EXAMINING THE CAUSES AND CONSEQUENCES OF VARIATION IN OFFSPRING GROWTH AND SURVIVAL IN AN ALTRICIAL BIRD

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ABSTRACT

Robert Allen Aldredge: EXAMINING THE CAUSES AND CONSEQUENCES OF VARIATION IN OFFSPRING GROWTH AND SURVIVAL IN AN ALTRICIAL BIRD
(Under the direction of Keith Sockman)

Understanding why parents often produce supernumerary offspring and increase the variation in offspring growth and survival is a fundamental question in evolutionary ecology. This behavior occurs primarily in species that live in unpredictable environments and is thought to be an adaptive strategy used by parents to maximize the number of offspring that survive to breed. In this dissertation, I explore both how and why female birds increase the variation in growth and survival of supernumerary offspring. To do this, I collected observational and experimental data over four years in a free-living population of house sparrows. In Chapter 2, I developed a novel technique to uncover variation in growth (e.g. mass change) not detected by conventional analyses. I show that variation in growth occurs when some offspring increase mass slowly, likely owing to a lack of food resources. Some of these offspring recover and increase mass rapidly to approach a similar pre-fledging mass as offspring that do not delay development, whereas others continue to increase mass slowly and are light at fledging. This plasticity in growth likely increases the number of high quality offspring that fledge. In Chapter 3, I tested whether female birds begin embryonic development (incubation) before all eggs are laid either 1) as an adaptive strategy to maximize the number of

embryos that survive to hatch or 2) as an adaptive strategy to maximize the number or quality of hatched young that fledge. I show that early incubation occurs too late to maximize survival of all embryos but early enough to reduce growth and survival of late-hatched young. Thus, early incubation likely is a tradeoff between increasing embryo survival and maintaining growth and survival of late-hatched young. Overall, my dissertation shows that house sparrows exhibit considerable plasticity in offspring growth, and that early incubation likely maximizes the number of embryos that survive the incubation and nestling periods to fledge as high quality young. Thus, the variation in offspring growth caused by early incubation may occur as consequence of unique adaptations (prolonged oviposition and parental incubation) for offspring production and development in house sparrows and other birds.

To my wife, who cried with me when I failed, encouraged me when I was down, and celebrated with me when I succeeded. I could not have done this without you.

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CHAPTER 1: INTRODUCTION

The availability of resources for raising offspring often is unpredictable at the time of fertilization. In response to this unpredictability, many plants and animals produce as many or more offspring (embryos) than the environment can support, on average, and then eliminate surplus young when resources are limiting (Mock and Parker 1997). This strategy of eliminating supernumerary offspring allows parents to track resource availability and maximize the number of offspring that survive in unpredictable environments (Forbes 1990).

For many species, parents not only must 'decide' when to eliminate surplus young, but they also need to 'decide' who to eliminate when resources are limiting. These likely are not conscious 'decisions' but represent behaviors that have been selected by evolution because they increase the number of offspring that survive to reproduce. One strategy parents can adopt is to invest in all offspring equally and randomly pick which offspring to eliminate when resources are scarce. Unfortunately, equal investment in all offspring provides an inefficient mechanism to track resource availability because poor growth, which decreases survival and reproduction in all offspring, likely is the best signal that one or more young need to be eliminated. An alternative strategy is to invest in offspring based on size and competitive ability (Ricklefs 1965). Unequal investment in the most competitive offspring provides an efficient mechanism to track resource availability because parents can optimize growth in a core group of offspring and quickly and

selectively eliminate poor quality young when resources are scarce (Forbes et al. 2001). Thus, many species inhabiting unpredictable environments invest unequally in supernumerary offspring, a behavior that increases the variation in offspring growth and survival but likely maximizes the production of high quality young (Mock and Parker 1997).

Hatching (or birth) asynchrony provides one of the clearest examples of how parents invest unequally in supernumerary offspring. Hatching asynchrony occurs when siblings of the same cohort hatch (or are born) over an extended period, which allows some offspring to begin feeding and increase size before their siblings have finished embryonic development. In most species, the variation in offspring size caused by hatching asynchrony increases as offspring develop because large, early-hatched offspring outcompete their smaller siblings for access to resources provided by the parents (Myers and Master 1983, Szöllősi et al. 2007, Drake et al. 2008, Trillmich and Wolf 2008, Sharifi and Vaissi 2013). Thus, hatching asynchrony often is viewed as an adaptive strategy used by parents to differentiate offspring and maximize the production of high quality young in unpredictable environments (Lack 1954, Magrath 1989, Wiebe and Bortolotti 1995, Takata et al. 2014).

Hatching (or birth) asynchrony appears to have evolved independently in several taxa, including insects (Smiseth et al. 2006), cartilaginous fish (Gilmore 1993), reptiles (While et al. 2007), mammals (Fraser et al. 1979) and birds (Clark and Wilson 1981). Unlike in most taxa, hatching asynchrony is widespread in birds, which is facilitated by two unique adaptations shared by nearly all bird species: 1) unlike most oviparous species, birds have lost the ability to retain shelled eggs *in utero*, which causes females to

lay eggs as they are ovulated (Blackburn and Evans 1986) and 2) almost all birds regulate embryonic development by external heat provided by one or more parents (incubation; Avise 2013). These adaptations cause birds to lay at most one egg per day and also enable parents to control the timing of embryonic development, and hence hatching asynchrony, among offspring.

Over half a century ago, David Lack (1947, 1954) proposed that many temperate breeding birds produce as many eggs (embryos) as parents can raise in optimal conditions, and begin incubation and thus embryonic development before all eggs are laid. This early incubation causes eggs to hatch asynchronously (Clark and Wilson 1981), which allows parents to reduce the number of offspring when food resources are scarce. Thus, Lack suggested that hatching asynchrony was an adaptive strategy used by parents to track food resources that are unpredictable at the time of egg laying. Despite over fifty years of research, it remains unclear whether hatching asynchrony maximizes offspring growth and survival (Stoleson and Beissinger 1997). Consequently, more than nineteen hypotheses have been proposed to explain the prevalence of hatching asynchrony in birds, but none have overwhelming support (Stoleson and Beissinger 1995, Viñuela 2000). Most are derived from Lack's brood reduction hypothesis and predict that hatching asynchrony is a strategy used by parents to increase the number of hatched offspring that fledge. Collectively, they are called adaptive hatching pattern hypotheses.

One problem with contemporary tests of the adaptive hatching pattern hypotheses is that experimental manipulations often are done after hatching. Although this framework tests whether variation in hatching increases post-hatching growth and survival, the experimental manipulation does not test the specific behavior (i.e.

incubation onset) that causes variation in hatching. Thus, most experimental studies exclude the possibility that early incubation is selected for reasons other than creating an adaptive hatching pattern (Stoleson and Beissinger 1995, Stenning 1996, Viñuela 2000). Some of these alternative explanations include the possibility that early incubation is an adaptive behavior that maximizes embryo (pre-hatching) survival (egg viability hypothesis; Arnold et al. 1987) or that early incubation reduces the amount of time offspring are exposed to nest predation (nest failure hypothesis; Clark and Wilson 1981). Because hatching asynchrony is strongly associated with timing of incubation in altricial birds (Wang and Beissinger 2009 but see Bortolotti and Wiebe 1993), researchers can create variation in hatching by regulating when females begin incubation. Such manipulations can test whether early incubation maximizes post-hatching growth and survival or whether early incubation may have some other adaptive function.

For my dissertation I examined some causes and consequences of variation in offspring growth and survival in the house sparrow *Passer domesticus*. Specifically, I studied how natural patterns of hatching asynchrony were associated with offspring growth during the first two years of my study (2011-12), and then ran a manipulative experiment during the last two years of my study to test how timing of incubation influenced both pre-hatching survival and post-hatching survival and growth. Female house sparrows often produce more offspring than the environment can support, and begin incubation one or more days before all eggs are laid (O'Connor 1977). This early incubation onset often is associated with eggs hatching asynchronously over one or more days, which enables parents to feed nestlings based on the size hierarchy established at hatching (Veiga and Viñuela 1993). Post-hatching mortality is common, and sometimes

high, in house sparrows, and likely allows parents to track resources that are unpredictable during egg laying (Anderson 2006). Thus, the house sparrow is an appropriate species to investigate the causes and consequences of variation in offspring growth and survival.

I looked at variation in offspring growth in two ways, which comprise each of my two data chapters. In my first data chapter (Chapter 2) I describe a novel analytical technique for characterizing variation in offspring growth (e.g. developmental changes in mass). Specifically, I use random effect estimates from nonlinear mixed effects models to characterize alternative growth patterns. I show that offspring often delay their rapid mass increase soon after hatching and then either extend the period of rapid mass increase to approach an optimal pre-fledging mass or maintain a slow but steady mass increase and are light at fledging. This chapter provides a simple way to define alternate growth patterns in passerines, such as the house sparrow, and likely can be extended to other organisms that display sigmoidal growth. In my second data chapter (Chapter 3) I show how timing of embryonic development influences both pre- and post-hatching survival of offspring. This chapter involves the analysis of only the field data in 2013 and 2014, in which I experimentally manipulated when females could begin incubation. I found that embryo survival was high in all but the earliest-laid eggs in naturally asynchronous nests but remained low across the laying sequence when incubation was experimentally synchronized. The experimental manipulation had little effect on overall (i.e. mean) patterns of post-hatching growth and survival. Instead, early incubation caused a delay in hatching for late-laid eggs, and it was this delay in hatching that was associated with reduced post-hatching growth and survival in late-hatched young. Thus, early incubation

neither maximized embryo survival nor maximized post-hatching growth and survival, but appears to be a tradeoff between increasing embryo survival and maintaining growth and survival of late-hatched young (Sockman 2008, Aldredge et al. 2014). Finally, my last chapter (Chapter 4) discusses some overall conclusions from my dissertation and highlights what my studies contribute to our understanding of variation in offspring growth.

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CHAPTER 2: USING NON-LINEAR MIXED EFFECTS MODELS TO IDENTIFY PATTERNS OF CHICK GROWTH IN HOUSE SPARROWS

Summary

For many animals with determinate growth, adult size has an important influence on fitness. Thus, offspring that experience food restriction often delay development and then, following restriction, increase mass rapidly to approach an optimal size. These delayed growers can approach an optimal size by increasing mass faster than the peak growth of offspring that do not delay development (compensatory growth) or by extending the period of rapid growth to reach an optimal size (catch-up growth). Unfortunately, current statistical techniques make it difficult to identify these alternative growth patterns. Here, I show how to use random effect estimates from non-linear mixed effect models to identify variation in post-hatching growth in the house sparrow *Passer domesticus*. Specifically, I show that much of the between-individual variation in offspring growth can be explained by differences in the timing of peak growth and in final asymptotic mass. These results suggest that much of the variation in offspring growth may be explained by factors other than growth rate. I also show that offspring that delayed development either maintain a slow but steady growth rate across development and are relatively light at fledging or extended the period of rapid growth to reach the mass of offspring that do not delay development, indicative of catch-up growth. This pattern of maintaining a similar growth rate as offspring that do not delay development may allow these delayed growers to minimize cellular damage caused by compensatory

growth but still maximize size-related fitness benefits (e.g., increased survival and fecundity) prior to fledging.

Introduction

For many animals, adult size is an important determinant of fitness. Thus, growth often is optimized to increase size-related fitness traits (Blackenhorn 2005, Dmitriew 2010). In ideal conditions, growth is limited primarily by the ability of tissues to differentiate (Metcalfe and Monaghan 2001). However, conditions often are not ideal, resulting in considerable variation between individuals in their growth rates and final size (Schew and Ricklefs 1998). Individuals that experience nutritional deficits may delay growth until conditions improve and then increase mass rapidly to approach an optimal size (Hector and Nakagawa 2012).

Delayed growth occurs when individuals slow the rate at which tissues differentiate (Schew and Ricklefs 1998). Delayed growth can appear as a shift in the peak growth rate, and often results in a higher age-specific growth rate for offspring that delay development (i.e. suboptimal growers; Nicieza and Álvarez 2009). These suboptimal growers can approach the size of offspring that do not delay development (i.e. optimal growers) in one of three ways: 1) offspring can increase mass faster than the peak growth rate of optimal growers (compensatory growth), 2) offspring can extend the period of rapid growth to reach a similar size as optimal growers (catch-up growth) or 3) offspring can increase mass faster than the peak growth rate and extend the period of rapid growth to reach a similar size as optimal growers (compensatory + catch-up growth; Hector and Nakagawa 2012; Figure 2.1, Table 2.1). Compensatory growth can increase the

accumulation of cellular damage and reduce survival and fecundity (Mangel and Munch 2005). In contrast, catch-up growth may enable offspring to minimize the cellular damage caused by rapid growth and reproduce before the negative fitness consequences (e.g., reduced survival and fecundity) of delayed growth are realized (Wilbur and Rudolf 2006, Alonso-Alvarez et al. 2006, Drummond et al. 2003).

Like many animals, postnatal (i.e. post-hatching) growth in birds follows a sigmoidal pattern (Ricklefs 1967). The rapid growth of passerines is approximated best by the logistic growth function, which has three parameters: asymptotic mass (*A*), the location of the inflection point (*I*), and a growth rate constant (*K*). For studies that use the logistic growth function to examine differences in growth, researchers often use *K* as the single measurement of growth. However, comparisons of growth within species may benefit from analyzing multiple growth parameters simultaneously because of the potential importance of variation in timing of development (*I*) and pre-fledging mass (*A*; Sofaer et al. 2013). Within a species or population, nestlings that display optimal growth are characterized by an early (low) inflection point, fast (high) growth rate, and heavy (high) asymptotic mass. Alternatively, nestlings that display slow growth are characterized by a late (high) inflection point, slow (low) growth rate and light (low) asymptotic mass. Optimal and slow growth likely represent opposite ends of a continuum, making it important to identify ways to define alternative patterns of post-hatching growth (i.e. catch-up and compensatory growth).

Nonlinear mixed effects models may allow researchers to identify which aspects of growth can explain important variation in postnatal development (Sofaer et al. 2013). Mixed effects models analyze data at multiple levels to account for the correlated

structure in hierarchical datasets (Pinheiro and Bates 2000), as occurs when analyzing multiple measurements of the same individual or when analyzing measurements on groups of related individuals. At the first level, fixed effects are used to test the direct effect of some independent variable (e.g., population or treatment) on one or more aspects of growth (*A*, *I*, or *K*). At the second level, random effects are used to account for some of the variation in the hierarchically structured dataset that is not explained by the direct (fixed) effects of the model. As such, random effects often are used to understand individual variation in growth (Huchard et al. 2014, Vicenzi et al. 2014).

I used a nonlinear (logistic) mixed effects model to identify alternative growth patterns in the house sparrow *Passer domesticus*. Specifically, I used random effect estimates from a logistic growth function to identify alternative growth patterns in nestlings from a North Carolina breeding population. I tested whether offspring that delayed development exhibited compensatory, catch-up, or slow growth (Table 2.1). I controlled for differences in timing of development between optimal and suboptimal growers by calculating linear growth rates based on time since the period of peak growth (*I*). These development-specific growth rates show whether nestlings that delayed development increased mass faster than the peak growth rate of optimal growers, indicating compensatory growth, or simply shifted the timing of peak growth but maintained similar growth rates to those of optimal growers.

Methods

I studied nestling growth at two beef cattle farms near Yanceyville, North Carolina, USA (36.41^{o}N, 79.34^{o}W) from 2011 to 2014. Mean clutch size in this population was 4.80 (± 0.08; 1 SE) eggs, with a range of two to seven eggs per clutch.

The mean incubation period was 10.55 (± 0.08) days and the mean nestling period was 13.49 (± 0.24) days.

From 2011 to 2014, I studied growth for 362 nestlings from 81 nests. Nestling growth was measured as changes in mass because very young birds and their parents are highly sensitive to the presence of researchers at the nest. This sensitivity causes a trade-off in the amount of data that can be collected and the frequency with which those data are collected. I chose to collect mass data at a frequency sufficient to generate non-linear growth curves (almost daily) instead of collecting size measures at a lower frequency (two or three times over a 12d nestling period), which could not provide a continuous measure of offspring growth. Because increases in mass often are highly correlated with increases in offspring size during post-hatching development in birds, the measurements used in this study likely provide fairly robust estimates of offspring growth (Ricklefs 1969).

For all years of the study, I checked nests daily after females finished lining the nest until the first egg was laid. I recorded clutch size after females laid the last egg and checked nests five days after clutch completion. Beginning nine days after clutch completion, I visited nests at least once per day until all eggs hatched. I visited nests almost daily after hatching and weighed nestlings to the nearest 0.1g with a 60g Pesola scale. I identified individual nestlings by uniquely marking nestlings' toes until they were banded with a uniquely numbered identification band approximately seven days post-hatch. Because nestlings can fledge prematurely several days before the end of the nestling period, I stopped weighing nestlings after a majority of the nestlings in a brood failed to gain mass between two successive measurement days, which typically occurred

between 10 and 12 days post-hatch. The number of mass measurements per individual varied from 1 to 13, with a median of 9.5 measurements per nestling.

Statistical Analysis

All analyses were done using R 2.14.0 (R Development Core Team 2014). To evaluate a set of candidate models, I chose the model with the lowest Akaike Information Criterion (AIC) value (Burnham and Anderson 2002). All parameter estimates are reported as the mean ± 1 SE.

