Using non-linear mixed effects models to identify patterns of chick growth in House Sparrows Passer domesticus

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For many animals, adult size is an important determinant of fitness. Thus, after a period of food restriction, offspring often grow quickly to approach an optimal size. Offspring 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, the most common statistical techniques make it difficult to differentiate alternative growth patterns among developing offspring. Here, I show how random effect estimates can be used to uncover important variation in growth in a short-lived passerine, the House Sparrow Passer domesticus. Specifically, I show that much of the variation in offspring growth can be explained by differences in the timing of peak growth and in final adult size, both within a single population and within treatments of an experimental manipulation. 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 delay development either maintain slow but steady growth across development and reach a small adult size, or extend the period of rapid growth to reach an optimal size, indicative of catch-up growth. This pattern of extending the period of rapid growth may allow offspring to minimize the cellular damage caused by compensatory growth but still maximize size-related fitness benefits (e.g. increased survival and fecundity) prior to fledging.

Keywords: fixed effects, hatching asynchrony, logistic growth, post-hatching growth, suboptimal growth.

For many animals, adult size has an important influence on fitness. Thus, growth is often optimized to increase these size-related fitness traits (Blackenhorn 2005, Dmitriew 2010). In ideal conditions, growth is limited primarily by the ability of tissues to differentiate (Metcalfe & Monaghan 2001). However, conditions rarely are ideal and growth rate often exhibits considerable variation around an optimum (Schew & Ricklefs 1998). Individuals that experience nutritional deficits may delay growth until conditions improve and then increase mass rapidly to approach an optimal size (Hector & Nakagawa 2012).

Delayed growth occurs when individuals slow the rate at which tissues differentiate, causing offspring to be developmentally younger than their chronological age (Schew & Ricklefs 1998). Differences between developmental and chronological age can appear as a shift in the peak growth rate, and often result in a higher age-specific growth rate for offspring that delay development (i.e. suboptimal growers; Nicieza & Álvarez 2009). These suboptimal growers can approach the size of 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 an optimal size (catch-up growth) or (3) offspring can increase mass faster than the peak growth rate and extend the period of rapid growth to reach an optimal size (compensatory + catch-up growth; Hector & Nakagawa 2012, Table 1, Fig. S1). Compensatory growth can increase the accumulation of cellular damage and reduce survival and fecundity.

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Table 1. Change in growth parameters between House Sparrow nestlings that exhibit 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.

<table>
<thead>
<tr>
<th>Growth pattern</th>
<th>Timing ($I$)</th>
<th>Growth ($K$)</th>
<th>Size ($A$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catch-up</td>
<td>Delay</td>
<td>Slower/no change</td>
<td>No change</td>
</tr>
<tr>
<td>Compensatory</td>
<td>Delay</td>
<td>Faster</td>
<td>Smaller</td>
</tr>
<tr>
<td>Catch-up + compensatory</td>
<td>Delay</td>
<td>Faster</td>
<td>Smaller</td>
</tr>
<tr>
<td>Slow</td>
<td>Delay</td>
<td>Slower</td>
<td>Smaller</td>
</tr>
</tbody>
</table>

(Mangel & 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 (Drummond et al. 2003, Alonso-Alvarez et al. 2006, Wilbur & Rudolf 2006).

Like many animals, postnatal 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 analysing multiple growth parameters simultaneously because of the potential importance of variation in timing of development ($I$) and adult size ($A$; Sofaer et al. 2013). Within a species or population, nestlings that exhibit optimal growth are often characterized by an early (low) inflection point, fast (high) growth rate and heavy (high) asymptotic mass. Alternatively, nestlings that experience slow growth are characterized by a late (high) inflection point, slow (low) growth rate and light (low) asymptotic mass. Optimal and slow growth probably represent opposite ends of a continuum, making it important to identify ways to detect variation in postnatal growth.

Non-linear mixed effects models may allow researchers to assess which aspects of growth can explain important variation in postnatal development (Sofaer et al. 2013). Mixed effects models analyse data at multiple levels to account for the correlated structure in hierarchical datasets (Pinheiro & Bates 2000), as occurs when analysing multiple measurements of the same individual or analysing measurements on groups of related individuals. At the first level, fixed effects 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 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 are often used to examine variation in offspring growth (Huchard et al. 2014, Vicenzi et al. 2014).

