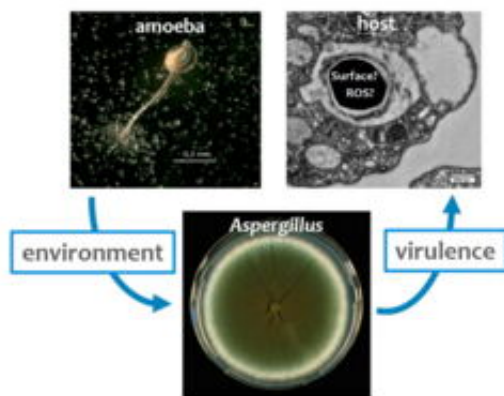


Until 2022



A large number of infections in humans and animals is caused by microorganisms that are actually considered to be harmless soil dwellers. Often their pathogenicity arises not only from the formation of special virulence factors, but rather results from complex interactions of the pathogen with the immune system of the infected host. How such microorganisms could have acquired properties to colonize or persist in the host is poorly understood.

Fungi such as *Aspergillus fumigatus* but also some yeasts can be regarded as such environmentally acquired pathogens. Their typical habitats include compost heaps or leaf litter, where they feed on decaying plant material. Nevertheless, they can cause severe systemic diseases in immunocompromised patients upon infection. In addition, the host range is rather broad and includes amphibians, as well as birds. What are the evolutionary driving forces behind general mechanisms to invasively colonize the host or resist the innate immune response?

We are analyzing to which extent fungivorous amoebae as naturally occurring predators could have exerted a selective pressure on fungi, which encouraged the development of virulent traits, e.g. the camouflage the cell surface by pigments or increased resistance to reactive oxygen species. Both are likely to be useful in the defense against natural predators and during interactions with cells of the immune system.

The research group [Miqwi](#) is integrated into the structural unit Evolution of Microbial Interactions. The group deals with the application-oriented projects to identify, isolate and characterise novel natural products from the secondary metabolism of amoebae-microbe-interactions.