Is the striatum involved in passive-avoidance behavior?

A commentary

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The role of the striatum in passive avoidance (PA) behavior has been reviewed in light of recent data by Olmstead and Villablanca (1980) reporting a lack of involvement of the caudate nucleus in this behavior. Evaluation of their data revealed that a small deficit in PA was demonstrated in the caudotomized adult cat group. Similarly, our own data with kainic-acid-induced striatal lesions in rats resulted in impairments in passive-avoidance acquisition and retention. It is concluded that the striatum is involved in PA behavior, although the underlying behavioral processes that contribute to this role are not clearly understood.

Recently, Olmstead and Villablanca (1980) suggested that the striatum (caudate nucleus and putamen) was not involved in passive-avoidance learning and that previous findings of PA deficits following striatal lesions probably reflected experimental artifacts. Given that these suggestions might have serious influences on the understanding of striatal functioning, the aim of this paper was to examine critically Olmstead and Villablanca's findings in the context of the problems involved in the interpretation of changes in avoidance learning following central nervous system lesions.

Olmstead and Villablanca's (1980) Study

Olmstead and Villablanca (1980) compared the PA performance of cats, including groups with large caudate and frontal cortical lesions. Eight cats with bilateral caudate lesions were operated on as kittens (kitten BAc), while 11 others were operated on as adults (adult BAc). Initially, for two trials daily over 2 days, the animals were placed on a platform and allowed to step down without shock. After the fourth trial, an inescapable shock was delivered, and PA was assessed as a tendency for the animals on later

trials to remain on the platform up to a cutoff of 300 sec. It was found that, in comparison with other groups, the adult BAc group was significantly slower to leave the platform on Trial 1 of Day 1. The PA performance of all participating subjects was measured on three occasions, Days 1, 2, and 7 after the learning trial. It was reported that there were no significant differences between the adult BAc and other groups on the first two testing sessions. However, on Test Day 7, the adult BAc group was significantly impaired. Olmstead and Villablanca did not interpret this significant impairment in the adult BAc group as indicating a PA deficit, but they noted that since 44% of the adult BAc animals avoided according to criterion latency, this indicated that as a group the cats had acquired and retained some learning. Olmstead and Villablanca argued that the "subtle" behavioral changes in the adult BAc subjects "merely reflect some of the peculiarities in reactivity and activity" (p. 342) and concluded that "we have a strong indication of a lack of a role of the striatum in passive avoidance" (p. 343).

Several conceptual issues are raised by Olmstead and Villablanca's conclusions.

The Question of Negative Findings

When assessing the pattern of avoidance changes as suggested by Olton (1973), it is clear that the ap-

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Table 1
Passive Avoidance Following Caudotomy

Retention-Extinction Trials	Percentage of Subjects Not Avoiding			
	N	1	2	3
Combined Sham-Operated Controls	11	9	0	0
Adult BA _c	9	33	33	56

Note—Data are from Olmstead and Villablanca (1980, Table 1, p. 341).

parent absence of involvement of a particular brain lesion in an avoidance task suggests crucial information concerning the behavioral role of that neural structure. Therefore, the question of Type II errors occurring in any given study should be raised. The design of Olmstead and Villablanca's (1980) study of PA involved the comparison of a relatively large number (nine) of groups, some of quite small size (ranging from five to nine subjects). The suggestion that a real, but rather small, effect might have been overlooked in the study is supported by the following comparison of the adult BA_c group with the combined PA performances of the two sham-lesioned control groups used in the study (Table 1).

Inspection of Table 1 suggests two points. First, the PA task was relatively easy since no extinction or retention deficits were demonstrated by the controls by the 2nd or 3rd day of PA testing. Second, in relation to the controls, there appears to be a tendency for the adult BA_c group to be impaired on PA, this effect being most marked on the third session. Taking into account that this group initially had a significantly greater tendency to remain on the platform, it is difficult to see how the effect can be labeled as "subtle." Therefore, an alternative interpretation of Olmstead and Villablanca's (1980) findings is that large bilateral caudate nucleus lesions in adult cats result in a PA retention deficit. The effect, at least initially, appears quite small for a particularly simple task, so that a rather powerful experimental design is necessary to uncover it. The fact that there was not a 100% deficit in PA in the caudate-damaged group in no way detracts from the above interpretation. There are valid reasons, such as the almost-certain involvement of other neural systems in the task or the arbitrary nature of the criteria used to assess PA learning. Olmstead and Villablanca's criterion was that of finding an absolute deficit in PA, instead of judging performance in relation to controls.

