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Systematic Assessment of Gamma Hydroxybutyrate (GHB) Effects During and After Acute Intoxication

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Abstract

We adapted and tested a previously published questionnaire battery eliciting sensory and cognitive symptoms during (acute) and immediately after (post-acute) GHB intoxication. Studying 125GHBusers, we assessed the instrument's internal consistency using Cronbach's alpha (CA) and responsiveness to change comparing acute and post-acute symptoms. The final 14-item battery demonstrated good internal consistency (CA ≥ 0.85 , both acute and post-acute). The median symptom score (possible range 0–64) was 30 (acute) and 6 (post-acute; difference $p < 0.001$). This modified substance-specific symptom battery, which is easily administered, demonstrated excellent performance characteristics. It can be used to study GHB and, potentially, related drugs of abuse.

INTRODUCTION

Gamma hydroxybutyrate (GHB) is a drug of abuse with a wide variety of well-recognized sensory and cognitive effects.^{1–3} Nonetheless, there are no established tools to systematically assess these GHB effects. Such a systematic approach is particularly relevant to the study of GHB intoxication in relation to correlates of abuse, such as drug co-ingestion and other drug-related high-risk behaviors, including GHB use in a pattern consistent with dependency.

As part of a larger study assessing the covariates and outcomes of GHB use, we modified and adapted a previously reported multi-item battery assessing acute GHB sensory and cognitive effects.⁴ In this study, we assessed the performance of this new instrument. This battery was included as part of a larger FORGE (Factors in Overdose Research into GHB Effects) survey instrument administered to a group of GHB users recruited through poison control center case referrals and through direct outreach to the GHB-using community.

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We have previously described the design and administration of the overall FORGE survey, a questionnaire that addressed GHB precursors and analogues, including gamma butyrolactone and 1,4-butanediol.⁵ In this paper, GHB refers to this specific compound as well as the related congeners. Our goal in the present analysis is to assess the performance characteristics of the GHB sensory and cognitive symptom battery that we administered. Specifically, we wished to assess the battery's internal consistency, its responsiveness to symptom change comparing the initial acute intoxication to the later immediate post-acute intoxication time frame, the concurrent validity of measured intensity of intoxication as a factor associated with selected drug-abuse associated behaviors, and the robustness of the battery when administered in a modified format in different settings.

METHODS

FORGE Survey Overview

The overall FORGE survey instrument was a structured, telephone-administered questionnaire consisting of 272 content items, the majority of which required simple yes–no responses. It was administered using computer-assisted telephone interview (CATI) software. The mean administration time was 33 ± 10 minutes. The duration of interviews was not normally distributed, however, ranging from 18 to 75 minutes (median time 31 minutes; 25th–75th percentiles, 26 to 38 minutes).

The questionnaire was intended to accomplish the following:

- address specific beliefs and practices related to GHB;
- detail the sources used in obtaining the drug and the circumstances surrounding GHB use;
- identify substances co-ingested with GHB, whether licit or illicit;
- capture slang terms for GHB and its congeners;
- delineate clinical symptoms related to acute and chronic GHB use;
- elicit subject demographics and socioeconomic status; and
- assess general physical and mental health status using standard quality of life measures.

To accomplish this ambitious set of goals, the FORGE survey utilized, wherever practical, validated batteries, including adapting questionnaire items specific to other drugs of abuse by making them applicable to GHB.

GHB Symptom Battery

In developing the FORGE questionnaire, we identified only one published survey instrument specific to GHB, that of Miotto et al.⁴ Its core component is a multi-item battery eliciting subjective cognitive and sensory symptoms experienced during and immediately after GHB use. The Miotto battery contained 25 items repeated twice (during and after use). Because of its multi-item length—especially if embedded in a large, parent questionnaire, as was our intended use—the original battery was cumbersome.

To address the length, we eliminated 12 items from the Miotto battery that we deemed too ambiguous or non-specific. Examples of deleted items include: tranquility, giddiness, craziness, optimism, placidity, and well-being. Based on our clinical experience, however, we did add two entirely new GHB-related items: one cognitive, eliciting “loss of memory or

amnesia,” and a second, sensory/motor, assessing “involuntary muscle jerking.” The final modified battery that we administered contained 15 distinct items.

