Original Investigation

Genome-wide Association Studies of Posttraumatic Stress Disorder in 2 Cohorts of US Army Soldiers

Murray B. Stein, MD, MPH; Chia-Yen Chen, ScD; Robert J. Ursano, MD; Tianxi Cai, ScD; Joel Gelernter, MD; Steven G. Heeringa, PhD; Sonia Jain, PhD; Kevin P. Jensen, PhD; Adam X. Malhofer, MS; Colter Mitchell, PhD; Caroline M. Nievergelt, PhD; Matthew K. Nock, PhD; Benjamin M. Neale, PhD; Renato Polimanti, PhD; Stephan Ripke, MD; Xiaoying Sun, MS; Michael L. Thomas, PhD; Qian Wang, PhD; Erin B. Ware, PhD; Susan Borja, PhD; Ronald C. Kessler, PhD; Jordan W. Smoller, MD, ScD; for the Army Study to Assess Risk and Resilience in Servicemembers (STARRS) Collaborators

IMPORTANCE Posttraumatic stress disorder (PTSD) is a prevalent, serious public health concern, particularly in the military. The identification of genetic risk factors for PTSD may provide important insights into the biological foundation of vulnerability and comorbidity.

OBJECTIVE To discover genetic loci associated with the lifetime risk for PTSD in 2 cohorts from the Army Study to Assess Risk and Resilience in Servicemembers (Army STARRS).

DESIGN, SETTING, AND PARTICIPANTS Two coordinated genome-wide association studies of mental health in the US military contributed participants. The New Soldier Study (NSS) included 3167 unique participants with PTSD and 4607 trauma-exposed control individuals; the Pre/Post Deployment Study (PPDS) included 947 unique participants with PTSD and 4969 trauma-exposed controls. The NSS data were collected from February 1, 2011, to November 30, 2012; the PDDS data, from January 9 to April 30, 2012. The primary analysis compared lifetime DSM-IV PTSD cases with trauma-exposed controls without lifetime PTSD. Data were analyzed from March 18 to December 27, 2015.

MAIN OUTCOMES AND MEASURES Association analyses for PTSD used logistic regression models within each of 3 ancestral groups (European, African, and Latino American) by study, followed by meta-analysis. Heritability and genetic correlation and pleiotropy with other psychiatric and immune-related disorders were estimated.

RESULTS The NSS population was 80.7% male (6277 of 7774 participants; mean [SD] age, 20.9 [3.3] years); the PPDS population, 94.4% male (5583 of 5916 participants; mean [SD] age, 26.5 [6.0] years). A genome-wide significant locus was found in ANKRD55 on chromosome 5 (rs159572; odds ratio [OR], 1.62; 95% CI, 1.37-1.92; P = 2.34 x 10^-8) and persisted after adjustment for cumulative trauma exposure (adjusted OR, 1.64; 95% CI, 1.39-1.95; P = 1.18 x 10^-8) in the African Americans samples from the NSS. A genome-wide significant locus was also found in or near ZNF626 on chromosome 19 (rs11085374; OR, 0.77; 95% CI, 0.70-0.85; P = 4.59 x 10^-8) in the European American samples from the NSS. Similar results were not found for either single-nucleotide polymorphism in the corresponding ancestry group from the PPDS sample, in other ancestral groups, or in transancestral meta-analyses. Single-nucleotide polymorphism-based heritability was nonsignificant, and no significant genetic correlations were observed between PTSD and 6 mental disorders or 9 immune-related disorders. Significant evidence of pleiotropy was observed between PTSD and rheumatoid arthritis and, to a lesser extent, psoriasis.

CONCLUSIONS AND RELEVANCE In the largest genome-wide association study of PTSD to date, involving a US military sample, limited evidence of association for specific loci was found. Further efforts are needed to replicate the genome-wide significant association with ANKRD55—associated in prior research with several autoimmune and inflammatory disorders—and to clarify the nature of the genetic overlap observed between PTSD and rheumatoid arthritis and psoriasis.
P

osttrumatic stress disorder (PTSD) is a common consequence of exposure to extreme, life-threatening events. Posttraumatic stress disorder is also frequently associated with other mental health problems such as major depressive disorder, substance abuse, and suicidality, with other adverse health sequelae such as obesity, cardiovascular disease, and type 2 diabetes mellitus, and with immune-related disorders, such as rheumatoid arthritis.

