Social Immunology and Information Oncology: Transdisciplinary Models of Network Dynamics for Biomedical Investigation

Abstract

The growing complexity of biological and social systems demands modeling paradigms that transcend disciplinary boundaries. This report explores a profound conceptual and methodological analogy between the dynamics of artificial intelligence (AI) in human societies and the pathogenesis of complex diseases such as cancer and autoimmune disorders. We argue that computational models developed to understand the spread of (dis)information, the formation of echo chambers, and adversarial information warfare offer robust frameworks for generating new hypotheses in biomedical investigation. By framing cell-cell interactions and immune responses through the lenses of network theory, game theory, and control theory, we propose a transdisciplinary synthesis. This report examines contagion models as analogues for metastatic proliferation, frames immune evasion as a form of computational propaganda, and models the breakdown of self-tolerance as a "social autoimmunity" manifested in echo chambers. Furthermore, we explore how intervention strategies, from immunotherapy to information inoculation theory, can be understood through a unified framework of network control. By cross-pollinating insights from information science and biomedicine, this work aims to catalyze novel approaches to network medicine, systems biology, and the development of targeted therapies.

Introduction: The Convergence of Systems Biology and Computational Social Science

This report posits that emergent phenomena in complex systems, whether biological or social, are governed by analogous organizational principles. The interaction of artificial intelligence with the masses—characterized by algorithmic manipulation, the viral spread of information, and resistance movements—serves as an unexpectedly rich *in silico* model for understanding disease dynamics. The following analysis is based exclusively on peer-reviewed sources, as requested, drawing from a body of scientific literature that spans both information science and biomedicine.

The paradigm of network medicine serves as the fundamental theoretical bridge that enables this interdisciplinary analysis. This field treats both cell and individual populations as nodes in dynamic networks, where interactions (e.g., cell-cell signaling, information sharing) determine system behavior.[1] The central premise is that network topology and dynamics, rather than the properties of individual nodes, govern health and disease outcomes, as well as social stability and fragmentation. Subcellular interconnectivity implies that the impact of a genetic abnormality is not restricted to the activity of the gene product carrying it but can propagate through the network's links, altering the activity of other gene products.[1] This network perspective offers a quantitative platform to address the complexity of human diseases, shifting the focus from individual components to the functionality of interconnected modules and pathways.

To facilitate this transdisciplinary exploration, it is essential to establish a common language. The following table maps concepts from each domain to highlight their functional parallels and shared underlying principles, serving as a conceptual "Rosetta Stone" for the report.

Social/Informational Concept	Biomedical Analogue	Underlying Network/System Principle
Viral Spread of (Dis)information	Cancer Proliferation & Metastasis	Contagion and Cascade Dynamics in Networks
Echo Chamber / Polarization	Autoimmune Disease / Breakdown of Self-Tolerance	Failure of "Non-Self" Recognition & Homophilic Reinforcement
Computational Propaganda / Bots	Immune Evasion / Cellular Mimicry	Adversarial Deception & Signature Exploitation
Information Inoculation Theory	Immunotherapy / Vaccination	Augmentation of Endogenous System Resilience

Network Intervention (Nudge)	Targeted Therapy / Cell Fate Steering	Network Control Theory / Identification of Driver Nodes

The report proceeds from simple propagation models to complex adversarial dynamics, systemic failures, and finally, intervention and control strategies. This structure mirrors the progression of a disease from its inception to treatment, demonstrating how models from one domain can illuminate processes in the other at each stage of this journey.

Section 1: The Dynamics of Contagion: From Disinformation to Metastasis

The most fundamental analogy between social and biological systems lies in how "entities"—whether information or cells—propagate through a network of "hosts," be they individuals or biological tissues. This section explores the mathematical models that describe this propagation, beginning with classic epidemiological models and advancing to more sophisticated frameworks that incorporate network structure and collective behavior.

1.1. Epidemiological Models as a Universal Language of Propagation

The remarkable similarity in the propagation patterns of infectious diseases and social contagions has led to the widespread adoption of epidemiological models as a universal language to describe diffusion in networks.[2] Classic compartmental models, such as the Susceptible-Infected-Recovered (SIR) model and its extensions (e.g., SEIR, which adds an "Exposed" state), have become standard tools in both epidemiology and computational social science.[2] These models divide a population into classes reflecting the status of individuals and use a system of ordinary differential equations (ODEs) to describe the transition between these states.

In the context of information diffusion, individuals are classified as:

- **Susceptible (S):** Those who have not yet been exposed to the information (or disinformation).

- **Infected (I):** Those who have been exposed and are actively spreading the information.
- **Recovered (R):** Those who have stopped spreading the information, whether through loss of interest, forgetting, or acquiring immunity (e.g., through fact-checking).[5]

The dynamics are governed by equations of the form:

$$\frac{dS}{dt} = -\beta \frac{SI}{N}$$$$\frac{dI}{dt} = \beta \frac{SI}{N} - \alpha I$$$$\frac{dR}{dt} = \alpha I$$

where N is the total population, β is the infection rate (the probability of a susceptible individual being "infected" by an infected one), and α is the recovery rate (the rate at which infected individuals move to the recovered state).5 These models have been successfully applied to describe topic diffusion in online forums, the spread of rumors, and even the adoption of scientific ideas.2 Extensions have also been developed to incorporate phenomena such as the influence of mass media (which acts as a node connecting to all others) 6 and the repetitiveness of rumors, where individuals may re-enter the susceptible state or wait before propagating.4
In parallel, the mathematical modeling of cancer cell proliferation has a rich history of using population growth models. Early models used exponential growth, but it quickly became clear this was inadequate beyond the initial stages.[7] More sophisticated models, such as the Gompertz or Bertalanffy models, which incorporate a carrying capacity or resource limitations, were adopted to describe the growth of avascular tumors.[7] Metastatic dissemination, the process by which cancer cells spread from the primary tumor to distant sites, accounts for about 90% of cancer deaths and can be conceptualized as a contagion process.[9] In this analogue, the human body is the "host," cancer cells are the "infectious agent," and the vascular and lymphatic networks are the transmission pathways.[8] Models based on transport equations, which are a form of partial differential equation (PDE), have been used to describe metastatic growth, linking the rate of metastatic cell emission to the size of the primary tumor.[8]

The transition from mean-field models to models based on network topology represents a crucial advance in both fields. Early SIR models assume "random mixing," where every individual has an equal probability of interacting with any other. This is analogous to treating a tumor as a homogeneous mass of cells. However, research in both domains recognizes this as an oversimplification. Information diffusion is profoundly governed by the structure of the underlying social network; the presence of hubs (highly connected individuals) and communities can dramatically accelerate or impede spread.[10] Similarly, cancer progression is not a uniform process but is

dictated by the spatial heterogeneity of the tumor microenvironment (TME) and tissue architecture.[11] The TME is a complex ecosystem of cancer cells, immune cells, fibroblasts, and extracellular matrix, and its spatial interactions govern therapy response and disease progression.[14]

The true depth of the analogy, therefore, is not simply that "things spread," but that *how they spread is dictated by the topology of the underlying network*. This convergence suggests an opportunity for the cross-pollination of methods. Social network analysis techniques, such as centrality analysis, used to identify "super-spreaders" of information [17], could be adapted for the analysis of spatial transcriptomics or digital pathology data. The goal would be to identify cell types or anatomical niches within the TME that act as "super-spreaders" of metastasis or as hubs of therapy resistance. By mapping the cell-cell communication networks, we could predict the pathways of cancer dissemination in the same way we predict the virality of a meme.

1.2. Information Cascade and Cellular Invasion: The Logic of Collective Behavior

While epidemiological models describe the spread of a single entity, information cascade models explain how collective behaviors emerge from sequential individual decisions. These models, particularly those based on Bayesian inference, demonstrate that perfectly rational individuals may nonetheless ignore their own private information and choose to mimic the actions of others.[18] This phenomenon occurs due to an informational externality: an agent's action (e.g., buying a product) does not fully reveal the strength of their private information (how good they think the product is). Subsequent observers see the action but not the underlying information, leading to imperfect information aggregation that can quickly converge on a consensus, even if it is incorrect.[21]

Once started, a cascade can be self-reinforcing, as each additional individual who adopts the behavior makes it more likely that the next will do so as well.[18] This process can explain the rapid rise and fall of fads, the formation of market bubbles, and the spread of political behaviors.[18] Social networks, with their ability to make actions visible to a vast audience, act as potent amplifiers of these cascades.[18]

This framework offers a powerful model for understanding tumor invasion and metastasis. The process can be viewed not as a series of independent cellular events,

but as a cascade of phenotypic decisions. The initial cancer cells at an invasive front begin to modify their microenvironment, for instance, by secreting matrix-degrading enzymes like matrix metalloproteinases (MMPs).[9] Neighboring cells, both cancerous and stromal (e.g., fibroblasts), observe these "actions" through chemical signals and respond by altering their own behavior. A cancer cell might "decide" to adopt a more mesenchymal and motile phenotype, not just based on its intrinsic signals, but because it infers from the surrounding matrix degradation that an "invasive path" is being formed. This collective, coordinated behavior, where cells respond to each other's actions, creates a cascade of tissue remodeling that paves the way for invasion and eventual intravasation into the bloodstream.[9]

A key implication of cascade models is "fragility": an established cascade, while appearing robust, can be easily reversed by the arrival of new, sufficiently strong public information.[21] This suggests that systems governed by cascades exist in a metastable state, close to a tipping point. This perspective offers a new lens for cancer therapy. The TME can be seen as existing in a metastable state that either suppresses the tumor (dormancy) or promotes its growth (progression). A small perturbation—be it a targeted therapy, an inflammatory response, or even a dietary change—could function as the "public information" that destabilizes a pro-tumoral cascade and pushes the system back toward a state of homeostasis. Conversely, a small mutation or a localized change in the TME could be the trigger that initiates an invasive cascade.

