Moerschbaecher, Joseph M.

From: Kaye, Alan D.
Sent: Tuesday, October 6, 2020 12:22 PM
To: Moerschbaecher, Joseph M.
Subject: Hari Koul..

Dear Joe,

There are various opinions about him. He was known here to be "arrogant" and "condescending" at times. Dr. Hari Koul was at U of Colorado School of Medicine. 6 years ago. He applied to be the chair of Biochemistry. He was funded in prostate cancer with multiple grants and was given a super chair at the Cancer Center. When he arrived, there was suspicion that he had data re-publication and it caught national attention on websites like "pubpeers" and "retraction watch". It resulted in an investigation at the medical school. Previous chairs of Biochemistry and others were very upset and long story short, it appears it was a postdoc that was involved who went back to India. It turns out there were similar allegations at U of Colorado and no investigation was ever opened there. LSUHSC and Colorado decided to retract a number of publications and they were actually never retracted to this day.

He obtained a lawyer and blamed the institution for trying to harm/malign him. At the end of the day, he did not keep his grants or research productivity over four years. LSUHSCS decided he was not meeting the standards of a Board of Regents chair and it was removed because he did not meet the criteria. He maintained an associate director capacity and tried to get his grants back. He heard about the opportunities in New Orleans and got his RO-1 back and he dropped the litigation against LSU Shreveport.

He is a "hard worker" and is "smart" per the people here. As for the data fabrication or falsification of data, he was never denied applying for a federal grant. To this day, they never found the postdoc who returned to India.

Alan

Dr. Alan David Kaye, MD, PhD, DABA, DABPM, DABIPP, FASA
Vice Chancellor of Academic Affairs, Chief Academic Officer, and Provost
Professor, Department of Anesthesiology and Pharmacology, Toxicology, and Neurosciences
Pain Fellowship Program Director, Louisiana State University School of Medicine
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Joseph M. Moerschbaecher, PhD
Vice Chancellor for Academic Affairs
Dean School of Graduate Studies
Professor of Pharmacology
LSU Health Sciences Center
1901 Perdido Street P7-1
New Orleans, LA 70112

70112-139301

June 15, 2020

Joseph M. Moerschbaecher, PhD
Vice Chancellor for Academic Affairs
Dean School of Graduate Studies
Professor of Pharmacology
LSU Health Sciences Center
1901 Perdido Street P7-1
New Orleans, LA 70112

RE: Dr. Hari K. Koul, Professor and VA Scientist

Louisiana State University Health Sciences Center, New Orleans

Dear Dr. Moerschbaecher,

We are writing this letter to draw your kind attention regarding the grant and research activities of Dr. Hari Koul, who has moved to New Orleans (LSUHSC, New Orleans and Southeast Louisiana Veteran Health Care System) from LUHSC Shreveport and Overton Brooks VA Medical Center, Shreveport. He is the Associate Director of the Stanley S. Scott Cancer center, LSUHSC, 1700 Tulane Ave, New Orleans, LA 70112.

1. Dr. Koul has obtain two federal grants (please see the abstracts below) from the US Veteran Affairs (5I01BX001258) and the National Institutes of Health (R01CA161880) for the same project. We have obtained the application under the freedom of information act. Both the proposals are same. Under the federal guidelines no one can obtain the funding for the same project from two different federal agencies. He was fired as a Chair of the Department of Biochemistry, LSHHSC Shreveport, and now joined LSUHSC New Orleans. LSUHSC must return the funds to the NIH and debar Dr. Koul for maximum allowable time. Before accepting the grant, he signed a legal document that there was no overlap between the two projects. It should be considered scam, waste, fraudulent and money laundering activities as per the federal guidelines. False claim, False statement, and embezzlement are punishable under federal law.

   18 U.S.C. § 1956: Monetary Transactions
   18 U.S.C. § 1957: Monetary Transactions
2. He has obtained a new NIH RO1 grant entitled "Prostate Cancer Health Disparity: Role of PDEF", No. 1R01CA242839-01. This grant is based on the research papers and subjects which have been retracted recently. It looks like NIH program directors are not paying any attention.

