Eur J Plant Pathol (2018) 150:21–37
DOI 10.1007/s10658-017-1273-y

Verticillium wilt caused by *Verticillium dahliae* in woody plants with emphasis on olive and shade trees

Mojtaba Keykhasaber · Bart P. H. J. Thomma · Jelle A. Hiemstra

Accepted: 13 June 2017 / Published online: 27 July 2017

Abstract Olive plantations and tree nurseries are economically and ecologically important agricultural sectors. However, Verticillium wilt, caused by *Verticillium dahliae* Kleb., is a serious problem in olive-growing regions and in tree nurseries worldwide. In this review we describe common and differentiating aspects of Verticillium wilts in some of the main economically woody hosts. The establishment of new planting sites on infested soils, the use of infected plant material and the spread of highly virulent pathogen isolates are the main reasons of increasing problems with Verticillium wilt in tree cultivation. Therefore, protocols for quick and efficient screening of new planting sites as well as planting material for *V. dahliae* prior to cultivation is an important measure to control Verticillium wilt disease. Furthermore, screening for resistant genotypes that can be included in breeding programs to increase resistance to Verticillium wilt is an important strategy for future disease control. Collectively, these strategies are essential tools in an integrated disease management strategy to control Verticillium wilt in tree plantations and nurseries.

M. Keykhasaber · B. P. H. J. Thomma
Laboratory of Phytopathology, Wageningen University & Research, 6708 PB Wageningen, The Netherlands

M. Keykhasaber · J. A. Hiemstra (✉)
Wageningen Plant Research, Wageningen University & Research, P.O. Box 200, 6670 AE Zetten, The Netherlands
e-mail: jelle.hiemstra@wur.nl

M. Keykhasaber
Department of Plant Biotechnology, Zabol University, Zabol, Iran

Keywords *Fraxinus* · *Acer* · *Olea* · Cotton · Defoliation · VCG

Introduction

Verticillium diseases are among the most devastating plant diseases affecting numerous species worldwide, ranging from herbaceous annuals to woody perennials (Bhat and Subbarao 1999; Pegg and Brady 2002; Smith et al. 1988; Fradin and Thomma 2006). After a recent revision of the genus *Verticillium* by Inderbitzin et al. (2011), ten *Verticillium* species are distinguished, namely *V. dahliae*, *V. albo-atrum*, *V. alfalfae*, *V. longisporum*, *V. nonalfalfae*, *V. tricorpus*, *V. zaregamsianum*, *V. isaacii*, *V. nubilum* and *V. klebahnii*. Among these species *V. dahliae* Kleb. (initially isolated from *Dahlia* and described by Klebahn 1913) has the greatest economic impact and is among the most widespread plant diseases worldwide (EFSA PLH Panel 2014; Klosterman et al. 2009; Pegg and Brady 2002; Smith et al. 1988). Moreover, with a few exceptions, e.g. wilt of *Ceanothus interregimus* Hook. & Arn. and *Acer pensylvanicum* L. caused by *V. nonalfalfae* (Hibben 1959; Harrington and Cobb 1984; Kasson et al. 2014), and wilt of *Liriodendron tulipifera* L. caused by both *V. dahliae* and *V. albo-atrum* (Donohue and Morehart 1978), all reports of Verticillium wilt in trees are related to *V. dahliae.* Although no exact statistics exist on the number of species that are susceptible to *Verticillium dahliae*, it is estimated that at least 300 (Berlanger and Powelson 2005) to 400 (Klosterman et al. 2009) plant species, ranging from herbaceous

annuals to woody perennials, are affected. All woody hosts that are susceptible to Verticillium wilt belong to the Dicotyledonaceae, whereas monocotyledonous trees and Gymnophytes are not affected (Hiemstra 1998a; Sinclair and Lyon 2005). Olive plantations in the Mediterranean Basin and tree nurseries in more temperate regions are the most important agricultural sectors that involve woody species affected by the disease (Goud et al. 2011; Hiemstra and Harris 1998; Jiménez-Díaz et al. 2012). In this review, we discuss Verticillium wilt of major tree hosts as well as symptomatology, genetic diversity and detection of the pathogen, the Verticillium wilt disease cycle, reactions of infected plants and recovery, and management of Verticillium in the main woody hosts. We will concentrate on Verticillium wilts of tree species with emphasis on olive as the most important fruiting species, and on ash and maple as the most important shade trees affected by *Verticillium dahliae*.

Major tree hosts and symptoms

Olive (*Olea europaea* L.), a member of the *Oleaceae* family, is considered as one of the economically, socially and ecologically most important trees within olive producing countries. It originates from the Persian high plateau and coastal Syria, from where it was spread throughout the Mediterranean Basin, at first by the Greeks and Phoenicians, later by the Carthaginians, Romans, and Arabs. Later olive cultivation expanded to the Americas, South Africa, China, Japan and Australia (Blázquez-Martínez 1996; Civantos 2004). Verticillium wilt of olive was first reported from Italy (Ruggieri 1946), and soon thereafter from various other regions, including California, European and Asian countries as well as Australia (López-Escudero and Mercado-Blanco 2011; Navas-Cortés et al. 2008), and Argentina (Docampo et al. 1981). Initially, Verticillium wilt mostly occurred in olive groves that were established in fields that were previously used for cultivation of crops that are susceptible to *V. dahliae*, especially cotton, or in groves established next to fields with susceptible crops (Jiménez-Díaz et al. 1998, 2012). Currently, Verticillium wilt is considered as the most important disease that threatens olive production, causing serious concern to growers, nursery companies and the olive-oil industry throughout the world (López-Escudero and Mercado-Blanco 2011; Jiménez-Díaz et al. 2012; Tsror 2011). This is particularly relevant since most olive cultivars are susceptible to *V. dahliae* (Antoniou et al. 2001, 2008; Cirulli et al. 2008; López-Escudero et al. 2004; López-Escudero and Mercado-Blanco 2011), although a number of relatively resistant genotypes have been identified in artificial inoculation assays (García-Ruiz et al. 2014; López-Escudero et al. 2004; Martos-Moreno et al. 2006) as well as in field experiments (López-Escudero and Mercado-Blanco 2011; Trapero et al. 2013). However, most of the agronomically and economically relevant olive cultivars are susceptible or extremely susceptible to highly virulent strains of *V. dahliae* (López-Escudero and Mercado-Blanco 2011).

In olive, two pathotypes of Verticillium wilt have been distinguished, namely the defoliating (D) and non-defoliating (ND) pathotypes (Navas-Cortés et al. 2008). The syndrome caused by isolates that belong to the D pathotype is characterized by early drop of asymptomatic, green leaves from individual twigs and branches that eventually give rise to complete defoliation and necrosis and death of the tree. These symptoms can develop from late fall through late spring. Conversely, the syndrome caused by isolates that belong to the ND pathotype comprises two symptom complexes: an acute form ('apoplexy') and a chronic form ('slow decline') (Blanco-López et al. 1984; Jiménez-Díaz et al. 1998; Tosi and Zazzerini 1998). The 'apoplexy' form, which mainly occurs in late winter and early spring, is characterized by rapid outbreaks involving severe wilting of main and secondary branches. Leaves first become chlorotic, and then turn light-brown and roll back towards the abaxial side while remaining attached to the branches. Ultimately, a rapid dieback of twigs, shoots and branches takes place, especially in young plants, which may result in death of the entire tree (Jiménez-Díaz et al. 1998; Jiménez-Díaz et al. 2012; López-Escudero and Blanco-López 2001). The 'slow decline' syndrome is characterized by necrosis of inflorescences, chlorosis of leaves and heavy defoliation of green or dull green leaves. On infected plants, flowers mummify and remain attached to the shoots. The bark of affected shoots may become reddish-brown, and the inner vascular tissues show a dark-brown discoloration. These symptoms usually begin in spring and slowly progress to early summer (Jiménez-Díaz et al. 1998; Jiménez-Díaz et al. 2012; López-Escudero and Mercado-Blanco 2011).

Verticillium wilt is also a major problem in shade tree nurseries in more temperate regions, and can occur also in landscape plantings, especially at

locations where susceptible field crops were grown previously (Hiemstra and Harris 1998; Riffle and Peterson 1989). Maples (*Acer* spp.) are popular trees for residential and commercial landscapes, but generally very susceptible to Verticillium wilt (Gleason and Hartman 2001; Harris 1998; Frank et al. 2012). Among the most frequently grown maple species, Norway maple (*A. platanoides*) is known as a highly susceptible species on which most of the investigations on Verticillium wilt of maple have been conducted (Harris 1998; Townsend et al. 1990). *Verticillium dahliae* can induce a range of symptoms in maple including leaf yellowing, curling, wilting and necrosis. Leaf scorch can also occur at leaf margins. Leaves on one side of the tree or on just an individual branch may suddenly wilt and die. Leaves are yellowish and smaller than normal. Also a dark olive-green discolouration develops in the sapwood that is more likely to be present in the larger branches than in the smaller twigs, and is more common near the bases of larger, symptomatic branches. Infected shoots may die back leading to death of branches, and possibly of the whole tree (Frank et al. 2012; Harris 1998; Pscheidt and Ocamb 2013a).

Ash (*Fraxinus* spp.), like olive a member of the *Oleaceae* family, is another widely cultivated genus with tree species that are well-known for their high quality timber and ornamental value. Several species in this genus, especially common ash (*F. excelsior*) which is the most widely distributed species in Europe (Fraxigen 2005), may be severely affected by Verticillium wilt (Heffer and Regan 1996; Hiemstra 1998a, 1998b; Worf et al. 1994). Wilting and defoliation are the earliest symptoms of this disease on ash trees. Leaves may turn to a lighter greyish green colour before wilting or falling off. In extreme cases complete necrosis of leaves may occur. Symptoms may affect the entire crown or only part of it. Ash rarely produces the wilting and discoloration of sapwood common to other trees such as maple. However, some affected trees show a discoloration in the cambial zone, the wood or the pith of stems or branches. In summer after the first heat stress of the year upper branches may die back in a random or one-sided distribution on the tree (Hiemstra 1998b; Pscheidt and Ocamb 2013b). In young trees, although death of affected trees may occur, most of the affected trees recover. Older trees, however, show more gradual disease progress and may decline over a period of months or even years whereas others may completely recover from infection (Hiemstra 1995b). This disease may occur in all kinds of plantations: nurseries, roadside, amenity and recreational plantations as well as forest stands of ash (Hiemstra 1995a, 1995b).

Apart from the above-mentioned major tree hosts, *V. dahliae* can attack fruit tree species including stone fruits, pistachio and cocoa, as well as other shade tree species including well known genera as *Catalpa*, *Tilia*, *Ulmus* and *Robinia* (Hiemstra 1998a; Sinclair and Lyon 2005). Wilt, leaf curling or dying, abnormal red or yellow colour of entire leaves, leaf scorch, defoliation, dieback and death, and sapwood discolouration are common symptoms in most of these woody hosts infected with Verticillium wilt (Hiemstra and harris 1998; Sinclair and Lyon 2005; Stipes and Hansen 2009). Eventually, particularly infected young trees may die slowly over a period of several years or suddenly within a few weeks (Douglas 2008; Dykstra 1997; Heimann and Worf 1997).

