How Do the Epidemics of *Gremmeniella abietina* Start?

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Abstract – *Gremmeniella abietina* type A caused two widespread epidemics in Nordic countries: in 1982 in Finland and in 2001 in Sweden. The reasons for these epidemics are discussed based on the inoculation experiments in Finland and the literature. The fungus has been inoculated by putting a piece of mycelium in the phloem or by spraying the conidia or ascospores on pine shoots. Mycelial inoculations cause cankers in dormant period inoculations, but not in summer inoculations. During the dormant period, pine cannot actively defend against the fungus. Spore inoculations are successful in summer, which is also the natural spreading time of the fungus infects the bract and during the dormant period it grows to the phloem through poorly developed cork layers between the dead bract and living phloem. A serious epidemic needs a rainy and cloudy summer and also the same kind of summer two years before. A mild winter enhances the growth in cankers, but a mild winter alone cannot cause the epidemics. *Gremmeniella abietina* damage is controlled by using local or a little bit of northern provenances.

inoculation/ Finland/ Sweden/ bract

Kivonat – Hogyan indulnak a *Gremmeniella abietina* járványok? A *Gremmeniella abietina* A típusa két nagykiterjedésű járványt okozott az északi országokban: 1982-ben Finnországban és 2001-ben Svédországban. Finnországi inokulációs kísérletek és a szakirodalom alapján elemezzük a járványok okait. A mesterséges fertőzéseket micelium darabkák háncsba helyezésével, illetve konídium, vagy aszkospóra szuszpenzió hajtásokra történő permetezésével végeztük. Miceliummal a nyugalmi időszakban végzett fertőzések nekrózist okoztak, de a nyári fetőzések nem. A nyugalmi időszakban a fa nem képes aktívan védekezni a gomba ellen. A spórával végzett fertőzések nyáron sikeresek, ami a gomba természetes terejdésének időszaka is. A fertőzés sikere a megfertőzött hajtások gyenge szerkezeti ellenállásával magyarázható. Kezdetben a gomba a fedőpikkelyeket fertőzi, majd a nyugalmi időszakban a gyengén fejlett kéregrétegeken keresztül az elhalt fedőpikkelyek és az élő háncs között behatol a háncsba. Egy komoly járvány esős és felhős nyarat igényel, és ugyanilyen nyarat az előző két évben is. Az enyhe tél elősegíti a nekrózisok növekedését, de egymagában nem okoz járványt. A *Gremmeniella abietina* okozta károk ellen a helyi, vagy kissé északibb származások alkalmazásával védekezünk.

Inokuláció / Finnország / Svédország / fedőpikkely

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1 INTRODUCTION

Gremmeniella abietina (Morelet) type A has caused serious damage in pole stage and bigger Pinus sylvestris L. trees. In Finland the worst epidemic was in 1982 and in Sweden in 2001 (Uotila 1988, Wulff et al. 2006). The previous weather conditions have been assumed to the main causes of Gremmeniella epidemics (Uotila 1988). The conidia and ascospores spread in the summer and so the rainy summer enhances the infections (Petäistö – Heinonen 2003, Nevalainen 1986, Uotila 1985). Also the spores germinate only in moist conditions (Dorworth 1972). It is obvious that the resistance of the diseased shoots has been weakened. In provenance experiments, southern origins suffer more than local and northern provenances (Uotila 1985). The summer frosts and shading are also important factors affecting epidemics (Petäistö – Repo 1990, Petäistö & Kurkela 1993, Read 1968, Uotila 1988, Sairanen 1990). The life cycle of the fungus lasts two years (Uotila 1985, Hellgren - Barklund 1992), so it is enough for a fungus to have good conditions for infections every second year. The infection happens after a latent period probably through the stomata of bracts on the base of needle fascicles (Patton et al. 1984). The same kind of infection process has been described on Diplodia pinea (Flowers et. al. 2006). After a first year infection the necrophylactic periderm can protect the surrounding phloem tissues from canker spreading. Gremmeniella infection needs enough so-called conducive days during the dormant period (temperature $+5^{\circ}C - -5^{\circ}C$) (Marosy et al. 1989). In Finland and Sweden we have enough conducive days every year and so the number of conducive days or dormant period weather cannot fully explain the variation in yearly disease level. Here we will synthesise the factors affecting the Gremmeniella epidemics based on the results of several inoculation experiments and the literature.

2 INOCULATION EXPERIMENTS

Several inoculation experiments have been done in Finland (Kurkela –& Norokorpi 1979, Petäistö – Kurkela 1993, Petäistö 1999, Petäistö and Laine 1999, Petäistö 1995, Petäistö et al. 2005, Uotila 1983, 1990, 1991). Type A and B isolates have been used in these inoculations. Most often the type is known. The mycelium inoculations in phloem have been done over the year (*Figure 1*). So we know that *Gremmeniella* mycelium causes the canker always if it is inoculated during the dormant period. In August type A cause more often cankers than type B, which is showing that type A is more aggressive pathogen (Terho – Uotila 1999). In the summer the pine can resist mycelial inoculations (Uotila 1990). This is logical because the fungus has then more time to grow without the active defence of the tree. In spring it seems that the defence activities begin in April, so the canker is mainly grown in late autumn and early spring. The mycelium cause serious cankers (Petäistö 1993). In spore inoculation experiments the delayed start of growth in the spring has increased infections (Petäistö – Laine 1999).

