Journal of Clinical Microbiology

Clinical Microbiology | Commentary

Nomenclature for human and animal fungal pathogens and diseases: a proposal for standardized terminology

Sybren de Hoog,[1,2] Thomas J. Walsh,[3,4] Sarah A. Ahmed,[1,2] Ana Alastruey-Izquierdo,[5,6] Maiken Cavling Arendrup,[7,8] Andrew Borman,[9] Sharon Chen,[10] Anuradha Chowdhary,[11,12] Robert C. Colgrove,[13] Oliver A. Cornely,[14,15] David W. Denning,[16] Philippe J. Dufresne,[17] Laura Filkins,[18] Jean-Pierre Gangneux,[19] Josepa Gené,[20] Andreas H. Groll,[21] Jaques Guillot,[22] Gerhard Haase,[23] Catriona Halliday,[24] David L. Hawksworth,[25,26,27,28] Roderick Hay,[29] Martin Hoenigl,[30,31] Vit Hubka,[32] Tomasz Jagielski,[33] Hazal Kandemir,[34] Sarah E. Kidd,[35,36] Julianne V. Kus,[37,38] June Kwon-Chung,[39] Shawn R. Lockhart,[40] Jacques F. Meis,[1,14,15] Leonel Mendoza,[41] Wieland Meyer,[34] M. Hong Nguyen,[42] Yinggai Song,[43] Tania C. Sorrell,[44] J. Benjamin Stielow,[45] Rachel Vilela,[41] Roxana G. Vitale,[46] Nancy L. Wengenack,[47] P. Lewis White,[48] Luis Ostrosky-Zeichner,[49] Sean X. Zhang,[50] on behalf of the ISHAM/ECMM/FDLC Working Group Nomenclature of Clinical Fungi

AUTHOR AFFILIATIONS See affiliation list on p. 14.

ABSTRACT Medically important pathogenic fungi invade vertebrate tissue and are considered primary when part of their nature life cycle is associated with an animal host and are usually able to infect immunocompetent hosts. Opportunistic fungal pathogens complete their life cycle in environmental habitats or occur as commensals within or on the vertebrate body, but under certain conditions can thrive upon infecting humans. The extent of host damage in opportunistic infections largely depends on the portal and modality of entry as well as on the host's immune and metabolic status. Diseases caused by primary pathogens and common opportunists, causing the top approximately 80% of fungal diseases [D. W. Denning, Lancet Infect Dis, 24:e428–e438, 2024, https://doi.org/10.1016/S1473-3099(23)00692-8], tend to follow a predictive pattern, while those by occasional opportunists are more variable. For this reason, it is recommended that diseases caused by primary pathogens and the common opportunists are named after the etiologic agent, for example, histoplasmosis and aspergillosis, while this should not be done for occasional opportunists that should be named as [causative fungus] [clinical syndrome], for example, *Alternaria alternata* cutaneous infection. The addition of a descriptor that identifies the location or clinical type of infection is required, as the general name alone may cover widely different clinical syndromes, for example, "rhinocerebral mucormycosis." A list of major recommended human and animal disease entities (nomenclature) is provided in alignment with their causative agents. Fungal disease names may encompass several genera of etiologic agents, consequently being less susceptible to taxonomic changes of the causative species, for example, mucormycosis covers numerous mucormycetous molds.

KEYWORDS nomenclature, fungal disease, proposal

Nomenclature of ascomycetous and basidiomycetous fungi has been dominated for more than a century by the system of separate names for sexual and asexual methods of propagation. These morphs often occur independently, for example, in nature or culture, and are enhanced by different requirements for growth and development. With the introduction of molecular taxonomy, the dual names for fungi were abandoned (1, 2) based on the "One-Fungus-One-Name" (1F1N) concept. Many stakeholders expected that nomenclature would be simplified and become more stable; however, that anticipated outcome has not always been realized. Unfortunately, full advantage has not yet been taken of adding names to the List of Protected Names of

Editor Romney M. Humphries, Vanderbilt University Medical Center, Nashville, Tennessee, USA

Address correspondence to Sybren de Hoog, sybren.dehoog@radboudumc.nl, or Sean X. Zhang, szhang28@jhmi.edu.

The authors declare no conflict of interest.

The views expressed in this article do not necessarily reflect the views of the journal or ASM.

Published 11 November 2024

Fungi, which when listed, are protected against any listed or unlisted competing names (3).

Nomenclature of medically important fungi has become a fiercely debated topic (4–8). Nomenclatural instability of medically important fungi has created uncertainty, prompting the establishment of an "international nomenclature committee for clinical fungi" as a Working Group of the International Society for Human and Animal Mycology (ISHAM) (6). It should be noted that taxonomy is a fundamental science, aiming to reflect biological relationships, with the ultimate goal of correctness of this representation, whereas nomenclature is a practical system to provide names for the units that taxonomy recognizes, so as to enhance communication between users, and where the ultimate goal is acceptance of a common language.

The uncertainty surrounding the name of the etiological fungus also affects the name of the disease, when it is directly associated with the fungal names. Nearly three decades ago, the dilemma regarding the impact of fungal name changes on the terminology of the associated disease was already acknowledged (9). We contend that a change in the name of the disease to align with the change in the name of a fungus would inaccurately suggest a change in the disease itself, potentially undermining communication among clinicians and clinical laboratories. Maintaining consistent language is crucial for diagnoses, epidemiology, statistics, and disease surveillance. To address this issue and to rectify this problem, Odds et al. (9) proposed the formula of "organism name [X] causing disease [Y]," a concept supported by another recent paper (10). Although this approach is rational, it neglects the common practice in medical mycology to refer to the prevalent common diseases with the generic name of the etiologic agent, the Odds system being used only for exceptional cases. This present paper aims to formalize this practice by (i) defining criteria for either choice (ii) fostering nomenclatural stability, (iii) providing a one health context, (iv) correlating with fungal pathogenicity, and (v) having medical and/or veterinary relevance.

Nomenclatural uncertainties

Impact of a nomenclatural change

The impact of a nomenclatural change of a fungus causing confusion in naming of the disease was raised by Denning (11) in describing the infections caused by former *Pseudallescheria* species, which previously were often termed "pseudallescheriasis" or "pseudallescheriosis." Since the name of this genus was changed to *Scedosporium* (12), the name "scedosporiosis" would be a more appropriate replacement. Judging from discussion among members of the ISHAM Nomenclature Working Group, the proposal to reallocate *Candida glabrata* to *Nakaseomyces* (13) seems to face particularly significant resistance from clinicians and clinical laboratory communities, primarily due to concerns regarding coherent public health policies (14) and disease tracking. Furthermore, there are cases where the disease name no longer accurately reflects the causative fungus. For example, "geotrichosis" has historically been used to describe infections caused by *Geotrichum* species (15) but localized infections by the generic type species, *G. candidum* (16), are exceedingly rare. The preponderant disseminated infections referred to members of this genus are caused by *G. capitatum* (17), which is now classified as *Magnusiomyces capitatus* (18). Despite this taxonomic reclassification, the disease name has not been updated, resulting in a discrepancy between the disease name and its etiologic agent.

Disease names may comprise very different disease entities

Another problem is that disease names may comprise very different disease entities, even across kingdoms, such that the name becomes practically uninformative other than the generic affiliation of the etiologic agent. For example, "cladosporiosis" has been applied for fatal human brain disease by *Cladophialophora bantiana* (19) as well as for a tomato disease by *Fulvia fulva* (20), and orthographically refers to the ubiquitous and

clinically insignificant saprobe, *Cladosporium*. A rational, informative system to name diseases in humans has long been codified in the International Classification of Diseases (ICD, www.who.int/standards/classifications/classification-of-diseases/).

The principle that has enabled "1F1N" to be widely adopted, namely DNA sequencing, also determines the current, prime method of classification: molecular phylogeny. It is essential to recognize that fungi with similar morphology can be phylogenetically unrelated. When two species are phylogenetically remote, their behavior is likely to be different, and they should not be classified in a single genus—which would erroneously imply behavioral and clinical similarity; a pertinent example is the diverse array of species within the traditional circumscription of the genus *Candida*. Of note, close kinship does not guarantee similar behavior. Molecular classification became general laboratory practice around the year 2000 when already over 120,000 species were known (21) of which only a fraction have been sequenced thus far. This implies that a significant task of reevaluating existing classifications lies ahead of us.

The reassessment of fungal nomenclature is currently facilitated through molecular phylogeny, serving as the initial step in delineating relationships. This approach has yielded significant benefits, particularly elucidating morphological genera such as *Sporothrix,* which contained ascomycetous as well as basidiomycetous members (22), and in the case of *Madurella* where classical members were found across different ascomycete subclasses (23). An extreme example of reallocation is *Calcarisporiella* from Ascomycota to the Mucoromycotina (24). In some cases, however, the comparative taxonomic approach impedes the phylogenetic method. Data sets by definition are subject to sampling effects, wherein the inclusion of species in analysis is contingent upon their perceived closeness to enable accurate tree reconstruction. When trees contain different members to show the relationships, the clade structure may differ. This phenomenon is particularly pronounced in areas with less practical significance that tend to be understudied, and where the discovery of new relatives can lead to considerable taxonomic instability over time.

Suggestions to prevent unnecessary changes of fungal names

The reallocation of species into different genera is meant to increase nomenclatural stability. However, as an inevitable outcome of scientific advancement, further changes will be proposed in years to come especially as undescribed species are discovered. Proposals have been made that could mitigate the impacts of taxonomic shifts, particularly concerning well-established infectious entities. One approach is to maintain large genera, thereby reducing susceptibility to reallocations solely based on narrow cladistic principles (25), a strategy also advocated for some large plant genera (26). A crucial initial step is to define the reference point for each genus, that is, the type species, defined by a type specimen. In bacteriology, the type should ideally be a living type strain, allowing for analysis with modern technology (27). However, the lack of appropriate type material is a common challenge (28). In such cases, efforts are made to redefine the reference species by neo- or epitypification, that is, depositing new reference material when the original is lost or does not show one or more diagnostic features (29, 30). Alternatively, genera lacking such reference material cannot be meaningfully defined and consequently discarded due to doubts regarding their identity (31). The International Code of Nomenclature for algae, fungi, and plants (3, 32) now provides mechanisms for the protection of well-known names at the rank of family and below for fungi, not only against any competing names but any name that may emerge in the future that might threaten them. These proposals came into effect in 2017 and are evaluated by specialized Working Groups of the International Commission for the Taxonomy of Fungi (ICTF) and ratified by the Nomenclature Committee for Fungi (NCF) (33). If proposed changes are adopted, they are implemented by internationally recognized databases including MycoBank (www.mycobank.org), Index Fungorum (www.indexfungorum.org), and Fungal Names (www.nmdc.cn/fungalnames).