3. He has been awarded a new VA merit award in spite of fraudulent activities and several retractions. What came out from the previously funded VA merit ward was fraudulent science and retracted papers. Now he has been awarded another VA merit award. Someone at the VA must investigate this immediately.
4. Several investigators who have been involved in fraudulent activities have been debarred and/or terminated from their jobs. Since these are the federal funds, US citizens have rights to demand fairness and equal penalties to everyone involved. US public must make sure that the grant funds are used for the right cause and utilized for the proper treatment of the disease.

   We hope that you please investigate this serious matter and take appropriate actions as needed.

VA Merit Award

Project # 5I01BX001258-04

Prostate Cancer: Targeting Androgen Receptor Signaling by Tetrandrine
Koul, Hari K.

Development of prostate cancer (PCa) and malignant progression requires altered regulation of many cellular processes, which are probably interrelated, including androgen receptor (AR) signaling, loss of growth control, and protection from apoptosis. Altered AR signaling is known to play a major role in progression of PCa to Castrate resistant phenotype. While screening for natural compounds for effects against prostate cancer cell growth, we found tetrandrine (Tet) to have selective effects against AR positive PCa cells. Tetrandrine (Tet), an active ingredient isolated from Stephania tetrandra is known to exhibit a broad range of pharmacological actions, and we are the first group to observe its effects against prostate cancer. In preliminary studies presented in this application, we also observed Tet, when injected to mice inhibited the growth of human prostate cancer xenografts in these mice, and dramatically decreased tumor volume. Tet inhibited Prostate Specific Antigen (PSA) synthesis and secretion, blocked cell cycle progression and growth of human PCa cells in culture, induced apoptosis, and inhibited cell migration and invasion, suggesting a direct effect of this compound on the neoplastic process. Based on these exciting preliminary data we hypothesize that Tetrandrine (Tet) targets Androgen Receptor signaling to modulate multiple molecular events in PCa cells that are probably interrelated, such as PSA expression, cell survival and anti-apoptotic signaling and deregulated cell cycle progression involved in uncontrolled PCa growth and malignant progression. As such, Tet may serve as a novel agent for prevention, growth control and therapy of PCa. In the current proposal we will conduct basic and pre-clinical research on Tet with an aim to understand the mechanisms of action against prostate cancer. These objectives will

be achieved in three Aims:
Aim 1, is to evaluate the effects of Tet on modulation of Androgen Receptor signaling in prostate cancer cells and to study specific molecular mechanisms by which Tet inhibits Prostate Specific Antigen (PSA);
Aim2, is to define, characterize, and establish molecular mechanism of the inhibitory effect of Tet on cell cycle progression and promotion of apoptosis in PCa cells.;
Aim 3, is to evaluate efficacy of Tet against androgen responsive and castrate resistant human PCa cell derived mouse Xenograft models in vivo.

We anticipate that proposed studies, together with our preliminary data, will identify Tet as a mechanism-based agent for the prevention, growth control and therapy of PCA, and will establish in vivo efficacy of Tet in pre-clinical human PCa cell derived xenograft models. It is important to emphasize here that an estimated 50,000 veterans being diagnosed with Prostate cancer every year and about 10,000 deaths result from prostate cancer in veteran population each year (based on Veterans Health education library and the National Prostate Cancer Coalition). Work proposed in this application, will contribute to development of a novel AR targeted therapy that may translate into an effective treatment regiment against prostate cancer and is therefore, highly relevant to Veteran health.

NIH RO1 Grant R01CA161880

Louisiana State University Health Sciences Center

Tetrandrine for the treatment of Prostate Cancer
Koul, Hari K.

In this proposal we seek to evaluate efficacy of tetrandrine as a novel targeted agent against prostate cancer in pre-clinical model systems. Altered AR signaling is known to play a major role in prostate cancer as well as progression of PCa to Castrate resistant phenotype. While screening for natural compounds for effects against prostate cancer cell growth, we found tetrandrine (Tet) to have selective effects against AR positive PCa cells. Tetrandrine (Tet), an active ingredient isolated from Stephania tetrandra is known to exhibit a broad range of pharmacological actions, and we are the first group to observe its effects against prostate cancer. In preliminary studies presented in this application, we also observed Tet, when injected to mice inhibited the growth of human prostate cancer xenografts in these mice, and dramatically decreased tumor volume. Tet inhibited Prostate Specific Antigen (PSA) synthesis and secretion, blocked cell cycle progression and growth of human PCa cells in culture, induced apoptosis, and inhibited cell migration and invasion, suggesting a direct effect of this compound on the

