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Comparison of promoter activities amongst different Merkel cell polyomavirus isolates

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Merkel cell polyomavirus (MCPyV) is common in the human population with seropositivity >50%. Infection occurs in childhood and the virus is part of the normal human skin flora. Its genome is functionally divided into: (i) early region encoding the onco-proteins large T- and small t- antigens, 57 kT and the ALTO protein, (ii) late region encoding the capsid proteins, and (iii) the non-coding control region (NCCR) encompassing the origin of replication and the promoters controlling the expression of the early and late genes. MCPyV is involved in the pathogenesis of Merkel cell carcinoma (MCC) as observed that approximately 80% of all examined MCC specimens are virus-positive. MCPyV-positive MCCs contain integrated viral genome and express truncated large T-antigen, whereas viral DNA is episomal and encodes full-length large T-antigen in other cell types. The NCCRs of most MCPyV isolates are 459-469 bp and differ by few point mutations. However, the NCCR of a strain isolated from healthy skin (strain 16b), feces (strain HB039C) and a Kaposi's sarcoma sample (strain TKS) is ~20 bp longer due to a repeated sequence. The early and late promoters of isolate MCC350 (464 bp) and 16b were cloned in a luciferase reporter plasmid and their promoter activities were compared in HEK293 cells. Our preliminary results demonstrate that the early promoters are stronger than the late promoters and the early 16b is stronger than the early MCC350, while their late promoters have similar activity. The effects of full-length as well as truncated large T-antigen are being examined.

Biography

Abdulsalam Ibrahim has studied Science Laboratory Technology and Biochemistry at Lagos State Polytechnic and Ladoke Akintola University respectively with excellent grades in Nigeria. He moved on to the Arctic University of Norway, to study a Master's degree program in Biomedicine and is presently a member of the Molecular Inflammation Research Group. His research focuses on signal transduction, inflammation and the novel Merkel cell polyomavirus. He co-authorized a publication "The Role of Merkel Cell Polyomavirus and Other Human Polyomaviruses in Emerging Hallmarks of Cancer" (*Viruses 2015*). His current study investigates the reciprocity of inflammation and Merkel cell polyomavirus in tumorigenesis.

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