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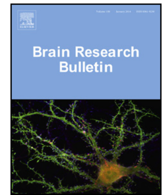

Research report

Administration of activated glial condition medium in the nucleus accumbens extended extinction and intensified reinstatement of methamphetamine-induced conditioned place preference

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ABSTRACT

Methamphetamine (METH) is a psychostimulant drug with significant abuse potential and neurotoxic effects. A high percentage of users relapse to use after detoxification and no effective medication has been developed for treatment of METH addiction. Developing evidences indicated the role of glial cells in drugs abused related phenomena. However, little is known about the role of these cells in the maintenance and reinstatement of METH-seeking behaviors. Therefore, the current study was conducted to clarify the role of glial cells in the maintenance and reinstatement of METH-induced conditioned place preference (CPP) in rats. Astrocyte condition medium (ACM) and neuroglia conditioned medium (NCM) are liquid mediums prepared from primary astrocyte and neuroglia cells. These mediums seem to contain many factors that release by glia cells. CPP was induced by systemic administration of METH (1 mg/kg for 5 days, s.c.). Following the establishment of CPP, the rats were given daily bilateral injections (0.5 μl/side) of either vehicle, ACM or NCM into the nucleus accumbens (NAc) and then were tested for the maintenance and reinstatement. Intra-NAc administration of ACM treated with METH, could extend the extinction period and also, intensified the magnitude of METH reinstatement. Furthermore, intra-accumbal administration of NCM treated with METH notably delayed the extinction period by four days and significantly increased the magnitude of CPP score in the reinstatement phase compared to the post-test phase. Collectively, these findings suggested that activation of glial cells may be involved in the maintenance and reinstatement of METH-seeking behaviors. It provides new evidence that glia cells might be considered as a potential target for the treatment of METH addiction.

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1. Introduction

Methamphetamine (METH) is a neurotoxic psychostimulant with highly addictive potential (Panenka et al., 2013). Psychosis, overdose and dependence are the major symptoms and disorders related to chronic use of METH. In addition, a high percentage of users relapse to drug taking after detoxification (Panenka et al., 2013). Unfortunately, no effective medication has been developed for treatment of METH addiction and symptoms associated with that (Gonzales et al., 2010; Ling et al., 2006).

Long-term exposure to psychostimulants can induce neuronal plasticity (Hyman and Malenka, 2001; Nestler, 2001). Alterations in neuronal structure, biochemistry and function in brain areas that are related to reward prossesing including medial prefrontal cortex (mPFC), nucleus accumbens (NAc), limbic structures and ventral tegmental area (VTA) are considered the base for the initiation, maintenance and also physiopathological mechanisms of drug addiction (Miguel-Hidalgo, 2009). The NAc by receiving dopaminergic projections from the VTA and glutamatergic projection from the mPFC play a crucial role in drug reward related phenomena as well as maintenance and reinstatement (Hsieh et al., 2014; Rocha and Kalivas, 2010). In addition, it has been shown that serotoninergic systems play a critical role in establishment of drug

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use-associated behaviors and transition from of METH use to addiction (Müller and Homberg, 2015)

Astrocytes exert a number of important functions during development of central nerve system (CNS). In the mature brain astrocytes perform a range of activities including maintenance of the blood-brain barrier, regulation of cerebral blood flow and CNS metabolism, involvement in synaptic transmission, neurotransmitter metabolism and turnover, glycogen storage and also regulation of extracellular pH and K^+ (Jensen et al., 2013; Sofroniew and Vinters, 2010).

Over the past decade accumulating evidence has demonstrated new critical roles for glial cells in neural function and their involvement in many diseases and disorders (Heneka et al., 2010). In addition to their well-known roles, glial cells by release of chemokines and neurotrophic factors can affect neuronal function (Aloisi, 2001; Dong and Benveniste, 2001). Furthermore, glial cells including astrocytes and microglia undertake a process of activation, proliferation and morphological changes as a consequence of many stimuli and disorders such as drug abuse (Kovacs, 2012; Narita et al., 2006).

