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Colony response to graded resource changes: an analytical model of the influence of genotype, environment, and dominance

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Abstract

Successful social groups must respond dynamically to environmental changes. However, a flexible group response requires the coordination of many individuals. Here we offer a static analytical model that integrates variation in environment-based cues for performance of a task with genetically and environmentally based variation in individual responses, and predicts the resultant colony behavior for that task. We also provide formulae for computing effective number of alleles in a haplo-diploid colony founded by any number of parents. Variable colony resources combined with variation among worker phenotypes generate known patterns of colony flexibility, allowing us to explicitly test how the number of loci, dominance/codominance, and the phenotype's environment influences group response. Our model indicates that the number of loci strongly influences colony behavior. For one or two loci, the proportion of workers foraging for pollen remain constant over vast increases in colony pollen stores, but then drops dramatically when the pollen stores increase past a specific threshold. As the number of loci controlling pollen foraging increases, graded increases in pollen stores result in a graded drop in the proportion of the worker population foraging for pollen. The effect of number of alleles is less strong, a result we discuss in light of the fact that a low number of effective alleles are expected in a colony. Comparisons of our model with empirical honey bee (*Apis mellifera*) data indicate that worker foraging response to pollen stores is driven by one or two loci, each with dominant allelic effects. The growing body of evidence that genotype has strong effects on task performance in social insect colonies, and the variation in within-colony genetic diversity across social insect taxa, make our model broadly applicable in explaining social group coordination.

1. Introduction

Social groups must respond quickly and flexibly to changes in their internal and external environment. Because social groups are made up of individuals, this flexible group response requires coordinated individual responses. The mechanisms groups use to integrate these individual responses remain key to our understanding of social group organization. In this paper we present a non-evolutionary analytical model in which we integrate variation in task stimulus cues with genetically based variation in individual task propensity, and determine how these two factors together can generate known patterns of colony flexibility. Our model focuses particularly on the effects of variation in locus and allele number, as well as dominance and codominance, on individual response. We model honey bee (*Apis mellifera*) worker behavior, specifically pollen foraging, because it allows us to directly compare the model's output to empirical data. However, we believe that our model has general applicability in explaining task organization across social groups.

Current models of behavioral organization in large social groups fall into two categories: those in which individuals vary task performance based on variation in environmental cues (Seeley, 1985; Seeley and Levien, 1987; Robinson and Page, 1989b; Page and Robinson, 1991; Seeley et al., 1991; Tofts and Franks, 1992; Franks and Tofts, 1994; Bonabeau et al., 1996; Gordon, 1996; Beshers and Fewell, 2001), and those that focus on genetic variation in individual task performance (Hellmich et al., 1985; Calderone and Page, 1988; Frumhoff

and Baker, 1988; Robinson and Page, 1988, 1989b; Fewell and Page, 1993). Environment-based models focus primarily on the roles of social information transfer and information from the physical environment in eliciting task performance (Camazine, 1991; Seeley et al., 1991; Gordon et al., 1992; Tofts and Franks, 1992; Tofts, 1993; Franks and Tofts, 1994). These models generally assume that, with the exception of age variation, individuals respond similarly to task cues and that variation in task performance is directly related to variation in environmental or social stimuli.

To date, environment-based models have not explicitly included any genetically based variation in motivation to perform a task. There is, however, a strong body of empirical evidence that workers within social insect colonies vary genetically in their sensitivity to task stimuli (Hellmich et al., 1985; Calderone and Page, 1988, 1991; Frumhoff and Baker, 1988; Robinson and Page, 1988, 1989b; Oldroyd et al., 1992; Fewell and Page, 1993, 2000; O'Donnell, 1998). In these studies, workers from the same genetic backgrounds (same patrilines or matrilines) are more likely to perform similar tasks than are less related workers. To explain the link between genetic variation and task performance, a set of response threshold models have been developed (Robinson and Page, 1989a; Page and Mitchell, 1991, 1998; Bonabeau et al., 1996, 1998). In these models, individuals within a colony vary intrinsically in the stimulus level (threshold) for a given task at which they begin to perform that task. Only a narrow subset of the worker pool may perform the task under low stimulus levels. However, as stimulus levels increase, the thresholds of more individuals are met and those workers begin performing the task (Robinson and Page, 1989a; Fewell and Page, 1993, 2000; Fewell and Bertram, 1999). Subsequent work has shown that, in honey bees, this variation is genetically linked; as the stimulus levels for a task increase, the genotypic diversity within that task group increases also (Fewell and Page, 1993, 2000; Fewell and Bertram, 1999). Thus genetic variation influences both task specialization and colony flexibility in response to changes in task need (Robinson and Page, 1989a, b; Fewell and Page, 1993, 1999; Bonabeau et al., 1996; Fewell, 1999; Fewell and Bertram, 1999).

Most response threshold models assume that variation in task sensitivity is a consequence of additive effects of genotype and environment (Robinson and Page, 1989b; Bonabeau et al., 1996), and that the distribution of genetic thresholds in the social group is continuous and normally distributed. However, recent genetic and phenotypic measures of honey bee foraging suggest that genotypic variation in foraging tasks involves a few major loci, and that phenotypic expression of these tasks may be closer to a model of Mendelian dominance (Hunt et al., 1995; Page et al., 2000).

Fewell and Bertram (1999) created a verbal model of how loci number and dominance or codominance may influence colony response to changing resource need. Honey bees collect two resources, pollen and nectar, and they regulate the number of pollen foragers around variation in colony need and pollen storage levels. Fewell and Bertram (1999) assumed that individual workers had specific genetically based response thresholds for pollen foraging. Thresholds were assumed to be either quantitative traits (with a normal distribution of thresholds) or traits expressing Mendelian dominance (a bimodal distribution of thresholds). They predicted that with quantitative traits, colonies would respond to graded increases in task stimuli by gradually increasing the number of workers in that task group. With Mendelian dominance, they predicted that colonies would respond to graded changes in task stimuli in a step-wise fashion. Their empirical data, based on gradually changing the colony pollen stores in several small honey bee (*Apis mellifera*) hives and measuring the behavioral and genotypic changes in the foraging population of workers, suggest that colonies show a sharp decrease (step-wise change) in foraging activity as pollen storage levels moved above a set point. Changes in pollen foraging were also accompanied by a corresponding change in the genotypic diversity of pollen foragers (Fewell and Bertram, 1999), consistent with a response threshold model.

