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# The increasing importance of grapevine trunk diseases

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**Summary:** Grapevine trunk diseases (GTDs) are destroying the woody parts of the plants, resulting decline or dieback of the grapevine. More detailed research of the GTD began in 1950s, when Hewitt et al. (1957) observed that specific symptoms cannot be detected on the diseased trunks every year. Latest results have also proved that abiotic factors affect the appearance and the severity of the disease. Moreover several pathogenic fungi may play role as causative agents (Bertsch et al., 2013). *Eutypa*, *Botryosphaeria*, *Phomopsis* dieback, esca disease complex, and Petri disease are considered the major GTDs, where a variety of pathogens attack the woody perennial organs of the vine and ultimately lead to the death of the plant (Lehoczky, 1974; Laignon & Dubos, 1997; Rolshausen et al. 2010; Kotze et al., 2011; Bertsch et al., 2013; Fontaine et al., 2015).

The GTD incidence has been reported to be increased during the last decades (Úrbez-Torres et al., 2014). The esca incidence has reached 60% to 80% in some old vineyards in southern Italy (Pollastro et al., 2000; Surico et al., 2000; Calzarano & Di Marco, 2007). The disease incidence of the esca was reported to be increased from 1.83% to almost 13%, between 2003 and 2007 in Hungary (Dula, 2011). There was detected a five times increase in the GTD disease incidence in the Tokaj Wine Region, Hungary between 2014 and 2016 (Bihari et al., 2016).

**Keywords:** grapevine trunk disease, *Eutypa*, *Botryosphaeria* and- *Phomopsis* dieback, esca disease complex, Petri disease, black dead arm, black foot, grapevine

## Introduction

The Grapevine Trunk Diseases (GTD) are one of the most serious disease worldwide for the grapevine (*Vitis vinifera* L.), which are causing considerable yield loss and economic losses in the wine industry (Rego et al., 2000; Bertsch et al., 2009; Abreo et al., 2011; Diaz et al., 2012; Kaliternam & Milicevic, 2013; Mondello et al., 2013; Ammad et al., 2014). More and more researchers mention the appearance of the diseases severity similar to the phylloxera in Europe. The GTD has been described first in California in 1895. Ravaz identified the disease in France in 1898, as “folletage”. The diseases were associated with the presence of the *Stereum hirsutum* (Willd.) Pers. (1800) (first described name: *Thelephora hirsuta* Willd. (1787) and the *Phellinus igniarius* (L.) Quél. (1886) (first described name: *Boletus igniarius* (L.) (1753) (syn.: *Fomes igniarius* (L.) Fr. (1849)) (Chiarappa, 2000). Intensive research of the GTD began from 1950s. Hewitt et al. (1957) observed that specific symptoms cannot be detected on the diseased trunks every year. Chiarappa et al. (1959) identified *Cephalosporium* and *Phellinus igniarius* (L.) Quél. (1886) (first described name: *Boletus igniarius* (L.) (1753), (syn.: *Fomitiporia mediterranea* M. Fischer (Fischer, 2002) species as potential pathogens from the symptomatic plants. The research of the esca complex was continued by Laignon & Dubos (1987). Their studies focused on the question, whether the asexually

reproducing (mitospore) fungi participate alone or together with basidiomycota in the evolving of the disease. The drastic increase of the disease was observed in 1990s in those countries, where the previously effectively used arsenite was banned (Mugnai et al., 1999). During this period the sodium-arsenite has been used in a limited extent in France, Portugal and in Spain, but the esca continues to spread in the winegrowing regions (Mugnai et al., 1999). Although the appearance and the type of GTD are different in the different wine region, the increase of the disease became robust at the beginning of 2000s (Chiarappa, 2000; Grosman, 2005; Graniti, 2006).

