Replication Project

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### **Introduction:**

Over the past decade in the game of baseball has undergone a player development revolution. This movement expands the audience of econometric principled practices to now serve players. Driveline is a principal example of this. The Washington State based company has risen to prominence throughout amateur, collegiate, and professional ranks despite lacking any official affiliation with MLB or any of its teams. It has done this by serving the players. By using expanded data capturing technology, Driveline (and now most MLB teams) are capable of measuring a player's growth and training practices over time to optimize their development and careers. A focus upon biomechanics and player health is critical to optimize this player development. Driveline itself truly got its start by rehabbing discarded professional pitchers with serious arm injuries like Tommy John's surgery. Because of this, it has become an economic priority of baseball organizations and players to understand pitching injuries (Lindbergh & Sawchik, 2019).

A long suspected leading factor in pitching injuries is workload management and fatigue.

One of the best ways to study fatigue effects is through relief pitchers in major league baseball.

Unlike starting pitchers, relievers often have irregular and abbreviated pitching outings with consequently irregular and abbreviated recovery periods.

Kyle Burris and Jacob Coleman explore these fatigue effects within the MLB relief pitcher population in their 2018 paper *Out of Gas: Quantifying Fatigue in MLB Relievers*. They set out to determine the effect of fatigue and prior workloads upon a relief pitcher's fastball velocity. Sampling the fastest pitch from the pitching arsenal of each reliever who threw 200 pitches in at least one of the 2015, 2016, or 2017 seasons. They constrained this data to outings with fewer than 10 days of rest. Using ideas and equations from Pharmacology and Toxicology,

Burris and Coleman treat pitch counts from prior relief outings as a dosage of a fatigue inducing toxin which has a decreasing concentration each additional day since its administration. Each player therefore ought to have a unique dosage and concentration effect of this fatigue inducing toxin. The way that they conducted this analysis was through Bayesian Inference that incorporated their toxicology equations for volume-dose model, a pharmacokinetic model of estimating concentration, and a dose-response model. Using loose priors as a basis for their Bayesian Inference, Burris and Coleman found posterior distributions of parameters associated for each toxicology equation. First, they found that dosage model indicated that higher pitch counts presented increasing effects of fatigue inducement that they modeled as a piecewise (linear spline) function corresponding to varying quantiles of pitch counts. They found that the posterior distribution of the population elimination effect was normally distributed with a center at an estimated recovery rate of 55% per day. This suggests that each day of rest eliminates roughly 55% of the current concentration of toxin in their system on that day. Finally, they found that each pitcher had varying degrees of dosage response. Burris and Coleman found that pitchers like Neftali Feliz, Jose Ramirez, and Tommy Hunter were most affected by fatigue while pitchers like Bryan Shaw, Corey Knebel, and Jim Johnson were least affected by fatigue.

Bayesian Inference is at the heart of Burris and Coleman's research. When attempting to replicate this paper I found that conducting a Bayesian Inference model through the R2Jags and R2winBUGS packages in R presented far too steep of a learning curve for me to accomplish in the limited time frame of our replication project. As a result, I chose to replicate as much of their process as possible through a Frequentist modeling discipline.

Bayesians and Frequentists represent the two main disciplines of statistical modeling and inference. Generally, Bayesians begin with an initial hypothesis upon the true values of study for

a population. Each sample they study afterwards is then tied as a probability of the prior hypothesis. This allows a Bayesian statistician to repeatedly sample with each ensuing sample returning a posterior distributional result that was built as a probabilistic reference to the previous distributional result and so on which is ultimately based upon the initial prior hypothesis of distribution. On the other hand, Frequentists follow a more traditional approach to statistical analysis where they gauge a hypothesis based upon the findings from one sample of data. The main distinction is that Bayesians operate on prior hypothesis to make their conclusions while a frequentist does not (Raleigh & Salt Lake City R Users Group, 2018). Because the Bayesian replication approach proved too complex to replicate in R, I have provided a similar Frequentist approach to the project. This is the main distinction between my project and the one that Burris and Coleman conducted.