Identifying alternate growth patterns using nonlinear mixed models

I wrote and implemented a self-starting function for the logistic curve typically used to analyze growth of passerine nestlings (Starck and Ricklefs 1998):

$$w_t = \frac{A}{1 + e^{K(I - t)}}$$

where w_t = mass at time t (g), A = asymptotic mass (g), K = growth rate constant (1/day), I = the inflection point of the growth curve (days), and t = nestling age (days). I fit all logistic growth curves using maximum likelihood in the nlme package of R (Pinheiro *et al*. 2011). In order to control for the non-independence of nestlings within the same nest and repeated measurements of individual nestlings, these models included nestling identity nested within brood as a random intercept. I included the three growth parameters (*A*, *I*, and *K*) as fixed effects and used the AIC value to determine which combination of growth parameters also should be included as random effects. Models that contained both *I* and *K* as random effects either failed to converge on a solution or exhibited a high correlation ($r > 0.9$). Thus, I chose the best model that contained random effects for either *I* or *K*.

Logistic growth parameters that did not include random effects were characterized

by a fixed effect estimate only. In contrast, growth parameters that included random effects were characterized by one fixed effect and one or more random effects (Table 2.2). For all models, the fixed effect (e.g., A) estimated the growth parameter of a typical (e.g. median) nestling from the population (Table 2.3). The brood-level random effect (e.g., A_i) estimated how the growth parameter of a typical nestling from an individual brood deviates from a typical nestling in the population (Table 2.3). The nestling-level random effect (e.g., A_{ij}) estimated how the growth parameter of an individual nestling deviates from a typical nestling in its brood (Table 2.3). Thus, the sum of the brood- and nestling-level random effects estimated how the growth parameter of an individual nestling deviates from a typical nestling in the population. I show results using the sum of the brood- and nestling-level random effects because I am interested in the variation in growth among individuals in this population.

Characterizing individuals as optimal, delayed or slow growers

I used random effect estimates (sum of brood- and nestling-level estimates) to identify alternative growth patterns in the house sparrow. I divided nestlings that survived the nestling period into one of three growth patterns: optimal, delayed, and slow growth. Nestlings exhibited optimal growth if the random effect estimate for the inflection point was less than one standard deviation above the fixed effect estimate of the population (indicating a normal to early inflection point) and the random effect estimate for asymptotic mass was greater than one standard deviation below the fixed effect estimate (indicating normal to heavy asymptotic mass; Figure 2.2). Nestlings exhibited delayed growth if the random effect estimate for the inflection point was greater than one standard deviation above the fixed effect estimate (indicating a late inflection point) and the

random effect estimate for the asymptotic mass was greater than one standard deviation below the fixed effect estimate (indicating normal to heavy asymptotic mass; Figure 2.2). These delayed growers are likely candidates for catch-up or compensatory growth. Nestlings exhibited slow growth if the random effect estimate of the asymptotic mass was less than one standard deviation below the fixed effect estimate (indicating a light asymptotic mass independent of the inflection point; Figure 2.2). I used a linear mixed effects model with clutch as a random intercept to determine whether a relationship existed between the random effect estimates.

Identifying growth patterns of suboptimal growers

To determine whether suboptimal (i.e. delayed and slow) growers displayed compensatory, catch-up or slow growth, I used a linear mixed effects model with nestling identity nested within brood as a random intercept and nestling identity as a random coefficient for day to investigate whether growth rate differed between optimal, delayed and slow growers. I divided the nestling period into four distinct growth phases (early, peak, late, and asymptotic growth) that corresponded to periods of approximately linear growth. The inflection point of each growth pattern was used to delineate these growth phases, which controlled for the shift in timing of peak growth for optimal, delayed and slow growers. The early growth rate was estimated as the linear change in mass from three to five days before the inflection point of each growth pattern. The peak growth rate was estimated as the linear change in mass that occurred three days before to three days after the inflection point of each growth pattern. This growth phase identified whether offspring displayed compensatory growth. The late growth rate was estimated as the linear change in mass from three to five days after the inflection point of each growth

pattern. Finally, the asymptotic growth rate was estimated as the linear change in mass from five to seven days after the inflection point of each growth pattern.

Results

Identifying alternate growth patterns using nonlinear mixed models

The nonlinear mixed effects model that included both brood- and nestling-level random effects for the asymptotic mass (*A*) and inflection point (*I*) was the best model (Table 2.2). The brood-level random effect explained more of the residual variation than the nestling-level random effect for both *A* and *I* (Table 2.4). A negative relationship existed between *A* and *I* (coefficient estimate: -2.53 ± 0.20; $t = -12.91$, $P < 0.001$), suggesting that light nestlings in this population reached their inflection point later than heavy nestlings (Figure 2.2).

Most nestlings that survived to fledge (71.3%; n = 239) displayed optimal growth. Approximately half of suboptimal growers (14.6% of surviving young; n = 49) were characterized by delayed growth, and the other half (14.0% of surviving young; n = 47) were characterized by slow growth. The inflection point was shifted -0.22, 0.98, and 0.36 days from the population-level fixed effect (4.44d) for optimal, delayed and slow growers, respectively. The asymptotic mass was shifted 0.77, -0.19, and -5.93g from the population-level fixed effect (26.63g) for optimal, delayed and slow growers, respectively.

Identifying growth patterns of suboptimal growers

Optimal growers increased mass faster than suboptimal growers (indicating no compensatory growth) during the peak growth phase. All suboptimal growers extended the period of rapid mass increase, but only delayed growers approached a similar mass as

optimal growers (indicating catch-up growth). Nestlings that displayed optimal growth increased mass more rapidly during the early growth phase (2.10 ± 0.04 g/d) than nestlings that displayed delayed (1.85 ± 0.08 g/d; t = 2.68, P = 0.008) and slow growth (1.42 ± 0.08 g/d; t = 7.31, P < 0.001; Figure 2.3). Nestlings that displayed optimal growth also increased mass more rapidly during the peak growth phase (2.91 ± 0.03 g/d) than nestlings that displayed delayed (2.57 ± 0.07 g/d; t = 4.65, P = 0.241) and slow growth (1.79 ± 0.07 g/d; t = 15.35, P < 0.001; Figure 2.3). Nestlings that displayed optimal and slow growth increased mass similarly (optimal: 1.59 ± 0.07 g/d; slow: 1.58 ± 0.15 g/d; t = 0.04, P = 0.970) but more slowly than nestlings that displayed delayed growth (2.16 ± 0.14 g/d; t = 3.74, P < 0.001; Figure 2.3) during the late growth phase. Finally, nestlings that displayed optimal growth increased mass more slowly during the asymptotic growth phase (0.34 ± 0.08 g/d) than nestlings that displayed delayed growth (0.73 ± 0.16 g/d; t = -2.22, P = 0.027), and nestlings that displayed delayed growth increased mass more slowly than nestlings that displayed slow growth (1.73 ± 0.17 g/d; t = 4.23, P < 0.001; Figure 2.3).

Discussion

I have shown how random effect estimates from nonlinear mixed effects models can be used to identify important variation in post-hatching growth. Specifically, asymptotic mass (*A*) and timing of peak growth (*I*) exhibit substantial variation among nestlings within a single population. In addition, more of the variation within this population can be explained by *A* and *I* than by *K*, which supports current theory that little variation exists around the optimal growth rate. These findings are consistent with a similar study, which showed that random effects for *A* and *I* explain much of the variation

in growth not explained by differences between populations (Sofaer et al. 2013). Thus, these results suggest that nonlinear mixed effects models may be a powerful analytical technique to identify important, but previously undetected variation in offspring growth.

Using a mixed modeling approach, I found evidence of catch-up growth in wild house sparrows, a strategy in which nestlings shifted the timing of peak growth and extended the period of rapid development to reach a similar mass as optimal growers (Lepczyk and Karasov 2000). I found no evidence of compensatory growth, likely because growth has been optimized evolutionarily at the peak growth rate of optimal growers (Ricklefs 1979). Some offspring maintained a fairly stable but slow growth rate and reached a light asymptotic mass prior to fledging. Despite the differences in asymptotic mass between nestlings that displayed catch-up and slow growth, all suboptimal growers increased mass faster than optimal growers toward the end of development.

Several studies in both wild and captive populations have shown that passerines can delay the period of peak growth but reach an optimal size prior to fledging (e.g. Lepczyk and Karasov 2000, Hegyi and Torok 2007, Alonso-Alvarez et al. 2007, Criscuolo et al. 2008). However, there is little direct evidence that passerines increase mass faster than the peak growth rate of optimal growers (although the results from at least one study suggest that compensatory growth may be possible (Killpack et al. 2014). Instead, most studies have shown that nestlings approach the mass of optimal growers by reaching a faster age-specific growth rate. I have shown that faster age-specific growth rates may occur as a consequence of offspring delaying development, and hence shifting the period of peak growth (Schew and Ricklefs 1998). Thus, future studies should control

for differences in timing of development and test directly whether offspring that delay development increase mass faster than optimal growers (Nicieza and Álvarez 2009), which will indicate whether offspring exhibit compensatory growth.

To control for differences in timing of development, researchers need to consider the trajectory of postnatal growth (Dmitriew 2010, Hector and Nakagawa 2012). In fact, some form of all three growth parameters (timing of peak growth [*I*], peak growth rate [*K*], and final mass [*A*]) can distinguish between alternative growth patterns (Table 2.1), even in studies that do not use nonlinear growth functions. Studies first need to determine whether offspring have shifted the timing of peak growth (e.g. delayed growth). For analyses using nonlinear growth functions, a shift in the location of the inflection point may indicate that offspring have delayed growth temporarily. For analyses not using nonlinear growth functions, delayed growth can be identified by a delay (or suspension) of growth during food restriction. Next, the peak growth rate can be estimated to identify whether offspring that delayed development increased mass faster than the peak growth rate of optimal growers (exhibit compensatory growth). By controlling for differences in timing of development, peak growth rates will be estimated at later ages for offspring that delayed development, which likely enable comparisons of growth at similar developmental stages (Nicieza and Álvarez 2009). Ideally, estimates of the peak growth rate should be averaged over several days and should be calculated during periods of linear growth. Finally, estimates of the final mass distinguish between offspring who attain a similar mass as optimal growers (exhibit catch-up growth) or those who are relatively light prior to fledging. Estimates of each of these growth parameters are derived easily from nonlinear mixed effects models, but also can be estimated by

concentrating mass measurements around the period of peak growth and the attainment of the asymptotic mass.

In this study, post-hatching growth was estimated as changes in offspring mass, which makes it difficult to compare these results with studies that focus on developmental changes in offspring size. Although increases in mass and size often are correlated in altricial birds during development (Ricklefs 1969), nestlings can increase size somewhat independently of post-hatching mass (Zach and Mayoh 1982, Skagen 1987). Because strong selection exists for altricial birds to reach a threshold size prior to fledging, offspring often bias investment into increasing size more than mass when food availability is low (Nilsson and Svensson 1996). Despite the importance of reaching a threshold size, much of the research investigating how post-hatching growth influences both short- and long-term survival in birds has been done using developmental differences in offspring mass. This likely occurs because mass provides an estimate of growth that is quick and easy to quantify in free-living birds. Previous studies have shown that offspring mass is an important predictor of post-fledging survival and recruitment into the breeding population (Magrath 1991, Mumme et al. 2015), suggesting that it may be as important that offspring reach a threshold mass by the end of post-hatching development than it is that they reach a threshold size.

The mechanisms by which compensatory growth reduces future survival and reproduction remain unclear (Mangel and Munch 2005). A potential explanation is that compensatory growth causes oxidative stress by increasing the production of reactive oxygen species (De Block and Stoks 2008), molecules associated with metabolism that can damage DNA, proteins and lipids (Finkel and Holbrook 2000). Additionally,

compensatory growth is thought to reduce future survival by shortening the length of telomeres (Geiger et al. 2012, Nettle et al. 2015) or by causing oxidative damage (Alonso-Alvarez et al. 2007), which can increase the rate of cell death, negatively affect normal cell cycle functioning, and decrease resistance to disease (Mangel and Munch 2005). In addition to negative effects at the cellular level, compensatory growth may cause developing offspring to produce morphological traits that reduce locomotor performance and increase susceptibility to predation (Arendt et al. 2001, Ficetola and De Bernardi 2006) or impair brain development and reduce performance of learning and other cognitive tasks (Fisher et al. 2006). Despite the lack of a clear understanding of the mechanism, compensatory growth has been shown to reduce future survival (Metcalfe and Monaghan 2003) and reproduction (Auer et al. 2010), and thus should be avoided unless the fitness benefits are relatively high. This may explain why house sparrow nestlings that delayed development did not display compensatory growth by increasing mass faster than optimal growers, but extended the period of rapid growth to reach a similar asymptotic mass prior to fledging.

It is unclear how suboptimal growth influences post-fledging survival and reproduction in this population of house sparrows. Survival until the first breeding season is strongly associated with mass near fledging in altricial birds (Schwagmeyer and Mock 2008, Dybala et al. 2013, Bouwhuis et al. 2015). I have shown that nestlings that exhibit suboptimal growth appear to extend the period of rapid growth to maximize pre-fledging mass, but only some of these nestlings reach a similar mass as optimal growers prior to fledging. Thus, the fitness costs of delaying development and extending the period of

rapid growth may not be realized in the short-lived house sparrow, but the potential fitness benefits of increasing pre-fledging mass likely are important.

House sparrows are cavity nesters and may experience relaxed selection pressure to fledge quickly. Low predation risk could enable house sparrows to slow growth temporarily and extend the developmental period in some nestlings (Schew and Ricklefs 1998, Bize *et al.* 2006), but still maintain an optimal growth rate and short developmental period for a core group of offspring (Forbes et al. 1997). The smallest, often last-hatched, young typically fledge one or more days after the other nestlings in the brood (Aldredge, personal observation). Thus, low predation risk may enable offspring to increase the variation in growth and timing of fledging, and might explain why clutch size is larger (Jetz et al. 2008) and developmental periods longer (von Haartman 1957) in cavity than in open cup nesters. Future studies should examine whether catch-up growth is more common than previously recognized in passerines, and whether this growth pattern occurs more frequently in species that have a low risk of nest predation. In addition, future research should test directly whether passerines are able to exhibit compensatory growth and increase mass faster than the peak growth rate of optimal growers. By using random effect estimates from nonlinear mixed effects models, researchers may be able to tease apart important differences in offspring growth and uncover developmental strategies that have evolved to maximize fitness.

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Table 2.1: Change in growth parameters between nestlings that exhibit optimal (not shown) and suboptimal growth. All suboptimal growers delay the timing of peak growth [Timing (I)]. In addition, compensatory growers develop faster than the peak growth rate [Growth (K)] of optimal growers, and offspring that exhibit catch-up growth reach an optimal size [Size (A)]. Slow growers increase mass slowly and reach a small adult size.

	Growth parameter		
Growth pattern	Timing (I)	Growth (K)	Size (A)
Catch-up	Delay	Slower/No change	No change
Compensatory	Delay	Faster	Smaller
Catch-up + Compensatory	Delay	Faster	No change
Slow	Delay	Slower	Smaller

Table 2.2: Model selection for the random effect structure of the nonlinear (logistic) mixed effects model examining post-hatching growth within a breeding population in North Carolina. Each model included a population-level fixed effect (e.g., A) for each growth parameter and clutch-level (e.g., A_i) and nestling-level (e.g., A_{ij}) random effects for at least one of the three growth parameters: asymptotic mass (A), inflection point (I), and growth rate (K). The best model included random effects for A and I. The model that included random effects for all three growth parameters failed to converge on a solution (NA), likely because I and K were highly correlated.

MODEL	Random Effects	AIC	ΔAIC
$w_{ijk} = \frac{A + A_i + A_{ij}}{1 + e^{K*((I+I_i+I_{ij})-t_{ijk})}} + \mathcal{E}_{ijk}$	A, I	12107	0
$w_{ijk} = \frac{A + A_i + A_{ij}}{1 + e^{(K+K_i+K_{ij})*(I-t_{ijk})}} + \mathcal{E}_{ijk}$	A, K	13298	1191
$w_{ijk} = \frac{A + A_i + A_{ij}}{1 + e^{K*(I-t_{ijk})}} + \mathcal{E}_{ijk}$	A	13485	1378
$w_{ijk} = \frac{A}{1 + e^{K*((I+I_i+I_{ij})-t_{ijk})}} + \mathcal{E}_{ijk}$	I	13860	1753
$w_{ijk} = \frac{A}{1 + e^{(K+K_i+K_{ij})*(I-t_{ijk})}} + \mathcal{E}_{ijk}$	K	16427	4320

Table 2.3: How the nonlinear mixed effects model partitions the variation in asymptotic mass. The asymptotic mass (A) for an individual (Ind) is the sum of the fixed effect estimate for population (Pop FE), random effect estimate for each brood (Brood RE), and random effect estimate for each nestling (Nstl RE). The brood-level random effect shows how much a typical nestling from a given brood deviates from a typical nestling in the population. The nestling-level random effect shows how much an individual nestling deviates from a typical nestling in its brood, and the sum of the brood- and nestling-level random effects (not shown) identifies how much an individual nestling deviates from a typical nestling in the population. Growth parameters that do not include any random effects were characterized by the fixed effects only.

