

How creative elements help the recovery of networks after crisis: lessons from biology¹

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In the middle of a world economic crisis, studies of biological crisis-survival strategies, which have proven their efficiency over a billion years of evolution, may provide novel ideas and solutions. In this chapter, we summarize our knowledge and the latest results which show that the “crisis of cells,” called stress in biology, and its extreme form, programmed cell death (also known as apoptosis), induce a decrease in the inter-modular contacts of protein–protein interaction networks of yeast and human cells, respectively. Moreover, programmed cell death leads to the decomposition of several key modules of human cells. This network disintegration fits well with the general scheme of network topological phase transitions upon a decrease of resources (and/or an increase of perturbations, stress). The modular disassembly resembles changes to social networks in crisis-like situations. Thus, the re-association of cellular networks after stress and the necessity of mobile, inter-modular elements (“creative elements”) for this process of “modular evolution” may give us adaptable schemes for crisis-survival strategies, proving the old observation that a crisis is not a disaster but a challenge providing a chance for development.

10.1 How can biological networks help our coping with crisis situations and our understanding of social networks?

From the second half of the twentieth century, the increasing complexity and the information-richness of our everyday life have got closer and closer to the limits of the information handling capacity of our logical, left-hemisphere brain. The Western civilization has been increasingly using (and actually, over-using) logical thinking

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from the very beginning and, especially, from the Enlightenment of the eighteenth century, signaling the birth of modern scientific thinking. Our schools socialize us and our children to accommodate a conceptual framework, which makes the logical assessment of the changing situations a Pavlovian conditioning of modern times. This way of human cognition is efficient, useful, and has enabled a lot of progress in the past centuries. However, the plethora and complexity of information we “enjoy” today easily saturates the left hemisphere of our brain, which is responsible for logical thinking. Our brain usually has only a few cognitive dimensions (five to seven in males and a slightly higher number in females; Dunbar, 2005), meaning that our left hemisphere can tackle only a few independent ideas simultaneously. The current information load easily surpasses this amount. The situation becomes even worse in times of crisis, since in crisis the generation speed of novel information, the need to get more and more information, and the number of seemingly important pieces of information all increase, which magnifies the perceived information load. We may sum all of this up in a rather extreme form: in crisis the logical brain gives up and emotions, instincts, and panic-like herding behavior become dominant.

How can networks help to cope with panic-like behavior in crisis-prone situations? Besides the logical analysis of the left brain hemisphere, the subconscious summary of billions of previous experiences in the form of instincts or emotions, and giving up any analysis, and simply copying what others do, herein we have another way of information perception, the holistic, pattern-like recognition of the right brain hemisphere. With this ability we “immediately” recognize the focal points of an incomprehensibly large dataset – as though it were converted to a picture-like form.

Networks became an increasingly popular method of analysis in recent decades, “conquering” scientific disciplines one after the other from the starting point, sociology. One of the major reasons for this network-revolution is that networks provide a dual way of cognition: they both allow the assessment of the dataset from an image (the visual image of the network), and provide an easy and straightforward path to “number-crunching” of the most important segments. In the visual images of networks we can easily recognize their

- hubs (Barabási and Albert, 1999);
- communities (Girvan and Newman, 2002; Kovács *et al.*, 2010; Palla *et al.*, 2005);
- bridges connecting distant elements and distant communities (Watts and Strogatz, 1998; Csermely, 2009);
- the highly versatile, inter-modular “creative elements” (Csermely, 2008),

and many other key features. After the identification of these points, networks provide a fast way to identify the exact data, such as the identity of the elements, the strength of their links, etc. Most of the above, key structural segments can