GTDs are caused by many different Ascomycete fungi, including Diatrypaceous and Botryosphaeriaceous species, *Phomopsis* species, *Phaeoconiella* and *Phaeoacremonium* species, as well as Basidiomycetous fungi such as *Fomitiporia mediterranea* (Kuntzmann et al., 2010; Kotze et al., 2011; Bertsch et al., 2013; Úrbez-Torres et al., 2014). GTD incidence may vary between closely located vineyards possibly due to microclimate, soil composition or water supply, suggesting abiotic environmental factors are likely to have a role in the evolve of the disease. The grape variety and the type of rootstock can also affect the appearance and severity of disease symptoms (Sosnowski et al., 2007; Van Niekerk et al., 2011).

Esca disease complex, Petri disease, *Eutypa*, *Botryosphaeria*, *Phomopsis* dieback, black dead arm (BDA) and the

black foot disease are considered the major GTDs worldwide, and the causal pathogens attack the woody perennial organs of the vine and ultimately lead to the death of the plant (Lehoczky, 1974; Fontaine et al., 2015).

According to Larignon et al. (2000) the esca complex was more widespread disease comparing to Eutypa dieback in France in 90's. Black dead arm was new disease in some French wine regions, including Champagne in 2000 (Larignon & Dubos, 2001b). The importance of esca and the BDA have been realized already in the majority of French wine region in 2003 (Fotre-Muller, 2006).

The effective control of GTD faces several problems. Although the susceptibility of the different cultivars are different (Murolo & Romanazzi, 2014) and the rootstock may also affect the frequency of the symptoms there is no grapevine cultivar known to be resistant to GTD. The protection restricts currently mainly to the prevention (healthy propagating material, prevention of pruning wounds infection).

We are summarizing the present knowledge about the different disease types of GTDs. Esca disease complex, Petri disease, Eutypa- Phomopsis- and Botryosphaeria dieback, Black Dead Arm (BDA), black foot are considered the major GTDs, are caused by a variety of pathogens which attack the woody perennial organs of the vine and ultimately lead to the death of the plant (Lehoczky, 1974; Mugnai et al., 1999; Rolshausen et al. 2005; Phillips et al., 2005; Halleen et al., 2006; Úrbez-Torres et al., 2006a; Úrbez-Torres et al., 2009; Varga, 2009; Kotze et al., 2011; Úrbez-Torres, 2011a; Hofstetter et al., 2012; Bertsch et al., 2013; Fontaine et al., 2015).

### Esca disease complex

Esca is a worldwide spreading disease. The disease was already described in the ancient Greek and Latin cultures, then in the middle ages (Mugnai et al., 1999). The research of the disease began at the end of nineteenth century with Ravaz's (1898) and Viala's (1926) work. The major pathogens, which are participating in the disease: *Phaeoacremonium aleophilum* (Crous et al., 1996), (syn.: *Calosphaeria minima* Tul. & C. Tul., (1863), *Togninia minima* Tul. & C. Tul. Berl., (1900), *Erostella minima* Tul. & C. Tul. (1906), *Pleurostoma minimum* Tul. & C. Tul. M.E. Barr, J.D. Rogers & Y.M. Ju (1993), *Longoa paniculata* Curzi (1927), a *Phaeomoniella chlamydospora* Crous & W. Gams (2000) (first described name: *Phaeoacremonium clamydosporum*), a *Phaeoacremonium* sp. W. Gams, Crous, M.J. Wingf. & Mugnai (Scheck et al., 1998c; Mugnai et al., 1999; Dupont et al., 2000). Basydiomycetes species also participate in the evolving of the esca. The most widespread fungi is *Fomitiporia mediterranea* (syn: *Phellinus punctatus* and *Fomitiporia punctata* M. Fischer (2002) (first described name: *Poria punctata* P. Karst. (1882). This species cause white rot (Mugnai et al., 1999; Fischer, 2006; Kuntzmann et al., 2010; Mikulás, 2014). Essakhi et al. (2008) identified a new *Phaeoacremonium* species from the Tokaj Wine Region, which described as *Phaeoacremonium hungaricum*. Beside

