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Memory-dependent coevolution: A non-Markovian paradigm for behavior-disease dynamics

Fang Wang,[1,2,3,4,*] Kexin Wei,[1] Zehui Zhang,[1] and You-Gan Wang[1,5]

[1]School of Mathematics and Computational Science, Xiangtan University, Xiangtan 411105, China

[2]National Center for Applied Mathematics in Hunan, Xiangtan 411105, China

[3]Key Laboratory of Intelligent Computing and Information Processing of Ministry of Education, Xiangtan 411105, China

[4]Hunan Research Center of the Basic Discipline Fundamental Algorithmic Theory and Novel Computational Methods, Xiangtan 411105, China

[5]School of Statistics and Data Science, Guangdong University of Finance & Economic, Guangzhou 510320, China

*Correspondence: fwang4@xtu.edu.cn

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The COVID-19 pandemic made clear that infectious-disease outbreaks are not purely biological processes but complex socio-technical phenomena shaped by continuous feedback between epidemiological dynamics and human behavior.[1] Changes in incidence rapidly triggered adaptive responses such as distancing, masking, and vaccination, which in turn reshaped transmission patterns and future risk perception. This recurrent feedback loop underscores a fundamental insight: disease spread and behavioral adaptation evolve jointly rather than sequentially. Understanding epidemics requires models where both behavioral decision-making and contagion dynamics are treated as coevolving, interconnected processes, rather than isolated components.[2] Importantly, while the coupling between behavioral adaptation and epidemic spreading has been widely recognized, the central argument of this editorial is that such coupling must be reformulated as a history-dependent, non-Markovian process, evolving on a shared temporal framework.

