Application No.	Drug	Applicant
ANDA 62–455	Polymyxin B Sulfate, USP (for prescription compounding)	Do.
ANDA 62–456	Bacitracin Powder, USP (for prescription compounding)	Do.
ANDA 74–084	Diltiazem Hydrochloride Tablets USP, 30 mg and 60 mg	Novopharm N.C., Inc., agent for Novopharm Ltd., 4700 Novopharm Blvd., Wilson, NC 27893.
ANDA 74–511	SULSTER (Sulfacetamide Sodium and Prednisolone Sodium Phosphate Ophthalmic Solution, 10%/eq. 0.23% phosphate)	Taylor Pharmaceuticals (an Akorn Co.), 150 South Wyckles Rd., P.O. Box 1220, Decatur, IL 62525–1220.
ANDA 80–025	Sulf-10 (Sulfacetamide Sodium Ophthalmic Solution, USP) 10%	Ciba Vision, 11460 Johns Creek Pkwy., Duluth, GA 30097–1556.
ANDA 83–648	Meprotabs (Meprobamate Tablets USP, 400 mg)	Wallace Laboratories, Division of Carter-Wallace, Inc., Half Acre Rd., P.O. Box 1001, Cranberry, NJ 08512–0181.
ANDA 85–136	Methocarbamol Tablets USP (750 mg)	Forest Laboratories, Inc., 909 Third Ave., New York, NY 10022–4731.
ANDA 85–137	Methocarbamaol Tablets USP (500 mg)	Inwood Laboratories, Inc., 909 Third Ave., New York, NY 10022–4731.
ANDA 86–228	Nitroglycerin Extended-release Capsules (2.5 mg)	Geneva Pharmaceuticals, Inc., 2655 West Midway Blvd., P.O. Box 446, Broomfield, CO 80038–0446.
ANDA 86–230	Nitroglycerin Extended-release Capsules (6.5 mg)	Do.
ANDA 87–797	Triamcinolone Acetonide Cream USP, 0.025%	Alpharma USPD, Inc., 333 Cassell Dr., suite 3500, Baltimore, MD 21224.
ANDA 88–220	Nitroglycerin Extended-release Capsules (9 mg)	Geneva Pharmaceuticals, Inc.

Therefore, under section 505(e) of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. 355(e)) and under authority delegated to the Director, Center for Drug Evaluation and Research (21 CFR 5.82), approval of the applications listed in the table in this document, and all amendments and supplements thereto, is hereby withdrawn, effective September 25, 1998.

Dated: September 14, 1998.

Janet Woodcock,

Director, Center for Drug Evaluation and Research.

[FR Doc. 98–25713 Filed 9–24–98; 8:45 am]

BILLING CODE 4160–01–F

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. 97N–0503]

Agency Information Collection Activities; Announcement of OMB Approval; New Animal Drug Application (NADA), Form FDA 356 V, 21 CFR Part 514

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is announcing that a collection of information entitled ''New Animal Drug Application (NADA), Form FDA 356 V, 21 CFR Part 514'' has been approved by the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995 (the PRA).

FOR FURTHER INFORMATION CONTACT: Denver Presley, Office of Information Resources Management (HFA–250), Food and Drug Administration, 5600 Fishers Lane, Rockville, MD 20857, 301–827–1472.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of June 9, 1998 (63 FR 31505), the agency announced that the proposed information collection had been submitted to OMB for review and clearance under section 3507 of the PRA (44 U.S.C. 3507). An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays currently valid OMB control number. OMB has now approved the information collection and has assigned OMB control number 0910–0032. The approval expires on July 31, 2001.

Dated: September 17, 1998.

William K. Hubbard,

Associate Commissioner for Policy Coordination.

[FR Doc. 98–25642 Filed 9–24–98; 8:45 am]

BILLING CODE 4160–01–F

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, Public Health Service, DHHS.

ACTION: Notice.

SUMMARY: The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

ADDRESSES: Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804; telephone: 301/496–7057; fax: 301/402–0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

Water Soluble Drugs and Methods of Preparing Same

DK Ho et al. (SAIC/NCI)
Serial No. 60/093,284 filed 17 Jul 98
Licensing Contact: Girish Barua, 301/496–7056, ext. 263

Many potential drugs of cancer chemotherapy intended for parenteral administration have been abandoned because the active ingredient is slightly soluble or water-insoluble. Various methods have been developed to allow these drugs to be dissolved in water; however, these methods can be complex and have negative impacts resulting from the use of cosolvents and complexing agents. The present invention addresses these problems by providing a method of producing water-soluble analogues of water-insoluble drugs through derivatization and conjugation with a polar moiety via a thiol ether bond with a

heterobifunctional linking molecule. In particular this invention provides a water-soluble analogue of the antitumor drug, geldanamycin. The analogue is expected to exhibit superior solubility under physiological conditions due to the unique configuration and thus permits the use of water-insoluble parent compounds.

