Novel Structural Elements Identified during Tail Resorption in *Xenopus laevis* Metamorphosis: Lessons from Tailed Frogs

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When the tail of the *Xenopus laevis* tadpole resorbs at the end of metamorphosis, various cell types, including muscle, fibroblasts, skin, and spinal cord, are lost at about the same time. However, feeding frogs with tails can be produced by inhibiting thyroid hormone production at the climax of metamorphosis with the goitrogen methimazole. These tails lose their fast muscle preferentially, showing that the different cell types of the tail have different fates and confirming that more than one cell death program is involved in tail resorption. Both normal and methimazole tails contain “cords,” novel structures that consist of two dorsal and two ventral parallel rows of slow muscle bundles joined by collagen fibers that run the length of the tail. The cords persist until the very end of tail resorption, being the last structure to dissolve. When thyroid hormone induces expression of proteolytic enzymes in the notochord sheath, the notochord, a structural rod that runs the length of the tail, begins to buckle, demonstrating that the tail is under tension. When sections of the tail that contain cords are surgically separated from the notochord, they contract *in vitro*, suggesting that the cords contribute to the tension that augments tail resorption. © 1999 Academic Press

Key Words: metamorphosis; *Xenopus*; notochord; slow muscle.

INTRODUCTION

Drastic remodeling occurs during anuran metamorphosis. While some organs, notably the limbs, undergo rapid growth, others, such as the gills and tail, are degraded and resorbed completely. The completeness and rapidity of the degeneration gives the impression that the pervasive loss of structure, due to death and resorption, is attributable to a single underlying mechanism.

In response to thyroid hormone (TH), four genes in *Xenopus laevis* have been identified that are down-regulated in the tail. All four genes are expressed exclusively in the apical cell layer of the larval epidermis, and their expression is down-regulated as part of the tail resorption program (Furlow et al., 1997). In contrast a greater number of genes is up-regulated by TH in the tail. These genes fall into several groups defined by their kinetics of regulation and the cells in which they are expressed (Wang and Brown, 1991; Brown et al., 1996; Berry et al., 1998). One group of genes, the delayed response genes, is up-regulated in fibroblasts associated with the two collagen lamellae in the tail, namely the notochordal sheath and the subepidermal layer, and is not expressed in epidermis or muscle. These observations led to the idea that tail resorption is controlled by multiple cell death pathways (Berry et al., 1998). The different programs would have to be coordinated during spontaneous tail resorption, since the whole tail is destroyed.

We show in this report that under certain conditions, these programs can be separated resulting in a persistent tail with an intact notochord and fins, but devoid of fast muscle. The notochord plays a central role in the structure and loss of the tail. The tail appears to be under tension that may facilitate its shortening once the notochord has collapsed. We have discovered two pairs of repeated muscles held together by collagen that are the last structures to resorb. These muscle “cords” may contribute to the tension in the shortening tail.

MATERIALS AND METHODS

Animals

*X. laevis* tadpoles were purchased from *Xenopus* I or raised in the lab from fertilized eggs. They were fed suspensions of nettle powder...
and crushed trout chow. Tadpoles were anesthetized with ice or with 0.01% MS222, prior to removal of their tail into 2X Stein-
berg's solution (Peng, 1991), and then immediately killed. Stein-
berg's solution (2X) was used when dissecting the large tails of
metamorphosing tadpoles, because its salt concentration is equiva-

cent to amphibian Ringer's. Metamorphic stages followed the
criteria of Nieuwkoop and Faber (NF) (1994).

Tadpoles were treated with methimazole (Buckbinder and Brown,
1993) to obtain incomplete metamorphosis. The tadpoles were kept at
a density of 10 per liter in 1 mM methimazole continuously from
NF58. The water with methimazole was changed every 2–3 days.
There was no toxicity due to methimazole, and some of the resulting
tailed frogs were kept in methimazole for several months.

Immunocytochemistry

Immunocytochemistry was performed on tail sections and on
whole mounts. For sections, pieces of tail were oriented in OCT
medium and frozen. Sections were cut at 12 µm on a cryostat and
attached to slides by drying at room temperature for 5–10 min. The
sections were fixed with either 4% formaldehyde in PBS or 70%
ethanol, depending on the primary antibody, and immunostained.
For whole mounts, tadpoles or tails were fixed in 4% formaldehyde
in PBS or in Dent's fixative (80% methanol, 20% dimethyl sulfox-
ide; Dent et al., 1989). They could be stored at −20°C in Dent's
indefinitely. Tadpoles, 2 weeks of age or younger, could be pro-
cessed intact, but larger tails from NF stages 57 and older were
dissected open to allow antibody access to all tissues. The dissection
often included peeling the skin off. In these instances, the cord muscles
described under Results usually remained attached to the skin.

