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SHORT COMMUNICATION

Drought heightens severity of diseases caused by *Botryosphaeria dothidea* and *Cryptostroma corticale* and needs to be factored in to properly assess pathogenicity or fulfill Koch's postulates

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Abstract
Climate change is driving the emergence of novel tree diseases at the global scale, requiring new approaches for the formal confirmation of the pathogenicity of novel pathogens on novel hosts. At the same time, predictive models need to account for the possible effect of environmental changes and of abiotic stressors on disease severity for all diseases. By wound-inoculating *Botryosphaeria dothidea* on potted California coast live oaks and *Cryptostroma corticale* on potted silver maples, simultaneously in well-watered and in water-deprived conditions, I show that drought conditions increase the severity of disease symptoms. I also show that, by including a water-stressed treatment, I can formally prove pathogenicity and fulfill Koch's postulates for putative pathogens that could not be confirmed in the absence of the stressor. Additionally, I show that the inclusion of data obtained in water stress conditions increases the differentiation between symptoms caused by fungal infection vs. symptoms caused by wound trauma, thus reducing the possible effect of outliers, a significant problem affecting many trials for fulfilling Koch's postulates conducted with a limited number of replicates. The availability of comparable datasets in the presence and the absence of an abiotic stressor allows for the calculation of an Environmental Disease Component Index. Positive values of the index indicate a significant role of environmental change in disease progression and identify those pathogens that must be modeled factoring in climatic stressors. I suggest that this index may be extremely valuable for identifying pathogens likely to become emergent as climate changes.

Keywords Climate change · Forest disease · Fungal pathogen · Latent pathogen

Climate change is driving the emergence of novel tree diseases at the global scale (Allen et al. 2015). While there is limited definitive evidence that climate itself may be directly causing large scale plant mortality (Hember et al. 2017), a growing number of reports links unprecedented plant and animal mortality outbreaks to the exacerbation of diseases once thought to be minor or to the emergence of new diseases caused by previously unreported "host x pathogen" combinations (Anderson et al. 2004). In a large number of cases, novel climate-driven mortality seems to be determined by emerging fungal diseases (Nnadi and Carter 2021), with several examples from forest ecosystems (Sturrock et al. 2011). Warmer temperatures and the intensification of the rapid and frequent alternation between very dry and very wet periods have been cited as factors potentially favoring fungal infections, and not just in plants (Coakley et al. 1999; Rosenzweig et al. 2001; Garcia-Solache and Casadevall 2010).

Milder winters may allow for longer infectious or disease-causing phases in the life cycle of pathogenic fungi (Kubiak et al. 2017) or may favor the regional movement and establishment of pathogens better adapted to warmer climates (Garrett et al. 2021; see Jeger 2022). Likewise, intense wet periods may elevate relative air humidity and plant surface wetness, thus favoring fungal sporulation and plant infection by fungal spores (see Pautasso et al. 2012). Conversely, longer and more intense droughts may favor disease progression in water-starved plants that were infected in the previous wet period (Haavik et al. 2015;

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Marques et al. 2022). Recent studies that compare disease progression at different temperatures are finding that disease caused by thermophilic or mesophilic fungi are exacerbated at higher temperatures (Almeida et al. 2019). In some cases, of course, the opposite may also be true, and individual diseases caused by psychrophilic fungi may be declining, due to increasing temperatures (Jeger 2022).

