

Researchers uncover insights into how moles change into melanoma

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Robert Judson-Torres, PhD. Credit: Huntsman Cancer Institute

Moles and melanomas are both skin tumors that come from the same cell called melanocytes. The difference is that moles are usually harmless, while melanomas are cancerous and often deadly without

treatment. In a study published today in *eLife Magazine*, Robert Judson-Torres, Ph.D., Huntsman Cancer Institute (HCI) researcher and University of Utah (U of U) assistant professor of dermatology and oncological sciences, explains how common moles and melanomas form and why moles can change into melanoma.

Melanocytes are cells that give color to the skin to protect it from the sun's rays. Specific changes to the DNA sequence of melanocytes, called BRAF gene mutations, are found in over 75% of moles. The same change is also found in 50% of melanomas and is common in cancers like colon and lung. It was thought that when melanocytes only have the BRAFV600E mutation the cell stops dividing, resulting in a [mole](#). When melanocytes have other mutations with BRAFV600E, they divide uncontrollably, turning into melanoma. This model is called "oncogene-induced senescence."

"A number of studies have challenged this model in recent years," says Judson-Torres. "These studies have provided excellent data to suggest that the oncogene-induced senescence model does not explain mole formation but what they have all lacked is an alternative explanation—which has remained elusive."

With help from collaborators across HCI and the University of California San Francisco, the [study](#) team took moles and melanomas donated by patients and used transcriptomic profiling and digital holographic cytometry. Transcriptomic profiling lets researchers determine molecular differences between moles and melanomas. Digital holographic cytometry helps researchers track changes in human cells.



Doug Grossman, MD, PhD | Robert Judson-Torres, PhD | David Lum, PhD | Matthew VanBrocklin, PhD | Sheri Holmen, PhD. Credit: Huntsman Cancer Institute

"We discovered a new molecular mechanism that explains how moles form, how melanomas form, and why moles sometimes become melanomas," says Judson-Torres.

The study shows [melanocytes](#) that turn into melanoma do not need to have additional mutations but are actually affected by environmental signaling, when cells receive signals from the environment in the skin around them that give them direction. Melanocytes express genes in different environments, telling them to either divide uncontrollably or stop dividing altogether.

"Origins of melanoma being dependent on environmental signals gives a new outlook in prevention and treatment," says Judson-Torres. "It also plays a role in trying to combat melanoma by preventing and targeting genetic mutations. We might also be able to combat melanoma by changing the environment."

These findings create a foundation for researching potential melanoma biomarkers, allowing doctors to detect cancerous changes in the blood at earlier stages. The researchers are also interested in using these data to better understand potential topical agents to reduce the risk of melanoma, delay development, or stop recurrence, and to detect [melanoma](#) early.

More information: Andrew S. McNeal et al, BRAFV600E induces reversible mitotic arrest in human melanocytes via microRNA-mediated suppression of AURKB, *eLife* (2021). DOI: [10.7554/eLife.70385](https://doi.org/10.7554/eLife.70385)

Provided by Huntsman Cancer Institute

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