

Interferon-gamma and Human Leukocyte Antigen-G genes promoter unravel for a better understanding of susceptibility to Human African Trypanosomiasis (Koffi: Senior Fellowship)

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Human African Trypanosomiasis (HAT), more commonly called sleeping sickness, is a debilitating and mortal disease that affects sub-Saharan African people living in remote rural areas far away from the formal health system. Thus patients with inadequate or limited access to health care are particularly disadvantaged. Despite the classical dichotomy between the chronic form of HAT caused by *T. b. gambiense* and the acute form attributable to *T. b. rhodesiense*, a diversity of clinical progression has been observed for *T. b. gambiense* ranging from asymptomatic forms to acute forms in Côte d'Ivoire. Recently, field based research demonstrated that untreated human infections by *T. b. gambiense* are not 100% fatal which raise concern about the role of individual who may control HAT infection in the maintenance or resurgence of the disease in foci. HLA-G and IFN-gamma are two key components of the innate immunity against trypanosome. The few studies that exist proved that IFN-gamma and HLA-G expression level and allelic polymorphism differ between clinical features such as long term seropositive without parasitological confirmation, HAT patient and healthy individual but did not highlight the genetic determinism of these differences. With regards to the importance of these gene in the human immunity, we decided to apply for a better understanding targeting the promoter of IFN-gamma and HLA-G genes which are known to have basic implication in the differential expression regulation) of the genes. This study will help to unravel mechanisms underlying differences between HAT individual phenotype groups and ultimately permit to design biomarkers for fine-tuning control strategies.

Projektbeteiligte

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