schwann-cell precursors, the authors also used *Cre*-based fate mapping in mice. The promoter of the gene for proteolipid protein is selectively active in Schwann cell precursors and Schwannn cells [8], and they used this element to drive expression of a tamoxifen-inducible Cre recombinase. These mice were then crossed to a yellow fluorescent protein (YFP) reporter line. Tamoxifen treatment of these mice results in permanent expression of YFP in Schwann cell precursors and all cells derived from them. They found that, after such treatment, many Schwann cells expressed YFP, as expected, but crucially, they also found that many of the melanocytes associated with hair follicles also expressed YFP. This is strong evidence that melanocytes indeed originate from Schwann cell precursors.

If Schwann cell precursors can generate both Schwann cells and meclanocytes, the question arises as to how this fate choice is controlled. Adameyko *et al.* [3] had noted that the MITF-expressing *Sox10*+ cells were also found in the proximity of the nerve fibres but not in direct contact with these axons. This would seem to suggest that the axons may play a role in directing the fate of Schwann cell precursors. To test this, they axotomised the nerves. They found that loss of the axons resulted in a great increase in the number of MITF+ cells on the operated side. Thus, it would seem that signals from the nerve maintain cells as Schwann cell precursors but in the absence of those signals some Schwann cell precursors will differentiate as melanocytes. Significantly, it was also found that in the adult, mature Schwann cells also retain the ability to differentiate into melanocytes. Some time after transection of the sciatic nerve, melanocytes derived from Schwann cells could be found in the vicinity of the nerve fragment.

Adameyko *et al.* [3] were further able to identify some of the signals emanating from the nerve that could play a role in regulating this fate choice. Neuregulin (NRG1) is a neuronally-derived signal that promotes Schwann cell development [9]. Mice lacking the ErbB3 receptor tyrosine kinase have compromised NRG1 signalling [8], and, although there is an overall reduction in Schwann cell precursors in these animals, there is also a significant increase in MITF+ cells. Furthermore, addition of NRG1 to cell cultures reduced the number of MITF+ cells. However, the addition of other factors to these cultures, such as the growth factors IGF1 and PDGF, which are produced by Schwann cells, could promote an increase in the number of MITF+ cells. Thus, it seems that there are opposing signals that will act to control the balance between Schwann cell and melanocyte differentiation.

This new study [3] is significant as it gives us profound new insights into the origin of melanocytes and more generally neural crest cells. It overturns our previous view that melanocytes were exclusively generated by a distinct population of neural crest cells; those that migrate last, and that these cells were committed to a melanogenic fate just after delamination from the neural tube. Rather it would seem that the process of melanocyte differentiation is more complex and plastic. This study also highlights the importance of Schwann cell precursors as the source of both glial cells and melanocytes. As the authors note, this may help explain the association between alteration in skin pigmentation and neurological disorders, such as is observed in patients with neurofibromatosis type 1.

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Evolution: Exposing the Buried Costs of Reproduction

Investigating the cost of reproduction and terminal reproductive investment is difficult in most species as individuals can respond plastically to most brood manipulations. Experiments in a burying beetle provide new insight into the allocation of resources towards current and future reproductive events.

Robert Brooks and Michael M. Kasumovic

Anybody who has carried a baby to term, helped a child through a bout of gastroenteritis, paid fees for daycare or sat through a school concert can tell you that reproduction is costly. The costliness of reproduction is the central assumption of life-history theory, yet these costs can be incredibly tricky to measure and thus to understand. Reproductive costs are manifested as trade-offs between offspring number and quality, and between current and future reproductive effort, so parents should optimize their investment in reproduction in relation to their age, the resources they have available to invest in reproduction, the environmental conditions they

face and the effort (if any) of their co-parent.

George Williams [1] made two important predictions about the trade-offs between current and future reproductive effort. First, a given amount of reproductive effort at one age should make a parent's future reproductive effort smaller than it otherwise would have been. The difference is the cost of reproduction. Second, as individuals age, the balance of the trade-off between current and future reproduction should tilt toward the current reproductive bout because the probability of future reproductive opportunities decreases. Although both predictions are straightforward, experimental tests that manipulate reproductive effort are difficult. Animals can be manipulated to produce or care for more offspring by physiologically stimulating mothers to produce more eggs [2] or by manipulating the number of eggs in a nest [3]. Although brood manipulation studies alter the number of offspring, parents are forced to lay or care for a manipulated brood that is not of their own making. It becomes impossible to disentangle the effects of the additional reproductive investment from any behavioral reaction to the change in brood size.

