the compound to inactive β-carboline-3-carboxylic acid ($K_i$, ~ 25,000 n$M$). This has been confirmed in our laboratories, and has stimulated the development of substantially longer lived, pharmacologically active β-carboline derivatives (*12*).

We have demonstrated that 3-HMC, which antagonizes the anxiolytic and anticonvulsant actions of diazepam, also antagonizes the sleep-inducing properties of the benzodiazepine flurazepam. Thus the hypnotic actions of flurazepam may be mediated through interaction with the benzodiazepine receptor. At slightly higher doses, 3-HMC increased wakefulness by significantly increasing sleep latency and reducing non-REM (but not REM) sleep (Fig. 1A and Table 1). Thus 3-HMC is not merely a benzodiazepine antagonist but exerts a pharmacological action on sleep opposite that produced by benzodiazepines (*13*). Although other drugs (such as amphetamines and methylxanthines) can reduce sleep (*14*), they also invariably cause profound alterations in behavior and motor activity (*15*). Compounds that reduce sleep without eliciting major changes in motor activity may, therefore, be more properly termed "somnolytics." The suggestion that benzodiazepine receptors (and, by implication, the endogenous substrates that subserve these receptors) are involved in both physiological and pharmacologically induced sleep could lead to the development of β-carbolines or related compounds for treating human sleep disorders, especially those characterized by excessive somnolence.

WALLACE B. MENDELSON
*Adult Psychiatry Branch*
and *Unit on Sleep Studies,*
*Clinical Psychobiology Branch,*
*National Institute of Mental Health,*
*Bethesda, Maryland 20205*

MICHAEL CAIN
JAMES M. COOK
*Department of Chemistry, University of Wisconsin, Milwaukee 53201*

STEVEN M. PAUL
*Neuroscience Branch,*
*National Institute of Mental Health*

PHIL SKOLNICK
*Laboratory of Bioorganic Chemistry,*
*National Institute of Arthritis, Diabetes, and Digestive and Kidney Diseases,*
*Bethesda, Maryland 20205*

