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Streamlining global germplasm exchange: Integrating scientific rigor and common sense to exclude phantom agents from regulation

Tzanetakis, I.E.^{1*} ▼ORCID: 0000-0002-5970-1763, Aknadibossian, V.² ▼ORCID: 0000-0002-4784-517X, Špak, J.^{3*} ▼ORCID: 0000-0002-2832-0853, Constable, F.^{4*} ▼ORCID: 0000-0002-8165-7500, Harper, S.J. ^{5*} ▼ORCID: 0000-0002-9355-2431, Hammond, J.^{6*} ▼ORCID: 0000-0001-7593-747X, Candresse, T.^{7*} ▼ORCID: 0000-0001-9757-1835, Folimonova, S. Y.² ▼ORCID: 0000-0001-7297-2253, Freitas-Astúa J.⁸ ▼ORCID: 0000-0002-0506-6880, Fuchs, M.⁹ ▼ORCID: 0000-0001-5332-6766, Jelkmann, W.¹⁰ ▼ORCID: 0000-0001-8073-807X, Maliogka, V.I.¹¹ ▼ORCID: 0000-0001-5714-2710, Marais, A.⁷ ▼ORCID: 0000-0003-2482-1543, Martin, R.R.¹² ▼ORCID: 0000-0002-2045-5470, Mollov, D.¹³ ▼ORCID: 0000-0002-4750-6194, Vidalakis, G.^{*14} ▼ORCID: 0000-0001-5821-7112, Aboughanem-Sabanadzovic, N.¹⁵ ORCID: 0000-0002-6141-3218, Al Rwahnih, M.¹⁶ ORCID: 0000-0003-1589-9234, Alabi, O.J.¹⁷ ORCID: 0000-0002-2471-7052, Alioto, D.¹⁸ ORCID: 0000-0002-4954-8363, Atanda H.Y. ¹ ORCID: 0000-0002-1215-8847, Bagi, F. ¹⁹ ORCID: 0000-0002-8646-1072, Baranwal, V.K. ²⁰ ORCID: 0000-0002-5251-0905, Barbosa, C.⁸, Bar-Joseph, M.²¹, Batista Le Riverend, L.²², Belien, T.²³ ORCID: 0000-0003-1274-4042, Benítez-Galeano, M.J.²⁴ ORCID: 0000-0002-6332-1632, Bennypaul, H.²⁵, Bertaccini, A.²⁶ ORCID: 0000-0002-5650-1512, Bester, R.²⁷ ORCID: 0000-0002-2226-0968, Blouin, A.G.²⁸ ORCID: 0000-0003-1360-1529, Blystad, D.-R.²⁹ ORCID: 0000-0001-5490-3334, Botermans, M.³⁰ ORCID: 0000-0002-7761-3855, Bozan, O.³¹ ORCID: 0000-0002-3274-5459, Brakta, A.³² ORCID: 0000-0002-5367-8689, Brans, Y.³³ ORCID: 0000-0002-6999-503X, Bulajić, A.³⁴ ORCID: 0000-0001-7680-9664, Caglayan, K.³⁵, Catara, A.³⁶ ORCID: 0000-0001-6551-203X, Choueiri, E.³⁷ ORCID: 0000-0001-8762-493X, Cieślińska, M.³⁸ ORCID: 0000-0001-6023-9070, Cook, G.³⁹ ORCID: 0000-0003-1702-479X, Cui, W.⁴⁰, da Graça, J.⁴¹, Davino, S.⁴² ORCID: 0000-0002-5926-6300, Delmiglio, C.⁴³ ORCID: 0000-0001-8959-7736, Dewdney, M.⁴⁴ ORCID: 0000-0003-1905-7616, Di Serio, F.⁴⁵ ORCID: 0000-0003-2822-704X, Diaz-Lara, A.⁴⁶ ORCID: 0000-0002-4736-8019, Digiaro, M.⁴⁷ ORCID: 0009-0006-3089-3606, Djelouah, K.⁴⁷, Donovan, N.⁴⁸ ORCID: 0000-0003-0941-2485, Druciarek, T.Z.⁴⁹ ORCID: 0000-0001-5607-1885, Duran-Vila, N.⁵⁰, Elçi, E.⁵¹ ORCID: 0000-0002-6434-6321, Esquivel-Fariña, A.⁵² ORCID: 0000-0002-7727-5243, Fall, M.L.⁵³ ORCID: 450-210-1847, Figueroa, J.⁵⁴, Fiore, N.⁴⁰, Fowkes, A.R.⁵⁵ ORCID: 0000-0001-7918-441X, Fox, A.⁵⁵ ORCID: 0000-0003-1280-1836, Fránová, J.³ ORCID: 0000-0001-8451-1454, Fuchs, R.⁵⁶ ORCID: 0000-0001-5056-5323, Gaafar, Y.Z.A.²⁵ ORCID: 0000-0002-7833-1542, García, M.L.⁵⁷ ORCID: 0000-0001-6468-6943, Ghosh, D.⁵⁸, Girardi, E.⁸, Glasa, M.⁵⁹ ORCID: 0000-0002-8495-7971, Gomez Talquenca, S.⁶⁰ ORCID: 0000-0001-9265-8333, Gratz, A.²⁵ ORCID: 0000-0002-2655-5755, Gritsenko, D.⁶¹ ORCID: 0000-0001-6377-3711, Hajeri, S.⁶² ORCID: 0000-0002-5993-118X, Hajizadeh, M.⁶³ ORCID: 0000-0002-0774-3523, Hamborg, Z.²⁹ ORCID: 0000-0001-7070-6621, Ho, T.⁶⁴ ORCID: 0000-0003-0914-306X, Holeva, M.⁶⁵ ORCID: 0000-0001-8645-5943, Holkar, S.K.⁶⁶ ORCID: 0000-0001-9197-8765, Horner, M.⁶⁷, Hurtado-Gonzales, O.P.⁶⁸ ORCID: 0000-0002-9561-7016, Ippolito, A.⁶⁹, Isac, V.⁷⁰ ORCID: 0000-0003-1789-1359, Iwanami, T.⁷¹, Jofre-y-Garfias, A.E.⁷², Jordan, R.⁶ ORCID: 0000-0003-1105-4066, Katis, N.¹¹, Koloniuk, I.³ ORCID: 0000-0002-5893-6683, Konings, H.⁷³, Križanac, I.⁷⁴ ORCID: 0000-0001-9185-8341, Krueger, R.⁷⁵ ORCID: 0000-0003-2570-7881, Kyrychenko, A.⁷⁶ ORCID: 0000-0002-6098-1467, Laranjeira, F.⁸ ORCID: 0000-0002-3584-6132, Lavagi-Craddock, I.¹⁵, Levy, A.⁴⁴ ORCID: 0000-0002-2846-9009, Licciardello, G.⁷⁷, Lu, Q.-Y.⁷⁸, MacFarlane, S.A.⁷⁹, Marcone, C.⁸⁰ ORCID: 0000-0002-1847-5296, Maree, H.J.²⁷ ORCID: 0000-0001-9639-4558, Margaria, P.⁸¹ ORCID: 0000-0001-8670-1331, Martić, A.⁸² ORCID: 0000-0003-4606-9735, Massart, S.⁸³ ORCID: 0000-0002-7153-737X, Mathioudakis, M.M.⁸⁴ ORCID: 0000-0002-8008-0228, Matić, S.⁸⁵ ORCID: 0000-0002-0885-8744, Mavric Plesko,