I used a non-linear (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 curve to identify whether nestlings in a North Carolina population exhibited optimal and suboptimal growth. I tested whether suboptimal growers exhibited compensatory, catch-up or slow growth. 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 grew 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 as optimal growers.

In addition, I used non-linear mixed effects models to identify variation in growth both within and between experimental treatments, by analysing growth data from an experiment in which I manipulated the time when females could begin incubation. Most female birds begin incubation before all eggs are laid, which often causes eggs to hatch (asynchronously) over one or more days and creates a developmental size hierarchy among offspring in a nest (Clark & Wilson 1981). Although hatching asynchrony increases the variation in offspring growth and survival, it has been proposed as an adaptive strategy used by parents to increase the number of high-quality offspring that fledge (Lack 1947, 1954, Magrath 1990, Stoleson & Beissinger 1995). Therefore, most studies that examine how early incubation (and hatching asynchrony) influences nestling growth in birds have looked for mean differences in growth...
between synchronously and asynchronously hatching nests. This approach is consistent with examining the fixed effect of treatment in mixed effects models. In addition to estimating these fixed effects, mixed effects models also allow researchers to characterize variation in growth within each experimental treatment using random effects. Thus, I used fixed effect estimates to uncover growth differences between experimental treatments (as is done in typical analyses), and random effects to disentangle variation in growth within each treatment. These random effect estimates may allow researchers to identify growth differences between synchronously and asynchronously hatching nests that typically are hidden in conventional analyses of offspring growth.

**METHODS**

I studied nestling growth at two beef cattle farms near Yanceyville, North Carolina, USA (36.41°N, 79.34°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.

In 2011 and 2012, I studied 182 nestlings from 44 un-manipulated nests. In 2013 and 2014, I performed a manipulative experiment with two treatments: an experimental treatment in which each egg was replaced with a dummy egg on the day it was laid and then the entire clutch was moved back to the original nest when females stopped laying eggs at clutch completion, and a control treatment in which each egg was replaced with a dummy egg on the day it was laid. I recorded clutch size after females laid the last egg and checked nests 5 days after clutch completion. Beginning 9 days after clutch completion, I visited nests at least once a day until all eggs hatched. I visited nests almost daily after hatching and weighed nestlings to the nearest 0.1 g with a 60-g Pesola scale. I identified individual nestlings by uniquely marking nestlings’ toes until they were ringed with a uniquely numbered identification ring approximately 7 days post-hatching. Because nestlings can fledge prematurely several days before the end of the nestling period, measurements stopped after mass became asymptotic for a majority of the nestlings in a brood, which typically occurred between 10 and 12 days post-hatching. The number of mass measurements per individual varied from 1 to 13, with a median of 9.5 measurements per nestling.

**Statistical analyses**

All analyses were conducted 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 & Anderson 2002). All values are reported as the mean ± 1 se.

**Identifying alternative growth patterns using non-linear mixed models**

To characterize the growth of House Sparrow nestlings in this population, I wrote and implemented a self-starting function for the logistic curve typically used to analyse growth of passerine nestlings (Starck & Ricklefs 1998):

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

where $w_t = \text{mass at time } t \ (g)$, $A = \text{asymptotic mass } (g)$, $K = \text{growth rate constant } (1/\text{day})$, $I = \text{the inflection point of the growth curve } (\text{days})$, and $t = \text{nestling age } (\text{days})$. I fitted all logistic growth curves using maximum likelihood in the nlme package of R (Pinheiro et al. 2011). 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 all three growth parameters ($I, A$ and $K$) as fixed effects in each model and used AIC 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 $K$. 

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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). For all models, the fixed effect (e.g. $A$) estimated the growth parameter of a typical (e.g. median) nestling from the population (Table S1). The brood-level random effect (e.g. $A_i$) estimated how the growth parameter of a typical nestling deviates from a typical nestling in the population (Table S1). The nestling-level random effect (e.g. $A_j$) estimated how the growth parameter of an individual nestling deviates from a typical nestling in its brood (Table S1). Thus, the sum of the brood- and nestling-level random effects estimated how the growth parameter of an individual nestling deviates from that of a typical nestling in the population. I show results using the sum of the brood- and nestling-level random effects because I was interested in the variation in growth among individuals in this population. I used a linear mixed effects model with brood as a random intercept to determine whether a relationship existed between the random effect estimates for $A$ and $I$.

