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Human influences on rates of phenotypic change in wild animal populations

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Abstract

Human activities can expose populations to dramatic environmental perturbations, which may then precipitate adaptive phenotypic change. We ask whether or not phenotypic changes associated with human-disturbed (anthropogenic) contexts are greater than those associated with more 'natural' contexts. Our meta-analysis is based on more than 3000 rates of phenotypic change in 68 'systems', each representing a given species in a particular geographical area. We find that rates of phenotypic change are greater in anthropogenic contexts than in natural contexts. This difference may be influenced by phenotypic plasticity — because it was evident for studies of wild-caught individuals (which integrate both genetic and plastic effects) but not for common-garden or quantitative genetic studies (which minimize plastic effects). We also find that phenotypic changes in response to disturbance can be remarkably abrupt, perhaps again because of plasticity. In short, humans are an important agent driving phenotypic change in contemporary populations. Although these changes sometimes have a genetic basis, our analyses suggest a particularly important contribution from phenotypic plasticity.

Keywords: contemporary evolution, Darwins, Haldanes, invasion, microevolution, rapid evolution

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Introduction

Natural populations are at least partly adapted to their local selective environments (Endler 1986; Rose & Lauder 1996; Schluter 2000). At any particular location, however, the intensity and direction of selection will fluctuate through time, making adaptation an ongoing and ever-changing necessity. This dynamic nature of adaptation has now been confirmed through numerous demonstrations of apparently adaptive phenotypic change on timescales as short as a few generations (reviews: Hendry & Kinnison 1999; Kinnison & Hendry 2001; Palumbi 2001; Reznick & Ghalambor 2001; Stockwell *et al*. 2003; Hairston *et al*. 2005). A remaining question, however, is whether or not adaptive phenotypic change can keep pace with the increasingly rapid and dramatic changes in selection that characterize our world. Our goal is to gain some insights into this question by examining phenotypic changes in populations experiencing environmental change, with the 'environment' construed broadly so as to include abiotic effects (e.g. temperature, moisture, light, nutrients, toxins), biotic effects (e.g. resources, competitors, predators), and other effects (e.g. harvesting of wild populations).

Environmental changes that alter selection may be particularly extreme in the case of human-caused disturbance (Pimm *et al*. 1995; Hughes *et al*. 1997; Vitousek *et al*. 1997). Populations facing these greater-than-normal disturbances may therefore manifest greater-than-normal phenotypic responses. Dramatic responses are certainly known for some populations facing human disturbance (Palumbi 2001; Stockwell *et al*. 2003) — but is this a general phenomenon? We address this question by comparing rates of phenotypic change between populations experiencing either human-induced (anthropogenic) or natural environmental change. We specifically test whether phenotypic changes associated with human disturbances rise above the baseline typical of more 'natural' environmental variation. We discuss our findings in relation to whether or not populations can adapt to the environmental changes wrought by humans.

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We next ask whether any observed differences between anthropogenic and natural contexts might have their origin in phenotypic plasticity, which occurs when environmental conditions directly influence phenotypic expression for a given genotype. We address this question by comparing phenotypic changes between anthropogenic and natural contexts based on two different subsets of the data. One subset includes only studies of wild-caught individuals, which integrate both genetic and plastic effects ('phenotypic' rates). The other subset includes only studies based on common-garden or quantitative-genetic methods, which presumably exclude most plastic effects ('genetic rates'). If phenotypic change differs between anthropogenic and natural contexts with respect to phenotypic rates but not genetic rates, then the difference may be the result of plasticity. We discuss our findings in relation to how plasticity may aid population persistence in the face of environmental change (Baldwin 1896; Robinson & Dukas 1999; Price *et al*. 2003; West-Eberhard 2003; Yeh & Price 2004; Ghalambor *et al*. 2007).

Finally, we ask whether phenotypic change following environmental change is typically abrupt or gradual. We make this comparison by examining studies conducted over different lengths of time. Under the 'abrupt' model, phenotypic change may be as great in studies evaluating short time intervals as in studies evaluating long time intervals. Under the 'gradual' model, phenotypic change may be small in studies evaluating short time intervals but increasingly large in studies evaluating long time intervals. The distinction between these two trajectories may have particular conservation importance—because populations capable of initially rapid change may have a greater chance of persistence (Bürger & Lynch 1995; Gomulkiewicz & Holt 1995; Price *et al*. 2003; Kinnison & Hairston Jr 2007).

Our analysis is limited in several respects. Principal among these is that we consider only animals, even though plants are certainly known to show dramatic phenotypic responses to environmental change (Bone & Farres 2001; Law & Salick 2005; Franks *et al*. 2007). In addition, we consider only continuous phenotypic traits, even though dramatic change is also known for discrete polymorphisms (e.g. Sinervo 2001; Balanyá *et al*. 2006). Another limitation is that our compilation of studies ceased in 2005. Although this cut-off should not introduce any particular bias, the recent acceleration of relevant studies will allow much more comprehensive analyses in the near future. Finally, we were limited in our data collection to studies in the peer-reviewed literature, which could lead to the sorts of the publication biases that often afflict meta-analyses: selective choice of study systems or traits and selective publication of large effects. Owing to these (and other) constraints, our analysis is intended to provide a first broad-brush pass at what will ultimately prove to be a much more complicated picture.