Asymptomatic infections with *V. dahliae* have been reported to occur in olive and several other host plants (Evans and Gleeson 1973; Mathre 1986; Malcolm et al. 2013; Karajeh and Masoud 2006; Karajeh 2006). Colonization of *V. dahliae* without inducing any symptoms has also been reported on monocotoledonous plant species, such as barley, oat and wheat (Krikun and Bernier 1987; Mol 1995), and also on numerous weeds, including dicotelydonous species such as common blackberry (*Rubus allegheniensis* Porter *ex* L. H. Bailey), nettle (*Urtica* spp.), Pennsylvania smartweed *(Polygonum pennsylvanicum* L.), lamb's quarters (*Chenopodium album*), common purslane (*Portulaca oleraceae*), and black nightshade (*Solanum nigrum*) (Malcolm et al. 2013; Pegg and Brady 2002; Vallad et al. 2005). This may be explained by the fact that *V. dahliae* can colonize plant species as an endophyte without inducing any visible symptoms of disease (Malcolm et al. 2013; Petrini 1991). Endophytic colonization of *V. dahliae* in plant hosts implies that asymptomatic plants may serve as a reservoir of inoculum and may potentially initiate epidemics of Verticillium wilt disease.

Genetic diversity and detection of *V. dahliae*

Little information is available about variation of the virulence among *V. dahliae* isolates causing wilt in trees. An exception to this is the classification of isolates from olive as defoliating (D) and non-defoliating (ND)

isolates (Rodríguez-Jurado et al. 1993). This dichotomy was first described by Schnathorst and Mathre (1966) for Verticillium infections on cotton (*Gossypium hirsutum* L.). Isolates belonging to the D pathotype are highly virulent and cause complete defoliation of affected plants, whereas isolates belonging to the ND pathotype are generally less aggressive and cause milder wilt symptoms that do not include defoliation (Schnathorst and Mathre 1966). Interestingly, although isolates of both types cause defoliation in olive, isolates that belong to the D pathotype on cotton are also highly virulent on olive, while isolates that belong to the ND pathotype on cotton are also less virulent on olive (Rodríguez-Jurado et al. 1993; Schnathorst and Sibbett 1971). However, despite the high virulence of isolates of the D pathotype on cotton and olive, different levels of virulence have been observed on other hosts. Moreover, on particular plant species D isolates can be highly virulent without inducing defoliation (Jiménez-Díaz et al. 2006; Korolev et al. 2008; Schnathorst and Mathre 1966). So far, presence of the D pathotype has been reported in North and South America, Europe, the Middle East, and Asia (Jiménez-Díaz et al. 2012). However, no information is available about the differential effects of these two types on other woody hosts.

Differentiation of *V. dahliae* pathotypes infecting cotton and olive from diverse regions has been conducted through the use of molecular markers (Mercado-Blanco et al. 2001, 2003; Pérez-Artés et al. 2000). Pérez-Artés et al. (2000) designed PCR primers specific for D and ND isolates of *V. dahliae,* based on sequences of pathotype-associated RAPD bands, and tested them on 67 *V. dahliae* isolates from cotton and olive collected from southern Spain, China, Italy and the USA. Subsequently, nested-PCR primers were designed and optimized for specific detection of D and ND pathotypes *in planta* and in soil samples (Mercado-Blanco et al. 2002; Pérez-Artés et al. 2005). However, although these primers have worked for several isolates tested worldwide, it was found that they do not produce the desired amplicon on all *V. dahliae* isolates (Collins et al. 2005), which may be explained by the high genetic variability that exists among *V. dahliae* isolates (De Jonge et al. 2012, 2013; Faino et al. 2015, 2016). Arguably, genetic differences between *V. dahliae* isolates are underlying their behaviour as D or as ND, and the most reliable molecular marker would be a marker on the gene(s) that is (are) responsible for the differential disease phenotype. Currently, however, the molecular mechanism that explains the occurrence of defoliation by some isolates and non-defoliation by others remains unknown.

PCR based assays for detection of *V. dahliae* have been developed by several groups for detection in soil (e.g. Pérez-Artés et al. 2005; DeBode and Van Poucke 2011; Bilodeau et al. 2012) as well as for detection in plant samples (e.g. Schena et al. 2004; Karajeh and Masoud 2006; Gayoso et al. 2007). So far, however, these protocols have not been developed into procedures for routine screening of planting stock or fields to be planted with crops susceptible to Verticillium wilt. If field soils are screened before planting this uHiemstra, 1995a sually is done using laborious and time consuming wet or dry sieving and plating techniques (Hiemstra, 2015a; Termorshuizen 1998).

Another way to characterize genetic diversity in fungi is to classify individual isolates in vegetative compatibility groups. According to the ability of individual fungal strains to undergo hyphal anastomosis and form stable heterokaryons they can be classified into vegetative compatibility groups (VCGs), such that compatible isolates are placed in the same VCG group (Joaquim and Rowe 1990; Katan 2000; Leslie 1993). *V. dahliae* isolates have been classified into six VCGs (VCG1 through VCG6). VCG1, VCG2 and VCG4 were further divided into subgroups A and B based on the frequency and vigour of complementation (Chandelier et al. 2003; Chen 1994; Dervis et al. 2010; Goud and Termorshuizen 2002; Jiménez-Díaz et al. 2006; Jiménez-Díaz et al. 2011; Jiménez-Díaz et al. 2012; Korolev et al. 2000, 2001, 2008; López-Escudero and Mercado-Blanco 2011). So far, vast numbers of *V. dahlia*e isolates from maple, ash, olive and some other woody hosts in USA and Europe have been analysed through complementation tests and classified into VCG1A, VCG1B, VCG2A, VCG2B and VCG4B (Chandelier et al. 2003; Chen 1994; Hiemstra and Rataj-Guranowska 2000; Jiménez-Díaz et al. 2011; Jiménez-Díaz et al. 2012; Neubauer et al. 2009). Recently, however, Papaioannou and Typas (2015) showed that although VCGs may be helpful in characterising different isolates, they are genetically not completely isolated.

Disease cycle of verticillium wilt of trees

The life cycle of *V. dahliae* consists of a parasitic part, in which the fungus lives in its host, and a non-parasitic part, in which it is dormant. For tree hosts the disease

cycle of *V. dahliae* has been described in detail by Hiemstra (1998a) (Fig. 1). During the non-parasitic phase in the soil, *V. dahliae* survives as microsclerotia, either as dispersed propagules or embedded within plant debris, mainly in the upper layer of the soil from where it can easily be spread by wind, rain or irrigation water, human and animal activities, and agricultural tools and machines (Pegg and Brady 2002; Schnathorst 1981; Wilhelm 1950). Microsclerotia are very persistent and enable the pathogen to attack new plantings even after a long period without hosts being present (Wilhelm 1955). The infection process of *V. dahliae* in woody plants is similar to that in herbaceous plants. Microsclerotia are stimulated to germinate by exudates from nearby growing roots (Schreiber and Green 1963). *V. dahliae* begins its parasitic phase when hyphae from germinating microsclerotia penetrate roots of a susceptible host (Lockwood 1977; Nelson 1990; Schreiber and Green 1963). Subsequently, hyphae grow inter- and intracellular within the root cortex to reach the xylem vessels and enter these (Prieto et al. 2009). Vallad and Subbarao (2008) reported that *V. dahliae* germ tubes can form appressoria before penetration and colonization of the cortical tissues. Also inflated structures that are thought to be functionally analogous to appressoria, and that are named "hyphopodia", were observed at the penetration sites of roots of cotton and olive inoculated with GFP-labelled isolates of *V. dahliae* (Reusche et al. 2014; Zhao et al. 2016; Keykhasaber 2017). It has recently been proposed that expression of the tetraspanin VdPls1 and VdNoxB, a catalytic subunit of membrane-bound NADPH oxidases for reactive oxygen species (ROS) production, in hyphopodia is essential for redirecting fungal growth toward host cells to penetrate roots and to colonize the host vascular system (Zhao et al. 2016).

Once inside the vessels, *V. dahliae* colonizes susceptible plants through conidia that are carried with the flow of xylem fluid (Keykhasaber 2017) until they are trapped at vessel ends or protruding parts of vessel elements. Here they may germinate and the new hyphae penetrate into adjacent vessel elements (Beckman et al. 1976). During this process of colonization of the xylem of infected

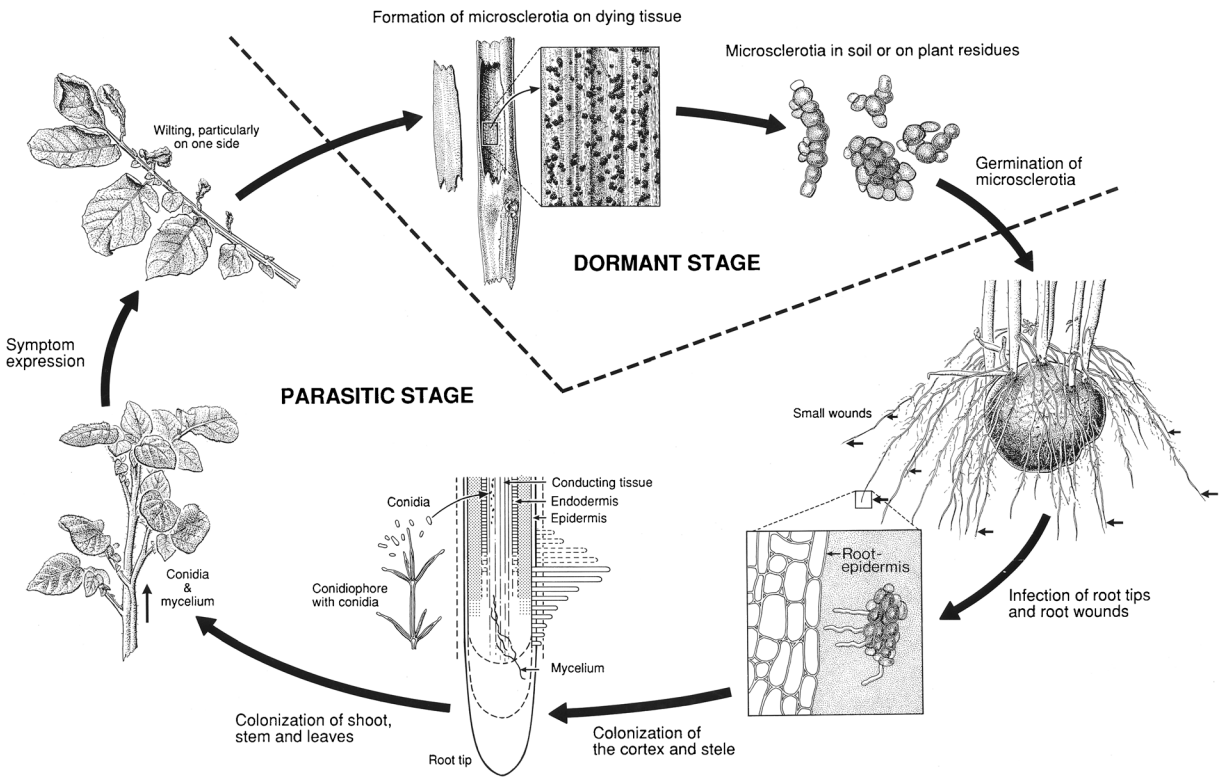

Fig. 1 Disease cycle of *V. dahliae* in trees (Drawing by P.J. Kostense; reprinted with permission from Hiemstra and Harris 1998)

plants cycles of fungal proliferation and (partial) fungal elimination (probably driven by plant defence responses) may occur (El-Zik 1985; Fradin and Thomma 2006; Klosterman et al. 2009; Heinz et al. 1998; Mercado-Blanco et al. 2003). Accumulation of *V. dahliae* hyphae, ultrastructural and chemical alterations resulting from defense reactions, and aggregates resulting from degradation of external material of the xylem vessel walls by fungal enzymatic activity may cause occlusion of *V. dahliae*-infected xylem vessels (Baídez et al. 2007; Hiemstra and Harris 1998; Pegg and Brady 2002). As a result, the water flow through the xylem is hampered and symptoms of water stress may develop. Wilting, defoliation and early senescence comprising chlorosis, necrosis, and stunting are the main symptoms of Verticillium wilt disease (Fig. 2). Moreover, sparse foliage and branch dieback may also occur (Berlanger and Powelson 2005; Hiemstra 1998a; Riffle and Peterson 1989; Sinclair and Lyon 2005). Plants with acute infections may start with symptoms on individual branches or on one side of the plant. This is often called "flagging", which can be diagnostic for Verticillium wilt disease. Furthermore, one or several branches may suddenly wilt and die and buds may fail to leaf out in spring (Douglas 2008; Himelick 1968; Piearce and Gibbs 1981).

