# Carcinoma-promoting FOXQ1 transcription factor found to suppress melanoma tumors

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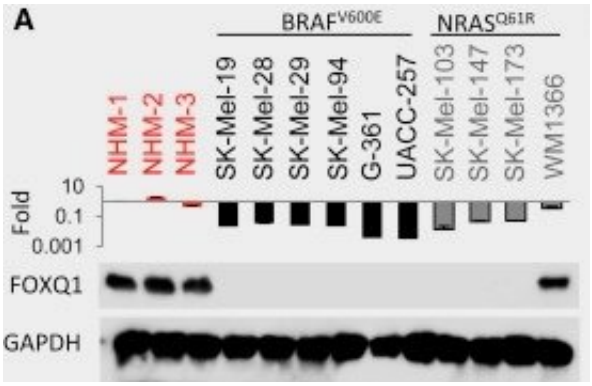


Figure from new research published in the journal Cell Reports, showing that levels of the FOXQ1 gene decrease as melanoma tumors progress. Credit: Roswell Park Cancer Institute

A treatment that works well for one cancer type can possibly make other cancers grow more quickly. That is the striking implication of new research from a team at Roswell Park Cancer Institute, published in the journal *Cell Reports*.

The transcription factor FOXQ1 is a known oncogene that has been previously associated with carcinomas, including many types of breast, colorectal, liver and ovarian cancers. Looking to better understand how this protein might be involved in additional cancer types, a team led by Mikhail Nikiforov, PhD, investigated FOXQ1's role in melanoma, a distinct cancer type that originates from different types of cells than

carcinomas.

What they found—that FOXQ1 suppresses the growth of melanoma cells—was quite unexpected.

"The most surprising finding from this work is that FOXQ1 suppresses exactly the same gene and processes in melanoma cells that it induces in carcinomas," says Dr. Nikiforov, a Professor of Oncology in the Department of Cell Stress Biology at Roswell Park. "I've never come across such a dramatic split in how cancer cells can respond to a single gene. The possible implications for cancer management are quite important. These findings may guide us in the future on how we can avoid use of drugs that, while eliminating one type of cancer, may at the same time induce another."

The team also reports findings about the mechanisms that enable FOXQ1 to inhibit the same processes in melanoma cells that they promote in carcinomas—processes that hinge on a balance between two types of proteins, the b-catenin and TLE family members. When interacting with FOXQ1, these proteins turn it either into a transcriptional activator (in carcinomas) or a repressor (in melanomas). This results in either induction or repression of N-cadherin (the gene CDH2)—a major regulator of tumor invasion and metastasis.

"Our hope is that with further study of these interactions, we will be able to exploit this inverse response and better understand how to better control both melanomas and carcinomas," adds Dr. Nikiforov.

**More information:** Archis Bagati et al. Melanoma Suppressor Functions of the Carcinoma Oncogene FOXQ1, *Cell Reports* (2017). DOI: 10.1016/j.celrep.2017.08.057

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