FORUM

"A new view of life-history evolution"? – A response

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In a recent article by Stearns several approaches to the study of life history evolution were discussed including the use of quantitative genetics in the analysis of genetic correlation between life history characters. Stearns readily dismisses the quantitative genetic approach to the study of fitness character variation and correlation even though many cases are documented in the literature. I suggest a reappraisal of existing studies in light of Fisher's Fundamental Theorem of Natural Selection, extend the list of published reports of genetic correlation and covariation, and evaluate many of the problems confronting further progress in understanding the genetics of life history evolution. I conclude that the study of ecology and quantitative genetics of natural populations is a prerequisite to the understanding of how life history characters might evolve and may lend some insight into the process of speciation.

How should the study of life history phenomena change in light of recent theoretical and empirical studies in ecology? Stearns (1976) stated that life history characters "are free to coevolve under the influence of purely demographic forces" into a life history tactic or "set of coadapted traits designed by natural selection to solve particular ecological problems". He suggested that life history or fitness characters have some genetic basis and thus can coevolve. Stearns (1980) discusses the evolution of life history traits in the context of quantitative genetics, development, and physiology. He warns about the pitfalls, the most important of which he suggests are the difficulties of understanding the causes of the variation and generalizing about nature from highly artifical laboratory experiments, of studying intraspecific genetic variation and correlation of fitness characters which may represent "life history tactics".

Responding to Stearns (1980), as well as introducing several alternate ideas, I will point out several flaws in Stearns' reasoning about genetic analyses of fitness characters and suggest that this area of study is not quite so perilous as Stearns' (1980) believes.

Fisher (1958) initially proposed that the average fitness of a population evolves at a rate proportional to the additive genetic variance in fitness at that time. Genetic variance in fitness should be at or near zero if the population has achieved maximal fitness (Fisher 1958, Lewontin 1965). Thus, additive genetic variance as reflected by realized heritability estimates should approach zero for fitness characters such as fecundity, age of first reproduction, egg weight, development time, etc. (Falconer 1960). The depletion of genetic variance in fitness has been interpreted to be a result of intense directional selection for maximum fitness (Lewontin 1965); however, the existence of appreciable additive genetic variance in fitness in natural populations has been interpreted as a means of adaptation to an unpredictable environment (Istock 1978, 1981). The issue at hand is not solely the existence of genetic variance for fitness; indeed the biometrical genetic literature provides many examples of genetic variance for individual fitness characters (Istock 1981). Of interest here is the significant genetic correlation and/or covariance between fitness characters which can be interpreted as products of natural selection for "life history tactics."

I believe it is premature to assume that "genetic correlations will tell us little about the existence or nonexistence of sets of coadapted traits" as Stearns (1980) asserts based on so few and incomplete examples. More examples of genetic correlation and covariation between fitness characters in domestic and natural populations can be found. For example, in domestic poultry, Dickerson (1957; cited in Falconer 1960) reported that pullets that delay maturity lay fewer, larger

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eggs and are heavier at 18 weeks of age. Bonnier and Johnson (1957) showed a significant negative correlation between survival and development time in *Drosophila melanogaster.* Speiss et al. (1952) showed a genotypic basis for a positive correlation between adult survivorship and fecundity in laboratory populations of *D. persimilis.* Ohba (1967) indirectly demonstrated a significant positive correlation between lifetime fecundity and longevity in *D. pseudoobscura.* Speiss and Schellein (1956) and Speiss and Speiss (1966) showed a positive correlation between preadult development time and age at first reproduction and development time and fecundity, respectively, in *D. persimilis.* Hiraizumi (1961) demonstrated a negative correlation between female fertility and developmental rate in *D. melanogaster* for both homozygous and heterozygous configurations of the second and third chromosomes. Istock et al. (1976) showed a very high positive correlation between development time and the tendency to diapause in experimental populations of the pitcher-plant mosquito, *Wyeomia smithii.* The latter example is exemplary because they showed the same effect in *natural* populations (Istock 1978). Englebert and Bell (1970) demonstrated realized genetic correlations between pupation time and 13 day larval weight to be consistently high and negative in *Tribolium castaneum.* Derr (1980) showed that low age at first reproduction and large egg clutches were both important in maximizing fitness in *Dysderous bimaculatus,* but could not show genetic covariation even though both characters showed significant heritability. From a sib analysis and artificial selection experiments, Rose and Charlesworth (1981a, b) showed negative correlations between early fecundity and lifespan, and mean egg laying rate and longevity in outbred populations of *D. melanogaster.* Although this is not intended to be a complete listing of such studies, these results are suggestive of exactly what Stearns doubts can be recognized as life history tactics within species.

