There has long been a dichotomy of opinion as to

Life-history costs of egg production

Many birds (called "indeterminate layers") will lay additional eggs if eggs are removed from the nest during laying, and this has been one of the main arguments against there being physiological limitations on egg production. However, it is now clear that females do incur costs associated specifically with reproductive effort during egg production, though studies are limited to seabirds and one or two passerine species to date. Reproduction can be manipulated by giving parents an extra chick (increasing only rearing costs) or by using egg removal to make females lay an additional egg (increasing both egg production costs and rearing costs). Birds with increased egg production costs have chicks with lower hatching mass (independent of egg size), reduced chick growth, increased early mortality, and decreased chick survival relative to those with only additional rearing costs (Monaghan et al. 1995). These effects are mediated through the parents' reduced brood-rearing or provisioning capacity (Monaghan et al. 1998). Furthermore, in several species, the females that are made to lay additional eggs (through egg removal) also have lower local return rates the following year (Nager et al. 2001, Visser and Lessells 2001). Although birds can lay additional eggs in response to egg removal, egg quality decreases rapidly with increasing egg number above the normal clutch size. For example, lesser black-backed gulls (*Larus fuscus*) will lay almost three times the normal clutch size of three eggs in response to continuous egg removal, but eggs laid late in the experimental laying sequence have 50% lower fledging success (Nager et al. 2000). This decrease in egg quality is due to changes in egg composition that are independent of changes in egg size (see also Williams and Miller 2003). Increased re-

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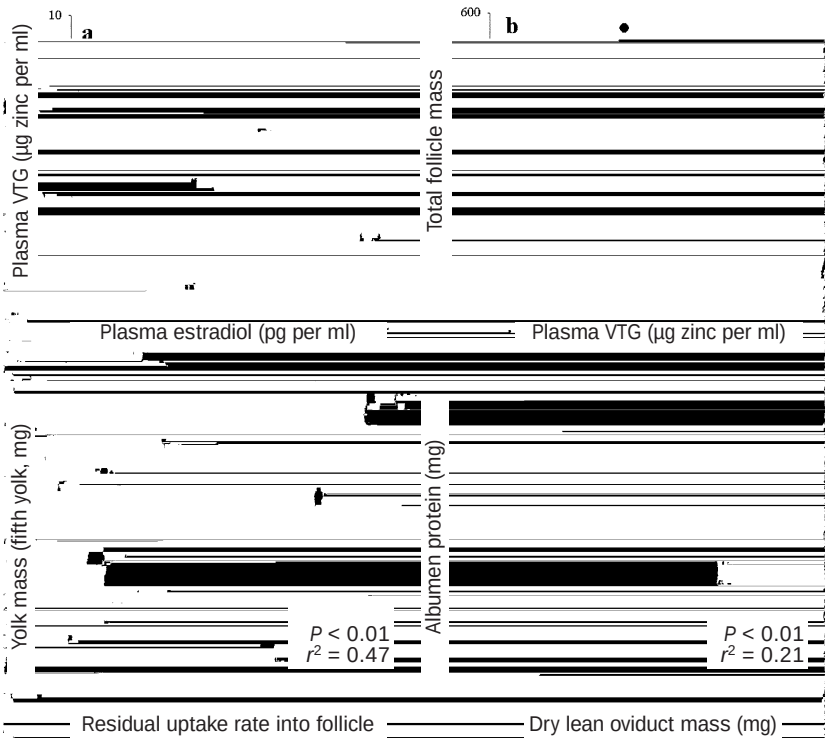
again, although numerous studies suggest that certain specific

itself cause higher parasite load, or are infected individuals in poorer condition and as a result likely to lay smaller eggs?). A resource-based trade-off between reproduction and immune function (immunocompetence) could operate through general energy or nutrient reallocation, or through reallocation of a specific limiting factor that plays a key role in regulating immune function in embryos or chicks as well as in the adult female (e.g., carotenoids or immunoglobulins), but few studies have addressed this issue in egg-producing birds. Martin and colleagues (2003) estimated the cost of an elevated cell-mediated immune response as 29% of RMR in captive, nonbreeding house sparrows (*Passer domesticus*), which they equated to a theoretically determined cost of production of half an egg. However, experimentally elevated primary antibody production during egg formation had no effect on egg or clutch size in free-living European starlings (Williams et al. 1999).

