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Area-level socioeconomic status in relation to outcomes in γ -hydroxybutyrate intoxication

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Abstract

Background—Area-level socioeconomic status (SES) may play an important role in drug abuse patterns, including related health outcomes. This may be particularly relevant for γ -hydroxybutyrate (GHB), which is prototypical of “party” drug abuse.

Methods—We retrospectively reviewed GHB-related cases reported to the California Poison Control System (CPCS; January 1, 1999 through June 30, 2007). We limited analysis to CPCS calls containing a residential zip code (ZC). The CPCS data were extracted for key case characteristics, including the residential ZC. We linked cases to corresponding 2000 U.S. Census data for area-level measures of SES and demographics. We used multiple logistic regression analysis to test the associations between area-level SES and GHB case severity, taking into account area-level demographics and individual-level GHB high-risk behaviors.

Results—We analyzed 210 cases. Taking into account area-level demographics (age and racial mix; urbanicity) and GHB-related high-risk behaviors (use of GHB congeners; GHB-dependence; co-ingestion of other agents), we associated higher area-level SES with greater GHB case severity. There was 40% increased likelihood of major GHB adverse health outcomes for every \$100,000 incremental increase in median home values (OR 1.41; 95% CI 1.1–1.8). For median annual household income (per \$10,000), the association was similar (OR 1.39; 95% CI 1.0–1.9).

Conclusion—Higher area-level SES is associated with greater GHB-related case severity. This study may serve as a model using a geographic information system (GIS) approach to study the population-based correlates of drugs of abuse reported through poison control surveillance.

Keywords

Intoxication; Demographic data; γ -Butyrolactone; γ -Hydroxybutyrate; Geocoding

Background

Socioeconomic status (SES) is a major determinant of health-related practices and outcomes. Moreover, SES effects operate at two levels: an individual's SES (e.g., personal or family income and assets) and the local neighborhood or "area" level SES (1). Area-level SES refers to the characteristics of the local population in which the individual lives, including measures of poverty status, employment patterns, and the quality or value of housing stock.

Although there has been increasing attention to the relationship between area-level SES and health status for a number of acute and chronic conditions, this relationship has not been well studied for poisoning and drug use. In particular, such analyses may reveal important area-level SES correlates of illicit drug use. Area-level SES direct measures, including household income and home values, may be estimated through census data and then linked to geocoded poison control data.

γ -Hydroxybutyrate (GHB) is an illicitly used drug in which identifying area-level SES associations may be particularly important in targeting prevention efforts by identifying populations at increased risk of morbidity and mortality. GHB and related GHB congeners (GHBP) are identified as "club drugs" by the National Institute on Drug Abuse (NIDA) (2). Our previous research has shown that GHB is also used in non-club group settings, with a particular niche among persons employed and relatively well educated (3). Prevention is important because serious GHB-related health effects (agitation, bradycardia, respiratory depression, coma, drug dependence) and fatal outcomes are well recognized (4-6). Outcome severity is also associated with GHB use combined with alcohol or other drugs (7). The health effects of GHB and GHBP are similar (6).

Recently, GHB abuse in the United States appears to have declined (4). Nonetheless, GHB abuse remains popular in some sectors and drug abuse trends are often cyclical (8). Moreover, internationally the use may be increasing (9). Thus, GHB poisoning, including toxicity due to drug dependence and to surreptitious administration, remains an ongoing concern (10-14).

