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Dynamic length changes of telomeres and their nuclear organization in Chronic myeloid leukemia

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Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm characterized by the translocation t(9:22). As in most cancers, short telomeres are one of the features of CML cells, and telomere shortening accentuates as the disease progresses from the chronic phase to the blastic phase. Although most individual telomeres are short, some of them are lengthened, and long individual telomeres occur non-randomly and might be associated with clonal selection. Telomerase is the main mechanism used to maintain telomere lengths, and its activity increases when CML evolves toward advanced stages. ALT might be another mechanism employed by CML cells to sustain the homeostasis of their telomere lengths and this mechanism seems predominant at the early stage of leukemogenesis. Also, telomerase and ALT might jointly act to maintain telomere lengths at the chronic phase, and as CML progresses, telomerase becomes the major mechanism. Finally, CML cells display an altered nuclear organization of their telomeres which is characterized by the presence of high number of telomeric aggregates, feature of genomic instability, differential positioning of telomeres. CML represents a good model to study mechanisms responsible for dynamic changes of individual telomere lengths and the remodeling of telomeric nuclear organization throughout cancer progression.

Biography

Oumar Samassekou has completed his medical degree at the age of 25 years from the University of Bamako (MALI) and Ph.D. studies from the University Sherbrooke (Canada). He is an Assistant Professor of oncology and genetics at the University of Bamako and is doing postdoctoral studies at the Manitoba Institute of Cell Biology. He has published more than 10 papers in peer review journals and presented more than 40 abstracts at scientific meeting.

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