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Reviving a failed network via microscopic interventions

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From mass extinction to cell death, complex networked systems often exhibit abrupt dynamic transitions between desirable and undesirable states. Such transitions are often caused by topological perturbations, such as node or link removal, or decreasing link strengths. The problem is that reversing the topological damage, namely retrieving the lost nodes/links or reinforcing the weakened interactions, does not guarantee the spontaneous recovery to the desired functional state. Indeed, many of the relevant systems exhibit a hysteresis phenomenon, remaining in the dysfunctional state, despite reconstructing their damaged topology. To address this challenge, we develop a two-step recovery scheme: first - topological reconstruction to the point where the system can be revived, then dynamic interventions, to reignite the system's lost functionality. Applied to a range of nonlinear network dynamics, we identify a complex system's recoverable phase, a state in which the system can be reignited by a microscopic intervention, *i.e.* controlling just a single node. Mapping the boundaries of this newly discovered phase, we obtain guidelines for our two-step recovery.

Complex systems, biological, social or technological, often experience perturbations and disturbances, from overload failures in power systems¹⁻⁴ to species extinction in ecological networks⁵⁻⁷. The impact of such perturbations is often subtle, the system exhibits a minor response, but continues to sustain its global functionality⁸⁻¹⁰. However, in extreme cases, a large enough perturbation may lead to a large-scale collapse, with the system abruptly transitioning from a functional to a dysfunctional dynamic state¹¹⁻¹⁶. (Fig. 1a-d). For instance, in cellular dynamics, genetic knockouts, beyond a certain threshold, lead to cell death^{17,18}; in ecological systems, changes in environmental conditions may, in extreme cases, cause mass-extinction^{5-7,19}; and in infrastructure networks, a cascading failure, at times, results in a major blackout^{11,20}. When such collapse occurs, the naïve instinct is to reverse the damage, retrieve the failed nodes and reconstruct the lost links. Such response however is seldom efficient, as (i) we rarely have access to all system components²¹, limiting our ability to reconstruct the perturbed network; (ii) even if we *could* reverse the damage, due to hysteresis²²⁻²⁴, in many cases, the system will not spontaneously regain its lost functionality²⁵.

To address this challenge, we consider here a two-step recovery process:

Step I. Restructuring (Fig. 1e). Retrieving the network topology and weights to a point where the system *can* potentially regain its functionality.

Step II. Reigniting (Fig. 1f). Introducing dynamic interventions to steer the system back to functionality.

The challenge in Step II is that in most practical scenarios we lack direct control over the dynamic activity of the majority of the nodes. Hence we seek to reignite the system via *micro-interventions*, *i.e.* controlling just a small number of components, typically - just a single or, at most, few nodes. We, therefore, need to characterize the conditions under which such single-node reigniting can be achieved, representing the primary focus of the current contribution. Along the way, we expose a new dynamic *phase* of complex systems - the *Recoverable* phase, a state in which the system can be driven towards functionality by controlling a microscopic set of nodes.

Unlike the classic controllability challenge^{26–32}, formulated in the context of linear dynamics, in recoverability we benefit from the nonlinear nature of the interactions. Indeed, when reigniting we do not aim to steer the system to a general arbitrary state, as done in the control theoretic setting, but rather towards one of its naturally occurring fixed-points - a problem of different nature, relying on fundamentally distinct mathematical tools²¹.

The challenge of irreversible collapse

Consider a complex system of N components (nodes), linked through A_{ij} , a sparse, binary and random network with arbitrary degree distribution $P(k)$. In A_{ij} the links represent dynamic interactions, whose nature depends on context. For instance, in a social system $A_{ij} = 1$ captures an acquaintance, and hence a potentially infectious interaction between individuals i and j , whereas in a sub-cellular network it may represent a regulatory interaction between gene i and gene j . To account for these dynamic distinctions we denote each node's *activity* by $x_i(t)$, quantifying, *e.g.*, individual i 's probability of infection or gene i 's instantaneous expression level. We then track the dynamics of the system via^{33–35}

$$\frac{dx_i}{dt} = M_0(x_i) + w \sum_{j=1}^N A_{ij} M_1(x_i) M_2(x_j), \quad (1)$$

a general equation, characterized by three potentially nonlinear functions $M_0(x)$, $M_1(x)$ and $M_2(x)$. The first function, $M_0(x_i)$ captures node i 's self-dynamics, describing mechanisms such as protein degradation³⁶ (cellular), individual recovery^{37,38} (epidemic) or birth/death processes³⁹ (population dynamics). The product $M_1(x_i)M_2(x_j)$ describes the i, j interaction mechanism, *e.g.*, genetic activation^{9,17,40}, infection^{37,38} or symbiosis⁴¹. The mean strength of these interactions is governed by the weight w . In the context of recoverability, we seek to revive the activity of all nodes by activating a selected set of nodes, hence we focus on *constructive* interactions, in which nodes positively contribute to each others activity. This is expressed in Eq. (1) through $wM_1(x)M_2(x) \geq 0$ (see Supplementary Section 1). Such conditions naturally arise in many relevant applications, social, biological or ecological, as exemplified in Figs. 3a and 4a,f.

Setting the derivative on the left hand side of (1) to zero, we obtain the system's fixed-points, $\mathbf{x}_\alpha = (x_{\alpha,1}, \dots, x_{\alpha,N})^\top$, which, if dynamically stable, represent different *states*, desirable or undesirable, in which the system can naturally reside. Transitions between these states often result from perturbations to A_{ij} and w , such as node/link deletion or weight change. When this occurs, it is often difficult to reverse the unwanted transition, since the system fails to spontaneously recover, even if we retrieve the lost nodes, links or the weights. To illustrate this difficulty we refer to a concrete example below.

Example 1. Cellular dynamics (Fig. 3). As our lead example we consider gene-regulation, capturing activation interactions between genes. Such genetic activation is often modeled within (1), using Michaelis-Menten dynamics⁴⁰ (Fig. 3a). Under this framework $M_0(x_i) = -Bx_i^a$, describing degradation

($a = 1$), dimerization ($a = 2$) or a more complex bio-chemical depletion process (fractional a), occurring at a rate B ; below we set $B = 1$. The activation interaction is captured by a Hill function of the form $M_1(x_i) = 1$, $M_2(x_j) = x_j^h/(1 + x_j^h)$, a *switch-like* function that saturates to $M_2(x_j) \rightarrow 1$ for large x_j , representing j 's positive, albeit bounded, contribution to i 's activity $x_i(t)$.

