Brief Communication

Predator (cat hair)-induced enhancement of hippocampal long-term potentiation in rats: Involvement of acetylcholine

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Extensive literature has demonstrated that arousal and fear modify memory acquisition and consolidation. Predator hair and odors increase arousal in rats and, therefore, may influence information encoding and synaptic plasticity in the rodent nervous system. In behavioral experiments, we confirm that laboratory-bred Long Evans rats avoid cat hair. Electrophysiological work in vivo showed that long-term potentiation (LTP) in the dentate gyrus induced by perforant path stimulation was enhanced for 5–7 days when LTP induction occurred in the presence of cat hair relative to fake hair. The muscarinic receptor antagonist scopolamine (i.p.) reversed the cat hair–elicited LTP enhancement without affecting weaker LTP elicited in the presence of fake hair. Thus, exposure to a predator stimulus elicits a cholinergically-dependent state of heightened plasticity that may serve to facilitate information storage in hippocampal circuits.

Memory formation is a dynamic and selective process, and the attentional selection and subsequent encoding of stimuli in memory systems are influenced by emotional arousal and stress (e.g., McGaugh 2000, 2004; Vuilleumier 2005; LeBar and Cabeza 2006). In several species, including humans, acute increases in arousal and related adrenal hormones can enhance the consolidation of stimuli experienced in close temporal proximity to the hormonal activation, leading to stronger encoding of emotionally arousing material relative to more neutral stimuli (Cahill et al. 1994, 2000; McGaugh 2000; McGaugh and Roozenendaal 2002).

Extensive evidence suggests that the effect of arousal to facilitate memory consolidation is mediated by the basolateral amygdala. Local, intra-amygdala application of arousal-related pharmacological agents (e.g., adrenergic, noradrenergic, or glucocorticoid agonists) can enhance memory consolidation, while lesions of the basolateral amygdala block arousal-induced memory modulation (Packard et al. 1994; Cahill and McGaugh 1998; McGaugh 2004). Further, the amygdala also influences synaptic plasticity in several forebrain systems, with electrical activation of amygdala neurons enhancing long-term potentiation (LTP) at hippocampal, thalamocortical, and corticostriatal synapses (Akirav and Richter-Levin 1999; Frey et al. 2001; Dringenberg et al. 2004; Popescu et al. 2007). Thus, the basolateral amygdala serves as a critical interface linking arousal states, plasticity mechanisms, and memory consolidation (Cahill and McGaugh 1998; Paré 2003; McGaugh 2004; LeBar and Cabeza 2006).

Here, we tested whether an insetingly arousing and aversive stimulus, cat hair (Dielenberg and McGregor 2001; Apfelbach et al. 2005), is effective in modulating hippocampal LTP in rats under in vivo conditions. Further, given the role of acetylcholine (ACh) in mediating arousal- and amygdala-induced memory consolidation and plasticity (Frey et al. 2001; Dringenberg et al. 2004; McGaugh 2004) and in light of recent data showing increased cortical ACh release with predatory odor exposure (Smith et al. 2006), we also examined the role of ACh in mediating effects of cat hair observed in the present investigation.

All experimental procedures were conducted in accordance with the guidelines of the Canadian Council on Animal Care and approved by the Queen’s University Animal Care Committee. Adult, male Long-Evans rats (300–450 g, Charles River, Quebec) were housed as pairs in a colony room (12:12-h reversed light-dark cycle) with free access to food and water.

Cat hair (compressed into pellets, 4 × 4 cm, −0.6 g) was obtained by shaving a domestic cat on each day prior to the experiment and kept in airtight (Zip-Lock) plastic bags until used. Color-matched fake hair (plastic fibers) was obtained at a local arts supply store.

To verify that rats respond to cat hair, exploratory activity in the presence of cat hair or fake hair was measured in an open field apparatus (80 × 40 × 30 cm, one half of the inside painted black and one half white, bottom covered with fresh sawdust). A pellet of either cat hair or fake hair was placed in the center of the black half of the box. The rat was placed in the center of the white half and allowed to explore the entire box for 30 min.

Sessions were videotaped and analyzed for the following measures: (1) time spent in the hair-containing half of the apparatus—the rat was considered to have entered this compartment when all four paws crossed the midline of the apparatus; (2) number of snout contacts—the rat’s snout touching the hair pellet; and (3) number of snout pick-ups—picking up and moving the hair pellet with the snout.