At least one recent study has shown that the magnitude in increase of disease severity as temperature increases is significantly greater than the magnitude of the increase in fungal growth rate (Martino et al. 2024). This incongruence is supposedly determined by the additional effect that increasing temperatures may have not on the fungus but on the plant host (Hennon et al. 2020). The multitude of effects of rising temperature on plant physiology has been studied (Adams et al. 2017). However, two less understood side effects of climate-induced modified plant response to infection may actually be to increase the virulence of drought-associated pathogens (Desprez-Loustau et al. 2006) or to activate the pathogenic phase of latent pathogens (Schoeneweiss 1978). While the end result of these two side effects may be strikingly similar, i.e. the emergence of novel diseases or the increase in the severity of symptoms of a disease that was once irrelevant, the mechanisms leading to disease may be very different. In the case of drought-induced pathogens, disease severity may be driven mostly by the reduced ability of the host to respond to infection (Nnejat and Mantri 2017). In the case of latent pathogens, it is the novel environment in the plant host, often reduced water pressure and increased presence of the gaseous phase in the plant tissue, that directly triggers an endophyte to become a pathogen (Hendry et al. 2002). It should also be clearly stated that the three mechanisms (rising temperature, compromised immune response and modified plant environment) are not mutually exclusive, thus complicating our understanding of disease that are emerging in response to climate changes and resulting in complex feedbacks and non-linear relationships.

In this study, I use the drought-induced fungal pathogen *Cryptostroma corticale* (Cc) and the latent pathogen *Botryosphaeria dothidea* (Bd) to highlight two considerable problems that arise when studying climate-driven diseases. The first problem consists in the inability to identify a pathogen as such in the absence of the appropriate environmental stressor. This is relevant, and has serious regulatory implications (Slippers and Wingfield 2007) because most pathogenicity tests performed in order to fulfill Koch's postulates are conducted in the absence of environmental stressors. The second problem lies in the inability to predict the true virulence of a pathogen unless the environmental stressor is applied or is present during a study. These two problems arise when the pathogen in question is strongly affected by climate or by environmental changes (e.g. latent pathogens), and may be less marked for aggressive primary pathogens. Based on the data I present below, I will suggest that from now on and for fungi known to be exacerbated by physiological stress, pathogenicity tests should be performed simultaneously in the absence and in the presence of a key abiotic stressor. Finally, I propose a simple Environment Disease Component Index that determines the magnitude of the effect that changing environmental conditions may have on disease severity. This index could be presented when performing pathogenicity tests under different environmental conditions and would help to identify pathogens that may be emergent, even before they actually emerge.

Botryosphaeria dothidea and *C. corticale* were chosen because they are both emergent and the diseases they cause have been reported to occur worldwide (Brooks et al. 2023; Marsberg et al. 2017; Muller et al. 2023). Bd is a latent pathogen known to cause stem or branch canker and dieback (Slippers and Wingfield 2007), a disease exacerbated by drought (Aguirre et al. 2024). Cc causes a disease known as Sooty Bark Disease on *Acer* spp. (maples) and other tree genera. Cc has been reported to be endophytic (Kelnarová et al. 2017) and to become increasingly aggressive during droughts (Dickenson and Wheeler 1981). The pathosystems or "pathogen x host" combinations I selected were "*Botryosphaeria dothidea* x *Quercus agrifolia* (Qa) (California coast live oak)" and "*Cryptostroma corticale* x *Acer saccharinum* (As) (silver maple)". The first pathosystem has been known since 1994 (Brooks and Ferrin 1994) but no formal pathogenicity studies have been completed yet, while the second one has been formally reported for the first time in 2024 (Garbelotto et al. 2024). The environmental stressor I chose was drought or water stress. This was an obvious choice, given that the literature reports that disease caused by both pathogens is intensified by drought. Both Bd and Cc are canker fungi, and stem decline and dieback of plants infected by these fungi is associated with the presence of cankers developing in the cambium and affecting the xylem as well. Cankers are visible and they can be accurately measured by gently peeling off the bark thus exposing the discolored phloem and xylem. Given that cankers are easily measured and that their size is positively correlated with disease severity and pathogens' virulence (Kranz 1988), these pathogens are ideal candidates for this study.