Eggs contain substantial amounts of various immune factors (carotenoids, immunoglobulins, lysozymes, etc.), which might be important in determining offspring fitness through effects on immune function in developing embryos or chicks. If laying females deplete their own stores of these factors, they might compromise their own ability to mount an immune response. Few studies have considered the effects of such factors on laying females, but in female barn swallows, plasma lysozyme activity does decline during the prelaying and laying periods (Saino et al. 2002). However, although plasma immunoglobulin concentrations increased during egg laying, postlaying plasma concentrations of immunoglobulins were similar to concentrations before laying, suggesting that females did not compromise their own immune system in order to allow passive transfer of immunity to offspring for this immune factor (Saino et al. 2001). In summary, although reproductive effort can lead to a decrease in immune function, only one study to date has linked long-term changes in immune function to future survival (i.e., to a measurable cost of reproduction), and this was in chick-rearing, not egg-producing, birds (Ardia et al. 2003). As with studies showing increases in energy demand during egg production, there is no evidence to show that transient, short-term decreases in immune function can have long-term consequences for future fecundity or survival.

Individual variation and the cost–benefit paradigm



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a positive relationship between albumen content of eggs and oviduct mass (figure 1d). Furthermore, *experimental* manipulation of yolk precursor levels, using the antiestrogen tamoxifen, does affect egg size, though even here a 50% decrease in yolk precursors levels reduces egg size by only 10% (Williams 2001), consistent with a buffered capacity for precursor production.

In general, these observations seem to question the idea of an "efficient" egg production system that has evolved through natural selection to match the capacity of physiological traits to variation in reproductive output. Moreover, no studies have found any relationship between variation in these physiological traits and clutch size (fecundity). If receptor-mediated uptake of yolk into follicles does represent a "costly" component of egg production (figure 1c), why would individuals vary in their ability to synthesize receptors? Are there developmental pathways that link lipoprotein receptor expression, such that upregulation of vitellogenin receptors causes upregulation of receptors in other tissues with potentially negative consequences? We know virtually nothing about the costs of the individual components of the reproductive system. However, two recent studies suggest that elevated yolk precursor and hormone synthesis are not associated with elevated energy expenditure in laying birds (Eising et al. 2003, Vézina et al. 2003).

This lack of a systematic relationship between many physiological and reproductive traits, which should be functionally linked, is not simply due to the fact that variation in the physiological trait is "neutral" or random. Plasma vitellogenin concentration in laying birds is a highly repeatable trait (Challenger et al. 2001), even though it has a low degree of genetic determination (Nestor et al. 1996). In other words, there appear to be "high-vitellogenin" and "low-vitellogenin" phenotypes (albeit at the ends of a continuum), but this phenotypic variation in lipoprotein physiology is unrelated to variation in egg size or clutch size. So why do some birds secrete tenfold higher estradiol or vitellogenin levels compared with other birds, when this has no apparent benefit in terms of increased reproductive output? Resolving the functional consequences of this large-scale, interindividual physiological variation in the reproductive system represents an exciting challenge for the future in terms of our understanding of costs of egg production, both from a physiological and an evolutionary perspective. In this context, it is also important to note that at the intraspecific level, interrelationships between "core" life-history traits (egg size, female size, incubation period, etc.) are often weak, or even nonexistent, whereas these relationships can be strong at the interspecific level (see figure 4.2 in Bennett and Owens 2002). This highlights a problem in applying results from traditional interspecific or comparative analyses to an understanding of intraspecific variation (another example is the very different mass exponents obtained for allometry of body composition from between- versus within-species studies; Guglielmo and Williams 2003). It is clear that we have to better understand the variability in physiological systems involved in egg production if we are to link these systems, and other physiological processes, to life-history costs of egg production.

