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The Oxygen Consumption and Metabolic Cost of Walking and Running in Adults With Achondroplasia

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The disproportionate body mass and leg length of Achondroplasic individuals may affect their net oxygen consumption ($\dot{V}O_2$) and metabolic cost (C) when walking at running compared to those of average stature (controls). The aim of this study was to measure submaximal $\dot{V}O_2$ and C during a range of set walking speeds (SWS; 0.56 – 1.94 m·s$^{-1}$, increment 0.28 m·s$^{-1}$), set running speeds (SRS; 1.67 – 3.33 m·s$^{-1}$, increment 0.28 m·s$^{-1}$) and a self-selected walking speed (SSW). $\dot{V}O_2$ and C was scaled to total body mass (TBM) and fat free mass (FFM) while gait speed was scaled to leg length using Froude’s number (Fr). Achondroplasic $\dot{V}O_{2\text{TBM}}$ and $\dot{V}O_{2\text{FFM}}$ were on average 29 and 35% greater during SWS ($P < 0.05$) and 12 and 18% higher during SRS ($P < 0.05$) than controls, respectively. Achondroplasic C$_{\text{TBM}}$ and C$_{\text{FFM}}$ were 29 and 33% greater during SWS ($P < 0.05$) and 12 and 18% greater during SRS ($P < 0.05$) than controls, respectively. There was no difference in SSW $\dot{V}O_{2\text{TBM}}$ or $\dot{V}O_{2\text{FFM}}$ between groups ($P > 0.05$), but C$_{\text{TBM}}$ and C$_{\text{FFM}}$ at SSW were 23 and 29% higher ($P < 0.05$) in the Achondroplasic group compared to controls, respectively. $\dot{V}O_{2\text{TBM}}$ and $\dot{V}O_{2\text{FFM}}$ correlated with Fr for both groups ($r = 0.984 – 0.999, P < 0.05$). Leg length accounted for the majority of the higher $\dot{V}O_{2\text{TBM}}$ and $\dot{V}O_{2\text{FFM}}$ in the Achondroplasic group, but further work is required to explain the higher Achondroplasic C$_{\text{TBM}}$ and C$_{\text{FFM}}$ at all speeds compared to controls.

**New and Noteworthy:** There is a leftward shift of oxygen consumption scaled to total body mass and fat free mass in Achondroplasic adults when walking and running. This is nullified when talking into account leg length. However, despite these scalars, Achondroplasic individuals have a higher walking and metabolic cost compared to age matched non-Achondroplasic individuals, suggesting biomechanical differences between the groups.

