

An Overview of Modularity in Artificial Evolutionary Systems

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Abstract

Modularity in biological systems exists on many levels, however it is very difficult to define or quantify. Loosely, evolutionary modularity is the compartmentalization of genotype-phenotype interactions such that pleiotropy is suppressed among genes that do not serve like functions. Evolutionary modularity is attributed to evolvability, specialization, and canalization in genetic systems. These effects, though conceptually separate, are highly interrelated, and often share a bijective relationship with modularity (e.g. an increase in the selection pressure for evolvability also increases modularity as a side effect). Little is known about how genetic modularity arises in biology, however recent research points to processes such as gene duplication, secondary selection, and symbiogenesis as possible factors. Since modularity seems to play such an important role in biology, it should also be studied with respect to neuro-evolution. In particular, since traditional neuro-evolution methods do not employ anything more complex than a one-to-one genotype-phenotype map, how can simplicity be maintained when modularity is introduced in the system?

1 Introduction

Modularity plays an important role in developmental and evolutionary biology on many different levels. For example, at low levels genes are composed of chemical bases organized into groups of three (codons), codons in different arrangements code for specific amino acids, sequences of amino acids codify proteins, etc[5]. Similarly, on the phenotypic level, most morphologies are highly specialized and organized in a hierarchical structure of interacting, but on the whole, functionally separate, modules. These effects are far reaching, however for artificial evolution, modularity's effects on the level of the genotype-phenotype map are particularly of interest.

Modularity can be intuitively defined as “the integration of functionally related structures and the dissociation of unrelated structures” [4]. More technically, modularity refers to a mapping in which “there are few pleiotropic effects among characters serving different functions, with pleiotropic effects falling mainly among characters that are part of a single functional complex” [14]. This definition is also over-simplistic, because to fully understand module function, hierarchies, gradations, and the overlapping of modules must also be taken into account. Regardless, however, since independent functions are coded independently using modularity, each function can be optimized separately with little interference with other already optimized functions. Thus modularity not only encourages specialization and phenotypic and mutational stability (i.e. canalization), but also increases evolvability through hierarchical complexification.

Three major types of biological modules have been identified[15], although the degree to which they overlap has not yet been fully explored. *Developmental modules* are parts of the embryo that develop independently of each other, regardless of the context in which they occur. For example,

limbs, eyes, etc. These features share the same genetic data, so for instance a mutation affecting one limb would likewise affect the other, but once the embryogenic process starts, the two modules would become separate. *Evolutionary modules* are “sets of phenotypic features that are highly integrated by pleiotropic effects of the underlying genes and are relatively isolated from other such sets by a paucity of pleiotropic effects.” Or in other words modules arising statistically in the genotype phenotype map. Finally, *functional modules* are independent units of physiological regulation, for example independent parts of the metabolic network. Symbiogenesis is thought to play a role in this kind of modularity.

The primary focus of this paper is on evolutionary modules, since they are more often used in current neuro-evolution systems. Although the developmental process also affects the genotype-phenotype map, most of its effects can be simulated using evolutionary modularity. That is, reusable modularity can be achieved without the need for an expensive developmental system.

This paper will explore the various functions that modularity serves, how it arises in biological evolution, how module specialization can occur, and the implications that all this has for Neuro-evolution.

2 Role of Modularity in Evolution

Although details about their emergence in biology are still mostly unknown, the role modules play in evolution can be reasonably well explained. The key effect of modularity is decreasing pleiotropy among less related characters, which has several side effects: increasing evolvability, allowing for more complete specialization of function, and aiding in genetic canalization.

2.1 Evolvability

In their paper on the evolution of evolvability, Wagner and Altenberg describe modularity as a possible partial solution to the *representation problem* (i.e. how a problem can be encoded such that it is amenable to optimization by genetic methods), an analog to the *evolvability* of a system[14]. Determining the best genotypic representation for phenotypic data also affects the eventual shape of the fitness landscape that will be searched. If a representation is used in which favorable mutations cannot easily accumulate, then evolutionary optimization will function poorly. If, on the other hand, the problem is coded more favorably to evolution, then search through mutation, selection and crossover should perform better.

The key to overcoming the representation problem is ensuring that on the whole, mutations that improve one area of the system must not cause problems in other areas. With regards to the canonical genetic algorithm, this is referred to as reducing epistatic interaction[8]. In order for the building blocks hypothesis to apply, the genotypic representation should have low epistasis, and more generally there should be low pleiotropy among unrelated characters in the genotype-phenotype map. To avoid excessive pleiotropy, there must be some kind of modularization that partitions off parts of the genotype-phenotype map so that genes that specify separate functions have few interactions. This not only reduces the effects of the representation problem, but also ensures that mutations for one function only affect the representation of that function. This allows for strictly local optimization, and structures the genotype-phenotype map such that harmful mutations cannot have such widespread effects.

