ID de Contribution: **28** Type: **Non spécifié**

Viral transactivators shape the nucleus of human B-cells for oncogenesis

Recently we discovered a novel mechanism explaining how B-cell lymphomas might be induced in HIV-infected persons. HIV-positive subjects have an increased risk to develop specific lymphoma subtypes including Burkitt lymphoma (BL). We found that the viral transactivator of transcription (Tat) protein, which is released by infected cells into the blood stream, could remodel the B-cell nucleus bringing together the potential translocation partners, the *MYC* loci at the chromosome 8 and the *IGH* loci at the chromosome 14, thus increasing the probability of the t(8;14) translocation characteristic of BL. Tat induces the mobility of the *MYC* locus in the nucleus via induction of a double strand break in the vicinity of the *MYC* gene and its further repair by NHEJ (Germini et al., 2017, Sall et al., 2019). We shall discuss this and other mechanisms by which HIV-1 Tat and its functional homologue Zta of the Epstein-Barr virus can induce oncogenesis.

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