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Mini review

Actinomycetes as plant pathogens

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Abstract. Biology, taxonomy, pathogenicity and control of plant disease inducing actinomycetes are reviewed. Recent progress in the study of potato, sweet potato, blueberry and fruit and forest tree diseases is illustrated. The role in potato scab pathogenesis of the newly discovered phytotoxins, thaxtomins, is discussed.

Abbreviations: ATCC = American Type Culture Collection, Rockville, Md, USA; IMRU = Institute of Microbiology, Rutgers University, NJ, USA; ISP = International Streptomyces Project; PCNB = pentachloronitrobenzene; 3,5-D = 3,5-dichlorofenoxyacetic acid.

1. Introduction

Actinomycetes, as causal agents of plant diseases,[1] are not a novelty. The detection, isolation and description of the organisms responsible for the scab disease of potato tubers date back to more than a century ago [Thaxter, 1891, 1892] and soil rot of sweet potato was first mentioned around the same time [Halsted, 1890]. This was just a few years after the discovery and description by Harz [1877] of the first actinomycete, *Actinomyces bovis*. It might seem a paradox that animal and plant pathogens were among the first actinomycetes to be identified, considering the overwhelming relevance saprophytic forms were to assume later on, particularly during the antibiotic era.

In comparison to other bacteria, apparently actinomycetes play a relatively minor role in plant diseases. However they represent major pathogens of certain crops in particular areas and, under special conditions, affect quality, palatability and market value of agricultural products, contribute to 'soil sickness' and produce phytotoxins.

In the past actinomycete phytopathology was restrained by taxonomic problems, essentially the lack of reliable criteria for species recognition, which led to considerable confusion as to the causal agents. Only in the last decade did things start to change slowly, with the adoption of methods for overall evaluation of reciprocal similarities and dissimilarities between the different microorganisms [Williams et al., 1983]. This approach has facilitated at least a preliminary clarification of the patterns characterising actinomycete pathoecology.

2. Pathology

With the exception of *Nocardia vaccinii*, all phytopathogenic actinomycetes so far recorded belong to the genus *Streptomyces* [Locci, 1989].

2.1 *Root crop diseases*

Economically root crop diseases are the most important ones incited by actinomycetes (Table 1). Up to a few years ago these comprised just potato scab and sweet potato soil rot (or pox). The situation was particularly unsatisfactory in the case of potato scab where distinct symptomatologies could be observed and phenotypically different streptomycetes isolated.

Table 1. Scab and rot diseases of root crops incited by actinomycetes

Disease	Agent	Host
Common scab	*Streptomyces scabies*	potato, red and sugar beet, carrot, parsnip, radish, rutabaga, turnip
Acid scab	*S. acidiscabies*	potato, table, fodder and sugar beets, carrot, radish, rutabaga, turnip
American russet scab	*S. aureofaciens*	potato
European russet (or netted) scab	*Streptomyces* spp.	potato
Soil rot (pox)	*S. ipomoeae*	sweet potato

2.1.1 *Common scab*

The disease is present in most potato growing areas of the world. Yield losses from severe infections have been reported, but usually just the grade quality of the tubers is affected. However, because of import regulations, in several countries the disease can represent a major economic problem. In addition the typical earthy odour of streptomycetes, caused by geosmin, can make the flesh inedible [Lechevalier, 1988].

Symptoms. A bewildering variety of symptoms has been described in the literature, possibly as a result of confusing diseases incited by different organisms. Roundish or star shaped lesions with cracked and torn edges are now recognised as typical of common scab. Cracks or furrows, 3–4 mm deep, may cut into the tuber when severe lesions coalesce [Lapwood, 1973].

Causal agents. Scab diseases of potatoes are incited by streptomycetes that historically have been associated with the *'Streptomyces scabies'* complex. Organisms causing common scab were first isolated by Thaxter [1891,

1892], characterised as melanin producers with grey spores borne in spiral chains and named *Oospora scabies*. No type culture was maintained. The species was subsequently transferred to the genus *Actinomyces* by Güssow [1914] and finally renamed *Streptomyces scabies* by Waksman and Henrici [1948]. Waksman [1961] redescribed the species, erroneously designating strain IMRU 3018 as the neotype strain. In fact this strain lacks spiral spore chains and does not produce melanin [Shirling and Gottlieb, 1968]. Further confusion was caused by the attribution of isolates from potato tubers to other *Streptomyces* species. More than thirty specific names are recorded by Bradbury [1986]. The situation has been clarified, at least in part, by the recent proposal to revive the name *Streptomyces scabies* for common scab strains from USA, Canada and Hungary [Lambert and Loria, 1989a]. The *S. scabies* strains were differentiated by a phenotypic analysis of 42 characters from atypical pathogenic strains, saprophytic streptomycetes isolated from scab lesions and reference strains of *S. griseus* and *S. tendae*. The strains form an homogeneous group characterised by smooth grey spores borne in spiral chains, melanin production and utilisation of all ISP [International Streptomyces Project, Shirling and Gottlieb, 1966] sugars. Most *S. scabies* isolates do not degrade xanthine and are susceptible to 25 μg^{-ml} of oleandomycin, 10 IU^{-ml} of penicillin G, 20 μg^{-ml} of streptomycin, 10 μg^{-ml} of thallium acetate and 0.5 μg^{-ml} of crystal violet. The type strain of *S. scabies* is ATCC 49173.

Histopathology and epidemiology. Penetration and infection by *S. scabies*, probably through stomata and young lenticels, have not been convincingly recorded. When first formed tuber internodes have stomata but, as the tissue expands, they are transformed into lenticels. Most workers agree with Fellows's [1926] suggestion that young lenticels can be infected. As the lenticels mature they become resistant, possibly because of the barrier formed by suberin deposits. Wounds may also be infection sites. Larval feeding aids initial penetration and progression through wound periderm layers.

The type of lesions, deep or shallow, is frequently determined by potato resistance or susceptibility. Resistance is apparently associated with the effectiveness of the periderm. In resistant cultivars a single periderm layer seems to prevent further infection, while in susceptible ones successive periderm layers form, as penetration progresses, resulting in deep scab lesions [Hooker, 1983]. According to Stein et al. [1993] penetration of cells is preceded by what appears to be enzymatic dissolution of walls.

It is possible to isolate *S. scabies* from affected tubers as well as from soil and there is some controversy as to whether scabby 'seed' or contaminated soil is more responsible for initiating the disease [Lapwood, 1973]. Undoubtedly the organism has been introduced into virtually all potato soils by infected tubers. Evidence exists however that pathogenic streptomycetes are present in some soils before potatoes are introduced. *S. scabies* is a

saprophytic pathogen able to survive for long periods on decaying plant parts in the soil and possibly on roots of living plants, in old feed lots and in fields heavily manured with animal wastes [Hooker, 1983].

The interaction of *S. scabies* and its potato host was examined under scab-conducive conditions in the greenhouse. In general disease severity increases linearly with an increase in log 10 of the inoculum, but both cultivar resistance and strain of *S. scabies* affected the slope of the regression line. Rhizosphere and rhizoplane populations of *S. scabies* also increase with increasing soil population. However rhizosphere population was not consistently related to scab severity, scab incidence, cultivar resistance or strain virulence [Keinath and Loria, 1991].

Zinc-inducible esterases and cutinase are produced by pathogenic *S. scabies* [McQueen and Schottel, 1987; Fett et al., 1992]. The production of phytotoxins (thaxtomins) and their role in pathogenicity will be discussed separately below.

Control. In practice preventive and control measures are directed against both seed and soil borne inocula. In addition potato varieties differ in their resistance to common scab and less susceptible cultivars are known.

