concerning the final effect of the HHS decision to designate a class of employees at the Y–12 Plant in Oak Ridge, Tennessee, as an addition to the Special Exposure Cohort (SEC) under the Energy Employees Occupational Illness Compensation Program Act of 2000. On August 15, 2008, as provided for under 42 U.S.C. 7384q(b), the Secretary of HHS designated the following class of employees as an addition to the SEC:

All employees of the Department of Energy (DOE), its predecessor agencies, and DOE contractors or subcontractors who worked at the Y–12 Plant in Oak Ridge, Tennessee from March 1, 1943 through December 31, 1947 for a number of work days aggregating at least 250 work days occurring either solely under this employment or in combination with work days within the parameters established for one or more other classes of employees in the Special Exposure Cohort.

This designation became effective on September 14, 2008, as provided for under 42 U.S.C. 7384*l*(14)(C). Hence, beginning on September 14, 2008, members of this class of employees, defined as reported in this notice, became members of the Special Exposure Cohort.

FOR FURTHER INFORMATION CONTACT: Larry Elliott, Director, Office of Compensation Analysis and Support, National Institute for Occupational Safety and Health (NIOSH), 4676 Columbia Parkway, MS C–46, Cincinnati, OH 45226, Telephone 513–533–6800 (this is not a toll-free number). Information requests can also be submitted by e-mail to *OCAS@CDC.GOV.*

Dated: September 22, 2008.

Christine M. Branche,

Acting Director, National Institute for Occupational Safety and Health.

[FR Doc. E8–23894 Filed 10–7–08; 8:45 am]

BILLING CODE 4163–19–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of the Secretary

Findings of Scientific Misconduct

AGENCY: Office of the Secretary, HHS.

ACTION: Notice.

SUMMARY: Notice is hereby given that the Office of Research Integrity (ORI) and the Assistant Secretary for Health have taken final action in the following case:

Peili Gu, PhD., Baylor College of Medicine: Based on the report of an investigation conducted by the Baylor College of Medicine (BCM) and an initial review conducted by the Office of Research Integrity (ORI), the U.S. Public Health Service (PHS) found that Dr. Peili Gu, former postdoctoral researcher, Department of Molecular and Cellular Biology, BCM, engaged in scientific misconduct in research supported by National Institute of Diabetes and Kidney Diseases (NIDDK), National Institutes of Health (NIH), grant R01 DK073524, National Institute of Child Health and Human Development (NICHD), NIH, grants T32 HD07165 and U54 HD07495, and National Institute of General Medical Sciences (NIGMS), NIH, grant R01 GM066099. ORI acknowledges Dr. Gu's full cooperation with the BCM misconduct proceedings.

Specifically, PHS found that the Respondent committed misconduct in science with respect to reporting falsified data in the following three papers:

1. Gu, P., LeMenuet, D., Chung, A., & Cooney, A.J. ''Differential Recruitment of Methylated CpG Binding Domains [MBDs] by the Orphan Receptor GCNF Initiates the Repression and Silencing of Oct4 Expression.'' *Mol. Cell. Biology* 26(24):9471–9483, December 2006 (hereafter referred to as the ''MBD paper''):

- Respondent falsified the relative expression level of Oct4 in differentiated P19 cells and embryonic stem cells treated with MBD2 and MBD3 small interfering RNA presented in Figures 5E and 6E, respectively.
- Respondent falsified Figure 6A depicting wild type and GCNF-/- embryonic stem cells to compare the binding of GCNF, MBD2, and MBD3 to the Oct4 gene and the measurement of expression at the RNA and protein levels by deleting in photoshop the GCNF Western blot data in the GCNF-/-cells (to match the lack of expression at the RNA level), and falsified the MBD 2 Western blot data in the GCNF-/-cells (or that depicted in Figure 7C, which shows the exact same data but reportedly from DNA methylation-deficient embryonic stem cells [Dnmt3A/Dnmt3B/ES cells]).
- Respondent falsified the MBD2 wild type and GCNF-/-chromatin Immunoprecipitation (ChIP) data in Figure 6B.

