1   **Eco-evolutionary Dynamics: Experiments in a model system**

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2

3   **Abstract (208 words)**

4   Understanding the consequences of environmental change on both long and short term ecological and
5   evolutionary dynamics is a basic prerequisite for any effective conservation or management
6   programme but inherently problematic because of the complex interplay between ecological and
7   evolutionary processes. Components of such complexity have been described in isolation or within
8   conceptual models on numerous occasions. What remains lacking are studies that characterise
9   effectively the coupled ecological and evolutionary dynamics, to demonstrate feedback mechanisms
10 that influence both phenotypic change, and its effects on population demography, in organisms with
11 complex life-histories. We present a systems-based approach that brings together multiple effects that
12 "shape" an organism's life history (e.g. direct and delayed life history consequences of environmental
13 variation) and the resulting eco-evolutionary population dynamics. Using soil mites in microcosms we
14 characterise ecological, phenotypic and evolutionary dynamics in replicated populations in response to
15 experimental manipulations of environment (e.g. the competitive environment, female age, male
16 quality). Our results demonstrate that population dynamics are complex and are affected by both
17 plastic and evolved responses to past and present environments, and that the emergent population
18 dynamic itself shaped the landscape for natural selection to act on in subsequent generations.
19 Evolutionary and ecological effects on dynamics can therefore be almost impossible to partition,
20 which needs to be considered and appreciated in research, management and conservation.

21 **Keywords:** eco-evolutionary, parental effects, maternal, phenotype, body size, offspring, population
22 dynamics, life histories, harvesting, natural selection, evolutionary rescue

23

Contents

43 **1.   Introduction**

44 A fundamental goal in evolutionary ecology is to understand the mechanisms responsible for

45 generating the phenotypic variation upon which selection acts. Similarly, a fundamental goal in

46 population ecology is to understand the role that individual phenotypic variation, created by density

47 independent and/or density dependent processes, plays in shaping population dynamic patterns. Thus,

48 understanding between-individual phenotypic variation is key to understanding both ecological and

49 evolutionary dynamics (Benton et al., 2006). Traditionally, an individual's phenotype has been

50 considered a consequence of interaction between its genes and the environment in which they are

51 expressed. Phenotypic variation has thus been envisaged as the sum of direct environmental and

52 genetic effects, plus their interactions. Despite this recognition, for most of the history of ecology it

53 has been assumed that the ways in which genes and environments interact are relatively unimportant

54 for population dynamics (i.e. the trait changes from life history evolution are either small or take too

55 long to influence short-term dynamics). Two major conceptual advances have recently occurred that

56 casts doubt on this traditional view. First, we now recognize that the environment experienced in

57 previous generations can have consequences for contemporary phenotypes (Beckerman et al., 2002),

58 reflecting the importance of non-genetic modes of inheritance that relate parental and offspring life-

59 histories (Qvarnstrom and Price, 2001, Bonduriansky and Day, 2009, Rasanen and Kruuk, 2007).

Second, there is a growing realisation that evolutionary change can occur over ecological timescales which has highlighted the need to better understand how ecological and evolutionary processes interact to drive population dynamics and demographic change (Coulson et al., 2010, Stockwell et al., 2003, Olsen et al., 2004, Bassar et al., 2010, Carroll et al., 2007, Coulson et al., 2006, Ellner et al., 2011, Ezard et al., 2009, Hairston et al., 2005, Schoener, 2011, Pelletier et al., 2007, Pelletier et al., 2009).

Teasing apart parental, plastic, ecological and reversible responses from evolved and irreversible responses of life-histories to environmental change is inherently problematic, as it is rarely possible to study parental environment effects, genetics, life histories and population dynamics simultaneously and in sufficient detail (Coulson and Tuljapurkar, 2008, Coulson et al., 2010, Morrissey et al., 2012, Andersen and Brander, 2009a, Andersen and Brander, 2009b, Bonenfant et al., 2009, Darimont et al., 2009, Becks et al., 2012, Ozgul et al., 2009, Ozgul et al., 2012, Uller, 2008). However, this is exactly what is required to understand how, or even if, populations will be able to respond to rapid anthropogenic environmental stressors such as selective harvesting (Andersen and Brander, 2009a, Andersen and Brander, 2009b, Coltman et al., 2003, Kinnison et al., 2009, Law, 2007, Ezard et al., 2009, Browman et al., 2008), the potential for species to respond to environmental change through evolution (Bell and Gonzalez, 2009, Ezard et al., 2009, Stockwell et al., 2003), and the role that parental effects have in those adaptive responses to environmental change (Uller, 2008).

Our research with an invertebrate model system has gone some way towards understanding the role of parental environments, and the significance of plastic responses and rapid evolution in delimiting individual phenotypic variation. Here we describe how we have approached these challenging questions by presenting our conceptual framework of eco-evolutionary population dynamics (**Figure 1**), and reporting on what progress we have made in determining each process within this framework. To this end we review previously published material, and report new results from ongoing empirical studies. We use our findings to identify new avenues for research necessary to properly understand how contemporary, historical, and evolutionary determinants of individual life histories interact to shape population level responses.

2.   Aims and scope

The aim of this chapter is to introduce the mite model system, a soil invertebrate microcosm based experimental system, and show how it has been used to test and develop our understanding of individual phenotypes, how they form, and how they scale up to population dynamics (i.e. **Figure 1**). We will begin by introducing our study organism, its general biology and the various experimental methods we have used to explore individual and population biology (section 3). In section 4 we will review our previously published work on the development of individual phenotypes as a function of resource availability. This has been a key empirical proof-of-principle of the L-shaped reaction norms predicted to arise when developmental thresholds determine age and size at maturity (Day and Rowe, 2002). Again referring to our published works, using this L-shaped age and size at maturity reaction

98   norm as a background measurement, we will describe our current understanding of when and how

99   parental environments shape offspring phenotypes. The role of non-genetic inheritance of parental

100   traits is important in the development of our later arguments that describe how current and historical

101   environmental effects interact with natural selection to create eco-evolutionary population dynamics.

102   If, and how, parental effects manifest themselves beyond effects on individual offspring will be

103   presented in section 5. Here we will present our published work on the magnitude and longitude of

104   detectable effects of ancestral environments on soil mite population dynamics.

105   In section 6 we will present a new analysis of how selection on individual phenotypes, caused by

106   feedbacks from population dynamics in the form of strong density dependent competition, leads to the

107   evolution of population dynamics. This extends the analysis of soil mite populations living in

108   periodically fluctuating resource environments and subject to experimental harvesting (Cameron et al.,

109   2013). Here we are able to present data across constant, randomly variable and periodically variable

110   resource environments. Crucially, it is the imposition of experimental harvesting that reveals that the

111   environmental variation is important in the evolutionary responses of populations to environmental

112   change. Finally, in section 7 we summarise what we have presented in the form of previously

113   published and new analyses and discuss how the different routes we have found to influence

114   population dynamics through changes in individual phenotypes might interact. The overall scope of

115   this contribution therefore, is to stress that it is by understanding how the different routes that lead to

116   phenotypic variation interact that we will come to a more than conceptual understanding on eco-

117   evolutionary population ecology.

