

**University of Tennessee, Knoxville [Trace: Tennessee Research and Creative](https://trace.tennessee.edu) [Exchange](https://trace.tennessee.edu)**

[Masters Theses](https://trace.tennessee.edu/utk_gradthes) [Graduate School](https://trace.tennessee.edu/utk-grad) (Graduate School (Graduate School (Graduate School (Graduate School (Graduate School

6-2011

# Why is there still so much confusion about VO2 plateau? A re-examination of the work of A.V. Hill

Richard Vincent Castle rcastle@utk.edu

#### Recommended Citation

Castle, Richard Vincent, "Why is there still so much confusion about VO2 plateau? A re-examination of the work of A.V. Hill. " Master's Thesis, University of Tennessee, 2011. https://trace.tennessee.edu/utk\_gradthes/955

This Thesis is brought to you for free and open access by the Graduate School at Trace: Tennessee Research and Creative Exchange. It has been accepted for inclusion in Masters Theses by an authorized administrator of Trace: Tennessee Research and Creative Exchange. For more information, please contact [trace@utk.edu.](mailto:trace@utk.edu)

To the Graduate Council:

I am submitting herewith a thesis written by Richard Vincent Castle entitled "Why is there still so much confusion about VO2 plateau? A re-examination of the work of A.V. Hill." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Kinesiology.

David R. Bassett, Jr., Major Professor

We have read this thesis and recommend its acceptance:

Dixie L. Thompson, Dawn P. Coe

Accepted for the Council: Dixie L. Thompson

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

Why is There Still So Much Confusion About VO<sub>2</sub> Plateau? A Re-examination of the Work of A.V. Hill

A Thesis Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

Richie Vincent Castle

August 2011

### **DEDICATION**

This work is dedicated to my mother who was on this earth 26 years of my life. You made me the person that I am today, and I have you to thank for so much of it. I will always love you and I know that you are watching me, proud of everything I am doing.

### **ACKNOWLEDGEMENTS**

I would like to acknowledge my primary professor, Dr. David Bassett Jr., who both conceived this idea and in no small way made it happen. From his near infinite patience and willingness to let an inexperienced student pursue questions, to his genuine concern about the quality and well-being of his students, he has far exceeded my expectations as both a mentor and professor.

I would also like to acknowledge those who assisted in testing, especially Scott Conger. Through his flexibility and desire to help others made my thesis doable instead of impossible. He is also has the talent of criticizing others without ever making them feel inferior.

I have had the support of both this department and my committee through every portion of it. My experiences have been essential in framing a view of a learning environment where we are all pushed to help each other succeed, and in return expect excellence. I want to acknowledge our department for instilling that value in me.

#### **ABSTRACT**

Maximal oxygen uptake (VO $_{2max}$ ) is regarded as the gold standard for assessing aerobic fitness. In 1923, A.V. Hill et al. proposed that  $VO<sub>2max</sub>$  represents the maximal ability of the body to take in and consume  $O<sub>2</sub>$  during strenuous exercise. Recently, however, controversy has arisen over the issue of whether a leveling off, or "plateau" in  $VO<sub>2</sub>$  is necessary to verify attainment of VO<sub>2max</sub>. Purpose: To compare two different VO<sub>2max</sub> protocols and determine if both protocols show direct evidence of an upper limit on  $O_2$  transport capacity. Methods: Nine runners (18-35 years old) completed a continuous graded exercise test (CGXT), followed by a discontinuous graded exercise test (DGXT). The CGXT consisted of gradually increasing treadmill running speed to the point of volitional exhaustion; the highest speed attained was labeled the peak treadmill speed. Over the next several days, participants ran at 80%, 90%, 100%, 105%, and 110% of peak treadmill speed for 10 minutes, or until volitional exhaustion was reached. Results: All participants (n=9) achieved a "VO<sub>2</sub> ceiling" (or upper limit) on the DGXT, while only 44% (n=4) achieved a "VO<sub>2</sub> plateau" on the CGXT. There was no significant difference between the VO<sub>2max</sub> obtained from a CGXT (57.4 ± 2.6 mL\*kg<sup>-1</sup>min<sup>-1</sup>) and DGXT (60.0 ± 3.1 mL\*kg<sup>-1</sup>min<sup>-1</sup>). There was no difference between maximal oxygen uptake (VO $_{2max}$ ) measured at 90%, 100%, 105%, and 110% of PTV (p>0.05). However, the highest  $VO<sub>2</sub>$  recorded at 80% PTV was significantly lower than that recorded at all other velocities ( $p$ <0.05). Conclusion: The VO<sub>2</sub> ceiling effect on a DGXT is inherently different than the  $VO<sub>2</sub>$  plateau effect on a CGXT. In this study, a ceiling was always seen on the DGXT, but a plateau was not always seen on the CGXT.

### **PREFACE**

"Bernard of Chartres used to say that we are like dwarfs on the shoulders of giants, so that we can see more than they, and things at a greater distance, not by virtue of any sharpness of sight on our part, or any physical distinction, but because we are carried high and raised up by their giant size."

--John of Salisbury, 1159

### **TABLE OF CONTENTS**



## **List of Figures**



#### Chapter 1

#### Thesis Introduction

 $VO<sub>2max</sub>$  is defined as the highest rate at which the body can take in and consume oxygen, measured during strenuous exercise  $[1]$ . VO<sub>2max</sub> is widely accepted as a measure of aerobic fitness, and is linked to reduction in all-cause mortality and cardiovascular disease [2-4]. In addition, it is considered and important factor in athletic performance of middle- and longdistance events. The concept of maximal oxygen intake (VO<sub>2max</sub>) can be traced back to the work of Hill and Lupton in 1923 [5]. In performing a discontinuous graded exercise test (DGXT), Hill and Lupton made several key observations that formed the Hill and Lupton  $VO_{2max}$  paradigm, which is still considered to be valid by most researchers.

The central tenets of the Hill and Lupton paradigm are as follows [6]:

- 1. There is an upper limit to oxygen intake
- 2. There are inter-individual differences in  $VO<sub>2max</sub>$
- 3. A high  $VO<sub>2max</sub>$  is a pre-requisite for successful distance running
- 4. VO<sub>2max</sub> is primarily limited by the circulatory and/or respiratory system

Attainment of VO<sub>2max</sub> must be confirmed by criteria established through rigorous research. Among those criteria is the VO<sub>2</sub> plateau. A VO<sub>2</sub> plateau signifies that, beyond a certain point, further increases in work rate fail to elicit further increases in  $VO<sub>2</sub>$ . The  $VO<sub>2</sub>$  plateau has been cited as a criterion for attaining  $VO_{2max}$  during a continuous graded exercise test (CGXT), by

several authors [7-17]. However, Day et al. [10] have shown that the lack of a VO<sub>2</sub> plateau on a CGXT does not necessarily invalidate a  $VO<sub>2max</sub>$  test. In their study, Day et al. found that participants lacking a  $VO<sub>2</sub>$  plateau had an equal  $VO<sub>2max</sub>$  when validated with a supramaximal test.

According to Noakes [18], the presence of a  $VO<sub>2</sub>$  plateau during a CGXT is essential to demonstrate an upper limit to oxygen intake. A plateau represents evidence that the upper limit to the oxygen intake has been reached. Because of the low prevalence of plateau during a CGXT, Noakes has questioned the entire  $VO_{2max}$  paradigm [18-32] set forth by Hill and Lupton [5]. He has maintained that, "…it is now established beyond doubt that the 'plateau phenomenon' is not a prerequisite for the identification of the 'true  $VO_{2max}$ ' in a majority of (but not all) subjects…we must now conclude that, according to the Hill model, the achievement of a 'limiting' cardiac output causing skeletal muscle anaerobiosis cannot be the exclusive reason why all subjects terminate maximal exercise. The prediction of the Hill model allow no other conclusion" [23], In more recent years, Noakes has acknowledged that  $VO<sub>2max</sub>$  exists, but he claims that it is limited by a "central governor" in the central nervous system, which limits the recruitment of skeletal muscles at high work rates [19, 22-32].

We believe that part of the confusion over the  $VO<sub>2</sub>$  plateau phenomenon results from the fact that the work Hill and Lupton [5]was based upon DGXTs, but most researchers today use CGXTs. The origin of the term "plateau" probably originated from Taylor et al. [33] who also used a DGXT protocol, but their results are not applicable to CGXT protocols. As modern

2

treadmills and metabolic measurement carts became available, researchers switched to using CGXTs, without rigorously examining whether the  $VO<sub>2</sub>$  plateau concept still applied.

By closely mirroring Hill and Lupton's protocol, we sought to determine whether taking runners to higher speeds than those achieved at the end of a CGXT would yield further increases in VO<sub>2</sub>. There were 3 hypotheses in this study:

- 1. There is not a statistically significant difference between  $VO_{2max}$  values achieved on a CGXT and DGXT protocol.
- 2. Having subjects run at supramaximal speeds does not result in further increases in VO<sub>2</sub>, beyond those seen at the end of a CGXT.
- 3. All participants display a  $VO<sub>2</sub>$  "ceiling" (or upper limit) on a DGXT, but not all participants display a plateau on a CGXT.

#### Chapter 2

#### Review of Literature

#### VO2max: Determinants and testing criteria

Maximal oxygen uptake is considered the "gold standard" for measuring cardiovascular fitness [34-35]. VO<sub>2max</sub> is a major determinant of performance in any aerobic endurance activity [36-39]. In addition, prospective observational studies demonstrate that high levels of VO<sub>2max</sub> are associated with lower rates of cardiovascular disease and all cause mortality [2-3, 40-43]. There is individual variance in cardiovascular fitness with clinical populations of individuals with cardiovascular, metabolic, and pulmonary disease having the lowest values, and elite endurance athletes having the highest values. In the sections that follow, the physiological determinants of VO<sub>2max</sub> will be discussed, as well as the various methods for measuring VO<sub>2max</sub>.

VO2max is the product of maximal cardiac output and arteriovenous oxygen difference [1]. Alterations in either of these variables will be associated with alterations in  $VO<sub>2max</sub>$ . The next section will discuss the physiological variables associated with these features.

#### Pulmonary Features

In healthy individuals, the pulmonary system typically does not limit  $VO<sub>2max</sub>$ . As predicted by Hill et al. [44], desaturation of arterial blood fails to occur during strenuous exercise based on the observation that the subjects have "never, even in the severest exercise, shown any signs of cyanosis", but also states that it would be dangerous to assume that arterial desaturation never occurs during strenuous exercise, as it could occur in those with high maximum cardiac output, due to the rapid movement of blood through pulmonary capillaries.

Though not confirmed by Hill himself, other researchers have found conditions that can cause arterial desaturation and limit  $VO_{2max}$  in healthy populations. Powers et al. [45] showed that elite athletes (VO<sub>2max</sub> mean = 70.1 ml\*kg<sup>-1\*</sup>min<sup>-1</sup>) experienced significantly greater arterial desaturation during maximal exercise compared to the recreational athletes (VO<sub>2max</sub> mean = 56.5 ml\*kg<sup>-1\*</sup>min<sup>-1</sup>). This arterial desaturation can be remedied with hyperoxic conditions (26% inspired  $O_2$  gas concentration), resulting in a significant increase of VO<sub>2max</sub> (6.7%) when compared to normoxic (21% inspired  $O<sub>2</sub>$  gas concentration) conditions. Unlike the elite athletes, the recreational athletes showed no difference in  $VO_{2max}$  or  $O_2$  saturation between hyperoxic and normoxic conditions.

Dempsey et al. [46] also showed that elite athletes are more likely to undergo arterial desaturation during maximal work, compared to individuals with normal fitness. Given the large difference in cardiac output between these two populations (40 vs. 25 L\*min<sup>-1</sup>), Dempsey et al. hypothesized the decreased time for diffusion of atmospheric oxygen to venous blood may be too short to allow an equilibrium to be established between the  $PO<sub>2</sub>$  in the alveoli and the pulmonary capillary blood. Thus, arterial desaturation occurs and this limits the VO<sub>2max</sub> of elite athletes.

In addition, high altitude is known to decrease arterial concentration, and thus  $VO<sub>2max</sub>$ . Due to the decreased atmospheric  $PO<sub>2</sub>$  at high elevations, the oxygen gradient that drives diffusion of  $O<sub>2</sub>$  from the alveoli into the pulmonary capillaries is reduced. Cymerman et al. [47] showed a decreased arterial saturation and greatly decreased  $VO<sub>2max</sub>$ , as a result of a decrease in arterial oxygen content.

Aside from elite athletes and high altitudes, pulmonary features play little to no part in limitations of  $VO<sub>2max</sub>$  in healthy populations.

#### Cardiac Output

Cardiac output refers to the rate at which the heart pumps blood through the body (i.e. the systemic circulation). Maximal cardiac output is a central factor affecting  $VO<sub>2max</sub>$ ; it is equal to the product of heart rate and stroke volume. Cardiac output has been proven to be a limiting factor of  $VO<sub>2max</sub>$  [1, 35, 48].

During maximal exercise, approximately 80-90% of arterial blood is re-directed to the working muscles [1]. In a study by Andersen and Saltin [48], , the quadriceps muscle group was isolated and exercised at work rates ranging from 10W to 60W . By cuffing the quadriceps just below the knee and measuring changes in the circumference of the quadriceps upon releasing the cuff, the researchers were able to determine blood flow to the quadriceps during exercise. The results showed that mean muscle VO<sub>2peak</sub> was 0.8 L/min for 2.3 kg of mass in the quadriceps. This calculates to a relative VO<sub>2</sub> of 347 ml\*kg of active muscle<sup>-1\*</sup>min<sup>-1</sup>, which far

exceeds the muscle's rate of oxygen uptake during maximal, whole body exercise. This suggests that blood flow to exercising skeletal muscle is limited by the finite amount of cardiac output, which is not an issue during exercise of isolated muscle groups.

Saltin [35] measured  $VO<sub>2max</sub>$  during various exercises, including cycling, running, arm cranking, and cycling plus arm cranking. Seven subjects completed a DGXT to measure  $VO<sub>2max</sub>$ using VO<sub>2</sub> measurements over 60 seconds. Cycling alone and cycling plus arm ergometry yielded a VO<sub>2max</sub> of 4.23 and 4.24 L/min of O<sub>2</sub>, respectively (p=0. 50). However, there was a significant difference (p<0.001) between the  $VO_{2max}$  of arm cranking alone (3.27 L/min) and cycling alone (4.66 L/min) in the 3 subjects that completed both these exercises. Combining arm-and-leg cycling failed to increase the VO<sub>2</sub> to amounts equal to the sum of the VO<sub>2</sub> of both activities. Thus, increasing the amount of muscle mass used (beyond a certain point) does not increase  $VO<sub>2max</sub>$  for that exercise, suggesting that a central factor, not the amount of muscle mass recruited, limits  $VO<sub>2max</sub>$ .

Saltin [49] demonstrated the limitations of cardiac output when comparing blood flow of single leg knee extensions versus two-legged cycling in nine healthy men. Saltin used the method previously described [48] to measure blood flow to a limb cuffing the leg just below the knee. During single-leg knee extensions, a work rate of 45 to 75 watts was maintained for 10 minutes, and no leveling off of whole body oxygen uptake could be detected with an increase of work rate. In a representative subject, the cardiac output during single-leg knee extensions was about 12 L/min, and 25 mL\*kg of active muscle  $1*$ min $1*$ , while the cardiac output during two-legged bicycling was 20 L/min and about 8 mL\*kg of active muscle  $1*$ min $1*$ . This

corresponds to a 67% increase in cardiac output with a 70% decrease in the cardiac output per kg of working muscle. In addition, doubling the muscle mass used from single-leg knee extensions to two-legged bicycling caused a 67% increase in  $VO<sub>2</sub>$  rather than a 100% increase. This strongly suggests that  $VO_{2max}$  is limited by the maximal cardiac output.

In 1977, Secher et al. [50] performed testing examining the effects of adding arm to leg exercises. Seven young male subjects were recruited. Subjects performed two, 20-minute bouts of exercise separated by an hour of rest. Each bout began with 10 minutes of either arm exercise (38-62% of VO<sub>2max</sub> for arm exercise) or leg exercise (58-78% of VO<sub>2max</sub> for leg exercise) and ended with 10 minutes of arm plus leg exercise (71-83% of VO<sub>2max</sub> for arm plus leg exercise). The results showed that adding sufficient arm exercise (accounting for 40% of VO<sub>2</sub> for combined exercise) to leg exercise caused a reduction in blood blow without a change in mean arterial blood pressure, while adding leg exercise to arm exercise decreased mean arterial blood pressure. The authors concluded that oxygen supply to a large muscle group is limited by vasoconstriction, when another large muscle group is exercising simultaneously. If the amount of exercising muscle mass increases, vasoconstriction will occur to allow redirection of the limited cardiac output of the body.

Maximal cardiac output is affected by maximal heart rate, left ventricular dimensions, and blood volume. These factors are discussed in detail below.

8

#### Maximal Heart Rate

Maximal heart rate is affected by age and genetic factors, more so than training [1]. Two Fitness tests on 31 male and 35 female subjects administered 20 years apart showed a reduction in VO<sub>2max</sub> by 20% and a reduction in maximal heart rate by 12-15 beats with great individual variability [51], linking age-reduced  $VO_{2max}$  with decreased maximal heart rate.

