Eurasia Specialized Veterinary Publication

Entomological Research Letters

ISSN: 3108-639X

2024, Volume 4, Issue 1, Page No: 115-124
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Available online at: www.esvpub.com/

Collective Defenses in Social Insects Operate as Distributed Risk-Management Systems Linking Behavior, Immunity, and Colony Organization

Lucas Meyer[1]*, Anna Schmid[2], Stefan Braun[1], Laura Keller[3]

[1]Department of Collective Defenses and Distributed Risk Management, Faculty of Life Sciences, ETH Zurich, Zurich, Switzerland.
[2]Department of Behavioral and Immunological Defenses, Faculty of Biosciences, University of Bern, Bern, Switzerland.
[3]Department of Colony Organization and Social Immunity, Faculty of Sciences, EPFL Lausanne, Lausanne, Switzerland.

***E-mail** ✉ lucas.meyer@ethz.ch

ABSTRACT

Social insects experience pathogen exposure through the same interaction networks that enable cooperative foraging, brood care, communication, and division of labor. Existing work has established diverse colony-level defenses, yet avoidance, sanitary behavior, physiological immunity, chemical recognition, nutritional regulation, and spatial organization are often interpreted as separate protective traits. This synthesis develops a multilevel account in which these defenses are treated as partially interacting components of a distributed risk-management architecture. The evidence does not support a centralized controller, universal optimization rule, or single evolutionary pathway. Instead, social-insect colonies can express context-dependent combinations of exposure avoidance, load-sensitive care, destructive sanitation, physiological activation, nutrient reallocation, contact-network reorganization, and state-dependent task performance. The architecture is therefore proposed as an analytical framework rather than an empirically validated mechanism. It emphasizes three distinctions: local detection versus colony-level outcome, evidence-supported defensive responses versus proposed cross-layer integration, and baseline colony organization versus pathogen-induced reconfiguration. Ant studies provide most mechanistic evidence, while honeybee and termite studies broaden several principles but do not establish universality. The resulting framework identifies how distributed defenses may complement, substitute for, or interfere with one another and specifies where pathogen evasion, coinfection, incomplete detection, laboratory-to-field transfer, and taxonomic bias constrain inference.

Keywords: Social immunity, Sanitary behavior, Disease recognition, Colony networks, Division of labor, Collective defense.

Received: 28 May 2024
Revised: 29 June 2024
Accepted: 30 June 2024

How to Cite This Article: Meyer L, Schmid A, Braun S, Keller L. Collective Defenses in Social Insects Operate as Distributed Risk-Management Systems Linking Behavior, Immunity, and Colony Organization. Entomol Appl Sci Lett. 2024;4(1):115-24. https://doi.org/10.51847/gWfta0wE3o

Introduction

Sociality creates an epidemiological paradox. Frequent contact, shared nest space, food exchange, and brood care can amplify opportunities for parasite transmission, yet social-insect colonies also express collective defenses that reduce exposure, remove infectious material, or buffer infection after exposure. Social immunity is therefore best understood as a colony-level property emerging from interacting behaviors and physiological processes rather than as a single hygienic act [1]. This framing also requires a strict distinction between individual immune function and colony-level protection: similarities between organismal and social immunity are analytically useful, but they do not imply that a colony reproduces the regulatory architecture of an individual organism [2].

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A second difficulty is conditionality. Social defenses need not be activated in the same way across colony states, reproductive systems, or social contexts. Theory and comparative synthesis distinguish relatively unconditional protection from defenses whose expression depends on threat, recipient value, or social organization [3]. More broadly, behavioral defenses against parasites vary with host social structure and ecological context, so avoidance, resistance, and tolerance cannot be collapsed into a single response axis [4]. Social-insect defense is also embedded in division of labor, because exposure, caregiving, waste handling, foraging, and brood contact are distributed unevenly among colony members [5]. These organizational features may contribute to disease control, but they also serve functions unrelated to infection and should not automatically be interpreted as adaptations for immunity.

