could represent a conserved ground state of αβ-tubulin. Together, these observations add further support to a model in which the role of GTP is to promote assembly by tuning the strength of polymerization contacts (*16*) and/or by decreasing the free-energy difference between straight and curved conformations (*17*).

The regions of curved αβ-tubulin that engage TOG1 move relative to each other in the transition to the straight conformation (Fig. 4A). Thus, TOG1:αβ-tubulin interactions might be sensitive to αβ-tubulin quaternary structure, with a preference for curved αβ-tubulin. If TOG1 binds preferentially to curved αβ-tubulin, it should inhibit in vitro microtubule formation by stabilizing a microtubule-incompatible conformation of αβ-tubulin. We used microtubule-assembly reactions to test this counterintuitive prediction, and we observed strong inhibition when TOG1 was present (Fig. 4B and fig. S8), consistent with earlier observations (*11*). We did not observe inhibition for TOG1 mutants (e.g., W23A or R200A) (fig. S9) that affect αβ-tubulin binding. TOG1 does not bind appreciably to straight αβ-tubulin in preformed microtubules (Fig. 4C), despite the apparent accessibility of the TOG1-interacting epitopes on the outside of the microtubule (fig. S7). Thus, TOG1 binds preferentially to curved αβ-tubulin. We obtained similar results for TOG2 (Fig. 4, B and C), indicating that it also binds preferentially to an αβ-tubulin conformation that cannot exist in the body of the microtubule.

TOG2 binds to GTP- or guanosine diphosphate–bound αβ-tubulin with approximately equal affinity (200 to 300 nM) (fig. S10), supporting a model in which the curvature of unpolymerized αβ-tubulin does not change appreciably as a function of the bound nucleotide. For the "hand-off" to the microtubule to be efficient, the affinity of αβ-tubulin:microtubule interactions must at least be comparable to that of TOG:αβ-tubulin interactions. We used analytical ultracentrifugation to demonstrate that TOG1-TOG2 and αβ-tubulin interact in a manner that is most consistent with a fast interchange between 1:1 and 1:2 TOG1-TOG2:αβ-tubulin complexes (Fig. 4D, red trace). The observation of a TOG1-TOG2:$(\alpha\beta\text{-tubulin})_2$ complex is surprising, because earlier studies (*5*, *7*) suggested that multiple TOG domains could simultaneously engage the same αβ-tubulin. Some of these earlier studies were conducted using a gel-filtration binding assay similar to the one we used, so it is possible that complexes with multiple αβ-tubulins were overlooked [we initially overlooked TOG2:αβ-tubulin interactions for the same reason (fig. S2)]. Our data also show that the complex formed depends on the relative stoichiometry of TOG domains to αβ-tubulin.

We hypothesize that the structure we determined provides a model for substrate recognition in which TOG1 [which is dispensable for plus-end binding (*5*)] of microtubule-bound Stu2p would capture unpolymerized subunits and/or stabilize a collision complex through its relatively strong interactions with naturally curved αβ-tubulin (Fig. 4E). Selective microtubule-end association is presumably the combined effect of a basic region in Stu2p providing microtubule lattice affinity (*5*) and TOG2 preferentially recognizing an end-specific conformation of αβ-tubulin. We speculate based on the polarity of TOG:αβ-tubulin engagement that the ordering of TOGs and the basic region dictates plus-end specificity. For TOG2 and the C-terminal basic domain to jointly mediate plus-end recognition, they must be able to engage the microtubule in a way that allows TOG2 to bind nonstraight αβ-tubulins at the very end and the basic region to simultaneously contact surfaces deeper in the polymer. This can only occur at the plus end. The conformational straightening in αβ-tubulin that accompanies lattice incorporation will result in lower-affinity TOG1 interactions (Fig. 4E). In this hand-off mechanism, polymer incorporation and release of TOG1 for a subsequent round of capture would be intrinsically coupled by virtue of the conformational preferences of TOG1. Hand-off will become efficient only when TOG1 is tethered to free αβ-tubulin binding sites at the end of the microtubule; this explains the requirement for at least two TOGs (*6*), as well as why isolated TOG1 or TOG2 inhibits microtubule assembly despite functioning to promote assembly when part of Stu2p.

Collectively, our observations indicate that Stu2p/XMAP215 family proteins use conformation-selective TOG:αβ-tubulin interactions to discriminate between unpolymerized and polymerized forms of αβ-tubulin. By extension, this result suggests that assembly dependent conformational change in αβ-tubulin plays an important role in dictating microtubule polymerization dynamics.

