A. COVER PAGE

Project Title: Investigating the association of posttraumatic stress disorder (PTSD) with chronic kidney disease (CKD) in World Trade Center (WTC) responders	
Grant Number: 5R21OH012237-02	**Project/Grant Period:** 07/01/2021 - 06/30/2023
Reporting Period: 07/01/2022 - 06/30/2023	**Requested Budget Period:** 07/01/2022 - 06/30/2023
Report Term Frequency: Final	**Date Submitted:** 03/12/2025
Program Director/Principal Investigator Information: FARRUKH MANSOOR KORAISHY , MBBS MD PHD **Phone Number:** 3135229036 **Email:** Farrukh.Koraishy@stonybrookmedicine.edu	**Recipient Organization:** STATE UNIVERSITY NEW YORK STONY BROOK STATE UNIVERSITY NEW YORK STONY BROOK The Office of Sponsored Programs STONY BROOK, NY 117943362 **UEI:** M746VC6XMNH9 **EIN:** 1146013200F7 **RECIPIENT ID:**
Change of Contact PD/PI: NA	
Administrative Official: MICHELE CANTON 100 Nicolls Road W5510 Melville Library Stony Brook, NY 11794 **Phone number:** 6316329029 **Email:** michele.canton@stonybrook.edu	**Signing Official:** MICHELE CANTON 100 Nicolls Road W5510 Melville Library Stony Brook, NY 11794 **Phone number:** 6316329029 **Email:** michele.canton@stonybrook.edu
Human Subjects: NA	**Vertebrate Animals:** NA
hESC: No	**Inventions/Patents:** No

B. ACCOMPLISHMENTS

B.1 WHAT ARE THE MAJOR GOALS OF THE PROJECT?

The objectives of this research proposal are to test the hypotheses that severity/course of PTSD can predict the risk of CKD. This study will also test the hypothesis that kidney disease and PTSD share genetic/protein biomarkers. Using all available WTC Responder data from > 10,000 patients over a longer follow-up period and studying the association of longitudinal changes in PTSD, medications and co-morbid conditions with changes in GFR, this study will establish the association between PTSD and incident CKD. This data will be used to develop risk prediction models. Using polygenic risk scores for PTSD and CKD and stored proteomic data, this study will identify gene/protein variants related to common pathways between PTSD and CKD/GFR decline.

B.1.a Have the major goals changed since the initial competing award or previous report?

No

B.2 WHAT WAS ACCOMPLISHED UNDER THESE GOALS?

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B.3 COMPETITIVE REVISIONS/ADMINISTRATIVE SUPPLEMENTS

For this reporting period, is there one or more Revision/Supplement associated with this award for which reporting is required?

No

B.4 WHAT OPPORTUNITIES FOR TRAINING AND PROFESSIONAL DEVELOPMENT HAS THE PROJECT PROVIDED?

NOTHING TO REPORT

B.5 HOW HAVE THE RESULTS BEEN DISSEMINATED TO COMMUNITIES OF INTEREST?

NOTHING TO REPORT

B.6 WHAT DO YOU PLAN TO DO DURING THE NEXT REPORTING PERIOD TO ACCOMPLISH THE GOALS?

Not Applicable

Award Number: 5R21OH012237-02

Project Title: Investigating the association of posttraumatic stress disorder (PTSD) with chronic kidney disease (CKD) in World Trade Center (WTC) responders

Principal Investigator (PI): FARRUKH M. KORAISHY, MD, PHD

Abstract

Background: Chronic kidney disease (CKD) is a prevalent and costly condition with high mortality rates. Identifying new risk factors for eGFR decline and CKD onset is crucial. Post-traumatic stress disorder (PTSD), a common diagnosis among World Trade Center (WTC) responders, may contribute to kidney function decline. This study aimed to establish PTSD as a new risk factor for eGFR decline and CKD development and examine how PTSD severity impacts kidney function using both clinical and genetic data.