The inferential problem is therefore not whether social insects possess multiple defenses; that point is well established. The unresolved problem is how to relate defenses operating at different biological levels without assuming that they are coordinated by a central controller or jointly optimized. Individual immune regulation itself reflects sensitivity–cost trade-offs, including the danger that excessive activation can generate physiological costs or immunopathology [6]. At colony scale, analogous language of "risk management" is useful only if it remains explicitly conceptual. In this article, distributed risk management denotes the proposed coordination problem created when local sensory information, heterogeneous exposure, physiological state, social care, and colony organization jointly determine which defensive processes are expressed, by whom, and under what conditions. The term does not imply cognition, formal calculation, or demonstrated colony-level maximization.

Materials and Methods

Defense-layer classification

This synthesis used a functional defense-layer classification to organize the 2017–2024 evidence assembled for this synthesis without treating the literature as a new empirical dataset. Evidence was assigned according to the biological level at which a response was measured, the threat information available to the responding individual or colony, the observed defensive action, and the inferential boundary between a measured response and its possible colony-level consequence. The classification distinguishes exposure management, social hygiene, chemical and physiological defense, spatial organization, and task-linked exposure, while detection–response feedback is treated as a cross-cutting process rather than as an additional defense category.

Threat recognition was classified separately from response because chemical cues can alter both behavior and immune state, yet cue detection is not equivalent to pathogen diagnosis. Reviews of insect disease recognition show that cuticular or microbial chemical signatures can act as cues for defensive behavior or immunomodulation while the sensory and mechanistic basis remains incomplete in many systems [7]. This separation prevents a detected cue from being interpreted automatically as a reliable estimate of infectious risk.

A second classification rule separates individual physiological protection from collective protection. In Lasius niger, bacterial challenge was associated with functional individual immune protection, whereas fungal challenge was effectively controlled through social immunity, illustrating pathogen-specific complementarity between defense layers [8]. That pattern is informative but cannot be treated as a universal evolutionary replacement of individual immunity. Conversely, work on Temnothorax curvispinosus found that colonies with stronger hygienic corpse removal had weaker individual-level protection and detected no pathogen avoidance in the tested assays [9]. Because the relationship was cross-colony and correlational, it supports neither a universal trade-off nor a causal allocation rule; instead, it demonstrates why defense layers should be allowed to vary independently.

The classification also permits cross-level signaling. In honeybees, cuticular hydrocarbon profiles associated with immune-challenged workers induced immune-gene activation in queens under direct-contact conditions [10]. Such findings justify links between chemical information and physiological response, but they do not establish pathogen-specific diagnosis or a universally conserved signaling route. Accordingly, the categories used here are analytical rather than claims of fixed evolutionary modules: evidence-supported processes remain tied to their measured taxon, stage, setting, and outcome, while any cross-layer architecture constructed later is explicitly proposed.

Results and Discussion

Individual behavioral avoidance

Avoidance is the earliest possible defense only when a relevant cue is detected before costly exposure and when withdrawal is behaviorally feasible. It should therefore be treated as a conditional route rather than the obligatory first stage of social immunity. Argentine ant colonies exposed to harmful food progressively reduced exploitation and actively abandoned the resource as adverse information accumulated [11]. Because the stimulus was harmful food rather than a pathogen, this experiment is used here only to establish that collective avoidance can emerge dynamically from local information; it does not demonstrate pathogen-specific avoidance.

Infection can also change social behavior in directions that are not uniformly protective. Israeli acute paralysis virus altered honeybee social interactions differently within and between colonies, generating patterns with potentially opposing consequences for transmission [12]. This result cautions against interpreting infection-induced withdrawal, aggression, or altered contact as host-beneficial defense by default. Pathogen effects on host signaling or behavior can coexist with defensive responses, and the epidemiological consequence depends on the interaction context.

Null evidence is equally important. In the acorn-ant system described above, neither individual nor colony-level pathogen avoidance was detected despite measurable variation in individual immunity and corpse-removal behavior [9]. This suggests that colonies may rely heavily on post-exposure defenses in some contexts, or that avoidance cues, costs, and assay conditions limit expression. The proposed architecture therefore does not require every defense layer to be active. Absence, weak expression, or failure of an early layer is treated as a condition that may increase reliance on later behavioral, physiological, or organizational processes rather than as evidence that the colony is undefended.