Seed disinfection with mercuric chloride, formaldehyde, mancozeb (8%) and organo-mercuric compounds has been suggested [see Hooker, 1983]. Some of the compounds however are not permitted in certain countries. The practice is of doubtful value since the causal agents are present in most agricultural soils. Disinfection might be of some use when introducing the crop into virgin or irrigated desert soils [Lapwood, 1973].

In glasshouse tests single foliar sprays of the growth retardant 3,5-dichlorofenoxyacetic acid (3,5-D) and of a series of its analogues greatly decrease scab incidence. Some compounds show promising activity at very low dosages, without causing deformation of the tubers and/or distortion of foliage. In particular 2,5-dichlorobenzoic acid does not appear to affect scab as a downward-moving systemic bactericide, but to act by altering the response of the host to infection [McIntosh et al., 1981, 1985, 1988]. The modified host metabolism probably involves biosynthesis inhibition of phenolics, but not of terpenoids [Burrell, 1984].

Chemical treatment of soil greatly depends on the proper incorporation of the chemicals and is not effective in all soils. Formaldehyde, urea formaldehyde, manganese sulphate and pentachloronitrobenzene (PCNB) have been used, but only the latter, despite some disadvantages due to the possibly carcinogenic breakdown products, has been widely tested. Results are generally beneficial, at least in the first year of application.

Sulphur has been applied to reduce soil pH. Its use decreases common scab, but may involve some risks for acid sensitive rotation crops, such as barley, and possibly act as a screen for selection and enrichment of pathogens such as *S. acidiscabies*. Similarly organic manures have been used to decrease the *S. scabies* population by increasing soil acidity,

moisture and multiplication of antagonistic organisms. The costs of the operation is usually high and the practice does not seem to be effective when the soil population of *S. scabies* is already large [Hooker, 1983].

Liming soils that have a low pH increases the severity of common scab. There has been some dispute as to whether the calcium concentration in soil, rather than pH, is responsible for scab susceptibility. The point is not simply of academic relevance since increasing the Ca content of tubers may improve quality and reduce susceptibility to *Erwinia carotovora* subsp. *carotovora* soft rot and to other tuber disorders. According to some authors [see Goto, 1985] the content of exchangeable calcium is a more reliable parameter than the soil pH to evaluate the severity of potato scab. Experiments on field plots treated with dolomitic lime and gypsum [Lambert and Manzer, 1991] show on the other hand that scab incidence is correlated to soil pH ($P \leq 0.001$) and not Ca concentrations in soil, tuber periderm or medulla tissue and that in low pH soils higher tissue Ca concentration is an effect rather than a cause of increased scab.

The incidence of scab is greatest in dry soils and irrigation after tuber set effectively reduces the disease. The effect of moisture on infection, although known, remains unexplained [Lapwood, 1973]. Periods when *S. scabies* can infect tubers in dry soil coincide with unusually small ratios of bacterial to actinomycete populations, while in wet soils the ratio is reversed. Consequently it has been suggested that *S. scabies* infection could be prevented by enhanced growth of bacterial antagonists [Lewis, 1970]. Alternatively failure to colonise tubers in wet soil might be the result of the poor competitive ability of *S. scabies* in soils of comparatively low temperature and restricted oxygen supply [Adams and Lapwood, 1978]. In the practice incidence of the disease can be significantly reduced by irrigation during the first month after tuber initiation [Lapwood et al., 1970].

Biological control. Data on suppressive and conducive soils [Menzies, 1959] and on disease decline [Deacon, 1983], as an alternative to crop rotation, are available. Though theoretically enticing this strategy should be confronted with care. Applying biological control against soil organisms is not an easy task and controlling opportunistic pathogens in the same environment is even more difficult. Often what functions in the laboratory fails under field conditions and if it works it is difficult to reproduce. Schober [1984] suggests the use of strains of *Bacillus subtilis* antagonistic to *S. scabies*, however doubts are expressed even by the author about the practical use of the procedure. Hayashida et al. [1989] claim the efficacy of an antibiotic biofertilizer, produced from swine manure containing *S. albidoflavus* CH-33, which causes reduction in scab severity and increase in potato production. Viable counts of *S. scabies* in the soil decrease in 80 days with a corresponding increase in the *S. albidoflavus* population. Two suppressive isolates of *Streptomyces* spp. from potato scab research plots were shown to produce bacteriocin-like reactions to pathogenic isolates of

S. scabies [Liu and Anderson, 1992]. A three-year field experiment was made using pots set into soil. The two suppressive isolates were grown on vermiculite plus oatmeal broth and added (1.5 and 10%, v:v) to scab conducive soil. Treatment gave highly significant levels of disease control. The suppressive isolates did not affect yield and were identified as *S. scabies*, and *S. fulvoviolaceus*.

Other hosts. In addition to potato, common scab affects other root crops, listed in Table 1 [Hooker, 1983]. According to Hanson and Lacy [1990] the causal agent of carrot scab shows echinulate rather than smooth spore surfaces. In radish (*Raphanus sativus* L.) scab represents an economically important disease in commercial production of the crop. Symptoms appear as circular white lesions, 0.5–1.5 cm in diameter, with raised edges and sunken centres on the surface of expanding hypocotyls. Although scab lesions are generally restricted to surface tissues, infected radishes are unsalable. The etiology of the disease is rather puzzling. The organisms isolated from affected tissues differ morphologically and in growth characteristics from *S. scabies*. A scab potato isolate, used for comparison, was pathogenic for radish, while radish scab inducers were not able to affect potato tubers. The case obviously deserves further investigation [Levick et al., 1985].

2.1.2 *Acid (or uncommon) scab*

Common scab is generally controlled by maintaining the soil pH below 5.2. An indistinguishable form of the disease (acid or uncommon scab), which occurs in soils with pH values as low as 4.5, was first detected in Maine in 1953. Early outbreaks of this disease were often associated with a particular Midwestern seed lot of the very susceptible variety Chippewa, suggesting that the pathogen was not native to Maine. However there were also instances of the disease in farms which had not used outside seed sources. Discontinuation of harsh types of seed treatment in the early 1950s may have led to the built-up of indigenous pathogens to perceptible levels in such cases [Lambert and Loria, 1989b], as the acid tolerant species persists primarily on infected tubers rather than in soil [Manzer et al., 1977].

Strains of *Streptomyces* species causing the disease were isolated and described by Bonde and McIntyre [1968] and by Manzer et al. [1977], who found differences between typical and acid scab strains in pigmentation, spore chain morphology, raffinose utilisation and tolerance of low pH. Acid scab is now present in various areas of the Northeast and upper Midwest of the USA [Lambert and Loria, 1989b]. The organisms inducing acid scab are distinct from those causing common scab in phenotype and ecology and a new species, *S. acidiscabies*, has been proposed [Lambert and Loria, 1989b]. In culture *S. acidiscabies* differs from *S. scabies*, having flexuous

spore chains, a growth medium-dependent spore mass colour ranging from white to salmon-pink, a red or yellow pH-sensitive diffusible pigment and no melanin. *S. acidiscabies* grows on agar media at pH 4.0 (versus pH 5.0 for *S. scabies*), does not utilise raffinose as a carbon source, and tolerates higher concentrations of crystal violet (0.5 μg^{-ml}), thallium acetate (10 μg^{-ml}), streptomycin (20 μg^{-ml}), oleandomycin (25 μg^{-ml}) and penicillin G (10 IU^{-ml}) than *S. scabies*. The level of similarity to *S. scabies* is only 64% and no strong similarities can be found with the major *Streptomyces* groups of Williams et al. [1983], *S. griseoruber* being most similar (67%) to *S. acidiscabies* [Lambert and Loria, 1989b]. The type strain is ATCC 49003. Additional hosts of *S. acidiscabies* (Table 1) have been reported by Lambert [1991].

