Published in final edited form as:
Int Biol Rev. 2015 ; 2015(1): .

On fuel choice and water balance during migratory bird flights

Cecilia Giulivi[a,b,*] and **Jon Ramsey**[a]

[a]University of California, School of Veterinary Medicine, Department of Molecular Biosciences, Davis, CA 95616, USA

[b]MIND Institute, Sacramento, CA

Abstract

It has been proposed that water loss during flight in migratory birds under high evaporative conditions can be offset by the production of water through increased protein catabolism. Indeed, oxidation of protein may supply 7-times more water/kJ than fat. However, the lack of a relative increase in protein catabolism over that of fat during long flights indicates that processes other than water balance may be the primary drivers of protein catabolism during long and strenuous flights. These processes include the release of stress hormones (which increase both protein and fat catabolism) and protein catabolism triggered by increased oxidative damage to muscle proteins from reactive oxygen species produced by mitochondria. Protein catabolism is an important source of water for birds during migratory flight, but it remains to be determined if this process is directly regulated by hydration status.

Keywords

migratory birds; fat; glycogen; water; fuel; exercise

Introduction

Preventing dehydration is a major challenge for birds that migrate long distances without stopping to consume water. It has been reported that the water loss during flight in migratory birds under high evaporative conditions (HEWL) is offset by the production of water through increased protein catabolism, as demonstrated by higher post-flight plasma uric acid levels (20). Essentially this indicates that when faced with a negative water balance, these birds increase protein oxidation to provide both energy and water since protein catabolism produces 5 to 6 times more water than fat catabolism. The purpose of this brief review is to provide an overview of water formation associated with substrate catabolism and discuss how shifts in substrate oxidation can influence water formation.

*Corresponding author: cgiulivi@ucdavis.edu (C. Giulivi).

Conflict of interest statement
None of the authors of this paper has a financial or personal relationship with other people or organizations that could inappropriately influence or bias the content of the paper.

Water Formation During Substrate Oxidation

For pedagogical purposes, it is important to clarify the origin of the water produced during tissue catabolism. To illustrate this issue, we have calculated the water contribution from various substrates (Table I). Total water is the sum of water released by oxidation of a particular substrate, also called *metabolic water* (Table I, A & C), and that released by the hydration shell associated with substrates and electrolytes that occurs primarily as a consequence of the loss of lean tissue mass (Table I, D & F).

In the case of birds, it is crucial to consider the energy density of the substrate (kJ/g substrate wet weight) since body weight may compromise flight performance. Given that fat produces 7–10 times higher energy/g than lean tissue (Table, I B), it is not surprising that flight (as a high ATP-demanding process) under all humidity conditions, is sustained primarily by fatty acid oxidation (>90%; (36, 50)). In regard to metabolic water production, fat produces 6 and 13.5 times more metabolic water/g than hydrated glycogen or lean mass, respectively (Table I, C). However, if total water generation (metabolic water plus substrate-associated water) were the prime factor determining fuel storage, glycogen would be the preferred substrate yielding 180- and 26-times more water/kJ compared to fat and protein, respectively (Table I, G). Reliance on glycogen, nonetheless, is not a viable option for birds undergoing sustained exercise since there is a limited amount of glycogen that can be stored in the body without affecting significantly body weight. The next best option for migrating birds would be to breakdown lean tissue, which yields 7-times more water/kJ than fat (Table I, G).