Grooming and social hygiene

Social hygiene illustrates most clearly why collective defense cannot be reduced to a fixed sequence. In founding Lasius niger colonies, queens can cannibalize infected brood before the brood becomes contagious, simultaneously removing a future infectious source and recovering nutrients [13]. This response is highly state dependent: it concerns founding queens and larvae, not the routine hygienic decisions of mature worker societies. Its importance lies in showing that the same broad problem—containment of infectious material—can be solved through different actions depending on colony stage and recipient state.

In worker-based care, response intensity can track local pathogen information. Experiments on ants exposed to fungal spores showed that caregivers preferentially groom individuals carrying higher infectious loads and that prior grooming by nestmates modifies subsequent grooming dynamics [14]. The resulting colony pattern can therefore arise through local detection and social feedback without invoking a centralized controller. Importantly, the informational substrate is imperfect. Experimental evolution of Metarhizium under social-immunity selection produced fungal lines that elicited less grooming and contained less ergosterol, demonstrating that pathogens can evolve reduced detectability under the tested laboratory regime [15]. This is a specific experimental result, not evidence that field pathogens generally evade social immunity.

Termites broaden the chemical-recognition evidence beyond ants. In Coptotermes formosanus, fungal conidia and the membrane sterol ergosterol increased allogrooming, supporting a role for pathogen-associated chemical information in initiating sanitary behavior [16]. Ergosterol should nevertheless be treated as a contributing cue rather than a universal or sufficient pathogen label. Comparative termite work further shows that allogrooming can compensate for relatively weak individual antifungal defense in tested systems [17]. This supports functional redundancy between defense layers, but whether compensation is common across termite lineages or pathogens remains unresolved.

Care is also conditional on the risk borne by caregivers. Ants facing superinfection altered the form of sanitary care rather than simply maximizing contact with infected nestmates, supporting genuinely risk-adjusted caregiving in that experimental context [18]. At later infection stages, sanitary behavior can become destructive. Lasius neglectus workers open infected pupal cocoons, apply antimicrobial poison, and damage infected brood in a process termed destructive disinfection, which prevents progression to contagious disease in the tested fungal system [19]. Routine grooming and destructive disinfection therefore represent distinct response states rather than different intensities of one behavior. One preserves the treated individual when feasible; the other can sacrifice brood to contain colony-level spread.

Chemical and immune defenses

Chemical and physiological defenses extend the colony response beyond removal of infectious particles. At the molecular level, perturbation of microRNA-related machinery in Reticulitermes chinensis alters social-immunity behaviors and antifungal outcomes, indicating that collective defense can depend on molecular regulation within individual workers [20]. The result links molecular state to colony-relevant behavior, but RNA interference can have pleiotropic effects and does not establish a conserved "master regulator" of social immunity.

Resource acquisition is another route by which infection can alter colony physiology. Fungal infection in Argentine ants shifts collective nutrient intake toward amino-acid-rich diets, and immune stimulation supports the interpretation that the host response contributes to this dietary change [21]. The nutritional shift is therefore consistent with infection-dependent resource allocation, not with a universal rule of adaptive self-medication. Short-term requirements of challenged ants may differ from the nutritional optimum of healthy workers, making nutritional defense a context-dependent adjustment with potential costs.

Defense also has temporal carry-over. In incipient Lasius niger colonies, early fungal exposure of founding queens altered developmental dynamics and increased protection of the later workforce under bounded challenge conditions [22]. Because protection can fail under sufficiently strong subsequent exposure, this evidence supports history-dependent colony protection rather than permanent or vertebrate-like immune memory. Pathogen context adds another layer of contingency: under fungal coinfection, social immunity altered competitive outcomes between pathogens, so the effect of a defense cannot always be inferred from single-pathogen experiments [23].

These cases make a mechanism–boundary comparison more useful than a list of "chemical defenses," because the measured processes span cue-mediated activation, molecular regulation, nutritional reallocation, exposure history, and pathogen interaction. The distinctions below separate what each mechanism permits us to infer from what remains untested.

