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*Original Citation:*

*Availability:*

This version is available <http://hdl.handle.net/2318/1695327> since 2019-03-23T08:21:50Z

*Published version:*

DOI:10.1007/s10658-018-1597-2

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[319 LIONE G., DANTI R., FERNANDEZ-CONRADI P., FERREIRA-CARDOSO J.V., LEFORT F., MARQUES G., MEYER J.B., PROSPERO S., RADÓCZ L., ROBIN C., TURCHETTI T., VETTRAINO A.M., GONTHIER P., 2019. The emerging pathogen of chestnut *Gnomoniopsis castaneae*: the challenge posed by a versatile fungus. *European Journal of Plant Pathology* 153, 671-685, DOI: 10.1007/s10658-018-1597-2]

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## The emerging pathogen of chestnut *Gnomoniopsis castaneae*: the challenge posed by a versatile fungus

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### Abstract

*Gnomoniopsis castaneae* is an emerging fungal pathogen currently scored as the major nut rot agent on chestnut, although it is also associated with cankers on both chestnut and hazelnut, as well as with necrosis on chestnut galls and leaves. Described for the first time in 2012, *G. castaneae* has been reported in several countries across Europe, Asia and Australasia, often in relation to severe outbreaks. The goal of this review is to provide a comprehensive summary of the state of the art about *G. castaneae*, highlighting the main results achieved by the research and stressing the most relevant knowledge gaps that still need to be filled.

This overview includes topics encompassing the taxonomy of the fungal pathogen, its host range and geographic distribution, the symptomatology and the diagnostic methods available for its detection, its impact, biology, ecology and epidemiology. The main interactions between *G. castaneae* and other organisms are also discussed, as well as the possible control strategies. In these past few years, relevant progresses in the knowledge of *G. castaneae* have been achieved, yet the complexity of the challenges that this pathogen poses to chestnut growers and to the scientific community advocates for further advances.

**Keywords:** canker, *Castanea* spp., *Dryocosmus kuriphilus*, *Gnomoniopsis smithogilvyi*, nut rot, review.

## 81 Introduction

82

83 The genus *Castanea* (hereafter referred to as chestnut) includes 13 woody species widely distributed across both  
84 hemispheres, as a result of their natural dispersal and cultivation by humans (Mellano et al. 2012). Despite being a  
85 multipurpose tree, chestnut has been cultivated and spread in association with the provision of specific goods such as edible  
86 nuts, timber and firewood (Conedera et al. 2004; Bounous and Torello Marinoni 2005; Mellano et al. 2012). To date, most  
87 of the economic relevance of chestnut relies on the production of marketable nuts for human consumption, mainly deriving  
88 from the cultivation of *C. sativa* Mill. (European or sweet chestnut), *C. crenata* Sieb. et Zucc. (Japanese chestnut), *C.*  
89 *mollissima* Blume (Chinese chestnut), and of their hybrids (Conedera et al. 2004; Bounous and Torello Marinoni 2005;  
90 Mellano et al. 2012).

91 The production of edible fruits may be compromised to variable extents as a consequence of abiotic stresses,  
92 pathogens and pests, whose presence can reduce fruit yield and quality in pre-harvest or post-harvest conditions. Some of  
93 the most damaging threats of chestnut affect tree health by significantly reducing its vitality and by determining substantial  
94 decline, not rarely leading to death. This is the case, for instance, of the onset of ink disease caused by the oomycetes  
95 *Phytophthora cambivora* (Petri) Buisman and *P. cinnamomi* Rands, of the chestnut blight epidemic due to the ascomycete  
96 *Cryphonectria parasitica* (Murrill) M.E. Barr and of the infestation of the Asian gall wasp *Dryocosmus kuriphilus*  
97 Yasumatsu (Vettraino et al. 2005a; Sartor et al. 2015; Rigling and Prospero 2018). Damages to chestnut may be substantial  
98 or even catastrophic. For instance, *C. dentata* (Marsh) Borkh. (American chestnut) got almost extinct by chestnut blight in  
99 the early 20<sup>th</sup> century in North America, where it was once largely widespread (Russell 1987). Other pathogens may act  
100 directly at fruit level, including many fungi associated with the spoilage of nuts, such as *Acrospeira mirabilis* Berk. &  
101 Broome, *Alternaria* spp., *Aspergillus* spp., *Botrytis cinerea* Pers., *Ciboria batschiana* (Zopf) N.F., *Colletotrichum acutatum*  
102 J.H. Simmonds, *Coniophora puteana* (Schumach.) P. Karst., *Cryptodiaporthe castanea* (Tul. & C. Tul.) Wehm. Buchw.,  
103 *Cytodiplospora castanea* Oudem., *Discula campestris* (Pass.) Arx, *Dothiorella* spp., *Fusarium* spp., *Mucor* spp.,  
104 *Neofusicoccum ribis* (Slippers, Crous & M.J. Wingf.) Crous, Slippers & A.J.L. Phillips, *Penicillium* spp., *Pestalotia* spp.,  
105 *Phoma castanea* Peck, *Phomopsis endogena* (Speg.) Cif., *Phomopsis viterbensis* Camici, *Rhizopus* spp., *Sclerotinia*  
106 *sclerotiorum* (Lib.) de Bary, *Trichoderma* spp., *Trichothecium roseum* (Pers.) Link, and *Truncatella* spp. (Hrubik and  
107 Juhasova 1970; Washington et al. 1997; Overy et al. 2003; Panagou et al. 2005; Rodrigues et al. 2012; Visentin et al. 2012;  
108 Donis-González et al. 2016; Gaffuri et al. 2017).

109 Until the early 2000s, one of the fungal species most frequently associated with the spoilage of chestnut nuts was  
110 the black rot agent *C. batschiana*, a latent pathogen that could be isolated from asymptomatic nuts, buds and bark tissues,  
111 as well as from rotten fruits (Hrubik and Juhasova 1970; Vettraino et al. 2005b; Blaiotta et al. 2014). In addition, *Phoma*  
112 spp. and *Phomopsis* spp. were reported as locally relevant in association with the spoilage or mummification of chestnut  
113 nuts (Washington et al. 1997; Visentin et al. 2012; Maresi et al. 2013). Although nut rots can be occasionally detrimental  
114 and challenging for chestnut growers and industry (Shuttleworth et al. 2013), they have generally not been considered as  
115 major threats to the cultivation of chestnut worldwide. Moreover, nut rots mostly occur as a post-harvest issue related to the  
116 storage conditions and to insects' infestations, while the harvest methods do not seem to play a relevant role on their  
117 incidence (Washington et al. 1997; Sieber et al. 2007; Migliorini et al. 2010).

