ACMD

Advisory Council on the Misuse of Drugs

ACMD Report – A review of the evidence on the use and harms of Carisoprodol

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1. Introduction

1.1. Carisoprodol is an orally active centrally acting muscle relaxant that was used clinically in the UK to treat painful muscle spasm.

1.2. In 2007 the European Medicines Agency recommended that Member States (including the UK at that time) suspend marketing authorisation for carisoprodol [EMA 2007]. After review, it was concluded that the benefits of medicines containing carisoprodol no longer outweighed the risks from its use. These risks include an increased likelihood of abuse and addiction as well as intoxication and psychomotor impairment. It was considered that there were safer alternatives for the treatment of acute musculoskeletal disorders where a muscle relaxant was needed. As a result, in the UK the marketing authorisations for carisoprodol were withdrawn in 2008 [MHRA, 2008].

1.3. Carisoprodol is still available as a prescription medicine in a number of countries including the USA [WHO 2023]. In 2019, more than 800,000 prescriptions for carisoprodol were issued in the USA [ClinCalc 2020].

1.4. The major metabolite of carisoprodol, meprobamate, was itself used clinically for short-term treatment of anxiety or painful muscle spasm. In 2012, the European Medicines Agency recommended the suspension of marketing authorisations for medicines containing meprobamate. In the UK the license for meprobamate production was cancelled in 2016. [MHRA 2016].

1.5. Carisoprodol is used non medically to produce feelings of relaxation, giddiness, and drowsiness. It is typically consumed orally, but when taken by insufflation (snorted), a more intense euphoria may be experienced which is likely due to more rapid absorption of the drug. It may be combined with other drugs such as opioids and benzodiazepines resulting in an enhanced ‘high’ but also with an increased likelihood of overdose [Horsfall 2017].

1.6. Carisoprodol induced relaxation and euphoria are generally short-lived due to fast metabolism to meprobamate and to other metabolites. The sedative effects of carisoprodol are longer lasting and are generally attributed to meprobamate which persists in the body for much longer than carisoprodol.

2. Legal Control

International Controls

2.1. Following a critical review report on carisoprodol published by the World Health Organization’s Expert Committee on Drug Dependence (ECDD) [WHO 2023; 2025] the United Nations Commission on Narcotic Drugs (UN CND) at its 68th session adopted Decision 68/6, confirming the international scheduling of carisoprodol under Schedule IV of the 1971 Convention on Psychotropic Substances [UNODC 2025].

2.2. Prior to the UN CND decision the United States had classified carisoprodol as a Schedule IV controlled substance under the Controlled Substances Act, following growing evidence of its misuse [Federal Register 2011].

2.3. As a result of the UN CND decision on the international scheduling of carisoprodol, Canada amended its Controlled Drugs and Substances Act, placing carisoprodol in Schedule V effective April 2025 [Government of Canada 2025]. Other jurisdictions have begun enacting similar controls, including Malta, which implemented national scheduling in June 2025 [Government of Malta 2025], and Hong Kong, which followed in July 2025 [Government of Hong Kong 2025].

United Kingdom

2.4. In the UK carisoprodol is not currently controlled under the Misuse of Drugs Act 1971 (MDA). However, it is psychoactive and as such may be subject to the Psychoactive Substances Act 2016 ("the PSA 2016"). The PSA 2016 makes it an offence to import, export, supply, and possess with intent to supply a psychoactive substance (with certain exceptions) where the person carrying out those actions knows, or is reckless as to whether, the psychoactive substance is likely to be consumed by some other person for its psychoactive effects. It also makes it an offence to possess psychoactive substances in a custodial institution. It should be noted, however, that compounds that meet the definition of a 'medicinal product' in Human Medicines Regulations of 2012 are exempted from the Act and this can include medicines licensed elsewhere in the world.

2.5. In the UK meprobamate is controlled under Class C, Schedule 3 of the MDA 1971.

2.6. The UN CND decision confirming the international scheduling of carisoprodol under Schedule IV of the 1971 Convention on Psychotropic Substances [UNODC 2025] obliges the UK, as a signatory, to consider and introduce appropriate legal control measures for this particular compound. Hence, the ACMD has undertaken a review of the harms associated with its use and has considered the legal control of carisoprodol to comply with our international obligations.

3. Chemistry

3.1. The IUPAC chemical name for carisoprodol is (RS)-[2-(carbamoyloxymethyl)-2-methylpentyl]-N-propan-2-ylcarbamate. It can also be referred to as N-isopropyl-2-methyl-2-propyl-1,3-propanediol dicarbamate or N-isopropylmeprobamate. When it was authorised for clinical use its brand name in the UK was Carisoma®.

3.2. The chemical structure of carisoprodol is shown in Annex A. It is a racemic mixture of the enantiomers, *S*-carisoprodol and *R*-carisoprodol, but which isomer is responsible for its biological activity has not been determined.

4. Pharmacology

Pharmacokinetics

4.1. Carisoprodol is well absorbed following oral administration (bioavailability ~90%). It has a rapid onset of action (0.5 - 1 h) and a time to maximum plasma concentration of ~1.5 h. It can also be taken by insufflation (snorting) which produces

a more rapid rise in the plasma concentration and thus the onset of drug-induced effects such as euphoria occurs much sooner, making this an attractive route of administration when taken for its pleasurable effects [Gupta 2020].

