The Impact of Ammonia Inhalants on Neuromuscular Function During Fatigued Isometric Contractions

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Abstract

Ammonia inhalants (AIs) are commonly used by athletes to increase arousal and prepare for maximal physical effort; however, evidence supporting their ergogenic effects remains insignificant and/or inconsistent, particularly when exercise is performed under fatigued conditions. The purpose of this study was to determine the effect of ammonia inhalation on neuromuscular function following fatiguing exercise. Fourteen male varsity athletes completed a randomized, counterbalanced crossover design consisting of an AI condition and a control condition separated by one week. During each visit, participants completed a baseline 30-s maximal voluntary isometric contraction (MVIC) of the elbow flexors, followed by a standardized dynamic biceps fatigue protocol and a subsequent 30-s fatigued MVIC. Force and surface electromyography (sEMG) were recorded concurrently. Maximal isometric force, rate of force development to peak force production (RTP), time to maximal force (T2M), biceps sEMG amplitude, rate of force decline ($RFD_{Decline}$), and mean power frequency (MPF) were assessed. Repeated-measures statistical analyses of variance were used to evaluate differences between treatment conditions and fatigue states.

Maximal isometric force significantly decreased following the fatigue protocol ($p < .001$), with reductions of approximately 20% in both treatment conditions. However, no significant treatment × fatigue state interactions were observed for maximal force, RTP, T2M, biceps sEMG amplitude, or $RFD_{Decline}$ ($p > .05$). MPF progressively decreased across the 30-s fatigued contraction ($p < .001$), consistent with the frequency shift phenomenon and the development of myoelectric fatigue; however, neither the treatment effect nor treatment × time interaction was significant ($p > .05$). Collectively, the results provided evidence that the experimental protocol produced measurable mechanical and myoelectric manifestations of

fatigue but did not provide evidence that ammonia inhalation significantly altered the measured indices of neuromuscular function during fatigued sustained isometric contractions in male varsity athletes. These finding suggest that athletes, coaches, and sport practitioners should not rely on AIs to preserve neuromuscular performance during sustained isometric exercise with performed under fatigue. Instead, performance preparation should prioritize evidence-based strategies for managing fatigue and recovery.

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List of Abbreviations

AI – Ammonia Inhalant

ATP – Adenosine Triphosphate

AMRAP – As Many Repetitions As Possible

BR – Breathing Rate

CMJ – Counter Movement Jump

CTE – Chronic Traumatic Encephalopathy

EMG – Electromyography

sEMG – Surface Electromyography

FFT – Fast Fourier Transform

FSP – Frequency Shift Phenomenon

HR – Heart Rate

IEMG – Integrated Electromyography

IMTP – Isometric Mid-Thigh Pull

MVC – Maximal Voluntary Contraction

MVIC – Maximal Voluntary Isometric Contraction

MAP – Mean Arterial Pressure

MCAv – Middle Cerebral Blood Flow Viscosity

MPF – Mean Power Frequency

MU – Motor Unit

ppm – Parts Per Million

PetCO$_2$ – Partial Pressure of End-Tidal CO_2

PSD – Power Spectrum Density

RFD – Rate of Force Development

$RFD_{Decline}$ – Rate of Force Decline

pRFD – Peak Rate of Force Development

RTP – Rate of Force Development to Peak Force Production

RM – Repetition Maximum

RMS – Root Mean Square

s – Second(s)

SNS – Sympathetic Nervous System

Introduction

While ammonia inhalants (AIs), or smelling salts, are pervasively used in sports (Velasquez, 2011), they are typically used based on anecdotal evidence (Bartolomei et al., 2018). Although there have been many studies examining the effects of AIs, the physiological mechanism with which AIs affect humans remains unknown (Malecek & Tufano, 2021).

Most often used in settings like powerlifting, recent literature describes smelling salts as being ineffective as an ergogenic aid to increase maximal power production (Bartolomei et al., 2018; Malecek & Tufano, 2021; Perry et al., 2016). Despite this, AIs are anecdotally used to achieve the "psyched up" feeling in sports competitions (Pritchard et al., 2014; Rogers et al., 2022). In other anaerobic settings (physical activity lasting 1-30 seconds (s)), conflicting evidence exists regarding the efficacy of AIs in improving performance variables such as peak power, mean power, speed, and fatigue over time (Groop, 2019; Richmond et al., 2014; Rogers et al., 2022; Secrest et al., 2015). Only one study exists where smelling salts were used to examine their effect on anaerobic power production from a fatigued state (Secrest et al., 2015). This study demonstrated that AIs may in fact be beneficial in a fatigued state as increases in peak and mean power were observed during a Wingate trial. Another study investigated the effects of AIs on power production during repeated high-intensity exercise (Rogers et al., 2022). The group found that AIs significantly increased mean and peak power compared to the control trial during 3 repeated 15s Wingate trials. However, the study only included trained, female participants, so it is unclear if this relationship exists in male athletes. Finally, a study by Bartolomei and colleagues (2018) found that during a maximal isometric mid-thigh pull, participants demonstrated a significant increase in peak rate of force development while AI were used, but not in any other evaluated indices (peak force, rate of force development, maximal effort

counter-movement jump power). Although this study did not demonstrate changes in peak force production, the observed increase in peak rate of force development suggests AIs may influence the neural activation of muscles during maximal effort exercises via increased motor unit recruitment.

Several theories exist about the potential physiological mechanism, but there is a large emphasis on AIs interaction with the cardiorespiratory and central nervous systems. Only one study suggests AIs have a possible influence in cerebral blood flow and glucose transport, as an indicator of neural activity, which was conducted within an animal model (Felipo & Butterworth, 2002). Many studies have examined AIs from the perspective of anaerobic power production rather than neuromuscular function (Bender & Popkin, 2023; Malecek & Tufano, 2021).

The studies done by Rogers et al. (2022), Secrest et al. (2015), and Bartolomei et al. (2018) form the basis for this current study, which evaluated the effects of AIs on neuromuscular function following fatiguing exercise. This study differs from that of Rogers and colleagues and Secrest and colleagues, by investigating the effects of AIs from a neuromuscular perspective and from Bartolomei and colleagues by incorporating the aspect of testing from a fatigued state. Concurrent assessment of isometric force and surface electromyography also enables the mechanical and myoelectric responses to AIs to be examined during a sustained contraction.

Thus, the aim of this study was to determine whether AIs altered neuromuscular function following arm fatigue. It was hypothesized that AIs would modify the effects of fatigue on force production and neuromuscular activation. The findings demonstrated that the fatigue protocol reduced maximal isometric force and produced progressing decline in mean power frequency; however, AIs did not significantly alter force production, rate of force development, myoelectric muscular activation, or the examined force indices. These findings are significant because they

extend the limited evidence provided by Rogers et al. (2022), Secrest et al. (2015), and Bartolomei et al. (2018) concerning AI use during fatigue during exercise. These results indicate that the perceived stimulatory effects of AIs may not translate into measurable preservation of neuromuscular performance under these specific conditions. As a result, athletes, coaches, and sport practitioners should not rely on AIs to counteract fatigue during sustained isometric efforts and should instead prioritize evidence-based training, fatigue-management, and recovery strategies. Importantly, these results also do not suggest that AIs hinder fatigued isometric exercise performance and should therefore be considered accordingly. The following section reviews research concerning the physiological and ergogenic effects of AIs, neuromuscular physiology and fatigue, and the methods used to evaluate neuromuscular responses to an intervention.

Review of Literature

In sports, ergogenic aids are utilized to enhance athletic performance. An ergogenic aid can be defined as any method of improving energy production and/or utilization (Silver, 2001). AIs are considered an ergogenic aid and are commonly used in explosive sports such as hockey, football, powerlifting and martial arts. AIs are often used in order to achieve the *psyched up* feeling during competition (P. McCrory, 2006; Pritchard et al., 2014; Tod et al., 2003; Velasquez, 2011). Although AIs are commonly used in sports, they are often used based on anecdotal evidence (Bender & Popkin, 2023; Malecek & Tufano, 2021; Velasquez, 2011). As a result, recent journal articles and several conference articles have been published examining the effect of AIs on human physiology (Bender & Popkin, 2023; Malecek & Tufano, 2021; Velasquez, 2011). While several publications present evidence against the efficacy of AIs (Campbell et al., 2022; Groop, 2019; McEvoy et al., 2019; Perry et al., 2016; Vigil et al., 2018), there are a few studies that have found specific results that may explain the unique circumstances where AIs hold potential benefits as an ergogenic aid (Bartolomei et al., 2018; Rogers et al., 2022; Secrest et al., 2015). These studies, that identified significant results, present a common theme that AIs may influence neuromuscular physiology during pre-fatigued and repetitive fatiguing contractions. As such, the following literature review describes the current understanding of AIs use in sport, physiological mechanism of action, risks and adverse effects, and a critical appraisal of studies that have been published examining its ergogenic potential. Furthermore, the proposed approach to neuromuscular assessment will be discussed to better understand the scope of conclusions that can be made based on different analysis.

Ammonia Inhalants

Chemically, AI compounds are any combination of ammonium carbonate ($(NH_4)_2CO_3H_2O$) mixed with ethanol and water (P. McCrory, 2006). AIs are comprised of 15-20% ammonia and 30-40% ethyl alcohol and are accompanied by various other inactive ingredients, described as the *aromatic spirits of ammonia*, such as eucalyptus, lemon oil, nutmeg oil, and/or lavender oil (Malecek & Tufano, 2021; P. McCrory, 2006). Inhalation of ammonia products in sport are most commonly done in the form of capsules, although alternative methods such as liquids, and powders exist (Pritchard et al., 2014).

Ammonia Inhalants in Sports

While the physiological effect of AIs is still in a theoretical state, their use in sport remains pervasive. Sports such as track and field, football, hockey, mixed martial arts, powerlifting and many more see frequent use of AIs in competition (Velasquez, 2011). Yet, the frequency of use in sports context has not been identified thoroughly. Pritchard and colleagues (2014) conducted an international survey to understand the frequency of AI use in powerlifters. The researchers found that approximately half of the surveyed athletes reported using AIs in competition. More detailed findings revealed that of those athletes who reported using AIs in competition, were in deadlifting events (86.9% of athletes). Interestingly, the frequency of AI use varied between events. In lifts such as the squat and bench press approximately half of those who reported using AIs incorporated AI use in these events (53.8%, and 44.6%, respectively). Pritchard and colleagues (2014) noted a reason for participants using AIs more frequently for the deadlift event is because it is the final lift to be conducted in competition. Competitors described that they are typically mentally and physically fatigued by this point in a regular competition and

they feel using AIs, *helps wake them back up* (Pritchard et al., 2014). These anecdotal reports suggest that athletes find AIs to be most beneficial when they are suffering from fatigue.

In more dynamic sports context such as football and hockey, AIs are often reported to be used for psychological purposes in competition. Athletes using AIs describe them as having a *psyching-up* or *pick me up* effect (P. McCrory, 2006; Tod et al., 2003; Velasquez, 2011). As theorized by Secrest and colleagues (2015), these reports may be due to potential cognitive and central drive enhancements. Regardless, these anecdotal claims have not been proven, and a greater effort is needed to understand the physiological effect of AIs.

Physiological Mechanism

Traditionally, acute inhalation of AIs has been used in medical practice as a method to stimulate the sympathetic response during times of syncope and excessive vagal output which fights feelings of dizziness and fainting (Coleridge & Coleridge, 1985; Peatfield, 1981). The response to AIs has been examined extensively, yet it remains unclear (Bender & Popkin, 2023; Felipo & Butterworth, 2002; Sugden & Newsholme, 1975). Research in animal models suggests AIs may increase cerebral blood flow and glucose delivery (Felipo & Butterworth, 2002). Other animal models indicate AIs may have a role in glycolytic metabolism (Sugden & Newsholme, 1975). Glycolytic metabolism refers to the series of reactions which produce ATP through the breakdown of glucose molecules. These findings suggest AIs may play a role as a mediator of glycolytic ATP production, though they are yet to be confirmed in human trials (Felipo & Butterworth, 2002). Nevertheless, the mechanism of action of AIs remains unclear.

