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COMMENTS

IS EVOLVABILITY INVOLVED IN THE ORIGIN OF MODULAR VARIATION?

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Abstract.—Lipson et al. (2002) presented an elegant linear algebraic formalism to define and study the evolution of modularity in an artificial evolving system. They employed simulation data to support their suggestion that modularity arises spontaneously in temporally fluctuating systems in response to selection for enhanced evolvability. We show analytically and by simulation that their correlate of modularity is itself under selection and so is not a reliable indicator of selection for modularity per se. In addition, we question the relation between modularity and evolvability in their simulations, suggesting that this modularity cannot confer enhanced evolvability.

Key words.—Adaptability, canalization, fluctuating selection, pleiotropy, robustness.

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Modularity is a major principle of design and abounds in nature. Functional separation of modules—from eukaryote organelles to *Drosophila* limbs to human cognitive faculties—may give robustness to changing inputs and facilitate future improvement. The question of the evolutionary origins of such modularity is important and the recent simulation study of Lipson et al. (2002) is therefore a welcome contribution. They introduce a potentially extremely useful formalism that allows one to quantify modularity and study its evolutionary origins. Environmental variables are described by a vector **E**, and phenotypic traits by a vector **P**. A matrix **A**, which premultiplies **E** to give **P**, then describes the organismal process of transforming environmental input into phenotypic output.

Lipson et al. argue that the ''blockiness'' of **A** and its correlate, the number of zero elements, are measures of modularity. By assigning fitnesses to realized phenotypes depending on their distance from an arbitrarily chosen optimum, Lipson et al. (2002) study the evolution of modularity. Their simulations show that the frequency of zero elements in the matrices deviates from the expected value (1/3, the frequency of zero elements at initialization and among random mutations) when the environment changes rapidly. Lipson et al. attribute these results to a ''second order (delayed) pressure for decomposition for adaptability,'' (p.1554) that is, the uncoupling of traits to allow independent optimization of each and hence increased ability to adapt to new environments. Enhanced evolvability is concluded to be a cause, as well as a fortunate outcome, of the preponderance of zero-element-rich matrices. We disagree with this conclusion and believe that an alternative explanation exists. In addition, we feel that modularity cannot influence evolvability in their study.

In the simulations of Lipson et al., the element values of **E** are restricted to -1 and $+1$ and the element values of **A** are restricted to -1, 0, and $+1$. The elements of the phenotype vector **P** are therefore restricted to the range $-n \rightarrow n$, where n is the number of dimensions of the vectors (eight in the simulations of Lipson et al.). They restrict the elements of **F**, the arbitrary optimal phenotype, to -1 and $+1$. The optimal phenotypes are therefore restricted to a small subset of all possible phenotypes, centered on the origin. We find that matrices with many zero elements tend to produce phenotypes that are closer to the zero vector, and therefore on average closer to the optimal phenotypes (mathematical details are given in the Appendix).

Rather than appealing to enhanced evolvability, the preponderance of zero-rich matrices can be explained by the advantage delivered to any **A** that can maintain a phenotype close to the origin, despite environmental perturbation (i.e., canalization; Waddington 1942). In Figure 1 we give the probability distribution of the value of an element of **P** as a function of ζ, the number of zero elements in the corresponding row of **A**. As ζ increases, the value of the focal element of **P** is more tightly distributed about the origin. Figure 2 reveals the relation between ζ and the mean scalar residual (negatively correlated with Lipson et al.'s measure of fitness) in a focal dimension: increasing ζ reduces the residual and thus increases fitness. Conducting simulations of our own, we have been able to demonstrate frequencies of zero elements significantly greater than 1/3, even when mutation is suppressed. Hence, individual lineages may thrive or decline, but cannot evolve and therefore cannot be under selection for enhanced evolvability (see Fig. 3 and Table 1).

Moreover, in the set-up of Lipson et al., it is unclear why enhanced evolvability is expected to play any role. Each element of the vector **P** is the result of (dot-) multiplying a separate row vector from **A** with **E**. Contrary to the suggestions of Lipson et al., manipulating the elements of such a row vector has no effect on the value of other elements of **P**. This means that when evolving **A** in the context of a certain environment **E** and a certain target phenotype **F**, every element of the actual phenotype **P** can be optimized independently. Interestingly, a different use of the same formalism was suggested by Lipson et al. and avoids this problem. Under this alternative scheme, vector **E** describes the genotype and matrix **A** describes the genetic architecture of the phenotype (e.g., pleiotropy), a framework similar to the multiple quantitative trait model proposed by Taylor and Higgs (2000). By allowing both **E** and **A** to evolve, one can study the evolution of modularity and evolvability under, for example, fluctuations in **F**.

