WHY DOES METABOLIC RATE INCREASE CURVILINEARLY
WITH RUNNING VELOCITY?

By

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Abstract

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Why Does Metabolic Rate Increase Curvilinearly With Running Velocity?

Thesis directed by Associate Professor *Emeritus*, Rodger Kram

The 'cost of generating force' model proposes that a major determinant of metabolic rate during running is the rate of muscular force production. However, the amount of muscle force needed during running is affected by the effective mechanical advantage (EMA), the ratio of the ground reaction force moment arm (R) to the muscle moment arm (r), R/r. The 'cost of generating force' model assumed that EMA and active muscle volume remain constant across velocity. With this assumption, the cost of generating force hypothesis explains 80% of the linear increase in metabolic rate in human runners across a moderate velocity range. Additionally, many studies have demonstrated a linear relationship between metabolic rate and running velocity for a diverse assortment of species. However, in humans there is less of a consensus of how to mathematically characterize the relationship. Using 7 sub-elite male runners, I performed a more systematic analysis of EMA over 6 different velocities (8, 10, 12, 14, 16 and 18km/hr) to explain both the remaining 20% and the curvilinear increase in metabolic rate. I hypothesized that the curvilinear metabolic rate pattern observed in elite runners at fast sub-maximal velocities can be explained by a decrease in EMA at the hip, knee and ankle joints, which necessitates a greater volume of active muscle recruitment. Over the velocity range, all subjects demonstrated a curvilinear increase in metabolic rate. Ankle EMA decreased by $14.5 \pm 4.1\%$, while hip EMA showed the largest magnitude decrease of $51.2 \pm 30.2\%$. Accordingly, the active volume of hip extensor muscles increased 50.1% from 448 ± 245 cm^3 to 898 ± 250cm^3 across the velocity range. The ankle extensor active muscle volume increased by 32.8% from 713 ± 145cm^3 to 1061 ± 159cm^3. I extended the cost of generating force model and found that in human runners, metabolic rate is proportional to the rate of force generation multiplied by the volume of muscle activated.

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Why Does Metabolic Rate Increase Curvilinearly With Running Velocity?

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Chapter I: Literature Review

The rate at which a runner consumes metabolic energy when they run at a submaximal velocity is most commonly approximated through the rate of oxygen uptake ($\dot{V}O_2$). This rate in mlO$_2$/kg/min is often referred to as an individual's running economy (RE) and is a key determinant of running performance (Daniels, 1985; Conley & Krahenbuhl, 1980). The intensity of exercise influences the relative rate of carbohydrate (CHO) and fat metabolism, and therefore the amount of energy made available per liter of oxygen uptake varies with intensity or running velocity (Blaxter, 1989). The respiratory exchange ratio (RER) is defined as the ratio of the rate of carbon dioxide production ($\dot{V}CO_2$) produced to the rate of O$_2$ consumed. From stoichiometry, a RER of 0.7 indicates 100% fat metabolism and 1.0 indicates 100% CHO metabolism. Thus, RER indicates the relative amount of CHO and fats utilized. It is well established that RER increases as submaximal running velocity increases (Saunders et al., 2004; Fletcher et al., 2009). Measuring only $\dot{V}O_2$ does not take into account the changes in substrate utilization that take place with changes in intensity. Thus, to quantify the rate of energy consumed in running, it is more accurate to use both $\dot{V}O_2$ and $\dot{V}CO_2$ to calculate how much of the energy is being metabolized from fats and CHOs. Fletcher et al. (2009) found that expressing RE in units of energy cost (W/kg) was more sensitive to changes in intensity than expressing RE in terms of just $\dot{V}O_2$ because it takes into account the different substrates used at different submaximal velocities. This conclusion was corroborated several years later by Shaw et al. (2014) who found oxygen cost *per distance* to be insensitive to changes in running velocity and therefore not a valid index for the actual energetic cost of running.

Traditionally, researchers have found that the rate of energy expenditure increases linearly at faster running velocities when using both $\dot{V}O_2$ (ml/kg/min) and the rate of energy expenditure (W/kg). Numerous studies have supported this over a submaximal range of velocities from ~2-4 $m \cdot s^{-1}$ in average to good runners (Margaria et al., 1963, Helgerud, 1994; Helgerud, et al., 2010; Menier & Pugh, 1968). However, there is disagreement about the nature of the relationship between metabolic cost and velocity in high-caliber runners who can sustain a wider range of submaximal velocities.

Steudel-Numbers and Wall-Scheffler (2009) reported that metabolic rate increases curvilinearly with running velocity in well-trained distance runners during treadmill running over a velocity range of ~2.01-4.9 $m \cdot s^{-1}$ (Figure 1). Similarly, Tam et al. (2012) reported significant increases in oxygen cost per distance at 5.0 $m \cdot s^{-1}$ compared to 3.33 $m \cdot s^{-1}$ in elite distance runners. Tam suggested that the finding could be explained by the increasing contribution of aerodynamic resistance to the metabolic rate during their over-ground running protocol. However, this does not explain the previous findings collected on treadmills, which involve negligible aerodynamic resistance.

Most recently, Batliner et al. (2017) compared average and sub-elite runners over a wide range of submaximal velocities. Average runners completed trials spanning a velocity range of 1.78-4.08 $m \cdot s^{-1}$ submaximally, while the sub-elite runners were capable of a wider velocity range of 1.78-5.14 $m \cdot s^{-1}$ submaximally. Over the wider range of velocities sustained by the sub-elite runners, both the oxygen uptake rate and metabolic rate vs. velocity relationships were best described by curvilinear fits (Figure 2 and 3). Even though Batliner et al. (2017) studied both average and sub-elite runners across their full range of submaximal velocities, the curvilinear increase in energetic cost was only observed in the sub-elite group of runners, who were able to

complete a wider range of velocities. This suggests that the sub-elite curvilinear increase in

