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Long-term effects of early nutrition and environmental matching on developmental and personality traits in zebra finches

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Developmental plasticity is a key feature of many organisms and individuals can benefit from early programming to optimize their phenotypes for the expected environmental conditions. However, environmental conditions may sometimes change unexpectedly. Mismatches between early and adult life, for example, can have important repercussions for adult phenotypes, potentially leading to better performance under matched than mismatched conditions as predicted by the predictive adaptive response hypothesis. We conducted a long-term experimental manipulation of dietary conditions in a population of zebra finches, *Taeniopygia guttata*. Broods were exposed to two early nutritional treatments until independence and we used a split-brood design to independently manipulate nutritional conditions after independence to create matched and mismatched nutritional environments in later life. Developmental trajectories of all individuals were followed for more than 5 years and we scored behavioural responses in trials in a special environment and while interacting with a special object three times during adult life. Overall, we found no evidence for early programming affecting morphology. Tarsus and wing length were exclusively influenced by the early nutrition. Body weight showed lasting effects of the early treatment and independent effects of nutritional condition during adulthood, but no effects of environmental matching or mismatching. Special-object trials showed effects of the adult nutritional treatment while environmental matching affected hopping activity in special environments. These behavioural responses showed substantial long-term individual stability over a 3-month period and were only marginally smaller when measured over a period of more than 4 years. Interestingly, survival of individuals from low-quality early nutritional condition was higher compared with high-quality early condition individuals, which became evident only after years of survival monitoring. Beyond the nutritional treatment itself, we found sizable brood identity effects that slowly but steadily declined with age, indicating a significant but decaying effect of natural variation in parental provisioning on adult phenotypes.

Phenotypic plasticity, the ability to modify the phenotype in response to environmental conditions, is a key feature of many organisms and an important component of phenotypic evolution (Bateson & Gluckman, 2011; West-Eberhard, 2003). Depending on whether phenotypic changes are reversible within an individual's lifetime, phenotypic plasticity can be categorized into reversible phenotypic flexibility or irreversible developmental plasticity (Piersma & Drent, 2003; West-Eberhard, 2003). While unlimited phenotypic flexibility seems ideal for optimizing phenotypes, there are often costs or constraints that limit the ability to change phenotypes reversibly (Piersma & Drent, 2003; West-Eberhard, 2003). This can make developmental plasticity more efficient for producing optimal phenotypes when the environment during early life is a sufficiently good predictor for conditions experienced later in life (Hanson & Gluckman, 2014). Individuals can thus be primed by the early environment and adjust their phenotypes adaptively. Such developmental priming may have adverse effects, however, if environmental conditions change unexpectedly (Monaghan, 2008). A key question is therefore when, how and why phenotypes are susceptible to early priming (DeWitt, Sih, & Wilson, 1998).

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A critical period for environmental priming is the growth phase during early ontogeny (Holveck & Riebel, 2009; Lindström, 1999; Naguib, Nemitz, & Gil, 2006), because important developmental decisions are made at this life stage. In species with determinate growth, this includes the size of the body and the relative proportions of its different components (Buchanan, Leitner, Spencer, Goldsmith, & Catchpole, 2004). Individuals may respond to poor initial conditions with compensatory growth, but compensation itself may invoke costs and trade-offs affecting other traits (Metcalfe & Monaghan, 2001), for example in physiology and metabolism (Criscuolo, Monaghan, Nasir, & Metcalfe, 2008). Ultimately, behaviour can also be permanently influenced by the early environment. Developmental plasticity can thereby contribute to the emergence of stable individual differences in behaviour ('animal personality') that have their own downstream consequences for individual performance (Réale, Reader, Sol, McDougall, & Dingemanse, 2007).

Early nutritional conditions may match or mismatch nutritional conditions during later life due to environmental changes and/or dispersal to environments varying spatially or temporally. The predictive adaptive response (PAR) hypothesis predicts that developmental trajectories are adaptively optimized to exploit the predictability of environmental conditions (e.g. Gluckman, Hanson, & Spencer, 2005; Hanson & Gluckman, 2014). Unexpected changes, however, might then have pathological consequences, in extreme cases leading to poor performance under apparently favourable adult conditions (Monaghan, 2008). A notable example of an apparently failed predictive adaptive response is type II diabetes and the metabolic syndrome in humans, which have a particularly high prevalence in cohorts of people that have experienced poor early conditions followed by substantially improved conditions later in life (e.g. Fall, 2001; Ravelli et al., 1998). It has been hypothesized that organisms that were metabolically primed to poor nutritional conditions develop a 'thrifty phenotype' that only performs well if nutritional conditions remain poor, but becomes pathological if conditions are better than anticipated (Hales & Barker, 1992, 2001). An environmental mismatch between early and late environmental conditions therefore exposes the putative predictive adaptive response.

Unfortunately, long-term studies under experimentally matched and mismatched juvenile and adult environments are largely lacking for vertebrates (Bertram & Hanson, 2001). We hence experimentally addressed this issue by testing the effects of the early and late environment for individual phenotypes over more than 5 years in an avian model species, the zebra finch, *Taeniopygia guttata*. By experimental dietary manipulation, we created matches and mismatches between early and late environmental conditions with a switch at nutritional independence from parental provisioning. We manipulated early dietary conditions at the brood level and subsequently used a split-brood design when manipulating nutritional conditions for the entire life span after independence from parental care. We focused on a skeletal trait (tarsus length) that is largely determined during early life and is linked to fitness in the zebra finch (e.g. Bolund, Schielzeth, & Forstmeier, 2011), as well as on wing length, body weight and exploratory behaviour, which can respond plastically throughout life, to a certain extent, and which are all also fitness relevant (e.g. Carere, Drent, Koolhaas, & Groothuis, 2005; van Oers, Drent, de Jong, & van Noordwijk, 2004; Stamps & Groothuis, 2010; Tinbergen & Boerlijst, 1990). Our analysis is based on more than 5 years of data collection.

Zebra finches are distributed over most of the Australian continent across different climate zones (Zann, 1996), without evidence for significant genetic population structure (Balakrishnan & Edwards, 2009). In some areas, they are exposed to seasonal changes (Zann & Straw, 1984), while in others environmental changes are highly variable (e.g. Morton & Davies, 1983). Although both the frequency and amplitude of the variability in relation to individual life span is imperfectly known, we believe that zebra finches are a suitable study system, because they naturally live under highly variable environmental conditions (Immelmann, 1965; Zann, 1996). We used a captive population of zebra finches that is comparatively little affected by domestication and has, until recently, been indistinguishable from native wild zebra finches in Australia with a panel of microsatellites (Forstmeier, Segelbacher, Mueller, & Kempenaers, 2007). We hence expected less adaptation to ad libitum food conditions in aviaries than in most domesticated populations which have lived in captivity for many decades (Tschirren, Rutstein, Postma, Mariette, & Griffith, 2009).

Zebra finches have been used to study long-term effects of early rearing conditions, but so far studies have either addressed (mis) matches between the nestling and fledging phase (Criscuolo et al., 2008; Honarmand, Goymann, & Naguib, 2010; Krause, Honarmand, Wetzel, & Naguib, 2009), or between the juvenile phase (nestling and fledgling phase) and the second month of life (immature phase) (Kriengwatana et al., 2014). No study has yet upheld the nutritional manipulation throughout the entire lifetime and it is therefore impossible to address long-term effects of developmental (mis)priming. Our study aimed to experimentally fill this gap and test for the relative importance of early conditions and the magnitude of (mis)matching effects on morphology and behaviour. Specifically, we tested for effects on tarsus length, wing length, body weight and body condition, latency to feed and hopping activity in a special-environment and in special-object trials, weight loss under mild stress and life span.

We predicted that structural body size would be largely shaped by the early environment; hence individuals under good nutritional conditions should attain larger sizes. Wing length, however, may be more phenotypically flexible and individuals may be able to compensate for a bad start during subsequent moults. Thus, for wing length, it seems possible that effects of a favourable environment early or late in life are complementary but not additive (Scharf, Braf, Ifrach, Rosenstein, & Subach, 2015), giving rise to a case where only individuals under low early and low late nutrition grow shorter wings. We expected body weight and condition to be most flexible and most prone to a predictive adaptive response with individuals under matched conditions reaching highest condition. Furthermore, we tested the extent to which both the early and current environment, as well as their interaction, affect traits of an animal personality phenotype. We predicted individuals in poor conditions to take greater risks in a mildly stressful situation and to lose most weight when exposed to food deprivation. Finally, we expected, based on the predictive adaptive response hypothesis, that individuals in matched environments would be longest lived.

METHODS

Subjects and Housing

We used zebra finches of wild Australian origin that have been bred at Bielefeld University for about 10–11 generations (referred to as 'Bielefeld-AUS' in Forstmeier et al., 2007). Pairs were randomly formed in 2010, ensuring that partners were unrelated to at least the grandparent level, and were allowed to breed in cages (83 × 30 cm and 40 cm high) equipped with wooden nestboxes (15 × 15 cm and 15 cm high). Coconut fibres and hay were provided as nesting material. During pair formation and incubation, all pairs received a high-quality diet as described below.

