Our field evidence for rapid onset of lacustrine deposition after impact (*24*) and the relatively rapid rates of accumulation recorded for such sediments (*25*) allow us to fix the date for the Haughton fossil assemblages well within the limit of error for our fission-track values. This provides an invaluable datum point for the hitherto poorly dated Neogene plant assemblages of the Arctic that will be of considerable importance for understanding paleogeography and biostratigraphy of the region during that time.

REFERENCES AND NOTES

1. T. Frisch and R. Thorsteinsson, *Arctic* **31**, 108 (1978).
2. P. B. Robertson and G. D. Mason, *Nature (London)* **255**, 393 (1975).
3. P. B. Robertson and J. F. Sweeney, *Can. J. Earth Sci.* **20**, 1134 (1983).
4. American Geological Institute, *Dictionary of Geological Terms* (Anchor/Doubleday, Garden City, NY, 1976).
5. P. B. Robertson *et al.*, *Proc. Lunar Planet Sci. Conf. 16th* (1985), p. 702.
6. M. R. Dawson *et al.*, *Can. Paleontol. Biostratigr. Sem. Quebec* (1985), p. 8.
7. L. V. Hills, in *Proceedings of the Circumpolar Conference on Northern Ecology*, (National Research Council Canada, Ottawa, 1975), p. I-65.
8. J. V. Matthews, *Geol. Surv. Can. Pap.* **87-1A**, 73 (1987).
9. C. W. Naeser, *U.S. Geol. Surv. Open-file Rep. 76-190* (1978).
10. B. S. Carpenter, *Natl. Bur. Stan. Spec. Publ. 260-292* (1984).
11. M. J. Kunk, J. F. Sutter, C. W. Naeser, *Geol. Soc. Am. Abstr. Programs* **17**, 636 (1985).
12. Dates were calculated from the age equation of R. L. Fleischer, P. B. Price, and R. M. Walker [*Nuclear Tracks in Solids: Principles and Applications* (Univ. of California Press, Berkeley, 1975)]. The constants used for age calculations are: $\lambda_D = 1.551 \times 10^{-10}$ year^{-1}, $^{235}U/^{238}U = 7.252 \times 10^{-3}$; and $\sigma^{235} = 580.2 \times 10^{-24}$ cm^2.
13. P. F. Green, *Nucl. Tracks* **5**, 77 (1981).
14. D. Storzer and G. A. Wagner, *Meteoritics* **12**, 368 (1977).
15. R. L. Fleischer, R. T. Wood, H. R. Hart, Jr., P. B. Price, N. M. Short, *J. Geophys. Res.* **79**, 339 (1974).
16. D. S. Miller and G. A. Wagner, *Earth Planet. Sci. Lett.* **43**, 351 (1979).
17. D. Stöffler, *J. Geophys. Res.* **76**, 5541 (1971).
18. C. W. Naeser, *Nucl. Tracks* **5**, 248 (1981).
19. A. J. W. Gleadow and I. R. Duddy, *ibid.*, p. 169.
20. A. J. W. Gleadow, I. R. Duddy, J. F. Lovering, *Aust. Pet. Explor. Assoc. J.* **23**, 93 (1983).
21. K. D. Crowley, C. W. Naeser, C. A. Babel, *Earth Planet Sci. Lett.* **79**, 329 (1986).
22. R. K. Dokka *et al.*, *Tectonics* **5**, 995 (1986).
23. C. W. Naeser, R. A. Zimmerman, G. T. Cebula, *Nucl. Tracks* **5**, 65 (1981).
24. This evidence includes the following features of the lacustrine sediments: a reverse-graded debris flow in the basal 2 m; the general lack of reworking or of primary depositional features; and the gradational contact with the underlying ejecta blanket.
25. At minimum rates of lacustrine sedimentation [D. E. Schindel, *Paleobiology* **6**, 425 (1980)] the maximum time to accumulate the 48 m of these sediments is 4.8×10^5 years.

26. We gratefully acknowledge comments by T. Lutz, R. Shagam, and R. Giegengack; S. Carpenter irradiated the samples; and A. Kelly and B. Tiffney identified the fruit and seed remains. Funding for fieldwork was provided by the National Geographic Society and the Polar Continental Shelf Project, Department of Energy, Mines and Resources of the Canadian Government. This is contribution 15287 of the Geological Survey of Canada.