2. Gu, P., Morgan, D.H., Sattar, M, Xu, X., Wagner, R., Raviscioni, M., Lichtarge, O., & Cooney, A.J. ''Evolutionary Trace-Based Peptides Identify a Novel Asymmetric Interaction that Mediates Oligomerization in Nuclear Receptors.'' *Journal of Biological Chemistry* 280(36):31818–31829, September 2005:

- In Figures 3C and 3D, depicting transfected wild-type and mutated HA–GCNF expression levels in undifferentiated and differentiated P19 cells, Respondent planned not to show the data for the Asp307 mutant (the data for the Asp307 mutant were deleted in panel D); however, she falsified Figure 3C by deleting the least intensive band instead of the Asp307 mutant in order to make the overall data appear more consistent and support the claim that there were no significant differences in the expression levels between the GCNF mutants and the wild type HA–GCNF in P19 cells.
- In Figure 4A, where Respondent intended not to show the data for the Asp307 mutant, she falsified the reported results by deleting the least intensive band instead of the Asp307 mutant in order to make the overall data appear more consistent in support of the claim that all mutants were expressed at similar levels in COS1 cells and that the various point mutations had not altered the stability of the protein.
- Respondent falsified Figures 4C and 4D depicting supershift of HA–GCNF homodimers expressed in COS1 cells using anti-GCNF and anti-HA antibodies, respectively, by inserting non-specific bands in each of three lanes of each figure where non-specific bands were not visible in the original data.
- Respondent falsified Figure 5A, which reported the detection of HA–GCNF point mutant expression in retinoic acid-differentiated P19 cells by Western blot with anti-HA antibody, by duplicating a series of lanes in the published figure: Lane 2 is the same as lane 4; lane 3 is the same as lanes 5, 7, and 9, and lane 6 is the same as lanes 8, 10, and 11.
- Respondent falsified Figure 6C, which reported on the dimerization abilities of various GCNF mutants, by cutting and pasting (in photoshop) bands into original lanes 7 and 8 to demonstrate the homodimer; certain of the comparisons reported in the text describing this figure do not appear to be confirmed in a repeat experiment.

3. Gu, P., LeMenuet, D., Chung, A., Mancini, M., Wheeler, D., & Cooney, A.J. Orphan Nuclear Receptor GCNF Is Required for the Repression of Pluripotency Genes during Retinoic Acid-Induced Embryonic Stem Cell Differentiation.'' *Mol. Cell. Biology* 25(19):8507–8519, October 2005:

- Respondent falsified Figure 1A by cutting out lanes and relocating them, wild type GCNF lanes 7 and 8 of the original data becoming lanes 1 and 2 in the published figure; the effect of the falsification was to demonstrate the inverse correlation with expression of Oct4, which did not appear to be confirmed in a repeat of the experiment.
- Respondent falsified Figure 4A by switching the 6 hour and 12 hour Oct4 expression data in the wild type embryonic stem cells (these falsified data also appear in Figure 5B).

Dr. Gu has entered into a Voluntary Settlement Agreement (Agreement) in which she has voluntarily agreed, for a period of three (3) years, beginning on September 12, 2008:

(1) To exclude herself from serving in any advisory capacity to PHS, including but not limited to service on any PHS advisory committee, board, and/or peer review committee, or as a consultant or contractor to PHS; and

(2) That any institution that submits an application for PHS support for a research project on which the Respondent's participation is proposed or that uses the Respondent in any capacity on PHS supported research, or that submits a report of PHS-funded research in which the Respondent is involved, must concurrently submit a plan for monitoring of the Respondent's research to the funding agency and ORI for approval. The monitoring plan must be designed to ensure the scientific integrity of the respondent's research contribution. Respondent agreed that she will not participate in any PHS-supported research until such a monitoring plan is submitted to ORI and the funding agency.

Dr. Gu also agreed that she would immediately cooperate with BCM officials to request retraction of the MBD paper. In the retraction letter, she will state that she alone was responsible for the falsification and fabrication of some of the data reported in the paper.

FOR FURTHER INFORMATION CONTACT: Director, Division of Investigative Oversight, Office of Research Integrity, 1101 Wootton Parkway, Suite 750, Rockville, MD 20852, (240) 453–8800.

Chris B. Pascal,

Director, Office of Research Integrity.

[FR Doc. E8–23819 Filed 10–7–08; 8:45 am]

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of the Secretary

Findings of Scientific Misconduct

AGENCY: Office of the Secretary, HHS.

ACTION: Notice.