In humans, AI exposure has been shown to increase irritation to the eyes and airways without stimulating cascading inflammatory responses (Sundblad et al., 2004). Widdicombe and Lee (2001), observed that upon acute nasal inhalation of AIs, there is irritation to the upper

respiratory tract through the mucosa which can result in lung irritation and a consequential increased respiratory rate and coughing. Due to the possible irritation to respiratory airways, it is hypothesized that there is an involvement of the sympathetic nervous system as a part of the autonomic nervous system in response to AIs (Velasquez, 2011). The autonomic stimulation, as a result of AI inhalation, is presumed to be associated with the activation of the olfactory and trigeminal nerves (Bensafi, 2002). The activation of the olfactory and trigeminal nerves is thought to result in the stimulation of adrenergic receptors in peripheral tissue, releasing norepinephrine, and increasing cardiac output and respiratory rate (Marshall, 1982). This theory is further supported by observed increases in heart rate, mean arterial blood pressure, breathing ventilation rate, and indices of sympathetic stimulation following acute AI exposure (Groop, 2019; Perry et al., 2016). Although this theory largely makes sense, the specific mechanisms are yet to be confirmed (Malecek & Tufano, 2021).

Risks and Adverse Effects

Although AIs are not fully understood as ergogenic aids, the risks associated with using AIs are well documented. A review by Malecek and Tufano (2021), detailed the potential risks associated with AI use and noted that adverse events can exist as anything from simple respiratory tract irritation to fatal responses to high doses of AIs. In general, adverse events occur based on a dose-dependent relationship, meaning they may occur based on the concentration, time, and amount of ammonia that is inhaled (Leduc et al., 1992). Previous studies in animal models and human trials suggest exposure to large doses (greater than 1500 parts per million (ppm) over 30 minutes) of concentrated ammonia vapours may result in acute injury including allergic reaction, respiratory airway burns, edema and distress (Herrick & Herrick, 1983; Leduc et al., 1992; Perkins et al., 2017; Public Health England, 2015). When consuming high doses of

AI, the response is typically severe, however, it remains individualized from case-to-case. One case of an individual inhaling 10,000 ppm of ammonia for an unknown amount of time experienced death however another case of an individual inhaling 20,000 ppm of ammonia experienced a collapsed lung and body weight loss (Perkins et al., 2017; Public Health England, 2015). Although this is concerning, commercially available AI capsules typically contain 50-100 ppm of ammonia vapours which is significantly less than the amount required to elicit severe injury.

Less severe adverse effects also exist as potential risks associated with commercially available AI exposure. In one case, a frequent user of AIs for powerlifting experienced an acute allergic reaction to a – likely low dose – commercially available AI where the female athlete experienced rhinorrhea, rhinitis, conjunctivitis, dizziness, and severe headaches following an attempt to set a national record (Herrick & Herrick, 1983). The athlete's condition progressed within the hour into shortness of breath, periorbital swelling, and a loss of vision (Herrick & Herrick, 1983). Medical treatment was required to address the symptoms and rectify the athlete's health. Although this is an isolated event of an adverse reaction to commercially available AI exposure, it highlights the possibility of experiencing allergic reactions due to the other components in AI products. Therefore, consideration for using AI products should incorporate cross-referencing of all components of the AI product in question, with all known allergies for anyone who may be exposed to the product (Groop, 2019).

A final consideration for the use of AIs is in contact sports such as American football, or ice hockey. In this context, the use of AIs may be particularly dangerous as their effects can mask the symptomology of head injuries and other traumas (Velasquez, 2011). The unnoticed symptoms can potentially lead to unsafe participation in competition, which may worsen the

severity of the injury. Secondary Impact Syndrome is a specifically relevant concern with AIs where an athlete sustains a head injury, and returns to competition to early then sustaining a second, more severe brain injury (P. R. McCrory & Berkovic, 1998). An investigation into the concussion history of an ex-NFL player who committed suicide, presumably due to the effects of chronic traumatic encephalopathy (CTE), lead to the resurfacing of statements he made about his concussions and AI use (Schwarz, 2007). When asked to recount his history of concussions the player reportedly acknowledged sustaining approximately 15 concussions during his career and described using smelling salts before returning to play without reporting his symptoms (Schwarz, 2007). This unfortunate, yet all too common, behaviour of athletes hiding head injuries may present a specific type of chronic AI misuse that could contribute to CTE development in contact sport athletes (Delaney et al., 2015; Gaetz, 2017; Schwarz, 2007).

In summary, the use of AIs may be accompanied by certain risks and adverse effects. However, these risks can be mitigated by adhering to commercially available doses (50-100 ppm ammonia), completing allergy screening prior to use, and refraining from use if the individual has sustained a head injury or has a respiratory condition. While the exact mechanism of how AIs influence these adverse events remains up to speculation, other assessments of AI exposure have been done to understand their effect on human physiology.

Researched Effects of AIs on Human Physiology and Performance

Cardiorespiratory Effects. Considering the proposed physiological mechanism of AIs interacting with the sympathetic nervous system and increasing cardiac output, it is surprising that few studies have been done to investigate the acute effects of AIs on the cardiorespiratory system. Perry and colleagues (2016) examined the effects of acute AI exposure on cardiorespiratory and cerebrovascular systems concurrently with anaerobic performance

measures. Incorporating 15 resistance trained males, the group evaluated participants over two trials. The first trial collected data before and after AI consumption where they assessed beat-to-beat middle cerebral artery blood flow viscosity (MCAv), heart rate (HR), mean arterial blood pressure (MAP), and partial pressure of end-tidal CO_2 ($PetCO_2$). The second trial had subjects perform an isometric mid-thigh pull (IMTP) immediately, 15, 30, and 60 s after either AI inhalation, or no inhalation (control). MCAv, HR, MAP, and $PetCO_2$ were also measured at the same intervals during the second trial. The study found that only MCAv and HR had significant increases 15 s after exposure, while all other variables remained constant across time points. As described by the authors, the changes in HR and MCAv with no change in MAP indicate that temporary cerebrovascular vasodilation occurred. In other words, AI exposure was shown to result in a transient increase in blood flow to the brain, however, it was not significant enough to alter MAP. The observed data suggest that this phenomenon did not last longer than 15-30 s.

K. Groop (2019) examined the cardiorespiratory response to AIs. Groop's study examined the cardiorespiratory response (HR and breathing rate (BR)) to AIs 5 s before and up to 60 s after AI exposure. The study also assessed reaction time (RT) 1-6 minutes post AI inhalation, and anaerobic power production via a 30 s Wingate 6 minutes post AI inhalation. Sixteen male hockey players were recruited to participate and completed the experimental protocol on two separate visits, once with AIs and once without AIs. The study identified significant increases in HR and BR post-AI exposure, with HR peaking at 7.5 and 10 s after inhalation. While significant differences were identified in cardiorespiratory indices, no changes to anaerobic performance or reaction time were observed.

(McEvoy and colleagues (2019) examined the influence of AIs on pulmonary responses in 22 college students. Participants (11 male and 11 female) completed 1 s forced expiratory

volume and forced vital capacity assessments before and after AI exposure. Results demonstrated no statistical differences in measured variables.

A more recent article by Campbell and colleagues (2022) detailed their study which examined the subjects response to AIs by measuring HR, alertness, isometric MVCs, counter movement jump (CMJ) power, perceived performance, electromechanical delay, and, therefore, overall RT. Fourteen non-resistance trained males completed three trials separated by 7 days each. The three trials differentiated by control, experimental (AI) and sham conditions. The group observed no changes to ergogenic indices, however, increases in HR, arousal, and perceived performance were demonstrated in the AI condition when compared to both control and sham conditions. Additional analysis revealed there was no correlation between HR and alertness. This suggested that the sympathetic nervous response was not solely responsible for the observed increase in arousal (Campbell et al., 2022).

In summary, three studies evaluating HR in response to AI exposure demonstrated significant increases after inhalation (Campbell et al., 2022; Groop, 2019; Perry et al., 2016). Two of these studies noted that the rise in HR was transient, only lasting 15-30 s before returning to baseline levels (Groop, 2019; Perry et al., 2016). These data present valuable insight into the potential duration with which AIs act on human physiology. As such, many subsequent studies have aimed to conduct testing within 30 s of AI exposure (Archer et al., 2017; Bartolomei et al., 2018; McEvoy et al., 2019; Rivera et al., 2017; Rogers et al., 2022; Vigil et al., 2018). Additionally, Campbell and colleagues' (2022) correlational findings between HR and arousal, or more specifically the lack thereof, suggest the sympathetic nervous response to AIs is not entirely responsible for their observed increases in arousal. This idea corresponds with the findings of a previous study which noted transient increases in MCAv immediately after AI

inhalation (Perry et al., 2016). As a result, Campbell and colleagues' (2022) findings suggest the observed increase in arousal following AI exposure may be attributed to an increase in arterial blood supply to the brain. Unfortunately, research investigating the respiratory system's response to AI exposure has similarly few studies and is arguably less understood. While K. Groop (2019) observed transient increases in BR in response to AI inhalation, and McEvoy and colleagues (2019) found no differences in pulmonary indices, no other studies, to our knowledge, have attempted to understand this relationship. Fortunately, other research assessing the effects of AI has been completed providing some insight into potential strength and power applications.

Strength and Ballistic Power. Literature supporting the notion that AIs provide ergogenic benefits to strength or power is sparse and is often accompanied by conflicting evidence in similar studies (Malecek & Tufano, 2021). Regardless of the lacking evidence, AIs are most frequently seen to be used in sports that demand large strength capacities and ballistic power production (Pritchard et al., 2014; Velasquez, 2011). As such, the majority of recent literature investigating the effects of AIs primarily examine its ergogenic potential for strength and/or ballistic power application (Bender & Popkin, 2023).

The earliest peer-reviewed study on the application of AI use for strength performance was conducted using 25-resistance trained male athletes, following a repeated measures, cross-over control study design (Richmond et al., 2014). The study included two trials separated by 2-4 days. Participants were randomly assigned to the AI condition or the placebo (Vick's VapoRub) condition on the first trial, then repeated the same protocol with the other condition on the second trial. Each trial, participants completed a standardized warm-up and were then asked to complete as many reps as possible (AMRAP) at 85% of their 1 repetition maximum (1RM) for the back squat, 3 s after inhalation. After 5 minutes rest, the same protocol was completed except

it was with the bench press exercise. The study found no differences in the number of repetitions completed between the AI or placebo conditions for both exercises. This suggests that AI do not provide an ergogenic effect to the AMRAP protocol. However, no conclusions can be made about the velocity of repetitions or power output throughout the series of repetitions completed. As the only study evaluating AIs over multiple strength-based repetitions, this provides an opportunity for future studies to evaluate AIs potential effects on velocity, power output, and fatigue in multiple strength-based repetitions (Malecek & Tufano, 2021).

Perry and colleagues (2016) examined the effect of AI exposure on muscle force, rate of force development (RFD), peak rate of force development (pRFD), and surface electromyography (sEMG) during a maximal effort IMTP. RFD and pRFD are variables which reflect the rate of explosive force generation during a given contraction while sEMG evaluates muscular activation deep to the sEMG electrode. The study included 15 resistance trained male participants, with no history of AI use. Five trials of IMTPs were conducted for each participant, each of which included an IMTP immediately, 15, 30, and 60 s after either AI inhalation or no AI inhalation (control). Each IMTP repetition was performed for 2 s. After completing a trial, 5 minutes of rest was given before beginning a subsequent trial. The treatment variable was randomly assigned to participants for each trial. The results demonstrated no statistical difference between AI or control treatments amongst all measured variables. However, there are potential limitations that should be considered regarding the results of this study. First, although the randomization technique used (Latin Square Design) in this study is well accepted, the Latin Square Design has certain assumptions that are violated in this study (Salkind, 2006). First, the Latin Square Design assumes that the number of conditions will be equal to the number of trials (columns) and number of participants (rows), creating a square matrix. Next, the Latin Square

Design is restricted so that each row and column can only have one treatment condition existing within it. This creates a balanced distribution of treatment conditions amongst participants and trials allowing the reduction (or elimination) of the order-effect in repeated measure studies with multiple conditions. This study has 2 treatment conditions, 15 participants, and 5 trials, where no participant completed equal trials in the AI or control conditions. As a result, the application of the Latin Square Design method for randomization in this study compromises its very purpose by creating an unbalanced distribution of treatment conditions which does not reduce the potential order effect. Another limitation to this study that may have influenced its results is the post-activation potentiation phenomenon (Lorenz, 2011; Malecek & Tufano, 2021). This is a phenomenon by which the force exerted by a muscle is increased due to its previous contraction (Lorenz, 2011). Although an insignificant positive effect may have been demonstrated in pRFD at 30s post-inhalation, post-activation potentiation may have been a contributing factor, irrespective of AI use (Malecek & Tufano, 2021). As such, the potential information derived about the timing and duration of AIs may not be accurate. In summary, this study did not identify any differences in IMTP indices or sEMG between AIs and the control, however limitations related to study design and confounding factors may have contributed to the results.