Table 1. Mechanistic boundaries separating chemical recognition, physiological protection, and colony-level resource responses

Recognition and individual protection			
Chemical cue → physiological activation			*Evidence-supported relation*
Biological level	Honeybee workers and queens		
Evidence crossing levels	Challenge-associated cuticular chemistry followed by queen immune-gene activation		
Social chemical information can alter physiological readiness		**COLONY INTERPRETATION**	
Competing explanation	Cue may signal challenge state rather than identify a specific pathogen	**Validation**	Test natural infections, cue specificity, and sensory pathways across colonies
Pathogen-specific partitioning of individual and social protection			*Evidence-supported; broader architecture proposed*
Biological level	Ant workers		
Evidence crossing levels	Differential functional protection against bacterial and fungal challenges		
Defense layers can complement one another differently across pathogen classes		**COLONY INTERPRETATION**	
Competing explanation	Species-specific immune loss or pathogen ecology may drive the pattern	**Validation**	Replicate across taxa, pathogens, and field-relevant exposure routes
Molecular regulation of collective defense			*Evidence-supported relation*
Biological level	Termite workers		
Evidence crossing levels	RNA-interference perturbation linked to altered sanitary behavior and antifungal outcome		
Individual molecular state can influence socially expressed defense		**COLONY INTERPRETATION**	
Competing explanation	Pleiotropy prevents assignment to a single dedicated social-immunity pathway	**Validation**	Resolve tissue, target-gene, and behavioral specificity
Colony resources and exposure history			
Infection-dependent nutrient reallocation			*Evidence-supported relation*
Biological level	Ant colonies		
Evidence crossing levels	Shift in collective amino-acid:carbohydrate intake during infection or immune activation		

Colony resource acquisition can change with physiological demand		**COLONY INTERPRETATION**	
Competing explanation	Pathogen manipulation and long-term dietary costs must remain separable	**Validation**	Compare short- and long-term fitness consequences under natural diets
Exposure-history carry-over			*Evidence-supported relation*
Biological level	Founding ant queens and later workforce		
Evidence crossing levels	Early challenge followed by altered development and later protection		
Defense state can persist across colony development		**COLONY INTERPRETATION**	
Competing explanation	Protection is challenge-dependent and not equivalent to durable immunological memory	**Validation**	Test duration, specificity, and mechanism under graded exposure
Compound infection			
Coinfection-dependent defense outcome			*Evidence-supported relation*
Biological level	Ant colonies with competing fungal pathogens		
Evidence crossing levels	Changed pathogen competition under social immune care		
A defense can alter pathogen–pathogen interactions as well as host protection		**COLONY INTERPRETATION**	
Competing explanation	Direction may reverse with pathogen combination	**Validation**	Use multispecies infection designs with independent burden measures

Spatial colony organization

The distribution of contacts can change disease risk even when no worker changes its intrinsic immunity. Automated tracking in Lasius niger showed that pathogen exposure reorganized colony social networks in a direction expected to reduce transmission, with changes in interaction structure occurring across exposed and unexposed nestmates [24]. This is strong evidence for pathogen-associated network plasticity in one ant system, but reduced transmission potential should not be equated automatically with demonstrated long-term fitness optimization.

Spatial organization also exists before any immediate infection. Comparative tracking and modeling across social insects showed that simple movement mechanisms can generate spatial division of labor, demonstrating that task-associated segregation need not originate as a disease-specific response [25]. This distinction is central to the proposed synthesis. Baseline colony organization can create an exposure-routing substrate on which pathogen-induced plasticity later acts; the same spatial pattern may therefore have both non-disease functions and epidemiological consequences.

Behavioral individuality further complicates exposure. In genetically clonal Ooceraea biroi colonies, pre-existing differences in behavior predicted infection risk, while infection itself also altered behavior [26]. Because genotype was controlled, the study provides unusually clear evidence that social position and behavioral state can generate heterogeneous exposure, yet the relationship is bidirectional and should not be reduced to a simple claim that task causes infection. Cross-species work likewise shows that major features of ant social-network structure are conserved across several lineages [27]. Conservation indicates that colonies possess repeatable organizational substrates, but it does not show that those substrates evolved for pathogen control.