I used the 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 reduced 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 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; Fig. 1). Nestlings exhibited delayed growth if the random effect estimate for the inflection point was greater than one standard deviation below 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; Fig. 1). These delayed growers were probable candidates for catch-up or compensatory growth. Nestlings exhibited reduced growth if the random effect estimate

Table 2. Model selection for the random effect structure of the non-linear (logistic) mixed effects model examining postnatal growth within a population of House Sparrows. Each model included a population-level fixed effect (e.g. $A$) for each growth parameter and brood-level (e.g. $A_i$) and nestling-level (e.g. $A_j$) 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, probably because $I$ and $K$ were highly correlated.

<table>
<thead>
<tr>
<th>Model</th>
<th>Random effects</th>
<th>AIC</th>
<th>$\Delta$AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>$W_{ijk} = \frac{A_i A_j}{1 - e^{-K_i - I_j}} + e_{ijk}$</td>
<td>$A, I$</td>
<td>12 107</td>
<td>0</td>
</tr>
<tr>
<td>$W_{ijk} = \frac{A_i A_j}{1 - e^{-K_i - I_j}} + e_{ijk}$</td>
<td>$A, K$</td>
<td>13 298</td>
<td>1191</td>
</tr>
<tr>
<td>$W_{ijk} = \frac{A_i A_j}{1 - e^{-K_i - I_j}} + e_{ijk}$</td>
<td>$A$</td>
<td>13 485</td>
<td>1378</td>
</tr>
<tr>
<td>$W_{ijk} = \frac{A_i A_j}{1 - e^{-K_i - I_j}} + e_{ijk}$</td>
<td>$I$</td>
<td>13 860</td>
<td>1753</td>
</tr>
<tr>
<td>$W_{ijk} = \frac{A_i A_j}{1 - e^{-K_i - I_j}} + e_{ijk}$</td>
<td>$K$</td>
<td>16 427</td>
<td>4320</td>
</tr>
</tbody>
</table>

Figure 1. Variation in growth within the North Carolina population of House Sparrows. 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 reduced growth (dark grey box) reached a light asymptotic mass independent of the location of the inflection point.
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; Fig. 1). These reduced growers are probable candidates for slow growth.

**Identifying growth patterns of suboptimal growers**

To identify whether suboptimal (i.e. delayed and reduced) 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 reduced growers. I divided the nestling period into four distinct growth phases (early, peak, late and asymptotic growth) that corresponded to periods of linear growth. The inflection point for each of optimal, delayed and reduced growers was used to delineate these linear growth phases, which controlled for the shift in timing of peak growth. The early growth rate was estimated as the linear change in mass from 3 to 5 days before the inflection point of each growth pattern. The peak growth rate was estimated as the linear change in mass that occurred 3 days before to 3 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 3 to 5 days after the inflection point of each growth pattern. Finally, the asymptotic growth rate was estimated as the linear change in mass from 5 to 7 days after the inflection point of each growth pattern.

**Examining alternative growth patterns between experimental treatments**

I used growth data from the experimental manipulation in 2013–2014 to assess whether non-linear mixed effects models can be used to identify alternative growth patterns within experimental treatments. I included treatment as a fixed effect for the three growth parameters, which identified growth differences between experimental and control nests, and AIC to identify the best random effects structure for this model (Table 3). In this analysis, the fixed effects (e.g. A and A + A1 * trt) estimated the growth parameter of a typical (e.g. median) nestling from each treatment (Table S1). The brood-level random effect (e.g. A1) assessed how the growth parameter of a typical nestling from an individual brood deviated from a typical nestling from the same treatment (Table S1). The nestling-level random effect (e.g. Aij) assessed how the growth parameter of an individual nestling deviated from a typical nestling in its brood (Table S1). Thus, the sum of the brood- and nestling-level random effects assessed how the growth parameter of an individual nestling deviated from a typical nestling from the same treatment. Again, I show results using the sum of the brood- and nestling-level random effects. However, the results from this analysis identify the variation in growth among individuals in each treatment instead of among individuals in a single population. I used a linear mixed effects model with brood as a random intercept to determine whether a relationship existed between the random effect estimates for A and I, and whether this relationship was influenced by treatment.