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Sincerely,

LSU Faculty
Shreveport, LA

CC: 1. HHS Secretary
2. Federal Bureau of Investigation
3. Office of Inspector general

1 University Place
Shreveport
LA 71115

Joseph M. Moerschbaecher, PhD
Vice Chancellor
LSU Health Sciences Center
433 Bolivar Street
New Orleans, LA 70112

Joseph M. Moerschbaecher, PhD
Vice Chancellor
LSU Health Sciences Center
433 Bolivar Street
New Orleans, LA 70112

Dear Dr. Moerschbaecher,

Re: Dr. Hari Koul, Professor, Stanley S. Scott Cancer Center, LSUHSC, New Orleans,
Scientist - Southeast Louisiana Veterans Health Care System, New Orleans, Louisiana
Fraudulent Activity, Data Manipulation, fraud and Money Laundering

This is to inform you that Dr. Hari Koul has joined the Louisiana State University Health Sciences Center, New Orleans and Southeast Louisiana Veterans Health Care System, New Orleans, Louisiana. He has moved from the LSUHSC, Shreveport and U.S. Department of Veterans Affairs, Shreveport, Louisiana. Dr. Koul was terminated from the chair position due to scientific misconduct, fraud and money laundering. An investigation was jointly carried out by the University of Colorado and the Louisiana State University Shreveport. His research was funded through the Veteran Affairs (VA Merit Award) and National Institutes of Health. The investigation committee never reported this event to the ORI/NIH and Inspector general, VA administration. The committee did not follow the proper protocols for reporting the event to the federal agencies.

His NIH and VA merit awards were exactly same. This is a misutilization of the federal government funds and considered as money laundering, fraud and not reporting truthfully. Nobody can receive the money for the same project from two federal agencies at the same time.

https://grantome.com/grant/NIH/R01-CA161880-04

Tetrandrine for the Treatment of Prostate Cancer
Koul, Hari K.
Louisiana State University Hsc Shreveport, Shreveport, LA, United States

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https://grantome.com/grant/NIH/I01-BX001258-04

Prostate Cancer: Targeting Androgen Receptor Signaling by Tetrandrine
Koul, Hari K.
VA Eastern Colorado Health Care System, Denver, CO, United States

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Furthermore, several of his papers have been retracted and corrected due to manipulation and fraud.
Please see the following information from PubPeer site.

2020 retraction. https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0234862

Following the publication of this article [1], concerns were raised regarding Fig 3:

Fig3B, first Hyper-oxaluric panel, there is an appearance of a vertical discontinuity between the first and second lane. Fig 3B, third Hyper-oxaluric panel, there is an appearance of vertical discontinuities on either side of the third lane. The authors provided underlying data that verified some but not all of the bands presented in Fig 3B. The underlying data for the remaining bands are no longer available. The corresponding author said that the underlying data are no longer available because the authors changed institutions.

The University of Colorado and the Louisiana State University Shreveport jointly investigated concerns regarding Fig 3B, but were unable to clarify the issues regarding this figure and how the experiment was conducted based on available laboratory records. The authors offered repeat experiment data for Fig 3B, but these repeat data do not resolve the concerns summarized above.

In light of the above concerns that call into question the integrity of results reported in Fig 3B, the PLOS ONE Editors retract this article.

LK and HKK agreed with the retraction. SK and RBM either did not respond directly or could not be reached. HKK stands behind the overall conclusions presented in the article.