**Keywords:** Achondroplasia, oxygen consumption, metabolic cost, walking, running
INTRODUCTION

Achondroplasia is a condition identified by a disproportionate shorter limb length relative to torso length compared to age matched average statured groups, hereafter referred to as ‘controls’ (Hoover-Fong et al., 2007). The absolute shorter lower limbs of the Achondroplastic population compared to controls is likely to affect functional tasks, such as walking, which in turn is likely to affect the oxygen consumption (\(\dot{V}O_2\)) and metabolic cost of locomotion (\(C\)). For example, increased stride frequency is observed in proportionally shorter statured groups compared to taller individuals during walking and running at the same speeds (Minetti et al., 1994). This in turn leads to a higher \(\dot{V}O_2\) in the shorter groups at set walking speeds (Rowland and Green, 1988). When gait speed is increased, such as during incremental walking and running, a positive curvilinear trend of absolute \(\dot{V}O_2\) exists in numerous cohorts (Rowland and Green, 1988; Minetti et al., 1994; Schepens et al., 2004; van den Hecke et al., 2007). In proportionally shorter statured groups there is a higher \(\dot{V}O_2\) compared to taller groups when walking and running at set speeds (Rowland and Green, 1988; Minetti et al., 1994; Schepens et al., 2004; Ludlow and Weyand, 2015). The differences in \(\dot{V}O_2\) between groups of different size can be accounted somewhat by total body mass (TBM) and fat free mass (FFM) (Goran et al., 2000; Browning et al., 2006). Scaling horizontal speed to leg length, as done with Froude's number (\(Fr\)), further nullifies the observed difference in \(\dot{V}O_2\) between shorter and taller groups during incremental or steady state exercise (Ferretti et al., 1991; Minetti et al., 1994; Steudel and Beattie, 1995; Steudel-Numbers et al., 2007; Kramer and Sylvester, 2012). The size, and therefore mass, of the torso between Achondroplastic individuals and controls is similar (Owen et al., 1990). But with the leg length of Achondroplastic individuals being shorter than controls (Horton et al., 1978), the ratio of torso-to-leg mass is likely to be greater in the Achondroplastic group, which has been shown to lead to a higher \(\dot{V}O_2\) and \(C\) (Griffin et al., 2003; Browning et al., 2006; Beeley et al., 2007; Peyrot et al., 2009; McCormick, 2014). Furthermore, the shorter Achondroplastic leg would likely lead to a higher \(\dot{V}O_2\) than controls when exercising at the same speed, as observed in other shorter to taller comparisons (Minetti et al., 1994). Therefore, separately scaling \(\dot{V}O_2\) to either leg length or body mass during incremental exercise would likely under- and over-predict an Achondroplastic individual’s \(\dot{V}O_2\) when compared to controls. To the authors’ knowledge though, there are no data pertaining to the measurement of \(\dot{V}O_2\) in Achondroplastic individuals during incremental exercise let alone the scaling of \(\dot{V}O_2\) during incremental exercise.

\(\dot{V}O_2\) is useful to describe the cardiovascular response during exercise, but \(C\) describes the oxygen demand over a given distance (Kramer and Sarton-Miller, 2008). For the same incremental walking that exhibits a positive trend of \(\dot{V}O_2\) described above, a U-shaped curve of \(C\) exists with clear local minima (Ferretti et al., 1991; Minetti et al., 1994; McCann and Adams, 2002; Kramer and Sarton-Miller, 2008). This minima suggests that respective slower and faster walking speeds are less economic, or have a higher \(C\) (i.e., more \(\dot{V}O_2\) is required for the given distance). A local minima is observed at different speeds when stride frequency is manipulated, suggesting an optimal stride frequency for different speeds (Minetti et al., 1995). The local minima of \(C\) during walking is observed around self-selected walking (SSW) speed (Ralston, 1958), but has not been measured alongside set walking speeds in adults groups of shorter stature (Ferretti et al., 1991; Minetti et al., 1994, 2002). In groups of shorter stature, their higher \(C\) can be accounted for by their higher stride frequency (Minetti et al., 1994). The inclusion of a leg length scaler, like Fr, can nullify the effect of stride frequency while SSW may explain some of the expected U-shape curve of walking \(C\) in Achondroplasia and controls.

Therefore, the overriding aim of this study was to observe the relationship between \(\dot{V}O_2\) and incremental walking and running in adults with Achondroplasia. The primary objectives were to: (1) collect submaximal \(\dot{V}O_2\) in Achondroplastic adults and controls at differing absolute and relative (SSW) intensities of walking and running; (2) convert \(\dot{V}O_2\) into \(C\) values and; (3) attempt to account for any differences in \(\dot{V}O_2\) and \(C\) by normalizing to body masses and leg length. It was hypothesized that Achondroplasia would have a higher \(\dot{V}O_2\) and \(C\) at all walking and running speeds, but differences would nullify once scaled appropriately to mass and leg length.

MATERIALS AND METHODS

Participants

Ten Achondroplasic adults and 17 age matched controls free from any lower limb injury volunteered to participate in the study (Table 1). All gave written informed consent, which was approved by the local committee (Manchester Metropolitan University) and conformed with the Helsinki declaration. All participants attended one laboratory session at Manchester Metropolitan University where anthropometric variables and \(\dot{V}O_2\) were collected during incremental exercise.

