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PROVIRAL INTEGRATION OF HTLV-1 INTO THE HOST GENOMIC DNA OF ADULT T-CELL LEUKEMIA CELLS FROM BRAZILIAN PATIENTS

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Introduction: HTLV-I virus, the etiologic factor of ATL, integrates its proviral DNA into host cell genome. ATL development is a consequence of monoclonal expansion of a transformed cell that contains an integrated HTLV-I genome. The impact of viral integration on cell phenotype is presumed to be dependent on the affected regions in host DNA. The aim of this study was to investigate whether the HTLV-I integration sites in patients with ATL might confirm diagnosis (monoclonal or oligoclonal) and whether they are completely random or show some specificity for genic or intergenic regions. **Materials and methods:** Viral integration was performed in DNA extracted from 12 PBMC samples and 2 cutaneous biopsies (tumoral lesion) from 14 patients with histopathologic diagnosis of ATL, by inverse PCR and inverse long PCR, as described by Takemoto et al, 1994 and Etoh et al, 1997, respectively, followed by sequencing of specific bands to confirm integration. **Results:** Monoclonal integration of HTLV-I provirus in host genomic DNA was detected in all 12 out of 14 patients studied, two using inverse PCR and ten using inverse long PCR. Proviral integration was detected in PBMC as well as cutaneous biopsies, independent of the clinical subtype of ATL. No chromosome bias was evident among different patients, no coding regions were interrupted. **Conclusion:** inverse long PCR was most efficient to detect proviral integration of HTLV-I and confirm diagnosis in ATL samples from Bahia, Brazil.