2.1.3 *Russet scab*

Russet scab has been reported in Europe and America since the beginning of the century [Harrison, 1962]. The causal agents were initially thought to be unfavourable environmental conditions, *Rhizoctonia solani* [Morse and Shapovalov, 1914] and finally soilborne streptomycetes, different from *S. scabies* [Harrison, 1962]. The disease is characterised by corky reticulations on the tuber surface. The infection is generally restricted to the skin affecting the quality of the crop. In Canada the disease does not reduce internal sales of potatoes, but severely affects exports [Faucher et al., 1993].

In contrast to common scab, the incidence of russet scab increases in moist soil. At the moment two types of russet scab are recognised.

2.1.3.1 *American russet scab.* Actinomycetes causing American Russet scab [Faucher et al., 1993] belong to the genus *Streptomyces* and are different from *S. scabies* (as they form pigmented mycelium, flexuous spore chains and no melanin) and from *S. acidiscabies* (mass spore colour, inability to grow at pH 4.5). The organisms are characterised by a bright yellow mycelium, which turns brown after about 2 weeks. The grey aerial mycelium has flexuous spore chains. The spore surface is smooth. The organisms do not produce melanin but degrade xanthine and xylan. Most strains utilise L-arabinose, D-fructose, D-glucose, D-mannitol, raffinose, rhamnose, sucrose and D-xylose and grow in the presence of NaCl (5%), tellurite (100 μg^{-ml}), penicillin (100 μg^{-ml}), phenol (0.1%) and oleandomycin (25 μg^{-ml}), but growth is inhibited by thallium (10 μg^{-ml}) and streptomycin (20 μg^{-ml}). By comparison with selected cluster groups of streptomycetes, as defined by numerical taxonomy [Williams et al., 1983], the russet scab-inducing organisms show the highest similarity level (78%) with Group 14 suggesting that they could be included in the *S. aureofaciens* cluster. With reference to the agents of common and acidic scab, the similarity level is 73% and 66% respectively.

2.1.3.2 *European russet (or netted) scab.* The organisms isolated by Harrison [1962] were not characterised in detail and therefore their specific identity remains uncertain. European russet scab is apparently a distinct disease from the American one [Sundheim, 1968; Bång, 1979; Scholte and Labruyère, 1985] with respect to cultivar susceptibility, root attack and optimum soil temperature. Scholte and Labruyère [1985] proposed the name of 'netted scab' for the former. No specific taxonomic investigations have so far been carried out on the causal streptomycetes.

2.1.4 *Relationships among scab inducers*

Relationships among the *Streptomyces* species causing potato scab have been investigated by DNA-DNA hybridisation [Healy and Lambert, 1991]. The levels of DNA relatedness among members of the major groups do not exceed 20% for any pair. Most *S. scabies* strains exhibit greater than 70% relatedness to the type strain, although values as low as 21% were obtained. The levels of homology between *S. scabies* and nonpathogenic type strains belonging to the *Diastatochromogenes* group range from 10 to 42%, while the reciprocal values obtained with labelled DNAs from the type strain of *S. bottropensis* (ATCC 25435) and of *S. eurythermus* (ATCC 14975) range from 37 to 74% and from 2 to 24%, respectively.

By convention, organisms which exhibit $\geq$ 70% DNA relatedness are considered members of the same species [Wayne et al., 1987]. While the characteristics of all of the cultures were consistent with the primary criteria for the species (spiral chains of smooth grey spores, use of all ISP sugars, melanin production), the strains which exhibited < 60% relatedness were significantly more likely ($P \leq 0.05$) to differ in secondary phenotypic traits. Pathogenic strains that differ from more typical *S. scabies* strains only in sugar utilisation patterns have been isolated and this may be another indicator of species diversity. Phenotypically, *S. scabies* most closely resembles the *S. diastaticus*, *S. cyaneus.* and *S. microflavus* groups of Williams et al. [1983]. Of the strains that exhibited low relatedness to the type culture, one was obtained from a russet (superficial) type of lesion, rather than a lesion with a typical raised or pitted appearance, while another differed from typical strains in the violet tinge of its grey spore colour. Thus, within the streptomycetes that are phenotypically consistent with *S. scabies*, pathogenicity would appear to occur in a subset of strains which have more genetic diversity than a conventional species. In addition, not all pathogenic strains can be differentiated from nonpathogenic strains on the basis of DNA relatedness data. By convention, pathogenic isolates obtained from tubers or roots have been attributed to *S. scabies* and saprophytes obtained from soil have been described as *S. diastatochromogenes*, which shares the same diagnostic characteristics [Shirling and Gottlieb, 1972]. At this point it is not possible to determine whether pathogenicity is an old trait which preceded divergence of these streptomycetes or whether pathogenicity is transferable among related organisms by some genetic mechanism.

In contrast to *S. scabies*, the levels of relatedness between *S. acidiscabies* isolates and their type strain were high in all cases. The levels of relatedness with all other species were ≤ 20% confirming the lack of close relationships between this species and other scab pathogens. This is consistent with the results obtained by Arias and Loria [1992]. In a search for monoclonal antibodies to *S. scabies* they identified a clone with high affinity for *S. scabies* which did not cross-react with *S. acidiscabies*. However the clone did cross-react with several of the nonpathogenic *Streptomyces* strains tested. Both *S. scabies* and *S. acidiscabies* produce the vivotoxin thaxtomin (see later), which induces scab symptoms and is associated only with streptomycetes that are pathogenic for potatoes. *S. acidiscabies* is also pathogenic for the other crop species attacked by *S. scabies*, a further indication that thaxtomin production may represent a primary pathogenicity mechanism in these two species. The toxin is unusual, and its production by unrelated species is an interesting example of either evolutionary convergence or genetic transfer.

The *S. albidoflavus* group, including *S. sampsonii*, *S. griseus* and the atypical common scab pathogens, appeared to be as diverse on the basis of DNA relatedness comparisons as on the basis of phenotypic comparisons. The pathogens belonging to this group are poorly understood. Until 1980, the putative type strain (strain IMRU 3018) and many or most of the other specimens deposited in culture collections as *S. scabies* were in fact flexuous-chained organisms that did not produce melanin. The strains which have been tested do not produce thaxtomin, although this ability might have been lost in culture.

A recent study on potato scab inducing agents in Israel [Doering-Saad et al., 1992] needs some comment, since it appears to overturn the patterns described above. In southern Israel scab became a limiting factor in potato production in the early 1960s. The disease was successfully controlled by improved irrigation techniques. However deep-pitted scab became a problem after 1981, even in well irrigated fields, and appeared to be incited by organisms different from *S. scabies*.

The results of a numerical taxonomy investigation, carried out on some thirty scab inducing organisms, demonstrated that potato scab in Israel is caused by a phenotypically diverse population of *Streptomyces* strains. The Israeli strains fell into different phena when analysed according to the classification of Kämpfert et al. [1991]. The majority of the pathogenic isolates were assigned to *S. violaceus* (57%) and *S. griseus* (22%), and some were allocated to *S. exfoliatus*, *S. rochei* and to single-member clusters. Reference strains of *S. scabies* and *S. acidiscabies*, obtained from R. Loria, were similarly recovered in different clusters and subclusters, though for some of them the probabilistic identification, carried out according to the scheme of Kämpfert and Kroppenstedt [1991], was admittedly ambiguous. The authors conclude that 'it appears likely that genes required for pathogenicity are spread by mobilization elements among

different *Streptomyces* species within their natural habitat'. The conclusion is more than acceptable, but it does not exclude that the scab inducing population might be a composite one and identifiable, in specific terms, with previously described agents of the disease in other parts of the world.

