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Biology 111: Briggs

The effects of exercise, gender, athleticism, and smoking on heart rate and recovery rate

Abstract

This experiment examined heart rates and recovery rates of students (mean age of 18.81) at a small Pennsylvania college. The effects of increasing duration step exercises along with gender, athleticism, and smoking are discussed. Heart rates and recovery rates increased with increasing exercise (P value = <0.001), although males had lower heart rates and recovery rates than females. Athletes had lower heart rates, recovery rates, and pulse rates than non-athletes. Finally, the heart rates and recovery rates of smokers were not different from non-smokers except for three instances (after 2 and 6 minutes of exercise and during the 6 minute recovery) when their rates were significantly higher. In addition, a negative correlation between body surface area and resting pulse was obtained ($P < 0.001$) with a correlation coefficient of – 0.186. In order to obtain these results, I used SPSS 17, Paired Sample T-tests, and Independent Sample T-tests. Age, gender, and the constraints of my study played a large role in interpreting my results.

Introduction

Because heart rate affects so many areas of the body, it is an important topic of research. Diseases such as onset atrial fibrillation can be caused by insufficient heart rate, especially when combined with other health issues such as obesity (Guglin et. al. 2010). Cigarette smoking, often associated with increased heart rate, also poses a risk for myocardial infarction or sudden death (Karakaya et. al. 2007). This clearly demonstrates the need to understand the ramifications of abnormal heart rate and find methods of addressing diseases arising because of abnormal heart rate.

Factors affecting heart rates and recovery rates include: gender (Theobald and Wandell 2007), smoking (Dietrich 2007), and athletic involvement (Rowland 2010). Men tend towards lower heart rates than women (Zhang 2007), although women have quicker recovery rates than men (Antelmi et. al. 2008). Similarly, smokers have higher blood pressure (Al-Safi 2005), higher heart rates (Dietrich et. al. 2007), and lower heart rate variability, suggesting decreased recovery rate effectiveness (e.g. longer recovery times) (Karakaya et. al. 2007). Athletes, however, have lower resting heart rates (Valentini and Gianfranco 2009). Antelmi (2008) demonstrates that heart rate recovery is rapid during the first 2 minutes after exercise stops and then slows significantly. In athletes, this process results in increased aerobic capacity, allowing for a more rapid recovery rate (Sloan et. al. 2009). Finally, increased surface area has been shown to impair heart rate recovery by causing a higher resting pulse and higher heart rate (Tigen et. al. 2009).

This experiment explores exercise's effect on heart rates and recovery rates. It also compares males and females, smokers and non-smokers, and athletes and non-athletes to ascertain exercise's effect on each group. Furthermore, this experiment explores the correlation between surface area and heart rate. In accordance with the above studies, I expect to find that exercise promotes increased heart rates and faster recovery rates. I also expect that men will have higher heart rates while women have higher recovery rates, that smokers will have higher heart rates and slower recovery rates than non-smokers, and that athletes will have a standard or lower heart rate and quicker recovery rate than non-athletes. Finally, I expect that greater surface area will result in higher resting pulse and heart rate.

Methods and Materials

This study was conducted with 733 students from a small undergraduate college in Pennsylvania. The average age of the 264 male participants and 469 female participants was 18.81. A total of 48 smokers and 685 non-smokers participated, as did 359 athletes (defined as a student participating in a fall or winter sports team and/or working out three times a week) and 374 non-athletes.

The students participated in a step exercise for 1, 2, or 6 minutes that involved stepping onto a block 17 cm high at a rate of 40 steps (one complete up and down cycle) per minute. After the exercise was finished, the students sat down within 5 seconds and had their pulse taken manually at the carotid artery. Every minute thereafter, the students had their pulse retaken until it was within +/- 6 beats of the student's resting pulse; resting pulse was the student's heart rate, taken before the exercise began.

The effect of increasing exercise duration on heart rate and recovery rate was analyzed using Paired Sample t-Tests. Independent Sample t-Tests analyzed differences between males and females, smokers and non-smokers, and athletes and non-athletes. Both tests were run through the computer program SPSS 17. The correlation between surface area and resting pulse was also tabulated using the same program.

