Genetic variation in total metabolic rate and correlations with other energy budget components and life history in *Daphnia magna*

Sigurd Einum[1], Erlend I. F. Fossen[1], Victor Parry[1,2] & Christophe Pélabon[1]

[1] Centre for Biodiversity Dynamics, Department of Biology, Norwegian University of Science and Technology, Trondheim, Norway

[2] Department of Ecology and Ecosystem Modelling, Institute of Biochemistry and Biology, University of Potsdam, Germany

Correspondence: S. Einum, Centre for Biodiversity Dynamics, Department of Biology, Norwegian University of Science and Technology, 7491 Trondheim, Norway. Tel.: +47 73590564; e-mail: Sigurd.einum@ntnu.no

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Abstract

Much is known about the genetic variance in certain components of metabolism, most notably resting and maximum metabolic rate. This is in stark contrast to the lack of information on genetic variance in total metabolic rate (TMR) and how this trait correlates with other components of the energy budget or life history traits. Here we quantify genetic variance in TMR, food consumption, juvenile somatic growth rate and age at maturation under *ad lib* food availability in a set of 10 clones of *Daphnia magna* from a natural population. Broad sense evolvabilities (0.16-0.56%) were on the same order of magnitude as those typically observed for physiological and life history traits, and suggests that all these traits have the potential to evolve within this population. We did not find support for the previously hypothesized positive genetic correlation between metabolic rate and growth rate. Rather, the patterns of genetic correlations suggest that genetic variance in food consumption is the single most influential trait shaping somatic growth rate, but that additional variance in growth can be explained by considering the joint effect of consumption and TMR. The genetic variance in consumption and TMR also translated into genetic variance in age at maturation, creating a direct link between these energy budget components and a life history trait with strong fitness effects. Moreover, a weak positive correlation between TMR and food consumption suggests the presence of substantial amounts of independent genetic control of these traits, consistent with results obtained using genomic approaches.

Key-words: Respiration, food intake, feeding rate, heritability, gross growth efficiency, assimilation efficiency, specific dynamic action

Introduction

Metabolic rate is one of the physiological traits that has received most interest among ecologist and evolutionary biologists. Well described sources of variation in metabolism includes environmental influences (e.g. temperature, Gillooly et al. 2001; habitat structure, Millidine et al. 2006) and the state of the organism (e.g. reproductive status, Vezina et al. 2006; body size, Gillooly et al. 2001; sex, Marhold and Nagel 1995; parasite infections, Scantlebury et al. 2007). Environmental influences and the state of the organism are likely responsible for parts of the pronounced and consistent (over time) individual variation in metabolism (Nespolo and Franco 2007; Metcalfe et al. 2016). These may be particularly prominent sources of variation for studies of field metabolic rate, which measures the total metabolic rate (TMR) of individuals performing their natural activity in the wild (Berteaux et al. 1996; Fyhn et al. 2001). There is also considerable evidence for genetically based variation in components of the TMR, with one such component being basal (for endotherms) or standard (for ectotherms) metabolic rate (hereafter collectively referred to as resting metabolic rate, RMR) (Ksiazek et al. 2004; Sadowska et al. 2005; Rønning et al. 2007; Nilsson et al. 2009; Careau et al. 2011). RMR represents the energetic cost of living in the absence of natural behavioural activity, and in the absence of the energetic costs of digestion and growth (i.e. specific dynamic action, Jobling 1981). Additional evidence of genetic variance comes from studies of maximum metabolic rate and aerobic scope (maximum minus resting metabolic rate) (Dohm et al. 2001; Sadowska et al. 2005).

Estimates of genetic variance in resting and maximum metabolic rates allow an understanding of their evolutionary potential, and how such variance might contribute to the consistent differences observed among individuals in studies where the genetic component can not be estimated. However, to our knowledge, there are no published estimates of within-