Poison control center (PCC) reporting is a major source of population-based data on drug abuse in general and GHB in particular, yet such data have not been used to address area-level SES linkages. Such surveillance data have been utilized to examine broad geographic patterns in U.S. drug abuse. For example, PCC data were analyzed from eight geographically dispersed areas in the United States (state or regional level) to identify patterns of opiate abuse (15). Another study of opiate abuse found that the prescription opiate abuse (predominantly oxycodone and hydrocodone) correlated with regional urbanicity (16). Poisoning surveillance data have also been linked to geospatial information to study within state regional clustering of pesticide cases (17). In addition, population-based data have been linked to PCC calls in order to estimate the utilization of services at the county level by ethnicity (18), population density (19), and SES (20,21). None of these approaches, however, have linked PCC surveillance data to area-level U.S. Census data for SES or even demographic data. Area-level linkages to PCC surveillance data are possible because these reports systematically document caller home zip codes (ZCs). Previously, in other disciplines, ZC data have been successfully linked with census demographics for area-level geocoding (22-24). This potential route of geocoding linkage to area-level data is important because individual-level SES data are almost never collected as a part of PCC reporting. In the analysis reported here, we use this approach, testing the hypothesis that area-level SES is associated with GHB intoxication reported through PCC surveillance. Specifically, we postulated that key case attributes, in particular, outcomes, would vary systematically in relation to area-level SES. Delineating such associations may be important for targeting prevention strategies for this specific drug. Moreover, this novel

approach to PCC data geocoding linkages could serve as a model to be applied to other research questions.

Methods

Poison center case record collection

We conducted a retrospective case record review analyzing all suspected exposure cases involving GHB or GHB congeners (e.g., γ -butyrolactone and 1,4 butane-diol) reported to the California Poison Control System (CPCS) over an 8.5-year period (January 1, 1999 through July 1, 2007). The CPCS is a 24-h emergency telephone consultation service offering consultation to the public and health care professionals. The CPCS staff systematically records consultation data in accordance with American Association of Poison Control Centers (AAPCCs) guidelines. The CPCS utilizes a standardized electronic case record documentation system based on numeric or closed-ended data entry, but that also allows for an open-ended text field describing the consultation. Data include demographics, caller site with ZC and city, product identification, patient disposition, and case severity outcome. Although open-ended text fields technically could allow for the entry of SES data or surrogates of SES status, such information is almost never recorded on a routine basis. This study was approved by the Committee on Human Research, University of California, San Francisco as well as by the CPCS's internal oversight Research Committee.

Case records were identified for evaluation by electronically searching the CPCS database for all case records coded as exposure and containing any of the AAPCC generic or product codes for GHB, GHB-related congeners, and GHB withdrawal. Prior to 2004, GHB cases were identified utilizing a more intensive search algorithm because, until that date, unique AAPCC codes for GHB and its congeners were not yet established or routinely implemented. This method of case identification has been previously described in detail (4). Each case record was reviewed for date, age, gender, product, drug co-ingestants, "reason" for exposure, caller site location, clinical course, case disposition, outcome, ZC and city of the case, and the caller (who could be a medical care provider, the patient, or a third party). Residential ZCs were verified during record review to ensure the ZC on the record originated from a residential site or was specifically entered into the open-ended text field. ZCs were included in this analysis when 1) the caller site was a non-health-care facility (non-HCF) (e.g., residence) and the patient ZC field was filled-in whether the case was referred to an HCF with subsequent follow-up care available or not or 2) the text field of the record specifically included the patient's home ZC even though the call originated from an HCF.

An approximate 50% of the sample (98/210 cases) were secondarily reviewed by two of three reviewers (IA, SK, JD) for accuracy and consistency. Case severity outcomes were coded ordinarily according to the AAPCC definitions (25) as follows (including typical GHB-related clinical case findings): 1) no effect; 2) minimal effect (self-limiting minor symptoms that resolved rapidly including lethargy, vomiting, or sinus tachycardia); 3) moderate effect (systemic symptoms that are not life threatening, including hallucinations, single seizure, agitation, myoclonus, and confusion); and 4) major effect (life-threatening symptoms, including cardiovascular or respiratory compromise or multiple seizures).

Cases were designated as ineligible for study inclusion for the following reasons: 1) duplicate case records with separate numeric identification identified by manual review of case content; 2) non-GHB case record based on manual review; 3) informational case only (no actual exposure occurred); 4) non-abuse related exposure (e.g., ocular or dermal exposure, suicidal attempt, adverse reaction); 5) HCF case records with unknown patient ZC; 6) case records with no personal ZC documented; and 7) cases telephoned in to the CPCS from outside of California. We also excluded potentially eligible cases based on the following criteria: 1) case symptoms

