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Search History

1. PsycInfo; exp ADDICTION/ OR DRUG ABUSE [+NT]/ OR DRUG USAGE; 39753 results.
2. PsycInfo; addict*.ti,ab; 37548 results.
3. PsycInfo; 1 OR 2; 67864 results.
1. Selective activation of the trace amine-associated receptor 1 decreases cocaine's reinforcing efficacy and prevents cocaine-induced changes in brain reward thresholds.

Citation: Progress in Neuro-Psychopharmacology & Biological Psychiatry, Dec 2015, vol. 63, p. 70-75, 0278-5846 (Dec 3, 2015)

Author(s): Pei, Yue; Mortas, Patrick; Hoener, Marius C.; Canales, Juan J.

Abstract: The newly discovered trace amine-associated receptor 1 (TAAR1) has emerged as a promising target for medication development in stimulant addiction due to its ability to regulate dopamine (DA) function and modulate stimulants' effects. Recent findings indicate that TAAR1 activation blocks some of the abuse-related physiological and behavioral effects of cocaine. However, findings from existing self-administration studies are inconclusive due to the very limited range of cocaine unit doses tested. Here, in order to shed light on the influence of TAAR1 on cocaine's reward and reinforcement, we studied the effects of partial and full activation of TAAR1 on (1) the dose–response curve for cocaine self-administration and (2) cocaine-induced changes in intracranial self-stimulation (ICSS). In the first experiment, we examined the effects of the selective full and partial TAAR1 agonists, RO5256390 and RO5203648, on self-administration of five unit-injection doses of cocaine (0.03, 0.1, 0.2, 0.45, and 1 mg/kg/infusion). Both agonists induced dose-dependent downward shifts in the cocaine dose–response curve, indicating that both partial and full TAAR1 activation decrease cocaine, reinforcing efficacy. In the second experiment, RO5256390 and the partial agonist, RO5263397, dose-dependently prevented cocaine-induced lowering of ICSS thresholds. Taken together, these data demonstrated that TAAR1 stimulation effectively suppresses the rewarding and reinforcing effects of cocaine in self-administration and ICSS models, supporting the candidacy of TAAR1 as a drug discovery target for cocaine addiction.

Subject Headings: Rewards
Rats
Side Effects (Drug)
Drug Self Administration
Dopamine
Addiction
Cocaine

Source: PsycInfo

2. Association of GABAA receptor α2 subunit gene (GABRA2) with alcohol dependence-related aggressive behavior.

Citation: Progress in Neuro-Psychopharmacology & Biological Psychiatry, Dec 2015, vol. 63, p. 119-125, 0278-5846 (Dec 3, 2015)

Author(s): Strac, Dubravka Svob; Erjavec, Gordana Nedic; Perkovic, Matea Nikolac; Sviglin, Korona Nenadic; Borovecki, Fran; Pivac, Nela

Abstract: Alcohol dependence is a common chronic disorder precipitated by the complex interaction between biological, genetic and environmental risk factors. Recent studies have demonstrated that polymorphisms of the gene encoding the GABAA receptor α2 subunit (GABRA2) are associated with alcohol dependence in different populations of European ancestry. As aggression often occurs in the context of alcohol dependence, the aim of this study was to examine the allelic and haplotypic association of GABRA2 gene with alcohol dependence and related aggressive behavior in subjects of Eastern European (Croatian) origin. Genotyping of the 3 single nucleotide polymorphisms (SNPs) across the GABRA2 gene (rs567926, rs279858 and rs9291283) was performed in patients with alcohol dependence (N = 654) and healthy control subjects (N = 574). Alcohol-dependent participants were additionally subdivided according to the presence/absence of aggressive behavior and type of alcohol dependence according to the Cloninger's classification. The association of rs279858 with alcohol dependence yielded nominal significance level. Haplotype analysis revealed a high degree of linkage disequilibrium (LD) for rs567926 and rs279858, but not for rs9291283 polymorphism in the GABRA2 gene. In patients with alcohol dependence, the A–C (rs567926 and rs279858) haplotype carriers were more
likely to demonstrate aggressive behavior. The same haplotype (present only in 1.6% of all subjects) was significantly more often present in patients with a combination of early onset alcohol abuse and aggression, corresponding to the Cloninger's type II alcoholism subgroup. These findings support the involvement of GABRA2 gene in alcohol dependence-related aggressive behavior. (PsycINFO Database Record (c) 2015 APA, all rights reserved) (journal abstract)

Subject Headings:  
Chronicity (Disorders)  
Polymorphism  
Gamma Aminobutyric Acid  
Haplotype  
Neural Receptors  
Genes  
Alcoholism  
Aggressive Behavior

Source:  
PsycInfo

3. Effects of prenatal immune activation on amphetamine-induced addictive behaviors: Contributions from animal models.

Citation:  

