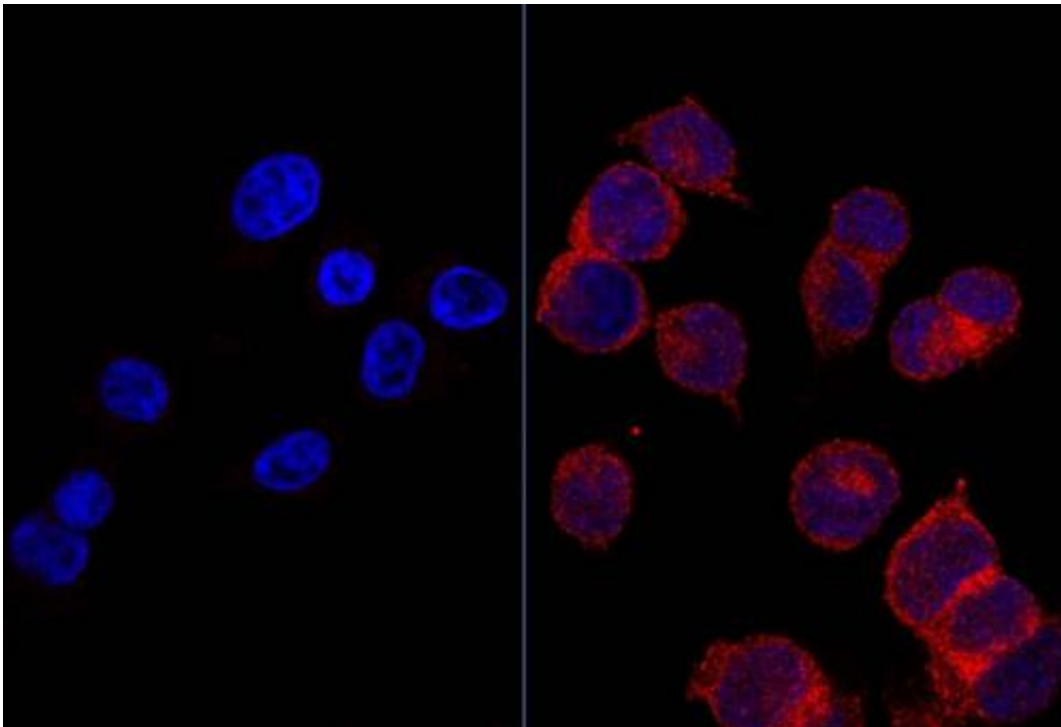


Discovery of a mechanism that makes tumor cells sugar addicted

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This shows, left, healthy cells with glucose receptors in red. Right, tumor cells with glucose in red receptors. Credit: PEBC-IDIBELL

For almost a hundred years ago is known that cancer cells feel a special appetite for a type of sugar called glucose. The tumor uses this molecule is like the gasoline which depends a sports car to burn faster and grows and multiplies rapidly. It is a little cash process from the energy point of view but allows a superaccelerated cancer cell division. It is what is

known as the Warburg effect, which was described in 1927.

Until now little was known about how [healthy cells](#) that have a balanced energy consumption depend on this "fast food" calorie in the tumor cell. Today, an article published in *Nature Communications* led by Manel Esteller, Director of Epigenetics and Cancer Biology, Bellvitge Biomedical Research Institute (IDIBELL), ICREA researcher and Professor of Genetics at the University of Barcelona, provides an important clue to understand this process. Research shows that in one in four human tumors, there is an excess of glucose receptors in the external face of the cell membrane and this protein acts as a magnet attracting all the glucose from the bloodstream.

"We were looking for genes that did not work in tumor cells and we found an altered one, but unaware what his function. we discovered that it was responsible for removing excess of glucose receptors" explained Esteller . "So what happens is that the gene that should degrade glucose receptor is inactivated in sound condition and quit, this tumor has an overactivation of this receptor that captures all the glucose molecules around it and used to obtain quick energy to proliferate. It is a cancer that has become addicted to this caloric molecule. "

"The interesting part is to study whether future treatments that fight the tumor and also quit the energy source of the [tumor cells](#), the tumor dies because it cannot easily be adapted to use other substrates for energy to survive," concludes Esteller.

More information: Lopez-Serra P, Marcilla M, Villanueva A, Ramos-Fernandez A, Palau A, Leal L, Wahi JE, Setien-Baranda F, Szczesna K, Moutinho C, Martinez-Cardus A, Heyn H, Sandoval J, Puertas S, Vidal A, Sanjuan X, Martinez-Balibrea E, Viñals F, Perales JC, Bramsem JB, Ørntoft TF, Andersen CL, Tabernero J, McDermott U, Boxer MB, Vander Heiden MG, Albar JP, Esteller M. A DERL3 Associated Defect

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