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P1345 Human Zika virus infection indicates a reduction of IFN-gamma producing CD4+ T-cells and a parallel expasion of Vdelta2 T-cells

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Background: Zika virus (ZIKV) has recently emerged as severe global health issue. Understanding host protective immunity to ZIKV may represent a key issue in vaccine efficacy definition and in identifying possible immune-pathogenetic mechanisms in severe infections. Aim of this study was to analyze the cellular immune response in the acute phase of ZIKV infection, and its role in the protection and/or pathogenesis.

Materials/methods: T cells profile was analyzed in 7 acute ZIKV-infected patients and compared with 5 acute Dengue virus (DEGV)-infected patients and with 10 healthy donors (HD). Phenotype/ Functionality of T-cells were analyzed by flow cytometry, Elispot and proliferation/degranulation assays.

Results: A significant activation of T-cells was observed during both ZIKV and DENV infections. ZIKV infection was characterized by a CD4 T cell differentiation toward effector cells and by a lower frequency of IFN-γ producing CD4 T cells. Moreover, a substantial expansion of CD3+CD4−CD8− T-cell subset expressing Vδ2 TCR was specifically observed in ZIKV patients. Vδ2 T cells presented a terminally differentiated profile, expressed granzyme B and maintained their ability to produce IFN-γ. Results showed that ZIKV infection induced a T-cell activation and differentiation by modulating the cytokine profile, and an expansion of cytotoxic Vδ2 T-cells, suggesting a protective role of these cells.

Conclusions: These findings provide new knowledge on the immune response profile during ZIKV infection pointing out the possible protective role of Vδ2 T-cells in controlling ZIKV replication.