### References and Notes

1. W. B. Mendelson, *The Use and Misuse of Sleeping Pills* (Plenum, New York, 1980), pp. 25–38.
2. R. F. Squires and C. Braestrup, *Nature (London)* **266**, 732 (1977); H. Möhler and T. Okada, *Science* **198**, 849 (1977).
3. C. Braestrup, M. Nielsen, C. Olsen, *Proc. Natl. Acad. Sci U.S.A.* **77**, 2288 (1980); in *GABA and Benzodiazepine Receptors*, E. Costa, G. Gessa, G. D. Chiara, Eds. (Raven, New York, 1981), pp. 147–156; P. Skolnick, E. Williams, J. Cook, M. Cain, K. Rice, W. Mendelson, J. Crawley, S. Paul, in *Beta-Carbolines and Tetrahydroisoquinolines*, E. Usdin, Ed. (Liss, New York, 1982), pp. 233–252.
4. S. S. Tenen and J. D. Hirsch, *Nature (London)* **288**, 609 (1980); P. Skolnick, S. Paul, J. Crawley, K. Rice, S. Barker, R. Weber, M. Cain, J. Cook, *Eur. J. Pharmacol.* **69**, 525 (1981).
5. P. Cowen, A. Green, D. Nutt, I. Martin, *Nature (London)* **290**, 54 (1981).
6. N. Oakley and B. Jones, *Eur. J. Pharmacol.* **68**, 381 (1980).
7. W. Mendelson, R. Guthrie, G. Frederick, R. J. Wyatt, *Pharmacol. Biochem. Behav.* **2**, 553 (1974).
8. No evidence of EEG spiking was observed after administration of 3-HMC, suggesting that this compound does not produce seizure activity. Recently, however, other β-carbolines that bind to benzodiazepine receptors with high affinity (for example, β-CCE) were shown to elicit tonic and clonic convulsions (*9, 10*).
9. M. Schweri, M. Cain, J. Cook, S. Paul, P. Skolnick, *Pharmacol. Biochem. Behav.*, in press.
10. C. Braestrup and M. Nielsen, *Nature (London)* **294**, 472 (1981).
11. The effects of smaller doses of 3-HMC (< 50 mg/kg) on motor activity are not known but presumably are even smaller than those observed with the higher doses. No evidence of stereotypical behavior was observed when 3-HMC (50 mg/kg) was administered daily for 3 weeks (P. Skolnick *et al.*, unpublished observations).
12. In an in vitro study, 3-HMC and β-CCE were incubated in rat plasma (37°C) at concentrations of 100 to 200 nmole/ml. Equal 500-μl aliquots were removed at intervals and treated with 25 μl of perchloric acid to precipitate the protein. After centrifugation, portions of the supernatant were withdrawn and the amount of pharmacologically active compound remaining was determined by an assay based on the displacement of [$^3$H]diazepam [P. Skolnick, S. Paul, F. Goodwin, *Arch. Gen. Psychiatry* **36**, 78 (1979)]. Greater than 80 percent of the assayable material was lost after 2.5 minutes of incubation with β-CCE. In contrast, 3-HMC appeared more stable, since approximately 70 percent of the original activity was still present after 30 minutes of incubation. Parallel experiments at 0° to 4°C confirmed the enzymatic nature of this degradation, since 70 percent of the initial activity of β-CCE was still present after a 15-minute incubation. Thus, despite the modest affinity of 3-HMC for benzodiazepine receptors ($K_i$, ~ 1470 n$M$) compared to that of β-CCE ($K_i$, ~ 1 n$M$) (*3*), the former compound appeared more suitable for sleep studies due to its slower rate of degradation. Other β-carbolines substituted at C-3, such as 3-acetyl-β-carboline, have now been synthesized. They may also prove valuable in defining the role of benzodiazepine receptors in sleep because of their high affinities ($K_i$, ~ 50 n$M$) and relative resistance to metabolic degradation.
13. The increased wakefulness observed after 3-HMC administration is blocked by the benzodiazepine receptor antagonist CGS 8216 [A. Czernik *et al.*, *Life Sci.* **30**, 363 (1982)] at a dose (5 mg/kg) that has no intrinsic action on sleep (W. B. Mendelson *et al.*, unpublished observation). Also, the convulsant action of β-CCE is antagonized by Ro 15-1788 and CGS 8216 (*9*). These findings strongly support the hypothesis that both the antagonism of the hypnotic actions of flurazepam and the increased wakefulness observed after 3-HMC injection are mediated by benzodiazepine receptors.
14. E. Hartmann and J. Cravens, *Psychopharmacology* **50**, 171 (1976); A. Rechtschaffen and L. Maron, *Electroencephalogr. Clin. Neurophysiol.* **16**, 438 (1964); I. Karacan, J. I. Thornby, M. Anch, G. H. Booth, R. L. Williams, P. J. Salis, *Clin. Pharmacol. Ther.* **20**, 682 (1976).
15. D. S. Segal, *Science* **190**, 475 (1975); S. H. Snyder, J. J. Katims, Z. Annau, R. F. Bruns, J. W. Daly, *Proc. Natl. Acad. Sci. U.S.A.* **78**, 3260 (1981).
16. We appreciate the technical assistance of D. Carroll.

17 June 1982; revised 30 August 1982

## Nigral Transplants Reinnervating the Dopamine-Depleted Neostriatum Can Sustain Intracranial Self-Stimulation

Abstract. *Transplants of embryonic substantia nigra reinnervated the striatum and were able to sustain intracranial self-stimulation in rats with brain lesions induced by 6-hydroxydopamine. Dopaminergic drugs and alterations in current intensity produced typical changes in response rates. Animals with electrodes implanted into cortical grafts or into the denervated striatum failed to exhibit self-stimulation. These findings suggest that transplanted dopamine neurons convey specific, temporally organized information axonally to the striatum.*