73 population genetic variance in the TMR to accompany estimates of individual variance in
74 field metabolic rates, despite the direct influence this trait has on energy budgets. Energy
75 budgets quantify how somatic growth rates depend on variation in food consumption (energy
76 intake), assimilation efficiency (proportion of consumed energy not lost through faeces and
77 urea), and TMR (energy loss through heat production). Energy loss through faeces, urea and
78 heat production influences how efficiently ingested food is transformed into somatic tissue,
79 which can be expressed as the gross growth efficiency (i.e. somatic growth divided by food
80 consumption). Genetic variation in growth efficiency has been a topic of interest in breeding
81 programs of domesticated species due to the economic importance of this trait (Bordas et al.
82 1992; Mrode and Kennedy 1993), and there is also some evidence for genetic differences in
83 growth efficiency among populations when reared in a common environment (Present and
84 Conover 1992; Jonsson et al. 2001; Finstad et al. 2004). However, due to the joint effect of
85 assimilation efficiency and metabolic rates on growth efficiency, such studies shed little light
86 on the question of whether there is genetic variance in TMR, and if so whether TMR is
87 genetically correlated with somatic growth rate.
88
89 The relationship between TMR and somatic growth rate is complex, partly because of their
90 reciprocal causal relationships, and partly because the relationship may depend on food
91 availability. First, for a given level of food consumption, having a high TMR will reduce
92 growth because more energy is lost through heat production, resulting in a negative genetic
93 correlation between these two traits. However, if food abundance is not restricted, a high
94 TMR may be associated with a higher food consumption. This may be the case if variation in
95 TMR is primarily driven by variation in RMR, and if RMR is positively genetically
96 correlated with food consumption (Ksiazek et al. 2004; Gebczynski and Konarzewski 2009).
97 Alternatively, a high TMR may be a *result of* high food consumption due to the effect of food

intake on the specific dynamic action (Jobling 1981). Both these effects would tend to create a positive correlation between TMR and food consumption, and in turn contributing positively to growth. Finally, there may be genetic variation in TMR that is not related to food consumption, such as costs associated with immune systems (Poulsen et al. 2002). Similarly, there may be genetic variation in food consumption that is not linked to TMR. As an example, a single gene in humans is shown to influence food consumption without influencing TMR (Haupt et al. 2009). These independent sources of variation in TMR and food consumption may weaken the phenotypic and genetic correlation between these two traits. It is therefore challenging to predict whether, and in which direction, TMR is genetically correlated with growth rate (and associated traits like age at maturation), and empirical data are lacking.

Here, using the highly suitable model organism *Daphnia magna*, we quantify genetic variance in TMR, food consumption, juvenile somatic growth rate and age at maturation under *ad lib* food availability among a set of 10 clones from a natural population, and test for genetic correlations among these traits.

Material and Methods

Study animals and husbandry

Ephippia of *D. magna*, containing up to two sexually produced resting eggs, were collected in November 2014 from the surface sediment of a shallow pond at Værøy Island (Sandtjønna, 1.0 ha, 67.687°N 12.672°E), northern Norway. Ten genotypes, hereby referred to as clones, each from a separate ephippia, were hatched in December 2014 and cultured separately for a minimum of three asexual generations at 17 °C with a 16L:8D photoperiod in 250 mL jars containing a modified ADaM medium (Klüttgen et al., 1994, SeO_2 concentration reduced by

50%). Being a result of sexual reproduction, each clone is genetically unique at the molecular level, and moreover these clones have previously been shown to vary genetically in thermal plasticity of life-history traits (Fossen et al., 2018). The clone lines, containing five adults per jar, were fed three times a week with Shellfish Diet 1800 (Reed Mariculture Inc, USA) at a algae concentration of 4×10^5 cells mL^{-1}, and the medium was changed weekly. All experiments and associated acclimation described below were at 17 °C, using the same medium and food as described here. During the period May 2015 – November 2016 we estimated clone-specific values of food consumption, somatic growth rate, age at maturation and total metabolic rate that allowed us to estimate genetic variance and genetic correlations among these traits.

Food Consumption

Prior to experiments, 8 replicate 250 mL jars of each clone were cultured separately for two asexual generations. Each clone line replicate started from animals born in different jars to ensure independent replicates of clones. Animals were fed three times a week (concentration in medium 4×10^5 cells mL^{-1}), and the medium was changed weekly. Food consumption was measured in five blocks during 22. – 28. August 2016. For each block, five individuals (second clutch juvenile females ≤ 24 hour old) from each of the 10 clones (i.e. 5×5 individuals per clone in total) were transferred from the culture jars into individual 50 mL centrifuge tubes and kept there for five days prior to measurements. Animals were fed every second day (concentration in medium 2.62×10^5 cells mL^{-1}) during this rearing. This feeding regime represents *ad libitum* concentrations during the juvenile growth stage (unpublished data). This procedure ensured standardization of the rearing environment prior to measurements. On the day of food consumption measurements, individuals were distributed individually into 3 mL wells of three spot plates. Each spot plate contained one or two individuals of every clone and two or four controls (i.e. wells without *Daphnia*) were present

in each spot plate. Each well contained an algae concentration of 3.12×10^5 cells mL^{-1}. *Daphnia* were kept in the wells for one hour before being removed and photographed using a stereomicroscope. We then sampled 2 mL from each spot plate well and mixed this with 8 mL isoton in a cuvette before measuring the number of algae left using a Beckman Coulter counter (Beckman Coulter Inc, USA). Food consumption for each individual was calculated as the average cell count of the control wells minus the cell count in their respective well. From the photographs we measured the gut length (*GL*, mm, measured from the top of midgut to the bottom of hindgut when the animal is relaxed) of each individual using ImageJ v1.48 (National Institutes of Health, Bethesda, MD). These length measurements were transformed to dry mass (*DM*, mg) using the following relationship between dry mass (*DM*) and gut length (*GL*): $DM = 0.00679GL^{2.75}$ (Fossen et al. 2018).

