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Booton, R.D., Iwasa, Y., Marshall, J.A.R. et al. (1 more author) (2017) Stress-Mediated Allee Effects Can Cause the Sudden Collapse of Honey Bee Colonies. *Journal of Theoretical Biology*. ISSN 0022-5193

<https://doi.org/10.1016/j.jtbi.2017.03.009>

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Stress-Mediated Allee Effects Can Cause the Sudden Collapse of Honey Bee Colonies

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Abstract

The recent rapid decline in global honey bee populations could have significant implications for ecological systems, economics and food security. No single cause of honey bee collapse has yet to be identified, although pesticides, mites and other pathogens have all been shown to have a sublethal effect. We present a model of a functioning bee hive and introduce external stress to investigate the impact on the regulatory processes of recruitment to the forager class, social inhibition and the laying rate of the queen. The model predicts that constant density-dependent stress acting through an Allee effect on the hive can result in sudden catastrophic switches in dynamical behaviour and the eventual collapse of the hive. The model proposes that around a critical point the hive undergoes a saddle-node bifurcation, and that a small increase in model parameters can have irreversible consequences for the entire hive.

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We predict that increased stress levels can be counteracted by a higher laying rate of the queen, lower levels of forager recruitment or lower levels of natural mortality of foragers, and that increasing social inhibition can not maintain the colony under high levels of stress. We lay the theoretical foundation for sudden honey bee collapse in order to facilitate further experimental and theoretical consideration.

Keywords: Honey bees, Colony collapse disorder, Allee effects, Saddle-node bifurcation, Population dynamics

Highlights

- We present a model of a functioning honey bee colony by considering the in-hive and forager adult bees.
- We introduce Allee-based stress effects alongside the fundamental regulatory processes governing the bee hive.
- We predict that the presence of a critical transition via a saddle-node bifurcation causes sudden collapse.
- Small increases in stress load can cause fundamental breakdowns in the normal regulatory functions of a colony.

1. Introduction

The pollination industry generates a total economic value of €153 billion per year (Gallai et al., 2009) and 75% of the leading global fruit, vegetable and seed crops rely on animal pollination, accounting for 35% of total global food production (Klein et al., 2007). The Western honey bee *Apis mellifera* *L.* is the most common pollinator, providing an additional service to native pollinators through managed colonies (Goodwin et al., 2011; Rucker et al., 2012). Hence, there are major concerns for the effects that decreasing honey bee colonies will have on future biodiversity and agriculture (Allen-Wardell et al., 1998; Biesmeijer et al., 2006; Potts et al., 2010; Burkle et al., 2013). Furthermore, it is normal for beekeepers to lose 15% of the total honey bee population per year (VanEngelsdorp et al., 2007), but more recently this decline has accelerated alarmingly to 30% per year (VanEngelsdorp et al., 2011). This has led to the definition of the term Colony Collapse Disorder (CCD) to describe the sudden mass disappearance of the worker honey bee population leading to colony failure (VanEngelsdorp et al., 2007).

Many potential stressors thought to cause CCD have been identified, although there has been no definitive explanation for every known symptom of collapsing hives. Pesticides (Suchail et al., 2001; Henry et al., 2012; Dai et al., 2010; Decourtye and Devillers, 2005), viruses (Highfield et al., 2009; Bromenshenk et al., 2010; Runckel et al., 2011; Moore et al., 2011), fungal diseases (Aronstein and Murray, 2010; Runckel et al., 2011; Fries, 2010; Paxton, 2010; Higes and Meana, 2013), microbes (Evans and Schwarz, 2011),

24 mite infections (Dainat et al., 2012; Eischen, 1987; Sammataro et al., 2000),
25 poor nutrition (Pernal and Currie, 2001; Alaux, Ducloz, Crauser and Le
26 Conte, 2010) and starvation (Mattila and Otis, 2007) have all been shown
27 to have adverse effects on honeybees. Recently the possibility of causes in-
28 volving several co-factors have been investigated. It has been suggested that
29 CCD could have its origins in multiple abiotic and biotic stressors interacting
30 with each other (Potts et al., 2010; Ratnieks and Carreck, 2010; Vanbergen,
31 2013). For example, the parasitic mite *Varroa destructor* and the viruses it
32 transmits (Nazzi et al., 2012; Francis et al., 2013), the interactions between
33 multiple pesticides having a synergistic effect on development and mortality
34 rate (Pilling and Jepson, 1993; Johnson et al., 2009, 2013; Wu et al., 2011),
35 and pesticides increasing the effect of pathogens in larvae and adult bees,
36 increasing the colony death rate (Pettis et al., 2012).