Based on the Flesch-Kincaid Readability Test,⁶ the reading levels for both batteries were similar. For the original (Miotto) battery, the composite text requires a 6.5 grade reading level, and for the FORGE revision, the text is at a 6.6 grade reading level. The range for the individual items was narrower for the FORGE survey (sixth to ninth grade) compared to the original (fourth to tenth grade). In addition, the term “auditory hallucinations” in the original battery, the only item in either battery at a tenth grade reading level, was modified in our survey to “voice or sound hallucinations.”

Of the 15 items, ten address cognitive, two sensory-motor, and three combined cognitive/sensory-motor experiences (see Table 1). As with the Miotto survey, the battery questions were asked twice, first to assess symptoms during acute GHB intoxication (“acute”) and again for symptoms experienced immediately following intoxication (“post-acute”). For acute intoxication, respondents were allowed to specify their own time frame, in hours, during which such effects were applicable. Respondents were asked to rank the likelihood, when GHB-intoxicated, of experiencing each item. Responses were elicited on a five-point Likert-type scale with “0” for never experience and “4” for always experience.

In addition to the administration of the battery within the structured telephone survey, we separately administered selected items from this battery as part of a brief, self-completed questionnaire. This was administered during intake to a separate component of the FORGE project involving ten focus groups studying GHB experiences and beliefs.⁷ The completion of questionnaires during the focus groups took 8 to 10 minutes, and the total length of focus groups averaged two hours. In order to compare these responses to the survey group, we analyzed only the 11 items in the focus group questionnaire that were identical to those in the telephone battery (see marked items in Table 1 indicating their additional administration in the focus group setting).

Last, we analyzed the likelihood that high-risk GHB behaviors would be associated with increasing intensity of GHB intoxication as reflected in the acute GHB symptom score. We focused on four “high-risk behaviors” that were linked to a statistically significant increased likelihood of hospitalization in a previous analysis of this cohort: co-ingestion of GHB with ethanol, driving under the influence of GHB, the use of GHB to treat withdrawal symptoms, and co-ingestion of GHB with ketamine.⁸ In that same previous study, we had also studied ten demographic and GHB user characteristics as potential covariates of high-risk GHB-associated behaviors, and we retained those covariates in the current analysis as well.

Subject Recruitment

We utilized two distinct recruiting strategies for subjects completing the FORGE survey, as reported previously.⁵ One pool of subjects was identified through California Poison Control System (CPCS) surveillance, comprised of persons sufficiently symptomatic from GHB to seek medical attention in a health care facility (and thus meet our study definition of experiencing an adverse outcome). A second, non-CPCS group was recruited via Internet postings on *Erowid* and *Project GHB* Web sites; through the placement of flyers in public places, such as bars, coffee shops, clubs and laundromats; through physician referrals; and by “snowball sampling,” that is, subject recruitment generated from other GHB users. Subject eligibility in this second group was confirmed through a brief telephone screening interview. These subjects might have experienced GHB-related hospital-based treatment in the past, but need not have to be eligible.

Subjects younger than 16 years old were excluded. Recruitment for the CPCS cohort was initiated in July 2003, the non-CPCS group in January 2004, and enrollment for both groups closed on March 1, 2006.

We recruited potential focus group participants from among those completing the FORGE telephone screening interview, most of whom also went on to complete the telephone survey. Recruitment was restricted to the greater San Francisco area, where the focus groups were conducted. Additional limited snowball recruitment for focus group interviews also occurred through participants' contacts. Volunteers for all components of the study provided written or verbal informed consent prior to enrollment depending on the recruitment cohort. The Committee on Human Research at the University of California, San Francisco, approved the study.