First broods were alternately assigned to either early high-quality or early low-quality nutritional treatments (see below). Pairs were allowed to rear second broods that were raised under

the other nutritional regime, such that each pair could contribute offspring to both treatments of early dietary conditions. All surviving offspring participated in the study. Parental breeding attempts were separated by breaks of 2 weeks without nestboxes for recovery after the offspring of first broods reached nutritional independence. Nestboxes were monitored on a daily schedule between 0900 and 1100 hours to record nest initiation, fresh eggs and newly hatched chicks. Thus, new eggs were weighed within a maximum of 26 h after laying and were individually marked using a nontoxic pen. Newly hatched chicks were marked by trimming down feathers (Adam, Scharff, & Honarmand, 2014). Numbered yellow plastic rings were attached to all birds for life-long identification around day 10. Daily checks ensured that hatchlings could be unambiguously assigned to eggs, except when more than one chick hatched on the same day, in which case we assigned the heavier chick to the heavier egg (these subjects were excluded when testing for effects of egg size on hatchling size).

On the day all individuals of a brood reached day 35, which is the time when zebra finches become nutritionally independent from their parents (Zann, 1996), juveniles were separated from their parents and transferred to tutor groups of 8–10 young birds of mixed sex that were housed together with an unfamiliar and unrelated adult pair (in cages 81 × 48 cm and 60 cm high). Birds were removed from tutor groups at the age of 65 days. From day 65 until about day 400 of life, birds were kept in mixed-sex peer groups of three or four individuals (in cages 83 × 30 cm and 40 cm high). At about 400 days of age, individuals had the opportunity to breed for 90 days and to raise one successful brood with an unrelated partner that had experienced the same early and late nutritional treatments. Thereafter, birds were kept under nonbreeding conditions in single-sex groups of three or four individuals. Nutritional treatments were maintained during the entire life (see below). The analyses of long-term effects are based on subjects that survived at least until day 35 (147 individuals from 70 broods from 39 breeding pairs) and thus were exposed to both early and late nutritional treatments.

Nutritional Manipulation

Early nutritional manipulations started when the oldest chick of a brood reached 3 days of age and were upheld until a median brood age of 35 days. Broods received either of two early nutritional treatments: (1) a high-quality nutritional treatment ($high_{early}$), comprising standard seed food (a mixture of yellow millet, red millet, canary seed and yellow panicum) ad libitum supplemented daily by germinated seeds and egg food (Egg Food for Tropical Finches, CéDé, Evergern, Belgium) and three times per week by fresh greens (chickweed, *Stellaria media*) (Krause, Honarmand, & Naguib, 2011; Krause & Naguib, 2014); (2) low-quality nutritional treatment (low_{early}), comprising standard seed food ad libitum that was supplemented only once per week by germinated seeds and egg food (Krause et al., 2011; Krause & Naguib, 2014). Standard seed mix had an average protein content of 10.9%, whereas the average protein content of egg food was 16.2% and thus about 1.5 times higher (for further dietary contents, see Appendix 1, Table A1). Adult passerines usually need a protein content of 10–14% in their diet for maintenance, whereas growing chicks are assumed to need protein contents of 15–20% for normal growth (Harper & Skinner, 1998). Zebra finches can rear chicks on seed food alone (Zann, 1996), but they use germinated seeds and protein-rich food such as insects as supplements (Immelmann, 1969). First broods of parents were randomly assigned to one of the two early nutritional treatments while second broods were assigned to the opposite nutritional treatment.

From day 35 and for the rest of their lives, half of the individuals were assigned to a high-quality nutritional treatment ($high_{late}$) and the other half to a low-quality nutritional treatment (low_{late}), ensuring that half of each brood made the switch in the nutritional treatment (high to low or low to high), while the other half remained in their original nutritional treatment. The assignment of individuals to late treatments was done at random. Late high-quality nutritional conditions were identical to early high-quality nutritional conditions, while late low-quality nutritional conditions differed from early low-quality nutritional conditions in the omission of the weekly provision of germinated seeds and egg food.

Morphological Measures

Subjects were measured repeatedly for body size and body weight from hatching (day 0) up to the age of 2000 days. Body mass was measured using a Sartorius PT120 balance to the nearest 0.01 g, tarsus length was measured with a digital calliper (PMS 150 Meßschieber) to the nearest 0.01 mm and (flattened) wing length was measured using a wing ruler to the nearest 0.5 mm. Body mass was measured on day 0, 5, 10, 17, 35, 65, 100, 150, 200, 400, 800, 1200, 1600 and 2000 while tarsus and wing length were measured on all occasions from day 10 to day 800 and on day 1600. Measures were taken at an individual's exact age, except for measurements on day 5, 10, 17 and 35, which were taken when the median brood age reached the defined age. The exact age of all individuals was known from hatching dates and was fitted as a covariate in statistical analyses. When calculating body condition as the residuals of body weight regressed on tarsus length, we used current tarsus length for dependent birds (up to day 35) and average tarsus length after independence in order to eliminate measurement error in the denominator as far as possible.

Behavioural Trials

Three times during an individual's lifetime, subjects were exposed to: (1) a special-environment trial; (2) a special-object trial on 2 subsequent days. The first episode of behavioural trials occurred at about 100 days of age (average age ± SD: 117 ± 13 days), the second at about 200 days of age (189 ± 16 days) and the third at about 1600 days of age (1649 ± 61 days). The trials were intended as novel-environment and novel-object trials, but the situations were truly novel only at first trials. Since it remains unknown whether or not the birds remember the trial context later in life, we decided on the more neutral labelling. In any case, the substantial repeatability among trials (see Results) suggests that the behavioural responses were qualitatively similar. We measured the latency of birds to obtain food and how active they were in these trials outside their home cages.

Four hours before the special-environment trials started, birds were food deprived in their home cage to increase their motivation to feed and to measure the relative body mass loss (based on weights before and after trials; treatment effects for absolute body mass loss are additionally presented in the Appendix 1, Table A4) in association with a mild stressor (Krause et al., 2009). Immediately after this period, each bird was individually introduced into a special test cage, which was visually but not acoustically separated from the home cage and any other conspecifics. The special test cage was a small, clean and unfamiliar cage (41 × 30 cm and 30 cm high) with two perches at a height of 21 cm. It differed from the home cage in size, perch number/location and coloration of the floor and potentially in scent. On the floor, we placed a standard food dish with seed food. We video-recorded trials for 1 h and scored: (1) latency to feed: (2) general activity, as hops/min (hops from perch to perch and from perch to floor, from trial start to first feeding). Similar trials have previously been used as proxies for exploration in zebra finches (Krause & Naguib, 2011).

After 1 h of testing, the birds remained individually in the experimental cages and received their respective food treatment. The food dish used for the experiment also remained in the cage. All feeders were removed on the following day and subjects were food deprived for 4 h in the experimental cage. After this period, the special-object trials started and were video-recorded for 1 h. Again, a standard food dish with seeds was placed on the floor, but on top of the seeds we placed a blue AA battery as a special object that the birds never encountered except in the three trial situations. The same two behavioural parameters as in the special-environment trials were scored. After the special-object trial, body mass was measured to estimate body mass change over the entire test period (approximately 24 h) and each bird was put back in its respective home cage. We expected the special object to trigger neophobic behaviour.

Ethical Note

All birds remained at the Department of Animal Behaviour, Bielefeld University, Germany, for their entire life. The study was carried out according to the German laws for experimentation with animals and with permission of the LANUV NRW (no. 8.87–51.05.20.11.011). Breeding and housing of the birds were done with permission of the Veterinäramt Bielefeld (no. 530.421630–1, 18.4.2002 and no. 530.4, 27.07.2014). Birds in both low-quality and high-quality nutritional conditions had ad libitum access to food. In our early low-quality nutritional group, supply of germinated seeds and egg food once per week ensured that subjects survived without causing unethical suffering. All birds were monitored daily.

Statistical Analysis

We analysed the treatment effect for the morphological traits using mixed-effects models with Gaussian error distribution for all age classes separately. The distribution of residuals was inspected visually and did not show marked deviations from normality (except for the hopping activity in the special-environment trials that showed slightly right-skewed residuals even after log transformation). Models included sex as a fixed effect and brood identity as a random effect. Our design was approximately balanced with respect to treatment and treatment effects were coded as −0.5 (low-quality nutrition) and 0.5 (high-quality nutrition), so that the marginal effects of the early and the late nutritional treatment could be evaluated in the same model that we used to test for their interaction (Schielzeth, 2010). During the dependent life phase (day 5–35), individuals were measured at slightly different time points and all models for these ages therefore include the deviation from the nominal age as a covariate.