In addition, I have pushed their research a step further by adding a Quantile Regression upon the effect that average pitcher velocity has upon fatigue concentration effects of velocity loss. I wanted to explore whether or not pitchers who throw harder are more susceptible to velocity decreases due to fatigue than pitchers who throw at lower velocity.

### Data:

In their paper Kyle Burris and Jacob Coleman obtained all of their data from MLB's Pitch f/x database. They were able to directly scrape pitch by pitch data using the pitchRx package in R, which has been succeeded by the Statcast supported baseballr package. Unfortunately, when MLB developed Statcast and baseballsavant in recent years, the pitchRx package became defunct and the Pitch f/x database was fused into Statcast and baseball savant. When replicating this project, I therefore was forced to use the baseballr package. The main issue with this, is that sample timeline of 2015-2017 is right around the time that TrackMan cemented itself as the analytical backbone of MLBs Statcast system. The effect of this was that current Statcast records seem to have certain dissimilarities to the original Pitch f/x data that Burris and Coleman used. I suspect that TrackMan ball tracking systems had a fair amount of missed data in the early stages of its system usage in certain MLB ball parks. The biggest issue that I have found is that my final sample included 223,097 total fastballs thrown, 24,024 unique relief outings, and 402 unique relief pitchers. Conversely, Burris and Coleman used a sample of 268,966 total fastballs, in 26,774 unique relief appearances, for 324 unique relief pitches. Therefore, my sample of study was different from the one that Burris and Coleman used in their paper. I am not certain as to why our samples were so different, but I do believe that differences between pitchRx-Pitch f/x data and baseballr-Statcast data, may be responsible for some of the variation.

The main variables of interest for study were derived from this original pitch by pitch data set. This included the release velocity, which was transformed to create the main dependent variable of study: change in velocity between season average and outing average. The player ID tag and player name variables were used as keys as well as the main random effect grouping

variable of study. The pitch type variable was used to filter the data to only include fastballs or the pitcher's fastest pitch. The game date variable was important to organize and group pitch by pitch data into outing averages.

### **Burris and Coleman's Methodology:**

In Burris and Coleman's paper, they used a series of toxicology formulas. It's important to understand each formula and parameter used. The first formula of interest is the volume-dose model which is a piecewise (linear spline) function:

$$\beta_{k} \sim Normal_{[0,\infty)}(0,10^{2}) \quad k = 1,2,3,4,5$$

$$d_{jt} = \begin{cases} \beta_{1}x_{jt} & x \leq 11 \\ 11\beta_{1} + \beta_{2}(x_{jt} - 11) & 11 < x \leq 15 \\ 11\beta_{1} + 4\beta_{2} + \beta_{3}(x_{jt} - 15) & 15 < x < 21 \\ 11\beta_{1} + 4\beta_{2} + 6\beta_{3} + \beta_{4}(x_{jt} - 21) & 21 < x \leq 27 \\ 11\beta_{1} + 4\beta_{2} + 6\beta_{3} + 6\beta_{4} + \beta_{5}(x_{jt} - 27) & 27 < x \end{cases}$$

In this equation, d expresses dosage for pitcher j on day t and is a product of workload  $x_{jt}$  which represents the number of pitches thrown in the outing. The ß coefficients are normally distributed positive parameter values. The spline is a product of pitch count knots produced at the 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, and 90<sup>th</sup> percentiles. Since my sample was a bit different, my knots for my linear spline came out to be at 10, 15, 20, and 27 pitches thrown.

