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PLANT DISEASE FEATURE

Streamlining Global Germplasm Exchange: Integrating Scientific Rigor and Common Sense to Exclude Phantom Agents from Regulation

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Abstract

This collaborative work by over 180 researchers from 40+ countries addresses the challenges posed by "phantom agents"—putative pathogenic agents named in literature without supporting data on their existence. Those agents remain on regulatory lists, creating barriers in trade and plant certification. Historically identified based solely on symptoms, these agents lack isolates or sequence data, making reliable detection or risk assessment impossible. After reviewing over 120 such agents across 10 key plant genera, we recommend their removal from regulatory lists and call for revised standards aligned with modern diagnostics. This effort seeks to streamline germplasm exchange, benefiting global agriculture by removing the constraints imposed by phantoms.

Keywords: fruit, ornamentals, small fruits, tree fruits, viruses and viroids

Introduction

Plant virology was established as a discipline in the late nineteenth century after the discovery of a novel entity causing tobacco mosaic disease described as "*contagium vivum fluidum*" (Beijerinck 1898). Since then, the field has undergone tremendous progress in understanding plant viruses and the diseases they cause. Accurate laboratory diagnostic tools, developed for the detection and identification of viruses and other systemic pathogens (e.g. viroids, phytoplasmas, and fastidious bacteria) in plants, have advanced our understanding of pathogen-vector-host interactions and are regularly employed in the production of plants that are free from regulated pathogens. Clean plants are crucial for the sustainable production of clonally propagated crops (Gergerich et al. 2015; Hammond et al. 2023), facilitating the safe exchange of plant material globally, and are often the basis of national or regional certification programs. Although accurate diagnostic tools are crucial for certification programs and for facilitating the safe exchange of plant material, current lists of regulated pathogens contain several "phantom" agents, which impede access to that material. Phantom agents have been associated with symptomatic plants and diseases of unknown etiology. Most are presumed to be of viral nature, yet (i) neither infected plant material nor reference isolates are available and (ii) no sequence information is accessible. The majority of the phantom agents listed among regulated pathogens are the result of a single report that describes symptoms and/or names a purported pathogen in the scientific literature. It is worth noting that many of the agents discussed herein have only been reported in scientific conference abstracts and proceedings and have not gone through the peer-review process. It is often futile to state the obvious, but for the purpose of this communication, it is important to remember that diagnosing a disease or detecting an agent that is alleged to exist but might not be real is impossible. Biological indexing is, other than in a very few exceptions, used to identify phantom agents. However, biological indexing is not the most appropriate of all available diagnostics technologies for clonally propagated crops, including those covered herein (Al Rwahnih et al. 2015; Bester et al. 2021; Rott et al. 2017; Villamor et al. 2022). However, when symptoms develop in grafted indicator plants, it is often impossible to determine whether the indexed agent is one of the so-called phantoms, owing to the absence of a positive control. With today's technology, if an indicator plant shows symptoms, it would undergo analysis by high-throughput sequencing (HTS). If this process identifies a novel agent, it is unlikely to be attributed to a phantom. Instead, it would be recognized as a new pathogen of the host. As a result, phantom agents tend to persist indefinitely. In the case of phantom agents and the disease they were named for, it is impossible to determine whether the disease is real and, if so, whether it is caused by a single or multiple agents or an agent known under a different name or even whether the said agent has been eliminated from the crop because of extensive sanitation and testing through certification programs, or the incorporation of resistance in modern cultivars. Subject experts from around the globe joined efforts in reviewing the relevant literature in their areas of expertise and prepared a list of phantom agents/diseases for 10 vegetatively propagated plant genera, grouped in eight crops. Based on our collective assessment and knowledge, we recommend that phantom agents and diseases be removed from regulatory lists based on the inability to (i) access diseased plant material/identify an agent (isolate) and (ii) obtain sequence information that would allow for their identification and the development of diagnostics. When we initiated this project, the core group of individuals named as the primary coauthors identified more than 140 agents. Communications with individuals from around the globe have triggered research efforts that have led to the identification of type isolates for some of these agents and characterization of others. A list of characterized agents is provided in Table 1. Herein, we provide information on more than 120 phantom agents/diseases for major clonally propagated crops and discuss why requiring their diagnosis from a biological perspective is unsound. Every disease/agent discussed herein fits the aforementioned definition of a phantom agent.

Citrus

Algerian navel orange virus

Algerian navel orange virus was reported in Algerian navel orange (*C. sinensis* [L.] Osbeck) trees in Florida, U.S.A. The suspected trees did not exhibit symptoms. Rather, a virus was presumed to be present based on symptoms consistently observed in indexed herbaceous indicator plants (Garnsey 1975). Mechanical inoculation of citrus using the material from the indexed herbaceous hosts did not result in symptoms. However, the agent from the inoculated citrus trees could be back-inoculated to herbaceous hosts with the development of similar symptoms as the original indicators. No further studies have been performed since 1975.

Bark pitting (inverse pitting)

Bark pitting (inverse pitting) was reported on satsuma mandarin (*Citrus unshiu* Marcow) in Japan (Tanaka and Yamada 1961). Affected trees were severely defoliated and exhibited pits on the cambial side of the bark and protruding pegs on the wood side. No research has been published since the 1960s.

Brittle twig yellows

Brittle twig yellows of sweet orange was reported to be graft transmissible in Iran. Affected trees were stunted, bushy, and yellowed with easily breakable, thick, and atypically branched shoots that exhibited symptoms with certain commonalities to citrus stubborn disease (*Spiroplasma citri*) and tristeza stem pitting (citrus tristeza virus) diseases, along with some distinguishing characteristics (Samadi et al. 1977). No further research has been published since the original report.

Citrus yellow mottle

Citrus yellow mottle was reported in Japan on satsuma mandarin trees, which developed distinct vein clearing with yellowish halos (Ushiyama et al. 1984). The disease could be transmitted to other citrus species by grafting and, occasionally, mechanically. Rod-shaped virus particles were detected in electron microscopy preparations from symptomatic leaves. The suspected viral agent was termed citrus yellow mottle virus. In later experiments, larger filamentous particles, similar to citrus leaf blotch virus particles, were observed. However, the disease has not been reported since, and the original isolates were lost, so this work was inconclusive (Toru Iwanami, *personal communication*). It is important to highlight here that the citrus yellow mottle–associated virus, with elongated flexuous particles, and size similar to mandariviruses, which was recently reported from Pakistan by Wu et al. (2020), was not

derived from the "citrus yellow mottle" materials, and the two are not synonymous.

Failure of rangpur lime on sweet orange

A failure of rangpur lime and citron (*C. medica* L.) scions grafted on sweet orange rootstocks was reported in California, U.S.A. (Frolich 1958; Frolich and Hodgson 1961). The growth of the sweet orange rootstock was greatly restricted, in comparison with the scion. The disorder was graft transmissible to rangpur lime plants grafted on the sweet orange rootstock, but not on rangpur lime scions grafted on the sour orange rootstocks or rooted rangpur lime cuttings. There have not been reports on the disease since the 1960s.

Fovea

The fovea disorder was reported in Florida on 'Murcott tangor' and other mandarin hybrids and was manifested as tree decline and inverse stem pitting (Timmer et al. 2000). The reported symptoms were like those observed in 'Murcott' infected by cachexia viroid (Timmer et al. 2000), but no causality was proved. There have been no further reports of fovea.

Grapefruit bark scaling

A disorder with bark scaling symptoms was reported on grapefruit (*C. paradisi* MacFadyen) in Florida. No pathogen has ever been associated with the disorder and graft-transmission tests did not yield any results (Timmer et al. 2000), and, to the best of our knowledge, reference isolates are not available.

Gum pocket, gummy pitting, and wood pitting

Gum pocket, gummy pitting, and wood pitting refer to a syndrome reported in various citrus-growing regions around the world, on trifoliate orange (*Poncirus trifoliata* [L.] Raf.) rootstocks. Symptoms were gum-filled pits in the trunk with gumming observed in both the xylem and phloem. These disorders were initially correlated with viroid infection (Duran-Vila et al. 2002; van Vuuren and da Graça 1996), yet follow-up research failed to confirm the association of viroids with the disease symptom. It is possible that viroids enhance these disorders; physiological or stress conditions may be their root cause (Duran-Vila et al. 2002; Vernière et al. 2002, 2004).

Leaf curl

Leaf curling, dieback of branches, and decline were reported on a small number of citrus trees in Brazil (Salibe 1959, 1965). The disorder was graft transmissible to other citrus species, where it reproduced the symptoms. Since the 1960s, no other studies have been performed.

Leathery leaf

A disorder with leathery leaf symptoms was reported in India (Ahlawat et al. 1979). The disorder was mechanically, graft, and aphid transmissible. The reported symptoms resembled those caused by satsuma dwarf virus (SDV) and psorosis-like vein clearing (Timmer et al. 2000). The disorder has not been reported since.

Milam lemon stem pitting

A disorder with severe stem pitting symptoms, similar to those associated with citrus tristeza virus (CTV), was reported in Milam lemon (*Citrus jambhiri* Lush) in Florida (Garnsey 1973). However, these symptoms were also observed in trees where CTV was not detected. Disease transmission trials were inconclusive. There have been no further reports of Milam lemon stem pitting.

Multiple sprouting

Multiple sprouting with dense proliferation of shoots was a graft-transmissible disorder observed in a 'Joppa' sweet orange tree in South Africa in 1970 (Timmer et al. 2000). The citrus multiple sprouting virus (Majorana and Schwarz 1972) was associated with this disorder but never confirmed as the causal agent. Multiple sprouting has also been reported as one of the symptoms of SDV infection in satsuma mandarin trees in Turkey (Önelge and Çinar 2010). However, SDV has not been established as the cause of the multiple sprouting disorder observed in South Africa. The original isolate has been lost (John daGraça, personal communication) and the disorder has not reappeared in the field (Glynnis Cook, *personal communication*).

