Research Article

Effects of Antimodularity and Multiscale Influence in Random Boolean Networks

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We investigate the effects of modularity, antimodularity, and multiscale influence on random Boolean networks (RBNs). On the one hand, we produced modular, antimodular, and standard RBNs and compared them to identify how antimodularity affects the dynamical behaviors of RBNs. We found that the antimodular networks showed similar dynamics to the standard networks. Confirming previous results, modular networks had more complex dynamics. On the other hand, we generated multilayer RBNs where there are different RBNs in the nodes of a higher scale RBN. We observed the dynamics of micro- and macronetworks by adjusting parameters at each scale to reveal how the behavior of lower layers affects the behavior of higher layers and vice versa. We found that the statistical properties of macro-RBNs were changed by the parameters of micro-RBNs, but not the other way around. However, the precise patterns of networks were dominated by the macro-RBNs. In other words, for statistical properties only upward causation was relevant, while for the detailed dynamics downward causation was prevalent.

1. Introduction

The structure of a network can significantly alter its function or behavior. Modularity is a structural property prevalent in many systems, where elements within modules have more connections among themselves than with other elements [1, 2]. Extrapolating from the notion of modularity, we can define antimodularity when elements in subsets have less connections among themselves than with other elements. Modularity has been extensively studied. It is related to the complexity of the systems and their capacity to evolve and adapt, but at the same time to resist perturbations in a robust fashion [3–7]. It maintains robustness through isolation of modules and allows faster adaptation via altering the connections among the modules. On the other hand, what would be the functional properties of antimodular networks?

In addition to modularity, another relevant property of complex systems is the causality between scales in a multilayer structure. It has attracted much attention in systems and computational biology how changes at lower layers affect system properties at upper layers and vice versa [8–10]. Gene therapy is an example showing “upward” causality, where the therapy at the genetic level cascades to an effect on upper levels such as tissues and organs. An example of “downward” causation would be the behavioral choices of an animal selecting a particular mate, which will partly determine which genes will be preserved in future generations.

In this study, we aim to investigate the effects of antimodularity and causality between layers in random Boolean networks (RBNs). Because RBNs have been used as models to represent dynamics of gene regulatory networks [11–13], our research could help better understand the characteristics of many living organisms showing modularity/antimodularity and causality between upper and lower levels [6, 9, 14].
2 Complexity

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Figure 1: An example RBN ($N = 6$, $K = 2$). The nodes and links are represented by circles and arrows, respectively.

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(a) (b) (c)

Figure 2: Adjacency matrixes and networks of standard, modular, and antimodular RBNs. All the networks consist of 6 nodes ($N = 6$) and 12 links. In the network, each node has two input nodes on average ($K = 2$). In the adjacency matrixes, blue means that there is a link between corresponding nodes, and yellow represents that there is no link in the defined module. (a) A standard RBN. The links are randomly distributed. (b) A modular RBN with three modules (AB, CD, and EF). There are 9 intramodular links and 3 intermodular links. (c) An antimodular RBN. The same modules as (b) are assumed. There are 3 intramodular links and 9 intermodular links.

2. Materials and Methods

2.1. Random Boolean Networks. RBNs were suggested as gene regulatory network models by Kauffman [11–13]. A RBN is composed of $N$ nodes and $K$ connections per node (or $K$ input nodes). An example RBN is shown in Figure 1. The nodes of the network can only have states of zero (off, inhibited) or one (on, activated). In RBNs, Boolean functions and topologies are randomly arranged. The states of the nodes are determined by the states of input nodes and the logical rules randomly assigned to each node.

2.2. Modularity/Antimodularity of RBNs. A module is a set of nodes that are more densely connected to each other than to nodes from other modules [14, 15]. Based on this definition, we produce modular, antimodular, and standard RBNs. Specifically, centering on diagonal elements in the adjacency matrix of a RBN, we define modules and then arrange links inside and outside the modules. Here we only consider modules which have the same size. Depending on node sizes, the number of modules to be defined in a network can be different. For example, in Figure 2, there are four possible homogeneous modules: 1 (i.e., ABCDEF), 2 (i.e., ABC, DEF), 3 (i.e., AB, CD, EF), and 6 (i.e., A, B, C, D, E, F), although the first and last are trivial, representing the network and node levels, respectively.

