

# Search Results

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## Search History

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1. MEDLINE; exp SUBSTANCE-RELATED DISORDERS/; 190082 results.
2. MEDLINE; addict\*.ti,ab; 30846 results.
3. MEDLINE; 1 OR 2; 200293 results.
4. MEDLINE; exp GREAT BRITAIN/; 259597 results.
5. MEDLINE; "United Kingdom".ti,ab; 19970 results.
6. MEDLINE; "Great Britain".ti,ab; 5453 results.
7. MEDLINE; "England".ti,ab; 25898 results.
8. MEDLINE; "Scotland".ti,ab; 9718 results.
9. MEDLINE; "Wales".ti,ab; 13517 results.
10. MEDLINE; UK.ti,ab; 48994 results.
11. MEDLINE; GB.ti,ab; 5203 results.
12. MEDLINE; ireland.ti,ab; 18758 results.
13. MEDLINE; IRELAND/; 10223 results.
14. MEDLINE; "British Isles".ti,ab; 627 results.
15. MEDLINE; "Channel islands".ti,ab; 78 results.
16. MEDLINE; 4 OR 5 OR 6 OR 7 OR 8 OR 9 OR 10 OR 11 OR 12 OR 13 OR 14 OR 15; 334744 results.
17. MEDLINE; 3 AND 16; 6079 results.

**1. Case-control association study of WLS variants in opioid and cocaine addicted populations.**

<b>Citation:</b>	Psychiatry Research, June 2013, vol./is. 208/1(62-6), 0165-1781;1872-7123 (2013 Jun 30)
<b>Author(s):</b>	Crist RC; Ambrose-Lanci LM; Zeng A; Yuan C; Kampman KM; Pettinati HM; Oslin DW; O'Brien CP; Ferraro TN; Doyle GA; Lohoff FW; Berrettini WH
<b>Institution:</b>	Center for Neurobiology and Behavior, Department of Psychiatry, University of Pennsylvania, Perelman School of Medicine, Philadelphia, PA 19104, USA. crist@mail.med.upenn.edu
<b>Language:</b>	English
<b>Abstract:</b>	The opioid receptor family is involved in the development and maintenance of drug addiction. The mu-opioid receptor (MOR) mediates the rewarding effects of multiple drugs, including opiates and cocaine. A number of proteins interact with MOR, potentially modulating MOR function and altering the physiological consequences of drug use. These mu-opioid receptor interacting proteins (MORIPs) are potential therapeutic targets for the treatment of addiction. The Wntless (WLS) protein was recently identified as a MORIP in a yeast two-hybrid screen. In this study, we conducted a case-control association analysis of 16 WLS genetic variants in opioid and cocaine addicted individuals of both African-American (opioid n=336, cocaine n=908) and European-American (opioid n=335, cocaine n=336) ancestry. Of the analyzed SNPs, three were nominally associated with opioid addiction and four were nominally associated with cocaine addiction. None of these associations were significant following multiple testing correction. These data suggest that the common variants of WLS analyzed in this study are not associated with opioid or cocaine addiction. However, this study does not exclude the possibilities that rare variants in WLS may affect susceptibility to drug addiction, or that common variants with small effect size may fall below the detection level of our analysis. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.
<b>Country of Publication:</b>	Ireland
<b>CAS Registry Number:</b>	0 (GPR177 protein, human); 0 (Intracellular Signaling Peptides and Proteins); 0 (Receptors, G-Protein-Coupled)
<b>Publication Type:</b>	Journal Article; Research Support, N.I.H., Extramural
<b>Subject Headings:</b>	"African Americans/ge [Genetics]" "African Americans/px [Psychology]" Case-Control Studies "*Cocaine-Related Disorders/ge [Genetics]" "European Continental Ancestry Group/ge [Genetics]" "European Continental Ancestry Group/px [Psychology]" Female Genetic Association Studies "*Genetic Predisposition to Disease/ge [Genetics]" Genotype Humans "*Intracellular Signaling Peptides and Proteins/ge [Genetics]" Male "*Opioid-Related Disorders/ge [Genetics]" "Polymorphism Single Nucleotide/ge [Genetics]" "*Receptors G-Protein-Coupled/ge [Genetics]"
<b>Source:</b>	MEDLINE
<b>Full Text:</b>	Available from <i>Elsevier</i> in <i>Psychiatry Research</i>

**2. Phosphoproteomic analysis of the striatum from pleiotrophin knockout and midkine knockout mice treated with cocaine reveals regulation of oxidative stress-related proteins potentially underlying cocaine-induced neurotoxicity and neurodegeneration.**

**Citation:** Toxicology, December 2013, vol./is. 314/1(166-73), 0300-483X;1879-3185 (2013 Dec 6)

**Author(s):** Vicente-Rodriguez M; Gramage E; Herradon G; Perez-Garcia C

**Institution:** Pharmacology Lab, Department of Pharmaceutical and Health Sciences, Facultad de Farmacia, Universidad CEU San Pablo, Madrid, Spain.

**Language:** English

**Abstract:** The neurotrophic factors pleiotrophin (PTN) and midkine (MK) are highly upregulated in different brain areas relevant to drug addiction after administrations of different drugs of abuse, including psychostimulants. We have previously demonstrated that PTN and MK modulate amphetamine-induced neurotoxicity and that PTN prevents cocaine-induced cytotoxicity in NG108-15 and PC12 cells. In an effort to dissect the different mechanisms of action triggered by PTN and MK to exert their protective roles against psychostimulant neurotoxicity, we have now used a proteomic approach to study protein phosphorylation, in which we combined phosphoprotein enrichment, by immobilized metal affinity chromatography (IMAC), with two-dimensional gel electrophoresis and mass spectrometry, in order to identify the phosphoproteins regulated in the striatum of PTN knockout, MK knockout and wild type mice treated with a single dose of cocaine (15mg/kg, i.p.). We identified 7 differentially expressed phosphoproteins: 5'(3')-deoxyribonucleotidase, endoplasmic reticulum resident protein 60 (ERP60), peroxiredoxin-6 (PRDX6), glutamate dehydrogenase 1 (GLUD1), aconitase and two subunits of hemoglobin. Most of these proteins are related to neurodegeneration processes and oxidative stress and their variations specially affect the PTN knockout mice, suggesting a protective role of endogenous PTN against cocaine-induced neural alterations. Further studies are needed to validate these proteins as possible targets against neural alterations induced by cocaine. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Carrier Proteins); 0 (Cytokines); 0 (Phosphoproteins); 134034-50-7 (pleiotrophin); 137497-38-2 (midkine); 42HK56048U (Tyrosine); I5Y540LHVR (Cocaine)