It would be ideal if we could manipulate reproductive investment by getting parents to alter the number of offspring themselves, and then alter the costs of this investment without the parents being aware of the manipulation involved. A recent study published in *The American Naturalist* by Creighton *et al.* [4] achieves this seemingly impossible feat by capitalizing on the interesting parental care strategies of a burying beetle (*Nicrophorus orbicollis*). In this species, parents find and bury the carcass of a small vertebrate and the mother lays eggs in the soil nearby. When the larvae hatch, parents regulate their number by cannibalizing some, such that the number of surviving offspring is correlated with carcass size. The carcass is then the only food source for parents and their larvae until the larvae pupate. Once parents produce and regulate the number of offspring through cannibalism, they no longer assess resource value (carcass volume) and manipulations can take place without them realizing it. Examining investment into current and future reproduction thus becomes simple: food the offspring eat is not available to parents and *vice versa*. The amount eaten by the offspring or regurgitated by the mother for the offspring is equivalent to investment in current reproduction, whereas the amount eaten by the mother is equivalent to investment in future reproduction. Such neat partitioning of investment is rare: in most animals it is the soma itself that is invested, and we cannot take before and after measures let alone manipulate resource levels without killing or profoundly disturbing the animal.

Creighton *et al.* [4] allowed females to assess a relatively large (30 g) carcass and reproduce accordingly, while removing males after mating. Once egg laying and cannibalism were complete, they reduced the size of the carcass to 20 g — mimicking a situation in which females overestimated the available resources by 50 percent. This manipulation dramatically decreases the females' number of successful reproductive bouts and lifetime offspring production compared to controls that buried and laid on 30 g or 20 g carcasses that were not subsequently manipulated. Females sacrifice their future reproduction by gaining much less weight on the carcass, and this comes at the expense of their longevity and future brood sizes. This has to be one of the cleanest experimental demonstrations of the costs of reproduction.

As females from all three groups got older, they produced smaller broods, and this effect was most dramatic in the experimental treatment, suggesting that the costs of earlier reproductive bouts begin to catch up with females. These costs could obscure the predicted shift in the balance between current and future reproduction toward terminal investment. The fact that the two control groups gained less weight on each subsequent carcass suggests just such a shift, but both declining weight gain and brood size could be incidental consequences of senescence rather than adaptation. To test the terminal investment hypothesis, Creighton *et al.* [4] compared the reproductive output of young and old first time breeders on large and small carcasses. Late breeders reared more larvae and ate far less of the carcass, as predicted.

Intriguingly the costs and terminal investment effects in this study were so substantial even though all females were fed *ad libitum* between reproductive events. It appears that reproductive costs cannot be alleviated or compensated for simply by eating excess food between reproductive events. The costliness of reproduction may come about through costly provisioning, mothers choosing to eat less, or both. In a related burying beetle, *N. vespilloides*, the time that females spend provisioning offspring by regurgitating pre-digested food is a good predictor of offspring fitness [5], and older mothers spend more time provisioning offspring than younger mothers do [6]. If the same is true in *N. orbicollis* [4], the shift toward greater investment in current reproduction with age may be driven by a shift in the amount of provisioning behaviour.

The burying beetles clearly provide an exceptional experimental system for studying reproductive trade-offs; in addition to the power to experimentally manipulate reproductive costs without parental knowledge, generation times are short and the most important aspects of the field can be duplicated in large samples in the lab. However, this does not mean that studies in other systems are either prohibitive or fruitless endeavors. The fact that Creighton *et al.*'s [4] results are consistent with a long history of brood manipulations in birds [3], for example, suggests that the problem of the manipulation being detected and adjusted for by parents in these studies has not led us to a flawed understanding of reproductive costs. This should be a comfort to empiricists working on reproductive investment in other species where the costs of reproduction can be easily if less directly measured.

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Neurobiology: Fly Gyro-Vision

Flies stay on course using a combination of high performance vision and a specialized sensory gyroscope. A new study reveals that these disparate modalities are wired together.

