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Early Nutrition Causes Persistent Effects on Pheasant Morphology

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ABSTRACT

Differences in growth conditions during early ontogeny have been suggested to cause permanent effects on the morphology and quality of birds. Yearly variation in growth conditions could thus result in morphological and quality differences between cohorts. In this study, we investigated the effect of small differences in the dietary protein content of captive ring-necked pheasants (*Phasianus colchicus*) during their first 8 wk posthatching. An experimental increase of the proportion of dietary protein during the first 3 wk of life accelerated growth, whereas a similar manipulation during the following 5 wk had only a limited effect. Compensatory growth during the postexperimental period equalized the size of chicks from different experimental treatments. However, a difference in tarsus length resulting from experimental treatment during the first 3 wk remained into adulthood. Furthermore, the protein content of the diet during the first 3 wk had an effect on the degree of fluctuating asymmetry in tarsus length, suggesting persistent effects on the quality of birds. The results of this study may explain size differences between cohorts that exist in pheasants and may also provide a link between the use of pesticides in agriculture and population effects on pheasants.

Introduction

Despite the theoretical prediction of negative trade-offs between fitness components, positive associations are common in nature (Roff 1992; Stearns 1992). Such positive association can be attributed to individual differences in the ability to acquire resources (van Noordwijk and de Jong 1986). One route by which such differences can develop is through variation in environmental conditions during growth, causing individuals to develop different phenotypic qualities (Lindström 1999). Such a "silver-spoon" effect has been suggested to account for quality differences observed between birds (Gebhart-Henrich and Richner 1998; Lindström 1999). In nature, intrasexual size/quality differences between individuals born in different habitats (e.g., Ulfstrand et al. 1981; Richner 1989) or belonging to different cohorts (e.g., Larsson and Forslund 1991; Witzell 1991) in the same population have been attributed to varying growth conditions. Systematic differences between habitats (e.g., Larsson and Forslund 1991) or years (e.g., Lepage et al. 1998) in food availability caused by, for example, density dependence (Cooke et al. 1995), variation in weather conditions (Witzell 1991), or timing of breeding (Sedinger et al. 1995) may produce these effects.

Recently, much attention has been focused on how various forms of environmental stress might affect developmental stability as reflected by fluctuating assymmetries in otherwise bilaterally symmetrical traits (e.g., Palmer and Strobeck 1986; Parsons 1990; Møller and Swaddle 1997; Fair et al. 1999). The level of fluctuating asymmetry might be an indicator of individual quality, either because fluctuating asymmetry itself affects fitness (e.g., Møller 1992; Morris 1998) or because it indicates some property of the developmental process that in turn relates to future fitness (Palmer and Strobeck 1986; Clarke 1995). Although it has been argued that fluctuating asymmetry may reflect an individual's ability to handle environmental stress during development (Palmer and Strobeck 1986; Leary and Allendorf 1989; Parsons 1990), the evidence is still scant (Taether 1996; Clarke 1998). Few studies have directly investigated the effect of variation in food availability or food quality during early ontogeny on subsequent development of fluctuating asymmetries (Møller and Swaddle 1997).

Pheasant chicks depend on access to dietary protein for their development and survival. The size of adult pheasants has been shown to differ between cohorts (Wittzell 1991). Wittzell showed that the size of juvenile birds in winter was related to weather conditions during chick development, most likely because of the effect of temperature on insect abundance and activity. In contrast, Woodard et al. (1977) found that a low-protein intake during the growth stage resulted in slower growth in pheasants but had no effect on final weight.

The aim of our experiment was to test whether differences in nutrition during early ontogeny could cause permanent effects on the morphology and quality of adult pheasants and thus explain the differences between cohorts that can be observed in nature (Witzell 1991). We tried to mimic natural variation in early food quality to determine whether the amount of dietary protein available during early ontogeny resulted in changes in

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growth rates that were not made up by compensatory growth and in permanent effect on fluctuating asymmetry.

Material and Methods

We manipulated chick nutrition in the form of protein content of the food during early ontogeny. Experimental studies have shown that pheasant chicks require at least 20% protein in their food during development; otherwise, mortality increases (Woodard et al. 1977). Therefore, chicks received two different kinds of food: a high plane of nutrition (H), of commercial turkey starter containing 27% protein, or a low plane of nutrition (L), consisting of commercial chicken starter containing 20.5% protein (Kalkon start and Fenix start, Skånska Lantmännen). The nutritive values of these fodders were otherwise similar, with the energy content being somewhat higher for the chicken starter (12.3 MJ/kg) compared to the turkey starter (11.8 MJ/kg). After the treatment periods, all chicks were fed a commercial fodder containing 16% protein. To distinguish the importance of food during different phases of early ontogeny, the experiment was designed as a 2^3 factorial, with randomly selected male and female chicks obtaining low- and high-protein content fodder during the first 3 wk (early treatment) and weeks 4–8 (late treatment; see below).

