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Multiple Stressors, One Superorganism: An Umbrella Review of Nutritional, Chemical, Parasitic, Pathogenic, and Climatic Threats to Honey Bees

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ABSTRACT

Honey-bee colonies function as integrated biological systems whose survival depends on coordinated nutrition, brood production, division of labour, immune defence, thermoregulation, and resource storage. This organization can buffer temporary disturbances, but it also permits stress originating in particular workers, life stages, or food-processing pathways to propagate through the colony. Evidence concerning honey-bee decline is distributed across reviews of nutritional limitation, landscape simplification, pesticide exposure, parasitic infestation, infectious disease, immune disruption, and climatic stress. These literatures frequently employ different endpoints and analytical scales, making apparent agreement difficult to interpret and preventing individual-level responses from being treated automatically as predictors of colony failure. This umbrella review critically integrates review-level evidence across the principal stressor domains while using selected mechanistic and colony-level studies to clarify biological pathways and interpretive boundaries. The synthesis indicates that nutritional scarcity, chemical exposure, parasites, pathogens, and adverse climatic conditions can each erode components of colony function, but their consequences depend strongly on exposure duration, season, colony demography, resource availability, infection pressure, and compensatory social mechanisms. Co-occurring stressors therefore should not be assumed to interact synergistically, and early molecular, physiological, or behavioural changes should not be interpreted as inevitable colony collapse. The strongest evidence supports a resilience-centred perspective in which colony risk reflects the balance between accumulated stress load and the superorganism's capacity to redistribute labour, regulate the nest environment, sustain brood care, preserve food quality, and control infection. Progress requires longitudinal colony-scale studies, explicit evaluation of overlapping evidence among reviews, and digital phenotyping systems that connect early biological signals with later functional outcomes without overstating predictive certainty.

Keywords: Honey-bee colony health, Superorganism, Colony resilience, Nutritional stress, Landscape resources, Chronic pesticide exposure.

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INTRODUCTION

A honey-bee colony is not merely an aggregation of exposed insects. It is a superorganism in which queens, workers, drones, brood, stored food, nest architecture, and collective behaviours form an interdependent physiological and organizational system. Environmental disturbance can therefore affect colony health through direct worker toxicity, altered nursing or foraging, impaired glandular function, disrupted brood

provisioning, or weakened regulation of the nest environment. Conversely, social organization may buffer substantial individual damage before an externally visible colony-level deficit appears. Toxicological interpretation must consequently consider both the exposed individual and the colony processes through which an effect may be amplified, compensated for, or concealed [1].

Contemporary evidence does not support a universal single-cause explanation for elevated colony losses. Varroa infestation and associated viruses, inadequate nutrition, pesticides, management conditions, queen problems, and weather may contribute in combinations that differ among regions and seasons. Even the outcome "colony loss" is not fully uniform: mortality, queen failure, dwindling populations, absconding, and beekeeper-mediated colony replacement can represent biologically different trajectories. This heterogeneity makes attribution difficult because a stressor associated with loss may operate as a primary driver, an aggravating condition, a marker of another process, or a context-dependent exposure whose importance changes with colony state [2].

The concept of colony resilience provides a more informative organizing principle than a simple inventory of hazards. Resilience encompasses the ability to absorb disturbance, reorganize activity, maintain essential functions, and recover without crossing into persistent decline. Division-of-labour flexibility, behavioural immunity, brood regulation, food storage, thermoregulation, and demographic replacement may delay or prevent failure. However, these mechanisms are finite. A colony facing sustained or simultaneous demands may progressively lose buffering capacity, particularly when the stressors affect long-lived workers, brood production, queen function, nutritional reserves, or the worker populations responsible for collective defence [3].

This article evaluates how review-level evidence characterizes nutritional, chemical, parasitic, pathogenic, and climatic threats, and whether reported effects can defensibly be connected to colony-scale resilience. Previous quantitative synthesis demonstrates that honey-bee stress research varies markedly in experimental unit, life stage, exposure design, duration, endpoint, and environmental realism [4]. Such variation is not statistical noise alone; it defines what each study can establish. The central argument developed here is that credible synthesis requires explicit separation of stressor presence from demonstrated interaction, individual impairment from colony decline, review convergence from independent confirmation, and early biological disruption from inevitable collapse.