### References and Notes


1. A. Desai, T. J. Mitchison, *Annu. Rev. Cell Dev. Biol.* **13**, 83 (1997).
2. D. L. Gard, M. W. Kirschner, *J. Cell Biol.* **105**, 2203 (1987).
3. H. Ohkura *et al.*, *EMBO J.* **7**, 1465 (1988).
4. P. J. Wang, T. C. Huffaker, *J. Cell Biol.* **139**, 1271 (1997).
5. J. Al-Bassam, M. van Breugel, S. C. Harrison, A. Hyman, *J. Cell Biol.* **172**, 1009 (2006).
6. P. O. Widlund *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **108**, 2741 (2011).
7. G. J. Brouhard *et al.*, *Cell* **132**, 79 (2008).
8. V. Johnson, P. Ayaz, P. Huddleston, L. M. Rice, *Biochemistry* **50**, 8636 (2011).
9. Materials and methods are available as supplementary materials on *Science* Online.
10. J. Al-Bassam, N. A. Larsen, A. A. Hyman, S. C. Harrison, *Structure* **15**, 355 (2007).
11. K. C. Slep, R. D. Vale, *Mol. Cell* **27**, 976 (2007).
12. K. A. Kosco *et al.*, *Mol. Biol. Cell* **12**, 2870 (2001).
13. R. B. G. Ravelli *et al.*, *Nature* **428**, 198 (2004).
14. H. Aldaz, L. M. Rice, T. Stearns, D. A. Agard, *Nature* **435**, 523 (2005).
15. J. Löwe, H. Li, K. H. Downing, E. Nogales, *J. Mol. Biol.* **313**, 1045 (2001).
16. L. M. Rice, E. A. Montabana, D. A. Agard, *Proc. Natl. Acad. Sci. U.S.A.* **105**, 5378 (2008).
17. R. M. Buey, J. F. Díaz, J. M. Andreu, *Biochemistry* **45**, 5933 (2006).



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### Supplementary Materials

www.sciencemag.org/cgi/content/full/337/6096/857/DC1
Materials and Methods
Figs. S1 to S10
Tables S1 to S4
References (*18*–*27*)


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# A Mechanism of Extreme Growth and Reliable Signaling in Sexually Selected Ornaments and Weapons


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**Many male animals wield ornaments or weapons of exaggerated proportions. We propose that increased cellular sensitivity to signaling through the insulin/insulin-like growth factor (IGF) pathway may be responsible for the extreme growth of these structures. We document how rhinoceros beetle horns, a sexually selected weapon, are more sensitive to nutrition and more responsive to perturbation of the insulin/IGF pathway than other body structures. We then illustrate how enhanced sensitivity to insulin/IGF signaling in a growing ornament or weapon would cause heightened condition sensitivity and increased variability in expression among individuals—critical properties of reliable signals of male quality. The possibility that reliable signaling arises as a by-product of the growth mechanism may explain why trait exaggeration has evolved so many different times in the context of sexual selection.**


The most elaborate male ornaments and weapons of sexual selection grow to exaggerated proportions (Fig. 1), especially in the largest and best-conditioned individuals. The size and conspicuousness of these traits make them likely candidates for intraspecific signals, used either by males to assess the size, condition, or status of rival males, or by females to assess the

relative genetic quality of potential mates (*1*, *2*). Not only are exaggerated traits easy to observe, they are unusually reliable signals of individual male quality (*2*–*4*), as their growth tends to be more sensitive to the nutritional histories and physiological conditions of individuals than is the growth of other traits (*5*–*7*). Exaggerated structures also tend to be more variable in their expression than other morphological structures (*8*–*10*). Hypervariability in trait size can amplify otherwise subtle differences in the body size or condition of males, further enhancing the utility of these traits as signals. Combined, these structural characteristics—extreme size, heightened condition sensitivity, and hypervariability among individuals—are the foundation for "handicap" and "good genes" models of sexual selection and a central tenet of modern theories of sexual selection and animal communication (*2*–*4*, *11*–*15*). We offer a developmental explanation for this phenomenon. We suggest that the evolution of trait exaggeration involves increased sensitivity to insulin/IGF signaling within a growing structure, and we show why such a change in mechanism should also confer both heightened condition sensitivity and hypervariablity to expression of the trait (Fig. 1B).