We obtained chicks by hatching pheasant eggs obtained from a commercial breeder. The chicks hatched in a standard incubator over a 2-wk period in the beginning of June 1998. All chicks were weighed when dry on the day of hatching. Chicks were matched for mass and then randomly assigned to the early treatment. Initially the high-protein treatment consisted of 25 males and 34 females, and the low-protein treatment consisted of 29 males and 22 females. The discrepancy in numbers between males and females was because of difficulties in correctly sexing newly hatched chicks; thus, chicks could not be matched for sex. After 3 wk, we randomly selected every second chick in each treatment for a high-protein treatment and the rest for a low-protein treatment during weeks 4–8 (late treatment). The pheasants were finally sexed at the ages of 4–7 wk.

The treatment groups of pheasants were initially kept indoors, in a large room that was divided into two similar sections of 3.5 m × 3.5 m. These sections, separated only by a thin wooden wall, were identical concerning windows, ambient light, and temperature. Later, at the age of 8 wk, they were transferred to an outdoor aviary. This aviary was divided into two similar sections, each containing one treatment group. The size of each aviary section was 40 m^2 and contained a house in which the birds could avoid inclement weather. Food and water were supplied ad lib. throughout the entire experiment.

Between 0 and 280 d old, at intervals, all chicks were weighed to the nearest 0.1 g using a digital balance (Mettler Toledo) and had their tarsus length measured to the nearest 0.01 mm using digital calipers. From day 30, both left and right tarsus lengths were measured. Males were weighed and measured 29 times and females 27 times. Since pheasants did not reach an asymptotic mass during the experiment, we estimated asymptotic mass by fitting the Gompertz equation to each chick (Zullinger et al. 1984).

As a measure of fluctuating asymmetry (FA) in tarsus length, the unsigned value of left minus right was used ($|L - R|$). Since any effect on FA is likely to be small, the importance of taking measurements blind has been emphasized (Palmer 1994; Swaddle et al. 1994). However, for practical reasons, the experiment could not be performed totally blind. At each occasion, the person taking measures was aware of the experimental status of birds but unaware of the individual measurements during previous occasions. Using the model described by Swaddle et al. (1994) to calculate the repeatability of estimated asymmetries after the tarsus had reached its asymptotic length (weeks 12–40, see below), we found them to be repeatable ($F_{106, 664} = 1.48$, $P < 0.005$). This suggests that our measure of FA is accurate. To obtain a blind measurement, we let a person with previous experience of measuring pheasants but with no knowledge of the experiment measure the tarsi of each male pheasant twice (when they were 40 and 41 wk old). Unfortunately, the same procedure could not be performed for females because an outbreak of coccidiosis caused heavy mortality among females. The blind measure of FA correlated with that obtained earlier (correlation between mean measurements, $r_{30} = 0.68$, $P < 0.001$).

Repeated measures of size were analyzed using restricted maximum likelihood estimation (REML) of mixed models (Littell et al. 1996). When selecting variance-covariance structures, we followed the recommendations for repeated-measures analyses by Littell et al. (1996); in all cases, a compound symmetrical variance-covariance structure was used. To control for initial size, we included either mass at hatching or the first measurement of tarsus length as covariate but excluded it unless significant. The first measurement of tarsus length was taken at slightly different times in relation to hatching, so we corrected for age at first measurement by using the residual tarsus length when regressing the first measurement against age (in hours) at the first measurement. FA was analyzed using a REML estimation of a mixed-regression model, following van Dongen et al. (1999) and using repeated-measures ANOVA. This method compares to the mixed-model approach of Palmer and Strobeck (1986) but has several computational advantages, including testing the statistical significance of FA (van Dongen 1999). In analyses containing more than one independent variable, we tested for higher-order interactions but excluded them unless significant. All statistical tests were performed using SYSTAT (Wilkinson 1987) or SAS (Littell et al. 1996). All probability distributions are two-tailed.