Statistical Analyses

To assess internal consistency, we calculated Cronbach's alpha for each of the two time frames. Our a priori assumption was that the battery assessed a single construct. We derived a summary symptom score, calculated separately for acute and post-acute periods, by adding together the responses (a five-point Likert scale scored numerically 0 through 4). To assess responsiveness over time, we compared the summary acute score to the post-acute score, testing this difference using the Wilcoxon rank sum test for non-normally distributed data. Our a priori assumption was that the score would decrease in the post-acute intoxication time frame, consistent with a decreased frequency/intensity of such sensations. Only after assessing the overall summary score change did we proceed to test differences in the individual items and assess statistical differences in that context. For this reason, we did not treat these as requiring statistical adjustment for multiple testing. To assess the robustness of the battery to a different method and setting of administration, we compared the distribution of responses in the abbreviated, self-completed format administered to focus group participants to the same items from the FORGE survey. Our a priori assumption was that the range and distribution should be similar, albeit not super-imposable.

To assess generalizability, we examined whether responses to the battery deviated substantially by demographic covariates. Our a priori assumption was that the nature of the GHB intoxication should be appreciated consistently across major demographic categories. Thus, if the content of the items captured a generalizable experience, responses would be consistent and not differ substantively by demographic factors. These differences were also tested using Wilcoxon rank sum test. Because this represented a series of ten comparisons, we considered significance adjusted conservatively at 0.05/10 ($p = 0.005$). To assess concurrent validity, we tested whether summary scores were associated with selected GHB-associated co-ingestions or other risk-taking behaviors. We selected the co-factors to be studied based on our previous analysis as described above: co-ingestion of GHB with ethanol, driving under the influence of GHB, the use of GHB to treat withdrawal symptoms, and co-ingestion of GHB with ketamine.⁸ Thus, because this analysis was based on data that informed our hypothesis testing a priori, we did not adjust for multiple comparisons. We tested these associations using logistic regression analysis. Multivariate models included covariates associated with the GHB symptom score ($p < 0.20$): age, gender, sexual orientation, and cigarette smoking status.

RESULTS

We analyzed responses from 125 FORGE structured telephone surveys. Data for one subject who did not complete the symptom battery were excluded. Six of these participants were recruited through CPCS surveillance of health care facilities for GHB treatment; the remainder was recruited through direct public outreach. We additionally analyzed data from 47 of 51 focus group participants, excluding four who did not complete the abbreviated battery.

In the FORGE interview data, six subjects responded “don’t know” to a single item, two subjects responded “don’t know” to two items, and two subjects responded “don’t know” to a maximum of three items. These responses were scored as “0” in this analysis. Only 11 items elicited any such response; the maximum number of “don’t know” responses elicited by a single item was three.

Immediately prior to the battery questions, the FORGE interview subjects were asked how long the usual effect of a single dose of GHB lasts. Most subjects, 73 (59%), indicated acute effects lasting for 2–4 hours after taking GHB; 34 (27%) noted effects for fewer than 2 hours; 10 (8%) reported acute effects for more than 4–6 hours, consistent with a standard anticipated time range of GHB effects.⁹ Only seven subjects (6%) reported acute effects of longer duration, while a single subject answered “don’t know” to this question.

Internal Consistency

The standardized Cronbach’s alpha coefficient for the 15-item survey battery was 0.83 and 0.86, respectively, for acute and post-acute symptoms. One item, “depression,” performed poorly, manifesting poor inter-item correlation within the battery at both the acute and post-acute time frames (0.07 and 0.27, respectively). With the removal of this item, the total adjusted Cronbach’s alpha coefficient increased to 0.85 and 0.86, respectively, consistent with its poor inter-item correlation. No other item in the reduced, 14-item acute intoxication battery demonstrated an inter-item correlation of less than 0.31 or, in the post-acute battery, an inter-item correlation of less than 0.34.

Responsiveness over Time

Given their internal consistency, we used the 14 items to calculate the total summary scores for acute and post-acute symptoms (see Table 2). The mean (median) total scores during acute intoxication were 28.6 (30.0). For post-acute symptoms, the mean (median) total scores were 8.26 (6.0; $p < 0.001$). Each of the 14 items was associated with scores that was greater for the acute compared to the post-acute period ($p < 0.001$), the anticipated direction of change in effect. Six items manifest particularly marked changes from the acute to the post-acute state (a decrease of two or more points out of a total range of 0–4). These items were: silliness, relaxation, increased sexuality, loss of inhibitions, increased sensitivity to touch, and euphoria.

