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

Honey bee social behaviour and the mechanisms that govern this are widely understood. Eusocial insects are typically defined by their intricate advanced division of labour (Robinson, 1992), and within honey bee colonies specific individuals have different roles in the hive (Visscher, 1983). Life for the honey bee begins with the queen laying eggs, from which a proportion will eclose within three weeks dependent upon the size of the adult workforce (Winston, 1991). The rate that a colony can grow is impacted by two central factors, the total number of adult workers and the laying rate of the queen (Fefferman and Starks, 2006). One of the most fundamental honeybee colony dynamics is the ability to structure the workforce according to age of the individuals, although this division of labour can change (Johnson, 2003) in response to stressors and in order to ensure colony survival. This regulation system, known as temporal polyethism allows honey bees to respond to stressors by either reverting to previous roles or taking on new ones. This flexibility in age structured task allocation is socially regulated (Huang and Robinson, 1996). Young honey bees tend to work on in-hive tasks such as cleaning, tending brood and eating pollen (Seeley, 1995) while remaining protected from potential outside stressors. Older adults will begin foraging at around 2-3 weeks (Winston, 1991), where natural mortality will most likely occur due to forager exhaustion (Neukirch, 1982) and the risks affiliated with foraging (Visscher and Dukas, 1997). Therefore, natural mortality in individual honey bees is age-dependent.

While an abundance of empirical work has been conducted addressing the individual effects of stressors, relatively few theoretical approaches have considered the underlying dynamics of collapse and the mechanisms regulating honey bee population dynamics. A simple model of in-hive and forager worker bees and the transitions between these showed that beneath a critical death rate of foragers, the colony can survive (Khoury et al., 2011). Developments upon this framework to include more complicated aspects of the hive were analysed with similar results (Khoury et al., 2013). Seasonal and annual fluctuations within another model predicted that death rates, food and transitions from in-hive to foraging tasks can influence colony survival (Russell et al., 2013). Population based Allee effects were shown to induce failure of the hive (Dennis and Kemp, 2016) and investigations into the effects of sublethal stress on colony function demonstrated that positive density dependence can cause either exponential growth or failure of the colony (Bryden et al., 2013). Other models incorporating the effects of stressors have been shown to cause colony failure such as infection (Kribs-Zaleta and Mitchell, 2014), American Foulbrood disease (Jatulan et al., 2015) and the interaction between Varroa destructor and Actute Bee Paralysis Virus (Ratti et al., 2015).

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

2. Methods

The structure of the honey bee hive is complex (Seeley, 1995), and many mathematical models have tried to express and explain the major regulatory systems observed in real hives. The model we present in Figure 1 extends previous model frameworks in Khoury et al. (2011) from which we formulate the basic processes governing the hive. We make the simplification to consider only the in-hive worker (H) and outside-of-the-hive worker or forager (F) populations, and assume all bees can be classified in this way. Because in-hive mortality is extremely low compared to that among foragers (Visscher and Dukas, 1997), we assume that all natural mortality occurs in the forager class, at a rate m. Honey bees enter the hive through the eclosion function E, and are recruited into the forager class through the recruitment function R. We assume that a proportion of the colony is lost to a generalised stress function, which induces a lethal effect (Staveley et al., 2014), through an individual's total disappearance from the colony caused by the effects of pesticides causing navigational problems for foragers never returning back to the hive (Bortolotti et al., 2003), or that of density dependent in-hive worker bee mass disappearance, present in CCD situations (VanEngelsdorp et al., 2009). We assume that stress S as a function of time t acts across both in-hive and forager compartments, as an Allee effect. As each individual stressor impacts different classes of honey bee in a different way, we make this assumption to simplify all stresses into a single function. We did this under the knowledge that the location of stress within the model does not impact the qualitative dynamics of the model (Supplementary Figure S6). We also model density-dependent limiting effects at large colony sizes via the function C. We can express the model with this additional general stressor term and additional large colony limiting effect as a two dimensional system of differential equations:

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

$$\frac{dH}{dt} = E(H,F) - R(H,F)H - S(H,F)H - C(H,F)H \tag{1}$$

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

$$\frac{dF}{dt} = R(H,F)H - mF - S(H,F)F \tag{2}$$

Following Khoury et al. (2011), we assume that the maximum eclosion of brood is equivalent to the laying rate L of the queen, and converges to L as $H + F$ gets large. Maximum eclosion occurs when the total size of the colony is large, representing the case when the total adult honey bee population is able to raise all eggs to adulthood (Winston, 1991). The parameter ω sets the speed at which total eclosion tends towards the maximum eclosion L. We make this assumption because the total number of eclosing eggs in honey bee hives is proportional to the number of adult bees in the colony (Allen and Jeffree, 1956; Harbo, 1986).

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

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

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

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

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

where per capita mortality is inversely proportional to the operational colony size. The rate of stress can be expressed as μ, and the low colony mortality can be controlled via ϕ. The limiting function at high densities is proportional to the total colony size

$$C(H,F) = \gamma(H+F) \tag{6}$$

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

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

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

These equations were analysed using the standard methods from dynamical systems theory. The equations were solved numerically with Wolfram Mathematica version number *10.0.2.0*. Numerical bifurcation plots were produced using the package MatCont in MATLAB version number *8.6 R2015b*. We parameterise the model according to previous empirical and theoretical studies as shown in Table 1.

3. Results

There are two fixed points in system (7)

$$(H, F) = (0, 0) \tag{8a}$$

$$(H, F) = (H^*, F^*) \tag{8b}$$

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

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

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

We calculate the Jacobian matrix for system (7) evaluated at the fixed point $(H, F) = (0, 0)$

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

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

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

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

or, when (12) holds true

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

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

Two qualitatively distinct dynamical outcomes are possible within our model. Either the colony size over time reaches a positive stable equilibrium which represents the optimal size of the colony or the population decreases rapidly around a critically low density colony size and the hive collapses. These two possibilities are dependent on initial conditions and parameter choice. This dynamical behaviour is summarised in Fig. 3a, which shows the effect of increasing the stress parameter μ on the total numbers of the adult in-hive and forager bees. In the stress free population and for stress levels less than the critical level, the model predicts that the population will reach equilibrium if the initial density is high enough. As the stress parameter μ is increased, total density drops, and then we observe a tipping point at the critical level of stress. If initial population sizes are below the unstable population size (Fig. 3b), then we predict the extinction of the hive. Otherwise, all populations will grow and tend towards the stable branch, and remain stable (Fig. 3b).

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

Fig. 5 shows the point of colony failure as a function of stress and other critical parameters, highlighting the relationship between the major regulatory functions of the honey bee hive and the hive's response to stress. Higher levels of laying by the queen, lower levels of forager recruitment and lower natural forager mortality can all counteract high levels of stress impacting the colony. Interestingly, our model predicts that varying the level of social inhibition can not save the colony from extinction at high stress levels.

4. Discussion

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

The regulatory functions governing honey bee hives are well understood. It is well documented that the hive will respond to higher levels of mortality of foragers by speeding up recruitment to form a workforce primarily made of precocious foragers (Huang and Robinson, 1996), a process that is thought to be one of the symptoms of CCD (Khoury et al., 2011). It is also understood that the queen is influenced by many factors including seasonality, total available resources, queen age, temperature in the hive, and photoperiod (Shehata et al., 1981; Kefuss, 1978). Therefore regulatory functions could have significant implications in maintaining the colony under stress, and could also be influenced by these stresses. Through investigating the relationship between stress and the major regulatory functions of honey bee hives, we make predictions reflecting the nature of colony collapse. Near the critical threshold inherent in our model, both an increase in recruitment to the forager class or a decrease in social inhibition can cause sudden colony failure. This suggests that CCD can be promoted by a breakdown in these simple regulatory functions that usually maintain honey bee hives under stress. We also predict that fluctuations in the queen's laying rate are highly sensitive in failing colonies. A small decrease in the laying rate of the queen subject to these natural fluctuations close to the bifurcation point could result in drastic switches in the dynamics of the hive, although colonies will normally replace the queen if she is not adequately laying enough brood (Winston, 1991), which could potentially occur before this critical point. In addition, the model proposes that a small increase in natural mortality can cause the sudden collapse of the colony, although the occurrence of mortality fluctuations are unlikely in summer conditions given the observed constant probability of death per unit time spent away from the hive (Visscher and Dukas, 1997), thus making natural mortality less likely to be subject to bifurcation-causing fluctuations.

