inflammation and cancer. Using a thioglycollate challenge as a measure of the impact of the deletion of MCP–1, MCP–1 deficient mice exhibit a 60% reduction in the number of monocytes/macrophages at 96 hours compared to wild type mice. Unlike previously generated MCP–1 deficient mice in which the expression of the neighboring gene for MCP–3 is down-regulated (our own data), the expression of MCP–3 is up-regulated in this mouse model.

Applications: This mouse may be useful as an in vivo model for evaluating the role of MCP–1 and MCP–3 in cancer or other diseases associated with inflammation due to the accumulation of monocytes.

Inventor: Teizo Yoshimura (NCI)

Patent Status: HHS Reference No. E–241–2005/0—Research Tool. Patent protection is not being pursued for this technology.

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

Licensing Contact: Betty Tong, PhD; 301–594–6565; *tongb@mail.nih.gov.*

Collaborative Research Opportunity: The National Cancer Institute, Center for Cancer Research, Laboratory of Molecular Immunoregulation, Cancer and Inflammation Program, is seeking statements of capability or interest from parties interested in collaborative research to further develop, evaluate, or commercialize agents useful to treat patients with inflammation or cancer. Please contact John D. Hewes, PhD at 301–435–3121 or *hewesj@mail.nih.gov* for more information.

DU145 Camptothecin (CPT)-Resistant Cell Line

Description of Technology: Drug resistance is a major limitation of chemotherapy. Understanding how drug resistance develops may lead to more effective treatments. This invention describes the DU145 Camptothecin (CPT)-resistant prostate cancer cell line that can be used to study mechanisms of drug resistance.

Inventor: Yves G. Pommier (NCI)

Related Publication: Y Urasaki *et al.* Characterization of a novel topoisomerase I mutation from a camptothecin-resistant human prostate cancer cell line. Cancer Res. 2001 Mar 1;61(5):1964–1969.

Patent Status: HHS Reference No. E–159–2005/0—Research Tool. Patent protection is not being pursued for this technology.

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

Licensing Contact: Betty Tong, PhD; 301–594–6565; *tongb@mail.nih.gov.*

Creation and Characterization of Carcinogen-Altered Mouse Epidermal Cell Lines

Description of Technology: The invention relates to the creation of three (3) cell lines that may be used as models of putative initiated cancer cells. The cell lines can be used in basic research assays and low/high throughput screening assays.

Cell line 308 evolved from a calcium-resistant focus from adult mouse epidermis that was exposed to the carcinogen, 7,12-dimethylbenz[a]anthracene (DMBA). Cell lines F and D were derived by treating primary newborn mouse epidermal cells in culture with *N*-methyl-*N*í-nitro-*N*-nitrosoguanidine (MNNG) and DMBA, respectively. These three (3) noncancerous cell lines derived from differentiation-resistant, carcinogen-induced foci may be considered to be putative initiated cells.

Inventor: Stuart H. Yuspa (NCI)

Related Publications:

1. SH Yuspa and DL Morgan. Mouse skin cells resistant to terminal differentiation associated with initiation of carcinogenesis. Nature 1981 Sep 3;293(5287):72–74.

2. H Hennings et al. Response of carcinogen-altered mouse epidermal cells to phorbol ester tumor promoters and calcium. J Invest Dermatol. 1987 Jan;88(1):60–65.

Patent Status: HHS Reference No. E–154–2004/0—Research Tool. Patent protection is not being pursued for this technology.

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

Licensing Contact: Betty Tong, PhD; 301–594–6565; *tongb@mail.nih.gov.*

A Mouse Model for Conditional Gene Deletion of c-Met Receptor

Description of Technology: c-Met oncogene has been implicated in a variety of human cancers as well as degenerative diseases. Signaling via the c-Met receptor is essential for survival as evidenced by the embryonal death of mice in which the c-Met has been deleted. Further analysis of the role of the signaling pathway supported by c-Met receptor in the adult organism is hindered by its embryonic lethality. The establishment of a mouse model for the conditional c-Met gene deletion will provide a unique opportunity to explore the function of c-Met in the adult mouse by selectively deleting the receptor gene in various tissues. Such a mouse model is established at the National Institutes of Health and available for licensing.

Applications:

- Animal model to study the physiological role of the c-Met receptor.
- Animal model for testing potential drug targeted to the c-Met signal transduction pathway.

Inventor: Snorri S. Thorgeirsson (NCI)

Patent Status: HHS Reference No. E–048–2003/0—Research Tool. Patent protection is not being pursued for this technology.

Licensing Status: c-Met receptor conditional KO mice are available for licensing.

Licensing Contact: Betty Tong, PhD; 301–594–6565; *tongb@mail.nih.gov.*

Dated: April 6, 2009.

Richard U. Rodriguez,

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

[FR Doc. E9–8212 Filed 4–9–09; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, March 26, 2009, 12 p.m. to March 27, 2009, 3 p.m., National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 which was published in the **Federal Register** on March 12, 2009, 74 FR 10748.

The meeting will be held April 16, 2009, 3 p.m. to April 17, 2009, 6 p.m. The meeting location remains the same. The meeting is closed to the public.

Dated: April 2, 2009.

Jennifer Spaeth,

Director, Office of Federal Advisory Committee Policy.

[FR Doc. E9–8018 Filed 4–9–09; 8:45 am]

BILLING CODE 4140–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Amended Notice of Meeting

Notice is hereby given of a change in the meeting of the Center for Scientific Review Special Emphasis Panel, April 20, 2009, 9 a.m. to April 21, 2009, 3 p.m., National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892 which was published in the **Federal Register** on March 31, 2009, 74 FR 14570–14571.