Materials and methods

Our database includes studies that quantified either (i) phenotypic change through time within a population (allochronic) or (ii) phenotypic differences between populations that had a common ancestry in the recent past (synchronic). In order to emphasize 'contemporary' effects while still including a reasonable range of time frames (Hendry & Kinnison 1999; Kinnison & Hendry 2001; Stockwell *et al*. 2003), we include only studies of change occurring over approximately 200 generations or fewer. For each trait in each comparison between samples in each study, we calculated (whenever possible) two standard metrics of phenotypic change. One metric is the 'Darwin', which is typically calculated by taking the natural logarithm of the trait means in two samples, calculating the difference, and then dividing this difference by the number of years in millions (Haldane 1949; Gingerich 1993). Darwins thus represent proportional change in units of e per million years. Another metric is the 'Haldane', which is typically calculated by taking the difference between two sample means, and dividing this difference by the within-population standard deviation and then by the number of elapsed generations for the organism (Haldane 1949; Gingerich 1993). Haldanes thus represent absolute change in standard deviations per generation. The resulting database, provided as Supplementary material, includes 75 studies of 68 systems, where a 'system' represents a particular species in a general geographical area. These studies together yielded 2847 rates in Darwins and 2414 rates in Haldanes. More details on an earlier version of the database are provided by Kinnison & Hendry (2001).

Each rate in the database was assigned to one of the following six categories according to the type of environmental change the populations were experiencing.

1 '*In situ* natural variation' was assigned when established populations were not subject to an obvious human impact ($N = 18$ systems). Most studies in this category involve the long-term monitoring of natural populations, such as Darwin's finches of the Galapágos (Grant & Grant 2002, 2006).
2 '*In situ* anthropogenic disturbance' was assigned when established populations were subject to an obvious human impact ($N = 17$). Examples here include harvesting, anthropogenic acidification, localized thermal inputs, and point-source chemical pollution.
3 'Introduction' was assigned when humans transferred a species to a new geographical location ($N = 17$), and comparisons were then made between introduced and ancestral populations.
4 'Introduction of a new host' was assigned when humans transferred an exotic host species into the range of a native species ($N = 2$), with comparisons then made

between populations on the native and introduced hosts. Examples in our database are currently limited to native phytophagous insects colonizing introduced host plants (e.g. Carroll *et al*. 2005).

5 'Self-induced range or host expansion' was assigned when new populations were established without any direct human influence ($N = 4$), and comparisons were then made among the new populations or among the new and ancestral populations. Examples include birds colonizing new habitats within their native range (Yeh & Price 2004) or new islands outside their native range (Clegg *et al*. 2002).

6 'Range expansion after introduction' was assigned when humans introduced a species to a new geographical location, and the species then spread on its own accord to occupy multiple sites ($N = 10$). Comparisons were here made among the self-colonized populations.

Assignment into these categories was sometimes ambiguous, such as in the case of climate change, and so readers are invited to reanalyse the database (see Table S1) as they see fit. Our primary goal in the present study is simply to determine *in general* whether anthropogenic disturbances are associated with greater rates of phenotypic change. We therefore maximized sample sizes by grouping subsets of the above six categories into two broader 'contexts' — anthropogenic or natural. The anthropogenic context included *in situ* anthropogenic disturbance, introduction, and introduction of a new host. The natural context included *in situ* natural variation, self-induced range or host expansion, and range expansion after introduction. The last of these categories could arguably be assigned to either context, but we treat it here as 'natural' because comparisons were made among populations that naturally begot one another within the introduced range. Regardless, this particular decision did not materially influence our conclusions. All statistical inferences are then based on comparisons between the two contexts. We also present the distribution of data in the six categories — but only to crudely evaluate whether any particular category has an obviously inordinate influence on our conclusions.

Rates of phenotypic change are slower over longer intervals, and so rate comparisons need to account for the length of elapsed time (Gingerich 1993; Kinnison & Hendry 2001; Sheets & Mitchell 2001). Our solution is to plot the absolute amount of phenotypic change, rather than the rate, against the length of time over which that change took place (Kinnison & Hendry 2001; Estes & Arnold 2007). These plots can be analysed using analysis of covariance (ANCOVA) to test for the effects of time interval (years for Darwins, generations for Haldanes) and context (natural or anthropogenic) on the absolute amount of phenotypic change (Darwin or Haldane numerators). These analyses were based on the maximum or mean amount of phenotypic change for a given system — in relation to the mean time interval for that system. This use a single value per system avoided the nonindependence of data points within a given system. Maximum changes might reflect those that can be *potentially* accomplished, whereas mean changes might reflect those that are *typically* accomplished (Kinnison & Hendry 2001).

