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Research article

Heat stress can change the competitive outcome between fungi: insights from a modelling approach

Felix Wesener[1], Matthias C. Rillig[2,3] and Britta Tietjen[1,3]

[1]Freie Univ. Berlin, Inst. of Biology, Theoretical Ecology, Berlin, Germany
[2]Freie Univ. Berlin, Inst. of Biology, Ecology of Plants, Berlin, Germany
[3]Berlin Brandenburg Inst. of Advanced Biodiversity Research (BBIB), Berlin, Germany

Correspondence: Britta Tietjen (britta.tietjen@fu-berlin.de)

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Under a changing climate, soil fungal communities will increasingly be subject to periods of heat stress. These periods can affect the performance of individual fungi and their competition for space and resources. Competition between fungi is strongly controlled by the exudation of inhibitory compounds, resulting in different competitive outcomes that range from overgrowth of the inferior competitor to a deadlock, where the competing fungi inhibit each other. As heat stress can alter the competitive outcome between fungi, the community composition can also change strongly. So far, a general understanding of the mechanisms that drive the competitive outcome between fungi under heat stress is still missing. However, this understanding is essential to assess important community functions, such as decomposition or mediation of plant nutrition, which strongly depend on the fungal community composition.

Here, we used a partial differential equation (PDE) model simulating two fungal competitors in a two-dimensional space, to mechanistically explain the observed change of fungal competition under heat stress. The model describes mycelial growth, the production and secretion of antifungal compounds and the synthesis of heat shock proteins of interacting colonies. We found a heat stress-induced lag phase favouring the accumulation of antifungal compounds and the build-up of inhibitor fields. This led to a qualitative change of the competitive outcome, reducing the occurrence of overgrowth by two thirds. The changes in competitive outcome favoured slower growing species, which benefit more strongly from the additional time during a stress-induced lag to build up a defence or block territory that would otherwise be quickly claimed by faster competitors.

Our work is an important step towards understanding how environmental changes may lead to qualitative changes in competitive outcomes. Our results show the importance of explicitly including species interactions into studies of climate change effects.

Keywords: fungal community, induced stress defence, mathematical model, microbiology, mycology, PDE model

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Introduction

Climate warming is increasing the frequency and intensity of heat periods, and will affect community dynamics and structure of soil fungal communities (Tedersoo et al. 2014, Cavicchioli et al. 2019). Soil fungi fulfil numerous important ecological functions, such as soil carbon cycling, mediation of plant nutrition or formation of soil structure (Boddy 2001, Ritz and Young 2004, Treseder and Lennon 2015). Because these soil functions have a high dependence on local-scale dynamics of soil fungal communities (Bradford et al. 2014, Wagg et al. 2014), understanding the drivers of community structure is an important step towards assessing how soil ecosystem functions are affected by environmental changes such as higher frequencies of heat stress events.

Due to the sessile nature of fungal individuals, fungi need to colonise territory to gain access to organic resources, and thus fungal competition for nutrients is synonymous with competition for space (Boddy 2000, Hiscox and Boddy 2017). This competition can be divided into two mechanisms: primary resource capture describes the colonisation of previously uncolonised territory, and is most effective for species that exhibit fast growth or highly efficient dispersal (Klepzig and Wilkens 1997, Boddy 2000, Prospero et al. 2003). Secondary resource capture, which is the capture of territory already inhabited by another fungus, is characterized by various antagonistic interactions between individuals. Laboratory experiments revealed the existence of different qualitative competitive outcomes between fungi (Stahl and Christensen 1992, Boddy 2000, Falconer et al. 2008). First, inhibition at contact or at a distance leads to a local cessation of growth for both species. This usually results in a deadlock in which neither species invades territory inhabited by the other (Schoeman et al. 1996, Hiscox et al. 2010, El Ariebi et al. 2016). Second, overgrowth describes the invasion of inhabited territory of an inferior competitor. The overgrown mycelium can be fully viable, but overgrowth can ultimately also lead to replacement of the inferior species (Maynard et al. 2017). Third, intermediate outcomes can be partial overgrowth of one species, as well as intermingling, when the hyphae of two species overlap and share certain regions. These fungal competitive outcomes are mediated by the exudation of various secondary metabolites, and their production varies not only between, but also within species (White and Boddy 1992, Marx 2004, Hiscox et al. 2010, Knowles et al. 2019). How exactly these different types emerge from the production of secondary metabolites, and how they affect competition between fungi, is yet to be fully understood.

Competitive outcomes in fungal pairs have been shown to change with temperature (A'Bear et al. 2013, Hiscox et al. 2016), and the metabolic response under changing temperature can differ markedly among community members (Rawlings et al. 2022). Induced heat stress defences can further influence the outcome of competition, if a species manages to build up a stress response earlier than its competitors (Wesener et al. 2021). In response to heat stress pulses of 45°C, fungi immediately stop growing, a pause that can last up to several days (Wesener et al. 2021), as cells are damaged, proteins are denatured and most cellular processes are halted (Plesofsky-Vig and Brambl 1985, Sharma et al. 2009). In contrast, many extracellular inhibitory compounds have been shown to be more heat resistant (San-Lang et al. 2002, Taechowisan et al. 2003, Marx 2004, Sena et al. 2011). During this so-called lag phase (Wesener et al. 2021), the production of different classes of heat shock proteins is upregulated in the fungus (Plesofsky-Vig and Brambl 1985). These heat shock proteins refold denatured proteins or protect nascent proteins (Tereshina 2005, Liberek et al. 2008, Tiwari et al. 2015). Other substances, such as the disaccharide trehalose, help stabilise proteins and membranes (Singer and Lindquist 1998, Elbein et al. 2003, Tereshina 2005).

After a heat pulse, a slow-growing species that exhibits a high production of heat shock proteins might be able to overgrow territory that has not yet been claimed by a faster growing competitor whose expansion is halted for a longer period. However, not only does the distribution of territory between species change under heat stress, but the outcomes of interaction have also been observed to change (Schoeman et al. 1996, Hiscox et al. 2016). Therefore, periods of heat stress might ultimately be a defining factor for fungal community assembly (Hiscox et al. 2016) and community composition (Allison and Martiny 2008, Shade et al. 2012). It is thus important to understand not only the effect of antifungal compounds on fungal competition, but also the interplay of heat stress and competition.

