

Memes as Structural Signals: A Chemical Model of Interpretive Compatibility

Eric Gladstone

Independent Researcher

Traverse City, Michigan, United States

David C. Thompson

Chemical Computing Group

Montreal, Quebec, Canada

To whom correspondence should be addressed:

E-mail: Eric.C.Gladstone@gmail.com

206-259-1912

Author Contributions

Both authors contributed equally to this work

Abstract

Cultural propagation models frequently fail to account for the emergence, recombination, and persistence of complex memetic structures across divergent interpretive contexts. Conventional memetics, reliant on genetic metaphors of high-fidelity replication, neglects the structural affordances and systemic constraints governing meaning-making within a host ecology. The pharmacophore-reactome framework introduced in the current work recharacterizes memes as structured, interpretable configurations operating within a distributed interpretive environment. Memetic transmission is thus determined not by copying accuracy, but by the binding affinity between substructural sign-motifs and the internalized semiotic architecture of the receiver. Grounded in Peircean triadic logic, this mechanism demonstrates how cultural signs acquire functional utility only through integration into compatible semantic receptors.

Such a conceptual shift accommodates selective inhibition, modular recombination, and functional clustering within a heterogeneous and evolving semantic ecology. Shifting the analytical focus from the replicator to the interpretable motif enables the simulation of docking interactions and structure-function prediction within cultural systems. Finally, the model provides a formal basis for explaining memetic resistance and non-linear signal propagation in dynamic digital landscapes.

Keywords: Memetics, Peircean semiotics, Biosemiotics, Pharmacophore model, Memetic reactome, Symbolic inheritance

1. Introduction

Contemporary cultural theory has struggled to account for the structural features that make some memes persist while others are quickly extinguished, a foundational challenge for both memetic theory and contemporary studies of digital culture (Blackmore, 1999, Jenkins, Ford, & Green, 2013, and Shifman, 2014). Indeed, figuring out the basis for why some content achieves virality, while other content does not, is an important question across academic and practitioner disciplines. Although the concept of the meme has circulated widely, it has remained conceptually and formally underpowered. Its treatment often defaults to analogies with replication, mutation, and selection drawn from genetic evolution (Dawkins, 1976, and Aunger, 2000), despite well-known limitations of these analogies when applied to meaning-bearing structures (Kull, 2000, Deacon, 1999). In many of these accounts, the meme is treated as a discrete unit of transmission whose survival depends on fidelity of replication, not on interpretive viability or structural affordances within a host. In this paper, rather than understanding memes as replicators, we model them as structured agents of interpretation, cultural pharmacophores that operate within an evolving memetic reactome. This framework borrows from computational drug design, where pharmacophores are not entire molecules but structural motifs that activate or inhibit functional responses when docked

into compatible receptors (Güner, 2014, and see also Wermuth, Ganellin, Lindberg, & Mitscher, 1998, for the formal IUPAC definition). We extend this idea into a semiotic ecology in which memes function as modular substructures that bind with, inhibit, or potentiate other memes (and minds) through interpretive docking. Cultural evolution then, we submit, is less a matter of raw replication than of successful interpretability within a structured semantic system.

This shift aligns with a broader tradition in biosemiotics that treats semiosis as a foundational property of life and cognition (Sharov & Vehkavaara, 2015, and Hoffmeyer, 2008). It echoes longstanding critiques of transmission-based models of communication, which presume too much symmetry between sender and receiver and too little structural differentiation within interpretive systems (Eco, 1976, Sperber & Wilson, 1995, and also see Brashears and Gladstone, 2016). From a Peircean perspective, replication is not semiosis. Meaning emerges not from the recurrence of a sign-form but from the differential logic of interpretation, where signs gain function only through their integration into a triadic relation involving object, interpretant, and sign (Peirce, 1931–1958).

We argue that memes, to function semiotically, must possess internal docking structures that allow for interpretive uptake, inhibition, or recombination. This approach enables a non-arbitrary structural theory of memes, grounded in their capacity to interact with the interpretive architecture of cultural agents. It also opens the door to operational modeling. If memes are pharmacophore-like structures, then cultural systems may be modeled as evolving reactomes whose internal configurations constrain or enable docking patterns. Such modeling could account not only for meme spread but also for meme resistance, modulation, and structural mutation within an interpretive landscape. Ultimately, this should provide a sufficient framework for the prediction of memetic fit within a given population or culture.

2. Theoretical Frame: Peircean Semiotics and Evolutionary Signs

In Peircean semiotics, a sign is not a static code or symbol, but a structured process. It mediates a relation between three components: the object (what is represented), the representamen (the sign vehicle), and the interpretant (the effect produced in a receiving system) (Peirce, 1931–1958). This triadic structure contrasts with dyadic models of communication, which presume a simple transmission between sender and receiver. Peirce's account foregrounds the conditions under which a sign becomes meaningful at all. Meaning is not embedded in the signal but arises through a system's capacity to produce interpretants in response.

