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Expression analysis of Activated protein Kinase C gene (LACK1) in antimony sensitive and resistant *Leishmania tropica* clinical isolates using Real-Time RT-PCR

H. Hajjaran[1], E. Kazemi-Rad[2], M. Mohebali[1], M. A. Oshaghi[3], M. B. Khadem Erfan[4], E. Hajialilo[1], H. Reissi[1], R. Raoofian[5]

[1]*Tehran University of Medical Sciences, Dep. Medical Parasitology and Mycology, Tehran, Iran, Islamic Republic Of*

[2]*Department of Parasitology, Pasteur Institute of Iran, Tehran, Iran, Islamic Republic Of*

[3]*Tehran University of Medical Sciences,, Departments of Medical Entomology and Vector Control , Tehran, Iran, Islamic Republic Of*

[4]*Kurdistan University of Medical Sciences, , Department of Medical parasitology and Mycology,, Sanandaj, Iran, Islamic Republic Of*

[5]*Legal Medicine Research Center, Legal Medicine Organization, Iran, Tehran,, Iran, Islamic Republic Of*

Background: Resistance to pentavalent antimonial drugs has become a serious problem in treatment of cutaneous leishmaniasis (CL) in some endemic areas. Investigations on molecular markers involved in drug resistance are essential for monitoring of the disease. *Leishmania* activated C kinase gene (LACK1) is involved in multiple central processes including signal transduction, RNA processing, and cell cycle control. According to the probable role of the LACK gene in antimony resistance, we used real-time RT-PCR to investigate the expression of this gene in clinical *L. tropica* strains which were resistant or sensitive to meglumine antimoniate (Glucantimee).

Methods: we analyzed the relative expression level of LACK in 18 sensitive and 14 resistant *Leishmania tropica* clinical isolates, were collected from anthroponotic cutaneous leishmaniasis (ACL) patients. After cDNA synthesis, gene expression analysis was performed by quantitative real-time PCR using SYBR® Green. In addition, full length of LACK gene from 6 reference strains was cloned and sequenced then deposited in NCBI database to confirm our strains.

Results: Real time RT-PCR revealed that the average of the RNA expression level of LACK gene in isolates from unresponsive and responsive patients were 0.479 and 4.583 respectively and expression of LACK was significantly down regulated (9.56 fold) in resistant isolates compared to sensitive ones.

Conclusion: The results of the present study suggest the probable role of LACK gene in antimony resistance. Moreover, it can be considered as a potential marker for monitoring of antimony resistance in clinical isolates. However, further studies are required to exploit the biological functions of it in antimony resistance.

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Shape and Force: Key to Transmission of the Malaria Parasite

M. Muthinja[1], K. Quadt[1], C. Moreau [1], J. Kehrer[1], U. Schwarz[2], M. Singer [1], J. Spatz [3], F. Frischknecht [1]

[1]*University of Heidelberg, Infectious Disease, Heidelberg, Germany*

[2]*University of Heidelberg, Institute for Theoretical Physics and Bioquant, Heidelberg, Germany*

[3]*University of Heidelberg, Biophysical Chemistry, Heidelberg, Germany*

The malaria-causing parasite (*Plasmodium*) is transmitted to vertebrate hosts by the infectious bite of a female *Anopheles* mosquito. The infectious forms, known as sporozoites are deposited in the skin and move at high speed (1-2 µm/s) to find and enter blood vessels [1]. Once in the blood they are transported to the liver, where they enter hepatocytes to differentiate into blood cell invading forms. The pre-erythrocytic stages of the malaria parasite are clinically silent but critical for establishing infection in the mammalian host. Sporozoites migrate using an uncommon mode of locomotion called gliding motility [2], which enables them to penetrate host tissues. Sporozoites are polarized crescent shaped cells that typically move in circles on two-dimensional substrates *in vitro*. In order to investigate sporozoite morphology and motility, we generated transgenic parasites expressing altered or fluorescently tagged proteins that are implicated in maintaining curvature or gliding. We use micro-patterned pillar arrays [3] as surrogate 3D tissues and blood capillary mimetics to study the role of curvature in sporozoite motility. To