Insulin and IGFs are essential regulators of tissue growth and body size (*16*). Circulating concentrations of insulin and IGFs are sensitive to nutrition, as well as stress and infection, and the insulin/IGF pathway has emerged as the central mechanism integrating physiological condition with growth in multicellular animal taxa. Insulin and IGF levels within a growing animal reflect the nutritional state and physiological condition of that individual, and circulating concentrations of these signals modulate tissue growth via the insulin receptor pathway in a graded, or dose-dependent, manner. Within an individual, growth will speed up or slow down in response to changes in nutritional or physiological state because of the action of this pathway. Across individuals, growth will differ between high-condition and low-condition individuals, resulting in population-level variation in body and trait sizes. Low-condition individuals have lower levels of these signals than higher-condition individuals, and as a result, they experience slower rates and lower overall amounts of tissue growth.

As long as the various organs and body parts (e.g., legs, eyes, wings) exhibit similar sensitivities to insulin/IGF signaling (*17*), their sizes will scale proportionally from individual to individual (*18*–*21*). But some traits deviate in their responsiveness to these signals, profoundly affecting the amount and nature of their growth. Genitalia are

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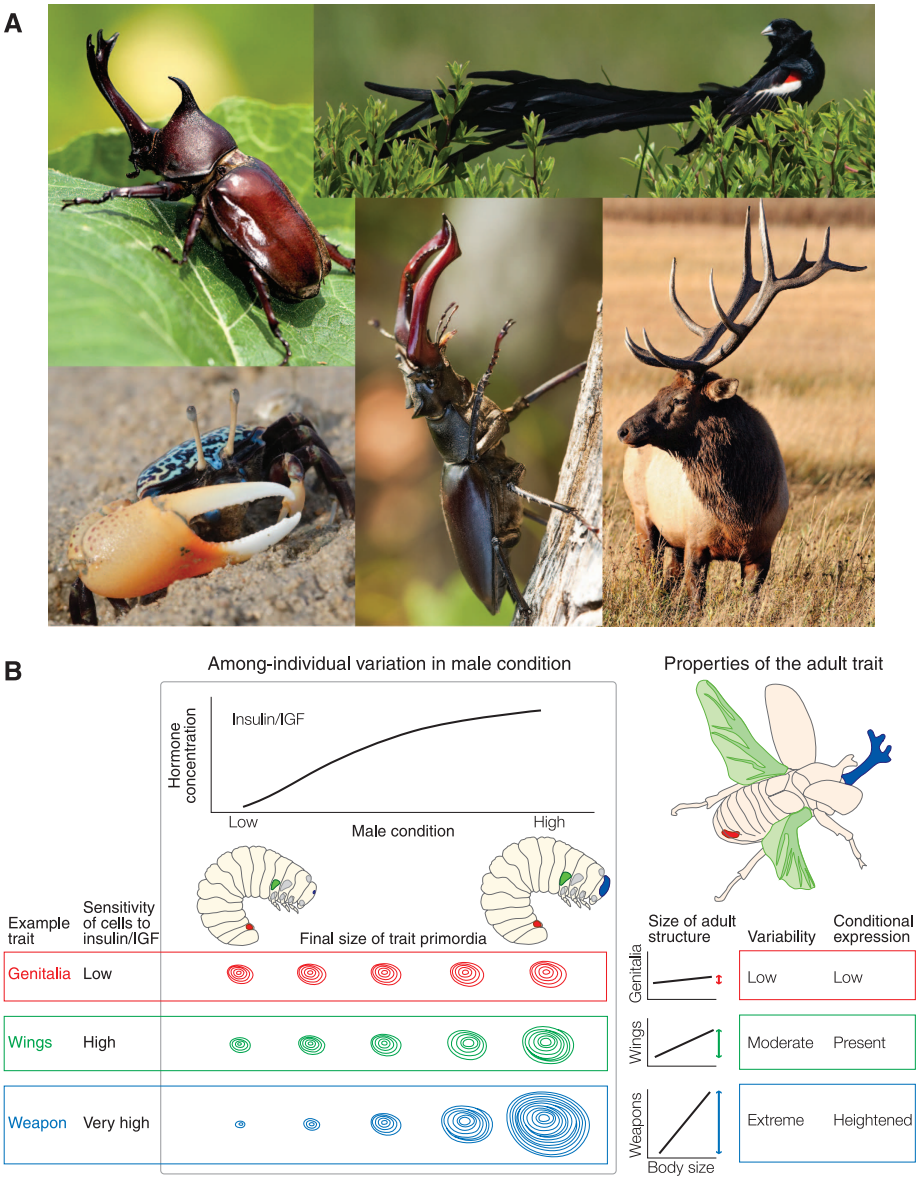