A 1984 study by Ades et al. [52] examined the effect of large and small doses of both pindolol and propranolol (β-adrenergic blocking drugs) on treadmill exercise. Compared to placebo, all drugs decreased exercise duration (p<0.001), but the drug or drug dosage did not make a difference in reducing the exercise duration. Though the differences in heart rate and blood pressure varied with dosage for each drug, both showed an increase in the rate-pressure product, indicating a compensation for lower maximal heart rate via increased stroke volume.

Joyner et al. [16] examined the effects of beta-blockade in 11 trained (VO<sub>2max</sub> of 63.3  $mL*kg<sup>-1</sup>*min<sup>-1</sup>$ ) and 11 untrained (VO<sub>2max</sub> of 44.5 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>) male subjects. The subjects performed two walking treadmill tests to exhaustion prior to any treatment. Subjects were administered a placebo, propanolol, or atenolol for 1 week then tested using the same treadmill protocol. All subjects were tested under all three treatments. Both the trained and untrained individuals experienced a significant decrease in  $VO<sub>2max</sub>$  with beta-blockade compared to placebo. Trained individuals decreased from 63.3 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>(placebo) to 59.3 mL\*kg<sup>-1</sup>\*min<sup>-1</sup> (atenolol) or 56.2 mL\*kg<sup>-1</sup>\*min<sup>-1</sup> (propanolol). They also experienced a significant reduction in maximum heart rate from 184 bpm (placebo) to 150 bpm (atenolol) or 143 bpm

(propanolol). These results were mirrored in the untrained group. They experienced a significant decrease in  $VO_{2max}$  from 44.5 (placebo) to 42.6 (atenolol) or 41.6 (propanolol), and a significant reduction in maximum heart rate from 197 bpm (placebo) to 149 bpm (atenolol) or 144 bpm (propanolol). The significant drop in maximum heart rate was accompanied by a lower, but still significant, drop in  $VO<sub>2max</sub>$ .

In a review by Tesch [53], administration of beta-blockers in patients, healthy and trained subjects causes a 30-35% reduction in maximal heart rate, 5 to 15% reduction in VO<sub>2max</sub> and an increase in maximal stroke volume. The decrease in maximal heart rate will affect  $VO<sub>2max</sub>$ , but the relative decrease in  $VO<sub>2max</sub>$  will be less than the relative decrease in maximal heart rate. Thus, acute reductions in maximal heart rate in an individual, due to pharmacological treatment, will cause reductions in  $VO<sub>2max</sub>$ .

Administration of beta-blockade is shown to reduce  $VO<sub>2max</sub>$  by reducing maximum heart rate. This shows that central factors—specifically maximal heart rate—have direct effects on VO<sub>2max</sub>. The reduction of VO<sub>2max</sub> from these studies is usually less than the magnitude of the reduction in maximum heart rate, suggesting a compensatory increase in stroke volume.

#### Left Ventricle Dimensions

Blomqvist and Saltin [54] reviewed several articles showing a correlation between high levels of VO<sub>2max</sub> and increased heart size and volume. Total heart size was not correlated with

VO2max, but left ventricular volume (LVV) was correlated. Endurance athletes demonstrated a higher stroke volume to heart volume ratio than power-trained athletes [55].

During training, the left ventricle increases in thickness. Ehsani et al. [56] used echocardiography to estimate left ventricle wall thickness in 8 competitive swimmers who underwent a 9-week training program. In the same study, they followed 6 runners who discontinued training for 3 weeks. The swimmers increased left ventricle end-diastolic dimension from 48.7 mm to 52.0 mm (p<0.005) and VO<sub>2max</sub> from 52 to 60 ml\*kg<sup>-1\*</sup>min<sup>-1</sup> (p<0.005) over the 9 weeks of training. The detrained running group decreased left ventricular end diastolic thickness from 51 mm to 46.3 mm and VO<sub>2max</sub> from 62 ml\*kg<sup>-1\*</sup>min<sup>-1</sup> to 57 ml\*kg<sup>-</sup>  $1*$ min<sup>-1</sup> over the 3 weeks of detraining.

The increased left ventricular volume after training allows the heart to maintain the same cardiac output at rest, with fewer beats per minute. Typically, endurance trained athletes often demonstrate sinus bradycardia (heart rate <60 beats/min) at rest. This increased efficiency also allows a person to maintain a work rate with a reduced heart rate response compared to the pre-trained state [57-58]. A study of 253 subjects showed a "minimal" decrease in resting heart rate (1.9 to 3.4 beats per minute) accompanying 20 weeks of endurance training [59]. However, a separate study showed that athletes in the peak of their training are more likely than a sedentary control group to exhibit resting sinus bradycardia [57]. This suggests that the increased left ventricular volume allows for a lower heart rate at rest.

#### Blood Volume and Blood Doping

Blood volume is shown to increase with endurance training. A review study done by Sawka et al. [60] analyzed 18 reports on the effects of endurance training on blood volume and found a 10% increase in blood, plasma, and erythrocyte volume within 30 days of endurance training. The same review showed significant correlation (p<0.05) between total blood volume and VO<sub>2max</sub> (r=0.65 to r=0.92 depending on the study) [60].

A 1994 study by Davy et al. [17] examined if aging causes a decrease in total blood volume. Blood volume was measured in 7 young non-obese (24.7 years of age) and 7 older (66.1 years of age) men matched for body mass index and physical activity. Trained subjects were excluded due to a possible masking effect on blood volume. Younger adults were shown to have significantly more blood (6.2L vs. 4.7L), plasma (3.7L vs. 3.0L), and erythrocyte (2.5L vs. 1.8L) volume than older men. Younger men also had significantly higher VO<sub>2max</sub> (48.7 vs. 32.4  $mL*kg<sup>-1</sup>*min<sup>-1</sup>$ , suggesting a link between age-related changes in VO<sub>2max</sub> with reduction in total blood volume.

Blood doping (the act of removing, storing, and then reinfusing blood), is shown to increase VO<sub>2max</sub>. Glenhill's review [61-62] of several blood doping articles show that a reinfusion of 900 to 1350 mL of blood increases  $VO_{2max}$  by 4 to 9%, respectively, compared to control groups (injected with saline) that showed no increase. In the 1970s, Ekblom et al. [63] conducted the first study to show that blood doping increases  $VO<sub>2max</sub>$ . They also showed that loss of 800 and 1200 mL of blood corresponded to a 13 and 18% drop in hemoglobin concentration and VO<sub>2max</sub>, respectively. Upon re-infusing the blood (which had been frozen and

12

stored for a couple of months while the subjects produced new red blood cells), there was a 13% increase in hemoglobin concentration and 9% increase in  $VO<sub>2max</sub>$ . Correlation between blood loss and  $VO<sub>2max</sub>$  was r=0.97.

#### Arterio-venous oxygen difference

The arterio-venous oxygen difference refers to the difference in the oxygen content (ml of  $O<sub>2</sub>$  per 100 ml blood) between the arteries and veins. It is an indirect measure of oxygen extraction at the cellular level. In most people of normal fitness level, the arterial hemoglobin is almost completely saturated with oxygen, even during maximal exercise [64].

A study done by Ekblom et al. [65] measured arterio-venous oxygen difference in 7 college-age males before and after 16 weeks of endurance training. The training consisted of cross-country running 3 times a week that was: (a) dash (30-60 second sprint repeated 5-10 times with 2-3 minutes rest between each sprint), (b) interval (70-75% of maximum speed for 3- 6 minutes with 3-4 minutes of rest between each run), or (c) distance running (45-75 minutes of continuous running). Arterial concentration was measured during maximal cycling (6-minute cycle to exhaustion) using two catheters placed into the antecubital vein. The results demonstrated an increase in their arterio-venous oxygen difference during maximal exercise, from 138 to 143 mL/liter and an increase of VO<sub>2max</sub> from 3.12 L/min to 3.5 L/min. Though not as significant as other factors such as blood volume and maximal heart rate, an increase in arteriovenous oxygen difference has been linked to increases in training-induced  $VO<sub>2max</sub>$ .

#### Capillary Density

Capillary density in the peripheral skeletal muscles affects  $VO_{2max}$  in several ways: it increases mean transit time for oxygen exchange in working muscles [49], and decreases peripheral resistance.

Tesch et al. [66] conducted a study in 1984 showing that endurance athletes demonstrate increased capillary density. Taking muscle tissue samples from 8 elite weight/power lifters, 8 endurance athletes, and 8 non-athletes, myofrillar ATPase, NADHtetrazolium reductase, and amylase-periodic acid-Schiff were used to determine fiber type distribution and capillary density. The number of capillaries per mm<sup>2</sup> of muscle fiber in untrained individuals (306 capillaries per mm<sup>2</sup>) was significantly greater than power/weight lifters (199 capillaries per mm<sup>2</sup>) and significantly lower than endurance athletes (401 capillaries per mm<sup>2</sup>). This was the first study linking endurance training to increased capillary density.

Brodal et al. [67] took muscle biopsies from the lateral part of the quadriceps in 12 untrained (VO<sub>2max</sub> of 72.0 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>) and 11 untrained (VO<sub>2max</sub> of 51.3 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>) young men and examined capillary density using ATPase staining. Compared to untrained men, the trained men had significantly more capillaries per mm<sup>2</sup> (821 vs. 585) and capillaries per fiber (2.49 vs. 1.77).

Ingjer and Brodal [68] examined the capillary density of the quadriceps femoris in 11 young women. Five were endurance trained (62.1 ml\*kg<sup>-1</sup>\*min<sup>-1</sup>) and 6 were untrained (43.9  $ml*kg^{-1*}min^{-1}$ ). The endurance trained athletes had significantly higher capillaries per fiber ratio

(1.69 vs. 1.11) and greater capillaries per mm<sup>2</sup> (404 vs. 301) compared to the untrained group. This provides a link between capillary density and  $VO<sub>2max</sub>$  that exists in females, as well as males.

Andersen and Henriksson [69] showed that endurance training increases capillary density. Taking 5 subjects, and training them on a cycle ergometer for 40 minutes a day at 80% of VO<sub>2max</sub> for 8 weeks, Andersen and Henriksson measured capillary density and muscle fiber composition before and after training using amylase-PAS method and myofribrillary ATPase. A 16% increase in VO<sub>2max</sub> (49.0 to 56.6 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>) was accompanied by a 20% increase in capillaries per mm<sup>2</sup> (329 to 395), which was increased across type I, type IIa, and type IIb fibers. Capillary density is related to the  $VO_{2max}$  increase caused by training.

In his J.B. Wolffe lecture in 1986 [70], Dempsey noted that these capillary density adaptations exceed the adaptations of the lung capillary density during training. Traininginduced increases capillary density in skeletal muscle increases the total cross-sectional surface area of the capillaries, allowing more blood to interact with the mitochondria, and allowing the muscles to maintain sufficient arterio-venous oxygen difference even at high rates of blood flow.

#### Skeletal Muscle Respiratory Capacity

Training increases both the mitochondrial density and concentration of mitochondrial enzymes [71]. Endurance training studies show that an increase in  $VO<sub>2max</sub>$ </sub> is generally coupled with increases in succinate dehydrogenase (SDH), phosphofructokinase [72], lactate dehydrogenase, monocarboxylate transporters (MCT) 1 and 4 [73], and mitochondrial density [74]. Although not as important as cardiac output in determining the  $VO<sub>2max</sub>$ , the increased enzyme activity increases the rate of oxygen utilization within the cell and helps to create the oxygen pressure gradient between pulmonary capillary blood and the mitochondria necessary to maintain arterio-venous oxygen difference.

Although mitochondrial density is associated with increased fitness, a 2.2 fold increase of these mitochondrial enzymes is normally associated with a 20 to 40% increase in  $VO<sub>2max</sub>$  [75]. Holloszy and Coyle have reinforced this by showing that individuals with identical VO<sub>2max</sub> will actually show two-fold differences in mitochondrial density [76-77]. Despite that possibility that skeletal muscle respiratory factors do not affect VO<sub>2max</sub>, Holloszy and Coyle [76] observed in a study of runners done by Costill [78] that the succinate dehydrogenase levels of elite runners were 2.5 times greater than untrained controls.

Saltin et al. [79] demonstrated the effects of peripheral limitations on  $VO<sub>2max</sub>$ . Thirteen male participants completed 4 to 5 training sessions per week over 4 weeks of training. Each leg was trained differently. One leg was always trained for sprint or endurance, and the other leg was trained oppositely or not at all. Muscle samples of the quadriceps were sampled before and after training and assayed for SDH and myofibrillar ATPase. Compared to baseline,  $VO<sub>2peak</sub>$ tests on the trained leg demonstrated a 27% increase in VO<sub>2max</sub> while the untrained leg demonstrated a 7% increase in  $VO<sub>2max</sub>$ . In addition, SDH activity increased in trained legs compared to untrained leg, and lactate was "only continuously released from the [untrained]

16

leg". The authors concluded that training increases local adaptations that affect the metabolic response to exercise.

Klausen et al. [80] examined the effects of 8 weeks of single-leg cycle ergometer training on maximal single-leg and double leg fitness. Six healthy young males showed a 19% VO<sub>2max</sub> increase in single-leg exercise and an 11% increase in double leg testing. In addition, heart rate during submaximal single-leg exercise dropped 11% while it dropped 2% during submaximal two-leg exercise. This implies that specificity of training can affect both the VO<sub>2max</sub> and cardiac adaptations to training.

#### Protocols for measurement of maximal oxygen uptake

#### Discontinuous vs. Continuous Protocols

Graded exercise tests (GXT) can be classified as either continuous (CGXT) or discontinuous (DGXT). CGXT are more commonly used today [9-10, 12-14], while DGXT were more common in  $VO<sub>2max</sub>$  studies done in the 1950s and 1960s [5, 33, 81-84].

A DGXT consists of working at a constant load (e.g. running at a single speed or cycling at a fixed wattage) until steady-state VO<sub>2</sub> (or an "apparent steady state" - i.e. leveling off of VO<sub>2</sub> at a supra-maximal work rate) is achieved. That steady state value is the VO<sub>2</sub> for that workload. Constant load tests are repeated until the subject fails to show an increase in  $VO<sub>2</sub>$  with an increase in workload (referenced as a plateau, which will be discussed later)[5, 33, 75, 79, 85], or failed to complete the workload for a duration [33]. The highest  $VO<sub>2</sub>$  achieved is considered

the  $VO_{2max}$ . This protocol typically requires the subject to repeat multiple tests separated by rest, usually a day or more in length.

A CGXT consists of a single continuous test. In it, the work rate or exercise intensity is gradually increased (usually at 1-minute intervals) until the participant reaches volitional exhaustion. The highest  $VO<sub>2</sub>$  achieved during any 15-, 30-, or 60-s period is termed the  $VO<sub>2max</sub>$ . The test usually lasts between 8 and 20 minutes, from start to finish.

#### Validity of CGXT compared to DGXT

Studies show that DGXT and CGXT yielded equally valid  $VO<sub>2max</sub>$  measurements [86-91]. Stamford [86] studied VO<sub>2max</sub> values of 10 males subjects using various DGXT and CGXT treadmill tests. Each participant was administered a DGXT, 2 CGXTs (test-retest), and 2 constant load tests. The DGXT was performed first to establish a maximal work rate. The DGXT consisted of running 7.0 mph for 3 minutes, resting 10 minutes, and then increasing the treadmill incline by 2.5% for each successive 3 minute run. The test was completed when subject could not complete the 3 minute run. Both CGXT were identical to the DGXT, but the stages were 2 minutes instead of 3 and there was no rest between the bouts. The constant load tests were run at the speed and peak incline from the DGXT, and 2.5% increased incline. The results of each test yielded VO<sub>2max</sub> of 50.14 (DGXT), 49.58 (best of either CGXT), 49.30 (constant load at peak incline), and 49.41 (constant load at peak incline plus 2.5%) ml\*kg<sup>-1\*</sup>min<sup>-1</sup> (p>0.05). Even

with the variance in protocol and supramaximal intensity, all protocols yielded equal VO<sub>2max</sub> results.

Shephard et al. [87] studied 24 Canadian males varying greatly in fitness (VO<sub>2max</sub> 30 to 65 ml\*kg<sup>-1\*</sup>min<sup>-1</sup>) across CGXT and DGXT in treadmill, bicycle, and step modes of exercise administered in a randomized order. The treadmill exercise consisted of running uphill at speed of 5 to 6 mph and slopes of 1% to 18%. Bicycle ergometer exercise was performed at 60 to 90 revolutions per minute with varying workloads. Step mode exercise consisted of a single 18 inch step climbed with two paces at a rate of 80 to 140 paces per minute. The CGXT and DGXT were performed by predicting the participants work rate at  $VO_{2max}$  based on the physiological response of a respective submaximal work rate. DGXT was a constant load at 110% of predicted maximum work rate, and further bouts were adjusted in light of the participant's performance, while the CGXT was set at 90% to 100% of the predicted maximum work rate and increased slightly every 2 minutes. The VO<sub>2max</sub> was not significantly different between CGXT (3.84 L/min) and DGXT (3.74 L/min), regardless of whether it was a treadmill, bicycle, or step exercise test.

Washburn and Seals [90] examined CGXT and DGXT in arm cranking in 20 males subjects. The CGXT portion increased power every minute, while the DGXT increased power every two minutes with one minute of rest. Initial power and power increases were identical between the DGXT and CGXT. The initial power for arm cranking was estimated from the assumption that the VO<sub>2peak</sub> of arm cranking is equivalent to 60% of the participant's treadmill  $VO<sub>2max</sub>$ , and the participant would reach  $VO<sub>2max</sub>$  by the fifth stage. Order of days was randomized, and no significant differences were found between the  $VO<sub>2peak</sub>$  in the CGXT (33.8

ml/kg/min) and DGXT (34.2 ml/kg/min). DGXT and CGXT yield equivalent VO<sub>2peak</sub> in arm cranking as well.

