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Short Communication

Aspergillus fumigatus Adaptation in a Warming Climate

Jonathan Sharma*

Department of Life Sciences, Avinashilingam Institute for Home Science and Higher Education for Women, Coimbatore, India

DESCRIPTION

Aspergillus fumigatus is a ubiquitous mold species found in soil, compost, and decaying vegetation. It thrives at temperatures up to ~ 50°C, with spores capable of surviving brief exposure near 70°C. Its innate thermotolerance has long been recognized as aiding its role as an opportunistic pathogen causing aspergillosis, particularly in immunocompromised individuals. As the global climate warms, habitats favorable for *A. fumigatus* are projected to shift [1]. Under high-emission climate scenarios (e.g. SSP5 8.5), this species may extend its range northward into cooler regions such as Scandinavia, Russia, Alaska, and parts of Canada and China-potentially increasing exposure to millions more people [2].

Modeling with species distribution tools like MaxEnt reveals that while *A. fumigatus* currently favors temperate regions of the Northern Hemisphere, warming trends will decrease its suitability in tropical zones and the Southern Hemisphere, and concentrate populations in cooler zones further north. This contrasts with *Aspergillus flavus* and *A. niger*, which currently dominate tropical environments but are expected to invade higher latitudes under global warming. Across global scenarios, suitable habitat for *A. fumigatus* could shift by up to 77% of land area by 2100, potentially exposing an additional ~9 million Europeans to inhaled spores [3].

Critical genes associated with high-temperature growth include *thtA*, required for viability near 50°C, and *cgrA*, which supports ribosome assembly at host-like temperatures (~37°C). Efficient ribosome biogenesis is central to rapid fungal growth under thermal stress, linking thermotolerance to pathogenic capacity [4]. Strain-level diversity influences temperature tolerance. Analyses show that strain mating type has minimal effect on thermal growth variability; however, reproductive capacity-particularly fertility in strains like AC3 4-may correlate with broader thermal adaptability. This may reflect a relationship between sexual reproduction at high compost-like temperatures (~65°C) and the selection of thermotolerant traits, though further genomic research is needed [5]. *A. fumigatus* has extremely high meiotic recombination rates (up to ~29 crossovers per chromosome), facilitating rapid dissemination of adaptive traits across populations and accelerating climate-driven adaptation.

Proteomics profiling during heat shock has identified dozens of differentially regulated proteins-including those involved in oxidative stress defense, translation, cellular signaling, and metabolism. Notably, *A. fumigatus* appears to downregulate carbohydrate metabolism genes at elevated temperatures while prioritizing chaperone synthesis for rapid stress response [6]. Additionally, accumulation of DHN melanin and pyomelanin within conidial walls enhances structural resilience against ultraviolet radiation, heat, oxidative agents, and antifungal compounds. Melanin also interferes with phagosomal acidification, promoting survival in host macrophages.

Climate-induced changes in precipitation, drought, and heavy rainfall impact soil moisture and compost dynamics, potentially triggering spore release events. Environmental disturbances like dust storms or extreme rains may facilitate episodic dispersal of *A. fumigatus*, heightening inhalation risks during sensitive climate periods. Currently, inhalation of *A. fumigatus* spores causes aspergillosis, with high mortality (up to ~90% in invasive cases) especially among immunocompromised individuals, such as transplant recipients, cancer patients, or those with respiratory conditions [7-9]. Projected climate-driven range expansion could expose millions more to infection risk. In Europe alone, ~ 9 million additional people may face elevated exposure while tropical declines occur elsewhere. Furthermore, rising thermotolerance and allergenicity intersect with antifungal drug resistance. Though primarily linked to drug pressure, enhanced thermal fitness may correlate with survival under treatment stress-complicating clinical outcomes [10]. Despite these threats, fungal pathogens remain understudied compared to bacterial or viral threats. The WHO has designated *A. fumigatus* as a critical fungal pathogen, and global funding for fungal research remains limited.

CONCLUSION

In a warming world, *Aspergillus fumigatus* stands out as a thermotolerant mold with the ability to adapt to higher temperatures, extend its ecological niche, and potentially cause more disease. Genetic mechanisms-including chaperones (e.g.

Correspondence to: Jonathan Sharma, Department of Life Sciences, Avinashilingam Institute for Home Science and Higher Education for Women, Coimbatore, India, E-mail: jonathan.Sharma08@gmail.com

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Hsp90), ribosomal assembly regulators (e.g. *cgrA*), and unknown thermotolerance proteins (e.g. *thtA*)-support its survival under heat stress. High recombination rates further enable rapid dissemination of adaptive traits. Addressing this threat demands improved environmental surveillance, genome-based monitoring of emergent thermotolerant strains, enhanced diagnostics, and investment in novel antifungal strategies that target thermal adaptation pathways. With climate change reshaping the patterns of fungal disease, understanding *A. fumigatus* at the intersection of ecology, evolution, and medicine is now more urgent than ever.

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