

A tiny RNA with a big role in melanoma

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will emerge as a better therapeutic option to treat melanoma patients," said senior author Narendra Wajapeyee, assistant professor of pathology at Yale School of Medicine and a member of Yale Cancer Center.

Provided by Yale University

Credit:

A Yale-led study has identified a key mechanism in the regulation of gene expression that promotes the proliferation of melanoma cells. The finding opens a possible avenue for development of treatments that target this mechanism. The study appears online Feb. 18 in the journal eLife.

Melanoma is the deadliest form of skin cancer. BRAF and NRAS genetic mutations are known to occur in a very large proportion of melanomas. But although scientists have identified the oncogenic mutations that trigger melanoma cells and their proliferation, the mechanism that causes normal cells to transform into cancerous melanoma cells and cause their spread has not been well defined.

The Yale team identified a microRNA called miR-146a that accelerates in activity in the presence of these oncogenes by activating a cancer promoting signaling pathway called Notch.

The authors suggest that Notch and another signaling pathway called MAP kinase, which is activated by these same oncogenes, represent a valuable drug development target. "Our study has identified a new genetic vulnerability in melanoma, and it is likely that simultaneous inhibition of MAP Kinase pathway and NOTCH signaling pathway



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