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The genome exposome microbiome–phenome (GEM–P): a conceptual framework for social bees

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Abstract

The gut microbiome of social bees is a small, specific set of symbionts that contributes to nutrition, detoxification, immune function and behaviour, with effects on colony performance and resilience. In recent years, the field has expanded, but studies still analyse host genetics and environmental exposures separately and often rely on correlations between microbes and health traits. This makes it difficult to distinguish when microbiome differences primarily reflect host or environmental factors from when they may contribute mechanistically to bee health. A key challenge is context dependence: similar microbiome shifts may be associated with different trait outcomes depending on life stage and task, exposure history, and colony transmission structure. We present the Genome Exposome Microbiome–Phenome (GEM–P) framework as an integrative organisational framework for social bees. GEM–P treats genome, lifetime exposures (exposome), and the gut microbiome as interacting components shaping phenotypes at both individual and colony scales. Phenotypes can, in turn, modify subsequent exposures and microbiome states. We use GEM–P to map evidence on genetic filtering, exposomal drivers, microbiome assembly and function, and phenotypic outcomes, and to highlight study designs that help distinguish among alternative biological pathways without requiring comprehensive multi-omics approaches. GEM–P provides a practical way to generate pathway-specific hypotheses and interpret microbiome–phenotype associations across contexts.

Key points • *An integrative framework linking genome, exposome, gut microbiome, and phenome in social bees.*
• *Highlights scale, timing, and social transmission as sources of context dependence.*
• *Supports pathway-specific hypotheses and sampling designs that help distinguish alternative explanations for microbiome–phenotype associations.*

Keywords Gut microbiota · Symbiosis · Microbial transmission · Resilience · Immunity · Colony health

Introduction

The gut microbiome of social bees is a small, host-specific community that supports nutrition, detoxification, immunity and behaviour, and therefore influences colony performance and resilience (Hammer et al. 2023; Mazel et al. 2025; Motta et al. 2022; Motta and Moran 2024; Zhang et al. 2022).Several reviews summarise microbiome composition and function and discuss links to health outcomes and potential applications, including probiotic/beneficial microbe approaches (Alberoni et al. 2016; Hamdi et al. 2011; Raymann and Moran 2018). However, these syntheses often treat environmental drivers and host genetic background as separate influences rather than as parts of one connected system.

A related challenge is that many studies assume mostly one-way effects, where the microbiome is viewed as an outcome of genes and environment rather than a potential mediator with feedback effects (Meehan and O'Toole 2025; Nguyen and Rehan 2023). Consequently, it can be difficult to disentangle the relative contributions of host genetic filters, environmental exposures, microbiome dynamics and social transmission, and to determine how these processes shape measurable traits at individual and colony levels (Kwong and Moran 2016; Motta and Moran 2024). Broader evolutionary ideas (e.g., niche construction, indirect genetic effects, maternal effects and multilevel selection) have been proposed as useful lenses for host–microbiome evolution, (Week et al. 2025), but these perspectives have not generally been integrated with an explicit treatment of lifetime exposures, gut microbiome dynamics and phenotypic feedbacks in social bees (Week et al. 2025). At the same time, several integrative perspectives have been developed specifically in social bee research. Hologenome based approaches, for example, emphasise the combined contribution of host and microbial genomes to honey bee fitness and their interactions with environmental stressors (Schwarz et al. 2015), while recent work on host specificity has synthesised the ecological and evolutionary processes that structure gut microbial associations across social bee lineages (Mazel et al. 2025).

Addressing these challenges requires integrating gene–environment interactions, exposome research, host–microbiome biology, social transmission perspectives and systems approaches that emphasise feedbacks and time lags (Baniel and Charpentier 2024; Gernat et al. 2018; Naug 2008; Sarkar et al. 2024). We therefore present the Genome Exposome Microbiome–Phenome (GEM–P) as an organisational framework for studying social bees. GEM–P links four components: host genome (G); lifetime exposures (the exposome, E); the gut microbiome (M); and host traits (the phenome, P), and considers their interactions across biological scales (population, colony and individual) and across time (immediate, delayed and evolutionary effects). GEM–P does not propose that the individual relationships among host genetics, environment, microbiome, and phenotype are themselves novel. Rather, it provides a social bee-specific structure in which these established processes can be considered simultaneously while remaining analytically distinct. Its purpose is to organise current evidence, support pathway-specific hypotheses and identify sources of context dependence in social bee gut microbiome research.

In this review, we map current knowledge about social bees onto GEM–P and show how it can guide future applied research. Table 1 compares GEM–P with the principal conceptual frameworks and perspectives on which it draws, highlighting both their shared elements and the specific organisational role of GEM–P in social bee research.

The GEM–P framework

The GEM–P framework describes how phenotypic traits at individual and colony scales (the phenome, P) can be shaped by interactions among host genome (G), lifetime exposures (the exposome, E), and the gut microbiome (M). Here, exposome refers to lifetime physical, chemical and biological exposures, including diet and microbial inputs, pathogens and parasites, and contact with hive substrates and nestmates, as well as socially mediated inputs (Wild 2005, 2012). In the present GEM–P formulation, M refers specifically and deliberately to the host-associated gut microbiome rather than to the entire microbial complement of the bee colony or nest environment.