Results

Growth

At hatching, there were no differences in nestling mass ($F_{3, 106} = 1.88$, $P = 0.14$) or tarsus length ($F_{3, 105} = 1.97$, $P =$

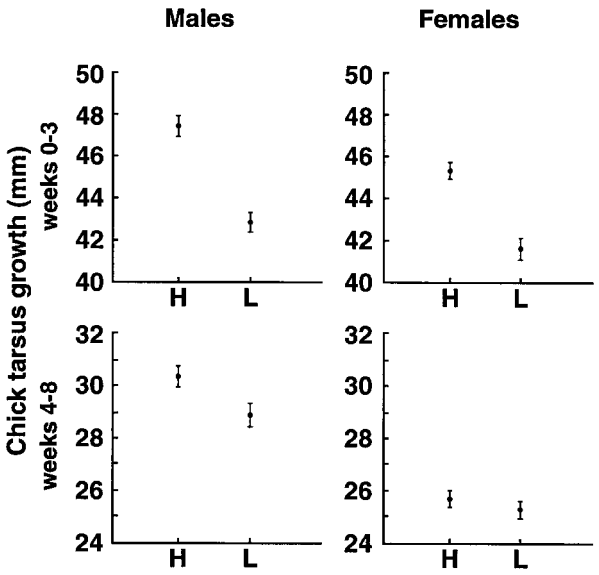

Figure 1. Growth of chick tarsus length (±SE) during the early experimental period (first 3 wk, *upper panels*) and during the late experimental period (subsequent 5 wk, *lower panels*) for males and females. The chicks received either high-protein food (*H*) or low-protein food (*L*). Means in the lower panels are adjusted for unequal sample size of the early treatment in each group. Error bars for the lower panels calculated using residuals from one-way ANOVAs including early treatment as factor.

0.12) between the four experimental groups. The manipulation of protein content during the first 3 wk had a significant effect on mass growth (ANCOVA with mass at 3 wk old as dependent variable, sex as factor, and initial mass as covariate; the effect of early treatment: $F_{1,105} = 105.40$, $P < 0.001$) and tarsus length (ANCOVA with tarsus length at 3 wk old as dependent variable, sex as factor, and initial tarsus length as covariate; the effect of early treatment: $F_{1,104} = 77.39$, $P < 0.001$; Fig. 1). Similarly, the late treatment affected mass growth (ANCOVA with mass at 8 wk old as dependent variable, sex as a factor, and mass at 3 wk old as covariate; the effect of the late treatment: $F_{1,106} = 5.67$, $P = 0.019$) and tarsus length (ANCOVA with tarsus length at 8 wk old as dependent variable, sex as a factor, and tarsus length at 3 wk old as covariate; the effect of the late treatment: $F_{1,106} = 4.18$, $P = 0.043$). The much larger effect of the early treatment had the result that size at 8 wk old was affected by the early treatment (ANCOVA with mass/tarsus length at 8 wk old as dependent variable, sex as factor, and initial mass/tarsus length as covariate; the effect of on mass: $F_{1,104} = 26.85$, $P < 0.001$; on tarsus length: $F_{1,103} = 23.19$, $P < 0.001$) but not by the late treatment (ANCOVA, mass: $F_{1,104} = 0.50$, $P = 0.48$; tarsus length: $F_{1,103} = 1.51$, $P = 0.22$).

The effect of experimental treatment on growth resulted in compensatory growth in tarsus length during the postexperimental period (from 8 to 20 wk old), such that chicks receiving less protein during early development grew faster (ANOVA with experimental treatments and sex as factors, the effect of the early treatment: $F_{1,91} = 36.14$, $P < 0.001$; the effect of the late treatment: $F_{1,91} = 3.32$, $P = 0.072$; Fig. 2). For mass, no significant effect could be detected (ANOVA as above, the effect of early treatment: $F_{1,89} = 1.72$, $P = 0.19$; late treatment: $F_{1,89} = 3.45$, $P = 0.067$). Growth in mass during the period from 20 wk old until 40 wk old was affected by the early treatment (ANOVA with experimental treatments and sex as factors: $F_{1,74} = 3.93$, $P = 0.051$), whereas the late treatment had no effect (ANOVA as above: $F_{1,74} = 0.09$, $P = 0.78$). For tarsus length, no similar trends could be detected (ANOVA, the effect of early treatment: $F_{1,76} = 0.03$, $P = 0.87$; late treatment: $F_{1,76} = 0.02$, $P = 0.89$) because tarsus length is fully grown at this age.

