Social Copying as the Collective Fight-or-Flight:

Ancient Stress Circuitry Repurposed for Individual and Collective Survival

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Abstract. The fight-or-flight (FoF) response is universally recognized as an innate, neurologically hardwired survival mechanism—a ~550-million-year-old system that automatically overrides rational deliberation to ensure individual survival under physical threat. Social copying of individuals within a social organism under stress and uncertainty constitutes a deeply conserved, largely automatic survival system—one that operates at the collective rather than individual level. Like FoF, social copying is triggered by stress, mediated by specific neurochemical pathways, and can override or bias individual rational behavior. Unlike FoF, social copying serves the survival of the social group rather than the individual organism, often by prioritizing group cohesion and coordinated response over individual survival. Scientists and the broader public readily accept that FoF "hijacks" rational thought for individual survival, yet resist the parallel claim that social group identity (SGI) mechanisms hijack rational thought for collective survival. By systematically demonstrating the structural, neurochemical, and functional parallels between these two systems—and grounding both within an evolutionary immunity framework where FoF represents individual immunity and SGI represents collective immunity to outside threats—this paper argues that social copying under stress is not cultural programming or personality weakness but a foundational feature of the biology of social organisms, analogously conserved from social amoebae through to human neural conformity circuits. The neurological comparison reveals that social copying and FoF recruit overlapping stress-detection circuitry: both share an amygdala–ACC–PVN–locus coeruleus core, but diverge at execution. FoF activates the sympathetic nervous system via catecholamines for physical action, whereas social copying activates ventral striatum–centered dopamine reward systems and perception–action coupling circuits (including mirror mechanisms) for behavioral conformity. Critically, FoF is typically inhibited by dlPFC-mediated control to allow rational behavior, whereas resisting social copying typically requires dlPFC-mediated control to maintain individual rational judgment—an inverted but parallel control logic (§4.2.2).

1. Introduction

When a human encounters a sudden physical threat—a car swerving into their lane, a loud unexpected noise—the body responds before conscious thought can intervene: heart rate spikes, muscles tense, pupils dilate, and blood redirects to skeletal muscles (Cannon, 1915; Godoy et al., 2018; Sapolsky, 2004). This is the fight-or-flight response, first described systematically by Walter Cannon in 1915, and it is now widely accepted as an innate, genetically hardwired survival mechanism that can override the slower rational deliberation to preserve the individual organism (Cannon, 1915; Ulrich-Lai & Herman, 2009).

Far less accepted—yet supported by converging evidence from neuroscience and evolutionary theory—is the proposition that social organisms possess an automatic social copying response under stress and uncertainty that can override individual rational behavior to preserve the collective (Mann, 2020; Mukherjee & Bassler, 2019; Strassmann & Queller, 2011). When humans face social uncertainty or perceived threats to their group, a characteristic neural cascade often biases cognition from individual assessment toward social copying: alignment with group consensus, conformity to group norms, and subordination of private judgment to collective behavior (Baddeley, 2010; Bond & Smith, 1996; Lin et al., 2018; Stallen & Sanfey, 2015; Wu et al., 2016).

The resistance to this idea is itself instructive. Academics and the general public readily accept that adrenaline and related stress hormones can "hijack" the prefrontal cortex during a physical emergency (Sapolsky, 2004). Yet the same populations resist the structurally similar claim that dopaminergic reward circuits and social-valuation networks can "hijack" prefrontal control during a social emergency—that group-identity mechanisms override rational thought just as fight-or-flight does (Chen et al., 2023; Klucharev et al., 2009; Ruff & Fehr, 2014; Zaki et al., 2011). This resistance likely reflects the very mechanism under discussion: acknowledging that one's own group affiliations operate through ancient, automatic neural circuits rather than through unconstrained rational choice threatens the narrative of individual cognitive autonomy (Baddeley, 2010; Bond & Smith, 1996; FeldmanHall & Shenhav, 2019).

This paper addresses that resistance by presenting social copying under stress as the structural and neurochemical parallel of fight-or-flight, systematically demonstrating that both systems share evolutionary origins, neural architecture, stress-triggered activation, and the capacity to override prefrontal deliberation (Loomis, 2014; Mukherjee & Bassler, 2019; Sgro et al., 2015; Strassmann & Queller, 2011; Toelch & Dolan, 2015; Wu et al., 2016). The argument is further grounded within the evolutionary immunity framework developed in (Johnson, 2026), where fight-or-flight represents individual immunity (Level 2A)—the organism's innate defense against physical threats—and social group identity represents collective immunity (Level 3)—the social organism's innate defense against threats to collective survival and cohesion (Beaumont et al., 2009; Boomsma & Gawne, 2018; Goodnight, 2005; Simons, 2011; Starrfelt & Kokko, 2012; Wilson & Wilson, 2007).

Throughout this paper, the Evolution of Immunity Framework of levels is used (Johnson, 2026): *Level 2A* denotes hardwired individual response (e.g., sympathetic outflow in FoF), *Level 2B* denotes adaptive individual deliberation (dlPFC, vmPFC), *Level 3A* denotes hardwired collective enforcement (e.g., the paired-gradient of ACC social pain + ventral striatum social reward), and Level 3B denotes the adaptive collective content (social group identity, group norms) that Level 3A enforces. Full development of the immunity framework — including why each system maps onto the level it does — follows in §5.

2. Fight-or-Flight: The Individual Survival Response

2.1 Evolutionary Status: Innate and Ancient

The fight-or-flight response is recognized as a fixed, innate survival mechanism shaped by natural selection across vertebrate evolution (Cannon, 1915; Godoy et al., 2018; Rodrigues et al., 2009; Sapolsky, 2004). It operates as what evolutionary psychologists call a "fixed action pattern"—an automatic, evolutionarily stable strategy that activates reflexively when organisms detect threats (Sapolsky, 2004; Schulkin, 2009). Unlike learned behaviors, the FoF response is genetically hardwired, requiring no conscious deliberation and proceeding automatically once triggered (Cannon, 1915; Ulrich-Lai & Herman, 2009). The evolutionary logic is straightforward: ancestral animals that responded quickly to predators or dangers without time for complex decision-making survived to reproduce, while slower responders did not (Godoy et al., 2018; Sapolsky, 2004). This

selective pressure embedded the response deeply into nervous system architecture across vertebrate lineages (Denver, 2009; Edens et al., 2024; Yu et al., 2023).

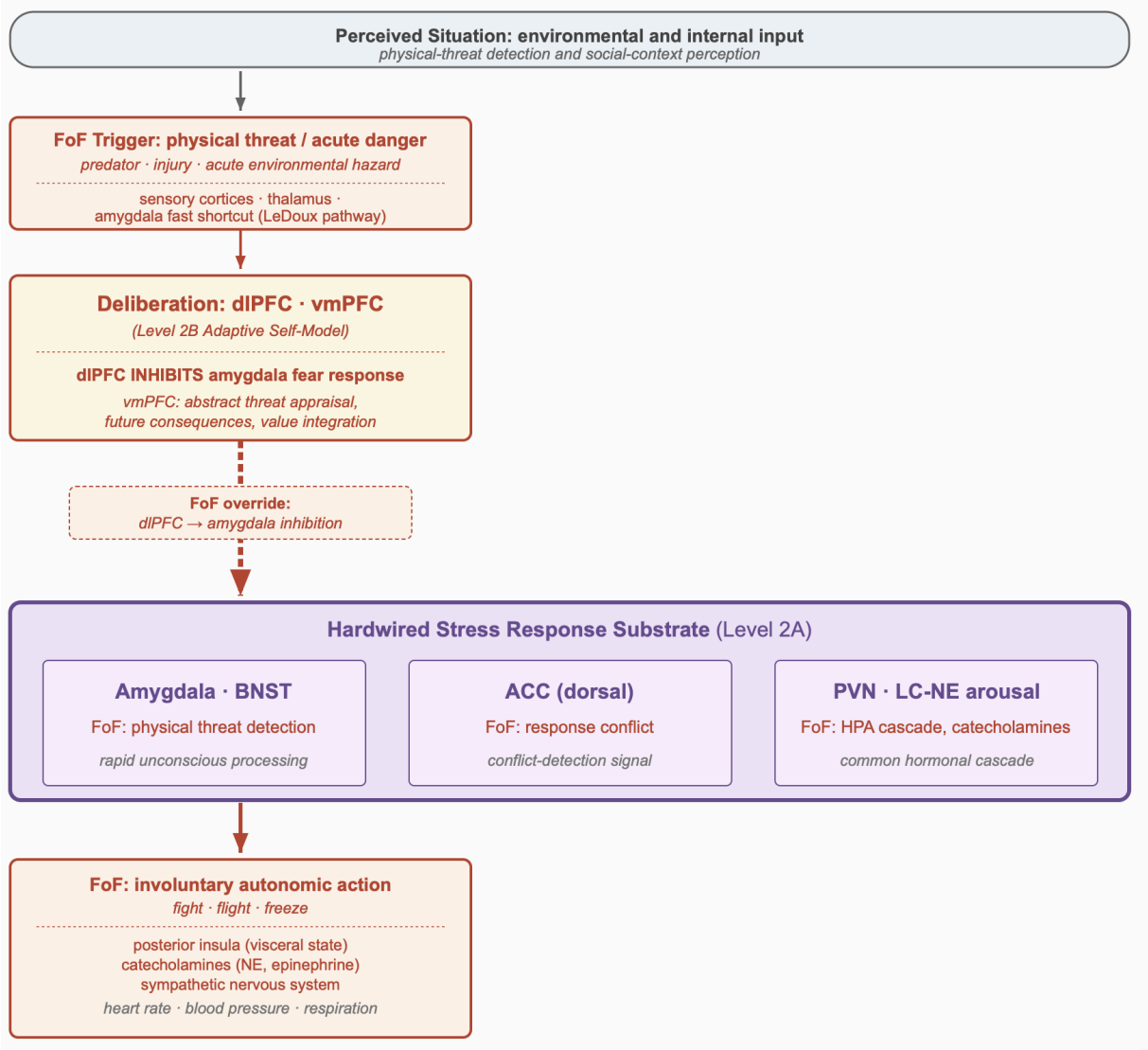

Figure 1. Neural implementation of the fight-or-flight response. Vertical stress pathway (Level-2A: amygdala threat detection → PVN/HPA cascade → hardwired sympathetic response, right column) with inhibitory override (Level-2B: dlPFC + vmPFC deliberation) that reduces fear when conditions permit deliberation. The shared Hardwired Stress Response Substrate (Level 2A, purple) is the response substrate that integrates both the FoF activation pathway shown here and the social copying override shown in Fig. 2.