Stearns (1980) mentions four studies which he interprets as evidence against coadaptation between life history traits within populations (cf. Lerner 1954, Dawson 1977, Birch et al. 1963, and Dobzhansky et al. 1964). Because these authors showed that "the heritability of fecundity is higher than the heritability of age at maturity," the traits "are probably not coadapted, at least not within that population" (Stearns 1980). In the latter two studies, the authors showed more differentiation between experimental populations in fecundity than in developmental time. However, Stearns has misevaluated both studies. Birch et al. (1963) showed fitness differences between five geographical races of *D. serrata* along a 3000 mile transect in Australia. That these races could be used to show within population fitness differences is doubtful; it is highly unlikely that these populations are contiguous. In fact, a northern and southern subspecies have been recognized (Dobzhansky and Mather 1961). The results of Birch et al. (1963) are not concerned with *within* population coadaptation of life history traits but with broad geographic differences in relative fitness, probably resulting from local adaptation to vastly different environments. Furthermore, Birch et al. (1963) did not explicitly show that the differences in life history traits they found were heritable. Certainly a portion of the variation was probably genetic as all of their measurements were made in uniform laboratory conditions. However, they provided no direct genetic evidence, such as estimates of heritability. Stearns (1980) has therefore inappropriately interpreted the results of Birch et al. (1963) as evidence against the genetic coadaptation of life history traits.

In the second example, Dobzhansky et al. (1964) did show direct genetic differences in fitness by comparing inversion types of *D. pseudoobscura.* They showed differences in fecundity between alternate third chromosome inversion homokaryotypes and a general heterokaryotype superiority, but could show no differences in developmental rate. Since developmental rate and fecundity in *Drosophila* may be assumed to be under polygenic control (Bell et al. 1955, Speiss and Speiss 1966) and the size of the third chromosome inversions in *D. pseudoobscura* are small relative to the rest of the genome, it is unlikely that most of the genes for fecundity or developmental rate are concentrated within these inversions. Together with the inability of Dobzhansky et al. (1964) to eliminate environmental variations between their replicates in their developmental time experiments and the known large effect of the genetic background in polygenic variation for developmental rate (Speiss and Speiss 1966), the results of Dobzhansky et al. (1964) should not be interpreted as negative evidence for the coadaptation of life history traits. It is interesting to note that heritable variation for developmental time in *D. pseudoobscura* does exist in one of the natural populations that Dobzhansky et al. (1964) sampled for their experiments. Marien (1965) showed a significant selective advance for fast development in eight generations, but was unable to show selection for slow developmental rate. That the extant polygenic variance for developmental rate in *D. pseudoobscura* can be viewed as part of a life history strategy is at present premature. Given the number of genetic markers in many species of *Drosophila,* studies of the genotypic breakdown of fitness variation in these species are most desirable. But to question "the reality of tactics as patterns that can be recognized within species" populations (Stearns 1980) based on the above two studies, is clearly unwarranted.

One of Stearns' (1980) major criticisms of the quantitative genetic approach is that measurement of genetic correlation between fitness characters yields results which are meaningless for most laboratory experiments because of the unsuitability of the laboratory environment. It is undeniable that such measurements are difficult in the laboratory and near impossible in the field because these characters simply do "not vary very

much" (Stearns 1980). However, laboratory exposure of either genetic correlation or genetic covariation between fitness characters by means of artificial selection does tell us such relationships are at least *feasible* genetic conditions in natural populations (Lewontin 1974, Istock 1981). The success of most artificial selection experiments with outbred natural populations attests to the widespread nature of additive genetic variance for fitness; the success in animal and plant breeding is evident (Pollack et al. 1977). Genetic correlation or covariation between fitness characters is likely to exist in nature if it can be detected in the laboratory.

