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The Immediate Effects of Jumping on Adrenaline (with RCBD)

Introduction:

In the pursuit of understanding how acute physical stressors like jumping influence physiological responses, specifically, adrenaline release, this study explores the immediate effects of different durations of jumping at maximum intensity (for the subject) on the blood adrenaline levels among the residents of Providence Island. Adrenaline, a critical hormone in the body's stress response system, is commonly known as the "fight or flight" hormone. It is known to increase strength and pain tolerance, helping individuals react swiftly to immediate threats or challenges. However, adrenaline is not only released in the presence of danger as many other factors can cause the release of adrenaline such as exercise or even stress-related actions. Exercise, a common non-threatening stimulus, is known to trigger significant adrenaline release yet the extent and immediacy of this response remains less understood in comparison to the stimuli that cause the "fight or flight" response.

The choice of Providence Island as the study's location provides a unique demographic and geographical setting with twelve different villages differing in size and geography. This allows us to test each region rather than one over the other as certain regions may influence physical stress responses differently such as a rural environment in comparison to an urban environment. This aspect of the study may additionally provide an insight that displays how different environments factor into physiological reactions to exercise, thus the random sampling from each region is to prevent a bias in the difference of blood adrenaline level readings.

Previous studies have often focused on the adrenaline response to prolonged endurance activities or extreme physical exertions, providing less insight to shorter, intense bursts of physical activity such as jumping. This project seeks to investigate if the response is linear or if there is a threshold effect where the increase in adrenaline levels off or even decreases with increased activity duration, to provide further insight for shorter duration exercises which can be utilized with prolonged exercise findings to conclude certain findings on the human's response in blood adrenaline to physical activities. .

The project was motivated by the need to elucidate the physiological dynamics triggered by short-term intense physical activities and to quantify how these activities affect adrenaline levels. Additionally, we would like to understand to what extent does adrenaline increase along with the duration of physical exercise. Does the adrenaline level plateau or continually increase the longer a person performs a physical activity? The overarching goal was to document the average difference between initial and post-jumping blood adrenaline levels along with the percentage change in blood adrenalin after various fixed intervals of jumping, examining the nuances of this response across a controlled, varied-time framework.

Our general plan for the project entailed utilizing a Randomized Complete Block Design, ensuring effective and efficient blocking, thus minimizing the variability and enhancing the reliability of the results. This design was chosen to systematically assess the impact of jumping for 30, 60, 90, and 120 second intervals on blood adrenaline levels measured in pg/mL. By undertaking this study, we aimed not only to contribute to the scientific understanding of exercise-induced hormonal changes but also to provide insights that could influence physical training regimes and health recommendations, and our general understanding of stress and recovery in sports science.

Design of the Experiment

To answer our questions about the relationship between jumping as exercise and adrenaline production, we adopted a Randomized Complete Block Design (RCBD) into our experimental process. We specifically chose this design because it's a decently balanced and simple design that accommodates many kinds of experiments, including our own, where resources and time are limited and our results were predictably straightforward. In our case, each participant represents a block because we'll be measuring

the adrenaline before and after treatment, and then recording the change; participants are randomly selected of course, hence the RCBD.

In more detail, our experiment begins by first randomly selecting participants for the experiment. While a power test indicates we should use *12* participants, we decided to use a larger sample size to help mitigate variability; we predicted that this might be an issue given the block we chose. Each group member randomly selected 3 houses from each village to select the first available person willing to be a participant, but if no one in the house consented to participate, then we moved the next house up to repeat the process until eventually obtaining all the participants we needed (n = 3*9*4 = 108). Since our only block is produced from measuring adrenaline before and after treatment, there's nothing preventing participants of different ages and health conditions from participating, meaning that there may be confounding variables in our experiment. To reiterate, we're using a larger sample size than recommended by the power test because the large variety of participants may influence variability. However, we can still use things like age and gender as predictors in our model as a way to observe their effects.

After gathering all the participants, each group member was assigned a treatment level to give to the participants they were responsible for selecting, jumping for 30s, 60s, 90s, and 120s for a total of 4 levels. Each group member first measured each participant's adrenaline (pg/mL), applied the treatment, and measured the adrenaline for a final time to compare adrenaline levels before treatment and after for each participant; this was our response variable. After collecting this data from all participants, we analyzed the results in R and found the following.