Umbrella review scope and review-level evidence

The review was structured as a synthesis of existing reviews addressing managed *Apis mellifera* colony health, supplemented selectively by primary evidence where it clarified mechanisms, exposure pathways, scale transitions, or unresolved disagreements. The analytical population was the honey-bee colony and its constituent life stages; the principal exposures were nutritional limitation, landscape resource loss, pesticides, parasites, pathogens, immune disturbance, heat, and drought. Outcomes were classified as molecular or microbial signals, individual physiological and behavioural effects, intermediate colony functions, and colony-level performance or survival. This hierarchy prevents endpoints at different biological scales from being treated as interchangeable. Umbrella-review methodology requires a clearly bounded question and systematic assessment of review-level evidence rather than an unstructured collection of narrative conclusions [5].

Eligible evidence had to provide sufficient information to identify the biological system, stressor or resource condition, study design, endpoint, and major limitation affecting interpretation. Reviews centred exclusively on non-*Apis* pollinators, unsupported management opinion, or isolated sensor outputs without colony-health interpretation were outside the analytical core. Searches were organized around combinations of the biological system, stressor domain, mechanism, outcome, evidence type, and colony scale. Review conclusions were extracted alongside their underlying study designs and contextual conditions. Primary studies were not used to inflate review-level agreement; they were used to determine whether a proposed pathway had direct support and whether a review conclusion remained consistent with colony-scale evidence.

Methodological quality and primary-study overlap were treated as determinants of

evidentiary confidence. Two reviews may reach similar conclusions because they examine independent bodies of evidence, but they may also repeatedly include the same influential experiments. Reviews can likewise differ because of search dates, eligibility criteria, endpoint definitions, or quality-appraisal methods rather than genuine biological contradiction. Guidance for overviews emphasizes that scope alignment, methodological quality, currency, and study overlap constrain what can be concluded from apparent convergence [6]. The present synthesis therefore uses qualitative confidence language tied to design and consistency rather than invented numerical grades.

The review questions, eligibility boundaries, search and screening logic, evidence-classification rules, and bias controls are specified in **Table 1**.

Table 1. Umbrella Review Scope and Review-Level Evidence: Review Questions, Eligibility Boundaries, Search Logic, Screening Rules, Evidence Classification, and Bias Controls

Review-method element	Operational definition	Inclusion rule	Exclusion rule	Search or screening implementation	Evidence-classification rule	Bias-control measure	Reporting requirement	Representative supporting reference(s)
Review question	How major stressor domains affect honey-bee colony function and resilience	Evidence addressing a named stressor, colony process, or interpretive boundary	Broad pollinator commentary lacking direct honey-bee relevance	Combine *Apis mellifera*, colony, stressor, mechanism, outcome, and review terms	Direct support, qualified support, contextual support, or proposed synthesis	Maintain predetermined construct and scale boundaries	State what is supported, uncertain, or contradicted	[5]
Biological boundary	Managed honey-bee colonies and biologically relevant constituent stages	Colony evidence and individual evidence with a plausible colony pathway	Non-*Apis* evidence without transferable colony relevance	Screen species, caste, life stage, and experimental unit	Molecular, individual, social-process, or colony level	Do not equate individual impairment with colony decline	Identify the biological scale of every principal claim	[1]
Exposure boundary	Nutritional, landscape, chemical, parasitic, pathogenic, immune, and climatic stress	Defined exposure or resource condition	Undefined "environmental stress" without interpretable exposure	Search each domain separately and in combination	Single stressor, co-occurrence, or tested interaction	Do not label co-occurrence as interaction	Specify exposure duration and relevant context	[2]
Outcome boundary	Colony function, resilience, performance, or survival and their mechanistic precursors	Outcomes connected to a defined biological pathway	Isolated measurements with no colony-health interpretation	Extract endpoint, timing, and analytical scale	Early signal, intermediate function, or colony outcome	Avoid treating biomarkers as deterministic predictions	State the inferential distance to colony-level consequences	[3]
Eligible evidence	Peer-reviewed reviews and claim-relevant primary studies	Reviews for synthesis; primary studies for mechanism and boundary testing	Non-peer-reviewed and non-journal evidence	Screen article type and claim relevance	Review-level or primary evidence	Keep the two evidence classes analytically distinct	Identify when a claim relies on primary rather than review evidence	[5]
Evidence overlap	Reuse of the same primary studies across reviews	Reviews with extractable evidence bases	Reviews whose support cannot be traced sufficiently	Compare scope, dates, major included studies, and conclusions	Independent convergence or potentially overlapping convergence	Do not count overlapping reviews as independent confirmation	Report overlap as an uncertainty affecting confidence	[6]
Contradictory evidence	Differences in direction, magnitude, or colony relevance	Conflicts that can be examined through context or design	Selective omission of inconvenient findings	Compare season, exposure, nutrition, infection, demography, and endpoint	Genuine contradiction or context dependence	Retain competing explanations	State what evidence could discriminate among explanations	[4]