**Fig. 1.** (**A**) Exaggerated growth of weapons and ornaments of sexual selection. Clockwise from top left: Rhinoceros beetle horns (*Trypoxylus dichotomus*); long-tailed widowbird tail (*Euplectes progne*); elk antlers (*Cervus elaphus*); stag beetle mandibles (*Lucanus cervus*); fiddler crab chela (*Uca tetragonon*). (**B**) Proposed mechanism for the evolution of trait exaggeration through increased cellular sensitivity to insulin/IGF signaling (shown for the disc-like appendage primordia of insects). Individual nutritional state and physiological condition are reflected in circulating concentrations of insulin-like peptides and IGFs, which modulate the rate of growth of each of the trait primordia. Traits whose cells are sensitive (*17*) to these signals [e.g., wings (green)] exhibit greater nutrition-dependent phenotypic plasticity and among-individual variability than other traits whose cells are less sensitive to these signals [e.g., genitalia (red)]. An increase in the sensitivity of cells within a particular trait [e.g., horns (blue); see text] would lead to disproportionately rapid growth of that trait in the largest, best-condition individuals (i.e., exaggerated trait size) and smaller trait sizes in low-condition individuals.

insensitive to circulating insulin/IGF signals in *Drosophila* (*20*, *21*). As a result, their growth is unresponsive to environmental conditions, such as nutrition, and genitalia size is largely invariant among individuals. In contrast, wings exhibit sensitivity to insulin/IGF signaling typical of the rest of the body; wing growth is sensitive to larval nutrition, and wing sizes scale isometrically with among-individual variation in body size (*21*).

We predicted that increased sensitivity to the insulin/IGF pathway might be a mechanism leading to the evolution of extreme growth in showy ornaments and weapons of sexual selection. In our model, individual males differ in their physiological state as a result of differences in their status, nutritional state, competitive ability, and/or health (parasite or pathogen loads), which translate into among-individual variation in circulating concentrations of insulin/IGF signals (Fig. 1B). During their respective periods of growth, the adult structures in these animals would be exposed to insulin/IGF signals, and the sensitivity of cells within each growing structure to these signals would determine both how and by how much each trait grew. Just as wings are more sensitive to insulin/IGF signaling than genitalia in *Drosophila* (*20*, *21*), so we predicted that exaggerated ornaments or weapons of sexual selection would be even more sensitive to insulin/IGF signaling than wings or other non–sexually selected body parts (Fig. 1B).

Male rhinoceros beetles (*Trypoxylus dichotomus*) wield a forked horn on their heads. During growth, horns in this species are more sensitive to larval nutrition than other body parts (wings, genitalia), and among adult males, horn size is hypervariable, ranging from tiny bumps to exaggerated structures two-thirds the length of a male's body (*22*). We tested whether growing rhinoceros beetle horns were more sensitive to insulin/IGF signaling than wings or genitalia using RNA interference to perturb transcription of the insulin receptor (*InR*) gene. Developing larvae were injected with a 398–base pair fragment of double-stranded RNA (dsRNA) of *T. dichotomus InR* as they commenced their transition from larval feeding to gut purge (the onset of the prepupal period and the beginning of metamorphosis). At this time, all growth in overall body size had ceased, but adult structures (including genitalia, wings, and horns) were still growing. Thus, any effects of manipulation of insulin/IGF signaling would be visible as reductions to genitalia, wing, or horn size relative to overall body size. If the evolution of exaggerated horn size resulted in part from an increase in cellular sensitivity to insulin/IGF signaling, then horns should be more sensitive than wings to perturbation of the activity of this pathway. We also predicted that genitalia would be relatively insensitive to pathway perturbation [sensu (*20*, *21*)].