**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** [Animals](#)  
[Blotting Western](#)  
["\\*Carrier Proteins/ge \[Genetics\]"](#)  
["\\*Carrier Proteins/ph \[Physiology\]"](#)  
[Chromatography Affinity](#)  
["\\*Cocaine/pd \[Pharmacology\]"](#)  
["\\*Cocaine/to \[Toxicity\]"](#)  
["\\*Cytokines/ge \[Genetics\]"](#)  
["\\*Cytokines/ph \[Physiology\]"](#)  
[Electrophoresis Polyacrylamide Gel](#)  
[Mice](#)  
[Mice Knockout](#)  
["\\*Neostriatum/me \[Metabolism\]"](#)  
["\\*Neurodegenerative Diseases/ci \[Chemically Induced\]"](#)  
["\\*Neurodegenerative Diseases/ge \[Genetics\]"](#)  
["\\*Neurodegenerative Diseases/pp \[Physiopathology\]"](#)  
["\\*Neurotoxicity Syndromes/ge \[Genetics\]"](#)  
["\\*Neurotoxicity Syndromes/pp \[Physiopathology\]"](#)  
["\\*Oxidative Stress/de \[Drug Effects\]"](#)  
["\\*Phosphoproteins/ge \[Genetics\]"](#)  
["\\*Phosphoproteins/me \[Metabolism\]"](#)  
[Phosphorylation](#)  
["\\*Proteomics/mt \[Methods\]"](#)  
[Spectrometry Mass Matrix-Assisted Laser Desorption-Ionization](#)  
["\\*Tyrosine/me \[Metabolism\]"](#)

**Source:** MEDLINE

**Full Text:** Available from *Elsevier* in [Toxicology](#)

### 3. Concentrations of drugs determined in blood samples collected from suspected drugged drivers in England and Wales.

<b>Citation:</b>	Journal of Forensic & Legal Medicine, May 2013, vol./is. 20/4(278-89), 1752-928X;1878-7487 (2013 May)
<b>Author(s):</b>	Burch HJ; Clarke EJ; Hubbard AM; Scott-Ham M
<b>Institution:</b>	The Forensic Science Service, London Laboratory, 109 Lambeth Road, London SE1 7LP, UK.
<b>Language:</b>	English
<b>Abstract:</b>	This communication reports the blood concentrations of alcohol and drugs from 376 cases of alleged driving under the influence of drugs analysed at the Forensic Science Service Chorley and London laboratories between February 2010 and March 2011. The samples were analysed for alcohol, amphetamine, benzodiazepines, cocaine, MDMA, opiates, -hydroxybutyrate (GHB), ketamine, methadone and methylenedioxymethamphetamine (the 4-isomer of which is known as mephedrone). The results were interpreted with respect to the number and type of drugs of abuse detected and the concentrations measured. Alcohol was quantified in 113 cases (30%), and of these a level in excess of the prescribed UK limit for driving of 80 mg% was present in 90 cases. In 80 cases, only the concentration of alcohol was measured, the concentrations of both drugs and alcohol were measured in 33 cases. In the remaining 263 cases, only the concentrations of relevant drugs of abuse were measured. The most common drug of abuse quantified was cocaine which was detected in 92 cases, either as the active drug or as its major metabolite benzoylecgonine, followed by diazepam which was quantified in 76 cases. Concentrations of some new drugs, and drugs rarely reported in driving under the influence cases are also presented. Crown Copyright 2012. Published by Elsevier Ltd. All rights reserved.
<b>Country of Publication:</b>	England
<b>CAS Registry Number:</b>	0 (Central Nervous System Depressants); 0 (Narcotics); 3K9958V90M (Ethanol); 44RAL3456C (Methamphetamine); 502-85-2 (Sodium Oxybate); 535318I6YS (benzoylecgonine); 67220MCM01 (Nordazepam); 690G0D6V8H (Ketamine); 8BA8T27317 (mephedrone); CK833KGX7E (Amphetamine); I5Y540LHVR (Cocaine); KE1SEN21RM (N-Methyl-3,4-methylenedioxyamphetamine); Q3JTX2Q7TU (Diazepam); UC6VBE7V1Z (Methadone)
<b>Publication Type:</b>	Journal Article
<b>Subject Headings:</b>	<a href="#">Adolescent</a> <a href="#">Adult</a> <a href="#">"Amphetamine/bl [Blood]"</a> <a href="#">"*Automobile Driving/lj [Legislation and Jurisprudence]"</a> <a href="#">"Central Nervous System Depressants/bl [Blood]"</a> <a href="#">Chromatography Liquid</a> <a href="#">"Cocaine/aa [Analogues and Derivatives]"</a> <a href="#">"Cocaine/bl [Blood]"</a> <a href="#">"Diazepam/bl [Blood]"</a> <a href="#">England</a> <a href="#">"Ethanol/bl [Blood]"</a> <a href="#">Female</a> <a href="#">Forensic Toxicology</a> <a href="#">Gas Chromatography-Mass Spectrometry</a> <a href="#">Humans</a> <a href="#">"Ketamine/bl [Blood]"</a> <a href="#">Male</a> <a href="#">"Methadone/bl [Blood]"</a> <a href="#">"Methamphetamine/aa [Analogues and Derivatives]"</a> <a href="#">"Methamphetamine/bl [Blood]"</a> <a href="#">"N-Methyl-3 4-methylenedioxyamphetamine/bl [Blood]"</a> <a href="#">"Narcotics/bl [Blood]"</a> <a href="#">"Nordazepam/bl [Blood]"</a>

"Sodium Oxybate/bl [Blood]"  
 \*Substance Abuse Detection  
 "\*Substance-Related Disorders/bl [Blood]"  
 "\*Substance-Related Disorders/di [Diagnosis]"  
 Wales  
 Young Adult

**Source:** MEDLINE

**Full Text:** Available from *Elsevier* in *Journal of Forensic and Legal Medicine*

#### 4. A review of drug-facilitated sexual assault evidence: an Irish perspective.

**Citation:** Journal of Forensic & Legal Medicine, May 2013, vol./is. 20/4(189-97), 1752-928X;1878-7487 (2013 May)

**Author(s):** McBrierty D; Wilkinson A; Tormey W

**Institution:** Chemical Pathology, Beaumont Hospital, Dublin 9, Ireland.  
 dermotmcbrierty@beaumont.ie

**Language:** English

**Abstract:** Drug-facilitated sexual assault (DFSA) is prevalent in Western society. There is a significant degree of confusion regarding the definition and prevalence of DFSA. It is a subject with medical, scientific and legal aspects. These facets are explored in this review through a detailed examination of published data. The legal issues are defined in the context of the Irish judicial system. Several key case-law studies are presented to aid in understanding unresolved difficulties that persist in this complex field of forensics. The aim of this paper is to aid individuals from disparate disciplines to increase their evidence base in the complex and evolving issue of DFSA. Copyright 2012 Elsevier Ltd and Faculty of Forensic and Legal Medicine. All rights reserved.