This focus on interpretability rather than transmission has become central to the biosemiotic reframing of evolution. In this view, genetic sequences, cellular signals,

behavioral displays, and cultural forms can all be treated as signs, so long as they elicit structured and meaningful responses within a living system. Semiosis, then, is not limited to symbolic language but applies across levels of life and cognition (Sharov & Vehkavaara, 2015). What matters is not replication fidelity, but compatibility with the interpretive machinery of a given system (Kull, 2009).

Interpretability is not a binary construct but a graded capacity. Following Kull's (2000) typology of semiotic thresholds, we treat interpretability as a continuous variable indexed by the probability that a sign produces an interpretant within a given reactome. It varies with sign structure, interpretive context, and the prior history of the host system. What makes this framing analytically useful, rather than merely descriptive, is that it permits interpretability to be ordered across cases and to be modeled as a distribution over possible interpretants rather than as a binary success/failure event. Some signs are inert, failing to trigger any interpretant. Others are catalytic, initiating cascades of interpretation, inhibition, or transformation depending on how they interact with prior signs. These dynamics create a selective landscape of interpretive constraints in which signs do not operate in isolation but through evolving systems of meaning-making. Such systems form ecologies of recognition, where certain forms persist not because they are copied accurately, but because they fit the interpretive architecture of the environment (Hoffmeyer, 2008). Relaxing requirements for isomorphic processes allows for the great cultural variation observed in general systems (Brashears and Gladstone, 2016). Peircean systems also support the idea of modular semiosis: the idea that interpretive processes can be segmented into smaller, reusable structures (Stjernfelt, 2007). Just as genes operate within regulatory networks, signs function within semiotic networks whose topology shapes the uptake and transformation of new elements. This enables interpretive recombination, inhibition, and resonance across levels of meaning. In such systems, evolutionary success depends not only on propagation, but on maintaining forms that remain legible and usable across shifting interpretive contexts. Culturally, many successful memes are indeed broken down into their constituent parts, and "remixed" to better dock with different populations.

The present argument builds on, and seeks to extend, a substantial body of prior Peircean and biosemiotic scholarship on memetic transmission. Deacon (1999) argues that Dawkinsian memes, lacking the triadic structure of sign action, cannot participate in genuine semiosis. What propagates, on his account, is not a replicator but an interpretant chain whose continuity depends on iconic, indexical, and symbolic relays. Kull (2000) develops a parallel critique, distinguishing copying (a dyadic operation) from translation (a triadic one), and arguing that cultural inheritance is inescapably the latter. Nathan Houser, writing in a broader pragmatist register, has argued that Dennett's treatment of memes recapitulates a dyadic information-transfer model that Peirce's semiotics explicitly rejects, and that any adequate account of cultural transmission must recover the full

triadic structure of sign action (Houser, 2003). Most directly relevant to the present paper, Cannizzaro (2016), in what remains the most sustained Peircean treatment of internet memes, develops a sign-typological account distinguishing memes that operate primarily as icons, indices, or symbols, and demonstrates that online memetic practice is structurally incompatible with the replication-fidelity model.

Deacon, Kull, Houser, and Cannizzaro collectively establish that memes are signs and require triadic analysis. They do not, however, specify the structural conditions under which particular signs bind with particular interpretive systems. Cannizzaro in particular classifies memes by sign type (iconic, indexical, symbolic) but does not offer a decompositional account of what within a meme does the binding work. The pharmacophore analogy developed below is offered as precisely this specification: a framework in which the sub-structural features of a sign and the receptor affordances of a host reactome together determine whether semiosis occurs at all, and if so, with what interpretive effect. Section 3 develops this specification. Section 5 demonstrates it in an applied case. This perspective opens the door to a more precise understanding of memetics. If memes are to be treated as signs, they must be analyzed not in terms of replication, but in terms of interpretive docking. Their stability depends on the structural features that allow them to activate specific interpretive responses within a given semiotic ecology. The next section develops this claim through the pharmacophore-reactome model, offering a formal account of what it means for a meme to bind to an interpretive system.

