to give a nucleus with 114 protons and 173 neutrons, which falls into slightly deeper water off the island. So far there have been two events. But the beauty of this experiment is that the decay properties of the new isotope could be calculated from the observed properties of the first isotope of element 114. The prediction of a single α-decay followed by spontaneous fission is confirmed convincingly[3].

In the latest LBNL experiment[2], the previous philosophy of using a ^{208}Pb target was adopted. This nuclide has a closed shell of neutrons ($N = 126$) and of protons ($Z = 82$) producing an unusually stable nucleus (see Box 1). The extra binding energy of this double-closed-shell system leads to a cooler compound nucleus that needs to evaporate only one neutron to survive fission. A projectile with 36 protons (^{86}Kr) enabled the group to leapfrog element 114 and reach $Z = 118$ (seen in three events), which then decays to $Z = 116$, 114 and so on. In terms of the question "Just how many stable elements is it possible to make?", this increases the maximum atomic number by a further four units. But, for the isotope of $Z = 114$ observed in the long Dubna decay chain (Fig. 2), the lifetime is over four orders of magnitude greater than that for the $Z = 114$ isotope in the LBNL chain. Similar comparisons hold for $Z = 112$ and $Z = 110$. Even the 114 isotope in the short Dubna decay chain (Fig. 2) has a lifetime enhanced by over three orders of magnitude. So, in terms of reaching the island of stability, the Dubna experiments appear so far to be the closest. □

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Behavioural ecology

Electrifying diversity

Michael J. Ryan

Sexual selection often favours signals that are conspicuous; the more conspicuous the signal, the more attractive the signaller is to mates[1]. But conspicuousness incurs a cost because predators can eavesdrop; the more conspicuous the signal, the more risky it is. So natural selection generated by predation is often viewed as a constraining force in signal evolution. In the face of predation, for example, crickets evolve non-calling strategies, guppies evolve dull colours, and túngara frogs produce less conspicuous calls[1]. In a study described on page 254 of this issue[2], however, Philip Stoddard argues that predation by electrolocating predators on gymnotiform electric fish has been a creative rather than a constraining force on the evolution of electric-organ discharges (EODs).

There are more than 100 species of gymnotiforms, or knife fish, in the fresh waters of Central and South America. These animals are nocturnal, and often live in murky waters where visual communication would be difficult even in the daytime. But poor visibility has little effect on them, as they rely on their ability to produce and detect weak electrical fields for sensing their environment as well as for communicating with one another. The EODs, however, make the fish vulnerable to electropredators such as electric eels and catfish.

It seems that the ancestral EOD was an intermittent monophasic pulse. But many gymnotiforms produce a biphasic EOD. What forces might favour the evolution of such a signal? Stoddard addresses three alternative hypotheses: electrolocation, sexual selection and predator avoidance. He argues that predator avoidance was the initial force favouring the evolution of the complex EODs, although sexual selection might have then been responsible for the subsequent sexual dimorphism in the EOD of some gymnotiforms.

Stoddard presents both correlational and experimental evidence that predation can favour the use of biphasic signals. One can change a monophasic EOD to a biphasic one by adding a negative-going second phase, which shifts the frequency upwards (Fig. 1). All electrolocating fish have ampullary organs, which are extremely sensitive to low frequencies and excel at detecting the weak electric fields of prey. But these receptors are less sensitive to the higher frequencies of the biphasic EODs that many gymnotiforms use in communication (Fig. 1). Gymnotiforms also evolved a second set of electroreceptors, tuberous organs, which are less sensitive than ampullary organs but are tuned to the higher frequency of the EOD. So both the signal and the receiver in the gymnotiform communication system operate in a frequency range above that to which ampullary organs are most sensitive. It would seem that such a frequency shift would reduce predation risk.