**Table 3.** Model selection for the non-linear (logistic) mixed effects model examining differences in postnatal growth of House Sparrows between treatments. Each model included treatment-level fixed effects for each growth parameter (treatment 1 (e.g. A; control) and treatment 2 (e.g. A + A1; manipulated)) and brood-level (e.g. A) and nestling-level (e.g. Aij) 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 brood- and nestling-level random effects for A and I. The model that included random effects for all three growth parameters had the lowest AIC value (5962), but was not considered a top model because the random effects for I and K were highly correlated (|r| > 0.9).

<table>
<thead>
<tr>
<th>Model</th>
<th>Fixed effects</th>
<th>Random effects</th>
<th>AIC</th>
<th>ΔAIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{W}<em>{ijk} = \frac{A + A</em>{ij} \cdot \text{trt} + A_{ij} \cdot A_{ij}}{1 + e^{-\left(\frac{A + A_{ij} \cdot \text{trt} + A_{ij} \cdot A_{ij}}{A_{ij}}\right)}} + e_{ijk} )</td>
<td>A, I, K</td>
<td>A, I</td>
<td>6403</td>
<td>0</td>
</tr>
<tr>
<td>( \text{W}<em>{ijk} = \frac{A</em>{ij} \cdot \text{trt}}{1 + e^{-\left(\frac{A_{ij} \cdot \text{trt}}{A_{ij}}\right)}} + e_{ijk} )</td>
<td>A, I, K</td>
<td>I, K</td>
<td>6480</td>
<td>0</td>
</tr>
<tr>
<td>( \text{W}<em>{ijk} = \frac{A</em>{ij} \cdot \text{trt} + A_{ij}}{1 + e^{-\left(\frac{A_{ij} \cdot \text{trt} + A_{ij}}{A_{ij}}\right)}} + e_{ijk} )</td>
<td>A, I, K</td>
<td>A, K</td>
<td>6991</td>
<td>588</td>
</tr>
<tr>
<td>( \text{W}<em>{ijk} = \frac{A</em>{ij} \cdot \text{trt} + A_{ij}}{1 + e^{-\left(\frac{A_{ij} \cdot \text{trt} + A_{ij}}{A_{ij}}\right)}} + e_{ijk} )</td>
<td>A, I, K</td>
<td>A</td>
<td>7022</td>
<td>619</td>
</tr>
<tr>
<td>( \text{W}<em>{ijk} = \frac{A</em>{ij} \cdot \text{trt}}{1 + e^{-\left(\frac{A_{ij} \cdot \text{trt}}{A_{ij}}\right)}} + e_{ijk} )</td>
<td>A, I, K</td>
<td>I</td>
<td>7625</td>
<td>1222</td>
</tr>
<tr>
<td>( \text{W}<em>{ijk} = \frac{A</em>{ij} \cdot \text{trt}}{1 + e^{-\left(\frac{A_{ij} \cdot \text{trt}}{A_{ij}}\right)}} + e_{ijk} )</td>
<td>A, I, K</td>
<td>K</td>
<td>9111</td>
<td>2708</td>
</tr>
</tbody>
</table>
Identifying treatment differences in growth patterns for optimal and suboptimal growers

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 assess whether growth rate differed between treatments for optimal, delayed and reduced growers. I divided the nestling period into four distinct growth phases (early, peak, late and asymptotic growth) that corresponded to periods of linear growth. The growth phases were delineated as previously described, which controlled for the shift in timing of peak growth for optimal, delayed and reduced growers in each treatment.

RESULTS

Identifying alternative growth patterns using non-linear mixed models

The non-linear mixed effects model that included both brood- and nestling-level random effects for the asymptotic mass (A) and inflection point (I) was the best supported model (Table 2). The brood-level random effect explained more of the residual variation than the nestling-level random effect for both A and I (Table 4). A negative relationship existed between A and I (coefficient estimate: $-2.53 \pm 0.20$; $t = -12.91$, $P < 0.001$), suggesting that light nestlings in this population reached their inflection point later than heavy nestlings (Fig. 1).

