Lecture 8: Introduction to hierarchical modeling part II

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As before, we need to load some packages and set some options prior to running any models:

```
library(rstan)
library(shinystan)
library(car)
rstan_options(auto_write = TRUE)
options(mc.cores = parallel::detectCores())
source("../utilityFunctions.R")
```

In the last class we started to incorporate priors who are themselves parameters to be estimated. In the simple and albeit contrived example, we learned about how we need to consider both joint and marginal effects on the posterior.

Now we can begin to put these models to effect for more realistic data.

Practical example of hierarchical models

Hierarchical models allow us to model not just individuals but populations through partial pooling.

- do this by relaxing *exchangability* (i.e., i.i.d) in the strict sense.
- We can permute any two individuals in the population, and the population still looks the same
 Not strictly iid but still pretty strong within population

Partial pooling shares information among populations, groups, or clusters with several benefits:

- 1. Improved estimates for repeat sampling: When more than one observation arises from same individual, location, or time, traditional single-level models either over- or underfit.
- 2. **Improved estimates for sampling imbalance:** When some individuals, locations, or times are sampled more than others, multilevel models implicitly deal with differing uncertainty across groups.
 - Prevents oversampled groups from dominating inference
- 3. **Estimating variation:** If our questions include variation among individuals or groups, multilevel models are the bee's knees because they explicitly model such variation.
- 4. **Avoid averaging, retain variation:** Often in traditional analyses groups are collapsed by pre-averaging to make variables. This is naughty because it discards variation, and because there are multiple ways to average.

As a motivating example, suppose we are interested in the infection prevalance of Ranavirus in Eastern newts (*Notophthalmus viridescens*)—the best animal ever!!!—in eastern Tennessee.

- Newts are censused across N = 60 ponds and scored as to whether they are infected or not.
- In some ponds, nary a newt is noticed because the density is low or the pond is hard to sample. Other ponds might be beside themselves with newts (the best ponds).
- Depending on the question, we might be interested in estimating the average infection rate among ponds.
 - We might also be interested in good estimates of within-pond infection rate if conservation efforts need to target the most infected ponds first.

To look at how hierarchical models work, I have simulated data (newtDat.csv) for this scenario so that we can check our estimates against the "true" values.

If you are interested in the simulation, here is the code:

```
omega <- 0.3 # population mode (avg. infection rate across sites)
kappa <- 20 # Concentration (how variable sites are)</pre>
nObs <- 60
             # total number of sites
a <- omega*(kappa-2) +1 # Beta shape parameter
b <- (1-omega)*(kappa-2) +1 # Beta shape parameter
set.seed(5) # for repeatability
# Total n. of newts sampled at a site. Sites have 5, 10, 25, or 40 newts
\mathbb{N} \leftarrow as.integer(rep(c(5, 10, 25, 40), each=15))
trueInfect <- rbeta(nObs, a, b)</pre>
                                     # true infection prevalance at a site
infect <- rbinom(nObs, N, trueInfect) # simulated number of infected newts</pre>
# Site indicator (not really necessary here but included for generality)
site <- 1:n0bs
newtDat <- data.frame(site=site, N=N, trueInfect=trueInfect,</pre>
                       infect=infect, propInf=infect/N)
newtDat <- read.csv("newtDat.csv")</pre>
```

```
site N trueInfect infect propInf
1
     1 5 0.2266825
                          1
                                0.2
2
     2 5 0.5166810
                          1
                                0.2
3
     3 5 0.1843030
                          1
                                0.2
                          2
4
     4 5 0.3283834
                                0.4
                          3
5
     5 5 0.5750651
                                0.6
     6 5
         0.2519853
                          2
                                0.4
```

head(newtDat)

We have repeated measures and heterogeneity. There is a lot of variation in the data across sites (because I simulated it thus) due to unmeasured factors affecting ranavirus prevalance. Thus site is a grouping variable with multiple observations (newts) within each site.

Ignoring sites by estimating one infection prevalence across all ponds means we ignore important variation and may underfit (complete pooling model).

Estimating an infection probability for each site separately (no pooling model) may introduce bias by overfitting noise and ignoring that sites may not be spatially independent of each other—newts migrate.

A partial pooling model compromises with an adaptive prior that learns from all site-level infection estimates.

Complete pooling

We are going to create all three models. We will start with the simplest, a complete pooling model that estimates one θ across all sites.

