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The Role of Proliferation-Associated Platelet-Receptors in Cancer Pathophysiology

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Here, a mechanism is presented that is used by proliferating malignant and normal cells to provide access to proliferation signaling molecules. This is demonstrated in simple experiments which show that HeLa cells and primary fibroblasts in the log phase possess proliferation-associated receptors for platelets. The experiments demonstrate that in vitro these human tumor cells lack self-sufficiency regarding proliferation signaling and that platelet-derived growth-promoting factors are necessary and sufficient for sustaining cell proliferation in cultures.

Previous experiments in our team have demonstrated the presence of such receptors on lectin-activated blood mononuclear cells and on leukemic blasts.

In vivo, these receptors must be part of a mechanism to provide access to factors involved in proliferation signaling at a level prior to the interaction between growth promoting factors and cell surface bound RTKs. This adds a new component to the known proliferation signal cascades. In vitro, where growth-promoting factors are included in the culture medium, platelet receptors appear to have no function. Liquid biopsies have shown that circulating tumor cells regularly have receptors for platelets. The present work shows that such receptors can be blocked pharmacologically. Clinical studies and animal studies elsewhere have shown that treatment with agents that can block this mechanism can delay and prevent tumor progression.