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FUNCTION OF THE CENTROMEDIAL AMYGDALA IN REWARD DEVALUATION AND OPEN-FIELD ACTIVITY

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Abstract—The present research aimed at determining the role played by the amygdala in reward devaluation using transient inactivation induced by lidocaine microinfusions into the centromedial region. Two situations involving reward devaluation were tested in rats: consummatory successive negative contrast (cSNC) and anticipatory negative contrast (ANC). In cSNC, rats exposed to a downshift from 32% to 4% sucrose consume less 4% sucrose than rats always exposed to 4% sucrose. Extensive evidence suggests that reward devaluation in the cSNC situation is accompanied by negative emotion. In ANC, rats consume less 4% sucrose when each session is closely followed by access to 32% sucrose rather than by 4% sucrose. Evidence suggests that reward devaluation in the ANC situation does not involve negative emotions; rather, ANC appears to involve Pavlovian anticipation of the higher value solution. To test the effects of lidocaine microinfusions in a situation known to induce negative emotion, but unrelated to reward devaluation, animals were also exposed to a lighted open field. Centromedial amygdala inactivation reduced the cSNC effect and increased exploratory behavior in the open field, both effects consistent with a reduction in negative emotional state. However, no detectable effects of amygdala inactivation were observed in the ANC situation. These results suggest that, first, the function of the amygdala is not unique to reward devaluation and, second, it is concerned with tagging the devaluation experience with aversive valence. © 2015 Published by Elsevier Ltd. on behalf of IBRO.

Key words: incentive contrast, reward devaluation, amygdala, consummatory successive negative contrast, anticipatory negative contrast, open-field activity.

INTRODUCTION

Reversible lesions produced by infusions of the sodium-channel blocker lidocaine in several amygdala locations disrupt the effects of reward devaluation on instrumental behavior. In one experiment (Salinas et al., 1993), rats exposed in a runway to a 10-to-1 pellet downshift decreased running speed relative to rats always reinforced with 1 pellet—an effect known as instrumental successive negative contrast (iSNC; Flaherty, 1996). While both lidocaine and vehicle rats exhibited comparable response latencies on the first downshift session, lidocaine-treated animals recovered faster from reward devaluation in the following sessions. In another experiment using the same procedure, Salinas and McGaugh (1996) infused bicuculline, a $GABA_A$-receptor antagonist, immediately after the first downshift session into the amygdala and observed an enhancement of the iSNC effect. Furthermore, restricted neurotoxic lesions uncovered differential effects. Whereas lesions of the central nucleus of the amygdala (CeA) enhanced the iSNC effect, lesions of the basolateral nucleus of the amygdala (BLA) reduced the iSNC effect (Salinas et al., 1996). Interestingly, none of the two lesions affected the initial response to the downshift. Consistent results were reported with similar manipulations of the amygdala in a related form of incentive contrast—consummatory successive negative contrast (cSNC). In cSNC, consummatory behavior for a small reward is reduced by prior access to a large reward, relative to unshifted controls always receiving the small reward (Flaherty, 1996). Large centromedial amygdala lesions reduced or even eliminated the cSNC effect (Becker et al., 1984), whereas intraamygdala infusion of the $GABA_A$ agonist diazepam reduced the size of the cSNC effect (Liao and Chuang, 2003).

These results suggest that output from the amygdala is a critical component of the negative emotional state induced by reward devaluation in both the iSNC and cSNC situations. Moreover, $GABA_A$ receptors are involved in the modulation of the response to reward devaluation in both situations, as also shown by systemic treatments with benzodiazepines (for cSNC: Flaherty and Driscoll, 1980; Flaherty et al., 1990; Pellegrini et al., 2004; Freet et al., 2006; Ortega et al., 2014; for iSNC: Rosen and Tessell, 1970; Vogel and Principi, 1971). However, these effects of amygdala manipulations on iSNC and cSNC situations differ in one respect. Whereas disruption of amygdala output did not seem to affect the initial response to the reward devaluation in the iSNC situation (Salinas et al., 1993), the cSNC effect was disrupted on the first downshift session

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Abbreviations: ANC, anticipatory negative contrast; BLA, basolateral nucleus of the amygdala; CeA, central nucleus of the amygdala; cSNC, consummatory successive negative contrast; GABA, gamma-aminobutyric acid; iSNC, instrumental successive negative contrast; PBS, phosphate-buffered saline.

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(Becker et al., 1984). Such differential effects are not surprising since these contrast situations respond differentially to a number of behavioral and neurobiological manipulations (Flaherty, 1996). For example, lesions of the hippocampus and nucleus accumbens disrupt iSNC without apparently affecting cSNC (Flaherty et al., 1998; Leszczuk and Flaherty, 2000), whereas the lesions of the gustatory thalamus disrupt cSNC without affecting iSNC (Sastre and Reilly, 2006). But the experiments involving the amygdala were based on different manipulations (i.e., lidocaine infusions vs. electrolytic lesions). Thus, the present experiment sought to understand the role of the amygdala in the cSNC effect by producing a reversible inactivation of the centromedial region just before the first reward devaluation experience. Compared to pretraining irreversible lesions, the current approach has the advantage that the consummatory behavior develops under normal amygdala conditions before and after disruption of its activity.

