prototype is significantly larger than the one based on voice adaptation. Moreover, this relationship was stronger than the relationship between TVA response and the distance to the androgynous prototype. These additional results strengthen the case for the norm-based coding of voice identity in the TVA.

Nonetheless, it is unlikely that norm-based coding and exemplar-based coding accounts are mutually exclusive. For instance, exemplar-based coding might predominate in certain brain regions, or when a prototype has not been formed or is difficult to form. Also if many 'prototypes' exist, norm-based coding is difficult to distinguish from exemplar-based coding.

Glimpse ahead and the 'Mysterious' Prototype

A psychological curiosity is that the brain apparently can form a prototype without ever experiencing it. In a classic study, Posner and Keele [19] presented participants with many dot patterns, which were synthesized by distorting a prototype pattern. Although participants never saw the prototype during an exposure phase, their responses to it afterwards suggested that they had abstracted it, merely by being exposed to the distorted dot patterns.

If the brain does rely on prototypical face or voice representations, even if it has never experienced them, a number of questions arise: How does the brain form and maintain prototypes? In this regard it is important to consider the form of learning; for instance, Andics and colleagues [10] suggest that the brain flexibly shifts the reference point as we become familiar with other people's voices. Also, what is the fidelity with which prototypes represent certain features, and which features, or life experiences, have a greater influence on the formation of prototypes?

No doubt, studying these issues will continue to provide insights into how we so effortlessly identity others and how this process comes to a grinding halt for people with face or voice recognition deficits. We will possibly also better understand how judges like Simon Cowell evaluate exceptional voices/faces, in relation to those that we as a society might not find as mesmerizing as the neurons in our brains.

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Genital Evolution: Cock-a-Doodle-Don't

Losing the penis in species with internal fertilization may seem paradoxical, but birds have managed to do it multiple times. A new study addresses one developmental mechanism responsible for penis reduction in birds, and opens the door to further examination of this little understood evolutionary phenomenon.

Patricia L.R. Brennan

Most amniotes with internal fertilization have a penis, with the exception of most birds [1]. What could have driven the loss of an organ that seems crucial to internal fertilization, and how exactly can a penis be lost? The answer to the first question is still largely unresolved [2,3]. However, in this issue of *Current Biology*, we get an answer to the second question in a paper by Herrera *et al.* [4] that describes a developmental mechanism responsible for the loss of intromittent function in the avian penis.

Only three percent of avian species belonging to two main clades have retained the ancestral copulatory organ: the Paleognathes (e.g., ostriches, kiwis and tinamous), and the Galloanseridae (e.g., chickens, turkeys, megapodes, cracids and ducks) [1]. All other birds have completely lost the penis. Avian penis evolution is complex

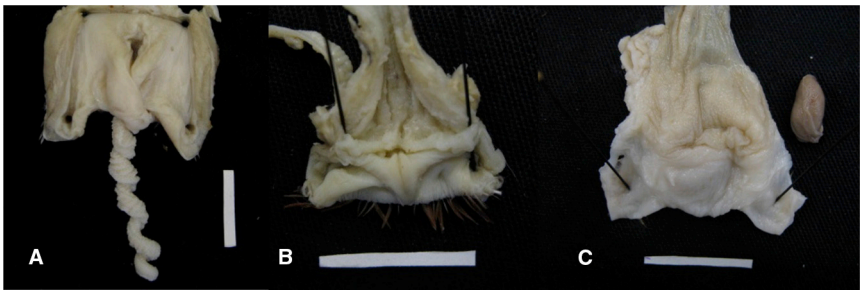

Figure 1. Avian penis diversity.

Avian penises can be intromittent (A; *Rhynchotus*, a tinamou), non-intromittent (B; *Crypturellus*), or completely absent (C; *Leipoa*).

(see Figure 1A in [4]). In addition to the loss of the penis in the ancestor of all Neoaves, birds have experienced multiple reductions of the penis' intromittent function (at least once in tinamous and twice in galliformes) and another complete penis loss (in a megapode) [5]. Herrera *et al.* provide a developmental picture of how birds can transition from a fully intromittent to a non-intromittent penis by examining genital development of chicken and duck embryos. The chicken penis is non-intromittent; and although it swells during copulation and may help to direct the ejaculate inside the female [1], it extends no further than the proximate portion of the vagina. In contrast, the duck penis is a fully intromittent copulatory organ with many remarkable features: it can achieve prodigious lengths [6], sometimes longer than the male himself [7], spirals in a counterclockwise direction [1], and features a unique explosive eversion and insemination mechanism [8].

According to Herrera *et al.*, the dramatically different copulatory organs in chickens and ducks share the same early development. Both begin growing at embryonic stage 26 as paired genital swellings between the anterior and posterior cloacal swellings. At stage 28, the paired swellings merge to form a single tubercle with a sperm channel. The tubercle continues to grow until stage 35 when it stops growing in chicks. By stage 45, the chicken has barely a noticeable bump in the cloaca, while the duck has a well-developed phallus.

An important discovery made by Herrera *et al.* is that the mechanism of external genital development is evolutionarily conserved in birds and mammals. This allowed them to hypothesize that changes in gene regulation of genital growth and patterning that are already known in mammals would explain the differences in morphology of the avian penis.