118

3.   Model system and methods

120   The soil mite *Sancassania berlesei* (Michael) is common in soil, poultry litter, and stored food

121   products. Populations of *S. berlesei* have been collected from a variety of sources in different years

122   since 1996 and have been kept in separate stock lines ever since (stock cultures kept in 10 cm diameter

123   containers maintained at 24°C in unlit incubators, number c1–2.5×10^5 individuals).

124

125   3.1 *The mite model system and generic methods*

126   The life cycle consists of five stages, beginning with eggs (length: $0.16 \pm$ SD 0.01mm), continuing

127   through a six-legged larvae (length: 0.22 ± 0.01mm), a protonymph, tritonymph, and then to adulthood

128   (female length at maturity: 0.79 ± 0.17mm, range 0.47 (low food) to 1.17 (high food), n = 64; males:

129   0.72 ± 0.11mm, range 0.55 (low food) to 1.02 (high food), n = 39). As indicated by the standard

130   deviations of the adult lengths, there is considerable variation in the life history and much of it is

131   governed by intake rates of food (Plaistow et al., 2004). An individual's intake rate is a function of a

132   number of factors: population density, stage structure, and the amount of food supplied and its spatial

133 configuration; together these factors create the individual's competitive environment (Benton and
134 Beckerman, 2005)

135 Eggs hatch 2 to 5 days after being laid. Juveniles can mature from as little as 4 to 50+ days after
136 hatching (Beckerman et al., 2003), depending on food and density. The longevity of the adults can also
137 vary from ç10 to ç50 days. Thus, total longevity varies from 3 weeks (high food, low density) to 7+
138 weeks (low food, high density). Fecundity is related to resources, and so to body size, and to survival.
139 The relationship between fecundity and the growth-survival trade-off is in itself dependent on
140 resources (Plaistow et al., 2006, Plaistow et al., 2007).

141

142 *3.2 General Experimental Procedures*

143 Generally, mite cultures are supplied food in the form of powdered or granulated yeast. Different
144 feeding regimes were used in different experiments and consisted of controlled feeding of balls or rods
145 of dried baking yeast, filtered to minimise variation in their size (diameter of 1.25-1.40 mm for
146 standard size balls). Experimental vessels are either glass tubes (20mm in diameter and 50mm in
147 height) or small non-static plastic vials (3-7ml). These are half-filled with plaster of Paris, which,
148 when kept moist, maintains humidity in the tubes. The tops of the tubes are sealed with a circle of
149 filter paper held in place by the tubes' cap with ventilation holes cut into it. For some shorter
150 experiments (24hr) the plastic vials were sealed with clingfilm. For population experiments, the mites
151 are censused using a Leica MZ8 binocular microscope and a hand counter. In each tube, a sampling
152 grid is etched into the plaster surface to facilitate more accurate counting and observation. All adults
153 are counted in the tube, but juveniles and eggs are counted in a randomly chosen quarter.

154 *3.2.1 Common garden environments*

155 Common garden tubes were used to both standardise and manipulate parental and offspring
156 environments prior to carrying out life history assays or population dynamic experiments. A common
157 garden was created by placing standardised numbers of eggs (from either stock culture females or
158 experimental animals) into identical tubes with controlled food access/competitor density and rearing
159 them until maturation. Upon maturation these individuals are paired and either placed in a new
160 common garden or in egg laying tubes for the collection of eggs for life history assays, reproduction
161 allocation measurements or population dynamic experiments i.e. (Plaistow and Benton, 2009, Plaistow
162 et al., 2004).

163

164 *3.2.2   Life history assays*

165 Life history assays are used to quantify the life history or phenotype of an individual, full-sib family or
166 population from a given treatment. Life history assays are conducted by placing individuals or groups

167 of random or full-sib eggs in a small vial that is half-filled with plaster (7-20ml plastic or glass vials).
168 These individuals are observed daily, either with density being standardised by replacement of dead
169 individuals or not. At maturation, individuals are photographed for later measurement and then
170 removed from the vial. We can collect data on age and size at maturity, fecundity at maturity or any
171 other stage of development (e.g. egg size, hatching, protonymphs). Reproductive allocation is a
172 measure of the differences between mite eggs laid by mothers from different parental environments
173 i.e. (Plaistow et al., 2007). We have measured reproductive allocation in terms of numerical, (e.g. total
174 eggs, eggs-at-age), physical (e.g. length, volume) and biochemical properties of eggs laid (e.g. total
175 protein). Measurements of individuals and eggs are made from digital images captured from the
176 microscope (e.g. Leica MZ8, Nikon SMZ15) and measured using ImageJ 1.28u
177 (http://rsb.info.nih.gov/ij) or Nikon Elements D software (v3.2 64bit).

178

3.2.3 Population dynamic experiments

180 Population dynamic experiments involve monitoring free-running populations over multiple
181 generations. Such experiments have been started in different ways depending on the purpose of the
182 experiment. Where the purpose was to investigate the timescale of parental effects, populations were
183 started with controlled numbers of eggs from parents of different environmental backgrounds or ages
184 (Plaistow et al., 2006, Plaistow et al., 2007, Pinder, 2009). To investigate the interplay between
185 population and phenotypic dynamics, populations were initiated with a mix of sexed adults (n=75-
186 150/sex) and juveniles (n=500-1000), approximately at stable stage distribution to minimise transient
187 dynamics. To investigate the links between ecological plasticity and life history change, populations
188 were initiated with mites recently collected from the wild to maximise genetic diversity (n=150 adult
189 /sex and 1000 juveniles).

190

191 In the population experiments, we have often manipulated stochasticity by varying the timing and
192 amount of food supplied, while trying to maintain other factors as close to constant as possible. Our
193 rationale for this is that many natural environmental factors will either vary the absolute food supply
194 (e.g., the weather), the requirement for food (e.g., temperature), or the availability of food (e.g.,
195 patchiness, territoriality, inter-specific competition). Each treatment supplied food at the same mean
196 daily rate (equivalent to one or two balls of yeast per day), but at a variable amount on different days.
197 The algorithms we developed were to supply balls of yeast randomly, or periodically, within each
198 window of time, such that over repeating window lengths, the cultures received a constant number of
199 balls of yeast. Other populations were maintained on constant food regimes either to act as contrasts to
200 those in the variable environments, or on their own for some parental effect experiments. Effects of the
201 different distributions of food supply on variation in population abundance are described elsewhere
202 (Benton et al., 2002).

4. Within and between individual phenotypic variation

In this section we review our previously published work explaining how environment induced changes in the growth rate and maturation decisions are responsible for generating a L-shaped age and size at maturity reaction norm. We then summarise our previously published work explaining how variation in age and size at maturity alters the provisioning of individual offspring and the developmental environment of those same offspring, leading to intergenerational phenotypic variation.

