• Less immunogenic targeted toxin results in improved efficacy during multiple administrations;

• Targeted therapy decreases non-specific killing of healthy, essential cells, resulting in fewer side-effects and healthier patients.

Development Status: Preclinical stage of development.

Inventors: Pastan (NCI) *et al.*

Patent Status:

• U.S. provisional application 61/241,620 (HHS technology reference E–269–2009/0–US–01);

• PCT patent application PCT/US2010/048504 (HHS technology reference E–269–2009/0–PCT–02).

For more information, see:

• U.S. Patent Publication US 20100215656 A1 (HHS technology reference E–292–2007/0–US–06);

• U.S. Patent Publication US 20090142341 A1 (HHS technology reference E–262–2005/0–US–06);

• U.S. Patent 7,777,019 (HHS technology reference E–129–2001/0–US–07).

Licensing Status: Available for licensing.

Licensing Contact: David A. Lambertson, PhD; 301–435–4632; *lambertsond@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Molecular Biology Section, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact John Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: June 15, 2011.

Richard U. Rodriguez,

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

[FR Doc. 2011–15492 Filed 6–21–11; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

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

ACTION: Notice.

SUMMARY: The inventions listed below are owned by an agency 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.

Mouse Model for Cerebral Cavernous Malformation, an Inherited Brain Disorder

Description of Technology: Cerebral Cavernous Malformation (CCM) is a brain disease affecting up to 0.5% of the worldwide population. CCM is characterized by grossly dilated vessels prone to leaking and hemorrhage which result in severe headaches, seizures, and strokes. Inherited forms of the disease are due to mutations in one of three loci, CCM1, CCM2, and CCM3. Prior efforts to develop mice with targeted null mutations in *Ccm1, Ccm2,* or *Ccm3* have been unsuccessful, as such mutations result in embryonic death.

The inventors have developed the first mouse model available for the study of CCM, in which mouse *Ccm2* can be conditionally deleted in blood-accessible and endothelial cells, resulting in neurological defects, ataxia, and brain hemorrhages consistent with the human disease. The model was generated through a cross of C57BL/6 *Ccm2*-floxed mice with C57BL/6 *MX–1–Cre* mice, which permits inducible ablation by polyinosinic:polycytidylic acid (pIpC).

Inventors: Ulrich Siebenlist (NIAID) and Yoh-suke Mukoyama (NHLBI).

Related Publications: In preparation.

Patent Status: HHS Reference No. E–158–2011/0—Research Material. Patent protection is not being pursued for this technology.

Licensing Status: Available for licensing under a Biological Materials License Agreement.

Licensing Contact: Tara L. Kirby, PhD; 301–435–4426; *tarak@mail.nih.gov.*

System to Increase Consistency and Reduce Variations in Contrast and Sensitivity in MRI Imaging

Description of Technology: The technology relates to the field of MRI. More specifically, the invention describes and claims system and methods related to the use of non-linear B_0 shims to improve excitation flip angle uniformity in high field MRI. The disclosed system and methods can be used in conjunction with existing multi-dimension excitation methods, including those that use parallel excitation to improve contrast and sensitivity in gradient echo magnetic resonance imaging. The technology is designed to overcome shortcomings associated with high field MRI, namely RF flip angle inhomogeneity due to wavelength effects that can lead to spatial variations in contrast and sensitivity.

Applications: High field MRI.

Advantages: The present system and methods will improve performance of high field MRI:

• Improve the transmit profile homogeneity, and therefore the uniformity of MRI images.

• The method is applicable to all MRI scanning with poor B1 uniformity. This includes situations when B1 variations are caused by the coil B1 profile, by the dielectric properties of the object (wavelength effects), or by a combination of both.

• The method is applicable with currently available single or multi-channel B1 coils.

Development Status:

• Proof of principle has been demonstrated on a prototype device.

• Demonstration of the application to human imaging is currently underway.

Inventors: Jeff Duyn (NINDS).

Relevant Publication: Duan Q, van Gelderen P, Duyn J. B_0 based shimming of RF flip angle in MRI. Submitted to Magnetic Resonance in Medicine.

Patent Status: U.S. Provisional Application No. 61/473,610 filed 08 Apr 2011 (HHS Reference No. E–129–2011/0–US–01).

Licensing Status: Available for licensing and commercial development.

Licensing Contacts:

• Uri Reichman, PhD, MBA; 301–435–4616; *UR7a@nih.gov.*

• John Stansberry, PhD; 301–435–5236; *js852e@nih.gov.*

Polyclonal Antibodies Against RGS7, a Regulator of G Protein Signaling, for Research and Diagnostic Use

Description of Technology: Investigators at the National Institutes of Health have generated a polyclonal antibody against the Regulator of G protein Signaling Protein 7 (RGS7). The RGS7 protein regulates neuronal G protein signaling pathways and inhibits signal transduction by increasing the GTPase activity of G protein alpha. RGS7 may play an important role in synaptic vesicle exocytosis and in the

rapid regulation of neuronal excitability and the cellular responses to stimulation. This polyclonal antibody was generated by using a purified fusion protein containing the regulator of guanine nucleotide-binding protein signaling (RGS) C-terminal region of bovine RGS. The antibody specifically recognizes RGS7 of mouse, rat, and human origin. The antibody is useful for studying the expression, functions, and interactions of RGS7 by Western blot and immunofluorescence analysis.