Negative vs. positive directions of phenotypic change (e.g. increases or decreases in body size) are not relevant to our main question — and so our analyses are based on absolute values. Although the use of absolute values can artificially inflate apparent change (owing to measurement error: Hereford *et al*. 2004; Hersch & Phillips 2004), our goal was simply to compare the amount of change *between* anthropogenic and natural contexts. As long as the contribution of measurement error is similar in the two contexts, no bias afflicts this comparison.

Different readers might favour different rate metrics or different data transformations (Kinnison & Hendry 2001). We attempt to cover the main options (and opinions) through the use of 24 different ANCOVAS that represent all possible combinations of the following distinctions: (i) raw values or log 10 values (values of zero assigned the next smallest value); (ii) the maximum or mean phenotypic change for each system; (iii) Haldane numerators vs. generations or Darwin numerators vs. years; and (iv) all rates, phenotypic rates (based on wild-caught individuals), or genetic rates (based on common-garden or quantitative-genetic methods). In each ANCOVA, we first tested whether the two contexts differed in their relationship between phenotypic change and time interval (i.e. slopes). Failing to uncover any significant heterogeneity (all $P > 0.10$), we removed the interaction term to test for effects of time interval and context (i.e. comparisons of least-squares means corrected for time). P values are not corrected for multiple comparisons because (i) we do not consider only a few significant P values as conclusive evidence for an effect, and (ii) we are not interested in which specific ANCOVAS are significant. Instead, we are interested in whether a diversity of tests reveals consistent trends in direction and significance (see below).

Results

Our first major finding is that phenotypic changes associated with human disturbance often rise above the baseline typical of natural environmental variation. We make this inference because least-squares means were greater for anthropogenic than for natural contexts in 23 of 24 ANCOVAS, with nine of these attaining $P < 0.05$ and two others attaining $P < 0.10$ (Figs 1–3; Tables 1 and 2). The binomial likelihood of obtaining nine or more values of $P < 0.05$ by chance is much less than 0.001. Moreover, the differences were not trivial, with phenotypic changes in anthropogenic

contexts exceeding those in natural contexts by a factor of 1.7 (averaged across the 12 ANCOVAS that used raw data).

The distribution of data in the six categories of environmental change (Fig. 4) suggests that the difference between anthropogenic and natural contexts is not driven by any particular category. In one logical contrast, phenotypic changes associated with *in situ* anthropogenic disturbance appear greater than those associated with *in situ* natural variation. In another, phenotypic changes associated with human-mediated introductions appear greater than those associated with self-induced range expansion or range expansion after introduction. Although our general conclusion of greater change in anthropogenic contexts thus appears robust, Fig. 4 does suggest some interesting variation that should be explored following the accumulation of more data.

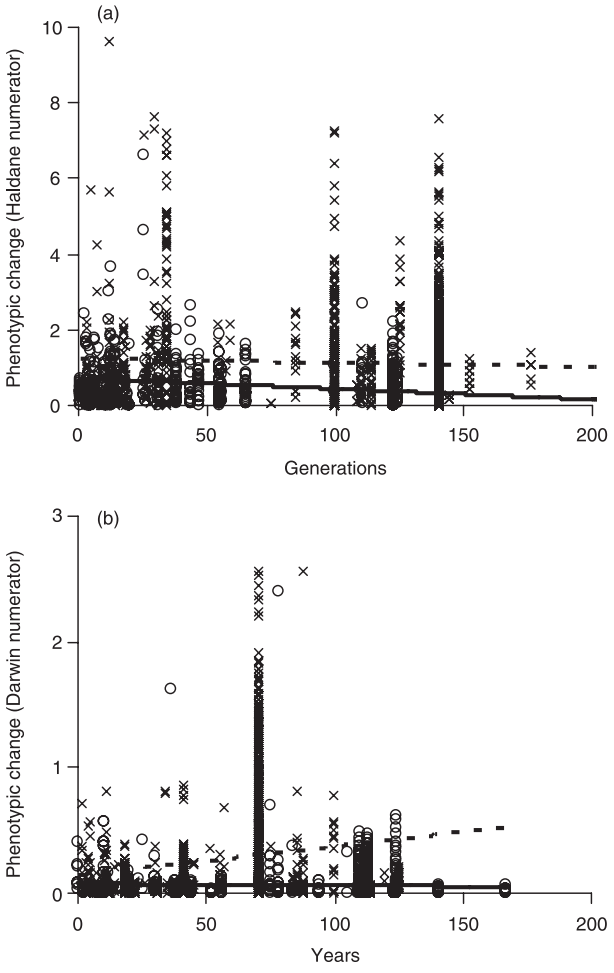

Fig. 1 A depiction of all phenotypic changes in the database. The top panel shows the amount of change in standard deviation units (Haldane numerator) vs. the time interval in generations. The *x*-axis is truncated at 200 generations for better visualization — only one interval was (slightly) longer. The bottom panel shows the proportional change in units of *e* (Darwin numerator) vs. the time interval in years. Changes in anthropogenic contexts are shown by crosses, whereas those in natural contexts are shown by open circles. Lines shown are least-squares regression relationships: — dashed for the anthropogenic context and solid for the natural context. These data are presented in full to give the reader a feeling for the entire database, whereas statistical analyses are based on a single value for each system (i.e. Figs 2 and 3).

