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# Status of Pathogenic and Physiologic Races of *Gremmeniella abietina*

The concept of different races or strains of *Gremmeniella abietina* (Lagerb.) Morelet with different pathogenic capacities can be traced back to 1945 in Europe when Ettlinger suggested the existence of two races in the Swiss Alps (10). Differences between the races in terms of conidium morphology and geographic and host disposition were noted. Results of work by Krywienczyk and me in 1975 (9) led us to conclude there are three races of *G. abietina*: the North American (Fig. 1), the European (Fig. 2), and the Asian (Fig. 3). *G. abietina* was found only recently in Japan (20), and further races may be defined there.

European and North American observations along these lines are similar but not parallel. With current knowledge, the racial groupings, if they can be so designated, may not be superimposed or intermixed. Accordingly, I shall treat them separately.

## Scientific Nomenclature

Before discussing the matter of race differentiation, I call attention to the taxonomic position assigned this fungus. There has been resistance to the change in name from *Scleroderris lagerbergii* Gremmen to *Gremmeniella abietina*, as there was earlier to the change from *Crumenula abietina* Lagerb. to *Scleroderris abietina* (Lagerb.) Gremmen to *Scleroderris lagerbergii*, and for the same reason in each instance: A change in scientific name is inconvenient for pathologists who must employ the terms in routine work. It is plain, though, that there no longer is, and never again will be, a valid genus *Scleroderris* according to the International Code of Botanical Nomenclature. Both *Scleroderris* and *Crumenula* are synonyms of the genus *Godronia* (13). As noted by Morelet (17), the perfect state of *G. abietina* has apparently never before been properly situated.

The most persistent reaction against such changes is based on the inconvenience they cause pathologists, foresters, and agriculturalists. We shall phase out *Scleroderris* rather quickly in written work but less quickly in discourse, with no harm to anyone in either case. In fact, the common term "Scleroderris canker" is achieving increasingly wide acceptance with time and has become the effective common name of the disease in North America. My suggestion to replace this common name with "Gremmeniella canker" encountered de facto rejection both within the profession and by other persons closely enough associated with the problem to voice an opinion. A common name is, by definition, the one accepted by the majority. The suggested change should be abandoned to stabilize this flux of common names in the interest of the layman and others who attempt to apply our research in the field.

Interestingly, the disease to which the term "Scleroderris canker" was initially applied (29) is caused by an entirely separate pathogen, formerly *Scleroderris abieticola* Zell. and Goodd., which was subsequently redisposed to the genus *Grovesiella* (12) and the associated disease renamed Grovesiella canker (2). Plainly, the matter of common names is not one that should be argued in depth. The term "Scleroderris canker" is surely descriptive of a prime North American race symptom and relates the disease and its consequent damage to a defunct but fully traceable genus.

This detailed consideration of common names should not be allowed to obscure the argument that there is an alternative to the scientific name *G. abietina*, which is *Ascocalyx abietina* Schläpfer-Bernhard (21). This particular segment of Schläpfer-Bernhard's work has received only limited support. Placement of this species (then a species of *Crumenula*) within *Ascocalyx* was rejected as far back as 1945 (10), and this rejection was reinforced by later observations and analyses (7). Addition of *Gremmeniella juniperina* K. and L. Holm (14) reinforces this formerly monotypic genus. Provided the choice of *G. abietina* and formation of a separate genus to accommodate the organism are biologically appropriate, the name will be protected under the provisions of the International Code of Botanical Nomenclature. The same is not true, however, for the names and systems of fungus races.

## Physiologic Races

The code does not seriously regulate the assignment of race names among fungi, much less the erection of race structures. Presumably, one congress or another will deal with this problem, but not until there is sufficient cause. At present, we are merely separating biotypes or serovars of *G. abietina* and not identifying pathovars, much less natural relationships. Furthermore, in most instances we are not assigning terms or identifying systems below the species level in even quasi-scientific fashion on a worldwide basis. One of the points made in 1975 (9), however, was that one cannot elaborate taxonomic systems of permanent value at the fringes of a taxon by exotic means without thorough knowledge of the taxon acquired by the same method. Browder et al (3), in fact, expressed the view that assignment of formal taxonomic status to races of fungi is not required.