The separate analysis by age class might seem unusual at first compared to the alternative of analysing the pooled data using mixed models while controlling for the repeated measures as a random effect. Reasons for this decision are presented in more detail in the Appendix. To verify the robustness of our analysis, we also conducted the pooled analysis using mixed models that were different from the above models only by the addition of age class as a multilevel fixed factor and individual identity as a random effect. We did so separately for the early growth phase that was only exposed to the early treatment (all data up to day 35) and for postindependence life stages (all data day 65+). Results of this analysis are not qualitatively different and are presented in detail only in the Appendix (Table A3).

Brood size might influence growth and behaviour, and brood size at a median age of 5 days gives a good representation of brood size at the most competitive phase, because mortality was low after this age. We hence fitted brood size as a covariate, but this was never a significant term in the models and the inclusion or exclusion of brood size did not affect the significance of the treatment effect, so we decided to present only models without brood size. We present (marginal) treatment differences and their standard errors on the original scale of measurements and as percentage differences relative to the average trait value. Random effect variances are presented as the proportion of the phenotypic variance explained by a random effect after controlling for all fixed effects in the model (as specified above).

The repeatability R of behavioural data was analysed using mixed-effects models (Nakagawa & Schielzeth, 2010), controlling for the series of measurements (three levels, day 100, 200 and 1600), and sex as a fixed effect and individual identity as a random effect. We also fitted models that included nutritional treatments in the analysis and hence allowed the estimation of treatment-adjusted repeatabilities, but these repeatabilities differed by less than 0.02 in all cases, hence we do not report these results here. The treatment effects on behavioural phenotypes were estimated from mixed models controlling for series as a three-level (day 100, 200 and 1600) fixed effect and controlling for individual and brood identity in a pooled analysis across all three repeated measures.

Treatment effects on survival were estimated using Gompertz models of aging. Gompertz models fit two parameters to describe each survival curve and we used likelihood ratio tests (LRT) when testing for treatment differences on survival. This was done by fitting a full model with all parameters (two Gompertz parameters for each treatment) and models constraining the parameters of the two levels of a treatment to be equal (hence reducing the number of parameters by two per treatment). We then compared the relative likelihoods of the two models against a chi-square distribution with two degrees of freedom.

All analyses were done in R 3.3.1 (R Core Team, 2016) using the lme4 package for fitting mixed-effect models (Bates, Maechler, Bolker, & Walker, 2015), the lmerTest package for quantifying P values for fixed effects via Satterthwaite approximation of degrees of freedom (Kuznetsova, Brockhoff, & Christensen, 2016), the rptR package for estimating repeatabilities and their standard errors based on parametric bootstrapping (Stoffel, Nakagawa, & Schielzeth, 2017) and the flexsurv package for fitting proportional hazard models (Jackson, 2016). For morphological traits, including body weight, that were measured across many age classes, we present effect sizes for group means in figures along with statistical significance with respect to $\alpha = 0.05$, while the text of the corresponding results section highlights relevant maximum and/or minimum differences. The full statistics for all age classes are presented in the Appendix (Tables A2 and A4).

RESULTS

Treatment Effects on Wing and Tarsus Length

Wing length during the early growth phase was strongly influenced by the early nutritional treatment (Fig. 1a, Table A2), with nestlings under low-quality conditions having significantly shorter wings (e.g. 2.86 ± 0.73 mm shorter on day 17, a 6.0% difference; Table A2). By day 150, juvenile flight feathers are fully grown and the difference had reduced to 0.87 ± 0.37 mm (1.6% difference), but was still statistically significant (Table A2). There were no significant differences in wing length explained by the early treatment on day 200, 400 and 1600 measurements (Table A2), although there was a slight, statistically significant, difference on day 800 (a 1.3% difference; Fig. 1a, Table A2). There was no indication of any effect of the late nutritional treatment (after day 35), either as a main effect or in interaction with the early nutritional treatment (Fig. 1a, Table A2).

Peak tarsus length was reached near our day 17 measurements, declined slightly after fledging and stayed virtually constant after

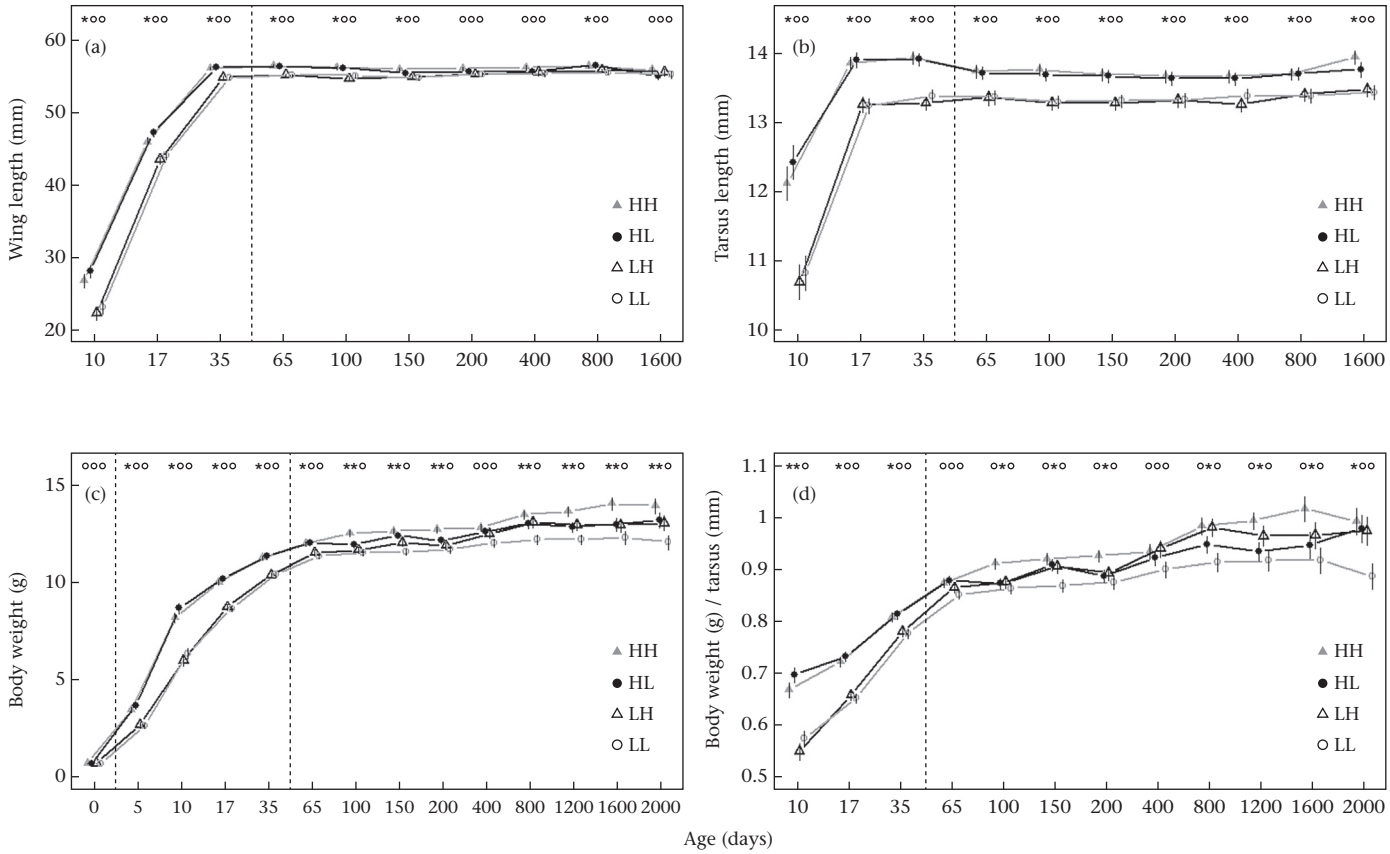

Figure 1. Age-dependent patterns of the nutritional treatment effects on morphological traits (LL = Low_{early}–Low_{late}, LH = Low_{early}–$High_{late}$, HL = $High_{early}$–Low_{late}, HH = $High_{early}$ $High_{late}$): (a) wing length, (b) tarsus length, (c) body weight and (d) condition. Means and SE are shown. The significance of the early treatment effect, the late treatment effect and their interaction (in this sequence) is shown at the top, where ° indicates $P > 0.05$ and $^*P \leq 0.05$. Vertical dashed lines mark the beginning (for body weight only) and end of the early nutritional manipulation. Note that we fitted the same model to data from all age classes for consistency, hence all significant late treatment and interaction effects before independence (one of 28 tests) evidently represent false positives.

the day 65 measurements (Fig. 1b). Before independence, tarsus length was significantly influenced by the early treatment: nestlings kept under low-quality conditions had shorter tarsi with a maximum difference of 0.61 ± 0.12 mm on day 17 (a 4.5% difference; Table A2). By day 100, the difference had reduced to 0.43 ± 0.10 mm (a 3.2% difference; Fig. 1b, Table A2). There was no indication of any effect of the late nutritional treatment, either as a main effect or in interaction with the early nutritional treatment (Fig. 1b, Table A2).