Mark A. Frye

So you think you can see? Human retinal ganglion cells are roughly predicted to transmit visual information at the equivalent of 10 bits per second [1], which is likely an underestimate of photoreceptor capacity. Foveal photoreceptors may transmit up to 100 bits per second in full daylight, but this performance nevertheless pales in comparison to the 1000 bits per second transmitted by the photoreceptors of a flesh fly [2]. Put another way, the 16 Hz flicker fusion cut-off for human cones in part enables us to be fooled into perceiving smooth motion in movies displayed at 30 frames per second. Yet flies perceive image frequencies well in excess of 100 Hz, and would therefore see our movie as something akin to a slide show.

Though fly visual transduction is the fastest yet measured in any animal, the extreme retinal image speeds achieved during routine flight maneuvers [3] are well beyond those which can be effectively compensated by visuo-motor reflexes [4]. Therefore, like humans, flies reduce the corrupting influence of image blur during locomotion by actively moving their heads to stabilize their gaze [5]. Just like a ballet dancer in a pirouette fixes his gaze on one spot to maintain stability, a fly steering its body into a turn contra-rotates its head to keep the visual world reasonably still [6]. A new study [7] shows that the extreme visual capabilities of flies are due in part to the convergence of multiple sensory modalities upon the control of head posture for stable gaze.

Maintaining stable gaze while chasing down a visual target, such as a territorial invader or potential mate, requires adjusting head posture to fixate the visual world and also to counteract movements of the body. Visual inputs from the compound eyes are segregated into parallel processing pathways specialized to encode patterns of panoramic optic flow generated during self motion [8,9], or small moving targets generated by prey or conspecifics [10]. Body dynamics are encoded by gyroscopic sensory organs called halteres that beat back and forth like the wings and during body rotation generate out of plane reaction forces that are detected by mechanoreceptors at their base [11].

Visual and mechanosensory signals converge on the neck musculoskeletal system to pivot the head [7]. Thus, if a visual target drifts laterally (Figure 1A), visual activation of the neck motor system produces a compensatory head turn (Figure 1B). Similarly, mechanical deflection of the body and haltere sensors by a gust of wind evokes a contra-rotating compensatory head movement (Figure 1C). It would thus appear that the visual and mechanosensory systems are well synchronized for the task of stabilizing gaze.

"Ay, there's the rub": haltere sensory neurons respond to stimulation within microseconds [12], and in turn mediate changes in head postural position within three milliseconds of a sensory disturbance [13]. This behavioral latency is ten times shorter than the activation delay within visual motion processing neurons [14]. The time discrepancy is evident within the very earliest stages of sensory transduction. In contrast to the rapid direct activation of ion channels in mechanoreceptors, photoreceptor signaling in flies uses a comparatively sluggish G-protein signaling cascade. Add to that the

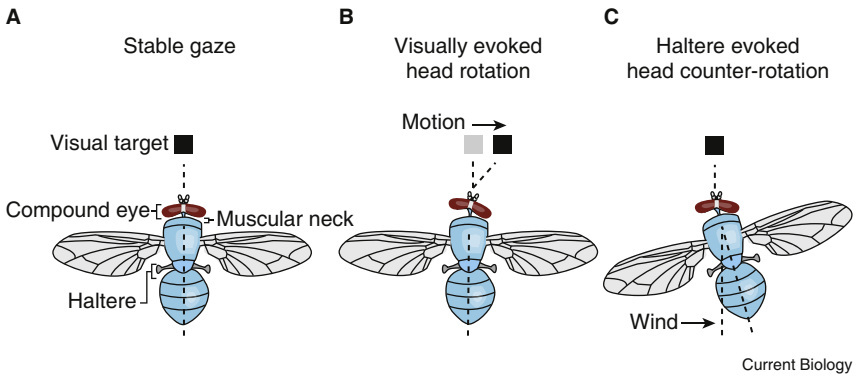

Figure 1. Head movements that stabilize the direction of gaze are evoked by two sensory systems.

(A) A visual target such as another fly is fixated on the forward-looking compound eye. The articulated head is moved by muscles that receive input from the compound eyes and also from gyroscopic sense organs called halteres. (B) Movement of the visual target activates the neck muscles to turn the head and stabilize gaze. (C) A wind gust on the body is detected by the halteres and results in coaxial contra-rotation of the head to stabilize gaze.