For species names, the International Code of Nomenclature for algae, fungi, and plants, subject to various exclusions, is based on priority of publication, that is, the oldest validly published legitimate name is to be used, regardless of whether this is based on a sexual or an asexual morph. This provision dates from 2011; prior to 2011, only sexually and asexually typified names were applied to the morph they represented. When this change was implemented, provisions to reduce the number of name changes were made by a mechanism of "protection." This meant that when a well-known sexually typified name was jeopardized by older asexually typified synonyms, they could be put on lists established by a series of Working Groups of the ICTF. For example, *Hyphopichia burtonii*, based on *Pichia burtonii* 1965, is preceded by *Sporotrichum anglicum* 1937 and *Trichosporon behrendii* 1952. In this case, if *Hyphopichia burtonii* was on a protected list, the name could be retained. Likewise, *Pichia kudriavzevii* has older competing synonyms: *Candida krusei*, *Monilia inexpectata,* and *Trichosporon dentriticum*; in all these cases, protection of the scientifically correct and clinically meaningful name is necessary. Protected names are safeguarded against any others that might compete, whether listed or unlisted by the Working Group. This possibility only exists for fungi and is stronger than the process of conservation. A conserved name is safeguarded only from the names listed as its synonyms. The conservation process requires a lengthy procedure including publication in *Taxon*, which needs to be ratified by the next International Botanical Congress, as was the case for the protection of the name *Coccidioides posadasii* against seven older listed synonyms (25).

Nomenclatural stability and One Health

One Health is a collaborative multidisciplinary approach to achieving optimal human, animal, and environmental health outcomes by understanding and optimizing medical, veterinary, zoological, agricultural, and botanical health within a shared environment (https://www.cdc.gov/one-health/about/?CDC_AAref_Val=https://www.cdc.gov/onehealth/basics/index.html). Integrating nomenclatural stability into the One Health approach, particularly within the context of zoonotic diseases, is essential for enhancing global health security. Zoonoses represent a significant public health threat and are a key indicator of One Health, which underscores the interconnectedness of human, animal, and environmental health. Maintaining consistent taxonomic names across disciplines facilitates the accurate tracking and communication of disease outbreaks, enhancing the effectiveness of One Health initiatives aimed at disease prevention and control, particularly in the light of the efforts conducted by the World Health Organization (www.who.int/news-room/fact-sheets/detail/one-health) and the World Organization Animal Health (https://doc.woah.org).

Recent studies, such as those developing the One Health Index on Zoonoses (OHIZ) and the Global One Health Index (GOHI) (34–36), highlight the importance of establishing a robust framework for evaluating One Health performance, particularly in the management of zoonotic diseases. These indices incorporate a variety of indicators, including source of infection, route of transmission, and environmental factors like air quality and climate, which are crucial for modeling the spread of diseases. The stability in species nomenclature would directly support these efforts by ensuring that data pertaining to these indicators are reliable and comparable over time and across regions.

Furthermore, the creation of evaluation tools and the inclusion of nomenclatural stability within these tools can help identify gaps where One Health capacity building is most urgently needed. For instance, the GOHI integrates various health dimensions, including human, animal, and environmental health, using a comprehensive indicator framework; however, this and other indicators do not yet explicitly integrate or recognize standardized nomenclature and disease naming as a standalone variable (34, 37, 38). Stable taxonomic and disease names enhance not only the utility of future indicator systems but must be considered an essential component of such, ensuring that indicators used are based on unequivocal and widely recognized species identifiers. Therefore, promoting nomenclatural stability aligns with the objectives of One Health

by enhancing the clarity and consistency of data, which, in turn, supports the development of effective policies and practices. One Health initiatives could more effectively utilize global indices and frameworks to measure performance and implement strategic interventions across interconnected health domains.

As a result, a revised naming system for infrequent opportunistic pathogens not only supports the objectives of One Health but also addresses the need for precise and actionable health data. This approach promotes a unified understanding of disease dynamics across various health domains, ultimately contributing to improved health outcomes and enhanced global health security. By adopting a naming convention that combines the causative agent with the clinical syndrome, the scientific community can ensure that communication is accurate and that disease management strategies are effectively implemented across all relevant sectors.

Types of pathogenicity

Importance of a stable nomenclature for fungal diseases

Informative classification and stable naming of fungi are essential for a stable nomenclature for fungal diseases. The disease should encompass a recognizable clinical syndrome, allowing the name to cover specific features. Broadly, two types of pathogenicity may be distinguished in medical mycology (Fig. 1); that is, primary pathogenic infections in healthy hosts, where the fungus plays the major role in the course of pathogenesis, and opportunistic infections where the disease is mainly dependent on host factors such as immune status, on the portal of entry, or a combination of both. In general, in primary pathogens, host infection is part of their life cycle, while opportunistic pathogens naturally occur on other substrates and are found on animal/human hosts incidentally, for example, by trauma, as transient colonizers, or when the hosts are immunocompromised. Most primary pathogens occur preferably in non-human hosts. They may be asymptomatic in their preferred animal host, like *Malassezia* species that assimilate cutaneous host's products but not the host itself, and zoophilic dermatophytes in cat fur. Another example of a principally non-pathogenic organism is provided by the unculturable genus *Pneumocystis,* of which its species can colonize the respiratory tract of humans and other mammals, with high degrees of host-specificity, and showing signs of co-evolution (39, 40). In humans, exposure to *P. jirovecii* is frequent and can lead to pulmonary colonization but its behavior may change depending on the conditions of the host: *P. jirovecii* can cause fulminant pneumonitis (usually referred to as pneumonia) if a person becomes immunocompromised; thus, it is considered as an opportunist (41).

In recognition of the complex pathogenesis of *Pneumocystis*, there is evidence for colonization, latent infection, subclinical infection, and *de novo* inhalation from environmental sources leading to a dichotomous outcome of eradication of the organism in healthy animals and infection in immunocompromised hosts (41–45). These different aspects of its pathogenesis are nonetheless consistent with *Pneumocystis* as an opportunistic fungus that can cause severe and lethal respiratory infections in immunocompromised hosts.

Host-dependent disease manifestations

Fungi also tend to behave differently in humans compared to their natural, non-human host. For example, the AIDS-associated fungus *Talaromyces marneffei* causes no symptoms in bamboo rats (46). Geophilic- and zoophilic dermatophytes tend to be asymptomatic on animal hosts but are often highly inflammatory when infecting a human (47). While host-fungus interactions demonstrate numerous variations, whether animals play a role in the fungus' natural lifecycle seems a firm criterion of primary pathogenicity.

From the fungal perspective, primary pathogenicity is a means to exploit and expand. In this scenario, a vertebrate host can serve as a way of increasing fungal dispersal and survival. A prime facilitator of adaptation is the transmission of the fungus to a subsequent host, following infection. This can take place either (i) directly, as in host-to-host

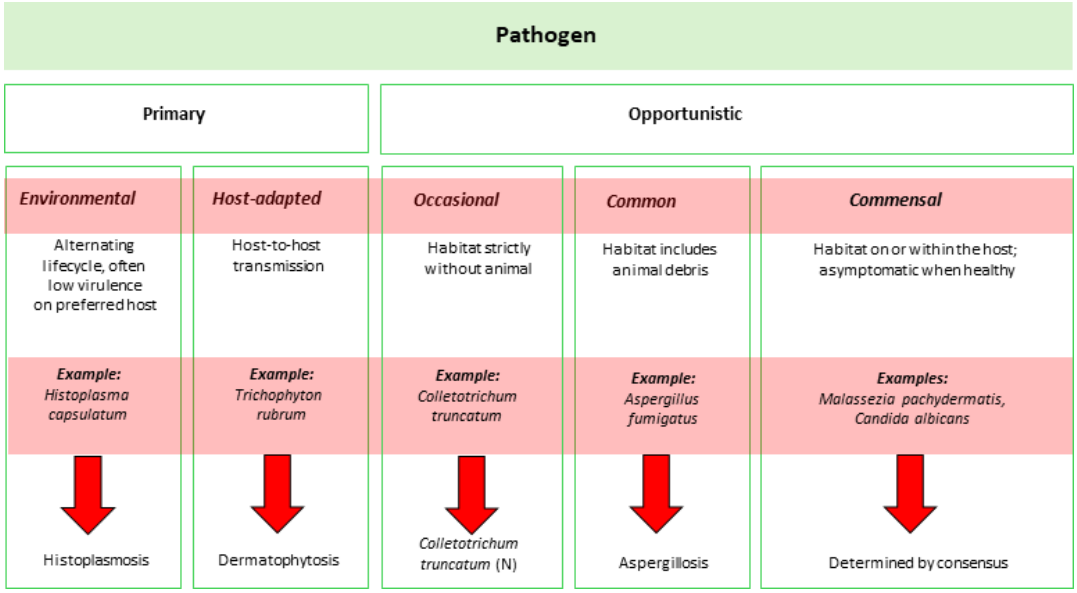

FIG 1 Types of pathogenicity of fungal pathogens in medical mycology.

contact transmission of, for example, anthropophilic dermatophytes, which constitute one of the very rare groups of human-adapted pathogens (48), or (ii) indirectly, inhaled *via* the environment, as with *Histoplasma* and related dimorphic pathogens (mostly having non-human preferred host animals and are known as environmental pathogens, Fig. 1). Third, (iii) opportunistic fungi have an environmental habitat (Fig. 1). In the latter case, human infection in immunocompetent hosts is accidental, the lesion usually following trauma and remaining localized, as for an example in chromoblastomycosis. The infection may become invasive when the host defense is breached or compromised; Sobianski Herman et al. (49) recently suggested that patients with severe chromoblastomycosis may carry a CARD9-related inherited disorder. The fungi of the category (iii) presumably die with their hosts (although no hard data are available to confirm this) and thus transmission would be unlikely. In this case, opportunistic infections are detrimental to the fungal population since the infecting fungal cells are lost for reproduction. Primary pathogens that follow infection pathways (i = direct) and (ii = *via* the environment) have a specific, relatively consistent relationship with their host, while with opportunists (iii = preferred environmental habitat) the disease may be more variable. It may be noted that obligate pathogens unable to produce an assimilative thallus outside the host, such as the frog pathogen *Batrachochytrium dendrobatidis*, are extremely rare among fungi, as most species can also thrive in the environment.

