Dated: May 2, 2007.

Judith Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. 07–2299 Filed 5–8–07; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: Use of Licensee's proprietary delivery formulation containing synthetic peptides of the CEA antigen (or modifications, derivatives, fragments, or immunogenic epitopes thereof) as claimed in the Licensed Patent Rights, alone or in combination with at least one other synthetic peptide for use in the prevention and/or treatment of adenocarcinomas in humans. For the avoidance of doubt, said delivery formulation specifically excludes all poxviruses, eukaryotic expression vectors, and recombinant yeast

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

ACTION: Notice.

SUMMARY: This is notice, in accordance with 35 U.S.C. 209(c)(1) and 37 CFR Part 404.7(a)(1)(i), that the National Institutes of Health, Department of Health and Human Services, is contemplating the grant of an exclusive patent license to practice the inventions embodied in U.S. Patent 6,756,038 and PCT Application Serial No. PCT/US98/19794 and foreign equivalents thereof, entitled ''Agonist and Antagonist Peptides of Carcinoembryonic Antigen (CEA)'' (E–099–1996/0), to Immatics Biotechnologies, GmbH, which is located in Tuebingen, Germany. The patent rights in these inventions have been assigned to the United States of America.

The prospective exclusive license territory may be worldwide and the field of use may be limited to the use of Licensee's proprietary delivery formulation containing synthetic peptides of the CEA antigen (or modifications, derivatives, fragments, or immunogenic epitopes thereof) as claimed in the Licensed Patent Rights, alone or in combination with at least one other synthetic peptide for use in the prevention and/or treatment of adenocarcinomas in humans. For the avoidance of doubt, said delivery formulation specifically excludes all poxviruses, eukaryotic expression vectors, and recombinant yeast.

DATES: Only written comments and/or applications for a license which are received by the NIH Office of Technology Transfer on or before July 9, 2007 will be considered.

ADDRESSES: Requests for copies of the patent application, inquiries, comments, and other materials relating to the contemplated exclusive license should be directed to: Michelle A. Booden, PhD., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852–3804; Telephone: (301) 451–7337; Facsimile: (301) 402–0220; E-mail: *boodenm@mail.nih.gov.*

SUPPLEMENTARY INFORMATION: The technology describes the composition and use of nucleic acid sequences that encode agonist and one antagonist peptide variants of the human carcinoembryonic antigen (CEA) peptide, including but not limited to CAP–1. CEA is an antigen, which is expressed on the surface of various types of cancer cells. It is capable of stimulating a specific cytolytic T cell response, as is CAP–1, which is a highly immunogenic epitope of CEA. Therefore, CAP–1 agonists which are capable of eliciting a CEA-specific cytolytic T cell response, such as those identified by the inventors, may represent potential immunogens for use as therapeutic agents or vaccines against various cancers, and possibly also for use against autoimmune diseases. In fact, at least one of the agonist peptides appears to be more immunogenic than the native CAP–1 peptide. CAP–1 antagonists which are capable of reducing or eliminating this T cell response, such as the antagonist peptide variant identified by the inventors, may represent potential agents for use against autoimmune responses to CEA or to agonist peptide variants thereof.

The prospective exclusive license will be royalty bearing and will comply with the terms and conditions of 35 U.S.C. 209 and 37 CFR Part 404.7. The prospective exclusive license may be granted unless within sixty (60) days from the date of this published notice, the NIH receives written evidence and argument that establishes that the grant of the license would not be consistent with the requirements of 35 U.S.C. 209 and 37 CFR Part 404.7.

Applications for a license in the field of use filed in response to this notice will be treated as objections to the grant of the contemplated exclusive license. Comments and objections submitted to this notice will not be made available for public inspection and, to the extent permitted by law, will not be released under the Freedom of Information Act, 5 U.S.C. 552.

Dated: May 1, 2007.

Steven M. Ferguson,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. E7–8888 Filed 5–8–07; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of an Exclusive License: Development and Commercialization of Therapeutic Products for Rheumatoid Arthritis (RA)

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

ACTION: Notice.

SUMMARY: This notice, in accordance with 35 U.S.C. § 209(c)(1) and 37 CFR Part 404.7(a)(1)(i), announces that the Department of Health and Human Services is contemplating the grant of an exclusive license to practice the inventions embodied in PCT Application No. PCT/US01/04125, filed February 9, 2001, entitled ''Identification of a Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-ligand Receptor Assembly and Function'' [E–095–2000/0–PCT–02]; U.S. Patent No. 7,148,061, issued December 12, 2006, entitled ''Identification of Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-ligand Receptor Assembly and Function'' [E–095–2000/0–US–03]; U.S. Patent Application No. 11/637,272, filed December 12, 2006, entitled ''Identification of Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-ligand Receptor Assembly and Function'' [E–095–2000/0–US–08]; PCT Application No. PCT/US06/24909, filed June 26, 2006, entitled ''A Potential Novel Therapeutic Protein Molecule of Inflammatory Arthritis Targeting the Pre-ligand Assembly Domain (PLAD) of Tumor Necrosis Factor Receptor Type 1'' [E–095–2000/4–PCT–01]; European Patent Application No. 01910476.9, filed February 9, 2001, entitled ''Identification of Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-ligand Receptor Assembly and Function'' [E–095–2000/0–EP–06]; Australian Patent Application No. 2001238076, filed on February 9, 2001, entitled ''Identification of Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-

ligand Receptor Assembly and Function'' [E–095–2000/0–AU–04]; Australian Patent Application No. 2006203490, filed on August 11, 2006, entitled ''Identification of Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-ligand Receptor Assembly and Function'' [E–095–2000/0–AU–07]; and Canadian Patent Application No. 2399388, filed February 9, 2001, entitled ''Identification of Novel Domain in the Tumor Necrosis Factor Receptor Family that Mediates Pre-ligand Receptor Assembly and Function'' [E–095–2000/0–CA–05] to Welson Pharmaceuticals, Inc.