The intrinsic bistability and sensible ecological behaviour present within our model implies an alternative route to colony collapse through the presence of a saddle-node bifurcation, not seen in other theoretical studies of honey bee population dynamics (Khoury et al., 2011; Bryden et al., 2013). Although some empirical replications of CCD have observed sudden declines in honey bee populations exposed to stressors such as the neonicotinoid imidacloprid (Lu et al., 2012), an insecticide thought to cause abnormal foraging behaviour (Yang et al., 2008), further work is needed to understand the mechanisms governing honey bee failure. Our generalised approach to modelling stress can, in theory, be thought of as acting through any possible mortality-based hive-wide stressor, such as other pesticides (Henry et al., 2012), the mite *Varroa destructor* (Dainat et al., 2012) or the pathogen *Nosema ceranae* (Bromenshenk et al., 2010). If it shown that honey bee hives exhibit bistability, we may be able to forecast the period leading up to the critical transition, and provide new ways of detecting imminent CCD.

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

Currently, we concentrate on the two most significant distinct adult classes (Seeley, 1995), the in-hive and forager worker bees. We make this simplification as there is a clear distinction in mortality rates and behaviour between these two populations, and together they express the most important regulatory processes in the hive (Seeley, 1995). However we could extend the model to include the population dynamics of bees from either the nest centre (cleaning and feeding) or nest periphery (receiving, packing and storing nectar) (Seeley, 1995). For example, we did not consider the regulatory processes governing receiver honey bees for which the dynamics are well known. Forager bees collect nectar and transfer it to receiver bees who then proceed to store this material in cells (Ratnieks and Anderson, 1999). Under higher influxes of nectar, the colony can allocate more honey bees into the receiver bee class (Seeley et al., 1996), and thus can be thought of as another regulatory process maintaining the colony. This introduction of a new classification of honey bee into our modelling framework would help describe the breakdown in regulatory processes of a honey bee hive under CCD conditions in more detail.

In recent years, researchers have become interested in forecasting transitions of state in the underlying dynamics of a wide range of systems (Venegas et al., 2005; Litt et al., 2001; McSharry et al., 2003; May et al., 2008; Scheffer et al., 2001). If bistability is important in understanding the general mechanisms governing a honey bee hive under stress, then we should be able to predict the onset of colony collapse. The model described in this paper has the required properties needed to detect critical transitions before they drastically alter the population dynamics of the system. The existence of a set of predictors called early warning signals (EWS) can be applied to any system with sudden changes in state (Scheffer et al., 2001). When a system undergoes significant change from one state into another state, just before the transition it approaches the tipping point or critical threshold, as shown in the dynamics of our model. Sometimes these changes in state can be catastrophic and widespread, having a detrimental ecological impact on the system as a whole (Scheffer et al., 2001; Folke et al., 2004), with the system sometimes never returning to its original state, even after pre-collapse conditions have been restored (Scheffer et al., 2001). The potential implications and applications of these EWS combined with our model are numerous and may provide the much needed insight into the complex problem of CCD.