Methods: First Study: Examined WTC responders with multiple eGFR measurements. PTSD severity was categorized using the PTSD Checklist (PCL), and multinomial logistic regression assessed associations between PTSD and eGFR changes. Second Study: Analyzed 640 older adults with cardiovascular disease, hypertension, and diabetes over a 5-year period. The study explored the link between PTSD and eGFR decline using generalized estimating equations and logistic regression, adjusting for demographics, medical conditions, and psychiatric factors. Third Study: Investigated genetic influences on kidney function decline in 1,601 WTC responders with European ancestry. Polygenic risk scores (PRS) from the largest genome-wide association study of CKD progression assessed associations between genetic predisposition and kidney function decline. Fourth Study: Tracked PTSD symptoms in WTC responders over 20 years to explore their relationship with rapid eGFR decline. Models adjusted for demographics, comorbidities, and genetic factors assessed whether PTSD symptom changes were linked to kidney function decline.

Results: First Study: Found that PTSD (16.5% of participants) was associated with greater risk of both eGFR decline and rise. "Hyperarousal" PTSD symptoms showed the strongest link to GFR decline, especially in individuals over 50. Second Study: PTSD was associated with a greater decline in eGFR (2.97 vs. 2.11 ml/min/1.73 m^2 per year, $p = .022$). PTSD increased the likelihood of rapid eGFR decline by 91%, even after controlling for demographic and medical factors. Third Study: Lower baseline eGFR and increased risk of CKD progression were linked to polygenic risk scores. PRS was associated with lower eGFR, higher CKD stages, and a higher likelihood of eGFR decline. Fourth Study: In a cohort of 7,509 WTC responders, PTSD symptoms over 20 years were significantly associated with rapid eGFR decline. This association remained even after adjusting for genetic risk factors, suggesting PTSD is an independent risk factor for kidney function decline.

Conclusions: These studies indicate that PTSD is a significant risk factor for accelerated kidney function decline in both younger, relatively healthy WTC responders and older adults with existing health conditions. The findings persist even after accounting for traditional CKD risk factors. Additionally, genetic variants were linked to eGFR decline in middle-aged WTC responders with low comorbidity. The longest study tracking PTSD symptoms confirmed that worsening PTSD trajectories are independently associated with rapid GFR decline, regardless of traditional kidney disease risk factors or genetic predisposition.

Section 1

Significant or Key Findings

Aim 1: Determine if severity of PTSD is associated with increased risk of incident CKD and GFR decline.

In our first study, WTC responders with two or more eGFR measures were included. PTSD severity was classified using the PTSD Checklist (PCL): no PTSD (PCL < 40), mild PTSD (40 ≤ PCL < 50), and severe PTSD (PCL ≥ 50). We analyzed PTSD subtypes based on symptom clusters and assessed the association between PTSD and eGFR changes (decline or increase) using multinomial logistic regression. Among 2,266 participants, with an average age of 53.1 years (8.2% female, 89.1% White), those with PTSD (16.5%) had no significant baseline eGFR difference from those without PTSD (89.73 vs. 90.56 mL/min/1.73 m²; p = 0.29). Over an average follow-up of 2.01 years, the average eGFR decline was -1.51 mL/min/1.73 m²/year. PTSD was associated with eGFR decline (adjusted relative risk [aRR] = 1.74; p < 0.001), particularly with "hyperarousal" symptoms (aRR = 2.11; p < 0.001). PTSD was also linked to eGFR rise (aRR = 1.47; p = 0.009), with stronger effects in participants over 50 years. This study suggests PTSD as a novel risk factor for eGFR changes in healthy adults.

In a second study, we validated our findings in the Heart and Soul Study (640 participants, mean age 66.2 years), which includes individuals with high cardiovascular disease and comorbidities. We found PTSD was associated with greater eGFR decline over a 5-year follow-up (2.97 vs. 2.11 mL/min/1.73 m²/year; p = 0.022) and 91% higher odds of rapid eGFR decline (p < 0.001). This association persisted after adjusting for demographics, comorbidities, and psychiatric factors. Our results support that PTSD independently contributes to eGFR decline in middle-aged adults with significant comorbidities.

