EFFECT OF DEER ANTLER VELVET ON AEROBIC, ANAEROBIC AND STRENGTH PERFORMANCE

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Abstract. Deer antler velvet (DAV) supplementation purportedly increases athletic performance; however, little data support this claim. The primary aim of our study is to examine DAV and exercise performance. We randomized 32 men (18–35 y) participating exclusively in resistance training (≥4 y) to 10-weeks of randomly assigned, double blind, DAV (1350 mg, 2×/day) or placebo treatments. Primary outcomes included maximal aerobic capacity (VO_2max), maximal strength (1RM; bench press and squat) and anaerobic cycling power. Secondary outcomes included comprehensive blood profiles and body composition. We used general linear models to determine changes following treatment.

Eighteen participants (n = 9) completed the study with DAV participants showing significant improvements in VO_2max (4.30 ±0.45 to 4.72 ±0.60 L/min, $P < 0.04$). The placebo and DAV groups increased bench press and squat 1RM (both, $P < 0.04$); yet, when expressed relative to body mass, only the DAV group showed significant bench press (4%) and squat (10%; both, $P \leq 0.02$). Neither group improved cycling performance or showed adverse changes in blood chemistries. We did observe a significant reduction in LDL-C (12%) accompanying DAV supplementation and both groups significantly reduced percent body fat ($P < 0.05$). Our results suggest that DAV may have ergogenic effects in men participating solely in resistance training.

Key words: Antler velvet, strength training, performance, aerobic power, anaerobic power

Introduction

Deer antler "velvet" (DAV) is a soft skin, with a texture similar to velvet, and is considered a mammalian organ owing to its annual regeneration (Goss 1984; Suttie et al. 1989). Anecdotal evidence and some research reports suggest that antler velvet promotes health, alleviates anemia, reduces arthritis, increases growth rates in children,

and improves some aspects of athletic performance (Allen et al. 2002; Sleivert et al. 2003; Wang et al. 1988a; Wang et al. 1988b; Zhou et al. 1999). Obtained during the growing process, DAV is produced systemically by insulin-like and transforming growth factors is subsequently used for various medicinal purposes (Francis and Suttie 1998; Li 2013; Li and Suttie 2000; Sadighi et al. 1994; Suttie et al. 1989; Suttie and Haines 2004). Overall, DAV derived from the antler tip and upper sections are lower in ash, calcium, and phosphorous than the mid and base sections. In contrast, the tip has greater lipid, nitrogen reflecting protein content, and selenium than all other sections. Finally, the tip and upper sections of the velvet antler are rich sources of iron. Several studies also reveal a variety of growth factors in antler velvet including insulin like growth factor, transforming growth factor beta, fibroblast growth factor, bone morphogenetic protein, vascular endothelial growth factor, and nerve growth factors (Francis and Suttie 1998; Lai et al. 2007; Sadighi et al. 1994). These constituents may be important factors for improving exercise performance and body composition.

Few reports exist regarding the efficacy and dosage required to promote ergogenesis in sport. Syrotuik et al. (2005) observed no significant improvements in maximal aerobic capacity, maximal strength, or rowing time following 560 mg/d of DAV (Syrotuik et al. 2005). In contrast, Sleivert et al. (2003) demonstrated significant improvements in isokinetic knee extensor strength following 10 weeks of strength training using higher doses (1,500 mg) compared to a placebo group (Sleivert et al. 2003). According to a review by Suttie and Haines, it has been suggested that dosing protocols <1,000 mg/d typically lack efficacy for health or sports performance, while dosages from 1,000–1,500 mg/d exert small, yet statistically meaningful performance effects, and dosages >2,000 mg elicit clear, statistically significant effects following ≥ 8-weeks of supplementation (Suttie and Haines 2004). Regardless, the efficacy of DAV remains unclear. The primary aim of our current study is to examine the efficacy of DAV on exercise performance, body composition and blood chemistries as a means of assessing supplement safety. We hypothesized that DAV will improve exercise capacity, as determined by assessments of aerobic, anaerobic and strength performance.