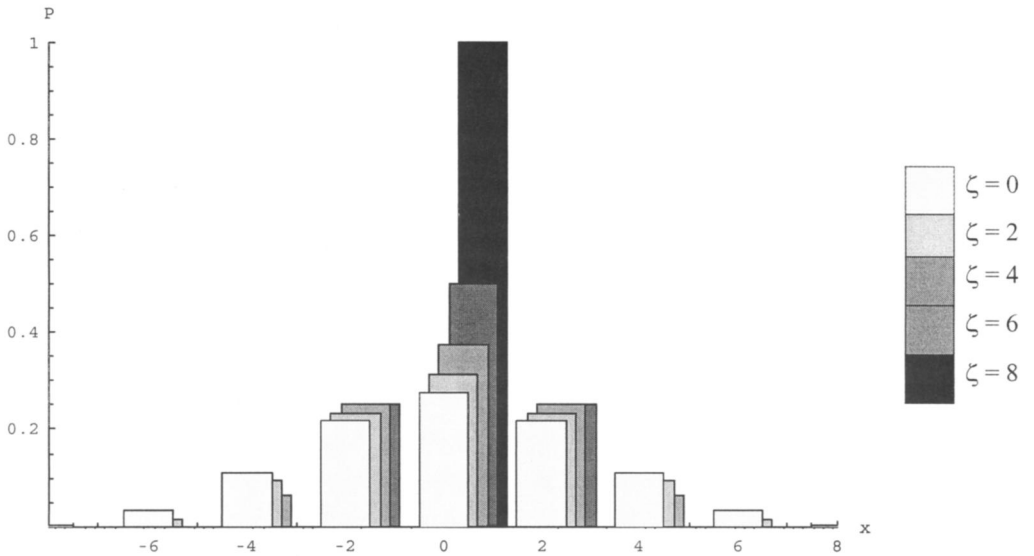

FIG. 1. The probability distribution of the value of $\mathbf{P}_\kappa$ as a function of the number of zero elements in the κ^{th} row of the 8 × 8 ternary matrix **A**, ζ. Here n (= 8) and every value of ζ (= 0, 2, 4, 6, 8) are even, so the values of $\mathbf{P}_\kappa$ are restricted to the set of even integers.

This is not to say that modularity is not under selection. It is possible that modularity confers robustness of fitness in response to the form of environmental change investigated by Lipson et al. When matrices are highly modular, such that there is a one-to-one correspondence between environmental characteristic and phenotypic trait, alteration of only one aspect of the environment will perturb the phenotype in one dimension only. Matrices that are less modular have environmental components each affecting more than one trait, and more than one trait being affected by several environmental components. They are therefore perturbed in multiple dimensions whenever a single aspect of the environment is altered. Because Lipson et al. change the sign of only one element of **E** at each environmental alteration, it is conceivable that selection for fitness robustness has given rise to an increase in modularity in their simulations. However, this is quite a different pressure than the supposed selection for enhanced evolvability.

In summary, Lipson et al. have presented an exciting and novel formalism that may yield quantitative, as well as qualitative insights into the evolution of evolvability and other problems. However, in their application of the model they have: (1) failed to demonstrate selection for modularity per se; and (2) not clearly established a link between modularity and evolvability. We suggest that enhanced evolvability can be neither a cause nor an outcome of the increase in their correlate of modularity.

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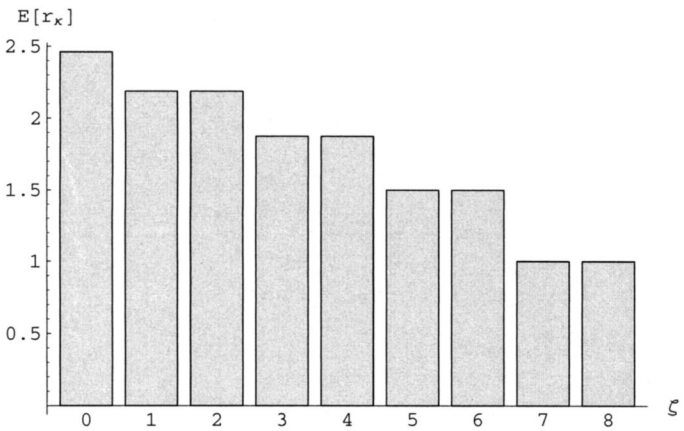

FIG. 2. The expectation of the residual r_κ as a function of ζ for an 8 × 8 ternary matrix. By ensuring that phenotype vectors are more tightly distributed around the origin, and hence closer to the optimum, matrix rows with more zero elements achieve reduced residual, on average.

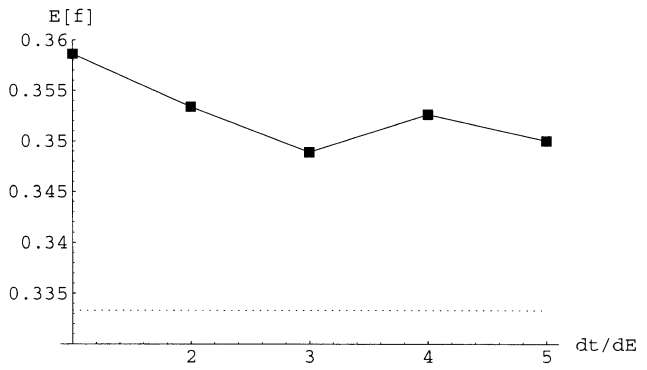

FIG. 3. The frequency of zero elements, averaged over 400 replicates, after 20 generations of evolution for a population of 50 8 × 8 matrices over a range of rates of environmental change dt/d**E**. The broken line indicates the null prediction 1/3. Simulations were devoid of mutation, but otherwise the evolutionary algorithm remained the same as that of Lipson et al.

