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## Expression of the *Aspergillus fumigatus* α-1, 2-mannosidase gene (*MsdS*) in *Trichoderma reesei* affects cell wall synthesis and polarized growth

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**Statement of the problem:**  $\alpha$ -1, 2-mannosidase is a very important enzyme, essential for N-glycan processing and plays a significant role in the biosynthesis and organization of fungal cell wall. Lack of  $\alpha$ -1, 2-mannosidase has been observed to cause cell wall defect in yeast and filamentous fungi. *Trichoderma reesei* is known to be non-toxic to human, and its N-glycan on mature secretory glycoprotein is GlcNAc2Man8, which is different from the GlcNAc2Man6 in the wild-type *Aspergillus fumigatus* and similar to the glycoform of the *MsdS* deletion mutant, which was characterized by cell wall defect and polarized growth.

**Methodology & Theoretical Orientation:** To gain insight into the physiological function of the N-glycan processing in *T. reesei*, in this study the *A. fumigatus* α-1, 2-mannosidase MsdS was introduced into *T. reesei*.

**Findings:** The mutant strain expressing MsdS produced a major glycoform of GlcNAc2Man6 on its secretory glycoproteins, instead of GlcNAc2Man8 in the wild-type. Although the cell wall content of the mutant was changed, it appeared that its cell wall integrity was not affected. However, multiple budding and random branching was observed in the mutant. In addition, the mutant showed less dense filamentous material on cell surface under favorable growth condition, while a complete loss of filamentous material accompanied by an increase of cell wall chitin was observed at elevated temperatures.

**Significance & Conclusion:** Our results, for the first time, indicated that processing of the N-glycan might play a major role in sorting and polarized transportation of certain glycoproteins in *T. reesei*, which is vital for the cell wall synthesis and polarity. Further, our results also indicated that the mechanism of the N-glycan-related sorting of glycoprotein in *T. reesei* is different from that in *A. fumigatus*.

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