The potential limitations of the previous study are particularly important to acknowledge as a study following that of Perry and colleagues (2016), identified contrasting results using similar test measures (Bartolomei et al., 2018). Bartolomei and colleagues (2018) conducted a counterbalanced crossover study examining the effects of AIs on power and maximal force production. The study consisted of three different assessments during each of the three trials conducted, which were separated 48 hours apart. Measuring power output, the study evaluated 20 resistance-trained male participants using a counter-movement jump (CMJ). This test

assessing the participants explosive lower body power by evaluating CMJ height relative to their body mass. To evaluate RFD and maximal strength, the study also asked these participants to complete a maximal IMTP. As previously described, RFD describes the rate of explosive force generation, while maximal strength is reflected as the peak force output during a contraction. Participants performed the maximal strength and power tests using either AI, placebo (Vick's VapoRub), or no inhalant, 10 s after treatment variable admission. The study was conducted in a randomized order, while incorporating double-blinding so that the treatment variable was unknown to both participants and investigators. The study found no significant difference relating to power production or maximal strength, however the authors identified a significant increase in pRFD for the AI condition during the IMTP. These results directly contrast those of the previous study of Perry and colleagues (2016). Although the results did not indicate AIs have an enhancing effect on maximal force production or lower-body peak power output, the results suggest AIs may increase explosive force output.

Another study investigating the influence of AIs on maximal strength assessed 20 undergraduate science students (10 male and 10 female), with a minimum of two years of resistance training history, through evaluating 1RM deadlift performance (Vigil et al., 2018). Participants completed 3 visits, separated by 72 hours of rest. The first trial was used to establish a baseline deadlift 1RM using traditional progressive 1RM protocol. The second two visits were counter-balanced so that participants received AI or placebo (water) treatments in a random order between trials. Also using the traditional progressive 1RM protocol during these trials, when attempting 102.5, 105, and 107.5% of their baseline 1RM, the participants received either AI or placebo treatments 15 s prior to the attempt. The authors purposely assessed 1RM deadlift performance 15 s after inhalation as this most similarly follows the timeline of AI use in

powerlifting competitions. However, the results of the study did not identify any ergogenic effect of AIs on 1RM deadlift performance for male or female participants. This suggests AIs do not provide any competitive advantage to powerlifters in the deadlift. However, it is important to consider that AIs are most frequently used in powerlifting competitions for the deadlift event because it is the last event to be conducted (Pritchard et al., 2014). Anecdotal reporting from powerlifting competitions suggests AIs act as a *pick me up* to counter the accumulated fatigue throughout a competition day (P. McCrory, 2006; Pritchard et al., 2014). Therefore, this study provides an opportunity for further research into the use of AIs in pre-fatigued deadlift performance.

More recently, Campbell et al., (2022) conducted a study a study on AIs assessing maximal isometric strength, sEMG, and peak power production. Fourteen non-resistance trained males participated in two trials, separated by 7 days, each taking place at the same time of day. The trials consisted of AI, or sham (water) conditions, where the order of trials were determined through participant block randomization. For each trial, participants completed a standardized submaximal warm-up followed by three sets of three tests including a knee extension maximal voluntary isometric contraction (MVIC), a handgrip MVIC, and a CMJ. For the MVICs, contractions lasted 5-s followed by 5 minutes of rest before attempting the next set of that test. Peak power was assessed via 3 maximal effort CMJs, each attempt being separated by 5 minutes of rest. Additionally, each test was separated by 20 minutes of rest. After inhalation of the AI (or water), a randomized audio cue was played within 30-s to evaluate reaction time and electromechanical delay. Assessment of the time from stimulus recognition (audio cue) to response (action) provides information about reaction time. On the other hand, electromechanical delay is described by the delay between sEMG onset and force production which provides

information about neuromuscular muscular activation. Although the authors revealed HR, alertness, and perceived performance were significantly increased by AIs, no other indices of functional performance changed. These measures included reaction time, electromechanical delay, RFD, peak force, and peak power. As a result, the study was unable to observe any improvements in maximal strength or peak power output following AI use.

One last peer-reviewed study investigated the effects of AI inhalation on repeated high-intensity exercise performance (Rogers et al., 2022). This study followed a single-blinded cross-over counterbalance design with 12 physically active female participants. Subjects completed two trials separated by at least 48 hours, each with different treatment variables (AI or control) ordered randomly. Each trial consisted of 3 × 15-s Wingate anaerobic cycle tests with 2 minutes of active recovery separating each bout. Participants received either treatment variable 10-s prior to each Wingate bout. When evaluating the individual repetitions of the Wingate test, there were no statistical differences observed between the AI and control conditions. However, the study found that acute AI inhalation significantly increased peak power and mean power on average over 3 × 15-s Wingate tests. Interestingly, it did not influence fatigue index. This study therefore suggests that AIs may possess ergogenic potential for increasing anaerobic power output for repeated high-intensity exercise in females.

Although there is a growing body of peer-reviewed literature examining the impact of AIs on strength and power enhancement. A large contribution to early AI research was presented in conference articles and a thesis dissertation. As many of the peer-reviewed studies incorporated information gleaned from these projects, they are certainly worth discussing. Interestingly, many of these articles demonstrate collaboration amongst authors such as studies by Mitchell et al., (2015); Richmond et al., (2014); and Witherbee et al., (2013), and also with

Archer et al., (2017) and Rivera et al., (2017). This further demonstrates how little knowledge and interest there was in examining AIs as ergogenic aids only 10 years ago.

For example, Witherbee and colleagues (2013) conducted a study evaluating the impact of AIs on the standard 30-s Wingate performance (Witherbee et al., 2013). The repeated measures study design evaluated 3 resistance trained male participants over 3 conditions in a randomized order. Subjects received AI, Vick's VapoRub, or no inhalant immediately before the test. The study did not identify any differences in Wingate indices. However, only having 3 participants may have been a contributing factor in establishing statistical significance if there truly was a change in the indices.

Another conference article study investigated the influence of AIs on vertical jump height and broad jump distance. Twelve subjects were told to inhale 1 of 3 randomly chosen vials containing either AIs, Vick's VapoRub, or a control (no scent) immediately prior to the maximal effort jumps. The study found that there were no significant differences in vertical jump height or broad jump distance.

Subsequently, a pair of conference papers were authored by M. Rivera and D. Archer in 2017. Although they published separate studies, the similarities in dates, participants characteristics (8 male and 3 female), identical treatment variables (AI, no inhalant, menthol, and high-potency ammonia), association with the same university, and co-authorship of both individuals on either article makes it reasonable to assume the same participants and potential bias existed in both studies or that the data were interpreted two different ways. First, Rivera et al., (2017) conducted a study examining the influence of inhalants on rate of force development and peak force during an IMTP. Eleven resistance trained individuals (8 male and 3 female) completed 4 IMTP on separate days for each treatment condition (AI, no inhalant, menthol, and

high-potency ammonia). Participants completed each IMTP 30 s after inhalation of the treatment variable. The findings suggest that inhalants have no effect on maximal or explosive isometric force production. Next, Archer et al., (2017) conducted a study, presumably using the same 11 participants and treatment variables, where CMJ height and 20-meter sprint time were assessed. Subjects completed 4 trial visits beginning with the no inhalant condition to establish a baseline. The subsequent 3 trials were randomly ordered by the remaining treatment conditions (AI, menthol, and high potency ammonia). During each trial, subjects performed 3 CMJs and 2 20-meter sprints. Participants were instructed to wait 30 s after inhalation prior to commencing each attempt. Results demonstrated no condition provided enhancements to CMJ height or sprint time.

Another conference article detailed a repeated measures study by Secrest (2015) which examined the influence of AIs on anaerobic power and fatigue. Ten anaerobically trained male participants complete a Wingate anaerobic cycling test following a simulated American football game via a sprint protocol. Participants completed two trials separated by a minimum of 48 hours. The first trial was conducted with the control condition and the second with the AI condition. The AI condition was administered by capsule immediately before the final Wingate. The results showed significant increases in peak power and mean power between the control and AI trials. This study demonstrates that AIs may be beneficial in increasing anaerobic power production when administered from a fatigued state. However, it is important to identify the potential for the order effect influencing results in this study (Salkind, 2006). Interestingly, this study is the only other one to demonstrate improvements in Wingate indices other than that of Rogers et al., (2022). Although the methodological validity of the two studies varies greatly, both studies observed improvements in peak power and mean power on the Wingate test when conducted from a fatigued state.

Finally a thesis dissertation study by Groop (2019) further examined the effects of AIs on Wingate performance. The study featured a counterbalance cross-over design where 16 male hockey players were recruited to participate and completed the experimental protocol on two separate visits, once with AIs and once without AIs, in a randomized order. Each trial consisted of a series of tests following the admission of the treatment variable. First the cardiorespiratory response to AIs was examined for 60 s after inhalation. Then reaction time was assessed 1-6 minutes post AI inhalation, and anaerobic power production via a 30 s Wingate 6 minutes post AI inhalation. While significant differences were identified in cardiorespiratory indices, no changes to anaerobic performance or reaction time were observed.

As documented in the literature, there are several studies whose results contradict each other regarding the potential benefits AIs may hold for strength and/or power. Some articles suggest they do not provide ergogenic potential for anaerobic power output (Archer et al., 2017; Campbell et al., 2022; Groop, 2019; Mitchell et al., 2015; Witherbee et al., 2013). While other articles demonstrated that AIs may increase power production (Rogers et al., 2022; Secrest et al., 2015). As suggested in the survey responses collected by Pritchard and colleagues (2014), AIs seem to act as a *pick me up* which implies that they are used to overcome fatigued states. This commonality is reflected in the work of Rogers et al., (2022) and Secrest et al., (2015). While many of these studies have evaluated AIs using Wingate protocols, only those of Rogers et al., (2022), and Secrest et al., (2015) encompass an aspect of fatigue induced before or throughout Wingate trials. The ability of AIs to potentially reduce the influence of fatigue on power production is exciting for practical applications, however more research is required before this theory can be accepted.

When examining the body of literature detailing the effects of AI on strength benefits, it is equally as unclear. While Bartolomei and colleagues (2018) produced the only study to demonstrate a significant increase in the rate of explosive force production (pRDF) during and IMTP, there are a pair of other studies examining AIs that contradict their findings (Perry et al., 2016; Rivera et al., 2017). Contradictions, though possibly due to methodological limitations, are contradictions, nonetheless. More research is needed to understand the relationship between AIs and potential strength benefits.

Cognitive Effects. While strength and power are two important components of athletic performance in many domains, arousal is particularly important in sports which require reactive movements and quick information processing. Athletic performance is influenced by many factors, however arousal is known to be an important modulator of both motor-performance and cognitive function (Finke & Schächinger, 2020). Increases in arousal/alertness, including increases mediated by AIs, have been shown to be highly correlated to performance (Campbell et al., 2022; Gould & Krane, 1992; Rogers et al., 2022). Additionally, in gross motor tasks lower levels of arousal is associated with poorer performance (Zaichkowsky & Naylor, 2004). Arousal is a complex psychological and physiological construct that is not directly measurable, however, there are specific physiological systems which influence arousal. The most relevant of these systems being *SNS-adrenal-medullary arousal* (Dienstbier, 1989). This system features the mediation of arousal via the hypothalamus acting to increase sympathetic activity to stimulate the adrenal medulla and release adrenaline. Furthermore, in this model synapses in the sympathetic nervous system are also stimulated to release noradrenaline. These two neurotransmitters are linked to various other physiological responses which can be measured as a means of indirect objective evaluation of arousal. For example, increases in heart rate, cardiac

output, and respiratory rate, are considered to be a result of increased sympathetic activity, and thus indicate increased levels of arousal. Alternatively, arousal can be directly assessed through subjective measures via visual analog scales (Rogers et al., 2022). In this approach, participants self-report their subjective feelings of arousal, alertness, or *psyched-up* energy on a 100 mm line where 0 represents the absence of these feelings and 100 represents the strongest possible feeling. While visual analog scales have been proven to be reliable and valid in the assessment of arousal, they are particularly vulnerable to participant bias (Aitken, 1969; Åkerstedt & Gillberg, 1990; Zhou et al., 2012). For example, when participant blinding is not accomplished between treatment variables, participant bias may present limitations concerned with validity. This is a particularly relevant concern with AI research due to the lack of an effective placebo control substance. Regardless, visual analog scales have still been used in AI research to assess arousal alongside other indirect measurements of arousal such as HR (Campbell et al., 2022; Rogers et al., 2022).