The Fewell and Bertram study provides an empirical basis to test a genetically based model of task regulation. However, the important question of how genotypic variation relates to colony response needs to be addressed quantitatively. We tested this question using a static analytical model that determines honey bee colony response to graded changes in task stimuli (colony pollen stores) for pollen collection. Our model is genetically based. We varied: (1) the number of loci and alleles controlling individual response thresholds for pollen foraging, (2) the allele frequencies within the colony, (3) the stimulus threshold phenotype generated by each allele, (4) the influence of dominance versus codominance, and (5) the level of variation in phenotype generated by random environmental effects. We then determined how these variables influenced colony level response to graded changes in pollen need using the proportion of workers in the hive that forage for pollen. We compared our model's output to empirical data collected on honey bee pollen foraging and behavioral genetics (from Fewell and Bertram, 1999) to determine how well it corresponds to reality.

2. Methods

Our model focuses on explaining the role of genetics and environment in how colonies respond quickly and

flexibly to internal and external environmental changes. We built a genetic model of worker foraging behavior using an analytical approach. The model output generates a colony-level response curve that relates changes in the proportion of workers in the hive foraging for pollen to variation in colony pollen stores. Because we are interested in colony response, our focus is on foragers, i.e., female workers. Our model does not extend across generations, but instead is evolutionarily static, because changes in colony pollen stores occur frequently within a generation.

In the model, workers were assigned genetically based thresholds for the performance of pollen foraging. Individual thresholds contained both genotypic and environmental components, determined by a specified number of loci (N) and alleles (k_m), as well a Gaussian variance (σ) around the genotypic effects to simulate a random environmental component. Phenotypes of each allele were input as the level of colony pollen stores below which an individual worker would forage (φ_i^m). These phenotypes were then combined for alleles at all loci. When colony stimulus levels met an individual's threshold, that individual was added to the already existing assemblage of pollen foragers. Model output was a function, $response(t)$, that related a given level of colony pollen stores t to the proportion of workers in the hive that forage for pollen. Following our description of the response function, we show how to compute the effective number of alleles in the colony, an important model parameter.

2.1. Computing the response function

This is a simple closed form solution, not an iterative model. Given the inputs of allele frequencies, phenotypes, and environmental variance, the output of the model is computed by the formula:

$$response(t) = \frac{1}{2} \sum_{(x_1 x_2 \ldots x_M)} f(x_1 x_2 \ldots x_M)$$
$$\times \left[1 - erf\left(\frac{t - \Phi(x_1 x_2 \ldots x_M)}{\sigma\sqrt{2}} \right) \right], \qquad (1)$$

where t is the amount of colony pollen stores, M is the number of loci, x_i is the genotype of a diploid locus, f is genotype frequency over all N diploid loci, Φ is phenotype over all M diploid loci, and erf is the conventional error function. The response is summed over all possible genotypes within the colony.

We modeled random environmental effects as a Gaussian distribution around the threshold generated by the genotypic effects component. To run the model without any environmental effects, we set the standard deviation to a very small positive number in comparison with the range of the phenotypic values (five orders of magnitude smaller than mean phenotype).

Eq. (1) can now be derived as follows. The inputs to the model are:

i = first of two alleles at a diploid locus,
j = second of two alleles at a diploid locus,
k_m = number of alleles at the mth locus,
M = number of loci,
σ = standard deviation of environmental effects on phenotype,
q_i^m = frequency of allele i at locus m,
φ_i^m = phenotype of allele i at locus m, which is the threshold at which an individual that is homozygous for this allele would begin foraging.

NB: To invoke the function $erf(t)$, we must have $\varphi_i^m \geq 0 \; \forall q_i^m > 0$. This can be relaxed by adding $min\,\varphi = |min\{\varphi_i^m\}|$ to all values of φ_i^m, and then computing $\underset{i,m}{response}\,(t - min\,\varphi)$, instead of $response\,(t)$.

We also introduce the following notation:

F = genotype frequency at a single diploid locus ("f" for frequency),
f = genotype frequency at all N diploid loci,
φ = phenotype of a single haploid allele ("phi" for phenotype),
ϕ = phenotype of a single diploid locus,
Φ = phenotype of all N diploid loci,
t = arbitrary threshold value ("t" for threshold),
x_m = an index of all the (i, j) diploid genotypes at the mth locus ("x" for index).

We first indexed the diploid genotypes at each locus. With k alleles, there were k homozygotes and $\frac{1}{2}k(k-1)$ heterozygotes at that locus. We first depicted these homozygotes and heterozygotes in a two-dimensional punnett square array. To ease computation in subsequent steps, we used indexing to convert the two-dimensional to a 1-dimensional array, as follows:

$$x_m = i \quad \forall i = j \text{ (i.e. homozygotes)},$$

$$x_m = j + \left(k_m - \tfrac{1}{2}i - \tfrac{1}{2}\right)i \quad \forall j > i \text{ (i.e. heterozygotes)}.$$

We assumed all workers were female and therefore diploid, and then computed the effects separately for each diploid locus in the population by computing the frequencies of all possible genotypes and their associated phenotypes. Allele frequencies were assumed to be in Hardy Weinberg equilibrium with no epigenetic effects. For a given locus, we either assumed additivity, where we averaged the phenotypes of the alleles on the two homologous chromosomes, or we assumed dominance. With additivity, any number of alleles was allowed at a locus; with dominance, only two alleles were allowed. Our model allows for a combination of additive and

dominant loci to generate an individual phenotype. We first compute the effects on a per locus basis, as follows:

Frequency of diploid genotypes at locus m:

$F_i^m = (q_i^m)^2$ for homozygotes,

$F_x^m = 2q_i^m q_j^m$ for heterozygotes.

Phenotype of diploid genotypes at locus m:

$\phi_x^m = \frac{1}{2}[\varphi_i^m + \varphi_j^m]$ for additivity,

$\phi_1^m = \varphi_1^m; \quad \phi_2^m = \varphi_2^m; \quad \phi_3^m = \varphi_1^m$ for dominance.