Treatment Effects on Body Weight and Condition

Hatchling weight on day 0 was heavily influenced by egg mass ($b = 0.60 \pm 0.10$, $t_{92.1} = 5.79$, $P < 0.0001$), but the weight of chicks on day 5 was no longer significantly influenced by egg mass ($b = 1.21 \pm 0.92$, $t_{101.3} = 1.32$, $P = 0.19$). The early treatment showed a significant effect already on day 5 (0.87 ± 0.20 g, a 28.0% difference) and throughout the period of nonindependence (Fig. 1c, Table A2). The difference between treatment groups was most pronounced on day 10, when hatchlings from low-quality nutritional conditions were 2.17 ± 0.35 g lighter (a 29.8% difference; Table A2). By the end of the dependent period (day 35), the difference had reduced to 0.90 ± 0.18 g (an 8.3% difference; Table A2), indicating that individuals under low-quality conditions had undergone partial catch-up growth. The early treatment had a significant effect on body weight throughout adult life (except for the day 400 measurements that were taken during the breeding period and were putatively affected by differences in breeding status; Table A2), with a difference of 1.04 ± 0.45 g (8.0% difference) detectable even on day 2000 (Table A2). The effect of the late treatment was detectable only from day 100 onwards (0.35 ± 0.15 g, a 2.8% difference; Table A2) and remained significant throughout life (e.g. day 2000: 0.80 ± 0.32 g, a 6.2% difference; Fig. 1c, Table A2), with birds from the late high-quality treatment being heavier. At no age class was there any indication of a significant interaction of the early and the late nutritional treatment (i.e. of environmental matching; Fig. 1c, Table A2).

Condition, here quantified as the residuals of body weight regressed against tarsus length, was lower in dependent young of the early low-quality nutritional treatment (Table A2), but after day 100, only the late nutritional treatment had an effect on condition, with individuals from the high-quality nutritional treatment showing higher weights for their sizes (Fig. 1d, Table A2). There was no effect of the early treatment on adult phenotypes indicating that while low-quality treatment individuals were smaller and lighter (see above), they were not lighter for their sizes. A notable exception was condition on day 2000, where high-quality early treatment individuals were, on average, of better condition than low-quality early treatment individuals (Fig. 1d, Table A2). There was no indication of any significant early and late treatment interactions (Fig. 1d, Table A2).

Brood Identity Effects Beyond the Treatment

We inspected the variance explained by brood identity after controlling for the nutritional treatments. This variance component

captures random variation between broods independent of the experimental treatments. All traits showed similar trends with substantial brood identity components during the nestling phase that gradually declined with age (Fig. 2a and b). However, the trend reversed for body weight after day 1200 with high brood identity effects on day 1600 and day 2000 (Fig. 2a).

Treatment Effects on Behavioural Responses and Body Mass Management

We quantified the latency to feed and the number of hops/min (all log transformed to better approach normality of the residuals) in a special-environment and a special-object trial each around day 100, 200 and 1600. All behavioural measurements were significantly repeatable across the first 100 days and only slightly less so across the entire 4-year period (Table 1). Latency to feed as well as hopping activity were correlated between the special-environment and the special-object trials (Table 2).

Latency to feed was associated with the late nutritional treatment in the special-object trials: individuals kept in the high-quality treatment showed longer latencies to feed (Fig. 3c, Table A4). However, latency to feed was not associated with the late nutritional treatment in the special-environment trials, where the sign of the estimated effect was even reversed (Fig. 3a, Table A4), suggesting strong context dependence (with $P = 0.003$ for the null hypothesis of equal effect sizes).

Hopping activity in the special-environment trials was affected by the interaction between early and late nutritional treatments (Fig. 3b). Birds from mismatching conditions showed an increased hopping rate compared to birds where early and late nutritional conditions matched (interaction early∗late treatment, Table A4). Hopping activity in special-object trials was not affected by the interaction of early and late treatment (Table A4) but only by the late treatment, with birds that had experienced high-quality conditions performing fewer hops than those kept under low-quality conditions (Fig. 3d, Table A4).

We quantified relative body mass loss after 4 h of food deprivation and across the 24 h of the two experiments. This relative body mass loss was repeatable both in the short term (a 100-day period) across the first two sessions (4 h: $R = 0.24 \pm 0.08$, $\chi^2_1 = 7.68$, $P = 0.006$; 24 h: $R = 0.30 \pm 0.08$, $\chi^2_1 = 12.27$, $P = 0.001$) and in the long term across the entire 4-year period (4 h: $R = 0.26 \pm 0.06$, $\chi^2_1 = 16.69$, $P < 0.0001$; 24 h: $R = 0.18 \pm 0.06$, $\chi^2_1 = 7.84$, $P = 0.005$).

The early treatment was linked to greater relative body mass loss during 4 h of food deprivation, with individuals of the high-quality treatment losing more weight relative to their body mass (Table A4), while the reverse was true for the late treatment where birds kept under low-quality conditions lost more relative body mass than those under high-quality food conditions (Fig. 4a, Table A4). There were no significant treatment effects on body mass loss over the 1-day trial period (Fig. 4b, Table A4).

Treatment Effects on Survival

By the time of the survival analysis cutoff date (31 July 2016), 98 of 147 individuals (67%) had died and those individuals still alive

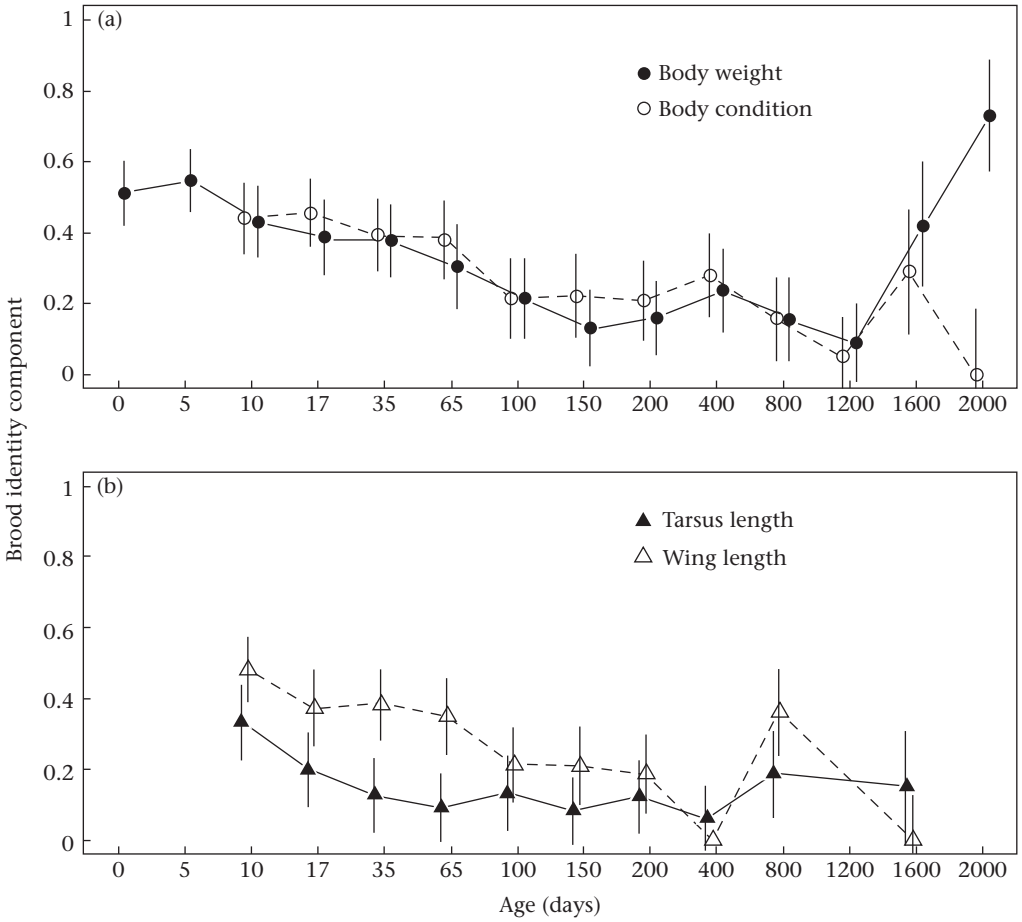

Figure 2. Age-dependent changes in the brood identity variance component relative to the total phenotypic variance (after accounting for the fixed treatment effects) for (a) body weight and body condition and (b) tarsus length and wing length. Means and SE are shown.