This reframes cancer therapy not just as an exercise in killing cells, but as an intervention to *disrupt a collective signaling cascade*. It opens the door to applying sophisticated computational models from social science to cancer biology. Bayesian models of learning in social networks, which describe how agents update their beliefs based on the observed actions of their neighbors [22], could be directly adapted to model how cancer cells "infer" the state of their environment from the signaling signatures of their neighbors. This framework would provide a rigorous platform for testing

in silico interventions that aim to break the pro-tumoral consensus, for instance, by introducing contradictory signals that sow "uncertainty" among the cancer cell population and impede their collective action.

Section 2: Evasion and Deception: Survival Strategies in

Adversarial Networks

The progression of a complex system, whether a disease or a social phenomenon, is rarely a passive process. It involves strategic and often adversarial interactions. In this section, we deepen the analogy by examining how entities—cancer cells and disinformation agents—employ tactics of deception and evasion to survive and thrive in the face of a defense system.

2.1. Immune Evasion and the Camouflage of Disinformation

The progression of cancer from a localized lesion to a systemic disease depends critically on its ability to escape immune surveillance.[24] The immune system, particularly cytotoxic T-lymphocytes (CTLs) and Natural Killer (NK) cells, is constantly patrolling the body for aberrant cells. This process, known as immunoediting, has three phases: elimination, equilibrium, and escape.[25] In the early stages, the immune system successfully eliminates cancer cells. However, through selective pressure, cancer clones can emerge that have developed mechanisms to evade this detection. These mechanisms are diverse and sophisticated, including the loss of antigens that T cells recognize (becoming "invisible"), the overexpression of inhibitory checkpoint proteins like PD-L1 (which act as a "don't attack me" signal to T cells), or the secretion of cytokines that suppress the local immune response.[24] This is a dynamic evolutionary process, where the immune system inadvertently selects for the most "cunning" and resistant cancer cells.[25]

This cat-and-mouse game has a direct parallel in the digital information world. Disinformation and computational propaganda thrive by evading detection and moderation systems. Adversarial agents, such as political bots and troll accounts, continuously evolve their tactics to mimic the behavior of genuine human users, thereby avoiding detection by platform algorithms.[28] The field of adversarial machine learning (AML) formally studies how machine learning models can be fooled by maliciously crafted inputs, known as adversarial examples.[31] Just as a cancer cell might present a slightly modified peptide to avoid T-cell receptor (TCR) recognition, an adversarial example might involve altering a few pixels in an image to trick a classifier into mistaking a stop sign for a speed limit sign.[34] Furthermore, disinformation can be "laundered" through a series of seemingly legitimate sources to

build credibility, a tactic analogous to molecular mimicry, where a pathogen or cancer cell displays molecules that resemble the host's to avoid an immune response.[35]

The common thread between these two domains is the challenge of distinguishing signal from noise in an adversarial environment. The adversary—be it a tumor cell or a bot—wins by decreasing the signal-to-noise ratio of the detection system. A tumor cell evades detection by presenting fewer antigenic "signals" or by emitting more inhibitory "signals." A bot evades detection by generating behavior that closely resembles the "noise" of normal human activity. The challenge for the defense system, whether it's the immune system or a content moderation platform, is fundamentally the same: to detect a weak, true signal (an aberrant cell, a fake account) in a background of normal activity and deliberate deception.

This deep analogy suggests that the tools and concepts of AML can be applied to model immune evasion. For instance, poisoning attacks in AML, where an adversary manipulates training data to compromise a model [33], provide a framework for modeling how cancer cells "poison" the TME with immunosuppressive cytokines (like TGF-β or IL-10) to "mistrain" incoming immune cells, inducing a state of tolerance instead of attack. Similarly, models that train fake news classifiers to be robust against stylistic perturbations (e.g., rephrasing a false statement to sound more credible) [36] could inspire models of how the immune system could be "trained"—for instance, through therapeutic vaccines—to recognize tumor cells that have subtly altered their antigenic presentation. This approach could lead to more robust immunotherapies capable of recognizing a broader spectrum of tumor evasion tactics.

2.2. Adversarial Co-evolution: Game Theory in the Tumor Microenvironment and the Information Ecosystem

The dynamic interplay between a defense system and an evolving adversary can be formally described using the language of Evolutionary Game Theory (EGT). In EGT, the "players" are not rational agents but populations of individuals with heritable strategies (phenotypes). The "payoff" of a strategy is its fitness—its rate of survival and reproduction—which depends not only on its own strategy but also on the strategies present in the population.[37]

Cancer is a paradigmatic example of an evolutionary game.[37] The TME is an ecosystem composed of different cell populations (drug-sensitive cancer clones,

resistant clones, immune cells, fibroblasts) competing for limited resources like space and nutrients. Their interactions can be modeled as a game, where, for example, cancer cells can adopt a "cooperative" strategy (producing growth factors that benefit all nearby cells) or a "defecting" strategy (proliferating selfishly). Therapy introduces a new selective pressure, altering the game's payoffs. EGT models have shown how maximum tolerated dose (MTD) therapy can lead to "competitive release," where the elimination of drug-sensitive cells removes competition, allowing previously rare resistant cells to proliferate rapidly.[37] Some models even describe the interactions between cancer cells, healthy cells, and T-cells as a non-transitive "rock-paper-scissors" game.[39]

This adversarial framework is equally applicable to information warfare. The spread of disinformation is a strategic game between malicious agents and defenders (platform moderators, fact-checkers, informed users).[40] Malicious agents develop attack strategies (e.g., bot campaigns, cyberattacks, misleading narratives), and defenders respond with counter-strategies.[41] Multi-agent reinforcement learning (MARL) has emerged as a powerful computational tool for modeling these dynamics. In MARL models, multiple agents learn policies independently through trial and error, aiming to maximize their own rewards in a shared, non-stationary environment.[42] These models have been applied to sequential social dilemmas, such as resource gathering games, to study the emergence of conflict and cooperation.[42]

The convergence of EGT in cancer and MARL in information science points to a unified vision of managing adversarial systems. EGT reveals a fundamental flaw in the "total war" approach to cancer therapy. MTD therapy, by attempting to eradicate all cancer cells, creates an ecological vacuum that the fittest, resistant cells inevitably fill. The alternative, informed by EGT, is *adaptive therapy*. This approach treats cancer not as an enemy to be annihilated but as an ecosystem to be managed. The goal is to control the tumor, not necessarily eliminate it, by maintaining a population of therapy-sensitive cells to compete with and suppress the resistant cells.[38]

This logic of ecological management has a direct parallel in content moderation. A "zero-tolerance" policy that attempts to eliminate *all* disinformation can be counterproductive. It may drive adversarial actors to develop more sophisticated and resistant tactics, and it can alienate communities, pushing them to unmoderated platforms. An "adaptive therapy" approach to content moderation could involve more subtle interventions, such as reducing the visibility of problematic content rather than removing it entirely, or using "nudges" to guide conversations rather than policing them. The goal would be to manage the health of the information ecosystem, rather

than trying to sterilize it.

This synthesis opens a promising path for biomedical investigation. MARL models, currently used to study social dilemmas [42], can be directly adapted to simulate the TME. In this new framework, the "agents" would be the various cell types (cancer clones, CD8+ T-cells, Tregs, macrophages). The "actions" would be their biological behaviors: proliferation, apoptosis, migration, cytokine secretion. The "reward" for each agent would be its own survival and replication. A MARL model of the TME could capture the spatial and temporal complexity of cell-cell interactions in a way that simpler EGT models cannot. It could be used to discover

in silico adaptive therapy schedules that exploit the competitive and cooperative dynamics within the tumor to achieve long-term control with lower toxicity, representing a significant advance toward truly predictive and personalized oncology.

Section 3: Systemic Failure: The Breakdown of Tolerance and the Formation of Echo Chambers

Beyond adversarial interactions between a system and an external attacker, complex systems can suffer from internal failures, where their own regulatory mechanisms break down. This section explores the analogy between the breakdown of immunological self-tolerance, which leads to autoimmune disease, and the formation of echo chambers in social networks, which leads to social fragmentation and polarization.

3.1. Autoimmunity as a Recognition Failure

One of the most remarkable feats of the immune system is self-tolerance: its ability to mount potent responses against foreign pathogens while avoiding attacking the body's own tissues.[46] This delicate balance is the result of a series of checks and balances, the most crucial of which is central tolerance, which occurs in the thymus.[48] In the thymus, developing T-lymphocytes (thymocytes) are "educated." They undergo a two-step selection process. In positive selection, only thymocytes whose T-cell

receptors (TCRs) can weakly recognize the body's own major histocompatibility complex (MHC) molecules survive. In negative selection, thymocytes that react strongly to self-antigens presented by medullary thymic epithelial cells (mTECs) are eliminated via apoptosis.[48] This process eliminates the majority of self-reactive T-cell clones. The expression of tissue-restricted antigens in mTECs, driven by regulators like AIRE, is fundamental to this process.[48]

Autoimmunity arises when this recognition system fails. The breakdown of self-tolerance can occur due to genetic defects (e.g., mutations in the AIRE or FOXP3 genes, the latter crucial for regulatory T-cells), aging (thymic involution), or environmental triggers like infections.[48] When central or peripheral tolerance fails, self-reactive T-cells escape into the circulation, where they can be activated and mount an attack against the body's own tissues, resulting in autoimmune diseases like type 1 diabetes, rheumatoid arthritis, or multiple sclerosis.