Methods

We recruited participants for this study with a minimum of four years of resistance training activity and not currently involved in aerobic training. Eligible participants began the study by signing an informed consent outlining the study procedures approved by the East Tennessee State University Institutional Review Board and performed in accordance with the Declaration of Helsinki. We initially screened eligible participants with an ECG monitored cardiac stress test and, upon clearance to participate; we randomized participants in a double-blind manner to treatment or placebo conditions. Treatment consisted of ingesting an encapsulated DAV supplement (1,350 mg, 2×/d) or matched placebo capsules of similar size and color for 10 weeks. We instructed participants to ingest one capsule at breakfast and one immediately before bed. We also instructed participants to maintain their current training regimen, while random supplement checks performed approximately every two weeks to verify their adherence to the supplement routine. We have presented all participant demographics, anthropometry, and performance indices in Table 1 and hematology indices in Table 2.

Table 1. Demographic and fitness characteristics of study participants

		All (N = 18)		Placebo (n = 9)		DAV (n = 9)	
		Mean	SD	Mean	SD	Mean	SD
Anthropometry							
Weight (kg)	Baseline	95.45	14.07	95.84	17.72	95.06	10.31
	Follow-up	94.94	13.69	94.92	17.12	94.97	10.26
BMI (kg/m^2)	Baseline	29.83	3.58	30.73	4.30	28.93	2.63
	Follow-up	29.73	3.25	30.47	3.84	28.99	2.55
Body Fat (%)	Baseline	20.52	5.55	21.51	6.13	19.53	5.06
	Follow-up	19.16*	5.08	20.08	5.79*	18.23	4.41
Fat Mass (kg)	Baseline	20.01	7.13	21.41	8.64	18.60	5.38
	Follow-up	18.57*	6.53	19.74	7.97*	17.40	4.89
Fat Free Mass (kg)	Baseline	75.49	9.47	74.54	9.87	76.45	9.54
	Follow-up	76.38	9.36	75.19	10.16	77.57	8.93
Fitness Indices							
VO_2max (L/min)	Baseline	4.21	0.50	4.10	0.56	4.31	0.45
	Follow-up	4.41	0.66	4.09	0.58	4.72*,**	0.60
VO_2max (ml/kg/min)	Baseline	44.84	7.52	43.16	6.90	46.53	8.14
	Follow-up	46.90	7.92	44.00	5.93	49.8*,**	8.91
Maximum HR (b/min)	Baseline	189.44	8.97	186.56	8.56	192.33	8.89
	Follow-up	188.44	6.62	188.67	7.28	188.22	6.32
Peak Power (W)	Baseline	733.78	167.92	690.67	196.35	776.89	131.08
	Follow-up	725.17	179.13	677.56	193.19	772.78	160.50
Time to Peak Power (sec)	Baseline	7.39	2.37	6.83	2.10	7.94	2.62
	Follow-up	6.94	1.52	6.84	1.68	7.04	1.44
Average Power (W)	Baseline	581.00	120.75	542.56	131.94	619.44	101.25
	Follow-up	560.83	129.71	515.44	144.38	606.22	101.36
Bench Press 1MR (kg)	Baseline	121.36	23.11	123.00	23.92	119.71	23.59
	Follow-up	126.38	25.84	128.01*	27.47	124.74*	25.66
Squat 1MR (kg)	Baseline	154.61	35.32	150.20	28.14	159.02	42.60
	Follow-up	165.82	37.66	156.24	30.34	175.40*	43.45
Total Lifting Volume (kg)		313,955	97,170	283,647	64,065	348,051	119,922

All data are express as mean ± SD. Body composition measures are determined by DXA.
* Represents a significant within group change from baseline ($P < 0.05$).
** Represents a significantly difference versus Placebo.