The use of AIs to enhance arousal is well documented however there is conflicting evidence as to whether it can enhance reaction time to a stimulus. Several studies support the notion that AIs stimulative properties on the sympathetic nervous system can increase psychological arousal (Bartolomei et al., 2018; Campbell et al., 2022; Groop, 2019; Rogers et al., 2022). A study performed by Campbell and colleagues (2022) investigated this phenomenon in the context of neuromuscular performance. The group found that although acute inhalation of AIs increased participant's subjective feelings of arousal, HR, and perceived performance though it did not significantly change reaction time. Similarly, Rogers et al., (2022) assessed perceived arousal concurrently with other anaerobic indices. The group found that perceived alertness was significantly higher over 3 repeated Wingate trials. However, other measures used to assess

psychological responses, such as rate of perceived exertion and HR, were not different between the AI and control conditions. These findings are contrasted by the findings of Campbell and colleagues (2022) however the differences may be due to the nature of the exercises used for examination in each study. Therefore, more research is needed to understand this relationship. Future studies might achieve this by combining the methodology of the studies by Campbell et al., (2022) and Rogers et al., (2022).

Neuromuscular Evaluation

The body of research investigating the potential benefits of AIs has produced substantially more evidence dismissing AIs as effective ergogenic aids, compared to the alternative (Bender & Popkin, 2023; Malecek & Tufano, 2021). However, the few articles that have observed significant ergogenic effects may be due to enhancements in muscle activation. Bartolomei and colleagues (2018) observed an increase in pRFD in response to AIs suggesting that there may have been an influence on motor unit recruitment. Studies by Secrest et al., (2015) and Rogers et al., (2022) observed increases in Wingate power output from a fatigued state and throughout consecutive fatiguing trials, respectively. While other studies exist dismissing beneficial effects of AIs on Wingate performance (Groop, 2019; Witherbee et al., 2013), the studies examining the effects of AIs throughout and after fatiguing exercise suggest they may alter the influence of fatigue on muscular activation patterns (Rogers et al., 2022; Secrest et al., 2015). Unfortunately, these studies did not directly evaluate muscular activity in the context of fatigue and were confined to speculation. To do so effectively, sEMG analysis could be utilized to assess myoelectric changes which reflect different muscular activation patterns.

sEMG analysis performed by Perry et al., (2016) and Campbell et al., (2022) were unable to identify myoelectric differences between AI and control conditions, however they both

conducted narrow EMG analyses within their studies. Perry and colleagues (2016) utilized time-domain analysis to evaluate percentage muscle activation in resistance trained males from a rested state with and without AIs. The inclusion of resistance trained participants may have hidden the influence of AIs on muscular activation because of the neuromuscular adaptations associated with training. Motor learning studies have demonstrated that resistance training and continued task development is characterized by decreased amplitude and duration of muscle activity, and decreased muscle co-activation (Bonacci et al., 2009). Assuming the participants in this study have trained enough to develop their neuromuscular characteristics as described by Bonacci and colleagues (2009), these adaptations may exceed the neuromuscular benefit of AIs from a rested state. Alternatively, Campbell and colleagues (2022) utilized sEMG analysis to exclusively assess electromechanical delay which describes the latency between the onset of myoelectric signals and force production. While this is a valid application of sEMG analysis in research, it only provides information about the state of neuromuscular activation at the beginning of a muscle contraction and not throughout its duration.

To best utilize sEMG in the assessment of AIs, it is important to understand relevant topics in neuromuscular physiology and how they are measured and analyzed thoroughly. Additionally, it is important to recognize how concepts like training adaptations, fatigue, and stimulants may alter myoelectric signatures so that valid conclusions can be made about observed changes in the participants neuromuscular physiology.

Neuromuscular Physiology

A motor unit (MU) is the functional unit of the neuromuscular system (Sherrington & Liddell, 1925). MUs are composed of an effector motor neuron and all of the muscle fibers that are activated by it (Buchthal et al., 1959). Activation of an MU is accomplished by electrical

action potentials which runs down from the motor neuron via the axon and extends to the terminal branches where the end plates and neuromuscular junction is found (Staudenmann et al., 2010). The electrical signal crosses the junction as acetylcholine is released from the endplates and attaches to ion channels on the muscle fiber. Initially in a resting state of electrical potential, the presence of acetylcholine opens ion channels which brings sodium ions into the cell, and potassium ions out of the cell. This results in depolarization of the designated muscle fiber membrane and initiates muscle contraction. There are two primary mechanisms with which the nervous system are able to activate muscles to produce force: 1) MU recruitment, where MUs are activated to increase the magnitude of force, and 2) rate coding, where already activated MUs are signaled to fire at a faster rate (Farina et al., 2016; Kukulka & Clamann, 1981; Van Bolhuis et al., 1997). MU recruitment patterns are task dependent and they change based on the rate of force development required for a given contraction (Desmedt & Godaux, 1977). MUs are recruited based on size, where MUs are activated in order from smallest to largest (Henneman, 1957). Known as the *size principle*, this rule allows the recruitment of small, fatigue-resistant MUs for fine motor control and the recruitment of large, fatigue-nonresistant MUs for explosive movements. While every muscle fiber within a MU has the same histochemical and biomechanical characteristics, it is also important to understand the differences in muscle fiber types (Staudenmann et al., 2010).

Muscle fibers exist in one of two classifications, type I (slow) muscle fibers and type II (fast) muscle fibers, based on histochemical analysis (Barany, 1967). While other subclassifications of muscle fibers exist, such as type IIA and type IIB, only the main classifications will be discussed for the purposes of this review (Scott et al., 2001). Type I and II fibers can be further differentiated based on morphological differences with type I fibers

appearing more red, while type II fibers appearing more white (McComas, 1996). The observed redness of type I muscle fibers is a result of high amounts of myoglobin and capillarization of the muscle. Consequently, the increase myoglobin and capillary content contribute to larger oxidation capacity of type I muscle which enables fatigue-resistant muscle contractions.

When examining force production capacity, the functional unit of the muscle fiber provides information on the rate at which a muscle can generate force (Scott et al., 2001). The sarcomere is the functional unit of a muscle fiber and is composed of thick myosin filaments and thin actin filaments (McComas, 1996). When a muscle fiber receives stimulus from its motor neuron, Ca^{2+} is released into the sarcoplasmic reticulum leading to the exposure of the myosin binding cite on the actin molecule (Plowman & Smith, 2013). When ATP is available, the myosin head binds to actin and pulls the filament along the sarcomere, releases, and reattaches (muscle contraction). This is known as cross-bridge cycling. The rate at which this cycle can occur is primarily limited to the rate at which the ATPase on the myosin head can hydrolyze ATP. In the case of type II muscle fibers, myosin ATPase hydrolysis rates are 2-3 times faster than those in type I fibers (Taylor et al., 1974). This allows type II fibers to engage in rapid cross-bridge cycling resulting in increased force production rates and capacities. As a result, type II muscle fibers are characteristically susceptible to fatigue due to the rate at which ATP is utilized within the sarcomere (Scott et al., 2001). Understanding these fine mechanics of neuromuscular physiology is important when utilizing techniques like electromyography to assess neuromuscular function.

Electromyography

Electromyography (EMG) is a technique that measures the electrical activity produced by skeletal muscles (Jang et al., 2018). It provides a means to evaluate the activation patterns of

motor units within a muscle and can be used to infer neuromuscular adaptations related to force production. By examining the EMG signals during isometric strength testing, researchers can gain information about the rate of force development and muscle activation patterns utilized during maximal or submaximal contractions (Lee et al., 2012). In the context of examining the effects of AIs on neuromuscular function, EMG studies may be used to measure the neuromuscular responses to AI exposure following fatiguing exercise.

Specifically, EMG evaluates changes in myoelectric signal amplitude and frequency characteristics, which can provide information regarding the overall level and temporal characteristics of muscular activation (Hwang et al., 2016). These signals reflect the summation of motor unit action potentials detected within the recording area; however, conventional sEMG does not directly distinguish the recruitment or discharge behaviour of individual motor units. sEMG was created in an effort to simplify EMG readings and reduce the invasiveness of the measurement procedure for the participant. Indwelling EMG readings involve the insertion of a needle electrode directly into the muscle tissue, whereas sEMG recordings place gel-adhesive electrode pads on the skin directly above the target muscle. Although sEMG measurement is less invasive, it is also less sensitive and specific due to noise, and proximity of the electrodes to the muscle. sEMG signals represent the sum of motor unit action potentials which travel along muscle fibers and trigger contractions (Costa-García et al., 2022). However, technological development and filtering techniques have enabled the application of sEMG devices to infer neuromuscular properties of contraction in the assessment of large muscles such as the biceps brachii, or the rectus femoris (Jang et al., 2018).

Although sEMG testing itself does not directly measure force production, combining it with isometric strength testing can offer valuable insights into various aspects of muscle function

closely associated with power production, such as neural activation of muscles and myoelectric manifestations of fatigue (Fengjun Bai et al., 2012; Hwang et al., 2016; Staudenmann et al., 2010). By analyzing sEMG signals, researchers can examine changes in the global myoelectric activity of a muscle during different phases of contraction (Staudenmann et al., 2010; Vigotsky et al., 2018). Changes in signal amplitude and spectral characteristics may reflect alterations in the underlying neuromuscular system; however, conventional surface EMG does not directly identify the recruitment or firing behaviour of individual motor units or the activation of specific muscle fibre types. Consequently, sEMG is most appropriately used in the present context to evaluate changes in overall muscular activation and the myoelectric manifestation of fatigue. Additionally, repeated testing between rested and fatigued states allows for analysis of myoelectric responses to a stimulus during fatigue muscle contractions.

While muscle force production and EMG signals do not always display a linear relationship, isometric contractions provide a unique opportunity for EMG researchers to analyze force production and EMG signals linearly (Hwang et al., 2016; Rantalainen et al., 2012; Roberts & Gabaldon, 2008). In the context of sustained isometric contractions, EMG analysis allows for more detailed understanding of the myoelectric manifestation of fatigue.

Manifestations of Fatigue. Muscle fatigue is a term used to describe a transient decrease in force production capacity by a muscle (Kallenberg et al., 2007; Lorist et al., 2002). Fatigue can occur as a result of impairment of several physiological processes (Abd-Elfattah et al., 2015). During maximal voluntary contractions, fatigue manifests as a gradual decrease in force production. However, during submaximal contractions fatigue is presented as the inability to maintain a certain level of force resulting in activity failure.

The inability of a muscle to generate sustained force is the result of a decrease in muscle firing from two main factors: 1) the central nervous system, and/or 2) the peripheral nervous system (McKenna & Hargreaves, 2008). In this context, central fatigue is defined as a decrease in the voluntary activation of muscles. This is presented as a decrease in frequency and synchronization of motor neurons, and reduced motor drive from the motor cortex in the brain. Conversely, peripheral fatigue describes a decrease in contractile strength of muscle fibers with changes in the mechanisms underlying the transmission of muscle action potentials (Ament & Verkerke, 2009; Korzeniewski, 2019). Peripheral fatigue manifests as a reduction in efficacy at the site of the neuromuscular junction and processes beyond as changes in metabolic and biochemical occur within the muscle.

Temporal Analysis of Force and Time. In the context of EMG analysis, fatigue manifestation is quantifiable through various techniques that reveal insight into the physiological mechanism that the muscle is responding with. The idea that EMG magnitude and intensity increases as muscle force production increases is well accepted in the scientific community (Hwang et al., 2016; Inman et al., 1952). In the time domain, however, fatigue development is also characterized by increased amplitude of the electrical voltage, which can be expressed as root mean square (RMS) values in EMG signals (deVries, 1968). During prolonged maximal muscle contractions, increased neural drive is required to overcome peripheral fatigue related loss of force production capacity of muscle (De Luca, 1984). Understanding muscle fiber type is critical to understanding mechanisms for fatigue. During sustained muscular contractions, changes in sEMG amplitude may occur as the neuromuscular system attempts to maintain force production despite the development of fatigue (De Luca, 1984). These changes reflect the combined influence of several physiological and methodological factors, including motor unit

recruitment and discharge behaviour, changes in muscle fibre action potentials, and amplitude cancellation. Consequently, increases or decreases in conventional sEMG amplitude during fatigue should not be interpreted as direct evidence of the recruitment of a specific muscle fibre type (Allen et al., 2008; Burke et al., 1973).

Spectrum Frequency Analysis. Alternative EMG analysis, in the frequency domain, can evaluate fatigue through a decrease in rate of action potential conduction velocity as shifts are observed toward lower frequencies during prolonged contractions (Petrofsky & Lind, 1980; J. Potvin, 1997). In spectral frequency analysis, the frequency content of the sEMG signal can be examined to characterize changes in the myoelectric signal during sustained contractions. During fatigue, the power spectrum commonly shifts toward lower frequencies, including reductions in higher-frequency components and increases in lower-frequency components (Kadefors, 1968; Kaiser & Petersen, 1962).This response is generally described as the frequency shift phenomenon. Measures such as mean power frequency and median frequency can therefore be used to characterize the temporal development of myoelectric fatigue. However, these spectral changes are influenced by several physiological factors and should not be interpreted as direct measurements of individual motor unit firing rates or the recruitment of specific muscle fibre types.