Although most Americans (50%-85%) experience traumatic events during their lifetime, the lifetime prevalence of PTSD is approximately 7%, suggesting differential vulnerability to the disorder. Rates of trauma exposure and PTSD are higher among US military personnel and veterans, particularly those exposed to combat. Much of the research on the risk for PTSD has focused on the differential impact of type, frequency, duration, and consequences (eg, extent of physical injury) of trauma exposures. Pretrauma risk factors, including personality characteristics and early life experiences, have also been scrutinized extensively, as have posttrauma factors, such as social support.

Twin studies have long established that genetic variation contributes to risk for PTSD symptoms, with heritability estimates in the range of 0.28 to 0.46. Genetic association studies have focused on a limited set of candidate genes and have been largely underpowered to detect loci of modest effect. More recently, several genome-wide association studies (GWAS) of PTSD have been reported in civilian and military or veteran samples, yielding several genome-wide significant associations that have yet to be widely replicated.

The present investigation uses data from the Army Study to Assess Risk and Resilience in Servicemembers (Army STARRS), a large, coordinated set of study components intended to improve understanding of suicide, PTSD, and related mental health risk and resilience in the US Army. Blood samples for DNA were provided by participants in the following 2 components of Army STARRS: a study of new soldiers during their first week of basic training (New Soldier Study [NSS]), and a study of 3 brigade combat teams before their deployment to Afghanistan (Pre/Post Deployment Study [PPDS]). Each of these studies has, to our knowledge, a larger PTSD-affected sample than any previously published genetic study of PTSD. We report herein results from within-ancestral-group and within-study genome-wide analyses, followed by meta-analyses across studies.

Methods

Participants

Detailed information about the design and conduct of Army STARRS is available in a separate report and in the eMethods in the Supplement. The recruitment, consent, human participant, and data protection procedures were approved by the Uniformed Services University of the Health Sciences, Harvard University, University of Michigan, and University of California, San Diego. Written informed consent was obtained from all participants.

Key Points

Findings: In a genome-wide association study, 2 genome-wide significant loci were associated with PTSD in a cohort of new soldiers, but these findings were not replicated in a separate sample of soldiers. We also found significant evidence of genetic pleiotropy between PTSD and rheumatoid arthritis and psoriasis.

New Soldier Study

The NSS was performed among new soldiers at the start of their basic training at 1 of 3 army installations from February 1, 2011, to November 30, 2012. Of 39784 NSS participants who completed the computerized, self-administered questionnaire, 33088 (83.2%) provided blood samples. Genotyping was conducted on samples from the first half of the cohort, from which 7999 samples were selected based on phenotype and case-control status (NSS1). When the remaining half of the cohort collection was completed, we selected an informative subset of 2835 samples for genotyping (NSS2) (eMethods in the Supplement).

Pre/Post Deployment Survey

The PPDS is a multiple-wave panel survey that collected baseline data (time 0) from US Army soldiers in 3 brigade combat teams from January 9 to April 30, 2012, within approximately 6 weeks of their deployment to Afghanistan. Seven thousand nine hundred twenty-seven PPDS soldiers with eligible self-administered questionnaire responses underwent genotyping for the GWAS.

Demographics and Case-Control Status

The population, sex, and age composition of our analyzed sample of cases and controls is shown in Table 1. Most of the participants were male, and we analyzed male and female participants together. A total of 3167 unique PTSD cases and 4607 trauma-exposed controls from NSS1 and NSS2 as well as 947 unique PTSD cases and 4969 trauma-exposed controls from PPDS entered the statistical analysis.