Immunostaining of whole mounts followed standard protocols
with washes in PBT (0.1% Triton X-100 in PBS), blocking in 10 mg/ml
bovine serum albumen (BSA) in PBS, incubation in primary antibody,
extensive washes in 2 mg/ml BSA in PBT, incubation in secondary
antibody, and extensive washes in 2 mg/ml BSA in PBT. Antibody
incubations and washes for whole mounts lasted several hours to
overnight. Triton X-100 was not used for sections, and antibody
incubations were 1 h followed by repeated 3-min washes in PBS.

Primary and secondary antibodies were diluted in 10 mg/ml BSA
in PBT. The primary antibodies were mouse monoclonals. F59,
S58, and S46 (gift from F. E. Stockdale) were used as culture
supernatants, diluted 1/10. These antibodies are directed against
chicken myosin heavy chains, with F59 recognizing fast isoforms
and S46 and S58 recognizing slow isoforms (Crow and Stockdale,
1986; Stockdale and Miller, 1987). They had been used previously
for X. laevis tadpoles, in which F59 recognizes both fast and slow
muscles, while S46 recognizes only slow muscles (Radice, 1995). In
our hands, S46 and S58 recognize the same muscles, and we have
used them interchangeably. TM228 (Sigma) was used as mouse
ascites fluid, diluted 1/100. It is directed against chicken tropomyo-
sin and had been used previously on metamorphosing X. laevis
(Nishikawa and Hayashi, 1994). S46 and S58 could only be used on

FIG. 1. Tail structure. (A) This cross section through the tail of a large premature tadpole (NF57) was immunostained (F59 antibody)
to reveal muscle. The large muscle masses (m) flank the notochord (n). (B) A similar section, immunostained with S58 antibody, shows slow
muscle at the periphery, as well as a ventral cord (v). (C) A section of a tail at NF64, immunostained with F59, shows the persistence of two
dorsal muscle cords. (D) A hematoxylin-stained section of a N F63 tail reveals two distinct muscle cords (c), flanking the dorsal blood vein
(v). Scale bar in (A), 1 mm for A, B, C; 0.25 mm for D.

FIG. 2. Tail muscle cords. (A) A NF64 tail, late in metamorphosis, was isolated, bisected, and immunostained as a whole mount with S46
antibody. The periodic staining appears as four rows, representing the two dorsal and the two ventral muscle cords. Scale bar, 1 mm. (B)
The periodic muscle cords are visible in a fixed, unstained NF63 tail. (C) When the skin is peeled from the tail, the muscle cords usually
isolate with the skin (NF62). (D) Immunostained cord isolates from a NF62 tail (top) and a NF63 tail (bottom) show that both the muscle
dark) and the intervening collagen (light) are reduced in size, as the tail is resorbed. Scale bar, 1 mm.

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ethanol or Dent's fixed tissue, while formaldehyde fixation sufficed for F59 and TM 228. M 1-B4 and 3A 10, obtained from the Developmental Studies Hybridoma Bank, were used as culture supernatants at dilutions of 1/10 and 1/2, respectively. M 1-B4, raised against a chicken collagen preparation, recognizes tenascin (Chiquet and Fambrough, 1984), while 3A 10 recognizes a neurofilament-associated antigen. The secondary antibody was a horseradish peroxidase-conjugated goat anti-mouse antibody (Sigma) at a dilution of 1/3000. Staining was done with a Vector DAB Substrate Kit.

Notochord Observation

Procedures used for bone-cartilage staining (Klymkowsky and Hanken, 1991) were followed, with or without staining with Alcian blue and Alizarin red. Animals were fixed in 3.7% formaldehyde in PBS, skinned, and eviscerated, digested with 1% trypsin in 2% Borax, and gradually cleared in mixtures of 0.5 N KOH and glycerol.

