TABLE 2—ESTIMATED ANNUAL RECORDKEEPING BURDEN [1] [2]—Continued

21 CFR section; activity	Number of recordkeepers	Number of records per recordkeeper	Total annual records	Average burden per recordkeeping	Total hours
§ 1.1138; laboratories—maintaining ISO/IEC 17025: 2017 accreditation. § 1.1154; laboratories—additional record-keeping requirements; a LAAF-accredited laboratory must maintain, for 5 years after the date of creation, records created and received while it is LAAF-accredited that relate to compliance with this subpart.	160	2	320	450.765 (450 hours and 46 minutes).	144,245
Total			345		145,241

[1] There are no capital costs or operating and maintenance costs associated with this collection of information.
[2] Totals may not sum due to rounding.

The burden we attribute to reporting and recordkeeping activities is assumed to be distributed among the individual elements of the respective information collection activities. Although we have not received a notice of intent to relinquish records since the last approval of this information collection, we include one response for the purpose of estimating burden.

New information technology applications have more accurately calculated the number of food testing laboratories seeking accreditation and as a result the number of respondents to the information collection decreased (from 170 respondents in the currently approved collection to 160 respondents). Consequently, we have adjusted our burden estimate, which results in a decrease of 227 responses and 9,303 burden hours from the currently approved information collection.

Dated: August 12, 2024.

Lauren K. Roth,

Associate Commissioner for Policy.

[FR Doc. 2024–18277 Filed 8–14–24; 8:45 am]

BILLING CODE 4164–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Office of the Secretary

Findings of Research Misconduct

AGENCY: Office of the Secretary, HHS.

ACTION: Notice.

SUMMARY: Findings of research misconduct have been made against Richard L. Eckert, Ph.D. (Respondent), who was a Professor, Chair of the Department of Biochemistry and Molecular Biology, and Deputy Director of the University of Maryland and Stewart Greenebaum Comprehensive Cancer Center, University of Maryland, Baltimore (UMB). Respondent 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 R01 CA211909, R01 CA184027, R01 CA131074, R01 CA131064, R01 CA092201, R01 CA109196, P30 CA134274, and P30 CA043703, National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), NIH, grants R21 AR065266, R01 AR046494, R01 AR053851, R01 AR060388, P30 AR039750, R01 AR041456, R01 AR049713, and R01 AR045357, National Eye Institute (NEI), NIH, grants P30 EY011373 and T32 EY007157, and National Institute of General Medical Sciences (NIGMS), NIH, grant R01 GM043751. The questioned research was included in two (2) grant applications submitted for PHS funds, specifically R01 CA233450–01 and R01 CA233450–01A1 submitted to NCI, NIH. The administrative actions, including debarment for a period of eight (8) years, were implemented beginning on August 1, 2024, and are detailed below.

FOR FURTHER INFORMATION CONTACT: Sheila Garrity, JD, MPH, MBA, 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:

Richard L. Eckert, Ph.D., University of Maryland, Baltimore (UMB): Based on the report of an investigation conducted by UMB and additional analysis conducted by ORI in its oversight review, ORI found that Dr. Richard L. Eckert (Respondent), former Professor, Chair of the Department of Biochemistry and Molecular Biology, and Deputy Director of the University of Maryland and Stewart Greenebaum Comprehensive Cancer Center, UMB, engaged in research misconduct in research supported by PHS funds, specifically NCI, NIH, grants R01 CA211909, R01 CA184027, R01 CA131074, R01 CA131064, R01 CA092201, R01 CA109196, P30 CA134274, and P30 CA043703, NIAMS, NIH, grants R21 AR065266, R01 AR046494, R01 AR053851, R01 AR060388, P30 AR039750, R01 AR041456, R01 AR049713, and R01 AR045357, NEI, NIH, grants P30 EY011373 and T32 EY007157, and NIGMS, NIH, grant R01 GM043751. The questioned research was included in two (2) grant applications submitted for PHS funds, specifically R01 CA233450–01 and R01 CA233450–01A1 submitted to NCI, NIH.