To shed light on the mechanisms underpinning fungal growth and competition, several studies have mathematically described fungal individuals using partial differential equations (PDEs). Edelstein (Edelstein 1982, Edelstein and Segel 1983, Edelstein-Keshet and Ermentrout 1989) is recognized as a pioneer of PDE models simulating mycelial growth in a two-dimensional space based on hyphal branching and merging, and her models have since inspired many other approaches. Some PDE models assessed macroscopic movement of biomass rather than hyphae (Davidson et al. 1996, 1997). Follow-up studies have combined both approaches, simulating different types of biomass (Boswell et al. 2002, 2003, Falconer et al. 2005, 2007). Later models have also included interactions between fungal competitors (Falconer et al. 2008, Boswell 2012, Choudhury et al. 2018, Kiziridis et al. 2020) by, for example, adding secondary metabolite production. So far, no modelling study has included induced stress defences in fungi. Therefore, a mechanistic explanation of changing fungal interactions under heat stress is still missing.

In this study, we extend the PDE model of Falconer et al. (2008) by adding a dynamic heat stress defence mechanism. We use the model to describe competitive growth between two soil fungi under different scenarios. Here, we focussed on a general understanding of the effect of different fungal characteristics (represented by traits) on competitive dynamics rather than on reproducing the behaviour of explicit species. Fungal traits are an important means to drive conceptual

understanding of fungal community development and functioning, as they provide generality and predictability (Crowther et al. 2014, Aguilar-Trigueros et al. 2015). In particular, because traits within functional groups are plastic (Naeem and Wright 2003), and many species show different traits and also different life strategies (e.g. ruderal, combative or stress-tolerant) at different developmental stages and in different environments (Pugh and Boddy 1988), a focus on traits is often more informative than predictions based solely on taxonomic classification.

With this study, we aim to answer the following questions: under which conditions does a heat stress change the specific competition between fungi? Which competitive strategies are most successful with and without heat stress? Do optimal stress responses and competitive strategies differ for fungi with different growth rates? Answering these questions will help in gaining an understanding of the mechanisms behind changing competitive outcomes. By disentangling the effects of induced defences and secondary metabolite production, we can explain how the physiological processes of a species affect competition dynamics.

Material and methods

We used a system of PDEs based on Falconer et al. (2005, 2007, 2008) describing the growth of two fungal species and their interactions in space and time, and we extended it by adding the impacts of heat stress. To parameterise our model, we used growth data of two soil fungi from a laboratory experiment. We then used the model to assess the effects of species' growth rates, their response to a heat shock and their production rate of antifungal compounds during fungal interactions.

Model description

The PDE model describes two fungi growing and competing for space on a Petri dish. It calculates the coupled dynamics of different types of biomass (all in units of mol): young, non-insulated hyphae (b_n), insulated hyphae that have undergone the process of rigidification of the cell wall (rigid hyphae b_r) and mobile biomass (b_m). The model also includes the uptake of substrate (s), and the dynamics of inhibitors (i) and heat shock proteins (hsp) of both species (Fig. 1). In the following section, we describe these variables and how we incorporated growth, the production of antifungal compounds and the heat shock defence. See Table 1 for a description of the model parameters and their units.

Fungal growth

For each of the two fungal species, the model assumes three different types of biomass growing on substrate s, which is replenished at a constant rate ($\omega > 0$ day^{-1}). With j referring to the simulated species, the non-insulated biomass b_{nj} represents the fungal hyphae and hyphal tips capable of high substrate uptake (rate λ_{nj}), that grow via a diffusion term scaled by the diffusion coefficient D_{nj}. The non-insulated biomass undergoes rigidification of the cell wall (at a rate c_j) and is converted to insulated, rigid biomass b_{rj} that describes hyphal sections that are significantly reduced in their uptake (rate λ_{rj}) of nutrients (Trinci 1978).

To assess the extension of a fungal mycelium, we defined a threshold mycelial biomass to differentiate between presence and absence of fungal biomass. Similar to Boswell (2012), who defined the leading edge of the fungal individual as the boundary in space where the mycelial density surpasses a certain value, we defined the leading edge where the biomass $b_{nj} + b_{rj} + b_{mj} > 10^{-1}$ mol.

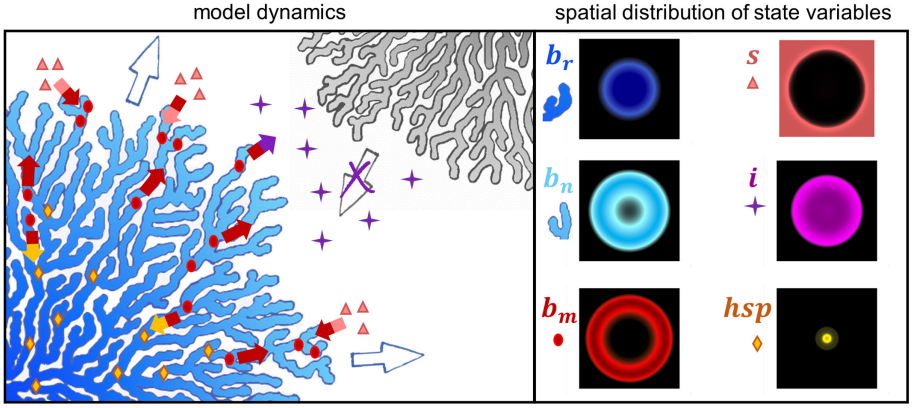

Figure 1. Schematic representation of the model dynamics. Non-insulated biomass (light blue, b_n) diffuses radially from the centre (indicated by white outlined arrows) and is transformed into insulated, rigid biomass (dark blue, b_r) over time. Mobile biomass (red circles, b_m) is moved through the hyphae and immobilised (i.e. converted to non-insulated biomass). Red arrows show the movement of mobile biomass through the hyphae. New biomass is generated from the underlying substrate (pink triangles, s) and is taken up and converted to mobile biomass (pink-red arrows). The inhibitors (purple stars, i), which are produced by mobile biomass (red-purple arrows), can diffuse and stop the local growth of other fungal biomass (shown as grey colony). In case of a heat pulse event, heat shock proteins (yellow diamonds, hsp) are synthesized from mobile biomass (red-yellow arrows). The right box shows an exemplary spatial distribution of the six state variables, i.e. the different compartments, of the same mycelium after ten days.