4.1 Age and size at maturity reaction norms

Population growth rates are intrinsically linked to the trade-off between the age and size at which individuals mature because age at maturity determines how quickly individuals start to reproduce and because fecundity is often closely associated with age and body size (Roff, 2002, Plaistow et al., 2006, Plaistow et al., 2007). Consequently, understanding how populations respond to environmental change is likely to depend upon how individuals, within those populations, respond to environmental change. Organisms that live in variable environments, due to environmental forcing or density dependence, for example, are expected to evolve plasticity in age and size at maturity because of fluctuations in resource availability (DeWitt et al., 1998, Via et al., 1995). We demonstrated that in soil mites, the trade-off between age and size at maturity is extremely plastic in response to food availability. Offspring reared on high food matured five times faster and at double the body size of offspring reared in a poor food environment. Moreover, the age and size at maturity reaction norm is L-shaped (Plaistow et al., 2004)**(Figure 2)**. This pattern arises because an individual's decision to mature is controlled by a developmental threshold, which is the minimum size below which maturation cannot occur (Day and Rowe, 2002). Fast growing individuals in good food environments overshoot the minimum threshold size considerably by the time maturation is complete. In contrast, slow-growing individuals in poor food environments have to delay maturation until the minimum threshold size is reached. Consequently, in good food environments all individuals mature at young age but individual differences in growth rates translate into variation in size at maturation. In contrast, in poor food environments, all individuals mature at the same minimum threshold size but individual differences in growth rates translate into differences in age at maturity (Plaistow et al., 2004). As we will see later, this fundamental difference in how environmental variation is translated into phenotypic variation has important implications for understanding how individual plasticity influences population dynamics.

4.2 Intergenerational parental effects on individual phenotypic variation

Parental effects are defined as any effect that parents have on the development of their offspring over and above directly inherited genetic effects (Uller, 2008). Two types of mechanisms can be involved in the transmission of parental effects to offspring phenotypes. In the first mechanism, parental effects

242   can arise from alterations of the developmental environment experienced by offspring through

243   variation in allocation of non-genetic resources such as nutrients, e.g. (Benton et al., 2005, Plaistow et

244   al., 2007), immune factors, e.g., (Hasselquist and Nilsson, 2009) and hormones e.g., (Meylan et al.,

245   2012). Traditionally, studies of environmental parental effects have focused on maternal influences on

246   her offspring's developmental environment because, in most species, females invest more resources in

247   offspring than males. However, a few examples of paternal effects arising from variation in food

248   provisioning, e.g. (Isaksson et al., 2006) and transmission of immune factors, e.g. (Jacquin et al., 2012,

249   Roth et al., 2012) exist in the literature. In addition, females can alter their investment in offspring in

250   response to males' characteristics, e.g. (Pinder, 2009, Gil et al., 1999), leading to indirect paternal

251   effects. In the second mechanism, parental effects can arise from alterations of gene expression

252   through epigenetic modifications of regulatory regions of the genome in the germline, for instance

253   mediated by DNA methylation and histone modifications, and without changes in DNA sequences

254   (Bonduriansky and Day, 2009). Transgenerational inheritance of epigenetic modifications have been

255   suspected to be involved in some parental age effects, e.g., (Bonduriansky and Day, 2009, Perrin et al.,

256   2007), in some heritable disorders, e.g. (Champagne, 2008, Olsen et al., 2012), and, more generally in

257   paternal effects transmitted through variation in allocation of non-genetic resources, e.g. (Rando,

258   2012). In addition, there is increasing evidence that maternal and paternal effects arising from

259   variation in offspring's provisioning or from epigenetic modifications are context-dependent, e.g.

260   (Badyaev and Uller, 2009), and can interact to shape offspring phenotype, e.g. (Ducatez et al., 2012).

261   In soil mites, we have explained how age and size at maturity is critically dependent on food

262   availability in the offspring's current environment (Plaistow et al., 2004). However, we have also

263   demonstrated how variation in the maternal provisioning of offspring and the age of the mother can

264   influence both offspring growth rates (Plaistow et al., 2006) and their decision to mature (Benton et

265   al., 2008). In this contribution, we are specifically dealing with the first mechanism described above

266   (i.e. alterations of the developmental environment). Consequently, individual variation in

267   developmental or somatic growth is not just a result of the environment that the individual

268   experiences, but also the environment experienced by its ancestors e.g. (Pinder, 2009) **(Figure 3a)**.

269   From a population dynamic perspective, these effects are important because they mean that a

270   population's response to environmental change may be time-lagged to some degree, with

271   intergenerational effects operating as a source of intrinsic delayed density dependence (Beckerman et

272   al., 2002, Rossiter, 1994).

273

274   *4.3 Understanding the context dependence of parental effects*

275   Our results have suggested that the importance of parental environments for the variation of offspring

276   phenotypes in soil mites is trait-dependent and may be highly context-dependent (Beckerman et al.,

277   2006, Plaistow et al., 2006). For instance, in low-food current environments, variation in egg size

278   produced by different parental food environments altered the trade-off between age and size at

279   maturity, but had little effect on the size of eggs produced in subsequent generations. Consequently,

280   the variation in egg size that affected intergenerational effects decreased over time. In contrast, in

281   high-food environments, variation in egg size predominantly influenced a trade-off between fecundity

282   and adult survival and generated increasing variation in egg size (**Figure 3b**). As a result, maternal

283   effects transmitted through variation in egg provisioning persisted and we have observed great grand-

284   maternal effects on descendant's life histories (Plaistow et al., 2006). We therefore predicted that the

285   persistence and significance of intergenerational effects for population dynamics would itself be

286   context-dependent. However, it is important to realize that in an eco-evolutionary sense 'context' is

287   itself something that is derived from the traits and maternal strategies that have evolved in the

288   population.

289   In viscous populations with overlapping generations, mothers and offspring are forced to compete for

290   the same resources and may, therefore, directly influence each other's probability of survival and

291   future reproductive success. The close covariation between the quality and number of offspring

292   produced and maternal survival means that any change in one offspring provisioning trait may have

293   consequences for the others (Beckerman et al., 2006). It is necessary, therefore, to understand how

294   females change their offspring provisioning strategy as a whole (e.g. egg numbers, egg size, maternal

295   survival) in order to interpret the adaptive significance of maternal responses to changes in their

296   environment. We have shown that in soil mites, offspring provisioning strategies are dynamic,

297   switching from investment in many small eggs in young females to fewer, better provisioned eggs in

298   older females (Plaistow et al., 2007). This strategy may be adaptive if it increases the survival of

299   younger offspring that must compete with older, larger siblings that had been laid previously. This

300   age-related dynamic shift in egg provisioning was greater in high food environments in which females

301   lived longer, creating a greater asymmetry in offspring competitive abilities. Such conditions are likely

302   to be common in an opportunistic species such as soil mites that have evolved a life history that

303   specializes in strong competition between individuals exploiting patchily distributed resources, such as

304   carcasses and dung (Houck and Oconnor, 1991). In the following section we examine the effects that

305   these complex environmentally driven parental effects have on patterns of population dynamics.

