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Effects of Fluoxetine on Play Dominance in Juvenile Rats

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In a series of three studies, we investigated the influence of a selective serotonin reuptake inhibitor (fluoxetine) on the rough-and-tumble play of juvenile rats. In Experiment 1, both members of eight pairs of solitary-housed juvenile rats received either vehicle, 2.5, 5, or 10 mg/kg fluoxetine in a counterbalanced within-subject design 20 min before being allowed to play for 5 min periods on four successive test days. The 5 and 10 mg/kg pretreatments significantly reduced incidence of pins during play without affecting dorsal contacts. In Experiment 2, one member of each of 19 established play pairs received 5 mg/kg fluoxetine 20 min before play, while the other member received vehicle. Dominant rats showed no reduction in pins as a result of fluoxetine treatment, but subordinate rats who received fluoxetine exhibited significant reductions in pins. Subsequent dyadic analyses indicated that in pairs where the subordinate animal received fluoxetine, dominant animals maintained their pinning advantage over the 10 days of testing, but in pairs where the dominant animals received fluoxetine, this pinning asymmetry diminished. In Experiment 3, we replicated the above procedure with inexperienced play pairs, to control for the effects of prior social learning. Fluoxetine treatment (5 mg/kg) significantly reduced both pins and dorsal contacts in all treated rats. The results indicate that fluoxetine can reduce the playful pins of juvenile rats, but that prior social learning mediates the strength of these effects. © 1996 Wiley-Liss, Inc.

Key words: fluoxetine, play, social dominance, serotonin, rat

INTRODUCTION

Selective serotonin reuptake inhibitors (SSRIs—e.g., fluoxetine/Prozac®) have gained clinical popularity because of their efficacy at reducing symptoms of depression and decreased incidence of side effects relative to older antidepressants such as the tricyclics [Preskorn, 1994]. Clinicians have speculated that SSRIs can ameliorate not only the vegetative symptoms of depression, but also temperamental vulnerabilities that contribute to depression, such as chronic irritability and hostility [Kramer, 1992]. Indeed, a few practitioners have utilized SSRIs successfully to remediate impulsive aggression in patients with personality-disorders [Kavoussi et al., 1989].

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Meanwhile, primate researchers have developed models to test the hypothesis that SSRIs can change temperamental behaviors as well as their social consequences. Raleigh et al. [1991] found that male vervet monkeys given SSRIs consistently attain dominant positions over conspecifics following removal of alpha males from established groups. Conversely, vervet males given serotonin antagonists under the same circumstances assume subordinate positions. Thus, increased serotonergic activity enhances social dominance in male vervet monkeys, as assessed by number of conflicts won. Interestingly, treated vervets attained dominance not by aggressing against other conspecifics, but rather by forming cooperative coalitions with them (mainly with females). Behaviorally, vervets who received SSRIs shows increased grooming, approaching, and proximity behaviors, but fewer aggressive behaviors. Thus, vervets treated with SSRIs appear to attain dominance via increased cooperation rather than competition.

However, researchers investigating the effects of serotonergic manipulations in the social behavior of rats have not routinely observed that interventions which enhance serotonergic activity also increase social dominance. For instance, Ellison [1976] lesioned the serotonergic systems of a subset of male rats with 5,6-dihydroxytryptamine and then reintroduced the lesioned rats to a colony of normal and dopamine-lesioned rats. In addition to spending more time in the open, running more in activity wheels, and approaching humans more often than controls, the serotonin-depleted animals fought more violently with their conspecifics and rose in dominance for the first month following their lesions (although they subsequently fell back to baseline dominance levels). More recently, Bonson and Winter [1992] found that pretreatment with the serotonin agonist quipazine diminished rats' success in competing with a partner for a high-incentive food. In sum, both of these studies indicate that a *reduction* in serotonergic tone encourages dominant behavior in rats.