More recent studies have verified this finding. Duncan et al. [11] compared the VO<sub>2max</sub> values from a the DGXT protocol used by Taylor et al. [33], and the CGXT protocol used by Stamford [86]. In the 10 males subjects tested, the VO<sub>2max</sub> of the DGXT (56.8 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>) was not significantly different from the CGXT (55.8 mL\*kg<sup>-1\*</sup>min<sup>-1</sup>). These results concur with Stamford [86].

Day et al. [10] performed a cycling CGXT on 71 participants. The protocol consisted of pedaling at 20W with an increase of 5W every 20, 15, or 12 seconds until the participant could not maintain a cadence of 60 revolutions per minute. Day et al. then followed up with 6 of the participants to perform a DGXT at varying supramaximal and maximal loads. Results showed that the VO<sub>2max</sub> of the CGXT (4.50 L/min) did not vary significantly from the DGXT (4.51 L/min).

Rossiter et al. [12] performed a cycling CGXT on 7 subjects followed by a 5-minute recovery at 20W, and then a constant load cycling bout at 95% or 105% of their peak work rate during the CGXT. The following constant load bout was similar to the bout of a DGXT that would be near or at VO<sub>2max</sub>. The results showed no significant difference between the VO<sub>2max</sub> of the CGXT (4.3 L/min), the 105% of peak work rate bout (4.3 L/min) and the 95% of peak work rate bout (4.1 L/min).

The validity of  $VO<sub>2max</sub>$  measurements between a DGXT and CGXT equally valid.

#### Various Exercise Modes

Research has shown that measured  $VO_{2max}$  is affected by the amount of muscle mass recruited during exercise. For this reason, various exercise modes will yield different  $VO<sub>2max</sub>$ values, depending on the amount muscle mass being used. Taylor et al. [33] showed that VO<sub>2max</sub> derived from a treadmill VO<sub>2max</sub> test increased from 4.0 to 4.2 L/min when arm ergometry was added.

McArdle et al. [92] tested 15 male students across several different exercise tests. They performed a treadmill and cycling CGXT; treadmill and cycling DGXT; Mitchell, Sproule and Chapman treadmill test; and a Balke treadmill test. They found that Balke, CGXT, and DGXT treadmill protocols yielded a VO<sub>2max</sub> that was 10-11% higher ( $p$ <0.01) than a cycling protocol, with no significant difference between the DGXT and CGXT cycling  $VO<sub>2max</sub>$  values.

Hermansen and Saltin [93] conducted a study that supported the findings of McArdle et al. [92]. They tested 55 male subjects ranging from 18 to 68 years of age. The participants completed a bicycling DGXT based on Astrand and Saltin's [81] protocol and a treadmill DGXT based on Taylor et al.'s protocol [33]. The resulting  $VO_{2max}$  values for bicycling, level treadmill running, and incline treadmill running were 4.34, 4.48, and 4.68 L/min (p<0.01 compared to bicycling), respectively. They concluded that inclined running yields a higher  $VO<sub>2max</sub>$  than bicycling due to the usage of more muscle during exercise.

Astrand and Saltin [82] compared  $VO<sub>2max</sub>$  in various exercises (sitting cycling, supine cycling, simultaneous leg and arm work, running on a treadmill, skiing, swimming, and arm

work) in seven subjects. VO<sub>2max</sub> was 15% lower in seated cycling compared to supine cycling, and 30% lower in arm ergometry than in cycling. Thus, the greater the difference in muscle mass recruitment, the greater the differences in measured  $VO_{2max}$ . In addition, even if the working muscle mass is equivalent, the method of work may affect  $VO<sub>2max</sub>$ , as demonstrated by the difference between seated and supine cycling.

Buchfuhrer et al. [94] tested 12 "normal men" by administering a cycling and treadmill CGXT, and found that  $VO_{2max}$  values measured on treadmill running were 6% higher compared to cycling. Bergh et al. [95] had 10 males perform  $VO_{2max}$  tests with cycling, cycling plus arm cranking, and treadmill running. Running yielded the highest  $VO_{2max}$  (4.44 L/min) which was significantly greater than cycling (4.12 L/min), but not significantly greater than cycling plus arm cranking (4.40 L/min). These were all significantly greater than arm cranking alone (VO<sub>2max</sub> of 3.01 L/min). They concluded that  $VO<sub>2max</sub>$  relies on the exercising muscle mass to a certain extent.

However, there are certain exceptions to the fact that running yields higher  $VO<sub>2max</sub>$  than cycling. If an athlete is particularly well trained in one exercise mode, he or she can achieve relatively high VO<sub>2max</sub> (compared to other modes) despite differences in muscle mass usage. For example, Stromme et al. [96] took 37 athletes (24 cross country skiers, 8 rowers, and 5 cyclists) and administered  $VO_{2max}$  tests using both their respective sport and treadmill incline protocol. The 5 cyclists achieved a VO<sub>2max</sub> on the cycle test that was about 5-10% higher than uphill running. All other athletes achieved VO<sub>2max</sub> in their respective sport that was higher than during uphill running, ranging from 2.5 to 6.0% higher. The cycling result contrasts with previous studies showing that running elicits a  $VO<sub>2max</sub>$  value that is 10% higher than cycling.

Magel et al. [97] trained 15 college-age male recreational swimmers in swimming for 10 weeks. Training consisted of 1 hour of practice 3 days per week. The first week was one set of ten 50-yard swims and five 100-yard swims. After the second week, 200- and 300-yard swims at maximum speed were added. After the fourth week, 300, 400, and 500 yard swims were added increasing the volume from750 yards to 2000 yards over the course of the training. Prior to the training, the participants completed treadmill (VO<sub>2max</sub> of 4.05 L/min) and swimming (VO<sub>2max</sub> of 3.44 L/min) VO<sub>2max</sub> tests. After training, their VO<sub>2max</sub> from the swimming test (3.82 L/min) increased significantly, but their VO<sub>2max</sub> on the treadmill test (4.11 L/min) did not. By training specifically for swimming, the participants'  $VO<sub>2max</sub>$  during swimming became relatively higher, and therefore  $VO_{2max}$  measurements would vary depending on the training of the exercise mode being tested.

Pechar et al. [98] enrolled 60 college age men, and randomly assigned them to 8 weeks of bicycle ergometer training, 8 weeks of treadmill training, or a no-training control group. Before and after training, they administered cycling and treadmill  $VO<sub>2max</sub>$  tests. The largest gains in cycle VO<sub>2max</sub> were seen in the bicycle-trained group, and the largest gains in treadmill VO<sub>2max</sub> were seen in the run-trained group. However, the bicycle-trained group showed significantly lower improvements in their treadmill VO<sub>2max</sub> compared to their cycling VO<sub>2max</sub>. This indicates that training for a specific activity will yield greater improvements in VO<sub>2max</sub>, with that specific exercise mode. Again, training causes relative increases in the trained exercise mode with little

change to other exercise modes. Aside from the exercise mode itself, the amount of training in the mode will affect the measured  $VO<sub>2max</sub>$ .

A 1974 study by Secher et al. [99] compared VO<sub>2max</sub> of 16 subjects performing running, cycling, arm, leg, and combined arm plus leg work. The  $VO_{2max}$  for arm ergometry was on average 85% of the VO<sub>2max</sub> achieved during leg exercise, while the VO<sub>2max</sub> of arm-plus-leg exercise was 99% to 117% of the VO<sub>2max</sub> achieved during leg exercise. The authors suggest that when two muscle groups are combined for exercise, the increase in oxygen uptake is proportional, but not equal to, the respiratory capacity of each muscle group individually. By increasing the amount of muscle used, the  $VO_{2max}$  of the test will increase.

In 1984, Nagle et al. [100] tested the relative contributions of the arms and legs in eliciting VO<sub>2max</sub>. 10 healthy non-arm-trained males performed CGXTs for arm only, leg only, 10% arms/90% legs, 20% arms/80% legs, and 30% arms/70% legs. The results showed that 10% arms/90% legs elicited significantly greater  $VO_{2max}$  than 100% legs, 100% arms and 30% arms/70% legs. The authors concluded that the significantly lower  $VO<sub>2max</sub>$  during exercise with the legs alone was caused by a reduction in the active muscle mass, while during 30% arms/70% legs, the cause of the lower  $VO_{2\text{max}}$  was excessive arm loading and insufficient leg loading. Depending on the distribution of work,  $VO<sub>2max</sub>$  can be affected by the relative division of work between muscle groups.

Altering the amount of muscles used during an exercise will affect the  $VO<sub>2max</sub>$ , with greater muscle usage yielding higher  $VO_{2max}$  (to a point). In addition, the specificity of training to certain exercise modes will affect the measured  $VO<sub>2max</sub>$ .

#### Criteria for achieving maximal oxygen uptake

Because maximal exercise tests rely on the participant to voluntarily work to maximal effort, there is a need for objective criteria to validate  $VO_{2max}$  measurements. If a participant failed to give a maximal effort, the  $VO_{2max}$  measurements would be underestimated, and the results of that test would be invalid. Without proper criteria for validation, the results of a VO2max test could actually be submaximal, and therefore, invalid.

#### VO2 Plateau

The operational definition of a VO<sub>2</sub> plateau can be ambiguous, as Howley et al. [101] observed. The assumption is that at the final intensity of a CGXT,  $VO<sub>2</sub>$  will be identical to the stage before it, yielding a perfectly flat slope. However, this type of a "true plateau" does not usually occur. The final stage may demonstrate an increase in  $VO<sub>2</sub>$  ranging from no increase to the same increase as seen on the previous stage. As a result, one must choose a cut-point for the increase in VO<sub>2</sub> that will constitute a VO<sub>2</sub> plateau. The criterion for VO<sub>2</sub> plateau was established in 1955 by Taylor et al. [33], and determined to be an increase less than 150 ml/min with an increase of 2.5% incline at 7 mph on a treadmill. Their reasoning was that the standard increase between stages was 300 ml/min, and they recognized that there is variability in  $VO<sub>2</sub>$ from minute-to-minute, and setting the plateau threshold at no  $VO<sub>2</sub>$  increase in the last stage

would not properly capture the phenomenon. Thus, a  $VO<sub>2</sub>$  plateau was operationally defined as being less than one-half of the expected increase.

Despite the VO<sub>2</sub> plateau criterion established by Taylor et al. [33], only 20-50% of VO<sub>2max</sub> tests result in a plateau by this definition [7-15]. Thus, although it has been considered by some to be a primary criterion for achieving  $VO_{2max}$  [16-17, 102-103], the ACSM Guidelines for Exercise Testing and Prescription [104] does not recognize it as such.

This has led some investigators to use the term VO<sub>2peak</sub> instead of VO<sub>2max</sub> to describe the key outcome variable resulting from a maximal GXT.  $VO_{2peak}$  refers to the highest VO<sub>2</sub> attained during a graded exercise test, without regard to whether an individual achieves a plateau, or any of the secondary criteria for attainment of  $VO<sub>2max</sub>$ . For example, most of the research on maximal exercise testing in children uses  $VO<sub>2peak</sub>$  instead of  $VO<sub>2max</sub>$ , due to the difficulty in showing evidence of a VO<sub>2</sub> plateau in children [105]. VO<sub>2peak</sub> is also commonly used to describe the results of maximal GXTs using smaller muscle groups (e.g. arm cranking or one-legged cycling).

#### Blood lactate

Blood lactate is an indicator of relative exercise intensity. High levels of blood lactic acid are associated with recruitment of fast-twitch muscle fiber, elevation in plasma epinephrine concentrations, and reduction in liver blood flow [101].

Astrand [106] established a criterion value for blood lactic acid levels 3 minutes following a maximal GXT. Out of all of the participants in the study, roughly 50% of them demonstrated a  $VO<sub>2</sub>$  plateau. Of those who did, the onset of the plateau was correlated to 3 minutes post-exercise lactate concentrations of 7.4-8.4 mM, while the blood lactate values 3 minutes post-exercise for the whole test ranged from 6.7 to 10.1 mM. However, the participants in Astrand's study were children ranging from 14-18 years of age, and so the results can only be applied to that group.

Other investigators have used a blood lactate of 7.3 mM [107-108] or 5.5 mM [109]. Also, other researchers have demonstrated lower blood lactate values during maximal testing, however they also used a single supramaximal test [110] or a CGXT [111] compared to the DGXT used by Astrand [106]. The criterion for blood lactate concentrations should be set after considering both the test and population being studied.

#### Respiratory Exchange Ratio

Respiratory exchange ratio (RER) is the ratio of the carbon dioxide production to oxygen consumption, measured at the mouth. It is mathematically computed by dividing  $VO<sub>2</sub> / VCO<sub>2</sub>$ . Although the body produces  $CO<sub>2</sub>$  as a result of metabolism, it is also produced by buffering hydrogen ions (produced by lactic acid) with bicarbonate anions, resulting in  $CO<sub>2</sub>$  and H<sub>2</sub>O formation. Thus, RER can be used as an indirect measure of lactic acid accumulation, and this was confirmed by Wasserman et al. [112]. Issekutz et al. [107, 113] established RER ≥ 1.15 as a
criterion for achieving VO<sub>2max</sub>. By following excess  $CO<sub>2</sub>$  and lactic acid concentrations pre to post exercise, they determined a linear relationship between the excess  $CO<sub>2</sub>$  and lactic acid concentrations [107]. Following up on this, Issekutz et al. [113] determined from a sample of 32 untrained subjects, all achieved a maximum RER of 1.15.

However, other research has shown that RER  $\geq 1.15$  is not achievable by everyone. Robinson [110] suggested that this criteria was reasonable for those aged 20 to 60 years, however younger participants could not achieve this RER. Cunningham et al. [111] showed that sixty-six, 10-year old hockey players could not achieve an RER >1.10 on a maximal treadmill exercise test. Sidney and Shephard [114] also demonstrated that men and women aged 60 to 83 who gave a "good effort" showed low prevalence of RER ≥ 1.15 (20% in women and 37% in men) during maximal treadmill GXT's. Though RER of 1.15 is an established criterion for attainment of  $VO<sub>2max</sub>$  from previous studies, an RER of 1.10 would be more appropriate for general populations

## Maximal Heart Rate

Maximal heart rate is predicted by the ACSM as 220 minus age in years [104]. This criterion is problematic because the standard deviation of this estimate is  $\pm$ 11 beats/min [115]. ACSM [104] suggests using it as a secondary criterion rather than a primary criterion for this reason.

Robergs and Landwehr [116] present the origins and subsequent basis of information for this age-predicted equation. One of the original works by Astrand et al. [106] measured heart rate during maximum exercise testing of 225 subjects (4 to 33 years of age), and found significant correlation (r=0.43), but also considerable variation (standard deviation =  $\pm 11$ beats/min). The most common citation of age-predicted maximal heart rate is derived from Fox et al. [117], who estimated the equation from 35 data points and no regression. Fox et al. states:

"…no single line will adequately represent the data on the apparent decline of maximal heart rate with age. The formula maximum heart rate=220–age in years defines a line not far from many of the data points…" [117]

Tanaka et al. [118] conducted a meta-analysis of studies that have examined changes in maximal heart rate with age. They found that the ACSM equation (220-age) was accurate in the early decades (20 and 30), but it under-estimated maximal heart rate in older individuals. These variations were not affected by gender or training state. In fact, Robergs and Landwehr [116] examined the origins of the 220-age and determined that it was based more on subjective and anecdotal information rather than on rigorous research. Tanaka et al. [118] suggested 208 – 0.7\*age in years as a more accurate estimate of heart rate based on age.

Rating of perceived exertion (RPE) is a subjective measure of exercise intensity [1]. It was developed by Gunnar Borg, a Swedish psychophysiologist [119]. The scale takes into account personal fitness level, environmental conditions, and general fatigue levels [120]. Ratings of perceived exertion can be affected by psychological factors, mood states, environmental conditions, exercise modes, and age, which can reduce its practicality [121].

Based on ACSM guidelines [104], RPE must be standardized to minimize misinterpretation. The following paragraph should be read to the participant:

"During the exercise test we want you to pay close attention to how hard you feel the exercise work rate is. This feeling should reflect your total amount of exertion and fatigue, combining all sensations and feelings of physical stress, effort, and fatigue. Don't concern yourself with any one factor such as leg pain, shortness of breath or exercise intensity, but try to concentrate on your total, inner feelings of exertion; be as accurate as you can" [104]

 Two RPE scales are commonly used today: the original category scale, which goes from 6 ("very, very light") to 20 ("very very hard"); and the revised or category ratio scale which goes from 0 ("Nothing at all") to 10 ("extremely strong"). A study of 28 healthy young adults showed that the Borg scale correlated closely ( $r=0.90$ ) with VO<sub>2max</sub> percentages and max heart rate percentages [122]. This data was repeated again at high workloads with equivalent success [123]. Typically, the criterion for RPE is >18 for original category scale, or >9 for the category ratio scale.

In summary, few of the criteria listed here can absolutely verify, beyond the shadow of a doubt, that a participant has attained  $VO_{2max}$ . Thus, over the past 5-10 years, exercise physiologists have begun to move away from using the aforementioned primary and secondary criteria. Presently, the only true method of verifying that  $VO<sub>2max</sub>$  has been attained is to retest the person at a supramaximal work rate. Rossiter et al. [12] did precisely this by having subjects perform a CGXT on the cycle ergometer, allowing a short 5-minute recovery, then taking subjects to 105% of the peak work rate achieved during the CGXT. This verifies the attainment of VO<sub>2max</sub> using the same principle as a VO<sub>2</sub> "ceiling" during a DGXT. Both of these show that further increases in work rate will not yield further increases in  $VO<sub>2</sub>$ .