the *Phaeoacremonium* and *Phaeomoniella* species can participate the *Botryosphaeria obtusa* Schwein (Shoemaker, 1964; Lehoczky, 1974; Cristinzio, 1978) (anamorph: *Diplodia seriata* de Not.) and other *Botryosphaeria* species (Bertsch et al., 2013), as well as *Cylindrocarpon* (*Ilyonectria*), *Campilocarpon* spp. (Halleen, 2006), the *Stereum hirsutum* (Ubrizsy, 1965; Mugnai et al., 1999; Armengol, 2001; Fischer & Kassemeyer, 2003; Surico et al., 2006) and *Phellinus igniarius* (Ubrizsy, 1965).

This species can cause different necrosis in the grapevine: central necrosis, punctual, sectoral necrosis or soft rot 'Amadou' (Maher et al., 2012). The esca pathogens are able to infect the plantations at different ages. The progression of the disease may take several years, and firstly appears on the woody areas, and then the destruction of the vegetative parts can be observed (Lima et al., 2010; Bertsch et al., 2013).

Different disease appearance have been described. The Petri disease is evolving in the younger plantation, the esca is forming in the older plantations (Mostert et al., 2006; Aroca et al., 2008). The apoplexy (Figure 1.) and the chronic form 'black measles' are described in the older plantation. The apoplexy appears in the middle of summer, with suddenly wilt, falling the leaves and wrinkling the berries (Mugnai et al., 1999; Fleurat-Lessard et al., 2010; Letousey et al., 2010; Mikulás, 2014). The leaves are first light green, than change to grey-green colour.



**Figure 1:** Apoplexy symptoms on Hárslevelű variety in Tokaj Wine Region (top) and on Merlot variety in Szekszárd Wine Region (under) Photo: Kovács and Sándor, 2015

The chronic form is usually evolving long period after the infection, typically in the 8 and 10 years old plantations (Mugnai et al., 1999; Fischer & Kassemeyer, 2003; Mikulás, 2008; Fleurat-Lessard et al., 2010; Letousey et al., 2010; Bertsch et al., 2013; Mikulás, 2014). According to Fussler et al. (2008), the esca attacks mainly the 15 and 25 years old plantations. The most frequent internal symptoms are the sponginess and the softness of woody tissues (Figure 2.). The necrosis can spread from up to down until the place of the grafting, but they did not attack the roots (Mugnai et al., 1999).



**Figure 2:** White rot in Chardonnay trunk  
Photo: Kovács, 2016

Chlorotic and necrotic lesions are visible on the leaves, which are called 'tiger stripe' (**Figure 3.**). These symptoms usually appear a few years after the infection, when the pathogen has colonized the vine stock, and are visible in the middle of the summer, when the dry and hot weather conditions are followed by rainy and wet one (Surico et al., 2006).



**Figure 3:** Esca symptoms on Furmint variety in Tokaj Wine Region  
Photo: Kovács and Balling, 2014

The sprouts and the twigs show decreased growth the in sprig, which they become woody in autumn. The clusters withered, the berries have low sugar content, lose their firmness, did not ripen. The disease may present just in case of some berries on the clusters (Mugnai et al., 1999).

The close relationship between the esca and the **Petri disease** has been reported worldwide (Ridgway et al., 2002; Halleen et al., 2003; Edwards & Pascoe, 2004). The disease was described first by Petri (1912) at the beginning of twentieth century in South-Italy, Sicily. The typical symptoms appear in one year old shoots (Bertsch et al., 2013; Dula, 2012; Gramaje & Armengol, 2011). The spreading of the disease is mainly through infected grafts and it cause rapid reduction in the yield (Mugnai et al., 1999). Castillo-Pando et al. (2001) and Phillips (2002) confirmed,