Testing Procedures

Baseline and follow-up testing included a series of tests to determine maximal cardiorespiratory fitness (VO_2max), anaerobic power output (PO, W), and upper and lower muscular strength. Participants performed all performance tests after abstaining from strenuous exercise for 12 hours and the consumption of a large meal four hours before testing. We performed all blood testing procedures under fasting conditions (8–10 hours) to assess blood cholesterol, blood glucose, liver and kidney function enzymes (Table 2).

Table 2. Hematology of study participants

		All (N = 18)		Placebo (n = 9)		DAV (n = 9)	
		Mean	SD	Mean	SD	Mean	SD
Triglycerides (mg/dL)	Baseline	104.61	52.86	115.78	62.21	93.44	42.27
	Follow-up	115.28	48.89	99.33	35.29	131.22	57.12
Total Cholesterol (mg/dL)	Baseline	177.28	46.64	175.11	56.30	179.44	37.99
	Follow-up	171.00	50.23	170.78	59.61	171.22	42.52
HDL-C (mg/dL)	Baseline	45.22	9.53	42.89	11.54	47.56	6.91
	Follow-up	43.39	5.95	41.22	6.34	45.56	4.95
LDL-C (mg/dL)	Baseline	111.11	38.17	109.00	43.32	113.22	34.77
	Follow-up	104.50	43.95	109.56	51.42	99.44*,**	37.44
Total-C/HDL-C Ratio	Baseline	4.01	1.10	4.17	1.16	3.85	1.08
	Follow-up	3.95	1.07	4.11	1.10	3.80	1.08
Glucose (mg/dL)	Baseline	94.00	5.37	95.67	4.74	92.33	5.70
	Follow-up	92.44	7.12	93.22	3.93	91.67	9.54
BUN [1]	Baseline	16.11	4.19	16.22	4.12	16.00	4.50
	Follow-up	16.06	3.30	16.22	2.44	15.89	4.14
Creatinine [1]	Baseline	1.22	0.15	1.23	0.17	1.21	0.14
	Follow-up	1.18	0.16	1.14	0.22	1.21	0.08
Albumin [2]	Baseline	4.44	0.21	4.46	0.19	4.42	0.25
	Follow-up	4.36	0.21	4.39	0.18	4.33	0.24
Globulin	Baseline	2.65	0.49	2.51	0.38	2.79	0.58
	Follow-up	2.63	0.49	2.59	0.45	2.67	0.55
Bilirubin [2]	Baseline	0.78	0.40	0.78	0.52	0.78	0.28
	Follow-up	0.85	0.59	0.98	0.77	0.72	0.35
ALP [2]	Baseline	66.28	13.95	60.00	9.54	72.56	15.29
	Follow-up	68.33	17.16	60.56	8.62	76.11	20.38
AST [2]	Baseline	23.33	7.66	19.56	3.24	27.11	9.06
	Follow-up	24.11	7.10	21.89	6.11	26.33	7.65
ALT [2]	Baseline	28.33	13.38	25.33	11.54	31.33	15.06
	Follow-up	26.78	13.85	25.67	14.32	27.89	14.15
Hematocrit	Baseline	44.78	3.44	46.27	3.53	43.29	2.76
	Follow-up	44.60	3.05	44.93	2.67	44.23	3.58
Hemoglobin	Baseline	15.27	1.27	15.71	1.31	14.82	1.13
	Follow-up	15.25	1.14	15.48*	1.03	15.02*	1.25

Indicators of (1) Kidney and (2) liver function: BUN, blood urea nitrogen; ALP, Alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine transaminase. To convert to SIU multiple respective variables by: Triglycerides (0.0113), total cholesterol, HDL-C, and LDL-C (all, 0.0259), glucose (0.055), BUN (0.357), Creatinine (88.4), Bilirubin (17.104), ALP, AST, and ALT (all, 0.0167), Hematocrit (0.01), Hemoglobin (10).

* Represents a significant within group change from baseline ($P < 0.05$).