Two months before each experiment, potted plants were placed in a lathehouse on the U.C. Berkeley campus and watered regularly. Oaks were in 20 L pots, had an average caliper of 13.12 mm and an average height of 97.43 cm. Maples were in 57 L pots, had an average caliper of 2.7 cm and an average height of 3.54 m. A loam potting soil was used for both species. Two weeks before pathogen inoculation, potted plants were assigned to one of two treatments. In Treatment 1 ww (ww = well-watered), plants were well

watered by ensuring the soil moisture was at or slightly above field capacity. Field capacity is at about 30% water content for loamy soils (NRCS 2019). This was accomplished by weighing the pots filled with dry soil (W) and by adding a volume of water equal to 35% of the dry pot weight (W) to potted plants. That amount of water was given to all potted plants, after plants were kept for 7–10 days without watering. Once watered, potted plants were weighted and that weight (Www1x, where 1 identifies the baseline weight and x identifies the individual pot) represented the weight of each potted plant at or slightly above field capacity. Twice a week, each pot was weighted and then the amount of water necessary to bring each potted plant x to its original Www1x was added to each pot x. In Treatment 2 ws (ws = water stressed), plants were minimally watered ensuring the soil moisture was just above the permanent wilting point, i.e. when the water content of a loam reaches 10% of water content. The weight W of the pot filled with dry soil measured above was used to calculate a volume of water corresponding to 15% of the weight of the dry soil pot. The threshold chosen was higher than 10%, based on our extensive experience, to ensure the soil moisture level never went below the permanent wilting point. That volume of water was poured in all potted plants in this treatment, after the plants were left unwatered for 7–10 days. Each pot was weighted after it was watered, and that weight (Wws1x) represented the baseline weight of each plant slightly above wilting point. Twice a week, each pot was weighted and then the amount of water necessary to bring each potted plant x to its original Wws1x was added to each pot x. The trial included 12 potted maples and 16 potted oaks. For each treatment, four plants were inoculated with a fungus and four were mock inoculated. Only two plants per treatment were used for mock inoculations of maples.

Potted plants were kept in the two watering regimes for 2 weeks. The day before pathogen inoculation, all pots were well watered. Inoculations were performed by using a 8 mm-diameter cork borer to excise a plug of bark on the stem of the potted plants and expose the phloem below it. The circular cuts were at least 50 cm above the soil line, to minimize contamination by soil fungi. Cultures of Bd (isolate SS4 from *Quercus lobata*) and Cc (isolate T6 from silver maple) were grown for 7–10 days in Petri dishes filled with half strength acidified PDA (APDA). A 6 mm-diameter cork-borer was used to excise plugs of inoculum from the edges of the fungal colonies. Plugs were placed in each circular inoculation wound with the aerial mycelium touching the plant phloem. The bark plug was then replaced on top of the inoculation point, with the bark side always towards the outside, and, finally, the inoculation point was sealed with softened grafting wax and wrapped with reflective silver tape (Fig. 1A). Mock inoculations were performed in an identical fashion, but inoculum plugs came from sterile Petri dishes filled with half strength APDA. Oaks were inoculated once in a single point, while maples were inoculated in three points 30 cm from one another. After plants were inoculated, they were immediately returned to their assigned water regime. Maple and oak inoculations were performed on August 23rd, 2023, and on October 4th, 2021, respectively. Six weeks post inoculation, watering stopped for the WS treatment and at eight weeks post inoculation, all plants were still alive and the experiment ended. At the end of the trial, the bark around the inoculation point was gently scraped with a sterile scalpel to uncover the underbark lesion. Then, lesions length above and below the inoculation point was carefully measured with a ruler (Fig. 1B). Finally, by using a sterile scalpel and sterile tweezers, small chips of wood at the top and bottom edges of the lesion were

Fig. 1 **A** Inoculation point of *Cryptostroma corticale* on the stem of a water-stressed potted silver maple, at week 6 of the experiment. Note the darker lesion visible on the outer bark, above and below the metallic tape. **B** The underbark lesion caused by *Botryospaheria dothidea* wound-inoculated on the stem of a well-watered potted California coast live oaks, at the end of the experiment, 8 weeks post inoculation