Data Filtering and Noise. sEMG data must be filtered to remove several sources of noise that may be influencing the data. Noise can be produced by movement of electrodes, cables and connectors, originating from the subject moving (Staudenmann et al., 2010). These sources are thought to contribute to relatively low frequencies (<10 Hz) of noise. Other sources of noise also exist such as common-mode noise. This is one such example where a reference electrode is placed over a non-muscular tissue to record common electrical signals passing through the body

(Stegeman et al., 1997). These signals are then subtracted from the target electrode recordings to eliminate common-mode noise (Botter & Vieira, 2015). However, common-mode noise is only of concern when using monopolar electrodes. Utilizing a bipolar electrodes, connected to a differential amplifier, accounts for and removes the common signals between the two electrodes, thus minimizing common-mode noise (Dimitrov et al., 2003). Another potential source of noise is muscle cross-talk, which refers to signal contribution from muscles near the muscle being investigated (Basmajian, 1988; Türker & Miles, 1990). Depending on the size and thickness of a muscle, cross-talk noise will vary. Signal contribution from other muscles is mainly the result of standing waves, which manifests as high-frequency content (Dimitrova et al., 2002). To minimize cross-talk, sEMG studies are best to investigate large, superficial muscles that are not surrounded by other large muscles or muscle groups.

In general, each muscle group is also associated with a particular frequency range that is accepted to be the truest myoelectric signal. As such, specific filters are applied to sEMG data based on the muscle group being investigated. For example, in the investigation of the biceps brachii, a common frequency range that is used is 10-500 Hz (Ahamed et al., 2015; Carr et al., 2016; Georgakis et al., 2003; Rantalainen et al., 2012). To filter out noise frequencies existing outside this range, a bandpass filter is used to remove both low (<10 Hz) and high (>500 Hz) frequency signals from sEMG data. Alternative filters also exist such as low-pass, and high-pass filters, which remove signal below or above a certain frequency, respectively (J. R. Potvin & Brown, 2004; Staudenmann et al., 2010). Finally, more specified bandpass filters can be created through conducting a power spectrum analysis to identify the dominant (true) frequency of the EMG signal. In this analysis, the upper and lower limits of the bandpass filter are created to exclude all sources of noise that exist outside of the true muscle frequency range. This approach

will account for unique sources of noise, such as electrical circuitry, which exist in the specific environment that the testing is being conducted. It is also important to recognize that data filtering techniques can only exclude noise that exist within the filter parameters. Any noise that is present within the accepted frequency range of the filter is more difficult to remove because it may interfere with the preservation of the desired signal. While many filtering techniques exist, they each present certain advantages, disadvantages, and limitations depending on the context of the data collection. As such, it is important to consider all potential sources of noise, the anatomical positioning of electrodes, and the type of electrode configuration used when filtering sEMG data.

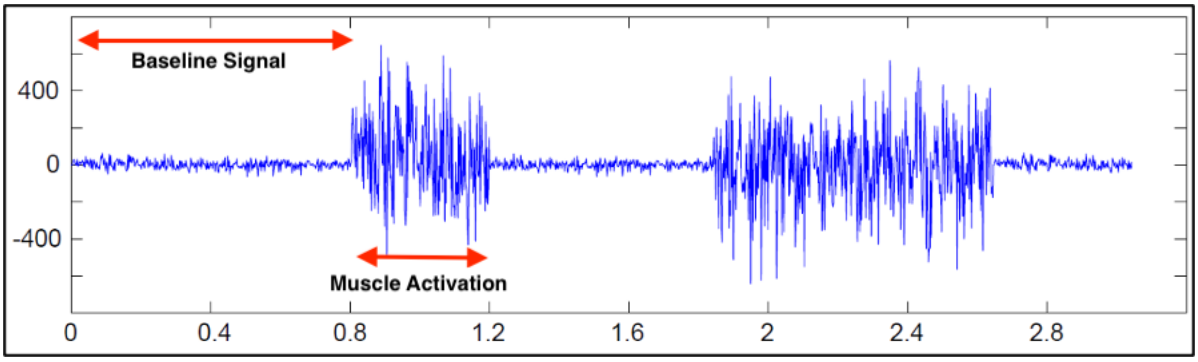
Electromyography Analysis. Changes in sEMG signals are based on the recruitment and firing rates of MUs within muscle (Hwang et al., 2016). Interpretation of these changes can provide insight into the degree of force produced by a muscle and its level of fatigue using time and frequency domain analysis.

Information regarding muscle force is analyzed using time-domain analysis where the amplitude of the EMG signal is examined (Soderberg, 1992; Winter, 2009). Integrated EMG (IEMG) or selected windows of data, and RMS provide information as to whether the muscle is active, and the relative amount of muscle activation. The mean RMS of IEMG windows is particularly useful evaluating changes in EMG amplitude over the course of a prolonged contraction (Ahmadi et al., 2021; Triwiyanto et al., 2018). For example, RMS can be integrated over 100-ms windows following the beginning of a MVIC. To consolidate data within these windows, a mean value can be calculated by dividing RMS data by 100-ms. As a result, the influence of post-filtered data noise is reduced, and the data is easier to plot along and contrasts against other data sets.

General fatigue analysis can also be examined by observing the slow of linear increases in IEMG windows and decreases in force production throughout a MVIC (Hwang et al., 2016). The time domain analysis can also provide information as to whether or not the muscle is active and the relative amount of activity of the muscle, as seen in Figure 1 (Hwang et al., 2016).

Figure 1.

Visualization of Muscle Activation on an EMG signal



Note. This figure depicts a raw EMG signal where the y-axis is in mV, and the x-axis is time in seconds.

Establishing an IEMG window (500-ms) centered around the peak force production of a MVIC can produce data related to an individual's maximal EMG (EMGmax) amplitude (Neyroud et al., 2013). In doing so, submaximal, and fatigued EMG data can be normalized as a percentage of rested EMGmax and therefore represents the relative activation of a muscle. Furthermore, antagonist muscle groups can be evaluated concurrently with the target muscle using the same analysis to examine co-activation percentage and timing associated with muscle contractions.

Meanwhile, in the frequency domain analysis, mean power frequency (MPF) can be examined using larger IEMG windows (6 s) where fatigue information can also be discerned by utilizing Fast Fourier Transform (Hwang et al., 2016). FFT is a method of data extraction where

the power density is examined, and fatigue level is assessed by quantifying the spectrum of frequency density during specific epochs of a muscle contraction. Subsequent quantification of the MPF within each time-window provides a consolidated data point for comparison during statistical analysis. Indices of fatigue can be demonstrated through observation of the frequency shift phenomenon (FSP). As muscles fatigue, the FSP describes a decrease in power density in the high-frequency range (>95 Hz) of EMG signals, and an increase in power density in the low-frequency range (15-45 Hz) (Cunningham et al., 2016; Kadefors, 1968; Kaiser & Petersen, 1962). The FSP can be observed throughout a contraction by quantifying changes in MPF during consecutive time-windows of IEMG data. Similarly, the FSP can be assessed by quantifying the ratio of high-frequency signals over the low frequency signals that exist withing consecutive IEMG windows. Therefore, the progression of fatigue can be monitored as the MPF value decreases throughout the time-windows of the MVIC.

As a result, using EMG analysis to examine the neuromuscular response to AIs following fatiguing exercise may identify changes in overall muscular activation and the progression of myoelectric fatigue.

Isometrics

Isometric strength testing is a widely used method for assessing muscle strength and function (Kim et al., 2014). It involves the static contraction of a muscle or group of muscles against a force gauge, such as a dynamometer. Through detailed analysis of isometric force production indices, AIs can be examined to further expose their impact on the physiological mechanisms which are mediated by fatigue.

Isometric strength testing largely evaluates linear force production during commonly encountered joint angels in exercise (Bartolomei et al., 2018; Hamada et al., 2000; Hwang et al.,

2016). With force being the primary index, researchers can evaluate aspects of force production beyond absolute force capacity by evaluating it across time. These measurements are factors such as the rate of force development (RFD). RFD is an important aspect of force production, particularly during explosive movements. It reflects how quickly force is generated in a muscle during the initial phase of contraction. A study by Bartolomei et al., (2018) took this principle and applied it to a study investigating the effect of ammonia inhalants on lower body power production. In the study, the researchers had participants perform an IMTP and evaluated the influence of AIs on force production during the test. By evaluating the various measures obtained by the force gauge, the group discovered that the use of AIs increased pRFD, but not total force production or mean RFD. The significant pRFD response suggested that ammonia inhalation may transiently influence explosive force production; however, the underlying neuromuscular mechanism was not directly measured.

Alternatively, isometrics can be used to examine the influence of muscular response to stimulus during fatigued contractions. A study by Heckathorne & Childress (1981) utilized concurrent isometric and EMG testing to examine muscular adaptations in the biceps brachii associated with prosthetic use in hand amputees. The authors concluded that observed increases in isometric force production during pre-fatigued contractions were due to increased recruitment of larger, fast type II muscle fibers with larger conduction velocities. As such, it is reasonable to expect that increases in EMG amplitude coupled with increases in MVIC force production are associated with increased in motor unit recruitment and firing rate during higher levels of force production (Hwang et al., 2016). While this conclusion was based on changes in EMG amplitude, it manifested as physical changes in force production during isometric contractions.

This conclusion provides greater power to the range of assumptions that can be made based on isometric analysis.

Although isometric strength testing does not directly measure isotonic strength capabilities, it is a safer alternative to maximal strength testing through dynamic exercises (Parai et al., 2016). While isotonic (dynamic) 1 repetition maximum (1RM) is the gold standard for strength testing, it is also associated with significant risk of injury (Tan et al., 2018). As such, research in isometric strength testing has developed to assess muscular strength through more controlled and safer methodologies.

Given that the force production capabilities of muscle are different between isometric, eccentric, and concentric muscle contractions, research investigating the relationship between isotonic 1RM and maximal isometric strength addresses this gap in knowledge (Kay et al., 2000). Utilizing regression analysis, several studies have been conducted demonstrating that isometric strength can be used as a predictor of 1RM strength for various muscle groups (Kanada et al., 2017; Parai et al., 2016; Petrović et al., 2020; Tan et al., 2018). Establishing a linear prediction equation for 1RM allows for the estimation of dynamic strength through assessment of maximal isometric strength. For example, study by Parai et al., (2016) developed a regression prediction equation for biceps 1RM based on isometric muscle strength. The prediction equation was found to have a significant moderate correlation ($r = 0.615$) between isometric strength and 1RM, with a low standard error ($SE = 1.12$ kg) suggesting it may be applicable to various populations. These findings allow researchers to make estimates of isotonic muscle strength from isometric strength data. Additionally, understanding isometric strength allows researchers and strength coaches to prescribe dynamic exercise load intensity without assuming the risks or time demands associated with maximal dynamic strength testing.

Implications

Concurrent isometric and sEMG testing provide an opportunity for detailed analysis of neuromuscular physiology and function. In the context of AI exposure during fatigued contractions, the use of this concurrent testing may provide more concrete insight into neuromuscular adaptations than either testing techniques individually. Concurrent isometric and sEMG testing provides an opportunity to examine both mechanical force production and global myoelectric responses during sustained contractions. In the context of AI exposure following fatiguing exercise, this approach may provide greater insight into whether changes in force production are accompanied by changes in muscular activation or the myoelectric manifestation of fatigue.

Utilizing detailed sEMG analysis alongside isometric force measurements may therefore help determine whether the performance effects previously reported by Secrest et al. (2015) and Rogers et al. (2022) are accompanied by measurable changes in neuromuscular function. Although conventional sEMG cannot directly identify individual motor unit recruitment, discharge behaviour, or specific muscle fibre activation, changes in amplitude and frequency-domain characteristics can provide complementary information regarding muscular activation and fatigue.

Alternatively, this analysis may infer AIs influence a greater recruitment of type II fast twitch muscle fibers when fatigue typically manifests as a greater recruitment of type I muscle fibers. Another possibility is that AIs influence central drive and make the effort associated with fatigued contractions easier to exert. While these scenarios are theoretically possible through increased sympathetic activation of muscle, the current body of literature investigating the effects of AIs has rested in speculation based on functional outcomes and propositions for more

detailed research (Bender & Popkin, 2023; Malecek & Tufano, 2021). Although EMG analysis may not be able to fully identify the mechanism with which they impact neuromuscular function, it may provide important information about which aspects of neuromuscular physiology are being affected either directly, or indirectly as a result of AI exposure.