The prospective exclusive license territory may be worldwide and the field of use may be limited to therapeutic applications for rheumatoid arthritis (RA) using Welson's proprietary platform.

DATES: Only written comments and/or license applications which are received by the National Institutes of Health on or before July 9, 2007 will be considered.

ADDRESSES: Requests for copies of the patent and/or patent applications, inquiries, comments and other materials relating to the contemplated exclusive license should be directed to: Mojdeh Bahar, J.D., M.A., Technology Licensing Specialist, Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852–3804. *Telephone:* (301) 435–2950; *Facsimile:* (301) 402–0220; E-mail: *baharm@od.nih.gov.*

SUPPLEMENTARY INFORMATION: The invention relates to methods and compositions that are useful for novel treatment of arthritis and other autoimmune diseases. This technology discloses the identification of a functional domain, Pre-ligand Assembly Domain (PLAD), an essential part in signaling involving receptors of the Tumor Necrosis Factor superfamily and its use in ameliorating rheumatoid arthritis (RA). PLAD is essential for signaling involving TFNR including TNFR–1 (p60), TNFR–2 (p80), Fas, TRAIL–R, LTR, CD40, CD30, CD27, HVEM, OX40 and DR4 and can be isolated as functional polypeptides which can be useful in inhibiting the first step in TNFR mediated signaling, ligand-independent assembly of members of the TNFR superfamily. The ability to inhibit TNFR signaling suggests that these PLAD polypeptides may be useful in development of new therapeutic molecules or as therapeutic molecules themselves used for modulation of immune responses, apoptosis, and inflammation. The inventors have discovered compounds that interfere with PLAD and can block the effects of TNF-alpha.

The prospective exclusive license will be royalty-bearing and will comply with the terms and conditions of 35 U.S.C. 209 and 37 CFR part 404.7. The prospective exclusive license may be granted unless within sixty (60) days from the date of this published notice, the NIH receives written evidence and argument that establish that the grant of the license would not be consistent with the requirements of 35 U.S.C. 209 and 37 CFR part 404.7.

Applications for a license in the field of use filed in response to this notice will be treated as objections to the grant of the contemplated exclusive license. Comments and objections submitted to this notice will not be made available for public inspection and, to the extent permitted by law, will not be released under the Freedom of Information Act, 5 U.S.C. 552.

Dated: April 30, 2007.

Steven M. Ferguson,

Director, Division of Technology Development and Transfer,Office of Technology Transfer,National Institutes of Health.

[FR Doc. E7–8889 Filed 5–8–07; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Prospective Grant of Exclusive License: Treatment of Inflammatory Bowel Disease (IBD) Using IL–13 Modulators and Inhibitors

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

ACTION: Notice.

SUMMARY: This is notice, in accordance with 35 U.S.C. 209(c) (1) and 37 CFR 404.7(a)(1)(i), that the National Institutes of Health (NIH), Department of Health and Human Services (HHS), is contemplating the grant of an exclusive license to practice the invention embodied in:

PCT patent application PCT/US2002/018790 filed 14 June 2002, entitled: ''Methods of Treating and Preventing Colitis involving IL–13 and NK–T Cells'' [HHS Reference Number: E–131–2002/0-PCT–01], to

Wyeth Pharmaceuticals, based in Madison, New Jersey. The field of use may be limited to the use of IL–13 modulators or NK–T cell modulators (such as antibodies) for the treatment or prevention of Inflammatory Bowel Disease, including ulcerative colitis and Crohn's disease. The United States of America is an assignee of the patent rights in these inventions.

DATES: Only written comments and/or application for a license, which are received by the NIH Office of Technology Transfer on or before July 9, 2007 will be considered.

ADDRESSES: Requests for a copy of the patent application, inquiries, comments and other materials relating to the contemplated license should be directed to: Susan Carson, D.Phil., Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, MD 20852–3804; E-mail: *carsonsu@od.nih.gov*; Telephone: (301) 435–5020; Facsimile: (301) 402–0220.

SUPPLEMENTARY INFORMATION: Ulcerative colitis (UC) is a chronic inflammatory disease of the colorectum and affects approximately 400,000 people in the United States. The cause of UC is not known, although an abnormal immunological response by the mucosal T cells responsive to bacterial antigens in the gut microflora, is thought to be involved. Present treatments for UC include anti-inflammatory therapy using aminosalicylates or corticosteroids, as well as immunomodulators and diet. However, 25–40% of ulcerative colitis patients must eventually have their colons removed due to massive bleeding, severe illness, rupture of the colon, risk of cancer or due to side effects of corticosteroids and novel treatments are still actively being sought. NIH scientists and their collaborators have used a mouse model of experimental colitis (oxazolone colitis, OC) to show that IL–13, a Th2 cytokine, is a significant pathologic factor in OC and that neutralizing IL–13 in these animals effectively prevents colitis (Immunity (2002) 17, 629–638).

OC is a colitis induced by intrarectal administration of a relatively low dose of the haptenating agent oxazolone subsequent to skin sensitization with oxazolone. A highly reproducible and chronic colonic inflammation is obtained that is histologically similar to human ulcerative colitis. Studies show that NKT cells rather than conventional CD4+T cells mediate oxazolone colitis and that NKT cells are the source of IL–13, and are activated by CD1 expressing intestinal epithelial cells. Tissue removed from UC patients was also shown to contain increased numbers of nonclassical NKT cells that produce markedly increased amounts of IL–13 and that in keeping with epithelial damage being a key factor in UC, these NKT cells are cytotoxic for epithelial cells (J Clin. Investigation (2004) 113,