings significantly shorter in *P. contorta* (3.9 cm) compared to *P. sylvestris* (10.4 cm). Furthermore, 47% of the *P. contorta* seedlings had developed new leader shoots in 2007, compared to 19% of the *P. sylvestris* seedlings. Histopathological examinations of infected shoots showed that both *P. contorta* and *P. sylvestris* produce ligno-suberised boundaries that are involved in the active defence in the shoots, preventing major crown dieback. The results indicate that *P. contorta* is more resistant to LTT *G. abietina* than *P. sylvestris.*

Keywords: defoliation, diameter increment, disease resistance, fungal pathogen, growth loss, lodgepole pine, *Pinus contorta*, sanitation, Scleroderris canker, Scots pine, slash.

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Appendix

Papers I – IV

This doctoral thesis is based upon the following papers, hereafter referred to by their respective Roman numerals:

I. Witzell, J., Bernhold, A. & Hansson, P. (2006). Survival and vitality of *Gremmeniella abietina* on *Pinus sylvestris* slash in northern Sweden. *Forest Pathology*. *36*, 406-412.

II. Bernhold, A., Witzell, J. & Hansson, P. (2006). Effect of slash removal on *Gremmeniella abietina* incidence on *Pinus sylvestris* after clear-cutting in northern Sweden. *Scandinavian Journal of Forest Research*. *21*, 489–495.

III. Bernhold, A., & Witzell, J. Tree mortality, increment loss and foliage recovery in middle-aged *Pinus sylvestris* following defoliation by *Gremmeniella abietina* and subsequent attack by *Tomicus piniperda* (Submitted manuscript)

IV. Bernhold, A., Hansson. P., Rioux, D., Simard, M. & Laflamme, G. Resistance to *Gremmeniella abietina* (EU race, large tree type) in introduced *Pinus contorta* and native *Pinus sylvestris* in Sweden (Submitted manuscript).

Introduction

The pathogen *Gremmeniella abietina* (Lagerb.) Morelet is the causal agent of Scleroderris canker on conifers in North America, Europe and Asia (Roll-Hansen, 1964; Yokota *et al.*, 1974; Setliff *et al.*, 1975; Uotila, 1983; Skilling *et al.*, 1986; Karlman, 1987; Barklund, 1990; Laflamme *et al.*, 1998). The disease has been found on several conifer species in the northern hemisphere, *e.g.* spruces, firs, larches, Douglas fir and juniper, but its economic damage is most significant on pines in northern Europe and north-eastern North America (Donaubauer, 1972b; Roll-Hansen, 1972; Skilling & Riemenschneider, 1984). In Fennoscandia, *G. abietina* is the most severe shoot pathogen on native *Pinus sylvestris* L. (Lagerberg, 1912; Roll-Hansen, 1964; Barklund, 1990; Wulff *et al.*, 2006) and introduced *Pinus contorta* var. *latifolia* Engelm. (Karlman *et al.*, 1994; Karlman, 2001), but also infects and damages *Picea abies* (L.) Karst. (Lagerberg, 1913; Björkman, 1963; Barklund & Rowe, 1981; Hansson, 1998). The most severe outbreak of *G. abietina* in Sweden so far took place in 2001–2003 and almost 500,000 ha of middle-aged pine-dominated forests were attacked. To control the disease many *P. sylvestris* stands were sanitary thinned or clear-cut (Wulff *et al.*, 2006), resulting in large costs for the Swedish forest owners due to additional sanitary management and reduction in income (Hansson *et al.*, 2005a). However, the knowledge of effective management of infected stands was scarce and general guidelines for sanitary fellings and regenerations were few and often based on research on other pest species. Thus, several applied research projects were immediately initiated to facilitate future control of the pathogen and serve as a knowledge base for future decision makers. To be able to prevent and control severe diseases it is, however, of great importance to have genuine knowledge of their biology and symptom development. Therefore, this thesis starts with a thorough introduction of the different types of *G. abietina* along with a historic background of outbreaks in Sweden.

Taxonomy and disease symptoms on *Pinus* spp.

The genus *Gremmeniella* has been classified into several species, varieties, races and types, more or less specified to environment and host species (Dorworth & Krywienczyk, 1975; Uotila, 1983; Petrini *et al.*, 1989; Hellgren & Högberg, 1995; Hamelin *et al.*, 1996) (Fig. 1). Such taxonomic knowledge is vital when attempting to control the disease since the epidemiology and symptoms of *G. abietina* differs among the recognised races and types.

So far, three species are recognised and confirmed within the genus *Gremmeniella*: *G. abietina* (Lagerberg) Morelet, *G. laricina* (Ettinger) Petrini *et al.* and *G. juniperina* L. Holm & Holm (Petrini *et al.*, 1989) (Fig. 1). According to Petrini *et al.* (1989), the latter two appear to be host specific on the *Larix* and *Juniperus* genera, respectively. In a review by Laflamme (2002), several new species within the genus *Gremmeniella* are proposed. Pending the acceptance of this proposal, however, the species *G. abietina* is comprised of two varieties: var.

abietina, found mostly on pines, and var. *balsamea*, found on spruces and firs (Petrini *et al.*, 1989) (Fig. 1).

Three races of *G. abietin*a var. *abietina* are described based on several serological and biochemical studies: the North American, European and Asian races (Dorworth & Krywienczyk, 1975; Petrini *et al.*, 1989; Hamelin *et al.*, 2000) (Fig. 1). In North America ("NA" hereafter), two out of the three races are present: the NA race, infecting seedlings and lower branches covered by snow in winter (Dorworth, 1971; Skilling *et al.*, 1986; Marosy *et al.*, 1989), and the introduced European (EU) race, infecting trees of all ages (Setliff *et al.*, 1975; Laflamme, 1993b; Hamelin *et al.*, 1998). The NA race has so far not been recorded in Europe (Petrini *et al.*, 1990; Hamelin *et al.*, 1996).

Three separate biotypes have been recognised within the European race of *G. abietina*: the small tree type (STT) (syn.: northern type, type B), the large tree type (LTT) (syn.: European type, type A) and the alpine type (Uotila, 1983; Hellgren & Högberg, 1995; Hamelin *et al.*, 1996) (Fig. 1).

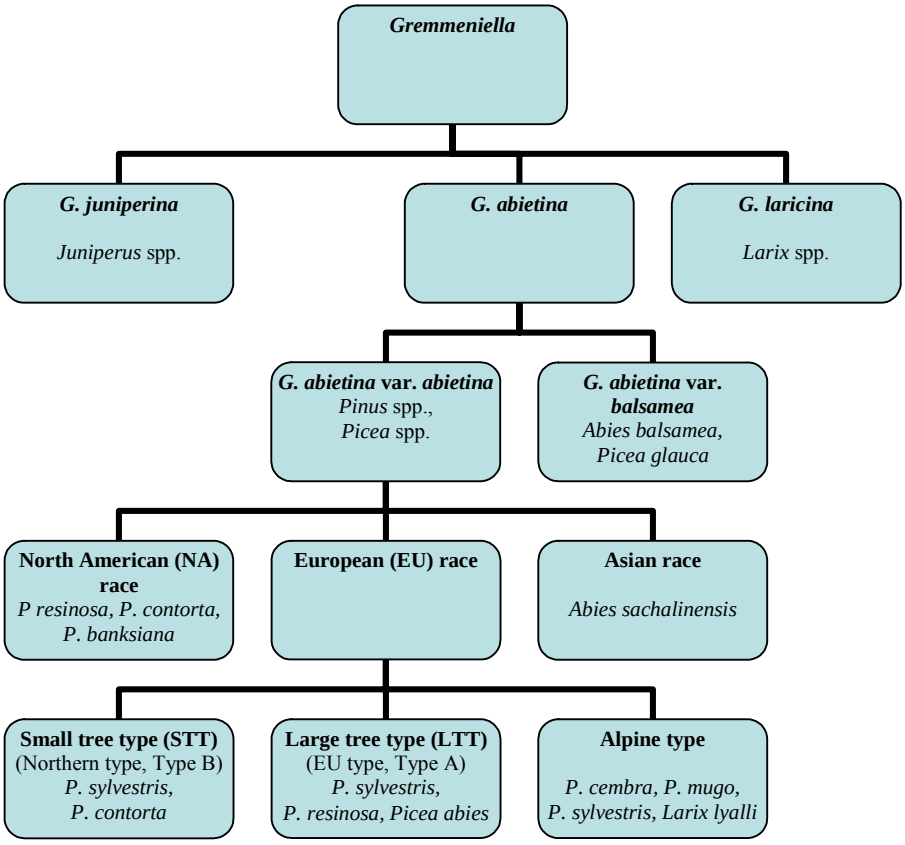

Fig. 1. Current *Gremmeniella* taxonomy and examples of common host species. A revision of this taxonomy, including a proposal for several new species, is found in Laflamme (2002).

STT mainly attacks young *P. contorta* and *P. sylvestris* plantations in high-altitude areas with harsh weather conditions in northern Sweden (Karlman *et al.*, 1994; Hamelin *et al.*, 1996; Hansson *et al.*, 1996). Symptoms of STT *G. abietina* are similar to those of the NA race and the alpine type of the EU race, with abundant canker formation and production of both pycnidia and apothecia on smaller trees, covered with snow in winter (Uotila, 1983; Marosy *et al.*, 1989; Karlman *et al.*, 1994; Hamelin *et al.*, 1996). LTT, the equivalent to the EU race in NA, infects both young and mature *P. sylvestris* along with the tops of understory *P. abies,* and is found in most parts of Sweden with the exception of alpine and the most northern areas (Barklund & Rowe, 1981; Barklund, 1990; Wulff *et al.*, 2006). Symptoms of LTT *G. abietina* on *P. sylvestris* in Sweden are similar to those in central and western Europe, with infections in the crown of both young and pole-sized pines and with a very limited production of apothecia (Uotila, 1983; Hellgren & Högberg, 1995; Hamelin *et al.*, 1996). The alpine type is found exclusively at high altitudes in the Alps of southern Europe, *e.g.* on *Pinus cembra* and *Pinus mugo* (Hamelin *et al.*, 1996).

The initial attack of LTT *Gremmeniella* on pole-sized *P. sylvestris* usually occurs in the lower parts of the crown and, in more severe cases, spreads to branches in the upper crown (Barklund, 1989). In the 2001–2003 outbreak in Sweden, however, the pathogen seemed to infect the whole crown at an initial state of infection, causing rapid tree mortality. Earlier investigations of genetic variation in *G. abietina* in northern Sweden recorded only the northern type (STT) on *P. contorta* and *P. sylvestris* (Hamelin *et al.*, 1996; Hansson *et al.*, 1996). During the 2001–2003 epidemic, however, LTT has been genetically identified as far north as Västerbotten (IV). Kaitera *et al.* (1998), has identified both LTT and STT on *P. sylvestris* saplings within the same stands in northern Finland and Russia. Even though hybridisation between LTT and STT was found possible by Uotila *et al.* (2000), the poor germination rate of the progeny led the authors to conclude that both types are genetically distinct populations. A future separation of LTT and STT *G. abietina* into two distinct species, based on their differences in epidemiology and biochemistry, was also suggested in Laflamme (2002).

Generally, *G. abietina* initially infects and kills buds and current year shoots. The pathogen can then remain latent for several years, or continue to grow along the branch towards the main stem (Blenis *et al.*, 1984; Skilling *et al.*, 1986). On young trees, the disease may spread rapidly over the whole tree and kill it within a few years after infection (Roll-Hansen, 1964; Dorworth, 1971; Skilling, 1972). Even if the trees survive, *G. abietina* may cause a reduction in stand productivity and height increment (Kurkela, 1984b; Witzell, 2001). The first symptoms of *G. abietina* are small spots of necroses forming under the bark (Roll-Hansen, 1964; Skilling *et al.*, 1986). The necrosis forming under the short shoots causes the needles to attach less strongly and cuts off all transport of water and nutrients into the needles (Roll-Hansen, 1964; Skilling *et al.*, 1986). Consequently, the needles are often bent down and coloured red, starting from the base and moving towards the tip (see cover page). After about one year, the dead shoots loose most or all of their red needles and appear dry.

Gremmeniella abietina also causes larger necroses in the inner tissue of stems and branches resulting in cankers on stems and branches (Skilling, 1972; Kurkela, 1981; Witzell, 2001). Stem cankers caused by LTT *G. abietina* are rare in Sweden (Hellgren, 1995b), even though small cankers on branches do occur (Kaitera & Jalkanen, 1994b). On the other hand, stem cankers caused by STT *G. abietina* are frequently found in northern Sweden, especially on *P. contorta* (Karlman *et al.*, 1992; Witzell, 2001). Cankers on the main stem can be formed after infection through the base of diseased branches and through bark damage caused by snow bending and frost and ice formation (Kurkela, 1981; Witzell, 2001). Visible damage is, however, not necessary for direct infection of the main stem, and *G. abietina* is believed to penetrate the needle-bases on the short-shoots as an additional way of infection (Skilling, 1972; Kurkela, 1984a; Witzell, 2001). Large stem cankers reduce height growth (Kurkela & Norokorpi, 1979; Witzell, 2001) and may eventually kill the tree by strangulation (Lagerberg, 1912; Roll-Hansen, 1964). Host reactions by canker formation seem to occur if infections do not kill the shoot during winter or spring (von Siepmann, 1972). Accordingly, infections in late summer seem to result in more and larger cankers than infections in early summer (Kurkela & Norokorpi, 1979; Uotila, 1990). *Gremmeniella* mycelia can survive up to eight years in *P. sylvestris* cankers (Kurkela, 1981) and cankers may, thus, serve as refuges for the pathogen during unfavourable conditions. Then, when conditions turn favourable, the pathogen can rapidly recolonise a stand (Hellgren & Barklund, 1992).