Measures

The self-administered questionnaire included a computerized version of the Composite International Diagnostic Interview Screening Scales and a screening version of the PTSD Checklist (PCL) (range, 17-85, with higher scores indicating worse symptoms). Trauma exposure was assessed from answers pertaining to childhood, adulthood civilian, and, for PPDS participants, military traumatic events (eMethods in the Supplement). A diagnosis of PTSD was assigned using multiple imputation methods that relied on the PCL and Composite International Diagnostic Interview Screening Scales data; our clinical reappraisal study found satisfactory concordance with independent clinical diagnoses based on blinded Struc...
sis) software tool. We tested the genetic correlation (propor-
tion using the GCTA (genome-wide complex trait analy-
sis) below).
lished military-relevant datasets (described in the Results sec-
analysis. We sought external replication with other pub-
sis of the PPDS sample is our internal attempt at replication.
regional plots\textsuperscript{58} for 300-kilobase regions around the 2 most significant (top-hit) SNPs (eFigure 7 in the Supplement). No significant associations were observed in the Latino NSS or PPDS samples or in any of the transethnic meta-analyses. Adjustment for lifetime trauma exposure slightly strengthened the genome-wide significant associations for the 2 lead SNPs (eTable 1 in the Supplement), whereas simultaneous adjustment for lifetime trauma exposure, sex, and age slightly attenuated the associations (eTable 2 and eFigure 8 in the Supplement).

The top SNP, rs159572 (eFigure 7A in the Supplement), on chromosome 5 is intronic to the ankyrin repeat domain 55 gene (\textit{ANKRD55} [NCBI Entrez Gene 79722]), and multiple other SNPs in this region were in LD with this SNP. ANKRD55 has been associated with several autoimmune diseases, including multiple sclerosis,\textsuperscript{59,60} type 2 diabetes mellitus,\textsuperscript{61} celiac disease,\textsuperscript{62} and rheumatoid arthritis.\textsuperscript{63} The top SNP, rs11085374, on chromosome 19 is located near the zinc finger protein 626 gene (\textit{ZNF626} [NCBI Entrez Gene 199777]). We found minimal LD between this SNP and surrounding SNPs (eFigure 7B in the Supplement), and no other SNPs in the region showed evidence of association.

### Meta-analysis With Other Military Data Sets
We performed a meta-analysis of the results for SNP rs159572 on chromosome 5 between 3 GWAS of African American military samples, including data reported herein from Army STARRS (the current analysis), the Marine Resiliency Study,\textsuperscript{31} and a recently published genetic study of Iraq-Afghanistan US veterans\textsuperscript{32} (Figure 2). The results were directionally consistent in the Army STARRS NSS and PPDS samples as well as the Marine Resiliency Study but not in the PTSD veteran GWAS (OR for meta-analysis, 1.17; 95% CI, 1.05-1.31).

### Alternate Phenotypic Characterization
To examine the robustness of our findings, we tested for association of the top 2 SNPs at the chromosome 19 and 5 loci with an alternate phenotypic characterization of PTSD; all par-
Participants in the respective ancestral groups were included. For this purpose, we chose a dimensional measure of lifetime worst PTSD severity from a 6-item version of the PCL (PCL-6) (range, 6-30, with higher scores indicating worse symptoms) that has been used in other published Army STARRS research reports. Among European American participants, the number of risk alleles (0-2) for rs11085374 was nominally significantly associated with lifetime PCL-6 severity in the NSS1 ($P = .007$) and NSS2 ($P < .001$) but not PPDS ($P = .82$) samples.

Among African American participants, the number of risk alleles (0-2) for rs159572 was nominally significantly associated with lifetime PCL-6 severity in the NSS1 ($P = .002$) and NSS2 ($P = .03$) but not PPDS ($P = .42$) samples.

**Heritability of Lifetime PTSD Phenotype**

We estimated SNP-based heritability ($h^2_g$) using the GCTA software tool in European American samples for the NSS1, NSS2, PPDS, and all cohorts pooled together. We found no significant $h^2_g$ estimates in overall ($h^2_g = 0.062 \text{ (SE, 0.049); } P = .10$) or sex-specific analyses (eTable 3 in the Supplement).

