THE HARDY-WEINBERG PRINCIPLE AND
ESTIMATING ALLELE FREQUENCIES

Introduction

To keep things relatively simple, we’ll spend much of our time in the first part of this course talking about variation at a single genetic locus, even though alleles at many different loci are involved in expression of most morphological or physiological traits. Towards the end of the course, we’ll study the genetics of continuous (quantitative) variation, but until then you can assume that I’m talking about variation at a single locus unless I specifically say otherwise.\(^1\)

The genetic composition of populations

When I talk about the genetic composition of a population, I’m referring to three aspects of genetic variation within that population:\(^2\)

1. The number of alleles at a locus.
2. The frequency of alleles at the locus.
3. The frequency of genotypes at the locus.

It may not be immediately obvious why we need both (2) and (3) to describe the genetic composition of a population, so let me illustrate with two hypothetical populations:

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\(^1\)You’ll see in a week or a week and a half when we talk about analysis of population structure that we start discussing variation at many loci. But you’ll also see that in spite of discussing variation at many loci simultaneously, virtually all of the underlying mathematics is based on the properties of those loci considered one at a time.

\(^2\)At each locus I’m talking about. Remember, I’m only talking about one locus at a time, unless I specifically say otherwise. We’ll see why this matters when I outline the ideas behind genome-wide association mapping.

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It’s easy to see that the frequency of $A_1$ is 0.5 in both populations, but the genotype frequencies are very different. In point of fact, we don’t need both genotype and allele frequencies. We could get away with only genotype frequencies, since we can always calculate allele frequencies from genotype frequencies. But there are fewer allele frequencies than genotype frequencies — only one allele frequency when there are two alleles at a locus. So working with allele frequencies is more convenient, but we can’t get genotype frequencies from allele frequencies unless . . .

**Derivation of the Hardy-Weinberg principle**

We saw last time using the data from *Zoarces viviparus* that we can describe empirically and algebraically how genotype frequencies in one generation are related to genotype frequencies in the next. Let’s explore that a bit further. To do so we’re going to use a technique that is broadly useful in population genetics, i.e., we’re going to construct a mating table. A mating table consists of three components:

1. A list of all possible genotype pairings.
2. The frequency with which each genotype pairing occurs.
3. The genotypes produced by each pairing.

<table>
<thead>
<tr>
<th>Female × Male</th>
<th>Frequency</th>
<th>Offspring genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A_1A_1 × A_1A_1$</td>
<td>$x_{11}^2$</td>
<td>$A_1A_1$ 0 $A_1A_2$ 0 $A_2A_2$ 0</td>
</tr>
<tr>
<td>$A_1A_2$</td>
<td>$x_{11}x_{12}$</td>
<td>$1/2$ $1/2$ 0</td>
</tr>
<tr>
<td>$A_2A_2$</td>
<td>$x_{11}x_{22}$</td>
<td>0 $1$ 0</td>
</tr>
<tr>
<td>$A_1A_2 × A_1A_1$</td>
<td>$x_{12}x_{11}$</td>
<td>$1/2$ $1/2$ 0</td>
</tr>
<tr>
<td>$A_1A_2$</td>
<td>$x_{12}^2$</td>
<td>$1/4$ $1/4$ $1/4$</td>
</tr>
<tr>
<td>$A_2A_2$</td>
<td>$x_{12}x_{22}$</td>
<td>0 $1/2$ $1/2$</td>
</tr>
<tr>
<td>$A_2A_2 × A_1A_1$</td>
<td>$x_{22}x_{11}$</td>
<td>0 1 0</td>
</tr>
<tr>
<td>$A_1A_2$</td>
<td>$x_{22}x_{12}$</td>
<td>0 $1/2$ $1/2$</td>
</tr>
<tr>
<td>$A_2A_2$</td>
<td>$x_{22}^2$</td>
<td>0 0 1</td>
</tr>
</tbody>
</table>

$p_1 = 2(50)/200 = 0.5$, $p_2 = (2(25) + 50)/200 = 0.5$.