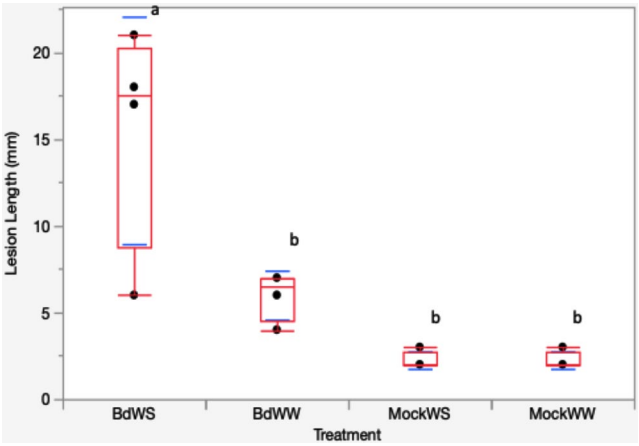

Fig. 2 A box plot graph of lesions lengths caused in 8 weeks by *Botryosphaeria dothidea* (Bd) wound-inoculated under the bark of potted California coast live oaks. The treatments are: Bd inoculated on well-watered plants (BdWW), mock-inoculated well-watered plants (MockWW), Bd inoculated on water-stressed plants (BdWS), mock-inoculated water-stressed plants (MockWS). See text for detailed explanation of watering regimes. ANOVA indicates there are significant differences among treatments ($P < 0.0003$), letters indicate homogeneous groups obtained with a Tukey test and with alpha set at 0.05

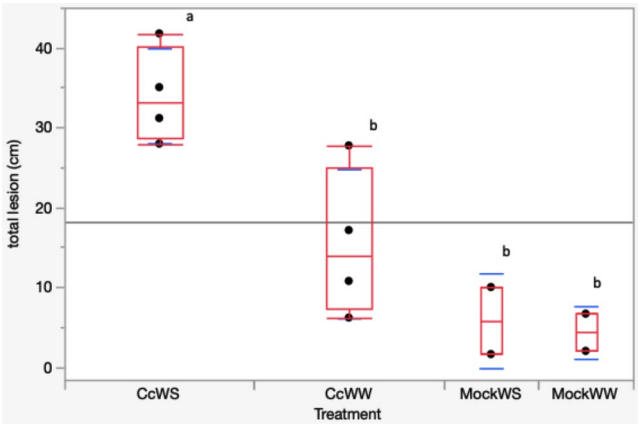

Fig. 3 A box plot graph of lesions lengths caused in 8 weeks by *Cryptostroma corticola* (Cc) wound-inoculated under the bark of potted silver maples. The treatments are: Cc inoculated on well-watered plants (CcWW), mock-inoculated well-watered plants (MockWW), Cc inoculated on water-stressed plants (CcWS), mock-inoculated water-stressed plants (MockWS). See text for detailed explanation of watering regimes. ANOVA indicates there are significant differences among treatments ($P < 0.0001$), letters indicate homogeneous groups obtained with a Tukey test and with alpha set at 0.05. Horizontal line represents the grand average of the entire dataset

plated on half strength APDA. Neither Bd nor Cc were ever reisolated from mock-inoculated controls, but re-isolation success was 100% both for Bd from oaks and for Cc from maples.