**Pleiotropy and Genetic Correlation**

We tested the pleiotropy shared by PTSD and 6 psychiatric disorders and 9 immune-related disorders in the European American samples (Table 3). Significant pleiotropy was observed for PTSD and rheumatoid arthritis ($P = 3.04 \times 10^{-7}$) and psoriasis ($P = 2.41 \times 10^{-8}$). No significant pleiotropy was observed between PTSD and the other psychiatric disorders tested. No sig-
significant genetic correlations were found in the same data sets (eTable 4 in the Supplement).

To further characterize the observed pleiotropy, we performed an enrichment analysis of SNPs with pleiotropy posterior probability of greater than 0.5. For pleiotropy for PTSD and rheumatoid arthritis, we observed several significant enrichments for medical subject headings for tissue and cell type annotations (eTable 5 in the Supplement) and gene ontology terms (eTable 6 in the Supplement) related to several immune systems and functions. No enrichment was present for pleiotropy for PTSD and psoriasis. Finally, we estimated that the probability for an SNP associated with PTSD to be a central nervous system SNP is 2.28 (SE, 0.24) times the probability for an SNP not associated with PTSD to be a central nervous system SNP. Similarly, the enrichment ratio for immune-related expression quantitative trait loci in PTSD is 2.36 (SE, 0.24).

**Abbreviations:** PGC, Psychiatric Genomics Consortium; PTSD, posttraumatic stress disorder.

### Table 3. Genetic Pleiotropy Analysis Between PTSD and Other Disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immune-related</td>
<td></td>
</tr>
<tr>
<td>Crohn disease</td>
<td>.64</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>.96</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>2.41 × 10⁻³</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>3.04 × 10⁻⁹</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>.87</td>
</tr>
<tr>
<td>Type 1 diabetes mellitus</td>
<td>.13</td>
</tr>
<tr>
<td>Ulcerative colitis</td>
<td>.38</td>
</tr>
<tr>
<td>Celiac disease</td>
<td>.049</td>
</tr>
<tr>
<td>Primary biliary cirrhosis</td>
<td>.09</td>
</tr>
<tr>
<td>Psychiatric</td>
<td></td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>.12</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>.78</td>
</tr>
<tr>
<td>Attention-deficit/hyperactivity disorder</td>
<td>.89</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>.78</td>
</tr>
<tr>
<td>Autism spectrum disorder</td>
<td>.84</td>
</tr>
<tr>
<td>PGC cross-disorder</td>
<td>.29</td>
</tr>
</tbody>
</table>

### Discussion

This study is, to our knowledge, the largest GWAS of PTSD conducted to date. Because the study is reflective of the US Army, the composition of the samples was ethnically diverse, obligating us to conduct initial association analyses within ancestral groups and then to attempt transancestral meta-analyses. We found no genome-wide significant loci at the level of the transancestral meta-analyses but found 2 genome-wide significant loci, one each in the African American and European American samples from the NSS.

In the African American NSS sample, we observed genome-wide significant association with PTSD for SNPs on chromosome 5 in ANKRD55. Inclusion of data from African American participants from additional military cohorts continued to yield a significant result of the meta-analysis at this locus, albeit attenuated compared with that of the NSS alone (Figure 2). This gene, whose function is currently unknown, has been reported to be associated with a range of autoimmune and inflammatory disorders, including multiple sclerosis, type 2 diabetes mellitus, celiac disease, and rheumatoid arthritis.

We also found evidence of significant pleiotropy between PTSD and 2 immune-related disorders, namely, rheumatoid arthritis and, to a lesser extent, psoriasis. We may have seen this genetic overlap in European American participants (whereas the ANKRD55 association finding was in African American individuals) because human populations can present ancestry-specific risk alleles in the context of similar underlying biological mechanisms of disease predisposition.

These novel findings are consistent with recent reports of pleiotropy between other mental disorders, such as schizophrenia, and immune disorders, such as rheumatoid arthritis and multiple sclerosis. In the context of new evidence that schizophrenia involves allelic variation in the major histocompatibility complex, these observations suggest that intensive scrutiny of immune factors, and perhaps in particular of complement component 4, should be the subject of further study in other mental disorders, such as PTSD.

