

The Discovery and Naming of *Candida albicans*

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“In no other group of medically important fungi is there any greater confusion than that concerned with the classification of the non-ascosporogenous mycelia-producing yeastlike organisms known to the medical men as the *Monilias*” (Martin and Jones, 1939).

The history of the discovery and naming of *Candida* extends from the ancient Greeks to modern day researchers. The perception of *Candida* has evolved from the presence of an exudate in the human host to a known infectious agent. 200 years of medical history was recorded before the etiological agent of oral thrush, the first form of candidiasis described, was correctly identified as a fungal pathogen. “Thrush” appears as whitish plaques within the oropharynx or the buccal mucosa or tongue. One of the main points of contention when defining thrush was whether it originated from the host or was an infectious agent, or a combination of the two.

The earliest reports of thrush predated the concept of a microbial pathogen. In “Of the Epidemics,” Hippocrates described oral candidiasis (around 400 B.C.) as “mouths affected with aphthous ulcerations” (5). In 1665, Pepys Diary reported “a patient hath a fever, a thrush and a hiccup” (8), perpetuating the idea that oral thrush originates from the host. Mycologists accepted this perception as late as the early 1900s where Castellani quoted previous accounts of thrush as “morbid secretions of the oral mucosa” (2).

However, a few clinicians and mycologists swayed popular belief towards the idea of an infectious agent causing thrush. In 1771, Rosen von Rosenstein defined an invasive form of thrush (2). In 1839, Langenbeck was credited with first recognizing a fungus in a patient with typhoid fever. Oropharyngeal and esophageal thrush with pseudomembranes were found at autopsy. “Under the microscope magnified, the pseudomembranes consisted of an immense number of fungi” (translated from German) (7). He describes in detail what is now referred to as septate hyphae, branched pseudohyphae and blastoconidia. However, he ascribed the entity to the typhoid bacterium rather than the fungus (7). In 1844, J.H. Bennett observed a similar fungus in the sputum and the lungs of a patient with a pneumothorax and criticized the conclusion by Langenbach (2). The morphologic description of Bennett was essentially that as described by Langenbeck. Bennett concluded that the disease was “indicative of great depression of the vital powers and impairment of the nutritive functions of the economy [host?]” (2). Two years later, Berg explicitly concluded that thrush was caused by a fungus and found that spread could occur from communal feeding bottles. Most importantly, he also stated “descriptions of the disease unsupported by demonstration of the fungus could not substantiate the diagnosis”. He was able to reproduce the infection in healthy children and thereby confirmed his hypothesis that the fungus caused the infection (2). After this discovery, other infections would be ascribed to this dimorphic fungus including vaginitis

and gastrointestinal candidiasis. Once the etiology was conclusively demonstrated by mycologists, the next point of contention was the identity of the pathogen.

While Langenbeck (1839) first documented the fungus associated with thrush, he failed to make the direct connection. In 1847, the distinguished French mycologist, Charles Philippe Robin, classified the fungus as *Oidium albicans* (6) using *albicans* (“to whiten”) to name the fungus causing thrush. Hill (4) and later Martin and Jones (8) misclassified *Candida* into the genus *Monilia*, a genus containing fungi that commonly grow in plants. Subsequently, clinicians erroneously referred to the etiology of thrush as “*Monilias*” despite the fact mycologists had already elucidated the morphological differences between the fungus associated with thrush and the fungus in the genus *Monilia*. Christine Berkhout and others noted these differences, particularly the ability of this fungus to infect humans. Berkhout reclassified it under the current genus *Candida* (1923) (1). *Candida* is derived from Latin where *toga candida* was a white robe worn by Roman Senators. Berkhout’s taxonomy was later heralded by the prominent French mycologists, Maurice Langeron and Paul Guerra, as “...the beginning of the rational systematics of the non-ascosporogenous yeasts” (1). However, it was not until 1954 that the Eighth Botanical Congress officially endorsed the binomial *Candida albicans* as the *nomen conservandum* formally ending the 200 year long uncertainty over the etiology and taxonomy of *Candida*.

Currently, there are some 200 organism species within the genus *Candida*. These yeast-like cells are anamorphic (sexual imperfect) fungi belonging to the form-class Blastomycetes. They are characterized by their polymorphic nature and ability to produce budding yeast cells (blastoconodia), mycelia, pseudomycelia, and blastospores (3). Of the nearly 200 species, six species, *C. albicans*, *C. glabrata*, *C. tropicalis*, *C. parapsilosis*, *Candida krusei*, and *C. lusitaniae* are the most commonly associated with human infection.

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