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REVIEW

MybA, a new player driving survival of the conidium of the human pathogen *Aspergillus fumigatus*

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Abstract *Aspergillus fumigatus* is an opportunistic human pathogen that causes various complications in patients with a weakened immune system functions. Asexual spores of *A. fumigatus* are responsible for initiation of aspergillosis. Long-term viability and proper germination of dormant conidia depend on trehalose accumulation, which protect the spores against thermal and oxidative stress. A putative Myb transcription factor, MybA has been recently found to be responsible for a variety of physiological and molecular roles ranging from conidiation, spore viability, trehalose accumulation, cell wall integrity and protection against reactive oxygen species. In this perspective review, we discuss the recent findings of MybA and its overlapping functions with the other regulators of conidia viability and trehalose accumulation. Therefore, the aim of this perspective is to raise interesting and stimulating questions on the molecular functions of MybA in conidiation and trehalose biogenesis and to question its genetic and physical interactions with the other regulators of conidial viability.

Keywords MybA · Spore viability · VelB-VosA · Cell wall · *Aspergillus fumigatus* · AtfA · WetA

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MybA background

Aspergillus fumigatus is a ubiquitous saprophytic filamentous fungus normally found in the soil in decaying vegetal materials (Dagenais and Keller 2009; Nierman et al. 2005; Sugui et al. 2014). Asexual sporulation is a major means of propagation and dispersal in natural habitats of the fungus. Conidia produced by this fungus can stay several months to a year dormant in the environment and capable of germinating into a mesh of hyphae upon landing on an appropriate substrate. Small size and abundant number of *A. fumigatus* conidia found in air help them to reach into deep alveolar cavities of animals and humans (Bultman et al. 2017; Cramer 2016). Especially, this landing of conidia into alveolar surfaces can lead to life threatening systemic diseases in individuals with a weakened immune system and serious allergic reactions such as Allergic broncho pulmonary aspergillosis in immunocompetent individuals (Heinekamp et al. 2015; Latge 2001). Germination of conidia in alveolar membrane is a key process in systemic aspergillosis and requires complex interaction of conidia with environment and alveolar macrophages (Amin et al. 2014). Long-term survival of the dormant conidia depends on accumulated osmolytes such as trehalose to cope with environmental as well as internal stressors such as reactive oxygen species (ROS) (Al-Bader et al. 2010; Eleutherio et al. 2015; Thammahong et al. 2017).

MybA is the first myb type transcription factor identified and characterized from opportunistic human pathogen *A. fumigatus* (Valsecchi et al. 2017). Deletion of *mybA* gene in this fungus leads to several important phenotypes. (1) Major defects are seen in sporulation processes in the absence of MybA transcription factor. Conidiation capacity of *mybA* mutant is drastically reduced (40 times less than a wild type strain), which is even more severe at higher temperatures

(50 °C). (2) Furthermore, *mybA* mutant loses its conidial viability (measured as an ability of each spores to germinate). This defect stem from the low levels of osmolyte (disaccharide trehalose, polyols mannitol and arabitol) accumulation in conidia of *mybA* mutant. (3) Cell wall organization of conidia is modified in *mybA* mutant due to the modification of the expression of many genes involved in cell wall metabolism leading to cell wall permeability defects in the conidia of the mutant. (4) Intracellular reactive oxygen species (ROS) levels soar in *mybA* mutant due to the fact that ROS scavenging enzymes such as catalases and superoxide dismutases are downregulated in *mybA* mutant. (5) *mybA* deletion strain shows reduced virulence in experimental aspergillosis murine model. In this mini review, we will discuss several overlapping functions and connections among MybA transcription factor, velvet family regulators VosA-VelB, asexual regulator WetA and stress response regulator AtfA as well as its upstream kinases SakA-MpkC and PbsB.