RESULTS

Tail Muscle Loss in Metamorphosis

The tadpole tail contains a notochord, flanked by large muscle masses (Fig. 1A). The more peripheral muscle fibers are slow muscle, while the bulk of the muscle is fast muscle (Kordylewski, 1986; Kordylewski et al., 1989; Sasaki, 1974, 1977; Muntz et al., 1989; Radice, 1995). Both fast and slow muscle were recognized by F59 antibody (Fig. 1A), while slow muscle was recognized specifically by antibodies against chicken slow myosin (S46, S58) and chicken β-tropomyosin (TM 228) (Fig. 1B). During premetamorphosis (NF 52–58), the limbs grow and differentiate, and at NF 58, the forelimbs break through the overlying operculum, signaling the start of metamorphic climax. By NF 62, the gills have resorbed, and the head and body have become froglike. The tail, however, remains long and fully muscled and is about 40 mm or two to three times the length of the body. During NF 63–64, the tail shrinks rapidly to less than 10 mm in 3 days. As it shrinks, immunostaining of fast muscle was lost. The only remaining muscle is four small patches of slow muscle (Fig. 1C) which are found in some but not all transverse sections symmetrically located on either side of the dorsal and ventral blood vessels (Fig. 1D). Immunostaining of NF 64 tails as whole mounts confirmed the periodic repetitive nature of these muscle cords (Fig. 2A).

Identification of the Cords, Novel Muscle Structures in the Tail

The four cords of slow muscle ran the full length of the tail with two on either side of the dorsal blood vessel and two on either side of the ventral blood vessel (Figs. 1D and 2B). The muscle bundles in the cords were separated by gaps that were larger than the myotendinous junctions between adjacent chevron-shaped blocks of fast muscle fibers that made up the bulk of the tail musculature (Fig. 2D). The gaps were stained with an antibody against tenascin (M 1-B4) and were largely filled with collagen fibers that linked together adjacent muscles in the cords (Fig. 3). The cords could be isolated from the rest of the muscle by carefully peeling the skin from the tail (Fig. 2C).

These slow muscles were not detected in an early tadpole (NF 38) but were present at the start of feeding (NF 46). They were the most dorsal fibers in each myotome in both the trunk and the tail (Figs. 4A and 4B), and they were also the most ventral fibers of each myotome in the tail as well as the most ventral fibers of the m. rectus abdominus in the trunk (Fig. 4C). The similarity of staining between the most dorsal myotomes in both the trunk and the tail of the developing tadpole suggested that the dorsal cords in the tail were a continuation of trunk musculature. The most dorsal muscle of the trunk is the m. longissimus dorsi (Ryke, 1953; Kordylewski and Grusza, 1986), and the tail dorsal cords were an attenuation of this muscle. The more medial fibers of the m. longissimus dorsi were slow fibers, whose pattern narrowed into the dorsal cords (Fig. 4D).

Tail Structure in Incomplete Metamorphosis

During the rapid shrinkage of the tail (NF 63–64), the dissolution of all cell types and structures occurs in just a few days, suggesting a general destruction of the tail. A different impression was gained by examining animals with incomplete metamorphosis. We found that tailed frogs could be produced by the addition of the gilrogen methimazole at just the right time during the climax of metamorphosis. This inhibitor blocks synthesis of TH by the thyroid gland. Treatment at NF 57 or earlier with 1 mM methimazole resulted in tadpoles that grew but did not continue metamorphosis, while treatment at stage NF 59 or later did not block completion of metamorphosis. Exposure of tadpoles at late NF 57 or NF 58 to 1 mM methimazole produced feeding frogs which retained their tail. Late NF 57 is just before forelimb emergence, which occurs at NF 58.

Of 294 tadpoles treated with methimazole at NF 57–58, 67% completed metamorphosis or retained only a small piece of tail. Ten percent arrested with full tails but with incomplete gill resorption. These animals failed to eat worms, a behavioral change in their feeding that normally occurs just as tail resorption is completed. These arrested tadpoles are similar to those seen when metamorphosis is inhibited by overexpressing the deiodinase III gene in transgenic X. laevis (Huang et al., 1999). Deiodinase III inactivates TH, reducing the endogenous hormone of climax to levels below the threshold needed to complete metamorphosis.