A procedure for transplanting dopamine (DA) cells from the substantia nigra to ectopic cortical sites was recently described (*1*). Such grafts can reinnervate the host brain extensively and ameliorate several behavioral deficits produced by 6-hydroxydopamine (6-OHDA)–induced depletions of DA in the host, including spontaneous and drug-induced rotation, sensorimotor impairments, and akinesia (*1, 2*). However, it is not clear whether transplanted neurons simply provide a tonic release of DA into the striatum or whether the grafts release DA from their terminals on activation of the cell bodies. The phenomenon of intracranial self-stimulation (ICSS) (*3*) may provide a useful means for investigating this question, since (i) DA systems of the brain have been implicated in ICSS (*4*), (ii) there is a requirement that the animal integrate a specific input with its behavior, and (iii) brain stimulation can provide such an input to the DA cell bodies in the graft. We report here that DA-rich nigral grafts can sustain self-stimulation. This suggests that transplanted DA neurons may indeed transmit specific information to the reinnervated striatum.

The right nigrostriatal pathway in 20 young adult female rats of the Sprague-Dawley strain was lesioned with 6-OHDA, and cavities were made through the right parietal cortex and corpus callosum, exposing the dorsal surface of the caudate-putamen. Three weeks later grafts of embryonic substantia nigra ($N$ = 14) or embryonic isotopic cortex ($N$ = 6) were placed into the cavities (*5*). After 4 months bipolar stimulating elec-

trodes were implanted into the surviving grafts under visual guidance ("nigral" and "cortical" groups). In four of the rats that had received nigral transplants the graft could not be detected and electrodes were implanted directly into the dorsal caudate-putamen ("caudate" group) (*6*).

After a 1-week period to allow for recovery from surgery, all the animals received daily 30-minute sessions of ICSS training over a range of 10 to 100 μA (root-mean-square) for 2 weeks (*7*). In the group with surviving nigral grafts the median current intensity required to maintain reliable ICSS was 60 μA (range, 50 to 100 μA). On the final 3 days of training all the animals were tested at a constant current intensity of 100 μA. Nine (of ten) nigral rats responded at a rate in excess of 80 responses per session, whereas all cortical and caudate rats responded below this level [$2\hat{I}(2) = 21.02$, $P < .001$] (Fig. 1) (*8*). Over the next 3 weeks, current intensities were increased further until a stable level of responding was observed or a current intensity of 300 μA was reached. The same nine nigral rats had a median maximum response rate of 287 responses per session (range, 188 to 767). The remaining nigral rat (SN-2), all the cortical rats, and three of the four caudate rats never exceeded 50 responses per session; the remaining caudate rat (CP-6) never exceeded 80 responses per session.

In the nigral rats experimenter-controlled delivery of stimulation-induced strong contralateral rotation and ineffective grooming of the snout with the contralateral forepaw. With self-stimulation, these rats tended to position themselves in the corner of the box such that the walls inhibited stimulus-bound turning away from the lever, which in every case was pressed with the ipsilateral paw (the paw under the control of the intact striatum and not influenced directly by the stimulation). This suggests that the lever-pressing behavior was goal-directed and not simply an involuntary response induced by the stimulation.

A function relating rate of responding to current intensity was derived for the nigral rat showing the highest rates (Fig. 2) (*9*). This animal manifested a sigmoidal rate-intensity function with ascending and descending hysteresis, which is characteristic of ICSS in normal animals (*10*).

The rate-enhancing effect of amphetamine (*11*) and its blockade by the neuroleptic α-flupenthixol were investigated in the nine animals showing a positive ICSS response (*12*). Injections of *d*-amphetamine produced a dose-dependent enhancement of response rates [$F(4, 32) = 4.59$, $P < .01$] that was blocked by prior treatment with α-flupenthixol [main amphetamine effect, $F(1, 6) = 7.12$; main α-flupenthixol effect, $F(2, 12) = 6.15$; amphetamine × α-flupenthixol interaction, $F(2, 12) = 4.39$; all $P$'s $< .05$].