In the Pharmacokinetic model of concentration estimate, Burris and Coleman used the following formulas:  $\phi \sim Gamma(0.1,0.1)$   $c(t)=d_0e^{-\phi t}$ 

$$c_{jt} = \sum_{k=1}^{\infty} d_{j(t-k)} e^{-\phi(t-k)}$$

Where phi is the elimination rate parameter that follows a Gamma distribution. K is a measure of how many days ago the dose was administered. C<sub>jt</sub> is a measure of the total toxin concentration present in player j on day t. This equation was incredibly difficult to translate to my Frequentist

modeling approach, because it is so dependent upon the elimination rate parameter phi, which must be estimated using the gamma distribution. Because of this, I simply used Burris and Coleman's findings for elimination rate parameter phi, in order to continue with my frequentist model. They found that phi had a normal distribution centered at 0.55. In other words, they found that a pitcher eliminated 55% of the toxin present in their body each day. In other words, toxin concentration present in the body each day was roughly 45% of the toxin concentration present in the body on the previous day. Therefore, the concentration function that I ended up using in my replication project was:  $c_{jt} = \sum_{k=1}^{\infty} d_{j(t-k)} 0.45^{(t-k)}$ 

The final model that Burris and Coleman used was the dose-response model. This model is predicated on normal distribution of the response variable  $v_{jt}$ . As such, the formula is:

$$1/\sigma^{2} \sim Gamma(0.1, 0.1)$$

$$v_{jt} \sim Normal(\mu_{jt}, \sigma^{2}/n_{jt})$$

$$\mu_{jt} = \alpha + m_{j}c_{jt}$$

Under this formula, mu represents the true average pitch velocity of pitcher j on day t. Variable  $n_{jt}$  represents the number of fastballs thrown by pitcher j on day t. Each pitcher has a unique slope m and a unique intercept alpha, that create the mu value from the pitcher's concentration level that day. In addition, Burris and Coleman fit their Bayesian inference with a number of diffuse priors.  $\alpha_j \sim \text{Normal}(\mu_\alpha, \tau_\alpha^2); \ \mu_\alpha \sim \text{Normal}(93, 3^2); \ \tau_\alpha \sim \text{HC}(0,1)$   $m_j \sim \text{Normal}_{\{0,\infty\}}(1, \tau_m^2); \ 1/\tau_m^2 \sim \text{Gamma}(10,1)$ 

Because I used a frequentist approach, there was no method to incorporate priors and specific distributions. I ignored this section because I could not incorporate it into my modeling approach. However, there are some interesting conclusions to draw from it. Burris and Coleman mostly used a collection of loosely fitted priors. The player's alpha coefficient for the dose

response equation is expected to follow a normal distribution around the mean value of all alpha coefficients and hold a standard deviation of tau alpha squared. That tau value is perhaps the loosest fitted parameter. The choice of a Half-Cauchy distribution beginning at 0 affords that all error terms should have no true mean value, and all terms should be positive values. This is because the Cauchy distribution, while it may appear similar to a normal bell curve distribution differs in that is has no true mean value. The boundaries of the Cauchy distribution do not grow closer to 0. Essentially this means that tau alpha will be a positive value that loosely may represent a half normal distribution, but there is no mean value that binds or deflates extreme values closer to 0 than they ought to be ("Cauchy distribution: Simple definition, PDF, uses," 2021). Additionally, when we look at mu alpha, we see that it is centered around 93 with a distribution of nine. This seems to represent the average velocity distribution of Burris and Coleman's pitching sample. This demonstrates that they used a prior assumption each pitcher velocity sample would follow a normal distribution of probability around the sample mean and standard deviation. While priors are merely hypothesis that can change through the process of Bayesian inference, I think this is an odd assumption. A pitcher's mean velocity should be tied to a distribution of their own true mean velocity and not a total sample mean velocity. Furthermore, there exists a wide spread of appearance numbers for each pitcher. Some pitchers had more appearances in a season than others. Therefore, it may not be responsible to assume that many pitchers threw enough pitches and appeared in enough games to achieve a normal distribution, even if we can assume that pitching velocity follows normal distribution in the first place.

Burris and Coleman also assume that pitcher specific velocity slope is positive, centered at 1, and normally distributed. Additionally, they assume that the standard deviation of that distribution is an inverse function of the gamma function.