Comparison with Focus Group Data

We assessed the distribution of responses to the abbreviated 11-item questionnaire administered to focus group participants, comparing these to the same items from the FORGE survey. Table 1 identifies the items that were also included in the focus group reduced battery. We analyzed the median and the 25th–75th percentile (IQR) scores of both the FORGE survey ($n = 125$) and the focus group participants ($n = 47$). The median (IQR) score during acute intoxication for the original survey group was 21 (15–27), while for the focus groups, the median was 24 (19–28, $p < 0.001$). For post-acute states, the median (IQR) scores for the survey and focus groups were 4 (1–8) and 7 (2–14), respectively ($p < 0.001$).

Generalizability

Table 3 summarizes the results for ten key demographic variables in relation to the mean subjective scores for acute GHB intoxication. For nine out of ten variables, there were no significant differences in the mean acute scores among dichotomous demographic divisions. The only variable with a significant difference in the acute scores was for current smokers compared to ex-smokers/never smokers (summary scores 32 for current smokers compared to 26 for all others; $p = 0.002$, < 0.005 significance adjusting for the ten comparisons carried out).

To further assess this finding, we analyzed smokers compared to ex-smokers/never smokers for each of the 14 items. Current smokers had significantly higher scores during acute intoxication for the following: silliness ($p < 0.02$), happiness ($p < 0.008$), tendency to talk ($p < 0.002$), and feeling stimulated or energetic ($p < 0.004$). Overall, the scores for current smokers were numerically higher for all 14 items. In the post-acute stage, current smokers had slightly higher, although not statistically significant, summary scores (9 for current smokers compared to 7 for all others, $p = 0.17$). Nonetheless, because of the association between smoking status and the acute symptom score, we also analyzed the items individually for the post-acute time frame. For the post-acute stage, current smokers had significantly higher scores for involuntary muscle jerking ($p < 0.004$), increased sexuality ($p < 0.04$), and loss of inhibitions ($p < 0.02$). However, in this same post-acute stage, current smokers had lower, although not statistically significant, scores for tendency to talk, stimulated or energetic, relaxation, and happiness.

Concurrent Validity

Lastly, using logistic regression analysis, we separately assessed the associations between high symptom scores during the acute or the post-acute stages of GHB intoxication and the likelihood of reporting any of the high-risk behaviors as defined previously. The results of this analysis are presented in Table 4. Overall, adjustment for demographic covariates and smoking status had little effect on the estimated associations. For both the acute and post-acute stages, the highest odds ratio (OR) for high symptoms scores was associated with using GHB to treat withdrawal symptoms (adjusted OR 3.09 and 2.25 for acute and post-acute, respectively; $p < 0.05$ in both cases). The next highest OR was for using GHB together with ketamine (adjusted OR 1.78 and 1.55, respectively; $p < 0.05$ in both cases). Although the acute score was statistically associated with driving while under the influence of GHB (adjusted OR 1.72; $p < 0.05$), this was not the case in the post-acute period (adjusted OR 1.36; $p > 0.05$). Neither acute nor post-acute scores were statistically associated with ethanol use.

DISCUSSION

The modified, final 14-item GHB symptom battery demonstrates a high degree of internal consistency and appears to assess a single construct, as intended. The comparison of summary scores between acute and post-acute time frames indicates that it is responsive to change in intoxication status over time. Scores from self-administered battery by focus group members compared to telephone-administration indicate that the battery is robust in two different settings. Further, analysis of the responses stratified by demographic covariates validates our assumption that the nature of the GHB intoxication is appreciated consistently across age, gender, race, education, and sexual orientation, indicating generalizability to differing demographic mixes in other study groups to whom this might be applied. Finally, concurrent validity was supported by the association between high summary symptom scores and previously identified high-risk behaviors associated with poor GHB outcomes, specifically using GHB to prevent withdrawal symptoms, co-ingestion of GHB with ketamine, and driving while under the acute influence of GHB.