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An in Vitro Neurite-Promoting Antigen Functions in Axonal Regeneration in Vivo

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The function of the neurite growth–promoting antigen INO has been tested in an in vivo neurite regeneration system, the rat iris. The sympathetic innervation of the irides was removed by a single systemic injection of 6-hydroxydopamine. The subsequent regeneration of sympathetic axons into the iris of one eye bathed by the INO antibody, which inhibits neurite growth in vitro, was compared with the regrowth of sympathetic axons into the iris of the animal's other eye, which contained control antibody. Antibodies were released within the eye by implanted hybridoma cells. Neurite regeneration was measured by assaying [^{3}H]norepinephrine uptake into freshly explanted irides. The blockage of the function of the INO antigen by the antibody resulted in a decreased rate of axonal regeneration, thus suggesting the involvement of the INO antigen in the process of neurite regeneration in vivo.

IN THIS REPORT WE PRESENT EXPERIMENTAL evidence for the role of the neurite growth–promoting INO antigen in the regeneration of sympathetic nerve fibers in vivo. The INO antibody, which was generated to the neurite promoting activity found in non-neuronal cell conditioned media, recognizes a laminin–heparin sulfate proteoglycan complex (*1*) and inhibits the in vitro neurite growth of sympathetic neurons over cryostat sections of sciatic nerve (*2*). To test the possible function of the INO antigen in vivo, regenerating axons were exposed to the INO antibody by injecting hybridoma cells into the anterior chamber of rat eyes. Treatment with 6-hydroxydopamine (6-OHDA) caused degeneration of sympathetic terminals in the irides; subsequent regeneration, in the presence of three different hybridomas that secreted immunoglobulin M (IgM) monoclonal antibodies, was assayed by explanting irides into culture media containing radiolabeled norepinephrine.

The iris is innervated by sympathetic fibers from the superior cervical ganglion, by parasympathetic fibers from the ciliary ganglion, and by sensory fibers from the trigeminal ganglion (*3*). The axons, in association with Schwann cells (*4*), enter the iris at the ciliary margin, course radially toward the pupil in the stroma, forming a "ground plexus," which lacks Schwann cells (*4*). The sympathetic innervation of the iris can be selectively lesioned by systemic administration of high doses of 6-OHDA, a catecholamine congener, which causes the degeneration of preterminal and terminal axons (*5–7*). Because neuronal cell bodies survive 6-OHDA administration in adults (*6*), sympathetic axons begin to regenerate soon after this chemical sympathectomy; complete return of the sympathetic ground plexus to peripheral organs is usually achieved after about 2 months (*8*). The regeneration of sympathetic nerve fibers has been monitored by the return of norepinephrine content (*7*) and high-affinity norepinephrine uptake (*9*) in peripheral organs; the recovery of these biochemical properties parallels the density of catecholamine histofluorescent axons.

The iris separates the anterior and posterior chambers of the eye and is bathed in aqueous humor. A "blood-aqueous barrier" limits exchange of materials between the blood and the aqueous humor of the eye (*10*) and at least partially protects the chambers from immune surveillance (*11*). Mouse hybridoma cells can grow in the anterior chamber and provide a continuous, long-

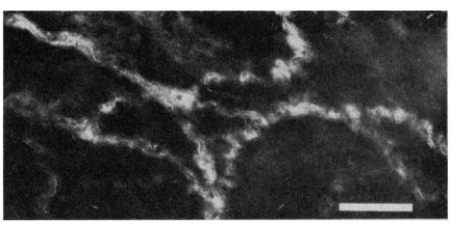

Fig. 1. Localization of the INO antigen in an iris whole mount. Irides were removed from killed animals and placed in 2% paraformaldehyde in 0.1*M* phosphate buffer, *p*H 7.4. After a 15-minute incubation at room temperature, the fixative was removed with three 10-minute rinses of L15 medium containing 10% fetal calf serum. The irides were incubated in 3 ml of hybridoma supernatant for 24 hours at 4°C. After three 1-hour rinses with PBS at room temperature, the irides were rinsed for an additional 24 hours with PBS at 4°C. The irides were incubated in 2 ml of fluorescein-conjugated goat antiserum to mouse immunoglobulins (1:50 dilution in PBS containing 10% rat serum; Antibodies, Inc.). After a 4-hour incubation at room temperature, the irides were rinsed with three 1-hour rinses of PBS at room temperature, gently stretched (stroma side up) on a glass slide and mounted in a 50:50 (v/v) mixture of glycerol and *p*-phenylenediamine (1 mg/ml) in 0.1*M* carbonate buffer, *p*H 8.3. Scale bar, 50 μm.