For calculations of the final tarsus length, we included measurements from weeks 12 to 40; at 12 wk old, male pheasants have reached 98.4% and female pheasants 98.9% of the size at 40 wk old. The final tarsus length depended on the initial experimental category (repeated-measures mixed model with treatments and sex as factors; the effect of early treatment: $F_{1,103} = 8.94$, $P = 0.004$), with a difference between treatments of 1.7 mm (mean tarsus length 88.6 ± 2.8 mm for males and

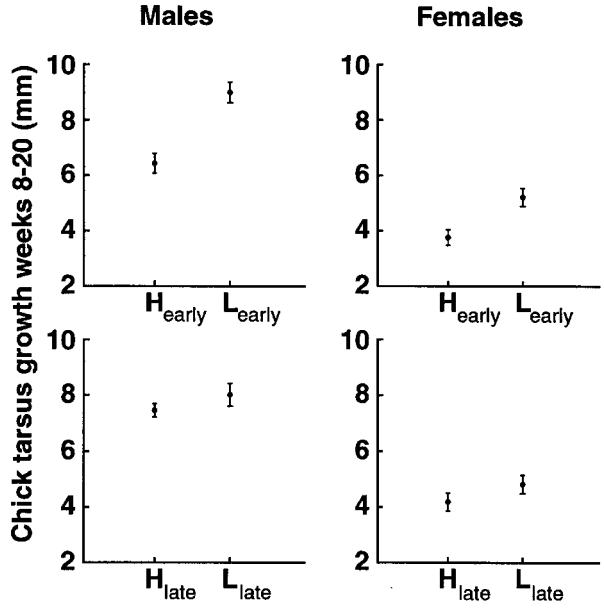

Figure 2. Compensatory growth in tarsus length (±SE) between 8 and 20 wk old for male and female chicks previously receiving high- (*H*) or low- (*L*) protein food during the early (first 3 wk, *upper panels*) or late experimental period (subsequent 5 wk, *lower panels*). Means adjusted for unequal sample size and error bars calculated as in Figure 1.

78.5 ± 2.8 mm for females). When including initial size as covariate, tarsus length was still larger in chicks fed high-protein food during the first 3 wk (repeated-measures mixed model with treatments and sex as factors, initial tarsus length as covariate, the effect of early treatment: $F_{1,99.9} = 4.25$, $P = 0.042$). The late treatment did not affect final tarsus length ($P > 0.1$ in all cases). The blind measurements on males also demonstrated an effect of the early treatment on tarsus length ($F_{1,29} = 5.12$, $P = 0.031$). This was not confounded by an effect of initial tarsus length ($F_{1,26} = 1.65$, $P = 0.21$), but when including initial tarsus length as covariate, tarsus length was nonsignificantly larger in chicks fed the high-protein fodder the first 3 wk ($F_{1,26} = 3.08$, $P = 0.091$). Mass did not reach its asymptote until late in the winter. As a measure of final size, we therefore used the measurement obtained from the Gompertz equation. Final mass, as measured in this way, was unaffected by both experimental treatments (ANOVA with sex as factor, early treatment: $F_{1,96} = 2.15$, $P = 0.15$; late treatment: $F_{1,96} = 0.50$, $P = 0.48$). Since calculations of asymptotic mass using growth equations should be viewed with caution, we repeated the analysis using the actual mass at 40 wk old. This yielded the same results (ANOVA, with mass at 40 wk old as dependent variable and sex as factor, the effect of early treatment: $F_{1,80} = 3.42$, $P = 0.07$; the effect of late treatment: $F_{1,80} = 0.002$, $P = 0.96$).

FA

In the following analyses, we included measures when birds had reached asymptotic size (weeks 12–40), but results were the same if including younger ages (see Fig. 3). Signed asymmetry demonstrated no skewness ($g_1 = -0.24$, $t_\infty = 1.00$, $P > 0.1$) but was leptokurtic ($g_2 = 4.02$, $t_\infty = 8.49$, $P < 0.001$). The degree of FA was unrelated to tarsus length (partial correlation after removing the effect of sex: $r_{104} = 0.038$). A mixed-model ANOVA (Palmer and Strobeck 1986) demonstrated a significant directional asymmetry in females ($F_{1,54} = 13.94$, $P < 0.001$) but not in males ($F_{1,51} = 0.14$, $P = 0.71$). Since this is a potentially serious problem for the analyses that is not easily corrected for (Palmer and Strobeck 1992), males and females were analyzed separately. Males demonstrated a measurable FA ($F_{51,467} = 1.55$, $P = 0.011$), whereas females did not ($F_{54,290} = 0.91$, $P = 0.65$), most likely because of a confounding effect of the directional asymmetry.

