Behavioural Brain Research 252 (2013) 439–443

Contents lists available at SciVerse ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Research report

Lesions of the nucleus accumbens disrupt reinforcement omission effects in rats

Danielle M. Judice-Daher*, José Lino O. Bueno

Departamento de Psicologia, Faculdade de Filosofia, Ciências e Letras de Ribeirão Preto, Universidade de São Paulo, Avenida Bandeirantes, 3900; 14049-901, Ribeirão Preto, São Paulo, Brazil

HIGHLIGHTS

- Nucleus accumbens is involved in the ROEs modulation.
- Lesions of the nucleus accumbens do not prevent ROEs, but interfere with them.
- Rats with nucleus accumbens lesions are less sensitive to the ROEs.
- ROEs cannot be explained only in terms of the frustration effect theory.
- ROEs can be driven by multiple processes.

ARTICLE INFO

Article history:
Received 28 March 2013
Received in revised form 17 June 2013
Accepted 20 June 2013
Available online 21 June 2013

Keywords:
Reinforcement omission effects
Nucleus accumbens
Rats
Operant behavior

ABSTRACT

The reinforcement omission effects (ROEs) have been attributed to both motivational and attentional consequences of the surprising reinforcement omission. Some studies have been showed amygdala is part of a circuit involved in the ROEs modulation. The view that amygdala lesions interfere with the ROEs is supported by evidence involving amygdala in responses correlated with motivational processes. These processes depend on the operation of separate amygdala areas and their connections with other brain systems. It has been suggested the interaction between the amygdala and the nucleus accumbens (NAC) is important to the modulation of motivational processes. Recent neuroimaging studies in human revealed reward delivery enhances activity of subcortical structures (NAC and amygdala), whereas reward omission reduces the activity in these same structures. The present study aimed to clarify whether the mechanisms related to ROEs depend on NAC. Prior to acquisition training, rats received bilateral excitotoxic lesions of NAC (NAC group) or sham lesions (Sham group). Following postoperative recovery, the rats were trained on a fixed-interval with limited hold signaled schedule of reinforcement. After acquisition of stable performance, the training was changed from 100% to 50% schedule of reinforcement. Both NAC and Sham groups presented the ROEs. However, after nonreinforcement, the response rates of the NAC group were lower than those registered in the Sham group. The performance of the NAC group decreased in the period following nonreinforcement when compared to the period preceding reinforcement omission. These findings suggest the NAC is part of the neural substrate involved in the ROEs modulation.

© 2013 Elsevier B.V. All rights reserved.

1. Introduction

The reinforcement omission effects (ROEs) have been attributed to motivational/emotional and attentional consequences of the surprising reinforcement omission. For instance, Amsel and Roussel [1] reported the introduction of partial reinforcement in the first goal of a double runway led to greatest response in the second runway immediately after omission than after delivery reinforcement; this effect was explained by increments in drive induced by primary frustration [1–3]. However, ROEs can be interpreted in terms of multiple processes involving behavioral facilitation after nonreinforcement, and behavioral transient inhibition after reinforcement induced by demotivation or reset of internal clock [4–7].

Some studies have showed amygdala is part of a circuit involved in the ROEs modulation [8–12]. The view that amygdala lesions interfere with ROEs is supported by evidence involving this area in responses correlated with motivational processes [13–15]. However, these processes depend on the operation of separate amygdala

* Corresponding author at: Faculdade de Filosofia, Ciências e Letras de Ribeirão Preto, Departamento de Psicologia, Universidade de São Paulo, Avenida Bandeirantes, 3900, 14049-901 Ribeirão Preto, São Paulo, Brazil.
Tel.: +55 16 36023697; fax: +55 16 36335668.
E-mail addresses: dadatrabalho@yahoo.com.br (D.M. Judice-Daher), jldobuen@usp.br (J.L.O. Bueno).

0166-4328/$ – see front matter © 2013 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.bbr.2013.06.028

areas and their connections with other brain systems [16]. It has been suggested the connections between different regions of the amygdala and cortical (prefrontal cortex) and subcortical (nucleus accumbens) structures are involved in processes related to reward [17–19], memory [20,21] and attention [22]. These regions are highly interconnected and together can be considered as an integrated network [23].

The nucleus accumbens (NAC) has been suggested to represent a limbic–motor interface [24]. It is a recipient of information from considerable limbic structures (including the amygdala, hippocampal formation, and regions of the prefrontal cortex) that also projects to structures known to be involved in behavioral expression [25]. The NAC is believed to contribute to the control of operant behavior by reinforcers. Recent evidence suggests that NAC is not crucial for determining the incentive value of immediately available reinforcers, but it has an important role in the control of behavior by delayed reinforcers [19,26].

Recent findings from human neuroimaging have been concerned the role of a highly interconnected network of brain areas including prefrontal cortex, amygdala, NAC and dopaminergic midbrain in reward processing [23]. Studies in human adults reveal that reward receipt enhances activity of subcortical structures (NAC and amygdala) and of cortical regions (prefrontal cortex), whereas reward omission tends to reduce activity of these same structures [27–30]. Spicer et al. [31] examined whether ventral frontostriatal regions differentially code expected and unexpected reward outcomes. Both the NAC and orbitofrontal cortex showed greater activation to rewarded relative to nonrewarded trials, but the NAC appeared to be most sensitive to violations in expected reward outcomes.