Rubbery wood

A graft-transmissible disorder was reported in India in limes (*C. aurantifolia* [Christm.] Swingle) and lemons (Ahlawat and Chenulu 1985). The symptoms were atypically flexible and downward-bent tree limbs (Ahlawat and Pant 2003). The disorder was associated with the presence of a phytoplasma. However, although phytoplasmas have been present in citrus groves, rubbery wood has been absent for many years (Garnier et al. 1991; Ghosh et al. 1999, 2013). There have not been additional efforts to characterize the causal agent of rubbery wood, and given the lack of a reference isolate, further characterization of this disorder is not possible (Dilip Ghosh, *personal communication*).

Shell bark

Shell bark was reported in Eureka and Lisbon lemon trees in Australia and Argentina (Fernández Valiela 1961; Olson 1968). These symptoms were later reported in other countries and in different citrus hosts such as orange and grapefruit (Timmer et al. 2000). Specific symptoms were not described in detail in these reports, but bark shelling usually signifies cracking and peeling of the bark. The disorder was often associated with one or more viroids, primarily, citrus exocortis viroid (CEVd) (Olson 1968). Shell bark could have been caused by a mixture of viroids, including CEVd, yet this is only a hypothesis, and the causal agent was not established. In Australia, when viroids were eliminated through nucellar embryony, shell bark ceased being a problem in lemons (Broadbent and Dephoff 1992). There have not been recurring reports of this disorder.

Tarocco pit

An atypical disease, characterized by significant concavities or inferior depressions in the wood, was reported on 'Tarocco' orange (*C. sinensis* [L.] Osbeck) trees in Sicily, Italy (Russo and Klotz 1963). Circular cavities filled with a cork-like material were observed in the center of these depressions. Oak-leaf pattern and leaf flecking, which are also symptoms of concave gum disease, were observed on the affected trees. Upon graft inoculation with budwood from the affected trees, sweet orange indicators developed leaf flecking and oak-leaf pattern. However, they did not develop the pits observed on 'Tarocco' trees. The authors stated: "It was evident that concave gum virus was present in the affected Tarocco trees." Consequently, they suggested that a more virulent strain of the virus or the combination of concave gum virus with another virus was the cause of this atypical phenotype. It is not clear, however, whether the virus mentioned in Russo and Klotz (1963) was citrus concave gum–associated virus (CCGaV) characterized by Minutolo et al. (2021) or citrus virus A (Navarro et al. 2018). CCGaV and citrus virus A have been associated with concave gum (Navarro et al. 2018) and impietratura diseases (Bester et al. 2021), respectively. There have been no further reports of Tarocco pit since the 1960s.

Currant and Gooseberry (*Ribes*)

Gooseberry mosaic

Gooseberry mosaic was reported from a single gooseberry 'Lady Delamare' plant, with a bright yellow mottle and vein yellowing, in Poland (Basak and Maszkiewicz 1980). The disease was graft transmitted to several gooseberry and black currant cultivars, which developed with symptoms of localized, patchy yellowing that gradually spread to most of the leaf area to resemble black currant yellows (Adams 1987; Posnette 1952), and was apparently latent in red currant cultivars. Attempts to transmit the virus to herbaceous species were not successful. Basak and Maszkiewicz (1980) concluded the host range and symptoms of the disease were different from gooseberry vein banding disease, the causal agent of which was later identified as the

gooseberry vein banding–associated virus (GVBaV; Jones et al. 2001; Petrzik et al. 2012). Since 1980, no other studies have been performed on identifying the causal agent of the disease.

Leaf malformation of gooseberry

The "claw leaf" or "hawthorn leaf" condition in England mentioned by Thresh (1970) was not graft transmitted. There is no information on the properties of the causal agent(s).

Vein clearing and vein net of black currant

Vein clearing and vein net disease was described by Thresh (1966) and attributed to infection with GVBaV. Gooseberry and red currant develop symptoms including vein banding or vein clearing. Although this disease was widespread in gooseberry and red currant, infected black currant bushes were rare in Britain (Thresh 1966) and in continental Europe (Adams and Thresh 1987a).

The disease has only been transmitted within the genus *Ribes* by grafting and by several aphid species from gooseberry to black currant and vice versa but not by sap (Adams and Thresh 1987a). Gooseberry and red currant develop symptoms typical of vein banding (Thresh 1970), suggesting vein clearing and vein net of black currant as a possible synonym of gooseberry vein banding disease caused by GVBaV.

Black currant yellows

Black currant yellows disease was found in a single English nursery (Posnette 1952) and subsequently in several plantations established with bushes distributed from it. The disease, a yellowish-green mottle affecting large sectors of whole laminae, has not been reported in other countries. The disease has only been graft transmitted between black currants, and different cultivars react similarly to infection. No natural vector has been found, and experimental tests with five aphid species were unsuccessful (Cropley et al. 1964; Thresh 1987). There is no information on the properties of the causal agent. With hindsight and based on the mite observations by Cropley et al. (1964), black currant yellows are most likely associated with black currant reversion virus.

Yellow leaf spot disease of red currant (synonyms Aucuba mosaic and European currant mosaic)

Yellow leaf spot of red currant has been described and referred to as Aucuba mosaic or European currant mosaic in several European countries (van der Meer 1987a). Experimentally, the disease could be transmitted between currants by grafting. Negative results of sap inoculation experiments, reported by several authors, suggest that the causal agent is not mechanically transmissible, and there is no information on the way and rate of natural spread in red currants. Jacob (1976) isolated potato virus Y (PVY) from five red currant cultivars that showed yellow leaf spot but also from asymptomatic plants. His results, however, have not been confirmed by other authors (van der Meer 1987a), and neither PVY nor any other potyvirus has been reported in currants since (Špak et al. 2021). Yellow leaf spot disease has not been observed since 1968.

Infectious variegation of black currant (synonym gold dust, Campbell and Adam 1968)

The symptoms of infectious variegation were described by Posnette (1952). Black currant is the only known host, and 'Daniel's September' and 'Laxton's' develop a bright chrome or pale-yellow mosaic of the early leaves. This is followed in summer by a broad yellow banding of the main veins, forming a vein net pattern. Symptoms differ greatly in severity between years. The disease was reported to be graft transmitted by Ellenberger (1962) and Kristensen et al. (1962), but confirmatory evidence is lacking (Wood 1991). Sap inoculation to herbaceous hosts failed, and no experiments on insect transmission have been reported (Adams and Thresh 1987b).

Red currant vein banding

Symptoms of vein banding in red currants were first described in the former Czechoslovakia (Blattný 1930). In most red currant cultivars, symptoms are vein banding and vein clearing, which are often restricted to parts of the leaves. The supposed virus was transmitted by aphids and by grafting but not by sap inoculation; however, particles were never observed. In black currant and gooseberry seedlings, red currant vein banding causes symptoms that resemble those of GVBaV. Both red currant and gooseberry vein banding are transmitted by the same aphid species and show the same virus-vector relationships (van der Meer 1987b). Therefore, it was concluded that most vein banding diseases described in black and red currants and gooseberries are probably caused by GVBaV.

Grapevine (*Vitis*)

Little leaf

A little leaf disease was described in grapevine cv. Merlot Noir that was held at the Indian Institute of Horticultural Research, Bangalore (Singh et al. 1975). The disease was graft transmitted to *Vitis vinifera* 'Emperor'. Heat treatment at 45 and 50°C for an hour and treatment with oxytetracycline resulted in recovery from symptoms, suggesting an association with a phytoplasma, but there appears to be no further confirmatory work to support this hypothesis. It appears that the variety is no longer held in this collection (Maul and Töpfer 2015) and there are no further reports of this disease in India or elsewhere.

Infectious chlorosis and leaf reddening of 'Pinot Noir'

Infectious chlorosis and leaf reddening was reported in France upon grafting of *V. vinifera* 'Pinot Noir' with other *V. vinifera* cultivars including 'Cinsaut'. Foliar symptoms consisted of a chlorosis and then dark reddening of the entire leaf blade and veins (Bovey et al. 1981; Martelli and Boudon-Padieu 2006). Given that there are several biotic agents causing leaf reddening in grapevine, it would be impossible to identify the agent(s) without a type isolate.

Flat trunk

The trunk of diseased grapevines was described as elliptical or flattish on opposite sides. The disease was described in Italy, Israel, Hungary, and the United States. Graft transmission has been reported (Hewitt 1975), but the etiological agent and its economic importance are not known (Bovey et al. 1981; Martelli and Boudon-Padieu 2006).

Summer mottle

Summer mottle was reported only in the red table grape 'Siderites' in Australia with no evidence of field spread, although the agent is graft transmissible (Krake and Woodham 1978). Affected *V. vinifera* cultivars express pale green interveinal discolorations, producing a typical vein feathering and vein banding, particularly during hot summer weather. Indicators used were 'Cabernet Franc', 'Cabernet Sauvignon', and 'Mission', and infection was latent in rootstocks. Sanitation through apical meristem culture eliminated the agent. Similarities between summer mottle and vein mosaic have been described, yet unlike the former, vein mosaic was not transmissible to *V. rupestris* 'St George' (Krake and Woodham 1978; Martelli 1993; Martelli and Boudon-Padieu 2006; Woodham and Krake 1983). Summer mottle–affected 'Siderites' vines were eradicated in Australia, and the disease is no longer known to occur.