The Israeli situation is intriguing and could only be explained by admitting the existence in that habitat of a peculiar streptomycete population, an assumption however to be refuted on the grounds of the behaviour of the reference strains. The obvious consequence would be a complete revision of the pathological patterns recently established in other areas. Some alternatives however are worth noting. From the introduction of the paper by Doering-Saad et al. [1992], it appears that the *Streptomyces* population is a complex one, at first consisting of forms kept under control by irrigation (as happens in the case of *S. scabies*) and then of organisms able to thrive under moist soil conditions (like the agents of russet scab). In addition the authors stress the fact that strain micromorphology is diverse, some showing spiral, some flexuous and others straight spore chains. The same applies to spore ornamentation and to spore mass colour. No mention is made, but melanin production probably shows the same dissimilarity. None of these features however is taken into consideration by the authors either in their numerical study or for the probabilistic identification of the organisms, not even as additional characters to the 329 exclusively physiological ones employed. How far this approach is correct in the case of scab pathogens is at least debatable, since one of the principles of numerical taxonomy is that as many characters as possible [Sneath and Sokal, 1973] should be taken into account. Obviously to consider all is impossible, but clearly some groups of characters (morphology, cultural characteristics, etc.) should not be excluded *a priori*. A more comprehensive approach might show alternative relationships between the scab strains. Obviously further studies are needed to clarify this problem.

2.1.5 *Sweet potato soil rot (pox)*

Soil rot or pox is an economically important disease of sweet potato. It causes substantial reductions in yield as well as disfiguring lesions on the fleshy storage roots, which render them unmarketable [Clark and Matthews, 1987]. In Louisiana the causal agent, *S. ipomoeae*, ranks as a major pathogen [Kennedy and Alcorn, 1980]. The organism apparently persists for long periods, even in the absence of the host plant. A possible explanation is the fact that *S. ipomoeae* also causes disease in other *Convolvulaceae* which are frequent weeds of crops (such as soybeans and cotton) that are grown in rotation with sweet potato [Clark and Watson, 1983].

Symptoms. The soil rot disease effects are represented by dwarfed plants with little or no vine growth and small discoloured leaves. Many of the plants die before the end of the season. The diseased plants are easily pulled from the ground and the root system is very poorly developed, most

of the roots being entirely rotted and many of them breaking off when the plant is lifted from the soil. Small elongated, dark-coloured lesions may also be present on the stem below the soil line. The disease moreover is present on the mature potatoes in the form of pits or cavities with irregular jagged or roughened margins. The lesions may vary from one-quarter to more than an inch in diameter, sometimes coalescing and covering most of the surface of the potato. In the early stages of the disease the lesions are covered by the skin of the root, but this later breaks away, exposing the pits. These are slightly sunken and the new epidermal layer, apparently perfectly normal, is covered with the black, granular remnants of the old dead tissue. A potato infected while young, may be entirely girdled and as it continues to grow, may enlarge on each side of the infection centre and thus become badly misshapen [Person and Martin, 1940]. More recent studies have shown that isolates from Louisiana, North Carolina, California and Texas are identical according to ISP determinations [Shirling and Gottlieb, 1966], although they vary in aggressiveness, interaction phenotypes and plasmid profiles [Clark, 1992].

Causal agents. Apparently the first published mention of the 'soil rot' of sweet potato was made by Halsted [1890] who attributed it to the fungus *Acrocystis batatas*. The presence of an actinomycete, *Actinomyces poolensis*, was recorded by Taubenhaus [1918] who considered it a 'superficial wound parasite usually found following the pox spots produced by *Cytospora batatas*', the causal agent previously identified by Elliott [1916]. Manns and Adams [see Person and Martin, 1940], after restaining with carbol-fuchsin slides prepared by Elliott, discovered the constant presence of an actinomycete. Following further studies they proved that the disease was induced by an actinomycete, which they named *Actinomyces Pox.* Having compared it with a culture supplied by S.A. Waksman, Adams [1929] concluded that *Actinomyces poolensis* was non pathogenic. Following a thorough survey in Louisiana, Person and Martin [1940] were able to isolate and describe the causal agent, named *Actinomyces ipomoea.* The organism was subsequently transferred to the genus *Streptomyces* as *S. ipomoea* (Person and Martin) Waksman and Henrici and the spelling corrected to *S. ipomoeae* by Waksman [1957].

Histopathology and epidemiology. Histopathology of the root infection has been investigated by scanning and transmission electron microscopy [Clark and Matthews, 1987]. The actinomycete apparently does not produce appressoria or infection cushions analogous to those of most fungi. Hyphae of *S. ipomoeae* grow within host cells, often adjacent to the cell wall, without penetrating the host wall. Penetration of cell walls apparently occurs as a result of growth of lateral branches arising from the hyphae at certain sites where the hypha is in contact with the host wall. There appears to be some cell wall dissolution at these sites, suggesting that

enzymatic cell wall degradation may be an important, if not essential, penetration mechanism. Because channels in host walls are sometimes similar in appearance to host plasmodesmata, perhaps hyphae penetrate through them following enlargement of the plasmodesmata channel by enzymatic degradation of some of the surrounding cell wall.

The observation that *S. ipomoeae* is not restricted to the cortex of the roots, as are many other root-rotting pathogens, may partially explain the pathogen's ability to drastically reduce growth and yield of the plant. Destruction of the cortex may reduce selective absorption of water and nutrients, but destruction of the vascular system also reduces translocation to other parts of the plant. The sweet potato plant produces a complex root system that consists primarily of fibrous and storage roots [Kays, 1985]. Both root types are susceptible to the pathogen. Infections on storage roots originate from infected secondary fibrous roots. In the field the severity of disease on fibrous roots has an important effect on marketable yield, being positively correlated to the percentage of diseased storage roots and negatively correlated to the number of storage roots produced per plants. Yield of marketable storage roots is negatively correlated to both the severity of disease on fibrous roots and the percentage of diseased storage roots produced. These data demonstrate the importance of fibrous root disease in the particular pathosystem. Management strategies, that reduce disease on fibrous roots, may ultimately lead to increased yield of storage roots [Ristaino and Averre, 1992].

The severity of soil rot is modified very considerably by two environmental factors, the water content and hydrogen-ion concentration of the soil. A high or satisfactory water content of the soil stimulates a more rapid development of roots and also makes it possible for a diseased plant with a deficient root system to absorb the water and essential mineral salts more easily from the soil. In a rainy season, sweet potato plants affected with soil rot are able to make vines and often fairly satisfactory yields. In Louisiana, soil rot does not develop in soils of pH 5.2 or below [Person and Martin, 1940].

Serological assays for the diagnosis of *S. ipomoeae* have been proposed [Moyer and Echandi, 1984; Weicht et al., 1992] as well as greenhouse and laboratory methods for detecting host resistance [Moyer et al., 1982, 1984].

Control. Trials on the influence of irrigation, lowering of soil pH with sulphur and fumigation with Telone C-17 [Ristaino and Averre, 1992] show that irrigation reduces disease on fibrous roots to the greatest extent in plants not treated with sulphur or fumigated and increases the number of storage roots produced. Irrigation also reduces the number of diseased storage roots produced per plant in low rainfall years, but does not significantly increase yields.

Addition of sulphur reduces the severity of the disease on fibrous roots in non-fumigated plots, it also reduces yields, because of the lower number

of storage roots produced per plant. Fumigation reduces the percentage of diseased storage roots per plot, the number of diseased storage roots produced per plant and the severity of the disease on fibrous roots.

2.2 *Other crop diseases*

2.2.1 *Blueberry galls and bud proliferation*

This unusual disease was first observed in 1944 in Beltsville, Maryland in seedlings resulting from crossing two blueberry species, *Vaccinium australe* Small and *V. ashei* Reade, with an average incidence of 26% in lots totalling over 1,700 individuals [Demaree, 1947; Demaree and Smith, 1952].

Symptoms. Galls (½–2 inches in diameter) occur at or immediately below ground line and rarely on roots. Bud proliferations are of common occurrence. The abnormal buds generally abort at an early stage, others grow into thin, weak shoots, 1 to 6 inches high, forming a witches'-broom effect at the base of the plant.

