Cancer cells deactivate their 'Velcro' to go on the attack

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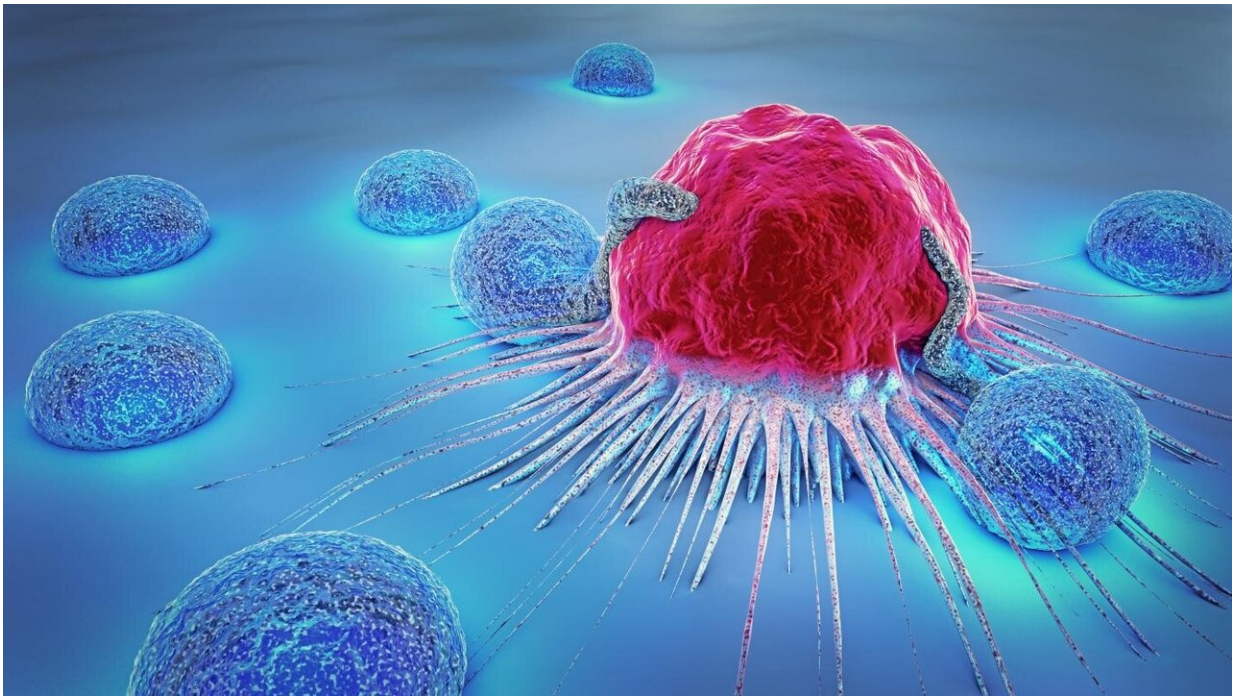

To form metastases, cancer cells must be able to migrate. But cancer cells are connected to each other by 'Velcro'University of Louvain (UCLouvain) researchers have discovered that certain cancer cells manage to suppress this 'Velcro' effect so that they can migrate more easily. It's a mechanism called endocytosisThe next step? Understanding the role of this mechanism (endocytosis) in the formation of metastases, which could ultimately help fight them! Credit: University of Louvain

Cancer cells remain clumped together via a sort of 'Velcro' which allows them to adhere to each other wherever they appear. In order for cancer cells to leave a tumour and spread throughout the body during metastatic processes, cancer cells must reduce their adhesion and increase their ability to migrate. They can do this by changing the amount and type of proteins on their surface.

University of Louvain (UCLouvain) research conducted by Henri-François Renard and François Tyckaert and directed by Professor Pierre Morsomme targeted these surface proteins, particularly one called CD166, a kind of microscopic 'Velcro' which allows the cells to stick together.

UCLouvain researchers observed that certain cancer cells are, in fact, capable of decreasing the abundance of this CD166 on their surface. More specifically, these cells have found a way to reduce the adhesion of their surface by redirecting it into small internal vesicles: this is a mechanism called endocytosis, which UCLouvain's researchers highlighted in their study published in the prestigious scientific journal *Nature Communications*.

If there is less adhesion, owing to less CD166 on the surface of cancer cells, they stick together less and therefore migrate more easily.

The next step? This fundamental mechanism still has many secrets to reveal. But UCLouvain researchers hypothesise that it could contribute to the formation of metastases which allow cancer to spread. And who knows, in the future this will perhaps make it possible to develop new solutions that block metastases and thus slow down the development of certain cancers.

More information: Henri-François Renard et al, Endophilin-A3 and

Galectin-8 control the clathrin-independent endocytosis of CD166, *Nature Communications* (2020). DOI: 10.1038/s41467-020-15303-y