International Journal for Parasitology: Parasites and Wildlife 17 (2022) 319–326

Contents lists available at ScienceDirect

International Journal for Parasitology: Parasites and Wildlife

journal homepage: www.elsevier.com/locate/ijppaw

The threat of pesticide and disease co-exposure to managed and wild bee larvae

Monika Yordanova[a,*], Sophie E.F. Evison[b], Richard J. Gill[a], Peter Graystock[a]

[a] Imperial College London, Silwood Park, Buckhurst Road, Berks, SL5 7PY, UK
[b] School of Life Sciences, University Park, Nottingham, NG7 2TQ, United Kingdom

ARTICLE INFO

Keywords:
Larval parasite
Neonicotinoid
Haemocytes
Larval pathogen
Agrochemical

ABSTRACT

Brood diseases and pesticides can reduce the survival of bee larvae, reduce bee populations, and negatively influence ecosystem biodiversity. However, major gaps persist in our knowledge regarding the routes and implications of co-exposure to these stressors in managed and wild bee brood. In this review, we evaluate the likelihood for co-exposure to brood pathogen and pesticide stressors by examining the routes of potential co-exposure and the possibility for pollen and nectar contaminated with pathogens and pesticides to become integrated into brood food. Furthermore, we highlight ways in which pesticides may increase brood disease morbidity directly, through manipulating host immunity, and indirectly through disrupting microbial communities in the guts of larvae, or compromising brood care provided by adult bees. Lastly, we quantify the brood research bias towards *Apis* species and discuss the implications the bias has on brood disease and pesticide risk assessment in wild bee communities. We advise that future studies should place a higher emphasis on evaluating bee brood afflictions and their interactions with commonly encountered stressors, especially in wild bee species.

1. Introduction

Bees (Anthophila) are a diverse and important clade encompassing over 20,000 species and are vital for the pollination services they provide to wild and managed landscapes. Many commercial orchard crops such as almonds, apples and peaches rely heavily on bees to increase yields (Allsopp et al., 2008; Higo et al., 2004; Life, 2021). Therefore, bees are crucial to produce diverse crops and provide a variety of nutrients essential for the human diet (Eilers et al., 2011). Although the primary bee species pollinating croplands are managed species, wild pollinators can also be used to increase crop yields in some commercial set-ups (Sánchez et al., 2001; McGrady et al., 2020). Furthermore, wild native bees are vital for unmanaged ecosystem biodiversity as they have a higher propensity to seek out native and rare plant species, therefore maintaining heterogeneity in these habitats through facilitating plant diversity (Mogren et al., 2020).

Bee populations have suffered declines in recent years primarily due to increasing pressure from stressors including pathogen spillover and pesticide exposure (Hristov et al., 2021; Olynyk et al., 2021; Wood et al., 2020c; Graystock et al., 2016). Worryingly, pathogen and pesticide research commonly ignore the impact these stressors have on the larval/brood stage of bee development in favour of focusing on adult bees. The period of larval development and pupation of a bee (commonly referred to as brood-stage) is a vulnerable yet essential life stage of all bee species. Due to their immobility, bee brood are dependent on adult bees to forage on flowers and provide them with food, however, pathogens and pesticides can become incorporated in brood food leaving them vulnerable to co-exposure to both stressors (James, 2011; Mullin et al., 2010). Pathogens can include viruses, bacteria, fungi or mites. Those that cause disease in the brood are known as 'brood pathogens' and have important effects on the health of both solitary and social bee populations. In solitary bees, a female will construct a nest and collect provisions that are deposited in cells within the nest along with an egg (Royauté et al., 2018). All solitary bee brood then develop in isolation into reproductively capable adults. Therefore, brood infections for such species can directly reduce the number of reproductive individuals available for subsequent generations, thereby reducing the populations of solitary bees and diminishing the pollination services they provide (Evison and Jensen, 2018). Eusocial species live in colonies where reproductive females can lay both reproductive and sterile brood (Hartfelder et al., 2006). In such colonies, sterile adult bees (worker bees) forage to provide for the developing offspring (Hartfelder et al.,

---

* Corresponding author.
E-mail address: mpy20@ic.ac.uk (M. Yordanova).

https://doi.org/10.1016/j.ijppaw.2022.03.001
Received 26 November 2021; Received in revised form 28 February 2022; Accepted 1 March 2022

2006). Therefore, brood infections can either directly reduce the population through affecting reproductive individuals (Murray et al., 2019), or indirectly through diminishing colony strength (number of workers) (Evison, 2015). Some pathogens can pose greater spillover risk due to their high adaptability and wide distribution, increasing the risks they pose to a wider range of bee species (Evison and Jensen, 2018). The presence of a stressor in addition to brood disease, such as co-exposure to pesticides can increase infection-induced mortality, however, our understanding of such co-exposures is limited (López et al., 2017). This knowledge gap is further widened for non-*Apis* species which may have major implications on our understanding and management of brood disease and pesticide exposure. Many wild bees have shown significant population declines, increasing the urgency to address this area of concern (Zattara and Aizen, 2020).

In this review we:

1) discuss how developing brood may commonly become co-exposed to brood pathogens and pesticides;
2) highlight the mechanisms underlying the interactions between these factors contributing to reduced brood survivorship;
3) quantify the scale of taxonomic bias prevalent in bee brood pathogen and pesticide research and examine the implications of this bias on our understanding of these research areas and the management practices that result from them.

2. Brood pathogen and pesticide co-exposure

Pathogen and pesticide exposure are implicated as major drivers of decline in bee populations, health, and range (Hristov et al., 2021; Olynyk et al., 2021; Wood et al., 2020c). For bee larvae, the route of exposure to both stressors can be the same, increasing the likelihood of their co-exposure. Gaining a deeper understanding of how co-exposure can occur, and evidence of its frequency and implications to bee health is important for developing effective conservation and management strategies.

Flowers are a shared food resource among bees, and will typically be visited multiple times per day by various species, but they may expose bees to more than just food. When contaminated adult bees forage on flowers, they can shed the transmission stage of pathogens, i.e. spores or virions, thus contaminating the flowers and facilitating their transmission to other foraging bees (Alger et al., 2019; Graystock et al., 2015, 2020). The initial shedding of pathogens may be via defecation, oral transfer, or via cuticle contamination, and this mechanism of horizontal transmission seems prevalent across multiple species of adult bee pathogens (Bodden et al., 2019; Schorkopf et al., 2007). Once pathogen-contaminated food is collected, it is deposited in the nest alongside the brood and fed to larvae. Though studies have not yet shown the role of flowers in brood pathogen transmission, the primary way larvae become infected with brood diseases is known to be through contaminated food (pollen/nectar) (Aronstein and Murray, 2010; Forsgren, 2010), and a diversity of viral, bacterial, fungal and mite pathogens and pests have been detected on flowers or in pollen (Graystock et al., 2013; Peck et al., 2016; Singh et al., 2010; Yousefi and Fouks, 2019). This route of pathogen exposure likely also poses a pesticide exposure risk to larvae. Pesticides including insecticides, fungicides, and herbicides are frequently sprayed onto plants and their residues are found to remain in pollen and nectar (McArt et al., 2017; Pohorecka et al., 2012; Tort et al., 2005). Likewise, systematic insecticides applied to seeds can be taken up into plant nectar and pollen (Wood and Goulson, 2017). Furthermore, pesticide residues can remain in water and soil, where the spores of some brood pathogens can also persist, providing an additional route of co-exposure in species which forage for water or nest in soil (Sglostra et al., 2019). Honey bees can also be exposed to high concentrations of miticides within their nests to control *Varroa* mites (Mullin et al., 2010). The pesticide residues collected from the foraging landscape are readily integrated into brood food and bee nest materials (McArt et al., 2017; Mullin et al., 2010; Traynor et al., 2021). A survey of pesticide residues in North American hives showed that 10 pesticides had higher than a tenth of their LD_{50} doses prevalent in stored pollen, indicating a high potential for exposure to the developing larvae (Mullin et al., 2010). Within honey bee hives miticides and fungicides are encountered at the highest rates (Mullin et al., 2010; Traynor et al., 2021). As both pesticides and brood pathogens are encountered in the environment and provisioned to larvae through brood food, the pollen and nectar collected by bees can likely be contaminated with both stressors posing possible synergistic risks.