Somatic growth rate and age at maturation

Juvenile somatic growth rate and age at maturation were measured during May-June 2015 in two blocks with four replicates for each of the 10 clones in each block (i.e. 8 individual per clone in total). These data constitute a part of a larger data set from an experiment describing the genetic variance in thermal reaction norms (Fossen et al. 2018), and here we only use the data obtained at 17 °C (i.e. same temperature as for the other traits). Prior to experiments, 13 to 14 replicate 250 mL jars of each clone were cultured separately for three asexual generations. Each clone line replicate started from animals born in different jars to ensure independent replicates of clones. Animals were fed three times a week (concentration in medium 4×10^5 cells mL^{-1}), and the medium was changed weekly.

Fourth generation female neonates (<24 hours old) from the second or later clutches born at 17°C were transferred to individual 50 mL centrifuge tubes with 17°C ADaM medium. These

juveniles were haphazardly chosen within each clonal line and from different mothers within the same clone to minimize maternal effects in the estimation of the genetic (clonal) variance. For each clone, female neonates (<24 hours old) from the second or later clutches were photographed for size measurements and transferred to individual 50 mL centrifuge tubes. These juveniles were haphazardly chosen within each clonal line and from different mothers within the same clone to minimize maternal effects in the estimation of the genetic (clonal) variance. Animals were fed every second day (concentration in medium $2.62\times10_5$ cells mL_{-1}). We checked individuals daily at the same time to estimate the age at maturation, defined as the time when eggs were first visible in the brood chamber. Mature individuals were photographed for size measurements. Initial and final dry mass was calculated as above. Using dry mass of neonates (DM_{start}), dry mass at maturation (DM_{end}) and the number of days between the two measurements (*duration*), the somatic growth rate (SGR) was calculated as: $\mathrm{SGR} = \frac{\ln(DM_{end}) - \ln(DM_{start})}{duration}$ and represented the proportional increase in dry mass per day.

Total metabolic rate

Animals used for metabolic rate measurements were reared in a climate cabinet at 17°C for a minimum of three asexual generations. Each generation was started from juveniles from the second or later clutches born in different 250 mL jars to obtain independent replicates of clones. Animals were fed every second day throughout the experimental period (concentration in medium $2.62\times10_5$ cells mL_{-1}). Female juveniles from second or later clutches and from independent jars were used for the measurements. Total metabolic rate (TMR) was measured as oxygen consumption of fed, free-swimming individuals (second or later clutch) following the method described in Yashchenko et al. (2016) during June-November 2016. To account for the effect of body mass on TMR, we estimated the allometric relationship between the two traits. We conducted a total of 15 runs with 20

individuals per run, resulting in 27-30 individuals per clone and a total sample size of 288 measurements. To increase the range of sizes and estimate the allometric relationship between body mass and metabolic rate with high precision, each experimental run consisted of one large (close to maturity, mean dry mass: 0.053 mg) and one small (recently born, mean dry mass: 0.007 mg) juvenile female from each of the 10 clones. Dry mass of individuals was determined as above. By using juveniles we avoided using females with eggs/embryos which are known to have lower metabolic rates than the female's somatic tissue (Glazier 1991).

Oxygen consumption was measured using a sealed glass microplate equipped with planar oxygen sensor spots with optical isolation glued onto the bottom of 200 µl wells (Loligo Systems, Denmark) integrated with a 24-channel fluorescence-based respirometry system (SDR SensorDish® Reader, PreSens, Germany). *Daphnia* were transferred into wells with air-saturated ADaM, which were sealed using an adhesive PCR film (Thermo Scientific, Waltham, MA, USA) while ensuring no air bubbles in the wells. The reader was placed inside a Memmert Peltier-cooled incubator IPP (Memmert, Germany). Oxygen concentrations inside wells were measured in darkness every 3 min for a duration of 120-150 min by using SDR v38 Software (PreSens, Germany). In each run, four wells with medium but without animals were used to control for temporal changes in pressure and temperature, as well as microbial respiration. Oxygen consumption was estimated from the decline in oxygen concentration during the interval of time where this decline was linear after controlling for oxygen diffusion into the wells (Yashchenko et al. 2016).