306

307   **5.   From phenotypic variation to population dynamics**

308

309   Parental effects may be especially important from a population dynamic perspective because they

310   generate a lag in the response of a population to an environmental change (Beckerman et al., 2006,

311   Beckerman et al., 2002, Benton et al., 2005). This could make it harder to predict changes in

312   population size, but may also theoretically lead to long-term deterministic population dynamic

313   patterns, such as population cycles (Ginzburg, 1998, Ginzburg and Taneyhill, 1994, Inchausti and

314   Ginzburg, 1998). Consequently, we have been interested in how parental effects might influence

315   population dynamics (Benton et al., 2001). This is not easy to study in the wild, or in many laboratory

316   systems, due to the difficulty of measuring parental effects and following population dynamics in

317   sufficient demographic detail. However, it is possible in the soil mite system because replicated

318   populations can first, be initiated with different numbers of eggs, changing the initial environment

319   experienced by offspring; but also initiated with eggs from different types of mothers, enabling us to

320   experimentally manipulate parental effects e.g. (Benton et al., 2005, Benton et al., 2008, Plaistow and

321   Benton, 2009).

322

323   *5.1 Transient population dynamics and parental effects*

324   In the first of these types of experiments, all replicated populations were initiated with 250 eggs.

325   However, half the populations were set-up with large eggs from mothers experiencing low food, the

326   other half were set-up with small eggs from well-provisioned mothers (see Benton et al., 2005 for

327   details). This manipulation of the maternal effect alone was sufficient to generate differences in the

328   transient population dynamics of the populations that were still present after three generations, even

329   though the populations were experiencing the same constant environment with respect to the food

330   supplied to them each day. Such deviations in population dynamics arise because differences in the

331   hatching success, growth rate, size and fecundity and survival in the initial cohort generate differences

332   in the competitive environment experienced by offspring produced in the second cohort. Changes in

333   the competitive environment creates further phenotypic variation between individuals from the two

334   treatments that ultimately leads to large differences in the population dynamics of the populations

335   sustained over multiple generations (Benton et al., 2005).

336   In a second experiment, but this time using similarly sized eggs that either came from young (3 days)

337   or old (9 days) mothers, the effects on transient population dynamics again lasted three generations

338   (Benton et al., 2008) (**Figure 4**). The results clearly demonstrate that deterministic differences in eggs,

339   which are not obviously related to their size, and so may be undetectable in a population setting, may

340   have a significant effect on population dynamics. Comparing these two experiments, the effects of

341   parental background or age were of a similar magnitude. However, as we discussed earlier, our

342   individual-level studies of maternal effects in soil mites suggested that the exaggeration and the

343   transmission of maternal effects from one generation to the next increased in high-food environments,

344   but decreased in low-food environments (Plaistow et al., 2006). Consequently, we hypothesized that

345   maternal effects would be more likely to persist, and have a bigger influence on population dynamics,

346   in high-food environments compared to low-food environments. In order to test this hypothesis we

347   created maternal effects by initiating populations with eggs from young mothers or old mothers but we

348   also simultaneously manipulated the initial resource environment by changing the initial density from

349   high (500 eggs, low food) to low (50 eggs, high food) (see Plaistow *et al*., 2009 for details). The

350   results clearly supported our hypothesis that the importance of maternal effects for population

351   dynamics is context-dependent. An influence of maternal age treatment on both population and egg

352   and body-size dynamics was only observed in the populations initiated under low density rather than

353   high density (Plaistow and Benton, 2009).

354   In summary, we have explained how an interaction between current and historical maternal states

355   (transmitted as parental effects) interact to shape patterns of individual phenotypic variation (e.g. size-

356   at-hatch, growth rate to maturity, size-at-maturity, offspring's own egg provisioning patterns) and how

357   this phenotypic variation is then translated into fluctuations in population size. Understanding the

358   various factors that can determine such fluctuations is crucial for predictive modelling of populations

359   for management purposes. From an eco-evolutionary perspective, it is also critical because it is those

360   fluctuations in the number, size and age structure of populations that determine the temporal resource

361   heterogeneity that ultimately shape how individual traits and life history strategies evolve (Roff,

362   2002). In the following section we summarise our current understanding of how differences in

363   temporal resource heterogeneity, created by environmental variation and harvesting, influence the

364   evolution of mite life histories and, in turn, how this evolution influences population dynamics.

365

6. Eco-evolutionary population dynamics – the full loop

367

368 Debate on the role of genetic change in ecological dynamics is not new (Lenski, 1984, Pimentel, 1961,

369 Pimentel et al., 1978, Pimentel and Stone, 1968, Wilcox and Maccluer, 1979), and includes predictions

370 of cyclic consumer-resource dynamics caused by evolution (Lenski, 1984, Abrams and Matsuda,

371 1997). It is only more recently that the search for the role of the gene in ecology has been termed "eco-

372 evolutionary dynamics".

373 It has largely been assumed that this emerging field of eco-evolutionary dynamics has demonstrated

374 that evolutionary "loops" exist in nature, where loops are defined as genetic selection pressures placed

375 on populations from ecological interactions that have significant effects on population dynamics,

376 additive to that of the ecological interaction itself (Kinnison and Hairston, 2007). For example, while a

377 predator can reduce population growth by killing individuals, does it have an additional detectable

378 effect on prey population growth rate by causing the average somatic growth rate to maturation to

379 evolve? Such an evolutionary response of the prey life history, causing a feedback to prey population

380 dynamics, and subsequently predator dynamics would be an evolutionary loop (Post and Palkovacs,

381 2009).

382 There is however a dearth of robust empirical evidence for such evolutionary loops. An early study by

383 Nelson Hairston Jr. described the pattern of rapid evolution of toxin resistance in *Daphnia galeata* in

384 Lake Constance in response to eutrophication (Hairston et al., 2001, Hairston et al., 1999). While not

385 evidence of a loop *per se*, the Lake Constance study led to a series of experiments on zooplankton-

386 phytoplankton interactions that demonstrated that rapid evolution in response to an ecological

387 interaction can alter predator-prey cycles (Yoshida et al., 2003), that rapid evolution can mask

388 interactions normally identified through changes in predator and prey abundance (Yoshida et al., 2007)

389 and that rapid prey evolution can affect predator dynamics more than changes in prey abundance

390 (Becks et al., 2012). Other studies on microcosm based asexual communities have followed to show the

391 generality of the importance of rapid evolution on ecological dynamics e.g. (Friman et al., 2014).

392   A common thread across all these aquatic predator-prey studies, with few exceptions e.g. (Fussmann et

393   al., 2003), is the evolution of traits associated with either defence from predators or digestion of prey.

