analogous to forward and backward mutation at the individual level; and the intensity of selection for other traits such as oogamy that may cause uniparental inheritance secondarily. None of these parameters is known for any organism.

In fact, all of these factors must be considered in order to achieve a complete explanation for the relative frequencies of uniparental and biparental inheritance of organelle genes. Moreover, the explanation must be sought in a phylogenetic context in which the ancestral state of the organisms can be reconstructed. It may be more fruitful to analyze the evolution of mechanisms of uniparental inheritance (organelle exclusion from the zygote, selective silencing, and so on) as opposed to patterns. Given the complexity of the task, it will almost certainly be necessary to treat one limited group of organisms at a time, and combine them into successively larger trees to achieve more general explanations, or more likely, to show that the evolutionary consequences of uniparental inheritance vary from one group to another, just as the mechanisms vary.

It should be clear from this Primer that uniparental inheritance is a quantitative trait with many different underlying mechanisms; moreover it is potentially subject to any or all of the evolutionary forces of mutation, random genetic drift, and selection within and between species. Unraveling the evolutionary history, causes, and consequences of the trait will almost certainly be much more difficult than we thought, and should provide many years of good scientific fun.

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Barotrauma is a significant cause of bat fatalities at wind turbines

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Bird fatalities at some wind energy facilities around the world have been documented for decades, but the issue of bat fatalities at such facilities — primarily involving migratory species during autumn migration — has been raised relatively recently [1,2]. Given that echolocating bats detect moving objects better than stationary ones [3], their relatively high fatality rate is perplexing, and numerous explanations have been proposed [1]. The decompression hypothesis proposes that bats are killed by barotrauma caused by rapid air-pressure reduction near moving turbine blades [1,4,5]. Barotrauma involves tissue damage to air-containing structures caused by rapid or excessive pressure change; pulmonary barotrauma is lung damage due to expansion of air in the lungs that is not accommodated by exhalation. We report here the first evidence that barotrauma is the cause of death in a high proportion of bats found at wind energy facilities. We found that 90% of bat fatalities involved internal haemorrhaging consistent with barotrauma, and that direct contact with turbine blades only accounted for about half of the fatalities. Air pressure change at turbine blades is an undetectable hazard and helps explain high bat fatality rates. We suggest that one reason why there are fewer bird than bat fatalities is that the unique respiratory anatomy of birds is less susceptible to barotrauma than that of mammals.

As with any airfoil, moving wind-turbine blades create zones of low pressure as the air flows over them. Animals entering these low pressure areas may suffer barotrauma. To test the decompression hypothesis, we collected hoary (*Lasiurus cinereus*) and silver-haired bats (*Lasionycteris noctivagans)* killed at a wind energy facility in south-western Alberta, Canada, and examined them for external and internal injuries.

Of 188 bats killed at turbines the previous night, 87 had no external injury that would have been fatal, for example broken wings or lacerations (Table 1). Of 75 fresh bats we necropsied in the field, 32 had obvious external injuries, but 69 had haemorrhaging in the thoracic and/or abdominal cavities (Table 1). Twenty-six (34%) individuals had internal haemorrhaging and external injuries, whereas 43 (57%) had internal haemorrhaging but no external injuries. Only six (8%) bats had an external injury but no internal haemorrhaging.

Among 18 carcasses examined with a dissecting microscope, ten had traumatic injuries. Eleven bats had a haemothorax, seven of which could not be explained by a traumatic event. Ten bats had small bullae — air-filled bubbles caused by rupture of alveolar walls — visible on the lung surface (Figure 1A). All 17 bats examined histologically had lesions in the lungs consistent with barotrauma (Table 1), with pulmonary haemorrhage, congestion, edema, lung collapse and bullae being present in various proportions (Figure 1). In 15 (88%), the main lesion was pulmonary haemorrhage, which in most cases was most severe around the bronchi and large vessels.

Although the pressure reduction required to cause the type of internal injuries we observed in bats is unknown, pressure differences as small as 4.4 kPa are lethal to Norway rats (*Rattus norvegicus*) [6]. The greatest pressure differential at wind turbines occurs in the blade-tip vortices which, as with airplane wings, are shed downwind from the tips of the moving blades [7]. The pressure drop in the vortex increases with tip speed, which in modern turbines turning at top speed varies from 55 to 80 m/s. This results in pressure drops in the range of 5–10 kPa (P. Moriarty, personal communication), levels sufficient to cause serious damage to various mammals [6].