Enation

Vines affected by enation show slow growth and delayed opening of the buds. Symptomatic vines often recover later in the season. Enations develop primarily on the underside of the leaves at the base of the shoot. Mature leaves are often misshapen and display prominent veins. Enation is graft transmissible, but with difficulty (Graniti et al. 1966; Martelli 1993). The disease has been reported in Europe, Israel, the United States, Venezuela, North Africa, South Africa, New Zealand, and Australia in several cultivars (Martelli 1993). No relationship exists between grapevine fanleaf virus and enation disease. The etiology of enation disease remains undetermined, although a hormonal imbalance has been suggested as a causal factor or a synergistic effect among several viruses in mixed

infections (Chiumenti et al. 2012, 2013; Martelli 1993; Martelli and Boudon-Padieu 2006). Some *Enamovirus* species are associated with enation in other crops. Recently, two grapevine Enamoviruses, Grapevine enamovirus 1 and Grapevine enamovirus 2, were described (Diaz-Lara et al. 2023; Silva et al. 2017); however, they are often found in mixed infections with other viruses, and the presence of enations was not reported. Improved nursery and/or field cultivation practices have apparently eliminated the disease.

Bushy stunt

A transitory form of graft incompatibility on certain clones of the rootstock 140 Ruggeri (*V. berlandieri* × *V. rupestris*) was reported in Italy and described as bushy stunt (Savino et al. 1991). Scion buds of *V. vinifera* grafted onto 140 Ruggeri rootstocks showed a stunted and bushy vegetation owing to the contemporary proliferation of apical and axillary buds, and reduced yield, but the canopy remained green. Normal growth resumed with the second or third leaf, but the yield was reduced. The disease was latent in 140 Ruggeri and erratically transmitted by grafting to some grape indicators. 'Italia' and 'Sangiovese' top grafted on some of these 140 Ruggeri accessions reproduced the disease symptomatology (Savino et al. 1991). The disease has been eliminated by therapeutic treatment, but its etiology is not known (Martelli 1993; Martelli and Boudon-Padieu 2006). There are no reports of bushy stunt since the original publication as a conference proceeding.

Pome Fruit (*Malus/Pyrus/Cydonia*)

Malus robusta No. 5 decline

There were observations of poor performance of *M. robusta* No. 5 rootstock trials in British Columbia, Canada, in the 1950 and 1960s. In severe cases, pruned trees showed leaf flecking, ringspot and interveinal chlorosis, thin foliage, bark scaling, and internal necrosis, with tree death occurring approximately 4 years after inoculation (Welsh and Spangelo 1971). It was speculated that this disease was caused by viruses present in the scions. However, this disorder has not been reported since, possibly because *M. robusta* No. 5 is less commonly used in modern plantings.

Apple (McIntosh) depression

This disease was described from a single 'McIntosh' tree in New York, U.S.A., in the 1960s, which exhibited mosaic and puckering of leaves, and deep depressions on the fruit, flattening on one or more sides (Palmiter 1969). The disease was found to be graft transmissible to other 'McIntosh' trees but was reported on one tree and has not been observed again.

Apple bumpy fruit of Ben Nevis

This disease is assumed to be similar to apple green crinkle described on *M. domestica* cv. 'Ben Davis' in Israel in the 1960s (Blodgett et al. 1965). It was graft transmissible and reproducible on 'San Jacinto' but has not been reported since. Another "bumpy fruit" disorder was reported on 'Golden Delicious' in India, which was associated with apple scar skin viroid (Behl et al. 1998), but it is unknown whether the two are the same.

Apple bunchy top/necrotic spot and mottle

Both diseases in apple were reported from Himachal Pradesh, India, by Sharma et al. (1979). There is no information on the causal agents nor whether these disorders are truly distinct from other graft-transmissible virus diseases reported in apple (Howell et al. 2011) or the newly identified apple necrotic mosaic virus (Noda et al. 2017).

Apple dead spur

Dead spur is a disease first observed in Washington state, U.S.A., in the 1960s and subsequently reported across North America, Poland, and China (Parish et al. 1983; Parish 1989a). Fruiting spurs fail to emerge from dormancy or are weak and subsequently die. Symptoms are concentrated on buds on the interior of the tree; buds on the tips of limbs may develop normally, leading the tree to take on a willow-like appearance (Parish 1989a). No field spread has been observed.

Apple freckle scurf

Apple freckle scurf was reported from a single 'Winesap' tree in Wenatchee, Washington state, U.S.A., in 1960. The disease, a bark necrosis and scaling disorder, was reproduced by graft transmission on 'Winesap' 4 years after inoculation; 'Red Delicious' and 'Golden Delicious' were asymptomatic (Parish 1989b).

Apple internal bark necrosis

Transmissible internal bark necrosis is a disease that appears visually similar to apple measles, a manganese or boron toxicity, on 'Red Delicious' (Parish 1981). It is graft transmissible, with symptoms appearing on older wood 3 to 4 years after inoculation of 'Red Delicious'; no other hosts are known. No further reports have been made subsequent to the original report.

Apple leaf pucker agent/related disorders

This disorder was reported from Canada and India in the 1950 and 1960s affecting 'Ballarat', 'McIntosh', 'Spartan', 'Stayman', and 'Winesap'. Symptoms included chlorotic vein flecking, leaf puckering and distortion, reduced bloom, and dimpled and distorted fruit (Nagaich and Vashisth 1965; Welsh and May 1973; Wood 1972b). The disease was graft transmissible, but no in-field spread was reported.

Apple little leaf

A graft- but not sap-transmissible disease that induces the production of small, distorted, and mottled leaves as well as delayed bud break was reported in India in the 1950 to 1960s (Nagaich and Vashisth 1965). Small or "little leaf" symptoms are also associated with zinc deficiency, which may have contributed to its persistence in the literature.

Apple 'Newton' wrinkle (apple fruit wrinkle)

This fruit marking disease only affects 'Yellow Newton' and is characterized by dimples or linear depressions on the skin of the fruit (Welsh and May 1970). Although it was graft transmissible to 'Yellow Newton', symptoms, which appeared 2 years after inoculation, varied considerably in severity between individual fruits on the same tree (Welsh and May 1970; Zawadzka and Millikan 1989). The original trees were reported to have been eradicated, and no additional reports have been published (Zawadzka and Millikan 1989).

Apple platycarpa scaly bark

M. platycarpa was widely used as an indicator to detect the presence of viruses in other apple cultivars (Campbell 1963). Scaly bark was a syndrome widely described from grafting from multiple sources in the United Kingdom and subsequently in other countries (Wood 1974). The agent was controlled by heat therapy of propagative material (Waterworth and Posnette 1989).

Apple red ring

A circular red-ring color disorder was observed on the fruit of six 'Red Delicious' trees in Oregon, U.S.A., in 1962, which faded as the fruit matured and the normal red color developed. Although this syndrome was graft transmissible and reproducible in 'Red Delicious' and 'Starkrimson' (Coyier et al. 1967; Zawadzka and Millikan 1989), no evidence of field spread was reported. Apple chlorotic ring spot, apple stem pitting, and apple stem grooving viruses have been associated with red ring, but this has yet to be confirmed (Németh 1986).

Apple ring spot

This graft-transmissible disorder induces pale brown ring spots on the fruit, which are rough on the surface. At fruit ripening, brown rings and lines were observed to form around these spots. This disorder was reported on multiple apple varieties including 'Abbondanza', 'Cox Orange', 'Golden Delicious', Granny Smith', and 'Sturmer Pippin' (Kegler 1977) but was symptomless on other varieties. The symptoms differed in color from a similar syndrome described in Bulgaria as

apple line mosaic, which consisted of light green–colored ring spots (Kegler 1977). Similar disorders were reported in New Zealand, Italy, and the former Yugoslavia and Czechoslovakia, but it is unknown whether the causal agent was the same in all countries. There have been no new reports for nearly 50 years.

Apple rosette

In 1950, two graft-transmissible disorders were reported from the Netherlands on the apple variety 'Belle de Boskoop', consisting of rosetting, leaf distortion, and sterility, whereas healthy controls remained symptomless (van Katwijk 1953). Although subsequent reports have been made from Denmark, Italy, and the former Soviet Union (Németh 1986), its current status is unknown.

Apple russet wart

An agent that induced raised russetted warts on fruit was identified from a single orchard in the United Kingdom in the 1960s. This agent was found to be graft transmissible to 'Cox's Orange Pippin', 'Golden Delicious', and 'M.2' rootstocks, where it produced similar symptoms, with necrotic russet rings occasionally developing on small immature 'Cox's Orange' fruit and black necrotic spots on 'Golden Delicious'. Other symptoms observed were similar to leaf pucker, russet ring, and green crinkle diseases, but the necrotic spots on leaves of the three varieties and 'M.2' rootstock distinguished it from these syndromes (Posnette and Cropley 1969). Similar symptoms had been reported across much of Europe (Németh 1986), but the current status of the disease is unknown.

'Apple' or 'Granny Smith' leaf fleck, bark blister, fruit russet, and distortion

An agent(s) that caused pale green leaf flecking, bark blisters, and cankers on both current-season and older wood, and severe fruit distortion and russet symptoms, was described in New Zealand in 1961. The disease was cultivar specific, affecting only 'Granny Smith', 'Red Statesman', and 'Lord Wolseley' (Wood 1972b). The original field trees were destroyed (Wood 1972b).

'Jubilee' ring and line pattern, 'Newton' ring russeting, 'Stayman' blotch (Bateman blotch), russet ring on 'Yellow Newton' or 'Golden Delicious', and 'Starking delicious' ring russeting

These names represent an arbitrary grouping of leaf pucker and fruit russet ring disorders described on and from the eponymous apple cultivars in Canada in the 1960s (Welsh and May 1967). Graft transmission to 'Jubilee', 'Spartan', 'Red Delicous' and 'Golden Delicious', 'Stayman', 'Yellow Newton', and 'McIntosh' from each source indicated both commonalities and differences in the appearance and severity of leaf vein flecking, vein blotching, oak-leaf patterns, and puckering of leaves, as well as in the extent and severity of russet ring networks formed on the fruit (Welsh and May 1967). Given the results reported, it is likely that each symptomatology represented mixtures and/or variants of common apple viruses, probably including apple chlorotic leaf spot virus (ACLSV), as it has been associated with russet ring in other apple cultivars (Li et al. 2020).