Infection biology and dispersal capacity

Gremmeniella abietina is an ascomycete and generally produces both asexual pycnidia and sexual apothecia on infected tissues of the host. Pycnidia are mainly produced one year after infection (Dorworth, 1972b; Kaitera *et al.*, 1997), and apothecia two years after infection (von Siepmann, 1972; Hellgren & Barklund, 1992). The production of *G. abietina* pycnidia and apothecia differs considerably between regions, races and types. In continental regions, with cold winters and much snow, *G. abietina* seems to produce apothecia more often than in more coastal regions, with mild winters (Hellgren & Barklund, 1992; Roll-Hansen, 1993). It was initially proposed that a harsh climate would promote the production of apothecia (Roll-Hansen, 1982), but later studies indicate that apothecia production, and thus sexual breeding, is genetically determined (Uotila, 1992). Thus, populations of STT *G. abietina*, along with the alpine type and the NA race seem to produce high amounts of apothecia, whereas LTT *G. abietina* (syn. the EU race in North America) mainly regenerate through pycnidia (Skilling *et al.*, 1986; Uotila, 1992; Hellgren & Högberg, 1995; Hansson *et al.*, 1996).

The main dispersal of conidia normally coincides with the development of current year pine shoots in spring and early summer (Donaubauer, 1972a; Luley & Manion, 1984; Hellgren & Barklund, 1992; Kaitera *et al.*, 1997). In southern and central Sweden most conidia are released between April and July, and sporulation ends already in August. (Hellgren & Barklund, 1992). There are no similar studies in northern Sweden but in northern Finland conidia sporulation begins in late June

or early July and continues to late autumn (Kaitera *et al.*, 1997), while in south-central Finland sporulation begins a bit earlier, in late May and early June, and continues to early September (Petäistö & Heinonen, 2003). The peak release of ascospores generally occurs somewhat later in the summer compared to conidia (Laflamme & Archambault, 1990; Hellgren & Barklund, 1992).

Rain and high air humidity enhance the release of both conidia and ascospores of *G. abietina* (Dorworth, 1972b; Skilling, 1972; Luley & Manion, 1984; Petäistö & Heinonen, 2003). Laflamme and Archambault (1990) found surface moisture to be the best indicator variable for ascospore release. Both conidia and ascospores are mainly dispersed by rain splash, which disseminates the spores in small droplets (Dorworth, 1972b; Skilling, 1972; Luley & Manion, 1984). Ascospores can also be dispersed in dry weather. Wind-dispersed *G. abietina* ascospores have been shown to spread 400–700 m, whereas conidia generally only disperse a few meters (Skilling, 1972; Skilling *et al.*, 1986; Wang *et al.*, 1997). However, some investigations have also recorded conidia in dry air (Bergdahl, 1984; Luley & Manion, 1984; Skilling *et al.*, 1986), indicating a limited wind transport. Bergdahl (1984) hypothesises that dispersal of conidia in dry air could involve some kind of redistribution of previously dispersed conidia.

Infection by *G. abietina* in current year shoots and buds occurs in spring and summer, but the pathogen does not colonise the living cells of the host until winter dormancy when defence mechanisms are absent or less effective (Lang & Schütt, 1974; Patton *et al.*, 1984). On living trees *G. abietina* can survive and produce apothecia and pycnidia for at least two years in dead shoots (Hellgren & Barklund, 1992; Kaitera *et al.*, 1997), and in cankers, the fungus can survive even longer (Kurkela, 1981; Hellgren & Barklund, 1992; Witzell, 2001). The pathogen is thus able to infect shoots during several years and survive at least one year of unfavourable weather conditions. Concerning dead branches (slash) on the ground, however, information on *G. abietina* survival is scarce. This question is addressed in study I.

Environmental factors influencing disease outbreak

Gremmeniella abietina seems to be most successful on trees that are stressed by adverse environmental conditions and both pathogen and host resistance are dependent on prevailing weather conditions (Karlman, 1986; Karlman *et al.*, 1994; Senn, 1999). Rainy, cool and cloudy growing seasons often precede increases in damage by *G. abietina* (*e.g.* Uotila, 1988) and have proven conducive for both STT (Karlman *et al.*, 1994) and LTT *G. abietina* (Hansson & Ottosson Löfvenius, 2005) in Sweden (Fig. 2). High humidity and rainy conditions promote conidia and ascospore dispersal and survival in the pathogen (Dorworth, 1972b; Skilling, 1972; Bergdahl, 1984), and reduces the resistance of the host trees (Kurkela, 1984a). Mild winter temperatures have also been demonstrated to promote disease development by *G. abietina* and enhance symptom development the following spring (Marosy & Patton, 1986; Marosy *et al.*, 1989). However, according to Hellgren and Barklund (1992), conducive wintertime temperatures alone are not

sufficient to cause symptom development in pole-sized *P. sylvestris*. Warm and dry growing seasons in between mild winters seem to enable the trees to increase their vitality and resistance against fungal invasion (Hellgren & Barklund, 1992; Karlman *et al.*, 1994). Warm and dry weather reduces spore dispersal capacity and survival (Dorworth, 1972b; Bergdahl, 1984), and prolonged periods of high summer temperatures are likely to kill the fungus at an early stage of infection in outer, unprotected shoot tissues.

Severe *G. abietina* infections are often found in pine forests at high-altitude sites (Karlman, 1986; Roll-Hansen *et al.*, 1992; Wulff & Walheim, 2003) and a strong negative correlation between the severity of STT *G. abietina* and site temperature sum was found in pine plantations in northern Fennoscandia during the 1980s (Uotila, 1988; Karlman *et al.*, 1994). In such northern or high-altitude regions, *G. abietina* seems to be most severe in local cold air depressions in the terrain (Dorworth, 1973; Kaitera & Jalkanen, 1995; Nevalainen, 2002) with deep, long-lasting snow covers (Karlman *et al.*, 1994; Senn, 1999). A prolonged snow cover reduces the growing season and allows the fungus to grow for a longer time under favourable moisture and temperature conditions (Marosy *et al.*, 1989; Senn, 1999). According to Marosy *et al.* (1989), at least 44 days of conducive temperatures (between +6º and -5ºC) are needed between October and February for symptoms to develop. Damage by STT *G. abietina* in young *P. contorta* plantations is often exemplified by young pines whose long-needled, dense crowns are burdened with heavy, wet snow and bent down into the snow (Hansson & Karlman, 1997). Importantly, however, symptom development of LTT *G. abietina* on pole-sized pines also seems to be favoured by mild winter temperatures and cool, rainy growing seasons (Luley & Manion, 1984; Hellgren & Barklund, 1992; Petäistö & Heinonen, 2003; Hansson & Ottosson Löfvenius, 2005).

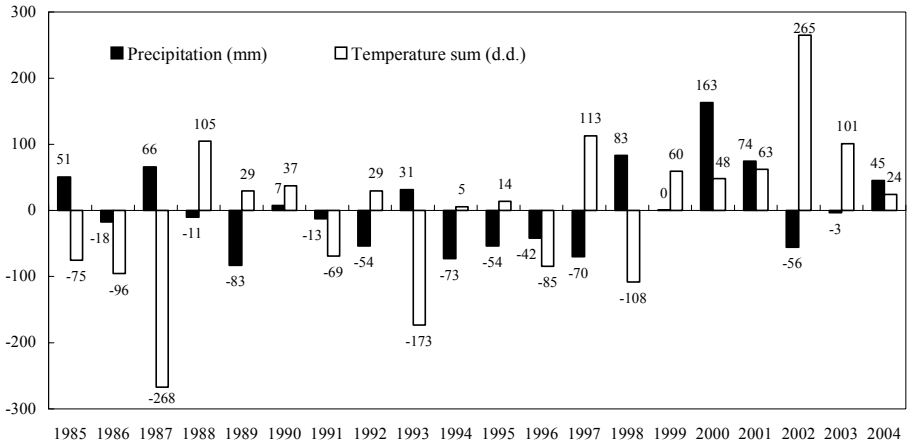

Fig. 2. Divergence in precipitation (mm) and temperature sum (day degrees) during the vegetation period in relation to a 20-year-mean in Sweden. Based on data from 50 weather stations in Sweden (Swedish Institute for Meteorology and Hydrology). Diagram produced by P. Hansson (2005).

Topographic depressions also accumulate water, resulting in moist, nutrient-rich soils. In Fennoscandia, such sites are natural habitats for spruce (*P. abies*) and when the less nutrient demanding *P. contorta* and *P. sylvestris* are planted on such "spruce sites" they have proven more susceptible to *G. abietina* (Lähde, 1974; Karlman *et al.*, 1994; Witzell & Karlman, 2000). The correlation between site fertility and *G. abietina* infection was supported in Nevalainen (1999) and fertilisation increased the damage by *G. abietina* on *P. sylvestris* in Kallio *et al.* (1985). Kaitera and Jalkanen (1995) also found an enhanced vulnerability of *P. sylvestris* to disease infection with increased pH of the humus layer.

Brief history of *G. abietina* outbreaks in Sweden

Gremmeniella was first described in the imperfect (conidial) state in the late 19th century, mainly in plantations of exotic tree species (Rostrup, 1883; Karsten, 1884; Brunchorst, 1888). The first detailed description was given by the Norwegian plant pathologist J. Brunchorst (Brunchorst, 1888) and the conidial state was named *Brunchorstia pinea*. Thirty years later in Sweden, Lagerberg (1912) described the perfect (ascospore) state, which he identified as *Crumenula pinicola*. According to Lagerberg (1912) this disease was common on pine heaths in northern Sweden, and apart from identifying fruiting body morphology he also described stem canker formation on young *P. sylvestris*. Since then the taxonomic status and scientific names has been revised several times. However, *Gremmeniella*, with *G. abietina* as the type species, was proposed by Morelet (1969) and later confirmed in Petrini *et al*. (1989).

The first major *G. abietina* outbreaks in Sweden occurred on *P. sylvestris* in nurseries during the 1950s and early 1960s (Björkman, 1959; Kohh, 1964). During a ten-year-period as many as 75 million pine seedlings were lost in northern Swedish nurseries (Kohh, 1964). The attacks on nurseries led to the development and use of fungicides against *G. abietina*. During the same period, 30- to 50-year-old German provenances of *P. sylvestris* in southern Sweden were infected with *G. abietina* (Kohh, 1964). In northern Sweden, however, damage by *G. abietina* in the 1950s was mainly found on young natural regenerations of *P. sylvestris* on poor heath land (Kohh, 1964). In 1977, *G. abietina* also caused top-dying in *P. abies* stands in southern and south-western Sweden (Barklund & Rowe, 1981). In the late 1980s, a period of extremely cool and rainy summers followed by a mild winter with exceptionally deep snow cover caused a severe outbreak by STT *G. abietina* in young *P. contorta* plantations in northern Sweden (Karlman *et al.*, 1992; Karlman *et al.*, 1994) (Fig. 2). Damage was often predisposed by unstable plants bent over by heavy snow loads (Karlman *et al.*, 1994; Hansson & Karlman, 1997). Studies in 110 conventional plantations in northern Sweden showed that infection by *G. abietina* increased with decreasing temperature sum, diminishing angle of slope, decreasing frequency of birch and on sites where *P. abies* gave better wood yield than *P. sylvestris* (Karlman *et al.*, 1994). The aggressive outbreak led to changes in Swedish policy and limited the possibility of planting *P. contorta* at high elevations areas with harsh climatic conditions (*cf*. Karlman, 2001; Anon., 2006). At the same time, several outbreaks

of LTT *G. abietina* in stands of *P. sylvestris*, and to a smaller extent *P. abies*, were recorded in southern and central Sweden (Barklund, 1990). Stands of all ages were attacked but the disease was most prevalent in 20- to 40-year-old *P. sylvestris*. The outbreaks peaked in 1981, 1985 and 1988, the latter being the most severe (Barklund, 1990).

The severe *G. abietina* outbreak in Sweden in 2001–2003

The largest known *G. abietina* outbreak in Sweden so far occurred in 2001–2003. According to the Swedish National Forest Survey of 2003, an area of 484,000 ha *P. sylvestris*-dominated stands (mainly 30- to 60-years-old) was attacked and 70,000 ha severely damaged by the pathogen (Wulff *et al.*, 2006). This massive attack forced forest owners to sanitary thin and clear-cut at least 50,000 ha of *P. sylvestris* stands during the first two years of the outbreak (Wulff *et al.*, 2006). The most severe infections were reported in central Sweden, in the region of Bergslagen, but the disease was common from the county of Småland in the south up to central Norrland (Fig. 3).

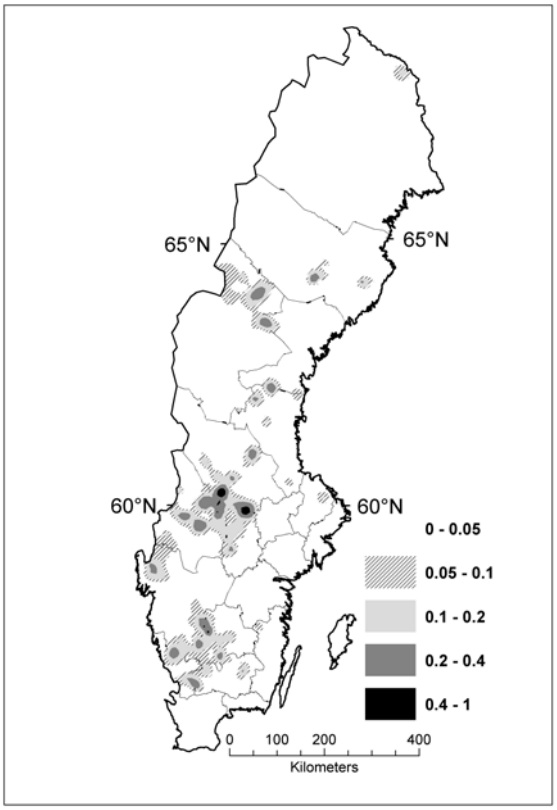

Fig. 3. Distribution of *G. abietina* infection in pine-dominated (>70%) forests in Sweden in 2001–2003. Legend shows proportion of trees with >25% crown defoliation. Produced by P. Hansson, SLU (2004) based on data from the Swedish National Forest Inventory.

There are several possible causes for the massive impact of the latest *G. abietina* outbreak. A major factor that probably influenced both the time and magnitude of the outbreak was the weather conditions. The period preceding the outbreak was characterized by a very cold and wet growing season in 1998 followed by a mild and long winter period in 1998/99 and an extremely wet growing season in 2000 (Hansson & Ottosson Löfvenius, 2005) (Fig. 2). Furthermore, areas most exposed to damage were correlated with areas with more precipitation than average during the dry growing season 1999, and with lower temperature than average during the warm and wet growing season of 2000. Thus, a probable scenario is that the weather conditions in 1998 contributed to the increased disease incidence observed already in 1999. The disease build-up and mass pycnidia production in the infected areas then resulted in a large quantity of mature spores in the early summer of 2000. The extremely rainy growing season in 2000 then greatly facilitated the mass sporulation that resulted in the major *G. abietina* outbreak in 2001.