Conidial viability program: MybA, AtfA, VosA-VelB, WetA, PbsB-MpkC-SakA

In Aspergilli, asexual sporulation program is mainly governed by a cascade of three transcription factors BrlA, AbaA and WetA (Etxebeste et al. 2010; Oiartzabal-Arano et al. 2016; Park and Yu 2016; Rohrig et al. 2013). BrlA is a key component for conidiophore development (Alkhayyat et al. 2015; Mah and Yu 2006). C_2H_2 type transcription factor BrlA is necessary for appropriate expression of *abaA*, *wetA* and velvet family genes *vosA* and *velB* (Tao and Yu 2011). BrlA activates AbaA transcription factor that is important for separation of clonal asexual spores from each other. AbaA finally controls expression of WetA transcription factor, which is required for the last step of conidiogenesis. Expression of *wetA* increases at late stage of conidiation. Lack of *wetA* results in defective cell wall and reduced trehalose levels (Tao and Yu 2011). Several years ago, two velvet family proteins VosA-VelB were found to be responsible for trehalose accumulation, conidial viability and thermotolerance of conidia in *Aspergillus nidulans* (Ni and Yu 2007; Sarikaya Bayram et al. 2010). Both genes have AbaA response element (ARE) in their promoters and *abaA* is important for their expression. Later, they were also found to be essential for conidial viability and stress response in *A. fumigatus* (Park et al. 2012). *mybA* also positively affects expression of WetA and VelB-VosA heterodimer (Valsecchi et al. 2017). Although MybA was shown to play an important role in the regulation of conidiation (Valsecchi et al. 2017), MybA is totally independent of BrlA and AbaA.

Not many regulators specifically controlling conidia viability and trehalose accumulation have been identified in *A. fumigatus*. Fission yeast stress transcription factor Atf1 homolog, AtfA and its upstream kinases PbsB, MpkC-SakA, conidial regulator WetA and the velvet family proteins VelB-VosA are the regulatory proteins which are involved in conidial viability, trehalose biogenesis and response to oxidative stress (Hagiwara et al. 2014; Park et al. 2012; Sarikaya Bayram et al. 2010; Tao and Yu 2011). Interestingly, the role of MybA in conidiation and trehalose biogenesis overlaps with these regulators. AtfA and its upstream connecting kinases PbsB, MpkC-SakA, which are central part of high osmolarity glycerol (HOG) pathway, are involved in trehalose accumulation, thermotolerance, oxidative stress response and conidial viability (Hagiwara et al. 2008, 2014; Lara-Rojas et al. 2011). It is also interesting that two kinases MpkC and SakA have redundant functions for trehalose accumulation and conidial viability. Because only double deletion of them result in drastically reduced conidial viability as well as trehalose accumulation (Hagiwara et al. 2014). Surprisingly, *mpkC*/*sakA* double mutant show a reduced virulence in a murine model of experimental aspergillosis, underlining that trehalose accumulation and virulence is highly coordinated processes (Bruder Nascimento et al. 2016). Furthermore, in *A. nidulans* SakA interact with AtfA and MpkC under oxidative stress conditions (Jaimes-Arroyo et al. 2015; Lara-Rojas et al. 2011). Although many stress response genes are downregulated in *atfA* mutant in *A. fumigatus*, trehalose biosynthetic genes are not downregulated. Therefore, reduced trehalose levels were attributed to posttranslational modifications of trehalose synthesizing enyzmes (Hagiwara et al. 2014).

The velvet domain transcription factors were studied in many fungi (Bayram and Braus 2012; Calvo 2008), however, more mechanistic knowledge come from the model filamentous fungus *A. nidulans* which is closely related to *A. fumigatus* (Sarikaya-Bayram et al. 2015). There are four of them, founding member VeA, VelB-VosA heterodimer and VelC. VeA is required for sexual fruit body formation, asexual sporulation and secondary metabolite production in *A. nidulans* but it is not vital for conidial viability and trehalose biogenesis (Kim et al. 2002; Park et al. 2012; Sarikaya Bayram et al. 2010). VeA-VelB together with a methyltransferase LaeA constitutes a heterotrimeric velvet complex which controls fungal development and secondary metabolite production in *A. nidulans* (Bayram et al. 2008). VelB-VosA form a heterodimer in *A. nidulans* and both proteins are required for viability of conidia both in *A. nidulans* and *A. fumigatus*. VelB-VosA heterodimer is mostly active during vegetative growth and required for expression of trehalose biosynthetic genes (*tps*), which support trehalose accumulation in conidia (Sarikaya Bayram et al. 2010). Furthermore, in *A. nidulans* they bind to promoters of conidiation genes as well as *tpsA* and *treA* genes responsible for trehalose biosynthesis (Ahmed et al. 2013).

