they contain small pieces of food, this likely contributes to their chance performance in their choices between strips in a support problem task, even in the simplest configurations.

I should add that many of the problems encountered when comparing distantly related species may also apply to comparing members of the same taxonomic group or even the same species in different labs. Prosocial choices in chimpanzees, for example, seem to be highly sensitive to context and task. Even observational studies on different groups may reveal different results, as the post-conflict behaviour in chimpanzees has been shown to function as consolation for the victims of aggression in some groups, but as protection from redirected aggression in other groups. As flexibility is one of the key characteristics of higher cognitive systems, such variability should not be surprising. But understanding the contextual variation of given skills is everything else but simple, and seems likely to become a very hot topic in future research.

***Could it be that laboratory data are biased in one or the other direction, and thus do not provide the 'true' picture of corvid intelligence?*** Sure, but it depends a lot on what you mean by 'true' picture. Laboratory results give us insight into the mind of particular subjects tested under particular circumstances and with particular experimental histories. That's a 'true' enough picture, as long as we are careful in not over-generalizing to the entire species or even broader taxonomic groups on the basis of a few results. Unfortunately, this is what is often done. For instance, if one out of ten captive ravens solves a particular problem in a complex task, we may conclude that it is in the range of cognitive capacities of ravens and thus of corvids; however, it says little about how relevant this skill is for ravens, or corvids in general, under daily life conditions.

***How do you cope with this problem?*** For me personally, the best way to keep laboratory results in context is to also investigate the patterns of interest in the wild. Studying animals under field conditions is quite challenging but it gives us a richer idea of when and how abilities are actually used, and very often inspires set-ups and further questions for the lab. That is why I am particularly proud of the advances made at our field sites, where we have access to a population of about 220 individually marked ravens and almost 300 marked crows now. But both field sites are in human-influenced environments, so some of my colleagues are rightly questioning the generality of the findings obtained under those conditions. So we are back to the question of what is a 'true' picture, in this case what is the 'natural' environment for highly generalist feeders and scavengers like crows and ravens.

***You are a co-founder of the Department of Cognitive Biology: is studying animals in the wild what you mean with a biological approach to cognition?*** No, studying animals under field conditions is only one aspect of our approach: we also do plenty of lab work. Our general aim is to foster comparative, evolutionary thinking in cognitive research. While much has been achieved in this respect in the last decades, we see ample room for improvement. Most notably, many theories about the evolution of 'higher forms' of cognition remain relatively vague, and many core concepts are biased towards primates and/or constrained by definitions based on human standards. As you have probably guessed, we also strongly support truly fair comparisons, not only between non-human animals but also when comparing the abilities of animals with those of humans. This means, for example, testing humans without verbal instructions. And, of course, we aim to contribute to the integration of different approaches and to the bridging of fields, in my case combining the powerful testing paradigms of psychology with standardized ethological observations under daily life, and also non-invasive physiological measures such as hormone metabolites in saliva or feces. So we intend to promote a very integrative and inter-disciplinary approach to studying cognition and its evolution.

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**Quick guide**

# Dermatophytes

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***What are dermatophytes?***
Dermatophytes are fungal pathogens (…phytes) that cause diseases of the skin (dermato…). These fungi grow as filaments or hyphae, forming molds. Dermatophytes are the causative agent of cutaneous mycoses, including athlete's foot, ringworm, and nail infections (Figure 1). The scientific name of the disease is given by the word 'tinea' followed by the location of the infection; for example, *Tinea pedis* refers to athlete's foot and *Tinea capitis* refers to scalp ringworm. Dermatophytes are the most common cause of fungal infections worldwide, although the type of infection varies. Developed countries have a higher prevalence of athlete's foot, while developing countries have a higher prevalence of *Tinea capitis* and *Tinea corporis* (body).

The dermatophytes include three genera of molds in the class Euascomycetes — *Trichophyton, Microsporum*, and *Epidermophyton* — although the genera are not distinct within the phylogeny. The dermatophytes occupy three different ecological niches, classified as anthropophilic (human-associated), zoophilic (animal-associated) or geophilic (soil-dwelling). Species from all three niches are associated with clinical human disease.

***Why do dermatophytes cause so many different types of disease?*** All dermatophytes infect host surfaces containing keratin, including skin, hair, and nails. As such, the same infecting organism could cause disease in the foot, fingernail, body, or head. Both climate and lifestyle contribute to the prevalence of dermatophyte infections. Tropical climates and overcrowding predispose populations to dermatophyte infections. Increased urbanization, including the use of occlusive footwear, community showers, and participation in sports, has been linked to higher prevalence