Another potential factor influencing the severity of the outbreak is the low diversity in the Swedish pine forests. Ever since the mid 20th century, plantation forestry with clear-cuts and even-aged monocultures of *P. sylvestris* and *P. abies* has been the dominant method of silviculture in Sweden (Enander, 2003). In 2001–2003, 60% of the harvested forest area in Sweden was regenerated by planting and 37% by natural regeneration (Anon., 2004). The practice of plantation forestry eases the production process and helps predict forest yields (Savill *et al.*, 1997). It is also a fairly reliable way of securing a quick regeneration by using productive nursery stock. In Sweden, 2003, the rate of regenerations accepted by the Swedish National Board of Forestry was 87% for plantations compared to 71% for natural regenerations (Anon., 2004). However, since monocultures are generally considered more susceptible to disease build-up and the evolution of more aggressive pathogens than mixed stands, due to closer spacing of susceptible host trees and a limited genetic diversity (Karlman, 1981; Skilling, 1988; Savill *et al.*, 1997; Ennos, 2001), a higher species diversity in the Swedish pine plantations may have prevented some of the substantial losses caused by *G. abietina*.

Insufficient site adaptation may also have reduced stand susceptibility to *G. abietina* (Witzell & Karlman, 2000). Since *P. sylvestris* has produced more valuable timber than *P. abies*, it has frequently been planted on fertile sites more suitable for spruce (Lähde, 1974; Aalto-Kallonen & Kurkela, 1985; Witzell & Karlman, 2000). Like in the epidemic on young *P. contorta* in the late 1980s (Karlman *et al.*, 1994), "spruce sites" proved more susceptible to disease also in the recent outbreak on pole-sized trees (Wulff & Walheim, 2003). Furthermore, on many of the plantations originating from 1950s–1970s, provenances of too southern an origin, from a disease resistance point of view, were used. The positive correlation between latitude of origin and resistance of *Pinus* spp. to *G. abietina*, STT as well as LTT, has been confirmed in several studies (Dietrichson, 1968; Björkman, 1972; Karlman, 1984; Uotila, 1988), and such a correlation was found also in the 2001–2003 epidemic (Hansson *et al.*, 2005b).

The latest *G. abietina* outbreak has, more than previous outbreaks, attacked productive, pole-sized *P. sylvestris* stands in northern Sweden, and sanitary fellings has resulted in large quantities of infected slash remaining on the clear-cuts. To set standards for future sanitation management it is therefore important to study *G. abietina* survival in *P. sylvestris* slash and to assess infection rate and mortality of seedlings planted after sanitary clear-cuts. Furthermore, the large economic losses from these stands has highlighted the importance of considering aggressive pathogens in regular stand management. In heavily infected stands, a firm knowledge of survival capacity and growth losses in relation to defoliation will help foresters in the choice of trees and stands for sanitary cuttings.

Resistance to *G. abietina* in *P. contorta* and *P. sylvestris*

Although *G. abietina* has proven capable of infecting several conifers, including several species of *Picea* and *Larix*, damage is by far most significant within the genus *Pinus,* and most pine species have proven more or less susceptible (Donaubauer, 1972b; Roll-Hansen, 1972; Skilling & Riemenschneider, 1984). In a review paper on resistance to *Scleroderris lagerbergii* (syn. *G. abietina*) in different pine species (Roll-Hansen, 1972), the general resistance was believed to be somewhat lower in *P. contorta* than in *P. sylvestris*, but it was stressed that the resistance is very dependent on meteorological and other ecological conditions. This review was, however, written before the separation of *G. abietina* into two genetically and epidemiologically different biotypes (Uotila, 1983; Hellgren & Högberg, 1995; Hamelin *et al.*, 1996). STT *G. abietina* is not host specific to the species *P. sylvestris* and *P. contorta* in northern Sweden (Hellgren, 1995a; Hansson *et al.*, 1996) but several field experiments in Fennoscandia indicate that *P. contorta* is more susceptible than *P. sylvestris* (Karlman, 1987; Jalkanen, 1990; Karlman *et al.*, 1994; Hansson & Karlman, 1997; Witzell & Karlman, 2000). However, *P. contorta* seems to survive and overcome physical damage, such as *G. abietina* stem wounds, better than *P. sylvestris*, even though the quality of the surviving *P. contorta* trees declines, with cankers, dead tops and double stems (Ericsson, 1991; Witzell & Karlman, 2000). In Hansson (1998), however, *in situ* inoculations with STT *G. abietina* caused significantly higher mortality and infection severity on *P. sylvestris* seedlings compared to *P. contorta* seedlings even though the proportion of infected seedlings was equal between the two species. The author concluded that *P. contorta* and *P. sylvestris* probably have the same "genuine" susceptibility to STT *G. abietina*, although the introduced *P. contorta* is more affected by predisposing stress factors as extreme weather conditions, due to its wide and dense crowns. Accordingly, the severe outbreak of STT *G. abietina* in northern Sweden in the late 1980s occurred after a period of extremely cool and rainy summers followed by a mild winter with heavy snow loads on the trees (Karlman, 1987, 1990; Karlman *et al.*, 1994).

The higher susceptibility of introduced species to *G. abietina*, as a result of a lower adaptation to the environmental conditions, has been described in several additional publications over the years (Brunchorst, 1888; Roll-Hansen, 1972;

Karlman, 1981). Regarding LTT *G. abietina*, a screening experiment of conifers in New York State found both *P. contorta* and *P. sylvestris* to be highly susceptible, based on the proportion of infected seedlings, and the authors expressed a fear of severe damage in the natural range of *P. contorta* if the pathogen were to spread into western North America (Skilling & Riemenschneider, 1984). Later studies, however, have found *P. contorta* to be more resistant to LTT *G. abietina* compared to *P. sylvestris* (Aitken, 1993) and *P. resinosa* (Laflamme *et al.*, 2006) based on higher survival rates and lower disease incidence on individual trees. Laflamme *et al.* (2006) showed that infected *P. contorta* shoots may form ligno-suberised defence barriers, limiting the infection to the outermost part of the shoots, and preventing major crown dieback. If *P. contorta* would prove more resistant than *P. sylvestris* to LTT *G. abietina*, that caused the most severe epidemic ever recorded in Sweden, this could be vital information for risk assessments in future planting programs with *P. contorta* in Sweden.

Preventive management

The most effective control of *G. abietina* is by prevention. Effective preventive measures are the choice of proper tree species for each specific site (Aalto-Kallonen & Kurkela, 1985; Karlman *et al.*, 1994; Witzell & Karlman, 2000) and the use of healthy seedlings of hardy provenances (Dietrichson, 1968; Roll-Hansen, 1972; Hansson, 1998). Furthermore, proper management by cleaning, thinning and, in some cases, pruning, is believed to improve stand resistance to *G. abietina* by increasing the fitness of the trees and by creating a less suitable environment for the pathogen (Bergdahl & Ward, 1984; Skilling *et al.*, 1986; Niemelä *et al.*, 1992).

One major preventive action is to avoid planting *P. sylvestris* and *P. contorta* on fertile sites more suitable for *P. abies* and broadleaved species. There are, however, other alternative conifer species that are suitable for such “spruce sites”, and even less prone to infection by *G. abietina* than *P. abies*, *e.g*. Siberian larch (*Larix sibirica* Ledeb.) in northern Sweden and Sitka spruce (*Picea sitchensis* (Bongard) Carriére) in coastal areas of southern Sweden. Even though these two species were found susceptible to the EU race of *G. abietina* (=LTT) in a screening study by Skilling and Riemenschneider (1984), no reports of damage from commercial plantations have yet been reported.

Several studies have found naturally regenerated stands to be less affected by *G. abietina* compared to plantations (Lähde, 1974; Kallio *et al.*, 1985). Additionally, as many severe *G. abietina* outbreaks in harsh climates are found on open, exposed sites with low temperature (Kohh, 1964; Karlman *et al.*, 1994), some kind of shelterwood system, *e.g.* seed trees, may reduce infection build-up; as long as the previous stand is not infected (Skilling, 1988). When planting, the choice of provenance is vital and susceptibility to *G. abietina* decreases with increasing latitude of seed origin, as shown in both *P. sylvestris* (Dietrichson, 1968; Björkman, 1972; Uotila, 1988; Hansson, 1998) and *P. contorta* (Hagner & Fahlroth, 1974; Dietrichson & Solheim, 1987; Karlman, 1987). However, even the

most northern provenances of *P. contorta* (from the Yukon territory in Canada), where severely damaged by STT *G. abietina* in harsh climatic regions of Sweden during the 1987–92 outbreak (Karlman *et al.*, 1994). In a study on scarification systems, mortality of *P. contorta* seedlings, after a severe attack of STT *G. abietina,* was found lower on mounded plots compared to untreated plots or plots with patch scarification (Hansson & Karlman, 1997). Early planting in harsh boreal climates has proven crucial for high survival of *P. sylvestris* (Ericsson *et al.*, 1983; Hansson & Karlman, 1997), but insignificant for survival of *P. contorta* (Hansson & Karlman, 1997), possibly due to the faster root establishment phase of *P. contorta* (Norgren, 1996).

One potential way of controlling *G. abietina* infection is by thinning before the stands become too dense (Skilling, 1988). Finnish studies have found that dense pure stands of *P. contorta* (Kujala, 1950) and *P. sylvestris* (Niemelä *et al.*, 1992) are more infected by *G. abietina* than stands with wider spacing. The density effect might be correlated with shading. Shading is known to decrease carbohydrate content in the buds of conifer species, causing a reduced defence against, for example, pathogens (Read, 1968). However, as stated in Donaubauer (1972b), shade alone cannot be the decisive factor inducing a *G. abietina* epidemic. Another potential factor affecting tree resistance in dense stands is the increasing intraspecific competition (Niemelä *et al.*, 1992). Thus, pines growing in high-density stands may allocate fewer resources to resistance, than for example height growth, and thus become more susceptible to *G. abietina*. However, even though dense stands may have a higher proportion of suppressed trees, more likely to be damaged, they could also have a greater number of recoverable trees after *G. abietina* outbreaks, as shown in Kallio *et al.* (1985).

Management of infected stands

According to present thinking, forest managers can minimize the impact of *G. abietina* by using various silvicultural practices (*e.g.* Skilling, 1988). Early detection of new *G. abietina* infections, by the use of remote sensing and regular forest inventory actions, offers the opportunity to eradicate or greatly decrease the disease by sanitary clear-cuts, thinnings or by pruning of infected branches. Thinning of a diseased stand improves its healthiness by changing the microclimatic conditions and by removing susceptible trees and tree species likely to be re-infected in the future (Uotila & Uitamo, 1993). *Pinus sylvestris* trees severely weakened by *G. abietina* are also likely to be attacked by the bark beetle *Tomicus piniperda* (L) (Kaitera & Jalkanen, 1994a; Sikström *et al.*, 2005). Secondary damage caused by *Tomicus* spp. can be reduced if the stand is thinned during the winter after severe *G. abietina* symptoms have been found (Uotila & Uitamo, 1993). In heavily infected areas, total sanitation may be the only way to control the disease. The use of more disease-resistant planting material may prolong this sanitary effect. How much of the crown that has to remain green to ensure survival of infected trees is essential knowledge for sanitation management and addressed in this thesis. During the 2001–2003 epidemic, no clear guidelines based on *G. abietina* research were available and Swedish forest owners were

given inconsistent recommendations on defoliation limits. In Sweden there is a law stating the minimum timber volume in a forest stand at a given tree height (Anon., 2006). If a thinning would cause the remaining timber volume to fall below that limit, an early clear-cut and regeneration of the stand was recommended.

To control the disease during the last epidemic in Sweden, many middle-aged *P. sylvestris* stands have been sanitary thinned or clear-cut (Wulff & Walheim, 2003). At present, however, there is little knowledge of the risks of leaving *G. abietina*-infected slash on the clear-cuts in Fennoscandia. Current knowledge is primarily derived from pruning-studies of *P. resinosa* in north-eastern USA and eastern Canada, where pruning is used as a silvicultural tool to reduce *G. abietina* infection in pine plantations (French & Silverborg, 1967; Bergdahl & Ward, 1984; Laflamme, 1993a). Both Bergdahl & Ward (1984) and Laflamme (1993a), studying the EU race of *G. abietina*, equivalent to LTT in Sweden, claim that pruned branches do not have to be removed from the plantations to prevent further infection within the pruned stand; According to Laflamme (1993a) this is true if the distance to remaining symptom free branches exceeds 60 cm. French and Silverborg (1967), however, noted re-infection by *G. abietina* (NA race) after pruning *P. resinosa* without slash disposal and suggested follow-up sanitation to remove the inoculum potential from the stand. Accordingly, Donaubauer (1960) and Dorworth (1972b), studying the EU race *G. abietina* on *Pinus nigra* Arnold and the NA race *G. abietina* on *P. resinosa*, respectively, recommended slash removal or prescribed burning to prevent re-infection on remaining trees or planted seedlings. Both authors also suggested that spore production might be enhanced in the moist layer of air forming in the piles of slash left after felling operations. Since these former studies: (a) primarily focused on sanitary pruning and re-infection on branches of pruned trees and not on planted seedlings on clear-cuts, (b) were conducted outside Fennoscandia and on non-Fennoscandian tree-species, and (c) resulted in conflicting conclusions, it was important to carry out new studies relevant to Fennoscandian conditions.

Objectives

All the recent sanitary harvest operations have resulted in great amounts of *Gremmeniella*-infected *P. sylvestris* slash left on the clear-cuts. However, due to the lack of adequate research, there are no general guidelines for regeneration to prevent *G. abietina* re-colonisation of *P. sylvestris* seedlings. Currently in Sweden it is common to wait two to four years between harvest and reforestation to facilitate soil scarification and reduce damage by pine weevil (*Hylobius abietis*). However, this rest period increases competition from surrounding vegetation and reduces stand production. Some forest managers therefore choose to plant during the first growing season after harvesting, often by using insecticides or mechanical protection of the seedlings.