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 reduced growth. The inflection point was shifted by a mean of $-0.22$, $0.98$ and $0.36$ days from the population-level fixed effect (4.44 days) for optimal, delayed and reduced growers, respectively (Table 5). The asymptotic mass was shifted by a mean of $0.77$, $-0.19$ and $-5.93$ g from the population-level fixed effect (26.63 g) for optimal, delayed and reduced growers, respectively (Table 5).

Identifying growth patterns of suboptimal growers

The peak growth rate was faster in optimal than in suboptimal growers (indicating no compensatory growth; Fig. S2). All suboptimal growers extended their period of rapid growth (Fig. S2), but only delayed growers approached an optimal size (indicating catch-up growth). The early growth rate increased more rapidly in nestlings that displayed optimal growth (2.10 $\pm$ 0.04 g/day) than in nestlings that displayed delayed growth (1.85 $\pm$ 0.08 g/day; $t = 2.68$, $P = 0.008$) and reduced growth (1.42 $\pm$ 0.08 g/day; $t = 7.31$, $P < 0.001$; Fig. 2). The peak growth rate also increased more rapidly in nestlings that displayed optimal growth (2.91 $\pm$ 0.03 g/day) than in nestlings that displayed delayed (2.57 $\pm$ 0.07 g/day; $t = 4.65$, $P < 0.001$) and reduced growth (1.79 $\pm$ 0.07 g/day; $t = 15.35$, $P < 0.001$; Fig. 2).

Examining alternative growth patterns between experimental treatments

Growth rate was faster in control (0.48 $\pm$ 0.005) than in manipulated nests (0.45 $\pm$ 0.004; $t = 4.04$, $P < 0.001$) and was similar between treatments for the location of the inflection point (control: $4.57 \pm 0.11$ days; manipulated: $4.73 \pm 0.11$ days; $t = -1.07$, $P = 0.282$) and asymptotic mass (co-
trol: 26.52 ± 0.80 g; manipulated: 26.31 ± 0.78 g; \( t = 0.18, \ P = 0.856 \); Fig. 3a). The best random effect structure included both brood- and nestling-level random effects for the asymptotic mass (\( A \)) and inflection point (\( I \); Table 3). A negative relationship existed between the random effects for \( A \) and \( I \) (coefficient estimate = \(-1.38 \pm 0.42\); \( t = -3.33, \ P = 0.001 \) and this relationship was similar between treatments (treatment by random effect interaction: \( t = -0.005, \ P = 0.996 \); Fig. 4).

Consistent with the population-level analysis, most nestlings that survived to fledge exhibited optimal growth in both control (61 nestlings; 69.4% of control young) and manipulated nests (59 nestlings; 65.6% of experimental young). In addition, approximately half the remaining nestlings in control nests exhibited delayed growth (14 nestlings; 16.5% of control young) and the other half exhibited reduced growth (12 nestlings; 14.1% of control young). In control nests, the inflection point was shifted by a mean of \(-0.20, 0.82\) and \(-0.09\) days from the treatment-level fixed effect and the asymptotic mass was shifted by a mean of 1.15, \(0.07\) and \(7.02\) g from the treatment-level fixed effect for optimal, delayed and reduced growers, respectively (Table 5). Half the remaining nestlings in manipulated nests exhibited delayed growth (16 nestlings; 17.2% of experimental young) and the other half exhibited reduced growth (16 nestlings; 17.2% of experimental young). In manipulated nests, the inflection point was shifted by a mean of \(-0.16, 0.94\) and \(-0.31\) days from the treatment-level fixed effect and the asymptotic mass was shifted by a mean of \(0.30, 7.50\) g from the treatment-level fixed effect for optimal, delayed and reduced growers, respectively (Table 5).