A hypothetical immune-related or inflammatory cause of PTSD has, in fact, gained some empirical support. Two recent studies have found elevated levels of the inflammatory biomarker C-reactive protein in individuals at risk for PTSD or with PTSD. Other studies have found abnormal cytokine regulation or other evidence of a proinflammatory milieu in PTSD. Posttraumatic stress disorder is itself highly comorbid with several of the disorders associated with ANKRD55, including type 2 diabetes mellitus and rheumatoid arthritis. Moreover, a recent epidemiologic study of Iraq and Afghanistan military veterans found PTSD to be associated with a broad range of autoimmune disorders, including inflammatory bowel disease, rheumatoid arthritis, and multiple sclerosis. Further research is needed to determine whether variation in ANKRD55—or other genes contributing to the observed pleiotropy—accounts for these associations.

Why the association of ANKRD55 with PTSD is apparently restricted to African American individuals should also be determined, although this restriction may be explained by differing LD block structure and increased minor allele frequency.

We also detected in the European American NSS samples 1 genome-wide significant SNP on chromosome 19 near ZNF626, a gene thought to be involved in the regulation of RNA transcription. The regional plot showed no other associated SNPs in LD with this result. This finding may represent a spurious association, but it also may reflect a lack of nearby variants in LD with the index SNP, which requires further study.

Genome-wide significant results from the NSS were not replicated in the PPDS. The PPDS sample was smaller and distinct in important ways from the NSS sample. Participants in the NSS were younger (mostly 18-20 years of age), and their trauma exposure and resultant PTSD were entirely premilitary. In contrast, PPDS participants were older, their mean nonmilitary trauma exposure was higher than in the NSS (reflect-
tial limitations. First, samples sizes—especially within ances-

tal groups—are likely to be insufficiently powered to de-
tect loci of modest effect. Given our total sample size, we would
have 80% power to detect associations for SNPs with 20% mi-
nor allele frequency with an OR of 1.2 or higher. Second, the
genetic correlation of the risk for trauma exposure with risk
for PTSD is well established.24,86,87 Therefore, although ex-
clusion of control individuals with no trauma exposure should
have improved our power to detect PTSD risk loci given trauma
exposure, it may have reduced our ability to detect loci that
contribute to PTSD by increasing the risk for trauma. Third, our
finding of no apparent heritability emanating from the GCTA
analyses may be owing to insufficient power. Fourth, the use
of the GPA package to detect pleiotropy is quite novel, and her-
tofoe unappreciated limitations in this approach may exist.

Conclusions

We found no genome-wide significant evidence that tran-
scended ancestry and replicated across studies. We did, how-
ever, find genome-wide significant evidence of an associa-
tion of ANKRD55, a gene previously associated with inflam-
atory and immune disorders, with PTSD in African
American participants. This association was observed in a pre-
military PTSD sample (NSS), not replicated in a mixed premili-
tary and military PTSD sample (PPDS), but showed similar effec-
t size and directionality in an independent sample of Marines
with PTSD (Marine Resiliency Study).31 This association is small
in magnitude and, even if replicated, would be of no obvious
clinical utility at present. Its value may lie, however, in event-
ual elucidation of the nature of PTSD and its association with
other illnesses. The finding of pleiotropy between PTSD and
rheumatoid arthritis and psoriasis should further motivate the
study of immune-related factors in PTSD, their potential con-
tribution to comorbidity with inflammatory disorders, and a
possible role for anti-inflammatory treatments in PTSD.88

ARTICLE INFORMATION

Correction: This article was corrected on July 6, 2016, to fix 2 typographical errors in the Abstract.

Submitted for Publication: October 10, 2015; final revision received February 6, 2016; accepted February 9, 2016.