4.2. Carisoprodol is metabolised in the liver by the cytochrome P450 enzyme CYP2C19. It has a biological half-life of ~2.5 h whereas meprobamate, its active metabolite, has a half-life of ~10 h. In individuals with reduced or zero CYP2C19 activity the levels of carisoprodol are higher and those of meprobamate reduced. Poor CYP2C19 metabolisers constitute ~5% of Caucasians and those of African origin but rises to ~20% in Asians (both South Asians and East Asians) [WHO 2023].

4.3. A number of drugs, including fluoxetine, topiramate, sertraline and tricyclic antidepressants and oral contraceptives, inhibit CYP2C19 and increase the levels of carisoprodol in the body [WHO 2023].

Pharmacodynamics

4.4. Carisoprodol exerts a dual action on $GABA_A$ receptors in the central nervous system (CNS). $GABA_A$ receptors are ligand-gated chloride channels mediating neuronal inhibition. Carisoprodol acts both as a positive allosteric modulator enhancing the action of $GABA_A$ on the receptor and as a direct activator of the $GABA_A$ receptor thus enhancing inhibitory neurotransmission in the CNS [Kumar 2015]. The allosteric modulatory effects are seen with slightly lower concentrations than those required for direct activation.

4.5. Carisoprodol is metabolised in the liver; its primary metabolite, meprobamate (chemical structure is shown in Annex A), also acts as a positive allosteric modulator and direct activator of $GABA_A$ receptors but shows a slightly different selectivity for $GABA_A$ receptor subtypes [Kumar 2016]. Barbiturates (e.g. pentobarbital) and benzodiazepines (e.g. diazepam) also modulate $GABA_A$ receptors but not in an identical manner to carisoprodol. The differences in receptor activation between these drugs likely underlies some of the differences in the behavioural effects they produce.

4.6. In an animal model of drug discrimination used to predict abuse potential, bemegride, a barbiturate antagonist, reversed the effect of carisoprodol, but the benzodiazepine antagonist flumazenil did not [Gonzalez 2009].

4.7. In mice carisoprodol induced a loss of the righting reflex, a measure of sedation [Gatch 2012]. When the dose of carisoprodol was increased daily over 4 days, tolerance to the loss of righting reflex was observed i.e. the effect of the drug decreased, indicating the development of tolerance.

Behavioural effects

4.8. Carisoprodol reduces anxiety and produces a feeling of well-being (euphoria), drowsiness and sedation. However, the euphoria is generally short-lived due to rapid metabolism of the drug. At high doses confusion, disorientation, partial amnesia and respiratory depression occur [Nebhinani 2013]. Its prolonged sedative effects are generally attributed to its major active metabolite meprobamate.

4.9. The subjective effects of carisoprodol are similar to those of other CNS depressants that potentiate gabaergic transmission such as barbiturates and benzodiazepines. The combination of carisoprodol with benzodiazepines and opioids (colloquially referred to as 'The Holy Trinity') has been reported to increase the intensity of the "high" [Horsfall 2017]. The effect of carisoprodol in combination with alprazolam and hydrocodone is described as being similar to that experienced when taking heroin [Recovery Village 2020].

4.10. Illicitly manufactured pills containing carisoprodol and the opioid analgesic tapentadol (colloquially referred to as 'red apples') are currently available on the illicit drug market in the UK [WEDINOS; PHS 2025].

5. Detection and Analysis

5.1. Methods based on liquid chromatography (LC) coupled to either tandem mass spectrometry (MS) or high-resolution MS have been described for quantitative determination of carisoprodol and meprobamate in biological samples [Skinner et al 2004; WHO 2023]. Deuterated reference standards of each are commercially available [Essler 2012].

5.2. As a result of carisoprodol being rapidly and extensively metabolised to meprobamate, its concentration in blood, urine or other tissues rapidly decreases (its half-life in blood is approximately 2 hours) and so, depending upon the length of time before post mortem sample collection, the concentration of carisoprodol may be below the level of detection. However, as meprobamate persists for much longer (its half-life in blood is approximately 10 hours) it can be detected at much longer time intervals than carisoprodol [WHO 2023]. For this reason, testing for both carisoprodol and meprobamate rather than carisoprodol alone is important to avoid missing overdose cases in which carisoprodol is involved (false negatives).

6. Medical Use

6.1. Carisoprodol can be used clinically to treat muscle spasm in painful musculoskeletal conditions. It also reduces anxiety. Adverse drug reactions include drowsiness, sedation, headache, dizziness, addiction and dependence. A withdrawal response is precipitated upon abrupt discontinuation following prolonged use [Reeves 2010; Gupta 2020].

6.2. Although the UK marketing authorisations for carisoprodol were withdrawn in 2008, ongoing supply could be provided on a named patient basis for those previously prescribed and physically dependent on the drug [MHRA, 2014].

