

Spatial pattern and disease severity of charcoal canker in Hyrcanian forests, north of Iran

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ABSTRACT: This research investigated the population density of infected trees with charcoal canker disease and the interaction across the severity of the disease on *Quercus castaneifolia* in Hyrcanian forests, north of Iran. We used two-dimensional spatial analysis tools with data gathered in point-centred quarter format in 2013. We investigated the co-occurrence of the severity of the disease symptoms using the paired quadrat covariance analysis. The results showed that nearly 54% of the oak trees were affected by the disease so that the dead trees and high disease severity were strongly clustered at broader scales (800 m), the trees with only limited exudates and signs of the activity of *Biscogniauxia mediterranea* (De Not.) Kuntze occurred in the clusters away from the dead trees. The crown mortality and beetle-infested trees co-occurred with mortality and were strongly correlated. Also, the findings of this research showed that the density of trees per hectare was one of the influential factors on the damage severity of the charcoal canker disease, so that reducing the density could increase the damage severity of the disease.

Keywords: *Biscogniauxia mediterranea*; oak decline; point-centred quarter method; *Quercus castaneifolia*

In the Mediterranean area, the main risk seems to be related to a potential reduction of precipitation and the consequences of this are that trees become more susceptible to opportunistic organisms such as fungal pathogens (BOYER 1995). Among them, *Biscogniauxia mediterranea* (De Not.) Kuntze is well known as the causative agent of charcoal canker in the oak and it is a serious problem in the oak forest (COLLADO et al. 2001; MAZZAGLIA et al. 2001; NUGENT et al. 2005; CAPRETTI, BATTISTI 2007). *B. mediterranea* is a common cause of the charcoal canker diseases and it is involved in the massive decline of oaks (*Quercus* spp.) and other trees in the forests of Europe, North America, Africa, New Zealand, and Asia (COLLADO et al. 2001; PORTA et al. 2008; MIRABOLFATHY et al. 2011). The spatial analysis of the plant diseases has been used to describe the spatial pattern proposing improvements in management and control methods (PETHYBRIDGE et al. 2005), identifying the factors that influence the spread of the plant diseases (ROUMAGNAC et al. 2004) or improving the sampling strategy

(FRANKLIN et al. 1995). In this regard, KELLY et al. (2008) investigated the spatial pattern dynamics of the oak mortality affected by sudden oak death and the results of their research showed that in the early process of the disease invasion, dead trees were strongly clustered at smaller scales and after three years this clustering was less pronounced. GIENCKE et al. (2014) implemented the bivariate point pattern analysis to examine the spatial patterns of beech thicket formation and the beech bark disease spread. Their results indicated that the disease severity of beech saplings was highest in close proximity to highly cankered canopy beech trees. In 2011, the charcoal canker was first reported on *Q. castaneifolia* trees that represent a new host of *B. mediterranea* in the Golestan forest in the north of Iran (MIRABOLFATHY et al. 2011). A few years after the first report, nevertheless, we do not know yet the rate of oak mortality and density of tree infections in the oak forests. Thus, the overall objectives of this study are as follows: (1) to quantify the population densities of trees with different degrees of disease

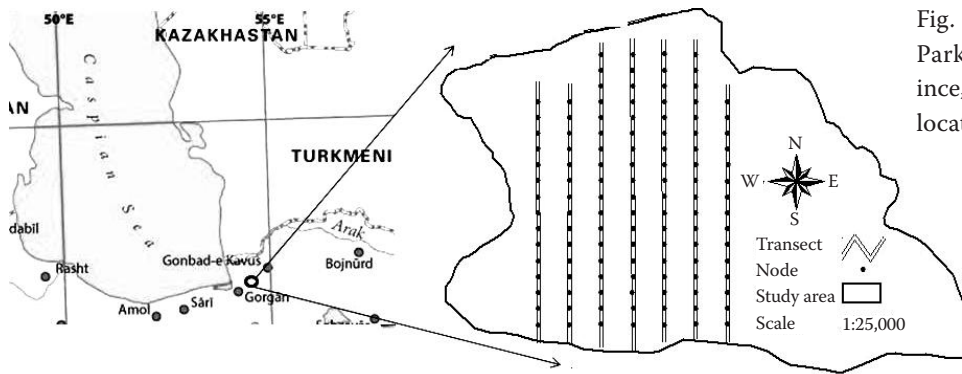


Fig. 1. Study area of the Forest Park Qoruq in Golestan province, showing transect and node locations

symptoms (disease categories) across the study area, (2) to determine the severity of co-occurrence of the crown and stem symptoms and (3) to determine the extent of clustering of the disease categories.