ORI found that Respondent engaged in research misconduct by intentionally, knowingly, or recklessly falsifying and/or fabricating data in the following thirteen (13) published papers and two (2) PHS grant applications:

- Inhibition of YAP function overcomes BRAF inhibitor resistance in melanoma cancer stem cells. *Oncotarget.* 2017 Nov 22; 8(66):110257–110272. doi: 10.18632/oncotarget.22628 (hereafter referred to as ''*Oncotarget* 2017'').
- The Bmi-1 helix-turn and ring finger domains are required for Bmi-1 antagonism of (-) epigallocatechin-3-gallate suppression of skin cancer cell survival. *Cell Signal.* 2015 Jul;27(7):1336–44. doi: 10.1016/j.cellsig.2015.03.021 (hereafter referred to as ''*Cell Signal* 2015''). Erratum in: *Cell Signal.* 2021 Jun;82:109952. doi: 10.1016/j.cellsig.2021.109952.
- P38δ regulates p53 to control p21Cip1 expression in human epidermal keratinocytes. *J Biol Chem.* 2014 Apr 18; 289(16):11443–11453. doi: 10.1074/jbc.M113.543165 (hereafter referred to as ''*J Biol Chem.* 2014'').

• Methylosome protein 50 and PKCδ/p38δ protein signaling control keratinocyte proliferation via opposing effects on p21Cip1 gene expression. *J Biol Chem.* 2015 May 22;290(21):13521–30. doi: 10.1074/jbc.M115.642868 (hereafter referred to as ''*J Biol Chem.* 2015'').

• Transamidase site-targeted agents alter the conformation of the transglutaminase cancer stem cell survival protein to reduce GTP binding activity and cancer stem cell survival. *Oncogene.* 2017 May 25;36(21):2981–2990. doi: 10.1038/onc.2016.452 (hereafter referred to as ''*Oncogene* 2017''). Erratum in: *Oncogene.* 2021 Apr;40(13):2479–2481. doi: 10.1038/s41388–021–01709–5.

• Suppression of AP1 transcription factor function in keratinocyte suppresses differentiation. *PLoS One.* 2012;7(5):e36941. doi: 10.1371/journal.pone.0036941 (hereafter referred to as ''*PLoS One* 2012''). Retraction in: *PLoS One.* 2021 Feb 11;16(2):e0247222. doi: 10.1371/journal.pone.0247222.

• Suppressing AP1 factor signaling in the suprabasal epidermis produces a keratoderma phenotype. *J Invest Dermatol.* 2015 Jan;135(1):170–180. doi: 10.1038/jid.2014.310 (hereafter referred to as ''*J Invest Dermatol.* 2015''). Erratum in: *J Invest Dermatol.* 2021 Jul; 141(7):1862. doi: 10.1016/j.jid.2021.05.008.

• Protein kinase C (PKC) delta suppresses keratinocyte proliferation by increasing p21(Cip1) level by a KLF4 transcription factor-dependent mechanism. *J Biol Chem.* 2011 Aug 19; 286(33):28772–28782. doi: 10.1074/jbc.M110.205245 (hereafter referred to as ''*J Biol Chem.* 2011'').

• The Bmi-1 polycomb protein antagonizes the (-)-epigallocatechin-3-gallate-dependent suppression of skin cancer cell survival. *Carcinogene*sis. 2010 Mar;31(3):496–503. doi: 10.1093/carcin/bgp314 (hereafter referred to as ''*Carcinogenesis* 2010'').

• PKC-delta and –eta, MEKK–1, MEK–6, MEK–3, and p38-delta are essential mediators of the response of normal human epidermal keratinocytes to differentiating agents. *J Invest Dermatol.* 2010 Aug;130(8):2017–30. doi: 10.1038/jid.2010.108 (hereafter referred to as ''*J Invest Dermatol.* 2010'').