The remainder of the animals (23%) completed gill resorption, ate worms, and grew even though they had retained most of their tail (Fig. 5). The tails continued to show the distal flickering, characteristic of X. laevis tadpole swimming. When tailed frogs were transferred out of methimazole 2 to 3 weeks after feeding began, the tails gradually resorbed in most cases. The tailed frogs often had bends in their tails (Fig. 5). The central part of the tail including the notochord was bent, while the more dorsal
and ventral tissues, including the cords, followed a shorter course relative to the notochord. This could be seen by the straighter run of the blood vessels (Fig. 5). Similar bends were occasionally observed at NF63/64 of normal metamorphosis in animals not treated with methimazole.

The tails of these frogs were thinner than normal tadpole tails and lacked the large chevron-shaped blocks of fast muscle. Histological examination revealed a large notochord and a paucity of muscle. A methimazole-treated frog with a full tail (body:tail::28 mm:41 mm) and one starting tail resorption after methimazole withdrawal (body:tail::33 mm:23 mm) were examined by immunostaining. Both tails had a large notochord, with little muscle (Figs. 6A and 6B). Most of the muscle in the former frog and all in the latter frog was slow muscle (Fig. 6C). Both the slow peripheral muscle and the periodic dorsal and ventral muscle cords were present. The selective loss of fast muscle clearly demonstrated that resorption programs of different cell types in the tail could be separated.

**Degeneration of the Notochord and Tail Contraction**

The persistence of the notochord in tailed frogs that have no muscle mass suggested that the normal rapid shrinkage of the tail at NF63/64 was due to degeneration of the notochord. To visualize notochord degeneration, animals were fixed at NF64 and cleared, according to the protocol normally used in bone-cartilage staining. When viewed in this way, the notochord was folded and wrinkled, suggesting that the notochord was being compressed as it weakened (Fig. 7A). When unfixed tails were cut open to expose the notochord, the notochord had a wrinkled appearance at NF63 (Fig. 7B), but not at NF62. This appearance suggested that the notochord had been weakened in the interim and could now be compressed like an accordion. These observations, along with those of bent tails (Fig. 5), indicated that there was tension in the tail acting on a weakened notochord.

**Contractility of the Dorsal and Ventral Cords**

The persistence of the dorsal and ventral cords of slow muscle raised the possibility that they could supply the compressive force on the notochord. As the tail shrank, so too did the cords, and both the repeated muscle bundles and the gaps between them were reduced in size from NF62 through NF64 (Fig. 2D). As a simple test of the ability of these muscles to contract, tails were amputated from anesthetized metamorphosing tadpoles, placed in 2X Steinberg's solution, and cut with a scalpel to isolate regions of the tail. The portions containing the dorsal and ventral cords shrank back as soon as they were freed from the central piece, containing the notochord (Fig. 7B). Shrinkage was about 2–3 mm or 20% of the cut length, and the cord containing pieces often curled, suggesting that one side of the piece was more contractile than the other. These observations suggested that the cords remain contractile during NF63–64, when rapid shortening of the tail occurred.

The cords were closely associated with the large mass of degenerating fast muscle, so it was difficult to separate the contribution of either one to the contraction. In order to test better the activity of the cords, the dissections were repeated on tails from methimazole-induced animals, in which most if not all of the fast muscle was gone. Separation of pieces containing the dorsal or ventral cords from the notochord led to some shrinkage and curling of the cut pieces. In addition to containing the cords, however, these cut pieces retained the dorsal and ventral fins that could inhibit shrinkage. When further cuts were made to isolate the cords from the fins, the fins expanded to their original lengths, approximately equivalent to that of the notochord, and the pieces with the cords were curled and shortened (Fig. 8). The cords shrank by 2–3 mm or 26 ± 6% (n = 34) of their cut length.

The cords consisted of muscles and gaps. The large size of the gaps, containing collagen and tenascin, raised the possibility that the gap materials could be elastic (Coyne et al., 1997; Oberhauser et al., 1998) and contribute to the contraction. To test whether both units are capable of contraction, dorsal cords plus skin were either isolated from fixed intact tails or isolated from living, freshly amputated tails and then fixed. The former were uncontracted when fixed, while the latter contracted before fixation. Since both the muscle and the gap decrease in size with tail resorption, comparisons were made between matched NF63 animals, with body/tail ratios of 0.69–1.2 (12 animals). In 5/6 pairs, the muscles were significantly smaller in the contracted cases, while in 2/6 pairs, the gaps were significantly smaller (Table 1). The muscles in four of the contracted cases were half the length of those of the uncontracted case, while in the two cases of smaller gaps, the decrease was less than 25%. These results indicate that the muscle is responsible for the contraction of isolated cords.