Research Problem

Previous studies by Rogers et al., (2022) and Secrest et al. (2015) revealed AIs may improve anaerobic power output following and throughout repeated fatiguing exercise, respectively. These observations indicated that AIs may alter the neuromuscular response to fatigue to allow for increased force production of fatigued muscles. While these are valid speculations, more detailed research is needed to understand the neuromuscular response to AIs. Further evidence that AIs interact with neuromuscular physiology was identified by Bartolomei et al., (2018) where they observed increases in peak rate of force development during a maximal isometric contraction. The authors speculated that the observed increase in explosive force production, without increasing peak force production, may be a result of an increased rate of motor unit recruitment. Similarly, more detailed investigation is needed to support this theory.

Thus, the purpose of this study was to determine the effect ammonia inhalants on fatigued neuromuscular function. This was achieved by measuring the differences in force production, and sEMG signals following a fatigue protocol during a maximal isometric muscle contraction held for 30 s with and without AIs (Bartolomei et al., 2018; Rogers et al., 2022; Secrest et al., 2015). Specifically, this study assessed differences in maximal isometric strength of the biceps brachii, rate of muscle force production, muscular activation, and fatigue indices obtained from both force gauge data and sEMG data.

Maximal isometric strength was defined as the peak force production achieved during a maximal voluntary isometric contraction. Next, the rate of muscle force development was defined as the rate at which force production increases between the onset of a maximal voluntary isometric contraction and the peak force production during that contraction. Additionally, muscular activation was quantified in time domain analysis as a percentage of the amplitude in EMG signal compared to rested state maximal voluntary isometric contraction testing. Finally, fatigue was assessed using both force production and sEMG-derived indices. Changes in force production across the sustained isometric contraction was quantified using rate of force decline ($RFD_{Decline}$). $RFD_{Decline}$ was defined as the slope of a first-order linear regression fitted across the complete 30 s contraction, with more negative values representing a greater overall rate of force loss across the contraction. Additionally, fatigue was evaluated through frequency-domain analysis of the sEMG signal. Mean power frequency (MPF) was quantified across five consecutive 6 s windows throughout the 30 s isometric MVC to assess the progression of fatigue through the frequency shift phenomenon.

Guiding Questions

1. Is there a difference in responses from those who use AI and no AI in maximal isometric strength production during an isometric MVC from a fatigued state?
2. Is there a difference in responses from those who use AI and no AI in rate of muscle force development during an isometric MVC from a fatigued state?
3. Is there a difference in responses from those who use AI and no AI in normalized muscular activation during an isometric MVC from a fatigued state?
4. Is there a difference in responses from those who use AI and no AI in fatigue indices during an isometric MVC from a fatigued state?

Methods

Study Design

This study followed a randomized, counterbalanced crossover design. Participants attended two separate testing visits and completed the same experimental procedures during each visit. The treatment condition differed between visits, with each participant completing one ammonia inhalant condition and one control condition in a randomized order. Treatment order was randomized using an online research randomizer to minimize potential order effects associated with repeated-measures study designs (Urbaniak & Plous, 2024). Testing visits were conducted at approximately the same time of day to minimize potential diurnal variation in muscle activation and force production (Racinais et al., 2005).

The time between testing visits was 1-week. This washout period between trials was included for two reasons. First, the 1-week washout was determined based on the concept that most training programs for varsity athletes are structured so that similar workouts occur on the same days from week-to-week. By doing this, participants attended the second testing visit in a similar physical condition to their first testing visit based on their existing athletic training program. Second, the 1-week washout was determined to give participants sufficient rest to avoid fatigue from the first trial confounding the results of the second trial. Finally, literature suggests AIs potential effects only last approximately 30 s (Groop, 2019; Perry et al., 2016). This washout period provides ample time to avoid any potential carry-over effect resulting from AI exposure.

Participants

Participants were recruited in Thunder Bay, Ontario from varsity athletes. Participants were only recruited from teams whose training programs incorporated upper body resistance

training exercises. A sample of 14 male participants were recruited using purposive sampling. This sample size met the minimum sample required to achieve adequate power (n = 14) as calculated via a priori power analysis. The following parameters were used in this priori power analysis via GPower software: test = RM ANOVA, *p* value (α) = 0.05, effect size (η^2) = 0.0377, power of rejection (1-β) = 0.8, correlation value r = 0.50 (Ballmann et al., 2019; Faul et al., 2009; Rogers et al., 2022).

The study protocol was reviewed and approved by the Lakehead University Research Ethics Board (REB file no. 1470455). All participants provided written informed consent prior to participation.

Inclusion Criteria

Participants included in the study were 18 years old or older and a varsity athlete at Lakehead University at their time of completion of the study. Participants must have had a two-year minimum history of and current participation in upper limb resistance training programs determined by self-reporting. Participants were also able to attend two separate testing visits separated by 1 week.

Exclusion Criteria

Participants were excluded from the study if they had any history of cardiovascular, neurological, or upper limb disorders (Hwang et al., 2016). Next, participants experiencing any acute upper limb injuries affecting their ability to maximally contract their biceps muscle were also excluded. Additionally, participants were excluded if they had any history of neuromuscular disorders. Participants experiencing acute or chronic respiratory conditions, such as a cold or asthma, were excluded from the study to avoid the risk of adverse respiratory reactions to the ammonia inhalants. Finally, participants were screened for allergies to the aromatic compounds

included in the commercially available ammonia capsule used in this study. These inclusion and exclusion criteria were screened with a self-report tool before the study commenced for each participant that responded to the recruitment strategy. None of these criteria were present in any of the participants who completed this study.

Instrumentation

Isometric Force Production

Isometric force production was evaluated using a PUSHTON High Precision Load Cell. This load cell has a 300 kg capacity with a combined error of ±0.09 kg and is presented with a certificate of calibration. The calibration certificate is guaranteed accurate with A2LA and NVLAP accreditations. These accreditations ensure that calibration procedures are accurate and traceable through the National Institute of Standards and Technology, the gold standard for calibration.

Surface Electromyography

Three Delsys Trigno Avanti Wireless Biofeedback Sensor Systems served as the sEMG electrodes being used to record myoelectric signals throughout testing (Delsys Inc., 2024). The Delsys Trigno Avanti Wireless Biofeedback Sensor System is regarded as the gold standard for wireless sEMG devices in terms of reliability and validity (Bawa & Banitsas, 2022; Bayram & Kaykayoglu, 2022; Fuentes del Toro et al., 2019; Toledo-Peral et al., 2024). The Avanti sensors have a maximum sampling rate of 4370 Hz with a recording bandwidth of 10-850 Hz. The electrodes are 99.9% silver in a bipolar bar arrangement. The inter-electrode spacing measures out to be 10 mm with dual on-board stabilizing reference. Delsys Avanti electrodes utilize double-sided adhesive skin interfaces to ensure minimum sensor movement on the skin.

Procedures

Treatment Variables

This study used AI and control treatment variables. The first treatment variable was the AI. Each administration of this variable was done in capsule form which is activated upon cracking the vial in the capsule and releasing the ammonia vapour. Each capsule contained 0.3 ml ammonia inhalant (35% alcohol and 15% ammonia). To ensure each participant inhaled equal concentrations of the vapour, the cracked capsule was inserted into an opaque, air-tight container (an empty disposable water bottle, wrapped in black tape) and left for 5 minutes prior to testing. This approach allowed the ammonia vapour ample time to fully saturate the air in the container prior to the treatment delivery. The purpose of this was two-fold; first, this method is a common practice among athletes who use smelling salts, and second, it ensures equal inhalation of ammonia between participants. To administer the AI, the researcher removed the lid from the container and placed it approximately 10 cm from the nose of the participant. Participants were then instructed to inhale deeply through their nose for 3 s (Bartolomei et al., 2018; Groop, 2019; Rogers et al., 2022).

The control condition followed the same preparation and administration procedures as the ammonia inhalant condition; however, no ammonia inhalant capsule was placed in the container. The control treatment followed identical preparation and administration procedures except there was no AI capsule in the container during preparation or administration of the treatment variable. Although other studies have incorporated the use of a placebo control such as Vick's VapoRub, and Menthol oil, each of these alternatives interact with the respiratory system which may confound the results by stimulating a response from the respiratory system (Malecek & Tufano, 2021). Additionally, these placebo substances are reported to remain discriminant from AIs among participants, thus negating the purpose of a placebo substance. While the concept of a

placebo control treatment variable is desirable for repeated measures studies, the lack of an effective placebo control creates a limitation by enabling participants to determine which treatment variable they have received during testing.

Pre-Trial Procedures

Participants were asked to refrain from engaging in vigorous upper body exercise at least 24 hours before testing and to refrain from irregular consumption of caffeine, nicotine, and alcohol at least 12 hours of testing (Dumar et al., 2021; Rogers et al., 2022). Written and informed consent was obtained from each participant prior to data collection.

Experimental Procedures

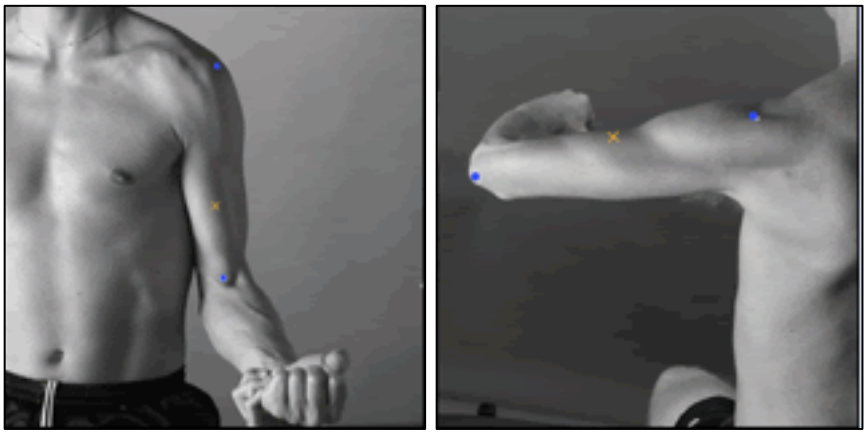
The experimental protocol of each testing visit was separated into five segments. The first segment being the *Warm-up Period*, then *sEMG Placement,* then *Baseline Testing Procedures*, then the *Fatigue Protocol*, and finally the *Fatigued Testing Procedures*. Each segment occurred in the order that they are described. The elbow joint angle for the entire experimental protocol was 90° of flexion to maximize force production and muscular cross-bridge formation (Squire, 1975). This was measured and verified with a plastic goniometer.

Electrode Placement. sEMG electrode placement was the next segment of procedures to occur on a testing visit. Prior to being equipped with an sEMG device, the skin on the anterior aspect of the participant's biceps was shaved and cleansed with an alcohol swab (Hwang et al., 2016). Participants were fitted with three bipolar sEMG electrodes over the biceps brachii, as the agonist, the triceps brachii, as the antagonist, and the ulnar styloid process, as the reference electrode (Ahmadi et al., 2021). Placement of the electrodes adhered to the guidelines of the *Surface EMG for non-invasive assessment of muscles (SENIAM)* (Hermens et al., 2000). The biceps electrode was fitted on the area with the greatest muscle bulk along the longitudinal

midline of the biceps muscle. The triceps electrode was placed on the lateral head of the triceps at 50 % on the line between the posterior crista of the acromion and the olecranon, with 3.5cm of lateral displacement to the line. Palpation of each respective anatomical landmark was conducted by hand, by the researcher to ensure accurate placement of the electrodes. See Figure 2 for a visual of the electrode placement locations and relevant anatomical landmarks.

Figure 2.

Electrode Placement Locations on the Biceps Brachii and Lateral Head of the Triceps Brachii



Note. The image on the left depicts the electrode placement for the biceps brachii, while the image on the right depicts the electrode placement for the triceps brachii. Blue dots indicate anatomical landmarks used to determine the electrode placement which is represented by the yellow markers (Hermens et al., 2000).