The discrepancy between serotonin's role in the dominant behavior of primates vs. rats could be explained in several ways. First, Bernstein [1981] has noted that the means of attaining social dominance may vary among species. Thus, even if serotonergic manipulations have similar behavioral effects in different species, behaviors that lead to dominance in one species may lead to subordination in another. For instance, in "harem" species such as baboons, where aggressive behavior appears to lead to dominance, serotonergic intervention might reduce dominance. However, in a "pair-bonded" species such as vervet monkeys, where affiliative behavior may lead to dominance, serotonergic intervention may promote dominance. Second, even in the same species, the means of attaining social dominance may vary across different situations (e.g., feeding, guarding territory, mating). For instance, high serotonergic tone may encourage affiliative dominance behaviors such as play, but discourage aggressive dominance behaviors such as territorial fighting. To evaluate such possibilities in rats, we conducted three studies that focused on the effects of SSRIs on play dominance in juvenile rats, an assertive form of social interaction that is a positive incentive for both participants [Calcagnetti and Schechter, 1992; Normansell and Panksepp, 1989].

Rough-and-tumble play presents a non-agonistic social-dominance paradigm in which assertive behaviors can be reliably measured in young animals [Panksepp, 1981; Panksepp et al., 1985]. Previously isolated juvenile rats show vigorous play activities when placed together in a chamber for brief observation periods. The rats first direct play attacks towards the nape of each others' necks. An attacked rat will rotate to a supine position to defend the nape of its neck against these dorsal contacts at which

point the attacker can pin the other animal to the ground momentarily [see Pellis and McKenna, 1992; Siviy and Panksepp, 1988]. During play, these pins and dorsal contacts can be coded with high reliability. Dominance has been inferred from the number of times one rat "pins" another [e.g., Panksepp, 1981], although this affiliative type of play dominance does not necessarily translate into the aggressive dominance more typically seen in adult rats [Pellis and Pellis, 1991].

To determine whether an SSRI would increase or decrease pins as a measure of play dominance and dorsal contacts as a measure of play solicitation, we conducted three experiments. First, we conducted a dose-response analysis of fluoxetine's effects on rough-and-tumble play. Second, we treated one member of pre-established play pairs with fluoxetine and the other with vehicle, to determine whether the fluoxetine effect varied as a function of prior play dominance. Third, we replicated the same procedure with rats who had had no prior play experience, to determine the extent to which fluoxetine affected play dominance in the absence of an established dominance relationship.

EXPERIMENT 1

Method

Eight pairs of juvenile Long-Evans rats (four male, four female) were separated from their mother at 22 days of age and individually housed in separate 23 × 10 × 13 cm steel mesh cages with water and food provided ad libitum. The rats were matched by litter, sex, and weight (to within 5 g of each other). Matched pairs received 3 days of habituation to the play paradigm. During habituation, each rat received an injection of vehicle (1 ml/kg distilled water) intraperitoneally 20 min before being placed in a 31 × 32 × 32 cm lucite test cage situated in a soundproof chamber and illuminated with a 25 W red light, where they were observed for five min. An experimenter scored each rat's number of pins, the duration of the pins, and number of dorsal contacts as described previously [Panksepp et al., 1984]. For the experimental sessions, both members of each pair received vehicle, 2.5, 5.0, or 10.0 mg/kg fluoxetine intraperitoneally 20 min before each of four successive daily play sessions. Each pair received a different treatment sequence for each of the four test days, as determined by a randomized Latin Squares design.

Result

The effect of fluoxetine on number of pins was assessed with a one-way repeated measures analysis of variance (ANOVA) that compared the number of pins across the four drug conditions (see Table I). A significant main effect indicated that type of treatment significantly affected a rat's number of pins during play, $F(3,45) = 5.71$, $P <$

TABLE I. Mean Pins, Pin Duration, and Dorsal Contacts by Drug Treatment (Experiment 1)†

	Drug treatment			
	Vehicle	2.5 mg/kg	5 mg/kg	10 mg/kg
Pins	20.00 (1.9)	19.75 (1.7)	12.25 (2.2)*	13.87 (2.0)*
Pin duration	1.14 (.1)	1.22 (.1)	1.38 (.2)	1.42 (.2)
Dorsal contacts	46.9 (3.4)	46.0 (3.6)	39.3 (5.5)	39.7 (5.0)

†Standard errors of the mean in parentheses.
*Significantly different at $P < .005$.

.01. Linear contrasts indicated that rats given 10 and 5 mg/kg fluoxetine pinned significantly less than rats given 2.5 mg/kg or vehicle, $F(3,45) = 11.51$, $P < .005$ (see Table I). Similar analyses of pin duration and number of dorsal contacts revealed no significant effects of fluoxetine treatment. As in our previous work, no significant differences in any of the dependent variables were evident for male versus female rat pairs.