Table 1
Behavioural repeatabilities in special-environment and special-object trials over short-term (3-month) and long-term (4-year) periods

Trial type	Trait	Individual (total)				Brood identity subcomponent			
		Short term		Long term		Short term		Long term	
		R±SE	P	R±SE	P	R±SE	P	R±SE	P
Special environment	Latency feeding	0.44±0.07	$<10^{-6}$	0.36±0.06	$<10^{-8}$	0.08±0.06	0.13	0.05±0.06	0.21
	Hops	0.38±0.07	$<10^{-3}$	0.32±0.06	$<10^{-4}$	0.11±0.07	0.062	0.09±0.06	0.089
Special object	Latency feeding	0.52±0.06	$<10^{-9}$	0.50±0.06	$<10^{-15}$	0.08±0.06	0.13	0.05±0.06	0.21
	Hops	0.38±0.06	$<10^{-3}$	0.39±0.06	$<10^{-9}$	0.09±0.07	0.1	0.06±0.06	0.17

Table 2
Phenotypic cross-context correlations in special-environment and special-object trials at three different ages of testing

Age (days)	Trait	N	r	t	P
100	Latency feeding	133	0.28	3.37	$<10^{-3}$
	Hops	133	0.32	3.48	$<10^{-3}$
200	Latency feeding	132	0.32	3.85	$<10^{-3}$
	Hops	132	0.43	5.48	$<10^{-6}$
1600	Latency feeding	80	0.59	6.42	$<10^{-8}$
	Hops	80	0.53	5.56	$<10^{-6}$

varied in age between 1976 and 2232 days. Fourteen individuals (three $high_{early}$–$high_{late}$, two $high_{early}$–low_{late}, four low_{early}–$high_{late}$ and five low_{early}–low_{late}) died at an age of 35–62 days, while the next individuals only died at an age of 171, 283 and 284 days, respectively. This early postindependence mortality was not significantly affected by treatment (chi-square test of equal shares among treatments: $\chi^2_1 = 1.14$, $P = 0.29$; Fig. 5). The fit of survival models was impaired when including this early postindependence mortality, because a simple survival model cannot capture the transitionary high mortality during early life. We therefore excluded early postindependence mortality from the main survival analysis of adults and analysed survival during the youth phase (before day 100, including preindependence mortality) separately. There was no significant effect of environmental matching on adult mortality (LRT: $\chi^2_2 = 0.04$, $P = 0.98$) and no effect of the late, i.e. current, nutritional treatments ($\chi^2_2 = 2.62$, $P = 0.26$), but there was a significant effect of the early nutritional treatment on adult survival, with individuals from the low-quality treatment outliving those from the high-quality treatment ($\chi^2_2 = 6.72$, $P = 0.035$; Fig. 5). Survival during the youth phase (before day 100, thus including nestling, postfledging and early postindependence mortality) was not significantly affected by the early nutritional treatment (LRT: $\chi^2_2 = 0.84$, $P = 0.66$; Fig. 5).

DISCUSSION

Life histories of individuals are crucially affected by the conditions they experience during their early development (Lindström, 1999; Mainwaring & Hartley, 2012), but also by the current conditions they experience. A key question is whether early environmental conditions shape the adult phenotype as an adaptive response to the expected environmental conditions (predictive adaptive response: Gluckman et al., 2005; Hales & Barker, 1992; Hanson & Gluckman, 2014). Here, we addressed the question of what happens when environmental conditions change unexpectedly between early ontogeny and adult life in zebra finches, by life-long manipulation of nutritional conditions that were either matched or mismatched with their early nutritional conditions. Our results provide no support for the predictive adaptive response hypothesis, as we did not find any robust interactive effects between early and late nutritional treatments. What we found instead are substantial life-long effects of early nutritional treatments on the adult structural size. Tarsus length, a skeletal trait that is fixed after determinate growth in zebra finches, and, to a lesser degree, wing length were affected by only the early treatment, while phenotypically flexible traits such as body mass were additively affected by both the early and late nutritional treatments, again with no evidence of interactive effects.

The clear lack of early adaptive programming in our study is supported by independent evidence from other zebra finch studies. One line of reasoning behind the predictive adaptive response hypothesis (Hanson & Gluckman, 2014) is that early nutrition primes the metabolism and prepares it for similar conditions in the future. There is previous evidence that metabolic rates are affected directly or indirectly (e.g. via costs from compensatory growth) by poor early conditions in zebra finches (Criscuolo et al., 2008; Verhulst, Holveck, & Riebel, 2006). However, zebra finches raised under early dietary stress show no adaptive responses when such stress reappears in adulthood (Krause et al., 2009), illustrating that the overall evidence is at least ambiguous in zebra finches. Although we have not directly examined metabolic changes, we did not detect any exhaustive fattening nor any specific response in relative body mass loss (Krause et al., 2009), but it may be possible that there are subtler changes affecting other traits.

Results on humans suggest that the period of gestation is the critical period for nutritional priming (e.g. Fall, 2001; Ravelli et al., 1998). Prenatal priming is also possible in birds (Adkins-Regan, Banerjee, Correa, & Schweitzer, 2013; Griffith & Buchanan, 2010; Groothuis & Schwabl, 2008; Saino, Dall'Ara, Martinelli, & Moller, 2002), but this was not affected by our postnatal manipulation. Our treatment started when the oldest chick in the nest was 3 days old, hence for many individuals (40.8%) even less than 3 days after hatching. Similar manipulation of only early nutritional conditions has been successfully used in several other studies to affect developmental trajectories (e.g. Birkhead, Fletcher, & Pellatt, 1999; Boag, 1987; Honarmand et al., 2010; Krause & Naguib, 2011, 2015). We upheld our early nutritional treatment until day 35, which covers the entire nestling and fledgling phase in which zebra finches are nutritionally dependent on their parents (Zann, 1996). This should offer sufficient time for efficient priming if early postnatal programming happens in zebra finches, although it is evidently possible that the most important priming phase is prenatal (Griffith & Buchanan, 2010), or that it requires stronger manipulation of nutrition (that we did not apply for ethical reasons). With these limitations in mind, we found no evidence of interactive effects in the predicted direction of better performance in matched environments, as would have been expected under the predictive adaptive response hypothesis (Hanson & Gluckman, 2014).

At the behavioural level, we found high long-term repeatability in behavioural traits (latency to feed in special-environment and special-object trials), which were also correlated across contexts (see also Wuerz & Krüger, 2015). We interpret the latency to feed as a measure of explorative behaviour (Krause & Naguib, 2011) and/or neophobia, and hopping activity as a combination of overall activity with arousal in a mildly stressful situation. We see the special-