37 Honey bee social behaviour and the mechanisms that govern this are
38 widely understood. Eusocial insects are typically defined by their intricate
39 advanced division of labour (Robinson, 1992), and within honey bee colonies
40 specific individuals have different roles in the hive (Visscher, 1983). Life for
41 the honey bee begins with the queen laying eggs, from which a proportion
42 will eclose within three weeks dependent upon the size of the adult work-
43 force (Winston, 1991). The rate that a colony can grow is impacted by two
44 central factors, the total number of adult workers and the laying rate of the
45 queen (Fefferman and Starks, 2006). One of the most fundamental honey-
46 bee colony dynamics is the ability to structure the workforce according to

47 age of the individuals, although this division of labour can change (John-
48 son, 2003) in response to stressors and in order to ensure colony survival.
49 This regulation system, known as temporal polyethism allows honey bees to
50 respond to stressors by either reverting to previous roles or taking on new
51 ones. This flexibility in age structured task allocation is socially regulated
52 (Huang and Robinson, 1996). Young honey bees tend to work on in-hive
53 tasks such as cleaning, tending brood and eating pollen (Seeley, 1995) while
54 remaining protected from potential outside stressors. Older adults will be-
55 gin foraging at around 2-3 weeks (Winston, 1991), where natural mortality
56 will most likely occur due to forager exhaustion (Neukirch, 1982) and the
57 risks affiliated with foraging (Visscher and Dukas, 1997). Therefore, natural
58 mortality in individual honey bees is age-dependent.

59 While an abundance of empirical work has been conducted addressing
60 the individual effects of stressors, relatively few theoretical approaches have
61 considered the underlying dynamics of collapse and the mechanisms regulat-
62 ing honey bee population dynamics. A simple model of in-hive and forager
63 worker bees and the transitions between these showed that beneath a critical
64 death rate of foragers, the colony can survive (Khoury et al., 2011). Develop-
65 ments upon this framework to include more complicated aspects of the hive
66 were analysed with similar results (Khoury et al., 2013). Seasonal and an-
67 nual fluctuations within another model predicted that death rates, food and
68 transitions from in-hive to foraging tasks can influence colony survival (Rus-
69 sell et al., 2013). Population based Allee effects were shown to induce failure

70 of the hive (Dennis and Kemp, 2016) and investigations into the effects of
71 sublethal stress on colony function demonstrated that positive density depen-
72 dence can cause either exponential growth or failure of the colony (Bryden
73 et al., 2013). Other models incorporating the effects of stressors have been
74 shown to cause colony failure such as infection (Kribs-Zaleta and Mitchell,
75 2014), American Foulbrood disease (Jatulan et al., 2015) and the interac-
76 tion between *Varroa destructor* and Acute Bee Paralysis Virus (Ratti et al.,
77 2015).

78 While previous theoretical studies capture some elements of CCD and fail-
79 ure of the colony, particularly the existence of thresholds where the colony
80 will either grow or fail, real collapse dynamics appear to be sudden (Lu et al.,
81 2012) rather than the gradual decline observed in most modelling studies.
82 Bistability, or the presence of two alternate stable states, where one state
83 corresponds to a stable positive population equilibrium, and the other to
84 the extinction of the hive, could be crucial to understanding the suddenness
85 observed in CCD. That is, CCD could be caused by sudden switches in stabil-
86 ity around a critical point. We present a model that exhibits these positive-
87 extinction stable states. We consider a generalised density-dependent stressor
88 causing adult worker bees to disappear from the hive, and density-dependent
89 mortality acting on high-density populations. We investigate the codepen-
90 dence of stress with the major regulatory functions in bee hives, such as the
91 laying rate of the queen, recruitment to the forager class, natural mortality
92 and social inhibition, and how these regulatory functions can counteract high

93 stress levels in honey bee colony units.

94 **2. Methods**

95 The structure of the honey bee hive is complex (Seeley, 1995), and many
96 mathematical models have tried to express and explain the major regulatory
97 systems observed in real hives. The model we present in Figure 1 extends
98 previous model frameworks in Khoury et al. (2011) from which we formulate
99 the basic processes governing the hive. We make the simplification to consider
100 only the in-hive worker (H) and outside-of-the-hive worker or forager (F)
101 populations, and assume all bees can be classified in this way. Because in-
102 hive mortality is extremely low compared to that among foragers (Visscher
103 and Dukas, 1997), we assume that all natural mortality occurs in the forager
104 class, at a rate m . Honey bees enter the hive through the eclosion function
105 E , and are recruited into the forager class through the recruitment function
106 R . We assume that a proportion of the colony is lost to a generalised stress
107 function, which induces a lethal effect (Staveley et al., 2014), through an
108 individual's total disappearance from the colony caused by the effects of
109 pesticides causing navigational problems for foragers never returning back to
110 the hive (Bortolotti et al., 2003), or that of density dependent in-hive worker
111 bee mass disappearance, present in CCD situations (VanEngelsdorp et al.,
112 2009). We assume that stress S as a function of time t acts across both
113 in-hive and forager compartments, as an Allee effect. As each individual
114 stressor impacts different classes of honey bee in a different way, we make

115 this assumption to simplify all stresses into a single function. We did this
 116 under the knowledge that the location of stress within the model does not
 117 impact the qualitative dynamics of the model (Supplementary Figure S6).
 118 We also model density-dependent limiting effects at large colony sizes via the
 119 function C . We can express the model with this additional general stressor
 120 term and additional large colony limiting effect as a two dimensional system
 of differential equations:

121

The rate of change of the in-hive population as functions of eclosion E ,
 recruitment R , stress S and limiting function C

$$\begin{array}{l}
 122 \\
 123
 \end{array}
 \frac{dH}{dt} = E(H, F) - R(H, F)H - S(H, F)H - C(H, F)H \quad (1)$$