Anthropometric Measures

Leg length (m) of all participants was measured as the distance from the anterior iliac spine to the medial malleolus of the ankle while standing. Participants’ TBM (kg) was obtained using electronic scales (SECA 813, CA 91710 Chino, United States) while barefooted and wearing minimal clothing. FFM was measured using Dual Energy x-ray absorptiometry (DEXA). Following a fasting period of ~8 h, participants were positioned...
on a DEXA scanner (Hologic Discovery, Vertec Scientific Ltd, United Kingdom) to measure FFM (kg) of the whole body. Briefly, participants wore a loose-fitting cotton gown and lay supine in a predefined position that ensured enough space was between each arm and the torso and between each leg. The feet were positioned in an internally rotated position and to maintain participant comfort, and reduce the muscle activity during the scan, medical tape (Transpore™ Medical Tape, 3M™, United States) was wrapped around both feet. A default whole body scan (EF 8.4 lSv) was selected for all trials; the scan emitted duel energy (140/100 kVp) fan-beam x-rays and lasted for ~7 min. The scanning region was adjusted depending on stature (Achondroplasia ~160 cm x 65 cm; controls ~195 cm x 65 cm) with 1.3 cm line spacing and a 0.2 cm point resolution and each participant was exposed to ~8.4 µSv (Blake et al., 2006). Following all scans manual segmentation of the body was completed using Physician’s View v6.1 software in accordance with Hologic’s recommendations. Glickman et al. (2004) showed that DEXA gives a reliable measurement of TBM (r = 0.940) and FFM (r = 0.890) against computer tomography, while the interrater reliability has been shown to be in excess of 0.998 (Hart et al., 2015).

**Speed of Locomotion**

Selected walking speed trials were completed by participants conforming to a habitual walking pace around the laboratory (~40 m). Each participant passed through two timing gates (1 m apart), three times. SSW speeds (m·s⁻¹) were calculated and recorded as an average of the three trials and used in VO₂ assessment where individuals’ SSW intersected absolute speeds described below. VO₂ collection apparatus (described in the next section) was worn throughout all exercise trials which were conducted on a motorized treadmill (Woodway PPS70). Treadmill speeds were set at 0.56 – 1.94 m·s⁻¹ (increment 0.28 m·s⁻¹) for walking and 1.67 – 3.33 m·s⁻¹ (increment 0.28 m·s⁻¹) for running, as described in Ferretti et al. (1991) and Minetti et al. (1994, 2002). All trials were completed at 1% gradient to replicate outdoor conditions (Jones and Doust, 1996) with each stage being 4 min in duration to attain steady state, again replicating Ferretti et al. (1991) and Minetti et al. (1994, 2002). Participants rested for ~5 min following all walking trials to reduce VO₂. Where participants could not maintain running speed for the entirety of the 4 min during any walking and running intensity, the stage was omitted from analysis and the testing protocol terminated.

**Oxygen Uptake, Metabolic Cost and Scaling**

Expired gasses were collected and analyzed using portable breath-by-breath indirect calorimetry (Metamax 3B, Cortex, Leipzig Germany), which was calibrated to the manufacturer’s guidelines prior to testing. The portable indirect calorimeter (weight = 1 kg) and a fitted face mask (Hans Rudolph V2, dead space between 125 – 143 ml) were worn by participants during the exercise bout. Prior to exercise testing, participants lay supine for 5 min so that resting metabolic rate (L·min⁻¹) could be measured. Gross VO₂ for each intensity was recorded with net VO₂ calculated by subtracting resting metabolic rate from gross VO₂ as conducted in Minetti et al. (1994, 2002), hereafter net VO₂ is referred to as ‘˙VO₂’. Steady state ˙VO₂ was determined by a respiratory exchange ratio < 1.0 and by a visual plateau of VO₂ over the final minute of exercise, with VO₂ recorded as a rolling average of 6 measurements (every 10 s) for each exercise intensity. C was presented as the amount of VO₂ required to complete 1 km at each gait speed, given as (L·km⁻¹). VO₂ and C were then normalized to TBM (VO₂TBM and C₇TBM, respectively) and FFM (VO₂FFM and CFFM, respectively). All VO₂ and C values were presented against absolute walking and running speeds, and against dimensionless Fr, given as: velocity² (m·s⁻²) ÷ leg length (m) · 9.81 (m·s⁻¹).

