

Strategic decision-making under uncertainty: A game-theoretic information analysis of poker and cancer systems

Introduction

Both poker players and cancer cells face analogous strategic challenges: maki optimal decisions under incomplete information while adapting to opposing for seek to exploit their vulnerabilities. In poker, "under the gun" (UTG) players mu choose opening strategies against unknown opponent types while concealing intentions. Similarly, cancer cells navigate treatment pressures by developing resistance through genetic mutations, epigenetic switching, and immune evasion Cancer resistance emerges via three mechanisms, selection of pre-existing va induced adaptations, and de novo mutations, creating evolutionary arms races comparable to the adaptation cycles between poker players and their opponen systems exhibit fundamental information-processing parallels: incomplete environmental sensing, strategic concealment of true states, temporal vulnerat exploitation, and continuous adaptation based on outcome feedback. We hypo that poker strategy optimization and cancer treatment resistance represent inst a universal computational algorithm for decision-making under uncertainty, when success requires balancing exploration of new strategies against exploitation approaches while managing information asymmetry and deception capabilities framework suggests that game-theoretic principles governing poker success, opponent modeling, mixed strategies, and adaptive protocols, may inform nove treatment designs that exploit evolutionary constraints and information-theoret vulnerabilities in tumor dynamics.





Methods

We developed a parallel computational framework comparing poker strategy a cancer treatment as information-processing games using Python with NumPy, and NetworkX libraries. The poker component simulates Texas Hold'em pre-fle scenarios where the UTG player employs four strategies (Standard Raise, Lim Over-Raise, Mixed Strategy) against up to nine opponent archetypes character fold/raise thresholds, adaptation rates, and risk tolerance parameters. The car component models heterogeneous tumor populations with up to nine cell types distributed across a 10×10 spatial grid with environmental gradients. Each cell defined by 13 parameters such as treatment resistance, growth rate, immune and epigenetic plasticity. Four treatment strategies (Standard Dose, Low-Dose Metronomic, High-Dose Pulse, Adaptive Protocol) are implemented with distin efficacy profiles, spatial penetration factors, and immune stimulation effects. T framework incorporates a comprehensive immune system with four cell types, intercellular signaling networks modeling ten major pathways, and evolutionary dynamics enabling mutation events and resistance development. Both system strategies based on outcome feedback using reinforcement learning mechanis Information-theoretic analysis quantifies system behaviors through Shannon er transfer entropy, mutual information, and Fisher information. Each simulation re rounds with systematic parameter sweeps across complexity levels (2-9 player types), generating comprehensive datasets of population dynamics, strategy e information metrics, and cross-domain correlation patterns with consistent ran seeding (seed=42) ensuring reproducibility.