### The  $VO<sub>2</sub>$  Plateau

## Validity of the VO2 plateau concept

Despite the continued use of VO<sub>2</sub> plateau as a criterion for the attainment of VO<sub>2max</sub>, several recent studies have shown that lack of  $VO<sub>2</sub>$  plateau prevalence does not invalidate a CGXT.

The low prevalence of  $VO<sub>2</sub>$  plateau in CGXT's is best shown in a study performed by Day et al. [10] . Seventy-one male subjects aged 19 to 61 performed a CGXT bike incremental ramp exercise test. The intensity was increased by 5W every 12, 15, or 20 seconds—depending on the fitness of the subject—to volitional exhaustion (unable to maintain cadence of ≥60 rpm).

After completing the CGXT, 38 of the 71 subjects completed a constant-load exercise at 90% of the peak work rate achieved in the CGXT. Then, 6 of the 38 subjects completed 5 more constant-load tests at varying submaximal, maximal, and supramaximal workloads. To test for the presence of a VO<sub>2</sub> plateau, a linear regression was fit to the "main portion" VO<sub>2</sub> response, which excluded the first 4 minutes and last 3 minutes. The first 4 minutes were excluded because the beginning of a CGXT demonstrates a steep increase of  $VO<sub>2</sub>$  from rest (called the fast portion), and including it in the VO<sub>2</sub> response creates an artificially high VO<sub>2</sub> increase. The last 3 minutes were considered the "tail portion" to appraise the  $VO<sub>2</sub>$  plateau.

A line of best fit was drawn for the main portion and extended through the tail portion. A VO<sub>2</sub> plateau was present if the tail portion deviated from the line of best fit enough to consider the tail portion significantly different than the main portion. The results showed that of the 71 participants, 12 individuals (17%) achieved a plateau, 40 individuals (56%) showed no linear variance, and 19 individuals (27%) showed an increase in  $VO<sub>2</sub>$  uptake. In addition, when 38 of the subjects performed a constant load test-to-exhaustion at 90% of the peak work load they showed a "ceiling" effect, in which their highest VO<sub>2</sub> did not exceed their VO<sub>2peak</sub> from their cycling test.

Following up on this study Rossiter, et al. [12] again examined the validity of the VO<sub>2</sub> plateau. Seven male adults performed a CGXT cycle ergometry protocol (increase 20W/min) until exhaustion, recovered for 5 minutes at 20W, then performed a constant load test at 105% of the final work rate achieved in the ramp incremental protocol. By doing this, they tested whether the lack of a plateau in the CGXT would yield a difference in peak  $VO<sub>2</sub>$  between

constant load and ramp incremental tests. Despite the fact that none of the seven participants achieved a VO<sub>2</sub> plateau by methods previously described [10], VO<sub>2peak</sub> was not significantly different between the CGXT and 105% constant load tests. The authors concluded that the presence of a plateau is not necessary to attain the  $VO<sub>2peak</sub>$ , showing that a plateau is not necessary for reaching VO<sub>2max</sub>.

Criticism by Noakes of the VO<sub>2</sub> plateau phenomenon and its implications

Since 1987, Timothy Noakes M.D. has been critical of the entire concept of VO<sub>2max</sub> as it

was understood by Hill and Lupton, citing the low prevalence of  $VO<sub>2</sub>$  plateau as a reason for

this.

His book Lore of Running [20] has criticized both the foundation and concept of VO<sub>2max</sub>.

 "On the basis of certain assumptions that were probably incorrect [21], Hill and Lupton postulated that shortly before the individual reached maximum work capacity, or running speed, the rate of oxygen consumption reached a plateau and did not increase further. Although able to exercise a little harder the athlete took up no more oxygen. At this point the athlete was said to have reached maximum oxygen consumption…However, modern studies suggest that only approximately half of all tested subjects show true plateaus in oxygen consumption during maximal exercise; in the remainder no such plateaus are present, and the factors that determine these subjects' maximal exercise performances are presently unclear. I have suggest that factors related to muscle not the cardiovascular system (and thus not oxygen transport) limit the maximal exercise performance of these persons[21]…This would explain the observations by Lydiard and Gilmour [124] and Pirie [125] that the distance runners who are fastest over the shortest distances will also be the fastest at all longer distances, including the ultramarathon… Obviously, these ideas are quite heretical, and it will be some time before they are either proven to be incorrect or are accepted by the international community of exercise physiologists"

Since then, Noakes has continued to criticize the  $VO_{2max}$  paradigm of Hill and Lupton [5] in his scientific publications. His maximal exercise testing of 43 marathon and ultra-marathon runners showed that peak treadmill velocity predicted running performance better than VO2max [19], suggesting that  $VO<sub>2max</sub>$  was irrelevant altogether in predicting performance. In 1996, Noakes delivered the J.B. Wolffe Memorial lecture [18], and again criticized Hill and Lupton's work:

"Hill and Lupton concluded that the constant VO2 they measured at the fastest running speed (16km/h in Fig. 1) represented an apparent, not a true, steady state. The basis for this conclusion was a circular argument based on Hill's subconscious model explaining fatigue during exercise (Fig. 2). For Hill began with the subliminal premise that fatigue during exercise is caused by an oxygen deficiency…Not only did Hill et al. [5] fail to measure concurrently either the oxygen debt or muscle or blood lactate levels during these subsequent studies, they also failed to subject their hypothesis to the accepted process of refutation. For the next logical study would have been to measure Hill's VO2 when he ran at a speed faster than 16km/h. Their hypothesis would have been supported if the VO2 at that higher speed was either the same or lower than that measured at 16 km/h"

Afterwards, Noakes entered into a debate concerning an article published by Bassett and Howley [6] titled, "Maximum oxygen uptake: 'classical' versus 'contemporary' viewpoints". In it, he continues to criticize the work of Hill and Lupton [22]. In 2008, he published two works that call into question both the work of Hill and Lupton, and the concept of  $VO_{2max}$  [23-24]. The articles titled "Testing for maximum oxygen consumption has produced a brainless model of human exercise performance" [24] and "How did A V Hill understand the VO<sub>2max</sub> and the 'plateau phenomenon'? Still no clarity?" [23]

At this point, Noakes introduced a new theory to refute the A. V. Hill paradigm: the central governor theory (CGT) [23]. Previously he had argued that peak treadmill velocity (PTV) predicted marathon performance better than  $VO_{2max}$ , and thus factors that affect PTV (including the percentage of type II muscle fibers) were the real determinants of  $VO<sub>2max</sub>$ . If only an individual could run faster, Noakes argued, his  $VO<sub>2max</sub>$  would go higher. In 2008, Noakes began to propose the CGT, which he claimed incorrectly had first been proposed by A.V. Hill. (In fact, he called it the Noakes-Hill theory.) The CGT states that aerobic performance is limited by the central nervous system's recruitment of skeletal muscle fibers, rather than by cardiac factors previously explained. The purpose of the central governor is to prevent ischemia of the heart during maximal exercise.

Again in 2008, Noakes published a letter to the editor of Medicine and Science in Sports and Exercise [25] proposing his CGT in response to a study showing that supramaximal tests yielded identical VO<sub>2max</sub> values to a CGXT [126], Noakes stated that even at 130% of VO<sub>2max</sub> the muscular forces generated and muscle fiber recruitment are less than those generated by a maximal voluntary contraction. Thus, he reasons, our performance is regulated by a "central governor" that limits motor unit recruitment, rather than by the maximal cardiac output. Although Noakes is correct in the assumption that forces generated during maximal volitional contraction are higher than those created at 100% of  $VO<sub>2max</sub>$ , the primary fuel during a single contraction at 100% MVC is ATP/CP. This energy system is intrinsically different than the systems that are used during supra-maximal, high-intensity exercise at 110-150% VO<sub>2max</sub>.

Later in 2008, Noakes published another letter to the editor of European Journal of Applied Physiology [29]. Noakes claims that the purpose of the central governor is not to protect the heart from ischemia (despite previously stating that was the reason for a central governor). He then finished by saying, "The point of course is that the brain could always choose to continue the exercise bout by increasing the extent of muscle recruitment until 100% of all available motor units were active. But clearly it chooses not to adopt that strategy". Noakes finished the year with another letter to the editor of European Journal of Applied Physiology [32] again describing his view of the central governor.

In 2009, the Journal of Applied Physiology published a point-counterpoint debate between Noakes [30] and BT Ekblom [127] with commentary by RJ Shephard [128], WG Hopkins [129], C. Foster and A. Lucia [130], JA Calbet [131], J. Gonzalez-Alonzo, SP Mortensen [132] and numerous letters written by other researchers. Noakes titled his piece "Maximal oxygen uptake is/is not limited by a central nervous system governor".

Noakes claimed that, contrary to popular teaching, maximal oxygen uptake is limited by a "central governor" rather than the ability of the cardiac system to deliver oxygen to working muscles. In it, Noakes claims that a central governor prevents the body from reaching maximal exertion because doing so would cause myocardial damage, which is in direct contradiction to his letter to the editor in the previous year [29], which stated "The [central governor model] does not predict, as Brink-Elfedoun et al. [133] incorrectly claim: 'To prevent the heart muscle from becoming ischemic…". He finished the point/counterpoint still espousing his CGT [31]. Ekblom's elegant and short final argument in the debate stated,

<sup>&</sup>quot;If the aim of a Central Governor mechanism was to prevent the heart from myocardial ischemia during maximal exercise, why use a complicated system of "peripheral" regulations—a system we know can fail in different environments? A more secure system for prevention would have been concentrated directly on the initiation of the heart beat. There are direct and indirect possibilities through the autonomic nervous system or even an intramyocardial feed back system. But there are no such signs. Therefore, the overwhelming results from many observations are that the healthy heart

is the bottle neck for  $VO_{2max}$  with good reserve capacity of the heart muscle without risk of heart failure and hypoxic damage."[127]

Aside from Noakes and Marino, all other respondents to the point/counterpoint refuted the CGT and its tenets. Noakes has continued to publish reviews and letters endorsing the CGT, with five articles published between 2009 and 2011 [26, 28, 134-135]. In addition to directly supporting the CGT, he has been listed as a secondary author on other papers that endorse this idea.

In 2009, Swart et al. [134] performed exercise tests with methylphenidate, and found that those who were administered methylphenidate (a psychostimulant used in treatment of ADHD) cycled 32% longer before achieving fatigue (defined as cycling at 70% of original power output). They concluded that exercise is "highly regulated" by the central nervous system.

Later in 2009, Noakes co-authored another paper published in the British Journal of Sports Medicine, concluding that mechanisms of peripheral fatigue could not be explained by physiological factors [135], hinting at an inhibition from the central nervous system.

In a reply to an article by Roy Shephard [26], Noakes stated "Furthermore, to advance the case for the [central governor model], I now include evidence from more than 30 studies, which, in my opinion, can only be interpreted according to a model of exercise regulation where the [central nervous system], acting in an anticipatory manner, regulates the exercise behavior by altering skeletal muscle recruitment…". Our body will react "in anticipation" to expected workloads, such as athletes who—despite training daily under high heat conditions fail to present with heat stroke more often. He cites the several psychological factors that can affect performance of an athlete. Noakes criticizes the science community as a whole of being

unwilling to accept his ideas, and he believes that over time his work will be accepted. He claims, "First they say that what you claim is wrong. Second, they say that what you claim is true but irrelevant. Finally they say that what you say is true but 'we have always known it' ".

His most recent work pushes to abandon the entire A.V. Hill paradigm altogether [27]. His results are summarized in the following paragraph.

"The cardinal weakness of the Hill model is that it allows no role for the brain in the regulation of exercise performance. As a result, it is unable to explain at least 6 common phenomena, including (i) differential pacing strategies for different exercise durations; (ii) the end spurt; (iii) the presence of fatigue even though homeostasis is maintained; (iv) fewer than 100% of the muscle fibers have been recruited in the exercising limbs;  $(v)$  the evidence that a range of interventions that act exclusively on the brain can modify exercise performance; and (vi) the finding that the rating of perceived exertion is a function of the relative exercise duration rather than the exercise intensity"

Over the past 15 years, Noakes has been highly critical of the  $VO_{2max}$  paradigm of Hill and Lupton [5]. Despite a substantial body of scientific evidence that supports the classical model, Noakes rejects the central tenets of the  $VO<sub>2max</sub>$  model.

#### VO2 Plateau: DGXT versus CGXT Protocols

Older studies in the 1920's to 1960's [5, 33, 81-82] established the VO<sub>2</sub> plateau criterion

based on the results of DGXTs, and these studies showed that the vast majority of subjects

displayed a  $VO<sub>2</sub>$  plateau. More recent studies, however, have used CGXTs and have found that

only about one -quarter to one-half of subjects display a VO2 plateau.

Taylor, Henschel, and Buskirk [33] completed a DGXT on a large sample, and established the criterion measure for  $VO<sub>2</sub>$  plateau. Their protocol used a series of discontinuous 3-minute runs, and they only collected expired air between 1:45 and 2:45. Each time, the participant ran at steeper inclines until they could not maintain exercise for the full 3-minute duration or they demonstrated a  $VO<sub>2</sub>$  plateau (when the treadmill grade was increased, they failed to demonstrate an increase of > 150 ml/min compared to the previous stage,). Of 115 participants, 108 demonstrated a  $VO<sub>2</sub>$  plateau using this DGXT.

Earlier studies [5, 33, 81-82] used a DGXT, consisting of several constant-load tests over a number of days. With a DGXT, the participant runs at a constant speed until steady state is achieved, and once that steady state is achieved, that is considered the  $VO<sub>2</sub>$  demand for that workload. However, at supra-maximal speeds the subject will only attain an "apparent steady state"; the  $VO<sub>2</sub>$  levels off but the oxygen requirement exceeds the oxygen consumption so metabolic steady-state cannot be achieved. By plotting the workload on the X-axis, and the corresponding  $VO<sub>2</sub>$  on the Y-axis, you create a data point. After resting for a period of time, the participant will perform another constant load test at a higher intensity. This continues until the participant performs a test where the workload increases but the VO<sub>2</sub> does not. Taylor et al. [33], which first established the plateau criterion, ran subjects at 7 mph for 3 minutes on the first visit, then the next visit would increase the incline by 2.5%. The incline would be increased for every visit until the subject showed a VO<sub>2</sub> increase that was less than 150 ml\*kg<sup>-1\*</sup>min<sup>-1</sup> or could not complete the full 3 minutes, assuming that to be the stage to yield a  $VO<sub>2</sub>$  plateau.

Earlier tests used a DGXT because a CGXT would have been nearly impossible to perform with equipment at that time. Hill and Lupton [5] used a grass track with a timer calling out splits to the subject, relying on the runner's experience to maintain a certain average pace over the bout. Without a treadmill, they could not gradually increase the velocity every minute, as required by a CGXT. In addition, metabolic carts were not available. So in order to examine the  $VO<sub>2</sub>$  kinetics during the run, the participant would have to repeat the run several times with one Douglas bag for a 30-second portion of the run. For example, when running at 9.9 miles per hour for 8 minutes, the subject would run at this speed and open up a valve to breath into the Douglas bag for 0:15 to 0:45, before switching the valve to breath into the atmosphere. The participant would have to repeat the run, but open the valve from 1:15 to 1:45. This was repeated several times until enough data could give an accurate picture of the  $VO<sub>2</sub>$  kinetics.

Taylor et al. [33] had access to a motorized treadmill, and even with this tool, performing a CGXT still would have been challenging and time-consuming. They measured a single Douglas bag for each constant-speed run (from 1:45 to 2:45). With each of their participants performing 2 to 4 constant load runs, this demanded two to four bags per subject. To perform a CXGT would have required 1 Douglas bag for each stage, which could be as few as 8, or as many as 20 depending on the protocol. A CGXT could require as many as 12 different stages, depending on the fitness of the subject. That would demand 12 different Douglas bags to be used for a single test, and 12 different gas analyses, which would be incredibly time consuming for results from a single test. As a result, the use of the DGXT during this time was

40

not necessarily because it was the most valid or appropriate method, but rather because it was easier to do with the equipment available during that time.

Once the methods and technology (treadmills, oxygen measuring devices, and other exercise equipment that could have an adjustable workload) became available, tests were done to compare the validity of the DGXT to CGXT [13, 86-91]. Since it was shown that they both yield the same values for  $VO_{2max}$ , researchers switched over to using a CGXT because it takes far less time and fewer visits to complete. Reasearchers failed to realize that notion of a  $VO<sub>2</sub>$ plateau developed by Taylor et al. [33] on a DGXT cannot be applied to the CGXT protocols.

#### The origin of the VO<sub>2</sub> plateau from the work of Hill, Lupton, and Taylor et al.

The first recorded instance of  $VO_{2max}$  testing came from Hill and Lupton [5] when Hill described the concept of  $VO<sub>2max</sub>$  by saying, "however much the speed be increased beyond this limit, no further increase in oxygen intake can occur". This described the notion of an upper limit (or "ceiling") during a DGXT. Although Hill and Lupton never tested this hypothesis (because they did not push the participant to supra-maximal speeds), it was inferred that running at higher speeds would not increase the  $VO<sub>2</sub>$  once you reached  $VO<sub>2max</sub>$ . Hill and Lupton never used the word "plateau" to describe the phenomenon, and "ceiling" is a more appropriate term to use to describe the upper limit on  $VO<sub>2</sub>$  on a DGXT.