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term source of monoclonal antibody to the aqueous humor. Thus, the sympathetic innervation of the iris provides an excellent system to examine the role of the INO antigen in promoting regeneration.

In a whole mount of iris the INO antibody stains a tortuous network in the stromal layer (Fig. 1). This network is distinct from blood vessels containing erythrocytes and resembles nerve fiber bundles, which course through the stroma. Iris whole mounts were also stained with two other IgM monoclonal antibodies, RN1A2 and RN3B3, which were used as controls for subsequent experiments. The RN1A2 antibody recognizes an antigen in sciatic nerve but has no effect on neurite growth in the in vitro bioassay. It stains material in the iris stroma in a pattern indistinguishable from that obtained with the antibody INO. Antibody RN3B3, which binds to sections of the distal stump of transected sciatic nerve and inhibits neurite growth over such sections in vitro (*12*), does not show detectable staining in iris whole mounts from normal or 6-OHDA–treated rats.

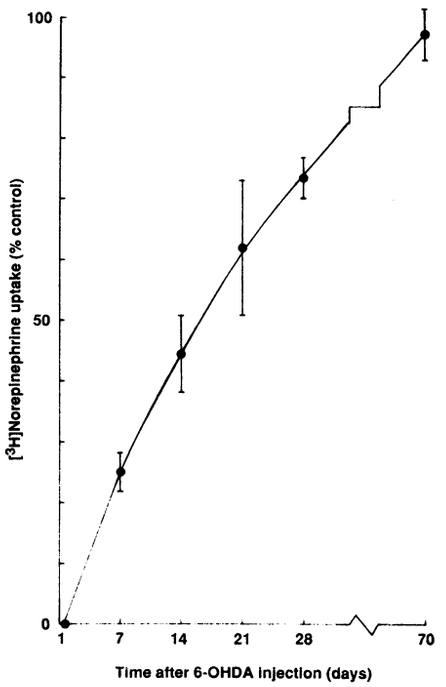

Fig. 2. The recovery of norepinephrine uptake by whole rat irides after 6-OHDA treatment. Each rat was injected intraperitoneally with 6-OHDA · HCl (100 mg/kg) freshly dissolved in 0.05% ascorbic acid, 0.9% NaCl. Irides were removed at various times after sympathectomy and placed in 200 μl of ice-cold L15 medium supplemented with 0.6% (w/v) glucose, 2 m*M* glutamine, penicillin (100 U/ml), and streptomycin (100 U/ml). The uptake assay was started by transferring each iris into L15-CO_2 medium containing 0.1 μ*M* [^{3}H]norepinephrine (44.4 Ci/m*M*, NEN) supplemented as above, plus L-ascorbic acid (10 mg/ml) in a humidified, 5% CO_2, 37°C incubator. Since the high affinity uptake pump of the axolemma is nearly completely blocked at 0°C (*13*), background uptake values into extraneuronal tissues were obtained by transferring irides into the wells of a different microtiter plate containing identical solutions maintained at 0°C. After 30 minutes (*16*) the assay was stopped by transferring the irides into wells containing 2 ml of 10^{-5}*M* unlabeled norepinephrine in ice-cold L15 medium. After a 10-minute incubation, the irides were rinsed with two changes of 2 ml of ice-cold L15 medium. Each iris was gently blotted on filter paper, transferred to a scintillation vial containing 1 ml of 1% SDS in 1 m*M* HCl, frozen, and thawed. After the addition of 10 ml of Ultrafluor (National Diagnostics) to each vial, radioactivity was measured for 1 minute in a liquid scintillation counter.

Irideal norepinephrine uptake was measured at various times after chemical sympathectomy to assess the extent of axon depletion after 6-OHDA treatment and to estimate the rate of subsequent regeneration (Fig. 2). The norepinephrine uptake in the irides of animals treated with 6-OHDA 24 hours earlier is indistinguishable from that in normal irides incubated at 0°C; the high affinity uptake system does not function at 0°C (*13*). Therefore, at the dose injected (100 mg/kg), 6-OHDA appears to remove the entire sympathetic innervation of the iris. Over the subsequent 10-week period, irideal norepinephrine uptake gradually returns to control values, reaching half-maximal values at approximately 2.5 weeks.