2.2 Organismal Requirements

The FoF response requires specific anatomical and physiological structures that define vertebrate biology.

Neural Crest Cells. The fundamental requirement is the neural crest, an embryonic stem cell population unique to vertebrates that emerged at the origin of the vertebrate lineage. Neural crest cells migrate from the developing spinal cord and differentiate into sympathetic neurons, adrenal chromaffin cells, and sensory ganglia. (Bronner & LeDouarin, 2012; Edens et al., 2024).

Sympathetic Nervous System. Derived from neural crest cells, the sympathetic nervous system mediates the FoF response through sympathetic chain ganglia containing noradrenergic neurons; catecholamine synthesis pathways (particularly tyrosine hydroxylase and dopamine β-hydroxylase) to produce norepinephrine and epinephrine; adrenal/interrenal chromaffin tissue for stress hormone secretion; and the hypothalamic–pituitary–adrenal (HPA) axis for neuroendocrine coordination. (Alshak & Das, 2025; Denver, 2009; Kvetnansky et al., 2009; Reid et al., 1998; Scott-Solomon et al., 2021)

Brain Structures. The response requires the amygdala (threat detection), hypothalamus (coordinating autonomic and endocrine outputs), and brainstem centers regulating sympathetic outflow (Janak & Tye, 2015; Phelps & LeDoux, 2005; Totah et al., 2018; Ulrich-Lai & Herman, 2009). In mammals, these subcortical structures interact with cortical regions that can modulate, delay, or in some contexts suppress FoF responses (Hartley & Phelps, 2010; Motzkin et al., 2015).

2.3 Evolutionary Depth: From Lampreys to Mammals

Recent research dramatically revises the understanding of how ancient the FoF system is.

Jawless Vertebrates (~550 MYA). A 2024 study overturned a century of scientific consensus by discovering sympathetic neurons in sea lampreys (*Petromyzon marinus*), the most primitive living vertebrates (Edens et al., 2024). Lampreys possess sympathetic neurons expressing catecholamine-synthesizing enzymes, neural crest-derived sympathoblasts, a rudimentary sympathoadrenal system capable of norepinephrine production, and the core transcriptional program (ASCL1, PHOX2, HAND genes) characteristic of sympathetic function, pushing the sympathetic nervous system—and by extension FoF—back to the earliest vertebrates (Edens et al., 2024).

Jawed Vertebrates. All jawed vertebrates (fish, amphibians, reptiles, birds, mammals) possess elaborated sympathetic nervous systems with extensive sympathetic chain ganglia and chromaffin tissue (Goldstein, 2025; Reid et al., 1998). The HPA (or analog HPI) axis shows remarkable conservation despite different anatomical arrangements, indicating early stabilization of the core stress circuitry (Denver, 2009; Herman et al., 2003; Herman & Tasker, 2016).

Invertebrates—Limited Analogues. Invertebrates lack the vertebrate-specific neural crest and sympathetic nervous system but exhibit stress responses using different molecular systems. Many use octopamine as their primary stress neurohormone rather than norepinephrine; these represent convergent evolution of stress responses rather than homologous FoF systems. (Adamo, 2017; Adamo & Baker, 2011; Ottaviani & Franceschi, 1996)

2.4 Evolutionary Innovations

While the basic FoF machinery dates to early vertebrates, it underwent significant elaboration. Epinephrine biosynthesis (requiring PNMT) appears only in jawed vertebrates, adding a potent systemic catecholamine signal to the stress response (Kvetnansky et al., 2009). Bone-derived osteocalcin was discovered to be essential for the acute stress response in bony vertebrates, revealing an unexpected skeletal contribution to FoF physiology (Berger et al., 2019). Cortical brain regions expanded in mammals, adding cognitive appraisal and flexible regulation to primitive reflexive responses (Hartley & Phelps, 2010; Yu et al., 2023). HPA-axis refinements allowed anticipatory responses to predicted threats, making stress responses partially preparatory rather than purely reactive (Herman & Tasker, 2016; Schulkin, 2009).

Functional Significance. The FoF response exemplifies how evolution builds complex behaviors on ancient neural scaffolding. Modern humans retain this ~550-million-year-old emergency system, which still activates during psychological stressors like public speaking despite no physical threat,

reflecting the evolutionary principle that once established, core stress circuits are highly conserved (Cannon, 1915; Denver, 2009; Sapolsky, 2004).

3. Social Copying Under Stress: The Collective Survival Response

Just as the fight-or-flight response represents an ancient, neurologically embedded survival system present at the dawn of vertebrate evolution, mounting evidence suggests that social copying under *coordination stress* (uncertainty about norms, group threat, coordination demands) constitutes a fundamental behavioral feature of social organisms—one that appears across evolutionary scales from the most primitive social entities to advanced human societies, and may in its simplest biochemical forms predate vertebrate fight-or-flight (Loomis, 2014; Mukherjee & Bassler, 2019; Sgro et al., 2015; Strassmann & Queller, 2011; Whiteley et al., 2017; Yamamoto & Vernier, 2011). This social conformity response, like fight-or-flight, operates through specific neural or signaling mechanisms and, critically, can override individual rational decision-making in ways that enhance the survival probability of the collective group (Baddeley, 2010; Bond & Smith, 1996; Overgaauw et al., 2019; Toelch & Dolan, 2015; Wu et al., 2016).

3.1 Evolutionary Status: Ancient and Conserved

Across taxa, social copying under coordination stress is better understood as an evolutionarily conserved strategy that repeatedly emerges wherever organisms aggregate and must coordinate behavior (Mann, 2020; Mukherjee & Bassler, 2019; Strassmann & Queller, 2011). The parallel to fight-or-flight is striking: both represent partly innate, rapid responses to environmental challenges—physical threats in one case, informational or social uncertainty in the other—that natural selection has embedded into nervous system or signaling architecture because they confer survival advantages (Beaumont et al., 2009; Goodnight, 2005; Simons, 2011; Starrfelt & Kokko, 2012; Tufto, 2015).

The fundamental social copying logic mirrors fight-or-flight. Individual organisms facing uncertainty about optimal choices—where to forage, how to respond to ambiguous threats, when to aggregate—often achieve better outcomes by pooling information through social copying than by relying solely on individual, potentially error-prone assessments (Mann, 2020; Toelch & Dolan, 2015). This "wisdom of crowds" phenomenon appears even in very simple social systems, where local copying rules produce group-level decisions that are more accurate or more robust than those of most individuals (Beaumont et al., 2009; Popat et al., 2015; Simons, 2011).

3.2 Evolutionary Depth: Examples from Bacteria to Humans

3.2.1 Bacteria: Quorum Sensing as Collective Sensing Under Uncertainty

Even more primitive than amoebae, bacteria employ quorum sensing—a system where individual cells release and detect autoinducer molecules to assess population density and environmental conditions (Mukherjee & Bassler, 2019; Whiteley et al., 2017). Recent theoretical and experimental work reinterprets quorum sensing not merely as density estimation but as collective environmental sensing under uncertainty: individual bacteria have noisy, imperfect estimates; by secreting autoinducers encoding their assessments and sensing the pooled concentration, bacteria effectively aggregate each other's information, allowing the population to make more reliable decisions about costly functions such as biofilm formation, virulence factor expression, and antibiotic resistance (Bruger & Waters, 2016; Popat et al., 2015). In this light, quorum sensing can be seen as an proto biochemical implementation of social copying under stress or uncertainty, where individual autonomy is partially subordinated to population-level decisions (Beaumont et al., 2009; Mukherjee & Bassler, 2019; Whiteley et al., 2017).

3.2.2 Social Amoebae: The Foundation of Stress-Induced Collective Behavior

The most compelling evidence for the primitiveness of stress-induced social copying comes from *Dictyostelium discoideum*, the social amoeba (commonly known as slime mold)—a unicellular organism that transforms from independent cells into coordinated multicellular aggregates specifically under starvation stress (Loomis, 2014; Strassmann & Queller, 2011). When *Dictyostelium* cells experience nutritional stress, they undergo a dramatic behavioral transformation driven by cyclic AMP (cAMP) signaling: individual cells begin producing and releasing cAMP in response to starvation; neighboring cells detect extracellular cAMP and copy this behavior; this creates propagating waves of cAMP signaling that coordinate movement of up to 100,000 cells toward aggregation centers; cells synchronize through collective oscillations; and the stressed collective forms multicellular structures in which roughly 20% of cells sacrifice themselves while ~80% survive as spores (Gregor et al., 2010; Loomis, 2014; Sgro et al., 2015).

This system can be interpreted as an early form of social copying under stress where some individuals self-sacrifice for the collective survival: it is triggered by uncertainty and starvation, uses chemical signaling for coordination, entails individual sacrifice for collective survival, and implements bet-hedging in which the population partitions into distinct fates (Beaumont et al., 2009; Simons, 2011; Strassmann & Queller, 2011). Critically, up to ~15% of cells remain unicellular — an evolved bet-hedging strategy (§6.1) (Dubravcic et al., 2014).