Our second major finding is that phenotypic plasticity may contribute to the above difference between anthropogenic and natural contexts. This inference emerges when comparing the two contexts based on studies of wild-caught individuals ('phenotypic' rates) or studies employing common-garden or quantitative-genetic methods ('genetic' rates). In these comparisons, the difference between anthropogenic and natural contexts attained $P < 0.05$ for five of the eight 'phenotypic' ANCOVAS but for none of the eight 'genetic' ANCOVAS (Figs 2,3; Tables 1,2). This difference between study types is not just a function of sample size differences because least-squares means for phenotypic change in anthropogenic contexts exceed those in natural contexts by a factor of 2.2 for phenotypic studies but only 1.3 for genetic studies (averaged across the ANCOVAS that used raw data).

Our third major finding is that the abrupt model of phenotypic change seems more appropriate than the gradual model. We make this inference because relationships between phenotypic change and time interval were never significant (Tables 1 and 2), nor were any noteworthy trends obvious (Figs 1–3). In short, the amount of phenotypic change can be substantial, but the distribution of these changes remains similar all the way from short to long time intervals.

Discussion

Human influences

The available data suggest that phenotypic changes associated with human disturbance often rise above the baseline typical of natural environmental variation. On the one hand, this difference might reflect a publication bias — if investigators are more likely to focus on large perturbations in anthropogenic contexts than in natural contexts. We do not consider this to be a particularly likely general explanation given that studies of natural contexts also tend to emphasize responses to dramatic environmental change (e.g. Bell *et al*. 2004; Hargeby *et al*. 2004; Grant & Grant 2006). On the other hand, the difference between contexts may have a biological basis, perhaps reflecting a combination of what might be called 'acceleration' and 'winnowing.' Acceleration occurs if the expectation for a typical population is to show a greater phenotypic response in an anthropogenic context than in a natural context. Winnowing occurs if populations that manifest small phenotypic responses are more likely to perish in an anthropogenic context than in a natural context. Acceleration would thus inflate the response of a typical population

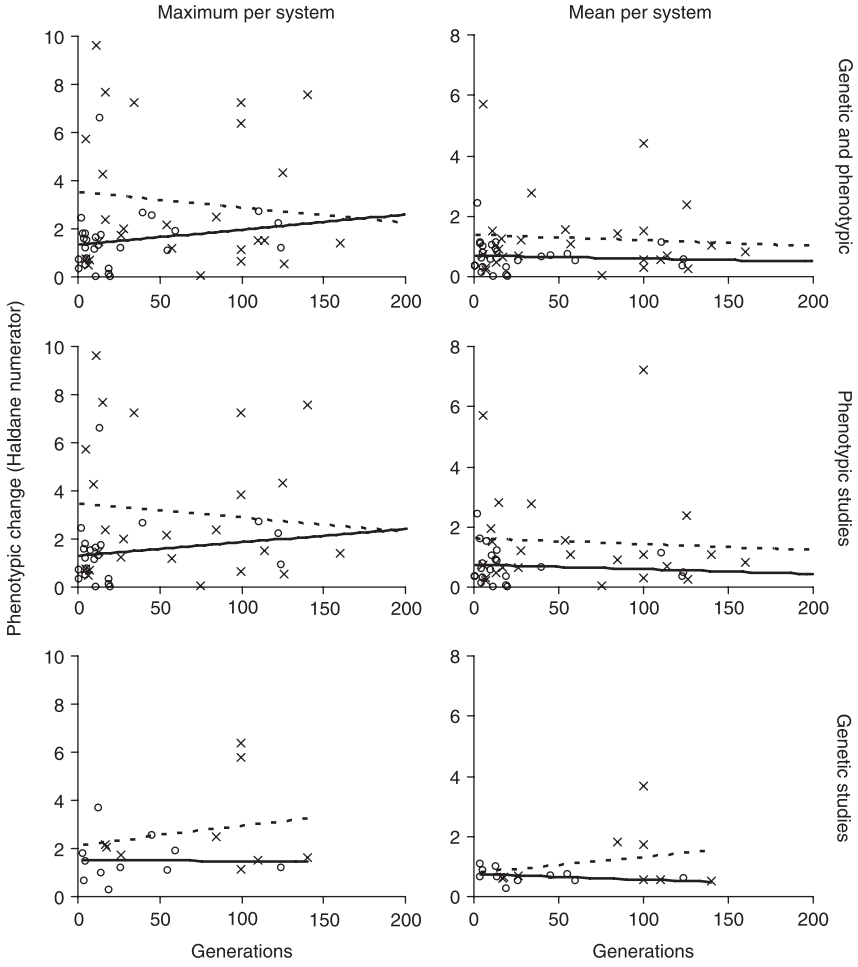

Fig. 2 Phenotypic changes in each system expressed as the maximum (left-hand panels) or mean (right-hand panels) amount of change in standard deviation units (Haldane numerator) vs. the time interval in generations. The top panels show all studies ('Genetic and Phenotypic'). The middle panels show studies based on wild-caught individuals ('Phenotypic'). The bottom panels show studies using common-garden or quantitative-genetic methods ('Genetic'). All other conventions are as described in the caption for Fig. 1.

whereas winnowing would remove populations showing the smallest responses.

