survival, and the use of drive mechanisms to achieve persistence of the construct in target populations. Characteristics of the accessible ecosystem (that is, the ecosystem receiving a GEO or engineered construct) must also be considered in evaluating the safety of release. For example, the presence of potential wild mates, predators, competitors, prey, suitable open niches, etc. can all accelerate or impede the spread of a GEO. Tragic cascades will move some GEOs (or their constructs, or novel products), through the accessible ecosystem once introduced. The expression of secondary or unintentional traits could significantly alter the behavior of GEOs in the field. Similarly, pleiotropy could produce unintended effects. The ultimate mutability of genetic constructs suggests that GEOs in the environment will change over time, thereby changing the relative risk.

An additional concern lies in the nexus between environmental effects and human health effects. For example, the human food chain cannot be wholly isolated from elements in the larger environment, and novel genes or gene products introduced for purposes other than human consumption could (and will) eventually find their way into the human food supply. The effects of novel proteins on human health and development are largely untested, but at least some novel proteins are likely to confer allergenicity or other negative impacts on human health.

The group recognized that the likelihood of safe, long-term releases of GEOs could diminish as the technology expands and as human need increases. Indeed, safe release will become a shifting target as technology changes to meet need. Risk assessment and management strategies must accommodate these changes if a market can be found for GEOs (or their constructs, or novel products) in the larger environment. The challenge to consumers, policy-makers and scientists alike is to find a balance between what is clearly beneficial and that which is potentially detrimental to human health and the environment. This is a great challenge, and one that can only be met through commensurate amounts of attention and effort from all concerned.

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Reference

Ethology: at 50 and beyond

When a dictionary of ethology describes the field as having ‘wide range, baffling diversity, and fuzzy outlines’, its practitioners are justifiably unamused. And yet there is some truth in this image. The richness of ethological explanations has provided such ample seed for the generation of other disciplines that ethology itself might seem to be in danger of disappearing. When Tinbergen famously posed ethology’s four central questions (cause, function, development and evolutionary history), he framed the research program for many years to come. But, disappointed at the loss of Lorenz’s grand theory of instinct and enticed by the predictive power of gene thinking, interest among ethologists and behavioral ecologists has more recently focused on adaptive function and has largely overcome the bias to identify where significant behavioral variation has often been viewed as a phylogenetic character. Emilia Martins (University of Oregon, Eugene, USA) exemplifies how the ‘naive intuition’ of the classical ethologists has more usefully matured into strategies for inferring the evolution of behavior. Her generalized linear model provides a smorgasbord of realistic microevolutionary models, allowing us to identify where significant behavioral change has occurred, and pose questions concerning ancestral states, the degree of phylogenetic effect, the relationship between traits and environment, and the rate of change. For example, she has proposed that the cradle of ethology. Browsing through the published abstracts verifies that ethology remains one of the broadest-based areas of modern biology, embracing neurobiology, phylogenetic character, embracing neurobiology, phylogenetic effect, the relationship between traits and environment, and the rate of change. For example, she has proposed that the cradle of ethology. Browsing through the published abstracts verifies that ethology remains one of the broadest-based areas of modern biology, embracing neurobiology, phylogenetic effect, the relationship between traits and environment, and the rate of change. For example, she has proposed that the cradle of ethology. Browsing through the published abstracts verifies that ethology remains one of the broadest-based areas of modern biology, embracing neurobiology, phylogenetic effect, the relationship between traits and environment, and the rate of change.

