RESEARCH LETTER

Recent Trends in the Prevalence of Marijuana Use and Associated Disorders in the United States

Using data from 2 waves of the National Epidemiologic Survey of Alcohol and Related Conditions (NESARC), Hasin and colleagues1 reported that the past-year prevalence of marijuana use in the United States has more than doubled in a decade (2002-2003 to 2012-2013), along with a comparable increase in the past-year prevalence of marijuana use disorder as defined by the DSM-IV. Because a previous report documented large discrepancies between drug use estimates from the NESARC and those provided by the National Survey on Drug Use and Health (NSDUH),2 we sought to directly compare prevalence estimates and trends in marijuana use and marijuana use disorder from the NSDUH those from the NESARC.

Methods| Between October 22 and November 14, 2015, we analyzed data from the adult samples (aged ≥18 years) of the 2002-2013 NSDUH (N = 451160), which is an annual survey representative of the household-dwelling population of the United States and is a primary source of information about prevalence and trends in drug use. Similar methods have been used annually since 2002, with typical response rates around 75%.2-4 Trends were analyzed using the surveylogistic procedure in SAS, version 9.4 (SAS Institute, Inc), modeling each outcome variable as a function of year of assessment. Procedures were approved by the Washington University Human Research Protection Office.

Results| Annual past-year prevalences of marijuana use, marijuana use disorder as defined by the DSM-IV, and marijuana use disorder among past-year users (conditional prevalence) are plotted in the Figure. Although the trends are not necessarily linear, we fitted each series to a trend line to evaluate the mean annual change between 2002 and 2013. The trend for prevalence of marijuana use was positive and significant (β = 0.021; 95% CI, 0.017-0.025; P < .001), reflecting a relative increase of 19%. The trend for prevalence of marijuana use disorder was flat (β = 0.006; 95% CI, -0.015 to 0.003; P = .23). There was a net decrease in the conditional prevalence of marijuana use disorder (β = -0.018; 95% CI, -0.027 to -0.009; P < .001). The Table lists prevalence estimates for each out-

Table. Past-Year Prevalence Estimates of Marijuana Use, Marijuana Use Disorder, and Marijuana Use Disorder Among Past-Year Marijuana Users

<table>
<thead>
<tr>
<th>Source*</th>
<th>Prevalence (95% CI)b</th>
<th>Marijuana Use</th>
<th>Marijuana Use Disorder</th>
<th>Marijuana Use Disorder Among Past-Year Users</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSDUH</td>
<td></td>
<td></td>
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<tr>
<td>2002</td>
<td>10.5 (9.9 to 11.0)</td>
<td>1.6 (1.4 to 1.8)</td>
<td>14.9 (13.5 to 16.4)</td>
<td></td>
</tr>
<tr>
<td>2013</td>
<td>12.5 (12.0 to 13.0)</td>
<td>1.5 (1.3 to 1.6)</td>
<td>11.6 (10.4 to 12.8)</td>
<td></td>
</tr>
<tr>
<td>Relative change, %</td>
<td>19 (12 to 22)</td>
<td>-8 (~22 to 8)</td>
<td>~23 (~13 to ~35)</td>
<td></td>
</tr>
<tr>
<td>NESARC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2001-2002 (wave 1)</td>
<td>4.1 (3.8 to 4.4)</td>
<td>1.5 (1.3 to 1.7)</td>
<td>35.6 (32.8 to 38.4)</td>
<td></td>
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<tr>
<td>2012-2013 (wave 3)</td>
<td>9.5 (9.0 to 10.0)</td>
<td>2.9 (2.6 to 3.2)</td>
<td>30.6 (28.5 to 32.7)</td>
<td></td>
</tr>
<tr>
<td>Relative change, %</td>
<td>131 (117 to 146)</td>
<td>93 (73 to 113)</td>
<td>~14 (~5 to ~23)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: NESARC, National Epidemiologic Survey of Alcohol and Related Conditions; NSDUH, National Survey on Drug Use and Health.