Discussion

Pretreatment with 5.0 and 10.0 mg/kg fluoxetine reduced juvenile rats' number of pins, but not pin durations or dorsal contacts during 5 min play sessions. Other antidepressants (e.g., imiprimine, amytriptilene, clomiprimine) have been shown to reduce both pins and dorsal contacts [Panksepp et al., 1987]. However, the contribution of pharmacological manipulations of serotonin in these previous findings is hard to interpret, since these drugs also have anticholinergic as well as other neurotransmitter effects, and anticholinergic drugs powerfully reduce play [Thor and Holloway, 1984; Panksepp et al., 1984]. The fact that fluoxetine, an SSRI, also reduced pins indicates that a more specific facilitation of serotonin can reduce play dominance, which is consistent with other work using serotonin receptor agonists and antagonists [e.g., Normansell and Panksepp, 1985]. On the basis of these findings, we selected the minimal effective dose of 5 mg/kg to investigate whether daily fluoxetine treatment of only one member of an established play pair would cause a specific reduction in that member's pins, yet leave the other member's pins unaffected.

EXPERIMENT 2

Pairs of rats engaged in four pre-drug play sessions. Based on mean pins during these four trials, one animal was designated as dominant, and the other as subordinate. Half of the dominant rats and half of the subordinate rats received 5 mg/kg fluoxetine, while their partners received vehicle before each of 10 successive daily play trials. This allowed analysis of differential fluoxetine effects on play for dominant and subordinate animals.

Method

Nineteen pairs of rats (ten male, nine female) completed the same procedure as in the previous experiment, with the following modifications: (1) one member of each pair received 5 mg/kg fluoxetine 20 min prior to each play session, while the other received vehicle (in half of the pairs, the dominant animal received fluoxetine and the subordinate received vehicle, while in the remaining half, the subordinate animal received fluoxetine and the dominant received vehicle); (2) rat pairs played for 10 consecutive daily 5 min sessions.

Result

On the day prior to drug treatment, the dominant animals exhibited a mean of 14.7 ± 2.1 (SEM) pins while the subordinate animals exhibited a mean of 7.8 ± 1.9 pins ($t(22) = 2.66$, $P < .05$, all figures quoted hereafter as mean ± standard error of the mean). Effects of fluoxetine on number of pins for dominant and subordinate animals was assessed with two separate 2 (drug treatment) × 10 (day) repeated

measures ANOVAs. Similar analyses were conducted for pin duration and dorsal contacts.

Dominant animals showed no significant differences in pinning whether they had been administered vehicle or fluoxetine prior to play sessions (Fig. 1a), nor did they differ in terms of dorsal contacts or pin duration (data not shown). As one would expect from the normal play ontogeny curve, both groups' pinning decreased overall during testing from a mean of 20.8 ± 2.2 to a mean of 14.0 ± 2.8, $F(9,153) = 3.76$, $P < .001$. Dorsal contacts similarly decreased over testing for both groups from a mean of 46.5 ± 2.8 to a mean of 33.2 ± 2.8 during testing ($F(9,153) = 2.00, P < .05$), while pin duration