and clinical scenario wholly inconsistent with GHB, indicating an initially inappropriate exposure assignment; 2) cases involving purported surreptitious poisoning without GHB laboratory confirmation; and 3) case records with a post office box address and its ZC only (Table 1). The CPCS staff collects the ZC of origin from all non-HCF callers. In situations where the city of origin is obtained rather than the ZC, a computer program generates a default ZC based on city and telephone prefix of the caller (when available). All case records were evaluated to determine whether the ZC was “unique” or, alternatively, could potentially have been generated automatically through computer algorithm. After all ineligible cases and exclusions were removed, 132 of 210 (67%) ZCs were determined to not fall within the potential default assignment algorithm. It was not possible to determine whether the remainder (78) of the ZCs were the callers' actual ZCs or computer-generated ZCs corresponding to the case's locale (and telephone exchange), but not necessarily to the precise ZC of residence. Of these 78 ZCs, 37 appeared only once, 9 ZCs were repeated once, 4 ZCs were repeated twice, 1 ZC was repeated 5 times, and 1 ZC was repeated 8 times in the data set.

Area-level measures derived from 2000 census data

The 2000 U.S. Census data were linked to CPCS caller ZCs utilizing ZIP*Data (CENSUS.DAT) from the Melissa database (26). ZIP*Data links the geographic latitude and longitude coordinates at the geographic center of the corresponding U.S. postal service five-digit ZCs to U.S. 2000 census blockgroup data for selected variables. Based on ZCs, we linked the CPCS case records to five variables from the 2000 census data that represented either area-level measures of SES or key demographic variables. The two area-level SES census data variables we analyzed were median annual household income and median home value. The demographic variables were urbanicity (percent population within the ZC that was non-urban defined by standard census criteria), adult proportion of all those above childhood (persons 30 years of age or more as a percentage of all persons 15 years of age and older within the entire population of the ZC; those less than 15 years of age were excluded because this age group would be unlikely to include any GHB users), and race (percent of non-White population).

Outcomes derived from the CPCS GHB case data set

Individual-level demographic characteristics derived from the CPCS case records were dichotomous based on age (>30 years vs. all others) and gender (female vs. male). We also extracted data related to four GHB-related high-risk behaviors: context of GHB use (collapsed to GHB dependence vs. all other scenarios), product involved (GHB vs. GHB congener), co-ingestion of GHB with ethanol, and co-ingestion of GHB with other agents [these included amphetamines, benzodiazepines, cocaine, marijuana, methylenedioxymethamphetamine (MDMA), opiates, ketamine, thyroid, and selective serotonin reuptake inhibitors (SSRIs)]. These four GHB-associated risky behaviors were selected on an *a priori* basis, because of data indicating their relevance to GHB outcomes. Specifically, our previous analysis of CPCS data revealed an increased risk of GHB hospital treatment associated with use of GHB to treat withdrawal symptoms (consistent with GHB dependence), co-ingestion of GHB with ethanol, and co-ingestion of GHB with ketamine (27). We also wished to include a measure of GHB dependence because of increasing reports of such dependence and withdrawal (28) and because GHB dependence has been associated with lengthier hospitalization (4). In 2000, GHB access became more restricted following its schedule I listing, a change temporally tied to increased GHB congener use. Given the emergence of GHB congeners, we were also particularly interested in evaluating their potential association with SES. Lastly, we extracted subject treatment disposition [non-HCF (e.g., treated at home) vs. HCF treatment (e.g., an emergency department)] and case outcome as coded according to standard AAPCC criteria (major effect vs. all others). The latter two variables comprised the ultimate key outcome variables of study interest.

Data analysis

In order to assess the generalizability of our observations to HCF-treated GHB overdose cases, we used the χ^2 to test differences in the frequencies for key variables comparing those included in the final analysis with HCF cases without residential ZCs ($n = 873$).

We used logistic regression analysis to test the independent associations among area-level demographic and SES variables derived from geocoding links to U.S. census data and two different levels of dependent variables. We did this in order to tease out the potential role of area-level SES on GHB-related health outcomes, independent of potential confounding demographic factors and potential GHB-associated behaviors that could be driven by upstream SES factors that mediate downstream health outcomes. Our underlying theoretical model presumes that SES is associated with both certain high-risk behaviors and GHB-related health outcomes. We did not have, however, an *a priori* assumption as to whether the SES link to health outcomes would be largely or only minimally mediated by the behaviors we were modeling.