TABLE 1. Simulation data and the one-tailed sign test for significant departure from null prediction "frequency of zero elements = 1/3".

dt/d**E**	Mean frequency of zero elements (from 400 replicates)	No. of replicates (out of 400) with frequency of zero elements >1/3	*P*
1	0.359	268	4.700×10^{-12}
2	0.353	243	9.979×10^{-6}
3	0.349	233	5.639×10^{-4}
4	0.353	250	3.266×10^{-7}
5	0.350	228	2.946×10^{-3}

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APPENDIX

The Distribution of $\mathbf{P}_\kappa$

A is a nxn ternary matrix (element values are −1, 0, and +1) and **E** is a *n*-element column vector with element values +1 and −1. The product of the premultiplication of **E** by **A** gives the phenotype vector **P**. The κ^{th} element of **P** is given by $\mathbf{P}_\kappa = \mathbf{A}_\kappa.\mathbf{E} = \Sigma_i \mathbf{A}_{\kappa i}.\mathbf{E}_i = \zeta.0 + m.(+1) + (n - \zeta - m).(-1)$ where ζ is the number of zero elements in $\mathbf{A}_\kappa$ and $m \sim Bin(n - \zeta, 1/2)$ is the number of same-sign pairs of $\mathbf{A}_{\kappa i}$ and $\mathbf{E}_i$ (i.e., those pairs of elements multiplying to give +1). Rearranging, the probability distribution of $\mathbf{P}_\kappa$ is found to be

$$\mathrm{P}[\mathbf{P}_\kappa = x] = \binom{n - \zeta}{\frac{n - \zeta - x}{2}} 2^{\zeta - n}, \tag{A1}$$

for $n = 8$, the distribution of P_κ as a function of ζ is shown in Figure 1.

E[r_κ*] as a function of* ζ

Lipson et al. define fitness as a decreasing function of the (scalar) distance between realized phenotype **P** and an arbitrary optimum **F**. The residual in the κ^{th} dimension is $r_\kappa = |\mathbf{F}_\kappa - \mathbf{P}_\kappa|$ where $\mathbf{F}_\kappa$ takes value +1 or −1 with equal probability. The probability density function of r_κ is then

$$\mathrm{P}[r_\kappa = y] = \frac{1}{2}\mathrm{P}[|\mathbf{P}_\kappa| - 1 = y] + \frac{1}{2}\mathrm{P}[|\mathbf{P}_\kappa| + 1 = y]$$

$$= \frac{1}{2}\Big(\mathrm{P}[|\mathbf{P}_\kappa| = y + 1] + \mathrm{P}[|\mathbf{P}_\kappa| = y - 1]\Big). \tag{A2}$$

Because P_κ is symmetrical about the origin, $\mathrm{P}[\mathbf{P}_\kappa = z] = \mathrm{P}[\mathbf{P}_\kappa = -z]$ and so for $z > 0$, $\mathrm{P}[|\mathbf{P}_\kappa| = z] = 2\,\mathrm{P}[\mathbf{P}_\kappa = z]$, that is, for $y > 1$,

$$\mathrm{P}[r_\kappa = y] = \mathrm{P}[\mathbf{P}_\kappa = y + 1] + P[\mathbf{P}_\kappa = y - 1]. \tag{A3}$$

For $y \leq 1$;

$$\mathrm{P}[r_\kappa = 1] = \mathrm{P}[\mathbf{P}_\kappa = -2]\mathrm{P}[F_\kappa = -1] + \mathrm{P}[\mathbf{P}_\kappa = +2]\mathrm{P}[F_\kappa = +1]$$

$$+ \mathrm{P}[\mathbf{P}_\kappa = 0] = \mathrm{P}[\mathbf{P}_\kappa = +2] + \mathrm{P}[\mathbf{P}_\kappa = 0]$$

$$\mathrm{P}[r_\kappa = 0] = \mathrm{P}[\mathbf{P}_\kappa = -1]\mathrm{P}[\mathbf{F}_\kappa = -1] + \mathrm{P}[\mathbf{P}_\kappa = +1]\mathrm{P}[\mathbf{F}_\kappa = +1]$$

$$= \mathrm{P}[\mathbf{P}_\kappa = +1]. \tag{A4}$$

Because $r_\kappa = \mathbf{P}_\kappa \pm 1$, and $\mathbf{P}_\kappa$ is restricted to values of the same parity as $n - \zeta$, r_κ is only evaluated for those integers with parity opposite to $n - \zeta$. For $n = 8$, the mean of r_κ is revealed as a function of ζ in Figure 2.