Pear bark split

Ring-like rough bark and foliar ring-pattern mosaic symptoms on pear cultivar 'Marianne' on 'Beurré Hardy' interstocks were observed in 1962 in Germany. Transmission experiments at the time indicated that, based on leaf symptoms on 'Schraderhof 603' and 'Williams', the disorder was caused by the bark split agent alone or in combination with the ring pattern mosaic agent. It appears that the bark split on 'Beurré Hardy' was dissimilar to pear bark diseases described from England and Denmark (Kegler 1965b).

Pear concentric ring pattern

In the late 1960s in Maryland, U.S.A., tan to dark brown–colored rings were observed on pear fruit, near the calyx. Symptoms appeared in the middle of summer, and affected fruit were also smoother-skinned and yellow. The disease was graft transmissible, although interestingly, a similar disorder on pear cultivar 'Packham's Triumph' reported in Australia and New Zealand was not transmitted by budding (Van Der Zwet et al. 1971). There have been no further reports on the agent.

Pear freckle pit

Freckle pit is a graft-transmissible disorder characterized by small green pits near the calyx, with green thread-like streaks extending into the fruit tissue below that become brown as the fruit ripens; in mild symptomatology, they fade as the fruit matures (Hansen and Waterworth 1989; Parish and Raese 1986). Identified in both the U.S. Pacific Northwest and Canada, slow field spread has been reported, although no vectors were identified (Hansen and Waterworth 1989; Parish and Raese 1986; Wilkes and Welsh 1965). Hansen and Waterworth (1989) assumed that given the slow rate of spread, the disease may be eliminated through the use of clean planting stock, as there have been no further reports.

Pear rough bark

This graft-transmissible disease showed pronounced splits and furrows on young shoots and older branches of several sensitive varieties in Denmark and England. For indexing, 'Williams' or 'Nouveau Poiteau' were used (Kristensen 1963). There is no further information on the similarity to other graft-transmissible bark diseases on pear.

Pear mild mosaic

Mild mosaic was reported from pear in the Punjab and Uttar Pradesh states in India on both cultivated and wild material, expressing leaf mottle, chlorotic spots, and oak-leaf patterns, as well as feathered leaf edges (Nagaich and Vashisth 1962). The authors suggested similarity between these symptoms and pear mosaic in the United Kingdom, which was later identified as apple chlorotic ringspot virus (Cropley et al. 1963). However, this similarity has not been confirmed, and pear mild mosaic has not been reported subsequently.

Quince wood pitting

This disorder was first reported in Israel and subsequently in California, U.S.A. In both cases the agent was not graft transmissible, and no further reports have been made (Fleisher et al. 1975; Waterworth and Fridlund 1989).

Quince yellow mosaic/quince yellow blotch

Yellow mosaic was reported in India in the 1960s. Graft transmissible to quince seedlings, the agent induced yellow flecking of the leaves, similar to apple mosaic virus, followed by necrosis of the affected areas and leaf wilt (Waterworth and Fridlund 1989). There are no further reports on the putative agent.

Raspberry, Blackberry, and Their Hybrids (*Rubus*)

Raspberry leaf curl virus

There have been diseases linked to 'raspberry leaf curl virus' in both the Americas and Europe. With the identification of raspberry ringspot virus and tomato black ring virus linked to raspberry leaf curl in Europe (Murant 1974), the following information refers to the synonymously named virus in the Americas. The history of the agent(s) associated with the homonymous symptoms goes back to the nineteenth century, but the seminal report on leaf curl is from Bennett (1930), who performed several transmission experiments with *Aphis rubicola*, the primary vector of the disease. The leaf curl agent is presumed to have two strains, α and β, which in challenge experiments did not cross protect against each other. In addition, depending on the aphid vector, acquisition may take 2 to 24 h (Stace-Smith and Converse 1987). This indicates that at least two viruses/agents are associated with the observed symptoms: severe downward leaf cupping and general epinasty of the affected plants. There are speculations that the disease is caused by a luteovirus (in the older, broad-spectrum definition that includes small, spherical, aphid-transmissible, and phloem-limited viruses) and/or a rhabdovirus. Such viruses have been recovered from leaf curl–displaying plants (Di Bello et al. 2017; Guzman et al. 2018), yet there were other

plants with similar symptoms that were infected by a complex of known viruses (R.R. Martin, *unpublished*). Given the complexity of the disease when it comes to the causal agent(s), it is clear that it is not possible to associate any virus, new or previously described, with the name raspberry leaf curl virus.

Thimbleberry ringspot virus

A virus-like agent was isolated from a single site in British Columbia, Canada (Stace-Smith 1958). It was transmitted semi-persistently by three aphid species but not by the major virus vector in the area, *Aphis agathonica*. Symptoms are generic, ranging from oak-leaf patterns to mosaic, ringspots, and chlorotic spots.

Bramble yellow mosaic virus

Bramble yellow mosaic virus (BrYMV) has been reported once in conference proceedings. BrYMV was discovered in South Africa and was mechanically transmitted from a wild trailing blackberry showing yellow mosaic and line pattern symptoms (Engelbrecht 1976). The virus was transmitted to several indicators and was seedborne in *Chenopodium murale*. Partial purification revealed elongated particles, and the virus had physicochemical properties resembling potyviruses. Given the lack of any additional information on the virus, it is impossible to identify BrYMV as such.

Raspberry yellow spot virus

Raspberry yellow spot virus was described in a single report from Poland where the foliage was covered with irregular yellow spots that differed in size and leaf blade coverage (Basak 1974). The agent was transmissible by the large European raspberry aphid *Amphorophora idaei* and grafting. Given the symptom similarity and the *A. idaei* transmission, raspberry yellow spot virus could be an isolate of raspberry leaf mottle virus.

Alpine mosaic agent

This is an agent with a misleading name. A study on sterility of 'Darrow' blackberry (Converse 1986) led to the discovery of the agent. The putative causal agent of the disorder was not graft transmissible to blackberry, indicating that the disorder was genetic. Yet an agent was graft transmitted to *Fragaria vesca* 'Alpine' strawberry from both sterile and fertile 'Darrow' clones. In 'Alpine' it caused mosaic and vein clearing. No blackberry clones from those experiments are available for further analysis.

Necrotic fern-leaf mosaic virus

Fern-leaf mosaic was described from a single 'Cuthbert' plant from Ontario, Canada (Chamberlain 1941). The plant with 'fern-leaf'–like leaves also had chlorotic and necrotic spots and was stunted. The clone was not retained past the original study (Stace-Smith 1987).

Black raspberry streak virus

A disease described a century ago without a single reference since 1960 (Converse 1970). Symptoms on raspberry include leaf streaking. The putative virus could cause severe or mild symptoms, which complicates things further, as symptoms could be caused by two individual viruses or a combination of viruses. The disease is graft and dodder transmissible.

Rubus Chinese seedborne virus

Rubus Chinese seedborne virus (RCSV) was only studied in a single seedling. The paper by Barbara et al. (1985) states: "The Rubus seedling from which RCSV was originally isolated showed no symptoms. Unfortunately, the plant died shortly after the virus was first detected and before the plant could be propagated or the virus transmitted to other *Rubus* material by grafting. The virus has not been detected in any other *Rubus* material." Given this comment it is clear there is no isolate available. However, the authors were able to mechanically inoculate 23 herbaceous indicator species, develop antisera, and describe physicochemical properties of the virus, which was assumed to be most similar to, but distinct from, strawberry latent ringspot virus.

Rose (*Rosa*)

Rose streak

Rose streak disease occurred in Europe and the Eastern and Midwestern United States and was found infrequently in California, U.S.A. (Secor et al. 1977). The graft-transmissible disease was suspected to be caused by "rose streak virus," although no such virus has been identified and characterized. Symptoms associated with "rose streak" are brownish-green rings and vein banding. Ring patterns on stems had been observed as well. No reports on the occurrence of rose streak disease have appeared in recent years.

Rose ring pattern

Rose ring pattern (Secor and Nyland 1978) was reported in commercially grown roses in California and Oregon, U.S.A. Even in 1978, rose ring pattern was described as part of the rose mosaic complex, which itself describes diseases caused by any (or mixtures) of at least five distinct viruses. The symptoms of rose ring pattern are similar to those of rose mosaic, except that symptoms in 'Burr' multiflora rose are distinct from those of prunus necrotic ringspot virus (PNRSV).

Rose wilt

Rose wilt disease was first reported in New Zealand and Australia prior to 1931 (Grieve 1931) and has later been described in California (Cheo 1970; Fletcher and Kingham 1962; Gumpf and Weathers 1974; Khristova 1974; Lisa 1998; Slack et al. 1976). Reported symptoms include leaves curling downward, with significant vein clearing, epinasty, and early leaf drop. The identity of the causal agent is unknown. Epidemics in parts of Australia were reported at considerable intervals, with one in Sydney in 1922 and the next not until 1952. No reliable reports of the occurrence of rose wilt are available in the literature since the 1970s.