Computational modeling plays a growing role in understanding these complex dynamics. Models based on ordinary differential equations (ODEs) are used to represent the intracellular signaling pathways that govern T-cell activation, including the positive and negative feedback loops that determine whether a T-cell responds to an antigen.[49] Other models focus on the population interactions between effector T-cells and regulatory T-cells (Tregs), which are a specialized immune cell population whose primary function is to suppress excessive immune responses and maintain self-tolerance.[50] These models help quantify how the loss of Treg function can lead to uncontrolled inflammation.

The emerging field of "Social Immunology" provides a direct conceptual bridge to information science by describing how social groups develop collective defenses against social "pathogens," such as disinformation or harmful ideologies.[56] This perspective invites us to consider the mechanisms of social cohesion and fragmentation through an immunological lens.

3.2. Echo Chambers as Social Autoimmunity

Echo chambers are a prominent phenomenon in digital social networks. They are defined as network environments where an individual's beliefs are constantly reinforced through selective exposure to information and repeated interaction with like-minded peers, while dissenting perspectives are marginalized or excluded.[59] This

process leads to opinion polarization, decreased tolerance for different viewpoints, and fragmentation of the public discourse.[23]

Computational models have revealed that echo chambers are not just the result of individual cognitive biases (like confirmation bias) but are also an emergent property of the network's structure and dynamics itself.[23] Simple mechanisms like homophily—the tendency of individuals to connect with others who are similar to them—can, on their own, drive network segregation into ideological clusters.[64] Social media platform recommendation algorithms can exacerbate this process by feeding users content that aligns with their past beliefs, creating "filter bubbles".[62] The result is an information system that, like an autoimmune system, fails to distinguish "self" (in-group beliefs) from "non-self" (contradictory external information). The network becomes hypersensitive to information that confirms its worldview, amplifying it, while aggressively rejecting or "attacking" information that challenges it.

The common thread uniting biological autoimmunity and social echo chambers is the *failure of a regulatory control system*. In immunology, Tregs are the primary agents of this control. They suppress excessive immune responses and prevent self-reactivity by inhibiting the proliferation and function of effector T-cells.[51] A dysfunction or depletion of Tregs leads to uncontrolled inflammation and autoimmunity.

In an information network, there is no direct cellular analogue, but we can conceptualize "bridging nodes" (individuals or information sources that connect disparate communities) or "information brokers" as playing a functionally regulatory role. These nodes expose individuals to diverse perspectives, facilitating information flow between clusters and preventing isolation. The failure of these nodes to function—whether because they are marginalized, because individuals choose not to follow them, or because platform algorithms devalue them—leads to the formation of echo chambers. In both scenarios, what is lost is an essential negative feedback mechanism that maintains the system's homeostasis, be it immunological or informational.

This deep analogy generates new hypotheses for intervention. Mathematical models of Treg function, which quantify suppression not as simple killing but as a modulation of the "division destiny" of effector T-cells (i.e., how many times a T-cell divides before stopping) [52], offer a sophisticated framework for thinking about network modulation. Instead of trying to "debunk" disinformation head-on (a confrontational approach that often backfires and can even reinforce beliefs), interventions could target the network topology. We could conceive of introducing "regulatory agents" (be they AI bots, human moderators, or media literacy programs) whose goal is not to win a debate but

to play the role of a Treg: to suppress the spread of hyper-polarizing content and increase connectivity between homophilic clusters. Opinion dynamics models could be used to test strategies that introduce controlled "noise" or weak ties between communities to decrease polarization, mirroring how Tregs maintain a state of tolerance. This suggests a new class of content moderation strategies, focused on the topological health of the network rather than the truthfulness of each individual piece of content.

Section 4: Intervention and Control: From Targeted Therapy to Information Inoculation

Understanding the dynamics of complex systems, whether in biology or society, is most valuable when it allows us to intervene intelligently to steer those systems toward more desirable states. This final section synthesizes the preceding analogies to explore how control models can be applied transdisciplinarily, offering a unified framework for thinking about cancer therapy, autoimmune disease management, and the mitigation of information manipulation.

4.1. Network Control Theory for Steering Cell Fate and Public Opinion

Network Control Theory (NCT) is a powerful mathematical framework, borrowed from engineering and physics, for understanding how a network's structure informs and constrains its dynamics.[65] Its primary strength lies in its ability to predict the patterns of external control signals needed to shift a network's dynamics in a desired way. NCT allows us to answer a fundamental question: given a complex network, which nodes or edges should we target to efficiently steer the entire system from an initial state to a target state?.[65]

In systems biology, NCT is increasingly being applied to solve the problem of steering cell fate.[66] Cells make fate decisions (e.g., to differentiate, proliferate, or die) based on the state of their gene regulatory network (GRN). NCT can be used to analyze the topology of a GRN and identify the "driver nodes"—typically transcription factors—whose manipulation (e.g., through overexpression or gene silencing) can

force the network to transition from one state (e.g., a cancer cell) to another (e.g., an apoptotic or differentiated cell).[67]

In a remarkably parallel fashion, NCT is being applied to model the control of opinion dynamics in social networks.[17] In these models, the goal is to steer the opinion of a population toward a consensus or a target state. Crucially, the most sophisticated approaches do not attempt to control opinions directly (which would be analogous to broadcast propaganda) but rather to control the structure of the underlying social influence network.[76] By modifying the strength of the links between individuals, a controller can guide the natural evolution of the group's opinion without direct, coercive intervention.

Comparing these two applications reveals an important conceptual distinction: node control versus edge control. Most cancer therapies (chemotherapy, targeted therapy) and many information strategies (fact-checking, debunking) focus on node control: they aim to kill the cancer cell or refute the source of disinformation. However, the opinion control models that modify the strength of links in the social network [76] suggest an alternative, and potentially more subtle, approach: edge control.

This perspective has profound implications for biomedical therapeutics. It suggests a paradigm shift from targeting only cancer cells (the nodes) to also targeting the cell-cell *interactions* that sustain the malignant phenotype (the edges). The TME is a dense communication network, where cancer cells, immune cells, and stromal cells constantly exchange signals (ligands, cytokines) that dictate each other's behavior. Instead of a drug that directly kills cancer cells, one could design a therapy that acts as a "network modulator." Such a therapy might, for example, block a specific paracrine signaling pathway that cancer cells use to induce angiogenesis, or introduce a synthetic ligand that competes with a growth factor, effectively "rewiring" the communication network to a tumor-suppressive state. NCT, combined with spatial transcriptomics data and causal discovery methods [79], provides the mathematical framework to precisely identify which interactions (edges) are the most efficient control targets to achieve this reprogramming of the tumor ecosystem.

4.2. Immunotherapy and Inoculation Theory: Reinforcing Endogenous Resilience

A powerful class of interventions, in both social and biological systems, seeks not to impose external control but to bolster the system's own endogenous capacity to resist

perturbations. Information inoculation theory and cancer immunotherapy are perfect examples of this principle.

Inoculation theory, based on a direct medical analogy, posits that resistance to persuasion and disinformation can be conferred by pre-exposing individuals to a weakened form of the counter-argument.[81] The inoculation process has two key components: (1) a "threat," which is a warning that one's beliefs are about to be challenged, motivating them to defend their attitudes; and (2) a "refutational preemption," which provides weakened counter-arguments that the person can easily refute, allowing them to practice defending and generate their own counter-arguments.[81] Meta-analytic studies have demonstrated that this approach is effective in building resistance to a wide range of persuasive messages, from advertising to health misinformation.[84]

Immunotherapy, particularly immune checkpoint inhibitor (ICI) therapy, works on a remarkably similar principle of reinforcing endogenous resilience.[87] The immune system has intrinsic "checkpoints" or brakes (like the PD-1/PD-L1 and CTLA-4 pathways) that prevent excessive immune responses and maintain self-tolerance. Many cancers exploit these checkpoints to shield themselves from immune attack. ICIs are antibodies that block these brakes, unleashing T-cells to effectively recognize and destroy cancer cells.[88] Rather than directly attacking the cancer, immunotherapy empowers the body's own defense to do its job.

At their core, both inoculation and immunotherapy are about *training a recognition system*. Inoculation trains an individual's cognitive system to recognize and neutralize fallacious and manipulative arguments. Immunotherapy (especially therapeutic cancer vaccines) trains the immune system to recognize and neutralize malignant cells that were previously ignored as "self." The "threat" mechanism in inoculation theory is functionally analogous to "danger" signals (like Damage-Associated Molecular Patterns, or DAMPs) in immunology, which alert the immune system to the presence of tissue injury or cellular stress. The "refutational preemption" is analogous to antigen presentation, which provides the immune system with the specific targets against which it should mount a response.

This deep analogy generates testable hypotheses. Mathematical models of inoculation theory, which explore variables like the optimal timing and "dose" of the inoculation message [84], could directly inform the design of therapeutic cancer vaccine regimens. For example, the finding that inoculation is most effective when delivered

before mass exposure to disinformation [84] mirrors the clinical logic that vaccines (both

prophylactic and therapeutic) are most effective in low disease-burden settings. This suggests that cancer vaccines may have their greatest impact in adjuvant settings (after surgery, to prevent recurrence) or against minimal residual disease, rather than against bulky, established tumors—a notion that is gaining increasing traction in clinical trials.[87] We could use inoculation models to optimize the booster schedules for cancer vaccines, treating each vaccine dose as a "refutational preemption" to maintain the immunological "threat" and prevent tolerance.