3. Pharmacophores and the Memetic Reactome

If we adopt a semiotic view of evolution, in which propagation depends on interpretability rather than replication alone, then the relevant question for memetics becomes structural: what features of a cultural form make it legible, modular, and re-combinable across minds and contexts? To answer this, we model memes as pharmacophores. In molecular pharmacology, a pharmacophore is the minimal structural configuration of a compound required to trigger a biological response (Güner, 2014, and Wermuth et al., 1998). In this way, it is similar to the logic in formal hypothesis testing, a stimulus is sufficient, but perhaps not required (pending future findings), to elicit a given response. It is not a complete molecule, nor its origin, but represents the spatial arrangements of the 'features' of the molecule that confer an experimentally observed functional interaction. A meme, in this framework, is not a replicating particle or discrete message, but a substructural sign whose efficacy emerges only through docking with a compatible interpretive site. Its cultural function depends not on its origin or surface fidelity, but on whether it can elicit a response within a receptive system. Recognition is, therefore, conditional. It depends on the system's prior interpretive development, and on the presence of latent receptor structures such as schemas, sensibilities, or symbolic

expectations that are capable of activation. We call this system the memetic reactome: the distributed ensemble of semiotic constraints, receptor sites, and activation potentials that structure interpretive uptake.

Grounding the pharmacophore concept in Peircean triadic logic

The analogy between a pharmacophore and a sign requires justification. We ground it in recent biosemiotic accounts of protein folding by Lacková (2020, 2025) and Bennett (2025), who treat folding as a semiotic process rather than merely as a structural precondition for semiosis. This point matters for the pharmacophore concept because receptor-ligand recognition depends on folded molecular forms that mediate selective response. A pharmacophore is not simply a set of chemical features present in a ligand. It is a relational configuration that becomes functionally legible only within a receptor environment capable of discriminating among possible ligands.

The contrary view, developed by Pattee (2021), is that protein folding is a lawful physical process that "leaves neither freedom nor necessity for interpretation," and that Peircean vocabulary is "gratuitous" at the molecular level because interpretation requires the possibility of choosing alternatives. We do not adopt this view, for two reasons. First, following Bennett (2025), the absence of an interpreter does not entail the absence of an interpretant. Triadic structure is preserved at the molecular level by mediatedness, and the folded protein structure is itself, in Bennett's formulation, an "evolutionary habit, final interpretant of protein synthesis." Second, the selectivity Pattee reserves for higher levels is already present at the receptor-ligand interface, where receptors discriminate among ligands of similar gross structure on the basis of finer pharmacophoric features. The pharmacophore concept therefore depends on precisely the kind of selective discrimination Pattee places outside semiosis.

With these grounding moves established, three claims become available to the present argument. First, following Lacková (2020), the unfolded peptide chain is best understood as a continuum, a firstness, a domain of mere possibility without articulated identity. The folded structure is what produces the discrete dyadic identity of the amino-acid residues responsible for folding. Triadicity precedes dyadicity, and articulation follows folding rather than the reverse. Second, the folded protein structure is itself a Peircean final interpretant, an evolutionary habit stabilized into lawlike functional regularity (Bennett 2025). Third, a pharmacophore, in the chemical sense (Güner 2014), is a sub-structural feature of a folded protein responsible for receptor binding. It is therefore a sub-feature of an already-Peircean object, a structural relate within a final interpretant. The receptor-binding event is then a relation between two such final interpretants, ligand and receptor, both of which are semiotic by virtue of their evolutionary-habit status.

This grounding does two kinds of work for our argument. First, it answers the prior question of why pharmacophoric logic belongs in a Peircean account of cultural

transmission at all: the pharmacophore is already, at the molecular level, a sub-structural feature of a Peircean final interpretant. Cultural memes are not being forced into a chemical metaphor. They are being described using the same triadic logic that already applies to the chemistry itself. The analogy is not one of surface similarity but of shared formal structure, what Stjernfelt (2007) would call a diagrammatic identity across substrates. Second, it clarifies what we mean by a "receptor site" in the memetic reactome. A receptor site is not a passive container waiting to be filled. It is itself a folded final interpretant, a structural relate within an evolutionary habit, that, when engaged by a compatible representation, produces a stabilized response. In both the molecular and cultural cases, the sign's efficacy depends on this triadic completion, not on token replication.

This perspective reorients memetic theory away from transmission and toward binding. Unlike replication-based accounts, which treat memes as autonomous units of selection (Dawkins, 1976, and Blackmore, 1999), the pharmacophore model makes interpretive viability the central variable. A meme may be widely transmitted but rarely bind. It may inhibit rather than activate. Such antimemes (Asparouhova, 2025) go beyond simply being "non-memetic" (i.e., failing to spread), and they function as active inhibitors, preventing the binding of competing sign-structures. It may serve as a structural scaffold that enables other memes, without producing any direct expression itself. These are not transmission dynamics, rather, they are receptor-binding dynamics conditioned by structural constraints.