**Country of Publication:** England

**Publication Type:** Journal Article; Legal Cases; Review

**Subject Headings:** Adolescent  
 Adult  
 Age Distribution  
 Aged  
 "Alcohol Drinking/ep [Epidemiology]"  
 "Crime Victims/lj [Legislation and Jurisprudence]"  
 "Domestic Violence/sn [Statistics and Numerical Data]"  
 Female  
 Humans  
 Immunoassay  
 Ireland  
 Male  
 "Mental Competency/lj [Legislation and Jurisprudence]"  
 Middle Aged  
 Prevalence  
 Sex Distribution  
 "\*Sex Offenses/lj [Legislation and Jurisprudence]"  
 "\*Sex Offenses/sn [Statistics and Numerical Data]"  
 "Sex Offenses/td [Trends]"  
 Substance Abuse Detection  
 "\*Substance-Related Disorders/co [Complications]"  
 "Substance-Related Disorders/di [Diagnosis]"  
 "Unconsciousness/co [Complications]"  
 Young Adult

**Source:** MEDLINE

**Full Text:** Available from *Elsevier* in *Journal of Forensic and Legal Medicine*

### 5. The effect of mitragynine on cAMP formation and mRNA expression of mu-opioid receptors mediated by chronic morphine treatment in SK-N-SH neuroblastoma cell.

**Citation:** Journal of Ethnopharmacology, June 2013, vol./is. 148/1(135-43), 0378-8741;1872-7573 (2013 Jun 21)

**Author(s):** Jamil MF; Subki MF; Lan TM; Majid MI; Adenan MI

**Institution:** Malaysian Institute Pharmaceuticals and Nutraceuticals (IPharm), Ministry of Science, Technology and Innovation (MOSTI) Jalan Bukit Gambir, 11700 Gelugor Pulau Pinang, Malaysia.

**Language:** English

**Abstract:** UNLABELLED: ETHOPHARMACOLOGICAL RELEVANCE: Mitragynine is an indole alkaloid compound of *Mitragyna speciosa* (*M. speciosa*) Korth. (Rubiaceae). This plant is native to the southern regions of Thailand and northern regions of Malaysia and is frequently used to manage the withdrawal symptoms in both countries. AIM OF STUDY: To investigate the effect of mitragynine after chronic morphine treatment on cyclic AMP (cAMP) level and mRNA expression of mu-opioid receptor (MOR) in human neuroblastoma SK-N-SH cell. METHOD AND MATERIALS: Mitragynine was isolated from the *Mitragyna speciosa* plant using the acid-base extraction method. The cAMP level upon forskolin stimulation in the cells was determined using the Calbiochem() Direct Immunoassay Kit. The mRNA expression of the MOR was carried out using quantitative RT-PCR. RESULT: Cotreatment and pretreatment of morphine and mitragynine significantly reduced the production of cAMP level at a lower concentration of mitragynine while the higher concentration of this compound could lead to the development of tolerance and dependence as shown by the increase of the cAMP level production in forskolin stimulation. In MOR mRNA expression study, cotreatment of morphine with mitragynine significantly reduced the down-regulation of MOR mRNA expression as compared to morphine treatment only. CONCLUSION: These finding suggest that mitragynine could possibly avoid the tolerance and dependence on chronic morphine treatment by reducing the up-regulation of cAMP level as well as reducing the down-regulation of MOR at a lower concentration of mitragynine. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Analgesics, Opioid); 0 (RNA, Messenger); 0 (Receptors, Opioid, mu); 0 (Secologanin Tryptamine Alkaloids); 5688UTC01R (Tretinoin); 76I7G6D29C (Morphine); E0399OZS9N (Cyclic AMP); EP479K822J (mitragynine)

**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** ["\\*Analgesics Opioid/ad \[Administration and Dosage\]"](#)  
[Cell Differentiation](#)  
[Cell Line Tumor](#)  
["\\*Cyclic AMP/me \[Metabolism\]"](#)  
[Humans](#)  
[Mitragyna](#)  
["\\*Morphine/ad \[Administration and Dosage\]"](#)  
[Plant Leaves](#)  
["RNA Messenger/me \[Metabolism\]"](#)  
["\\*Receptors Opioid mu/ge \[Genetics\]"](#)  
["\\*Secologanin Tryptamine Alkaloids/ad \[Administration and Dosage\]"](#)  
[Substance-Related Disorders](#)  
["Tretinoin/ad \[Administration and Dosage\]"](#)

**Source:** MEDLINE

### 6. A randomized controlled trial of a smoking cessation intervention conducted among prisoners.

**Citation:** Addiction, May 2013, vol./is. 108/5(966-74), 0965-2140;1360-0443 (2013 May)

**Author(s):** Richmond R; Indig D; Butler T; Wilhelm K; Archer V; Wodak A

**Institution:** School of Public Health and Community Medicine, UNSW, Sydney, NSW, Australia.

**Language:** English

**Abstract:** AIM: To evaluate the efficacy of nortriptyline (NOR) added to a multi-component smoking cessation intervention, which included cognitive-behavioural therapy (CBT) and provision of nicotine replacement therapy (NRT).DESIGN: Randomized controlled trial (RCT) comparing two study groups with blinded follow-up at 3, 6 and 12 months. Both groups received a multi-component smoking cessation intervention comprising two half-hour individual sessions of CBT and NRT with either active NOR or placebo.SETTING: Prisons in New South Wales (17) and Queensland (one), Australia.PARTICIPANTS: A total of 425 male prisoners met inclusion criteria and were allocated to either treatment (n=206) or control group (n=219).MEASUREMENTS: Primary end-points at 3, 6 and 12 months were continuous abstinence, point prevalence abstinence and reporting a 50% reduction in smoking. Smoking status was confirmed by expired carbon monoxide, using a cut-point of <10 parts per million.FINDINGS: Participants' demographics and baseline tobacco use were similar in treatment and control groups. Based on an intention-to-treat analysis, continuous abstinence between the treatment and control groups was not significantly different at 3 months (23.8 versus 16.4%), 6 months (17.5 versus 12.3%) and 12 months (11.7 versus 11.9%).CONCLUSION: Adding nortriptyline to a smoking cessation treatment package consisting of behavioural support and nicotine replacement therapy does not appear to improve long-term abstinence rates in male prisoners. 2012 The Authors, *Addiction* 2012 Society for the Study of Addiction.