Sympathetic fibers regenerating within the iris were examined histologically by

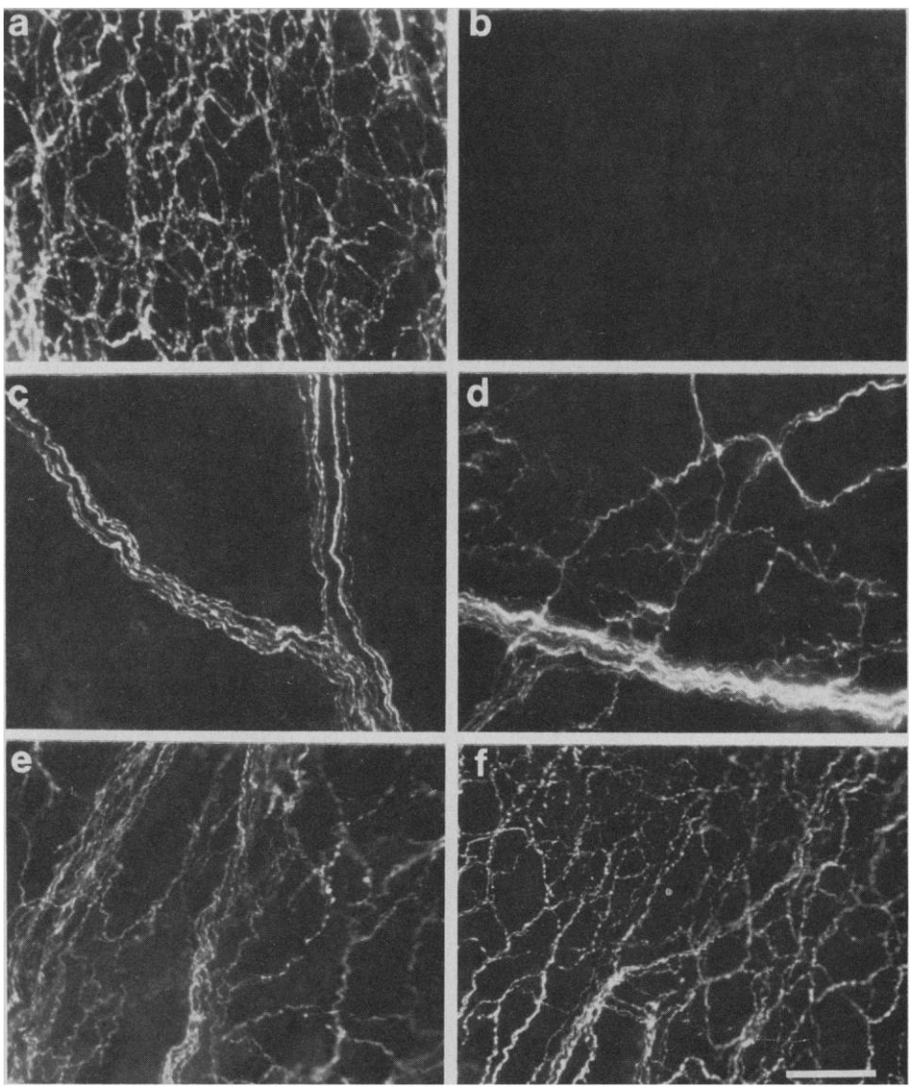

Fig. 3. Histochemical localization of irideal sympathetic nerve fibers. Animals were killed and enucleated. The eyes were placed in ice-cold Dulbecco's PBS where the irides were removed under a dissecting microscope. Each iris was gently stretched on a clean glass microscope slide with the anterior (stromal) side up and allowed to partially dry in room air for about 5 minutes. The iris whole mount was then treated with the glyoxylic acid solution of de la Torre and Surgeon (*17*) for 5 seconds and immediately placed under a hair dryer. After the iris had been rapidly and thoroughly dried, the slides were mounted in mineral oil, heated at 95°C for 2 to 3 minutes, and examined in a Zeiss fluorescence microscope equipped with epifluorescence and catecholamine filter. Photographs are of glyoxylic acid–induced catecholamine histofluorescence in (**a**) normal iris, and in irides of rats (**b**) 1 day, (**c**) 7 days, (**d**) 14 days, (**e**) 21 days, and (**f**) 70 days after systemic 6-OHDA treatment. Scale bar, 50 μm.