Author(s):  
Borcoi, Aline R.; Patti, Camilla L.; Zanin, Karina A.; Hollais, André W.; Santos-Baldaia, Renan; Cecon, Liliane M. B.; Berro, Lais F.; Wuo-Silva, Raphael; Grapiglia, Stephanie B.; Ribeiro, Luciana T. C.; Lopes-Silva, Leonardo B.; Frussa-Filho, Roberto

Abstract:  
Background: Prenatal environmental adversities may affect brain development and are associated with increased risk for schizophrenia, an illness with 50% comorbidity with addiction. Maternal immune activation by polyinosinic–citosilic acid (Poly(I:C)) exposure can promote behavioral alterations consistent with schizophrenia symptoms in rodents. Objectives: Considering the vulnerability to addiction in patients with schizophrenia, we evaluated the interactions between prenatal Poly(I:C) administration and addiction in two animal models (behavioral sensitization and conditioned place preference—CPP) in mice repeatedly treated with amphetamine (AMP). Additionally, stereotyped behavior and cross-sensitization with cocaine (COC) were also investigated. Methods: Swiss male mice offspring were submitted to prenatal administration of 5 mg/kg Poly(I:C) in the 9th day of pregnancy. At the age of 90 days, mice were treated with 2.5 mg/kg AMP for 9 days to evaluate behavioral sensitization or stereotyped behavior. Cross-sensitization with 10 mg/kg COC was evaluated 24 h after the last treatment day. For AMP-induced CPP evaluation, mice were treated during 8 consecutive days. Results: Prenatal Poly(I:C) administration potentiated both AMP-induced behavioral sensitization and CPP. Furthermore, Poly(I:C) increased cross-sensitization with COC. Conclusions: Prenatal administration of Poly(I:C) is able to potentiate vulnerability to addiction in two animal models, without however modulating stereotyped behavior. (PsycINFO Database Record (c) 2015 APA, all rights reserved) (journal abstract)

Subject Headings:  
Sensitization  
Drug Sensitivity  
Schizophrenia  
Animal Models  
Addiction  
Cocaine  
Amphetamine  
Mice

Source:  
PsycInfo

4. Recommendations for the prevention, detection, treatment and management of prescription opioid analgesic dependence: Outcomes from the opioid analgesic dependence education nexus (open) meeting.
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Citation: International Journal of Mental Health and Addiction, Dec 2015, (Dec 3, 2015), 1557-1874 (Dec 3, 2015)
Author(s): Kraus, Mark; Lintzeris, Nicholas; Maier, Christoph; Savage, Seddon
Abstract: The global consumption of opioids continues to rise, which has led to an increasing rate of diversion, misuse, addiction, and deaths related to prescription opioids. This has been particularly well documented in the USA; however, opioid analgesic dependence (OAD) is an increasing concern in Europe. More guidance is required for European healthcare professionals in the prevention, detection, treatment and management of OAD. The first Opioid Analgesic Dependence Education Nexus (OPEN) Mentor Meeting was held in Berlin in September 2014 to address this. An international Expert Panel, combining expertise in OAD from Australia, USA and Europe, invited 16 European experts in the pain and addiction fields to develop a best-practice approach to OAD that European practitioners can adopt. The outcomes from this meeting are presented here and included are a set of shared strategies that may be universally adopted by all healthcare professionals working with patients who use opioids. (PsycINFO Database Record (c) 2015 APA, all rights reserved)(journal abstract)

Subject Headings: No terms assigned
Source: PsycInfo

5. Extended nicotine self-administration increases sensitivity to nicotine, motivation to seek nicotine and the reinforcing properties of nicotine-paired cues.

Citation: Addiction Biology, Dec 2015, (Dec 2, 2015), 1355-6215 (Dec 2, 2015)
Author(s): Clemens, Kelly J.; Lay, Belinda P. P.; Holmes, Nathan M.
Abstract: An array of pharmacological and environmental factors influence the development and maintenance of tobacco addiction. The nature of these influences likely changes across the course of an extended smoking history, during which time drug seeking can become involuntary and uncontrolled. The present study used an animal model to examine the factors that drive nicotine seeking behavior after either brief (10 days) or extended (40 days) self-administration training. In Experiment 1, extended training increased rats’ sensitivity to nicotine, indicated by a leftward shift in the dose–response curve, and their motivation to work for nicotine, indicated by an increase in the break point achieved under a progressive ratio schedule. In Experiment 2, extended training imbued the nicotine-paired cue with the capacity to maintain responding to the same high level as nicotine itself. However, Experiment 3 showed that the mechanisms involved in responding for nicotine or a nicotine-paired cue are dissociable, as treatment with the partial nicotine receptor agonist, varenicline, suppressed responding for nicotine but potentiated responding for the nicotine-paired cue. Hence, across extended nicotine self-administration, pharmacological and environmental influences over nicotine seeking increase such that nicotine seeking is controlled by multiple sources, and therefore highly resistant to change. (PsycINFO Database Record (c) 2015 APA, all rights reserved)(journal abstract)

Subject Headings: No terms assigned
Source: PsycInfo
Full Text: Available from Wiley in Addiction Biology