Injections significantly reduced *InR* transcript abundances for 48 hours near the end of the period of trait growth (i.e., before *InR* transcript abundance normally drops in these tissues; Fig. 2, A to C). After metamorphosis was completed, we compared morphologies of treated and control animals. Genitalia did not respond to experimental perturbation of *InR* pathway activity (Wald statistic = 0.1245, df = 1, $P = 0.724$; Fig. 2D). Wings, which exhibit nutrition-sensitive growth patterns typical of most metric traits (e.g., eyes, legs, elytra), showed a significant reduction in size of ~ 2% (Wald statistic = 8.976, df = 1, $P = 0.003$; Fig. 2E). In contrast, male horns, the structures most sensitive to nutrition, were reduced by ~16% relative to controls (Wald statistic = 68.37, 1 df, $P < 0.0001$; Fig. 2, F and G). Using response to *InR* knockdown as a metric, we found that male horns were eight times as sensitive to insulin/IGF signaling as wings, consistent with our model for the evolution of disproportionate or exaggerated weapon size from enhanced tissue-specific sensitivity to the insulin/IGF pathway.

A growing body of research now implicates insulin/IGF signaling in the development of extreme animal structures (*23*). Insulin/IGF signaling is an ancient and conserved physiological pathway that has coupled rates of cell proliferation with available nutrients for at least 500 million years, and we suggest that this pathway has been co-opted repeatedly in lineages experiencing strong sexual selection to yield disproportionate growth in signaling structures. The insulin/IGF pathway would likely have controlled the rate of growth of these structures already; increased cellular sensitivity to these signals would therefore be an easy route to the evolution of accelerated growth if the structure came under directional sexual selection for increased size.

But such a route to exaggeration would only generate exaggerated trait sizes in high-condition individuals because low-condition individuals would have low circulating concentrations of insulin/IGF signals and attenuated rates of tissue and body growth. The same mechanism stimulating increased trait growth in high-quality individuals would also repress trait growth in low-quality individuals (Fig. 1B). This means that whenever exaggerated ornament or weapon size arises due to an increase in trait-specific sensitivity to insulin/IGF signaling,