Finally, at late stages of the disease, resting structures are formed in dying tissues. Melanized microsclerotia are the survival structures of *V. dahliae* (Klosterman et al. 2009). Light and electron microscopy analysis of morphological events during microsclerotia formation has shown that initially hyphae become swollen, vacuolated, and form numerous septa. Subsequently, clusters of hyphal cells form in these swollen hyphae. Finally, melanin particles are extruded into the interhyphal spaces of the microsclerotium, and the individual cells of mature microsclerotia also possessed a thickened cell wall surrounded by melanin (Klimes et al. 2008; Xiong et al. 2014). This melanin enables the microsclerotia to resist UV irradiation, temperature extremes, enzymatic lysis, and fungicidal activities (Bell and Wheeler 1986). The genes involved in melanin biosynthesis have been identified and their functions have been characterized (Gao et al. 2010; Tzima et al. 2012; Klimes et al. 2008; Xiong et al. 2014). It was also confirmed that *VDH1*, encoding a class II hydrophobin, is involved in the formation of microsclerotia (Klimes et al. 2008; Klimes and Dobinson 2006). In addition, many genes have been characterized that are involved in signal transduction pathways and regulation of microsclerotia formation of *V. dahliae*, such as *VMK1*, encoding a mitogen-activated

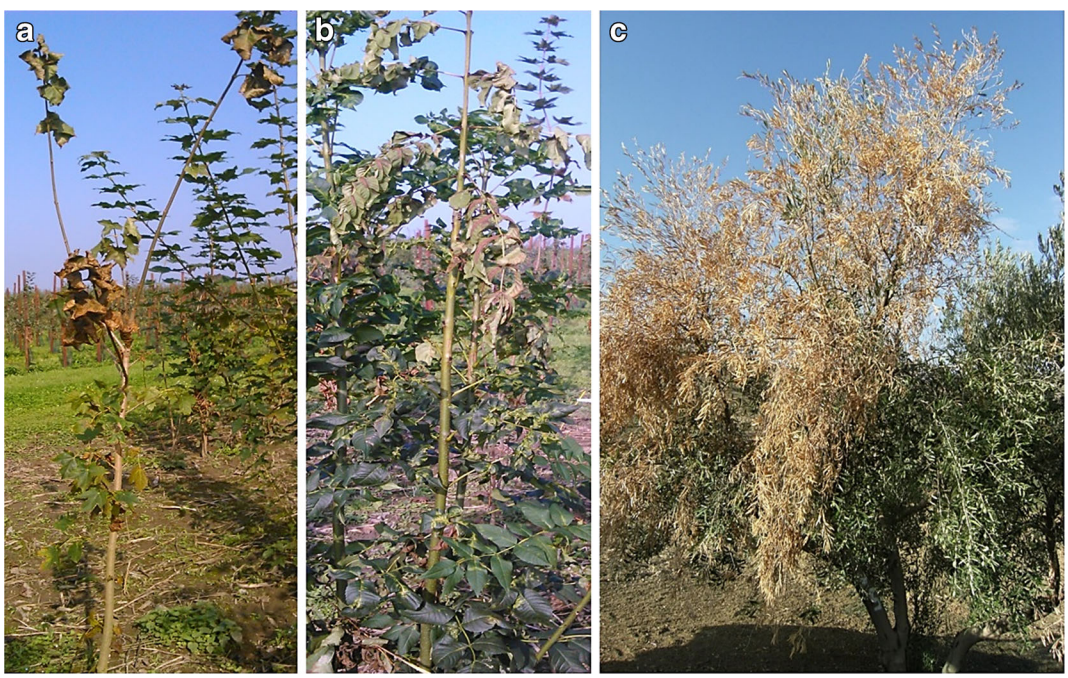

Fig. 2 Wilting and leaf necrosis in maple, ash and olive trees affected by *Verticillium dahliae*. **a** Wilting and desiccation of leaves in a young maple tree (photograph: M. Keykhasaber). **b** Necrosis and wilting of leaves in a young ash tree (photograph: M. Keykhasaber). **c** Partial dieback of shoots and branches in an olive tree (photograph: J.A. Hiemstra)

protein kinase (Rauyaree et al. 2005), *VdGARP1*, encoding a glutamic acid-rich protein (Gao et al. 2010), the G protein β subunit (named as VGB) (Tzima et al. 2012), *Vta2*, encoding a nuclear zinc finger protein, (Tran et al. 2014), the MADS-box transcription factor *VdMcm1* (Xiong et al. 2016), the mitogen-activated protein kinase (MAPK) *Hog1* (Wang et al. 2016), and the MAPK *Msb* and *Pbs2* (Tian et al. 2014; Tian et al. 2016). Unravelling the functional characterizations of these genes provides insight into the genetic control of microsclerotia formation in *V. dahliae.*

The presence of *V. dahliae* in petioles of infected trees in the form of microsclerotia has been demonstrated for several tree species including *Acer* (Hiemstra 1997), *Liriodendron tulipifera* (Morehart and Melchior 1982), olive (Prieto et al. 2009), and *Fraxinus* (Rijkers et al. 1992). Recently, formation of microsclerotia was also found inside peduncles and flowers of infected olive trees (Trapero et al. 2011). After incorporation of infected plant debris in the top soil layer and decomposition by the activity of soil-borne organisms, microsclerotia survive in the soil for prolonged times (years) and become available as inoculum for new infections (Hiemstra 1997; Hiemstra and Harris 1998; Morehart and Melchior 1982; Rijkers et al. 1992; Tjamos and Botseas 1987; Tjamos and Tsougriani 1990; Townsend et al. 1990).

Reactions of infected trees and recovery

In some cases trees infected by Verticillium wilt are able to recover from the disease (Hiemstra 1998a). This phenomenon has been reported in olive as re-growth from existing crowns that suffer from limited dieback, or from the stem base after complete dieback (López-Escudero and Blanco-López 2001, 2005; Levin et al. 2003; Markakis et al. 2009; Mercado-Blanco et al. 2001). Recovery has similarly been reported in *Catalpa bignonioides* and *Sassafras albidum* (Kasson et al. 2015) as re-growth from the crown, in *Acer platanoides* with re-growth from stem base after extensive dieback (Goud et al. 2011), and in *Fraxinus excelsior* as re-growth without dieback of twigs (Hiemstra 1998b). The inherent structure of the xylem and the ability of trees to produce new layers of xylem has a significant impact on the potential of recovery (Banfield 1968; Emechebe et al. 1974; Sinclair et al. 1981; Tippett and Shigo 1981). In ring-porous tree species (like robinia and ash) most of the water transport is in the most recent growth ring. This implicates that as long as these trees are able to produce new xylem vessels every year, they can substitute their blocked vessels with new ones, which enables complete recovery, often even without dieback of the crown. In tree species with a diffuse-porous structure of the xylem, such as maple, xylem vessels in each growth ring remain functional for several years. Hence loss of a major part of the water transport capacity in infected trees often cannot sufficiently be compensated by the wood in a new growth ring. Such trees therefore probably show much more dieback of the aerial parts and recovery starts by regrowth from healthy parts of the stem base or roots (Hiemstra and Harris 1998; Keykhasaber 2017). Compared to the healthy plants, however, recovered plants have higher probability of becoming diseased again (Goud et al. 2011).

Differences in the severity of symptoms and in the percentage of recovery in tree species may be related to differences in the capacity to compartmentalize infected xylem. Compartmentalization is a boundary-setting process that is activated following fungal vascular invasion and tends to limit the spread of infection and the loss of normal functioning of sapwood (Hiemstra 1998a; Nicole and Gianinazzi-Pearson 1996; Shigo 1984; Tippett and Shigo 1981). Compartmentalization was first proposed as a mechanism against spread of decay in trees by isolating the damaged tissues and replacing it by new functional tissues (Shigo and Marx 1977). Later it was reported that this mechanism that causes changes in anatomy and chemistry of xylem cells also has an important role in protecting trees against colonization by vascular pathogens (Bonsen et al. 1985; Shigo 1984; Tippett and Shigo 1981; Manion 2003; Smith 2006). The principle of the compartmentalization lies in the establishment of four types of “walls”. While wall 1 restricts pathogen movement longitudinally, wall 2 consists of the growth ring boundary and restricts pathogen movement centripetally, and wall 3 limits the tangential movement of pathogen and is associated with ray parenchyma. Wall 4 is the strongest and referred to as the “barrier zone” that is produced by cambial activity after injury or infection of the existing xylem, and separates the tissue present at the time of infection from new, uninfected tissue (Shigo 1984). It has been reported that, shifts in enzymes and growth regulators in the vascular cambium result in the formation of barrier zone (Shigo 1984; Shigo and Dudzik 1985; Smith and Shortle 1990; Tippett and Shigo 1981). Also a waxy layer of suberin may be synthesized at the boundary between healthy and infected sapwood (Pearce

1990). In fact, suberization response is a heritable trait under control of genetic elements that can be considered as part of a breeding program for increased compartmentalization (Biggs et al. 1992). Studies on clones of *Populus deltoides* Bartr. (eastern cottonwood) and *Liquidambar styraciflua* L. (sweetgum) have shown that different clones vary in their compartmentalization ability, suggesting that this phenomenon is under genetic control, and making it possible to screen species for genotypes that display superior compartmentalization traits (Garrett et al. 1979; Shain and Miller 1988; Smith 2006).