118 Since the mid-2000s, a steep raise in the incidence of rotten nuts has been extensively observed by chestnut growers  
119 in some regions of Europe and Australasia (Smith and Agri 2008; Smith and Ogilvy 2008; Gentile et al. 2009; Visentin et  
120 al. 2012). Spoiled kernels displayed symptoms not completely consistent with any common disease of chestnut fruits. In  
121 2012, the causal agent of these outbreaks was described as the novel fungal species *Gnomoniopsis castaneae* G. Tamietti  
122 (Visentin et al. 2012; Tamietti 2016). To date, *G. castaneae* is deemed the main nut rot agent of chestnut across vast  
123 geographic areas encompassing three continents (Visentin et al. 2012; Shuttleworth et al. 2012; Shuttleworth et al. 2013;  
124 Maresi et al. 2013; Dar and Rai 2015; Dennert et al. 2015; Lione et al. 2015; Shuttleworth and Guest 2017; Vannini et al.  
125 2017). Moreover, the same fungal species was also reported in association with the onset of chestnut bark cankers in Europe  
126 and Asia (Dar and Rai 2015; Pasche et al. 2016a). Hence, *G. castaneae* may be currently acknowledged as a serious  
127 emerging plant pathogen threatening the cultivation of chestnut and challenging researchers, policymakers and chestnut  
128 growers at a global scale. Under such a premise, the goal of this review is to provide a comprehensive overview of the state  
129 of the art about *G. castaneae*, while highlighting gaps, uncertainties and future perspectives.

130

## 131 Identity and taxonomy

132

133 Nut rots epidemics reported in Europe and Australasia since the mid-2000s were firstly attributed to *Gnomonia*  
134 *pascoe* species nova or to its anamorphic stage *Discula pascoe*, although both binomials were not formally and validly

135 assigned (Smith and Agri 2008; Smith and Ogilvy 2008; Gentile et al. 2009; Shuttleworth et al. 2015). The fungi responsible  
136 for the above epidemics were independently and validly described in 2012 as *Gnomoniopsis castaneae* (“*castanea*”) G.  
137 Tamietti *species nova* (Visentin et al. 2012) and *G. smithogilvyi* L.A. Shuttlew., E.C.Y. Liew & D.I. Guest *species nova*  
138 (Shuttleworth et al. 2012), in Europe and Australasia, respectively. Later, morphological observations, DNA sequencing  
139 and phylogenetic analyses demonstrated the synonymy between the two taxa (Shuttleworth et al. 2015), *G. castaneae* having  
140 priority over *G. smithogilvyi* (Tamietti 2016). The fungus is known in both the teleomorphic and anamorphic stages,  
141 producing ascomata (i.e. perithecia) and conidiomata (i.e. acervuli), respectively (Visentin et al. 2012).

142 Although clearly defined as a species, some ambiguities related to the taxonomy of *G. castaneae* still need to be  
143 elucidated. For instance, Meyer et al. (2015) and Ibrahim et al. (2017) listed *Amphiporthe castanea* (Tul. & C. Tul.) M.E.  
144 Barr as a synonym of *G. castaneae*. However, *Gnomoniopsis* and *Amphiporthe* are indicated as clearly distinct within the  
145 *Gnomoniaceae* according to the list of accepted genera of *Diaporthales* (Senanayake et al. 2017). Preliminary observations  
146 suggest that isolates of *A. castanea* display both morphological traits and sequences of the internal transcribed spacers (ITS)  
147 of ribosomal DNA identical to those of *G. castaneae*, although the possible synonymy could be unraveled only through  
148 more detailed analyses conducted by sequencing and comparing conserved DNA loci between the holotypes of the two  
149 species (T. Sieber, ETH Zürich, Switzerland, pers. comm.). Furthermore, the possibility that *P. endogena* and *G. castaneae*  
150 could be the same species was deemed likely based on a comprehensive analysis of the literature dealing with chestnut nut  
151 rots and on the examination of some common morphological and symptoms-related features (Maresi et al. 2013). If such  
152 speculations were proven, the emergence of the nut rots caused by *G. castaneae* might predate the 2000s and the known  
153 geographic distribution of the pathogen might be broader. However, further studies are required to confirm or reject the  
154 above hypotheses.  
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## 156 **Host range and geographic distribution**

157  
158 *Gnomoniopsis castaneae* has been reported on different tree and shrub species within the families Betulaceae,  
159 Fagaceae, Oleaceae, and Pinaceae including both cultivated and wild plants such as chestnut (*C. sativa*, *C. crenata* and  
160 hybrids between the two species), hazelnut (*Corylus avellana* L.), manna ash (*Fraxinus ornus* L.), holm oak (*Quercus ilex*  
161 L.), Turkey oak (*Quercus cerris* L.), and maritime pine (*Pinus pinaster* Aiton) (Table 1). It should be noted, however, that  
162 the fungus has been also reported as a saprobe or endophyte in addition to as a pathogen, depending on the host and plant  
163 tissue (Table 1). For instance, fungal endophyte communities inhabiting asymptomatic leaves of different tree species were  
164 investigated in southern Italy by analyzing Illumina-MiSeq generated fungal ITS1 sequences. The Operational Taxonomic  
165 Unit (OTU) assigned to *G. castaneae*, with the online BLAST web interface against the GenBank database, was detected  
166 in leaves of chestnut, Turkey oak, manna ash, and maritime pine (Fernandez-Conradi 2017; Fernandez-Conradi et al. 2017;  
167 Fernandez-Conradi unpublished). This result was consistent with the record of Ibrahim et al. (2017) reporting *G. castaneae*  
168 among the manna ash foliar endophytes.

169 The current geographic distribution of *G. castaneae* encompasses 12 countries scattered across three continents,  
170 including Europe, Asia and Australasia (Table 1). However, only some of the regions where the potential hosts of *G.*  
171 *castaneae* are widely distributed have been thoroughly surveyed. Despite different interpretations having been proposed to  
172 explain the current distribution and the possible intra- and inter-continental spread of *G. castaneae* (Pasche et al. 2016a;  
173 Seddaiu et al. 2017; Sillo et al. 2017), the origin of the fungus is still unknown.  
174

## 175 **Symptomatology and diagnosis**

176  
177 *G. castaneae* has been reported to cause symptoms including nut rot on chestnut, bark cankers on chestnut and  
178 hazelnut, and necrosis on chestnut leaves and galls. The association between the fungus and the symptoms on the different  
179 hosts has been repeatedly confirmed through the fulfillment of Koch’s postulates.