Rose leaf curl

Rose leaf curl was reported to be widely distributed throughout the United States in "antique" roses in public rose gardens (Cheo 1970; Gumpf and Weathers 1974; Slack et al. 1976). Characteristic symptoms resembling rose wilt were reported on hybrid tea roses but not on rootstock genotypes. Symptoms are first seen in the spring as reduced leaf size, easily detached leaflets, leaf epinasty, necrosis of shoot tips, and a yellow flecking of veins that may progress into necrosis. The causal agent of rose leaf curl is undetermined but likely distinct from rose leaf curl virus, reported from India and Pakistan (Khatri et al. 2014). In screening by HTS of roses showing a variety of symptoms, no begomovirus infections have been detected (Al Rwahnih, *personal observation*). The disease previously reported in the United States is therefore presumed to be distinct from that caused by rose leaf curl virus in Pakistan. Whether the disease seen in the United States has a biotic or an abiotic cause is still unclear, and the description as "rose leaf curl" is not useful because of potential confusion with the begomovirus-induced disease, which has a clear etiology.

Rose flower proliferation

Rose flower proliferation, in which the center of the flower elongates into a stem that bears additional flowers, has been described in Italy (Gualaccini 1963). However, similar symptoms can occur in some cultivars when grown under excessive nitrogen fertilization conditions. Another flower anomaly, in which petals turn green and leaflike, has been described in the United Kingdom. The cause of both types of symptoms is unknown, nor is it known if they are transmissible. The symptoms of this disease share similarities to diseases caused by phytoplasmas in other crops, but to date there is no evidence associating any phytoplasma with rose flower proliferation.

Mottled rose mosaic virus

Mottled rose mosaic virus (MRMV) was described in Washington state, U.S.A., to infect *R. rugosa* Thunb. (Kirkpatrick et al. 1968). The putative virus was associated with green mottled foliage on *R. rugosa*. The authors inoculated stone fruit trees and subsequently

inoculated herbaceous indicator plants including cucumber and *Physalis pubescens* L. (syn. *P. floridana*), in both of which symptoms were observed. Transmission from cucumber to mahaleb cherry and peach caused symptomless infection. Sap transmission from these stone fruit trees to herbaceous indicators 2 to 7 years later induced symptoms on *Physalis pubescens* L.; bud grafting from mahaleb cherry and peach to other woody indicators yielded no symptoms. Although MRMV was distinguished from prunus necrotic ringspot, prune dwarf, and plum line pattern viruses based on herbaceous host range and symptoms, there are no further reports of this agent since 1968.

Rose stunt/wilt

Rose stunt disorder was described in the United Kingdom and New Zealand (Ikin and Frost 1974). The disease was studied in the 1960s, but researchers could not associate a causal agent by either graft transmission or electron microscopy. It was hypothesized that rose stunt could be the same as the unidentified rose wilt virus described in New Zealand (Fry and Hammett 1971). The EPPO Global Database (https://gd.eppo.int/taxon/ROW000) lists rose wilt as synonymous with rose stunt.

Rose line pattern virus

Rose line pattern virus was described in England in 1961 (Fletcher and Kingham 1962). Wavy yellow lines were described on young leaves and symptoms were transmitted to *R. canina* L. from the 'New Yorker' variety. Additionally, cane necrosis and bud dying were reported before flower opening, associated with lateral shoot proliferation. During fall and winter, symptoms were not present and bud development was normal.

Rose X disease

Secor et al. (1977) described the disease in *R. rugosa* Thunb. in California and Oregon. When transmitted by grafting to *R. multiflora* Thunb., symptoms were severe stunting and microphylly with mottling. In hybrid tea roses, Secor et al. (1977) described association of symptoms with rose X agent with symptoms of line pattern and rings being similar to rose mosaic. Even though nearly 100% transmission rate by grafting is recorded, no causal agent has been identified since its description.

Stone Fruit (*Prunus*)

Almond virus bud failure

This disease was first reported in 1947 in the United States (Stout and Wilson 1947). It is characterized by a failure of blossom or leaf buds to develop. Parallels have been drawn to Drake almond bud failure, peach mule's ear, peach wild leaf, and peach willow twig diseases (Thomas 1951; Wilson and Stout 1951; Wilson and Wagnon 1955). This suggests that these diseases could represent the same agent in different hosts but lacks substantiation. The causal agent was named almond bud failure virus. However, in almonds affected by almond virus bud failure and in peaches affected by mule's ear disease, the presence of PNRSV has been repeatedly reported, although the association has yet to be confirmed (Nyland 1976).

Apricot bare twig and unfruitfulness

This is a graft-transmissible disorder of apricot causing bare limbs that lack lateral branches and spurs, reduced fruit set, and leaf rolling. Strawberry latent ringspot virus and cucumber green mottle mosaic virus have been isolated from symptomatic trees (Cech et al. 1979), yet no association has been established between virus occurrence and symptomatology.

Apricot chlorotic leaf mottle

This disease was initially observed in New Zealand in a single orchard in 1968/69 (Wood 1975), with trees exhibiting leaf scorch, deformation, and interveinal chlorosis, as well as spur death. An infectious agent could be experimentally transmitted by grafting to some apricot and peach varieties, causing late-season pale green chlorotic areas or a chlorotic mottle in grafted peach and apricots.

Apricot deformation mosaic

This apricot disease reported from Moldova in 1968 (Verderevskaja 1968) has been shown to be graft transmissible in apricot. There is no information on the causal agent or whether it is truly distinct from other disorders reported in apricot (Howell et al. 2011).

Apricot moorpark mottle

This was a low-incidence, graft-transmissible apricot disease observed in New Zealand in 1954, with scattered foliar chlorosis and misshapen fruit near the stem (Chamberlain et al. 1954). No other reports have been published since.

Apricot necrotic leaf roll or apricot yellow line pattern

This disease is listed in the quarantine regulations of several countries, despite the fact that the original publication describing the disease could not be traced and there are no further reports in recent years.

Apricot pucker leaf

This disease was found in several orchards in Utah, U.S.A., in 1966 (Wadley 1966). The infectious agent(s) was graft transmitted to apricot, peach, Manchu cherry, and Marianna plum. No additional infected trees had been found until 1976 (Wadley 1976a), and there are no reports in recent years.

Apricot witches' broom

This disease was identified in two apricot trees in the former Czechoslovakia in 1977 (Blattný 1977). It is characterized by witches' broom symptoms, small twigs and leaves. A phytoplasma etiology was suspected at the time but has not been investigated. Although the presence of phytoplasmas in Czech *Prunus* trees has since been reported (Navrátil et al. 2001), there have been no new reports of the disease.

Apricot yellow mosaic

This disease was identified in the Cape Province of South Africa in the 1960s (Wolfswinkel 1966). Little information is available on this disease, as the original publication could not be accessed and there are no further reports in recent years.

Cherry black canker

This is a graft-transmissible disease identified in the U.S. Northeast in the 1940s (Zeller et al. 1947). There were no further reports in recent years.

Cherry freckle fruit

This disease was found in two cherry trees in Oregon in 1952. Graft transmission was achieved to sweet cherry indicators (Williams and Cameron 1976). The report stated that the symptoms of very small fruit size and severely delayed maturation were reminiscent of little cherry disease. However, it remains unknown whether little cherry viruses 1 or 2 might incite the disease, alone or in combination with other agents.

Cherry fruit necrosis/sour cherry fruit necrosis

Fruit necrotic symptoms in sweet and sour cherry is known to be caused by severe isolates of ACLSV, and it has been suggested that the disease might be caused by a mixed infection of ACLSV and PNRSV (Németh 1986). Because it was not possible to unambiguously trace the original publication describing the disease, it is unclear what "cherry fruit necrosis agent" refers to.

Cherry line pattern and leaf curl

Little information is available on this disease, and the only report in eastern Europe could not be accessed (Paulechova 1984). The disease may be correlated with the later discovery of American plum line pattern virus in the Mediterranean (Myrta et al. 2003). No new reports have been published in recent years.

Cherry midleaf necrosis

A graft-transmissible disease was observed in sour cherry in Oregon, U.S.A. (Milbrath 1957). The fact that it was not mentioned in the extensive description of North American virus diseases of stone fruits published by the United States Department of Agriculture (USDA) in 1976 suggests the lack of any significance.

Cherry (sweet) mora

This is a graft-transmissible disease described in sweet cherry in Oregon in 1949 (Milbrath 1952). It was not mentioned in the extensive description of North American virus diseases of stone fruits produced by the USDA in 1976 (Gilmer et al. 1976).

Cherry pseudo leafroll

This is an agent/disease listed in at least one quarantine legislation, yet it was not possible to unambiguously identify the original publication describing it. There is no information on symptoms or country/region in which the disease was originally observed.

Cherry rough bark

This disease was described in two cherry trees in Oregon in 1952 (Nichols and McClain 1957). Graft transmission with symptom expression was achieved in some sweet cherry cultivars. PNRSV was identified in the affected trees, but it is not clear whether other agent(s) also might have been present and responsible for the disease symptoms (Nichols 1976).

Cherry rough fruit

This disease was initially described in a germplasm collection in Utah on cherry material introduced from Iran. It was later reported in cherry trees in Iran and Bulgaria. Graft transmission was achieved to sweet cherry indicators. Samples in the United States were destroyed in 1965 (Wadley 1976b).

Cherry rusty spot

A disease in which leaves exhibited small, purple leaf spots that became necrotic as the season progressed, with leaf rolling and premature drop occurring in the summer, was described in New Zealand in 1972 (Wood 1972a). This agent was found in high prevalence (67%) in cherry trees in Central Otago and was shown to be graft transmissible to cherry indicators. It causes rust-colored spots on spring leaves, which later fall out, leaving shot-holes. Leaves appearing later in the season are symptomless. The absence of characteristic symptoms of RNRSV, necrotic rusty mottle, and European rusty mottle tatter leaf led to the conclusion that "rusty spot" represented a novel disease/agent. It cannot be ruled out, however, that it might have been caused by mixed infections or by distinct isolates/strains of a known virus.