Human and Rat gb2 $GABAG_B$ Receptors

J Clark, T Bonner (NIMH)
Serial No. 60/087,274 filed 29 May 98
Licensing Contact: Charles Maynard, 301/496–7735, ext. 243

Disruption of GABAergic neurotransmission has been implicated in a number of neurological and psychiatric disorders. GABAergic neurotransmission is mediated by two very different types of GABA receptors, the ligand-gated ion channels or $GABA_A$ receptors, and the seven transmembrane domain G protein-coupled $GABA_B$ receptors. $GABA_B$ receptors have been shown to modulate adenylyl cyclase and phosphoinositide hydrolysis, inhibit voltage-sensitive calcium currents, and stimulate potassium currents and phospholipase A_2. New $GABA_B$ receptor cDNAs, designated hgb2 and rgb2 $GABA_B$, have been isolated from both rat and human. The rat and human gb2 receptors share ~95% amino acid identity with each other and 27% identity with the gb1.

Therapeutic Blockage of ICER Synthesis To Prevent ICER-Mediated Inhibition of Immune Cell Activity

PA Cohen, J Bodor, D Weng, GK Koski, BJ Czerniecki (NCI)
Serial No. 60/076,293 filed 27 Feb 98
Licensing
Contact: Girish Barua, 301/496–7056 ext. 263

This invention relates to the use of antisense to the ICER (Inducible cAMP Early Repressor) to protect cells of the immune system against ICER suppression by tumors and infectious pathogens.

Normal functioning of the host's immune cells encompasses the recognition and destruction of cancer cells and infectious pathogens. Such immunologic activities are critically dependent upon local antigen-presenting cell (APC) function and T cell restimulation. It is apparent, however, that tumors and infectious pathogens can escape recognition and rejection through local inhibition of APC and lymphocyte function, through diverse mechanisms including prostaglandin secretion. It has recently been discovered that sustained inhibition of APC and lymphocyte function is inducible with cAMP activating stimuli in tandem with other coordinate stimuli, resulting in sustained intracellular expression of the inhibitory nuclear regulatory molecule ICER (Inducible cAMP Early Repressor).

The present invention potentially prevents inhibitory effects of tumors and infectious pathogens on APC and lymphocyte function by utilizing ICER antisense to block ICER synthesis in cells of the immune system. The goal of such treatment is to prevent ICER synthesis in lymphocytes and APC responding to inhibitory stimuli secreted or induced by tumors and infectious pathogens, thereby rendering the immune system less vulnerable to ICER-mediated immunosuppression.

Signal Transduction Inhibitors of Allergic Reactions

B Vonakis, H Metzger, H Chen (NIAMS)
Serial No. 09/020,116 filed 06 Feb 98
Licensing Contact: Kai Chen, 301/496–7735 ext. 247

Allergic reactions affect nearly 40 million persons in the United States. Allergic reactions are due to a sequential interaction beginning with the extracellular aggregation of the high affinity receptor for IgE (FcεRI) followed by intracellular tyrosine phosphorylation which initiates a further cascade of events eventually leading to histamine and cytokine release. The reaction is initiated by Lyn kinase which is pre-associated with the FcεRI. It was shown that the introduction of a unique portion of the N-terminal region of Lyn A kinase into cells inhibits the receptor tyrosine phosphorylation in a dose and time-dependent manner. Without receptor phosphorylation, allergic reactions can not occur. The NIH is looking for a company to license and independently develop the technology or to work in collaboration with the NIH scientists via a Cooperative Research and Development Agreement to further research and develop the allergy treatment. It is believed that this technology may ultimately lead to an anti-allergy drug or allergy therapy.

Method and System for Identifying Acid-Fast Structures in Slide-Mounted Biological Specimens

AE Lash, LA Liotta (NCI)
Serial No. 60/066,234 filed 20 Nov 97
Licensing Contact: John Fahner-Vihtelic, 301/496–7735 ext. 270

The present application describes a system and method for screening subjects who are suspected of having a mycobacterial infection. After obtaining a specimen of interest, a digitized photomicrographic image of a magnified field of the specimen is color filtered to remove pixels in the red to magenta range. The pixels are grouped and analyzed to determine if they form any structures having an elongated shape associated with mycobacteria. Upon identification of target organisms, an alarm sounds and the section of interest is displayed by the system. Problems associated with locating mycobacteria on a slide and determining their morphological appearance, once found, are virtually eliminated with this invention.

Resonant Structure for Spatial and Spectral-Spatial Imaging of Free Radical Spin Probes Using Radiofrequency Time Domain Electron Paramagnetic Resonance Spectrometry

N Devasahayam et al. (NCI)
Serial No. 60/047,786 filed 27 May 97; PCT/US98/10467 filed 21 May 98
Licensing Contact: John Fahner-Vihtelic, 301/496–7735 ext. 270

The present application represents a significant improvement in resonators for use in electron paramagnetic resonance (EPR) imaging systems. This apparatus is designed to detect time domain EPR responses from spin probes after pulsed excitation using radiofrequency irradiation in the range of 60–400MHz. The invention is configured into an array of numerous surface coils of appropriate diameters connected in a parallel fashion with suitable spacing between individual surface coils to form a volume type resonator. This technology provides necessary capabilities and improvements in EPR systems and overcomes obstacles associated with implementation of EPR spectroscopy diagnostic imaging.

Dated: September 18, 1998.

Jack Spiegel,

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

[FR Doc. 98–25709 Filed 9–24–98; 8:45 am]
BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Cancer Institute; 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.