Statistical analyses

All statistical analyses were conducted with linear mixed-effects models using the package lme4 (v. 1.1-7, Bates *et al.*, 2015) in R v.3.1.1 (R Core Team, 2014). For somatic growth rate

and age at maturation, clone-specific values were obtained as best linear unbiased predictions (BLUPs) from models that included block as a categorical variable and clone as a random effect. TMR data were log-transformed, and log body mass was included as a covariate, plate ID as a fixed effect (two different plates were used), and well ID, run ID and clone as random intercepts. For food consumption, body size was included as a covariate, and plate ID and clone were included as random intercepts. The relationship between food consumption and body mass was not log-transformed because it was more linear and had a higher R^2 without log-transformation ($R^2 = 0.78$ for non-log transformed vs. $R^2 = 0.67$ for log-transformed). For both TMR and food consumption we also allowed for a difference among clones in the effect of body mass (i.e. random slope) in the initial model. However, model selection using log-likelihood contrasts (Zuur et al. 2009) showed that there was no variation in the body size effect among clones for either of these traits (food consumption $P = 0.97$; TMR $P = 1$). We could thus use BLUPs from the reduced models (i.e. without random slopes) to obtain body size adjusted clone-specific estimates for food consumption and TMR. The use of BLUPs for predicting individual breeding value has been criticized because bias arise due to effects that are not accounted for in the model (Hadfield et al. 2010). This problem is most likely limited in our case due to the similarity of the experimental conditions and the equal sample sizes among clones.

The population's evolutionary potential of the different traits was estimated as broad sense evolvability (clonal variance / $mean^2$) (Houle 1992; Hansen et al. 2011). Evolvability measures the expected percentage change in a trait per generation under a unit strength of selection. Compared to heritability, evolvability is independent from the environmental variance and represents a measure of the evolutionary potential that is directly comparable across traits, populations and species (Hansen et al. 2011). Genetic correlations between traits

were estimated as the Pearson product-moment correlation between the clone trait means (i.e. BLUPs for somatic growth rate and age at maturation, BLUPs from models including body size as a covariate for TMR and food consumption). In addition to the correlations between the directly measured traits, we were also interested in quantifying how strongly genetic variance in somatic growth rate and age at maturation were linked to the food consumption relative to the TMR. Thus, we calculated clone specific values of log(food consumption/TMR) based on the BLUPs for these variables.

Results

Food consumption increased with body mass (Fig. 1). Food consumption corrected for body mass varied among clones (significant variation in intercept among clones $P < 0.001$, Fig. 1), and the evolvability of this trait was estimated to 0.41%. On a log-log scale, the estimated allometric slope (± 1SE) between food consumption and body mass was 0.98 ± 0.05, suggesting that the scaling between these two traits is not significantly different from isometry. TMR was also positively related to body mass (Fig. 2, allometric slope of 0.94 ± 0.01 SE), and there was significant variation in the size corrected TMR (among clone variation in intercept, $P = 0.037$). Evolvability of TMR was estimated to be 0.16%. A similar level of genetic variance was observed for somatic growth rate, with estimated evolvability being 0.19% (Fig. 3a, $P = 0.037$). Finally, there was a somewhat larger genetic variance in age at maturation, with an estimated evolvability of 0.56% (Fig. 3b, $P < 0.001$).

The genetic correlation between TMR (corrected for body mass) and growth rate was weakly negative and statistically non-significant ($r = -0.30$, $P = 0.41$, $n = 10$). There was a weak, statistically non-significant positive correlation between TMR and food consumption ($r = 0.39$, $P = 0.26$, $n = 10$). However, a positive and statistically significant genetic correlation

was observed between food consumption and somatic growth rate (Fig. 4A, $r = 0.66$, $P = 0.039$, $n = 10$). Furthermore, when we accounted for the energy loss through heat production by considering the relative relative difference between food consumption and TMR this correlation with somatic growth rate became even stronger (Fig. 4B, $r = 0.88$, $P < 0.001$, $n = 10$). This translated into a trend of a negative correlation between food consumption and age at maturation (Fig. 4C, $r = -0.56$, $P = 0.090$, $n = 10$), and a significant negative correlation between the relative difference between food consumption and metabolic rate and age at maturation (Fig. 4D, $r = -0.83$, $P = 0.003$, $n = 10$).

