asia and North America. The primitive nature of *Miracinonyx* and the apparent relationship with *Puma* (which is restricted to the Western Hemisphere) suggest that cheetahs originated, not in Eurasia as previously believed (*17*), but in North America. It seems unlikely that *A. pardinensis* was ancestral to *Miracinonyx* because of the derived features that separate it from the American species. An extensive study of North American Hemphillian (early Ruscinian) felids is needed before this question can be resolved. Many of the American felids from this time are represented only by fragmentary fossils and remain largely undescribed (*2*).

The North American fossils share numerous derived characters with, and only with, the Old World felid genus *Acinonyx*, and referral to that genus is thereby warranted. Although now on the verge of extinction, *Acinonyx* has at various times been an important part of the faunas of Europe, Asia, Africa, and North America, and was once one of the most widely distributed of land mammals.

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Regrowth of Severed Axons in the Neonatal Central Nervous System: Establishment of Normal Connections

Abstract. *When pyramidal tract axons are cut in the adult hamster, fibers degenerate in both anterograde and retrograde directions from the lesion. If the same operation is performed on infant hamsters, however, there is massive regrowth of the severed axons via a new brainstem pathway to their appropriate terminal sites in the medulla and spinal cord. In contrast to previous studies, these results suggest that axons in the mammalian central nervous system damaged early in life may regenerate in a functionally useful way.*

Plasticity of axons in the mammalian central nervous system (CNS) often involves the sprouting of intact axons into a foreign territory that has been denervated by damage to its normal inputs (*1*). Removing target tissue early in development can also result in abnormal terminations of the axons that would normally innervate that tissue (*2*). By contrast, however, if fiber tracts of the CNS are cut directly, axons distal to the lesion will degenerate. Attempts to show subsequent regeneration of proximal axonal stumps across the lesion have generally met with little success (*3*), even when the experiments were performed on infant animals (*4*). Although damaged axons emit new collateral sprouts proximal to a cut, the sprouts, rather than reinnervating their normal synaptic territory, are usually abortive or form anomalous connections (*5*). We now present anatomical evidence that CNS axons severed in the infant hamster are capable of massive new growth extending for considerable distances to terminate in appropriate regions of the brain.

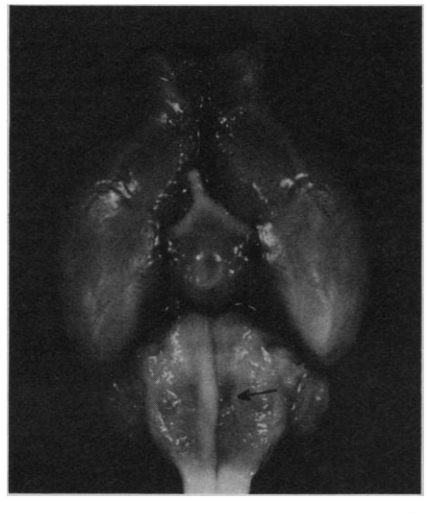

Fig. 1. Ventral view of adult hamster brain after a lesion (arrow) was placed in the animal's left pyramidal tract (on the right side of the photograph) at 5 days of age. Note the absence of pyramidal tract fibers below and ipsilateral to the lesion, in contrast to the normal fibers to the left of the midline.

To study the response of immature axons to injury, we chose the pyramidal tract. The fibers in this pathway are unidirectional, originating in the sensorimotor cortex (layer 5) and descending to terminal sites in the brainstem and spinal cord. The tract lies exposed on the ventral surface of the medulla before it enters the pyramidal decussation and crosses completely to the opposite side. Hence, the pyramidal fibers can be cut on one side by a ventral approach to the surface of the medulla with minimal or no injury to other brain structures. We studied infant Syrian hamsters (*Mesocricetus auratus*) because the immaturity of their nervous system at birth is well documented (*6*), and axons of the CNS would thus be likely to show greater plasticity.