The rate of change of the forager population as functions of recruitment R ,
 natural mortality m and stress S

$$\begin{array}{l}
 124 \\
 125
 \end{array}
 \frac{dF}{dt} = R(H, F)H - mF - S(H, F)F \quad (2)$$

126 Following Khoury et al. (2011), we assume that the maximum eclosion of
 127 brood is equivalent to the laying rate L of the queen, and converges to L as
 128 $H + F$ gets large. Maximum eclosion occurs when the total size of the colony
 129 is large, representing the case when the total adult honey bee population is
 130 able to raise all eggs to adulthood (Winston, 1991). The parameter ω sets

131 the speed at which total eclosion tends towards the maximum eclosion L .
 132 We make this assumption because the total number of eclosing eggs in honey
 133 bee hives is proportional to the number of adult bees in the colony (Allen
 and Jeffree, 1956; Harbo, 1986).

$$134 \quad E(H, F) = L \frac{H + F}{\omega + H + F} \quad (3)$$

135 The recruitment function $R(H, F)$ captures the effects of both natural age-
 136 dependent transitions to foraging and that of social inhibition. In-hive bees
 137 are recruited to the foraging class at rate α , and can switch back to in-hive
 138 tasks via social inhibition at a rate σ , proportional to the relative foraging
 139 capacity of the colony. We introduce a term k , which represents the rate at
 140 which the proportion of reverting foragers approaches the maximum social
 141 inhibition rate σ . Similarly to Khoury et al. (2011), the recruitment function
 can be modelled as

$$142 \quad R(H, F) = \alpha - \sigma \frac{F}{k + F + H} \quad (4)$$

143 Stress is modelled as a positive density-dependent mortality Allee effect,
 similarly to Bryden et al. (2013),

$$144 \quad S(H, F) = \frac{\mu}{\phi + H + F} \quad (5)$$

145 where per capita mortality is inversely proportional to the operational colony
 146 size. The rate of stress can be expressed as μ , and the low colony mortality

147 can be controlled via ϕ . The limiting function at high densities is proportional
 148 to the total colony size

$$148 \quad C(H, F) = \gamma(H + F) \quad (6)$$

149 We choose this high density effect γ to be extremely small. This large colony
 150 size limiting function represents the biological nature of hives, as populations
 151 do not grow indefinitely, with a typical colony size around 20 000 worker
 152 bees (Seeley, 1995), and often managed hives have limited comb space which
 153 are maintained by beekeepers. In addition, populations of honey bees often
 154 swarm, preventing the total population from growing indefinitely. The total
 155 combined mortality effect for the in-hive population ($S(H, F) + C(H, F)$)
 156 and the individual effects of both can be seen in Figure 2, where the overall
 157 mortality is very high for lower number of bees, and decreases before increas-
 158 ing again for large colony sizes. The final system of differential equations is
 159 therefore

$$160 \quad \frac{dH}{dt} = L \frac{H + F}{\omega + H + F} - H \left(\alpha - \sigma \frac{F}{k + F + H} \right) - \frac{\mu H}{\phi + H + F} - \gamma(H + F)H \quad (7a)$$

$$161 \quad \frac{dF}{dt} = H \left(\alpha - \sigma \frac{F}{k + F + H} \right) - mF - \frac{\mu F}{\phi + H + F} \quad (7b)$$

163 These equations were analysed using the standard methods from dynamical
 164 systems theory. The equations were solved numerically with Wolfram Math-

165 ematica version number *10.0.2.0*. Numerical bifurcation plots were produced
 166 using the package MatCont in MATLAB version number *8.6 R2015b*. We pa-
 167 rameterise the model according to previous empirical and theoretical studies
 168 as shown in Table 1.

169 **3. Results**

170 There are two fixed points in system (7)

$$171 \quad (H, F) = (0, 0) \quad (8a)$$

$$172 \quad (H, F) = (H^*, F^*) \quad (8b)$$

173
 174 with $H^*, F^* > 0$. Let us define the following functions

$$175 \quad g_1(H, F) = \frac{dH}{dt} \quad (9a)$$

$$176 \quad g_2(H, F) = \frac{dF}{dt} \quad (9b)$$

178 We calculate the Jacobian matrix for system (7) evaluated at the fixed point

$$179 \quad (H, F) = (0, 0)$$

$$180 \quad J = \begin{pmatrix} \left(\frac{dg_1}{dH}\right)_{(0,0)} & \left(\frac{dg_1}{dF}\right)_{(0,0)} \\ \left(\frac{dg_2}{dH}\right)_{(0,0)} & \left(\frac{dg_2}{dF}\right)_{(0,0)} \end{pmatrix} = \begin{pmatrix} -\alpha - \frac{\mu}{\phi} + \frac{L}{\omega} & \frac{L}{\omega} \\ \alpha & -m - \frac{\mu}{\phi} \end{pmatrix} \quad (10)$$

182 Calculating eigenvalues gives the condition for stability of the extinction of
 183 the population of honey bees. This happens when (11a) and (11b) hold true

184

$$185 \quad 0 < \omega < \frac{L(m + \alpha)}{m\alpha} \quad \& \quad (11a)$$

$$186 \quad \mu > \frac{L\phi + \omega \left(-\phi(m + \alpha) + \sqrt{\frac{\phi^2(L^2 + 2L\omega(m + \alpha) + (m - \alpha^2)\omega^2)}{\omega^2}} \right)}{2\omega} = \mu_{crit} \quad (11b)$$

187 or, when (12) holds true

$$188 \quad \omega \geq \frac{L(m + \alpha)}{m\alpha} \quad (12)$$

189

190 i.e. the population goes extinct when either the laying rate is too low (12)
 191 or when the laying rate is sufficiently high (11a) and the stress μ is higher
 192 than a critical level μ_{crit} (11b).