Ind	Nest	Pop FE	Brood RE	Nstl RE	A
1	1	26.63	-0.08	1.72	28.3
2	1	26.63	-0.08	1.82	28.4
3	1	26.63	-0.08	-0.53	26.0
4	1	26.63	-0.08	-3.18	23.4
5	2	26.63	0.86	0.04	27.5
6	2	26.63	0.86	0.66	28.2
7	2	26.63	0.86	-0.55	26.9
8	2	26.63	0.86	0.59	28.1
9	3	26.63	-1.36	-0.55	24.7
10	3	26.63	-1.36	0.63	25.9
11	3	26.63	-1.36	0.82	26.1
12	3	26.63	-1.36	-1.19	24.1
13	4	26.63	4.52	-0.27	30.9
14	4	26.63	4.52	0.29	31.4
15	4	26.63	4.52	0.93	32.1
16	4	26.63	4.52	0.97	32.1

Table 2.4: Table showing the variance (Var), standard deviation (Std dev), and correlation between the random effects (Corr) for asymptotic mass (A) and inflection point (I) at both the brood (Brood) and nestling (Nstl) levels. Overall, the brood-level RE explained more of the residual variation than the nestling-level RE for both A and I.

RE	Parameter	Var	Std dev	Corr
Brood	A	7.60	2.76	
	I	0.33	0.58	0.048
Nstl	A	3.21	1.79	
	I	0.15	0.39	-0.359
	Residual	1.09	1.04	

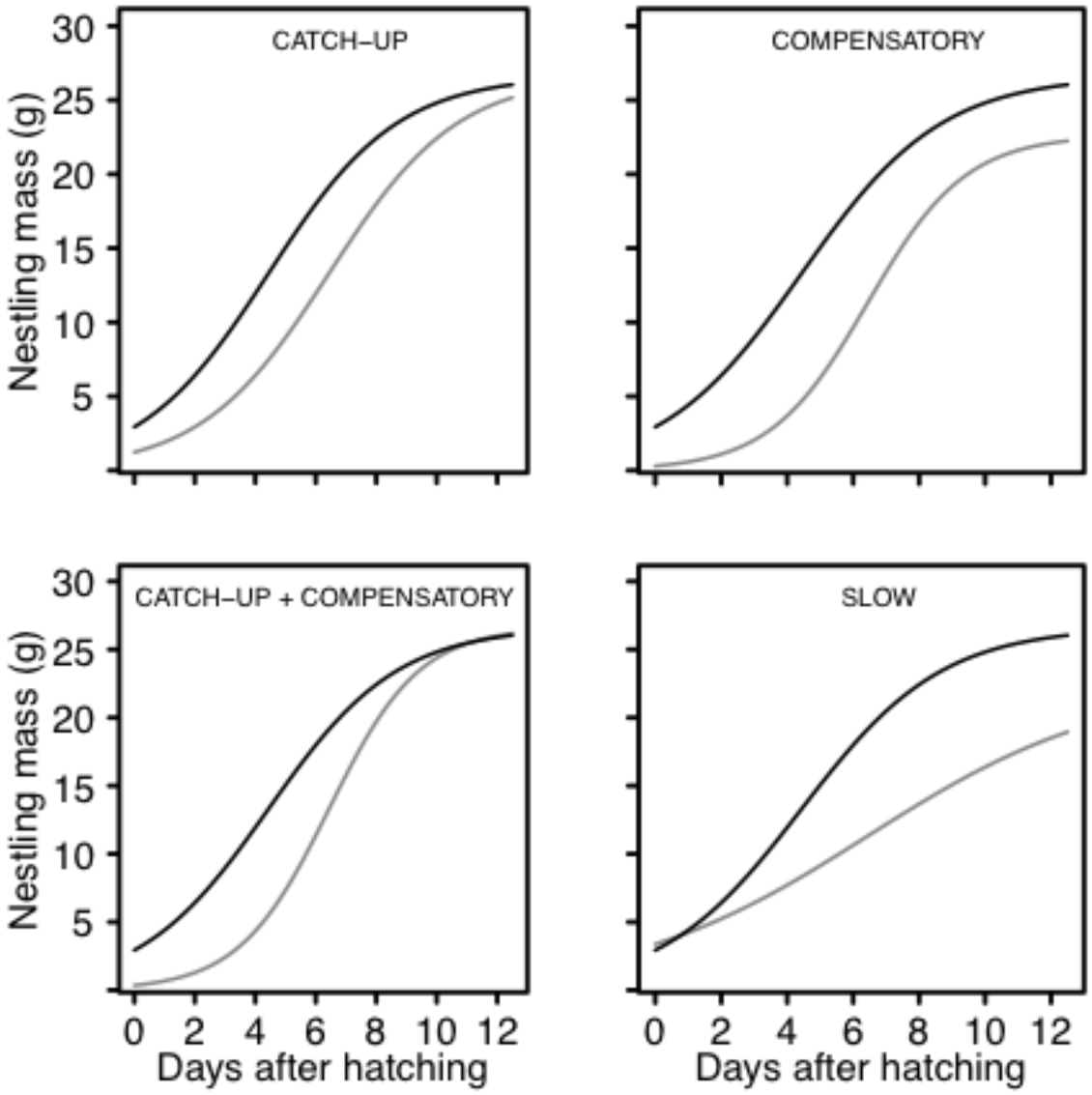

Figure 2.1: Theoretical growth patterns for offspring displaying optimal (black line) and suboptimal growth (grey lines). Offspring that displayed optimal growth had a growth rate (K) of 0.47, reached their period of peak growth (I) at 4.44d and reached an asymptotic mass (A) of 26.63g. All suboptimal growers delayed the period of peak growth by 2 days (i.e. 6.44d) compared to optimal growers. In addition, offspring that displayed catch-up growth (upper left panel) had the same growth rate and reached the same asymptotic mass as optimal growers. Offspring that displayed compensatory growth (upper right panel) had a faster growth rate (0.67) and reached a lighter asymptotic mass (22.63g) than optimal growers. Offspring that displayed catch-up and compensatory growth (lower left panel) had a faster growth rate (0.67) but reached the same asymptotic mass as optimal growers. Finally, offspring that displayed slow growth (lower right panel) had a slower growth rate (0.27) and reached a lighter asymptotic mass (22.63g) than optimal growers.

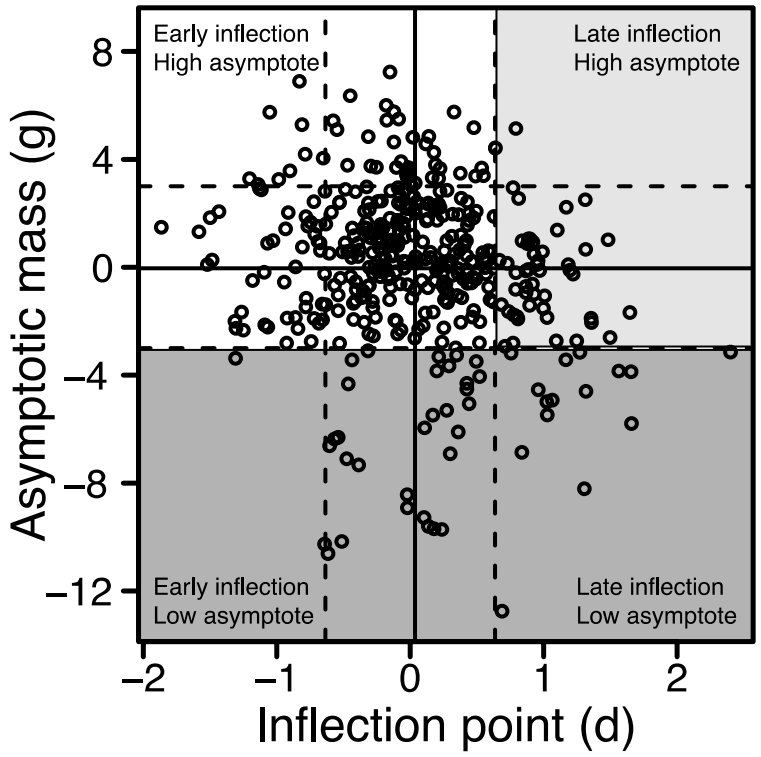

Figure 2.2: Variation in growth within a North Carolina breeding population. Points indicate random effect estimates of the asymptotic mass and location of the inflection point. Solid lines indicate nestlings that did not differ from the population-level fixed effect estimates and dashed lines indicate random effect estimates that were one standard deviation away from the population-level fixed effect estimates. Nestlings that displayed optimal growth (white box) reached a normal to early inflection point and normal to high asymptotic mass. Nestlings that displayed delayed growth (light grey box) reached a late inflection point and a normal to high asymptotic mass. Nestlings that displayed slow growth (dark grey box) reached a light asymptotic mass independent of the location of the inflection point.

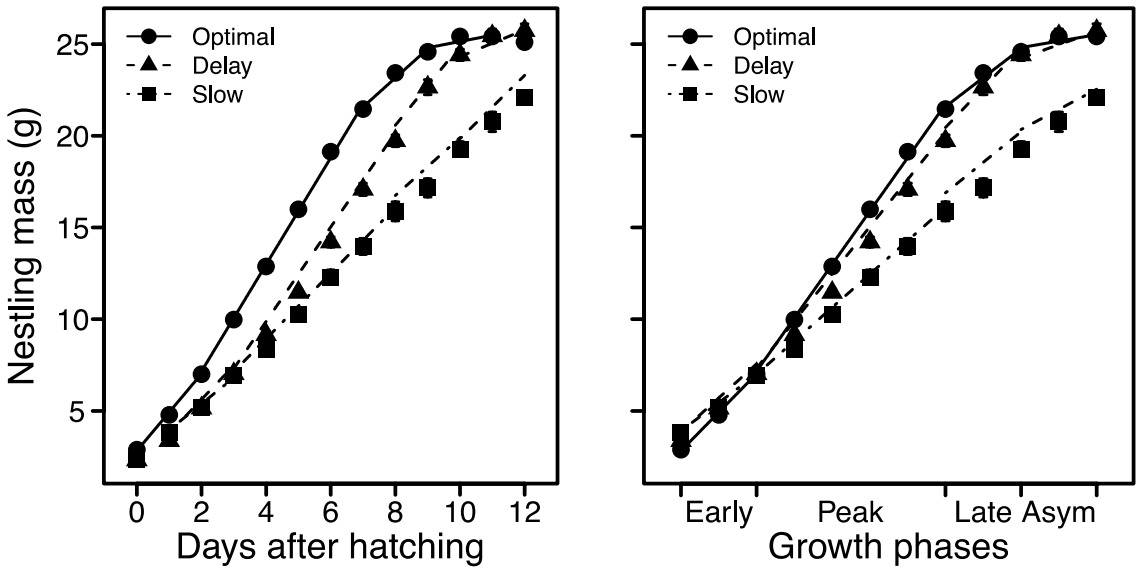

Figure 2.3: Linear growth rates at four different growth phases [Early, Peak, Late, and asymptotic (Asym)] for optimal (Optimal; circle, solid line), delayed (Delay; triangle, dashed line) and slow growers (Slow; square, dot-dash line). The left panel shows linear growth rates for absolute nestling age and the right panel shows linear growth rates after controlling for the shift in peak growth by delayed and slow growers. Nestlings that displayed optimal growth reached their period of peak growth earlier and growth rate also started to decline earlier than nestlings that displayed delayed and slow growth. Nestlings that displayed delayed growth did not reach a higher peak growth rate (no compensatory growth) but reached an asymptotic mass that was similar to optimal growing nestlings. Points and error bars are derived from actual data and lines are derived from estimates of the best fit model.

CHAPTER 3: INCUBATION ONSET IS A TRADEOFF BETWEEN MAINTAINING BOTH EMBRYO SURVIVAL AND GROWTH AND SURVIVAL OF LATE-HATCHED YOUNG

Summary

Hatching (or birth) asynchrony occurs primarily as a consequence of the timing of embryonic development (or parturition). Despite over 50 years of study, it is unclear why, ultimately, most birds initiate embryonic development (incubation) before all eggs are laid. One hypothesis focuses on pre-hatching (embryo) survival and predicts that early incubation maximizes embryo survival by reducing exposure of unincubated eggs (egg viability hypothesis). Another set of hypotheses focuses on post-hatching growth and survival and predicts that females time incubation to maximize the number or quality of hatched offspring that fledge (adaptive hatching pattern hypotheses). I experimentally manipulated when females could begin incubation to test how timing of embryonic development influences pre-hatching survival and post-hatching growth and survival in the house sparrow *Passer domesticus*. I found that early incubation maximized embryo survival in all but the earliest-laid eggs, suggesting that house sparrows begin incubation too late to maximize survival of all embryos. Early incubation had little effect on overall (i.e. mean) patterns of post-hatching growth and survival. However, early incubation increased the initial variation in offspring size because last-hatched young were relatively small when all eggs had completed hatching. Nestlings that were small at hatch completion grew slowly and exhibited a reduced probability of survival, suggesting that

house sparrows begin incubation too early to maximize growth and survival of hatched offspring. These results suggest that timing of incubation neither maximizes embryo survival nor maximizes post-hatching growth and survival. Instead, early incubation appears to be a tradeoff between maintaining both embryo survival and growth and survival of late-hatched offspring. Thus, house sparrow females likely time incubation as an adaptive strategy to maximize the number of embryos that survive the incubation and nestling periods to fledge.

Introduction

Hatching (or birth) asynchrony occurs when siblings of the same cohort hatch (or are born) at different times (Stenning 1996). Hatching asynchrony in oviparous species is caused primarily by differences in the timing of embryonic development associated with sequential ovulation (While et al. 2007, Sockman 2008), whereas birth asynchrony in viviparous species can be caused either by differences in timing of embryonic development (Gilmore 1993) or by differences in timing of birth associated with sequential parturition (Fraser et al. 1979). Hatching (or birth) asynchrony often increases the variation in offspring size at hatching, a pattern that can have important consequences for parental and offspring fitness.

Although it occurs in diverse taxa, including mammals (Fraser et al. 1979), reptiles (Duffield and Bull 1996, Chapple 2005), lamnoid sharks (Gilmore 1993), and insects (Smiseth et al. 2006), hatching asynchrony has been studied primarily in birds. Birds not only ovulate sequentially but also have lost the ability to retain shelled eggs *in utero*, which causes all birds to lay eggs sequentially (Blackburn and Evans 1986). Birds

lay at most one egg per day and in nearly all species, embryonic development is driven by parental incubation (Avise 2013). Instead of beginning incubation after all eggs are laid, most species initiate incubation before clutch completion, which may cause eggs to hatch over one or more days (Clark and Wilson 1981).

The relationship between timing of incubation and hatching asynchrony differs considerably among birds, based primarily on the species' developmental mode (Clark and Wilson 1981, Magrath 1990). Precocial species often begin incubation one or more days before clutch completion, but hatching patterns do not always reflect timing of incubation (Freeman and Vince 1974, Persson and Andersson 1999). Instead, embryos of most, but not all (Hussell and Page 1976), precocial species produce clicking noises at the end of embryonic development (McCoshen and Thompson 1968), a behavior that synchronizes development, and hence hatching within a clutch (Vince 1968). This hatching synchronization maximizes both parental and offspring fitness by increasing the number of hatched young that leave the nest (fledge). In contrast, no altricial species have been shown to use inter-egg communication to synchronize development, and hatching patterns often reflect timing of parental incubation (Magrath 1990, Wiebe et al. 1998, but see Bortolotti and Wiebe 1993). Thus, many altricial birds exhibit extensive hatching asynchrony as a result of early incubation onset (Clark and Wilson 1981). Unlike in precocial species, altricial offspring compete to monopolize parental care after hatching, which maximizes direct fitness of individual offspring but decreases indirect and possibly parental fitness (Mock 1985, Cotton et al. 1999, Ricklefs 2002). Thus, altricial offspring presumably have abandoned the ability to synchronize hatching and evolved in a competitive environment where each chick strives to maximize its own rate of embryonic

(pre-hatching) and post-hatching development. This may explain why the variation in offspring size caused by early incubation is maintained in altricial birds (Ricklefs, 1969, Ricklefs 1984, Ricklefs 1993), but also leads to the long-standing question: Why, ultimately, is early incubation, and hence hatching asynchrony, widespread in altricial species?

Hatching asynchrony was first described over a half century ago as an adaptive strategy that enables parents to selectively feed young based on size and maximize offspring production when food is abundant, but also quickly reduce the brood to a manageable size when food is limiting (Lack 1947, Ricklefs 1965). By beginning incubation before all eggs are laid, females increase the variation in offspring size at hatching, and as a consequence often increase the variation in offspring growth and survival. The increased variation in growth and survival caused by hatching asynchrony is thought to be maintained in most altricial species because it increases the number of high quality young that survive to breed (Lack 1954, 1966). Since its first description, the brood reduction hypothesis has spawned over a dozen similar hypotheses that attempt to explain the adaptive significance of hatching asynchrony (reviewed in Magrath 1990 and Stoleson and Beissinger 1995). Many of these hypotheses focus on how parents regulate timing of hatching to maximize the number or quality of hatched young that fledge. Collectively, they are called adaptive hatching pattern hypotheses and although many hypotheses have been proposed, none have clear experimental support that is applicable to the diversity of species that begin incubation prior to clutch completion (Stoleson and Beissinger 1997). Studies that test these hypotheses often include the experimental creation of within-nest variation in offspring size by exchanging nestlings between nests

to simulate synchronously and asynchronously hatching clutches (Magrath 1990, Stoleson and Beissinger 1995). These studies predict that synchronously hatching clutches will: 1) produce fewer offspring that survive to fledge, 2) produce lower quality (e.g. smaller or lighter) offspring at or near fledging, or 3) produce offspring that exhibit lower post-fledging survival (Stoleson and Beissinger 1997). Overall, hatching asynchrony may increase post-hatching growth and survival in environments or years when food is limiting (Magrath 1989, Wiebe and Bortolotti 1994, Amundsen and Slagsvold 1998, Hebert and McNeil 1999, Forbes et al. 2001, Podlas and Richner 2013), but this likely does not explain why most birds, including precocial species whose eggs hatch synchronously, begin incubation prior to clutch completion (Clark and Wilson 1981, Arnold et al. 1987). Furthermore, tests of the adaptive hatching pattern hypotheses often fail to account for the effects of incubation behavior on embryo survival (Viñuela 2000).