Discussion

In the current study we demonstrate significant within-population genetic variance in three important components of the energy budget and one life-history trait among clones of *D. magna*. The observed broad sense evolvabilities (0.16-0.56%) are on the same order of magnitude as those typically observed for physiological and life history traits (Hansen et al. 2011), and suggests that all these traits have the potential to evolve within this population. The patterns of genetic correlations suggest that genetic variance in food consumption is the single most influential trait shaping somatic growth rate, but that additional variance in somatic growth can be explained by considering the joint effect of consumption and TMR. Residual variation from this latter relationship is a combination of measurement errors and genetic variance in assimilation efficiency, although the relative magnitude of these two remains unknown. The genetic variance in food consumption and TMR also translated into genetic variance in age at maturation, creating a direct link between these energy budget components and a life history trait with strong fitness effects.

297 It has been hypothesized that there should be a positive correlation between resting metabolic
298 rate and growth rate under *ad lib* feeding conditions (Biro & Stamps 2010; Burton et al.
299 2011; but see Einum 2014). This is based on the assumption that a high resting metabolic rate
300 provides the ability to generate the high TMR required to take advantage of high food
301 availability. Hence, a positive correlation between TMR and growth would also be expected.
302 This was not supported in the current study, where the correlation between TMR and growth
303 rate was weakly negative. Empirical support for such positive correlation between resting
304 metabolic rate and growth rate is also weak in studies of phenotypic correlations. Three out of
305 four studies on phenotypic correlations under *ad lib* food conditions reviewed by Burton et al.
306 (2011) reported positive correlations between resting metabolic rate and growth rate
307 (Yamamoto et al. 1998; McCarthy 2000; Alvarez and Nicieza 2005), whereas the last one
308 showed a negative correlation (Steyermark 2002). It is noteworthy that all positive
309 correlations came from experiments where juvenile salmonid fish (*Salmo* sp.) were reared in
310 groups, and correlations were estimated within these groups. Juvenile salmonids show high
311 levels of intraspecific aggressiveness, and their social status depends on metabolic rate
312 (Metcalfe et al. 1995; Yamamoto et al. 1998). Variation in social status, in turn, creates
313 variation in food availability even when food is abundant (Metcalfe 1991). Thus, positive
314 effects of high metabolism on growth under *ad lib* food conditions may only be present in the
315 special case where variation in metabolism translates into variation in food availability
316 through interference competition (see also Reid et al. 2012).
317
318 Our quantification of genetic variance in TMR complements previous studies that
319 demonstrate genetic variance in resting or maximum metabolic rates (Dohm et al. 2001;
320 Ksiazek et al. 2004; Rønning et al. 2005; Sadowska et al. 2005; Nilsson et al. 2009; Careau et
321 al. 2011). One might ask what drives this empirical focus on genetic variance in the separate

metabolic rate components, which is also evident in studies of individual variation (Careau et al. 2008), rather than TMR? We suspect that the most important reason for this is the need to minimize 'noise' when estimating variance among individuals and genotypes which can be logistically challenging for TMR. For example, fish, birds, and mammals show irregular activity and feeding patterns that makes TMR highly variable over time. Thus, a common approach for estimating resting metabolic rate is to keep starved organism in metabolic chambers for an extended time period (e.g. over night), and then use the lowest average value over a short period as a proxy for resting metabolism (Careau et al. 2008). For maximum metabolic rate, one commonly applied approach is to stimulate intense activity until exhaustion prior to metabolism measurements (Norin and Clark 2016). However, given that such resting and maximal metabolic rates are likely infrequently expressed in the wild, it is not clear how often these traits are exposed to direct selection. This suggests that TMR could be just as, if not more, ecologically relevant because of the potential fitness consequences of its variance.

One particular advantage of using *Daphnia* as a model organism in ecological and evolutionary studies of energy budget components is that they perform a more or less continuous swimming activity that enables them to stay pelagic in the water column (resting *Daphnia* sink to the bottom), and which causes oxygen consumption to vary little through time during measurements (Yashchenko et al. 2016). This allowed us to quantify genetic variance in total metabolic rate based on short-term individual measurements of active individuals, and hence to include any contribution from genetic variation in activity (Sereni and Einum 2015). *Daphnia* also appear to show relatively little variation through time in food consumption (under a given feeding regime), as indicated by the large amount of variation in food consumption that could be explained by body size and clonal identity in our short-term

measurements (R_2 = 0.78). Further support for this arises from the genetic correlation between short-term food consumption and longer-term growth rate. This made it feasible to include the potential contribution from genetic variation in specific dynamic action to the variation in total metabolic rate, rather than measuring metabolic rates of starved individuals. Finally, the clonal nature of *Daphnia* enabled us to obtain truly independent estimates of the mean clonal value for the different traits (i.e. different individuals used to estimate each trait).