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Figures and Tables

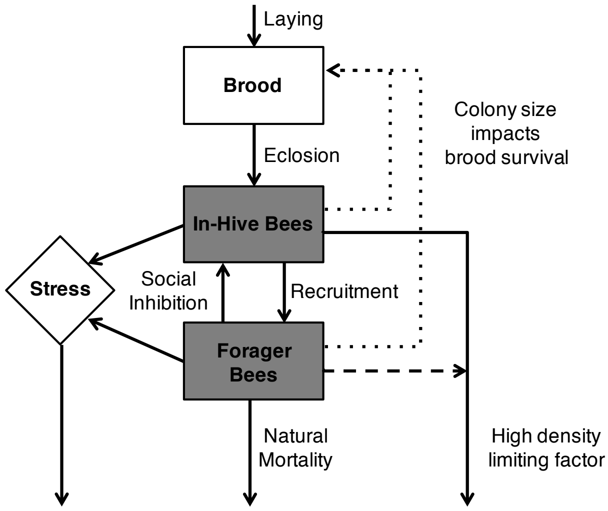


Figure 1: Dynamics of the model. The queen lays eggs which eclose into adult in-hive bees. Total adult population size impacts brood survival. A proportion of the in-hive bees are recruited into the foraging class by the natural age-dependent structure of the hive. Forager bees are able to make the switch 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.

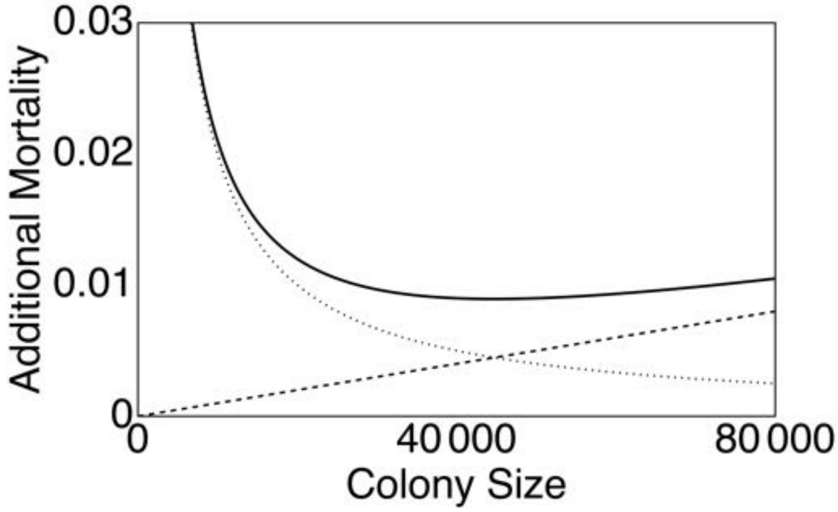
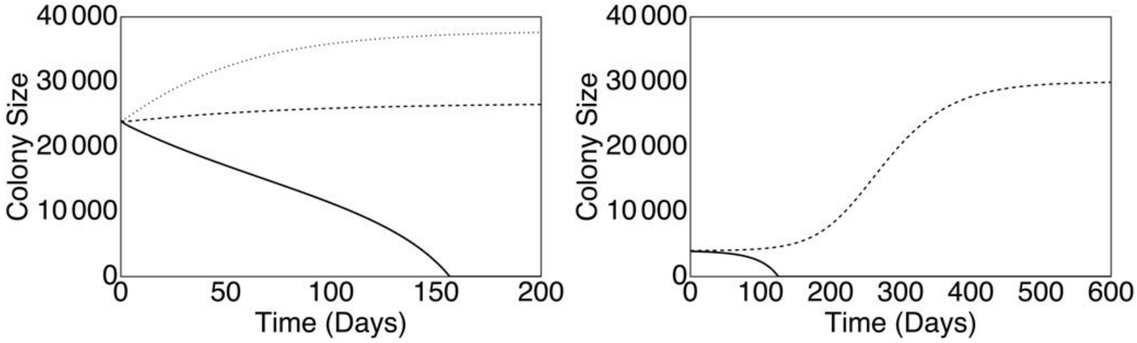


Figure 2: The effects of stress $S(H,F)$ (dotted) and high density function $C(H,F)$ (dashed) on in-hive mortality, and the combined effect (black), as the colony size (H+F) increases. Parameters are $\mu = 200$, $\phi = 0.402$, $\gamma = 0.0000001$. In our model, the stress function $S(H,F)$ acts strongly 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.



