Clusters: fish, type of treatment

Treatments: a, b, l, d

First level: outcome variable = p (proportion), parameters = a\_fish[].

Second level: outcome variable = vector of intercept parameters, a\_fish[]. Parameters = alpha and sigma, and they have their own priors (normal and halfCauchy). These two parameters = hyperparameters.

So our first varying effects model, where fish are allowed to vary by intercept, would have 23 + 2 parameters: one for each fish, one overall sample intercept alpha, and one for the standard deviation among individuals.

Benefits of multilevel approach: each fish provides information that can be used to improve the estimates for all of the other fish. Each fish helps in this way, because we made an assumption about how the varying log-odds of each fish related to all of the others. We assumed a distribution, the beta distribution in this case. Once we have a distributional assumption, we can use Bayes’ theorem to optimally share information among the clusters (fish).

From there, we can visualize the inferred population distribution of the proportion of time spent positively rheotactic under each treatment. This is done in

Regular regression approach (frequentist): we have measurements for each fish (23 fish) under each condition (1 measurement per fish). They’re organized by fish and treatment. The prediction about whether treatment affects positive rheotaxis is going to be We can lump all the fish together and run a regression over all that data at the same time and say we have an average positive rheotaxis value for all of them, and that average is maybe higher based on whether or not it was measured in a velocity condition or not, but that’s all we can tell you.

But let’s suppose that because green sturgeon are highly variable, we’re interested more in what is the probability of positive rheotaxis under a certain treatment, given that each individual fish is highly variable in its response? How do we separate the effect of the individual from the effect of treatment? There are fish-level effects, and treatment-level effects. We have multiple levels.

The answer is that you construct a multilevel model and allow the slope of the treatment effect to vary by individual fish.

Two variables: what fish are we trying to make a measurement for, and is this fish in treatment A, B, L, or D? We have 4 measurements for this fish: one at A, one at B, one at L, and one at D. Each treatment is allowed to have its own slope. Each treatment raises the prediction of proportion of positive rheotaxis by some set amount, and that set amount is determined by the slope of that line.

Now if you can picture it, we have 23 scatterplots; all with 4 diagonal lines, which are indicating how much treatment affects proportion positive rheotaxis.

What the multilevel model does is say: we don’t think that treatment A in fish 20 has any more or less effect than treatment A has on fish #22. So the slope for treatment A is going to be the same for all the fish. Same for the slope of treatment B, the slope of treatment L, etc. So then the statistical noise that you might get in the response variable for an individual fish can be smoothed over by our measurements of all the other fish that help us understand what the difference that each treatment makes for each fish, because the intercepts are allowed to vary for each fish. So fish #22 might have a higher intercept than fish #21 (meaning that fish #22 displays a higher “baseline” positive rheotaxis when there is no treatment present), but the effect

So when we’re fitting all of these slopes, any one of them is allowed to stray toward the extremes, but the model will pull those extremes closer to the average intercept across all the fish. So for that fish that we measured twice in experiment 1, it’s helping to inform the measurements of the fish for which we have less data. Alternatively, we have one fish that displayed zero positive rheotaxis, an extreme value. We can use partial pooling to regularize that value, without having to exclude the data from that fish (whose other 3 measurements closer to the mean).

Modeling the data gives us a more nuanced way to represent the data than null hypothesis testing. We’re not saying “we reject the null hypothesis that flow has no effect on positive rheotaxis.” Null hypothesis testing, in this case, would lead us to say “we fail to reject the null hypothesis that visual stimuli has no effect on positive rheotaxis.” But we can see from the response relative to the other treatments that it’s more nuanced than that; it’s not that visual stimuli has NO effect, it’s that it has little effect relative to the other treatments. Constructing a model allows us to more genuinely and accurately describe the data-generating process we observed in these experiments. We’re saying this is the probability of positive rheotaxis, conditional on the data.

Frequentist methods would preclude from the get-go what we actually observe: that the response variable is bimodal. Bayesian approach allows for these sorts of odd distributions as a matter of turn; we don’t wish to constrain the possibilities at the outset.