Based on the work by Taylor et al. [33],  $VO<sub>2</sub>$  plateau was established as a criterion measure for attainment of VO<sub>2max</sub> on DGXT protocols. Given the high prevalence of the plateau among Taylor et al.'s population, it was reasonable to believe that the presence of a plateau could be used to verify the attainment of a maximal effort. Thus, the  $VO<sub>2</sub>$  plateau criterion was established for DGXT.

## The difference between a  $VO<sub>2</sub>$  plateau and a  $VO<sub>2</sub>$  ceiling

The DGXT and CGXT are very different exercise protocols. Normally, the result of a DGXT is illustrated using a graph of time on the x-axis vs.  $VO<sub>2</sub>$  on the y-axis. For each constant-speed stage, a line graph is constructed, and several of these are overlaid onto the same graph. Once the subject reaches a work-rate where increases in exercise intensity fail to elicit any further rise in VO<sub>2</sub>, they have hit their upper ceiling (VO<sub>2</sub> max). Figure 1 represents an example of this.

In contrast, a CGXT does not rely on constant load tests, but rather a single incremental exercise bout. The data are normally illustrated using a graph of work rate or speed on the xaxis, versus VO2 on the y-axis. The lack of a VO<sub>2</sub> plateau does not necessarily mean that the participant has chosen to terminate the test prematurely (i.e. before attaining  $VO_{2max}$ ), and thus invalidate the attainment of  $VO_{2max}$ . This is why Day et al. [10], after finishing the CGXT, had the participant complete another constant-load test at a work rate 105% of the highest work rate achieved during the CGXT. If the VO<sub>2</sub> on the supra-maximal effort failed to elicit a higher VO<sub>2</sub>, then they could correctly conclude that the  $VO_{2max}$  had been reached. Rossiter et al. [12] repeated these results in 7 participants who, despite showing no  $VO<sub>2</sub>$  plateau on a CGXT, failed to increase VO<sub>2</sub> at a work rate 105% of the highest one achieved during the CGXT.

The key difference in CGXT and DGXT protocols is the capacity to work at supramaximal intensities during the test. During a CGXT, the participant is already fatigued when they approach  $VO_{2max}$  and therefore are unable to continue running at supramaximal speeds. Contrarily, during a DGXT, the participant begins each run without fatigue and can run at supramaximal speeds long enough to achieve steady-state VO<sub>2</sub> at VO<sub>2max</sub>. [insert chart showing an overlay of CGXT and DGXT]. In the example of subject X, the oxygen requirements at certain speeds were identical, but the DGXT continued to higher speeds than the DGXT. As a result the plateau phenomenon and ceiling phenomenon are different.

Whereas a CGXT will terminate at or near  $VO_{2max}$ , a DGXT actually attempt to measure the VO<sub>2</sub> response at intensities beyond VO<sub>2max</sub>. The ceiling of the DGXT accounts for supramaximal intensities that will fail to increase the  $VO<sub>2</sub>$  with increased work rate, but the CGXT plateau occurs near  $VO<sub>2max</sub>$  without necessarily reaching supramaximal speeds.

#### Chapter 3

#### Abstract

Maximal oxygen uptake (VO<sub>2max</sub>) is regarded as the gold standard for assessing aerobic fitness. In 1923, Hill et al. proposed that  $VO_{2max}$  represents the maximal ability of the body to take in and consume  $O_2$  during strenuous exercise. Recently, however, controversy has arisen over the issue of whether a leveling off, or "plateau" in VO<sub>2</sub> is necessary to verify attainment of VO<sub>2max</sub>. **Purpose:** To compare two different  $VO_{2max}$  protocols and determine if both protocols show direct evidence of an upper limit on  $VO<sub>2</sub>$ . Methods: Nine runners (18-35 years old) completed a continuous graded exercise test (CGXT), followed by a discontinuous graded exercise test (DGXT). The CGXT consisted of gradually increasing treadmill running speed to the point of volitional exhaustion; the highest speed attained was labeled the peak treadmill speed. Over the next several days, participants ran at 80%, 90%, 100%, 105%, and 110% of peak treadmill speed for 10 minutes, or until volitional exhaustion was reached. Results: All participants (n=9) achieved a "VO<sub>2</sub> ceiling" (or upper limit) on the DGXT, while only 44% (n=4) achieved a "VO<sub>2</sub> plateau" on the CGXT. There was no significant difference between the  $VO<sub>2max</sub>$  obtained from a CGXT (57.4  $\pm$  2.6 mL\*kg<sup>-1</sup>min<sup>-1</sup>) and DGXT (60.0  $\pm$  3.1 mL\*kg<sup>-1</sup>min<sup>-1</sup>). There was no difference between oxygen uptake measured at 90%, 100%, 105%, and 110% of PTV (p>0.05). However, the highest VO<sub>2</sub> recorded at 80% PTV was significantly lower than that recorded at all other velocities ( $p$ <0.05). Conclusion: The VO<sub>2</sub> ceiling effect on a DGXT is inherently different than the VO2 plateau effect on a CGXT. In this study, a ceiling was always seen on the DGXT, but a plateau was not always seen on the CGXT.

#### Introduction

Maximal oxygen intake (VO<sub>2max</sub>) is defined as the highest rate at which oxygen can be taken in and consumed by the body during strenuous, dynamic exercise [1].  $VO<sub>2max</sub>$  is widely accepted as a measure of aerobic fitness and has been linked to numerous health outcomes [2- 4]. In addition,  $VO_{2max}$  is an important determinant of athletic performance in middle- and longdistance athletic events. For these reasons,  $VO_{2max}$  is widely used in the field of exercise science.

Historically, the concept of  $VO<sub>2max</sub>$  can be traced back to the work of Hill and Lupton [5]. Their VO<sub>2max</sub> paradigm has four central tenets: (1) there is an upper limit to oxygen intake; (2) there are inter-individual differences in VO<sub>2max</sub>; (3) a high VO<sub>2max</sub> is a pre-requisite for successful distance running; and (4)  $VO_{2max}$  is primarily limited by the circulatory and/or respiratory systems [136]. In addition, their model states that it is possible to exercise at work rates in excess of  $VO<sub>2max</sub>$ , by increasing one's reliance on anaerobic metabolism. Thus, once the participant reaches a work rate that elicits  $VO_{2max}$ , any further increase in work rate will not yield a further increase in VO<sub>2</sub>. This is often referred to as the VO<sub>2</sub> plateau phenomenon [5, 9-10, 12-14, 33, 137-144].

For several decades, a VO<sub>2</sub> plateau was used as a criterion for the attainment of VO<sub>2max</sub>. However, the use of a  $VO<sub>2</sub>$  plateau as a criterion during continuous graded exercise tests (CGXT) has become problematic. Although several sources list the  $VO<sub>2</sub>$  plateau as a primary criterion for attainment of VO<sub>2max</sub> [16-17, 23, 33, 83-84, 93, 106], a review of studies in which VO<sub>2max</sub> was measured showed that most individuals do not show evidence of a  $VO<sub>2</sub>$  plateau at the end of a CGXT [7, 9, 13, 15, 137, 140, 145-146].

In addition, some researchers have concluded that the low prevalence of  $VO<sub>2</sub>$  plateau indicates that entire  $VO_{2max}$  paradigm proposed by Hill and Lupton [5] is incorrect [18, 20-23, 147-148]. Noakes et al. [18, 20, 22-27] has argued that since some individuals do not show evidence of a plateau during maximal exercise tests, then it cannot be stated that they have truly attained their  $VO_{2max}$ . This has led them to speculate that if only these individuals could run faster, their  $VO<sub>2</sub>$  would reach higher levels. As a consequence, Noakes [18-20, 22] has stated that  $VO_{2max}$  is actually limited by the physiological factors that determine maximal running velocity (such as muscle fiber type) or central nervous system factors, rather than the physiological factors that limit oxygen transport from the lungs to the mitochondria.

The classic studies of Hill and Lupton [5] introduced the concept of  $VO<sub>2max</sub>$ , but they used different maximal exercise testing protocols than those used currently.  $VO<sub>2max</sub>$  studies in the 1920's used a discontinuous graded exercise test (DGXT), which consisted of several constant load tests performed to exhaustion and separated by lengthy rest periods. In contrast, modern day researchers typically use a CGXT, in which the exercise intensity is gradually increased each minute, until volitional exhaustion is reached. The DGXT protocols yield a higher prevalence of VO<sub>2</sub> plateau [5, 33, 83-84, 106] than modern CGXT protocols [7, 9, 13, 15, 137, 140, 145-146].

The purpose of this study was to re-examine the classic work of Hill and Lupton [5], using modern-day techniques. We planned to compare their DGXT protocol to a modern-day CGXT protocol. There were 3 hypotheses in this study:

1. There is not a statistically significant difference between  $VO<sub>2max</sub>$  values achieved on a CGXT and DGXT protocol.

2. Having subjects run at supramaximal speeds does not result in further increases in VO<sub>2</sub>, beyond those seen at the end of a CGXT.

3. All participants display a  $VO<sub>2</sub>$  "ceiling" (or upper limit) on a DGXT, but not all participants display a plateau on a CGXT.

#### Methods

#### Participants

Nine runners (7 men, 2 women) were recruited from Knoxville, TN to participate in this study. Eligibility criteria included: 18-35 years of age, running at least 15 miles per week on average over the past 6 months, and low risk for cardiovascular disease as defined by ACSM/AHA [104]. Prior to participating in the study, participants signed an informed consent form that was approved by the university's Institutional Review Board (Appendix A). All testing was completed in the Applied Physiology Laboratory in the Health, Physical Education, and Recreation Building on the University of Tennessee campus.

### Exercise Protocol

During the initial visit, the participant's weight and body fat were assessed using a Tanita BCA-418 bioelectrical impedance analyzer (Tanita, Chiba, Japan) in the morning prior to exercise. All but 1 participant ate nothing the morning of the initial visit. Weight was measured then recorded to the nearest 0.1 kg and body fat was estimated to the nearest 0.1%. Height was measured to the nearest 0.1 cm using a wall-mounted standiometer. Both weight and height were measured in light running clothing, without shoes.

Participants were instructed not to exercise for 24 hours prior to each test. They were informed that testing would be vigorous and asked to adjust their personal workouts accordingly. All testing was completed on calibrated treadmill (Quinton Q65 series 90, Seattle, WA, USA). For all tests, the subject first straddled the treadmill belt while the speed was brought up to the appropriate setting. Once up to speed, the participant straddled the belt for 3 minutes while standing oxygen consumption was measured. Once testing began, the participant suspended himself or herself above the belt using the handrails. The participant then lowered himself or herself onto the belt and began running, releasing the hand rails once he or she felt comfortable with the speed. The bout started when the participant's foot touched the treadmill.

48

On the first day, participants completed a CGXT. The CGXT protocol began with treadmill running at 9.7 kilometers per hour (kph), and speed was increased by 0.8 kph each minute until the participant reached volitional exhaustion. Respiratory gas exchange was measured using an Oxycon Mobile [Viasys Healthcare, Hochberg, Germany] device. The Oxycon Mobile is a portable breath-by-breath system which can accurately determine the  $V_{E}$ , VO<sub>2</sub>, VCO<sub>2</sub>, FEO<sub>2</sub>, and FECO<sub>2</sub> of individual breaths [149-150]. The device was chosen for its ability to measure VO<sub>2</sub> responses during brief high-intensity exercise. Participants wore a facemask (model 7450 V2 Mask, Hans Rudolph, Shawnee, KS) that was held tight by a head strap. The facemask fits over the nose and mouth and allows for measurement of ventilation and gas fractions. When the mask was placed on the participant, the participant would cover the opening with his or her palm and exhale normally to make sure there were no gas leaks during exhalation. Participants were given verbal encouragement to continue as long as possible, and were able to see their speed and time spent exercising on the treadmill. The highest speed maintained for 40 seconds or more on the CGXT was considered the peak treadmill velocity (PTV). VO<sub>2max</sub> was determined from the highest measured VO<sub>2</sub> for any 15-second period.

Three more days of testing were performed in a randomized order. On these testing days, the participants ran at either (a) 90% and 100%, (b) 105%, or (c) 110% of PTV on the CGXT. Testing days were separated by 1 to 6 days of rest. Before each of these trials, participants warmed up by running at 80% of PTV for 10 minutes, followed by a 5-minute cooldown at a self-selected speed and 10 minutes of rest.

The 90% and 100% PTV trials began with the previously described warm-up and cooldown and rest. The participant ran at 90% of PTV for 10 minutes, followed by a 15-min cooldown and rest period. On the same day, the participant then completed a second warm-up run at 80% of PTV for 3 minutes to sufficiently warm up the body without exhausting it for the final bout. After 5 minutes of cool-down and rest, the participant ran at 100% of PTV for 10 minutes or until volitional exhaustion was reached.

The 105% PTV trial began with a warm-up, then after a 5-minute cool-down and rest, running at 105% PTV until volitional exhaustion. The 110% PTV trial was identical, except it was run at 110% of PTV.

Participants were allowed to view both speed and duration during the tests, and the researchers provided verbal encouragement to give a maximal effort. At the point of volitional exhaustion, the participant grabbed the handrails and straddled the treadmill belt while the speed was lowered to 2.0 mph. The participant then completed a cool-down at a self-selected pace.

## VO2 Plateau Determination

For the CGXT, we checked for a VO<sub>2</sub> plateau by graphing treadmill speed against VO<sub>2</sub>. For plateau determination, the first two velocities (6.0 and 6.5 mph) were excluded. If the participant failed to complete a full minute at the final velocity, the  $VO<sub>2</sub>$  measurements were excluded. Because the exercise started from rest, the initial velocities had large increases in  $VO<sub>2</sub>$  for the increases in work rate (i.e. the  $VO<sub>2</sub>$  increase from rest to 6 mph is much larger than the increase from 7 to 7.5 mph). Including them in the analysis would have yielded an artificially high VO<sub>2</sub> increase, so they were excluded. A plateau was defined by constructing a line of best fit for the graph of running speed versus  $VO<sub>2</sub>$ , excluding the first two velocities and peak treadmill velocity . We then extrapolated the line of best fit to the peak treadmill velocity. A plateau in VO<sub>2</sub> was defined as an increase in the measured VO<sub>2</sub> corresponding to less than onehalf of the "expected" increase in  $VO<sub>2</sub>$ .

## VO<sub>2</sub> Ceiling Determination

For the DGXT, the presence of a VO<sub>2</sub> ceiling was determined by examining the VO<sub>2max</sub> values for all 5 of the DGXT trials. If the VO<sub>2max</sub> of 105% and 110% of PTV were essentially the same as the VO<sub>2max</sub> measured at 100% of PTV, then this demonstrated the presence of a VO<sub>2</sub> ceiling. If the variation between these 3 values for each participant was less than 5% (i.e.- the day-to-day coefficient of variation generally associated with  $VO_{2max}$  testing [151-153], then they were considered to be essentially the same. A variation greater than 5% in these 3 values indicates that there is a difference greater than what can be attributed to day-to-day variation.

All data were analyzed using SPSS software (v16, SPSS, Insc, Chicago, IL). The VO<sub>2peak</sub>,  $VO<sub>2max</sub>$ , and RER values are presented in 15-second epochs. Comparisons between  $VO<sub>2peak</sub>$  and VO2max values for different constant speed runs and CGXT were conducted using a repeatedmeasures ANOVA to detect for significant difference between the groups. If significance was found (p<0.05), pairwise analysis with Bonferonni adjustments were made to identify which protocols or intensities yielded significantly different  $VO<sub>2peak</sub>$ , and  $VO<sub>2max</sub>$  measurements.

## Results

## Participant Description

Descriptive statistics of participants are described in Table 1.

Table 1 Physiological characteristics of participants (N=9; 7 males and 2 females)

Age (years)	Height (cm)	Weight (kg)	BMI ( $\text{kg/m}^2$ )	<b>Estimated Body</b> Fat (%)
$28.7 \pm 1.5$	$175.9 \pm 1.9$	71.6±2.8	$23.0 \pm 0.6$	14.9±1.7

\* Values are mean ± standard error; BMI = body mass index

## Continuous Graded Exercise Test

Four of the 9 participants achieved a plateau on the CGXT, as previously described.

Figure 1 illustrates an example of a participant who demonstrated a  $VO<sub>2</sub>$  plateau, while figure 2

illustrates and example of a participant who failed to demonstrate a  $VO<sub>2</sub>$  plateau.

### Discontinuous Graded Exercise Test

All 9 participants demonstrated an "upper ceiling" for  $VO<sub>2</sub>$  on the DGXT. This ceiling prevalence was significantly different from the plateau prevalence (p=0.013). The vast majority also achieved secondary criteria for attainment of  $VO_{2max}$  [101]. Eight were within 10 beats of age-predicted maximal heart rate, and 7 achieved an RER  $\geq$  1.10 during any of the constant-load tests, with 6 of them achieving both criteria.

The VO<sub>2peak</sub>, V<sub>Epeak</sub>, HR<sub>peak</sub>, and of the 10-min run at 80% of PTV were significantly lower (p<0.05) than all other constant speed runs, indicating that this was clearly a sub-maximal effort. The VO<sub>2peak</sub>, V<sub>Epeak</sub>, and HR<sub>peak</sub> of all other constant speed runs were not significantly different from each other, but were all higher than the values achieved at 80% of PTV . The 105% and 110% of PTV runs had significantly higher RER<sub>peak</sub> than 80%, 90%, and 100% of PTV, but no other significant differences in these variables were found. These results are displayed in Figures 3, 4, 5, and 6.