Evolvability can be defined as adaptive variability¹, or the genome’s ability to produce adaptive

¹Variability is a measure of how easily genotypic changes result in phenotypic changes. It should not be confused with the *variation* of a population.

variations when acted on by a genetic system. In the same vein as the representation problem, evolvability critically depends on the way genetic variation maps onto phenotypic variation.

Modularity is able to increase evolvability if functionally independent characters are also variational modules, in other words if modularization provides a good decomposition of the sub problems. The variational independence of distinct functional units avoids unwanted side effects if a functional unit undergoes some kind of adaptive evolution. Wagner provides a useful metaphor:

The situation is analogous to obtaining a verse of Shakespeare from monkeys banging away on typewriters. Typewriters make this far more likely than if the monkeys had pencil and paper. The typewriters at least constrain them to produce strings of letters.

Modularization of the genotype-phenotype map can lead to similar improvements (i.e. improve evolvability) in the types of phenotypes arising from genetic variation[14].

2.2 Specialization

In working out favorable representations in the genotype-phenotype map, modularity also provides a basis for the adaptation of different functions with little or no interference with other functions. This allows for the local optimization of each function, or, specialization.

Consider a gene-duplication scenario where a single gene is performing several functions prior to duplication. Then after duplication, the two independent copies are now able to more fully specialize without loss of phenotypic function. This is possible when the genetic information regarding two sub-problems can be decomposed into two independent modules. Of course, gene duplication is not necessarily required for specialization to occur. Another possible example is several genes performing the same family of functions, and over time evolution favoring a modularization in which each gene begins to take over a single function.

As modularity increases, functional interference decreases, and the capacity for a gene to specialize increases as a result.

2.3 Canalization

Evolutionary canalization is defined as phenotypic robustness against mutation. In biology, most genotype-phenotype maps are sufficiently canalized that small changes in genotype have little or no effect on phenotypes. In other words the genotype-phenotype map is not some kind of “house of cards,” rather, phenotypic variation occurs only when a significant number of mutations have accumulated. Canalization permits safer exploration of the genotypic space than would otherwise be possible[13]. Modularization of the genotype-phenotype map leads to one kind of canalization. Increasing evolvability by lowering detrimental variability has the side effect of making the genotype-phenotype map more robust to mutation, since the effects of genes will be highly compartmentalized.

Consider, for example, a highly adapted genotype-phenotype map where each module in the genotype serves a specific function and has a specific compartmentalized effect on the phenotype. A mutation on this genotype, as opposed to a mutation on a genotype where there is a high amount of pleiotropy, is going to have comparatively little expression in the phenotype. Therefore, canalization is directly correlated to evolvability. Specifically, there cannot be high evolvability unless the genotype-phenotype map is structured to protect against the effects of single mutations. However the map cannot be structured this way without being canalized.

This correlation between canalization and evolvability is just one manifestation of a larger correlation between all three effects. That canalization, specialization, and evolvability all arise

from modularity suggests that they are highly related effects, and that each one is necessary if the others are to be realized. This relationship also may play a role in the emergence of modularity; selection for evolvability, or selection for phenotypic stability may yield modularity as a result. However, whether or not these kinds of selection pressures exist in biological evolution remains to be seen.

3 Emergence of Modularity

Much research has been done recently on determining the origin of modules in biological evolution, however these results point in somewhat different directions [15]. The explanations for the emergence of modularity can be roughly divided into three categories: methods in which modularity is directly selected, or is a direct result of a related selective advantage, methods in which modularity arises strictly as a side effect of evolutionary dynamics, and methods where modularity arises due to external factors.

3.1 Direct Selection Methods

The existence of a selection pressure that gives rise to the emergence of modularity has been hypothesized, however none of these methods so far presented can completely explain the emergence of modularity. It should be noted, however, that a combination of several of these methods may lead to modularity in Nature. Several cases of how modularity could arise through direct selection are described here: selection for evolvability, constructional selection, selection for phenotypic stability (canalization), and “frustration” of genotypic characters.

3.1.1 Selection for Evolvability

Since embryogenic systems must have evolved at some point, it is most likely that the genotype-phenotype map is under genetic control, and is therefore evolvable. Thus variability, or the relative ability of a genome to generate variable phenotypes, is also under genetic control, and therefore, one would assume that there would be evolutionary strategies to evolve variability, i.e. strategies that increase or decrease variability based on the environment.