Causal agents. The organisms, isolated from galls, were able to infect healthy blueberry plants reproducing the typical symptoms, but not azalea, impatiens, kalanchoe, strawberry and vinca. The pathogen resembled *Nocardia minima*. A culture of the latter however did not cause any abnormal growth in blueberry plants. In addition to pathogenicity, the isolated organisms, named *Nocardia vaccinii*, differ in their ability to utilise glycerol, mannitol and citrates and to reduce nitrates to nitrites. To our knowledge, notwithstanding the severity of the infection and the striking symptomatology, the disease has not been reported again in the last fifty years [Bradbury, 1986].

2.2.2 *Replant diseases*

'Replant disease' of apple seedlings and the related 'soil sickness' of more mature plants are apparently correlated to the colonisation of roots by actinomycetes. Both diseases are most severe when planting takes place in the same soils [Otto and Winkler, 1977]. Actinomycete hyphae are found within the root epidermal cells of diseased young apple seedlings. Large numbers of *Microbispora* sp. could be isolated from the root systems of affected plants and it was at first suggested that these organisms might be the cause of the problem [Westcott and Beer, 1983, 1985]. Later work [Westcott et al., 1987], however, showed that the isolates were not pathogenic.

The term 'apple replant disease' is used to refer to problems resulting in their delayed establishment on sites where apple trees had previously been planted. Symptoms include retarded growth, deterioration of young roots and inhibition of lateral root development during the first few years after

transplanting. The syndrome is not specific, but the etiology of some forms of the disease definitely involves biotic agents. Affected apple seedling roots show light brown superficial lesions, consistently colonised by filaments, about 1 μm in diameter. Ultrastructural features of the organisms, present in the lesions, indicate their prokaryotic similarity with actinomycetes. Apparently the pathogens are able to invade and damage living cells of the root epidermis and cortex. Severity of infection of roots is associated with inhibition of both apple seedling growth and lateral root development. Cytological evidence supports a hypothesis involving release of substances injurious to plant cells by the causal agents which however have not been isolated and identified [Westcott et al., 1987].

2.3 *Actinomycetes as forest pathogens*

Actinomycetes are reported to plug the xylem vessels of silver, sugar and Norway maples leading to early decay and dieback of the tree branches [Blanchette et al., 1981a,b; Wallis, 1983]. A variety of streptomycetes belonging to different species (*S. parvullus*, *S. sparsogenes*, *Streptomyces* sp.) were isolated from the plugs. The isolates are capable of growth within the tree vessels and *in vitro* in the presence of several phenols. Although sugar maples in the North-Eastern USA are routinely 'tapped' to collect maple sap for conversion to maple syrup, the mode of penetration of the actinomycetes into the host is not known.

Similarly a lignocellulose-degrading streptomycete (*S. flavovirens*) was found to decompose the intact cell walls of the phloem of Douglas fir, and hyphae were found in the cavities deriving from the destruction of the walls of the parenchyma and sclereids [Sutherland et al., 1979].

3. Phytotoxins and pathogenesis

One of the problems of host-parasite interaction is the elucidation of the mechanisms by which the pathogen is able to induce the development of lesions. Already in the mid 1920s Fellows [1926] noted in potato scab the darkening of tuber cells in advance of colonisation of the pathogen and suggested that this was a response to the action of a toxin or enzyme secreted by the scab organism. Later on Shoemaker [1952] succeeded in inducing scab lesions by transferring small agar blocks taken from the proximity of non sporulating colonies of *S. scabies* to the surface of tubers maintained under sterile conditions. Samples taken from the developed lesions failed to yield any actinomycetes. However the evidence for the induction of scab by a 'diffusable metabolic substance' of the pathogen was questioned when it was subsequently revealed that samples taken from the 'sterile' tubers showed the presence of bacterial and fungal contaminants [Lawrence et al., 1990].

In an attempt to provide evidence for the role of toxins in the etiology of the scab disease, aseptically cultured minitubers were inoculated with cell-free extracts from scab lesions of field-grown and cultured tubers infected with *S. scabies*. Typical symptoms of common scab (cell proliferation and expansion, eruption and browning of tissue, cracking of tuber surface), indistinguishable from those incited by the actual causal agent, were reproduced. Isolation and fractionation of the active components in tissue extracts, by a combination of normal and reversed phase thin-layer chromatography, yielded two active compounds, thaxtomin A and B, characterised as unique 4-nitroindol-3-yl containing 2,5-dioxopiperazines [King et al., 1989; Lawrence et al., 1990]. The phytotoxic principles satisfy basic requirements of vivotoxins and cannot be induced by physical or chemical injury or by inoculation with non pathogenic *Streptomyces* species.

The role of thaxtomins in the pathogenesis of *S. scabies* is supported by the correlation between susceptibility to the actinomycete and sensitivity to the phytotoxins. Susceptible potato cultivars develop extensive necrosis, extending in some cases into the vascular tissue of the tuber; resistant ones show necrotic flecks around the lenticel, with no evidence of damage in the vascular ring [Delserone et al., 1991]. An analysis of 23 isolates, obtained from scab infected potato tubers representative of 6 sampling areas in Eastern and Central Canada and 5 ATCC *Streptomyces* strains, showed that pathogenicity is positively correlated to the ability to produce thaxtomin on potato slices, thus stressing the importance of the compounds in the scab disease [King et al., 1991]. More recently [Bukhalid and Loria, 1993] a cosmid (pKC505) library of total genomic DNA from *S. scabies* was expressed in *S. lividans* TK24. Transformants were screened for the ability to colonise and produce necrosis on potato tuber slices. Three clones out of 2,000 produced a necrotic reaction similar to that of the pathogen type strain from which the library was constructed. Thin layer chromatography of the chloroform-soluble fractions of potato tuber tissue colonised by two of the clones produced a band that comigrated with thaxtomin A, the phytotoxin produced by *S. scabies* but not *S. lividans* TK24.

The number of thaxtomins has recently increased and five compounds (1 and 4 corresponding to thaxtomin A and B respectively) have been isolated and purified from *S.scabies* grown on surface sterilised potato slices [King et al., 1992] and on oatmeal agar and broth media [Babcock et al., 1993]. The ability to produce thaxtomin in agar and broth media has facilitated the purification of the compounds and the elucidation of the pathway for phytotoxin biosynthesis [Eckwall et al., 1992; Babcock et al., 1993]. Production of thaxtomin A, the major product, is repressed at least 130-fold in oatmeal broth medium supplemented with 0.5% glucose and by tryptophan and tyrosine, precursors of which may be involved in feedback inhibition of early steps in biosynthesis. Phytotoxins are secreted by the organism when the cells reach late exponential to early stationary phases of culture growth. The time at which each thaxtomin compound is produced

suggests a pathway for the latter steps in thaxtomin biosynthesis (Fig. 1). The ability to produce thaxtomins in large quantities provides the means for potentially using them as a screen to test potato cultivars for resistance to scab disease.