Reference 1.Khandrika L, Koul S, Meacham RB, Koul HK (2012) Kidney Injury Molecule-1 Is Up-Regulated in Renal Epithelial Cells in Response to Oxalate In Vitro and in Renal Tissues in Response to Hyperoxaluria In Vivo. PLoS ONE 7(9): e44174. https://doi.org/10.1371/journal.pone.0044174

Retracted - **The transcription factor sterile alpha motif (SAM) pointed domain-containing ETS transcription factor (SPDEF) is required for E-cadherin expression in prostate cancer cells**
The Journal of biological chemistry (2013) - 18 Comments
pubmed: 23449978 doi: 10.1074/jbc.m112.434225

https://pubpeer.com/publications/C325F21E87D2C7B985EB8F1D201EC2

Not much came out from his federally funded grants. Using fraudulent data, he has been able to get another NIH RO1 grant (1RO1CA242839-01) and a New VA Merit Award at VA Medical Center, New Orleans. He has invited all the reviewers (working in prostate cancer field) of the NIH and the VA study sections to the SBUR society in which he was a president and active member. He has found the ways to influence the reviewers through the SBUR platform. Is there justice in the system? How a person with such a poor productivity can get an NIH RO1 first time. What is so unique about his research?

Considering the above facts, we request you to launch an inquiry and investigate this matter thoroughly. The public funds should not be mis-utilized and research should be conducted with honesty and integrity. He should be suspended from all the research activities. If you do not act, we will report this matter to the news media and other agencies.

Sincerely,

LSU Shreveport
Faculty

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Lakshmipathi Khandrika, Sweaty Koul, Randall B. Meacham, Hari K. Koul

PLoS ONE (2012)

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Loss of PDEF, a prostate-derived Ets factor is associated with aggressive phenotype of prostate cancer: regulation of MMP 9 by PDEF (/publications/A2F9C4FBCDB9BC2AE7BF8547B8FCBB#4)

Thomas R Johnson, Sweaty Koul, Binod Kumar, Lakshmipathi Khandrika, Sarah Venezia, Paul D Maroni, Randall B Meacham, Hari K Koul

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ERRATUM

You The transcription factor E2F3 suppresses prostate tumor metastasis (/publications/6E4BB6B12C436E9E7DB5095804E3B#3)

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The Journal of biological chemistry (2012)

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Mintu Pal, Sweaty Koul, Hari K. Koul

The Journal of biological chemistry (2013)

18 comments

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Oxalate selectively activates p38 mitogen-activated protein kinase and c-Jun N-terminal kinase signal transduction pathways in renal epithelial cells (/publications/C10020183BE1D0E77C992BAAB06ECD#2)

Lakshmi S. Chaturvedi, Sweaty Koul, Avtar Sekhon, Akshay Bhandari, Mani Menon, Hari K. Koul

The Journal of biological chemistry (2002)

2 comments

6 years ago

Differential effects of valproic acid on growth, proliferation and metastasis in HTB5 and HTB9 bladder cancer cell lines (/publications/CA58ABBF618A58349CCCE90E20C024#19)

Seok-Soo Byun, Fernando J. Kim, Lakshmipathi Khandrika, Binod Kumar, Sweaty Koul, Shandra Wilson, Hari K. Koul

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Lakshmipathi Khandrika, Fernando J. Kim, Adriano Campagna, Sweaty Koul, Randall B. Meacham, Hari K. Koul

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Research Integrity Officer
Louisiana State University Health Sciences Center
433 Bolivar Street
New Orleans, LA 70112

70112-702133

Moerschbacher

Research Integrity Officer
433 Bolivar Street,
New Orleans, LA 70112

Dear Sir/Madam,

Re: Dr. Hari Koul, Professor, Stanley S. Scott Cancer Center, LSUHSC, New Orleans,
Scientist - Southeast Louisiana Veterans Health Care System, New Orleans, Louisiana
Fraudulent Activity, Data Manipulation, fraud and Money Laundering

This is to inform you that Dr. Hari Koul has joined the Louisiana State University Health Sciences Center, New Orleans and Southeast Louisiana Veterans Health Care System, New Orleans, Louisiana. He has moved from the LSUHSC, Shreveport and U.S. Department of Veterans Affairs, Shreveport, Louisiana. Dr. Koul was terminated from the chair position due to scientific misconduct, fraud and money laundering. An investigation was jointly carried out by the University of Colorado and the Louisiana State University Shreveport. His research was funded through the Veteran Affairs (VA Merit Award) and National Institutes of Health. The investigation committee never reported this event to the ORI/NIH and Inspector general, VA administration. The committee did not follow the proper protocols for reporting the event to the federal agencies.