3.2.3 Social Insects: Pheromone-Based Coordination

Social insects—ants, bees, termites—represent intermediate evolutionary complexity with sophisticated pheromone-based copying systems (Boomsma & Gawne, 2018; Valentini et al., 2020). Alarm pheromone cascades in social insects provide a clear example: when a honeybee stings an intruder ensuring its own demise, it releases isoamyl acetate, recruiting nearby workers to the threat and escalating a coordinated defense response (Bouchebti & Arganda, 2020). Swarms and foraging parties coordinate through stigmergy and pheromone trails; under predation stress or resource scarcity, social insects increase coordination and sacrifice individual autonomy for collective survival, functioning at the colony level as superorganisms (Boomsma & Gawne, 2018; Valentini et al., 2020).

3.2.4 Fish Schools: Social Learning Under Predation

Fish provide clear evidence that vertebrates employ social copying, particularly under predation stress. Guppies and other small fishes from high-predation habitats show stronger schooling tendencies and greater reliance on social information than those from low-predation environments (Seghers, 1974; Webster & Laland, 2017). Fish schools use visual cues with light intensity and group composition modulating social interaction strength—copying is strongest under ambiguous or high-risk conditions—and social learning experiments demonstrate that fish preferentially copy others when facing uncertainty about food locations or threats (Brown et al., 2006; Webster, 2023). These patterns illustrate how stress and uncertainty shift the balance from individual exploration toward social copying in vertebrate groups (Mann, 2020; Seghers, 1974).

3.2.5 Mammals and Humans: The Neural Conformity Circuit

In mammals, particularly primates and humans, social copying under stress and uncertainty operates through highly conserved neural circuits that parallel the fight-or-flight system in their automaticity and their capacity to bias or sometimes override deliberative judgment (Lin et al., 2018; Overgaauw et al., 2019; Stallen & Sanfey, 2015; Wu et al., 2016). See Fig. 2.

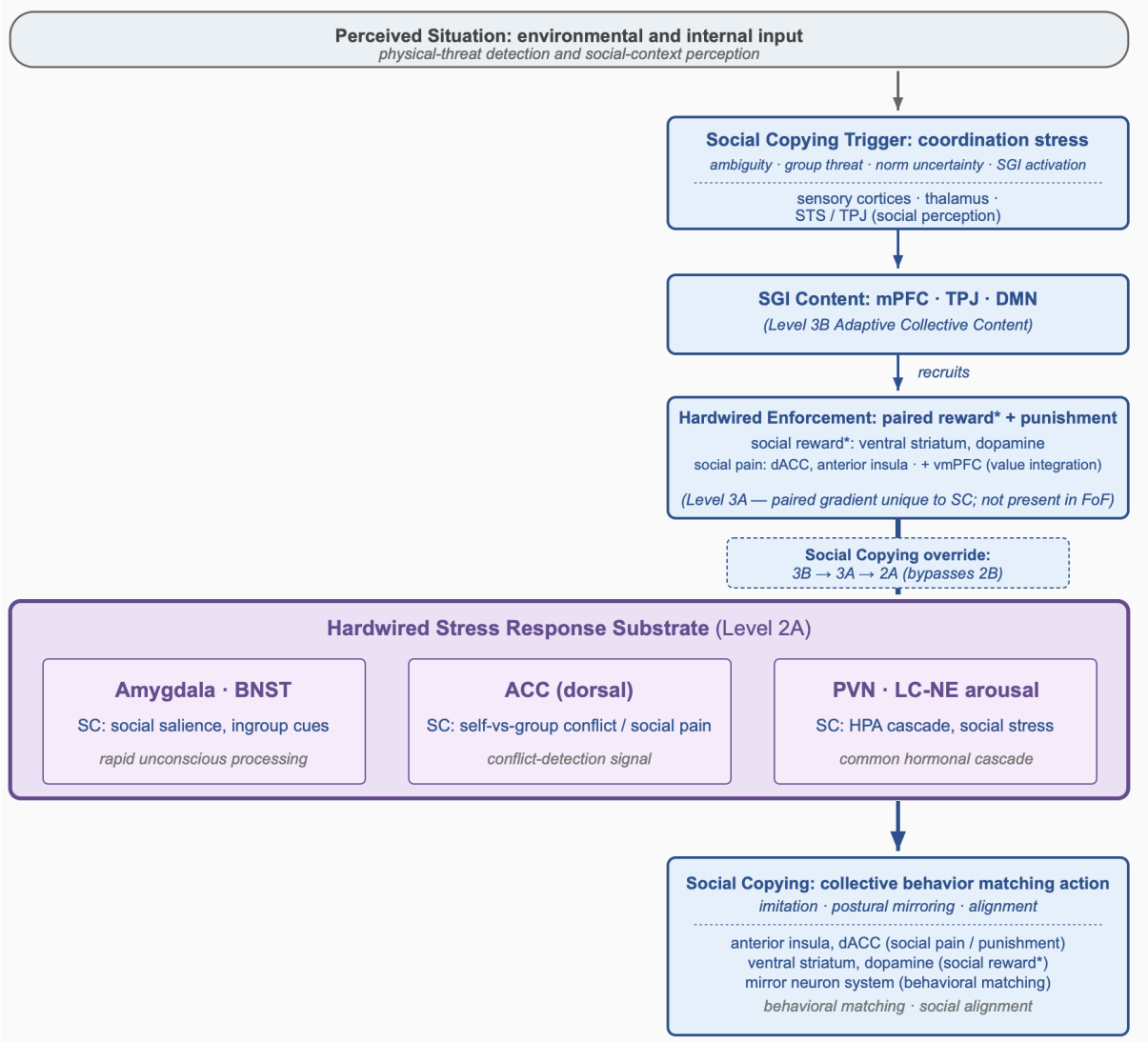

Figure 2. Neural implementation of social copying under coordination stress. Vertical Level-3 → Level-2A descending recruitment pathway: coordination stress engages SGI content (Level 3B: mPFC, TPJ, DMN), which recruits hardwired enforcement (Level 3A: *paired-gradient* of social reward via ventral striatum + dopamine and social punishment via dACC + anterior insula), which operates through the same Hardwired Stress Response Substrate (Level 2A, purple) — bypassing Level-2B deliberation entirely. *Social reward and punishment developed in §3.3 and §7.3 (ventral striatum, dopamine) is unique to social copying; FoF (Fig. 1) has no comparable intrinsic reward channel.

Ventromedial prefrontal cortex (vmPFC) and ventral striatum (VS). These regions process social rewards and track alignment between individual judgments and group consensus. When individuals conform to group opinions, ventral striatum and vmPFC show increased activity, providing positive reinforcement for social copying and, in some cases, reflecting genuine changes in subjective value (Chen et al., 2023; Fliessbach et al., 2007; Ruff & Fehr, 2014; Zaki et al., 2011).

Anterior cingulate cortex (ACC) and insula. These regions detect conflict between individual assessment and group consensus, generating negative affect and arousal that motivate conformity; activity increases when individuals deviate from group norms, creating discomfort that drives copying behavior. (Botvinick et al., 2004; Kerns et al., 2004)

Amygdala. The amygdala tracks social salience and the emotional significance of group alignment, with activation increasing during conformity to ingroup members and during social-evaluative threat. It signals the motivational relevance of social information and interacts with both valuation and control circuits. (Bickart et al., 2012, 2014; Janak & Tye, 2015; Phelps & LeDoux, 2005)

Dorsolateral prefrontal cortex (dlPFC). dlPFC is involved in the cognitive control required to override individual preferences when conforming and, conversely, to resist social pressure when maintaining independence; trusting outgroup members requires more dlPFC activation than trusting ingroup members, suggesting ingroup copying is more automatic. (Hartley & Phelps, 2010; Huang et al., 2014; Motzkin et al., 2015)

Mirror-related perception–action coupling. Distributed across premotor, motor, and parietal areas, mirror-like mechanisms support automatic imitation and behavioral synchronization with conspecifics, providing one motor pathway for rapid social matching and coordination. (Bonini et al., 2022; Gallese, 2009; Iacoboni, 2009; Rizzolatti & Fogassi, 2014)

These evolutionary-conserved neural conformity circuits must coordinate to achieve the desired survival of the collective: remarkably achievable in the simpler evolutionary environments but potentially failing in more complex environments.

3.3 Social Copying Overrides Individual Rationality

Behavioral evidence. Asch's classic conformity experiments show that roughly one-third of individuals conform to obviously incorrect group judgments, even when the correct answer is perceptually obvious (Bond & Smith, 1996). Time pressure increases conformity, indicating that copying is a default response that requires cognitive control to resist (Cone & Rand, 2014). Monetary incentives reduce but do not eliminate conformity errors, demonstrating that social pressure can override even financial self-interest (Bond & Smith, 1996). Herd behavior in financial markets can create bubbles and crashes as individuals abandon private information and copy the crowd (Baddeley, 2010; Baddeley et al., 2012; Bharti et al., 2025; Bharti & Kumar, 2022).