180 The nut rot of chestnut caused by *G. castaneae* displays the typical color alteration and texture degradation  
181 characterizing brown rots, although in some cases the kernel may appear as chalky and dehydrated (Visentin et al. 2012;  
182 Maresi et al. 2013; Shuttleworth et al. 2013). Iconographic tables showing the main symptoms on nuts are available (Smith  
183 and Agri 2008; Gentile et al. 2009; Shuttleworth et al. 2012; Visentin et al. 2012; Maresi et al. 2013; Shuttleworth and Guest  
184 2017). However, nut rot symptoms are visible only once the fruit has been excised and the kernel exposed. In addition,  
185 depending on the progression of the disease, the confusion with diseases caused by other fungal pathogens such as *P.*  
186 *endogena* or molds cannot be ruled out. Further complexity is added by the fact that *G. castaneae* can also live as an  
187 endophyte within asymptomatic nuts, hence hampering the visual detection of the disease (Dennert et al. 2015; Ruocco et

188 al. 2016). For instance, Dennert et al. (2015) reported a substantial underestimation of the incidence of *G. castaneae* (about  
189 30%) when the diagnosis was based on the mere visual inspection rather than on isolation.

190 Bark cankers caused by *G. castaneae* on young chestnut branches and scions are morphologically similar to those  
191 caused by the chestnut blight pathogen *C. parasitica*, hence the impact of *G. castaneae* as a canker agent may be difficult  
192 to appraise in the field (Pasche et al. 2016a). Not surprisingly, in most cases the presence of *G. castaneae* in association  
193 with cankers emerged almost accidentally during regular surveys targeting *C. parasitica* (Dar and Rai 2015; Pasche et al.  
194 2016a; Lewis et al. 2017; Trapiello et al. 2017). Nonetheless, a careful examination focused on the color and morphology  
195 of conidiomata, stromata and tendrils might provide clues to detect *G. castaneae* (Pasche et al. 2016a). It is still unknown  
196 if *G. castaneae* might trigger the onset of cankers as severe as those caused by *C. parasitica* on elder branches and trunks  
197 of chestnut in field conditions. However, preliminary results from inoculation trials conducted on 2-year-old chestnut plants  
198 showed that isolates of *G. castaneae* were threefold less aggressive than a virulent *C. parasitica* isolate (C. Robin,  
199 unpublished). *G. castaneae* was also observed in association with cankers on hazelnut, although in this case the fungus was  
200 described as a weak pathogen (Linaldeddu et al. 2016). In fact, pathogenicity tests pointed out that *G. castaneae* could  
201 qualitatively reproduce cankers on hazelnut, but their severity did not attain values significantly higher than those displayed  
202 by untreated controls (Linaldeddu et al. 2016).

203 A series of reports have shown the causal relation between *G. castaneae* colonization and the appearance of  
204 necrosis on chestnut leaves and galls, the latter induced by *D. kuriphilus*, an alien pest to Europe (Magro et al. 2010; Vinale  
205 et al. 2014; Seddaiu, et al. 2017; Vannini et al. 2017). Recent findings pointed out that some secondary metabolites produced  
206 by strains of *G. castaneae*, namely the abscisic acid (ABA) and the 1',4'-*trans*-diol ABA, display phytotoxic effects on  
207 chestnut leaves and could be involved in galls necrosis (Vinale et al. 2014). However, the onset of necrosis on *D. kuriphilus*  
208 galls are also associated with other fungi, including *Fusarium incarnatum-equiseti* species complex (FIESC), *Alternaria*  
209 *alternata* (Fr.) Keissl., and *Botrytis* sp. (Addario and Turchetti 2011).

210 Regardless of the disease type, the most reliable diagnostic methods for *G. castaneae* rely on field samplings,  
211 followed by isolation on substrates such as MEA (Malt Extract Agar), MYA (Malt Yeast Agar) and PDA (Potato Dextrose  
212 Agar), and subsequent identification of isolates through morphometric and/or biomolecular assays (Shuttleworth et al. 2012;  
213 Visentin et al. 2012). Macro- and micromorphology of perithecia and ascospores or acervuli and conidia have been  
214 extensively described (Shuttleworth et al. 2012; Visentin et al. 2012). Some observations can be performed directly *in*  
215 *planta*, possibly after incubation of infected host tissues in a damp chamber (Vannini et al. 2017), while others need to be  
216 conducted *in vitro*. Nonetheless, the correct identification of *G. castaneae* might not be successfully accomplished through  
217 the mere morphological characterization of the fungal isolates, since colonies of other fungi inhabiting the same hosts can  
218 display similar morphological traits, as remarked by Meyer et al. (2017) for isolates of *Sirococcus castaneae* comb. nov.  
219 J.B. Meyer & B. Senn-Irlet & T.N. Sieber (syn. *Diplodina castaneae* Prill. & Delacr.), just to cite an example. A taxon-  
220 specific molecular assay was designed, tested and validated for the identification of *G. castaneae* through a Polymerase  
221 Chain Reaction (PCR) based on a set of specific primers (Lione et al. 2015). Alternatively, the identification of the fungus  
222 may be achieved by a multilocus phylogenetic analysis of the internal transcribed spacers (ITS) of ribosomal DNA, the  
223 translation elongation factor 1-alpha (TEF1- $\alpha$ ) and the  $\beta$ -tubulin genes (Visentin et al. 2012; Linaldeddu et al. 2016; Pasche  
224 et al. 2016a).

## 225 226 **Impact**

227  
228 Nut rot caused by *G. castaneae* may occur both in pre-harvest and in post-harvest conditions, affecting nuts still  
229 on the tree, laying on the ground or stored prior to be marketed or processed. The incidence of *G. castaneae* on nuts has  
230 been reported to vary in space and time, but it is often associated with substantial yield losses. For instance, peaks of  
231 incidence between 71.4% and 93.5% have been reported in chestnut orchards in north western Italy (Visentin et al. 2012;  
232 Lione et al. 2015; Lione and Gonthier 2016), a peak of 49% was reported in north eastern Italy (Maresi et al. 2013), and  
233 levels as high as 72% and 91% were observed in Australasia and Switzerland, respectively (Shuttleworth et al. 2013;  
234 Dennert et al. 2015). Not surprisingly, *G. castaneae* is currently acknowledged as a major threat affecting chestnut nuts  
235 (Shuttleworth et al. 2013; Dennert et al. 2015). The incidence of cankers caused by *G. castaneae* may be locally relevant as  
236 well. As an example, Dar and Rai (2015) reported an average incidence of *G. castaneae* attaining 39% in symptomatic  
237 branches. While data about the frequency of the pathogen and the severity of symptoms on leaves are scanty, more  
238 throughout investigations have been carried out on galls induced by *D. kuriphilus*. Here, incidences of *G. castaneae* as high  
239 as 53.8%, 68%, and over 80% were recorded in Switzerland, Sardinia and central Italy, respectively (Meyer et al. 2015;  
240 Seddaiu, et al. 2017; Vannini et al. 2017).

