

Findings of Research Misconduct

Notice Number:

NOT-OD-20-165

Key Dates

Release Date:

August 17, 2020

Related Announcements

None

Issued by

Office of The Director, National Institutes of Health ([OD](#))

Purpose

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.

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- κ B 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- κ B 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- κ B 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- κ B 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 kappaB 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-kappaB, 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 cDNAtransfection 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-XL (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-XL (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-XL 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-XL, 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 D1 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-XL 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 IKK α and pIkB α 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 μ mol/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 μ mol/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 G₀-G₁ 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- κ B target gene expression by Notch-1 siRNA in BxPC-3 cells in Figure 23A in the Dissertation
- inhibition of IKK α and pIkB α 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 PDGF-D expression by PDGF-D siRNA in BxPC-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 G₀-G¹ 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 $\mu\text{mol/L}$ 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- κ B 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- κ B binding activity by Notch-1 plasmid (cDNA) transfection in Figures 11A and 14A in the Dissertation
- inhibition of NF- κ B 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- κ B DNA binding activity in Figures 11A and 14A in the Dissertation to also show NF- κ B 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- κ B DNA binding activity in Figures 11A and 14A in the Dissertation to also show NF- κ B 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- κ B 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- κ B DNA binding after transfection with Notch-1 cDNA
- a single gel shift band showing the effect of 0 μM genistein on NF- κ B 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- κ B binding activity in Figure 22C in the Dissertation
- a single gel shift band showing the effect of 10 μM genistein on NF- κ B 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- κ B 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- κ B 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- κ B 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- κ B activation in three types of pancreatic cancer cells (BxPC-3, HPAC, PANC-1) in Figure 3A in *Cancer Res.* 2006b
- inhibition of NF- κ B 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- κ B 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-[kappa]B 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-[kappa]B DNA binding activity in BxPC3, Colo-357, and MIA PaCa pancreatic cancer cells in Figure 5A in *Cancer Research* 2007c
- differences in NF- κ B 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- κ B 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- κ B 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-[kappa]B p65 antibody on NF- κ B 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- κ B p65 antibody on NF- κ B 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- κ B p65 antibody on NF- κ B 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

Inquiries

Please direct all inquiries to:

Elisabeth A. Handley
Office of Research Integrity
Telephone: 240-453-8200

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