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Review

Differential effects of MDMA and cocaine on inhibitory avoidance and object recognition tests in rodents

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ABSTRACT

Introduction: Drug addiction continues being a major public problem faced by modern societies with different social, health and legal consequences for the consumers. Consumption of psychostimulants, like cocaine or MDMA (known as ecstasy) are highly prevalent and cognitive and memory impairments have been related with the abuse of these drugs.

Aim: The aim of this work was to review the most important data of the literature in the last 10 years about the effects of cocaine and MDMA on inhibitory avoidance and object recognition tests in rodents.

Development: The object recognition and the inhibitory avoidance tests are popular procedures used to assess different types of memory. We compare the effects of cocaine and MDMA administration in these tests, taking in consideration different factors such as the period of life development of the animals (prenatal, adolescence and adult age), the presence of polydrug consumption or the role of environmental variables. Brain structures involved in the effects of cocaine and MDMA on memory are also described.

Conclusions: Cocaine and MDMA induced similar impairing effects on the object recognition test during critical periods of lifetime or after abstinence of prolonged consumption in adulthood. Deficits of inhibitory avoidance memory are observed only in adult rodents exposed to MDMA. Psychostimulant abuse is a potential factor to induce memory impairments and could facilitate the development of future neurodegenerative disorders.

1. Introduction

Currently, drug abuse continues to be a major public problem faced by modern societies with different social, health and legal consequences for the consumers (Everitt, 2014; Koob & Volkow, 2016). Drug addiction can be defined as a chronic and recurrent disorder characterized by a compulsive seeking and loss of control over drug intake, the presence of a negative emotional state when the substance is not available and a high rate of relapse even after periods of abstinence (Koob & Volkow, 2016).

Psychostimulant drugs as cocaine and amphetamines (and its derivatives, such as 3,4-methylenedioxymethylamphetamine, MDMA) are widely used in our society (United Nations Office on Drugs and Crime, UNODC, 2016). Cocaine is the Psychostimulant most commonly consumed in occidental societies; in Europe 2.4 millions of people (1.9% of young adults between 15 and 34 years) have used cocaine in the last year (European Monitoring Centre for Drugs and Drug Addiction, EMCDDA, 2016). Dates regarding cocaine consumption show that the number of cocaine users has increased, from about 14 million in 1998 to 18.8 million in 2014 (UNODC, 2016). Despite the fact that an overall shrinking of the cocaine market has been observed, the number of cocaine users in several emerging markets continues to rise. Cocaine blocks the re-uptake of dopamine (DA), the main neurotransmitter involved in reward, and other monoamines on the brain and exerts stimulant actions on the central nervous system (CNS). Regarding its physical effects it is remarkable that it induces tachycardia, tremors and hypertension on the users. After repeated consumption, addictive-like behaviours and negative neuropsychological effects, including cognitive impairments, have been reported (Potvin, Stavro, Rizkallah, & Pelletier, 2014; Spronk, van Wel, Ramaekers, & Verkes, 2013).

On the other hand, MDMA, also known as ecstasy, and other amphetamine-type stimulants constitute the second most commonly used group of illicit substances worldwide. The typical recreational use of MDMA is often characterized by a pattern of repeated frequent administrations during a short period of time, also known as a binge

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administration (Badon et al., 2002) in night parties, raves or discos, with short-term effects after consumption such as hyperactivity, alertness, reduced fatigue, euphoria and entactogenic properties (Davis & Rosenberg, 2014; Nichols, 1986). In fact, positive effects after consumption have also been demonstrated in experimental animals showing that MDMA induces different changes in social behaviour such as pro-social and anti-aggressive effects (Blanco-Gandía et al., 2015; Machalova, Slais, Vrskova, & Sulcova, 2012; Thompson, Hunt, & McGregor, 2009). Moreover, data from human and animal studies confirm that ecstasy is a drug of abuse with addictive potential (Davis & Rosenberg, 2014; Daza-Losada et al., 2007) and damaging effects on cognition after repeated consumption (Camarasa, Rodrigo, Pubill, & Escubedo, 2012; García-Pardo, Roger-Sánchez, Rodríguez-Arias, Miñarro, & Aguilar, 2017; Parrott, 2015). The effects of MDMA on the CNS are diverse because this substance interacts with different neurotransmission systems (Breivik et al., 2014; Liechti, 2015; Yubero-Lahoz et al., 2015). MDMA blocks serotonin (5-HT), noradrenaline (NA) and DA, transporters and stimulates the release of these monoamines by reverse transport (Breivik et al., 2014; Colado, O'shea, & Green, 2004; Lizarraga et al., 2015), in several regions of the brain mainly in the nucleus accumbens (NAcc) (KankaanpÄÄ, Meririnne, Lillsunde, & SeppÄlÄ, 1998; Marona-Lewicka, Rhee, Sprague, & Nichols, 1996; White, Obradovic, Imel, & Wheaton, 1996; Yamamoto & Spanos, 1988).

The type of drug administration (acute, chronic, extended access) and the period of life (prenatal/neonatal period, adolescence or adulthood) when an individual is exposed to a drug of abuse can be determinant factors on the effects of such exposure. Scientific evidence shows that prenatal life is a period of enhanced sensitivity, since drugs of abuse can alter the developing brain inducing long-term structural and neuro-behavioural consequences. These impairing effects on brain development have been demonstrated with different types of drugs: depressant drugs, such as alcohol (Alfonso-Loeches & Guerri, 2011; Marquardt & Brigman, 2016) or stimulant drugs, such as cocaine or MDMA (Singer et al., 2015).