metabolic rate was not caused by a physiological parameter, but a biomechanical factor.
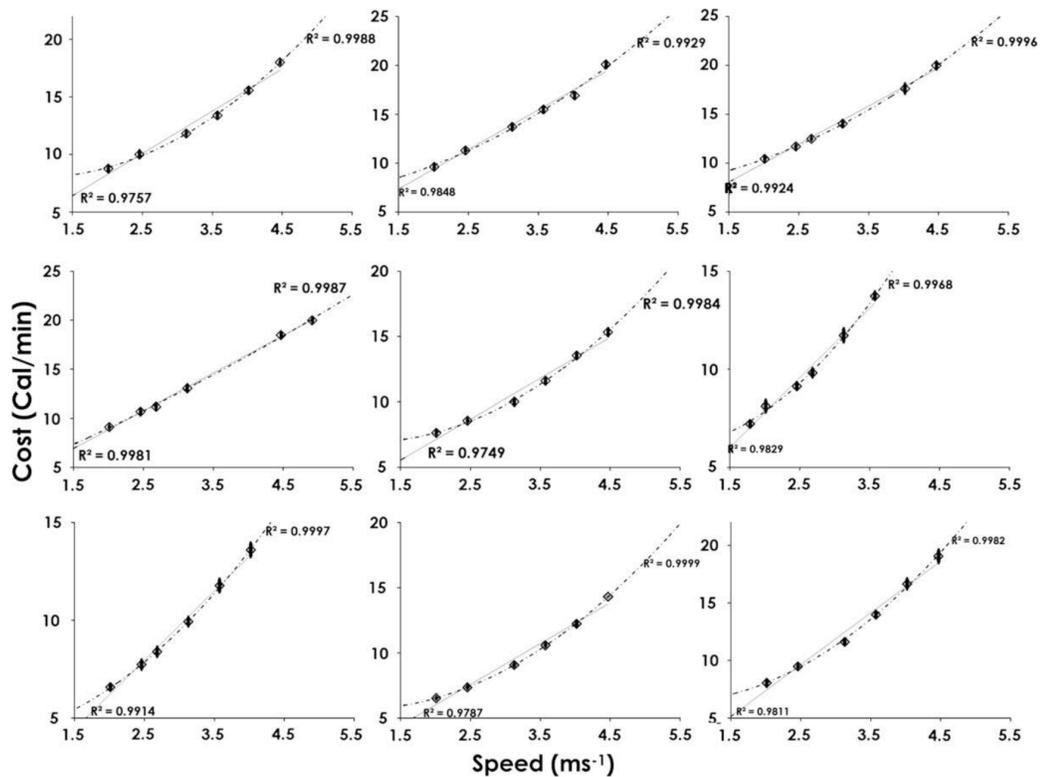
Figure 1. Each individual participant's metabolic rate (Cal/min) plotted vs. running speed with both linear and curvilinear line fits. Open diamonds are males; closed diamonds are females. The R^2 values on the bottom left are for the linear fit; the values on the top right are for the curvilinear fit. Data from Steudel-Numbers and Wall-Scheffler, 2009.
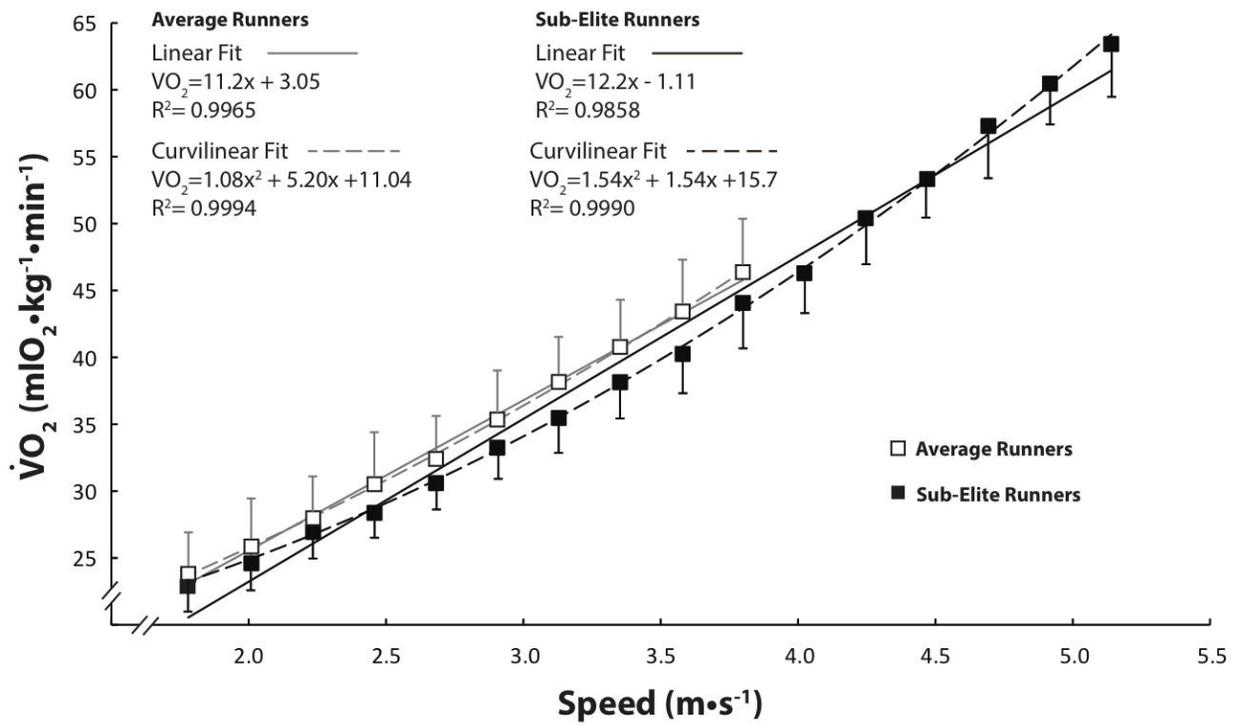
Figure 2. $\dot{V}O_2$ vs. running speed for Average and Sub-elite subjects calculated from mean slopes, intercepts, quadratic coefficients, linear coefficients, and R^2 values for linear and curvilinear (2nd order polynomial) fits. Data from Batliner et al., 2017.
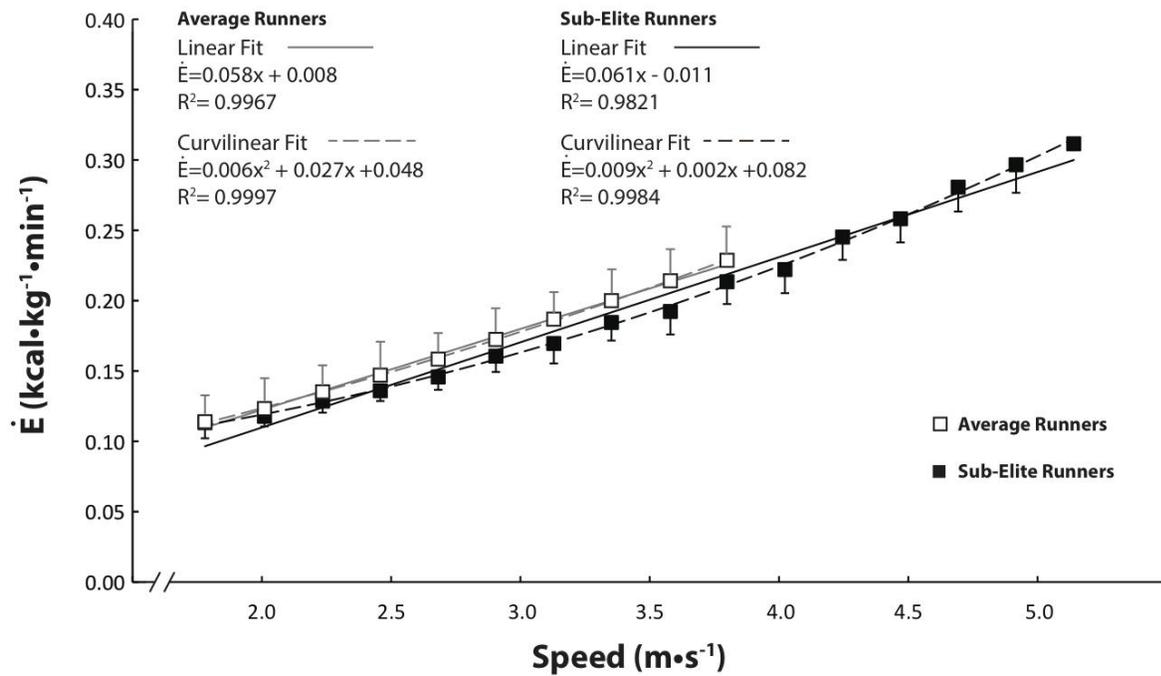
Figure 3. Metabolic rate vs. running speed for Average and Sub-elite groups calculated from mean slopes, intercepts, quadratic coefficients, linear coefficients, and R^2 values for linear and curvilinear (2nd order polynomial) fits. Data from Batliner et al., 2017.

What are the biomechanical determinants of the metabolic cost of running? When an individual runs, the leg muscles and tendons function in series to store and return mechanical energy similar to springs. With each step, kinetic energy is stored as elastic energy as the body's center of mass (CoM) is lowered towards the ground and slowed during each braking phase. Stored elastic energy is then converted to gravitational potential energy and kinetic energy as the body is lifted and accelerated during a propulsive phase. However, the amount of mechanical work the body appears to perform is not accurately reflected by the metabolic energy the body uses during running (Heglund et al., 1982). This is because the main determinant of metabolic cost is producing force to support body weight (Kram & Taylor, 1990). During the stance phase, most of the force is produced by muscles that act nearly isometrically, with little to no change in muscle length (Kram & Taylor, 1990; Roberts et al., 1998).

The idea that force and not mechanical work, determine the metabolic cost of running was developed by C.R. Taylor in 1980. He proposed the 'cost of generating force model' based on experiments in which he measured the metabolic cost of added weight to a group of different animal species that spanned a wide range of body weights. The rate of oxygen (O_2) consumption increased almost proportionately to the weight supported (Figure 4). That is, a 10% increase in body weight, resulted in a ~10% increases in the rate of O_2 consumption (L/min). It was noted that while there was an increase in O_2 consumption with the added weight, the average vertical acceleration of the center of mass did not change. This led Taylor to conclude that the muscular force developed by the animal during the stance phase increased proportionally with the load. Additionally, generating force on the ground was much more costly for the smaller animals than the larger animals. Taylor speculated that this was because compared to a horse, a little mouse must take many quick strides and generate force using fast, uneconomical muscle fibers. The

differences in the cost of running between animals of different sizes suggested that the time available for developing force is an important determinant of the energy cost of running.

A decade later, in their seminal paper, Kram and Taylor (1990) proposed that the metabolic rate during running is determined by the rate of muscular force production (calculated as the inverse of foot-ground contact time per step, $1/t_c$, where t_c = foot-ground contact time) and the volume of muscle activated. Presumably, shorter t_c requires muscle to activate fast-twitch fibers to generate the force since they have high rates of cross bridge cycling due to faster ATPase in the muscle cell. However, relying on these faster muscle fibers is more costly since they consume ATP at faster rates (Rall, 1985). Longer t_c presumably allows for the recruitment of slower, more economical muscle fibers. Kram and Taylor's "cost of generating force hypothesis" helped explained the paradox of why roughly the same amount of energy is consumed in running a mile regardless of the running velocity.
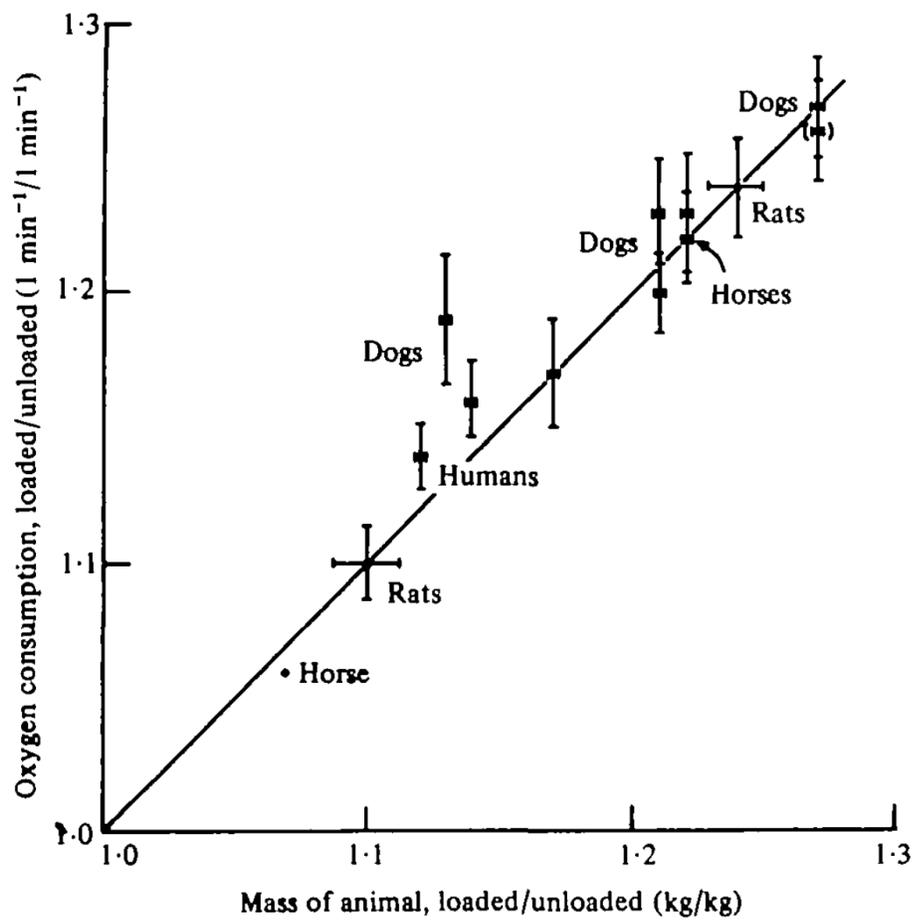
Figure 4. Oxygen consumption ratio (loaded/unloaded) vs. animal weight ratio (loaded/unloaded). The solid line is drawn to show direct proportionality of 1 between the two ratios, indicating that the muscular force developed by the animal during the stance phase increased proportionally. Data from Taylor, 1980.

