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Genetic regulation of lymphocyte specific proto-oncogene (LCK), differentially selects the right MHC class I variants against a disease

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LCK gene known as lymphocyte-specific proto-oncogene is associated with coordinated expression of Major Histocompatibility Complexes (MHC), class I and II T-lymphocytes, in response to physiological stimuli, mediated through a combined interaction of promoters, suppressors and enhancers. Proximal and distal promoters, functioning independently and developmentally regulated, favor proper *LCK* transcription. The former is thymocyte related while the latter involves all stages of T-cell development. *LCK* is genetically and functionally associated with several diseases including HIV and EBOLA, serious threats to human existence. *LCK* driven immune-selection research for therapeutic intervention derives little scientific interest. The last Ebola outbreak (2014), emerging from Guinea and expanding to Liberia, Sierra Leone, Nigeria and USA, pushed the scientific community to find an urgent therapy against the disease. Our work focused on looking at HLA class I & II subtypes expressed after the Ebola virus (EBOV) attack with focus on the most potent related antigenic epitopes generating favorable immune response to eliminate the disease. *LCK* dominantly observed in genetic interaction analysis was associated with T-cell antigen receptor (TCR)-linked signal transduction pathways hence differentially selecting for CD4 (MHC II) and CD8 (MHC I) immune variants. Data obtained from Gene Omnibus database at NCBI, showed two *LCK* related disease clusters, grouping EBOV with different cancer types and a down regulation of most CD8 immunogenic variants (HLA A, B, C) compared to CD4 variants. This MHC expression pattern, suggests a careful regulatory pattern for *LCK* and related expression of T-cell immune variants whose antigenic specificity is key to resolving the disease burden. We are currently using modern genetic technology to engineer the right *LCK* variants required to drive downstream expression of the required MHC class I immune variants for a specific pathogen, which could be further designed for therapeutic applications against Ebola and related diseases.

Biography

Daniel A Achinko has completed his PhD in Biochemistry from the University of Egerton, Njoro, Kenya in May 2013 at the International Center for Insect Physiology and Ecology (icipe) while working on the transgenesis of malaria vectors as a control technique against malaria. He took special interest in bioinformatics of therapy design leading him to a Post doctorate at the National Institutes of Health, USA. He has published several articles on cancer antigenic epitopes driving proper immune response.

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