## 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.

**Transforming Growth Factor Beta-1 (TGF-β1) Transgenic Mouse Model**

*Description of Technology:* Transforming Growth Factor-β1 (TGF-β1) is a multifunctional cytokine that is involved in many physiological processes such as immune regulation, cell proliferation, angiogenesis, apoptosis, and extracellular matrix deposition. Overexpression of activated TGF-β1 signaling pathway is known to play a role in many disease processes, such as inflammation, fibrosis and tumor metastasis.

NIH inventors have developed a transgenic mouse model, designated $\beta 1^{glo}$, which permits conditional, gene-specific overexpression of TGF-β1. The model features a TGF-β1 transgene for which expression is blocked by a floxed enhanced green fluorescent protein (EGFP) gene downstream of the promoter. Excision of the EGFP gene by Cre recombinase allows expression of TGF-β1. Thus, these mice may be cross-bred with a variety of Cre transgenic mouse lines in order to study the role of TGF-β1 in targeted organ systems and tissues.

*Inventors:* Ashok B. Kulkarni and Bradford E. Hall (NIDCR).

*Publication:* BE Hall, C Zheng, WD Swaim, A Cho, CN Nagineni, MA Eckhaus, KC Flanders, IS Ambudkar, BJ Baum, AB Kulkarni. Conditional overexpression of TGF-beta1 disrupts mouse salivary gland development and function. Lab Invest. 2010 Apr;90(4):543–555. [PubMed: 20142803].

*Patent Status:* HHS Reference No. E–016–2010/0—Research Tool. Patent protection is not being pursued for this technology.

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

*Licensing Contact:* Tara Kirby, PhD; 301–435–4426; *tk200h@nih.gov.*

**A Fertility Test To Detect Ovarian Autoimmune Disease Using Human Recombinant MATER Protein**

*Description of Technology:* The inventors have identified *MATER,* a gene that plays an important role in fertility, and have shown that antibodies against MATER protein are detected at higher frequencies in women experiencing infertility and irregular menstrual periods than in healthy women. The discovery of MATER as an important factor in autoimmune-mediated ovarian dysfunction will facilitate diagnosis and treatment of these disorders. In addition to its critical role in ovarian autoimmunity, the inventors have also discovered that the *MATER* gene plays an essential role in embryonic development.

The invention discloses the *MATER* gene, MATER protein and MATER-specific antibodies. Also disclosed are methods and kits for evaluating female infertility through detection of an abnormal autoimmune response, an abnormal *MATER* gene, or abnormal MATER protein expression.

*Applications*

- Diagnostic test for women suffering from infertility or irregular menstrual periods.
- Tool for the study of early embryonic development.
- Tool for the development of MATER-based contraceptives.

*Development Status:* Established research test, ready for additional clinical research and commercial development.

*Market:* Approximately 10% of women of reproductive age experience infertility, and approximately 5% per year experience menstrual irregularity.

*Inventors:* Lawrence M. Nelson and Zhi-bin Tong (NICHD).

*Publications*

1. Zhi-Bin Tong *et al.* A mouse gene encoding an oocyte antigen associated with autoimmune premature ovarian failure. Endocrinology. 1999 Aug;140(8):3720–3726. [PubMed: 10433232].

2. Zhi-Bin Tong *et al.* Developmental expression and subcellular localization of mouse MATER, an oocyte-specific protein essential for early development. Endocrinology. 2004 Mar;145(3):1427–1434. [PubMed: 14670992].

3. Zhi-Bin Tong *et al.* A human homologue of mouse Mater, a maternal effect gene essential for early embryonic development. Hum Reprod. 2002 Apr; 17(4):903–911. [PubMed: 11925379].

4. Zhi-Bin Tong *et al.* Mater, a maternal effect gene required for early embryonic development in mice. Nat Genet. 2000 Nov;26(3):267–268. [PubMed: 11062459].

*Patent Status*

- U.S. Patent 7,217,811 issued 15 May 2007 (HHS Reference No. E–239–2000/0–US–03).
- U.S. Patent 7,531,635 issued 12 May 2009 (HHS Reference No. E–239–2000/0–US–08).
- U.S. Patent 7,432,067 issued 07 Oct 2008 (HHS Reference No. E–239–2000/0–US–09).
- U.S. Patent 7,189,812 issued 13 Mar 2007 (HHS Reference No. E–239–2000/1–US–02).
- Foreign counterparts issued/pending in Australia, Canada, Europe, and Japan.

*Licensing Status:* Available for licensing.

*Licensing Contact:* Tara Kirby, PhD; 301–435–4427; *tk200h@nih.gov.*

Dated: August 17, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010–20863 Filed 8–20–10; 8:45 am]

**BILLING CODE 4140–01–P**

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**Matrix Metalloproteinase-9 Blade-1 Region Peptides: Use as Cell Migration Modulators**

*Description of Technology:* Matrix metalloproteinase-9 (MMP–9) is an enzyme integrally involved in many normal physiological processes that require degradation and remodeling of the extracellular matrix, such as cell migration and invasion, wound repair, bone remodeling, angiogenesis, and embryonic growth. MMP–9 is shown to be involved in the progression of several diseases including many cancers, cardiovascular diseases, CNS diseases, respiratory diseases, and arthritis. In cancer, MMP–9 is thought to promote growth, migration, and spread of cancer cells by catalyzing the degradation of extracellular matrix proteins, releasing bound growth factors, and allowing cancer cells to escape from the primary tumor.