**Statistical Analysis**

All data were collated onto a personal computer (Macintosh, MacBook Pro) and analyzed using SPSS (v22.0, IBM). Data were assumed parametric following Shapiro–Wilk and Levene’s tests. To avoid a Type I error in the comparisons between groups’ VO₂ and C measures, a repeated measures ANOVA with a between subject variance and C were then normalized to TBM (VO₂TBM and C₇TBM, respectively) and FFM (VO₂FFM and CFFM, respectively). All VO₂ and C values were presented against absolute walking and running speeds, and against dimensionless Fr, given as: velocity² (m·s⁻²) ÷ leg length (m) · 9.81 (m·s⁻¹).

**RESULTS**

**Participant Anthropometrics**

There was no difference in age between groups (P = 0.487, Table 1). Achondroplasia were 23% smaller in stature (P < 0.001), had a 38% shorter leg (P < 0.001), 19% less TBM (P < 0.001), 26% less FFM (P < 0.001) but a 25% greater body mass index (BMI, P < 0.001) than controls (Table 1).

**Self-Selected Walking**

Achondroplasia were 23% slower than controls at SSW [Achondroplasia, 1.02 (0.13) m·s⁻¹; control 1.33 (0.14) m·s⁻¹, P < 0.001].

**Incremental Exercise**

During VO₂ assessment, both groups completed all walking speeds. However, only 50% of the Achondroplasia group managed to obtain steady state running at 2.50 m·s⁻¹ and only 20% maintained steady state running at 2.78 m·s⁻¹. Therefore, VO₂ and C values collected at all walking speeds and at
running speeds $1.67 - 2.22 \text{ m/s}^{-1}$ were inferentially analyzed (Figures 1, 2).

**Oxygen Consumption**

$\dot{V}O_2_{TBM}$ were on average 29% greater in Achondroplasia at all absolute walking speeds apart from SSW where no difference was observed (Figure 1A). Similarly, Achondroplasia had an average 35% greater $\dot{V}O_2_{FFM}$ at all absolute walking speeds compared to the control group, with no difference being found between groups at SSW (Figure 1B). There was no difference in $\dot{V}O_2_{TBM}$ between groups at running speed $1.67 \text{ m/s}^{-1}$, but Achondroplasia had a 14 and 12% higher $\dot{V}O_2_{TBM}$ than controls at running speeds 1.94 and 2.22 m·s⁻¹ (Figure 1A). A higher $\dot{V}O_2_{FFM}$ was observed in Achondroplasia for all running speeds (Figure 1B).

**Metabolic Cost**

On average, Achondroplasia had a 29% higher walking $C_{TBM}$ and 33% higher walking $C_{FFM}$ when compared to controls (Figure 1C). Running $C_{TBM}$ were the same between groups at 1.67 and 2.22 m·s⁻¹ ($P > 0.05$) whereas Achondroplasia had a higher $C_{TBM}$ than controls at 1.94 m·s⁻¹ ($P < 0.05$, Figure 1D). Achondroplasic running $C_{FFM}$ was, on average, 18% higher at all running speeds compared to controls ($P < 0.05$, Figure 1D).