Although to be honest, we won’t see mating tables again after the first couple weeks of the semester.
Notice that I’ve distinguished matings by both maternal and paternal genotype. While it’s not necessary for this example, we will see examples later in the course where it’s important to distinguish a mating in which the female is $A_1A_1$ and the male is $A_1A_2$ from ones in which the female is $A_1A_2$ and the male is $A_1A_1$. You are also likely to be surprised to learn that just in writing this table we’ve already made three assumptions about the transmission of genetic variation from one generation to the next:

**Assumption #1** Genotype frequencies are the same in males and females, e.g., $x_{11}$ is the frequency of the $A_1A_1$ genotype in both males and females.\(^5\)

**Assumption #2** Genotypes mate at random *with respect to their genotype at this particular locus.*

**Assumption #3** Meiosis is fair. More specifically, we assume that there is no segregation distortion; no gamete competition; no differences in the developmental ability of eggs, or the fertilization ability of sperm.\(^6\) It may come as a surprise to you, but there are alleles at some loci in some organisms that subvert the Mendelian rules, e.g., the $t$ allele in house mice, segregation distorter in *Drosophila melanogaster,* and spore killer in *Neurospora crassa.* If you’re interested, a pair of papers describing work in *Neurospora* appeared in 2012 [3, 4].

Now that we have this table we can use it to calculate the frequency of each genotype in newly formed zygotes in the population,\(^7\) provided that we’re willing to make three additional assumptions:

**Assumption #4** There is no input of new genetic material, i.e., gametes are produced without mutation, and all offspring are produced from the union of gametes within this population, i.e., no migration from outside the population.

**Assumption #5** The population is of infinite size so that the actual frequency of matings is equal to their expected frequency and the actual frequency of offspring from each mating is equal to the Mendelian expectations.

**Assumption #6** All matings produce the same number of offspring, on average.

\(^{5}\)It would be easy enough to relax this assumption, but it makes the algebra more complicated without providing any new insight, so we won’t bother with relaxing it unless someone asks.

\(^{6}\)We are also assuming that we’re looking at offspring genotypes at the zygote stage, so that there hasn’t been any opportunity for differential survival.

\(^{7}\)Not just the offspring from these matings.
Taking these three assumptions together allows us to conclude that the frequency of a particular genotype in the pool of newly formed zygotes is
\[ \sum \text{(frequency of mating)} \times \text{(frequency of genotype produce from mating)}. \]

So

\[
\begin{align*}
\text{freq.}(A_1A_1 \text{ in zygotes}) &= x_{11}^2 + \frac{1}{2}x_{11}x_{12} + \frac{1}{2}x_{12}x_{11} + \frac{1}{4}x_{12}^2 \\
&= x_{11}^2 + x_{11}x_{12} + \frac{1}{4}x_{12}^2 \\
&= (x_{11} + x_{12}/2)^2 \\
&= p^2 \\
\text{freq.}(A_1A_2 \text{ in zygotes}) &= 2pq \\
\text{freq.}(A_2A_2 \text{ in zygotes}) &= q^2
\end{align*}
\]

Those frequencies probably look pretty familiar to you. They are, of course, the familiar Hardy-Weinberg proportions. But we're not done yet. In order to say that these proportions will also be the genotype proportions of adults in the progeny generation, we have to make two more assumptions:

**Assumption #7** Generations do not overlap.\(^8\)

**Assumption #8** There are no differences among genotypes in the probability of survival.

### The Hardy-Weinberg principle

After a single generation in which all eight of the above assumptions are satisfied

\[
\begin{align*}
\text{freq.}(A_1A_1 \text{ in zygotes}) &= p^2 \\
\text{freq.}(A_1A_2 \text{ in zygotes}) &= 2pq \\
\text{freq.}(A_2A_2 \text{ in zygotes}) &= q^2
\end{align*}
\]

It’s vital to understand the logic here.

\(^8\)Or the allele frequency is the same in generations that do overlap.
1. If Assumptions #1–#8 are true, then equations 1–3 must be true.

2. If genotypes are not in Hardy-Weinberg proportions, one or more of Assumptions #1–#8 must be false.

3. If genotypes are in Hardy-Weinberg proportions, one or more of Assumptions #1–#8 may still be violated.

4. Assumptions #1–#8 are sufficient for Hardy-Weinberg to hold, but they are not necessary for Hardy-Weinberg to hold.

Point (2) is why the Hardy-Weinberg principle is so important. There isn’t a population of any organism anywhere in the world that satisfies all 8 assumptions, even for a single generation. But all possible evolutionary forces within populations cause a violation of at least one of these assumptions. Departures from Hardy-Weinberg are one way in which we can detect those forces and estimate their magnitude.