Taken together, spatial organization should be separated into baseline structure, behaviorally generated exposure heterogeneity, and pathogen-induced reconfiguration. Only the last of these is directly demonstrated as an induced disease response. This separation prevents a common inferential error: reading every stable nest zone, task-associated contact pattern, or conserved network property as social immunity merely because it can influence transmission. In the proposed risk-management architecture, organization constrains who is exposed, who detects change, and which interactions become epidemiologically important, but the defensive meaning of those constraints must be demonstrated rather than assumed.

Task allocation

Task allocation contributes to disease defense principally by distributing exposure and care opportunities rather than by functioning as an immune system in itself. In clonal raider ants, behavioral individuality predicts infection

risk even when genetic variation is minimized, showing that workers occupying different behavioral states encounter unequal hazards [26]. Spatial task organization can likewise arise from simple movement rules without any pathogen-specific trigger [25]. Thus, baseline division of labor should be treated as an organizational substrate on which defensive responses operate, not automatically as evidence of selection for disease control.

Defensive work can nevertheless become specialized or conditional. Social-insect colonies distribute hygienic and defensive functions unevenly among workers and states [5]. More strikingly, Camponotus floridanus workers provide wound-dependent treatment: the intervention differs with injury location and infection dynamics, including amputation under particular conditions [28]. This demonstrates conditional task execution, but it does not establish a generalized surgical algorithm. Likewise, conserved features of ant social networks provide stable organizational structure across species [27] without proving that those structures evolved specifically to control pathogens.

The resulting distinction is between baseline task structure, task-linked exposure, and threat-contingent defensive work. These components can interact, but they are not interchangeable. The multilevel relationships are compressed below because prose alone does not readily show where evidence-supported organizational links end and the article's proposed cross-layer interpretation begins.