To meet the intense metabolic demands of migration, birds rely on the ability of adipose tissue (i) to store high amount of fat in a relatively short period (about 10-fold fat increase in a week) and (ii) to catabolize it in 2–3 days during nonstop migratory flights (36). Respiration and evaporative cooling primarily account for water losses in migratory birds (1, 21). While most energy will be provided by fat during long flights, the total water produced by fat oxidation is almost equal to that produced by the much smaller amount of lean mass catabolized:

$$[0.032\,\mathrm{g\,water/kJ} \times 0.9]_{\mathrm{fat}}+[0.220\,\mathrm{g\,water/kJ} \times 0.1]_{\mathrm{lean\ mass}}=0.029+0.022$$

Gaining enough fat would minimize the frequency and duration of stopovers (which increases the risk of predatorial activity) and provide better chances of breeding at the final destination (14). However, some loss of lean mass (second sum term in the above equation) seems the only option that birds on long flights have for generating more water (after glycogen is depleted) if they encounter HEWL conditions. The success of this strategy will depend on how much lean mass can be gained prior to migration and how much lean mass can be lost before exercise performance is compromised. Protein catabolism from flight muscles and other organs has the concomitant advantage that flight muscle mass can be continuously adapted to the decreasing body mass (37) and the body mass to be carried is reduced (39). Pectoral muscle thickness decreases, as expected, during long flights (27, 33). However, the energetic demands of long flight produce changes in both lean and fat tissue mass. High residual body mass loss occurs in parallel with high plasma levels of both protein and lipid catabolites in migrating birds (27), indicating that rates of both protein and lipid metabolism increase in concert with no major change in the proportion of substrates used for energy. Furthermore, during 10 h flights, residual energy expenditure was positively correlated with both lipid catabolite levels and body mass loss (30). Although protein catabolism is an important contributor to water production during migratory flight, it is not clear if water status is a factor regulating substrate utilization by the animal.

Alternative hypotheses

Does negative water balance influencing the mix of substrates oxidized for energy during migratory flight? This is the central question in determining if protein catabolism during long flights is a regulated process for supplying birds with water. Alternative hypotheses to this regulated protein catabolism for water generation concept include water generation simply as a useful byproduct of increased protein catabolism driven by the release of stress hormones and/or increased oxidative damage to muscle proteins during strenuous, long flights.

Catecholamines and glucocorticoids (such as corticosterone) have been reported to play a role in stimulating flight, regulating fuel utilization during flight and optimizing flight performance in birds (4, 6–8, 13, 15–17, 19, 23, 24, 40, 44, 48). However, there is not uniform agreement regarding the effect of long flights on these hormones with some studies reporting that levels are not increased from baseline, possibly indicating an adaptation to long flights to minimize both lean mass protein loss which can compromise flight performance (26, 31). The stress-related hormones increase lipid and protein catabolism to provide energy, sustain gluconeogenesis (glucogenic amino acids, glycerol) and (25, 27) compensate for water losses. Additional work is needed, however, to determine if stress-related hormones are the primary stimulus for protein catabolism and generation of water during migratory flight.

Oxidative damage to lipids, nucleic acids and proteins during long flights (or sustained exercise; (9–11, 28, 32)) could also contribute to protein catabolism and production of water. This damage, mediated by reactive oxygen species leaking from mitochondria (2, 5, 22, 29, 34, 38, 41, 45, 47, 49), could induce protein catabolism to clear the oxidatively modified targets (3, 18, 46). This activation of catabolic pathways functions to ensure the removal of dysfunctional proteins (42, 43), does not seem to be triggered by a putative negative water balance. It remains to be determined if an appreciable amount of water is generated secondary to protein catabolism in response to flight-related oxidative stress.

Conclusions

Catabolism of both fats and proteins generate water, which helps mitigate water loss during long migratory flights. Although protein catabolism is often a relatively minor contributor to energy supply during migration, the large amount of water generated during breakdown of lean versus fat tissue makes protein catabolism an important source of water during flight. While the protein-for-water strategy should work in all animals (not only uricotelic), the sustained, intense exercise that occurs in migratory birds (especially those flying over oceans with 40–60 h for > 1,000 km) without the consumption of drinking water makes this process much more important in birds than terrestrial animals which undergo less intense levels of sustained exercise. It remains to be determined if water status plays an important role in regulating protein catabolism in migrating birds or if water is simply a fortunate byproduct of protein catabolism induced by intense exercise.