Stearns concluded from three laboratory studies (Lerner 1954, Robertson 1957, and Gall 1975) that the "verdict is not unanimous, but it (the data) suggests a genetic correlation between age at maturity and fecundity." One would have predicted these results from life history theory if "demographic tactics are real" (Stearns 1980). Stearns presents several convincing arguments cautioning against certain biological interpretations of these genetic relationships. But, as he points out for genetic correlations, these estimates "tell us nothing about the forces that produced them" (Lewontin 1974). For a number of reasons, I think Stearns has misjudged the importance of many of the documented cases of fitness character variation.

First, and most important, the measurement of genetic correlation and covariation which may "mirror demographic tactics" is only a *statistical description* of a complex genetic relationship estimated in the laboratory. Using the estimates of realized heritability and additive genetic variance made by regression analysis for single fitness traits, we can only speculate about the mechanisms which *cause* the variation in natural populations. Low, yet widespread additive genetic variance for fitness characters may be of adaptive importance in nature (Istock 1978, Lumme 1978, Derr 1980). The maintenance of polygenic variation for fitness may be in some part due to the same forces which are thought to regulate polygenic variation for traits other than fitness characters such as stabilizing selection (Mather 1941, Lerner 1954, Allard and Jain 1962), genetic drift (Lande 1976a), gene flow (Endler 1977), and mutation rate (Lande 1976b, 1980). Istock (1978) postulated that stabilizing selection could be the mechanism for fitness variation in populations of the pitcher-plant mosquito, *Wyeomia smithii.* Mukai (1964, Mukai et al. 1972) has substantiated that the polygenic variation in viability for *Drosophila melanogaster* is partially controlled by mutation. Indeed, Lande (1976b) predicted that mutation rates in the range 10^{-2} or 10^{-3} per gamete per generation can be an important source of genetic variation in polygenic characters which are influenced by stabilizing selection. The extent to which drift, gene flow, mutation, and selection alone or in combination may influence additive genetic variance for fitness characters is not well understood. The extent to which these forces shape "life history tactics" is even more obscure. Needed are more studies designed to discriminate between the causes of polygenic variation such as drift and selection (cf. Lande 1976a).

Theoretical predictions (Lande 1980) explicitly show that the covariance between polygenic traits can respond to changes in the selection regime. As long as recombination rates are greater than selection on polygenic variation, pleiotropic mutation can be a significant source of genetic correlation, with linkage per se contributing relatively little. Under these conditions and the high mutability of polygenic traits, Lande (1980) concludes that covariances are easily influenced by selection. Further careful experimentation in the laboratory and possibly in the field is required before it will be possible to identify the causes and knowledgeably comment on the significance of fitness variation and genetic correlation and covariation between fitness characters.

Secondly, since heritabilities are intrinsically low for fitness characters, it will be difficult to detect very small amounts of polygenic variation, amounts which appear to be consistent with an interpretation of no heritability. Even a small amount of additive genetic variance, just bordering on statistical significance, may play a crucial role in the evolution of a single or suite of life history characters.

Consider a population in nature, diploid and undergoing sexual reproduction, expressing a typical amount of additive genetic variance for a fitness character (Falconer 1960). Over evolutionary time as environmental conditions change, mean population fitness waxes and wanes as an array of genotypes is exposed to selection each generation because of recombination. Depending on the present environmental circumstances and the population's past history of adaptation, the realized heritability will change. During episodes of environmental stasis, much longer than generation time, recombination and directional selection for maximal fitness should drive the additive genetic variance to zero (Fisher 1958). During environmental changes as new genotypes arising from recombination interact with new environments, selection exposes larger and larger frequencies of those genotypes which were previously selected against. Any measurement of heritability will therefore be dependent upon *when* it is measured during the evolutionary "progress" of the population. The phenotypic score may move around some optimum value conferring greater fitness on the population in the face of long term environmental change (Lerner 1954), but without knowledge of how polygenic variation for fitness traits reacts to changing environments, heritability estimates, though not meaningless, are very difficult to interpret. Genetic correlations will therefore change as the heritabilities shift. Low heritabilities pose a dilemma to the study of natural populations because without knowledge of the agents of selection the interpretation of low heritability for fitness remains open to question. Needed are studies designed to document

the evolution of heritability of polygenic traits in changing environments.