Consistent with this hierarchical model, the first level of dependent outcomes we tested with logistic regression analysis comprised the four GHB-associated high-risk behaviors described previously. We tested these as dependent dichotomous outcomes in relation to each of the five area-level variables in simple univariate models. Thus, in this step of the analysis we tested 20 separate models.

The second level of outcomes comprised two outcomes: “major” effects according to AAPCC criteria and treatment for GHB in an HCF. Because the latter required follow-up confirmation that such treatment took place, we limited that analysis to the subset of these data ($n = 111$) within the entire data set ($n = 210$). To test area-level SES associations with these outcomes, we separately modeled median annual income and median home values as SES measures. These could not be combined as independent predictors in the same model due to their collinearity. In the first level of multivariate modeling, we included area-level demographics (age distribution, race, and urbanicity) in order to take into account these factors as potential confounding variables. In the second, more complex multivariate model, we further added the three high-risk behaviors that had shown a relationship to SES in the previous stage of the analysis. For HCF treatment, we further tested a third model, adding “major” GHB case outcome status as a predictor. The rationale for this model was to examine whether case severity accounted for HCF care or whether an independent SES effect remained, for example, due to ability to pay for medical care.

We tested the effect of possible misclassification due to ZCs assigned by default CPCS procedures by re-running the key multiple logistic regression models excluding all subjects with any potential for having been assigned a default ZC. These sensitivity analyses were limited to 132 of 210 subjects for the re-analysis of SES in relation to major GHB outcomes and 75 of 111 subjects with both no potential default ZC and complete follow-up for the analysis SES in relation to HCF treatment. SAS 9.1 (SAS Institute, Cary, SC, USA) was used for all analyses.

Results

Table 1 summarizes case record identification, eligibility, and exclusions. A total of 3,735 cases were ineligible by study criteria of GHB exposure cases. Another 486 were excluded, which comprised 335 cases that purported surreptitious GHB exposure that could not be confirmed and 41 cases with ZCs for post office boxes rather than an actual residence. Ultimately, 210 exposure case records were included in the analysis. Total CPCS human

exposure calls during the 8.5-year study period were 1,894,767 and did not vary substantively year to year within that period.

Subject descriptive data

The dependent variables from the CPCS database describing the 210 subjects included in the final analysis are summarized in Table 2. Sixty-seven percent of the analyzed subjects were males, and 40% were over 30 years of age, consistent with our previous findings revealing the general pattern of GHB use among a predominantly male population with this age distribution (4). Overall, less than one in four subjects reported GHB dependence (18%), any use of GHB congeners (13%), co-ingestion at any time of GHB with other agents (16%), and co-ingestion of GHB with ethanol alone (20%). Polysubstance use was reported by history in 70 cases, 43 of which reflected ethanol as the single additional substance used. The remainder involved “other agents” including amphetamines (14), benzodiazepines (9), cocaine (6), marijuana (4), MDMA (3), opiates (2), ketamine (1), thyroid (1), and SSRIs (1). There were 134 subjects (64%) who had contacted the CPCS from a non-HCF and who were given advice from the CPCS to seek evaluation at an HCF. Follow-up information for subjects was available for 111 subjects, confirming health care center evaluation among one in three. Overall, 19 (9.1%) outcomes were classified as “major” according to AAPCC criteria. There were no deaths among the cases analyzed.

We compared the demographics, GHB-associated high-risk behaviors, and GHB outcome severity between the subjects we analyzed ($n = 210$) and the HCF cases that were excluded due to lack of a residential ZC ($n = 873$). The gender mix was similar in those studied compared with the excluded cases (33% compared to 32% female, respectively; $p > 0.60$), but those studied were older [40% compared with 27% over 30 years of age, respectively ($p < 0.001$)]. The use of a GHB congener was more common in the HCF excluded group (203 [23%]) than in the study group (13%) as shown in Table 2 ($p = 0.001$). Compared with the study subjects (Table 2), the frequencies of other GHB-associated high-risk behaviors among the excluded HCF cases did not differ statistically ($p \geq 0.10$): dependence use, 14%; co-ingestions other than ethanol, 17%; and ethanol co-ingestion, 16%. Consistent with treatment in an HCF, the frequency of major outcomes in that group (17%) was statistically higher than the frequency in the study group (9%; $p = 0.004$); there were also three deaths among the 873 HCF-treated, excluded cases.