References

1. Altshuler DL, Dudley R. The physiology and biomechanics of avian flight at high altitude. Integr Comp Biol. 2006; 46:62–71. http://www.ncbi.nlm.nih.gov/pubmed/21672723. [PubMed: 21672723]
2. Benzi G. Aerobic performance and oxygen free-radicals. J Sports Med Phys Fitness. 1993; 33:205–22. http://www.ncbi.nlm.nih.gov/pubmed/8107472. [PubMed: 8107472]
3. Bordel R, Haase E. Influence of flight on protein catabolism, especially myofilament breakdown, in homing pigeons. Journal of Comparative Physiology B-Biochemical Systemic and Environmental Physiology. 2000; 170:51–8.
4. Breuner CW, Hahn TP. Integrating stress physiology, environmental change, and behavior in free-living sparrows. Horm Behav. 2003; 43:115–23. http://www.ncbi.nlm.nih.gov/pubmed/12614641. [PubMed: 12614641]
5. Casimiro-Lopes G, Ramos D, Sorenson MM, Salerno VP. Redox balance and mitochondrial glycerol phosphate dehydrogenase activity in trained rats. Eur J Appl Physiol. 2012; 112:3839–46. http://www.ncbi.nlm.nih.gov/pubmed/22391683. [PubMed: 22391683]
6. Chin EH, Love OP, Verspoor JJ, Williams TD, Rowley K, Burness G. Juveniles exposed to embryonic corticosterone have enhanced flight performance. Proc Biol Sci. 2009; 276:499–505. http://www.ncbi.nlm.nih.gov/pubmed/18842541. [PubMed: 18842541]
7. Corbel H, Groscolas R. A role for corticosterone and food restriction in the fledging of nestling White storks. Horm Behav. 2008; 53:557–66. http://www.ncbi.nlm.nih.gov/pubmed/18313056. [PubMed: 18313056]
8. Cornelius JM, Boswell T, Jenni-Eiermann S, Breuner CW, Ramenofsky M. Contributions of endocrinology to the migration life history of birds. Gen Comp Endocrinol. 2013; 190:47–60. http://www.ncbi.nlm.nih.gov/pubmed/23602795. [PubMed: 23602795]
9. Costantini D, Cardinale M, Carere C. Oxidative damage and anti-oxidant capacity in two migratory bird species at a stop-over site. Comp Biochem Physiol C Toxicol Pharmacol. 2007; 144:363–71. http://www.ncbi.nlm.nih.gov/pubmed/17218158. [PubMed: 17218158]
10. Costantini D, Dell'ariccia G, Lipp HP. Long flights and age affect oxidative status of homing pigeons (Columba livia). J Exp Biol. 2008; 211:377–81. http://www.ncbi.nlm.nih.gov/pubmed/18203993. [PubMed: 18203993]
11. Costantini D, Monaghan P, Metcalfe NB. Loss of integration is associated with reduced resistance to oxidative stress. J Exp Biol. 2013; 216:2213–20. http://www.ncbi.nlm.nih.gov/pubmed/23470664. [PubMed: 23470664]
12. Delp MD, Duan C. Composition and size of type I, IIA, IID/X, and IIB fibers and citrate synthase activity of rat muscle. J Appl Physiol (1985). 1996; 80:261–70. http://www.ncbi.nlm.nih.gov/pubmed/8847313. [PubMed: 8847313]
13. DesRochers DW, Reed JM, Awerman J, Kluge JA, Wilkinson J, van Griethuijsen LI, Aman J, Romero LM. Exogenous and endogenous corticosterone alter feather quality. Comp Biochem Physiol A Mol Integr Physiol. 2009; 152:46–52. http://www.ncbi.nlm.nih.gov/pubmed/18804171. [PubMed: 18804171]
14. Drent RH, Eichhorn G, Flagstad A, van der Graaf AJ, Litvin KE, Stahl J. Migratory connectivity in Arctic geese: spring stopovers are the weak links in meeting targets for breeding. Journal of Ornithology. 2007; 148:S501–S14.
15. Eikenaar C, Bairlein F, Stowe M, Jenni-Eiermann S. Corticosterone, food intake and refueling in a long-distance migrant. Horm Behav. 2014; 65:480–7. http://www.ncbi.nlm.nih.gov/pubmed/24721337. [PubMed: 24721337]
16. Eikenaar C, Fritzsch A, Bairlein F. Corticosterone and migratory fueling in Northern wheatears facing different barrier crossings. Gen Comp Endocrinol. 2013; 186:181–6. http://www.ncbi.nlm.nih.gov/pubmed/23518480. [PubMed: 23518480]

