Energy Metabolism Modulation of Schwann Cells Infected by *Mycobacterium Leprae*

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**Introduction**: Leprosy, or Hansen's disease, is one of the oldest diseases afflicting humans, originated from the infection caused by *Mycobacterium leprae*. The bacillus affects mainly macrophages and Schwann cells, leading to segmental demyelination and axonal loss (WHO, 1998). Our group previous results have demonstrated the modulation of energy metabolism on ST8814 Schwann cells strain infected by *M. leprae*, evidencing the increase of glucose uptake. This increase was not followed by glycolytic increase, or lactate release, generating mitochondrial electrical potential. We analyzed in the present study the pentose cycle pathway modulation and its contribution to lactate and lipids homeostasis in host cell during infection. **Material and Methods**: Enzymes gene expression was analysed by Real Time PCR. Lactate amount was determined on culture supernatant by quinone imine generation at 550 nm using lactate kit liquiform. **Results and Discussion**: In this study, we observed that *M. leprae* infected Schwann cells showed a threefold increase on expression and activity of Glucose-6-phosphate dehydrogenase (G6PD), followed by increase on lipid synthesis and a twofold reduction on lactate production. G6PD is a regulatory enzyme catalyzing the first step of the pentose phosphate pathway that generates NADPH and pentoses. When we use 6-ANAM, G6PD enzyme inhibitor, we successfully recovery lactate liberation by infected Schwann cells, reducing *M. leprae* viability. **Conclusions**: The lactate production modulation by infected Schwann cells could be a new mechanism of neuronal injury, based on the fact that lower liberation of lactate by these cells in vivo will generate an energetic deprivation and subsequent axon's death. Pentose cycle inhibition could present therapeutic effects on leprosy, associated to neuronal lactate nutrition reestablishment and *M. leprae* loss of viability. **Key-words**: Leprosy, metabolism, infection