health care facilities searching for potential candidates to fill open health care job opportunities at their sites.

Burden Statement: Burden in this context means the time expended by persons to generate, maintain, retain, disclose or provide the information requested. This includes the time needed to review instructions; to develop, acquire, install and utilize technology and systems for the purpose of collecting, validating and verifying information, processing and maintaining information, and disclosing and providing information; to train personnel and to be able to respond to a collection of information; to search data sources; to complete and review the collection of information; and to transmit or otherwise disclose the information. The total annual burden hours estimated for this ICR are summarized in the table below.

TOTAL ESTIMATED ANNUALIZED BURDEN HOURS

Form name	Number of respondents	Number of responses per respondent	Total responses	Average burden per response (in hours)	Total burden hours
Account Creation	15,600	1	15,600	.08	1,248
Complete Profile	9,400	1	9,400	1	9,400
Total	[1] 15,600		15,600		10,648

[1] The 9,400 respondents who complete their profiles are a subset of the 15,600 respondents who create accounts.

HRSA specifically requests comments on (1) the necessity and utility of the proposed information collection for the proper performance of the agency's functions, (2) the accuracy of the estimated burden, (3) ways to enhance the quality, utility, and clarity of the information to be collected, and (4) the use of automated collection techniques or other forms of information technology to minimize the information collection burden.

Maria G. Button,

Director, Executive Secretariat.

[FR Doc. 2020–17635 Filed 8–11–20; 8:45 am]

BILLING CODE 4165–15–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of the Secretary

Findings of Research Misconduct

AGENCY: Office of the Secretary, Health and Human Services (HHS).

ACTION: Notice.

SUMMARY: Findings of research misconduct have been made against Zhiwei Wang, M.D. (Respondent), former postdoctoral fellow, Department of Pathology, Karmanos Cancer Institute, Wayne State University (WSU). Dr. Wang engaged in research misconduct in research supported by U.S. Public Health Service (PHS) funds, specifically National Cancer Institute (NCI), National Institutes of Health (NIH), grants P20 CA101936, P30 CA022453, R01 CA075059, R01 CA083695, R01 CA101870, R01 CA109389, R01CA131151, R01 CA132794, and U19 CA113317. The administrative actions, including debarment for a period of ten (10) years, were implemented beginning on July 21, 2020, and are detailed below.

FOR FURTHER INFORMATION CONTACT: Elisabeth A. Handley, Director, Office of Research Integrity, 1101 Wootton Parkway, Suite 240, Rockville, MD 20852, (240) 453–8200.

SUPPLEMENTARY INFORMATION: Notice is hereby given that the Office of Research Integrity (ORI) has taken final action in the following case:

Zhiwei Wang, M.D., Wayne State University: Based on the report of an investigation conducted by WSU and additional analysis conducted by ORI in its oversight review, ORI found that Dr. Zhiwei Wang, former postdoctoral fellow, Department of Pathology, Karmanos Cancer Institute, WSU, engaged in research misconduct in research supported by PHS funds, specifically NCI, NIH, grants P20 CA101936, P30 CA022453, R01 CA075059, R01 CA083695, R01 CA101870, R01 CA109389, R01CA131151, R01 CA132794, and U19 CA113317.

ORI found that Respondent engaged in research misconduct by knowingly, intentionally, and/or recklessly falsifying data that were included in grant applications R01 CA120008, R01 CA131151, and R01 CA131456 submitted to NCI, NIH; his 2006 Ph.D. dissertation (hereafter referred to as the ''Dissertation''); and the following published papers:

- Activated K-Ras and INK4a/Arf deficiency promote aggressiveness of pancreatic cancer by induction of EMT consistent with cancer stem cell phenotype. *J Cell Physiol.* 2013 Mar;228(3):556–62 (hereafter referred to as ''*J Cell Physiol.* 2013''). Erratum in: *J Cell Physiol.* 2014 Aug;229(8):1118. Retraction in: *J Cell Physiol.* 2016 Oct;231(10):2304.
- Activated K-ras and INK4a/Arf deficiency cooperate during the development of pancreatic cancer by activation of Notch and NF-κB signaling pathways. *PLoS One* 2011;6(6):e20537 (hereafter referred to as ''*PLoS One* 2011''). Erratum in: *PLoS One* 2014;9(6):e101032. Retraction in: *PLoS One.* 2018 Oct 2;13(10):e0205289.
- Down-regulation of Notch-1 is associated with Akt and FoxM1 in inducing cell growth inhibition and apoptosis in prostate cancer cells. *J Cell Biochem.* 2011 Jan;112(1):78–88 (hereafter referred to as ''*J Cell Biochem.* 2011''). Retraction in: *J Cell Biochem.* 2016 Aug;117(8):1962.
- Down-regulation of Notch-1 and Jagged-1 inhibits prostate cancer cell growth, migration and invasion, and induces apoptosis via inactivation of Akt, mTOR, and NF-κB signaling pathways. *J Cell Biochem.* 2010 Mar 1;109(4):726–36 (hereafter referred to as ''*J Cell Biochem.* 2010''). Retraction in: *J Cell Biochem.* 2016 Aug;117(8):1960.
- TW–37, a small-molecule inhibitor of Bcl-2, inhibits cell growth and invasion in pancreatic cancer. *Int J Cancer* 2008 Aug 15;123(4):958–66 (hereafter referred to as ''*Int J Cancer* 2008''). Retraction in*: Int J Cancer.* 2016 Nov 1;139(9):2146.
- Induction of growth arrest and apoptosis in human breast cancer cells by 3,3-diindolylmethane is associated with induction and nuclear localization of p27kip. *Mol Cancer Ther.* 2008 Feb;7(2):341–9 (hereafter referred to as ''*Mol Cancer Ther.* 2008'').
- Down-regulation of platelet-derived growth factor-D inhibits cell growth and angiogenesis through inactivation of Notch-1 and nuclear factor-κB signaling. *Cancer Res.* 2007 Dec 1; 67(23):11377–85 (hereafter referred to as ''*Cancer Res.*

2007c''). Retraction in: *Cancer Res.* 2018 Sep 15;78(18):5469.

• Down-regulation of Forkhead Box M1 transcription factor leads to the inhibition of invasion and angiogenesis of pancreatic cancer cells. *Cancer Res.* 2007 Sep 1;67(17):8293–300 (hereafter referred to as ''*Cancer Res.* 2007b''). Retraction in: *Cancer Res.* 2018 Sep 15; 78(18):5470.

• Inhibition of angiogenesis and invasion by 3,3′-diindolylmethane is mediated by the nuclear factor-κB downstream target genes MMP–9 and uPA that regulated bioavailability of vascular endothelial growth factor in prostate cancer. *Cancer Res.* 2007 Apr 1;67(7):3310–9 (hereafter referred to as ''*Cancer Res.* 2007a''). Retraction in: *Cancer Res.* 2018 Sep 15; 78(18):5471.

• Notch-1 down-regulation by curcumin is associated with the inhibition of cell growth and the induction of apoptosis in pancreatic cancer cells. *Cancer* 2006 Jun 1;106(11):2503–13 (hereafter referred to as ''*Cancer* 2006''). Retraction in: *Cancer* 2016 Oct 15;122(20):3247.

• Epidermal growth factor receptor-related protein inhibits cell growth and invasion in pancreatic cancer. *Cancer Res.* 2006 Aug 1;66(15):7653–60 (hereafter referred to as ''*Cancer Res.* 2006b''). Retraction in: *Cancer Res.* 2018 Sep 15;78(18):5474.

• Inhibition of nuclear factor kappaβ activity by genistein is mediated via Notch-1 signaling pathway in pancreatic cancer cells. *Int J Cancer* 2006 Apr 15;118(8):1930–6 (hereafter referred to as ''*Int J Cancer* 2006''). Erratum in: *Int J Cancer* 2014 Apr 15;134(8):E3. Retraction in: *Int J Cancer* 2016 Nov 1;139(9):2145.

• Down-regulation of Notch-1 inhibits invasion by inactivation of nuclear factor-kappaβ, vascular endothelial growth factor, and matrix metalloproteinase-9 in pancreatic cancer cells. *Cancer Res.* 2006 Mar 1;66(5):2778–84 (hereafter referred to as ''*Cancer Res.* 2006a''). Retraction in: *Cancer Res.* 2018 Sep 15;78(18):5476.

• Down-regulation of Notch-1 contributes to cell growth inhibition and apoptosis in pancreatic cancer cells. *Mol Cancer Ther.* 2006 Mar;5(3):483–93 (hereafter referred to as ''*Mol Cancer Ther.* 2006''). Retraction in: *Mol Cancer Ther.* 2018 Oct;17(10):2268.