In the latest study, we explored the 20-year course of PTSD in WTC responders and its link to rapid eGFR decline. PTSD symptoms were measured in 7,509 responders over a mean observation period of 4.3 years. We found that PTSD symptom progression was associated with rapid eGFR decline (risk ratio [RR] = 1.09; p = 0.002), even after adjusting for demographic and health factors. This study confirms the long-term impact of PTSD on kidney function, independent of traditional kidney disease risk factors.

Aim 2: Investigate the shared pathogenesis between CKD and PTSD using genomics and proteomics.

In the first study, we analyzed genetic data from 1,601 adult WTC responders of European ancestry (mean age 49.7 years, 93% male, 23% hypertensive, 7% diabetic, and 1% with cardiovascular disease), all of whom had at least three serum creatinine measurements. Polygenic risk scores (PRSs) were calculated using single nucleotide polymorphisms (SNPs) from a large genome-wide association study (GWAS) focused on rapid eGFR decline. Generalized linear models evaluated the association between PRS and renal outcomes such as baseline eGFR, CKD stage, and the rate of eGFR change over a 3-5 year period. eGFR decline was classified as "clinical" (> -1.0 ml/min/1.73 m²/year) or "empirical" (lowest quartile of eGFR slopes). The mean baseline eGFR was approximately 86 ml/min/1.73 m². Participants with eGFR decline were more likely to have diabetes. PRS was linked to lower baseline eGFR, higher CKD stage, and greater eGFR decline after adjusting for established CKD risk factors. This study suggests that common genetic variants are associated with eGFR decline in middle-aged adults with few comorbidities.

In the most recent study, we examined whether the 20-year PTSD course in WTC responders was associated with rapid eGFR decline. A sensitivity analysis of 4,381 responders included PRSs for PTSD, chronic kidney disease, and rapid eGFR decline to assess whether PTSD symptoms were linked to eGFR decline, independent of genetic predispositions. After adjusting for three PRS models, the 20-year trajectory of PTSD symptoms was significantly associated with rapid eGFR decline in fully adjusted models (RR = 1.15 [1.00–1.33], p = 0.049). This study, the longest and largest continuous tracking of PTSD symptoms, suggests that worsening PTSD symptom trajectory is linked to rapid eGFR decline. The findings indicate that the association between PTSD and kidney function decline is independent of genetic predisposition to PTSD, CKD, and rapid eGFR decline.

Translation of Findings

The research indicates that PTSD, particularly severe forms with hyperarousal symptoms, is linked to kidney function decline as measured by eGFR, positioning it as a possible risk factor for kidney dysfunction. Healthcare providers should consider PTSD in their monitoring of kidney health, especially in older individuals or those with underlying conditions such as diabetes and hypertension, as PTSD may accelerate eGFR decline. Early kidney disease screening for PTSD patients, especially those with comorbidities, could help prevent further kidney deterioration. Genetic findings also reveal that polygenic risk scores (PRSs) are associated with eGFR decline, offering the potential for personalized care through genetic assessments to predict kidney disease risk. Moreover, the long-term relationship between PTSD symptom progression and rapid eGFR decline highlights the need for continuous monitoring of both mental health and kidney function in PTSD patients, particularly over extended periods. This emphasizes the importance of integrating psychiatric and nephrological care in treating PTSD patients, ensuring a comprehensive approach to addressing both mental health and kidney health.

Research Outcomes/Impact

The research highlights a significant link between PTSD and the decline in kidney function, positioning PTSD as a potential new risk factor for chronic kidney disease (CKD) and decreased glomerular filtration rate (GFR). Across several studies involving different cohorts, the findings consistently showed that PTSD is associated with an increased risk of eGFR decline over time, even after adjusting for demographic factors, comorbidities, and other mental health disorders. Specifically, individuals with PTSD were more likely to experience rapid eGFR decline, especially those with severe PTSD symptoms and older adults. These results suggest that PTSD symptoms, particularly hyperarousal, may worsen kidney function decline, which has important clinical implications. Early detection and management of PTSD could potentially help prevent or slow the progression of kidney disease. Additionally, genetic analyses revealed that certain common genetic variants linked to eGFR decline might help identify individuals at higher risk, underscoring the importance of more comprehensive screening and personalized treatment strategies. Furthermore, the studies indicated that the relationship between PTSD and kidney function decline is independent of genetic susceptibility to both PTSD and CKD, highlighting the potential for therapeutic approaches targeting PTSD symptoms as part of kidney disease management. These findings suggest that integrating mental health care, particularly for individuals with PTSD, into kidney disease management could improve patient outcomes by addressing both mental and physical health factors.