Results and Interpretation:

We enter the data using Excel then process the data using R. We first model the analysis of variance(ANOVA) table with the response set as the change in blood adrenaline and the response predictors being jumping time(seconds), age(year), and gender.

```
##     Min      1Q  Median      3Q     Max
## -3311.3  -301.0   162.8   511.0   889.4
##
## Coefficients:
##                   Estimate Std. Error t value Pr(>|t|)
## (Intercept)       1397.295    215.318   6.489 2.98e-09 ***
## treatments_nofac    62.350      2.004  31.116  < 2e-16 ***
## age                  2.453      2.908   0.844    0.401
## genderM          -1800.411    134.825 -13.354  < 2e-16 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 689.7 on 104 degrees of freedom
## Multiple R-squared:  0.9262, Adjusted R-squared:  0.9241
## F-statistic:   435 on 3 and 104 DF,  p-value: < 2.2e-16
```

Fig 1. ANOVA Table of lm(response ~ treatments + age + gender)

In our analysis, the model coefficients significantly highlighted the influence of jumping time and gender on adrenaline levels. Specifically, as jumping time increases by 1 second, there is an increase of approximately 62.35 pg/mL in blood adrenaline, indicating a strong positive relationship between the duration of physical exertion and adrenaline secretion. Conversely, being male is associated with a substantial decrease of 1800.411 pg/mL in comparison to females, suggesting notable gender differences in physiological responses to exercise. Interestingly, the effect of age on adrenaline secretion was not statistically significant ($p = 0.401 > 0.05$), indicating that within the age range of our study population, age did not play a significant role in the change of blood adrenaline in response to jumping.

Additionally, we tested to see whether or not our model violates any statistical assumptions such as homoscedasticity(constant variance) or normality. To do so, we have created plots to illustrate the validity of our model.

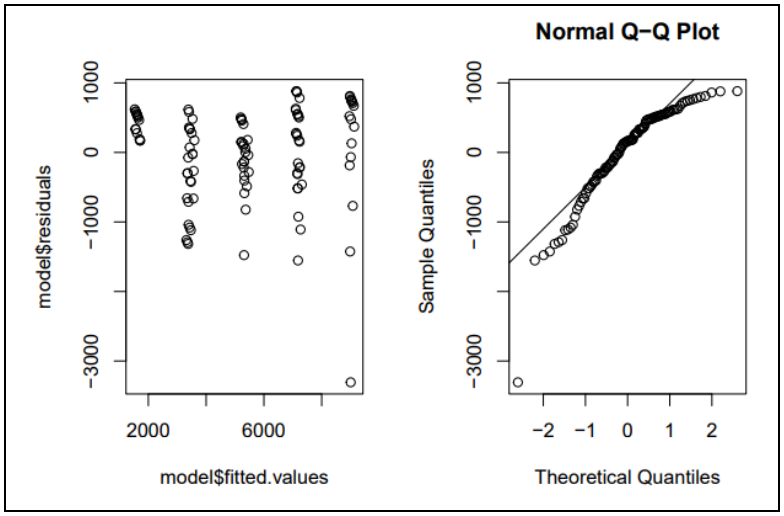

Fig 2. Plots for Model Adequacy(left to right: Residuals vs. Fitted and Normal Q-Q Plot)

As observed from the model adequacy plots in Figure 2, we can state that the plots support the key assumptions required for our linear regression model. The residuals vs. fitted plot shows a random spread of residuals, suggesting that the homoscedasticity assumption is met. Additionally, the Normal Q-Q PLot displays the residuals along the theoretical line with only slight deviations at the extremes indicating that our model does not violate the normality assumption. These observations affirm that our model does not violate the assumptions of constant variance and normality, ensuring the validity of our regression model analysis.

Additionally, we conducted two post-hoc tests utilizing the Tukey and Fisher LSD method. By observing Tukey's test, we can assess the significance of the differences between pairs of group means. Another method of viewing the differences between groups is to observe the Fisher LSD results which allows us to view the confidence intervals for all of the pairwise differences between group means while also controlling the individual error rate.

```
##
## $gender
##          diff    lwr.ci    upr.ci    pval
## M-F -857.1706 -1106.193 -608.1482 1.6e-08 ***
##
## $timejumped
##             diff    lwr.ci    upr.ci    pval
## 60-30   1027.8316  676.6282 1379.0349 5.1e-07 ***
## 90-30   1959.9597 1608.7564 2311.1630 2.1e-14 ***
## 120-30 2579.4469 2228.2435 2930.6502 < 2e-16 ***
## 90-60    932.1281  580.9248 1283.3315 3.2e-06 ***
## 120-60 1551.6153 1200.4120 1902.8187 2.5e-11 ***
## 120-90  619.4872  268.2838  970.6905 0.00093 ***
##
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

Fig 3. Fisher LSD Method

```
$gender
          diff       lwr       upr p adj
M-F -857.1706 -1106.193 -608.1482     0