For sufficiently dense A_{ij} or large ω the system exhibits an *active* state $\mathbf{x}_1$, in which all nodes have positive activity ($x_{1,i} > 0$ for all i), representing a *living* cell. However, perturbations to the network topology, such as link/node removal or weight reduction can cause a sharp transition to the *inactive* state $\mathbf{x}_0 = (0, \dots, 0)^\top$, in which all nodes have zero activity, *i.e.* cell death. To track this transition systematically we measured the average activity $\bar{x}_\alpha = (1/N) \sum_{i=1}^N x_{\alpha,i}$, which follows $\bar{x}_\alpha > 0$ for $\alpha = 1$ and $\bar{x}_\alpha = 0$ for $\alpha = 0$, as we subject it to increasing levels of topological stress : removing a q -fraction of nodes (Fig. 3b); deleting a q -fraction of links (Fig. 3d) or reducing ω by a factor q (Fig. 3c). In all cases we observe a sudden transition, as the perturbations exceed a critical threshold q_c , from $\bar{x}_\alpha > 0$ (green), representing the active state $\alpha = 1$, to $\bar{x}_\alpha = 0$ (red), representing the inactive state $\alpha = 0$.

To drive the system back to the desired $\mathbf{x}_1$, the instinctive approach is to reverse the topological perturbations, namely reconstruct the deleted nodes/links or push the weight ω to its original strength. In Fig. 3f we do precisely that, finding that the system fails to recover. The reason is that while $\mathbf{x}_1$ is only stable above q_c , $\mathbf{x}_0$ is *always* stable - both below and above the critical point. This leads to a hysteresis phenomenon that causes the system to remain inactive, despite the reversal of the perturbation, *e.g.*, recovering the original weight ω .

Example 1, above, while representing a specific scenario, illustrates the family of challenges that we tackle here: system's with irreversible topologically driven transitions, *e.g.*, from a desired $\mathbf{x}_1$ to an undesired $\mathbf{x}_0$. To revive such systems, we must dynamically *reignite* them by exerting external control over the activities $x_i(t)$, in order to drive them back towards the basin of attraction of $\mathbf{x}_1$ (Fig. 1h).

Recoverability - single-node reigniting

After restructuring A_{ij}, ω , the most natural way to reignite the system is to drive all activities x_i towards an initial condition from which the system will naturally recover to the desired $\mathbf{x}_\alpha$. Namely, we must steer the system into $\mathbf{x}_\alpha$'s basin of attraction (Fig. 1h)

$$\mathfrak{B}_\alpha = \left\{ \mathbf{x}(t=0) \mid \mathbf{x}(t \rightarrow \infty) = \mathbf{x}_\alpha \right\} \quad (2)$$

which comprises all initial conditions $\mathbf{x}(t=0)$ from which Eq. (1) converges to $\mathbf{x}(t \rightarrow \infty) = \mathbf{x}_\alpha$. The problem is that such level of control over the dynamics of all nodes is seldom attainable, hence we seek to recover the system's functionality by driving just a microscopic fraction $f \rightarrow 0$ of *forced* nodes.

To achieve this, we consider the limit of $f \sim 1/N$, in which case our reigniting is attempted through, typically, a single, randomly selected *source* node s . To reignite the system we artificially set s 's activity to $x_s(t) = \Delta > 0$, namely we externally *hold* its activity, forcing it to equal Δ . The remaining $N-1$ nodes continue to follow the *natural* system dynamics, *i.e.* Eq. (1), responding to the s -forcing. In technical terms, the failed state of the system, $\mathbf{x}_0$, captures Eq. (1)'s initial condition, and the forced node imposes a strict boundary condition at s .

In a recoverable system, after some time, the activities will enter $\mathfrak{B}_1$, at which point we can cease our external control and allow the system to naturally transition to $\mathbf{x}_1$, based on its internal dynamics. This captures a successful reigniting of the system's activity. If, however, the system is non-recoverable, such single-node reigniting is insufficient, the system remains at the basin $\mathfrak{B}_0$, and once we discontinue our external forcing, it relaxes back to $\mathbf{x}_0$, a failed reigniting.

To analytically track the system's response to our forcing at s , we divide the rest of the network into

shells $K_s(l) = \{j \mid L_{sj} = l\}$, comprising all nodes located at distance l from s (Fig. 2a). In this notation $K_s(0) = \{s\}$, $K_s(1)$ is the group of s 's nearest neighbors, $K_s(2)$ its next neighbors, and so on. Then, starting with $x_s(t) = \Delta$, we track the average activity of nodes in $K_s(l)$, via

$$x_s(l, t) = \frac{1}{|K_s(l)|} \sum_{i \in K_s(l)} x_i(t), \quad (3)$$

where $|K_s(l)|$ represents the number of nodes in $K_s(l)$. The shells adjacent to the source, *i.e.* small l , will be forced to respond to s 's activation Δ , exhibiting a gain in their activity $x_s(l, t)$. However, such response may, under certain conditions, decay as l is increased, leaving the distant shells almost unaffected, and therefore still within the basin $\mathfrak{B}_0$. Under these conditions, upon termination of our Δ -forcing, all shells retreat back to $\mathbf{x}_0$, rendering the system unrecoverable. Successful reigniting, therefore, requires that

$$x_s(l \rightarrow \infty, t \rightarrow \infty) \in \mathfrak{B}_1, \quad (4)$$

capturing a state in which the forcing at s was able to penetrate the network and impact the state of even the most distant nodes at $K_s(l \rightarrow \infty)$. This represents a recoverable system that will naturally revert to $\mathbf{x}_1$ once the forcing Δ is deactivated.

In Supplementary Section 2.1 we use Eq. (1) to derive a direct set of equations for the shell states $x_s(l, t)$ in (3), providing

$$\begin{cases} x_s(0, t) = \Delta \\ \frac{dx_s(l, t)}{dt} = M_0(x_s(l, t)) + \omega M_1(x_s(l, t)) \left(M_2(x_s(l-1, t)) + \kappa M_2(x_s(l+1, t)) \right) \end{cases}, \quad (5)$$

a set of continuous time discrete space differential equations, with a set boundary condition at $l = 0$, capturing our s -forcing. The functions $M_0(x)$, $M_1(x)$, $M_2(x)$ and the weight ω in (5) are taken from Eq. (1), and the parameter κ is extracted from A_{ij} via

$$\kappa = \frac{1}{N} \sum_{i=1}^N \frac{1}{k_i} \sum_{j=1}^N A_{ij} k_j - 1, \quad (6)$$

capturing the average neighbor's residual degree^{10,42}. Starting from an initial condition, where $\mathbf{x}_s(l, t = 0) \in \mathfrak{B}_0$ for all $l > 0$, Eq. (5) helps us track the response of the initially failed system to our reigniting.