glyoxylic acid–induced catecholamine histofluorescence (Fig. 3). In agreement with the biochemical data and in contrast to normal irides (Fig. 3a), no fluorescent fibers were observed in the irides of animals that had been sympathectomized 24 hours before (Fig. 3b). At 1 week after sympathectomy, regenerating fluorescent fibers were visible in the dilator region near the ciliary margin of the iris (Fig. 3c). These fibers were in the anterior stromal layer of the iris and were consistently observed in bundles of two to ten fibers. As the sympathetic reinnervation of the iris proceeded, the regenerating sympathetic axons tended to grow radially toward the pupil. As the reinnervation of the iris continued, individual fibers left the bundles and arborized (Fig. 3, d and e), eventually reforming the ground plexus. By week 10 (Fig. 3f), the sympathetic plexus was indistinguishable from that in normal, undamaged irides (Fig. 3a), and the axonal bundling that was so prominent at earlier time points was difficult to discern.

Hybridoma cells secreting the in vitro neurite growth–inhibiting antibody INO and antibody RN3B3, as well as those secreting the control antibody RN1A2, were injected into anterior chambers to compare the effect of a function-blocking antibody (in one eye) with that of a control antibody (in the other eye of the same animal). One week later, rats with undamaged eyes that contained clumps of hybridoma cells in the anterior chamber were chemically sympathectomized, and sympathetic regeneration in the iris of each eye was estimated by norepinephrine uptake at 5, 10, and 20 days after sympathectomy. Hybridoma cells secreting the INO antibody caused a substantial reduction in the regeneration of sympathetic axons into the iris; the effect was most pronounced at time intervals soon after sympathectomy—5 and 10 days after sympathectomy (Fig. 4). In contrast, hybridoma cells secreting the RN3B3 antibody had no effect on the rate of sympathetic nerve regeneration at any time.

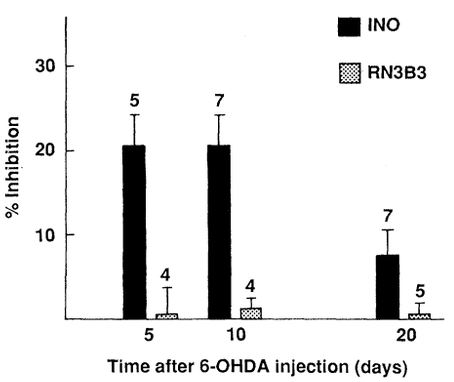

Fig. 4. Histograms of the percent reduction in the recovery of norepinephrine uptake by whole irides at 5, 10, and 20 days after chemical sympathectomy, in the presence of INO, RN3B3, and RN1A2 (control) hybridoma cells in oculo. Hybridoma cells were grown and implanted as described in (*18*). [^{3}H]Norepinephrine uptake into whole irides was measured at various times after systemic 6-OHDA administration. The percent reduction of norepinephrine uptake by the iris of the eye that had contained experimental hybridoma (INO and RN3B3) cells, in comparison to the uptake by the iris of the contralateral eye, which had contained control hybridoma (RN1A2) cells, was calculated for each animal; the values given are means (+ SEM) derived from the number of animals in each group at the top of each histogram bar.

To determine whether the INO antibody had exerted its effects by actually penetrating the stroma and binding to antigens that might normally contact the nerve fiber bundles, irides from animals that had received in oculo injections of INO hybridoma cells 10 days earlier were rinsed thoroughly with phosphate-buffered saline (PBS), and fixed and stained with fluorescein-conjugated goat antiserum to mouse immunoglobulins. These irides displayed the characteristic network in the stroma, indistinguishable from the INO staining pattern in iris whole mounts after standard immunocytochemical procedures (Fig. 1).