Across a landscape, different plant species vary in their likelihood to become pathogen transmission hubs or to be treated with pesticides (Lentola et al., 2017; Graystock et al., 2020). Land management schemes developed to encourage healthy bee populations are often focussed on improving bee nutrition and reproductive success, rather than alleviating pesticide and pathogen exposure risks (European Commission, 2017). Wildflowers planted to improve bee foraging success may, if grown near agricultural lands, become contaminated with pesticides either through drift from sprays or dust from abraded seeds treated with pesticides (Wood and Goulson, 2017). Ornamental flowers sold as “pollinator-friendly” are often treated with a wide mix of pesticides (Lentola et al., 2017). Furthermore, different floral species can be more likely to act as transmission hubs for adult bee pathogens and floral traits seem to be important in augmenting transmission potential, however, little is known about how flower species and floral traits influence brood pathogen transmission (Adler et al., 2018; Graystock et al., 2020; Figueroa et al., 2020). Research should therefore aim to address the gaps in this knowledge to identify floral traits that influence brood pathogen and pesticide exposure risks to bees, enabling informed land-management and conservation schemes that may alleviate co-exposure to these stressors.

3. Pesticide effects on brood disease

Most research on the interactions between pesticides and pathogens focuses on adult bees, however, co-exposure to these stressors in developing brood could influence brood health through a variety of direct and indirect mechanisms (Fig. 1). These interactions ultimately reduce the survivorship prospects of larvae and could therefore pose risks to bee populations. Brood exposure to pesticides can directly reduce their cellular and humoral immune responses to pathogens (López et al., 2017). This lessens their ability to quickly or adequately respond to pathogen exposure and increases the lethality of infections (López et al., 2017; Wood et al., 2020a). Brood health may also be diminished indirectly by pesticide exposure. This is mediated either through disrupting the health of adult bees which larvae are dependent on (Al Toufailia et al., 2016; Medina et al., 2009) or perturbing the microbial composition of larval guts (Vásquez et al., 2012). Because of their impact on larval survivorship, some pesticides indirectly reduce the prevalence of brood pathogens in the environment by lowering the abundance of their hosts or reducing the survivorship of certain pathogens.

3.1. *Pesticides directly affect brood disease through manipulating larval immune responses*

For honey bees, immunity is modulated through four non-autonomous pathways linked to different aspects of host defence, responsible for pathogen recognition, signalling, and the production of effectors such as antimicrobial peptides (Evans et al., 2006). Exposure of adult honey bees to neonicotinoid insecticides can downregulate transcription factors involved in these pathogen defence systems, thereby increasing the susceptibility of bees to pathogens (di Prisco et al., 2013; Pamminger et al., 2018). Whilst this direct interference of adult bee immunity following pesticide exposure could act in similar fashion in larvae, there are few studies characterising the mechanisms by which

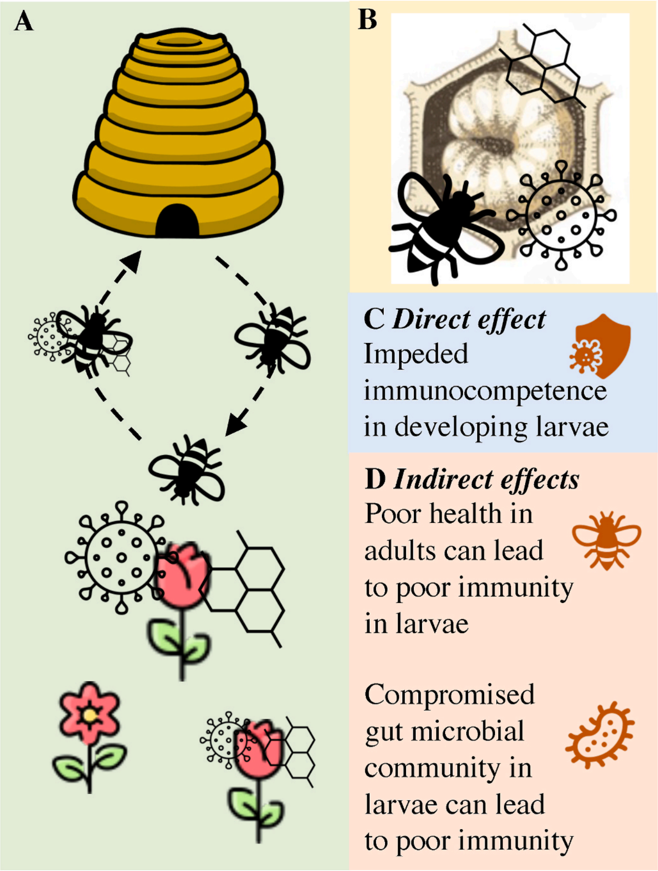


Fig. 1. Flowers contaminated with brood pathogens and pesticides can lead to simultaneous exposure to pesticides and brood pathogens from flowers in adult foraging bees (A). This leads to brood being co-exposed to the stressors via food provisioning (B). Pesticides may increase larval mortality from brood infections directly through compromised immunocompetence (C), and/or indirectly through manipulating microbial communities and compromising food provisions from adult bees (D) (Icons8, 2022).

pesticides influence larval immunity and particularly how they interact with brood infections.

Some insecticides can exacerbate the effect that brood pathogens have on larval health, by reducing haemocyte production. Haemocytes are an important component of bee larval immunity whereby upon infection haemocytes can replicate and migrate to kill pathogens via phagocytosis (Hillyer, 2016; Wilson-Rich et al., 2008). This response is stronger in larvae, relative to any other life stages in honey bees, indicating its potential importance for tackling brood infections (Wilson-Rich et al., 2008). The interactive effect between brood pathogens and pesticides on haemocyte production has been demonstrated for honey bee larvae inoculated with *P. larvae* bacteria, the causative agent of the destructive American foulbrood (AFB) disease (López et al., 2017). Larvae exposed to the organophosphate, dimethoate, or the neonicotinoid, clothianidin, had reduced haemocyte cell counts leading to increased larval mortality in a synergistic manner (López et al., 2017). A similar reduction in haemocyte production has been observed in the fruit fly *Drosophila melanogaster* responding to neonicotinoid and organophosphate exposure (Rajak et al., 2015; Walderdorff et al., 2019). Further work should aim to assess the role of haemocyte production in mitigating bee brood disease for a wider range of brood infections and investigate which pesticides can negatively influence this response to ensure the introduction of adequate regulatory measures.

