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Editorial

Variability and Disturbances as Key Factors in Forest Pathology and Plant Health Studies

Matteo Garbelotto [1,*] and Paolo Gonthier [2]

[1] Department of Environmental Science, Policy and Management, University of California at Berkeley, 54 Mulford Hall, Berkeley, CA 94720, USA

[2] Department of Agricultural, Forest and Food Sciences, University of Torino, Largo P. Braccini 2, I-10095 Grugliasco, Italy; paolo.gonthier@unito.it

* Correspondence: matteog@berkeley.edu; Tel.: +1-510-6434282

Received: 13 November 2017; Accepted: 14 November 2017; Published: 15 November 2017

Abstract: The plant disease triangle (PDT) is as old as the field of modern plant pathology, and it postulates that any plant disease is the outcome of the interaction between a pathogen, a host, and the environment. Recently, the need has emerged to study not only how the three elements of the PDT directly influence disease, but to focus on how they indirectly affect one another, consequently modifying the final outcome. It is also essential to structure such analyses within three major external frameworks provided by landscape level disturbances, climate change, and anthropogenic effects. The studies included in this issue cover a wide range of topics using an equally varied list of approaches, and they showcase the important role these indirect and often non-linear processes have on the development of forest diseases.

Keywords: biological invasions; climate; disease triangle; epidemiology; forest; Geographic Information System; modeling; variability

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A SCOPUS (https://www.scopus.com/) search using the key words "forest pathogen", "invasive", and "variability" reveals a recent reborn interest in the concepts of variability and disturbances as major drivers of infectious forest diseases (Figure 1). Although it is still convenient to partition such variability according to the three main elements of the plant disease triangle (PDT), that is, pathogen, host, and environment [1], our interest is spiked not so much by the study of the individual variables per se, but rather by their dynamic interaction. Advancements in computational and statistical approaches provide a solid framework to focus on those effects that may have been previously discarded or considered marginal because of being too difficult to measure using standard passive analytical approaches [2,3]. These advancements allow us to compute the outcomes of multiple interactions with greater confidence than in the past, and they have provided a considerable push to cross-over across fields. Additionally, this renewed interest in the disease triangle is occurring in a broader framework provided by the awareness of the importance of both anthropogenic and climate change effects [4,5]. It should be noted that the disease triangle may be used to predict epidemiological outcomes not only in plant health, but also in public health, both in local and global communities [6]. The main aim of this special issue was to focus on disturbances and variability as important factors determining the final outcome of forest diseases.