The volume of active muscle recruited to generate the required force is the product of fascicle length and active cross-sectional area. The metabolic cost of generating force is dependent on the active cross-sectional area and the average length of the activated muscle fascicles. For muscle fascicles similar in composition, and acting under similar levels of activation and shortening velocities, muscles with shorter fascicles consume proportionally less ATP per unit force generated compared to muscles with longer fascicles (Roberts et al., 1998).

The amount of total muscle force the body must produce when we run is determined by how our lower limbs act as a series of levers (Biewener, 1989). Each of the leg segments: thigh, shank, and foot, act as a lever with a fulcrum at a joint center. Over the stance phase, changes in limb posture affect the ground reaction force moment arm (R), or the perpendicular distance the resultant ground reaction force is away from the respective joint center. The distance that the muscle force is exerted relative to the joint center defines the internal muscle-tendon moment arm (r). The effective mechanical advantage (EMA) is the ratio of these two moment arms, r/R (Figure 5). In bipeds, EMA is calculated about the hip, knee, and ankle. Because r, the internal muscle-tendon arm changes very little throughout t_c, EMA is primarily determined by how R changes throughout t_c based upon how flexed the joint angles are and the orientation of the resultant ground reaction force vector. Changes in EMA change the amount of muscle volume that must be activated. The amount of active muscle volume required to generate force underlies differences in metabolic costs. Smaller EMAs require a greater muscle force needed to exert force on the ground and thus more active muscle volume and higher metabolic costs (Biewener, 1989). This normally occurs when the limb segments are more flexed and less aligned with the resultant GRF. As an individual's limb posture becomes more upright (straighter legs), the GRF

becomes more aligned with the limbs and joint centers, and the force that the muscles must exert to support body weight decreases.

Biewener (1989) showed that EMA does not vary significantly with changes in velocity or gait in quadrupeds. Thus, previous studies have assumed that EMA is constant over a wide range of velocities (Kram & Taylor, 1990). However, in humans there is some evidence that EMA and active muscle volume might change over a velocity range and between walking and running gaits.

Biewener et al., (2004) studied 4 male humans to quantify EMA and the muscle force generating requirements during walking and running. They allowed the subjects to self-select their 3 running velocities: slow, preferred, and fast. Over these three velocities, EMA declined at the hip from the slow to the preferred velocity. Knee EMA also decreased slightly at the fastest measured velocity (Figure 6). Overall, Biewener et al., (2004) observed increases in active muscle volume across the three velocities (Figure 7). In a related study, McMahon et al., (1987) found that humans use more energy when they run in a crouched posture ('Groucho running') directly because of the poor mechanical advantage and increased muscle recruitment at the knee.
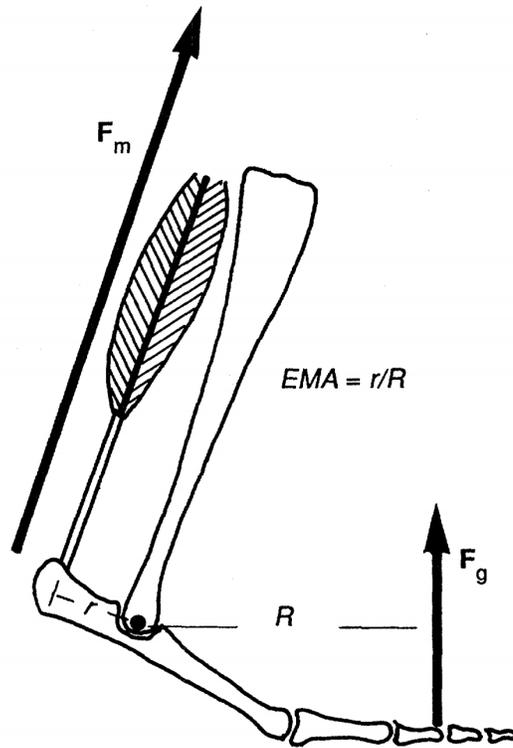
Figure 5. Example of effective mechanical advantage (EMA) of the ankle. EMA is defined as the ratio (r/R) of the weighted mean agonist muscle moment arm (r) of the muscle force (F_m) to the moment arm (R) of the ground reaction force (F_g). Data from Biewener 1989.
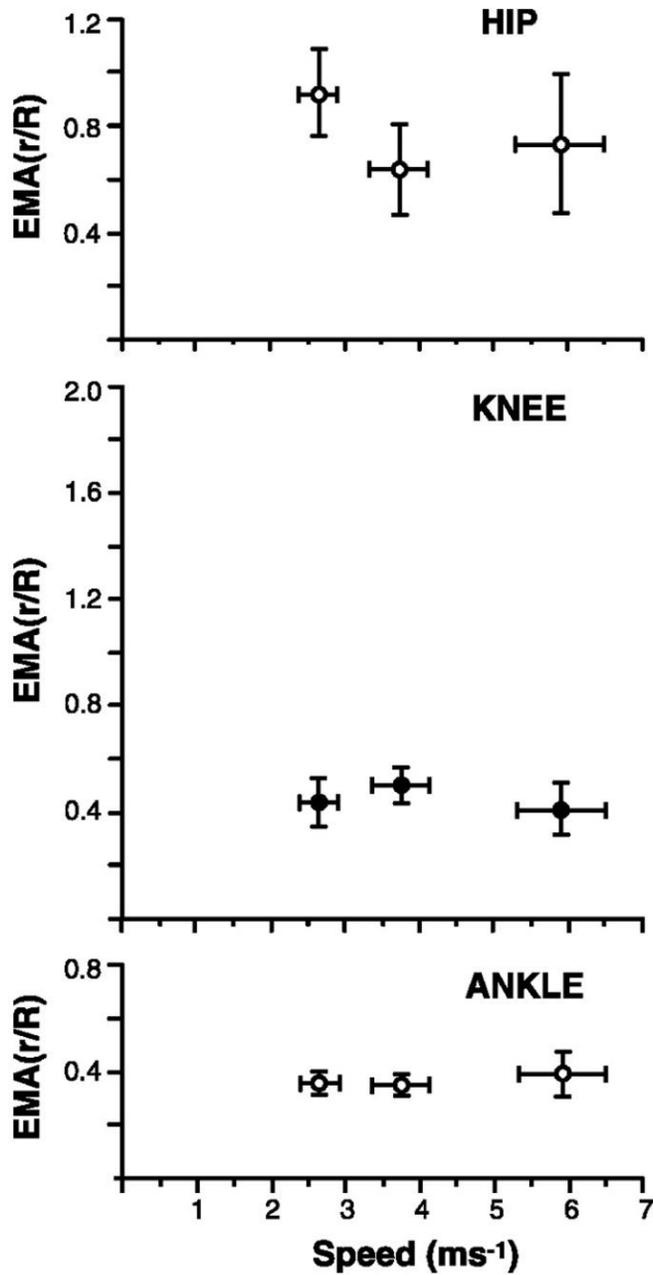
Figure 6. Changes in muscle EMA at the hip (top panel), knee (middle panel), and ankle (lower panel) joints as a function of speed for 1 subject. Over the three running speeds (~2.5- $5.75\,\mathrm{m\cdot s^{-1}}$) EMA declined slightly about the hip for the slow and preferred speed. Knee EMA declined slightly at the faster running speed. Data from Biewener et al., 2004.
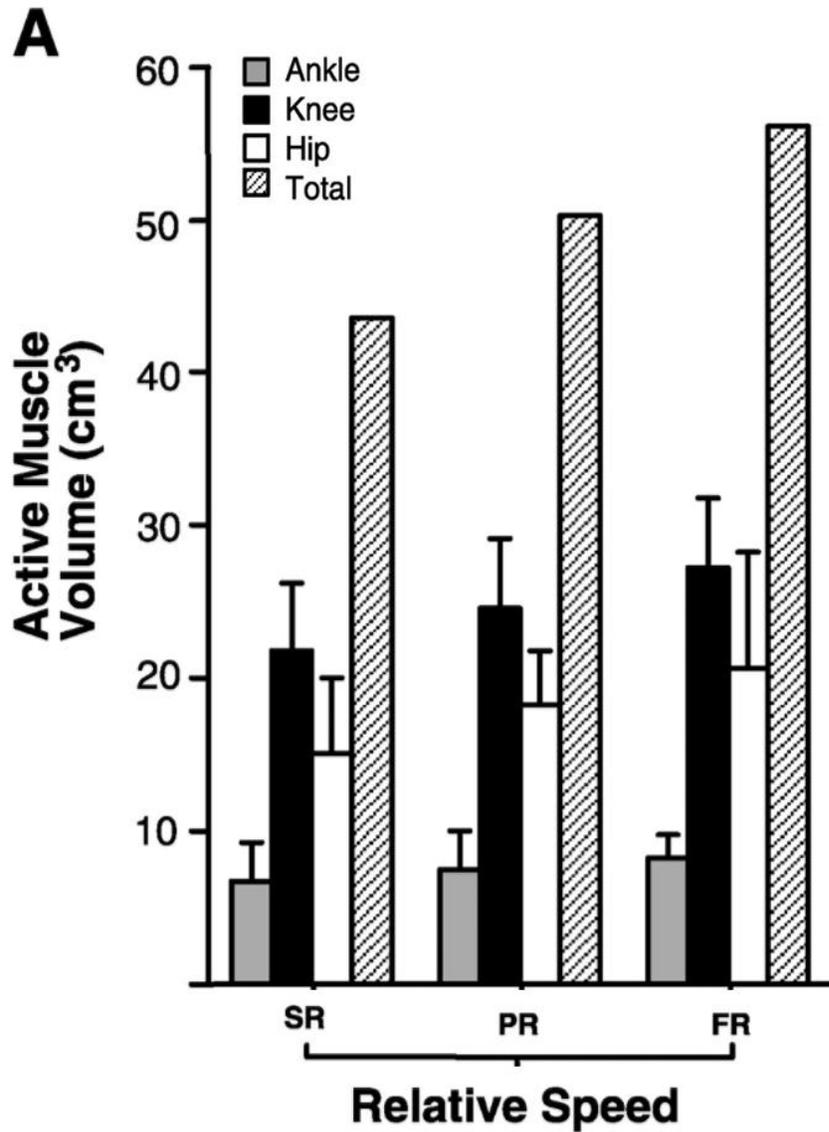
Figure 7. A: Estimated active volume of muscles at the hip, knee, and ankle joints increased over the three self-selected running speeds: slow running (SR), preferred running (PR) and fast running (FR) speed. Data from Biewener 2004. Note: it appears that a calculation error occurred because the muscle volumes are so small.