Table 1. Model parameters and their description and derivation. Parameters with several values were varied to obtain different scenarios: lower and upper boundaries of ranges are indicated with a hyphen, and discrete values are separated by a dash. See Supporting information for sources marked with an asterisk (*).

Parameter	Description	Value	Units	Source
λ_n	Substrate intake rate of non-insulated biomass	0.97	mol^{-1} day^{-1}	Falconer et al. (2008)
λ_r	Substrate intake rate of rigid biomass	0.1	mol^{-1} day^{-1}	Falconer et al. (2008)
c	Rate of conversion of non-insulated to insulated, rigid biomass	0.5	day^{-1}	Boswell et al. (2002)
D_n	Diffusion coefficient of non-insulated biomass	3 – 21	mm^2 day^{-1}	Calibrated
D_m	Diffusion coefficient of mobile biomass	$10^{-7} \times D_n$	mm^2 day^{-1}	Falconer et al. (2005)
α	Immobilisation rate of biomass	0.87	day^{-1}	Falconer et al. (2008)
π	Ratio of mobile biomass to hyphal biomass	$\frac{b_m}{b_n + b_r}$	–	Falconer et al. (2005)
ω	Replenishment rate of external substrate	0.01	day^{-1}	Falconer et al. (2005)
s_m	Amount of maximum substrate per cell	10	mol	Fixed*
Competition				
Ω	Inhibitor production rate	0.1	mol^{-1} day^{-1}	Varied
D_i	Inhibitor diffusion rate	0/10	mm^2 day^{-1}	Varied
ψ	Resistance to competitor's inhibitor	10^{-8}	mol	Calibrated*
Heat stress response				
δ	Max. production rate of *hsp*	0.01–0.2	mol^{-1} day^{-1}	Calibrated
hsp_{lim}	Minimal amount of *hsp* needed to allow normal process rates	0.001	mol	Fixed*
Initial conditions				
b_{n0}	Initial amount of non-insulated biomass	1	mol	Fixed*
b_{r0}	Initial amount of rigid biomass	0	mol	Fixed*
b_{m0}	Initial amount of mobile biomass	1	mol	Fixed*
s_0	Initial amount of substrate	10	mol	Fixed*
r	Radius of initial mycelial plug	3	mm	Experimental design

For the sake of simplicity, we assume that all substrate s taken up by the hyphae and hyphal tips is converted to mobile biomass b_{mj}, which is then transported via diffusion through the hyphae. The diffusion coefficient D_{mj} of mobile biomass b_{mj} is nonlinearly dependent on the local amount of mobile biomass to account for limited transport pathways of the mycelial network. Similarly to Falconer et al. (2005), we assumed a simple nonlinear dependence:

$$D_{mj} = \begin{cases} D_{nj}, & b_{mj} \le b_{m\_lim} \\ 10^{-7} D_{nj}, & b_{mj} > b_{m\_lim} \end{cases}$$

where b_{m_lim} describes a local threshold of mobile biomass concentration.

Parts of the mobile biomass b_{mj} are immobilised (converted to non-insulated biomass b_{nj}). Because immobilisation is conducted by the non-insulated hyphal biomass, it is proportional to immobile biomass b_{nj}. Moreover, following Falconer et al. (2005), we assume that mobile biomass (transporting elements such as vesicles) is needed to conduct conversion, and therefore the immobilisation rate α is modified by π_j:

$$\alpha\pi_j \text{ with } \pi_j = \frac{b_{mj}}{b_{rj} + b_{nj}}$$

If the concentration of mobile biomass, b_{mj} in the hyphae ($b_{rj} + b_{nj}$) is low, the immobilisation of biomass will be reduced as a result of a dilution effect.

Note that two opposed processes are described here: while an increased amount of immobile biomass b_{nj} increases immobilisation $\alpha\pi_j b_{nj}$, it also leads to a decrease via dilution. See Supporting information for a more detailed explanation of the implementation of these processes.

Interactions

To realize fungal interactions, fungi can convert mobile biomass to inhibitor molecules i_j at a rate Ω_j, which can diffuse and halt a competitor's growth. Because we assume that the conversion of mobile biomass to inhibitors happens within non-insulated hyphae, the conversion is dependent on both b_m and b_n. Given the general nature of the model, we did not use i to represent a certain antifungal compound, but instead aimed at capturing the universal dynamics of antifungal compounds such as mycotoxins, chitinolytic enzymes or small antifungal proteins (Taechowisan et al. 2003, Marx 2004, Sena et al. 2011, Knowles et al. 2019). For the sake of simplicity, we did not include the effects that volatile organic compounds (VOCs) can have on fungal interactions (Effmert et al. 2012, Sridharan et al. 2020).

If a competitor's k inhibitor concentration $i_{k \neq j}$ is higher than a species' resistance ψ_j, the local diffusion coefficient of that species' non-insulated biomass is set to zero:

$$D_{nj} = \begin{cases} D_{nj}, & i_{k\neq j} < \psi_j \\ 0, & i_{k\neq j} \geq \psi_j \end{cases}$$

In this study we did not consider the possible effects of autophagy upon encounter with an antagonist, as proposed in some scenarios by Falconer et al. (2008), and thus did not include any other effects of *i*. We assumed that a species is fully resistant to its own toxins and can grow unhindered, even though some fungi might locally disrupt their own mycelium when inhibiting other species (Hiscox and Boddy 2017).

Heat shock response

We expanded the original model to include the effects of heat stress and stress defence mechanisms, i.e. the arrest of growth leading to a lag phase (due to protein misfolding and aggregation) and a return to growth when enough heat shock proteins have been produced. We assumed that, under heat stress, low concentrations of biomass in the periphery of the fungal individual (i.e. beyond the leading edge) are degraded immediately and therefore local biomass that is too small is set to zero.