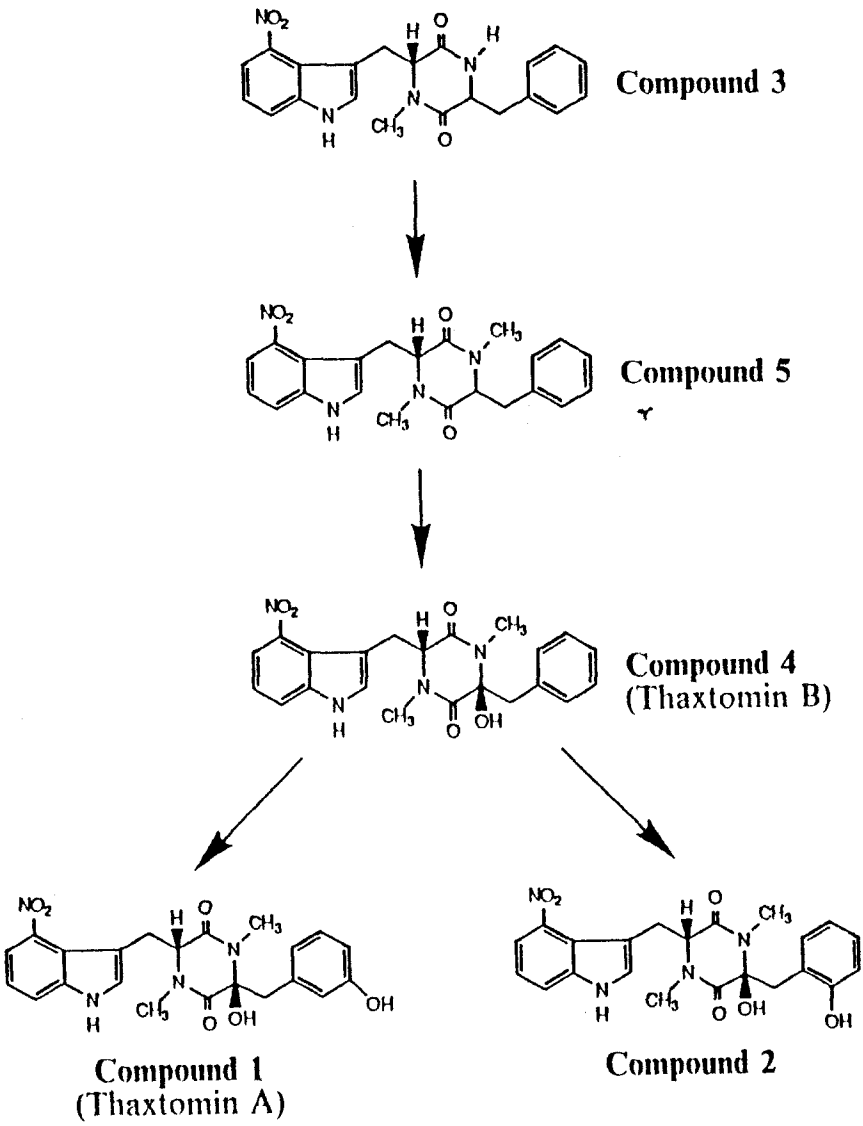

Fig. 1. Proposed pathway for the later stages of thaxtomin biosynthesis [from Babcock et al., 1993].

4. Conclusions

There is no doubt that phytopathogenic actinomycetes are enjoying a revival; after years of relative neglect, there has been in the last decade an upsurge of interest particularly with reference to the inducers of the different forms of potato scab. It has been argued that their incidence is of minor economical impact and that they often represent a problem of so-called 'cosmetic' phytopathology [Baker and Cook, 1974]. Actually potato scab can affect economically producing countries. The internal market is influenced by grading systems that reduce profits and export is hindered with obvious consequences. In addition scab inducing organisms have invaded every country where the potato is grown. The pathogens are extremely adaptable to different environments. Soil water content, pH, temperature are not obstacles to their diffusion independently of whether this capacity is overcome by different specific pathogens or by the spreading of pathogenicity genes within natural habitats. Reports show that potato is becoming a world food crop and progressing at very fast rates in the developing countries. In thirty years (1950–1980) annual production of potatoes in India has increased eight times. Another quite indicative parameter is the relative potato/rice price for the same years, nearly five times in favour of the potato crop on the Bangkok market [Niederhauser, 1993]. In these countries the scarcity and greater cost of high quality seed tubers may force self-dependence with all the relative phytopathological implications.

Turning now to the biology of the causal agents, notwithstanding the results obtained during the last ten years, the situation though improved is far from being completely clarified and taxonomic and ecological problems still exist. Taxonomy plays an important role in biology, not just as an labelling service, but as a complex and lively science involved in the unique task of integrating biological phenomena into a complete system [Baldacci and Locci, 1970]. It is a fundamental biological discipline in that it allows communication. The status of taxonomy is closely connected with the progress of knowledge so that any existing classification cannot be but a child of its time, reflecting in addition the purpose of the operation and the ability of the investigator. Consequently it is relative, provisional and imperfect and bound to be further developed, if not improved. The vicissitudes of the taxonomy of pathogenic actinomycetes, again exemplified most dramatically by the scab inducers, have already been illustrated. Particularly confusing was the situation following the transfer of the causative agents to the genus *Streptomyces*. This was not an unique trend but proceeded in parallel with that of typical saprophytic actinomycetes. Poor reproducibility of taxonomic characters led to a proliferation of the number of species described and made actinomycete taxonomy such a subjective exercise. While this was at least justifiable in practical terms, i.e., in order to fulfil patent requirements for antibiotic producers, it also 'contaminated' less

profit oriented areas. The habit of creating new species on the basis of few phenological divergences, invaded also streptomycete phytopathology. To restrict the case to the potato scab agents, a number of specific taxa have appeared in the literature, many of them trying to find justification by apparent relationships with saprophytic species whose taxonomic status was suffering from the same basic handicap. This led to the invalidation of *S. scabies* as species *incertae sedis* [Pridham and Tresner, 1974], because of the availability of 'many taxonomically different reference strains'. The revival of *S. scabies* by Lambert and Loria [1989a] appears commendable in that it has clarified, at least in part, the situation of common scab agents. However the possible existence of other less virulent streptomycetes, their role in the overall syndrome of the disease, the possibility of transferring pathogenicity genes between different species are still grey areas awaiting clarification. The fact that *S. scabies* resides in soil where the actinomycete community is numerous and active does represent a pathoecological complication. In addition comparison with previously isolated organisms is not always feasible and reliable, due to the possible loss of pathogenicity in culture.

The recent discovery of phytotoxin production by strains of *S. scabies* and *S. acidiscabies* deserves a final comment. Thaxtomins are potentially very promising tools not only for understanding the dynamics of pathogenesis, but also for screeening pathogen virulence and host susceptibility.

Note

1. The present review deals with phytopathogenic actinomycetes *sensu stricto*, that is with the exclusion, e.g., of the organisms previously classified as plant pathogenic 'corynebacteria' [Locci et al., 1989; Firrao and Locci, 1989a,b].

References

Adams JF (1929) An actinomycete, the cause of soil rot or pox in sweet potatoes. Phytopathology 19: 179–190

Adams MJ and Lapwood DH (1978) Studies on the lenticel development, surface microflora and infection by common scab (*Streptomyces scabies*) of potato tubers growing in wet and dry soils. Ann. Appl. Biol. 90: 335–343

Arias I and Loria R (1992) Production of monoclonal antibodies to *Streptomyces scabies*. Phytopathology 82: 241 (Abstr.)

Babcock MJ, Eckwall EC and Schottel JL (1993) Production and regulation of potato-scab-inducing phytotoxins by *Streptomyces scabies*. J. gen. Microbiol. 139: 1579–1586

Baker KF and Cook RJ (1974) Biological Control of Plant Pathogens. W.H. Freeman & Co., San Francisco

Baldacci E and Locci R (1970) Micromorphology of *Actinomycetales* and their genera arrangement. In: Prauser H (ed.) The Actinomycetales (pp. 419–424) G. Fischer Verlag, Jena

Bång H (1979) Studies on potato russet scab. 1. A characterization of different isolates from northern Sweden. Acta Agric. Scand. 29: 145–150

Blanchette RA, Sutherland JB and Crawford DL (1981a) Actinomycetes in discolored wood of living silver maple. Can. J. Botany 59: 1–7

Blanchette RA, Sutherland JB and Crawford DL (1981b) The role of actinomycetes in the discoloration and decay process of living silver maple trees. Phytopathology 71: 204 (Abstr.)