Lesions' sizes caused by Bd on Qa were overall different (ANOVA $P = 0.0003$), depending on treatment and presence/absence of fungal inoculation (Fig. 2). The average lesion length in Bd-inoculated and well-watered oaks was 6 mm (SE = 1.41) and, although larger than that equaling 2.25 mm (SE = 0.5) in well-watered mock-inoculated oaks, it was not statistically significant. Because most of the trials for fulfilling Koch's postulates are performed in the absence of an abiotic stressor, this result would lead to the erroneous conclusion that Bd is not a significant pathogen of California coast live oak. However, when looking at the results of the water-stressed treatment, the average lesion length caused by Bd on Qa was 15.5 mm (SE = 6.5) and different ($P = 0.05$) from the average lesion measuring 2.25 mm (SE = 0.5) in the controls. Furthermore, the average lesion caused by Bd on water-stressed oaks was seven times greater than that caused by Bd on well-watered oaks and that difference was significant ($P = 0.05$). It is obvious that without factoring in drought, we may fail to identify emerging pathogens with a biology and ecology similar to Bd. This failure will on one side result in the failure of phytosanitary programs and, on the other, it may prevent land managers to deploy the best available disease management programs. Based on the data, we would also underestimate by a factor of 7 the virulence of Bd on California coast live oaks in the presence of drought conditions and this would bias any predictive model based on laboratory data. The Environmental Disease Component Index I will explain below may be useful to identify those "pathogen x host" combinations for which abiotic stressor may truly be game changers in terms of disease severity and host morbidity.

Results of the second experiment, inoculating Cc on As, were comparable to those of the first trial (Fig. 3). Difference among the various treatments, with each lesion nested within the individual potted tree where it had been inoculated, were overall significant (ANOVA $P < 0.0001$). When limiting the analysis to the well-watered treatment dataset, average lesion length caused by Cc on well-watered maples was 15.4 cm (SE = 4.5) and larger ($P = 0.04$) than the lesion in mock-inoculated plants measuring 4.3 cm (SE = 2.3). Thus proof of pathogenicity was obtained by running a standard pathogenicity test without the added abiotic stress as reported in Garbelotto et al. (2024). However, when the entire dataset was included in the analysis, while average lesion length in the water stressed Cc-inoculated treatment measured 36.4 cm (SE = 2.38) and was larger ($P < 0.0001$) than the average lesion in water-stressed mock-inoculated plants measuring 5.8 cm (SE = 3.4), the difference between fungus-inoculated and mock-inoculated well-watered plants became insignificant (Fig. 3). Furthermore, when lesions caused by Cc on well-watered maples were compared to Cc lesions caused on water-stressed maples, the average lesion size of the latter treatment was 2.5 times greater and larger ($P < 0.0001$) than the average lesion size Cc caused on well-watered maples. This experiment primarily shows that drought will greatly accelerate sooty bark disease of trees,

hence it needs to be factored in when formulating predictions. Additionally, I believe that, as indicated by the much lower P value obtained when including data from the water-stressed treatment ($P < 0.0001$ vs. $P < 0.04$), the data also show that differentiating lesions of mock-inoculated trees from those of fungus-inoculated trees becomes much more robust when applying the abiotic stressor. This aspect may be particularly important when designing trials for fulfilling Koch's postulates, given that often they include a limited number of plants and hence are more susceptible to the effect of outliers.

Given the ongoing global climate change, it is a current challenge to produce information on what kind of changes we may expect in disease severity for any given change in climatic and environmental conditions. I propose a simple Environmental Disease Component Index (EDCI) as a way to provide insights on how much an environmental stressor may affect disease in any given pathosystem. The EDCI ranges between 0 (pathogen is not affected by the presence of an abiotic stressor) to 1 (environmental stressor is entirely driving pathogenicity). In order to calculate the EDCI, one needs to have a)- a pathosystem in which disease severity can be confidently assessed using a measurable metric; and b)- a stressor that is known to interact with disease expressions and that can be either measured accurately in nature or can be created and controlled experimentally. Trials have to be performed in the same place, at the same time and on plant stock with identical provenance and history. Finally, the EDCI should be calculated when there are significant differences in disease severity among treatments. We believe that the two experiments presented here can be used to provide an example of how to calculate the EDCI as summarized in Table 1.