Both acceleration and winnowing may occur in anthropogenic contexts. Acceleration seems likely given that humans cause extreme environmental change (Vitousek *et al*. 1997), and that a greater environmental change should cause a greater phenotypic response (West-Eberhard 2003). Winnowing seems likely given that humans cause the extinction of many populations and species (Pimm *et al*. 1995; Hughes *et al*. 1997), that human-mediated introductions often fail (Williamson & Fitter 1996; Sax & Brown 2000), and that extinction is more likely for populations that are less capable of rapid adaptation (Bürger & Lynch 1995; Gomulkiewicz & Holt 1995; Boulding & Hay 2001; Price *et al*. 2003; Kinnison & Hairston Jr 2007). Note that acceleration and winnowing are not mutually exclusive even within a single population. That is, acceleration may initially occur but the population may still be winnowed away — if adaptation is too slow to offset the demographic costs of selection (Pease *et al*. 1989; Bürger & Lynch 1995; Gomulkiewicz & Holt 1995; Boulding & Hay 2001; Kinnison & Hairston Jr 2007). It is also important to recognize that phenotypic change in response to a disturbance can be maladaptive (Grether 2005; Ghalambor *et al*. 2007), and so acceleration in some phenotypic directions may actually *increase* the chances of winnowing.

The distinction between acceleration and winnowing is critical for conservation. If the patterns we documented are mostly the result of acceleration that achieves sustainability, then many populations may be able to respond adaptively to the challenges presented by human disturbance. If the patterns we documented are mostly the result of winnowing (or acceleration that cannot achieve sustainability), then adaptation may often fail to rescue populations experiencing human disturbance. Our data do not allow a definitive conclusion regarding the relative contributions of acceleration and winnowing — but a signature of acceleration may be evident in the greater number of large for phenotypic changes in anthropogenic contexts (Figs 2,3). A missing piece in this puzzle is the extent to which adaptive changes in natural populations actually influence their probability of establishment and persistence (Lee 2002; Cox 2004; Richards *et al*. 2006; Kinnison & Hairston Jr 2007). This should be a major target for future research.

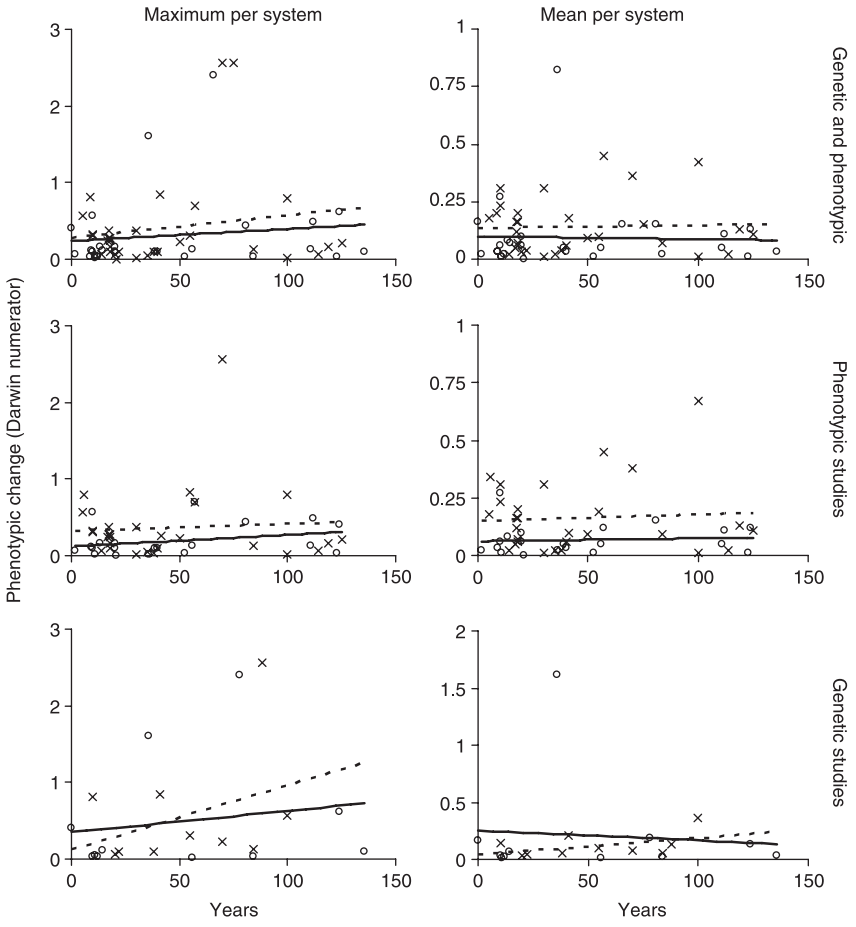

Fig. 3 Phenotypic changes in each system expressed as the maximum (left-hand panels) or mean (right-hand panels) proportional change in units of *e* (Darwin numerator) vs. the time interval in years. All other conventions are as described in the captions for Figs 1 and 2.

