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

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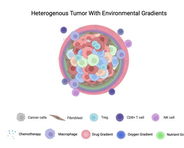
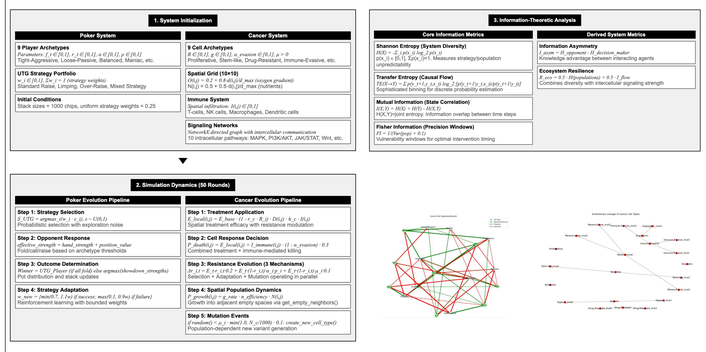
Introduction

Both poker players and cancer cells face analogous strategic challenges: making optimal decisions under incomplete information while adapting to opposing forces that seek to exploit their vulnerabilities. In poker, “under the gun” (UTG) players must choose opening strategies against unknown opponent types while concealing their own 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 variants, induced adaptations, and de novo mutations, creating evolutionary arms races comparable to the adaptation cycles between poker players and their opponents. Both systems exhibit fundamental information-processing parallels: incomplete environmental sensing, strategic concealment of true states, temporal vulnerability exploitation, and continuous adaptation based on outcome feedback. We hypothesize that poker strategy optimization and cancer treatment resistance represent instances of a universal computational algorithm for decision-making under uncertainty, where success requires balancing exploration of new strategies against exploitation of proven approaches while managing information asymmetry and deception capabilities. This framework suggests that game-theoretic principles governing poker success, including opponent modeling, mixed strategies, and adaptive protocols, may inform novel cancer treatment designs that exploit evolutionary constraints and information-theoretic vulnerabilities in tumor dynamics.



Methods

We developed a parallel computational framework comparing poker strategy and cancer treatment as information-processing games using Python with NumPy, SciPy, and NetworkX libraries. The poker component simulates Texas Hold'em pre-flop scenarios where the UTG player employs four strategies (Standard Raise, Limping, Over-Raise, Mixed Strategy) against up to nine opponent archetypes characterized by fold/raise thresholds, adaptation rates, and risk tolerance parameters. The cancer component models heterogeneous tumor populations with up to nine cell types distributed across a 10×10 spatial grid with environmental gradients. Each cell type is defined by 13 parameters such as treatment resistance, growth rate, immune evasion, and epigenetic plasticity. Four treatment strategies (Standard Dose, Low-Dose Metronomic, High-Dose Pulse, Adaptive Protocol) are implemented with distinct efficacy profiles, spatial penetration factors, and immune stimulation effects. The 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 systems adapt strategies based on outcome feedback using reinforcement learning mechanisms. Information-theoretic analysis quantifies system behaviors through Shannon entropy, transfer entropy, mutual information, and Fisher information. Each simulation runs 50 rounds with systematic parameter sweeps across complexity levels (2-9 players/cell types), generating comprehensive datasets of population dynamics, strategy evolution, information metrics, and cross-domain correlation patterns with consistent random seeding (seed=42) ensuring reproducibility.

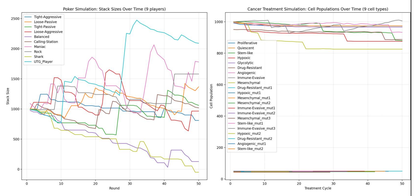


Results

Stack Size and Cell Population Results

Poker Stack Size Results
- UTG Player exhibits greatest degree of dominance
- General convergence of strategies toward 0 or 1000-1500 indicates equilibrium establishment

Cancer Cell Population Totals
- Cell populations remain more stable than poker strategies
- Mutant variants survive and persist



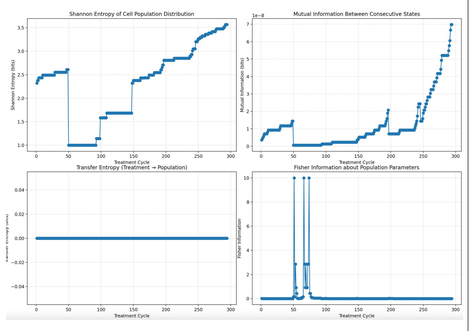
Information-Theoretic Analysis

Shannon Entropy
- Discontinuous jumps indicate discrete evolutionary events
- Plateaus suggest stable population states

Mutual Information
- Late stage increase suggests emergence of strong state-to-state correlations

Transfer Entropy
- Consistently near zero values throughout simulation indicate absence of directed information flow from treatment to population changes

Fisher Information
- Extreme spikes present at certain cycles
- Seem to be decoupled form major evolutionary transitions



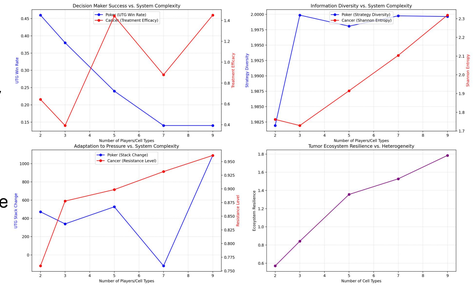
System Complexity

Decision Maker Success
- UTG win rate decreases monotonically
- Cancer treatment success peaks at higher complexity

Information Diversity
- Poker strategy diversity decreases with complexity while cancer Shannon entropy increases linearly

Adaptation
- Cancer resistance levels increase steadily with complexity while poker shows volatile performance changes

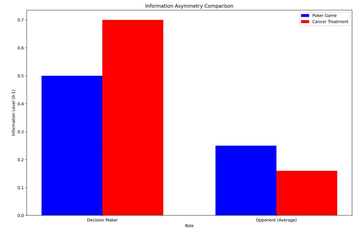
Resilience
- Scales linearly with increasing cell type diversity



Informational Asymmetry

Decision Maker
- Cancer treatment possesses superior environmental knowledge than UTG poker player

Opponent
- Poker players reveal more information than cancer cells conceal from treatment



Discussion

Poker and Cancer Systems Behave Differently Under Complexity

- 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 sweet spots

Treatments Act Indirectly

- Near-zero transfer entropy from treatments to population changes challenges conventional resistance models
- Treatments operate through fitness landscape modification rather than direct selective pressure, which contrasts sharply with poker's immediate strategy coupling between players

Temporal Vulnerability Windows

- Fisher information spikes at cycles 80-90 reveal discrete periods when tumors become temporarily predictable
- Unlike poker's continuous information advantages, cancer shows discrete vulnerability windows
- Static treatment protocols fundamentally misalign with tumor evolution dynamics

Clinical Applications

- 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

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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