Title (en)

KILLING HUMAN LYMPHOMA AND LEUKEMIA CANCER CELLS AND TCR-ACTIVATED NORMAL HUMAN CELLS BY DOPAMINE D1R AGONISTS

Title (de)

ABTÖTEN HUMANER LYMPHOM- UND LEUKÄMIE-KREBSZELLEN SOWIE TCR-AKTIVIERTER NORMALER HUMANER ZELLEN DURCH DOPAMIN-D1R-AGONISTEN

Title (fr)

ELIMINATION DES CELLULES CANCÉREUSES HUMAINES DU LYMPHOME ET DE LA LEUCÉMIE ET DES CELLULES HUMAINES NORMALES ACTIVÉES PAR LE TCR AU MOYEN D'AGONISTES DE LA DOPAMINE D1R

Publication

EP 1917277 A4 20090805 (EN)

Application

EP 06800733 A 20060803

Priority

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Abstract (en)

[origin: WO2007019267A1] Blocking the voltage-gated potassium channel Kv1.1 of T-cells causes the robust and exclusive production of TNF-a, and thus can be used for eradication of cancer, improved eradication of infectious organisms, increased permeability of blood vessels and the blood brain barriers to given molecules and cells, and improved neuronal features, regeneration function and development. Blocking the voltage-gated potassium channel Kv1.1 of T-cells causes the robust and exclusive production of TNF-a. Similarly, unblocking of a blocked Kv1.1 channel or opening of a Kv1.1 channel will prevent the T-cells from producing and secreting excess amounts of TNF-a, thus being useful in the treatment of conditions such as rheumatoid arthritis and for treating neurological diseases associated with defected functioning and/or pathological block of the Kv1.1 channel, among them PNH associated with Kv1 Abs; Encephalitis associated with Kv1 Abs; and Episodic-ataxia type 1 (EA-1), in all of which the T-cell blocked Kv1.1 channel may secrete excess TNFa and thereby contribute to the pathology. Blocking of the Kv1.1 channel may be achieved in vivo or ex vivo by contact with a selective Kv1.1 channel blocking molecule such as Dendrotoxin-K or a selective monoclonal antibody against the Kv1.1 channel. Preventing the Kv1.1. block would be achieved by Kv1.1 openers, or by molecules that would prevent the closure of the Kv1.1 channel.

IPC 8 full level

C07K 14/705 (2006.01)

CPC (source: EP)

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Citation (search report)

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