Cherry short stem

This disease was described in Oregon and Montana, U.S.A., in the late 1950s and early 1960s (Parish and Cheney 1976). Symptoms were noted as resembling those of tomato bushy stunt virus (TBSV), but TBSV was not present in affected cherry trees. Graft transmission was achieved in cherry indicators and Japanese plum, whereas symptomless carriers included almond, apricot, peach, and some wild *Prunus* species. Natural spread was reported in the field, but the vector is unknown. The latest report of the disease dates back to 1994 (Li 1994).

Cherry sickle leaf

This is a quarantine listed agent/disease in New Zealand for which it was not possible to identify the original publication describing the disease. Thus, there is no information on symptoms or country/region in which the disease was originally observed.

Spur cherry

This disorder was identified in a single 'Bing' cherry tree with unusual compact, spur-type growth in Washington state, U.S.A., in 1962 (Cheney et al. 1967). As of 1976, the natural occurrence had been limited to that single tree (Blodgett and Aichele 1976a), although some graft-inoculated trees had been commercialized as dwarf planting material. Not all cherry cultivars showed growth reduction. Symptoms reported as "typical of apricot ring pox" were also observed on inoculated apricots, leading to the notion that the same agent could be responsible for the two diseases (Cheney et al. 1969). This conclusion was not substantiated, and there are no further reports in recent years.

Cherry stem pitting

Stem pitting symptoms have been observed in cherry and in other *Prunus* hosts in the United States and Canada (Mircetich et al. 1978; Mircetich and Fogle 1969). Viruses identified as associated with such symptoms include tomato ringspot virus (ToRSV) and, less frequently, TBSV. However, work performed in California indicated a significant disease incidence in cherry trees without the apparent presence of ToRSV infection (Uyemoto et al. 1995). In 1998, the analysis of double-stranded RNAs in five infected trees failed to identify a common pattern, leading to the conclusion that "the primary causal agent(s) remain(s) unknown" (Zhang et al. 1998).

Cherry stunt

Even though the disease is identified in quarantine regulations of several countries, including New Zealand, it was not possible to identify the original publication describing the disease. No reports were traced in the literature in recent years.

Cherry vein clearing and rosette

It was not possible to unambiguously identify the original publication describing the disease. A virus named cherry rosette has been described in Germany as a possible (Kunz 1988) and now verified nepovirus (Blouin, Table 1). It is, however, unclear whether the term "Cherry vein clearing and rosette agent" refers to cherry rosette virus.

Cherry white spot

White spot disease was described as graft transmissible in cherry in Switzerland and Germany (Mallach 1957). The disease was present in mirabelles and was widespread in cherry in Bavaria, Germany, as well as "Altes Land" in northern Germany (Mallach 1956). In Switzerland a similar symptomatology for sweet cherry cultivar Rigikirsche was reported with small light green spots turning into white spots. A possible relationship to plum white spot described in the United States was not confirmed. The economic impact of this disease was evaluated as being low (Mallach 1956), and there are no new reports traced in the literature.

Cherry xylem aberration

Even though the disease is identified in quarantine regulations of several countries, including New Zealand, the original publication describing the disease could not be identified.

Peach bark and wood grooving

A single disease report identified this disease in two orchards in Michigan, U.S.A., in 1976, with no obvious tree-to-tree spread (Rosenberger and Jones 1976).

Peach chlorotic spot

Identified in New Zealand on peach trees that had been heat-treated to eliminate cherry green ring mottle virus (CGRMV) (Wood 1975). The disease was considered of minor significance. A new virus named peach chlorotic leaf spot virus, closely related to ACLSV, has been identified and its genome sequence determined in the 2010s from peach trees in China (Zhou et al. 2018). There is no information available as to whether this virus might be related to the disease identified in New Zealand.

Peach enation

A study in Japan of peach trees showing leaf enation symptoms led to the characterization of a putative new virus with icosahedral

particles (Kishi et al. 1973a). The virus was mechanically transmissible to herbaceous hosts and reproduced the disease symptoms when back transmitted to peach. It was given the name peach enation virus and, based on the wide host range and particle morphology, tentatively ascribed to the genus *Nepovirus*. The virus has not been further studied and has since only been inconclusively reported in China on the basis of enation symptoms alone (Ruan et al. 1998).

Peach leaf necrosis in plum

A disease identified in Germany (Kegler 1965a) with a graft-transmissible agent latent in plum but causing severe stunting in peach seedlings. Shoots of the previous year died off over winter and new shoots were stunted and showed leaf rolling with yellow and brown necrotic spots. No other reports were traced in the literature.

Table 1. Agents/diseases caused by infectious agents previously misidentified, synonymized, or characterized and that now have a confirmed pathogen ID

Crop	Agent/disease name	Confirmed pathogen ID	References
Citrus	Citrus dieback disease, leaf mottle yellows disease, likubin, citrus vein-phloem degeneration	'*Candidatus* Liberibacter asiaticus'	Wallace 1978
Citrus	Oleocellosis-like symptoms of satsuma orange, summer orange dwarf (a.k.a., satsuma dwarf-like disease on citrus natsudaidai in Yamaguchi Prefecture), citrus mosaic, and Natsudaidai dwarf	Satsuma dwarf virus	Iwanami 2010, 2023; Le Gall et al. 2005; Tanaka and Yamada 1961; Toru Iwanami (*personal communication*)
Citrus	Mechanically transmitted citrus ringspot virus	Citrus psorosis virus	Garnsey 1975; Garnsey and Timmer 1980
Citrus	Bahia bark scaling	*Lasiodiplodia iraniensis* in grapefruit	Gama et al. 2019a, b; Moreno et al. 2015; Nickel et al. 2007; Passos 1965; Passos et al. 1974; Cristiane Barbosa (*personal communication*)
Cydonia	Quince deformation, sooty ringspot	Apple stem pitting virus	Morelli et al. 2017
Cydonia	Quince stunt	Apple chlorotic leaf spot virus, apple stem pitting virus	Yaegashi et al. 2011
Grapevine	Grapevine virus C	Grapevine leafroll-associated virus 2	Masri et al. 2006
Grapevine	Grapevine leafroll-associated virus 8	Not a pathogen but partial sequence of grapevine genome	Bertsch et al. 2009
Malus	Apple rubbery wood, flat limb	Apple rubbery wood virus-1 and apple rubbery wood virus-2	Rott et al. 2018
Malus	Flat apple	Cherry rasp leaf virus	James et al. 2001
Malus	Green crinkle	Apple stem pitting virus	Li et al. 2020
Malus	Russet ring and associated disorders	Apple chlorotic leaf spot virus	Li et al. 2020
Malus	Union necrosis	Tomato ringspot virus	Stouffer and Uyemoto 1976
Malus	Apple blister bark	Apple fruit crinkle viroid	Koganezawa and Ito 2011
Rubus	Blackberry calico	Blackberry calico virus	GenBank accession OR026033; Fager and Mollov (*unpublished*)
Rubus	Black raspberry latent virus	Strawberry necrotic shock virus	Tzanetakis et al. 2004b, 2007b
Rubus	Raspberry mottle virus, raspberry leaf spot virus, raspberry yellow spot virus	Raspberry leaf mottle virus	McGavin and MacFarlane 2010; Tzanetakis et al. 2007a
Rubus	Wineberry latent virus	Blackberry virus E	GenBank accession OQ877124 (MacFarlane, McGavin and Jones, *unpublished*)
Prunus	Peach blotch, peach calico, peach latent mosaic, peach yellow mosaic	Peach latent mosaic viroid	Flores et al. 2017
Prunus	Peach willow leaf and rosette	Strawberry latent ringspot virus	Belli et al. 1986
Prunus	Amasya cherry disease, cherry chlorotic rusty spot, cherry leaf scorch	*Apiognomonia erythrostoma*	Minoia et al. 2014
Prunus	Cherry rosette virus	Nepovirus	GenBank accession PP393046-7; Blouin (*unpublished*)
Prunus	Plum leaf roll	Phytoplasmas ('*Candidatus* Phytoplasma prunorum', '*Ca*. P. ziziphi')	Hong et al. 2011; Marcone et al. 2002
Prunus	Apricot fruit blotch; apricot Butteratura; apricot viruela	Apple chlorotic leaf spot virus	Cañizares et al. 2001; Desvignes et al. 1990; Furia and Ragozzino 1974; Peña-Iglesias 1988
Prunus	Cherry detrimental canker	Petunia asteroid mosaic virus	Jelkmann 2011
Prunus	Peach wart	Cherry mottle leaf virus	Mekuria et al. 2013
Prunus	Cherry twisted leaf	Cherry twisted leaf-associated virus	James et al. 2014; Villamor and Eastwell 2013
Prunus	Cherry mottle leaf	Cherry mottle leaf virus	James et al. 2000
Prunus	Cherry rusty mottle	Cherry rusty mottle-associated virus	Villamor and Eastwell 2013
Prunus	Cherry necrotic rusty mottle	Cherry necrotic rusty mottle virus	Rott and Jelkmann 2011
Prunus	Shirofugen stunt, Kwanzan stunt	Little cherry virus-1	Candresse et al. 2013; Matic et al. 2009
Pyrus	Pear corky pit, stony pit	Apple stem pitting virus	Paunović et al. 1999
Strawberry	Strawberry pallidosis	Strawberry pallidosis associated virus, beet pseudo-yellows virus	Tzanetakis et al. 2003, 2004a
Strawberry	Strawberry chlorotic fleck	Strawberry chlorotic fleck–associated virus	Tzanetakis and Martin 2007
Strawberry	Strawberry pseudo mild yellow edge	Strawberry pseudo mild yellow edge	GenBank accession PP763440; Sierra-Mejia and Tzanetakis (*unpublished*)

Peach line pattern and leaf curl

A virus was identified in Hungary and recorded in the early 1980s infecting peach and sweet cherry (Németh et al. 1983), with the leaves of affected trees showing yellowish-green discolorations and wrinkling. It was experimentally transmitted to herbaceous hosts and partially characterized, showing isometric particles of 33-nm diameter and composed of a single, ca. 33-kDa capsid protein subunit. Back inoculation to peach indicated this virus as the causal agent of the disease (Kerlan et al. 1986). Further characterization has not been conducted.

