Dear Editor,

I read an interesting recent article by the Colombian authors Muñoz JF et al., (2018) about the evolutionary mechanisms of adaptation in systemic dimorphic fungi. Based on the emergence of *Emmonsia*-like species causing systemic human mycoses worldwide, the authors selected one strain of *Ea. parva* (UAMH130; CBS139881; type strain) from the lungs of a rodent in the USA and one additional strain of *Ea. crescens* (UAMH4076; CBS139868) from a greenhouse source in Canada. By sequencing the genomes of *Ea. parva* and *Ea. crescens*, they evaluated how the changes in their gene content can be correlated with transitions to the pathogenesis in mammals. Their study revealed that *Ea. parva* isolates (UAMH130 and UAMH139) do not constitute a single well-defined clade, therefore confirming that *Ea. parva* may not be a single species.

Dimorphism is a morphogenetic phenomenon by which some fungi can both grow in the environment, as well as to become pathogens for animal and human hosts. Among the dimorphic fungi, the *Ajellomycetaceae* family includes some rarely pathogenic species as *Ea. parva* and *Ea. crescens*, which undergo a thermal transition to produce adiaspores instead of yeasts, and may cause the disease adiaspiromycosis. Worthy of note, compared with the soil or the animal excrements, human hosts constitute a very different habitat for these fungi, and interactions with the environment may origin a capacity for survival in animal hosts and become pathogenic to humans.

In this scenery, one must emphasize the growing number of reports about lung adiaspiromycosis affecting diverse mammals and other animals all over the world. Hughes K and Borman AM (2018) described an *Oryctolagus cuniculus* with pulmonary and tracheobronchial lymph node adiaspiromycosis and reviewed the related literature. Tissue samples stained by hematoxylin and eosin (H&E), periodic acid–Schiff (PAS), and Grocott methenamine silver (GMS) showed adiaspores with a bi- or trilaminar wall (a thin brightly eosinophilic outer layer; a thick pale eosinophilic layer; and a variabe inner basophilic layer, surrounding a core of basophilic granular-to-foamy material. Therefore, the final diagnosis was consistent with infection caused by *Ea. crescens*; nevertheless, microdissection of adiaspores, DNA purification, and PCR amplification utilizing *Emmonsia* specific primers failed to identify the *Emmonsia*-specific DNA. The authors highlighted the possibility...
that the confirmation of the etiologic agent of this rare mycosis by PCR using the formalin fixed tissue may not be possible in all cases.

Finally, comments are added about the Brazilian contribution to the morphologic diagnosis of pulmonary adiaspiromycosis utilizing mucicarmine, picro-sirius, and Congo red, in addition to the routine methods. By mucicarmine the inner and middle layers of the wall are discreetly positive, without birefringence to polarized light; with picro-sirius the wall has orange birefringence to polarized light; by red Congo there are more than three layers in the wall, with intense yellowish birefringence in polarized light; and phase contrast microscopy may reveal clear trilaminar wall structure, even in H & E. Data herein included can be useful to solve diagnostic challenges mainly in low-income regions where more expensive resources are not available in daily practice.

References