**Comparison of Froude’s Number (Fr)**

Strong positive correlations existed for the Achondroplasic group between Fr and $\dot{V}O_2_{TBM}$ when walking ($r = 0.998$, $P = 0.001$) and running ($r = 0.994$, $P = 0.070$), and also for controls when walking ($r = 0.997$, $P < 0.001$) and running ($r = 0.985$, $P < 0.001$, Figure 2A and Table 2). There were also strong positive correlations for the Achondroplasic group between Fr and $\dot{V}O_2_{FFM}$ when walking ($r = 0.999$, $P < 0.001$) and running ($r = 0.993$, $P = 0.036$) and for the control group when walking ($r = 0.997$, $P < 0.001$) and running ($r = 0.984$, $P < 0.001$, Figure 2B and Table 2).

**DISCUSSION**

The main aim of this study was to attain $\dot{V}O_2$ and $C$ profiles during incremental walking and running in adult males with Achondroplasia and compare the results to controls. Further to this, we aimed to scale $\dot{V}O_2$ and $C$ to body mass and leg length in both groups. Our hypotheses were partially met in that: (1) Achondroplasia had a greater $\dot{V}O_2_{TBM}$ and $\dot{V}O_2_{FFM}$ during walking and running compared to controls and had a higher $C_{TBM}$ and $C_{FFM}$ (i.e., a greater $\dot{V}O_2$ for a given distance) at all walking speeds compared to controls; and, (2) leg length can
explain some of the difference in $\dot{V}O_2^{TBM}$ and $\dot{V}O_2^{FFM}$ between groups but not for $C_{TBM}$ and $C_{FFM}$.

### Oxygen Consumption

Achondroplasic $\dot{V}O_2^{TBM}$ and $\dot{V}O_2^{FFM}$ was higher at every walking and running speed compared to controls other than SSW which were the same between groups. This is similar to previous reports, where shorter statured groups have higher $\dot{V}O_2^{TBM}$ than taller counterparts during locomotion (Rowland and Green, 1988; Minetti et al., 1994; Schepens et al., 2004; Ludlow and Weyand, 2015). The higher Achondroplasic $\dot{V}O_2^{TBM}$ and $\dot{V}O_2^{FFM}$ at set speeds is most likely due to a higher stride frequency of the Achondroplasic group, which is a result of their shorter legs, as Minetti et al. (1994) partially confirmed that higher stride frequency was the cause of higher $\dot{V}O_2^{TBM}$ in their African Pygmy group. While stride frequency was not measured in the present study, the data presented by Minetti et al. (1994) would infer some similarities between groups. The Pygmies included in Minetti et al. (1994) were 16 cm taller than the presented Achondroplasic group while the control groups were 1 cm different. With Pygmies being smaller and having a higher stride frequency compared to controls, it is likely that the even smaller Achondroplasic group had a similar, if not higher, stride frequency to the Pygmies.

While this study does not directly measure factors that explain the higher $\dot{V}O_2$ of the Achondroplasic group for a given speed of locomotion, there are a number of mechanisms that could explain these data. Firstly, the probable higher stride frequency of the Achondroplasic group would lead to a greater amount of internal mechanical work being done compared to controls, as observed in other shorter statured groups (Ferretti et al., 1991; Minetti et al., 1994; DeJaeger et al., 2001; Minetti et al., 2002; Schepens et al., 2004; Weyand et al., 2010). A higher internal work not only requires energy to complete the work but would also elicit a greater rate of muscular contraction. Assuming the fiber type distribution is the same between groups, the rate of muscular contraction is likely to alter the force-velocity relationship of the Achondroplasic muscle (Fletcher and MacIntosh, 2017). In this scenario, the Achondroplasic muscle would be producing less force due to the quicker movement of the limbs. Muscle activation could therefore be higher in the Achondroplasic group to recruit the fibers required to maintain locomotion forces, therefore leading to a higher oxygen demand (Mian et al., 2006). However, internal work, force production (relative to gait requirements) and muscle activation have not been measured in Achondroplasic individuals during gait and therefore more work is required in this area to help confirm these theories. To try an account for the assumed higher Achondroplasic stride frequency,
TABLE 2 | $R^2$ values for Froude and scaled $\dot{V}O_2$ relationships in Achondroplasic individuals and controls during walking and running.