Recent evidence suggests that prolonged exposure of undeveloped embryos (i.e. unincubated eggs) increases the risk of embryo mortality (the egg viability hypothesis; Arnold et al. 1987, Veiga 1992). Among those risks, exposure to pathogens (Cook et al. 2003, Godard et al. 2007, Shawkey et al. 2009) and suboptimal temperatures (Webb 1987, Hebert 2002) are most commonly cited (but see Beissinger et al. 2005). Although species and populations may differ in the amount of exposure that increases embryo mortality, experimental (Arnold et al. 1987, Veiga 1992, Veiga and Viñuela 1993, Arnold 1993, Stoleson and Beissinger 1999, Viñuela 2000, Beissinger et al. 2005, Walls et al. 2011, Wang et al. 2011) and observational (Sockman 2008, Wang and Beissinger 2009, Aldredge et al. 2012) evidence shows that females can reduce embryo mortality (hatching

failure) by beginning incubation before clutch completion, which decreases the length of exposure for undeveloped embryos. Hatching failure increases in as few as two days of exposure, which is the time required to lay a 3-egg clutch in many species (Beissinger et al. 2005). Regardless of the mechanism, embryo mortality increases when eggs are exposed to the environment for prolonged periods prior to incubation, which can have important consequences for parental fitness.

I experimentally manipulated when females could begin incubation to test predictions of the egg viability and adaptive hatching pattern hypotheses in the house sparrow (*Passer domesticus*). The house sparrow, like many temperate breeding passerines, typically lays clutches of four to six eggs and displays extensive variation in relative hatching times (Anderson 2006). In addition, the house sparrow was the first altricial species shown to exhibit declining egg viability with increased exposure prior to incubation (Veiga 1992). Thus, the house sparrow is an appropriate species to examine predictions of both the egg viability and adaptive hatching pattern hypotheses in the same study. I compared patterns of embryo survival and post-hatching growth and survival between clutches in which incubation was naturally asynchronous and clutches in which I experimentally synchronized incubation. If the egg viability hypothesis is correct, then hatching success should be lowest in early-laid eggs because these eggs would experience increased exposure prior to incubation. In addition, hatching success should be lower in experimentally synchronized nests because experimental eggs experience increased exposure relative to eggs from naturally asynchronous nests. If the adaptive hatching pattern hypotheses are correct, then naturally asynchronous nests should either produce

more fledged young or should produce more high quality young that fledge compared to experimentally synchronized nests.

In most altricial species, early incubation influences post-hatching growth and survival primarily by increasing the variation in offspring size at hatching (Clark and Wilson 1981). Because even experimentally synchronized clutches exhibit some variation in size caused by asynchronous hatching (Harper et al. 1993), I also examined how timing of incubation influenced the variation in offspring size at hatching and then whether this variation was associated with differences in post-hatching growth and survival. To quantify the variation in offspring size, I used the mass difference (in grams) between an individual chick and the heaviest nestling in the brood one day after the first egg hatched, which provides a direct and ecologically relevant estimate of a nestling's mass relative to its nestmate(s) when all eggs in a clutch had completed hatching (hereafter referred to as hatch completion). The heaviest nestling in the brood had no mass difference (0g), and a large mass difference indicated that a nestling was relatively light at hatch completion. I predicted that naturally asynchronous nests would exhibit greater variation in offspring size at hatch completion because these nests would contain more relatively light nestlings. I also predicted that the increased variation would enable nestlings from asynchronous nests to grow better, on average, than nestlings from experimentally synchronized nests.

Although both the egg viability and adaptive hatching pattern hypotheses attempt to explain why, ultimately, early incubation occurs, they are not mutually exclusive. Indeed, it is possible that naturally asynchronous nests exhibit both reduced embryo survival in early-laid eggs and reduced growth and survival of late-hatched young caused

by hatching asynchrony. Such a result would suggest that early incubation does not support either hypothesis but likely occurs as a tradeoff between maintaining both embryo survival and post-hatching survival and growth (Sockman 2008, Aldredge et al. 2014).

Methods

I studied a population of house sparrows near Yanceyville, North Carolina (36.41^{o}N, 79.34^{o}W) during the breeding seasons (late March-late June) of 2013 and 2014. This population has been studied since 2009 and contains approximately 35 wooden nestboxes over ~15,500m^2, half to two-thirds of which are occupied at some point during a single breeding season. In 2013 and 2014, I checked nests daily from nest completion (nest lined) to the day the female laid the last egg (clutch completion). When a female laid the first egg of her first clutch, I randomly assigned the nest either to an experimental or control treatment. When females re-laid after successfully fledging young, I assigned the second clutch to the opposite treatment of the first. Less than half of females (14 of 33 females) relaid a second clutch after successfully fledging young. I used 205 eggs from 41 clutches (91 eggs from 19 control nests and 114 eggs from 23 experimental nests) to examine predictions of the egg viability hypothesis. For the experimental treatment, each egg was marked with an indelible marker to identify laying order and replaced with a dummy egg on the day it was laid and then carried to a nestbox that was 100-200m from the focal nest and open to the environment but inaccessible to predators, house sparrows, or other birds. Each clutch of eggs was placed in an artificial nest cup lined with fresh cotton. When females laid the last egg, which can be identified by the diffuse spotting on the last-laid egg (Lowther 1988), I returned each clutch of eggs to the focal nest where it

was exchanged with the clutch of dummy eggs. Thus, incubation was delayed until clutch completion in experimental clutches. For the control treatment, each egg was marked with an indelible marker, replaced with a dummy egg and carried approximately 100m from the nest before returning it to the focal nest and exchanging it with the dummy egg. This procedure controlled for the transportation and handling manipulation but allowed females to begin incubation prior to clutch completion. Unfortunately, eggs from experimental nests were moved twice (i.e. once at laying and once at clutch completion) during the manipulation whereas control eggs were moved only once at laying. This difference in egg handling between experimental and control nests allowed me to avoid jostling embryos from control eggs that had initiated embryonic development. Because embryos from experimental eggs should not have initiated development prior to the second handling at clutch completion, this difference should have no effect on embryo survival.

Beginning nine days after clutch completion, I visited nests once in the morning and once in the evening until at least one egg hatched and then visited these nests four to five times daily (approximately every three to six hours during daylight) until all viable eggs hatched. I identified laying order of individual nestlings, when known, uniquely clipped nestlings' toenails for individual identification, and weighed all nestlings in a clutch to the nearest 0.1g with a 60g Pesola scale when hatching was complete. I used 83 nestlings from 24 experimental nests and 76 nestlings from 24 control nests to examine predictions of the adaptive hatching pattern hypotheses. I banded all nestlings in a clutch with a uniquely numbered identification band approximately seven days post-hatch. Because nestlings can fledge prematurely several days before the end of the nestling

period, I stopped weighing nestlings after a majority of the nestlings in a brood failed to gain mass between two successive measurement days, which typically occurred between 10 and 12 days post-hatch. However, I continued to monitor nests daily to identify when all nestlings had fledged. To estimate post-hatching size and condition, I measured the length of both tarsi, the length of both wing chords, head length and head width to the nearest 0.01mm using a Mitutoyo dial caliper at two, six and ten days after the first egg of a clutch hatched. All procedures were reviewed and approved by the Institutional Animal Care and Use Committee of the University of North Carolina at Chapel Hill (Permit Number: 12-046.0)

Statistical analysis

All analyses were done using R 2.14.0 (R Development Core Team 2014). To evaluate a set of candidate models, I chose the model with the lowest Akaike Information Criterion (AIC) value for models that estimated parameters based on maximum likelihood (Burnham and Anderson 2002). I chose the model with the lowest Quasi-Akaike Information Criterion (QIC) value for generalized estimating equations that implemented a quasi-likelihood approach (Pan 2001). For all analyses, I included data only from clutches of four to six eggs because house sparrows in this population rarely lay fewer than four or more than six eggs in a clutch (Aldredge unpub. data). All coefficient and parameter estimates are reported as the mean ± 1 SE.

Effects of experimental manipulation on offspring production

I used linear mixed effects models with female as a random intercept to examine whether treatment influenced clutch size and whether treatment, clutch size or their interaction influenced the number of young that fledged.

Effects of experimental manipulation on embryo and post-hatching survival

I used a generalized linear mixed effects model with a binomial error distribution and clutch nested within female as a random intercept and clutch as a random coefficient for laying order to examine whether laying order, treatment, clutch size or the interactions between treatment and laying order and treatment and clutch size influenced the probability that an embryo survived the incubation period (i.e. the egg hatched). Unfortunately, few eggs failed to hatch in this study, which precluded testing patterns of embryo survival for individual eggs in the laying sequence. However, I subtracted a value of one from each egg in the laying sequence, which caused the first-laid egg in each clutch to occur at the y-intercept. This approach allowed me to test whether the experimental manipulation influenced embryo survival in first-laid eggs (i.e. by testing whether the y-intercept differed between experimental treatments), which is the egg that is predicted to have the lowest rate of embryo survival. I also used a generalized linear mixed effects model with a binomial error distribution and clutch nested within female as a random intercept and clutch as a random coefficient for laying order to examine whether the probability that an embryo survived changed from last- to early-laid eggs (i.e. reverse laying order) or was influenced by treatment, clutch size or the interactions between treatment and reverse laying order and treatment and clutch size.

I used a generalized linear mixed effects model with a binomial error distribution and clutch nested within female as a random intercept and clutch as a random coefficient for laying order to examine whether treatment, clutch size or their interaction influenced the probability that a nestling survived to fledge.

Effects of experimental manipulation on post-hatching growth

I wrote and implemented a self-starting function for the Logistic curve typically used to analyze growth for passerine nestlings (Starck and Ricklefs 1998):

$$w_t = \frac{A}{1 + e^{K(I - t)}}$$

where w_t = mass at time t (g), A = asymptotic mass (g), K = growth rate constant (1/day), I = the inflection point of the growth curve (days), and t = nestling age (days). To remove the possibility that growth curves were influenced by differences in timing of hatching created by the experimental manipulation, I estimated growth curves based on the chronological age (i.e. time since hatching in days) of each nestling. I used a nonlinear (logistic) mixed effects model that controlled for 1) the non-independence of nests from the same female, 2) nestlings within the same nest and 3) repeated measurements of individual nestlings by including nestling identity nested within clutch nested within female as a random intercept. I included random intercepts only for *A* and *I* because the model that included random intercepts for all three growth parameters failed to converge on a solution. I used fixed effects to examine whether treatment influenced each of the three Logistic growth parameters simultaneously (*A*, *K* and *I*), which is possible using a mixed modeling framework (Sofaer et al. 2013). Because variance in nestling mass often increases across the nestling period, I relaxed the normal distribution assumption and allowed the variance to change as a function of 1) a fixed proportionality constant on age, 2) an estimated power of age, 3) a constant added to the estimated power of age, and 4) an exponential function of age. All of the models provided a better fit than using a fixed variance, but the best model contained a constant (1.404) added to the estimated power (0.700) of age.

To determine whether the experimental manipulation resulted in differences in offspring size or condition, I used a principal components analysis to collapse the six morphological measurements (both tarsi, both wings, head length and head width) into fewer linearly uncorrelated variables at each of two, six and ten days after the first egg hatched. Because size characteristics were measured during only three days of the nestling period, I examined treatment differences in size and condition for nestlings that hatched within 24h of the first-hatched egg. This approach removed the possibility that treatment differences in size and condition were influenced by differences in nestling age between experimental and control nests. The first principal component (PC1) explained most (> 79%) of the variation in the six size measures and all size variables had similar weights and were positive for all three measurement days (Table 3.1). Thus, PC1 appeared to reflect overall body size. I used PC1 to examine whether treatment influenced nestling body size at each measurement day. I also extracted residuals from a linear regression between offspring size (PC1) and mass to estimate nestling body condition at two, six, and ten days after the first egg hatched. I used this condition index to determine whether treatment influenced nestling condition at each measurement day.

Effects of experimental manipulation on within-nest variation in offspring mass, size and condition

To determine whether the experimental manipulation influenced the variation in offspring mass within nests, I calculated the standard deviation (hereafter variation) in offspring mass of each nest for each of the first ten days of development, starting one day after the first egg hatched (i.e. when all nests had completed hatching). Because the within-nest variation in offspring mass appeared to increase non-linearly with age, I used a mixed effects quadratic regression with brood nested within female as a random

intercept and brood as a random coefficient for nest age to examine whether age of the nest (in days), treatment, age^2 and the interactions between age and treatment and age^2 and treatment influenced the within-nest variation in offspring mass over the course of post-hatching development. Because the variance associated with the standard deviation estimates increased across the nestling period, I relaxed the normal distribution assumption and allowed the variance to change as a function of 1) a fixed proportionality constant on age, 2) an estimated power of age, 3) a constant added to the estimated power of age, and 4) an exponential function of age. All of the models provided a better fit than using a fixed variance, but the best model contained an estimated power (0.603) of age.

I also determined whether the experimental manipulation influenced the within-nest variation in offspring size and condition. For these analyses, nestlings that hatched more than 24h after the first-hatched egg were included because I was interested in how the experimental manipulation influenced the within-nest variation in size and condition. I used a general linear model to examine whether treatment influenced the within-nest variation in offspring size at each of two, six and ten days after the first egg hatched. I also used a general linear model to examine whether treatment influenced the within-nest variation in offspring condition at each of two, six and ten days after the first egg hatched.

In order to measure the within-nest variation in mass irrespective of developmental changes in the mean mass of the clutch, I also calculated the coefficient of variation in mass of each nest for each of the first ten days of development, starting one day after the first egg hatched. Because the within-nest coefficient of variation in offspring mass appeared to decrease non-linearly with age, I used a mixed effects

quadratic regression with brood nested within female as a random intercept and brood as a random coefficient for nest age to examine whether age of the nest (in days), treatment, age^2 and the interactions between age and treatment and age^2 and treatment influenced the coefficient of variation in mass over the course of post-hatching development. I also used general linear models to examine whether treatment influenced the within-nest coefficient of variation in offspring 1) size and 2) condition at each of two, six and ten days after the first egg hatched.

To examine possible reasons for treatment differences in the within-nest variation and coefficient of variation in mass at hatch completion, I examined whether treatment influenced patterns of hatching asynchrony and also whether treatment and hatching asynchrony influenced a nestling's relative mass at hatch completion. To determine whether the experimental manipulation influenced patterns of hatching asynchrony, I used a linear mixed effects model with clutch nested within female as a random intercept and clutch as a random coefficient for laying order to examine whether laying order, treatment, laying $order^2$, and the interactions between laying order and treatment and laying $order^2$ and treatment influenced the delay in hatching (in hours) from the first-hatched egg (hereafter referred to as hatching delay). I included laying $order^2$ in this analysis because the relationship between laying order and hatching delay appeared to be non-linear. To determine whether the experimental manipulation influenced the relative mass of nestlings within a clutch, I used a generalized estimating equation with a quasi-Poisson error distribution and clutch as a random intercept to determine whether treatment influenced a nestling's relative mass at hatch completion. To determine whether patterns of hatching asynchrony influenced a nestling's relative mass at hatch completion,

and whether the relationship between hatching asynchrony and a nestling's relative mass at hatch completion was influenced by the experimental manipulation, I used a generalized estimating equation with a quasi-Poisson error distribution and clutch as a random intercept to determine whether hatching delay, treatment or their interaction influenced a nestling's relative mass at hatch completion. Finally, I used a general linear model to examine whether the within-nest variation and coefficient of variation in mass at hatch completion was influenced by the relative mass of the last-hatched young, treatment or their interaction.

Effects of relative offspring mass on post-hatching survival and growth

I used a generalized linear mixed effects model with a binomial error distribution and clutch nested within female as a random intercept and clutch as a random coefficient for the mass difference at hatch completion to examine whether a nestling's relative mass at hatch completion, clutch size, treatment or their interactions influenced the probability that a nestling survived to fledge.

I used a nonlinear (logistic) mixed effects model with nestling identity nested within clutch nested within female as a random intercept for A and I to examine whether a nestling's relative mass at hatch completion, treatment or their interaction influenced any of the three growth parameters (A, K and I). Again, I relaxed the normal distribution assumption and allowed the variance to change as a function of 1) a fixed proportionality constant on age, 2) an estimated power of age, 3) a constant added to the estimated power of age, and 4) an exponential function of age. All of the models provided a better fit than using a fixed variance, but the best model contained a constant (1.248) added to the estimated power (0.682) of age. I used a linear mixed effects model with clutch nested

within female as a random intercept and clutch as a random coefficient for the mass difference at hatch completion to examine whether a nestling's relative mass at hatch completion, treatment or their interaction influenced nestling body size (PC1) at each of two, six and ten days after the first egg hatched. Lastly, I used a linear mixed effect model with clutch nested within female as a random intercept and clutch as a random coefficient for the mass difference at hatch completion to examine whether a nestling's relative mass at hatch completion, treatment or their interaction influenced nestling body condition at each of two, six and ten days after the first egg hatched.