**Country of Publication:** England

**CAS Registry Number:** 0 (Adrenergic Uptake Inhibitors); BL03SY4LXB (Nortriptyline)

**Publication Type:** Journal Article; Multicenter Study; Randomized Controlled Trial

**Subject Headings:** [Adolescent](#)  
["\\*Adrenergic Uptake Inhibitors/tu \[Therapeutic Use\]"](#)  
[Adult](#)  
[Aged](#)  
[Australia](#)  
[Cognitive Therapy](#)  
[Follow-Up Studies](#)  
[Humans](#)  
[Male](#)  
[Middle Aged](#)  
["\\*Nortriptyline/tu \[Therapeutic Use\]"](#)  
["Prisoners/px \[Psychology\]"](#)  
["\\*Smoking/dt \[Drug Therapy\]"](#)  
["Smoking/th \[Therapy\]"](#)  
["\\*Smoking Cessation/mt \[Methods\]"](#)  
[Tobacco Use Cessation Products](#)  
[Treatment Outcome](#)  
[Young Adult](#)

**Source:** MEDLINE

**Full Text:** Available from *Wiley* in *Addiction*; Note: ; Collection notes: Offsite access: Type "Homerton" into box entitled "Institution Name" at lower right of the screen and select "Homerton Hospital"

#### 7. Lost in translation? Learning from the opioid epidemic in the USA.

**Citation:** Anaesthesia, December 2013, vol./is. 68/12(1215-9), 0003-2409;1365-2044 (2013 Dec)

**Author(s):** Weisberg D; Stannard C

**Institution:** Yale University School of Medicine, New Haven, CT, USA.

**Language:** English

**Country of Publication:** England

**CAS Registry Number:** 0 (Analgesics, Opioid)  
**Publication Type:** Editorial  
**Subject Headings:** "Analgesics Opioid/ae [Adverse Effects]"  
 "\*Analgesics Opioid/tu [Therapeutic Use]"  
 Great Britain  
 Humans  
 "\*Opioid-Related Disorders/et [Etiology]"  
 "\*Pain/dt [Drug Therapy]"  
 "Pain/ec [Economics]"  
 "Pain Management/ec [Economics]"  
 "\*Pain Management/mt [Methods]"  
 "Prescription Drug Misuse/ec [Economics]"  
 "\*Prescription Drug Misuse/sn [Statistics and Numerical Data]"  
 United States

**Source:** MEDLINE

**Full Text:** Available from Wiley in *Anaesthesia*; Note: ; Collection notes: Offsite access: Type "Homerton" into box entitled "Institution Name" at lower right of the screen and select "Homerton Hospital"  
 Available from *Anaesthesia* in *Newcomb Library & Information Service*

#### 8. Brain nitric oxide metabolites in rats preselected for nicotine preference and intake.

**Citation:** Neuroscience Letters, June 2013, vol./is. 545/(102-6), 0304-3940;1872-7972 (2013 Jun 17)

**Author(s):** Keser A; Nesil T; Kanit L; Pogun S

**Institution:** Ege University, Center for Brain Research, Bornova, Izmir, Turkey.

**Language:** English

**Abstract:** Nicotine addiction is a serious health problem resulting in millions of preventable deaths worldwide. The gas messenger molecule nitric oxide (NO) plays a critical role in addiction, and nicotine increases nitric oxide metabolites (NOx) in the brain. Understanding the factors which underlie individual differences in nicotine preference and intake is important for developing effective therapeutic strategies for smoking cessation. The present study aimed to assess NO activity, by measuring its stable metabolites, in three brain regions that express high levels of nicotinic acetylcholine receptors in rats preselected for nicotine preference. Rats (n=88) were exposed to two-bottle, free choice of oral nicotine/water starting either as adolescents or adults; control animals received only water under identical conditions. Following 12 or six weeks of exposure, levels of NOx (nitrite+nitrate), were determined in the hippocampus, frontal cortex, and amygdala. Since the rats were singly housed during oral nicotine treatment, naive rats were also included in the study to evaluate the effect of isolation stress. Isolation stress increased NOx in the hippocampus. Nicotine preference did not have a significant effect on NO activity, but rats with adolescent exposure had higher NOx levels in the frontal cortex compared to adult-onset rats. Our findings suggest that nicotine exposure during adolescence, regardless of the amount of nicotine consumed, results in higher NO activity in the frontal cortex of rats, which persists through adulthood. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 31C4KY9ESH (Nitric Oxide); 54-11-5 (Nicotine)

**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** Administration Oral  
 Animals  
 "Brain/de [Drug Effects]"  
 "\*Brain/me [Metabolism]"  
 "\*Drug-Seeking Behavior/de [Drug Effects]"  
 Female

Male  
 "\*Nicotine/to [Toxicity]"  
 "\*Nitric Oxide/me [Metabolism]"  
 Rats  
 Rats Sprague-Dawley  
 "Tissue Distribution/de [Drug Effects]"  
 "Tobacco Use Disorder/et [Etiology]"  
 "\*Tobacco Use Disorder/me [Metabolism]"

**Source:** MEDLINE

### 9. Peripheral toxicity in crack cocaine use disorders.

**Citation:** Neuroscience Letters, June 2013, vol./is. 544/(80-4), 0304-3940;1872-7972 (2013 Jun 7)

**Author(s):** Narvaez JC; Magalhaes PV; Fries GR; Colpo GD; Czepielewski LS; Vianna P; Chies JA; Rosa AR; Von Diemen L; Vieta E; Pechansky F; Kapczinski F

**Institution:** Bipolar Disorders Program & INCT Translational Medicine, Hospital de Clinicas de Porto Alegre, Universidade Federal do Rio Grande do Sul, Porto Alegre, Brazil.

**Language:** English

**Abstract:** A growing body of evidence suggests that crack cocaine misuse has widespread systemic and cognitive consequences, but little attention has been given to its systemic pathophysiology. We report here changes in inflammation markers, oxidative damage and brain derived neurotrophic factor in a sample of outpatients with crack cocaine use disorders. Fifty-three outpatients were recruited for this cross-sectional study and matched with fifty control subjects. The focus of this report is in between group differences in cytokines, oxidative damage and brain-derived neurotrophic factor (BDNF). Crack cocaine use was associated with higher BDNF levels when compared to controls, present only in those who used crack cocaine in the last month. Patients also had higher circulating levels of IL-1beta, TNF-alpha and IL-10 when compared to controls. There were no significant differences in oxidative damage between patients and controls. These results represent a first demonstration that crack cocaine use disorders entail an activation of the reward, immune and inflammatory systems. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Biological Markers); 0 (Crack Cocaine); 0 (Cytokines)

**Publication Type:** Comparative Study; Journal Article

**Subject Headings:** Adult  
 "Biological Markers/bl [Blood]"  
 "Brazil/ep [Epidemiology]"  
 Causality  
 "\*Cocaine-Related Disorders/bl [Blood]"  
 "\*Cocaine-Related Disorders/ep [Epidemiology]"  
 Comorbidity  
 "\*Crack Cocaine/to [Toxicity]"  
 "\*Cytokines/bl [Blood]"  
 Female  
 Humans  
 "\*Inflammation/bl [Blood]"  
 "\*Inflammation/ep [Epidemiology]"  
 Male  
 Risk Assessment  
 Risk Factors