For humans, the cost of generating force hypothesis can explain about 80% of the increase in the rate of metabolic energy consumption, assuming that EMA and thus active muscle volume remains constant across running velocity (Roberts, 1998). Thus, it is likely that the assumption of constant EMA and thus constant active muscle volume is not a valid assumption for human runners. Changes in limb posture affect muscle forces by altering the mechanical advantage of the ground reaction force and therefore the EMA for muscle force production. A decrease in EMA across running velocities results in a greater magnitude of muscular force needed to support the body. It is possible that a more systematic analysis of EMA over a wide velocity range might explain the curvilinear increase in metabolic rate across the full sub-maximal velocity range that elite runners are capable of sustaining.

Chapter II:

Introduction

Nearly all terrestrial vertebrates exhibit a linear relationship between metabolic rate and running velocity (Taylor et al., 1970; Taylor et al., 1981; Heglund et al., 1982). Linear relationships have been reported for a diverse assortment of species including: ground squirrels and white rats (Taylor et al., 1970), chipmunks and turkeys (Heglund et al., 1982), domestic dogs (Zuntz, 1897; Slowtzoff et al., 1903; Taylor et al., 1981; Heglund et al., 1982), baboons, domestic cats, sheep, zebu cattle, wolves, waterbucks and wildebeests (Taylor et al., 1981). When comparing horses, humans, and dogs of different sizes, Zuntz (1897) (and later Taylor et al., 1970) noted that while each species showed a linear relationship between metabolic rate and velocity, smaller animals used more energy per unit of body weight to run a given distance compared to larger animals.

To explain the differences across body size, Taylor (1980) proposed the 'cost of generating force hypothesis'. His hypothesis was based on experiments that measured changes in metabolic rate when running animals carried extra weights. Their rates of oxygen consumption ($\dot{V}O_2$) increased in almost direct proportion to the weight supported. That is, a 10% increase in body weight increased the rate of O_2 consumption by 10%. Taylor noted that while there was an increase in O_2 consumption rate with the added weight, the average vertical acceleration of the center of mass did not change. That led Taylor to conclude that the average muscular force developed by the animal during the stance phase increased proportionally with the load. However, generating a Newton of force on the ground was much more costly for the smaller animals than the larger animals. Taylor reasoned that this was because smaller animals (e.g. a

mouse) must take many quick strides and generate force using fast, uneconomical muscle fibers

compared to larger animals. The differences in the metabolic cost of running between animals of

different sizes suggested that the time available for developing force is an important determinant

of the energy cost of running.

A decade later, Kram and Taylor (1990) refined the cost of generating force hypothesis to

also explain why metabolic rate increases at faster running velocities. They found that, for a

variety of species, the metabolic rate during running is determined by the rate of muscular force

production (calculated as the inverse of foot-ground contact time per step, $1/t_c$, where t_c= foot-

ground contact time) and the volume of muscle activated. Presumably, a shorter t_c requires

muscle fibers with faster intrinsic shortening velocities, due to high rates of crossbridge cycling

and faster myosin ATPase in the muscle cells. However, relying on faster muscle fibers is more

costly since they consume ATP at faster rates when generating force (Rall, 1985). Longer t_c

allows for the recruitment of slower, more economical fibers. The cost of generating force

hypothesis states that the rate of energy consumption per Newton of body weight (BW) is

inversely proportional to the time the foot is on the ground multiplied by a cost coefficient (c)

(Equation 1). The cost coefficient is nearly constant across velocity and BW for a diverse

assortment of mammals ranging in size, indicating that metabolic rate is inversely proportional to

the time that the foot is on the ground.

$$\dot{E}_{met,net} \cdot BW^{-1} = c \cdot 1/t_c \qquad \text{(Equation 1)}$$

While most terrestrial animals show a linear relationship between metabolic rate and

velocity over a wide velocity range, there is less agreement on how the relationship between

metabolic rate and running velocity should be mathematically characterized for running humans. Although many studies have found linear relationships for human runners (Margaria et al., 1963, DiPrampero et al., 1986, Helgerud, 1994, Roberts et al., 1998), Steudel-Numbers and Wall-Scheffler (2009) reported that metabolic rate increases curvilinearly with running velocity in well-trained distance runners during treadmill running at velocities ranging from ~2.01-4.9 m/s. Similarly, a re-analysis of Tam et al. (2012) produces a curvilinear relationship between metabolic rate and running velocities for elite human runners up to 5.0 m/s. Tam et al. suggested that their findings could be explained by the increasing contribution of aerodynamic resistance to the metabolic rate during their over-ground running protocol. However, that does not explain the curvilinear relationships for data collected on treadmills, which involve negligible aerodynamic resistance.

Most recently, Batliner et al. (2017) compared average and sub-elite human runners over a wider range of submaximal velocities. Average runners completed a velocity range of 1.78 to 4.08 m/s submaximally, while the sub-elite runners completed a velocity range of 1.78 to 5.14 m/s. Even though both the average and sub-elite runners completed a full range of submaximal velocities up to ~85% of their maximal aerobic capacity ($\dot{V}O_2$ max), the curvilinear increase in metabolic rate was only observed in the sub-elite runners, who were able to run faster than 4.08 m/s submaximally. This suggests that the sub-elite curvilinear increase was not caused by a physiological parameter such as muscle glycogen depletion or lactate accumulation, but by biomechanical factor(s).

To explain the increase in metabolic rate with running velocity in humans, Roberts et al. (1998) applied the Kram and Taylor cost of generating force approach. That model proposes that the major determinant of metabolic rate during running is the rate of muscular force production

($1/t_c$) and the volume of muscle activated. Roberts et al. assumed that EMA (and thus active muscle volume) remained constant across velocity. They found that the $1/t_c$ approach alone explained ~80% of the linear increase in metabolic rate in running humans across a moderate velocity range (2-4 m/sec).

In running animals, the volume of muscle activated is determined by the amount of muscle force the body must produce to support body weight and the architecture of the muscles. The amount of muscle force needed is determined by how the limbs act as a series of levers (Biewener, 1989). Each of the leg segments: thigh, shank, and foot, act as a lever with a fulcrum at a joint center. Over the stance phase, changes in limb posture affect the ground reaction force moment arm, R (defined as the perpendicular distance from the resultant ground reaction force vector to the respective joint center). The lever arm (perpendicular distance) of the muscle force relative to the joint center, defines the internal muscle-tendon moment arm (r). The effective mechanical advantage (EMA) is the ratio of these two moment arms, r/R.

Changes in EMA change the muscle force required and thus the amount of active muscle volume. Smaller EMAs require a greater muscle force to exert a specified force on the ground, requiring greater active muscle volume and thus presumably greater metabolic rates. This occurs when the limb segments are more flexed and less aligned with the resultant ground reaction force (GRF). When an animal's limb posture becomes more upright (straighter legs), the GRF becomes more aligned with the limbs and joint centers and the force that the muscles must exert to support body weight decreases.

Biewener (1989) showed that EMA does not vary significantly with changes in velocity or gait in quadrupeds. Thus, previous studies have assumed that EMA is constant over a wide range of velocities (Kram & Taylor, 1990; Roberts et al., 1998). However, in humans there is

evidence that EMA decreases, and thus active muscle volume increases, over both a velocity range and when switching from walking to running gaits (Biewener et al., 2004).