** Represents a significantly difference versus Placebo.

Maximal Oxygen Uptake

To measure VO_2max we performed a maximal ECG monitored treadmill exercise test. Oxygen consumption was assessed via open circuit spirometry (SensorMedics 2900, Yorba Linda, CA). Maximal aerobic capacity was defined as a plateau in VO_2 (<2–3 ml) with increasing workloads, a respiratory exchange ratio >1.1, and heart rate within 10 beats of each participant's age predicted maximum heart rate (220-age). If a participant did not meet the above criteria, they returned to the laboratory to repeat the test.

Anaerobic Power Testing

We assessed anaerobic power on a Computrainer (Seattle, WA), whereby participants were asked to cover a standardized distance (320 m) as quickly as possible. Testing consisted of three cycling trials interspersed with light tension recovery until heart rate dropped below 100 bpm. The trial eliciting the best performance was used to record each individual's maximum and average power output. Forty-eight hours after their first test, participants repeated the test to verify their power output. No significant differences were observed between the repeated power trials; therefore, the data reported herein are the mean for both trials.

Prior to testing, the Computrainer was calibrated at 1.47 kg of resistance for each participant's trials with the cycle rear tire inflated to the maximal pressure allowed to minimize friction related power variance. After the calibration procedure, we instructed participants on how to perform the test before beginning the protocol. Participants initiated each test with a light intensity warm-up lasting five minutes. During the test, the participant remained seated on the cycle and began cycling in a 52/23-gear combination at 60 rpm for one minute. Participants continued to cycle for another minute in order to change gearing before subsequently cycling at maximal effort.

Strength Assessment

Upper and lower body maximal strength was determined using a one repetition maximum (1MR) protocol for both the bench press and leg squat. All lifts were performed with a spotter. The initial weight lifted was estimated based on the previous lifting experience of the participants and what they normally used for each respective movement. A successful bench press attempt involved fully lowering the weight to the chest and then extending fully upward until the arms were straight without assistance from the spotter. For the maximal squat attempt the participant was instructed to squat until the leg reached a 90° degree angle. Between lifts, participants recovered for 1–3 minutes of rest to ensure recovery as they neared their maximal effort. We considered a successful 1MR as the weight lifted immediately prior to a failed attempt.

Anthropometry

Height, body mass and body mass index (BMI) were recorded using standardized methodology and body composition was determined using DXA (Lunar System, GE Corporation, Wauwatosa, WI) in order to examine percent body fat and fat free mass.

Statistical Analyses

We used general linear models to examine changes in all parameters from baseline and within-group statistical significance was established by examining 95% confidence interval (95% CI) and regression analyses were performed in order to examine relationships between changes in outcomes as appropriate. Between group differences were assed using least squared differences. Owing to a high dropout rate (described below), we covaried all of our analyses using respective baseline analysis measures reasoning that the number of dropouts would offset the randomness of our initial treatment assignments. In addition, we boot strapped our analyses using 1,000 imputations in order to better examine the surrounding confidence intervals of our analyses. Lastly, we performed Pearson correlations to examine respective changes candidate variables to denote significant relationships regarding potential mechanisms of action. Effect sizes were calculated and presented as partial eta

squared. We have reported all data as means ± SD or 95% confidence intervals when appropriate. Statistical significance is set at $P \leq 0.05$.

Results

We initially recruited 32 individuals into our study. However, 14 participants were excluded due to: Failure to complete all testing requirements (n = 7), lack of adherence to the study protocol (n = 3), study dropout due to scholastic time commitments (n = 2), an injury obtained outside the study (n = 1), desire to change their work routine (n = 1). We have presented participant demographics, anthropometry, and performance indices in Table 1 and hematology indices in Table 2. Changes from baseline and individual response values for VO_2max and 1MR strength measures are presented in Figure 1.