Peach Mexican spot

This disease was identified in France (Desvignes 2004) in a single accession of peach originating from Mexico (Agua 4N6) that showed cross reaction with antiplum pox virus polyclonal antisera (James et al. 1994). HTS has since shown this source to be coinfected by three viruses, Asian prunus virus 1, peach mosaic virus, and CGRMV (T. Candresse, *unpublished*). There is no evidence for the presence of any additional agent. Although it is unclear whether the diffuse chlorotic spotting symptoms observed were caused by a combination of agents or by one of the identified agents, there is no longer a logic in maintaining the separate peach Mexican spot agent terminology because it does not correspond to a distinct virus.

Peach mottle

This disease was identified in few peach trees in Idaho in the late 1930s and early 1940s (Blodgett 1941). Graft transmission achieved infection of peach and sweet and sour cherry cultivars. No natural spread was reported, and the unidentified causal agent was considered of minor significance (Helton 1976).

Peach oil blotch

This is a graft-transmissible disease identified in Japan in 1973 (Kishi et al. 1973b) for which little information is available. Peach latent mosaic viroid (PLMVd) was later detected in all seven evaluated oil blotch sources, leading to the suggestion that PLMVd might be the causal agent (Osaki et al. 1999). However, in these experiments PLMVd was detected in 94% of all peach accessions tested, greatly reducing the solidity of the oil blotch–PLMVd association.

Peach pseudo stunt

A graft-transmissible agent causing stunting without visible leaf symptoms on peach seedlings leaves was identified in Germany (Kegler 1965a). The agent did not show any symptoms on 'Shirofugen' and was not sap transmissible to herbaceous host plants. No new reports have been traced in the literature.

Peach seedling chlorosis

This disease was identified in New Zealand on 'Golden Queen' peach seedling indicators that had been grafted with symptomless plum and cherry samples (Fry and Wood 1973; Wood and Fry 1973). Sections of leaves that developed in late spring exhibited zonate chlorosis, although curiously, the symptoms did not occur on leaves produced during the summer flush. The current status of this disease is unknown.

Peach seedling necrosis

Despite the fact that the disease is identified in the quarantine regulations of several countries, such as New Zealand, the original publication describing the disease could not be identified and there are no reports traced in the literature.

Peach star mosaic

This is a graft-transmissible disease identified in Japan and reported in 1973 for which little information is available (Kishi et al. 1973b) and for which no new reports of the disease have been traced in the literature.

Peach stubby twig/false yellow leaf roll

The disease, shortened internodes and the production of pale green to chlorotic leaves with terminal dieback in later seasons, was first noted in California in 1952 (Wagnon et al. 1958). It was also observed in Missouri and Oregon in the United States, as well as in Italy and Turkey. Graft transmission was possible to apricot, almond, and 'Myrobalan', 'Marianna', and 'Damson' plum without causing symptoms. The agent was shown to be eliminated by a 5-week thermotherapy period at 38°C. Observation indicated very limited, if any, field spread (Wagnon et al. 1976). A superficial resemblance of symptoms with those of peach X-disease was reported, and the vein swelling of affected leaves is reminiscent of phytoplasma infections.

Weak peach

A graft-transmissible agent was identified in a single peach orchard showing weaker-than-normal trees in Georgia, U.S.A., in 1952 (Kenknight 1962). Infected trees were stunted, with delayed spring growth, chlorosis of leaves during summer, and dieback in later seasons. This agent is considered as likely being the same as the disease known as peach short life (Okie et al. 1985), which is apparently caused by a complex of viral, fungal, and nematode infections and cultivation factors (Howell et al. 2011; Kenknight 1976).

Peach yellow mottle

This disease was identified by graft inoculating 'Golden Queen' peach seedlings with a single symptomless apricot from Central Otago in New Zealand (Fry and Wood 1973). No symptoms could be observed upon further propagation on 'Golden Queen' peach seedlings or on a range of *Prunus* indicators, raising the question of the validity of the original disease observation or description. No other disease source was identified since 1975.

Plum chlorosis and wilt

Even though the disease is identified in the quarantine regulations of several countries, including New Zealand, the original publication describing the disease could not be identified and there are no new reports of the disease.

Plum enation mottle

Even though the disease is identified in the quarantine regulations of several countries, including New Zealand, the original publication describing the disease could not be identified and there are no new reports of the disease.

Plum fruit crinkle

This graft-transmissible disease was present on three Japanese plum trees from two orchards in New Zealand in 1951 (Chamberlain et al. 1959). No new reports of the disease have been made in recent years.

Plum mottle leaf

This graft-transmissible disease was identified in New Zealand in 1979 (Wood 1979; Wood and Fry 1984). There is little information available on the disease because the original report could not be accessed. A report from 1998 in the United States indicates that a source obtained from New Zealand induced symptoms in the *Prunus tomentosa* indicator (Damsteegt et al. 1998).

Plum ochre mosaic

This disease was identified in the former Czechoslovakia and reported in 1961 as both graft and aphid transmissible by *Myzus persicae* (Blattný 1961). No new reports have been identified in recent years in any country, and there is no information on the causal agent.

Plum ringspot and shot hole

Even though the disease is identified in the quarantine regulations of several countries, including New Zealand, neither the original publication describing the disease nor any other report could be identified.

Plum white spot

A disease very loosely described from Californian plums in 1939 (Thomas and Rawlins 1939) in a paper that only provided symptoms

description and an indication of graft transmissibility to 'Golden Queen' peach seedlings. This disease was not mentioned in the large-scale synthesis of virus diseases and noninfectious disorders of stone fruits in North America published in 1976 (Gilmer et al. 1976).

Prune diamond canker

Diamond canker was noted from French (Agen) prune in California in 1915. Investigations culminated in a report in 1941 providing evidence of its viral nature in 1941 (Smith 1941). The incidence of the disease then decreased by more careful selection of budwood, and there was little further research. Grafting experiments showed that other *Prunus* species did not develop symptoms (Wagnon and Williams 1976).

Sour cherry bark splitting

The disease was recorded on 'Montmorency' sour cherry in Oregon in 1954 (Cameron 1954). When graft transmitted to apricot, the symptoms are reported to be similar to those of apricot gummosis (Blodgett and Twomey 1958). No symptoms are recorded on graft-inoculated sweet cherry, Italian plum, Japanese plum, flowering cherry, and peach seedlings (Cameron 1976).

Sour cherry gummosis

This disease was discovered through the indexing of seven symptomless *Prunus mahaleb* trees from the U.S. Pacific Northwest on 'Montmorency' sour cherry (Blodgett et al. 1964). Natural spread of the disease has been recorded, but since 1976 the graft-transmissible agent had only been recorded in the seven original trees (Blodgett and Aichele 1976b).

Sour cherry line pattern

The disease was identified in the former Czechoslovakia (Paulechova 1968). Little information is available because the original report could not be accessed and there are no other further reports of the disease.

Sour cherry pink fruit

The disease was first recorded in Washington state, U.S.A., in 1934 and shown to be associated with a graft-transmissible agent in 1940 (Reeves 1943). Sweet cherry indicators are symptomless carriers, whereas Italian plum, peach, and *P. mahaleb* are reported as unsusceptible. In 1976 a synthesis indicated that "some diseased trees are still found scattered [mostly] in backyard orchards" (Cheney et al. 1976). The timing and appearance of symptoms as well as plant issues affected are indicative of X-disease ('*Candidatus* Phytoplasma pruni'), although the pink fruit agent was described as asymptomatic in many other cherry and peach cultivars (Parish 1995).

Sour cherry rusty splitting

Despite the fact that the disease is identified in the quarantine regulations of several countries, the original publication describing the disease could not be identified and there are no reports of the disease.

Strawberry (*Fragaria*)

Strawberry latent C virus

In a virus survey using graft indexing, Demaree and Marcus (1951) described two symptom types. Type 2 symptoms eventually became type 2 virus, with Smith (1952) reporting it to be aphid transmissible in a persistent manner. McGrew (1956) followed up on the Demaree and Marcus symptom types and determined that an agent named latent C could cause symptoms on *Fragaria vesca* in mixed infections with latent A (now strawberry crinkle virus). Further studies revealed that latent C was able to cause symptoms in some *F. vesca* East Malling Clone (EMC) clones and not in others. Yoshikawa and Inouye (1989) identified latent C in Japan based on the fact that it only caused symptoms on *F. vesca* indicator UC-5. However, the virus is supposed to cause symptoms (epinasty, reduced runner numbers, chlorosis, dwarfing) on several *F. vesca* and *F. virginiana* indicators. Those *F. vesca* plants were infected by a nucleorhabdo-like virus. There are clones labeled as latent C in the National Clonal Repository in Oregon, but multiple HTS analyses failed to identify any new agents in those plants (Diaz-Lara et al. 2021; R.R. Martin and I.E. Tzanetakis, *unpublished*). Given the inconsistencies of the agent description and the exhaustive analysis of "infected" clones that were labeled as latent C, it was concluded that characterization of latent C virus as described in the literature is not currently possible.