MATERIALS AND METHODS

Study area. The study area is located at the Forest Park Qoruq (FPQ) in the Golestan province of Iran input the map (54°43'N, 36°52'E; 105–600 m a.s.l.). Mean annual temperature from 1982 to 2012 was 17.2°C and mean annual precipitation was 700.81 mm, with wetter months occurring between October and June, and a dry season from July to September. Average monthly temperature ranges from 7.5°C in February to 27.3°C in August (Golestan Regional Water Authority 2013). The area is on the unpaved and uneven terrain with slope (0–50%). The soils are deep, moderately well drained and stone-free with silty clay loam and clay loam textures. The forests in this region are dominated by oak (*Q. castaneifolia*), hornbeam (*Carpinus betulus*), and is co-dominated by Persian ironwood (*Parritia persica*) and Caucasian elm (*Zelkova carpinifolia*).

Data collection. We collected data using the point-centred quarter (PCQ) method in FPQ in the summer of 2013. Therefore, parallel linear transects were established 200 m apart, along the north-south line of the study area (Fig. 1), which served as the anchor point for seven transects. Sampling centre points (nodes) were located at the distance of 100 m from each other in each transect so that to reduce edge effects, the nodes that were situated at least at a distance of 100 meters from the forest boundary were selected. The transects ranged from 4 to 22 nodes in length. The location of each centre point was recorded with a global positioning system (GPS) device. At each node, cardinal compass directions defined four quadrants. The closest oak tree (> 7.5 cm DBH) in each quadrant was labelled

with a number, crown cover mortality percent, diameter at breast height (DBH) and disease status were determined. The distance from the node centre and azimuth of each tree were recorded. Only the trees that were within a maximum distance of 50 m from a node centre were recorded (COTTAM, CURTIS 1956; KELLY et al. 2008).

Symptom severity evaluation of charcoal canker. The first symptoms of charcoal disease that may be observed are discolorations and browning of the leaves, resulting in the drying of foliage, viscous liquid exudates and lengthwise bark cracks. Subsequently, the outer bark begins to slough off in the area of the infection, and pieces of bark can be seen at the base of the tree (RAGAZZI et al. 2012). This bark loss exposes the first sign of the fungus, which is a brownish fungal stroma where conidia (or asexual spores) of the pathogen are produced. This area may be several inches to several feet long on limbs and trunks. In all infected trees were dead and exhibited symptoms of charcoal disease with carbonaceous, perithecial stromata erupting from the bark on stems (MIRABOLFATHY et al. 2011). Therefore, visual disease symptoms for each of the trees at each node were evaluated and recorded. Charcoal canker disease severity on individual trees was ranked from 1 (no disease) to 5 (dead) based on stem health and crown loss. Includes of 1: asymptomatic; 2: only limited exudates and signs of the fungal activity of *B. mediterranea* (discoloration and browning of leaves, drying of foliage, viscous liquid exudates observed on trunks) were found; 3: exudates and signs of the fungal activity plus longhorn beetles (Coleoptera, Cerambycidae); 4: exudates and signs of the fungal activity plus beetles and deep canker (caused by the beetles and an associated fungal pathogen) and wood-eating beetle activity have increased the range; 5: dead (McPHERSON et al. 2005; KELLY et al., 2008, MIRABOLFATHY et al. 2011). We analysed the data from the PCQ method in two ways: we estimated the

population density of trees across the study area, and then we examined the spatial pattern of the crown mortality and associated symptoms.

Population density estimation. The population density of the oak trees at each of the five different symptom levels was estimated from the PCQ data in three steps (COTTAM, CURTIS 1956). First, the mean of all node centre-to-plant distances (the mean distance from the centre node to all four sample trees) was calculated for all sample points, regardless of the symptom, using the Equation 1:

$$\bar{d} = \frac{1}{n} \sum_{i=1}^n d_i \quad (1)$$

where:

n – overall sample size,

d_i – point-to-plant distance.