Results

Effects of experimental manipulation on offspring production

The experimental manipulation had little effect on offspring production. Specifically, clutch size was similar between experimental (5.42 ± 0.13) and control (5.17 ± 0.13; t = 1.34, P = 0.203) nests. In addition, the number of young that fledged increased with clutch size (coefficient estimate = 0.58 ± 0.24; t = 2.48, P = 0.035) but was similar between experimental (4.59 ± 0.23) and control (4.40 ± 0.23; t = 0.22, P = 0.827) nests.

Effects of experimental manipulation on embryo and post-hatching survival

The experimental manipulation primarily influenced how embryo survival changed across the laying sequence (treatment X laying order interaction: z = 2.09, P = 0.037; Figure 3.1). Overall, embryo survival was similar between experimental (92% of eggs) and control (96% of eggs; coefficient estimate = 0.81 ± 0.63; z = 1.29, P = 0.196) nests. Despite this similarity between treatments, the probability that an embryo survived (i.e., egg hatched) increased with laying order in control nests (coefficient estimate = 0.96 ± 0.50; z = 1.92, P = 0.054), but changed little with laying order in experimental nests

(coefficient estimate = -0.16 ± 0.20; z = -0.81, P = 0.419). Embryo survival was similar in first-laid eggs from experimental and control nests (z = 1.22, P = 0.223), and did not appear to be influenced by clutch size in either control (coefficient estimate = 0.46 ± 0.75; z = 0.60, P = 0.545) or experimental (coefficient estimate = -0.51 ± 0.58; z = -0.89, P = 0.376; treatment X clutch size interaction: coefficient estimate = 0.97 ± 0.95; z = 1.02, P = 0.308) nests. Consistent with the results for laying order, embryo survival decreased from last- to early-laid eggs in control nests (coefficient estimate = -0.96 ± 0.50; z = -1.92, P = 0.054) but changed little from last- to early-laid eggs in experimental nests (coefficient estimate = 0.16 ± 0.20; z = 0.81, P = 0.419; reverse laying order X treatment: z = 2.09, P = 0.037; Figure 3.1). In addition, clutch size influenced embryo survival differently between experimental and control nests (reverse laying order X treatment: z = 1.95, P = 0.052). Specifically, embryo survival increased non-significantly with clutch size in control nests (coefficient estimate = 1.42 ± 0.91; z = 1.56, P = 0.118) but decreased non-significantly with clutch size in experimental nests (coefficient estimate = -0.67 ± 0.57; z = -1.17, P = 0.242).

Overall, post-hatching survival was similar between experimental (95% of nestlings) and control (90% of nestlings; coefficient estimate = 0.78 ± 0.61; z = 1.28, P = 0.202) nests, and did not appear to be influenced by clutch size (coefficient estimate = -0.33 ± 0.54; z = -0.61, P = 0.541).

Effects of experimental manipulation on post-hatching growth

The experimental manipulation influenced growth rate (K) but appeared to have little effect on overall changes in post-hatching mass, size, and condition. Control nestlings displayed a faster growth rate (coefficient estimate: 0.019 ± 0.006; t = 3.00, P =

0.003) than experimental nestlings (Figure 3.2). However, both control and experimental nestlings reached a similar inflection point (coefficient estimate: 0.22 ± 0.15d; t = 1.43, P = 0.152) and a similar asymptotic mass (coefficient estimate: 0.22 ± 1.41g; t = 0.153, P = 0.879; Figure 3.2). Consistent with patterns of mass increase, control and experimental nestlings reached a similar size at two (coefficient estimate: 0.42 ± 0.50; t = 0.86, P = 0.415), six (coefficient estimate: 0.36 ± 0.76; t = 0.47, P = 0.643), and ten (coefficient estimate: 0.16 ± 0.68; t = 0.24, P = 0.817) days after the first egg hatched (Figure 3.3). Control and experimental nestlings also exhibited a similar body condition at two (coefficient estimate: 0.19 ± 0.20; t = 0.96, P = 0.362), six (coefficient estimate: 0.19 ± 0.52; t = 0.36, P = 0.722), and ten (coefficient estimate: 0.05 ± 0.33; t = 0.14, P = 0.891) days after the first egg hatched (Figure 3.4).

Effects of experimental manipulation on within-nest variation in offspring mass, size and condition

The experimental manipulation affected the (initial) within-nest variation in mass at hatch completion, but it did not affect the rate at which the variation in mass increased over the course of post-hatching development (Figure 3.5). Specifically, the initial variation in mass was higher in control (0.63 ± 0.06g) than in experimental (0.48 ± 0.06g; t = 2.35, P = 0.035) nests. The within-nest variation in mass increased non-linearly with age (age: coefficient estimate: 0.41 ± 0.05; t = 8.01, P < 0.001; age^2: coefficient estimate: -0.028 ± 0.006; t = -4.79, P < 0.001), a relationship that was similar between experimental and control nests (treatment X age: t = 1.36, P = 0.174; treatment X age^2: t = 1.26, P = 0.208).

The experimental manipulation also affected the within-nest variation in offspring size, but had little effect on variation in offspring condition. Specifically, the variation in offspring size was higher in control than in experimental nests at two (coefficient estimate: 0.99 ± 0.24; $t = 4.16$, $P < 0.001$) and ten (coefficient estimate: 0.64 ± 0.27; $t = 2.38$, $P = 0.026$) days after the first egg hatched, but was similar between experimental and control nests at six days after the first egg hatched (coefficient estimate: 0.52 ± 0.46; $t = 1.14$, $P = 0.270$). The variation in offspring condition was similar between control and experimental nests at each of two (coefficient estimate: 0.14 ± 0.14; $t = 1.03$, $P = 0.310$), six (coefficient estimate: 0.21 ± 0.24; $t = 0.88$, $P = 0.390$) and ten (coefficient estimate: 0.03 ± 0.17; $t = 0.17$, $P = 0.860$) days after the first egg hatched.

In contrast to the results for the within-nest variation in mass, the experimental manipulation affected both the initial coefficient of variation in mass and the rate at which the coefficient of variation in mass decreased over the course of post-hatching development (treatment X age: $t = -0.96$, $P = 0.340$; treatment X age^2: $t = 2.00$, $P = 0.046$; Figure 3.6). Specifically, the initial coefficient of variation in mass was higher in control (0.201 ± 0.017) than in experimental (0.124 ± 0.014; $t = 4.05$, $P = 0.001$) nests. In addition, the coefficient of variation in mass decreased non-linearly with age in control nests (age: coefficient estimate: 0.007 ± 0.005; $t = 1.44$, $P = 0.151$; age^2: coefficient estimate: -0.0020 ± 0.0005; $t = -4.01$, $P < 0.001$), but displayed little change with age in experimental nests (age: coefficient estimate: 0.0007 ± 0.004; $t = 0.15$, $P = 0.879$; age^2: coefficient estimate: -0.0007 ± 0.0005; $t = -1.38$, $P = 0.168$). The experimental manipulation had little effect on the within-nest coefficient of variation in offspring size at each of two (coefficient estimate: 0.75 ± 0.96; $t = 0.78$, $P = 0.440$), six (coefficient

estimate: 0.76 ± 1.23; t = 0.62, P = 0.540) and ten (coefficient estimate: 3.96 ± 3.59; t = 1.10, P = 0.281) days after the first egg hatched. The experimental manipulation also had little effect on the within-nest coefficient of variation in offspring condition at each of two (coefficient estimate: 0.33 ± 2.57; t = 0.13, P = 0.900), six (coefficient estimate: 2.69 ± 2.57; t = 1.05, P = 0.310) and ten (coefficient estimate: 6.11 ± 7.05; t = 0.87, P = 0.390) days after the first egg hatched.

The greater variation and coefficient of variation in mass at hatch completion in control nests appeared to be a result of differences in hatching patterns created by the experimental manipulation (laying order X treatment interaction: t = 0.76, P = 0.448; laying order^2 X treatment interaction: t = 3.49, P < 0.001). In control nests, first-laid eggs hatched first (0.20 ± 0.20h; t = 0.16, P = 0.872) and late-laid eggs hatched later than early-laid eggs (coefficient estimates: egg = -1.24 ± 1.71; t = -0.73, P = 0.468 and egg^2 = 1.63 ± 0.36; t = 4.54, P < 0.001; Figure 3.7). Alternatively, in experimental nests, first-laid eggs were not the first to hatch (4.65 ± 2.07h; t = 3.20, P = 0.002) and little difference existed in the hatching delay of early- and late-laid eggs (coefficient estimates: egg = 0.51 ± 1.54; t = 0.33, P = 0.742 and egg^2 = -0.04 ± 0.31; t = -0.12, P = 0.906; Figure 3.7).

Nestlings that experienced a delay in hatching were light relative to other nestlings in the brood (Wald = 34.25, P < 0.001), and the relationship between hatching delay and a nestling's relative mass at hatch completion was similar between control (coefficient estimate: 0.038 ± 0.006) and experimental (coefficient estimate: 0.055 ± 0.008; hatching delay X treatment interaction; Wald = 2.84, P = 0.092; Figure 3.8) nests. As expected, control nestlings were relatively light (0.84 ± 0.08) at hatch completion

compared to experimental nestlings (0.50 ± 0.04; Wald = 20.19, P < 0.001), likely because control nests exhibited greater hatching delays in late-laid eggs (i.e. increased hatching asynchrony) and hence contained more light nestlings than experimental nests at hatch completion (Figure 3.9). Consistent with this hypothesis, the (initial) within-nest variation in mass at hatch completion was higher when last-hatched young were relatively light at hatch completion (coefficient estimate: 0.152 ± 0.049; t = 3.11, P = 0.003), a relationship that was similar between experimental and control nests (mass difference X treatment interaction: coefficient estimate: 0.216 ± 0.141; t = 1.54, P = 0.133; Figure 3.10). In addition, the initial within-nest coefficient of variation in mass was higher when last-hatched young were relatively light at hatch completion (coefficient estimate: 0.097 ± 0.009; t = 10.55, P < 0.001), a relationship that was similar between experimental and control nests (mass difference X treatment interaction: coefficient estimate: 0.03 ± 0.02; t = 1.07, P = 0.290; Figure 3.10).

Effects of relative offspring mass on post-hatching survival and growth

A nestling's relative mass at hatch completion was associated with reduced post-hatching survival and growth, a relationship that appeared to be relatively independent of the experimental manipulation. Post-hatching survival declined in nestlings that were relatively light at hatch completion (coefficient estimate: -1.00 ± 0.47; z = -2.12, P = 0.034; Figure 3.9), and the relationship between post-hatching survival and relative mass at hatch completion was similar between experimental and control nestlings (coefficient estimate: 1.60 ± 0.90; z = 1.77, P = 0.076). After including the effect of a nestling's relative mass at hatch completion, control nestlings had both a faster growth rate (0.49 ± 0.006) and earlier inflection point (4.29 ± 0.12d) than experimental nestlings (K: 0.47 ±

0.005; t = 3.85, P < 0.001; I: 4.58 ± 0.10d; t = -2.46, P = 0.014), but nestlings continued to reach a similar asymptotic mass between control (27.0 ± 0.98g) and experimental (26.7 ± 0.98g; t = 0.21, P = 0.835; Figure 3.11) nests. In addition, a nestling's relative mass at hatch completion was associated with changes in all aspects of post-hatching growth. Relatively light nestlings had a slower growth rate (coefficient estimate: -0.019 ± 0.005; z = -3.52, P < 0.001), later inflection point (coefficient estimate: 0.19 ± 0.06d; z = 3.10, P = 0.002) and lighter asymptotic mass (coefficient estimate: -1.95 ± 0.33; z = -5.86, P < 0.001) than the heaviest nestling(s) in the brood at hatch completion (Figure 3.11). Nestlings that were relatively light at hatch completion also were smaller than the heaviest nestling(s) in the brood at each of two (coefficient estimate = -1.59 ± 0.23; z = -7.07, P < 0.001), six (coefficient estimate = -1.92 ± 0.30; z = -6.47, P < 0.001) and ten (coefficient estimate = -1.59 ± 0.23; z = -7.07, P < 0.001) days after the first egg hatched (Figure 3.12). The relationship between a nestling's size and its relative mass at hatch completion was similar between experimental and control nestlings at all three measurement days (Day 2: coefficient estimate = 0.74 ± 0.46; z = 1.60, P = 0.143; Day 6: coefficient estimate = 0.51 ± 0.83; z = 0.62, P = 0.543; Day 10: coefficient estimate = 0.32 ± 0.67; z = 0.48, P = 0.651). Nestling condition did not appear to be influenced by the relative mass at hatch completion for any of the three measurement days (Day 2: coefficient estimate = 0.0007 ± 0.0946; t = 0.01, P = 0.994; Day 6: coefficient estimate = 0.07 ± 0.27; z = 0.25, P = 0.803; Day 10: coefficient estimate = 0.08 ± 0.26; z = 0.30, P = 0.763).

Discussion

Timing of incubation had little effect on the number of offspring that fledged. Instead, timing of incubation mainly affected which eggs failed to hatch and the initial variation in offspring size associated with asynchronous hatching. Females maximized embryo survival in all but the earliest-laid eggs by beginning incubation prior to clutch completion (Figure 3.1). However, early incubation caused last-hatched young to be light at hatch completion (Figure 3.8), which increased the variation in offspring size when all eggs had completed hatching (Figure 3.10). Timing of incubation had little effect on growth and survival of the heaviest, early-hatched nestlings in the brood, but late-hatched nestlings that were light at hatch completion grew slowly (Figures 3.11 and 3.12) and exhibited a reduced probability of survival (Figure 3.9). Overall, most nestlings maintained a high probability of survival in both asynchronous and experimentally synchronized nests. Thus, these results suggest that early incubation does not maximize embryo survival or maximize post-hatching survival and growth, but may be a tradeoff between maintaining both embryo survival and growth and survival of late-hatched young (Stoleson and Beissinger 1997, Arnold 2011).

Early-laid eggs from control nests experienced lower hatching success than late-laid eggs, supporting evidence that prolonged exposure to the environment decreases embryo survival (Arnold et al. 1987, Veiga 1992). Hatching success was similar for first-laid eggs in naturally asynchronous and experimentally synchronized clutches. However, hatching success changed little across the laying sequence when incubation was delayed experimentally. Taken together, these results suggest that embryo survival may not decrease as a linear function of pre-incubation exposure but may decrease gradually after

some threshold of exposure has been reached (Wang et al. 2011). However, this explanation fails to explain why survival remained low in embryos of last-laid eggs from experimentally synchronized clutches, which had little to no exposure prior to incubation. Treatment differences were not caused by the handling and transportation manipulation because both treatments experienced the same procedure. In addition, the experimental nest environment did not cause abnormal patterns of embryo mortality (Veiga 1992) because few [one (n = 9 nests) or two (n = 1 nest)] eggs failed to hatch in experimentally synchronized nests, consistent with patterns found in asynchronous nests. Low sample size may have precluded strong conclusions about the effect of pre-incubation exposure because most eggs hatched in this study. Despite the small sample size and potentially low power to detect an effect, house sparrow females displayed an incubation strategy that maintained embryo survival in all but the earliest-laid eggs (Aldredge et al. 2012). Thus, house sparrow females appear to begin incubation too late to maximize survival in all embryos.

Although early incubation maintains survival of most embryos, this behavior also increases the variation in offspring size caused by asynchronous hatching (Veiga and Viñuela 1993, Stoleson and Beissinger 1999, Sockman 2008, Aldredge et al. 2014). Embryos from late-laid eggs could reduce the delay in hatching by decreasing the length of embryonic development (Ricklefs 1993). However, this strategy is likely to occur only if embryos from late-laid eggs hatch at a similar time and size as other nestlings in the brood. I found no evidence of faster embryonic development in late-laid eggs. Rate of development may not change with laying order because it is maximized in house sparrow embryos (Lepczyk and Karasov 2000). Although strong selection should exist for

embryos from late-laid eggs to reduce the variation in size caused by early incubation, selection also should exist for embryos from early-laid eggs to maintain the competitive size advantage caused by asynchronous development and increase the chance of survival during food shortages (Lloyd and Martin 2003). This should result in maximal growth rates for all embryos (Ricklefs 1969). Thus, maximal growth rates in altricial embryos may represent a form of parent-offspring conflict whereby offspring that begin development relatively early gain the direct fitness benefits of increased survival caused by asynchronous hatching (Ricklefs 1993, 2002). However, the enhanced growth and survival of early-hatched offspring come at a cost to parents of decreasing the probability that small, late-hatched offspring survive to fledge. Possibly as a response to this conflict, female house sparrows in this (Aldredge unpub data) and other populations (Anderson 2006) increase the size of late-laid eggs, which can reduce the developmental size hierarchy caused by early incubation and hence increase the probability of survival for small, late-hatched young (Forbes and Wiebe 2010).

Nestlings that were relatively small at hatching experienced slower growth and lower post-hatching survival than large, early-hatched nestlings in both naturally asynchronous and experimentally synchronized nests. Hatching often occurred over 5-10h in experimentally synchronized nests (compared to 24-36h in control nests), which resulted in a small size difference (median mass difference = ~0.5g) between the lightest and heaviest nestling in the brood. Despite the similar size of experimental nestlings at hatch completion, post-hatching growth and survival declined as a consequence of the size difference caused by asynchronous hatching, a pattern that has been shown in other studies (Howe 1976, Clark and Wilson 1981, Slagsvold 1982, Bancroft 1985). These

results suggest that early incubation is not necessary for parents to regulate growth and survival of hatched offspring (Bengtsson and Ryden 1983, Amundsen and Slagsvold 1991), which begs the question: Why is hatching asynchrony large in house sparrow clutches? A clear benefit of initiating incubation before all eggs are laid (in addition to reducing the risk of embryo mortality) is that it ensures that late-laid eggs hatch last. This enables females to predict hatching order (Figure 3.7) and allocate resources preferentially to late embryos, which may increase the probability that late-hatched offspring survive (Sockman et al. 2006, Forbes and Wiebe 2010, Hadfield et al. 2013).