To test this hypothesis, Stoddard and his graduate student Marina Olman trained an electric eel, a predator, to respond to electrical discharges; the reward was associated with approach to either a monophasic or biphasic EOD. The predator was more likely to approach the monophasic pulse. These results suggest that there is an advantage to producing biphasic pulses, but do not

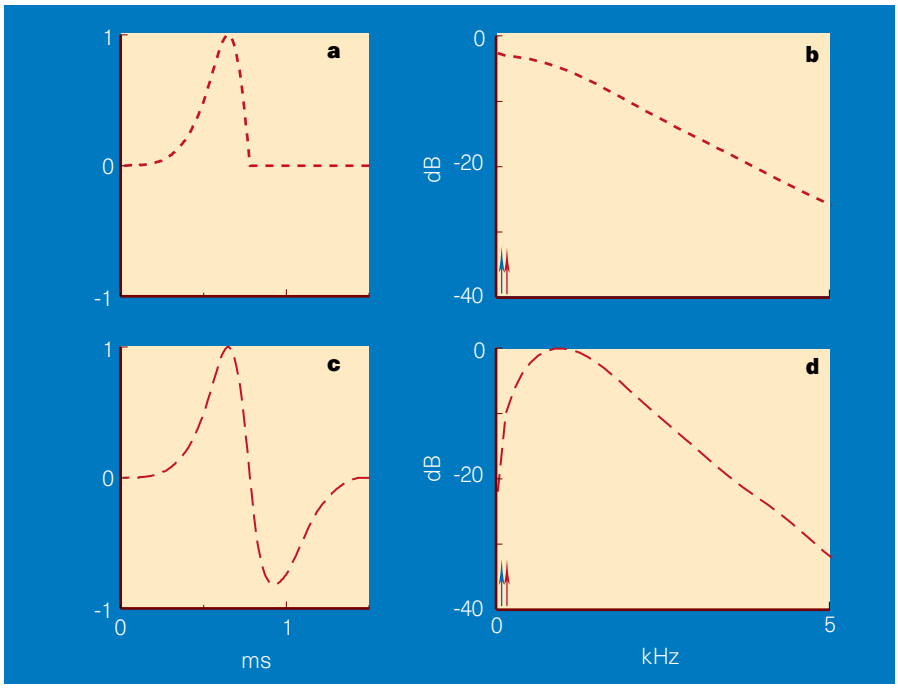

Figure 1 **Signalling in electric fish. The waveform of a monophasic** (a) **and biphasic** (c) **electric-organ discharge (EOD) with the corresponding power spectra (monophasic,** b**; biphasic,** d**). The arrows show the sensitivity of two types of electrosensitive predators, catfish at about 8 Hz (blue) and gymnotiforms at about 30 Hz (red). The biphasic EOD is from *Brachypopomus pinnicaudatus*, gymnotiform prey; the monophasic waveform is that same waveform with the negative component deleted. The figures are modified from Figs 3c and 3d of Stoddard[2], who argues that predation has favoured an increase in the complexity of EODs because the biphasic signals are less detectable by predators than are the monophasic signals.**

explain why three species of gymnotiform still produce monophasic EODs. These, Stoddard argues, might be the exceptions that prove the rule. Of the three monophasic species, one is an electropredator, one lives in an area devoid of electropredators, and one produces an EOD which resembles that of an electric eel.

This study describes a convincing way in which predation may have promoted rather than constrained signal diversity. But it is not a unique case, as predation is known to have had similar effects in other systems. It caused moths to evolve ears to detect, and signals to deter, bat predation; these traits were then co-opted for communication, resulting in males that communicate their presence to females with ultrasonics (in some species, males and females even conduct an ultrasonic duet)[3]. Also, it has been suggested that offspring might evolve signals that are more conspicuous to predators in order to manipulate parental behaviour[4]; bird nestlings giving loud, conspicuous begging calls, and children holding their breath in parental defiance, are two examples.

Stoddard's study gives us a glimpse into the complicated world of signal evolution of one system; a more general understanding, however, is far off. Consider Zuk and Kolluru's[5] review of the predation costs of sexual signals. They pointed out that there are many more examples of predators attracted to signals in the acoustic mode than in the visual mode (electrical communication is more similar to acoustic than visual communication). This bias is probably because it is easier to study acoustic than visual communication. Nevertheless, it is crucial to know how predator effects on communication systems might vary among sensory modalities.