The prevalence of the achievement of criteria for both the DGXT and CGXT are presented in Table 2.

53



# Table 2. Achievement of criteria on CGXT and DGXT

Duration

Among the constant-speed tests, the duration of the runs at 80% of PTV and 90% of PTV did not differ from each other, but they were significantly less (p<0.05) from all other run durations. None of the bouts at 80% of PTV, as well as 3of the bouts at 90% PTV, were not to carried out to volitional exhaustion, but were terminated at 10 minutes. The durations of the 100% of PTV run and 105% of PTV run were not significantly different from each other, but they were significantly different from all other runs (p<0.05). The duration of 110% of PTV was significantly shorter than all other runs. These results are displayed in Figure 7.

# Time course of  $VO<sub>2</sub>$  response

The time course of the  $VO<sub>2</sub>$  response for each of the constant-speed runs for each subject can be seen in Figures 8 though 16. Comparisons of the mean values for the CGXT and DGXT are

shown in Table 3.





All values expressed as mean ± standard error

\*Significantly different from CGXT (p=0.011)

For the DGXT, the VO<sub>2max</sub>, V<sub>Emax</sub>, and RER<sub>max</sub>, we took the highest values reached on any

of the 5 DGXT bouts to represent the maximal values for the test.

There was no significant difference in the  $VO<sub>2max</sub>$  between the CGXT or DGXT. The CGXT and DGXT also did not differ on RER<sub>max</sub> maximum heart rate, or maximum heart rate expressed as a percentage of age-predicted maximum. However,  $V_{Emax}$  was significantly higher on the DGXT compared to the CGXT.

#### **Discussion**

All of the participants demonstrated a  $VO<sub>2</sub>$  ceiling (i.e. an upper limit on  $VO<sub>2</sub>$ , with increasing running speeds) during the DGXT. However, only 44% of them showed a VO<sub>2</sub> plateau during the CGXT, despite the fact that there was no significant difference between the  $VO<sub>2max</sub>$ values measured on both protocols. Thus, while the  $VO<sub>2</sub>$ </sub> ceiling on a DGXT appears to be a valid criterion for the attainment of  $VO_{2max}$ , the lack of  $VO_2$  plateau on a CGXT does not invalidate the  $VO<sub>2max</sub> obtained during such a test.$  The presence of the  $VO<sub>2</sub>$  ceiling in the DGXT shows that, contrary to what has been hypothesized by Noakes [18], VO<sub>2</sub> will not continue to increase indefinitely as velocity increases.

Our results are in close agreement with those of Snell et al. [154]. In their study, 52 participants (36 men, 16 women) performed a treadmill CGXT, starting at 8 or 9 miles per hour with 0% grade. The treadmill grade was then increased by 2% every 2 minutes until volitional exhaustion ensued. The participants returned for a second day of testing where they ran at the same velocity as on the CGXT but with an additional 8% increase in the treadmill incline. The results showed no difference between the  $VO<sub>2</sub>$  of the CGXT and the brief, supramaximal test. Thus, they concluded that, on average, CGXT and DGXT protocols yield the same values for VO<sub>2max</sub>. They also confirmed A.V. Hill's hypothesis that at high exercise intensities eventually the VO2 reaches a maximal value, beyond which no effort can drive it.

Stamford's 1976 study [86] was similar to that of Snell et al. [154] and yielded similar results. Ten male subjects completed a DGXT, 2 CGXTs, and 2 constant load tests. The DGXT

consisted of running at 7.0 mph for 3 minutes, resting for 10 minutes, and then increasing the treadmill incline by 2.5% for each successive 3-minute bout (with 10-minute rests in between). The test continued until the subject could not complete the full 3 minutes, and the final completed stage was considered the maximal work rate. The CGXT was identical to the DGXT in work rate increments, but each stage lasted 2minutes instead of 3 and there was no rest between stages. The supra-maximal, constant load test was conducted using the peak speed and incline of the DGXT with an extra 2.5% incline added on. The results of the test showed no significant difference between the  $VO<sub>2max</sub>$  of each test whether it was DGXT, CGXT, or supramaximal.

Shephard et al. [87] confirmed these findings with a study of 24 Canadian males. The participants were administered a CGXT and a DGXT in treadmill, bicycle, and step exercise. Each test was preceded by a submaximal warm-up, which was used to estimate the maximal work rate during the exercise. In the case of the DGXT, a constant load of 110% of predicted maximum work rate was administered, and further bouts were adjusted in relation to the participant's performance. The CGXT began at 90% to 100% of maximal work rate with slight increases in work rate every 2 minutes. Regardless of the mode of exercise, there was no significant difference between DGXT and CGXT results. Thus, the concurrent validity of the DGXT and CGXT has been consistently demonstrated, across several modes of exercise and testing protocols.

The fact that a participant can show a  $VO<sub>2</sub>$  ceiling on a DGXT, but fail to show a  $VO<sub>2</sub>$ plateau on a CGXT indicates that these two events represent different phenomena. We believe

57

that subjects are more likely to show a "ceiling" or upper limit on VO2 with a DGXT, because at the beginning of each stage they are well rested and can summon anaerobic metabolism to a greater extent. This enables them to reach higher exercise intensities than they would with a CGXT, thus increasing the likelihood that they will attain supra-maximal running speeds where the oxygen requirement far exceeds the subjects  $VO<sub>2max</sub>$ . Furthermore, the lack of significant difference between the  $VO_{2max}$  of the CGXT and DGXT is consistent with previous research [13, 86-91].

Day, et al. [10] found that the  $VO<sub>2</sub>$  plateau only occurs in only a small percentage of individuals who perform a maximal CGXT on the cycle ergometer. The methods used by Day et al. [10] were meticulous and rigidly controlled. By using a small increment of increase (5W every 12-20 seconds) on a cycle ergometer, they were able to show a low-residual linear response to the exercise during the "linear portion" (they omitted the first four minutes and last three minutes of the CGXT to arrive at the linear portion). Most protocols use larger jumps in work rate at 1- or 2-minute intervals, creating more of a "step" shape rather than a "true" linear response. In addition, the breath-by-breath response, which typically exhibits a fair amount of "noise", was normalized by removing errant breaths. Without this tight control, it would be much more difficult to determine the presence of a  $VO<sub>2</sub>$  plateau. Their results clearly showed that a relatively small percentage of people (17%) demonstrate a VO<sub>2</sub> plateau on a CGXT. Most individuals demonstrate a linear increase in VO<sub>2</sub> throughout exercise (56%), while some actually demonstrate an acceleration in  $VO<sub>2</sub>$  response at the end of a CGXT (27%).

Rossiter et al. [12] showed that, in addition to the low prevalence of VO<sub>2</sub> plateau during a cycling CGXT, the lack of VO<sub>2</sub> plateau does not invalidate the attainment of VO<sub>2max</sub>. Seven subjects completed a modified cycle ergometer CGXT. They began at 20 W with an increase of 1 W every 3 seconds until volitional exhaustion was reached. After achieving exhaustion, they recovered by cycling at 20W for 5 minutes, and then completed a constant load bout at 105% of the peak work rate achieved in the CGXT. After at least 5 days of rest, the protocol was repeated, but using 95% of the peak work rate achieved during the CGXT. Results showed no significant difference between the  $VO<sub>2</sub>$  achieved during the CGXT or 105% bout. This was true despite the fact that of the 14 tests, only 2 had a "tendency toward a plateau". This study demonstrated that exercising at supramaximal rates does not increase the  $VO<sub>2</sub>$  above values measured on a CGXT. In addition, the lack of a VO<sub>2</sub> plateau on a CGXT did not invalidate VO<sub>2max</sub> values obtained during the test.

Our study confirms the findings of Day et al. [10] and Rossiter et al. [12] by demonstrating that a VO<sub>2</sub> plateau is not a necessary criterion for attainment of VO<sub>2max</sub>, and that subjecting participants to supramaximal work rates results in them achieving  $VO<sub>2max</sub>$  values that are no higher than those achieved on a CGXT.

Duncan, et al. [11] sought to determine if there was a difference in  $VO<sub>2</sub>$  plateau and ceiling prevalence between CGXT and DGXT. Their DGXT mirrored that of Taylor et al. [33], even including the collection of expired respiratory gases in Douglas bags. The participants ran at 7 miles per hour for three minutes with gas collection between 1:45 and 2:45. Each successive test was repeated at the same speed with an incline increased 2.5% from the previous bout. If

the participant showed an increase of VO<sub>2</sub> less than 150 mL/min, then they were said to have demonstrated a plateau. Duncan et al. used a CGXT that was similar to their DGXT, except that it had 1-minute stages rather than 3-minute stages, and there was no rest between each stage. Though Duncan et al. showed little difference in VO<sub>2</sub> plateau prevalence between protocols (5 of 10 for CGXT and 6 of 10 for DGXT), this is likely due to premature termination of the DGXT. Both Taylor et al. and Duncan et al. failed to show 100% plateau prevalence in the DGXT because only bouts of at least 2.75 minutes were included. Thus, if a participant's final bout was 2 minutes and 44 seconds or less it was excluded. However, in our study, 2 out of 9 (22%) participants were not able to run at 100% of PTV for 2.75 minutes, but they were still able to attain VO<sub>2max</sub> within about two minutes, which allowed them to show evidence of a VO<sub>2</sub> ceiling on the DGXT. For these individuals, the maximal DGXT protocols of Taylor et al. and Duncan et al. would not have included the data from runs at supramaximal running speeds. As a result, they would have failed to demonstrate a plateau; not because their body was incapable of reaching a plateau during a DGXT, but rather because the supramaximal bouts were excluded. This likely explains why some of the participants in the studies of Taylor et al. and Duncan et al. failed to show a ceiling effect, while all of the individuals in the present study did.

Noakes has been an ardent opponent of the  $VO<sub>2</sub>$  paradigm proposed by Hill and Lupton for over a decade. Part of his criticism was based on the fact that there is low prevalence of  $VO<sub>2</sub>$ plateau when CGXT are used. In his J.B. Wolffe Memorial lecture [18], Noakes stated,

<sup>&</sup>quot;Not only did Hill et al. [5] fail to measure concurrently either the oxygen debt or muscle or blood lactate levels during these subsequent studies, they also failed to subject their hypothesis to the accepted process of refutation. For the next logical

study would have been to measure Hill's VO<sub>2</sub> when he ran at a speed faster than 16 km/h." [18]

The major purpose of the present study was to conduct the critical experiment that would answer this question. As our results clearly showed, taking subjects to supra-maximal speeds (where the  $O_2$  requirement exceeded their VO<sub>2max</sub>) yielded a "ceiling effect" just as Hill and Lupton [5] had predicted, not a continued linear increase in  $VO<sub>2</sub>$  as hypothesized by Noakes [18]. Hill and Lupton described in  $VO_2$ -vs-running speed graph as follows, "oxygen requirement rises continuously at an increasing rate, as the velocity increases, attaining enormous values…however much the speed be increased beyond this limit, no further increase in oxygen intake can occur: the heart, lungs, circulation, and the diffusion of oxygen to the active musclefibres have attain their maximum activity."

The DGXT protocol used for the present study closely resembled that of Hill and Lupton [5]. They used a protocol designed around changes in running speed, rather than the more common practice of increasing percent grade on the treadmill [11, 33]. This procedure was used for both the CGXT and DGXT, which meant that the participants reached volitional exhaustion while running on a level surface. Nevertheless, there were some differences between our methods and those of Hill and Lupton [5]. First, rather than running around a grass track, our participants ran on a motor-driven treadmill so that the speed and environmental conditions could be controlled more easily. Second, we used a portable indirect calorimeter system that employs breath-by-breath gas analysis, rather than the Douglas bag method used by Hill and Lupton. Lastly we pushed our runners to supra-maximal speeds, while Hill and

61

Lupton did not (at least not in the experiment where they studied the time course in the VO<sub>2</sub> response to running at various speeds). Our purpose in doing this was to test the validity of Hill and Lupton's assertion that "however much the speed be increased beyond this limit, no further increase in oxygen intake can occur."

Because of the similarities between our protocol and that of Hill and Lupton, the results of this study are comparable to Hill and Lupton's work and can properly validate their results. However, it should be noted that several excellent studies have examined the time-course of the VO<sub>2</sub> response to DGXT protocols on cycle ergometers [5, 10, 12, 33, 81]. One caution, however, is that the VO<sub>2max</sub> values measured during treadmill running are generally 5-10% higher than those measured during cycle ergometry, especially for individuals not trained in bicycling. Thus, we sought to examine the  $VO<sub>2</sub>$  response to DGXT protocol during level running, to extend the work of Hill and Lupton.

 In conclusion, the results of this study reaffirm the findings of Hill and Lupton [5]. It also confirms previous work by Day et al. [10] and Rossiter et al. [12] showing that a VO<sub>2</sub> plateau is not a necessary criterion for the attainment of  $VO<sub>2max</sub>$ . Furthermore, it shows that at supramaximal running speeds,  $VO<sub>2max</sub>$  values do not exceed those achieved in a maximal CGXT, confirming the work of Snell et al. [154] and other researchers. These results strongly refute Noakes' hypothesis that running at speeds beyond 100% of PTV would yield a further increase in VO<sub>2</sub>. Hill and Lupton were absolutely correct in asserting that, "...oxygen requirement rises continuously at an increasing rate, as the velocity increases, attaining enormous values…however much the speed be increased beyond this limit, no further increase in oxygen

intake can occur: the heart, lungs, circulation, and the diffusion of oxygen to the active musclefibres have attain their maximum activity."
**List of References** 

