Evolution's fast track toward slow flight

Moving slowly can take a toll on anything that flies. A plane traveling at low speed generates little lift and has a hard time staying in the air. Birds, however, evolved a solution to this aerodynamic impasse extremely early in their evolutionary history, according to evidence gleaned from a 115-million-year-old fossil discovered in Spain.

During slow flight, birds make use of a special structure called a bastard wing, or alula, which consists of several feathers attached to the first digit of the wing bones. By moving that digit, a bird can separate the feathers of the alula from the rest of the wing, creating a slot that helps channel air over the flight feathers. The improved airflow enhances lift and prevents the bird from stalling during takeoff and landing.

Because feathers rarely fossilize, scientists have had trouble tracing the evolution of this important flight adaptation. The Spanish discovery, however, features remarkably well preserved alula feathers in their original position, report José L. Sanz of the Universidad Autónoma of Madrid, Luis M. Chiappe of the American Museum of Natural History in New York, and their colleagues.

The paleontologists named the new species *Eoalulavis*, because this goldfinch-sized bird shows the earliest evidence of an alula. They describe the fossil in the Aug. 1 NATURE.

"It's a significant find, and it helps to bridge the gap between *Archaeopteryx* and more modern birds," comments Lawrence M. Witmer, an evolutionary biologist at Ohio University in Athens.

Archaeopteryx, the earliest known bird, lived about 150 million years ago, during the late Jurassic period, and apparently lacked an alula. The Spanish discovery "suggests that some portions of the flight apparatus appeared quite early after *Archaeopteryx*," says Witmer.

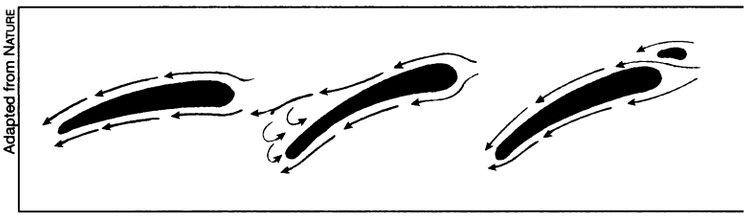
With its long legs, long tail, claws, and teeth, *Archaeopteryx* looked like the small, bipedal dinosaurs from which birds evolved, according to a theory popular among paleontologists. Although *Archaeopteryx* had feathers and could fly, researchers think this early bird was an awkward aerialist at best.

Eoalulavis falls between *Archaeopteryx* and today's birds. "What's particularly interesting is that it retains many of the primitive dinosaurian features that are absent in modern birds, but it has wing and shoulder [adaptations] that are very avian," says Chiappe.

Until recently, the early history of birds has remained obscure, with few fossils filling the interval between *Archaeopteryx* and the late Cretaceous. But discoveries in Argentina, China, Korea, Mongolia, Madagascar, and Spain have given paleontologists a wealth of new fossils from this critical period.

Eoalulavis is the third bird species to come out of Spain's Las Hoyas deposits—the remains of a large lake surrounded by tropical forests. Paleontologists have deduced the diet of *Eoalulavis* from the fragments of crustaceans found in its abdominal region. "We imagine these birds fed in the water or close to the water," says Chiappe. —*R. Monastersky*

When a flying bird (wing shown on left) slows and tilts its wing (center), air passing over the wing separates from the surface and forms eddies. The alula (shown in black on right) channels air over the wing surface.



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Missing protein helps mice to bone up

The vertebrate skeleton plays host to a constant tug-of-war. Bone-forming cells called osteoblasts counter the efforts of their bone-destroying counterparts, the osteoclasts.

Through most of life, this tussle allows vertebrates to remodel and strengthen their skeletons as they grow. Yet if osteoclasts gain a competitive edge over osteoblasts, as can happen in elderly people, bones weaken and diseases such as osteoporosis result.

Working with genetically engineered mice, investigators have now found one of the generals issuing orders to osteoblasts. Osteocalcin, a protein that osteoblasts themselves secrete, appears to inhibit the bone-creating action of the cells, Gerard Karsenty of the University of Texas M.D. Anderson Cancer Center in Houston and his colleagues report in the Aug. 1 NATURE.

The study has surprised bone researchers, because earlier studies had indicated that high concentrations of osteocalcin in the blood paralleled increased activity of osteoblasts.

"These findings are interesting in that they suggest entirely different things than we had suspected before. . . . Osteocalcin might be a negative regulator of bone formation," says Stavros C. Manolagas of the Center for Osteoporosis and Metabolic Bone Diseases at the University of Arkansas for Medical Sciences in Little Rock.

Bone is living tissue formed of cells, including osteoblasts and osteoclasts, enmeshed in an extracellular matrix. A fibrous protein called collagen makes up most of this matrix. Minerals deposited around the collagen give bone its hardness and strength.

Osteocalcin is the most abundant of several noncollagen proteins in the extracellular matrix of bone. In mice, two genes independently encode the protein. To study osteocalcin's function, the researchers made so-called knockout mice. "We deleted the two genes and generated a mouse strain that had no osteocalcin," explains Karsenty.

At birth and at age 3 months, these knockout mice were indistinguishable from normal mice. When checked at 6 months, however, the knockouts displayed a difference: Their bones were denser than those of normal mice. Moreover, their bones proved superior to those of normal mice on a biomechanical stress test. "The bone is of better quality," says Karsenty. "It's the opposite of osteoporosis in many respects."

Since osteoblasts secrete osteocalcin, the researchers originally thought the slow improvement in bone formation stemmed from an inhibition of osteoclast activity by the protein; however, the osteoclasts behaved normally.

Further tests showed that the osteoblasts in the knockout mice were simply more effective. They constructed more mineralized bone than did the osteoblasts in mice that had osteocalcin. "Each osteoblast is laying down more matrix. That's the only abnormality of this mouse," says Karsenty,

To explain the results, Karsenty's group suggests that osteocalcin binds to molecules on the surface of osteoblasts, slowing their bone-forming activity. If so, compounds that thwart osteocalcin might prove useful in treating osteoporosis and other bone diseases, the researchers observe.

That aid may be some time in coming, however. "I don't think we have the cure or explanation for osteoporosis. We just have the function of one protein," cautions Karsenty.

In addition to probing how osteocalcin dampens osteoblast activity, the investigators plan to create more knockout mice to understand the roles of the other noncollagen bone proteins. —*J. Travis*