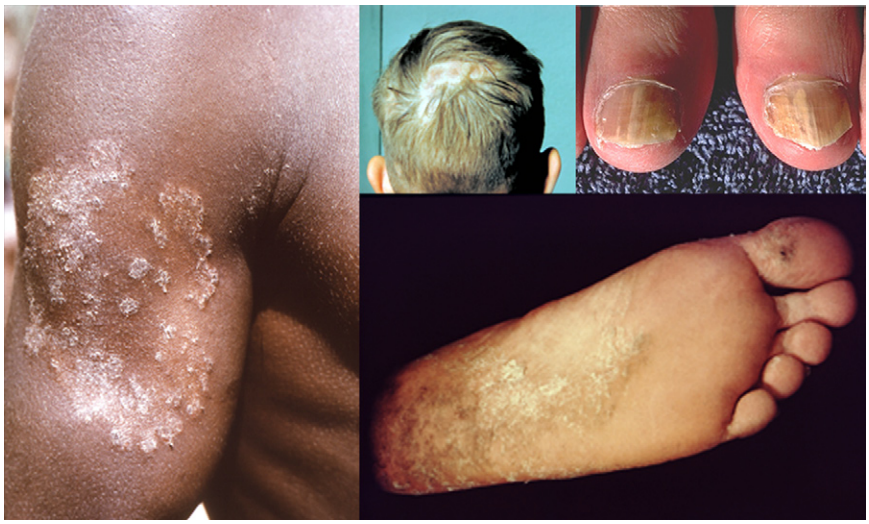

Figure 1. Skin diseases caused by dermatophytes.
Dermatophyte disease is named after the site of infection. Clockwise from left: *Tinea corporis*, *Tinea capitis*, *Tinea unguium*, and *Tinea pedis*. (Images courtesy of the Public Health Image Library.)

of *Tinea pedis* and onychomycosis (nail infections). It is very rare for a dermatophyte to cause an invasive or disseminated infection, although cases in immunocompromised patients have been reported.

Interestingly, disease severity correlates with the ecological niche of the infecting dermatophyte. Anthropophiles, such as *Trichophyton rubrum*, cause the most human cases and present as a chronic disease with mild inflammation. Nail infections in particular are difficult to cure and have a high rate of recurrence. In contrast, human disease caused by geophilic or zoophilic organisms present as a severe disease with acute inflammation and can be self-healing.

***How do dermatophytes cause disease?*** In order to cause disease, dermatophytes must adhere to a surface, such as epithelial cells, and then obtain nutrients for growth from these cells. The exact mechanism by which they do so has not been described in detail. It is known that dermatophytes secrete keratinases and other proteases, which are thought to play a role during infection; however, the temporal expression of these proteases appears to vary between species.

For chronic infection, such as that caused by anthropophiles, the immune system must be inhibited or avoided. It has been observed that epidermal keratinocytes respond differently to co-culture with a representative anthropophile than they do with a zoophile. The recent sequencing and analysis of several dermatophyte species provides a clue as to how dermatophytes might avoid the host immune response. However, the mechanism by which anthropophiles do this more effectively than zoophiles or geophiles remains to be discovered.

***How does the genome sequence help us understand dermatophyte virulence?*** The annotated genomes of several dermatophyte species have recently been published. As expected, the genomes of dermatophyte strains are more closely related to each other than to the related dimorphic fungi such as Coccidioides. Analyses of the dermatophyte genomes show an expansion of protease-encoding genes, including secreted proteases, in dermatophytes compared to related fungi. This fits with what is known about the ability of these fungi to grow on keratinized surfaces and highlights protein degradation as an important aspect of dermatophyte virulence. In comparison to other fungi, dermatophyte genomes contain a reduced number of genes involved in catabolism of plant sugars, as expected for fungi that are now specific to mammalian hosts.

The genome sequences also inform our hypotheses about which gene products play a role in virulence and niche adaptation. For example, genomic analysis shows an overall expansion of kinases in the dermatophytes. This suggests that signaling and regulation play a key role in adaptation to different environmental niches, as well as pathogenesis in the host. Clues as to how dermatophytes might interact with the host immune system were found in the genome sequences, since they are enriched with proteins containing LysM binding domains. These domains appear to mask surface proteins from the immune system. Because of this, proteins containing LysM domains have been implicated in the evasion of the host innate immune response in plant pathogens. Finally, dermatophyte genomes exhibit an expansion of genes involved in secondary metabolism. Some secondary metabolites from other species are involved in immune suppression, while others have antimicrobial activities. The dermatophyte secondary metabolites may play a role during infection in regulation of the immune system or in controlling secondary infections with other pathogens. With the completion of the dermatophyte genomes, researchers now have the tools to probe the role of specific gene products in the dermatophytes, and to elucidate their roles in pathogenesis and disease.

## *Where can I find out more?*

Achterman, R.R., and White, T.C. (2012). A foot in the door for dermatophyte research. PLoS Pathog. *8*, e1002564.

Achterman, R.R., and White, T.C. (2012). Dermatophyte virulence factors: identifying and analyzing genes that may contribute to chronic or acute skin infections. Int. J. Microbiol. *2012*, 358305.

Martinez, D.A., Oliver, B.G., Gräser, Y., Goldberg, J.M., Li, W., Martinez-Rossi, N.M., Monod, M., Shelest, E., Barton, R.C., Birch, E. *et al*. (2012). Comparative genome analysis of Trichophyton rubrum and related dermatophytes reveals candidate genes involved in infection. MBio. *3*, e00259–12.

Weitzman, I., and Summerbell, R.C. (1995). The dermatophytes. Clin. Microbiol. Rev. *8*, 240–259.

White, T.C., Oliver, B.G., Gräser, Y., Henn, M.R. (2008). Generating and testing molecular hypothesis in the dermatophytes. Euk. Cell *7*, 1238–1245.

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