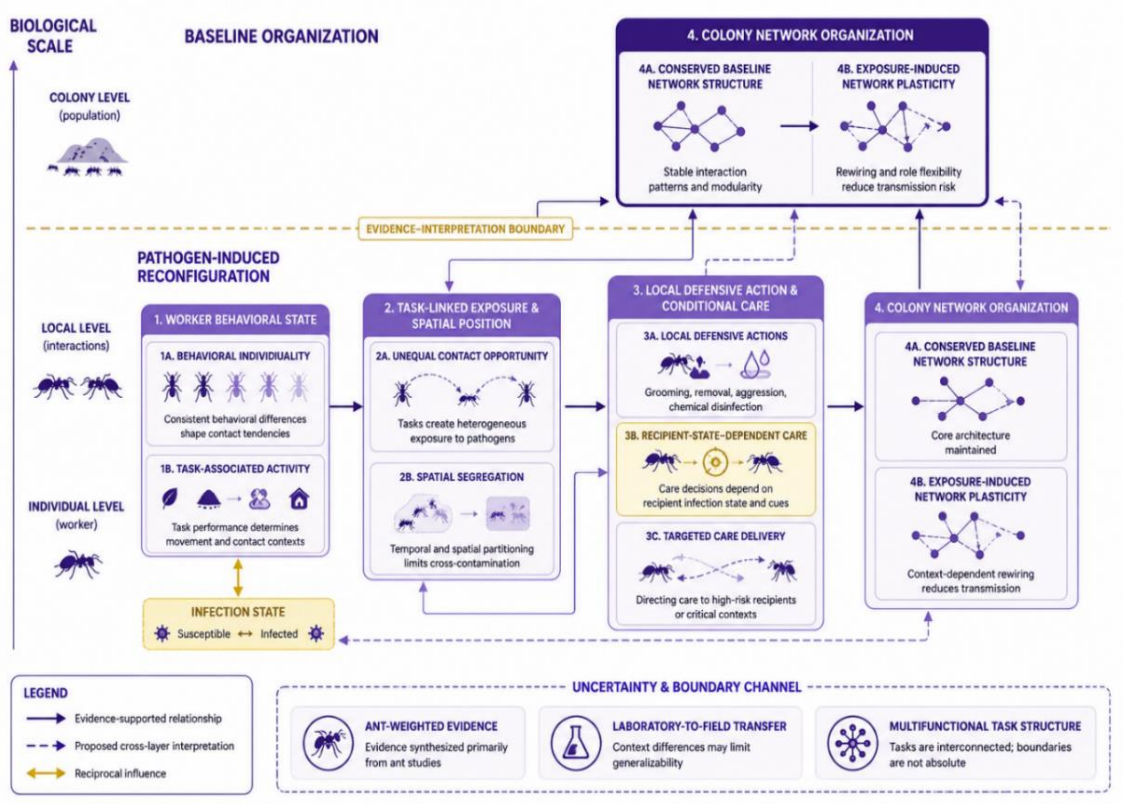


Figure 1. Task-linked exposure, conditional care, and organizational constraints on distributed colony defense

Detection–response feedback

Collective defense depends not only on detecting a threat but also on how local responses alter subsequent information. A useful mechanistic comparator comes from Ooceraea biroi, in which a collective thermal-response threshold emerges through local excitation and global inhibition [29]. Because this experiment did not involve pathogens, it cannot demonstrate a disease-response threshold; it shows only that colony-level nonlinear responses can arise from distributed local interactions.

Disease-specific evidence provides the stronger foundation. Load-sensitive grooming and caregiver feedback alter subsequent sanitary responses [14], while previous infection can modify later collective hygiene. In Atta sexdens, repeated fungal challenges produced finite, partly pathogen-specific history effects that diminished with time [30]. Risk-adjusted sanitary care also changes when superinfection conditions change [18], whereas pathogen evolution

can weaken the cues that elicit grooming [15]. Detection–response systems are therefore neither perfectly informed nor temporally fixed.

The cross-scale distinctions below are needed because superficially similar changes in collective behavior can originate from different processes and justify different inferences.

Table 2. Cross-scale evidence boundaries for interpreting detection, feedback, response updating, and failure in collective defense

Detection and collective threshold		
Detection-response dimension	**Load-sensitive sanitary response**	**Collective threshold formation**
Observable feedback	Care increases with infectious burden and is modified by previous grooming	Nonlinear collective response generated by local excitation and inhibition
Inference permitted	Local information can generate coordinated hygiene	Distributed interactions can create colony-level thresholds
Biological scale	Individual caregiver → colony	Individual interaction → colony response
Failure mode	Cue reliability and local information remain incomplete	Demonstrated with thermal rather than infectious stimuli
Discriminating test	Manipulate cue strength independently of viable pathogen load	Test comparable interaction rules under pathogen-associated cues
Standing	Evidence-supported	Comparator evidence; disease extension proposed
History-dependent updating		
Detection-response dimension	**History-dependent updating**	**Superinfection-sensitive care**
Observable feedback	Rechallenge changes later hygienic response	Sanitary behavior changes with subsequent infection risk
Inference permitted	Collective defense can retain temporary exposure history	Response selection can depend on caregiver risk context
Biological scale	Colony through time	Caregiver–recipient pair
Failure mode	Physiological carry-over, learning, or colony-state change may contribute	Experimental exposure may simplify natural pathogen communities
Discriminating test	Separate mechanism, specificity, and duration experimentally	Test graded and naturally mixed exposure regimes
Standing	Evidence-supported	Evidence-supported
Cue failure and absent avoidance		
Detection-response dimension	**Cue erosion by pathogen evolution**	**Incomplete or absent early avoidance**
Observable feedback	Reduced grooming elicitation after social-immunity selection	No measurable avoidance despite other defenses
Inference permitted	Detection itself can become a target of counterselection	Later defenses can remain relevant when early detection or withdrawal is weak
Biological scale	Pathogen → caregiver detection	Individual/colony
Failure mode	Laboratory passage may not represent field prevalence	True absence cannot always be separated from assay sensitivity
Discriminating test	Compare natural pathogen populations differing in social exposure	Replicate across cues, doses, environments, and colony states
Standing	Evidence-supported under experimental evolution	Evidence-supported boundary

Distributed risk-management architecture

The central contribution of this synthesis is a proposed distributed risk-management architecture. It does not describe a centralized controller. Instead, it treats exposure routing, detection, response selection, physiological defense, resource allocation, and organizational reconfiguration as partially coupled processes whose interactions can alter colony-level disease risk.