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## Biology

*G. castaneae* is an ascomycete whose mycelium can colonize different host tissues (Table 1). The fungus has been identified as a minor component of the endophytic community of manna ash (Ibrahim, et al. 2017), while it has been extensively reported as the main, or among the major endophytes of chestnut (Visentin et al. 2012), with isolation frequencies varying depending on the tissue, year and geographic location but as high as 70% in Europe and 80% in Australasia (Maresi et al. 2013; Pasche et al. 2016a; Shuttleworth and Guest 2017). The fungus has the ability to move from cell to cell within parenchymatic tissues, medullar rays and the vascular network (Pasche et al. 2016a). Both the teleomorphic and anamorphic stages of *G. castaneae* have been observed and described in chestnut (e.g. Shuttleworth et al. 2012; Visentin et al. 2012; Pasche et al. 2016a). Although ascospores can develop both on rotten nuts and burrs (Visentin et al. 2012), the latter may represent the main substrate for perithecia formation and subsequent release of infectious ascospores (Shuttleworth and Guest 2017). While ascospores can be produced all the day long, their release shows peaks approximately at sunrise and sunset (Shuttleworth and Guest 2017). In the field, the anamorphic stage of *G. castaneae* has been observed on the galls of *D. kuriphilus* (Maresi et al., 2013) and on bark cankers (Pasche et al. 2016a), while on nuts conidiomata have been detected only after incubation into damp chambers (Vannini et al. 2017). Hence, it was suggested that the anamorphic stage of the fungus could be rather frequent in the field too, provided that long-lasting conditions of high relative humidity are met (Vannini et al. 2017). However, based on the outcomes of a population genetics study conducted in Europe, the high genetic differentiation within populations along with the absence of significant linkage disequilibrium pointed to a prevailing role of sexual reproduction in *G. castaneae* (Sillo et al. 2017). Hence, in the long term, *G. castaneae* could be a high-risk pathogen at global level since it is likely to be endowed with a remarkable evolutionary potential fostered by the prevailing sexual reproduction (McDonald and Linde 2002; Sillo et al. 2017). Clonal spread through dissemination of conidia may also be relevant at the local scale, especially in association with site-specific factors (Sillo et al. 2017). For instance, conidiomata of *G. castaneae* developing on galls of *D. kuriphilus* might release conidial loads promoting the clonal spread of the fungus (Maresi et al. 2013; Vannini et al. 2017). Interestingly, conidiomata have not been extensively observed in Australia (Shuttleworth and Guest 2017), where *D. kuriphilus* is still absent (Csóka et al. 2017). Experimental evidence showed that conidia infect flowers at blossoming time and the same is likely for ascospores (Visentin et al. 2012; Shuttleworth and Guest 2017).

Based on the outcomes of isolation trials and spore trapping assays, an attempt of description of the infection process of *G. castaneae* on chestnut nuts was published (Shuttleworth and Guest 2017). Depending on the inoculum pressure and chestnut flowering time, ascospores released from perithecia harbored on burrs should be responsible of primary infections, while conidial loads should determine secondary infections on flowers, leaves and branches (Shuttleworth and Guest 2017). Wind, insects and rain should play a key role as carriers of infectious airborne inoculum, i.e. both ascospores and conidia (EPPO 2017; Shuttleworth and Guest 2017). Although intriguing and consistent with some previous speculations (Smith and Agri 2008; Smith and Ogilvy 2008; Gentile et al. 2009; Shuttleworth et al. 2013), as well as with experimental results showing the likelihood of conidial infections through the floral pathway (Visentin et al. 2012), this model of infection and disease spread would probably need further confirmations. For instance, to date, neither observational nor experimental evidence support the possibility that insects or other arthropods could act as vectors of *G. castaneae*. Although this eventuality cannot be ruled out, extensive isolation trials from *D. kuriphilus*, which is recognized as a major pest of chestnut, failed to detect viable inoculum of *G. castaneae* on adults, even when these insects emerged from galls colonized by the fungus (Lione et al. 2016). Vehiculation by pollen has also been hypothesized, although *ad hoc* experiments are still lacking (Shuttleworth and Guest 2017). Nonetheless, when appraising the risk associated with *G. castaneae* at global or local scale (EPPO 2017), the precautionary principle suggests to account for potential biotic interactions until they are not ruled out by dedicated studies. There is no information on the pathways of infection leading to cankers and to leaves and gall necrosis, although in this last case it was suggested that necrosis may occur on galls following endophytic colonization rather than from an external source of inoculum (Vannini et al. 2017). In addition, while the fungus has been often defined as a latent pathogen, the mechanisms underlying the hypothesized switch from the endophytic to the pathogenic phase are still largely unknown (Maresi et al. 2013; Lione et al. 2016; Pasche et al. 2016a,b; Shuttleworth and Guest 2017; Vannini et al. 2017).

The first evidence of intraspecific genetic differentiation within *G. castaneae* was detected by Dennert et al. (2015) in Switzerland. Based on the analysis of concatenated  $\beta$ -tubulin and calmodulin sequences, several haplotypes could be identified coexisting in the same trees at each sampling site (Dennert et al. 2015). This was also observed by Pasche et al. (2016a). A population genetics study conducted across a wider geographic area including southern Switzerland, north-western Italy and south-eastern France showed that two distinct subpopulations of *G. castaneae* could be identified combining simple sequence repeat (SSR) with high resolution melting (HRM) analyses (Sillo et al. 2017). Based on data of

297 allelic diversity, it was speculated that either both subpopulations, or at least one, could have been introduced to Europe  
298 (Sillo et al. 2017). In such a scenario and in agreement with the hypothesis proposed by Pasche et al. (2016a), north-western  
299 Italy could have represented the area of first introduction (Sillo et al. 2017).

300

## 301 **Ecology and epidemiology**

302

303 The influence of abiotic factors on the epidemics of nut rot of chestnut caused by *G. castaneae* has been partially  
304 investigated, with emphasis on climatic variables. By combining isolation trials and molecular diagnostic assays with  
305 statistical and geostatistical approaches, Lione et al. (2015) suggested that the incidence of *G. castaneae* at orchard level  
306 could be related to site-dependent factors exerting their influence at a scale of few kilometres (approximately 7.5-15.5).  
307 Further analyses revealed that the average mean, maximum and minimum temperatures of the months preceding nut  
308 harvesting (from January to October) were significantly correlated to the nut rot incidence at harvesting in north-western  
309 Italy (Lione et al. 2015). Based on different combinations of such temperatures, a series of predictive models (*GnoMods*)  
310 assessing the incidence of *G. castaneae* at site level was fitted and validated (Lione et al. 2015). *In silico* simulations carried  
311 out with *GnoMods* suggested that an overall increase of the average temperatures would likely trigger a raise of the nut rot  
312 incidence (Lione et al. 2015). The role of temperature as a key driver boosting disease incidence is in agreement with the  
313 findings reported by Maresi et al. (2013) and Vannini et al. (2017). The former suggested that warm temperatures and  
314 drought might be related to an exacerbation of nut rot in sites infested by *G. castaneae* in northern Italy. The latter showed  
315 that, in central Italy, the frequency of galls necrosis associated with *G. castaneae* increased exponentially, with a steep raise  
316 in the early summer to July, which was the warmest month reported during the timeframe of the study.