The squared value of this mean distance gives the mean grid area occupied by one tree. Second, the population density of the oak trees at all symptom levels can be estimated as the ratio between a unit area (1 ha) and the mean grid area. The overall population density of the oak trees ρ (tree per ha) was calculated as Equation 2:

$$\rho = \frac{10,000 \text{ m}^2/\text{ha}}{(\bar{d}) \times \text{m}^2/\text{tree}} \quad (2)$$

where:

\bar{d} – mean point-to-plant distance.

given the units of mean distance and area are meters and square meters, respectively.

Finally, the population density of the oak trees at a particular symptom level is calculated as the product of the overall population density ρ and the percentage of the trees at that symptom level. Thus, the population density of symptom level i is denoted by ρ_i , and $\rho_i = \rho \times \rho_i$, where Equation 3:

$$\rho_i = n_i/n \times 100\% \quad (3)$$

where:

n_i , n – sample size of trees at symptom level i ($i = 1-5$) and the overall sample size, respectively.

Spatial pattern analysis of mortality. The quadrat variance methods such as paired quadrat variances (PQV), “two-term local quadrat variance” (TTLQV) as well as 3TLQV and others, are used predominantly for one-dimensional regularly-spaced transect data. In order to apply the quadrat variance methods to the transect data for detection of the scale of disease clustering, we adjusted the PCQ transect data in two steps: first, we treated the nodes along each transect as the centres of the quadrats, and the width of quadrats is determined as the maximum point-to-plant distance. Second,

the quarter-wise observations were converted to quadrat-wise observations by replacing the four-quarter records with the mean or maximum value for each node. We applied two quadrat variance analysis methods including two-term local quadrat variances (TTLQV) and three-term local quadrat variance (3TLQV), to the adjusted transects data. The two methods were defined in the Equations (4–5) (DALE, MACISAAC 1989):

$$3\text{TTLQV}(r) = \frac{\sum_{i=1}^{n+1-3r} (\sum_{j=i}^{i+r-1} x_j - 2 \sum_{j=i+r}^{i+2r-1} x_j + \sum_{j=i+2r}^{i+3r-1} x_j)^2}{8r(n+1-3r)} \quad (4)$$

$$\text{TTLQV}(r) = \frac{\sum_{i=1}^{n+1-2r} (\sum_{j=1}^{i+r-1} x_j - \sum_{j=i+r}^{i+2r-1} x_j)^2}{2r(n+1-2r)} \quad (5)$$

where:

x_j – observation on the j -th quadrat,

n – total number of quadrats,

r – scale parameter, which is the integer-times of the quadrat size.

Paired quadrat variances are mathematically identical to a one-dimensional variogram analysis, detecting the scale of the pattern under investigation (BRYANT et al. 2005). Just as the variogram can be extended to a cross-variogram for covariance analysis, the PQV can also be extended to a paired quadrat covariance (PQC) in order to study the co-variance among oak trees at different symptom severity. To do this, the PCQ transect data were adjusted as follows: for each symptom severity level, the quadrat value at each node was recorded as 1 or 0 (presence or absence of a symptom). This generated five binary transects of quadrats, each of which corresponded to one symptom. The PQC is similarly defined in the Equation 6:

$$V_{\text{PCQ}}(i,j,r) = \frac{\sum_{k=1}^{n-r} (x_{i,k} - x_{i,k+r})(x_{j,k} - x_{j,k+r})}{2(n-r)} \quad (6)$$

where:

$x_{i,k}$, $x_{j,k}$ – presence/absence values on the k -th quadrat for symptom severity level i and j , respectively,

n – total number of quadrats,

r – scale parameter, which is the integer-times of the quadrat size.

We used the formulae above to analyse data along the longest transect in our study area.

RESULTS

Population density estimation

In total, data from 400 trees with an average diameter of 52.55 cm with a diameter range of 15–140 cm were registered in seven transects with

Table 1. Tree density and mean disease state (oaks·ha⁻¹) for seven transects that have four nodes at least. Mean disease state is the mean of the disease rating scale (1–5) recorded for the oaks in each transect

Transect	No. of nodes	Mean (trees·ha ⁻¹)	SD	Minimum–maximum	Mean disease state ± (SD)
1	14	17.55	12.63	1.4–53	2.5 ± (1.1) ^{a*}
2	14	18.61	10.32	1–38.8	1.9 ± (1.4) ^b
3	17	17.38	9.96	3.7–49.48	2.3 ± (1.4) ^{ab}
4	22	19.65	11.53	1.7–47	2.2 ± (1.7) ^b
5	16	17.28	12.75	1–56	2.0 ± (1.4) ^b
6	15	19.11	11.43	2.26–53.5	2.0 ± (1.4) ^b
7	4	21.40	14.47	3–52	1.8 ± (1.4) ^b

*significant at $P = 0.01$

102 nodes. The mean node centre-to-plant distance (for all oaks) is $17.8 \pm (11.8)$ m, and the population density of all oak trees in the study area is 32.2 trees per hectare (Table 1).