In particular, early exposure to psychostimulant drugs has complex and long-lasting implications for brain structure and function. For example, prenatal cocaine exposure causes profound changes in behaviour as well as synaptic function and brain structure with compromised glutamatergic transmission (Stucky, Bakshi, Friedman, & Wang, 2016). Cocaine prenatal exposure affects attention, learning and memory processes and induces morphological alterations in the brain (Kabir, Katzman, & Kosofsky, 2013; Zhao et al., 2015). Long-term behavioural and cognitive alterations induced by prenatal cocaine exposure are caused by altered cell proliferation, migration, differentiation and dendritic growth processes (Scott-Goodwin, Puerto, & Moreno, 2016). Similarly, a brief period of postnatal cocaine exposure during infancy on postnatal days (PND) 11–20 in rats can impair spatial cognition in adulthood (Melnick, Kubie, Laungani, & Dow-Edwards, 2001) and offspring of cocaine-using fathers showed alterations in brain circuitry underlying mood regulation (Killinger, Robinson, & Stanwood, 2012). In the same way, prenatal exposure to MDMA results in a behavioural phenotype in adult rats characterized by anxiety, a heightened response to novelty and "hyper-attentiveness" to environmental cues during spatial learning (Thompson et al., 2009). Prenatal MDMA exposure also induces physiological and neurobiological changes (Dzietko et al., 2010; Heuland, Germaux, Galineau, Chalon, & Belzung, 2010; Kaizaki, Tanaka, Yoshida, & Numazawa, 2014; Ádori et al., 2010) as well as structural and functional changes in noradrenergic structures involved in attentional processing (Thompson et al., 2012). Decreased exploratory activity, impaired memory and alterations in the dentate gyrus of the hippocampus were also observed in offspring from mothers treated with the combination of alcohol and MDMA during pregnancy (Canales & Ferrer-Donato, 2014).

Adolescence is a very important period in the life of individuals in which the brain is also highly vulnerable (Schneider, 2013). During puberty, neuronal maturation of the brain, which began during perinatal development, is completed so that the behavioural potential of the adult organism can be fully achieved (Schneider, 2013; Spear, 2013). The transition from childhood to adulthood involves extensive developmental changes and re-organisation of the brain (McCormick, 2010). During this period maturational processes in the prefrontal cortex (PFC) and limbic regions take place, which are characterized by both progressive and regressive changes, e.g. myelination, synaptic pruning (Spear, 2000) and grey matter reductions in the striatum and other subcortical structures (Rodríguez-Arias & Aguilar, 2012; Spear, 2013). Human adolescence is commonly considered to be from 12 to 18 years of age, although even up to 25 years is sometimes considered late adolescence (Baumrind, 1987) or as young adults. In animals, the period of adolescence is around 21–60 PND, which can be divided into early adolescence, between 21–34 PND, middle adolescence at 34–46 PND and young adults between 46–60 PND (Laviola, Macrì, Morley-Fletcher, & Adriani, 2003). While higher rates of drug consumption are observed during adolescence, drug consumption at this age can alter brain development and induce different cognitive and behavioural consequences (Santucci & Rabidou, 2011). However, developmental plasticity in this period of life can also have protective effects on the impairing effects on memory induced by cocaine (Kantak, Barlow, Tassin, Brisotti, & Jordan, 2014). For example, Santucci et al. (2004) showed that cocaine administration produces residual memory impairments in adolescent rats that are reversible with time.

Besides critical developmental periods, several studies have demonstrated that administration of cocaine and MDMA on adult life can also induce memory impairments in rats (Harper, Wisnewski, Hunt, & Schenk, 2005) in which oxidative stress could be involved (Muriach et al., 2010). Thus, it is important to determine how the exposure to cocaine and MDMA through lifetime can induce short- or long-term memory impairments. The effects of psychostimulant exposure on memory in animal models can have a translational value to evaluate the possible influence of drug consumption on the development of future cognitive decline or premature neurodegenerative disorders such as Alzheimer's and Parkinson's disease in drug dependent individuals.

Exposure to different environmental variables (for example, stress exposure or environmental enrichment) can also have an influence on the effects of psychostimulant drugs on cognitive performance. In this regard it has been demonstrated that social stress can boost the cognitive impairment induced by MDMA (Garcia-Pardo et al., 2017). On the other hand, it seems that environmental enrichment promotes the formation of long-lasting memories (Melani, Chelini, Cenni, & Berardi, 2017) and decreases the activating effects of different types of drugs (Laviola, Hannan, Macrì, Solinas, & Jaber, 2008). Recent studies have demonstrated that environmental enrichment ameliorates the spatial memory deficit induced by methamphetamine (Hajheidari, Miladi-Gorji, & Bigdeli, 2017) and the object recognition deficit induced by toluene, the main component of inhalants (Montes, del Carmen Solís-Guillén, García-Jácome, & Páez-Martínez, 2017).

This work was aimed to provide an update of studies on the effects of cocaine and MDMA on two popular memory tasks in rodents, the inhibitory avoidance and the object recognition tests, to compare and contrast the effects of these drugs in function of the memory task under study and the stage of development of the animal. Thus, we will focus our attention in different aspects like the age of the animals during drug exposure (prenatal, adolescence and adult age), the type of drug administration (acute, chronic, extended access) and the environmental variables. First, we will describe the representative animal paradigms, the inhibitory avoidance and the object recognition test, used to evaluate different types of memory. Next, we will describe the main literature in the last ten years, about the differential effects of cocaine or MDMA exposure throughout the lifetime of the organism on memory using these animal models. Later, we will describe the main effects of cocaine and MDMA on brain reward- and memory-related structures.

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