Results

Table 1. Paired Sample T-test of all participants, comparing heart rates, recovery times, and resting pulse rates after increasing increments of exercise.		
Comparisons	Mean +/-S.E.	P
Normal Pulse Pulse 1	78.09 +/- 0.47 134.69 +/- 1.06	<0.001
Normal Pulse Pulse 2	78.09 +/- 0.48 146.90 +/- 1.06	<0.001
Normal Pulse Pulse 6	77.97 +/- 0.48 162.42 +/- 1.14	<0.001
Pulse 1 Pulse 2	134.62 +/- 1.07 146.79 +/- 1.06	<0.001
Pulse 1 Pulse 6	134.05 +/- 1.08 162.30 +/- 1.13	<0.001
Pulse 2 Pulse 6	146.58 +/- 1.07 162.33 +/- 1.14	<0.001
Recovery 1 Recovery 2	1.82 +/- 0.04 2.73 +/- 0.06	<0.001
Recovery 1 Recovery 6	1.82 +/- 0.04 3.92 +/- 0.07	<0.001
Recovery 2 Recovery 6	2.71 +/- 0.06 3.92 +/- 0.07	<0.001

Heart rates and recovery rates increased significantly after increased durations of exercise.

Table 2. Independent Sample T-test comparing mean +/- SE pulse and recovery rates of males and females after increasing exercise increments			
Factor	Mean Males +/- S.E.	Mean Females +/- S.E.	P
Rest	74.59 +/- 0.70	80.03 +/- 0.61	< 0.001
Pulse after 1 minute	123.03 +/- 1.62	141.25 +/- 1.30	< 0.001
Pulse after 2 minutes	136.89 +/-1.71	152.43 +/- 1.28	< 0.001
Pulse after 6 minutes	153.63 +/- 1.89	167.30 +/- 1.37	< 0.001
Recovery after 1 minute	1.60 +/- 0.06	1.96 +/- 0.05	< 0.001
Recovery after 2 minutes	2.50 +/- 0.10	2.87 +/- 0.08	0.003
Recovery after 6 minutes	3.67 +/- 0.12	4.07 +/- 0.08	0.008

The recovery rate, rest rate, and pulse rate of males was significantly lower than those of females.

Table 3. Independent Sample T-Test comparing mean +/-SE heart rates and recovery rates of athletes and non-athletes.			
Factor	Mean Athletes +/- S.E.	Mean Non-Athletes +/- S.E.	P
Rest	76.35 +/- 0.66	79.72 +/- 0.67	< 0.001
Pulse after 1 minute	129.55 +/- 1.52	139.61 +/-1.45	< 0.001
Pulse after 2 minutes	139.44 +/- 1.55	154.00 +/- 1.34	< 0.001
Pulse after 6 minutes	155.86 +/- 1.69	168.72 +/- 1.45	< 0.001
Recovery after 1 minute	1.56 +/- 0.05	2.09 +/- 0.06	< 0.001
Recovery after 2 minutes	2.23 +/- 0.08	3.23 +/- 0.09	< 0.001
Recovery after 6 minutes	3.51 +/- 1.00	4.33 +/- 1.00	< 0.001

The pulse rate, resting rate, and recovery rate of athletes was significantly lower than those of the non-athletes.

Table 4. Independent Sample T-Test comparing mean +/- SE heart rates and recovery rates of smokers and non-smokers.			
Factor	Mean Smokers +/- S.E.	Mean Non-Smokers +/- S.E.	P
Rest	78.79 +/- 1.71	78.02 +/- 0.49	0.687
Pulse after 1 minute	140.04 +/- 4.07	134.31 +/- 1.10	0.182
Pulse after 2 minutes	157.02 +/- 3.47	146.15 +/- 1.10	0.011
Pulse after 6 minutes	171.17 +/- 4.39	161.72 +/- 1.17	0.041
Recovery after 1 minute	1.81 +/- 0.15	1.84 +/- 0.04	0.902
Recovery after 2 minutes	3.00 +/- 0.20	2.72 +/- 0.06	0.257
Recovery after 6 minutes	4.72 +/- 0.26	3.87 +/- 0.07	0.003

The heart rates and recovery rates of smokers and non-smokers were not different except when the pulse was checked after 2 minutes and 6 minutes, and during the recovery after 6 minutes. For these factors, the smokers' rates were significantly higher than those of the non-smokers.

