Effect of caffeine on breathing and pulse rate biology essay

ASSIGN BUSTER

Caffeine is a natural chemical substance known to have stimulant properties within the body (Addicott MA & Laurienti PJ, 2009). Prior research focused on caffeine's ability to affect ventilation, blood pressure, mood, and mental stress (Kennedy MD et al. 2008; Addicott MA & Laurienti PJ, 2009). Their findings concluded that caffeine actually spiked blood pressure, ventilation and heart rate (Kennedy MD et al., 2008); while others claimed that it had no effect on heart rate or breathing (Ratliff-Crain J et al., 1989). Some gaps in these studies were that very few actually measured the rate of breathing, but rather focused on the volume of breathing and the percent oxygen saturation/diffusion (Chapman R & Stager JM, 2008), as well as blood pressure, rather than heart rate (Ratliff-Crain J et al., 1989).

This experiment aimed at finding a clear relationship between ingesting caffeine, and its effect, if any, on breathing and heart rate, after the subjects performed incremental stepping exercises. The significance of this study is vital in sport, particularly caffeine's role in the Olympics (Spriet LL, 1995), and its potential as a prophylactic in treating asthma (Welsh EJ et al., 2010).

Methods:

See School of Biological Sciences (2010). Refer to Appendix (1) for the hypotheses.

Results:

Using the t-test it was found that the mean (+/- range) change in pulse rate was not significantly different between non-caffeine (Group A) and caffeine

(Group B) treated subjects (t= 0. 4, df= 161, P= 0. 7 ie P> 0. 05). (Refer to Appendices 2a, 2b, 3a, and 3b for the data and t-test)

Figure 1: The mean of changes in the pulse rate of non-caffeinated (control) and caffeinated (treatment) participants after performing a stepping exercise. Error bars are the range. N= 140 and n= 75 for the non-caffeinated and caffeinated groups respectively.

Using a paired two sample for mean t-test it was found that the mean (+/- range) change in respiration rate was not significantly different between non-caffeine (Group A) and caffeine (Group B) treated subjects (t=-0. 7, df= 132, P= 0. 5 ie P> 0. 05). (Refer to Appendices 2a, 2b, 3a, and 3b for the data and t-test)

Figure 2: The mean of changes in the breathing rate of non-caffeinated (control) and caffeinated (treatment) participants after doing a stepping exercise. Error bars are the range. N= 140 and n= 75 for the non caffeinated and caffeinated groups respectively.

Discussion:

The results of the investigation revealed that the difference in mean pulse rate change between the two groups was 1. 1 BPM and the mean breathing rate was 1 BRPM. This indicated that there were no significant difference between mean change in pulse rate and breathing rate between the group that was taking the caffeinated drink (treatment), and the group that ingested the non -caffeinated beverage (control). Therefore, since (P> 0. 05)

the null hypothesis (HO) was accepted, and the alternative hypothesis (HA) was rejected.

Prior research revealed that caffeine had no significant effect on the ventilator responsiveness or exercise ventilation on exercise -induced subjects following the administration of a moderate dose of caffeine (8mg per kilogram of body weight). Any modest increase in breathing rate was attributed to caffeine's secondary effect on the Central Nervous System, rather than a direct effect on the respiratory system (Chapman R & Stager JM, Caffeine stimulates ventilation in athletes with exercise-induced hypoxemia, 2008). However, other studies have suggested that caffeine acted in the manner of a ventilator stimulant and increased the breathing rate of individuals (Chapman R & Mickleborough TD, 2009).

Similar studies found that caffeine increased the mean change in pulse rate by 6 beats/min an hour after ingesting caffeinated coffee (350mL, 140mg of Caffeine). This revealed that while caffeine may affect the heart rate, its relative impact had no significant effect (Kennedy MD et al., 2008). Further research concluded that regular consumption of coffee could not increase the heart rate and blood pressure; unless consumed chronically and excessively (Ratliff-Crain J et al., 1989).

The experimental data revealed that naturally there were a large range of values, beginning from as low as {3 BPM, 1 BRPM} and peaking to {110 BPM, and to 62 BRPM} respectively. The issue with having such a large range was that it masked any significance to the mean. The extreme data points would distort the mean value, even if caffeine had proven to have a

significant effect. Thus, its effect would not be reflected in the results. Whilst increasing the sample size and repeating the experiment a number of times would improve the reliability of the experiment, it would do little to increase the precision of results. This is due to the presence of natural variation. Different people have differing athletic ability, sensitivity to caffeine, or gender differences, which the sample sizes (n= 140 and n= 75), did not account for. The failure to take account for the nature of the sample sizes greatly reduced the power of the experiment. Research suggests that males and females may actually have distinct responses (Kennedy MD et al., 2008), and so must be accounted for separately. This could be improved by separating the caffeinated and non- caffeinated groups into gender types, and perhaps only observing its effect on athletes (Chapman R & Stager JM, Caffeine stimulates ventilation in athletes with exercise-induced hypoxemia, 2008).

The amount of coffee may have been too small experimentally to produce a noticeable effect. Switching the dose to a full cup of coffee could enhance the effects of caffeine (Kennedy MD et al., 2008), or perhaps the length of time between ingestion and exercise was too short. Increasing the period from 1 hour to 2 hours could give caffeine more time to produce its effects (Kennedy MD et al., 2008). The accuracy of the results could have been improved by using more sophisticated counting methods to measure heart rate like using a digital pulse readout (i. e. an ECG), or percentage arterial oxyhemoglobin saturation to measure ventilation, which would produce more accurate results (Chapman R & Stager JM, Caffeine stimulates ventilation in athletes with exercise-induced hypoxemia, 2008).

The implication of this study is particularly important in the issue of caffeine bans from the International Olympic Committee, and whether or not it provides legal ergogenic advantage to athletes in short distance running or other aerobic activities. The rules governing caffeine ingestion prior to exercise may be revised if caffeine is proven to have substantially enhanced the athlete's pulmonary capabilities (Spriet LL, 1995). Other future studies of caffeine include developing prophylactics for asthma (from its anti-inflammatory and bronchodilator properties). Randomised clinical trials have already confirmed improvements in lung function after using caffeine. This could produce new ' front line' drugs to treat asthma (Welsh EJ, 2010).

In conclusion, the caffeinated and non caffeinated groups showed a slight mean difference of 1. 1 BPM and 1 BRPM, but statistically, there were no significant change in the mean heart rate or the mean breathing rate, which means the null hypothesis was accepted (P> 0. 05), and the effects of caffeine was not proven conclusive on either.

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