Based on the formula in Table 1, we calculate the EDCI values as follows:

$$\text{“}B.\,dothidea \times Q.\,agrifolia\text{”}\ \text{EDCI}_{\text{ws}} = [1 - (43.20 - 14.14)/(354.21 - 14.14)] = [1 - (29.06/340.07)] = 0.91$$

$$\text{“}C.\,corticola \times A.\,saccharinm\text{”}\ \text{EDCI}_{\text{ws}} = [1 - (14.738 - 4.33)/(35.98 - 5.83)] = [1 - (10.408/30.15)] = 0.65.$$

The EDCI values above indicate that both fungi increase their virulence in drought conditions, but Bd much more than Cc. I note that the relationship between the increase in disease severity associated with a changed environment and the value of the ECDI is not linear, as exemplified in Fig. 4. I also note that the ECDI informs on the relative increase of pathogen virulence associated with environmental change, but does not per se define how aggressive a pathogen may be in the absence of the environmental stressor. That information needs to be gathered from the literature or assessed experimentally. In spite of its obvious limitations, a positive EDCI value may help to identify those pathogens that should be analyzed in the presence of environmental stressors and that may become emergent as climate changes. The dual synchronous test in the absence and presence of key abiotic stressor will help to identify pathogens that otherwise would be defined as non-pathogens based on the results of

Table 1 Definitions and formula for the calculation of the Environmental Disease Component index associated with water-stress (drought)

Term	Definition
$EDCI_{ws}$	Environmental Disease Component Index associated with water stress (ws)
ww	Soil at slightly above field capacity or baseline well-watered (ww) environmental condition
ws	Soil at slightly above permanent wilting point or condition of water stress (ws)
L_{ws}	Average lesion size in water-stressed plants inoculated with a pathogen
LC_{ws}	Average lesion size in water-stressed mock-inoculated control plants
L_{ww}	Average lesion size in well-watered plants inoculated with a pathogen
LC_{ww}	Average lesion size in well-watered mock-inoculated control plants

EDCI Formula* *When the nominator or the denominator are zero or negative, use the minimum unit of the metric used to assess disease severity

$$EDCI_{ws.} = [1 - (L_{ww} - LC_{ww}) / (L_{ws.} - LC_{ws})]$$

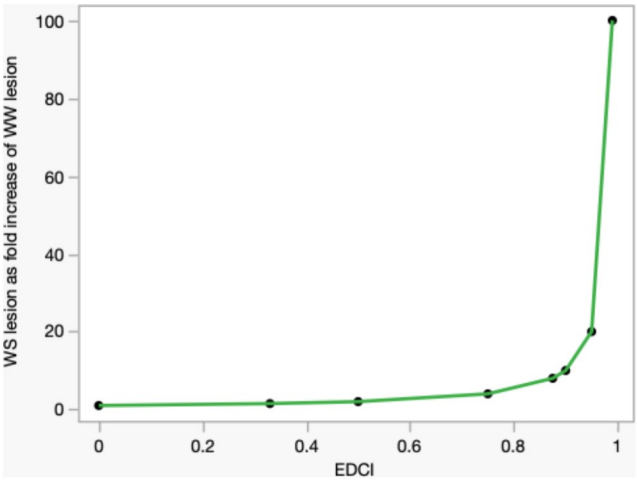

Fig. 4 Positive values of the Environmental Disease Component Index (EDCI) on the x axis and the increase in disease symptom severity associated with the presence of an environmental stressor (water-stressed or WS lesion) on the y axis, expressed as fold-increase (1×, 2×, 10×, etc.) of the disease symptom severity measured in the absence of the stressor (well-watered WW lesion). Note that the EDCI is asymptotic as it approaches its maximum positive value of 1. If disease symptoms severity decreases in the presence of a stressor, the EDCI will be negative

traditional pathogenicity tests. Our inoculation study under water stress proves that, in fact, *Botryosphaeria dothidea* is a true pathogen of Coast live oaks (*Quercus agrifolia*). This study further indicates that *Cryptostroma corticale* is likely to be more virulent on silver maple in drought conditions and provides a dataset that more convincingly proves its pathogenicity on this recently reported host (Garbelotto et al. 2024).

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Data Availability Data available upon request.

Declarations

Competing Interests The authors declare there are no conflicts of interest.

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