The population density of the trees at different symptom levels is reported in Table 2. The results showed that the frequency and density (tree·ha⁻¹) of the asymptomatic trees were much higher than in the other trees and the trees with symptom levels 4 (exudates and sign of the fungal activity plus beetles plus deep canker caused by the beetles and an associated fungal pathogen and wood-eating beetle activity had an increased range) were the lowest. The results also showed that trees with higher disease severity accounted for the lowest percentage of oak trees in the study area allocated (Table 2). Nevertheless, the results showed that 54 percent of the trees were infected, so that 17.3% of these trees could not recover his health and 13.0 percent was dead (Table 2).

Spatial pattern analysis of mortality

The results from both TTLQV and 3TLQV methods of transect analysis are very similar. The 3TLQV and TTLQV plots show strong clustering of the crown mortality within the distance of 800 m, as shown in Fig. 2. 3TLQV and TTLQV plots of both mean symptom severity number of 5 (trees from all four quadrants at a node had dead crowns) and maximum of

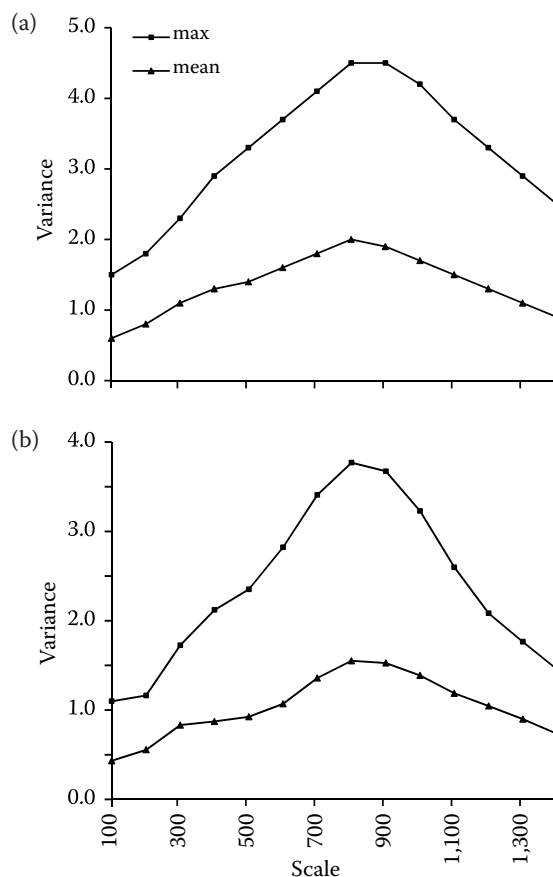


Fig. 2. Results of the transect analysis for clustering of overstorey mortality: (a) two-term local quadrat variance (TTQLV) analysis of the longest transect using the mean symptom severity from each node and the maximum symptom severity from each node (b) three-term local quadrat variance (3TQLV) analysis of the longest transect using the mean symptom severity from each node

5 (at least one of the trees in the four quadrants at a node had a dead crown) showed strong clustering at 800 m, or in other words (Fig. 2), distinguished clusters with large open spaces between overstorey mortality clusters are available.

We compared co-occurrence in space for trees with the symptoms: asymptomatic and crown mortality (1 and 5), exudates and crown mortality (2 and 5), exudates plus beetles and crown mortality (3 and 5), deep canker plus trunk bark isolated and crown mortality (4 and 5) by using the PQC analysis (Fig. 3a). Besides this, co-occurrence in space for trees with

Table 2. The frequency, percentage and population density of trees with different symptom levels

	Symptom level				
	asymptomatic	with exudates	with beetles	exudates and beetle has increased	dead
Frequency	166	102	50	38	56
Absolute frequency	76.3	57.7	35	26	35
Relative density	40.2	24.65	12.2	9.35	13.6
Absolute density (tree·ha ⁻¹)	12.9	7.9	3.9	3	4.4