I.⁸⁶ORCID: 0000-0003-2874-3562, Meekes, E.T.M.⁷³, Mehle, N.⁸⁷ORCID: 0000-0002-3028-0280, Melzer, M.J.⁸⁸ORCID: 0000-0003-0390-3857, Meng, B.⁸⁹, Menzel, W.⁸¹ORCID: 0000-0002-3614-161X, Miljanić, V.⁹⁰ORCID: 0000-0002-4398-3364, Minafra, A.⁴⁵ORCID: 0000-0002-0547-7129, Minutolo, M.⁹¹ORCID: 0000-0001-7816-2657, Mitra, A.¹⁵, Moreno, P.⁵⁰ORCID: 0000-0003-3091-6207, Navarro, L.⁵⁰ORCID: 0000-0001-5163-5960, Navarro, B.⁴⁵ORCID: 0000-0001-8981-1018, Nerva, L.⁹², Okić, A.⁹³ORCID: 0000-0003-4609-4527, Olmos, A.⁵⁰ORCID: 0000-0001-8406-7963, Onelge, N.³¹, Osundahunsi, B.¹ORCID: 0000-0002-2454-5887, Palacios, M.F.⁵⁴, Pallas, V.⁹⁴ORCID: 0000-0003-4954-989X, Panno, S.⁴²ORCID: 0000-0002-8941-7050, Perez-Egusquiza, Z.⁴³, Poudel-Ward, B.⁹⁵ORCID: 0000-0002-2365-2859, Radišek, S.⁹⁶ORCID: 0000-0002-9333-7985, Ramos-González, P.L.⁹⁷ORCID: 0000-0002-9640-6587, Ramteke, P.⁹⁸, Ranabhat, N.B.⁹⁹ORCID: 0000-0001-5929-9578, Rivarez, M.P.S.¹⁰⁰ORCID: 0000-0002-8544-5740, Rivas, F.¹⁰¹, Roenhorst, A.³⁰ORCID: 0009-0004-6029-0801, Roy, A.¹⁰²ORCID: 0000-0002-2268-9713, Ruiz-García, A.B.⁵⁰ORCID: 0000-0001-5704-2138, Sabanadzovic, S.¹⁰³ORCID: 0000-0002-2995-2633, Šafářová, D.¹⁰⁴ORCID: 0000-0002-9677-0530, Saldarelli, P.⁴⁵ORCID: 0000-0002-8633-7813, Salem, N.¹⁰⁵ORCID: 0000-0003-3778-2213, Sanahuja Solsona, G.¹⁰⁶, Schoen, R.³⁰ORCID: 0000-0003-4135-609X, Sharma, S.K.²⁰ORCID: 0000-0002-9114-4231, Shilts, T.⁴⁴ORCID: 0000-0002-3872-3891, Sierra-Mejia, A.¹ORCID: 0000-0003-0893-5882, Singh, S.¹ORCID: 0000-0002-2053-3838, Skelton, A.⁵⁵ORCID: 0000-0002-5418-1930, Škorić, D.¹⁰⁷ORCID: 0000-0002-1625-1598, Stainton, D.¹ORCID: 0000-0002-7472-6029, Štajner, N.⁹⁰, Starović, M.¹⁰⁸ORCID: 0000-0002-1700-2702, Stuchi, E.⁸, Svoboda, P.¹⁰⁹ORCID: 0000-0001-6368-7039, Tahzima, R.⁸³ORCID: 0000-0002-0115-8366, Tang, J.⁴³ORCID: 0000-0001-9876-572X, Tessitori, M.¹¹⁰ORCID: 0000-0003-4285-8569, Thermo, J.-P.¹¹¹ORCID: 0000-0001-5494-3096, Thompson, E.¹¹², Thompson, J.⁴³, Trebicki, P.¹¹³ORCID: 0000-0002-7075-6650, Turina, M.⁸⁵ORCID: 0000-0002-9659-9470, Ulubas Serce, C.⁵¹ORCID: 0000-0001-5337-5883, Umble, J.¹⁰⁶, Valiunas, D.¹¹⁴ORCID: 0000-0001-7625-3766, Varallyay, E.¹¹⁵ORCID: 0000-0002-9085-942X, Varma, A.²¹, Varveri, C.⁶⁵ORCID: 0000-0001-6317-0734, Vasquez-Gutierrez, U.¹¹⁶ORCID: 0000-0001-8281-5075, Vazquez-Iglesias, I.⁵⁵ORCID: 0000-0003-1258-1810, Veerakone, S.⁴³, Villamor, D.E.¹, Vives, M.C.⁵⁰ORCID: 0000-0001-6850-9696, Vončina, D.¹¹⁷ORCID: 0000-0001-6093-5471, Wang, J.¹³, Wenxing, X.¹¹⁸ORCID: 0000-0002-5984-5724, Westenberg, M.³⁰ORCID: 0000-0002-6458-9809, Wetzel, T.¹¹⁹ORCID: 0000-0003-1559-975X, Winter, S.⁸¹ORCID: 0000-0003-0309-9934, Wright, G.⁹⁵, Wulff, N.A.¹²⁰ORCID: 0000-0002-4557-2075, Xu Dong, F.¹²¹ORCID: 0000-0001-9385-1928, Ya Feng, D.¹²⁰, Yokomi, R.¹²²ORCID: 0000-0002-5658-2279, Zhou, C.¹²³, Zikeli, K.¹⁰ORCID: 0009-0007-2324-9991, Zindović, J.¹²⁴ORCID: 0000-0002-3193-950X