In spite of the potential fitness benefits of synchronous hatching for maintaining post-hatching survival and growth, females began incubation before clutch completion and increased the (initial) variation in offspring mass at hatching. The within-nest variation in offspring mass increased more than three-fold over the course of post-hatching development, likely as a consequence of the exponential increase in offspring mass. Unexpectedly, this variation increased similarly between asynchronous and experimentally synchronized nests, suggesting that the within-nest variation in mass may increase independently of hatching patterns created by the female.

Treatment differences in the within-nest variation in mass became apparent primarily after controlling for the increase in offspring mass across development. Although the variation in mass at hatch completion was only 33% higher in asynchronous than in experimentally synchronized nests, the coefficient of variation in mass, which is the variation in offspring mass divided by the mean mass of the brood, was almost twice as large in asynchronous nests immediately after hatching. Thus, the small size of nestlings at hatch completion can cause even small amounts of variation to seem large

relative to the size of offspring at the beginning of post-hatching development (Amundsen and Slagsvold 1991). As nestlings aged, the mean mass of nestlings increased more rapidly than the variation in mass within nests, which caused the coefficient of variation in mass to decline over the course of post-hatching development. This decline occurred more rapidly in asynchronously hatching nests, likely owing to the higher coefficient of variation in mass at the beginning of post-hatching development. As a consequence of this faster decline in asynchronous nests, the coefficient of variation in mass was similar between asynchronous and experimentally synchronized nests by the end of post-hatching development (Hebert 1993). Thus, the variation in mass caused by early incubation may have its greatest effect on offspring growth and survival immediately after hatching, an effect that diminishes as offspring age and increase mass exponentially. In addition, these results suggest that studies that examine the effects of within-nest variation in offspring size should manipulate this variation early in development or should take into account the smaller relative effect that the variation in size may have as nestlings age.

Consistent with the hypothesis that the initial variation in offspring size may have a relatively large effect on post-hatching development, I found that a nestling's relative mass at hatch completion had a more robust effect on post-hatching growth and survival than timing of incubation. In fact, post-hatching growth and survival decreased in nestlings that were relatively light at hatch completion but was not directly influenced by when females began incubation. Because late-hatched nestlings were lighter at hatch completion in naturally asynchronous than in experimentally synchronized nests, late-hatched nestlings from asynchronous nests likely grew slower and reached a smaller adult

size than late-hatched nestlings from experimentally synchronized nests (Greig-Smith 1985). Importantly, slow growth and small adult size are associated with low post-fledging survival, as well as poor survival prior to breeding (Magrath 1991). Timing of incubation had little effect on growth and survival of the largest, early-hatched nestlings in asynchronous and experimentally synchronized nests, and also appeared to have little effect on overall (e.g. mean) patterns of growth and survival of asynchronous and experimentally synchronized nestlings. Thus, I was able to uncover a pattern hidden by conventional analyses by investigating not only the mean effects of the experimental treatment, but also how variation within those treatments influenced post-hatching growth and survival. Overall, it appears that early incubation may reduce the quality but not the quantity of offspring that fledge, a finding that contradicts predictions of the adaptive hatching pattern hypotheses.

The similarity in the within-nest coefficient of variation in mass between asynchronous and experimentally synchronized nests at the end of post-hatching development supports the idea that house sparrows adopt a strategy of facultative brood reduction (Anderson 2006). In fact, much of the evidence from this study suggests that females adopt an incubation strategy that maintains post-hatching growth and survival in most, if not all, offspring. Although survival declined with increasing size differences associated with asynchronous hatching (and early incubation), post-hatching survival remained high (> 80%) even for the smallest, last-hatched young from naturally asynchronous nests. Thus, timing of incubation may be constrained by the ability of small, late-hatched young to overcome the initial size difference caused by asynchronous hatching and fledge. Further evidence that small, late-hatched young can overcome the

initial size difference caused by asynchronous hatching comes from the decline in the within-nest variation in mass near the end of the nestling period. These results suggest that small, late-hatched young increase mass faster than their larger, older nestmates at the end of post-hatching development. Thus, small, late-hatched young in both asynchronous and experimentally synchronized nests appear to be able to catch-up in mass, and likely in size, to their older siblings prior to fledging (Aldredge in press). Altogether, these results suggest that female house sparrows begin incubation too early to maximize growth and survival of all offspring, but late enough to maintain growth and survival of most young.

Thus, my results predict that timing of incubation may be influenced by two factors: 1) the rate at which embryo survival decreases with pre-incubation exposure and 2) the rate at which post-hatching growth and survival decrease with mass differences caused by hatching asynchrony. Precocial birds appear to take full advantage of the fitness benefits of early incubation. Not only does early incubation increase embryo survival (Arnold et al. 1987), but precocial embryos also communicate with each other to synchronize development, which increases the number of offspring that hatch and fledge. Thus, incubation onset in precocial birds likely is constrained by the ability of embryos to overcome developmental differences caused by early incubation and hatch synchronously. Early incubation also increases embryo survival in altricial birds, but altricial embryos have evolved to maximize growth, which maintains the variation in offspring size caused by asynchronous development. Thus, incubation onset in altricial birds may be constrained by the ability of late-hatched nestlings to overcome the initial size difference caused by asynchronous hatching and fledge. Overall, incubation onset in

both precocial and altricial species appears to be an adaptive strategy used by female birds to maximize the number of embryos that survive the incubation and nestling periods to fledge (i.e. parental fitness).

Hatching asynchrony is predicted to have its greatest effect on post-hatching growth and survival in years or environments when food availability is low (Magrath 1989, Forbes 1994). It is possible that food was relatively abundant for raising nestlings in 2013 and 2014, which might mask any adaptive benefits of early incubation on post-hatching growth and survival. Offspring increased mass faster, on average, during this study than during the previous two breeding seasons (2011-2012; Aldredge unpub. data), suggesting that food availability may have been relatively high in 2013 and 2014. Despite these annual differences in post-hatching growth, survival was similar across all four years, providing further support that early incubation may have a greater effect on quality than on the quantity of post-hatching young. Annual differences in food availability also are likely to influence within-nest patterns of variation and coefficient of variation in mass. However, it is unclear how food availability may influence the variation in offspring mass across post-hatching development. Thus, studies should be conducted in environments or years with a variable food supply or studies should be done in conjunction with experimental manipulations of food availability (Magrath 1989). Such studies can test how early incubation influences developmental changes in the variation in offspring mass, and also can test whether parents can regulate post-hatching development independently of hatching patterns created by the female (as seen in this study).

This study highlights the importance of considering both pre- and post-hatching survival and growth when testing adaptive explanations that are caused by timing of embryonic development. Embryo survival decreases with pre-incubation exposure, which should select for early incubation. However, early incubation increases the size differences caused by asynchronous hatching, which can reduce post-hatching growth and survival. In response to these different sources of mortality, female house sparrows appear to begin incubation early enough to maximize embryo survival in all but the earliest-laid eggs but late enough to maintain growth and survival of late-hatched offspring. Thus, incubation onset likely is a tradeoff between maintaining both embryo survival and post-hatching survival and growth of late-hatched young.

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Table 3.1: The first principal component (PC1) of six morphological measurements characterized overall body size at 2, 6, and 10 days after the first egg hatched. Numbers indicate the factor loadings of each of six morphological measurements: Lt tarsus = left tarsus (leg), Rt tarsus = right tarsus (leg), Lt wing chord = left wing, Rt wing chord = right wing. Var explained = Variance explained by PC 1.

	Day 2	**Day 6**	**Day 10**
Lt tarsus	0.403	-0.421	-0.404
Rt tarsus	0.404	-0.423	-0.406
Lt wing	0.420	-0.422	-0.435
Rt wing	0.416	-0.421	-0.429
Head width	0.394	-0.350	-0.355
Head length	0.411	-0.408	-0.414
Var explained	0.827	0.878	0.795

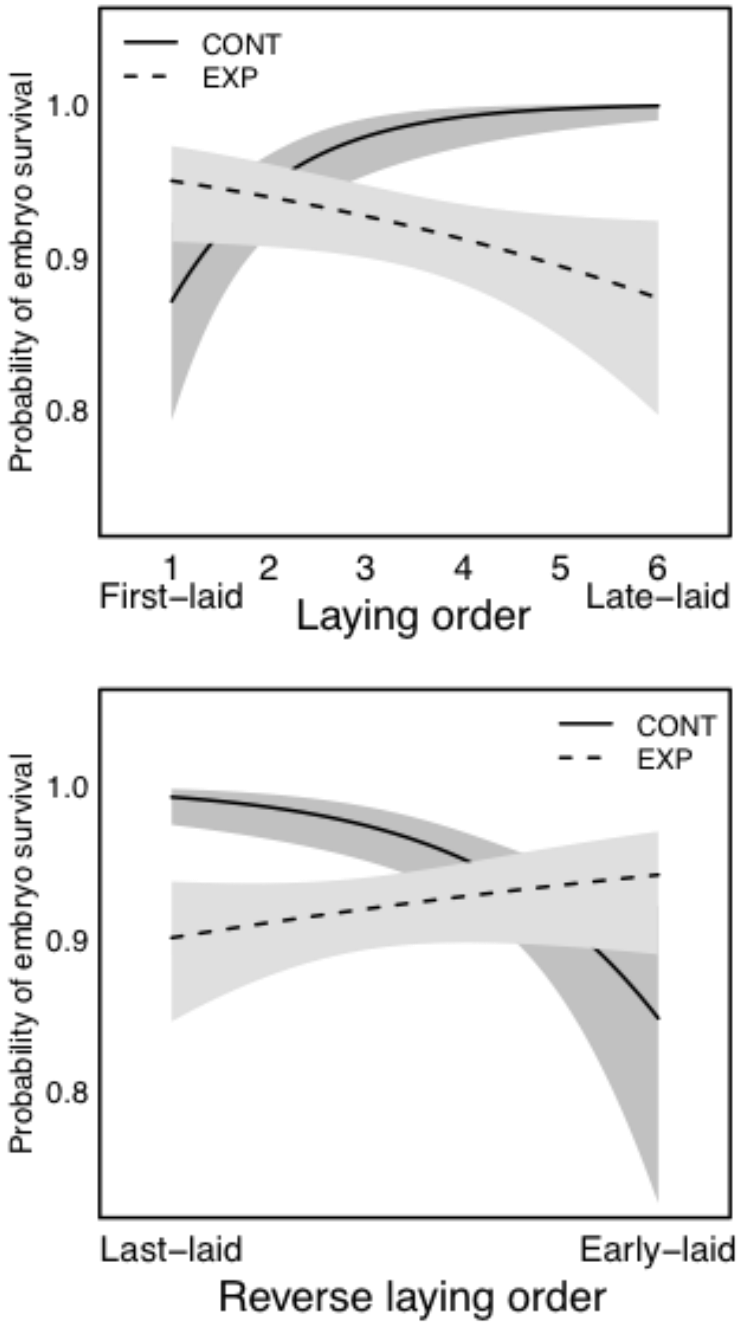

Figure 3.1: Relationship between laying order (top panel) and reverse laying order (bottom panel) and embryo survival in control (CONT; solid line, dark gray polygon) and experimental (EXP; dashed line, light gray polygon) nests. Embryo survival (hatching success) was lower in early- than in late-laid eggs in control nests. Embryo survival was similar in first-laid eggs from experimental and control nests and changed little across the laying sequence in experimental nests. Lines are approximated using parameter estimates of the best model, and colored polygons indicate one standard error above and below the mean probability of embryo survival (i.e., line).

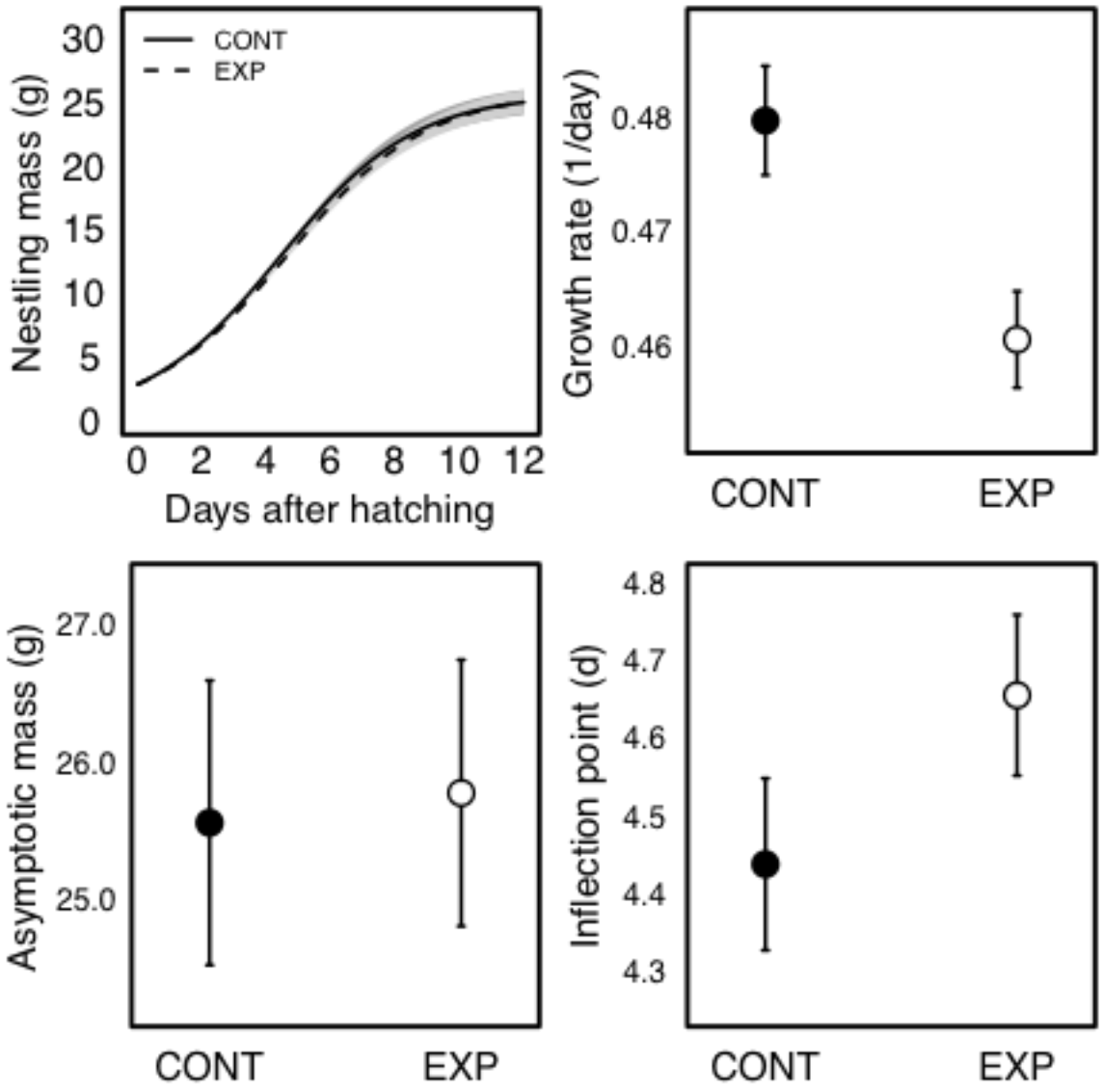

Figure 3.2: Differences in offspring growth between control (CONT; filled circles, solid line, dark gray polygon) and experimental nests (EXP; open circles, dashed line, light gray polygon). Nestlings from control nests reached a faster growth rate than nestlings from experimental nests. However, the inflection point and asymptotic mass were similar between control and experimental nests, which resulted in little difference in patterns of offspring growth. All results are approximated using parameter estimates from the nonlinear mixed effects model that allowed all three growth parameters to vary simultaneously between treatments. Colored polygons indicate one standard error above and below the mean offspring mass (i.e., line).