In vascular diseases of annual plants, it has been observed that infection may induce transdifferentiation of bundle sheath cells to novel, functional xylem vessels, or may increase xylem cells within the vascular bundle as a result of prolonged or renewed activity of the vascular cambium (Reusche et al. 2012). This phenomenon allows novel vegetative growth of affected stems and branches and enhances recovery (Tjamos et al. 1991; López-Escudero and Blanco-López 2001). Seven putative NAC (for NAM, ATAF1/2, and CUC2) transcription factors have been identified in *Arabidopsis thaliana*, which are involved in transdifferentiation and fall into the VND subfamily (Vascular related NAC Domain) (Demura et al. 2002; Kubo et al. 2005; Yamaguchi et al. 2010). Within this subfamily, VND6 and VND7 seem to have specific roles on *Verticillium*-triggered transdifferentiation of bundle sheath cells, with VND6 regulating metaxylem (xylem tissue that consists of rigid thick-walled cells and occurs in parts of the plant that have finished growing) formation, and VND7 inducing protoxylem (the first-formed xylem tissue, consisting of extensible thin-walled cells thickened with rings or spirals of lignin) development (Kubo et al. 2005; Reusche et al. 2012). Interestingly, homologs of NAC domain protein genes (*PtVNS/PtrWND*) have been identified in poplar (*Populus trichocarpa*) and their role in differentiation of the xylem vessel element has been demonstrated (Hu et al. 2010; Ohtani et al. 2011). This implies that similar mechanisms may occur in tree species resulting in increased numbers of vascular elements being formed after vascular infection. Thus, studying the distribution of these genes or their homologs in other trees, and also their impact on *Verticillium*-triggered changes in differentiation of cells from the cambium or even within existing tissues, may help to design strategies to stimulate recovery of susceptible trees.

Expansion of *V. dahliae* in xylem vessels of infected plants triggers defense reactions, including ultrastructural and chemical alterations (Adams and Thomas 1985; Hiemstra and Harris 1998; Pegg and Brady 2002). Host plants may deposit coating materials (such as lipid-rich or fibrillar coatings) onto xylem vessel walls and into pit membranes (Robb et al. 1982; Street et al. 1986), and accumulate gums and form tyloses to prevent pathogen spread (Baídez et al. 2007). Infected plants also secrete phytoalexins, terpenoid and phenolic substances that have antimicrobial activity during pre-vascular and vascular phases of infection (Daayf et al. 1997; Laouane et al. 2011; Mace et al. 1989; Mansfield 2000; El-Zik 1985; Rodríguez-Jurado et al. 1993; Ryan and Robards 1998; Treutter 2006). In olive trees infected with *V. dahliae* it was observed that the level of phenolic components such as verbascoside, quercetin, luteolin aglycons, rutin, oleuropein, luteolin-7-glucoside, tyrosol, p-coumaric acid and catechin increased in vascular tissues during infection and colonization (Baídez et al. 2007; Markakis et al. 2010). Their antifungal activity against *V. dahliae* was substantiated by in vitro studies, suggesting they are involved in defense (Baídez et al. 2007). Moreover, chemical composition of wood plays a role in the variation of effectiveness of pathogen compartmentalization between tree genotypes (Rolshausen et al. 2008). Plant phenolics contribute substantially to structural lignin as well as many seasonal pigments and defensive compounds in plants (Cheynier et al. 2013). The tannin group of phenolic compounds is widespread in both constitutive protection and induced compartmentalization process. Tannins in foliage and wood are reactive pro-oxidant compounds (i.e., chemicals that induce oxidative stress, either by generating reactive oxygen species or by inhibiting antioxidant systems) that disrupt microbial metabolism (Antilla et al. 2013). Additionally, it has been determined that trees with small xylem vessels have stronger ability than trees with large xylem vessels to compartmentalize infections (Eckstein et al. 1979).

Management of the disease

Control of Verticillium wilt is very difficult due to the characteristics of the pathogen and the nature of the infection. Especially the long survival time of microsclerotia in soil, the long lifetime of a tree with continuous exposure to inoculum present in the soil, and the absence of methods to cure infected trees are important factors. Consequently, the use of an integrated strategy is the best way to deal with this

disease. This includes the employment of resistant cultivars or rootstocks, cultural practices (i.e., avoid intercropping with *V. dahliae* susceptible crops; minimise cultivation practices that damage the roots; avoid contaminated equipment; and avoid irrigation that may disseminate the pathogen) to avoid spreading of the disease, and measures (i.e., disinfestation of *V. dahliae*-infested soil with fumigants, soil solarisation) to avoid build-up of soil inoculum and to reduce soil inoculum levels wherever possible (Barranco et al. 2010; Hiemstra 2015b; Jiménez-Díaz et al. 1998; López-Escudero and Mercado-Blanco 2011; Tjamos and Jiménez-Díaz 1998). Green amendments or biological soil infestation would be also promising methods for control of Verticillium wilt in tree nurseries, but these methods are costly and the efficacy relies on the soil type (Blok et al. 2000; Hiemstra et al. 2013). Moreover, accurate quantification of inoculum in soil would provide valuable information for disease prediction, since density of inoculum in soil is correlated with final disease incidence values (Goud et al. 2011; López-Escudero and Blanco-López 2007). Replacement of diseased trees with non-host plants might also be an environmentally friendly management solution to control Verticillium wilt. Studies on replacement of dead or severely diseased olive trees with apple trees revealed that this would be an appropriate approach in an integrated disease management to control Verticillium wilt disease (Karajeh and Owais 2012). Use of biological control agents, including beneficial bacteria is another approach to manage Verticillium wilts (Markakis et al. 2015; Mercado-Blanco et al. 2004; Prieto et al. 2009). However, the use of resistant cultivars and the screening of new planting sites and planting stock for infection by *V. dahliae* are the most efficient tools for control of Verticillium wilt of trees (Hiemstra 2015b; López-Escudero and Mercado-Blanco 2011; Tjamos and Jiménez-Díaz 1998). Measures to be included in integrated strategies for control of Verticillium wilt in trees are summarized in

Table 1 Elements in integrated strategies to control Verticillium wilt in trees (compiled from Barranco et al. 2010, Hiemstra 2015b; López-Escudero and Mercado-Blanco 2011)

General principles	
	• Only use Verticillium free propagation and planting material • Avoid introduction of Verticillium into fields • Prevent build-up of soil inoculum of Verticillium
Specific measures	
Before planting	• Screen field soil and planting stock for presence of Verticillium • If soils are infested soil inoculum levels may be reduced by cultural measures such as fumigation, biofumigation, solarisation or biological disinfestation
At planting	• Plant susceptible species only on soils free of Verticillium • On infested soils resistant species or cultivars (if available) may be used • Care should be taken to avoid Verticillium being introduced through contaminated soil or crop residues on machinery • Do not plant susceptible crops next to fields with highly susceptible crops such as cotton or tomato, or on fields with a history of such crops
After planting	• Avoid remains of diseased crops from nearby fields being blown into the field • Prevent leaves and fruits from infected plants from being spread within the field • Keep soils free from weeds by using herbicides, superficial disking or cover crops • Do not intercrop with vegetables or cover crops that are susceptible to Verticillium • Remove and destroy diseased plants and replace by resistant varieties or non-hosts • Before replanting soil inoculum may be reduced locally by applying fumigation, biofumigation, solarisation or biological disinfestation • Clean machinery before moving to another field • Disinfect pruning tools before moving to another tree • Do not use pruning material from infected trees for mulching and check organic amendments for contamination with Verticillium • Avoid soil or fallen leaves being moved by run-off water by using reduced tillage systems or growing cover crops • Minimize dose and limit frequency of irrigation and fertilize in a balanced manner

Fig. 3 Inoculation methods in trees. **a** rood-dipping inoculation. **b** stem-inoculation of a tree by making a horizontal incision through the bark into the xylem of the stem by using a snap-off cutter, and putting the conidiospore suspension on the knife with a disposable transfer pipette. **c** stem-inoculation of a tree by drilling a trunk hole through the bark of the stem into the xylem and injecting a conidial suspension

Table 1. More elaborated decision schemes for control of Verticillium wilt especially in olive are provided by Hiemstra (2015b) and López-Escudero and Mercado-Blanco (2011).

To evaluate the level of resistance of particular plant genotypes, the pathogen inoculation method that is used, the virulence of the isolates, and environmental conditions all influence the response of the genotype (Levin et al. 2003; López-Escudero et al. 2010; Blanco-López et al. 1998; López-Escudero and Mercado-Blanco 2011). In all programs for evaluating host resistance to *V. dahliae* it is important to include isolates with different levels of virulence, since different *V. dahliae* isolates may be differentially pathogenic to different host genotypes (Barbara et al. 1998). Root-dipping and stem inoculation are the most common methods to inoculate trees (Rodríguez-Jurado et al. 1993; Resende et al. 1995) (Fig. 3). In the root-dipping method, the bare root system is inoculated with conidiospore suspension, microsclerotia, or a semisolid fluid mass of mycelium (García-Ruiz et al. 2014), while in the stem inoculation method the stem of tree is inoculated by drilling a trunk hole or making a horizontal incision of a few millimetres deep through the bark of the stem into the xylem to inject a conidiospore suspension (Antoniou et al. 2008; Hiemstra 1995b). Both of these methods have characteristics that must be taken into account during the evaluation of disease progression. Stem inoculation results in a rapid development of disease due to injecting a dense conidial pathogen suspension directly into the vascular tissue of the trees to enable fast resistance evaluation (Antoniou et al. 2008). Upon this type of inoculation, the pathogen escapes the resistance mechanisms operating in roots (Chen et al. 2004; Gold and Robb 1995; Heinz et al. 1998), while upon root dipping the first fungal elimination that occurs in the roots may affect the distribution of the pathogen and progression of the disease. The type of inoculum used for root dipping may also impact colonization by the pathogen and disease progression (Markakis et al. 2009). Also, there is a correlation between inoculum density and final disease incidence values (López Escudero and Blanco-López 2007; Bejarano-Alcázar et al. 1995). Moreover, it has been observed that for a given inoculum density, disease incidence varies greatly depending on the crop (Berbegal et al. 2007; Xiao and Subbarao 1998; Grogan et al. 1979; Harris and Yang 1996; Atallah et al. 2011). Consequently, validation and standardization of *V. dahliae* inoculation methods and inoculum density is needed to provide accurate assessment of wilt resistance in tree hosts.

Conclusion

Over the last decades, a strong increase of Verticillium Wilt of olive was associated with the establishment of new olive orchards on infested soils, the use of infected plant material, and the spread of highly virulent pathogen isolates. Also tree nurseries are often established on former agricultural land where hosts of *V. dahliae* may have been grown, and therefore may serve as infection sources. Therefore, improving the resistance of cultivars, as well as developing protocols for fast and reliable detection of *V. dahliae* in planting stocks and at planting sites are of the highest importance for establishing effective integrated disease management strategies. PCR-based methods for sensitive and accurate detection and discrimination of *V. dahliae* isolates allow for the rapid and reliable

assessment of soil contaminations and plant infections by *V. dahliae.* Furthermore, revealing the genetics and molecular background of resistance mechanisms, and of the recovery phenomenon, may provide essential information that can be used in breeding programs to increase the natural resistance of trees against Verticillium wilt. Collectively, these two approaches will provide essential tools for integrated disease management strategies to control Verticillium wilt in tree plantations and nurseries.

Acknowledgements Work on Verticillium wilts of trees at Wageningen Plant Research was supported financially by a scholarship of the ministry of science and technology of Iran to MK. BPHJT acknowledges support of the Research Council Earth and Life Sciences (ALW) of the Netherlands Organization of Scientific Research (NWO).

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

Adams, G., Thomas, C. (1985). *Verticillium wilt of shade trees and woody ornamentals*. Extension Bulletin E-1860, Cooperative Extension Service. USA: Michigan State University.

Antilla, A.-K., Pirttilä, A. M., Häggman, H. M., Harju, A., Venäläainen, M., Haapala, A., et al. (2013). Condensed conifer tannins as antifungal agents in liquid culture. *Holzforschung, 67*, 825–832.