The three types of RBNs are as follows:

(i) Standard RBNs: regardless of modules, links are randomly distributed in the adjacency matrix. Figure 2(a) shows an example of a standard RBN.

(ii) Modular RBNs: a greater number of links are randomly placed inside the modules than outside the modules with probability $p_m$. Figure 2(b) represents an example of a modular RBN with three modules, where each module consists of two nodes.

(iii) Antimodular RBNs: contrary to modular RBNs, a larger number of links are distributed outside the
defined modules than inside the modules with probability $p_m$. Figure 2(c) refers to an example of an antimodular RBN.

In this study, we consider arbitrarily $p_m = p_o = 0.9$.

We quantitatively measure modularity $M$ as follows:

$$M = \frac{\%i}{\%o} \quad \text{while} \quad \%o > 0,$$

where $\%i$ is the percentage of possible links within the defined modules (intermodular) and $\%o$ is the percentage of possible links outside the modules (intradmodular). If there are no connections outside the modules, that is to say $\%o = 0$, it is considered that the network is infinitely modular, because modules are completely isolated. Figure 3(a) presents an example showing how to calculate the modularity. Antimodularity $\bar{M}$ is defined by the following equation:

$$\bar{M} = \frac{1}{M}.$$  

To check the realism of the modules we define and the modularity measure, we compare our modular/antimodular RBNs with biological Boolean networks. In the case of biological networks, we find their modules using Fortunato’s community detection algorithm [16], which allows the detection of homogeneous and nonhomogeneous modules. Figure 3(b) is an example showing how to compute the modularity in nonhomogeneous modules.

2.3. Multilayer RBNs. To study the causality between upper and lower levels, we developed multilayer RBN model where there are different RBNs in the nodes of a higher scale RBN. We call the meta RBN macronetwork and the RBNs in the nodes of the macronetwork micronetwork. The macronetwork consists of disconnected RBNs, although they may interact through the macronetwork (the $M$ of the macronetwork would be infinite). In our model, the node states of the macronetwork and micronetworks are updated through the interactions between them. Specifically, the following processes are repeated:

(1) Random initial states are assigned to micronetworks (Figure 4(a)).

(2) The node states of the micronetworks are updated at the microlevel during $t$ time steps (Figure 4(b)).

(3) The node states of the macronetwork are determined based on the node states of micronetworks at time $t$. If the node states of the macronetwork in the node of the macronetwork have a larger number of 1s (0s), the node state of the macronetwork becomes 1(0). In other words, the state of the nodes of the macronetwork is determined by the majority of the nodes of each lower scale RBN. (Figure 4(c)).

(4) The node states of the macronetwork are updated during $T$ time steps. The node states of the macronetwork at time $T$ determine the node states of each RBN at the lower scale. If the node state of the macronetwork is 1(0), the states of all its lower scale nodes become 1(0) (Figure 4(d)).

2.4. Experiments. To investigate the effect of modularity/antimodularity, we performed 100 independent simulation runs for each parameter combination. For each simulation, we generated modular, antimodular, and standard RBNs composed of 240 nodes. Because the number of nodes is 240, all the possible numbers of modules to be defined in the network are $2, 3, 4, 5, 6, 8, 10, 12, 15, 16, 20, 24, 30, 40, 48, 60, 80, 120, 240$. Varying $K$ from 1 to 7, we measured modularity, the number of attractors, the average length of attractors, and complexity for all the cases of modules.