The significant differences in summary scores during acute intoxication between current smokers compared to ex-smokers and never smokers are interesting findings that warrant further investigation. Although current smoking might simply be a potential confounder by being a marker for other “risky” behaviors,^{10,11} we did not observe a substantive change in the estimated associations between GHB symptoms score and key high-risk behaviors, even when adjusting for smoking status.

There may be a nicotine-GHB interaction such that the overall perceived effects of acute GHB intoxication, and certain specific effects in particular, are magnified in current smokers, a relationship that could be consistent with the known pharmacological effect of nicotine on

GABA-B receptor sites. Gamma-aminobutyric acid, subtype B (GABA-B) receptors play an important role in regulating brain reward function,¹² reinforcing the effects of drugs of abuse. The pharmacologic effects of both GHB and nicotine appear to be mediated, at least in part, via GABA-B. GHB exerts direct agonist effects at the GABA-B receptors, as well as activity at GHB-specific receptors.⁹ Nicotine-GABA-B interactions are likely to be complex. Dopaminergic activity in the mesolimbic pathway and GABA-B receptor expression in the rat prefrontal cortex are modulated by nicotine and cigarette smoke.¹³ Baclofen, a GABA-B agonist, antagonized nicotine-induced dopamine release in the nucleus accumbens of rat.¹⁴ In addition, baclofen and another experimental GABA-B agonist decreased nicotine self-administration in rats.^{12,15} In a relevant human study, while baclofen administration did not change nicotine craving or the number of cigarettes smoked, it did alter the sensory properties of cigarettes in a contradictory fashion, decreasing ratings of “like cigarette’s effects” while increasing “feeling relaxed.”¹⁶ Another intriguing human study found that endogenous levels of urinary GHB in smokers almost doubled when compared to nonsmokers and to alcohol drinkers.¹⁷ The authors of that study speculated that the elevated GHB levels were due to stimulatory effects of nicotine on the central nervous system. Given that, in our analysis, higher GHB symptom scores were linked to greater likelihood of reporting risky GHB-associated behaviors and that smoking may promote a more intense GHB “high,” this may have a substantive public health impact.

Our study limitations should be kept in mind. Although we interviewed more than one hundred participants, our study population was relatively small and geographically concentrated. Importantly, selection effects of both CPCS referral and non-randomized population outreach likely indicate that our group used GHB more frequently and with more severe outcomes. In addition, the lengthy nature of the telephone survey may have selected for the more functional subset of GHB users capable of maintaining focus for the duration of the survey. However, we did elicit a wide range of responses with sufficient study power to observe statistically significant associations in both stratified and multivariate analyses. Although we did carry out multiple statistical tests, our analytic strategy did take this complexity into account.

In summary, the final 14-item symptoms battery has excellent performance characteristics in terms of consistency, responsiveness, robustness, generalizability, and concurrent validity. Its administration to other cohorts of GHB users will be necessary in order to assess its performance in different settings. Finally, although designed to be specific to GHB, adaptation of this battery to other drugs of abuse by inclusion or exclusion of other selected items may be feasible.

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TABLE 1

GHB cognitive and sensory-motor items included in the structured interview administered by telephone (asked in random order)

Item	Content type
Involuntary muscle jerking *	Sensory-motor
Silliness *	Cognitive
Happiness	Cognitive
Loss of memory or amnesia *	Cognitive
Voice or sound hallucinations *	Cognitive
Relaxation *	Cognitive/sensory-motor
Increased sexuality *	Cognitive/sensory-motor
Sight or image hallucinations *	Cognitive
Tendency to talk *	Cognitive
Loss of inhibitions *	Cognitive
Heightened sense of touch *	Sensory-motor
Increased sensitivity to sound *	Cognitive/sensory-motor
Stimulated or energetic	Cognitive
Euphoric	Cognitive
Depressed or lethargic	Cognitive

The trunk question was worded as: "During the time of GHB's effects, using a scale of 0 for "never" to 4 for "always," how likely are you to feel these sensations?"