Paired gradient: reward of alignment, pain of misalignment. The brain enforces social conformity through a coupled carrot-and-stick architecture, developed in detail at §7.3. On the reward side, conformity activates ventral striatum and vmPFC even when the individual privately disagrees, with social influence modulating value representation so that group opinions change how individuals neurally encode options — changes that track subsequent behavioral shifts toward group norms (Chen et al., 2023; Klucharev et al., 2009; Ruff & Fehr, 2014). On the punishment side, the dACC and anterior insula respond robustly to social exclusion and non-alignment in a pattern overlapping with the affective component of physical pain, providing a negative-valence signal that discourages sustained independence — noted is controversy about whether this ACC activation represents "pain" versus salience or conflict detection (Eisenberger, 2012; Rotge et al., 2015). Reinforcement-learning models show that this paired gradient operates as a coupled learning signal: prediction errors for social agreement (carrot) and disagreement (stick) jointly drive updating toward group consensus, so that social copying is sustained not just because conformity feels good, but because persistent divergence neurally and phenomenologically hurts (Klucharev et al., 2009; Toelch & Dolan, 2015).

3.4 Evolutionary Depth of Social Copying

At each level (Table 1)—bacteria, social amoebae, insects, fish, and mammals—the system exhibits four recurrent features: (1) stress or uncertainty as the trigger, (2) chemical or neural signaling for coordination, (3) automatic or strongly biased copying that can override individual assessments, and (4) collective survival benefits that can exceed individual costs.

Table 1. Summary of social copying mechanisms across evolutionary scales, showing a conserved pattern of stress-triggered collective coordination.

Organism	Stress Signal	Copying Mechanism	Collective Benefit	References
Bacteria	Resource depletion	Autoinducer molecules (quorum sensing)	Coordinated gene expression, antibiotic resistance	(Mukherjee & Bassler, 2019; Popat et al., 2015)
Social amoebae	Starvation	cAMP oscillations and chemotaxis	Multicellular aggregation, spore dispersal	(Loomis, 2014; Strassmann & Queller, 2011)
Insects	Predation, resource scarcity	Pheromone trails and stigmergy	Optimal foraging, nest defense	(Bouchebti & Arganda, 2020; Valentini et al., 2020)
Fish	Predation threat	Visual coordination, schooling	Predator confusion, group vigilance	(Brown et al., 2006; Seghers, 1974; Webster & Laland, 2017)
Mammals / Humans	Social uncertainty, threat	Neural conformity circuits (vmPFC, ACC, amygdala)	Group cohesion, cultural transmission, collective decisions	(Lin et al., 2018; Overgaauw et al., 2019; Stallen & Sanfey, 2015)

4. Neurological Comparison: Shared Architecture, Distinct Functions

The fight-or-flight and social copying responses share a surprising amount of common neural architecture, reflecting their parallel evolutionary functions as survival systems—one for individual physical threats, the other for collective social challenges. At the same time, key differentiations reveal how the same brain structures can be repurposed for fundamentally different adaptive purposes, depending on whether the relevant "threat" is to the individual body or to the social group.

4.1 Shared Core Architecture: Ancient Stress Circuitry

Both fight-or-flight and social copying responses rely on ancient subcortical stress systems that operate largely automatically and can override deliberative reasoning. See Fig. 3.

Amygdala. The amygdala functions as a primary threat detector in both systems but processes different content. For fight-or-flight, it detects physical threats through rapid, unconscious processing, projecting to hypothalamic and brainstem nuclei that initiate autonomic and endocrine responses (Janak & Tye, 2015; Phelps & LeDoux, 2005). For social copying, the same structure encodes social salience, evaluates ingroup versus outgroup cues, and participates in the emotional evaluation of social alignment or exclusion (Bickart et al., 2012, 2014). In both contexts, amygdala activation can precede and bias conscious appraisal, making it a shared entry point for physical and coordination stress cascades (Ulrich-Lai & Herman, 2009).

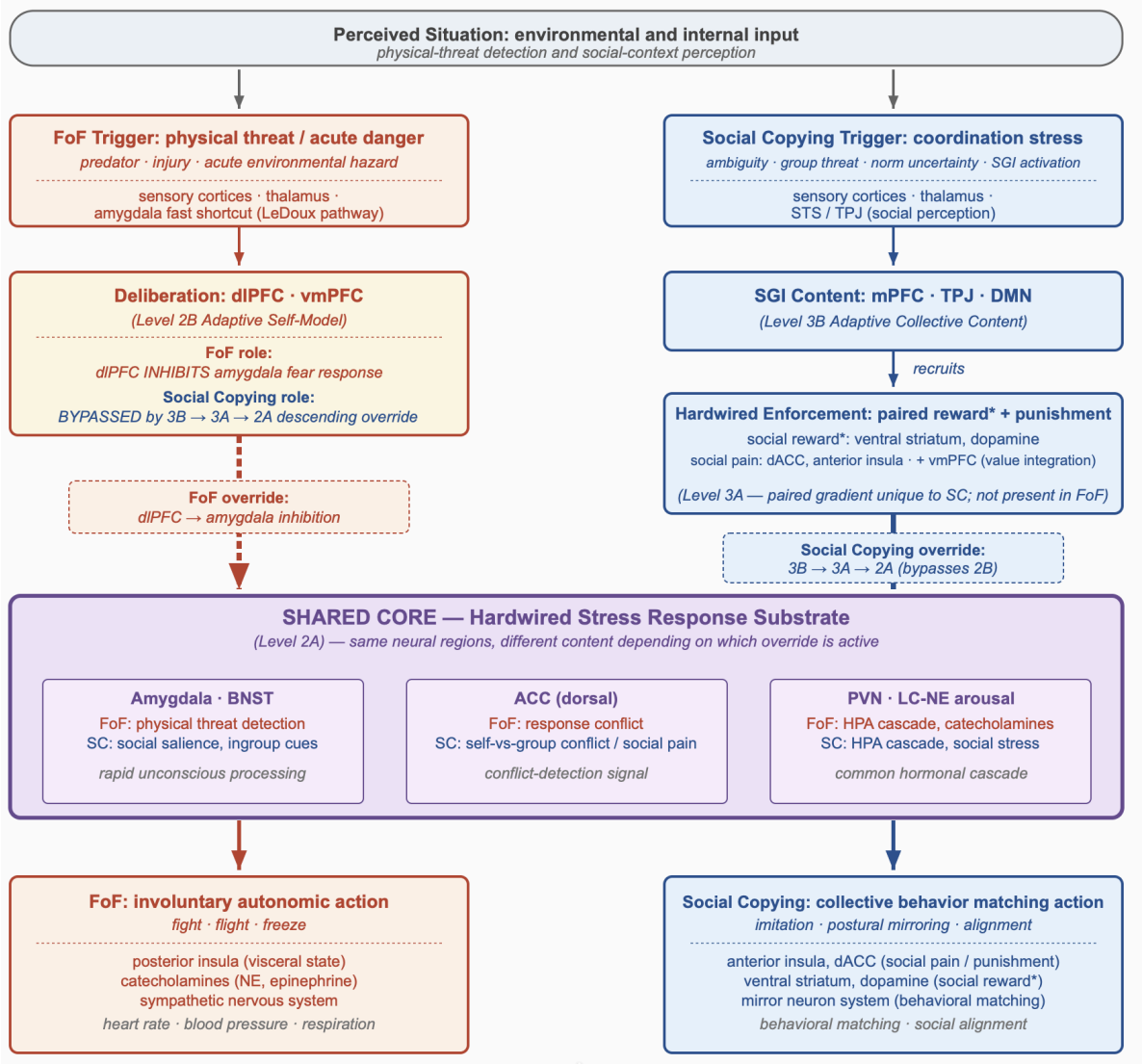

Figure 3. Shared architecture, inverted control logic. Fight-or-Flight and Social Copying use overlapping neural substrates (purple SHARED CORE) with distinct override mechanisms: the FoF dlPFC → amygdala inhibitory override (left, red) and the Social Copying 3B → 3A → 2A descending recruitment override (right, blue). *Social reward (ventral striatum, dopamine) appears only in the Social Copying pathway (Hardwired Enforcement box and the SC output box). FoF has no comparable intrinsic paired reward + punishment gradient.

Anterior Cingulate Cortex (ACC). The dorsal ACC contributes to conflict detection in both systems but monitors different types of conflict. In fight-or-flight, it detects response conflict—competing motor or action tendencies—and signals the need for increased control (Botvinick et al., 2001, 2004; Kerns et al., 2004). In social copying, ACC activity tracks the discrepancy between an individual's judgment and group consensus—self-versus-group conflict—and overlaps with the pattern seen in social rejection tasks, where exclusion or non-alignment reliably activates dACC in a way that predicts subjective distress (Eisenberger, 2012; Rotge et al., 2015). This ACC-mediated "social pain" signal is the punishment limb of the paired-gradient enforcement developed at §7.3 (Eisenberger, 2012; Rotge et al., 2015).

Paraventricular Nucleus of the Hypothalamus (PVN). PVN represents the apex of the stress response for both behaviors. It initiates HPA axis activation in response to physical stressors and social-evaluative stressors, releasing corticotropin-releasing hormone (CRH) that drives pituitary ACTH and adrenal glucocorticoid secretion (Herman et al., 2003; Herman & Tasker, 2016; Ulrich-Lai & Herman, 2009). Thus, both FoF and social copying ultimately converge on a common hormonal cascade (PVN → pituitary → adrenal), differing more in upstream triggers and downstream targets than in core endocrine machinery (Godoy et al., 2018; Het et al., 2005).

Locus Coeruleus–Norepinephrine (LC–NE) System. This brainstem arousal system activates during both responses. LC neurons release norepinephrine throughout cortex and subcortex during physical and social stress, modulating arousal, alertness, and vigilance (Totah et al., 2018; Ulrich-Lai & Herman, 2009). LC hyper-responsivity predicts vulnerability to stress-related pathology, and catecholaminergic modulation of prefrontal circuits influences both rapid defensive reactions and the regulation of social decisions under uncertainty (Chandler et al., 2014; Kvetnansky et al., 2009).