To include these processes, we introduced a binary variable z_j, which controls all affected cellular processes. Under non-stressed temperature conditions, $z_j = 1$. By setting $z_j = 0$ upon transgression of a critical temperature, all modelled processes are immediately halted, except for the production of heat shock proteins, the replenishment of substrate and the diffusion of inhibitors, which are heat resistant. At the same time, the term controlling heat shock protein production is $(1 - z_j) = 1$, and therefore part of the mobile biomass b_{mj} is converted to heat shock proteins hsp_j at a rate δ_j. When a certain threshold $hsp_j \geq hsp_{lim}$ is reached, the local cellular processes revert to pre-disturbance levels, i.e. $z_j = 1$, representing the protection of nascent proteins and the unfolding or disaggregation of denatured proteins. Simultaneously, the production of hsp_j is halted because $(1 - z_j) = 0$, assuming self-regulation of heat shock protein production (Tereshina 2005). Therefore, if a heat pulse treatment occurs at time t_T,

$$z_j = \begin{cases} 1, & t < t_T \\ 0, & t \geq t_T \text{ and } hsp_j < hsp_{lim} \\ 1, & t > t_T \text{ and } hsp_j \geq hsp_{lim} \end{cases}$$

The stress mediating variable hsp_j integrates several different stress defence mechanisms, such as different classes of heat shock proteins and other substances involved in stabilising proteins and membranes.

The PDE system

The processes described above resulted in the following set of equations for a species *j*:

$$\frac{\partial b_{rj}}{\partial t} = \overbrace{c b_{nj} z_j}^{\text{rigidification}},$$

$$\frac{\partial b_{nj}}{\partial t} = \overbrace{D_{nj}\left(\frac{\partial 2 b_{nj}}{\partial x^2} + \frac{\partial 2 b_{nj}}{\partial y^2}\right) z_j}^{\text{tip movement}} + \overbrace{\alpha \pi_j b_{nj} z_j}^{\text{immobilisation of biomass}} - \overbrace{c b_{nj} z_j}^{\text{rigidification}},$$

$$\frac{\partial b_{mj}}{\partial t} = \overbrace{D_m\left(\frac{\partial^2 b_{mj}}{\partial x^2} + \frac{\partial^2 b_{mj}}{\partial y^2}\right) z_j}^{\text{transport of biomass}} - \overbrace{\alpha \pi_j b_{nj} z_j}^{\text{immobilisation of biomass}} + \overbrace{\left(\lambda_n b_{nj} + \lambda_r b_{rj}\right) s z_j}^{\text{substrate uptake}} - \overbrace{\Omega b_{mj} b_{nj} z_j}^{\text{inhibitor production}} - \overbrace{\delta_j b_{mj} b_{nj}\left(1 - z_j\right)}^{\text{hsp production}},$$

$$\frac{\partial s}{\partial t} = \overbrace{\omega\left(s_m - s\right)}^{\text{replenishment}} - \overbrace{\left(\lambda_n b_{nj} + \lambda_r b_{rj}\right) s z_j}^{\text{substrate uptake}},$$

$$\frac{\partial i_j}{\partial t} = \overbrace{\Omega b_{mj} b_{nj} z_j}^{\text{production}} + \overbrace{D_i\left(\frac{\partial^2 i_j}{\partial x^2} + \frac{\partial^2 i_j}{\partial y^2}\right)}^{\text{inhibitor diffusion}},$$

$$\frac{\partial \text{hsp}_j}{\partial t} = \overbrace{\delta_j b_{mj} b_{nj}\left(1 - z_j\right)}^{\text{production}},$$

where

$$\pi_j = \frac{b_{mj}}{b_{nj} + b_{rj}}$$

We discretized the system of PDEs over a grid of 172 × 172 cells with one grid cell representing an area of 0.5 × 0.5 mm^2 to approximate fungal growth on a Petri dish 86 mm in diameter. To calibrate growth and heat shock responses, we assessed species in isolation with initial inoculum of 6 mm diameter placed in the centre of the dish. For dual cultures, two initial inocula of 6 mm diameter were placed on the horizontal diameter equidistant from each other and the border of the Petri dish. The initial conditions of the state variables describing the biomass b_{nj}, b_{rj}, b_{mj} were therefore set to b_{n0}, b_{r0}, b_{m0} within a radius of 3 mm around the centre of the initial inocula, and 0 everywhere else. The substrate *s* was distributed homogeneously over the grid, and initially no inhibitors *i* and heat shock proteins *hsp* were present. We applied Neumann boundary conditions preventing any flux into or out of the system; that is, at the borders of the system, the rate of change of the dependent variables was zero.

The system was solved numerically with the finite element method using the Python package 'FiPy' (Guyer et al. 2009).

Parameterisation and calibration

We parameterised our model using a parameter set of a fungal trait type from Falconer et al. (2008) (Table 1). To define

the rate of cell wall rigidification c, we assumed a conversion rate of 0.5 day^{-1} from active to inactive mycelium used by Boswell et al. (2002). We assessed the robustness of our results towards parameter value changes of ± 20% in a local sensitivity analysis. In three of the 64 new scenarios, the parameter change led to a change from intermingling to inhibition at contact due to a slight decrease in overgrown biomass. In all other cases, the competitive outcome remained stable (Supporting information).

In this study we aimed to gain a general understanding of the mechanisms behind changing competitive outcomes and did not want to reproduce the behaviour of specific fungi. For our simulation experiment, we varied different fungal traits. To derive a realistic range of trait values as a starting point, we first parameterised our model to match growth rates and stress responses experimentally observed in Wesener et al. (2021): we separately calibrated the diffusion coefficient D_{nj} regulating the extension of biomass, and the heat shock protein production rate δ_j, by using a differential evolution algorithm. The biomass diffusion coefficient D_{nj} was optimized to find the best match between the modelled extension rate and the observed extension under control conditions (22°C constantly) in isolation. The production of heat shock proteins δ_j was calibrated under stress conditions (a 2-h heat pulse of 45°C 1 day after inoculation) in isolation, to best correspond to the duration of the post-stress lag phase, i.e. the time it took the species to produce enough heat shock protein to restart growth. For further details regarding the experimental setup, see Wesener et al. (2021).