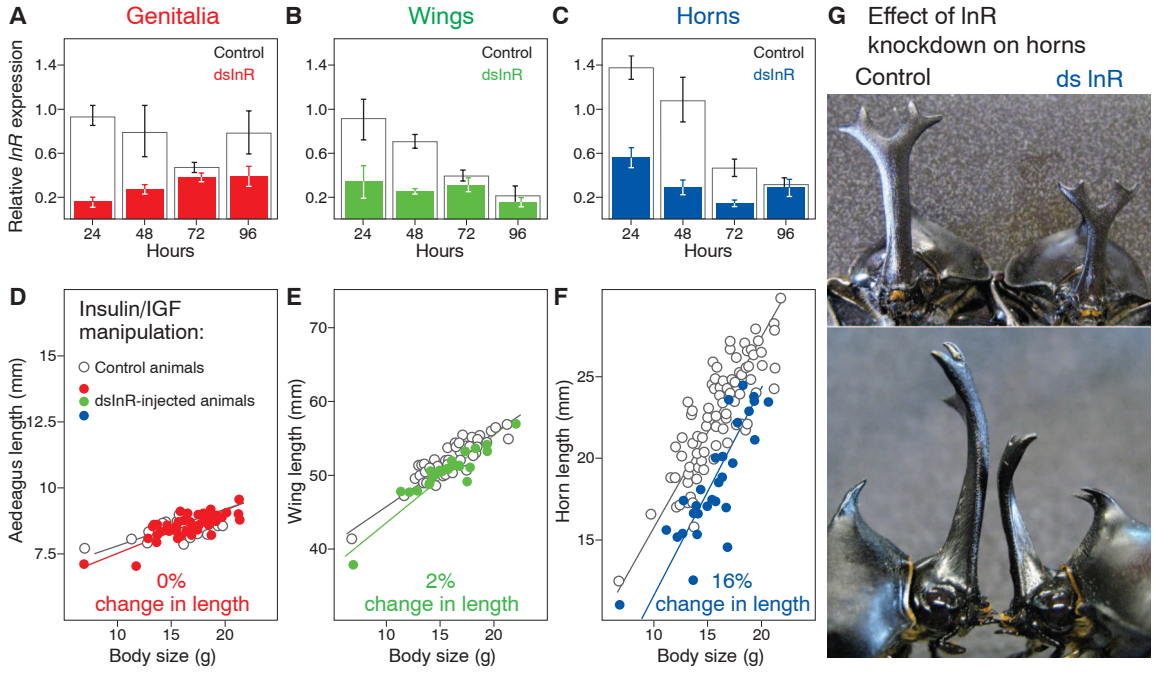


**Fig. 2.** Effect of *insulin receptor* (*InR*) knockdown on growth of adult structures in rhinoceros beetles. (**A** to **C**) Relative transcript abundances for the *insulin receptor* (*InR*) gene in genitalia (A), wings (B), and horns (C), measured 24, 48, 72, and 96 hours after the onset of the prepupal period in control (open bars) and ds*InR*-injected (solid bars) animals. Injection with dsRNA significantly reduced transcript abundances for 48 hours after injection in all three tissues. (**D** to **F**) Effects of ds*InR* knockdown on trait growth. Genitalia were insensitive (D); wings responded significantly but moderately to interrupted insulin/IGF signaling (E) (average reduction in wing length = 2%); and horns responded markedly (F), with an average reduction in horn length of 16%. (**G**) Head and thorax shown in two orientations (top and bottom) for same-sized control (left) and ds*InR*-injected (right) males.

then the exaggerated trait should also show enhanced (or "heightened") condition-sensitive expression and higher relative variability in trait size between low- and high-condition individuals (as compared to other, nonexaggerated, traits). Signal reliability would be an intrinsic property of these structures because of the developmental mechanism regulating their growth.

Theoretical considerations of sexual selection and animal signaling argue that escalated evolution of signals is most likely when signals are reliable, and it is difficult or impossible for low-quality males to "cheat" by producing full-sized structures (Fig. 3). Signal reliability can be evolutionarily stable under two sets of conditions: Either the signal is sufficiently costly to produce or wield that it is not cost-effective for low-quality individuals to cheat ("handicap" signals),

| Category of models: | Model conclusions: | Model predictions: | Contribution of the insulin/IGF mechanism: |
|---|---|---|---|
| **Corroborated by an insulin/IGF mechanism for trait exaggeration:** | | | |
| **Index signal models**<br>Maynard Smith & Harper 2003 (*3*) | Sexually selected traits can evolve to exaggerated sizes if bigger versions of the trait are more effective signals than smaller versions, and if trait size is a physiologically or physically unfakable 'index' of the quality of the signaler. Trait size is a reliable signal because it is mechanistically impossible for low quality signalers to cheat. | Should be mechanistically impossible for low quality males to produce large signal traits. Why the size of a signal trait is unfakable is often unclear/unspecified. | Provides an explicit mechanism for unfakable signal expression in exaggerated morphological structures. |
| **Good genes/indicator models**<br>Andersson 1986 (*26*)<br>Iwasa & Pomiankowski 1999 (*13*)<br>Lorch et al. 2003 (*27*) | Females benefit if they choose mates based on conditionally expressed ornaments because variation in the expression of these traits reliably indicates the overall quality of a male. Low quality males produce smaller signal traits because they are in poor condition. | Exaggerated traits should exhibit 'heightened' conditional expression. | Provides an explicit mechanism for heightened conditional expression of exaggerated morphological structures. |
| **Genic capture models**<br>Rowe & Houle 1996 (*12*)<br>Lorch et al. 2003 (*27*)<br>Tomkins et al. 2004 (*28*) | Evolution of ornaments persists (genetic variation is not depleted) because in their expression these traits "capture" genetic variation for overall body condition, including health, resistance to parasites, competitive ability, nutrition, etc. | Genetic variation among males affecting their body condition, resistance to parasites, competitive ability, etc., should translate into differences in ornament size. | Provides an explicit mechanism for genic capture, since all of these aspects of body condition are channeled into a common endocrine signal regulating trait growth. |
| **Assessment/arms race models**<br>Parker 1974 (*29*)<br>Enquist & Leimar 1983 (*30*) | Male weapons can evolve to exaggerated sizes if weapon size reliably signals the fighting ability of a male. | Males should use relative weapon size as a basis for assessment; fights should be most likely to escalate if rival males are similarly armed. Not clear from the models why weapon size should remain reliable. | Suggests that weapons will become increasingly reliable signals of fighting ability as they increase in size, facilitating arms races. |
| **Modified by an insulin/IGF mechanism for trait exaggeration:** | | | |
| **Handicap models**<br>Zahavi 1975 (*24*)<br>Pomiankowski 1987 (*31*)<br>Grafen 1990 (*25*)<br>Johnstone 1995 (*11*)<br>Iwasa & Pomiankowski 1999 (*13*) | Females benefit if they choose mates based on costly ornaments. A given increase in trait size costs low quality males more (or benefits them less) than it does high quality males, resulting in ornament sizes that reliably signal male quality. | Costs should be present, and they should be relatively highest for low quality males. | Suggests that costs may not be necessary for maintaining signal reliability*, and the handicap principle may only be relevant when exaggeration is extreme. |
| **Allometry evolution models**<br>Bonduriansky & Day 2003 (*32*)<br>Kodric-Brown et al. 2006 (*33*) | Exaggerated ornaments/ weapons will have steep allometry slopes when small males pay higher costs (or derive fewer benefits) than large males for the same increase in trait size. | Costs of ornaments/weapons should trade-off with allocation to overall growth or body maintenance, and these costs should be relatively highest for small males. | Suggests that costs may not be necessary for steep allometry slopes (they should arise as a byproduct of the mechanism of exaggeration)*. This should expand the conditions for which steep allometry slopes are expected. |