6.3. The ACMD Secretariat contacted the Medicines and Healthcare products Regulatory Agency (MHRA) to obtain information regarding the current licensing status of carisoprodol. The MHRA has confirmed that carisoprodol remains an unlicensed medicine in the United Kingdom, with the last licence cancelled in April 2012. The most recent Notification of Intent (NOI) submitted to the MHRA for its importation was received in July 2022.

6.4. In Australia, carisoprodol is no longer a licensed medicine but can be accessed through the Special Access Scheme. Indonesia withdrew carisoprodol from the market in 2013 due to problems with diversion, dependence and side effects.

6.5. Carisoprodol is no longer a licensed medicine in Canada. In 2025, Health Canada removed it from the Prescription Drug List and added it to Schedule V of the Controlled Drugs and Substances Act (CDSA).

6.6. In the USA, carisoprodol was classified under Schedule IV of the US Controlled Substances Act in 2012 meaning that it has a recognised medical use but also carries a risk of abuse and dependence [Conermann 2024]. Currently it is available on prescription. Since the Drug Enforcement Administration (DEA) scheduling, cases of carisoprodol abuse as recorded by the National Poison Data System (NPDS) have declined by 97% [Thornton et al. 2025].

6.7. Carisoprodol is not a licensed medicine in China. It is currently available as a medicine in other countries worldwide [WHO 2023].

7. Misuse

7.1. For use in the UK carisoprodol can be obtained from non-UK legitimate pharmaceutical sources or by purchasing counterfeit versions from online sources.

7.2. Carisoprodol is used non medically to produce feelings of relaxation, well-being (reduced anxiety), giddiness, and drowsiness. The experience has been described as being similar to that experienced with barbiturates (e.g. pentobarbital) [WHO 2023]. Carisoprodol is typically consumed orally, but when snorted intense euphoria may be experienced which is likely due to more rapid absorption of the drug.

7.3. Carisoprodol can be taken alone or with other drugs such as aspirin, ketamine, benzodiazepines, gabapentinoids and opioids [WHO 2023]. The combination of carisoprodol with a benzodiazepine (e.g. alprazolam) and an opioid (e.g. hydrocodone, oxycodone and tramadol or tapentadol) is said to produce an enhanced euphoric experience but increases the risk of overdose [Horsfall 2017].

7.4. In the UK, tablets containing carisoprodol alone are known as ‘somas’. Tablets containing carisoprodol and tapentadol are known as ‘red apples’ and first appeared on the UK drug market in 2021 [WEDINOS].

7.5. Precise data on the extent of carisoprodol misuse alone or in combination with other drugs in the UK is currently lacking. However, the submission of carisoprodol positive samples to the Welsh Emerging Drugs and Identification of Novel Substances (WEDINOS) programme suggests that it is likely to be occurring but only to a limited extent at present.

7.6. In the USA carisoprodol is often obtained by diversion of prescribed drug or obtained from pharmacies in Mexico which issue it without a prescription. In 2019, the DEA reported that carisoprodol was one of the most commonly diverted drugs in the USA [DEA 2019]. In the USA, the carisoprodol metabolite meprobamate is no longer commonly used to treat anxiety but is still available on prescription [Mayo Clinic 2025].

8. Health Harms

8.1. High doses, either supra-therapeutic dosing or intentional misuse, can result in euphoria, hypomania, confusion, disorientation, myoclonus, agitation and partial amnesia [Nebhinani 2013; Roth 1998; Gupta 2008]. Very high doses result in respiratory failure, coma and death [DEA 2019].

8.2. The features of carisoprodol toxicity (hypertension, tachycardia, involuntary robot-like choreiform movements, shivering and tremor) are different from those observed in meprobamate toxicity (cardiogenic hypotension, absent tendon reflexes, flaccid muscles, and CNS-depression) [Bramness 2005]. This may indicate that carisoprodol toxicity does not result simply from over activation of $GABA_A$ receptors. There is however one case report of the benzodiazepine antagonist flumazenil reversing severe carisoprodol overdose [Roberge 2000]. Flumazenil is not licenced in the UK, nor recommended by the UK National Poisons Information Service, for the reversal of carisoprodol (or meprobamate) acute toxicity in the UK. The use of flumazenil in this setting may be associated with severe adverse effects, including precipitating carisoprodol withdrawal in regular/long-term users.

8.3. High doses of carisoprodol can result in a fatal overdose due to respiratory depression [DEA 2019] but this is less common than in overdoses where carisoprodol, and other CNS depressants such as opioids and benzodiazepines, are also present [WHO 2023].

8.4. High doses of carisoprodol may result in the development of serotonin toxicity, and in severe cases it can be life threatening [Bramness 2005]. This toxicity occurs when individuals use medications and/or drugs that elevate extracellular levels of serotonin in the CNS and is greater if people use/misuse more than one medicine or drug that increases serotonin levels.

8.5. An analysis of 3,982 calls to Texas poisons centres between 1998 and 2003 involving carisoprodol assessed severity as: no effects – 861; mild effects – 2,036; moderate effects – 797; severe effects – 383; and death – 15 [Forrester 2006].

8.6. When carisoprodol is taken along with benzodiazepines and opioids, the risk of overdose is greater as all three classes of drug depress respiration [Horsfall 2017].