One caveat with the present study is that sample sizes for genetic correlation analyses were restricted, and thus only strong genetic correlations can be expected to show up as being statistically significant. In other words, non-significant correlations in the present study should not be interpreted as demonstrating a lack of correlation, but rather that the sample size may have been insufficient to detect them with sufficient confidence. It is also unknown to what extent non-additive genetic variation (due to epistasis and dominance) influenced our estimates of genetic correlations. However, given the close empirical correspondence between genetic and phenotypic correlations (i.e. 'Cheverud's conjecture'; Cheverud 1988, Roff 1995) it seems unlikely that narrow sense genetic correlations would deviate much from our estimated broad sense genetic correlations.

In conclusion, the present study provides insights into the genetic variation of, and genetic correlations between total metabolic rate, food consumption, growth rate and age at maturation. We observed that genotypes that have a high food consumption relative to TMR achieve a high growth rate and a low age at maturation. Thus, this relationship is not strongly influenced by genetic variance in assimilation efficiency, which would weaken such correlations. We are not aware of any studies quantifying genetic variance in assimilation efficiency, but the current study suggests that such variation, if present, has relatively minor

effects on genetic variation in energy budgets. Furthermore, the genetic correlation between TMR and growth rate was weakly negative (although non-significant), suggesting that it is unlikely to have turned significantly positive with a larger sample size. Finally, the weak correlation between TMR and food consumption suggests the presence of substantial amounts of independent genetic control of these traits, as suggested by genomic methods (Haupt et al. 2009). This should allow these traits to evolve rather independently, which enables divergent evolution of growth efficiencies among populations (Present and Conover 1992; Jonsson et al. 2001; Finstad et al. 2004).

Ethical standards

Animals were hatched from resting eggs, previously collected in the wild, and grown under healthy conditions in the laboratory. Laboratory conditions and procedures are not regulated by law for this particular crustacean species, as it is considered to be a less sentient animal.

Conflict of interest

The authors declare that they have no conflict of interest.

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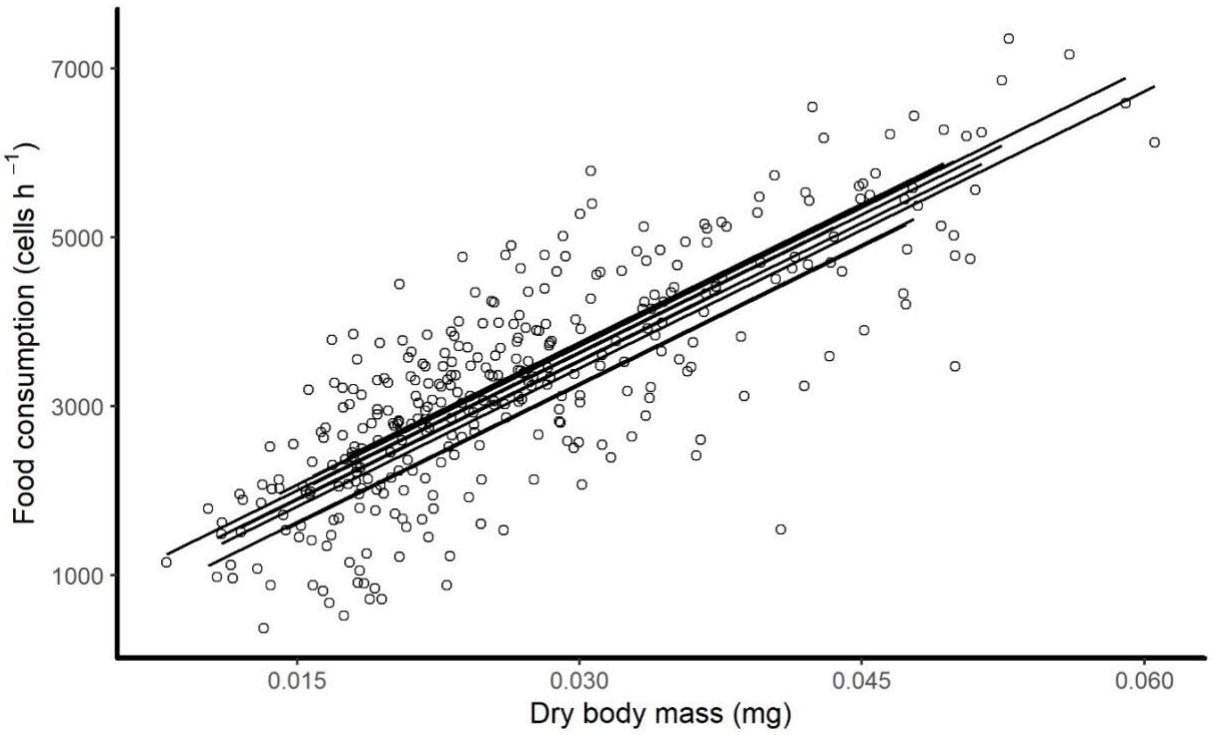

514

515 **Figure 1.** The relationship between dry body mass and food consumption in 10 clones of *D.*

516 *magna*. Separate regression lines are given for each clone.