Since the expression of genetic variation is under genetic control, it is likely that one can also evolve strategies of how to structure the phenotypic effects of mutations. In other words, variability can be used to give rise to evolvability. This restructuring is accomplished through modularity, and thus modularity would increase if evolvability were selected for. Wagner found, however, that overwhelmingly, the selective advantage of suppressing pleiotropy coincided more with a direct selectional advantage in the phenotype rather than with increasing evolvability. He concludes, “even if natural selection can be effective in removing pleiotropic effects, the resulting increase in evolvability is not explained by selection for evolvability, i.e. it is not due to differences in genotypes.” [15].

3.1.2 Constructional Selection

Altenberg suggests that “genes with fewer pleiotropic effects have a higher probability to establish duplicated copies of themselves in the genome” [1] [15]. That is, it is more likely for independent genes to be expressed elsewhere, because they will have less far-reaching effects. This model also predicts that over the course of evolution, pleiotropy will decrease on average, which corresponds to genetic canalization.

The flaw of this model, admitted by Altenberg, is that it considers pleiotropy to be a heritable aspect of genes rather than an effect emergent in the genotype-phenotype map. The fact that genes do not usually contain their own pleiotropy information suggests that the canonical genetic algorithm with one-to-one genotype-phenotype mapping may be unable to make use of the correct set of selection pressures to encourage modularity.

3.1.3 Canalization

Selection for phenotypic stability, which can also be thought of as selection for canalization, is proposed by Ance and Fontana[3] as an operator that leads to modularity in the evolution of RNA secondary structures. This is provided as a counter-example to the thought that modularity cannot arise under stabilizing selection.

In RNA, there is a correlation between modularity and robustness against thermal noise, and mutation (i.e. modularity occurs as a resistance to sustained environmental or genetic perturbation[15]). Selection for phenotypic robustness yields mutational robustness and modularity as a side-effect. The primary rationale for this, is that without a modular architecture the phenotype has low plasticity, which can be easily selected against in RNA.

Ance goes on to note that modularity is not only a mechanism for evolutionary stability, but a tool for further complexification, since once the basic structure is modularized, the only way to innovate is by finding novel combinations of the modules, which in turn themselves modularize, etc.

3.1.4 “Frustration”

A final way that modularity can be selected for directly arises from a mathematic argument due to Rice[12]. For sets of two characters, correlation between the characters can either be positive or negative. That is, either one character increases directional selection on the other in a synergistic relationship, or one character decreases directional selection on the other, an antagonistic relationship. For sets of three characters with pairwise interactions, however, this can not be the case. In three dimensions it is impossible to have negative correlations among three or more characters simultaneously. The evolution of negative correlations is said to be “frustrated” [15]. In this case, the only stable solution is one in which the characters evolve variational independence.

A simple example of this phenomenon comes from the equation $C = x + y + z$, where C is a composite character, and is under stabilizing selection. In this case, there is antagonism between all three characters, and Rice’s theory predicts that selection will favor independence between the characters. If x were increased, then there must be a corresponding decrease spread out over y and z . Either they both will decrease, or one will decrease and the other will increase. In both cases, two of the characters would be have a positive correlation. Thus modularity could arise as from antagonistic fitness interactions among three or more characters.

3.2 Modularity Through Evolutionary Dynamics

In addition to the possibility of some kind of direct or indirect selection leading to a rise in modularity, another possible scenario is that modularity arises as a byproduct of evolutionary dynamics. Two possible cases are described here: gene duplication, and secondary selection (evolution on multiple time scales).

3.2.1 Gene Duplication

Calabretta et. al. [6][5] propose a system in which neural networks are evolved to control a Khepera robot in a “garbage collection” domain. This domain involves searching for objects in a confined environment, picking them up, and moving them outside the arena.

Three different neural network architectures are tested, a non-modular architecture, a *hardwired modular architecture*, and a *duplication based modular architecture*. The hardwired architecture is composed of two copies of the non-modular architecture, with an extra set of control output neurons to determine which copy is used for each output (left/right motors, pick up, and release). The duplication based architecture has the capacity for modularity, however, instead of having the modules hardwired into the topology, the modules can be added by duplicating the already existing module during the evolutionary process. It should be noted that in neither case is modularity directly selected for, although it is explicitly added to the phenotype.

Under these conditions, the two modular architectures outperform the non-modular architecture[11]. In addition, there is no clear evidence for the duplication based architecture performing any better overall than the hardwired architecture, although performance growth is slightly slower (this is attributed to the fact that it has to spend time initially growing the modules)[6].