$timejumped
             diff       lwr       upr     p adj
60-30   1027.8316  562.4340 1493.229 0.0000030
90-30   1959.9597 1494.5621 2425.357 0.0000000
120-30 2579.4469 2114.0493 3044.844 0.0000000
90-60    932.1281  466.7306 1397.526 0.0000184
120-60 1551.6153 1086.2178 2017.013 0.0000000
120-90  619.4872  154.0896 1084.885 0.0049537
```

Fig 4. Tukey HSD Method

As observed from Figure 3, Fisher's LSD test provides a straightforward approach to pairwise comparisons showing crucial information such as differences and p-values. It can be observed that all of the p-values are extremely small, indicating that all of the pairwise comparisons made are statistically significant. The greatest difference is between the duration of 120-30 (roughly 2800 pg/mL) and the smallest difference between the duration of 120-90(roughly 620 pg/mL). To further support these findings, Tukey's HSD test finds similar differences between these two time durations. Both the Tukey HSD and Fisher LSD tests provide compelling evidence that different durations of jumping significantly affect blood adrenaline levels with longer durations causing a bigger increase. It can also be noted that the

difference between 60-30(roughly 1030 pg/mL) and 120-90(roughly 620 pg/mL) decreases as the duration increases. This suggests that there is some sort of plateau in which an increased duration of jumping may not result in an increase of blood adrenaline relative to the previous jumping duration.

In assessing the physiological responses to different durations of physical activity, we utilize two types of visualizations to depict the impact of jumping time on the blood adrenaline levels. These visualizations assist in illustrating the relationships and distributions of adrenaline changes across varying exercise intensities.

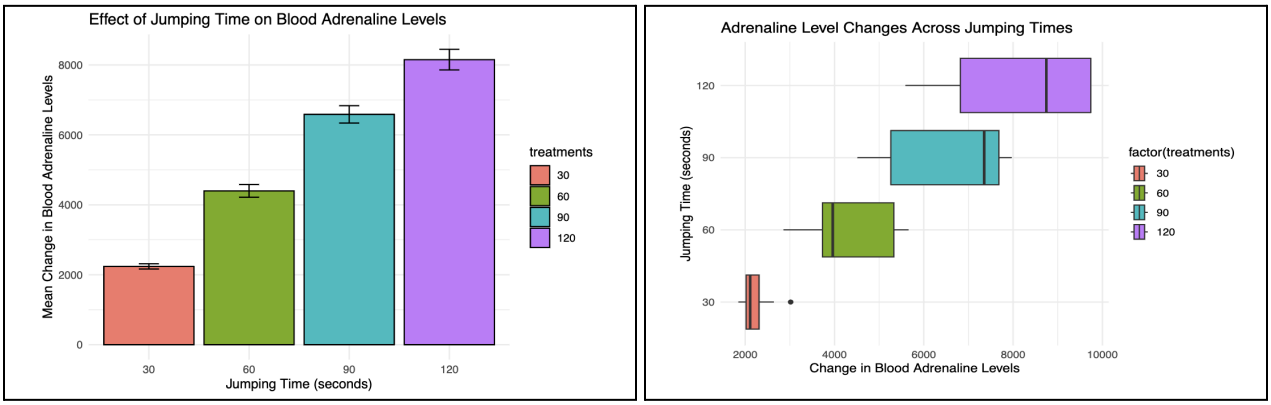

Figure 5. Bar Graph and Box-plot for Change in Blood Adrenaline Levels

The bar graph visualizes the mean change in blood adrenaline for four different jumping durations: 30, 60, 90, and 120 seconds. A clear response between increasing jumping time leading to progressively higher adrenaline levels can be observed. This suggests that longer durations of physical exertion are more effective in stimulating adrenaline secretion. Additionally, the error bars are relatively small, indicating that the mean values are reliably estimated. Accompanying the bar graph is the boxplot with details of the distribution of blood adrenaline changes for each specified jumping time. Similar to the bar graph, the median and IQR range for each category rises with increasing duration of exercise time, further supporting the relationship between jumping time and an increase in blood adrenaline levels. Another item to note is the outlier present in the 30 second jumping time level, indicating that factors such as baseline fitness or stress levels may also influence these outcomes. Together, these figures demonstrate that blood adrenaline levels significantly increase with longer durations of jumping and are helpful in understanding how different durations of exercise can be utilized to manipulate physiological responses for health improvements and/or athletic performance.

In our study, power analysis is a critical statistical method to determine the minimum sample size required to detect an effect of a given size with a predetermined level of confidence. This ensures that our experiment is adequately powered, meaning it has a sufficiently high probability of detecting statistically significant differences in blood adrenaline among the different levels of jumping time.