8.7. Long-term therapeutic use and/or excessive use/misuse of carisoprodol can lead to the development of physical dependence, and abrupt cessation or significant reduction in dose can precipitate an acute physical withdrawal syndrome. The symptoms of this acute physical withdrawal syndrome include anxiety, insomnia, tremors, muscle twitching and, in severe cases hallucinations and seizures [Reeves 2004; Eleid 2010; Ni 2007; Reeves 2007; Venugropal 2000; Vo 2017; Paul 2016]. Management of the acute physical withdrawal syndrome has typically involved benzodiazepines, in a similar way to the treatment of alcohol and/or benzodiazepine withdrawal.

9. Social Harms

9.1. There is little evidence currently available on the social harms caused by carisoprodol use. It is anticipated these will be similar to those of other controlled drugs that also act on the $GABA_A$ receptor such as barbiturates and benzodiazepines.

9.2. Carisoprodol may impair the ability to drive a motor vehicle which may put the individual or the public at risk. A study utilising the Norwegian Prescription Database, Road Accident Registry and Central Population Registry between April 2004 and September 2005 showed that having a prescription for carisoprodol dispensed increased the standardized incidence ratio for being involved in an accident with person injury to 3.7 (95% CI 2.9-4.8) in the first week after the date of dispensing, which was similar to that seen with diazepam (2.8; 2.2-3.6) [Bramness 2007]. Withdrawal of carisoprodol from the Norwegian market reduced the presence of carisoprodol in cases of impaired driving [Høiseth 2009].

9.3. The acute toxicity described with exposure to carisoprodol involves drowsiness, sedation and coma. Therefore, there is the potential that individuals may be exposed intentionally to carisoprodol by other individuals to facilitate crimes such as robbery, assault and sexual assault.

10. Prevalence

Prevalence in UK

10.1. From 2020 to 2024 there has been a year-on-year increase in the number of carisoprodol detections in samples obtained from drug seizures (Table 1). In that time there has been one report of a sample positive for meprobamate and one sample containing carisoprodol and meprobamate, indicating that carisoprodol, rather than meprobamate, is the main drug entering the UK illicit drug market.

Table 1. Detections of carisoprodol and/or meprobamate in drug seizures in UK (Redacted version)

Year	2020	2021	2022	2023	2024	2025*	Total
Carisoprodol	6	11	15	25	52	14	123
Carisoprodol and Meprobamate	0	0	0	1	0	0	1
Meprobamate	0	0	1	0	0	0	1

*Data for 2025 are for the first 6 months of the year.
Data were obtained from Border Force[1], Eurofins, National Crime Agency (NCA), Office for Health Improvement and Disparity (OHID) and Scottish Police Authority (SPA). The Scottish Prison Service, MANchester Drug Analysis and Knowledge Exchange (MANDRAKE), TicTac and Emerging Drugs and Technologies

[1] Border Force data has been redacted from the published version of this report.

programme (EDAT) provided nil returns for both carisoprodol and meprobamate in the period 2020-2025.

10.2. Funded by Public Health Wales, the WEDINOS programme offers laboratory testing of drug samples voluntarily submitted by the community. Samples are received anonymously by post from individuals or participating organisations, including substance misuse services, housing providers, young people's services, educational institutions, nightclubs and bars, mental health community teams, local authorities, ambulance services, and the police. Anonymised test results are made publicly available online. A potential limitation of the WEDINOS dataset is that it depends on voluntary sample submissions, which may not fully represent the wider drug landscape.

10.3. In September 2025, the publicly available 'sample results' section on the WEDINOS website was reviewed to identify analysed samples where carisoprodol and meprobamate had been detected. Since 2020, there has been an increase in the number of drug samples containing carisoprodol voluntarily submitted to WEDINOS for checking (Table 2).

10.4. Prior to 2024, samples submitted to WEDINOS (35 samples, all white tablets) contained only carisoprodol but in 2024/2025 a minority of white tablets tested (5 of 19 samples) also contained tapentadol. In contrast from 2020 to 2025 all red tablets ('red apples') (15 samples) contained carisoprodol and tapentadol.

Table 2. Detections of carisoprodol and/or meprobamate in samples voluntarily submitted for drug checking in UK

Year	2020	2021	2022	2023	2024	2025*	Total
Carisoprodol	2	13	18	12	15	7	68
Meprobamate	0	0	0	0	0	0	0

* Data for 2025 are up to the 2nd of September 2025.
Data obtained from WEDINOS.

10.5. Public Health Scotland (RADAR Intelligence Reports) recorded the appearance on 14 occasions from 2022-2025 of carisoprodol in pills ('somas') or carisoprodol and tapentadol ('red apples'). [PHS, RADAR quarterly report – July 2025]

10.6. The National Poisons Information Service (NPIS) provides information on the number of accesses to information held on its on-line poisons information database TOXBASE® and numbers and details of telephone enquiries made to the service by health professionals. These numbers reflect (but do not measure directly) the frequency of contacts between health professionals and patients presenting following suspected exposure. Limited data on characteristics of exposure are also available from call records. Online and app TOXBASE® accesses for carisoprodol and meprobamate are summarised in Table 3 below:

Table 3. Online and app TOXBASE® accesses for carisoprodol and meprobamate

Substance	Number of TOXBASE® accesses						
	2019	**2020**	**2021**	**2022**	**2023**	**2024**	**2025***
Carisoprodol	48	82	196	216	299	336	231
Meprobamate	10	0	6	5	19	23	7

***** 2025 data reflect 1st Jan- 31st March only

10.7. Very few telephone enquiries have been received by the NPIS concerning carisoprodol, with only two calls recorded between October and December 2024. No enquiries have been received in relation to meprobamate. Further details regarding the circumstances of use, clinical presentation, or patient outcomes are not available.