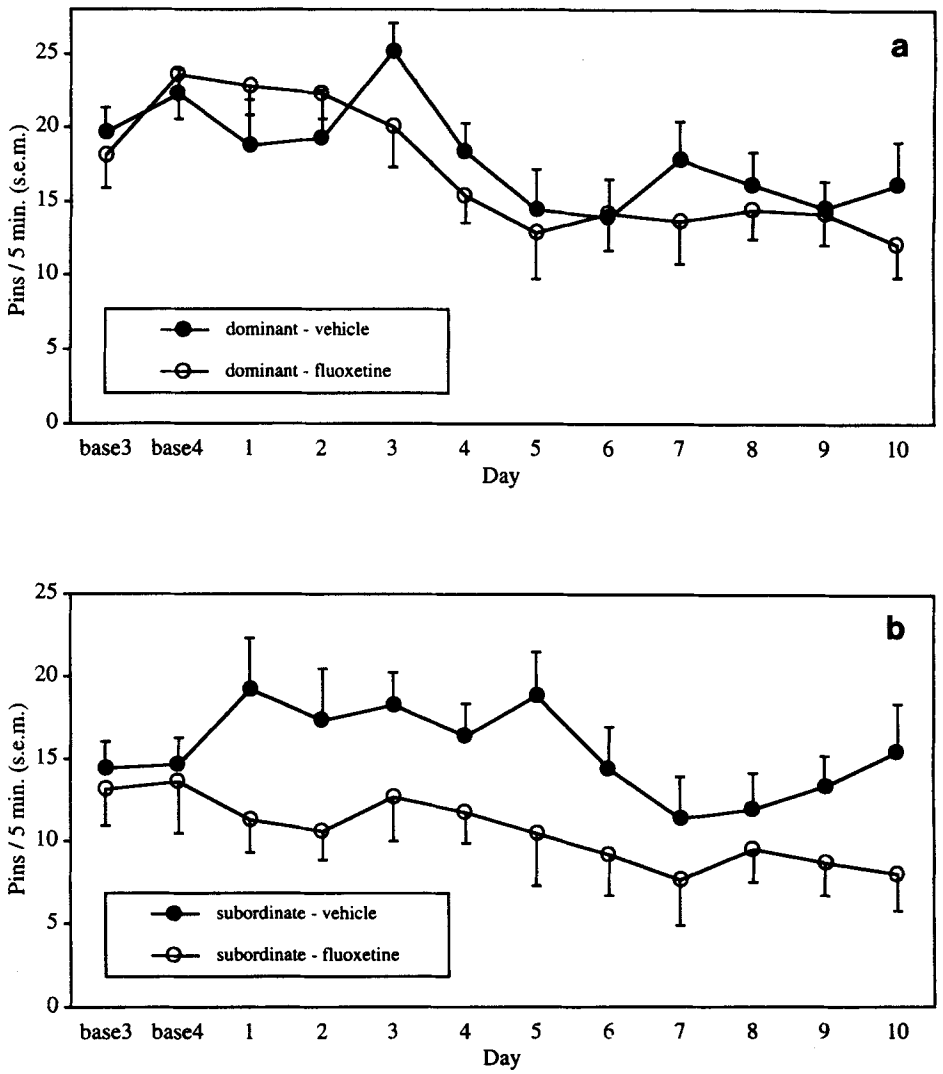

Fig. 1. **A:** Mean pins for dominant animals treated with vehicle vs. fluoxetine (mean ± SEM, Experiment 2). **B:** Mean pins for subordinate animals treated with vehicle vs. fluoxetine (mean ± SEM, Experiment 2).

increased for both groups from a mean of 1.4 ± .2 to a mean of 1.8 ± .3, $F(9,153) = 2.59, P < .01$.

On the other hand, subordinate animals did show significant differences in pinning as a function of fluoxetine treatment (Fig. 1b). Subordinates treated with fluoxetine pinned less overall (10.0 ± 1.6) than those treated with vehicle (15.7 ± 1.7), $F(1,17) = 5.28, P < .05$. However, fluoxetine- vs. vehicle-treated subordinates did not differ in pin durations or dorsal contacts (data not shown). As with dominant animals, both groups of subordinate rats also showed an overall decrease in pins from a mean of 15.1 ± 2.0 to a mean of 11.6 ± 2.0 over the course of testing, $F(9,153) = 4.44, P < .001$. Subordinates' dorsal contacts similarly decreased overall from a mean of 39.2 ± 2.9 to a mean of 32.8 ± 2.6, $F(9,153) = 4.44, P < .001$. Unlike the dominant animals, subordinates' pin durations did not significantly increase over the course of testing.

These data suggested that fluoxetine largely affected play dominance by specifically reducing subordinate partners' pins. However, there is an alternative explanation for these findings. Fluoxetine may have exerted its effects not only by changing one partner's behavior, but rather, by altering the pre-established relationship between partners.[1] As mentioned previously, play pairs usually develop stable asymmetries in pinning. Just as one can assess individual play dominance with absolute number of pins per subject, one could also assess the dyadic play dominance relationship with a pairwise measure of pinning asymmetry. To this end, we constructed a pinning asymmetry ratio (PAR), which was calculated as follows: (dominant pins – subordinate pins) / (dominant pins + subordinate pins). We hypothesized that if fluoxetine treatment altered not just individual behavior, but also the dyadic behavioral balance, PAR should differ between pairs in which the dominant partner received fluoxetine treatment and pairs in which the subordinate partner received fluoxetine.