<table>
<thead>
<tr>
<th></th>
<th>Achondroplasia</th>
<th>Control</th>
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<tbody>
<tr>
<td></td>
<td>Walking</td>
<td>Running</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>0.997</td>
<td>0.988</td>
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<tr>
<td>$\dot{V}O_2$ (ml·kgFFM$^{-1}$·min$^{-1}$)</td>
<td>0.997</td>
<td>0.987</td>
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All correlations significant at $P < 0.001$.

and therefore eliminate some of the above, we normalized horizontal speed using leg length in the form of Fr.

Comparisons of Froude’s Number (Fr)

In the current study, Fr could explain the variability of the groups’ $\dot{V}O_2$ and $\dot{V}O_2$ as little as 98.7% (Figures 2A, B and Table 2). This matches much of the literature where Fr has been used to compare the relationship between leg length and $\dot{V}O_2$ during locomotion. Ferretti et al. (1991) and Minetti et al. (1994) showed African Pygmy’s $\dot{V}O_2$ and Fr trends are similar to controls, with Minetti et al. (2002) also observing similar findings in patients with GHD. The similarity of the $\dot{V}O_2$ and Fr trends between groups suggests that the difference in groups’ $\dot{V}O_2$ and $\dot{V}O_2$ in the shorter Achondroplasic leg. The similarity in the slopes are not surprising given that $\dot{V}O_2$ correlates well with TBM and FFM (Goran et al., 2000; Weibel and Hoppeler, 2005). It is important to note here though, that although the relationships appear similar between the groups and between mass scalers (Table 2), each mass scaler should not be used to estimate another. For example, in both groups a Fr of 0.30 elicits a $\dot{V}O_2$ in excess of 15 ml·kg$^{-1}$·min$^{-1}$, whereas the same Fr elicits a $\dot{V}O_2$ in excess of 20 ml·kg$^{-1}$·min$^{-1}$. While the inclusion of Fr accounted for the difference in Fr by providing similar slopes for both $\dot{V}O_2$ and $\dot{V}O_2$ in the shorter Achondroplasic leg, Fr did not explain the difference in C between groups.

Metabolic Cost

While $\dot{V}O_2$ is a useful parameter, it only describes the rate at which O$_2$ is used, not the usage per unit distance. In contrast, C provides a measure of energy cost, assuming an energy equivalent of 20.9 J per 1 ml O$_2$ (Figures 1B, D) and describes the energetic cost per unit mass and distance. Despite accounting for mass and distance traveled, the Achondroplasic group exhibited a higher C during walking and running than controls. In addition, C at SSW occurred near the local minima of the trend lines for both groups but remained different between groups. The factors that may account for the difference in C between groups are again not investigated directly in this paper, but the available literature does allow insight into potential reasons to why these differences exist.

The torso size of Achondroplasic people and controls is the same when measured as sitting height (Owen et al., 1990). With the Achondroplasic legs being shorter than controls, one would assume that Achondroplasic people have a larger torso to TBM ratio than controls. Where additional mass is added to the torso of individuals exercising on a treadmill, a higher C is observed compared to individuals without additional mass (Griffin et al., 2003; Browning et al., 2006, 2007; Beekley et al., 2007; Peyrot et al., 2009; McCormick, 2014); the converse is observed when body weight is reduced through assisted treadmill running (Grabowski and Kram, 2008). While the present Achondroplasic participants were lighter than the control group, the additional torso mass of the Achondroplasic group would have implications for the contractile properties of their smaller lower leg muscles during stance. Firstly, relative to lower leg muscle mass, a greater vertical ground reaction force (vGRF) would be observed in the Achondroplasic group. The smaller muscle fibers of the Achondroplasic lower limbs (Sims et al., 2017) would therefore do more external work during the braking phase of stance to compensate for the relatively larger vGRF, eliciting a higher C (Pontzer, 2005; Grabowski and Kram, 2008). Furthermore, we recently showed that Achondroplasic adults have a greater co-activation of their antagonists during knee extension (Sims et al., 2017). Although this was during maximal voluntary contraction, it is possible that during the propulsion phase of gait, there is greater co-activation of the hamstrings. Previously, a higher co-activation during gait contributes to negative work which is partly associated with a higher C (Mian et al., 2006). These theories though, require further work to help explain the difference in C between Achondroplasic individuals and controls.