(a) (b)

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.

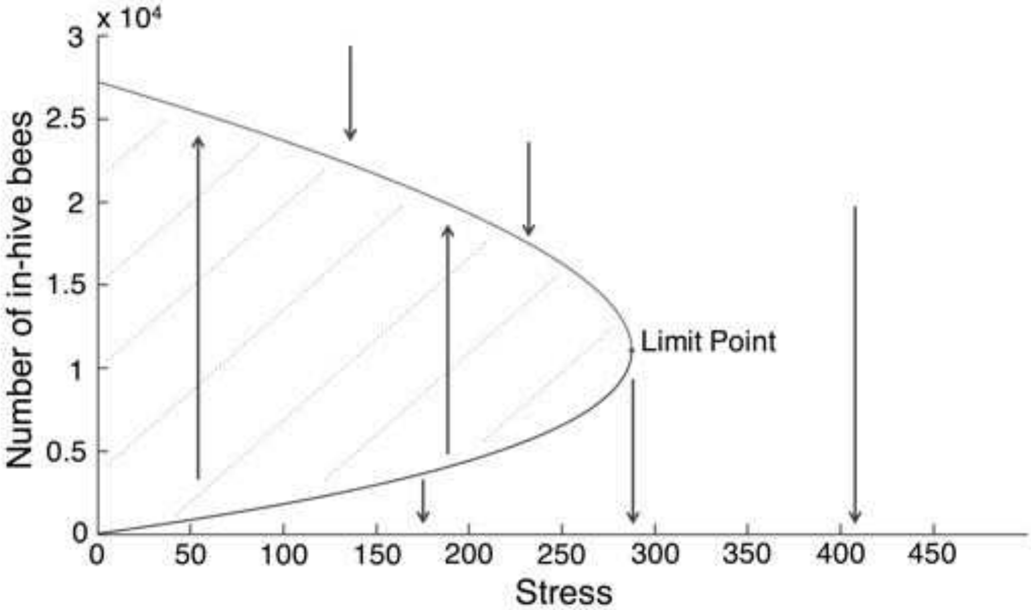
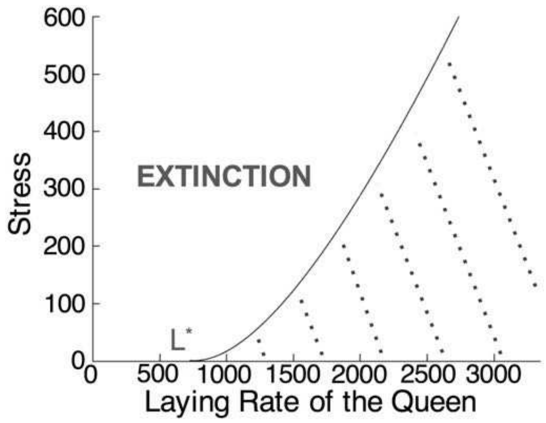
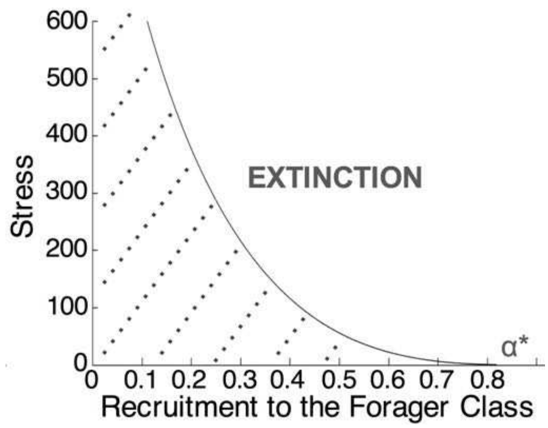