The RN3B3 antibody inhibits neurite growth in vitro; however, it did not inhibit sympathetic fiber regeneration in the iris. This antibody, unlike the INO and RN1A2 antibodies, stains a network in the stroma of the iris only after this tissue has been cultured for 4 days; it did not stain normal irides or irides from animals treated with 6-OHDA. Presumably, the total denervation that results from the removal of the iris from the animal causes the induction of this antigen. It is possible that more elaborate manipulations, such as the surgical removal of the sensory or parasympathetic innervations, performed at or before the time of sympathectomy, would reveal a detectable effect of this antibody on irideal sympathetic nerve fiber regeneration. Because the RN3B3 antigen cannot be detected in sympathectomized animals, it is not surprising that this monoclonal antibody could not perturb axonal regeneration under the conditions tested.

The INO antibody is most effective at delaying the reinnervation of the iris at early times after sympathectomy and is less effective at day 20. This correlates with the initial phase of regeneration, when bundled axons grow within the stroma (Fig. 3, c and d), as opposed to later times when the ground plexus is being formed (Fig. 3, e and f). One interpretation is that the INO antigen, which is restricted to a network within the stromal layer of the iris (Fig. 1), promotes the regeneration of fiber bundles within the stroma at early times after sympathectomy, whereas other factors promote arborization at later times.

One important question remains. Are the "pathways" followed by fascicles of regenerating sympathetic fibers the same as those that are stained by the INO antibody? It is unfortunate that the technique of catecholamine histofluorescence precludes concurrent immunohistochemical staining procedures; however, dual-label histological procedures with antisera against one of the catecholamine biosynthetic enzymes and the INO antibody would directly answer this question. The facts that the pathways (Fig. 3c) are similar in appearance and distribution to the profiles stained by the INO antibody (Fig. 1) and that neither coincides with blood vessels indicate that the pathways and the antibody staining patterns are probably the same.

The network stained by the INO antibody resembles the structures in cultured rat irides that are labeled with antisera to nerve growth factor (NGF) (*14*). It has been proposed that the NGF-like immunoreactivity resides in denervated Schwann cells, which increase their production of NGF in irides after they have been in culture for several days. NGF can serve to promote neurite growth as either a soluble or substrate-bound factor (*15*). Our findings suggest that regenerating sympathetic axons follow well-defined pathways through the irideal stroma because of the presence of substrate-attached molecules, such as those recognized by the INO antibody. In addition, axons might be guided by concentrations of other neurite growth–promoting factors or by trophic factors such as NGF.

REFERENCES AND NOTES

1. W. D. Matthew and P. H. Patterson, *Cold Spring Harbor Symp. Quant. Biol.* **48**, 625 (1983); A. Y. Chiu, W. D. Matthew, P. H. Patterson, *J. Cell Biol.* **103**, 1383 (1986).
2. A. W. Sandrock and W. D. Matthew, *Soc. Neurosci. Abstr.* **11**, 592 (1985); *Proc. Natl. Acad. Sci. U.S.A.*, in press.
3. B. Falck *et al.*, *J. Histochem. Cytochem.* **10**, 348 (1962); A. Huhtala, T. Tervo, A. Palkama, *Acta Ophthalmol. Suppl.* **125**, 47 (1975); A. Miller, M. Costa, J. B. Furness, I. W. Chubb, *Neurosci. Lett.* **23**, 243 (1981); C. S. Ayer-le Lievre, A.-Ch. Granholm, A. Sieger, *J. Anat.* **138**, 309 (1984).
4. K. C. Richardson, *Am. J. Anat.* **114**, 173 (1964).
5. C. C. Porter, J. A. Totaro, C. A. Stone, *J. Pharmacol. Exp. Ther.* **140**, 308 (1963); C. R. Creveling, J. W. Daly, B. Witkop, *ibid.* **158**, 46 (1967); L. L. Iversen, *Br. J. Pharmacol. Chemother.* **25**, 18 (1965); H. Thoenen and J. P. Tranzer, *Arch. Pharmakol. Exp. Pathol.* **261**, 271 (1968).
6. R. Laverty, D. F. Sharman, M. Vogt, *Br. J. Pharmacol. Chemother.* **24**, 549 (1965).
7. J. de Champlain, *Can. J. Physiol. Pharmacol.* **49**, 345 (1971).
8. G. Haeusler, W. Haefely, H. Thoenen, *J. Pharmacol. Exp. Ther.* **170**, 50 (1969); G. Jonsson and C. Sachs, *Eur. J. Pharmacol.* **9**, 141 (1970).
9. G. Jonsson and C. Sachs, *J. Neurochem.* **19**, 2577 (1972).
10. T. Vegge, *Zeit. Zellforsch. Mikrosk. Anat.* **121**, 74 (1971).
11. L. Olson and T. Malmfors, *Acta Physiol. Scand.*