```
##      Balanced one-way analysis of variance power calculation
##
##               k = 4
##               n = 27
##               f = 8.706012
##       sig.level = 0.05
##           power = 1
##
## NOTE: n is number in each group
```

Fig 6. Initial Power Analysis Results

```
##      Balanced one-way analysis of variance power calculation
##
##               k = 4
##               n = 2.182995
##               f = 2.208578
##       sig.level = 0.05
##           power = 0.95
##
## NOTE: n is number in each group
```

Fig 7. Adjusted Power Analysis Results

Through our experimentation, we obtain a relatively large effect size(f = 8.706012) which indicates that the group means have a very large difference relative to the variability within groups. This initial power calculation resulted in an estimated power of 1.0 when using all of the obtained data (n = 27 per group), reflecting an excessively high statistical power due to the large effect size(Fig 6). Therefore, our sample size is quite adequate and more than capable of detecting a statistically significant difference between the levels of jumping time. However, to find the minimum required sample size for this example, we set the maximum difference threshold to 1500(Fig 7), which resulted in a smaller effect size relative to the original(f = 2.208578). Although the effect size is still large, we are able to run the power analysis and obtain the number of samples per group (n = 2.182995). Ultimately suggesting that our sample size be 12 samples(round up n = 2.182995 to n = 3 then multiply by k = 4) which can adequately detect differences across varied jumping times.

Discussion:

To reiterate the aim of our project and its findings, we were able to successfully use an RCBD design to generate a linear relationship that predicted an individual's change in blood adrenaline levels given their sex, age, and duration of jumping. This is expressed through the equation Y = 1397.295 + 62.35 * Treatment + 2.45 * age - 1800.411 * Gender M + e, where Y is the change in blood adrenaline levels. While all our predictors were significant at the 0.05 alpha level except age, we decided to use all our predictors in the model to compare the full picture with current day scientific literature done on blood adrenaline released in the body.

To investigate the applicability of our findings in a real world setting, we examined current research to see if our findings on the relationship between our three predictors and a change in blood adrenaline levels were scientifically valid. Firstly, we theorized that an individual's jumping time would be the most significant predictor of the rise in their blood adrenaline levels. This turned out to be false when compared with research from the National Library of Medicine, which stated that "we have confirmed that there is very little rise in venous plasma adrenaline levels during mild or moderate exercise"(Warren, Dalton, Turner, Clark, Toseland). Our belief that sex was another significant determiner of blood adrenaline response after exercise, with it causing a much higher rise in males than females was partially true. An article published by Oxford University supported the idea that sex did play a significant role in adrenaline response after exercise, but that it occurred in the opposite way of what we predicted: "adrenaline and pancreatic polypeptide all increased substantially more with exercise in males than females"(Dart, Anthony M, et al.). The last predictor we examined was age, with our prediction being this would have a limited to nonexistent impact on the changes in blood adrenaline levels. This assumption was proven true when comparing with literature published from the National Institute of Medicine, which states that "Epinephrine and norepinephrine plasma concentrations become lower or don't change significantly with advancing age"(Yiallouris, Andreas, et al.). Overall, our cross-analysis with current scientific studies indicates that our predictions for the variables of sex and duration of exercise were incorrect, but were accurate for our predictions on age.

Following this, the largest limitation of our project is that it was ultimately modeled after data that was not scientifically valid, so it can not be used to model the changes in adrenaline levels in a real world setting. This issue suggests that the Islands Simulator website we used produced data that is not reflective of the true adrenaline levels and its changes of the human population. Some other limitations and weaknesses of our model include the scope to which it is applicable in the context of the simulation, potential weakness in the Residual Plot, our model design, and the threshold of blood adrenaline levels rising. Our testing was only conducted on members of the island Providence, so it may not be applicable to people of all the islands especially if there are significant variations in the blood adrenaline responses of people in other locations. Another area of improvement could be our model design as it could have been better organized with more blocking and another setup like a crossover design. However, we were limited in this regard to only being allowed to use a design taught in class. The last potential weakness of our model was the lack of an upper ceiling for our response variable values as it is likely the blood adrenaline levels would have hit a maximum at a certain point of time as the time spent jumping continued to increase. To improve this, we can measure at higher jumping times until this upper limit is reached to add a cap to our equation.

Works Cited:

Dart, Anthony M, et al. “Gender, Sex Hormones and Autonomic Nervous Control of the Cardiovascular System.” *OUP Academic*, Oxford University Press, 15 Feb. 2002, academic.oup.com/cardiovascres/article/53/3/678/328102.

Warren JB;Dalton N;Turner C;Clark TJ;Toseland PA; “Adrenaline Secretion during Exercise.” *Clinical Science (London, England : 1979)*, U.S. National Library of Medicine, pubmed.ncbi.nlm.nih.gov/6690194/. Accessed 1 June 2024.

Yiallouris, Andreas, et al. “Adrenal Aging and Its Implications on Stress Responsiveness in Humans.” *Frontiers in Endocrinology*, U.S. National Library of Medicine, 7 Feb. 2019, www.ncbi.nlm.nih.gov/pmc/articles/PMC6374303/#:~:text=In%20one%20study%2C%20adrenaline%20production,that%20 observed%20in%20 young%20 controls.