Press Release
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PRESS RELEASE EMBARGOED UNTIL 14 JULY

The prestigious journal Nature Cell Biology has published

Keys to reprogramming cells and suppressing tumour invasion

Experts of the IMIM-Hospital del Mar Cancer Research Programme have collaborated on a study that identifies new keys in breast cancer tumour invasion.

Researchers and doctors of the IMIM-Hospital del Mar Cancer Research Programme, with the participation of the Jiménez Díaz Foundation of Madrid, have been the Spanish contribution and played a fundamental role in this study published by the prestigious journal Nature Cell Biology. Led by Jonas Fuxe from Stockholm, the study explains, for the first time, **new keys in tumour invasion in cancer, specifically breast cancer**. **This discovery opens new doors to a possible reprogramming of this tumour invasion with the aim of reversing the process, thanks to the elimination of the expression of genes responsible for the chain of actions that are necessary for this invasion.**

This study is a prime example of the translational research that is conducted at Hospital del Mar and its IMIM Research Centre. Together, those in the Cancer Research Programme work to find all the scientists' possible clinical applications. In this study, the results from in vitro samples, animal models, as well as tumour samples from breast cancer patients have been studied and validated.

A localized breast tumour can metastasise thanks to a set of cellular mechanisms that allow the invasion of nearby tissues (called epithelial-mesenchymal transition). As a result of these mechanisms, the cell loses some of its own characteristics and acquires new ones. This change in the cells and tissues makes it possible for the new transformed cell to acquire migratory properties (due to a loss in adhesion) and thus invasive properties. This change is what favours the appearance of metastasis.

The trigger that causes a localized breast tumour to invade the nearby tissues is a TGFβ factor, involving the transformation of the tissues when they become malignant. This study identifies proteins that are dependent on TGFβ and their interaction with an already-known promoter of these cellular changes, as well as the formation of a complex that activates or inhibits the transformation of the tissues. This complex acts as an switch for the formation of two proteins (CAR and E-cadherin) which are responsible for maintaining the union or adhesion between the epithelial tissue cells and thus prevent the invasion of the nearby, healthy tissues. Therefore, the key is in deactivating this complex in order to protect the manufacturing of the two proteins that will prevent the cells of a tissue in tumour transformation from disconnecting from their neighbouring cells, increasing their mobility and thus the ability to cause metastasis.

For more information: **www.hospitaldelmar.cat** i **www.imim.es**
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