Confidence appraisal	Qualitative judgement based on relevance, consistency, scale, and design	Claims with traceable supporting evidence	Unsupported numerical ranking	Integrate design quality and transferability	Higher, moderate, limited, or indeterminate confidence expressed narratively	No invented scores or certainty estimates	Explain the basis and limitations of confidence statements	[6]

Nutritional limitation and landscape resource loss

Honey-bee nutrition involves more than the presence or absence of forage. Nectar supplies carbohydrates, whereas pollen provides proteins, lipids, sterols, vitamins, minerals, and other compounds required for brood rearing and adult physiology. Workers regulate nutrient intake individually and collectively, transform pollen and nectar into stored foods, and redistribute nutrients through glandular secretions. Consequently, dietary quality, diversity, timing, and accessibility may be as important as gross resource abundance. Nutritional physiology also changes with worker role, age, reproductive demand, and season, making a diet adequate under one colony state insufficient under another [7].

Landscape resource loss can create temporal gaps even when flowers remain locally abundant during short crop-bloom periods. Mapping studies demonstrate that forage supply varies across land-cover types and seasons, requiring analysis at spatial scales relevant to colony foraging [8]. Field evidence across agricultural gradients further indicates that worker nutritional condition can differ with surrounding land use, but landscape associations should not be interpreted as simple causal effects because weather, management, colony strength, and resource phenology may covary [9]. A nutritionally poor worker sample signals biological strain; it does not by itself demonstrate that a colony will decline.

Colony consequences emerge when resource shortages persist long enough to constrain brood production, worker replacement, storage, or the physiological quality of long-lived bees. Mechanistic modelling shows that crop identity, diversity, spatial arrangement, and flowering continuity can alter predicted colony viability by changing when and where food is available [10]. Nutritional stress may also reshape the gut microbial community and immune responses and can modify susceptibility to *Nosema ceranae*, illustrating a pathway through which diet can condition responses to another stressor [11]. Nevertheless, altered microbiota or infection intensity in sampled workers remains an intermediate outcome. Demonstrating reduced colony resilience requires longitudinal evidence connecting these changes to sustained functional impairment.

The evidence dimensions and interpretive boundaries for nutritional limitation and landscape resource loss are summarized in **Table 2**.

Table 2. Nutritional Limitation and Landscape Resource Loss: Colony-Level Pathways, Diagnostic Signals, Resilience Outcomes, Management Relevance, Validation Needs, and Interpretive Boundaries

Colony-health component	Signal, stressor, or resource	Biological pathway	Indicator or evidence	Colony-level implication	Management relevance	Uncertainty	Interpretive boundary	Representative supporting reference(s)
Nutrient acquisition	Nectar and pollen availability	Foraging and transfer of carbohydrates, proteins, lipids, and micronutrients	Resource maps, pollen intake, food stores	Determines the material base for maintenance and brood production	Preserve seasonally complementary forage	Resource accessibility may differ from mapped abundance	Floral presence does not establish nutritional adequacy	[7, 8]
Diet quality	Nutrient composition and botanical diversity	Nutrient balancing and physiological allocation	Pollen composition, worker nutrient status	May influence worker condition and brood-support capacity	Evaluate quality as well as quantity of forage	Colony requirements vary by season and demographic state	Greater plant diversity is not automatically a complete diet	[7, 9]