Simulation experiments

To thoroughly assess the parameter space, we investigated slow or fast extension of a focal species f (D_{nf} = 5 mm^2 day^{-1} or D_{nf} = 17.5 mm^2 day^{-1}, which equals an extension of 6 or 11 mm day^{-1}, respectively), and low or high heat shock protein production (δ_f = 0.02 mol^{-1} day^{-1} or δ_f = 0.05 mol^{-1} day^{-1}, which equals a lag phase of 2 days or 1 day, respectively). We let the focal species compete against a fungus k of variable extension rate and stress defence, with the trait range based on trait values observed in Wesener et al. (2021). We therefore varied D_{nk} from 3 to 21 mm^2 day^{-1} in increments of 2 mm^2 day^{-1} (leading to extension rates from 4 to 13 mm day^{-1}) and δ_k from 0.01 to 0.19 mol^{-1} day^{-1} in increments of 0.02 mol^{-1} day^{-1} (post stress lag phases from 4 to 0.5 days). We investigated both the production of diffusible and non-diffusible inhibitors (D_{ij} = 0 mm^2 day^{-1} and D_{ij} = 10 mm^2 day^{-1}, respectively) to include both an offensive strategy where a species actively exudes inhibitors, and a defensive strategy where a species produces intracellular molecules to fend off overgrowing competitors.

As model output, we determined the qualitative outcome of competition ten days after inoculation. In previous experiments, after this time the Petri dish was fully covered with fast-growing pairs (Wesener et al. 2021). This agrees with our simulation model. Also, after ten days of simulation, inhibitors have spread throughout the dish and stable interactions have formed. We differentiated between four competitive outcomes, namely inhibition at contact, inhibition at a distance, intermingling and overgrowth. Moreover, we determined the cover ratio under control conditions compared to a stress treatment for each species. Here, we differentiated between area covered by a single species and shared areas, which occur for the competitive outcomes overgrowth and intermingling. These metrics allowed us to assess the effect of heat stress on competition for space.

Determination of competitive outcomes

To define the competitive outcome, we first assessed the amount of shared area, i.e. grid cells that were occupied by biomass of both fungi. If no shared cells existed or if the amount of shared territory was less than 1% of the smaller fungus, we assumed the fungi to inhibit each other. We further defined the outcome as 'inhibition at distance' in those cases where there was a distance of more than 4 mm between the two fungi (Magan and Lacey 1984), and in other cases as 'inhibition on contact'. If the amount of shared territory was larger than 1%, intermingling or overgrowth took place. Intermingling is usually defined as neutral interaction in the periphery without adverse effect on either fungus (Stahl and Christensen 1992, Robinson et al. 1993), or as a special case of overgrowth (Choudhury et al. 2018). Here, we defined intermingling as slight overgrowth in the periphery, i.e. shared territory constituting 1–5% of the overall territory of the smaller fungus. For all cases of intermingling, we found a sufficient inhibitor concentration to prevent the fungi from growing further into the territory of the other fungi.

If more than 5% of the territory of a fungus was also occupied by its competitor, we defined the outcome as 'overgrowth'. Our model does not consider whether the overgrown biomass is still viable or not and assigns overgrown area to both competitors. We therefore do not differentiate between overgrowth and replacement.

Results

Under control conditions, competition resulted in intermingling or overgrowth in 93% of the investigated scenarios (points in Fig. 2, Supporting information). The growth strategy of both competitors influenced how much area would be overgrown, with the faster species being able to overgrow the slower species. If a species exuded inhibitors that diffused, it could prevent overgrowth by a faster competitor. For example, the focal species was overgrown in fewer scenarios when it produced diffusing inhibitors compared to no diffusion, even when its competitor grew very fast (compare yellow points in Fig. 2A and C). The same applies to the competitor which, when it grew slowly, could reduce overgrowth by a faster focal species through the production of diffusing inhibitors (compare redpoints in Fig. 2B and F). Slow-growing species could use diffusive inhibitors offensively to claim territory that the species

