

New data on the regulation of the genetic activity that protects against lung cancer

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A research project led by the University of Granada, which includes researchers from Harvard and Yale, has provided new data for a better understanding of the alterations produced during the development of lung cancer, the tumour with the highest yearly death rate in Spain. This research clears the path for the development of new antitumoral therapeutic strategies based on microRNAs activity

Scientists at the University of Granada, in collaboration with the universities of Harvard and Yale (United States) have provided new data for a better understanding of the alterations produced during the [development](#) of lung cancer, the tumour with the highest yearly death rate in Spain.

This research has found that certain small RNA molecules called microRNAs can deactivate the function of the SMARCA4 gene, which protects healthy cells from becoming tumour cells.

These findings, which were developed in pre-clinical models, constitute the foundation for the development of future applications for the diagnosis and prognostication of [lung cancer](#).

"We had previously discovered that [lung](#) tumours in patients lost the activity of the SMARCA4 gene, which carries out tasks that protect normal cells from turning into [tumour cells](#). This new research proves that this loss in the tumour-suppression activity of SMARCA4 could be attributed to the [activity](#) of certain microRNAs", says prof. Pedro P.

Medina, the principal investigator in this project, and a researcher at the Molecular Biochemistry and Biology I Department at the University of Granada.

"This result has opened up a new research line in our lab, by means of which we aim to explore new therapeutic pathways based on the regulation conducted by microRNAs", he added.

More information: "Expression inactivation of SMARCA4 by microRNAs in lung tumors" *Hum. Mol. Genet.* (2014) [DOI: 10.1093/hmg/ddu55](https://doi.org/10.1093/hmg/ddu55)

Provided by University of Granada

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