Together, these structures form a shared stress-detection and arousal core that can be recruited either for individual-level FoF responses or for social-level copying and conformity. The primary differences between the two systems emerge in how this core is coupled to valuation, reward, and motor-output circuits (Ruff & Fehr, 2014; Wu et al., 2016).

4.2 Critical Differentiations

Despite their shared infrastructure, the two systems diverge in crucial ways that reflect their distinct evolutionary fitness purposes. See Fig. 3.

4.2.1 Insula Regionalization: Physical vs. Emotional Interoception

The insula exhibits a posterior-to-anterior gradient. Fight-or-flight primarily involves the posterior and mid-insula, which process physical visceral states such as heart rate, breathing, and pain (Gasquoine, 2014; Simmons et al., 2013). Social copying more strongly recruits the anterior insula (AIC), which integrates interoceptive signals with emotional and social context to generate feelings such as embarrassment, guilt, and social pain during rejection or non-alignment with others (Eisenberger, 2012; Simmons et al., 2013). Together with dACC, the AIC helps implement an aversive, pain-like response to social misalignment that motivates individuals to restore conformity to group norms (Eisenberger, 2012; Rotge et al., 2015).

4.2.2 Dorsolateral Prefrontal Cortex (dlPFC): Divergence in Control Logic

The dlPFC plays almost opposite control roles in the two systems. In fight-or-flight, dlPFC and related prefrontal regions regulate amygdala-driven fear responses through inhibitory pathways, allowing deliberative assessment to override automatic reactions when conditions permit (Chandler et al., 2014; Hartley & Phelps, 2010; Motzkin et al., 2015). In social copying, dlPFC is required to resist conformity: ingroup alignment and copying are relatively automatic, whereas maintaining independent judgments—especially when they conflict with group norms—demands active control (Huang et al., 2014; Toelch & Dolan, 2015; Wu et al., 2016). This suggests that fight-or-flight is typically inhibited to allow rational behavior, whereas social copying is typically resisted to maintain individual judgment, an “inverted but parallel” control logic (Chen et al., 2023; Hartley & Phelps, 2010; Huang et al., 2014).

4.2.3 Ventromedial Prefrontal Cortex (vmPFC): Abstract Threat vs. Dual Value Coding

In fight-or-flight, vmPFC contributes to the appraisal of future and abstract threats, integrating long-term consequences into value computations; damage impairs evaluation of delayed outcomes while leaving immediate defensive reactions relatively intact (Bechara et al., 2000; Hartley & Phelps, 2010). In social copying, vmPFC exhibits dual coding patterns in social influence tasks, with activity scaling both with subjective value (personal preference) and with normative value (alignment with others), and shifting as social information changes (Ruff & Fehr, 2014; Zaki et al., 2011). This opponent or multiplexed coding allows the same region to represent conflicts between personal desires and social norms, providing a plausible substrate for social-value reweighting during conformity (Chen et al., 2023; Konovalov et al., 2018).

4.2.4 Primary Neurotransmitters: Catecholamines vs. Dopamine

The systems also differ in their primary neuromodulators. Fight-or-flight is dominated by catecholamines—norepinephrine and epinephrine—released via sympathetic and adrenal pathways, preparing the body for immediate physical action (Godoy et al., 2018; Kvetnansky et al., 2009; Ulrich-Lai & Herman, 2009). Social copying is centered on dopamine in ventral striatum and related circuits, with dopaminergic signals tracking social reward, social prediction errors, and value updates induced by group feedback (Barron et al., 2010; Fliessbach et al., 2007; Klucharev et al., 2009; Ruff & Fehr, 2014; Yamamoto & Vernier, 2011). Although both systems may engage multiple neuromodulators, the balance between catecholaminergic arousal and dopaminergic social reward is markedly different (Chandler et al., 2014; Schultz, 2007; Wise, 2004).

4.2.5 Reward Feedback: Outcome vs. Process

In fight-or-flight, "reward" is primarily outcome-based: successful threat elimination or escape reduces stress and returns the organism toward homeostasis, but the acute stress response itself is not intrinsically rewarding (Godoy et al., 2018; Sapolsky, 2004). In social copying, the behavior itself often carries intrinsic reward: conforming to group norms activates ventral striatum and vmPFC even when material outcomes are unchanged or objectively disadvantageous, indicating that alignment with others is treated as valuable in its own right (Chen et al., 2023; Fliessbach et al., 2007; Ruff & Fehr, 2014; Zaki et al., 2011). This shift from outcome reward to process reward is central to understanding how social copying can persist even when it conflicts with individual instrumental interests (Baddeley, 2010; Toelch & Dolan, 2015).

4.2.6 Motor/Behavioral Output

Fight-or-flight primarily engages involuntary autonomic and somatic outputs: the sympathetic nervous system increases heart rate, blood pressure, respiration, muscle tone, and glucose mobilization, preparing the body for rapid physical action (Denver, 2009; Kvetnansky et al., 2009; Reid et al., 1998). Social copying, by contrast, relies more on voluntary (though often automatic) motor systems that implement behavioral matching, such as postural mirroring, gaze following, and imitative actions (Bonini et al., 2022; Rizzolatti & Fogassi, 2014). Mirror-related perception–action coupling provides one candidate pathway for rapid synchronization with conspecifics, even though the broader social copying response likely depends on more distributed motor and associative networks (Bonini et al., 2022).

4.3 Neural Component Comparison

The result (Table 2) is a pair of parallel survival architectures: one tuned to immediate physical danger for individual bodies, the other tuned to social uncertainty and threats to the collective entity (Chen et al., 2023; Ruff & Fehr, 2014; Ulrich-Lai & Herman, 2009).

Table 2 Summary of commonalities and differences, highlighting how a shared stress-detection core (amygdala, ACC, PVN, LC) is coupled to distinct valuation, neuromodulatory, and motor subsystems in fight-or-flight versus social copying.

Neural Component	Fight-or-Flight	Social Copying	Common (C) vs. Differentiated (D)
Primary Threat Detection	Amygdala (fear/threat detection, rapid unconscious processing)	Amygdala (social threat detection, ingroup/outgroup salience)	C: Same structure, different content
Conflict Detection	Dorsal ACC (response conflict monitoring)	ACC (self vs. group conflict detection)	C: Same structure, different conflicts
Interoceptive Awareness	Posterior insula (visceral/bodily state)	Anterior insula (emotional awareness, social interoception)	D: Posterior (physical) vs. Anterior (social)
Command Center	PVN—HPA/SAM axis initiation	PVN—stress under social uncertainty	C: Same nucleus, both stressors
Brainstem Arousal	Locus coeruleus (NE surge)	Locus coeruleus (arousal during social stress)	C: Both activate LC under stress
Cognitive Control	dlPFC inhibits amygdala response	dlPFC required to RESIST conformity	D: Inhibits FoF; resists social copying
Value/Reward	vmPFC appraises future/abstract threat	vmPFC encodes subjective AND normative value (dual coding)	D: Single vs. dual value coding
Primary Neurotransmitter	Catecholamines (NE, epinephrine)	Dopamine (ventral striatum, NAcc)	D: Physical action vs. social reward
Reward Structure	Outcome-based (escape/elimination)	Process-based (conforming IS the reward)	D: Outcome vs. behavior reward
Motor Output	Sympathetic NS (involuntary autonomic)	Mirror neuron system (automatic imitation)	D: Autonomic vs. voluntary motor

5. The Immunity Framework: Individual and Collective Survival

The parallels between fight-or-flight and social copying become most theoretically coherent when taking an immunity perspective within a multi-level evolutionary framework (Boomsma & Gawne, 2018; Goodnight, 2005; Simons, 2011; Wilson & Wilson, 2007), in which "immunity" refers to any mechanism that protects the "self" from threats at a given level of biological organization (Johnson, 2026). In this *Immunity Framework*, immune-like systems exist at multiple levels, from molecular and cellular defenses up through individual organisms and social collectives, and can be compared in terms of the units they protect, the threats they detect, and the mechanisms they deploy (Strassmann & Queller, 2011, 2014).

Within this lens, fight-or-flight maps onto **Level 2A individual innate immunity**: a defense against acute physical threats, triggered by amygdala detection, mediated by catecholamines and the sympathetic nervous system, and capable of overriding prefrontal deliberation to ensure rapid survival-oriented action (Cannon, 1915). Social copying under coordination stress, by contrast, maps onto **Level 3 collective immunity**: a defense of the social organism against threats to collective survival and cohesion. Mechanistically, social copying implements a *Level-3 → Level-2A override*: Level-3 social-identity content (mPFC, TPJ, DMN) recruits Level-3A enforcement (ACC social pain + ventral striatum reward, the paired-gradient developed at §7.3) that operates through the Level-2A response substrate (amygdala, PVN/LC-NE, sympathetic outflow), bypassing Level-2B deliberation (dlPFC, vmPFC) entirely — a structurally distinct override pathway from the Level-2B-inhibits-Level-2A control logic of FoF (§4.2.2). The Level-3→Level-2A override is triggered by coordination stress (amygdala plus ACC), mediated by dopamine and perception–action coupling circuits, and capable of overriding or biasing individual deliberation to secure coordinated group responses (Mukherjee & Bassler, 2019; Strassmann & Queller, 2011; Toelch & Dolan, 2015).

The structural parallel can be summarized schematically: fight-or-flight detects threats to individual survival (e.g., predators, physical danger) and mobilizes bodily resources for escape or defense; social copying detects threats to collective integrity (e.g., ambiguity about norms, external group threats) and mobilizes behavioral alignment to maintain cohesion (Boomsma & Gawne, 2018; Goodnight, 2005; Wilson & Wilson, 2007). In both cases, stress-sensitive circuitry "hijacks" deliberative control to enforce a canonical response at an appropriate level of organization, trading some local optimality for robustness at the protected level (Beaumont et al., 2009; Sapolsky, 2004; Simons, 2011).