3.2. Pesticides can indirectly increase brood disease lethality

Pesticides that negatively influence the health and behaviour of adult bees can indirectly diminish brood health due to reducing care or food provisions by adult bees which has cascading effects on the ability of larvae to combat pathogens. Honey bee workers may be more vulnerable to pesticide toxicity relative to larvae (Wood et al., 2020b), however, due to their roles in maintaining larval health this can result in indirect negative consequences to larval health. Adult bees of social species have a repertoire of behaviours that provide a ‘social immunity’ such as the removal of infected brood to reduce infection risks, a behaviour common in stingless bees (Al Toufailia et al., 2016; Medina et al., 2009), and the sterilisation of brood food to reduce exposure to pathogens (López-Uribe et al., 2017). However, a variety of pesticides including neonicotinoid insecticides and chlorothalonil fungicides influence adult cognition, behaviour, and ability to forage (Gill and Raine, 2014; Lima et al., 2016; Stanley et al., 2016; O’Neal et al., 2019). Pesticide exposure in larvae can also have delayed effects for future generations of larvae due to their potential to disrupt development and cognition leading to impaired behavioural performance in later-life as adult bees (Smith et al., 2020). The inability to properly feed or provision brood will have obvious effects on their general health and ability to fight pathogens. However, impacts on the way that adult bees interact with larvae or how they provision larval food may also influence the ability of the developing larvae to acquire important gut microbes (Hroncova et al., 2015; Kwong and Moran, 2016; Menezes et al., 2015). Bee larvae acquire their gut microbial communities from the adults that feed them, but adults exposed to some pesticides can lose their own key gut microbes both reducing their own health and their likelihood to transmit key microbes, which could lead to compromised brood health (Hroncova et al., 2015; Kwong and Moran, 2016).

Bee larvae immunity may be mediated through microbial communities in their guts, therefore, pesticides that influence these communities could influence brood disease susceptibility. Microbial communities in the guts of adult honey bees can play important roles in development, nutrient acquisition, and pathogen avoidance (Daisley et al., 2020a). Shifts in the microbial communities in adults can lead to dysbiosis, which is characterised by a loss of functionally important microbes in bee guts (Anderson and Ricigliano, 2017). Although less widely explored, microbial communities in larval guts may also impact the proliferation of pathogenic microbes and their microbial communities may also suffer as a result of pesticide exposure and brood infections (Yu et al., 2021; Vásquez et al., 2012; Floyd et al., 2020; Erban et al., 2017). Therefore, a pesticide that can induce dysbiosis in bee larvae may increase their vulnerability to brood pathogens through the loss of symbionts which mediate immune function, nutrition, or development (Yu et al., 2021).

Gaining a greater understanding of the indirect interactions between brood pathogens and pesticide exposure could improve the health of both managed and wild bee species. To understand the effects pesticides and pathogens have on brood health, further work should examine the role of microbial symbionts in mediating their interactions. If microbes affect the interaction between pesticide and brood pathogens, this could pave the way for the use of probiotic supplements to alleviate the effects of these stressors in managed bee species (Daisley et al., 2020a; Daisley et al., 2020b; Floyd et al., 2020). Furthermore, given the importance of adult bees to brood health, researchers could utilise the more easily accessible adult bees as bioindicators of brood infections. Gaining a greater understanding of the symptoms of brood infections in adult bees can be useful for monitoring brood infections in wild bee species, where brood may not be as easily accessible to researchers as foraging adults. Through this, monitoring schemes can be introduced to moderate stressors which increase brood disease incidence such as pesticide exposure.

3.3. Pesticides may reduce brood disease incidence by reducing host prevalence or pathogen survivorship

As pesticides can reduce bee populations, they may in some cases decrease brood pathogen prevalence. A UK-based study surveying pollinator communities across agricultural field sites found diminished species richness across insect pollinators and lower abundances of solitary bees, honey bees, hoverflies and wasps in agricultural sites with higher use of pesticides (Evans et al., 2018). This is unsurprising as high pesticide use can reduce insect survivorship and pollinator diversity (Rundlöf et al., 2015; Arce et al., 2017). However, surveying pathogen prevalence in *Osmia bicornis* and *Megachile* spp. within those field sites showed reduced prevalence of *Ascosphaera* in field-sites with increased pesticide use (Evans et al., 2018). *Ascosphaera* is a fungal pathogen that can cause chalkbrood disease in developing larvae of a wide range of bee species (Jensen et al., 2009; Reynaldi et al., 2015; Rust and Torchio, 1992). One explanation for this reduced pathogen prevalence could be a reduction of the fungal pathogen due to high fungicide use (Evans et al., 2018). Another study found that high fungicide use similarly reduces *Ascosphaera* incidence in adult bees of *Osmia cornifrons* (Krichilsky et al., 2021). However, they also found an additive effect in larval mortality as a result of *Ascosphaera* infection and fungicide exposure (Krichilsky et al., 2021). This may indicate that the reduced incidence of the pathogen in adults may be due to increased lethal effects in larvae (Evans et al., 2018; Krichilsky et al., 2021). Furthermore, reduced density of adult bees can lessen the pathogen's ability to be transmitted across the environment, as adults can also disperse the pathogen (James and Pitts-Singer, 2005; Maxfield-Taylor et al., 2015; Evans et al., 2018). The reduction of pollinator species richness and abundance can further minimise the prevalence of the fungus as other pollinators can also carry it across the foraging landscape (Evison et al., 2012; Evans et al., 2018). Therefore, the contribution that pesticides may have on lowering survivorship in bee larvae, pollinators and microbes could in some cases lead to reduced brood pathogen prevalence. However, further work should be undertaken to examine this effect across bee genera, brood pathogens, and pesticide types.

4. Research bias

We conducted systematic searches of studies across bee taxa and terms associated with brood pathogens or pesticide exposure to determine research biases. Logically, absence of bias would mean the number of research papers found in various bee genera and families should proportionally reflect the number of species in these taxa. A list of bee family and genus names were obtained from the Discover Life database (Life, 2021) and each was individually used on *The Web of Science* to identify the spread of research focus across scientific literature since 1900. Each taxa was searched for alongside terms related to brood disease and pesticide exposure. Manuscripts from the resulting searches were identified and manually curated to produce data sets for each search conducted (full details in Supplementary Methods, Supplementary Fig. 1, Supplementary Table 1).

We found the distribution of research published across each bee taxa to not be proportional to bee diversity, suggesting a bias in research towards some, and away from other bee taxa (Fig. 2, Supplementary Table 1, Supplementary Fig. 2). Research assessing pesticide exposure only covered 40 (24.69%) of the 162 bee genera (Life, 2021), and only 14 genera had research assessing their brood pathogen prevalence or defences (8.64%). Furthermore, 83.69% (13,309 species) belong to genera that have no studies related to their brood infections and 43.66% (6944 species) belong to genera that have no studies related to their pesticide exposure risks. As expected, the brood disease and pesticide exposure research bias strongly favours *Apis* bees (Fig. 2, Fig. 3 and Fig. 4).