Although studies from human neuroimaging have been suggested the involvement of the NAC in processes related to violations in expected reward outcomes, its role in ROEs modulations remains unexplored. Studies conducted on animals suggesting that NAC has an important role in the control behavior by delayed reinforcers support the hypothesis that NAC can be involved in the ROEs modulation. If it is showed the functional impairment linked to NAC activation in lesioned animals interferes with ROEs, the processes related to these effects could be better understood. The present study therefore aimed to clarify whether or not the mechanisms underlying the ROEs depend on NAC. Prior to acquisition training, rats received bilateral excitotoxic lesions of NAC. Subsequently postoperative recovery, rats were trained on a fixed-interval 12 s with limited hold 6 s signaled schedule of reinforcement (acquisition training). On a fixed-interval schedule with a limited-hold contingency (FI LH), reinforcement is available for only a specified period of time after the FI terminates. Consequently, on a FI 12 s LH 6 s schedule, all responses occurring between 0 and 12 s after the start of the FI have no effect on reinforcement; however, the first response occurring between 12 and 18 s is followed by reinforcement [32]. After acquisition of stable performance, the training was changed from 100% to 50% reinforcement schedules (Testing: Partial Reinforcement). The role of the NAC on ROE is examined by comparison of the performance of the rats with NAC lesions and of the rats of the Sham groups, after reinforcement and after nonreinforcement.

2. Method

2.1. Subjects

The subjects were 28 experimentally naive male Wistar rats, 90 days old at the beginning of the experiments, weighing from 416 g to 433 g. Throughout the experiment, animals were housed singly in steel cages in the laboratory colony room, on a 12-h light schedule (lights on from 8:00 to 20:00). The rats were maintained on a water deprivation schedule at 85% of their ad libitum body weight by limiting access to water. Food was available at all times in their cages.

2.2. Surgical procedures

The animals were anesthetized by an intraperitoneal injection of a mixture containing 0.8 ml of ketamine hydrochloride (0.028 mg/ml) and 0.7 ml of xylazine (3.33 mg/ml). Each rat received 0.1 ml of anesthetic for each 100 g body mass. Excitotoxic lesions of the NAC ($n = 19$) were made by injecting 0.75 μl of 0.09 M quinolinic acid through a micropipette at coordinates 1.0 mm anterior to bregma, ± 1.6 mm from the midline, and 6.8 mm below the skull surface at bregma. The toxin had been dissolved in 0.1 M phosphate buffer (composition 0.07 M Na2HPO4, 0.028 M NaH2PO4 in double-distilled water, sterilized by filtration) and adjusted with NaOH to a final pH of 7.2–7.4. Toxin was injected over 3 min and the micropipette was left in place for 2 min following injections. Sham lesions ($n = 9$) were made in the same manner except that that no solution was infused [33]. At the end of the operation, animals were given a single subcutaneous injection of Banamine (2.15 mg/ml; 0.1 ml for each 100 g body mass) for amelioration of pain. They were given a week to recover, with free access to water and food.

2.3. Apparatus

The experiment was conducted in operant chambers (Lafayette model 80201) equipped with a speaker, which delivered a 1000 Hz, 30 dB tone, a 5 W house-light lamp, and a retractable 5 cm lever. Each chamber was in a soundproof wooden box provided with a transparent acrylic window; these chambers were located in soundproof experimental rooms. An electrical interface (MRA-Electronic Equipment, Ribeirao Preto, Brazil) connected the experimental chambers to a PC. This system used a program prepared with Microsoft QuickBasic 4.0 designed for this experiment, which controlled the reinforcement mechanisms registered and recorded lever presses.

2.4. Behavioral training procedures

2.4.1. Preoperative training

To avoid the potential effects of the lesions on learning, rats were trained preoperatively [34]. Preoperative training was carried out over two sessions. In the first session, each rat was placed into the operant chamber and trained to lever press for one 0.05 ml drop of water. The following session consisted of continuous reinforcement (CRF training, with a single water drop – 0.05 ml delivered with each lever press), for a total of 100 lever presses. Each session lasted a maximum of 1 hour.

2.4.2. Acquisition training

After recovery from surgery (approximately 7 days), rats were trained on a fixed-interval 12 s with limited hold 6 s schedule (FI 12 s LH 6 s) which was signalized by an auditory stimulus of 18 s. The first lever press occurring between 12 s and 18 s was always followed by the water delivery (0.05 ml). Each session consisted of 20 trials which were interpolated with variable inter-trial intervals (mean: 75 s). Each acquisition training session lasted for 30 min. All rats received a single training session per day for 24 days. At the end of each session, the rats were returned to their home cages and given access to water sufficient to maintain them within body weight schedule. Thus, the rats were water-deprived for approximately 23 h before the beginning of each session. The rats went

Download English Version:

https://daneshyari.com/en/article/4312635

Download Persian Version:

https://daneshyari.com/article/4312635

Daneshyari.com