### Table 5

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Inflection point (d)</th>
<th>Asymptotic mass (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fixed</td>
<td>Opt</td>
</tr>
<tr>
<td>POP</td>
<td>NA</td>
<td>4.44</td>
</tr>
<tr>
<td>MANIP</td>
<td>EXP</td>
<td>4.73</td>
</tr>
<tr>
<td>CONT</td>
<td>4.57</td>
<td>-0.20</td>
</tr>
</tbody>
</table>

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0.73, 0.30 and $-7.50$ g from the treatment-level fixed effect for optimal, delayed and reduced growers, respectively (Table 5).

**Identifying treatment differences in growth patterns for optimal and suboptimal growers**

**Optimal growers**
Optimal growers in asynchronous (control) nests displayed a faster peak growth rate than optimal growers in experimentally synchronized (manipulated) nests (Fig. 3b). However, optimal growers in synchronized nests extended the period of rapid growth to reach an optimal size. (c)Delayed growers from naturally asynchronous nests also increased mass faster than delayed growers from experimentally synchronized nests. (d) Reduced growers grew equally poorly in both naturally asynchronous and experimentally synchronized nests. Points and error bars are derived from actual data and lines are derived from estimates of the best-fit model.

Figure 3. Differences in House Sparrow offspring growth between nests in which incubation was naturally asynchronous (CONT) and nests in which incubation was experimentally synchronized (EXP). (a) Overall, control nestlings increased mass faster than experimental nestlings but had a similar inflection point and asymptotic mass. (b) Optimal growers in naturally asynchronous nests increased mass faster than optimal growers from experimentally synchronized nests. However, optimal growers from synchronous nests extended the period of rapid growth to reach an optimal size. (c) Delayed growers from naturally asynchronous nests also increased mass faster than delayed growers from experimentally synchronized nests. (d) Reduced growers grew equally poorly in both naturally asynchronous and experimentally synchronized nests. Points and error bars are derived from actual data and lines are derived from estimates of the best-fit model.

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mentally synchronized nests (2.37 ± 0.09 g/day; Fig. 3c). The experimental treatment had little effect on the early (control = 1.80 ± 0.16 g/day, manipulated = 1.75 ± 0.13 g/day; t = 0.23, P = 0.821), late (control = 1.98 ± 0.49 g/day, manipulated = 2.25 ± 0.37 g/day; t = −0.43, P = 0.670) and asymptotic growth rates (control = 0.50 ± 0.70 g/day, manipulated = 1.18 ± 0.34 g/day; t = −0.86, P = 0.396) for delayed growers.

**Reduced growers**

Reduced growers grew similarly poorly in both asynchronous and experimentally synchronized nests (Fig. 3d). Specifically, the experimental treatment had little effect on the early (control = 1.43 ± 0.12 g/day, manipulated = 1.44 ± 0.10 g/day; t = −0.05, P = 0.964), peak (control = 1.77 ± 0.08 g/day, manipulated = 1.74 ± 0.08 g/day; t = 0.28, P = 0.784), late (control = 1.53 ± 0.50 g/day, manipulated = 1.39 ± 0.55 g/day; t = 0.17, P = 0.863) and asymptotic growth rates (control = 0.09 ± 0.53 g/day, manipulated = 2.53 ± 0.63 g/day; t = −0.86, P = 0.396) for reduced growers.

**DISCUSSION**

Random effect estimates from non-linear mixed effects models can be used to uncover important variation in postnatal growth. Specifically, asymptotic mass (A) and timing of peak growth (I) exhibit substantial variation among nestlings within a single population and among nestlings within experimental treatments. In addition, more of the variation within each population (or treatment) 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 also are supported by 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 non-linear mixed effects models may be a powerful analytical technique to identify important but undetected variation in postnatal growth.

Using a mixed modelling 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 growth to reach an optimal size (Lepczyk & Kara-sov 2000). I found no evidence of compensatory growth, probably because growth has been optimized evolutionarily at the peak growth rate of optimal growers (Ricklefs 1979). Approximately half of all offspring that delayed development maintained a slow but fairly stable growth rate across development and reached a small adult size. Despite the differences in final size between nestlings that displayed catch-up and reduced (i.e. slow) growth, all suboptimal growers increased mass faster than optimal growers toward the end of development.