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Table 1 Relationship of GEM–P to established conceptual frameworks and perspectives relevant to social bee host–microbiome research. The comparison summarises the primary emphasis of each framework and perspective, the extent to which genome (G), environmental exposure (E), microbiome (M) and phenotype (P) are explicitly represented, and the treatment of temporal, feedback and social processes

Conceptual framework/Perspective	Primary emphasis	Genome	Environment/exposure	Microbiome	Phenotype	Time/feedback/social context	Relationship to GEM–P	Representative References
Genotype × environment interaction	Variation in phenotypic response among genotypes across environments	Central	Central	Usually not explicit	Central	Can include context dependence. Microbiome/social transmission not generally explicit	GEM–P incorporates G × E as an established relationship while adding microbiome-mediated and feedback pathways	1
Holobiont / hologenome perspectives	Host and associated microbiota as an integrated biological unit	Host and microbial genomes emphasised	Environmental context may be considered	Central	Often linked to host fitness/function	Evolutionary interactions emphasised	GEM–P retains host and gut microbiome as analytically distinct nodes and separately represents lifetime exposure and phenotype	2–4
Exposome paradigm	Cumulative environmental exposures across a lifetime	May modify susceptibility	Central	Can be part of exposure or mediator depending on formulation	Exposure related outcomes	Strong temporal/lifetime emphasis	GEM–P places lifetime exposure alongside G, M and P and includes socially mediated exposure	5–6
Social microbiome perspectives	Microbial transmission through social interaction and group structure	Usually not explicit	Social contact structures microbial exposure	Central	Behaviour/social organisation important	Social transmission and feedback central	GEM–P incorporates social transmission as P → E → M relationships within the broader G–E–M–P structure	7–8
Systems/feedback approaches	Interacting components, feedbacks and dynamic responses	Potential component	Potential component	Potential component	Potential component	Feedback and temporal ordering central	GEM–P applies this logic to a social-bee-specific organisation of G, E, M and P	9–10

Table 1 (continued)

Conceptual framework/Perspective	Primary emphasis	Genome	Environment/exposure	Microbiome	Phenotype	Time/feedback/social context	Relationship to GEM–P	Representative References
GEM–P	Organising host genetics, lifetime exposures, gut microbiome and phenotypic traits within one social-bee-specific framework	Explicit G node	Explicit cumulative E node	Explicit gut M node	Explicit P node	Explicit temporal ordering, behavioural feedback and social transmission	Integrative organisational scaffold rather than a replacement for the above theories	

Representative references: [(1)] Via and Lande 1985;[(2)] Zilber-Rosenberg and Rosenberg 2008;[(3)] Theis et al. 2016;[(4)] Schwarz et al. 2015;[(5)] Wild 2005;[(6)] Wild 2012;[(7)] Baniel and Charpentier 2024;[(8)] Sarkar et al. 2024;[(9)] Gernat et al. 2018;[(.10)] Naug 2008

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This choice reflects the comparatively well-resolved composition, transmission routes and functional relationships of the social bee gut microbiome, which provide a strong empirical basis for examining relationships with host genetics, environmental exposures and host phenotypes. In GEM–P, microbial communities outside the focal gut microbiome, including hive, food, flower and environmentally associated microbial reservoirs, are represented as biotic components of the exposome when they act as sources of microbial exposure to the host.

We use GEM–P to examine how these components interact over a bee's lifetime, with an emphasis on three microbiome-centred relationships: how host genotype can shape microbiome assembly (G → M), how exposures can alter the microbiome (E → M), and how microbiome variation can influence host traits (M → P). Host genotype and exposures can also affect traits directly (G → P; E → P), and their combined effects are often described as genotype-by-environment interactions (G × E) (Via and Lande 1985). GEM–P incorporates G × E as an established relationship within its broader organisational representation. The principal direct, microbiome-mediated and feedback relationships represented in GEM–P are summarised in Fig. 1.

A defining feature of GEM–P is that P is treated not only as an outcome of G, E and M, but also as a potential driver of subsequent exposure and microbiome states; these feedback relationships are developed below and in Section. 2.4.

Representing GEM–P over time

Time is an important dimension of GEM–P because exposures, microbiome states and phenotypic traits can change over a bee's lifetime and across biologically relevant colony periods. These changes may occur across developmental or task transitions, such as emergence, nursing and foraging, or across seasonal periods such as spring bloom, summer dearth and overwintering. By contrast, the host genome is treated as fixed within an individual's lifetime. Changes in host genetic composition occur over longer evolutionary timescales across generations.

GEM–P therefore distinguishes current relationships from feedbacks that affect later states. For example, current behavioural or physiological traits can alter subsequent exposures by changing diet, habitat use, social contact or interaction with hive substrates (P → E). These altered exposures may in turn influence later microbiome states (E → M), while behavioural and physiological traits can also modify conditions for microbial acquisition, transmission and persistence (P → M).

External conditions such as weather, land use configuration and management practices can modify the exposures experienced by bees but are not themselves generated by colony state over the timescale considered. GEM–P distinguishes these contextual drivers from the specific physical, chemical and biological exposures experienced by the focal bee or colony. The importance of time as a scale is discussed in more detail in Section. 2.2.

Recurring pathways in GEM–P

To summarise this framework operationally, we describe seven recurring pathways. Arrows represent hypothesised directional relationships between GEM–P. Where a pathway describes feedback, the downstream node refers to a subsequent state rather than necessarily a simultaneous change. Where temporal ordering is relevant, t denotes a current state and $t+1$ a subsequent state; these subscripts indicate relative temporal order only. G′ denotes the host genetic composition in subsequent generations, operating on longer timescales than within a bee's lifetime dynamics. Individual arrows may encompass intermediate mechanisms not represented as separate nodes.

Pathway 1. Phenotypic effects within a bee's lifetime ($G, E_t, M_t \rightarrow P_{t+1}$): phenotypes at the next time point arise from combined genomic, exposomal and microbial effects during an individual's life, mainly through gene expression, cellular state, and physiology (Fig. 1B).