Barotrauma helps explain the high fatality rates of bats at some

Table 1. Injuries observed in bats killed at wind turbines in south-western Alberta, Canada.

	L. cinereus	*L. noctivagans*	*Other species*	*Total*
No external injury	38% (103)	55% (77)	75% (8)	46% (188)
Internal haemorrhage	90% (48)	96% (26)	100% (1)	92% (75)
Pulmonary lesions	100% (6)	100% (8)	100% (3)	100% (17)

Internal haemorrhage was detected by visual examination of dissected carcasses, while pulmonary lesions were detected using stained histological sections. Numbers in parentheses are sample sizes.

wind energy facilities. Even if echolocation allows bats to detect and avoid turbine blades, they may be incapacitated or killed by internal injuries caused by rapid pressure reductions they can not detect.

Birds are also killed at wind turbines, but at most wind energy facilities fewer birds than bats are killed [8], and barotrauma has not been suggested as a cause of bird fatalities. This may be explained partly by differences in the respiratory anatomy and susceptibility to barotrauma of birds and bats. Bats have large lungs and hearts, high blood oxygen-carrying capacity, and blood-gas barriers thinner than those of terrestrial mammals [9]. These flight adaptations suggest that bats are particularly susceptible to barotrauma. Although birds have even thinner blood-gas barriers, they have compact, rigid lungs with unidirectional ventilation and a cross-current blood-gas relationship, as opposed to mammals which have large pliable lungs with the blood-gas relationship in a uniform pool in the pulmonary alveoli [9,10]. In addition, the pulmonary capillaries of birds are exceptionally strong compared to those of mammals, and do not change as much in diameter when exposed to extreme pressure changes [10]. Bats' large pliable lungs expand when exposed to a sudden drop in pressure, causing tissue damage, whereas birds' compact, rigid lungs do not.

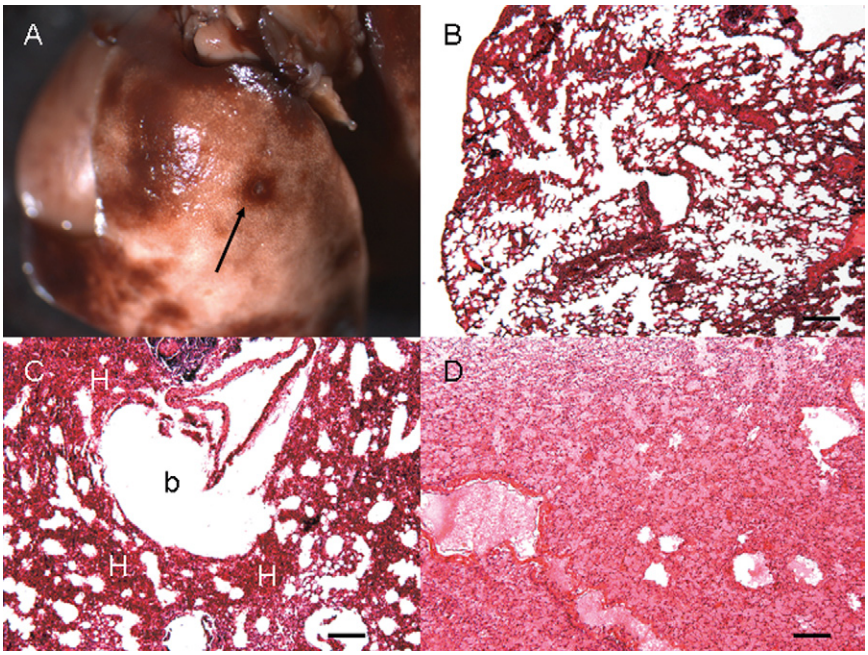

Figure 1. Pulmonary barotrauma in bats killed at wind turbines.

(A) Formalin-fixed *L. noctivagans* lung with multifocal hemorrhages and a ruptured bulla with hemorrhagic border (arrow). Histological sections of bat lungs stained with hematoxylin and eosin (100X). (B) Normal lung of an *L. noctivagans*. (C) Lung of *Eptesicus fuscus* found dead at a wind turbine with no traumatic injury. There is extensive pulmonary hemorrhage (H), congestion, and bullae (b). (D) Lung of *L. cinereus* found dead at a wind turbine with a fracture of the distal ulna and radius. 90% of the alveoli and airways are filled with edema. Bar = 100 μm.

Supplemental data

Supplemental data are available at http://www.current-biology.com/cgi/content/full/18/16/R695/DC1

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