NIH *Inventors* have discovered that specific polypeptides corresponding to Blade-1 region of MMP–9 hemopexin domain can stimulate migration of cells, specifically the migration of cells expressing β1 integrin. The present technology can be used to develop novel therapeutic candidates for the prevention and treatment of human disease conditions mediated by MMP–9 promoted cell migration, e.g., cancer, inflammation, fibrotic diseases, cardiovascular diseases, CNS diseases, respiratory diseases, angiogenesis and arthritis.

*Applications:* Development of therapeutics for treating or preventing human diseases (cancer) using MMP–9 Blade-1 domain polypeptides or peptide analogs.

*Development Status:* Early-stage.

*Inventors:* SK Akiyama *et al.* (NIEHS)

*Patent Status:* U.S. Provisional Application No. 61/360,328 filed 30 Jun 2010 (HHS Reference No. E–146–2010/0–US–01)

*Licensing Status:* Available for licensing.

*Licensing Contact:* Suryanarayana Vepa, PhD, J.D.; 301–435–5020; *vepas@mail.nih.gov*.

*Collaborative Research Opportunity:* The National Institute of Environmental Health Sciences, Laboratory of Molecular Carcinogenesis, Cell Adhesion Group, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize this technology. Please contact Elizabeth M. Denholm, PhD at 919–541–0981 or *denholme@mail.nih.gov* for more information.

**Melanocyte Pigmentation or Proliferation With Neuregulin: Compositions and Methods to Treat Skin Disorders, Including Skin Cancer**

*Description of Invention:* Human skin pigmentation is regulated by complex and intricate interactions among melanocytes and keratinocytes in the epidermis and fibroblasts in the dermis. A number of factors secreted from keratinocytes and/or from fibroblasts have been shown to be involved in regulating skin pigmentation after UV exposure. NIH investigators have previously demonstrated that the less pigmented and thicker skin on the palms and soles is regulated by underlying fibroblasts in those areas, specifically via a secreted factor (DKK1) that modulates Wnt signaling. Now, using microarray analysis to compare gene expression patterns in 15 different primary dermal fibroblast populations derived from the dorsal trunk skin of three different skin phototypes (I, III and VI), these investigators have identified a number of genes that differ dramatically in expression. One among them, neuregulin 1 (NRG–1), secreted by fibroblasts derived from dark skin, effectively increases the pigmentation of melanocytes in tissue culture and in an artificial skin model and regulates their growth, suggesting it is one of the major factors determining human skin color. NRG-l was observed to be highly expressed by fibroblasts derived from darker skin. NIH investigators believe that NRG–1 increases the proliferation of human melanocytes via the phosphorylation of Akt. These results suggest a potential role for NRG–1 in regulating constitutive human skin color and perhaps its dysfunction in pigmentary skin diseases. Based on these observations, NIH investigators are currently developing compositions and methods of modulating pigmentation and proliferation of a melanocyte to prevent or treat skin disorders, including skin cancer.

*Applications:*

- Therapeutics for skin disorders.
- Therapeutics for skin cancer.

*Development Status:* Early stage and studies on reconstructed skin model and in melanocytes.

*Inventors:* Vincent J. Hearing and Wonseon Choi (NCI)

*Related Publications:*

1. Choi W, Wolber R, Gerwat W, Mann T, Hearing VJ. Characterization of the influence of fibroblasts on melanocyte function and pigmentation. In: Proc. XXth Intl. Pigment Cell Conf., edited by K. Jimbow, Bologna, Italy: Medimond, 2008, p. 79–82.

2. Choi W, Wolber R, Gerwat W, Mann T, Batzer J, Smuda C, Liu H, Kolbe L, Hearing VJ. A novel fibroblast-derived melanogenic paracrine factor neuregulin-1 (NRG–1) that modulates the constitutive color and melanocyte function in human skin. J. Cell Sci. in press, 2010.

*Patent Status:* U.S. Provisional Application No. 61/357,846 filed 23 Jun 2010 (HHS Reference No. E–100–2010/0–US–01).

*Licensing Status:* Available for licensing.

*Licensing Contact:* Suryanarayana Vepa, PhD, J.D.; 301–435–5020; *vepas@mail.nih.gov*.

*Collaborative Research Opportunity:* The Center for Cancer Research, Laboratory of Cell Biology, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize the use of NRG–1 (or modifiers of its function) to regulate skin pigmentation. Please contact John Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

Dated: August 17, 2010.

**Richard U. Rodriguez,**

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

[FR Doc. 2010–20862 Filed 8–20–10; 8:45 am]

**BILLING CODE 4140–01–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### Food and Drug Administration

**[Docket No. FDA–2010–N–0001]**

### Food and Drug Administration Clinical Trial Requirements, Regulations, Compliance, and Good Clinical Practice; Public Workshop

**AGENCY:** Food and Drug Administration, HHS.

**ACTION:** Notice of public workshop.