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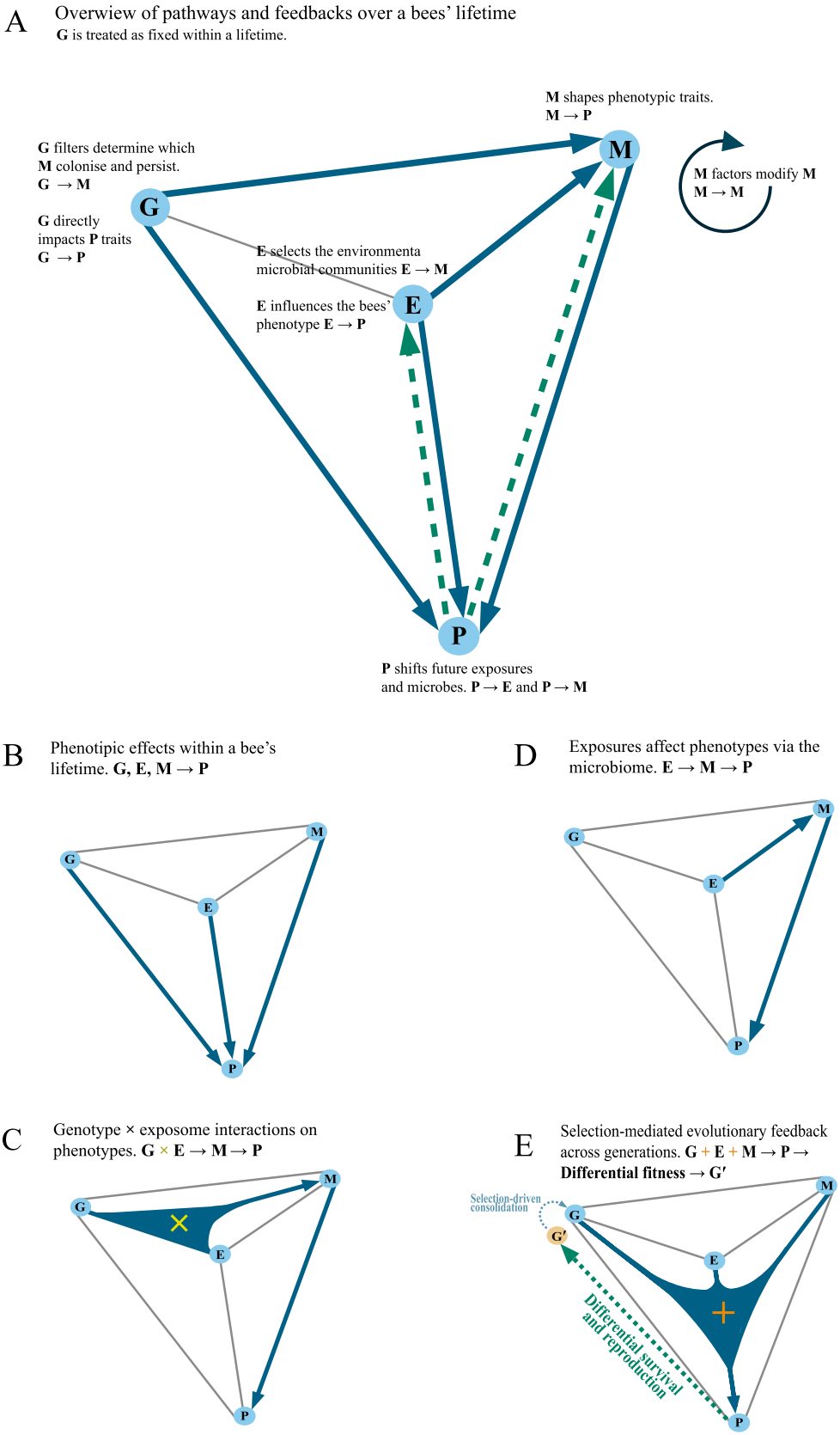

Fig. 1 Key GEM–P relationships and recurring pathways. Nodes represent Genome (G), Exposome (E), Microbiome (M) and Phenome (P). Solid arrows indicate directional relationships represented within a bee's lifetime; dashed arrows indicate feedbacks that act with time lags (e.g., P reshaping later E and M); in Panel **E** the dashed link to G′ represents across generation change; grey arrows stand for other plausible links not highlighted in the panel. Panel **A**—Integrated overview of interactions and feedbacks within a bee's lifetime (encompassing multiple recurring pathways, including Pathways 1–4), whereas Panels **B–E** highlight representative recurring pathways defined in the main text. Panel **B**—Pathway 1, phenotypic effects within a bee's lifetime (**G, E, M → P**); **Panel C—**Pathway 5, genotype × exposome interactions (**G × E → M → P**); Panel **D—**Pathway 6, exposures affecting phenotype via the microbiome (**E → M → P**); and **Panel E—**Pathway 7, selection-mediated evolutionary feedback across generations, in which natural selection acting on phenotypic variation can alter host genotype frequencies through differential survival and reproduction (**G + E + M → P → Differential fitness → G′**)

Pathway 2. Phenotypes shape future exposures ($G, E_t, M_t \rightarrow P_t \rightarrow E_{t+1}$): genomes, exposure, and microbiomes shape phenotypes, that determine where and how bees interact with their environment (e.g., diet choice and habitat use), thereby altering subsequent exposures (Fig. 1A).