Separate groups of rats were surgically prepared (sodium pentobarbital anesthesia, 65 mg/kg, i.p.; buprenorphine analgesia, 0.01 mg/kg, i.p.) with chronic electrode implants of a twisted bipolar stimulation electrode (125-μm Teflon-insulated wire) aimed at the perforant path (AP = −8.1, L +4.3, V = −3.0 from bregma) and a monopolar recording electrode aimed at the ipsilateral dentate gyrus (AP = −3.2, L +1.6, V = −3.5; both ventral placements adjusted to yield maximal field excitatory postsynaptic potential [fEPSP] amplitude). Reference and ground connections were placed in the bone over the cerebellum. Rats had seven recovery days before commencement of the experiments.
Following seven recovery days, electrophysiological procedures were carried out in freely moving rats contained in a round Plexiglas bowl (40-cm diameter). Dentate gyrus fEPSPs were recorded differentially against the cerebellar reference connection (ML 136 Bio Amp and PowerLab/8 s data acquisition system running Scope v. 3.6.4 software, digitizing rate of 1 kHz; ADInstruments). The PowerLab also provided constant current stimulation of the perforant path (ML 180 Stimulus Isolator) in the form of either single, monopolar 0.2-msec test pulses at 0.2 Hz, or theta burst stimulation (TBS) consisting of 10 pulses (100 Hz) per burst, which were repeated 10 times at 5 Hz.

Initially, rats were habituated to the room and recording apparatus for 7 consecutive days (~1 h/d). Subsequently, input-output curves (0.1–1.0 mA in 0.1-mA increments, 10 fEPSPs/ intensity) were established for three consecutive days. Following the completion of the input-output curve on day 3, LTP was induced using TBS (see above) at the intensity that produced 50%–60% of the maximal population spike amplitude. TBS was delivered during alert, behavioral immobility, defined as the absence of gross movements of head, trunk, and limbs (see Leung et al. 2003). One minute prior to TBS, a pellet of cat or fake hair was placed in the container holding the rat, where it remained until 1 min after TBS for total of 2-min hair exposure. Some rats received injections of scopolamine hydrochloride (1 mg/kg, i.p., dissolved in saline; obtained from Sigma/RBI; dose chosen based on previous work showing anti-muscarinic effects in vivo with systemic administration) (see Vanderwolf 1988; Dringenberg and Vanderwolf 1996) or an equivalent volume of saline on the day of LTP induction, administered 30 min prior to TBS delivery. Recordings of fEPSPs elicited by single perforant path pulses (intensity to elicit 50%–60% of maximal population spike amplitude) were repeated 5 min after and for 5–7 d following LTP induction. A further control group underwent similar electrophysiological procedures, with the exception that cat hair exposure occurred in the absence of TBS delivery in order to assess if cat hair alone exerted lasting effects on baseline (nonpotentiated) dentate gyrus fEPSPs. For all rats, standard perfusion and histological techniques were used to assess electrode placements, and all fEPSP data were scrutinized for significant changes and instability over the recording period, indicative of electrode movement or injury to the implantation site. Scope software (see above) computed population spike amplitude and average fEPSP slope. Based on these analyses, a total of 10 rats were excluded from the experiment, for the following reasons: instability of recordings, apparent either as development of excessive potentiation (500%–700% of baseline, three rats); clear deterioration of recordings (values of <50% of baseline, three rats); or postmortem histological examination indicating inaccurate electrode placements (four rats). Following the exclusion of these animals, the remaining data were subjected to ANOVA and, where statistically appropriate, simple effects tests, and are presented as mean ± SEM.

In the open field, all rats showed high levels of exploratory locomotor activity and rearing, regardless of the type of hair placed in the apparatus. Rats exposed to fake hair (n = 6) spent an average of 629 ± 91 sec (35 ± 5%) of the 30-min test session in the half of the apparatus containing the hair pellet (Fig. 1, top). In contrast, for cat hair, rats (n = 6) spent only 147 ± 36 sec (8 ± 2%) in the hair-containing half of the open field. Rats also readily approached and explored the fake hair, as evident in the high incidence of snout contacts (28 ± 5) and snout pick-ups (9.7 ± 3.2) (Fig. 1, middle and bottom, respectively). The number of times these behaviors were exhibited was significantly lower for cat hair, with 13 ± 3 snout contacts and 0.3 ± 0.2 snout pick-ups. Thus, laboratory-bred Long-Evans rats show an avoidance response to cat hair, even though it is noteworthy that, in both the behavioral and electrophysiological experiments, cat hair exposure did not result in behavioral freezing responses (i.e., sudden arrest of all movement).

Analyses of electrophysiological data showed that rats given saline injections 30 min prior to TBS and untreated rats did not differ on any fEPSP measure, and they were combined for the purpose of data presentation and statistical analyses (all Fs < 2.0 and all Ps > 0.2 for group and group by time effects comparing population spike amplitude and fEPSP slope in untreated and saline rats). Following 3 d of baseline recordings, rats that received TBS in the presence of fake hair (n = 16) showed a modest enhancement of the fEPSP slope (Fig. 2A) and population spike amplitude (Fig. 2B). When TBS was delivered in the presence of cat hair (n = 15), however, greater potentiation was observed for both measures. For the fEPSP slope, cat hair–induced LTP facilitation was particularly pronounced between 2 and 5 d after TBS (Fig. 2A), whereas population spike amplitude showed LTP enhancement up to 7 d following TBS (Fig. 2B). There was no significant difference for the TBS intensity used for the two groups of rats, with mean intensities of 0.26 ± 0.05 mA and 0.24 ± 0.05 mA for fake hair– and cat hair–exposed animals, respectively.