17. Eikenaar C, Klinner T, Stowe M. Corticosterone predicts nocturnal restlessness in a long-distance migrant. Horm Behav. 2014; 66:324–9. http://www.ncbi.nlm.nih.gov/pubmed/24956025. [PubMed: 24956025]

18. Evans WJ, Cannon JG. The metabolic effects of exercise-induced muscle damage. Exerc Sport Sci Rev. 1991; 19:99–125. http://www.ncbi.nlm.nih.gov/pubmed/1936096. [PubMed: 1936096]

19. Falsone K, Jenni-Eiermann S, Jenni L. Corticosterone in migrating songbirds during endurance flight. Horm Behav. 2009; 56:548–56. http://www.ncbi.nlm.nih.gov/pubmed/19782685. [PubMed: 19782685]

20. Gerson AR, Guglielmo CG. Flight at low ambient humidity increases protein catabolism in migratory birds. Science. 2011; 333:1434–6. http://www.ncbi.nlm.nih.gov/pubmed/21903811. [PubMed: 21903811]

21. Giladi I, Pinshow B. Evaporative and excretory water loss during free flight in pigeons. Journal of Comparative Physiology B-Biochemical Systemic and Environmental Physiology. 1999; 169:311–8.

22. Giulivi C, Boveris A, Cadenas E. The steady-state concentrations of oxygen radicals in mitochondria. 1999:77–102.

23. Gray JM, Yarian D, Ramenofsky M. Corticosterone, foraging behavior, and metabolism in dark-eyed juncos, Junco hyemalis. Gen Comp Endocrinol. 1990; 79:375–84. http://www.ncbi.nlm.nih.gov/pubmed/2272460. [PubMed: 2272460]

24. Haase E, Rees A, Harvey S. Flight stimulates adrenocortical activity in pigeons (Columba livia). Gen Comp Endocrinol. 1986; 61:424–7. http://www.ncbi.nlm.nih.gov/pubmed/3956993. [PubMed: 3956993]

25. Jenni L, Jenni-Eiermann S, Spina F, Schwabl H. Regulation of protein breakdown and adrenocortical response to stress in birds during migratory flight. Am J Physiol Regul Integr Comp Physiol. 2000; 278:R1182–9. http://www.ncbi.nlm.nih.gov/pubmed/10801285. [PubMed: 10801285]

26. Jenni-Eiermann S, Hasselquist D, Lindstrom A, Koolhaas A, Piersma T. Are birds stressed during long-term flights? A wind-tunnel study on circulating corticosterone in the red knot. Gen Comp Endocrinol. 2009; 164:101–6. http://www.ncbi.nlm.nih.gov/pubmed/19481083. [PubMed: 19481083]

27. Jenni-Eiermann S, Jenni L, Kvist A, Lindstrom A, Piersma T, Visser GH. Fuel use and metabolic response to endurance exercise: a wind tunnel study of a long-distance migrant shorebird. Journal of Experimental Biology. 2002; 205:2453–60. [PubMed: 12124368]