Examples of host-dependent disease manifestations

Paracoccidioides spp.

Pathogenicity as described in categories (i) and (ii) (Fig. 1) leads to particular clinical syndromes in the preferred host. An example of types of pathogenicity suggestive of different disease names applies to an unculturable *Paracoccidioides* species, formerly known as *Lacazia loboi*, restricted to South America. This species was recently found to comprise two unculturable cryptic species, *P. lobogeorgii,* infecting human subcutaneous tissues, and *P. ceti*, causing subcutaneous infections in dolphins (50). The traditional disease name for the infections caused by culturable *Paracoccidioides* species (*P. brasiliensis* and relatives) that are clinically very different from those caused by the unculturable species is paracoccidioidomycosis, and therefore the investigators proposed the disease names "paracoccidioidomycosis lobogeorgii (PCML)" for the

human disease, and "paracoccidioidomycosis ceti (PCMC)" to denote the disease in dolphins.

As a caveat, we also underscore that clinical manifestations of a given invasive fungal disease may vary as a function of a pathogen's inter-strain genetic variability and differential host responses depending upon the type of immune impairment, immunogenetics, and pharmacological immunosuppression.

Aspergillus spp.

In practice, the above distinction is less obvious, particularly with the most common opportunistic fungal pathogens, *Aspergillus fumigatus* and *Candida albicans*, where the host/fungus interaction is much more complex. *A. fumigatus* can cause invasive pulmonary aspergillosis, which is chronic or acute if the disease is manifested in less than 1 month and subacute if 1–3 months. Many patients are demonstrably immunocompromised, but certain numbers of patients in intensive care units following viral and other infections (e.g., influenza, SARS-CoV-2), or with chronic obstructive pulmonary disease (COPD) are not classified as being classically immunocompromised. Invasive aspergillosis usually affects the lungs, but sometimes the paranasal sinuses with corresponding terms of invasive pulmonary aspergillosis and invasive *Aspergillus* rhinosinusitis. Dissemination from the lungs to the brain, frequently in severely immunocompromised patients, leads to "cerebral aspergillosis" which manifests as one or more acute abscesses, areas of hemorrhagic infarction, meningitis, mycotic aneurysm, and cerebral granuloma. Dissemination is also reported from almost every other organ of the body, including the skin (cutaneous aspergillosis), thyroid (*Aspergillus* thyroiditis), heart valve (*Aspergillus* endocarditis), and other locations. Because the unifying disease pathology is demonstrable, or inferred, the term "invasive aspergillosis" is universally used, regardless of disease entity.

A. fumigatus also causes disease in immunocompetent patients. In patients with impaired lung tissue architecture, thus resembling the traumatic opportunistic scenario, it causes various forms of chronic pulmonary infection, *Aspergillus* (tracheo)bronchitis or *Aspergillus* nodule following long- or short-term colonization. It can be involved in onychomycosis, keratitis, and postoperative infections as a result of contaminated air-conditioning in hospitals. In patients with hypersensitivity and an over-reacting immune system, *Aspergillus* can cause allergic manifestations, granulomatous sinusitis (though usually *A. flavus*), fungus ball of the maxillary of sphenoid sinuses, and otitis externa (most often *A. niger* complex). In cystic fibrosis and COPD patients, the fungus causes allergic bronchopulmonary aspergillosis (ABPA), *Aspergillus* bronchitis, and *Aspergillus* sensitization, all linked to worsening lung function. *A. fumigatus* is the most common fungal allergenic species and is the most common "allergen" linked to severe asthma. Repetitive occupational exposure to *A. fumigatus* can cause "farmer's lung" and to *A. clavatus* "malt worker's lung," two of many fungal examples of hypersensitivity pneumonitis (extrinsic allergic alveolitis).

In summary, from the patient's perspective, *A. fumigatus* can be classified as (i) an opportunistic pathogen in terms of immune dysfunction (the most usual meaning), causing serious and lethal infection in infected, damaged, and susceptible tissue, (ii) a superficial pathogen of the airways with minimal invasion, (iii) a long-term colonizer of the upper and lower respiratory tract without causing infection, and (iv), an immune sensitizer. In the environment, its natural habitat is in self-heated decaying organic matter (51). Supposing a nutritional similarity to pulmonary debris, this might explain the prevalence of the species in human infection. With respect to its saprobic character, *A. fumigatus* is listed as a common opportunist, with the disease name aspergillosis when infection follows inhalation. Cases of implantation or localized lesions do not follow this route and are therefore not named after the fungus.

Candida spp.

Candida albicans is also a pleiotropic opportunistic pathogen and colonizer. As a normal component of the gut microbiome and frequent colonizer of mucosal membranes, it may be present for years without ever causing disease or perhaps even being beneficial; this is the situation in the majority of people. Medical self-experimenters demonstrated that oral consumption of 10^{12} *C. albicans* cells enabled immediate translocating from the intestine to the bloodstream, but remained limited and without known organ involvement (52). Certain conditions associated with severe illness, chemotherapy, or use of antibiotics lead to translocation through the gut mucosa, resulting in candidemia, and invasive candidiasis involving other organs. Candidemia may also occasionally follow catheter infection with its associated biofilm (catheter-associated candidemia). Transit of *C. albicans* from the blood through the kidney can lead to positive urine cultures, but urinary tract infection may also occasionally follow ascending infection through the urethra (both called urinary candidiasis). Once in the bloodstream, any organ can be infected with the more common demonstrable localization being bone, the eye, and heart valves (*Candida* osteomyelitis, *Candida* endophthalmitis, and *Candida* endocarditis, respectively), or rarely cerebral infection (53). Intra-abdominal candidiasis is a catch-all phrase covering intra-abdominal abscesses, secondary peritonitis, and occasionally primary peritonitis, infected pancreatic necrosis and cholecystitis/cholangitis and complicating chronic ambulatory peritoneal dialysis. However, the vast majority of *C. albicans* infections are mucosal—oral, esophageal, and vaginal and rarely in the bronchi (oral and esophageal candidiasis, vulvovaginal candidiasis, and *Candida* bronchitis). Other specific manifestations of oral candidiasis include angular cheilitis, median rhomboid glossitis, chronic hyperplastic, and atrophic candidiasis/denture stomatitis, the last linked to denture biofilm formation. Cutaneous infection with *C. albicans* is also common in infants in the crural area (nappy rash, or diaper dermatitis), and in adults in moist intertriginous areas (axillae, under breasts, toe webspace, groin), also known as intertrigo. *C. albicans* is also a cause of fungal keratitis, often linked to contact lens wear, keratoplasty, and xerophthalmia, as well as being found in diabetic foot ulcers and otitis externa, typically associated with other microorganisms.

In summary, *C. albicans* and many other *Candida* spp. are (i) frequent, if not universal, colonizers of the normal human gastrointestinal tract and mucosal membranes, (ii) causes of bloodstream infection, with involvement to almost all organs, (iii) a cause of abdominal cavity infection, usually linked to gut wall damage or perforation, (iv) common mucosal opportunistic pathogens of the mouth and vagina, or (v) a cause of cutaneous and ocular infection. Supposing a main lifestyle as an internal commensal, infections upon any change in host immunity may lead to infection; *Candida albicans* is therefore classified as a common saprobe, with its frequent tissue invasion after commensalism therefore being referred to as candidiasis. There needs to be clarity if bloodstream infections caused by taxonomically reallocated, previous "*Candida*" species, should be referred to as fungemia or candidemia and if this complicates treatment and surveillance. Cases of superficial infection (i.e., keratitis, otitis) do not follow the commensal route and are therefore not named after the fungus.

Diversity of fungi and disease

Looking forward, a revision of the taxonomy of the ascomycetous yeasts causing candidiasis is under consideration. We suggest that the term candidiasis (and its various forms as described above) should be preserved, regardless of any generic name changes, if that agent causes a significant disease burden currently labeled as candidiasis (Table 1). The medical, public health, and coding arguments in favor of this are set out in detail for *C. glabrata* (14), recently proposed to be re-classified as *Nakaseomyces glabratus*, and would certainly apply to *C. auris* (recently proposed to be re-classified as *Candidozyma auris* (54)), *C. albicans*, *C. tropicalis,* and *C. parapsilosis* as well as to *C. krusei* (*Pichia kudriavzevii*). The clinical umbrella syndrome can be referred to as candidiasis. The same principle would apply to the agents of *Fusarium*, where well-established disease

terminology remains in place despite controversial taxonomic systems being discussed. The principle is already practiced with mucormycosis which covers all relevant genera within the Mucoromycotina where significant differences in antifungal susceptibility are also noted (31), as explained below.

Judging from the foregoing concepts, diseases caused by primary fungal pathogens are likely to be more consistent and predictable than those caused by opportunistic fungi—with the notion that this bipartition is not strict, and some of the major invasive fungi are difficult to categorize. Although several *Candida*-like, *Aspergillus*, *Cryptococcus*, and mucoralean species are opportunistic, they cause distinctive patterns of disease in immunocompromised patients. To a certain extent, human infection by these environmental fungi might be in accordance with their natural behavior. As a compost fungus, that is, a degrader of bio debris at elevated temperatures, the thermotolerant *A. fumigatus* may find in damaged human lungs a habitat that is sufficiently close to its natural niche, albeit in degenerate form. This differentiates the common opportunists from the occasional opportunists. Similar reasoning may be applied to *C. albicans* colonizing the gastrointestinal tract and mucocutaneous tissues, and to *P. jirovecii* transiently colonizing the lungs, both naturally being colonizers in healthy individuals rather than pathogens. Invasive candidiasis, acute pulmonary aspergillosis, chronic pulmonary aspergillosis, cryptococcal meningitis, *Pneumocystis* pneumonia, and rhinocerebral mucormycosis are well-established clinical entities. Diseases such as chromoblastomycosis or sporotrichosis are not unambiguously characterized as either primary pathogenic or opportunistic (55). Following earlier papers by Casadevall and colleagues (56, 57), it was surmised that the fungi had shifted to another advantageous habitat, that is, the animal. The distinction between diseases caused by primarily pathogenic fungi *versus* those caused by accidental opportunists is obvious, but the largest category in clinical practice is the intermediate group of "common" opportunists.