The potential role of plasticity

A striking pattern in our data is that the difference in phenotypic change between anthropogenic and natural contexts is greater when evaluated using wild-caught individuals than when using common-garden or quantitative genetic methods (Figs 2 and 3). Part of this difference between study types may reflect different sample sizes, and yet estimated effect sizes are also considerably greater when based on wild-caught individuals (Tables 1 and 2). One possibility is that common-garden environments lack the stresses needed to release important genetic variation (Hoffmann & Merilä 1999). Alternatively, the greater environmental challenges that typify anthropogenic contexts may be more easily bridged by plasticity than by genetic change (Price *et al*. 2003; West-Eberhard 2003; Richards *et al*. 2006). We therefore suggest that the greater phenotypic changes in anthropogenic contexts than in natural contexts may be partly the result of phenotypic plasticity.

Strong plastic responses to human-induced environmental change might either increase or decrease the likelihood of population persistence, and might also alter the strength of selection and the rate of genetic change (West-Eberhard 2003; Ghalambor *et al*. 2007). If plastic responses are sufficiently adaptive, they may substantially reduce the fitness costs of human-induced environmental change. In this case, plasticity may aid population persistence, reduce the strength of selection, and slow genetic change for the plastic trait (Price *et al*. 2003). If, in contrast, plastic responses are maladaptive, they may increase fitness costs, contribute to population declines, and increase selection and genetic change for the plastic trait (Grether 2005). And, of course, plastic responses in one trait may precipitate genetic changes in other traits (Price *et al*. 2003). Formally acknowledging these alternatives highlights the need to examine how plasticity influences the fate of populations confronted by human disturbance (Ghalambor *et al*. 2007).

Abrupt changes

Our analyses suggest that phenotypic change is often abrupt — because the amount of change was seemingly independent of the time interval. Although previous studies have found that individual populations can certainly show increasing phenotypic change with increasing time, this effect here appears much weaker than the variation across studies for a given timescale (see also Gingerich 2001;

Table 1 ANCOVA comparisons of 'anthropogenic' (anthro.) and 'natural' contexts based on raw data. Analyses are conducted using the 'Maximum' or 'Mean' amount of phenotypic change for a given system, expressed as either standard deviations (Haldane numerator) or units of *e* (Darwin numerator). Analyses are conducted using either 'Phenotypic' estimates (based on wild-caught individuals), 'Genetic' estimates (based on common-garden or quantitative-genetic studies), or 'Both' types of estimate. Shown are sample sizes (*N*) for each context; *F*-values for the effects of context (anthropogenic vs. natural), time interval (generations for Haldanes; years for Darwins), and their interaction; and least-squares estimates of mean values for each context while controlling for time

Specific data set			*N*: anthro./ natural	ANCOVA with interaction			ANCOVA without interaction		Least–squares means	
				Context	Time	Interaction	Context	Time	Anthro.	Natural
Haldane	Maximum	Both	27/27	6.548†	0.000	0.828	6.696†	0.184	3.141	1.454
Haldane	Maximum	Phenotypic	25/22	5.717†	0.001	0.577	5.956†	0.167	3.155	1.395
Haldane	Maximum	Genetic	9/11	0.240	0.163	0.232	1.894	0.221	2.661	1.596
Haldane	Mean	Both	27/27	3.266*	0.209	0.018	5.185†	0.328	1.294	0.638
Haldane	Mean	Phenotypic	25/22	2.784	0.158	0.002	4.358†	0.225	1.532	0.671
Haldane	Mean	Genetic	9/11	0.005	0.155	1.670	1.215	0.242	1.152	0.730
Darwin	Maximum	Both	32/26	0.031	1.300	0.116	0.444	1.258	0.411	0.309
Darwin	Maximum	Phenotypic	28/20	1.210	0.597	0.042	2.212	0.590	0.367	0.191
Darwin	Maximum	Genetic	10/11	0.123	1.278	0.310	0.041	1.011	0.565	0.496
Darwin	Mean	Both	31/27	0.373	0.997	0.077	1.591	0.001	0.141	0.094
Darwin	Mean	Phenotypic	28/20	2.188	0.182	0.032	6.501†	0.199	0.163	0.068
Darwin	Mean	Genetic	10/11	0.523	0.017	0.250	0.318	0.011	0.120	0.208

*$P < 0.10$, †$P < 0.05$.