Correlation of body surface area to resting pulse:

The correlation of body surface area to the resting pulse is negative; the P value is < 0.001 and the correlation coefficient is – 0.186.

Discussion

My results in Tables 1, 2, 3, and 4 demonstrate that exercise affects heart rate and recovery rate. As Table 1 shows, heart rate and recovery rate both increased after increased durations of exercise. This is an important factor, as increased heart rate and recovery rate allows for increased oxygen uptake (Rowland 2010). Dimpka and Ibhazehiebo (2009) also found that heart rate increased after exercise, but that heart rate declined to its normal level again once exercise stopped. Similarly, Antelmi et. al. (2008) found that heart rate recovery following exercise was caused by an increase of parasympathetic drive and a decrease of sympathetic drive. Likewise, Valentini and Gianfranco (2009) noted a gradual increase of heart rate until it peaked and also that regular exercise reduced resting heart rate. Variables that can affect heart rate and recovery rate include gender and body mass (Nielson et. al. 2010), age, exercise intensity, aerobic capacity, and resting heart rate (Dimpka and Ibhazehiebo 2009). At its simplest level, exercise causes heart rate to increase until exercise stops; however, other factors can influence and perhaps even alter this outcome.

Gender is a significant factor in heart rate; as Table 2 demonstrates, male heart rates, recovery rates, and resting pulse rates were lower than those of females. Valentini and Gianfranco (2009) concur, saying that women have higher resting heart rates. Sloan et. al. noted that women have lower VO2 max (the maximum ability to intake oxygen during exercise) than men (Sloan et. al. 2009; George et. al. 2009), also supporting my findings. Other studies, however, show that heart rate recovery is more rapid in women (Antelmi et. al. 2008). An important factor to consider, though, is that Antelmi et. al. (2008) studied participants from ages 15-82. Age strongly affects heart rate, as I will later explain, and the large variation of ages and concentration of certain age groups probably caused this unexpected result.

Dubowy et. al. (2008) demonstrated that, until puberty, males and females display the same exercise performance. They further explain that male oxygen uptake (VO2 max) increases rapidly for a short time after puberty due to the sudden influx of testosterone. The authors also found that this uptake eventually plateaus and then begins to decrease, although this decrease occurs at a later age in men than it does in women. Furthermore, the authors demonstrated that the higher body and muscle mass of men (and the greater amount of body fat and ovarian hormones negatively affecting muscle mass in women) is directly linked to men's lower heart rates. In addition, Sloan et. al. (2009) suggest that estrogen causes diminished autonomic responses in women. My data correlated with the above findings, as men were shown to have lower heart rates and faster recovery rates than women.

According to Table 3, athletes' pulse rates, resting rates, and recovery rates were significantly lower than those of non-athletes, as was expected. As Valentini and Gianfranco (2009) explain, regular exercise activity leads to lower heart rates, even though exercise increases heart rate when an exercise regimen is first undertaken. The heart rate pattern after exercise does not vary: recovery rate is initially quick and then slows down (Dimpka and Ibhazehiebo 2009). Rowland and Roti (2010) further demonstrate that enough regular exercise with suitable intensity causes the formation of the "athlete's heart." The "athlete's heart" is marked by greater cardiac mass and volume and results in increased oxygen uptake, causing lower pulse rates, recovery rates, and resting rates. Interestingly, the authors show that male athletes have a slightly larger "athlete's heart" than women. Amounts of testosterone and estrogen, blood volume, body surface area, and amount of body fat are all believed to influence this difference. Similarly, Sloan et. al. (2009) report that men receive more cardioprotection from aerobic conditioning than women. Regular exercise creates lower heart rates over a period of time, as I found, even though exercise begins by increasing heart rate.