To compute the effects between diploid loci, we assumed no linkage or epistasis. Lack of linkage presumes that each locus is on a separate chromosome. Independent assortment implies that genotype frequencies at one locus are independent of those at other loci. Therefore, we multiplied per locus genotype frequencies to compute the overall genotype frequency over all diploid loci. Lack of epistasis implies additivity between loci, therefore phenotypic effects amongst all diploid loci were averaged. We then combine the effects of all diploid loci, by running the following computations for all possible values of $(x_1 x_2 \ldots x_M)$:

$$f(x_1 x_2 \ldots x_M) = \prod_{j=1}^{M} F_{x_j}^{j}$$

is the frequency of the genotype $(x_1 x_2 \ldots x_M)$,

where

$$x_j = 1, \ldots, k_j + \binom{k_j}{2},$$

i.e. $x_j = 1, \ldots, [k_j + \frac{1}{2}k_j(k_j - 1)]$,

$$\Phi(x_1 x_2 \ldots x_M) = \frac{1}{M} \sum_{j=1}^{M} \phi_{x_j}^{m_j}$$

is the phenotype of $(x_1 x_2 \ldots x_N)$,

$$response(t) = \frac{1}{2} \sum_{(x_1 x_2 \ldots x_M)} f(x_1 x_2 \ldots x_M)$$
$$\times \left[1 - erf\left(\frac{t - \Phi(x_1 x_2 \ldots x_M)}{\sigma\sqrt{2}}\right)\right],$$

where this final summation is over all possible values of $(x_1 x_2 \ldots x_M)$, where

$$erf(t) = \frac{1}{\sqrt{\pi}} \int_0^t e^{-x^2} dx,$$

where t is the foraging threshold and the cumulative distribution function of $N(\mu, \sigma)$ is

$$\frac{1}{2}\left[1 - erf\left(\frac{t - \mu}{\sigma\sqrt{2}}\right)\right].$$

We set the colony storage levels to match empirical data on the levels of pollen stored in small colonies (10–20,000 workers) to allow us to directly compare our model output with results from Fewell and Bertram's (1999) empirical research. This allowed us to compare changes in the number of pollen foragers predicted by the model to actual experimental data resulting from changing the amount of pollen stored in hives. Appendix A contains the MatLab source code for this section.

2.2. Computing average number of effective alleles in the colony

Although population genetic studies may tell us the number of alleles per locus throughout a *population*, they do not necessarily indicate how many fewer alleles there are in a single *colony*. We, therefore, must take into account this sampling, including the breeding system. In particular, there is typically only one diploid honey bee queen per colony, which reduces the number of alleles contributed by females to two per locus. Although there is typically more than one male contributing genes to workers in a colony, there are still only a finite number and they only contribute one allele per haploid male. Throughout this section, we only consider a single locus at a time and assume that there are N alleles at that locus throughout the population.

Before delving into how to estimate number of alleles in the colony, we first deal with the problem that Eq. (1) only specifies a single number of alleles per locus, not the number of alleles per locus for females and males, separately. Our model specified a single number of alleles, k_m, per locus for all parents, in which case the number of diploid genotypes per locus equals $G = \frac{1}{2}k_m(k_m + 1)$. We could reformulate our model in Eq. (1) with separate numbers of alleles for female and male parents. But, instead, we continue using the model implicit in Eq. (1) by producing a single effective number of alleles per locus from the separate numbers of alleles for female and male parents. To do this, note that $\frac{1}{2}k_m(k_m + 1) = \frac{1}{2}(k_m + \frac{1}{2})^2 - \frac{1}{8}$, therefore

$$k_m^* = \sqrt{2G + \frac{1}{4}} - \frac{1}{2}, \tag{2}$$

where k_m^* represents the effective number of alleles in the colony (for a single locus). With a single queen and an infinite number of males, the effective G must be less than $2k_m$, where here k_m represents the number of alleles in the population. Therefore $k_m^* < \sqrt{4k_m + \frac{1}{4}} - \frac{1}{2}$. With a single queen and β males (where $\beta < k_m$), $G < 2\beta$ hence $k_m^* < \sqrt{4\beta + \frac{1}{4}} - \frac{1}{2}$. With α queens (where $2\alpha < k_m$) and an infinite number of males, $G < 2\alpha k_m$ hence $k_m^* < \sqrt{4\alpha k_m + \frac{1}{4}} - \frac{1}{2}$. Generally, with α queens and β males, $G < 2\alpha\beta$ hence $k_m^* < \sqrt{4\alpha\beta + \frac{1}{4}} - \frac{1}{2}$.

The upper bounds given in the previous paragraph for k_m^* are overestimates because, thus far, we have severely overestimated the number of alleles found amongst the parents in the colony. First consider the case of a colony with a single queen and β males, within a population that contains N alleles. It is highly unlikely that all β males will have different haploid genotypes at a locus. We compute the average number of distinct alleles that will be drawn from a sample of random males. Assume that all allele frequencies are identical in the population (not in the colony), i.e. a uniform distribution of alleles. This assumption of a uniform distribution provides the most generous number of possible alleles in the β males in the colony. Other distributions will yield smaller estimates for the upper bound of k_m^*.

Next we compute the probability of the males in the colony collectively containing exactly $\beta - k$ distinct alleles given that the population at-large from which the males are drawn contains a total of N alleles. We shall call this.

$$Pr_{\text{males}}(\beta - k; N) = Pr(\beta - k \text{ alleles in colony};$$
$$\text{given } N \text{ alleles in population})$$

To do this, we first compute the probability of there being at least $\beta - k$ distinct alleles in the colony as

$$Pr_{\text{male}}^{\text{cum}}(\beta - k; N) = \frac{N!}{(N - \beta + k)!}\left(\frac{1}{N}\right)^{\beta - k}. \tag{3}$$

For $\beta \leqslant N$, perform the above computation for all integer values of k from zero to $\beta - 1$.
For $\beta > N$, perform the above computation for all integer values of k from $\beta - N$ to $\beta - 1$.