Task Parameters and Avoidance Performance. Evidence from Previous Studies Concerning the Effects of Caudate Lesions on PA

Olmstead and Villablanca reviewed four lesion studies relating caudate nucleus lesions in adult cats to avoidance performance. Three of these studies

(Laursen, 1963; Thompson, 1959; Thompson & Mettler, 1963) demonstrated avoidance deficits, but they were *active*-avoidance deficits and not relevant to the question of PA deficits (see Olton, 1973). The remaining study (Fox, Kimble, & Lickey, 1964) reported significant deficits in caudate-damaged cats on a PA task. Olmstead and Villablanca (1980) reported that, in agreement with their results, Reyes-Vasquez, Zarco-Coronado, and Brust-Carmona (1978) found that 6-hydroxydopamine injections into the anteroventral caudate nuclei did not result in deficits for a PA task. However, given other reports that DA depletion does not disrupt PA but, rather, disrupts active-avoidance behavior (e.g., Cooper, Breese, Grant & Howard, 1973; Fibiger & Phillips, 1976; Neill, Boggan & Grossman, 1974; Zis, Fibiger, & Phillips, 1974), the lack of a PA deficit reported by Reyes-Vasquez et al. (1968) is not surprising. The weight of evidence suggests that, while DA apparently is not involved in PA, there are other neurotransmitter systems in the striatum, such as the cholinergic and GABAergic systems (Coyle, McGeer, McGeer, & Schwarcz, 1978) that might well mediate PA behavior (Deadwyler, Montgomery, & Wyers, 1972; Haycock, Deadwyler, Sideroff, & McGaugh, 1973; Vazquez, Tellez, De La Mora, & Brust-Carmona, 1979).

In relation to the findings concerning the effects of caudate damage on avoidance in rats, Olmstead and Villablanca (1980) draw two conclusions. The first is that the literature is quite unclear, and the second is that the behavioral findings are obscured by the artifact of damage to capsular fibers coursing through the rat's striatum.

With regard to the literature, Olmstead and Villablanca (1980) failed to distinguish between the pattern of findings concerning the different avoidance paradigms. In fact, in all the studies in the area quoted by Olmstead and Villablanca (Kirkby & Kimble, 1968; Mitcham & Thomas, 1972; Sanberg, Pisa, & Fibiger, 1979; Winocur, 1974), there is a clear consensus that striatal lesions in the rat do result in PA deficits. This view has also been supported by similar findings of Glick and Greenstein (1973) in mice and Rothman and Glick (1976) in rats. The conditions under which a PA deficit has been most reliably found were for PA tasks involving the inhibition of a learned rather than a simple unpracticed response (Kirkby & Kimble, 1968; Winocur & Mills, 1969). In fact, the reason for the small PA deficit found by Olmstead and Villablanca (1980) and the differences between their findings and those of Fox et al. (1964) might well reflect the "unpracticed" PA paradigm chosen by Olmstead and Villablanca (1980).

Olmstead and Villablanca (1980) were justified in pointing out the possibility that damage to fibers of passage by striatal lesions in the rat complicates the interpretation of avoidance performance. However,

they were mistaken in asserting that "to date ... there have been no adequately controlled studies for that fiber damage" (p. 343). Investigators in the area have been well aware of this complication and have attempted to control for it. For example, there have been numerous studies involving the intrastriatal injections of substances that are known to disrupt specific neurotransmitter functioning without, presumably, affecting fibers of passage, which have demonstrated the involvement of striatal systems in both active (e.g., Neill & Grossman, 1970; Neill et al., 1974) and passive (Haycock et al., 1973; Prado-Alcala, Cruz-Morales, & Lopez-Miro, 1980; Vazquez et al., 1979) avoidance.

In a more novel study using kainic-acid-induced lesions of the striatum, which apparently spare fibers of passage (Coyle et al., 1978), Sanberg, Lehmann, and Fibiger (1978) demonstrated PA deficits in the rat. However, in a subsequent study (Sanberg et al., 1979), some of the striatally lesioned rats also had hippocampal damage as a result of the intrastriatal kainic acid injections. While the presence of hippocampal damage in some rats may complicate the attributing of the PA deficits to the striatal pathology per se, Sanberg et al. (1979) observed that the deficit appeared independently of hippocampal damage. Also, in subsequent studies using rats with striatal lesions induced by kainic acid, but without any hippocampal damage, PA impairments were found using tasks involving the inhibition of learned (Pisa, Sanberg, & Fibiger, 1978; Sanberg, 1978) or unpracticed (see Table 2) responses.