1. System Initialization		3. Information-Theoretic Analysis	
Poker System	Cancer System	Core Information Metrics	Derived Sys
9 Player Archetypes Parameters: $f_t \in [0,1]$, $r_t \in [0,1]$, $\alpha \in [0,1]$, $\rho \in [0,1]$ Tight-Aggressive, Loose-Passive, Balanced, Maniac, etc.	9 Cell Archetypes $R \in [0,1], g \in [0,1], \alpha_evasion \in [0,1], \mu > 0$ Proliferative, Stem-like, Drug-Resistant, Immune-Evasive, etc.	Shannon Entropy (System Diversity) $H(X) = -\Sigma_i p(x_i) \log_2 p(x_i)$ $p(x_i) \in [0, 1], \Sigma p(x_i)=1.$ Measures strategy/population unpredictability	Information Asymmetry <i>I_asym</i> = <i>H_opponent</i> - <i>H_decision</i> Knowledge advantage between i
UTG Strategy Portfolio $w_i \in [0,1], \Sigma w_i = 1 (strategy weights)$ Standard Raise, Limping, Over-Raise, Mixed Strategy	Spatial Grid (10×10) $O(i,j) = 0.2 + 0.8 \cdot d(i,j)/d_max (oxygen gradient)$ $N(i,j) = 0.5 + 0.5 \cdot d(i,j)/d_max (nutrients)$	Transfer Entropy (Causal Flow) $TE(X \rightarrow Y) = \sum p(y \ t+1, y \ t, x \ t) \ log \ 2 \ [p(y \ t+1 y \ t, x \ t)/p(y \ t+1 y \ t)]$ Sophisticated binning for discrete probability estimation	Ecosystem Resilience $R_eco = 0.5 \cdot H(populations) + 0.5$ Combines diversity with intercell
Initial Conditions Stack sizes = 1000 chips, uniform strategy weights = 0.25	Immune SystemSpatial infiltration: $I(i,j) \in [0,1]$ T-cells, NK cells, Macrophages, Dendritic cells	Mutual Information (State Correlation) $I(X; Y) = H(X) + H(Y) - H(X, Y)$ $H(X, Y)$ =joint entropy. Information overlap between time steps	
	Signaling Networks NetworkX directed graph with intercellular communication 10 intracellular pathways: MAPK, PI3K/AKT, JAK/STAT, Wnt, etc.	Fisher Information (Precision Windows) $FI = 1/(Var(pop) + 0.1)$ Vulnerability windows for optimal intervention timing	
2. Simulation Dy Poker Evolution Pipeline	vnamics (50 Rounds) Cancer Evolution Pipeline		
2. Simulation Dy Poker Evolution Pipeline Step 1: Strategy Selection S. UTG = armar, i/w i < s. i) s ~ U(0.1)	ynamics (50 Rounds) Cancer Evolution Pipeline Step 1: Treatment Application E local(ii) = E base (l = r. c : R ii) : D(ii) : h. c : l(ii)	Cancer Cell Signating Network	Evolutionary Linea
Poker Evolution Pipeline Step 1: Strategy Selection $S_UTG = argmax_i(w_i \cdot \varepsilon_i), \varepsilon \sim U(0,1)$ Probabilistic selection with exploration noise Step 2: Opponent Response effective_strength = hand_strength + position_value	ynamics (50 Rounds) Cancer Evolution Pipeline Step 1: Treatment Application $E_local(i,j) = E_base \cdot (1 - r_c \cdot R_i) \cdot D(i,j) \cdot h_c \cdot I(i,j)$ Spatial treatment efficacy with resistance modulation Step 2: Cell Response Decision $P_death(i,j) = E_local(i,j) + I_immune(i,j) \cdot (1 - a_evasion) \cdot 0.3$	Cancer Cat Signaling Network	Evolutionary Lineag Mesench@nal_mm Mesench@nal_mm1 Mesench@nal_mm1
2. Simulation Dy Poker Evolution Pipeline Step 1: Strategy Selection $S_UTG = argmax_i(w_i \cdot \varepsilon_i), \varepsilon \sim U(0,1)$ Probabilistic selection with exploration noise Step 2: Opponent Response effective_strength = hand_strength + position_value Fold/call/raise based on archetype thresholds Step 3: Outcome Determination Winner = UTG_Player (if all fold) else argmax(showdown_strengths) Pot distribution and stack updates	ynamics (50 Rounds) Cancer Evolution Pipeline Step 1: Treatment Application $E_local(i,j) = E_base \cdot (1 - r_c \cdot R_j) \cdot D(i,j) \cdot h_c \cdot I(i,j)$ Spatial treatment efficacy with resistance modulation Step 2: Cell Response Decision $P_death(i,j) = E_local(i,j) + I_immune(i,j) \cdot (1 - a_evasion) \cdot 0.3$ Combined treatment + immune-mediated killing Step 3: Resistance Evolution (3 Mechanisms) $dr_i, t = E_tr_i, t \cdot 0.2 + E_t(1 - r_i, t) \cdot a_ip_i + E_t(1 - r_i, t) \cdot µ_i \cdot 0.1$ Selection + Adaptation + Mutation operating in parallel	Cacar Cal Signaling Batewart	Evolutionary Lineag Mesench al muta Protifeative Stem laganut2
2. Simulation Dy Poker Evolution Pipeline Step 1: Strategy Selection $S_UTG = argmax_i(w_i \cdot \varepsilon_i), \varepsilon \sim U(0,1)$ Probabilistic selection with exploration noise Step 2: Opponent Response effective_strength = hand_strength + position_value Fold/call/raise based on archetype thresholds Step 3: Outcome Determination Winner = UTG_Player (if all fold) else $argmax(showdown_strengths)$ Pot distribution and stack updates Step 4: Strategy Adaptation $w_new = \{min(0.7, 1.1w) \ if success; max(0.1, 0.9w) \ if failure\}$ Reinforcement learning with bounded weights	ynamics (50 Rounds) Cancer Evolution Pipeline Step 1: Treatment Application $E_local(i,j) = E_base \cdot (1 - r_c \cdot R_i) \cdot D(i,j) \cdot h_c \cdot I(i,j)$ Spatial treatment efficacy with resistance modulation Step 2: Cell Response Decision $P_death(i,j) = E_local(i,j) + I_immune(i,j) \cdot (1 - a_evasion) \cdot 0.3$ Combined treatment + immune-mediated killing Step 3: Resistance Evolution (3 Mechanisms) $dr_i,t = E_tr_i,t\cdot0.2 + E_t^{-}(1 - r_i,t) \cdot a_i \cdot p_i + E_t^{-}(1 - r_i,t) \cdot \mu_i \cdot 0.1$ Selection + Ādaptation + Mutation operating in parallel Step 4: Spatial Population Dynamics $P_growth(i,j) = g_rate \cdot n_efficiency \cdot N(i,j)$ Growth into adjacent empty spaces via get_empty_neighbors()		Evolutionary Lines Mesench end mut2 Mesench end mut2 Prolifestive Stem line_mut2ste Quidgent