1. Dept. of Entomology and Plant Pathology, University of Arkansas System Division of Agriculture
2. University of Florida, Department of Plant Pathology, Gainesville, FL 32611, USA
3. Biology Centre, Czech Academy of Sciences, České Budějovice, Czech Republic
4. Agriculture Victoria Research, Department of Energy, Environment and Climate Action
5. Dept. of Plant Pathology, Washington State University
6. Floral and Nursery Plants Research, US National Arboretum, USDA-ARS
7. Université de Bordeaux, INRAE, UMR BFP, Villenave d'Ornon, France
8. Embrapa Cassava and Fruits, Cruz das Almas, BA, Brazil
9. Plant Pathology, Cornell University, Geneva, NY 14456, USA
10. Julius Kühn-Institute, Institute for Plant Protection in Fruit Crops and Viticulture, Dossenheim, Germany

11. Plant Pathology Laboratory, School of Agriculture, Aristotle University of Thessaloniki, Greece
12. Department of Botany and Plant Pathology, Oregon State University, Corvallis, Oregon 97339
13. USDA-APHIS-Plant Protection and Quarantine, Riverdale, MD 20737
14. Department of Microbiology and Plant Pathology, University of California, Riverside, CA, USA
15. Institute for Genomics, Biocomputing and Biotechnology, Mississippi State University, USA
16. Foundation Plant Services, Dept. of Plant Pathology, University of California-Davis, Davis, USA
17. Dept. of Plant Pathology & Microbiology, Texas A&M AgriLife Research and Extension Center, Weslaco, TX 78596, USA
18. Dept. of Agricultural Sciences, University of Naples Federico II
19. Department of Plant and Environmental Protection, Faculty of Agriculture, University of Novi Sad, Serbia
20. Advanced Centre of Plant Virology, ICAR-Indian Agricultural Research Institute, New Delhi -110012, India
21. The Volcani Center, Israel
22. Grupo de Fitopatología, Instituto de Investigaciones en Fruticultura Tropical, La Habana, Cuba
23. Proefcentrum Fruitteelt vzw (pcfruit vzw), Zoology Department, 3800 Sint-Truiden, Belgium
24. Unidad de Genómica y Bioinformática, Departamento de Ciencias Biológicas, Universidad de la República, CENUR Litoral Norte, Salto, Uruguay.
25. The Sidney Laboratory - Centre for Plant Health, Canadian Food Inspection Agency (CFIA), 8801 East Saanich Road, North Saanich, British Columbia V8L 1H3, Canada
26. Alma Mater Studiorum, University of Bologna, Italy
27. Department of Genetics, Stellenbosch University, Private Bag X1, Matieland 7602, South Africa
28. Research Group Virology, Bacteriology and Phytoplasmology, Plant Protection Department, Agroscope, Nyon, Switzerland
29. NIBIO - Norwegian Institute of Bioeconomy Research, Ås, Norway
30. Netherlands Institute for Vectors, Invasive plants and Plant health (NIVIP), National Plant Protection Organization (NPPO), Netherlands Food and Consumer Product Safety Authority (NVWA), Geertjesweg 15, 6706 EA Wageningen, The Netherlands
31. Department of Plant Protection, Agriculture Faculty, Cukurova University 01330/Adana/Turkiye
32. Plant Virology Laboratory, Department of Plant Pathology, Dr. Yashwant Singh Parmar University of Horticulture & Forestry, Nauni Solan. Himachal Pradesh. 173230. India.
33. CTIFL, Laboratoire de virologie fruitière, Centre de Lanxade, 24130 Prigonrieux, France
34. Department of Phytopathology, Institute of Phytomedicine, University of Belgrade – Faculty of Agriculture, 11080 Belgrade, Serbia
35. Department of Plant protection, Agriculture Faculty, Hatay Mustafa Kemal University, Turkeye
