Cannabinoids As Neuroprotectants

A Hampson, J Axelrod, M Grimaldi (NIMH)
DHHS Reference Nos. E–287–97/0 filed 21 Apr 98 and E–287–97/1 filed 10 Aug 98
Licensing Contact: Stephen Finley, 301/496–7735 ext. 215

This technology describes the neuroprotective properties of cannabidiol (CBD), 2-[3-Methyl-6-(1-methylethenyl)-2-cyclohexen-1y1]-5-pentyl-1,3-benzenediol. Cannabidiol is a neuroprotective cannabinoid that does not possess the psychoactive qualities which have previously hampered the development of cannabinoid-based therapeutics. Cannabidiol is an effective blood-brain barrier permeable antioxidant, that is more potent than either tocopherol or ascorbate. As reported in PNAS 95, 8268–73 (July 1998), CBD can protect neurons from both glutamate and free radical induced toxicity. It is believed that CBD may present a viable alternative for treatment of ischemia or physical traumas. This technology is currently available for either licensing or collaborative efforts under a Cooperative Research and Development Agreement (CRADA).

Methods and Compositions for Inhibiting Inflammation and Angiogenesis

K Kelly (NCI)
PCT/US97/19772 filed 24 Oct 97 (claiming priority of USSN 60/027,871 filed 25 Oct 96)
Licensing Contact: Charles Maynard, 301/496–7735 ext. 243

The invention provides compositions and methods directed to isolated α subunits of the 7TM protein CD97. CD97 is a heterodimer existing in three isoforms, namely three forms of α subunit and one invariant β subunit. The invention provides compositions and methods for detecting a subunit of CD97, a T-cell protein which is upregulated in activated T-cells and is involved in the onset and maintenance of inflammation and angiogenesis. The invention provides an isolated protein comprising a soluble CD97 α subunit, and an isolated nucleic acid encoding a soluble CD97 α subunit protein. The invention also provides methods for identifying compounds which inhibit soluble CD97 α subunit expression. The invention may be used to inhibit angiogenesis associated with chronic inflammation in a mammal by administering a therapeutically effective amount of a CD97 antagonist. Another application includes determining the degree of inflammation at a site in a mammal with an antibody composition specifically reactive to a soluble CD97 α subunit. Further, it should be noted that these compositions and methods have in vitro utility in the construction of proteins and subsequences thereof for the construction of antibodies, and nucleic acids and subsequences thereof for use as probes.

Genetic Polymorphisms Of Interleukin-1 Alpha And Beta Associated With Early Onset Periodontitis

SR Diehl, HA Schenkein, YF Wang (NIDR)
Serial No. 09/035,220 filed 05 Mar 97
Licensing Contact: Dennis Penn, 301/496–7056 ext. 211

Periodontal disease occurs in 10–20% of adults, and constitutes a major cause of tooth loss. About 0.5% of U.S. adolescents between the ages of 14 to 17 years old (about 70,000) have localized early onset periodontitis and 0.1% (17,000) have the more destructive form known as generalized early onset periodontitis. Both types of early onset periodontitis often lead to tooth loss before the age of 20. Extrapolation of these figures up to age 35 leads to estimates of early onset periodontitis having a major impact on the dental health of 400,000 individuals in the U.S. population. Discovery of genetic polymorphisms at the interleukin 1 alpha and 1 beta genes significantly associated with disease risk allows genetic testing to be used to predict disease prior to onset. This can be used to target clinical efforts for disease prevention to those individuals at greatest risk. The genetic test can also justify more aggressive therapeutic treatments for individuals already affected by the early onset periodontitis who, based on their genetic profile, are predicted to exhibit very rapid disease progression.

Dated: October 24, 1998.

Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer.

[FR Doc. 98–29072 Filed 10–29–98; 8:45 am]

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Diabetes and Digestive and Kidney Diseases; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552(b)(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute of Diabetes and Digestive and Kidney Diseases Special Emphasis Panel.

Date: December 7–9, 1998.

Time: December 7, 1998, 7:30 PM to Adjournment.

Agenda: To review and evaluate grant applications.

Place: Durham Hilton, 3800 Hillsborough Road, Durham, NC 27705.

Contact Person: FRANCISCO O. CALVO, PHD, Chief, S.E.P. Section, Chief, Special Emphasis Panel, Review Branch, DEA, NIDDK, Natcher Building, Room 6AS–37E, National Institutes of Health, Bethesda, MD 20892–6600, (301) 594–8897.

(Catalogue of Federal Domestic Assistance Program Nos. 93.847, Diabetes, Endocrinology and Metabolic Research; 93.848, Digestive Diseases and Nutrition Research; 93.849, Kidney Diseases, Urology and Hematology Research, National Institutes of Health, HHS)

Dated: October 26, 1998.

LaVerne Y. Stringfield,

Committee Management Officer, NIH.

[FR Doc. 98–29067 Filed 10–29–98; 8:45 am]

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute of Dental Research; Notice of Closed Meetings

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended (5 U.S.C. Appendix 2), notice is hereby given of the following meetings.

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