Supplementary information to:

Food availability affects adult survival trajectories depending on early developmental conditions Michael Briga, Egbert Koetsier, Jelle J Boonekamp, Blanca Jimeno & Simon Verhulst Groningen Institute for Evolutionary Life Sciences, University of Groningen, Nijenborgh 7, 9747 AG Groningen, the Netherlands.

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Supplementary information S1: Effect developmental conditions (brood size) on chick development and survival prior to the foraging cost manipulation

We used general linear models to analyze the effect of the brood size manipulation on chick mass during growth. Birth nest and rear nest were included as random effects. All analyses were done in R, v. 2.15.2 or later [1] using function 'lmer' in the package 'lme4' (version 1.1-7, Bates *et al.* 2015). Residuals were checked for normality and homogeneity of variance. Growing up in a large brood resulted in 1.4g (12%) lower mass at age 15 days, i.e. just before fledging (Fig. S1, N=477 χ^2 =71.4, p<0.0001) in agreement with earlier reports [3,4]. Growing up in large broods shifted the whole distribution of chick weights downwards (Fig. S1) as shown by the similar standard deviations of 1.4 g for both groups. The difference in weight was due to differential growth, because at the time of brood size manipulation there was no discernible difference in mass (N=523, χ^2 =1.00, p=0.27). At age 120 days (early adulthood), shortly before birds were housed in the experimental aviaries, individuals reared in large broods were 0.6g (4%) lighter than individuals reared in small broods (Fig. S1, N=508, χ^2 =15.1, p=0.0001). Standard deviations were similar for both groups (1.51 vs. 1.58 for small and large broods respectively). Thus growing up in poor developmental conditions impaired growth and this effect persisted into adulthood. Small and large broods thus reflect benign and harsh developmental conditions respectively.

We tested if manipulated brood size affected chick survival up to adulthood (3 months), including all manipulated chicks in the breeding batches from which birds were allocated to the foraging cost manipulation (n= 877 chicks in 293 nests). Of the 422 young reared in small broods, 21 (5.0 %) died before the age of 3 months. Of the 455 young reared in large broods, 41 (9.0%) died before the age of 3 months. Although there was a mortality difference in the expected direction, it is statistically far from being statistically significant (logistic regression: z=0.31, p=0.76). More importantly, the absolute difference is small, and we therefore consider it safe to assume that there was no bias from selective disappearance of individuals from large broods before the start of the foraging cost experiment during adulthood. Furthermore, the direction of the mortality difference is such that this will have decreased the difference in phenotypic quality between birds reared in small and large broods, making our statistical tests more conservative.

Fig. S1 Birds reared in large broods attained a lower mass as chicks (age 15 days, just before fledging,) and as young adult (age 120 days, i.e. just before the start of the manipulation of adult conditions, i.e. the foraging treatment). Boxplots show median, first and third quartiles and whiskers show 95% confidence interval.

Supplementary information S2: Cox proportional hazard analyses

To identify which covariates and/or random effects affected mortality in addition to the experimental we treatments we performed survival analyses using the counting process formulation of the Cox proportional hazard (CPH) model (Cox 1972; Andersen et al. 1993; Therneau and Grambsch 2000). The counting process formulation allows the coefficient to be estimated at each time point and thus timedependent covariates, such as age, can be included. Age was partitioned into 'starting age' and 'time in treatment', with day 1 for all birds being the day they started the foraging cost experiment, as advocated for randomized experiments [8]. Survival was checked daily and as time base we therefore used daily intervals. Deaths that occurred due to accidents (N=7) and birds still alive were rightcensored.

Analyses were done in R, v. 3.2.1 [1] using function 'coxme' in the package coxme (version 2.2-3; Therneau 2012). To find the model best supported by the data, we used the function 'dredge' of the package 'MuMIn' [10]. In brief, this is a hypothesis-based approach that generates, given a global model, subset models that best fit the data. This makes it possible to assess model support for each hypothesis. Model support is shown here by ranking all subset models within six AICc of the best model fit. CPH assumptions were checked for the best fitting models using scaled deviance and martingale residual plots [6,7].

There was potential non-independence at several levels in our data set (shared birth nest, genetic mother, genetic father, rear nest, rear mother, rear father, birth batch and aviary), which we checked for by entering these factors as random effect. Note however that the experiment was balanced with respect to all these effects, except aviary, because adult treatment was varied at the aviary level. We therefore performed all analyses with aviary as random effect, and subsequently tested effects of all other potential random effects by adding these one at the time to the final model. Adding other random effects to the final model in no case improved model fit or otherwise altered the conclusions.