That is, if $\beta > N$ and $0 < k < \beta - N$, then set $Pr_{\text{male}}^{\text{cum}}(\beta - k; N) = 0$. The first factor in the above equation represents the number of ways one can select $\beta - k$ distinct alleles. The second factor represents the probabilities of choosing each of the $\beta - k$ distinct alleles. Had we included a third factor representing the probabilities for choosing the remaining k alleles, then $Pr_{\text{male}}^{\text{cum}}(\beta - k; N)$ would have represented the probability of picking exactly $\beta - k$ distinct alleles, i.e. $Pr_{\text{male}}(\beta - k; N)$. But, since we are not specifying the remaining k alleles, Eq. (3) yields the cumulative distribution $Pr_{\text{male}}^{\text{cum}}(\beta - k; N)$ of $Pr_{\text{male}}(\beta - k; N)$, and we can compute $Pr_{\text{male}}(\beta - k; N)$ by taking successive differences:

$$Pr_{\text{male}}(\beta - k; N) = Pr_{\text{male}}^{\text{cum}}(\beta - k + 1; N) - Pr_{\text{male}}^{\text{cum}}(\beta - k; N).$$

The expected number of alleles contributed by β males is therefore

$$E_{\text{males}}(\beta; N) = \sum_{k=1}^{\beta-1} k \cdot Pr_{\text{male}}(\beta - k; N). \tag{4}$$

The corresponding equation for the probability that α queens will contribute at least $2\alpha - j$ alleles is slightly more complicated because queens are diploid:

$$Pr_{\text{female}}^{\text{cum}}(2\alpha - j; N) = \frac{N!}{(N - 2\alpha + j)!}\left(\frac{1}{N}\right)^{2\alpha - j}. \tag{5}$$

For $\alpha \leqslant \frac{1}{2}N$, perform the above computation for all integers from j from zero to $2\alpha - 1$.
For $\alpha > \frac{1}{2}N$, perform the above computation for integer values of j from $2\alpha - N$ to $2\alpha - 1$.
That is, if $\alpha > 1/2N$ and $0 < j < 2\alpha - N$, then set $Pr_{\text{female}}^{\text{cum}}(2\alpha - j; N) = 0$.

As we did for males, compute $Pr_{\text{female}}(2\alpha - j; N)$ from its cumulative distribution $Pr_{\text{female}}^{\text{cum}}(2\alpha - j; N)$ by computing successive differences:

$$Pr_{\text{female}}(2\alpha - j; N) = Pr_{\text{female}}^{\text{cum}}(2\alpha - j + 1; N)$$
$$- Pr_{\text{female}}^{\text{cum}}(2\alpha - j; N)$$

and the expected number of alleles contributed by α females is therefore

$$E_{\text{females}}(\alpha; N) = \sum_{j=1}^{\alpha-1} j \cdot Pr_{\text{female}}(\alpha - j; N). \tag{6}$$

When a colony is formed by α queens and β males, both drawn from a large population containing a total of N alleles, the expected number of diploid genotypes in their offspring equals

$$\bar{G}(\alpha, \beta; N) = E_{\text{females}}(\alpha; N) \cdot E_{\text{males}}(\beta; N). \tag{7}$$

Here, we assumed random mating, i.e. $E(\alpha, \beta; N) = E_{\text{females}}(\alpha; N) \cdot E_{\text{males}}(\beta; N)$. Eq. (7) provides an estimate of the expected number of diploid genotypes and hence for the effective number of alleles per locus in the colony, which we compute (using Eq. (2)) as $k_m^* = \sqrt{2\bar{G}(\alpha, \beta; N) + \frac{1}{4}} - \frac{1}{2}$. This value of effective allele number can then be used as an input to Eq. (1):

$$response(t) = \frac{1}{2} \sum_{(x_1 x_2 \ldots x_M)} f(x_1 x_2 \ldots x_M)$$
$$\times \left[1 - erf\left(\frac{t - \Phi(x_1 x_2 \ldots x_M)}{\sigma\sqrt{2}}\right)\right]$$

Appendix B contains the MatLab source code for this section.

3. Results

The proportion of workers foraging for pollen decreases monotonically with increasing pollen stores. The response function can take on many shapes, ranging from a step function at one end of a continuum to a gradual sloping smooth curve at the other end. The shape of the response curve is dependent upon the number of loci and alleles, allele frequencies and phenotypic values, whether the alleles express
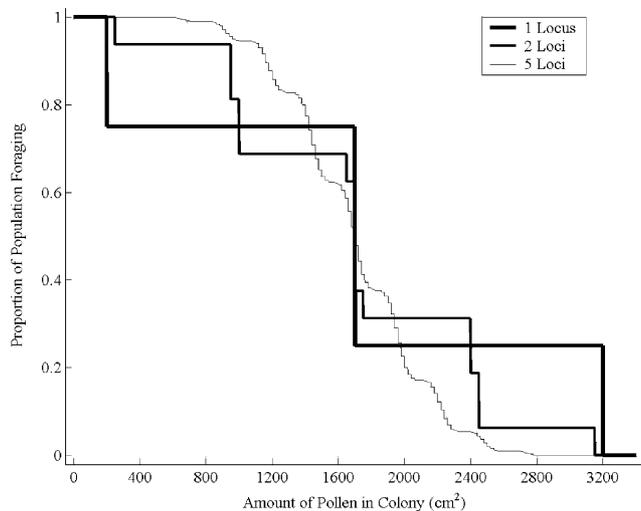
Fig. 1. The influence of varying the number of loci on the colony's response to changes in pollen stores. Each locus had two alleles at equal frequencies, and allele phenotypes at each locus were specified as: Locus 1: 200, 3200; Locus 2: 300, 3100; Locus 3: 400, 3000; Locus 4: 500, 2900; Locus 5: 600, 2800. Environmental variation was minimal (sigma = 0.001).
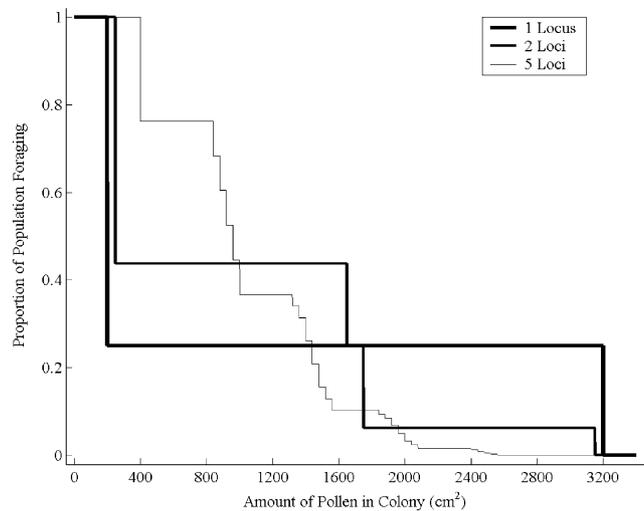

Fig. 2. The influence of dominance on the colony's response to changes in pollen stores. All variables are run with identical values of Fig. 1, except that at each locus, one allele is dominant over the other. Each locus had two alleles at equal frequencies, and allele phenotypes at each locus were specified as: Locus 1: 200, 3200; Locus 2: 300, 3100; Locus 3: 400, 3000; Locus 4: 500, 2900; Locus 5: 600, 2800. Environmental variation was minimal (sigma = 0.001).

dominance or codominance, and the amount of environment influencing phenotypic expression.