10.8. The Identification of novel psychoactive substances (IONA) study collected and analysed biological (blood and/or urine) samples from consenting patients attending over 30 participating emergency departments (EDs) in England, Wales and Scotland following illicit drug use. The study ran from May 2015 to March 2023, during which samples from a total of 1,815 patients were analysed. In a 2022 case, carisoprodol and meprobamate was detected in a 38-year-old female alongside multiple other substances including cocaine and nitazenes. In 2023, meprobamate was detected in an 18-year-old male who presented to the ED with reported exposure to etizolam and nitrazepam sourced from the internet. Additional substances detected included codeine and oxycodone.

10.9. A Surveillance Study of Illicit Substance Toxicity (ASSIST) is conducted at Queen Elizabeth University Hospital in NHS Greater Glasgow and Clyde. The study monitors drug trends and associated clinical features through the use of prospective surveillance of emergency department attendances due to acute illicit drug toxicity. Between August 2022 and May 2025, ASSIST identified 6,027 drug detections in 1,152 serum samples. Notably, two cases were positive for both carisoprodol and meprobamate - one in July 2023 and another in March 2024.

10.10. Between October 2013 and December 2023, there were 82,613 presentations reported to the Euro-DEN Plus registry, of which four involved the self-reported use of carisoprodol. These presentations were to London, UK (2 presentations in June 2019) and Oslo, Norway (one in April 2017 and one in January 2020). All four presentations included at least one other drug, all of which were sedatives: diazepam (2 presentations), alprazolam (1), GHB/GBL (1), methadone (1), tapentadol (1) and zolpidem (1) [Wood 2025].

10.11. The available data for the number of fatal overdoses in the UK (2020 - 2025) positive for carisoprodol alone or in combination with other drugs are provided in Table 4. These data do not however provide an accurate estimate of the involvement of

carisoprodol in drug overdoses in the UK because i) testing for carisoprodol and its metabolite, meprobamate, is not performed routinely by all forensic testing laboratories across the UK, ii) not all forensic testing laboratories responded to requests for data from ACMD and iii) there may be double counting in the data shown due to different organisations providing data from the same sources. What can be concluded is that deaths involving carisoprodol did occur between 2020 and 2024 and that some of these deaths also involved an opioid and/or a benzodiazepine.

Table 4. Fatal overdoses in UK in which carisoprodol and/or meprobamate were detected

Year	2020	2021	2022	2023	2024	2025	Total
Carisoprodol	0	0	1	2	0	0	3
Meprobamate	1	0	2	4	3	2	12
Carisoprodol and meprobamate	0	0	1	2	3	2	8
Carisoprodol and Meprobamate also containing an opioid and/or benzodiazepine	1	0	1	4	7	6	19

Data obtained from National Programme on Substance Use Mortality (NPSUM), National Records of Scotland (NRS), Organisation: Office of National Statistics (ONS), European-Wide, Monitoring, Analysis and Knowledge Dissemination on Novel/Emerging Psychoactive (EU-MADNESS), Public Health Scotland - Rapid Action Drug Alerts & Response (PHS RADAR), Laboratory of the Government Chemist (LGC) Standards and Office for Health Improvement and Disparity (OHID)

Prevalence in EU and North America

10.12. Between January 2018 and 7 September 2025, there were 1,463 reports involving carisoprodol and 757 involving meprobamate submitted to EudraVigilance, the European database that collects information on suspected adverse drug reaction reports for medicines authorised for use in Europe. This database was established in 2001 prior to the suspension of the European marketing authorisation for carisoprodol. Therefore, it is not possible to determine what proportion of these were therapeutic use related adverse drug reactions (similar to the UK MHRA Yellow Card reporting system) rather than non-medical use related.

10.13. In the USA, the FDA Adverse Event Reporting System (FAERS) recorded 39 overdoses involving carisoprodol between 2002 and 2023, 9 of which were fatal. Additionally, from 2003 to 2025, there were 116 deaths attributed to carisoprodol alone. Over the overlapping period (2003 to 2020), four overdose cases were reported involving carisoprodol in combination with other CNS depressants, 3 of which were fatal [FAERS 2025].

11. Conclusions

11.1. In the UK there are no legitimate uses of carisoprodol and meprobamate in human or veterinary medicine. There may however be visitors to the UK who have been prescribed carisoprodol in another country.

11.2. Acute administration of high doses of carisoprodol can produce hypomania, confusion, disorientation, agitation and partial amnesia. Very high doses may result in respiratory failure, coma and death. When carisoprodol is taken along with benzodiazepines and opioids the risk of overdose is higher as all three classes of drug depress respiration.