Independent census SES predictive variables

Table 3 presents descriptive statistics for the area-level SES measures derived from geocoding linkages to 2000 U.S. census data. As reflected in these area-level data, the subjects were predominantly urban. Although the proportional age mix fell within a fairly narrow range, there was a wide range in racial diversity. For area-level SES variables, median annual household incomes and home values varied approximately by a two-fold factor ranging from the 25th to 75th percentile about the median value.

SES census variables predicting risky behaviors

Table 4 presents univariate models testing the associations between area-level demographics and SES and each of the four high-risk GHB-associated behaviors studied: GHB dependency, GHB congener use, co-ingestion of GHB with other agents, and co-ingestion of GHB with ethanol. The most consistent finding was for area-level SES. Median annual household income was significantly associated with three of the four high-risk behaviors: GHB dependence ($p < 0.001$), GHB congener use ($p = 0.03$), and co-ingestion of GHB with other agents ($p = 0.004$), all in the direction of higher SES being associated with increased likelihood of high-risk behaviors. The direction of association was also similar for co-ingestion of alcohol, although this was not statistically significant. Area-level SES measured by median home values was

also associated with co-ingestion of GHB together with other agents ($p = 0.01$). An older population demographic mix, as reflected in the percentage of those 30 years or older among all adults (15 years and over), was strongly associated with GHB dependence ($p < 0.001$) and GHB congener use ($p = 0.02$). There were no statistical associations between urbanicity or ethnicity and any of the four GHB-associated high-risk behaviors.

Predictors of major outcomes and health care facility treatment

Using multivariate logistic regression, we tested the association between area-level SES, as measured by median household income and home values, and major outcomes (19 such outcomes among 210 subjects). As shown in Table 5, area-level SES was associated with an increased likelihood of poison center-assessed major (severe) outcomes following GHB use. This was true both for annual household income and for home values as measures of SES and remained so after taking into account area-level urbanicity, age, and racial mix.

In order to test whether the SES association with case severity was mediated through selected high-risk behaviors (because three of these were related to SES, as shown in Table 4), we retested the multivariate models including GHB dependency, co-ingestion, and congener use (Model 2, as indicated). The estimated associations between SES and severe GHB outcomes were only minimally impacted by inclusion of these potential mediators.

We also tested the associations between area-level SES and HCF treatment for GHB, limiting this analysis to the 111 subjects for whom final disposition had been confirmed (Table 6). Including demographic variables in the multivariate model, we associated area-level SES with increased likelihood of HCF treatment for GHB (home value $p = 0.04$; income $p = 0.07$). Adding GHB-associated high-risk behaviors reduced the risk estimates somewhat (only minimally for median home value as a measure of SES), whereas further including case severity reduced the SES effect to a negligible point estimate of increased risk. This final model (Model 3) is consistent with case severity being the mediator for the SES association with HCF treatment for GHB shown in Model 1 and, to a lesser extent, Model 2.

In order to examine the impact of any ZCs that might have been assigned to CPCS cases by default (see Methods), we re-estimated the models testing the associations between SES and major GHB outcomes, limited to the 132 subjects for whom there was no possibility of an assigned ZC. For home value, there was a modest increase in the estimated OR taking into account demographics and GHB-associated high-risk behaviors (Model 2): OR 1.61 (95% CI 1.2–2.2; $p = 0.01$). A similar pattern was evident for annual household income, although the re-estimated 95% CI did include 1.0: OR 1.53 (0.96–2.4; $p = 0.07$) (data not shown in tables). A similar analysis of SES as a predictor of HCF treatment, limited to 75 subjects with both confirmed follow-up status and no possibility of an assigned ZC, also reconfirmed the association between median home value and this outcome, taking into account demographics and GHB-associated risk behavior: OR 1.43 (95% CI 1.02–2.0; $p = 0.04$). As in the main analysis shown in Table 6 ($n = 111$), this association was accounted for by case severity, with the estimated OR falling to 1.0 after addition of severe outcome to the model.