Practical application

This categorization might be used to build a nomenclatural system for fungal diseases. If infection by a primary pathogen is a relatively invariant clinical entity, it may be named after the infecting agent as a neologism, for example, histoplasmosis. In most opportunistic infections, this is less informative because of the variable clinical patterns. Consequently, it will be necessary to define which fungi are designated as (i) primary pathogens according to the above definition, (ii) common opportunists, and (iii) coincidental opportunists (Fig. 1). Infections by environmental fungi that are very frequent among susceptible host populations have a relatively consistent pattern, such as pulmonary infections by *A. fumigatus* (Table 1), but the clinical manifestations of infections by, for example, *Paecilomyces variotii* or *Trichosporon asahii,* are highly variable (31) (Table 2). The descriptor "common" is certainly skewed by the fact that some fungi are ubiquitous; this is determined by clinical practice rather than scientific correctness.

Fusariosis and aspergillosis

"Fusariosis" varies from mild, almost imperceptible nail colonization (58) to potentially fatal dissemination in compromised patients (59–61), caused by *Fusarium* or fusarioid species. If "fusariosis" is applicable to a disseminated, blood-carried infection, then a superficial nail infection by *F. oxysporum* is perhaps better referred to as *Fusarium oxysporum* onychomycosis, instead of fusariosis (or *Fusarium* onychomycosis when the species is not identified). The same would apply to a common manifestation of disease caused by *Fusarium* species such as fungal keratitis. In many instances, the adjective "disseminated" is used for clarity, as in disseminated fusariosis, sometimes with acute or subacute, to indicate a time frame. If disease names are limited to the rather few classic fungal diseases by primary pathogens, they might be less subject to variation. The same holds true for the names of common opportunists such as *A. fumigatus*. Also here, the prevalent, pulmonary infection can be referred to as aspergillosis, while *Aspergillus flavus* keratitis is a more appropriate term than aspergillosis since broader application

TABLE 1 Recommended names of diseases caused by primary pathogens and common opportunists in mammals[a,e]

General disease	Concise definition	Prevalent subtypes/notes
Adiaspiromycosis 🐾	Preponderantly rodent, rare human pulmonary invasion with giant cells by *Emmonsia crescens*	
Aspergillosis	Tissue invasion or inflammation by *Aspergillus* spp.	Allergic bronchopulmonary aspergillosis (ABPA)
		Chronic pulmonary aspergillosis (CPA)
		Chronic fibrosing pulmonary aspergillosis (CFPA)
		Chronic cavitary pulmonary aspergillosis (CCPA)
		Chronic necrotizing pulmonary aspergillosis (CNPA)
		Severe asthma with fungal sensitization (SAFS)
		Invasive pulmonary aspergillosis (IPA)
		Aspergilloma
		Allergic *Aspergillus* sinusitis
		Invasive *Aspergillus* sinusitis (*A. flavus, A. fumigatus*)
		Rhino-orbital-cerebral aspergillosis (ROCA)
		Disseminated[b] aspergillosis
		Aspergillus keratitis
		Aspergillus (Y[c]) infection
		Canine/feline sinonasal aspergillosis (SNA) 🐾
		Feline sinoorbital aspergillosis 🐾
Basidiobolomycosis	Tissue invasion by *Basidiobolus* spp.	Gastrointestinal basidiobolomycosis
		Subcutaneous basidiobolomycosis
Black piedra	Sclerotium formation on hair shaft by *Piedraia* spp.	
Blastomycosis	Tissue invasion or inflammation by *Blastomyces* spp.	Pulmonary blastomycosis
		Disseminated[b]blastomycosis
Candidiasis	Tissue invasion or inflammation by common ascomycetous yeasts	Mucocutaneous candidiasis
		Cutaneous (intertriginous) candidiasis
		Vulvovaginal candidiasis (VVC)
		Candida balanitis
		Oropharyngeal candidiasis (OPC)
		Esophageal candidiasis
		Candida fungemia (candidemia)
		Device-related candidemia
		Acute disseminated[b] candidiasis
		Chronic disseminated[b] candidiasis
		Candida meningoencephalitis
		Candida endocarditis
		Candida cystitis
		Renal candidiasis
		Hepatosplenic candidiasis
		Intra-abdominal candidiasis (abscess ad peritonitis)
		Candida empyema
		Candida arthritis
		Candida osteomyelitis
		Candida chorioretinitis
		Candida keratitis
Chromoblastomycosis	Subcutaneous invasion with muriform cells by the order of Chaetothyriales	
Chytridiomycosis 🐾	*Batrachochytrium* disease of frogs and salamanders	
Coccidioidomycosis	Tissue invasion or inflammation with spherules by *Coccidioides* spp.	Acute pulmonary coccidioidomycosis
		Chronic pulmonary coccidioidomycosis
		Disseminated coccidioidomycosis

(*Continued on next page*)

TABLE 1 Recommended names of diseases caused by primary pathogens and common opportunists in mammals[a,e] (*Continued*)

General disease	Concise definition	Prevalent subtypes/notes
Conidiobolomycosis	Tissue invasion of the sinus by *Conidiobolus* spp.	
Cryptococcosis	Tissue invasion or inflammation by *Cryptococcus* (*s.str.*) spp.	Pulmonary cryptococcosis
		Cerebral cryptococcosis
		Cryptococcus meningoencephalitis
		Cryptococcus fungemia
		Disseminated[b] cryptococcosis
		Cryptococcoma
		Cutaneous cryptococcosis
		Sinoorbital cryptococcosis 🐾
Dermatophytosis	Tissue invasion or inflammation by dermatophyte (*Epidermophyton*, *Microsporum*, *Nannizzia*, *Trichophyton* spp.)	See this list under Tinea
Emergomycosis	Tissue invasion or inflammation by *Emergomyces* spp.	
Eumycetoma	Subcutaneous invasion with grains due to various fungi of widely different agents *(Madurella*, *Scedosporium*, *Trematosphaeria, Curvularia, Bipolaris, Exophiala, Rhinocladiella, Acremonium, Sarocladium, Fusarium* spp.)	
Fungemia	Blood-carried fungus, wide diversity	
Fusariosis	Hematogenous dissemination by *Fusarium* and relatives	Disseminated[b] fusariosis
		Pulmonary fusariosis
		Fusarium fungemia
		Fusarium keratitis
		Fusarium onychomycosis
Histoplasmosis	Tissue invasion by *Histoplasma* spp.	Acute pulmonary histoplasmosis
		Chronic pulmonary histoplasmosis
		Progressive disseminated[b] histoplasmosis
		Primary cutaneous histoplasmosis
		Histoplasma mediastinal fibrosis
	Tissue invasion by *Histoplasma duboisii*	African histoplasmosis
		Epizootic lymphangitis in horses 🐾
Hyalohyphomycosis	Tissue invasion with hyaline hyphae of widely different agents	This term may be superfluous.
Lagenidiosis 🐾	Canine acute tissue invasion by *Lagenidium*[d] and *Paralagenidium*[d] spp.	
Malassezia dermatitis	Inflammation by *Malassezia* spp.	*Malassezia* seborrheic dermatitis
		Malassezia folliculitis
		Malassezia otitis
		Malassezia fungemia
Mucormycosis	Acute tissue invasion by the orders of Mucorales and Mortierellales	Cutaneous mucormycosis
		Disseminated[b] mucormycosis
		Gastrointestinal mucormycosis
		Pulmonary mucormycosis
		Rhino-orbital-cerebral mucormycosis (ROCM)
Onychomycosis	Nail invasion with yeast cells or hyphae of widely different agents	
Ophidiomycosis 🐾	Tissue invasion by *Ophidiomyces ophidiicola* in snakes	
Paracoccidioidomycosis PCM)	Tissue invasion by *Paracoccidioides* spp.	(Sub)acute paracoccidioidomycosis
		Chronic paracoccidioidomycosis
Paracoccidioidomycosis ceti 🐾	Tissue invasion by the unculturable *Paracoccidioides ceti* in dolphins (PCMC)	

(*Continued on next page*)

TABLE 1 Recommended names of diseases caused by primary pathogens and common opportunists in mammals[a,e] (*Continued*)

General disease	Concise definition	Prevalent subtypes/notes
Paracoccidioidomycosis lobogeorgi	Tissue invasion by the unculturable *Paracoccidioides lobogeorgii* in humans (PCML)	
Penicilliosis 🐾	Disseminated infection in dogs by *Penicillium canis* and *P. labradorum*	
Phaeohyphomycosis	Tissue invasion with melanized hyphae of widely different agents	This term may be superfluous
Pityriasis versicolor	Colonization and inflammation by *Malassezia* spp.	Also commonly known as tinea versicolor
Pneumocystis pneumonia	Pulmonary infection by *Pneumocystis* spp.	
Protothecosis 🐾	Preponderantly bovine tissue inflammation by the algae *Prototheca*[d] spp. In humans, protothecosis may be localized or disseminated	In humans, localized infection commonly involves the olecranon bursa
Pythiosis 🐾	Tissue invasion and inflammation by *Pythium*[d] spp. In humans, vascular and ocular pythiosis are common clinical manifestations.	
Rhinosporidiosis 🐾	Acanthosis by *Rhinosporidium*[d] spp. mainly in bird and horses	
Sporotrichosis	Tissue invasion by members of pathogenic clade of *Sporothrix* spp.	Lymphocutaneous sporotrichosis
		Fixed sporotrichosis
		Pulmonary sporotrichosis
		Disseminated[b] sporotrichosis
		Feline sporotrichosis 🐾
Talaromycosis	Disseminated intracellular infection by *Talaromyces marneffei*	
Tinea	Cutaneous infection by dermatophyte (*Microsporum, Trichophyton, Epidermophyton, Nannizzia* spp.)	Tinea capitis
		Tinea corporis
		Tinea cruris
		Tinea faciei
		Tinea manuum
		Tinea pedis
Tinea nigra	Cutaneous colonization by *Hortaea werneckii*	
White nose disease 🐾	Disease of hibernating bats	
White piedra	Growth on hair shaft by *Trichosporon* spp.	