• Sulforaphane suppresses PRMT5/MEP50 function in epidermal squamous cell carcinoma leading to reduced tumor formation. *Carcinogenesis.* 2017 Aug 1;38(8):827–836. doi: 10.1093/carcin/bgx044 (hereafter referred to as ''*Carcinogenesis* 2017''). Erratum in: *Carcinogenesis.* 2023 Oct 20;44(7):626–627. doi: 10.1093/carcin/bgad044.

• Localization of the TIG3 transglutaminase interaction domain and demonstration that the amino-terminal region is required for TIG3 function as a keratinocyte differentiation regulator. *J Invest Dermatol.* 2008 Mar;128(3):517–29. doi: 10.1038/sj.jid.5701035 (hereafter referred to as ''*J Invest Dermatol.* 2008'').

• Transglutaminase interaction with α6/β4-integrin stimulates YAP1-Dependent ΔNp63α stabilization and leads to enhanced cancer stem cell survival and tumor formation. *Cancer Res.* 2016 Dec 15;76(24):7265–7276. doi: 10.1158/0008–5472.CAN–16–2032 (hereafter referred to as ''*Cancer Res.* 2016'').

• R01 CA233450–01, ''Sulforaphane suppression of PRMT5 epigenetics to reduce cancer stem cell survival,'' submitted to NCI, NIH, on 01/26/2018, administratively withdrawn by NCI on 07/01/2020

• R01 CA233450–01A1, ''Sulforaphane suppression of PRMT5 epigenetics to reduce cancer stem cell survival,'' submitted to NCI, NIH, on 10/30/2018, administratively withdrawn by NCI on 03/01/2021

Specifically, ORI found that Respondent intentionally, knowingly, or recklessly falsified and/or fabricated Western blot image data and microscopy image data by:

• using images representing unrelated experiments, with or without manipulating them, and falsely relabeling them as data representing different proteins and/or experimental results as follows:

—In Figure 3F of *Oncotarget* 2017, the bands in rows 4 and 7 of the A375–PLX–R right-side panel, representing expression of TAZ-*P* (row 4) and ERK1/2 (row 7), are falsified and/or fabricated by using unrelated bands from a source image representing different proteins in an unrelated experiment

—In Figure 2B of *J Biol Chem.* 2014, the bands in row 2 in the top panel, representing MEK3 expression in normal human keratinocytes (KERn) infected with Ad5–EV, Ad5–MEK3, and Ad5–PKCδ (from left to right), are falsified and/or fabricated by compiling unrelated bands from a source image representing p44 expression in an unrelated experiment

—In Figure 2B of *J Biol Chem.* 2014, the bands in row 3 in the top panel, representing p38δ expression in KERn infected with Ad5–EV, Ad5–MEK3, and Ad5–PKCδ (from left to right), are falsified and/or fabricated by compiling unrelated bands from a source image representing β-actin expression in an unrelated experiment

—In Figure 1B of *J Biol Chem.* 2015, the bands in rows 1–3 in the upper panel, representing expression of MEP50 (row 1), FLAG (row 2), and β-actin (row 3), are falsified and/or fabricated by compiling different bands from source images representing expression of different proteins in unrelated experiments

—In Figure 7C of *J Biol Chem.* 2011, the bands in row 2 in the right panel, representing $p21^{Cip1}$ expression under treatments of Control-siRNA or hKLF4-siRNA, are falsified and/or fabricated by using unrelated bands from a source image representing p21 expression in cells treated with Ad5–EV or Ad5–PKCd

—In Figure 1B of *PLoS One* 2012, the bands in row 1, representing TAM67–FLAG expression, are falsified and/or fabricated by using unrelated bands from a source image representing CyclinA expression