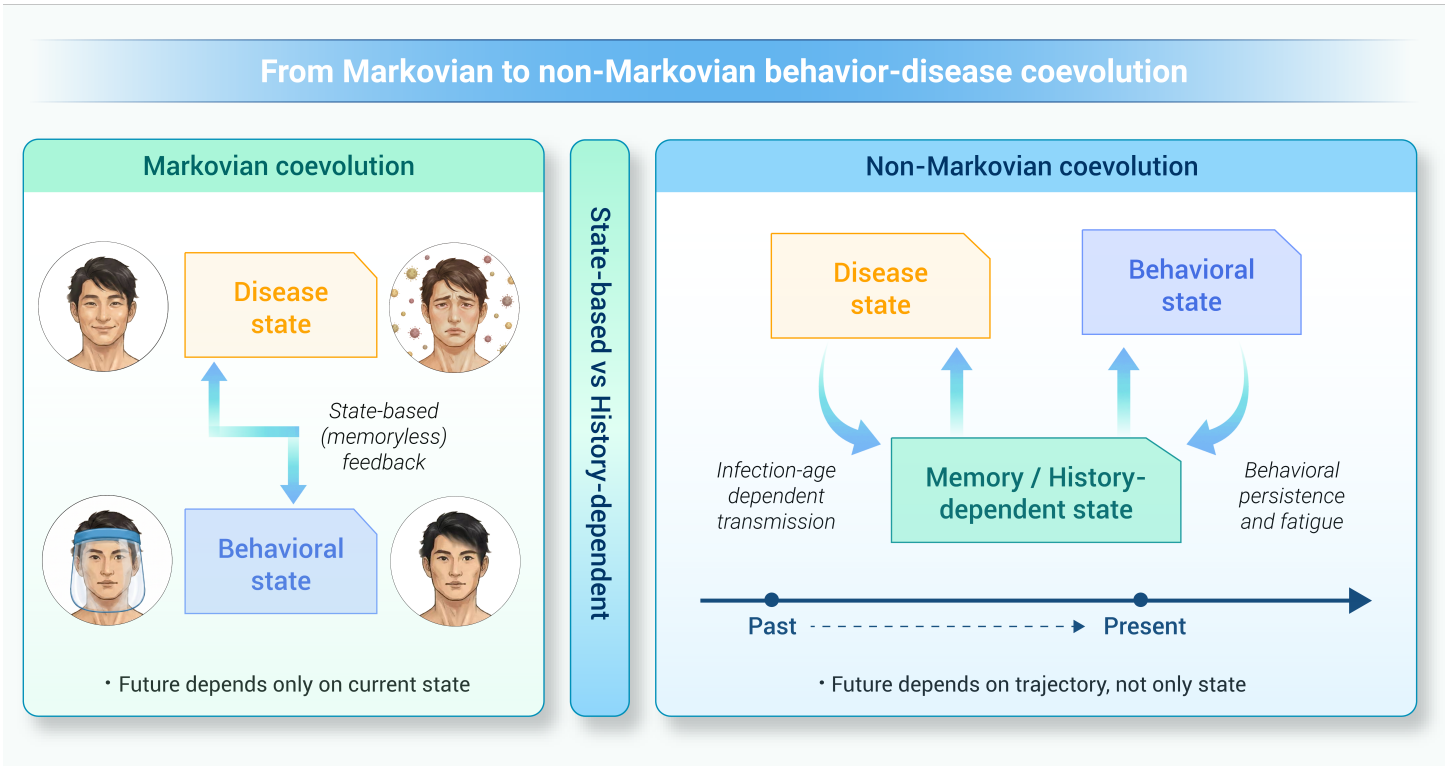

Figure 1. Markovian vs non-Markovian behavior-disease coevolution It contrasts memoryless, state-based coupling in Markovian models with history-dependent coevolution in the non-Markovian paradigm, where past states influence both transmission dynamics and behavioral adaptation, creating bidirectional feedback.

Most classical epidemiological models adopt a separation-of-scales perspective. Behavioral effects are often incorporated through static or slowly varying parameters, for example, assuming a fixed fraction of protected individuals or a constant reduction in transmission rate, while disease dynamics are described using Markovian assumptions for analytical convenience.[3] Although such formulations have proven useful for short-term forecasting and control analysis, they struggle to capture several empirically observed features of real epidemics, including recurrent waves driven by behavioral relaxation, protective fatigue, and strong timing effects of interventions.[4] A central limitation is the assumption of memorylessness. In reality, both transmission and behavior exhibit pronounced temporal structure: infection generation times, recovery processes, and behavioral responses often follow non-exponential distributions with clustering, heavy tails, and long-range dependence. Ignoring these temporal dynamics suppresses the feedback mechanisms that drive complex epidemic patterns.[5]

These observations motivate a shift toward tightly coupled, non-Markovian models of behavior–disease coevolution. This editorial does not aim to propose a specific modeling algorithm or technical solution. Rather, it seeks to articulate a unifying conceptual lens for understanding how strategic behavior and contagion dynamics interact through memory and feedback on a unified time frame. In particular, the key conceptual advance lies not in coupling per se, but in recognizing that both transmission risk and behavioral decision-making depend explicitly on past trajectories, rather than solely on current states. To convey this perspective intuitively, Figure 1 provides a conceptual comparison between Markovian and non-Markovian behavior–disease coevolution, illustrating the proposed tightly coupled, history-dependent framework. As illustrated, behavior-disease interaction is regarded as a tightly coupled, non-Markovian coevolutionary process.

Epidemiological and behavioral signals are continuously integrated through memory-dependent temporal structure, such that past states shape present transmission risk and strategic incentives. This perspective shifts the modeling focus from static behavioral assumptions to dynamic feedback operating on this shared temporal scaffold, providing a conceptual foundation for informatics-driven approaches to epidemic modeling.

In this paradigm, behavioral strategies and epidemiological states are not layered as separate modules but are embedded within a unified dynamical system evolving over time. Non-Markovian dynamics are not treated as technical complications to be averaged out, but as core organizing principles through which past states shape present risk and decision-making. This reframing shifts the central questions from how behavior modifies transmission parameters to how adaptive decision-making and contagion dynamics jointly shape long-term system behavior. Unlike Markovian formulations, which rely solely on the current state to determine future dynamics, the non-Markovian perspective incorporates duration, accumulation, and temporal ordering of past states as essential components of the system state itself.