11.3. Prolonged use of carisoprodol induces addiction and dependence. In individuals who have been taking carisoprodol over a prolonged period, cessation of drug use will likely precipitate a withdrawal response.

11.4. Data from UK drug checking (seizures and voluntarily submitted samples) indicate that carisoprodol is available on the illicit drug market either on its own or in combination with the opioid tapentadol.

11.5. Evidence for the involvement of carisoprodol in non-fatal and fatal overdoses in the UK, alone or in combination with other drugs, is incomplete due to insufficient routine forensic testing for carisoprodol and its metabolite meprobamate.

11.6. Accurate information on the involvement of carisoprodol in drug overdoses in the UK will only be available when routine testing for carisoprodol and its metabolite meprobamate is performed across the country.

11.7. Because of the recent increase in the numbers of detections of carisoprodol in the illicit drug supply in the UK and the likelihood of further increases in its prevalence as well as the potential health and social harms associated with its use, the ACMD advises that control of carisoprodol via the Misuse of Drugs Act 1971 is necessary. Harms are considered to be broadly equivalent to those of other sedatives such as benzodiazepines and zopiclone.

12. Recommendations

Recommendation 1:
Given the harms associated with its use, carisoprodol should be added to Class C of the Misuse of Drugs Act 1971 and placed in Schedule 4 Part 1 of the Misuse of Drugs Regulations 2001 (as amended).

Leads: Home Office

Measure of outcome: The inclusion of the listed compounds in Class C of the Misuse of Drugs Act 1971, Schedule 4 of the Misuse of Drugs Regulations 2001.

Recommendation 2:

a) Toxicological testing for both carisoprodol and its metabolite meprobamate should be performed routinely in cases of non-fatal and fatal overdose. Non-comprehensive screening hinders our capacity to understand trends in drug deaths.
b) The presence or absence of either an opioid or a benzodiazepine should be recorded for all deaths involving carisoprodol and/or meprobamate.

Leads: Home Office, Coroners in England, Wales and Northern Ireland and Procurators Fiscal in Scotland.

Measure of outcome: All post-mortem toxicology to include carisoprodol and meprobamate analogues on the testing panel.

Recommendation 3:
Resources should be developed for health professionals and drug services on the danger of carisoprodol overdose, and increased risk when combined with benzodiazepines and opioids. These resources to include information on how overdoses that may involve carisoprodol alone or in combination with other drugs should be treated.

Leads: UK Health Security Agency (UKHSA), Office for Health Improvement and Disparities (OHID), Public Health Wales, Public Health Scotland, the Department of Health Northern Ireland, the Association of Directors of Public Health and National Poisons Information Service (NPIS).

Measure of outcome: Information available for health professionals, and drug services.

Recommendation 4:
Information on the harm of carisoprodol and the risks of overdose should be developed in formats suitable for people who are currently using carisoprodol, those who may use it in future, their family and friends as well as for the general public.

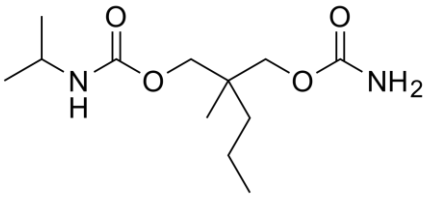
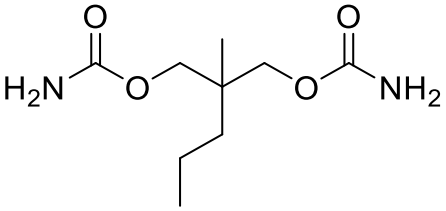
Information for people who are currently using carisoprodol or who may use it should highlight risk of addiction, dependence and overdose, and the increased harms of

combining with benzodiazepines and opioids. Information for the general public should be provided through updates to national drug information channels (FRANK, DAN 24/7, Know the Score).

Leads: UK Health Security Agency (UKHSA), Office for Health Improvement and Disparities (OHID), Public Health Wales, Public Health Scotland, the Department of Health Northern Ireland and the Association of Directors of Public Health.

Measure of outcome: Readily accessible information being available for people who use drugs, their families and friends, and the general public.

Annex A: Chemical structure of carisoprodol and meprobamate

Names (including IUPAC name)	Structure
• Carisoprodol • (RS)-[2-(carbamoyloxymethyl)-2-methylpentyl]-N-propan-2-ylcarbamate • N-isopropyl-2-methyl-2-propyl-1,3-propanediol dicarbamate • N-isopropylmeprobamate.	
• Meprobamate • [2-(carbamoyloxymethyl)-2-methyl-pentyl] carbamate	

Carisoprodol exists as a racemic mixture of two stereo isomers - (2S)-[2-(carbamoyloxymethyl)-2-methylpentyl] N-propan-2-ylcarbamate and (2R)-[2-(carbamoyloxymethyl)-2-methylpentyl] N-propan-2-ylcarbamate.