193 Two qualitatively distinct dynamical outcomes are possible within our
 194 model. Either the colony size over time reaches a positive stable equilibrium
 195 which represents the optimal size of the colony or the population decreases
 196 rapidly around a critically low density colony size and the hive collapses.
 197 These two possibilities are dependent on initial conditions and parameter
 198 choice. This dynamical behaviour is summarised in Fig. 3a, which shows
 199 the effect of increasing the stress parameter μ on the total numbers of the
 200 adult in-hive and forager bees. In the stress free population and for stress
 201 levels less than the critical level, the model predicts that the population
 202 will reach equilibrium if the initial density is high enough. As the stress
 203 parameter μ is increased, total density drops, and then we observe a tipping

204 point at the critical level of stress. If initial population sizes are below the
205 unstable population size (Fig. 3b), then we predict the extinction of the hive.
206 Otherwise, all populations will grow and tend towards the stable branch, and
207 remain stable (Fig. 3b).

208 Fig. 4 shows the saddle-node bifurcation present in our system, high-
209 lighting the location of the stable and unstable branches with respect to the
210 stress parameter for the in-hive population. This shows the way that the
211 total in-hive population changes as a function of stress, and where the limit
212 point is formed as the stable and unstable equilibria branches collide and
213 disappear, leaving only the stable zero solution. This dynamical behaviour
214 and the presence of the stable-unstable-stable equilibria is related to initial
215 conditions (Fig. 6), for both low and high stress levels. For lower stress lev-
216 els all solutions tend towards either stable equilibria dependent upon initial
217 conditions, and for high stress levels all solutions tend towards the stable ex-
218 tinction of the hive. Other saddle-node bifurcations can be caused by changes
219 to the parameters representing the natural mortality of foragers (Fig. S1),
220 and recruitment to the forager class (Fig. S2). The direction of the saddle-
221 node bifurcation is reversed for the laying rate of the queen (Fig. S4), and is
222 also reversed for the bifurcation of the social inhibition parameter (Fig. S3).

223 Fig. 5 shows the point of colony failure as a function of stress and other
224 critical parameters, highlighting the relationship between the major regula-
225 tory functions of the honey bee hive and the hive's response to stress. Higher
226 levels of laying by the queen, lower levels of forager recruitment and lower

227 natural forager mortality can all counteract high levels of stress impacting
228 the colony. Interestingly, our model predicts that varying the level of social
229 inhibition can not save the colony from extinction at high stress levels.

230 **4. Discussion**

231 In this paper we show how stress-mediated Allee effects bring about sud-
232 den collapse in the population dynamics of honey bee colonies. The stress
233 induced bistability created by our model forced dependence on the initial
234 population sizes of both in-hive and forager bees. This led to a sensitive
235 threshold around the unstable population size where colonies would either
236 persist or fail. In addition, we show that CCD can be triggered by small per-
237 turbations in regulatory hive functions through changes in hive parameters
238 indicating that the honey bee hive is highly sensitive to such changes under
239 density-dependent stress.

240 The regulatory functions governing honey bee hives are well understood.
241 It is well documented that the hive will respond to higher levels of mortality
242 of foragers by speeding up recruitment to form a workforce primarily made of
243 precocious foragers (Huang and Robinson, 1996), a process that is thought to
244 be one of the symptoms of CCD (Khoury et al., 2011). It is also understood
245 that the queen is influenced by many factors including seasonality, total avail-
246 able resources, queen age, temperature in the hive, and photoperiod (Shehata
247 et al., 1981; Kefuss, 1978). Therefore regulatory functions could have signif-
248 icant implications in maintaining the colony under stress, and could also be

249 influenced by these stresses. Through investigating the relationship between
250 stress and the major regulatory functions of honey bee hives, we make pre-
251 dictions reflecting the nature of colony collapse. Near the critical threshold
252 inherent in our model, both an increase in recruitment to the forager class or
253 a decrease in social inhibition can cause sudden colony failure. This suggests
254 that CCD can be promoted by a breakdown in these simple regulatory func-
255 tions that usually maintain honey bee hives under stress. We also predict that
256 fluctuations in the queen's laying rate are highly sensitive in failing colonies.
257 A small decrease in the laying rate of the queen subject to these natural
258 fluctuations close to the bifurcation point could result in drastic switches in
259 the dynamics of the hive, although colonies will normally replace the queen
260 if she is not adequately laying enough brood (Winston, 1991), which could
261 potentially occur before this critical point. In addition, the model proposes
262 that a small increase in natural mortality can cause the sudden collapse of
263 the colony, although the occurrence of mortality fluctuations are unlikely
264 in summer conditions given the observed constant probability of death per
265 unit time spent away from the hive (Visscher and Dukas, 1997), thus making
266 natural mortality less likely to be subject to bifurcation-causing fluctuations.

