

because newly arising variation modifies existing organismal blueprints, large differences between taxa imply differences in the kinds and amounts of new variation that can arise. The new variation immediately available to a metazoan population, for example, is obviously different from that immediately available to a single-celled eukaryote population. It follows that the evolvabilities of metazoans and single-celled eukaryotes are probably different at present, at least in the short term. It would be far more interesting, though, to know whether differences in evolvability explain in the first place why some single-celled lineages became metazoans whereas others remained single-celled, and this is a much more difficult problem.

Invoking variability as a retrospective explanation for why one clade has diversified or changed more than another does not rule out the possibility that the clades evolved differently for reasons unrelated to variability. And finding isolated examples of evolutionary novelties related to distinctive variability mechanisms — for example, mutations of major phenotypic effect caused by transposable elements — provides only anecdotal evidence for the importance of such variability mechanisms in evolution. As other commentators on evolvability have noted, there is a need for quantitative, testable predictions concerning evolvability rather than retrospective and anecdotal arguments. Approaches such as computer simulation and long-term experimental evolution may yield some progress in this direction because they allow direct manipulation and assessment of the effects of variability differences on evolution, but even these kinds of approaches may not provide dependable insights into whether and how variability differences have actually affected the evolution of natural populations.

Conclusion

Our knowledge of molecular mechanisms that affect the

origin of variation in populations has grown very rapidly in recent decades; in contrast, our fundamental genetic understanding of natural selection developed before 1950 and has not changed in major ways since then. To some, this historical disjunction suggests that evolutionary theory cannot account for the origin and maintenance of mechanisms affecting variability and is overdue for major revision. It is indeed attractive to suppose that the most important evolutionary feature of organisms — their very capacity to evolve and adapt — is itself an adaptation, but this is probably only true in highly restricted circumstances. Instead, variability is probably most often a byproduct of the messy and intricate ways in which genomes have evolved. And the possibility that incidental differences in variability between populations have caused differences in evolvability with profound consequences for evolutionary history remains an interesting — but largely untested — hypothesis.

Further reading

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Neural basis of time changes during saccades

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Normal vision consists of periods of fixation (around 300 ms) interspersed with rapid eye movements called saccades. Saccades create special problems for the visual system, such as rapid, whole field motion across the retina and changes in the relationship between object positions in space and image positions on the retina [1]. Changes to visual processing occur around the time of saccades to cope with these problems. Two time-related phenomena resulting from this altered processing are perceptual time compression during a saccade and slight post-saccadic time expansion [2]. We show that neurons in visual areas of primate parietal cortex have reduced latencies to visual stimulation at the time of a saccade [3,4]. This observation provides a neural explanation for the time related perceptual changes.

Morrone *et al.* [2] demonstrated time compression of visually presented stimuli (but not of audible clicks) during saccades. They presented successive flashed visual stimuli to people and found that the inter-stimulus interval was underestimated if the flashes were presented slightly before or during a saccade. Observers underestimated a 110 ms interval by up to 60 ms. Interestingly, the precision of time estimations was increased during saccades, and for critical time intervals there was an inversion of time. The inversion was observed by asking subjects to report the temporal order of the flashed bars: observers consistently reported the second flash as

occurring first for inter-flash intervals of 20–75 ms.

We recorded the spiking activity of neurons in the middle temporal (MT) and medial superior temporal (MST) regions of the parietal lobe of macaque cortex in alert behaving animals [3,4]. We recorded the visual responses generated when monkeys made saccades across textured visual stimuli. We measured all saccade metrics and replayed the saccadic-image-motion sequence to the monkeys while they were fixating a spot. This method allowed comparisons between monkey-generated image motion when it moved its eyes (active case) and passive viewing when we moved the stimulus (passive case) (Figure 1A).

For 62 MT neurons, the average response latencies during active and passive stimulation were 30 ± 5 (SD) ms and 67 ± 15 ms, respectively [3,4]. The difference was significant for all cells (t-test, $p < 0.01$). For 42 MST neurons [4], 35 showed response latencies that were significantly shorter in the active case (t-test, $p < 0.01$). The mean latencies were 38 ± 11 ms (active) and 69 ± 18 ms (passive) [4]. The remaining cells showed no latency changes around saccades (active: 79 ± 18 ms; passive: 76 ± 14 ms). Optimized stimulation with moving patterns 50 ms after a saccade produced latencies of 48 ± 10 ms, suggesting latency starts to return to normal soon after saccades.

