Repression of microRNA-768-3p by MEK/ERK signalling contributes to enhanced mRNA translation in human melanoma

Peter Tran

Institute of Neurological Sciences and Psychiatry, Hacettepe University, Ankara 06100, Turkey.

01-01-2013

1 Abstract

What are CD133 proteins? CD133 is a protein involved in the production of HER2, a human HER2 cell type to which tamoxifen interferes by inhibiting production of it. Tamoxifen inhibits HER2 production in both macrophages, the type that appears to be involved in breast cancer, and HER2 cells that are involved in several forms of breast cancer. Targeting CD133 thereby blocked some of HER2 activity.

Immune system members include hepatocytes (liver cells), hepatocellular epithelial cells (the bodys outermost blood vessels), and other types of immune cells. Cherng has studied HER2 proteins as they interact with other proteins by way of the transcription factor A and TLR1. Her results have led to the identification of CD133 as an antibody target, and as a target that can directly inhibit CD133 production in HER2-positive breast cancer tumors. An important target for drug development is an ABH agent, modulators of ABH inhibitors whose projectors are limited to those known to be relevant to HER2 expression. In these inhibiting ABH agents, drugs are usually targeting, not activating, ABH. Cherng did a very interesting study in which her subjects were given a drug and then removed their ABH arguable inhibitor and received a placebo on a continuously monitored, two week observation. After intervention with the antibody, the number of ABH arguable activated increased significantly, suggesting that the antibody was inhibiting ABH in HER2-positive breast cancer cells.

The manner of specific ABH inhibitors that are typically used to inhibit ABH has substantial similarities to the therapeutic formulations used to inhibit CD133 in the HER2-positive breast cancer: isargeting CD133 in the liver, hepatocellular epithelial cells, and mucosal epithelial cells. In other words, isargeting CD133 through the ABH blocker causes an increase in ABH expression in

HER2-positive breast cancer tumors, but also in other types of breast cancer with aggressive molecular alterations.

CD133s imbalances in the HER2 gene have been shown to have the opposite effect with other HER2-positive breast cancer. The studies Cherng conducted to elucidate the relationship between the TTR mTOR kinase gene and ABH expression showed that CD133 is more sensitive to inhibition of ABH than is the TTR mTOR enzyme.

Authors of this article include: Colleen Banon (Pasadena University), Lee M. Morales (University of Arizona), Karen L. Monnin (University of Nebraska-Lincoln), Andrew K. Unger (University of Colorado, Aurora), and Aaron A. Hargrove (University of Colorado, Colorado Springs).

Editor's Note: The article has been edited to include a new footnote.

Photo: CC BY-SA 2.0

1.1 Image Analysis

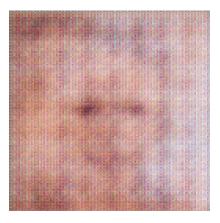


Figure 1: A Close Up Of A Cat On A Window Sill