267 The intrinsic bistability and sensible ecological behaviour present within
268 our model implies an alternative route to colony collapse through the pres-
269 ence of a saddle-node bifurcation, not seen in other theoretical studies of
270 honey bee population dynamics (Khoury et al., 2011; Bryden et al., 2013).
271 Although some empirical replications of CCD have observed sudden declines

272 in honey bee populations exposed to stressors such as the neonicotinoid imi-
273 dacloprid (Lu et al., 2012), an insecticide thought to cause abnormal foraging
274 behaviour (Yang et al., 2008), further work is needed to understand the mech-
275 anisms governing honey bee failure. Our generalised approach to modelling
276 stress can, in theory, be thought of as acting through any possible mortality-
277 based hive-wide stressor, such as other pesticides (Henry et al., 2012), the
278 mite *Varroa destructor* (Dainat et al., 2012) or the pathogen *Nosema cer-*
279 *anae* (Bromenshenk et al., 2010). If it shown that honey bee hives exhibit
280 bistability, we may be able to forecast the period leading up to the critical
281 transition, and provide new ways of detecting imminent CCD.

282 There are many potential extensions to the modelling framework we
283 present. We do not consider the effects of seasonality, instead concentrating
284 on the colony in the favourable spring and summer conditions. Indeed, it has
285 been shown that honey bee survival depends upon the time of year (Mattila
286 and Otis, 2007) and that the proportion of brood reared to adulthood de-
287 pends upon the supply of pollen which decreases in the autumn and winter
288 seasons (Seeley, 1985). In order to better understand how the risk of colony
289 collapse varies across the seasons, we suggest extending the model to include
290 seasonality in similar ways in which other models have proven useful in this
291 context (Russell et al., 2013; Ratti et al., 2015). This combination of known
292 ecological behaviour and bistability within our model could provide insight
293 into the mechanisms governing colonies which commonly collapse in winter
294 conditions (VanEngelsdorp et al., 2009).

295 Currently, we concentrate on the two most significant distinct adult classes
296 (Seeley, 1995), the in-hive and forager worker bees. We make this simplifica-
297 tion as there is a clear distinction in mortality rates and behaviour between
298 these two populations, and together they express the most important reg-
299 ulatory processes in the hive (Seeley, 1995). However we could extend the
300 model to include the population dynamics of bees from either the nest cen-
301 tre (cleaning and feeding) or nest periphery (receiving, packing and storing
302 nectar) (Seeley, 1995). For example, we did not consider the regulatory pro-
303 cesses governing receiver honey bees for which the dynamics are well known.
304 Forager bees collect nectar and transfer it to receiver bees who then proceed
305 to store this material in cells (Ratnieks and Anderson, 1999). Under higher
306 influxes of nectar, the colony can allocate more honey bees into the receiver
307 bee class (Seeley et al., 1996), and thus can be thought of as another regula-
308 tory process maintaining the colony. This introduction of a new classification
309 of honey bee into our modelling framework would help describe the break-
310 down in regulatory processes of a honey bee hive under CCD conditions in
311 more detail.

312 In recent years, researchers have become interested in forecasting transi-
313 tions of state in the underlying dynamics of a wide range of systems (Venegas
314 et al., 2005; Litt et al., 2001; McSharry et al., 2003; May et al., 2008; Schef-
315 fer et al., 2001). If bistability is important in understanding the general
316 mechanisms governing a honey bee hive under stress, then we should be able
317 to predict the onset of colony collapse. The model described in this paper

318 has the required properties needed to detect critical transitions before they
319 drastically alter the population dynamics of the system. The existence of a
320 set of predictors called early warning signals (EWS) can be applied to any
321 system with sudden changes in state (Scheffer et al., 2001). When a system
322 undergoes significant change from one state into another state, just before
323 the transition it approaches the tipping point or critical threshold, as shown
324 in the dynamics of our model. Sometimes these changes in state can be
325 catastrophic and widespread, having a detrimental ecological impact on the
326 system as a whole (Scheffer et al., 2001; Folke et al., 2004), with the system
327 sometimes never returning to its original state, even after pre-collapse con-
328 ditions have been restored (Scheffer et al., 2001). The potential implications
329 and applications of these EWS combined with our model are numerous and
330 may provide the much needed insight into the complex problem of CCD.

331 **5. Acknowledgments**

332 We would like to thank the anonymous reviewers for their thoughtful
333 comments and suggestions which helped improve this study. This work was
334 supported by a Japan Society for the Promotion of Science (JSPS) Summer
335 Fellowship to R.D.B.