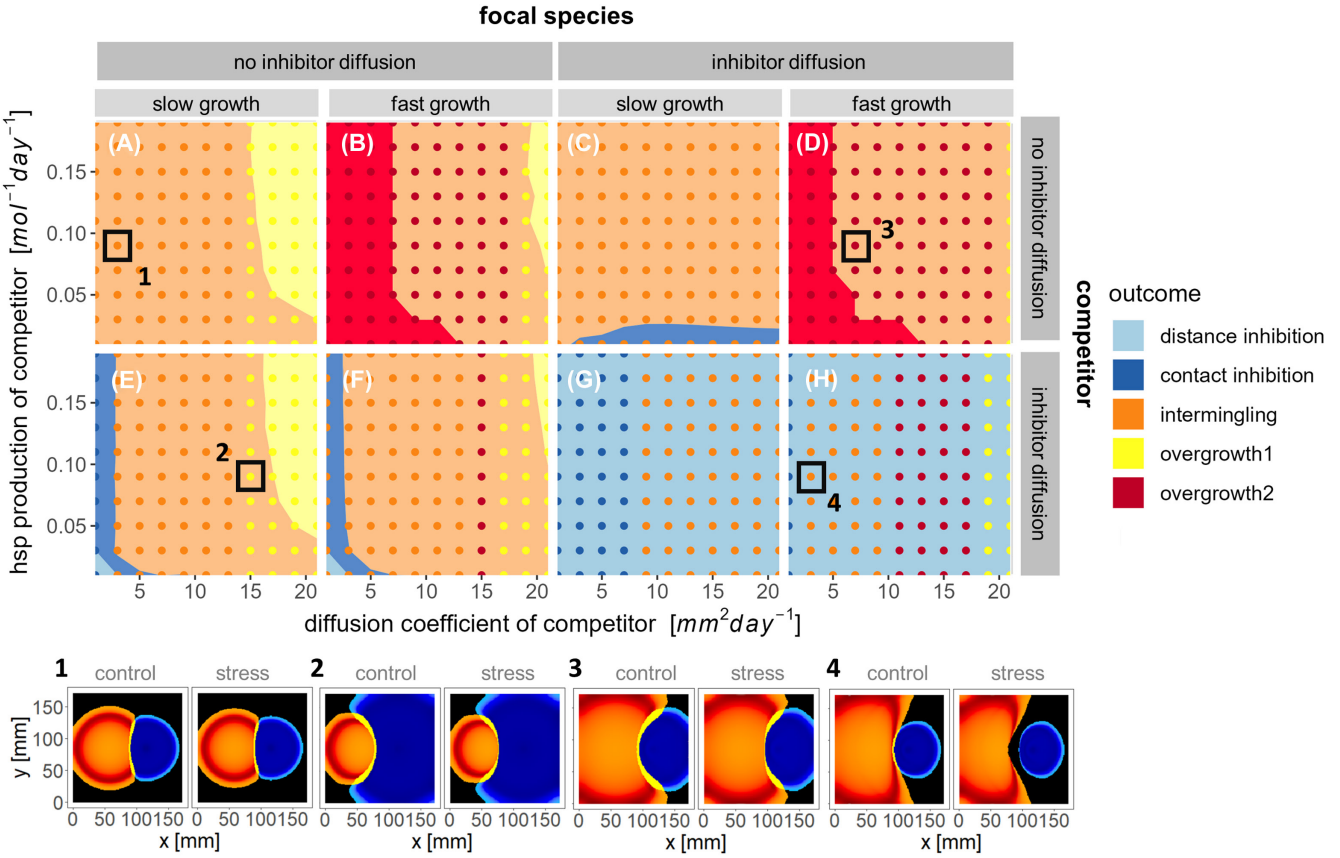

Figure 2. Competitive outcomes in dual culture under control and heat stress conditions ten days after inoculation. Competition for space is simulated between a focal species with intermediate heat shock protein production ($\delta = 0.05$, leading to a lag phase of 1 day) and slow growth ($D_n = 5$, leading to an extension of 6 mm day^{-1}) or fast growth ($D_n = 17.5$, 11 mm day^{-1}) and a competitor of variable growth and stress defence. Points represent the competitive outcome under control conditions, and the colour of the underlying area represents the outcome after a heat pulse. 'Overgrowth 1' describes outcomes where the focal species is overgrown, while 'overgrowth 2' refers to the competitor being overgrown. Numbered squares 1–4 show snapshots from the simulation to illustrate different heat effects on competing fungi ten days after inoculation: 1) no change of competitive outcome after heat stress, 2) a reduction of overgrowth of the slower focal species, 3) a reduction of overgrowth of the slower competitor and 4) emergence of distance inhibition.

had not yet grown on, and counter overgrowth of the faster species.

A heat stress pulse reduced overgrowth (45–14%) and led to an increase in inhibition at a distance (0–25%, compare points to underlying areas in Fig. 2). When both species produced diffusive inhibitors, competition under stress led to distance inhibition in all scenarios (compare Fig. 2G and H). Surprisingly, the growth strategy again influenced the competitive outcome under stress conditions, while increases in heat stress defence had no effect on the outcome if the heat stress defence was already high (for example, in Fig. 2B different outcomes can be observed along the x-axis; along the y-axis different outcomes are only apparent for heat shock protein production values ≤ 0.07 mol^{-1} day^{-1}).

Change in area under stress

To assess the benefit of a stress pulse on species under competition, we compared the territory covered by each species under stress to control conditions (Fig. 3). Even though its trait values were fixed within one panel, the benefit of the focal species was highly dependent on competitor trait values (Fig. 3A–D and I–L). Again, our results showed that a disruptive stress can be beneficial when competing against a fast competitor. Faster growth of the competitor increased the benefit of the focal species (Fig. 3C), and vice versa: the competitor benefitted most when the focal species was fast growing and reached highest values when the competitor itself was slow growing (Fig. 3N). The parameter space of beneficial stress effects for the focal species was reduced if its competitor exudes diffusing inhibitors (compare Fig. 3D and L), and vice versa (compare Fig. 3N and P).

The stress defence influenced how much territory a species could gain from stress but, as in the competitive outcome (Fig. 2), this influence was reduced for higher stress defences (for example, in Fig. 3C, an increase in *hsp* production only has an effect for low production values). Both species reached the highest benefit for low heat shock protein production rates of their competitor (Fig. 3, Supporting information).

