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13TH INTERNATIONAL CONFERENCE ON THROMBOSIS AND HEMOSTASIS ISSUES IN CANCER, 2026 ABSTRACT N. PO02

MECHANISMS OF THROMBOSIS AND BLEEDING IN CANCER

SYNERGISTIC INTERACTION OF ENDOTHELIAL AND CANCER CELLS IN THE FORMATION AND STRUCTURE OF THE FIBRIN CLOT SHIELDS

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Introduction. Cancer cells (CaCe) express tissue factor (TF) and release TF-bearing extracellular vesicles (CaCe-dEVs), triggering thrombin generation and fibrin network formation within the tumor microenvironment (Tran et al., Thromb Res, 2024). Fibrin clot shields (FCS) act as protective barriers and scaffolds that support CaCe migration. FCS also exert evolutionary pressure, selecting CaCe with increased procoagulant potential. In addition, CaCe-dEVs activate endothelial cells (EC), inducing TF expression and further enhancing local hypercoagulability.

Aim. To investigate the contribution of EC to the formation and structural characteristics of cancer cell–induced FCS and to cancer cell migration within the fibrin network.

Materials and Methods. Viability and proliferation of pancreatic (BXPC3) and breast (MCF7, MDA-MB-231) CaCe, as well as HUVEC cultured alone or in co-culture, were assessed using the alamarBlue® assay. Cells were cultured in RPMI with 10% normal platelet-poor plasma (PPP). Co-culture experiments were performed using CaCe:EC ratios of 2:1, 1:1, and 1:2. Procoagulant activity of cells in PPP was evaluated by calibrated automated thrombography (CAT®, Diagnostica Stago). FCS architecture and CaCe invasion within the fibrin network were analyzed by scanning electron microscopy (SEM), as previously described.

Results. EC exposure to CaCe-dEVs induced a marked procoagulant shift, most pronounced with BXPC3-dEVs compared with MDA-MB-231-dEVs or MCF7-dEVs. In co-cultures, EC significantly amplified thrombin generation compared with CaCe alone. SEM analysis showed cell-dependent differences in FCS: BXPC3 formed dense networks with thin fibers, small pores and long fibers, whereas MCF7 and MDA-MB-231 generated looser structures. EC co-culture induced cell-specific remodeling, with thicker and less complex fibers in BXPC3, increased network complexity in MDA-MB-231, and enhanced intersections and fiber length in MCF7. CaCe migrated within the fibrin network and formed distant colonies, confirming fibrin-guided invasion.

Conclusions. Cancer cells “educate” EC to acquire a potent procoagulant phenotype, amplifying thrombin generation and promoting the formation of robust FCS. EC co-culture does not impair cancer cell migration or colony formation within the fibrin network. These findings identify endothelial cell education as an additional mechanism enhancing FCS formation and contributing to FCS-mediated cancer cell resistance and migration.