317 Field observations led to hypothesize that rainfall could trigger the incidence of the nut rot by raising the airborne  
318 inoculum of *G. castaneae* at blossoming time, hence fostering floral infection by ascospores (Smith and Agri 2008; Smith  
319 and Ogilvy 2008; Gentile et al. 2009). In Australia, isolation trials from chestnut flowers pointed out that a higher frequency  
320 of isolation of *G. castaneae* corresponded to a subsequent higher incidence of nut rot (Shuttleworth and Guest 2017). This  
321 finding confirmed previous results (Shuttleworth et al. 2013), showing through the fitting of a linear model that rainfall  
322 during chestnut blossoming in December was significantly associated with the incidence of nut rot, despite the correlation  
323 between the two variables being mild. Maresi et al. (2013) suggested that also drought might foster the incidence of nut rot.  
324 Nonetheless, investigations focused on other ecological factors might help in clarifying the drivers of *G. castaneae*  
325 outbreaks (Shuttleworth et al. 2013; Lione et al. 2015).

326 A study conducted in Italy with the aid of the newly developed Mean Distance Tests (MDT) showed that different  
327 chestnut patches displayed the same randomized spatial pattern of infection by *G. castaneae* regardless of their plantation  
328 density, suggesting that long-distance transmission of *G. castaneae* could be more likely than short-distance transmission  
329 (Lione and Gonthier 2016), which is also supported by the spatial distribution of the disease observed by Vannini et al.  
330 (2017). In addition, the hypothesis of a large-scale spread is consistent with findings showing that the same haplotype of *G.*  
331 *castaneae* can be present in chestnut stands separated by distances of many kilometers (Dennert et al. 2015; Sillo et al.  
332 2017).

333 High temperatures and relative humidity have been suggested to boost synergistically the development of bark  
334 cankers (Pasche et al. 2016a), whereas the occurrence of galls necrosis might be mainly influenced by temperatures, since  
335 the same exponential development of the symptoms was observed notwithstanding the different rainfall patterns (Vannini  
336 et al. 2017).

337 The epidemiology of *G. castaneae* could be even more complex than hypothesized so far because of its status of  
338 latent or weak pathogen and endophyte on different hosts, some of which share common habitats and an overlapping  
339 geographic distribution with chestnut (Linaldeddu et al. 2016). The possibility that such hosts may act as transmissive hosts  
340 has been suggested. For instance, the presence of hazelnut may have favored the establishment of *G. castaneae* on chestnut  
341 in Sardinia, despite the reverse process being equally likely (Seddaiu et al. 2017). Detecting the presence of transmissive  
342 hosts and unraveling their epidemiological role might be pivotal to clarify and predict the spread of the pathogen (Garbelotto  
343 et al. 2017). It is worth noting that ecology, infection processes and epidemiology of *G. castaneae* are likely to be variable  
344 within and among different biogeographical frames (Lione et al. 2015) depending on hosts presence and distribution,  
345 climate, effects of biotic interactions and availability of natural substrates for endophytic/saprobic/pathogenic colonization  
346 and for the development of the teleomorphic and anamorphic stages. Anthropogenic activities could also favor the spread of *G.*  
347 *castaneae* at the local or global scale through the movement of plants for planting/grafting and plant commodities (Pasche  
348 et al. 2016a; EPPO 2017), although these pathways deserve to be extensively investigated.

349

350

351



## 352 **Biotic interactions**

353

354 Interspecific interactions may drive the dynamics of plant diseases by influencing the outcomes of epidemics,  
355 especially when native hosts and plant microbiomes are challenged with alien or emerging threats, including insect pests  
356 and plant pathogenic fungi (Quacchia et al. 2008; Sillo et al. 2015; Garbelotto et al. 2017; Zampieri et al. 2017). The spatial  
357 and temporal overlapping between the outbreak of *G. castaneae* and the invasion by the alien pest *D. kuriphilus* in Europe  
358 (Brussino et al. 2002; Visentin et al. 2012) has triggered the research on the possible interactions between the two species.  
359 While it can be excluded that *D. kuriphilus* may act as a vector of viable inoculum of *G. castaneae* (Lione et al. 2016), a  
360 series of experiments revealed that *G. castaneae* can colonize chestnut buds asymptotically before the pest oviposition,  
361 and independently from this latter (Lione et al. 2016), although the colonization process still need to be further investigated.  
362 Nonetheless, the incubation under controlled conditions of chestnut galls collected in the field showed that the number of  
363 emerging adults of *D. kuriphilus* was significantly higher in galls colonized by *G. castaneae* than in those not colonized,  
364 suggesting a possible synergy between the pathogen and the pest (Lione et al. 2016). Such synergistic interaction is in  
365 agreement with the observation that the sites more severely infested by *D. kuriphilus* tend to display higher levels of nut rot  
366 incidence caused by *G. castaneae*, probably in relation to an increased availability to the fungus of a natural substrate (i.e.  
367 galls) for the production of ascomata and conidia (Maresi et al. 2013; Vannini et al. 2017). Interestingly, studies conducted  
368 on the endophytic communities in green galls induced by *D. kuriphillus* and in the associated surrounding leaf tissue pointed  
369 out that OTU richness and diversity were lower in galls, with a significantly different composition between chestnut galls  
370 and surrounding leaf tissues. Remarkably, the *G. castaneae* OTU was found in all sampled galls (84 samples, with a mean  
371 relative abundance equal to 0.73) and in 84% of the associated leaf samples (mean abundance 0.54). Results from this study  
372 suggest that *D. kuriphilus* act as an ecological filter selecting particular endophytic species, as *G. castaneae*, from a pool of  
373 species initially present in plant buds or galls (Fernandez-Conradi 2017; Fernandez-Conradi et al. 2017; Fernandez-Conradi  
374 unpublished).

375 Some studies documented the co-occurrence between the onset of galls necrosis and mortality of *D. kuriphilus*  
376 individuals inhabiting galls (Magro et al. 2010; Vannini et al. 2017), hence suggesting antagonisms in a broad sense between  
377 the fungus and the pest. The adverse effect exerted by *G. castaneae* against *D. kuriphilus* was not ascribed to a direct  
378 entomopathogenic activity of the fungus, but rather to an increased compactness and toughness of necrotic galls through  
379 dehydration preventing the emergence of the adults which remain trapped inside (Vannini et al. 2017). However, no  
380 detrimental effects of galls necrosis on the vitality and emergence of *D. kuriphilus* resulted from the experimental trials  
381 carried out by Seddaiu et al. (2017). Noteworthy, in addition to *G. castaneae*, several other fungal species have been isolated  
382 from necrotic galls, some potentially playing a role in the frame of this complex interspecific interaction (Vannini et al.  
383 2017). Moreover, Vannini et al. (2017) reported that the frequency of *G. castaneae* did not display significant and/or  
384 substantial differences between asymptomatic and symptomatic galls, thus adding further complexity to the interpretation  
385 of the interspecific interaction between the fungus and the pest. The previously documented mechanisms of synergy or  
386 antagonism between the fungus and the insect pest (Lione et al. 2016; Seddaiu et al. 2017; Vannini et al. 2017) would need  
387 further experimental support.