The main objectives in study I and II were to:
(I) investigate survival and vitality of *G. abietina* on *P. sylvestris* slash after clear-cutting severely infected *P. sylvestris* stands in northern Sweden.
(II) investigate *G. abietina* disease incidence on *P. sylvestris* seedlings planted shortly after sanitation felling, with and without removal of infected *P. sylvestris* slash.

During the 2001–2003 epidemic, no clear guidelines for sanitation thinning of *G. abietina*-infected stands were available and recommendations were based on data from studies on pine shoot beetle (*T. piniperda)* attack patterns.

The main objective in study III was to improve the accuracy of predictions of mortality and growth losses in middle-aged *P. sylvestris* forests infected with *G. abietina*. A specific goal was to identify limits of defoliation by *G. abietina* and subsequent attack by *T. piniperda*, beyond which the trees will not survive. In addition, we wanted to study the rate of foliage recovery and quantify losses of diameter increments following different levels of defoliation during a four-year-period.

There is currently an increasing interest in the use of the fast-growing *P. contorta* as an alternative to *P. sylvestris* in Swedish forests. If *P. contorta* would prove more resistant than *P. sylvestris* to LTT *G. abietina*, this could be vital information for risk assessments in future planting programs with *P. contorta* in Sweden.

The main objective in study IV was to examine the resistance of the introduced *P. contorta* var. *latifolia* exposed, under field conditions, to LTT *G. abietina* (EU race) in northern Sweden.

Materials and methods

Paper I

The study of longevity of LTT *G. abietina* in *P. sylvestris* slash was conducted by cutting and debranching infected 40-year-old trees, continuously collecting recently killed shoots with *G. abietina* pycnidia from the slash and performing spore (conidia) germination tests in the lab from the sampled pycnidia. The field experiment was conducted on an east-facing sandy till slope near Vindeln (64°17'N, 19°50'E, 285 m a.s.l.), 55 km northwest of Umeå in northern Sweden.

Seventeen trees with a mean height of 15 m and scattered symptoms of recent *G. abietina* infection in the crowns were selected and left after a clear-cut in September 2003. Approximately once a month between September 2003 and April 2004, 2–3 of the selected trees were cut down and debranched. On the cut branches, shoots with *G. abietina* pycnidia were marked for coming sampling. At regular intervals between September 2003 and October 2004, infected shoots were randomly sampled for vitality tests in the Forest Pathology Lab, SLU, Umeå. Normally at least two infected shoots with pycnidia were sampled from each group of 2–3 trees on each sampling occasion. Furthermore, a small number of *P. sylvestris* shoots with *G. abietina* pycnidia were sampled from slash on three neighbouring clear-cuts on 22–25 May 2004, 13–18 months after sanitation felling.

The vitality test of the collected *G. abietina* pycnidia was done by studying conidial germination capacity on 20% vegetable juice agar incubated at 15°C. The germination percentage of at least 100 conidia from each pycnidium, after 24, 48 and 72 hours' incubation, was estimated through a light microscope. Spores were regarded as germinated when showing visible hyphae (Fig. 4).

Fig. 4. Germinating *G. abietina* conidia after 72h incubation on Granini vegetable juice agar in 15° C. Photo: A. Bernhold.

In the statistical analysis, three factors were tested against conidial germination capacity at 24, 48 and 72 hours' incubation, respectively: (i) Time of felling, (ii) time of sample collection and (iii) time on ground ("slash age"). Analysis of variance and Tukey-tests as well as linear regression analysis, were performed to statistically test for the effects of these variables.

Paper II.

The infection risk on *P. sylvestris* seedlings planted after sanitary clear-cuts with or without cleaning of LTT *G. abietina*-infected slash was studied in three middle-aged *P. sylvestris* stands in northern Sweden, all severely infected in 2000–2001. In the early summer of 2003, two to eight months after the infected stands had been cut and debranched, one-year-old containerised *P. sylvestris* seedlings were planted on the sites. In a randomised block design, each site was divided into two plots; one with (treated) and one without (control) slash removal. Four subplots, each with 25 *P. sylvestris* seedlings, were randomly placed within the treated and control plots, the total number of seedlings reaching 600. The infected slash was piled in rows on all four sides of the treated plots. The occurrence of different inoculum sources surrounding the indicator seedlings was assessed in late May 2004 as follows: (i) coverage of *P. sylvestris* slash in 5x5 and 10x10 squares surrounding control subplots; (ii) distance between subplots and surrounding piles of infected slash on treated plots; and (iii) distance between all subplots and the closest stand of infected *P. sylvestris*. To control for snow cover as a major factor influencing disease incidence, snow depth was measured on all subplots in the spring of 2005.

The seedlings were checked for symptoms of *G. abietina* on two occasions one year after planting; once in June–July, to detect early symptoms, and once in October–November, when pycnidia had developed. Seedlings with visible *G. abietina* pycnidia on dead shoots and cankers were classified as infected. Dead seedlings with pycnidia were classified as *G. abietina*-induced mortality, while all dead seedlings were counted in total mortality.

The effect of slash cleaning was tested using analysis of variance performed by block (site) and the effect of different infection sources (i–iii above) was tested using covariance analysis. The dependent variables tested were infection rate (with and without stem cankers) and mortality rate (total and *G. abietina*-induced).

Paper III.

Tree mortality, diameter increment loss and foliage recovery was studied in four 2–4 ha stands of 35- to 40-year-old *P. sylvestris* following defoliation by LTT *G. abietina* and subsequent attack by *T. piniperda* during the outbreak in 2001–2003 and two consecutive years. The four stands had been commercially thinned in 1999 and 2000 and were sparsely stocked. Five to ten circular plots with radii of 10 m were laid out systematically along transects in the plantations, resulting in

86–223 sample trees, in total, per site. Within each circular plot the degree of crown defoliation of each tree, *i.e.* the percentage of needles affected by *G. abietina*, was assessed, together with the condition of the top shoot and the occurrence of stem-boring insects (predominantly *Tomicus* spp.). In addition, each tree's diameter at breast height, and basal area and dominant height for each circular plot, were measured.

The significance of between-site and between-year differences in levels of defoliation was tested using ANOVA (GLM) and Tukey's test of comparisons. For each tree, defoliation each year in 2001–2004 was related to mortality one year after defoliation assessments. Colonisation by *T. piniperda* and the mortality of top-shoots were related to defoliation in the year of, and one year prior to, the time of damage. The relationship between mean defoliation and the factors tree size (diameter) and diameter increment were analysed using linear regression. Furthermore, the relationship between increment and site was tested using multiple regression with the sites as dummy variables. To visualise the reduction in increment due to needle defoliation an index was constructed (Fig 14), in which the increment of trees with very little or no defoliation (0–19%) was used as reference.

Paper IV.

Field experiments

To test the resistance of *P. contorta* to LTT *G. abietina*, indicator seedlings were planted in gaps in a severely infected 40-year-old *P. sylvestris* stand following the 2001–2003 *G. abietina* epidemic. The study was conducted in Kulbäcksliden Experimental Forest near Vindeln (64°09'N, 19°34'E, 300 m a.s.l.), 60 km northwest of Umeå in northern Sweden. In each of the three 15x15 m gaps, 100 one-year-old seedlings were planted on 23 June 2005; 33–34 seedlings each of two provenances *P. contorta* (C 703 and C 704) and one provenance of *P. sylvestris* (T 406) as control. The *P. contorta* seedlings originated from seed orchards in Sweden using Canadian pine clones from the following latitude ranges: 57°60'– 60°90'N (C703) and 54°40'– 58°80'N (C 704). The *P. sylvestris* seedlings were taken from a local seed orchard, using pine clones from high altitude areas of northern Sweden (lat. 64°00'– 65°30'). Each seedling was checked for symptoms of *G. abietina* in autumn two consecutive years after planting. On infected seedlings, lengths and proportions of infected tissue (tip/shoot blight) and the production rate of new leader shoots were additionally assessed. Uncertain field identifications were confirmed by laboratory diagnostics. During the two years of observations, only anamorph fructifications (pycnidia) were found on the seedlings.

Biotype identification

To confirm LTT *G. abietina* of the European race as the damaging agent, infected needles of 14 seedlings (ten *P. contorta* and four *P. sylvestris*) were sampled for race and type identification. DNA extraction directly from infected needles,

followed by PCR amplification and Msp I digestion of the ITS of the ribosomal DNA (Hamelin *et al.* 2000) allowed the detection of *G. abietina* of the European race on all samples. Nine of the samples (five *P. contorta* and four *P. sylvestris)* were tested and identified as LTT *G. abietina* using STS markers and low-ionic-strength single-strand conformation polymorphis (LIS-SSCP) (Dusabenyagasani 1998). Race and biotype identification was performed at the Laurentian Forestry Centre (LFC) (of the Canadian Forest Service) in Quebec, Canada.

Processing for microscopy

Shoot samples for microscopic analyses of defence reactions were collected in late August 2007. The samples were cut transversally from the transition zone of the infected shoots and fixed in 4% paraformaldehyde in 0.1M cacodylate buffer (pH 7.2) for one week during transport from Sweden to LFC in Quebec. Tissue from 10 infected samples (four *P. contorta* and six *P. sylvestris*) were prepared and analysed in microscopy using two different methods: (i) Epoxy sections: Fixed samples were post-fixed in 1% osmium tetroxide, dehydrated in an ethanol series and embedded in Jembed 812 according to standard procedures. Serial sections, between 1 and 3µm, were obtained with an Ultracut E microtome, stained with a commercial Epoxy Tissue Stain (toluidine blue & basic fuchin) and examined with an Orthoplan light microscope. (ii) Cryostat sections: Fixed samples were rinsed with cacodylate buffer, embedded in Tissue-Tek O.C.T. compound and frozen at -20ºC. Serial sections (30 µm each) were then cut with a Histostat cryomicrotome, stained with phloroglucinol-HCl and used for histochemical tests in a light microscope. Lignin (red) was observed under brightfield light and suberin (blue) was observed under violet light excitation. Additionally, two symptom-free samples (one *P. contorta* and one *P. sylvestris*) were processed according to both methods and used as controls.

Data analysis

All statistical analyses of between-species and between-provenience differences in relation to disease incidence (mortality rate, infection rate, infection length) were performed by ANOVA (GLM), using a randomised block design. A separate ANCOVA (GLM), including "2006 shoot length" as covariate, was used for the 2006 dataset due to the significant correlation ($p<0.01$; $r=0.49$) between the top shoot length and the infection length on the seedlings infected in 2006. To further visualise the differences in disease severity between tree species, infected seedlings were classified in four infection classes: tip blight (<15 mm), shoot blight <50% (>15 mm, <50% of seedling size), shoot blight >50% (>50% of seedling size) and dead (killed by *G. abietina*), and displayed in a diagram.

Results and discussion

Survival and vitality of *G. abietina* in *P. sylvestris* slash

It is shown in paper I that LTT *G. abietina* can survive and reproduce through pycnidia in up to 18-month-old *P. sylvestris* slash in northern Sweden. The vitality of *G. abietina* conidia in the slash remained high the whole period from the felling of the sample trees, starting in August 2003, until the release of conidia in the summer of 2004, regardless of the time on the ground (Fig. 5). In fact, conidial germination capacity in the lab was as high in pycnidia collected in 13- to 18-months-old slash as in pycnidia collected in fresh slash at the time of felling (Fig. 5). Mean germination percentage of 265 pycnidia, sampled from the same clear-cut in 0- to 13-months-old slash after 24h, 48h and 72h incubation, respectively, was 10%, 90% and 98%. The regression analysis showed no significant decline in germination capacity by number of months the slash had been on the ground. In fact, the lowest germination percentages were found after 4–5 months on the ground while the highest were found after 6–7 months on the ground (Fig. 5).

Survival and vitality of *G. abietina* in *P. sylvestris* slash has previously not been studied in Fennoscandia. In Ontario, Canada, Dorworth (1972b) found the North American race of *G. abietina* could survive for at least 10 months in *P. resinosa* slash. However, since apothecia are believed to be more persistent than pycnidia on dead shoots and branches on living pines (Roll-Hansen, 1964; Hellgren & Barklund, 1992), it was unexpected to see the high vitality of conidia in slash over one year old.

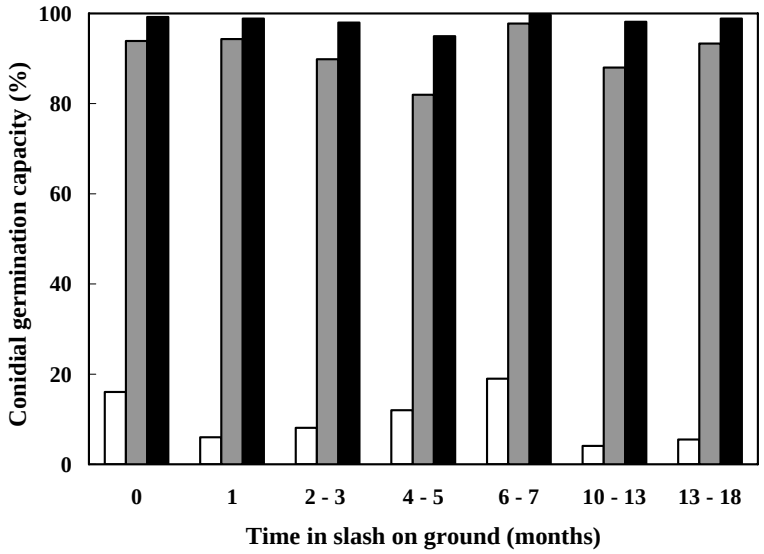

Fig. 5. Germination capacity of LTT *G. abietina* conidia after 24 (white), 48 (grey) and 72 (black) h incubation in each of seven age classes of *P. sylvestris* slash. Samples from 13- to 18-months-old slash were collected at three additional sites in the same region as the main site.

There is reason to believe that *G. abietina*-infected slash in areas with a higher proportion of sexual reproduction may cause even more damage to pine seedlings after sanitation cuttings. Dorworth (1972b) also reports of apothecia production in the *P. resinosa* slash. In the current study, the findings of fresh pycnidia in slash in fall, several months after the time of conidial sporulation, indicate that also pycnidia may be produced on dead pine branches. This hypothesis was, however, only based on general observations and not specifically tested. Production of pycnidia in *P. sylvestris* slash could explain the high vitality of the pycnidia found in over one-year-old *P. sylvestris* slash. Pycnidia with ripe conidia were also found in late May 2004 in the piles of slash on the three sites in study II, 13–18 months after clear-cutting. These pycnidia were probably produced in the slash during summer and fall of 2003. If they had been produced before the stands were cut in the winter and spring of 2002/2003, they would most likely have released their spores already in the summer of 2003.