Non-linear mixed effects models can also disentangle variation in growth within manipulative experiments. Using the fixed effects estimates from a non-linear mixed effects model, nestlings from naturally asynchronous (control) nests increased mass faster and reached a similar inflection point and asymptotic mass than nestlings from experi-
mentally synchronized nests. This pattern is common in experimental manipulations of asynchronous development (e.g. incubation onset, hatching asynchrony) and may be explained by decreased competition for food in asynchronously hatching nests (Magrath 1989, Hébert 1993, Gilby et al. 2011). However, substantial individual and brood-level variation existed in the asymptotic mass and location of the inflection point, and this variation made it possible to identify nestlings that displayed optimal, delayed and reduced growth within each treatment. Both optimal and delayed growers from asynchronous nests increased mass faster than optimal and delayed growers from experimentally synchronized nests, which is likely to explain why growth rate differed between asynchronous and experimentally synchronized nests. Despite the treatment differences in growth rate, optimal, delayed and reduced growers displayed similar growth trajectories in naturally asynchronous and experimentally synchronized nests. Thus, fixed effect estimates can be used to identify differences in growth between experimental treatments whereas random effect estimates can be used to uncover variation in growth within each treatment. As seen in other taxa (Huchard et al. 2014, Vicenzi et al. 2014), random effect estimates may be a powerful analytical technique to detect important variation in growth that often is undetected by conventional analyses. Furthermore, this approach can be used to identify variation in growth between two or more populations of the same species.

Several studies in both wild and captive populations have shown that passerines can delay development but reach an optimal size prior to fledging (e.g. Lepczyk & Karasov 2000, Alonso-Alvarez et al. 2007, Hegyi & Torok 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 an optimal size by reaching a faster age-specific growth rate. I show that faster age-specific growth rates can be achieved by temporarily delaying development, which can uncouple the relationship between developmental and chronological age (Schew & Ricklefs 1998). Thus, future studies should control for differences in timing of development and directly test whether differences exist between peak growth rates (Nicieza & 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 & Nagawa 2012). In fact, some form of all three growth parameters (timing of growth (I), peak growth rate (K) and final size (A)) can be used to distinguish between alternative growth patterns (Table 1), even in studies that do not use non-linear growth functions. Researchers first need to determine whether offspring have shifted the timing of peak growth (e.g. delayed growth). For analyses using non-linear growth functions, a shift in the location of the inflection point may indicate that offspring have delayed growth temporarily. For analyses not using non-linear growth functions, delayed growth can be identified by a delay (or suspension) in increase of mass or size during food restriction. Next, the peak growth rate can be estimated to identify whether offspring that delayed development displayed a faster peak growth rate than optimal growers (exhibit compensatory growth). By controlling for differences in timing of development, peak growth rates will be estimated at later ages for suboptimal growers, but these analyses will enable comparisons of growth at similar developmental stages (Nicieza & 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 identify whether offspring attain an optimal size (exhibit catch-up growth) or reach a small size prior to fledging. Estimates of each of these growth parameters are derived easily from non-linear mixed effects models but also can be estimated by concentrating mass measurements around the period of peak growth and the attainment of final adult size.

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 size near fledging in altricial birds (Schwagmeyer & Mock 2008, Dybala et al. 2013, Bouwhuis et al. 2015). In addition, nestlings that are large enough to survive to independence often have a random chance of recruiting into the breeding population (Magrath 1991). I have shown that nestlings that exhibit suboptimal growth appear to extend the period of rapid growth to approach an optimal size, but only some of these nestlings reach an
optimal size 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 size are probably important.

House Sparrows are cavity-nesters and may have 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 & 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 (R.A. Aldredge unpubl. data). 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 by increasing mass faster than the peak growth rate of optimal growers. By using random effect estimates from non-linear mixed effects models, researchers may be able to tease apart important variation in offspring growth and uncover developmental strategies that have evolved to maximize fitness.

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**SUPPORTING INFORMATION**

Additional Supporting Information may be found in the online version of this article:

**Figure S1.** Theoretical growth patterns for offspring displaying optimal and suboptimal growth.

**Figure S2.** Daily growth rate for optimal, delayed and reduced growers.

**Table S1.** Table showing how the non-linear mixed effects models partition the variation in asymptotic mass for the population-level and treatment-level analyses.