336 **6. References**

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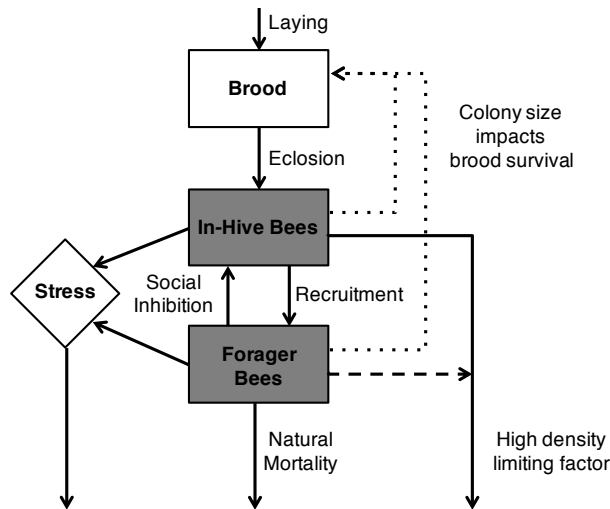


Figure 1: Dynamics of the model. The queen lays eggs which eclose into adult in-hive bees. Total adult population size impacts brood survival. A proportion of the in-hive bees are recruited into the foraging class by the natural age-dependent structure of the hive. Forager bees are able to make the switch back to the in-hive class via social inhibition. Natural mortality occurs within the forager class, but high density mortality occurs within the in-hive class. The generalised stress term acts over both adult classes and causes both in-hive and forager mortality or disappearance from the hive.

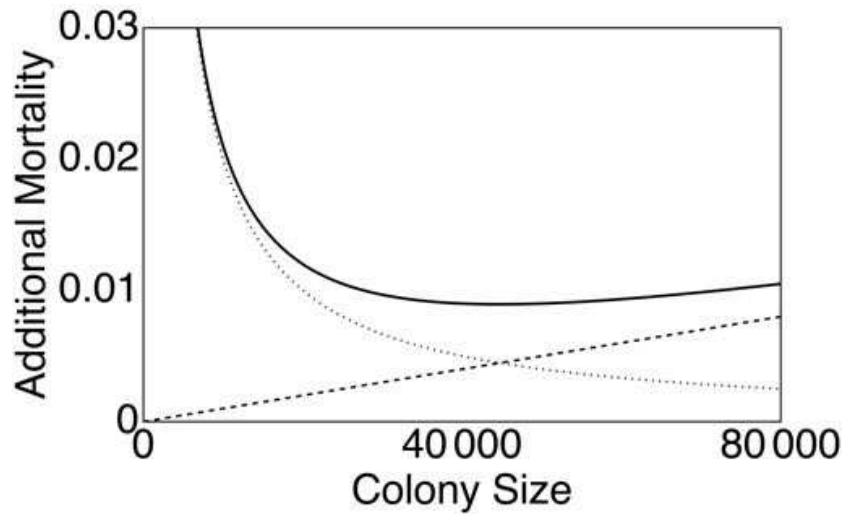


Figure 2: The effects of stress $S(H, F)$ (dotted) and high density function $C(H, F)$ (dashed) on in-hive mortality, and the combined effect (black), as the colony size ($H+F$) increases. Parameters are $\mu = 200$, $\phi = 0.402$, $\gamma = 0.0000001$. In our model, the stress function $S(H, F)$ acts strongly at very small populations, whereas the large population size limiting factor $C(H, F)$ is small at low populations. At high population sizes, the limiting effect reduces the population which results in the population declining rapidly whereas the stress term has a small effect. The combined impact is high additional mortality at low population sizes, then a decrease for intermediate population sizes before higher mortality again at high population sizes.

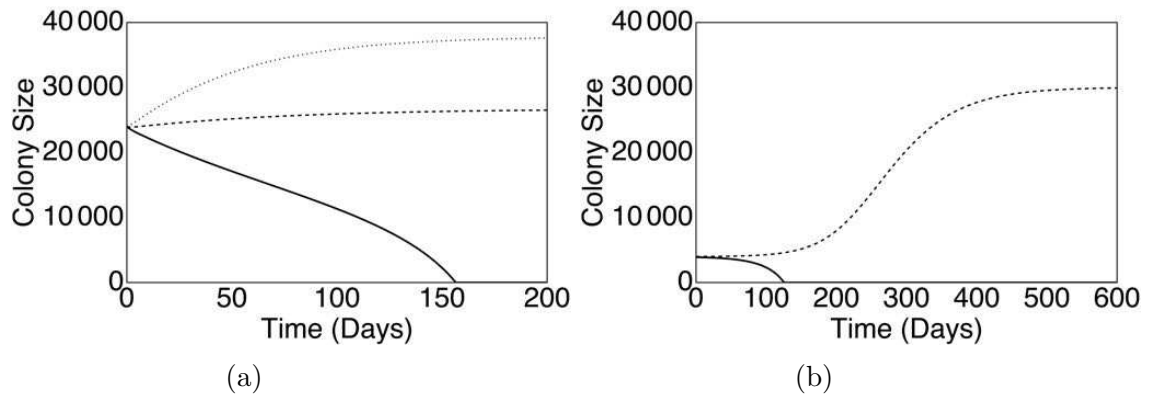


Figure 3: Numerical simulations of the model for (a) increasing stress levels and (b) sensitivity of initial conditions. In (a) we plot 3 stress levels $\mu = 0$ (dotted), $\mu = 200$ (dashed) and $\mu = 400$ (black). Failure of the colony is initiated by the high stress level ($\mu = 400$). Initial conditions are $H(0) = 16000$, $F(0) = 8000$. In (b), dependence upon initial conditions is illustrated with a fixed stress $\mu = 150$ for $H(0) = 3000$, $F(0) = 1000$ (dashed) and $H(0) = 2900$, $F(0) = 1000$ (black). A decrease in 100 initial in-hive bees causes the colony to fail. Parameters are taken from Table 1.

