



An observed rise in γ -hydroxybutyrate-associated deaths in London: Evidence to suggest a possible link with concomitant rise in chemsex



Joanna Hockenhull^{a,*}, Kevin G. Murphy^b, Sue Paterson^a

^aToxicology Unit, Imperial College London, St. Dunstan's Road, London W6 8RP, UK

^bSection of Endocrinology and Investigative Medicine, Imperial College London, Commonwealth Building, Hammersmith Hospital, Du Cane Road, London W12 0NN, UK

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ABSTRACT

Background: Gamma-hydroxybutyrate (GHB) is the drug most linked to acute harm out of those used in chemsex, the incidence of which is reported to be increasing. However, there have been few systematic studies of the harms associated with GHB use. We investigated GHB-associated deaths from London coroners' jurisdictions between 2011 and 2015.

Methods: Blood and urine samples were collected by pathologists and submitted for toxicological analysis at the request of coroners. Data from the Toxicology Unit, Imperial College London was retrospectively analysed. This comprised of 6633 cases from seven out of eight coroners' jurisdictions in London that underwent toxicological analysis between January 2011 and December 2015.

Results: A total of 61 GHB-associated deaths (0.92% of total cases), 184 cocaine-associated deaths (2.8% of total cases) and 83 MDMA-associated deaths (1.3% of total cases) were identified. There was a 119% increase in the proportion of GHB-associated deaths detected in 2015 compared to 2014. Over the same time period there was a 25% increase in cocaine-associated deaths and a 10% decrease in MDMA-associated deaths.

Conclusions: Our data suggest that GHB-associated deaths are increasing in London, and that this is likely at least in part due to increasing use of GHB for chemsex. Further studies on the use of GHB are urgently required to understand the extent of its use, whether this is as prevalent in other major urban areas in the UK, and the full extent of the harms it causes.

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1. Introduction

Gamma-hydroxybutyrate (GHB) is a central nervous system depressant produced naturally in the body in small quantities and is also an illegal drug used recreationally for its aphrodisiac, euphoric and relaxant effects [1]. Regular use of GHB is thought to be relatively higher in the lesbian, gay, bisexual, and transgender community, though other populations also use it [1]. Anecdotal evidence suggests that GHB use is increasing, and this may be driven in part by an increase in the incidence of chemsex.

Chemsex is a specific form of recreational drug use involving specific drugs – GHB/ γ -butyrolactone (GBL), methylamphetamine and mephedrone alone or in combination to enhance or prolong sexual sessions. Such sessions can last for several days, involve multiple partners and include high risk behaviour. Chemsex mainly occurs amongst men who have sex with men (MSM) [2]. It

is recognised that MSM are more likely to take drugs than the general population [3]. In the United States and Australia the practice is known as 'party and play' (PNP) and is more common in urban areas with a large gay population [4,5].

Whilst chemsex is not a new phenomenon, healthcare professionals are worried that increased availability and accessibility of drugs and sex through websites and mobile applications has increased the acceptability, fashionableness and incidence of chemsex [6,7]. The mainstream media are becoming increasingly aware of the issue and drug treatment centres report being overwhelmed by the number of men seeking help [8]. However, it is difficult to know how harmful chemsex is. The Chemsex Study (2014) reported that whilst chemsex has received significant media attention, evidence on the extent of the issue and potential harms was limited [7]. There is a lack of reliable statistics about the number of men involved, though it is believed that the proportion of MSM participating in chemsex is relatively small [9].

GHB is the drug most linked to acute harm out of those used in chemsex. However, though recent publications have highlighted chemsex-associated harms to health including addiction and

* Corresponding author. Fax: +44 203 311 7110.

E-mail address: j.hockenhull@imperial.ac.uk (J. Hockenhull).

sexually transmitted diseases [2,7,10], the risks and rates of overdose have been relatively little explored.

Data collected over a 12 month period (2013–2014) from the European Drug Emergencies Network found that GHB/GBL was the fourth most common acute drug toxicity presentation (after heroin, cocaine and cannabis), based on self-reported use. For the two centres located in London, GHB/GBL was the most common drug reported, with a combined total of 380 presentations over the 12 month period [11].

The onset of GHB effects usually occurs within 15 min of ingestion. Effects last for 3 h on average with a typical dose but can be indefinitely prolonged through repeated dosing [12]. Previous studies have shown that GHB is rapidly eliminated from the body with reported half-lives ranging from 20 to 53 min and being completely eliminated, and therefore undetectable in blood, within 4–8 h following ingestion [13].