ORI found by a preponderance of evidence that Respondent engaged in research misconduct by intentionally, knowingly, and/or recklessly falsifying and/or fabricating images representing protein expression, invasion and migration assays, and electrophoretic mobility shift assays (EMSA) in experiments designed to identify underlying mechanisms controlling cell proliferation, differentiation, and apoptosis in cancer so that novel targeted therapeutic agents could be identified.

Specifically, Respondent reused and relabeled:

• The same protein bands to represent experimental conditions in:

—Figure 6D (upper panel) in the Dissertation; Figure 1D (upper panel) in *Mol Cancer Ther.* 2006: Down-regulation of Notch-1 expression by siRNA in BxPC–3, HPAC, and PANC–1 cells

—Figure 6D (lower panel) in the Dissertation; Figure 1D (lower panel) in *Mol Cancer Ther.* 2006: Up-regulation of Notch-1 expression by cDNA transfection in BxPC–3, HPAC, and PANC–1 cells

—Figure 8A in *Mol Cancer Ther.* 2006: Down-regulation of Notch-1 expression by genistein and Notch-1 siRNA

—Figure 4 in *Int J Cancer* 2006: Down-regulation of Notch-1 expression by genistein and Notch-1 siRNA

• inhibition of Bcl-X_L (0–72 hours with genistein) in BxPC–3 cells in Figure 20 in the Dissertation, Figure 7B in *Mol Cancer Ther.* 2006, and Figure 3C in *Int J Cancer* 2006 to also represent:

—Inhibition of Bcl-X_L (0–13 uM curcumin) in PANC–1 cells in Figure 3D in *Cancer* 2006

—inhibition of Notch-1 expression (ERRP and Notch-1 siRNA transfection) in BxPC–3 cells in Figure 5A in *Cancer Res.* 2006b

• inhibition of Hes-1 (0–72 hours genistein) in BxPC–3 cells in Figure 7B in *Mol Cancer Ther.* 2006 to also represent:

—Inhibition of Cyclin D1 (0–72 hours with genistein) in BxPC–3 cells in Figure 20 in the Dissertation and Figure 3C in *Int J Cancer* 2006

—inhibition of Cyclin D1 (0–13 uM curcumin in PANC–1 cells) in Figure 3D in *Cancer* 2006

• inhibition of Cyclin D1 (0–72 hours with genistein) in BxPC–3 cells in Figure 7B in *Mol Cancer Ther.* 2006 to also represent inhibition of Hes-1 (0–72 hours with genistein) in BxPC–3 cells in Figure 20 in the Dissertation and Figure 3C in *Int J Cancer* 2006

• expression of Bcl-2 in control and Notch-1 siRNA transfected pancreatic cell lines (BxPC–3, HPAC) in Figure 10 in the Dissertation and Figure 5 in *Mol Cancer Ther.* 2006 to represent expression of Notch-1 in control and PDGF–D siRNA transfected pancreatic cells in Figure 4A in *Cancer Res.* 2007c.

• representing expression of Cyclin D1 and Bcl-X_L in control and Notch-1 siRNA transfected pancreatic cell lines (BxPC–3, HPAC, PANC–1) in Figure 10 in the Dissertation and Figure 5D in *Mol Cancer Ther.* 2006 to represent expression of Hes-1 and Cyclin D1 in control and ERRP-incubated pancreatic cells in Figure 2C in *Cancer Res.* 2006b

• expression of p27 in control and Notch-1 siRNA transfected pancreatic cell lines (HPAC) in Figure 10 in the Dissertation and Figure 5 in *Mol Cancer Ther.* 2006 to represent VEGF protein expression in control and Notch-1 plasmid transfected BxPC–3 cells in Figure 4B in *Cancer Res.* 2006a

• expression of Cyclin D1 in control and Notch-1 siRNA transfected pancreatic cell lines in Figure 10 in the Dissertation and Figure 5 in *Mol Cancer Ther.* 2006 to represent the expression of uPAR genes in control siRNA and FoxM1 siRNA transfected pancreatic cancer cells in Figure 5B in *Cancer Res.* 2007b

