Researchers have had more success in developing hypotheses about Compositae's evolutionary sequence (phylogeny) by using molecular techniques to study genetic similarities among existing species. These studies initially identified two related families: Goodeniaceae, centered in Australia; and Calyceraceae, which are in southern South America and are closer to Compositae (6, 7). Calyceraceae, a small family of just four genera and about 60 species (8), stands in contrast to the huge sunflower assemblage of 1500 genera. The phylogenetic approach has helped researchers identify what appears to be the oldest known familial relative: Barnadesiinae, a little known subtribe of Mutisieae; it consists of nine genera and 91 species from southern and Andean South America (9, 10).

All data suggested that the common ancestor of Goodeniaceae, Calyceraceae, and Compositae developed in Antarctica, when it had mixed temperate and tropical forests (11, 12). As Antarctica cooled during Eocene-Oligocene (~56 million to 23 million years ago) (13), the ancestral form dispersed and migrated eastward into Australia, resulting eventually in Goodeniaceae, and westward into southern South America, leading to the progenitor of Calyceraceae and Compositae. The splitting of Calyceraceae and Compositae, therefore, would have occurred in southern South America during the Eocene (~56 million to 34 million years ago). These hypotheses have been buttressed by "molecular clock" studies, which suggest that Compositae diverged in the Eocene, approximately 50 million years ago (14).

Given this background, Barreda et al.'s report of an Eocene fossil from southern South America showing clear flowering heads with phyllaries and pappus is important. At long last, there is clear macrofossil evidence of the sunflower family at an early stage of its diversification, just where it had been hypothesized to originate. The fossil does not allow unequivocal assignment, but the authors suggest that its large, conical heads and types of pappus and phyllaries are broadly compatible with Mutisieae. There is also dispersed pollen found in the matrix with the fossil, and its features are also suggestive of Mutisieae (or possibly Carduoideae). Detailed scanning and transmission electron microscopic studies on the pollen would be helpful for deeper understanding of relationships, as would finding pollen in situ in anther sacs of a better preserved fossil.

Much remains to be learned about the evolution and biogeography of the sunflower family. A new book (15) has synthesized molecular phylogenetic studies and, in consort with the new fossil reported here, provides strong stimuli for further research. Even if researchers accept the sunflower's origin in southern South America, it is still unclear how the family quickly colonized the entire planet and became so incredibly diverse.

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GENETICS

A Bit of Texas In Florida

Florida's inbred panthers benefited from the import of Texas pumas.

Craig Packer

Harassed, hunted, and restricted to ever smaller areas, most populations of large carnivores are fragmented into archipelagoes of parks and reserves. Biologists have long warned of the negative genetic consequences of inbreeding in such small populations. To restore genetic health, they have prescribed "active management," including moving, or translocating, individuals into inbred populations. In a time of budget cuts and inadequate funding for effective conservation, however, is translocation worth the costs? Moving a lion from Namibia to South Africa is not a trivial exercise, nor is the translocation of cougars from one part of the United States to another. But it may be worth the trouble, Johnson et al. (1)

report on page XXX of this issue. In the most comprehensive study ever conducted on the effects of inbreeding in wild carnivores, they find convincing evidence that the "quality" of a population of Florida panthers was successfully improved by the addition of panthers from Texas.

Florida panthers (also called cougars, pumas, or mountain lions) have been studied in considerable detail since the 1970s and provide an exceptionally clear example of the genetic consequences of prolonged inbreeding. By the early 1990s, Florida's population of 20 to 25 adult panthers was showing lower genetic variation than other puma populations. Biologists observed a range of problems—including heart defects, poor sperm quality, poor fecundity, and many adult males with one or no descended testes—that led to predictions that the population could go extinct within decades. In a bid to stem the

tide, managers introduced eight female Texas cougars to Florida in 1995.