This model, completePooling.stan, is specified as:

$$infected \sim \text{Binomial}(N, \theta)$$

 $\theta \sim \text{Beta}(\alpha, \beta).$ (1)

where θ is the infection probability for all sites and α and β are constants. This should be simple to code in stan by now.

```
data {
  int<lower=0> nObs;
                           // No. obs.
                             // No. sampled newts
  int<lower=0> N[nObs];
  int<lower=0> obs[n0bs];  // No. infected newts
  real<lower=0> alpha;
                             // priors on theta
  real<lower=0> beta;
                             // priors on theta
}
parameters {
  real<lower=0, upper=1> theta; // grand infect prob.
}
model {
  theta ~ beta(alpha, beta); // prior for thetas
  obs ~ binomial(N, theta);
}
```

No pooling

The second model we will build is a no-pooling model that estimates separate $\theta's$ for each site without cross-talk among sites. This model will be called noPooling.stan where

$$infected \sim \text{Binomial}(N, \theta_i)$$

 $\theta_i \sim \text{Beta}(\alpha, \beta)$ (2)

• Each site $theta_i$ will be given a weak Beta(2, 2) prior.

```
int<lower=0> nObs;
                             // No. obs.
  int<lower=0> nSites;
                            // No. groupings (redundant here)
  int<lower=0> site[nSites]; // Indicator values for groupings
 int<lower=0> N[n0bs];
                         // No. sampled newts
  int<lower=0> obs[n0bs];
                             // No. infected newts
 real<lower=0> alpha;
                             // priors on thetas
                            // priors on thetas
 real<lower=0> beta;
}
parameters {
 vector<lower=0, upper=1>[nSites] theta; // w/in-site infect prob.
}
model {
 theta ~ beta(alpha, beta);
                                 // prior for thetas
 // lik. (loop not necessary here but used to show indexing)
 for(n in 1:n0bs)
 obs[n] ~ binomial(N[n], theta[site[n]]);
}
```

Partial pooling

Finally, we will build a partial pooling model, partialPooling.stan, as specified below:

$$infected \sim \text{Binomial}(N, \theta_i)$$

$$\theta_i \sim \text{Beta}(a, b)$$

$$a = \omega(\kappa - 2) + 1$$

$$b = (1 - \omega)(\kappa - 2) + 1$$

$$\omega \sim \text{Beta}(\alpha_\omega, \beta_\omega)$$

$$\kappa \sim \text{Normal}^+(2, \sigma_\kappa)$$
(3)

where θ_i is the infection probability at the *i*th site and α_{ω} , β_{ω} , and σ_{κ} are constants.

- Here ω will be given a weak Beta(2, 2) prior
- Because κ must be positive and > 2, it has a folded normal distribution and will be given a regularizing prior of $\sigma = 2.5$.

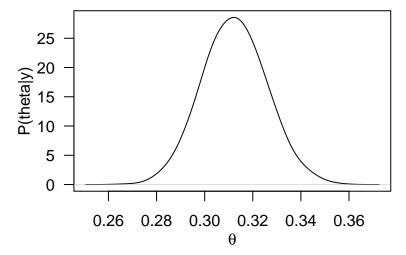
```
data {
  int<lower=0> nObs;
                             // No. obs.
                            // No. groupings (redundant here)
  int<lower=0> nSites;
  int<lower=0> site[nSites]; // Indicator values for groupings
  int<lower=0> N[nObs];
                             // No. sampled newts
 int<lower=0> obs[n0bs]; // No. infected newts
 real<lower=0> alpha;
                            // priors on Omega
 real<lower=0> beta;
                            // priors on Omega
 real<lower=0> sigma;
                          // prior on kappa scale
}
parameters {
 real<lower=0, upper=1> omega; // avg. amg-site infect prob.
 real<lower=2> kappa;
                                   // similarity of sites
 vector<lower=0, upper=1>[nSites] theta; // w/in-site infect prob.
}
transformed parameters {
 real<lower=0> a;
 real<lower=0> b;
   a = omega * (kappa - 2) +1;
   b = (1 - omega) * (kappa - 2) + 1;
}
model {
  omega ~ beta(alpha, beta);
                                         // prior on omega
 kappa ~ normal(2, sigma);
                                          // prior on kappa
 theta ~ beta(a,b);
                                          // prior for thetas
 // lik. (loop not necessary here but used to show indexing)
 for(n in 1:n0bs)
 obs[n] ~ binomial(N[n], theta[site[n]]);
}
```

Lets set up the data and look at the simplest model first:

```
# set up the data
nObs <- nrow(newtDat)
nSites <- max(newtDat$site)
     <- newtDat$site</pre>
site
N
       <- newtDat$N
      <- newtDat$infect</pre>
obs
                              # weak beta
alpha <- 2
     <- 2
                              # priors
beta
sigma <- 2.5
                              # regularizing scale prior
dat <- list(nObs=nObs, nSites=nSites, site=site, N=N, obs=obs,
```