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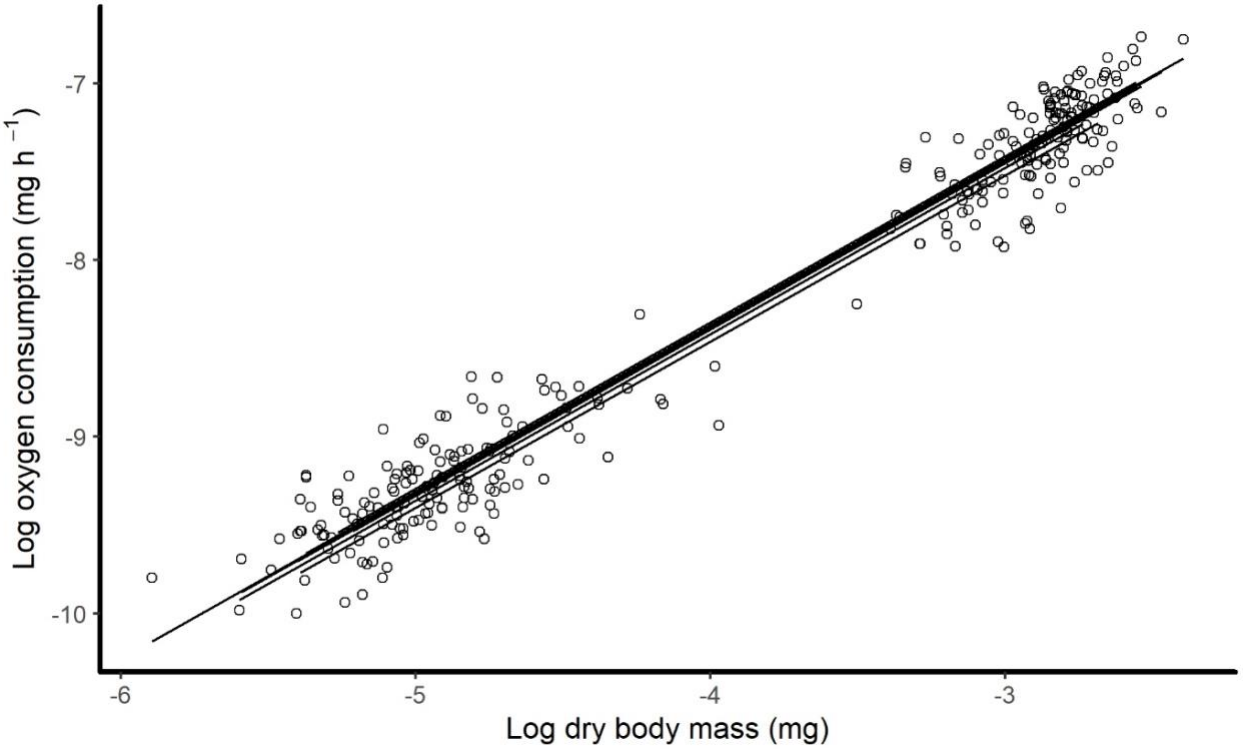
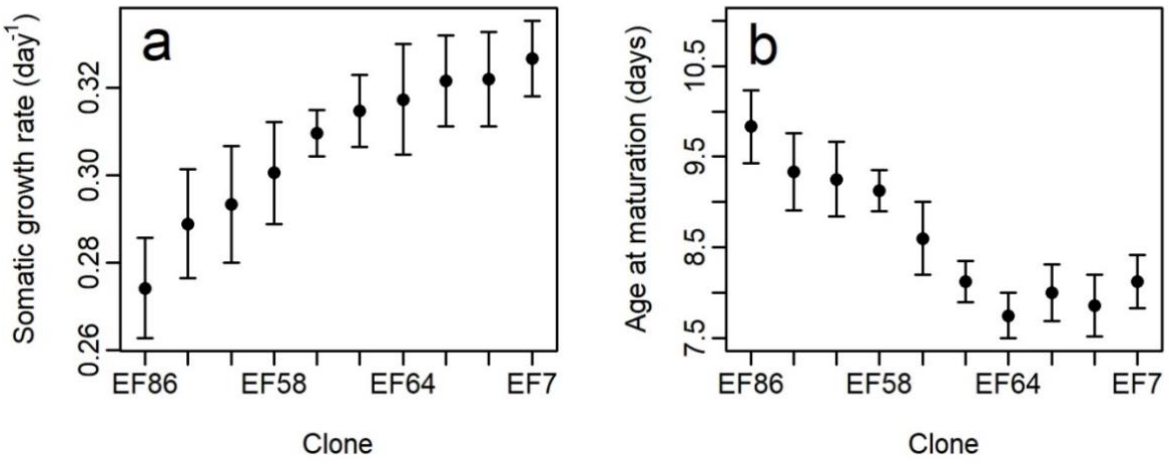