The cost of generating force hypothesis can explain about 80% of the increase in the rate of metabolic energy consumption in humans, assuming that EMA and active muscle volume remain constant across running velocity (Roberts, 1998). But, it is likely that the assumption of constant EMA and constant active muscle volume is not correct for human runners. Here, I performed a more systematic analysis of EMA over a wide velocity range to try and explain the remaining 20% and the curvilinear increase in metabolic rate across the full velocity range that elite runners are capable of sustaining. Specifically, I explored if the remaining 20% increase in metabolic rate can be attributed to EMA changes. My main purpose was to quantify how metabolic rate, the rate of force production, and active leg muscle volume change across a wide range of running velocities in order to more fully test the cost of generating force hypothesis in human runners. I hypothesized that at faster running velocities, EMA at the hip, knee and ankle joints would decrease, which necessitates a greater volume of active muscle recruitment. I incorporated an active muscle volume (V_m) term into the original cost of generating force hypothesis (Equation 1) and introduced a new version of the cost coefficient, c*:

$$\dot{E}_{met,net} \cdot BW^{-1} = c* \cdot 1/t_c \cdot V_m \qquad \text{(Equation 2)}$$

I hypothesized that c* would be constant across running velocity. Additionally I hypothesized that the curvilinear metabolic rate pattern observed in elite runners at fast sub-maximal velocities can be explained by a curvilinear increase in volume of active muscle recruitment.

Methods

Participants

Seven healthy human male runners participated (27.4±2.7 years, 64.6±4.6 kg, 178.8±5.8 cm). All participants could run a 10-kilometer race in less than 31 minutes at sea level, or less than 32 minutes at the local altitude (~1600 m). Participants gave written informed consent that followed the guidelines of the University of Colorado Boulder IRB.

Experimental set-up and protocol

Over two visits, subjects performed a series of running trials on a motorized, force-measuring treadmill with a rigid deck (Treadmetrix, Park City, UT). During their first visit, subjects habituated to the treadmill and expired gas set-up, and I verified that they were running submaximally. Subjects performed 5-minute running trials at 14, 16 and 18km/hr while breathing through a standard expired gas mouthpiece and wearing a nose clip. I obtained 50uL of venous blood from the subject's fingers to determine lactate concentration ([La]) at rest and at the completion of each 5-minute stage. Subjects rested for 5-minutes between each running trial. I monitored [La] to assure a primary reliance on oxidative metabolism at the three fastest velocities. Subjects who could run all three velocities with a blood lactate level below 4mmol (Heck, 1985) and an RER< 1.0 were considered capable of running at all velocities submaximally. I analyzed blood samples in duplicate with a YSI 2300 lactate analyzer (YSI, Yellow Springs, OH).

Following the three 5-minute running trials, I placed 40 reflective markers on the subject's lower limbs. I assigned subjects to a randomized order for six velocities: 8km/hr, 10km/hr, 12km/hr, 14km/hr, 16km/hr, and 18km/hr. Subjects then ran at each of these six velocities for 2 minutes with *ad libitum* rest in-between. I used a three-dimensional motion

capture system (Vicon 512 System, Oxford, UK) to determine the positions of the ankle, knee and hip joints of both legs relative to the force-measuring treadmill.

During a second visit, subjects arrived to the laboratory 2 hours post-prandial to help control for potential effects of diet on metabolic rate. With the subject shod and in their running attire, I measured their weight with a beam scale. Subjects ran at the same six velocities as the first visit in the same randomized order. Trials lasted 5-minutes for each velocity, and subjects took 5-minute breaks between trials. After completing the 6 submaximal trials, subjects recovered for 10-minutes and then completed a $\dot{V}O_2$ max test. Subjects ran at 16km/hr on a level treadmill for one minute. Then, I increased the grade by 1% each minute until subjects reached voluntary exhaustion.

Data Analyses
Physiology

During the experimental trials, I measured rates of oxygen uptake and carbon dioxide production with an open circuit expired gas analysis system (Parvomedics TrueOne 2400, Sandy, UT). I then calculated metabolic rates (Watts) using the energetic equivalents (Brockway, 1987) from the last 2-minutes of each trial when metabolic power had reached steady state. I defined $\dot{V}O_2$ max as the greatest 30-second mean value obtained. My criteria for reaching $\dot{V}O_2$ max was a plateau in oxygen consumption and/or a respiratory exchange ratio (RER) over 1.15 (Issekutz, 1962).

Mechanics

I collected ground reaction forces (GRF) at 1000 Hz and limb kinematics at 200Hz for 10 strides (20 steps) during the last 30 seconds of each trial (Vicon 512 System, Oxford, UK). I used

a critically damped low-pass filter (12 Hz) for GRF and a Butterworth low-pass filter (7 Hz cutoff frequency) for target-marker data (Visual 3D software, C-Motion Inc., MD, USA). I calculated stride kinematic data (i.e. t_c, stride frequency) from GRF traces using a custom MATLAB script (MathWorks Inc, Natick, MA).

I determined muscle force (F_m) acting at each joint using equation 3 during the stance phase by averaging joint moments when they were greater than 25% of their maximum ($M_{net\ joint}$) and dividing by the muscle moment arm (r). I determined r by palpation of muscle attachments relative to estimates of joint centers for each subject and compared it to direct measures from cadavers. Even though our measures were similar to the cadavers, I used the more precise measurements taken from the cadavers.

$$F_m = M_{net\ joint} / r \hspace{3cm} \text{(Equation 3)}$$

I calculated EMA about the hip, knee and ankle joints as the ratio (r/R) of the muscle moment arm (r) to the ground reaction force (GRF) moment arm (R), over the same period of the stride used to calculate F_m (Biewener, 1989; Roberts et al., 1998).

$$EMA = r/R = GRF/F_m \hspace{3cm} \text{(Equation 4)}$$

To estimate the volume of actively recruited muscle (V_m, cm^3) at each joint during stance, I used the morphological data of lower extremity muscles of male human cadavers from Biewener et al. (2004). The weighted average of fascicle length (L, cm) was determined from multiple agonist muscles (Table 1). I assumed that muscles exert an equivalent force per section

of cross sectional area of active fibers (σ) of 20 N/cm^2 based on Perry et al. (1988).

$$V_m = LF_m / \sigma \qquad \text{(Equation 5)}$$

Statistics

I calculated means and standard deviations (mean ± SD) for all tested variables and tested for normality using the Shapiro-Wilk normality test. I fit individual subject linear and 2nd order curvilinear regressions to the metabolic power vs. velocity values and used R^2 values for each subject to assess the strength of both regression methods. I used a paired samples t-test to compare the means of individual R^2 values for linear and curvilinear fits.

To determine whether the slope of both calculated cost coefficient regression lines differed significantly from zero, I used linear regression t-tests. I considered results significant at a $p < 0.05$. I performed statistical analyses using RStudio (version 0.99.892, Boston, MA, USA) software.

Table 1. Muscle data from 4 male cadavers (mean age 78 years) used from Biewener et al., 2004. Volumes assume a constant muscle density of $1.06 g/cm^3$ (Mendez and Keys, 1960).

Joint and Muscle	Mean r (cm)	Mass (kg)	Volume (cm^3)	Fascicle Length (cm)
Hip				
Gluteus maximus		0.481	453.7	18.7
Semitendinosus		0.109	102.8	14.3
Semimembranosus		0.168	158.5	7.4
Biceps femoris		0.148	139.6	8.3
Total	**5.7**	**0.906**	**854.7**	**11.7**
Knee				
Vastus lateralis		0.410	386.8	8
Vastus intermedius		0.224	211.3	7.3
Vastus medialis		0.248	233.9	8.4
Rectus femoris		0.128	120.8	7.2
Total	**5.5**	**1.01**	**952.8**	**7.3**
Ankle				
Lateral gastrocnemius		0.094	88.7	5.5
Medial gastrocnemius		0.155	146.2	4.6
Soleus		0.326	307.5	4.1
Flexor digitorum longus		0.022	20.8	4.4
Tibialis posterior		0.068	64.2	3.2
Flexor digitorum longus		0.063	59.4	5.1
Peroneus Longus		0.064	60.4	5.5
Peroneus Brevis		0.028	26.4	3.8
Total	**3.7**	**0.82**	**773.6**	**4.1**