To extract insight from (5), we seek its steady state $x_s(l) = x_s(l, t \rightarrow \infty)$, by setting the derivatives on the left hand side to zero. We show that we can approximate this state via the recurrence relation (Supplementary Section 2.3)

$$\begin{cases} x_s(0) = \Delta, \\ F(x_s(l)) = M_2(x_s(l-1)), \end{cases} \quad (7)$$

where

$$F(x) = \frac{1}{\omega} R(x) - \kappa M_2 \left(R^{-1}(\omega M_2(x) + \omega \kappa M_2(\bar{\mathbf{x}}_0)) \right), \quad (8)$$

$R(x) = -M_0(x)/M_1(x)$ and $R^{-1}(x)$ is its inverse function. The parameter $\bar{\mathbf{x}}_0$ in the right hand side of (8) represents the mean activity of nodes in $K_s(l > 1)$ under the failed state $\mathbf{x}_0$. Hence, we arrive at a

direct equation for the l -shell steady states $x_s(l)$ under any given forcing Δ .

Equations (7) and (8) represent our key result. They reduce the recoverability of (1), a multidimensional nonlinear dynamic equation, into a manageable first order recurrence relation. This recurrence takes the system's topology and weights (ω, κ) and its nonlinear dynamic mechanisms $(M_0(x), M_1(x), M_2(x))$ and hence $R(x)$ as input, and predicts the system's recoverability as output. For any given forcing Δ , the recurrence (7) either leads to $x_s(l \rightarrow \infty) \in \mathfrak{B}_1$, satisfying the recoverability condition (4), or to $x_s(l \rightarrow \infty) \in \mathfrak{B}_0$, and hence indicating a failed recovery.

To obtain $x_s(l \rightarrow \infty)$, and observe whether it is in $\mathfrak{B}_0$ or $\mathfrak{B}_1$, we seek the potential fixed-points of the recurrence equation (7). These points can be extracted from the solutions of

$$F(x) = M_2(x), \quad (9)$$

capturing stationary states in which $x_s(l) = x_s(l-1)$ ^{43,44}. Therefore, to analyze the system's recoverability we extract the intersections of the two functions $F(x)$ and $M_2(x)$ in (9), observing two characteristic behaviors (Fig. 2): if, for example, $F(x)$ and $M_2(x)$ have only a single intersection $x \in \mathfrak{B}_0$, then the series inevitably converges to that point (Fig. 2c). This represents a non-recoverable system, that, regardless of the magnitude of Δ , resorts back to $\mathbf{x}_0$. If, on the other hand the functions have several intersections, then its convergence depends on the boundary condition $x_s(0) = \Delta$. For $\Delta < \Delta_c$ it will approach $\mathfrak{B}_0$, a failed reigniting, and for $\Delta \geq \Delta_c$ it will reach $\mathfrak{B}_1$, capturing a successful reigniting (Fig. 2d,e; see also Supplementary section 2.3). Therefore, our formalism predicts both whether the system is recoverable or not, and in case it is - it predicts the required critical forcing Δ_c for reigniting. Most crucially, the patterns of intersection of (9) depend on the values of κ and ω in (8). This means that the system can transition between the recoverable and the non-recoverable phases following appropriate manipulations of ω and κ - precisely the desired guidelines for the restructuring step, which must be carried out prior to reigniting.

Next we demonstrate our framework on a range of relevant systems, starting from gene regulation (Example 1), then advancing to neuronal and ecological dynamics. We show that tracking the $l \rightarrow \infty$ limit of Eq. (7) can help us obtain direct insights into reviving collapsed systems, specifically:

- *Recoverability.* Given the state of the network, *i.e.* κ, ω , we predict whether the system can be revived by single-node reigniting or not.
- *Reigniting force.* In case the system *is* recoverable, we obtain the minimal magnitude of the forcing $\Delta \geq \Delta_c$ required for recovery.
- *Restructuring.* If non-recoverable, the equations offer guidelines on relevant structural interventions affecting the topology/weights, to steer ω and κ towards the recoverable phase.

Applications

Cellular dynamics (Fig. 3; see also Supplementary Section 3.1). As our first application we return to Example 1, regulatory dynamic, where $M_0(x) = -x^a$, $M_1(x) = 1$ and $M_2(x) = x^h/(1+x^h)$, and therefore $R(x) = x^a$ and $R^{-1}(x) = x^{1/a}$. Equation (9) becomes

$$\frac{1}{\omega} x^a - \frac{\kappa x^{\frac{h^2}{a}}}{x^{\frac{h^2}{a}} + \omega^{-\frac{h}{a}} (1+x^h)^{\frac{h}{a}}} = \frac{x^h}{1+x^h}, \quad (10)$$

whose roots (x) determine the potential fixed points of the reignited system. Clearly, $x = 0$ represents a solution to (10), capturing the fact that the failed state $\mathbf{x}_0 = (0, \dots, 0)^\top \in \mathfrak{B}_0$ is always stable. Hence,

the question is, under what conditions do we observe a second solution $x > 0$, representing a potential convergence to $\mathfrak{B}_1$. To answer this, we first note that while the right hand side of Eq. (10) is independent of the network topology, its left hand side, $F(x)$, is affected by A_{ij} through κ and by its link weights through ω . Therefore, in Fig. 3f,h we plot $M_2(x)$ vs. x (yellow) and observe its intersections with $F(x)$ (purple) as we vary the values of these two structural parameters. This allows us to observe, graphically, the potential convergence of the system to $\mathfrak{B}_0$ or $\mathfrak{B}_1$.

First we consider $\omega = 0.8, \kappa = 4$ (Fig. 3g), conditions under which the system is bi-stable, and hence, in principle, can reside both in $\mathbf{x}_0$ or $\mathbf{x}_1$. Despite this bi-stability, we find that (10) exhibits only one solution, represented by the single intersection at $x = 0$. This guarantees that (7) converges to $x_s(l \rightarrow \infty) = 0$, independently of Δ . Consequently, the system is non-recoverable, regardless of the strength of our forcing. The meaning is, that even though the system *can* potentially feature a stable $\mathbf{x}_1$, it cannot be reignited from an $\mathbf{x}_0$ initial condition by single-node activation. Indeed, this prediction is confirmed in Fig. 3h, which indicates that the system fails to recover, despite the forcing Δ at s .

Increasing the network density to $\kappa = 7$, however, changes the picture, as now (10) features three intersection points (Fig. 3i): an unstable intermediate point (grey) and the two stable points at $x = 0$ (red) and at $x > 0$ (green), representing convergence to $\mathfrak{B}_0$ and $\mathfrak{B}_1$, respectively. This predicts a critical forcing Δ_c (vertical grey dashed line), above which all x_i reactivate to $\mathfrak{B}_1$, and below which they remain within $\mathfrak{B}_0$. If $\Delta > \Delta_c$ the system will be successfully reignited by controlling just a single node, as all shells will, eventually be driven into $\mathfrak{B}_1$, and then naturally converge to the desired $\mathbf{x}_1$. Fig. 3j shows just that, as now the system responds to our forcing, and, as predicted, recovers its lost functionality.