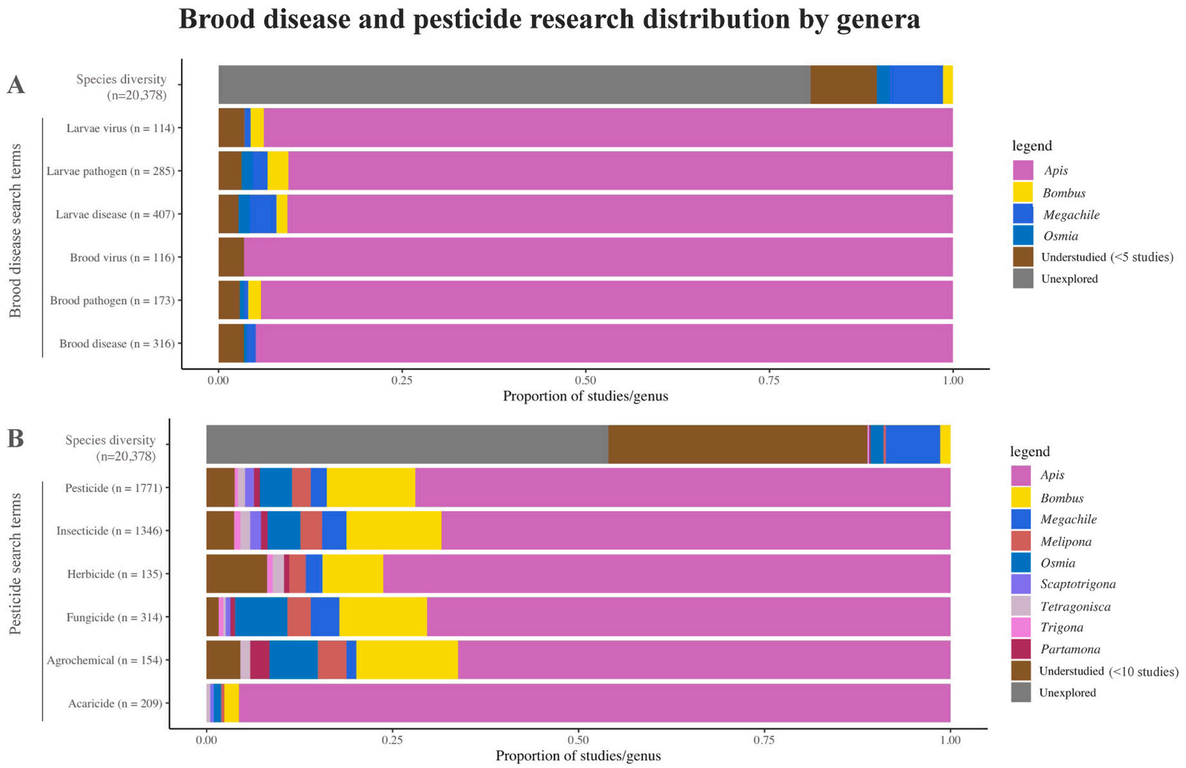
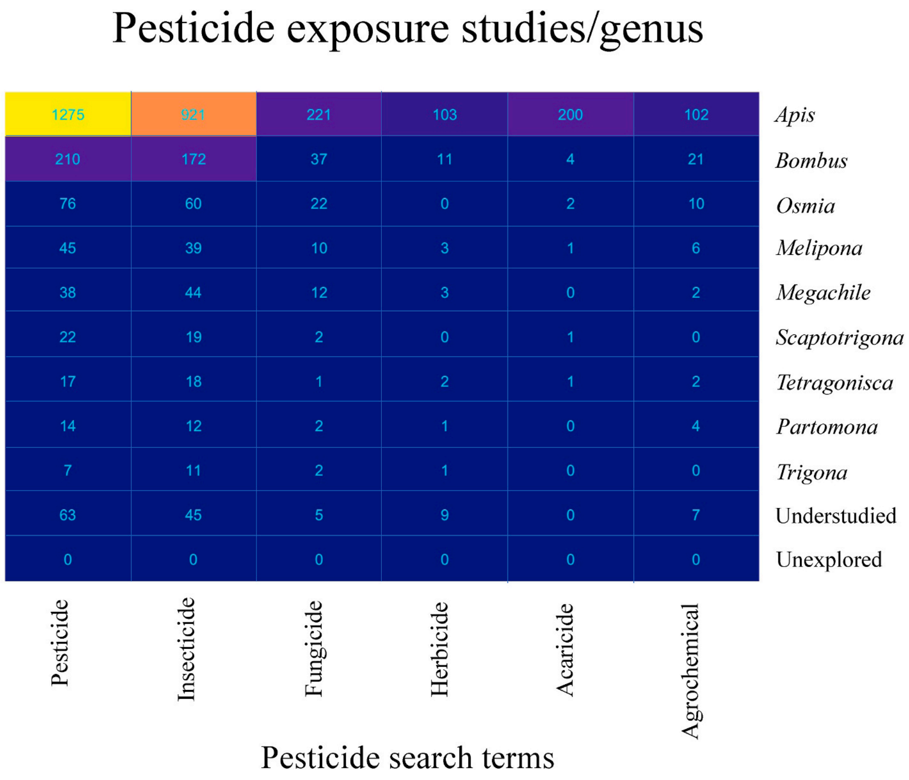


Fig. 2. Proportion of results per search term across bee genera on the *Web of Science* search engine (n = the total number of studies corresponding to each search term). Search terms related to brood disease (A) and pesticide exposure (B) are both compared to species diversity at the genus level. Genera with less than 5 studies in any brood disease search term (A) and less than 10 studies across any pesticide exposure search term (B) have been classified as 'Understudied' (brown) and grouped. Genera with no studies related to any brood disease search term (A) and no studies related to any pesticide exposure search term (B) have been classified as 'Unexplored' (grey) and grouped. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)



	Pesticide	Insecticide	Fungicide	Herbicide	Acaricide	Agrochemical
Apis	1275	921	221	103	200	102
Bombus	210	172	37	11	4	21
Osmia	76	60	22	0	2	10
Melipona	45	39	10	3	1	6
Megachile	38	44	12	3	0	2
Scaptotrigona	22	19	2	0	1	0
Tetragonisca	17	18	1	2	1	2
Partomona	14	12	2	1	0	4
Trigona	7	11	2	1	0	0
Understudied	63	45	5	9	0	7
Unexplored	0	0	0	0	0	0

Fig. 3. Pesticide research bias across bee genera depending on pesticide type based on *Web of Science* searches. The number of studies per search term is indicated for each genus with at least 10 studies across search terms; Understudied is a cumulative group including genera which contain less than 10 studies; Unexplored is a cumulative group including genera which have no published papers for any pesticide exposure category. Warmer colours are used to indicate a higher number of studies related to a genus for the corresponding pesticide search term, while colder colours indicate a lower number of studies found. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

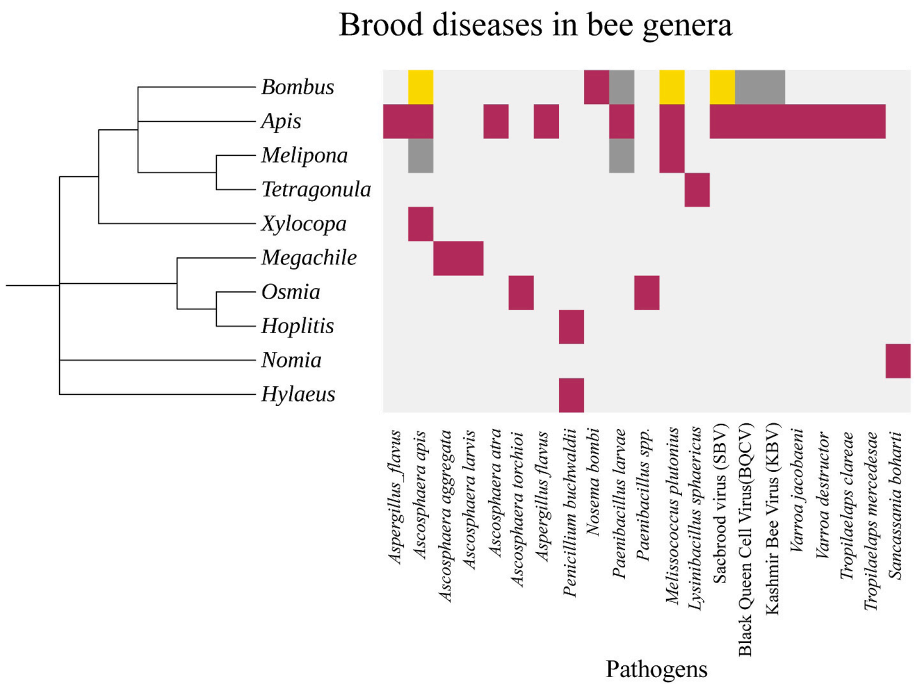


Fig. 4. Brood pathogens studies per bee genera found from *Web of Science* searches using search terms “brood disease”, “brood pathogen”, “brood virus”, “larvae disease”, “larvae pathogen” and “larvae virus” across bee genera. Red squares indicate that a pathogen on the x-axis has been found to infect at least one species in the bee genus corresponding to its position in the phylogeny shown on the y-axis; yellow squares indicate that the pathogen on the x-axis has been found in individuals from at least one species in the genus on the y-axis but no symptoms were reported in the studies; dark grey squares indicate that the pathogen on the x-axis has been tested for in at least one species in the genus corresponding on the y-axis, however has not been found; white squares indicate that the searches found no studies where any bee species of the genus on the y-axis were tested for in the corresponding pathogens on the x-axis. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

