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Arctic waders are not capital breeders

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Ornithology

Arctic waders are not capital breeders

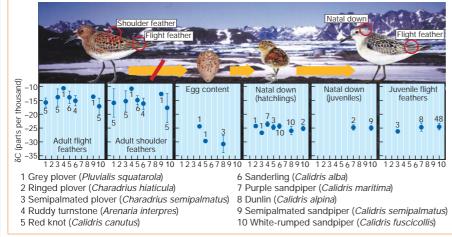
irds prepare their eggs from recently ingested nutrients ('income' breeders) or from body stores ('capital' breeders)¹. As summers are short at Arctic latitudes, Arctic migrants have been presumed to bring nutrients for egg production from their previous habitats, so that they can start breeding immediately upon arrival^{1–3}. But we show here that eggs laid by 10 different wader species from 12 localities in northeast Greenland and Arctic Canada are produced from nutrients originating from tundra habitats, as inferred from carbon stable-isotope ratios in eggs, natal down, and juvenile and adult feathers.

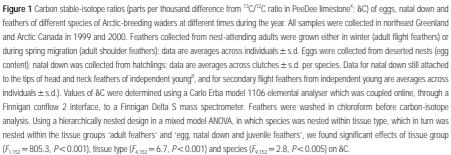
During winter and migration, most Arctic-breeding waders eat estuarine invertebrates, shifting to terrestrial and limnic invertebrates on tundra breeding grounds. Invertebrates from estuarine and tundra habitats have distinctly different carbonisotope ratios⁴. Diet-based differences in carbon-isotope ratios are expressed in bird tissues, including eggs and feathers^{4.5}. We investigated whether Arctic-breeding wader species that use estuarine habitats during the non-breeding season are capital breeders, producing eggs and hatchlings with carbon-isotope ratios that are typical of estuaries.

As expected, the flight feathers produced during early winter show ratios that are typical of estuarine systems (Fig. 1). The same is true for shoulder feathers, which are among the last to be moulted before migration to breeding grounds⁶. However, the carbon-isotope signatures of eggs and natal down are typical of terrestrial and limnic systems. The eggs and resultant hatchlings therefore seem to be produced from local nutrients. This conclusion is supported by the strong resemblance of the carbon-isotope signatures of natal down and juvenile flight feathers, showing that eggs and post-hatch tissues have the same nutritional source.

Our results also indicate that the use of a mixed capital/income breeding strategy by these waders is unlikely. Egg-laying females use a single, local nutrient source, as indicated by the low within-clutch variation compared with the among-clutch variation in carbon-isotope ratios of natal down. (We used a hierarchically nested design in a mixed-model ANOVA, in which brood is nested within species as a random factor, and found significant effects of species ($F_{6,30}$ = 4.8, P < 0.002) and brood ($F_{29,93}$ = 16.8, P < 0.001) on the carbon stable-isotope ratios of natal down.)

We conclude that, with respect to egg production, the Arctic-breeding waders investigated here are income, rather than capital, breeders. For these waders, the fitness costs of transporting extra nutrient stores to breeding grounds⁷ outweigh the potential benefits¹⁻³. The capital strategy may still be used by large species of Arctic breeding migrants, such as geese^{1,2} — it is likely that larger species need relatively





smaller body stores for egg production. Also, larger species, although constrained by the same fixed period of opportunity as smaller birds, require longer to complete their breeding.

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Intracellular signalling

Key enzyme in leptin-induced anorexia

eptin is a key hormonal regulator of energy balance that acts upon hypothalamic neurons to reduce food intake, but the intracellular mechanisms involved are incompletely understood. Here we show that systemic administration of leptin in rats activates the enzyme phosphatidylinositol-3-OH kinase (PI(3)K) in the hypothalamus and that intracerebroventricular (i.c.v.) infusion of inhibitors of this enzyme prevents leptin-induced anorexia. Our results indicate that PI(3)K is a crucial enzyme in the signal-transduction pathway that links hypothalamic leptin to reduced food intake.

Leptin, a hormone derived from adipocytes (fat cells), is known to activate PI(3)K in non-neuronal cells and to elicit cellular responses that are blocked by PI(3)K inhibitors *in vitro*^{1,2}; it also activates the transcription factor STAT3 (ref. 3). To determine whether leptin activates PI(3)K, as well as STAT3, in the hypothalamus *in vivo*, we observed the effect of systemically administered leptin on their activity in male Wistar rats. As before³, leptin induced rapid tyrosine-phosphorylation of STAT3