REGULATION OF HYPHAL MORPHOGENESIS AND THE DNA DAMAGE RESPONSE BY THE ASPERGILLUS NIDULANS ATM HOMOLOG ATMA.

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Ataxia telangiectasia (A-T) is an inherited disorder characterized by progressive loss of motor function and susceptibility to cancer. The most prominent clinical feature observed in A-T patients is the degeneration of Purkinje motor neurons. Numerous studies have emphasized the role of the affected gene product, ATM, in the regulation of the DNA damage response. However, in Purkinje cells, the bulk of ATM localizes to the cytoplasm and may play a role in vesicle trafficking. The nature of this function, and its involvement in the pathology underlying A-T, remain unknown. Here we characterize the homolog of ATM (AtmA) in the filamentous fungus Aspergillus nidulans. In addition to its expected role in the DNA damage response, we find that AtmA is also required for polarized hyphal growth. We demonstrate that an atmA mutant fails to generate a stable axis of hyphal polarity. Notably, cytoplasmic microtubules display aberrant cortical interactions at the hyphal tip. Our results suggest that AtmA regulates the function and/or localization of landmark proteins required for the formation of a polarity axis. We propose that a similar function may contribute to the establishment of neuronal polarity.

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