

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 natural 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](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.

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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

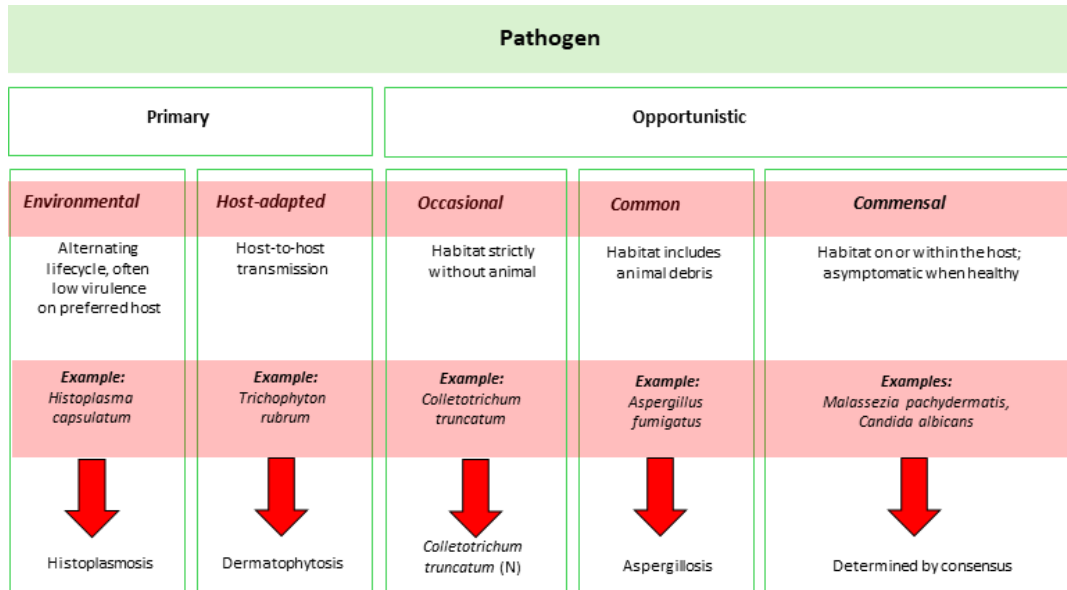


FIG 1 Types of pathogenicity of fungal pathogens in medical mycology.

