ANIMAL BEHAVIOUR

Some begging is actually bragging

A meta-analysis of 143 bird species finds huge variation in parental responses to chicks' begging signals, and shows that parental strategies depend on environmental factors, such as the predictability and quality of food supplies.

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Within just two weeks of its eggs hatching, each songbird parent delivers around 2,000 prey animals to the nest. The food transforms the cold-blooded, naked and tiny hatchlings into warm-blooded, fully feathered flying machines 20 times their original size. Each arrival of a parent at the nest is met with 'begging' signals. But are these signals actually begging, and if not, what do they indicate?

For a quarter of a century, the predominant answer has been that each chick expresses an honest signal of its own need, and parents, thus informed, help weaklings to feed first. However, writing in *Nature Communications*, Caro *et al.*[1] reveal that this may be an oversimplification. They find that, in many species, parents actively disfavour their weakest offspring so that the family food budget covers others adequately. Thus, although the hatchlings of some species may beg, others brag. Offspring signals to parents are regarded as a test paradigm for ideas about animal communication in general, so Caro and colleagues' results may reset the picture.

The signal-of-need model[2] was proposed in 1991 as a provocative and mathematically elegant variant of an earlier argument devised to explain some species' flamboyant sexual advertisements. According to that 'handicap principle', male self-aggrandizement during courtship is curtailed by the costs of the signal (for example, predation risk limits the size of a peacock's fan), such that only the best males can afford to display such finery.

But when applied to begging, this core logic has to be flipped, such that the costly signals are performed mainly by those least able to afford them. This theory assumes that stronger nestmates desist from displaying the signals. Conceding the spotlight in this manner can be explained by kin selection (in which individuals perform behaviours that are costly to themselves but beneficial to relatives), but only if the indicated returns — in this case, extra nieces and nephews — are forthcoming. This assumption of indirect compensation was overlooked as field and laboratory empiricists flocked to study begging. The literature exploded: before the signal-of-need hypothesis was published, a Web of Science search for 'offspring' and 'begging' shows only a handful of papers; from 1992 on, several hundred have been published, most of which support the hypothesis.

When phenomena are studied with only one hypothesis in mind, standards can slide. Offspring 'need' was originally defined in formal evolutionary terms (an individual's prospects