- 1. Powers, S.K. and E.T. Howley, Exercise Physiology: Theory and Application to Fitness and Performance. 2009, New York, NY: McGraw-Hill.
- 2. Lee, I.M., C.C. Hsieh, and R.S. Paffenbarger Jr, Exercise intensity and longevity in men. The Harvard Alumni Health Study. JAMA: the journal of the American Medical Association, 1995. 273(15): p. 1179.
- 3. Paffenbarger Jr, R.S., et al., Physical activity, all-cause mortality, and longevity of college alumni. New England Journal of Medicine, 1986. 314(10): p. 605-613.
- 4. Lee, I.-M. and R.S. Paffenbarger, Associations of Light, Moderate, Vigorous Intensity Physical Activity with Longevity. American Journal of Epidemiology, 2000. 151(3): p. 293-299.
- 5. Hill, A.V. and H. Lupton, Muscular Exercise, Lactic Acid, and the Supply and Utilization of Oxygen. Quarterly Journal of Medicine, 1923. 16: p. 135-171.
- 6. Bassett, D.R. and E.T. Howley, Maximal oxygen uptake: "classical" versus "contemporary" viewpoints. Medicine and Science in Sports and Exercise, 1997. 29(5): p. 591-603.
- 7. Vella, C.A., D. Marks, and R.A. Robergs, Oxygen cost of ventilation during incremental exercise to VO2 max. Respirology, 2006. 11(2): p. 175-81.
- 8. Bassett, D.R., Jr., Scientific contributions of A. V. Hill: exercise physiology pioneer. J Appl Physiol, 2002. 93(5): p. 1567-82.
- 9. Astorino, T.A., et al., Elucidating determinant of the plateay in oxygen consumption at VO2max. Br J Sports Med, 2005. 39: p. 665-660.
- 10. Day, J.R., et al., The maximally attainable VO2 during exercise in humans: the peak vs. maximum issue. J Appl Physiol, 2003. 95(5): p. 1901-7.
- 11. Duncan, G.E., E.T. Howley, and B.N. Johnson, Applicability of VO2max criteria: discontinuous versus continuous protocol. Medicine and Science in Sports and Exercise, 1997. 29(2): p. 273-278.
- 12. Rossiter, H.B., J.M. Kowalchuk, and B.J. Whipp, A test to establish maximum O2 uptake despite no plateau in the O2 uptake response to ramp incremental exercise. J Appl Physiol, 2006. 100: p. 764-770.
- 13. Astorino, T.A., Alterations in VO2max and the VO2 plateau with manipulation of sampling interval. Clinical physiology and functional imaging, 2009. 29(1): p. 60-67.
- 14. Astorino, T.A., et al., Incidence of the oxygen plateau at VO 2 max during exercise testing to volitional fatigue. Methods, 2000. 3(4).
- 15. Poole, D.C., D.P. Wilkerson, and A.M. Jones, Validity of criteria for establishing maximal O 2 uptake during ramp exercise tests. European journal of applied physiology, 2008. 102(4): p. 403-410.
- 16. Joyner, M.J., et al., Effects of beta-blockade on exercise capacity of trained and untrained men: a hemodynamic comparison. Journal of Applied Physiology, 1986. 60(4): p. 1429.
- 17. Davy, K.P. and D.R. Seals, Total blood volume in healthy young and older men. Journal of Applied Physiology, 1994. 76(5): p. 2059.
- 18. Noakes, T.D., Challenging beliefs: ex Africa semper aliquid novi. Medicine and Science in Sports and Exercise, 1996. 29(5): p. 571-590.
- 19. Noakes, T., K. Myburgh, and R. Schall, Peak treadmill running velocity during the VO2 max test predicts running performance. Journal of sports sciences, 1990. 8(1): p. 35.
- 20. Noakes, T.D., Lore of Running. 1987, Ed. Cape Town: Oxford University Press.
- 21. Noakes, T.D., Implications of exercise testing for prediction of athletic performance: a contemporary perspective. Medicine and Science in Sports and Exercise, 1988. 20(4): p. 319-330.
- 22. Noakes, T.D., Maximal oxygen uptake: "classical" versus "contemporary" viewpoints: a rebuttal. Medicine and Science in Sports and Exercise, 1998. 30(9): p. 1381-98.
- 23. Noakes, T.D., How did A V Hill understand the VO2max and the "plateau phenomenon"? Still no clarity? Br J Sports Med, 2008. 42(7).
- 24. Noakes, T.D., Testing for maximum oxygen consumption has produced a brainless model of human exercise performance. British journal of sports medicine, 2008. 42(7): p. 551.
- 25. Noakes, T.D., Maximal oxygen uptake as a parametric measure of cardiorespiratory capacity: comment. Medicine & Science in Sports & Exercise, 2008. 40(3): p. 585.
- 26. Noakes, T.D., Is it Time to Retire the AV Hill Model?: A Rebuttal to the Article by Professor Roy Shephard. Sports Medicine, 2011. 41(4): p. 263-277.
- 27. Noakes, T.D., Time to move beyond a brainless exercise physiology: the evidence for complex regulation of human exercise performance. Applied Physiology, Nutrition, and Metabolism, 2011. 36(1): p. 23-35.
- 28. Noakes, T.D., H. Crewe, and R. Tucker, The brain and fatigue. 2009.
- 29. Noakes, T.D. and F.E. Marino, Does a central governor regulate maximal exercise during combined arm and leg exercise? A rebuttal. European journal of applied physiology, 2008. 104(4): p. 757-759.
- 30. Noakes, T.D. and F.E. Marino, Point: Counterpoint: Maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of Applied Physiology, 2009. 106(1): p. 338.
- 31. Noakes, T.D. and F.E. Marino, Last word on Point: Counterpoint: maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of Applied Physiology, 2009.  $106(1)$ : p. 347.
- 32. Noakes, T.D. and R. Tucker, Do we really need a central governor to explain brain regulation of exercise performance? A response to the letter of Dr. Marcora. European journal of applied physiology, 2008. 104(5): p. 933-935.
- 33. Taylor, H.L., E. Buskirk, and A. Henschel, Maximal Oxygen Intake as an Objective Measure of Cardio-Respiratory Performance. 1955.
- 34. Saltin, B., Physiological effects of physical conditioning. Medicine & Science in Sports & Exercise, 1969. 1(1): p. 50.
- 35. Saltin, B., Maximal oxygen uptake and heart rate in various types of muscular activity. Journal of Applied Physiology, 1961. 16(6): p. 977.
- 36. Paavolainen, L., A. Nummela, and H. Rusko, Muscle power factors and VO2max as determinants of horizontal and uphill running performance. Scandinavian journal of medicine & science in sports, 2000. 10(5): p. 286-291.
- 37. JE, M., et al., Test of the classic model for predicting endurance running performance. Medicine and Science in Sports and Exercise, 2010. 42(5): p. 991-997.
- 38. Butts, N., B. Henry, and D. McLean, Correlations between VO2max and performance times of recreational triathletes. The Journal of sports medicine and physical fitness, 1991. **31**(3): p. 339.
- 39. Ingham, S., et al., Determinants of 2,000 m rowing ergometer performance in elite rowers. European journal of applied physiology, 2002. 88(3): p. 243-246.
- 40. Blair, S.N., et al., *Physical fitness and all-cause mortality*. JAMA: the journal of the American Medical Association, 1989. 262(17): p. 2395.
- 41. Lee, I., Associations of light, moderate, and vigorous intensity physical activity with longevity. American Journal of Epidemiology, 2000. 151(3): p. 293.
- 42. Blair, S.N., et al., Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA: the journal of the American Medical Association, 1996. 276(3): p. 205.
- 43. Blair, S.N., et al., Changes in physical fitness and all-cause mortality. JAMA: the journal of the American Medical Association, 1995. 273(14): p. 1093.
- 44. Hill, A.V., et al., Muscular Exercise, Lactic Acid, and the Supply and Utilisation of Oxygen.--Parts VII - VIII. Proceedings of the Royal Society: B, 1924. 97: p. 155-176.
- 45. Powers, S.K., et al., Effects of incomplete pulmonary gas exchange on VO2 max. Journal of Applied Physiology, 1989. 66(6): p. 2491.
- 46. Dempsey, J., P. Hanson, and K. Henderson, Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. The Journal of physiology, 1984. 355(1): p. 161.
- 47. Cymerman, A., et al., Operation Everest II: maximal oxygen uptake at extreme altitude. Journal of Applied Physiology, 1989. 66(5): p. 2446.
- 48. Andersen, P. and B. Saltin, Maximal perfusion of skeletal muscle in man. The Journal of physiology, 1985. 366(1): p. 233.
- 49. Saltin, B., Hemodynamic Adaptations to Exercise. American Journal of Cardiology, 1985. 52: p. 42D-47D.
- 50. Secher, N.H., et al., Central and regional circulatory effects of adding arm exercise to leg exercise. Acta Physiologica Scandinavica, 1977. 100(3): p. 288-297.
- 51. Astrand, I., et al., Reduction in maximal oxygen uptake with age. Journal of Applied Physiology, 1973. 35(5): p. 649.
- 52. Ades, P.A., et al., Effect of beta blockade and intrinsic sympathomimetic activity on exercise performance\*. The American journal of cardiology, 1984. 54(10): p. 1337-1341.
- 53. Tesch, P., Exercise performance and beta-blockade. Sports medicine (Auckland, NZ), 1985. 2(6): p. 389.
- 54. Blomqvist, C.G. and B. Saltin, Cardiovascular adaptations to physical training. Annual Review of Physiology, 1983. 45(1): p. 169-189.
- 55. Keul, J., et al., Effect of static and dynamic exercise on heart volume, contractility, and left ventricular dimensions. Circulation research, 1981. 48(6 Pt 2): p. I162.
- 56. Ehsani, A.A., J.M. Hagberg, and R.C. Hickson, Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. The American journal of cardiology, 1978. 42(1): p. 52-56.
- 57. Smith, M., et al., Exercise training bradycardia: the role of autonomic balance. Medicine & Science in Sports & Exercise, 1989. 21(1): p. 40.
- 58. Hickson, R., et al., Time course of the adaptive responses of aerobic power and heart rate to training. Medicine & Science in Sports & Exercise, 1981. 13(1): p. 17.
- 59. Wilmore, J., et al., Endurance exercise training has a minimal effect on resting heart rate: the HERITAGE study. Medicine & Science in Sports & Exercise, 1996. 28(7): p. 829.
- 60. Sawka, M.N., et al., Blood volume: importance and adaptations to exercise training, environmental stresses, and trauma/sickness. Medicine & Science in Sports & Exercise, 2000. 32(2): p. 332.
- 61. Gledhill, N., Blood doping and related issues: a brief review. Medicine & Science in Sports & Exercise, 1982. 14(3): p. 183.
- 62. Gledhill, N., The influence of altered blood volume and oxygen transport capacity on aerobic performance. Exercise and sport sciences reviews, 1985. 13(1): p. 75.
- 63. Ekblom, B., A.N. Goldbarg, and B. Gullbring, Response to exercise after blood loss and reinfusion. Journal of Applied Physiology, 1972. 33(2): p. 175.
- 64. Krogh, M., The diffusion of gases through the lungs of man. The Journal of physiology, 1915. 49(4): p. 271.
- 65. Ekblom, B., et al., *Effect of training on circulatory response to exercise*. Journal of Applied Physiology, 1968. 24(4): p. 518.
- 66. Tesch, P., A. Thorsson, and P. Kaiser, Muscle capillary supply and fiber type characteristics in weight and power lifters. Journal of Applied Physiology, 1984. 56(1): p. 35.
- 67. Brodal, P., F. Ingjer, and L. Hermansen, Capillary supply of skeletal muscle fibers in untrained and endurance-trained men. American Journal of Physiology-Heart and Circulatory Physiology, 1977. 232(6): p. H705.
- 68. Ingjer, F., Maximal aerobic power related to the capillary supply of the quadriceps femoris muscle in man. Acta Physiologica Scandinavica, 1978. 104(2): p. 238-240.
- 69. Andersen, P. and J. Henriksson, Capillary supply of the quadriceps femoris muscle of man: adaptive response to exercise. J Physiol, 1977. 270: p. 677-690.
- 70. Dempsey, J.A., Is the lung built for exercise? Medicine & Science in Sports & Exercise, 1986. 18(143-155).
- 71. Saltin, B. and P.D. Gollnick, Skeletal muscle adaptability: significance for metabolism and performance. 1983.
- 72. Gollnick, P., et al., Effect of training on enzyme activity and fiber composition of human skeletal muscle. Journal of Applied Physiology, 1973. 34(1): p. 107.
- 73. Dubouchaud, H., et al., Endurance training, expression, and physiology of LDH, MCT1, and MCT4 in human skeletal muscle. American Journal of Physiology-Endocrinology And Metabolism, 2000. 278(4): p. E571.
- 74. Ingjer, F., Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in man. The Journal of physiology, 1979. 294(1): p. 419.
- 75. Saltin, B., et al., FIBER TYPES AND METABOLIC POTENTIALS OF SKELETAL MUSCLES IN SEDENTARY MAN AND ENDURANCE RUNNERS\*. Annals of the New York Academy of Sciences, 1977. 301(1): p. 3-29.
- 76. Holloszy, J.O. and E.F. Coyle, Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology, 1984. 56(4): p. 831.
- 77. Holloszy, J.O. and F.W. Booth, Biochemical adaptations to endurance exercise in muscle. Annual Review of Physiology, 1976. 38(1): p. 273-291.
- 78. Costill, D., W. Fink, and M. Pollock, Muscle fiber composition and enzyme activities of elite distance runners. Medicine & Science in Sports & Exercise, 1976. 8: p. 96-100.
- 79. Saltin, B., et al., The Nature of the Training Response; Peripheral and Central Adaptations to One Legged Exercise. Acta Physiologica Scandinavica, 1976. 96(3): p. 289-305.
- 80. Klausen, K., et al., Central and regional circulatory adaptations to one-leg training. Journal of Applied Physiology, 1982. 52(4): p. 976.
- 81. Astrand, P.O. and B. Saltin, Oxygen uptake during the first minutes of heavy muscular exercise. J Appl Physiol, 1961. 16: p. 971-6.
- 82. Astrand, P.O. and B. Saltin, Maximal oxygen uptake and heart rate in various types of muscular activity. J Appl Physiol, 1961. 16: p. 977-81.
- 83. Glassford, R., et al., Comparison of maximal oxygen uptake values determined by predicted and actual methods. Journal of Applied Physiology, 1965. 20(3): p. 509.
- 84. Mitchell, J.H., B.J. Sproule, and C.B. Chapman, The physiological meaning of the maximal oxygen intake test. Journal of Clinical Investigation, 1958. 37(4): p. 538.
- 85. Saltin, B. and P. Astrand, Maximal oxygen uptake in athletes. Journal of Applied Physiology, 1967. 23(3): p. 353.
- 86. Stamford, B., Step increment versus constant load tests for determination of maximal oxygen uptake. European Journal of Applied Physiology and Occupational Physiology, 1976. 35(2): p. 89-93.
- 87. Shephard, R.J., et al., The maximum oxygen intake: An international reference standard of cardio-respiratory fitness. Bulletin of the World Health Organization, 1968. 38(5): p. 757.
- 88. Fardy, P.S. and H.K. Hellerstein, A comparison of continuous and intermittent progressive multistage exercise testing. Medicine & Science in Sports & Exercise, 1978. 10(1): p. 7.
- 89. Patton, J., J. Vogel, and R. Mello, Evaluation of a maximal predictive cycle ergometer test of aerobic power. European Journal of Applied Physiology and Occupational Physiology, 1982. 49(1): p. 131-140.
- 90. Washburn, R. and D. Seals, Comparison of continuous and discontinuous protocols for the determination of peak oxygen uptake in arm cranking. European Journal of Applied Physiology and Occupational Physiology, 1983. 51(1): p. 3-6.
- 91. Pierce, S.J., et al., Prolonged incremental tests do not necessarily compromise V [combining dot above] O2max in well-trained athletes. Journal of Science and Medicine in Sport, 1999. 2(4): p. 356-363.
- 92. McArdle, W., F. Katch, and G. Pechar, Comparison of continuous and discontinuous treadmill and bicycle tests for max VO2. Medicine & Science in Sports & Exercise, 1973. 5(3): p. 156.
- 93. Hermansen, L. and B. Saltin, Oxygen uptake during maximal treadmill and bicycle exercise. J Appl Physiol, 1969. 26(1): p. 31-7.
- 94. Buchfuhrer, M.J., et al., Optimizing the exercise protocol for cardiopulmonary assessment. Journal of Applied Physiology, 1983. 55(5): p. 1558.
- 95. Bergh, U., I. Kanstrup, and B. Ekblom, Maximal oxygen uptake during exercise with various combinations of arm and leg work. Journal of Applied Physiology, 1976.  $41(2)$ : p. 191.
- 96. Stromme, S., F. Ingjer, and H. Meen, Assessment of maximal aerobic power in specifically trained athletes. Journal of Applied Physiology, 1977. 42(6): p. 833.
- 97. Magel, J., et al., Specificity of swim training on maximum oxygen uptake. Journal of Applied Physiology, 1975. 38(1): p. 151.
- 98. Pechar, G., et al., Specificity of cardiorespiratory adaptation to bicycle and treadmill training. Journal of Applied Physiology, 1974. 36(6): p. 753.
- 99. Secher, N.H., et al., Maximal oxygen uptake during arm cranking and combined arm plus leg exercise. Journal of Applied Physiology, 1974. 36(5): p. 515.
- 100. Nagle, F., J. Richie, and M. Giese, VO2max responses in separate and combined arm and leg airbraked ergometer exercise. Medicine & Science in Sports & Exercise, 1984. 16(6): p. 563.
- 101. Howley, E.T., J. David R. Bassett, and H.G. Welch, Criteria for maximal oxygen uptake: review and commentary. Medicine & Science in Sports & Exercise, 1995. 27(9): p. 1292-1301.
- 102. Midgley, A.W., et al., Criteria for determination of maximal oxygen uptake: a brief critique and recommendations for future research. Sports Medicine, 2007. 37(12): p. 1019-1028.
- 103. Cristina Bisi, M., R. Stagni, and G. Gnudi, Automatic detection of maximal oxygen uptake and ventilatory threshold. Computers in Biology and Medicine, 2011.  $41(1)$ : p. 18-23.
- 104. Medicine, A.C.o.S., Guidelines for Graded Exercise Testing and Prescription. 1975, Philadelphia: Lea & Febiger.
- 105. Eisenmann, J.C. and R. Malina, Secular Trend in Peak Oxygen Consumption Among United States Youth in the 20th Century. Am J. Hum. Biol, 2002. 14: p. 699-706.
- 106. Astrand, P.-O., Experiemental Studies of physical working capacity in relation to sex and age. 1952, Copenhagen: Ejnar Munksgaard.
- 107. Issekutz, B. and K. Rodahl, Respiratory quotient during exercise. Journal of Applied Physiology, 1961. 16(4): p. 606.
- 108. Astrand, I., P.O. Astrand, and K. Rodahl, Maximal Heart Rate during work in older men. J Appl Physiol, 1959. 14: p. 562-566.
- 109. Doblen, W., I. Astrand, and A. Bergstrom, An analysis of age and other factors related to maximal oxygen uptake. J Appl Physiol, 1967. 22: p. 934-938.
- 110. Robinson, S., Experimental studies of physical fitness in relation to age. European Journal of Applied Physiology and Occupational Physiology, 1938. 10(3): p. 251-323.
- 111. Cunningham, D., et al., Reliability and reproducibility of maximal oxygen uptake measurement in children. Medicine & Science in Sports & Exercise, 1977. 9(2): p. 104.
- 112. Wasserman, K., Lactate and related acid base and blood gas changes during constant load and graded exercise. Canadian Medical Association Journal, 1967. 96(12): p. 775.
- 113. Issekutz, B., N. Birkhead, and K. Rodahl, Use of respiratory quotients in assessment of aerobic work capacity. Journal of Applied Physiology, 1962. 17(1): p. 47.
- 114. Sidney, K. and R. Shephard, Maximum and submaximum exercise tests in men and women in the seventh, eighth, and ninth decades of life. Journal of Applied Physiology, 1977. 43(2): p. 280.
- 115. Londeree, B. and M. Moeschberger, Influence of age and other factors on maximal heart rate. J Cardiac Rehabil, 1984. 4: p. 44-49.
- 116. Robergs, R.A. and R. Landwehr, The surprising history of the "HRmax= 220-age" equation. J Exerc Physiol, 2002. 5(2): p. 1-10.
- 117. Fox 3rd, S., J.P. Naughton, and W.L. Haskell, Physical activity and the prevention of coronary heart disease. Annals of clinical research, 1971. 3(6): p. 404.
- 118. Tanaka, H., K.D. Monahan, and D.R. Seals, Age-predicted maximal heart rate revisited. Journal of the American College of Cardiology, 2001. 37(1): p. 153.
- 119. Borg, G., Psychophysical bases of perceived exertion. Medicine and Science in Sports and Exercise, 1982. 14(5): p. 377.
- 120. Noble, B., et al., A category-ratio percieved exertion scale: relationship to blood and muscle lactates and heart rate. Medicine & Science in Sports & Exercise, 1983. 15: p. 523-528.
- 121. Robertson, R. and B. Noble, Perception of physical exertion: methods, mediators, and applications. Exerc Sport Sci Rev, 1997. 25: p. 407-452.
- 122. Eston, R.G., B.L. Davies, and J.G. Williams, Use of perceived effort ratings to control exercise intensity in young healthy adults. European Journal of Applied Physiology and Occupational Physiology, 1987. 56(2): p. 222-224.
- 123. Eston, R. and J. Williams, Reliability of ratings of perceived effort regulation of exercise intensity. British journal of sports medicine, 1988. 22(4): p. 153.
- 124. Lydiard and A.G. Gilmore, Running the Lydiard Way. 1978, Mountain View California: World Publications.
- 125. Pirie, G., Running Wild. 1961, London: W.H. Allen.
- 126. Hawkins, M., et al., Maximal xygen uptake as a parametric measure of cardiorespiratory capacity. Medicine & Science in Sports & Exercise, 2007. 39(1): p. 103-107.
- 127. Ekblom, B.T., Last word on Point: Counterpoint: maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of Applied Physiology, 2009. 106(1): p. 348.
- 128. Shephard, R.J., Comments on Point: Counterpoint: maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of Applied Physiology, 2009. 106(1): p. 343.
- 129. Hopkins, W.G., The improbable central governor of maximal endurance performance. Sportscience, 2009. 13: p. 9-12.
- 130. Foster, C. and A. Lucia, Comments of point: counterpoint: maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of applied physiology (Bethesda, Md.: 1985), 2009. 106(1): p. 343.
- 131. Calbet, J., Comments of point: counterpoint: maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of applied physiology (Bethesda, Md.: 1985), 2009. 106(1): p. 346.
- 132. Gonzalez-Alonso, J. and S. Mortensen, Comments of point: counterpoint: maximal oxygen uptake is/is not limited by a central nervous system governor. Journal of applied physiology (Bethesda, Md.: 1985), 2009. 106(1): p. 344.
- 133. Elfegoun, T.B., et al., Neuromuscular and circulatory adaption during combined arm and leg exercise with different maximal work loads. Eur J Appl Physiol, 2007. 101: p. 103-611.
- 134. Swart, J., et al., Exercising with reserve: evidence that the central nervous system regulates prolonged exercise performance. British journal of sports medicine, 2009. 43(10): p. 782.
- 135. Baron, B., et al., Why does exercise terminate at the maximal lactate steady state intensity? British journal of sports medicine, 2008. 42(10): p. 828.
- 136. Bassett, D.R., Jr. and E.T. Howley, Limiting factors for maximum oxygen uptake and determinants of endurance performance. Medicine & Science in Sports & Exercise, 2000. 32(1): p. 70-84.
- 137. Gordon, D., et al., Incidence of the Plateau at V O2max is Dependent on the Anaerobic Capacity. International journal of sports medicine, 2011. 32(01): p. 1-6.
- 138. Howley, E.T., VO2 max and the plateau-needed or not. Medicine & Science in Sports & Exercise, 2007. 39(1): p. 101-2.
- 139. Lacour, J.R., L. Messonnier, and M. Bourdin, The leveling-off of oxygen uptake is related to blood lactate accumulation. Retrospective study of 94 elite rowers. Eur J Appl Physiol, 2007. 101(2): p. 241-7.
- 140. Lucia, A., et al., Frequency of the VO2max plateau phenomenon in world-class cyclists. Int J Sports Med, 2006. 27(12): p. 984-82.
- 141. Rivera-Brown, A.M., et al., Anaerobic power and achievement of VO2 plateau in pre-pubertal boys. Int J Sports Med, 2001. 22(2): p. 111-5.
- 142. Rowland, T.W. and L.N. Cunningham, Oxygen uptake plateau during maximal treadmill exercise in children. Chest, 1992. 101(2): p. 485-9.
- 143. Weir, J.P., et al., VO2 plateau detection in cycle ergometry. Medicine & Science in Sports & Exercise, 2001. 33(5): p. S45.
- 144. Yoon, B.-K., L. Kravitz, and R. Robergs, VO2max, Protocol Duration, and the VO2 Plateau. Medicine and Science in Sports and Exercise, 2007. 39(7): p. 1186-92.
- 145. Rossiter, H., J. Kowalchuk, and B.J. Whipp, A test to establish O2 uptake despite no plateau in the O2 uptake response to ramp incremental exercsie. Journal of Applied Physiology, 2006. 100: p. 764-770.
- 146. Day, J.R., et al., The maximally attainable VO2 during exercise in humans: the peak vs. maximum issue. J Appl Physiol, 2003. 95: p. 1901-1907.
- 147. Noakes, T.D., Peak treadmill running velocity during the VO2max test predicts running performance. Journal of Sports Science, 1990. 8: p. 35-45.
- 148. Noakes, T.D., Implications of exercise testing for prediction of athletic performance: a contemporary perspective. Medicine and Science in Sports and Exercise, 1997. 20(4): p. 319-330, 1988.
- 149. Perret, C. and G. Mueller, Validation of a new portable ergospirometric device (Oxycon Mobile®) during exercise. International journal of sports medicine, 2006. 27(5): p. 363-367.
- 150. Rosdahl, H., et al., Evaluation of the Oxycon Mobile metabolic system against the Douglas bag method. European journal of applied physiology, 2010. 109(2): p. 159-171.
- 151. Fielding, R., et al., The reproducibility of the Bruce protocol exercise test for the determination of aerobic capacity in older women. Medicine & Science in Sports & Exercise, 1997. 29(8): p. 1109.
- 152. Hickey, M., et al., Day to day variation in time trial cycling performance. International journal of sports medicine, 1992. **13**: p. 467-467.
- 153. Skinner, J., et al., Reproducibility of maximal exercise test data in the HERITAGE Family Study. Medicine & Science in Sports & Exercise, 1999. 31(11): p. 1623.
- 154. Snell, P.G., et al., Maximal oxygen uptake as a parametric measure of cardiorespiratory capacity. Medicine and Science in Sports and Exercise, 2007. 39(1): p. 103-107.