As anticipated, *Apis* dominate the scientific literature because of the wide array of studies devoted to the managed and commercially important honey bees, *Apis mellifera*. As crop pollinators, honey bees increase farm yields by £191.8 million in the UK alone, and the production of honey bee products including honey, propolis and royal jelly enhances their economic value (DEFRA, 2009; Leska et al., 2021). This economic importance is a primary driver for this disproportionate research focus. This has facilitated a greater depth of understanding of their brood diseases and susceptibility to pesticides more than any other bee genera (Leska et al., 2021; Wilson-Rich et al., 2008). For example, *in vitro* rearing techniques of honey bee larvae have been clearly outlined and are continuously improved which has made it easier to assess the effects of brood pathogens and pesticides on honey bee larvae relative to other bee species (Schmehl et al., 2016; Crailsheim et al., 2013; Wood et al., 2020a). However, in contrast to *A. mellifera*, the majority of bee species are solitary (Life, 2021; Sgolastra et al., 2019). This means the vast majority of brood disease and pesticide exposure research focuses on non-typical bee species, overlooks differences in life-history that likely have major implications, and limits our understanding of the severity of harm brood pathogen and pesticide exposure can have across bee species.

Nesting behaviour can vary greatly across bee species and may affect pesticide and pathogen exposure risks. Pesticides applied to the soil or in water can become integrated into the soil matrix, increasing risks for ground-nesting bees (Sgolastra et al., 2019). As these pesticides remain in the soil, they can incorporate into plant materials that can be used for nest building by a variety of bee species (Sgolastra et al., 2019). Some bees can produce substances that enable them to regulate the environment of their larvae, which may also serve to reduce pathogen exposure to the brood. Examples of this include the Colletidae family which produce a cellophane-like substance and stingless bees which produce an involucrum (Almeida, 2008; Cham et al., 2019). However, the role of such substances in preventing brood disease and pesticide exposure has not been examined. A lack of understanding of the factors which mitigate or enhance brood disease and pesticide co-exposure risks can impede the implementation of adequate conservation or management practices for non-*Apis* species.

A disproportionate research effort for *A. mellifera* relative to wild bees can result in management practices tailored to honey bee health, which can affect pathogen exposure dynamics. Bee species can vary in their niche breadth and foraging range - wild species have narrower ranges and interact with a more limited number of flowers compared to the generalist *A. mellifera* (Zemenick et al., 2021; Zurbuchen et al., 2010). A large number of generalists increase the prevalence of pathogens in the foraging landscape as they interact with a wider range of flowers and connect different microbial (and pathogen) networks (Graystock et al., 2020; Zemenick et al., 2021). Therefore, practices that favour honey bees relative to other pollinators, such as pesticide exposure risk assessments tailored to honey bee health, can result in increased pathogen spillover risks for wild bees (Sgolastra et al., 2019). Further efforts should therefore be directed to gaining a greater understanding of the brood pathogens and pesticides which impact wild bee health, and how these stressors interact with one another, in order to promote healthy bee communities.

5. Conclusion

This review outlines the gaps that persist in our knowledge of bee brood disease and pesticide exposure research. We highlight that further work should examine the risks for pesticide and brood disease co-exposure. As bees are likely to encounter pathogens and pesticides simultaneously in their environments, future research should aim to examine the potential means of interaction between these stressors across a wider range of pathogens and pesticides. Furthermore, an increased emphasis should be placed on evaluating these effects for non-*Apis* species to improve management practices for both wild and commercially used bees.

Funding

Funded jointly by the C. B. Dennis British Beekeeper's Research Trust and Bee Disease Insurance Limited.

Declaration of competing interest

No conflict of interest.

Acknowledgements

We would like to thank the Gill lab group for their comments and suggestions. We are grateful to the two anonymous reviewers for their positive and insightful comments which helped improve the content of the manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijppaw.2022.03.001.

References

Adler, L.S., Michaud, K.M., Ellner, S.P., Mcart, S.H., Stevenson, P.C., Irwin, R.E., 2018. Disease where you dine: plant species and floral traits associated with pathogen transmission in bumble bees. Ecology 99 (11), 2535–2545. https://doi.org/10.5061/dryad.0vm264s.

Al Toufailia, H., Alves, D.A., Bento, J.M.S., Marchini, L.C., Ratnieks, F.L.W., 2016. Hygienic behaviour in Brazilian Stingless bees. Biol. Open 5 (11), 1712–1718. https://doi.org/10.1242/bio.018549.

Alger, S.A., Burnham, P.A., Brody, A.K., 2019. Flowers as viral hot spots: honey bees (*Apis mellifera*) unevenly deposit viruses across plant species. PLoS One 14 (9), e0221800. https://doi.org/10.1371/journal.pone.0221800.

Allsopp, M.H., de Lange, W.J., Veldtman, R., 2008. Valuing insect pollination services with cost of replacement. PLoS One 3 (9), 3128. https://doi.org/10.1371/journal.pone.0003128.

Almeida, E.A.B., 2008. Colletidae nesting biology (Hymenoptera: Apoidea). Apidologie 39 (1), 16–29. https://doi.org/10.1051/apido:2007049.

Anderson, K.E., Ricigliano, V.A., 2017. Honey bee gut dysbiosis: a novel context of disease ecology. Curr. Opin. Insect. Sci. 22, 125–132. https://doi.org/10.1016/j.cois.2017.05.020.

Arce, A.N., David, T.I., Randall, E.L., Rodrigues, A.R., Colgan, T.J., Wurm, Y., Gill, R.J., 2017. Impact of controlled neonicotinoid exposure on bumblebees in a realistic field setting. J. Appl. Ecol. 54, 1199–1208. https://doi.org/10.1111/1365-2664.12792.

Aronstein, K.A., Murray, K.D., 2010. Chalkbrood disease in honey bees. J. Invertebr. Pathol. 103, S20–S29. https://doi.org/10.1016/j.jip.2009.06.018.

Bodden, J.M., Hazlehurst, J.A., Wilson Rankin, E.E., Rehan, S., 2019. Floral traits predict frequency of defecation on flowers by foraging bumble bees. J. Invertebr. Pathol. 19 (5), 1–3. https://doi.org/10.1093/jisesa/iez091.

Cham, K.O., Nocelli, R.C.F., Borges, L.O., Viana-Silva, F.E.C., Tonelli, C.A.M., Malaspina, O., Menezes, C., Rosa-Fontana, A.S., Blochtein, B., Freitas, B.M., Pires, C.S.S., Oliveira, F.F., Contrera, F.A.L., Torezani, K.R.S., Ribeiro, M.D.F., Siqueira, M.A.L., Rocha, M.C.L.S.A., 2019. Pesticide exposure assessment paradigm for stingless bees. Environ. Entomol. 48 (1), 36–48. https://doi.org/10.1093/ee/nvy137.

Crailsheim, K., Brodschneider, R., Aupinel, P., Behrens, D., Genersch, E., Vollmann, J., Riessberger-Gallé, U., 2013. Standard methods for artificial rearing of *Apis mellifera* larvae. J. Apicult. Res. 52 (1), 1–16. https://doi.org/10.3896/IBRA.1.52.1.05.

Daisley, B.A., Chmiel, J.A., Pitek, A.P., Thompson, G.J., Reid, G., 2020a. Missing microbes in bees: how systematic depletion of key symbionts Erodes immunity. Trends Microbiol. 28 (12), 1010–1021. https://doi.org/10.1016/j.tim.2020.06.006.