For example, is it as likely that potential prey can evolve electrical (say, biphasic pulses) or acoustic (say ultra- or subsonic) signals out of the range of their predators than it is for prey to evolve visual signals (say in the ultraviolet) to escape predators in that modality? One part of the answer might depend on the lability of signals in a given modality. Another must derive from the predator's receiving systems. Do the demands of communication on a sensory system constrain the uses of that system in other tasks, and does this vary among sensory modalities? For example, does tuning an electroreceptive or auditory system to one type of signal, a weak electric field or an echolocation call, constrain this system from being used to locate prey making very different types of sounds? It might be so in electric eels, but appears not to be in frog-eating bats[6].

Also, more generally, we can ask how constrained different sensory systems are in their ability to evolve. Can we compare the changes in the inner ear of a vertebrate, needed to allow that animal access to ultrasonic emissions made by its prey, to changes in the retina of the same animal that would give it access to ultraviolet signals of different prey? Probably not. Only studies of the entire biology of communication systems can allow an appreciation of their diversity — an argument that provides strong support for the kind of integrative approach taken by Stoddard. □

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Signal transduction

Neither straight nor narrow

Mark Peifer

Back when I was a boy, things were simpler. Web browsers fancied spiders, cell phones were used by prisoners to call their lawyers, and signal transduction — the mechanism by which information is transferred from the surface of a cell to its nucleus — proceeded though a linear series of simple steps. But times have changed, and five papers in *Nature*[1–5] (the latest of which are found on pages 271, 276 and 281 of this issue) provide a dramatic example of this. The discovery of new components and connections is converting the stepwise, linear pathways of information transfer into increasingly complex networks.

Most of our cells do not act in isolation — rather, they respond to signals from their neighbours. Much of the cellular machinery is devoted to receiving these signals and then transducing them, relaying information to the components that mediate the cell's response. In the simplest view, these signal-transduction pathways are envisaged as linear wiring diagrams, like telephone lines that link individual customers to the phone company.

The decisions that cells make are dominated by just a few families of cell–cell signals. Among these are members of the Wingless signal-transduction pathway, inappropriate activation of which contributes to human cancers. (On a lighter note, this pathway may also offer a cure for baldness[6].) The genes required for Wingless signalling were first identified in the fruitfly *Drosophila melanogaster* (and, for simplicity, only the *Drosophila* names of components in the pathway are mentioned here). The pathway initially seemed simple and linear (Fig. 1; and reviewed in ref. 7). A protein called Armadillo is the key regulated component. Normally, Armadillo is unstable inside cells because it is targeted for destruction by a kinase known as Zeste-white3 (Zw3). This kinase is somehow counteracted by another protein, Dishevelled, which is activated when Wingless binds at the cell surface. The receptors for Wingless signals are members of the Frizzled protein family, distant relatives of guanine-nucleotide-binding (G)-protein coupled receptors. So, when Wingless binds to Frizzled receptors, Dishevelled is activated, somehow counteracting Zw3 and stabilizing Armadillo. Armadillo then enters the nucleus, where it interacts with transcription factors and activates Wingless-responsive genes.

Figure 1 **Our humble beginnings — early ideas about the Wingless signalling pathway. Initially this pathway was thought to be a simple, linear cascade. Binding of Wingless to its receptor leads to activation of Dishevelled, which counteracts the repressive effects of the Zeste-white3 (Zw3) kinase and allows expression of Armadillo. After crossing into the nucleus, Armadillo can then activate Wingless-responsive genes.**

But this simple, linear picture has changed dramatically — additional regulatory inputs have been discovered at each level in the pathway. There are, for example, many receptors in the Frizzled family, as well as many Wingless ligands, and we are only just beginning to discover how these proteins can be mixed and matched. Moreover, secreted antagonists of Wingless (a subset of which are secreted analogues of Frizzled receptors), negatively regulate signalling (reviewed in ref. 7). The papers by Tsuda *et al.*[2] and Lin and Perrimon[3] further complicate the issue, because they suggest that the Wingless receptor is, in fact, a protein complex, with