In 1975, Janina Krywienczyk, a research scientist in the Department of the Environment at the Forest Pest Management Institute in Sault Ste. Marie, and I proposed an outline of physiologic races of *G. abietina* based on serologic comparisons (9) and supported in limited fashion by observations of gross culture morphology. We proposed three races. The North American race was based on a group of serologically similar isolates from Canada and the United States that differed predictably in

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serologic reaction from a group of isolates from Europe, termed thereafter the European race. The term "Asian race" was applied to a group of five isolates from Hokkaido, Japan.

Since that time we have traced the North American race across the North American continent; it is transcontinental. The Old World European race extends from Lapland to the middle of the Italian peninsula, from 2,000 m or more above sea level in the Alps near Tyrol to an elevation below 100 m at the southern extreme, as determined from tests of a collection of isolates selected by Francesco Moriondo of the Universita Degli Studi di Firenze.

Whereas the concept of both the European and the North American races is open to partial or total revision with the addition of new information, "Asian race" is clearly a qualified distinction, based solely on five isolates from a relatively small geographic area. I tried unsuccessfully to obtain isolates from the People's Republic of China and the Soviet Union, each of which bears contiguous zones of climate and host species favorable to development of the pathogen. The pathogen existed as well in New York, in Great Britain, and in *Picea* spp. in northern Quebec, but I was unable to obtain cultures at the time of original testing.

The grouping of three races has proved useful thus far. The original work revealed further differences among isolates but the test was ultimately interpreted in terms of its least sensitive response in order to yield fully reproducible results. Further intraspecies distinctions become visible as the method is increasingly refined. Those dealing with the technique may, by refining it further, distinguish a greater number of races at the same level of confidence as in the original application, or at a higher level. Skilling (24) and Wendler et al (27) have, in fact, characterized an "intermediate race" that responds both to European and to North American antisera when subjected to serologic examination.

Much as was the case with the change in scientific name, there has been some reaction among pathologists to the term "physiologic race." At present we are, by definition, dealing with physiologic races or serovars in North America, and it is important to recognize the fact. Stakman and Harrar (25) define physiologic race as: "A biotype or group of biotypes within a species or variety that can be distinguished with reasonable certainty and facility by physiologic characters, including pathogenicity, and, in some fungi, by growth characters on artificial media." All pathogenic races are physiologic races then, but the converse may not be true.

## Pathogenic Races

The chemotaxonomic approach to race characterization was adopted because conducting field inoculations with imported isolates in order to define virulence was considered inadvisable, even though *G. abietina* supposedly existed as a homogeneous species on a worldwide basis. That the European race was characterized in North America after

