Interaction of PAX1 (P6) and Mitochondrial Metabolism With Immunosuppressive Therapies

Authors: Kenneth Mills Jeffrey Clark Kelly Davis Casey Juarez Stephanie Jackson

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Humboldt State University

School of Mathematics

This paper presents how the pathways of tumorigenesis are activated by mitochondrial metabolism activated by PAX1 acting through nitric oxide channels induced by cancer initiation, metastasis, tumor burden, and tumor treatment, and process tumorigenesis through immunosuppressive cytokinesis. A specific molecular function of PAX1 is to regulate the continuous activation of the Aurora-A and BRCA2 promoters and to convert their activatory properties to suppress-induced inhibition by oxidative stress. However, the modulation of the protein stability hinges on the expression of selected proteins.

AEGR1, MeX6, and melanopyrimidine-2 are the three proteins whose inhibition may protect against ion-type activation and development of tumorigenesis. In this study, post-expression analysis of mice bred to grow tumors activated by MAPK1 (or its derivative) and MeX6 showed in vivo survival significantly more than did mice with activated MAPK1 or MeX6. This study showed that the stable PAX1 (P6) expression in pre- and post-expression both activates Aurora-A and BRCA2 promoters, and suppresses the activation of PI3K and phospholipase-A2. PAX1 is the promoter of PI3K1 and 2, the driver of endothelial destruction and cell death, and is also sufficient for binding the MCK4a, and hence efectively validates eVersantâ€™s mechanistic model of oxidative stress induced runaway proliferation and tumorigenesis.

A Large Black Bear Standing Next To A Tree