• expression of Hes-1 in control and ERRP-incubated pancreatic cancer cells in Figure 2C in *Cancer Res.* 2006b to represent the expression of uPAR genes in control siRNA and FoxM1 siRNA transfected pancreatic cancer cells in Figure 5B in *Cancer Res.* 2007b

• expression of Hes-1 in control and ERRP-incubated pancreatic cells in Figure 2C in *Cancer Res.* 2006b to represent control, TGF-α, and TGF-α+ERRP effects on Notch-1 activation in BxPC–3 cells in Figure 2D in *Cancer Res.* 2006b

• inhibition of Bcl-X_L, Hes-1, and Cyclin D protein expression by genistein in BxPC–3 cells at 0, 24, 48, and 72 hours in three different experiments in Figure 7B in *Mol Cancer Ther.* 2006 to represent the same protein expressions in one experiment in Figure 3C in *Int J Cancer* 2006

• up-regulation of Notch-1 in cDNA-transfected BxPC–3 cells in Figure 5C in *Cancer Res.* 2006b to also show that ERRP inhibits the expression of MMP–9 in Figure 6 in *Cancer Res.* 2006b

• expression of Notch-1 when transfected with Jagged-1 siRNA in PC–3 cells in Figure 5A in *J Cell Biochem.* 2010 to also show the expression of Notch-1 when transfected with Notch-1 siRNA in C4–2B cells in Figure 3A in *J Cell Biochem.* 2011

• expression of Notch-4 in a genetically modified mouse model (KCI) in Figure 1D in *PLoS One* 2011 to also

show the expression of Bcl-2 in the same mouse model in Figure 3A in the same paper
- expression of EZH2 in IC, KC, and KCI transgenic mice to also represent the expression of E-cadherin in the same mouse types in Figure 4B in *J Cell Physiol.* 2013

Respondent reused and relabeled one set of β-actin bands to represent loading controls for the following experiments showing:
- Inhibition of VEGF in Notch-1 siRNA transfected BxPC–3 cells in Figure 16B in the Dissertation
- inhibition of cyclin D_1 in genistein-treated BxPC–3 cells over time in Figure 7B in *Mol Cancer Ther.* 2006
- inhibition of Notch-1 in genistein-treated BxPC–3 cells over time in Figure 8A in *Mol Cancer Ther.* 2006
- down-regulation of MMP–9 expression in Notch-1 siRNA transfected BxPC–3 cells in Figure 17A (left) in the Dissertation and Figure 3B in *Cancer Res.* 2006a
- up-regulation by cDNA transfection and down regulation by Notch-1 siRNA transfection in BcPC–3 cells in Figure 4B in *Cancer Res.* 2006a
- down-regulation of MMP–9 in ICN-transfected BxPC–3 cells in Figure 15B in the Dissertation and Figure 5A in *Cancer Res.* 2006a
- inhibition of Notch-1, Hes-1, Cyclin D1, and Bcl-X_L protein expression after 72 hours of curcumin treatment in pancreatic cancer cells in Figure 3D in *Cancer* 2006
- down-regulation of Notch-1 expression by curcumin and Notch-1 siRNA in Notch-1 siRNA-transfected BxPC–3 cells in Figure 5A in *Cancer* 2006
- down-regulation of Notch-1 expression in Notch-1 siRNA-transfected BxPC–3 cells compared with control in Figure 5A in *Cancer Res.* 2006b
- inhibition of Hes-1, Cyclin D1 and Bcl-xL in genistein-treated BxPC–3 cells over time in Figure 20C in the Dissertation and Figure 3C in *Int J Cancer* 2006
- inhibition of Bcl-xL, Bcl-2, Cyclin D1, COX–2, Survivin and MMP–9 protein expression by Notch-1 siRNA in BxPC–3 cells in Figure 6A in *Int J Cancer* 2006
- inhibition of IKKα and pIκBα protein expression by Notch-1 siRNA in BxPC–3 cells in Figure 6B in *Int J Cancer* 2006

Respondent reused and relabeled a second set of β-actin bands to represent loading controls for the following experiments showing:
- Increasing inhibition of Notch-1 by 25 μmol/l genistein at 24, 48, and 72 hours in BxPC–3 cells in Figure 20A in the Dissertation, Figure 7B in *Mol Cancer Ther.* 2006, and Figure 3A in *Int J Cancer* 2006
- up-regulation of Notch-1 in Notch-1 cDNA transfected BxPC–3 cells, with or without 10 μmol/l curcumin, in Figure 6A in *Cancer* 2006