Results

　　Across the velocity range, every subject's gross metabolic power increased curvilinearly by more than two-fold (Fig. 1). The R^2 values for every subject's curvilinear fits for metabolic power vs. velocity (average R^2=0.999) were statistically greater than the R^2 values for linear fits (average R^2=0.981) (p<0.05). At 18km/hr, subjects' oxygen uptakes averaged 82.7% of their $\dot{V}O_2$ max values (average $\dot{V}O_2$ max = 72.0 +/- 3.3 mlO2/kg/min). Further, at 18km/hr average RER was 0.94 +/- 0.03, and average blood lactate concentration was 3.56 ± 0.34 mmol. No subject exceeded an RER of 1.0 or a blood lactate value of 4.0mmol. Additionally I compared the last two minutes of each trial to rule out a contribution of a slow component. I found no significant difference when comparing minute 3-4 to minute 4-5, indicating the subjects had reached steady state. Thus, it is clear that the trials were all sub-maximal and at steady-state. Table 2 reports all metabolic variables.
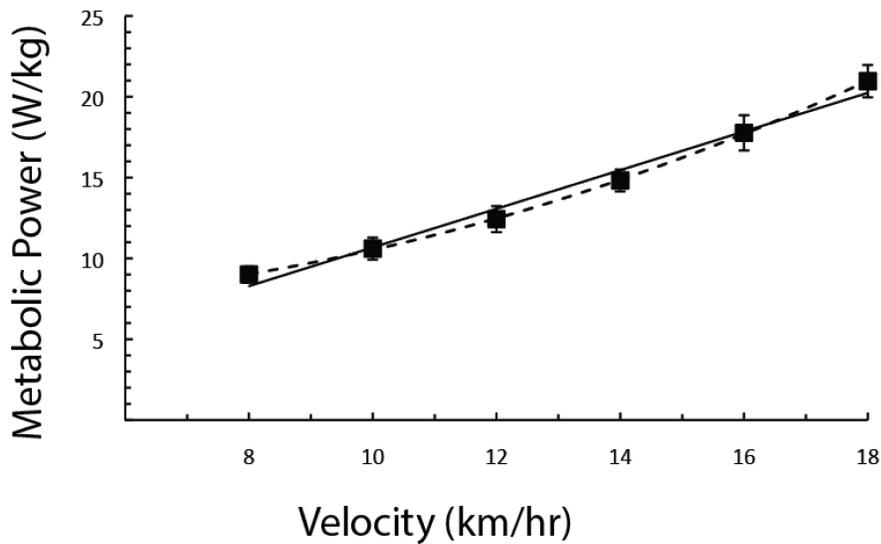
Fig. 1 Metabolic rate ($\dot{E}_{met,gross}$) vs. velocity (v) in km/hr. The solid line represents a linear least squares regression of the data ($\dot{E}_{met,gross}$=1.19v-1.28; R^2=0.981). The dashed line represents a 2nd order polynomial regression ($\dot{E}_{met,gross}$=0.055v^2-0.251v+7.47; R^2=0.999). Equivalent equations in m/s are as follows, linear fit: $\dot{E}_{met,gross}$=4.31v-1.28; 2nd order polynomial regression: $\dot{E}_{met,gross}$=0.721v^2-0.904v+7.47. Error bars indicate ±1 SD.

Ground contact time (t_c) decreased over the velocity range, while the rate of force production ($1/t_c$) increased (Fig 2). The R^2 values for curvilinear fits for both ground contact time (t_c) and rate of force production ($1/t_c$) vs. velocity (R^2=0.990;R^2=0.996) were not statistically different than the R^2 values for linear fits (R^2=0.996; R^2=0.998) (p>0.05) (Fig. 2).

Vertical ground reaction force peaks were greater at faster velocities (Fig. 3). Several individuals transitioned from a rearfoot to a midfoot strike pattern at the fastest velocities, as indicated by a disappearance of the impact peaks in the vertical GRF trace. All other subjects maintained their same footstrike pattern over the entire range of velocities.
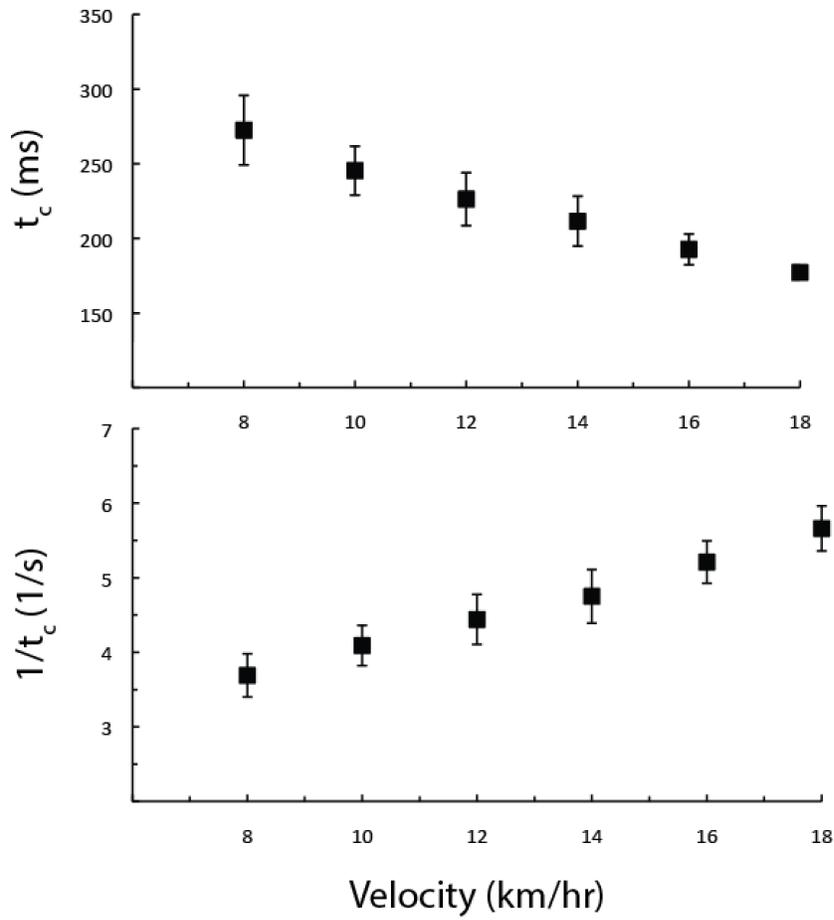
Fig. 2 Ground contact time (t_c) and rate of force production ($1/t_c$) across velocity. Error bars indicate ±1 SD.
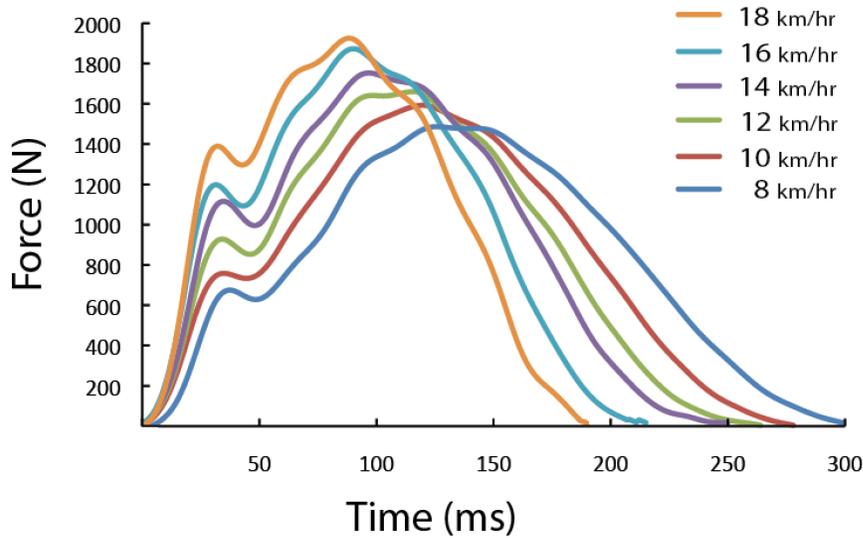
Fig. 3 Example vertical GRF traces vs. time over the velocity range for one subject.