388 While testing the interaction between the chestnut blight pathogen *C. parasitica* and *D. kuriphilus* in Switzerland,  
389 the fungal community of galls abandoned by the pest was investigated, revealing that *G. castaneae* was prevalent (Meyer  
390 et al. 2015). In addition to *G. castaneae*, a second, much rarer species firstly attributed to the genus *Gnomoniopsis* (Meyer  
391 et al. 2015), but later referred to as *S. castaneae* (Meyer et al. 2017), was isolated. Interestingly, the abundance of both *G.*  
392 *castaneae* and *S. castaneae* taken together was negatively and significantly correlated to the abundance of *C. parasitica* in  
393 abandoned galls (Meyer et al. 2015). The above findings suggest that *G. castaneae* might have a competitive advantage  
394 over *C. parasitica* as endophytic colonizer of galls, hence potentially limiting the amount of infectious inoculum that could  
395 be produced by the chestnut blight pathogen on that substrate (Meyer et al. 2015). On the other side, a lower abundance of  
396 *G. castaneae* was found on older galls, suggesting that fungi with better saprotrophic ability, including *C. parasitica*, might  
397 outcompete it. In any case, the use of *G. castaneae* as a biocontrol agent against other pathogens or pests of chestnut is  
398 unfeasible and not recommended due to its pathogenic side effects on the same host (Vannini et al. 2017).  
399

## 400 **Control strategies**

401

402 Studies focused on testing if the management practices could influence the incidence of spoiling fungi are notably  
403 few for chestnut (Sieber et al. 2007). Screening and testing host varieties or cultivars either resistant, or at least more tolerant  
404 to *G. castaneae* might help in preventing the disease in new plantations. In this perspective, a first attempt was carried out  
405 in Australia with some among the most important chestnut varieties cultivated in that region for nuts production

406 (Shuttleworth et al. 2013; Shuttleworth and Guest 2017). Despite being all susceptible to *G. castaneae*, differences in the  
407 severity of symptoms were detected depending on the biogeographical origin of the fungal strains used for the pathogenicity  
408 tests (Shuttleworth and Guest 2017). In Europe, preliminary results from a survey conducted within a varietal collection  
409 field suggested that the susceptibility profiles to nut rot caused by *G. castaneae* are comparable between the *C. sativa*  
410 wildtype and some chestnut cultivars of local or global relevance (Lione 2016). However, further analyses are needed before  
411 drawing definitive conclusions.

412 The lack of association between the plantation density and the spatial pattern of nut rot caused by *G. castaneae*  
413 suggests that the attempt of controlling this pathogen by fine-tuning the orchard plantation density is likely to fail (Lione  
414 and Gonthier 2016). Conversely, considering the prevalence of sexual reproduction in *G. castaneae* (Sillo et al. 2017), an  
415 effective strategy could be represented by the removal of the fallen burrs on which the teleomorph stage develops (Visentin  
416 et al. 2012; Shuttleworth et al. 2013; Shuttleworth and Guest 2017; Sillo et al. 2017). However, this and other similar  
417 practices proposed in the literature (Shuttleworth et al. 2013) to prevent ascospores release might not lead to the expected  
418 outcomes because of the potential long-distance dispersal of the pathogen and of the local relevance of asexual reproduction  
419 (Sillo et al. 2017). Nonetheless, specific trials are needed to test which management options could be effective to control *G.*  
420 *castaneae* in the field.

421 Nut rot incidence may considerably increase during the post-harvest storage (Maresi et al. 2013; Shuttleworth et  
422 al. 2013; Dennert et al. 2015). The first attempt to test a post-harvest control strategy to reduce the incidence of the disease  
423 on chestnut nuts was reported in Ruocco et al. (2016). In this study, a traditional method based on the thermic treatment of  
424 nuts in water (i.e. “curatura”) was customized by adding to the water a cell-wall degrading enzyme mixture gathered from  
425 cultures of the fungus *Trichoderma harzianum* Rifai strain T22. The improved treatment resulted in a significant reduction  
426 of nut rot incidence, whose main agent had been previously detected as *G. castaneae* (Ruocco et al. 2016), hence providing  
427 new and intriguing perspectives to reduce the post-harvest losses caused by the pathogen.

428 The efficacy of biological control against *G. castaneae* was explored also in relation to its endophytic presence in  
429 grafting scions of chestnut (Pasche et al. 2016b). A series of observations led to the hypothesis that the bacterium *Bacillus*  
430 *amyloliquefaciens* (ex Fukumoto 1943) Priest et al. 1987 emend. Wang et al. 2008 and the fungus *Trichoderma atroviride*  
431 P. Karst. could act as antagonists against *G. castaneae* (Pasche et al. 2016b). By treating chestnut scions with inoculum  
432 suspensions of either *B. amyloliquefaciens* or *T. atroviride* prior to grafting, it was observed that *G. castaneae* was absent  
433 where such species colonized endophytically the woody tissues (Pasche et al. 2016b). Bark canker symptoms associated  
434 with *G. castaneae* were also slowed in their progression on treated plants (Pasche et al. 2016b). Consequently, the authors  
435 hypothesized that both *B. amyloliquefaciens* and *T. atroviride* could prevent or inhibit the development of *G. castaneae*,  
436 suggesting that preventive inoculations of these antagonistic endophytes could be effective in the biocontrol of the fungal  
437 pathogen (Pasche et al. 2016b).

438

## 439 **Conclusions and perspectives**

440

441 The current state of the art points out that *G. castaneae* is an emerging pathogen posing a major threat to chestnut  
442 cultivation worldwide. The nut rots and cankers associated with *G. castaneae* are likely to determine relevant losses in  
443 orchard and coppices challenging chestnut growers, foresters, researchers and policymakers. In spite of the remarkable  
444 progress achieved by the scientific research in the last years, there is a need to push the knowledge about *G. castaneae* far  
445 beyond its current status, especially with the aim of designing effective control strategies.