*Costs will still work to ensure signal reliability in both cases (i.e., the models are not wrong); however, for insulin-sensitive exaggerated structures, costs may not be necessary. These structures should be intrinsically reliable due to the proximate mechanism regulating their growth.

**Fig. 3.** Sexual selection models whose relevance is affected by the proximate mechanism responsible for trait exaggeration.

or the signal is intrinsically unfakable ("index" signals, "good genes" signals) (*2–4*, *11–13*, *24–33*). The largest ornaments and weapons are generally assumed to be handicap signals of male quality, such that the cost of these structures enforces signal reliability (*2–4*, *24–33*). However, for even the largest structures, the process of escalation must have started when these structures were small, and at that early stage, these costs would likely have been minimal. Moreover, several recent studies of exaggerated male ornaments and weapons have failed to find appreciable costs (*34*, *35*), forcing a reconsideration of the question, why don't low-quality males cheat?

We suggest that exaggerated animal structures may be unfakable signals of quality because of the developmental mechanism responsible for their accelerated growth. If true, then our hypothesis of "intrinsic reliability" could help explain why so many different signal traits embark on an evolutionary trajectory of bigger and bigger size. We suggest that whenever receivers responded to variation in insulin/IGF-sensitive structures, they fared relatively well due to the intrinsic reliability of these traits as signals of underlying male quality. As these traits became larger under selection, their utility as signals would have increased, enhancing the benefits to receivers and accelerating the rate of signal evolution still further. Once these structures become large enough to be costly, they may also act as handicap signals, and costs could contribute to signal reliability (Fig. 3). However, as long as the traits exhibit heightened sensitivity to insulin/IGF signals, costs may not be necessary for signal reliability (*36*). This means that subsequent evolution of compensatory structures alleviating costs to the signaling males (*37*) need not undermine the reliability of these traits as signals and could explain why some exaggerated sexually selected structures function as reliable signals even when no discernable costs are apparent (*34*, *35*).