Both males and females that were given the high-protein food during their first 3 wk of life (early treatment) grew more symmetrical tarsi than those that were given the low-protein food (males: $\chi^2 = 19.32$, df = 1, $P < 0.001$, $n = 49$; females: $\chi^2 = 6.17$, df = 1, $P < 0.025$, $n = 55$). There was a similar effect of the late treatment for females ($\chi^2 = 8.16$, $P < 0.005$) but not for males ($\chi^2 = 1.51$, $P > 0.1$). REML tests may be sensitive to leptokurtic deviations from normality (van Dongen 1999); we therefore repeated the tests using Levene's test (Palmer and Strobeck 1992). There was a significant effect of the early treatment for males ($F_{1,50} = 17.53$, $P < 0.001$) and females ($F_{1,53} = 4.21$, $P = 0.045$) but not of the late treatment (males: $F_{1,50} = 0.25$, $P = 0.62$; females: $F_{1,53} = 0.67$, $P = 0.42$). Since there was a significant directional asymmetry for females, the result for females should be viewed with caution. Using the blind measures, the effect of the early treatment held true for males ($\chi^2 = 10.25$, $P < 0.005$, $n = 32$), whereas the late treatment had no effect ($\chi^2 = 0.41$, $P > 0.1$, $n = 32$). Levene's test gave the

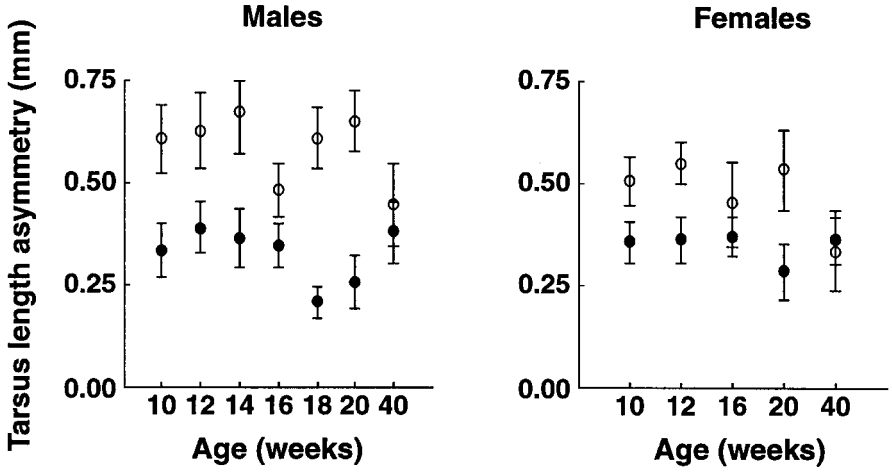

Figure 3. Fluctuating asymmetry ($|L - R|$, ±SE) in tarsus length of male and female pheasants in relation to experimental treatment during the first 3 wk of life. Chicks either received high- (*closed circles*) or low- (*open circles*) protein food. Only males were measured at weeks 14 and 18.

same result (early treatment: $F_{1,30} = 7.95$, $P = 0.008$; late treatment: $F_{1,30} = 2.52$, $P = 0.12$).

Discussion

Morphology

The protein content of food during the early development of pheasant chicks had a large impact on growth of both tarsus and mass, a result also found in studies of other precocial birds (Ricklefs et al. 1998), including the pheasant (Warner and Darda 1982). However, chicks demonstrated compensatory growth (Schew and Ricklefs 1998) after the experimental periods. The growth rate of chicks receiving low-protein food was accelerated compared to growth of chicks receiving high-protein food but may not have been so in relation to physiological age (Schew and Ricklefs 1998). Precocial birds may be more flexible in their developmental program than altricial birds and also other studies have demonstrated compensatory growth following experimental manipulation of dietary protein (Schew and Ricklefs 1998). The net result of food manipulation and compensatory growth was that final mass was unaffected by experimental manipulations, whereas final tarsus length was affected. Thus, our study demonstrates that, in spite of the occurrence of compensatory growth in pheasants, the diet during the first few weeks of life may have lifelong consequences for morphology.