Burris and Coleman then derived their posterior distribution through Gibbs Sampling with three Markov chains, ran for 10,000 iterations, after a burn-in of 1,000 iterations, and were thinned every 10 iterations. By using a Gibbs Sampling method, Burris and Coleman are placing added preference and predication on the sampling distributions of their priors. This is because the Gibbs sampling method iteratively calculates values based upon the probability of one parameter value given other parameter values. Because of this, it is important to know each parameter distributional probability. This process is achieved by their prior parameter assumptions as well as the sampler's initial 1,000 burn-in iterations (Ritvikmath, 2021). While the prior parameter assumptions we previously covered were all fairly loose in their probabilistic claims, they still fell under certain distributional classifications like Normal, Gamma, and Half-Cauchy. This means that they are limited in their ability to change based upon those distributional classifications. This can be a little bit concerning because there is not a lot of discussion in Burris and Coleman's paper as to why they assigned each distribution to each prior parameter.

Aside from splitting hairs upon prior distributions, I think there are a couple of limitations to Burris and Coleman's approach. The biggest flaw to their approach is that it does not seem to incorporate the potentially far more important factor of gameday pitch counts upon velocity loss effects. A fully rested reliever who is asked to throw 50+ pitches in an outing may achieve an average fastball velocity that is lower than if they had previously pitched once or several times in the past five days but were only asked to throw ten pitches in that outing. If anything, Burris and Coleman seemed to invert this dosage response effect because they included the number of gameday pitches as a denominator to the standard deviation of the Normal distribution prior of dose response: v<sub>it</sub>. That would effectively increase the probability of

achieving a velocity closer to the dose concentration predicted velocity as the number of pitches increased in a given outing. This doesn't make much sense. If anything, they should have defined pitches thrown on Day 0 as a part of the original concentration formula and weighted it above previous day pitch counts. Ultimately the number of pitches that are thrown in an outing ought to have a larger effect on a pitcher's velocity than the number of pitches the pitcher threw in previous days outings.

### My Methodology:

In order to navigate issues of coding the Bayesian Inference, I substituted Burris and Coleman's Bayesian approach for a Frequentist approach. This was done by using a series of linear mixed effects models. The dependent variable of interest in these models was velocity change in an outing from the pitcher's season average velocity. The random effects variables were the player IDs and player names from Statcast. The first linear mixed effects model was run to find the beta values of the piecewise function used to define pitching dosage for each of the previous 6 days. The fixed effect in this model was the number of pitches thrown on each of the previous 6 days. These beta values were found by the unique random effects intercepts and coefficients calculated by the linear mixed effects model. The beta values were then supplied to Burris and Coleman's previously specified piecewise function to calculate dosage from each previous day. Because my data sample ended up being different from Burris and Coleman, I resulted in slightly different knot values for my piecewise function as was previously discussed in the coverage of that function.

The next linear mixed effects model was used with the same dependent variable and random effects but now specified the dosage on each of the previous 6 days as the fixed effects.

The unique random effect coefficients and intercepts of the model were once again pulled and

supplied as player specific toxin concentration factors. These player specific toxin concentrations of their pitching day were then calculated using the elimination rate phi that Burris and Coleman found to be centered at 0.45. By multiplying that rate upon the dosages for each of the prior 6 days, the total toxin concentration within the body could be found.

The final linear mixed effects model used the same dependent variable and random effects but supplied that new concentration value as the fixed effect. The resulting coefficients and intercepts represented the player specific response magnitude to fatigue. The three linear mixed effects models are:

1.

$$\Delta Velo_{jt} = \mu_{js} - \mu_{jt}$$
 
$$\Delta Velo_{jt} = (n_{j_1} + n_{j_2} + n_{j_3} + n_{j_4} + n_{j_5} + n_{j_6} | PlayerIDs) + n_1 + n_2 + n_3 + n_4 + n_5 + n_6$$

2.