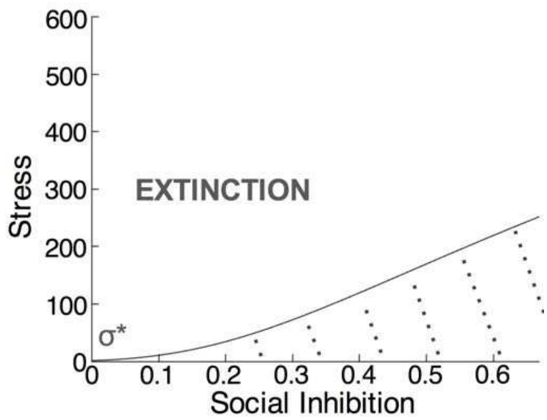
Figure 4: The saddle-node bifurcation through the stress parameter μ for the total numbers of in-hive honey bees. Parameters are taken from Table 1. The location of the limit point represents a critical stress level after which the total number of in-hive bees will become 0. The existence of the unstable 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.



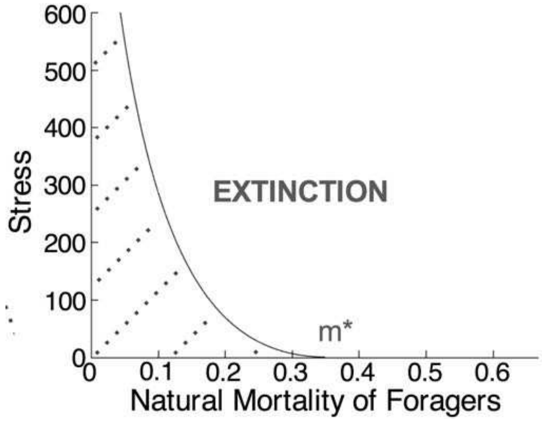
(a) Laying rate L and stress μ.



(b) Recruitment α and stress μ.

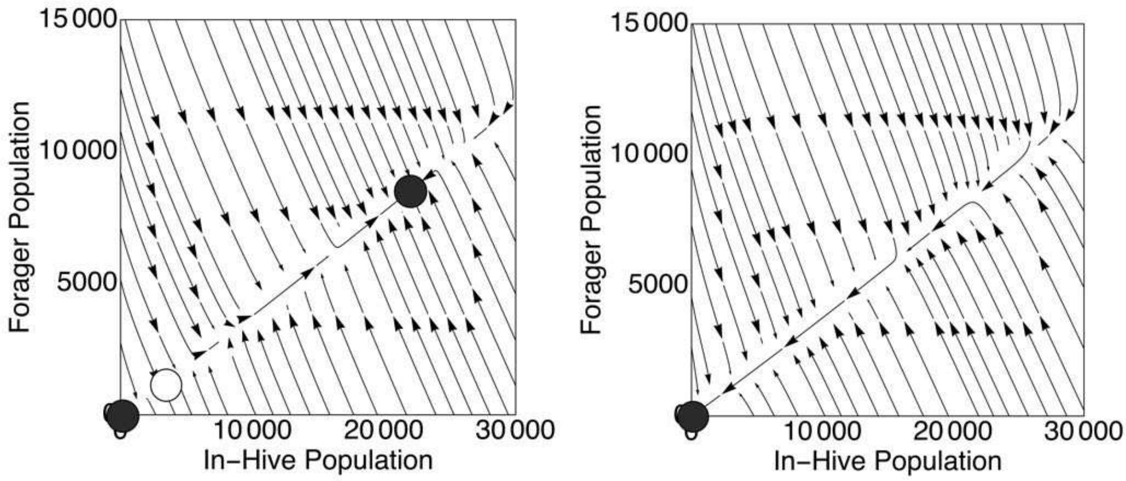


(c) Social inhibition σ and stress μ.



(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.



(a) Stress $\mu = 150$ (b) Stress $\mu = 400$

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.

Parameter	*Description*	*Value*	*Reference*
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	*chosen to be small*
k	Rate at which social inhibition tends to maximum	0.1	*arbitrary*
γ	High density effect	0.0000001	*chosen to be very small*
μ	Stress	$[0, 2000]$	*varying*

Table 1: Model Parameters