Guiding the swing in golf putting

Golfers control the pace of a putt by comparing sensory data with an internal guide.

Actions that involve making contact with surfaces often demand perceptual regulation of the impact — for example, of feet with ground when walking or of bat with ball when hitting. Here we investigate how this control of impact is achieved in golf putting, where control of the clubhead motion at ball impact is paramount in ensuring that the ball will travel the required distance. Our results from ten professional golfers indicate that the clubhead motion is spatially scaled and perceptually regulated by coupling it onto an intrinsic guide generated in the nervous system.

Our model of motor control in putting considers two fundamental but largely ignored issues: the role that information gathered through the senses plays in guiding actions, and the control processes used by the nervous system to solve the guidance problem. This model (Box 1, overleaf) is based on a general theory that any guiding mechanism is assumed to be generated in the nervous system. The golfer controls the motion pattern of the forward swing of the putting action by coupling them onto an intrinsic τ guide (Fig. 1b). In self-paced actions like putting, this τ guide is assumed to be generated in the nervous system (for example, by modulating energy levels). Neural mechanisms are implicated in the intrinsic timing of movements and in their spatial specification, and there are similarities in brain activity when the performance of an action is being imagined and when it is subsequently executed.

The golfer controls the motion pattern of the forward swing of the putting action by constantly sensing the τ of the gap between the club and the end of the follow-through, and keeping this τ in constant ratio with an intrinsic τ guide (Box 1, Fig. 1a). Spatially scaling the forward-swing pattern generated by the intrinsic τ guide could be achieved by adjusting one or more of four possible parameters (Box 1): the amplitude, ΔD, of the forward swing (more specifically, ΔD²); the duration, T, of the forward swing (more specifically, 1/T²); the relative time, Tτ, at which the ball is hit; and the τ-coupling constant, k. The first two parameters are the most plausible as they alone are linearly related to the distance the ball will travel (equation (2) in Box 1).

Figure 1 τ-guiding the forward swing. Ten golfers’ club movements were recorded at 200 Hz when putting to four distances, ten times, on an artificial green. a, Motion parameters defined in Box 1. Horizontal distance/velocity ratio for the clubhead during the forward swing was a measure of the Tτ. For each putt, Tτ was linearly regressed onto the intrinsic τ guide (τg) to determine the degree of linearity (r²). b, Relation between the parameters (ΔD², 1/T², Pτ and k) and putt distance, dp, for each of the ten golfers.
Evolution of lifespan in C. elegans

It was proposed almost 50 years ago that ageing is non-adaptive and is the consequence of a decline in the force of natural selection with age. This led to the theory that ageing results from detrimental effects late in life of genes that act beneficially in early life, so any genetic alteration that increases lifespan might be expected to reduce fitness, for example. We show here that a mutation which greatly increases the lifespan of the nematode Caenorhabditis elegans does indeed exhibit a fitness cost, as demonstrated during starvation cycles that may mimic field conditions, thereby validating the pleiotropy theory of ageing.

C. elegans is a soil-dwelling nematode with a facultative, self-fertilizing mode of reproduction. Mutation of the age-1 gene, which encodes a phosphatidylinositol 3-OH kinase catalytic subunit component of the insulin-like signalling pathway, can extend adult lifespan by up to 80% (ref. 4). The age-1 gene, and other genes encoding components of the insulin-like signalling pathway, not only influence ageing, but also control progress of normal development5–7 and determine adult stress resistance4.

If worms carrying the weak mutant allele age-1(hx546) are grown at 27 °C, they develop into dauer larvae (a diapause stage) that does not feed or reproduce, but if they are grown at 20 °C, they develop normally into adults. At this growth temperature, mutant and wild-type worms are essentially identical in appearance, development rates, activity levels and total fertility5–9.

Alleles that confer an extended lifespan but appear otherwise normal are at odds with the pleiotropy theory4–5. We have tested this theory by measuring the relative fitness of the hx546 and wild-type alleles of age-1 in strains that are otherwise isogenic.

We established synchronously ageing populations of hermaphrodite worms, each containing wild-type and age-1(hx546) worms on the same agar plates at 20 °C. As there were no males in these populations, all progeny are the result of self-fertilization. To determine the allele frequency at the age-1 locus in each generation, 100 eggs were removed and shifted to 27 °C, where after 3 days wild-type worms developed into adults and age-1(hx546) worms developed into dauer larvae. The ratio of adults to dauer thus indicated the age-1 allele frequency in the populations. Meanwhile, the populations were maintained by transferring eggs to new plates at 20 °C. When the populations were maintained over 10 generations on agar plates spotted with Escherichia coli as a constant food source, there was no consistent change in allele frequency (Fig. 1a), even when the hx546 allele frequency in the founding populations was varied from 0.1 to 0.9. Thus, there is no evidence of a trade-off between longevity and other life-history traits under these conditions.

This was not the case when mixed populations were maintained under conditions of cyclical starvation. We allowed the worms to exhaust the bacterial food and starved them for four days. Only eggs laid within 24 hours were used to initiate the next cycle. After six starvation cycles, a homogeneous response (G = 7.20, d.f. = 4, P = 0.13) was observed in all populations, with hx546 changing from an initial frequency of 0.50 to a mean of 0.06 (Fig. 1b). Such a large reduction in allele frequency suggests a substantial difference in relative fitness under starvation conditions.

To test which life-cycle stage contributed to the change in allele frequency, we picked starved worms from population plates, allowed them to feed and examined them 12 and 24 h later. Only young adults laid eggs over this period, indicating that the trade-off occurs during this life-cycle stage.

We conclude that the extension of lifespan, by mutation of a single gene, is associated with reduced fitness. This fitness cost is only apparent in an environment thought