Figure 2. The relationship between dry body mass and metabolic rate in 10 clones of *D. magna*. Separate regression lines are given for each clone.



523

524 **Figure 3.** Mean ± SE (a) juvenile somatic growth rate and (b) age at maturation among 10

525 clones of *D. magna*. Clones are sorted from lowest to highest somatic growth rate.

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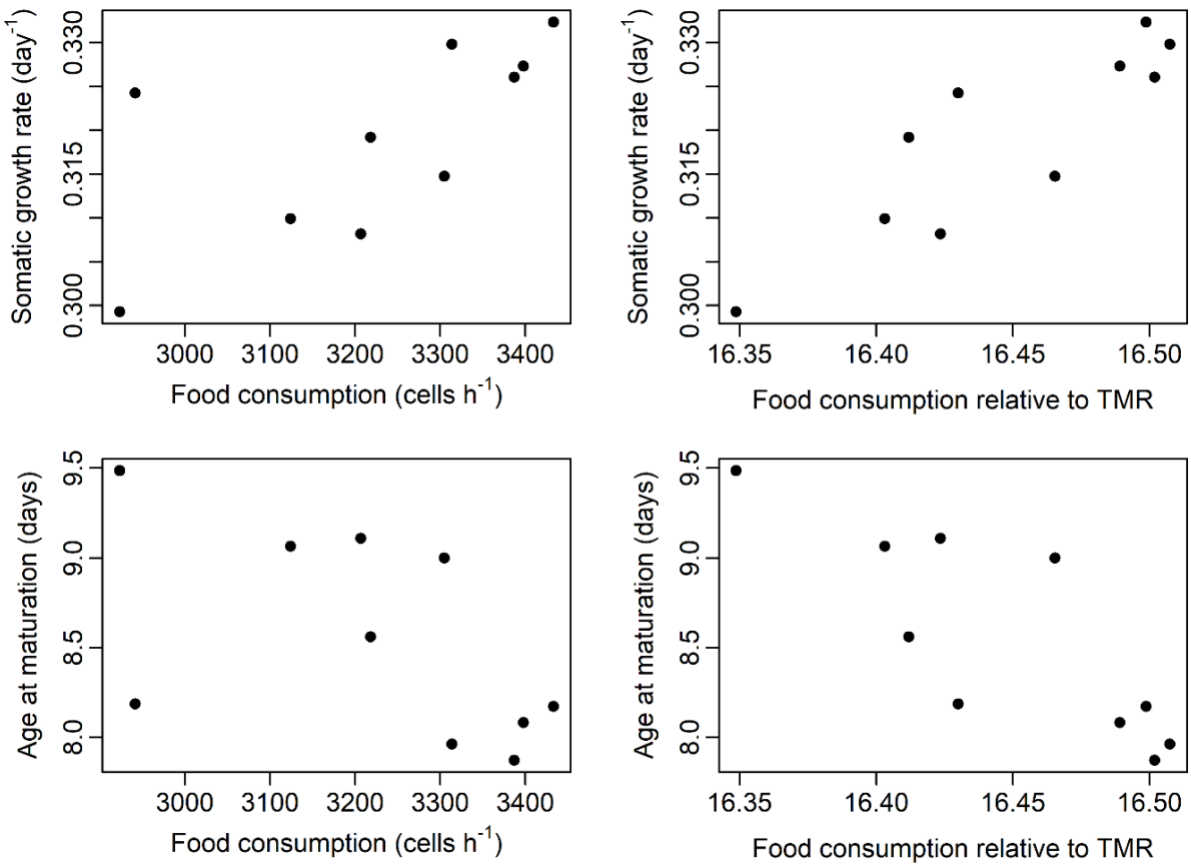

527

528 **Figure 4.** Clone-specific estimates of food consumption (left panels) and food consumption
529 relative to total metabolic rate (right panels) correlated against somatic growth rates (top
530 panels) and age at maturation (bottom panels) among 10 clones of *D. magna* from a single
531 population.

532