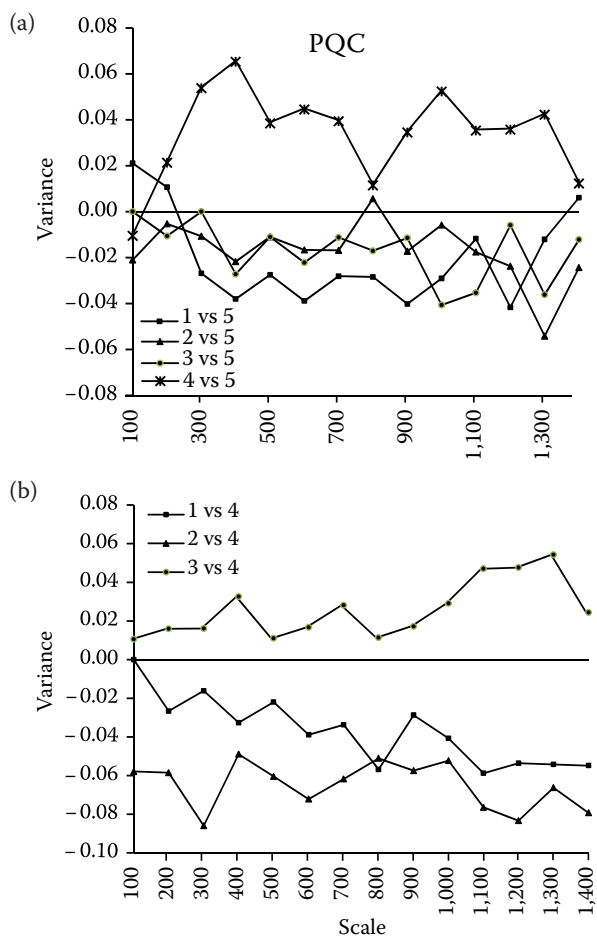


Fig. 3. Results of PQC: (a) data comparing nodes with exudates and mortality, symptom level 5, and symptoms 4 and 5, (b) data comparing nodes with symptom level 1 and 4, symptom level 2 and 4, and symptom level 3 and 4

the symptoms: asymptomatic and deep canker plus trunk bark isolated (1 and 4), limited exudates and deep canker plus trunk bark isolated (2 and 4), exudates plus beetles and deep canker plus trunk bark isolated, (3 and 4), by using the PQC analysis (Fig. 3b), and we review the results symptom-by-symptom. The strength of the association with mortality is in relation to the “0” variance x-axis, where a strong association is positive and a weak association is negative. For both analyses by PQC, we see an increasingly strong association of the symptom state with mortality as the tree transition progresses from exudates to beetle infestation, to deep canker plus trunk bark isolated. Exudates: earlier in the epidemic of the charcoal disease, exudates did not co-occur with trees which had deep canker plus trunk bark isolated and dead trees. But at 800 m exudates occurred close to the dead trees, indicating perhaps a new cohort of infected trees in the forest. The relationship of the disease severity was even more pronounced: crown mortality and deep canker plus trunk bark isolated (4 and 5), and exudates, canker development and deep

canker plus trunk bark isolated (3 and 4), occurred in close. Beetles: beetle-infested trees did not co-occur with mortality, but were associated with trees that had deep canker plus trunk bark isolated in clusters at 400, 700 and 1,100–1,300 m. The trees which had deep canker plus trunk bark isolated displayed much spatial patterning, and were found throughout the forest and were strongly correlated with dead trees across all scales.