Evolutionary lability
Ethology arose in the service of phylogenetic reconstruction and intraspecific variation has often been viewed as a stumbling block to the use of behavior as a phylogenetic character. Emilia Martins (University of Oregon, Eugene, USA) exemplifies how the ‘naive intuition’ of the classical ethologists has more usefully matured into strategies for inferring the evolution of behavior. Her generalized linear model provides a smorgasbord of realistic microevolutionary models, allowing us to identify where significant behavioral change has occurred, and pose questions concerning ancestral states, the degree of phylogenetic effect, the relationship between traits and environment, and the rate of change. For example, she has proposed that the cradle of ethology. Browsing through the published abstracts verifies that ethology remains one of the broadest-based areas of modern biology, embracing neurobiology, phylogenetic effect, the relationship between traits and environment, and the rate of change. For example, she has proposed that the cradle of ethology. Browsing through the published abstracts verifies that ethology remains one of the broadest-based areas of modern biology, embracing neurobiology, phylogenetic effect, the relationship between traits and environment, and the rate of change. For example, she has proposed that the cradle of ethology. Browsing through the published abstracts verifies that ethology remains one of the broadest-based areas of modern biology, embracing neurobiology, phylogenetic effect, the relationship between traits and environment, and the rate of change.
At least two of these three major QTLs appear to be affecting the response thresholds of individual workers, which determine their roles as foragers (i.e. whether they collect pollen, water, or nectar), and, in the latter case, the preferred sugar concentration of the nectar.

The machinery of behavior

For classical ethologists, species-typical behaviors evolved through natural selection and represented the outward manifestation of inherited hardware. However, contributions explored aspects of the neural mechanisms subserving social behaviors. Firstly, using a combination of pharmacological and intracellular stimulation experiments, Berthold Heldewig (University of Gottingen, Germany) elegantly unraveled the neuronal control of complex courtship displays in gompohere grasshoppers. These involve three patterns of hindleg movements and associated sound signals (i.e. three classical fixed action patterns, or FAPs). He showed that recording from three types of interneurons in the brain reliably elicits just one of the three behaviors. Courtship sequence is therefore controlled by successive activation of the three neuronal types, each constituting a necessary part of the FAPs of stridulatory behavior. Even more surprising, it seems that in contrast to the distributed control seen in many other systems, this stridulatory behavior is hierarchically organized: the motor patterns for leg movement may emanate from the metathoracic ganglion but their production is controlled by the brain. Neural mechanisms subserving more enduring social relationships are the province of Michael Riehle (University of California, Los Angeles, USA). Experimental alteration of serotonergic function showed that this system underlies social effectiveness in vervet monkeys. Males with high serotonin levels show less frequent but more effective aggression, form and maintain more coalitions and social alliances, and are more likely to attain high social status in new groups following dispersal. Moreover, serotonergic system receptor density in the frontal cortex and amygdala correlates strongly with substantial and persistent individual differences in aggression and social rank. Those individuals possessing high receptor densities exhibit less destructive, aggressive behavior over their lifetime, and have an enhanced capacity for cooperative behavior.

The general superiority of the tit-for-tat strategy is well recognized and has become the prototypical cooperative strategy in theoretical and empirical studies of the evolution of cooperation11. However, this model is sometimes counterintuitive to those studying motivational mechanisms. For instance, the more cooperative and larger cognitive capacities did not lead to more intricate patterns of social interactions. While the generality of this mechanism is not yet clear, Hemelrijk’s study challenges ethologists to generate parsimonious hypotheses for animal behavior; studies aiming to explain social complexity must ask what part is explicitly encoded in qualities of individuals (genetic or cognitive) and what part determined by interactions between them.

There was a sense of rejuvenation at this meeting, of a reorientation in ethology stemming from the striking success of the interdisciplinary approaches. It was somewhat surprising, though, that with few exceptions, delegates tended to view behavior purely as a result rather than a potential cause of evolution. Mayr saw behavior as the pacemaker of evolution22 (but provided no data), and Wright developed models23 in which ecological opportunity (read ‘behavioral adaptation’) was the sine qua non of evolutionary change. Recent population genetic models incorporate behavior more explicitly24, but without further empirical efforts from ethologists, the claim that behavior is an evolutionary pacemaker will not survive another 40 years25. If ethology is to fulfill its historical aspirations we must put behavior back at the center of evolution. It is gratifying to recognize that ethologists are providing the means to do just that.

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References
Counting the cost of disease resistance

A well-known fictitious race of predatory alien would always admonish the species they intended to subjugate with the unforgettable phrase: ‘RESISTANCE IS USELESS’. A recent paper in Nature shows that an animal’s ability to mount a robust immunological response to incoming pathogens, while being far from useless, may indeed be less than useful in certain circumstances. Evolu­tionary biologists have suspected for some time that the central observation fuelling these suspicions is that genetic variation exists among conspecics in the capacity to repel or control infection successfully. If resistance is useful, in the sense that it contributes positively towards an individual’s fitness, then why are some genotypes refractory to disease and others congenitally defenceless? Why does natural selection not fix genes conferring resistance throughout animal populations?