446 The endophytic presence of *G. castaneae* within asymptomatic plant tissues, as well as the difficulties in the  
447 diagnosis of the pathogen in symptomatic plants, might have led to a substantial underestimation of both its host range and  
448 geographic distribution. However, a full screening seeking for other potential host species might be difficult to implement  
449 on the large scale. On the contrary, extensive surveys targeting *G. castaneae* on its main confirmed hosts could be profitably  
450 carried out across regions where these species are abundant and play a key economic, social and environmental role. For  
451 instance, no records of *G. castaneae* are available for some countries accounting for the most relevant chestnut nuts  
452 production worldwide, including China, the Korean peninsula, Japan, Turkey and Portugal (Bounous and Torello Marinoni  
453 2005). Similarly, surveillance for *G. castaneae* might be important also in countries where chestnut has been recently  
454 introduced or reintroduced, such as USA, just to cite an example (Gold et al. 2006). In addition, investigations focused on  
455 hazelnut could unravel whether *G. castaneae* is a canker-related pathogen associated with mild symptoms on this host only  
456 at local level (i.e. Sardinia) (Linaldeddu et al. 2016), or if it could represent an emerging risk at the global scale.

457 The effectiveness of extensive surveys mostly depends on the availability of diagnostic techniques able to provide  
458 a reliable and reproducible outcome combining accuracy, versatility and technical/economical sustainability. As previously  
459 mentioned, only laboratory analyses and molecular-based approaches can satisfy the majority of the above requirements in  
460 the case of *G. castaneae*. Nonetheless, innovative diagnostic methods could be designed, customized and implemented for

461 rapid in-field applications. For instance, Loop-mediated isothermal AMPlification of DNA (LAMP) assays (Notomi et al.  
462 2000) might provide an intriguing perspective, as recently shown in studies focused on the diagnosis of emerging and  
463 invasive plant pathogens (Tomlinson et al. 2010; Sillo et al. 2018). LAMP-based tools might also help in preventing the  
464 circulation of plant commodities or other putative carriers of *G. castaneae* in non-infested areas, allowing for the timely  
465 detection of the pathogen even in the absence of symptoms and without the need of the fungal isolation step.

466 A phylogeographic investigation with the ultimate goal of clarifying the possible origin of the pathogen as well as  
467 its most likely transmission pathways would provide helpful insights. The intensive trade of plants for planting, wood, fruits  
468 and transformed products might foster the spread of the pathogen unless its carriers are identified and their epidemiological  
469 role elucidated. In spite of the considerable efforts devoted to investigate the biology, reproduction strategy, population  
470 structure, ecology and epidemiology of *G. castaneae*, relevant knowledge gaps still need to be filled. Such gaps include,  
471 but are not limited to, the detection of the possible vectors of the pathogen, the characterization of its spore deposition  
472 patterns at seasonal level, the identification of the mechanism allowing for its penetration within the different hosts tissues,  
473 the elucidation of the epidemiological role played by asymptomatic hosts/host tissues, the clarification of the factors  
474 triggering the switch from the endophytic to the pathogenic stage and their relation to the onset of nut rots, cankers and  
475 necrosis of green tissues. Moreover, the possibility that the level of pathogenicity of *G. castaneae* could be strain-dependent  
476 is worth of being fully explored.

477 The interpretation and prediction of disease outbreaks caused by *G. castaneae* could be substantially improved  
478 through the clarification of its interaction with other organisms potentially exerting a synergistic or antagonistic effect,  
479 possibly mediated by varying environmental conditions. While some biotic interactions with *D. kuriphilus* (Lione et al.  
480 2016; Seddaiu et al. 2017; Vannini et al. 2017) and *C. parasitica* (Meyer et al. 2017) have been investigated, no information  
481 is available about the possible interactions of the fungus with other arthropods or relevant chestnut pathogens affecting  
482 either nuts (e.g. *C. batschiana*), leaves [e.g. *Mycosphaerella maculiformis* (Pers.) J. Schröt], cambial or woody tissues (e.g.  
483 *Phytophthora* spp.). In addition, while in the case of *C. parasitica* the antagonism with *G. castaneae* is consistently  
484 supported by the available lines of evidence, at least at gall level (Meyer et al. 2015), for *D. kuriphilus* the results reported  
485 in the literature are partially discordant in defining possible synergistic or antagonistic interactions, hence requiring further  
486 investigations.

487 Another relevant aspect still largely unexplored is related to the susceptibility profiles of different chestnut cultivars  
488 to *G. castaneae*. A rank of differential susceptibilities supported by experimental trials and statistical evidence could provide  
489 the chestnut growers with helpful criteria to select the propagating material for new plantations. Under the same practical  
490 perspective, comparing the effects of different management practices on the incidence of *G. castaneae* might help in  
491 designing effective control strategies both in orchards and in coppices. In addition, control strategies could be profitably  
492 improved by testing both traditional methods, such as the application of fungicides, manures or other chemicals, and more  
493 sustainable approaches based on biological control, including the promising treatments with *B. amyloliquifaciens* and *T.*  
494 *atroviride* (Pasche et al. 2016b). In post-harvest, the use of bioproducts aimed at inhibiting pests and diseases has provided  
495 interesting results in controlling *G. castaneae* in chestnut nuts (Ruocco et al. 2016), thus offering new outlooks that are  
496 worth exploring to customize different nuts treatments based on hydrotherapy, thermotherapy, refrigeration in normal or  
497 controlled atmosphere, exposition to carbon dioxide (CO<sub>2</sub>) fluxes, freezing and drying (Bounous and Torello Marinoni  
498 2005). Finally, control treatments should also be tested in relation to potential mycotoxins contamination. In fact, despite  
499 the mycotoxigenic potential of *G. castaneae* is unknown, it cannot be excluded, as other mycotoxin-producing fungi have  
500 been isolated from chestnut nuts and derived products (Prencipe et al. 2018).

## 501 502 **Acknowledgements**

503  
504 This work was carried out within the activities of the Eurochestnut - Fungi Thematic Group. The Authors wish to thank  
505 Thomas Sieber of ETH Zürich (Switzerland), Anne Chandelier of the Walloon Agricultural Research Centre (Belgium),  
506 Patricia van Rijswick of the National Plant Protection Organization (the Netherlands) for proving personal communications  
507 and comments useful to this review. The Authors wish to thank the anonymous Reviewers for their useful comments and  
508 suggestions which contributed to improve the manuscript.

## 509 510 **Compliance with Ethical Standards**

511  
512 Funding: The work was co-funded by the F.E.A.S.R. 2014/2020, Project #castagnopiemonte, and by Regione Piemonte  
513 through the activity of the Chestnut Growing Centre.