Respondent reused and relabeled a third set of β-actin bands to represent loading controls for the following experiments showing:
- The level of expression of seven known G_0-G_1 cell cycle regulatory factors in Figure 10 in the Dissertation and Figure 5 in *Mol Cancer Ther.* 2006
- overexpression of Notch-1 in Notch-1 cDNA transfected BxPC–3 cells in Figure 22A in the Dissertation and Figure 9A in *Mol Cancer Ther.* 2006
- inhibition of NF-κB target gene expression by Notch-1 siRNA in BxPC–3 cells in Figure 23A in the Dissertation
- inhibition of IKKα and pIκBα protein expression by Notch-1 siRNA in BxPC–3 pancreatic cancer cells in Figure 23B the Dissertation
- overexpression of Notch-1 in Notch-1 siRNA–transfected BxPC–3 cells in Figure 1C in *Cancer Res.* 2006a
- down-regulation of VEGF by siRNA transfection in ICN-transfected BxPC–3 cells in Figure 5A (right) in *Cancer Res.* 2006a
- up-regulation of Notch-1 in cDNA-transfected and cDNA and ERRP transfected BxPC–3 cells in Figure 5C in *Cancer Res.* 2006b
- inhibition of MMP-2, MMP-9, and uPAR genes by FoxM1 siRNA in BxPC–3, HPAC, and PANC–1 cells in Figure 5B in *Cancer Res.* 2007b

Respondent reused and relabeled a fourth set of β-actin bands to represent loading controls for the following experiments showing:
- FoxM1 expression in AsPC–1, BxPC–3, Colo-357, HPAC, L3.6pl, MIA PaCa and PANC–1 cells in Figure 1A in *Cancer Res.* 2007b
- PDGF–D expression in PDGF–D cDNA transfected BxPC–3, Colo-357, and MIA PaCa cells in Figure 2C in *Cancer Res.* 2007c
- Bcl-2 expression in AsPC–1, BxPC–3, Colo-357, HPAC, L3.6pl, MIA PaCa and PANC–1 cells in Figure 1C in *Int J Cancer* 2008

Respondent reused and relabeled a fifth set of β-actin bands to represent loading controls for the following experiments showing:
- Down regulation of PDFG–D expression by PDGF–D siRNA in BcPC–3, HPAC, and Colo-357 cells and up-regulation of PDGF–D expression by PDGF–D cDNA in BxPC–3, Colo-357, and MIA PaCa cells in Figure 2C in *Cancer Res.* 2007c
- inhibition of Notch-1 expression by PDGF–D siRNA in BxPC–3, HPAC, and Colo-357 cells in Figure 4A in *Cancer Res.* 2007c

Respondent reused and relabeled a sixth set of β-actin bands to represent loading controls for the following experiments showing:
- Up-regulation of Notch-1 expression by cDNA in BxPC–3, HPAC, and PANC–1 cells in Figure 6D (bottom) in the Dissertation and Figure 1D in *Mol Cancer Ther.* 2006
- down-regulation of Notch-1 expression by Notch-1 siRNA and genistein in BxPC–3 cells in Figure 21 in the Dissertation and Figure 4A in *Int J Cancer* 2006

Respondent reused and relabeled a seventh set of β-actin bands to represent loading controls for the following experiments showing:
- Down-regulation of Notch-1 expression by Notch-1 siRNA in BxPC–3, HPAC, and PANC–1 cells in Figure 6D (top) in the Dissertation and Figure 1D in *Mol Cancer Ther.* 2006
- expression of Notch-1, Hes-1, and Cyclin D1 after incubation with recombinant ERRP in BxPC–3, HPAC, and PANC–1 cells in Figure 2C in *Cancer Res.* 2006b
- effects of ERRP, Erbitux, or Herceptin followed by exposure to TGF-α or HB–EGF on Notch-1 expression in BxPC–3 cells in Figure 2D in *Cancer Res.* 2006b
- down-regulation of FoxM1 expression by FoxM1 siRNA in BxPC–3, HPAC, and PANC–1 cells in Figure 1D in *Cancer Res.* 2007b
- the level of expression of seven known G_0-G_1 cell cycle regulatory factors (Survivin, cdc25A, p27, p21, Cyclin D1, Cyclin B, and CDK2) in Figure 4C in *Cancer Res.* 2007b