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Koul, Hari K.
Louisiana State University Hsc Shreveport, Shreveport, LA, United States

In this proposal we seek to evaluate efficacy of tetrandrine as a novel targeted agent against prostate cancer in pre-clinical model systems. Altered AR signaling is known to play a major role in prostate cancer as well as progression of PCa to Castrate resistant phenotype. While screening for natural compounds for effects against prostate cancer cell growth, we found tetrandrine (Tet) to have selective effects against AR positive PCa cells. Tetrandrine (Tet), an active ingredient isolated from Stephania tetrandra is known to exhibit a broad range of pharmacological actions, and we are the first group to observe its effects against prostate cancer. In preliminary studies presented in this application, we also observed Tet, when injected to mice inhibited the growth of human prostate cancer xenografts in these mice, and dramatically decreased tumor volume. Tet inhibited Prostate Specific Antigen (PSA) synthesis and secretion, blocked cell cycle progression and growth of human PCa cells in culture, induced apoptosis, and inhibited cell migration and invasion, suggesting a direct effect of this compound on the neoplastic process. Based on these exciting preliminary data we hypothesize that 'Tetrandrine (Tet) targets Androgen Receptor signaling to modulate multiple molecular events in PCa cells that are probably interrelated, such as PSA expression, cell survival and anti-apoptotic signaling and deregulated cell cycle progression involved in uncontrolled PCa growth and malignant progression.' As such, Tet may serve as a novel agent for prevention, growth control and therapy of PCa. In the current proposal we will conduct basic and pre-clinical research on Tet with an aim to understand the mechanisms of action against prostate cancer. These objectives will be achieved in three specific aims:
Aim 1, is to evaluate the effects of Tet on modulation of Androgen Receptor signaling in prostate cancer cells and to study specific molecular mechanisms by which Tet inhibits Prostate Specific Antigen (PSA);

Aim 2, is to define, characterize, and establish molecular mechanism of the inhibitory effect of Tet on cell cycle progression and promotion of apoptosis in PCa cells.;
Aim 3, is to evaluate efficacy of Tet against androgen responsive and castrate resistant human PCa cell derived mouse Xenograft models in vivo. We anticipate that proposed studies, together with our preliminary data, will identify Tet as a mechanism-based agent for the prevention, growth control and therapy of PCA, and will establish in vivo efficacy of Tet in pre-clinical human PCa cell derived xenograft models. It is important to emphasize here that an estimated 40,000 men die of Prostate cancer every year in the US. Work proposed in this application, will contribute to development of a novel AR targeted therapy that may translate into an effective treatment regiment against prostate cancer and is therefore, highly relevant to the mission of NCI/NIH.

https://grantome.com/grant/NIH/I01-BX001258-04

Prostate Cancer: Targeting Androgen Receptor Signaling by Tetrandrine
Koul, Hari K.
VA Eastern Colorado Health Care System, Denver, CO, United States