Analysis of the evolved architectures yields significant differences, however. The modules in the hardwired architecture have no correspondence to sub-behaviors of the robot, whereas in the duplication based method, there is a much higher degree of specialization visible in the modules. Furthermore, there is evidence to support the hypothesis that different modules are used in different environmental situations (i.e. meaningful modularity).

The logical sequence of steps for this kind of modularity can be summarized as follows[9]:

1. A gene has several functions prior to duplication, these functions are not optimal because of conflicting adaptive demands.
2. The gene is duplicated.
3. The new genes are released from the conflicting demands and are able to further specialize.

Thus gene duplication can aid directly in modular specialization, however modular specialization alone does not account for a major increase in evolvability, since the hard-wired modular architecture performed just as well.

3.2.2 Secondary Selection

Kvasnička and Pospíchal propose an elegant example of how modularity could arise in nature, even in cases where some sort of direct or indirect selection for modularity does not occur. They show analytically that modularity can arise as a byproduct of evolutionary optimization on multiple time scales when evolving both the genotype and the genotype-phenotype map [10]. Specifically, short time scale genotypic evolution coupled with long time scale genotype-phenotype map optimization yields modularity in the genotype-phenotype map as a way to suppress interactions between decomposable sub problems.

Kvasnička performed a simple experiment in which two instances of the Kauffman NK function[2] were optimized simultaneously. The first half of the phenotype was used to seed the first Kauffman NK fitness function, and the second half of the phenotype used for the second. Initially, the genotype-phenotype maps are random, so optimization of one Kauffman function may effect the optimization of the other. This linkage is mainly detrimental, because the probability that both functions can be optimized the same way is very low. The evolution method used was:

- Randomly generate a population of epigenotypes (a genotype and a genotype-phenotype map).
- For a fixed amount of time, do:
 - Locally optimize the genotypes for each epigenotype using Pareto optimization.
 - Perform roulette selection and crossover on epigenotypes.
 - Mutate epigenotypes.

Thus, the genotypes evolve on a much faster time scale than the genotype-phenotype maps since they are completely optimized at every time step. Logically, this two time-scale evolution method gives rise to modularity because epigenotypes which allow local optimization (i.e. those with more modular structures) dramatically accelerate the evolution of genotype. Since the phenotype map is only evaluated on optimized genotypes, the main selection pressure that exists is one that encourages the suppression of interference between both halves of the problem, or modularization.

This result is important because it shows clearly how an evolutionary system that does not favor modularity explicitly can still be made to produce modular solutions when there is implicit modularity in the fitness function.

3.3 Modularity Through Symbiogenesis

A third method through which modular morphologies can evolve, separate from both direct selection and indirect emergence, is *symbiogenesis*, or the combination of two or more evolutionary distinct entities into a new larger system. This process of symbiotic composition is considered to be the method through which the complex interlocking systems, for example organs in higher animals, evolved.

Watson proposes an algorithm called the Symbiogenetic Evolutionary Adaptation Model (SEAM) in which symbiotic composition, not sexual recombination or accumulation of favorable mutations is the primary operator for evolution[16]. In this algorithm, entities start out specifying a single bit of genetic information, with all other bits unspecified. These entities are combined, at random, and if their combination represents a Pareto optimal solution under the current population, then the combination is admitted into the population. A simplified version of the procedure then is:

- Initialize ecosystem E with random, single feature entities.
- Repeat until some stopping condition:
 - Remove two entities a and b at random from the ecosystem.
 - Produce their symbiotic union, $a + b$, using composition.
 - If the union is *unstable* return a and b to the population, otherwise add $a + b$ to the population.

where a union is considered unstable if there exists another entity in the population θ that can form a better union than $a + b$ with either a or b .

Under the shuffled hierarchical if-and-only-if domain, Watson found that SEAM significantly outperforms both a one point crossover genetic algorithm with deterministic crowding, and the random mutation hill-climber.

In terms of modularity, symbiogenesis decomposes the problem into partially separated sub-problems, performs local optimization, and then constructs a solution based on the composition of

the two modules. Note that this can occur regardless of whether the problem is *separable* or *decomposable*. Separable sub problems are sub problems that can be expressed as two disjoint modules, whereas decomposable sub problems are for the most part disjoint, but may contain a non-trivial number of crosslinks. However, this decomposition occurs *before* composition, so symbiogenesis is a form of constructional modularization rather than decompositional modularization.

