The shocking predatory strike of the electric eel
Kenneth Catania

Electric eels can incapacitate prey with an electric discharge, but the mechanism of the eel’s attack is unknown. Through a series of experiments, I show that eel high-voltage discharges can activate prey motor neurons, and hence muscles, allowing eels to remotely control their target. Eels prevent escape in free-swimming prey using high-frequency volleys to induce immobilizing whole-body muscle contraction (tetanus). Further, when prey are hidden, eels can emit periodic volleys of two or three discharges that cause massive involuntary muscle contraction in nearby animals. The temporal patterns of eel electrical discharges resemble motor neuron activity that induces fast muscle contraction, suggesting that eel high-voltage discharges have been selected to most efficiently induce involuntary muscle contraction in nearby animals.

The electric eel (Electrophorus electricus) is one of just a few species that uses electrical discharges to capture prey and defend against predators. It is the most powerful electrogenic fish, with most of its body composed of electrocytes (muscle-derived biological batteries), providing a combined discharge of up to 600 V (1). Early attempts to understand electricity made use of electric eels (2), and more recently, eels were important for identifying acetylcholine receptors (3) and for providing insights into the evolution of electric organs (4), but little is known about how the eel’s electrical discharge affects prey. In this study, I designed a set of experiments to explore the impacts of the electric eel discharges on potential prey and the mechanism that operates during such attacks.

Electric eels emit three distinct types of electric organ discharges: (i) low-voltage pulses for sensing their environment, (ii) pairs and triplets of high-voltage pulses given off periodically while hunting in complex environments, and (iii) high-frequency volleys of high-voltage pulses during prey capture or defense (movie S1) (5–9). Under most conditions, eels attack free-swimming prey with the latter strategy, using high-voltage volleys combined with a suction-feeding strike. To explore this more common behavior, I simultaneously recorded eel behavior and electric organ discharges in a naturalistic experimental environment (10). Eels began their attack with a high-frequency (~400 Hz) volley of high-voltage pulses 10 to 15 ms before their predatory strike. In response to these volleys, prey voluntary movement was completely arrested 3 to 4 ms after the first strong discharge (Fig. 1 and movie S2). Fish that were not successfully captured during this period of immobility were often able to escape.

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Fig. 1. Eel’s discharge and strike. (A) Electric organ discharge corresponding to plates below. Arrow indicates low-amplitude discharge. (B) Video frames showing that fish movement is arrested by discharge. Red frames indicate electric organ discharge (movie S1). (C) The utility of the discharge illustrated. Shown are the prey fish at 40 ms (green) and later, the position and velocity of the eel and fish at 160 ms (red fish). Green dotted fish outline shows velocity and location of uninterrupted escaping fish matched in time, size, and position from 40 ms, suggesting that the eel would have missed without the discharge.
return to previous movement patterns and escape (movie S2).

To characterize the mechanism by which high-voltage volleys cause this remote immobilization of prey (10), anesthetized fish were pithed (to destroy the brain), the hole was sealed with cyanoacrylate, and the fish was attached to a force transducer. An eel in the aquarium was separated from the fish by an electrically permeable agar barrier (Fig. 2A) (11) and fed earthworms, which it attacked with volleys of its high-voltage discharge. The discharge directed at the earthworms induced strong muscular contractions in the fish preparation, precisely correlated in time with the volley (no tension developed during the weak discharge). A steep rise in fish tension occurred with a mean latency of 3.4 ms ($n = 20$ trials) after the first strong pulse (Fig. 2B), which is similar to the 2.9-ms mean immobilization latency ($n = 20$ trials) observed in free-swimming fish. Tension induced by the eel in the fish preparation was similar to the maximum that could be induced experimentally (fig. S1) (10). This result indicates that fish are immobilized by massive, involuntary muscle contraction.

To further investigate the fidelity of prey muscle contractions relative to the electric organ discharge, the fish preparation was subjected to curare (D). The injection of curare (red) reduced the fish tension (green) and increased the latency of fish tension development (fig. S3).
discharge, and the mechanism of the contractions’ induction, two pithed-fish preparations were stationed side by side (Fig. 2C). The high-voltage discharge reliably created muscle tension with similar form and time course in both fish (Fig. S2). As the discharge frequency decreased, individual fish twitches often emerged on the tension trace, each corresponding to a single discharge (Fig. 2B and Fig. S2). To determine whether the discharge induced muscle contractions by initiating action potentials directly in prey muscles or through activation of some portion of fish motor neurons, one of two similarly sized fish was injected with curare (an acetylcholine antagonist) so as to block the acetylcholine gated ion channels at the neuromuscular junction, whereas the other fish was sham-injected (Fig. 2D). In each of four cases, tension responses in the curarized fish dropped to near zero, whereas the sham-injected fish continued to respond (fig. S3). These findings indicate that fish motor neuron activation is required to induce tetanus in prey. To determine whether this activation of prey motor neurons was the result of central nervous system (spinal) activity or activity in efferent branches of motor neurons, the dual tension experiment was repeated twice with extensively double-pithed fish (in which both the brain and spinal cord were destroyed, but the branches of motor efferents were left intact within the fish body) and compared with a brain-pithed fish. No diminution in contractile response, or difference in contractile response latency, was observed for the double-pithed fish relative to the brain-pithed fish (fig. S2). These experiments suggest that the electric eel’s strong electric organ discharge remotely activates motor neuron efferents of its prey, although this activation could occur anywhere between the spinal cord and the presynaptic side of the neuromuscular junction. Given that the eel’s strong electric organ discharge remotely activates motor neuron efferents of its prey, although this activation could occur anywhere between the spinal cord and the presynaptic side of the neuromuscular junction. Given that the eel’s strong electric organ discharge remotely activates motor neuron efferents of its prey, although this activation could occur anywhere between the spinal cord and the presynaptic side of the neuromuscular junction. Given that the eel’s strong electric organ discharge remotely activates motor neuron efferents of its prey, although this activation could occur anywhere between the spinal cord and the presynaptic side of the neuromuscular junction.