394   This is clearly important in a community setting, but it is difficult to make the jump from proof of

395   principle in these systems to studies that consider the role of environmental change (e.g. trends in mean

396   annual temperature) or high rates of harvesting against life history traits such as somatic growth rate in

397   well-studied populations of fishes, birds and mammals (Darimont et al., 2009). Other differences

398   between demonstrated eco-evolutionary dynamics in freshwater microorganisms and proposed eco-

399   evolutionary dynamics in larger animals exist, not least of which is asexual vs. sexual reproduction and

400   *more* complex life histories based on significant growth from birth. Experimental studies have shown

401   that rapid life history evolution in vertebrates is possible, through response to selection caused by

402   predation (Reznick et al., 1996) and harvesting (van Wijk et al., 2013), but trait change from selection

403   on vertebrates in itself is not an eco-evolutionary loop. Analyses of empirical data demonstrates that

404   eco-evolutionary feedback from an environmental change to population dynamics could explain

405   observed trait distributions and population sizes (Coulson et al., 2010, Ozgul et al., 2010, Ozgul et al.,

406   2012), but this generally lacks evidence of genetic selection, but see similar studies of trait demography

407   in birds (Charmantier et al., 2008, Nussey et al., 2005). Other studies have identified where eco-

408   evolutionary dynamics are likely to occur, for example by demonstrating how changes in selection have

409   led to changes in animal behaviour and/or distribution (Strauss et al., 2008). Fewer studies, however,

410   have been able to manipulate the eco-evolutionary loop in more complex organisms and ask what role

411   ecological conditions have on selection on traits, and does this trait change feed-back to influence

412   population dynamics (Cameron et al., 2013, Walsh et al., 2012).

413   The role of predation in life history evolution has long been recognised (Law, 1979, Michod, 1979,

414   Reznick, 1982, Stenson, 1981), and remains a contemporary interest (Beckerman et al., 2013). There

415   has been a fever of interest in the role of high rates of trait-selective exploitation on shifts in the trait

416   distributions of many harvested animal populations, in particular of body size or age and traits that

417   would otherwise be under sexual selection, such as male ornamentation (Biro and Post, 2008,

418   Bonenfant et al., 2009, Bunnefeld et al., 2009, Ciuti et al., 2012, Darimont et al., 2009, Hamilton et al.,

419   2007, Milner et al., 2007, Olsen et al., 2009, Pelletier et al., 2007, Coltman et al., 2003). There has also

420   been a concomitant interest in the role that these shifts in trait distributions may play in eco-

421   evolutionary dynamics (Coulson et al., 2006, Coulson et al., 2010). In those animal species that we

422   exploit at some of the highest rates, specifically the marine and freshwater fishes, there is an ongoing

423   debate about the mechanisms that lead to these shifts in body size distributions (Andersen and

424   Brander, 2009a, Andersen and Brander, 2009b, Anderson et al., 2008, Browman et al., 2008, Kinnison

425   et al., 2009, Kuparinen and Merila, 2007, Kuparinen and Merila, 2008, Law, 2007). There are several

426   more robust explanations for reduced mean body size-at-age in exploited fishes including body

427   condition effects (Marshall and Browman, 2007), size structured community interactions (De Roos et

428   al., 2003, Persson et al., 2007, Van Leeuwen et al., 2008, Anderson et al., 2008), and fisheries-induced

429   evolution (Jorgensen et al., 2007). Intuitively these more prominent explanations are not mutually

430   exclusive and have each been more plausible an explanation for responses to harvesting in different

431   case studies. Here, we will investigate the role of evolutionary responses of phenotypes to

432   exploitation, and in particular to stage-selective harvesting.

433

434   Stage-selective harvesting, occurring at times of the year or in places where particular life history

435   stages dominate the harvest (e.g. adult Barents Cod at spawning ground), or where there are other

436   stage-based vulnerabilities in likelihood of harvest mortality (e.g. in cryptic selection of hunted birds

437   (Bunnefeld et al., 2009), or killing only adults or juveniles of pest species) is predicted to lead to shifts

438   in growth rate to maturity that are distinct from size-selection harvesting. Here it is expected that life

439   histories will evolve such that individuals who minimise their time in the most vulnerable stages will

440   be selected for (Stearns, 1992). So we expect that harvesting of juveniles will lead to faster

441   developmental growth to maturity, while harvesting adults will reduce developmental growth via a

442   trade-off with increased juvenile survival and adult fecundity (Ernande et al., 2004).

443

444   Previous investigations with soil mites in seasonal environments where we exposed populations to

445   adult or juvenile mortality resulted in statistically different growth rates to maturity in harvested

446   populations, and compared to unharvested populations, the shifts in growth rate were exactly as

447   predicted by theory (Cameron et al., 2013). Here we extend this analysis to the evolved responses of

448   growth rate to maturity when harvesting juveniles or adults across constant, random and periodic

449   environments. Mite populations were harvested at a rate of 40% per week (proportional harvest) or as

450   an additional threshold harvest treatment in randomly variable environments of all adults above 60%

451   of the long term adult population size. We estimated these rates to be close to the maximum soil mite

452   populations can sustain without collapsing (Benton, 2012). We report the life history results on Low

453   food conditions as we assume this is most representative of the conditions in long term experimental

454   populations e.g. (Cameron et al., 2013).

455

456   In summary of this introduction we present new empirical data from the mite model system where we

457   have investigated the role evolution plays in the contemporary responses of population dynamics to

458   environmental change. We will summarise our main finding on the role of phenotypic evolution on

459   population responses to highly competitive environments and building on this we will discuss the roles

460   of environmental variation (i.e. variation in food availability) and harvesting on the development of

461   the eco-evolutionary feedback loop.

462   *6.1 Methods*

463   Soil mites were collected from several wild populations and allowed to mate for two generations in the

464   laboratory before being placed in our standard microcosm population tubes (see section 3)(Cameron et

465   al., 2013). Sixty populations were started with 150 of each sex of adult and approximately 1000

466   juveniles in order to minimise transient dynamics. Each population received the same average access

467   to resources of 2 balls of yeast per day, but was randomly assigned to one of three experimentally

468   induced levels of resource variability (i.e. environmental variation): constant (replicates (n) =18);

469   periodically variable (n=18) and randomly variable (n=24). The periodically variable treatment was

470 designed to represent seasonality as best as possible by having a 28 day cycle e.g. (Cameron et al.,

471 2013). The randomly variable treatment was designed to be entirely unpredictable with daily food

472 provisions being chosen from a random distribution with mean of two balls over a 56 day window,

473 with a maximum daily provision of 12 balls (Benton et al., 2002). The mite populations were censused

474 each week for 2 years, where a generation is approximately 5 weeks (Ozgul et al., 2012).

475

476 From week 13 to 83 the populations from each environmental variation treatment were subjected to a

477 factorial stage-structured harvest treatment where: populations were either unharvested; juveniles were

478 proportionally harvested (where 40% of juveniles were removed each week) or adults were

479 proportionally harvested (where 40% of adults were removed each week). In the randomly variable

480 treatment there was an additional treatment of a threshold adult harvest, sometimes called a fixed-

481 escapement harvest (Fryxell et al., 2005), where all adults above 60% of the long term mean number

482 of adults were removed. This number was set to 176 adults based on 60% of the long term mean adult

483 population size from previous studies on the same mean resources (Benton and Beckerman, 2005).

484 Threshold harvest strategies have been said to be more conservative in affecting the variance in

485 population size and therefore minimise extinction risks to harvested populations (Lande et al., 1997),

486 but such claims have not been tested experimentally in variable environmental conditions.