DISCUSSION AND CONCLUSIONS

This work provides an estimate of the density of symptomatic trees at different severity of disease across a forested landscape affected by the charcoal canker. The results showed that 54% of the trees in the surveyed area were infected so that 17.3% of these trees could not recover their health and 13.0% were dead. According to these results, the Forest Park Qoruq can be one of the charcoal canker foci in the hardwood forests of Iran introduced. Because less than two years have passed since identifying the pathogen of charcoal canker in the forest where the severity of the damage has increased. The non-uniform distribution of the disease severity may indicate the factors of the local destruction such as humans (soil compaction, logging and thinning of canopy) (ANDERSON et al. 2004) and pests (*Lymantria dispar*) and abiotic factors (drought) with charcoal canker (DESPREZ-LOUSTAU et al. 2006; HAJIZADEH et al. 2013). Meteorological data, an average increase in temperature (0.6°C) and a decrease in mean monthly rainfall (11.5 mm) and drought in the hot months of the year (August) in the past 5 years, the 30-year average shows that these can be the factors that increase the spread of charcoal canker disease in the region. However, the abundance and density of healthy trees and trees with lower disease severity indicate the start of the first wave of the disease in the study area in recent years (MIRABOLFATHY et al. 2011). In addition, the results showed that the severity of charcoal canker in less than seven transects was lesser but the highest density of trees per hectare was observed in seven transects instead. Also in transect one with the greatest degree of damage, tree density on the surface of transects was too low. Therefore, based on these results it can be concluded that the density of trees per hectare is one of the factors affecting the disease severity and the negative correlation of disease severity with the density of trees per hectare. The results of this study with the results of MCBRIDE, APPEL (2009) showed that with the mass and density of trees in natural forests and

plantations, the disease severity of the oak becomes higher and the spread of charcoal canker increases. So the management and conservation of the forest tree density per hectare should be maintained at an appropriate level until the outbreak of charcoal canker is slower. Also our results in accordance with KELLY et al. (2008) indicate that the disease severity of the charcoal canker in the oak forest is not randomly distributed. Therefore, based on the results obtained, we can acknowledge that in the earlier epidemic, the dead trees are strongly clustered at large scales (800 m) (GIENCKE et al. 2014). The results of the relationship between the severity of the disease symptom classifications of the dead oak trees using the paired quadrat covariance (PQC) showed that the trees with great damage caused by the disease have a strong association with dead trees so that healthy trees or with low severity of the disease were far from dead trees (KOT et al. 1996; HAVEL et al. 2002; KELLY et al. 2008; MEENTEMEYER et al. 2008). The close relationship with the severity of the tree damage and the dead trees were perhaps indicative of the prevalence of the disease in a specific location which may be due to differences in microclimatic conditions and the tree resistance against the disease in each location (MEENTEMEYER et al. 2008). Similar results have been reported in other plant ecosystems exhibiting passive pathogen dispersal, whereby the dispersion slope favours new infections closer to the source of the inoculum (CARLSSON, THRALL 2002; HASTING et al. 2005). However, the interpretation of the role of fungi and insects in relation to other biotic and abiotic factors in oak death is difficult because many factors and interventions are involved in the oak mortality. Many of these organisms can cause physiological abnormalities that are not easily visible, but they can induce handicapping trees and increase their susceptibility to infection with other parasites. These findings support the forest decline models developed by MANION in 1991 and show that it is important to take into account the role of opportunistic pathogens in tree mortality processes. According to the results of this study, the mean level of 800 m clusters of trees with the crowns dried to producing can be a good guide for identifying and mapping the oak mortality. The strong correlation between the severities of great damage with the dead trees may indicate the focus of infection, providing the optimal conditions for the fungal growth and sporulation of *B. mediterranea*. Large inoculums to dead trees provide the place and trees near the centre will be more contaminated. The evidence presented in this study, new insights into the highly complex spatial distribution and co-occurrence se-

verity of the charcoal canker disease in the oak forest. Because we have shown that the trees with great damage severity are closely related to each other, these findings might be useful in the control of the charcoal canker disease and restoration of forests affected by the pathogen, which the oak declining the cluster occurs. Specifically, these findings confirmed the results of the research which showed that the infected trees with great damage severity of the charcoal canker disease should be cut and burned to the ground nearest the trunk and if possible chumps should also be removed and burned from the increased pathogen inoculation in order to prevent the spread of the disease. Therefore, it is essential to identify the foci of the infection and they should be managed to prevent the outbreak.