Mean net joint moments ($M_{net\ joint}$) increased with velocity at the ankle and hip (Fig 4). However, at the knee, $M_{net\ joint}$ increased up to 14 or 16 km/hr and then slightly decreased (Table 4). I observed these trends in every subject. Accordingly, ankle EMA decreased by 14.5 ± 4.1%, while hip EMA showed the largest magnitude decrease of 51.2 ± 30.2%, over the velocity range. Knee EMA showed less of a distinct decrease due to the joint moment decreasing at the faster velocities. (Fig. 5).
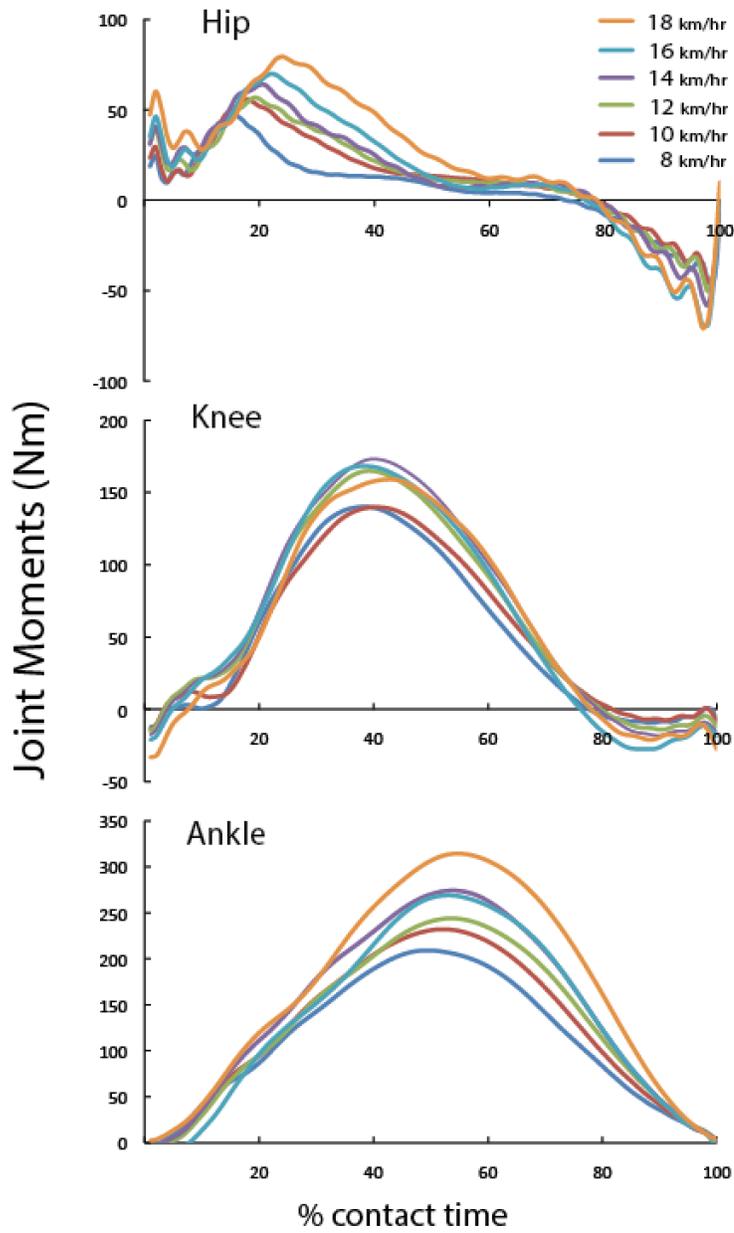
Fig. 4 Example traces of net joint moments over the velocity range at the hip, knee and ankle. Positive values indicate net extensor muscle moments, while negative values are net flexor muscle moments.
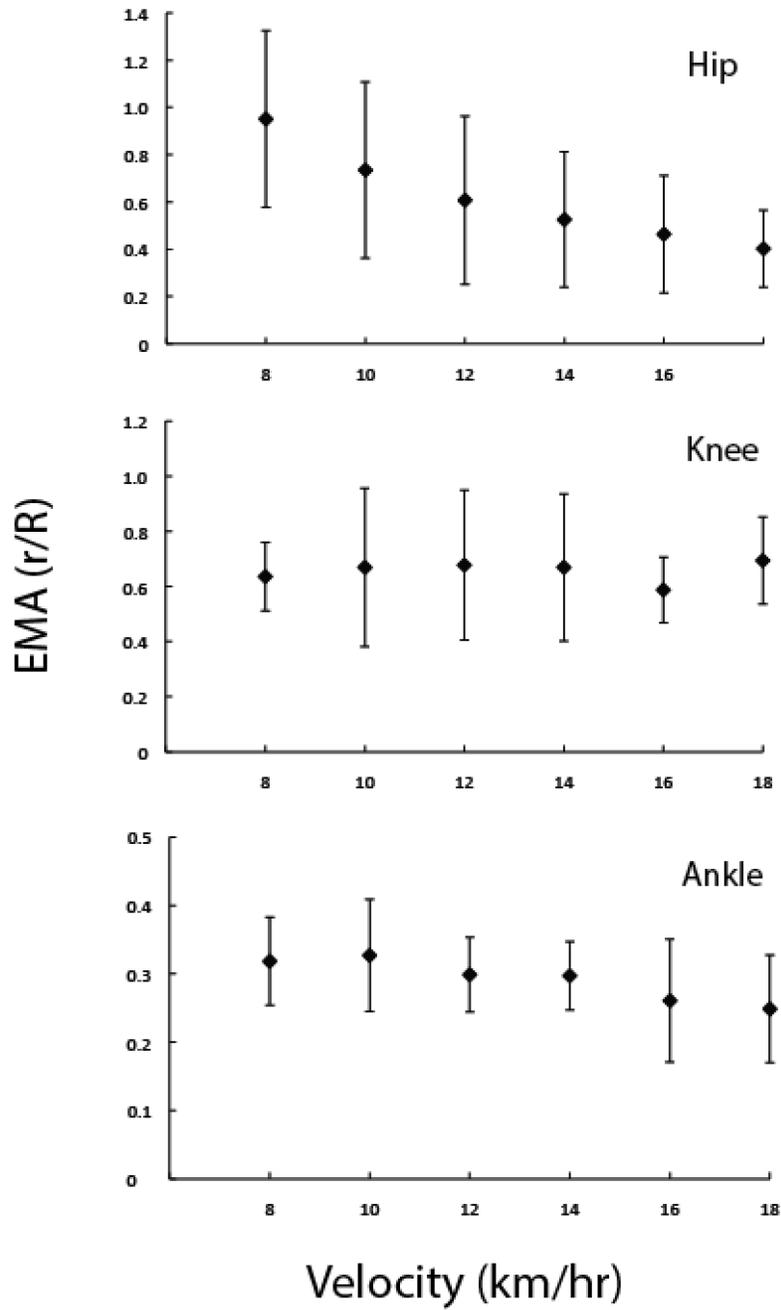
Fig. 5 EMA across velocity for ankle, knee, and hip joints. Error bars indicate ±1 SD.

Total mean active muscle volume increased across velocity by $31.5 \pm 2.6\%$ across the velocity range (Fig. 6). Active muscle volume increased at the ankle and hip $32.6 \pm 5.1\%$ and $45.8 \pm 6.2\%$, respectively across the velocity range, while slightly decreasing at the knee. The ankle's contribution to the total active muscle volume only increased 1% from 8km/hr to 18km/hr. while the hip was the largest contributor, increasing from 26.6% of the total active muscle volume at 8km/hr, to 33.8% of the total active muscle volume at 18km/hr.
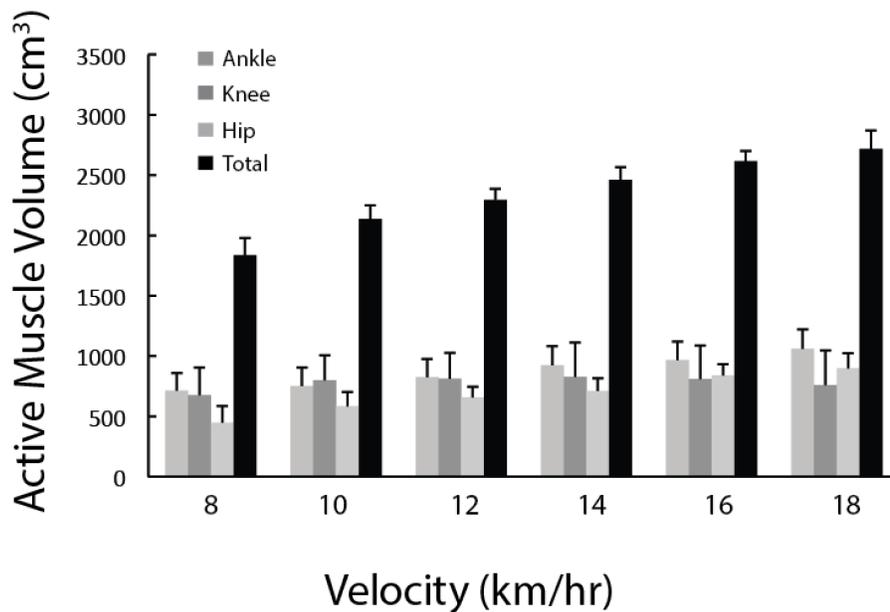
Fig. 6 Mean estimated active muscle volume across velocity for ankle, knee, and hip extensor muscles. Error bars indicate ±1 SD.

I calculated the classic cost coefficient (c) for each velocity by dividing the mean net metabolic power ($\dot{E}_{met,net}$) normalized to body weight (BW) by $1/t_c$ as proposed by Kram and Taylor (1990) (Equation 1). In this equation, $\dot{E}_{met,net}$ was calculated by subtracting the y-intercept. Furthermore, I calculated a new cost coefficient (c*) by additionally dividing by V_m (Equation 2).

$$\dot{E}_{met,net} \cdot BW^{-1} = c* \cdot 1/t_c \qquad\qquad \text{(Equation 1)}$$

$$\dot{E}_{met,net} \cdot BW^{-1} = c* \cdot 1/t_c \cdot V_m \qquad\qquad \text{(Equation 2)}$$

However, because gross metabolic power takes into account all of the energy being expended as a subject runs, I calculated two additional versions of the cost coefficients (c' and c'*) based upon the mean gross metabolic power, $\dot{E}_{met,gross}$ (Equation 6 and 7).

$$\dot{E}_{met,gross} \cdot BW^{-1} = c'* \cdot 1/t_c \qquad\qquad \text{(Equation 6)}$$

$$\dot{E}_{met,gross} \cdot BW^{-1} = c'* \cdot 1/t_c \cdot V_m \qquad\qquad \text{(Equation 7)}$$

Linear regression t-tests showed that the slopes for c and c', were significantly different from zero ($p<.001$), while the new cost coefficients, c* and c'*, were nearly constant across the velocity range, and not statistically different ($p=0.176$) (Fig 7).