This model also permits screening. Just as pharmacophores can be evaluated for their binding affinity across receptor classes, memes can be assessed for their interpretive resonance across cultural systems. Some bind widely, activating shared inferential pathways. Others require scaffolding, emotional priming, or prior exposure. Still others interfere with competing memes, constraining interpretive bandwidth or closing off semiotic routes. These dynamics are context-dependent, asymmetric, and often latent until triggered. Put most broadly, the receptivity conditioners are contained in one's culture, and one's time.

The memetic reactome, like the proteome, evolves. It may be sensitized, desensitized, or rendered dormant through saturation, repetition, or prior engagement. The structure of the reactome shifts over time, reshaping the conditions under which new memes can bind. From this perspective, cultural propagation is not a function of copying fidelity but of system-level docking potential. Transmission without docking is inert. Cultural evolution depends not on spread alone, but on interpretive fit.

By reframing memes as pharmacophores, one moves from imitation to activation, from fidelity to affordance. This approach can shed light as to why some memes persist while others fade, not due to a lack of persistence, but due to a lack of sustained structural

resonance within dynamic interpretive systems. Generally, this also speaks to why the creation of viral content is, broadly, a see-what-sticks process. The next section develops the consequences of this reorientation, including the logic of inhibition, coactivation, and sequence-dependence across memetic ecologies.

4. The Memetic Reactome: Compatibility, Inhibition, and Interpretive Ecology

If memes are understood as pharmacophoric sign-structures, then their propagation should be conceptualized not as a linear transmission process, but rather, as a distributed network of interpretive interactions. We introduce the term memetic reactome to describe the semiotic environment in which these interactions unfold. Borrowed from systems biology, where a reactome refers to the full set of biochemical reactions active within an organism, the memetic reactome denotes the ensemble of interpretive sites, constraints, and affordances that determine whether and how a meme exerts influence.

We can now specify the Peircean structure of the reactome. The receptor site, the meme (as representamen), and the binding event (producing the interpretant) together form a triadic relation, but each element corresponds to a characteristic Peircean category. The unbound receptor state is an instance of firstness: a qualitative potential for interpretation that exists independently of any particular sign engaging it. The binding event itself is secondness: an actual dyadic contact between sign and receptor, here-and-now, in a particular reactome at a particular time. The stabilization of that event into a recurrent, rule-governed interpretant (the pattern by which subsequent encounters with the same pharmacophoric structure reliably produce the same interpretive effect) is thirdness, the lawlike regularity that makes the sign a sign rather than a passing stimulus. Extended over time, these regularities constitute what Hoffmeyer (2008) calls the semiotic scaffold of a culture, and what the biosemiotic tradition following Uexküll calls its Umwelt. The reactome, on our account, is the collection of firstness-affordances within a given Umwelt that stand ready to produce thirdness-stabilized interpretants when engaged by compatible pharmacophoric structures.

In this framework, memetic efficacy does not depend on exposure alone, but on binding compatibility, a meme's structural fit with latent interpretive receptors. These receptors may include cognitive schemas, emotional sensitivities, ideological commitments, or symbolic expectations. Importantly, said receptors are not static. They evolve with the interpretive capacities of their host systems and are modulated by prior semiotic activity. Exposure to one meme may sensitize or desensitize a system to another, shifting the conditions under which binding can occur.

This perspective draws a non-trivial distinction between transmission fidelity and interpretive viability. Traditional models often assume that a meme must replicate accurately to persist. Indeed, many computational or agent-based models of idea

transmission do just this. The pharmacophoric approach rejects this, proposing instead that persistence depends on whether a meme retains the minimal structural features necessary for activation. A sign may be degraded, paraphrased, or visually altered, yet still bind effectively if its pharmacophoric constraints remain intact. This logic underpins the ways in which smaller modules within a given meme can be substituted for other content, while the essence of the meme (built on larger, more foundational modules) remains.

This also introduces inhibition as a distinct mode of memetic influence. Not all signs activate interpretive responses, and some block them. A satirical meme may prevent a serious claim from gaining traction, not through argument but by preemptively altering the interpretive terrain. Here, inhibition functions not by displacing content, but by reconfiguring the receptor environment such that competing memes no longer bind. The analogy to molecular inhibitors is precise: both function by altering local binding conditions without requiring replication of form.

The memetic reactome further supports coactivation and combinatorial effects. Some memes exhibit little influence in isolation but become catalytic when paired with other structures. A phrase may be inert on its own yet trigger a cascade when paired with a recognizable image or shared narrative frame. These dependencies produce non-linear propagation patterns such as threshold effects, feedback loops, and synergistic chains, none of which are accounted for by single-message exposure models.

In sum, the memetic reactome reframes cultural transmission as a process of semiotic binding, inhibition, and recombination within a dynamic interpretive ecology. It offers a middle path between replication-based and purely constructivist models by formalizing how interpretive compatibility governs memetic uptake. Rather than asking whether a meme is faithfully copied, the reactome model asks whether a sign remains recognizably functional under systemic constraints. This shift in focus lays the groundwork for the applied analysis that follows.

