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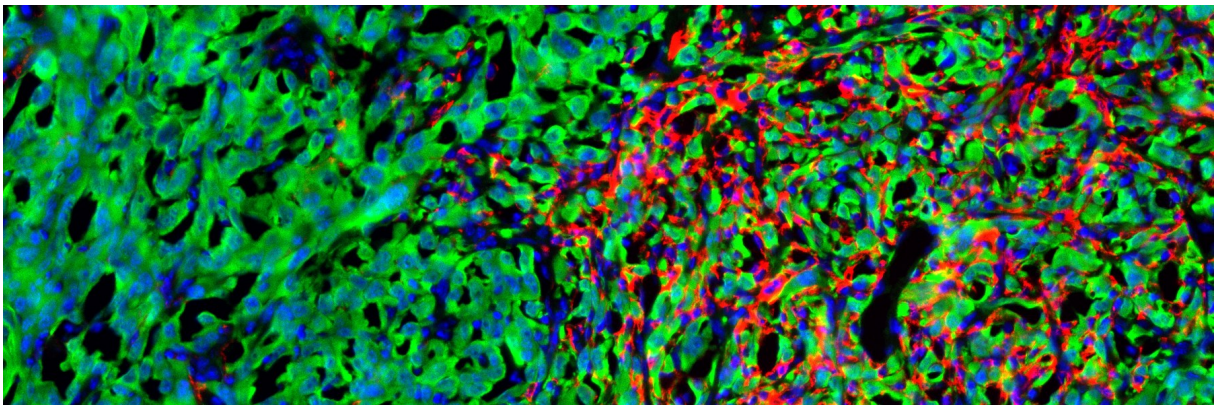

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Bad neighbors: how immune cells help cancer cells get ready to spread

Published in Nature Cancer: Researchers at the Université libre de Bruxelles (ULB) have discovered that immune cells build distinct "neighborhoods" around cancer cells. In one of these neighborhoods, immune cells communicate with cancer cells to promote their ability to spread to distant organs and mediate metastasis formation. This research could eventually help develop new therapies to target metastasis.

Most cancer deaths are not caused by the original tumor, but by metastases, which correspond to the cancer cells that leave the original tumor and disseminate to other organs. To do so, cancer cells must loosen their attachment with the other cancer cells and become more motile, to be able to move away from the tumors, a process called epithelial-to-mesenchymal transition, or EMT. EMT is a progressive phenomenon and cancer cells go through an intermediate "hybrid" stage, which is the best at forming metastases.

The study, published in the prestigious journal Nature Cancer, was led by Prof. Cédric Blanpain, MD/PhD, investigator at the WEL Research Institute, director of the Laboratory of Stem Cells and Cancer and professor at the Université libre de Bruxelles.

The cancer is made of cancer cells and different normal cells, such as immune cells and blood vessels, which usually help cancer cells to grow. The researchers wanted to know which are these normal neighboring cells that surround cancer cells during EMT, and how these normal cells communicate with cancer cells to give rise to metastasis.

Each stage has its own neighborhood

Like a city divided into districts, each with its own residents, the tumor is organized into distinct neighborhoods. Hybrid EMT cancer cells are surrounded by a subset of white blood cells called neutrophils whereas in the most advanced stage of EMT, cancer cells are surrounded by another type of white blood cells called macrophages.
To assess the role of these different white blood cells to regulate EMT and metastasis, the researchers experimentally removed these different immune cells. When the researchers removed neutrophils in mice, hybrid EMT cancer cells decreased, which led to fewer lung metastases. In contrast, when they removed the macrophages, this led to an increase of hybrid EMT cancer cells, but had no effect on lung metastases.

"It was striking to see that neutrophils and macrophages occupy different territories around cancer cells. When we removed neutrophils in mice, the hybrid cancer cells were strongly reduced but more importantly this led to a strong decrease in metastasis, suggesting that the dialogue between neutrophils and cancer cells controls their metastatic potential," explains Dr. Anqi Dong, co-first author of the study.

Two messages, two neighborhoods

The researchers then looked for the molecules that these two types of white blood cells use to "talk" to cancer cells. They found that neutrophils release a molecule called oncostatin M, which pushes cancer cells into the hybrid state of EMT. Indeed, when the researchers gave mice a drug that blocks oncostatin M, the number of hybrid cancer cells went down. Macrophages, in contrast, release another molecule, TGFβ1, which starts EMT and drives cancer cells towards its most advanced stage.

"By dissecting for the first time the molecular dialogue between the immune cells and the tumor cells during EMT, we uncovered oncostatin M and TGFβ1 as two of the key molecules that regulate EMT and metastatic dissemination," explains Dr. Sebastiaan Vanuytven, co-first author of the study.

Also present in human cancers

To find out whether their findings also apply to human cancers, the researchers looked for similar neighborhoods and the same molecular dialogue around hybrid cancer cells in human tumors. Strikingly, they found neutrophils producing oncostatin M next to hybrid cancer cells in human breast, pancreatic, lung and bladder cancers. This suggests that the same mechanism is present in at least some human cancers.

Why it matters

Hybrid EMT cancer cells are the most likely to spread and to resist anti-cancer therapy. Knowing which signals keep cancer cells in this dangerous state opens new avenues to prevent metastasis and sensitize tumor cells to therapy.

"Our study shows that EMT in cancer cells and the ability to give rise to metastasis are not only decided by the cancer cells, but are also influenced by the molecular dialogue between white blood cells and the cancer cells. It will be important to assess whether targeting the signals we have identified in this study, such as oncostatin M, could help to prevent metastasis and improve response to anti-cancer therapy," says Prof. Cédric Blanpain, who led the study.

Collaborations and funding

This study was carried out by the team of Prof. Blanpain (ULB, Belgium) in collaboration with the team of Sandrine Rorive and Isabelle Salmon at DIAPath (Center for Microscopy and Molecular Imaging, ULB) and CurePath (CHIREC, CHU Tivoli and ULB, Belgium).

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Journalists must credit Nature Cancer as the source of the article covered.

Reference

Myeloid-derived TGFβ1 and oncostatin M establish two distinct stromal niches controlling late and hybrid EMT states
Anqi Dong#, Sebastiaan Vanuytven#, Andrea Pérez Gonzalez, Kevin Bevant, Lieselot Van de Putte, Zahra Zahedi, Justine Allard, Christine Dubois, Isabelle Salmon, Sandrine Rorive and Cédric Blanpain.
# These authors contributed equally.
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Contact

Prof. Cédric Blanpain, MD, PhD
Professor of Stem Cell and Developmental Biology
Investigator at WEL Research Institute, WELBIO Department
Director of the Laboratory of Stem Cells and Cancer
Université libre de Bruxelles (ULB)
808, route de Lennik, Bldg GE, G2 4.205, 1070 Brussels, Belgium
Office: +32 2 555 41 75
Email: Cedric.Blanpain@ulb.be
Assistant: Marylène Poelaert – Marylene.Poelaert@ulb.be
Lab website: http://blanpainlab.ulb.ac.be/index.htm