These experiments were repeated with animals administered scopolamine (1 mg/kg, i.p.) 30 min prior to TBS. In rats exposed to fake hair during TBS, LTP induced in the presence of scopol-
Figure 2. Dentate gyrus long-term potentiation (LTP) induced in the presence of either cat hair or fake hair and the effect of scopolamine treatment at the time of LTP induction. (A) Analysis of the slope of the field excitatory postsynaptic potential (fEPSP) prior to and after theta burst stimulation (TBS) in rats exposed to a predator odor following scopolamine administration. Exposure to cat hair produced an enhancement of LTP relative to rats exposed to fake hair. The enhancement of LTP by cat hair exposure during TBS, scopolamine (Scopol., 1 mg/kg, i.p., administered 30 min prior to TBS), exposure to cat hair (n = 16) failed to enhance LTP relative to fake hair–exposed animals (n = 6), as measured by the fEPSP slope (group effect: F_{1,14} = 0.5, P = 0.48; time effect: F_{2,28} = 2.7, P < 0.01; interaction: F_{2,28} = 0.8, P = 0.59). (D) Similarly, population spike amplitude analyzed in scopolamine-treated rats exposed to cat (n = 11) or fake hair (n = 7) also failed to show an enhancement for cat hair–exposed animals (group effect: F_{1,14} = 0.4, P = 0.53; time effect: F_{2,28} = 5.6, P < 0.01; interaction: F_{2,28} = 0.4, P = 0.93).

amine (n = 7) was not different from that in saline-treated and untreated rats (all Ps < 1.4, all Ps > 0.2 for group effects and group × time interactions comparing fEPSP slope and population spike amplitude in untreated/saline-treated and scopolamine-treated rats). However, for rats exposed to cat hair during TBS, scopolamine (n = 11) blocked the enhancement of LTP by cat hair exposure during TBS, so that cat hair and fake hair rats no longer differed at any time point following TBS (Fig. 2C,D). It is noteworthy, however, that for fake hair animals, fEPSP slope values on days 2, 3, and 5 were somewhat higher in scopolamine-treated rats relative to saline/no drug treatment (see Fig. 2 A,C), even though these differences were statistically nonsignificant (see above). We acknowledge that this trend minimizes differences in slope values between cat and fake hair groups following scopolamine treatment, even though it clearly does not account for the lack of differences between hair groups in population spike amplitude following drug treatment. There was no significant difference for the TBS intensity used for scopolamine-treated rats exposed to the two different hair types, with mean intensities of 0.24 ± 0.04 mA and 0.31 ± 0.05 mA for fake and cat hair–exposed animals, respectively.

An additional group of rats (n = 6) received cat hair exposure without concurrent TBS to assess whether hair alone affects non-potentiated dentate gyrus fEPSPs. For these animals, neither fEPSP slope nor population spike amplitude showed significant changes following hair exposure (Fig. 3). Thus, the LTP enhancement induced by cat hair does not appear to reflect a long-lasting facilitation of baseline dentate gyrus responses following hair exposure.

The present experiments show that cat hair, confirmed to produce an innate avoidance response, enhances hippocampal LTP in vivo when present at the time of induction. This effect appears to depend on activation of muscarinic receptors since scopolamine administration prevented LTP facilitation by cat hair, an interpretation consistent with recent data showing enhanced cortical ACh release in the presence of a predator odor (Smith et al. 2006). Thus, a biologically significant, aversive stimulus can facilitate hippocampal plasticity, an effect that requires the engagement of the cholinergic system.

Pharmacological experiments have shown that elevated levels of arousal/stress can enhance memory consolidation by a complex series of hormonal and neurochemical mechanisms.
effects on either fEPSP slope ($F_{top}$; time effect: $P = 0.83$) or population spike amplitude (bottom; time effect: $P = 0.7$).