**Source:** MEDLINE

### 10. Oxidative stress and inflammatory markers are associated with depression and nicotine dependence.

**Citation:** Neuroscience Letters, June 2013, vol./is. 544/(136-40), 0304-3940;1872-7972 (2013 Jun 7)

**Author(s):** Vargas HO; Nunes SO; de Castro MR; Vargas MM; Barbosa DS; Bortolasci CC; Venugopal K; Dodd S; Berk M

**Institution:** Department of Clinical Medicine, Psychiatry Unit, Londrina State University, Brazil. hebervargas@sercomtel.com.br

**Language:** English

**Abstract:** To determine if oxidative stress and inflammation are linked with major depressive disorder, nicotine dependence and both disorders combined. This study comprised 150 smokers and 191 never smokers. The instruments were: a socio-demographic questionnaire, diagnoses of mood disorder and nicotine dependence according to DSM-IV, (SCID-IV), and the Alcohol, Smoking and Substance Involvement Screening Test. Laboratory assessments included: nitric oxide metabolites (NOx), lipid hydroperoxides, malondialdehyde (MDA), total reactive antioxidant potential (TRAP), advanced oxidation protein products (AOPP), fibrinogen concentrations, homocysteine, erythrocytes sedimentation rate (ESR) and high-sensitivity C-reactive protein (hs-CRP) were assayed from blood specimens. Statistically significant differences were found among depressed smokers who had more severe depressive symptoms, a higher risk of alcohol consumption, more suicide attempts, and more disability for work than non-depressed never smokers. Depressed smokers had significantly higher levels of NOx, fibrinogen, hs-CRP, AOPP, ESR and lower levels of TRAP compared to non-depressed never smokers. Depressed smokers had significant levels of oxidative stress and inflammatory biomarkers after adjusting for gender, age, years of education, disability for work, and laboratory measures. The levels of NOx, lipid hydroperoxides, AOPP, and fibrinogen were substantially higher, whereas levels of TRAP were lower in depressed smokers compared to non-depressed never smokers. (1) Depressed smokers exhibited altered concentrations of NOx, lipid hydroperoxides, AOPP, TRAP, and fibrinogen. (2) Depressed smokers were more unable to work, showed more severe depressive symptoms and attempted suicide more frequently. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Biological Markers); 0 (Cytokines); 0 (Reactive Oxygen Species)

**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** [Adolescent](#)  
[Adult](#)  
[Age Distribution](#)  
["Biological Markers/bl \[Blood\]"](#)  
["Brazil/ep \[Epidemiology\]"](#)  
[Comorbidity](#)  
["Cytokines/bl \[Blood\]"](#)  
["\\*Depressive Disorder Major/bl \[Blood\]"](#)  
["\\*Depressive Disorder Major/mo \[Mortality\]"](#)  
[Female](#)  
[Humans](#)  
["\\*Inflammation/bl \[Blood\]"](#)  
["\\*Inflammation/mo \[Mortality\]"](#)  
[Male](#)  
[Middle Aged](#)  
[Oxidative Stress](#)  
["\\*Reactive Oxygen Species/bl \[Blood\]"](#)  
[Risk Factors](#)  
[Sex Distribution](#)  
["Sick Leave/sn \[Statistics and Numerical Data\]"](#)  
["Suicide Attempted/sn \[Statistics and Numerical Data\]"](#)  
[Survival Analysis](#)  
[Survival Rate](#)

"\*Tobacco Use Disorder/bl [Blood]"  
 "\*Tobacco Use Disorder/mo [Mortality]"  
 Young Adult

**Source:** MEDLINE

### 11. Reduced cocaine-seeking behavior in heterozygous BDNF knockout rats.

**Citation:** Neuroscience Letters, June 2013, vol./is. 544/(94-9), 0304-3940;1872-7972 (2013 Jun 7)

**Author(s):** St Laurent R; Helm SR; Glenn MJ

**Institution:** Department of Psychology, Colby College, Waterville, ME 04901, USA.

**Language:** English

**Abstract:** Cocaine generates drug-seeking behavior by creating long-lasting changes in the reward pathway. The role of the growth factor, brain-derived neurotrophic factor (BDNF) in facilitating these changes was investigated in the present report with a genetic rat model. Using conditioned place preference, the current study investigated the hypothesis that a partial knockout of the BDNF gene in rats (BDNF(+/-)) would attenuate the rewarding effects of cocaine. Wildtype rats exposed to cocaine exhibited normal cocaine-seeking responses one day after conditioning and cocaine-seeking behavior was reinstated with drug priming following drug abstinence. In contrast, BDNF(+/-) rats did not show cocaine-seeking behavior one day after conditioning, nor did they respond to drug priming. A median split of rats based on BDNF levels in sera collected prior to behavioral procedures revealed that wildtype rats with high BDNF levels showed stronger conditioned place preference and reinstatement to cocaine. Together, the results support the hypothesis that a partial knockout of the BDNF gene attenuates the rewarding properties of cocaine. Additionally, individual differences in BDNF levels may predict future cocaine-seeking behavior. An underlying mechanism of these effects may be a reduction of the amount of synaptic changes made in the reward pathway. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Brain-Derived Neurotrophic Factor); E78ZFF4KQ0 (Reward); I5Y540LHVR (Cocaine)

**Publication Type:** Journal Article; Research Support, N.I.H., Extramural

**Subject Headings:** [Animals](#)  
["\\*Brain-Derived Neurotrophic Factor/bl \[Blood\]"](#)  
["\\*Brain-Derived Neurotrophic Factor/ge \[Genetics\]"](#)  
["\\*Cocaine/pd \[Pharmacology\]"](#)  
["\\*Cocaine-Related Disorders/bl \[Blood\]"](#)  
["\\*Drug-Seeking Behavior/de \[Drug Effects\]"](#)  
["\\*Drug-Seeking Behavior/ph \[Physiology\]"](#)  
[Female](#)  
[Rats](#)  
[Rats Sprague-Dawley](#)  
["\\*Repetition Priming/de \[Drug Effects\]"](#)  
["\\*Repetition Priming/ph \[Physiology\]"](#)  
[\\*Reward](#)

**Source:** MEDLINE

### 12. A tobacco extract containing alkaloids induces distinct effects compared to pure nicotine on dopamine release in the rat.

**Citation:** Neuroscience Letters, June 2013, vol./is. 544/(85-8), 0304-3940;1872-7972 (2013 Jun 7)

**Author(s):** Khalki H; Navailles S; Piron CL; De Deurwaerdere P

**Institution:** Laboratory of Pharmacology, Neurobiology and Behavior (URAC-37), University Cadi Ayyad, Faculty of Sciences Semlalia, Marrakesh, Morocco.

