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1: [Cell](#). 2006 Mar 24;124(6):1169-81. [Related Articles, Links](#)

**Cell Press**

**A genetic screen implicates miRNA-372 and miRNA-373 as oncogenes in testicular germ cell tumors.**

[Voorhoeve PM](#), [le Sage C](#), [Schrier M](#), [Gillis AJ](#), [Stoop H](#), [Nagel R](#), [Liu YP](#), [van Duijse J](#), [Drost J](#), [Griekspoor A](#), [Zlotorynski E](#), [Yabuta N](#), [De Vita G](#), [Nojima H](#), [Looijenga LH](#), [Agami R](#).

Division of Tumour Biology, The Netherlands Cancer Institute, Amsterdam, The Netherlands.

Endogenous small RNAs (miRNAs) regulate gene expression by mechanisms conserved across metazoans. While the number of verified human miRNAs is still expanding, only few have been functionally annotated. To perform genetic screens for novel functions of miRNAs, we developed a library of vectors expressing the majority of cloned human miRNAs and created corresponding DNA barcode arrays. In a screen for miRNAs that cooperate with oncogenes in cellular transformation, we identified miR-372 and miR-373, each permitting proliferation and tumorigenesis of primary human cells that harbor both oncogenic RAS and active wild-type p53. These miRNAs neutralize p53-mediated CDK inhibition, possibly through direct inhibition of the expression of the tumor-suppressor LATS2. We provide evidence that these miRNAs are potential novel oncogenes participating in the development of human testicular germ cell tumors by numbing the p53 pathway, thus allowing tumorigenic growth in the presence of wild-type p53.

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