To test for the boundary of the effects of lidocaine on reward devaluation, amygdala inactivation was also studied in the anticipatory negative contrast (ANC) situation and in the open field. The ANC task involves the same rewards used in the cSNC situation, but delivered in a different arrangement (Flaherty, 1996). In the ANC effect, consumption of 4% sucrose is suppressed in a group for which each trial is followed shortly thereafter by access to 32% sucrose (4–32 condition), relative to a group for which both trials provide access to 4% sucrose (4–4 condition). Such consummatory suppression does not depend on the last reward received a day earlier, but on the relative value of the forthcoming reward (Flaherty et al., 1995). The ANC effect develops over sessions and it is immune to pharmacological manipulations that eliminate, reduce, or exacerbate the cSNC effect, including treatments with benzodiazepine anxiolytics (Flaherty and Rowan, 1988) and corticosterone (Ruetti et al., 2009). There is also an unpublished report suggesting that electrolytic lesions of the central nucleus of the amygdala do not affect ANC (Coppotelli and Flaherty, cited in Flaherty, 1996, p. 121). Flaherty (1996) suggested that ANC is an anterograde phenomenon, that is, that consumption of the first reward is inhibited by anticipation of a forthcoming reward of a greater value. Thus, although the ANC effect involves reward devaluation, there is no evidence that the effect is accompanied by negative emotion. It was expected that amygdala inactivation would not affect ANC.

The effects of amygdala inactivation were also tested in the open-field situation. This task was chosen because it is known to induce behaviors indicative of negative emotion (Suarez and Gallup, 1981; Pare, 1994; Ramos, 2008). Rats exposed to a well-lighted open-field arena showed reduced activity in the central area, an indication of heightened unconditioned fear (Bouwknecht et al., 2007), and increased c-Fos expression in the BLA (Hale et al., 2006). These results, therefore, lead to the expectation that inactivation of the amygdala before open-field testing would enhance activity, especially in the central area of the arena.

EXPERIMENTAL PROCEDURES

Subjects

The subjects were 48 male Wistar rats, experimentally naïve and about 90 days of age at the start of the experiment. They were bred from animals purchased at Harlan Labs (Indianapolis, IN, USA), maintained in same-sex groups in polycarbonate cages after weaning, and moved to individual wire-bottom cages around postnatal day 40. The colony room was maintained at a relatively constant temperature (18–23 °C) and humidity (50%), and subject to a 12:12-h light cycle (lights on at 07:00 h). Rats were tested during the light portion of the daily cycle. Water was freely available throughout their lives. Food was freely available until they were approximately 90 days of age. In preparation for surgery (see below), all animals were food deprived to 90% of their free-food weight. After recovery from surgery and in preparation for behavioral testing, animals were further deprived to an 81–84% of their original free-food weight. This stepwise deprivation procedure was implemented to reduce the number of postsurgical days and thus minimize the risk of loose cannula implants. Supplemental food was given every day at least 15 min after behavioral sessions; the amount of food was determined by an empirically derived formula aimed at keeping animals within the preestablished range of food deprivation. While on deprivation, animals were weighed daily.

Apparatus

cSNC and ANC testing took place in eight conditioning boxes (MED Associates, St. Albans, VT, USA) made of aluminum and Plexiglas, and measuring 29.4 × 28.9 × 24.7 cm (L × H × W). The floor was made of steel rods, 0.5 cm in diameter and 1.2 cm apart, placed perpendicular to the feeder wall. A bedding tray filled with corncob bedding and placed underneath the rods collected fecal pellets and urine; the bedding was replaced as needed. An elliptical opening 1 × 2 cm (W × H), 3.5 cm from the floor and located on the feeder wall served to preset a sipper tube (diameter: 1 cm). When fully inserted, the sipper tube was flush against the wall. A house light (GE 1820) located in the center of the box's ceiling provided diffuse light. A computer in an adjacent room controlled the presentation and retraction of the sipper tube, and recorded contacts with the sipper tube. Each conditioning box was placed in a sound-attenuating chamber containing a speaker (white noise) and a fan (ventilation), and producing masking noise with an intensity of 80.1 dB (SPL scale C).

Open-field testing was carried out in three units (MED Associates, St. Albans, VT, USA), between 9:00 and 15:00 h. The dimensions of each chamber were 43 × 30 × 43 cm (L × H × W). Rats were tested in squads of three whenever possible. A light bulb (100 W) was suspended on top of each field, above the central area. The open field was cleaned immediately after each session.

UNCORRECTED PROOF

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