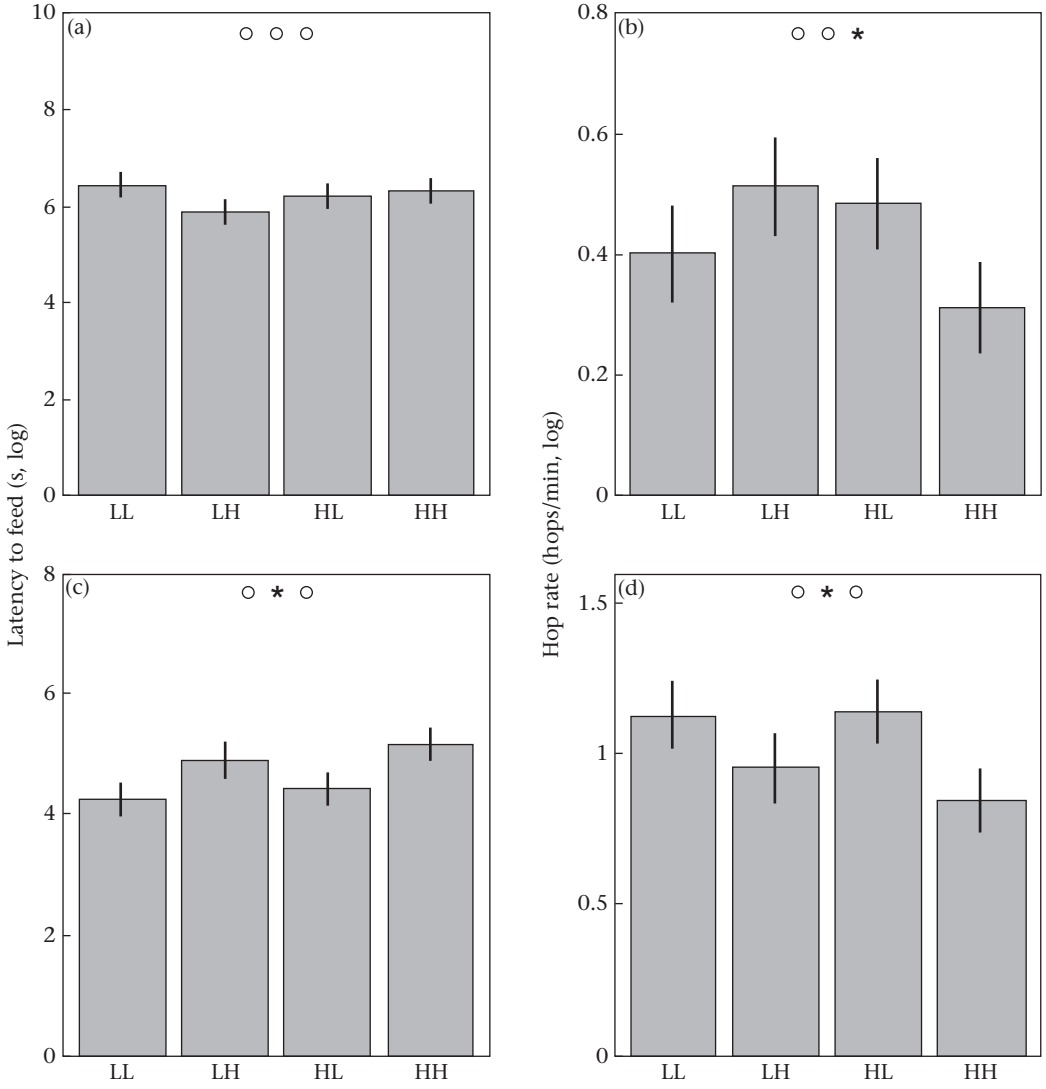

Figure 3. Nutritional treatment effects (LL = Low_{early}–Low_{late}, LH = Low_{early}–$High_{late}$, HL = $High_{early}$–Low_{late}, HH = $High_{early}$–$High_{late}$) on behavioural phenotypes: (a, b) latency to feed and hop rate in the special-environment trials, and (c, d) latency to feed and hop rate in the special-object trials. Means and SE are shown. The significance of the early treatment effect, the late treatment effect and their interaction (in this sequence) is shown at the top, where ° indicates $P > 0.05$ and $^*P \leq 0.05$. Note that the Y axes depict a log-scale.

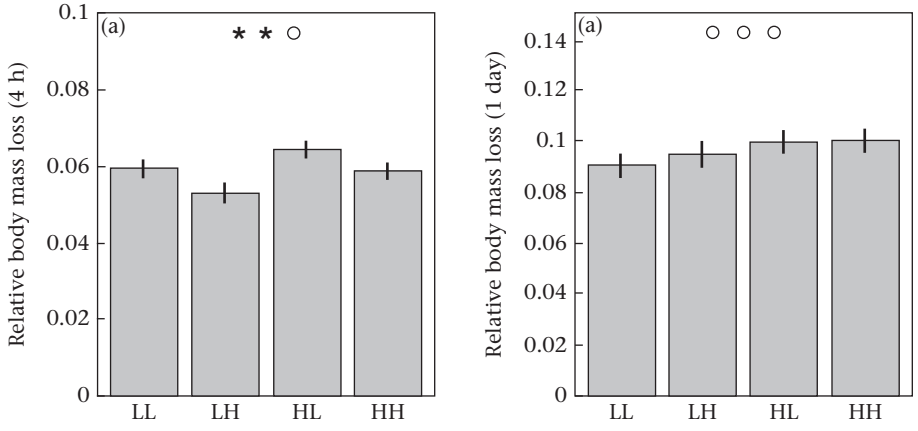

Figure 4. Nutritional treatment effects (LL = Low_{early}–Low_{late}, LH = Low_{early}–$High_{late}$, HL = $High_{early}$–Low_{late}, HH = $High_{early}$–$High_{late}$) on relative body mass loss after (a) 4 h of food deprivation and (b) 1 day. Means and SE are shown. The significance of the early treatment effect, the late treatment effect and their interaction (in this sequence) is shown at the top, where ° indicates $P > 0.05$ and $^*P \leq 0.05$.

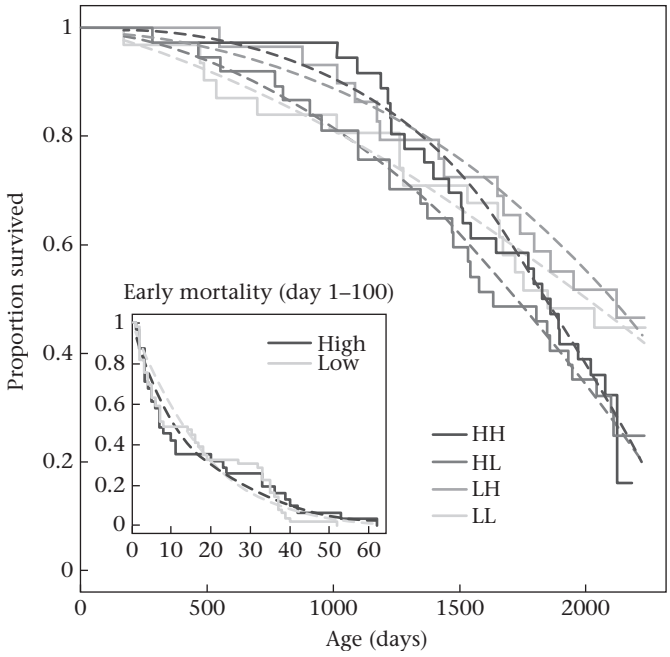

Figure 5. Nutritional treatment effects on survival. Kaplan–Meier survival estimates are shown by solid lines and fitted Gompertz survival curves are shown by dashed lines. Short horizontal lines indicate censored data for individuals that were still alive. The main plot only considers individuals that survived at least 100 days, since early mortality (day 35–100) was poorly modelled by Gompertz survival curves. Early mortality (day 1–100) is shown as an insert with separate curves for the two early nutritional conditions.

environment and special-objects trials as different contexts for testing a similar underlying personality-related axis, even though they may also be considered as different traits with significant cross-trait correlations. We found that responses to special-environment and special-object trials were highly repeatable, even over several years, and were correlated across contexts. Such traits, linked to a certain extent to exploration (Krause & Naguib, 2011), are often considered as proxies for animal personality and an intensely studied research question is how such consistent interindividual differences arise (Réale et al., 2007).

Latency to feed was affected only by the current (late) treatment (and this only in the special-object context), and since there were no significant brood identity effects, there do not seem to be substantial shared early environmental effects on this trait. Hopping activity, however, showed a significant brood identity component, at least in the special-environment context, and a significant effect of environmental matching in the same context. This was the only indication of any treatment interactions, with individuals from matched treatments being less active than those from mismatched treatments. This might represent a genuine, context-specific effect, but since there was no equivalent trend in the special-object context, we believe that the results should not be over-interpreted.

Overall, the lasting environmental effects on the behavioural phenotypes are certainly small in our study, but interestingly the influence of current nutritional conditions was also small. Small early-rearing effects, but sizable heritable variation, was reported for novel-object trials in another zebra finch population (Schielzeth, Bolund, Kempenaers, & Forstmeier, 2011). Other studies found direct and indirect effects of early life conditions on exploration behaviour, personality and cognition (Brust, Krüger, Naguib, & Krause, 2014; Brust, Würz & Krüger, 2013; Fisher, Nager, & Monaghan, 2006; Krause et al., 2009; Mainwaring & Hartley, 2013). Zebra finches experiencing nutritional stress as nestlings have been found to be faster in exploring to obtain food as adults in a test aviary (Krause et al., 2009). In addition, the compensatory growth for early deficits, but not the early treatment itself, has been shown to affect exploratory behaviour in zebra finches (Krause & Naguib, 2011, 2015). Relative body mass loss was affected by both treatments. Notably, the direction of an early nutritional treatment effect was previously reported in the opposite direction, i.e. that birds from early good conditions lost less relative body mass (Krause et al., 2009). The late treatment effect occurred, however, in the expected direction.

The late treatment, i.e. the current treatment at the time of mortality, had no significant effect on survival rates, either as a main effect or in interaction with the early treatment. However, there was a significant effect for individuals raised under low-quality nutritional conditions to outlive individuals from early high-quality nutritional conditions, an effect that became apparent only late in life. Previous studies have reported reduced survival of domesticated male zebra finches as a consequence of early poor conditions (Birkhead et al., 1999), even when following the respective cohorts for only 500 days. At this time, about 20% of the males from early good conditions died, compared with 50% of the males from early poor conditions. Thus, the average survival to age 500 days for all birds was around 35% (Birkhead et al., 1999), which is comparable to the total mortality in our population after more than twice the time (1200 days). It is remarkable that the early treatment effects on survival became apparent only very late in life, a period that is not covered by most studies. This early treatment effect is slightly unexpected. One could have expected, if the early treatment matters, that early good conditions would have a positive effect on survival, i.e. the so-called silver-spoon effect (Grafen, 1988; Monaghan, 2008), but we found the opposite. However, for morphological phenotypic traits, such beneficial effects of early good conditions (Grafen, 1988; Monaghan, 2008) are strongly supported by our results. One might speculate that beneficial effects of good early nutrition for physiology and behaviour (as known from other studies, e.g. Krause et al., 2009; Kriengwatana, Farrell, Aitken, Garcia, & MacDougall-Shackleton, 2015) trade off with reduced survival as predicted by the pace-of-life hypothesis (e.g. Réale, Garant, Humphries, Bergeron, Careau & Montioglio, 2010; Ricklefs & Wikelski, 2002).