Applications:

• *Basic research tool for the study of RGS7.* Reagent for diagnostic applications such as Western Blotting, ELISA, immunofluorescence and immunohistochemistry in fixed tissue samples.

• *Reagent for biochemical techniques such as immunoprecipitation.* Development of diagnostics or therapeutics for diseases of the nervous system linked to RGS protein-regulated signaling including Parkinson's disease, schizophrenia, seizure disorders, multiple sclerosis, and opiate addiction.

Inventors: William F. Simonds and Jianhua Zhang (NIDDK).

Relevant Publications

1. Rojkova AM, Woodard GE, Huang TC, Combs CA, Zhang JH, Simonds WF. Ggamma subunit-selective G protein beta 5 mutant defines regulators of G protein signaling protein binding requirement for nuclear localization. J Biol Chem. 2003 Apr 4;278(14):12507–12512. [PMID: 12551930]

2. Nini L, Waheed AA, Panicker LM, Czapiga M, Zhang JH, Simonds WF. R7-binding protein targets the G protein beta 5/R7-regulator of G protein signaling complex to lipid rafts in neuronal cells and brain. BMC Biochem. 2007 Sep 19;8:18. [PMID: 17880698]

3. Panicker LM, Zhang JH, Posokhova E, Gastinger MJ, Martemyanov KA, Simonds WF. Nuclear localization of the G protein beta 5/R7-regulator of G protein signaling protein complex is dependent on R7 binding protein. J Neurochem. 2010 Jun;113(5):1101–1112. [PMID: 20100282]

Patent Status: HHS Reference No. E–077–2011/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: This technology is available as a research tool under a Biological Materials License.

Licensing Contact: Jaime Greene, M.S.; 301–435–5559; *greenejaime@mail.nih.gov.*

Collaborative Research Opportunity: The NIDDK Metabolic Diseases Branch is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize polyclonal antibodies against the Regulator of G protein Signaling Protein 7 (RGS7). Please contact Anna Z. Amar at 301–451–2305 or *aa54d@nih.gov* for more information.

Oligonucleotide Compounds that Enhance Immunity to Cancer and Reduce Autoimmunity

Description of Technology: Suppressive cells, including macrophages and other myeloid-derived suppressor cells, regulatory T cells, and dendritic cells (DCs), have been attributed to tumor growth. DCs in particular are known to be associated with the induction of T cell tolerance in cancer, but molecular mechanisms that control DC dysfunction are complex and a better understanding of DC mechanisms in tumors is needed. Recently FOXO3, originally identified as a tumor suppressor, was associated with DC dysfunction. Additionally, therapeutics targeting FOXO3 are known to be effective at killing many tumors types, synergize with traditional therapies, and show efficacy against tumors that are otherwise resistant to conventional treatments.

The researchers at the NIH have demonstrated for the first time that FOXO3 expression by DC coincides with expression of suppressive genes that negatively regulate T cell function. They have also demonstrated that silencing FOXO3 simultaneously changes DC function, eliminating tolerogenicity and enhancing their immunostimulatory capacity. Specifically, the inventors have developed siRNAs or oligonucleotides that enhance an immune response and neutralize the activity of FOXO3 in DCs by converting suppressive cells into immunostimulatory cells. This novel approach could be applied to cancer vaccines, where dendritic cells could be treated with these small molecules prior to use in clinical therapies. Alternatively, small molecules that stimulate FOXO3 expression could be used for inducing immune suppression for autoimmune diseases like type I diabetes or multiple sclerosis.

Applications

• An adjuvant to neutralize FOXO3 and elicit a more potent response to cancer immune-based therapies, either at the time of vaccination or during an on-going anti-tumor immune response.

• Suppressing an immune response through the induction of FOXO3 expression to prevent tissue-specific autoimmune diseases like type I Diabetes or Multiple sclerosis, where known target antigens have been identified.

Advantages

• The ability to treat multiple tumor types linked to FOXO3 expression.

• siRNAs can be delivered to different organs with minimal cytotoxicity.

• Through the modulation of FOXO3 gene expression, therapeutics for both cancer and autoimmune diseases can be developed.

Development Status: Pre-clinical proof of principle.

Inventors: Arthur A. Hurwitz (NCI) *et al.*

Publication: Watkins SK, Zhu Z, Riboldi E, Shafer-Weaver KA, Stagliano KE, Sklavos MM, Ambs S, Yagita H, Hurwitz AA. FOXO3 programs tumor-associated DCs to become tolerogenic in human and murine prostate cancer. J Clin Invest. 2011 Apr 1;121(4):1361–1372. [PubMed: 21436588]

Patent Status

• U.S. Provisional Application No. 61/293,098 filed January 7, 2010 (HHS Reference No. E–086–2010/0–US–01).

• PCT Application No. PCT/US2011/020315 filed January 6, 2011 (HHS Reference No. E–086–2010/0–PCT–02).

Licensing Status: Available for licensing.

Licensing Contact: Whitney Hastings; 301–451–7337; *hastingw@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute Cancer and Inflammation Program is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize agents that both block FOXO3 function and enforce FOXO3 expression. Please contact John Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: June 14, 2011.

Richard U. Rodriguez,

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

[FR Doc. 2011–15477 Filed 6–21–11; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

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

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