Respondent reused and relabeled:
- Invasion assay results showing a high level of penetration of Notch-1 cDNA-transfected cells through a Matrigel matrix in Figure 1D in *Cancer Res.* 2006a, to also represent control siRNA-transfected cells (controls) not transfected with MMP–9 or VEGF siRNA in Figure 5B in *Cancer Res.* 2006a
- sections from one image of an invasion assay to show a lower level of penetration of C4–2B cells through a Matrigel matrix after treatment with 10 μmol/L of B–DIM than in the control condition (DMSO) in Figure 6B in *Cancer Res.* 2007a

- sections from one image to show the penetration of both control and ERRP-treated HPAC cells through a Matrigel matrix in Figure 4 in *Cancer Res.* 2006b
- one image to show the penetration of ERRP-treated PANC–1 cells through a Matrigel matrix in Figure 4 in *Cancer Res.* 2006b to also show the penetration of TW–37 treated Colo-357 cells in Figure 5b in *Int J Cancer* 2008
- images of assays of endothelial tube formation after HUVACs were trypsinized and seeded with control siRNA transfected BxPC–3 or HPAC cells in Figure 6c in *Cancer Res.* 2007b
- a single gel shift band showing the no treatment control condition (CS) in an EMSA assay using BxPC–3 cells showing down regulation of NF-κB DNA binding by Notch-1 siRNA in Figures 11A and 14A in the Dissertation to also show:

—The control conditions (CP) in assays showing activation of NF-κB binding activity by Notch-1 plasmid (cDNA) transfection in Figures 11A and 14A in the Dissertation

—inhibition of NF-κB DNA binding activity after treatment with 25 μM genistein for 48 hours in Figure 19B in the Dissertation

- a single gel shift band showing the effect of Notch-1 siRNA transfection of BxPC–3 cells, showing inhibition of NF-κB DNA binding activity in Figures 11A and 14A in the Dissertation to also show NF-κB binding activity in BxPC–3 cells after treatment with 25 μM genistein in Figure 22C in the Dissertation
- a single gel shift band showing the effect of Notch-1 cDNA transfection of BxPC–3 cells, showing activation of NF-κB DNA binding activity in Figures 11A and 14A in the Dissertation to also show NF-κB binding activity in BxPC–3 cells in the no treatment control condition in an experiment showing the effect of genistein on binding in Figure 22C in the Dissertation
- a single gel shift band showing the no treatment control condition in an EMSA assay using HPAC cells showing down regulation of NF-κB DNA binding by Notch-1 siRNA in Figure 11A in the Dissertation to also show the no treatment control condition in the activation of NF-κB DNA binding after transfection with Notch-1 cDNA
- a single gel shift band showing the effect of 0 μM genistein on NF-κB binding activity in BxPC3 cells in Figure 19A the Dissertation to also show the effect of:

—25 μM of genistein for 0 hours in HPAC cells in Figure 19B in the Dissertation

—Notch-1 cDNA on NF-κB binding activity in Figure 22C in the Dissertation

- a single gel shift band showing the effect of 10 μM genistein on NF-κB binding activity in BxPC3 cells in Figure 19A in the Dissertation to also show the effect of:

—25 μM genistein for 24 hours in HPAC cells in Figure 19B in the Dissertation

—Notch-1 cDNA plus 25 μM genistein on NF-κB binding activity in Figure 22C in the Dissertation

- a single gel shift band showing the effect of Bcl-2 siRNA transfection of Colo-357 cells showing down-regulation of NF-κB DNA binding activity to also show the same effect with 500 nM TW–37 on Colo-357 cells in Figure 3a in *Int J Cancer* 2008

Respondent reused and relabeled images representing the retinoblastoma control protein (Rb) levels from one EMSA in multiple figures. Respondent used the same loading controls assay blots, in different orders with some flipped horizontally, showing:

