Study identifies potential targets for treating triple negative breast cancer

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No specific treatments are currently available for triple negative breast cancer (TNBC), a type of tumor that lacks the receptors targeted by many breast cancer therapies. Although many TNBC tumors lack two tumor suppressors, RB1 and p53, the specific downstream pathways that can be targeted as potential treatments for these tumors have not been identified.

In this issue of the *JCI*, a team led by Eldad Zacksenhaus at Toronto General Research Institute discovered that the growth of TNBC-like breast tumors is supported by enhanced mitochondrial function. Mice carrying tumors that were deficient in both RB1 and p53 displayed upregulation of a pathway that controls the synthesis of mitochondrial proteins.

They then identified an FDA-approved drug, tigecycline, that blocks this upregulation and reduces the growth of TNBC-like tumors in mice. This work suggests that inhibiting mitochondrial protein translation could potentially be a successful treatment for TNBC.

More information: Robert A. Jones et al, RB1 deficiency in triple-negative breast cancer induces mitochondrial protein translation, *Journal of Clinical Investigation* (2016). DOI: 10.1172/JCI81568

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