Placement of the biceps sEMG device was recorded by measuring the distance of the proximal aspect of the device to the acromion process in the anterior shoulder. Similarly, the placement of the triceps sEMG device was recorded by measuring the distance between the posterior crista of the acromion and the olecranon, with 3.5cm of lateral displacement at exactly halfway down the line. These measurements guided the placement of the sEMG device in the second testing visit to ensure consistent device placement. Additionally, a small temporary ink

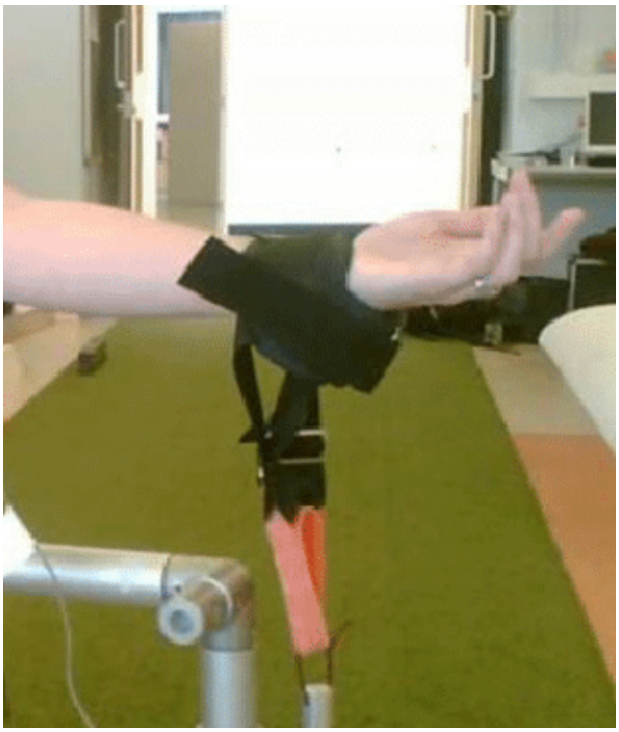
tattoo was used to mark the exact position of the sEMG electrode. The tattoo ink was selected based on a manufacturer statement that it will last 1-2 weeks before disappearing (Inkbox, 2015). The temporary tattoo was only placed on the subject during the first testing visit, after completing the testing protocol. Participants were instructed not to wash the area where the ink was applied for at least 1 hour after its application, as recommended by the manufacturer.

Warm-up Period. Prior to beginning the testing procedures, participants completed a 5-minute isometric warm-up. The warm-up consisted of three sets of short 5 s, submaximal elbow flexion at approximately 75% of maximal exertion, separated by 1 minute rest intervals (Hwang et al., 2016).

Baseline Testing Procedures. After participants were fitted with the sEMG device they began with baseline testing. This component of testing was used to evaluate their rested capacity to perform a maximal voluntary isometric contraction (MVIC) of the biceps. Participants were fitted with a wrist cuff which is attached to a PUSHTON High Precision Load Cell by a non-stretch strap material. The load cell was fixed to the floor directly below the participants' dominant wrist, using the same strap material, and adjusted to allow the participant to stand with their back to a wall with their dominant arm at their side in 90° of elbow flexion. During the contraction, participants were asked to maintain back, elbow, and shoulder contact with the wall to eliminate changes in elbow joint angle. The decision to connect the force gauge to the participant's wrist instead of their hand was made to avoid wrist flexors fatiguing earlier than elbow flexors during testing (See Figure 3).

Figure 3.

Example of the Wrist Strap that was Used During Testing



Note. This figure is an example of the wrist strap used in a study conducted by Qi et al., (2010) where biceps MVIC were examined.

To determine the participant's rested MVC, they completed one 30 s MVC. The decision to evaluate a 30 s contraction was made based on previous research indicating AI inhalation produces transient changes in the cardiorespiratory system for no longer than 30 s (Groop, 2019; Perry et al., 2016). During all MVC attempts, participants did not receive any form of encouragement. The results of this testing were used for three reasons. First, the baseline MVC peak force output results were used to develop the load required during the fatigue protocol which was necessary for the next segment of testing. Additionally, the baseline testing results were used as additional data to compare the fatigued testing results against. Finally, *Baseline Testing Procedures* sEMG data were used for normalization of *Fatigued Testing Procedures* data. This was done to account for minor variations that may occur in sEMG electrode placement and individual variation between visits.

Fatigue Protocol. A standardized fatigue protocol was used to ensure participants were equally fatigued for both testing visits. After completing the 30 s baseline MVC, an estimate of the participants isotonic 1RM was calculated using a linear regression equation. Developed by Parai et al., (2016), the regression equation functions to estimate an individual's dominant arm 1RM isotonic bicep curl based on an isometric MVC. The regression equation is as follows,

$$Y = 0.391X + 1.472 \quad (1)$$

where Y = 1RM in kilograms, X = peak isometric force in kilograms.

Following the estimation of the individual's 1RM, a 10RM load was estimated at 75% of the individual's 1RM. The calculated 10RM load was used throughout the fatigue protocol. Due to limitations in precise dumbbell weights, the 10RM load was rounded to the nearest 5 pounds increment of standard exercise dumbbells.

The fatigue protocol consisted of 4 sets of biceps curls at the 10RM load to failure with each set. For example, repetitions of 10, 8, 6, and 5 may be achieved throughout the 4 sets respectively. Participants competed each set with their back against a wall to minimize force contributions from other muscle groups. Participants were also asked to complete repetitions following a 3 s eccentric, and 2 s concentric cadence guided by a metronome (Robbins et al., 2010). Each set was separated by 3 minutes of rest between sets. Failure of a set was defined as the inability of a participant to match the pace of the metronome during a set for 2 consecutive repetitions. All repetitions were supervised by the principal investigator to ensure proper technique was used by participants.

The 10RM load has been determined to evoke similar central fatigue responses compared to a heavier 5RM load, however it may result in greater peripheral fatigue compared to the heavier 5RM load (Robbins et al., 2010). The descending volume of repetitions over 4 sets has been observed to evoke significant peripheral and central fatigue responses in participants completing traditional biceps curls. This outcome was determined based on observed decreases in isometric MVC force production, and inferred motor unit activation, following the fatigue protocol.

Fatigued Testing Procedures. To capture the greatest neural deficits, fatigued testing procedures began within 30 s of completing the fatigue protocol (Behm et al., 2002; Robbins et al., 2010). After completing the fatigue protocol participants immediately began the fatigued testing procedures. The fatigued testing procedures used identical equipment and positioning to the *Baseline Testing Procedures*. After participants were in the correct position and sEMG and force data collection devices were prepared, the participants were given one of the treatment variables. Upon receiving the treatment variable, participants immediately began a 30 s isometric MVC of their dominant arm biceps.

Data Collection

Force data recorded in kilograms and was collected using a PUSHTON High Precision Load Cell at a sampling rate of 2000 Hz (Hwang et al., 2016). sEMG data were collected using Delsys Trigno Avanti sensors at a sampling rate of 2000 Hz. Both force data and sEMG data were collected concurrently and sent to a host PC using LabChart Pro.

Data Analysis

Baseline Testing Procedures Data

Data collected during the *Baseline Testing Procedures* was recorded and analyzed on LabChart Pro to identify each participant's maximal isometric strength. This value was then transferred to an excel file accordingly where the individual's estimated 1RM load was calculated with the same following equation,

$$Y = 0.391X + 1.472 \quad (2)$$

where Y = 1RM load in kilograms, and X = maximal isometric force in kilograms. This equation was also modified to concurrently estimate the individual's 10RM load.

$$Y = \frac{0.391X + 1.472}{0.75} \quad (3)$$

where Y = 10RM load in kilograms, and X = maximal isometric force in kilograms. The estimated 10RM load was then rounded to the nearest 5-pound increment to match the available dumbbell weights for use in the *Fatigue Protocol*.

Isometric Force Quantification

Force data were recorded as the voltage output of the PUSHTON High Precision Load Cell. Prior to each experimental trial, the load cell was calibrated using a series of known applied loads, with the corresponding voltage output recorded for each calibration load. A total of 28 calibration procedures were completed across experimental trials. Individual calibration procedures demonstrated a highly linear relationship between voltage output and applied load (mean R^2 = .99987; range = .99963–.999997).

To provide a standardized conversion of voltage to force across participants and experimental conditions, calibration data collected across trials were used to derive a common linear calibration function. The common calibration slope was 668.462 lb/V, while the mean calibration intercept was −84.830 lb. Accordingly, recorded voltage was converted to pounds using:

$$F_{lb} = 668.462V - 84.830 \quad (4)$$

where F_{lb} represents the calibrated load in pounds and V represents the recorded load-cell voltage. Pounds were subsequently converted to kilograms using a conversion factor of 0.45359237 kg/lb. Mass-equivalent values were then converted to force in newtons according to:

$$F_N = m \times g \quad (5)$$

where F_N represents force in newtons, m represents mass in kilograms, and g represents gravitational acceleration (9.80665 $m \cdot s^{-2}$)(Rodgers & Cavanagh, 1984). Combining these conversions resulted in the standardized equation:

$$F_N = 2973.456V - 377.343 \quad (6)$$

This standardized calibration equation was applied uniformly to force measurements prior to statistical analysis.

Rate of Force Development. Temporal characteristics of force production were quantified using two variables: rate of force development to peak force production (RTP) and time to maximal force (T2M). RTP represented the rate of increase in force from contraction onset to maximal force production. Following identification of contraction onset and maximal force, force data spanning this interval were fitted using a first-order linear regression, with the slope of the fitted line representing RTP. Regression slopes calculated relative to sample number were converted to force per second using the 2000-Hz sampling frequency and subsequently converted to N/s using the standardized load-cell calibration.

T2M was quantified as the elapsed time between the identified onset of the contraction and maximal force production. The sample index corresponding to maximal force was divided by the 2000-Hz sampling frequency to express T2M in seconds.

Rate of Force Decline. $RFD_{Decline}$ was used as a force-based measure of the overall force trajectory during the sustained isometric contraction. A first-order linear regression was fitted to the force signal across the complete 30 s contraction, beginning at contraction onset and continuing through the end of the contraction. The slope of the fitted regression line represented $RFD_{Decline}$. Negative values indicated an overall decline in force production across the contraction, with more negative values representing a greater rate of force loss over time. Because the regression incorporated the complete contraction, including the initial rise in force toward maximal force production, $RFD_{Decline}$ represents the overall force-time trend across the sustained contraction rather than an isolated measure of force decline following peak force. Regression slopes calculated relative to sample number were converted to force per second using the 2000-Hz sampling frequency and subsequently converted to N/s using the standardized load-cell calibration.

Electromyography Analysis

Surface EMG data collected during the *Baseline Testing Procedures* and the *Fatigued Testing Procedures* were processed using MATLAB R2023b scripts (The MathWorks Inc., 2023). Raw sEMG signals were recorded at a sampling frequency of 2000 Hz. Separate processing procedures were used for the time- and frequency-domain analyses.

Time Domain Analysis. For the time-domain analysis, the mean value of each raw sEMG signal was first removed to correct for signal offset. The signal was then scaled by a factor of 1000, full-wave rectified, and processed using a fourth-order low-pass Butterworth filter with a 100-Hz cut-off frequency. Zero-phase digital filtering was applied to avoid introducing a temporal phase shift into the processed signal. Biceps sEMG amplitude associated with maximal force production was quantified within a 500-ms window centred around the sample corresponding to maximal force. The mean of the processed sEMG signal within this interval was used to represent biceps sEMG amplitude. For comparison between fatigue states, fatigued sEMG amplitude was additionally normalized to the corresponding baseline value for each treatment condition. (Ahamed et al., 2015; Hwang et al., 2016; Rantalainen et al., 2012)

Frequency Domain Analysis. Frequency-domain analysis was performed separately for five consecutive 6-s windows spanning the 30-s sustained isometric contraction. Each window contained 12,000 samples based on a sampling frequency of 2,000 Hz. Before transformation, a 12,000-point Hamming window was applied to each segment to reduce spectral leakage. A 12,000-point fast Fourier transform (FFT), without zero-padding, was then used to generate a single-sided spectrum spanning 0 to 1,000 Hz, with a frequency resolution of approximately 0.167 Hz. Mean frequency was calculated independently for each window as the amplitude-weighted average of the frequency bins:

$$\mathrm{MPF} = \frac{\sum_{i=1}^{n} \mathrm{f}_i \, \mathrm{P}_i}{\sum_{i}^{n} \mathrm{P}_i} \tag{7}$$

where f_i represents the frequency associated with spectral bin i, P_i represents the corresponding spectral power, and n represents the total number of frequency bins included in the calculation. MPF was calculated independently for each of the five windows. The frequency shift phenomenon (FSP) refers to the fatigue-related redistribution of the sEMG power spectrum toward lower frequencies during sustained muscle activation. Accordingly, progressive reductions in MPF across the consecutive windows were used to characterize the myoelectric manifestation of fatigue during the sustained contraction (Arendt-Nielsen & Mills, 1985; Cunningham et al., 2016; De Luca, 1984; Kadefors, 1968; Kaiser & Petersen, 1962; Petrofsky & Lind, 1980).