5. A Worked Example: "Okay, Boomer"

The theoretical claims developed above can be evaluated for their operational value only through applied analysis. Using only the vocabulary established in the preceding sections we demonstrate that the pharmacophore-reactome framework surfaces structural features of memetic propagation that replication-fidelity accounts do not, and that its Peircean grounding generates predictions that can in principle be tested.

Identifying the pharmacophore

"Okay, Boomer" emerged in late 2019 and achieved peak circulation across English-language social platforms between November 2019 and early 2020. The meme's surface form is a two-word dismissive reply, typically deployed in response to a statement

attributed to, or heard as coming from, an older interlocutor. Following the pharmacophoric logic developed in Section 3, we propose that the meme's efficacy depends not on the exact phrase but on the preservation of a minimal set of structural features: (i) a concessive-dismissive particle that ostensibly grants the interlocutor's point while structurally refusing to engage it ("okay," "sure," "alright"), (ii) a generational or stereotype-laden proper-noun address that marks the interlocutor as a member of an outgroup defined by birth cohort or social type ("Boomer," "Karen," and in some variants "Zoomer"), and (iii) a prosodic flatness, rendered in text through absence of punctuation, emphasis, or elaboration, that signals exhaustion rather than argument. Variants that preserve these three features (for example, "Sure, Karen" and "Alright, Gen X") continue to bind effectively. Variants that lose them (an earnest "I hear your concerns, Boomer," or a hostile "Shut up, Boomer") fail to activate the characteristic interpretant. This pattern is consistent with pharmacophoric logic in the Wermuth et al. (1998) sense: the minimal activating configuration is preserved across variation in tokens, and loss of any one feature attenuates the response.

Specifying the receptor site

The pharmacophore is inert outside a compatible reactome. The receptor in this case is not a single cognitive schema but a compound structure: (i) a latent schema of intergenerational power asymmetry, in which older cohorts are perceived as holding institutional authority while remaining unpersuadable, (ii) an affective disposition toward exhaustion, coupled with the expectation that substantive engagement with the addressee will be unproductive, and (iii) a higher-order meta-interpretive frame that treats public argument itself as a game in which dismissal can score points that rebuttal cannot. In the Peircean terms developed in Section 4, this receptor is not a single object-relation but a nested firstness-thirdness assembly: the schema (thirdness, a rule-governed expectation) conditions the affective readiness (firstness, a qualitative potential for dismissal), which is then actualized in the binding event (secondness, the actual dismissive reply). Crucially, this receptor is unevenly distributed. It is richly developed in cohorts that came of age online between roughly 2012 and 2020, sparse among cohorts whose intergenerational interpretive frames were formed before the emergence of platform-mediated public argument, and effectively absent outside English-dominant internet cultures. The meme's famous cohort specificity is, on this analysis, not a feature of the meme itself but a feature of its reactome.

The binding dynamic

Why does the pharmacophore, once placed into a compatible reactome, succeed? The binding-affinity story turns on a structural feature the existing memetics literature has not isolated: "Okay, Boomer" is a response that refuses the frame of argument while acknowledging the addressee. Classical rebuttal requires a propositional target. It engages a claim in order to defeat it. "Okay, Boomer" provides no propositional target. It grants the speaker's point (the concessive "okay") and then declines to contest it, while marking

the speaker as structurally incapable of receiving contestation (the generational address). The receptor responds with a stable interpretant: the recognition "I have been dismissed in a way I cannot rebut." Once produced, this interpretant cannot easily be undone by further argument. The binding event is, in Peircean terms, a completed triad: the representamen (the phrase) engages the object-relation (the intergenerational schema) to produce an interpretant (the felt, and largely ineradicable, experience of being frame-excluded). This is the kind of semiotic event that Cannizzaro (2016) argues is distinctive of internet memes but does not structurally specify. The pharmacophore framework provides the specification.

Inhibition and allosteric dynamics

This model distinguishes itself from traditional semiotic perspectives specifically through its analysis of inhibition. Once "Okay, Boomer" is deployed, subsequent substantive claims by the addressee do not bind differently on their own merits. They bind differently because the receptor environment has been reconfigured. The meme does not refute the Boomer's claim, it changes the shape of the interpretive site such that any subsequent claim by the same speaker is now pre-read as an instance of the type "unpersuadable-elder speech." This is directly analogous to allosteric inhibition in molecular pharmacology: the inhibitor does not occupy the primary binding site and does not displace the substrate, it binds elsewhere on the receptor and changes the receptor's conformation such that the substrate can no longer bind productively. We propose that a substantial class of political and cultural memes function allosterically in precisely this sense. Satirical memes in particular characteristically operate by reconfiguring the receptor environment around a target claim rather than by addressing the claim itself. Replication-fidelity accounts have no natural place for this dynamic, and the pharmacophore-reactome framework derives it directly from the structural logic of receptor-ligand interaction.