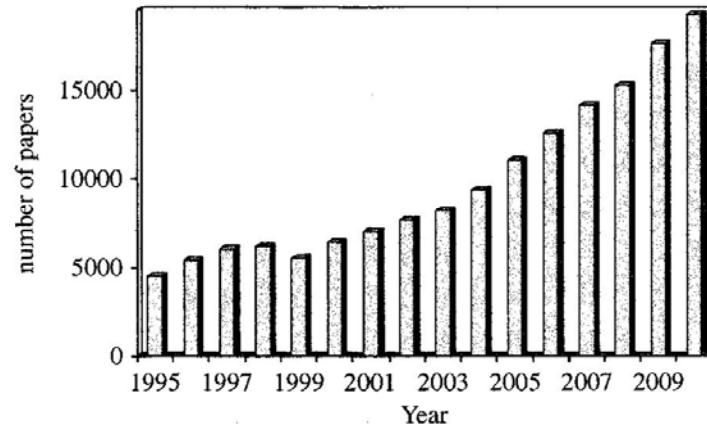


Figure 10.1. Propagation of the network method. The number of publications containing the words “network” or “networks” in their title or abstract was collected from the database www.pubmed.com. The 2010 data is an extrapolation. Notice that the two seminal papers on networks were published in the late nineties (Watts and Strogatz, 1998 and Barabási and Albert, 1999). We should highlight that the quality of data are somewhat compromised since the word “network” may also refer to scientific collaboration networks, and the total number of articles per year in PubMed also increased over this period.

also already be assigned using precise algorithms. With these dual possibilities, networks help to mobilize both hemispheres of our brains to cope with the increased information-load in crisis.

The search for the “essence” is as old as human culture itself. Why would networks provide a better solution for this evergreen problem than the myriads of other approaches? Their visual image alone would not explain the tremendous success networks enjoyed and enjoy in the last decade (Figure 10.1). The visualization of the hundred-million users of a mobile-phone network would make a rather incomprehensible picture, even with the hierarchical zooming-in techniques of most modern visualization options (Kovács *et al.*, 2010; Pavlopoulos *et al.*, 2008). In past decades, one of the most important and most surprising conclusions of network studies was that several key network features are highly similar in many networks which describe entirely different complex systems. Thus, networks of macromolecules, cells, organisms, ecosystems, and social networks, are similar

- in their small-worldness (Watts and Strogatz, 1998);
- in their close-to scale-free degree distribution (Barabási and Albert, 1999);
- in the hierarchy and nestedness of their layers of increasing complexity (Ravasz *et al.*, 2002);
- in the stabilizing role of their weak links (Csermely, 2009), and in many other features.

This similarity allows for very exciting play: if we find a novel feature of a network of a certain complex system, we may try to find a similar feature of a network of an entirely different complex system. This not only gives a broader scope to our findings, but also allows us to grade their importance. Highly specific network features may not claim general applicability. Additionally, these trans-disciplinary, network-aided studies also help our general understanding. Human cognition is many times hindered by the cognitive barriers of our words. The meaning of words requires a certain context. This gives a rich texture of meaning, but also imprisons the meaning giving it limited scope. Let me give an example. If I say, “aging” every one of us immediately imagines something, an old grandpa, wrinkles, whatever. This immediate recognition of aging as a human feature makes us uneasy about thinking about questions like: “Does the Internet age?”; “Is the structure of our society aged?”; “Can the ecosystem of the whole Earth, Gaia age?” Network science may give us generally applicable tests for phenomena like aging, which make such questions testable. In our chapter we will sum up our knowledge and our own results, giving another example, where a network of a certain layer of complexity (i.e. the protein–protein interaction network of our cells), can teach us patterns (i.e. in this case cellular crisis-recovery patterns), which can be applied to an entirely different situation, the recovery from the current economic and social crisis.

10.2 Crisis responses of networks

At the time of writing this chapter, world-economy gurus had started to convince each other that we had slowly emerged from the 2008 monetary and economic crisis. We will start to get those datasets whereby the 2008 crisis and recovery behavior of human societies can be studied only some years later. However, we need answers now. Regrettably, during previous crisis events data collection and analysis were not at the point that they are today, and we have only very few reliable network-like datasets, useful for studying social crisis events of the past. What can we do in such a situation? We may use the analogies described in the introduction to our chapter and examine crises in other networks in the hope that the features we find will be applicable to social networks.

The crisis in cells may provide a good example of this cross-disciplinary approach. When does a cell suffer a crisis? The easy answer is: “always”. An event of cellular crisis is called stress. We can induce cellular stress rather easily. We just have to heat or chill the cell, provide too much or too little salt, sugar, oxygen, etc., all of these will lead to a cellular crisis, if we apply them abruptly enough. Our own studies examined the changes in yeast and human protein–protein interaction networks under various forms of stress.