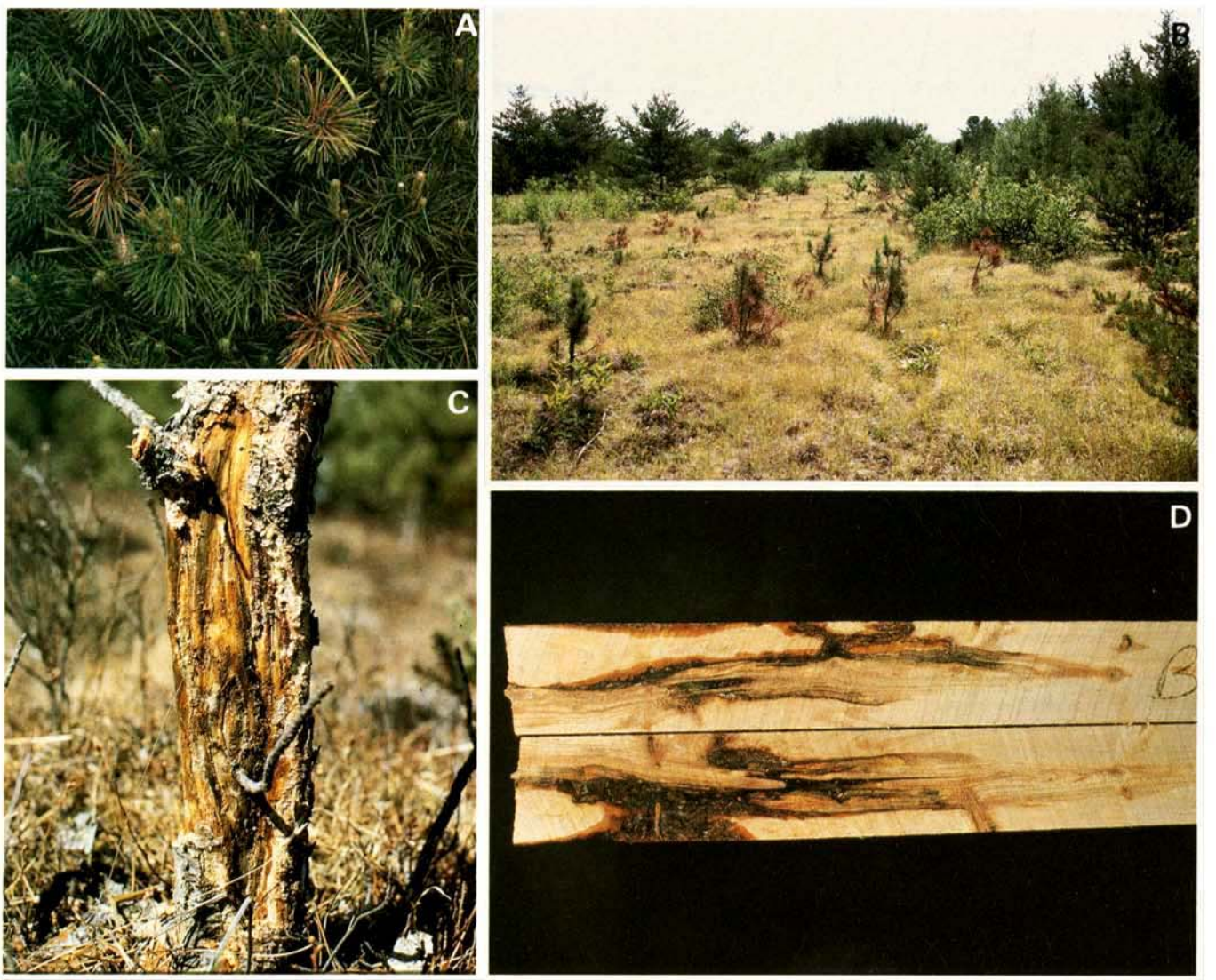


**Fig. 1. Manifestations of North American race of *Gremmeniella abietina*: (A) Needle base discoloration on 4-year-old red pine. (B) Depleted plantation of 8-year-old red pine, with branch mortality of survivors. (C) Chronic basal canker of 16-year-old red pine. (D) Ingrown bark, resin pockets, discolored wood, and distorted grain on 2 × 4 in. boards from 24-year-old red pine.**


**Fig. 2. Manifestations of European race of *Gremmeniella abietina*: (A) Infected young Scots pine (left) and lodgepole pine (right) and (B) lodgepole pine with green stain and apothecia, in Norway. (Courtesy F. Roll-Hansen)**

a disease syndrome was recorded similar to that in Europe (23) lends credibility to the serologic method as a valid basis for defining a "New World European" versus a "North American" pathogenic race, but this remains circumstantial evidence. The observation that the North American race invades only the lower crowns of *Pinus* spp. whereas the European race invades both upper and lower crowns of various conifers of all ages (4–6,8) lends additional support. The Asian race was described as a top invader of pole-sized *Abies sachelinensis* Mast. weighted to or near the ground during winter by snow at higher altitudes in Hokkaido (28). The most pronounced symptom, upper-crown dieback, appeared after the trees resumed an erect position in the spring (28; S. Yokota, *personal communication*). In addition, the *G. abietina*-linked disease of white pine in Japan may involve a separate race of the pathogen (S. Yokota, *personal communication*). Hence, two and possibly three discrete disease syndromes accompany the three physiologic races.