Bonde MR and McIntyre GA (1968) Isolation and biology of a *Streptomyces* sp. causing potato scab in soils below pH 5.0. Am. Potato J. 45: 273–278

Bradbury JF (1986). Guide to Plant Pathogenic Bacteria. CAB International Mycological Institute, Kew

Bukhalid RA and Loria R (1993) Cloning of a pathogenicity factor from *Streptomyces scabies*. Phytopathology 83: 1360 (Abstr.)

Burrell MM (1984) Inhibition of browning, phenoxyacetic acids and phenolic metabolism in potato tuber discs: a model system to study chemicals that control common scab. Plant Pathology 33: 325–336

Clark CA (1992) Variability of *Streptomyces ipomoeae* for cultural characteristics, pathogenicity, interaction phenotypes, and plasmid profiles. Phytopathology 82: 1125 (Abstr.)

Clark CA and Matthews SW (1987) Histopathology of sweet potato root infection by *Streptomyces ipomoea*. Phytopathology 77: 1418–1423

Clark CA and Watson B (1983) Susceptibility of wood species of *Convolvulaceae* to root-infecting pathogens of sweet potato. Plant Disease 69: 907–909

Deacon JW (1983) Microbial Control of Plant Pests and Diseases. American Society for Microbiology, Washington, DC

Delserone LM, Loria R and Arias I (1991) Correlation between susceptibility of potato cultivars to *Streptomyces scabies* and sensitivity to thaxtomins. Phytopathology 81: 1193 (Abstr.)

Demaree JB (1947) A proliferating gall on blueberry plants caused by an *Actinomyces*. Phytopathology 37: 438 (Abstr.)

Demaree JB and Smith NR (1952) *Nocardia vaccinii* n. sp. causing galls on blueberry plants. Phytopathology 42: 249–252

Doering-Saad C, Kämpfer P, Manulis S, Kritzman G, Schneider J, Zakrzewska-Czerwinska J, Schrempf H and Barash I (1992) Diversity among *Streptomyces* strains causing potato scab. Appl. Environ. Microbiol. 58: 3932–3940

Eckwall EC, Babcock MJ and Schottel JL (1992) Production of the common scab phytotoxin, thaxtomin A, by *Streptomyces scabies*. Phytopathology 82: 1153 (Abstr.)

Elliott JA (1916) The sweet potato 'soil rot' or 'pox', a slime mold disease. Del. Agric. Exp. Stn. Bull. 114

Faucher E, Otrysko B, Paradis E, Hodge NC, Stall RE and Beaulieu C (1993) Characterization of streptomycetes causing russet scab in Québec. Plant Disease 77: 1217–1220.

Fellows H (1926) Relation of growth in the potato tuber to the potato scab disease. J. Agric. Res. 32: 757–781

Fett WF, Gerhard HC, Moreau RA, Osman SF and Jones LE (1992) Cutinase production by *Streptomyces* spp. Curr. Microbiol. 25: 165–171

Firrao G and Locci R (1989a). Identification by probabilistic methods of plant pathogenic corynebacteria. Ann. Microbiol. 39: 81–92

Firrao G. and Locci R (1989b) Diagnosis of the potato ring rot agent by DNA dot-blot hybridization using non-radioactive probes. In: Klement Z (ed.) Plant Pathogenic Bacteria (pp. 589–592) Akadémiai Kiadó, Budapest

Goto K (1985) Relationships between soil pH, available calcium and prevalence of potato scab. Soil Sci. Plant Nutr. 31: 411–418

Güssow HT (1914) The systematic position of the organism of the common potato scab. Science 39: 431–432

Halsted BD (1890) Some fungous diseases of sweet potato. N.J. Agric. Exp. Stn. Bull. 76 [after Person and Martin, 1940]

Hanson LE and Lacy ML (1990) Carrot scab caused by *Streptomyces* spp. in Michigan. Plant Disease 74: 1037

Harrison MD (1962) Potato russet scab, its cause and factors affecting its development. Am. Potato J. 39: 368–387

Harz CO (1877) *Actinomyces bovis* ein neuer Schimmel in dem Geweben des Rindes. Deutsche Zeitschrift für Thiermedizin 5: 125–140

Hayashida S, Choi MY, Nanri N, Yokoyama M and Uematsu T (1989) Control of potato common scab with an antibiotic biofertilizer produced from swine feces containing *Streptomyces albidoflavus* CH-33. Agric. Biol. Chem. 53: 349–354

Healy FG and Lambert DH (1991) Relationships among *Streptomyces* spp. causing potato scab. Int. J. Syst. Bacteriol. 41: 479–482

Hooker WJ (1983) Common scab. In: Hooker WJ (ed.) Compendium of Potato Diseases (pp. 33–34) American Phytopathological Society, St. Paul

Kämpfert P and Kroppenstedt RM (1991) Probabilistic identification of streptomycetes using miniaturized physiological tests. J. gen. Microbiol. 137: 1893–1902

Kämpfert P, Kroppenstedt RM and Dott W (1991) A numerical classification of the genus *Streptomyces* and *Streptoverticillium* using miniaturized physiological tests. J. gen. Microbiol. 137: 1831–1891

Kays SJ (1985) The physiology of yield in the sweet potato. In: Bouwkamp JC (ed.) Sweet Potato Products. A Natural Resource for the Tropics (pp. 79–132) CRC Press, Boca Raton

Keinath AP and Loria R (1991) Effects of inoculum density and cultivar resistance on common scab of potato and population dynamics of *Streptomyces scabies*. Amer. Potato J. 68: 515–524

Kennedy BW and Alcorn SM (1980) Estimates of US crop losses to procaryote plant pathogens. Plant Disease 64: 674–676

King RR, Lawrence CH and Calhoun LA (1992) Chemistry of phytotoxins associated with *Streptomyces scabies*, the causal organism of potato common scab. J. Agric. Food Chem. 40: 384–387

King RR, Lawrence CH and Clark MC (1991) Correlation of phytotoxin production with pathogenicity of *Streptomyces scabies* isolates from scab infected potato common tubers. Amer. Potato J. 68: 675–680

King RR, Lawrence CH, Clark MC and Calhoun LA (1989) Isolation and characterization of phytotoxins associated with *Streptomyces scabies*. J. Chem. Soc. (London) 13: 849–850

Lambert DH (1991) First report of additional hosts for the acid scab pathogen *Streptomyces acidiscabies*. Plant Disease 75: 750 (Abstr.)

Lambert DH and Loria R (1989a) *Streptomyces scabies* sp. nov., nom. rev. Int. J. Syst. Bacteriol. 39: 387–392

Lambert DH and Loria R (1989b) *Streptomyces acidiscabies* sp. nov. Int. J. Syst. Bacteriol. 39: 393–396

Lambert DH and Manzer FE (1991) Relationship of calcium to potato scab. Phytopathology 81: 632–636

Lapwood DH (1973) *Streptomyces scabies* and potato scab disease. In: Sykes G and Skinner FA (eds.) Actinomycetales: Characteristics and Practical Importance (pp. 253–260) Academic Press, London

Lapwood DH, Wellings LW and Rosser WR (1970) The control of common scab of potatoes by irrigation. Ann. Appl. Biol. 66: 397–405

Lawrence CH, Clark MC and King RR (1990) Induction of common scab symptoms in aseptically cultured potato tubers by the vivotoxin, thaxtomin. Phytopathology 80: 606–608

Lechevalier MP (1988) Actinomycetes in agriculture and forestry. In: Goodfellow M, Williams ST and Mordarski M (eds.) Actinomycetes in Biotechnology (pp. 327–358) Academic Press, San Diego

Levick DR, Evans TA, Stephens CT and Lacy ML (1985) Etiology of radish scab and its control through irrigation. Phytopathology 75: 568–572

Lewis BG (1970) Effects of water potential on infection of potato tubers by *Streptomyces scabies* in soil. Ann. Appl. Biol. 66: 83–88

Liu D and Anderson N (1992) Biological control of potato scab with suppressive isolates of *Streptomyces*. Phytopathology 82: 1108 (Abstr.)