Table 4 demonstrates an unanticipated result: heart rates and recovery rates of smokers and non-smokers were not different except for a total of three factors, although these three factors were all significantly higher in smokers. Smokers are known to have higher blood pressure (Al-Safi 2005). Cardiovascular morbidity and mortality are also associated with long-term cigarette smoking, caused by high pulse rates and high blood pressure (Karakaya et. al. 2007). Karyakaya et. al. (2007) further explain that nicotine immediately increases heart rate and blood pressure. The authors also state that heart rate variability decreases, suggesting a slower recovery rate. Interestingly, Al-Safi (2005) demonstrates that the higher heart rate caused by smoking is not as significant for females as it is for men. Furthermore, even passive, or second-hand, smoke can harm the cardiovascular system, as particles of smoke become absorbed in the lungs (Dietrich et. al. 2007).

The contradictory findings of my study result largely from the subjects themselves. Students who said that they smoked were not required to note the amount of smoking or what substance they smoked. Since our participants were generally young, the majority simply did not yet have the chance to exhibit the symptoms associated with long-term chronic cigarette use. These factors have a large influence on this issue, especially since smoking in lower doses is a controversial topic that some researchers have not found overly harmful (Al-Safi 2005). It is clear, however, that heavy smoking causes severe health issues that could result in a predisposition to cardiovascular complications or sudden death (Karakaya et. al. 2007). Even my findings indicate that smoking resulted in higher heart rates and slower recovery times after longer exercise intervals for at least some students.

I expected to see that greater surface area would result in a higher resting pulse, but my results showed resting pulse decreased as surface area increased. Age and body surface area are related to heart rate changes (Sarnari et. al. 2009), and increased surface area (BMI) is shown to increase heart rate and decrease heart rate variability (suggesting a slower recovery rate), resulting in increased cardiac mortality (Sztajzel et. al. 2009). Tigen et. al. (2009) demonstrate that a larger surface area results in a higher resting pulse and higher heart rate after exercise. Furthermore, in a study done by Jenkins et. al. (2009) involving 6-minute walking distance, higher BMI negatively affected the distance achieved by women. Interestingly enough, the negatives associated with a larger BMI can be completely reversed by losing excess body fat (Guglin et. al. 2010). All of this information is in direct opposition to my findings. In my study, however, the students with a larger surface area were mostly male (as per the norm), and not obese; in addition, males naturally have lower heart rates than females.

Age also affects heart rate, as Cui et. al. (2008) show. Although the pattern of heart rate remains identical (recovery starts quickly and then tapers off), younger people have faster heart rate declines than older people (Dimpka and Ibhazehiebo 2009). Oxygen uptake is also higher in younger people, but there is no significant difference regarding resting heart rate (Dimpka and Ibhazehiebo 2009). Accordingly, the differences of younger people may be due to a lower BMI (Dimpka and Ibhazehiebo 2009). Women also have a lower heart rate than males, as their maximum heart rate begins to decline before a male’s heart rate (Dubowy 2008). Similarly, Valentini and Gianfranco (2009) also state that maximum heart rate declines with age. This was not reflected in my study because the subjects were all very close in age and therefore lacked any sort of age variability.

There are some possible errors to my study. The close age of all the participants excluded data due to the lack of certain affects that become prominent only in older or younger participants. Also, despite timing, there was variability in how quickly the students performed the step exercise. Furthermore, there was a two week difference between exercise 1 and exercises 2 and 6. Finally, students were not required to be specific regarding why they considered themselves athletes, and students were not asked to give further information regarding their smoking habits.

Clearly, this is an important research topic that should continue to be explored. As George et. al. (2009) show, cardio-respiratory fitness is important for educational purposes regarding fitness levels and fitness goals, while also lessening cardiovascular risk. Increased heart rate represents an increased mortality risk due to cardiovascular disease, especially in men (Theobald and Wandell 2007). These issues cannot be taken lightly, and further research is needed in the area of age and heart rate. Age is one of the greatest effecters of heart rate; as illustrated by my study and by reports of other studies, the age of participants can skew results greatly and cause different health concerns for different age groups. Also, the reasons behind the differences observed between men and women regarding heart rate is not well known and requires more research. In conclusion, heart rate is affected by exercise, gender, age, BMI, smoking, and athleticism, among many other factors, and the factors of age and gender both merit further study.

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