Figure 3. Dentate gyrus field excitatory postsynaptic potentials (fEPSPs) before and after exposure to cat hair in the absence of theta burst stimulation ($n = 6$). Cat hair exposure alone did not exert significant effects on either fEPSP slope (top; time effect: $F_{top} = 0.5$, $P = 0.83$) or population spike amplitude (bottom; time effect: $F_{top} = 0.6$, $P = 0.7$).

creased release of peripheral stress hormones and arousal-related neurotransmitters (e.g., NA, ACh) act on the basolateral amygdala, which, in turn, influences plasticity in cortical and subcortical target regions that mediate different aspects of memory encoding, including hippocampus, caudate putamen, thalamus, and neocortex (Packard et al. 1994; Cahill and McGaugh 1998; Akirav and Richter-Levin 1999; Frey et al. 2001; Paré 2003; Dringenberg et al. 2004; McGaugh 2004; Popescu et al. 2007). Similarly, enhancement of LTP has been demonstrated for a variety of situations related to increases in exploratory activity and arousal. For example, transient early-phase LTP can be converted to stable (≥8-h) late-phase LTP by exploration of novel environments or objects (Li et al. 2003; Straube et al. 2003; Lemon and Manahan-Vaughan 2006). Inducing LTP during more active behavioral states (walking vs. immobility) also produces greater levels of potentiation (Leung et al. 2003). Our data demonstrating enhanced LTP with cat hair support the notion that LTP, like memory encoding, is a dynamic phenomenon that is influenced by the external and internal states of the animal. In our experiments, rats were well acquainted with the room and electrophysiological apparatus, and LTP was induced during immobility, suggesting that the effect we observed is not related to novelty of the test environment and procedure or to differences in gross motor activity of the animal.

Muscarinic receptor activation is known to enhance hippocampal LTP in vitro and in vivo (Huerta and Lisman 1993; Auerbach and Segal 1994; Leung et al. 2003; Ovsepian et al. 2004). In the present experiments, the muscarinic antagonist scopolamine prevented the enhancement of LTP by cat hair without affecting weaker LTP induced in the presence of fake hair. The latter observation indicates that scopolamine, at the dose used here, does not exert general suppressant effects on LTP at perforant path-dentate gyrus synapses. Cat hair is known to act on neurons in the amygdala (Vazdarjanova et al. 2001; Takahashi et al. 2005). In turn, excitation of the amygdala stimulates the cholinergic inputs to the hippocampus to elicit hippocampal EEG theta activity (Dringenberg and Vanderwolf 1996), an oscillatory state that favors that induction of LTP (Huerta and Lisman 1993; Leung et al. 2003). The interactions between the amygdala and cholinergic inputs to the hippocampus responsible for theta generation are likely mediated by polysynaptic pathways, given the lack of significant, direct projections from the amygdala to the medial septum (Jolkkonen et al. 2002).

Muscarinic receptor activation can facilitate plasticity induction by several, distinct mechanisms, including direct depolarization of postsynaptic membranes, convergence of muscarinic-coupled and NMDA receptor–coupled intracellular second messenger pathways, elevation of intracellular calcium, and further downstream effects on gene expression (Rasmusson 2000; Gu 2003). Importantly, concurrent activation of NMDA and muscarinic receptors synergistically interacts to induce de novo protein synthesis in hippocampal neurons (Feig and Lipton 1993), an essential requirement for the induction of long-lasting LTP (Frey et al. 2001) like that observed here. Thus, it is likely that cat hair–induced, cholinergic activation of the hippocampus is critical for electrophysiological effects noted in the present experiments, even though the systemic drug application used does not allow us to rule out the involvement of cholinergic mechanisms in extra-hippocampal areas. Detailed studies employing local drug application are required to characterize the precise targets of scopolamine and validate the anatomical model hypothesized to mediate cat hair influences on hippocampal plasticity. We also do not rule out the participation of additional neurochemical mechanisms, such as the effect of NA to promote synaptic enhancement in the dentate gyrus (Chauk and Harley 1998). In fact, reinforcement of hippocampal LTP by electrical stimulation of the amygdala is sensitive to both muscarinic and β-adrenergic receptor antagonists (Frey et al. 2001), consistent with the view that multiple, converging mechanisms act to facilitate hippocampal plasticity.

It is important to note that arousal and stress exert complex, biphasic effects on hippocampal functions and plasticity. While brief, moderate arousal can enhance LTP and memory consolidation, intense or chronic stressors generally result in memory impairments and hippocampal dysfunction, including suppression of LTP and adult neurogenesis, as well as neuronal atrophy (Diamond and Rose 1994; Xu et al. 1997; McEwen 2000; Artola et al. 2006). For example, exposing rats to a live cat, using a procedure to maximize sensory contact between the animals, produces impairments in hippocampal plasticity and memory functions (Diamond et al. 1999; 2006; Vouimba et al. 2006). Assuming that this procedure induces higher stress levels than those experienced by rats in the present experiments, this pattern of results is consistent with a hypothesized, biphasic relation between stress and memory/plasticity mechanisms.

To summarize, the present experiments demonstrate that predator-elicited arousal induces a state of heightened, ACh-dependent hippocampal plasticity, an effect that could serve to facilitate the encoding of biological significant information in hippocampal networks.

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