Individual Innate Immunity (Fight-or-Flight). At this level (Level 2A), the focal unit is the individual organism. Threats are typically physical (predators, injury, acute environmental hazards), detection is implemented by fast subcortical systems (amygdala, brainstem, PVN), and the effector mechanisms include sympathetic outflow, HPA activation, and rapid motor responses (Herman & Tasker, 2016; Ulrich-Lai & Herman, 2009). The evolutionary payoff is straightforward: individuals that rapidly mobilize FoF responses have higher survival probability in the face of acute dangers, even if those responses sometimes misfire in modern environments (Godoy et al., 2018; Sapolsky, 2004).

Collective Immunity (Social Group Identity). At this level (Level 3), the focal unit is the social group or "superorganism" (Boomsma & Gawne, 2018; Strassmann & Queller, 2011). Threats include not only external competitors or predators but also internal sources of fragmentation such as norm defection and coordination failure (Goodnight, 2005; Wilson & Wilson, 2007). Detection mechanisms involve circuits sensitive to social uncertainty, norm conflict, and group threat—amygdala and ACC for conflict and salience; anterior insula for social discomfort; and vmPFC/striatum for social value (FeldmanHall & Shenhav, 2019; Wu et al., 2016). Effector mechanisms include conformity, ingroup alignment, and sometimes outgroup exclusion,

implemented via dopaminergic reward signals and perception–action coupling that bias behavior toward group norms (Iacoboni, 2009; Klucharev et al., 2009; Ruff & Fehr, 2014; Zaki et al., 2011).

The broad analogy comparing and contrasting the two evolutionary systems can be summarized as follows:

- *Threat detected*: physical danger to the organism vs. social uncertainty or threat to the group (FeldmanHall & Shenhav, 2019; Martinez-Saito & Gorina, 2022).
- *Detection mechanisms*: amygdala-centered circuits monitoring physical threat vs. amygdala–ACC–insula circuits monitoring social conflict and uncertainty (Janak & Tye, 2015; Phelps & LeDoux, 2005).
- *Primary neuromodulators*: catecholamines (norepinephrine, epinephrine) vs. dopamine-dominated social reward and prediction-error signals (Fliessbach et al., 2007; Kvetnansky et al., 2009; Schultz, 2007; Wise, 2004).
- *What is overridden*: prefrontal deliberation in both cases, but with opposite control logic (inhibiting FoF vs. resisting conformity) (Hartley & Phelps, 2010; Huang et al., 2014; Motzkin et al., 2015; Toelch & Dolan, 2015).
- *Survival unit*: individual organism vs. social group or higher-level collective (Boomsma & Gawne, 2018; Goodnight, 2005; Wilson & Wilson, 2007).
- *Reward structure*: outcome-based escape/elimination vs. process-based conformity reward (Ruff & Fehr, 2014; Sapolsky, 2004; Zaki et al., 2011).
- *Modern maladaptation*: panic attacks and chronic stress vs. polarization, herd behavior, and groupthink (Baddeley, 2010; Bharti & Kumar, 2022; Nesse, 2019; Sapolsky, 2004).

The structural parallel can be summarized as follows:

Feature	FoF Acting at the Individual Level	Social Copying Acting at the Collective Level
Threat detected	Physical danger to organism	Social uncertainty / threat to group
Detection mechanism	Amygdala (rapid unconscious)	Amygdala + ACC (social conflict)
Primary neurotransmitter	Catecholamines (NE, epinephrine)	Dopamine (ventral striatum)
What is overridden	Prefrontal deliberation	Prefrontal deliberation
Survival unit	Individual organism	Social group / collective
Evolutionary depth	~550 MYA (vertebrate SNS)	>1 BYA (bacterial quorum sensing)
Reward structure	Escape/elimination (outcome)	Conformity itself (process)
Modern maladaptation	Panic attacks, chronic stress	Polarization, herd behavior, groupthink

5.1 A Testable Framework: Well-Established, Strongly Suggested, and Hypothesized Components

For clarity, components can distinguish between this framework that are (1) well-established by existing evidence, (2) strongly suggested by converging data, and (3) hypothesized but testable.

5.1.1 Well-established components.

- Fight-or-flight is an evolutionarily ancient (≥550 MYA), largely automatic stress response mediated by sympathetic catecholamines and conserved vertebrate circuitry (amygdala, hypothalamus, sympathetic nervous system) (Cannon, 1915; Denver, 2009; Edens et al., 2024; Ulrich-Lai & Herman, 2009).
- Humans and other social animals show robust social conformity and copying, especially under uncertainty and stress, with consistent neural correlates in vmPFC, ventral striatum, ACC, insula, amygdala, and dlPFC (Baddeley, 2010; Bond & Smith, 1996; Wu et al., 2016).
- Quorum sensing in bacteria, *Dictyostelium* aggregation under starvation, social-insect pheromone coordination, and vertebrate schooling provide multiple examples of stress-linked, signal-mediated coordination that can entail individual sacrifice for group-level benefit (Loomis, 2014; Mukherjee & Bassler, 2019; Popat et al., 2015; Seghers, 1974; Sgro et al., 2015; Strassmann & Queller, 2011).
- Both responses recruit a shared stress-detection core (amygdala, ACC, PVN, LC-NE), developed in §4.1 (Ulrich-Lai & Herman, 2009).

5.1.2 Strongly suggested components.

- Social conformity in humans is partly automatic, engages reward circuitry, and can bias or override explicit deliberation under conditions of uncertainty and stress (Chen et al., 2023; Klucharev et al., 2009; Ruff & Fehr, 2014; Zaki et al., 2011).
- Social copying intensity increases under coordination stress and recruits the same stress-detection core (§4.1) (FeldmanHall & Shenhav, 2019; Wu et al., 2016).
- Across taxa, stress-linked coordination mechanisms function as "collective immunity" strategies that reduce variance and pool information at the group level (§6.1) (Beaumont et al., 2009; Tufto, 2015).
- The dlPFC plays opposite control roles: inhibiting fight-or-flight versus resisting social conformity — an inverted but parallel control architecture (§4.2.2) (Hartley & Phelps, 2010; Huang et al., 2014).

5.1.3 Hypothesized but testable components.

- Social copying constitutes an evolutionarily ancient "Level 3 social immunity" system that is functionally (though not necessarily mechanistically) homologous to individual-level fight-or-flight across diverse taxa, from microbial collectives to human societies (Mukherjee & Bassler, 2019; Strassmann & Queller, 2011); the multilevel-selection mechanism supporting this is developed at §6.1.
- Social copying mechanisms, in some form, predate vertebrate fight-or-flight biochemistry, with origins traceable to early social life such as bacterial quorum sensing and stress-induced aggregation in social amoebae (Loomis, 2014; Sgro et al., 2015; Whiteley et al., 2017).
- The shared neural architecture (amygdala–ACC–PVN–LC plus valuation circuits) exists partly because social copying evolutionarily recruited or "piggybacked on" fight-or-flight infrastructure, reusing ancient stress circuitry for social threats (Chen et al., 2023; Ulrich-Lai & Herman, 2009).

- Modern polarization, financial herding, and groupthink can be modeled as maladaptive expressions of this Level-3 social immunity apparatus in environments mismatched to ancestral conditions, analogous to how panic and anxiety disorders represent misfiring of fight-or-flight systems (Baddeley, 2010; Bharti et al., 2025; Bharti & Kumar, 2022; Nesse, 2019; Sapolsky, 2004).

This framework yields testable predictions: for example, that manipulations enhancing physical stress should increase social conformity via shared PVN–LC pathways, that genetic or pharmacological disruption of shared stress circuits should affect both fight-or-flight and conformity, and that cross-species comparative studies should reveal graded elaboration of social-conformity circuitry tracking social complexity (Herman & Tasker, 2016; Ulrich-Lai & Herman, 2009).

6. Evolutionary Function and Modern Implications

6.1 Group Survival Through Individual Subordination

The adaptive logic of social copying under uncertainty parallels fight-or-flight but operates at the collective level. Social copying can sacrifice individual optimality (e.g., an average of individual fitness) to reduce variance in collective outcomes, functioning as a bet-hedging strategy at the group scale (Beaumont et al., 2009; Simons, 2011; Starrfelt & Kokko, 2012; Tufto, 2015). Under unpredictable environments, bet-hedging strategies that reduce fitness variance outcompete strategies that maximize average fitness but risk occasional catastrophic failure; the same logic applies when groups trade some individual flexibility for greater reliability of coordinated response (Beaumont et al., 2009; Simons, 2011; Tufto, 2015).

Within a multilevel-selection framework, social copying evolves when selection among groups (favoring coordinated, cohesive collectives) overcomes selection within groups (favoring individual defection or idiosyncratic behavior) (Goodnight, 2005; Kramer & Meunier, 2016; Wilson & Wilson, 2007). Groups with strong conformity can coordinate responses to threats, efficiently exploit resources, and maintain social cohesion even when this requires individual sacrifice, whereas groups with weak conformity may fragment or fail under stress (Boomsma & Gawne, 2018; Strassmann & Queller, 2011; Valentini et al., 2020). The superorganism concept in social insects—where colonies function as single organisms with individuals as expendable parts—represents an extreme expression of this principle and illustrates how strong collective immunity can emerge when between-group selection is intense (Boomsma & Gawne, 2018; Strassmann & Queller, 2011).