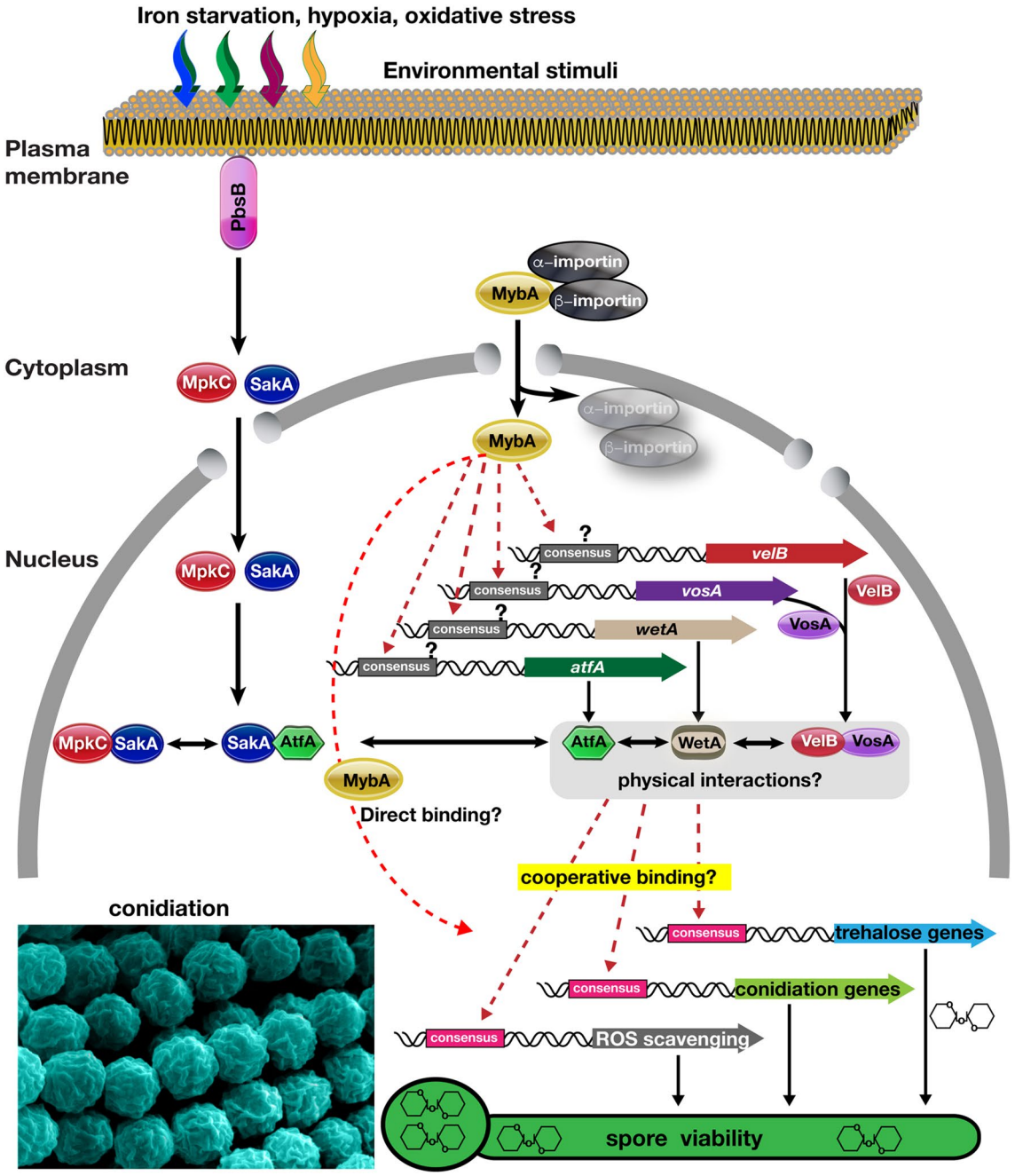

Fig. 1 Current picture of molecular control of spore viability regulators in Aspergilli. The model was drawn based on the current literature from both *A. nidulans* and *A. fumigatus*. There are two levels of control in spore viability network. (1) Upstream elements: yeast Pbs22 homolog PbsB MAP Kinase Kinase (MAPKK), and two downstream MAP kinases (MAPKs) of HOG pathway SakA and MpkC initiate the signal for trehalose accumulation. Possible signals may include various environmental stimuli (shown as *colored arrows*). MpkC and SakA interact with each other and SakA additionally interact with stress transcription factor AtfA, which possibly activate the AtfA itself. (2) Downstream regulators: BrlA and AbaA central pathway (not shown here) turn on expression of at least *vosA-velB* and *wetA* transcription factors. MybA interacts with two nuclear importins and turn on expression of *velB-vosA*, *wetA* and *atfA* by possibly binding to the promoters of these genes. MybA might also bind to the promoters of further downstream conidiation and trehalose genes. There might be physical interactions among these regulators. These transcription factors either individually or cooperatively induce expression of genes involved in conidiation, trehalose biogenesis, cell wall, ROS scavenging which all together contribute to spore viability, stress tolerance and virulence. Please see further discussion in the text