There are numerous explanations: for example, large asymmetries between host and parasite generation times may leave hosts ‘lagging’ behind pathogens in coevolutionary arms races. Alternatively, substan­tial genetic variance, for instance due to the effects of dominance, can remain at the limits of artificial selection, and similar constraints might obtain in nature. None­theless, a major possibility is that resist­ance correlates negatively with other important fitness components (a so-called ‘cost’ of resistance). Consequently, so far, so good. The great problem with the cost of resistance model, however, has been a continuing lack of direct evidence identifying the all-important costs themselves. Recent years have seen a quickening of interest in this problem, and now in an elegant experiment with an insect host–parasitoid system, Kraaij­eveld and Godfray add convincingly to a small but expanding body of empirical data in support of the ‘cost’ hypothesis.

Using the parasitoid wasp Asobara tabi­da, a common biological enemy of several D. melanogaster species, the authors selected replicate lines of D. melanogaster for increased resistance to parasitoid at­tack. Ovipositing females of A. tabida lay their eggs in the body cavity of larval flies. The young wasp then develops within its host, ultimately causing its death. Occa­sionally, however, a larval fly successfully contains the intruder within multiple layers of immune cells and deposits a dark pigment upon its surface. If this process, known as melanotic encapsulation, is suc­cessful, the invading parasitoid is de­stroyed and the larval fly can develop to adulthood. The dark melanotic capsule re­ mains visible through the fly’s abdominal wall so that as an adult, a larva that sur­vives parasitoid display is the little black spot like a badge of honour.

Kraaij­eveld and Godfray used these spots as the phenotypic marker in their se­ lection regime, choosing only those flies with a melanotic capsule to parent sub­sequent generations. The response to se­ lection was rapid and substantial. In the original field isolate, c. 5% larvae flies encap­sulated wasp eggs, a figure typical of north­ern European D. melanogaster populations. After eight generations, encapsulation rates in the selected lines exceeded 50%. Aside from confirming the genetic basis of en­ capsulation ability, the magnitude of this response suggests that in wild populations there may be considerable constraints on the evolution of resistance.

The authors then turned their attention to locating possible costs associated with the resistant phenotype. Comparison of a battery of traits between selected and con­ trol lines revealed that in high population densities resistant larvae suffer a signifi­ cant decline in ability to compete for a limited food supply when measured against a genetically marked ‘tester’ strain of D. melanogaster. According to Kraai­­jeveld and Godfray, the population densi­ ties imposed in these competition assays are frequently encountered by developing larvae in the field. By demonstrating a negative genetic correlation between lar­val encapsulation ability and competitive performance, these experiments provide hard evidence of a trade-off between resistance and other components of fitness.

Data pointing to a cost of resistance are now found in a diverse assemblage of host-pathogen systems. The conditional interplay of different genotypes has been demonstrated in the interactions of bac­teria with bacteriophages and with viruses as well as mosquitoes with prote­zoans and nematode parasites. These lat­ ter two results have implications for the successful control of debilitating human diseases – specifically malaria and the tropical diarrheas, in which mosquitoes act as vector. Eradication programmes based on the release of pathogen-resistant vec­ tors to the field could ultimately prove fu­tile if resistant mosquitoes pay too high a fitness cost in the absence of parasites. In plant and vertebrate biology, the genetics of resistance mechanisms have been intensively studied. Our under­standing of the genetics underlying host resistance mechanisms in invertebrates is less impressive, but progress is being made. In the case of mosquito refractori­ ness to Plasmodium sp., both suscep­tibility and resistance respond to selec­tion in the laboratory and, more recently, QTL mapping suggests a complex basis to the mode of inheritance of resistance. For some parasitoid systems at least, the genetic basis of resistance may be much simpler. For example, melanotic encapsu­lation ability of D. melanogaster larvae

honey bees Apis mellifera, Behav. Ecol. Sociobiol. 30, 219–226

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