Pathway 3. Phenotypes shape future microbiome states ($G, E_t, M_t \rightarrow P_t \rightarrow M_{t+1}$): phenotypic behaviours and physiological traits (e.g., trophallaxis, grooming, social contact patterns, gut conditions) affect microbial acquisition, transmission and colonisation conditions, thereby shaping the subsequent microbiome state (Fig. 1A).

Pathway 4. Microbiome assembly and restructuring ($G, E_t, P_t, M_t \rightarrow M_{t+1}$): microbiome state at the next time step reflects joint effects of host genetic filtering, realised exposures and microbial inputs, phenotype-mediated gut conditions and transmission, and within-community dynamics (e.g., competition, priority effects, cross-feeding) (Fig. 1A).

Pathway 5. Microbiome-mediated genotype × exposome effects on phenotype ($(G \times E_t) \rightarrow M \rightarrow P_{t+1}$): genes and exposures often work together. The same exposure can produce different microbiome changes in different genotypes because genotypes differ in gut conditions and processing of food or toxins. These microbiome differences may then lead to differences in phenotypes (Fig. 1C).

Pathway 6. Exposures affect phenotypes via the microbiome ($E_t \rightarrow M \rightarrow P_{t+1}$): exposures can shift microbiome composition and function, which then affects host physiology and behaviour (e.g., nutrition, immune activity), generating delayed or indirect exposome effects on the phenome (Fig. 1D).

Pathway 7. Selection-mediated evolutionary feedback across generations ($(G, E_t, M_t) \rightarrow P_{t+1} \rightarrow$ *differential fitness* $\rightarrow G'$): host–microbiome interactions and environmental exposures generate phenotypic variation that can affect reproductive success. Where fitness differences are associated with heritable host variation, natural selection can shift genotype frequencies across generations. Thus, G′ represents a change in host genotype frequencies in subsequent generations rather than direct modification of an individual's genome (Fig. 1E). In Sections. 2.1–2.4 we map evidence from social bees onto these pathways for each component (G, E, M, P).

Host genome (G): evolutionary and unctional filters on microbiome acquisition

Studies increasingly show that host genotype contributes to gut microbiome composition and function in social bees (Bridson et al. 2022; De Iorio et al. 2024; Wu et al. 2021; Yang et al. 2021). However, formal heritability estimates for bee gut microbiome traits are still limited. Where they exist, they usually focus on individual taxa or strains (often with small effect sizes), rather than at the whole-community properties. Even so, the available evidence suggests that host genetic effects on microbiome assembly operates across multiple biological scales, forming a layered and hierarchical structure (Su et al. 2022; Wu et al. 2021).

To make the multi-layered structure explicit within GEM–P, Table 2 summarises the main scales at which host genetics can influence the microbiome, the likely primary mechanisms at each scale, and the expected microbiome outcomes. In GEM–P, the pathway G → M refers to host genetic effects on microbiome acquisition that act through genetically influenced host traits (here termed genetically mediated host features, GMHF), such as immune filtering and gut physiology. By contrast, effects that occur because genotype shapes behaviour and therefore changes exposure are represented separately as G → P → E → M (for example, social organisation or foraging behaviour altering microbial contact and transmission).

Overall, host genetic effects on the social bee gut microbiome are best understood as layered across scales. Here lineage refers to genetic lineages within the species (e.g., subspecies or geographically structured populations).

Table 2 Multi-layered host-genomic influence on gut microbiome assembly in GEM–P. Rows summarise the genetic unit, the dominant mechanism(s), and the expected microbiome outcomes at each biological scale. 'GEM–P path' indicates the primary GEM–P pathway emphasised (e.g. G → M via GMHF versus G → P → E → M via exposure and transmission)

Scale	Genetic unit	Primary mechanism(s)	GEM–P path	Microbiome outcome	References
Phylogeny (host identity)	Species ancestry	Long-term host–microbe association; host habitat	$\mathbf{G} \overset{\mathbf{GMHF}}{\rightarrow} \mathbf{M}$	Core membership; host-specific strains	1, 2
Lineage (subspecies)	Subspecies background	Host–microbe matching (adhesion/colonisation)	$\mathbf{G} \overset{\mathbf{GMHF}}{\rightarrow} \mathbf{M}$	Strain divergence under shared E	3
Colony	Queen & patrilines	Indirect genetic effects via social transmission	G → P → E → M	Transmission-biased strain frequencies	4
Individual	Patriline genotype	Immune/epithelial filtering; gut physiology	G → P → M	Differential persistence/clearance	5

Representative references: (1) Kwong et al. (2017); (2) Motta and Moran (2024); (3) Wu et al. (2021); (4) Bridson et al. (2022); (5) Guo et al. (2023).
*GMHF: genetically mediated host features, genetic effects not mediated by the environment.

1. **Species and lineage effects:** Host identity constrains core community membership and strain background, consistent with long-term host–microbe associations.
2. **Host–microbe compatibility effects:** Even when colonies share similar foraging environments, host genomic differences can be associated with consistent differences in colonisation and persistence of particular strains. For example, in apiaries with mixed subspecies, workers retain distinct *Snodgrassella alvi* strains despite sharing the same foraging environments (Wu et al. 2021). Similar host–microbe genotype matching predicts colonisation success of *S. alvi* in *Bombus*, supporting host–microbe compatibility effects beyond *Apis* (Sauers and Sadd 2019)
3. **Colony and individual effects:** Genetic influences are often context dependent and may act indirectly via immune and physiological traits, as well as through social organisation that shapes exposure and transmission routes (both G → M and G → P → E → M).

A detailed description of these effects across layers is provided in Supplementary Appendix 1.

Exposome (E): environmental and social inputs shaping microbiome and phenome.