SUMMARY: Notice is hereby given that the Office of Research Integrity (ORI) and the Assistant Secretary for Health have taken final action in the following case:

Kirk Sperber, M.D., Mount Sinai School of Medicine: Based on the report of an investigation conducted by the Mount Sinai School of Medicine (MSSM) and additional analysis conducted by the Office of Research Integrity (ORI) in its oversight review, the U.S. Public Health Service (PHS) found that Dr. Kirk Sperber, former Associate Professor, Department of Medicine, Division of Clinical Immunology, MSSM, engaged in scientific misconduct while supported by National Institute of Allergy and Infectious Diseases (NIAID), National Institutes of Health (NIH), grants R01 AI45343 and P01 AI44236, and National Cancer Institute, NIH, grant R29 CA256990.

PHS finds the Respondent engaged in scientific misconduct by falsifying and fabricating data that were included in NIAID, NIH, grant applications R01 AI45343–01A1, R01 AI45343–04A2, and P01 AI44236–05. Respondent's scientific misconduct occurred while he was a faculty member at MSSM. Respondent is no longer employed at MSSM.

Specifically, PHS found that Respondent engaged in scientific misconduct by falsifying and fabricating data in the following publications:

1. In multiple figures reported in Sperber, K., Beuria, P., Singha, N., Gelman, I., Cortes, P., Chen, H., & Kraus, T. ''Induction of apoptosis by HIV–1–infected monocytic cells.'' *Journal of Immunology* 170:1566–1578, 2003 (''2003 *J. Immunol. paper*'') (Retracted in December 2005); by duplicating and reusing panels of FACS data in Figures 1A, 2, 4A, 4B, and 7; by duplicating and reusing lanes of polyacrylamide gels in Figure 3, of Western blot analyses in Figures 5A, 5C, 6C, and 9, and of agarose gels in PCR analyses in Figure 5B; and by duplicating and reusing laser confocal micrographs in Figures 10 and 11. Respondent's claims that Figures 1A, 2, 4A, and 7 were representative of experiments repeated five times and that Figures 3, 4B, 5A, 6C, and 9 were representative of experiments repeated three times constitute additional falsifications. The effect of these misrepresentations was to falsely demonstrate the proapoptotic activity of a protein from a novel cDNA clone isolated from an HIV-infected human macrophage cell line and to falsify its presence in brain and lymphoid tissue from patients with HIV-associated dementia.

2. In Figure 10 reported in Rakoff-Nahoum, S., Chen, H., Kraus, T., George, I., Oei, E., Tyorlin, M., Salik, E., Beuria, P., & Sperber, K. ''Regulation of Class II Expression in Monocytic Cells after HIV–1 Infection.'' *J. Immunol.* 167:2331–2342, 2001 (Retracted in November 2006), by duplicating and reusing four confocal micrographs to misrepresent different panels for the Cath D, 43pol and CD–63, 43neve data; for the Cath D, 43gag and Cath D, 43nef data; for the DAMP, 43 nef and M6PR, 43nef data; and for the M6PR, 43gag and the CD–63, 43gag data. Respondent's reported claim that the results were representative of an experiment repeated five times constitutes an additional falsification.

3. In Figures 3B, 4B, and 6B reporting flow cytometry analyses (FACS) in Chen, H., Yip, Y.K., George, I., Tyorkin, M., Salik, E., & Sperber, K. ''Chronically HIV–1–Infected Monocytic Cells Induce Apoptosis in Cocultured T Cells.'' *J. Immunol.* 161:4257–4267, 1998 (Retracted in November 2006); by reusing two FACS histograms, each to represent 2 different experiments in Figure 3B; by reusing the same FACS histogram as the negative control for CD–4 cells and for the CD–8 cells in Figure 4B; and by duplications of the top two panels, the middle two panels, and the bottom two panels of data as graded dilutions of different fractions in Figure 6B to falsely show that a soluble factor from 43HIV cells induced apoptosis. Figure 6B was also presented in grant application AI45343–01A1 as Figure 5B. Respondent's reported claims that the results in Figures 3B, 4B, and 6B were each representative of experiments that were repeated three times constitute additional falsifications.

PHS also finds that Respondent engaged in scientific misconduct by falsifying and fabricating the following data in NIAID, NIH, research applications R01 AI45343–04A2 and P01 AI44236–05:

4. The results of Figures 1, 6C, 7, 9, 10 and 11 from the 2003 *J. Immunology* paper were reported in NIAID, NIH, grant application R01 AI45343–04A2; nearly all of the figures in the paper were falsified, so that the claims in the