New study links PKC ϵ activation in cancer to aberrant FGFR2 expression

Danilo Ranieri
Monica Nanni
Flavia Persechino
Maria Rosaria Torrisi
Francesca Belleudi

Video Byte

Keywords: Cell Communication and Signaling, Sapienza University of Rome, FGFR2c, PKC ϵ , epithelial-mesenchymal transition, keratinocytes, EMT, fibroblast growth factor receptor 2, aberrant expression, siRNA, isoform, phosphorylation, post-translational modification, splicing, oncogenic profile, oncosuppression, protein kinase C, tyrosine kinase, carcinoma, cancer progression

Posted Date: October 28th, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-99198/v1>

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
[Read Full License](#)

Abstract

When functioning normally, the protein FGFR2 helps regulate cell growth and division, bone growth, and the formation of blood vessels. But alterations in FGFR2 activity have been linked to certain cancers, including breast and prostate cancer, most notably because of a switch between two forms of the protein: from FGFR2b to FGFR2c. To understand the effects of this transition, a recent study examined how the aberrant expression of FGFR2c affects another protein known as PKC ϵ . PKC ϵ is overexpressed in several carcinomas, dramatically increasing the growth rate and mobility of the same cells regulated by FGFR2c. Test-tube experiments revealed that faulty expression of FGFR2c strongly activated PKC ϵ through phosphorylation, a process in which phosphoryl groups are attached to a protein, modifying its function in most cases. Phosphorylation of PKC ϵ coaxed cells to transform from epithelial cells, which line the surfaces of organs, including the skin, into mesenchymal cells, stem cells that can differentiate into a variety of cell types. Understanding how this transformation plays out in the human body could help researchers develop ways of blocking the tumor-promoting effects of FGFR2c.