In protein–protein interaction networks, elements are cellular proteins and their links are physical interactions between them. Regrettably, the yeast protein–protein interaction network (interactome; Stark *et al.*, 2011) has not yet been measured after stress. Therefore, we had to choose an indirect method: we modeled the changes in weight of protein–protein interaction links as changes in the abundance of the two messenger ribonucleic acids (mRNA-s) coding the two constituting proteins in stress (Gasch *et al.*, 2000; Holstege *et al.*, 1998). The multitude of our control examinations (Palotai *et al.*, 2008; Mihalik and Csermely, 2011) and other studies (Halbeisen and Gerber, 2009) show that this assumption holds fairly true. We analyzed the overlapping modules of the yeast interactome using our ModuLand method (Kovács *et al.*, 2010). In the first phase of work we had dozens of highly lucrative hypotheses. However, after the application of various controls (different stress types, starting conditions, interactomes, averaging and weight calculating methods, etc.) all but one of our ideas were proved to be false. The only finding, which survived all controls was that the overlap of yeast interactome modules significantly decreases in many types of stress, including heat shock, osmotic shock, and oxidative stress.

Does a decrease in modular overlaps have any importance? Modules of yeast protein–protein interaction network correspond nicely to various key functions of the yeast cell, such as protein synthesis, protein degradation, regulation of the cell cycle, and others (Palotai *et al.*, 2008; Kovács *et al.*, 2010). A decrease in the overlap means that we have many fewer proteins in stress, participating in more than one cellular function. This helps cellular adaptation to stress in the following ways:

- A decrease in inter-modular links is an important factor in sparing system resources, since the maintenance of all contacts requires additional resources from the cell. Energy is a very high-cost item during cellular crisis, when mitochondria (the cellular power stations) become damaged.
- A decrease in overlap between network communities enables more independent functioning. This increases the adaptation potential of the cell, since various segments may explore a larger adaptation space, if they are no longer bound so tightly to each other.
- The decrease in overlap prevents the easy spread of damage. If a cellular module becomes damaged (e.g. one of the proteins is oxidized by the free radicals coming from aberrant mitochondria, the free radicals emanating from the oxidation of this protein will not damage its previous partner in the adjacent module). Similarly, the increased noise coming from the malfunctioning of damaged proteins, membranes, and other cellular components will not disturb all of the cellular communities, since it is arrested at the modular boundaries (Szalay *et al.*,

2007). This latter feature makes isolation of modules similar to the well-known quarantine applied to prevent the spread of infectious maladies.

If cellular crisis is extremely severe, cells often go along a programmed cell pathway called apoptosis (Sóti *et al.*, 2003). This death is altruistic, since apoptosis makes a “neat package” from the cell, preventing the spread of its internal content to the neighborhood. The alternative pathway, cellular necrosis, leads to a break in the cell wall, and to widespread inflammation due to the leak of the internal cellular content.

In programmed cell death, a family of protease enzymes, called caspases, cleaves several hundreds of cellular proteins. Many of these proteins are essential for cellular life, therefore one may wonder why we need cleavage of so many of them. In our studies, we examined the changes in protein–protein interaction networks of human cells after the removal of these caspase-substrate proteins, cleaved during the process of programmed cell death. To make a long story short, we concluded that the apoptosis of human cells separates the modules of the human interactome rather similarly to the modular disassembly we have observed in yeast stress. However, as an additional feature, caspases also induced the disassembly of key modules, leading to a much more complete disintegration of the whole network than in the case of “simple” stress in yeast. Moreover, caspases were extremely efficient in finding the most important human proteins to break the network structure of the human interactome.

10.3 Network rearrangements after crisis

So far we have shown two examples, where protein–protein interaction networks are disassembled in a moderate way (stress), or in a more complete fashion (programmed cell death). What happens after the initial, deteriorating phase of crisis? Obviously, the cell must then be re-assembled. Understandably, this task is impossible after the completion of programmed cell death. However, re-assembly of the yeast interactome after stress is a common, straightforward process (Figure 10.2).

How does the cell “know” how the disassembled modules should re-assemble? Obviously, not all inter-modular contacts are destroyed during moderate stress, such as those examined in our studies. Therefore, the remaining contacts guide re-assembly of modules. This ensures that the “new,” re-assembled cell will behave in a “cell-like” way, i.e. similarly to the cell before stress.