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 or 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 <i>Emmonsia crescens</i>	
Aspergillosis	Tissue invasion or inflammation by <i>Aspergillus</i> 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 <i>Aspergillus</i> sinusitis Invasive <i>Aspergillus</i> sinusitis (<i>A. flavus</i> , <i>A. fumigatus</i>) Rhino-orbital-cerebral aspergillosis (ROCA) Disseminated ^b aspergillosis <i>Aspergillus</i> keratitis <i>Aspergillus</i> (Y ^c) infection Canine/feline sinonasal aspergillosis (SNA) 🦠 Feline sinoorbital aspergillosis 🦠
Basidiobolomycosis	Tissue invasion by <i>Basidiobolus</i> spp.	Gastrointestinal basidiobolomycosis Subcutaneous basidiobolomycosis
Black piedra	Sclerotium formation on hair shaft by <i>Piedraia</i> spp.	
Blastomycosis	Tissue invasion or inflammation by <i>Blastomyces</i> spp.	Pulmonary blastomycosis Disseminated ^b blastomycosis
Candidiasis	Tissue invasion or inflammation by common ascomycetous yeasts	Mucocutaneous candidiasis Cutaneous (intertriginous) candidiasis Vulvovaginal candidiasis (VVC) <i>Candida</i> balanitis Oropharyngeal candidiasis (OPC) Esophageal candidiasis <i>Candida</i> fungemia (candidemia) Device-related candidemia Acute disseminated ^b candidiasis Chronic disseminated ^b candidiasis <i>Candida</i> meningoencephalitis <i>Candida</i> endocarditis <i>Candida</i> cystitis Renal candidiasis Hepatosplenic candidiasis Intra-abdominal candidiasis (abscess ad peritonitis) <i>Candida</i> empyema <i>Candida</i> arthritis <i>Candida</i> osteomyelitis <i>Candida</i> chorioretinitis <i>Candida</i> keratitis
Chromoblastomycosis	Subcutaneous invasion with muriform cells by the order of Chaetothyriales	
Chytridiomycosis 🦠	<i>Batrachochytrium</i> disease of frogs and salamanders	
Coccidioidomycosis	Tissue invasion or inflammation with spherules by <i>Coccidioides</i> 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 <i>Conidiobolus</i> spp.	
Cryptococcosis	Tissue invasion or inflammation by <i>Cryptococcus</i> (s.str.) spp.	Pulmonary cryptococcosis Cerebral cryptococcosis <i>Cryptococcus</i> meningoenkephalitis <i>Cryptococcus</i> fungemia Disseminated ^b cryptococcosis Cryptococcoma Cutaneous cryptococcosis Sinoorbital cryptococcosis 🦠
Dermatophytosis	Tissue invasion or inflammation by dermatophyte (<i>Epidermophyton</i> , <i>Microsporum</i> , <i>Nannizzia</i> , <i>Trichophyton</i> spp.)	See this list under Tinea
Emergomycosis	Tissue invasion or inflammation by <i>Emergomyces</i> spp.	
Eumycetoma	Subcutaneous invasion with grains due to various fungi of widely different agents (<i>Madurella</i> , <i>Scedosporium</i> , <i>Trematosphaeria</i> , <i>Curvularia</i> , <i>Bipolaris</i> , <i>Exophiala</i> , <i>Rhinochadiella</i> , <i>Acremonium</i> , <i>Sarocladium</i> , <i>Fusarium</i> spp.)	
Fungemia	Blood-carried fungus, wide diversity	
Fusariosis	Hematogenous dissemination by <i>Fusarium</i> and relatives	Disseminated ^b fusariosis Pulmonary fusariosis <i>Fusarium</i> fungemia <i>Fusarium</i> keratitis <i>Fusarium</i> onychomycosis
Histoplasmosis	Tissue invasion by <i>Histoplasma</i> spp.	Acute pulmonary histoplasmosis Chronic pulmonary histoplasmosis Progressive disseminated ^b histoplasmosis Primary cutaneous histoplasmosis <i>Histoplasma</i> mediastinal fibrosis
	Tissue invasion by <i>Histoplasma duboisii</i>	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 <i>Lagenidium</i> ^d and <i>Paralagenidium</i> ^d spp.	
<i>Malassezia</i> dermatitis	Inflammation by <i>Malassezia</i> spp.	<i>Malassezia</i> seborrheic dermatitis <i>Malassezia</i> folliculitis <i>Malassezia</i> otitis <i>Malassezia</i> 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 <i>Ophidiomyces ophidiicola</i> in snakes	
Paracoccidioidomycosis PCM)	Tissue invasion by <i>Paracoccidioides</i> spp.	(Sub)acute paracoccidioidomycosis Chronic paracoccidioidomycosis
Paracoccidioidomycosis ceti 🦠	Tissue invasion by the unculturable <i>Paracoccidioides ceti</i> 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 <i>Paracoccidioides lobogeorgii</i> in humans (PCML)	
Penicilliosis 🦠	Disseminated infection in dogs by <i>Penicillium canis</i> and <i>P. labradorum</i>	
Phaeohyphomycosis	Tissue invasion with melanized hyphae of widely different agents	This term may be superfluous
Pityriasis versicolor	Colonization and inflammation by <i>Malassezia</i> spp.	Also commonly known as tinea versicolor
<i>Pneumocystis</i> pneumonia	Pulmonary infection by <i>Pneumocystis</i> spp.	
Protothecosis 🦠	Preponderantly bovine tissue inflammation by the algae <i>Prototheca</i> ^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 <i>Pythium</i> ^d spp. In humans, vascular and ocular pythiosis are common clinical manifestations.	
Rhinosporidiosis 🦠	Acanthosis by <i>Rhinosporidium</i> ^d spp. mainly in bird and horses	
Sporotrichosis	Tissue invasion by members of pathogenic clade of <i>Sporothrix</i> spp.	Lymphocutaneous sporotrichosis Fixed sporotrichosis Pulmonary sporotrichosis Disseminated ^b sporotrichosis Feline sporotrichosis 🦠
Talaromycosis	Disseminated intracellular infection by <i>Talaromyces marneffe</i>	
Tinea	Cutaneous infection by dermatophyte (<i>Microsporum</i> , <i>Trichophyton</i> , <i>Epidermophyton</i> , <i>Nannizzia</i> spp.)	Tinea capitis Tinea corporis Tinea cruris Tinea faciei Tinea manuum Tinea pedis
Tinea nigra	Cutaneous colonization by <i>Hortaea werneckii</i>	
White nose disease 🦠	Disease of hibernating bats	
White piedra	Growth on hair shaft by <i>Trichosporon</i> spp.	

^aDefinitions 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.

^bDisseminated refers to translocation to multiple sites other than the original site of infection.

^c(Y***) denotes the clinical picture concerned.

^dOrganisms that are non-fungal but conventionally treated under medical mycology.

🦠, (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 marneffe*. “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) <i>Cutaneotrichosporon cutaneum</i> [<i>Trichosporon cutaneum</i>] (Y) infection
(X) <i>Geotrichum candidum</i> (Y) infection
(X) <i>Magnusiomyces capitatus</i> [<i>Saprochaete capitata</i>] (Y) infection
(X) <i>Trichosporon asahii</i> (Y) infection
Filamentous fungi
(X) <i>Alternaria alternata</i> (Y) infection
(X) <i>Bipolaris</i> ^b (Y) cerebral infection
(X) <i>Cladophialophora bantiana</i> (Y) cerebral infection
(X) <i>Colletotrichum gloeosporioides</i> keratitis
Disseminated (X) <i>Phialophora verrucosa</i> (Y) infection
(X) <i>Fusarium oxysporum</i> (Y) infection
(X) <i>Lomentospora prolificans</i> (Y) infection
(X) <i>Madurella mycetomatis</i> mycetoma
(X) <i>Paecilomyces variotii</i> sinusitis
(X) <i>Scedosporium apiospermum</i> (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/>.