Strawberry feather leaf/sparkle virus

Feather leaf is one of the most elusive agents reported in strawberry. It causes marginal chlorosis in *F. virginiana* clones UC-10 and -12, whereas in *F. vesca* clones, it causes dwarfing and strap-like leaves with serrated margins. Yet those symptoms are not diagnostic unless there is also vein clearing and translucent spots on young leaves. McGrew (1970) noticed that feather leaf does not affect the symptoms caused by aphid-borne viruses, and Frazier (1974) hypothesized that the disease has multiple causal agents. Furthermore, there is no information on the natural transmission of the agent and no known source/clone of a feather leaf plant, possibly because symptoms are not persistent and, after the initial acute stage, newly emerged leaves appear normal.

Strawberry vein necrosis virus (Nepo 1)

In a virus survey by Stingl and King (1965) in Minnesota, U.S.A., a single plant of presumed cultivar 'Champion' displayed severe virus-like symptoms including vein chlorosis, necrosis, and epinasty during fall and winter. During summer, symptoms—other than reduced vigor—disappeared. The agent was graft transmissible to *F. vesca*, *F. virginiana*, and two strawberry (*F.* × *ananassa*) cultivars; mechanically transmissible to 14 indicator plant species but not transmissible by *Chaetosiphon fragaefolii* or *Amphorophora rubi* aphids. The physical properties of the purified virus align with those of nepoviruses infecting berry crops.

Strawberry Nepo 2

Canova and Tacconi (1965) identified an agent from field-collected plants through mechanical inoculations and grafting onto strawberry and herbaceous indicators. Strawberries developed symptoms similar, but not identical, to those reported for strawberry vein necrosis including vein banding, chlorosis, and epinasty. The host range of the putative virus did overlap with that of vein necrosis, yet symptoms on bean, the primary indicator host for the virus, were quite distinct and included stem galls, something not observed in vein necrosis. Virus purification yielded infectious material that could be inoculated to bean and had similar physical properties to nepoviruses. Yet this is the only report of the agent.

Strawberry band mosaic virus

This disease was described once in an old cultivar in Hungary in the 1960s. The cultivar Hl. Anton von Padua displayed band mosaic symptoms that resembled strawberry vein banding based on the single picture present in the publication (Maassen and Németh 1961). The graft-transmissible putative virus failed to be transmitted mechanically or by aphids. Given the limited information provided in the single report, the agent cannot be studied further.

Strawberry necrosis virus

The necrosis agent was mechanically transmitted to bean and other indicators from an *F. vesca* EMC that was aphid and graft inoculated with a vein necrosis agent (Maasen 1959). Back inoculations were unsuccessful, and thus, the indicator-infecting virus and the virus causing symptoms on strawberry may not be the same, especially when considering that the bean-infecting virus was not aphid transmissible. Other than some physical properties of the agent, no other information is available, making it impossible to characterize it any further. The necrosis agent should not be confused with strawberry necrotic shock virus.

Noninfectious phyllody disease of strawberry

This disease has been observed in several European countries and Australia. The symptoms of affected plants strongly resemble those induced by phytoplasmas. Attempts to transmit the disease to periwinkle or healthy strawberry plants were unsuccessful. Symptomatic plants tested negative for phytoplasma infection using fluorescence microscopy and direct and nested PCR assays. Thus, the disease is likely to be of physiological etiology (Marcone et al. 1996, 1998).

Strawberry leaf curl virus

In 2008, a begomovirus-like disease was observed in *Fragaria* in Egypt (El-gaied et al. 2008). Infected plants tested positive by enzyme-linked immunosorbent assay and PCR for general detection of whitefly-transmitted geminiviruses. The disease was transmitted by *Bemisia tabaci* (sweet potato or silverleaf whitefly) under experimental conditions and by mechanical transmission to tomato (*Solanum lycopersicum*). Begomovirus-like particles were observed using electron microscopy. Particles were isolated and the coat protein molecular mass was ~32 kD. A tomato yellow leaf curl virus (TYLCV) PCR showed that TYLCV was not the causal agent of the observed disease. No sequencing technique was used during this study, so no sequencing information is available and there are no known isolates of the virus. This study showed that a begomovirus that was not TYLCV could infect strawberry, but it is unclear whether this was a previously unreported virus or one of the characterized begomoviruses. No begomoviruses have been reported in strawberry since 2008.

Discussion

This communication provides evidence that many diseases and/or agents reported in the scientific literature must be considered "phantom agents," because no known isolates are available in pathogen/disease collections or elsewhere, and sequence data are lacking. This implies that no further studies can be performed nor diagnostic tests developed.

In international standards, the basic criterion for a pathogen to be considered as either a quarantine pest or a regulated nonquarantine pest during a pest risk analysis is the characterization of the pest to ensure that it has an unambiguous taxonomic position or, in case the agent has not been characterized, that a specific and reliable assay for the detection of the disease is available (IPPC 2004, 2006). The phantom agents listed herein were mostly postulated as phytopathogens before the routine application of molecular diagnostics and sequencing technologies. In many cases, these findings included some preliminary biological characterization but lack supporting data to enable unambiguous detection or taxonomic classification of the agents. This can lead to confusion over the identity of the agent(s). In some cases, the pathogen may be rediscovered by sequencing many years after the initial description with no way to connect the new findings to the original record (Jones et al. 2021). There are multiple examples emerging where pathogens have been regulated under a tentative name and later, through sequencing, shown to be another, already characterized pathogen (Table 1). There have also been examples where newly discovered pathogens with molecular characterization are considered for regulation and later shown to be synonymous with previously reported yet unsequenced pathogens (EFSA Panel on Plant Health (PLH) et al. 2019; Hammond et al. 2021; Sabanadzovic et al. 2011). In both scenarios there may have been regulatory listing of pathogenic agents, leading to trade barriers.

There have been concerted attempts to generate sequence data from historical virus isolate collections to link to these phantom agents (Table 1), and in certain cases, Koch's postulates have been fulfilled by using infectious clones, as shown for apple russet ring and apple green crinkle diseases caused by isolates of ACLSV and apple stem pitting virus, respectively (Li et al. 2020).

Contemporary samples with similar maladies have been investigated using HTS approaches. For example, Rott et al. (2018) investigated the uncommon apple rubbery wood disease and the associated flat limb disease. This investigation revealed two novel viruses, apple rubbery wood virus 1 and apple rubbery wood virus 2, in affected samples, often in association with other common viruses. Yet demonstrating causal associations by fulfilling Koch's postulates can be challenging because of confounding coinfections, without mentioning that epidemiological observations may not be possible for rare diseases (Evans 1976; Fox 2020; Fredricks and Relman 1996). Given the discontinuity in the criteria between the pre- and postsequencing diagnostic eras for agents listed here, there is a need to revise them for selecting a regulated pest. The revisions should consider both biological and molecular data as well as evidence of pathogenicity and impact, before a pest is added to the regulatory list. In any event, none of the phantom agents highlighted here would meet the current or revised criteria.

Many phantom agents could be considered "historical curiosities." Indeed, phantom agents are often linked to specific varietal reactions, and in many cases, they have ceased to be observed for several decades either because the cultivars involved have fallen out of popularity or possibly because the agent has been eliminated through improved sanitary practices and the expansion of clean plant programs and certification schemes (Fuchs et al. 2021; Gergerich et al. 2015; Martin et al. 2016). With the gradual adoption of HTS in support of certification (Maree et al. 2018), it is likely that these agents, if still present in varietal stocks, will be detected and recharacterized. At the same time, the increased use of HTS analysis has led and will lead to the discovery of an increasing number of novel viruses and other agents, thereby creating a new pitfall as there is a risk that these will become regulated as pests on the basis of sequence data only. For this reason a framework has been developed to recommend the type of biological characterization that should be generated to support regulatory decisions (Fontdevila Pareta et al. 2023).

We collectively aim to mainstream the movement of clean, high-quality propagation material to improve yields and assist stakeholders, from the producers who will have access to a wider selection of clean material to grow better and more productive crops to consumers who will be able to choose from a greater variety of high-quality fruits. It is anticipated that this communication will facilitate the establishment of more synergistic interactions between the scientific and regulatory communities so that regulatory standards will resonate with producers and enhance their level of confidence in our collective ability to accompany the safe exchange of germplasm they desire.

We wish policymakers and regulatory authorities consider the present evidence for excluding phantom agents from regulatory standards. It is important to set sound and realistic standards that take into consideration the latest advancement in diagnostics and disease biology and ecology. It is equally important to collectively learn the lessons from past mistakes and continuously refine standards based on scientific progress. This list of phantom agents for eight major crops is extensive. However, we believe that science and pragmatism should guide the establishment of regulatory standards. This is critical to facilitate the safe exchange of germplasm, while simultaneously reducing barriers to trade, and to reduce the costs and delays of certification programs. If current regulations are not revised, producers, who often have little trust in current standards, will continue circumventing them to access their desired germplasm via illegal "suitcase" introductions. Illegal introductions are well documented to have repeatedly caused agriculture disasters across the globe (Dimou et al. 2002; Levy et al. 2000). In conclusion, this article provides valuable information for evaluating current regulation, to improve compliance with future scientifically substantiated regulation and effective protection of fruit crops against harmful diseases.

Furthermore, if, despite the extensive research carried out in support of this review, reference material of one of these phantom agents is still available in any local repository, the authors would be grateful for any feedback to further investigate their existence or etiology. To prevent future problems of the emergence of possible new phantom agents, we also call for the public submission of

material of newly described agents to appropriate collections or bioresource centers for further investigations and for securing and establishing necessary reference material.

Authors' note

For an extensive literature review dating back to 1913 and a research summary of the past 20 years concerning 55 suspected citrus phantom disorders, please refer to the article by Aknadibossian et al. (2023) - https://gd.eppo.int/reporting/article-7741.

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