However, besides the above mechanism of conservation of cellular “rules, norms, and morale” re-assembly has another element too, which inflicts change. The cell will re-assemble in a slightly different structure from that before the stress. These changes may be called an adaptation, or learning process of the cell. They provide

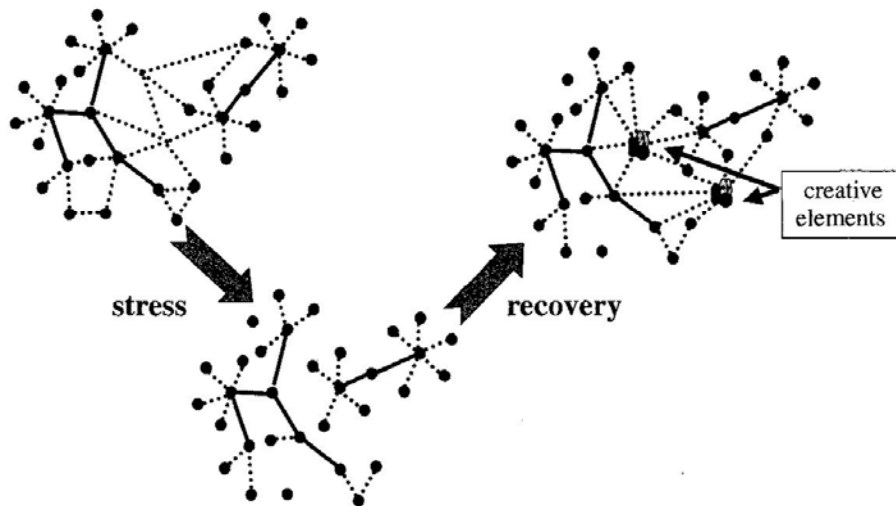


Figure 10.2. Changes in network structure during and after crisis. The illustrative figure shows the behavior, of the yeast interactome during and after a stressful event. During stress, network communities become disassembled, while after stress they re-assemble again – in a slightly different manner. Solid and dashed lines represent strong and weak links, respectively.

a “stress memory” or “stress history” of the given cell, which makes it unique amongst all other cells with identical genetic content (among many other reasons, this is also why two cloned humans may not be entirely identical after the first stressful event their zygotes survived). In the long run the “stress history” of cellular genealogies contributes to the gene-independent, epigenetic mechanisms of evolutionary change, called “modular evolution” (Korcsmáros *et al.*, 2007; Szalay *et al.*, 2007).

What type of behavior of network elements is necessary for changes in the reassembly process after stress? The ability to build “quasi-random,” low affinity contacts (weak links) plays a prominent role in this process. If an element has a large variety of weak links, and these links point to distant segments of the network, this element has the ability to change one or a few of its contacts, thus having a large adaptation potential in stress or crisis. Naturally, if this element has contacts to distant network segments, than it is in the overlap of multiple modules. Having this central position it may have a decisive role in building up the novel elements of inter-modular contacts after stress. The features of such elements have been summarized and are called “creative elements” (Csermely, 2008), since they behave similarly to creative people requiring the presence of many independent views and ideas so as not to be bored to death. Excitingly, these creative elements have a major role in introducing innovations to networks from protein structures, where they form the active centers of enzymes, to social networks and ecosystems (Csermely, 2008).

In cells, creative elements are well exemplified by stress proteins. These proteins are bona fide helpers for the survival of cells during stress. Their pivotal role in

evolutionary changes had been uncovered a decade ago (Rutherford and Lindquist, 1998). In the past 4–5 years it has become increasingly clear that the scope of creative elements of protein–protein interaction networks goes much beyond stress proteins (Csermely, 2009). As a recent example the yeast genome, which contains approximately 6,000 genes in total, has roughly 300 creative elements (Levy and Siegal, 2008).

10.4 How can we adapt the crisis responses of biological networks to social networks and to our own behavior?

The crisis responses we have shown so far are not specific to protein–protein interaction networks. Another type of cellular networks, mitochondrial networks, also first assemble, and then disassemble later, as the stress increases (Tondera *et al.*, 2009). The crisis-induced disintegration of networks can be observed in various types of inter-firm contacts (Saavedra *et al.*, 2008; Stark and Vedres, 2006). In fact, the essence of such transitions is reminiscent of the Schumpeterian term, “creative destruction” (Schumpeter, 1942).