The spore inoculations with conidia or ascospores have been made over the year in spite of the period from January to April. It is interesting that these inoculations have been successful in the same time as the spores are spreading in nature (*Figure 1*). The successful period of spore inoculations is just opposite than that of the mycelial inoculations. At first this sounds confusing, but this fact gives a good opportunity to understand epidemics. The infection happens in the cases when the pine has not developed structural resistance against the fungus, which is waiting latent in the bract. This sounds too simple. We need find more facts to support this theory. The first-year nursery seedlings are most susceptible to spore inoculations during late summer simultaneously with bud development (Petäistö 1999, Petäistö 2005). This difference is noticed in container

seedlings and in bareroot seedlings. The needles of these first-year seedlings are primary needles. There is no structural resistance between the primary needle and the shoot. Primary needles are not developed as needles after the first summer, but they form a bract at the base of the needle fascicle. The bract initials are already in the bud in the first growing season. Older seedlings have been more susceptible to early summer inoculations. The infection of these seedlings probably happens via bracts (Patton et al. 1985) or via the scales of the long shoot (Siepmann 1976). Why does not the infection happen via the base of the needles? Is latent infection possible in the needles? In diseased nursery seedlings pycnidia are common on primary needles. In nature pycnidia are not common in the needles. The reason for this could be that the diseased needles drop down before pycnidia develop or that the infection really occurs via the bracts and the *Gremmeniella* infection is that the needle bases turn brown in the spring. This happens only in those needles which are connected to infected phloem. The tip of needle is still green which probably means that the fungus has not originally penetrated into the needle.

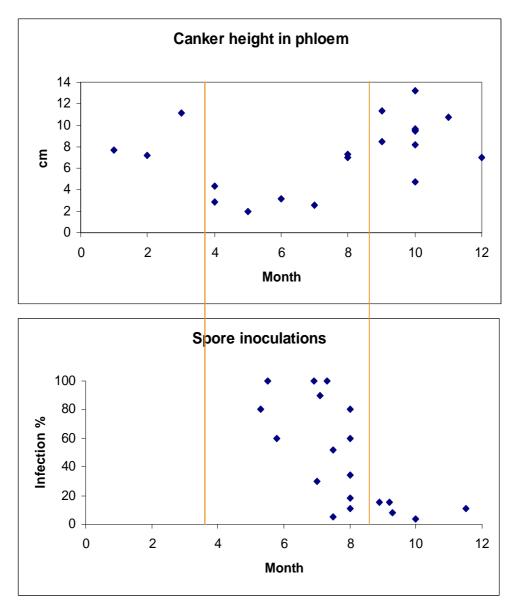


Figure 1. The shown results originate from several experiments in different years by the authors. In the upper picture the results of mycelium inoculations in phloem are shown and in lower picture are the results of spore inoculations sprayed on shoots

3 WEATHER CONDITIONS

The following weather conditions were common for major Nordic epidemics: a big rainfall during the previous summer and low solar radiation during the growing season. The winter of 1981-1982 in Finland was cold and the winter of 2000-2001 in Sweden was mild. So we can conclude that the epidemics can occur in spite of the winter conditions. The damage in Sweden was very severe and it is possible that the fungus causes larger cankers during a mild winter compared to the cankers in a cold winter. The winter conditions are not the main factors, because *Gremmeniella* spores spread in summer and the structural resistance of pine is developed during the summer. Type B Gremmeniella damage is a little bit different because type B Gremmeniella typically grows in perennial cankers below the snow.

In winter storage experiments the seedlings have been infected even more in cold winter temperatures (Petäistö – Laine 1999). The cold winter temperatures delay the start of growth in spring and so the fungus has time to invade the host tissues. This corresponds with the late appearance of visible symptoms.

Single stands were destroyed in the areas where most stands were not severely infected. This is clearly caused by the seed origin coming from too southern conditions or nitrogen fertilisation (Kallio et al. 1985, Aalto-Kallonen – Kurkela 1985). In both epidemics the fungus was present almost everywhere in the lower branches and the pine understory. So the inoculum was everywhere, but the surviving shoots had developed their structural resistance to the disease.

4 CONCLUSIONS

The main cause of *Gremmeniella* epidemics is the rainy weather during the summer in conditions where there is a big inoculum potential due an epidemic two years before. If the rainy season occurs just occurs at the same time as shoot lengthening the *Gremmeniella* infections will increase. Late summer frosts increase also the pine susceptibility. We can control the future damage by using local or north to south transferred provenances. The altitude is also important. A new risk is that foresters react to global warming by planting southern provenances since these provenances are still susceptible to *Gremmeniella abietina*.

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