Coactivation and modular recombination

The three features identified above (the concessive particle, the generational or type-based address, and the prosodic flatness) are modular. They can be recombined with other features to produce a family of related pharmacophores: "Sure, Jan" (with a sitcom-referential address rather than a generational one), "Cool, cool, cool" (with an exaggerated concessive repeat rather than an address), and so on. Each variant binds to a somewhat different receptor structure while preserving the core inhibitory function. This is consistent with the claim in Section 4 that memes coactivate and recombine combinatorially: the modular decomposition is not imposed by the analyst but is visible in the actual propagation history, in which features migrate across memes while retaining their pharmacophoric identity. No replication-fidelity account can explain why "Sure, Jan" and "Okay, Boomer" feel like cousins while sharing almost no lexical material.

Reactome evolution: saturation and desensitization

Finally, the framework makes a prediction that a replication account does not naturally generate: memes should decay not only through loss of carriers but through receptor desensitization. "Okay, Boomer" is, as of this writing in 2026, no longer fully potent. Deployed now, it reads as dated. Not because the underlying intergenerational schema has disappeared (it has not), but because the receptor has been saturated by repeated exposure. The interpretant it produces is now attenuated: the phrase reads as referring to the 2019–2020 moment rather than activating fresh dismissal. This is the cultural analog of pharmacological desensitization: the receptor remains structurally present but the signal-to-response coupling has been down-regulated by exposure history. The prediction generalizes. Memes that depend heavily on novelty (a large contribution of firstness to the binding event) should desensitize quickly. Memes that depend on thirdness-level regularities (deeply established cultural rules) should desensitize slowly or not at all. This is a testable empirical claim, and one that no purely replicative account of memetic propagation naturally produces.

What the framework did

The worked analysis above did six specific things that a transmission- or replication-based treatment of "Okay, Boomer" would not naturally do. It identified the minimal sign-features necessary for activation rather than treating the whole phrase as a unit. It specified the receptor structure as a compound assembly of schema, affect, and meta-frame rather than as a single belief or representation. It characterized the binding event as a frame-excluding completion of a Peircean triad rather than as a persuasion move. It isolated inhibition as an allosteric reconfiguration of the receptor rather than as mere displacement of content. It treated modular recombination as visible in the propagation record rather than as post-hoc analyst stipulation. And it generated a testable prediction about decay through receptor desensitization. None of these analytical operations is available within a replication-fidelity account.

6. Implications for Biosemiotic Models of Inheritance and Cultural Propagation

The pharmacophore-reactome analogy is an attempt to reframe cultural inheritance in ways that align with foundational commitments in biosemiotics. Rather than defining propagation through the lens of high-fidelity replication (a notoriously difficult process for humans), this approach centers on structural compatibility, interpretive affordance, and ecological contingency. These emphases resonate with a body of work that positions evolution not solely as a genetic process but as a semiotic phenomenon spanning signs, contexts, and interpretive systems (Kull, 2000, and Hoffmeyer & Emmeche, 1991).

Within this framework, cultural evolution is not driven by discrete units competing for replication but by configurations that persist when they remain interpretable within evolving environments. The emphasis falls not on the meme as a replicator, but on the

conditions under which interpretive binding occurs. This shift allows symbolic inheritance to be described in terms of binding sites, functional activation, inhibitory pathways, and re-combinatory structures, concepts that better reflect the systemic character of meaning propagation. Inheritance is best defined, we argue, as propagation of meaning independent of constituent ingredients.

For biosemiotics, the implications are twofold. First, the pharmacophoric model contributes to the formalization of symbolic dynamics, which remains a well-recognized area for development within biosemiotics when compared to genetic or physiological semiosis (Kull & Favareau, 2015). By treating memes as pharmacophoric signs, it is possible to identify the minimal structural conditions required for interpretive activation without presupposing agreement on form, content, or fidelity. This enables the study of cultural signs using the same formal tools applied to biochemical systems, thresholds, constraints, and activation functions. In doing so, we are concretizing the meme as an object of conceptual inquiry.

Secondly, the memetic-reactome model provides a way to connect molecular and cultural systems without collapsing their differences. The analogy is not intended to biologize culture, but instead to clarify the types of structural relationships that support meaning across systems. In this sense, the model supports a central aim of biosemiotics: to describe the continuity of semiosis from molecules to minds, not by assuming identity of substance, but by tracking continuity in function (Hoffmeyer, 2008).