Annex B: Stakeholder Engagement and Data Request

Mortality and Chemical Analysis Data:

- Eurofins
- European-Wide, Monitoring, Analysis and Knowledge Dissemination on Novel/Emerging Psychoactive (EU-MADNESS)
- Forensic Science Northern Ireland (FSNI)
- Laboratory of the Government Chemist (LGC) Standards
- National Programme on Substance Abuse Deaths (NPSAD)
- National Records of Scotland (NRS)
- Crown Office and Procurator Fiscal Service (COPFS) and NHS Grampian
- Northern Ireland Statistics and Research Agency (NISRA)
- Office for National Statistics (ONS)
- Scottish Police Authority (SPA), Criminal Toxicology
- Scottish Police Authority (SPA), Post-Mortem Toxicology
- TICTAC
- Welsh Emerging Drugs and Identification of Novel Substances (WEDINOS)

Drug Seizure Data:

- Border Force
- Defence Science and Technology Laboratory (DSTL)
- Drugs Team at Public Health Scotland (PHS) (RADAR)
- EDAT (Emerging Drugs and Technologies programme)
- Manchester Drug Analysis and Knowledge Exchange (MANDRAKE)
- National Crime Agency (NCA)
- Office for Health Improvement and Disparities (OHID)
- Scottish Police Authority (SPA), Drug Detections
- Scottish Prison Service, University of Dundee's Leverhulme Research Centre

Drug Poisoning Data:

- Identification of Novel Psychoactive Substances (IONA) Study
- National Poisons Information Service (NPIS)

Legitimate Use:

- Medicines and Healthcare products Regulatory Agency (MHRA)

Annex C: List of Abbreviations Used in This Report

ACMD	Advisory Council on The Misuse of Drugs
CDSA	Controlled Drugs and Substances Act
CNS	Central Nervous System
CYP2C19	Cytochrome P450 enzyme
DEA	Drug Enforcement Administration
DSTL	Defence Science and Technology Laboratory
ECDD	Expert Committee on Drug Dependence
ED	Emergency Department
EUDA	European Union Drugs Agency
EU-MADNESS	EUropean-wide, Monitoring, Analysis and knowledge Dissemination on Novel/Emerging pSychoactiveS
GABA	Gamma-Aminobutyric Acid
LC	Liquid Chromatography
IONA	Identification Of Novel Psychoactive Substances
IUPAC	International Union of Pure and Applied Chemistry
MANDRAKE	Manchester Drug Analysis and Knowledge Exchange
MDA	Misuse Of Drugs Act 1971
MDR	Misuse Of Drugs Regulations 2001
MHRA	Medicines and Healthcare products Regulatory Agency
NCA	National Crime Agency
NISRA	Northern Ireland Statistics and Research Agency
NPIS	National Poisons Information Service
NPS	Novel Psychoactive Substances
NPSUM	The National Programme on Substance Use Mortality
NRS	National Records of Scotland
OHID	Office for Health Improvement and Disparities
ONS	Office for National Statistics
PHS	Public Health Scotland
PSA	Psychoactive Substances Act 2016
SPA	Scottish Police Authority
UNODC	United Nations Office on Drugs and Crime
WEDINOS	Welsh Emerging Drug & Identification Of Novel Substances
WHO	World Health Organization

Annex D: Chair and Members of ACMD Carisoprodol working group.

Chair of Working Group	
Professor Graeme Henderson	**Honorary Professor of Pharmacology, School of Physiology, Pharmacology & Neuroscience, University of Bristol**
Members of Working Group	
Dr John Corkery	**Associate Professor in Pharmacy Practice at University of Hertfordshire; mortality and epidemiological lead for EU-MADNESS project**
Professor Amira Guirguis	**Professor of Pharmacy, MPharm Programme Director and Deputy Pro Vice Chancellor at Swansea University**
Dr Hilary Hamnett	**Associate Professor in Forensic Science and Forensic Toxicologist at the University of Lincoln**
Professor Fabrizio Schifano*	**Professor of Clinical Pharmacology, Centre for Health Services and Clinical Research, University of Hertfordshire**
Mr Ric Treble	**Retired Laboratory of the Government Chemist (LGC) expert**
Professor David Wood	**Professor of Clinical Toxicology and Consultant Physician, Guy's and St Thomas' NHS Foundation Trust and King's College London**

***denotes co-opted member of the ACMD working group**

Acknowledgements:

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Annex E: ACMD Novel Psychoactive Substances Committee membership, at time of publication

Chair of NPS Committee	
Professor Simon Thomas	**NPS Committee Chair, Emeritus Professor of Clinical Pharmacology and Therapeutics, Newcastle University**
Members of NPS Committee	
Mr Paul Bunt**	**Director of Casterton Event Solutions Ltd, Former Drug Strategy Manager for Avon and Somerset Constabulary**
Mr Peter Cain**	**Drugs Scientific Advisor, Eurofins Forensic Services**
Dr Caroline Copeland	**Senior Lecturer in Pharmacology and Toxicology, King's College London. Director, National Programme on Substance Use Mortality**
Dr John Corkery**	**Associate Professor in Research (Psychoactive Substances' Epidemiology, Toxicology and Mortality), University of Hertfordshire; mortality. Epidemiological lead for EU-MADNESS project**
Professor Colin Davidson	**Professor of Neuropharmacology, University of Central Lancashire**
Professor Amira Guirguis	**Professor of Pharmacy, MPharm Programme Director and Deputy Pro Vice Chancellor at Swansea University**
Dr Hilary Hamnett	**Associate Professor in Forensic Science and Forensic Toxicologist at the University of Lincoln**
Professor Graeme Henderson	**Honorary Professor of Pharmacology, School of Physiology, Pharmacology & Neuroscience, University of Bristol**
Dr Simon Hill	**Consultant in Clinical Pharmacology, Therapeutics and Clinical Toxicology. Honorary Clinical Senior Lecturer, Newcastle University**