The results above uncover the existence of a novel, previously overlooked, dynamic phase. Indeed, the regulatory system of Fig. 3a has been previously shown to follow two phases, *inactive*, where only $\mathbf{x}_0$ is stable vs. *bi-stable* where both $\mathbf{x}_0$ and $\mathbf{x}_1$ are stable, depending on initial conditions (Fig. 3b-d). Our analysis here uncovers a third phase: *recoverable*, a subspace of the bi-stable phase, in which the system can be reignited from $\mathbf{x}_0$ to $\mathbf{x}_1$ by controlling a microscopic set of nodes, here just one single node. The parameters driving this three state phase space are ω, κ and Δ , which together determine the existence (and if exists - the value) of the critical forcing required to revive the inactive system. In Fig. 3k we show these phases in the ω, κ space, as obtained from numerically simulating the states of the system for $\sim 10^3$ different combinations of ω and κ (Supplementary Section 4.3). The white solid line represents our theoretical prediction, based on analyzing the intersections of (10), indicating that the boundaries of recoverability can be well-approximated by our analytical framework. We also present the ω, Δ and κ, Δ phase diagrams, further indicating the agreement between simulation and theory (Fig. 3l,m).

To further test our predictions in an empirical setting, we collected data on two real biological networks, capturing protein interactions in Human⁴⁵ ($\kappa = 21.7$) and Yeast⁴⁶ ($\kappa = 10.7$) cells. In these networks the interaction topology A_{ij} is extracted from empirical data, but the average weight ω , determining the interaction *rates* can change due to environmental conditions. We, therefore set, for each network, two different weights (Fig. 3n): for Human we set $\omega_1 = 0.2$ (Human UR orange) and $\omega_2 = 1$ (Human R blue), the former in the unrecoverable phase, and the latter - recoverable. Similarly, for Yeast we set $\omega_1 = 0.4$ (Yeast UR, orange, unrecoverable) and $\omega_2 = 1$ (Yeast R, blue, recoverable). As predicted, we find in Fig. 3n,o that, indeed, Yeast UR and Human UR remain inactive under reigniting. This is while Yeast R and Human R, both predicted to reside in the recoverable phase, can be successfully revived via single-node reigniting.

Neuronal dynamics (Fig. 4a-e, Supplementary Section 3.2). As our second example we consider the Cowan-Wilson neuronal dynamics^{47,48}, in which (1) follows the form shown in Fig. 4a. The system naturally exhibits three dynamic phases. The *inactive* state $\mathbf{x}_0$, in which all activities are suppressed, is obtained when the network is extremely sparse, *i.e.* small κ, ω . The *active* $\mathbf{x}_1$, in which x_i are relatively large, is observed when ω, κ are high. In between these two extremes the system features a *bi-stable* phase,

in which both $\mathbf{x}_0$ and $\mathbf{x}_1$ are potentially stable. In Fig. 4b,c we observe these phases on a set of random networks with varying ω , obtaining two critical points $\omega_{c,1} < \omega_{c,2}$ (dashed grey lines), in which the system transitions between active, inactive and bi-stable. This phase-space predicts a hysteresis phenomenon: if ω is driven below $\omega_{c,1}$ and the system fails, it will not spontaneously recover unless we retrieve ω to be above $\omega_{c,2}$.

Our formalism, however, predicts an additional, previously unknown dynamic phase, in which the system is *recoverable*. This phase, shown in Fig. 4d (blue) represents a sub-space of the bi-stable regime, in which the failed system, if untouched, remains at $\mathbf{x}_0$ as per the above hysteresis. Yet, under single-node reigniting, it can be forced back towards activity ($\mathbf{x}_1$). To demonstrate this we collected data on the structure of a human brain network⁴⁹ to construct A_{ij} ($\kappa = 6.6$), and simulated neuronal dynamics using $\omega_1 = 2$, in the unrecoverable phase (Brain UR, orange), and $\omega_2 = 4$, which is recoverable (Brain R, blue). Indeed, Fig. 4e confirms that Brain UR remains inactive, while Brain R's activity is successfully revived.

Mutualistic population dynamics (Fig. 4f-j, Supplementary Section 3.3). As our final example we examine symbiotic interactions in ecological networks, such as plant-pollinator relationships⁵⁰. Once again, we find a *window* of recoverability, in which one can steer a collapsed system towards activity via single-node reigniting. Here we used an empirical plant-pollinator network⁵¹, Eco, to examine recoverability (Fig. 4i,j), a low ω unrecoverable Eco UR (orange) and an increased ω recoverable Eco R (blue).

Taken together, these examples demonstrate the predictive power of our framework, which allows to systematically map the conditions for recoverability. Next, we show how to use the obtained recoverability maps to provide direct insights into our proposed two-step recovery, extracting guidelines for reviving a collapsed network.

Restructuring guidelines

To successfully reignite a failed system we must first steer it from the non-recoverable to the recoverable phase. This can be achieved through appropriate restructuring interventions to impact ω or κ , for instance, increasing weights or adding nodes/links. The phase diagrams of Figs. 3k-m and 4d,i can provide guidelines for such restructuring, as, indeed, they indicate what interventions can potentially push us closer to the recoverable phase.

To illustrate this, in Fig. 5a-d we simulate a cellular network (Yeast) that has been driven towards inactivity due to major topological perturbations, such as node or link deletion (grey nodes/links). Some of the removed components are inaccessible (red), and hence when restructuring we cannot retrieve them. To revive the system, under these constraints, we incorporate our proposed two-step recovery:

- **Step I. Restructuring.** First we conduct topological interventions, to bring the system to the recoverable phase. As explained above, certain nodes or links are inaccessible to us (red), hence our potential interventions are restricted. The challenge is, therefore, to design a set of accessible interventions that will enable us to revive the system's dynamic activity. As recoverability is driven by two relevant parameters, κ and ω , we map all potential intervention to their effect on these two parameters (Fig. 5e). Indeed, a sequence of such interventions represents a *path* in the κ, ω space. Therefore, in the restructuring step we seek paths of accessible interventions originating in the present state of the system, and delivering the network into the bounds of the recoverable phase (Fig. 5f). Our goal, we emphasize, is not to simply retrieve the lost nodes and links, but to achieve recoverability. This designates, not a single point, but rather an entire sub-space in κ, ω (Fig. 5f, blue), affording us some level of restructuring flexibility. As a result, despite the constraints imposed by the network's irretrievable components, we are able to design three distinct restructuring paths, all originating in the current state of the failed system (Yeast perturbed, white), but leading to different destinations - Net 1,2 or 3 - within the recoverable sub-space.

- **Step II. Reigniting.** Once in the recoverable phase we can revive the system via single-node reigniting, and retain its lost activity, as shown in Fig. 5g for the three restructured networks Net 1,2 and 3.