Several studies have shown that food availability during development may cause lifelong effects on morphology (reviewed by Gebhardt-Heinrich and Richner 1998). In our experiment, we only manipulated dietary content, not food availability. Some other studies have found effects of dietary content on subsequent morphology (Boag 1987; Richner 1989; Dahlgren 1990; Larsson and Forslund 1991; Lepage et al. 1998). In contrast to these studies, we only manipulated diet during the earliest part of the ontogeny. Furthermore, the manipulation during the first 3 wk had a larger impact than the subsequent manipulation. Specific developmental periods may be more vulnerable than others (Schew and Ricklefs 1998), and environmental stress may have a larger impact the earlier it occurs (Desai and Hales 1997).

Although the differences in final size were small, they may be larger in the wild, where differences in dietary protein content may be larger than the small differences we used for ethical reasons. An additional explanation for the small differences found in our study could be that the unlimited postexperimental food availability could allow compensatory growth. In addition, chicks may compensate for poorer diet by different allocation of resources (Schew and Ricklefs 1998). For example, zebra finch nestlings (*Poephila guttata*) on a seed-only diet demonstrated impaired immune function, without demonstrating any detectable differences in adult morphology (Birkhead et al. 1999).

Witzell (1991) found that several morphological characters in pheasants born in the same year were more similar than between years and suggested that this was a result of the summer temperature during the chick's early development. In warm summers, the abundance of insects was high, and the birds grew better and reached a larger final size. This study demonstrated that dietary differences might indeed cause these cohort differences.

The difference in tarsus length caused by the experiment is permanent and may be important for future reproductive success. First, large males may be better intrasexual competitors (e.g., Røskaft 1983; Richner 1992). Second, large females may be more fecund by laying eggs of higher quality/weight (e.g., Cooch et al. 1991; Sedinger et al. 1995).

FA

It has been suggested that FAs of bilateral structures may be caused by nutritional stress (e.g., Parsons 1990; Møller and Swaddle 1997). Relatively few studies have investigated the development of FAs in relation to nutritional stress (but see Swaddle and Witter 1994 for a study on molt), and we are aware of no studies that have found differences in the degree of FA in relation to experimental differences of nutritional stress during early ontogeny.

In our experiment, we manipulated the amount of protein in the food during the earliest phase of development and found permanent effects on tarsus asymmetry. Thus, the experiment shows that nutrition during the earliest phase in development may have lifelong effects on the morphology and quality of individuals. It has been suggested that compensational growth may reduce initial higher degrees of FAs during development (Swaddle and Witter 1994; Taether 1996). However, in spite of compensational growth as regards the size of the trait, no change was found in the degree of FA over time. Similarly, a study of house sparrow (*Passer domesticus*) feather growth did not support the existence of a mechanism of compensational growth (Aparicio 1998).

This study also shows that there might be a connection between nutritional stress, FAs, and certain growth phases. The degree of FA in tarsus length was related to the treatment during the first 3 wk of life but not to the subsequent treatment. Furthermore, we did not find any differences in spur-length FA (T. Ohlsson and H. G. Smith, unpublished data), probably because these start to grow later in the ontogeny.

It has been suggested that FAs may be directly related to fitness, for example, because FAs in ornaments affect mate choice (e.g., Møller 1992; Watson and Thornhill 1994) or that FAs may be indirectly related to fitness because some common factor affects both fitness and FAs (Clarke 1995). We have no evidence directly relating to this but suggest that the small differences in tarsus length observed here affect neither performance or mate choice but, instead, may be indirectly related to fitness. However, a firm conclusion requires that the degree

of FA be directly compared to fitness-related traits (cf. Clarke 1998). For the pheasant, this has to await further studies.

Conclusion

Pheasant chicks require a considerable amount of protein during early growth. In the wild, pheasant chicks' protein requirement is met by a diet of invertebrates during the first weeks of life (e.g., Scott et al. 1954). There is some evidence that the use of pesticides in modern agriculture is responsible for the declines in naturally reproducing populations of pheasants by negatively affecting the abundance of invertebrates available to young chicks (e.g., Richard et al. 1999). Woodard et al. (1977) showed that mortality was substantially higher in pheasant chicks fed only 16% protein in the diet compared to control birds (20% protein). Our study demonstrates that lack of dietary protein may cause additional fitness costs because of permanent effects on both morphology and FA later in life. Thus, only measuring the effects of agricultural methods on chick production may underestimate the effects of pesticide use on pheasant population dynamics.

Acknowledgments

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