Since germination capacity of *G. abietina* conidia in the slash remained high regardless of the month of felling of the sample trees, it is not possible to recommend a time of year for harvesting infected stands to minimize survival and vitality of *G. abietina* in the slash.

Conidial germination capacity is often used to analyse the vitality of *G. abietina* isolates (*e.g.* Petäistö, 1993; Hansson, 1998; Uotila *et al.*, 2000). It is, however, important to recognise the effects of variation in incubation environment, growth medium and isolate origin. The isolates of September 2003, collected at the time of felling, were additionally incubated in water agar as comparison. The mean germination percentage after 48h and 72h, respectively, was 39% and 62%, compared to 98% and 100% for vegetable juice agar. Compared to previous germination tests on water agar, this germination capacity was low. In Petäistö (1993) and Hansson (1998), over 90% of the conidia germinated after 48h incubation on water agar. However, in both these tests *in vitro* cultivated conidia was used.

One reason for the variation in germination capacity can be traced to the initial state of conidial germination in the lab. During the three-day germination period on vegetable juice agar, some spores showed a tendency for abnormal growth. Instead of producing long-celled hyphae the cells multiplied and swelled so that each spore took the form of a chain of sphere-shaped cells. This phenomenon is illustrated in Lagerberg (1912), and described as a result of a surplus of nutrients. Most of these abnormal cells were, however, found in the large aggregations of conidia excluded from the germination study due to overlapping spores and poor visibility. Thus, the effect of the abnormal cells in somewhat reducing the germination percentage was rather low.

Effect of slash removal on disease incidence in infected *P. sylvestris* plantations

Replanting LTT *G. abietina*-infected *P. sylvestris* stands within one year after a sanitary clear-cut may severely damage or kill the planted *P. sylvestris* seedlings. In study II, conducted in Västerbotten, *G. abietina* pycnidia were found on 32% of the control seedlings and total infection, including stem cankers, reached 44%, just one year after planting (Fig. 6). Furthermore, total and *G. abietina*-induced mortality was 15 and 10%, respectively. However, infection rates of 50–90% were at the same time reported from one-year-old *P. sylvestris* plantations in Dalarna (Stenström, 2004), the most heavily infected region in Sweden. Thus, in these stands, the effect of a sanitary clear-cut is greatly reduced, unless planting is delayed or careful slash-cleaning is conducted. In study II, the removal and piling of infected slash reduced, in relation to control, the number of infected seedlings by 50% and seedling mortality by 27% one year after planting (Fig. 6).

These findings are not consistent with Bergdahl & Ward (1984), who studied conidial infection levels of *G. abietina* of the EU race (=LTT) on pruned *P. resinosa* in Vermont, USA. They found no effect of slash cleaning and as little as 1% infection on the *P. resinosa* seedlings in the pruning slash, compared to 45% in the unpruned control. The studies of Bergdahl & Ward (1984) leads to the conclusion that slash infected with LTT *G. abietina* only causes minor damage to *Pinus* spp. seedlings. Our results, however, show that this is not the case in *P. sylvestris* plantations of northern Sweden. In northern Sweden, slash cleaning may additionally have a sanitary effect with respect to snow blight (*Phacidium infestans* Karst.) infection, reducing the vegetative spread of mycelia between *P. sylvestris* seedlings in the snow (Hansson, 2006).

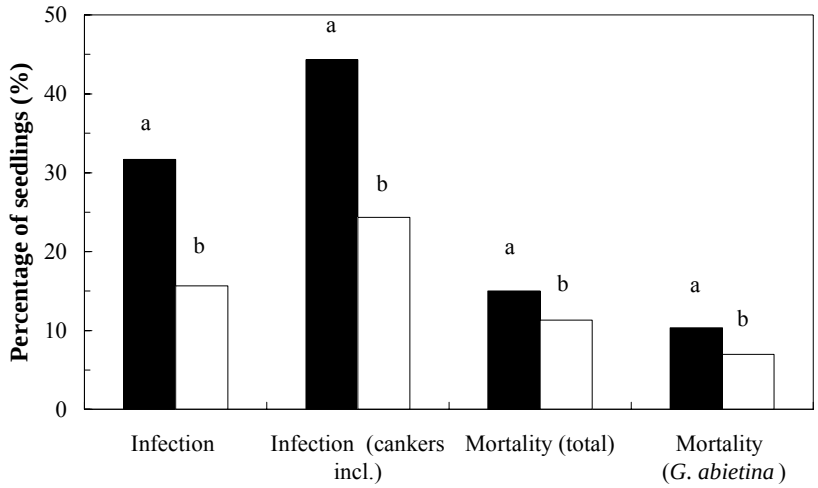

Fig. 6. Gremmeniella abietina (LTT) infection rate (with and without including stem cankers) and mortality (total and *G. abietina*-induced) of *P. sylvestris* seedlings one year after planting. Significant difference ($p<0.05$) was found between control plots (■) and slash-cleaned plots (□).

Furthermore, there is an increasing market for slash as a bio-energy source in Sweden. In 2006, 18.5% of the energy produced in Sweden derived from biofuels (Anon., 2007b).

Gremmeniella abietina-induced stem cankers on pine seedlings have not been properly studied yet, but were shown to be common on young *P. sylvestris* seedlings in study II. The 5–25 mm long cankers were mainly found on one-year-old green shoots and on some cankers, mainly larger ones, *G. abietina* pycnidia had developed (Fig. 7). In total, stem cankers were found on 15% of the seedlings. On one site, however, stem cankers were found on as much as 40% and 21% of the seedlings on the control and slash-cleaned plots, respectively. Frequent canker formation may be the effect of high seedling vitality and good resistance to pathogen colonisation (Manion, 1991). Consistent with this, the site with most cankers also had the lowest seedling mortality. A parallel can be drawn to infection studies of STT *G. abietina* on *P. sylvestris* and *P. contorta*. Witzell and Karlman (2000) found a higher infection rate but a lower mortality rate on *P. contorta* saplings compared to *P. sylvestris* saplings, and subsequent stem cankers were frequent on the surviving *P. contorta* trees.

A significant correlation ($p<0.05$, $R^2>0.72$) between infection rate and amount of infected slash on control plots, within both 5x5 m and 10x10 m squares, was found (II). This relation indicates that clear-cuts with large amounts of infected slash should be given priority for slash cleaning and that total slash sanitation is not always needed to reduce seedling infection. Neither the distance to closest pine stand nor any of the variables on distance to surrounding piles of infected slash on slash-cleaned plots had any effect on seedling disease incidence (II). However, the effects of neighbouring stands on disease incidence need to be further investigated without influx from infected slash on the study site.

Fig. 7. Stem canker with LTT *G. abietina* pycnidia on one-year-old green shoot of a *P. sylvestris* seedling. Picture taken in Nov 2004, one year after planting. Photo: A. Bernhold.

A prolonged snow cover reduces the growing season and allows pathogens as *G. abietina* and *P. infestans* to grow for a longer time in conducive moist conditions (Marosy *et al.*, 1989; Roll-Hansen, 1989). The significantly higher seedling infection and mortality on the control plots could, however, not be related to a longer lasting snow cover. On site 1 and 2, the snow cover was equally deep on the control and cleaned plots and on site 3, the snow cover was twice as deep on the cleaned plot as on the control plot in late April 2005.

Since it is common practice in Sweden today to wait 2–4 years between harvest and reforestation, even in healthy stands, re-infection of LTT *G. abietina* on planted *P. sylvestris* seedlings from remaining slash may be a minor problem. However, the method of planting in green slash, or otherwise planting within two years after harvesting, should be avoided if *G. abietina* infection is present in the regeneration area. Prior to the latest *G. abietina* epidemic of 2001–2003, mainly *young* pine plantations in harsh climatic areas were infected in northern Sweden (Karlman, 1986; Karlman *et al.*, 1994). During the latest outbreak, however, a large part of the damaged *P. sylvestris* stands were 30–60 years old, resulting in large quantities of infected slash remaining on the sanitary cleared areas. Slash cleaning may reduce the infection rate up to 50% even if the slash is still left in piles at the site (II). Even so, *G. abietina* may survive for one and a half years in slash (I), and since such piles of slash may provide a favourable moist and shady environment for the pathogen, it is recommended to prioritise these sanitary cut stands when gathering slash for bio-energy and, if possible, process the slash into chips at the site before transport. Another possibility is to use prescribed burning on these infected regeneration areas; a regeneration method that has increased in Sweden recently due to its conservation benefits as recognised by FSC certification policy. The effect of burning of infected slash on disease incidence was originally meant to be measured in study II but could not be performed due to unfavourable weather conditions for burning at the study sites.

Due to the sporulation capacity of *G. abietina* in *P. sylvestris* slash and the risk of seedling infection and mortality, both one and two years after sanitary clear-cuts (I, II), it is recommended to wait at least two years before regenerating *G. abietina*-infected stands with *P. sylvestris.* Thus, the method of planting in green slash should be avoided in these stands. However, if the stand is regenerated already the first vegetation period after a sanitary clear-cut, removal of infected slash may reduce infection with up to 50% (II).

Resistance to LTT *G. abietina* in *P. contorta* and *P. sylvestris*

In the study of natural infection by LTT *G. abietina* (IV), the proportion of infected seedlings was higher in *P. contorta* (45%) than in *P. sylvestris* (32%). However, the infected *P. contorta* seedlings proved to be more resistant in terms of higher survival rates, shorter lengths of shoot blight (Fig. 8) and quicker recovery by production of new leaders.

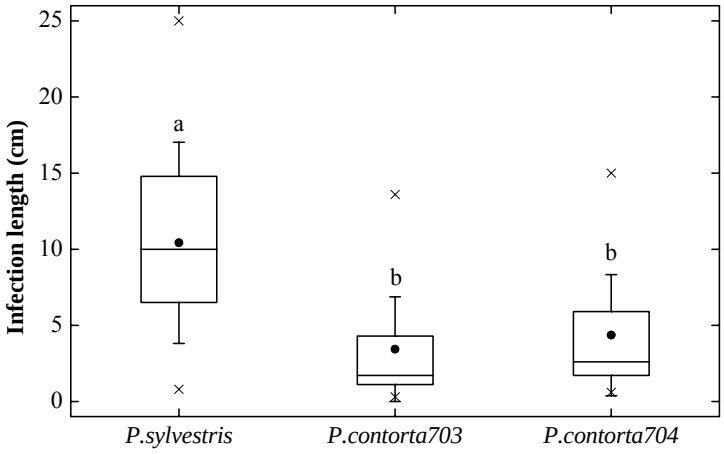

Fig. 8. Length of LTT *G. abietina*-infected tissue in relation to tree species and *P. contorta* provenances. Standard deviation (error bars), max/min values (x), interquartile range boxes and arithmetic mean (black dots) values are displayed. Statistically significant differences between mean values ($p<0.05$) are shown by different letters.

This is in accordance with Aitken (1993), who found *P. contorta* to be less affected by *G. abietina* compared to both *P. sylvestris* and *P. abies* in Britain. On most *P. contorta* seedlings in study IV, the damage was restricted to shoot blight on the current year shoots and a majority of the infected seedlings showed signs of recovery after 1–2 years. Similar results were found in recent studies in Quebec, Canada, where *P. contorta*, and to an even larger extent *P. banksiana*, were found highly resistant to the EU race of *G. abietina* (=LTT) (Laflamme *et al.*, 2000; Laflamme *et al.*, 2006). In Laflamme *et al.* (2006), gaps in a severely infected, 25-year-old *P. resinosa* stand were planted with *P. resinosa, P. banksiana* and *P. contorta*. After five years, all the *P. resinosa* seedlings were killed, compared to 19% of the *P. contorta* and only 1% of the *P. banksiana* seedlings. Prior to these studies, *P. contorta* was regarded as susceptible to the EU race of *G. abietina* in North America (*cf.* Skilling & Riemenschneider, 1984) and there was a debate regarding the possibilities of a large scale spread of the pathogen into the *P. contorta* forests of western Canada (Dorworth & Muir, 1993). The risk scenarios presented in Dorworth and Muir (1993) were, however, partly based on experiences from the *G. abietina* outbreak on *P. contorta* in northern Sweden at the time. It is now evident that the damage to the Swedish *P. contorta* plantations in the 1980s was caused by STT *G. abietina* and not LTT, which is the type that is introduced to North America (Hamelin *et al.*, 1996). Even if these two types are currently regarded as the same race of *G. abietina,* they clearly differ genetically and in their epidemiology. Only STT has caused any significant damage to *P. contorta* in Sweden and thus there are no reasons to predict a major outbreak of the European race in North America based on experiences from Sweden. This example highlights the importance of taxonomic knowledge in pest management and the need for thorough resistance studies, including monitoring of disease

severity and recovery, as complements to screenings of possible host species. Thus, according to current knowledge, *P. contorta* seems to have a well developed resistance to LTT *G. abietina* even though shoot blight may occur in infected plantations.

Regarding STT *G. abietina*, however, several field trials (Hansson & Karlman, 1997; Witzell & Karlman, 2000) and large-scale monitoring of pine plantations in northern Sweden (Karlman *et al.*, 1994) found *P. contorta* to be more susceptible than *P. sylvestris* in terms of higher infection and mortality rates. Interestingly, though, an inoculation trial by Hansson (1998), found a higher resistance in *P. contorta* also to STT *G. abietina* and concluded that the two species probably have the same "genuine" susceptibility to STT *G. abietina*, although the introduced *P. contorta* is more affected by predisposing stress factors as extreme weather conditions. Accordingly, the severe outbreak of STT *G. abietina* in northern Sweden in the late 1980s occurred after a period of extremely cool and rainy summers followed by a mild winter with extreme snow loads (Karlman *et al.*, 1994).