Daisley, B.A., Pitek, A.P., Chmiel, J.A., Al, K.F., Chernyshova, A.M., Faragalla, K.M., Burton, J.P., Thompson, G.J., Reid, G., 2020b. Novel probiotic approach to counter *Paenibacillus larvae* infection in honey bees. ISME J. 14 (2), 476–491. https://doi.org/10.1038/s41396-019-0541-6.

DEFRA, National Audit Office, 2009. The health of livestock and honeybees in England, hc 288 session 2008-2009, March. London: The Stationery Office.

di Prisco, G., Cavaliere, V., Annoscia, D., Varricchio, P., Caprio, E., Nazzi, F., Gargiulo, G., Pennacchio, F., 2013. Neonicotinoid clothianidin adversely affects insect immunity and promotes replication of a viral pathogen in honey bees. PNAS USA 110 (46), 18466–18471. https://doi.org/10.1073/pnas.1314923110.

Eilers, E.J., Kremen, C., Greenleaf, S.S., Garber, A.K., Klein, A.M., 2011. Contribution of pollinator-mediated crops to nutrients in the human food supply. PLoS One 6 (6), 21363. https://doi.org/10.1371/journal.pone.0021363.

Erban, T., Ledvinka, O., Kamler, M., Hortova, B., Nesvorna, M., Tyl, J., Titera, D., Markovic, M., Hubert, J., 2017. Bacterial community associated with worker honeybees (*Apis mellifera*) affected by European foulbrood. PeerJ 5, e3816. https://doi.org/10.7717/peerj.3816.

European Commission, 2017. Directorate-General for Environment, Agri-Environment Schemes: Impacts on the Agricultural Environment. Publications Office. https://doi.org/10.2779/633983.

Evans, J.D., Aronstein, K., Chen, Y.P., Hetru, C., Imler, J.-L., Jiang, H., Kanost, M., Thompson, G.J., Zou, Z., Hultmark, D., 2006. Immune pathways and defence mechanisms in honey bees *Apis mellifera*. Insect Mol. Biol. 15 (5), 645–656. https://doi.org/10.1111/j.1365-2583.2006.00682.x.

Evans, A.N., Llanos, J.E.M., Kunin, W.E., Evison, S.E.F., 2018. Indirect effects of agricultural pesticide use on parasite prevalence in wild pollinators. Agric. Ecosyst. Environ. 258, 40–48. https://doi.org/10.1016/j.agee.2018.02.002.

Evison, S.E., 2015. Chalkbrood: epidemiological perspectives from the host-parasite relationship. Insect Mol. Biol. 10, 65–70. https://doi.org/10.1016/j.cois.2015.04.015.

Evison, S.E., Jensen, A.B., 2018. The biology and prevalence of fungal diseases in managed and wild bees. Curr. Opin. Insect. Sci. 26, 105–113. https://doi.org/10.1016/j.cois.2018.02.010.

Evison, S.E.F., Roberts, K.E., Laurenson, L., Pietravalle, S., Hui, J., Biesmeijer, J.C., Smith, J.E., Budge, G., Hughes, W.O.H., 2012. Pervasiveness of parasites in pollinators. PLoS One 7 (1), e30641. https://doi.org/10.1371/journal.pone.0030641.

Figueroa, L.L., Grab, H., Ng, W.H., Myers, C.R., Graystock, P., McFrederick, Q.S., McArt, S.H., 2020. Landscape simplification shapes pathogen prevalence in plant-pollinator networks. Ecol. Lett. 23 (8), 1212–1222. https://doi.org/10.1111/ele.13521.

Floyd, A.S., Mott, B.M., Maes, P., Copeland, D.C., McFrederick, Q.S., Anderson, K.E., 2020. Microbial ecology of uropean foul brood disease in the honey bee (*Apis mellifera*): towards a microbiome understanding of disease susceptibility. Insects 11 (9), 1–16. https://doi.org/10.3390/insects11090555.

Forsgren, E., 2010. European foulbrood in honey bees. J. Invertebr. Pathol. 103, S5–S9. https://doi.org/10.1016/j.jip.2009.06.016.

Gill, R.J., Raine, N.E., 2014. Chronic impairment of bumblebee natural foraging behaviour induced by sublethal pesticide exposure. Funct. Ecol. 28 (6), 1459–1471. https://doi.org/10.1111/1365-2435.12292.

Graystock, P., Yates, K., Evison, S.E.F., Darvill, B., Goulson, D., Hughes, W.O.H., 2013. The Trojan hives: pollinator pathogens, imported and distributed in bumblebee colonies. J. Appl. Ecol. 50 (5), 1207–1215. https://doi.org/10.1111/1365-2664.12134.

Graystock, P., Blane, E.J., McFrederick, Q.S., Goulson, D., Hughes, W.O.H., 2016. Do managed bees drive parasite spread and emergence in wild bees? Int. J. Parasitol. Parasites Wildl. 5, 64–75. https://doi.org/10.1016/j.ijppaw.2015.10.001.

Graystock, P., Goulson, D., Hughes, W.O.H., 2015. Parasites in bloom: flowers aid dispersal and transmission of pollinator parasites within and between bee species. Proc. Royal Soc. B 282. https://doi.org/10.1098/rspb.2015.1371, 1813.

Graystock, P., Ng, W.H., Parks, K., Tripodi, A.D., Muñiz, P.A., Fersch, A.A., Myers, C.R., McFrederick, Q.S., McArt, S.H., 2020. Dominant bee species and floral abundance drive parasite temporal dynamics in plant-pollinator communities. Nat. Ecol. Evol. 4 (10), 1358–1367. https://doi.org/10.1038/s41559-020-1247-x.

Hartfelder, K., Makert, G.R., Judice, C.C., Pereira, G.A.G., Santana, W.C., Dallacqua, R., Bitondi, M.M.G., 2006. Physiological and genetic mechanisms underlying caste development, reproduction and division of labor in stingless bees. Apidologie 37 (2), 144–163. https://doi.org/10.1051/apido:2006013.

Higo, H.A., Rice, N.D., Winston, M.L., Lewis, B., 2004. Honey bee (Hymenoptera: Apidae) distribution and potential for supplementary pollination in commercial tomato greenhouses during winter. J. Econ. Entomol. 97 (2), 163–170. https://academic.oup.com/jee/article/97/2/163/2217947.

Hillyer, J.F., 2016. Insect immunology and hematopoiesis. Dev. Comp. Immunol. 58, 102–118. https://doi.org/10.1016/j.dci.2015.12.006.

Hristov, P., Shumkova, R., Palova, N., Neov, B., 2021. Honey bee colony losses: why are honey bees disappearing? Sociobiology 68 (1), e-5851. https://doi.org/10.13102/SOCIOBIOLOGY.V68I1.5851.

Hroncova, Z., Havlik, J., Killer, J., Doskocil, I., Tyl, J., Kamler, M., Titera, D., Hakl, J., Mrazek, J., Bunesova, V., Rada, V., 2015. Variation in honey bee gut microbial diversity affected by ontogenetic stage, age and geographic location. PLoS One 10 (3), e0118707. https://doi.org/10.1371/journal.pone.0118707.

Icons8, 2022. Creator: vitaly Gorbachev. https://icons8.com/icons/set/flower.

James, R.R., 2011. Chalkbrood transmission in the alfalfa leafcutting bee: the impact of disinfecting bee cocoons in loose cell management systems. Environ. Entomol. 40 (4), 782–787. https://doi.org/10.1603/EN10138.

James, R.R., Pitts-Singer, T.L., 2005. *Ascosphaera aggregata* contamination on alfalfa leafcutting bees in a loose cell incubation system. J. Invertebr. Pathol. 89 (2), 176–178. https://doi.org/10.1016/j.jip.2005.02.008.