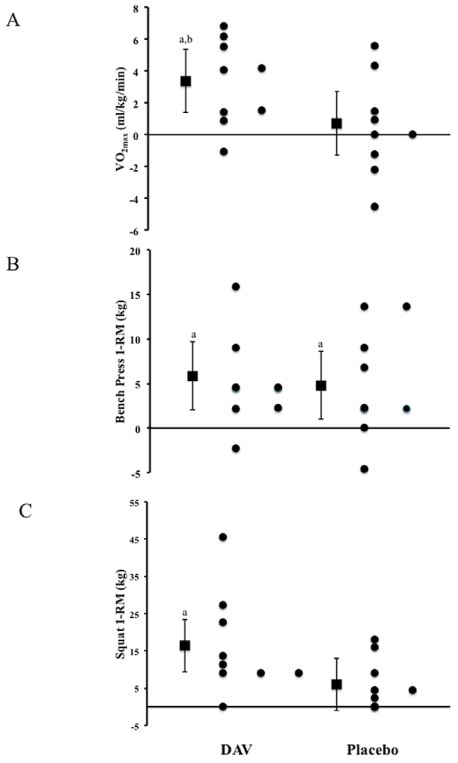

Figure 1. Data represent mean change from baseline (±95% CI) for relative VO2max and one repetition maximum bench press and squat performance. Black circles represent individual participant responses. Statistical significance is represented as (a) significant within group change and (b) significantly different from placebo

Overall, we observed no significant between group differences at baseline for age (27 ±6 y), height (1.79 ±0.1 m), weight (95.5 ±14 kg), BMI (29.8 ±3.5 kg/m^2) or dietary intake measures including total energy intake (2491 ±803 kcal), protein (128 ±61 g) and carbohydrate (314 ±96 g). Analysis of participant training records showed

that each group trained ~4 d/wk. At follow-up, we found that the DAV group exhibited a greater total lifting volume (348,051 ±119,922 kg) during the intervention compared to the placebo group (283,647 ±64,065 kg, $P < 0.05$). We did not observe any significant changes in body mass at follow-up; however, both the placebo (–1.30, 95% CI, –2.75, –0.16) and DAV group (–1.43%, 95% CI, –2.52, –0.49) demonstrated significant reductions in percent fat mass, as well as for percent body fat ($P < 0.05$). No significant between group differences were noted for either parameter, nor for changes in fat free mass (Table 1).

Maximal Cardiorespiratory Capacity. We observed no significant between group differences at baseline for absolute or relative VO_2max. At follow-up, the placebo group showed no significant difference in VO_2max (–0.012 L/Min, 95% CI, –0.20, 0.19), while the DAV group exhibited a significant improvement (0.41 L/min, 95% CI, 0.26, 0.60) that was also different versus placebo ($P < 0.05$). A similar pattern of improvement was noted when VO_2max was expressed in relative terms (Figure 1a), while we did not observe any significant difference in maximal heart rate obtained during exercise testing. When expressed as percent improvement, the DAV group exhibited a 9.6% (95% CI, 6.1, 13.5) and 7.4% (95% CI, 3.5, 11.8) for absolute and relative VO_2max, respectively. Respective responses for the placebo group were –0.2% (95% CI, –5.2, 4.6) and 2.1% (95% CI, –0.3, 7.0).

Anaerobic Power and Muscular Strength. We observed no significant improvements for time to peak power, peak power, or average power for either treatment group. For the bench press, both groups exhibited a significant improvement in 1RM bench press (Figure 1b). Only the DAV group improved squat performance (Figure 1c). While no between group differences were noted, the DAV group exhibited a numerical trend toward greater improvement (16.40 kg, 95% CI, 7.9, 25.5) versus the placebo group (6.02 kg, 95% CI, 1.7, 11.2; $P = 0.07$) although statistical significance was not reached. When we analyzed our data in relative terms (wt/kg), we found the same patterns for improvement (data not shown).