Table 2 ANCOVA comparisons of anthropogenic and natural contexts based on log-10 transformed data. See the caption of Table 1 for more details

Specific data set			*N*: anthro./ natural	ANCOVA with interaction			ANCOVA without interaction		Least–squares means	
				Context	Time	Interaction	Context	Time	Anthro.	Natural
Haldane	Maximum	Both	27/27	1.727	0.032	0.459	3.137*	0.075	0.265	–0.029
Haldane	Maximum	Phenotypic	25/21	1.416	0.000	0.282	2.848*	0.000	0.272	–0.026
Haldane	Maximum	Genetic	9/11	0.076	0.186	0.019	2.452	0.180	0.348	0.110
Haldane	Mean	Both	27/27	0.465	0.078	0.014	4.126†	0.091	–0.068	–0.367
Haldane	Mean	Phenotypic	25/22	0.617	0.260	0.018	4.771†	0.271	–0.022	–0.405
Haldane	Mean	Genetic	9/11	0.803	0.177	1.709	1.764	0.020	–0.024	–0.194
Darwin	Maximum	Both	32/26	0.388	0.024	0.098	1.179	0.101	–0.715	–0.875
Darwin	Maximum	Phenotypic	28/20	3.008*	0.003	1.763	2.645	0.009	–0.705	–0.949
Darwin	Maximum	Genetic	10/11	0.055	0.310	0.252	1.050	0.078	–0.543	–0.848
Darwin	Mean	Both	31/27	0.948	0.292	0.092	5.289†	0.201	–1.038	–1.328
Darwin	Mean	Phenotypic	28/20	2.403	0.033	0.817	5.953†	0.023	–1.009	–1.352
Darwin	Mean	Genetic	10/11	0.184	0.183	0.443	0.763	0.010	–1.032	–1.242

*$P < 0.10$, †$P < 0.05$.

Kinnison & Hendry 2001; Estes & Arnold 2007). Some of the apparent abruptness may be the result of biases inherent in meta-analysis, one being measurement error that inflates apparent change. This effect is probably not overriding, however, because the amount of phenotypic change in many studies is clearly greater than measurement error. Another possibility is that investigators focusing on short timescales preferentially target systems where dramatic changes are expected. Similar biases may attend the choice of traits examined and whether or not the findings are published. Although these biases may well contribute to the apparently abrupt changes on very short timescales, they cannot explain why greater changes are not observed over longer timescales. Future work could reduce potential biases by correcting for measurement error, selecting populations and traits independent of

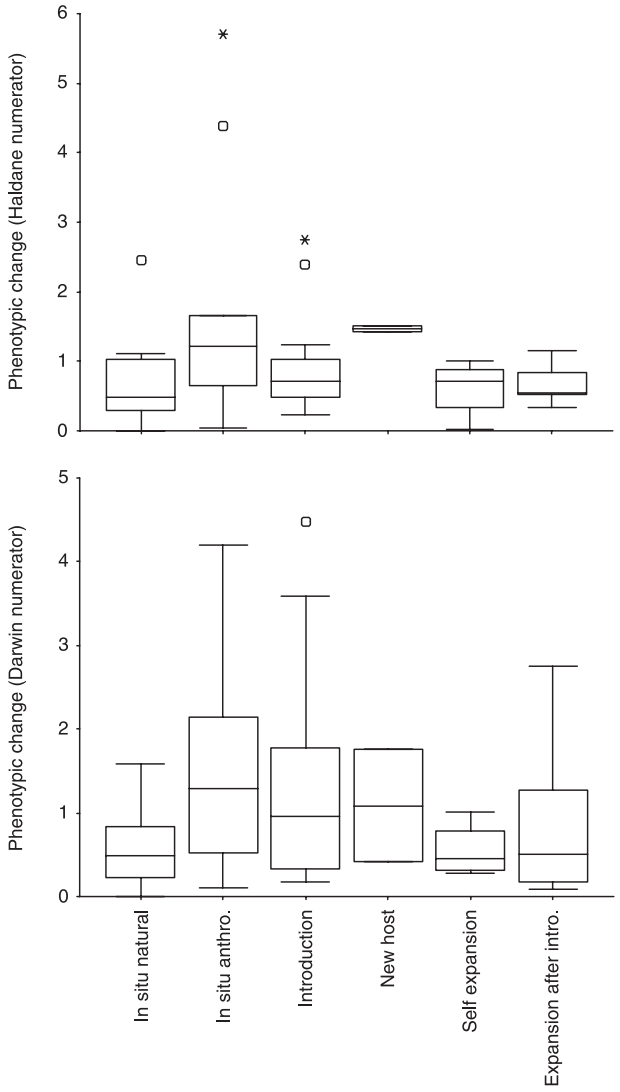

Fig. 4 Phenotypic changes associated with each category of environmental change (see text for details). The data summarized here are based on the mean value per system for the absolute change in standard deviation units (Haldane numerator; top panel) or the proportional change in units of *e* (Darwin numerator; bottom panel). Conventions include boxes that contain 50% of the data, central horizontal lines that represent medians, and whiskers that contain the remainder of the data — excluding outliers (open circles) and extreme values (asterisks). One extreme value (Darwin numerator = 8.2) is not shown for *in situ* natural variation in the bottom panel so as to aid comparison among the categories. Note that these data are shown as box plots rather than scatter plots (as in Figs 1–3) because six groups would be hard to visually compare in scatter plots and because our analyses showed that the amount of phenotypic change was not correlated with time interval (see text).

expected change, and publishing results irrespective of the observed change. We should also point out that some rates in the database were calculated for systems where a disturbance might have occurred at any time during the interval (i.e. not just at the start). This might make the amount of change less dependent on the length of the time interval (see also Estes & Arnold 2007).