that *Botryosphaeria lutea* participate in the evolving of the symptoms. The foliar symptoms are between June and September in the vegetation period. The visible symptoms are not detected in all trunks and in each year (Petit et al., 2006). The presence of the brown stripes is visible in case of rudimentary esca symptom, and it can be found mainly in the older plantations (Mugnai et al., 1999; Surico et al., 2006; Bertsch et al., 2013). The young esca is the most destructive disease in case of the new plantations (Larignon & Dubos, 1997; Mugnai et al., 1999; Halleen et al., 2003; Martin & Cobos, 2007). *Phaeoacremonium* species cause brown stripes on the trunks, while *Fomitipora mediterranea* causes the white rot (Mugnai et al., 1999; Mikulás, 2014). The presence of the Petri disease was detected on Merlot variety in Eger Wine Region, Hungary since 1997, resulting the necessity of replantation for 30-50% in the vineyards (Dula, 2003, 2004). Esca was detected 11.6% in a high cordon cultivated 18 years Red Traminer variety in wine region of southern part of Balaton with Lehoczky & Makó (1983). Later Aponyi et al. (1999) investigated the esca infection in nine wine regions between 1997 and 1998, where the infection was between 8 and 21%. Rábai et al. (2008) examined the appearance of the disease between 2003 and 2005 in 13 wine regions, the highest esca presence (38.10%) was in Pannon Wine Region Balatonboglár area. The lowest infection was in Tokaj Wine Region (<2%) in this period. The abiotic and biotic factors affect the appearance of the disease (Maher et al., 2012).

### *Eutypa dieback*

*Eutypa dieback* has been described as the most common diseases, caused by *Eutypa lata* (Pers.: Fr.) Tul. & C. Tul. (syn.: *E. armeniaca* Hansf. & M.V. Carter; anamorph: *Libertella blepharis* Tul. & C. Tul (1863) A.L. Smith, Rappaz (1984) (Carter, 1988; 1991; Dula, 2012). Trouillas et al. (2011) described that other species (*Eutypella microtheca*, *Eutypella citricola*, *Diatrypella vulgaris*) can participate in the development of the disease in Australia. The pathogen can be found beyond Europe in other countries: Australia, Brazil (Paradela et al., 1993), Canada, Israel, Mexico, New-Zealand, South-Africa, USA (California, Oregon, Washington, Michigan, New York) (Carter, 1991; Munkvold, 2001). *Eutypa lata* infects through the pruning wounds (English & Davis, 1978; Lehoczky & Moller, 1979). The disease is mainly in more than one year old, typically 4 and 5 years old plantation. The most intensive infection is in the autumn period (Ramos et al., 1975b). Black stroma of the fungus is forming in the woody tissues without bark. Disease can be expected in areas where the annual precipitation is more than 250 mm (Bertsch et al., 2013), according Vajna (1983) 350 mm. The ascospores are released from the perithecia and infect the pruning wounds (English & Davis, 1978; Moller & Kasimatis; 1978; Gubler et al., 2005). Lehoczky & Moller (1979) showed in their research that the annual distribution of the precipitation correlated with the disease appearance in the Hungarian vineyards in 1970s. The ascospores spread with wind in rainy weather (Lehoczky & Moller, 1979; Gubler

et al., 2005). The optimum temperature for the germination of the ascospores is between 22 and 25°C, but according to Gubler et al. (2005) the pathogen can infect above 1°C. The largest amount of spores can be detected during winter time (Ramos et al., 1975b; Petzholdt et al., 1982; Gubler et al., 2005). The typical symptoms of the disease are the wedge shaped, zonal brownish longitudinal necrotic death in the cordon, the stunted shoots and chlorotic points on the leaves (Figure 4.) (Lehoczky, 1974; Bolay et al., 1977; Moller & Kasimatis, 1981; Duthie et al., 1991; Creaser & Wicks, 2001; 2004; Gubler et al., 2005; Rábai et al., 2005; Hluchy, 2007; Sosnowski et al., 2009; 2011). According to Gubler et al. (2005), the stromata, producing the perithecia are in the older plantations. The perithecia was observed in some Californian vineyards on the dead plants in 1937 (Lehoczky & Moller, 1979). Later, the stunted shoot resulted fruitless, small clusters (Lehoczky & Moller, 1979).