Estimating allele frequencies

Before we can determine whether genotypes in a population are in Hardy-Weinberg proportions, we need to be able to estimate the frequency of both genotypes and alleles. This is easy when you can identify all of the alleles within genotypes, but suppose that we’re trying to estimate allele frequencies in the ABO blood group system in humans. Then we have a situation that looks like this:

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>A</th>
<th>AB</th>
<th>B</th>
<th>O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotype(s)</td>
<td>aa</td>
<td>ao</td>
<td>ab</td>
<td>bb</td>
</tr>
<tr>
<td>No. in sample</td>
<td>$N_A$</td>
<td>$N_{AB}$</td>
<td>$N_B$</td>
<td>$N_O$</td>
</tr>
</tbody>
</table>

Now we can’t directly count the number of $a$, $b$, and $o$ alleles. What do we do? Well, more than 50 years ago, some geneticists figured out how with a method they called “gene counting” [1] and that statisticians later generalized for a wide variety of purposes and called the EM algorithm [2]. It uses a trick you’ll see repeatedly through this course. When we don’t know something we want to know, we pretend that we know it and do some calculations.

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9There may be some that come reasonably close, but none that fulfill them exactly. There aren’t any populations of infinite size, for example.

10Actually, there’s a ninth assumption that I didn’t mention. Everything I said here depends on the assumption that the locus we’re dealing with is autosomal. We can talk about what happens with sex-linked loci, if you want. But again, mostly what we get is algebraic complications without a lot of new insight.
with what we just pretended to know. If we’re lucky, we can fiddle with our calculations a bit to relate the thing that we pretended to know to something we actually do know so we can figure out what we wanted to know. Make sense? Probably not. Let’s try an example and see if that helps.

If we knew $p_a$, $p_b$, and $p_o$, we could figure out how many individuals with the $A$ phenotype have the $aa$ genotype and how many have the $ao$ genotype, namely

\[
N_{aa} = n_A \left( \frac{p_a^2}{p_a^2 + 2p_ap_o} \right),
\]

\[
N_{ao} = n_A \left( \frac{2p_ap_o}{p_a^2 + 2p_ap_o} \right).
\]

Obviously we could do the same thing for the $B$ phenotype:

\[
N_{bb} = n_B \left( \frac{p_b^2}{p_b^2 + 2p_bp_o} \right),
\]

\[
N_{bo} = n_B \left( \frac{2p_bp_o}{p_b^2 + 2p_bp_o} \right).
\]

Notice that $N_{ab} = N_{AB}$ and $N_{oo} = N_O$ (lowercase subscripts refer to genotypes, uppercase to phenotypes). If we knew all this, then we could calculate $p_a$, $p_b$, and $p_o$ from

\[
p_a = \frac{2N_{aa} + N_{ao} + N_{ab}}{2N},
\]

\[
p_b = \frac{2N_{bb} + N_{bo} + N_{ab}}{2N},
\]

\[
p_o = \frac{2N_{oo} + N_{ao} + N_{bo}}{2N},
\]

where $N$ is the total sample size.

Surprisingly enough we can actually estimate the allele frequencies by using this trick. Just take a guess at the allele frequencies. Any guess will do. Then calculate $N_{aa}$, $N_{ao}$, $N_{bb}$, $N_{bo}$, $N_{ab}$, and $N_{oo}$ as described in the preceding paragraph. That’s the Expectation part the EM algorithm. Now take the values for $N_{aa}$, $N_{ao}$, $N_{bb}$, $N_{bo}$, $N_{ab}$, and $N_{oo}$ that you’ve calculated and use them to calculate new values for the allele frequencies. That’s the Maximization part of the EM algorithm. It’s called “maximization” because what you’re doing is calculating maximum-likelihood estimates of the allele frequencies, given the

\[\text{Chances are } N_{aa}, N_{ao}, N_{bb}, \text{ and } N_{bo} \text{ won’t be integers. That’s OK. Pretend that there really are fractional animals or plants in your sample and proceed.}\]
observed (and made up) genotype counts.\footnote{If you don’t know what maximum-likelihood estimates are, don’t worry. We’ll get to that in a moment.} Chances are your new values for $p_a$, $p_b$, and $p_o$ won’t match your initial guesses, but\footnote{Yes, truth \textit{is} sometimes stranger than fiction.} if you take these new values and start the process over and repeat the whole sequence several times, eventually the allele frequencies you get out at the end match those you started with. These are maximum-likelihood estimates of the allele frequencies.\footnote{I should point out that this method \textit{assumes} that genotypes are found in Hardy-Weinberg proportions.}