Temporal continuity	Gaps between flowering periods	Depletion of stores and reduced incoming forage	Seasonal resource curves and colony food dynamics	Persistent gaps may constrain brood rearing and worker replacement	Design landscapes for continuous forage rather than isolated bloom	Weather and management alter realized shortages	Short resource gaps need not cause irreversible decline	[8, 10]
Landscape structure	Crop composition and semi-natural habitat	Alters foraging distance, choice, and resource continuity	Land-cover associations and mechanistic models	Can modify exposure to nutritional deficits and other stressors	Retain or restore forage-rich habitat near apiaries	Associations may be confounded; models depend on assumptions	Landscape composition is not a deterministic predictor of survival	[9, 10]
Immune competence	Nutritional restriction	Reduced investment in immune and maintenance processes	Immune markers, infection response, survival	May lower tolerance or resistance under pathogen pressure	Avoid severe nutritional restriction during high disease risk	Marker changes vary with diet, age, and infection conditions	Immune change is not equivalent to colony disease	[11]
Microbial homeostasis	Diet-associated microbiota alteration	Changes in gut-community structure and host–microbe interactions	Microbiota composition and pathogen burden	May influence individual health and stress responsiveness	Treat microbial indicators as contextual diagnostic signals	Direction and functional meaning of microbial shifts may be unclear	Microbiome difference is not proof of colony dysfunction	[11]
Resilience reserve	Stored food and healthy long-lived workers	Buffers temporary resource shortage and seasonal stress	Store levels, demography, worker physiology	Determines how long essential functions can be maintained	Integrate forage planning with colony-demographic monitoring	Buffering thresholds are colony- and season-specific	Early nutritional strain does not imply inevitable collapse	[7, 10]

Pesticides and chronic chemical exposure

Honey-bee colonies encounter chemicals through contaminated pollen, nectar, water, wax, stored food, and in-hive treatments. National monitoring demonstrates that real-world exposure is characterized by changing mixtures rather than a single constant compound [12]. Residue detection establishes contact but not biological harm: interpretation requires concentration, frequency, duration, route, compound toxicity, life stage, food processing, and colony condition. Conversely, low concentrations should not be dismissed automatically when exposure is prolonged or when the affected function is essential to brood care, navigation, reproduction, or social immunity.

Field-realistic work near treated crops has linked chronic neonicotinoid exposure with worker mortality, weakened social-immunity indicators, and increased queenlessness, while also demonstrating that co-exposure to a fungicide can modify toxicity [13]. Mixture experiments have reported changes in worker activity and foraging efficiency that would not be captured by acute mortality alone [14]. These findings establish biologically plausible routes to colony impairment, but neither behavioural inefficiency nor altered worker lifespan alone specifies the final colony trajectory. Colonies may compensate through recruitment and demographic reorganization, or may fail to compensate when exposure overlaps with nutritional scarcity, infection, or critical seasonal transitions.

Chemical exposure can also operate indirectly through social food production. Colony-level exposure to a pesticide mixture has been associated with altered royal-jelly quantity and nutritional composition, indicating that nurse-bee physiology can transmit effects to queen developmental nutrition even when residues are not the only relevant endpoint [15]. This pathway is consequential because queen performance influences brood production and colony continuity, yet altered jelly composition should not be treated as proof of subsequent queen failure without longitudinal confirmation. Reviews of pesticide assessment consequently recommend expanding beyond lethality to behavioural and reproductive endpoints while emphasizing standardization and ecological relevance [16]. A defensible colony-level assessment must combine such sensitive endpoints with exposure realism, temporal follow-up, and direct measures of colony function rather than assuming that every sublethal change predicts loss.

Parasites, pathogens, and immune disruption

Among biological threats, *Varroa destructor* is especially consequential because it combines direct parasitism with virus amplification and transmission. Infestation alters worker and brood condition, changes pathogen dynamics, and can weaken the demographic foundations of overwintering success [17]. The mite's principal feeding target is honey-bee fat-body tissue, which performs functions analogous to nutrient storage, detoxification, metabolism, and immune regulation; damage therefore extends beyond the removal of circulating fluids [18].