The exposome encompasses the totality of environmental and socially mediated inputs that influence an organism and its microbiome across its lifetime (Wild 2012). In social bees, this includes nutrition, chemical exposures (e.g., agrochemicals and antimicrobials), pathogens and parasites, seasonal and climatic variations, and landscape context. It also includes microbially relevant inputs acquired through contact with hive substrates and social contact with nestmates. In GEM–P, these socially mediated microbial inputs are treated as part of the exposome (E). Microbial reservoirs outside the focal gut community are therefore not automatically included within microbiome (M); when they act as sources of microbial exposure to the host, they are represented as biotic components of exposome (E). At the same time, the exposome is strongly shaped by phenotypes since behaviour and colony organisation change the exposure that bees experience (P → E). These shifts in realised exposure can then alter microbial inputs and transmission, with consequences for microbiome state (P → E → M) consistent with the 'social microbiome' perspectives (Baniel and Charpentier 2024; Sarkar et al. 2024).

Within GEM–P, exposures influence the system through two main routes (Hotchkiss et al. 2022).

1. **Exposures reshape the microbiome (E → M):** Exposures change which microbes bees encounter and alter selection pressures inside the gut, leading to shifts in microbiome composition and function.

2. **Exposures affect phenotypic traits directly (E → P), often with downstream microbiome effects:** Exposures can influence bee physiology and behaviour (e.g., nutrition and immune activity), which can in turn modify gut conditions and transmission routes (E → P → M) (Hotchkiss et al. 2022). Because behaviour also changes exposures, feedbacks can occur (P → E → M).

Exposures vary across time and space

Developmental stage and chronological age are not themselves treated as components of the exposome in GEM–P; rather, they provide temporal and ontogenetic (developmental stages) context that can modify behaviour, physiology and social interactions, thereby structuring the realised exposures experienced by the bee. Realised exposures are not uniform within a colony. They vary over time because developmental and task-related transitions (e.g., nurse to forager) alter behaviour, social interactions and contact with the external environment; they also vary with seasonality (Hotchkiss et al. 2022). They also vary across space, both within the hive (substrates and microhabitats) and across the surrounding landscape (foraging locations and landscape context) (Jones et al. 2018). As a result, exposomal effects are expected to change across time and space rather than being homogeneous (Supplementary Box S1). Figure 2 summarises these shared temporal and spatial axes and their implications.

It is also useful to distinguish the realised exposome experienced by a bee or colony from broader external environmental conditions, such as weather, land-use context and management regime. These conditions can shape realised exposures but are generally external to colony-generated feedbacks over the timescale considered. This distinction helps separate variation arising from colony behaviour and organisation from variation imposed by broader environmental conditions.

Evidence and common patterns

Across studies, a consistent pattern emerges: diverse exposomal drivers (such as changes in diet quality and forage diversity, infection pressure, chemical disturbance, and other external micobiomes) can disrupt gut microbiome stability and function, with downstream consequences for health-relevant traits. Diet effects can also be context dependent: pollen botanical origin and nutritional composition can modulate stress responses even when pollen carries chemical residues (Janam et al. 2026). Infection pressure can also restructure gut communities by favouring opportunists; for example, *Vairimorpha (Nosema) ceranae* infection has been associated with marked shifts in gut community structure and can facilitate *Serratia* proliferation (Alberoni et al. 2023; Braglia et al. 2024). These effects are often strongest when exposures co-occur, when the timing or order of exposure matters (early events shaping later states), and when colony transmission dynamics amplify microbiome change (Castelli et al. 2021; Favaro et al. 2023; Rouzé et al. 2019).

In honey bees, landscape- and forage-associated variation has been linked to reproducible differences in gut community structure and colony performance (Bridson et al. 2022).Comparable geography- and landscape-associated differences have also been reported in honey bees sampled across Brazilian biomes and in wild South American bumble bees, and environment linked variation has been described in Australasian stingless bees and New Zealand bumble bees (Felden et al. 2021; Fernandez de Landa et al. 2024; Liu et al. 2023; Soares et al. 2025). Chemical and antimicrobial exposures also provide well-characterised routes by which disturbance can propagate from exposures to microbiome change and phenotypic traits, e.g., (Alberoni et al. 2021; Motta et al. 2018; Raymann et al. 2017). Finally, external microbiomes such as the colony microbiome (Anderson and Copeland 2024) and the flower microbiome (Smutin et al. 2022) can interfere with the gut microbiome of social bees.

Together, these studies emphasise that realised exposures are heterogeneous and context dependent, particularly with respect to timing, co-occurring stressors, developmental/task context and landscape. A structured synthesis