Developing evidence has indicated the role of glial cells in drug reward related phenomena. In a study by Johnson et al. they showed that chronic morphine treatment increased expression of glial fibrillary acidic protein (GFAP), a specific marker of astrocytes, in the VTA (Johnson et al., 1993). Also it has been shown that glial activation is involved in opioid analgesia, reward and dependence (Hutchinson et al., 2007). Repeated amphetamine treatment increases the level of GFAP in caudate putamen (Armstrong et al., 2004). In addition, in a clinical study it has been shown that astrocyte and microglia changed in brain of METH users (Kitamura et al., 2010). Astrocyte condition medium (ACM) and neuroglia conditioned medium (NCM) are liquid mediums prepared from primary astrocyte and neuroglia cell culture respectively. Many stimuli can activate glia cells and induce the release of many factors including proinflammatory cytokines from them (John et al., 2003; Thomas et al., 2004a). It seems ACM and NCM, particularly their activated forms contain these factors. Recently, we showed that intra-NAc administration of NCM can prolong the extinction period and also increases the magnitude of morphine reinstatement (Arezoomandan et al., 2015). Another study by Narita et al. showed that chronic administration of METH significantly increased the GFAP expression in the NAc and cingulate cortex (CG). Furthermore, they showed that intra-NAc administration of ACM intensified the rewarding effects induced by METH and morphine (Narita et al., 2006). Many studies have mentioned that modulation of glia activation could attenute behavioral effects induced by METH (Fujita et al., 2012; Narita et al., 2006; Zhang et al., 2006). In our previous studies we reported that intra-NAc adminsteration of glia modulator, minocycline, attenuated the maintenance and blocked the drug-induced reinstatement of METH-CPP (Attarzadeh-Yazdi et al., 2014). Also, glial cell modulator, ibudilast, could attenuate drug- and cue-induced reinstatement of METH- CPP (Beardsley et al., 2010). Altogether, these data imply that glial cell activation may contribute to the synaptic plasticity and behavioral changes during the acquisition, extinction and reinstatement of METH. Therefore, in the present study the involvement of glial cells in the maintenance and reinstatement of METH was investigated using conditioned place preference (CPP) paradigm in rats.

2. Materials and methods

2.1. Animals

The experiments were carried out on male adult Wistar rats weighing 250–300 g (Pasteur Institute, Tehran, Iran). Animals were acclimated to the vivarium (a climate-controlled environment on a 12 h light/dark cycle), for at least one week prior to the onset of the experiments. The animals were randomly assigned to different experimental groups, habituated to their new environment and handled for one week before the experimental procedure was started. Each experimental group consisted of 5–7 animals. The tests were done between 9:00 a.m. and 2:00 p.m. Animal care procedures were conducted in accordance to the guide for the care and use of laboratory animals (National Institutes of Health Publication No. 80-23, revised 1996) and were conducted with the approval of an institutional animal care and use committee at the Research and Ethics Committee of Shahid Beheshti University of Medical Sciences. Additionally, all efforts were made to minimize animals' suffering and to use only the number of animals necessary to produce reliable scientific data.

2.2. Tissue processing

Primary cells were cultured using cerebral hemispheres of neonatal Wistar rat (1–2 days old). Briefly, the brains were removed and meninges and blood vessels were cleaned under a dissection microscope. After mechanical dissociation, tissues were treated with trypsin (0.025%, Gibco, USA) dissolved in phosphate-buffered saline (PBS) solution containing 0.02% L-cysteinemonohydrate (Sigma-Aldrich, USA), 0.5% glucose, and 0.02% bovine serum albumin (Gibco, USA) at 37 °C for 15 min. Then, the cells were placed in a flask containing Dulbecco's Modified Eagle Medium (DMEM) at a concentration of 15×10^6 cells and incubated at 37 °C in a moist 5% CO_2, 95% air atmosphere for 48–72 h. DMEM is a medium that contains glucose, amino acids, vitamins and additional supplementary components. The medium was changed every other day until cells reached an appropriate confluency and could be taken for harvesting different cell populations (Narita et al., 2006; Skaper et al., 2012).

To prepare cortical neuroglia conditioned medium (NCM), the cultures were incubated for 5 days. After reaching to appropriate confluency, the cells moved to the other flasks and after reaching appropriate confluency, the cells were washed with Dulbecco's Modified Eagle Medium (DMEM) and covered with an equal volume of serum-free medium for 3 days at 37 °C and 5% CO_2 in the presence of the 10 or 100 μM METH. After 3 consequence days' treatments with METH, supernatant was and centrifuged for 10 min at 1000 rpm. The final supernatant was used as NCM.

Purified cortical astrocyte was grown as follows: after seeding and reaching an appropriate confluency, the cells were washed and the medium was changed completely. To separate the microglia and oligodendrocytes, the flasks were shaken at 180 rpm for 12 h. Contents of shaken flasks were aseptically poured into a new flask at density of 1×10^5 cells/cm^3 in DMEM supplemented with 5% FBS, 5% horse serum (HS), 10 U/ml penicillin and 10 mg/ml streptomycin in a humidified atmosphere of 95% air and 5% CO_2 at 37 °C. Then, the cells were identified by immunofluorescence using mouse anti-GFAP antibody.

The astrocyte-conditioned medium (ACM) used in experimental procedure was prepared as follow; after reaching confluency, the cells were washed with DMEM and then covered with an equal volume of serum-free medium in the presence of the METH 10 or 100 μM treatment and were incubated for 3 days at 37 °C and 5% CO_2. The supernatant was collected and centrifuged 10 min at 1000 rpm. The final supernatant was used as ACM.

2.3. Immunocytochemistry

After seeding in eight-well chamber slides, astrocyte cells were fixed in 4% paraformaldehyde in PBS for 20 min at room temperature and then rinsed with PBS. Then, cells were permeabilized

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