In all cases the animal's left pyramidal tract (originating from the left sensorimotor cortex) was cut 2 to 3 mm rostral to the pyramidal decussation with a fine knife (Fig. 1). A total of 27 animals were operated on at 2, 3, 4, 5, 8, 12, and 20 days. Several additional animals received pyramidal tract lesions in adulthood. The animals were reared for at least 3 months, and then a small quantity of [^{3}H]proline (10 to 20 μCi in 0.5 to 1 μl)

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(specific activity, 30 Ci/mmole, New England Nuclear) was injected by means of a Hamilton 10 μl syringe into the left sensorimotor cortex ipsilateral to the lesion. After 3 to 5 days, the hamsters were perfused through the heart with 10 percent formalin. Frozen sections of the entire brains and cervical spinal cords were processed by the autoradiographic method for tracing labeled pathways.

The results at all ages showed no labeled axons below and ipsilateral to the lesion or growing through the lesion, demonstrating that the tract was completely severed and that axons did not regenerate directly across the cut. Instead, we observed a massive new growth of the severed axons originating from the pyramidal tract approximately 1 to 3 mm rostral to the cut. As shown in the autoradiographs from an animal operated on at 5 days (Fig. 2, E and F), labeled axons left the tract and sprayed out over a large region of the brainstem in a fanlike array. Comparable autoradiographs (Fig. 2, A and B) from the same levels through the brainstem of a normal animal whose left cortex was labeled with a similar quantity of [^{3}H]proline in adulthood show no such fiber system in the normal brain (*7*). Although the anomalous fibers in the brain operated on during infancy fanned out to both sides of the medulla, most of the fibers crossed to the opposite side. Whereas the labeling of ipsilaterally directed axons appears to extend no farther than 100 to 200 μm caudal to their origin from the labeled pyramid, the axons crossing to the opposite side of the brain coalesce into a compact bundle, which descends for a distance of 6 to 7 mm until it reaches the cervical spinal cord. Some fibers may have descended to lower spinal levels as in the normal animal, but we did not examine the thoracic or lumbar cord. In all experimental animals, the trajectory of the anomalous pathway was consistent, descending just medial to the spinal trigeminal nucleus.

Although the course of the regrowing pyramidal tract fibers is completely abnormal, their pattern of termination appears normal. In the normal adult hamster, as in other rodents (*8*), the pyramidal tract, after decussating, gives off some rostrally ascending fibers to the contralateral dorsal column nuclei (DCN). The majority of the pyramidal tract fibers descend through the ventral zone of the dorsal column and then project mediolaterally into the dorsal horn of the spinal cord. By contrast, in the experimental infants the newly formed pyramidal pathway decussates in an anomalous location rostral to the lesion and travels caudally in an aberrant position but on the correct side of the brain. The new fibers do not cross in the normal decussation, nor do they enter the dorsal column. Nevertheless, they terminate appropriately in the DCN and cervical spinal cord by entering these sites in a lateral-to-medial direction. Figure 2 (C and G) shows the similarity between the stripelike normal pattern of pyramidal terminations in the DCN and the terminations formed by the regrowing axons. Terminations formed by the normal and aberrant pyramidal tract in the cervical spinal cord are also similar in their pattern of organization (Fig. 2, D and H). This innervation of the DCN and spinal cord by the newly formed pathway occurs rapidly. By injecting the cortex at 2-day intervals after the pyramidal tract was cut at 8 days of age, we were able to calculate an axonal growth rate of approximately 1 mm per day.