By comparing genetic data collected from 591 Florida panthers between 1978 and 2009, Johnson et al. show that Texas-Florida hybrid offspring have replaced the original inbred stock. The researchers documented increased levels of genetic heterozygosity (having different versions of the same gene), and the hybrid offspring enjoyed greater viability and fewer genetic abnormalities. The adult hybrids were also superior competitors: The pure-bred Florida panthers suffered greater mortality from fights with outbred cougars, and hybrids were better able to climb trees when pursued by scientists.

The size of the panther population has also increased since the translocation, but this result is more difficult to interpret. The amount of land available to the Florida panther has increased in the past few decades due

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Gene flow. The Florida panther got a genetic boost from introductions of pumas from Texas, but other big cats still face serious problems around the world.

to a variety of conservation measures. More land supports more cougars, so it is hard to estimate how much of the population growth resulted from the influx of "fresh blood" as opposed to range expansion. A similar translocation of 16 lions into a highly inbred population in Hluhluwe iMfolozi Park, a fenced reserve in South Africa, also improved the reproductive performance of the lions in the park, but population size did not increase in the short term (*2*).

Although translocation looks to be an effective technique for ameliorating the genetic consequences of small population size, the larger problem still remains. Big cats may be popular in places where they've become scarce and most people live in cities, but the rest of the world still struggles to deal with the dangers that man-eaters and cattle-killers pose to rural residents. Lions attacked more than 100 Tanzanians every year for the first few years of this millennium (*3*), and thousands of livestock are killed by lions, leopards, and jaguars throughout the world each year.

Retaliatory poisoning is increasingly common in Africa, greatly reducing the number of large carnivores outside national parks, and trophy hunting is excessive and poorly regulated, resulting in rapid population declines in many jurisdictions (*4*). Even in the United States, attitudes toward cougars vary from state to state. Montana paid bounties for dead cougars between 1908 and 1911; the take averaged about 140 animals per year. In contrast, between 1997 and 1999, trophy hunters in Montana killed an average of 800 cougars per year—virtually at the same time as the translocation from Texas to Florida. In 2006, Oregon announced plans to increase trophy hunting in order to decrease the state's cougar population by 40% and thereby reduce livestock depredation.

Although 21st-century Floridians may be willing to enlarge panther habitat, the story is still quite different in the rest of the world. We can perhaps take some consolation from Johnson *et al*.'s study: Once the entire planet reaches the same state of economic development and urbanization as the United States, wildlife managers all over the world can look forward to carting rare species from one park to another until the end of time.

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GENETICS

Exposing A DUX Tale

Mani S. Mahadevan

A DNA sequence stabilizes the expression a gene that may affect muscle development and lead to muscular dystrophy.

Facioscapulohumeral muscular dystrophy (FSHD), the third most common muscular dystrophy, is characterized by progressive weakness that starts in the facial muscles, proceeds to the upper back (scapula) and shoulder-upper arm regions (humeral), and eventually affects the trunk and lower extremities. Since 1992, this disorder has been associated with an array of repeated DNA sequences (called D4Z4) on

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chromosome 4 (*1*). An unaffected chromosome 4 has between 11 to more than 100 repeat units within D4Z4, but when this is shortened to 1 to 10 units, disease develops. How this contraction leads to disease has been a mystery. Over the past three years, analyses of chromosome 4q35 has identified a combination of DNA sequences (haplotype 4A161) associated with susceptibility to FSHD, suggesting that specific sequence variations are coupled to disease pathogenesis in conjunction with D4Z4 contraction (*2*). On page XXXX of this issue, Lem-

mers *et al*. (*3*) provide an intriguing unifying model for FSHD pathogenesis based on very high resolution haplotype mapping and sequence analyses, and careful study of exceptional pedigrees.

FSHD pathogenesis has been one of the most puzzling enigmas in human genetics for the past two decades, but there was always a consensus that the disease was caused by a gain of function mutation (*1*). Each D4Z4 repeat unit has a sequence called *DUX4* that potentially encodes a double homeobox gene putatively involved in developmental regu-