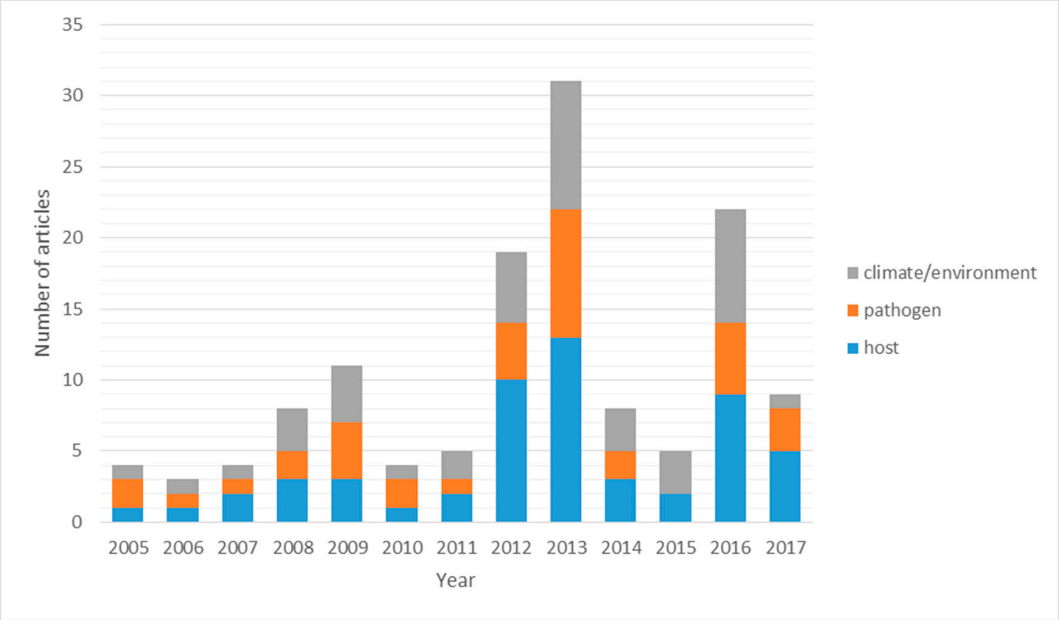

Figure 1. Number of articles retrieved on SCOPUS (https://www.scopus.com/) using the search terms "forest pathogen", "invasive" and "variability" (as of 27 July 2017) partitioned according to the three main elements of the plant disease triangle.

"Landscape-scale disturbances such as wind, fire, or land use can (i) modify the impacts of a disease, or (ii) can be influenced by disease in a manner which increases or decreases the ecological impacts of these disturbances" [7]. The above quote clearly summarizes the two-way interconnectivity between heterogeneity at the landscape level and diseases. However, it is not only the heterogeneity of the landscape that drives the epidemiology and the final outcome of forest diseases. In fact, variability in the environment, as well as in host and pathogen populations, can also have a profound impact on the spread and impacts of forest diseases. This special issue attempts to summarize some of the knowledge on this broad and novel aspect of forest pathology while providing some provoking case studies investigating several different aspects of this variability.

We have long known that hosts and pathogens are in a constant arms race through which resistance and virulence are in continuous evolution, and it has been repeatedly postulated that shorter generation times will accelerate the process [8,9]. In this issue, Soularue et al. [10] through a convincing model show that humans can also play a role in the co-evolutionary arms race, by shortening the rotation time of plantations and thus accelerating the evolution of virulence in the pathogen. This information is novel because it goes well beyond the known effects that humans can have on disease severity by altering ecosystems. Such more widely studied effects are also described in this special issue, for example, the dense monoculture of young trees and off-site plantings caused by the use of exotic species, leading to an increased susceptibility to both exotic and native emergent pathogens [11,12]. At the same time, emergent diseases are far from being in a stationary phase; climate change is currently affecting several pathosystems, especially where pathogens and/or hosts may be at the fringes of their natural or naturalized range [13]. For instance, Kubiak et al. [14] point out that increasing temperatures will allow the root rot pathogens *Armillaria* spp. to grow all year round and to decay wood more effectively, thus significantly enhancing their spread rate and pathogenicity.

Likewise, the changing climate may negatively affect the physiology of native trees, making them more attractive to insects and more susceptible to diseases caused by endophytic fungi that have turned into pathogens [15]. In addition, in such a predicament, contagion may be further compounded by the fact that the rates of vectoring of such fungi by insects also increase, as a result of a greater frequency of encounters between fungi and insects in weakened plants [15].

This is a new world we are living in: a globalized world, a changing world, and a world that requires new approaches to maximize the return of scientific investigation. The application of landscape ecology approaches [7] is greatly enhancing our insights into non-linear processes [16]. Likewise, the use of crowdsourced data provides an opportunity to generate datasets of an inconceivable scale until recently. Lione et al. [17] used crowdsourced data to uncover the non-linear progression of emergent diseases. In this paper, the authors identify precise environmental and topographic conditions that result in a reversion of infection status (from positive to negative) for the invasive and destructive forest disease Sudden Oak Death in California.

Often, even when the variability of the landscape, environment, and main ecological parameters are all embedded in our research, it is arduous to include variability of the host response in our studies. Most of the literature in this area in fact has focused on gene-for-gene resistance, but the effects of other types of resistance have been less widely studied [18]. The paper on the Eucalyptus rust *Austropuccinia psidii* [19] provides solid evidence of a phenological type of resistance present in older leaves, while even more complex is the report by Chieppa et al. [20] suggesting that genetically inherited susceptibility to a vascular fungus results in great susceptibility to changes in water availability. While interspecific competition driven by pathogens is well known [21], the example by Chieppa et al. [20] is a classic example of virtually unstudied intraspecific competition cryptically driven by a pathogen.