Locci R (1989) Section 29. Streptomycetes and related genera. In: Williams ST, Sharpe ME and Holt JG (eds.) Bergey's Manual of Systematic Bacteriology (pp. 2451–2452) Williams & Wilkins, Baltimore

Locci R, Firrao G, Petrolini B and Sardi P (1989) Numerical taxonomy of phytopathogenic corynebacteria. Ann. Microbiol. 39: 59–79

Manzer FE, McIntyre GA and Merriam DC (1977) A new potato scab problem in Maine. Maine Agric. Exp. Stn. Bull. 85: 1–24

McIntosh AH, Bateman GL, Chamberlain K, Dawson GW and Burrell MM (1981) Decreased severity of potato common scab after foliar sprays of 3,5-dichlorophenoxyacetic acid, a possible antipathogenic agent. Ann. Appl. Biol. 99: 275–281

McIntosh AH, Chamberlain K and Dawson GW (1985) Foliar sprays against potato common scab: compounds related to 3,5-dichlorophenoxyacetic acid. Crop Protection 4: 473–480

McIntosh AH, Bateman GL and Chamberlain K (1988) Substituted benzoic and picolinic acids as foliar sprays against potato common scab. Ann. Appl. Biol. 112: 397–401

McQueen DAR and Schottel JL (1987) Purification and characterization of a novel extracellular esterase from pathogenic *Streptomyces scabies* that is inducible by zinc. J. Bacteriol. 169: 1967–1971

Menzies JD (1959) Occurrence and transfer of a biological factor in soil that suppresses potato scab. Phytopathology 49: 648–652

Morse WJ and Shapovalov M (1914) The rhizoctonia disease of potatoes. Maine Agric. Exp. Stn. Bull. 230 [after Faucher et al., 1993]

Moyer JW and Echandi E (1984) Serodiagnosis of *Streptomyces ipomoea*-infected sweet potato roots. Phytopathology 74: 630–631 (Abstr.)

Moyer JW, Campbell CL, Echandi E and Collins WW (1982) Greenhouse and laboratory methods for identifying resistance in sweet potato to *Streptomyces ipomoea*. Phytopathology 72: 990

Moyer JW, Campbell CL, Echandi E and Collins WW (1984) Improved methodology for evaluating resistance in sweet potato to *Streptomyces ipomoea*. Phytopathology 74: 494–497

Niederhauser JS (1993) International cooperation in potato research and development. Ann. Rev. Phytopathol. 31: 1–21

Otto G and Winkler H (1977) Investigations about cause of specific replant disease of fruit trees. Zbl. Bakt. Par. Infekt. Hyg. II Abt. 132: 593–606

Person LH and Martin WJ (1940) Soil rot of sweet potato in Louisiana. Phytopathology 30: 913–926

Pridham TG and Tresner HD (1974) Genus I. *Streptomyces* Waksman and Henrici 1943, 339. In: Buchanan RE and Gibbons NE (eds.) Bergey's Manual of Determinative Microbiology. VIII ed. (pp. 748–829) Williams & Wilkins, Baltimore

Ristaino JB and Averre CW (1992) Effects of irrigation, sulfur, and fumigation on *Streptomyces* soil rot and yield components in sweetpotato. Phytopathology 82: 670–677

Schober B (1984) Kartoffelschorf. Information zum integrierte Pflanzenschutz. Nachrichtenblatt des Deutschen Pflanzschutzdienstes 36: 89

Scholte K and Labruyère RE (1985) Netted scab: a new name for an old disease in Europe. Potato Res. 28: 443–448

Shirling EB and Gottlieb D (1966) Methods of characterization of *Streptomyces* species. Int. J. Syst. Bacteriol. 16: 313–340

Shirling EB and Gottlieb D (1968) Cooperative description of type cultures of *Streptomyces*.

III. Additional species description from first and second studies. Int. J. Syst. Bacteriol. 18: 279–392

Shirling EB and Gottlieb D (1972) Cooperative description of type strains of *Streptomyces*. V. Additional descriptions. Int. J. Syst. Bacteriol. 22: 265–394

Shoemaker RA (1952) *Streptomyces scabies* and its relationship to common scab of potato. Ph.D. Thesis, University of Toronto

Sneath PHA and Sokal RR (1973) Numerical Taxonomy: The Principles and Practice. W.H. Freeman & Co., San Francisco

Stein B, Hammerschmidt R and Douches D (1993) Ultrastructure of *Streptomyces scabies* infection of *in vitro* cultured potato tubers. Phytopathology 83: 1414 (Abstr.)

Sundheim L (1968) *Streptomyces* sp. causing russet scab of potatoes in Norway. Eur. Potato J. 11: 194

Sutherland JB, Blanchette RA, Crawford DL and Pometto AL (1979) Breakdown of Douglas fir phloem by a lignocellulose degrading *Streptomyces*. Curr. Microbiol. 2: 123–126

Taubenhaus JJ (1918) Pox, or pit (soil rot) of the sweet potato. J. Agric. Res. 13: 437–450

Thaxter R (1891) The potato scab. Conn. Agric. Exp. Stn. Rept. 1890: 81–95

Thaxter R (1892) Potato scab. Conn. Agric. Exp. Stn. Rept. 1891: 153–160

Waksman SA (1957) Family *Actinomycetaceae* Buchanan and family *Streptomycetaceae* Waksman and Henrici. In: Breed RS, Murray EGD and Smith (eds.) Bergey's Manual of Determinative Microbiology. VII ed. (pp. 744–825) Williams & Wilkins, Baltimore

Waksman SA (1961) The Actinomycetes. Volume II. Classification, Identification and Description of Genera and Species. Williams & Wilkins, Baltimore

Waksman SA and Henrici AT (1948) Family II. *Actinomycetaceae* Buchanan and family *Streptomycetaceae* Waksman and Henrici. In: Breed RS, Murray EGD and Hitchens AP (eds.) Bergey's Manual of Determinative Microbiology. VI ed. (pp. 892–980) Williams & Wilkins, Baltimore

Wallis G (1983) Actinomycetes and actinomycete-like vessel occlusions of sugar maple, *Acer saccharum*. MS thesis, SUNY Coll. Env. Sci. & Forestry, Syracuse, NY, 51 pp. [after M.P. Lechevalier, 1988]

Wayne LG, Brenner DJ, Colwell RR, Grimont PAD, Kandler O, Krichevsky MI, Moore LH, Moore WEC, Murray RGE, Stackebrandt E, Starr MP and Trüper HF (1987) Report of the *ad hoc* committee on reconciliation of approaches to bacterial systematics. Int. J. Syst. Bacteriol. 37: 463–464

Weicht TR, Moyer JW and Restaino JB (1992) Detection of *Streptomyces ipomoea* in mixed cultures, soil, and sweetpotato fibrous roots with a serological assay. Phytopathology 82: 1145 (Abstr.)

Wescott SW and Beer SV (1983) Association of an actinomycete with apple replant disease. Phytopathology 72: 1348 (Abstr.)

Wescott SW and Beer SV (1985) Association of *Microbispora* spp. with roots of apple seedlings. Phytopathology 75: 1290 (Abstr.)

Wescott SW, Beer SV and Israel HW (1987) Interactions between actinomycete-like organisms and young apple roots grown in soil conducive to apple replant disease. Phytopathology 77: 1071–1077

Williams ST, Goodfellow M, Alderson G, Wellington EMH, Sneath PHA and Sackin MJ (1983) Numerical classification of *Streptomyces* and related genera. J. gen. Microbiol. 129: 1743–1813