[a]Definitions are intended to be used in the Atlas of Clinical Fungi and are consistent with the EORTC/MSG-ERC Revised Definitions, as well as several major guidelines of the IDSA, ISHAM, and ECMM and authoritative reviews.
[b]Disseminated refers to translocation to multiple sites other than the original site of infection.
[c](Y***) denotes the clinical picture concerned.
[d]Organisms that are non-fungal but conventionally treated under medical mycology.
[e]🐾, (nearly) exclusively animal disease.

of the term "aspergillosis" jeopardizes the definition of the disease name. In general, implantation diseases or localized infections, determining most opportunistic infections, are to be excluded from naming after the fungus. For those *Fusarium* and *Aspergillus* cases, and the exceptional, occasional, or rare opportunists, the disease should then be defined with the name of the causative fungus, followed by the descriptive term for the type of infection "*Colletotrichum truncatum* keratitis."

Genus-dependent disease names

With certain disease names, an entire genus or multiple genera are implicated, as is the case with histoplasmosis, coccidioidomycosis, and mucormycosis. But often, fungus-based disease names are linked to a particular species within a large genus, such as "talaromycosis" pertaining to only one species, *Talaromyces marneffei*. "Sporotrichosis" refers exclusively to the infections caused by any of the four species (*S. brasiliensis, S.*

TABLE 2 Examples of names of diseases caused by uncommon fungal opportunists[a]

Etiologic agent (X) and disease (Y)
Yeasts
(X) *Cutaneotrichosporon cutaneum* [*Trichosporon cutaneum*] (Y) infection
(X) *Geotrichum candidum* (Y) infection
(X) *Magnusiomyces capitatus* [*Saprochaete capitata*](Y) infection
(X) *Trichosporon asahii* (Y) infection
Filamentous fungi
(X) *Alternaria alternata* (Y) infection
(X) *Bipolaris*[b] (Y) cerebral infection
(X) *Cladophialophora bantiana* (Y) cerebral infection
(X) *Colletotrichum gloeosporioides* keratitis
Disseminated (X) *Phialophora verrucosa* (Y) infection
(X) *Fusarium oxysporum* (Y) infection
(X) *Lomentospora prolificans* (Y) infection
(X) *Madurella mycetomatis* mycetoma
(X) *Paecilomyces variotii* sinusitis
(X) *Scedosporium apiospermum* (Y) infection

[a](X) denotes fungal agent; (Y) denotes the clinical picture concerned. A more complete list of disease indications will be available on https://www.atlasclinicalfungi.org/nomenclaturenames/.
[b]Some *Bipolaris* species have been proposed to be re-classified as Curvularia species.

globosa, S. luriei, and *S. schenckii*) of the "pathogenic clade" of *Sporothrix* (62) because other species of this genus have an entirely different ecology (63). "Dermatophytosis" covers the cutaneous infections caused by several genera in the family Arthrodermataceae that are frequently involved in human infection; this would exclude the geophilic members in *Arthroderma* of the same family. In this specific example, dermatophyte infections are referred to using the general term dermatophytosis rather than one derived from the genus. Following long-standing and widespread usage, the term "tinea" can also be used as a disease descriptor often followed by the part of the body affected (in Latin), for example, tinea capitis (head), tinea pedis (foot). Likewise, one of the common skin infections caused by *Malassezia* species is widely known as pityriasis versicolor or tinea versicolor, the former name is recommended here. "Mucormycosis" stands for the acute, severe infections caused by members of several remotely related genera within the order Mucorales, albeit only a few species in this order are involved. There are several prevalent types based on affected body sites, which can be referred to as rhinocerebral mucormycosis, pulmonary mucormycosis, gastrointestinal mucormycosis, etc. *Basidiobolus* and *Conidiobolus* species are phylogenetically diverse, and their clinical manifestations are very different and are therefore preferably referred to as basidiobolomycosis and conidiobolomycosis, respectively. Despite their overarching classification in Entomophthoromycotina, the name entomophthoromycosis is not recommended since this term is also used for insect infections. Chromoblastomycosis is a subcutaneous disease caused by various members of the black fungal order Chaetothyriales. Although phylogenetically separated, members of *Cladophialophora, Fonsecaea, Phialophora,* and *Rhinocladiella* can cause chromoblastomycosis with the common feature of muriform cell formation in tissue. "Fusariosis" is recommended to apply to systemic infections by fusarioid species, implying that the disease name does not change upon reclassification of some members of the classic genus *Fusarium* in *Neocosmospora* (64). Likewise, "candidiasis" could be maintained as a clinical entity that results from infection due to phylogenetically widely different ascomycetous yeasts. The above-described clinical variation due to the single species, *Candida albicans,* exceeds that manifested between yeasts belonging to different genera. In addition to the infection site, specification of the disease type is required, leading to subtype nomenclature, such as ABPA designating allergic bronchopulmonary aspergillosis. If possible, the use of the recommended disease name must be accompanied by identification of the etiologic agent, including new and previous names, as required.

Diseases by unrelated agents

A number of disease names used exclusively in mycology do not refer to a particular agent. "Mycetoma" represents a subcutaneous implantation disease where the fungus or bacterium (aerobic actinomycetes) occurs in the form of grains regardless of the etiologic agent. "Eumycetoma" adds the diagnostic feature that the infection is caused by a fungus. The standard disease type is therefore already in use as "*Madurella fahalii* mycetoma," analogous to the designation of "*Colletotrichum gloeosporioides* keratitis." General fungal diseases designated as phaeo- and hyalohyphomycosis have limited diagnostic value, only distinguishing between melanized *versus* non-melanized filamentous etiologic agents. The clinical manifestations of phaeohyphomycosis may vary from insignificant nail infection to fatal cerebral infection caused by phylogenetically widely different dematiaceous fungi (65). Some of the diseases in the phaeohyphomycotic category have been linked to the etiologic agent, such as "alternariosis" (66). It remains unclear why diseases by related, equally common *Curvularia* species have not been named after the fungus. Names such as "alternariosis" appears to imply particular clinical type or course of disease, while this is not the case: most infections reported are (sub)cutaneous in patients receiving corticosteroid therapy (67) although other types of infection can also occur (31). The use of the term alternariosis is not recommended.

Conclusions and recommendations

According to the above-described concepts, we suggest restricting fungus-based disease names to the cases listed in Table 1. These definitions are intended to be consistent with the EORTC/MSG-ERC Revised Definitions, as well as several major guidelines of the IDSA, ISHAM, and ECMM, and authoritative reviews (68–75) and WHO guidelines for International Classification of Diseases (ICD). All other fungal diseases are then designated by the name of the etiologic agent followed by the clinical type. Pertinent examples are given in Table 2. This system is straightforward and can be publicly accessed through a database that is focused on medical and veterinary fungi (https://www.atlasclinicalfungi.org/nomenclaturenames/). In due course, numerous synonymous disease names can also be included, improving standardized reporting of fungal infections. This model for naming diseases caused by medically important fungi may help in improving the International Classification of Diseases (ICD) codes for fungal infections. The ICD codes are especially important for more accurately defining fungal diseases for patient care, epidemiology, public health, healthcare economics, and research funding priorities.

ACKNOWLEDGMENTS

The ISHAM/ECMM/FDLC Working Group Nomenclature of Clinical Fungi consists of the International Society for Human and Animal Mycoses (ISHAM), European Confederation of Medical Mycology (ECMM), and Fungal Diagnostic Laboratory Consortium (FDLC) (https://www.isham.org/working-groups/nomenclature-clinical-fungi).

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

AUTHOR AFFILIATIONS

[1]Radboudumc-CWZ Centre of Expertise for Mycology, Nijmegen, the Netherlands
[2]Foundation Atlas of Clinical Fungi, Hilversum, the Netherlands
[3]Center for Innovative Therapeutics and Diagnostics, Richmond, Virginia, USA
[4]University of Maryland School of Medicine, Baltimore, Maryland, USA
[5]Mycology Reference Laboratory, Spanish National Centre for Microbiology, Madrid, Spain
[6]Centro de Investigación Biomédica en Red de Enfermedades Infecciosas, Instituto de Salud Carlos III, Madrid, Spain

[7]Department of Microbiology and Infection Control, Statens Serum Institut, Copenhagen, Denmark
[8]Department of Clinical Microbiology, Copenhagen University Hospital, Copenhagen, Denmark
[9]National Mycology Reference Laboratory, Public Health England, Bristol, United Kingdom
[10]Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead, Australia
[11]Medical Mycology Unit, Department of Microbiology, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India
[12]National Reference Laboratory for Antimicrobial Resistance in Fungal Pathogens, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India
[13]Division of Infectious Diseases, Mount Auburn Hospital, and Harvard Medical School, Cambridge, Massachusetts, USA
[14]University of Cologne, Faculty of Medicine, Institute of Translational Research, Cologne, Germany
[15]Excellence Center for Medical Mycology, Department I of Internal Medicine, University of Cologne, Cologne, Germany
[16]Manchester Fungal Infection Group, Academic Health Science Centre, University of Manchester, Manchester, United Kingdom
[17]Laboratoire de Santé Publique du Québec, Institut National de Santé Publique du Québec, Sainte-Anne-de-Bellevue, Canada
[18]Department of Pathology, University of Texas Southwestern Medical Center, Dallas, Texas, USA
[19]Department of Mycology, Centre Hospitalier Universitaire de Rennes, Centre National de Référence Aspergilloses chroniques, ECMM Excellence Center in Mycology, Rennes, France
[20]Unitat de Micologia i Microbiologia Ambiental, Facultat de Medicina i Ciènces de la Salut, Universitat Rovira i Virgili, Reus, Spain
[21]Infectious Disease Research Program, Department of Pediatric Hematology and Oncology, University Children's Hospital Münster, Münster, Germany
[22]Oniris, VetAgroBio Nantes, IRF, SFR ICAT, Université d'Angers, Angers, France
[23]Laboratory Diagnostic Center, RWTH Aachen University Hospital, Aachen, Germany
[24]Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead, Australia
[25]Royal Botanic Gardens, Kew, Richmond, United Kingdom
[26]Natural History Museum, London, United Kingdom
[27]University of Southampton, Southampton, United Kingdom
[28]Jilin Agricultural University, Chanchung, China
[29]St. John's Institute of Dermatology, King's College London, London, United Kingdom
[30]Division of Infectious Diseases, Medical University of Graz, Graz, Austria
[31]Translational Medical Mycology Research Unit, Medical University of Graz, Graz, Austria
[32]Department of Botany, Charles University, Prague, Czechia
[33]Department of Medical Microbiology, University of Warsaw, Warsaw, Poland
[34]Westerdijk Fungal Biodiversity Institute, Utrecht, the Netherlands
[35]National Mycology Reference Centre, SA Pathology, Adelaide, Australia
[36]School of Biological Sciences, Faculty of Sciences Engineering and Technology, University of Adelaide, Adelaide, Australia
[37]Public Health Ontario Toronto, Toronto, Canada
[38]Department of Laboratory Medicine and Pathobiology, University of Toronto, Toronto, Canada
[39]National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland, USA
[40]Centers for Disease Control and Prevention, Atlanta, Georgia, USA