514 Conflict of interest: the authors declare that they have no conflict of interest.

515 Ethical approval: this article does not contain any studies with human participants or animals performed by any of the  
516 authors.  
517 Informed consent: informed consent was not applicable to this article since no information regarding individual participants  
518 was included in the study.  
519

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**Table 1.** Host range, trophic attitude, symptomatology and geographic distribution of *G. castaneae*. Each row reports data from publications including the hosts on which *G. castaneae* was detected, the trophic attitude displayed by the fungus, the presence of disease symptoms and the country, region or state where the fungal species was found. Based on the available information, publications predating the first description of the species in 2012 are included when probably referring to *G. castaneae* or to its synonym *G. smithogilvyi* under a different or incomplete specific epithet. Rows are ranked based on the associated reference, using the chronological order per year and the alphabetical order within year. Acronyms next to the region/state indicate their associated country (AU - Australia, CH - Switzerland, FR - France, IT - Italy, NZ - New Zealand). If molecular analyses were conducted on strains already mentioned in, or clearly referable to other publications, the strains origin was omitted in the Country and Region/State columns.

Host	Trophic attitude	Symptoms	Country	Region/State	Reference
<i>C. sativa</i> <i>C. sativa</i> × <i>C. crenata</i>	pathogen	nut rot	Australia New Zealand	not specified	Smith and Agri (2008)
<i>Castanea</i> spp.	pathogen	nut rot	Australia	New South Wales	Smith and Ogilvyi (2008)
<i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on pistils and flowers, fruit stems, developing nuts, external burr tissues, and shoots bark	Italy	Piedmont	Gentile et al. (2009)
<i>Castanea</i> spp.	pathogen	necrosis on leaves and galls of <i>D. kuriphilus</i> , blight symptoms on twigs (artificial inoculation)	Italy	Lazio	Magro et al. (2010)
<i>Castanea</i> spp. <i>Q. ilex</i>	pathogen (on chestnut); saprobe (on chestnut); not specified (on holm oak)	nut rot; asymptomatic on dead burrs	Australia	New South Wales	Shuttleworth et al. (2012)
<i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on shoots bark and on flowers (artificial inoculation)	France Italy Switzerland	Alpes-de-Haute-Provence (FR) Piedmont (IT) Ticino (CH)	Visentin et al. (2012)
<i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on bark and young shoots	Italy	Piedmont Trentino-South Tyrol Tuscany	Maresi et al. (2013)
<i>C. sativa</i> <i>C. crenata</i> × <i>C. sativa</i>	pathogen	nut rot	Australia	New South Wales Victoria	Shuttleworth et al. (2013)
<i>Castanea</i> spp.	not specified	not specified on galls of <i>D. kuriphilus</i>	Italy	Campania	Vinale et al. (2014)
<i>C. sativa</i>	pathogen	canker on sprouts and branches	India	Jammu and Kashmir	Dar and Rai (2015)
<i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on ripened nuts	Switzerland	Glarus Graubünden Ticino	Dennert et al. (2015)
<i>C. sativa</i>	pathogen	nut rot	France Italy	Alpes-Maritimes (FR) Aosta Valley (IT) Piedmont (IT)	Lione et al. (2015)
<i>C. sativa</i>	not specified	not specified on abandoned necrotic galls of <i>D. kuriphilus</i>	Switzerland	Ticino Valais Vaud	Meyer et al. (2015)
<i>C. crenata</i> <i>C. crenata</i> × <i>C. sativa</i> <i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on nuts	Australia France New Zealand	Bay of Plenty (NZ) New South Wales (AU) Oise (F) Victoria (AU) Waikato (NZ)	Shuttleworth et al. (2015)
<i>C. avellana</i>	weak pathogen	canker on twigs and branches	Italy	Sardinia	Linaldeddu et al. (2016)
<i>C. sativa</i>	endophyte	asymptomatic in buds and galls of <i>D. kuriphilus</i>	Italy	Aosta Valley Piedmont	Lione et al. (2016)
<i>C. sativa</i>	pathogen	nut rot	Italy	Piedmont	Lione and Gonthier (2016)
<i>C. sativa</i>	pathogen; endophyte	canker on twigs and scions; asymptomatic on twigs and scions, in wood, bark and leaves, also at vascular level	Switzerland	Geneva Ticino	Pasche et al. (2016a,b)
<i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on ripened nuts; not specified on galls of <i>D. kuriphilus</i>	Italy	Campania	Ruocco et al. (2016)
<i>C. sativa</i> <i>C. crenata</i> × <i>C. sativa</i>	pathogen	nut rot; canker on branches	Slovenia	not specified	EPPO (2017)
<i>C. sativa</i> <i>F. ornus</i> <i>P. pinaster</i> <i>Q. cerris</i>	endophyte	asymptomatic on leaves	Italy	not specified	Fernandez-Conradi (2017); Fernandez-Conradi et al. (2017); Fernandez-Conradi, unpublished
<i>F. ornus</i>	endophyte	asymptomatic on leaves	Italy Switzerland	Ticino (CH) Trentino-South Tyrol (IT)	Ibrahim et al. (2017)

<i>C. sativa</i>	pathogen	canker on shoots	United Kingdom	not specified	Lewis et al. (2017)
<i>C. sativa</i>	not specified	isolated from canker	Switzerland	Valais Vaud	Meyer et al. (2017)
<i>C. sativa</i>	pathogen; endophyte	necrosis or asymptomatic on galls of <i>D. kuriphilus</i>	Italy	Sardinia	Seddaiu et al. (2017)
<i>C. sativa</i> <i>C. crenata</i> × <i>C. sativa</i>	pathogen; endophyte	nut rot; asymptomatic on female flowers, male flowers, styles, pedicels, burr equators, shell equators, kernels, terminal leaf petioles, terminal leaf mid-veins, terminal leaf margin, bark and vascular cambium of young branches, dormant terminal buds	Australia	New South Wales	Shuttleworth and Guest (2017)
<i>C. sativa</i>	pathogen	nut rot	France Italy Switzerland	Alpes-de-Haute-Provence (F) Aosta Valley (IT) Piedmont (IT) Ticino (CH)	Sillo et al. (2017)
<i>C. sativa</i> × <i>C. crenata</i>	pathogen	canker on branches	Spain	Asturias	Trapiello et al. (2017)
<i>C. sativa</i>	pathogen; endophyte	nut rot; necrosis on galls of <i>D. kuriphilus</i> ; asymptomatic on bark, buds, leaves, galls of <i>D. kuriphilus</i> and nuts	Italy	Lazio	Vannini et al. (2017); Vannini et al. (2018)
<i>C. sativa</i>	pathogen	bark canker	Belgium	not specified	Chandelier et al. (2018)
<i>C. sativa</i>	pathogen; endophyte	canker on branches and sprouts; asymptomatic on leaves	the Netherlands	not specified	P. van Rijswijk, National Plant Protection Organization, the Netherlands, pers. comm.
<i>C. sativa</i>	pathogen	nut rot	Czech Republic	not specified	P. Gonthier, University of Torino and L. Jankovský, Mendel University, Czech Republic, unpublished

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