Beside the initial experimental question, we want to further highlight two auxiliary results of our study that are of interest in the context of animal personality research and for uncontrolled shared early environmental effects. First, there has been a lot of interest in the stability of interindividual differences over different timescales (e.g. Araya-Ajoy, Mathot, & Dingemanse, 2015; Boulton, Grimmer, Rosenthal, Walling, & Wilson, 2014; Schielzeth et al., 2011; Wexler, Subach, Pruitt, & Scharf, 2016). We found that the decay of repeatability over time was remarkably mild when moving from a 3-month period to an almost 4-year period. This illustrates that, at least in some cases, short-term repeatability estimates can be predictive for individual consistencies over most of the expected life span.

The second point refers to the slight, but steady, decline of the brood identity random effect variance component. In quantitative genetics, clutch or brood identity effects are used to model shared environmental effects. Shared environmental effects, by definition, affect members of the same group in similar ways and thereby produce differences between groups. The brood identity effect in our study covers all effects beyond the experimental treatment (which was statistically controlled for) that make individuals from different families differ from each other. This includes all maternal effects and early rearing conditions that were not explicitly manipulated, but possibly also genetic differences between families. It is remarkable that family identity effects on body weight

and morphology are detectable even when birds were measured at an age of 4–5 years, but also that the effect tended to decline when individuals grew older. Relatively few animal studies have explicitly reported the temporal decline in the variance component (but see Réale, Festa-Bianchet, & Jorgenson, 1999; Wilson, Kruuk, & Coltman, 2005) and we hope that our finding stimulates some research on this important topic and the routine reporting of random effect variance components (Schielzeth & Nakagawa, 2013).

DATA ACCESSIBILITY STATEMENT

The data set is available online in the Dryad Digital Repository, http://dx.doi.org/10.5061/dryad.6j700. (Krause, E.T., Krüger, O., Schielzeth, H. 2017. Data from: Long-term effects of early nutrition and environmental matching on developmental and personality traits in zebra finches.)

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APPENDIX. AGE-CLASS SPECIFIC VERSUS POOLED ANALYSIS

In the main text, we present the analysis of morphological traits separated by age class. This may seem unusual compared with the alternative of pooling these data and analysing them in a single mixed model controlling for age class as a categorical fixed effect and for repeated measures by a random effect of individual identity. We think that our approach offers more robustness and conveys more information than the alternative. We explain our reasoning here.

The advantage of the pooled analysis is clearly that it reduces the analysis to single hypothesis tests for each of the three core research hypotheses (the main effects of the early and the late nutritional treatment and their interaction). This comes with the drawback, however, that we implicitly have to assume that the treatment effect is constant across the range of age classes that are pooled, an assumption that may well be violated. For example, if we pool all postindependence data (day 65 to day 2000), we implicitly assume that the early treatment is equally influential across the entire range. However, it is not unlikely that the influence decays with time. If the early treatment influences only ages up to day 200, for example, then pooling all data will probably result in (unnecessary) false negative outcomes. Similarly, the late treatment may take some time to take effect (e.g. only after moult into the adult plumage) and pooling data from too-young ages may cause false negative results, too. The advantage of reducing the analysis to single hypothesis tests comes at the cost of susceptibility to the details of pooling.

The age class-specific analysis allows the identification of even unexpected trends in effect sizes, since effects are estimated independently and the age patterns only emerge from the view of the results across age classes (Fig. 1). Notably, if we had an idea of how effect sizes change across age, it would be possible to model these effects in a mixed model, but the age class-specific analysis allows the identification of new patterns. Three concerns may be brought forward against this analysis. One is that the data are not independent, the second is that multiple tests of the same hypothesis are applied and the third is that the test results are not independent. The latter two are valid concerns, while the first is not a real issue, at least not as far as nonindependence of data within age class is concerned. Each separate model for each age class uses perfectly independent data at the level of the individual, because no individual contributes more than a single data point.

Multiple testing and nonindependence of test results are potentially real problems. We certainly warn against splitting a single hypothesis like the one about late treatment effects on body weight into nine age class-specific hypotheses and claim a single significant effect as evidence for a biologically relevant effect (as a single significant outcome is more likely to be a type I error). It is also difficult to control for nine independent tests by Bonferroni correction. Bonferroni and other multiple testing corrections were developed for independent tests and applying them to related tests will result in too stringent thresholds. We instead suggest considering multiple tests as partially replicated results and looking for runs of significant tests in equal directions in adjacent age classes. The results are indeed independent with respect to measurement error, even though they are not independent with respect to assignment of individuals to treatment. Random biases in the assignment, however, affect the pooled analysis as well.

An additional issue is the implicit assumption of equal residual variance across age classes. This can be problematic in cases where trait means change substantially during development as in our data. For example, the early treatment effect on wing length at ages 10–35 days is undeniably strong and clearly visible in Fig. 1, yet in the pooled analysis it is not significant when the raw data are analysed (Table A3). The effect is recovered when the data are log transformed ($b = 0.21 \pm 0.067$, $t_{574.0} = 3.22$, $P = 0.001$). Log transformation is an obvious solution in this case, but the point is that we need to worry about (un)equal residual variances in the pooled analysis, while this is clearly less of an issue in the age class-specific analysis.

Finally, why did we analyse the morphological data separately by age class, but pool the behavioural data? The reason is the expected amount of measurement error. Morphological traits are typically measured with little error. Age class-specific analyses thus have decent power to detect real effects. Behavioural scores, however, typically contain a large proportion of measurement error and the power for the test thus comes from pooling data across multiple measures of the same trait. The traits are hence different and therefore deserve different analyses.

Table A1
Dietary contents of the three food sources used for the nutritional treatments

	Crude protein (%)	Crude fat (%)	Crude fibre (%)	Moisture (%)	Crude ash (%)
Standard seed mix	10.9	4.13	9.15	9.41	2.98
Germinated seeds	8.20	3.05	6.60	31.88	2.03
Egg food	16.2	5.00	4.20	11.00	5.00

Standard seed mix that consists of a seed mixture of seeds of yellow millet, red millet, canary seed and yellow panicum. Germinated seeds are standard seeds that were germinated for at least 24 h and the egg food was a commercial mixture (Egg Food for Tropical Finches, CéDé, Evergern, Belgium) that consists, among other things, of eggs, egg derivatives, vegetable protein, insects, fruits, oils, etc.