Hematology. Overall, we observed no significant changes in hematological variables with the exception of blood lipids and hematocrit (Table 2). For total cholesterol, we observed a significant reduction within the DAV group (–8 mg/dL, 95% CI, –14.74, –1.30). No significant reduction was noted for the placebo group (–4 mg/dL, 95% CI, –8.17, 16.38), nor were any between group differences otherwise noted. The observed reduction in total cholesterol was associated with a significant reduction in low density lipoprotein cholesterol (LDL-C) within the DAV group (–13.96 mg/dL, 95% CI, –24.37, –3.54), with only minor changes observed within the placebo group (0.74 mg/dL, 95% CI, –6.66, 9.07). No significant changes in triglycerides are noted (Table 2). For hematocrit, the DAV group exhibited a significant improvement (1.28%, 95% CI, 0.60, 2.64), whereas the placebo group showed a significant reduction (–1.33%, 95% CI, –2.040, –0.51), significant differences observed between the treatment groups ($P = 0.003$). We also observed significant correlations between changes in VO_2max and hematocrit ($r = 0.68$, $\beta = 0.11$, $P = 0.003$) versus changes in body mass ($r = –0.075$, $\beta = 0.011$, $P = 0.70$) and total lifting volume ($r = 0.46$, $\beta = 0.008$, $P = 0.72$).

Discussion

The primary aim of our study was to examine in the effect of DAV supplementation on parameters of aerobic, anaerobic and muscular strength performance. Secondary to this purpose we also performed a body composition and blood chemistry analyses in order to gain insight into potential safety issues associated with DAV supplementation. Overall, we found that DAV supplementation increased aerobic capacity when expressed in absolute (9.6%) and relative terms (7.4%). While we did not observe significant improvements for various indices

of anaerobic power, both groups did exhibit a significant improvement in bench press and squat performance. However, no differences between the placebo and DAV group existed despite a numerical trend for between group differences for the squat (P = 0.07). Of particular importance to our study is the observation that 10 weeks of DAV supplementation was not associated with negative alterations in blood chemistries associated with hepatorenal function. Additionally, DAV supplementation was associated with lowered plasma LDL-C levels. Based on these results, we accept the hypothesis that DAV improves aerobic capacity; reject the hypothesis that DAV will improve anaerobic power and view the hypothesis that DAV will increase strength performance as equivocal based on the inconsistency of responses for bench press and squat performances observed in the current study.

The most intriguing finding of our study is the observed improvement in the DAV group VO_2max. However, the mechanisms responsible for these changes remain unclear. A simplistic reason for these changes may simply be the increase in total training volume observed in the DAV group. While extensively reviewed elsewhere, heavy strength training can improve running performance in non-endurance athletes and may be related to delayed activation of less efficient type II fibers, improved neuromuscular efficiency, conversion of fast-twitch type IIX fibers into more fatigue-resistant type IIA fibers, or improved musculotendinous stiffness (Aagaard and Andersen 2010; Ronnestad and Mujika 2013). Collectively, these observations explain the current findings as the participants in this study participated exclusively in resistance training. Thus, it is reasonable to anticipate a modest increase in maximal cardiorespiratory capacity in conjunction with the greater levels of exercise energy expenditure associated with 10 weeks of DAV supplementation.

A second possible reason for the increase in VO_2max may be related to changes in hemoglobin (1.4%, non-significant) and hematocrit concentrations (2.7%) as the observed change in VO_2max was correlated with a small, yet significant increase in hematocrit (r = 0.67, mean 1.28%, 95% CI, 0.60, 2.64) accompanying DAV supplementation. However, given that our calculations were performed to Dill and Costill, it is hard to reconcile the changes in VO_2max to blood parameters alone and the observed improvements in VO_2max may be due to the combined effects between increased training volume (r = 0.46, β = 0.008) and hematocrit (r = 0.68, β = 0.11) (Costill et al. 1974; Costill and Fink 1974).

Our results for the anaerobic and strength performance are difficult to interpret. While it is clear that DAV has no ergogenic effect on anaerobic performance as assessed by a Wingate-like test, changes in upper and lower body strength are less certain. These differences could be accounted for by examining the dose of DAV associated with various studies as well as the respective training status of the study participants.