**Figure 4:** Eutypa dieback  
Photo: Kovács, 2014, South-Australia, Barossa-Valley Wine Region

The foliar symptoms was detected in spring, on 3 and 8 years old plantations (Carter, 1988; Tey-Rulh et al., 1991). The water supply of the plant collapse due to the destruction of the pathogen. The produced metabolites obstruct the nutrient delivery in the vascular tissues (Mahoney et al. 2005).

### *Botryosphaeria dieback*

The Botryosphaeria dieback has been described as several name: „Bot”, Black Dead Arm (BDA) excoryose, Botryosphaeria dieback, Diplodia dieback, Grapevine decline syndrome, Diplodia cane dieback, bunch rot, Botryosphaeria cancer (Phillips, 1998; Van Niekerk et al., 2003; Savocchia et al, 2007; Úrbez-Torres, 2011a; Van Niekerk et al., 2006). Several pathogens cause the necrosis (Auger et al., 2004; Van Niekerk et al., 2004). The species is latent pathogen. More than 20 several *Botryosphaeria* species were identified worldwide (Úrbez-Torres, 2001a), of which *Diplodia mutila* (Fr.) Mont. (1834) (teleomorph: *Botryosphaeria stevensii* Shoemaker (1964)), *Diplodia seriata* de Not. (1842) (teleomorph: *Botryosphaeria obtusa* Schwein Shoemaker

(1964) (Lehoczky, 1974; Cristinzio, 1978.), *Neofusicoccum parvum* Pennycook & Samuels (1985) (teleomorph: *Botryosphaeria parva* (Crous et al. 2006)) are the most widespread (Bertsch et al., 2013) worldwide, and *D. seriata* in the Tokaj Wine Region, Hungary (Kovács et al. 2014).

The characteristics (shape, size, surface of the conidia) of anamorph form take into consideration for the taxonomical identification (Jacobs & Rehner, 1998; Denman et al., 2000; Phillips, 2002), because the teleomorph form found rarely in nature (Shoemaker, 1964). According Alves et al. (2005) the identification based on the anamorph form is not enough for the correct identification. The species are parasites, endophytes or saprophytes (Barr, 1987; Denman et al., 2000; Phillips, 2002). Phillips (1998) has shown that the *Botryosphaeria dothidea* responsible for the whitish crust formation and the destruction of the buds and the shoots. The disease symptoms will be diverse and cannot detected on all infected trunks (Gubler et al., 2005; Sosnowski & Loschiavo, 2010). First necrosis and spotting is visible on the leaves, then the leaves begin to dry (Figures 5/a, 5/b). The spout growth decrease. Cross or longitudinal dark brown discoloration present in the vascular tissue (Figure 5/c) (Larignon, 2004). This symptom is caused by *B. obtusa* (Castillo-Pando et al. (2001) and Larignon et al. (2001a; c). The necrosis can be observed in case of infected plants at the bud burst time. The bark turns white, where the pycnidia of the *Botryosphaeria* species appear in dark spots (Gubler et al., 2005), then the clusters begin to rot (Figures 6/d, 6/e) (Gubler et al., 2005; Bonfiglioli & McGregor, 2006; Van Niekerk et al. 2006; Wunderlich et al. 2009; Mundy & Manning, 2010).