The main difference in symbiogenesis and other evolutionary methods is that the symbionts are already specialized and distinct, whereas modules begin homologous and become more distinct over time. Thus implicit evolutionary modularity may provide more benefits during early evolutionary stages than symbiogenesis.

4 Implications for Modularity in Neuro-Evolution

Modularity, if used correctly, could certainly benefit Neuro-evolution. However, much work has yet to be done comparing different methods that could lead to the emergence of modularity. Depending on the method used, modularity can be realized in different ways, for example in the duplication based modularity vs. hard-wired modularity experiment, it was found that modularity of functions only occurred under the duplication based method. Thus, several aspects of modularity, in particular, isolating the three effects of evolutionary modularity, and separating developmental and evolutionary modularity, could yield interesting experimental results.

4.1 Isolating the Effects of Modularity

In Nature, modularity primarily is a function of the mapping between the genotype and phenotype, and modular reuse is a function of both evolution, and the developmental system used to produce the phenotype. Under this dichotomy, modularity is responsible for providing a way for circumventing the representation problem, for decomposition and subsequent local optimization, and for canalizing the phenotype against detrimental mutation. These three effects are intrinsically linked in biology, however in computer science, it may be worthwhile to pull them apart and study them individually. For example, is there a way to allow for modularity without canalization, or strict specialization without the positive effects on evolvability and phenotypic stability?

4.2 Separating Developmental and Evolutionary Modularity

Several NE methods have been proposed that allow for reusable modularity in artificial developmental systems. For example, Bongard designed a system for Artificial Ontogeny[4], and Gruau et al. designed a method using cellular encoding[7], however little work has been done to study reusable modularity with a more neutral genotype-phenotype map. Since reusable genetic modularity can arise in the presence of just a simple genotype-phenotype mapping, it follows that studying modularity in a genetic system without a complex developmental system (such as the canonical genetic algorithm) would be worthwhile. This way, evolutionary modularity could be abstracted away from developmental modularity, and its effects could be studied in isolation, highlighting the limitations of purely evolutionary modularity.

4.3 Analysis of the Modular Neuro-Evolution Method

One way of allowing for a modular NE method is to explicitly allow for modularity and reuse in the genotype by creating a population of modules (partial genotypes) and chromosomes (genotype-phenotype maps). These two populations undergo cooperative coevolution to arrive at optimal

compositional solutions. This system allows for serial modular decompositions, but does not allow for hierarchical decompositions, which may be critical in exploiting phenotypic regularities such as bilateral symmetry.

The main downfall of this system may be that modularity does not develop implicitly, rather it is an explicit component from the start of evolution. Because of this, rather than starting with monolithic genotypes and specializing and canalizing genes into modules, the evolutionary system is given hundreds of modules and asked to put them together into a working solution, much like symbiogenesis. This severely retards module specialization until at least a partially useful decomposition has been found and therefore the system cannot completely canalize until much later in evolution. Since this also reduces evolvability early during the evolutionary cycle, the modular NE method may take longer to converge than more traditional NE methods.

One possible solution to this problem is to use some form of secondary selection in conjunction with the symbiogenetic composition and to find the optimal module population for a population of chromosomes at one time step. This constrain-and-evolve method could also work the other way around, i.e. constraining the module population and evolving the chromosomes.

5 Conclusion

By decreasing pleiotropy among disjoint functions, evolutionary modularity has been attributed to increasing the overall evolvability of a genotype, allowing separate functions to optimize individually, and to further canalizing the system against harmful mutations. Building on already modularized components, more complex genomes can be searched without massive genetic changes. Since modularity seems to be the key to solving the representation problem and providing for unbounded complexification in the form of evolvability, understanding the factors that contribute to it is necessary to further the field of artificial evolutionary systems.

Several methods have been proposed for how modularity arises internally in natural systems. These methods generally fall under two categories, methods which employ some kind of direct selection for modularity, and methods that do not. Another category that can possibly give rise to modularity occurs external to the genotype-phenotype map. This is symbiogenesis, or the combination of distinct evolutionary lineages into one system. It has been shown that modularity cannot arise from direct selection for a single feature, though it may arise from a combination of selections for several features. More likely, however, modularity arises from a mechanism implicit in evolution, such as genetic duplication, or evolutionary optimization on more than one time scale.

Finally, outside of gene-duplication, little has been done to study evolutionary modularity applied to NE. Although several methods exist utilizing developmental modularity, Artificial Embryogeny had not yet been well mapped out, so these methods may suffer from unnecessary complication. Studying other systems like secondary selection and symbiogenesis applied to NE may yield more efficient algorithms.

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