Investigating the Opposing Roles of Oncoprotein gC1qR and Tumor Suppressive cC1qR as Mechanisms for Inhibiting Cancer Pathogenesis

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Current cancer treatment methods remain flawed, oftentimes lacking tumor specificity and causing undesirable side effects. Consequently, elucidating the mechanisms behind tumorigenesis is an attractive, immunotherapeutic approach for transforming targeted cancer treatment. Previous studies have suggested that complement pathway-activating protein C1q and its ubiquitously distributed multiligand-binding cellular receptors, collagenous cC1qR and globular gC1qR, are regulators in cancer cell survival. However, their specific functions remain unclear. This study investigates the role these two critical receptors play in cancer pathogenesis as a means of ending cancerous proliferation. Adenocarcinomas were tested by antigen-capture assays, flow cytometry, immunofluorescence microscopy, and tumor necrosis factor-a assays to determine differential gC1qR and cC1qR secretion and expression in proliferating cancer cells. Results indicate atypical gC1qR overexpression and cC1qR underexpression, suggesting their relevance in cancer's immunosurveillance evasion. Data confirms that cC1qR is diminished and gC1qR upregulated in cancer cell survival and progression, providing a newfound understanding of the mechanisms by which cancer cells maximize proliferation, metastasize, sustain angiogenesis, and evade immune detection and phagocytosis. Transfection of gC1qR and cC1qR antibodies and proteins uncovered the ability to control and counter cancerous proliferation. Taken together, these results unveil the significance of gC1qR and cC1qR in cancer pathogenesis and potential as novel targets for immune treatments, which have universal implications as useful cancer therapeutic modalities.

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