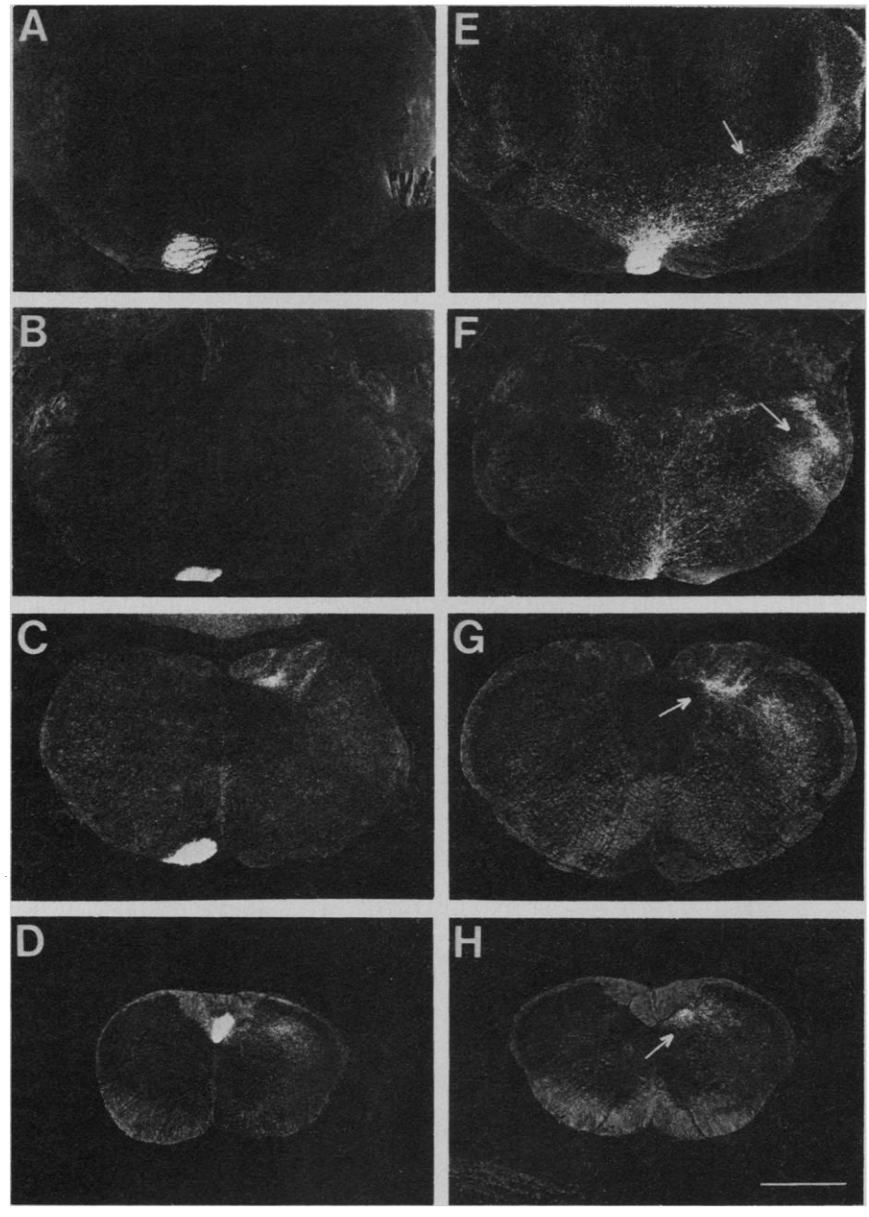

Fig. 2. Dark-field autoradiographs of brainstem and spinal cord sections from adult hamster brains 3 days after [^{3}H]proline was injected into the left sensorimotor cortex. In the normal brain (A to D), the labeled left pyramidal tract descends through the medulla on the base of the brain without giving off brainstem collaterals. Terminations in the dorsal column nuclear (C) and cervical spinal cord (D) are completely crossed. Note the final position of the pyramidal tract in the ventral zone of the dorsal column (D). (E to H) Brain of an adult that had sustained a pyramidal tract lesion at 5 days of age; the lesion was located just caudal to the level shown in (F). Labeled axons regrowing from the severed tract crossed to the opposite side (E), formed a compact bundle (F), and terminated appropriately in the contralateral dorsal column nuclear (G), and cervical spinal cord (H) in a pattern resembling the normal terminations (C and D). In (H), the dorsal column on the right side of the spinal cord is somewhat shrunken because of the absence of the normal pyramidal tract. Scale bar, 1 mm.