36. Formerly, Science and Technology Park of Sicily, ZI Blocco Palma I, Via V. Lancia 57, 95131 Catania, Italy
37. Department of Plant Protection, Lebanese Agricultural Research Institute, Tal Amara, P.O.Box 287, Zahlé, Lebanon
38. Department of Plant Pathology, The National Institute of Horticultural Research, Skierniewice, Poland
39. Citrus Research International, Disease Management, Graft Transmissible Diseases, South Africa

- 132 40. Facultad de Ciencias Agronómicas, Universidad de Chile, Avenida Santa Rosa 11315, Santiago 8820808, Chile
133 41. Texas A&M University-Kingsville, USA
134 42. Department of Agricultural, Food and Forest Science - University of Palermo, Italy
135 43. Plant Health & Environment Laboratory, Ministry for Primary Industries, New Zealand
136 44. Citrus Research and Education Center and Department of Plant Pathology, University of Florida, Lake Alfred, FL
137 33850, USA
138 45. Institute for Sustainable Plant Protection-CNR, Bari, Italy
139 46. School of Engineering and Sciences, Tecnológico de Monterrey, Mexico
140 47. CIHEAM, Istituto Agronomico Mediterraneo di Bari, Italy
141 48. New South Wales Department of Primary Industries, Australia
142 49. Department of Plant Protection, Warsaw University of Life Sciences, Poland
143 50. Instituto Valenciano de Investigaciones Agrarias, 46113 Moncada, Valencia, Spain
144 51. Department of Plant Production and Technologies, Niğde Ömer Halisdemir University, Türkiye
145 52. Facultad de Ciencias Agrarias (FCA) de la Universidad Nacional de Asunción (UNA), Paraguay
146 53. Agriculture and Agri-Food Canada/Université de Sherbrooke, Canada
147 54. Estación Experimental Agroindustrial Obispo Colombres, Argentina
148 55. Fera Science Ltd, York Biotech Campus, York, United Kingdom
149 56. State Institute of Viticulture and Oenology (WBI), Freiburg, Germany
150 57. Instituto de Biotecnología y Biología Molecular, Universidad Nacional de La Plata-CONICET, La Plata, Argentina
151 58. ICAR – Central Citrus Research Institute, India
152 59. Institute of Virology, Biomedical Research centre, Slovak Academy of Sciences, Bratislava, Slovakia
153 60. Instituto Nacional de Tecnologia Agropecuaria - EEA Mendoza, Mendoza, Argentina
154 61. Laboratory of molecular biology, Institute of plant biology and biotechnology, Kazakhstan
155 62. Alliance of Pest Control Districts
156 63. Department of Plant Protection, Faculty of Agriculture, University of Kurdistan, Sanandaj, Iran
157 64. Driscoll's Inc, Watsonville, California, USA
158 65. Benaki Phytopathological Institute, Scientific Directorate of Phytopathology, Laboratory of Bacteriology, Athens,
159 Greece
160 66. ICAR-National Research Centre for Grapes, Pune, Maharashtra, India
161 67. The New Zealand Institute for Plant and Food Research Limited, New Zealand
162 68. United States Department of Agriculture, Animal Plant Health and Inspection Services, Beltsville, Maryland,
163 20705, USA
164 69. Department of Soil, Plant, and Food Sciences, University of Bari Aldo Moro, Italy
165 70. Research Institute for Fruit Growing, Pitesti, Arges, Romania
166 71. Faculty of Agriculture, Tokyo University of Agriculture, Funako 1737, Atsugi, Kanagawa 243-0034, Japan
167 72. Agrogenomic Sciences, National School of Higher Studies Leon Unit, National Autonomous University of Mexico
168 73. Naktuinbouw, Sotaweg 22, 2371 GD Roelofarendsveen, The Netherlands
169 74. Centre for Plant Protection, Croatian Agency for Agriculture and Food, Zagreb, Croatia