It is becoming increasingly obvious, however, with the addition of new information, that the "three race/three disease/three continent" set originally described is only a partial explanation. According to Roll-Hansen (*personal communication*): ". . . I would conclude that both races (or similar races) exist in Norway. We have for decades been aware of this difference. Dr. Ivar Jørstad thus emphasized the differences between the fungus in West Norway (similar to your European race) and the fungus in East Norway (similar to your North American race)." Roll-Hansen and Roll-Hansen (19) also drew attention to the possibility that abundance of the perfect state of *G. abietina* in Norway and Great Britain may be related to climate.

Ettlinger (10) considered the possibility that separate races of *G. abietina* were responsible for mortality of *Pinus cembra* L. at higher altitudes and *P. nigra* Arnold at lower altitudes in Switzerland, as did Donaubauer in Austria (5). Barbacovi et al (1) inoculated *Pinus* spp. with isolates from various hosts and various locations and reported differences in virulence or infectivity. Barbacovi et al (1), Donaubauer (5), and Ettlinger (10) described or illustrated conidia from *P. cembra* at higher elevations with approximately twice as many septa as conidia collected from other hosts at lower elevations. Several of these isolates as well as two Japanese isolates—one with conidia with three or four septa and the other with conidia with eight or fewer septa—were recorded and illustrated (9). This contrast in degree of septation of the two Japanese forms had been described earlier and in greater detail by Takahashi (26). Roll-Hansen noted stem cankers on *P. sylvestris* in Norway (18) and reproduced the symptom by mycelial inoculation. Kurkela and Norokorpi (16) incited formation of stem cankers of Scots pine in Finland by mycelial inoculation and recorded decreased terminal growth where canker size exceeded 20% of stem diameter. Skilling (*personal communication*) identified stem cankers on *P. resinosa* Ait. from an area in New York where only the European race was recovered. Similarly, Bergdahl recorded upper and lower stem cankers of *P. resinosa* in Vermont where only the intermediate race was found (*personal communication*). Morelet (17) proposed varietal subdivision of *Brunchorstia pinea* (Karst.) Höhn. the imperfect state of *G. abietina*, into *B. pinea* var. *cembrae* and *B. pinea* var. *typica* on the basis of a study of the imperfect reproductive state.

The proposals and descriptions from Europe, then, refer primarily to what could be termed pathogenic races, with the possible exception of Morelet's work,

**Fig. 3. Manifestations of Asian race of *Gremmeniella abietina*: Canker on 2-year-old shoot of Todo fir, with many pycnidia. (Courtesy S. Yokota)**

which may relate directly or indirectly to any subdivision of pathogenic races. There remains the task of amalgamating these reports and proposals into a single definitive system and correlating it with the system of physiologic races from North America to yield an efficient, reliable outline of pathogenic races or pathovars on a worldwide basis. Such amalgamation will undoubtedly occur in Europe shortly, in view of the degree of interest directed to the problem recently and a background of nearly a century of research and observation.

Browder et al (3) present a thoughtful argument in favor of ultimately basing distinctions evolving from host-parasite specificity studies on pathogen genotype with respect to pathogenicity. This

should provide a stable fundamental basis from which to develop race systems, but application of this procedure in cases of forest crop diseases could present a challenge. That third component of the classic disease interaction picture—environment—gains particular importance when forest crops are considered. While not insurmountable, logistics assume major proportions when a subject crop matures over decades rather than in a single season, is probably subjected within a single rotation and at various stages in plant development to all extremes of climatic predisposition, and itself progressively and formidably alters the site and (at least) the microclimate during maturation.

Correlation of the North American and European information will be facilitated by the increasing pool of results being generated as more and more European collections are serologically typed. A single major problem remains in North America: definition of etiology and epidemiology of all races on various host species and under various environmental conditions. This can be done with impunity only in an area where all the races to be used in inoculation are known to exist.

In general, the original physiologic race structure has been substantiated by subsequent work (27). This is encouraging, as the serologic test is subject both to the consequences of intraspecies fungus variation, as is any mode of classification, and to variation in response of the experimental animals. As with aspects of classic taxonomic systems, handling of the subject material bears elements both of a science and of an art. Extensive experience with the subject animals and with serologic systems overall is usually reflected in the quality of results obtained. It will be partly good fortune, nonetheless, if this race structure remains unchanged, because, to my knowledge, the pathogen was not even sought, much less diagnosed, in South America and Africa. Equally possible, the current scheme could survive yet be amended if further research shows that each physiologic race is comprised of more than one pathogenic race.