This example illustrates how the phase diagrams of recoverability provide direct guidelines for restructuring. For example, in Fig. 5f path 1 builds mainly on controlling the interaction strength (ω), but assumes little freedom to add nodes or links (κ). In contrast path 3 involves a significant component of adding nodes/links to A_{ij} , affecting not just ω but also κ . The optimal restructuring path is, therefore, determined by the nature of our constraints, for example, the relative difficulty in adding weights as in path 1, vs. adding nodes/links, *a là* path 3. The crucial point is, that, knowing the phase-boundaries of recoverability, we were able to set different end-points for each path (Net 1,2,3), providing us with a spectrum of potential interventions - either focusing on ω , and hence aiming for end-point 1, or focusing on κ , and thus seeking to reach end-point 3. Such flexibility, enabled thanks to our phase-diagrams, is crucial for real-world restructuring.

While the potential space of structural interventions in Step I is incomprehensibly vast, our phase diagrams reduce this space into just two relevant control parameters - κ , characterizing A_{ij} 's density, and ω , capturing its link weights. This allows us to assess the contribution of all potential interventions by quantifying their effect on these two parameters - enabling to seek optimal pathways for crossing the recoverability phase boundary.

Interestingly, our phase diagrams indicate that κ and ω may play unbalanced roles: for example Fig. 5f shows that the phase boundary from unrecoverability to recoverability in the κ, ω space becomes almost flat in the limit of large κ (white solid line). This implies that while it may only require a small change in ω , *i.e.* pushing the system in the vertical direction, to transition a network towards recoverability, doing so through κ , along the horizontal axis, is by far more difficult. Indeed, in Supplementary Section 3.1.3 we show that in the limit of large h in Fig. 3a, the phase boundary becomes almost independent of κ , and the effect of increasing ω becomes dominant. Hence, under cellular dynamics, increasing weights, when possible, is a preferred strategy over adding links. This offers a quite general restructuring guideline, that can help design efficient κ, ω -paths in our two-step recovery scheme.

Discussion and outlook

While the structure of complex networks has been deeply investigated over the years, our understanding of their dynamics is still emerging. The challenge is often focused on *prediction*, aiming to foresee a network's dynamic behavior. Here, we go a step further, and focus on *influence*, showing how to steer a system towards a desired behavior.

At first glance, this challenge seems to be associated with network controllability. There are, however two crucial distinctions between recoverability and controllability: (i) Control theory revolves around linear dynamics, and is therefore fully characterized by the network structure/weights (A_{ij}, ω). Recoverability, in contrast, is tailored for nonlinear systems within the form (1), hence driven by the interplay of this structure with the system's intrinsic dynamics. Indeed, the phase boundaries of recoverability strongly depend on the system's interaction mechanisms $M_0(x), M_1(x), M_2(x)$, as observed in Figs. 3k-m and 4d,i. (ii) On the other hand, while controllability aspires to drive a system between any two arbitrary states, an unsolved challenge under nonlinear dynamics, recoverability is limited to the system's naturally occurring fixed-points.

The main advantage of item (ii) above, is that it allows us to revive a failed system using an extremely simple control signal, namely the constant forcing Δ . Had we needed a more delicate fine-tuned input, our strategy would have likely been too sensitive and impractical. Fortunately in recoverability, one only needs to *kick* the system out of its undesired basin, then let it relax to $\mathbf{x}_1$ independently²¹ (Fig. 1h).

Such crude level of control, we find, is possible even under the rather broad conditions captured by Eq. (1).

The microscopic behavior of complex networks is driven by countless parameters, from the fine-structure of A_{ij} to the specific rates of each node's dynamic processes. Our analysis, however, shows, that their large-scale functionality can be traced to a manageable set of *relevant parameters*, *e.g.*, κ, ω and Δ . Such dimension reduction is the fundamental premise of statistical physics, allowing to analyze systems with endless degrees of freedom using a limited set of statistical entries. We believe, that a similar approach to network dynamics, can help us understand, predict, and ultimately influence the behavior of complex multi-dimensional systems.

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Figure 1: Reviving a failed network. (a) The components of our modeling framework: The network topology is captured by the adjacency matrix A_{ij} and the link weights ω (grey terms); the interaction mechanisms, *i.e.* the dynamics, are captured by the nonlinear functions $M_0(x), M_1(x), M_2(x)$ (orange terms). (b) Depending on the dynamics - *e.g.*, cellular, neuronal or ecological - the system exhibits distinct fixed-points, active (x_1 , green) or failed (x_0 , red); intermediate unstable points are marked by grey dashed lines. The transitions between these states are driven by perturbations to the topology (A_{ij}, ω). (c) Under the unperturbed topology the system resides in the functional state x_1 , in which all nodes are active, *i.e.* $x_i > 0$. In this presentation, which we also use throughout the paper, the network nodes are laid out on the x, y plane, and their activities x_i are captured by the z coordinate. Hence, an active system has all nodes spread out along the positive z -axis, while a failed network is laid out around $z = 0$. We also use color coding from red (small x_i) to blue (large x_i) as visual aid. (d) Perturbations to the topology, such as node/link removal or weight reduction, result in a collapse to the inactive x_0 , here having all activities vanish ($z = 0$). (e) **Restructuring.** To revive the failed system we must first restructure its topology to a point where it *can* recover, namely a point where x_1 is potentially stable. This, however, is insufficient, as often the system will not spontaneously retain its lost activity. (f) **Reigniting.** After restructuring we *reignite* the active state x_1 by controlling a microscopic set of nodes, here the single node s . The controlled node is *forced* to sustain a constant activity $x_s(t) = \Delta$. This activating force then spreads to impact all other nodes in the network and push them towards x_1 . (g) The state of the network in three different time-points following single-node reigniting, forcing $x_s(t) = \Delta$ (black node at center). The reigniting force gradually impacts the network, until the system's activity x_1 is restored. (h) In this process we use the natural basin structure of our dynamics. The basin $\mathfrak{B}_\alpha$ captures all initial states, from which the system (1) converges to the fixed-point x_α . Therefore, to reignite x_1 we seek to steer the system from $\mathfrak{B}_0$ (red) to any point within $\mathfrak{B}_1$ (green). Once in $\mathfrak{B}_1$, we can cease our forcing, and the system will spontaneously transition to the desired x_1 (dashed arrows).