All of the surviving rats were killed for fluorescence histochemistry (*13*) 6 to 8 weeks after the completion of behavioral testing. Many fluorescent DA cells were identified in the grafts of all six surviving nigral animals, and reinnervation extending through one-eighth to one-third of the total volume of the head of the dorsal caudate-putamen was seen in each animal. The remainder of the denervated caudate-putamen on the lesioned side was devoid of fluorescence. Relations between electrode placement, DA cell bodies, striatal reinnervation, and ICSS rates are shown for each rat in Fig. 1. In the nigral rat with the highest ICSS rates (SN-8), the tip of the electrode was in the middle of the DA cell clusters (Fig. 2). In two nigral rats the track of the electrode was not clearly identifiable, but necrosis on the dorsal surface of the graft indicated its probable position. In one of these animals (SN-2)—the one nigral rat that failed to self-stimulate—all fluorescent DA cells were located > 1 mm from the electrode tip; in the other rat (SN-1)—which manifested moderate ICSS—fluorescent DA cells were close to the electrode tip. Thus in all five of the nigral animals that self-stimulated, the electrode tip was positioned close to DA cells or outgrowing DA fibers (Fig. 1).

In the three surviving caudate rats small grafts with very few or no DA neurons survived, adhering to the cavity walls, and the caudate-putamen was de-

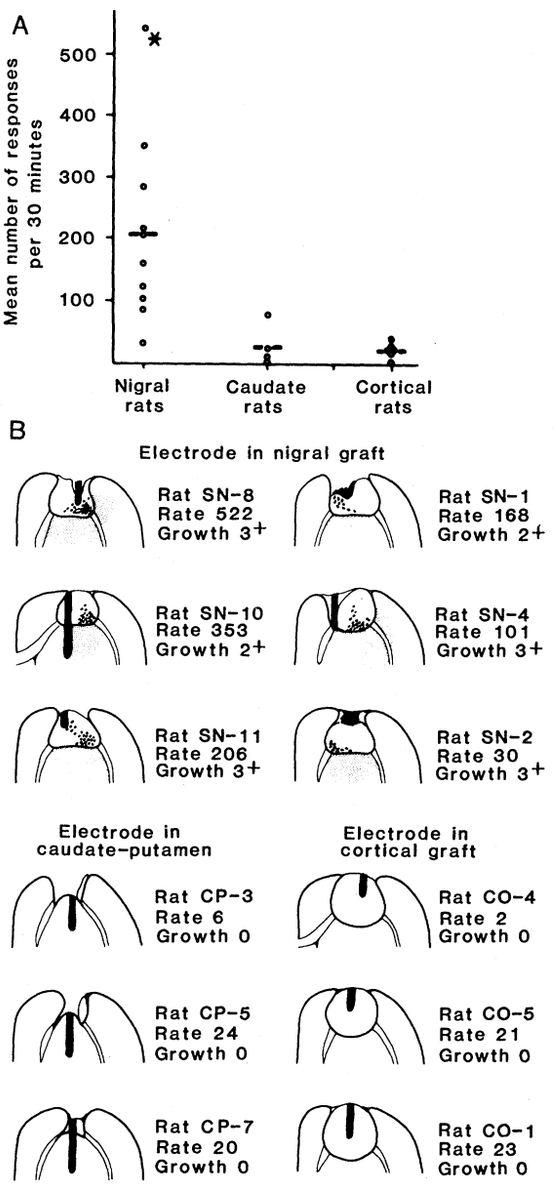


Fig. 1. Intracranial self-stimulation by rats with electrodes implanted into nigral grafts, into the dorsal caudate-putamen, or into cortical grafts. All animals had a unilateral 6-OHDA lesion of the nigrostriatal pathway on the same side as the electrode (and the graft). Tests were conducted at a constant current intensity (100 μA) after 2 weeks of initial training. (A) Mean response rate for each rat, averaged over three consecutive days. Asterisk indicates result for rat SN-8 (Fig. 2A). (B) Schematic diagrams of grafts, showing electrode placement and DA cell reinnervation for each rat killed for fluorescence histochemistry. The rate of self-stimulation (over 30 minutes at 100 μA) is indicated beside each diagram, together with a rating of the extent of DA cell ingrowth from the graft (*16*): 0, no ingrowth; 2+, one-eighth to one-fifth of the head of the caudate-putamen; 3+, one-fourth or more of the head of the caudate-putamen. Stippled areas, DA cell bodies; shaded areas, DA axons and terminals.

void of fluorescence. The electrodes were well-positioned in the dorsal neostriatum. Three cortical rats had large surviving grafts with well-positioned electrodes, and, as in the caudate rats, the caudate-putamen was devoid of fluorescence (Fig. 1).