Author Affiliations: Department of Psychiatry, University of California, San Diego (UCSD), La Jolla (Stein, Malhofer, Nievuerget, Thomas); Department of Family Medicine and Public Health, UCSD, La Jolla (Stein, Jain, Sun); Psychiatry Service, Veterans Affairs San Diego Healthcare System, San Diego, California (Stein); Department of Psychiatry, Massachusetts General Hospital and Harvard Medical School, Boston (Chen, Smoller); Stanley Center for Psychiatric Research, Broad Institute of Massachusetts Institute of Technology and Harvard, Cambridge (Chen, Neale, Ripke, Smoller); Department of Psychiatry, Uniformed Services University of the Health Sciences, Bethesda, Maryland (Ursano); Harvard T. H. Chan School of Public Health, Boston, Massachusetts (Cai); Department of Psychiatry, Yale University, New Haven, Connecticut (Gelernter, Jensen, Polimanti, Wang); Department of Genetics, Yale University, New Haven, Connecticut (Gelernter, Polimanti, Wang); Department of Neurology, Yale University, New Haven, Connecticut (Gelernter, Polimanti, Wang); Institute for Social Research, University of Michigan, Ann Arbor (Heeringa, Mitchell, Ware); Department of Psychology, Harvard University, Cambridge, Massachusetts (Nock); currently with Department of Computational Biology and Bioinformatics, Graduate School of Arts and Sciences, Yale University, New Haven, Connecticut (Wang); National Institute of Mental Health, National Institutes of Health, Bethesda, Maryland (Borja); Department of Health Care Policy, Harvard Medical School, Boston, Massachusetts (Kessler).

Author Contributions: Dr Stein had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Stein, Chen, Ursano, Gelernter, Heeringa, Jain, Mitchell, Nock, Borja, Kessler, Smoller.

Acquisition, analysis, or interpretation of data: Stein, Chen, Ursano, Gelernter, Heeringa, Jain, Jensen, Malhofer, Mitchell, Nievuerget, Neale, Polimanti, Ripke, Sun, Thomas, Wang, Ware, Borja, Kessler, Smoller.

Drafting of the manuscript: Stein, Chen, Ursano, Mitchell, Nievuerget, Wang, Smoller.

Critical revision of the manuscript for important intellectual content: Stein, Chen, Ursano, Cai, Gelernter, Heeringa, Jain, Jensen, Malhofer, Mitchell, Nievuerget, Neale, Polimanti, Ripke, Sun, Thomas, Wang, Ware, Borja, Kessler, Smoller.

Copyright 2016 American Medical Association. All rights reserved.
Mitchell, Niev erget, Nock, Neale, Polimanti, Ripke, Sun, Thomas, Ware, Borja, Kessler, Smoller. 

Streefland, Ninne, Chen, Cai, Heeringa, Jain, Malhofer, Mitchell, Niev erget, Neale, Polimanti, Ripke, Sun, Wang, Ware, Kessler, Smoller. 

Obtained funding: Stein, Ursano, Geier ler, Heeringa, Nock, Kessler. 

Administrative, technical, or material support: Ursano, Heeringa, Mitchell, Thomas, Borja, Kessler. 

Study supervision: Ursano, Ger lenthal, Mitchell, Niev erget, Smoller. 

Conflict of Interest Disclosures: Dr Stein reports serving as a consultant for Healthcare Management Technologies, Actelion, Dart Neuroscience, Janssen, Oxeia Biopharmaceuticals, Pfizer, Resilience Therapeutics, and Toxi Pharmaceuticals in the last 3 years. Dr Kessler reports serving as a consultant for Hoffman-La Roche, Inc., Johnson & Johnson Wellness and Prevention, and Sanofi Groupe in the last 3 years; serving on advisory boards for Mensante Corporation, Plus One Health Management, Lake Nona Institute, and US Preventive Medicine; and ownership of a 25% stake in Data Science, Inc. Dr Smoller reports serving as an unpaid member of the Scientific Advisory Board of PsyBrain, Inc. No other disclosures were reported. 

Funding/Support: This study was sponsored by the Department of the Army and supported by cooperative agreement U01MH087981 from the US Department of Health and Human Services, National Institutes of Health, National Institute of Mental Health (NIMH) (Dr Stein and Ursano). The GPA analyses were supported by grant ROI DA12690 from the National Institute of Drug Abuse (Dr Geier ler). 