A 2 (dominant received fluoxetine vs. subordinate received fluoxetine) × 10 (day) repeated measures ANOVA revealed that, indeed, pinning asymmetry ratio differed depending on which partner received fluoxetine treatment. Pairs in which the subordinate partner received fluoxetine and the dominant partner received vehicle had a mean PAR of .25 ± .04, while pairs where the dominant partner received fluoxetine and the subordinate partner received vehicle had a mean PAR of 0 ± .04, $F(1,17) = 5.49, P < .05$ (see Fig. 2). PAR did not change reliably over the course of testing. Thus, when the subordinate partner received fluoxetine, pinning asymmetry remained at baseline levels (e.g., the dominant rat executed an average of 25% more pins than the subordinate rat), but when the dominant partner received fluoxetine, pinning asymmetry collapsed to zero.

Discussion

On an individual basis, fluoxetine pretreatment significantly reduced subordinate rats' pinning, but not that of dominant rats. However, on a dyadic basis, fluoxetine treatment did not affect pinning asymmetry when the subordinate animal received treatment, but the initial pinning asymmetry collapsed when the dominant animal received treatment. Thus, the individual effect was not only due to reduction in the pins of fluoxetine-treated subordinates but also an increase in pins by the vehicle-treated subordi-

[1]We wish to thank an anonymous reviewer who brought this interpretation to our attention.

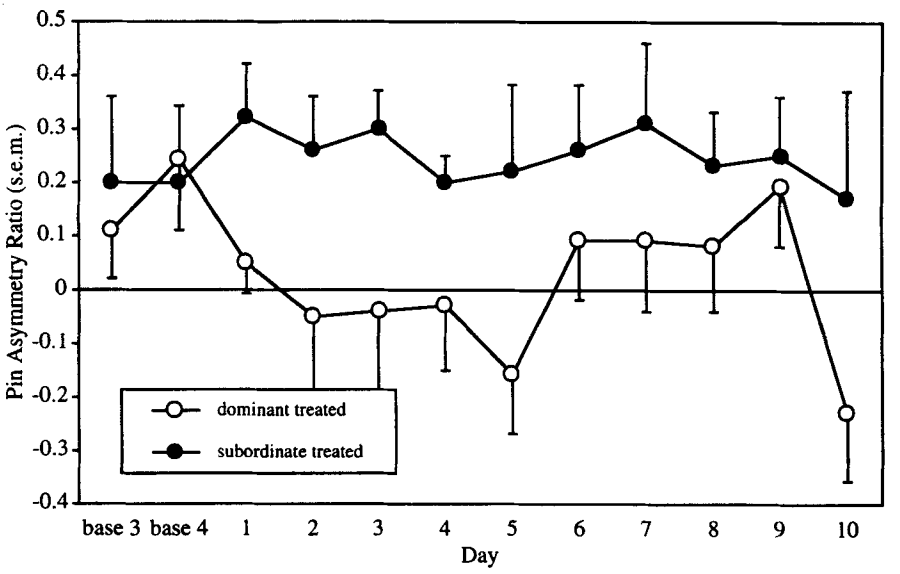

Fig. 2. Pin asymmetry ratio for pairs in which the dominant vs. subordinate partner received fluoxetine (mean ± SEM, Experiment 2).

nates who played with fluoxetine-treated dominants. Notably, fluoxetine did not reduce dorsal contacts, which appears to be associated with a desire to play.

These findings strongly suggested that a pre-existing social relationship can alter the effects of fluoxetine on play dominance. To further test this possibility, we removed the influence of prior social learning by conducting the same experiment with play-naive rats. We predicted that all rats treated with fluoxetine would show a decrement in pinning.

EXPERIMENT 3

In the following experiment, rat pairs played after one member received fluoxetine and the other received vehicle, but without having established a prior play dominance relationship. This procedural modification allowed us to evaluate whether fluoxetine treatment would systematically affect play dominance in the absence of prior social learning.

Method

Procedures were identical to Experiment 2 with the exception that eight pairs of rats were tested (four male, four female) with no prior play experience. One member of each pair of 22-day-old rats received 5 mg/kg fluoxetine, while its partner received vehicle 20 min before each play session. Daily testing continued for 10 consecutive days.