Table A2
The full statistics for all age classes for morphological traits

Trait	Age (days)	Early nutritional treatment				Late nutritional treatment				Early∗late interaction			
		b	*t*	*df*	*P*	*b*	*t*	*df*	*P*	*b*	*t*	*df*	*P*
Wing	10	4.71±1.17	4.03	60.7	<0.001	−1.05±0.72	−1.45	82.7	0.15	−0.44±1.46	−0.30	82.7	0.77
	17	2.86±0.73	3.89	59.4	<0.001	−0.92±0.48	−1.92	83.2	0.058	−0.73±0.96	−0.76	83.1	0.45
	35	1.33±0.30	4.41	66.2	<0.001	−0.03±0.20	−0.15	90.3	0.88	−0.18±0.41	−0.44	90.4	0.66
	65	1.24±0.30	4.14	69.1	<0.001	0.00±0.20	0.02	89.1	0.98	0.07±0.40	0.16	88.8	0.87
	100	1.32±0.36	3.69	71.1	<0.001	−0.18±0.28	−0.63	96.7	0.53	0.52±0.56	0.92	96.6	0.36
	150	0.87±0.37	2.33	62.3	0.023	0.31±0.30	1.06	90.7	0.29	0.43±0.60	0.72	90.7	0.47
	200	0.54±0.31	1.72	62.6	0.091	0.22±0.26	0.84	93.6	0.41	0.52±0.52	0.99	93.6	0.32
	400	0.46±0.44	1.05	125.0	0.30	0.34±0.44	0.77	125.0	0.44	0.39±0.89	0.45	125.0	0.66
	800	0.75±0.32	2.35	61.2	0.022	0.07±0.23	0.28	80.2	0.78	−0.46±0.47	−0.97	80.8	0.34
	1600	0.05±0.52	0.09	78.0	0.93	0.60±0.52	1.14	78.0	0.26	0.50±1.05	0.48	78.0	0.63
Tarsus	10	1.50±0.29	5.12	56.7	<0.001	−0.22±0.21	−1.07	84.1	0.29	−0.17±0.42	−0.40	84.4	0.69
	17	0.63±0.13	5.00	58.9	<0.001	−0.02±0.10	−0.16	90.0	0.87	−0.07±0.19	−0.38	90.4	0.71
	35	0.59±0.10	5.74	64.3	<0.001	−0.04±0.09	−0.50	98.6	0.62	0.12±0.17	0.73	99.4	0.47
	65	0.36±0.11	3.31	63.2	0.002	0.01±0.10	0.07	96.7	0.94	0.03±0.20	0.15	96.8	0.88
	100	0.43±0.10	4.16	64.8	<0.001	0.03±0.09	0.30	95.2	0.76	0.08±0.18	0.47	95.3	0.64
	150	0.39±0.10	3.75	65.8	<0.001	0.00±0.09	−0.03	99.3	0.98	0.06±0.19	0.31	99.5	0.76
	200	0.33±0.11	3.01	66.7	0.004	0.02±0.10	0.17	99.3	0.86	0.04±0.19	0.20	99.4	0.84
	400	0.33±0.10	3.10	65.6	0.003	−0.05±0.10	−0.48	100.8	0.63	0.16±0.20	0.81	101.0	0.42
	800	0.31±0.11	2.88	57.9	0.006	0.01±0.09	0.09	87.4	0.93	−0.02±0.19	−0.13	88.7	0.90
	1600	0.40±0.11	3.51	46.8	0.001	0.11±0.10	1.06	60.8	0.29	0.13±0.21	0.63	60.7	0.53
Body weight	0	0.00±0.03	0.04	69.4	0.97	0.02±0.02	1.02	90.1	0.31	−0.02±0.03	−0.71	89.8	0.48
	5	0.90±0.20	4.46	64.6	<0.001	−0.09±0.11	−0.82	83.5	0.41	−0.24±0.23	−1.06	83.4	0.29
	10	2.24±0.35	6.41	59.5	<0.001	−0.42±0.21	−1.99	80.6	0.050	−0.14±0.43	−0.33	80.5	0.74
	17	1.40±0.20	7.13	65.2	<0.001	−0.05±0.12	−0.44	87.6	0.66	−0.25±0.25	−0.99	87.5	0.33
	35	0.91±0.19	4.90	61.0	<0.001	−0.04±0.12	−0.34	84.0	0.73	−0.05±0.24	−0.22	84.0	0.82
	65	0.56±0.17	3.24	61.6	0.002	0.06±0.13	0.52	85.7	0.61	−0.20±0.25	−0.78	85.4	0.44
	100	0.64±0.19	3.32	59.5	0.002	0.36±0.15	2.37	87.3	0.020	0.45±0.30	1.50	87.2	0.14
	150	0.72±0.19	3.85	51.3	<0.001	0.35±0.17	2.12	87.8	0.037	−0.23±0.33	−0.69	88.1	0.49
	200	0.64±0.20	3.12	53.8	0.003	0.39±0.18	2.16	89.0	0.033	0.39±0.36	1.09	89.1	0.28
	400	0.46±0.25	1.88	51.6	0.066	0.35±0.20	1.72	82.0	0.089	−0.27±0.40	−0.66	81.9	0.51
	800	0.62±0.27	2.28	51.4	0.027	0.67±0.24	2.76	83.9	0.007	−0.42±0.49	−0.86	85.5	0.39
	1200	0.67±0.28	2.39	37.5	0.022	0.77±0.27	2.89	70.2	0.005	0.08±0.54	0.14	70.2	0.89
	1600	0.90±0.40	2.26	36.5	0.030	0.86±0.30	2.89	32.5	0.007	0.41±0.60	0.68	32.1	0.50
	2000	1.01±0.43	2.35	26.6	0.027	0.85±0.34	2.51	14.9	0.024	−0.28±0.69	−0.41	14.7	0.69
Condition	10	0.12±0.02	6.11	63.3	<0.001	−0.03±0.01	−2.36	82.7	0.020	0.00±0.02	−0.18	82.6	0.86
	17	0.07±0.01	6.05	66.1	<0.001	0.00±0.01	−0.30	86.8	0.76	−0.02±0.01	−1.07	86.6	0.29
	35	0.03±0.01	2.73	59.5	0.008	0.00±0.01	−0.10	83.3	0.92	−0.01±0.02	−0.64	83.3	0.53
	65	0.02±0.01	1.66	57.7	0.10	0.00±0.01	0.48	79.3	0.63	−0.02±0.01	−1.22	79.0	0.23
	100	0.02±0.01	1.85	54.7	0.070	0.03±0.01	2.55	84.0	0.013	0.03±0.02	1.39	84.0	0.17
	150	0.03±0.01	1.96	56.4	0.055	0.02±0.01	2.24	85.3	0.028	−0.03±0.02	−1.15	85.3	0.25
	200	0.02±0.01	1.58	54.9	0.12	0.03±0.01	2.40	85.4	0.018	0.02±0.02	0.91	85.3	0.37
	400	0.01±0.02	0.51	51.8	0.61	0.03±0.01	1.88	79.2	0.063	−0.03±0.03	−1.02	79.0	0.31
	800	0.02±0.02	0.99	49.8	0.33	0.05±0.02	3.07	83.0	0.003	−0.03±0.03	−0.95	84.6	0.34
	1200	0.02±0.02	1.24	38.2	0.22	0.05±0.02	2.96	71.9	0.004	0.01±0.04	0.33	71.9	0.74
	1600	0.04±0.03	1.41	37.8	0.17	0.06±0.02	2.63	39.7	0.012	0.02±0.05	0.48	39.4	0.64
	2000	0.05±0.03	2.12	51.0	0.039	0.05±0.03	1.93	51.0	0.059	−0.07±0.05	−1.41	51.0	0.17

Estimates ± SEs, test statistics, degrees of freedom (based on Satterthwaite approximation) and *P* values for treatment effects on morphological traits are provided.

Table A3
The full statistics for the pooled analysis for the morphological traits

Trait	Age (days)	Early nutritional treatment				Late nutritional treatment				Early*late interaction			
		b	*t*	*df*	*P*	*b*	*t*	*df*	*P*	*b*	*t*	*df*	*P*
Wing	10–35	2.87±1.84	1.56	429.0	0.11	−0.87±1.76	−0.49	429.0	0.62	−0.31±2.59	−0.12	429.0	0.90
	65–1600	0.92±0.37	2.51	111.81	0.013	0.28±0.30	0.92	88.76	0.36	0.18±0.45	0.41	93.4	0.69
Tarsus	10–35	0.91±0.20	4.58	154.8	<0.001	−0.11±0.19	−0.57	421.7	0.57	−0.01±0.27	−0.04	428.3	0.97
	65–1600	0.40±0.14	2.91	115.6	0.004	0.04±0.12	0.41	94.4	0.68	0.07±0.18	0.39	98.8	0.70
Body weight	5–35	1.36±0.38	3.63	574.0	<0.001	−0.22±0.36	−0.62	574.0	0.54	−0.08±0.53	−0.15	574.0	0.88
	65–2000	064±0.25	2.57	121.6	0.003	0.50±0.21	2.39	77.7	0.019	0.03±0.31	0.10	83.2	0.92
Condition	10–35	0.07±0.02	4.73	139.0	<0.001	−0.01±0.01	−1.09	414.6	0.28	−0.01±0.02	−0.33	425.3	0.74
	65–2000	0.02±0.02	1.13	102.5	0.26	0.03±0.01	2.52	72.3	0.013	−0.00±0.02	−0.21	77.9	0.83

Estimates ± SEs, test statistics, degrees of freedom (based on Satterthwaite approximation) and *P* values for treatment effects on morphological traits are provided, when pooled across age classes controlling for individual identity and brood identity as random effects and sex, age class and (for models on young ages) exact age as fixed effects.

Table A4
The full statistics for the behavioural trials

Trial	Trait	Early nutritional treatment				Late nutritional treatment				Early*late interaction			
		b	*t*	*df*	*P*	*b*	*t*	*df*	*P*	*b*	*t*	*df*	*P*
SE	Feeding	0.10±0.23	0.43	56.3	0.67	−0.23±0.22	−1.04	89.0	0.30	0.68±0.44	1.56	89.3	0.12
SE	Hopping	−0.06±0.08	−0.73	53.6	0.47	−0.03±0.07	−0.44	80.5	0.66	−0.28±0.14	−2.04	80.5	0.045
SO	Feeding	0.21±0.27	0.77	63.6	0.45	0.69±0.25	2.73	99.1	0.008	0.09±0.51	0.17	99.4	0.86
SO	Hopping	−0.05±0.11	−0.43	60.2	0.67	−0.23±0.10	−2.32	93.3	0.022	−0.12±0.20	−0.60	93.5	0.55
_	RBML 4 h	0.0053±0.0025	2.10	58.1	0.040	−0.0060±0.0019	−3.19	86.0	0.002	0.0001±0.0038	0.18	85.7	0.85
_	RBML 1 d	0.0075±0.0049	1.54	57.6	0.13	0.0025±0.0038	0.66	81.2	0.51	−0.0038±0.0077	−0.50	81.0	0.62
_	BML 4 h	0.06±0.03	1.70	59.2	0.095	−0.08±0.02	−3.46	86.7	<0.001	0.00±0.05	−0.03	83.3	0.97
_	BML 1 d	−0.03±0.06	−0.43	53.5	0.67	−0.06±0.04	−1.29	73.0	0.20	−0.05±0.09	−0.60	70.1	0.55

Estimates ± SEs, test statistics, degrees of freedom (based on Satterthwaite approximation) and *P* values for treatment effects on behavioural traits and body mass loss are provided. Body mass loss was modelled as relative body mass loss (relative to starting body weight; RBML) or as absolute body mass loss while controlling for starting body weight (BML). SE: special-environment trial; SO: special-object trial.