- 170 75. USDA-ARS-NCGRCD, 1060 Martin Luther King Blvd, Riverside, CA 92507
171 76. Plant Virus Laboratory, Institute of Microbiology and Virology, National Academy of Sciences of Ukraine, Kyiv,
172 Ukraine
173 77. CREA Olivicoltura, Frutticoltura e Agrumicoltura, Acireale (CT), Corso Savoia 190, 95024 Acireale (CT), Italy
174 78. Department of Moriculture, Jiangsu University of Science and Technology, Zhenjiang, Jiangsu, China
175 79. Cell and Molecular Sciences Department, The James Hutton Institute, Dundee DD2 5DA, UK
176 80. Department of Pharmacy, University of Salerno, I-84084 Fisciano (Salerno), Italy
177 81. Leibniz-Institute DSMZ, German Collection of Microorganisms and Cell Cultures, Plant Virus Department,
178 Braunschweig, Germany
179 82. Development sector, 13. jul Plantaže ad. Podgorica, Montenegro
180 83. Plant Pathology laboratory, Gembloux Agro-Bio Tech, Liège University, Belgium
181 84. Institute of Olive Tree, Subtropical Crops and Viticulture, Greece
182 85. Institute for Sustainable Plant Protection-CNR, Torino, Italy
183 86. Agricultural Institute of Slovenia, Ljubljana, Slovenia
184 87. National Institute of Biology, Slovenia
185 88. University of Hawaii, Honolulu, HI 96822, USA
186 89. Department of Molecular and Cellular Biology, University of Guelph, Guelph, Ontario, Canada
187 90. Department of Agronomy, Biotechnical Faculty, University of Ljubljana, Slovenia
188 91. Dept. of Agricultural Sciences, University of Naples Federico II
189 92. Council for Agricultural Research and Economics, Research Centre for Viticulture and Enology, Conegliano, Italy
190 93. University of Sarajevo, Faculty of Agriculture and Food Sciences, Sarajevo, Bosnia and Herzegovina
191 94. Instituto de Biología Molecular y Celular de Plantas, Consejo Superior de Investigaciones Científicas, Universitat
192 Politècnica de Valencia, Valencia, Spain
193 95. School of Plant Sciences - Yuma Agriculture Center, 6425 W. 8th Street, Yuma, University of Arizona, AZ 85364
194 96. Department of Plant Protection, Slovenian Institute of Hop Research and Brewing, Slovenia
195 97. Instituto Biológico, Avenida Conselheiro Rodrigues Alves, 1252, Vila Mariana, 04014-900, São Paulo, Brazil
196 98. Department of Biotechnology, Hislop College, Civil Lines, Nagpur 440001, India
197 99. Dept. of Entomology and Plant Pathology, University of Tennessee, Knoxville, TN
198 100. Philippine Genome Center-Visayas, Road 8, Miagao, 5023, Iloilo, Philippines Caraga State University, Butuan
199 City, 8600, Agusan del Norte, Philippines
200 101. National Institute of Agricultural Research, Uruguay
201 102. Molecular Plant Pathology Laboratory, Beltsville Agricultural Research Center, Agricultural Research Service,
202 United States Department of Agriculture, Beltsville, MD 20705, USA
203 103. Department of Biochemistry, Molecular Biology, Entomology and Plant Pathology, Mississippi State University,
204 USA
205 104. Department of Cell Biology and Genetics, Faculty of Science, Palacký University Olomouc, Czech Republic
206 105. Department of Plant Protection, School of Agriculture, the University of Jordan, Amman 11942, Jordan
207 106. Fall Creek Farm and Nursery Inc, Oregon, USA