[41]Microbiology and Molecular Genetics, Biomedical Laboratory Diagnostics, Michigan State University, East Lansing, Michigan, USA
[42]University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA
[43]Department of Dermatology, Peking University First Hospital, Peking University, Beijing, China
[44]Sydney Infectious Diseases Institute, Faculty of Medicine and Health, University of Sydney, Sydney, Australia
[45]Bioinformatics, Helmholtz Institute for One Health, Greifswald, Germany
[46]CONICET (Consejo Nacional de Investigaciones Científicas y Tecnológicas), Hospital JM Ramos Mejía, Buenos Aires, Argentina
[47]Division of Clinical Microbiology, Mayo Clinic, Rochester, Minnesota, USA
[48]Public Health Wales Microbiology, Cardiff, United Kingdom
[49]McGovern Medical School, Division of Infectious Diseases, University of Texas Health Science Center, Houston, Texas, USA
[50]Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

AUTHOR ORCIDs

Sybren de Hoog http://orcid.org/0000-0002-5344-257X
Thomas J. Walsh http://orcid.org/0000-0002-4142-8711
Ana Alastruey-Izquierdo http://orcid.org/0000-0001-8651-4405
Maiken Cavling Arendrup http://orcid.org/0000-0002-4747-0144
Andrew Borman http://orcid.org/0000-0003-0585-5721
Laura Filkins http://orcid.org/0000-0002-0406-4947
Josepa Gené http://orcid.org/0000-0001-6195-9299
Andreas H. Groll http://orcid.org/0000-0003-1188-393X
Martin Hoenigl http://orcid.org/0000-0002-1653-2824
Sarah E. Kidd http://orcid.org/0000-0002-5957-4178
Julianne V. Kus http://orcid.org/0000-0001-6033-7244
June Kwon-Chung http://orcid.org/0000-0001-6671-0994
Shawn R. Lockhart http://orcid.org/0000-0002-4383-5994
Jacques F. Meis http://orcid.org/0000-0003-3253-6080
Leonel Mendoza http://orcid.org/0000-0002-7769-2151
M. Hong Nguyen http://orcid.org/0000-0003-2065-3239
Roxana G. Vitale http://orcid.org/0000-0001-8425-0856
Nancy L. Wengenack http://orcid.org/0000-0003-2853-8219
P. Lewis White http://orcid.org/0000-0003-3056-4205
Sean X. Zhang http://orcid.org/0000-0003-1166-2563

AUTHOR CONTRIBUTIONS

Sybren de Hoog, Conceptualization, Writing – original draft, Writing – review and editing | Thomas J. Walsh, Conceptualization, Writing – original draft, Writing – review and editing | Sarah A. Ahmed, Writing – review and editing | Ana Alastruey-Izquierdo, Writing – review and editing | Maiken Cavling Arendrup, Writing – review and editing | Andrew Borman, Writing – review and editing | Sharon Chen, Writing – review and editing | Anuradha Chowdhary, Writing – review and editing | Robert C. Colgrove, Writing – review and editing | Oliver A. Cornely, Writing – review and editing | David W. Denning, Writing – review and editing | Laura Filkins, Writing – review and editing | Jean-Pierre Gangneux, Writing – review and editing | Josepa Gené, Writing – review and editing | Andreas H. Groll, Writing – review and editing | Jaques Guillot, Writing – review and editing | Gerhard Haase, Writing – review and editing | Catriona Halliday, Writing – review and editing | David L. Hawksworth, Writing – review and editing | Martin Hoenigl, Writing – review and editing | Vit Hubka, Writing – review and editing | Tomasz Jagielski, Writing – review and editing | Hazal Kandemir, Writing – review and editing | Sarah E. Kidd, Writing – review

and editing | Julianne V. Kus, Writing – review and editing | June Kwon-Chung, Writing – review and editing | Shawn R. Lockhart, Writing – review and editing | Jacques F. Meis, Writing – review and editing | Leonel Mendoza, Writing – review and editing | Wieland Meyer, Writing – review and editing | M. Hong Nguyen, Writing – review and editing | Yinggai Song, Writing – review and editing | Tania C. Sorrell, Writing – review and editing | J. Benjamin Stielow, Writing – review and editing | Roxana G. Vitale, Writing – review and editing | Nancy L. Wengenack, Writing – review and editing | P. Lewis White, Writing – review and editing | Luis Ostrosky-Zeichner, Writing – review and editing | Sean X. Zhang, Conceptualization, Writing – original draft, Writing – review and editing.

REFERENCES

1. Hawksworth DL. 2011. A new dawn for the naming of fungi: impacts of decisions made in Melbourne in July 2011 on the future publication and regulation of fungal names. MycoKeys 1:7–20. https://doi.org/10.3897/mycokeys.1.2062
2. Hawksworth DL, Crous PW, Redhead SA, Reynolds DR, Samson RA, Seifert KA, Taylor JW, Wingfield MJ, Abaci Ö, Aime C, et al. 2011. The amsterdam declaration on fungal nomenclature. IMA Fungus 2:105–112. https://doi.org/10.5598/imafungus.2011.02.01.14
3. May TW, Redhead SA, Bensch K, Hawksworth DL, Lendemer J, Lombard L, Turland NJ. 2019. Chapter F of the International code of nomenclature for algae, fungi, and plants as approved by the 11th International mycological congress, San Juan, Puerto Rico, July 2018. IMA Fungus 10:21. https://doi.org/10.1186/s43008-019-0019-1
4. Borman AM, Johnson EM. 2021. Name changes for fungi of medical importance, 2018 to 2019. J Clin Microbiol 59:01811–01820. https://doi.org/10.1128/JCM.01811-20
5. de Hoog GS, Chaturvedi V, Denning DW, Dyer PS, Frisvad JC, Geiser D, Gräser Y, Guarro J, Haase G, Kwon-Chung K-J, Meis JF, Meyer W, Pitt JI, Samson RA, Taylor JW, Tintelnot K, Vitale RG, Walsh TJ, Lackner M, ISHAM Working Group on Nomenclature of Medical Fungi. 2015. Commentaries: name changes in medically important fungi and their implications for clinical practice. J Clin Microbiol 53:1056–1062. https://doi.org/10.1128/JCM.02016-14
6. de Hoog S, Walsh TJ, Ahmed SA, Alastruey-Izquierdo A, Alexander BD, Arendrup MC, Babady E, Bai F-Y, Balada-Llasat J-M, Borman A. 2023. A conceptual framework for nomenclatural stability and validity of medically important fungi: a proposed global consensus guideline for fungal name changes supported by ABP, ASM, CLSI, ECMM, ESCMID-EFISG, EUCAST-AFST, FDLC, IDSA, ISHAM, MMSA, and MSGERC. J Clin Microbiol 61:e0087323. https://doi.org/10.1128/jcm.00873-23
7. Kidd SE, Abdolrasouli A, Hagen F. 2023. Fungal nomenclature: managing change is the name of the game. Open Forum Infect Dis 10:fac559. https://doi.org/10.1093/ofid/ofac559
8. Kidd SE, Halliday CL, McMullan B, Chen S-A, Elvy J. 2021. New names for fungi of medical importance: can we have our cake and eat it too? J Clin Microbiol 59:e02730–02720. https://doi.org/10.1128/JCM.02730-20
9. Odds FC, Arai T, Disalvo AF, Evans EG, Hay RJ, Randhawa HS, Rinaldi MG, Walsh TJ. 1992. Nomenclature of fungal diseases: a report and recommendations from a sub-committee of the International society for human and animal mycology (ISHAM). J Med Vet Mycol 30:1–10. https://doi.org/10.1080/02681219280000021
10. Kidd SE, Abdolrasouli A, Hagen F. 2023. Reply to "Re: fungal nomenclature: managing change is the name of the game." Open Forum Infect Dis 10:ofad394. https://doi.org/10.1093/ofid/ofad394
11. Denning DW. 2023. Fungal nomenclature: managing change is the name of the game. Open Forum Infect Dis 10:ofad395. https://doi.org/10.1093/ofid/ofad395
12. Lackner M, de Hoog GS, Yang L, Ferreira Moreno L, Ahmed SA, Andreas F, Kaltseis J, Nagl M, Lass-Flörl C, Risslegger B, et al. 2014. Proposed nomenclature for *Pseudallescheria, Scedosporium* and related genera. Fungal Divers 67:1–10. https://doi.org/10.1007/s13225-014-0295-4
13. Takashima M, Sugita T. 2022. Taxonomy of pathogenic yeasts *Candida*, *Cryptococcus, Malassezia, and Trichosporon*. Med Mycol J 63:119–132. https://doi.org/10.3314/mmj.22.004
14. Denning DW. 2024. Renaming *Candida glabrata*-a case of taxonomic purity over clinical and public health pragmatism. PLoS Pathog 20:e1012055. https://doi.org/10.1371/journal.ppat.1012055
15. Kandi V, Vaish R, Gurrapu P, Koka SS, Bhoomigari MR. 2020. Geotrichosis presenting as funguria and asymptomatic urinary tract infection in a patient with renal cyst. Cureus 12:e7616. https://doi.org/10.7759/cureus.7616
16. Keene S, Sarao MS, McDonald PJ, Veltman J. 2019. Cutaneous geotrichosis due to *Geotrichum candidum* in a burn patient. Access Microbiol 1:e000001. https://doi.org/10.1099/acmi.0.000001
17. Becchetti C, Ferrarese A, Cattelan A, Barbieri S, Feltracco P, Saluzzo F, Cillo U, Senzolo M, Germani G, Burra P. 2020. *Geotrichum capitatum* invasive infection early after liver transplant. Exp Clin Transplant 18:737–740. https://doi.org/10.6002/ect.2019.0170
18. Groenewald M, Hittinger CT, Bensch K, Opulente DA, Shen X-X, Li Y, Liu C, LaBella AL, Zhou X, Limtong S, et al. 2023. A genome-informed higher rank classification of the biotechnologically important fungal subphylum *Saccharomycotina*. Stud Mycol 105:1–22. https://doi.org/10.3114/sim.2023.105.01
19. Banerjee U, Mohapatra AK, Sarkar C, Chaudhery R. 1989. Cladosporiosis (cerebral phaeohyphomycosis) of brain--a case report. Mycopathologia 105:163–166. https://doi.org/10.1007/BF00437249
20. Kurkina YN. 2020. Phytoncide action of essential oils of medicinal plants on the causative agents of broad beans alternariosis and cladosporiosis. Ovoŝ Rossii 3:73–76. https://doi.org/10.18619/2072-9146-2020-3-73-76
21. Hawksworth DL, Lücking R. 2017. Fungal diversity revisited: 2.2 to 3.8 million species. Microbiol Spectr 5. https://doi.org/10.1128/microbiolspec.FUNK-0052-2016
22. de Beer ZW, Begerow D, Bauer R, Pegg GS, Crous PW, Wingfield MJ. 2006. Phylogeny of the *Quambalariaceae* fam. nov., including important eucalyptus pathogens in South Africa and Australia. Stud Mycol 55:289–298. https://doi.org/10.3114/sim.55.1.289
23. Ahmed SA, van de Sande WWJ, Stevens DA, Fahal A, van Diepeningen AD, Menken SBJ, de Hoog GS. 2014. Revision of agents of black-grain eumycetoma in the order *Pleosporales*. Persoonia 33:141–154. https://doi.org/10.3767/003158514X684744
24. Tedersoo L, Sánchez-Ramírez S, Kõljalg U, Bahram M, Döring M, Schigel D, May T, Ryberg M, Abarenkov K. 2018. High-level classification of the fungi and a tool for evolutionary ecological analyses. Fungal Divers 90:135–159. https://doi.org/10.1007/s13225-018-0401-0
25. de Hoog GS, Taylor JW, Redhead SA. 2023. Proposal to conserve the name *Coccidioides posadasii* against *Posadasia esferiformis*, *Coccidium posadasi*, *Pseudococcidioides mazzae*, *Geotrichum louisianoideum*, *Glenospora meteuropea*, *Glenospora metamericana* and *Trichosporon proteolyticum* (Ascomycota)(2952). Taxon 72:656–660. https://doi.org/10.1002/tax.12961
26. Muñoz - Rodríguez P, Wood JRI, Wells T, Carruthers T, Sumadijaya A, Scotland RW. 2023. The challenges of classifying big genera such as *Ipomoea* . Taxon 72:1201–1215. https://doi.org/10.1002/tax.12887
27. Yurkov A, Alves A, Bai F-Y, Boundy-Mills K, Buzzini P, Čadež N, Cardinali G, Casaregola S, Chaturvedi V, Collin V, et al. 2021. Nomenclatural issues concerning cultured yeasts and other fungi: why it is important to avoid unneeded name changes. IMA Fungus 12:18. https://doi.org/10.1186/s43008-021-00067-x