Section 2

Scientific Report and Publications

Aim 1: Determine if severity of PTSD is associated with increased risk of incident CKD and GFR decline.

In our first study, WTC responders were included if they had two or more measures of eGFR. The PTSD Checklist (PCL) was used to classify PTSD severity: no PTSD (PCL < 40), mild PTSD (40 ≤ PCL < 50), and severe PTSD (PCL ≥ 50). PTSD subtypes based on symptom clusters were also analyzed. Multinomial logistic regression was used to assess the association between PTSD and two eGFR change outcomes (decline or increase) compared to stable eGFR. Among 2,266 participants, the mean age was 53.1 years, 8.2% were female, and 89.1% were White. Individuals with PTSD (n = 373; 16.5%) did not differ significantly in mean baseline eGFR from those without PTSD (89.73 vs. 90.56 mL min^{-1} 1.73 m^{-2}; p = 0.29). During a mean follow-up of 2.01 years, the average eGFR decline was -1.51 mL min^{-1} 1.73 m^{-2} per year. In multivariable-adjusted models, PTSD was associated with eGFR decline (adjusted relative risk [aRR] = 1.74 [1.32-2.30], p < 0.001) compared to stable eGFR, with "hyperarousal" symptoms showing the strongest association (aRR = 2.11 [1.40-3.19], p < 0.001). Dose-response effects were observed when comparing mild versus severe PTSD and when comparing PTSD with and without depression. PTSD was also associated with eGFR rise (aRR = 1.47 [1.10-1.97], p = 0.009). The association between PTSD and eGFR change was stronger in participants older than 50 years. The conclusion of this study is that PTSD may be a novel risk factor for exaggerated longitudinal eGFR change in young, healthy adults.

This work was subsequently published in a peer-reviewed journal:

The Association of Posttraumatic Stress Disorder With Longitudinal Change in Glomerular Filtration Rate in World Trade Center Responders. Farrukh M Koraishy , Steven G Coca, Beth E Cohen, Jeffery F Scherrer, Frank Mann, Pei-Fen Kuan, Benjamin J Luft, Sean A P Clouston. Psychosom Med. 2021 Nov-Dec;83(9):978-986. doi: 10.1097/PSY.0000000000000968. PMID: 34297009.

We conducted a second study to validate our findings in an independent cohort of 640 adult participants from the Heart and Soul Study (mean baseline age 66.2 years), a population with high prevalence of cardiovascular disease, hypertension, and diabetes. We examined the association between PTSD and eGFR decline over a 5-year follow-up. Generalized estimating equations were used to estimate the association between PTSD diagnosis and eGFR decline slope, and logistic regression was used to estimate the association between PTSD and the odds of "rapid" versus "mild" eGFR decline. Six sequential adjustment models were used: unadjusted (Model 1), adjusted for demographics (age, gender, race) (Model 2), medical comorbidities (BMI, hypertension, diabetes, congestive heart failure, left ventricular hypertrophy, stroke, and angina) (Model 3), other psychiatric diagnoses (major depressive disorder and generalized anxiety disorder) (Model 4), psychiatric medication use (Model 5), and alcohol intake and smoking (Model 6). We observed significantly greater estimated eGFR decline over time in those with PTSD compared to those without (2.97 vs. 2.11 mL/min/1.73 m^2/year; p = 0.022). PTSD was associated with 91% (95% CI 12%-225%) higher odds of rapid eGFR decline (defined as >3.0 mL/min/1.73 m^2/year) compared to mild decline (defined as <3.0 mL/min/1.73

m²/year). These associations remained consistent after controlling for demographics, medical comorbidities, other mental disorders, and psychiatric medications. In conclusion, this study provides evidence that PTSD is independently associated with eGFR decline in middle-aged adults with a high comorbidity burden, suggesting the need for further research in larger cohorts with longer follow-up periods.

