gene therapy

	NCT Number	Title	Authors	Description	Identifier	Dates
1	pubmed:36115073	Combined HASPIN and mTOR inhibition is synergistic against KRAS-driven carcinomas	Chenyue Xu Qiongmei Gao Zhengming Wu Weijuan Lou Xiaoyan Li Menghui Wang Nianhong Wang Qingquan Li	CONCLUSIONS: These findings indicate that increased DNA damage and mitotic catastrophe are the basis for the effective synergistic effect observed with mTOR and HASPIN inhibition, and support the clinical evaluation of this dual therapy in patients with KRAS-mutant tumors.	pmid:36115073 doi:10.1016/j.tranon.2022.101540	Sat, 17 Sep 2022 06:00:00 -0400
2	pubmed:36115112	A designed peptide-based vaccine to combat Brucella melitensis, B. suis and B. abortus: Harnessing an epitope mapping and immunoinformatics approach	Hossein Tarrahimofrad Javad Zamani Michael R Hamblin Maryam Darvish Hamed Mirzaei	Vaccines against Brucella abortus, B. melitensis and B. suis have been based on weakened or killed bacteria, however there is no recombinant vaccine for disease prevention or therapy. This study attempted to predict IFN- epitopes, T cell cytotoxicity, and T lymphocytes in order to produce a multiepitope vaccine based on BtpA, Omp16, Omp28, virB10, Omp25, and Omp31 antigens against B. melitensis, B. abortus, and B. suis. AAY, GPGPG, and EAAAK peptides were used as epitope linkers, while the	pmid:36115112 doi:10.1016/j.biopha.2022.113557	Sat, 17 Sep 2022 06:00:00 -0400
3	pubmed:36115593	NEK2 inactivates the Hippo pathway to advance the proliferation of cervical cancer cells by cooperating with STRIPAK complexes	Yan-Ru Zhang Peng-Sheng Zheng	The never in mitosis gene A (NIMA)-related kinase 2 (NEK2) protein has been reported to be an oncoprotein that plays different oncogenic roles in multiple cancers. Here, we confirmed that NEK2 highly expressed in cervical cancer cells rather than in normal epithelial basal layer cells in cervical tissues and correlated with worse outcomes. We also demonstrated that NEK2 promoted the in vivo growth of subcutaneous xenograft tumors stemming from cervical cancer cells and the in vitro cell	pmid:36115593 doi:10.1016/j.canlet.2022.215917	Sat, 17 Sep 2022 06:00:00 -0400
4	pubmed:36115656	Tumor pH-functionalized and charge-tunable nanoparticles for the nucleus/cytoplasm-directed delivery of oxaliplatin and miRNA in the treatment of head and neck cancer	Yu-Li Lo Hua-Ching Lin Wei-Hsuan Tseng	Prospective tumor pH-responsive and charge-convertible nanoparticles have been utilized to reduce side effects and improve the active tumor-targeting ability and nuclear/cytoplasmic localization of chemo-and gene therapeutics for the treatment of head and neck cancer (HNC). Oxaliplatin (Oxa) is a third-generation platinum compound that prevents DNA replication. miR-320 may regulate cancer cell apoptosis, resistance, and progression. Innovative nanoparticles incorporating miR-320 and Oxa were	pmid:36115656 doi:10.1016/j.actbio.2022.09.027	Sat, 17 Sep 2022 06:00:00 -0400
5	pubmed:36115690	Clonality in immune aplastic anemia: Mechanisms of immune escape or malignant transformation	Jibran Durrani Emma M Groarke	Aplastic anemia (AA) is the prototypic bone marrow failure syndrome and can be classified as either acquired or inherited. Inherited forms are due to the effects of germline mutations, while acquired AA is suspected to result from cytotoxic T-cell mediated immune attack on hematopoietic stem and progenitor cells. Once thought to be a purely "benign" condition, clonality in the form of chromosomal abnormalities and single nucleotide variants is now well recognized in AA. Mechanisms underpinning	pmid:36115690 doi:10.1053/j.seminhematol.2022.08.001	Sat, 17 Sep 2022 06:00:00 -0400