Our monkey data suggest explanations for the findings of Morrone *et al.* [2]. If two flashed stimuli are presented consecutively 100 ms apart, the neurons will respond to both flashes with the same latency (say 65 ms) so that the inter-response interval remains 100 ms (Figure 1B; red trace). If the second flash is presented at saccade onset the response latency to the first flash will be 65 ms, but that to the second flash will be shorter (say 35 ms); thus, the interval between flash responses will be reduced to 70 ms (Figure 1B; blue trace).

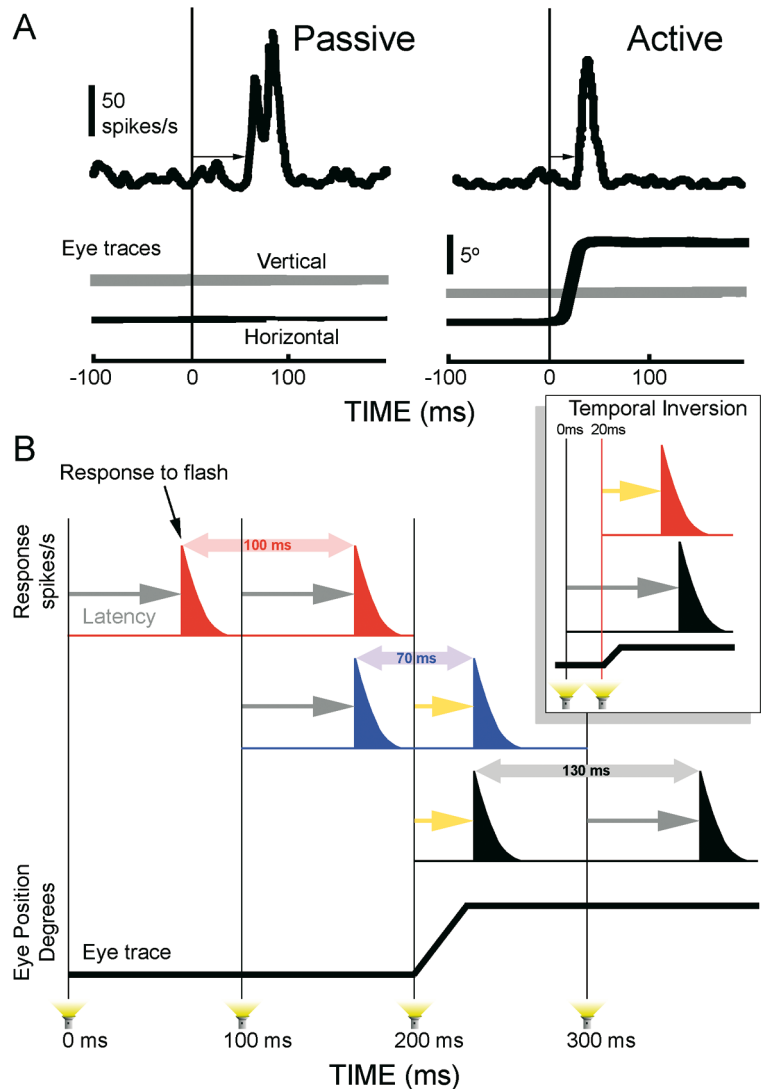


Figure 1. Neural explanation of time compression and inversion during saccades.

(A) Responses of an MT neuron to passive and active stimulation. It responds vigorously to both types of stimulation but the latencies differ (active: 32 ms, passive: 61 ms). The traces show spike rate and eye positions against time. (B) Theoretical flashed stimuli are presented at 100 ms intervals (vertical lines). A saccade begins at the third flash (deviation in eye trace). The cell responds to each flash with a burst of spikes (filled response profiles). The latency to the response is 65 ms (horizontal grey arrows) except at saccade onset where the latency is 35 ms (yellow arrows). For the three flash intervals shown the perceived inter-flash intervals are 100 ms (control; red responses), 70 ms (time compression; blue responses) and 130 ms (time expansion; black responses). The inset panel shows how time inversion could occur for flashed stimuli with an inter-stimulus interval of 20 ms. The response to the second flash (red) arrives before the response to the first flash (black).

