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# Modulation of Sexual Development in Association of Newly Isolated IndB/D with a GATA Transcription Factor, NsdD in *Aspergillus nidulans*

Nak-Jung Kwon* and Suhn-Kee Chae

*Department of Biochemistry and Bio-med RRC, PaiChai University*

NsdD is an auto-regulated putative GATA transcription factor with a zinc finger domain and plays an important role as a positive regulator during sexual development of the filamentous fungus, *Aspergillus nidulans*. Three interacting proteins of NsdD were isolated using the yeast two-hybrid assay and named IndA, IndB, and IndD (Interactor of NsdD). Sequencing analysis of their cDNAs predicted that the *indA, indB,* and *indD* genes encoded 335, 268, and 282 amino acids, respectively. IndA displayed sequence homology to the FAR1 family of plant nuclear proteins involved in phytochrome signal transduction, yet IndB or IndD showed no homology to any genes or proteins characterized to date. Interactions of NsdD-IndB and NsdD-IndD were verified *in vitro* and *in vivo* using a GST-pull down assay and co-immunoprecipitation, respectively. Furthermore, mapping of NsdD interaction domain(s) demonstrated that IndB and IndD bound to the zinc finger domain of NsdD. Other GATA type proteins of LreA, LreB, and AreA failed to interact with IndB/D, indicating that NsdD-IndB and -IndD interactions are specific. *In vitro* and *in vivo* chromatin immunoprecipetation (ChIP) assay revealed that binding of IndB or IndD to NsdD inhibited auto-regulation of NsdD by binding to the GATA elements in its own promoter. Vegetative growth was reduced in *ΔindD* but not in *ΔindB*. Null mutants of *indB* or *indD* were normal in sexual development, however, mutants disrupted both in *indB* and *indD* displayed uncontrolled cleiostothecial formation during sexual development, resulting in continuing production of mature and immature cleistothecia. In contrast, over-expression of IndB or IndD blocked sexual development in $veA^{+}$ strains. Interestingly, expression levels of *indB* and *indD* were significantly increased in the sexually-impaired *ΔveA* mutant. Thus, the novel proteins, IndB and IndD, function as negative regulators of sexual development through interaction with NsdD. The incresed expression of *indB* and *indD* may explain the defective sexual development in the *ΔveA* mutant.