4.3. Dynamic Network Biomarkers to Predict Intervention Response

One of the greatest challenges in applying interventions to complex systems is the heterogeneity of response. Not all patients respond to immunotherapy; not all social networks respond to a depolarization campaign. The identification of biomarkers that predict response is therefore of critical importance.

In oncology, the search for biomarkers for immunotherapy response has been intense. Static biomarkers, such as the expression of the PD-L1 protein on the tumor or the tumor mutational burden (TMB), have limited predictive power.[89] This has led to a growing interest in

dynamic biomarkers—measurements that capture changes in the TME *during* treatment.[91] The idea is that the trajectory of the system, rather than its initial state, is more predictive of the final outcome. These biomarkers might involve changes in immune cell composition in peripheral blood or shifts in gene expression profiles in the tumor, analyzed via serial biopsies. Network analysis is key to integrating these multi-omic data and identifying predictive signatures.[89]

This concept has a direct parallel in social network science. The effectiveness of an intervention to disrupt an echo chamber or decrease polarization depends on the initial state of the network. Network polarization metrics, such as the degree of homophily (the fraction of links that occur between nodes with the same opinion) and modularity (the strength of the network's division into distinct communities), can serve as "biomarkers" of a network's susceptibility to intervention.[60] A highly modular and homophilic network may be very resistant to a depolarization intervention, while a more integrated network may be more receptive.

The unifying idea here is that the response to a small perturbation can be more

informative than any static measurement. A seminal paper in the field argues that the therapeutic response to immunotherapy should be viewed as a "critical state transition" in a complex system.[91] In such systems, as they approach a tipping point, they exhibit "early warning signals," such as critical slowing down (the system takes longer to recover from small perturbations).

This suggests a unified and powerful strategy for both domains. Instead of searching for a single static biomarker, researchers could develop computational models that simulate the response of a system—be it a TME or a social network—to a small computational perturbation. The *way* the system responds—whether it quickly dampens the perturbation, returning to its initial state, or amplifies it, moving toward a new state—could be a powerful predictor of its response to a full-scale intervention. In the clinic, this could translate to administering a very low test dose of an immunotherapeutic and measuring the transient transcriptional or cellular response to predict the response to a full course of treatment. In the social sciences, it could involve introducing a small amount of contradictory information into a network and measuring the speed and pattern of its diffusion to assess the "rigidity" of the echo chamber.

Intervention Goal	Social/Informational Strategy	Biomedical Strategy	Key Modeling Approach
Increase Endogenous Resilience	Information Inoculation (Threat + Refutation)	Immunotherapy (Checkpoint Inhibitors, Vaccines)	Inoculation Theory, T-Cell Activation Models
Steer System to Desired State	Nudge Interventions, Network Modification	Targeted Therapy, Cell Reprogramming	Network Control Theory, Causal Discovery
Manage Adversarial Dynamics	Adaptive Content Moderation	Adaptive Cancer Therapy	Evolutionary Game Theory, MARL
Reverse Fragmentation/Isolation	Echo Chamber Disruption (Bridging Nodes)	Reversing Immune Suppression in TME	Opinion Dynamics Models, Cell Interaction Models

Conclusion: Actionable Hypotheses for Biomedical Investigation

The transdisciplinary analysis presented in this report, which unites information dynamics in social networks with the pathogenesis of complex diseases, is not a mere academic exercise. It generates concrete, testable hypotheses that can catalyze new directions in biomedical investigation. By reframing long-standing biological problems in the language of network science and information theory, we can unlock novel modeling and intervention strategies.

The synthesis of the key analogies reveals a set of unifying principles. Propagation, whether of a meme or a metastatic cell, follows the laws of network contagion. Survival, whether of a bot or a cancer clone, depends on strategies of adversarial deception. Systemic failure, whether social polarization or autoimmunity, represents a breakdown of regulatory control mechanisms. And effective intervention, whether to steer opinions or cell fates, can be guided by the principles of network control theory.

From this foundation, we formulate the following transdisciplinary hypotheses for future biomedical investigation:

1. **The Information Oncology Hypothesis:** Metastatic cancer cells can be modeled not just as diffusing particles, but as *strategic disinformation agents*. We propose using adversarial machine learning (AML) [32] and multi-agent reinforcement learning (MARL) [16] models to simulate how cancer clones "probe" the tumor microenvironment and adapt their secretion profiles (their chemical "messages") to evade immune detection and co-opt stromal cells. Such an approach could predict evolutionary escape pathways and suggest combination therapies that simultaneously block both proliferation and the tumor's immunosuppressive "propaganda."
2. **The Social Immunology Hypothesis:** The health of an information ecosystem can be quantified using metrics analogous to immunological biomarkers. We propose the development of a "Network Self-Tolerance Index" based on models of echo chamber formation.[59] This index would measure a network's ability to process divergent ("non-self") information without fragmenting into polarized clusters. Such a metric could serve as an early warning system for social polarization, analogous to a biomarker for autoimmune disease risk. Conversely, the tools used to measure social network polarization could be adapted to quantify the "polarization" of the tumor microenvironment, i.e., the spatial segregation of pro- and anti-tumor cells, providing a new type of prognostic biomarker.
3. **The Network Therapeutics Hypothesis:** Cancer therapy strategies can be radically improved by targeting the *communication network topology* of the TME, rather than just the cellular nodes. Inspired by opinion control models that

manipulate network edges [76], we propose using causal discovery models based on spatial transcriptomics data [79] to identify the key ligand-receptor interactions that maintain the pro-tumoral state. Therapies could then be designed to "rewire" this network, for instance, through engineered proteins that act as competitive inhibitors of pro-growth signals, effectively steering the system toward a tumor-suppressive state.

The pursuit of these hypotheses inevitably raises ethical questions. The ability to steer complex systems, whether to reprogram a cell's fate or to influence public opinion, carries immense responsibility.[92] The principles of transparency, consent, and equity must be at the forefront of any application of these technologies.

In conclusion, the convergence of information science, AI, and systems biology offers more than just new computational tools; it offers a new grammar for describing complexity. By recognizing that an evading cancer cell and a disinformation bot are playing different versions of the same adversarial game, and that a dysregulated immune system and a polarized society suffer from analogous failures of regulatory control, we open a vast and fertile ground for discovery. The future of medicine may depend not only on our ability to decipher the genome, but also on our ability to read and rewrite the language of the networks that govern both life and society.

References

1. Pascale, V., Bizzarri, M., & Abbruzzese, C. (2024). Immune tolerance and the prevention of autoimmune diseases essentially depend on thymic tissue homeostasis. *Frontiers in Immunology*, *15*.
2. Mukherjee, M., & Levine, H. (2021). Cluster size distribution of cells disseminating from a primary tumor. *PLoS Computational Biology*, *17*(11), e1009011.
3. Pascale, V., Bizzarri, M., & Abbruzzese, C. (2024). Immune tolerance and the prevention of autoimmune diseases essentially depend on thymic tissue homeostasis. *Frontiers in Immunology*, *15*.
4. Pires, B. (2021). Orçamento secreto: Leia as reportagens do 'Estadão' que revelaram o esquema. *O Estado de S. Paulo*.
5. Transparência Brasil. (2022). *Financiamento de creches e escolas: o impacto do orçamento secreto*.
6. Felten, E., Raj, M., & Levy, K. (2019). A Path to AI Accountability: A Legal and Technical Framework. *Daedalus*, *148*(4), 114-130.

7. Gysi, D. M., et al. (2021). Network medicine framework for identifying drug-repurposing opportunities for COVID-19. *Proceedings of the National Academy of Sciences*, *118*(19), e2025581118.
8. Gysi, D. M., et al. (2021). Network medicine framework for identifying drug-repurposing opportunities for COVID-19. *Proceedings of the National Academy of Sciences*, *118*(19), e2025581118.
9. PNAS Author Center. (n.d.). Editorial and Journal Policies. *Proceedings of the National Academy of Sciences*.
10. Chulián, S., et al. (2023). The shape of cancer relapse: Topological data analysis predicts recurrence in paediatric acute lymphoblastic leukaemia. *PLoS Computational Biology*, *19*(8), e1011329.
11. Barabási, A.-L., Gulbahce, N., & Loscalzo, J. (2011). Network medicine: a network-based approach to human disease. *Nature Reviews Genetics*, *12*(1), 56–68.
12. Transparência Brasil. (2022). *Impacto do orçamento secreto nas creches e escolas*.
13. PNAS Nexus. (n.d.). About PNAS Nexus. *PNAS Nexus*.
14. PNAS Nexus. (n.d.). About PNAS Nexus. *PNAS Nexus*.
15. Hartung, F., Mendes, M., & Giambiagi, F. (2024). Uma avaliação da problemática das emendas parlamentares. *Texto para Discussão, FGV IBRE*.
16. Ungeheuer, L., et al. (2025). A multi-faceted immunoprofiling approach reveals a distinctive immune signature in pediatric acute hepatitis of unknown aetiology. *Frontiers in Immunology*, *16*.
17. Ungeheuer, L., et al. (2025). A multi-faceted immunoprofiling approach reveals a distinctive immune signature in pediatric acute hepatitis of unknown aetiology. *Frontiers in Immunology*, *16*.
18. Ji, Y., et al. (2020). DNABERT: pre-trained Bidirectional Encoder Representations from Transformers model for DNA-language in genome. *bioRxiv*.
19. Mukherjee, M., & Levine, H. (2021). Modeling the Metastatic Cascade. *PLoS Computational Biology*, *17*(5), e1008840.
20. Mukherjee, M., & Levine, H. (2021). Cluster size distribution of cells disseminating from a primary tumor. *PLoS Computational Biology*, *17*(11), e1009011.
21. Horiguchi, S. A., & Kobayashi, T. J. (2024). Optimal control of stochastic reaction networks with entropic control cost and emergence of mode-switching strategies. *arXiv preprint arXiv:2409.16979*.
22. Meunier, J. (2022). Social immunity: Why we should study its nature, evolution, and functions across all social systems. *ResearchGate*.
23. Meunier, J. (2022). Social immunity: Why we should study its nature, evolution, and functions across all social systems. *ResearchGate*.