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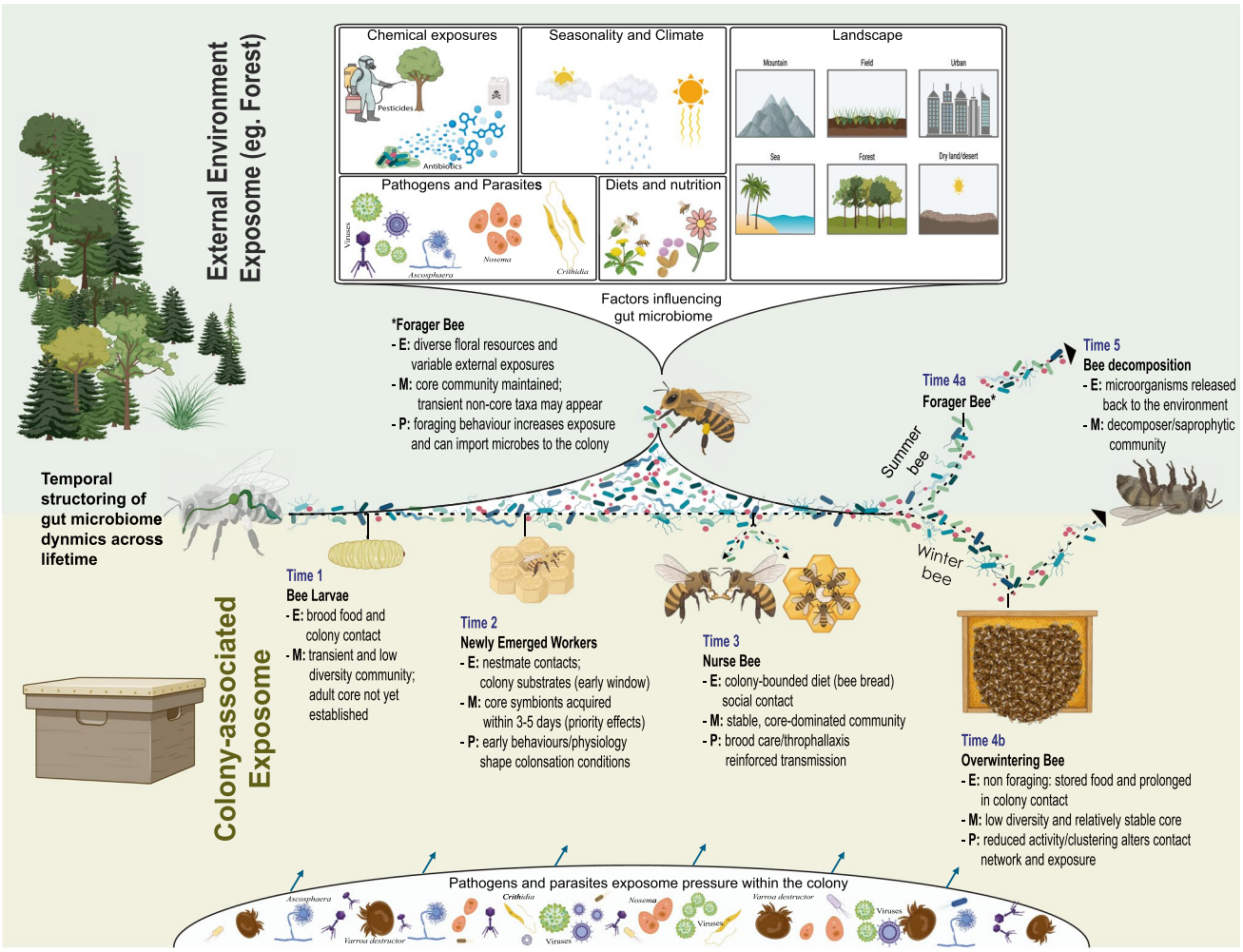

Fig. 2 Temporal and environmental structuring of gut microbiome dynamics across the social bee life cycle. The figure illustrates how gut microbiome assembly and restructuring are shaped over time by colony-associated and external environmental exposures. During early life, larvae and newly emerged workers are primarily exposed to brood food, nestmates and colony substrates, whereas nurse bees experience predominantly within-colony dietary and social inputs. With the transition to foraging, workers become a major interface between the colony and the external environment and encounter diverse exposures, including floral resources, chemicals, pathogens and parasites, climatic and landscape conditions, and microbial reservoirs associated with flowers, other wild bees, soil and water. The factors illustrated are representative rather than exhaustive; likewise, pathogens and parasites are highlighted within the colony environment as important biotic exposures but do not represent all colony-associated exposures. Although external factors are shown primarily in relation to foragers for visual simplicity, they can also influence the colony environment through materials transported back to the nest. Foragers can therefore contribute to colony-associated microbial reservoirs and to exposures experienced by nestmates and subsequent colony stages. Seasonal changes further modify these patterns, producing distinct trajectories during active foraging and overwintering periods. The final post-mortem stage illustrates the potential release of host-associated microorganisms back into the environment following decomposition. Throughout the figure, E denotes realised exposures, M gut microbiome state, and P phenotypic or behavioural traits that can modify subsequent exposure and microbial transmission. Figure created with BioRender.com

linking exposome drivers to microbiome shifts and phenotype outcomes (including directionality, context dependence and bee taxa) is provided in Supplementary Appendix 2.

Gut microbiome (M): mechanistic mediator linking genome and exposome to the phenome

In GEM–P, the gut microbiome is a key mediator linking the host genome (G) and lifetime exposures (E) to observable traits (P). Host genotype can shape microbiome assembly by altering gut conditions and immune filtering, which favours some strains and functions over others (G → M). Exposures can also restructure the microbiome by altering microbial inputs and gut conditions (E → M). In turn, core gut symbionts metabolise dietary and environmental inputs into bioactive metabolites (Kešnerová et al. 2017; Zheng et al. 2018) and interact with host immune and neural pathways (M → P). Together, this provides a mechanistic route by which G and E can influence phenotypic traits via the microbiome (G/E → M → P), alongside direct G → P and E → P effects.

A holobiont perspective treats the host and its associated microbiota as an integrated functional unit, emphasising that many traits we measure (e.g., digestion, detoxification, immune activity) arise from host–microbe interactions rather than from the host alone (Braglia et al. 2025; Theis et al. 2016; Zilber-Rosenberg and Rosenberg 2008). We use this as a functional lens, but GEM–P keeps the host genome (G), exposome (E), microbiome (M) and phenome (P) as distinct nodes to maintain analytical clarity. This separation allows us to distinguish direct effects (e.g., G → P, E → P) from microbiome-mediated pathways (e.g., G → M → P, E → M → P) and to represent feedback explicitly.

