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Vaccines-2015 : Identification of highly immunogenic and protective *Leishmania major* antigen that elicits strong T cell recall responses in recovered human patients - Jude E Uzonna - University of Manitoba

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Despite a plethora of publication on immunology of leishmaniasis, there is still no clinically effective vaccine against the disease. We used reverse immunology and proteomics approaches to identify naturally processed L. major peptides presented by MHC II molecules on infected mouse dendritic cells. One of the peptides derived from highly conserved glycosomal phosphoenolpyruvate carboxykinase (PEPCK), induced strong proliferation and IFN-γ production by CD4+ T cells from infected mice. PEPCK is expressed in glycosomes of L. major promastigotes and amastigotes and also induced proliferation, IFN-γ and granzyme production in PBMCs from infected human patients that healed cutaneous leishmaniasis. Vaccination with PEPCK peptide, DNA or recombinant protein induced strong protective immunity against L. major challenge in both the resistant and susceptible mice. Importantly, we generated PEPCK peptide-MHC II tetramer and reliably demonstrate the activation, expansion, effector activity, contraction and stable maintenance of PEPCK-specific CD4+ T cells in L. major-infected mice. Thus, reverse immunology and proteomics-based antigenic peptide identification approach is a potentially rewarding strategy to find new vaccine candidates for infectious pathogens. The primary composed reference to the prominent side effects of cutaneous leishmaniasis surfaced in the Paleotropics inside oriental writings going back to the seventh century BC (purportedly deciphered from sources a few hundred years more seasoned, somewhere in the range of 1500 and 2000 BC). Because of its wide and diligent commonness all through artifact as a baffling malady of differing suggestive results, leishmaniasis has been named with different names extending from "white sickness" to "dark fever". A portion of these names recommend connections to negative social convictions or folklore, which despite everything feed into the social criticism of leishmaniasis today. Individuals from an old sort of the Leishmania parasite, Paleoleishmania, have been recognized in fossilized sand flies going back to the early Cretaceous period, be that as it may, the causative operator for the infection was just found in 1901 as a simultaneous finding by William Boog Leishman and Charles Donovan. They autonomously pictured infinitesimal single-celled parasites (later called Leishman-Donovan bodies) living inside the phones of tainted human organs. The parasitic sort would later be classed as trypanosomatid protozoans under the phylogenetic assignment, Leishmania donovani. A few animal types have since been ordered and assembled under two significant subgenera for example Leishmania Viannia (for the most part situated in the Neotropics) or Leishmania (by and large situated in the Paleotropics, with the significant special case of the L. mexicana subgroup). The subtleties of the advancement of this sort are discussed, yet Leishmania clearly developed from a tribal trypanosome genealogy. The most seasoned heredity is that of the Bodonidae, trailed by Trypanosoma brucei, the last being limited to the African mainland. Trypanosoma cruzi bunches with trypanosomes from bats, South American well evolved creatures, and kangaroos recommend a starting point in the Southern Hemisphere. These clades are just indirectly related. The rest of the clades in this tree are Blastocrithidia, Herpetomonas, and Phytomonas. The four genera Leptomonas, Crithidia, Leishmania, and Endotrypanum structure the terminal branches, proposing a generally late starting point. A few of these genera might be polyphyletic and may require further division.The birthplaces of variety Leishmania itself are unclear. One hypothesis proposes an African starting point, with movement to the Americas. Another proposes movement from the Americas to the Old World through the Bering Strait land connect around 15 million years prior. A third hypothesis proposes a Palearctic origin. Such relocations would involve resulting movement of vector and repository or progressive adjustments en route. A later movement is that of L. infantum from Mediterranean nations to Latin America (known as L. chagasi), since European colonization of the New World, where the parasites got their current New World vectors in their separate ecosystems. This is the reason for the plagues now obvious. One late New World pandemic concerns foxhounds in the USA. In spite of the fact that it was recommended that Leishmania may have developed in the Neotropics. This is most likely valid for species having a place with the subgenera Viannia and Endotrypanum. In any case, there is proof that the

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essential advancement of the subgenera Leishmania and Sauroleishmania is the Old World. While the Mundinia species give off an impression of being progressively widespread in their advancement. One hypothesis is that various heredities became detached geologically during various periods and it is this that offered ascend to this transformative mosaicism. Be that as it may, there is no uncertainty that the Leishmaniinae are a monophyletic gathering. An enormous informational index examination proposes that Leishmania advanced 90 to 100 million years prior in Gondwana. The reptile contaminating species started in mammalian clades. Sauroleishmania species were initially characterized on the premise that they contaminated (reptiles) instead of warm blooded creatures. In view of atomic confirmations, they have been moved to subgenus status inside Leishmania. This subgenus most likely developed from a gathering that initially tainted mammals.

Biography

Jude E Uzonna obtained DVM and PhD in Immunology from the University of Saskatchewan, Canada. After a Postdoctoral fellowship at the University of Pennsylvania, USA, he was recruited to the Department of Immunology, University of Manitoba in 2004. His research program focuses on understanding cellular and molecular mechanisms that regulate the induction, maintenance and loss of protective immunity to protozoan parasites, with a view to exploiting the information gained from these studies for the development of effective vaccines and vaccination strategies against these infections. He is currently an Associate Professor of Immunology and the Leader of Parasite Vaccines Development Research Group.

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