107. Department of Biology, Faculty of Science, University of Zagreb, Croatia
108. Institute for Plant Protection and Environment, Belgrade, Serbia
109. Hop Research Institute Co., Ltd., Kadanska 2525, 438 14 Zatec, Czech Republic
110. Department of Agriculture, Food and Environment (Di3A), University of Catania, Italy
111. INRAE, Agap Antenne Corse, 20230, San Giuliano, France
112. Hortifrut Genetics Ltd., Dublin, Ireland
113. Applied BioSciences, Macquarie University, Sydney, New South Wales, Australia
114. Laboratory of Plant Pathology, Nature Research Centre, Akademijos St. 2, LT-08412 Vilnius, Lithuania
115. Genomics Research Group, Department of Plant Pathology, Institute of Plant Protection, Hungarian University of Agriculture and Life Sciences, Hungary
116. Plant Virology Area, Department of Agricultural Parasitology, Universidad Autonoma Agraria Antonio Narro, Coahuila, Mexico
117. Division of Phytomedicine - Department of Plant Pathology, University of Zagreb Faculty of Agriculture, Croatia
118. College of Plant Science and Technology, Huazhong Agricultural University, Wuhan 430070, China
119. DLR Rheinpfalz, Institute of Plant Protection, Neustadt an der Weinstrasse, Germany
120. Fundo de Defesa da Citricultura - FUNDECITRUS, Brazil
121. National Center for Eliminating Viruses from Deciduous Fruit Tree, Research Institute of Pomology, Chinese Academy of Agriculture Sciences, Liaoning, Xingcheng 125100, People's Republic of China
122. USDA-ARS, Parlier, CA, USA
123. Citrus Research Institute, China
124. Department of Plant Protection, Biotechnical Faculty, University of Montenegro, Montenegro

▼ These authors contributed equally to this work

*Corresponding authors: Vidalakis, G. (vidalg@ucr.edu, Citrus), Špak, J. (spak@umbr.cas.cz; currants and gooseberry), Constable, F. (Fiona.Constable@agriculture.vic.gov.au, grapevine), Harper, S.J. (scott.harper@wsu.edu, Pome fruits), Hammond, J. (john.hammond@usda.gov, Rose), Candresse, T. (thierry.candresse@inrae.fr, Stone fruits), Tzanetakis I.E. (itzaneta@uark.edu; Raspberry, blackberry, strawberry)

Plant virology was established as a discipline in the late 19th 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 amongst 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 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; Rott et al., 2017; Bester et al., 2021; Villamor et al., 2022). However, when symptoms develop in grafted indicator plants, it's often impossible to determine whether the indexed agent is one of the so-called phantoms, due 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's 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, 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 co-authors 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 over 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, USA. 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, USA (Frolich, 1958; Frolich and Hodgson, 1961). The growth of the sweet orange rootstock was greatly restricted, in comparison to 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 (van Vuuren and da Graca, 1996; Duran-Vila et al., 2002), 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 satsuma dwarf virus (SDV) infection in Satsuma mandarin trees in Turkey (Onelge and Çınar, 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. aurantiifolia* (Christm.) Swingle) and lemons (Ahlawat and Chenulu, 1985). The symptoms were atypically flexible and downwards-bent tree limbs (Ahlawat and Pant, 2003). The disorder was associated with the presence of a phytoplasma. However, while 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 (Fernandez-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 (Beris et al., 2021), respectively. There have been no further reports of Tarocco pit since the 1960s.

Currant and Gooseberry (*Ribes*)

Gooseberry mosaic: Reported from a single gooseberry 'Lady Delamare' plant, with a bright yellow mottle and vein yellowing in Poland (Basak and Maskiewicz, 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 (Posnette, 1952, Adams 1987), and was apparently latent in red currant cultivars. Attempts to transmit the virus to herbaceous species were not successful. Basak and Maskiewicz (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 (Blatný, 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°C 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. 1980, 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 USA. Graft transmission has been reported (Hewitt 1975) but the etiological agent and its economic importance are not known (Bovey et al. 1980, 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; Woodham and Krake 1983; Martelli 1993,

Martelli and Boudon-Padieu 2006). 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, USA, 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, Chiumenti et al. 2013, Martelli 1993, Martelli and Boudon-Padieu 2006). Some *Enamovirus* species are associated with enation in other crops. Recently two grapevine enamovirus 1 and grapevine enamovirus 2 were described (Silva et al 2017, Diaz-Lara 2023), 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* x *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 due 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 1950s 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 about four 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: A disease described from a single 'McIntosh' tree in New York, USA, 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 bunched 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, USA in the 1960s, and subsequently reported across North America, Poland, and China (Parish et al., 1982; 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, USA in 1960. The disease, a bark necrosis and scaling disorder, was reproduced by graft transmission on 'Winesap' four years after inoculation; Red 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 3-4 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 1950s and 1960s affecting 'Ballarat', 'McIntosh', 'Spartan', 'Stayman', and 'Winesap'. Symptoms included chlorotic vein flecking, leaf puckering and distortion, as well as reduced bloom and dimpled and distorted fruit (Nagaich and Vashisth, 1965; Wood, 1972b; Welsh and May, 1973). 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 1950s-1960s (Nagaich and Vashith, 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). While it was graft-transmissible to 'Yellow Newton', symptoms, which appeared two 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, USA, in 1962, which faded as the fruit matured and the normal red color developed. While this syndrome was graft transmissible and reproducible in 'Red Delicious' and 'Starkrimson' (Coyier et al., 1967; Zawadzka and Milligan, 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, 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 fifty 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 while healthy controls remained symptomless (van Katwijk, 1953). While 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 'M2' 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 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, 1965).

