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Carta al editor

Pulmonary adiaspiromycosis – yet a challenging condition?
Adiaspiromicose pulmonar - ainda uma condição desafiadora?

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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[1]. 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[1].

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[1]. 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[1]. 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[1].

In this scenery, one must emphasize the growing number of reports about lung adiaspiromycosis affecting diverse mammals and other animals all over the world[1-5]. Hughes K and Borman AM (2018) described an *Oryctolagus cuniculus* with pulmonary and tracheobronchial lymph node adiaspiromycosis and reviewed the related literature[3]. 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

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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[3].

Finally, comments are added about the Brazilian contribution to the morphologic diagnosis of pulmonary adiaspiromycosis utilizing mucicarmin, 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[5]. 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.

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