Another type of "hidden" polygenic variation is that due to closely linked alleles which cancel out each other's effect on the phenotype (Lande 1976b). Allelic effects are assumed to be negatively correlated, yet after recombination and environmental change will produce new genotypic combinations. As Istock (1981) points out, natural populations may harbor large amounts of such genetic variation as a consequence of adaptation to fluctuating environments and recombination and altered gene expression many provide a genetic basis for response to long term environmental change. Whether or not much genetic variation is only expressed upon a change of environment, can only be known by careful studies of how polygenic variation is expressed.

I believe, as does Stearns (1980), that the study of life history evolution is now at a point of potentially rapid development. The emergence of Fisher's Fundamental Theorem to the forefront of life history theory has indeed provided a kind of new approach to the study of life history evolution sustaining the continuum from micro- to macroevolution. We are standing at the crossroads of several avenues of research which Stearns (1980) has elegantly summarized. But before we turn away prematurely from approaches to the genetic study of life history evolution, attempts must be made to unravel the causes for the widespread genetic variance in fitness which has already been documented. Therefore, a better comprehension of the nature of life history evolution will at first demand coordinated, rigorous attention to ecology and quantitative genetics. Once the genetics of the complex systems which constitute fitness characters and suites of interacting fitness characters are understood, we may then be in a position to analyze larger irreversible genetic changes in life history characters which may accompany speciation.

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Reply to Etges

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Stearns, S. C. 1981. Reply to Etges. – Oikos 38: 122–124.

Etges constructively points out errors in my interpretations of Birch et al. and of Dobzhansky et al., and usefully extends the list of studies on the heritability of fitness traits. We still differ on several points. First, I disagree with him over the reliability of genetic correlations. Measuring them is essential to progress in life-history work, but measurements done in single environments are not sufficient. To be useful, genetic correlations must be measured in several environments, one of which should be the field. The influence of developmental plasticity on genetic correlations in different environments remains fundamental. Second, Etges relies extensively on Fisher's fundamental theorem. While it certainly would strengthen the inferences we could make if it were true, its general applicability remains questionable because of the effects of strong selection, linkage, age-structure, and density-dependence. Third, the genetical point of view that permeates both Etges' paper and the Modern Synthesis is not be be preferred as an *exclusive* alternative. It must be combined with the organismal point of view to be fruitful. Fourth, recent discoveries in molecular biology may bring about a sweeping re-assessment of population and quantitative genetics, and are at least sufficiently unsettling to make an agnostic stance wise in the interim.

I found several aspects of Etges' (1981) comment constructive and useful. We differ on the interpretation of several important assumptions, but we agree at least as much as we disagree. His compilation of an extended list of estimates of heritabilities and genetic correlations of fitness characters will benefit everyone who works on life-history evolution. His criticism of my interpretation of Birch et al. (1963) and of Dobzhansky et al. (1964) is appropriate. I stand corrected.

His main point is that my criticism of genetic correlations as an unreliable tool was too strong. He cites both empirical studies that document the existence of genetic correlations in fitness characters and theoretical studies that demonstrate that the correlations themselves can evolve through pleiotropic mutations. He also emphasizes the importance of Fisher's Fundamental Theorem. To this I have several replies on several levels.