Keeping the above issues in mind, it nevertheless seems useful to postulate biological reasons for abrupt phenotypic change. For studies of wild-caught individuals, this pattern is exactly as expected when a shift in the environment leads to a large initial plastic response, which then either obviates the need for genetic change or is followed by cryptic genetic change (Baldwin 1896; Waddington 1961; Robinson & Dukas 1999; Pigliucci & Murren 2003; Price *et al*. 2003; West-Eberhard 2003). This interpretation is consistent with recent studies that have found much larger plastic than genetic responses to environmental change (Trussell & Smith 2000; Both & Visser 2001; Réale *et al*. 2003) — although this is not always the case (Merilä *et al*. 2001). In order to draw broad generalizations about these different responses, more studies will need to examine plasticity, genetic change, and their interaction in populations experiencing environmental change.

Interestingly, increasing change with time was also undetectable in the data set based on common-garden and quantitative genetic studies — where plasticity should be limited. Several possible explanations come to mind. First, genetic change may be greatest immediately after a disturbance because this is when selection is strongest, i.e. the population is farthest from its phenotypic optimum. Indeed, studies examining individual populations through time clearly show that evolutionary change slows down with increasing time following a disturbance (Reznick *et al*. 1997; Kinnison & Hendry 2001). Second, particularly rapid changes may not be sustainable over long time periods owing to very high mortality or the depletion of genetic variation (Bürger & Lynch 1995; Boulding & Hay 2001; Kinnison & Hairston Jr 2007). Third, natural populations may experience strong selection on short time intervals, but the direction of selection may vary through time and therefore cancel out over longer time intervals (Gingerich 2001; Kinnison & Hendry 2001; Grant & Grant 2002; Estes & Arnold 2007). Regardless of the precise reason, the abruptness of phenotypic change suggests that studies spanning more than even a few generations may miss the critical early stages of this process.

Where to next?

Although our basic conclusions seem well supported by the existing data, we nevertheless view them as preliminary. One reason is that the studies we compiled are too few and diverse to be definitive. Greater confidence will require the inclusion of many more studies, which should be feasible given the expanding wave of interest in contemporary evolution. At the same time, we must recognize that meta-analyses will remain perpetually

susceptible to publication bias. We therefore suggest an additional, and perhaps more powerful, method of inference. Specifically, it would be useful to compare phenotypic changes in conspecific populations that are or are not exposed to human influences. Such comparisons may be able to uncover the specific conditions that cause more or less rapid change in the face of particular types of human or natural disturbance.

Our results suggest that conservation biologists should increase their attempts to understand how adaptive phenotypic change influences the persistence of populations. To do so, one might ideally determine the nature of selection on specific traits, the contribution of these traits to fitness, the tendency of traits to show adaptive plasticity, and the potential for traits to respond genetically to selection (e.g. genetic variances and covariances). This set of objectives is unattainable in many crisis-driven conservation scenarios. Even here, however, an invaluable first step might be to assess how traits respond plastically to changing environmental conditions, and how this plasticity influences survival and reproductive success. Even more advisable would be to start the above process of enquiry for populations that are not yet in drastic decline but that are potentially susceptible to intensifying disturbances. Here we have some excellent examples to emulate (e.g. Etterson & Shaw 2001; Réale *et al*. 2003; Both *et al*. 2006).

Conclusion

Conservation biology often focuses on maintaining viable populations in the face of environmental change, an outcome that will depend at least in part on the adaptive fit of phenotypes to their selective environment (Bürger & Lynch 1995; Gomulkiewicz & Holt 1995; Stockwell *et al*. 2003; Kinnison & Hairston Jr 2007). This fit may be particularly challenged in anthropogenic contexts, where environmental changes are acute (Pimm *et al*. 1995; Hughes *et al*. 1997; Vitousek *et al*. 1997). Our analysis suggests that plasticity may be a critical component in the adaptive response of populations to human-induced environmental change. Of course, population persistence may also be influenced by the capacity for genetic change (Bürger & Lynch 1995; Pigliucci & Murren 2003; Stockwell *et al*. 2003; Grether 2005; Kinnison & Hairston Jr 2007). When disturbances are modest, many populations may be able to persist through phenotypic plasticity or genetic change. As anthropogenic influences intensify, plasticity and genetic adaptation may be pushed to their limits. Determination of these limits should be a major research priority.

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Andrew Hendry and Michael Kinnison both study the evolution and ecology of populations in the wild and under anthropogenic influences. Most of their work examines fish populations and evolutionary processes observed over contemporary time scales. Thomas Farrugia was an undergraduate student working with Andrew Hendry, and has now started to work on the ecology and physiology of elasmobranches.

Supplementary material

The following Supplementary material is available for this article:

Table S1 Evolutionary rate database used by Hendry, Farrugia and Kinnison

This material is available as part of the online article from:
http://www.blackwell-synergy.com/doi/abs/10.1111/j.1365-294X.2007.03428.x
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