The cords appeared to be innervated based on immunocytochemical detection of nerves running along the cords (antibody 3A10). We tested whether neural stimulation was required for either the in vitro contractions or the tail resorption. Stage 59 tadpoles were paralyzed by intraperitoneal injection of 5 mg of α-bungarotoxin or by addition to their water of 1 mg/ml atropine or 10 mg/ml 2,3-butanedione monoxime. An hour after they were completely immobilized, their hearts were still beating. Then their tails were amputated and subjected to the in vitro contraction assay. None of these inhibitors inhibited contraction. As an in vivo test, the spinal cord was severed in the proximal third of the tail at NF61–63. The tail could still move, presumably by muscles at its base, but the flickering of the distal part of the tail ceased. Despite this, there was no obvious effect on tail resorption, suggesting that neural-mediated muscle activity is not required.

**DISCUSSION**

Our observations on the resorption of the normal X. laevis tadpole tail at metamorphosis indicate a morphologi-
cal order to its resorption. First, fast muscles are preferen-
tially lost compared to slow muscles. Second, a novel set of
four cordlike tail muscles is the last to go. The notochord,
which provides structural support for the tail, appears to be
under a compressive force, so that when the notochord
weakens, the tail collapses.

FIG. 4. Origin of the cords. (A) A just-feeding tadpole (N F46), immunostained for slow muscle (S58), shows staining of the most dorsal fibers of each myotome. Scale bar, 1 mm. (B) Higher magnification of dorsal immunostaining. Scale bar, 0.25 mm. (C) The ventralmost fibers are also slow muscle. Scale bar, 0.25 mm. (D) Dorsal view of a large N F60 tadpole, immunostained for slow muscle, shows the attenuation of the m. longissimus dorsi of the trunk (right) into the dorsal cords of the tail (left). Scale bar, 1 mm.

FIG. 5. Tailed frog produced by methimazole treatment. (A) This tailed frog had been feeding for about a month. Its snout–vent length was 24 mm. (B) A higher magnification of the bend in the tail, in the same orientation as in (A), shows the dorsal and ventral blood vessels running a straighter course than the central part of the tail, which contains the notochord.
Differential Tail Muscle Response during Resorption

As the muscle rapidly degenerates at NF63–64, the slow cords persist longer than either the fast or the slow peripheral muscle. It has not been possible to distinguish whether there is a difference in timing of loss of the fast muscle and the slow peripheral muscle during spontaneous metamorphosis because of the rapidity of the process. However, this difference is obvious in tailed frogs that are produced by

FIG. 6. Tail structure in a tailed frog, produced by methimazole. (A) A hematoxylin-stained cross section shows a large notochord (n), peripheral muscle, dorsal and ventral cords (c), and the absence of the large chevron muscles. (B) A cross section, immunostained with F59 for fast and slow muscle, shows the paucity of muscle. Compare this section to Fig. 1A. (C) A cross section, immunostained with S46, shows that most if not all of the muscle, seen in (B), is the slow type. Scale bar in (A), 0.5 mm for A, B, C.

FIG. 7. Compression of the notochord. (A) In this cleared animal at NF64, the notochord in the tail is bent and folded. (B) This tail was cut from the animal (NF63) and carefully bisected, so as not to injure the notochord. The muscle-containing pieces contracted back, and the notochord had a wrinkled appearance. Scale bar in (A), 2 mm for A, 1 mm for B.
partially blocking metamorphosis with methimazole. These animals retain both the cords and the slow peripheral muscles but lose their fast muscle. A tailed frog was also produced by overexpressing the deiodinase III gene that was introduced by transgenesis (Huang et al., 1999). This animal's tail showed the same differential loss of fast muscle and retention of the cords (Huang et al., unpublished data).

It is clear that tadpole tail muscle cannot be regarded as a homogeneous tissue, as the fast muscle, the slow peripheral muscle, and the dorsal and ventral cords have different developmental histories and different responses to thyroid hormone.