Suppl. **348**, 1 (1970); R. Y. Moore, *Exp. Neurol.* **49**, 617 (1975); T. Ebendahl, L. Olson, A. Seiger, K.-O. Hedlund, *Nature (London)* **286**, 25 (1980).
12. W. D. Matthew and A. W. Sandrock, *Soc. Neurosci. Abstr.* **11**, 1254 (1985).
13. C. Sachs, *Acta Physiol. Scand. Suppl.* **341**, 1 (1970); in *6-OHDA and Catecholamine Neurons*, T. Malmfors and H. Thoenen, Eds. (North-Holland, Amsterdam, 1971).
14. P. J. Finn *et al.*, *J. Neurocytol.* **15**, 169 (1986); R. A. Murphy *et al.*, *Dev. Biol.* **114**, 369 (1986).
15. R. W. Gundersen, *J. Neurosci. Res.* **13**, 199 (1985); A. W. Sandrock, W. D. Matthew, *Brain Res.*, in press.
16. The level of norepinephrine uptake by explanted irides is an accurate measure of the density of its sympathetic innervation if it is assayed when the uptake rate is constant. Therefore, the rate of uptake of 0.1 μM [^{3}H]norepinephrine into irides was determined. The level of norepinephrine uptake was linear for incubation periods up to 40 minutes. Thus, we assayed irideal norepinephrine uptake during a 30-minute incubation period to maximize the signal while remaining within the linear portion of the curve.
17. J. C. de la Torre and J. W. Surgeon, *Histochemistry* **49**, 81 (1976).
18. Hybridomas were maintained in logarithmic growth in L15-CO_2 medium supplemented with 0.6% glucose, 2 mM glutamine, penicillin (100 U/ml), streptomycin (100 U/ml), and 10% fetal calf serum, until the time of transplantation. The cells were washed twice with serum-free L15 medium and resuspended in a small volume of serum-free medium (supplemented with the same concentrations of glucose, glutamine, and antibiotics) at a final concentration of 10^7 to 10^8 cells per milliliter. Male albino Sprague-Dawley rats weighing 175 to 225 g were each anesthetized with halothane U.S.P. (Halocarbon Labs.) and placed under a dissecting microscope. After a corneal reflex could no longer be elicited, a clean 10-μl glass syringe equipped with a 33-gauge needle (Hamilton) was used to puncture both corneas. For each eye, the extruded aqueous humor was wiped from each eye, and the same syringe was filled to capacity with a suspension of the appropriate hybridoma cells. Approximately 10^5 hybridomas were injected into the anterior chamber of one eye through the original puncture wound in about 5 μl, immediately followed by the injection of about 0.5 μl of air (the air bubble in the anterior chamber lodges in the puncture wound and prevents the cell suspension from leaking out of the eye). One eye received experimental hybridoma cells—those secreting a function-blocking monoclonal antibody (either INO or RN3B3)—while the contralateral eye received control hybridomas (RN1A2). One week after hybridoma injection, the rats were anesthetized with halothane and both eyes were examined under a dissecting microscope for signs of tissue damage, as well as for the presence of characteristic clumps of growing hybridoma cells. Only those rats with apparently normal eyes containing visible clumps of hybridoma cells in the anterior chambers of both eyes were retained for sympathectomy. Each rat was injected intraperitoneally with 6-OHDA · HCl (100 mg/kg, Sigma) freshly dissolved in 0.05% ascorbic acid, 0.9% NaCl. Only those rats that exhibited marked ptosis of the eyelids on the next day were considered adequately sympathectomized and retained for further study.
19. Expert assistance was provided by J. Gagliardi and M. LaFratta. We thank many colleagues for helpful discussions, especially E. Furshpan, S. Landis, P. Patterson, and D. Potter. Supported by research grant NS02253 from the National Institutes of Health, and aided by a McKnight Foundation Scholars Award, an Alfred P. Sloan Foundation Fellowship, and Basil O'Connor Starter Research Grant 5-523 from the March of Dimes Birth Defects Foundation. A.W.S. is the recipient of Public Health Service, National Research Service Award 2T 32 GM07753-07 from the National Institute of General Medical Sciences. In all cases, procedures performed on animals and the care of animals were in accordance with the guidelines of Harvard Medical School.