This work was subsequently published in a peer-reviewed journal:

The association of post-traumatic stress disorder with glomerular filtration rate decline. Farrukh M Koraishy, Beth E Cohen, Jeffery F Scherrer, Mary Whooley, Janos Hajagos, Cassianne Robinson-Cohen, Wei Hou. Nephrology (Carlton). 2023 Mar;28(3):181-186. doi: 10.1111/nep.14140. Epub 2023 Jan 20. PMID: 36594760

In the most recent study, we tested whether the 20-year course of PTSD in WTC responders is associated with rapid eGFR decline. PTSD symptoms were measured using the PCL-17 from July to December 2022, and initial levels and rates of change in PTSD symptoms were operationalized using random intercepts and random slopes from a generalized multi-level model. Rapid eGFR decline was defined as eGFR decline of < -5 mL/min/1.73 m²/year, and stable eGFR was defined as change between -1.0 and +1.0 mL/min/1.73 m²/year, based on serial measures of eGFR (minimum of two) from 2015 to 2023. A series of robust quasi-Poisson regressions assessed whether linear rates of change in PTSD symptoms were associated with rapid eGFR decline compared to stable eGFR, adjusting for demographics (age, sex, race/ethnicity), comorbidities (diabetes, hypertension, cardiovascular disease, obesity), and severity of PTSD. In the primary cohort of 7,509 responders (91% male, 83% White), 41% had stable eGFR and 11% had rapid eGFR decline over a mean observation period of 4.3 ± 1.7 years. The 20-year linear slope of PTSD symptoms was significantly associated with rapid eGFR decline (risk ratio [RR] = 1.09 [1.03-1.14], p = 0.002) and remained significant in fully adjusted models (RR = 1.08 [1.01-1.17], p = 0.035). The conclusion of this study is that the association between PTSD and kidney function decline is independent of traditional kidney disease risk factors.

This work is now completed in a manuscript form to be soon submitted for publication in a peer-reviewed journal.

Aim 2: Investigate the shared pathogenesis between CKD and PTSD using genomics and proteomics.

In the first study related to this aim, we analyzed genetic data from 1,601 adult WTC responders of European ancestry (mean age 49.68 ± 8.79 years, 93% male, 23% hypertensive, 7% diabetic, and 1% with cardiovascular disease), all of whom had at least three serial measurements of serum creatinine. Polygenic risk scores (PRSs) were calculated based on an aggregation of single nucleotide polymorphisms (SNPs) derived from a large-scale genome-wide association study (GWAS) focused on rapid eGFR decline. Generalized linear models were used to evaluate the association between PRS and renal outcomes, including baseline eGFR, CKD stage, rate of change in eGFR, and stable versus declining eGFR over a 3–5-year observation period. eGFR decline was defined in separate analyses as "clinical" (> -1.0 ml/min/1.73 m²/year) or "empirical" (the lowest quartile of eGFR slopes). The mean baseline eGFR was approximately 86 ml/min/1.73 m². Subjects who experienced eGFR decline were more likely to have diabetes. PRS was significantly associated with lower baseline eGFR (B = -0.96, p = 0.002), higher CKD stage (OR = 1.17, p = 0.010), eGFR decline (OR = 1.14, p =

0.036) compared with stable eGFR, and the lowest quartile of eGFR slopes (OR = 1.21, p = 0.008), after adjusting for established CKD risk factors. The conclusion of this study is that common genetic variants are associated with eGFR decline in middle-aged adults with relatively low comorbidity burdens.

This work was subsequently published in a peer-reviewed journal:

Polygenic association of glomerular filtration rate decline in world trade center responders. Farrukh M Koraishy, Frank D Mann , Monika A Waszczuk, Pei-Fen Kuan, Katherine Jonas, Xiaohua Yang, Anna Docherty, Andrey Shabalin, Sean Clouston, Roman Kotov, Benjamin Luft. BMC Nephrol. 2022 Oct 28;23(1):347. doi: 10.1186/s12882-022-02967-5. PMID: 36307804.