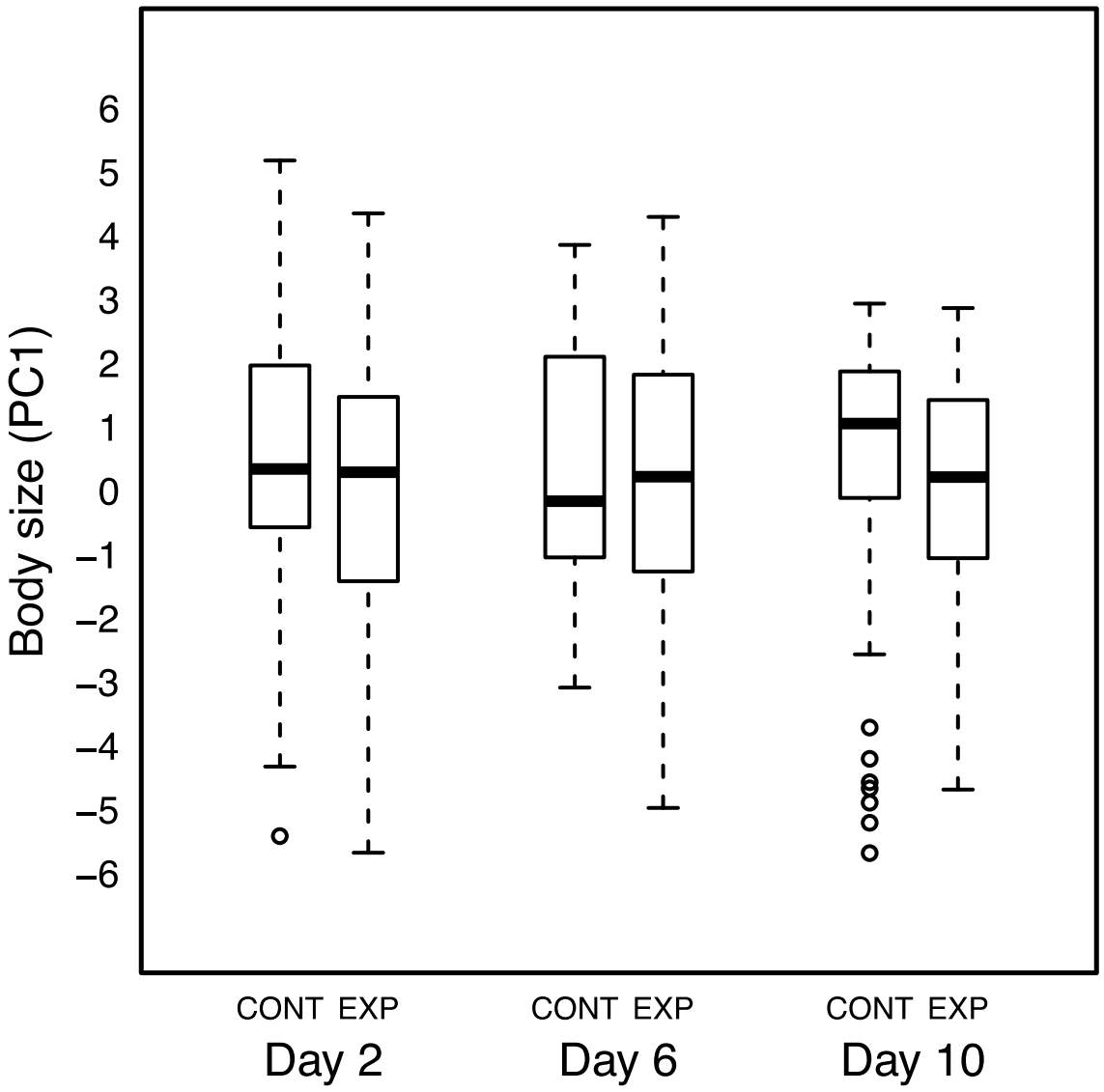

Figure 3.3: Differences in body size (PC1) between control and experimental nestlings at each of two, six, and ten days after the first egg hatched. Nestling body size was similar between control and experimental nests at each measurement day. Boxplots are the median (thick line), first and third quartiles (box) and 95% confidence intervals around the median (dashed line), as well as any outliers (points) of body size estimates.

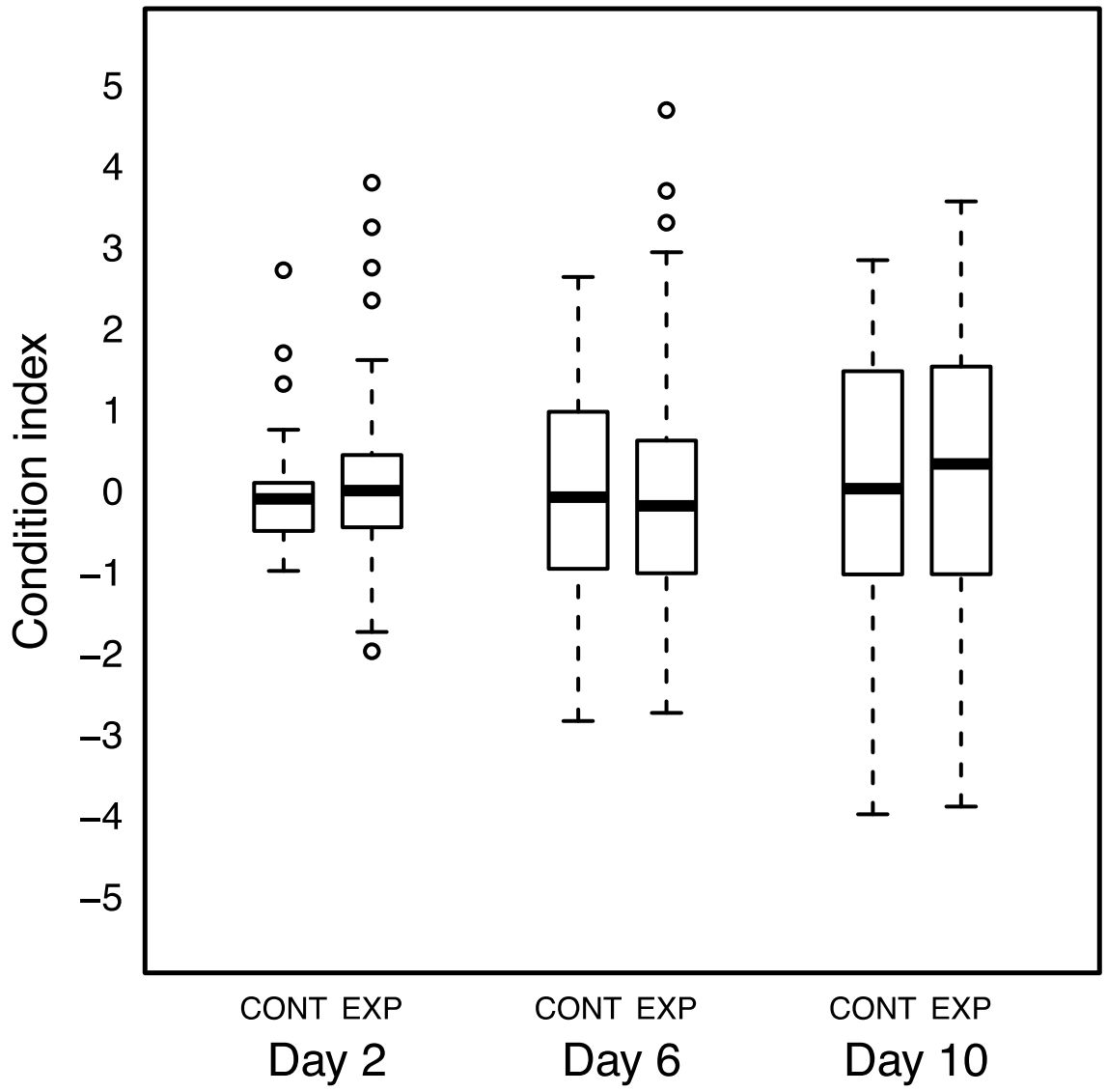

Figure 3.4: Differences in body condition (residuals from linear regression of body size (PC1) and mass) between control and experimental nestlings at each of two, six, and ten days after the first egg hatched. Nestling condition was similar between control and experimental nests at each measurement day. Boxplots are the median (thick line), first and third quartiles (box) and 95% confidence intervals around the median (dashed line), as well as any outliers (points) of body condition estimates.

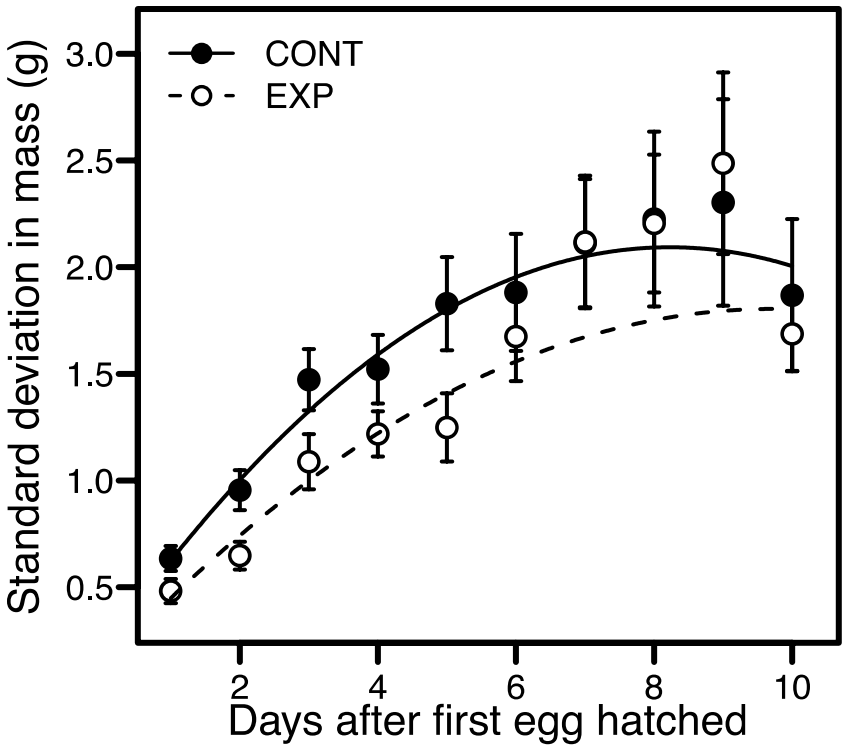

Figure 3.5: Change in within-nest standard deviation (i.e. variation) in mass over the course of post-hatching development for control (CONT; closed points, solid line) and experimental (EXP; open points, dashed line) nests. Standard deviation in mass was higher in control than in experimental nests at hatch completion but increased similarly across the nestling period in both treatments. Points and standard errors are derived from actual data and lines are approximated using parameter estimates of the best model.

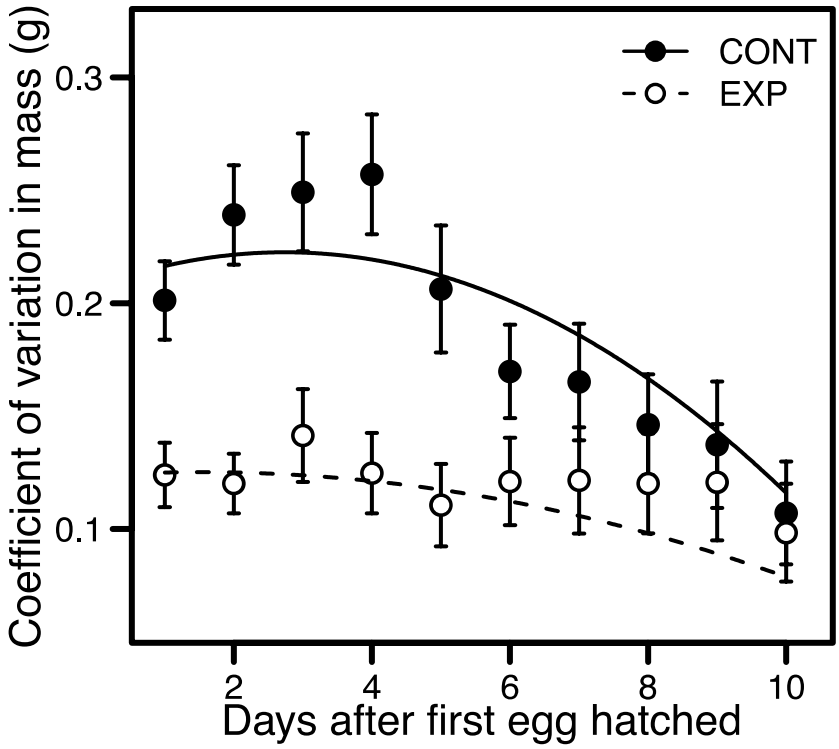

Figure 3.6: Change in within-nest coefficient of variation (i.e. size-specific variation) in mass over the course of post-hatching development for control (CONT; closed points, solid line) and experimental (EXP; open points, dashed line) nests. Coefficient of variation in mass was higher in control than in experimental nests at hatch completion. In addition, the coefficient of variation in mass decreased more rapidly in control than in experimental nests across the nestling period. Points and standard errors are derived from actual data and lines are approximated using parameter estimates of the best model.

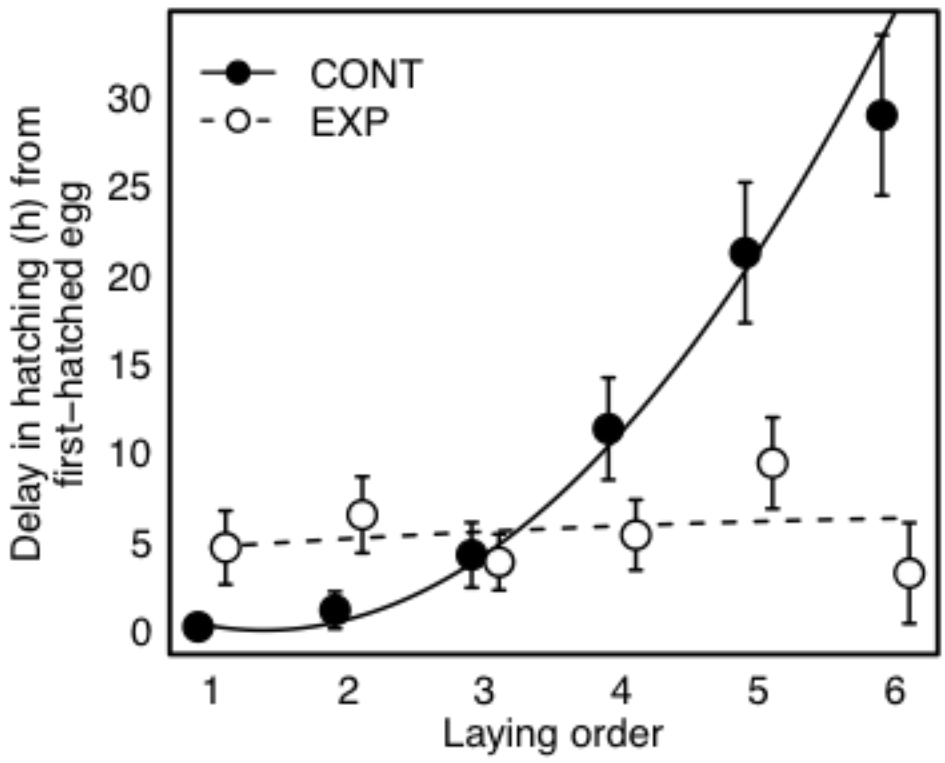

Figure 3.7: Relationship between laying order and hatching delay in control (CONT; closed circles, solid line) and experimental (EXP; open circles, dashed line) nests. Late-laid eggs hatched later than early-laid eggs in control nests but laying order had little effect on hatching delay in experimental nests. Points and standard errors are derived from actual data and lines are approximated using parameter estimates of the best model.

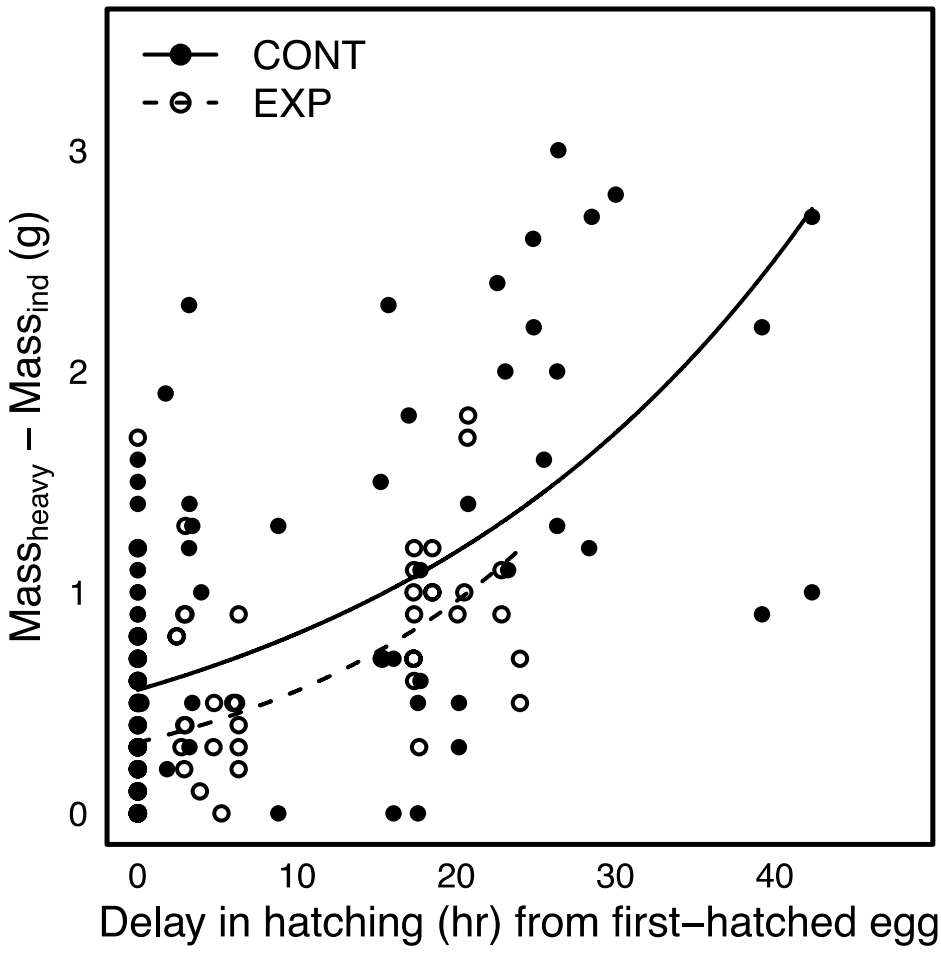

Figure 3.8: Relationship between hatching delay (Delay in hatching (hr) from first-hatched egg) and a nestling's relative mass at hatch completion [$Mass_{heavy}$ - $Mass_{ind}$ (g)]. A nestling's relative mass at hatch completion increased similarly with hatch delay in control (CONT) and experimental nests (EXP). Points are drawn from actual data and lines are estimated from the best fit generalized estimating equation.