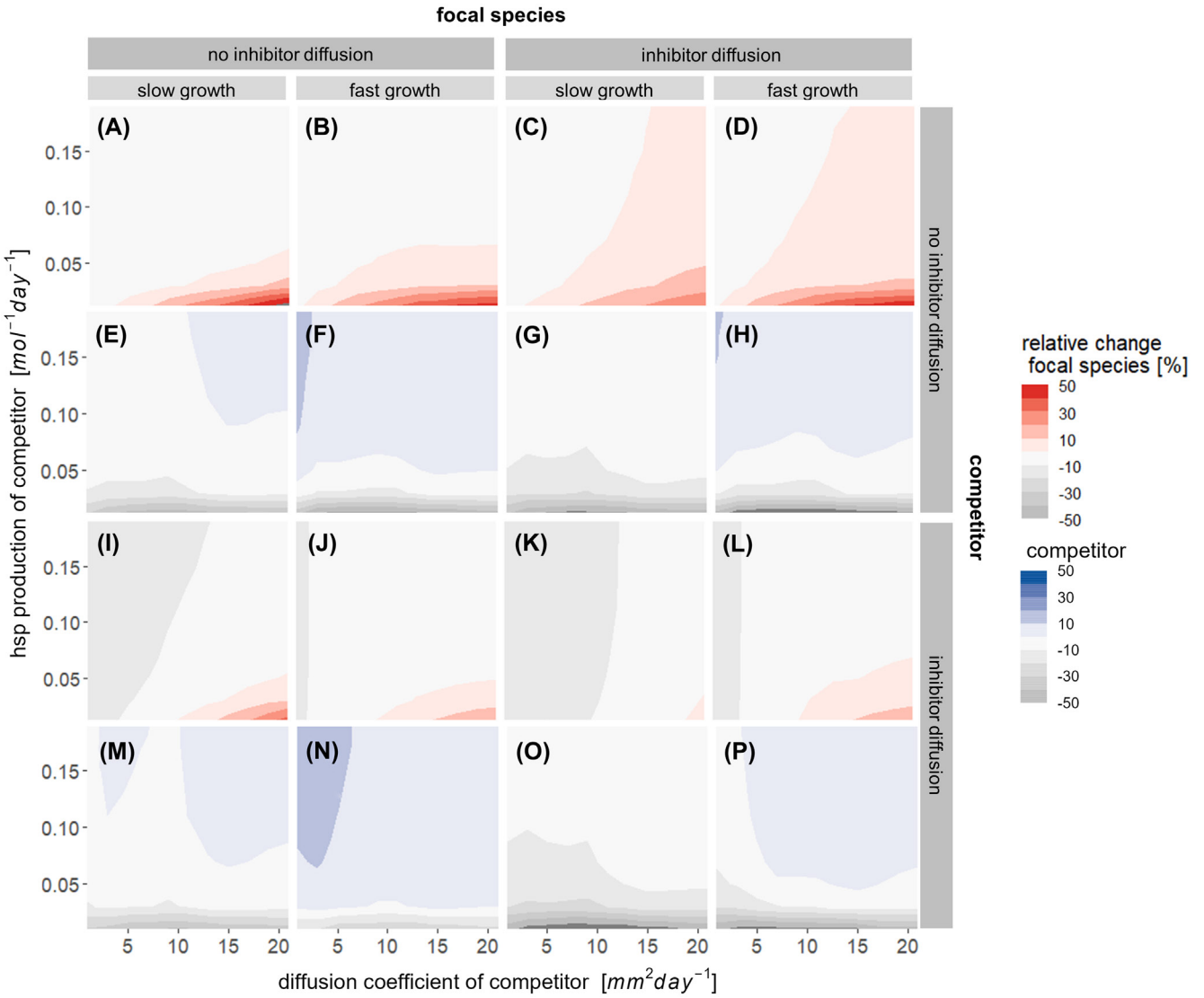

Figure 3. Effect of heat stress on the area of two competitors. Relative change of total area under stress treatment compared to control conditions ten days after inoculation of a focal species with intermediate heat shock protein production ($\delta = 0.05$, leading to a lag phase of 1 day) and slow growth ($D_n = 5$, leading to an extension of 6 mm day^{-1}) or fast growth ($D_n = 17.5$, 11 mm day^{-1}) and a competitor of variable growth and stress defence. Panels in the first and third rows describe the area change of the focal species, while panels of the second and fourth rows describe the area change of its competitor. Red and blue tones indicate a gain in area after stress for the focal species and its competitor, respectively, and grey tones indicate a loss in area.

The amount of shared area was reduced under stress conditions for all investigated scenarios (Supporting information), which is reflective of the reduction of overgrowth as competitive outcome. The production of diffusive inhibitors, in particular, had a large impact on the reduction of overgrowth.

Temporal dynamics of the inhibitor field

Even though competitive outcomes could change under heat stress, the temporal and spatial distribution of inhibitors did not change (Fig. 4). This discrepancy is caused by the lag in biomass extension. After a stress-induced lag, biomass reached territory later than under control conditions, i.e. at a point in time when more inhibitory compounds had already accumulated. This shows that, rather than the stress resistance of a species, the inhibitors that had been produced before the species start interacting influenced the resulting competitive outcome. That is, if a fungus had already produced enough inhibitors to suppress the other species, these could also be also effective during its phase of no growth.

Discussion

The PDE model could reproduce the typical response of a lag phase after a heat pulse and changing fungal competitive

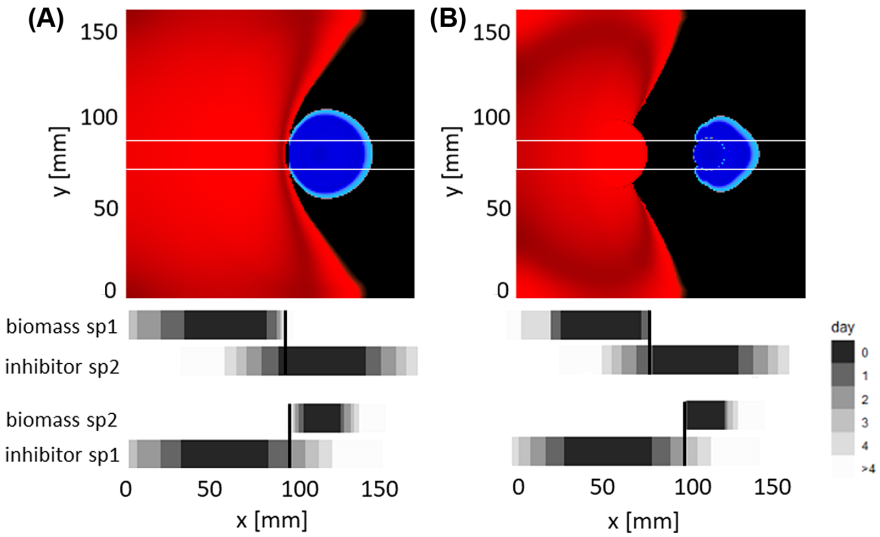

Figure 4. Inhibitor and biomass distribution on the horizontal axis ($y=86$) over time for inhibitor diffusivity $D_i=10$ (A) under control conditions and (B) under stress treatment. Square panels show the two-dimensional distribution of biomass of a fast and stress-susceptible species (sp1, red, $D_n=22$, $\delta=0.01$) and a slow and stress-resistant species (sp2, blue, $D_n=3$, $\delta=0.02$). Darker red and blue shades describe higher biomass density of the two respective species. The white lines frame the horizontal transect along which inhibitor and biomass distribution over time was observed. Horizontal bars show when inhibitory concentrations of antifungal compounds ($i=\psi$) and biomass concentrations of $b_{ni}+b_{ri}+b_{mi} > 10^{-1}$ reach a certain x coordinate. Darker shades indicate earlier days, i.e. the compound that has reached an x coordinate earlier is darker. Vertical bars show the x coordinate where biomass stopped growing because it had reached inhibitory concentrations of antifungal compounds.

outcomes under heat stress. We varied the production of antifungal compounds and heat stress proteins, the type of antifungal compounds produced and the extension rates of the competing fungi.