$$\beta_{jtk} = Coef(RANEF(Model1))$$

$$d_{jt} = \begin{cases} \beta_{jt1}x_{jt} & x \le 10 \\ 10\beta_{jt1} + \beta_{jt2}(x_{jt} - 10) & 10 < x \le 15 \\ 10\beta_{jt1} + 5\beta_{jt2} + \beta_{jt3}(x_{jt} - 15) & 15 < x < 20 \\ 10\beta_{jt1} + 5\beta_{jt2} + 5\beta_{jt3} + \beta_{jt4}(x_{jt} - 20) & 20 < x \le 27 \\ 10\beta_{jt1} + 5\beta_{jt2} + 5\beta_{jt3} + 7\beta_{jt4} + \beta_{jt5}(x_{jt} - 27) & 27 < x \end{cases}$$

$$\Delta Velo_{jt} = (d_{j_1} + d_{j_2} + d_{j_3} + d_{j_4} + d_{j_5} + d_{j_6} | PlayerIDs) + d_{j_1} + d_{j_2} + d_{j_3} + d_{j_4} + d_{j_5} + d_{j_6}$$

$$+ d_{j_7} + d_{j_6}$$

3.

 $CONFactors_j = Coef(RANEF(Model2) + Int(RANEF(Model2))$ 

$$c_{jt} = \sum_{k=1}^{\infty} CONFactors_{j(t-k)} 0.45^{(t-k)}$$

$$\Delta Velo_{jt} = (c_{jt}|PlayerIDs) + c_{jt}$$

In addition to supplying a frequentist approach to Burris and Coleman's modeling procedure, I wanted to further their study by measuring the effect that a pitcher's average fastball velocity had upon their fatigue base expected velocity lost. In essence, I wanted to study whether pitchers that threw harder were more susceptible to greater velocity losses than pitchers who may throw their fastballs at a slower velocity. In order to study this, I used a quantile regression, with quantiles set at 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, and 90<sup>th</sup> percentiles. The formula for this model followed this rudimentary equation:

 $Fatigue\_Concentration\_Velocity\_Effect_{Coefficient_j} = \alpha + Seaon\_Avg\_Velo_j$ 

Results:
Linear Mixed Effects Model 1 Output:

# Linear Mixed Effects Model 2 Output:

	Dependent variable:		Dependent variable:
	SeasonVeloDiff		SeasonVeloDiff
Pitches_Thrown_Day1	-0.011***	PitchingDosageDay1	-0.027***
	(0.001)		(0.003)
Pitches_Thrown_Day2	-0.003***	PitchingDosageDay2	-0.008***
	(0.001)		(0.002)
Pitches_Thrown_Day3	-0.0004 PitchingDosageDay		-0.002
	(0.001)		(0.001)
Pitches_Thrown_Day4	0.001	PitchingDosageDay4	-0.0003
	(0.001)		(0.001)
Pitches_Thrown_Day5	0.002***	PitchingDosageDay5	0.003**
	(0.001)		(0.001)
Pitches_Thrown_Day6	0.002***	PitchingDosageDay6	0.004***
	(0.001)		(0.001)
Constant	0.023**	Constant	0.033***
	(0.010)		(0.009)
Observations	24,024	Observations	24,024
Log Likelihood	-30,539.930	Log Likelihood	-30,532.130
Akaike Inf. Crit.	61,131.850	Akaike Inf. Crit.	61,116.260
Bayesian Inf. Crit.	61,342.110	Bayesian Inf. Crit.	61,326.520
Note:	*p<0.1; **p<0.05; ***p<0.01	Note:	*p<0.1; **p<0.05; ***p<0

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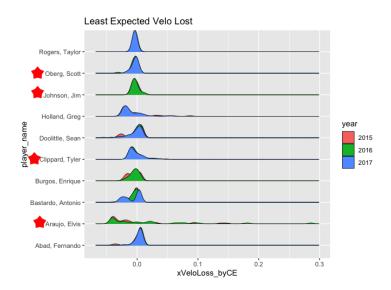
## Linear Mixed Effects Model 3 Output:

