

Publication

A Trk/HKT-type K⁺ transporter from *Trypanosoma brucei*

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Author(s) Mäser, P.

Author(s) at UniBasel [Mäser, Pascal](#) ;

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The molecular mechanisms of K⁽⁺⁾ homeostasis are only poorly understood for protozoan parasites. *Trypanosoma brucei* subsp. parasites, the causative agents of human sleeping sickness and nagana, are strictly extracellular and need to actively concentrate K⁽⁺⁾ from their hosts' body fluids. The *T. brucei* genome contains two putative K⁽⁺⁾ channel genes, yet the trypanosomes are insensitive to K⁽⁺⁾ antagonists and K⁽⁺⁾ channel-blocking agents, and they do not spontaneously depolarize in response to high extracellular K⁽⁺⁾ concentrations. However, the trypanosomes are extremely sensitive to K⁽⁺⁾ ionophores such as valinomycin. Surprisingly, *T. brucei* possesses a member of the Trk/HKT superfamily of monovalent cation permeases which so far had only been known from bacteria, archaea, fungi, and plants. The protein was named TbHKT1 and functions as a Na⁽⁺⁾-independent K⁽⁺⁾ transporter when expressed in *Escherichia coli*, *Saccharomyces cerevisiae*, or *Xenopus laevis* oocytes. In trypanosomes, TbHKT1 is expressed in both the mammalian bloodstream stage and the Tsetse fly midgut stage; however, RNA interference (RNAi)-mediated silencing of TbHKT1 expression did not produce a growth phenotype in either stage. The presence of HKT genes in trypanosomatids adds a further piece to the enigmatic phylogeny of the Trk/HKT superfamily of K⁽⁺⁾ transporters. Parsimonious analysis suggests that the transporters were present in the first eukaryotes but subsequently lost in several of the major eukaryotic lineages, in at least four independent events

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