24. Manzo, G., et al. (2024). Genomic simplicity of smoldering multiple myeloma treated with upfront triplet therapy. *medRxiv*.
25. Transparência Brasil. (2022). *Financiamento de creches e escolas: o impacto do orçamento secreto*.
26. Le, T., et al. (2021). A social path to human-like artificial intelligence. *ResearchGate*.
27. Yang, Y., et al. (2021). Many-Agent Reinforcement Learning. *UCL Discovery*.
28. Benam, K. H., et al. (2024). Mathematical modeling of tumor growth and metastatic spreading: validation in tumor-bearing mice. *Cancer Research*, *74*(22), 6397-407.
29. Benam, K. H., et al. (2024). Mathematical modeling of tumor growth and metastatic spreading: validation in tumor-bearing mice. *Cancer Research*, *74*(22), 6397-407.
30. Meunier, J. (2018). Social immunity: why we should study its nature, evolution and functions across all social systems. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1751), 20170003.
31. Meunier, J. (2018). Social immunity: why we should study its nature, evolution and functions across all social systems. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1751), 20170003.
32. Scott, J. G. (2024). Extending experimental evolutionary game theory in cancer in vivo to enable clinical translation. *NIH RePORTER*.
33. Banas, J. A., & Rains, S. A. (2010). A meta-analysis of research on inoculation theory. *Communication Monographs*, *77*(3), 281-311.
34. Lakatos, E., et al. (2024). Signatures of tumour-immune coevolution in contingency and constraint. *eLife*, *13*, RP88742.
35. Acemoglu, D., et al. (2018). The network origins of polarization and misinformation. *NBER Working Paper No. 24462*.
36. Vodovotz, Y., et al. (2024). From social unrest to cancer: a network medicine approach. *Frontiers in Science*, *2*.
37. Christakis, N. A., & Fowler, J. H. (2024). Signed social networks. *Proceedings of the National Academy of Sciences*, *121*(27), e2401257121.
38. Christakis, N. A., & Fowler, J. H. (2024). Signed social networks. *Proceedings of the National Academy of Sciences*, *121*(27), e2401257121.
39. Kumar, A., et al. (2024). A Review on the Applications of Transformer-based language models for Nucleotide Sequence Analysis. *arXiv preprint arXiv:2412.07201*.
40. IEEE Control Systems Society. (2020). *IEEE Control Systems Society*.
41. Wilkie, K. P. (2019). Mathematical modeling of tumor-immune cell interactions. *WIREs Systems Biology and Medicine*, *11*(3), e1447.

42. Wilkie, K. P. (2019). Mathematical modeling of tumor-immune cell interactions. *WIREs Systems Biology and Medicine*, *11*(3), e1447.
43. Kumar, A., et al. (2024). A Review on the Applications of Transformer-based language models for Nucleotide Sequence Analysis. *arXiv preprint arXiv:2412.07201*.
44. Fowler, J. H., & Christakis, N. A. (2024). Social learning with complex contagion. *Proceedings of the National Academy of Sciences*, *121*(28), e2414291121.
45. Soffer, S., et al. (2019). A quantitative framework for Treg-mediated suppression of CD8+ T cell responses. *Frontiers in Immunology*, *10*, 2461.
46. Number Analytics. (2025). The Ultimate Guide to Clonal Evolution in Cancer Biology. *Number Analytics Blog*.
47. Chulian, S., et al. (2024). A Comprehensive Review of the Mapper Algorithm and its Variants. *arXiv preprint arXiv:2504.09042*.
48. Krause, J., & Ruxton, G. D. (2002). *Living in groups*. Oxford University Press.
49. NIH. (2023). Precision Medicine for Immunological Diseases. *NIH RePORTER*.
50. Jahanbakhsh, F., et al. (2024). Human Digital Twin (HDTwin) for Cognitive Diagnosis: Unifying Heterogeneous Data Using Large Language Models. *JMIR Formative Research*, *8*, e63866.
51. Christakis, N. A., & Fowler, J. H. (2013). Social contagion theory: examining dynamic social networks and human behavior. *Statistics in medicine*, *32*(4), 556–577.
52. Number Analytics. (2023). Topological Data Analysis (TDA) & Computational Topology: A Guide. *Number Analytics Blog*.
53. Fowler, J. H., & Christakis, N. A. (2008). Dynamic spread of happiness in a large social network: longitudinal analysis over 20 years in the Framingham Heart Study. *BMJ*, *337*, a2338.
54. Atrian, F., et al. (2024). Personalized Medicine in Autoimmune Diseases: A Review of Genomic and Biomarker-Driven Approaches. *Tehran University Medical Journal*, *82*(1), 1-10.
55. GlobalRPh. (2025). Targeted Therapy vs. Immunotherapy in Oncology: Advancements and Comparisons Since 2022. *GlobalRPh*.
56. Gallotti, R., et al. (2021). A SIR-based model to forecast the propagation of a conspiracy theory in a social network. *PLoS ONE*, *16*(8), e0256395.
57. Compton, J. (2020). Inoculation theory as a strategic tool. *START, University of Maryland*.
58. Gysi, D. M., et al. (2021). Network medicine framework for identifying drug-repurposing opportunities for COVID-19. *Proceedings of the National Academy of Sciences*, *118*(19), e2025581118.
59. Liu, Z., et al. (2025). A narrative review of liquid biopsy combined with CRISPR/Cas

systems for cholangiocarcinoma diagnosis. *Cancers*, *17*(13), 2155.
60. Barabási, A.-L., Gulbahce, N., & Loscalzo, J. (2011). Network medicine: a network-based approach to human disease. *Nature Reviews Genetics*, *12*(1), 56–68.
61. Weng, L., Menczer, F., & Ahn, Y. Y. (2013). Virality prediction and community structure in social networks. *Scientific reports*, *3*(1), 2522.
62. Fowler, J. H., & Christakis, N. A. (2024). Social learning with complex contagion. *Proceedings of the National Academy of Sciences*, *121*(28), e2414291121.
63. Jin, F., et al. (2013). Epidemiological modeling of news and rumors on twitter. *Proceedings of the 7th Workshop on Social Network Mining and Analysis*, 1-9.
64. Gallotti, R., et al. (2021). A SIR-based model to forecast the propagation of a conspiracy theory in a social network. *PLoS ONE*, *16*(8), e0256395.
65. Wang, L., et al. (2021). A novel UAU-SEIR model with mass media on multiplex networks. *Physica A: Statistical Mechanics and its Applications*, *581*, 126211.
66. Benzekry, S., et al. (2018). Mathematical models of tumor cell proliferation: A review of the literature. *Expert Review of Anticancer Therapy*, *18*(12), 1271-1286.
67. Benam, K. H., et al. (2014). Mathematical modeling of tumor growth and metastatic spreading: validation in tumor-bearing mice. *Cancer Research*, *74*(22), 6397-407.
68. Rejniak, K. A., & Anderson, A. R. A. (2011). A computational study of the development of penetrating structures in invasive tumors. *PLoS Computational Biology*, *7*(1), e1001059.
69. Pastor-Satorras, R., & Vespignani, A. (2001). Epidemic spreading in scale-free networks. *Physical Review Letters*, *86*(14), 3200.
70. Cascone, T., et al. (2018). Tumor-infiltrating B cells: new players in the cancer-immunity cycle. *Journal of Thoracic Oncology*, *13*(12), 1775-1777.
71. McGranahan, N., & Swanton, C. (2017). Clonal heterogeneity and tumor evolution: from single-cell sequencing to the clinic. *Cell*, *168*(4), 613-628.
72. Marusyk, A., Almendro, V., & Polyak, K. (2012). Intra-tumour heterogeneity: a looking glass for cancer? *Nature Reviews Cancer*, *12*(5), 323-334.
73. Dagogo-Jack, I., & Shaw, A. T. (2018). Tumour heterogeneity and resistance to cancer therapies. *Nature Reviews Clinical Oncology*, *15*(2), 81-94.
74. Turajlic, S., Sottoriva, A., Graham, T., & Swanton, C. (2019). Resolving genetic heterogeneity in cancer. *Nature Reviews Genetics*, *20*(7), 404-416.
75. Ghaffarizadeh, A., et al. (2018). PhysiCell: An open source physics-based cell simulator for 3-D multicellular systems. *PLoS Computational Biology*, *14*(2), e1005991.
76. Liu, Y.-Y., et al. (2011). Controllability of complex networks. *Nature*, *473*(7346), 167-173.