Physiological mechanisms for non-resource-based costs of egg production

Most studies, and therefore much of this review, have focused on resource-based trade-offs as the basis of physiological costs of egg production. In general, much less attention has been paid to non-resource-based costs, even though these can often lead to very different interpretations. For example, it has been suggested that immunosuppresion during laying might be adaptive if it prevents inappropriate autoimmune responses analogous to protection of the fetoplacental unit in mammals (Raberg et al. 1998). Potential mechanisms for non-resource-based costs of egg production come from the broad pleiotropic effect of reproductive hormones (Ketterson and Nolan 1999). Egg formation involves large cyclical changes

in many hormones, associated with reproductive behaviors, ovarian and oviductal growth and regression, and onset of incubation. Steroid hormones and gonadotropins increase rapidly to a peak at the time of courtship, territoriality, and egg laying, and then rapidly decrease, whereas prolactin increases at onset of incubation and is high through incubation (see figure 7 in Williams 1999). Many, if not all, hormones involved

(1974) reported an increase in thymus gland size during incubation and chick rearing—that is, after plasma estradiol would have returned to basal levels, which they argued facilitated a recovery of hematocrit through increased erythropoiesis. A prolonged decrease in hematocrit might therefore be due to a direct estradiol-dependent inhibition of red blood cell production (Kern et al. 1972, Williams et al. 2004b). The relatively long life span of avian red blood cells (30–42 days) supports the idea that a transient inhibition of erythropoiesis could induce a prolonged reduction in hematocrit. Females are particularly vulnerable to further decreases in hematocrit when exposed to additional stressors during egg production (figure 2b), and this is associated with a reduction in breeding success. Since hematocrit can affect flight ability and aerobic performance in general (Carpenter 1975, Hammond et al. 2000), decreased hematocrit might provide an explanation for long-term costs of egg production, such as decreased chick-provisioning ability, renesting probability, or survival.

Conclusions

It is clear that birds can and do incur costs of reproduction specifically associated with egg production, although comprehensive studies have so far been limited to a few taxa (seabirds and some passerines). If egg production is indeed costly, it should be possible to identify the components of the physiological system underlying reproduction, or the specific reproductive traits themselves, that explain these costs. This has proved to be surprisingly difficult, primarily because of the marked individual variation that occurs in reproductive and physiological traits (perhaps suggesting that these traits are not costly and that they are not under strong selection). Many studies continue to focus on factors, such as food availability, social environment, and temperature, that actually contribute relatively little to variability in reproductive traits (e.g., less than 10% of variability in egg size) compared with the much greater true variability among individuals (e.g., variability of 60%–100% in egg size; Christians 2002). Although egg and clutch size are, to varying degrees, genetically determined, the mechanism or mechanisms through which genotype determines intraspecific, phenotypic variation are unknown. We need a much better characterization of this intraspecific variability, and descriptive and correlational studies will be informative, although experimental manipulations of physiological mechanisms are really needed (Williams 2000, 2001). The many recent studies on other aspects of egg quality, such as egg composition, egg carotenoid content, and egg hormone content, have simply added more evidence of unexplained phenotypic variation rather than advancing our knowledge of the mechanisms underlying this variation or their associated costs.

Several mechanisms have been proposed for the cost of egg production (e.g., protein depletion and impaired flight muscle function; immunosuppression), but the focus to date has been on resource-based costs. These proposed mechanisms assume that animals have finite resources and that the increased resource demand for egg production reduces the resources available for other functions. It is clear that the energy

ated (weeks to months or years) surely argue against a simple resource-allocation basis for trade-offs. Instead, I suggest that a more productive approach for future research will be to focus on non-resource-based costs of egg production, such as those mediated by the pleiotropic effects of reproductive hormones. These generate fundamental questions with regard to mechanisms, such as why individuals cannot dissociate positive and negative effects of hormones through tissue-specific receptor expression. But I believe that non-resource-based mechanisms (e.g., lipoprotein phenotype, accumulation of oxidative stress, and estrogen-induced anemia) are more consistent with the longer time scales over which biological costs operate. They might also explain why costs can be related to reproduction in an all-or-nothing way—that is, they are related to egg production per se but are independent of the level of variation in fecundity (Ladyman et al. 2003). Finally, there has been something of a disconnect between the studies of costs of reproduction in birds and those in other taxa. This is, in part, because much of this work has been carried out in an ecological or evolutionary context (e.g., by field behavioral ecologists) rather than a genetic, physiological, or molecular biology context. Work on reproduction and life span in model species (e.g., *Caenorhabditis elegans*, *Drosophila*) is far more mechanistically sophisticated and should inform future studies in nonmodel species, such as free-living birds.

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