487

488 In tandem with the population census, we conducted less frequent common garden life history assays

489 to measure the development to maturation of seven full sib families for two of the six replicate

490 populations per treatment combination. For the common garden, 100 juveniles were removed from

491 populations and reared to the F2 generation on fixed per-capita resources to standardise parental

492 effects e.g. (Plaistow et al., 2006). Single F2 male-female pairs were allowed to mate and their eggs

493 were collected. Twenty offspring from each pair were each reared collectively in either High or Low

494 food resource availability. Only the results from the Low food life history assay will be presented in

495 this paper as this was found to best represent the competitive conditions in experimental populations.

496 Age (days) and body sizes (body length in mm) at maturity were recorded for each adult individual of

497 each sex. Daily survival rates until maturity of the cohort of 20 juveniles were calculated using

498 standard methods (e.g. Mayfield estimates). Fecundity at maturity was estimated for each female

499 individual using a linear regression of the age and size at maturity with cumulative fecundity from day

500 3-7 post eclosion from existing data (Plaistow et al., 2006, Plaistow et al., 2007). These data led to

501 average trait values representing family and treatment phenotypes.

502

503   Twenty four adult females per population were sampled from the common garden F3 generation in

504 weeks (i.e. time-points) 0, 18, 37, 63 and 95 and their genotype characterised using amplified fragment

505 length polymorphisms (AFLP). The assay used 299 loci and the methodology has been described in

506 detail elsewhere (Cameron et al., 2013), but here incorporated the constant, periodic and random

507 environmental variation treatments.

508

509 *6.1.1 Quantitative methods and statistical analysis*

510   Life history trait data on age and size at maturity are presented in the text as full-sib female or

511   treatment means with standard deviations at the beginning (week 0) and end (week 95) of the

512   experiment (e.g. Plaistow et al. 2004). Statistical differences in daily Mayfield survival estimates

513   between environmental or harvesting treatments was most appropriately tested using a generalised

514   linear model with a quasipoisson error distribution. Significance of treatments was tested while

515   correcting for the highly overdispersed distribution using F tests (Crawley, 2007). The significance of

516   environmental variation and harvesting treatments on the mean female phenotype and the age and size

517   at maturity of each family per treatment at the end of the study was assessed using MANOVA to

518   jointly model log(age) and log(size) in Low food conditions while controlling for population density in

519   the life history assay tubes by using tube covariates (weighted density, median density and total tube

520   survival), see Cameron et al. (2013). Owing to the extra threshold harvest treatment in random

521   variation treatments, a full model was first built without this one treatment to independently test for an

522   environment*harvest interaction. Following this, and for predictions of treatment means, a separate

523   MANOVA was built for each environmental variation treatment. Age and size at maturity trait values

524   were then plotted as model predicted means with associated standard errors of the model estimates.

525

526   To test for any link between Low food phenotypic change and changes in observed population growth,

527   we estimated the mean and confidence intervals of the basic reproductive rate per treatment, R_0 ($R_0 =$

528    $\exp((\ln (l_x * m_x))/T_c$, where l_x is the chance of an individual surviving to age x, m_x is the number of

529   offspring produced during age x-1 to x and T_c is the average generation time) (Stearns, 1992). R_0 was

530   corrected by the average generation time due to the overlapping generations. For further details of this

531   method refer to supplementary material associated with Cameron et al. (2013). Average population

532   growth rate (pgr = Nt + 1/Nt) was calculated from a smoother fitted across replicate population time

533   series per treatment (observed population growth = change in total population size from week to week,

534   over a 10 week window around assay time-points), and a Pearson's correlation test between the two

535   estimates of population growth were undertaken.

536   For each environmental variation treatment, genetic diversity in age-at-maturity in a Low food assay

537   was apportioned using an analysis of molecular variation (AMOVA) approach into: 1) differences

538   among individuals within replicate populations; 2) differences among replicate populations within

539   time-points within harvesting regimes; 3) differences among time-points within harvesting treatment;

540   and 4) differences among harvesting treatments across time-points (AMOVA, Arlequin Version 3.5

541   (Excoffier and Lischer, 2010)). The relative magnitude of differences can highlight the effects of

542   deterministic and stochastic microevolution acting across the populations. It is expected that drift

543   would cause significant differences to accumulate among replicates within time-points for any

544   treatment, whereas selection would cause significant differences across time-points within a treatment

545   or among the treatments themselves.

546

547   *6.2 Results- Evolution of population dynamics in variable environments*

548   All mite populations initially declined across all three environments and then recovered (**Figure 5**).

549   Before the recovery, the mean population growth rate of the populations was 0.980 (=2% decline per

550   week), 0.978 and 0.980 at week 20 for the constant, periodic and random environments respectively.

551   During the recovery, the population growth had increased to 1.010 (= 1% increase per week), 1.013

552   and 1.012 respectively by week 60. At the start of the experiment, in low food and hence highly

553   competitive conditions, soil mites took an average of 12.3 days to mature. By the end of the

554   experiment we observed a large reduction in the growth rate to maturity of the average mite family

555   from all three environments, equating to a 35%, 76% and 83% delay in age-at-maturity in the constant

556   (16.6±2.6s.d. days), periodic (22.1±3.6s.d. days) and variable environments (21.6±4.27s.d. days)

557   respectively. The observed increasing delays in developmental growth rate over the course of the

558   experiment in resource poor conditions are positively correlated with increases in fecundity in adult

559   mites (Cameron et al., 2013, Plaistow et al., 2006, Plaistow et al., 2007). This is suggestive that the

560   delays in maturity are adaptive. There was no significant difference in daily survival rate between

561   families from the three environments (Quasipoisson GLM:$F_{env}=0.29_{2,123,}$ P>0.7). Consequently, while

562   the earlier maturation phenotype we see in constant environments would have reduced fecundity

563   compared to other environment phenotypes, this appears to be offset by increased overall survival to

564   maturity. The question of interest, that separates our experiment from only demonstrating that the

565   traits of mites change when they are placed in different laboratory environments, was to determine if

566   the change in growth rates observed were caused by selection and if that selection led to the recovery

567   of the populations after only eight generations.

568   The basic reproductive rates R_0 estimated from the common garden life history data at weeks 0, 18,

569   37, 63 and 95 were highly correlated with the average of observed population growth rates estimated

570   from replicated experimental time series (Pearson's = 0.88, $t_{2,13}$ = 4.81, P<0.001). Furthermore, there

571   is no significant difference between the estimates of population growth from life history data or the

572   time series (e.g. R_0 vs pgr, paired t-test, p=0.34). Given that the phenotype data used to estimate R_0

573   (i.e. age and size at maturity, survival to maturity, reproduction at maturity) are collected in similar

574   competitive conditions to those in the population experiments but after 3 generations in a common

575   garden environment, this is very strong evidence that we are observing evolved changes in mean life

576   history that lead to changing population dynamics; a requirement for the demonstration of an eco-

577   evolutionary feedback loop (Schoener, 2011). However, it does not prove that the phenotypic change

578   observed is being caused by genetic evolution e.g. (Chevin et al., 2010). The AMOVA analysis on

579   AFLP variation confirms that both genetic drift and selection are operating in concert to affect the

580   levels and distribution of genetic variation in growth rates within the microcosm system (**Figure 6**).