This continuity does not rest on replication. Rather, it rests on interpretive viability, the capacity of a structure to participate in a functional semiotic ecology. Cultural forms persist not because they are faithfully copied, but because they continue to bind to interpretive receptors, often in altered form. The pharmacophore-meme makes this process visible. A sign structure does not need to remain identical to remain active. It must retain the requisite sub-features necessary to trigger recognition within the interpretive field.

7. Limitations of the Analogy

A productive analogy in theory-building must also be a falsifiable one. The pharmacophore-reactome framework, like all cross-domain borrowings, depends on structural isomorphism between two systems, and where that isomorphism breaks down, the framework either loses explanatory grip, or must be re-specified. We identify here four such points, both as an honest account of the framework's current limits and as an agenda for its further development.

First, measurement asymmetry. Molecular pharmacology possesses a mature toolkit for quantifying binding interactions (dissociation constants, half-maximal inhibitory concentrations, receptor occupancy curves) that has no direct cultural analog. Binding

affinity between a pharmacophore and a receptor can be measured. Binding affinity between a meme and an interpretive receptor cannot, at least not yet. The present framework therefore operates at the level of structural prediction rather than quantitative modeling. Whether culturally tractable proxies for binding affinity (e.g., time-to-interpretant measured in reaction latencies, variance in interpretant content across exposures, cohort-specific uptake rates) can be developed is an open empirical question and, in our view, the most important single avenue for subsequent work.

Second, receptor instability. In pharmacology, receptor architectures are stable over the timescale of binding events. A receptor assay run this morning will yield the same result run this afternoon. Memetic reactomes are not stable in this sense. They are themselves evolving, responsive to prior binding events, and internally heterogeneous even within a single interpreter. The pharmacophoric framework accommodates this through the notion of reactome evolution (Sections 3 and 4), but the accommodation is conceptual rather than formal. A full formalization would require treating the reactome not as a fixed receptor ensemble but as a time-indexed distribution, which raises modeling challenges that current agent-based frameworks are not yet fully equipped to meet.

Third, the boundary of the unit. In pharmacology, the pharmacophore is defined relative to a known molecular series whose composition can be specified exhaustively. In culture, the boundary of a meme is itself interpretively variable: the same propagation event may be described as one meme, two co-activating memes, or three modular sub-memes depending on the analyst's decomposition strategy. The framework treats this as a feature. Memes are pharmacophores precisely because they are sub-structural motifs rather than whole units. But the absence of a principled decomposition criterion remains a live methodological issue that future work will need to address.

Fourth, intentionality. Molecular pharmacophores do not intend to bind, and they are structural objects acted upon by physical laws. Cultural producers sometimes do intend to engineer binding, through targeting, A/B testing, and explicit design for virality. The framework is agnostic about intentionality. Binding either occurs or does not, regardless of the producer's goals. But this agnosticism is itself a substantive theoretical commitment that deserves explicit treatment. We note it here rather than resolve it.

These limits do not undermine the framework. Rather, they delimit its scope. The analogy between pharmacophoric action and semiotic action is most productive where both are triadic, structural, and receptor-dependent. It becomes less productive where pharmacology depends on quantitative parameters that have no direct cultural analogue. Recognizing this distinction allows the framework to be applied where it has analytic purchase and extended, rather than merely defended, where the analogy reaches its limits.

8. Conclusion

The meme, often criticized for its conceptual vagueness, becomes more analytically tractable when treated as a structurally constrained sign rather than a replicating unit. Through analogy with the concept of a molecular pharmacophore, we have attempted to reframe memetic action in terms of interpretive compatibility and ecological affordance, rather than fidelity or virality. The memetic reactome provides a way to model this environment formally, capturing propagation, inhibition, and coactivation across distributed interpretive networks.

The worked analysis of "Okay, Boomer" presented in Section 5 demonstrates that this reorientation is not merely conceptual: applied to a specific meme, the framework generates structural distinctions (pharmacophoric sub-features, compound receptor assemblies, allosteric inhibition, and receptor desensitization) that are unavailable within replication-based accounts. The limitations discussed in Section 7 identify where that distinctive contribution ends and where further development is needed. A computational instantiation of the framework, applying pharmacophoric structure-activity probing to image and text embeddings drawn from naturalistic memetic data, is the subject of a companion paper currently in preparation.

This framework is not offered as a replacement for existing models of cultural evolution. Rather, it clarifies a neglected level of analysis: the level at which signs must be structurally legible to function as components of semiotic life. Instead of asking whether memes resemble genes, we ask what makes a sign durable, recombinable, or suppressive within a given interpretive system. That question is not limited to memetics. It bears on the broader project of biosemiotics and on the problem of explaining how structured signs propagate within and across living systems.

