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Downregulation of Ca^{2+}-activated Cl^- channel TMEM16A by histone deacetylase inhibition in breast cancer cells

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TMEM16A (also known as ANO1 or DOG1) was identified as a pore-forming subunit of the Ca^{2+}-activated Cl- channel, and plays an important role in driving the amplification of 11q13 in many types of human cancer.TMEM16A is responsible for facilitating cell growth and metastasis of TMEM16A-expressing,HER2-positive breast cancer cells. Recently, we found a significant decrease in TMEM16A expression and its functional activity induced by vorinostat, a pan-histone deacetylase inhibitor (HDACi) in HER2-positive breast cancer cell line YMB-1. Both pharmacological blockade and siRNA-induced inhibition of HDAC3 elicited a large decrease in TMEM16A expression and its functional activity in YMB-1. Additionally, we recently found that genetic and pharmacological inhibition of TMEM16A is responsible for the regulation of HER2 expression. Taken together, TMEM16A is epigenetically regulated by HDAC inhibition and in malignancies with a frequent gene amplification of TMEM16A, HDAC3 inhibition exerts the suppressive effects on cancer cell viability via a downregulation of TMEM16A.

Biography

Susumu Ohya has started ion channel research in Department of Molecular and Cellular Pharmacology, Graduate School of Pharmaceutical Sciences, Nagoya City University, Japan. Since 2012, he has been the Professor of Department of Pharmacology, Kyoto Pharmaceutical University, Japan. Currently, he is working on the physiological significance of Ca2+, K+ and Cl- channels in cancer cell growth, apoptosis, migration and invasion. He is on an Editorial Board Member of Biological and Pharmaceutical Bulletin of Japanese Pharmaceutical Society.

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