Professor Stephen Husbands	**Professor of Medicinal Chemistry, University of Bath**
Professor Roger Knaggs	**Professor of Pain Management, University of Nottingham and Specialist Pharmacist in Pain Management, Primary Integrated Community Services**
Professor Fiona Measham**	**Professor and chair in criminology at the University of Liverpool; co-founder and co-director, the Loop**
Dr Richard Stevenson	**Emergency Medicine Consultant, Glasgow Royal Infirmary**
Mr Ric Treble**	**Retired Laboratory of the Government Chemist (LGC) expert**
Professor David Wood	**Professor of Clinical Toxicology and Consultant Physician, Guy's and St Thomas' NHS Foundation Trust and King's College London**

****denotes co-opted member of ACMD Novel Psychoactive Substances Committee**

Annex F: ACMD membership at the time of publication

Chair of ACMD	
Professor Owen Bowden-Jones, CBE	**Consultant psychiatrist, Central North-West London NHS Foundation Trust**
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Professor Anne Campbell, MBE	**Professor in substance use at Queens University Belfast School of Social Sciences, Education and Social Work**
Dr Caroline Copeland	**Senior Lecturer in Pharmacology and Toxicology, King's College London and Director, National Programme on Substance Use Mortality**
Professor Colin Davidson	**Professor of Neuropharmacology, University of Central Lancashire**
Professor Karen Ersche	**Professor of Addiction Neuroscience, University of Cambridge**
Mr Mohammed Fessal	**Chief pharmacist, Change Grow Live**
Professor Amira Guirguis	**Professor of Pharmacy, MPharm Programme Director at Swansea University Medical School**
Dr Hilary Hamnett	**Associate Professor in Forensic Science and Forensic Toxicologist at the University of Lincoln**
Mr Jason Harwin	**Director and Co-founder of E-T-E Solutions Limited**
Professor Katy Holloway	**Professor of Criminology at the University of South Wales**
Professor Graeme Henderson	**Honorary Professor of Pharmacology, School of Physiology, Pharmacology & Neuroscience, University of Bristol**
Dr Carole Hunter	**Retired. Chair SDF Board and Doping Control Officer UK Antidoping**
Professor Stephen Husbands	**Professor of Medicinal Chemistry, University of Bath**

Professor Sunjeev Kamboj	**Professor of Translational Clinical Psychology Research Department of Clinical, Educational and Health Psychology, University College London**
Professor Roger Knaggs	**Professor of Pain Management, University of Nottingham and Specialist Pharmacist in Pain Management, Primary Integrated Community Services**
Mrs Sapna Lewis	**Senior Lawyer, Welsh Government Legal Services Department**
Dr Lorna Nisbet	**Senior Lecturer and Principal Investigator for Forensic Toxicology at the Leverhulme Research Centre for Forensic Science, University of Dundee**
Mr Jon Privett	**Detective Sergeant, Metropolitan Police Service**
Mrs Fiona Spargo-Mabbs OBE	**Director and Founder, Daniel Sargo-Mabbs Foundation and Chair, Drug Education Forum**
Dr Richard Stevenson	**Emergency medicine consultant, Glasgow Royal Infirmary**
Professor Paul Stokes	**Professor of mood disorders and psychopharmacology, King's College London**
Professor Harry Sumnall	**Professor in Substance Use, Liverpool John Moores University (LJMU)**
Professor Simon Thomas	**Emeritus professor of clinical pharmacology and therapeutics, Newcastle University**
Professor Derek Tracy	**Chief Medical Officer, South London and Maudsley NHS Foundation Trust**
Ms Rosalie Weetman	**Group Manager (Public Health Principal) Inclusion Public Health at Derbyshire County Council; currently on secondment to Office for Health Improvement and Disparities, as programme manager, Drug and Alcohol Improvement Support Team**
Professor David Wood	**Professor of Clinical Toxicology and Consultant Physician, Guy's and St Thomas' NHS Foundation Trust and King's College London**

Annex G: Quality Of Evidence

This report draws on evidence from peer-reviewed literature (UK and international publications) and government reports and considered international approaches when drafting its recommendations. The evidence gathered was considered in accordance with the ACMD's standard operating procedure for quality of evidence [ACMD, 2024b].

To evidence the identification and prevalence in the UK of carisoprodol, the ACMD's NPS Committee wrote to stakeholders requesting available data on carisoprodol and its metabolite meprobamate. Details of organisations contacted are provided in Annex B.

The ACMD also sought information on medicinal use of carisoprodol from the Medicines and Healthcare products Regulatory Agency (MHRA).

It is important to note that forensic analysis is inconsistent across the UK and as a result, the presence of carisoprodol and meprobamate in samples may not be identified. As a result, information being fed into reporting agencies that were approached may not be representative.

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