(1) The fact that heritabilities and genetic correlations are functions of the environments in which the organisms are raised remains fundamental. Many of the organisms that have been used in studying the quantitative genetics of fitness characters are small ectothermic animals or plants. Such organisms have notoriously high levels of developmental plasticity. If various fitness traits differ in their plastic responses, then the phenotypic correlations of the traits in nature can differ from their phenotypic correlations in the laboratory. Since we infer genetic correlations from phenotypic correlations, I contend that laboratory measurements can still mislead us about the nature and the direction of a coordinated response to selection in nature. For example, my own work with mosquitofish indicates that within each of several populations age at maturity is much more plastic than length at maturity. Furthermore, in comparisons among four populations of mean

fecundity, length at maturity, and size of offspring, the ranks of the four populations were different in the laboratory than they were in the field, and the rankings changed differently for different traits. Those observations mean that the phenotypic correlations observed among populations must differ in field and laboratory, and make it plausible that they also differ within single populations, although that point remains to be established.

These comments should not be taken as a rejection of quantitative genetics: we must measure the genetic correlations of fitness traits if we are to understand life-history evolution. However, that measurement will certainly not be sufficient. It will have much greater reliability if it is done in several different environments, and such measurements will have the added benefit of simultaneously estimating the phenotypic plasticities of the traits. An extremely important achievement in this field of research would be the measurement of genetic correlations for fitness traits in one population in several laboratory environments and in the field. I would consider that data base – difficult to get in the best of circumstances – sufficient grounds for reliable inference on some points.

To summarize this point: measurements of the genetic correlations of fitness traits are essential to the progress of life-history work, but they are not sufficient, and to be reliable they must be done in several environments, one of which should be the field. The empirical difficulty of this procedure does not alter its logical necessity at all.

(2) Much of traditional inference about the quantitative genetics of fitness traits in natural populations is based on Fisher's (1958) so-called "Fundamental Theorem of Natural Selection": the rate of change of average fitness in a population equals the additive genetic variance in fitness in the population at that time. A corollary, due to Robertson, is that the rate of change of a trait in a population is proportional to its correlation with fitness times the additive genetic variance for that trait. Etges (1981) relies extensively on these statements.

However, opinions about the theorem are divided. For example, Karlin (1975) states that "the fundamental theorem of natural selection *can only prevail* when there exists a stable equilibrium valid independent of the recombination fraction whose chromosomal frequencies are in linkage phase equilibrium. Such conditions are rarely satisfied" (pp. 394–395). Thus one problem with Fisher's theorem is posed by linkage, and the problem shrinks if recombination rates are greater than selection coefficients (Lande 1980). We do not yet know whether that is often the case, and I agree with Etges that we must find out.

Charlesworth (1980) has shown that, under certain circumstances, a version of Fisher's theorem holds for selective differences up to 40% per generation, but his thorough analysis turned up other problems, and his summary is worth quoting at length:

> "The general conclusion is, therefore, that for the case of weak selection and random mating with respect to age, the intrinsic rate of increase of a genotype or, more generally, the mean of the male and female intrinsic rates, provides an adequate measure of fitness in a density-independent and constant environment. This parameter can be used in much the same way as the discrete generation fitness to predict, to a good approximation, the rate of change of frequency and ultimate composition of populations with respect to single loci. Provided that linkage is either very tight or is rather loose, in comparison with the selection intensity, . . . this is true for the two-locus model as well, with some minor qualifications.
>
> If selection is strong, however, there is no unitary measure of fitness . . . [T]he intrinsic rate of increase provides a good indicator of the rate of spread of a rare non-recessive gene, but a different fitness measure must be used for a rare recessive gene. Furthermore, in the neighborhood of a polymorphic equilibrium, the intrinsic rate of increase will not predict the rate of change of gene frequency accurately . . ., and it also gives an inaccurate prediction of the composition of the equilibrium population. This conclusion is important for experimenters who wish to study selection by following gene frequency changes in a continuously breeding population such as a *Drosophila* population cage" (pp. 196–197).

As Charlesworth notes, some of the difficulties of Fisher's approach can be gotten around by weighting individuals by their reproductive value (Crow 1978), but at this point all we can say about the general applicability of Fisher's theorem is that linkage, strong selection, age-structure, and density-dependence all have implications that result in significant qualifications of it.