**Language:** English

**Abstract:** It has been suggested that minor alkaloids in plants play a role in the biological and neuronal actions of nicotine. We hypothesized that these molecules modulate the effect of nicotine on the activity of central dopamine (DA) neurons, one of the main cellular targets in addiction to drugs. In this study the effect of a single intraperitoneal injection of either nicotine or an alkaloid extract of the tobacco plant (0.5 mg/kg) on the efflux of DA were investigated. DA was measured in vivo by intracerebral microdialysis in the nucleus accumbens and the striatum of freely-moving rats. Results show that nicotine enhanced accumbal and striatal DA extracellular levels (+47 and 20% above baseline, respectively). The extract also evoked a significant increase in DA extracellular levels in both regions (+33 and +38% above baseline). However, this effect was significantly higher compared to nicotine in the striatum only. In conclusion, the tobacco extract enhanced the neurochemical effect of nicotine alone in the striatum, a response that could underlie the higher propensity of developing addictive-like behavior using nicotine with tobacco alkaloids. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Alkaloids); 0 (Plant Extracts); 54-11-5 (Nicotine); VTD58H1Z2X (Dopamine)

**Publication Type:** Comparative Study; Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** ["\\*Alkaloids/pd \[Pharmacology\]"](#)  
[Animals](#)  
["Corpus Striatum/de \[Drug Effects\]"](#)  
["Corpus Striatum/me \[Metabolism\]"](#)  
["\\*Dopamine/bi \[Biosynthesis\]"](#)  
["Dopaminergic Neurons/de \[Drug Effects\]"](#)  
["\\*Dopaminergic Neurons/me \[Metabolism\]"](#)  
[Male](#)  
["\\*Nicotine/pd \[Pharmacology\]"](#)  
["Nucleus Accumbens/de \[Drug Effects\]"](#)  
["Nucleus Accumbens/me \[Metabolism\]"](#)  
["\\*Plant Extracts/pd \[Pharmacology\]"](#)  
[Rats](#)  
[Rats Sprague-Dawley](#)  
["\\*Tobacco/ch \[Chemistry\]"](#)

**Source:** MEDLINE

### 13. Protein kinase G increases AMPA receptor GluR1 phosphorylation at serine 845 after repeated cocaine administration in the rat nucleus accumbens.

**Citation:** Neuroscience Letters, June 2013, vol./is. 544/(147-51), 0304-3940;1872-7972 (2013 Jun 7)

**Author(s):** Seo SY; Oh JH; Choe ES

**Institution:** Department of Biological Sciences, Pusan National University, 63-2 Pusandaehak-ro, Kumjeong-gu, Pusan 609-735, Republic of Korea.

**Language:** English

**Abstract:** The regulation of alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor GluR1 subunit phosphorylation at serine 845 (GluR1-Ser845) by protein kinase G (PKG) activation was investigated in the nucleus accumbens (NAc) after repeated cocaine administration. Intra-NAc injection of the cyclic guanosine monophosphate (cGMP) analog, Rp-8-Br-PET-cGMPS (5 nmol) and the PKG inhibitor, KT5823 (2 nmol), prior to the final drug injection significantly decreased GluR1-Ser845 phosphorylation elevated by repeated systemic injections of cocaine (20mg/kg) once a day for seven consecutive days. The inhibition of PKG also attenuated Ca(2+)-calmodulin-dependent protein kinases II (CaMKII) phosphorylation, however inhibition of CaMKII with KN62 (20 nmol) did not alter the phosphorylation state of GluR1-Ser845. Similarly, inhibition of cGMP or PKG attenuated the repeated cocaine-induced increase in locomotor activity. These findings suggest that the AMPA receptor provides a PKG-sensitive phosphorylation site on GluR1-Ser845 in the NAc after repeated cocaine, thus

contributing to behavioral alterations. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Receptors, AMPA); 0 (glutamate receptor ionotropic, AMPA 1); 452VLY9402 (Serine); EC 2-7-11-12 (Cyclic GMP-Dependent Protein Kinases); EC 2-7-11-17 (Calcium-Calmodulin-Dependent Protein Kinase Type 2); I5Y540LHVR (Cocaine)

**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** [Animals](#)  
["\\*Behavior Animal/de \[Drug Effects\]"](#)  
["\\*Calcium-Calmodulin-Dependent Protein Kinase Type 2/me \[Metabolism\]"](#)  
["Cocaine/to \[Toxicity\]"](#)  
["\\*Cocaine-Related Disorders/me \[Metabolism\]"](#)  
["Cyclic GMP-Dependent Protein Kinases/ai \[Antagonists and Inhibitors\]"](#)  
["\\*Cyclic GMP-Dependent Protein Kinases/me \[Metabolism\]"](#)  
[Male](#)  
["Nucleus Accumbens/de \[Drug Effects\]"](#)  
["\\*Nucleus Accumbens/me \[Metabolism\]"](#)  
["Phosphorylation/de \[Drug Effects\]"](#)  
[Rats](#)  
[Rats Sprague-Dawley](#)  
["\\*Receptors AMPA/me \[Metabolism\]"](#)  
["\\*Serine/me \[Metabolism\]"](#)

**Source:** MEDLINE

#### 14. Employers should help prevent misuse of alcohol by employees.

**Citation:** BMJ, 2013, vol./is. 347/(f6590), 0959-535X;1756-1833 (2013)

**Author(s):** Shenker D

**Language:** English

**Country of Publication:** England

**Publication Type:** Journal Article

**Subject Headings:** ["Alcohol Drinking/ae \[Adverse Effects\]"](#)  
["\\*Alcohol Drinking/pc \[Prevention and Control\]"](#)  
["Alcohol-Related Disorders/et \[Etiology\]"](#)  
["\\*Alcohol-Related Disorders/pc \[Prevention and Control\]"](#)  
[Great Britain](#)  
[Humans](#)  
[\\*Occupational Health](#)  
["\\*Preventive Health Services/mt \[Methods\]"](#)  
[Questionnaires](#)  
[Workplace](#)

**Source:** MEDLINE

**Full Text:** Available from *Highwire Press* in *BMJ*  
 Available from *BMJ* in *Newcomb Library & Information Service*

#### 15. mGluR1/5 receptor densities in the brains of alcoholic subjects: a whole-hemisphere autoradiography study.

**Citation:** Psychiatry Research, June 2013, vol./is. 212/3(245-50), 0165-1781;1872-7123 (2013 Jun 30)

**Author(s):** Kupila J; Karkkainen O; Laukkanen V; Tupala E; Tiihonen J; Storvik M

**Institution:** Department of Forensic Psychiatry, University of Eastern Finland, Niuvanniemi Hospital, Kuopio, Finland.