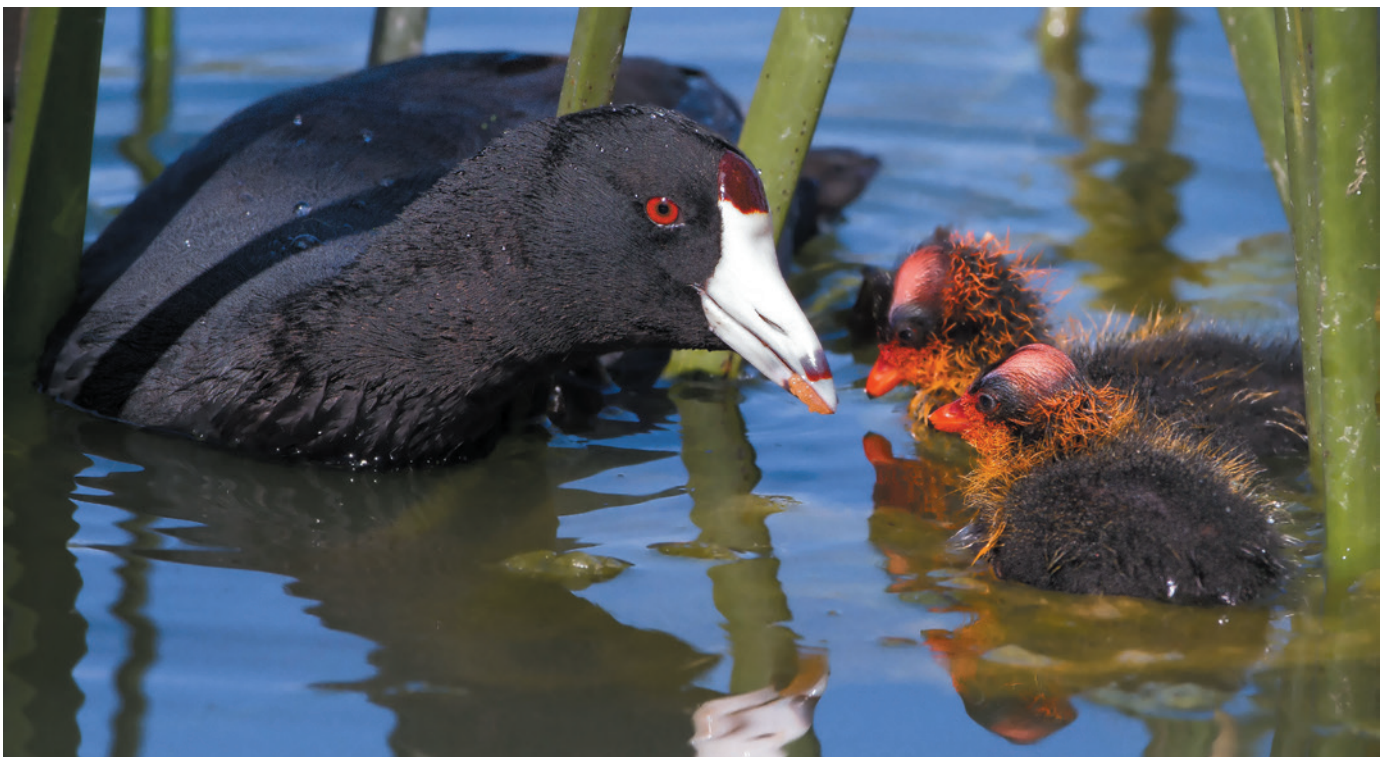

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Figure 1 | Feeding strategies. American coot (*Fulica americana*) parents switch their strategy for feeding their highly mobile and exceptionally colourful chicks midway through the cycle. Initially, parents give food to the nearest chick, which automatically confers a competitive advantage on the larger (earlier-hatched) siblings. But after starvation claims one or two runts, the parents reverse that favouritism — preferentially feeding the weakest survivors and sometimes even attacking the strongest ones[7].

for future reproduction), but such factors are impractical for accurate measurement. Instead, much of the support for signal-of-need came from substituting hunger for need. The problem here is that an offspring can be crammed with food even as it dies of malnutrition, and a robust nestling can be made hungry through brief deprivation[3]. It has been shown for dozens of species[4] that depriving youngsters of food induces escalated begging, but that may not reveal future reproductive potential. Desire is not a synonym for need.

Ironically, the opposite view — 'signal-of-quality', wherein parents generally favour stronger offspring over weaklings — had been proposed a year earlier[5], albeit buried in a long paper. Echoing the advertisement roots of sexual signals, that hypothesis requires no inversion of message and no voluntary abstention. Instead, it proposes that strong offspring are essentially bragging. The signal-of-quality concept also aligns with classic life-history theory[6], in which parents engineer offspring disparities that often facilitate brood reduction, for example by hatching some eggs 1–2 days later than the others. If food availability is unpredictable, competitive mismatches expedite the deferred correction of family size.

Caro *et al.* show that both types of offspring-signalling system may exist in nature, because ecological realities constrain what parents can hope to accomplish. In their meta-analysis, the authors assessed key environmental features and the quality and predictability of food supply for each of 143 species. Variation in environmental quality was scored on the basis of high versus low offspring survival and/or experimental manipulations (additions or subtractions of brood or food), and food predictability was inferred from parental strategies (mainly, whether broods hatched synchronously).