Syrotuik et al. (2005) showed no significant improvements in leg press strength, bench press strength or circulating concentrations of testosterone or insulin-like growth factor following a lose dose (560 mg/d) DAV supplementation routine for 10 weeks while also participating in strength and rowing training.(Syrotuik et al. 2005) In contrast, Sleivert et al. (2003) used a higher dose (1,500 mg/d) of DAV per day and demonstrated significant improvements in knee extension strength and endurance following after 10 weeks of supplementation(Sleivert et al. 2003). Thus, dose appears to be an issue as both Sleivert and our study used DAV doses ~2.7 and 4.8 fold higher to Syrotuik. Finally, training status cannot be discounted. While Sleivert et al. (2003) examined “active males,” we specifically focused on chronically resistance trained participants not currently participating in aerobic conditioning. Thus, chronic resistance training may not elicit as great a supplement effect in chronically resistance-trained men owing to a plateauing of neurological and potential for musculoskeletal strength gains compared lesser-trained individuals (Sale 1988).

Both groups in our current study showed a similar change in percent body fat; therefore, DAV supplementation does not appear to offer any direct or indirect (i.e., via increased training volume) affect on anthropometry. Supplementation with DAV also does not appear to adversely affect indices associated with hepatorenal function. Curiously, however, DAV supplementation did affect LDL-C (–12%), which subsequently altered the LDL/HDL cholesterol ratio in the DAV (3.8%) versus the placebo conditions (<1%). Given that blood parameters were a tertiary are of research interest, we are unable to offer a direct mechanism of action responsible for these change in LDL-C. However, IGF concentrations have been shown previously to be inversely associated with LD-C in fasted individuals (Savendahl and Underwood 1999). While beyond the scope of this article, IGF levels in DAV vary with the antler growth cycle, and therefore, may be an area of future interest with regard to potential alterations in lipid metabolism (Francis and Suttie 1998; Suttie et al. 1989).

A strength of our study is that we have extended the small body of research examining DAV using a clinically focused trial. While our study shows a positive effect on aerobic capacity accompanying DAV supplementation, some caution is advised to readers given the high number of dropouts associated with our trial. Though we considered using various imputation techniques to overcome this obstacle, the breadth of imputation compared to the number of dropouts within the study outweighs the utility of imputation. Thus, we opted to covary our analyses using the respective baseline variable for each assessed parameter. In addition, we used bootstrapping techniques to improve the confidence intervals surrounding our outcomes. Finally, we re-examined all of our initial analyses by also co-varying for total lifting volume a posteriori, which only served to inflate the responses of the DAV group further. We believe our current approach is conservative means for addressing our study findings. We are further limited by our inability to expound on hormonal responses to DAV supplementation, nor generalize our findings beyond those involved in chronic resistance training only. This latter limitation however, is also a strength to our study, as we explored the relationship between DAV supplementation, exercise performance and blood safety factors associated with hepatorenal function in those individuals most likely to use DAV as a nutrition supplement; specifically, those heavily involved in strength training. Additional strengths of our study include using DAV in higher doses than previously reported, which not only improved aerobic capacity, but also appear to modulate LDL-C concentrations, subsequently the LDL/HDL cholesterol ratio. Taken as a whole, our findings suggest that DAV shows potential ergogenic effects in athletic populations. The use of DAV should also be explored in clinical populations targeted for improved muscular strength and effective lipid management.

Acknowledgements and Disclosures

Professor Earnest works as the director of research for Nutrabolt International and serves on the graduate faculty at Texas A&M University. Nutrabolt International is a commercial entity that sells nutrition supplements. However, the work presented herein was conceived, performed, and completed several years prior to his employment with Nutrabolt and does not represent a product of interest to the company.

Dr. Broeder presented this work previously as a conference presentation that was not peer reviewed. Since that time the paper has been substantially re-written and re-analyzed.

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