28. Dayarathne M, Boonmee S, Braun U, Crous P, Daranagama D, Dissanayake A, Ekanayaka A, Jayawardena R, Ebg J, Maharachchikumbura S, Perera R, Phillips A, Stadler M, Thambugala K, Wanasinghe D, Zhao Q, Hyde K, Jeewon R. 2016. Taxonomic utility of old names in current fungal classification and nomenclature: conflicts, confusion & clarifications. Mycosphere 7:1622–1648. https://doi.org/10.5943/mycosphere/7/11/2
29. Crous PW, Giraldo A, Hawksworth DL, Robert V, Kirk PM, Guarro J, Robbertse B, Schoch CL, Damm U, Trakunyingcharoen T, Groenewald JZ. 2014. The genera of fungi: fixing the application of type species of generic names. IMA Fungus 5:141–160. https://doi.org/10.5598/imafungus.2014.05.01.14
30. Crous PW, Hawksworth DL, Wingfield MJ. 2015. Identifying and naming plant-pathogenic fungi: past, present, and future. Annu Rev Phytopathol 53:247–267. https://doi.org/10.1146/annurev-phyto-080614-120245
31. Hoog GS, Guarro J, Gené J, Ahmed SA, Al-Hatmi AMS, Figueras MJ, Vitale RG. 2020. Atlas of clinical fungi, p 1599. In Foundation atlas of clinical fungi. Vol. 4. Hilversum.
32. Turland NJ, Wiersema JH, Barrie FR, Greuter W, Hawksworth DL, Herendeen PS, Knapp S, Kusber W-H, Li D-Z, Marhold K. 2018. International Code of Nomenclature for algae, fungi, and plants (Shenzhen Code) adopted by the Nineteenth International Botanical Congress Shenzhen, China, July 2017. In Koeltz bot books
33. Kirk PM, Stalpers JA, Braun U, Crous PW, Hansen K, Hawksworth DL, Hyde KD, Lücking R, Lumbsch TH, Rossman AY, Seifert KA, Stadler M. 2013. A without-prejudice list of generic names of fungi for protection under the International Code of Nomenclature for algae, fungi, and plants. IMA Fungus 4:381–443. https://doi.org/10.5598/imafungus.2013.04.02.17
34. Zhang X-X, Liu J-S, Han L-F, Xia S, Li S-Z, Li OY, Kassegne K, Li M, Yin K, Hu Q-Q, et al. 2022. Towards a global one health index: a potential assessment tool for one health performance. Infect Dis Poverty 11:57. https://doi.org/10.1186/s40249-022-00979-9
35. Zhao H-Q, Fei S-W, Yin J-X, Li Q, Jiang T-G, Guo Z-Y, Xue J-B, Han L-F, Zhang X-X, Xia S, Zhang Y, Guo X-K, Kassegne K. 2022. Assessment of performance for a key indicator of one health: evidence based on one health index for zoonoses in Sub-Saharan Africa. Infect Dis Poverty 11:109. https://doi.org/10.1186/s40249-022-01020-9
36. Feng J, Guo Z, Ai L, Liu J, Zhang X, Cao C, Xu J, Xia S, Zhou X-N, Chen J, Li S. 2022. Establishment of an indicator framework for global one health intrinsic drivers index based on the grounded theory and fuzzy analytical hierarchy-entropy weight method. Infect Dis Poverty 11:121. https://doi.org/10.1186/s40249-022-01042-3
37. Jato-Espino D, Mayor-Vitoria F, Moscardó V, Capra-Ribeiro F, Bartolomé del Pino LE. 2023. Toward one health: a spatial indicator system to model the facilitation of the spread of zoonotic diseases. Front Public Health 11. https://doi.org/10.3389/fpubh.2023.1215574
38. Sun Z-S, Wan E-Y, Agbana YL, Zhao H-Q, Yin J-X, Jiang T-G, Li Q, Fei S-W, Wu LB, Li X-C, et al. 2024. Global one health index for zoonoses: a performance assessment in 160 countries and territories. iScience 27:109297. https://doi.org/10.1016/j.isci.2024.109297
39. Demanche C, Berthelemy M, Petit T, Polack B, Wakefield AE, Dei-Cas E, Guillot J. 2001. Phylogeny of *Pneumocystis carinii* from 18 primate species confirms host specificity and suggests coevolution. J Clin Microbiol 39:2126–2133. https://doi.org/10.1128/JCM.39.6.2126-2133.2001
40. Guillot J, Demanche C, Hugot JP, Berthelemy M, Wakefield AE, Dei-Cas E, Chermette R. 2001. Parallel phylogenies of *Pneumocystis* species and their mammalian hosts. J Eukaryot Microbiol Suppl:113S–115S. https://doi.org/10.1111/j.1550-7408.2001.tb00475.x
41. Cushion MT. 2010. Are members of the fungal genus *Pneumocystis* (a) commensals; (b) opportunists; (c) pathogens; or (d) all of the above? PLoS Pathog 6:e1001009. https://doi.org/10.1371/journal.ppat.1001009
42. Morris A, Norris KA. 2012. Colonization by *Pneumocystis jirovecii* and its role in disease. Clin Microbiol Rev 25:297–317. https://doi.org/10.1128/CMR.00013-12
43. Chen W, Gigliotti F, Harmsen AG. 1993. Latency is not an inevitable outcome of infection with *Pneumocystis carinii*. Infect Immun 61:5406–5409. https://doi.org/10.1128/iai.61.12.5406-5409.1993
44. Gigliotti F, Harmsen AG, Wright TW. 2003. Characterization of transmission of *Pneumocystis carinii* f. sp. muris through immunocompetent BALB/c mice. Infect Immun 71:3852–3856. https://doi.org/10.1128/IAI.71.7.3852-3856.2003
45. Chabé M, Dei-Cas E, Creusy C, Fleurisse L, Respaldiza N, Camus D, Durand-Joly I. 2004. Immunocompetent hosts as a reservoir of *Pneumocystis* organisms: histological and rt-PCR data demonstrate active replication. Eur J Clin Microbiol Infect Dis 23:89–97. https://doi.org/10.1007/s10096-003-1092-2
46. Cao C, Liang L, Wang W, Luo H, Huang S, Liu D, Xu J, Henk DA, Fisher MC. 2011. Common reservoirs for *Penicillium marneffei* infection in humans and rodents, China. Emerg Infect Dis 17:209–214. https://doi.org/10.3201/eid1702.100718
47. Tang C, Zhou X, Guillot J, Wibbelt G, Deng S, Kandemir H, Gräser Y, Feng P, Kang Y, de Hoog GS. 2023. Dermatophytes and mammalian hair: aspects of the evolution of *Arthrodermataceae*. Fungal Divers 125:139–156. https://doi.org/10.1007/s13225-023-00526-3
48. Carpouron JE, de Hoog S, Gentekaki E, Hyde KD. 2022. Emerging animal-associated fungal diseases. J Fungi (Basel) 8:611. https://doi.org/10.3390/jof8060611
49. Sobianski Herman T, Azevedo CMPS, Lorenzetti Bocca A, Jacomel Favoreto de Souza Lima B, Pavini Beato-Souza C, Razzolini E, Marques GS, Nunes N, Paulo Rodrigues Lustosa B, Wagner Castro Lima Santos D, Queiroz-Telles F, de Hoog S, Gomes R, Aparecida Vicente V. 2024. CARD9 mutations and T cell immune response in patients with chromoblastomycosis. OHM 1:14–22. https://doi.org/10.63049/OHM.24.11.3
50. Vilela R, de Hoog S, Bensch K, Bagagli E, Mendoza L. 2023. A taxonomic review of the genus *Paracoccidioides*, with focus on the uncultivable species. PLOS Negl Trop Dis 17:e0011220. https://doi.org/10.1371/journal.pntd.0011220
51. Zhang J, Debets AJM, Verweij PE, Snelders E. 2021. Azole-resistance development; how the *Aspergillus fumigatus* lifecycle defines the potential for adaptation. J Fungi (Basel) 7:599. https://doi.org/10.3390/jof7080599
52. Krause W, Matheis H, Wulf K. 1969. Fungaemia and funguria after oral administration of *Candida albicans*. Lancet 1:598–599. https://doi.org/10.1016/s0140-6736(69)91534-7
53. Fennelly AM, Slenker AK, Murphy LC, Moussouttas M, DeSimone JA. 2013. *Candida* cerebral abscesses: a case report and review of the literature. Med Mycol 51:779–784. https://doi.org/10.3109/13693786.2013.789566
54. Liu F, Hu Z-D, Zhao X-M, Zhao W-N, Feng Z-X, Yurkov A, Alwasel S, Boekhout T, Bensch K, Hui F-L, Bai F-Y, Wang Q-M. 2024. Phylogenomic analysis of the *Candida auris- Candida haemuli* clade and related taxa in the *Metschnikowiaceae,* and proposal of thirteen new genera, fifty-five new combinations and nine new species. Persoonia 52:22–43. https://doi.org/10.3767/persoonia.2024.52.02
55. de Hoog S, Tang C, Zhou X, Jacomel B, Lustosa B, Song Y, Kandemir H, A Ahmed S, Zhou S, Belmonte-Lopes R, Quan Y, Feng P, A Vicente V, Kang Y. 2024. Fungal primary and opportunistic pathogens: an ecological perspective. FEMS Microbiol Rev 48:fuae022. https://doi.org/10.1093/femsre/fuae022
56. Casadevall A, Pirofski L. 2007. Accidental virulence, cryptic pathogenesis, martians, lost hosts, and the pathogenicity of environmental microbes. Eukaryot Cell 6:2169–2174. https://doi.org/10.1128/EC.00308-07
57. Casadevall A. 2007. Determinants of virulence in the pathogenic fungi. Fungal Biol Rev 21:130–132. https://doi.org/10.1016/j.fbr.2007.02.007
58. Lu L-Y, Ou J-H, Hui RC-Y, Chuang Y-H, Fan Y-C, Sun P-L. 2023. High diversity of *Fusarium* species in onychomycosis: clinical presentations, molecular identification, and antifungal susceptibility. J Fungi (Basel) 9:534. https://doi.org/10.3390/jof9050534
59. Fanci R, Pini G, Bartolesi AM, Pecile P. 2013. Refractory disseminated fusariosis by *Fusarium verticillioides* in a patient with acute myeloid leukaemia relapsed after allogeneic hematopoietic stem cell transplantation: a case report and literature review. Rev Iberoam Micol 30:51–53. https://doi.org/10.1016/j.riam.2012.05.003
60. Jacobs SE, Wengenack NL, Walsh TJ. 2020. Non-*Aspergillus* hyaline molds: emerging causes of sino-pulmonary fungal infections and other invasive mycoses. Semin Respir Crit Care Med 41:115–130. https://doi.org/10.1055/s-0039-3401989
61. Nucci M, Anaissie E. 2023. Invasive fusariosis. Clin Microbiol Rev 36:e0015922. https://doi.org/10.1128/cmr.00159-22