Figure 2: Can a system be reignited by a single node? (a) During reigniting we select a single *source* node s and, for a limited time, we artificially force it to sustain a permanent activity Δ . To track the system's response to such forcing, we divide the network into *shells* $K_s(l)$ comprising all nodes at distance l from s . (b) The reignited activity $x_s(l)$ of all nodes at $K_s(l)$ is tracked via the recurrence relation of Eq. (7), starting from our forcing at s (top equation), and tracking its propagation as it penetrates the network shells (bottom equation). Successful reigniting requires $x_s(l \rightarrow \infty) \in \mathfrak{B}_1$, *i.e.* that the distant shells were driven towards the desired basin. (c) - (e) We can track the convergence of the recurrence relation graphically by plotting $F(x)$ (purple) and $M_2(x)$ (yellow). The forcing Δ determines our initial starting point, and the recurrence can be tracked via the red or green trajectories. The final state $x_s(l \rightarrow \infty)$ is reached when the two functions, $F(x)$ and $M_2(x)$, intersect. We observe three potential scenarios (Supplementary Section 2.3): **Case 1.** $F(x)$ and $M_2(x)$ have a single intersection point in $\mathfrak{B}_0$ (red). Under these conditions, regardless of the initial value (Δ) the recurrence converges to $\mathfrak{x}_s(l \rightarrow \infty) \in \mathfrak{B}_0$, and hence the system is unrecoverable. **Case 2.** $F(x)$ and $M_2(x)$ exhibit two intersections, corresponding to the system's two stable fixed points $\mathfrak{x}_0$ (red) and x_1 (green); the intermediate intersection (grey) is unstable. Here, for $\Delta < \Delta_c$ the system converges to $\mathfrak{B}_0$, *i.e.* unrecoverable (red trajectory), while for $\Delta \geq \Delta_c$ it approaches $\mathfrak{B}_1$ - hence it is recoverable (green trajectory). **Case 3.** In case $F(x)$ is non-monotonic the critical forcing Δ_c is determined by $F(x)$'s local maximum point (grey dashed lines), see Supplementary Section 2.3 for a detailed analysis of Case 3.

Figure 3: Recoverability of cellular dynamics. (a) Gene regulation as modeled via the Michaelis-Menten dynamics. (b) - (d) The average steady-state activity $\bar{x}$ as obtained from cellular dynamics following topological perturbation: deletion of a q -fraction of nodes; factor q reduction of weights and removal of a q -fraction of links. While the active $\mathbf{x}_1$ is only stable for $q < q_c$ (green), the inactive $\mathbf{x}_0$ is always stable (red). Hence the system undergoes an irreversible collapse at $q = q_c$. (e) We track three specific states, capturing the steady-state activities x_i under varying levels of weight loss q . When q is small (left) all x_i are positive and the system is in the *active* state $\mathbf{x}_1$. x_i gradually decline as q increases (center), until the system collapses into the inactive state $\mathbf{x}_0$, in which all x_i vanish (right). (f) Reversing the perturbation does not revive the system. Here we retrieve the lost weights, however, since $\mathbf{x}_0$ is always stable, the system remains in the collapsed state, avoiding spontaneous recovery. To revive the failed system we apply single-node reigniting: (g) The functions $F(x)$ (purple) and $M_2(x)$ (yellow) as obtained from the left/right hand sides of Eq. (10), setting $\kappa = 4, \omega = 0.8$. The system follows Case 1 of Fig. 2c, exhibiting a single intersection ($\mathfrak{B}_0$, red), and hence it is unrecoverable. (h) Indeed, forcing node s to a permanent Δ activity (black node), the remaining shells fail to reignite. (i) Under a denser A_{ij} with $\kappa = 7$ the function $F(x)$ takes a different form and the system now follows Case 3. It is now recoverable if the forcing $\Delta \geq \Delta_c = 0.9$ (vertical dashed line). (j) As predicted, now the system is successfully activated via single-node reigniting. (k) The phase diagram in the κ, ω plane (Supplementary Section 4.3). We observe the three predicted phases: inactive (red), where only $\mathbf{x}_0$ is stable, unrecoverable (yellow), where $\mathbf{x}_1$ is potentially stable, but unattainable via single-node reigniting, and recoverable (blue), where the system can be revived by a single node. The theoretical prediction obtained from the roots of Eq. (10) is also shown (white solid line). (l) The phase diagram in the ω, Δ space. Here, for $\omega < 0.9$ the system is never recoverable. Above that, the higher is ω the smaller is the forcing Δ required for reigniting. Our theoretical prediction is also shown (white solid line). (m) The κ, Δ phase diagram. The slight discrepancy between theory (white solid line) and simulation (yellow-blue transition) is likely due to the discrete nature of κ . (n) To examine our prediction in an empirical setting we used the human protein interaction network⁴⁵ ($\kappa = 21.7$) to construct A_{ij} under two values of ω : Human UR with $\omega = 0.2$, in the unrecoverable phase, and Human R, with $\omega = 1$, which is recoverable (see panel k, orange vs. blue squares). Indeed, while Human UR cannot be revived (top), Human R is successfully reignited by a single node (bottom). (o) Similar results are observed also for the Yeast network⁴⁶ ($\kappa = 10.7$, orange vs. blue circles in panel k) under $\omega = 0.4$ (Yeast UR, unrecoverable) and $\omega = 1$ (Yeast R, recoverable).

Figure 4: Recoverability of neuronal and ecological dynamics. (a) We model neuronal interactions via the Cowan-Wilson dynamics. (b) The system exhibits three phases: An inactive state $\mathbf{x}_0$ (red) when κ, ω are small, and an active state $\mathbf{x}_1$ (green) under large κ, ω . Between these two extremes we observe a bi-stable state (center, grey shaded) in which the system can reside in both $\mathbf{x}_0$ and $\mathbf{x}_1$. (c) The κ, ω phase diagram, featuring the three states, inactive (red), bi-stable (grey) and active (green). (d) Our analysis predicts a new fourth phase, splitting the bi-stable state into two distinct dynamic phases - unrecoverable (yellow) vs. recoverable (blue). Simulation results (yellow-blue transition) are in good agreement with our theoretical prediction (white solid line). To test our prediction in an empirical setting we used our Brain network⁴⁹, diluted to reside in the bi-stable phase (Supplementary Section 4.4). The resulting network with $\kappa = 6.6$ was simulated under two values of ω : Brain UR with $\omega = 2$, which is unrecoverable (orange circle), and Brain R with $\omega = 4$ - recoverable (light blue circle). (e) Indeed, we find that Brain UR fails to reignite (top), while Brain R is successfully reactivated (bottom). **Ecological dynamics.** (f) We consider symbiotic ecological interactions. The self-dynamics is captured by migration (F) complemented by logistic growth and the Alley effect; the interaction follows the Lotka-Volterra response function⁴¹ (Supplementary Section 3.3). (g) The system exhibits two phases: The active $\mathbf{x}_1$ (green) when κ, ω are large, and bi-stability of $\mathbf{x}_1$ and $\mathbf{x}_0$ (red) when κ, ω are reduced (grey shaded). (h) The κ, ω phase diagram. In the active phase (green) the system is guaranteed to reside in $\mathbf{x}_1$, however in the bi-stable phase (grey) it can be in both states, and hence, within this phase, once collapsed, the system will not recover spontaneously. (i) Once again, our analysis shows that the bi-stable phase is split, giving rise to our new recoverable phase (blue). To further support this we used an empirical plant-pollinator network⁵¹ (Eco, $\kappa = 11.1$, Supplementary Section 4.4) with two different weights: Eco UR ($\omega = 0.15$, unrecoverable, orange) and Eco R ($\omega = 0.3$, recoverable, light blue). (j) As predicted, Eco UR remains in $\mathbf{x}_0$ (top), while Eco R is successfully reignited (bottom).