Increasing the number of loci while keeping all other parameters constant alters the response curve from a simple step function to a gradual decline in forager numbers with increasing pollen stores (Fig. 1). For one or two loci, the proportion of workers foraging for pollen remains constant over vast increases in colony pollen stores, but then drops dramatically when the pollen stores increase past a specific threshold (setpoint). As the number of loci controlling pollen foraging increases, graded increases in pollen stores result in a graded drop in the proportion of the workers in the colony foraging for pollen.

With the one locus, two allele model, with little environmental influences, we see three distinct classes of individuals, AA, Aa and aa, whose frequencies are 25%, 50%, and 25%, respectively for codominance (Fig. 1 solid line), and two classes of individuals whose frequencies are 75% and 25% for dominance (Fig. 2 solid line). These classes of individuals result in distinct step functions in the model, where the number of workers foraging for pollen changes suddenly with graded changes in colony pollen stores.

When the alleles express dominance instead of additivity, the number of steps in each function is reduced by 25%, except in the one locus two-allele example, where the dominance function was comprised of only two steps while the additivity function included three steps (Fig. 2). Even under a model of dominance, however, with two or more loci, the function shows a gradual decrease in the proportion of workers foraging for pollen with increasing pollen stores.
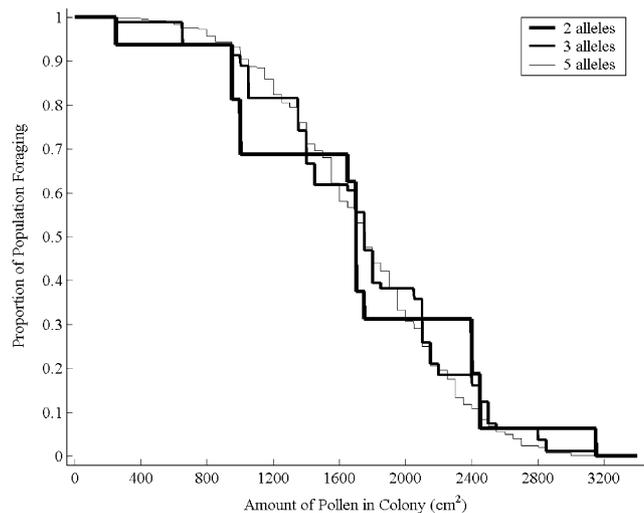

Fig. 3. The influence of varying allele number on the colony's response to changes in pollen stores. There are two loci, each with varying numbers of alleles at equal frequencies, and allele phenotypes at each locus were specified as: Locus 1: 200, 3200, 1600, 800, 2400; Locus 2: 300, 3100, 1500, 900, 2500. Environmental variation was minimal (sigma = 0.001).

Increasing the effective number of alleles, while holding all other parameters constant, alters the response curve from a simple step function to one that incorporates numerous steps (Fig. 3). Allele number does not, however, influence the general shape of the whole response function. In all cases there is a dramatic drop in the number of foragers from 80% to less than 20% of the colony at 1600 cm^2 of pollen stores.

Provided a haplo-diploid colony contains only one queen, it will contain low numbers of effective alleles,
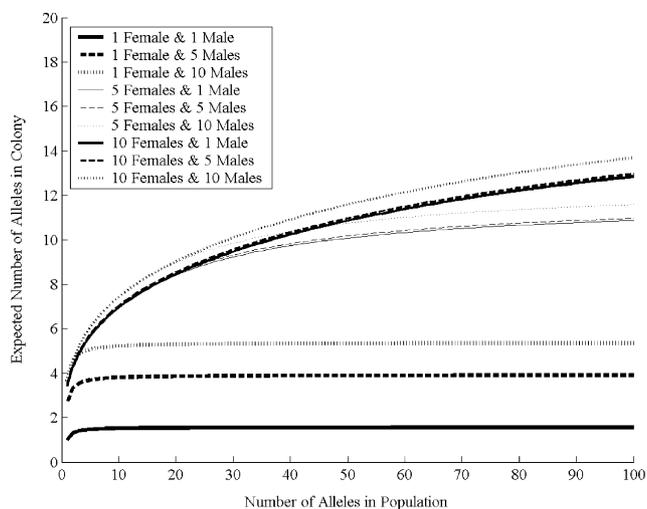
Fig. 4. The influence of varying the number of males a queen mates with, the number of queens in the colony, and the number of alleles per locus in the population at large on the number of effective alleles per locus in the colony.
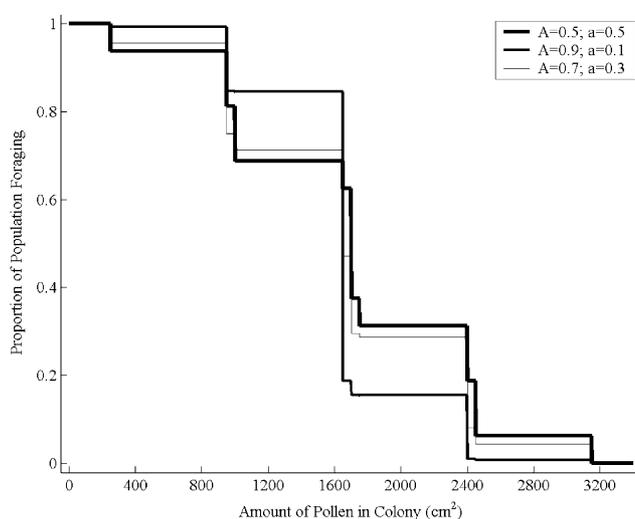