The results indicate that nigral grafts reinnervating the caudate-putamen can sustain ICSS, provided the electrode is positioned adjacent to DA neurons. Rats in which the electrode was distal to surviving DA neurons, rats in which the nigral graft failed to provide dopaminergic reinnervation of the caudate-putamen, and rats that received grafts not containing DA neurons failed to exhibit ICSS. Therefore the presence of DA neurons appears to allow the reinnervated striatum to be activated in such a way as to support ICSS, suggesting that this DA pathway carries information axonally to the striatum and activates it in synchrony with each stimulation train.

It could be argued that the DA cells tonically release DA into the striatum, rendering it receptive to the stimulation, which spreads radially through the transplanted tissue to that part of the striatum receiving the dopaminergic innervation. However, this seems unlikely in view of the fact that the one nigral rat that failed to self-stimulate did have extensive dopaminergic reinnervation of the striatum, but the electrode was positioned distal to the DA cells in the graft. The effect of dopaminergic drugs on ICSS rates is compatible with our interpretation, although by itself the drug data cannot be used to distinguish specific from nonspecific mechanisms. ICSS can be obtained from the substantia nigra in the normal rat (*4*, *14*), but there has been some controversy over the relative involvement of DA neurons and of ascending and descending fibers of passage (*15*). The present results suggest that DA neurons isolated from their normal location are sufficient to sustain ICSS.