^bSome *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 *Rhinoctadiella* 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.

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AUTHOR AFFILIATIONS

¹Radboudumc-CWZ Centre of Expertise for Mycology, Nijmegen, the Netherlands

²Foundation Atlas of Clinical Fungi, Hilversum, the Netherlands

³Center for Innovative Therapeutics and Diagnostics, Richmond, Virginia, USA

⁴University of Maryland School of Medicine, Baltimore, Maryland, USA

⁵Mycology Reference Laboratory, Spanish National Centre for Microbiology, Madrid, Spain

⁶Centro de Investigación Biomédica en Red de Enfermedades Infecciosas, Instituto de Salud Carlos III, Madrid, Spain

⁷Department of Microbiology and Infection Control, Statens Serum Institut, Copenhagen, Denmark

⁸Department of Clinical Microbiology, Copenhagen University Hospital, Copenhagen, Denmark

⁹National Mycology Reference Laboratory, Public Health England, Bristol, United Kingdom

¹⁰Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead, Australia

¹¹Medical Mycology Unit, Department of Microbiology, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

¹²National Reference Laboratory for Antimicrobial Resistance in Fungal Pathogens, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

¹³Division of Infectious Diseases, Mount Auburn Hospital, and Harvard Medical School, Cambridge, Massachusetts, USA

¹⁴University of Cologne, Faculty of Medicine, Institute of Translational Research, Cologne, Germany

¹⁵Excellence Center for Medical Mycology, Department I of Internal Medicine, University of Cologne, Cologne, Germany

¹⁶Manchester Fungal Infection Group, Academic Health Science Centre, University of Manchester, Manchester, United Kingdom

¹⁷Laboratoire de Santé Publique du Québec, Institut National de Santé Publique du Québec, Sainte-Anne-de-Bellevue, Canada

¹⁸Department of Pathology, University of Texas Southwestern Medical Center, Dallas, Texas, USA

¹⁹Department of Mycology, Centre Hospitalier Universitaire de Rennes, Centre National de Référence Aspergilloses chroniques, ECMM Excellence Center in Mycology, Rennes, France

²⁰Unitat de Micologia i Microbiologia Ambiental, Facultat de Medicina i Ciències de la Salut, Universitat Rovira i Virgili, Reus, Spain

²¹Infectious Disease Research Program, Department of Pediatric Hematology and Oncology, University Children's Hospital Münster, Münster, Germany

²²Oniris, VetAgroBio Nantes, IRF, SFR ICAT, Université d'Angers, Angers, France

²³Laboratory Diagnostic Center, RWTH Aachen University Hospital, Aachen, Germany

²⁴Centre for Infectious Diseases and Microbiology Laboratory Services, Westmead, Australia

²⁵Royal Botanic Gardens, Kew, Richmond, United Kingdom

²⁶Natural History Museum, London, United Kingdom

²⁷University of Southampton, Southampton, United Kingdom

²⁸Jilin Agricultural University, Changchun, China

²⁹St. John's Institute of Dermatology, King's College London, London, United Kingdom

³⁰Division of Infectious Diseases, Medical University of Graz, Graz, Austria

³¹Translational Medical Mycology Research Unit, Medical University of Graz, Graz, Austria

³²Department of Botany, Charles University, Prague, Czechia

³³Department of Medical Microbiology, University of Warsaw, Warsaw, Poland

³⁴Westerdijk Fungal Biodiversity Institute, Utrecht, the Netherlands

³⁵National Mycology Reference Centre, SA Pathology, Adelaide, Australia

³⁶School of Biological Sciences, Faculty of Sciences Engineering and Technology, University of Adelaide, Adelaide, Australia

³⁷Public Health Ontario Toronto, Toronto, Canada

³⁸Department of Laboratory Medicine and Pathobiology, University of Toronto, Toronto, Canada

³⁹National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland, USA

⁴⁰Centers for Disease Control and Prevention, Atlanta, Georgia, USA

⁴¹Microbiology and Molecular Genetics, Biomedical Laboratory Diagnostics, Michigan State University, East Lansing, Michigan, USA

⁴²University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

⁴³Department of Dermatology, Peking University First Hospital, Peking University, Beijing, China

⁴⁴Sydney Infectious Diseases Institute, Faculty of Medicine and Health, University of Sydney, Sydney, Australia

⁴⁵Bioinformatics, Helmholtz Institute for One Health, Greifswald, Germany

⁴⁶CONICET (Consejo Nacional de Investigaciones Científicas y Tecnológicas), Hospital JM Ramos Mejía, Buenos Aires, Argentina

⁴⁷Division of Clinical Microbiology, Mayo Clinic, Rochester, Minnesota, USA

⁴⁸Public Health Wales Microbiology, Cardiff, United Kingdom

⁴⁹McGovern Medical School, Division of Infectious Diseases, University of Texas Health Science Center, Houston, Texas, USA

⁵⁰Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

AUTHOR ORCID*s*

Sybre 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

Sybre 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.

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