28. Jenni-Eiermann S, Jenni L, Smith S, Costantini D. Oxidative stress in endurance flight: an unconsidered factor in bird migration. PLoS One. 2014; 9:e97650. http://www.ncbi.nlm.nih.gov/pubmed/24830743. [PubMed: 24830743]

29. Johnson F, Giulivi C. Superoxide dismutases and their impact upon human health. Mol Aspects Med. 2005; 26:340–52. http://www.ncbi.nlm.nih.gov/pubmed/16099495. [PubMed: 16099495]

30. Kvist A, Lindstrom A, Green M, Piersma T, Visser GH. Carrying large fuel loads during sustained bird flight is cheaper than expected. Nature. 2001; 413:730–2. http://www.ncbi.nlm.nih.gov/pubmed/11607031. [PubMed: 11607031]

31. Landys-Ciannelli MM, Ramenofsky M, Piersma T, Jukema J, Wingfield JC, Castricum Ringing G. Baseline and stress-induced plasma corticosterone during long-distance migration in the bar-tailed godwit, Limosa lapponica. Physiol Biochem Zool. 2002; 75:101–10. http://www.ncbi.nlm.nih.gov/pubmed/11880983. [PubMed: 11880983]

32. Larcombe SD, Tregaskes CA, Coffey JS, Stevenson AE, Alexander L, Arnold KE. The effects of short-term antioxidant supplementation on oxidative stress and flight performance in adult budgerigars Melopsittacus undulatus. J Exp Biol. 2008; 211:2859–64. http://www.ncbi.nlm.nih.gov/pubmed/18723545. [PubMed: 18723545]

33. Lindstrom A, Kvist A, Piersma T, Dekinga A, Dietz MW. Avian pectoral muscle size rapidly tracks body mass changes during flight, fasting and fuelling. J Exp Biol. 2000; 203:913–9. http://www.ncbi.nlm.nih.gov/pubmed/10667974. [PubMed: 10667974]