—In Figure 2C of *PLoS One* 2012, the bands in rows 3 and 4, representing negative expression of junB (row 3) and junD (row 4), are falsified and/or fabricated by using blank areas that were far from the target molecular weight in a source image

—In Figure 6a of *J Invest Dermatol.* 2015, the bands 1–4 in the bottom row, representing β-Actin expression under treatments of Loricrin, TAM67-rTA, and/or Dox, are falsified and/or fabricated by:

—➢ using 3 bands from a source image representing β-actin expression in an unrelated experiment for bands 1–3

—➢ duplicating band 3 to create band 4

—In Figure 1B of *Carcinogenesis* 2010, the bands in rows 1, 2, and 5 in the left panel, representing expression of Ezh2 (row 1), H3 K27–3M (row 2), and β-actin (row 5) in two different cell types treated with 60 μM EGCG, are falsified and/or fabricated by using unrelated bands from a source image representing expression of the same proteins under an unrelated experiment

—In *Carcinogenesis* 2010, the bands in row 3 in the right panel of Figure 1B and the bands 1–5 in row 3 in the upper panel of Figure 2A are falsified and/or fabricated by using unrelated bands from a source image. Specifically:

—➢ the bands 1–4 in the upper panel of Figure 2A, representing Ezh2 expression treated with 0, 10, 20, and 40 μM EGCG are used from the bands representing the same protein

but treated with different doses of EGCG in the source image

—➢ the bands 1 and 5 in the upper panel of Figure 2A, representing Ezh2 expression, are reused and relabeled in the bands in Figure 1B, row 3 in the right panel to represent Suz12 expression

—In Figure 4A of *Carcinogenesis* 2010, the bands in rows 6 and 7, representing expression of cyclin E (row 6) and cyclin A (row 7) in cells treated with 60 μm EGCG plus other reagents, are falsified and/or fabricated by reusing and relabeling the bands from a source image representing cyclin E expression in cells treated with 150 μm EGCG plus other reagents

—In Figure 7a of *J Invest Dermatol.* 2008:

—➢ bands 1 and 5 (including the empty lanes) in the COX4 panel, representing expression of COX4 treated with EV (band 1) and TIG3 1–134 (band 5), are falsified and/or fabricated by reusing a band labeled as TGI C377 sample 3 from the primary data

—➢ band 8 (including the empty lanes) in the Cytochrome c panel, representing expression of Cytochrome c treated with TIG3 124–164, is falsified and/or fabricated by using an unrelated band from unknown source

• reusing the same source images, with or without manipulating them to conceal their similarities, and falsely relabeling them as data representing different proteins or experimental results as follows:

—In Figure 2 of *Cell Signal* 2015, two control samples in the bottom panel, representing cells in tAd5–FLAG-hBmiΔRF condition (left) and tAd5–FLAG-hBmi-1ΔHT condition (right), are reused from different fields of a same source image

—In *J Biol Chem.* 2014, Figure 2B, bands 2 and 3 in row 1 of 3rd panel, representing ATF2-*P* expression, and Figure 6C, bands 1 and 2 in row 2 of the 3rd panel, representing p38α expression, are identical

—In *J Biol Chem.* 2014, Figure 2C, bands 1 and 3 in row 3 of the upper panel, representing MEK3 expression, and Figure 6C, bands 1 and 2 in row 2 of the top panel, representing p38α expression, are identical

—In Figure 3C of *Oncogene* 2017, band 9, representing TG2 expression treated with total CP4d, is falsified and/or fabricated by reusing and relabeling band 3, representing TG2 expression treated with NC9 (total) in the same figure

—In *Carcinogenesis* 2010, Figure 3C, the bands in row 2, representing β-actin expression, and Figure 4C, the bands in row 3, representing procaspase 9 expression, are identical

—In Figure 7b of *J Invest Dermatol.* 2010, the bands in the upper panel, representing expression of MEKK1 and its β-Actin control, are falsified and/or fabricated by reusing and relabeling the bands in the middle panel, representing expression of MEK6 and its