Statements and Declarations

Competing Interests

The authors have no competing interests to declare that are relevant to the content of this article.

Funding

No funding, grants, or other support was received for conducting this study or for the preparation of this manuscript.

Data Availability

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

Author Contributions

Both authors contributed equally to the creation of this work.

References

- Asparouhova, N. (2025). *Antimemetics: why some ideas resist spreading*. Dark Forest Collective.
- Aunger, R. (2000). *The Electric Meme: A New Theory of How We Think*. Free Press.
- Bennett, L. L. (2025). Toward a biosemiotics framework for AI: Folding and the dynamics of meaning. *Sign Systems Studies*, 53(3-4), 444-463.
- Blackmore, S. (1999). *The meme machine*. Oxford University Press.
- Brashears, M. E., & Gladstone, E. (2016). Error correction mechanisms in social networks can reduce accuracy and encourage innovation. *Social Networks*, 44, 22–35.
- Cannizzaro, S. (2016). Internet memes as internet signs: A semiotic view of digital culture. *Sign Systems Studies*, 44(4), 562–586.
<https://doi.org/10.12697/SSS.2016.44.4.05>
- Dawkins, R. (1976). *The Selfish Gene*. Oxford University Press.
- Deacon, T. W. (1999). Memes as signs in the dynamic logic of semiosis: Sign evolution and development. In R. Aunger (Ed.), *Darwinizing Culture: The Status of Memetics as a Science* (pp. 41–64). Oxford University Press.
- Eco, U. (1976). *A Theory of Semiotics*. Indiana University Press.
- Güner, O. F., & Bowen, J. P. (2014). Setting the record straight: the origin of the pharmacophore concept. *Journal of Chemical Information and Modeling*, 54(5), 1269–1283.
- Hoffmeyer, J. (2008). *Biosemiotics: An Examination into the Signs of Life and the Life of Signs*. University of Scranton Press.
- Hoffmeyer, J., & Emmeche, C. (1991). Code-duality and the semiotics of nature. In M. Anderson & F. Merrell (Eds.), *On Semiotic Modeling* (pp. 117–166). Mouton de Gruyter.
- Houser, N. (2003). Pragmatism and the Loss of Innocence. *Cognitio* (São Paulo), 4(2), 197-210.

- Jenkins, H., Ford, S., & Green, J. (2013). *Spreadable media: Creating value and meaning in a networked culture*. New York University Press.
- Kull, K. (2000). Copy versus translate, meme versus sign: Development of biological textuality. *European Journal for Semiotic Studies*, 12(1), 101–120.
- Kull, K. (2009). Biosemiotics: To know, what life knows. *Cybernetics and Human Knowing*, 16(3–4), 81–88.
- Kull, K., & Favareau, D. (2015). Theses on the semiotic study of behaviour. *Biosemiotics*, 8(3), 329–335. <https://doi.org/10.1007/s12304-015-9244-6>
- Lacková, I. (2020). Folding of a peptide continuum: Semiotic approach to protein folding. *Semiotica*, 233, 79–90. <https://doi.org/10.1515/sem-2018-0116>
- Lacková, I. (2025). *Language of Life: A Peircean Approach to Living Organisms*. Berlin: Peter Lang.
- Pattee, H. H. (2021). Symbol grounding precedes interpretation. *Biosemiotics*, 14, 561–568. <https://doi.org/10.1007/s12304-021-09458-4>
- Peirce, C. S. (1931–1958). *Collected Papers of Charles Sanders Peirce* (Vols. 1–8, C. Hartshorne, P. Weiss, & A. W. Burks, Eds.). Harvard University Press.
- Sharov, A. A., & Vehkavaara, T. (2015). Semiotic and cybernetic aspects of life: Toward a unified view. *Biosemiotics*, 8(2), 259–271.
- Shifman, L. (2014). *Memes in digital culture*. The MIT Press.
- Sperber, D. (1996). *Explaining Culture: A Naturalistic Approach*. Blackwell.
- Sperber, D. (2000). An objection to the memetic approach to culture. In R. Aunger (Ed.), *Darwinizing Culture: The Status of Memetics as a Science* (pp. 163–173). Oxford University Press.
- Sperber, D., & Wilson, D. (1995). *Relevance: Communication and Cognition* (2nd ed.). Blackwell.
- Stjernfelt, F. (2007). *Diagrammatology: An Investigation on the Borderlines of Phenomenology, Ontology, and Semiotics*. Springer.
- Wermuth, C. G., Ganellin, C. R., Lindberg, P., & Mitscher, L. A. (1998). Glossary of terms used in medicinal chemistry (IUPAC Recommendations 1998). *Pure and Applied Chemistry*, 70(5), 1129–1143.