**Cancer cells reprogram to metastatic state through the acquisition of platelet mitochondria**

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**Background**：

Tumor metastasis has long been considered an important factor of death. In osteosarcoma, some patients still develop lung metastasis after chemotherapy, which presents a challenge to cancer treatment. Since then, more and more studies have been trying to figure out the mechanisms of tumor metastasis and how the process is regulated in order to search for a new way to prevent cancer metastasis.

Platelets have been reported to play a key role in tumorigenesis, as platelets can attach to the surface of cancer cells to promote metastasis and immune evasion. However, mitochondria are known to be transferable and may become pathological under certain circumstances. Research has indicated that cancer cells can acquire mitochondria from adjacent normal cells to evade immune response in human body. This kind of mitochondrial transfer can also occur between platelets and mesenchymal stem cells (MSCs) to enhance MSCs ability towards tissue repair and angiogenesis.

Therefore, with the support of the known information, the author tried to determine if mitochondria can transfer from platelets to cancer cells. Moreover, they tried to investigate the mechanism and involved metabolites of the transfer progress.

**Objective**：

To investigate the mechanism of mitochondrial transport from platelets to cancer cells and the metabolites remodeling related to this process.

**Results**：

The author first confirmed that the mitochondria can be transferred from platelets to K7 cells. Then, according to previous studies, Mfn2, a GTPase, has been reported as a necessary protein involved in mitochondrial transport. They found that the mitochondrial transfer is also related to Mfn2 and its relative PINK1/parkin pathway. Moreover, they also found that the taken-up mitochondria can remodel the metabolism of cancer cells, allowing them to transform into a metastatic state.

**Conclusion**：

Osteosarcoma cells turn into metastatic states after the acquisition of platelet mitochondria. The process weakens proliferation ability of cancer cells but enhances their metastatic capability by changing their metabolic way.