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Dinosaurs on the North Slope, Alaska: High Latitude, Latest Cretaceous Environments

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Abundant skeletal remains demonstrate that lambeosaurine hadrosaurid, tyrannosaurid, and troodontid dinosaurs lived on the Alaskan North Slope during late Campanian–early Maestrichtian time (about 66 to 76 million years ago) in a deltaic environment dominated by herbaceous vegetation. The high ground terrestrial plant community was a mild- to cold-temperate forest composed of coniferous and broad leaf trees. The high paleolatitude (about 70° to 85° North) implies extreme seasonal variation in solar insolation, temperature, and herbivore food supply. Great distances of migration to contemporaneous evergreen floras and the presence of both juvenile and adult hadrosaurs suggest that they remained at high latitudes year-round. This challenges the hypothesis that short-term periods of darkness and temperature decrease resulting from a bolide impact caused dinosaurian extinction.

CLOSELY RELATED LATE CRETACEOUS (about 66 to 76 million years before the present) terrestrial vertebrates found in mid-latitudes of North America and Asia suggest that Alaska was part of an intercontinental route of dispersal and supported diverse faunas (*1*). Until recently, dinosaurian footprints and skin impressions were the only published records of Alaskan Late Cretaceous terrestrial vertebrates (*2*). The first discovery of dinosaurian bones in Alaska was made in 1961 by the late R. L. Liscomb at a site on the Colville River. The significance of his discovery went unnoticed until 1984 when Liscomb's collection was sent to C. A. Repenning, who recognized that the collection documented an exceptionally high latitude occurrence of dinosaurs and forwarded the material to W. Langston, Jr., for further study (*3*).

Dinosaur-bearing sediments crop out in bluffs (Fig. 1) along the Colville River (*4, 5*) and are part of the type section of the upper Kogosukruk Tongue, Prince Creek Formation, upper Colville Group (*6*). This unit is composed of delta plain silts, channel sands, local stringers of coal, beds of tephra, and nearshore marine sands and silts (*7*). Skeletal remains of dinosaurs are preserved at six known stratigraphic levels and within three distinct fault blocks (Fig. 2). The precise stratigraphic relationships of strata between these fault blocks are not known (*8*).

The bone bed discovered by Liscomb ranges from 0.5 to 1.0 m in thickness. The disarticulated dinosaur bones are remarkably well preserved, and, except for post-depositional breakage, are not distorted. The bones are stained dark brown to brownish black, with rare permineralization. They are size-sorted, with the largest elements and densest concentration near the base of the bed. The matrix is a dark brown, carbonaceous siltstone containing scattered calcareous concretions; both lithologies contain large amounts of plant debris and roots, but rare fossil wood. Total organic carbon content of the siltstone is high and dominated by terrestrial carbon sources (*9*). The bone bed shows an abrupt lateral facies change northward into a lighter colored siltstone that contains no bones and has markedly less plant debris. Three other bone-bearing horizons consist of similar carbonaceous, dark brown siltstones with concretions, these three organic-rich beds and the Liscomb bone bed are thought to represent soils (*8*). The remaining two bone-bearing horizons are in sand and represent fluvial channel lag deposits (*8*). All of the bone beds are laterally discontinuous, ranging from 25 m to more than 100 m in horizontal extent (*8*).

Most identifiable vertebrate fossils are remains of lambeosaurine hadrosaurids (hollow-crested duck-bill dinosaurs). No skeletal elements diagnostic at the genus or species level have been recovered. Some bones are parts of the skeletons of individuals as large as an adult *Parasaurolophus* or *Corythosaurus* (about 9 to 10 m in length); others represent individuals one-half to one-third the size and show morphological traits of juvenile individuals. Carnivorous or scavenging dinosaurs are represented by isolated teeth of a tyrannosaurid and *Troodon*. In lower latitudes the known stratigraphic ranges of these dinosaurs fall within the Upper Cretaceous. To date, remains of other kinds of vertebrates have not been found; we have yet to determine whether the absence of

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