Pathogenic effects may remain hidden before visible deformity or colony deterioration occurs. Covert deformed-wing-virus infection has been associated with reduced foraging performance and shortened worker survival under natural conditions [19]. *Nosema ceranae* intensity has also been linked to particular gut bacterial taxa, although its relationship with overall microbiome structure appears comparatively weak and context dependent [20]. These findings support mechanistic concern but do not make infection detection a deterministic forecast of colony loss.

Colony resistance depends partly on grooming, hygienic removal of infested brood, suppressed mite reproduction, virus tolerance, and other socially organized defences. Evidence for these traits varies among populations and measurement protocols, and no single phenotype fully captures survival capacity [21]. Parasite control, pathogen burden, host genotype, colony demography, and environmental resources must therefore be evaluated jointly. Immune-marker changes may indicate altered defence, but they do not independently establish immune failure or irreversible colony decline.

Heat, drought, and climate-related stress

Climate-related stress operates through both direct thermal exposure and indirect changes in floral phenology, water availability, forage continuity, parasite dynamics, and management conditions. Review-level evidence remains dominated by individual-bee experiments conducted over relatively short temporal and spatial scales, limiting inference about long-term colony adaptation and regional beekeeping outcomes [22]. Climatic suitability should therefore not be inferred from isolated temperature responses alone.

Colonies can respond to temporary heat waves by modifying fanning, water collection, worker activity, and brood-area thermoregulation, although these responses impose energetic and labour costs [23]. Heat-shock-protein expression further demonstrates physiological responses whose magnitude can vary among worker roles and locally adapted or imported bee lineages [24]. Climate stress thus intersects with nutrition, chemical exposure, infection, demography, and social regulation rather than operating as an isolated hazard.

Figure 1 classifies major stressor domains within the analytical logic developed in this section.

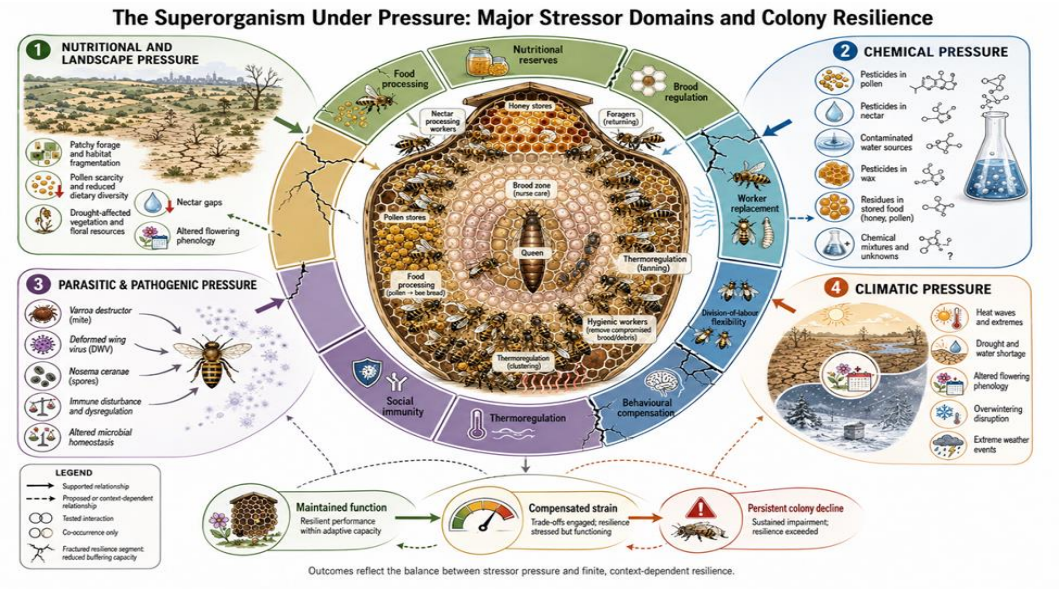

Figure 1. Major stressor domains

Alt text

A structured conceptual diagram that classifies major stressor domains, with labelled components, directional relations, contextual modifiers, uncertainty points, and a clear boundary between observed evidence and proposed synthesis.

