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Point:Counterpoint: High altitude is/is not for the birds

POINT: HIGH ALTITUDE IS FOR THE BIRDS!

Birds arose from their dinosaur ancestors and took to the skies over 100 million years ago. Since that time, birds have become abundant at high elevation, they have acquired the ability to migrate over the world's highest mountains, and they have even found reason to soar over 11,000 m above sea level (18). These anecdotal observations alone may convince some of an avian supremacy at elevation. Here, however, we provide physiological evidence for the superior ability of birds to adapt and thrive at elevation compared with mammals. Two lines of evidence are provided to support this opinion, showing that birds have *1*) a greater tolerance of the hypoxia at high altitudes and *2*) a greater capacity for exercise at high altitudes.

Birds Have a Greater Tolerance of Hypoxia

Early evidence that birds are more tolerant of hypoxia than mammals showed that sparrows behaved normally and could even fly in a wind tunnel at a simulated altitude (6,100 m) that rendered mice comatose (24). Subsequent comparisons of the lowest survivable O_2 tensions of many species also supported the conclusion that birds are generally more hypoxia tolerant than mammals and that the most tolerant birds (e.g., bar-headed goose; ~20 Torr) survive lower O_2 tensions than the most tolerant euthermic mammals (e.g., mole rats; ~35 Torr; Ref. 23). Consideration of two of the most dangerous consequences of high-altitude exposure in humans—cerebral and pulmonary edema (9, 17)—suggests that tolerance is largely dictated by effects of hypoxia on the brain and lungs. Perhaps not surprisingly, the superior hypoxia tolerance of birds coincides with unique physiological characteristics that protect against cerebral and pulmonary dysfunction.

Hypoxia tolerance and the brain. The avian brain appears to be protected against the problems experienced by the mammalian brain during hypoxia. Neural function can be compromised in mammals during high-altitude hypoxia by changes in cerebral blood flow (CBF), which arise primarily in response to *1*) hypoxemia, which stimulates CBF in an attempt to maintain cerebral O_2 delivery, and *2*) hypocapnia (a secondary consequence of elevated breathing at high altitudes), which constricts cerebral blood vessels and offsets the hypoxemic stimulation of CBF (4). These conflicting mechanisms pose two potential problems in mammals. First, cerebral O_2 delivery and tissue oxygenation will be impaired if CBF does not increase during hypoxemia. Second, however, increases in CBF contribute to vasogenic edema and a rise in intracranial pressure that are thought to cause many of the neurological deficits at high altitudes (29). In contrast, CBF is insensitive to hypocapnia in birds, resulting in higher CBF and maintained cerebral O_2 delivery during hypoxemia (4). Should any deficit in O_2 supply occur, avian neurons are also more tolerant of low O_2 than mammalian neurons (11). Although the consequences of increases in CBF on intracranial pressure have not been directly studied in birds, some evidence suggests that intracranial pressure is exceptionally well maintained (10). If this is true at high altitude, birds should be capable of greater increases in CBF to avoid O_2 lack while not risking intracranial hypertension.

Hypoxia tolerance and the lungs. The avian lung does not seem to suffer the same dysfunction in hypoxia as the mammalian lung. Mammalian pulmonary vessels normally constrict in response to regional decreases in O_2 to divert blood flow toward the most oxygenated regions of the lungs. During hypoxia, when low O_2 occurs throughout the lungs, this response results in pulmonary hypertension and can lead to pulmonary edema and impaired gas exchange (12, 17). In contrast, birds do not exhibit hypoxic pulmonary vasoconstriction, so pulmonary arterial pressure increases only in response to heightened pulmonary blood flow (3, 5, 28). Pulmonary hypertension is also less likely to cause edema in birds than in mammals, because the avian blood-gas barrier is mechanically stronger and more resistant to stress failure (26).

Birds Have a Greater Capacity for Exercising at High Altitudes

Far more impressive than their superior tolerance compared with mammals is the ability of birds to perform intense exercise during hypoxia. The highest peaks in the Himalayas represent the altitudinal limit for humans, above which even basal aerobic metabolism cannot be sustained (27). Similar limits probably constrain exercise performance in other mammals as well (8). Some birds fly at these altitudes (18, 22)—an impressive feat considering that flight is more costly than other forms of vertebrate locomotion and that its cost increases as barometric pressure declines (1, 7, 25). The ability to sustain high rates of metabolism in hypoxia appears to arise from unique enhancements across the O_2 cascade.

O_2 transport and the respiratory system. The lungs of birds have a higher capacity for transporting O_2 during hypoxia than the lungs of mammals. Airflow is unidirectional through the terminal gas-exchange units of avian lungs (parabronchioles), and the arrangement of airway and vascular vessels creates a functionally cross-current gas exchanger (13). This mechanism of gas exchange is inherently more efficient than that in mammalian lungs during moderate hypoxia, such that O_2 tensions in the arterial blood of birds can exceed those in the expired gas (15, 16). The capacity for pulmonary O_2 diffusion is also higher in birds due to the exceptional thinness and large surface area of the gas-exchange tissue (26). The advantage of the avian respiratory system for loading O_2 into the blood is well exemplified by the bar-headed goose, a species that flies over the Himalayas at nearly 9,000 m elevation (22). This bird has particularly large lungs and a pronounced hypoxic ventilatory response, which help it realize arterial O_2 tensions in severe hypoxia that are indistinguishable from those in *inspired* gas (20, 21).

O_2 transport and the cardiovascular system. Birds should be capable of higher cardiac outputs and convective O_2 delivery than mammals. Birds have larger hearts and cardiac stroke volumes than mammals of similar body size (6), and during free flight (which is presumably submaximal exercise) birds can sustain heart rates that are similar to those of mammals during maximal exercise (2). Capillarity is also higher in the hearts of birds compared with mammals (4), and there are further increases in high fliers (21), suggesting that cardiac

output may be better sustained in birds during exercise in hypoxia. Birds do not have a higher hemoglobin-O_2 affinity in general, but the affinities of several highland species are enhanced compared with lowland birds (18), which should further improve O_2 delivery during hypoxia. The flight muscle of birds also has a superior capacity for O_2 diffusion than the locomotory muscle of mammals, largely due to a high degree of branching between adjacent capillaries (14), and this capacity is increased even further in the highest flying species (19). These differences should increase the overall ability to supply O_2 to the active muscle during exercise in hypoxia.

Conclusions

Better able to tolerate and exercise in hypoxia, we conclude that birds are far superior to mammals at adapting and thriving at high altitudes. This should not be surprising when considering that the ability to fly has for millions of years enabled birds access to altitudes that were unattainable to humans and other mammals. Perhaps the simplest explanation for why birds excel at elevation is that evolution has had much more time and reason to equip them with the physiology to do so!

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COUNTERPOINT: HIGH ALTITUDE IS NOT FOR THE BIRDS!

The best evidence for the adaptation of a species to its environment is that it reproduces efficiently and passes genes to future generations. This has occurred in mammals and birds resident at