Histopathological examinations of infected shoots in study IV showed that *P. contorta* from Swedish breeding programs produce similar defence reactions in infected shoots (Fig. 9) as showed on *P. contorta* in Quebec, Canada (Laflamme *et al.*, 2006). Infections in *P. contorta* was shown to be limited to the outermost parts of the shoots by the production of ligno-suberised barrier walls extending from healthy needle bases transversally across the shoots and, thus, prevented major dieback. Interestingly, however, we found traces of ligno-suberised barriers also in the transition zone of infected *P. sylvestris* shoots (Fig. 9). It is reasonable to assume that this active defence of *P. contorta* and *P. sylvestris* against LTT *G. abietina* constrains the infection to the youngest shoots and prevents the pathogen from reaching larger branches or the stem, where it could cause lethal cankers, as often seen on *P. resinosa* infected with LTT *G. abietina* in Canada (Laflamme, 1991). However, during a severe outbreak, such as the one in Sweden in 2001–2003, significant defoliation may cause mortality of pole-sized *P. sylvestris* without strangulation of the stem (III, Sikström *et al.* 2005).

A general conclusion based on the results of study IV and of other works on resistance (Skilling & Riemenschneider, 1984; Simard *et al.*, 2001; Laflamme *et al.*, 2006) is that there seems to be a gradient of resistance among the following commercially-important, two-needled pine species: *Pinus banksiana* seems to be most resistant, followed by *P. contorta, P. sylvestris* and finally the highly susceptible *P. resinosa*. An interesting conceptual approach was put forth by Wu *et al.* (1996). They found that the closer a *P. contorta* provenance was to the edge of the natural *P. banksiana* distribution, the higher was its resistance to pests like western gall rust (*Endocronartium harknessii* (J.P. Moore) Y. Hiratsuka), stalactiform blister rust (*Cronartium coleosporioides* Arth.) and needle cast (*Lophodermella concolor* (Dearn.) Darker).

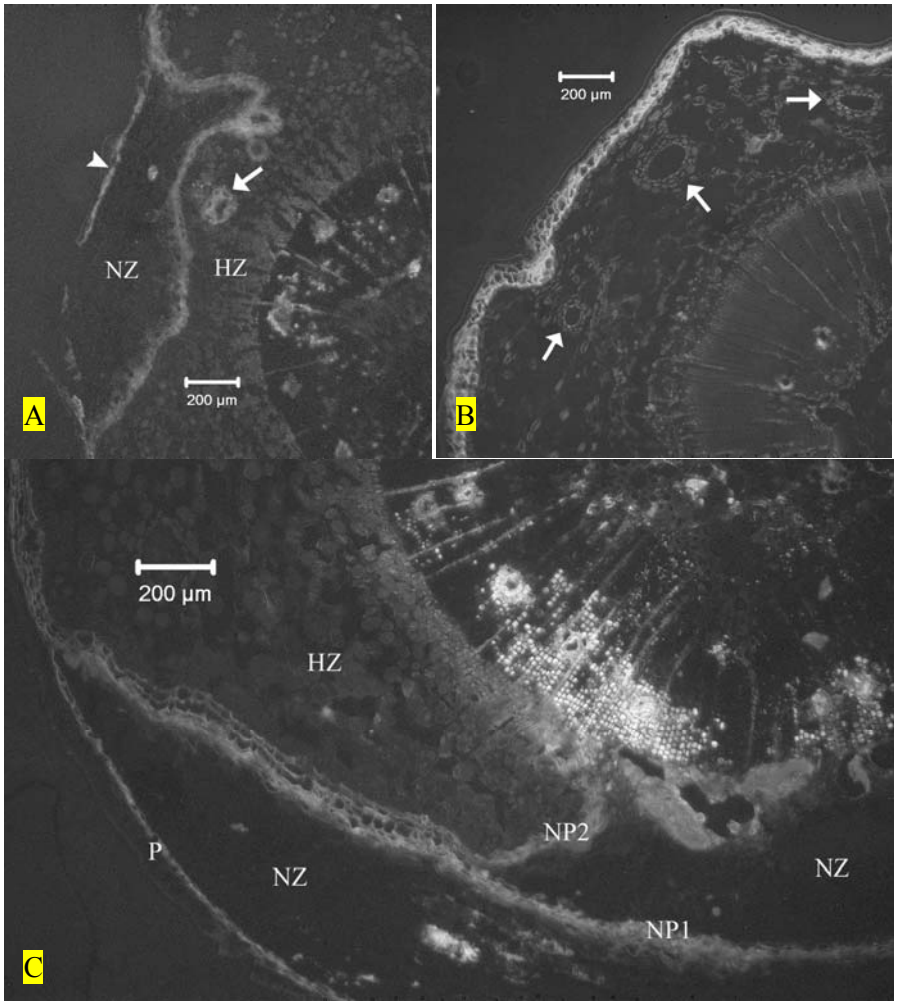

Fig. 9. Cross-sections from the transitional zones of LTT *G. abietina*-infected pine shoots, stained with phloroglucinol-HCl and observed under violet light excitation.

A. Infected *P. contorta* shoot. A necrophylactic periderm has formed as a barrier between the necrotic zone (NZ) and the healthy zone (HZ). Fluorescence is also seen around resin canals (arrow), in infected xylem tracheids and in the broken periderm (arrowhead).

B. Healthy *P. contorta* shoot. Fluorescence is seen in the well-defined periderm and in the xylem resin canals. Suberin is *not* observed in xylem tracheids or in cortex resin canals (arrows).

C. Infected *P. sylvestris* shoot. Three suberised barriers are seen: the initial periderm (P), the first necrophylactic periderm (NP1), separating the two areas of necrotic zone (NZ), and the second necrophylactic periderm (NP2), separating the necrotic and healthy zones (HZ). Fluorescence is also seen around resin canals and in infected xylem tracheids, though in the latter it was also partly the result of phenols that filled these cells.

Wu *et al.* (1996) hypothesised that *P. banksiana* introgression may have played an important role in the evolution of defence towards pests in *P. contorta*. An interesting comparison is that Hansson (1998) found that seedlings of the most south-eastern *P. contorta* provenance, close to the western distribution limit of *P. banksiana*, showed lower frequencies of STT *G. abietina* damage compared to the most north-western provenance. Even though there has been no host-pathogen co-evolution between *P. contorta* and the European race of *G. abietina*, it would be interesting to investigate a possible effect of *P. banksiana* introgression on the *P. contorta* resistance to LTT *G. abietina*.

In study IV, 47% of the *P. contorta* seedlings that survived infection had developed a new leader shoot in 2007, compared to 19% of the infected *P. sylvestris* seedlings. This is in accordance with Hansson (1998) and may be explained by a combination of the confirmed higher tolerance to physical damage and the shorter length of shoot infection found in the *P. contorta* seedlings. The mean infection length on surviving seedlings was 3.9 cm for *P. contorta* compared to 10.4 cm for *P. sylvestris*, corresponding to 23% and 49%, respectively, of the total seedling length (Fig. 8). Thus, the pathogen rarely managed to spread past the annual shoots in *P. contorta* seedlings, enabling the seedlings to swiftly produce new leaders from lateral shoots or sprouting dormant buds. On *P. sylvestris* seedlings, however, the pathogen more often spread to older shoots and thus reduced the possibility of fast recovery. One hypothesis is that this shorter length of shoot infection enables the *P. contorta* to retain a large proportion of green needles in the crown even though a large part of the current year shoots are infected. A large-scale infection trial with LTT *G. abietina* on pole-size *P. contorta* is needed to test this hypothesis.

As mentioned earlier, susceptibility of *P. contorta* to *G. abietina* has a significant correlation with latitude of seed origin and northern provenances have proven more resistant (Karlman, 1984; Dietrichson & Solheim, 1987). In the current study, however, we found no significant differences in disease incidence or mortality between the two provenances of *P. contorta*. These two provenances are currently being used in plantation forestry in northern Sweden (Västerbotten and Västernorrland) and originate from Swedish seed orchards with Canadian pine clones from British Colombia (57°60'– 60°90'N (C703) and 54°40'– 58°80'N (C 704)). Earlier studies on transfer of *P. contorta* provenances indicated that a 3–10° (Hagner & Fahlroth, 1974) or 2–5° (Lindgren *et al.*, 1988) northern transfer into Sweden seemed optimal. However, a direct comparison of the provenance of imported seed and seed from Swedish orchards cannot be made due to the improved genetic and physiological qualities in the selected seed trees in the Swedish seed orchards (Tore Ericsson pers. comm., Jan 2008). Even so, it is reasonable to assume that hardier *P. contorta* provenances, *e.g.* from Yukon territory (>60° N), would prove even more resistant to *G. abietina* on the location of study IV (64°09'N, 19°34'E, 300 m a.s.l.) (*cf.* Hansson, 1998).

It is becoming more and more evident that a high forest production is beneficial not only for the pulp and paper industry but also for the increase in uptake of atmospheric carbon to reduce global warming (IPCC, 2007). Accordingly, there is

currently an increasing interest in the use of fast-growing exotic tree species, as *P. contorta*, in Swedish forestry. There is, however, also a restriction in the use of exotic species, partly due to the risk of pest epidemics. The aggressive outbreak of STT *G. abietina* on *P. contorta* plantations in northern Sweden in the 1980s contributed to these restrictions (in area and geographical locations of *P. contorta* plantations) in the Swedish Forestry Act (Karlman, 1987, 2001). Infections were most severe in areas with low temperature sums and long lasting snow covers, previously covered with spruce (Karlman *et al.*, 1994). If we want to increase the use of *P. contorta* in Swedish forests it is important to identify areas with low risk of disease outbreak along with a proper choice of plant material. However, since current research indicates that *P. contorta* is so far more resistant than *P. sylvestris* to LTT *G. abietina* (IV) and several other severe pathogens in Sweden, *P. contorta* is, from a pathological point of view, an interesting alternative to *P. sylvestris* on sites suitable for pine, and especially in regions with extensive damage by LTT *G. abietina*.

Aspects of *P. contorta* as an alternative to *P. sylvestris* in Sweden

There are several reasons why a well developed resistance in *P. contorta* to LTT *G. abietina* (IV) would be beneficial for Swedish forestry.

Firstly, LTT *G. abietina* caused the most severe outbreak of a shoot disease ever recorded in Sweden (480,000 ha middle-aged *P. sylvestris* was infected in 2001–2003) and similar outbreaks cannot be excluded in the future. Previously, *G. abietina* on pole-sized *P. sylvestris* were mainly found in southern and central Sweden, but during this latest epidemic severe damage was found as far north as central Norrland. It is not unlikely that a change in the climate is contributing to this increase in disease incidence in the north. *Gremmeniella* is favoured by cool and rainy summers and mild winters, and a climatic change, with higher summer precipitation and winter temperatures (Kjellström *et al.*, 2005), is likely to favour the development of the disease in the north. However, diseases that are linked to a thick and long-lasting snow cover, especially snow blight (*P. infestans*) but to some extent also STT *G. abietina*, may become less frequent with an increase in winter temperatures (Burdon *et al.*, 2006). Thus, warmer temperatures may reduce the overall severity of damage on young *P. contorta* plantations in northern Sweden in the future. However, extreme snow falls are likely to become more frequent (Kjellström *et al.*, 2005) and since heavy wet snow is predisposing *P. contorta* for STT *G. abietina* infection, more conducive conditions for disease development may occur locally in northern Sweden.

Secondly, the use of *P. contorta* is likely to increase rapidly in Sweden in the near future due to the increased demand for fast-growing forests to satisfy the timber demand of industry, and at the same time increase the uptake of atmospheric carbon. *Pinus contorta* is estimated to produce 36% more wood than *P. sylvestris* and shows a higher survival at young age, even though it is less stable against wind and snow when planted (Elfving *et al.*, 2001). Despite the severe outbreak of STT *G. abietina* on young *P. contorta* in the late 1980s, the

introduced *P. contorta* has so far proven more resistant to fungal pathogens in Fennoscandia, compared to the native *P. sylvestris* (Roll-Hansen, 1978; Karlman, 1986; Kaitera & Nuorteva, 2008). For example, *P. contorta* is not damaged by the two major rust fungi on *P. sylvestris* in Fennoscandia: resin top disease (*Cronartium flaccidum* (Alb. & Schwein) G. Winter and *Peridermium pini* (Pers.) Lév.) and pine twisting rust (*Melampsora pinitorqua* (Braun) Rostr.). Both of these diseases are caused by biotrophic fungi that are likely to be favoured by a future increase in foliar nitrogen and tree production levels along with an extension of the growing season (Mattila *et al.*, 2001; Burdon *et al.*, 2006). A recent Finnish inoculation study confirms the resistance of *P. contorta* to both *C. flaccidum* and *P. pini*, and recommends *P. contorta* as an alternative to *P. sylvestris* for cultivation on disease-prone areas in northern Fennoscandia (Kaitera & Nuorteva, 2008). Furthermore, mortality by snow blight (*P. infestans*) has proven less severe in *P. contorta* regenerations compared to *P. sylvestris* due to faster growth during the early stages, enabling the seedlings to more quickly reach above the snow cover (Karlman, 1986; Roll-Hansen *et al.*, 1992; Elfving & Norgren, 1993). This, along with greater frost hardiness and a lower predisposition to browsing by moose are, according to Elfving *et al.* 2001, the most important reasons for the higher survival of *P. contorta*, compared to *P. sylvestris*. The rapid height growth of young *P. contorta* trees should also be beneficial for the resistance to STT *G. abietina*. However, unstable trees, snow bending and vole damage has proven to be a large problem in the early *P. contorta* plantations in northern Sweden, partly due to poor seedling quality and site adaptation (Karlman *et al.*, 1994). Even though potential diseases may yet be discovered during the later stages of the *P. contorta* rotation, there is currently no major fungal shoot pathogen on pole-sized *P. contorta*. With a careful regeneration management, including the choice of proper sites and hardy plant material from Swedish breeding programmes, subjected to screening for pathogens, it is likely that the damage in the young *P. contorta* plantations will be less severe during future STT *G. abietina* outbreaks.