In the most recent study, we tested whether the 20-year course of PTSD in WTC responders was associated with rapid eGFR decline. A sensitivity analysis was conducted on a sub-cohort of 4,381 responders, which included PRSs for PTSD symptoms, chronic kidney disease, and rapid eGFR decline, to assess whether the association between PTSD symptom trajectory and rapid eGFR decline was robust to genetic predispositions for PTSD and kidney disease. In the PRS cohort (European ancestry), after adjusting for three PRS models, the 20-year linear trajectory of PTSD symptoms remained significantly associated with rapid eGFR decline (vs. stable eGFR) in fully adjusted models (RR = 1.15 [1.00–1.33], p = 0.049). This study represents the longest and largest continuous tracking of PTSD symptoms since exposure, and our findings suggest that worsening PTSD symptom trajectory is associated with rapid GFR decline. The conclusion from this study is that the association between PTSD and kidney function decline is independent of genetic predisposition to PTSD, CKD, and rapid eGFR decline.

This work is now completed in a manuscript form to be soon submitted for publication in a peer-reviewed journal.

Subsequent grant applications resulting from this work:

Intra-mural:

We recently obtained a seed grant from the Dialysis Clinic Inc. (DCI). DCI Reserve funds. RF#C-421 " Identification of post-traumatic stress disorder and its impact on outcomes in hemodialysis patients". The objective is to study PTSD in the local Stony Brook University hemodialysis population using machine learning. The PI of the R21 grant (Dr. Farrukh Koraishy) is also the PI of this grant proposal.

Extra-mural:

In 2024, we applied for an NIH R01 on the association of PTSD and Depression with CKD that was not funded. We have re-submitted the proposal for the NIH R01 grant after major updates again in the spring of 2025. The PI of the R21 grant (Dr. Farrukh Koraishy) is also the PI of this grant proposal.

C. PRODUCTS

C.1 PUBLICATIONS

Are there publications or manuscripts accepted for publication in a journal or other publication (e.g., book, one-time publication, monograph) during the reporting period resulting directly from this award?

Yes

Publications Reported for this Reporting Period

Public Access Compliance	Citation
N/A: Not NIH Funded	Koraishy FM, Coca SG, Cohen BE, Scherrer JF, Mann F, Kuan PF, Luft BJ, Clouston SAP. The Association of Posttraumatic Stress Disorder With Longitudinal Change in Glomerular Filtration Rate in World Trade Center Responders. Psychosomatic medicine. 2021 November;83(9):978-986. PubMed PMID: 34297009; PubMed Central PMCID: PMC8578353; DOI: 10.1097/PSY.0000000000000968.
N/A: Not NIH Funded	Koraishy FM, Mann FD, Waszczuk MA, Kuan PF, Jonas K, Yang X, Docherty A, Shabalin A, Clouston S, Kotov R, Luft B. Polygenic association of glomerular filtration rate decline in world trade center responders. BMC nephrology. 2022 October 28;23(1):347. PubMed PMID: 36307804; PubMed Central PMCID: PMC9615399; DOI: 10.1186/s12882-022-02967-5.
N/A: Not NIH Funded	Koraishy FM, Cohen BE, Scherrer JF, Whooley M, Hajagos J, Robinson-Cohen C, Hou W. The association of post-traumatic stress disorder with glomerular filtration rate decline. Nephrology (Carlton, Vic.). 2023 March;28(3):181-186. PubMed PMID: 36594760; PubMed Central PMCID: PMC9974752; DOI: 10.1111/nep.14140.

C.2 WEBSITE(S) OR OTHER INTERNET SITE(S)

NOTHING TO REPORT

C.3 TECHNOLOGIES OR TECHNIQUES

NOTHING TO REPORT

C.4 INVENTIONS, PATENT APPLICATIONS, AND/OR LICENSES

Have inventions, patent applications and/or licenses resulted from the award during the reporting period? No

If yes, has this information been previously provided to the PHS or to the official responsible for patent matters at the grantee organization? No

C.5 OTHER PRODUCTS AND RESOURCE SHARING

NOTHING TO REPORT

D. PARTICIPANTS

D.1 WHAT INDIVIDUALS HAVE WORKED ON THE PROJECT?