Discussion

Using a geocoding approach, we found that higher area-level SES was associated with certain GHB-associated risk behaviors (notably, GHB dependence and GHB congener use) and, independent of these high-risk behaviors, greater severity in GHB outcomes. Although SES was also associated with GHB-related evaluation in an HCF, this appeared to be driven by case severity, rather than other SES effects, such as SES-mediated access to health care. Put in monetary terms, for every \$10,000 incremental increase in median household income or for

every \$100,000 incremental increase in median home values, at the area level, there was a 40% increased risk of a severe GHB-related outcome reflected in PCC center surveillance data.

GHB is not the first drug to be associated with higher SES. In the early 1980s cocaine was relatively “pricey” and use was more prevalent among the affluent. Only later, with the introduction of crack as a relatively inexpensive form of cocaine, the use shifted to a lower SES stratum (29). Injectable drugs of abuse are strongly linked to lower SES (30). The older age area-level association with GHB behaviors that we observed also warrants comment and may be interrelated to the SES pattern. Adolescents and younger adults are presumed to be more likely to use a variety of different drugs. Indeed, younger age and SES have been noted to track together in certain patterns of usage. For example, substance abuse and mental health services administration (SAMHSA's) 2003 National Survey on Drug Use and Health reported on the use of club drugs and SES, including MDMA, LSD (D-lysergic acid diethylamide), GHB, ketamine, and flunitrazepam (31). SAMHSA found that youths aged 16–23 years living in lower-income families (<\$20,000/year) were the most likely to have used two or more club drugs in the past year (32). In addition, adolescents aged 16–19 years were most likely to use LSD if their family income was reportedly less than \$40,000 annually, although surprisingly, there was no direct link found between SES and the use of methamphetamine or MDMA (32). In contradistinction to these findings, however, according to another governmental report, most ecstasy users are of middle to upper SES (33). Club drug age-specific use by race ethnicity has also been studied. In New York City, a predominance of club drug use among White adolescents and young adults was identified, although use among Hispanics and Blacks was not uncommon (34). In terms of GHB specifically, others have reported a mixed association with SES and age: middle-class “youth” are reportedly the population most associated with GHB, GHB congeners, and ketamine use (33). This is consistent with our previous qualitative research on GHB, indicating that regularly employed professional adults over 30 years of age are an important SES-demographic stratum among users of this drug (3).

Limitations

It is important to recognize the potential limitations of this study. These cases were collected from California and may not be representative of the rest of the United States. Nonetheless, California does include 12% of the U.S. population (35) with a wide range of SES. Second, we linked 8.5 years of CPCS data with census data obtained in the year 2000. It is possible that neighborhood demographics may have changed during the time frame of this study, an inherent limitation of using decennial census data. However, other area-level studies using census data have used similar time frames (36). In addition, we were not able to include all GHB-related PCC calls, including many originating from an HCF (thus precluding the availability of a residential ZC). The excluded HCF cases were younger, more likely to have reported GHB congener use, and, not surprisingly, manifested greater severity. Of note, however, excluding HCF cases did not appear to have a selection effect in terms of gender, dependence use, and co-ingestion frequencies. Although we did not find a strong link between GHB use and population density, the heavily urban characteristics of areas from which the cases originated make it difficult to study such as association in this cohort. Also of note, we studied only two measures of SES, income and home values. Another more salient study limitation is potential imprecision in the geocoding linkages to census data. We faced two principal challenges in this regard. First, CPCS protocols allowed for the possibility of default ZC assignment in some cases. This would have occurred if a subject lived in an area with multiple ZCs (e.g., an urban center), declined to provide a ZC, and was assigned a ZC in that city that was not, in fact, the correct ZC of the caller. We addressed this possible misclassification through reanalysis excluding all subjects in whom this was possible. Importantly, these sensitivity analyses did not appreciably change our findings. The second source of imprecision is that of ZC-based geocoding linkages. ZC boundaries do not coincide exactly with census blockgroup data and,