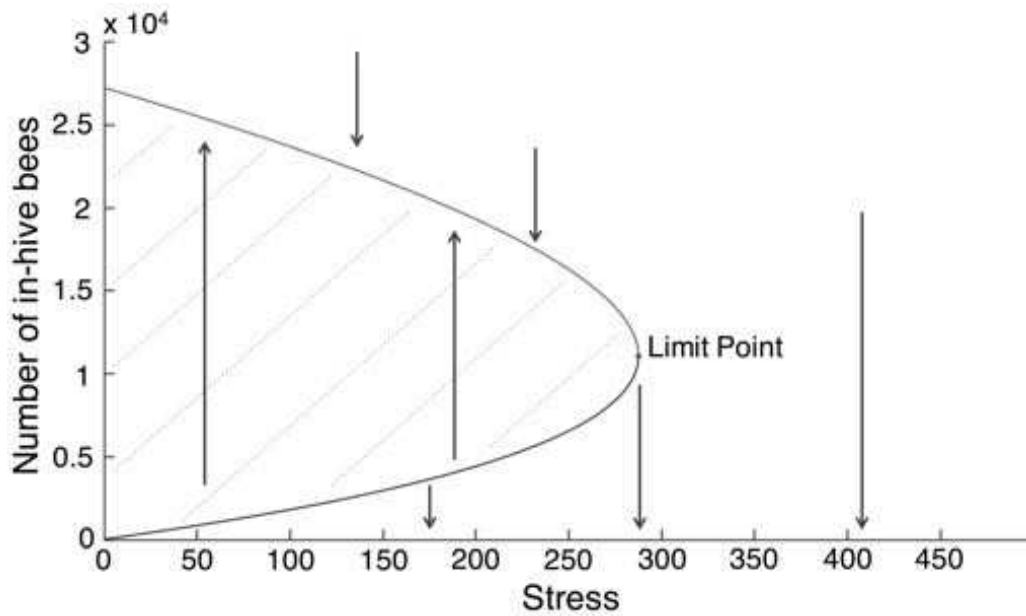
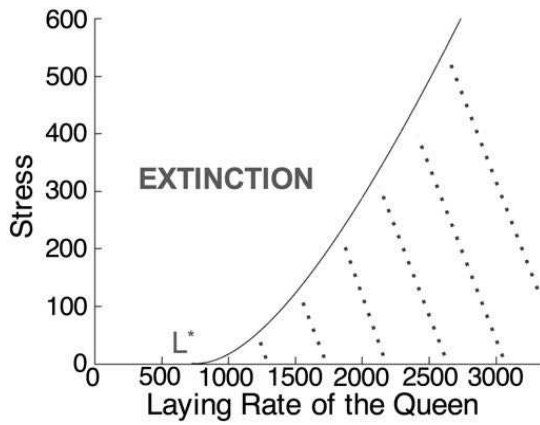
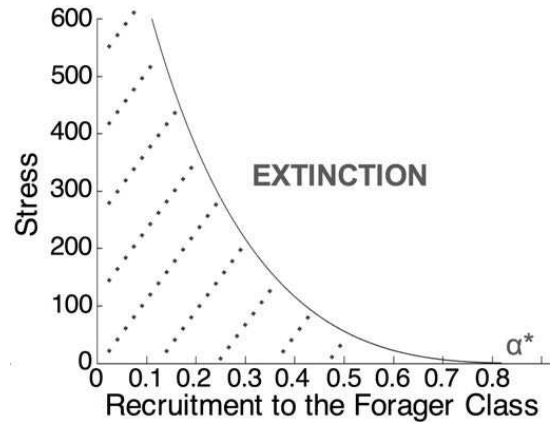


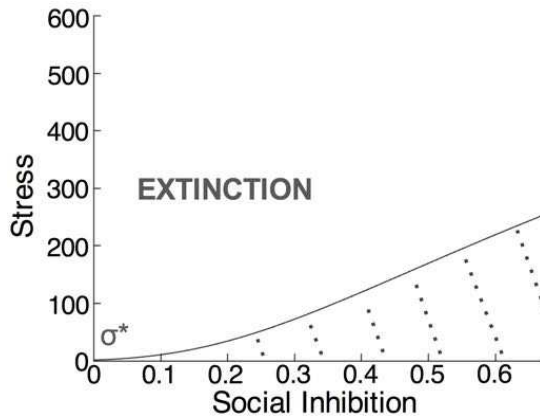
Figure 4: The saddle-node bifurcation through the stress parameter μ for the total numbers of in-hive honey bees. Parameters are taken from Table 1. The location of the limit point represents a critical stress level after which the total number of in-hive bees will become 0. The existence of the unstable branch pushes all solutions onto the stable branch, unless initial conditions lie below this unstable branch. Around the critical stress level, we see a rapid decline in the number of in-hive bees.