Fig. 5. The influence of varying allele frequency on the colony's response to changes in pollen stores. There are two loci, each with two alleles, the frequencies of which were varied (0.9 and 0.1; 0.7 and 0.3; 0.5 and 0.5). The allele phenotypes at each loci were specified as Locus 1: 200, 3200; Locus 2: 300, 3100. Environmental variation was minimal (sigma = 0.001).
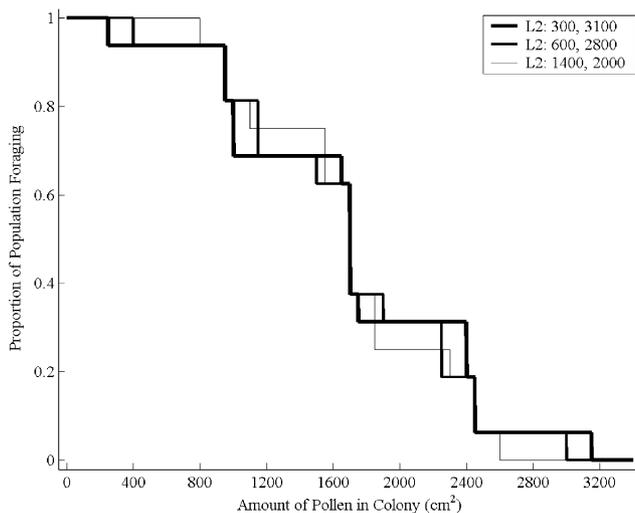

Fig. 6. The influence of varying allele phenotype on the colony's response to changes in pollen stores. There are two loci, each with two alleles at equal frequencies. The phenotypic values of the first loci's alleles are held constant (Locus 1: 200, 3200), and the second loci's alleles are changed with each line-plot. Environmental variation was minimal (sigma = 0.001).

regardless of the number of males a queen mates with or the number of alleles in the population at large (Fig. 4). The number of effective alleles in a colony will be very small (just over one) if there are few alleles in the population or if the queen mates with only one male. Even if there are a large number of alleles in the population and the queen mates with numerous males, the colony will only contain less than six effective alleles. However, if a haplo-diploid colony contains multiple queens each mated multiple times, the number of effective alleles contained in the colony rises dramatically (Fig. 4).

Changing the allele frequency from equal to unequal proportions within the colony alters the fraction of foragers that collect pollen at specific thresholds (Fig. 5), while changing the allele phenotypes alters the colony's set-point (Fig. 6). Neither change, however, alters the step-like nature of the response function to a smoother or more gradual curve.

All aforementioned results include only an infinitesimal level of environmental variation influencing individual phenotypes. Adding an environmental component to phenotypic expression profoundly affects the shape of the response function (Fig. 7). As the environmental component of threshold phenotypic expression increases, the function shifts to a smoothly monotonic response, even when only one or two loci are parameterized.

4. Discussion

In our model, genetic variation strongly influenced how honey bee colonies responded to changes in pollen stores. As colony pollen stores increased, the proportion of workers foraging for pollen decreased. However, the shape of the response function varied between a step-wise decreasing function, and a gradually decreasing function. This shape change depended primarily upon the number of loci; increasing the number of loci beyond four produced a graded smooth response curve.

Shifting from additive effects to dominance altered the curve from a gradually decreasing function to a more step-wise function, but this was again dependent on the number of loci. The relationship between genetic variation and environmental variation in individual
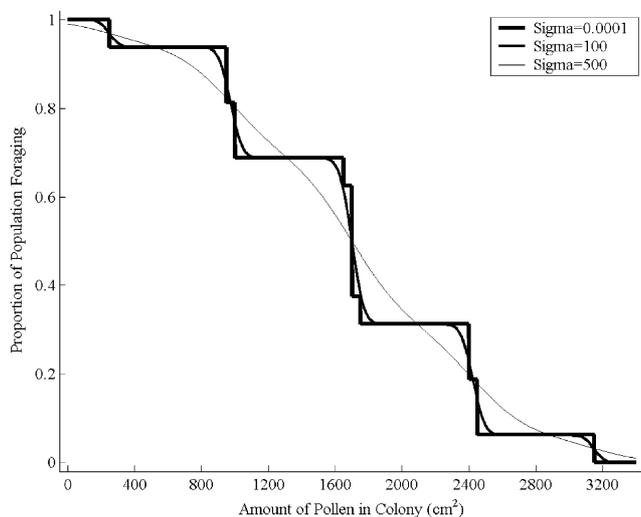
Fig. 7. The influence of increasing environmental effects on the colony's response to changes in pollen stores. All variables are run with 2 loci and 2 alleles at equal frequencies (Locus 1: 200, 3200; Locus 2: 300, 100). Environmental variation was increased with each line plot from sigma = 0.001 to sigma = 1000.

phenotype strongly affected colony-level foraging response. Increasing the environmental variation resulted in a response similar to increasing the number of loci; the curve smoothed from a step-wise to a graded decrease in the proportion of pollen foragers with increasing pollen stores.

Allele number also results in a shape change in the response curve. However, our model suggests that the honey bee breeding system limits the effective number of alleles. In honey bees, one diploid queen mates with several haploid males (average is 10 males with a range of 2–20; Page, 1986). The computations regarding effective allele number show how the number of mates can quantitatively alter the output of our model. Assuming that only one queen founds a colony, Fig. 4 shows that an increase in the number of patrilines from one to ten results in roughly five times more effective alleles. Although this may not seem like a large effect, Fig. 3 shows that the output response becomes smoother as effective allele number is increased from 1 to 5. Our estimates of effective allele number per locus in the colony provide an upper bound, due to our assumption that the alleles in the population at-large are drawn from a uniform distribution. Had we chosen a more biologically realistic—albeit computationally less tractable distribution, such as a Poisson distribution—the effective number of alleles in the colony would be smaller. If a colony were to be comprised of several multiply mated queens the estimate of the effective number of alleles would quickly grow large enough to result in a very smooth response function.

Throughout, we assumed no epistasis, but our results would have been strikingly different had we allowed for non-linear interactions between loci. For example, if a

threshold number of paralogous genes were required to be in a certain state in order to trigger foraging, then the response function would remain step-like even with a large number of loci. Such models are entirely plausible, but such epistatic models for honey bee foraging are *empirically* unknown. This is largely due to it being difficult to estimate epistatic coefficients.