	Dependent variable:		
	SeasonVeloDiff		
concentration	-0.051***		
	(0.005)		
Constant	0.058***		
	(800.0)		
Observations	24,024		
Log Likelihood	-30,508.650		
Akaike Inf. Crit.	61,029.310		
Bayesian Inf. Crit.	61,077.830		
*	** ***		

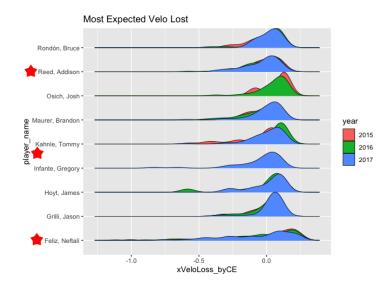
*Note:* \*p<0.1; \*\*p<0.05; \*\*\*p<0.01

Quantile Regression											
	25%		50%		75%		90%				
Variabl e	Pr(> t )	Coeff	Pr(> t	Coef ficie nt	Pr(> t	Coeff	Pr(> t	Coeff			
Interce pt Value	0.00	91.34	0.00	93.1 1160	0.00	94.43 916	0.00	96.10 842			
Season Averag e Velo	0.00	- 17.67 571	0.00	- 15.8 2610	0.00	- 22.57 044	0.00	- 21.17 793			

### Graph 1:



Graph 2:



\*Red stars mark pitchers that were correctly found in both Burris and Coleman's magnitude leaders, as well as our expected velo leaders.

\*\*Graph 2 is inverted, the pitchers with strongest negative velocity effect are at the bottom of the graph starting with Neftali Feliz

### **Discussion:**

Our results presented two main conclusions. Firstly, pitching exposure within the two previous days had the most negative effect upon a reliever's velocity. After those 2 previous days, it seems that there was not much statistical significance to suggest that pitchers were losing velocity. In fact, pitching exposure on days 5 and 6 was found to have statistically positive effects upon velocity. This suggests that relievers may regain most of their pitching stamina after at least 2-3 days of rest. This is interesting but also makes sense in comparison to the 5-6 day starting pitcher cycle that Burris and Coleman proposed. Starters need more time to recover stamina after outings, because they typically throw significantly more pitches than relievers would in those outings. Therefore, we would expect a relievers recovery timeline to be merely a fraction of the recovery time that a start requires.

Our project's second main conclusion can be drawn from the density plots. These present the true conclusions upon how are modeling process compared to that of Burris and Coleman. The red stars on the graph mark the pitchers that we correctly predicted on our leaderboards of most and least expected velocity lost. What I find interesting is that it really didn't matter how these stats ended up being factored, Neftali Feliz still lost the most velocity due to fatigue of any pitcher. In fact, he was the one pitcher that we correctly placed in the exact same rank as Burris and Coleman did in their leaderboards. However, this points to the main conclusion of our replication project which is that the Frequentist approach provides significantly different conclusions to Burris and Coleman's Bayesian approach. There are a number of pitchers who I found to be significant, (Taylor Rodgers, Greg Holland, Jason Grilli, and James Hoyt) that are nowhere to be found with Burris and Coleman's conclusions.

While I am unable to view the entirety of Burris and Coleman's rankings to discern how close my unique leaders were from being included by them, I am able to discern how close some of their unique leaders were to being included in my leaderboards. Bryan Shaw just missed the leaderboard as the reliever that was 12<sup>th</sup> least susceptible to fatigue. Corey Knebel missed the mark significantly, coming in at 339<sup>th</sup> and much closer to the bottom of the leaderboard than the top. Simon Castro was closer at 45<sup>th</sup>, while Brad Ziegler and Matt Bowman were quite close at 26<sup>th</sup> and 23<sup>rd</sup> respectively. On the other side, my model predicted Jose Ramirez to be far less susceptible to fatigue than Burris and Coleman found. He ranked 238<sup>th</sup> in most expected velocity lost to fatigue. Tommy Hunter was closer being ranked 45<sup>th</sup> in most expected velocity lost to fatigue. Hunter Strickland was next closer ranking 38<sup>th</sup>, Aaron Loup followed at 18<sup>th</sup>, and finally Scott Alexander, just missed the cut with the 10<sup>th</sup> most expect velocity lost to fatigue.