Extreme weather may affect losses through temperature anomalies, altered overwintering conditions, resource deficits, and interactions with parasites or pesticide exposure. Large-scale observational analysis has associated colony loss with extreme weather alongside biological and chemical stressors, but such associations cannot identify a universal causal sequence or exclude differences in management and land use [25]. Drought is particularly difficult to isolate because its effects are often mediated through forage quantity, nutritional quality, water demand, and seasonal timing.

Multiple-stressor interactions at colony scale

A demonstrated interaction requires comparison of the combined response with an explicitly defined additive or independent expectation. Controlled experiments have shown that nutritional restriction can intensify neonicotinoid-associated mortality in individual workers [26]. Pathogen–pesticide combinations have likewise produced interactive effects on survival and immunity under particular laboratory conditions [27]. These studies establish biological plausibility, but their results cannot be generalized automatically across compounds, pathogens, diets, exposure schedules, or whole colonies.

Colony-level responses may differ because workers redistribute tasks, adjust brood production, regulate food use, and replace impaired individuals. A large field study found that colonies could buffer short-term effects of pollen restriction and fungicide exposure, although temporary changes in development and microbiome-related outcomes remained detectable [28]. Buffering is therefore an active biological response rather than evidence that exposure was harmless, and its energetic or reproductive costs may emerge outside a short observation period.

Resilience can decline when stress alters the timing of brood rearing, worker emergence, growth, or reproduction and thereby weakens the colony's capacity to reorganize [29]. Colony state is consequently both an outcome and a modifier of future exposure. The evidence dimensions and interpretive boundaries for multiple-stressor interactions at colony scale are summarized in **Table 3**. **Figure 2** shows pathways through which interacting stressors affect individual bees and colony-level resilience within the analytical logic developed in this section.

Table 3. Multiple-Stressor Interactions at Colony Scale: Colony-Level Pathways, Diagnostic Signals, Resilience Outcomes, Management Relevance, Validation Needs, and Interpretive Boundaries

Colony-health component	Signal, stressor, or resource	Biological pathway	Indicator or evidence	Colony-level implication	Management relevance	Uncertainty	Interpretive boundary	Representative supporting reference(s)
Nutritional reserve	Food restriction plus pesticide exposure	Reduced energetic capacity to tolerate toxic stress	Survival and physiological responses	May narrow the colony's compensatory margin	Maintain forage during exposure periods	Colony translation remains context dependent	Individual synergy is not proof of colony collapse	[26]
Immune defence	Pathogen plus pesticide	Altered immune response and survival	Infection, immune markers, mortality	Could increase pressure on worker replacement	Integrate disease and exposure management	Effects vary by agent and design	A tested interaction is not universal	[27]
Colony buffering	Pollen restriction plus fungicide	Task redistribution and demographic compensation	Brood development and microbiome signals	Short-term function may be maintained	Monitor recovery after exposure	Delayed costs may be missed	Buffering does not mean absence of harm	[28]
Social resilience	Accumulated stress load	Disrupted timing of brood, emergence, and reproduction	Colony growth, survival, reproduction	Reduced capacity to absorb further disturbance	Track trajectories rather than snapshots	Thresholds vary among colonies	Early disruption is not inevitable collapse	[29]
Climate interaction	Heat, drought, parasites, and chemicals	Increased energetic demand and	Weather, infestation,	Risk may rise under	Couple environmental	Observational attribution is limited	Co-occurrence is not	[25]

		altered exposure context	residues, colony loss	unfavourable combinations	and biological monitoring		demonstrated interaction	
Demographic reserve	Worker abundance and age structure	Replacement of impaired individuals	Adult population and brood balance	Determines compensatory capacity	Assess worker and brood structure together	Hidden costs may precede visible decline	Stable size can conceal internal strain	[28]
Recovery capacity	Resource return or stress removal	Restoration of food, labour, and homeostasis	Post-exposure development	Distinguishes transient disturbance from persistent decline	Extend monitoring beyond exposure	Recovery time is poorly standardized	A reversible response is not colony failure	[29]