34. Mankowski, RT., Anton, SD., Buford, TW., Leeuwenburgh, C. Dietary Antioxidants as Modifiers of Physiologic Adaptations to Exercise. Med Sci Sports Exerc. 2015. http://www.ncbi.nlm.nih.gov/pubmed/25606815
35. Morrison SD. A method for the calculation of metabolic water. J Physiol. 1953; 122:399–402. http://www.ncbi.nlm.nih.gov/pubmed/13118549. [PubMed: 13118549]
36. Odum EP. Adipose tissue in migratory birds. Handbook Physiol. 1965; 5:37–43.
37. Pennycuick CJ. Towards an optimal strategy for bird flight research. Journal of Avian Biology. 1998; 29:449–57.
38. Pereira B, Costa Rosa LF, Safi DA, Medeiros MH, Curi R, Bechara EJ. Superoxide dismutase, catalase, and glutathione peroxidase activities in muscle and lymphoid organs of sedentary and exercise-trained rats. Physiol Behav. 1994; 56:1095–9. http://www.ncbi.nlm.nih.gov/pubmed/7824577. [PubMed: 7824577]
39. Piersma T, Lindstrom A. Rapid reversible changes in organ size as a component of adaptive behaviour. Trends in Ecology & Evolution. 1997; 12:134–8. [PubMed: 21238009]
40. Piersma T, Reneerkens J, Ramenofsky M. Baseline corticosterone peaks in shorebirds with maximal energy stores for migration: a general preparatory mechanism for rapid behavioral and metabolic transitions? Gen Comp Endocrinol. 2000; 120:118–26. http://www.ncbi.nlm.nih.gov/pubmed/11042017. [PubMed: 11042017]
41. Powers SK, Sollanek KJ, Wiggs MP, Demirel HA, Smuder AJ. Exercise-induced improvements in myocardial antioxidant capacity: the antioxidant players and cardioprotection. Free Radic Res. 2014; 48:43–51. http://www.ncbi.nlm.nih.gov/pubmed/23915097. [PubMed: 23915097]
42. Radak Z, Kaneko T, Tahara S, Nakamoto H, Ohno H, Sasvari M, Nyakas C, Goto S. The effect of exercise training on oxidative damage of lipids, proteins, and DNA in rat skeletal muscle: evidence for beneficial outcomes. Free Radic Biol Med. 1999; 27:69–74. http://www.ncbi.nlm.nih.gov/pubmed/10443921. [PubMed: 10443921]
43. Radak Z, Toldy A, Szabo Z, Siamilis S, Nyakas C, Silye G, Jakus J, Goto S. The effects of training and detraining on memory, neurotrophins and oxidative stress markers in rat brain. Neurochem Int. 2006; 49:387–92. http://www.ncbi.nlm.nih.gov/pubmed/16564605. [PubMed: 16564605]
44. Rees A, Hall TR, Harvey S. Adrenocortical and adrenomedullary responses of fowl to treadmill exercise. Gen Comp Endocrinol. 1984; 55:488–92. http://www.ncbi.nlm.nih.gov/pubmed/6468924. [PubMed: 6468924]
45. Sachdev S, Davies KJ. Production, detection, and adaptive responses to free radicals in exercise. Free Radic Biol Med. 2008; 44:215–23. http://www.ncbi.nlm.nih.gov/pubmed/18191757. [PubMed: 18191757]
46. Uchiyama S, Tsukamoto H, Yoshimura S, Tamaki T. Relationship between oxidative stress in muscle tissue and weight-lifting-induced muscle damage. Pflugers Arch. 2006; 452:109–16. http://www.ncbi.nlm.nih.gov/pubmed/16402246. [PubMed: 16402246]
47. Venditti P, Masullo P, Di Meo S. Effect of training on H(2)O(2) release by mitochondria from rat skeletal muscle. Arch Biochem Biophys. 1999; 372:315–20. http://www.ncbi.nlm.nih.gov/pubmed/10600170. [PubMed: 10600170]
48. Viswanathan M, John TM, George JC, Etches RJ. Flight effects on plasma glucose, lactate, catecholamines and corticosterone in homing pigeons. Horm Metab Res. 1987; 19:400–2. http://www.ncbi.nlm.nih.gov/pubmed/3692434. [PubMed: 3692434]
49. Walsh B, Tonkonogi M, Sahlin K. Effect of endurance training on oxidative and antioxidative function in human permeabilized muscle fibres. Pflugers Arch. 2001; 442:420–5. http://www.ncbi.nlm.nih.gov/pubmed/11484774. [PubMed: 11484774]
50. Weis-Fogh T. Energetics and Aerodynamics of Flapping Flight a Synthesis. 1976:48–72.
51. Widdowson, EM. Body composition in animals and man. Washington: National Academy of Sciences Press; 1968. Biological implications of body composition; p. 71-86.

TABLE I

Water formation by oxidation of storage substrates

	A	B	C	D	E	F	G
Substrates	Metabolic H_2O in g/kJ of oxidized substrate[a]	Energy in kJ/g substrate wet wt	Metabolic H_2O in g/g fuel (A × B)	H_2O content in g/g dry wt substrate[d]	Energy in kJ/g dry wt	H_2O content in g/kJ (D/E)	Total H_2O in g/kJ (A + F)
Fat	0.0272	39.6[e]	**1.08**	0.20	39.6	0.005	**0.032**
Protein	0.0220	3.7[b]	**0.08**	3.65	18.4	0.198	**0.220**
Carbohydrate	0.0318	5.6[c]	**0.18**	2.10	17	0.124	**5.632**

[a]Values for calculating metabolic water production from indirect respiration calorimetry (35).

[b]Assuming that lean tissue contains approximately 73% water (12, 51).

[c]Assuming that 2/3 of glycogen wet weight is water.

[d]Water associated with electrolytes and hydration shells.

[e]Assuming that fatty acids can be mobilized from adipose tissue without loss of tissue water.