Even if this special issue purposefully does not focus primarily on diseases caused by an exotic organism, it would be impossible not to include this topic in an issue on the effect of variability on the epidemiology and impacts of forest diseases. Variability certainly is the issue when studying most exotic pathosystems; in fact, how can exotic pathogens be so successful in novel environments despite their limited genetic variability? There is no a single answer to this question, and it is our belief that simply invoking a lack of coevolution does a disservice to our learning of the complex mechanisms driving the invasion by exotic pathogens. In this issue, several hints are provided regarding factors other than the lack of coevolution to explain successful invasions by pathogens. In the case of cypress canker, the use of the artificially created hybrid Leyland cypress has significantly increased the severity of outbreaks, even where the causal agent is native (e.g., in California) [22]. In the case of laurel wilt, the high susceptibility of cultivated avocados has accelerated the spread of the disease in natural ecosystems [23]. On top of this, and unfortunately, the exotic laurel wilt pathogen, introduced in conjunction with the introduction of an exotic ambrosia beetle, has been picked up by multiple native beetles, thus immediately broadening its host range. Finally, exotic diseases can often emerge because humans have provided an abundance of exotic hosts. Such hosts, being exotic, generally can be regarded as planted off-site. This is the case for *Austropuccinia psidii* on Eucalyptus planted in South America [19], for *Lecanosticta acicola* on *Pinus radiata* grown in Spain [12], and likely for the many cultivated hosts of *Lasiodiplodia theobromae* [24].

This special issue contains 13 articles that we hope will be thought-provoking in more than a single way. The articles include widely different approaches, scales, and technical methodologies, and they well represent the cutting edge of contemporary forest pathology. The expectation of this special issue was to represent a range of approaches currently employed to study variability in tree diseases. We hope the reader will agree that this expectation has been met, and we hope he/she will concur that in the process of compiling this issue, we may have put together an excellent textbook for an advanced class in forest pathology.

Acknowledgments: The authors of this paper and co-editors of the special issue "Forest Pathology and Plant Health" are grateful to all the authors and reviewers of the special issue.

Author Contributions: M.G. and P.G. contributed equally to this article.

Conflicts of Interest: The authors declare no conflict of interest. The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript; or in the decision to publish the results.