Antoniou, P.P., Tjamos, E.C., Kaltsis, J., Tjamos, S.E. (2001). Resistance evaluation to Verticillium dahliae in young olive cultivars or in root stocks of established olive orchards. Proceedings of the 8th International Verticillium Symposium, 2001. Córdoba, p 31.

Antoniou, P. P., Markakis, E. A., Tjamos, S. E., Paplomatas, E. J., & Tjamos, E. C. (2008). Novel methodologies in screening and selecting olive varieties and root-stocks for resistance to *Verticillium dahliae*. *European Journal of Plant Pathology, 110*, 79–85.

Atallah, Z. K., Hayes, R. J., & Subbarao, K. V. (2011). Fifteen years of Verticillium wilt of lettuce in America's salad bowl: A tale of immigration, subjugation, and abatement. *Plant Disease, 95*(7), 784–792.

Baídez, A. G., Gomez, P., Del Rio, J. A., & Ortuno, A. (2007). Dysfunctionality of the xylem in *Olea europaea* L. plants associated with the infection process by *Verticillium dahliae* Kleb. Role of phenolic compounds in plant defense mechanism. *Journal of Agricultural and Food Chemistry, 55*, 3373–3377.

Banfield, W. M. (1968). Dutch elm disease recurrence and recovery in American elm. *Phytopathologische Zeitschrift, 62*, 21–60.

Barbara, D. J., Paplomatas, E. J., & Jiménez-Díaz, R. M. (1998). Variability in *V. dahliae*. In J. A. Hiemstra & D. C. Harris (Eds.), *A compendium of Verticillium wilts in tree species* (pp. 43–45). Wageningen: Ponsen and Looijen.

Barranco, D., Fernandez-Escobar, R., Rallo, L. (2010). Olive growing. MundiPrensa/Australian Olive Association Ltd., Junta de Andalucía, 756 pp.

Beckman, C. H., Vandermolen, G. E., & Mueller, A. C. (1976). Vascular structure and distribution of vascular pathogens in cotton. *Physiology of Plant Pathology, 9*, 87–94.

Bejarano-Alcázar, J., Melero-Vara, J. M., Blanco-López, M. A., & Jiménez-Díaz, R. M. (1995). Influence of inoculum density of defoliating and non-defoliating pathotypes of *Verticillium dahliae* on epidemics of Verticillium wilt of cotton in southern Spain. *Phytopathology, 85*, 1474–1481.

Bell, A. A., & Wheeler, M. H. (1986). Biosynthesis and functions of fungal melanins. *Annual Review of Phytopathology, 24*, 411–451.

Berbegal, M., Ortega, A., García-Jiménez, J., & Armengol, J. (2007). Inoculum density-disease development relationship in Verticillium wilt of artichoke caused by *Verticillium dahliae*. *Plant Disease, 91*, 1131–1136.

Berlanger, I., Powelson, M.L. (2005). Verticillium wilt. The Plant Health Instructor. DOI: 10.1094/PHI-I-PHI-I-2000-0801-01.

Bhat, R. G., & Subbarao, K. V. (1999). Host range specificity in *Verticillium dahliae*. *Phytopathology, 89*, 1218–1285.

Biggs, A. R., Miles, N. W., & Bell, R. L. (1992). Heritability of suberin accumulation in wounded peach bark. *Phytopathology, 82*, 83–86.

Bilodeau, G. J., Koike, S. T., Uribe, P., & Martin, F. N. (2012). Development of an assay for rapid detection and quantification of *Verticillium dahliae* in soil. *Phytopathology, 102*, 331–343.

Blanco-López, M. A., Jiménez-Díaz, R. M., & Caballero, J. M. (1984). Symptomatology, incidence and distribution of Verticillium wilt of olive trees in Andalucía. *Phytopathologia Mediterranean, 23*, 1–8.

Blanco-López, M. A., Hiemstra, J., Harris, D., López-Escudero, F. J., & Antoniou, P. (1998). Selection and screening for host resistance. In J. Hiemstra & D. Harris (Eds.), *Compendium of Verticillium wilt in tree species* (pp. 51–54). Ponsen & Looijen: Wageningen.

Blázquez-Martínez, J.M. (1996). Evolution and history, p. 17–54. In: International Olive Oil Council (IOOC) (eds.). World olive encyclopedia. Madrid.

Blok, W. J., Lamers, J. G., Termorshuizen, A. J., & Bollen, G. J. (2000). Control of soilborne plant pathogens by incorporating fresh organic amendments followed by tarping. *Phytopathology, 90*, 253–259.

Bonsen, K.J.M., Scheffer, R.J., Elgersma, D.M. (1985). Barrier zone formation as a resistance mechanism of elms to Dutch elm disease. International Association of Wood Anatomists Bulletin N.S., 6, 71-77.

Chandelier, A., Laurent, F., Dantinne, D., Mariage, L., Etienne, M., & Cavelier, M. (2003). Genetic and molecular characterization of *Verticillium dahliae* isolates from woody ornamentals in Belgian nurseries. *European Journal of Plant Pathology, 109*, 943–952.

Chen, W. (1994). Vegetative compatibility groups of *Verticillium dahliae* from ornamental woody plants. *Phytopathology, 84*, 214–219.

Chen, P., Lee, B., & Robb, J. (2004). Tolerance to a non-host isolate of *Verticillium dahliae* in tomato. *Physiology and Molecular Plant Pathology, 64*, 283–291.

Cheynier, V., Comte, G., Davies, K. M., Lattanzio, V., & Martens, S. (2013). Plant phenolics: Recent advances on their biosynthesis, genetics, and ecophysiology. *Plant Physiology and Biochemistry, 72*, 1–20.

Cirulli, M., Colella, C., D'Arnico, M., Amenduni, M., & Bubici, G. (2008). Comparison of screening methods for the evaluation of olive resistance to *Verticillium dahliae* Kleb. *Journal of Plant Pathology, 90*, 7–14.

Civantos, L. (2004). Olive growing in the world and in Spain. In D. Barranco, R. Fernández-Escobar, & L. Rallo (Eds.). (2010). Olive Growing (pp. 17–35). Australia: RIRDC.

Collins, A., Mercado-Blanco, J., Jiménez-Díaz, R. M., Olivares, C., Clewes, E., & Barbara, D. J. (2005). Correlation of molecular markers and biological properties in *Verticillium dahlia*e and the possible origins of some isolates. *Plant Pathology, 54*, 549–557.

Daayf, F., Nicole, M., Boher, B., Pando, A., & Geiger, J. P. (1997). Early vascular defense reactions of cotton roots infected with defoliating mutant strain of *Verticillium dahliae*. *European Journal of Plant Pathology, 103*, 125–136.

De Jonge, R., Peter van Esse, H., Maruthachalam, K., Bolton, M. D., Santhanam, P., Keykha Saber, M., Zhang, Z., Usami, T., Lievens, B., Subbarao, K. V., & Thomma, B. P. H. J. (2012). Tomato immune receptor Ve1 recognizes effector of multiple fungal pathogens uncovered by genome and RNA sequencing. *Proceedings of the National Academy of Sciences of the United States of America, 109*(13), 5110–5115.

De Jonge, R., Bolton, M. D., Kombrink, A., van den Berg, G. C. M., Yadeta, K. A., & Thomma, B. P. H. J. (2013). Extensive chromosomal reshuffling drives evolution of virulence in an asexual pathogen. *Genome Research, 23*(8), 1271–1282.

DeBode, J., & Van Poucke, K. (2011). Detection of multiple Verticillium species in soil using density flotation and real-time polymerase chain reaction. *Plant Disease, 95*, 1571–1580.

Demura, T., Tashiro, G., Horiguchi, G., Kishimoto, N., Kubo, M., Matsuoka, N., Minami, A., Nagata-Hiwatashi, M., Nakamura, K., Okamura, Y., Sassa, N., Suzuki, S., Yazaki, J., Kikuchi, S., & Fukuda, H. (2002). Visualization by comprehensive microarray analysis of gene expression programs during transdifferentiation of mesophyll cells into xylem cells. *Proceedings of the National Academy of Sciences of the United States of America, 99*(24), 15794–15799.

Dervis, S., Mercado-Blanco, J., Erten, L., Valverde-Corredor, A., & Perez-Artes, E. (2010). Verticillium wilt of olive in Turkey: A survey on disease importance, pathogen diversity and susceptibility of relevant olive cultivars. *European Journal of Plant Pathology, 127*, 287–301.

Docampo, D.M., Vázquez, A.A., Laguna, I.G. (1981). *Verticillium dahliae* Kleb. causal de la paralisis del olivo en el area olivera centro oeste Argentina. ?Rev Invest Agropecuarias XVI (2):197-204 (in Spanish).

Donohue, F.M., Morehart, A.L. (1978). Pathogenicity of isolates of *Verticillium* spp. to yellow poplar. Phytopathology news, 12, 68 (abstract).

Douglas, S.M. (2008). Verticillium wilt of ornamental trees and shrubs. The Connecticut Agricultural Experiment Station. www.ct.gov/caes.

Dykstra, M.D. (1997). Verticillium wilt of maple. Pest diagnostic clinic, laboratory services division, University of Guelph, California: 4.

Eckstein, D., Liese, W., & Shigo, A. (1979). Relationship of wood structure to compartmentalization of discolored wood in hybrid poplar. *Canadian Journal of Forest Research, 9*, 205–210.

EFSA PLH Panel (EFSA Panel on Plant Health), (2014). Scientific Opinion on the pest categorisation of *Verticillium dahliae* Kleb. EFSA Journal 2014; 12(12):3928, 54 pp. doi:10.2903/j.efsa.2014.3928.

El-Zik, K. M. (1985). Integrated control of Verticillium wilt of cotton. *Plant Disease, 69*, 1025–1032.

Emechebe, A. M., Leakey, C. L. A., & Banage, W. B. (1974). Verticillium wilt of cacao in Uganda: Wilt induction by mechanical vessel blockage and mode of recovery of diseased plants. *East African Agricultural and Forestry Journal, 39*, 337–343.

Evans, G., & Gleeson, A. C. (1973). Observations on the origin and nature of *Verticillium dahliae* colonizing plant roots. *Australian Journal of Biological Sciences, 26*, 151–161.

Faino, L., Seidl, M. F., Datema, E., van den Berg, G. C. M., Janssen, A., Wittenberg, A. H. J., & Thomma, B. P. H. J. (2015). Single-molecule real-time sequencing combined with optical mapping yields completely finished fungal genome. *MBio, 6*(4), e00936–e00915. doi:10.1128/mBio.00936-15.

Faino, L., Seidl, M. F., Shi-Kunne, X., Pauper, M., van den Berg, G. C. M., Wittenberg, A. H. J., & Thomma, B. P. H. J. (2016). Transposons passively and actively contribute to evolution of the two-speed genome of a fungal pathogen. *Genome Research, 26*(8), 1091–1100.

Fradin, E. F., & Thomma, B. P. H. J. (2006). Physiology and molecular aspects of Verticillium wilt diseases caused by *V. dahliae* and *V. albo-atrum*. *Molecular Plant Pathology, 7*, 71–86.

Frank, S.D., Klingeman, W.E., White, S.A., Fulcher, A.F. (2012). Reduced risk insecticides to control scale insects and protect natural enemies in the production and maintenance of urban landscape plants. Published by the southern nursery IPM working group, Knoxville, TN; in cooperation with the southern region IPM center. Chapter 10, ISBN: 978-0-9854998-1-5.