The researchers found that these ecological factors were strongly associated with offspring signalling and within-brood patterns of feeding bias that support two very different parental strategies. If food is relatively predictable, natural selection will favour parents that match family size to the indicated family food budget (creating fewer eggs when food is scarce). In this scenario, survival of the whole brood is the best outcome for everyone, such that a lagging chick should beg more and be fed preferentially, without sibling interference. Conversely, in volatile conditions, parents probably do best by overproducing initially and then pruning later, if necessary, on the basis of offspring size or other physical markers (which devalue the role of behavioural signals). Some species, such as American coots (*Fulica americana*; Fig. 1), actually switch their game mid-cycle, initially letting larger young enjoy their parentally conferred size advantage until brood reduction occurs, and then actively catering to the smallest that remain[7].

By validating pluralism in the explanation for offspring signals, Caro *et al.* encourage further expansion of hypotheses. One to consider is simpler than either signal-of-quality or signal-of-need because it does not require the nestling to possess any 'insider information' about its own long-term prospects, either high (indicating quality) or low (indicating need). Instead, a system could work on the basis of the only 'cryptic' information already known to exist — hunger pangs. In tandem with 'public-domain' cues such as body size, offspring signals might simply answer the mundane but useful question, "Who's ready for another worm?", and thus help parents to make fast allocation decisions. Parents already have knowledge of current food conditions, and for the darker question about who is most expendable, they could rely on visible cues such as size and vigour. This signal-of-hunger hypothesis has strong empirical support[4], and may prove a fine example of Occam's razor — the philosophy that the hypothesis that requires the fewest assumptions is often the most plausible. ■

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CANCER GENOMICS

Hard-to-reach repairs

Two studies find that the molecular machinery that initiates gene transcription prevents repair proteins from accessing DNA, resulting in increased mutation rates at sites of transcription-factor binding. SEE LETTERS P.259 & P.264

EKTA KHURANA

The genetic mutations that lead to cancer are caused by diverse, often poorly understood processes, some of which involve exposure to external agents. Excessive ultraviolet light is linked to melanoma, for example, and tobacco smoke to lung cancer. A molecular mechanism called nucleotide excision repair deals with UV- and smoke-induced genetic damage by removing damaged pieces of DNA, preventing mutations from arising. However, this process is complicated by the fact that repair occurs alongside other crucial genetic activities, such as DNA transcription. Two papers[1,2] in this issue of *Nature* demonstrate how interplay between the DNA-repair and transcription-initiation machinery leads to an increased mutation rate in regulatory regions of the genome.

Although most cancer studies have focused on mutations in protein-coding DNA, there is a growing understanding of the importance of the non-coding DNA regions that regulate gene expression[3–6] — promoter sequences, which are located close to genes, and distant elements called enhancers. Binding of these regions by transcription factors modulates the expression levels of associated genes. On page 264, Sabarinathan *et al.*[1] describe the use of whole-genome sequences from human melanoma samples to analyse mutations in regulatory regions. They found that the cores of the regulatory regions, where transcription factors are predicted to bind, have a mutation rate five times higher than the flanking sequences.

Because of the major role of nucleotide excision repair (NER) in fixing UV-induced DNA damage, Sabarinathan and colleagues next analysed the locations of NER activity[7]. This revealed that the increased mutation rates at transcription-factor binding sites were caused by reduced levels of NER. The authors reasoned that mutations in other cancers that rely on NER should also exhibit this pattern. And indeed, they found increased mutation rates at transcription-factor binding sites in lung-cancer samples, particularly for mutations linked to smoking.

On page 259, Perera *et al.*[2] report the analysis of mutations in regulatory elements in multiple cancer types. They found increased mutation density in the centres of active promoters associated with reduced levels of NER. Moreover, the authors' data suggest that mutation density in regulatory regions is linked not only to transcription-factor binding, but also to the level of transcription initiation.

Thus, two independent studies show that NER at regulatory DNA regions is inhibited by the bound transcription-initiation machinery. This discovery is especially interesting in light of a previous study[8] that showed that