Our results showed that the change in competitive outcome, as well as the gain in area under heat stress conditions, differed greatly for different fungal response trait combinations (Fig. 2). In particular, if fast-growing species were present, the competitive outcome was likely to change, because they suffered relatively more from the stress-induced phase of no growth (reduction of overgrowth when fast species are present, Fig. 2D or E).

The production of extracellular inhibitors proved to be a successful strategy under stress, as they acted as heat-stable agents and could be used to claim territory (Fig. 3A versus C). For species of low heat resistance, particularly, an increase in heat shock protein production proved beneficial, as a reduction of a long lag phase granted time to counter faster species (Fig. 3N).

While slower species generally benefitted more, they were also more dependent on high stress defence to restart growth earlier than their faster competitors. Also, slower species benefitted more from diffusive inhibitors as they can defensively stop overgrowth by faster competitors (for example, a slow growing competitor benefitted in 2F versus 2B).

The effect of heat stress on competition and biodiversity

The results of this study suggest that induced defences favour less competitive, slower-growing species that may gain a head start to overgrow more territory or build up a defence. Because fast-growing species focus on rapid extension, a lag phase of no growth is advantageous for their slower-growing competitors, who may benefit from the interruption if they can revert to growth earlier or produce diffusive inhibitors. A heat pulse can therefore have an equalizing effect that promotes coexistence and increases the diversity of a community. Additionally, we found that overgrowth is reduced and intermingling between competitors was a more common outcome after a heat stress than under control conditions, as a species had additional time to accumulate antifungal compounds and to stop competitors from overgrowing them. When both species produced diffusive inhibitors, competition under stress led to distance inhibition in all scenarios. The inhibitors acted as heat-stable agents and could diffuse while fungi were lagged, leading to both species blocking each other from claiming free territory. The importance of the amount of inhibitors is corroborated by a study on fungal secondary metabolite production, in which a mutant with reduced secondary metabolite production was overgrown by its competitor (Knowles et al. 2019). Our study supports the finding that heat stress can favour biodiversity by decreasing the exclusion of community members, as competitive exclusion in spatially structured communities of sessile organisms is often the result of overgrowth (Maynard et al. 2017). We described several mechanisms that explain observed, positive effects of fluctuating environmental conditions on biodiversity in communities of fungi (Heilmann-Clausen and Boddy 2005, Toljander et al. 2006, Letten et al. 2018) and microbial communities in general (Jiang and Morin 2007, Nguyen et al. 2021).

In this study, we investigated short-term effects of heat stress pulse, and it is unclear how environmental fluctuations affect a community in the long term (Schimel et al. 2007). The competitive outcomes observed after a disturbance might change over longer periods of time (White and Boddy 1992, Hiscox et al. 2016, Jurburg et al. 2017), possibly caused by active degradation of antimicrobial proteins or nutrient depletion that affects competitive outcomes (Choudhury et al. 2018). These processes could be implemented in the existing PDE model, similar to another PDE model including biomass degradation through rivals (Boswell 2012).

Fungal competitive traits

We parameterised our model for theoretical species and varied response trait combinations in a full-factorial manner, resulting in a generalisable trait-based approach. In their review, Crowther et al. (2014) suggest that fungal combativeness is controlled by traits such as extension rate and toxic secondary metabolite production. The results of this study corroborate this proposition, as they show that a high extension rate granted dominance that could partly be countered by production of antifungal compounds. However, we found that a heat pulse might function as an environmental filter favouring the production of antifungal compounds. Environmental conditions are never static, and we therefore propose to include the ability to respond to environmental fluctuations in the set of traits influencing microbial competition.

Additional combative traits such as the ability to form barriers to keep competitors from overgrowing (White and Boddy 1992, El Ariebi et al. 2016, Hiscox and Boddy 2017) or the ability to degrade toxins of competitors (Hiscox and Boddy 2017) could be integrated into the model. These defensive strategies might benefit a species that can fend off inhibitors of antagonists and would thus decrease the efficacy of inhibitor production.

Other studies have used simulation models to examine which mechanisms are responsible for changing fungal competition, and found that nutrient content impacted competitive outcomes (Falconer et al. 2008, Choudhury et al. 2018) and biodiversity (Halley et al. 1994, Falconer et al. 2011). This study is the first to assess mechanisms responsible for changing interactions after a heat disturbance.

Priority effects

In our model, the timing of diffusion rather than the distribution of inhibitors determined the competitive outcome of interaction between competitors. This could explain why Hiscox et al. (2010) found no clear differences in enzyme activity for different competitive outcomes if looking at one point in time. Previous studies have described the effects of disturbances on priority effects (Tucker and Fukami 2014, Hiscox et al. 2016) and we identified induced defences as an important factor for fungal community assembly via priority effects in an earlier study (Wesener et al. 2021). Here, we additionally show that this influence is applicable to the expansion of biomass as well as the production of secondary metabolites.

In this study, we present the first PDE model that includes induced stress defences in a simulation of fungal growth. We successfully reproduced shifts of competition under heat stress and showed that this change was dependent on the interplay of inhibitor dynamics, induced heat stress response and fungal growth strategy. In recent years several studies have highlighted the importance of including species interactions into assessments of the impacts of climate change (Urban et al. 2013, Diamond et al. 2017, Engelhardt et al. 2020). Here, we provide an important step towards a mechanistic understanding of how induced heat stress defences affect species interactions and, vice versa, how competition between species influences the effect of heat on a community. The results of our study highlight the significance of fungal secondary metabolism and stress defence mechanisms as the basis of fungal interactions under changing climatic conditions.

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Author contributions

Felix Wesener: Conceptualization (equal); Data curation (equal); Formal analysis (lead); Methodology (lead); Validation (lead); Visualization (lead); Writing – original draft (lead); Writing – review and editing (equal). **Matthias C. Rillig**: Data curation (equal); Funding acquisition (equal); Writing – review and editing (equal). **Britta Tietjen**: Conceptualization (equal); Data curation (equal); Formal analysis (supporting); Funding acquisition (equal); Project administration (lead); Supervision (lead); Validation (supporting); Visualization (supporting); Writing – review and editing (equal).

Data availability statement

Data are available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.3j9kd51m9 (Wesener et al. 2023).

Supporting information

The Supporting information associated with this article is available with the online version.

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