when linked, may lead to potential biases. In addition, despite increasing use of geocoding technology (37), the best method to institute regarding the assignment of socio-demographic measures for analyzing social disparities is debatable (37-39). Nonetheless, it is generally accepted that linkages based on precise street address, either to census tract or to census blockgroup data, are preferable to ZC-based linkages, because the latter demarcation is less homogenous in regard to demographic and SES factors. This is a limitation inherent in using PCC data in which residential addresses are not captured. The net effect of these sources of imprecision, however, would have been to bias our findings toward the null. Thus, it is unlikely that the strong and consistent SES associations that we did observe were, in fact, attributable to this potential limitation.

Conclusion

Our study uses geographic information system (GIS) methodology in an innovative way to link drug abuse reports received by a statewide PCC with U.S. census data in order to determine associations of drug abuse incidence with surrounding area-level SES variables. PCC routinely collect ZC data for cases called-in from home (as opposed to an HCF), allowing for identification of the location at which these exposures occur. However, PCC do not routinely collect SES information. Using the ZC data, our study was able to link GHB exposures with area-level socio-demographic data using a geocoding information technology approach. Our study highlights the potential uses of PCC surveillance data through GIS linkages in order to analyze population-based correlates of poisoning and drug overdose. In conclusion, we identified a strong association between higher area-level SES and GHB-associated outcomes. These findings may help target future prevention strategies geared to mitigating the adverse health effects associated with GHB abuse.

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Table 1

GHB and congener-related cases reported to the California Poison Control System (CPCS), January 1999–July 2007

Case records by study eligibility status and by exclusion criteria	N	%
Total potential case records identified via key word computerized record search	4,431	100%
<i>Ineligible cases</i>		
Duplicate case records on basis of case record number	1,176	
Duplicate case records eliminated following manual review	34	
Non-GHB case records based on manual review	1,107	
Information case records only (no actual exposure)	484	
Non-abuse related exposure: ocular/dermal (8), suicide (16), adverse reaction (2)	26	
Health care facility case records with unknown patient zip code	873	
Case records with no zip code documented	24	
Non-California case records	11	
Total ineligible cases	3,735	84.3%
Total potentially eligible cases	696	
<i>Excluded cases</i>		
Presentation unrelated to GHB based on review of case record	110	15.8%
Surreptitious poisoning without GHB laboratory confirmation	335	48.1% ^a
Case record containing only a Post Office Box zip code	41	7.0% ^a
Total excluded cases	486	69.8% ^a
Total eligible cases included in this analysis	210	30.2% ^a

^aPercentage calculated as a proportion of eligible cases.

Table 2

Individual-level characteristics derived from California Poison Control System (CPCS) case records

	Frequency (%)
All subjects	210 (100)
Demographics	
Age (over 30 years ^a)	84 (40.0)
Female ^a	70 (33.3)
GHB-associated high-risk behaviors (potential outcome mediators)	
GHB dependence	38 (18.1)
Use of a GHB congener	27 (12.9)
Co-ingestion of GHB with other agent(s) ^b	34 (16.2)
Co-ingestion of GHB with ethanol	43 (20.5)
GHB severity outcomes	
Major GHB-associated health outcome ^c	19 (9.1)
Referred to a health care facility by CPCS	134 (63.8)
Final outcome status determined by CPCS	111 (52.9)
Treated in health care facility, outcome status determined ^d	35 (31.5)

^a Age missing for 21 subjects; gender missing for 6 subjects.

^b Other agents co-ingested included amphetamines, benzodiazepines, cocaine, marijuana, methylenedioxymethamphetamine, opiates, ketamine, and selective serotonin reuptake inhibitors.

^c Major outcome involved life-threatening symptoms including cardiovascular or respiratory compromise or multiple seizures.

^d Percent of n from preceding row.