**Language:** English

**Abstract:** Increased glutamatergic neurotransmission and hyper-excitability during alcoholic withdrawal and abstinence are associated with increased risk for relapse, in addition to compensatory changes in the glutamatergic system during chronic alcohol intake. Type 5 metabotropic glutamate receptor (mGluR5) is abundant in brain regions known to be involved in drug reinforcement, yet very little has been published on mGluR1/5 expression in alcoholics. We evaluated the densities of mGluR1/5 binding in the hippocampus and striatum of post-mortem human brains by using [(3)H]Quisqualic acid as a radioligand in whole hemispheric autoradiography of Cloninger type 1 (n=9) and 2 (n=8) alcoholics and healthy controls (n=10). We observed a 30-40% higher mGluR1/5 binding density in the CA2 area of hippocampus in type 1 alcoholics when compared with either type 2 alcoholics or healthy subjects. Although preliminary, and from a relatively small number of subjects from these diagnostic groups, these results suggest that the mGluR1/5 receptors may be increased in type 1 alcoholics in certain brain areas. Copyright 2012 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (Receptor, Metabotropic Glutamate 5); 0 (Receptors, Metabotropic Glutamate); 0 (metabotropic glutamate receptor type 1)

**Publication Type:** Journal Article

**Subject Headings:** [Adult](#)  
[Aged](#)  
["Alcoholism/ge \[Genetics\]"](#)  
["\\*Alcoholism/me \[Metabolism\]"](#)  
[Autoradiography](#)  
["\\*Brain/me \[Metabolism\]"](#)  
[Female](#)  
[Humans](#)  
[Male](#)  
[Middle Aged](#)  
["Receptor Metabotropic Glutamate 5/ge \[Genetics\]"](#)  
["\\*Receptor Metabotropic Glutamate 5/me \[Metabolism\]"](#)  
["Receptors Metabotropic Glutamate/ge \[Genetics\]"](#)  
["\\*Receptors Metabotropic Glutamate/me \[Metabolism\]"](#)

**Source:** MEDLINE

**Full Text:** Available from *Elsevier* in [Psychiatry Research](#)

#### 16. Impact of psychiatric disorders and chronic physical conditions on health-related quality of life: Singapore Mental Health Study.

**Citation:** Journal of Affective Disorders, May 2013, vol./is. 147/1-3(325-30), 0165-0327;1573-2517 (2013 May)

**Author(s):** Subramaniam M; Abdin E; Vaingankar JA; Nan L; Heng D; McCrone P; Chong SA

**Institution:** Institute of Mental Health, Buangkok Green Medical Park, 10 Buangkok View, Singapore 539747, Singapore. mythily@imh.com.sg

**Language:** English

**Abstract:** BACKGROUND: Few studies have established Quality Adjusted Life Years (QALY) losses associated with mental and chronic physical conditions in the community. The aim of the current study was to establish and compare the QALY losses associated with select mental and chronic physical conditions in Singapore. METHODS: The Singapore Mental Health Study was a cross-sectional epidemiological survey of a nationally representative sample. The diagnosis of psychiatric disorders was established using the Composite International Diagnostic Interview (CIDI 3.0) and health related quality of life (HRQoL) was measured using the Euro-QoL-5D (EQ-5D). RESULTS: Pain conditions, hypertension and MDD were associated with the highest loss of QALYs in Singapore at a population level. The marginal effect on HRQoL by - Major Depressive Disorder (MDD), Obsessive Compulsive Disorder (OCD) and Bipolar Disorder was higher than the effect of any physical condition. LIMITATIONS: The presence of chronic physical diseases was

established using a check-list rather than with more objective measures and UK tariffs were used as local tariffs are not available and this might introduce some cultural bias. CONCLUSIONS: QALY losses associated with psychiatric disorders were high, emphasizing the need for recognizing them as major public health concerns and the need for appropriate resource allocation. Copyright 2012 Elsevier B.V. All rights reserved.

**Country of Publication:** Netherlands

**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't

**Subject Headings:** Adolescent  
Adult  
Aged  
"\*Alcohol-Related Disorders/ep [Epidemiology]"  
"Alcohol-Related Disorders/px [Psychology]"  
"\*Chronic Disease/ep [Epidemiology]"  
"Chronic Disease/px [Psychology]"  
Cross-Sectional Studies  
Depressive Disorder Major  
Female  
Humans  
Male  
"\*Mental Disorders/ep [Epidemiology]"  
"Mental Disorders/px [Psychology]"  
Middle Aged  
Quality of Life  
\*Quality-Adjusted Life Years  
"Singapore/ep [Epidemiology]"  
Young Adult

**Source:** MEDLINE

**Full Text:** Available from Elsevier in *Journal of Affective Disorders*

#### 17. The influence of stimulants on truck driver crash responsibility in fatal crashes.

**Citation:** Forensic Science International, May 2013, vol./is. 228/1-3(15-20), 0379-0738;1872-6283 (2013 May 10)

**Author(s):** Gates J; Dubois S; Mullen N; Weaver B; Bedard M

**Institution:** Centre for Research on Safe Driving, Lakehead University, Thunder Bay, Ontario, Canada P7B-5E1.

**Language:** English

**Abstract:** INTRODUCTION: Given the monotony and extended driving periods inherent in transport truck driving, drivers might rely on stimulants to sustain attention and combat fatigue. Research indicates that stimulant use improves some cognitive functions but impairs driving ability and is linked to crashes. The research on crash responsibility among stimulant-positive truck drivers is inconclusive due to small sample sizes and a lack of control over confounding variables. The present study investigated the influence of stimulants on unsafe driving actions (UDAs) in fatal crashes contained in the Fatality Analysis Reporting System (FARS) database. METHODS: Logistic regression was used to calculate the odds ratio of an UDA (cases committed an UDA; controls did not) by stimulant status (present; absent) while accounting for the influence of confounding variables (age, previous driving record, and other drug use). RESULTS: For all truck drivers, we found that 372 truck drivers tested stimulant-positive representing 0.57% of the entire truck driver sample and 3.7% of truck drivers who were actually tested for drug use. Stimulant-positive truck drivers had a greater proportion of driving record infractions and narcotic drug use compared to stimulant-negative truck drivers. The adjusted odds of committing an UDA were 78% greater for truck drivers who were stimulant-positive (OR: 1.78, 95% CI: 1.41-2.26) compared to truck drivers stimulant-negative. CONCLUSION: The results suggest stimulants are associated with crash responsibility and warrant further study into their impact on truck drivers. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland  
**CAS Registry Number:** 0 (Central Nervous System Stimulants)  
**Publication Type:** Journal Article; Research Support, Non-U.S. Gov't  
**Subject Headings:** [\\*Accidents Traffic](#)  
[Adult](#)  
[\\*Automobile Driving](#)  
["\\*Central Nervous System Stimulants/bl \[Blood\]"](#)  
[Databases Factual](#)  
["Fatigue/pc \[Prevention and Control\]"](#)  
[Humans](#)  
[Logistic Models](#)  
[Middle Aged](#)  
[Motor Vehicles](#)  
[North America](#)  
[Prevalence](#)  
[Records as Topic](#)  
[Substance Abuse Detection](#)  
["Substance-Related Disorders/ep \[Epidemiology\]"](#)

**Source:** MEDLINE

**Full Text:** Available from *Elsevier* in [Forensic Science International](#)  
Available from *ProQuest* in [Forensic Science International](#); Note: ; Collection notes: If asked to log in click "Athens Login" and then select "NHSEngland" in the drop down list of institutions.