**Figure 5:** Botryosphaeria dieback in Furmint variety in Tokaj Wine Region  
Photo: Kovács, Hongtao, 2013  
(a, b: necrosis on the leaves, c: necrosis on the woody tissues, d, e: rotting berries on the cluster)

The **Black Dead Arm** (BDA) described first by Lehoczky in 1974 in Hungary, then Cristinzio (1978), Rovesti & Montermini (1987) identified it in Italy. The disease was identified in France in 1999 (Larignon et al., 2001c). The *D.*

*mutila* was described as BDA's pathogen earlier, but some researchers also confirmed the appearance other pathogens (*D. seriata*, *B. dothidea*, *L. theobromae*) (Larignon et al., 2001c; Van Niekerk et al., 2006). The presence of Botryosphaeria species is mainly in the rainy period after the pruning (Lehoczky, 1974; 1988; Alfonzo et al., 2009). *Diplodia mutila* overwinters on the grapevine, generates pycnidia, and the rainy period favours for the release of the spores (Lehoczky, 1974).

### Black foot disease

The disease may be present in the nursery or in the plantation (Halleen et al., 2006). Primary pathogens are *Ilyonectria destructans* (Zinssm.) Rossman, L. Lombard & Crous (2015) (syn.: *Cylindrocarpon destructans* (Zinssm.) Sholten (1964); *Ramularia destructans* (Zinssm.) (1918), *Cylindrocarpon macrodidymum* Schroers, Halleen & Crous (Halleen et al., 2004c) (syn.: *Cylindrocarpon macrodidymum* Schroers, Halleen & Crous (2004); *Ilyonectria macrodidyma* Halleen, Schroers & Crous (2011); *Neonectria macrodidyma* Halleen, Schroers & Crous (2004). Furthermore other two species were identified: *Campylocarpon fasciculare* Halleen, Schroers & Crous (2004) and *Campylocarpon pseudofasciculare* Halleen, Schroers & Crous (2004) (Halleen et al., 2004c). The disease was described in France in 1961 (Maluta & Larignon, 1991), then in Tasmania (Sweetingham, 1983), Sicily (Grasso, 1984), Portugal (Rego, 1994; Rego et al., 2000; 2001) were also found. Guginò & Travis (2003) identified the disease in the USA, Pennsylvania. *Cylindrocarpon obtusisporum* Cooke & Harkn., Wollenw. (1916) (currently name: *Neonectria obtusispora* Cooke & Harkn., Rossman, L. Lombard & Crous (2014) (syn.: *Fusarium obtusisporum* Cooke & Harkn. (1884)); *Ramularia obtusispora* Cooke & Harkn., Wollenw. (1916) was identified in Sicily (Grasso & Magnano di San Lio, 1975) and in California (Scheck et al., 1998a). Several *Cylindrocarpon* sp. was identified from the young, necrosis, basal rot and root necrosis showing trunks in Chile (Auger et al., 1999), Greece (Rumbos & Rumbou, 2001), Spain (Armengol et al., 2001), South-Africa (Fourie et al., 2000; Fourie & Halleen, 2001) and Australia (Edwards & Pascoe, 2004).

The symptoms are in the growing season. The new sprouts had decreased growth activity (Picture 6/a), so the plant will die by middle-summer (Halleen et al., 2006). The plants have short internodes, small leaves, on which chlorosis, necrosis, bud lack and root lesion were in the vegetation period (Halleen et al., 2006).

Black discoloration was in the woody tissue (Scheck et al., 1998c; Halleen et al., 2006), and brown, dark strips in case of the rootstocks (Rego et al., 2000). The longitudinal vascular streaking caused by *Cylindrocarpon* species in the young plants (Halleen et al., 2006).

*C. destructans* pathogen is basal part of the rootstocks in the older plantation (Rego et al., 2000). This symptom was found in 2 and 5 years old plantation in California (Scheck et

al., 1998). Typical symptoms are the striation of the venation of leaves in the nursery (Grasso & Magnano di San Lio, 1975).