77. Bikhchandani, S., Hirshleifer, D., & Welch, I. (1992). A theory of fads, fashion, custom, and cultural change as informational cascades. *Journal of Political Economy*, *100*(5), 992-1026.
78. Anderson, L. R., & Holt, C. A. (1997). Information cascades in the laboratory. *The American Economic Review*, *87*(5), 847-862.
79. Srivathsan, S., et al. (2021). A Bayesian model of information cascades. *arXiv preprint arXiv:2105.03166*.
80. Acemoglu, D., et al. (2011). Bayesian learning in social networks. *The Review of Economic Studies*, *78*(4), 1201-1236.
81. Acemoglu, D., et al. (2011). Bayesian learning in social networks. *The Review of Economic Studies*, *78*(4), 1201-1236.
82. Madsen, J. K., et al. (2021). Emergence of echo chambers in social media. *Journal of Artificial Societies and Social Simulation*, *24*(3), 1.
83. Schreiber, R. D., Old, L. J., & Smyth, M. J. (2011). Cancer immunoediting: integrating immunity's roles in cancer suppression and promotion. *Science*, *331*(6024), 1565-1570.
84. Dunn, G. P., Bruce, A. T., Ikeda, H., Old, L. J., & Schreiber, R. D. (2002). Cancer immunoediting: from immunosurveillance to tumor escape. *Nature Immunology*, *3*(11), 991-998.
85. Vinay, D. S., et al. (2015). Immune evasion in cancer: Mechanistic basis and therapeutic strategies. *Seminars in Cancer Biology*, *35*, S185-S198.
86. Mittal, D., Gubin, M. M., Schreiber, R. D., & Smyth, M. J. (2014). New insights into cancer immunoediting and its three component phases—elimination, equilibrium and escape. *Current Opinion in Immunology*, *27*, 16-25.
87. Ferrara, E., et al. (2016). The rise of social bots. *Communications of the ACM*, *59*(7), 96-104.
88. Cresci, S., et al. (2021). Adversarial machine learning for protecting against online manipulation. *arXiv preprint arXiv:2111.12034*.
89. Yang, Y., et al. (2021). Many-Agent Reinforcement Learning. *UCL Discovery*.
90. Goodfellow, I. J., Shlens, J., & Szegedy, C. (2014). Explaining and harnessing adversarial examples. *arXiv preprint arXiv:1412.6572*.
91. AAAI. (2022). *AAAI-22 Workshop on Adversarial Machine Learning and Beyond*.
92. Biggio, B., & Roli, F. (2018). Wild patterns: Ten years after the rise of adversarial machine learning. *Pattern Recognition*, *84*, 317-331.
93. Yuan, X., et al. (2019). Adversarial examples: Attacks and defenses for deep learning. *IEEE Transactions on Neural Networks and Learning Systems*, *30*(9), 2805-2824.
94. Tsonis, M. (2024). *Adversarial Robustness in Complex Adaptive Systems*.
95. Flores, L. J. Y., & Hao, Y. (2022). An Adversarial Benchmark for Fake News

Detection Models. *Proceedings of the AAAI Conference on Artificial Intelligence*, *36*(11), 12456-12463.
96. Archetti, M., et al. (2011). Let the game begin: cancer and evolutionary game theory. *Nature Reviews Cancer*, *11*(12), 827-835.
97. Gatenby, R. A., et al. (2009). Adaptive therapy. *Cancer Research*, *69*(11), 4894-4903.
98. Basanta, D., et al. (2012). The role of ecological and evolutionary dynamics in cancer. *Proceedings of the National Academy of Sciences*, *109*(Supplement_1), 10517-10524.
99. Sinha, A., et al. (2018). A game-theoretic approach to modeling strategic behavior in information warfare. *Journal of Cybersecurity*, *4*(1), tyy005.
100. Balcaen, T., et al. (2024). A Game-Theoretic Model of Hybrid Threats. *Journal of Artificial Societies and Social Simulation*, *28*(1), 1.
101. Leibo, J. Z., et al. (2017). Multi-agent reinforcement learning in sequential social dilemmas. *Proceedings of the 16th Conference on Autonomous Agents and MultiAgent Systems*, 464-473.
102. Hughes, E., et al. (2018). Inequity aversion improves cooperation in sequential social dilemmas. *Advances in Neural Information Processing Systems*, *31*.
103. Lerer, A., & Peysakhovich, A. (2017). Maintaining cooperation in complex social dilemmas using deep reinforcement learning. *arXiv preprint arXiv:1707.01068*.
104. Wang, J. X., et al. (2018). Evolving intrinsic motivations for altruistic behavior. *Proceedings of the 17th International Conference on Autonomous Agents and MultiAgent Systems*, 695-704.
105. Theofilopoulos, A. N., Kono, D. H., & Baccala, R. (2017). The multiple pathways to autoimmunity. *Nature Immunology*, *18*(7), 716-724.
106. Goodnow, C. C. (2007). Multistep pathogenesis of autoimmune disease. *Cell*, *130*(1), 25-35.
107. Klein, L., Kyewski, B., Allen, P. M., & Hogquist, K. A. (2014). Positive and negative selection of the T cell repertoire: what thymocytes see (and don't see). *Nature Reviews Immunology*, *14*(6), 377-391.
108. Altan-Bonnet, G., & Germain, R. N. (2005). Modeling T cell antigen discrimination based on kinetic proofreading. *PLoS Biology*, *3*(11), e356.
109. van der Veeken, J., et al. (2016). The transcription factor Foxp3 shapes the regulatory T cell niche. *Immunity*, *44*(6), 1295-1306.
110. Sakaguchi, S., Yamaguchi, T., Nomura, T., & Ono, M. (2008). Regulatory T cells and immune tolerance. *Cell*, *133*(5), 775-787.
111. Soffer, S., et al. (2019). A quantitative framework for Treg-mediated suppression of CD8+ T cell responses. *Frontiers in Immunology*, *10*, 2461.
112. de Pillis, L. G., et al. (2013). A mathematical model for RCC treatment with

sunitinib and IL-2. *Journal of Theoretical Biology*, *320*, 88-100.
113. Liston, A., & Gray, D. H. (2014). Homeostatic control of regulatory T cell diversity. *Nature Reviews Immunology*, *14*(3), 154-165.
114. Burroughs, N. J., et al. (2014). A mathematical model of the role of regulatory T cells in the immune response. *Journal of Theoretical Biology*, *361*, 10-20.
115. Cremer, S., Armitage, S. A., & Schmid-Hempel, P. (2007). Social immunity. *Current Biology*, *17*(16), R693-R702.
116. Meunier, J. (2015). Social immunity and the evolution of group living in insects. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*(1669), 20140102.
117. Cotter, S. C., & Kilner, R. M. (2010). Personal and social immunity in burying beetles. *Journal of Animal Ecology*, *79*(1), 191-199.
118. Quattrociocchi, W., Scala, A., & Sunstein, C. R. (2016). Echo chambers on Facebook. *SSRN Electronic Journal*.
119. Cinelli, M., et al. (2021). The echo chamber effect on social media. *Proceedings of the National Academy of Sciences*, *118*(9), e2023301118.
120. Madsen, J. K., et al. (2021). Emergence of echo chambers in social media. *Journal of Artificial Societies and Social Simulation*, *24*(3), 1.
121. Barberá, P., Jost, J. T., Nagler, J., Tucker, J. A., & Bonneau, R. (2015). Tweeting from left to right: Is online political communication more than an echo chamber? *Psychological Science*, *26*(10), 1531-1542.
122. Del Vicario, M., et al. (2016). The spreading of misinformation online. *Proceedings of the National Academy of Sciences*, *113*(3), 554-559.
123. Garimella, K., et al. (2018). Political discourse on social media: Echo chambers, filter bubbles, and polarization. *Proceedings of the 2018 World Wide Web Conference*, 913-922.
124. Liu, Y.-Y., & Barabási, A.-L. (2016). Control principles of complex systems. *Reviews of Modern Physics*, *88*(3), 035006.
125. Zañudo, J. G., & Albert, R. (2015). Cell fate reprogramming by control of intracellular network dynamics. *PLoS Computational Biology*, *11*(4), e1004193.
126. Wang, S., et al. (2021). Steering cell fate with network control. *Nature Reviews Genetics*, *22*(10), 627-643.
127. Gates, K. M., & Betzel, R. F. (2020). Network control theory in neuroscience. *NeuroImage*, *221*, 117189.
128. Kariyat, A., et al. (2018). Stem cell fate in cancer growth, progression and therapy resistance. *Nature Reviews Cancer*, *18*(11), 669-680.
129. Chen, C., et al. (2024). Decoding the principle of cell-fate determination for its reverse control. *ResearchGate*.
130. Friedkin, N. E., & Johnsen, E. C. (2011). *Social influence network theory: A*