Consider the following example:

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>A</th>
<th>AB</th>
<th>AB</th>
<th>O</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. in sample</td>
<td>25</td>
<td>50</td>
<td>25</td>
<td>15</td>
</tr>
</tbody>
</table>

We’ll start with the guess that $p_a = 0.33$, $p_b = 0.33$, and $p_o = 0.34$. With that assumption we would calculate that $25(0.33^2/(0.33^2 + 2(0.33)(0.34))) = 8.168$ of the A phenotypes in the sample have genotype $aa$, and the remaining 16.832 have genotype $ao$. Similarly, we can calculate that 8.168 of the B phenotypes in the population sample have genotype $bb$, and the remaining 16.832 have genotype $bo$. Now that we have a guess about how many individuals of each genotype we have,\footnote{Since we’re making these genotype counts up, we can also pretend that it makes sense to have fractional numbers of genotypes.} we can calculate a new guess for the allele frequencies, namely $p_a = 0.362$, $p_b = 0.362$, and $p_o = 0.277$. By the time we’ve repeated this process four more times, the allele frequencies aren’t changing anymore, and the maximum likelihood estimate of the allele frequencies is $p_a = 0.372$, $p_b = 0.372$, and $p_o = 0.256$.

What is a maximum-likelihood estimate?

I just told you that the method I described produces “maximum-likelihood estimates” for the allele frequencies, but I haven’t told you what a maximum-likelihood estimate \textit{is}. The good news is that you’ve been using maximum-likelihood estimates for as long as you’ve been estimating anything, without even knowing it. Although it will take me a while to explain it, the idea is actually pretty simple.

Suppose we had a sock drawer with two colors of socks, red and green. And suppose we were interested in estimating the proportion of red socks in the drawer. One way of approaching the problem would be to mix the socks well, close our eyes, take one sock from the drawer, record its color and replace it. Suppose we do this $N$ times. We know that the number of red socks we’ll get might be different the next time, so the number of red socks we actually get is a random variable. Let’s call that random variable $K$. Now suppose in our actual experiment we find $k$ red socks, i.e., the value our random variable takes on is $k$. 
or putting it in an equation: \( K = k \). If we knew \( p \), the proportion of red socks in the drawer, we could calculate the probability of getting the data we observed, namely

\[
P(K = k|p) = \binom{N}{k} p^k (1 - p)^{(N-k)}.
\] (4)

This is the binomial probability distribution. The part on the left side of the equation is read as “The probability that we get \( k \) red socks in our sample given the value of \( p \).” The word “given” means that we’re calculating the probability of our data conditional on the (unknown) value \( p \).

Of course we don’t know \( p \), so what good does writing (4) do? Well, suppose we reverse the question to which equation (4) is an answer and call the expression in (4) the “likelihood of the data.” Suppose further that we find the value of \( p \) that makes the likelihood bigger than any other value we could pick.\(^\text{16}\) Then \( \hat{p} \) is the maximum-likelihood estimate of \( p \).\(^\text{17}\)

In the case of the ABO blood group that we just talked about, the likelihood is a bit more complicated

\[
\left( \frac{N}{N_A N_AB N_B N_O} \right) (p_a^2 + 2p_a p_o)^{N_A} 2p_a p_b^{N_AB} (p_b^2 + 2p_b p_o)^{N_B} (p_o^2)^{N_O}
\] (5)

This is a multinomial probability distribution. It turns out that one way to find the values of \( p_a, p_b, \text{and} p_o \) is to use the EM algorithm I just described.\(^\text{18}\) There isn’t a simple formula that allows us to write down an expression for the maximum-likelihood estimate of the allele frequencies in terms of the phenotype frequencies. We have to use an algorithm to find them, and the EM algorithm happens to be a particularly convenient algorithm to use.

### An introduction to Bayesian inference

Maximum-likelihood estimates have a lot of nice features, but they are also a slightly backwards way of looking at the world. The likelihood of the data is the probability of the data, \( x \), given parameters that we don’t know, \( \phi \), i.e., \( P(x|\phi) \). It seems a lot more natural to think about the probability that the unknown parameter takes on some value, given the data, i.e., \( P(\phi|x) \). Surprisingly, these two quantities are closely related. Bayes’ Theorem tells us that

\[
P(\phi|x) = \frac{P(x|\phi)P(\phi)}{P(x)}.
\] (6)

---

\(^{16}\)Technically, we treat \( P(K = k|p) \) as a function of \( p \), find the value of \( p \) that maximizes it, and call that value \( \hat{p} \).