4.1. Comparisons with empirical data

Knowledge of how the underlying genetics influences adaptive behavioral traits is important for understanding the processes of behavioral evolution. We therefore compared the response curves from our analytical model to the response curves from empirical field data (Fewell and Bertram, 1999). Fewell and Bertram measured the foraging behavior of honey bee colonies in which the level of pollen stores within the colony was systematically increased and/or decreased. They found that colonies did not respond gradually to changes in pollen stores; instead, between 400 and 800 cm^2 of pollen stores there was a dramatic two-fold stepwise change in the number of pollen foragers. The change in pollen foraging activity was best fit by a regression model that included a stepwise change. In each colony, the step component accounted for the largest portion of the variance. The colonies showed virtually identical responses to changing pollen levels, even though the pollen levels were increased through the experiment for one colony and decreased through the experiment for the second colony. This response was observed for the foraging portion of the colony as a whole, as well as for individually identified workers that were followed through the course of the experiment.

The only response curves produced by our model that match these step-wise results were produced with one or two loci, each with non-additive (dominant) allelic effects. The stepwise model also appears to be a best fit for colony changes in allocation to nectar collection in response to varying nectar quality at resource stations (Seeley et al., 1991). Therefore, our model suggests that a limited number of loci produce the major genetic effects on this foraging trait. This result is consistent with molecular analyses of Hunt et al. (1995) and Page et al. (2000) indicating that two quantitative trait loci account for the majority of observed variation in pollen collection. This result is also consistent with the findings of Page and Fondrk (1995) that selection for or against high pollen storage levels results in a plateau for colony pollen intake rates within a limited number of generations (four). Current evolutionary thought, however, assumes that most adaptive trait variation results from large numbers of additive gene loci, each with infinitesimal small effects (Falconer, 1981; Barton and Turelli, 1989), although this paradigm has been challenged in the last decade (Orr and Coyne, 1992).

When few loci affect the task threshold, the set-point of a colony's response to changes in task need appears to depend greatly on both the phenotypic threshold for which the allele codes and on the allele frequencies within the colony. When alleles coding for a low threshold phenotype are most common, the colony will have many workers foraging for pollen at high colony storage levels. Conversely, when alleles coding for a high threshold phenotype are most common, the colony will have a lower response set point, resulting in the majority of pollen foragers only starting to forage for pollen when the stores are substantially lower than in the previous scenario.

If so, then a simple switch in either allele frequency or the response threshold for which it codes can generate large colony-level differences in pollen foraging behavior. Such a shift may explain the large differences in African and European honey bee foraging strategy. African colonies have much higher pollen intake rates than European colonies (Danka et al., 1987; Pesante et al., 1987; Schneider, 1989; Schneider and McNally, 1992), which can be traced to genetically based differences in the propensity of African and European workers to forage for pollen (Fewell and Bertram, 2002).

4.2. General applicability

Our modeling approach has philosophical under-pinnings in neurophysiology and theoretical computer science. Two decades ago, researchers investigated how a collection of imprecise and inflexible neurons or electronic components could result in precise and flexible responses (Enright, 1980), work which continues (Needleman et al., 2001). Enright's work has found application in task activity of eusocial insects (Cole, 1991). Our model is supported by one of these applications, in which, "with self-synchronized patterns of activity, a task may be fulfilled more effectively with non-synchronized activity" (Delgado and Solé, 2000). We have built a model with inflexible, non-synchronized individuals in which task activity of the entire colony is finely honed. What is fundamentally new here is that we explicitly model the genetics of the colony.

We base our model on the assumption that the individuals in the colony possess variation in genotype. How does this genetic variation arise? Extensive genetic variation within the colony likely results from polyandrous mating (reviewed by Page, 1986). Queens simultaneously use the sperm from several drones as they lay eggs (Page and Metcalf, 1982; Laidlaw and Page, 1984), resulting in colonies containing numerous subfamilies, each consisting of the offspring of the queen and one of her mates. The assertion of genetic variation in individual propensity to perform a task is valid for diverse social insect taxa, including honey bees (Hellmich et al., 1985; Calderone and Page, 1988, 1991; Frumhoff and Baker, 1988; Robinson and Page, 1988, 1989b; Oldroyd et al., 1992; Fewell and Page, 1993).

Our approach of using a multiple-locus model differs from previous threshold response models. Most models to date make the assumption that individual propensity to perform a task is normally distributed in the population, suggesting that the phenotypes are controlled by quantitative genetics (Robinson and Page, 1989b; Page and Mitchell, 1998). However, recent research suggests that genotypic variation in foraging tasks is most strongly affected by a few major loci, and that phenotypic expression of these tasks may be closer to a model of Mendelian dominance (Hunt et al., 1995; Page et al., 2000).

Because our model focuses on a specific, limited part of the process that leads to worker task performance, it incorporates some simplifying assumptions. For example, it assumes that response thresholds are fixed, but there is evidence that the response thresholds for some stimuli can vary over worker lifetime (Collins, 1980; Allan et al., 1987; Robinson, 1987).

Our model also assumes that all workers are equally likely to encounter all tasks. However, mechanisms for transmitting information about task opportunity and nest mate performance should also be considered, as should the effects of the spatial distribution of tasks and of worker movements. Models of information transfer in social groups often distinguish between two types of information transfer: from the physical environment to the worker (Tofts and Franks, 1992), and via social interactions (Seeley et al., 1991; Gordon, 1996). Foraging is likely to involve both social and physical cues. For example, bees respond directly to changes in pollen amounts encountered in the hive (Camazine, 1993). Workers are also stimulated to collect pollen via changes in brood hunger pheromones (Pankiw and Page, 2001), potentially by tropholactic exchange with the nurse bees that feed brood (Camazine et al., 1998), and by information received on the dance floor from multiple foragers on the location and quality of both pollen and nectar resources.

How would the mechanism of information transfer affect colony level response? Our expectation is that social information transfer should produce a much faster and more universal response to changes in colony stimuli. Social groups in which individuals receive information only as they encounter it in the physical environment are likely to respond more slowly to changes in environmental conditions. In contrast, the combination of an information center model of information exchange coupled with the Mendelian based response thresholds in our model should generate a faster and more stepwise pattern of colony response. This interaction of environmental conditions and individual behavioral patterns seems a remarkably good fit with the empirical systems seen in the honey bee and other complex social groups.