Three relationship types are especially important. First, defense layers can be complementary: in Lasius niger, individual and social protection differ across pathogen classes [8]. Second, local feedback can coordinate collective action without central instruction, as demonstrated for load-sensitive sanitary care [14]. Third, colony organization can change after pathogen exposure, altering the network through which future transmission occurs [24]. These observations justify integrating the layers analytically, but no cited study tests the complete architecture as a single system.

The framework also accommodates substitution, conflict, and failure. Nutritional reallocation may support challenged colonies while imposing different costs on healthy workers [21]. Coinfection can alter pathogen competition under social immune care [23]. Pathogens can reduce detectability [15], and some systems show weak or absent avoidance [9]. "Risk management" therefore denotes distributed adjustment under incomplete information, not optimal allocation. The framework is intended to generate testable cross-level questions, particularly whether changes in one layer systematically alter reliance on another under field-realistic exposure.

The strongest evidence supports interactions among particular components rather than validation of a complete colony-wide architecture. Pathogen-specific complementarity between individual and social protection [8], feedback-dependent grooming [14], and infection-associated network plasticity [24] each demonstrate cross-level coupling, but they occur in different experimental systems. Their integration here is therefore conceptual.

Taxonomic imbalance is a major limitation. Ants supply most mechanistic evidence, whereas honeybees and termites provide narrower demonstrations of chemical signaling, infection-dependent social interaction, fungal-cue detection, and grooming-mediated compensation. These findings justify comparison across social insects but not universality. Life stage also matters: founding queens, brood, routine workers, and wounded workers face different constraints, so sanitary decisions observed in one state should not be projected onto another.

Laboratory-to-field transfer is equally important. Experimental fungal doses, controlled coinfections, serial pathogen passage, defined diets, and simplified social environments provide causal leverage on specific processes, yet natural colonies experience variable pathogen mixtures, environmental stressors, dispersal, seasonal turnover, and heterogeneous resources. Transmission-reducing network changes or short-term nutritional benefits therefore should not be equated automatically with lifetime colony fitness.

The proposed architecture also resists adaptationist interpretation. Baseline task organization can arise from movement mechanisms with functions unrelated to disease [25], and conserved network properties need not be immune adaptations [27]. Infection can alter behavior in ways that may either reduce or facilitate transmission [12]. Defensive responses may additionally impose physiological or nutritional costs [6]. Consequently, apparently protective patterns require alternatives such as pathogen manipulation, multifunctional organization, developmental constraint, or experimental context to remain visible.

Future validation should measure several layers simultaneously. The most informative designs would combine pathogen burden, cue availability, worker state, contact networks, care behavior, physiological activation, resource use, and subsequent transmission within the same colonies. Comparative replication across ants, bees, termites, developmental stages, and natural field conditions would test whether proposed cross-layer relations recur or are lineage-specific. Manipulating one layer while measuring compensatory or conflicting changes in others would be especially important for determining whether the architecture has predictive value beyond descriptive integration.

Conclusion

Collective defense in social insects is best interpreted as a multilevel problem rather than a catalogue of hygienic traits. Behavioral avoidance, sanitary care, chemical recognition, physiological protection, nutritional change, spatial organization, and task-linked exposure can interact, but they do not form a universally demonstrated control system. The proposed distributed risk-management architecture organizes these processes around local information, heterogeneous exposure, conditional response, and cross-level feedback while retaining failure, cost, and uncertainty as integral features. Its main value is therefore analytical: it separates established mechanisms from proposed integration and identifies where comparative, multilevel, and field-based tests are required before stronger claims about colony-wide coordination, optimization, or evolutionary generality can be made.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

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