The strongest evidence to date that muscle degeneration is cell autonomous comes from cultures of myoblasts derived from tadpole tail that respond to thyroid hormone by undergoing apoptosis (Yaotoa and Nakajima, 1997). In contradiction to this idea, Niki et al. (1982) reported that the epidermis is required for thyroid hormone-induced tail resorption. Similarly, genes involved in tissue destruction, such as stromelysin-3 and collagenase-3, are up-regulated in a layer of subepidermal fibroblasts, adjacent to muscle, but not in tail muscle (Berry et al., 1998), suggesting that the muscle could be killed by proteins secreted from another tissue. These genes are also up-regulated in the myotendinous junctions to which the muscle fibers are attached, and the loss of these junctions may lead to programmed cell death of the muscles (Berry et al., 1998).

The persistence of the slow peripheral muscles in the tailed frogs is surprising. They are closest to the subepidermal fibroblasts, so it is expected that they would be destroyed earlier by secreted enzymes than the more internal fast muscles. One possibility is that the slow peripheral muscles are particularly resistant to stromelysin-3 and collagenase-3. Another possibility is that the slow peripheral muscle regenerates from satellite cells in the tailed frogs, while the fast muscle is unable to do so. A more careful examination of the fate of the slow peripheral muscle in both normal metamorphosis and in the methimazole-treated animals is required to resolve this issue.

### Collapse of the Notochord

The tadpole tail is distinguished from that of other vertebrates by its lack of vertebrae, either as bone or as cartilaginous precursors (Wassersug, 1989). The tail is mainly muscle with a notochord at its core. The existence of tailed frogs, with a notochord but lacking fast muscle, demonstrates that the notochord is the main structural element of the tadpole tail. The destruction of the collagen notochord sheath occurs after fibroblasts in the sheath express a group of TH-induced genes including secreted proteases such as collagenase-3 (Berry et al., 1998). These cells then invade the acellular sheath. The notochord weakens, but before it dissolves, the notochord bends, wrinkles, and buckles (Figs. 5 and 7), indicating that it is under a compressive force. The tail shortens by half of its length each day, and its disappearance is the final event of metamorphosis.

The buckled appearance of the X. laevis notochord is similar to that seen in some species of ascidian tadpoles.

### Table 1

<table>
<thead>
<tr>
<th>Pair</th>
<th>Body/tail (mm)</th>
<th>Muscle (mm)</th>
<th>Gap (mm)</th>
<th>Body/tail (mm)</th>
<th>Muscle (mm)</th>
<th>Gap (mm)</th>
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<td>0.69</td>
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<tr>
<td>b</td>
<td>0.77</td>
<td>0.24</td>
<td>0.23</td>
<td>0.77</td>
<td>0.12**</td>
<td>0.22</td>
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<tr>
<td>c</td>
<td>0.77</td>
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<td>0.27</td>
<td>0.80</td>
<td>0.14**</td>
<td>0.28</td>
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<tr>
<td>d</td>
<td>0.82</td>
<td>0.23</td>
<td>0.33</td>
<td>0.82</td>
<td>0.18**</td>
<td>0.27*</td>
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<tr>
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<td>0.18</td>
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<td>0.09**</td>
<td>0.18</td>
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<tr>
<td>f</td>
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<td>0.17</td>
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Note. Dorsal cords were isolated from tadpole tails after (Uncontracted) or before (Contracted) fixation, and the anterior to posterior length of each repeating unit of a cord was measured. From 6 to 17 muscle and gap units were measured for each dorsal cord, and their size was compared between pairs of tadpoles with similar body/tail ratios. Statistically significant size differences (t test, **P < 0.001; *P < 0.05) between uncontracted and contracted muscle or gap units in paired samples are indicated.
Tail Resorption in Xenopus Metamorphosis

ACKNOWLEDGMENTS

We thank Frank Stockdale (Stanford University) for the F59, S46, and S58 antibodies; Michael Sepanski for electron microscopy; Deborah Berry for the section in Fig. 1D; and members of the Brown lab for comments and suggestions. The M1-B4 (D. M. Fambrough) and 3A10 (T. M. Jessell, J. Dodd) antibodies were obtained from the Developmental Studies Hybridoma Bank developed under the auspices of NICHD and maintained by the Department of Biological Sciences, University of Iowa, Iowa City, 52242. This work was supported by grants from NSERC, Canada (R.P.E.), and the NIH and the G. Harold and Leila Y. Mathers Charitable Trust (D.D.B.).

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Received for publication July 26, 1999
Revised September 1, 1999
Accepted September 1, 1999