With regard to the effect of age on the capacity of severed axons to regrow, the difference between animals operated on as infants and as adults is dramatic. The most massive regrowth seems to have occurred if the axons were cut during the first week of life. Thereafter the development of new growth declined until, by 20 days of age, only a small bundle of fibers projected across the brainstem rostral to the cut. None of these axons, however, could be followed caudally into the DCN or spinal cord. Axons cut in the adult animal showed no capacity for regrowth. Rather, the pyramidal tract rostral to the cut degenerated severely so that we could observe no significant transport of label from the cortex beyond the level of the pons. This result agrees precisely with a previous experiment in which fiber degeneration methods were used with adult hamsters to show that degeneration of the severed pyramidal tract occurs in the retrograde direction as far back as pontine levels (*9*).

In order to determine whether sprouting of intact axons from the contralateral side would occur simultaneously with the massive new growth from the damaged fibers, we injected [^{3}H]proline into the cortex contralateral to the lesion in another series of infants. Sprouting did indeed take place (*10*). In animals as old as 1 week of age, we observed a small but distinct anomalous ipsilateral projection arising from the labeled pyramidal tract at the decussation (Fig. 3). The abnormally directed ipsilateral fibers, however, reached their target sites on the incorrect side of the brain by means of the normal dorsal columnar pathway.

We have demonstrated that axons severed at an early stage of development are capable of extensive new growth. Most previous attempts to investigate the regenerative capacity of damaged CNS axons have shown that if new growth occurs at all, it is abortive or inappropriate. In fact, the formation of premature, abnormal synapses has often been invoked as an explanation for the inhibition of further axonal growth (*3*-*5*). Even in instances of growth from axons severed early in life, the growing sprouts do not necessarily establish their normal connections. For example, Devor showed that the collateral sprouts emitted by damaged axons of the lateral olfactory tract in infant hamsters could form anomalous connections outside as well as within the normal olfactory projection cortex (*11*). After lesions are made mechanically in the adult rat spinal cord, growing monoamine axon sprouts enter the necrotic area but do not grow through it into the distal part of the cord

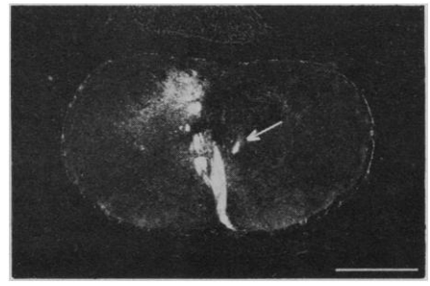

Fig. 3. Dark-field autoradiograph of a section through the pyramidal decussation of an adult hamster brain to show sprouting from intact pyramidal fibers after an early lesion was made in the contralateral pyramid. Lesion of the left pyramid was made at 2 days of age. The right sensorimotor cortex was injected with [^{3}H]proline in adulthood. The arrow shows a small, anomalous, ipsilateral pathway arising from the pyramidal decussation; this ipsilateral bundle projects to the incorrect side of the brain. Scale bar, 1 mm.

to their original sites (*12*). Regeneration into original sites has been suggested for the bulbospinal monoamine neurons in the adult rat, but since the original lesions were chemically induced, the actual disintegration of the axons (and hence their true regeneration) remains uncertain (*13*). Similarly, sprouts from axons in the visual system often have anatomically and functionally inappropriate terminations (*14*). Thus, in contrast to previous studies, our results show that regrowing CNS axons in the infant animal can reach their appropriate targets even over long distances. Although the axons made an anomalous decussation and did not follow their original pathway, they crossed to the correct side of the brain to innervate their normal terminal sites.