Based upon these results, it seems that my model had fairly similar conclusions to Burris and Coleman. However, the biggest outliers were clearly Jose Ramirez and Corey Knebel. With Corey Knebel, I believe that his calculation had something to do with his high number of outings with less than 3 days of rest. As our model output displays above, having less than 3 days of rest significantly negatively influenced a pitcher's expected velocity. Roughly 55% of all of Corey Knebel's outings in our study occurred with either 1 or 2 days of rest. Therefore, it makes sense that our model may have miscalculated him to be far more susceptible to fatigue than Burris and Coleman did.

On the other hand, Jose Ramirez had a far more balanced spread of days of rest. His error seems a bit more nuanced. Like Knebel, he was also featured in a relatively disproportionate number of games with less than 3 days of rest. However, unlike Knebel and perhaps unlike with Burris and Coleman concluded, Jose Ramirez had a positive coefficient for velocity effects in

games with 1 day of rest. This may not simply suggest that Ramirez had strong stamina in games with only one day of rest but rather may illustrate the flaws to our modeling procedure. Despite his positive coefficient, Jose Ramirez in fact has a very negative intercept for velocity effects in games with 1 day of rest. This suggests that going into these games with a lower concentration of fatigue toxin from prior games, Ramirez was expected to have lower velocity than if he had entered the game with a high concentration of fatigue toxin from prior games. This doesn't seem to make much sense though. It implies that in 1 day of rest outings, Ramirez pitched better when he had to throw a lot in the prior games than in the 1 day of rest outings where he threw very little in prior games. I think this flaw arises from the fact that the sample size of 1 day of rest outings for Jose Ramirez was rather small at 23 occurrences. This is not a significant enough sample size to make conclusions about the concentration effects for fatigue in these 1 day of rest games for Ramirez. However, the linear mixed effects modeling method doesn't concern itself with these small subsample sizes and runs its regressions anyways. Burris and Coleman circumvent this issue with their Markov Chain Monte Carlo Gibbs Sampling method that Bayesian analysis affords. They are able to simulate their sampling to increase their level of certainty in the results that they find. It seems that Frequentist modeling procedures are less successful when sample sizes and subsample sizes are small, whereas the Bayesian approach may excel in this sector of statistical analysis.

Finally, our quantile regression results provided some insight into how a pitcher's average velocity factors into his fatigue effects. Our table shows that typically faster velocity pitchers suffered from fatigue more, but the most resilient pitchers were not the slowest pitchers, but rather pitchers representing the 50<sup>th</sup> percentile of velocity. This seems to suggest that pitchers with particularly fast or slow velocity tended to suffer from fatigue more than average pitching

velocity counterparts. Since this does not support a hypothesis that high velocity fastballs are comparatively more fatiguing than lower velocity fastballs, it may rather demonstrate effects of different pitching styles. I believe very generally that uniquely fast or slow velocities are more likely to draw swings and misses than average pitching velocity. This is because with all other factors being equal a hitter is more adequately prepared to hit average pitching velocities than extremely fast or slow speeds. While many other factors go into pitch effectiveness and design, I believe that the quantile regression results may be demonstrating this theory. If a reliever has average velocity, hitters are more likely to put the ball in play which translates to quicker outs or quicker hits and runs. Since relievers are typically situationally called upon, they don't often have extended outings in which they are not pitching well. So, either way when they pitch to contact, they are likely to throw less pitches than their strikeout motivated counterparts. Because average velocity pitchers throw less pitches, they develop less fatigue inducing toxin concentrations and therefore see lower velocity deficits than their strikeout motivated counterparts.

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