Figure 5: Two-step recovery for reviving a failed system. (a) The unperturbed Yeast protein interaction network. For visibility we focus on the circled sub-network. (b) The dynamic states x_i of all nodes. As expected, the unperturbed network is in the active state $\mathbf{x}_1$, hence all $x_i > 0$. (c) - (d) Following extensive perturbation in which 30% of nodes and 30% of links (grey and red) were deleted the state of the network collapses to the inactive $\mathbf{x}_0$, *i.e.* all $x_i = 0$. The challenge is that some of the deleted components (nodes/links) are inaccessible, and hence cannot be retrieved (red). This captures the restructuring constraints that are, indeed, inevitable in realistic scenarios. Circle at center - we focus on the same sub-network shown in (a), the unperturbed network components are highlighted, the deleted nodes links appear in grey and red. (e) **Step I.** Within the given constraints we restructure the network by reintroducing nodes/links or strengthening link weights. Such structural interventions can be mapped to their impact on the two relevant control parameters κ (blue) and ω (orange). Here we track a sequence of restructuring steps. For illustration, we show the highlighted sub-network in (c) as it restructures, acquiring nodes, links and increased weights (sub-networks along the x -axis). (f) Each such sequence of restructuring steps can be mapped into a path in the κ, ω phase diagram. A successful restructuring path must lead the network from the collapsed phase (red) into the recoverable phase (blue). Our predicted phase diagram helps us design several alternative paths, affording us the flexibility to, *e.g.*, focus mainly on increasing weights ω (Net 1, light blue path) or also on enhancing network density κ (Net 3, purple path), all depending on the nature of our constraints. The resulting networks Net 1 - 3 may differ from the original unperturbed network, as indeed, our goal is not to simply reverse the damage, but to revive the system's dynamic activity. (e) **Step II.** Once the network is brought to the recoverable phase, we can revive it via single-node reigniting, demonstrated here on each of our restructured networks, Net 1 - 3.

FIGURES - FULL SIZE

Figure 1. Reviving a failed network.

Figure 2. Can a system be reignited by a single node?

figure 3. Recoverability of cellular dynamics.

Figure 4. Recoverability of neuronal and ecological dynamics.

Figure 5. Two-step recovery for reviving a failed system.

Figures

Figure 1

Reviving a failed network. (a) The components of our modeling framework: The network topology is captured by the adjacency matrix A_{ij} and the link weights ω (grey terms); the interaction mechanisms, i.e. the dynamics, are captured by the nonlinear functions $M_0(x)$, $M_1(x)$, $M_2(x)$ (orange terms). (b) Depending

on the dynamics - e.g., cellular, neuronal or ecological - the system exhibits distinct fixed-points, active (x_1 , green) or failed (x_0 , red); intermediate unstable points are marked by grey dashed lines. The transitions between these states are driven by perturbations to the topology (A_{ij} , ω). (c) Under the unperturbed topology the system resides in the functional state x_1 , in which all nodes are active, i.e. $x_i > 0$. In this presentation, which we also use throughout the paper, the network nodes are laid out on the x, y plane, and their activities x_i are captured by the z coordinate. Hence, an active system has all nodes spread out along the positive z -axis, while a failed network is laid out around $z = 0$. We also use color coding from red (small x_i) to blue (large x_i) as visual aid. (d) Perturbations to the topology, such as node/link removal or weight reduction, result in a collapse to the inactive x_0 , here having all activities vanish ($z = 0$). (e) Restructuring. To revive the failed system we must first restructure its topology to a point where it can recover, namely a point where x_1 is potentially stable. This, however, is insufficient, as often the system will not spontaneously retain its lost activity. (f) Reigniting. After restructuring we reignite the active state x_1 by controlling a microscopic set of nodes, here the single node s . The controlled node is forced to sustain a constant activity $x_s(t) = \Delta$. This activating force then spreads to impact all other nodes in the network and push them towards x_1 . (g) The state of the network in three different time-points following single-node reigniting, forcing $x_s(t) = \Delta$ (black node at center). The reigniting force gradually impacts the network, until the system's activity x_1 is restored. (h) In this process we use the natural basin structure of our dynamics. The basin B_a captures all initial states, from which the system (1) converges to the fixed-point x_a . Therefore, to reignite x_1 we seek to steer the system from B_0 (red) to any point within B_1 (green). Once in B_1 , we can cease our forcing, and the system will spontaneously transition to the desired x_1 (dashed arrows).

Figure 2

Can a system be reignited by a single node? (a) During reigniting we select a single source node s and, for a limited time, we artificially force it to sustain a permanent activity Δ . To track the system's response to such forcing, we divide the network into shells $K_s(l)$ comprising all nodes at distance l from s . (b) The reignited activity $x_s(l)$ of all nodes at $K_s(l)$ is tracked via the recurrence relation of Eq. (7), starting from our forcing at s (top equation), and tracking its propagation as it penetrates the network shells (bottom equation). Successful reigniting requires $x_s(l \rightarrow \infty) \in B1$, i.e. that the distant shells were driven towards the desired basin. (c) - (e) We can track the convergence of the recurrence relation graphically by plotting $F(x)$ (purple) and $M2(x)$ (yellow). The forcing Δ determines our initial starting point, and the recurrence can be tracked via the red or green trajectories. The final state $x_s(l \rightarrow \infty)$ is reached when the two functions, $F(x)$ and $M2(x)$, intersect. We observe three potential scenarios (Supplementary Section 2.3): Case 1. $F(x)$ and $M2(x)$ have a single intersection point in $B0$ (red). Under these conditions, regardless of the initial value (Δ) the recurrence converges to $x_s(l \rightarrow \infty) \in B0$, and hence the system is unrecoverable. Case 2. $F(x)$ and $M2(x)$ exhibit two intersections, corresponding to the system's two stable fixed points $x0$ (red) and $x1$ (green); the intermediate intersection (grey) is unstable. Here, for $\Delta < \Delta_c$ the system converges to $B0$, i.e. unrecoverable (red trajectory), while for $\Delta \geq \Delta_c$ it approaches $B1$ - hence it is recoverable (green trajectory). Case 3. In case $F(x)$ is non-monotonic the critical forcing Δ_c is determined by $F(x)$'s local maximum point (grey dashed lines), see Supplementary Section 2.3 for a detailed analysis of Case 3.