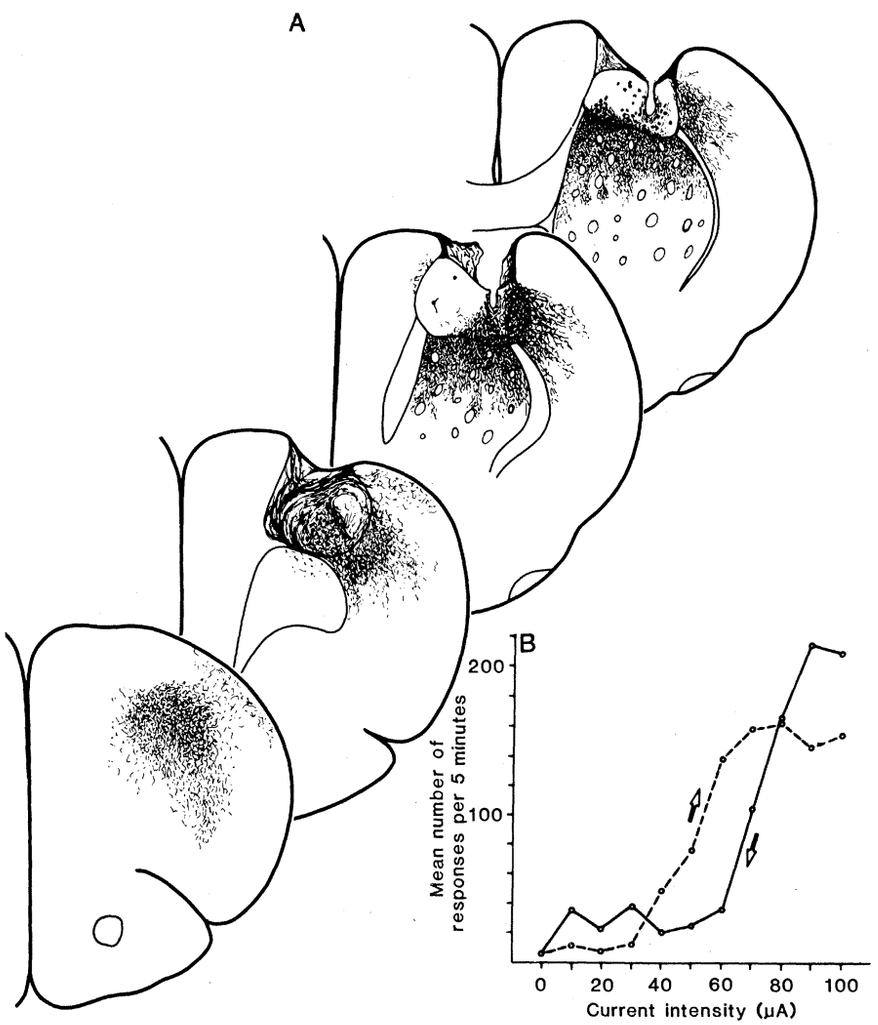


Fig. 2. (A) Camera lucida drawings showing a nigral graft providing dopaminergic reinnervation of the dorsal caudate-putamen. The animal involved, rat SN-8, had the highest ICSS rate of all the rats tested (Fig. 1A). The electrode tip is positioned adjacent to a large cluster of DA cell bodies in the center of the graft. (B) Rate-intensity function for rat SN-8. Each descending current intensity (solid line) and each ascending intensity (dashed line) was tested for 5 minutes during the test session and averaged over three sessions on consecutive days.


PAUL J. FRAY

*Department of Anatomy, University of Cambridge, Cambridge, United Kingdom CB2 3DY*

STEPHEN B. DUNNETT

SUSAN D. IVERSEN

*Department of Experimental Psychology, University of Cambridge*

ANDERS BJÖRKLUND

ULF STENEVI

*Department of Histology, University of Lund, S-223 62 Lund, Sweden*


**References and Notes**


1. A. Björklund and U. Stenevi, *Brain Res.* **177**, 555 (1979); U. Stenevi, A. Björklund, S. B. Dunnett, *Peptides* **1**, 111 (1980).
2. A. Björklund, U. Stenevi, S. B. Dunnett, S. D. Iversen, *Nature (London)* **289**, 497 (1981); S. B. Dunnett, A. Björklund, U. Stenevi, S. D. Iversen, *Brain Res.* **215**, 147 (1981); S. B. Dunnett *et al.*, *ibid.* **229**, 209 (1981).
3. J. Olds and P. Milner, *J. Comp. Physiol. Psychol.* **47**, 419 (1954).
4. T. J. Crow, *Psychol. Med.* **2**, 414 (1972); *Brain Res.* **36**, 265 (1972); D. C. German and D. M. Bowden, *ibid.* **73**, 381 (1974); A. G. Phillips, D. A. Carter, H. C. Fibiger, *ibid.* **104**, 221 (1976); B. R. Cooper, R. J. Konkol, G. R. Breese, *J. Pharmacol. Exp. Ther.* **204**, 592 (1978); G. F. Koob, P. J. Fray, S. D. Iversen, *Brain Res.* **146**, 123 (1978); D. Corbett and R. A. Wise, *ibid.* **185**, 1 (1980).
5. All surgery was conducted under Brietal anesthesia. Lesions were made by stereotaxic injection of 4 μg of 6-OHDA in 1 μl of ascorbate solution (0.2 mg/ml) into the rostral medial substantia nigra, where the ascending nigrostriatal DA fibers converge. All rats manifested strong ipsilateral rotation (more than seven turns per minute over 1 hour) in response to amphetamine, indicating > 98 percent DA cell denervation in the striatum (*2*). The cortical cavities were made by aspiration through the dorsal parietal cortex and corpus callosum to expose the dorsal surface of the caudate-putamen. The cavities measured approximately 3 by 4 mm and were filled with Gelfoam until subsequent transplantation. The grafts were dissected from the ventral mesencephalon of 16- to 17-day rat fetuses of the same inbred strain as the hosts and placed immediately into the cavities, which were again plugged with Gelfoam (*1*, *2*).
6. Medium-gauge, bipolar, insulated stimulating electrodes of stainless steel (Plastic Products) were implanted into the grafts (or into the dorsal caudate-putamen) under visual guidance and anchored to the skull with dental cement and stainless steel screws.
7. All behavioral testing was conducted in operant chambers with a single large response lever along one wall. A flexible cable connected the rat electrode to a commutator above the center of the chamber so that the animal could move freely. Each press of the lever resulted in the delivery of a single 300-msec train of 50-Hz sine wave stimulation at a constant current intensity.
8. S. Kullback, *Information Theory and Statistics* (Dover, New York, 1968).
9. Rat SN-8 received three daily 105-minute test sessions, on each of which the current intensity was reset every 5 minutes in descending and ascending series of 10-μA steps over the range 0 to 100 μA. The response rates for each current intensity were averaged separately for the descending and ascending series.
10. G. F. Koob, *Physiol. Behav.* **18**, 131 (1977).
11. L. Stein, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **23**, 836 (1964); M. E. Olds, *Neuropharmacology* **9**, 519 (1970).
12. The five doses of amphetamine (0, 0.125, 0.25, 0.5, and 1.0 mg/kg), dissolved in 0.9 percent saline (1 ml/kg), were administered in a fully balanced order over 15 days. The injection was administered intraperitoneally 15 minutes before the daily test session. Two days without injec-

