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Editorial

Patent Highlights in Cancer Gene Therapy's

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APTAMER-siRNA Chimera Molecules for Breast Cancer Treatment

USA Patent Application US2017275629, Methods and Compositions for the Treatment of Cancer (Inventors: Lieberman J, Gilboa-Geffeb A, Wheeler LA; applicant: Children's Medical Center Corp., USA; published: September 28, 2017).

RNA interference offers the opportunity to treat disease, e.g. cancer, by knocking down disease-causing genes. Chimeric aptamer-siRNA molecules target cancer cell markers to direct the siRNA specifically to the cancer cells. The Boston Children's Hospital Center Corp operates as a non-profit organization that offers health care services such as breast cancer, autism, brain injury, cardiac anesthesia, and pediatrician, filed the application underlying this patent disclosure on August 28, 2015. The concept is interesting: a chimeric aptamer-siRNA molecules (AsiCs) comprising a cancer EPCAM-binding aptamer domain (GCGACUGGUUACCCGGUCGUUU) and an inhibitory nucleic acid that inhibits the expression of a gene selected from the group consisting of: Plk1 (NCBI Gene ID: 5347), MCL1 (NCBI Gene ID: 4170), EphA2 (NCBI Gene ID: 1969), PsmA2 (NCBI Gene ID: 5683), MSI1 (NCBI Gene ID: 4440), BMI1 (NCBI Gene ID: 648), XBP1 (NCBI Gene ID: 7494), PRPF8 (NCBI Gene ID: 10594), PFPF38A (NCBI Gene ID: 84950), RBM22 (NCBI Gene ID: 55696), USP39 (NCBI Gene ID: 10713), RAN (NCBI Gene ID: 5901), NUP205 (NCBI Gene ID: 23165), and NDC80 (NCBI Gene ID: 10403). Particularly, EpCAM AsiC targeting PLK1 specifically inhibits cell proliferation in basal A breast cancer cells when the effect of EpCAM-AsiC targeting PLK1 on cell proliferation was tested on 10 breast cancer cell lines representative of basal A, B and luminal cell lines. In addition, EPCAM AsiCs are stable in human serum and do not stimulate innate immunity which allows an increase in administration efficiency and therapeutic efficacy while reducing the potential for side effects.

NCBI Gene ID: 4072 (EPCAM: epithelial cell adhesion molecule).

siRNA Silencing the Expression of Large Tumor Suppressor Kinase for Use to Treat Breast Cancer

USA Patent Application US2017298360, LATS and Breast Cancer (Inventors: Bentires-Al M, Britschgi A; applicant: Friedrich Miescher Institute for Biomedical Research, Switzerland; published: October 19, 2017).

Breast cancer originates in the epithelium of the mammary gland, which consists of differentiated luminal epithelial and basal myoepithelial cells, as well as undifferentiated stem cells, and more restricted progenitors. The Friedich Miescher Institute, a center dedicated to biomedical research, filed the application underlying this patent disclosure on September 23, 2015. Interestingly it is described that the elimination of large tumor suppressor kinases (LATS) 1 and 2, which are part of the Hippo pathway promotes the luminal phenotype and increases the number of luminal and bipotent progenitors, which are the proposed cells-of-origin of most breast cancers. Particularly, patent application describes a method of treating breast cancer in patients having ERα negative type through the use of siRNAs (siRNA IDs: siYAP s20366 and s20368, siTAZ s13807 and s13806, siERα s4823 and s4825) as silencers of expression of LATS.

NCBI Gene ID: 9113 (LATS 1: large tumor suppressor kinase 1).

NCBI Gene ID: 26524 (LATS 2: large tumor suppressor kinase 2).

Knockdown of ANLN Reduces Liver Tumor Development

International Patent Application WO2017213851, Inhibitory RNA-based therapeutics targeting ANLN for cancer treatment (Inventors: Zhu H, Zhang S; applicant: University of Texas, USA; published: December 14, 2017).

The blocking of cytokinesis, the last step of mitosis, has been an unsuccessful modality for the treatment of cancer because it involves damage to normal cells that are in division. However, since the human liver naturally harbors a significant number of polyploid cells, cytokinesis blockade is likely to function as a treat-

ment method in patients with hepatocellular carcinoma (HCC). University of Texas, a leading institution in cancer research, filed the application underlying this patent disclosure on June 9, 2016. The invention provides an interfering RNA (GGCUCUCUGCAGAUACUAATT) that targets an anillin actin binding protein (ANLM) mRNA. In the first instance it was observed that ANLM was overexpressed (2-fold higher) in human HCC tissues compared to normal liver. Since ANLN participates in the process of cytokinesis, it was tested whether suppression of ANLN expression could be an effective strategy against cytokinesis and, therefore, tumor cell formation of immortalized mouse liver cells (H2.5). Indeed, silencing by siRNAs (10 distinct ANLN shRNAs) demonstrated that the cells in division required ANLN to complete the cytokinesis and proliferate. H2.5 cells transfected with ANLN shRNA were injected via splenic in immunosuppressive fumarylacetoacetate hydrolase-deficient (FRG <"/ _>) mice observing the appearance of liver tumors. *In vivo* knockdown of ANLN did not interfere with liver function or regeneration.

NCBI Gene ID: 54443 (ANLN: anillin actin binding protein).

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