Implications and Future Work

In the present study, the difference in C between groups could possibly be explained by biomechanical differences that have previously been shown to alter or determine C, such as mass distribution and inertial properties (Griffin et al., 2003; Browning et al., 2006, 2007; Beekley et al., 2007; Peyrot et al., 2009; McCormick, 2014), tendon compliance (Arampatzis et al., 2006; Fletcher et al., 2010; Albracht and Arampatzis, 2013) and internal and external work (Ferretti et al., 1991; Minetti et al., 1994, 2002); further work is required in Achondroplasic populations to determine whether these factors play a role in explaining the present results. The current data does show that leg length can explain some of the variance in $\dot{V}O_2$ and $\dot{V}O_2$ between the presented groups, i.e., longer legs equate to a lower walking and running $\dot{V}O_2$ and in turn suggests a lower walking and running C. Therefore, the lengthening of the Achondroplasic leg would possibly lower C and make some ambulatory based activities of daily living less energetically demanding. Although a viable option, leg lengthening surgery for an Achondroplasic individual is, however, a highly invasive long-term surgical procedure involving prolonged recovery and imposed sedentary behavior (Park et al., 2015). Were other interventions explored to help improve Achondroplasic C during activities of daily living, it is probably that such tasks would be completed more often. It is probable thereafter that their physical activity levels would be improved, as has been observed in an obese population (Weinsier et al., 2000), which is then likely to lower the risk of health complications (Wilmot et al., 2012). Further work should therefore be directed toward identifying exercise interventions that are associated with lowering C.
in Achondroplasic populations, such as those identified in control populations described above, and then be appropriately addressed in Achondroplasic populations.

CONCLUSION

This study aimed to observe the relationship between VO$_2$ and incremental walking and running speeds in adult males with Achondroplasic. To the author's knowledge, this is the first study to scale and compare VO$_2$ and C during walking and running between Achondroplasic males and controls. The main findings are that (1) Achondroplasic VO$_2$TBM and VO$_2$FFM are higher at all walking and running speeds compared to controls; (2) VO$_2$TBM and VO$_2$FFM is not different between groups at SSW; (3) C$_{TBM}$ and C$_{FFM}$ are higher in Achondroplasia than controls at all walking and running speeds, and 4) the inclusion of leg length as a scaler helps explain VO$_2$ differences but not C differences between groups. The higher Achondroplasic VO$_2$TBM and VO$_2$FFM is most likely due to a higher stride frequency, while their higher C$_{TBM}$ and C$_{FFM}$ is likely due to anthropometrical differences compared to controls.

REFERENCES


AUTHOR CONTRIBUTIONS

DS: Lead author, contributed to the inception of the study, participant recruitment and testing, data analysis and interpretation, writing of the main body of work and approval of the article. GO-P and AB: Contributed to the inception of the study, data analysis and interpretation, and revisions of the main body of work and approval of the article. CP: Contributed to data analysis and interpretation and revisions of the main body of work and approval of the article. CM: Contributed to the inception of the study, participant recruitment and testing, data analysis and interpretation, and writing of the main body of work and approval of the article.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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