### Phomopsis dieback

The *Phomopsis dieback* was described first by Lehoczky in a plantation in southern Hungary (Villány Wine Region). The disease is mainly in British Columbian Wine Region (Úrbez-Torres et al., 2009). The disease was caused by *Diaporthe perijuncta* Niessl. (1846) (Rawnsley et al., 2004) and *Phomopsis viticola* Sacc. (1905), (syn.: *Diaporthe neoviticola* Udayanga, Crous & K.D. Hyde (2012)), *Diaporthe ampelina* Berk. & M.A. Curtis R.R. Gomes, Glienke & Crous (2013) (Gomes et al., 2013; Dissanayake et al., 2015). These species are cosmopolitan, they cause the root-and fruit rot and the necrosis of the woody tissue (Uecker, 1988).

The presence of *Phomopsis viticola* was reported at the beginning of 1920s and the middle of 1960s almost in all grapevine producing region: in the eastern part of the USA, Canada (Coleman, 1928; Anderson et al., 1943; Braun, 1961; Barnes, 1963; Chamberlain et al., 1964; Willison et al., 1965), California (Hewitt, 1935), Japan (Hiura, 1924) and South-Africa (Du Plessis, 1938) areas. *Diaporthe perijuncta* causes the stunted sprouts and symptoms on the leaves and other spouts (Rawnsley et al., 2004). *Phomopsis velata* (Sacc.) Traverso, 1906 syn.: *Diaporthe ambigua* Nitschke (1870), *Diaporthe eres* Nitschke (1870) and *D. neotheicola* A.J.L. Phillips & J.M. Santos (2009) species were identified from the symptomatic woody tissue in California (Úrbez-Torres et al., 2013). *Diaporthe eres* was isolated from GTD symptomatic plants in the Tokaj Wine Region (Kovács et al., 2014).

The pycnidia are on the dead crust, under the crust and on the woody tissue. The ripened grapes cane turns white, with pycnidia on its surface (Varga, 2009; Úrbez-Torres et al., 2013). The wedge-shaped brown discoloration is on the woody tissue. The diseased plant shows decreased sprout growth and it is observed dark, blackened, irregular shape (Úrbez-Torres et al., 2013) brownish discoloration on the sprouts and leaf stalk, which become longitudinal, drying cracks (Úrbez-Torres et al., 2013). The mycelia is in the green sprouts mainly in the parenchyma tissue of the crust. Greenish-yellow, rounded, irregular chlorotic spots are on the leaves (Reddick, 1909; Úrbez-Torres et al., 2013). The symptom of the berries is in harvest period (Anco, 2011).

### Discussion

The GTD incidence has been reported to be increased during the last decades worldwide (Úrbez-Torres et al., 2014; Pollastro et al., 2000; Surico et al., 2000; Calzarano & Di Marco, 2007), and Hungary (Dula, 2011; Bihari et al., 2016). GTD incidence may vary between closely located vineyards possibly due to microclimate, soil composition

or water supply. This suggests that not only biotic, but also abiotic environmental factors are likely having role in the development of the disease (Bertsch et al., 2013).

The GTDs are caused by different Ascomyceteous pathogens, like *Diplodia seriata*, different *Phomopsis* species, *Phaeoconiella* and *Phaeoacremonium* species, moreover Basidiomyceteous fungi (e.g. *Fomitiporia mediterranea*) (Kuntzmann et al., 2010; Kotze et al., 2011; Bertsch et al., 2013; Úrbez-Torres et al., 2014). Different forms of GTDs have been detected in Hungary. Lehoczky (1974) described the black dead arm disease of grapevine, which is caused by *Diplodia mutila* (R. A. Schoemaker), in the Tokaj Wine Region. *D. seriata*, *Diaporthe eres* and *D. mutila* were identified from GTD symptomatic grapevines in the Tokaj Wine Region, Hungary (Kovács et al. 2014). *Phaeoacremonium hungarium* (S. Essakhi, L. Mugnai, G. Surico & P. W. Crous (Pers.)) has been identified as new *Phaeoacremonium* species in the Tokaj Wine Region (Essakhi et al., 2008). A new GTD pathogen, *Seimatosporium vitis* was recently described in Hungary (Váczy, 2016).

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