It is, however, of great importance to consider the long-term risks involved with the introduction of an exotic tree species into a novel environment with pathogens not previously encountered (Karlman, 1981; von Weissenberg, 1982). This is of special concern when there is a close taxonomic relationship between the exotic and native species, as in the case of *P. sylvestris* and *P. contorta* (Ennos, 2001). In von Weissenberg (1982), it is recommended that 1–2 rotation periods, including thorough investigations of plant material of different heritage and age-class, should precede a large-scale cultivation of exotic tree species. As stressed by Karlman (1986) and Ennos (2001), the greatest risk with the introduction of *P. contorta* to Fennoscandia, is the possibility of a transmission of its native pathogens from western North America to the Swedish *P. sylvestris* stands. As mentioned in Ennos (2001), experience has shown that the introduction of an exotic species on a large scale is often followed by a later introduction of its own pathogens. A well known example is the arrival of the needle cast pathogen *Dothistroma pini* Hulbary in exotic *Pinus radiata* D. Don. plantations, about 30–40 years after plantation establishment. However, all *P. contorta* seedlings planted in Sweden today are produced in Swedish nurseries, and most accidental imports

of exotic pathogens occur from plant transportations between continents. Even so, it is important to continue the evaluation of pathogenicity of *P. contorta* pathogens on native *P. sylvestris* that was initiated in 1986 as a joint Swedish-Canadian research project (Karlman *et al.*, 1997). In Karlman *et al.* (1997), some of the rust fungi on *P. contorta* in western Canada, *e.g. E. harknessii*, were found capable of also infecting *P. sylvestris*. Although these rusts were not as virulent on *P. sylvestris* as on *P. contorta* in the cited study, it is of utmost importance to prevent the introduction of these exotic pathogens into Sweden and at the same time promote a rapid development of more resistant Swedish "land races" of *P. contorta*. Another potential risk of introducing *P. contorta* to Sweden is that the native pathogens might become more virulent on *P. contorta* and then spread back and cause severe damage to *P. sylvestris* (Ennos, 2001; Karlman, 2001). According to Ennos (2001), however, the risk of evolving more aggressive pathogens is not higher on introduced *P. contorta* than it is on native *P. sylvestris* growing under stressed conditions.

In terms of future research on resistance of *P. contorta* and *P. sylvestris* to *G. abietina* it would be interesting to perform a study on larger trees and include both STT and LTT, as well as the importance of snow cover, for development of the disease. Such a study was originally included in paper IV. Two sites with 10- to 15-year-old *P. contorta* and *P. sylvestris* saplings and with the lower branch whorls covered by snow in winter were chosen for the study. Branches on the lowest 2–3 whorls were fixed at a height of 20–30 cm above ground to secure a long lasting snow cover in winter. On both sites pine shoots above and below the snow cover limit was inoculated with both STT and LTT *G. abietina* (EU race) in the summer of 2006. The main hypotheses would be to confirm if *P. contorta* is more resistant to LTT *G. abietina* compared to *P. sylvestris* also on larger trees. Additionally, it would have been interesting to see if it was possible to observe any symptoms of STT *G. abietina* above the snow, as well as to compare the virulence of the two types below the snow cover. Unfortunately this study had to be cancelled due to the small number of infected shoots. The most probable reason for this was the extremely hot and dry summer of 2006, conditions unsuitable for *G. abietina* spore germination and colonisation.

Mortality of middle-aged *P. sylvestris* following defoliation by *G. abietina* and subsequent attack by *Tomicus piniperda*

Defoliation and mortality

There was a significant correlation between defoliation by *G. abietina* and mortality in middle aged *P. sylvestris* (paper III). Infected trees with more than 90% defoliation were at high risk of mortality, caused either directly by LTT *G. abietina* or by secondary damaging agents, such as the pine shoot beetle (*T. piniperda*) (Fig. 10). This high critical threshold of defoliation supports a recent study of *G. abietina* on *P. sylvestris* (Sikström *et al.*, 2005), but greatly exceeds the defoliation limit of 67% (2/3) recommended for sanitation of young *G. abietina*-infected *P. resinosa* plantations in Quebec, Canada (Laflamme, 1993a). *Pinus resinosa*, however, has proven extremely susceptible to the introduced

European race of *G. abietina* (=LTT) in Canada, compared to other pine species (Laflamme *et al.*, 2006). Trees with defoliation levels lower than 90% were only killed in the initial state of the epidemic, in 2001–2002. Surviving trees could withstand high defoliation rates (>90%) for several years, and no trees with less than 90% defoliation died in 2003–2005 (Fig. 10). It seems reasonable to assume that the fungus affected the trees more severely in the two years at the beginning of the epidemic (2000–2001), due to several years in a row with high spore pressure, than in the following years, regardless of the defoliation level. Furthermore, most of the killed trees had lower than average diameters (Fig. 12). Thus, smaller trees seemed to be less resistant to *G. abietina* and were killed at lower defoliation rates. As comparison, STT *G. abietina* is known to cause severe mortality in young *P. contorta* and *P. sylvestris* plantations (Hansson & Karlman, 1997; Witzell & Karlman, 2000). Even though there seems to be a threshold for survival of around 90–95% defoliation, it is important to note that heavily infected trees are potent sources of further infection, and should be prioritised in sanitation cuttings.

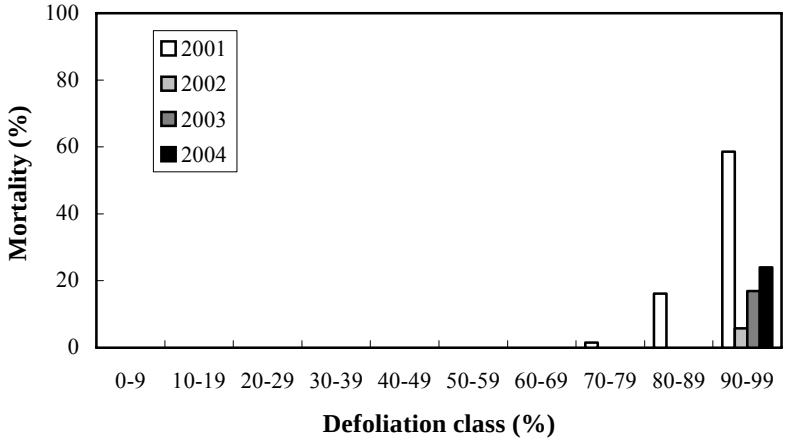

Fig. 10. Percentage of trees in each defoliation class in 2001–2004 that were dead one year after disease assessment.

Causes of mortality

In study III, 66.3% of the trees that died were killed directly by *G. abietina* shoot blight and the rest, 33.7%, following colonisation by *T. piniperda*. In addition, most of the *G. abietina*-induced mortality occurred in 2002, whereas most of the trees that died in 2003–2005 had been attacked by *T. pinperda*. In contrast, *P. sylvestris* trees normally survive one year of total defoliation by needle-eating insects unless they are attacked by secondary bark beetles (Långström *et al.*, 2001; Cedervind & Långström, 2003). Furthermore, in a study of *P. sylvestris* stands defoliated by the pine looper, *Bupalus piniaria* (L.), Cedervind *et al.* (2003) found that more than 50% of the intermediate and dominant trees with 90–100% defoliation survived *T. piniperda* attacks. In study III, however, practically all (99%) *G. abietina*-defoliated trees colonised by *T. piniperda* died.

Susceptibility to beetle attack

Reductions in tree vigour due to defoliation are known to reduce the resistance of *P. sylvestris* to secondary attacks by insects, most frequently and seriously *T. piniperda* (Långström *et al.*, 2001; Cedervind *et al.*, 2003; Sikström *et al.*, 2005). However, the defoliation has to be extensive in order to have significant effects. In study III, 66% of the colonised trees had already died, and 92% were at least 95% defoliated, in the year before beetle colonisation (Fig. 11). This is in accordance with Sikström *et al.* (2005), where *T. piniperda* only successfully colonised *P. sylvestris* trees with more than 97% defoliation. Since no colonised trees with less than 90% defoliation were found, higher beetle pressure cannot have been the reason for the unexpected mortality in 2002 of trees with defoliation levels lower than 90% (Fig. 10). Furthermore, *T. piniperda* colonisation did not peak until 2003, two years after the initial outbreak. In general, there seem to be a critical threshold of 90% defoliation for successful *T. piniperda* colonisation of *P. sylvestris* (Annila *et al.*, 1999; Långström *et al.*, 2001; Cedervind *et al.*, 2003). In the study described by Annila *et al.* (1999), not even simulation of high beetle densities by baiting led to insect colonisation of trees with more than 10% crown foliage. However, Cedervind *et al.* (2003) found that some *P. sylvestris* trees carrying as much as 30% of their original foliage were colonised by *T. piniperda* in the second, third and fourth years after defoliation. These same authors suggest that a build-up of the *T. piniperda* population during the years following defoliation enables recovering trees with more needle biomass to be colonised. This conclusion is not supported in study III, in which no trees with more than 10% foliage were successfully colonised by 2005. However, it should be stressed that reduction in original foliage is not the sole determinant of tree vigour, and Cedervind *et al.* (2003) focused on the young, most severely damaged stands, with densities greatly exceeding those of the sample plots in study III. Furthermore, fire-damaged (Långström *et al.*, 1999), and pruned (Långström & Hellqvist, 1988) *P. sylvestris* trees with up to 20% remaining foliage have been shown to be susceptible to *T. piniperda* attacks.

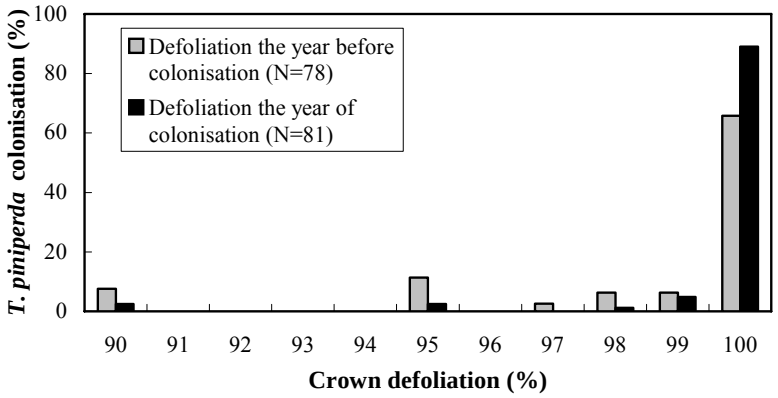

Fig. 11. Distribution of crown defoliation of *G. abietina*-infected trees that were colonised by *T. piniperda* in 2001–2005 (N=81). Due to lack of defoliation data from year 2000, trees colonised in 2001 were not included in "defoliation the year before colonisation".

In conclusion, the results in study III supports the findings of previous studies (Långström *et al.*, 2001; Cedervind *et al.*, 2003; Sikström *et al.*, 2005), in that healthy *P. sylvestris* stands are highly resistant to *T. piniperda* attacks even after severe defoliation of over 90% of the crown. However, the threshold for successful resistance is dependent on the overall vigour of the trees.

Effect of tree size on defoliation and mortality

We found a significant negative relationship between mean defoliation (2001–2005) and tree diameter in study III. This is in accordance with Hansson *et al*. (2005b) in which suppressed trees had the highest frequency of *G. abietina* symptoms. Furthermore, mortality was significantly higher and more rapid among small trees than among larger trees (Fig. 12), in accordance with the findings of similar studies on defoliation caused by *G. abietina* (Kurkela, 1984b; Sikström *et al.*, 2005) and needle-eating insects (Långström *et al.*, 2001; Cedervind & Långström, 2003). Increment losses caused by *G. abietina* could not have been the main cause of the smaller diameter of the dead trees in study III since diameter was measured in the year of the outbreak. Earlier studies have shown that dense pine stands are less resistant to *G. abietina* than stands with wider spacing (Kujala, 1950; Niemelä *et al.*, 1992). The greater susceptibility of suppressed trees in dense stands is believed to be due to a more moist and shady microclimate (Read, 1968; Gremmen, 1972) and high levels of intraspecific competition (Niemelä *et al.* 1992). In our recently thinned study plots, however, mortality was higher among smaller trees even though the number of suppressed trees was low.

The results of the present study support the hypothesis that early and regular thinnings reduce *G. abietina* damage in high-risk areas (Gremmen, 1972; Niemelä *et al.*, 1992; Sikström *et al.*, 2005). Secondary damage caused by *T. piniperda* can be best avoided if the stands are thinned in the winter following the first appearance of *G. abietina* symptoms (Uotila & Uitamo, 1993).

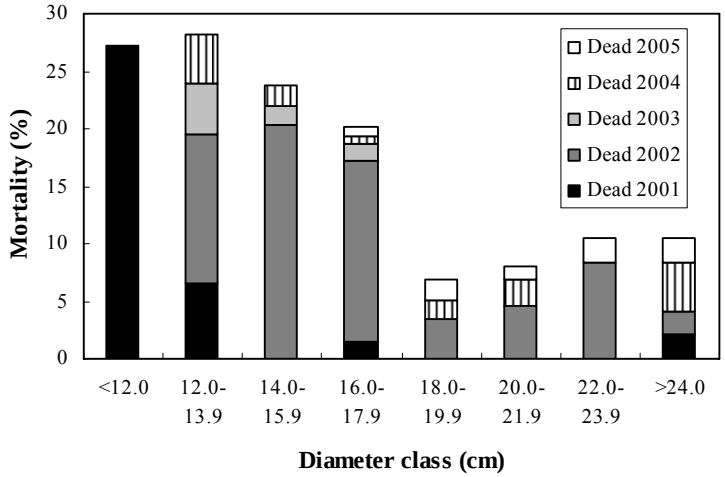

Fig. 12. Proportions of dead trees in different diameter (breast height) classes.

Foliage recovery and increment losses in diseased stands

Foliage recovery

The total defoliation data in paper III did not provide any signs of foliage recovery during the study period (Table 1). However, highly defoliated trees that survived the initial state of the epidemic had slowly started to recover. Our results suggest that slow foliage recovery, along with the ability to kill trees without secondary pests, are major reasons for the severity of *G. abietina* outbreaks in *P. sylvestris* forests (III). After the outbreak in 2001, the percentage of *P. sylvestris* with more than 20% defoliation dramatically increased in Sweden according to national statistics of forest damage (Anon., 2007a) (Fig. 13). Even five years later the forests had just started to recover, and defoliation levels were still higher than normal. No other major reasons for the increased defoliation level were mentioned in the cited text. In comparison, foliage seems to recover faster after defoliation by needle-eating insects. Långström *et al.* (2001) found that more than 75–85% of the needle biomass had recovered four years after almost total defoliation (90–100%) of a *P. sylvestris* stand by the conifer sawfly, *Diprion pini* (L.).