Here complexity was calculated based on our previous approach [17, 18]. Complexity $C$ ($0 \leq C \leq 1$) is defined as follows:

$$E_i = -(p_0 \log_2 p_0 + p_1 \log_2 p_1)$$

$$C = 4 \times E \times (1 - E)$$

where $E_i$ is the emergence of node $i$ and $p_{0(1)}$ is the probability that the state of the node is 0 (1). $C$ is the complexity of the network. It is calculated based on average ($0 \leq E \leq 1$) of the emergence values for all the nodes. 4 is added to normalize the values of $C$ between 0 and 1.

Figure 5 shows how complexity is affected by the number of nodes of a network. Although the number of nodes increases, the networks have a similar trend: complexity
increases until reaching the maximum at $K = 2.3$ and after the peak, it decreases monotonically with a growing $K$. However, the precise complexity values do change with the network size, probably due to a finite size effect, which is clearly seen around $K = 2.3$.

To examine the causality between micro- and macronetworks, we conducted 55 independent simulation runs. For each simulation, we produced a multilayer RBN model where each micronetwork has 125 nodes, and the macronetwork has 55 nodes. In the simulation, $T$ of the macronetwork was set to 3. $T$ of the micronetworks was set to 1, 2, 3, 5, and 10. Changing $K$ from 1 to 7, we measured complexity of micro- and macro-RBNs.

### 3. Results and Discussion

Figure 6 shows modularity, numbers of attractors, complexity, and lengths of attractors for modular RBNs. In a first set of experiments, as $K$ increased, the modularity decreased. In the three plots of modularity, when the modules are more (i.e., the size of the modules is smaller), the modularity drops overwhelmingly as the connections ($K$) increase. This is because a greater number of links end up being placed outside the modules.

To illustrate how links are distributed in modular networks, we present a few simple examples in Figure 7. The figure represents link distributions in the modules depending on $K$ and module size. As mentioned in the Introduction, $K$ is
the number of connections per node. Thus, in the examples, we assume that \( K \) connections are distributed per row in the modules. If \( K \) is larger than the module size, the rest of the connections are placed outside the modules, which causes the decrease of modularity. As seen in the figure, as the module size gets smaller from 4 to 1 (i.e., the number of modules increases), a tendency can be identified.

In a second set of experiments, the number of attractors is limited to a maximum number of 1,000. In other words, if 1,000 attractors are found, the search for attractors is halted. As seen in the figure, the numbers of attractors found were zero at \( K > 3 \), which resulted from very long transients and attractors caused by chaotic dynamics of RBNs. Meanwhile, the attractors increased as the number of modules grew at \( K = 2 \). In the case of 80 and 120 modules, the maximum number of 1,000 attractors was found. However, when the number of modules was 240, the numbers of attractors dramatically decreased.

In a third set of experiments, the complexity gradually increased until reaching their peaks at \( K = 2.5 \), which is expected as complexity is maximized at the phase transition in standard RBNs; i.e., it reflects criticality. Since then the values started to decrease regardless of the number of modules for the most cases. However, the complexity was affected by the number of modules for a few cases. When the numbers of modules were 40, 48, and 60, the complexity values were the largest, especially in regions where chaotic dynamics would be expected. When the numbers were 120 and 240, the values decreased. This confirmed our previous results [4], where we saw that modularity extends the critical regime (with a high complexity) towards the chaotic region.

In another set of experiments, the lengths of attractors were the highest at \( K = 2 \) but converged to zero when \( K \) was larger than 3. Except for 120 and 240 modules, the more modules a network had, the more increased the lengths of attractors were. When the modules were 120 and 240, the values were decreased.

Figure 8 shows characteristic examples of state transitions depending on the number of modules. As seen in the figure, the states of the network with 240 modules converged to the attractor faster than the states of the network with two modules.

Figure 9 represents numbers of attractors, complexity, and lengths of attractors for antimodular RBNs. The numbers of attractors gradually increased and reached the peaks at \( K = 2 \) as the number of modules grew (Figure 9(a)). Since then, the values decreased and started to converge into 1 from \( K = 3 \).