If we plot out the posterior distribution of θ , we see that the median infection rate across sites is 0.31.

```
plot(density(thetaCP, adj=2), main="", xlab="", ylab="",las=1)
mtext(text = expression(paste(theta)), side=1, line = 2)
mtext(text = "P(theta|y)", side=2, line = 2.1)
```

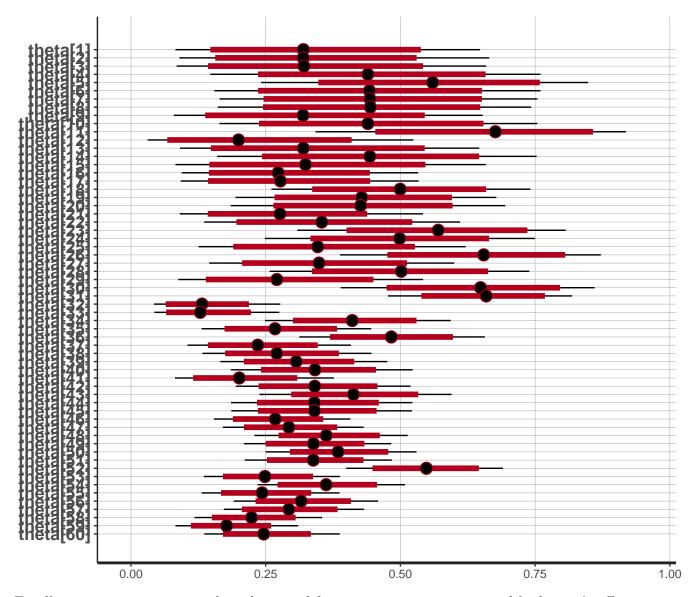


However, this ignores among-site heterogeniety.

Next let's look at the no-pooling model:

A plot of the θ 's reveals there is substantial variation among sites, with more uncertainty concentrated in sites with fewer observations (top to bottom):

```
plot(modNP, pars="theta")
```



Finally we can run our partial pooling model partialPooling.stan and look at it's effects.

To show the impact of partial pooling, we will first plot the empirical proportion of newts infected and then overlay the partial-pooling medians.

```
# Display no-pooling estimates of infection for each pond
plot(newtDat$propInf, col="blue", pch=16, xaxt="n", las=1, xlab="", ylab="")
axis(1, at=c(1, 15, 30, 45, 60))
mtext(text = "site", side=1, line = 2, cex=1)
```

```
mtext("p(infection)", side=2, line=2.2, cex=1)
# Draw vertical dividers between site sample sizes
abline(v=c(15.5, 30.5, 45.5))
text(8, 0, "N=5")
text(22.5, 0, "N=10")
text(37.5, 0, "N=25")
text(53, 0, "N=40")
points(medianPP)
abline(h=median(omega), lty=3, lwd=2)
for(i in 1:length(medianNP)) {
  arrows(x0=i, x1=i, y0=newtDat$propInf[i], y1=medianPP[i], length = 0.05)
}
  8.0
  0.6
p(infection)
0
7.
                                                   8
  0.2
  0.0
                 N=5
                                   N = 10
                                                     N=25
                                                                         N = 40
          1
                           15
                                             30
                                                                45
                                                                                  60
                                             site
```

Here the filled blue points represent the raw infection proportions and the black circles indicate the posterior θ medians. The horizontal line indicates the median of ω , the estimated median infection rate across east Tennessee.

Notice that the estimated $\theta's$ are closer to the dashed line than the raw estimates. This is called *shrinkage*.

- Because of information sharing, the estimates will be pulled towards the overall mean.
- Estimates from sites with less information (smaller N's) are shrunk more. Also, the greater the distance from the overall mean, the more shrinkage wil happen.