Conversely, if the first flash is presented at saccade onset and the second after the saccade, the interval between flash responses will be 130 ms (Figure 1B; black trace). Using the same logic, if the inter-flash interval was 20 ms and the second flash occurred at saccade onset, the response to the first flash would arrive 65

ms after the first flash while the response to the second flash would arrive only 55 ms after the first flash (20 ms interval plus 35 ms latency). Thus, the temporal order of flash arrival will reverse (Figure 1B; inset).

The theory outlined above assumes that a downstream clock remains unaffected by

saccades. Neurons in the higher-order lateral intraparietal area keep track of elapsed time between behavioural events [5]. It is not established whether the time coding in these cells is influenced by saccades; however, our data reveal some MST neurons that do not have saccade-related reductions in latency, thus showing that some neurons in the parietal cortex operate independent of eye movements.

Morrone *et al.* [2] showed that temporal precision was improved around the time of saccades. The standard deviations of the mean latencies for MT/MST neurons in the active case are significantly smaller than those for the passive case (*F* ratio test, $P \ll 0.01$). These data indicate an increase in the precision of response timing during saccades, which could account for the peri-saccadic perceptual improvement in temporal precision.

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Archerfish shots are evolutionarily matched to prey adhesion

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Archerfish are renowned for their unique hunting technique: with a simple blow tube (Figure 1A) they fire precisely aimed jets of water at distant aerial prey to catch their dislodged victims on the water surface [1–3]. The tube is thought capable only of delivering an all-or-none shot of fixed force [4]. But archerfish shoot down an impressive range of different organisms from flies to small lizards [1], can estimate their absolute size [2], and would save energy by tuning their shots accordingly. Studying for the first time the forces transferred to prey, we discovered that archerfish do not fire all-or-none shots but fine-tune their surprisingly costly shots to prey size. This tuning is strikingly lacking of plasticity and innately matched to a constant key property of archerfish feeding ecology: the universal scaling [5] of adhesive forces of their various prey organisms.

By imaging the impact of archerfish shots at frame rates of 5000 s^{-1} (see Supplemental experimental procedures in the Supplemental data available on-line with this issue) we were able to derive for the first time the forces acting on prey and discovered that archerfish transfer systematically larger maximum forces to larger targets (Figure 1B). Strikingly, forces were strictly tuned to target-size even in fish that had grown up in an artificial situation in which we removed all advantages of adjusting force. Under these conditions firing a weak, size-independent shot sufficed to receive a reward of constant nutritional value, regardless which target the fish were firing at. Because of their impressive cognitive performance in other tasks [2,3] we expected the

fish to readily adjust to such conditions and to not tune their force-transfer. Nevertheless, even after two years in this setting, all fish continued to increase their maximum instantaneous forces (Figure 1C, $r^2 = 0.88$, $P < 0.001$) and the total momenta transferred ($r^2 = 0.97$, $P < 0.001$, data not shown) in strict proportion to target size.

This puzzling lack of plasticity could be understood as an evolutionary match to a stable key factor in archerfish hunting: The maximum adhesive forces in animals as diverse as flies and lizards have recently been shown to follow a universal scaling rule [5]. As a consequence of the self-similar structure of their attachment pads, terminal elements occur in a density N_p that universally increases with the animal's mass $m^{2/3}$, and the total adhesive forces increase proportional to $N_p^{1/2}$. Hence, the maximum adhesive forces an archerfish's shot must overcome in order to actually dislodge prey increase linearly with prey's size (i.e. with its linear dimensions or $m^{1/3}$). Archerfish force-scaling closely matches this prediction, ensuring a reasonable safety margin: for any given size of prey, the fish apply about ten times the forces the adhesive organs of prey of that size could maximally sustain (Figure 1C).

Our findings do not support the views that archerfish shooting has been significantly shaped either by components of prey adhesion that are not mediated by specialized organs, or by an attempt of the fish to achieve a mass-independent speed level of its dislodged prey — these would predict force to increase with, respectively, the square or the third power of the prey's size. Moreover, because the first shot hits prey unprepared in an average posture, the fish needed not to adjust to the probably much larger forces some prey might exert by clawing to the substrate.

The evolutionary pressures for adjusting the shots at all, instead of firing an all-or-none shot of sufficient maximum force, became evident when