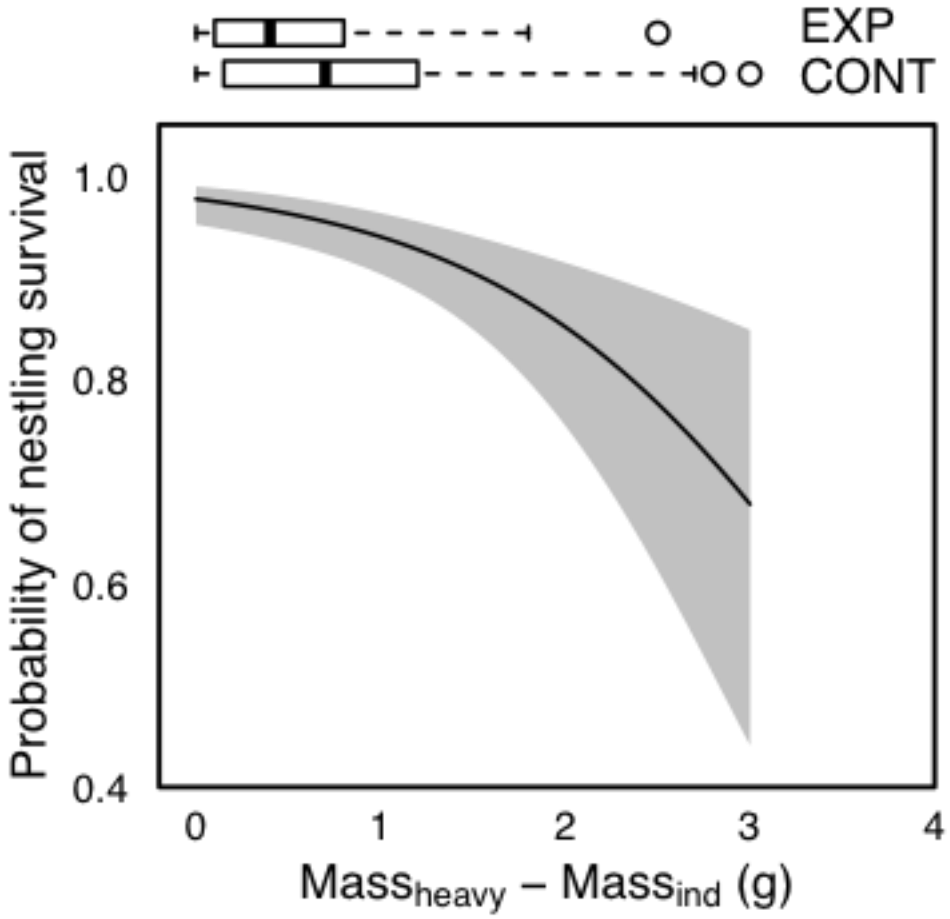

Figure 3.9: Relationship between post-hatching (nestling) survival and a nestling's relative mass at hatch completion [$Mass_{heavy}$ - $Mass_{ind}$ (g)]. The relationship was similar between control and experimental nests. Boxplots are the median (thick line), first and third quartiles (box) and 95% confidence intervals around the median (dashed line), as well as any outliers (points) of relative nestling sizes for each treatment. The line is approximated using parameter estimates of the best model, and the colored polygon indicates one standard error above and below the mean probability of nestling survival (i.e., line).

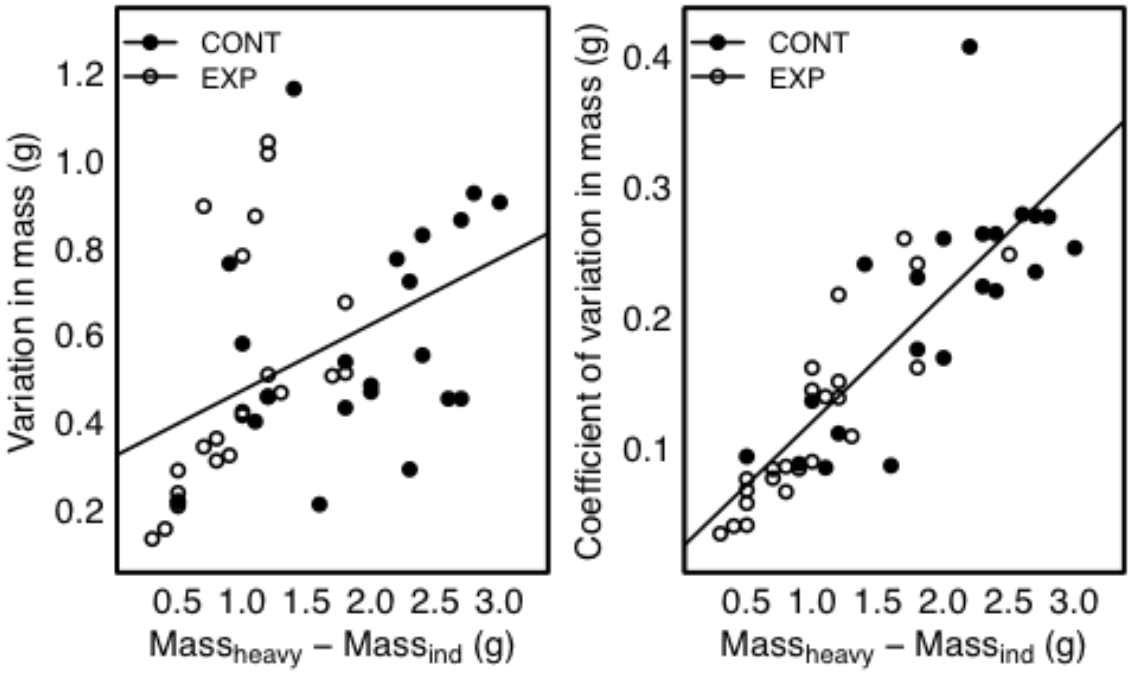

Figure 3.10: The (initial) variation (left panel) and coefficient of variation (right panel) in mass at hatching completion was influenced by the relative mass of the last-hatched nestling at hatch completion [$Mass_{heavy}$ - $Mass_{ind}$ (g)]. The initial within-nest variation and coefficient of variation in mass was higher when last-hatched young were relatively light at hatch completion, a relationship that was similar between experimental and control nests.

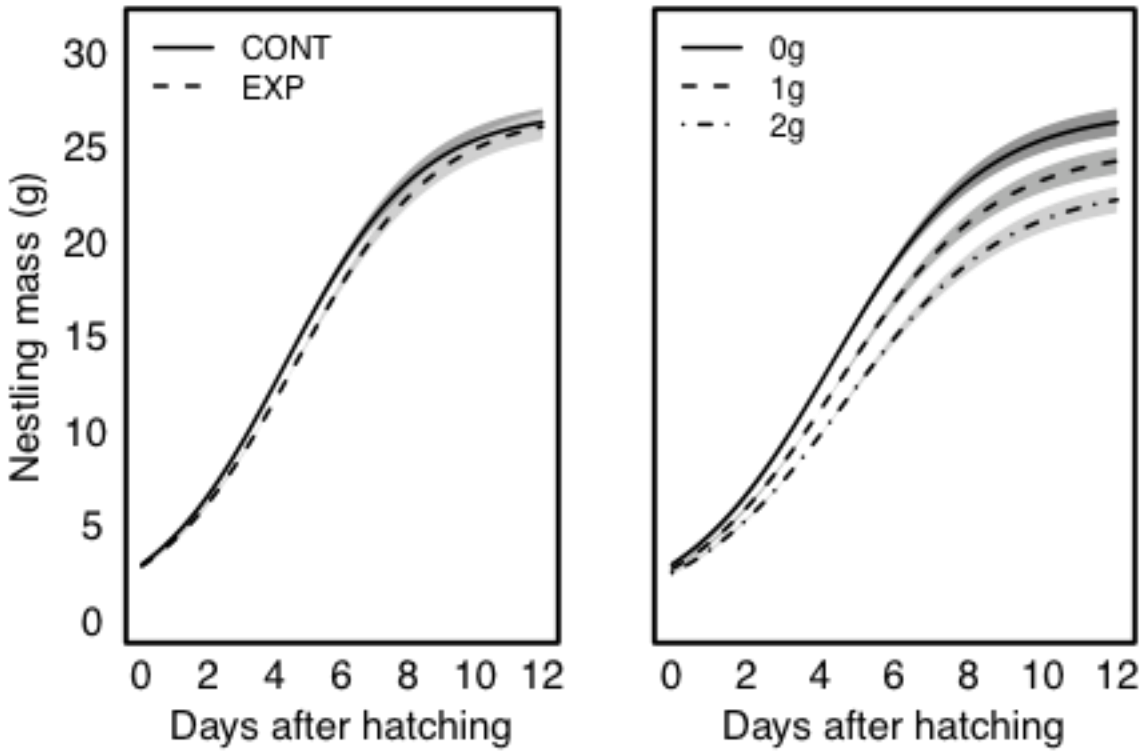

Figure 3.11: Nestling growth was influenced by treatment (left panel) and a nestling's relative mass at hatch completion (right panel). The left panel shows the average growth of the heaviest nestling at hatch completion in each treatment and the right panel shows how the relative mass at hatch completion influenced offspring growth in control nests. Overall, nestlings from control nests (CONT; solid line, dark gray polygon) reached a faster growth rate and earlier inflection point than nestlings from experimental nests (EXP; dashed line, light gray polygon). In addition, relatively light nestlings at hatch completion (1 or 2g; medium and dark-gray polygon, respectively) reached a slower growth rate, later inflection point and lighter asymptotic mass than the heaviest nestling in the brood at hatch completion (0g, light gray polygon). All lines are approximated using parameter estimates from the nonlinear (Logistic) mixed effects model, and colored polygons indicate one standard error above and below the mean nestling mass (i.e., line).

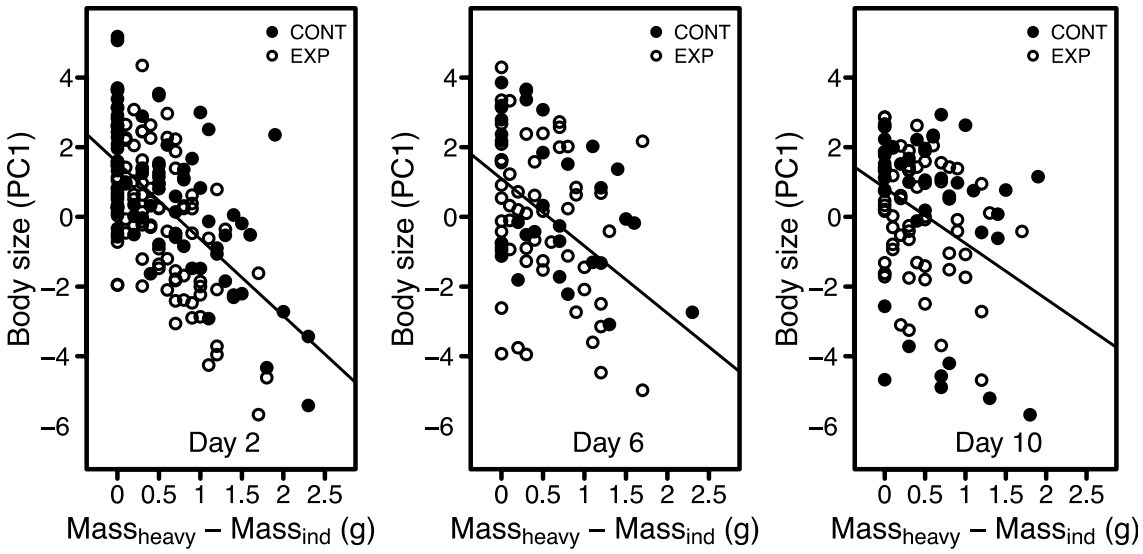

Figure 3.12: Relationship between nestling size (PC1) and a nestling's relative mass at hatch completion for control (CONT; closed symbols) and experimental (EXP; open symbols) nestlings at each of two (left panel), six (middle panel), and ten days (right panel) after the first egg hatched. Relatively light nestlings were small at each measurement day, and the relationship between size and relative mass at hatch completion was not influenced by treatment. Points are derived from actual data and lines are approximated using parameter estimates of the best models.

CHAPTER 4: CONCLUSIONS

Most plants and animals produce more offspring than can be supported by local resource availability. The production of supernumerary offspring causes siblings to compete for access to limited resources provided by the parents and increases the variation in offspring growth and survival (Mock and Parker 1997). Evolutionary ecologists seek to understand both how this variation is produced and why behaviors that increase variation in offspring growth and survival are maintained in free-living populations. The research I have presented in this dissertation fills in some of the gaps in our current knowledge about variation in offspring growth, including how to measure variation in growth and how and why some avian parents increase this variation directly by regulating timing of embryonic development (incubation). Specifically, I show that random effect estimates from nonlinear mixed effects models can be used to uncover variation in offspring growth not detected by conventional analyses (Chapter 2), and that early incubation increases the variation in offspring size primarily at hatching and likely occurs as a parental strategy that maintains both pre-hatching survival and post-hatching survival and growth (Chapter 3).

In Chapter 2, I developed a novel analytical technique to characterize variation in offspring growth. Most studies that analyze differences in growth between populations or experimental treatments focus primarily on differences in growth rate. However, these analyses ignore the potential importance of other aspects of growth that might cause

variation in offspring development (Sofaer et al. 2013). To overcome this problem, I used nonlinear mixed effects models to analyze multiple aspects of growth (e.g. mass change) simultaneously and show that much of the variation in offspring growth within populations may be explained by between-individual differences in timing of peak mass increase (i.e. inflection point) and final adult mass (i.e. asymptotic mass). These results highlight the importance of parameters other than growth rate for explaining variation in offspring development, and show the utility of nonlinear mixed effects model for uncovering previously undetected variation in offspring growth. In addition, this analytical technique can be adapted to other species and taxa that display sigmoidal growth and thus should have broad application for studies interested in understanding intraspecific variation in growth.

I also proposed clear definitions that characterize alternative growth patterns for developing offspring. A common problem with the literature describing variation in offspring growth is that the words used to describe suboptimal growth (e.g. catch-up growth, compensatory growth) are not clearly defined and hence often are used interchangeably (Hector and Nakagawa 2012). This may stem from the fact that alternative growth patterns have been difficult to characterize with conventional analytical techniques. Thus, I showed that alternate growth patterns can be defined clearly using the three growth parameters that characterize many nonlinear (sigmoidal) growth functions: timing of development, growth rate and final adult size. Importantly, the definitions for these suboptimal growth patterns make a clear distinction between compensatory, catch-up growth, and slow growth, which should allow researchers to document the prevalence of these alternative growth patterns. In addition, by clarifying

the definitions of suboptimal growth, future studies can determine which aspects of growth (e.g. delayed development, faster than optimal peak growth rates, or small body size) have the greatest impact on future survival and fecundity.

In Chapter 3, I used a manipulative experiment to test ultimate (i.e., evolutionary) explanations about why parent birds often initiate embryonic development (incubation) before all eggs have been laid. More than fifty years ago, David Lack (1947) proposed that female birds increase the variation in offspring size at hatching by beginning incubation prior to the last-laid egg. This increased variation reduces competition between siblings, which allows parents to feed offspring based on size and competitive ability and is thought to maximize the number of high quality offspring that fledge (Clark and Wilson 1981, Magrath 1990). Thus, Lack (1947, 1954) suggested that early incubation is an adaptive strategy used by parents to maximize the production of high quality young. I manipulated when female could begin incubation to test two of the leading hypotheses that seek to explain the adaptive significance of early incubation (Stoleson and Beissinger 1995). The first hypothesis, the egg viability hypothesis, suggests that early incubation maximizes pre-hatching (embryo) survival by reducing the amount of time undeveloped embryos are exposed to the environment (Arnold et al. 1987). The second set of hypotheses, the adaptive hatching pattern hypotheses, suggest that early incubation establishes a competitive size hierarchy among offspring at hatching that enables parents to maximize the number of high quality young that fledge (Stoleson and Beissinger 1997). I found that early incubation caused embryo survival to be high in all but the earliest-laid eggs, suggesting that females begin incubation too late to maximize survival in all embryos. I found no evidence that early incubation increased the

overall (e.g. mean) number or quality of hatched offspring that fledge. Instead, nestlings that hatched late were smaller than their siblings at hatching and suffered reduced growth and survival. However, nestlings that were relatively light at hatching exhibited reduced growth and survival in both asynchronously and synchronously hatching nests. Overall, these results suggest that females can increase offspring growth and survival by reducing the competitive size hierarchy caused by early incubation. Thus, I found that females began incubation too late to maximize embryo survival but too early to maximize the number of high quality young that fledge. Taken together, these results suggest that timing of incubation may occur as a tradeoff between maintaining both embryo survival and post-hatching survival and growth. This may explain why most birds that lay eggs over multiple days begin incubation before all egg are laid, and suggests that early incubation may be an adaptive strategy used by parents to maximize the number of embryos that survive the incubation and nestling periods to fledge. In addition, these results suggest that the widespread adoption of early incubation may be a consequence of unique adaptations in birds associated with offspring production (prolonged oviposition) and embryonic development (parental incubation).

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