

How can a pathogen infect humans and plants? The role of omics in deciphering the cross-kingdom behaviour of the fungus *Lasiodiplodia hormozganensis*

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FIGURE 1

Asexual spores of *Lasiodiplodia* spp. in different stages of maturation, young (colorless and aseptate) and mature (brown with 1 transverse septum).

FIGURE 2

Schematic representation of the effects of temperature on the fungus *Lasiodiplodia hormozganensis*. Adapted from Félix et al (2024) <https://doi.org/10.1016/j.scitotenv.2024.171917>

Back in the beginning of the XX century, the fungus *Lasiodiplodia theobromae* was described for the first time [1]. In 2008 [2], strain CBS 339.90 (the star of this story) was included in the species *L. theobromae*. It is a geographically widespread species, infecting nearly 500 plant hosts leading to disease and death. But CBS 339.90 was recognized as the causative agent of a human infection [3]. In early 2024 [4], we reassigned CBS 339.90 to the species *Lasiodiplodia hormozganensis*. Human body temperature is one of the main barriers preventing most fungi from infecting humans, so we investigated the effect of temperature on this cross-kingdom pathogen.

Genome analysis of CBS 339.90 revealed that it has the machinery to infect both plants and humans. At optimal temperature (25°C) or “human” temperature (37°C), different proteins & transcripts are expressed: at 25°C, proteins related to primary metabolism, while at 37°C, proteins related to pathogenesis. Notably, plant cell wall degradation proteins are more expressed at 25°C, while pathogenesis-related proteins dominate at 37°C.

L. theobromae strains isolated from plants induce mammalian cell death at 25°C, but only temporarily. In contrast, *L. hormozganensis* consistently causes 90% mammalian cell mortality during fungus growth [5]. *L. hormozganensis* alters its metabolism to cope with increased temperature, expressing lipids that decrease

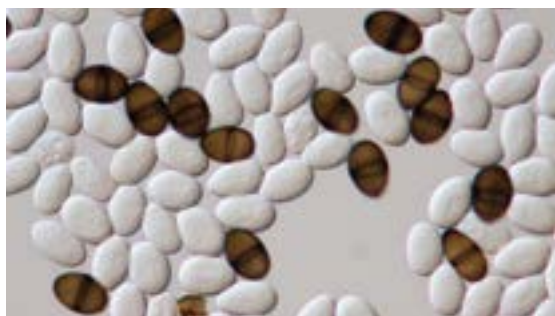
membrane fluidity at 37°C. But also, increased temperature leads to an increase in the accumulation of lipids known as essential players during fungal infection of humans.

L. hormozganensis, has the molecular mechanisms that allow it to infect both plants and humans, demonstrating its ‘cross-kingdom pathogen’ nature. We showed that higher temperatures lead to a higher expression of proteins and lipids related to pathogenicity.

As environmental temperatures rise due to climate change, fungal behaviour, particularly in the case of *L. hormozganensis*, may enhance human pathogenesis.

References

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Temperature modulates the expression of pathogenesis-related molecules