Jensen, A.B., James, R.R., Eilenberg, J., 2009. Long-term storage of *Ascosphaera aggregata* and *Ascosphaera apis*, pathogens of the leafcutting bee (*Megachile rotundata*) and the honey bee (*Apis mellifera*). J. Invertebr. Pathol. 101 (2), 157–160. https://doi.org/10.1016/j.jip.2009.03.004.

Krichilsky, E., Centrella, M., Eitzer, B., Danforth, B., Poveda, K., Grab, H., 2021. Landscape composition and fungicide exposure influence host-pathogen dynamics in a solitary bee. Environ. Entomol. 50 (1), 107–116. https://doi.org/10.1093/ee/nvaa138.

Kwong, W.K., Moran, N.A., 2016. Gut microbial communities of social bees. Nat. Rev. Microbiol. 14 (6), 374–384. https://doi.org/10.1038/nrmicro.2016.43.

Lentola, A., David, A., Abdul-Sada, A., Tapparo, A., Goulson, D., Hill, E.M., 2017. Ornamental plants on sale to the public are a significant source of pesticide residues with implications for the health of pollinating insects. Environ. Pollut. 228, 297–304. https://doi.org/10.1016/j.envpol.2017.03.084.

Leska, A., Nowak, A., Nowak, I., Górczyńska, A., 2021. Effects of insecticides and microbiological contaminants on *Apis mellifera* health. Molecules 26 (16), 5080. https://doi.org/10.3390/molecules26165080.

Life, Discover, 2021. Bee genera. https://www.discoverlife.org/mp/20q?guide=Bee_genera.

Lima, M.A.P., Martins, G.F., Oliveira, E.E., Guedes, R.N.C., 2016. Agrochemical-induced stress in stingless bees: peculiarities, underlying basis, and challenges. J. Comp. Physiol. 202 (9–10), 733–747. https://doi.org/10.1007/s00359-016-1110-3.

López, J.H., Krainer, S., Engert, A., Schuehly, W., Riessberger-Gallé, U., Crailsheim, K., 2017. Sublethal pesticide doses negatively affect survival and the cellular responses in American foulbrood-infected honeybee larvae. Sci. Rep. 7, 40853. https://doi.org/10.1038/srep40853.

López-Uribe, M.M., Fitzgerald, A., Simone-Finstrom, M., 2017. Inducible versus constitutive social immunity: examining effects of colony infection on glucose oxidase and defensin-1 production in honeybees. R. Soc. Open Sci. 4 (5), 170224. https://doi.org/10.1098/rsos.170224.

Maxfield-Taylor, S.A., Mujic, A.B., Rao, S., 2015. First detection of the larval chalkbrood disease pathogen *Ascosphaera apis* (Ascomycota: eurotiomycetes: Ascosphaerales) in adult bumble bees. PLoS One 10 (4), e0124868. https://doi.org/10.1371/journal.pone.0124868.

McArt, S.H., Fersch, A.A., Milano, N.J., Truitt, L.L., Böröczky, K., 2017. High pesticide risk to honey bees despite low focal crop pollen collection during pollination of a mass blooming crop. Sci. Rep. 7, 46554. https://doi.org/10.1038/srep46554.

McGrady, C.M., Troyer, R., Fleischer, S.J., Strange, J., 2020. Wild bee visitation rates exceed pollination thresholds in commercial cucurbita agroecosystems. J. Econ. Entomol. 113 (2), 562–574. https://doi.org/10.1093/jee/toz295.

Medina, L., Hart, A., Ratnieks, F., 2009. Hygienic behavior in the stingless bees *Melipona beecheii* and *Scaptotrigona pectoralis* (Hymenoptera: Meliponini). Genet. Mol. Res. 8 (2), 571–576. https://doi.org/10.4238/vol8-2kerr010.

Menezes, C., Vollet-Neto, A., Marsaioli, A.J., Zampieri, D., Fontoura, I.C., Luchessi, A.D., Imperatriz-Fonseca, V.L., 2015. A Brazilian social bee must cultivate fungus to survive. Curr. Biol. 25 (21), 2851–2855. https://doi.org/10.1016/j.cub.2015.09.028.

Mogren, C.L., Benítez, M.S., McCarter, K., Boyer, F., Lundgren, J.G., 2020. Diverging landscape impacts on macronutrient status despite overlapping diets in managed (*Apis mellifera*) and native (*Melissodes desponsa*) bees. Conserv. Physiol. 8 (1) https://doi.org/10.1093/conphys/coaa109 coaa109.

Mullin, C.A., Frazier, M., Frazier, J.L., Ashcraft, S., Simonds, R., van Engelsdorp, D., Pettis, J.S., 2010. High levels of miticides and agrochemicals in North American Apiaries: implications for honey bee health. PLoS One 5 (3), e9754. https://doi.org/10.1371/journal.pone.0009754.

Murray, E.A., Burand, J., Trikoz, N., Schnabel, J., Grab, H., Danforth, B.N., 2019. Viral transmission in honey bees and native bees, supported by a global black queen cell virus phylogeny. Environ. Microbiol. 21 (3), 972–983. https://doi.org/10.1111/1462-2920.14501.

Olynyk, M., Westwood, A.R., Koper, N., 2021. Effects of natural habitat loss and edge effects on wild bees and pollination services in remnant prairies. Environ. Entomol. 50 (3), 732–743. https://doi.org/10.1093/ee/nvaa186.

O'Neal, S.T., Reeves, A.M., Fell, R.D., Brewster, C.C., Anderson, T.D., 2019. Chlorothalonil exposure alters virus susceptibility and markers of immunity, nutrition, and development in honey bees. J. Insect Sci. 19 (3), 1–8. https://doi.org/10.1093/jisesa/iez051.

Pamminger, T., Botías, C., Goulson, D., Hughes, W.O.H., 2018. A mechanistic framework to explain the immunosuppressive effects of neurotoxic pesticides on bees. Funct. Ecol. 32 (8), 1921–1930. https://doi.org/10.1111/1365-2435.13119.

Peck, D.T., Smith, M.L., Seeley, T.D., 2016. *Varroa destructor* mites can nimbly climb from flowers onto foraging honey bees. PLoS One 11 (12), e0167798. https://doi.org/10.1371/journal.pone.0167798.

Pohorecka, K., Skubida, P., Miszczak, A., Semkiw, P., Sikorski, P., Zagibajło, K., Teper, D., Kołtowski, Z., Skubida, M., Zdańska, D., Bober, A., 2012. Residues of neonicotinoid insecticides in bee collected plant materials from oilseed rape crops and their effect on bee colonies. J. Apicul. Science 56 (2), 115–134. https://doi.org/10.2478/v10289-012-0029-3.

Rajak, P., Dutta, M., Roy, S., 2015. Altered differential hemocyte count in 3rd instar larvae of *Drosophila melanogaster* as a response to chronic exposure of Acephate. Interdiscipl. Toxicol. 8 (2), 84–88. https://doi.org/10.1515/intox-2015-0013.

Reynaldi, F.J., Lucia, M., Genchi Garcia, M.L., 2015. *Ascosphaera apis*, the entomopathogenic fungus affecting larvae of native bees (*Xylocopa augusti*): first report in South America. Rev. Iberoam. De. Micol. 32 (4), 261–264. https://doi.org/10.1016/j.riam.2015.01.001.

Royauté, R., Wilson, E.S., Helm, B.R., Mallinger, R.E., Prasifka, J., Greenlee, K.J., Bowsher, J.H., 2018. Phenotypic integration in an extended phenotype: among-individual variation in nest-building traits of the alfalfa leafcutting bee (*Megachile rotundata*). J. Evol. Biol. 31 (7), 944–956. https://doi.org/10.1111/jeb.13259.