Christian Gensbigler, Randy Liu

Department of Mathematics, Dartmouth College

	Results	
king	Stack Size and Cell Population Results	
rces that ust	Poker Stack Size Results	
their own	 OTG Player exhibits greatest degree of dominance General convergence of strategies toward 0 or 1000-1 	500
ion	indicates equilibrium establishment	
ariants,	Cancer Cell Population Totals	
S	 Cell populations remain more stable than poker strategy 	gies
nts. Both	 Mutant variants survive and persist 	
bility		
othesize stances of		
nere	Information-Theoretic Analysis	
of proven	 Discontinuous jumps indicate discrete evolutionary 	
including	events	3.5 -
vel cancer	 Plateaus suggest stable population states 	3.0 -
tic	Mutual Information	2.5-
	Late stage increase suggests emergence of strong	2.0
		1.0 -
	Transfer Entropy	ò
	 Consistently near zero values throughout simulation indicate absence of directed information flow from 	04 -
	treatment to population changes	00 -
	Fisher Information	02 -
	 Extreme spikes present at certain cycles 	04 -
and	Seem to be decoupled form major evolutionary	0
, SCIPy, op	u ansiuons	
nping,		
erized by	System Complexity	
S	Decision Maker Success	
Il type is	UTG win rate decreases monotonically	
evasion, e	Cancer treatment success peaks at higher complexity	
nct The	0.35 - 월	
ne	 Information Diversity Poker strategy diversity decreases with complexity 	
У У	while cancer Shannon entropy increases linearly	
is adapt sms.	Adaptation	2 3
entropy,	Cancer resistance levels increase steadily with	
runs 50 ars/cell	complexity while poker shows volatile performance	
evolution,		
dom	Resilience	2 3
	 Scales linearly with increasing cell type diversity 	
m Metrics		
ker acting agents	Informational Acumanation	
low signaling strength	Decision Maker	
	 Cancer treatment possesses superior 	
	environmental knowledge than UTG poker player	el (0-1)
	Opponent	Information Lev
ncer Cell Types	Poker players reveal more information than cancer colls concool from treatment	
Angioneric Immune & sive mut3 Immune Expassive Immune Expassive mut1		
Gly@ytic Stem{@_mut1		
⊷osers.ngrai_mut2 Hypose_mut1		



Discussion

Poker and Cancer Systems Behave Differently Under Complexity

sweet spots

Treatments Act Indirectly

- conventional resistance models
- between players

Temporal Vulnerability Windows

become temporarily predictable vulnerability windows

Clinical Applications

Paradigm Shift

• Framework represents shift from reactive resistance treatment toward proactive strategic intervention • Conceptualizes treatments as fitness landscape architects guiding tumor evolution along therapeutically favorable trajectories

Conclusion

This project demonstrates that information-theoretic analysis of poker strategy and cancer treatment resistance reveals fundamental differences in how adaptive systems optimize under uncertainty, rather than direct parallels. While both systems process incomplete information and adapt continuously under environmental pressure, their distinct implementations illuminate domain-specific constraints that drive different strategies. The critical finding that treatments modify tumor fitness landscapes indirectly rather than through direct selection pressure, contrasting with poker's immediate strategy coupling, fundamentally reframes cancer therapy from reactive resistance management to proactive evolutionary guidance. These information-theoretic insights enable translating poker principles to oncology: exploiting temporal Fisher information vulnerability windows for precision timing, maintaining optimal tumor complexity within therapeutic sweet spots, and employing game-theoretic mixed strategies that remain unpredictable to evolutionary processes, transforming cancer treatment from static protocol-driven approaches to dynamic strategic intervention systems guided by cross-domain optimization principles.

Future Directions

Develop sequential combination treatments that mimic poker's strategic deception use initial "bluff" therapies (low-dose or targeted agents) that appear weak to cancer cells but deliberately reshape tumor fitness landscapes by forcing evolutionary pressure toward specific phenotypes, then exploit the resulting vulnerabilities with precisely-timed second-line "value bet" interventions (high-efficacy combinations) during Fisher information spikes when tumors become predictable. This approach maintains optimal tumor heterogeneity during the "bluff" phase, keeping diversity within therapeutic sweet spots, then capitalizes on temporary evolutionary constraints when cancer cells commit to suboptimal strategies.

References

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Acknowledgement: We would like to acknowledge the support and guidance provided by Professor Feng Fu. His feedback and advice was integral to our successful completion of this project.



Both systems operate with moderate information asymmetry (0.5-0.7 levels) • Poker: UTG win rates decline from 45% to 14% as opponents increase Cancer: Treatment efficacy peaks at higher heterogeneity, suggesting therapeutic

• Near-zero transfer entropy from treatments to population changes challenges

• Treatments operate through fitness landscape modification rather than direct selective pressure, which contrasts sharply with poker's immediate strategy coupling

• Fisher information spikes at cycles 80-90 reveal discrete periods when tumors

• Unlike poker's continuous information advantages, cancer shows discrete

Static treatment protocols fundamentally misalign with tumor evolution dynamics

 Monitor tumor states to exploit Fisher information vulnerability windows Maintain heterogeneity within therapeutic sweet spots through adaptive therapy Employ sequential treatments that exploit fitness landscape modifications Incorporate mixed strategies that remain unpredictable to evolutionary processes

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