6.2 Stress as the Shared Trigger

Critically, like fight-or-flight activation under threat, social copying is specifically triggered under stress or uncertainty (uncertainty is treated here as a source of coordination stress; see §3). Uncertainty about correct choices increases reliance on social information and conformity; people and animals rely more on others when private signals are noisy or ambiguous (FeldmanHall & Shenhav, 2019; Mann, 2020; Martinez-Saito & Gorina, 2022; Toelch & Dolan, 2015). Social stress is transmissible: stressed individuals induce physiological and behavioral stress responses in group-mates, creating cascades of arousal that can rapidly synchronize groups in both humans and other social species (Godoy et al., 2018; MacLeod et al., 2023). Fear and anxiety increase conformity and reduce independent judgment, particularly when threat is framed in group-relevant terms (Baddeley, 2010; Bond & Smith, 1996; FeldmanHall & Shenhav, 2019). Ambiguous threat environments increase ingroup favoritism and conformity to ingroup norms, as individuals treat alignment with their group as a protective response (Huang et al., 2014; Lin et al., 2018).

Under coordination stress, the paired-gradient enforcement (§7.3) is operative: dopaminergic reward circuits make alignment with the group feel intrinsically good, while ACC/insula social-pain

circuits make persistent non-alignment feel aversive, jointly biasing individuals toward rapid conformity when coordination demands are high.

Neurologically, coordination stress and physical threat recruit the same shared core (§4.1), suggesting shared circuitry for managing different forms of threat and uncertainty (Wu et al., 2016).

6.3 Modern Maladaptations

Just as the fight-or-flight response can malfunction in modern contexts (panic attacks, chronic stress from non-physical threats), social copying under uncertainty produces maladaptive outcomes when ancient mechanisms encounter novel environments (Nesse, 2019; Sapolsky, 2004).

- *Financial panics and herd behavior*. Contemporary financial markets exhibit irrational copying that creates bubbles and crashes, as individuals abandon private information in favor of crowd behavior reinforced by social and reputational incentives (Baddeley, 2010; Baddeley et al., 2012; Bharti et al., 2025; Bharti & Kumar, 2022).
- *Political polarization*. Ingroup conformity and outgroup derogation amplify under perceived threat, reducing rational evaluation of political information and hardening identity-based divisions (Bond & Smith, 1996; FeldmanHall & Shenhav, 2019).
- *Social media amplification*. Digital environments algorithmically amplify social feedback and visibility, exploiting conformity mechanisms to create viral cascades that bypass reflective judgment and magnify collective misperceptions (Baddeley, 2010; Ruff & Fehr, 2014; Zaki et al., 2011).
- *Groupthink in organizations*. Conformity pressure suppresses dissent and critical thinking in teams and institutions, leading to collective errors that mirror panic-like misfires at the group level (Baddeley, 2010; Goodnight, 2005; Wilson & Wilson, 2007).

These maladaptive phenomena can be viewed as social analogues of anxiety and stress-related disorders—ancient adaptive systems operating in environments they did not evolve for, where mismatches between ancestral selection pressures and modern conditions lead to systematic distortions in both individual and collective decision-making (Bharti et al., 2025; Nesse, 2019; Sapolsky, 2004).

7. Dopamine: The Ancient Neurochemistry of Social Reward

The centrality of dopamine to the social copying response raises the question of its evolutionary origins and its role within a broader enforcement architecture. In social contexts, dopaminergic circuits provide the positive limb of a *paired-gradient* enforcement: alignment with group norms is intrinsically rewarding, while ACC and anterior insula circuits provide the negative limb of social pain when individuals deviate or are excluded (Eisenberger, 2012; Klucharev et al., 2009; Zaki et al., 2011). Dopamine as a neuromodulator that influences behavior is very ancient, arising with the first nervous systems more than 500 million years ago, while the underlying biosynthetic and receptor machinery has deeper roots in non-neuronal organisms (Roshchina, 2010; Yamamoto & Vernier, 2011). Across phyla, dopamine and related catecholamines regulate reward-seeking, motivation, and approach behavior, suggesting that social reward mechanisms in vertebrates are built on a conserved biochemical foundation (Barron et al., 2010; Schultz, 2007; Wise, 2004).

7.1 Biochemical Origins

Dopamine is a catecholamine derived from the amino acid tyrosine, synthesized via tyrosine hydroxylase and dopa decarboxylase as part of an aromatic amino-acid hydroxylase pathway (Kvetnansky et al., 2009; Squire, 2013). This pathway and its cofactors appear early in eukaryote evolution and are deployed in diverse cell types, including microbes and plants, where

catecholamine-like molecules participate in stress responses and interkingdom signaling (Roshchina, 2010). Comparative work shows that dopamine and related compounds modulate reward-seeking and learning across animal phyla, from insects to mammals, indicating a deep evolutionary continuity in the use of dopaminergic signals to bias action selection (Barron et al., 2010; Wise, 2004).

7.2 Evolution of Dopamine Systems

In vertebrates, dopamine neurons arise within conserved development, and dopamine receptor families have undergone duplications and specializations that parallel major transitions in vertebrate brain organization (Callier et al., 2003; Squire, 2013). Evolutionary analyses of chordate dopamine systems show both strong conservation of core midbrain circuits and diversification of projection targets and receptor subtypes, supporting increased behavioral flexibility over time (Yamamoto & Vernier, 2011). This neurological history suggests that the human dopaminergic substrates of social copying represent a relatively recent functional reuse of very old molecular machinery, adapted to encode not only primary rewards but also socially constructed values and norms (Barron et al., 2010; Ruff & Fehr, 2014).

7.3 Dopamine, Prediction Error, and Social Reward

At the circuit level, dopaminergic neurons in midbrain structures such as the ventral tegmental area signal reward prediction errors: transient increases or decreases in firing when outcomes are better or worse than expected (Schultz, 2007; Wise, 2004). These signals drive reinforcement learning by updating value estimates for actions and cues, a mechanism that has been extensively characterized in nonsocial settings (Schultz, 2007). In social contexts, ventral striatum and vmPFC show analogous error-like signals when feedback from others differs from one's expectations or preferences, and these signals predict subsequent conformity toward group opinions (Fliessbach et al., 2007; Klucharev et al., 2009; Zaki et al., 2011). In social settings, this reinforcement architecture is not operating in isolation: positive prediction errors for agreement (the carrot) are complemented by ACC/insula-mediated social-pain responses to disagreement or exclusion (the stick), so that value updates reflect both the pull of alignment and the cost of divergence.

Importantly, the dopaminergic system responds not only to material rewards but also to social comparisons and alignment. Ventral striatum activity increases when individuals receive favorable social comparison information or align with group norms, even when tangible outcomes are unchanged, indicating that social agreement itself is encoded as intrinsically rewarding (Fliessbach et al., 2007; Ruff & Fehr, 2014; Zaki et al., 2011). At the same time, divergence from the group or social exclusion activates dACC and anterior insula in a pattern overlapping with the affective component of physical pain, providing an aversive signal that discourages sustained independence (Eisenberger, 2012; Rotge et al., 2015). Taken together, these findings establish the *paired-gradient* architecture as the canonical enforcement mechanism for social group identity. Ancient dopamine-based reinforcement mechanisms (ventral striatum, VTA) reward alignment, while ACC/insula-mediated social pain punishes misalignment, so that every act of copying or dissent is evaluated by a coupled reward–punishment signal. This carrot-and-stick architecture ensures that social group identity is enforced not through a single aversive channel but through bidirectional gradient pressure: alignment is pulled toward the group by intrinsic reward, and deviation is pushed back by pain-like aversive states — constituting the Level-3 collective-immunity enforcement that the Immunity Framework (§5) identifies as functionally homologous to the Level-2A fight-or-flight response.

8. Insights, Applications, and Predictions

The neural architecture described in this paper yields several key insights with direct implications for modeling and managing collective behavior.

1. *Parallel but inverted control logic.* Fight-or-flight is typically inhibited to allow rational behavior, whereas social copying typically must be resisted to maintain individual judgment; both are automatic defaults requiring prefrontal override, but in opposite directions (§4.2.2) (Hartley & Phelps, 2010; Huang et al., 2014).
2. *Stress amplifies both systems*. The shared PVN–LC axis means that any significant stressor—physical or social—primes both responses, helping explain why social-evaluative stress can trigger physiological FoF reactions and why physical stress may increase social conformity (Herman & Tasker, 2016; Ulrich-Lai & Herman, 2009).
3. *Common substrate, different content*. The shared core (amygdala–ACC–PVN; §4.1) likely evolved once but now processes different types of threat: concrete physical danger for FoF and social uncertainty or misalignment for social copying, reflecting evolutionary efficiency in reusing ancient circuits for newer social challenges (Phelps & LeDoux, 2005).
4. *Valuation system repurposing*. The vmPFC–striatal valuation network, originally evolved for assessing physical rewards and threats, has been recruited for social valuation; social approval and agreement activate the same reward circuitry as food or money, allowing conformity to hijack individual decision-making through evolutionarily ancient reward mechanisms (Bechara et al., 2000; Chen et al., 2023; Fliessbach et al., 2007; Ruff & Fehr, 2014; Zaki et al., 2011).

This layered architecture—ancient subcortical stress circuits overlaid with cortical valuation and social-cognitive systems—explains why both responses can be automatic yet also cognitively modulated, and why they interact so extensively under conditions of stress or uncertainty (FeldmanHall & Shenhav, 2019; Ulrich-Lai & Herman, 2009). It leads to specific predictions: manipulations that jointly increase social-evaluative threat and coordination demands should produce especially strong conformity by co-activating ACC/insula social pain, dopaminergic reward of alignment, and dlPFC-mediated control, yielding steeper and more persistent social-learning effects than either factor alone (Eisenberger, 2012; Klucharev et al., 2009; Zaki et al., 2011).