Acknowledgments

Appendix A. MatLab source code for Section 2.1

```
clear variables;

%% Define the environmental influence
global sigma;
sigma = 0.001;

%% Define the phenotypes of the different alleles
%% change values for different phenotype numbers
Phnll(1,1) = 200; % phenotype locus 1 allele 1
Phnll(2,1) = 3200; % phenotype locus 1 allele 2
% Phnll(3,1) = 1600; % phenotype locus 1 allele 3
% Phnll(4,1) = 800; % phenotype locus 1 allele 4
% Phnll(5,1) = 2400; % phenotype locus 1 allele 5
% Phnll(1,2) = 300; % phenotype locus 2 allele 1
% Phnll(2,2) = 3100; % phenotype locus 2 allele 2
% Phnll(3,2) = 1500; % phenotype locus 2 allele 3
% Phnll(4,2) = 900; % phenotype locus 2 allele 4
% Phnll(5,2) = 2500; % phenotype locus 2 allele 5
% Phnll(1,3) = 400; % phenotype locus 3 allele 1
% Phnll(2,3) = 3000; % phenotype locus 3 allele 2
% Phnll(1,4) = 500; % phenotype locus 4 allele 1
% Phnll(2,4) = 2900; % phenotype locus 4 allele 2
% Phnll(1,5) = 600; % phenotype locus 5 allele 1
% Phnll(2,5) = 2800; % phenotype locus 6 allele 2

%% Determine the range of phenotypes
min_phen = min(min(Phnll))−200;
max_phen = max(max(Phnll)) + 200;
range_phen = max_phen−min_phen;
y = min_phen:0.25:max_phen;

%% Define the alleles
N = 1; % N = Number of Loci
for m = 1:N
    k(m) = 2;
end; % end loci loop
```

```
%% Unequal allele frequencies, define matrix qq
for m = 1:N % loci loop
    for i = 1:k(m) % allele loop
        qq(i,m) = 1/k(m);
    end; % end allele loop
end;
q = qq;

%% HOMOZYGOTES
for m = 1:N
    for i = 1:k(m)
        x = i;
        Freqhom(x,m) = q(i,m)^2;
        Phenhom(x,m) = Phnll(i,m);
    end;
end;

%% HETEROZYGOTES
for m = 1:N
    for i = 1:k(m)
        for j = (i + 1):k(m)
            x = j + (k(m)-.5*i-.5)*i;
            Freqhet(x,m) = 2*q(i,m)*q(j,m);
            Phenhet(x,m) = .5*(Phnll(i,m) + Phnll(j,m));
        end;
    end;
end;

for m = 1:N
    totgen(m) = k(m) + .5*k(m)*(k(m)−1);
    totheterogen(m) = totgen(m)-k(m);
    tothomogen(m) = k(m);
end

%% Build homozyg & heterozyg combnd mtrcs
A = zeros(N,totheterogen(m));
A = A';
Phenhom = [Phenhom;A];
Freqhom = [Freqhom;A];
Phen = Phenhet + Phenhom;
Freq = Freqhet + Freqhom;
CDL1 = zeros(size(y')); % Cum Dist for Line 1
for c = 1:totgen(1)
    Freqgen(c) = Freq(c,1);
    Phengen(c) = (Phen(c,1));
    Phengenelm = Phengen(c)*ones(size(y'));
    Freqgenelm = Freqgen(c);
    CDL1 = CDL1 + Freqgenelm*true_erf(y'-
        Phengenelm);
end;

%% Plot
x = 3400:−0.25:0
plot(x,CDL1)
axis([−50 3450 −0.02 1.02])
```

Appendix B. MatLab source code for Section 2.2

```
clear variables;

NumFmls = 1
NumMls = 1
NumAlleles = 100

%% Calculate cum dnsty functns & prob dnsty functn
PCum Fmls(2*NumFmls + 1,NumAlleles) = zeros;
PCum Mls(NumMls + 1,NumAlleles) = zeros;
Prob Fmls(2*NumFmls,NumAlleles) = zeros;
Prob Mls(NumMls,NumAlleles) = zeros;

for alpha = 1:NumFmls % counter number Fmls
  for beta = 1:NumMls counter number Mls
    for N = 1:NumAlleles % counter number alleles
      if beta < = N % Equation (1)
        k = 0;
        E1P1 Mls = (factorial(N)/(factorial(N-
          beta + k)));
        E1P2 Mls = ((1/N)^(beta-k));
        PCum Mls(beta,N) = E1P1 Mls*E1P2 Mls;
        for k = 1:beta-1
        E1P1 Mls = (factorial(N)/(factorial(N-
          beta + k)));
        E1P2 Mls = ((1/N)^(beta-k));
        PCum Mls(beta-k,N) = E1P1 Mls*E1P2 Mls;
        end
        Prob Mls = -diff(PCum Mls);
      end
      if beta > N % Equation (2)
        for k = 1:(beta-N)-1
          PCum Mls(beta-k,N) = 0;
        end
        for k = beta-N:beta-1
          E1P1 Mls = (factorial(N)/(factorial(N-
            beta + k)));
          E1P2 Mls = ((1/N)^(beta-k));
          PCum Mls(beta-k,N) = E1P1 Mls*E1P2 Mls;
        end
        Prob Mls = -diff(PCum Mls);
      end;
      if alpha < = N/2
        j = 0;
        E1P1Fmls = (factorial(N))/(factorial(N-
          2*alpha + j));
        E1P2Fmls = ((1/(N))^(2*alpha-j));
        PCumFmls(2*alpha-j,N) =
          E1P1Fmls*E1P2Fmls;
        for j = 0:2*alpha-1
          E1P1Fmls = (factorial(N))/(factorial(N-
            2*alpha + j));
          E1P2Fmls = ((1/(N))^(2*alpha-j));
          PCumFmls(2*alpha-j,N) =
            E1P1Fmls*E1P2Fmls;
        end
        ProbFmls = -diff(PCumFmls);
      end
      if alpha > N/2
        for j = 0:(2*alpha-N)-1
          PCumFmls(2*alpha-j,N) = 0;
        end
        for j = 2*alpha-N:2*alpha-1
          E1P1Fmls = (factorial(N))/(factorial(N-
            2*alpha + j));
          E1P2Fmls = ((1/(N))^(2*alpha-j));
          PCumFmls(2*alpha-j,N) =
            E1P1Fmls*E1P2Fmls;
        end
        ProbFmls = -diff(PCumFmls);
      end
    end
  end
end

ProbFmls
ProbMls

for i = 1:2*alpha;
  VS(i) = i;
end
for i = 1:beta;
  VT(i) = i;
End

Exp Fmls = VS*ProbFmls
Exp Mls = VT*ProbMls
ExpColony = ExpFmls'*ExpMls
ksubm = sqrt(2*ExpColony + 1/4)-1/2
```

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