This is the first instance, to my knowledge, in which chemotaxonomic techniques have formed a basis for imposing international quarantines to constrain movement of plant-pathogenic fungi. Regulations both in Canada and in the United States were written initially to exclude and constrain spread of the European race of *G. abietina*. Circumstantial evidence fully supports the premise that the New World European race has a counterpart throughout Europe. The sudden appearance of severe and conspicuous upper-crown dieback of mature pines in the state of New York nearly a century after the first similar record appeared in Europe forces us to regard the possibility that the European race is an exotic in North America. Nonetheless, serologic identity and disease syndrome should be directly correlated to substantiate use of serology as a basis for imposing quarantine statutes.

At present we are loath to conduct such tests in Canada because of the danger of introducing the European race, the potential of which is incompletely understood, into the transcontinental host continuum of northern Canada. We discussed this earlier: "No degree of scientific curiosity or precaution will justify field inoculations with foreign isolates"(9). The consequences of such an adventure appear particularly awesome if one considers Skilling's work conducted in New York State (*personal communication*), in which species of conifers that predominate in the western Cordilleran Range were recorded as hosts of the European race of *G. abietina*, and the work of Shichkina and Tzanava (22), who recorded *Brunchorstia pinea* on *Cedrus deodara* (Roxb.) Loud; cedars were previously regarded as immune.

## Diseases Caused by *G. abietina*

I noted in 1971 (6) that *G. abietina* apparently caused different diseases in Europe (termed dieback, topkill, bud drought) and in North America (termed Scleroderris canker). The overall pathogenic interaction is strongly affected by environmental conditions (5,6,8,11,15), and the pathogen has been termed a facultative saprophyte both in Europe (15) and in North America (9). Donaubauer indicated (*personal communication*) that the pathogen may exist at an endemic level during extended periods when weather conditions are less than optimum for disease intensification, damaging the main stem and lower crown alone, only to become epidemic in the upper crowns during periods of weather highly favorable to reproduction of and infection by the pathogen. Presumably the somewhat resistant *P. banksiana* Lamb. will serve as an "endemic refuge" in the Great Lakes area of North America during periods of adverse weather and, subsequently, as a widespread source from which inoculum is dispersed during periods of damp weather most suitable for inoculum development. Such periods are equally good for inoculum dissemination and infection and all susceptible species suffer, but the likely site for a subsequent epidemic and the attendant logarithmic increase of inoculum would be monocultures of the highly susceptible *P. resinosa*. This would parallel the *P. sylvestris*/*P. nigra* sequence in Europe and might be projected as well to the *P. contorta* Dougl./*P. ponderosa* Laws. interface of the Cordilleran Range. The notion of an endemic refuge is implicit in our professional dialogue but it assumes discrete form when applied to a facultative saprophyte of extended host range (9) such as *G. abietina*.

On the basis of present knowledge, disease development in response to infection by the North American race would be a logical standard on which to construct a host-response model. Restriction of the pathogen to the lowermost 2 m of crown has remained a consistent aspect of the North American race syndrome over nearly two decades of observation. The European race syndrome, by contrast, is more variable. An obvious weakness appears in this argument: We do not know if the European race will

also be limited to the lowermost 2 m of crown in areas where the North American race now predominates. Further, we are directed toward restriction of the European race to its present range. Attempts to test the effect of the European race outside its present range would contravene not only our professional ethics but Canadian law as well.

It may be sufficient to note that the European race causes upper-crown dieback of mature pines in northern Europe. In any case, if the North American physiologic race remains restricted to the lower crown of a limited selection of *Pinus* spp. and the European physiologic race or its components infect both upper and lower crown tissues of most mature conifers, then it would be reasonable to assign separate titles to the diseases incited by each race. Whether or not separate disease names (canker and dieback) are applied, however, is entirely a matter of convenience, in view of the list of races we are acquiring and the broad audience to which a uniform and simplified frame of reference must be extended. If any basis can be established for retention of a common disease name, utility might best serve that purpose. Only further research will reveal the extent to which the biology of the various races differs and the extent to which forest managers will need to vary control procedures according to the race encountered.

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