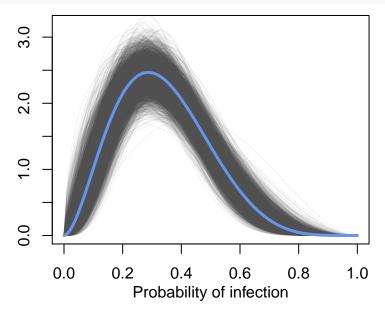
We can also look at what the inferred infection proportions for East TN as a whole should look like:

```
# calculate a & b for each posterior iteration and turn them into a matrix
a <- omega * (kappa - 2) + 1
b <- (1-omega) * (kappa - 2) + 1
propTN <- cbind(a,b)

plot(0:1,0:1, type="n", ylim=c(0,3.2)) # plot empty plot
mtext(text = "Probability of infection", side=1, line = 2, cex=1)

# Overlay infection probability densities for each posterior draw
for (n in 1:nrow(propTN)) {
    curve(dbeta(x, propTN[n,1], propTN[n,2]), add=TRUE, col="#50505010")
}

# Overlay median posterior probability density
medianTN <- apply(propTN,2,median)
curve(dbeta(x,medianTN[1], medianTN[2]), add=TRUE, col="cornflowerblue", lwd=3)</pre>
```



Note that these are different from the densities of ω (can plot as line if you want).

- The density of ω only encompasses uncertainty in the estimation of the posterior mode.
- Estimation of the infection probability for all of TN requires uncertainty about both the mode and the variation among sites, κ .
 - All of the uncertainty is then propagated together.

Bias vs. variance

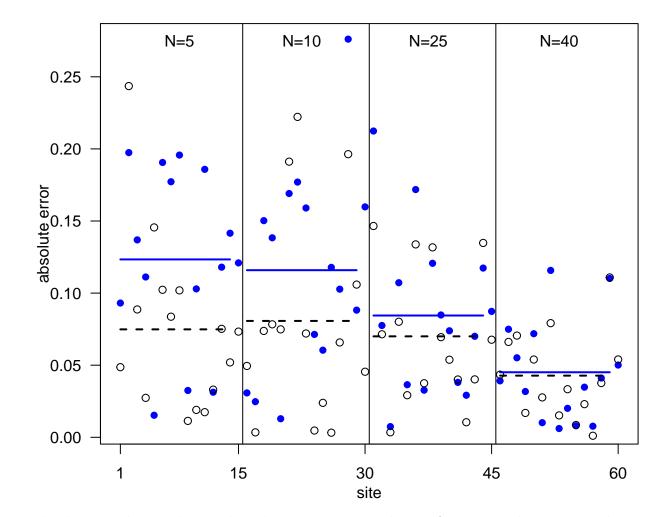
Because the complete pooling approach ignores among-group heterogeneity, it ignores information and tends to underfit.

Conversely, the no-pooling model can result in individual estimates that are imprecise because very little information goes into each estimate of θ —especially for smaller ponds.

Because I simulated the data, we have estimates of the "true" population infection probabilities 'trueInfect'. Therefore we can see how the partial pooling model compromises between over- and underfitting.

We can calculate an estimate of absolute error by taking the absolute value of the difference between each median estimate and the "true" value: $|\theta_{est} - \theta_{true}|$.

```
# calculate the error for no-pooling and partial pooling models
errorNP <- abs(medianNP - newtDat$trueInfect)</pre>
errorPP <- abs(medianPP - newtDat$trueInfect)</pre>
# calculate the average error for each sample size
avgErrNP <- aggregate(errorNP~N, FUN=mean)$errorNP</pre>
avgErrPP <- aggregate(errorPP~N, FUN=mean)$errorPP
# plot the no pooling error in blue
plot(errorNP, col="blue", pch=16, xaxt="n", las=1, xlab="", ylab="")
axis(1, at=c(1, 15, 30, 45, 60))
mtext(text = "site", side=1, line = 2, cex=1)
mtext("absolute error", side=2, line=2.5, cex=1)
# denote the different sample sizes
abline(v=c(15.5, 30.5, 45.5))
text(8, 0.275, "N=5")
text(22.5, 0.275, "N=10")
text(37.5, 0.275, "N=25")
text(53, 0.275, "N=40")
points(errorPP) # plot the partial pooling error estimates
# plot the average error
segments(x0=c(1, 16, 31, 46), x1=c(14, 29, 44, 59), y0=avgErrPP,
         y1=avgErrPP, lty=2, lwd=2)
segments(x0=c(1,16,31,46), x1=c(14, 29, 44, 59), y0=avgErrNP,
         y1=avgErrNP, lty=1, lwd=2, col="blue")
```



First, both no-pooling and partial-pooling estimates are better for sites with more samples. More data means more information and better estimation.

Second, note that the dashed line is always lower than the blue lines. This means the partial pooling models always do a better job (lower error than the no pooling models).

Overall, information sharing means better estimation for sites with less information by borrowing from sites with more information. Sites with more information need less correction and so pooling doesn't affect them very much.

However, partial pooling models are not panaceas, and there may be times when no-pooling is better. We are assuming, after all, that groups belong to a large population which may or may not be the case.