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DSTYK INHIBITION ALTERS CELLULAR CYTOSKELETON AND REDUCES METASTASIS IN VIVO IN NON-SMALL CELL LUNG CANCER

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Lung cancer is the leading cause of cancer death among both men and women. The 5 year relative survival rate is 15%. Personalized medicine and new targeted therapies have demonstrated long-term survival in patients. We identified **DSTYK**, a dual serine/threonine and tyrosine non-receptor protein kinase as a potential **novel target** for lung cancer. This protein is altered in **6%** of non-small cell lung cancer (NSCLC) patients, and we showed that it could be a new targetable protein. Our **objective** is to determine the molecular mechanisms by which DSTYK inhibition affects to tumoral cells and how can be used as a therapeutic target. We used high DSTYK expression NSCLC human and mouse cell lines. DSTYK inhibition was achieved through CRISPR-Cas9 technology. In these models, we clearly show that DSTYK inhibition alters cell **cytoskeleton** and **integrin** expression, decreasing cell **adhesion**, **migration** and **invasion** of tumoral cells. Moreover, we have verified *in vivo* mouse lung cancer models that DSTYK inhibition **decreases** tumor cell **metastasis.** Our **results** suggest the molecular mechanisms through which DSTYK is involved in promoting lung cancer progression and gives more light about the way through which **DSTYK inhibition** may act as a **novel therapeutic strategy** for lung cancer patients management.