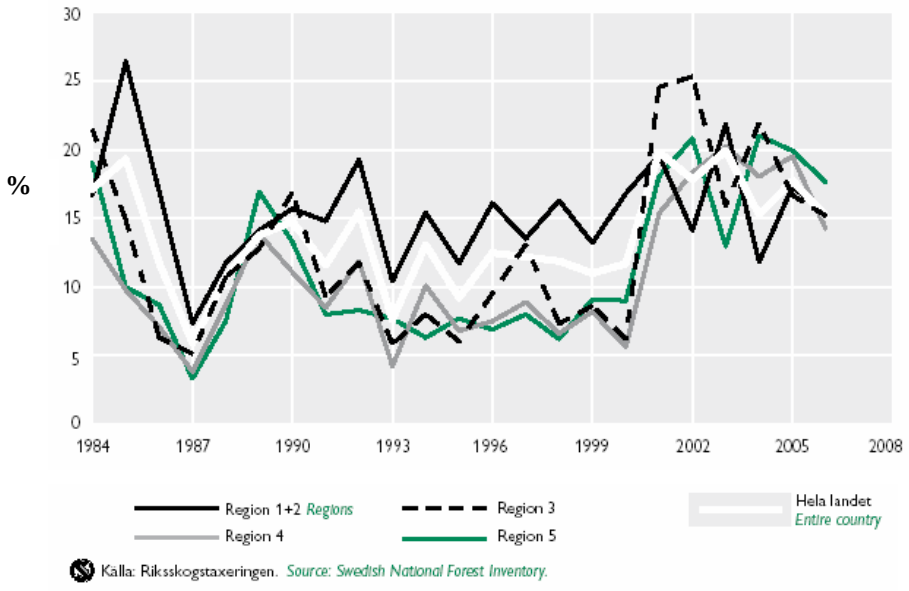

Fig. 13. Percentage of trees with more than 20% defoliation in *P. sylvestris* thinning stands and mature forests in 5 different regions in Sweden (1–5: north–south). Note the drastic increase in defoliation in central Sweden (Region 3) after the severe *G. abietina* outbreak in 2001. From: Swedish Statistical Yearbook of Forestry 2006 (Anon., 2007a).

Table 1. *Mean defoliation (%) for each site and year, including both living and dead trees. Significant between-site and between-year differences (p<0.05) are indicated by different superscript letters*

Site	2001	2002	2003	2004	2005	Mean
1	59,9	62,2	68,9	63,2	63,9	**63.6** [a]
2	55,1	52,8	67,9	64,1	59,1	**59.8** [a]
3	12,5	11,0	34,0	33,2	33,7	**24.9** [b]
4	65,1	69,7	71,0	64,1	68,8	**67.7** [a]
Mean	**51.5** [a]	**52.9** [a]	**62.4** [b]	**57.6** [ab]	**58.7** [ab]	

Defoliation and increment losses

We found a significant reduction in diameter increment with increasing mean defoliation of living trees in study III. Regression analysis indicated that mean defoliation of 2/3 of the crown resulted in a 50% loss in diameter increments, on average, during the study period (Fig. 14). In comparison, two consecutive years of total defoliation of *P. sylvestris* by the pine sawfly, *Neodiprion sertifer* (Geoffr.), caused 56% reductions in diameter increment in a 40-year-old stand during nine years of recovery (Austarå *et al.*, 1987). We did not study the effect of the fungus on height growth, but earlier studies have found similar correlations between height growth and disease incidence on *P. sylvestris* to those between radial growth and disease incidence (Kurkela, 1984b). However, the disease seemed to affect radial growth earlier than height growth in the cited text.

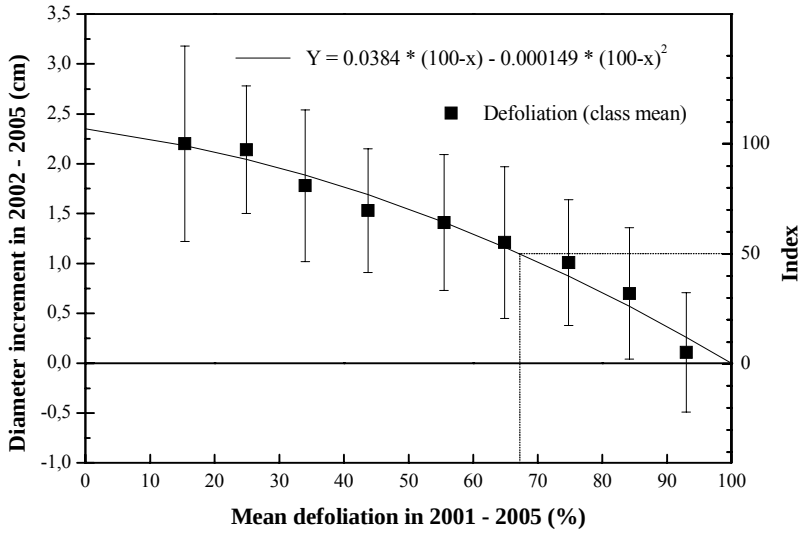

Fig. 14. Diameter increment at breast height of surviving trees (N=478) as a function of mean defoliation in 2001–2005. Black squares indicate calculated increments for trees with defoliation equal to the means of the defoliation classes 0–19%, 20–29%, 30–39%...90–99%. The regression function ($p<0.001$, $r^2=0.84$) was constrained to pass through the origin at x=100. The index shows reductions in increment, associated with increases in defoliation level, relative to trees with very little or no defoliation (class 0–19% = index 100).

Trees with 0–19% defoliation were used as controls to provide reference data and to facilitate comparison of the results with those of similar studies (Söderberg, 1991; Anon., 2007a). The *G. abietina*-induced defoliation caused greater growth losses than those observed by Söderberg (1991) in a study on general defoliation using data from the Swedish National Forest Inventory (NFI) 1984–1990. The two studies agree that 20–40% defoliation leads to *ca.* 10% reductions in increment, but 40–60% defoliation caused slightly higher reductions in the present study (*ca.* 33%) than in the earlier study (*ca.* 20%). The studies are not fully comparable, however, because Söderberg (1991) assessed the upper 2/3 of the crown, while we assessed the whole crown of the sample trees. If we had also assessed only the upper 2/3 of the crowns our estimates of increment losses would have increased, leading to even greater deviations from those reported in Söderberg (1991). One obvious reason for the higher increment reduction in trees infected by *G. abietina*, compared to general defoliation, is that the majority of the defoliation takes place in current-year shoots with vital needles.

Sanitary management in middle-aged *P. sylvestris* stands

We recommend a 75–80% defoliation limit for sanitation cutting in initial stages of a severe *G. abietina* outbreak. If the cutting is done two to three years after the outbreak, and the weather is not conducive for another infection peak, the defoliation limit could be raised to 90%. Attacks of *G. abietina* are known to be predisposed by low temperatures and high precipitation during the vegetation periods (Uotila, 1988; Karlman *et al.*, 1994) and a correlation between high precipitation and disease incidence was also found in the outbreak of 2001–2003 (Hansson & Ottosson Löfvenius, 2005). Due to recent thinning operations, the study sites in paper III were close to the lowest timber volume permitted by the Swedish National Forestry Act (Anon., 2006) already before the *G. abietina* outbreak; the number of stems per hectare was 462–710 in the four sites. Removing the trees with ≥90% defoliation in 2001 would have resulted in the volume being lower than permitted in a majority of the sites and removing the trees with ≥75% defoliation would have resulted in the volume being lower than permitted in all four sites. These stands would therefore either have had to be clear-felled or left without any sanitary action. Thus, in Sweden it is important to consider the lowest timber volume limits permitted by the Swedish National Forestry Act when considering sanitation measures. This is in accordance with two Finnish studies that concluded that it is not profitable to grow a *P. sylvestris* stand with less than 600 stems/ha after the first thinning (Aalto-Kallonen & Kurkela, 1985; Uotila & Uitamo, 1993). However, if the stand is diseased after thinning, as the case in our study area, a minimum density of 700–800 stems/ha was recommended (Aalto-Kallonen & Kurkela, 1985).

In earlier reports, *G. abietina* (EU race, LTT) initially infected the lower branches and then progressed up in the crown (Bergdahl & Ward, 1984; Barklund, 1989). In the 2001–2003 epidemic in Sweden, however, infections were often found throughout the trees' crowns, often with severe reductions of shoots in the uppermost parts. In study III, the top-shoots were often found dead even though

up to 40% of their crowns were still green at the time. This behaviour strongly reduces the possibilities to control the disease by pruning (*cf*. Laflamme, 1993a), and pruning was, therefore, not recommended in Sweden during the 2001–2003 outbreak. A small-scale sanitation trial was, however, performed within this project, even though not presented as a manuscript in this thesis. In a middle-aged *P. sylvestris* stand with moderate infections of LTT *G. abietina*, plots were laid out where trees with at least 67% (2/3) defoliation were thinned and all other trees pruned to a height of 6–7 m. At the time of disease assessment in the fall of 2006, two years after pruning, no effects of pruning, in terms of lower rates of new infections, could be seen compared to the control plots. The main reason for this was the fact that progression of the disease had ceased also on the untreated plots during the years since the peak of the epidemic in 2001–2002 and there were hardly any new infections in the stand. Therefore this trial can not fully be evaluated until a new infection wave hits the stand.

Conclusions and management applications

- *Pinus sylvestris* seedlings planted the first growing season after sanitary clear-cuts will suffer from *G. abietina* infection, causing shoot blight, stem canker formation and mortality, already within one year.

- *Gremmeniella abietina* may survive and reproduce in *P. sylvestris* slash for at least 18 months, thus being able to damage seedlings planted also a second year after sanitation clear-cuts. It is, therefore, recommended to wait at least two years after sanitation clear-cuts before replanting an infected *P. sylvestris* stand.

- If the stand is regenerated already the first vegetation period after a sanitary clear-cut, careful removal of infected slash may reduce infection rates by up to 50%. Seedlings on clear-cuts with large amounts of infected slash were more heavily infected and should be given priority for slash cleaning.

- It is not possible to recommend a time of year to harvest infected stands to minimize survival and vitality of *G. abietina* in the slash. Germination capacity of *G. abietina* conidia from the slash was equally high regardless of the month of felling of the sample trees.

- The proportion of seedlings infected with LTT *G. abietina* is not necessarily lower in *P. contorta* compared to *P. sylvestris* regenerations. However, infected *P. contorta* seedlings seem more resistant as evidenced by their higher survival rates, shorter lengths of shoot infection and quicker recovery by production of new leaders.

- Infection by LTT *G. abietina* in *P. contorta* is mainly limited to the outermost parts of the shoots. In our study the mean infection length on the main stem of surviving seedlings was 39 mm for *P. contorta* compared to 104 mm for *P. sylvestris*, corresponding to 23% and 49%, respectively, of the total seedling length.

- Forty-seven percent of the *P. contorta* seedlings that survived infection by LTT *G. abietina* in 2005–2006 had developed a new leader shoot in 2007, compared to 19% of the infected *P. sylvestris* seedlings.

- Resistance to LTT *G. abietina* in *P. contorta* is mediated by the production of ligno-suberised barrier walls and phenol-filled cells around the necrotic tissue. Similar defence mechanisms were also found in infected *P. sylvestris* shoots.

- The results indicate that *P. contorta* has a more developed resistance to LTT *G. abietina* compared to *P. sylvestris*. The extent of this resistance needs to be further tested on larger trees under more severe climatic conditions.

- Healthy, middle-aged *P. sylvestris* trees normally survive up to 90–95% defoliation caused by a single outbreak of *G. abietina* shoot blight. However, trees with 80–90% defoliation may be killed in the initial phases of severe outbreaks.

- Small and/or suppressed trees die more frequently and more quickly than larger trees and should be prioritised in sanitary thinnings.

- Most of the middle-aged trees that die in a severe *G. abietina* epidemic are killed directly by *G. abietina* shoot blight. The pine shoot beetle (*Tomicus piniperda*) mainly attacks dead or occasionally severely weakened trees; in our study trees with more than 95% defoliation.

- There is a highly significant correlation between defoliation by *G. abietina* and growth reduction in middle-aged *P. sylvestris*. Trees with a mean defoliation of 1/3 and 2/3 of the crown suffer *ca.* 20% and 50% losses in mean diameter increment, respectively.

- Foliage recovery seems to be slower after attack by *G. abietina* than after attacks by needle-eating insects.

Praktiska råd för skogsbruket

- Då *Gremmeniella*-svampen (syn. ”tallens knopp- och grentorka”) visat sig kunna överleva åtminstone 18 månader i infekterat avverkningsris av tall, samt svåra skador visat sig på plantor satta första sommaren efter avverkning, rekommenderas en hyggesvila på åtminstone 2 år om *Gremmeniella*-skadade bestånd ska föryngras med samma trädslag.

- Om *Gremmeniella*-skadade bestånd föryngras inom två år efter sanerande avverkning kan bortförande av infekterat hyggesavfall reducera antalet skadade plantor med upp till 50%.

- Våra infektionsförsök på plantor bekräftade att contortatallen är mer resistent mot large tree type (LTT) *Gremmeniella* än svensk inhemsk tall. Med den virulens som LTT visar idag kan contortatallen anses vara ett intressant alternativ till svensk tall, särskilt i områden som drabbats hårt av LTT *Gremmeniella*.

- För att säkra överlevnaden av enskilda träd rekommenderas saneringsgallring av träd med minst 75–80% barrförlust efter utbrott av en *Gremmeniella*-epidemi. För träd som överlevt skadorna från de första åren av utbrottet kan gränsen höjas till 90% barrförlust.

- Små och undertryckta träd dör oftare och snabbare av *Gremmeniella*-skador än större träd och bör prioriteras vid sanerande gallringar.

- De flesta träden som dör under allvarliga *Gremmeniella*-epidemier dör som en direkt följd av själva svampen. Större märgborren angriper främst redan döda eller svårt skadade träd med en barrförlust på minst 95%. Sanerande gallring av kraftigt infekterade träd redan första vintern efter det att årsskotten dött och färgats röda kan förhindra att virket skadas av märgborrar och medföljande blånadssvampar.

- Diametertillväxten för enskilda infekterade tallar avtar med *ca*. 20% vid en barrförlust av 1/3 av kronan och *ca.* 50% vid en barrförlust av 2/3 av kronan, vilket kan ligga till grund för uträkning av produktionsförluster i *Gremmeniella*-skadade bestånd.

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