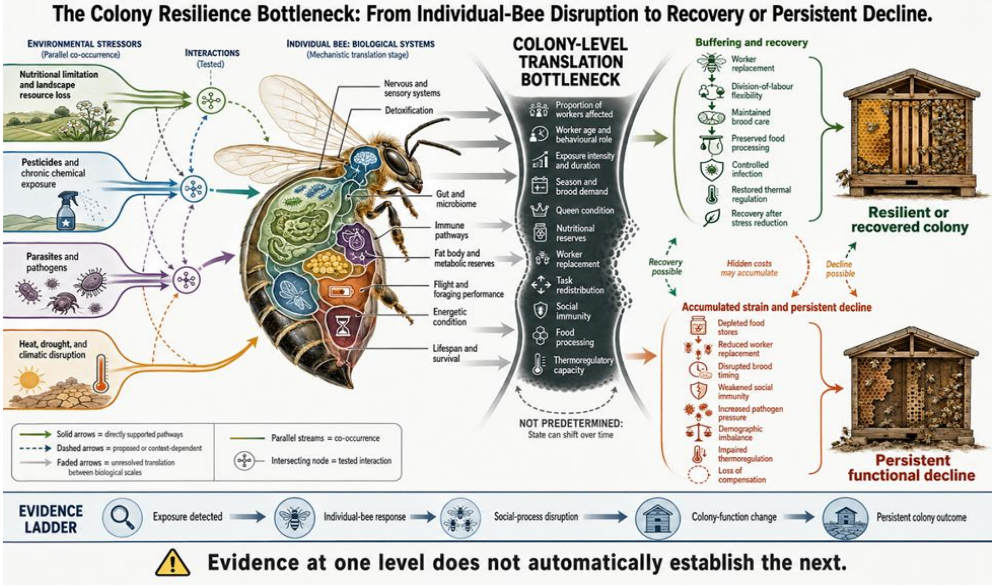

Figure 2. Pathways through which interacting stressors affect individual bees and colony-level resilience

Alt text

A structured conceptual diagram that shows pathways through which interacting stressors affect individual bees and colony-level resilience, with labelled components, directional relations, contextual modifiers, uncertainty points, and a clear boundary between observed evidence and proposed synthesis.

Review-level synthesis and evidence confidence

Across reviews, the most consistent conclusion is that colony health is shaped by multiple biological and environmental pressures whose relative importance changes across regions, seasons, and management systems. *Varroa destructor* and associated viruses receive particularly strong support, while nutrition, pesticides, weather, queen condition, and management modify vulnerability [30]. Agreement on multifactorial causation does not, however, prove that every factor interacts or contributes equally in every colony.

The superorganism perspective explains why decline may be nonlinear. Population models show that stress-mediated reductions in worker function can generate threshold behaviour: colonies may appear stable while compensation remains possible and then deteriorate rapidly after positive social feedbacks weaken [31]. Such models identify plausible dynamics rather than forecast inevitable outcomes for individual colonies, because parameters and initiating stressors remain context dependent.

Experimental evidence similarly demonstrates that individual impairment and colony performance can diverge. Colonies exposed to clothianidin partly compensated for reduced larval survival through increased brood initiation, preserving capped-brood numbers over the short term while potentially incurring longer-term reproductive costs [32]. This illustrates why colony resilience should be measured through both maintained function and the cost of maintaining it.

Confidence decreases when laboratory biomarkers, short exposure periods, or restricted colony conditions are extrapolated to field loss. Imidacloprid-associated gene-expression responses differed between laboratory and field contexts, emphasizing environmental modulation [33]. Broad synthesis also identifies substantial variation in stressor definitions, biological scales, and interaction evidence [34]. The convergent findings, context-dependent results, methodological limitations, and remaining uncertainties are synthesized in **Table 4**.