One possible developmental route for the chicken penis to become non-intromittent would be for it to simply stop growing. Expression of Shh (a morphogenic protein) and Hoxa13 and Hoxd13 (transcription factors), is known to be involved in promoting genital growth in mammals [9,10]; however, their expression was no different between chicken and duck. In addition, cell proliferation response of genital tissues to these signals was similar in both groups. An alternative mechanism is for differential cell-death/apoptosis to occur in the developing chicken penis. Bone morphogenic proteins (Bmps) are known to be involved in apoptosis in mice genitalia [11]. Herrera *et al.* report that indeed a derived pattern of Bmp expression results in reduction of chicken genitalia via induced apoptosis in the distal end of the genital tubercle starting at stage 36. Their study demonstrates that Bmp4 and Bmp7 have different patterns of expression in the genital tubercle of chicken and duck, but Bmp4 appears to be primarily responsible for apoptosis in the distal cells of the chicken genital tubercle. By determining that Bmp4 expression in emu and alligator embryos (both have fully intromittent penises) is virtually identical to ducks, Herrera *et al.* established that Bmp4 expression and apoptosis in the distal genital tubercle evolved in chickens, rather than being lost in ducks. Further, functional experiments using Bmp antagonists rescued the chicken penis from cell death and regression, while activation of Bmp signaling in ducks resulted in apoptosis of their genital tubercle, elegantly demonstrating that Bmp4 expression is both necessary and sufficient to regress growth of the penis.

Bmps are involved in the development of several other organs [12], so their role in genital reduction suggests the possibility that the loss of the intromittent function of the avian penis may have resulted from pleiotropic selection on another organ system, rather than selection for reduced genitalia *per se*. If true, this could explain the seemingly paradoxical loss and regression of genitalia in birds. However, the five independent evolutionary reductions and losses of intromittent penis function have occurred in avian groups that differ greatly in ecology, morphology and behavior. While most tinamous have an intromittent penis (Figure 1A), those in the genus *Crypturellus* have a non-intromittent one that is very different from the non-intromittent chicken penis (Figure 1B) [5]. *Crypturellus* are small ground-dwelling forest birds with exclusive male incubation and parental care, and mating systems ranging from monogamy to promiscuity [13]. Megapodes are large mound incubators from Australasia; they also have a range of mating systems, but in all species males build and defend the mound [14]. Some megapodes have a non-intromittent penis, while in at least one species (*Leipoa ocellata*) it is completely lost (Figure 1C) [5]. The Phasianoidea superfamily (e.g., chicken, quail and turkey) have non-intromittent penises [1], variable body sizes, female incubation and offspring care, and varied mating systems [14]. In the face of these diverse life histories, the pleiotropy hypothesis does not seem plausible, as there is no obvious characteristic shared only by these groups. However, an intriguing possibility is that perhaps the timing of Bmp4 expression in the chicken genital tubercle is dictated by preventing pleiotropic effects of its expression in other organ systems that may be developing earlier and that also rely on Bmps (for example, the limbs) [10,15]. If so, this may explain why some birds end up with non-intromittent penises that do not appear to offer a clear functional advantage, rather than completely losing the penis.

Adaptive explanations of penis loss and reduction in birds include

both natural and sexual selection hypotheses. A reduced penis may increase copulatory efficiency if copulating becomes faster or easier, or it may increase flight performance due to weight reduction if the penis and associated machinery are heavy [2,3]. The penis may have been lost to reduce the risk of acquiring sexually transmitted diseases (STDs) that may be common in birds [2,3]. Female choice may have favored males with reduced genitalia if these males were less able to manipulate reproduction and coerce females [2,3,16]. While we lack sufficient data to rigorously test these hypotheses, preliminary examinations have not yielded universal support for any one of them [2,3,5,16]. However, there is no reason to expect that all the independent penis reduction events in birds are the result of the same selective pressures. Increased copulatory efficiency may have been important for the small *Crypturellus* if shorter copulation — compared to the more prolonged affair in their closest relatives, *Tinamus* — resulted in less predation. Reducing the incidence of STDs may have been important in promiscuous species, such as some megapodes and tinamous. Perhaps the reduction of the penis resulted from female choice for less coercive males in some Galliformes if sexual conflict over forced copulation was as rampant in their last common ancestor as it is in modern waterfowl [6,17].

There may have been more than one ultimate reason why the penis was reduced or lost in birds, and similarly there may be more than one developmental mechanism by which reduction of the penis has occurred. Thanks to Herrera *et al.* we now have one piece of the puzzle, but studying the developmental mechanism of penis loss in Neoaves, and penis reduction in tinamous and megapodes, would help to complete the picture. The diversity of morphologies of bird genitalia suggests that evolution has likely come up with more than one way to lose the penis.

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Evolutionary Genetics: Inheritance of a Complex Pollination Syndrome

How adaptive traits that are controlled by multiple genes evolve is an intriguing question in evolutionary genetics. A recent study shows that tight linkage allows genes that contribute to a multitrait pollination syndrome to be inherited together as a unit.

Kevin M. Wright and Kirsten Bomblies

How do complex, multicomponent traits evolve? Does variation in such traits arise due to changes at single genes or combined effects in many independent loci? In the case of species that can hybridize, do appropriate trait combinations remain associated in the face of gene flow? Understanding the evolution and inheritance of complex adaptive traits, such as the multicharacter floral syndromes that define pollinator interactions in plants, is an intriguing puzzle in evolutionary genetics.

There have been numerous studies of the genetic basis of local adaptation and reproductive isolation among species. Complex adaptive traits have been found in several studies to be multigenic, rather than being caused by variation at single large-effect regulatory genes. In some cases, causal genes map together to inverted genomic regions