comprising a pyrrolidinyl ring, compositions and methods for treatment of Central Nervous System (CNS) dysfunctions, neurotoxic damage, or neurodegenerative diseases. These compounds are particularly useful for treating neurotoxic injury which follows periods of hypoxia, anoxia or ischemia associated with stroke, cardiac arrest or perinatal asphyxia. In addition these compounds are also useful as antipsychotics and anticonvulsives.

Unlike other tissues which can survive extended periods of hypoxia, brain tissue is particularly sensitive to deprivation of oxygen or energy. Permanent damage to neurons can occur during brief periods of hypoxia, anoxia or ischemia. Neurotoxic injury is known to be caused or accelerated by certain excitatory amino acids (EAA) found naturally in the CNS. Compounds as described herein block the action of EEA synaptic receptors and thus can prevent neurotoxic injury.

Treatment of CNS disorders and diseases such as cerebral ischemia, psychotic disorders, convulsions and parkinsonism, as well as prevention of neurotoxic damage and neurodegenerative diseases, may be accomplished by administration of a therapeutically-effective amount of a compound of a class described herein.

Severe Renal Glomerular Disease in Mice Homozygous for Targeted Disruption of Uteroglobin Gene: A Model for Human Hereditary Glomerulopathies

AB Mukherjee, Z Zhang (NICHD)

OTT Reference No. E–164–96/0

Licensing Contact: David Sadowski; *phone:* 301/496–7735 ext. 288; *e-mail:* DS27A@NIH.GOV

Uteroglobin (UG) is a protein fraction of pregnant uterine fluid which can induce and regulate blastocystic development and also possesses important anti-inflammatory properties. This invention describes a novel physiological function of UG, which is its role in preventing severe fibronectin (Fn) deposit-associated renal glomerular disease. Uteroglobin binds to Fn thereby inhibiting the formation of Fn-Fn aggregates and Fn-collagen aggregates, thus preventing the disease. Uteroglobin knockout mice (UG-/-) were generated by targeted disruption of the UG gene. These mice developed glomerular disease, became cachectic and died within 4–5 weeks after birth.

This mouse could potentially be a valuable model system for the study and treatment of glomerular disease.

A description of this research may be found in Science, vol. 276, pp. 1408–1412, 1997.

A Method for Producing Retrovirus RNA Packaging Cassettes Amplified in the Cytoplasm by Autocatalytic Togavirus Vectors

R Morgan, J Wahlfors, K Xanthopoulos (NHGRI)

OTT Reference No. E–135–96/0 filed 25 Sep 96

Licensing Contact: Larry Tiffany; *phone:* 301/496–7056 ext. 206; *e-mail:* LT10X@NIH.GOV

Retroviral vectors are currently the most advanced system available for mammalian gene therapy. The major obstacle with the previous methods is that the transfer of complex or large genomic elements is virtually impossible. This technology obviates the need for the retrovirus DNA provirus stage of the life cycle via retroviral RNA vectors. Specifically, this invention utilizes Togaviruses, especially the Semliki Forest virus (SFV), to produce recombinant retroviral vector RNA in the cytoplasm of a retrovirus packaging cell. Using the SFV system, a retroviral cassette with a heterologous gene is cloned into an SFV expression vector. This in vitro transcribed RNA vector is used to transduce packaging cells. The retroviral RNA vector is amplified in the cytoplasm using the SFV system, and packaged into infectious viral particles. This system represents a means by which large fragments of viral RNA, or complex gene structures, can be transferred via retroviral vectors. An additional advantage is that by using the SFV production system, it is able to produce high titers of retrovirus particles, due to its self-amplification capabilities.

Potential areas of application include: *ex vivo* and *in vivo gene* therapy for infectious (e.g., HIV) and noninfectious (e.g., cancer, birth defects) disease; untranslated genomic regions of DNA may be important for regulation of gene expression.

Dated: August 5, 1997.

Barbara M. McGarey,

Deputy Director, Office of Technology Transfer.

[FR Doc. 97–21401 Filed 8–12–97; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Eye Institute

Notice of the meeting of the National Advisory Eye Council Pursuant to Pub. L. 92–463, notice is hereby given of the meeting of the National Advisory Eye Council (NAEC) on September 11–12, 1997, Executive Plaza North, Conference Room G, 6130 Executive Boulevard, Bethesda, Maryland.

The NAEC meeting will be open to the public on September 11, from 8:30 a.m. until approximately 11:30 a.m. Following opening remarks by the Director, NEI, there will be presentations by the staff of the Institute and discussions concerning Institute programs and policies. Attendance by the public at the open session will be limited to space available.

In accordance with provisions set forth in sec. 552b(c)(4) and 552b(c)(6), Title 5, U.S.C. and sec. 10(d) of Pub. L. 92–463, the meeting of the NAEC will be closed to the public on September 11 from approximately 11:30 a.m. until adjournment at approximately 5:00 p.m. for the review, discussion, and evaluation of individual grant applications. These applications and the discussions could reveal confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Ms. Lois DeNinno, Council Assistant, National Eye Institute, EPS, Suite 350, 6120 Executive Boulevard, MSC–7164, Bethesda, Maryland 20892–7164, (301) 496–9110, will provide a summary of the meeting, roster of committee members, and substantive program information upon request. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should contact Ms. DeNinno in advance of the meeting.

(Catalog of Federal Domestic Assistant Program No. 93.867, Vision Research: National Institutes of Health)

Dated: August 7, 1997.

LaVerne Y. Stringfield,

Committee Management Officer, NIH.

[FR Doc. 97–21423 Filed 8–12–97; 8:45 am]

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