Table 4. Review-Level Synthesis and Evidence Confidence: Convergent Findings, Context Dependence, Methodological Limitations, Evidence Confidence, and Residual Uncertainty

Evidence domain	Convergent finding	Contradictory or context-dependent finding	Study-design basis	Main methodological limitation	Strength of inference	Residual uncertainty	Implication	Representative supporting reference(s)
Nutrition and landscape	Resource continuity supports colony function	Landscape effects vary with season, weather, and management	Field studies and models	Resource availability and accessibility are difficult to separate	Moderate	Nutritional thresholds remain unclear	Measure quality, timing, and accessibility together	[7, 10]
Pesticides	Sublethal exposure can alter behaviour, physiology, or reproduction	Colony outcomes vary with compound, exposure, and compensation	Laboratory and field experiments	Exposure realism and follow-up differ	Moderate	Long-term colony consequences remain unevenly resolved	Link sensitive endpoints to colony trajectories	[13, 32]
Parasites and pathogens	*Varroa*–virus pressure is a major threat	Survival differs among host populations and management contexts	Reviews, mechanistic studies, and field evidence	Pathogen and mite effects are difficult to disentangle	Relatively strong	Contributions of tolerance and resistance vary	Integrate infestation, viruses, and colony demography	[17, 21]
Climate stress	Heat and extreme weather can challenge colony regulation	Colonies may buffer temporary events; regional responses differ	Experiments, reviews, and observational analyses	Long-term and large-scale studies remain limited	Moderate to limited	Adaptation under repeated extremes is uncertain	Use longitudinal climate–colony monitoring	[22, 25]
Multiple stressors	Interactions occur under defined combinations	Additive, synergistic, and antagonistic outcomes all remain possible	Factorial experiments and field studies	Many studies test individuals or few combinations	Limited to specific contexts	Transferability across exposures is low	Require explicit interaction models	[26, 27]
Colony resilience	Social regulation can delay functional decline	Compensation may carry delayed demographic costs	Colony experiments and models	Recovery and compensation are inconsistently measured	Moderate	Failure thresholds are colony specific	Measure resilience costs and recovery	[29, 32]
Review-level confidence	Several reviews identify common threat domains	Primary-study overlap may inflate apparent convergence	Umbrella-review comparison	Review quality, scope, and currency differ	Conditional	Independence of evidence is often unclear	Report overlap and scale explicitly	[5, 6]

Future priorities for honey bee health

The first priority is longitudinal digital phenotyping that connects continuous signals

with inspected biological states. Internal temperature can contribute to estimates of colony strength, but its interpretation depends on sensor placement, season, brood status, and external conditions [35]. Entrance imaging and temporal models may support early warning from population-loss patterns, yet predictions require validation across apiaries, climates, colony types, and management systems [36].

A second priority is multimodal validation. Molecular indicators should be assessed alongside infestation, pathogen burden, nutrition, demography, behaviour, temperature regulation, and subsequent colony outcomes. Immune-gene profiles have shown potential as markers of how colonies respond to *Varroa* and deformed wing virus, but the evidence remains preliminary and context specific [37]. Biomarkers should therefore support diagnosis and hypothesis generation rather than be presented as stand-alone predictions of failure.

A third priority is interoperable, transparently documented monitoring infrastructure. Open longitudinal datasets containing hive weight, temperature, humidity, management records, and confirmed colony events can support reproducibility and cross-site testing [38]. Future studies should predefine interaction hypotheses, distinguish exposure from effect, measure compensatory costs, document review overlap, and retain negative or mixed findings. Progress should be judged by improved transferability and earlier recognition of recoverable stress, not merely by increased sensor or biomarker sensitivity.

CONCLUSION

Honey-bee colony health cannot be understood by counting hazards independently or by treating every altered worker-level endpoint as evidence of impending collapse. Nutritional limitation, landscape resource loss, pesticides, parasites, pathogens, immune disruption, heat, and drought can all weaken components of superorganism function, but their consequences are conditioned by timing, duration, demography, resource reserves, infection pressure, management, and the colony's capacity for social compensation. The strongest synthesis is therefore resilience centred: colony risk emerges when accumulated demands erode the collective mechanisms that sustain brood care, food processing, thermoregulation, defence, worker replacement, and recovery. Co-occurrence must remain distinct from demonstrated interaction, early biological change from inevitable collapse, and review agreement from independent evidentiary confirmation. The highest-priority advance is longitudinal, multimodal colony monitoring that links mechanistic signals to later functional outcomes while preserving uncertainty and context.

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