Fraxigen. (2005). Ash species in Europe: Biological characteristics and practical guidelines for sustainable use. Oxford Forestry Institute, University of Oxford, UK. 128 pp.

Gao, F., Zhou, B., Li, G., Jia, P., Li, H., Zhao, Y., Zhao, P., Xia, G., & Guo, H. (2010). A glutamic acid-rich protein identified in *Verticillium dahliae* from an insertional mutagenesis affects microsclerotial formation and pathogenicity. *PloS One, 5*, e15319.

García-Ruiz, G. M., Trapero, C., Del Río, C., & López-Escudero, F. J. (2014). Evaluation of resistance of Spanish olive cultivars to *Verticillium dahliae* in inoculations conducted in greenhouse. *Phytoparasitica, 42*, 205–212.

Garrett, P.W., Randall, W.K., Shigo, A.L., Shortle, W.C. (1979). Inheritance of compartmentalization of wounds in sweet gum

(Liquidambar styraciflua L.) and eastern cottonwood (Populus deltoides Batrt.). USDA Forest Service research paper NE- 443, 4 pp.

Gayoso, C., Martínez de Illárduya, O., Pomar, F., & Merino de Cáceres, F. (2007). Assessment of real-time PCR as a method for determining the presence of *Verticillium dahliae* in different Solanaceae cultivars. *European Journal of Plant Pathology, 118*, 199–209.

Gleason, M., Hartman, J. (2001). Chapter 60, maple diseases, In: Diseases of woody ornamentals and trees in nurseries, APS Press. P. 236–241.

Gold, J., & Robb, J. (1995). The role of the coating response in Craigella tomatoes infected with *Verticillium dahliae*, races 1 and 2. *Physiology and Molecular Plant Pathology, 47*, 141–157.

Goud, J. K. C., & Termorshuizen, A. J. (2002). Pathogenicity and virulence of the two Dutch VCGs of *Verticillium dahliae* to woody ornamentals. *European Journal of Plant Pathology, 108*, 771–782.

Goud, J. K. C., Termorshuizen, A. J., & van Bruggen, A. H. C. (2011). Verticillium wilt in nursery trees: Damage thresholds, spatial and temporal aspects. *European Journal of Plant Pathology, 131*, 451–465.

Grogan, R. G., Ioannou, N., Schneider, R. W., Sall, M. A., & Kimble, K. A. (1979). Verticillium wilt on resistant tomato cultivars in California: Virulence of isolates from plants and soil and relationship of inoculum density to disease incidence. *Phytopathology, 69*, 1176–1180.

Harrington, T. C., & Cobb Jr., F. W. (1984). Host specialization of three morphological variants of Verticicladiella wageneri. *Phytopathology, 74*, 286–290.

Harris, D.C. (1998). Maple. In: Hiemstra JA, Harris DC, 1998. A compendium of Verticillium wilts in trees: 35-36.

Harris, D. C., & Yang, J. R. (1996). The relationship between the amount of *Verticillium dahliae* in soil and the incidence of strawberry wilt as a basis for disease risk prediction. *Plant Pathology, 45*, 106–114.

Heffer, V. J., & Regan, R. P. (1996). First report of Verticillium wilt caused by *Verticillium dahlia*e of ash trees in Pacific Northwest nurseries. *Plant Disease, 80*, 342.

Heimann, M.F., Worf, G.L. (1997). Maple and other trees disorder: Verticillium wilt. University of Wisconsin-Extension, cooperative Extension, 608-262-8076.

Heinz, R., Lee, S. W., Saparno, A., Nazar, R. N., & Robb, J. (1998). Cyclical systemic colonization in *Verticillium*-infected tomato. *Physiological and Molecular Plant Pathology, 52*, 385–396.

Hibben, C. R. (1959). A new host for *Verticillium albo-atrum* Reinke & berth. *Plant Disease Report, 43*, 1137.

Hiemstra, J. A. (1995a). Recovery of Verticillium-infected ash trees. *Phytoparasitica, 23*, 64–65.

Hiemstra, J.A. (1995b).Verticillium wilt of *Fraxinus excelsior*. PhD Thesis, Wageningen Agricultural University, The Netherlands.

Hiemstra, J.A. (1997). Petioles from infected trees spread Verticillium dahliae. 7th. International Verticillium Symposium, Athens. Book of abstracts, p 47.

Hiemstra, J.A. (1998a). Some general features of Verticillium wilts in trees. In: Hiemstra JA, Harris DC, 1998. A compendium of Verticillium wilts in trees: 5-11.

Hiemstra, J.A. (1998b). Ash. In: Hiemstra JA, Harris DC, 1998. A compendium of Verticillium wilts in trees: 35-36.

Hiemstra, J.A. (2015a). Der schnelle Nachweis von Verticillium. Jahrbuch der Baumpflege 2014: 108–120.

Hiemstra, J.A. (2015b). Control of Verticillium wilt in olive; guide of best practices. Vertigeen project, programme for research and technological development of the European Union, 25 Pages.

Hiemstra, J.A., Harris, D.C. (Eds.) (1998). A compendium of Verticillium wilts in tree species. Wageningen: Centre for Plant Breeding and Reproduction Research (CPRO-DLO)/ West Malling: HRI-East Malling.

Hiemstra, J. A., & Rataj-Guranowska, M. (2000). Vegetative compatibility groups in *Verticillium dahliae* in the Netherlands. In E. C. Tjamos, R. C. Rowe, J. B. Heale, & D. R. Fravel (Eds.), *Advances in Verticillium: Research and disease management* (pp. 100–102). St. Paul: APS Press.

Hiemstra, J.A., Korthals, G.W., Visser, J.H.M., van Dalfsen, P., van der Sluis, B.J., Smits, A.P. (2013). Control of Verticillium in tree nurseries through biological soil disinfestation. 11th International Verticillium Symposium, Georg-August-Universität Göttingen, Germany, 5–8 May 2013. - Göttingen: DPG Spectrum Phytomedizin, 2013 - ISBN 9783941261129 - p. 62.

Himelick, E.B. (1968). Verticillium wilt of trees and shrubs. Illinois natural history survey, Bl-1968, Botany and Plant Pathology Section, Urbana, Illinois. 2 pp.

Hu, R., Qi, G., Kong, Y., Kong, D., Gao, Q., & Zhou, G. (2010). Comprehensive analysis of NAC domain transcription factor gene family in *Populus trichocarpa*. *BMC Plant Biology, 10*, 145.

Inderbitzin, P., Bostock, R. M., Davis, R. M., Usami, T., Platt, H. W., & Subbarao, K. V. (2011). Phylogenetics and taxonomy of the fungal vascular wilt pathogen Verticillium, with the descriptions of five new species. *PloS One, 6*, e28341.

Jiménez-Díaz, R.M., Tjamos, E.C., Cirulli, M. (1998). Verticillium wilt of major tree hosts. In: Hiemstra JA, Harris DC, 1998. A compendium of Verticillium wilts in trees: 13-16.

Jiménez-Díaz, R. M., Mercado-Blanco, J., Olivares-García, C., ColladoRomero, M., Bejarano-Alcázar, J., Rodríguez-Jurado, D., Giménez-Jaime, A., García-Jiménez, J., & Armengol, J. (2006). Genetic and virulence diversity in *Verticillium dahliae* populations infecting artichoke in eastern-central Spain. *Phytopathology, 96*, 288–298.

Jiménez-Díaz, R. M., Olivares-García, C., Landa, B. B., Jiménez-Gasco, M. M., & Navas-Cortés, J. A. (2011). Region-wide analysis of genetic diversity in *Verticillium dahliae* populations infecting olive in southern Spain and agricultural factors influencing the distribution and prevalence of vegetative compatibility groups and pathotypes. *Phytopathology, 101*, 304–315.

Jiménez-Díaz, R. M., Cirulli, M., Bubici, G., Jiménez-Gasco, M., Antoniou, P. P., & Tjamos, E. C. (2012). Verticillium wilt a major threat to olive production: Current status and future prospects for its management. *Plant Disease, 96*(3), 304–329.

Joaquim, T. R., & Rowe, R. C. (1990). Reassessment of vegetative compatibility relationships among strains of *Verticillium dahliae* using nitrate-nonutilizing mutants. *Phytopathology, 80*, 1160–1166.

Karajeh, M. R. (2006). Seed transmission of *Verticillium dahliae* in olive as detected by a highly sensitive nested PCR-based assay. *Phytopathologia Mediterranea, 45*, 15–23.

Karajeh, M. R., & Masoud, S. A. (2006). Molecular detection of *Verticillium dahliae* Kleb. In asymptomatic olive trees. *Journal of Phytopathology, 154*, 496–499.

Karajeh, M. R., & Owais, S. J. (2012). Reaction of selected apple cultivars to wilt pathogen *Verticillium dahliae*. *Plant Protection Science, 48*, 99–104.

Kasson, M. T., Short, D. P. G., O'Neal, E. S., Subbarao, K. V., & Davis, D. D. (2014). Comparative pathogenicity, biocontrol efficacy, and multi-locus sequence typing of *Verticillium nonalfalfae* from the invasive *Ailanthus altissima* and other hosts. *Phytopathology, 104*, 282–292.

Kasson, M. T., O'Neal, E. S., & Davis, D. D. (2015). Expanded host range testing for *Verticillium nonalfalfae*: Potential biocontrol agent against the invasive *Ailanthus altissima*. *Plant Disease, 99*(6), 823–835.

Katan, T. (2000). Vegetative compatibility in populations of Verticillium: an overview. In E. C. Tjamos, R. Rowe, J. B. Heale, & D. R. Fravel (Eds.), *Advances in Verticillium: Research and disease management. Proceeding of 7th International Verticillium Symposium* (pp. 69–86). St. Paul: The American Phytopathological society.

Keykhasaber, M. (2017). Unravelling aspects of spatial and temporal distribution of *Verticillium dahliae* in olive, maple and ash trees and improvement of detection methods. PhD thesis Wageningen University & Research. 163 pp. ISBN: 978–94–6343-014-2. DOI: 10.18174/396604.

Klebahn, H. (1913). Beiträge zur kenntnis der fungi imperfecti. 1. Eine Verticillium-krankheit auf dahlien. *Mycologisches Centralblatt, 3*, 49–66.

Klimes, A., & Dobinson, K. F. (2006). A hydrophobin gene, *VDH1*, is involved in microsclerotial development and spore viability in the plant pathogen *Verticillium dahliae*. *Fungal Genetics and Biology, 43*, 283–294.

Klimes, A., Amyotte, S. G., Grant, S., Kang, S., & Dobinson, K. F. (2008). Microsclerotia development in *Verticillium dahliae*: Regulation and differential expression of the hydrophobin gene *VDH1*. *Fungal Genetics and Biology, 45*, 1525–1532.

Klosterman, S. J., Atallah, Z. K., Vallad, G. E., & Subbarao, K. V. (2009). Diversity, pathogenicity and management of *Verticillium* species. *Annual Review of Phytopathology, 47*, 39–62.

Korolev, N., Katan, J., & Katan, T. (2000). Vegetative compatibility groups of *Verticillium dahliae* in Israel: Their distribution and association with pathogenicity. *Phytopathology, 90*, 529–536.

Korolev, N., Pérez-Artés, E., Bejarano-Alcázar, J., Rodríguez-Jurado, D., Katan, J., Katan, T., & Jiménez-Díaz, R. M. (2001). Comparative study of genetic diversity and pathogenicity among populations of *Verticillium dahliae* from cotton in Spain and Israel. *European Journal of Plant Pathology, 107*, 443–456.