As described above, hunting eels often pause and give off isolated high-voltage doublets (9), particularly in complex environments, when seeking hidden prey or when exploring conductors (movie S3). In the course of the present study, eels stationed behind the agar barrier in the fish tension experiments occasionally emitted such isolated doublets or triplets and then attempted to break through the barrier to reach the fish preparation (movie S4). This suggested that eels were able to detect fish movements through the thin agar barrier, which was not designed to mask
mechanosensory cues. To identify the function of this additional behavior, eels were presented with prey hidden below a thin agar barrier (Fig. 3C). In some cases, eels detected prey through the barrier and attacked directly, but in other cases, the eel investigated the agar surface with a low-amplitude electric organ discharge and then produced a high-voltage doublet. The doublet invariably caused prey movement. Stimulated prey movement was closely followed (in 20 to 40 ms) by a full predatory strike consisting of a strong electric discharge volley and directed attack (Fig. 3 and movie S5), as characterized in the first experiments. The distinct form of the discharge trace in these trials consisted of a doublet (or triplet) followed by a 20- to 40-ms pause (during which prey moved) and then a full discharge volley (Fig. 3D and F).

The results of the doublet experiment suggest that the eels may use doublet and triplet discharges to detect cryptic prey by inducing movement. To test this hypothesis, a pithed fish was placed in a thin plastic bag to isolate it from the eel’s discharge. The electrically isolated fish was positioned below an agar barrier; with electrical leads embedded in the head and tail region (10) that allowed production of artificial fish twitch by the experimenter. Artificial fish twitch was triggered remotely through a stimulator (Fig. 4A), allowing control over its timing and occurrence. When the stimulating electrodes were inactive, eel doublets caused no response in the pithed fish and eels did not attack the preparation (Fig. 4B and movie S6). However, when the stimulator was configured to trigger fish twitch when the eel produced a doublet, the eel’s full “doublet attack” behavior was replicated (Fig. 4C and movie S6). The attack pattern consisted of a doublet, followed by a short pause, during which the prey moved (resulting from the triggered stimulator), followed by a high-voltage volley and strike. This key experiment showed that eels never (10 of 10 trials for each of two eels) followed a doublet with an attack volley without a mechanosensory stimulus, allowing it to cancel the very escape response it has generated. Overall, this study reveals that the electric eel has evolved a precise remote control mechanism for prey capture, one that takes advantage of an organism’s own nervous system.

REFERENCES AND NOTES
10. Materials and methods are available as supplementary materials on Science Online.

INFLAMMATION

Neutrophils scan for activated platelets to initiate inflammation

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Immune and inflammatory responses require leukocytes to migrate within and through the vasculature, a process that is facilitated by their capacity to switch to a polarized morphology with an asymmetric distribution of receptors. We report that neutrophil polarization within activated venules served to organize a protruding domain that engaged activated platelets present in the bloodstream. The selectin ligand PSGL-1 transduced signals emanating from these interactions, resulting in the redistribution of receptors that drive neutrophil migration. Consequently, neutrophils unable to polarize or to transduce signals through PSGL-1 displayed aberrant crawling, and blockade of this domain protected mice against thrombinoinflammatory injury. These results reveal that recruited neutrophils scan for activated platelets, and they suggest that the neutrophils’ bipolarity allows the integration of signals present at both the endothelium and the circulation before inflammation proceeds.

Neutrophils are primary effectors of the immune response against invading pathogens but are also central mediators of inflammatory injury (1). Both functions rely on their remarkable ability to migrate within and through blood vessels. The migration of neutrophils is initiated by tethering and rolling on inflamed venules, a process mediated by endothelial selectins (2). Selectin- and chemokine-triggered activation of integrins then allows firm adhesion, after which leukocytes actively crawl on the endothelium before they extravasate or return to the circulation (3). A distinct feature of leukocytes recruited to inflamed vessels is the rapid shift from a symmetric morphology into a polarized form, in which intracellular proteins and receptors rapidly segregate (4). In this way, neutrophils generate a moving front or leading edge where the constant formation of lamellipodia (actin projections) guides movement, and a uropod or trailing edge where high glycosylated receptors accumulate (5, 6). We deemed it unlikely that this dramatic reorganization served to exclusively generate a front-to-back axis for directional movement, and we explored the possibility that neutrophil polarization functions as an additional checkpoint during inflammation.
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