Figure 1, Demonstration of VO<sub>2</sub> Plateau



Figure 2, Demonstration of Lack of VO<sub>2</sub> Plateau



Figure 3, VO<sub>2</sub> Comparison between DGXT and CGXT



Figure 4,  $V_E$  Comparisons between CGXT and DGXT



Figure 5, Heart Rate Comparisons between DGXT and CGXT



Figure 6, RER Comparisons Between CGXT and DGXT



Figure 7, Duration of Each DGXT Run



Figure 8, Subject 1 Time Course of the VO<sub>2</sub> Response for Discrete Continuous Speeds



Figure 9, Subject 2 Time Course of the VO2 Response for Discrete Continuous Speeds



Figure 10, Subject 3 Time Course of the VO2 Response for Discrete Continuous Speeds



Figure 11, Subject 4 Time Course of the VO2 Response for Discrete Continuous Speeds

\*Subject 4 had an invalid 110% of PTV run due to equipment malfunction



Figure 12, Subject 5 Time Course of the VO2 Response for Discrete Continuous Speeds



Figure 13, Subject 6 Time Course of the VO2 Response for Discrete Continuous Speeds



Figure 14, Subject 7 Time Course of the VO2 Response for Discrete Continuous Speeds



Figure 15, Subject 8 Time Course of the VO2 Response for Discrete Continuous Speeds



Figure 16, Subject 9 Time Course of the VO2 Response for Discrete Continuous Speeds

# **APPENDIX A**

# **Screening Questionnaire:**

1. Have any of the following occurred with your father or first-degree male relative before age 55, or with your mother or first-degree female relative before the age of 65: Myocardial infarction, coronary revascularization, or sudden death?

Yes No

2. Do you currently smoke, or quit smoking within the last 6 months? Yes No

3. Has a doctor ever told you that you have hypertension, systolic blood pressure  $\geq 140$ mmHg, diastolic blood pressure  $\geq 90$ mmHg, or do you currently take antihypertensive medications?

Yes No

4. Has a doctor ever told you that you have hyperlipidemia ("High cholesterol") defined as  $LDL > 130$  mg/dL, or been told you have HDL < 40 mg/dL, or are on lipid-lowering medication? If you have only been told total serum cholesterol, has it been >200mg/dL?

Yes No

5. Has a doctor ever told you that you have diabetes, or impaired fasting glucose defined as fasting glucose  $\geq 100$  mg/dL measured on two separate occasions?

No No

6. What is your height and weight? Height (inches):  $*0.0254 =$  Height (meters): Weight (lb.):  $/2.2$  = Weight (kg): Height (meters)\*Height (meters) =  $[H \text{ eight}$  (meters)]<sup>2</sup>: Weight(kg) /[Height(meters)]<sup>2</sup>  $=$  BMI: Is the participant's  $BMI > 30$ Yes No

7. Do you run at least 15 miles per week on average, and have done so over the past 6 months?

Yes No

8. During exercise, do you experience chest discomfort, unreasonable breathlessness, dizziness, fainting, or blackouts?

Yes No

9. Do you take heart medications? Yes No

10. Are you older than 35? No No

If the participant answered "no" to all questions, except question 7, read the following text:

"*According to your screening, we have determined that you are eligible to participate in the study. We can set up a time with you in which you will be able to commit 30 minutes to an hour of your time for your first testing"* 

If the participant answered "yes" to any question, except question 7, read the following test:

*"According to your screening, we have determined that it might be unsafe for you to participate in this study given the presence of risk factors. Thank you for taking the time to contact us."*

Participant name

Time and date of first meeting

Date

 $\overline{a}$ 

 $\overline{a}$ 

 $\overline{a}$ 

## APPENDIX B

Sponsored by the Department of Kinesiology, Recreation, and Sports Studies

# Are you a runner?

You can participate in a research study at the University of Tennessee, and get a free  $VO_{2max}$  test and body fat analysis in the process!

#### Requirements

- o Run at least 15 miles a week regularly
- o Be in good health, and 18-35 years old
- o Willing to commit to 4 separate running tests
- o Do interval training 1 or more days a week



### Interested?

Contact Richie Castle at the number below to see if you are eligible



## APPENDIX C

#### **Research Team Member's Pledge of Confidentiality**

As a member of this project's research team, I understand that I will be assisting in confidential fitness tests. The data and personal information in these fitness tests has been recorded from research participants who participated in this project on good faith that their test results and personal information would remain strictly confidential. I understand that I have a responsibility to honor this confidentially agreement. I hereby agree not to share any information in these fitness tests with anyone except the primary researcher of this project, his primary advisor, or other members of this research team. Any violation of this agreement would constitute a serious breach of ethical standards, and I pledge not to do so.

\_ \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

Research Team Member Date
## APPENDIX D

#### Recruitment E-Mail

Dear Runner or runners,

 I would like to invite you to participate in a study for the University of Tennessee looking to examine methods of testing aerobic fitness. If you choose to participate, you will receive free body fat, body mass index, and VO<sub>2max</sub> testing. The testing consists of coming to our lab on the University of Tennessee campus for four separate days of testing. Testing will take 30 to 60 minutes for each day, and will consist of running at various high-intensity speeds to determine  $VO<sub>2max</sub>$ .

 To be eligible to participate you must be 18 to 35 years of age, healthy, and have been running an average of 15 miles a week for the past 6 months. If you would like to participate, please call Richie Castle at  $(865)$  974-5091, or email me at reastle@utk.edu.

Richie Castle, B.S. Graduate Student in Exercise Physiology University of Tennessee, Knoxville

# APPENDIX E

### **INFORMED CONSENT FORM**

**TITLE:** Why is there so much confusion about VO2 plateau? A re-examination of the work of A.V. Hill

Investigators: Richie Castle, B.S. David R. Bassett, Jr., Ph.D.

Address:

 Kinesiology, Recreation, and Sport Studies 322 HPER Building University of Tennessee 1914 Andy Holt Ave., Knoxville, TN 37996-2700

Phone: (865) 974-5091

#### **PURPOSE**

You are invited to participate in a research study. The purpose of this study is to look at your rate of oxygen uptake near the end of a maximal exercise test to look for a plateau (i.e. leveling off) in oxygen uptake. You will be asked to participate in exercise testing on four separate days, while we study the effects of running speed on oxygen uptake.

#### **PROCEDURES**

On four separate days you will be asked to come to the Applied Physiology Laboratory in the Health, Physical Education & Recreation (HPER) building. Prior to your participation, you will be screened by the primary researcher to determine whether you have any risk factors that prevent you from taking part in this study. On the first day of testing, you will perform a maximal treadmill running test to determine your aerobic fitness. A researcher will measure your height, weight and body fat. Following these measurements you will undergo a maximal exercise test on a treadmill. This involves running on a treadmill until exhaustion while wearing a face mask, a vest housing two portable measurement devices, and heart rate monitor.

The next test will consist of a discontinuous exercise test. The laboratory test will consist of running on a treadmill at a single speed, resting, then repeating for faster speeds. While running, you will wear a face mask that will funnel all breathing through a mouth piece and chest harness that will house two light devices used in measuring oxygen content. In addition, the heart rate monitor will be worn around your chest. You will run for 10 minutes at a submaximal speed as a warm-up before running at each submaximal or maximal speed. Each 10 minute run will be separated by 15 minutes of rest, and the speed will be increased with each successive bout. Each bout will be performed for 10 minutes or until you are unable to continue, whichever comes first. The fastest speed will be equal to the fastest speed you attained on your first visit.

The final two tests will consist of you running at speeds that are approximately 5% and 10% faster than highest speed attained during the first test. Each test will be preceded by a 10 minute warm-up at a speed that is approximately 80% of the peak speed attained on the continuous run. The final two tests will be performed for 10 minutes until you are unable to continue, whichever comes first.

Participant initials

The total time commitment will be approximately 3 hours. One to six days of rest will be allowed between tests.

#### **BENEFITS OF PARTICIPATION**

By participating in this study, you will learn your current aerobic fitness level  $(VO_{2max})$ , body mass index, and percent body fat.

#### **RISKS OF PARTICIPATION**

Risks are similar to those experienced during a typical vigorous workout. Some risks include muscle soreness, dizziness, headache, leg discomfort, gastrointestinal discomfort, and a very rare possibility of a cardiac event. Sudden cardiac death during exercise among 18 to 35 year old persons could occur, but this is very rare. Researchers collecting data will be CPR/AED certified. Falling while running is the most likely injury. During running, one person will spot you, and there will be padding at the rear of the treadmill. During any part of the test, you may stop for any reason by signaling the researchers, or jumping off and straddling the treadmill belt. If the primary researcher feels it is unsafe for you to continue a test, he will terminate the test immediately. The University of Tennessee does not "automatically" reimburse subjects for medical claims or other compensation. If physical injury is suffered in the course of research, or for more information, please notify Richie Castle at (865) 974-5091 or rcastle@utk.edu.

#### **CONFIDENTIALITY**

Only the researchers will have access to your test results. All information will eventually be used for a research report, however, your private information will not be presented in the report.

#### **CONTACT INFORMATION**

If you have questions or concerns at any time during the course of the testing procedures or after completion of the testing procedures, you may contact Dr. David Bassett at (865) 974-8766. If you have questions concerning your rights as a participant, contact the Compliance Section of the Office of Research at (865) 974-3466.

#### **PARTICIPATION**

Your participation in this study is strictly voluntary. You have the option to withdraw from the study at any time without penalty. If you withdraw from the study, then all data will be given to you or destroyed.

## **AUTHORIZATION**

By signing this informed consent form, I am indicating that I have read and understood this document and have received a copy of it for my personal records. I have been given the opportunity to ask questions about the research study. By signing this form I indicate that I agree to serve as a participant in this research study.

Participant's name

Participant's signature Date

\_

\_ \_\_\_\_\_\_\_\_\_\_\_\_\_\_

\_ \_\_\_\_\_\_\_\_\_\_\_\_\_\_

Investigator's signature **Date** 

## **Vita**

Richie Castle often spends his time wishing that he were Bruce Wayne and unsuccessfully picking up women at bars. It has not occurred to him that those two reasons may be linked.

Although aspiring to become a physician, life dealt him a different hand and he followed up on his undergraduate work and pursued a master's in exercise physiology. Be began the process arrogant and ignorant. As he learned more, the adage, "the more you know, the less you realize you know," rang true for him, and he learned the core of humility: no matter how much you know, there is always somebody that knows more.

He was accepted into Nova Southeastern University School of Osteopathic Medicine in February of 2011. Before you ask him, no he is not doing it for the money, and no he does not know what specialty he wants to do. However, should you ask him, he will be willing to see you as a patient in four years. Also, to be honest, he doesn't really understand the difference between osteopathic and allopathic medicine, but supposes he will in a couple of years.

He loves his mother who died just a day after defending his thesis. It was not unexpected, but the timing was. One of his pet peeves is when a person says, "this is the last thing I need right now," about an event that is never advantageous (e.g. when exactly would you need your car to break down?). His mother's death will never be a happy event, but he appreciates that the timing was as good as it was.

He is incredibly happy to finally become a doctor after several medical school rejections, hours of volunteer work, days spent on classes necessary for medical school and unnecessary for graduation, and answering the essay question "why do you want to become a doctor" written in ten similar but still intrinsically different forms. He is also happy to move. Knoxville is a college town and he graduated from college four years ago. Finally, he is happy to submit this monster of a learning experience. I'll close with a quote from my mother's husband (who enjoys driving for the sake of driving), "The thing about taking a trip, is that once I get where I'm going, there is nothing for me to do except pack it up and go home. You spend this time thinking the destination is the fun part, then I realize it was the drive."