- Down-regulation of Notch-1 gene expression by Notch-1 siRNA in siRNA- and cDNA-transfected BxPC–3, HPAC, and PANC–1 cells in Figure 11 in the Dissertation and Figure 6 in *Mol Cancer Ther.* 2006
- down-regulation of Notch-1 by genistein in BxPC–3 cells in Figure 7E in *Mol Cancer Ther.* 2006
- Notch-1 induced NF-κB DNA binding in Figure 14 in the Dissertation and Figure 2 in *Cancer Res.* 2006a
- down-regulation of Notch-1 by curcumin in BxPC–3 and PANC–1 cells in Figures 4, 5D, and 6D in *Cancer* 2006
- inhibition of NF-κB activation in three types of pancreatic cancer cells (BxPC–3, HPAC, PANC–1) in Figure 3A in *Cancer Res.* 2006b
- inhibition of NF-κB DNA binding activity by genistein (by dose and time) in Figure 19 in the Dissertation and Figure 2 in *Int J Cancer* 2006
- inhibition of NF-κB DNA-binding activity by Notch-1 siRNA in BxPC–3 pancreatic cancer cells in Figure 22 in the Dissertation and Figure 5 in *Int J Cancer* 2006
- decreased NF-κB DNA-binding activity through down-regulation of PDGF–D by siRNA transfection in BxPC–3, HPAC, and Colo-357 pancreatic cancer cells, activation of NF-κB DNA binding activity in BxPC3, Colo-357, and MIA PaCa pancreatic cancer cells in Figure 5A in *Cancer Research* 2007c
- differences in NF-κB activation in a panel of pancreatic cancer cell lines (AsPC–1, BxPC–3, Colo-357, HPAC, L3.6pl, MIA PaCa, PANC–1 in Figure 1d in *Int J Cancer* 2008
- inhibition of NF-κB activation by Bcl-2 siRNA in Colo-357 cells and by TW–37 (by dose and time) in Colo-357 and BXPC–3 pancreatic cancer cells in Figure 3a in *Int J Cancer* 2008
- inhibition of NF-κB activation by TW–37 in Colo-357 tumor xenografts from SCID mice in Figure 6c in *Int J Cancer* 2008

In addition, Respondent used these same images to represent β-actin in a figure showing that FoxM1 protein levels were up-regulated by FoxM1 cDNA plasmid in AsPC–1, PANC–1, and Colo-357 cells in Figure 1D in *Cancer Res.* 2007b.

Respondent reused and relabeled one image to represent multiple supershift assays done at different times for different experiments to show the effect of anti-NF-κB p65 antibody on NF-κB DNA-binding activity in:

- Figure 2B in *Cancer Res* 2006a
- Figure 5A in *Cancer Res.* 2007c

Respondent reused and relabeled a second image to represent multiple supershift assays done at different times for different experiments to show the effect of anti-NF-κB p65 antibody on NF-κB DNA-binding activity in:

- Figure 6D in *Mol Cancer Ther.* 2006
- Figure 4C in *Cancer* 2006
- Figure 3A in *Cancer Res.* 2006b
- Figure 2C in *Int J Cancer* 2006
- Figure 1d in *Int J Cancer* 2008

Respondent reused and relabeled the Rb levels in multiple supershift assay figures representing different experiments done at different times. Respondent used the same loading control assay blots in the supershift assays that came from the EMSAs to show the effect of anti-NF-κB p65 antibody on NF-κB DNA-binding activity in:

- Figure 6D in *Mol Cancer Ther.* 2006
- Figure 2B in *Cancer Res.* 2006a
- Figure 4C in *Cancer* 2006
- Figure 3A (right) in *Cancer Res.* 2006b
- Figure 2C in *Int J Cancer* 2006
- Figure 5A (right) in *Cancer Res* 2007c
- Figure 1d (right) in *Int J Cancer* 2008

The institution revoked the Respondent's Ph.D. degree and procured retractions or errata for all of the affected papers except *Mol Cancer Ther.* 2008.

Dr. Wang entered into a Voluntary Exclusion Agreement (Agreement) and agreed to the following:

(1) Respondent agreed to exclude himself voluntarily for a period of ten (10) years beginning on July 21, 2020, from any contracting or subcontracting with any agency of the United States Government and from eligibility for or involvement in nonprocurement programs of the United States Government referred to as ''covered transactions'' pursuant to HHS's Implementation (2 CFR part 376) of OMB Guidelines to Agencies on Governmentwide Debarment and Suspension, 2 CFR part 180 (collectively the ''Debarment Regulations'');

(2) Respondent agreed to exclude himself voluntarily 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 for a period of ten (10) years, beginning on July 21, 2020; and

(3) as a condition of the Agreement, Respondent will request that the following paper be corrected or retracted in accordance with 42 CFR 93.407(a)(1):

- *Mol. Cancer Ther.* 2008 Feb;7(2):341–9

Dated: August 7, 2020.