* Prevalence estimates from the NSDUH were estimated from public-use files using Estimates from the NESARC are from Hasin et al.1

b 95% CIs for relative change estimates were calculated from pooled standard errors.
come from the NSDUH for the years 2002 and 2013, along with comparable results from the NESARC.\textsuperscript{1}

**Discussion** | In contrast with results from 2 waves of the NESARC covering roughly the same period, estimates from the NSDUH suggest a more modest increase in marijuana use and no increase in the prevalence of marijuana use disorder. It is well known that individuals underreport socially proscribed behaviors in face-to-face interviews such as those used by the NESARC.\textsuperscript{5} In contrast, the NSDUH uses an interview that is self-administered via a computer as well as other methods known to enhance privacy and reduce underreporting of socially proscribed behaviors.\textsuperscript{2,3,5} Accordingly, a previous comparison of estimates of drug use prevalence from the NSDUH and NESARC (wave I) showed that NSDUH estimates were 2 to 5 times higher for all drugs.\textsuperscript{2} Because use of marijuana has become more socially acceptable, people may now be more willing to disclose its use to an interviewer than they were in 2002. This change likely led to a partial closing of the gap in the reported prevalence of use between the NSDUH and NESARC (Table).

If marijuana use was underreported in the NESARC, it likely led to bias in the prevalence estimate of marijuana use disorder because only those who report past-year use are assessed for marijuana use disorder. If underreporting was more prevalent in the first wave of the NESARC than in the most recent wave (wave 3), it could explain the apparent increase in the prevalence of marijuana use disorder. Trends in the past-year conditional prevalence of marijuana use disorder were similar across surveys, although estimates from the NESARC were substantially higher, suggesting that its diagnostic assessment is more sensitive than the one used in the NSDUH.\textsuperscript{1,2}

There were also methodological differences between the 2 NESARC waves. For example, in wave I, interviews were conducted by US Census Bureau employees, whereas wave 3 used a private company.\textsuperscript{1,2} It is possible that individuals are less likely to disclose illegal behaviors to government employees than to private sector interviewers.

In summary, changes in the social acceptability of marijuana use and methodological changes likely account for much of the apparent doubling in prevalence of marijuana use and disorders reported by Hasin and colleagues.\textsuperscript{1}

Richard A. Grucza, PhD
Arpana Agrawal, PhD
Melissa J. Krauss, MPH
Patricia A. Cavazos-Rehg, PhD
Laura J. Bierut, MD

**Author Affiliations:** Department of Psychiatry, Washington University School of Medicine, St Louis, Missouri.

**Corresponding Author:** Richard A. Grucza, PhD, Department of Psychiatry, Washington University School of Medicine, 660 S Euclid Ave, PO Box 8134, St Louis, MO 63110 (gruczar@psychiatry.wustl.edu).

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**Author Contributions:** Dr Grucza 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:** Grucza, Agrawal, Bierut.

**Acquisition, analysis, or interpretation of data:** Agrawal, Krauss, Cavazos-Rehg.

**Drafting of the manuscript:** Grucza, Cavazos-Rehg.

**Critical revision of the manuscript for important intellectual content:** Grucza, Agrawal, Krauss, Bierut.

**Statistical analysis:** Grucza, Agrawal.

**Obtained funding:** Grucza, Cavazos-Rehg.

**Administrative, technical, or material support:** Grucza, Krauss.

**Study supervision:** Grucza.

**Conflict of Interest Disclosures:** Dr Bierut is listed as an inventor on issued US Patent 8,080,371, “Markers for Addiction,” covering the use of certain single-nucleotide polymorphisms in determining the diagnosis, prognosis, and treatment of addiction. No other disclosures were reported.

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**COMMENT & RESPONSE**

**One Effect Size Does Not Fit All—Is the Depression-Inflammation Link Missing in Racial/Ethnic Minority Individuals?**

**To the Editor** | In their analysis of the National Health and Nutrition Survey (NHANES), Jokela and colleagues\textsuperscript{1} address a timely question: Is systemic inflammation more strongly associated with particular depressive symptoms? While C-reactive protein (CRP) was positively associated with all 9 depressive symptoms in separate models, it was associated with only sleep disturbance, fatigue, and appetite changes in models adjusting for the other 8 symptoms. Because these associations are likely bidirectional,\textsuperscript{2,3} this line of research has the potential to (1) clarify the pathophysiology of inflammation-related depression and inform the development of new depression interventions targeting inflammation and (2) elucidate the mechanisms through which depression increases cardiometabolic disease risk and inform the development of new primary prevention approaches.

We also examined associations between CRP and depressive symptoms in the study by Case and Stewart\textsuperscript{4} using the same NHANES data (2005-2010) and, not surprisingly, found nearly identical results. Of note, we excluded more NHANES participants than Jokela et al\textsuperscript{5} to minimize the possibility of

Agrawal, Krauss, Cavazos-Rehg.