#### 18. Analysis of 4-MEC in biological and non-biological material--three case reports.

**Citation:** Forensic Science International, May 2013, vol./is. 228/1-3(e11-5), 0379-0738;1872-6283 (2013 May 10)

**Author(s):** Gil D; Adamowicz P; Skulska A; Tokarczyk B; Stanaszek R

**Institution:** Institute of Forensic Research, Westerplatte 9, 31-033 Krakow, Poland.

**Language:** English

**Abstract:** 4-Methylethcathinone (4-MEC) is a designer drug that is structurally similar to mephedrone. This substance was identified in many drug seizures analyzed in the Institute of Forensic Research (IFR). This paper describes three of the first cases in which both powders and biological material were secured at the same time and delivered to the IFR for toxicological analysis. The first case concerned a man who died in a car crash. The second case describes a death associated with multiple-drug intake, including 4-MEC. In this case, however, the death was the result of an overdose of para-methoxyamphetamine (PMA). In the third case, the man was arrested for possession of illicit drugs. Analysis of powders was carried out using gas chromatography-mass spectrometry (GC-MS) and high pressure liquid chromatography with diode array detection (HPLC-DAD). The purity of 4-MEC found in powder samples was 51% and 78%. Analyses of biological material were carried out using liquid chromatography-tandem mass spectrometry (LC-MS/MS). 4-MEC was found in blood samples at concentrations of 46, 56 and 152 ng/mL. Copyright 2013 Elsevier Ireland Ltd. All rights reserved.

**Country of Publication:** Ireland

**CAS Registry Number:** 0 (4-methylethcathinone); 0 (Alkaloids); 0 (Amphetamines); 0 (Central Nervous System Stimulants); 0 (Designer Drugs); 0 (Powders); 0 (Propiophenones); 540EI4406J (cathinone)

**Publication Type:** Case Reports; Journal Article

**Subject Headings:** [Adult](#)  
["\\*Alkaloids/an \[Analysis\]"](#)  
["Alkaloids/ch \[Chemistry\]"](#)  
["\\*Amphetamines/an \[Analysis\]"](#)

"Amphetamines/ch [Chemistry]"  
 "\*Central Nervous System Stimulants/an [Analysis]"  
 "Central Nervous System Stimulants/ch [Chemistry]"  
 Chromatography High Pressure Liquid  
 "\*Designer Drugs/an [Analysis]"  
 Forensic Toxicology  
 Gas Chromatography-Mass Spectrometry  
 "Gastrointestinal Contents/ch [Chemistry]"  
 Humans  
 Male  
 Molecular Structure  
 "Powders/ch [Chemistry]"  
 "\*Propiophenones/an [Analysis]"  
 "Propiophenones/ch [Chemistry]"  
 Substance Abuse Detection  
 "Substance-Related Disorders/bl [Blood]"  
 "Substance-Related Disorders/ur [Urine]"

**Source:** MEDLINE

**Full Text:** Available from *Elsevier* in *Forensic Science International*  
 Available from *ProQuest* in *Forensic Science International*; Note: ; Collection notes: If asked to log in click "Athens Login" and then select "NHSEngland" in the drop down list of institutions.

#### 19. [Mephedrone: a new synthetic drug]. [French] La mephedrone: une nouvelle drogue de synthese.

**Original Title:** La mephedrone: une nouvelle drogue de synthese.

**Citation:** Presse Medicale, October 2013, vol./is. 42/10(1310-6), 0755-4982;0755-4982 (2013 Oct)

**Author(s):** Petit A; Karila L; Sananes M; Lejoyeux M

**Institution:** AP-HP, hopital Bichat, service de psychiatrie, addictologie et tabacologie, 75018 Paris, France; Universite Paris-VII, faculte de medecine, 75018 Paris, France. Electronic address: a.petit@bch.aphp.fr.

**Language:** French

**Abstract:** Mephedrone is a synthetic psychostimulant derived from cathinone belonging to the family of phenylethylamines. Sold on the Internet, it has recently emerged in France in recreational settings, and is mostly consumed by young people from the gay community and festive environment. Identified in 2008 by the European Monitoring Centre for Drugs and Drug Addiction as a new drug on the market, the use of mephedrone has attracted media attention following the suspicious deaths of two young adults in Sweden and in England. Its legal aspect, ease of getting it on the Internet and cheap price coupled and an alternative-seeking to other psychostimulants make mephedrone a prime target for these populations and a source of abuse, with psychiatric and somatic complications. There is no curative pharmacological treatment approved by health authorities. Copyright 2013 Elsevier Masson SAS. All rights reserved.

**Country of Publication:** France

**CAS Registry Number:** 0 (Central Nervous System Stimulants); 0 (Street Drugs); 44RAL3456C (Methamphetamine); 8BA8T27317 (mephedrone)

**Publication Type:** English Abstract; Journal Article; Review

**Subject Headings:** Adult  
 "Central Nervous System Stimulants/cs [Chemical Synthesis]"  
 "Central Nervous System Stimulants/pd [Pharmacology]"  
 "Central Nervous System Stimulants/to [Toxicity]"  
 \*Central Nervous System Stimulants  
 Europe  
 "France/ep [Epidemiology]"  
 Humans  
 "\*Methamphetamine/aa [Analogues and Derivatives]"

"Methamphetamine/cs [Chemical Synthesis]"  
"Methamphetamine/pd [Pharmacology]"  
"Methamphetamine/to [Toxicity]"  
"Street Drugs/lj [Legislation and Jurisprudence]"  
"Street Drugs/pd [Pharmacology]"  
"Street Drugs/to [Toxicity]"  
"Substance Withdrawal Syndrome/co [Complications]"  
"Substance Withdrawal Syndrome/ep [Epidemiology]"  
"Substance Withdrawal Syndrome/th [Therapy]"  
"Substance-Related Disorders/co [Complications]"  
"Substance-Related Disorders/ep [Epidemiology]"  
"Substance-Related Disorders/mo [Mortality]"  
"Substance-Related Disorders/th [Therapy]"  
Young Adult

**Source:**

MEDLINE