Table 3
Area-level demographic and SES measures based on the zip code of case origin

	Median (%)	(25th, 75th%)
% Non-urban population	0.0	(0.0, 0.2%)
Age distribution of persons aged 15 years or older		
% Adolescent to young adult (15–29 years)	21.8	(18.0, 26.5%)
% Adult 30 years or more	72.9	(65.5, 77.8%)
Race		
% White	50.1	(0.0, 76.7%)
% Non-white	33.7	(19.1, 48.3%)
Annual household incomes, dollars ^a × \$10,000	5.2	(3.6, 6.4)
Home values, dollars ^a × \$100,000	3.4	(2.1, 3.9)

^aDollars are reflective of value in 2000.

Table 4
Univariate models – socioeconomic census variables predicting four risky behaviors

n = 210	Dependent on GHB		Used GHB congener		Co-ingested GHB with other agent(s)		Co-ingested GHB with ethanol	
	OR95% CI	p value	OR95% CI	p value	OR95% CI	p value	OR95% CI	p value
% Non-urban	1.660.32–8.60	0.55	4.290.92–20.04	0.06	0.01<0.001–790.44	0.41	3.910.91–16.80	0.07
Age (% adult ≥30 years) ^a	>999.996.46–>999.9	<0.001	502.952.40–>999.9	0.02	19.210.18–>999.9	0.21	24.780.35–>999.9	0.14
Race (% White)	0.970.36–2.59	0.95	0.670.22–2.09	0.49	0.960.34–2.69	0.94	0.900.35–2.30	0.83
Household income, dollars ^b × \$10,000	1.401.17–1.66	<0.001	1.221.02–1.48	0.03	1.291.08–1.53	0.004	1.120.96–1.32	0.15
Home value, dollars ^b × \$100,000	1.070.89–1.28	0.48	1.110.90–1.36	0.33	1.281.07–1.53	0.01	1.010.85–1.22	0.88

^a As a percentage of all persons aged 15 years and older (denominator excludes 0–14-year-olds).
^b Dollars are reflective of value in 2000.

Table 5
Area-level SES as a predictor of major GHB-related outcomes: multivariate logistic regression analysis

	Model 1 (includes area-level demographics ^d)			Model 2 (includes area-level demographics ^d and GHB-associated high-risk behaviors ^b)		
	OR	95% CI	p value	OR	95% CI	p value
All subjects (n = 210)						
Household income, dollars ^c × \$10,000	1.43	1.05–1.96	0.02	1.39	1.00–1.94	0.05
Home value, dollars ^c × \$100,000	1.48	1.16–1.90	0.002	1.41	1.09–1.82	0.01

^a Area-level demographics: % non-urban, % adult 30 years or more, and % White.
^b High-risk behaviors: dependence on GHB, used GHB congener, and co-ingested other agents.
^c Dollars are reflective of value in 2000.

Area-level SES as a predictor of health care facility treatment for GHB among 111 subjects with verified outcomes: multiple logistic regression analysis including area-level demographics, GHB-associated high-risk behaviors, and case severity

Table 6

	Model 1 (includes demographics ^a)			Model 2 (includes demographic ^a and GHB-associated high-risk behaviors ^b)			Model 3 (includes demographics ^a , GHB-associated high-risk behaviors ^b , and case severity ^c)		
	OR	95% CI	p value	OR	95% CI	p value	OR	95% CI	p value
Subjects with verified outcomes (n = 111):									
Household income, dollars ^d × \$10,000	1.29	0.98–1.70	0.07	1.15	0.85–1.56	0.38	1.03	0.71–1.51	0.88
Home value, dollars ^d × \$100,000	1.30	1.02–1.66	0.04	1.26	0.97–1.63	0.08	1.07	0.76–1.50	0.72

Major outcomes were noted for 17 of the 111 subjects included in this analysis.

^aDemographics (Models 1, 2, and 3): area-level data for % non-urban, % adults 30 years or more, and those 15 years or older, and % White.

^bHigh-Risk behaviors (Models 2, 3): dependence on GHB, used GHB congener, and co-ingested other agents.

^cCase severity (Model 3): cases coded as moderate to severe by AAPCC criteria (see Methods).

^dDollars are reflective of value in 2000.