Korolev, N., Pérez-Artés, E., Mercado-Blanco, J., Bejarano-Alcázar, J., Rodríguez-Jurado, D., Jiménez-Díaz, R. M., Katan, T., & Katan, J. (2008). Vegetative compatibility of cotton defoliating *Verticillium dahliae* in Israel and its pathogenicity to various hosts. *European Journal of Plant Pathology, 122*, 603–617.

Krikun, J., & Bernier, C. C. (1987). Infection of several crop species by two isolates of *Verticillium dahliae*. *Canadian Journal of Plant Pathology, 9*, 241–245.

Kubo, M., Udagawa, M., Nishikubo, N., Horiguchi, G., Yamaguchi, M., Ito, J., Mimura, T., Fukuda, H., & Demura, T. (2005). Transcription switches for protoxylem and metaxylem vessel formation. *Genes and Development, 16*, 1855–1860.

Laouane, H., Lazrek, H. B., & Sedra, M. H. (2011). Synthesis and toxicity evaluation of cinnamyl acetate: A new phytotoxin produced by a strain of *Verticillium dahliae* pathogenic on olive tree. *International Journal of Agriculture and Biology, 13*, 444–446.

Leslie, J. F. (1993). Fungal vegetative compatibility. *Annual Review of Phytopathology, 31*, 127–150.

Levin, A. G., Lavee, S., & Tsror, L. L. (2003). Epidemiology and effects of Verticillium wilt on yield of olive trees (cvs. Barnea and Souri) irrigated with saline water in Israel. *Phytoparasitica, 31*, 333–343.

Lockwood, J. L. (1977). Fungistasis in soils. *Biological Reviews, 52*, 1–43.

López-Escudero, F. J., & Blanco-López, M. A. (2001). Effect of a single or double soil solarisation to control Verticillium wilt in established olive orchards in Spain. *Plant Disease, 85*, 489–496.

López-Escudero, F. J., & Blanco-López, M. A. (2005). Recovery of young olive trees from *Verticillium dahliae*. *European Journal of Plant Pathology, 113*, 367–375.

López-Escudero, F. J., & Blanco-López, M. A. (2007). Relationship between the inoculum density of Verticillium dahliae and the progress of Verticillium wilt of olive. *Plant Disease, 91*, 1372–1378.

López-Escudero, F. J., & Mercado-Blanco, J. (2011). Verticillium wilt of olive: A case study to implement an integrated strategy to control a soil-borne pathogen. *Plant and Soil, 344*, 1–50.

López-Escudero, F. J., Del Río, C., Caballero, J. M., & Blanco-López, M. A. (2004). Evaluation of olive cultivars for resistance to *Verticillium dahliae*. *European Journal of Plant Pathology, 110*, 79–85.

López-Escudero, F. J., Mercado-Blanco, J., Roca, J. M., Valverde-Corredor, A., & Blanco-López, M. A. (2010). Verticillium wilt of olive in the Guadalquivir Valley (southern Spain): Relation with some agronomical factors and spread of *Verticilliunm dahliae*. *Phytopathologia Mediterranea, 49*, 70–380.

Mace, M. E., Stipanovic, R. D., & Bell, A. A. (1989). Histochemical localization of desoxyhemigossypol a phytoalexin in *Verticillium dahlia*e infected cotton stems. *New Phytologist, 111*, 229–232.

Malcolm, G. M., Kuldau, G. A., Gugino, B. K., & Jiménez-Gasco, M. M. (2013). Hidden host plant associations of soilborne fungal pathogens: An ecological perspective. *Phytopathology, 103*, 538–544.

Manion, P. D. (2003). Evolution of concepts in forest pathology. *Phytopathology, 93*, 1052–1055.

Mansfield, J. W. (2000). Antimicrobial compounds and resistance: The role of phytoalexins and phytoanticipins. In A. Slusarenko, R. Fraser, & L. van Loon (Eds.), *Mechanisms of resistance to plant diseases* (pp. 325–370). Dordrecht: Kluwer Academic Publishers.

Markakis, E. A., Tjamos, S. E., Antoniou, P. P., Paplomatas, E. J., & Tjamos, E. C. (2009). Symptom development, pathogen isolation and real-time qPCR quantification as factors for evaluating the resistance of olive cultivars to Verticillium pathotypes. *European Journal of Plant Pathology, 124*, 603–611.

Markakis, E. A., Tjamos, S. E., Antoniou, P. P., Roussos, P. A., Paplomatas, E. J., & Tjamos, E. C. (2010). Phenolic

responses of resistant and susceptible olive cultivars induced by defoliating and Nondefoliating Verticillium dahliae Pathotypes. *Plant Disease, 94*, 1156–1162.

Markakis, E. A., Tjamos, S. E., Antoniou, P. P., Paplomatas, E. J., & Tjamos, E. C. (2015). Biological control of Verticillium wilt of olive by Paenibacillus alvei, strain K165. *BioControl, 61*, 293–303.

Martos-Moreno, C., López-Escudero, F. J., & Blanco-López, M. A. (2006). Resistance of olive cultivars to the defoliating pathotype of *Verticillium dahliae*. *Hortscience, 41*, 1313–1316.

Mathre, D. (1986). Occurrence of *Verticillium dahliae* on barley. *Plant Disease, 70*, 981.

Mercado-Blanco, J., Rodríguez-Jurado, D., Perez-Artes, E., & Jiménez-Díaz, R. M. (2001). Detection of the nondefoliating pathotype of *Verticillium dahliae* in infected olive plants by nested PCR. *Plant Pathology, 50*, 609–619.

Mercado-Blanco, J., Rodríguez-Jurado, D., Pérez-Artés, E., & Jiménez Díaz, R. M. (2002). Detection of the defoliating pathotype of *Verticillium dahliae* in infected olive plants by nested PCR. *European Journal of Plant Pathology, 108*, 1–13.

Mercado-Blanco, J., Collado-Romero, M., Parrilla-Araujo, S., Rodríguez-Jurado, D., & Jiménez-Díaz, R. M. (2003). Quantitative monitoring of colonization of olive genotypes by *Verticillium dahlia* pathotypes with real-time polymerase chain reaction. *Physiological and Molecular Plant Pathology, 63*, 91–105.

Mercado-Blanco, J., Rodriguez-Jurado, D., Hervas, A., & Jimenez-Diaz, R. M. (2004). Suppression of Verticillium wilt in olive planting stocks by root-associated fluorescent pseudomonas spp. *Biological Control, 30*, 474–486.

Mol, L. (1995). Formation of microsclerotia of *Verticillium dahliae* on various crops. *Netherlands Journal of Agricultural Science, 43*, 205–215.

Morehart, A. L., & Melchior, G. L. (1982). Influence of water stress on Verticillium wilt of yellow-poplar. *Canadian Journal of Botany, 60*, 201–209.

Navas-Cortés, J. A., Landa, B. B., Mercado-Blanco, J., Trapero-Casas, J. L., Rodríguez-Jurado, D., & Jiménez-Díaz, R. M. (2008). Spatiotemporal analysis of spread of infections by *Verticillium dahliae* pathotypes within a high tree density olive orchard in southern Spain. *Phytopathology, 98*, 167–180.

Nelson, E. B. (1990). Exudate molecules initiating fungal responses to seeds and roots. *Plant and Soil, 129*, 61–73.

Neubauer, C., Vogel, C., & Heitmann, B. (2009). Morphology, vegetative compatibility and pathogenicity of *Verticillium dahliae* isolates from Woody ornamentals in Germany. *Journal of Plant Diseases and Protection, 116*, 109–114.

Nicole, M., Gianinazzi-Pearson, V. (1996). Histology, ultrastructure and molecular cytology of plant-microorganism interactions. Dordrecht: Kluwer. 261 pp.

Ohtani, M., Nishikubo, N., Xu, B., Yamaguchi, M., Mitsuda, N., Goué, N., Shi, F., Ohme-Takagi, M., & Demura, T. (2011). A NAC domain protein family contributing to the regulation of wood formation in poplar. *The Plant Journal, 67*, 499–512.

Papaioannou, I. A., & Typas, M. A. (2015). Barrage formation is independent from heterokaryon incompatibility in *Verticillium dahliae*. *European Journal of Plant Pathology, 141*, 71–82.

Pearce, R. B. (1990). Occurrence of decay-associated xylem suberization in a range of woody species. *European Journal of Forest Pathology, 20*(5), 275–289.

Pegg, G. F., & Brady, B. L. (2002). *Verticillium wilts*. Wallingford: CABI publishing.

Pérez-Artés, E., Garcia-Pedrajas, M., Bejarano-Alcazar, D., & Jime-nez-Díaz, R. M. (2000). Differentiation of cotton-defoliating and nondefoliating pathotypes of *Verticillium dahliae* by RAPD and specific PCR analyses. *European Journal of Plant Pathology, 106*, 507–517.

Pérez-Artés, E., Mercado-Blanco, J., Ruz-Carrillo, A. R., Rodríguez-Jurado, D., & Jiménez-Díaz, R. M. (2005). Detection of the defoliating and nondefoliating pathotypes of *Verticillium dahliae* in artificial and natural soils by nested PCR. *Plant and Soil, 268*, 349–356.

Petrini, O. (1991). Fungal endophytes of tree leaves. Pages 179–187 in. Microbial ecology of leaves. J. H. Andrews and S. S. Hirano, eds. Springer-Verlag, New York.

Piearce, G.D., Gibbs, J.N. (1981). Verticillium wilt of trees and shrubs. Arboricultural leaflet 9. Dept. of the Environment, Forestry Commission, HMSO, London. 8 pp.

Prieto, P., Navarro-Raya, C., Valverde-Corredor, A., Amyotte, S. G., Dobinson, K. F., & Mercado-Blanco, J. (2009). Colonization process of olive tissues by *Verticillium dahliae* and its *in planta* interaction with the biocontrol root endophyte *Pseudomonas fluorescens* PICF7. *Microbial Biotechnology, 2*(4), 499–511.

Pscheidt, J.W., Ocamb, C.M. (2013a). Maple (Acer spp.)-Verticillium wilt. Pacific Northwest plant disease management handbook.

Pscheidt, J.W., Ocamb, C.M. (2013b). Ash (Fraxinus spp.)-Verticillium wilt. Pacific Northwest plant disease management handbook.

Rauyaree, P., Ospina-Giraldo, M. D., Kang, S., Bhat, R. G., Subbarao, K. V., Grant, S. J., & Dobinson, K. F. (2005). Mutations inVMK1, a mitogen-activated protein kinase gene, affect microsclerotia formation and pathogenicity in *Verticillium dahliae*. *Current Genetics, 48*, 109–116.

Resende, M. V. L., Flood, J., & Cooper, R. M. (1995). Effect of methods of inoculation, inoculum density and seedling age at inoculation on the expression of resistance of cocoa (*Theobroma cacao* L.) to *Verticillium dahliae* Kleb. *Plant Pathology, 43*, 104–111.

Reusche, M., Thole, K., Janz, D., Truskina, J., Rindfleisch, S., Drübert, C., Polle, A., Lipka, V., & Teichmann, T. (2012). Verticillium infection triggers VASCULAR-RELATED NAC DOMAIN7–dependent de novo xylem formation and enhances drought tolerance in Arabidopsis. *The Plant Cell, 24*, 3823–3837.