For the complexity, the values had little variation against the number of modules (Figure 9(b)). That is, modularity did not affect complexity in antimodular networks. The complexity increased until reaching the maximum at \( K = 2.3 \). After that, it decreased monotonically as \( K \) grew. The lengths of attractors were not affected by the number of modules (Figure 9(c)). The values increased until reaching their peaks at \( K = 2 \). After that, it started to decrease and became zero for \( K \) larger than 3. Figure 10 shows the complexity of standard and antimodular RBNs. The complexity of antimodular RBNs was very similar to that of standard networks.

To compare the results acquired from our modular/antimodular RBNs and the properties of biological networks, we collected seven biological networks from CellCollective.org [19–25]. Figure 11 shows the adjacency matrix and modularity of one biological network related to treatment of infections. In the adjacency matrix, blue and red boxes indicate nonhomogeneous modules detected by Fortunato’s algorithm.

Figure 12 shows the complexity and the modularity of seven biological networks. To study the correlation between complexity and modularity, we calculated Pearson correlation coefficient (\( r = 0.4764 \)). Figure 13 illustrates the correlation graphically. We found that the complexity and the modularity have a moderate positive correlation. This result
Figure 6: Modularity, numbers of attractors, average length of attractors, and complexity for modular RBNs.
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**Figure 7:** Link distributions depending on $K$ and a module size.

**Figure 8:** State transitions of networks with $N = 240$ and $K = 2$ depending on the number of modules. The states were calculated during 100 timesteps. Time moves downward, columns represent nodes, and colors show states (white represents 0 and black represents 1). (a) Network with 2 modules. (b) Network with 240 modules.

**Figure 9:** Continued.

**Attractors**

- Mod4
- Mod5
- Mod6
- Mod8
- Mod10
- Mod12
- Mod15
- Mod16
- Mod20
- Mod24
- Mod30
- Mod40
- Mod48
- Mod60
- Mod80
- Mod120
- Mod240

**Complexity**

- Mod4
- Mod5
- Mod6
- Mod8
- Mod10
- Mod12
- Mod15
- Mod20
- Mod24
- Mod30
- Mod40
- Mod48
- Mod60
- Mod80
- Mod120
- Mod240
Figure 9: Numbers of attractors, complexity, and lengths of attractors for antimodular RBNs.

Figure 10: Complexity of standard and antimodular RBNs. (a) Standard network. (b) Modular networks. (c) Overlapped plots.
Potential intramodular links = 75
Potential intermodular links = 70
Intramodular links = 27
Intermodular links = 1

(a)

(b)

**Figure 11:** Adjacency matrix and modularity of a biological network related to infection treatment.

**Figure 12:** Modularity and complexity of seven biological networks.
supports our finding that the number of modules has an impact on complexity in the modular RBNs.

Figure 14 shows heat maps on the complexity of micro- and macro-RBNs. When $K$ of the macronetwork (macro-$K$) was larger than 4, its complexity was changed by $K$ of the micronetworks (micro-$K$). For micro-$K$ smaller than 2, the complexity was close to zero. However, as the value of $K$ increased, the complexity became higher. This trend stayed the same although $t$ (i.e., the period for the update of the node states of the micronetworks) was varied. In other words, the dynamics of the lower scale affected the higher scale only when ordered dynamics were present at the lower scale: they inhibited the expected chaotic dynamics of the higher scale.

On the contrary, the complexity of the micronetworks was not influenced by macro-$K$ but mostly depended on the micro-$K$. The complexity of micronetworks increased until micro-$K$ became 2. Since then, the value decreased as micro-$K$ got bigger. This trend was manifested for larger $t$. Thus, downward causation was not detected in this study for complexity, or at least an effect of the macronetwork on the micronetwork was not detected.

As another experiment for multiscale influence, an analysis of the attractors was performed. What can be seen in Figure 15 is that the attractors of micro- and macronetworks are similar but for the micronetwork are shorter. This result indicates that the attractors of the macronetwork dominate the dynamics of the micronetwork.