It might be argued that our results simply represent the labeling of late-arriving fibers undamaged but redirected by the lesion. We consider this unlikely because concurrent experiments have demonstrated that the pyramidal decussation is well developed by 5 days of age. Using autoradiographic labeling methods, we have traced the growth of the hamster's pyramidal tract axons into the spinal cord throughout the first 2 weeks of life and have found that by 5 days of age the decussation is massively labeled (*15*). Moreover, results obtained with the more sensitive horseradish peroxidase techniques (*16*) reveal that as early as 2 days of age, the pyramidal decussation is heavily labeled with the anterograde transport of horseradish peroxidase (*17*). Thus, the observation that regrowth could occur from axons damaged as late as 20 days argues further against a hypothesis of late-arriving fibers. Rather, it seems likely that continued growth of the cut axons occurs as long as the axon is in its growth phase. Thus, the reduction of regenerative capability after the first postnatal week may coincide with the formation of corticospinal synaptic connections and hence the cessation of axonal growth. Lacking the ability to regrow, some axons severed at later developmental stages and all of those cut in adulthood undergo retrograde degeneration from the site of injury.

Preliminary behavioral results indicate that the morphological plasticity exhibited by the immature axons may have important functional consequences. Previous experiments showed that hamsters subjected to unilateral pyramidal tract lesions as adults exhibit a marked deficit in digital function from which they do not recover (*18*). By contrast, observations of our animals operated on as infants suggest that they are able to develop normal motor function of the forepaw. Further experiments will be needed to determine precisely the role of new axonal growth in maintaining normal motor behavior, particularly the fine motor control of the digits.

The factors guiding the newly growing fibers to the correct side of the brain are unknown at present. It is likely, however, that these factors operate during normal development. Therefore, the pyramidal tract in newborn hamsters may provide a useful fiber system that can be manipulated early in development to allow us to study the mechanisms regulating the decussations of axonal pathways.

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High-Speed Cinematographic Evidence for Ultrafast Feeding in Antennariid Anglerfishes

Abstract. *Analyses by means of high-speed, light cinematography at 800 and 1000 frames per second have shown that members of the shallow-water anglerfish genus* Antennarius *are capable of producing an enormous suction pressure for prey capture by means of an extraordinarily rapid expansion of the buccal and opercular cavities. Prey is totally engulfed at speeds considerably greater than those recorded for any other fish. The structural adaptations responsible for this rapid prey engulfment provide anglerfishes with one of the fastest known vertebrate feeding mechanisms.*

Early comparative studies of the biomechanics of feeding in fishes were based solely on anatomical data (*1*). More recently, however, anatomical data have been integrated with functional data obtained through the use of living material and cinematographic and electromyographic techniques (*2*). The addition of these new techniques to anatomical analyses largely avoids the problems of extrapolating function from form and has provided a more accurate assessment of the role of individual bones, muscles, and ligamentous connections during feeding activity. In the past, cinematographic analyses have been limited to film speeds of 18 to 250 frames per second (*2a*). It has been suggested, however, that higher speeds in filming might reveal that single feeding events in fishes occur at greater speeds than previously believed (*2*). We report single feeding events in three species of shallow-water anglerfishes of the genus *Antennarius* filmed at speeds of 800 and 1000 frames per second, showing that mouth cavity (buccal and opercular cavities) expansion and subsequent prey capture take place at speeds that are more than four times greater than those described for fishes (*3*).

The Antennariidae, largest of the four families of the lophiiform suborder Antennarioidei, includes with few exceptions, shallow to moderately deep-water bottom dwellers with representatives in tropical and temperate waters of all major oceans and seas of the world (*4*). They are structurally and chromatically cryptic forms whose piscivorous