tions separated each drug test, and the mean response rate over the ten intervening days provided baseline scores against which each animal's response to the drug was compared. The neuroleptic response was tested similarly over a further 18 days. α-flupenthixol (0, 0.1, or 0.3 mg/kg) and amphetamine (0 or 0.5 mg/kg) were injected 45 and 15 minutes, respectively, before the daily test session in a balanced sequence. During this second series of drug tests two animals became sick; they are not included in the data.
13. I. Lorén, A. Björklund, B. Falck, O. Lindvall, *J. Neurosci. Methods* **2**, 277 (1980).
14. A. Routtenberg and C. M. Malsbury, *J. Comp. Physiol. Psychol.* **68**, 22 (1969); Y. H. Huang and A. Routtenberg, *Physiol. Behav.* **7**, 419 (1971).
15. D. C. German and F. A. Holloway, *Science* **179**, 1345 (1973); J. D. Belluzzi, S. Ritter, C. D. Wise, L. Stein, *Behav. Biol.* **13**, 103 (1975); R. M. Clavier and A. Routtenberg, *Brain Res.* **105**, 325 (1976).
16. A. Björklund, S. B. Dunnett, U. Stenevi, M. E. Lewis, S. D. Iversen, *Brain Res.* **199**, 303 (1980).
17. Supported by grants from the Swedish Medical Research Council, the Syskonen Svensson Foundation, and the European Training Programme in Brain and Behaviour Research. S.B.D. and P.J.F. are supported by the Medical Research Council in Britain. We thank T. W. Robbins for helpful discussions and U. Jarl and G. Stridsberg for technical assistance.

8 January 1982; revised 28 July 1982

## Recruitment and Population Dynamics of a Coral Reef Fish

Abstract. *Daily otolith increments were used to determine the daily pattern of settlement of the bluehead wrasse (*Thalassoma bifasciatum*), a Caribbean coral reef fish. Recruitment occurs in brief and sporadic episodes even though bluehead wrasses spawn every day. Patterns of recruitment do not correspond to patterns of mortality on the reef. The composition of the adult population directly reflects the relative rates of recruitment of juveniles the year before. The population dynamics of this species may therefore be determined by the supply of recruits and not by the supply of space or some other resource on the reef.*

Virtually all coral reef fishes settle onto the reef after spending some time as planktonic larvae (*1*). This process of recruitment is one of the most important and yet least studied aspects of reef fish ecology. Recently, some descriptions of settlement patterns have been reported (*2*). However, many questions are unanswered, such as what determines patterns of recruitment and what impact these patterns have on adult populations. I report that recruitment in the bluehead wrasse, *Thalassoma bifasciatum*, occurs in brief and sporadic episodes that are not related either to patterns of reproduction or to mortality on the reef. Furthermore, I show that these brief episodes of recruitment can have a profound impact on adult populations in the future. These findings are incompatible with the view that reef fish communities are stable and primarily limited by resources (*3*).