Table S1 (next page) Cox proportional hazard analyses of manipulations effects on lifespan

For birds from benign developmental conditions, there was little evidence that adult environment affected lifespan (Table S1A: ΔAICc=+0.7). In contrast, birds from harsh developmental conditions lived shorter in harsh than in benign adult environments (Table S1B: ΔAICc=-3.6). In the benign adult environment, the best fitting model did not include an effect of developmental conditions (Table S1C: ΔAICc≥+1.8). In the harsh adult environment, birds from benign developmental conditions live longer than birds from harsh developmental conditions (Table S1D: ΔAICc=-10.9). The interaction between the developmental conditions and adult environment obtained moderate support (Table S1E: ΔAICc≥1.2). For table 1E, only models within 6AICc of the best fitting model are shown. Values indicate model coefficients and are missing when the term was excluded from the model.

Note that these are Cox proportional hazards models and model coefficients are therefore hazard ratios relative to a baseline hazard, which always is a benign group. A hazard ratio of one implies no effect and for example a hazard ratio of 1.37 for *Devel.* (manipulation during development) means that the hazard rate increases with 37% between benign and harsh developmental conditions. Note that there is no main effect *Age* since it is included in the baseline mortality curve. All models included aviary as random effect. Results indicating how best to include *AgeStart* can be found in Table S2. Abbreviations: Devel.: Developmental conditions (i.e brood size manipulation); Adult: adult conditions (i.e. foraging cost manipulation); AgeStart: age at start of the foraging treatment. Interaction terms are indicated by *.

Table S2 Cox proportional hazard analyses to determine how to account for age at start of the experiment

The model best fitting the data did not include experiment-specific *AgeStart* effects. In contrast, model support for including *AgeStart* was strong, since excluding *AgeStart* gave the worst possible model (ΔAICc =14.73; model 11). *Age* and *AgeStart* variables are per year. Further table specifications as in Table S1.

Table S3 Cox proportional hazards analyses to show that sex dependent survival is independent of experimental manipulations

There was considerable model support for female biased mortality in the six best fitting models and model fit deteriorated when sex was not included (ΔAICc =3.1; model 7). Yet, the female biased mortality seems most pronounced at older ages since the 3 best fitting models also include a sex*age interaction (ΔAICc ≥1.8; model 4). In contrast, the model support sex-specific manipulation effects was weak: models 1 (best fitting) and 4 do not include interactions between sex and experimental manipulations. All '*Age'* and '*AgeStart'* terms are per year. Further table specifications as in Table S1.

Supplementary information S3: Comparison of parametric mortality model fits

Table S4 Model selection results for parametric model fits using maximum likelihood approach of the R package fitdistrplus [11]. Shown numbers are AIC values (Akaike's 'An Information Criterion' [12]). Results in bold are best fits. Multiple 'best fits' indicate that these models fit approximately equally well (ΔAIC<2; [12]). Consistent with Bayesian methods, the Gompertz function fitted the data better than the exponential function.

Supplementary information S4: Gompertz fits with R package BaSTA

Fig. S2 Parameter trace plot of the MCMC optimization for Gompertz fits with BaSTA as in Fig 2. Settings were 4 parallel runs with 500,000 iterations, 100,000 burn in period and a thinning of 1000. Abbreviations: B: Benign and H: Harsh, in chronological order such that e.g. the HB group indicates harsh developmental followed by benign adult conditions. Note variation in Y-axes between panels.

Supplementary information S5: Comparison of survival and mortality of the four experimental groups

Fig. S3 Survival (A) and mortality (B) trajectories of the 4 experimental groups show that the HH group differs most from all other groups. Grey lines represent the benign adult environment, black lines the harsh adult environment, full and dotted lines show the benign and harsh developmental conditions respectively. Group abbreviations: B: benign conditions and H: harsh conditions, in chronological order, such that e.g. the BH group indicate benign developmental followed by harsh adult conditions.

Supplementary information S6: Sex-specific mortality trajectories

Fig. S4 Survival curve and instantaneous mortality rate in relation to sex:

(A) Proportion of birds surviving since entering the adult treatment. (B) Instantaneous mortality rate as a function of time in treatment. In panel (B), dots represent mortality data and lines show Gompertz fits. (C) Posterior distributions of Gompertz parameters, showing that the sexes have very similar age independent mortality rate (Gompertz A, KLD=0.50) but that the rate of actuarial senescence is higher in females (Gompertz B, KLD=0.92).

Supplementary information 7: References

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