—β-Actin control in the same figure

—In Figure 1D of *Carcinogenesis* 2017, Figure 5B of R01 CA233450–01 and Figure 3B of R01 CA233450–01A1, the bands in rows 3 in both the upper and bottom panels, representing H4 expression, are falsified and/or fabricated by reusing and relabeling the same source images that are used for the bands in row 2 in Figure 3J of *Carcinogenesis* 2017, representing PRMT5 expression

—In Figure 1c of *J Invest Dermatol.* 2008, the background area between molecular weight 20–45 in the TIG3 (41–164) lanes of the right panel is falsified and/or fabricated by reusing and relabeling the background area of TIG3 WT group with flipping

—In Figure 1c of *J Invest Dermatol.* 2008, the bands in lanes 7–8 of the left panel, representing expression of TIG3 monomer under TIG3 (100–164) condition, are falsified and/or fabricated by reusing and relabeling the bands in lanes 9–10 of the left panel, representing expression of TIG3 monomer under TIG3 (41–164) condition

—In *Cancer Res.* 2016, bands 2–3 in the bottom row in Figure 3C, representing β-actin expression treated with Integrin α6-siRNA (band 2) and Integrin β4-siRNA (band 3), and bands 1–2 in the bottom row in Figure 3D, representing β-actin expression treated with Control-siRNA (band 1) and FAK-siRNA (band 2), are identical

• manipulating the data to exclude the band from a source image to falsely show a favorable result in Figure 2C of *PLoS One* 2012 by erasing the band in the left lane of the top row to falsely represent a lack of TAM67–FLAG expression

Respondent entered into a Voluntary Exclusion Agreement (Agreement) and voluntarily agreed to the following:

(1) Respondent will exclude himself voluntarily for a period of eight (8) years beginning on August 1, 2024 (the ''Exclusion Period'') from any contracting or subcontracting with any agency of the United States Government and from eligibility for or involvement in nonprocurement or procurement transactions referred to as ''covered transactions'' in 2 CFR parts 180 and 376 (collectively the ''Debarment Regulations'').

(2) During the Exclusion Period, Respondent will not apply for, permit his name to be used on an application for, receive, or be supported by funds of the United States Government and its agencies made available through contracts, subcontracts, or covered transactions.

(3) During the Exclusion Period, Respondent will exclude himself voluntarily from serving in any advisory or consultant capacity to PHS including, but not limited to, service on any PHS advisory committee, board, and/or peer review committee.

(4) Respondent will request that the following papers be corrected or retracted:

• *Oncotarget* 2017 Nov 22;8(66):110257–110272. doi: 10.18632/oncotarget.22628.

• *J Biol Chem.* 2014 Apr 18;289(16):11443–11453. doi: 10.1074/jbc.M113.543165.

• *J Biol Chem.* 2015 May 22;290(21):13521–30. doi: 10.1074/jbc.M115.642868.

• *J Biol Chem.* 2011 Aug 19;286(33):28772–28782. doi: 10.1074/jbc.M110.205245.

• *Carcinogenesis* 2010 Mar;31(3):496–503. doi: 10.1093/carcin/bgp314.

• *J Invest Dermatol.* 2008 Mar; 128(3):517–29. doi: l 0.1038/sj.jid.5701035.

• *J Invest Dermatol.* 2010 Aug;130(8):2017–30. doi: 10.1038/jid.2010.108.

• *Cancer Res.* 2016 Dec 15;76(24):7265–7276. doi: 10.1158/0008–5472.CAN–16–2032.

Respondent will copy ORI and the Research Integrity Officer at UMB on the correspondence with the journals.

Dated: August 12, 2024.

Sheila Garrity,

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

[FR Doc. 2024–18289 Filed 8–14–24; 8:45 am]

BILLING CODE 4150–31–P