Transcripts of *velB-vosA* heterodimer, *wetA* and *atfA* are all downregulated in the absence of *mybA*. Further evidence for this regulation comes from a reporter assay where lack of MybA causes a reduction in expressions of VelB-VosA heterodimers as well as WetA transcription factor in this reporter assay in *A. fumigatus*. However, *atfA* promoter was overlooked in this study although its expression was downregulated at the same level with *vosA-velB* heterodimer in an RNA-seq study (Valsecchi et al. 2017). It is highly possible that there is a consensus binding sequence in the promoters of these genes responsive to MybA (Fig. 1). There are two palindromic sequences (CAGTT and AACTG) in promoters of *wetA*, *velB* and *vosA* identified. *atfA* promoter has a degenerate palindromic sequence. It is likely that MybA binds to these consensus sequences or other currently unknown motifs to activate these genes. However, functionality of these consensus sequences and binding of MybA remain to be shown.

Besides similar/overlapping functions of these regulators, there are also divergent control mechanisms for conidiation network. First of all, expression of *mybA* is not influenced by the lack of major asexual regulators BrlA and AbaA whereas *velB* and *vosA* are influenced by BrlA and AbaA. Furthermore, AbaA binds to the promoter of *velB* in vitro (Tao and Yu 2011). Normally, Aspergilli do not sporulate under liquid culture conditions and only able to form conidiophore on air surfaces. *velB* and *vosA* mutants both show a derepressed asexual development in liquid media whereas *mybA* mutant does not show any sign of conidiation in submerged medium. There has not been any report how *atfA*, *mpkC*, *sakA* and *pbsB* mutants behave in submerged liquid medium yet.

What is about the physical interactions between these regulators? It was shown in MybA-GFP and -HA pull-downs that MybA does not physically interact with VosA-VelB heterodimer, WetA, AtfA or any of MpkC-SakA kinase system. VosA-VelB heterodimer and SakA affinity purifications do not recruit MybA homolog in *A. nidulans* (Jaimes-Arroyo et al. 2015; Sarikaya Bayram et al. 2010). However, WetA, MpkC interaction partners remain to be shown both in *A. nidulans* and *A. fumigatus*. The lack of interaction does not completely exclude that they will not interact in other means or conditions, because MybA pull-downs were performed from 24-h grown vegetative hyphae. It is still likely that these proteins might interact with each other transiently, which could not be identified via pull-down and mass spectrometry or these interactions take place at a short time window during late vegetative or early asexual phase. Furthermore, their interactions might also require several stress conditions such as oxidative or osmotic stress.

What is next?

Conidial viability is an essential part of survival of *A. fumigatus* and other fungi. Moreover, conidia, which are resistant to harsh environmental conditions, are the initiators of aspergillosis by germinating and passing through blood barriers in lung tissues. Discovery of MybA showed once more that trehalose biogenesis influences conidial viability and virulence in *A. fumigatus*. MybA together with VelB-VosA heterodimer, WetA and AtfA-MpkC-SakA-PbsB system are four important players of osmolyte regulation and conidial viability in *A. fumigatus*. It will be important to learn genome wide binding sites of MybA as well as VelB-VosA heterodimer, WetA and AtfA by doing a chromatin immunoprecipitation (ChIP) and high throughput sequencing. However, there is currently only one paper reporting ChIP results in *A. fumigatus*, which might not be an easy task to apply in this fungus (Chung et al. 2014). ChIP will reveal the binding motif of these regulators and their target genes in the genome. It will be also interesting to reveal whether these regulators bind to some promoters such as trehalose biosynthetic genes in a cooperative manner. It will be also interesting to know the upstream regulators (e.g., protein kinases or mitogen-activated kinases (MAPK)) of conidial viability and trehalose accumulation and their interaction with the downstream elements. HOG pathway (Fig. 1) is an important candidate for the upstream regulation because there is strong evidence that this pathway governs the stress responses together with trehalose biogenesis in yeast and *Aspergilli* (Ho and Gasch 2015). However, solid links should be established in near future between the MpkC-SakA and MybA.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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