References

1. Agrios, G. *Plant Pathology*, 5th ed.; Academic Press: New York, NY, USA, 2005.
2. Yun, L.; Zeger, S.L. On the equivalence of case-crossover and time series methods in environmental epidemiology. *Biostatistics* **2006**, *8*, 337–344.
3. Roche, B.; Guégan, J.-F.; Bousquet, F. Multi-agent systems in epidemiology: A first step for computational biology in the study of vector-borne disease transmission. *BMC Bioinform.* **2008**, *9*, 435. [CrossRef] [PubMed]
4. Desprez-Loustau, M.-L.; Aguayo, J.; Dutech, C.; Hayden, K.J.; Husson, C.; Jakushkin, B.; Marçais, B.; Piou, D.; Robin, C.; Vacher, C. An evolutionary ecology perspective to address forest pathology challenges of today and tomorrow. *Ann. For. Sci.* **2016**, *73*, 45–67. [CrossRef]
5. Pautasso, M.; Doring, T.; Garbelotto, M.; Pellis, L.; Jeger, M. Impacts of climate change on plant diseases—Opinions and trends. *Eur. J. Plant Pathol.* **2012**, *133*, 295–313. [CrossRef]
6. Scholthof, K.G. The disease triangle: Pathogens, the environment and society. *Nat. Rev. Microbiol.* **2007**, *5*, 152–156. [CrossRef] [PubMed]
7. Cobb, R.C.; Metz, M.R. Tree diseases as a cause and consequence of interacting forest disturbances. *Forests* **2017**, *8*, 147. [CrossRef]
8. Parker, I.M.; Gilbert, G.S. The evolutionary ecology of novel plant-pathogen interactions. *Annu. Rev. Ecol. Evol. Syst.* **2004**, *35*, 675–700. [CrossRef]
9. Oliva, J.; Boberg, J.; Hopkins, A.J.M.; Stenlid, J. Concepts of epidemiology of forest diseases. In *Infectious Forest Diseases*; Gonthier, P., Nicolotti, G., Eds.; CABI: Wallingford, UK, 2013; pp. 1–28.
10. Soularue, J.-P.; Robin, C.; Desprez-Loustau, M.-L.; Dutech, C. Short rotations in forest plantations accelerate virulence evolution in root-rot pathogenic fungi. *Forests* **2017**, *8*, 205. [CrossRef]
11. Munck, I.A.; Luther, T.; Wyka, S.; Keirstead, D.; McCracken, K.; Ostrofsky, W.; Searles, W.; Lombard, K.; Weimer, J.; Allen, B. Soil and stocking effects on Caliciopsis canker of *Pinus strobus* L. *Forests* **2016**, *7*, 269. [CrossRef]
12. Ortíz de Urbina, E.; Mesanza, N.; Aragonés, A.; Raposo, R.; Elvira-Recuenco, M.; Boqué, R.; Patten, C.; Aitken, J.; Iturritxa, E. Emerging needle blight diseases in Atlantic *Pinus* ecosystems of Spain. *Forests* **2017**, *8*, 18. [CrossRef]
13. Thomas, C.D. Climate, climate change and range boundaries. *Div. Distr.* **2010**, *16*, 488–495. [CrossRef]
14. Kubiak, K.; Żółciak, A.; Damszel, M.; Lech, P.; Sierota, Z. *Armillaria* pathogenesis under climate changes. *Forests* **2017**, *8*, 100. [CrossRef]
15. Panzavolta, T.; Panichi, A.; Bracalini, M.; Croci, F.; Ginetti, B.; Ragazzi, A.; Tiberi, R.; Moricca, S. Dispersal and propagule pressure of *Botryosphaeriaceae* species in a declining oak stand is affected by insect vectors. *Forests* **2017**, *8*, 228. [CrossRef]
16. Holdenrieder, O.; Pautasso, M.; Weisberg, P.J.; Lonsdale, D. Tree diseases and landscape processes: The challenge of landscape pathology. *Trends Ecol. Evol.* **2004**, *19*, 446–452.
17. Lione, G.; Gonthier, P.; Garbelotto, M. Environmental factors driving the recovery of bay laurels from *Phytophthora ramorum* infections: An application of numerical ecology to citizen science. *Forests* **2017**, *8*, 293. [CrossRef]
18. Laine, A.L.; Burdon, J.J.; Dodds, P.N.; Thrall, P.H. Spatial variation in disease resistance: From molecules to metapopulations. *J. Ecol.* **2011**, *99*, 96–112. [CrossRef] [PubMed]
19. Ruiz Silva, R.; Costa da Silva, A.; Antônio Rodella, R.; Eduardo Serrão, J.; Cola Zanuncio, J.; Luiz Furtado, E. Pre-infection stages of *Austropuccinia psidii* in the epidermis of eucalyptus hybrid leaves with different resistance levels. *Forests* **2017**, *8*, 362. [CrossRef]
20. Chieppa, J.; Eckhardt, L.; Chappelka, A. Simulated summer rainfall variability effects on loblolly pine (*Pinus taeda*) seedling physiology and susceptibility to root-infecting ophiostomatoid fungi. *Forests* **2017**, *8*, 104. [CrossRef]
21. Gilbert, G.S. Evolutionary ecology of plant diseases in natural ecosystems. *Annu. Rev. Phytopathol.* **2002**, *40*, 13–43. [CrossRef] [PubMed]
22. Danti, R.; Della Rocca, G. Epidemiological history of Cypress Canker Disease in source and invasion sites. *Forests* **2017**, *8*, 121. [CrossRef]

23. Ploetz, R.C.; Kendra, P.E.; Choudhury, R.A.; Rollins, J.A.; Campbell, A.; Garrett, K.; Hughes, M.; Dreaden, T. Laurel wilt in natural and agricultural ecosystems: understanding the drivers and scales of complex pathosystems. *Forests* **2017**, *8*, 48. [CrossRef]
24. Mehl, J.; Wingfield, M.J.; Roux, J.; Slippers, B. Invasive everywhere? Phylogeographic analysis of the globally distributed tree pathogen *Lasiodiplodia theobromae*. *Forests* **2017**, *8*, 145. [CrossRef]