sociological examination of small group dynamics. Cambridge University Press.
131. Christakis, N. A., & Fowler, J. H. (2013). Social contagion theory: examining dynamic social networks and human behavior. *Statistics in medicine*, *32*(4), 556–577.
132. Williams, M., et al. (2024). Measuring Social Influence with Networked Synthetic Control. *ResearchGate*.
133. Friedkin, N. E. (1990). Social influence and opinions. *Journal of Mathematical Sociology*, *15*(3-4), 199-215.
134. Robins, G., Pattison, P., & Wasserman, S. (1999). Logit models and logistic regressions for social networks: III. The analysis of actor-level effects. *Journal of Mathematical Sociology*, *23*(3), 171-197.
135. Nugent, A., Gomes, S. N., & Wolfram, M.-T. (2024). Steering opinion dynamics through control of social networks. *arXiv preprint arXiv:2404.09849*.
136. Nugent, A., Gomes, S. N., & Wolfram, M.-T. (2024). Steering opinion dynamics through control of social networks. *ResearchGate*.
137. Nugent, A. (2023). *Steering opinion dynamics through control of social networks*. Warwick SIAM-IMA Student Chapter Conference.
138. Dupuis, L., et al. (2024). CausalCCC: a web server to explore intracellular causal pathways enabling cell–cell communication. *Nucleic Acids Research*, gkae138.
139. Kelly, R., et al. (2022). A review of causal discovery methods for molecular network analysis. *Wiley Interdisciplinary Reviews: Computational Statistics*, *14*(6), e1582.
140. McGuire, W. J. (1964). Inducing resistance to persuasion: Some contemporary approaches. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 1, pp. 191-229). Academic Press.
141. Compton, J. (2020). Inoculation theory. In *The international encyclopedia of media psychology*. John Wiley & Sons.
142. Compton, J., et al. (2021). Inoculation theory as a strategic tool. *ResearchGate*.
143. Banas, J. A., & Rains, S. A. (2010). A meta-analysis of research on inoculation theory. *Communication Monographs*, *77*(3), 281-311.
144. Ivanov, B., et al. (2021). Affect and the process of resistance: A state-of-the-art review of inoculation theory. *International Journal of Communication*, *15*, 23.
145. van der Linden, S., et al. (2017). Inoculating the public against misinformation about climate change. *Global Challenges*, *1*(2), 1600008.
146. Farkona, S., Diamandis, E. P., & Blasutig, I. M. (2016). Cancer immunotherapy: the beginning of the end of cancer? *BMC Medicine*, *14*(1), 73.
147. Pardoll, D. M. (2012). The blockade of immune checkpoints in cancer

immunotherapy. *Nature Reviews Cancer*, *12*(4), 252-264.
148. Gibney, G. T., Weiner, L. M., & Atkins, M. B. (2016). Predictive biomarkers for checkpoint inhibitor-based immunotherapy. *The Lancet Oncology*, *17*(12), e542-e551.
149. Havel, J. J., Chowell, D., & Chan, T. A. (2019). The evolving landscape of biomarkers for checkpoint inhibitor immunotherapy. *Nature Reviews Cancer*, *19*(3), 133-150.
150. Chen, P. L., & Roh, W. (2017). Dynamic versus static biomarkers in cancer immune checkpoint blockade: unravelling complexity. *OncoImmunology*, *6*(1), e1254333.
151. Smajdor, A., & Villalba, A. (2023). The Ethics of Cellular Reprogramming. *Cell Reprogramming*, *25*(5), 190-194.
152. MacKay, K. (2023). What are the ethical and moral quandaries of synthetic human embryos? *University of Sydney News*.
153. Nuffield Council on Bioethics. (2016). *Genome editing: an ethical review*.
154. Douglas, T., & Savulescu, J. (2024). The Ethics of Synthetic DNA. *Journal of Medical Ethics*, *50*(1), 1-8.

Referências citadas

1. Network Medicine: A Network-based Approach to Human Disease ..., acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC3140052/
2. Epidemic model for information diffusion in web forums: experiments ..., acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC4723377/
3. Social and biological contagions - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC4229037/
4. SEIR-based model for the information spreading over SNS - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/289288868_SEIR-based_model_for_the_information_spreading_over_SNS
5. Understanding and countering the spread of conspiracy theories in ..., acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC8360523/
6. The coupled dynamics of information dissemination and SEIR-based epidemic spreading in multiplex networks - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC8559433/
7. Mathematical Models of Tumor Cell Proliferation: A Review of the Literature - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC6295418/
8. Mathematical modeling of tumor growth and metastatic spreading: validation in tumor-bearing mice - PubMed, acessado em junho 27, 2025, https://pubmed.ncbi.nlm.nih.gov/25217520/
9. A Mathematical Framework for Modelling the Metastatic Spread of Cancer - PubMed Central, acessado em junho 27, 2025,

https://pmc.ncbi.nlm.nih.gov/articles/PMC6503893/
10. [2403.07881] Epidemic modelling requires knowledge of the social network - arXiv, acessado em junho 27, 2025, https://arxiv.org/abs/2403.07881
11. A tutorial review of mathematical techniques for quantifying tumor heterogeneity, acessado em junho 27, 2025, https://www.aimspress.com/article/id/5115
12. (PDF) Quantification of tumor heterogeneity: from data acquisition to metric generation, acessado em junho 27, 2025, https://www.researchgate.net/publication/357391023_Quantification_of_tumor_heterogeneity_from_data_acquisition_to_metric_generation
13. Integrated analysis of single-cell RNA-seq and spatial transcriptomics to identify the lactylation-related protein TUBB2A as a potential biomarker for glioblastoma in cancer cells by machine learning - Frontiers, acessado em junho 27, 2025, https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2025.1601533/full
14. Quantifying Intratumoral Heterogeneity and Immunoarchitecture Generated In-Silico by a Spatial Quantitative Systems Pharmacology Model - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC10216176/
15. Spatial colocalization and combined survival benefit of natural killer and CD8 T cells despite profound MHC class I loss in non-small cell lung cancer, acessado em junho 27, 2025, https://jitc.bmj.com/content/12/9/e009126
16. Multiscale Agent-Based and Hybrid Modeling of the Tumor Immune Microenvironment - PMC - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC6349239/
17. Social network theory in the behavioural sciences: potential applications - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC7079911/
18. A Bayesian model of information cascades - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/351449470_A_Bayesian_model_of_information_cascades
19. Bayesian Learning in Social Networks - NYU Stern, acessado em junho 27, 2025, https://pages.stern.nyu.edu/~ilobel/bayesian-learning-social-networks.pdf
20. Bayesian Learning in Social Networks | MIT Economics, acessado em junho 27, 2025, https://economics.mit.edu/sites/default/files/publications/Bayesian%20Learning%20in%20Social%20Networks.pdf
21. Information Cascades and Social Learning - National Bureau of Economic Research, acessado em junho 27, 2025, https://www.nber.org/system/files/working_papers/w28887/w28887.pdf
22. A Bayesian Model of Information Cascades | Request PDF - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/364582972_A_Bayesian_Model_of_Information_Cascades
23. Cascades Across Networks Are Sufficient for the Formation of Echo Chambers:

An Agent-Based Model, acessado em junho 27, 2025, https://www.jasss.org/24/3/1.html
24. Mathematical Modeling of Tumor-Immune Cell Interactions - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC6579737/
25. The Immune System and Responses to Cancer: Coordinated Evolution - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC7735224/
26. Agent-Based Modeling of Virtual Tumors Reveals the Critical Influence of Microenvironmental Complexity on Immunotherapy Efficacy, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC11394213/
27. Cancer-immune coevolution dictated by antigenic mutation accumulation - eLife, acessado em junho 27, 2025, https://elifesciences.org/reviewed-preprints/103970
28. Political Communication, Computational Propaganda, and Autonomous Agents - Introduction, acessado em junho 27, 2025, https://ijoc.org/index.php/ijoc/article/download/6298/1809
29. Political Communication, Computational Propaganda, and Autonomous Agents - Introduction - NSF Public Access Repository, acessado em junho 27, 2025, https://par.nsf.gov/servlets/purl/10021331
30. Bots and Computational Propaganda: Automation for Communication and Control (Chapter 5) - Social Media and Democracy - Cambridge University Press, acessado em junho 27, 2025, https://www.cambridge.org/core/books/social-media-and-democracy/bots-and-computational-propaganda-automation-for-communication-and-control/A15EE25C278B442EF00199AA660BFADD
31. GameSec 2025 - Conference on Game Theory and AI for Security, acessado em junho 27, 2025, https://www.gamesec-conf.org/
32. Adversarial Machine Learning in Real-World Computer Vision Systems and Online Challenges (AML-CV) | Workshop at CVPR 2021, acessado em junho 27, 2025, https://advml-workshop.github.io/aaai2022/
33. Functionality-Preserving Adversarial Machine Learning for Robust Classification in Cybersecurity and Intrusion Detection Domains: A Survey - MDPI, acessado em junho 27, 2025, https://www.mdpi.com/2624-800X/2/1/10
34. Adversarial machine learning for protecting against online manipulation - arXiv, acessado em junho 27, 2025, https://arxiv.org/pdf/2111.12034
35. Renée DiResta: "Computational Propaganda" - The Yale Review, acessado em junho 27, 2025, https://yalereview.org/article/computational-propaganda
36. Adversarial Style Augmentation via Large Language Model for Robust Fake News Detection, acessado em junho 27, 2025, https://openreview.net/forum?id=c9f8LmRgnD&referrer=%5Bthe%20profile%20of%20Sungwon%20Park%5D(%2Fprofile%3Fid%3D~Sungwon_Park1)
37. The contribution of evolutionary game theory to understanding and treating cancer, acessado em junho 27, 2025, https://www.researchgate.net/publication/346931043_The_contribution_of_evolutionary_game_theory_to_understanding_and_treating_cancer
38. Optimal adaptive cancer therapy based on evolutionary game theory | PLOS One, acessado em junho 27, 2025,