Pear concentric ring pattern: In the late 1960s in Maryland, USA, 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 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 (Parish and Raese, 1985; Hansen and Waterworth, 1989). Identified in both the USA Pacific Northwest and in Canada, slow field spread has been reported although no vectors were identified (Wilkes and Welsh, 1965; Parish and Raese 1985; Hansen and Waterworth 1989). 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, USA. In both cases the agent was not graft-transmissible, and no further reports have been made (Fleisher et al., 1973; Waterworth, 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, 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 19th 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 downwards 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 seed-borne 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 seed-borne virus:

Rubus Chinese seed-borne 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, the Eastern and Midwestern USA and was found infrequently in California, USA (Secor et al., 1977). The graft-transmissible disease was suspected to be caused by 'rose streak virus' though 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, USA. 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 downwards, 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 USA is therefore presumed to be distinct from that caused by rose leaf curl virus in Pakistan. Whether the disease seen in the USA has a biotic or an abiotic cause is still unclear, and the

description as 'rose leaf curl' is not useful due to 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 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 at 2-7 years later induced symptoms on *Physalis pubescens* L.; bud grafting from mahaleb cherry and peach to other woody indicators yielded no symptoms. While "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 either by 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 microphyllly 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 USA (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 Wagon, 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 (ABFV). However, in almonds affected by almond virus bud failure and in peaches affected by mule's ear disease, the presence of prunus necrotic ringspot virus (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, along with 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 (Verderevskaia, 1968), has been shown to be graft transmissible in apricot. There is no information on the causal agent nor 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 identified 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, USA 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 (Blattny, 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 (Navratil 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 USA Northeast in the 1940's (Milbrath and Kienholz, 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 apple chlorotic leaf spot virus (ACLSV) and it has been suggested that the disease might be caused by a mixed infection of ACLSV and PNRSV (Nemeth, 1986). Since 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, USA (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 suggesting 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.

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 & 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 also other agent(s) 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 in Bulgaria. Graft transmission was achieved to sweet cherry indicators. Samples in the USA 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 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 season are symptomless. Absence of characteristic symptoms of RNRSV, necrotic rusty mottle, and European rusty mottle tatter leaf and 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, USA in the late 1950's and early 1960's (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, while 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. So, 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, USA in 1962 (Cheney et al., 1967). As

of 1976, the natural occurrence had been limited to that single tree (Blodgett and Aichele, 1976), while 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 USA and Canada (Mircetich and Fogle, 1969; Mircetich et al., 1978). 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: Cherry white spot: White spot disease was described as a 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 USA 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 in two orchards in Michigan, USA in 1976 were 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 2010's 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, 1964) 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.

Peach line pattern and leaf curl: A virus was identified in Hungary and recorded in the early 1980's infecting peach and sweet cherry (Nemeth 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 anti-plum pox virus (PPV) polyclonal antisera (James et al., 1994). High-throughput sequencing has since showed this source to be co-infected 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. While it is unclear if 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 (Blodget, 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., 2001). 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, 1964). 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 produces 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 USA, 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, USA 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, nematode infections and cultivation factors (Kenknight, 1976; Howell et al., 2011).

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, 1974). 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 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 USA 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* (Blattny, 1961). No new reports identified in recent years in any country and 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, nor the original publication describing the disease neither any other report could not 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 USA 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, 1976).

Sour cherry line pattern: The disease was identified in the former Czechoslovakia (Paulechova, 1968). Little information is available since 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 in 1934 and shown to be associated with a graft-transmissible agent in 1940 (Reeves, 1943). Sweet cherry indicators are symptomless carriers while Italian prune, 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* EMC clones and not in others. Yoshikawa and Inouye (1988) 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 high throughput sequence 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, USA 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. x 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 (Maassen, 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 whereas. symptomatic plants were tested negative for phytoplasmal infection using fluorescence microscopy and direct and nested PCR assays and all . 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 ELISA 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 which 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 non-quarantine 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; IPPC, 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

1382 later shown to be synonymous with previously reported, yet unsequenced pathogens
1383 (EFSA, 2019; Hammond et al., 2021; Sabanadzovic et al., 2011). In both scenarios
1384 there may have been regulatory listing of pathogenic agents, leading to trade barriers.

1385 There have been concerted attempts to generate sequence data from historical virus
1386 isolate collections to link to these phantom agents (Table 1) and in certain cases Koch's
1387 postulates have been fulfilled by using infectious clones as shown for apple russet ring
1388 and apple green crinkle diseases caused by isolates of ACLSV and ASPV, respectively (Li
1389 et al., 2020).

