New Laboratory Tool Opens Door to Better Treatments for Rare and Deadly Melanomas

TURKU, FINLAND, June 29, 2026 /EINPresswire.com/ -- Researchers have created a powerful new cellular model to study rare tumours that are resistant to immunotherapies. The tool could change how therapies are developed for aggressive melanomas that currently have almost no effective treatment options.

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> *Dr Carlos R. Figueiredo*

A research team at the University of Turku in Finland have developed a reliable laboratory model to study BAP1-deficient melanomas, which are a rare type of melanoma that evade the immune system once they have metastasized and are universally resistant to current state-of-the-art immunotherapies.

Metastatic BAP1-deficient melanoma is the most common intraocular malignancy in adults, but it remains rare and extremely difficult to treat once it spreads. When the disease reaches the liver, as it does in roughly half of patients, median survival is measured in months. Unlike common melanomas, BAP1-deficient melanomas do not respond to the immune checkpoint therapies that have transformed cancer care over the past decade.

The key driver behind the tumour's immune evasion is the loss of a gene called BAP1 (BRCA1-associated protein 1). When BAP1 is lost, tumours become more aggressive, grow faster, and suppress the immune cells that would otherwise attack them. BAP1 loss is the most important molecular event in uveal melanoma progression, and it also plays a role in mesothelioma, renal cell carcinoma, and other cancers.

However, scientists have been unable to study the consequences of BAP1 loss properly in the laboratory, because no suitable immune-competent model existed.

Gene Editing Solved the Missing Puzzle Piece

To address the issue, the research team used CRISPR-Cas9 gene editing to delete BAP1 from normal melanocyte cells, creating a new pre-clinical tumour model that behaves like human BAP1-deficient melanoma in an animal with a fully functioning immune system.

“BAP1 loss is associated with poor prognosis and resistance to immunotherapy in melanoma. Until now, there has been no preclinical model that faithfully reproduces the tumour–immune interactions seen in patients. Our model fills this gap, for the first time, by recapitulating the human tumour immune microenvironment in vivo. This provides a powerful platform to study how BAP1 loss drives immune evasion and to test novel immunotherapy combinations that may overcome treatment resistance,” explains lead researcher of the study, Dr Mona Wang Meng from the University of Turku.

The absence of a proper laboratory model has been one of the biggest bottlenecks slowing down drug development for BAP1-deficient melanoma and related rare tumours. Previous models either lacked a functional immune system, making them useless for immunotherapy studies, or carry too many confounding mutations that obscure the specific role of BAP1.

“The implications go well beyond melanoma. BAP1 loss is a shared vulnerability across several hard-to-treat cancers. This platform allows us, and research groups worldwide, to rationally design and test new immunotherapy combinations — something that simply was not possible before,” says Dr Carlos R. Figueiredo, the Principal Investigator of the study.

The study was published in the journal Communications Biology. It is part of the broader research programme of MIORG within the InFLAMES Research Flagship at the University of Turku, which focuses on harnessing the immune system to fight cancer.

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