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6	pubmed:36115765	Under the hood: The molecular biology driving gene therapy for the treatment of sickle cell disease	Evan Waldron Yvette C Tanhehco	Gene therapy will soon become the dominant modality for treating of sickle cell disease (SCD). Currently, three technologies are the most promising: expression of transgenic globin genes via a lentiviral vector, controlled mutation of the -globin control cluster by transgenic CRISPR-based ribonucleoprotein, and suppression of BCL11a mRNA by shRNA. In this review, we discuss the mechanism of each technology and how they correct the SCD pathology at the molecular level. We conclude by discussing	pmid:36115765 doi:10.1016/j.transci.2022.103566	Sat, 17 Sep 2022 06:00:00 -0400
7	pubmed:36115838	Spatio-temporal analysis of prostate tumors in situ suggests pre-existence of treatment-resistant clones	Maja Marklund Niklas Schultz Stefanie Friedrich Emelie Berglund Firas Tarish Anna Tanoglidi Yao Liu Ludvig Bergenstråhle Andrew Erickson Thomas Helleday Alastair D Lamb Erik Sonnhammer Joakim Lundeberg	The molecular mechanisms underlying lethal castration-resistant prostate cancer remain poorly understood, with intratumoral heterogeneity a likely contributing factor. To examine the temporal aspects of resistance, we analyze tumor heterogeneity in needle biopsies collected before and after treatment with androgen deprivation therapy. By doing so, we are able to couple clinical responsiveness and morphological information such as Gleason score to transcriptome-wide data. Our data-driven analysis	pmid:36115838 doi:10.1038/s41467-022-33069-3	Sat, 17 Sep 2022 06:00:00 -0400
8	pubmed:36115843	PFKFB4 interacts with FBXO28 to promote HIF-1 signaling in glioblastoma	Emma Phillips Jörg Balss Frederic Bethke Stefan Pusch Stefan Christen Thomas Hielscher Martina Schnölzer Michael N C Fletcher Antje Habel Claudia Tessmer Lisa-Marie Brenner Mona Göttmann David Capper Christel Herold-Mende Andreas von Deimling Sarah-Maria Fendt Violaine Goidts	Glioblastoma is a highly aggressive brain tumor for which there is no cure. The metabolic enzyme 6-Phosphofructo-2-Kinase/Fructose-2,6-Biphosphatase 4 (PFKFB4) is essential for glioblastoma stemlike cell (GSC) survival but its mode of action is unclear. Understanding the role of PFKFB4 in tumor cell survival could allow it to be leveraged in a cancer therapy. Here, we show the importance of PFKFB4 for glioblastoma growth in vivo in an orthotopic patient derived mouse model. In an evaluation of	pmid:36115843 doi:10.1038/s41389-022-00433-3	Sat, 17 Sep 2022 06:00:00 -0400
9	pubmed:36115852	Protein tyrosine kinase inhibitor resistance in malignant tumors: molecular mechanisms and future perspective	Yang Yang Shuo Li Yujiao Wang Yi Zhao Qiu Li	Protein tyrosine kinases (PTKs) are a class of proteins with tyrosine kinase activity that phosphorylate tyrosine residues of critical molecules in signaling pathways. Their basal function is essential for maintaining normal cell growth and differentiation. However, aberrant activation of PTKs caused by various factors can deviate cell function from the expected trajectory to an abnormal growth state, leading to carcinogenesis. Inhibiting the aberrant PTK function could inhibit tumor growth	pmid:36115852 doi:10.1038/s41392-022-01168-8	Sat, 17 Sep 2022 06:00:00 -0400

NCT Number	Title	Authors	Description	Identifier	Dates
10 pubmed:36115937	GBP2 acts as a member of the interferon signalling pathway in lupus nephritis	Yuan Zhang Yinping Liao Qing Hang Dong Sun Ya Liu	Lupus nephritis (LN) is a common and serious clinical manifestation of systemic lupus erythematosus. However, the pathogenesis of LN is not fully understood. The currently available treatments do not cure the disease and appear to have a variety of side effects in the long term. The purpose of this study was to search for key molecules involved in the LN immune response through bioinformatics techniques to provide a reference for LN-specific targeted therapy. The GSE112943 dataset was downloaded	pmid:36115937 doi:10.1186/s12865-022-00520-5	Sat, 17 Sep 2022 06:00:00 -0400