1390 Contemporary samples with similar maladies have been investigated using HTS
1391 approaches. For example Rott et al. (2018) investigated the uncommon apple rubbery
1392 wood disease, and the associated flat limb disease. This investigation revealed two
1393 novel viruses, apple rubbery wood virus 1 and apple rubbery wood virus 2, in affected
1394 samples, often in association with other common viruses. Yet demonstrating causal
1395 associations by fulfilling Koch's postulates can be challenging because of confounding
1396 co-infections, without mentioning that epidemiological observations may not be possible
1397 for rare diseases (Evans, 1976; Fox, 2020; Fredericks and Relman, 1996). Given the
1398 discontinuity in the criteria between the pre- and post- sequencing diagnostic eras for
1399 agents listed here, there is a need to revise them for selecting a regulated pest. The
1400 revisions should consider both biological and molecular data as well as evidence of
1401 pathogenicity and impact, before a pest is added to the regulatory list. In any event,
1402 none of the phantom agents highlighted here would meet the current or revised
1403 criteria.

1404 Many phantom agents could be considered "historical curiosities". Indeed, phantom
1405 agents are often linked to specific varietal reactions, and in many cases, they have
1406 ceased to be observed for several decades either because the cultivars involved have
1407 fallen out of popularity, or possibly because the agent has been eliminated through
1408 improved sanitary practices and the expansion of clean plant programs and certification
1409 schemes (Fuchs et al., 2021; Gergerich et al., 2015; Martin et al., 2016). With the
1410 gradual adoption of HTS in support of certification (Maree et al., 2018) it is likely that
1411 these agents, if still present in varietal stocks, will be detected and re-characterized. 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 policy makers 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 (Levy et al., 2000; Dimou et al., 2002). In conclusion, this paper provides valuable information

1441 for evaluating current regulation, to improve compliance with future scientifically
1442 substantiated regulation and effective protection of fruit crops against harmful diseases.

1443 Furthermore if, despite the extensive research carried out in support of this
1444 review, reference material of one of these phantom agents is still available in any local
1445 repository, the authors would be grateful for any feedback to further investigate their
1446 existence or etiology. To prevent future problems of the emergence of possible new
1447 phantom agents, we also call for the public submission of material of newly described
1448 agents to appropriate collections or bioresource centers for further investigations, and
1449 for securing and establishing necessary reference material.

1450

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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2267 Table 1. Agents/Diseases caused by infectious agents previously misidentified,
 2268 synonymized or characterized and which 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	<i>'Candidatus Liberibacter asiaticus'</i>	Wallace, 1978
Citrus	Oleocellosis-like symptoms of Satsuma orange, Summer orange dwarf (a.k.a., Satsuma dwarf-like disease on citrus natsudaikai in Yamaguchi Prefecture), citrus mosaic, and Natsudaikai dwarf	satsuma dwarf virus	Tanaka & Yamada, 1961; Le Gall et al., 2005; Iwanami, 2010; Iwanami, 2023; Toru Iwanami (personal communication)
Citrus	Mechanically transmitted citrus ringspot virus	citrus psorosis virus	Garnsey, 1975; Garnsey & Timmer, 1980
Citrus	Bahia bark scaling	<i>Lasiodiplodia iraniensis</i> in grapefruit	Passos, 1965; Passos et al., 1974; Nickel et al., 2007; Moreno et al., 2015; Gama, 2019, Gama et al., 2019; 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 & Uyemoto 1976
Malus	Apple blister bark	apple fruit crinkle viroid	Koganezawa & 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., 2004; 2007b

Rubus	raspberry mottle virus, raspberry leaf spot virus, raspberry yellow spot virus	Raspberry leaf mottle virus	McGavin et al., 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	Cherry Amasya disease – cherry chlorotic rusty spot – Cherry leaf scorch	<i>Apiognomonina erythrostoma</i>	Minoia et al., 2014
Prunus	Cherry rosette virus	Nepovirus	GenBank Accession PP393046-7 (Blouin, unpublished)
Prunus	Plum leaf roll	Phytoplasmas (<i>Candidatus</i> Phytoplasma prunorum, Elm yellows group phytoplasmas)	Marccone et al., 2002; Hong et al., 2011
Prunus	Apricot fruit blotch; Apricot Butterature; Apricot viruela	apple chlorotic leaf spot virus	Furia and Ragozzino, 1974; Desvignes et al., 1990; Pena-Iglesias, 1988, Cañizares et al., 2011
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	Villamor & Eastwell, 2013; James et al., 2014
Prunus	Cherry mottle leaf	cherry mottle leaf virus	James et al., 2000
Prunus	Cherry rusty mottle	cherry rusty mottle-associated virus	Villamor & Eastwell, 2013
Prunus	Cherry necrotic rusty mottle	cherry necrotic rusty mottle virus	Rott & Jelkmann, 2011
Prunus	Shirofugen stunt, Kwanzan stunt	little cherry virus-1	Matic et al. 2009; Candresse et al., 2013
Pyrus	Pear corky pit, stony pit	apple stem pitting virus	Paunovic et al. 1999
Strawberry	Strawberry pallidosis	strawberry pallidosis associated virus, beet pseudo-yellows virus	Tzanetakis et al., 2003; 2004
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)