Figure 3

Recoverability of cellular dynamics. (a) Gene regulation as modeled via the Michaelis-Menten dynamics. (b) - (d) The average steady-state activity $\bar{x}$ as obtained from cellular dynamics following topological perturbation: deletion of a q -fraction of nodes; factor q reduction of weights and removal of a q -fraction of links. While the active x_1 is only stable for $q < q_c$ (green), the inactive x_0 is always stable (red). Hence the system undergoes an irreversible collapse at $q = q_c$. (e) We track three specific states, capturing the

steady-state activities x_i under varying levels of weight loss q . When q is small (left) all x_i are positive and the system is in the active state x_1 . x_i gradually decline as q increases (center), until the system collapses into the inactive state x_0 , in which all x_i vanish (right). (f) Reversing the perturbation does not revive the system. Here we retrieve the lost weights, however, since x_0 is always stable, the system remains in the collapsed state, avoiding spontaneous recovery. To revive the failed system we apply single-node reigniting: (g) The functions $F(x)$ (purple) and $M_2(x)$ (yellow) as obtained from the left/right hand sides of Eq. (10), setting $\kappa = 4$, $\omega = 0.8$. The system follows Case 1 of Fig. 2c, exhibiting a single intersection (B_0 , red), and hence it is unrecoverable. (h) Indeed, forcing node s to a permanent Δ activity (black node), the remaining shells fail to reignite. (i) Under a denser A_{ij} with $\kappa = 7$ the function $F(x)$ takes a different form and the system now follows Case 3. It is now recoverable if the forcing $\Delta \geq \Delta_c = 0.9$ (vertical dashed line). (j) As predicted, now the system is successfully activated via single-node reigniting. (k) The phase diagram in the κ, ω plane (Supplementary Section 4.3). We observe the three predicted phases: inactive (red), where only x_0 is stable, unrecoverable (yellow), where x_1 is potentially stable, but unattainable via single-node reigniting, and recoverable (blue), where the system can be revived by a single node. The theoretical prediction obtained from the roots of Eq. (10) is also shown (white solid line). (l) The phase diagram in the ω, Δ space. Here, for $\omega < 0.9$ the system is never recoverable. Above that, the higher is ω the smaller is the forcing Δ required for reigniting. Our theoretical prediction is also shown (white solid line). (m) The κ, Δ phase diagram. The slight discrepancy between theory (white solid line) and simulation (yellow-blue transition) is likely due to the discrete nature of κ . (n) To examine our prediction in an empirical setting we used the human protein interaction network⁴⁵ ($\kappa = 21.7$) to construct A_{ij} under two values of ω : Human UR with $\omega = 0.2$, in the unrecoverable phase, and Human R, with $\omega = 1$, which is recoverable (see panel k, orange vs. blue squares). Indeed, while Human UR cannot be revived (top), Human R is successfully reignited by a single node (bottom). (o) Similar results are observed also for the Yeast network⁴⁶ ($\kappa = 10.7$, orange vs. blue circles in panel k) under $\omega = 0.4$ (Yeast UR, unrecoverable) and $\omega = 1$ (Yeast R, recoverable).

Figure 4

Recoverability of neuronal and ecological dynamics. (a) We model neuronal interactions via the Cowan-Wilson dynamics. (b) The system exhibits three phases: An inactive state x_0 (red) when κ , ω are small, and an active state x_1 (green) under large κ , ω . Between these two extremes we observe a bi-stable state (center, grey shaded) in which the system can reside in both x_0 and x_1 . (c) The κ , ω phase diagram, featuring the three states, inactive (red), bi-stable (grey) and active (green). (d) Our analysis predicts a

new fourth phase, splitting the bi-stable state into two distinct dynamic phases - unrecoverable (yellow) vs. recoverable (blue). Simulation results (yellow-blue transition) are in good agreement with our theoretical prediction (white solid line). To test our prediction in an empirical setting we used our Brain network⁴⁹, diluted to reside in the bi-stable phase (Supplementary Section 4.4). The resulting network with $\kappa = 6.6$ was simulated under two values of ω : Brain UR with $\omega = 2$, which is unrecoverable (orange circle), and Brain R with $\omega = 4$ - recoverable (light blue circle). (e) Indeed, we find that Brain UR fails to reignite (top), while Brain R is successfully reactivated (bottom). Ecological dynamics. (f) We consider symbiotic ecological interactions. The self-dynamics is captured by migration (F) complemented by logistic growth and the Allee effect; the interaction follows the Lotka-Volterra response function⁴¹ (Supplementary Section 3.3). (g) The system exhibits two phases: The active x_1 (green) when κ, ω are large, and bi-stability of x_1 and x_0 (red) when κ, ω are reduced (grey shaded). (h) The κ, ω phase diagram. In the active phase (green) the system is guaranteed to reside in x_1 , however in the bi-stable phase (grey) it can be in both states, and hence, within this phase, once collapsed, the system will not recover spontaneously. (i) Once again, our analysis shows that the bi-stable phase is split, giving rise to our new recoverable phase (blue). To further support this we used an empirical plant-pollinator network⁵¹ (Eco, $\kappa = 11.1$, Supplementary Section 4.4) with two different weights: Eco UR ($\omega = 0.15$, unrecoverable, orange) and Eco R ($\omega = 0.3$, recoverable, light blue). (j) As predicted, Eco UR remains in x_0 (top), while Eco R is successfully reignited (bottom).

Figure 5

Two-step recovery for reviving a failed system. (a) The unperturbed Yeast protein interaction network. For visibility we focus on the circled sub-network. (b) The dynamic states x_i of all nodes. As expected, the unperturbed network is in the active state x_1 , hence all $x_i > 0$. (c) - (d) Following extensive perturbation in which 30% of nodes and 30% of links (grey and red) were deleted the state of the network collapses to the inactive x_0 , i.e. all $x_i = 0$. The challenge is that some of the deleted components (nodes/links) are

inaccessible, and hence cannot be retrieved (red). This captures the restructuring constraints that are, indeed, inevitable in realistic scenarios. Circle at center - we focus on the same sub-network shown in (a), the unperturbed network components are highlighted, the deleted nodes links appear in grey and red. (e) Step I. Within the given constraints we restructure the network by reintroducing nodes/links or strengthening link weights. Such structural interventions can be mapped to their impact on the two relevant control parameters κ (blue) and ω (orange). Here we track a sequence of restructuring steps. For illustration, we show the highlighted sub-network in (c) as it restructures, acquiring nodes, links and increased weights (sub-networks along the x-axis). (f) Each such sequence of restructuring steps can be mapped into a path in the κ, ω phase diagram. A successful restructuring path must lead the network from the collapsed phase (red) into the recoverable phase (blue). Our predicted phase diagram helps us design several alternative paths, affording us the flexibility to, e.g., focus mainly on increasing weights ω (Net 1, light blue path) or also on enhancing network density κ (Net 3, purple path), all depending on the nature of our constraints. The resulting networks Net 1 - 3 may differ from the original unperturbed network, as indeed, our goal is not to simply reverse the damage, but to revive the system's dynamic activity. (e) Step II. Once the network is brought to the recoverable phase, we can revive it via single-node reigniting, demonstrated here on each of our restructured networks, Net 1 - 3.

Supplementary Files

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