References

- Anderson P.K., Cunningham A.A., Patel N.G., Morales F.J., Epstein P.R., Daszak P. (2004): Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends in Ecology & Evolution*, 19: 535–544.
- Boyer J.S. (1995): Biochemical and biophysical aspects of water deficits and the predisposition to disease. *Annual Review of Phytopathology*, 33: 251–274.
- Bryant D.M., Ducey M.J., Innes J.C., Lee T.D., Eckert R.T., Zarin D.J. (2005): Forest community analysis and the point-centered quarter method. *Planet Ecology*, 175: 193–203.
- Capretti P., Battisti A. (2007): Water stress and insect defoliation promote the colonization of *Quercus cerris* by the fungus *Biscogniauxia mediterranea*. *Forest Pathology*, 37: 129–135.
- Carlsson U., Thrall P.H. (2002): The spatial distribution of plant populations, disease dynamics and evolution of resistance. *Oikos*, 97: 97–110.
- Collado J., Platas G., Pelaez F. (2001): Identification of an endophytic *Nudulisporium* sp. from *Quercus ilex* in central Spain as the anamorph of *Biscogniauxia mediterranea* by rDNA sequence analysis and effect of different ecological factors on distribution of the fungus. *Mycologia*, 93: 875–886.
- Cottam G., Curtis J.T. (1956): The use of distance measures in phytosociological sampling. *Ecology*, 37: 451–460.
- Dale M.R.T., Macisaac D.A. (1989): New methods for the analysis of spatial pattern in vegetation. *Journal of Ecology*, 77: 78–91.
- Desprez-Loustau M., Marcais B., Nageleisen L.M., Piou D., Vannini A. (2006): Interactive effects of drought and pathogens in forest trees. *Annals of Forest Science*, 63: 597–612.
- Franklin S.B., Gibson D.J., Robertson R.A., Pohlmann J.T., Fralish J.S. (1995): Parallel analysis: a method for determining significant principal components. *Journal of Vegetation Science*, 6: 1–8.

- Giencke L.M., Dovciak M., Mountrakis G., Cale J.A., Mitchell M.J. (2014): Beech bark disease: spatial patterns of thicket formation and disease spread in an aftermath forest in the northeastern United States. *Canadian Journal of Forest Research*, 44: 1042–1050.
- Hajizadeh G., Kavosi M.R., Jalilvand H. (2013): Evolution of oviposition behavior in gypsy moth (*Lymantria dispar*) in Hyrcanian forests, North of Iran. *Biodiversity*, 2: 101–105.
- Hasting A., Cuddington K., Davies K.F., Dugaw C., Elmendorf S., Freestone A., Harrison S., Holland M., Lambrinos J., Malvadkar U., Melbourne B., Moore K., Taylor C., Thomson D. (2005): The spatial spread of invasions: new developments in theory and practice. *Ecology Letters*, 8: 91–101.
- Havel J., Shurin J., Jones J. (2002): Estimating dispersal from patterns of spread: spatial and local control of lake invasions. *Ecology*, 83: 3306–3318.
- Kelly M., Liu D., McPherson B., Wood D., Standiford R. (2008): Spatial pattern dynamics of oak mortality and associated disease symptoms in a California hardwood forest affected by sudden oak death. *Journal Forest Research*, 13: 312–319.
- Kot M., Lewis M.A., Driessche P. (1996): Dispersal data and the spread of invading organisms. *Ecology*, 77: 2027–2042.
- Manion P.D. (1991): *Tree Disease Concepts*. New Jersey, Prentice Hall: 402.
- Mazzaglia A., Anselmi N., Gasbarri A., Vannini A. (2001): Development of polymerase chain reaction (PCR) assay for the specific detection of *Biscogniauxia mediterranea* living as an endophyte in oak tissues. *Mycological Research*, 105: 952–956.
- McBride S., Appel D. (2009): Understanding, recognizing and keeping *Hypoxylon* Canker of oaks at bay. Available at <http://plantclinic.tamu.edu/files/2010/09/Hypoxylon-Fact-Sheet3.pdf>
- McPherson A., Mori R., Wood L., Storer J., Svihra P., Kelly M., Standiford R.B. (2005): Sudden oak death in California: disease progression in oaks and tanoaks. *Forest Ecology Management*, 213: 71–89.
- Meentemeyer R.K., Anacker B.L., Mark W., Rizzo D.M. (2008): Early detection of emerging forest disease using dispersal estimation and ecological niche modeling. *Ecological Applications*, 18: 377–390.
- Mirabolfathy M., Groenewald J.Z., Crous P.W. (2011): The occurrence of Charcoal canker caused by *Biscogniauxia mediterranea* on chestnut-leaved oak (*Quercus castaneifolia*) in the Golestan Forests, North of Iran. *Plant Disease*, 95: 876–876.
- Ragazzi A., Ginetti B., Moricca S. (2012): First Report of *Biscogniauxia mediterranea* on English Ash in Italy. *Plant Disease*, 96: 1694.

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