A common conceptual starting point is to consider individuals characterized jointly by an epidemiological state (e.g., susceptible or infectious) and a behavioral strategy (e.g., adopting or not adopting protection). In non-Markovian transmission processes, infection risk depends on infection age rather than being constant in time. Tight coupling arises when this temporally structured transmission process is directly conditioned on behavioral states. In this view, behavior does not merely affect whether transmission occurs, but when transmission events are likely to occur. The resulting feedback is immediate and bidirectional: strategic decisions reshape temporal transmission patterns, while evolving infection pressure alters strategic incentives.

The scope of tight coupling extends beyond contact-level effects. A broader perspective allows behavioral dynamics to act directly on node-level states and latent memory variables that evolve alongside epidemiological processes. This node-centric view captures phenomena such as behavioral persistence, habituation, and compliance fatigue that cannot be reduced to static parameter changes. By expanding the locus of coupling from edges to individuals, the framework moves toward a more expressive representation of adaptive behavior in epidemic systems.

The interaction between memory and tight coupling gives rise to system-level behaviors that are difficult to capture with simpler formulations. Within this framework, non-Markovian coevolution can qualitatively alter epidemic dynamics, including shifts in epidemic thresholds due to generation-interval structure, changes in peak timing arising from misalignment between infectiousness and behavioral response, the emergence of endogenous oscillations driven by delayed behavioral adaptation, and the formation of history-dependent steady states. Temporally heterogeneous transmission can lead to early surges that outpace behavioral response, while delayed relaxation of protective behavior can suppress outbreaks even under favorable biological conditions. At longer timescales, coevolutionary steady states emerge in which endemic prevalence and protective adoption are jointly shaped by strategic incentives. Importantly, these outcomes arise as emergent properties of feedback and memory, rather than as artifacts of irrational behavior.

Viewing epidemics through this lens shifts attention from isolated metrics toward system-level regimes and transitions. Questions of interest become: What stable coevolutionary states exist between behavior and disease? Under what conditions do tipping points separate disease-free and endemic regimes? How does social connectivity amplify or dampen behavioral feedback? In tightly coupled systems, network structure acts not merely as a conduit for transmission but as a social amplifier that can intensify feedback and promote polarized outcomes.

Looking ahead, significant challenges and opportunities remain. Analytically, the stability and phase behavior of tightly coupled, non-Markovian systems are still poorly understood. Nevertheless, emerging evidence suggests that under appropriately structured behavioral modulation mechanisms, analytical or even closed-form descriptions can still be obtained, including in networked settings. Computationally, efficient methods for simulating and inferring long-memory processes on adaptive networks are needed. From an innovation informatics perspective, the conceptual structure outlined here naturally complements data-driven and AI-based approaches by providing interpretable constraints and principled inductive bias. In particular, data-driven methods can assist in (i) inferring temporal response structures such as memory kernels, (ii) estimating latent behavioral states and heterogeneous response patterns, and (iii) supporting calibration and surrogate prediction in complex non-Markovian systems. Hybrid strategies that integrate mechanistic insight with temporal learning may offer a promising path forward.

Ultimately, the goal of a tightly coupled, non-Markovian paradigm is not merely descriptive realism but predictive and decision-relevant insight. By formalizing the temporally structured interaction between behavior and contagion, this framework advances toward a science of adaptive resilience—one in which social response and biological spread are understood as inseparable components of a single complex system.

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AUTHOR CONTRIBUTIONS

F. Wang: Writing–original draft, Methodology, Project administration, Funding acquisition, Conceptualization, Formal analysis. K. X. Wei: Visualization and Writing – review & editing. Z. H. Zhang:Validation and Writing – review & editing. Y. G. Wang: Writing – review & editing. All authors contributed to the manuscript and approved the final version.

DECLARATION OF INTERESTS

You-Gan Wang is an Editorial Board member of The Innovation journals and was blinded from reviewing or making final decisions on the manuscript. Peer review was handled independently of this member and their research group. The other authors declare no conflicts of interest.

DATA AND CODE AVAILABILITY

No new data were created or analyzed in this study.