Elisabeth A. Handley,

Director, Office of Research Integrity, Office of the Assistant Secretary for Health.

[FR Doc. 2020–17602 Filed 8–11–20; 8:45 am]

BILLING CODE 4150–31–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Institute on Aging; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended, notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: National Institute on Aging Special Emphasis Panel; AD Analysis.

Date: September 4, 2020.

Time: 2:00 p.m. to 5:00 p.m.

Agenda: To review and evaluate grant applications.

Place: National Institute on Aging, Gateway Building, 7201 Wisconsin Avenue, Bethesda, MD 20892 (Video Meeting).

Contact Person: Nijaguna Prasad, Ph.D., Scientific Review Officer, Scientific Review Branch, National Institute on Aging, National Institutes of Health, 7201 Wisconsin Avenue, Gateway Building, Suite 2W200, Bethesda, MD 20892, (301) 496–9667, *nijaguna.prasad@nih.gov*.

(Catalogue of Federal Domestic Assistance Program Nos. 93.866, Aging Research, National Institutes of Health, HHS)

Dated: August 6, 2020.

Miguelina Perez,

Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2020–17586 Filed 8–11–20; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Fogarty International Center; Notice of Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended, notice is hereby given of a meeting of the Fogarty International Center Advisory Board.

The meeting will be open to the public, with attendance limited to space available. Individuals who plan to attend and need special assistance, such as sign language interpretation or other reasonable accommodations, should notify the Contact Person listed below in advance of the meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute clearly unwarranted invasion of personal privacy.

Name of Committee: Fogarty International Center Advisory Board.

Date: September 10–11, 2020.

Closed: September 10, 2020, 12:00 p.m. to 3:00 p.m.

Agenda: To review and evaluate grant applications.

Place: Fogarty International Center, National Institutes of Health, 31 Center Drive, Bethesda, MD 20892 (Virtual Meeting).

Open: September 11, 2020, 12:00 p.m. to 3:00 p.m.

Agenda: Update and discussion of current and planned FIC activities.

Place: Fogarty International Center, National Institutes of Health, 31 Center Drive, Bethesda, MD 20892 (Virtual Meeting).

Meeting Access: https://www.fic.nih.gov/About/Advisory/Pages/default.aspx.

Contact Person: Kristen Weymouth, Executive Secretary, Fogarty International Center, National Institutes of Health, 31 Center Drive, Room B2C02, Bethesda, MD 20892–7952, (301) 496–1415, *kristen.weymouth@nih.gov*.

Any interested person may file written comments with the committee by forwarding the statement to the Contact Person listed on this notice. The statement should include the name, address, telephone number and when applicable, the business or professional affiliation of the interested person.

Information is also available on the Institute's/Center's home page: *http://www.fic.nih.gov/About/Advisory/Pages/default.aspx* where an agenda and additional information for the meeting will be posted when available.

(Catalogue of Federal Domestic Assistance Program Nos. 93.106, Minority International Research Training Grant in the Biomedical and Behavioral Sciences; 93.154, Special International Postdoctoral Research Program in Acquired Immunodeficiency Syndrome; 93.168, International Cooperative Biodiversity Groups Program; 93.934, Fogarty International Research Collaboration Award; 93.989, Senior International Fellowship Awards Program, National Institutes of Health, HHS)

Dated: August 6, 2020.

Miguelina Perez,

Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2020–17589 Filed 8–11–20; 8:45 am]

BILLING CODE 4140–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Human Genome Research Institute; Notice of Closed Meeting

Pursuant to section 10(d) of the Federal Advisory Committee Act, as amended, notice is hereby given of the following meeting.

The meeting will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: Center for Inherited Disease Research Access Committee Grant Review.

Date: September 11, 2020.

Time: 11:30 a.m. to 2:30 p.m.

Agenda: To review and evaluate grant applications.

Place: National Human Genome Research Institute, National Institutes of Health, 6700B Rockledge Drive, Room 3185, Bethesda, MD 20892 (Telephone Conference Call).

Contact Person: Barbara J. Thomas, Ph.D., Scientific Review Officer, Scientific Review Branch, National Human Genome Research