Results

Effects of fluoxetine on number of pins was assessed with a 2 (drug treatment) × 10 (day) repeated measures ANOVA. Similar analyses were conducted for pin duration

and number of dorsal contacts. A significant main effect for drug treatment indicated that rats who received fluoxetine pinned less overall (19.1 ± 1.2) than rats who received placebo (14.3 ± .9), $F(1,12) = 5.25$, $P < .05$ (see Fig. 2). Although the treatment × trial interaction was not significant ($F(9,108) = 1.51$), there was considerable daily fluctuation of this effect, with the most pronounced differences occurring on days 2 and 7 (see Fig. 3a). Analysis of dorsal contacts yielded a similar main effect, in which fluoxetine-treated animals showed fewer dorsal contacts (31.2 ± 1.5) than those who received placebo (38.7 ± 1.4), $F(1,12) = 7.58$, $P < .05$, with a comparable fluctuation of the effect

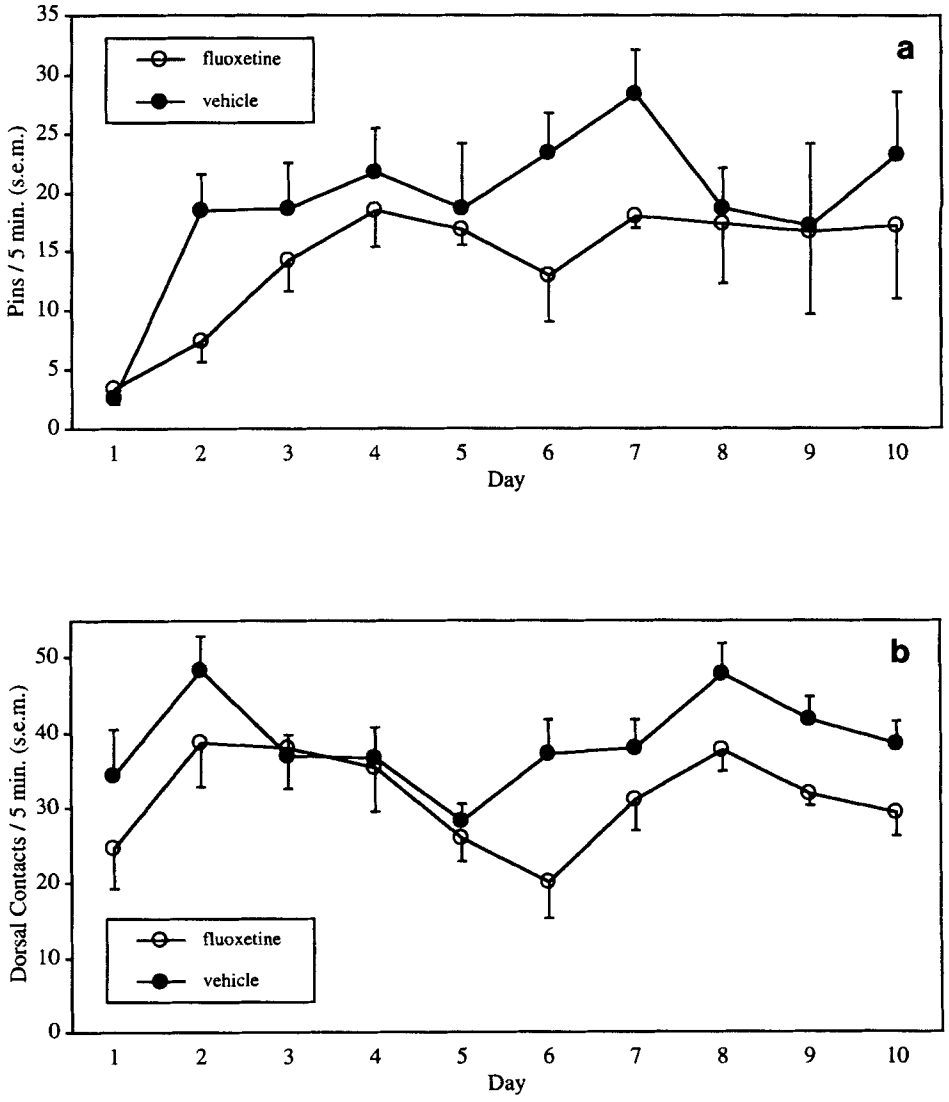

Fig. 3. **a:** Mean pins for rats treated with vehicle vs. fluoxetine (mean ± SEM, Experiment 3). **b:** Mean dorsal contacts for rats treated with vehicle vs. fluoxetine (mean ± SEM, Experiment 3).

across days (see Fig. 3b). There was no significant effect of fluoxetine treatment on pin duration.

