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Hyperdiploid tumors as a driving force in genomic instability

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Abstract

In this lecture we address attention to human breast tumor progression during continuously increasing genomic instability reflected in three ploidy entities. These entities are identified based on DNA-Index (DI) intervals as diploid (D-type-), tetraploid (T-type) and aneuploid tumors (A-type). Following increase in genomic instability and proliferative activity as a reference in a simulation a transition from D-type to T-type tumors was found and a subsequent turn over to A-type tumors. In the breast tumors analyzed this occurred particularly in a limited tumor size interval between 10 to 20 mm. The parameter used to reflect genomic instability we have defined as Stemline-Scatter-Index (SSI). It includes the addition of the parameters of G1-coefficient of variation, percentage of S-phase fraction and the percentage of exceeding G2 cells based on image analysis of Feulgen stained tumors cells. After the invasiveness of the tumor and increasing SSI values T-type tumors seem to lose DNA-content and become included in the A-type DI region. When established as a solid growing tumor an increasing anoxia has been reported in the literature as a driving force for genomic instability. Reaching the tumor size interval of 10 to 20 mm a new wave of T-type tumors was observed. We found these T-type tumors to be recruited from a hyper-diploid (D-type) population within a relatively narrow DI interval. We deduced this to be caused by increasing anoxia due to lack of enough blood vessel in-growth, a theory well established in the literature. In the next and final interval of high SSI values the A-type tumors dominated the tumor population.

Biography

Associate professor Roland B Sennerstam entered research in the seventies at the Institution of Cell Research at the laboratory of professor Torbjorn Caspersson Karolinska Institutet. In his research he has focused on the proliferation of the cell cycle and the development of tumors during increasing genomic instability. In the nineties he developed a model for the cell cycle together with prof Jan-Olov Strömberg and a computer simulating program was created where several parameters could be analyzed over many cell generations. Lately dr Sennerstam has focused on growth and development of breast cancer related to genomic instability.