An important scope limitation of the present GEM–P formulation is that the microbial dimension is represented by a single gut microbiome node. Social bee colonies contain multiple microbial compartments that differ in persistence, transmission, ecological assembly and degree of host association, and GEM–P does not assume that these communities are biologically equivalent. The gut microbiome is used here as a deliberately restricted focal compartment because it currently provides the strongest mechanistic basis for linking host genotype, exposure history and measurable host phenotypes. Where other microbial compartments are central to a particular hypothesis, the framework can be expanded by representing them as additional interacting microbial nodes.

Microbiome acquisition and early life assembly

Microbiome assembly in social bees across developmental stage and task, season, and landscape. These are the same spatiotemporal axes that structure the exposome (Supplementary Box S1; Fig. 2).

Social bees harbour a relatively simple, specialised gut microbiome dominated by a small set of core host-associated taxa. These microbes are acquired primarily through nestmate contact and hive substrates, rather than through direct uptake from the wider environment (Engel et al. 2016; Kwong and Moran 2015, 2016; Zheng et al. 2018). Newly emerged adults are nearly germ-free and acquire core symbionts within a short early window (≈3–5 days post-emergence (Kwong and Moran 2016). During this period, the order and timing of exposure can generate persistent, strain-level differences (Jones et al. 2025; Powell et al. 2016). In GEM–P terms, microbiome establishment reflects both the available microbial inputs (E) and how exposure occurs (P) through social contact and behaviour (E + P → M). This contributes to stable colony-structured microbiome profiles.

Functional links from microbiome to traits

Functionally, the microbiome contributes to nutrition, immune function, and behaviour (M → P). In many cases, strain-level functional differences are more closely associated with phenotypic outcomes than broad taxonomic shifts (Kwong and Moran 2016; Motta and Moran 2024) Experimental work, including gnotobiotic (germ-free) and mono-colonisation designs, supports effects on metabolic signalling and growth, colonisation resistance and immune homeostasis, and neurochemical and behavioural phenotypes (e.g., Raymann and Moran 2018; Raymann et al. 2017; Steele et al. 2021; Wu et al. 2021; Zheng et al. 2017) Comparative metagenomics further indicates

substantial functional diversity among strain-discrete populations, with implications for digestion, defence, and neuroactive metabolite potential. Functionally, the microbiome contributes to nutrition, immune function, and behaviour (M → P). In many cases, strain-level functional differences are more closely associated with phenotypic outcomes than broad taxonomic shifts predict phenotypic outcomes more directly than broad taxonomic shifts.

Overall, the bee gut microbiome is characterised by a small, specialised core, complemented by low-abundance and transient taxa. Social transmission and spatial niche structure help stabilise microbiome states at the colony level. Assembly is strongly shaped by early-life exposure routes and priority effects (E + P → M) (Jones et al. 2025) and is further stabilised by within-community interactions (e.g., competition, cross-feeding) and host filtering (G/P → M; M → M). Functionally, microbial metabolism, defence, and neuro-behavioural modulation provide major routes by which genome and exposure are translated into phenotypes (G/E → M → P), with strain-level gene repertoires often determining realised outcomes. Detailed mechanistic examples and supporting evidence for this section are provided in Supplementary Appendix 3.

Phenome (P): individual- and colony-level traits as readouts and feedback drivers

In GEM–P, the phenome (P) refers to measurable traits (Houle et al. 2010) of individual bees and colonies that emerge from interactions among the genome (G), exposome (E) and microbiome (M). Within GEM–P, chronological age and developmental stage are treated primarily as temporal or developmental context rather than as exposures; associated physiological and behavioural states are represented within P, while task transitions are represented as changes in P. These traits include physiological, immune and behavioural states at the individual level, and emergent performance outcomes at the colony level; expanded definitions and examples are provided in Supplementary Appendix 4. Conceptually, P has two complementary roles. First, it provides the observable outcomes through which the effects of G, E and M are assessed. Second, it feeds back on the system because phenotypic traits, especially behaviour and social organisation, reshape later exposures and routes of microbial transmission (P → E → M), often with time lags (Naug 2008).

Physiological and behavioural traits as both outcomes and mediators

Physiological and immune states (e.g., basal immune activity, barrier integrity, gut homeostasis) translate genomic and exposomal inputs into within-host conditions that can favour or suppress particular microbial strains (G/E → P → M) while also capturing microbiome-mediated effects on host health and function (G/E → M → P) (Evans and Spivak 2010; Guo et al. 2023). Behavioural phenotypes play a similar dual role. Many behaviours are heritable or genotype-biased (G → P), yet behaviour and colony organisation also determine how exposures circulate within the colony (G → P → E). By shaping contact networks, behaviours directly structure microbial transmission (P → E → M) (Johnson 2010). By shaping contact networks, behaviours directly structure microbial transmission (P → E → M).

Age-related division of labour illustrates this clearly: nurse bees experience dense within-hive contact and repeated exposure to nestmate-associated microbes, whereas foragers encounter a broader and more variable set of external inputs. Thus, a behavioural or task transition, such as the nurse-to-forager transition, is represented as a change in P, whereas the resulting changes in diet, social contact and environmental encounter constitute changes in realised E. These changes in realised exposure can increase inter-individual variation in exposure and microbial composition.