Rundlöf, M., Andersson, G.K.S., Bommarco, R., Fries, I., Hederström, V., Herbertsson, L., Jonsson, O., Klatt, B.K., Pedersen, T.R., Yourstone, J., Smith, H.G., 2015. Seed coating with a neonicotinoid insecticide negatively affects wild bees. Nature 521 (7550), 77–80. https://doi.org/10.1038/nature14420.

Rust, R., Torchio, P., 1992. Effects of temperature and host developmental stage on *Ascosphaera torchioi* Youssef and McManus prevalence in *Osmia lignaria propinqua* Cresson (Hymenoptera: Megachilidae). Apidologie 23, 1–9. https://doi.org/10.1051/apido:19920101.

Sánchez, A.L., Judith Slaa, E., Sandí, M., Salazar, W., 2001. Use of stingless bees for commercial pollination in enclousers: a promise for the future. Environ. Int. 89–90 (561), 7–11.

Schmehl, D.R., Tomé, H.V.V., Mortensen, A.N., Martins, G.F., Ellis, J.D., 2016. Protocol for the in vitro rearing of honey bee (*Apis mellifera* L.) workers. Journal of J. Apic. Res. 55 (2), 113–129. https://doi.org/10.1080/00218839.2016.1203530.

Schorkopf, D.L.P., Jarau, S., Francke, W., Twele, R., Zucchi, R., Hrncir, M., Schmidt, V. M., Ayasse, M., Barth, F.G., 2007. Spitting out information: *trigona* bees deposit saliva to signal resource locations. Proc. Royal Soc. B. 274 (1611), 895–898. https://doi.org/10.1098/rspb.2006.3766.

Sgolastra, F., Hinarejos, S., Pitts-Singer, T.L., Boyle, N.K., Joseph, T., Luckmann, J., Raine, N.E., Singh, R., Williams, N.M., Bosch, J., 2019. Pesticide exposure assessment paradigm for solitary bees. Environ. Entomol. 48 (1), 22–35. https://doi.org/10.1093/ee/nvy105.

Singh, R., Levitt, A. L., Rajotte, E. G., Holmes, E. C., Ostiguy, N., van Engelsdorp, D., Lipkin, W. I., de Pamphilis, C. W., Toth, A. L., Cox-Foster, D. L., 2010. RNA Viruses in Hymenopteran Pollinators: Evidence ofInter-Taxa Virus Transmission via Pollen and PotentialImpact on Non-ApisHymenopteran Species. PLoS ONE 5 (12), e14357. https://doi.org/10.1371/journal.pone.0014357.

Smith, D.B., Arce, A.N., Ramos Rodrigues, A., Bischoff, P.H., Burris, D., Ahmed, F., Gill, R.J., 2020. Insecticide exposure during brood or early-adult development reduces brain growth and impairs adult learning in bumblebees. Proc. R. Soc. B. 287, 20192442. https://doi.org/10.1098/rspb.2019.2442.

Stanley, D.A., Russell, A.L., Morrison, S.J., Rogers, C., Raine, N.E., 2016. Investigating the impacts of field-realistic exposure to a neonicotinoid pesticide on bumblebee foraging, homing ability and colony growth. J. Appl. Ecol. 53 (5), 1440–1449. https://doi.org/10.1111/1365-2664.12689.

Tort, N., Öztürk, İ., Güvensen, A., 2005. Effects of some fungicides on pollen morphology and anatomy of tomato (*Lycopersicon esculentum* mill.). Pakistan J. Bot. 37 (1), 23–30.

Traynor, K.S., Tosi, S., Rennich, K., Steinhauer, N., Forsgren, E., Rose, R., Kunkel, G., Madella, S., Lopez, D., Eversole, H., Fahey, R., Pettis, J., Evans, J.D., vanEngelsdorp, Dennis, 2021. Pesticides in honey bee colonies: establishing a baseline for real world exposure over seven years in the USA. Environ. Pollut. 279, 116566. https://doi.org/10.1016/j.envpol.2021.116566.

Vásquez, A., Forsgren, E., Fries, I., Paxton, R.J., Flaberg, E., Szekely, L., Olofsson, T.C., 2012. Symbionts as major modulators of insect health: lactic acid bacteria and honeybees. PLoS One 7 (3). https://doi.org/10.1371/journal.pone.0033188.

Walderdorff, L., Laval-Gilly, P., Wechtler, L., Bonnefoy, A., Falla-Angel, J., 2019. Phagocytic activity of human macrophages and *Drosophila* hemocytes after exposure to the neonicotinoid imidacloprid. Pestic. Biochem. Physiol. 160, 95–101. https://doi.org/10.1016/j.pestbp.2019.07.007.

Wilson-Rich, N., Dres, S.T., Starks, P.T., 2008. The ontogeny of immunity: development of innate immune strength in the honey bee (*Apis mellifera*). J. Insect Physiol. 54 (10–11), 1392–1399. https://doi.org/10.1016/j.jinsphys.2008.07.016.

Wood, T.J., Goulson, D., 2017. The environmental risks of neonicotinoid pesticides: a review of the evidence post 2013. Environ. Sci. 24 (21), 17285–17325. https://doi.org/10.1007/s11356-017-9240-x.

Wood, S.C., Chalifour, J.C., Kozii, I.V., de Mattos, I.M., Klein, C.D., Zabrodski, M.W., Moshynskyy, I., Guarna, M.M., Veiga, P.W., Epp, T., Simko, E., 2020a. *In Vitro* effects of pesticides on European foulbrood in honeybee larvae. Insects 11, 252. https://doi.org/10.3390/insects11040252.

Wood, S.C., de Mattos, I.M., Kozii, I.V., Klein, C.D., Dvylyuk, I., Folkes, C.D.A., de Carvalho Macedo Silva, R., Moshynskyy, I., Epp, T., Simko, E., 2020b. Effects of chronic dietary thiamethoxam and prothioconazole exposure on *Apis mellifera* worker adults and brood. Pest Manag. Sci. 76, 85–94. https://doi.org/10.1002/ps.5501.

Wood, T.J., Michez, D., Paxton, R.J., Drossart, M., Neumann, P., Gérard, M., Vanderplanck, M., Barraud, A., Martinet, B., Leclercq, N., Vereecken, N.J., 2020c. Managed honey bees as a radar for wild bee decline? Apidologie 51 (6), 1100–1116. https://doi.org/10.1007/s13592-020-00788-9.

Yousefi, B., Fouks, B., 2019. The presence of a larval honey bee parasite, *Ascosphaera apis*, on flowers reduces pollinator visitation to several plant species. Acta Oecol. 96, 49–55. https://doi.org/10.1016/j.actao.2019.03.006.

Yu, L., Yang, H., Cheng, F., Wu, Z., Huang, Q., He, X., Yan, W., Zhang, L., Wu, X., 2021. Honey bee *Apis mellifera* larvae gut microbial and immune, detoxication responses towards flumethrin stress. Environ. Pollut. 290, 118107. https://doi.org/10.1016/j.envpol.2021.118d107.

Zattara, E.E., Aizen, M.A., 2020. Worldwide Occurrence Records Reflect a Global Decline in Bee Species Richness. bioRxiv Preprint. https://doi.org/10.1101/869784.

Zemenick, A.T., Vanette, R.L., Rosenheim, J.A., 2021. Linked networks reveal dual roles of insect dispersal and species sorting for bacterial communities in flowers. Oikos 130 (5), 697–707. https://doi.org/10.1111/oik.06818.

Zurbuchen, A., Landert, L., Klaiber, J., Müller, A., Hein, S., Dorn, S., 2010. Maximum foraging ranges in solitary bees: only few individuals have the capability to cover long foraging distances. Biol. Conserv. 143 (3), 669–676. https://doi.org/10.1016/j.biocon.2009.12.003.