Across species and institutions, systems exposed to more volatile or competitive environments should show tighter coupling between stress markers and conformity behaviors, stronger multilevel selection on coordination traits, and greater vulnerability to modern maladaptations such as polarization and herd behavior when informational environments diverge from ancestral structure (Baddeley, 2010; Bharti & Kumar, 2022; Mukherjee & Bassler, 2019; Seghers, 1974; Strassmann & Queller, 2011).

9. Limitations and Future Directions

9.1 Proximate vs. ultimate explanations.

The neural evidence reviewed here is primarily proximate and largely correlational: activation of reward, control, and perception–action coupling circuits during social influence shows how social information is implemented in the brain, but does not by itself establish that these circuits evolved for collective-level benefits rather than for individual social reward learning or generic conflict monitoring (Bonini et al., 2022; Lin et al., 2018; Overgaauw et al., 2019; Rizzolatti & Fogassi, 2014; Stallen & Sanfey, 2015; Toelch & Dolan, 2015). The proposal that social copying functions as a Level-3 "collective immunity" system within a multilevel-selection framework is therefore an evolutionary hypothesis layered on top of more neutral proximate findings, not a conclusion

compelled by the neuroscience alone (Beaumont et al., 2009; Goodnight, 2005; Kramer & Meunier, 2016; Simons, 2011; Starrfelt & Kokko, 2012; Tufto, 2015; Wilson & Wilson, 2007). Future work should link neural mechanisms more directly to fitness-relevant outcomes across levels of organization, for example via comparative, longitudinal, or experimental-evolution designs that track both individual and group consequences of conformity-like behaviors (Beaumont et al., 2009; Mukherjee & Bassler, 2019; Seghers, 1974; Simons, 2011; Strassmann & Queller, 2011; Valentini et al., 2020).

9.2 Strength and generality of the override claim.

The characterization of social copying as a *Level-3 → Level-2A override* that can bias or transiently capture rational deliberation (§5) is supported by behavioral data (e.g., Asch-type paradigms) and converging neuroimaging findings (Bond & Smith, 1996; Klucharev et al., 2009; Lin et al., 2018; Overgaauw et al., 2019; Wu et al., 2016). However, the analogy to the fight-or-flight override of prefrontal processing is not fully symmetric: conformity is probabilistic rather than universal, and many individuals resist even under strong social pressure (Bond & Smith, 1996). Evidence that social copying genuinely overrides deliberation in the same mechanistic sense as FoF remains indirect and task-dependent, particularly outside tightly controlled laboratory paradigms (Huang et al., 2014; Toelch & Dolan, 2015; Wu et al., 2016). More work combining time-resolved neural measures, causal perturbations, and fine-grained behavioral assays is needed to determine when and how social influence actually pre-empts or reshapes deliberative processing, and how this varies across individuals, cultures, and threat contexts (FeldmanHall & Shenhav, 2019; Martinez-Saito & Gorina, 2022; von Dawans et al., 2012) – noted that (Bond & Smith, 1996) found conformity rates vary substantially across cultures (collectivist > individualist societies).

9.3 dlPFC and "inverted but parallel" control logic.

The claim that fight-or-flight is typically inhibited by prefrontal control whereas social copying typically must be resisted by prefrontal control is better supported on the FoF side than on the conformity side. For fear regulation, the role of prefrontal regions (including dlPFC and vmPFC) has been probed using lesions, stimulation, and connectivity analyses, providing relatively strong causal evidence (Hartley & Phelps, 2010; Motzkin et al., 2015). For social conformity, most evidence linking dlPFC to resistance is correlational and task-specific, and the exact processes implemented by dlPFC in balancing social reward and social pain remain incompletely specified (Huang et al., 2014; Toelch & Dolan, 2015; Wu et al., 2016). Future studies using causal interventions, cross-task generalization, and individual-differences approaches are needed to test whether an "inverted but parallel" control architecture is robust and generalizable (FeldmanHall & Shenhav, 2019; Konovalov et al., 2018; Wu et al., 2016).

9.4 Reward–punishment structure and reinforcement-learning accounts.

The suggestion that fight-or-flight reward is predominantly outcome-based while conformity often involves a combination of process-like social reward and punishment through social pain is interpretive and not yet firmly established (Eisenberger, 2012; Ruff & Fehr, 2014; Ulrich-Lai & Herman, 2009). Ventral striatum activation during conformity could reflect intrinsic value of alignment, anticipation of social approval, avoidance of ostracism, or some mixture of these, while ACC/insula activation during non-alignment may reflect salience, pain, or more general aversive conflict (Rotge et al., 2015; Zaki et al., 2011). Likewise, reinforcement-learning–inspired models that treat discrepancies between personal and group opinions as prediction errors have yielded mixed results, with some studies failing to find clear evidence for a single, shared mechanism between social conformity and standard reinforcement learning (Konovalov et al., 2018; Toelch & Dolan, 2015; Wu et al., 2016). Disentangling intrinsic social reward, avoidance of social pain, and generic

prediction-error processes will require tasks that orthogonally manipulate social and nonsocial payoffs, combined with computational modeling and multimodal imaging (Chen et al., 2023; Konovalov et al., 2018; Ruff & Fehr, 2014).

9.5 Mirror mechanisms and motor implementation.

Attributing motor aspects of social copying to a "mirror neuron system" is controversial. Strong versions of mirror-neuron theory as a general account of social cognition have been widely critiqued, and current evidence favors more distributed perception–action coupling across premotor, motor, parietal, and associative regions (Bonini et al., 2022; Rizzolatti & Fogassi, 2014). In this paper, mirror-like mechanisms are treated as one candidate motor pathway contributing to rapid imitation and synchronization, but the overall account does not hinge on the existence of a dedicated mirror system (Bonini et al., 2022; Iacoboni, 2009). Future work should clarify which specific sensorimotor circuits support stress-sensitive social matching, how automatic their engagement is, and whether their contribution is necessary, sufficient, or merely facilitative for conformity behaviors (Rizzolatti & Fogassi, 2014; Valentini et al., 2020).

9.6 Multilevel selection vs. gene-centric alternatives.

Interpreting social copying as a Level-3 collective-immunity mechanism that evolves through multilevel selection is one of the most contentious aspects of the thesis. Inclusive-fitness and kin-selection frameworks offer alternative explanations in which the same conformity phenomena are driven by individual or gene-level payoffs, with apparent group-level adaptations arising from relatedness and assortment rather than direct group-level selection (Goodnight, 2005; Kramer & Meunier, 2016; Wilson & Wilson, 2007). Discriminating among these accounts will require empirical work that measures and models selection gradients at multiple levels (individuals, groups, possibly higher-level units) and that examines how conformity and coordination traits respond to manipulations of relatedness, group composition, and between-group competition (Beaumont et al., 2009; Seghers, 1974; Strassmann & Queller, 2011).

9.7 Scope of generalization across taxa and contexts.

The cross-taxa parallels—from bacterial quorum sensing to human neural conformity—are developed at a high level of functional abstraction. While this illuminates recurring patterns of stress-linked coordination, it risks overextending the social-copying concept across mechanisms that are only loosely analogous (Adamo, 2017; Adamo & Baker, 2011; Ottaviani & Franceschi, 1996). More detailed comparative work is needed to map which specific features of the human social-copying response (e.g., stress sensitivity, default status, neural substrates, carrot-and-stick enforcement) are genuinely shared with simpler systems and which are unique elaborations (Mukherjee & Bassler, 2019; Seghers, 1974; Strassmann & Queller, 2011). Similarly, the current synthesis focuses on threat and uncertainty; the extent to which the same mechanisms operate in positive, opportunity-driven contexts (e.g., innovation waves, prosocial cascades) remains to be clarified (MacLeod et al., 2023; Ruff & Fehr, 2014; von Dawans et al., 2012).

Overall, the framework offered here should be read as a structured set of hypotheses that organize existing behavioral, neural, and evolutionary findings into a unified picture of social copying as a candidate collective-survival system enforced by both reward and pain signals. Many of its components—especially the precise control logic, the reward–punishment structure, the computational implementation, and the evolutionary pathway—remain open to revision as more discriminating data and models become available.

10. Conclusion

The fight-or-flight response and social copying under stress share a conserved stress-detection core but diverge sharply in their implementation, targets, and evolutionary level of action. FoF relies on catecholamine-driven sympathetic outputs to protect individual organisms from physical threats, whereas social copying uses a combination of dopaminergic valuation and ACC/insula-mediated social pain to bias individuals toward alignment with their groups under social uncertainty (Eisenberger, 2012; Klucharev et al., 2009; Schultz, 2007). Seen through the lens of an evolutionary-immunity framework and multilevel selection (Johnson, 2026), these systems can be interpreted as parallel survival mechanisms—one at individual innate immunity (Level 2A) and one at collective immunity (Level 3)—that repurpose ancient biochemical pathways to produce fast, stereotyped responses at different organizational scales.

At the same time, the analogy is intentionally constrained. The evidence for an "inverted but parallel" control logic and for a genuinely collective-immunity function in social copying is suggestive rather than definitive, and alternative, more gene-centric interpretations remain viable (Hartley & Phelps, 2010; Kramer & Meunier, 2016; Motzkin et al., 2015). By making these assumptions explicit and framing them as testable hypotheses rather than settled facts, the current synthesis is meant to provide a structured agenda for empirical and theoretical work on how stress, social identity, and neural valuation combine—via both reward and pain—to shape the remarkable power and modern pathologies of collective behavior (Baddeley, 2010; Bharti et al., 2025; Bond & Smith, 1996; FeldmanHall & Shenhav, 2019; Martinez-Saito & Gorina, 2022). Further research in the neurological foundation of social copying has significance in modern society where the polarization of social and political groups can trigger maladaptive social conformity, serving neither the individuals in groups or society at large.

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