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0320677
39. Gaming the cancer-immunity cycle by synchronizing the dose schedules - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/385515666_Gaming_the_cancer-immunity_cycle_by_synchronizing_the_dose_schedules
40. Containing misinformation: Modeling spatial games of fake news - PMC - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC10924450/
41. Using Agent-Based Modelling and Reinforcement Learning to Study Hybrid Threats, acessado em junho 27, 2025, https://www.jasss.org/28/1/1.html
42. Multi-agent Reinforcement Learning in Sequential Social Dilemmas - Papers With Code, acessado em junho 27, 2025, https://paperswithcode.com/paper/multi-agent-reinforcement-learning-in/review/
43. [1702.03037] Multi-agent Reinforcement Learning in Sequential Social Dilemmas - arXiv, acessado em junho 27, 2025, https://arxiv.org/abs/1702.03037
44. Multi-Agent Reinforcement Learning: A Review of Challenges and Applications - MDPI, acessado em junho 27, 2025, https://www.mdpi.com/2076-3417/11/11/4948
45. Selectively Sharing Experiences Improves Multi-Agent Reinforcement Learning - NIPS, acessado em junho 27, 2025, https://proceedings.neurips.cc/paper_files/paper/2023/file/baa7fc022f35b6ea7b8b2a2fe60babe0-Paper-Conference.pdf
46. Self-Tolerance - GenScript, acessado em junho 27, 2025, https://www.genscript.com/self-tolerance.html
47. 6. Tolerance and Autoimmunity - Immunopaedia, acessado em junho 27, 2025, https://www.immunopaedia.org.za/immunology/special-focus-area/6-tolerance-and-autoimmunity/
48. Immune tolerance and the prevention of autoimmune ... - Frontiers, acessado em junho 27, 2025, https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2024.1339714/full
49. Understanding the Dynamics of T-Cell Activation in Health and ..., acessado em junho 27, 2025, https://pubmed.ncbi.nlm.nih.gov/30689404/
50. miRNA/mRNA analysis of increased TGF-β pathways drive epithelial-mesenchymal transition and regulatory T cell differentiation | bioRxiv, acessado em junho 27, 2025, https://www.biorxiv.org/content/10.1101/2025.06.23.661210v1
51. Mathematical Modeling of the Regulatory T Cell Effects on Renal Cell Carcinoma Treatment - Scholarship @ Claremont, acessado em junho 27, 2025, https://scholarship.claremont.edu/cgi/viewcontent.cgi?article=1438&context=hmc_fac_pub
52. Regulatory T Cells Suppress Effector T Cell Proliferation by Limiting Division Destiny - Frontiers, acessado em junho 27, 2025, https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2018.02461/full

53. Regulatory T cells: immune suppression and beyond - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC2868372/
54. Regulatory T Cells: Regulation of Identity and Function - Frontiers, acessado em junho 27, 2025, https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2021.750542/full
55. Theoretical modeling reveals that regulatory T cells increase T-cell interaction with antigen-presenting cells for stable immune tolerance - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC6794947/
56. Social immunity: why we should study its nature, evolution and functions across all social systems - PubMed, acessado em junho 27, 2025, https://pubmed.ncbi.nlm.nih.gov/30551759/
57. Social immunity: Why we should study its nature, evolution and functions across all social systems - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/323690105_Social_immunity_Why_we_should_study_its_nature_evolution_and_functions_across_all_social_systems
58. Towards a conceptual framework for innate immunity - arXiv, acessado em junho 27, 2025, https://arxiv.org/pdf/1006.1568
59. Recursive patterns in online echo chambers - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC6934735/
60. The echo chamber effect on social media - PNAS, acessado em junho 27, 2025, https://www.pnas.org/doi/10.1073/pnas.2023301118
61. Emergence of echo chambers in social media communication involving multiple topics, acessado em junho 27, 2025, https://www.researchgate.net/publication/388426487_Emergence_of_echo_chambers_in_social_media_communication_involving_multiple_topics
62. Network polarization, filter bubbles, and echo chambers: an annotated review of measures and reduction methods - IC-Unicamp, acessado em junho 27, 2025, https://ic.unicamp.br/~ruben/publications/2023%20-%20Network%20polarization%20filter%20bubbles%20and%20echo%20chambers%20An%20annotated%20review.pdf
63. Large networks of rational agents form persistent echo chambers - PMC - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC6098126/
64. Modeling Echo Chambers and Polarization Dynamics in Social Networks | Phys. Rev. Lett., acessado em junho 27, 2025, https://link.aps.org/doi/10.1103/PhysRevLett.124.048301
65. Using network control theory to study the dynamics of the structural connectome - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC10473719/
66. Control Theory and Cancer Chemotherapy: How They Interact - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC7841331/
67. (PDF) Decoding the principle of cell-fate determination for its reverse control - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/380392884_Decoding_the_principle_of

_cell-fate_determination_for_its_reverse_control
68. (PDF) Steering cell-state and phenotype transitions by causal disentanglement learning, acessado em junho 27, 2025, https://www.researchgate.net/publication/383206974_Steering_cell-state_and_phenotype_transitions_by_causal_disentanglement_learning
69. Stem cell fate in cancer growth, progression and therapy resistance - PubMed, acessado em junho 27, 2025, https://pubmed.ncbi.nlm.nih.gov/30228301/
70. Cell Fate Reprogramming by Control of Intracellular Network Dynamics - PLOS, acessado em junho 27, 2025, https://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1004193
71. Social Influence Network Theory - Cambridge University Press & Assessment, acessado em junho 27, 2025, https://www.cambridge.org/core/books/social-influence-network-theory/2602561B361D1ABF985E712B14392D2C
72. Social Influence Networks and Opinion Change, acessado em junho 27, 2025, https://www2.cs.siu.edu/~hexmoor/classes/CS539-F10/Friedkin.pdf
73. Measuring Social Influence with Networked Synthetic Control - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/391910980_Measuring_Social_Influence_with_Networked_Synthetic_Control
74. Network models for social influence processes, acessado em junho 27, 2025, http://explorer.bee.oregonstate.edu/Topic/InfluenceNetworks/Documents/Robins2001_Article_NetworkModelsForSocialInfluenc.pdf
75. A natural experiment of social network formation and dynamics - PNAS, acessado em junho 27, 2025, https://www.pnas.org/doi/10.1073/pnas.1404770112
76. (PDF) Steering opinion dynamics through control of social networks - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/381884016_Steering_opinion_dynamics_through_control_of_social_networks
77. Steering opinion dynamics through control of social networks - YouTube, acessado em junho 27, 2025, https://www.youtube.com/watch?v=xlhyv3IBOZY
78. [2404.09849] Steering opinion dynamics through control of social networks - arXiv, acessado em junho 27, 2025, https://arxiv.org/abs/2404.09849
79. Causal Discovery in Gene Regulatory Networks with GFlowNet: Towards Scalability in Large Systems - OpenReview, acessado em junho 27, 2025, https://openreview.net/pdf?id=jbamDZ374t
80. CausalCCC: a web server to explore intracellular causal pathways enabling cell–cell communication | Nucleic Acids Research | Oxford Academic, acessado em junho 27, 2025, https://academic.oup.com/nar/advance-article/doi/10.1093/nar/gkaf404/8131115
81. Inoculation Theory: Quick Look - DTIC, acessado em junho 27, 2025, https://apps.dtic.mil/sti/trecms/pdf/AD1118511.pdf
82. Protecting Against Disinformation: Using Inoculation to Cultivate Reactance Towards Astroturf Attack - Courtney Boman - The University of Alabama, acessado em junho 27, 2025,

https://cboman.people.ua.edu/uploads/1/3/7/2/137203799/boman__2023_.pdf
83. Neutralizing misinformation through inoculation: Exposing misleading argumentation techniques reduces their influence - PMC, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC5419564/
84. A Meta-Analysis of Research on Inoculation Theory - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/233050264_A_Meta-Analysis_of_Research_on_Inoculation_Theory
85. Inoculation Theory and Affect - International Journal of Communication, acessado em junho 27, 2025, https://ijoc.org/index.php/ijoc/article/download/19094/3828
86. Psychological inoculation can reduce susceptibility to misinformation in large rational agent networks - PubMed Central, acessado em junho 27, 2025, https://pmc.ncbi.nlm.nih.gov/articles/PMC9363981/
87. Advancing Adjuvant Immunotherapy in Hepatocellular Carcinoma: A Comprehensive Review - Dove Medical Press, acessado em junho 27, 2025, https://www.dovepress.com/advancing-adjuvant-immunotherapy-in-hepatocellular-carcinoma-a-compreh-peer-reviewed-fulltext-article-ITT
88. Biomarkers and computational models for predicting efficacy to tumor ICI immunotherapy, acessado em junho 27, 2025, https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2024.1368749/full
89. Towards the Prediction of Responses to Cancer Immunotherapy: A Multi-Omics Review, acessado em junho 27, 2025, https://www.mdpi.com/2075-1729/15/2/283
90. Immuno-Oncology at the Crossroads: Confronting Challenges in the Quest for Effective Cancer Therapies - MDPI, acessado em junho 27, 2025, https://www.mdpi.com/1422-0067/26/13/6177
91. (PDF) Dynamic versus static biomarkers in cancer immune checkpoint blockade: Unravelling complexity - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/312097194_Dynamic_versus_static_biomarkers_in_cancer_immune_checkpoint_blockade_Unravelling_complexity
92. (PDF) Stem Cell Engineering: Potential and Ethical Considerations - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/388316404_Stem_Cell_Engineering_Potential_and_Ethical_Considerations
93. The Ethics of Cellular Reprogramming | Request PDF - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/374119128_The_Ethics_of_Cellular_Reprogramming
94. The Ethics of Cellular Reprogramming - PubMed, acessado em junho 27, 2025, https://pubmed.ncbi.nlm.nih.gov/37738321/
95. Computational propaganda and misinformation: AI technologies as tools of media manipulation - ResearchGate, acessado em junho 27, 2025, https://www.researchgate.net/publication/388029509_Computational_propaganda_and_misinformation_AI_technologies_as_tools_of_media_manipulation