Development of prostate cancer (PCa) and malignant progression requires altered regulation of many cellular processes, which are probably interrelated, including androgen receptor (AR) signaling, loss of growth control, and protection from apoptosis. Altered AR signaling is known to play a major role in progression of PCa to Castrate resistant phenotype. While screening for natural compounds for effects against prostate cancer cell growth, we found tetrandrine (Tet) to have selective effects against AR positive PCa cells. Tetrandrine (Tet), an active ingredient isolated from Stephania tetrandra is known to exhibit a broad range of pharmacological actions, and we are the first group to observe its effects against prostate cancer. In preliminary studies presented in this application, we also observed Tet, when injected to mice inhibited the growth of human prostate cancer xenografts in these mice, and dramatically decreased tumor volume. Tet inhibited Prostate Specific Antigen (PSA) synthesis and secretion, blocked cell cycle progression and growth of human PCa cells in culture, induced apoptosis, and inhibited cell migration and invasion, suggesting a direct effect of this compound on the neoplastic process. Based on these exciting preliminary data we hypothesize that """""""""""Tetrandrine (Tet) targets Androgen Receptor signaling to modulate multiple molecular events in PCa cells that are probably interrelated, such as PSA expression, cell survival and anti-apoptotic signaling and deregulated cell cycle progression involved in uncontrolled PCa growth and malignant progression.""""""""""" As such, Tet may serve as a novel agent for prevention, growth control and therapy of PCa. In the current proposal we will conduct basic and pre-clinical research on Tet with an aim to understand the mechanisms of action against prostate cancer. These objectives will be achieved in three Aims:
Aim 1, is to evaluate the effects of Tet on modulation of Androgen Receptor signaling in prostate cancer cells and to study specific molecular mechanisms by which Tet inhibits Prostate Specific Antigen (PSA);
Aim2, is to define, characterize, and establish molecular mechanism of the inhibitory effect of Tet on cell cycle progression and promotion of apoptosis in PCa cells.;
Aim 3, is to evaluate efficacy of Tet against androgen responsive and castrate resistant human PCa cell derived mouse Xenograft models in vivo. We anticipate that proposed studies, together with our preliminary data, will identify Tet as a mechanism-based agent for the prevention, growth control and therapy of PCA, and will establish in vivo efficacy of Tet in pre-clinical human PCa cell derived xenograft models. It is important to emphasize here that an estimated 50,000 veterans being diagnosed with Prostate cancer every year and about 10,000 deaths result from prostate cancer in veteran population each year (based on Veterans Health education library and the National Prostate Cancer Coalition). Work proposed in this application, will contribute to development of a novel AR targeted therapy that may translate into an effective treatment regiment against prostate cancer and is therefore, highly relevant to Veteran health.

Furthermore, several of his papers have been retracted and corrected due to manipulation and fraud.
Please see the following information from PubPeer site.

2020 retraction. https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0234862

Following the publication of this article [1], concerns were raised regarding Fig 3:

Fig3B, first Hyper-oxaluric panel, there is an appearance of a vertical discontinuity between the first and second lane. Fig 3B, third Hyper-oxaluric panel, there is an appearance of vertical discontinuities on either side of the third lane. The authors provided underlying data that verified some but not all of the bands presented in Fig 3B. The underlying data for the remaining bands are no longer available. The corresponding author said that the underlying data are no longer available because the authors changed institutions.

The University of Colorado and the Louisiana State University Shreveport jointly investigated concerns regarding Fig 3B, but were unable to clarify the issues regarding this figure and how the experiment was conducted based on available laboratory records. The authors offered repeat experiment data for Fig 3B, but these repeat data do not resolve the concerns summarized above.

In light of the above concerns that call into question the integrity of results reported in Fig 3B, the PLOS ONE Editors retract this article.

LK and HKK agreed with the retraction. SK and RBM either did not respond directly or could not be reached. HKK stands behind the overall conclusions presented in the article.

Reference 1.Khandrika L, Koul S, Meacham RB, Koul HK (2012) Kidney Injury Molecule-1 Is Up-Regulated in Renal Epithelial Cells in Response to Oxalate In Vitro and in Renal Tissues in Response to Hyperoxaluria In Vivo. PLoS ONE 7(9): e44174. https://doi.org/10.1371/journal.pone.0044174

Retracted - **The transcription factor sterile alpha motif (SAM) pointed domain-containing ETS transcription factor (SPDEF) is required for E-cadherin expression in prostate cancer cells**
The Journal of biological chemistry (2013) - 18 Comments
pubmed: 23449978 doi: 10.1074/jbc.m112.434225

https://pubpeer.com/publications/C325F21E87D2C7B985EB8F1D201EC2

Not much came out from his federally funded grants. Using fraudulent data, he has been able to get another NIH RO1 grant (1R01CA242839-01) and a New VA Merit Award at VA Medical Center, New Orleans. He has invited all the reviewers (working in prostate cancer field) of the NIH and the VA study sections to the SBUR society in which he was a president and active member. He has found the ways to influence the reviewers through the SBUR platform. Is there justice in the system? How a person with such a poor productivity can get an NIH RO1 first time. What is so unique about his research?

Considering the above facts, we request you to launch an inquiry and investigate this matter thoroughly. The public funds should not be mis-utilized and research should be conducted with honesty and integrity. He should be suspended from all the research activities. If you do not act, we will report this matter to the news media and other agencies.

Sincerely,
LSU Shreveport
Faculty
CC: PubPeer