Let us recall that an attractor is a set of states that are repeated. If a timestep of the macronetwork does not produce a change in a particular node, this will force its micronetwork to back into a state of only ones or zeroes. Thus, a regular pattern will be observed even if the micronetwork is chaotic. In other words, without the macroinfluence, it would be actually in a transient.

Even if there is a change in the node of the macronetwork, this would push its micronetwork to back into one of two possible states. Thus, on average micronetworks’ attractors are shorter because macronodes that do not change force the same initial state over the micronetwork, while the macronetwork can have longer attractors composed of the combinations of the microattractors, as shown in Figure 16.

4. Conclusions

Our computational experiments showed that modularity increases the “diversity” of the network dynamics. That is, there are more and longer attractors as the number of modules increases. For the antimodularity, the increase of the modules does not influence the length or number of attractors.

Networks with a median number of modules extend the complexity of RBNs. All networks have a “critical” region where complexity is high (around 2.3 for standard networks due to a finite size effects, as the theoretical phase transition occurs at $K = 2$ at the thermodynamic limit, i.e., for infinite networks). Networks with very few or a lot of modules have topologies which are more similar to standard topologies, while a median number of modules implies a structure that allows for a better balance of robustness and adaptability, which is precisely a characteristic of standard RBNs with a high complexity. We can conclude that modularity “extends” criticality in RBNs, since our measure of complexity is maximal at the critical region of parameters (not only in RBNs).

Antimodular RBNs showed similar dynamics as standard RBNs. We can infer the reason from Figure 17. In the figure, adjacency matrixes of standard, modular, and antimodular RBNs are presented. In the adjacency matrixes of the standard network and the antimodular one, a greater number of links are distributed outside the diagonal modules. Because the antimodular network has relatively larger space for the distribution of links in the adjacency matrix, especially as the network size increases, it is more similar to the standard network where the links are randomly arranged compared to the modular network.

For the causality between micro- and macro-RBNs, the complexity of the macronetwork was varied by $K$ of the micronetworks when $K$ of the macronetwork was larger than 4. This trend was maintained irrespective of the period for the update of the node states in the micronetworks. Meanwhile, the complexity of the micronetworks was only determined by their own structure. This suggests that there is no downward effect between the two scales in the complexity analysis.
Figure 14: Heat maps of the complexity of micro- and macro-RBNs.
However, the study of the attractors for the multiscale networks showed that the attractors in the macronetwork affect how the micronetwork behaves. Our complexity measure is based on Shannon’s information, and thus it is a statistical approximation of the dynamics of a network. In other words, it does not distinguish the precise arrangement of bits and just focuses on their probability distribution. For example, a random bit sequence has the same information (maximal) and complexity (minimal) than an ordered string which has precisely one-half of all zeroes and another half of all ones.

In our experiments, the statistical properties of the network dynamics (i.e., the complexity) were determined mainly by the lower scale. However, the precise order of the dynamics (i.e., the attractors) was determined mainly by the higher scale.

Our results suggest that studying only the lower scale of systems would be meaningful only if we are interested in certain statistical properties. Nevertheless, if we want to understand and attempt to predict the precise dynamics, we need to study both scales and how they interact. This can be illustrated with the classical example of different arrangements of carbon atoms, which are the same at the lower scale, but depending on their structure at the higher scale (charcoal, diamond, graphene, etc.) they can have very different properties at both scales. Still, studying whether our results for RBNs can be generalized to all phenomena is an ambitious task which is beyond the scope of this paper.

For further study, we plan to investigate a relationship between modularity/antimodularity and diverse topologies such as cyclic or star shapes and multiscale effects of micro- and macronetworks with different structures. Also, we will study how to control the states of modular, antimodular, and multilayer networks so that our research can be applied to more fields. Specifically, using mathematical approaches like the technique of semitensor product (STP) [26–28], we will examine the dynamics of networks and design controllers for them.

**Data Availability**

Our simulator and data are available at https://github.com/angelEsc/RBN. Biological networks were obtained from https://research.cellcollective.org.
Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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