581   All of the partitions explained a significant proportion of the variation observed (e.g. more than 5%)

582   except for the difference among harvesting treatments within the constant food environment. This

583   need not reflect a lack of selection caused by harvesting acting on growth rates in constant

584   environments, but that among individual variation is likely masking its importance in this treatment.

585   This highlights that within each environmental variation treatment, genetic drift is acting to force

586   populations into different evolutionary trajectories (given that replicate populations within harvesting

587 treatments within time-points and within environments accumulated significant genetic differences). It

588 also demonstrates that selection operates to generate differences in the growth rate to maturity across

589 time-points, within harvesting regimes, in the different environment treatments as well as between

590 environments across time-points.

591

592 *6.3 Results - Life history responses to harvesting in variable environments*

593 We found a significant interaction between environmental variation and harvesting treatment on the

594 age and size at maturity (MANOVA: age-at-maturity $F_{env:har}=2.45_{4,123}$ P<0.05; size-at-maturity

595 $F_{env:har}=3.15_{4,123}$ P<0.02). To understand this interaction, and by controlling for stochastic differences

596 in mite densities between life history assay tubes, we standardised survival and density covariates to

597 the mean values per environmental treatment and predicted the mean and variance of trait values from

598 a MANOVA for each environment.   In both constant and randomly variable environments harvesting

599 adults or juveniles led to a significant delay in maturation in comparison to unharvested controls

600 (**Figure 7**, left and centre panels). This contrasts with what was observed in periodic environments

601 where harvesting juveniles reduced age at maturity in line with reducing risk of increased harvesting

602 mortality (**Figure 7**, right panel). In both constant and randomly variable environments there was no

603 significant effect of harvesting on size at maturation (constant: $F_{har}=2.25_{2,28}$ P>0.1; random:

604 $F_{har}=0.76_{3,40}$ P>0.5), unlike the small but significant increase in size at maturity in adult harvested

605 phenotypes from periodic environments originally described in Cameron et al. (2013). As we

606 discussed in the previous section, we detected a statistically significant effect of selection caused by

607 harvesting on the variation in developmental growth rates in both random and periodically variable

608 environments (**Figure 6**). It is surprising that given the clear phenotypic differences found between

609 unharvested and harvested constant environment populations at the end of the experiment, that the

610 AFLP response was not more pronounced. However, selection was observed, and this assay method is

611 a blunt tool given that we only have a snapshot of phenotype and genotype differences from a small

612 number of individuals from two of six replicate populations at the F3 generation.

613

614 *6.4 Discussion of Evolution of life histories in response to environmental variation and harvesting*

615

616 Life history research increasingly focusses on understanding the links between environmental

617 variation and population demography. Stochastic demography is a matrix based approach to estimate

618 optimum life histories that maximise fitness averaged over variable environments, when variable

619 environments lead to variation in vital rates (Caswell, 2010, Haridas and Tuljapurkar, 2005, Trotter et

620 al., 2013, Tuljapurkar et al., 2009, Tuljapurkar et al., 2003). Not all such approaches have focussed or

621 presented the same traits we have considered here, i.e. developmental growth. However, stochastic

622 demographic approaches have shown that the generation time, measured variously as cohort

623 generation time (T_c) or longevity, buffers against the negative effects of environmental variation on

624 fitness (Morris et al., 2008, Tuljapurkar et al., 2009). Shertzer & Ellner present a dynamic energy

625 budget approach that, while not strictly evolving per se, sought out optimum energy allocation

626 strategies to growth, storage or reproduction that maximised R_0 in a genetic algorithm model of a

627 rotifer population (Shertzer and Ellner, 2002). In the Shertzer & Ellner study, what is relevant is that

628 environmental variation was experienced over the time scale of an individual's lifetime, as in soil

629 mites (e.g. day-to-day variation instead of between generation or inter-annual variation). Life history

630 strategies that delayed age to maturity were optimum in more variable environments and/or

631 environments with periods of resource limitation (Shertzer and Ellner, 2002). Tenhumberg and

632 colleagues also focussed on stochastic variation in prey availability within a predators lifetime that led

633 to a negative relationship between growth rate and mortality arising from the physiological constraints

634 of 'digestion and gut capacities' in syrphids (Tenhumberg et al., 2000). The negative relationship led

635 to increased fitness of those strategies that delayed growth rate to maturity in variable environments.

636 Negative relationships between vital rates have been suggested to increase fitness in variable

637 environments in other analytical approaches (Tuljapurkar et al., 2009). In *Caenorhabditis elegans*,

638 mutants that aged slower were also found to have higher fitness in more stressful environments,

639 including when food availability was variable. This is suggested to lead to altered allele frequencies in

640 more heterogeneous environments in ecological time that feeds into evolutionary dynamics (Savory et

641 al., 2014). All these predictions fit with our main result that strong competition and more variable food

642 supply led to larger delays in maturity, which led to increased population growth rates. There is great

643 consistency therefore, across a number of empirical and theoretical approaches that the evolution of

644 slow life histories is likely in variable environments. However the relative importance of the

645 magnitude of environmental variability, its predictability or autocorrelation in the evolution of slow

646 life histories is not yet clear and should be an interesting avenue of future research.

647 While our experiment was designed to investigate potential links between phenotypic change and

648 population dynamics, it shows the potential for populations to recover from an extinction trajectory

649 through evolution: evolutionary rescue (Bell and Gonzalez, 2009). Across all three of our

650 environmental variation treatments, the initial trajectory of population growth is negative (i.e. an

651 extinction trajectory), but becomes positive after evolution in response to laboratory conditions leads

652 to delayed maturity and increased fecundity.

653 It is a key result that increased juvenile mortality can generate faster or slower life histories relative to

654 controls depending on the temporal variability in the strength of resource competition. The constant

655 and random environments produced more similar juvenile harvested mite life histories when compared

656 to the periodic treatment. While the variation in food provision in the constant and random treatments

657 was different (Coefficient of Variation (CV): zero vs. 0.36), the resulting variation in mite abundance

658 was more similar due to demographic noise in constant populations (Benton et al., 2002, Cameron,

659 Submitted)(CV_{adults}:0.20 vs. 0.34; $CV_{juveniles}$:0.46 vs. 0.50). In periodic environments the variation of

660 food provision, and therefore adult and juvenile mite abundance is much greater (CV = 0.86, 0.46 and

661 0.76 respectively). However, the greatest difference between constant, random and periodic variation

662 is that periodicity is caused by highly autocorrelated resource provisioning. We predict that this is

663 where the different life history responses to harvesting arise, in the interaction between density

dependent demographic responses to mortality and evolutionary responses to more (periodic) or less (noisy-constant and random) predictable resource pulses between harvesting events. Such interactions could increase the positive relationship between age-at-maturity and fecundity if the increase in risk of harvesting mortality from delaying maturity was less than the potential gains to lifetime fitness from receiving a glut of resources just before maturation. Theoretical understanding of the interaction between intra-generation environmental noise and selective mortality at this temporal scale is currently lacking, largely due to the taxonomic bias in evolutionary demography studies towards long lived mammals and birds.