From individual traits to colony outcomes

At the colony scale, outcomes such as brood production, productivity and overwinter survival reflect the aggregation of individual phenotypes and the structure of social organisation. Colony genetic structure (e.g., polyandry) can also influence contact networks and task allocation, thereby shaping exposure patterns and microbial flow (Mattila and Seeley 2007; Seeley and Tarpy 2007). At the colony scale, outcomes such as brood production, productivity and overwinter survival reflect the aggregation of individual phenotypes and the structure of social organisation. Colony genetic structure (e.g., polyandry) can also influence contact networks and task allocation, thereby shaping exposure patterns and microbial flow.

Additional detail on phenotypic readouts and feedback pathways is provided in Supplementary Appendix 4, and illustrative hypotheses derived from GEM–P are provided in Supplementary Table S1.

Operationalising GEM–P

GEM–P provides a structured way to move from observed associations to alternative pathway-specific hypotheses. It does not constitute a formal causal-inference method in itself and does not establish the direction or strength of a relationship; rather, it makes alternative direct, mediated and feedback pathways explicit, thereby helping researchers identify which measurements, temporal contrasts, and experimental manipulations would be needed to distinguish among them. A central implication of this framework is context dependence: similar microbial change may have different phenotypic consequences depending on life stage, task, exposure history and colony transmission structure. Since pathway plausibility is context dependent, study designs should account for relevant differences in life stage or task, exposure history and colony transmission structure.

Study design: testing GEM–P pathways

Operationally, GEM–P highlights study designs that reduce uncontrolled heterogeneity and improve discrimination among alternative pathway-specific explanations without requiring comprehensive multi-omics. Each GEM–P pathway can be translated into a testable hypothesis. Illustrative hypothesis templates are provided in Supplementary Table S1. Where feasible, sampling stratification by temporospatial factors repeatedly linked with microbiome variation in social bees (e.g., age/task class, season, landscape context) can improve interpretability. By contrast, pooling across such states may conflate distinct microbiome regimes and obscure biologically meaningful shifts.

In parallel, exposures should be treated as measurable variables. Even coarse indices, such as diet breadth, pesticide or treatment history, pathogen load, foraging opportunity and management context, can help separate exposure-driven variation from host-genetic or transmission-related effects. Since the colony is a transmission unit, study designs should account for colony identity and genetic structure (e.g., queen lineage and mating regime, and patriline structure where feasible), together with traits that shape contact networks and microbial flow.

Where microbiome-to-phenotype hypotheses are evaluated, combining a perturbation with microbiome readouts (functional and strain-resolved where feasible) and intermediate phenotypes (e.g., gut homeostasis, immune tone, metabolic condition; see Supplementary Appendix 4 for phenotype readouts) helps distinguish mechanistic microbiome effects from patterns that primarily track upstream exposure. A practical measurement checklist aligned to GEM–P pathways is provided in Supplementary Box S2.

Applied and evolutionary extensions

GEM–P also provides an applied framework for considering the conditions under which nutritional, chemical and microbiome-targeted interventions may succeed or fail under different exposure and transmission regimes (Supplementary Box S3). For example, supplementation with beneficial strains and/or plant extracts has been reported to shift gut microbiota and improve colony-level outcomes, improve vitellogenin accumulation in haemolymph, and reduce *Nosema* infection, but effects are context dependent (Alberoni et al. 2018; Garrido et al. 2024).

The framework also links within-lifetime G–E–M–P relationships to fitness and selection across generations, motivating longitudinal and comparative study designs (Supplementary Box S4).

Scope and limitations of GEM–P

GEM–P is intended as an organisational and hypothesis-generating framework, and its utility depends on the quality and temporal resolution of the data used to evaluate specific pathways. A major practical limitation is the exposome, which represents a heterogeneous set of physical, chemical, biological and socially mediated exposures that usually cannot be measured comprehensively. In practice, studies will often rely on selected exposure variables or proxies relevant to the pathway being tested. Similarly, distinguishing temporally ordered and feedback relationships may require repeated sampling or longitudinal designs that can be difficult to implement at both individual and colony scales. GEM–P therefore does not require simultaneous measurement of all G, E, M and P components; rather, measurements should be selected according to the specific pathway and competing explanations under study.

The present formulation also deliberately restricts M to the gut microbiome and therefore does not capture the full microbial heterogeneity of the social bee colony. This limitation, and the potential extension of GEM–P to additional microbial compartments, are discussed in Sect. 2.3. More broadly, representing genomic, exposomal, microbial and phenotypic variation as four components necessarily simplifies biological variation within each component and across levels of organisation.

Conclusions

GEM–P is an integrative conceptual framework for organising evidence on how host genomes, lifetime exposures and gut microbiomes jointly shape phenotypic variations in social bees, while explicitly incorporating feedbacks via behaviour and social transmission. The individual relationships represented in GEM–P are largely grounded in established host–microbiome, gene–environment and ecological theory; the contribution of the framework lies in bringing these relationships into a common structure specific for social bees and making temporal and biological contexts explicit. By integrating evidence across biological scales and accounting for temporospatial variation, GEM–P provides a coherent way to interpret context dependence, formulate pathway-specific hypotheses and identify alternative explanations for gut microbiome–phenotype associations. It also guides study design choices, including sampling stratification, temporal measurements and targeted perturbations, that can improve mechanistic interpretation and help distinguish among competing explanations. Together, this positions GEM–P as an organisational and hypothesis-generating scaffold for research on social bees.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Ethics approval None.

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Declaration of generative AI and AI-assisted technologies in the writing process During the preparation of this manuscript, the authors used ChatGPT for language editing (typo correction and readability improvement). All content was subsequently reviewed and edited by the authors, who take full responsibility for the final manuscript. Biorender.com was used to assist in generating elements of Fig. 2 (Created in BioRender. Alberoni, D. (2026) https://BioRender.com/eu3qhzn).

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