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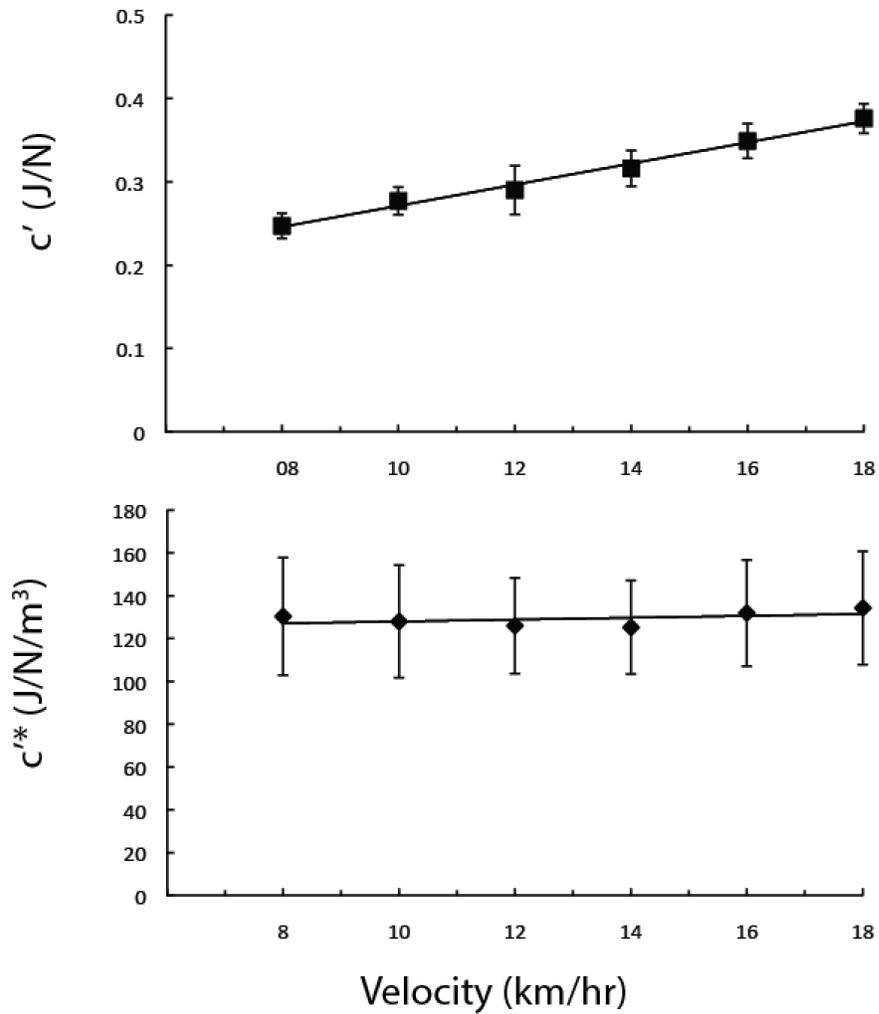
Fig. 7. The cost coefficient (c') vs. the new cost coefficient (c'*), calculated using gross metabolic power. Equations for the cost coefficients across velocity, v: c'= 0.013v + 0.144; c'*= 0.439v + 123.6. Error bars indicate ±1 SD.

Table 2. Metabolic variables for 7 subjects across velocity (mean ± SD).

Velocity (km/hr)	$\dot{V}O_2$ (L/min)	$\dot{V}O_2$ (ml/kg/min)	RER	Metabolic Power (W)	Metabolic Power (W/kg)
Rest	0.33 ± 0.04	5.1 ± 0.5	0.851 ± 0.094	112.5 ± 11.7	1.75 ± 0.16
8	1.69 ± 0.15	26.3 ± 1.7	0.821 ± 0.048	580.4 ± 38.0	9.00 ± 0.49
10	1.99 ± 0.16	30.8 ± 1.7	0.829 ± 0.037	683.1 ± 49.3	10.6 ± 0.68
12	2.33 ± 0.24	36.1 ± 2.7	0.841 ± 0.031	801.8 ± 69.0	12.4 ± 0.80
14	2.77 ± 0.23	42.9 ± 2.3	0.851 ± 0.029	956.0 ± 65.9	14.8 ± 0.68
16	3.29 ± 0.27	51.1 ± 3.1	0.888 ± 0.041	1145 ± 71.9	17.8 ± 1.09
18	3.84 ± 0.33	59.5 ± 3.3	0.941 ± 0.034	1351 ± 89.2	21.0 ± 0.99

Table 3. Biomechanical variables for 7 subjects across velocity (mean ± SD).

Velocity (km/hr)	t_c (ms)	Peak Vertical GRF (N)	Peak Vertical GRF (BW)	Stride Frequency (strides/s)
8	272.6 ± 23	1530 ± 148	2.43 ± 0.23	1.35 ± 0.03
10	245.5 ± 16	1593 ± 124	2.53 ± 0.21	1.36 ± 0.03
12	226.2 ± 17	1670 ± 129	2.66 ± 0.25	1.39 ± 0.03
14	211.5 ± 16	1783 ± 156	2.83 ± 0.28	1.42 ± 0.05
16	192.6 ± 10	1820 ± 152	2.89 ± 0.25	1.47 ± 0.04
18	177.1 ± 9	1895 ± 142	3.01 ± 0.22	1.52 ± 0.05

Table 4. Net joint moments for the Ankle, Knee and Hip. Net joint moments are defined as the average joint moments for the period when the moments are greater than 25% of the peak joint moment at that velocity. Ground contact time (t_c) and peak vertical ground reaction force (GRF) in units of body weight (BW).

Velocity (km/hr)	Net Joint Moment (Nm)		
	Ankle	Knee	Hip
8	128.7 ± 26.2	101.7 ± 34.5	43.8 ± 13.4
10	135.8 ± 27.5	120.6 ± 31.0	51.8 ± 11.4
12	148.7 ± 27.4	122.6 ± 32.2	56.2 ± 8.5
14	166.5 ± 28.9	124.7 ± 43.0	61.8 ± 10.2
16	174.4 ± 27.8	122.1 ± 41.8	70.1 ± 9.0
18	191.6 ± 28.7	114.2 ± 43.4	76.1 ± 12.2

Discussion

I accept my overall hypothesis that the increase in metabolic power at faster running velocities can be explained by increases in the rate of force generation and the active muscle volume. The new cost coefficient, c*, from equation 2 was nearly constant across the velocity range, indicating that both the rate of force production ($1/t_c$), and active leg muscle volume together can explain the energetic requirements of running.

I observed every subject's gross metabolic power increase curvilinearly by more than two-fold across the velocity range. This is in agreement with previous studies who reported a nonlinear increase in metabolic rate for good to elite runners over a wide range of velocities (Steudel-Numbers & Wall-Scheffler, 2009; Batliner et al., 2017; Tam, 2012,). My second objective was to explain the curvilinear metabolic rate pattern observed in elite runners at fast sub-maximal velocities. Because active muscle volume increased linearly, I reject my second

hypothesis; the curvilinear increase cannot be attributed to a curvilinear increase in volume of active muscle recruitment. Additionally, I tested the relationship between the rate of force development $(1/t_c)$ and velocity and found no difference between linear and curvilinear fits. Thus, neither the active muscle volume or the rate of generating force alone can explain the curvilinear increase in metabolic cost.

I extended previous studies that suggested changes in the effective mechanical advantage of the lower limb could influence the metabolic cost of running (Biewener, 1990, Full et al., 1990; McMahon et al., 1987). From 8 to 18km/hr, ankle EMA decreased by 14.5 ± 4.1%, while hip EMA showed the largest magnitude decrease of 51.2 ± 30.2%. Accordingly, the ankle extensors increased active muscle volume by 32.1% from 712 ± 146cm^3 to 1049 ± 157cm^3, while the hip extensors muscles showed the greatest increase in the active muscle volume needed to generate force on the ground, increasing 45.8% from 489 ± 126 cm^3 to 898 ± 174cm^3 across the velocity range. The hip muscles also increased their contribution to the total active muscle volume from 26.6% to 33.8% over the velocity range. The knee extensor muscle volume only increased a modest 8.7%, likely due to the decreasing moments at the faster velocities. Biewener et al., (2004) also showed an increase in active muscle volume at all three joints over three running velocities. However, in contrast to my results, they showed the active muscle volume at the knee comprises 48% of the total active muscle volume, while the hip and ankle made up 36% and 16% of the total active muscle volume. The magnitudes of the active muscle volume reported by Biewener et al., (2004) are puzzlingly small and are almost surely a calculation error.

Additionally, the relative contributions of ankle/knee/hip muscles that I measured for running are not similar to those for walking. Griffin et al. (2003), reported that active muscle volume at the ankle made up 50% of total active muscle volume, and the hip made up 32% of

total volume at all walking velocities (0.5- 2.0m/s). This difference is likely due to the fundamental difference in limb postures in walking (straight knee) vs. running (bent knee).

A limitation of this study was the use of muscle data obtained from cadavers. Although all of the cadavers were reportedly in good musculoskeletal health at the time of their death, given their average age (78 years) and my calculations of their total muscle volume, it is evident that substantial muscle mass had been lost due to age. My young healthy subjects were activating more muscle volume at the fastest velocity (mean $2658.8 \pm 169cm^3$) than the cadavers had available ($2581.1cm^3$). However, my analysis of how active muscle volume varies as a function of velocity depends only on the relative size and architecture of the muscles within the limb, which are less likely to be affected by age and muscle atrophy.

I did not look at differences in EMA and active muscle volume in average or recreational populations. But because most biomechanics variables such as stride length, stride frequency and contact time do not differ significantly between elite and good recreational runners (Cavanagh et al., 1977), I would not expect significant differences in EMA between the two populations. I would still predict that both the rate of force generation ($1/t_c$) and active muscle volume to better predict the energetic cost of running than just $1/t_c$ for the average to good running populations.

Biewener (1989) showed that EMA does not vary significantly with gait changes in quadrupeds. Thus, further research should seek to establish if a decrease in EMA occurs with other bipedal species (i.e. birds) that cannot switch between walking, trotting or galloping gaits like quadrupeds can.

In conclusion, I sought to evaluate changes in limb mechanical advantage and their effect on the muscle force-generation requirements via estimated active muscle volume in humans as running velocity increases. I calculated a new cost coefficient for approximating metabolic rate

that accounts for active muscle volume changes. The new cost coefficient was nearly constant across the velocity range, indicating that both the rate of force production ($1/t_c$), and active leg muscle volume together better explain the energetic requirements of running.

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