It has been suggested that recruitment patterns of coral reef fishes closely parallel spawning patterns and are thus predictable (*4*). This may be true for species with infrequent and sharply defined spawning bouts, because outside of spawning periods, eggs are not being produced and larvae are not available for settlement. However, how much of the variability in recruitment is due to reproductive patterns and how much to other processes can be elucidated by examining a species that spawns every day. Daily spawning is a common phenomenon among coral reef fishes, particularly in the wrasses (Labridae), parrotfishes (Scaridae), and the basses (Serranidae) (*5*). I examined recruitment of the bluehead wrasse on coral reefs in the San Blas Archipelago, on the Caribbean coast of Panama, where the daily spawning behavior of the species has been monitored for many years.

I used the daily otolith increment technique (*6*) to calculate the pattern of settlement. Since there are daily lines and a prominent transition mark corresponding to settlement on the otoliths of the bluehead wrasse (*7*), the settlement pattern of populations of fish, including adults, can be easily reconstructed. The date of settlement of each individual is obtained by subtracting the number of lines after the settlement mark from the date of capture. For this study I collected 103 juveniles from a large patch reef near the island of Porvenir in late 1980.

Recruitment of bluehead wrasses occurred in brief and sporadic episodes (Fig. 1) (*8*) and did not reflect the daily spawning pattern of this species. It has been reported that reef fishes are limited by the availability of resources such as space and that recruits settle from a pool of superabundant larvae into spaces made available by the death of residents (*3*, *9*). According to these views, the brief episodes of settlement of bluehead wrasses would be in response to sudden die-offs among the reef population. Mortality rates of bluehead wrasses on patch reefs around the study area were monitored (*10*). To prevent recruitment, newly settled fish were continuously removed; at the same time changes in the adult population were monitored by monthly censuses. Populations declined somewhat steadily (86.5 percent survival per month; standard deviation, 6.7 percent; $N = 8$ censuses). During the monitoring period occasional large peaks of settlement occurred on these and surrounding reefs without corresponding changes in the mortality rate. It is likely that some process occurring in the plank-

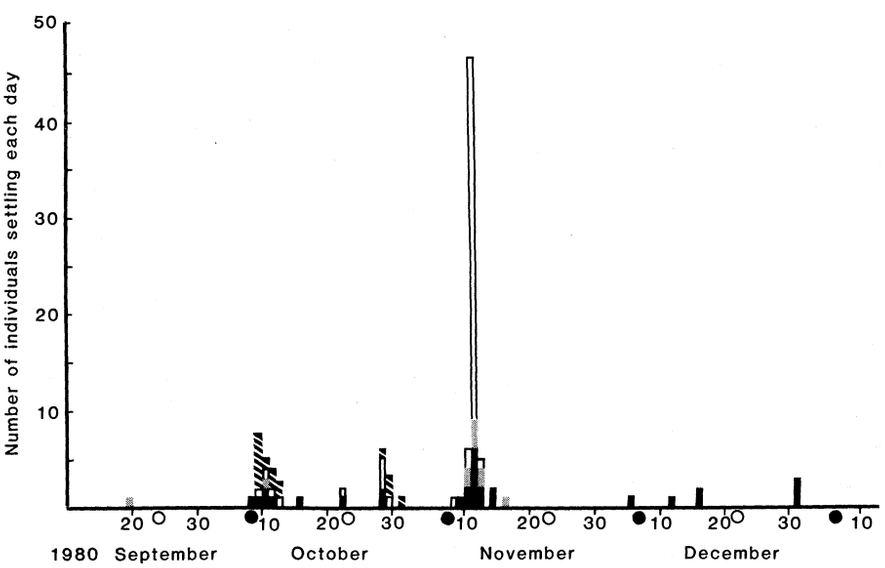


Fig. 1. The recruitment of juvenile *Thalassoma bifasciatum* onto a single patch reef in the San Blas Islands, Panama. Bars represent the number of individuals that settled each day, calculated by subtracting the age since settlement from the date of capture for each individual. Hatched bars represent fish caught in early November 1980, open bars those caught the third week of November, shaded bars those caught in late December, and solid bars those caught in early January. Solid circles denote new moon and open circles, full moon.