* Items that were included in the 11-item focus group version of the instrument.

TABLE 2

Changes in mean scores for subjective feelings during (“acute”) and just after (“post-acute”) GHB intoxication among 125 interviewed GHB users

Symptoms	Acute mean scores	Post-acute mean scores	Changes in mean scores*
Summary score	28.60	8.26	20.34
Loss of inhibitions	2.78	0.52	2.26
Euphoric	2.82	0.58	2.24
Increased sexuality	2.64	0.58	2.06
Relaxation	3.10	1.06	2.04
Heightened sense of touch	2.41	0.40	2.01
Silliness	2.50	0.50	2.00
Happiness	3.00	1.26	1.74
Tendency to talk	2.27	0.71	1.56
Stimulated or energetic	2.06	0.74	1.32
Increased sensitivity to sound	1.58	0.42	1.12
Involuntary muscle jerking	0.93	0.38	0.55
Sight or image hallucinations	0.75	0.22	0.54
Loss of memory or amnesia	1.14	0.70	0.43
Voice or sound hallucinations	0.62	0.20	0.42

Single-item score minimum = 0, maximum = 4; summary score possible range 0–64.

Items presented in order of magnitude of change.

* All changes in scores for summary and for individual items, $p < 0.001$, Wilcoxon rank sum test.

TABLE 3

Acute GHB symptom scores in relation to demographics and cigarette smoking status among 125 interview subjects

Variable	N	Mean	± SD	<i>p</i>
Female	34	26.35	11.30	0.14
Male	91	29.44	9.83	
30-years-old or less	67	27.06	10.45	0.07
>30 years old	58	30.38	9.90	
White, non-Hispanic	90	27.92	9.77	0.24
All other races	35	30.34	11.50	
Some college or less	72	29.18	10.61	0.47
College graduate or more	53	27.81	9.90	
Employed	83	29.21	10.08	0.36
Not working	42	27.41	10.73	
Annual income <\$20,000	46	28.07	11.18	0.71
Annual income >\$20,000	75	28.77	9.71	
Household size 1–2 people	80	28.68	10.69	0.90
Household size 3 or more	45	28.44	9.66	
Gay, bisexual, or transgender	35	31.14	9.53	0.09
Other sexual orientation	90	27.61	10.46	
Body mass index normal	89	28.34	10.43	0.66
Overweight/obese	36	29.25	10.06	
Never/former smoker	66	25.94	10.30	0.002 *
Current smoker	59	31.58	9.52	

* $p < 0.005$, a cut-off taking into account the 10 comparisons made.

TABLE 4
Association of GHB symptom scores and selected high-risk, GHB-associated behaviors: logistic regression analysis among 125 subjects

High-risk behavior	Unadjusted logistic regression model		Logistic model adjusted for demographics and smoking*	
	OR [†]	95% CI	OR [†]	95% CI
Acute GHB symptom score				
Ethanol co-ingestion	1.18	0.90–1.57	1.22	0.90–1.66
Ketamine co-ingestion	1.86	1.26–2.75	1.78 [‡]	1.18–2.68
Driving under the influence of GHB	1.63	1.16–2.29	1.72 [‡]	1.17–2.52
GHB used to treat withdrawal	3.05	1.74–5.35	3.09 [‡]	1.73–5.51
Post-acute GHB symptom score				
Ethanol co-ingestion	1.07	0.57–1.53	1.06	0.73–1.53
Ketamine co-ingestion	1.58	1.06–2.35	1.55 [‡]	1.03–2.34
Driving under the influence of GHB	1.39	0.96–2.02	1.36	0.90–2.05
GHB used to treat withdrawal	2.24	1.40–3.59	2.25 [‡]	1.38–3.69

* Variables from Table 3 associated with GHB symptom score at $p < 0.20$: age, gender, sexual orientation, and cigarette smoking status (current vs. former or never smoker).

[†] OR values expressed per 8 points in score (approximately 1 SD).

Statistically significant ($p < 0.05$).