Commons ID	Sr/Key	Name	Degree(s)	Role	Cal	Aca	Sum	Foreign Org	Country	SS
FKORAISHY	Y	Koraishy, Farrukh Mansoor	PHD,MBBS,MD	PD/PI	1.8	0.0	0.0			NA
BJLUFT	Y	LUFT, BENJAMIN J	MD,BA	Co-Investigator	0.1	0.0	0.0			NA
PFKUAN	Y	Kuan, Pei-Fen	PHD	Co-Investigator	0.0	0.3	0.0			NA
	N	Espinoza, Melissa	MA	Clinical Research Assistant	6.0	0.0	0.0			NA

Glossary of acronyms:
Sr/Key - Senior/Key
Cal - Person Months (Calendar)
Aca - Person Months (Academic)
Sum - Person Months (Summer)
Foreign Org - Foreign Organization Affiliation
SS - Supplement Support
RS - Reentry Supplement
DS - Diversity Supplement
OT - Other
NA - Not Applicable

D.2 PERSONNEL UPDATES

D.2.a Level of Effort

Not Applicable

D.2.b New Senior/Key Personnel

Not Applicable

D.2.c Changes in Other Support

Not Applicable

D.2.d New Other Significant Contributors

Not Applicable

D.2.e Multi-PI (MPI) Leadership Plan

Not Applicable

E. IMPACT

E.1 WHAT IS THE IMPACT ON THE DEVELOPMENT OF HUMAN RESOURCES?

Not Applicable

E.2 WHAT IS THE IMPACT ON PHYSICAL, INSTITUTIONAL, OR INFORMATION RESOURCES THAT FORM INFRASTRUCTURE?

NOTHING TO REPORT

E.3 WHAT IS THE IMPACT ON TECHNOLOGY TRANSFER?

Not Applicable

E.4 WHAT DOLLAR AMOUNT OF THE AWARD'S BUDGET IS BEING SPENT IN FOREIGN COUNTRY(IES)?

NOTHING TO REPORT

G. SPECIAL REPORTING REQUIREMENTS SPECIAL REPORTING REQUIREMENTS

G.1 SPECIAL NOTICE OF AWARD TERMS AND NOTICE OF FUNDING OPPORTUNITIES REPORTING REQUIREMENTS

NOTHING TO REPORT

G.2 RESPONSIBLE CONDUCT OF RESEARCH

Not Applicable

G.3 MENTOR'S REPORT OR SPONSOR COMMENTS

Not Applicable

G.4 HUMAN SUBJECTS

G.4.a Does the project involve human subjects?

Not Applicable

G.4.b Inclusion Enrollment Data

NOTHING TO REPORT

G.4.c ClinicalTrials.gov

Does this project include one or more applicable clinical trials that must be registered in ClinicalTrials.gov under FDAAA?

G.5 HUMAN SUBJECTS EDUCATION REQUIREMENT

NOT APPLICABLE

G.6 HUMAN EMBRYONIC STEM CELLS (HESCS)

Does this project involve human embryonic stem cells (only hESC lines listed as approved in the NIH Registry may be used in NIH funded research)?

No

G.7 VERTEBRATE ANIMALS

Not Applicable

G.8 PROJECT/PERFORMANCE SITES

Not Applicable

G.9 FOREIGN COMPONENT

No foreign component

G.10 ESTIMATED UNOBLIGATED BALANCE

Not Applicable

G.11 PROGRAM INCOME

Not Applicable

G.12 F&A COSTS

Not Applicable

I. OUTCOMES

I.1 What were the outcomes of the award?

Our work as a result of this grant lead to a significant enhancement of our knowledge on the association of post-traumatic stress disorder with kidney function decline. These are some of initial studies ever done is this important medical area.
Our work was published in peer-reviewed journals and lead to a new grant to study the risk of kidney disease in individuals with mental health disorders like PTSD and depression. in national cohorts.