\(^{17}\)You’ll be relieved to know that in this case, \( \hat{p} = k/N \).

\(^{18}\)There’s another way I’d be happy to describe if you’re interested, but it’s a lot more complicated.
We refer to $P(\phi|x)$ as the **posterior distribution** of $\phi$, i.e., the probability that $\phi$ takes on a particular value given the data we’ve observed, and to $P(\phi)$ as the **prior distribution** of $\phi$, i.e., the probability that $\phi$ takes on a particular value before we’ve looked at any data. Notice how the relationship in (6) mimics the logic we use to learn about the world in everyday life. We start with some prior beliefs, $P(\phi)$, and modify them on the basis of data or experience, $P(x|\phi)$, to reach a conclusion, $P(\phi|x)$. That’s the underlying logic of Bayesian inference.

### Estimating allele frequencies with two alleles

Let’s suppose we’ve collected data from a population of *Protea repens* and have found 7 alleles coding for the *fast* allele at a enzyme locus encoding glucose-phosphate isomerase in a sample of 20 alleles. We want to estimate the frequency of the *fast* allele. The maximum-likelihood estimate is $7/20 = 0.35$, which we got by finding the value of $p$ that maximizes

$$P(k|N,p) = \binom{N}{k} p^k (1 - p)^{N-k},$$

where $N = 20$ and $k = 7$. A Bayesian uses the same likelihood, but has to specify a prior distribution for $p$. If we didn’t know anything about the allele frequency at this locus in *P. repens* before starting the study, it makes sense to express that ignorance by choosing $P(p)$ to be a uniform random variable on the interval $[0, 1]$. That means we regarded all values of $p$ as equally likely prior to collecting the data.

Only a little less than 30 years ago it was necessary to do a bunch of complicated calculus to combine the prior with the likelihood to get a posterior. Since the early 1990s statisticians have used a simulation approach, Monte Carlo Markov Chain sampling, to construct numerical samples from the posterior. For the problems encountered in this course, we’ll mostly be using the freely available software package **JAGS** to implement Bayesian analyses. For the problem we just encountered, here’s the code that’s needed to get our results:

```r
model {

A few of you may recognize that I didn’t choose that species entirely at random, even though the “data” I’m presenting here are entirely fanciful.

If we had prior information about the likely values of $p$, we’d pick a different prior distribution to reflect our prior information. See the Summer Institute notes for more information, if you’re interested.

You are probably balking at my use of the word “only” in combination with the phrase “30 years.” Please cut me a little slack. I know that most of you weren’t born 30 years ago, but I’d already taught this course two or three times by the time the paper I’m about to refer to was published.

This code and other **JAGS** code used in the course can be found on the course web site by following the links associated with the corresponding lecture.

}
# likelihood
k ~ dbin(p, N)

# prior
p ~ dunif(0,1)

}

Running this in JAGS with k = 7 and n = 20 produces these results:

> source("binomial.R")
Compiling model graph
  Resolving undeclared variables
  Allocating nodes
  Graph Size: 5

Initializing model

|**************************************************| 100%
Inference for Bugs model at "binomial.txt", fit using jags,
  5 chains, each with 2000 iterations (first 1000 discarded)
n.sims = 5000 iterations saved
  mu.vect sd.vect  2.5%  25%  50%  75%  97.5% Rhat n.eff
  p       0.363  0.099  0.187  0.290  0.358  0.431  0.567  1.001  3800

For each parameter, n.eff is a crude measure of effective sample size,
  and Rhat is the potential scale reduction factor (at convergence, Rhat=1).

DIC info (using the rule, pD = var(deviance)/2)
pD = 0.8 and DIC = 5.1
DIC is an estimate of expected predictive error (lower deviance is better).
>

The column headings should be fairly self-explanatory, except for the one last two. mu.vect is the posterior mean. It’s our best guess of the value for the frequency of the

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23Kristen will show you how to run JAGS through R in lab.
fast allele. sd.vect is the posterior standard deviation. It’s our best guess of the uncertainty associated with our estimate of the frequency of the fast allele. The 2.5%, 50%, and 97.5% columns are the percentiles of the posterior distribution. The [2.5%, 97.5%] interval is the 95% credible interval, which is analogous to the 95% confidence interval in classical statistics, except that we can say that there’s a 95% chance that the frequency of the fast allele lies within this interval. Since the results are from a simulation, different runs will produce slightly different results. In this case, we have a posterior mean of about 0.36 (as opposed to the maximum-likelihood estimate of 0.35), and there is a 95% chance that \( p \) lies in the interval [0.19, 0.57].

**Returning to the ABO example**

Here’s data from the ABO blood group:

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>A</th>
<th>AB</th>
<th>B</th>
<th>O</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observed</td>
<td>862</td>
<td>131</td>
<td>365</td>
<td>702</td>
<td>2060</td>
</tr>
</tbody>
</table>

To estimate the underlying allele frequencies, \( p_A \), \( p_B \), and \( p_O \), we have to remember how the allele frequencies map to phenotype frequencies:

\[
\begin{align*}
\text{Freq}(A) &= p_A^2 + 2p_Ap_O \\
\text{Freq}(AB) &= 2p_Ap_B \\
\text{Freq}(B) &= p_B^2 + 2p_Bp_O \\
\text{Freq}(O) &= p_O^2 .
\end{align*}
\]

Hers’s the JAGS code we use to estimate the allele frequencies:

```jags
model {
    # likelihood
    pi[1] <- p.a*p.a + 2*p.a*p.o
    pi[2] <- 2*p.a*p.b
    x[1:4] ~ dmulti(pi[],n)
}
```

---

24If you don’t understand why that’s different from a standard confidence interval, ask me about it.
25This is almost the last time! I promise.
26Assuming genotypes are in Hardy-Weinberg proportions. We’ll relax that assumption later.
# priors
\[
\begin{align*}
  a_1 & \sim \text{dexp}(1) \\
  b_1 & \sim \text{dexp}(1) \\
  o_1 & \sim \text{dexp}(1) \\
  p.a & \leftarrow a_1/(a_1 + b_1 + o_1) \\
  p.b & \leftarrow b_1/(a_1 + b_1 + o_1) \\
  p.o & \leftarrow o_1/(a_1 + b_1 + o_1) \\
  n & \leftarrow \text{sum}(x[]) \\
\end{align*}
\]

The \texttt{dmulti()} is a multinomial probability, a simple generalization of the binomial probability to samples when there are more than two categories. The priors are some mumbo jumbo necessary to produce the rough equivalent of uniform \([0,1]\) priors with more than two alleles.\textsuperscript{27} \texttt{sum()} is a built-in function that saves me the trouble of calculating the sample size and ensures that the \(n\) in \texttt{dmulti()} is consistent with the individual sample components. The \(x=c()\) produces a vector of counts arranged in the same order as the frequencies in \(\pi[]\). Here are the results:

```r
> source("multinomial.R")
Compiling model graph
   Resolving undeclared variables
   Allocating nodes
   Graph Size: 20

Initializing model

|++++++++++++++++++++++++++++++++++++++++++++++++++| 100%
|**************************************************| 100%

Inference for Bugs model at "multinomial.txt", fit using jags, 5 chains, each with 2000 iterations (first 1000 discarded)
n.sims = 5000 iterations saved

\begin{tabular}{lcccccccccc}
 & mu.vect & sd.vect & 2.5\% & 25\% & 50\% & 75\% & 97.5\% & Rhat & n.eff \\
p.a & 0.282 & 0.008 & 0.266 & 0.276 & 0.282 & 0.287 & 0.297 & 1.001 & 5000 \\
p.b & 0.129 & 0.005 & 0.118 & 0.125 & 0.129 & 0.133 & 0.140 & 1.001 & 5000 \\
p.o & 0.589 & 0.008 & 0.573 & 0.584 & 0.589 & 0.595 & 0.606 & 1.001 & 5000 \\
deviance & 27.811 & 2.007 & 25.830 & 26.363 & 27.229 & 28.577 & 33.245 & 1.001 & 4400 \\
\end{tabular}

\textsuperscript{27}It produces a Dirichlet(1,1,1), if you really want to know.
For each parameter, n.eff is a crude measure of effective sample size, and Rhat is the potential scale reduction factor (at convergence, Rhat=1).

DIC info (using the rule, pD = var(deviance)/2)
pD = 2.0 and DIC = 29.8
DIC is an estimate of expected predictive error (lower deviance is better).

Notice that the posterior means are very close to the maximum-likelihood estimates, but that we also have 95% credible intervals so that we have an assessment of how reliable the Bayesian estimates are. Getting them from a likelihood analysis is possible, but it takes a fair amount of additional work.

References


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