Role of the Funder/Sponsor: The funding sources had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication. 

Group Information: The Army STARRS Collaborative Team consists of co principal investigators Robert J. Ursano, MD (Uniformed Services University of the Health Sciences) and Murray B. Stein, MD, MPH (University of California, San Diego [UCSD]) and Veterans Affairs San Diego Healthcare System; site principal investigators Steven G. Heeringa, PhD (University of Michigan) and Ronald C. Kessler, PhD (Harvard Medical School); NIMH collaborating scientists Lisa J. Colpe, PhD, MPH, and Michael Schoenbaum, PhD; Army liaisons/consultants COL Steven Cersovsky, MD (Uniformed Services University of the Health Sciences), and Kenneth Cox, MD, MPH (USAPHC). Other team members include Pablo A. Allia, MA (Uniformed Services University of the Health Sciences), David M. Benedek, MD (Uniformed Services University of the Health Sciences), K. Nikkil Benevides, MA (Uniformed Services University of the Health Sciences), Laura Campbell-Sills, PhD (UCSD), Tianni Cai, ScD (Harvard T. Chan School of Public Health), Chia-Yen Chen, ScD (Massachusetts General Hospital), Catherine L. Dempsey, MD, MPH (Uniformed Services University of the Health Sciences), Julie O. Denenberg, MA (UCSD), Carol S. Fullerton, PhD (Uniformed Services University of the Health Sciences), Nancy Gebler, MA (University of Maryland) (Uniformed Services University of the Health Sciences), Robert K. Gifford, PhD (Uniformed Services University of the Health Sciences), Stephen E. Gilman, ScD (Harvard School of Public Health), Feng He, MS (UCSD), Marjan G. Holloway, PhD (Uniformed Services University of the Health Sciences), Paul E. Hurwitz, MPH (Uniformed Services University of the Health Sciences), Sonia Jain, PhD (UCSD), Tzu-Cheg Kao, PhD (Uniformed Services University of the Health Sciences), Karestan C. Koenen, PhD (Columbia University), Kevin P. Jensen, PhD (Yale University), Lisa Lewandowski-Romps, PhD (University of Michigan), Holly Herberman Mash, PhD (Uniformed Services University of the Health Sciences), Adam X. Malhofer, MS, UCSD), James E. McCarroll, PhD, MPH (Uniformed Services University of the Health Sciences), James A. Naifeh, PhD (Uniformed Services University of the Health Sciences), Matthew K. Nock, PhD (Harvard University), Rema Raman, PhD, UCSD), Holly J. Ramsay, PhD (Uniformed Services University of the Health Sciences), Anthony Joseph Roselli, PhD (Harvard Medical School), Nancy A. Sampson, BA (Harvard Medical School), LCRD Patcho Santiago, MD, MPH (Uniformed Services University of the Health Sciences), Michellea Scanlon, MBA (NIMH), Jordan W. Smoller, MD, ScD (Massachusetts General Hospital), Army Street, PhD (Boston University School of Medicine), Xiaoying Sun, MS (UCSD), Michael L. Thomas, PhD (UCSD), Pitti L. Veglia, MS, MA, Uniformed Services University of the Health Sciences), Lerning Wang, MS (Uniformed Services University of the Health Sciences), Erin B. Ware, PhD (University of Michigan), Christina L. Wassel, PhD (University of Pittsburgh), Simon Wessely, FMedSci (King’s College London), Hongyan Wu, MPH (Uniformed Services University of the Health Sciences), LTC Gary H. Wynn, MD (Uniformed Services University of the Health Sciences), Alan M. Zaslavsky, PhD (Harvard Medical School), Bailey G. Zhang, MS (Uniformed Services University of the Health Sciences), and Lei Zhang, PhD (Uniformed Services University of the Health Sciences). 

Disclaimer: The contents are solely the responsibility of the authors and do not necessarily represent the views of the Department of Health and Human Services, NIMH, the Department of Veterans Affairs, the Department of the Army, or the Department of Defense. 

REFERENCES: 