### References and Notes


1. C. Darwin, *The Descent of Man and Selection in Relation to Sex* (Random House, Modern Library, New York, 1871).
2. J. W. Bradbury, S. L. Vehrencamp, *Principles of Animal Communication* (Sinauer, Sunderland, MA, 2011).
3. J. Maynard Smith, D. Harper, *Animal Signals* (Oxford Univ. Press, Oxford, 2004).
4. W. A. Searcy, S. Nowicki, *The Evolution of Animal Communication: Reliability and Deception in Signaling Systems* (Princeton Univ. Press, Princeton, NJ, 2005).
5. S. Cotton, K. Fowler, A. Pomiankowski, *Evolution* **58**, 1038 (2004).
6. R. Bonduriansky, L. Rowe, *Evolution* **59**, 138 (2005).
7. R. J. Knell, N. Fruhauf, K. A. Norris, *Ecol. Ent.* **24**, 323 (1999).
8. R. V. Alatalo, J. Höglund, A. Lundberg, *Biol. J. Linn. Soc. Lond.* **34**, 363 (1988).
9. S. Fitzpatrick, *Biol. J. Linn. Soc. Lond.* **62**, 145 (1997).
10. J. J. Cuervo, A. P. Møller, *J. Evol. Biol.* **22**, 1503 (2009).
11. R. A. Johnstone, *Biol. Rev. Camb. Philos. Soc.* **70**, 1 (1995).
12. L. Rowe, D. Houle, *Proc. Biol. Sci.* **263**, 1415 (1996).
13. Y. Iwasa, A. Pomiankowski, *J. Theor. Biol.* **200**, 97 (1999).
14. S. Cotton, K. Fowler, A. Pomiankowski, *Proc. Biol. Sci.* **271**, 771 (2004).
15. R. Bonduriansky, *Am. Nat.* **169**, 9 (2007).
16. A more complete description of this pathway and references are provided in the supplementary materials.
17. For this study, we define tissue sensitivity as the extent to which variations in the level of hormone signal influence the rate of cell proliferation via activity of the insulin/IGF pathway. Insensitive tissues grow to roughly the same final size regardless of circulating insulin/IGF concentrations, whereas the amounts of growth of sensitive tissues are strongly regulated by signal levels. Tissue sensitivity is often equated with receptor density. However, in this case, altered expression of any number of downstream genes in the pathway could change the responsiveness of a tissue to insulin/IGF signals. Indeed, in the best-studied example to date, reduced insulin sensitivity in a specific tissue (genitalia) in *Drosophila*, resulted from lowered levels of expression of a "downstream" element of the insulin-signaling pathway, *FOXO*, and not from tissue differences in expression of the insulin receptor (*21*).
18. A. W. Shingleton, W. A. Frankino, T. Flatt, H. F. Nijhout, D. J. Emlen, *Bioessays* **29**, 536 (2007).
19. A. W. Shingleton, C. K. Mirth, P. W. Bates, *Proc. Biol. Sci.* **275**, 1875 (2008).
20. A. W. Shingleton, J. Das, L. Vinicius, D. L. Stern, *PLoS Biol.* **3**, e289 (2005).
21. H. Y. Tang, M. S. B. Smith-Caldas, M. V. Driscoll, S. Salhadar, A. W. Shingleton, *PLoS Genet.* **7**, e1002373 (2011).
22. Results, as well as all methods for this paper, are in the supplementary materials.
23. A description of these studies is in the supplementary materials.
24. A. Zahavi, *J. Theor. Biol.* **53**, 205 (1975).
25. A. Grafen, *J. Theor. Biol.* **144**, 517 (1990).
26. M. Andersson, *Evolution* **40**, 804 (1986).
27. P. D. Lorch, S. Proulx, L. Rowe, T. Day, *Evol. Ecol. Res.* **5**, 867 (2003).
28. J. L. Tomkins, J. Radwan, J. S. Kotiaho, T. Tregenza, *Trends Ecol. Evol.* **19**, 323 (2004).
29. G. A. Parker, *J. Theor. Biol.* **47**, 223 (1974).
30. M. Enquist, O. Leimar, *J. Theor. Biol.* **102**, 387 (1983).
31. A. Pomiankowski, *Proc. R. Soc. Lond. B Biol. Sci.* **231**, 123 (1987).
32. R. Bonduriansky, T. Day, *Evolution* **57**, 2450 (2003).
33. A. Kodric-Brown, R. M. Sibly, J. H. Brown, *Proc. Natl. Acad. Sci. U.S.A.* **103**, 8733 (2006).
34. J. S. Kotiaho, *Biol. Rev. Camb. Philos. Soc.* **76**, 365 (2001).
35. J. F. Husak, J. G. Swallow, *Behaviour* **148**, 1 (2011).
36. In principle, selection on poor-quality males to cheat could lead to evolutionary modifications to the underlying developmental mechanism that buffered expression of the exaggerated trait from the influence of male condition (i.e., that decreased sensitivity to insulin/IGF signals). In this event, the condition sensitivity of trait expression and among-male variability in trait size would decrease (as in male genitalia of these beetles), reducing the reliability of the size of the trait as a signal of male quality. We are aware of no instances in which exaggerated sexually selected signal traits presently display condition insensitivity and/or reduced among-individual variation. This could be because once the traits become exaggerated, their costs reinforce signal honesty and select against cheating males. Or it could reflect the fact that once subsequent insensitivity to insulin/IGF evolves in an exaggerated trait, its reliability as a signal diminishes, favoring receivers who ignore the trait and focus instead on other signals.
37. C. E. Oufiero, T. Garland Jr., *Funct. Ecol.* **21**, 676 (2007).


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### Supplementary Materials

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Materials and Methods
Supplementary Text
Figs. S1 to S3
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Editor's Summary

**Truthful Embellishments**

Exaggerated ornaments such as beetle horns, deer antlers, and extreme tail lengths in birds are typically assumed to be subject to sexual selection because they signal the quality of an individual's breeding status—but how? **Emlen *et al.*** (p. 860, published online 26 July) present a general mechanistic model for the evolution of exaggerated traits, proposing that sensitivity to the insulin response pathway can explain variation among individuals. The exaggerated size of such ornaments and their increased variability between individuals are a result of sexual selection for traits that are honest signals of the fitness of the individual.