62. Zhang Y, Hagen F, Stielow B, Rodrigues AM, Samerpitak K, Zhou X, Feng P, Yang L, Chen M, Deng S, Li S, Liao W, Li R, Li F, Meis JF, Guarro J, Teixeira M, Al-Zahrani HS, Pires de Camargo Z, Zhang L, de Hoog GS. 2015. Phylogeography and evolutionary patterns in *Sporothrix* spanning more than 14 000 human and animal case reports. Persoonia 35:1–20. https://doi.org/10.3767/003158515X687416
63. de Beer ZW, Duong TA, Wingfield MJ. 2016. The divorce of *Sporothrix* and *Ophiostoma*: solution to a problematic relationship. Stud Mycol 83:165–191. https://doi.org/10.1016/j.simyco.2016.07.001
64. Sandoval-Denis M, Lombard L, Crous PW. 2019. Back to the roots: a reappraisal of *Neocosmospora*. Persoonia 43:90–185. https://doi.org/10.3767/persoonia.2019.43.04
65. Revankar SG, Baddley JW, Chen SC-A, Kauffman CA, Slavin M, Vazquez JA, Seas C, Morris MI, Nguyen MH, Shoham S, Thompson GR III, Alexander BD, Simkins J, Ostrosky-Zeichner L, Mullane K, Alangaden G, Andes DR, Cornely OA, Wahlers K, Lockhart SR, Pappas PG. 2017. A mycoses study group international prospective study of phaeohyphomycosis: an analysis of 99 proven/probable cases. Open Forum Infect Dis 4:fx200. https://doi.org/10.1093/ofid/ofx200
66. Lopes L, Borges-Costa J, Soares-Almeida L, Filipe P, Neves F, Santana A, Guerra J, Kutzner H. 2013. Cutaneous alternariosis caused by alternaria infectoria: three cases in kidney transplant patients. Healthcare (Basel) 1:100–106. https://doi.org/10.3390/healthcare1010100
67. Machet L, Jan V, Machet MC, Vaillant L, Lorette G. 1996. Cutaneous Alternariosis: role of corticosteroid-Induced cutaneous fragility. Dermatology (Basel) 193:342–344. https://doi.org/10.1159/000246287
68. Chowdhary A, Meis JF, Guarro J, de Hoog GS, Kathuria S, Arendrup MC, Arikan-Akdagli S, Akova M, Boekhout T, Caira M, et al. 2014. ESCMID and ECMM joint clinical guidelines for the diagnosis and management of systemic phaeohyphomycosis: diseases caused by black fungi. Clin Microbiol Infect 20 Suppl 3:47–75. https://doi.org/10.1111/1469-0691.12515
69. Pappas PG, Kauffman CA, Andes DR, Clancy CJ, Marr KA, Ostrosky-Zeichner L, Reboli AC, Schuster MG, Vazquez JA, Walsh TJ, Zaoutis TE, Sobel JD. 2016. Executive summary: clinical practice guideline for the management of *Candidiasis*: 2016 update by the infectious diseases society of America. Clin Infect Dis 62:409–417. https://doi.org/10.1093/cid/civ1194
70. Patterson TF, Thompson GR III, Denning DW, Fishman JA, Hadley S, Herbrecht R, Kontoyiannis DP, Marr KA, Morrison VA, Nguyen MH, Segal BH, Steinbach WJ, Stevens DA, Walsh TJ, Wingard JR, Young J-AH, Bennett JE. 2016. Practice guidelines for the diagnosis and management of aspergillosis: 2016 update by the infectious diseases society of America. Clin Infect Dis 63:e1–e60. https://doi.org/10.1093/cid/ciw326
71. Queiroz-Telles F, de Hoog S, Santos DWCL, Salgado CG, Vicente VA, Bonifaz A, Roilides E, Xi L, Azevedo C de MPES, da Silva MB, Pana ZD, Colombo AL, Walsh TJ. 2017. Chromoblastomycosis. Clin Microbiol Rev 30:233–276. https://doi.org/10.1128/CMR.00032-16
72. Ahmed AA, van de Sande W, Fahal AH. 2017. Mycetoma laboratory diagnosis: review article. PLoS Negl Trop Dis 11:e0005638. https://doi.org/10.1371/journal.pntd.0005638
73. Cornely OA, Alastruey-Izquierdo A, Arenz D, Chen SCA, Dannaoui E, Hochhegger B, Hoenigl M, Jensen HE, Lagrou K, Lewis RE, et al. 2019. Global guideline for the diagnosis and management of mucormycosis: an initiative of the European Confederation of Medical Mycology in cooperation with the Mycoses Study Group Education and Research Consortium. Lancet Infect Dis 19:e405–e421. https://doi.org/10.1016/S1473-3099(19)30312-3
74. Donnelly JP, Chen SC, Kauffman CA, Steinbach WJ, Baddley JW, Verweij PE, Clancy CJ, Wingard JR, Lockhart SR, Groll AH, et al. 2020. Revision and update of the consensus definitions of invasive fungal disease From the European Organization for Research and Treatment of Cancer and the Mycoses Study Group Education and Research Consortium. Clin Infect Dis 71:1367–1376. https://doi.org/10.1093/cid/ciz1008
75. Hoenigl M, Salmanton-García J, Walsh TJ, Nucci M, Neoh CF, Jenks JD, Lackner M, Sprute R, Al-Hatmi AMS, Bassetti M, et al. 2021. Global guideline for the diagnosis and management of rare mould infections: an initiative of the European confederation of medical mycology in cooperation with the International society for human and animal mycology and the American Society for Microbiology. Lancet Infect Dis 21:e246–e257. https://doi.org/10.1016/S1473-3099(20)30784-2