

with longer period of treatment and the continuity of the investigation of new analogues.

doi:10.1016/j.ijid.2010.02.600

82.012

Alanine 163 in loop C of *Leishmania major* aquaglyceroporin LmAQP1 resides near the pore mouth of the channel

Y. Zhou¹, G. Mandal^{1,*}, V.S.R. Atluri¹, E. Beitz², R. Mukhopadhyay¹

¹ Florida International University, Miami, FL, USA

² University of Kiel, Gutenbergstrasse, Germany

Background: The *Leishmania major* aquaglyceroporin, LmAQP1, is responsible for the transport of trivalent metalloids, arsenite and antimonite. We have earlier shown that down regulation of LmAQP1 provides resistance to trivalent antimony compounds whereas upregulation of LmAQP1 in drug resistant parasites can reverse the resistance. We have implicated this transporter to volume regulation and osmotaxis, two important characteristics for successful infection. Recently we have shown that a single change in the loop C of LmAQP1 changes its substrate specificity. Beitz et al (2004) have shown that E125 in loop C of PfAQP is close to the mouth of the pore and is critical for high water permeability of PfAQP (Beitz, 2004). Loop C of PfAQP is twelve residues shorter than LmAQP1. Therefore, E125 of PfAQP lines up with A163 in the primary sequence. To confirm that A163 is near the pore mouth we have created different mutants of LmAQP1 and their transport properties were compared.

Methods: Different mutants of A163 were created using site directed mutagenesis method. Wild type and mutant LmAQP1 were transfected into wild type *L. donovani*. Metalloid sensitivity, transport and osmoregulation ability of the transfected parasites were measured. cRNA of wild type and mutated LmAQP1 were injected into *Xenopus levis* oocytes followed by the determination of metalloid, water and solute transport.

Results: Change of A163 to glutamate, glutamine or aspartate made it impermeable to water, metalloids and other solutes. However, changes to serine and threonine did not change the transport properties of the channel. Water transport through LmAQP1 is mercurial insensitive. Introducing cysteine (s) in loop C in the vicinity of A163 made the water transport through the channel mercurial sensitive.

Conclusion: We report that A163 in loop C is localized near the pore mouth of LmAQP1.

doi:10.1016/j.ijid.2010.02.601

82.013

Chagas disease: Mother to child transmission (MTCT). A single experience in a public hospital from Buenos Aires

M.T. Rodriguez Brieschke^{1,*}, M. Seoane¹, C. Nieto¹, V. Loggia¹, E. Bottaro², M. Giacco¹, J.C. Gonzalez¹, P.G. Scapellato¹

¹ Hospital D F Santojanni, Buenos Aires, Argentina

² Hospital General de Agudos D F Santojanni, Buenos Aires, Argentina

Background: Mother to child transmission of Chagas Diseases (ChD) rates was reported from 1 to >10%. Factors reported to increase risk include younger maternal age, human immunodeficiency virus infection, and parasite strain.

Methods: Retrospective analysis of children assisted in infectious diseases department from 2001 to 2008. Inclusion criteria was: every child born from a mother suffering of ChD with accessing a diagnostic procedures before 24 months of life and never traveled to endemics areas until finish diagnostic process. Diagnosis of ChD among mothers and children over 6 months old were performed by serology (ELISA and HAI). Diagnosis of ChD among children) 6 months was performed with parasitemia investigation.

Results: We studied 307 children born from mother with ChD, 165 were ≤ 24 months of life. Ratio man/woman 87/78. MTCT was 26/152 (17%). Diagnosis was performed by parasitemia in 20/26: the 1st parasitemia performed was positive in 13 cases, 2nd parasitemia was positive in 5 cases and 3rd parasitemia was positive in only a case. In six patients, ChD diagnosis was performed by serology.

Mothers were from: Argentina = 28, Bolivia = 96, Paraguay = 19 and without records = 22. Only 11/26 chagasic children were symptomatic. Most frequent symptoms were: anemia 9/11, hepatosplenomegaly 3/11, hydrops fetalis 1/11, sepsis 1/11, abnormalities in serum transaminases 1/11 and cardiac abnormalities 1/11.

Prematurity was observed in 5/26 with ChD vs 2/111 no ChD. Low birth weight was present in 5/26 (19.2%) with ChD vs 6/111 (5.4%) no ChD.

Three patients born from mother with HIV infection. MTCT were 3/3 (100%) vs 23/145 (15.8%) no HIV. No children were infected with HIV. The study of newborn allowed us performed study of ChD among 97 siblings never studied; in 11 of them ChD was diagnosed. Benzindazol was indicated in all cases of vertically acquired ChD; 9 of the were lost of follow up and 5 had adverse reactions that.

Conclusion: MTCT was higher than previously reported. Even if sample was small; it can be possible that mothers with HIV co-infection have more MTCT than no HIV. We have the opportunity to study all the members of family and bring treatment if it is necessary.

doi:10.1016/j.ijid.2010.02.602