Reusche, M., Truskina, J., Thole, K., Nagel, L., Rindfleisch, S., et al. (2014). Infections with the vascular pathogens Verticillium longisporum and Verticillium dahliae induce distinct disease symptoms and differentially affect drought stress tolerance of *Arabidopsis thaliana*. *Environmental and Experimental Botany, 108*, 23–37.

Riffle, J.W., Peterson, G.W. (1989). Diseases of trees in the Great Plains. Gun. Tech. Rep. RM-129. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station; 1986. 149 p.

Rijkers, A. J. M., Hiemstra, J. A., & Bollen, G. J. (1992). Formation of microsclerotia of *Verticillium dahliae* in petioles of infected ash trees. *Netherlands Journal of Plant Pathology, 98*, 261–264.

Robb, J., Smith, A., & Busch, L. (1982). Wilts caused by Verticillium species. A cytological survey of vascular alterations in leaves. *Canadian Journal of Botany, 60*, 825–837.

Rodríguez-Jurado, D., Blanco-López, M. A., Rappoport, H. F., & Jiménez-Díaz, R. M. (1993). Present status of Verticillium wilt of olive in Andalucía (southern Spain). *EPPO Bulletin, 23*, 513-516.

Rolshausen, P. E., Greve, L. C., Labavitch, J. M., Mahoney, N. E., Molyneux, R. J., & Gubler, W. D. (2008). Pathogenesis of *Eutypa lata* in grapevine: Identification of virulence factors and biochemical characterization of cordon dieback. *Phytopathology, 98*, 222–229.

Ruggieri, G. 1946. Una nuova malatia dell'olivo. L'Italia Agricola, 83, 369–372.

Ryan, D., & Robards, K. (1998). Phenolic compounds in olives. *Analyst, 123*, 31–44.

Schena, L., Nigro, F., & Ippolito, A. (2004). Real-time PCR detection and quantification of soilborne fungal pathogens: The case of *Rosellinia necatrix*, *Phytophthora nicotianae, P. citrophthora*, and *Verticillium dahliae*. *Phytopathologia Mediterranean, 43*, 273–380.

Schnathorst, W. C. (1981). Life cycle and epidemiology of Verticillium. In M. E. Mace, A. A. Bell, & C. H. Beckman (Eds.), *Fungal wilt diseases of plants* (pp. 81–111). New York: Academic Press.

Schnathorst, W. C., & Mathre, D. E. (1966). Host range and differentiation of a severe form of *Verticillium albo-atrum* in cotton. *Phytopathology, 56*, 1155–1161.

Schnathorst, W. C., & Sibbett, G. S. (1971). The relation of strains of *Verticillium albo-atrum* to severity of Verticillium wilt in *Gossypium hirsutum* and *Olea europaea* in California. *Plant Disease Report, 9*, 780–782.

Schreiber, L. R., & Green, R. J. (1963). Effect of root exudates on germination of conidia and microsclerotia of *Verticillium albo-atrum* inhibited by the soil fungistatic principle. *Phytopathology, 53*, 260–264.

Shain, L., & Miller, J. B. (1988). Ethylene production by excised sapwood of clonal eastern cottonwood and the compartmentalization and closure of seasonal wounds. *Phytopathology, 78*, 1261–1265.

Shigo, A. L. (1984). Compartmentalization: A conceptual framework for understanding how tree grow and defend themselves. *Annual Review of Phytopathology, 22*, 189–214.

Shigo, A. L., & Dudzik, K. R. (1985). Response of uninjured cambium to xylem injury. *Wood Science and Technology, 19*, 195–200.

Shigo, A.L., Marx, H.G. (1977). Compartmentalization of decay in trees. Agricultural information Bulletin USDA Forest Service. No.405.73pp.

Sinclair, W. A., & Lyon, H. H. (2005). *Diseases of trees and shrubs* (2nd ed.p. 659). Ithaca: Cornell University Press.

Sinclair, W. A., Smith, K. L., & Larsen, A. O. (1981). Verticillium wilt of maples: Symptoms related to movement of the pathogen in stems. *Etiology, 71*, 340–345.

Smith, K. T. (2006). Compartmentalization today. *Arboricultural Journal, 9*, 173–118.

Smith, K. T., & Shortle, W. C. (1990). IAA oxidase, peroxidase, and barrier zone formation in red maple. *European Journal of Forest Pathology, 20*(4), 241–246.

Smith, I.M., Dunez, J., Lelliott, R.A., Phillips, D.H., Archer, S.A. (1988). European handbook of plant diseases. Blackewell Scientific publications, Oxford etc., 583 S.

Stipes, R.J., Hansen, M.A. (2009). Verticillium wilt of shade trees. Virginia Polytechnic Institute and State University, publication 450-619.

Street, P. F. S., Robb, J., & Ellis, B. E. (1986). Secretion of vascular coating components by xylem parenchyma cells of tomatoes infected with *Verticilliurn albo-atrum*. *Protoplasma, 132*, 1–11.

Termorshuizen, A.J. (1998). Quantification of *Verticillium dahliae* in soil. In: Hiemstra JA, Harris DC, 1998. A compendium of Verticillium wilts in trees: 47.

Tian, L., Xu, J., Zhou, L., & Guo, W. (2014). VdMsb regulates virulence and microsclerotia production in the fungal plant pathogen *Verticillium dahliae*. *Gene, 550*, 238–244.

Tian, L., Wang, Y., Yu, J., Xiong, D., Zhao, H., & Tian, C. (2016). The mitogen-activated protein kinase kinase VdPbs2 of *Verticillium dahliae* regulates Microsclerotia formation, stress response, and plant infection. *Frontiers in Microbiology, 7*, 1532.

Tippett, J.T., Shigo, A.L. (1981). Barrier zone formation: A mechanism of tree defense against vascular pathogens. International Association of Wood Anatomists Bulletin N.S., 2, 163-168.

Tjamos, E. C., & Botseas, D. (1987). Occurrence of *Verticillium dahliae* in leaves of Verticillium-wilted olive trees. *Canadian Journal of Plant Pathology, 9*, 86.

Tjamos, E.C.; Jiménez-Díaz, R.M. (1998). Management of disease. In: Hiemstra JA, Harris DC, 1998. A compendium of Verticillium wilts in trees: 55-57.

Tjamos, E.C., Tsougriani, H. (1990). Formation of Verticillium dahliae microsclerotia in partially disintegrated leaves of Verticillium affected olive trees. 5th International Verticillium Symposium, Book of Abstracts, Leningrad, Soviet Union, p 20.

Tjamos, E. C., Biris, D. A., & Paplomatas, E. J. (1991). Recovery of olive trees from Verticillium wilt after individual application of soil solarization in established olive orchards. *Plant Diseases, 75*, 557–562.

Tosi, L., Zazzerini, A. (1998). An epidemiological study on Verticillium wilt of olive in Central Italy. Olivae, 71, 50–55. (In Spanish).

Townsend, A. M., Schreiber, L. R., Hall, T. J., & Bentz, S. E. (1990). Variation in response of Norway maple cultivars to *Verticillium dahliae*. *Plant Disease, 74*, 44–46.

Tran, V. T., Braus-Stromeyer, S. A., Kusch, H., Reusche, M., Kaever, A., Kühn, A., et al. (2014). Verticillium transcription activator of adhesion *Vta2* suppresses microsclerotia formation and is required for systemic infection of plant roots. *New Phytologist, 202*, 565–581.

Trapero, C., Roca, L. F., Alcantara, E., & Lopez-Escudero, F. J. (2011). Colonization of olive inflorescences by *Verticillium dahliae* and its significance for pathogen spread. *Journal of Phytopathology, 159*, 638–640.

Trapero, C., Serrano, N., Arquero, O., Del Río, C., Trapero, A., & López-Escudero, F. J. (2013). Field resistance to Verticillium wilt in selected olive cultivars grown in two naturally infested soils. *Plant Disease, 97*, 668–674.

Treutter, D. (2006). Significance of flavonoids in plant resistance: A review. *Environmental Chemistry Letters, 4*, 147–157.

Tsror, L. (2011). Epidemiology and control of Verticillium wilt on olive. *Israel Journal of Plant Sciences, 59*, 59–69.

Tzima, A. K., Paplomatas, E. J., Tsitsigiannis, D. I., & Kang, S. (2012). The G protein beta subunit controls virulence and multiple growth- and development related traits in *Verticillium dahliae*. *Fungal Genetic and Biology, 49*, 271–283.

Vallad, G. E., & Subbarao, K. V. (2008). Colonization of resistant and susceptible lettuce cultivars by a green fluorescent protein-tagged isolate of *Verticillium dahliae*. *Phytopathology, 98*, 871–885.

Vallad, G. E., Bhat, R. G., Koike, S. T., Ryder, E. J., & Subbarao, K. V. (2005). Weedborne reservoirs and seed transmission of *Verticillium dahliae* in lettuce. *Plant Disease, 89*, 317–324.

Wang, Y., Tian, L., Xiong, D., Klosterman, S. J., Xiao, S., & Tian, C. (2016). The mitogen-activated protein kinase gene, VdHog1, regulatesosmotic stress response, microsclerotia formation and virulence in *Verticillium dahliae*. *Fungal Genetics and Biology, 88*, 13–23.

Wilhelm, S. (1950). Vertical distribution of *Verticillium albo-atrum* in soils. *Phytopathology, 40*, 776–777.

Wilhelm, S. (1955). Longevity of Verticillium wilt fungus in the laboratory and field. *Phytopathology, 45*, 180–181.

Worf, G. L., Spear, R., & Heimann, S. M. F. (1994). Verticillium induced scorch and chlorosis in ash. *Journal of Environmental Horticulture, 12*, 124–130.

Xiao, C. L., & Subbarao, K. V. (1998). Relationship between *Verticillium dahliae* inoculum density and wilt incidence, severity, and growth of cauliflower. *Phytopathology, 88*(10), 1108–1115.

Xiong, D., Wang, Y., Ma, J., Klosterman, S. J., Xiao, S., & Tian, C. (2014). Deep mRNA sequencing reveals stage-specific transcriptome alterations during microsclerotia development in the smoke tree vascular wilt pathogen, *Verticillium dahliae*. *BMC Genomics, 15*(1), 324.

Xiong, D., Wang, Y., Tian, L., & Tian, C. (2016). MADS-box transcription factor VdMcm1 regulates Conidiation, Microsclerotia formation, pathogenicity, and secondary metabolism of *Verticillium dahliae*. *Frontiers in Microbiology, 7*, 1192.

Yamaguchi, M., Goué, N., Igarashi, H., Ohtani, M., Nakano, Y., Mortimer, J. C., Nishikubo, N., Kubo, M., Katayama, Y., Kakegawa, K., Dupree, P., & Demura, T. (2010). VASCULAR-RELATED NACDOMAIN6 and VASCULAR-RELATED NAC-DOMAIN7 effectively induce transdifferentiation into xylem vessel elements under control of an induction system. *Plant Physiology, 153*, 906–914.

Zhao, Y.-L., Zhou, T.-T., & Guo, H.-S. (2016). Hyphopodium-specific VdNoxB/VdPls1-dependent ROS-Ca2+ signaling is required for plant infection by *Verticillium dahliae*. *PLoS Pathogens, 12*(7), e1005793. doi:10.1371/journal.ppat.1005793.