To summarize this point: Fisher's theorem may or may not be true. While it is certain that if it were true it would greatly strengthen the inferences we could make, it is equally certain that the jury is still out on the question of its general applicability.

(3) An implicit assumption of the genetical approach that Etges advocates is that an understanding of gene frequency changes, heritabilities, and genetic correlations is the sine qua non of success in evolutionary biology. In this, he stands firmly in the tradition of the Modern Synthesis. To a certain extent, I disagree with this position.

I regard population genetics not as the central "hard core" of evolutionary theory, but as one of a number of equivalent specialities that can be derived from a small set of basic statements by the use of ancillary assumptions (Stearns et al. unpubl.). In choosing population genetics as the core of evolutionary theory, one emphasizes the role genes play in constructing organisms and in recording in their frequencies the history of

selection. Causation is seen as flowing upward to the organism from the gene. In choosing the organism as the unit of selection, one emphasizes the role that the phenotype plays in interacting with the environment, persisting, and producing more organisms. At this level, it does not matter which genes the phenotype contains so long as it can survive and reproduce. Causation is seen as flowing from the phenotype down to the genes.

In fact, causation flows in both directions, during ontogeny from the gene to the organism, during evolution from the organism to the gene, and to choose one or the other as *the* unit of selection seriously misrepresents this basic fact: selection consists of two processes, the mechanics of genetics and the interactions between organisms and environment. From this point of view, the habitual practice of viewing the same phenomenon from the alternative perspectives of the whole organisms and the genes is not simply an exercise beneficial to our mental health, but an essential reflection of the nature of organisms and of evolution.

What do we see when we forget genetics and look at the phenotype? Plasticity, canalization, and constraint (Stearns 1981). What do we see when we forget the phenotype and look at the genes? Pleiotropy, covariance, thresholds, and changes in heritabilities and correlations as environments change. Neither perspective provides an exclusive framework for evolutionary interpretation, because each interprets the same phenomena on a different level. The interpretations are not mutually exclusive but complementary. Quantitative genetics provides a phenomenological description of the summary effects of a great deal of developmental structure; for some purposes that summary is critical. In other situations, genetics is of little help (Alberch 1981), and an explicit description of developmental mechanisms is essential. This raises the obvious point that we need to know more about the quantitative genetics of developmental mechanisms. For life-history traits, the most wide-spread mechanism of importance is developmental plasticity. That it can interact with genetics in important and surprising ways is nicely shown in Berven's reciprocal transplant studies of green frogs (cf. Berven et al. 1979).

To summarize this point: the genetical point of view is not to be preferred as an exclusive alternative. It must be combined with the organismal point of view to be fruitful.

(4) Finally, new and surprising results from molecular biology may require a complete reconstruction of population genetics and quantitative genetics. We understand the function only of genes which code for proteins: about 1% of the DNA in the human genome, for example. The remainder of the genome is composed of short simple repeats of nucleotide sequences called satellite DNA, of longer, more complex repeat sequences called intermediate repetitive DNA, and of unique sequence, single copy DNA that is interspersed with intermediate repeats. The functions of these three classes of DNA, composing 99% of the genome, are not understood and are the subject of a great deal of current controversy (see the 7 August 1981 issue of Science 213: 634–636 for a report by Lewin). They may have implications for the significance of linkage, for the nature of gene regulation, and for speciation. They may not. Whatever their eventual significance, their existence suggests that an agnostic attitude towards the fundamental assumptions of population and quantitative genetics is appropriate.

In conclusion, I want to make clear my support of continuing work on the quantitative genetics of fitness traits. Such work is necessary, but not sufficient. I not only support it, I do it: I have been working on the heritabilities of fitness traits in mosquitofish for over two years, and I except to continue to work in this area for several more years. My intention in writing Stearns (1980) was not to discourage people from using quantitative genetics, but to encourage those who use it to think about its limitations and about alternative ways of interpreting the same phenomena. I thank Etges for helping me to make that point, which was evidently not sufficiently explicit in the earlier paper. I simply distrust *exclusive* reliance on *any* particular point of view, and quantitative genetics is a particular point of view.

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