The generality of network behavior after a decrease in network resources has already been suggested by the work of Tamás Vicsek and co-workers (Derényi *et al.*, 2004). The model shows that as resources available for the formation and maintenance of new contacts decrease (the amount of disassembly-inducing stress increases), the network topology changes from a random graph structure through a scale-free topology and a star network to a complete disassembly to subgraphs (Figure 10.3). These topological phase transitions can be generalized and a number of additional examples can be given, when similar topological changes occur in cellular networks, and in animal communities (Csermely, 2009). The crisis-induced changes of protein–protein interaction networks correspond well to the “scale-free to star” network transition in the case of yeast stress (marked by arrow 1 on Figure 10.3) and to the “star network to subgraphs” transition in the case of the human programmed cell death (marked by arrow 2 on Figure 10.3), respectively.

The changes we have described encourage us to think about the possible analogies of crisis-induced changes in social networks. Our common knowledge is in agreement with the possible consequences. The story of our cells is our own story too. In the first shock of crisis, we tend to become more isolated, and this relative isolation is true all the more to the contacts leading to distant social groups. The society breaks into more isolated “islands,” where the members of groups deal with their own problems. The isolation of social groups increases their tensions and possible conflicts.

How can we reassemble the social network after crisis? The first step is to enforce the islands of trust. Such islands can be very stable, even in a completely different

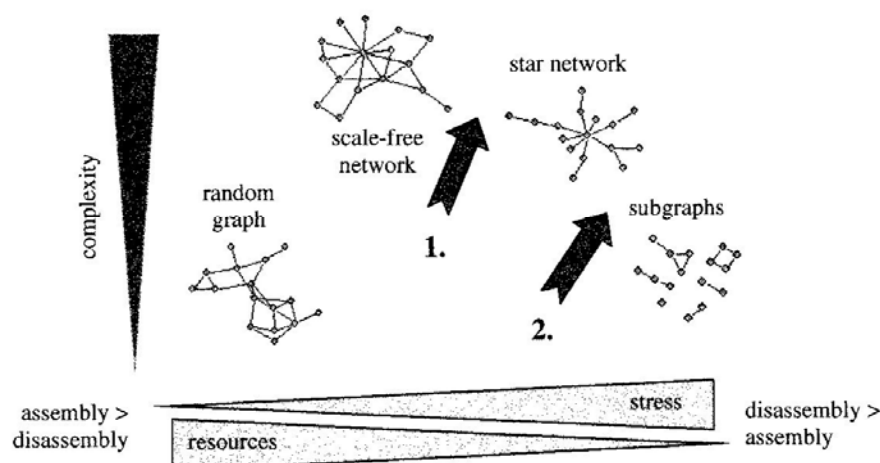


Figure 10.3. Changes of network structures upon increasing crisis (stress). The figure shows the topological phase transition of networks over a larger range than that shown in Figure 10.2. The “stress-scale” of the figure spans from the no-stress situation (left side, where assembly of contacts dominates over disassembly), to the completely severe, “lethal” stress (right side, where disassembly of contacts dominates over assembly). The vertical position reflects the approximate complexity of the network structure. Arrows 1 and 2 point to the approximate position of the yeast stress and the human programmed cell death conditions of our experiments, respectively.

environment (Wang *et al.*, 2008). However, this first step is not enough. From our own island of trust we have to be adventurous and discover the neighboring islands. For this we have to increase the number of social dimensions we have. With a rich structure of social dimensions we may reach a number of novel groups, with novel information, novel abilities, and solutions to the crisis. We may increase our value to be mediators and brokers for all of these conflicts and misunderstandings, which have been sadly enriched during the crisis. With each misunderstanding solved, we decrease the possibility of a future conflict. The novel contacts and the richness of social dimensions help us a lot to transform the conflicts to a new frame, where the original, adamant opposition can be seen, and ridiculed from a new distance. The wider scope of contacts also enables us to “think big,” and instead of competing, cooperate to explore novel options together.

Such behavior needs a stable and positive self-esteem. What can we suggest to those who do not yet have this? They should dare to make the very first step. They should leave those who are similarly desperate to themselves and they should make contacts with those who are already able to build trustful contacts. Give a positive gesture, give a smile. And a virtuous cycle begins: they get a positive feedback which enforces their positive self-esteem. And a more positive self-esteem helps the next smile. This “smile-propagation” is illustrated by the examination of Web-communities, where smiling faces are mostly surrounded by

other smiling faces, while sad figures draw other sad companions (Christakis and Fowler, 2009).

This summary has shown that the key to crisis recovery is in our own hands. Crisis is not a disaster, but a chance to develop something new, a chance to change. However, the key to crisis recovery is a funny one. None of us has it alone – but we all have it together. It is time to look around.

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