Melanin is an essential component for the integrity of the cell wall of Aspergillus fumigatus conidia

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Résumé en anglais	**BACKGROUND**: Aspergillus fumigatus is the most common agent of invasive aspergillosis, a feared complication in severely immunocompromised patients. Despite the recent commercialisation of new antifungal drugs, the prognosis for this infection remains uncertain. Thus, there is a real need to discover new targets for therapy. Particular attention has been paid to the biochemical composition and organisation of the fungal cell wall, because it mediates the host-fungus interplay. Conidia, which are responsible for infections, have melanin as one of the cell wall components. Melanin has been established as an important virulence factor, protecting the fungus against the host's immune defences. We suggested that it might also have an indirect role in virulence, because it is required for correct assembly of the cell wall layers of the conidia. **RESULTS**: We used three A. fumigatus isolates which grew as white or brown powdery colonies, to demonstrate the role of melanin. Firstly, sequencing the genes responsible for biosynthesis of melanin (ALB1, AYG1, ARP1, ARP2, ABR1 and ABR2) showed point mutations (missense mutation, deletion or insertion) in the ALB1 gene for pigmentless isolates or in ARP2 for the brownish isolate. The isolates were then shown by scanning electron microscopy to produce numerous, typical conidial heads, except that the conidia were smooth-walled, as previously observed for laboratory mutants with mutations in the PKSP/ALB1 gene. Flow cytometry showed an increase in the fibronectin binding capacity of conidia from mutant isolates, together with a marked decrease in the binding of laminin to the conidial surface. A marked decrease in the electronegative charge of the conidia and cell surface hydrophobicity was also seen by microelectrophoresis and two-phase partitioning, respectively. Ultrastructural studies of mutant isolates detected considerable changes in the organisation of the conidial wall, with the loss of the outermost electron dense layer responsible for the ornamentations seen on the conidial surface in wild-type strains. Finally, analysis of the conidial surface of mutant isolates by atomic force microscopy demonstrated the absence of the outer cell wall rodlet layer which is composed of hydrophobins. **CONCLUSION**: These results suggest that, in addition to a protective role against the host's immune defences, melanin is also a structural component of the conidial wall that is required for correct assembly of the cell wall layers and the expression at the conidial surface of adhesins and other virulence factors.
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