# Pathogenesis, Diagnosis, and Treatment of Amphibian Chytridiomycosis

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**KEYWORDS**

• *Batrachochytrium dendrobatidis* • Chytridiomycosis • Amphibian • Treatment
• Diagnosis

**KEY POINTS**

- Chytridiomycosis causes death in amphibians by disrupting osmoregulatory function of the skin.
- Polymerase chain reaction testing is the diagnostic method of choice and should be used both to confirm infection and confirm negative status after treatment.
- Itraconazole is the most commonly used antifungal agent for treatment of chytridiomycosis.
- Successful treatment of amphibians that are clinically ill with chytridiomycosis requires supportive care, especially with electrolyte therapy.

## INTRODUCTION

Amphibian chytridiomycosis, caused by the chytridiomycete fungus, *Batrachochytrium dendrobatidis* (Bd), is responsible for a global pandemic that has dramatically reduced global amphibian populations and diversity.[1–4] Species declines, extirpations, and extinctions attributed to chytridiomycosis have occurred in Australia, Europe, Latin America, and the United States.[5–8] The geographic origin of Bd is the subject of ongoing investigation. However, the spread of virulent Bd lineages to all continents (other than Antarctica) is largely believed to be caused by global trade in amphibians for food and research.[9–12] Global dissemination of this important

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pathogen has led to its designation as an internationally notifiable disease by the World Organization for Animal Health (OIE), making it subject to OIE standards in international trade.[13]

Veterinarians are engaged in response to this crisis through assistance with treatment, research, and conservation programs taking place at zoos, aquaria, international ex situ assurance colonies, and private hobby and agricultural industry. There has been a proliferation of reports published recently that describe research on Bd, its pathogenesis, and treatment. This review focuses on recent advances in information concerning Bd, with emphasis on diagnosis, clinical response, and treatment.

### *Amphibian Skin Physiology*

Understanding of normal skin physiology in amphibians is important in understanding the pathophysiology and treatment of chytridiomycosis as well as several other amphibian diseases.

Amphibian skin is highly adapted and arguably the most important organ in amphibian anatomy. Very permeable and well vascularized, the skin is a primary route of respiratory gas exchange and water and electrolyte balance.[14,15] In most species, the area of highest water uptake is the ventral pelvic region, also referred to as the pelvic or drink patch. Sodium, chloride, and calcium ions are actively transported from the environment in to the amphibian body through the skin via several mechanisms, including selective epithelial channels and ion pumps.[14] Potassium is passively regulated based on concentration differences across cell membranes.[14] Disruption of these processes can quickly lead to morbidity and mortality.[16,17]

## CHYTRIDIOMYCOSIS

### *Life Cycle of Bd*

The infective stage, a flagellated motile zoospore, encysts on the surface of stratified keratinizing epithelial cells of frog skin, the flagellum is resorbed, and a cell wall is formed. The cyst wall elongates to form a germination tube that penetrates a skin cell membrane within deeper cell layers beneath the stratum corneum.[18,19] Host cell cytoplasm is digested and the contents of the encysted zoospore migrate through the germ tube and develop into an intracellular thallus. The thallus matures from sporangium to zoosporangium, the contents of which divide to form more flagellated zoospores. As the zoosporangium is maturing, superficial stratum corneum cells are being shed, accelerated by effects of infection, and infected cells are brought to the skin surface.[18,20] Simultaneously, the thallus forms a discharge papilla that extends through the skin cell surface, from which the motile zoospores exit and disperse once sufficient moisture is present for zoospore migration. Established thalli can also directly infect deeper cells via projection of rhizoidlike structures that form new sporangia in a similar manner as described for the germination tube.[19]

### *Pathophysiology*

Infection with Bd is limited to the keratinizing stratified squamous epithelium that comprises the skin of postmetamorphic amphibians and the mouthparts of larvae (tadpoles). The skin of larval amphibians begins to keratinize during metamorphosis, and mortality as a result of chytridiomycosis is often observed at this time as the fungus moves from the mouthparts to colonize the skin.[21] In susceptible species, as motile zoospores are released and spread out on the surface of the skin, local reinfection quickly leads to exponential increase in zoospore burden and infection intensity.[22] Initial infection in terrestrial amphibians is on those skin surfaces in contact with the