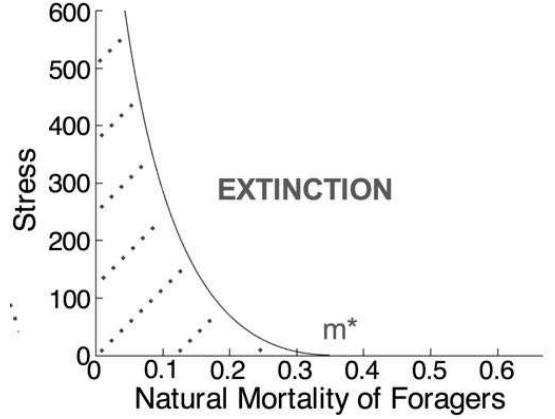
(a) Laying rate L and stress μ .



(b) Recruitment α and stress μ .



(c) Social inhibition σ and stress μ .



(d) Natural mortality m and stress μ .

Figure 5: The location of the limit point present in the saddle-node bifurcation within two dimensional parameter space (black line), and the conditions for extinction and persistence (dotted), with parameters taken from Table 1. In (a), the higher laying rate L counteracts stress and extremely high laying rates require exponential stress levels to cause failure. In (b), low levels of forager recruitment α can maintain the colony. This can be thought of as lower levels of 'panic' switching between tasks counteracting high stress levels. In (c), extinction of the hive is possible for all values of social inhibition σ . Low levels of social reversion are close to the limit point, even in the stress free hive. In (d), collapse of the hive is not possible for extremely low natural mortality m of foragers. Past the critical death rate all colonies will fail regardless of the stress level.

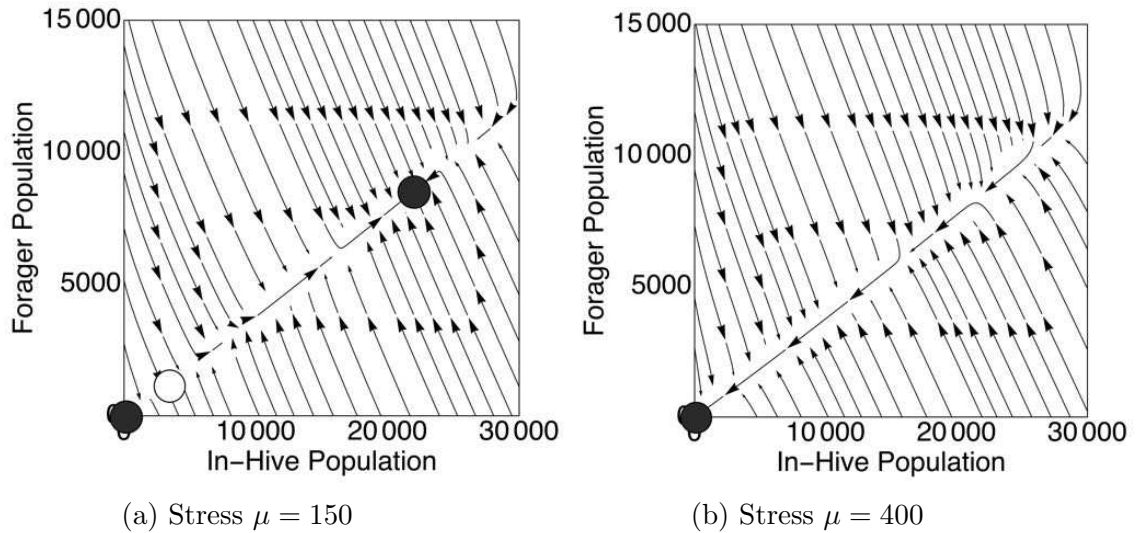


Figure 6: The comparison of two levels of stress on the in-hive - forager phase plane. Parameters are taken from Table 1. At the lower level of stress $\mu = 150$, the populations tend towards the positive stable equilibrium at $(H, F) = (21643, 8380)$ or to the stable origin $(H, F) = (0, 0)$ (black dots). The existence and location of the unstable equilibrium (white dot) suggests that for these parameters there can be a minimum of 2927 in-hive and 1064 foragers before extinction of the hive. In (b), all solutions tend towards $(0, 0)$ (black dot), regardless of the initial conditions suggesting that this level of stress $\mu = 400$ will cause extinction in all cases.

<i>Parameter</i>	<i>Description</i>	<i>Value</i>	<i>Reference</i>
L	Laying rate of the queen	2000	Cramp (2008)
α	Recruitment to forager class	0.25	Fahrbach and Robinson (1996)
σ	Social inhibition	0.75	Fahrbach and Robinson (1996)
ω	Rate at which eclosion tends to maximum	27000	Khoury et al. (2011)
ϕ	Control of low colony mortality	0.402	Bryden et al. (2013)
m	Natural mortality	0.1	<i>chosen to be small</i>
k	Rate at which social inhibition tends to maximum	0.1	<i>arbitrary</i>
γ	High density effect	0.0000001	<i>chosen to be very small</i>
μ	Stress	[0, 2000]	<i>varying</i>

Table 1: Model Parameters