The majority of capsular fibers traversing the anterodorsal striatum of the rat are likely to be passing to or from the anterior cortex (Knook, 1965). It is interesting to note that Olmstead and Villablanca (1980) did not report any PA deficits for their groups with very large bilateral anterior cortex lesions. Therefore, in terms of Olmstead and Villablanca's own findings, damage to capsular fibers does not adequately explain the PA deficits found following electrolytic lesions of the striatum (Kirkby & Kimble, 1968; Winocur, 1974).

We are not arguing here either that the anterior cortex is not involved in PA or that damage to capsu-

lar fibers does not have behavioral consequences. Rather, the point is that the findings discussed above support the conclusion that damage to striatal neuronal tissue alone is sufficient to disrupt avoidance learning, including PA.

We have reviewed above those lesioning studies demonstrating impairments in PA; however, there is also extensive evidence of PA retention deficits following posttrial electrical stimulation of the striatum (e.g., Gold & King, 1972; Kesner & Wilburn, 1974; Wyers & Deadwyler, 1971; Wyers, Deadwyler, Hirasuna, & Montgomery, 1973; Zornetzer & Chronister, 1973). These experiments concluded that the striatum was involved in aspects of acquiring and retaining a PA response.

Some Theoretical Considerations

Although the surgical technique adopted by Olmstead and Villablanca (1980) resulted in extremely large bilateral caudate nucleus lesions, it must be noted that the putamen remained intact. In strict neuroanatomical terms, there is no basis for their conclusion that the striatum, which consists of the caudate nucleus and putamen, is not involved in PA. While this point might seem pedantic, it has certain implications. It is quite likely that some studies that have shown PA deficits with lesions that involved the more ventral aspects of the striatum in the rat (Kirkby & Kimble, 1968; Winocur, 1974) damaged tissue that is homologous to the putamen in the cat.

One of the problems with using extirpation techniques to investigate striatal involvement in PA is that, following caudate nucleus damage, an active reorganization of the brain might occur in response to the surgical insult (see Schoenfeld & Hamilton, 1977). During the relatively long lesioning-testing interval (3 months) used by Olmstead and Villablanca, adaptive changes could have occurred in the caudate-nucleus-damaged brains, minimizing the PA deficits.

There are numerous, and sometimes apparently conflicting, interpretations of striatal involvement in learned behavior (Polgar & Kirkby, 1977). We agree with Olmstead and Villablanca (1980) that subjects with striatal damage can be characterized as showing heightened levels of reactivity to environmental and

Table 2
Effect of Bilateral Kainic Acid Lesions of the Striatum on Acquisition and Retention of a Step-Down Passive Avoidance Test

Group	N	Initial Step-Down Latency†		Time to Criterion†		Number of Descents to Criterion		24-h Retention: Step-Down Latency†	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Control	10	5.02	1.40	263.1	14.7	3.16	.32	151.3	16.1
Kainic Acid Striatal	10	6.62	1.73	394.2	28.3*	5.72	.63*	95.2	24.5*

Note—The rats were lesioned as described in Sanberg (1980), which included pretreatment with diazepam to avoid hippocampal damage, and tested for PA as described in Sanberg et al. (1979). *Significantly different from controls ($p < .01$, two-tailed test).

†In seconds.

physiological stimuli (Kirkby, 1973, 1976, 1978; Kirkby & Polgar, 1974; Pisa et al., 1980; Polgar & Kirkby, 1980; Sanberg et al., 1979). Therefore, it is possible that exaggerated arousal reactions may account for PA deficits in striatally lesioned animals. However, there are other possible interpretations of the PA deficits in animals with striatal lesions in terms of striatal involvement in memory functions (Pisa, Sanberg, & Fibiger, 1981; Prado-Alcala & Cobos-Zapiain, 1979; Prado-Alcala, Grinberg, Arditti, Garcia, Prieto, & Brust-Carmona, 1975; Zornetzer & Chronister, 1973). Therefore, the PA retention deficit in caudotomized adult cats in Olmstead and Villablanca's (1980) study could reflect a limited impairment in the retention of information for successful PA performance.

All the evidence reported here clearly implicates a role for the striatum in PA behavior. However, it is evident that further work is necessary to elucidate the underlying behavioral processes involved with striatal modulation of PA behavior.

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