What we have presented in section 6 by describing ecological dynamics of a wild population adapting to a controlled laboratory environment, provides a much higher level of resolution on the consequences of ecological and evolutionary interaction. We demonstrate how individuals maximise their lifetime fecundity in response to resource poor conditions, or high selective mortality and highlight how complex population dynamics can be maintained despite long term erosion of genetic diversity caused by both stochastic and deterministic processes. The latter is difficult to reconcile with classical ideas of extinction debt in conservation population genetics e.g. (Fagan and Holmes, 2006) whereby positive feedback occurs between reduced population growth rate and loss of genetic diversity that leads to an inevitable extinction. Clearly there is a need to address how evolutionary rescue can interrupt an on-going extinction vortex, and the limits to the recovery of populations in relation to extant and introduced genetic variation.

7   Summary

The aim of this contribution was to explore the complexity of the route from individual phenotypic variation to population dynamics and back again in a model system: the eco-evolutionary loop. The mite model system has provided a rich series of experiments that have highlighted the level of information on individual life histories we require to make predictions about transient population dynamics following environmental perturbations is often considerable. The study of ecology has been described as the investigation of variation in space and time of the abundance and density of organisms (Begon et al., 2005), and while demography may be a main objective of ecology, it is clear from our work and others in this volume that the proposal that all evolutionary biologists should be demographers goes both ways (Metcalf and Pavard, 2007).

We have presented the study of three distinct pathways between environments, phenotypes and population dynamics: the role of current and historical environments on offspring phenotypes; the multigenerational effects of environmentally determined phenotypes on short term population dynamics and finally the feedback between population abundance and resource availability to selection on phenotypes and evolution of population dynamics. In our diagram of eco-evolutionary interactions (Figure 1), we have represented those pathways as independent routes. It is, however,

702   clear from the context dependency of our results that the selection on life histories that determines

703   population dynamics will very much depend on the interaction between historical (parental effects)

704   and current environments (growth rate to developmental thresholds).

705   Through our demonstration that soil mite population trends are determined by their life

706   histories, which evolve in response to density dependent competition and predation (the eco-

707   evolutionary loop), we have shown that in populations in which density-dependent competition is

708   common, there is selection for individuals with life-history strategies that permit individuals to mature

709   later in low food conditions, but still retain the ability to mature early when conditions improve

710   (Cameron et al., 2013). If this is evidence of eco-evolutionary dynamics selecting for increased

711   phenotypic plasticity, it highlights the potential importance of the parental effects we previously found

712   to shape reaction norms such that selection can act on novel phenotypes e.g. (Plaistow et al., 2006).

713   Selection on more novel phenotypes would have the potential to allow more rapid feedbacks between

714   natural selection and population dynamics. This is particularly relevant in light of the interest in rapid

715   evolutionary responses to environmental change. Our current research in the mite model system is

716   examining how variation in the population dynamic patterns created in different environments

717   influences the evolution of offspring provisioning strategies and epigenetic variation in gene

718   expression during development and the effect that this has on later population dynamic patterns. This

719   should lead to a less conceptual, and more mechanistic, understanding of eco-evolutionary population

720   dynamics.

721   While we have identified much complexity, we have also shown when the role of environmentally

722   determined phenotypic variation is less important in a population dynamics context (e.g. when

723   resources are low), but it was only through experimentation that we were able to say this. This is in

724   some ways the most important conclusion of this review, that carefully planned experiments in well-

725   studied systems are what is required to separate potential consequences of eco-evolutionary dynamics

726   from those which are likely to have important consequences in natural populations.

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Figure Legends

Figure 1. A diagramatic representation of eco-evolutionary dynamics based on the results of mite model system experiments. The eco-evolutionary loop is moving between the three circled states: from (a) population structure is dependent on life history transition rates, and interacts with the environment (b) via an interaction between density depedent and independent mechanisms and parental effects to determine per capita resources (c). Per capita resources interact with genetic and environmental determinants of individual life histories (d), which leads to a closure of the eco-evolutionary loop by creating population structure. We consider here the effects of predation and harvesting as external to the loop (orange boxes and arrows), affecting the loop directly by selecting against life histories or changing population size and structure.

Figure 2. A model of the L-shaped developmental threshold model predicting growth rates to maturation along an environmental gradient of food availability (i.e. norm of reaction). This model, developed by Day and Rowe (2002), is supported by our results in the mite model system and captures the feedback caused by the interaction between population size and environmental quality on per-capita resources, and the resulting density dependent effects on individual phenotype (based on Beckerman et al. 2004, Plaistow et al. 2004).

Figure 3. A. Male age and condition influences female allocation patterns. 16 different males were mated to virgin females at each of 5 time-points during their lifetime ("time"). Males (subpanels) were well fed (males 11-18) or poorly fed (males 1-8) and are presented in the order of the two male conditions. Graphs show egg size (mm) as a function of male age. Lines are fitted values from mixed effects' model. Time, food and male are all significant. Virgin females mating with "prime" males (time class 3) laid larger eggs (Pinder, 2009). **B.** Vector plots of the factor loadings from a factor analysis of parental effects (variation in egg length) between life history traits for individuals reared in high- or low-food current environments. In high current food environments, variation in egg length predominantly influenced a negative trade-off between fecundity and adult survival and had little effect on recruitment or age and size at maturity. In contrast, in low-food environments variation in egg length translated into differences in the probability of recruiting and variation in age and size at maturity. Modified from Fig. 4 in Plaistow et al. 2006 with the kind permission of University of Chicago Press.

Figure 4. The intergenerational effects of variation in parental investment in offspring on population dynamics. The graphs show the transient dynamics of populations initiated with eggs that were laid by either younger 3 day old (white points) or older 9 day old mothers (black points). The error bars represent bootstrapped 95% confidence intervals. The individual cohorts are marked approximately on the figures as F1, F2 and F3 and were identified by inspection of the age-structured dynamics. Modified from Benton et al. 2008 with permission from Wiley and the British Ecological Society.

Figure 5. Mean age and size at maturity of full-sib females (top panel), and of harvesting treatment means and twice standard error bars predicted from MANOVA when controlling for differences in tube densities (bottom panel). Panels represent constant (left panels), randomly variable (centre panels) and periodically variable resource environments (right panels). Colours represent juvenile (green), adult (red), threshold adult (orange) and unharvested harvesting treatments (black).

Figure 6. Analysis of molecular variance for 299 AFLP loci for (black) differences among individuals within replicate populations; (back hatching) differences among replicate populations within time-points; (forward hatching) differences among time-points within harvesting regimes; (waves) differences among harvesting regimes. * indicates statistical significance of treatment group at $P<0.05$.

Figure 7. Adult population size ($\pm95\%$CI) from GAM fits across a 5 week centred moving average of replicate weekly counts per treatment (6 d.f., minimum model across all environments). All other stage

1085   counts show a similar pattern of initially decreasing in abundance then increasing. Arrows at weeks 13

1086   and 83 mark start and end of harvesting period respectively.