An Alba-domain protein required for proteome remodelling during trypanosome differentiation and host transition

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The transition between hosts is a challenge for digenetic parasites as it is unpredictable. For Trypanosoma brucei subspecies, which are disseminated by tsetse flies, adaptation to the new host requires differentiation of stumpy forms picked up from mammals to procyclic forms in the fly midgut. Here we show that the Alba-domain protein Alba3 is not essential for mammalian slender forms, nor is it required for differentiation of slender to stumpy forms in culture or in mice. It is crucial, however, for the development of T. brucei procyclic forms during the host transition. While steady state levels of mRNAs in differentiating cells are barely affected by the loss of Alba3, there are major repercussions for the proteome. Mechanistically, Alba3 aids differentiation by rapidly releasing stumpy forms from translational repression and stimulating polysome formation. In its absence, parasites fail to remodel their proteome appropriately, lack components of the mitochondrial respiratory chain and show reduced infection of tsetse. Interestingly, Alba3 and the closely related Alba4 are functionally redundant in slender forms, but Alba4 cannot compensate for the lack of Alba3 during differentiation from the stumpy to the procyclic form. We postulate that Alba-domain proteins play similar roles in regulating translation in other protozoan parasites, in particular during life-cycle and host transitions.
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