Discussion

As with all animals in Experiment 1 and the subordinate animals in Experiment 2, rats treated with fluoxetine across 10 days pinned less than vehicle-treated rats. Contrary to the results of Experiment 1, fluoxetine-treated rats in this study also exhibited fewer dorsal contacts than did vehicle-treated rats. Perhaps this is because animals were not given a chance to establish a playful relationship prior to receiving the fluoxetine treatment. In general, these results suggest that increases in serotonin may reduce the energization of play behavior, since other investigators have reported a general "calming" (but not necessarily sedating) effect of fluoxetine in other species [cf. Raleigh et al., 1985].

GENERAL DISCUSSION

Taken together, these findings indicate that fluoxetine treatment can reduce play dominance in juvenile rats, but these effects depend on the previously established play dominance relationship among the animals. When a subordinate member of a play pair received fluoxetine, pinning asymmetry remained at baseline levels, but when a dominant member received fluoxetine, pinning asymmetry collapsed to zero. Contrary to what individual analyses initially suggested in Experiment 2, this effect appeared to be due to an increase in the pins of vehicle-treated subordinates who were matched with the fluoxetine-treated dominants. Apparently, then, fluoxetine had some effect on the treated dominants that led their vehicle-treated partners to pin them more. Without prior social learning, all animals who received fluoxetine pinned less and made fewer dorsal contacts. Thus, social learning constrained the effects of fluoxetine on play dominance.

These findings parallel those found with vervet monkeys. For example, in an established social hierarchy, dominant males are more behaviorally affected by fluoxetine treatment than subordinate males. Specifically, they show more calm and nonthreatening behaviors such as approaching, grooming, resting, and eating [Raleigh et al., 1985]. Further, in a socially ambiguous situation (i.e., the dominant male had been removed from the group), male vervets who receive fluoxetine also behave more calmly, which enables them to form coalitions and ultimately elevates their dominance status [Raleigh et al., 1991]. So in both male vervets and rats, fluoxetine seems to have calming and prosocial effects.

Although fluoxetine treatment appeared to promote calmer behavior in both primates and rats, our findings create an apparent paradox. In primates, fluoxetine's calming effect ultimately served to increase dominance. In playing rats, on the other hand, fluoxetine treatment eliminated the dominant partner's pinning advantage. However, fluoxetine treatment did not reduce the absolute number of dominant rats' pins. Instead, it affected the dominant rat in such a way that his or her vehicle-treated partner pinned more. Thus, the collapse of pinning asymmetry may not result from a loss of dominance per se, but rather increased reciprocation on the part of the subordinate, a hallmark of high-quality play. Because of our relatively molar behavioral measures, we do not know how the fluoxetine-treated dominant rat encouraged his or her subordinate partner to pin more, but this question may be amenable to more sophisticated behavioral coding techniques such as those employed by Pellis and Pellis [1991]. When inter-

preted in this light, our findings may be consistent with the fact that fluoxetine treatment only enhanced primate dominance by increasing calm and nonthreatening social displays such as approaching and grooming.

Taken together, these findings suggest that the social effects of temperamental changes induced by serotonin depend on an animal's prior social status. In both primates and rats, serotonergic activation appears to calm animals. However, calmer behavior can have varying consequences, depending on species differences and situational circumstances (e.g., social status). For subordinate animals, fluoxetine may not change preexisting relationships, but may reduce perceived stress. For instance, chronic fluoxetine treatment (i.e., longer than 2 weeks) of subordinate adult male rats in a naturalistic colony setting reduces stress levels as indexed by corticosterone levels, and selectively reduces defensive but not offensive behavior [McKittrick et al., 1994]. For dominant animals, fluoxetine treatment may reduce threatening displays, which may create opportunities for increased social reciprocation. For instance, fluoxetine treatment of dominant adult male rats in a resident-intruder paradigm selectively reduces offensive aggressive behaviors while leaving social exploration intact or even increasing it [Sijbesma et al., 1991]. Thus, while fluoxetine may have similar effects on temperament in different mammals, the social repercussions of these effects appear to depend critically on an individual's pre-existing social status.

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