Recreational doses are reportedly taken 1–3 g of GHB but some users with tolerance can take up to 4–5 g. GHB is commonly available as a liquid of variable concentration and is often measured in doses such as a capful or teaspoon [14]. Online advice states that an initial safe recreational dose of GBL is 0.5–1 mL with no redosing within a 3 h period [14]. At low doses the effects of GHB are similar to alcohol. However, higher doses have anaesthetic effects, rapidly causing unconsciousness and can lead to coma, respiratory depression and apnoea [1]. Coma is relatively common with many men reporting becoming unconscious after taking GHB [7]. In the majority of cases these individuals awake spontaneously within approximately 1.5–3 h, but the coma can lead to death. GHB has a steep dose-response curve and there is large variability in effects of a particular dose between and within individuals [1]. This means that a euphoric dose for one person could be a sedative dose for another, and it is relatively easy to accidentally overdose [15]. In addition, GHB and GBL are used and described interchangeably with each other, GBL converts rapidly to GHB in vivo and the perceived effects are indistinguishable. Therefore, it is often difficult to know both for the user and healthcare professionals whether GHB or GBL has been consumed. However, GBL is absorbed much faster and eliminated more slowly than GHB, resulting in faster and more prolonged effects. Pure GBL is approximately three times more potent than GHB preparations [16]. In addition, alcohol or other psychoactive substances, both depressants and stimulants, may intensify the toxic effects of GHB [1,17].

The main risks of overdosing are thus from taking too much GHB/GBL, redosing too soon or taking the drug in combination with alcohol or other psychoactive substances.

GHB-associated deaths have been reported from the USA, Canada, Europe and Australia [18,19]. However, there has been little systematic investigation of the incidence of GHB-associated deaths. Zvosec et al. analysed 226 GHB-associated deaths that occurred between 1995 and 2005 in the USA, Canada, and the UK, but suggested that this was likely a significantly under-representation because, amongst other reasons, many routine toxicology tests do not include or request GHB analysis, and there is no central database recording such deaths [18]. Corkery et al. described 159 deaths associated with GHB and derivatives between 1995 and 2013 in the UK's National Programme on Substance Abuse Deaths database, providing comprehensive and detailed data, but acknowledged that their study likely underestimates the number of deaths due to the fact that GHB analysis is not routinely performed in post-mortem investigations in the UK [19].

As reports suggest that the use of GHB and the incidence of chemsex are increasing, and GHB is the drug most linked to acute harm out of those used in chemsex, we systematically investigated the numbers of GHB-associated deaths from London coroners' jurisdictions to see whether there was evidence of increased

deaths associated with GHB which suggest increased and/or more dangerous use of the drug.

2. Material and methods

2.1. Study design

The Human Tissue Act 2004 gives coroners consent only to conduct analysis relevant to the investigation into the cause of death. The analyses carried out were therefore determined on a case-by-case basis and were limited by the case history provided and analyses requested by the pathologist. Routine analyses included gas chromatographic measurement of ethanol in blood, urine, and/or vitreous humour; a general Gas Chromatography–Mass Spectrometry (GC–MS) screen of blood (or liver, gastric contents, vitreous humour if blood unavailable) for drugs including unknown, licit, and illicit drugs [20]; and a GC–MS screen of urine for illicit drugs [21]. Quantification and/or confirmation of any drugs was carried out according to the UK and Ireland Association of Forensic Toxicologists' guidelines [22]. Approval for the use of data generated from this analysis was granted by South West London Research Ethics Committee 1 (reference 11/LO/0033).

GHB is not detected in blood during routine analysis; specific analysis is required. GHB analysis was historically performed when requested or when ingestion was indicated from the history provided with the case. As we observed an increase in requests for GHB analysis, the drug was incorporated into the GC–MS urine illicit drugs screen in May 2012. This screen is performed on approximately half of all cases received (a total of 709 cases received in 2011, 974 cases in 2012, 1534 cases in 2013, 1624 cases in 2014 and 1792 cases in 2015).

Femoral blood samples from individuals with detectable GHB in their urine were subsequently analysed for GHB. If no urine was received, blood analysis was determined based on the history received with the case.

GHB concentrations in post-mortem blood can vary widely, and there appears to be no definable fatal concentration. It is generally accepted that post-mortem femoral blood concentrations greater than 50 mg/L are indicative of GHB/GBL ingestion rather than endogenous production [19]. A case was therefore considered to be a GHB-associated death if GHB was detected in the post-mortem femoral blood at a concentration greater than 50 mg/L.

2.2. Study population

The Toxicology Unit, Imperial College London routinely analysed samples from all of the coroners jurisdictions in London, with the exception of West London. This study took place between 2011 and 2015. The total number of cases analysed during this time was 6633.

Cases in which recreational use of either cocaine or methylenedioxymethylamphetamine (MDMA) was identified were also investigated for comparison. This included all cases where MDMA or cocaine was detected during the general screen (limit of detection 0.05 mg/L) but excluded 'body packers' and people with a history of heroin use who tend to take 'crack' cocaine rather than cocaine recreationally. While cocaine or MDMA may not have been the direct cause of death, they were present in the body at the time of death and were thus defined as associated deaths.

3. Results

3.1. Prevalence

We identified 61 GHB-associated deaths (0.92% of total cases), between 2011 and 2015, with post-mortem femoral blood concentrations ranging from 108 to 2444 mg/L (see Fig. 1).

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