

Researchers uncover novel role of BRCA1 in regulating the survival of skin stem cells

January 3 2013

Our DNA, which stores our genetic information, is constantly exposed to damage. If not properly repaired, DNA damage can lead to cell death. This, in turn, can lead to tissue exhaustion and ageing, or induce mutations resulting in uncontrolled cell proliferation and cancer. Brca1 is a key gene that mediates DNA repair, and mutations in Brca1 lead to familial and sporadic breast and ovarian cancer in humans.

In this study published in [Genes and Development](#), researchers reveal the critical role of Brca1 for the maintenance of hair follicle stem cells.

Researchers showed that upon deletion of the breast cancer associated gene Brca1 in the epidermis, hair follicle cells show high levels of DNA damage and cell death. This cell damage and death induces hyperproliferation and finally exhaustion of hair follicle stem cells, resulting in hair follicle degeneration. In contrast, the other types of stem cells located in the epidermis, which form the skin barrier and the sebaceous glands, are maintained and continue to function normally despite the absence of BRCA1. This variance displays the different requirement for BRCA1 in the distinct types of adult stem cells. "We were very surprised to see that distinct types of cells residing within the same tissue may exhibit such profoundly different responses to the deletion of the same, crucial gene for [DNA repair gene](#)" comments Peggy Sotiropoulou, the first author of this study.

This work is very important to understanding the [DNA repair mechanisms](#) in different types of [adult stem cells](#) and at different stages

of their activation. If other stem cells of the body also require BRCA1 for their survival, this result may potentially explain why [Brca1 mutations](#) in women lead preferentially to the development of only breast and ovarian cancers.

More information: Sotiropoulou, P. et al., BRCA1 deficiency in skin epidermis leads to selective loss of hair follicle stem cells and their progeny. *Genes and Development*, January 1, 2013.
www.ncbi.nlm.nih.gov/pubmed/23271346

Provided by Université libre de Bruxelles

Citation: Researchers uncover novel role of BRCA1 in regulating the survival of skin stem cells (2013, January 3) retrieved 9 April 2024 from <https://medicalxpress.com/news/2013-01-uncover-role-brca1-survival-skin.html>

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