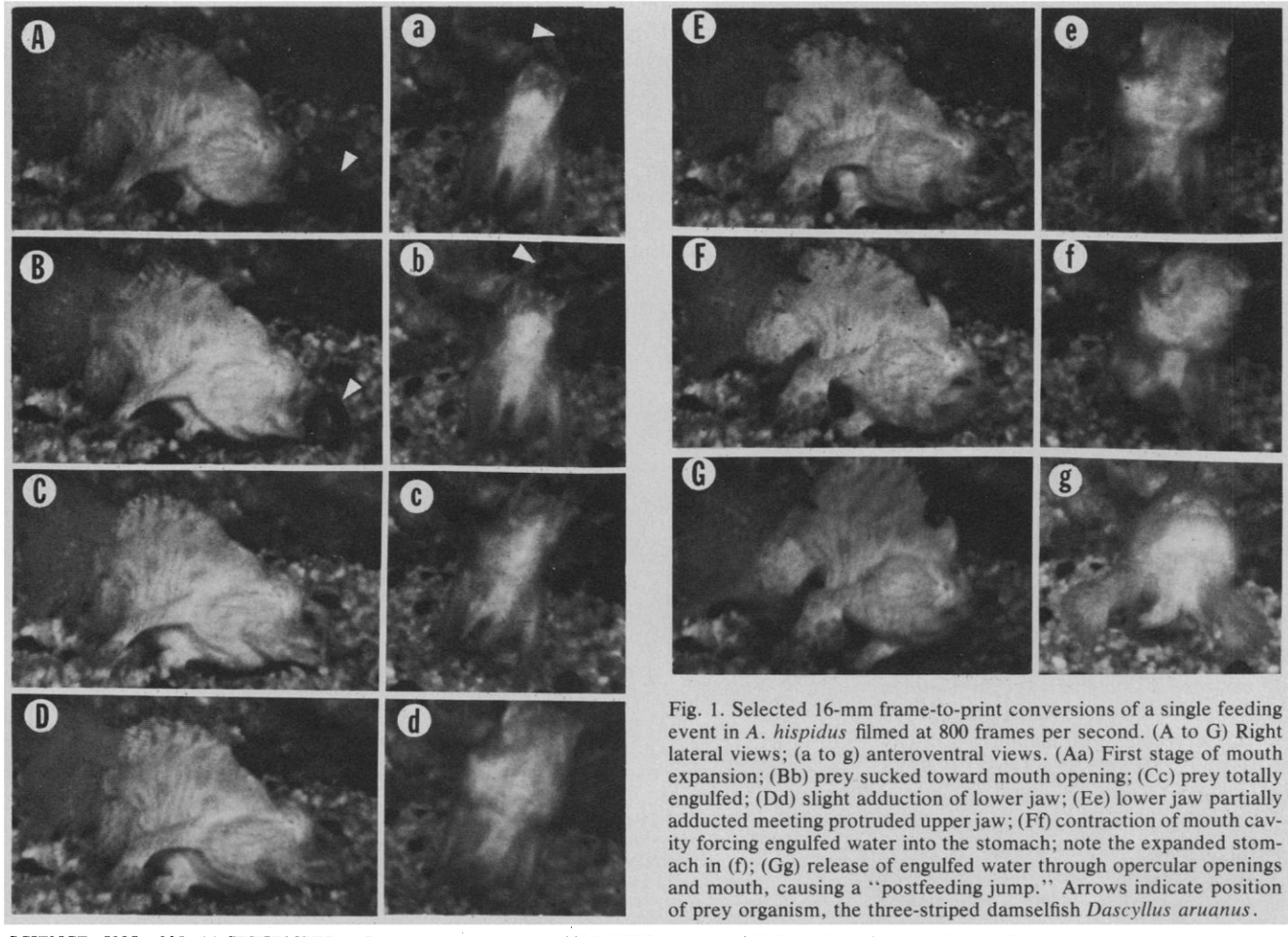

Fig. 1. Selected 16-mm frame-to-print conversions of a single feeding event in *A. hispidus* filmed at 800 frames per second. (A to G) Right lateral views; (a to g) anteroventral views. (Aa) First stage of mouth expansion; (Bb) prey sucked toward mouth opening; (Cc) prey totally engulfed; (Dd) slight adduction of lower jaw; (Ee) lower jaw partially adducted meeting protruded upper jaw; (Ff) contraction of mouth cavity forcing engulfed water into the stomach; note the expanded stomach in (f); (Gg) release of engulfed water through opercular openings and mouth, causing a "postfeeding jump." Arrows indicate position of prey organism, the three-striped damselfish *Dascyllus aruanus*.

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