Statistical Analysis

Descriptive statistics were conducted to provide means and standard deviations for all dependent variables within the respective treatment conditions. Inferential statistics were completed to address each of the research questions at $\alpha = .05$ for all dependent variables of interest. Data recorded during the Baseline Testing Procedures and Fatigued Testing Procedures were contrasted to identify differences between fatigue states (baseline and fatigued) and treatment conditions (AI and control). Where applicable, data were additionally normalized to the corresponding baseline value for comparison between treatment conditions. Force analyses included all 14 participants. Complete biceps sEMG data were available for 13 participants following the exclusion of one invalid ammonia inhalant fatigued biceps sEMG recording.

To evaluate differences in force-derived variables and time-domain sEMG amplitude, separate 2 × 2 repeated-measures analyses of variance (rmANOVA) were conducted for maximal isometric force production, RTP, T2M, $RFD_{Decline}$, and biceps sEMG amplitude. Treatment consisted of two levels (AI and control), and fatigue state consisted of two levels (*Baseline Testing Procedures* and *Fatigued Testing Procedures*). The treatment × fatigue state interaction was examined first for each dependent variable. If the interaction was not statistically significant, the main effects of treatment and fatigue state were subsequently examined. If a significant interaction was identified, simple-effects analyses were conducted to characterize differences between AI and control conditions at each fatigue state and differences between fatigue states within each treatment condition.

To further examine the magnitude of fatigue-related changes in maximal isometric force production, percentage change from baseline to the fatigued state was calculated separately for the AI and control conditions. Percentage-change values were compared between treatment conditions using a paired-samples *t*-test, with Cohen's *d* and the 95% confidence interval of the mean difference reported as measures of effect magnitude and precision. For biceps sEMG amplitude, fatigued values were additionally normalized to the corresponding baseline value within each treatment condition and compared between conditions.

To evaluate differences in the frequency-domain sEMG response during the fatigued contraction, MPF was assessed using a 2 × 5 rmANOVA. Treatment consisted of two levels (AI and control), while time consisted of five consecutive 6 s windows spanning the 30 s sustained isometric contraction. The treatment × time interaction was examined first to determine whether the temporal change in MPF differed between treatment conditions. If the interaction was not statistically significant, the main effects of treatment and time were subsequently examined.

When a significant main effect of time was identified, pairwise comparisons using the least significant difference (LSD) method were conducted to characterize differences in MPF between time windows. Mauchly's test was used to assess the assumption of sphericity, and Greenhouse–Geisser-adjusted results were reported when the assumption of sphericity was violated.

Effect sizes for rmANOVA analyses were reported using partial eta squared (ηp^2), consistent with institutional reporting conventions. All statistical analyses were conducted using IBM SPSS Statistics.

Results

This study aimed to investigate the effect ammonia inhalants on fatigued neuromuscular function in varsity athletes by evaluating differences in force production and sEMG signals through an isometric muscle contraction following a fatigue protocol. A total of 14 male participants aged 19-25 years old (M = 23.57, SD = 1.79) with a mean body mass index of 27.43 (SD = 2.47) completed the study.

Following randomization of treatment order, participants completed both experimental conditions. Descriptive statistics for the fatigue protocol are presented in Table 1.

Table 1.

Fatigue Protocol Descriptive Statistics

Trial	Mean Prescribed Fatigue-Protocol Load (kg)	Number of Repetitions of Biceps Curls									
		Set 1		Set 2		Set 3		Set 4		Total	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	15.88	9.14	1.70	6.71	1.44	5.43	1.09	5.00	0.96	26.29	3.87
2	15.88	8.86	2.07	6.71	1.27	5.57	0.85	4.86	1.03	26.00	4.13

Note. Values represent descriptive statistics for the fatigue protocol across the two experimental visits. Load is presented in kilograms (kg); SD = standard deviation.

Question #1: Is there a difference in responses from those who use AI and no AI in maximal isometric strength production during an isometric MVC from a fatigued state?

The treatment × fatigue state interaction was not significant, $F_{(1, 13)} = 0.019$, $p = .894$, $\eta_p^2 = .001$. There was no significant main effect of treatment, $F_{(1, 13)} = 0.562$, $p = .467$, $\eta_p^2 = .041$. However, a significant main effect of fatigue state was observed, $F(1, 13) = 71.59$, $p < .001$, $\eta_p^2 = .846$, indicating that maximal isometric force was lower in the fatigued condition than at baseline, regardless of treatment condition. Mean force values were lower in the fatigued condition than at baseline for both the control and ammonia inhalant trials (Table 2).

Table 2.

Maximal Isometric Force at Baseline and Following the Fatigue Protocol

Condition	Testing Stage	
	Baseline (*N*)	Fatigued (*N*)
Control (No AI)	349.24 ± 63.63	277.56 ± 61.01
Ammonia Inhalant	340.69 ± 68.49	271.06 ± 54.89

Note. Values are presented as mean ± standard deviation where *N* represents units in Newtons.

Mean force decreased by 20.29 ± 11.08% in the control condition and 20.09 ± 8.43% in the ammonia inhalant condition. A paired-samples *t*-test indicated no significant difference in percentage change between treatment conditions, $t_{(13)} = -0.058$, $p = .954$, Cohen's $d = -0.016$, 95% CI [−7.96, 7.54].

Question #2 Is there a difference between AI and no AI in rate of muscle force development during an isometric MVC from a fatigued state?

Descriptive values for RTP and T2M across treatment conditions and fatigue states are presented in Table 3.

Table 3.

Rate to Peak Force and Time to Maximal Force at Baseline and Following the Fatigue Protocol

Variable	Control		Ammonia	
	Baseline	Fatigued	Baseline	Fatigued
RTP (N/s)	156.78 ± 178.67	109.41 ± 93.11	94.50 ± 139.64	49.63 ± 64.93
T2M (s)	2.86 ± 4.43	3.50 ± 5.17	2.82 ± 2.63	4.21 ± 2.67

Note. Values are presented as mean ± standard deviation. RTP = rate of force development to peak force production; T2M = time to maximal force.

With respect to RTP, the treatment × fatigue state interaction was not significant, $F_{(1, 13)} = 0.002$, $p = .966$, $\eta_p^2 < .001$. There was also no significant main effect of treatment, $F_{(1, 13)} = 2.671$, $p = .126$, $\eta_p^2 = .170$, or fatigue state, $F_{(1, 13)} = 3.262$, $p = .094$, $\eta_p^2 = .201$. Therefore, RTP did not significantly differ as a function of treatment or fatigue state.

Regarding T2M, the treatment × fatigue state interaction was not significant, $F_{(1, 13)} = 0.099$, $p = .757$, $\eta_p^2 = .008$. There was also no significant main effect of treatment, $F_{(1, 13)} = 0.151$, $p = .704$, $\eta_p^2 = .011$, or fatigue state, $F_{(1, 13)} = 0.980$, $p = .340$, $\eta_p^2 = .070$. Therefore, T2M did not significantly differ as a function of treatment or fatigue state.

Question #3 Is there a difference between AI and no AI in muscular activation during an isometric MVC from a fatigued state?

Complete biceps sEMG data were available for 13 participants, as one participant was excluded from the sEMG analyses due to invalid biceps sEMG data during the ammonia inhalant fatigued trial. Descriptive values for biceps sEMG amplitude across treatment conditions and fatigue states are presented in Table 4.

Table 4.

Biceps sEMG Amplitude at Baseline and Following the Fatigue Protocol

Condition	Testing Stage	
	Baseline (mV)	Fatigued (mV)
Control (No AI)	545.69 ± 361.32	479.19 ± 276.11
Ammonia Inhalant	713.42 ± 326.67	581.30 ± 343.73

Note. Values are presented as mean ± standard deviation. sEMG = surface electromyography. mV= millivolts.

The treatment × fatigue state interaction for biceps sEMG amplitude was not significant, $F_{(1, 12)} = 0.435$, $p = .522$, $\eta_p^2 = .035$. There was also no significant main effect of treatment, $F_{(1, 12)} = 3.473$, $p = .087$, $\eta_p^2 = .224$, or fatigue state, $F_{(1, 12)} = 2.492$, $p = .140$, $\eta_p^2 = .172$. Therefore, biceps sEMG amplitude did not significantly differ as a function of treatment or fatigue state.

To further examine muscular activation following the fatigue protocol, fatigued biceps sEMG amplitude was normalized to the corresponding baseline value for each treatment condition. Normalized biceps sEMG amplitude was 0.940 ± 0.311 in the control condition and 0.861 ± 0.336 in the ammonia inhalant condition, corresponding to approximately 94.0% and 86.1% of baseline activation, respectively. There was no significant difference in normalized biceps sEMG amplitude between treatment conditions, $F_{(1, 12)} = 0.845$, $p = .376$, $\eta_p^2 = .066$.

Question #4: Is there a difference between AI and no AI in fatigue indices during an isometric MVC from a fatigued state?

Fatigue during the sustained isometric contraction was assessed using both force- and sEMG-derived measures. $RFD_{Decline}$ was used to quantify the change in force production across the sustained contraction, while biceps brachii MPF was used to assess the temporal progression

of neuromuscular fatigue across five consecutive 6 s time windows. Descriptive values for RFDDecline across treatment conditions and fatigue states are presented in Table 5.

Table 5.

Rate of Force Decline at Baseline and Following the Fatigue Protocol

Condition	Testing Stage	
	Baseline (N/s)	**Fatigued (N/s)**
Control (No AI)	−3.03 ± 2.27	−3.02 ± 1.38
Ammonia Inhalant	−3.41 ± 2.80	−2.52 ± 1.63

Note. Values are presented as mean ± standard deviation. $RFD_{Decline}$ = rate of force decline, calculated as the linear force-time slope across the complete 30 s contraction; more negative values indicate a greater overall rate of force loss.

For $RFD_{Decline}$, the treatment × fatigue state interaction was not significant, $F_{(1, 13)} = 1.935$, $p = .188$, $\eta_p^2 = .130$. There was also no significant main effect of treatment, $F_{(1, 13)} = 0.082$, $p = .779$, $\eta_p^2 = .006$, or fatigue state, $F_{(1, 13)} = 0.657$, $p = .432$, $\eta_p^2 = .048$. Therefore, $RFD_{Decline}$ did not significantly differ as a function of treatment or fatigue state.

To assess the myoelectric manifestation of fatigue, MPF was examined across five consecutive 6 s time windows during the fatigued contraction. Complete MPF data were available for 13 participants.

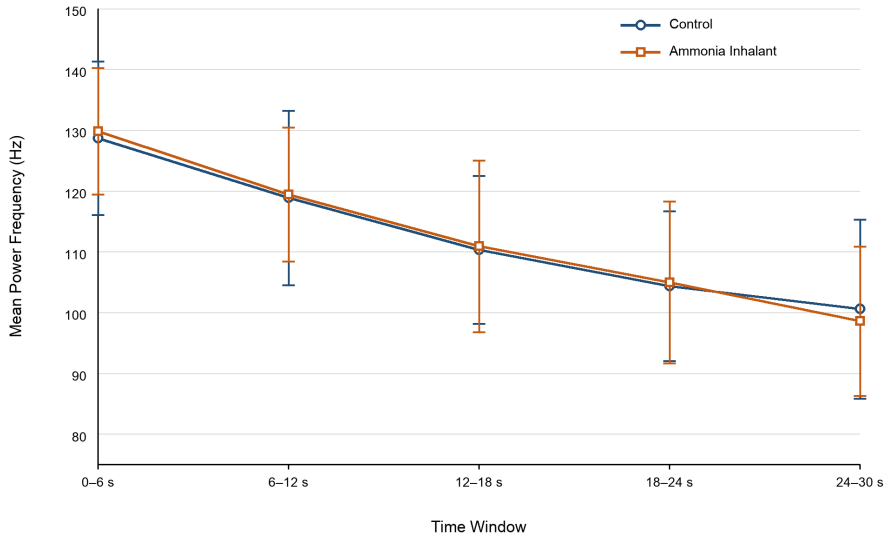
A two-way repeated-measures ANOVA was conducted to examine the effects of treatment condition and time on MPF. The treatment × time interaction was not significant, $F_{(4, 48)} = 1.093$, $p = .371$, $\eta_p^2 = .083$. There was also no significant main effect of treatment condition, $F_{(1, 12)} = 0.004$, $p = .954$, $\eta_p^2 < .001$. However, there was a significant main effect of time.

Mauchly's test indicated that the assumption of sphericity was violated for time, $W = .040$, $p < .001$; therefore, Greenhouse–Geisser-corrected results were reported. MPF significantly differed across the five time windows, $F_{(1.785, 21.417)} = 150.303$, $p < .001$, $\eta_p^2 = .926$.

When collapsed across treatment conditions, mean MPF progressively decreased from 129.25 Hz during the first 6 s window to 119.16 Hz, 110.62 Hz, 104.65 Hz, and 99.58 Hz during the subsequent windows, respectively. Pairwise comparisons indicated that MPF significantly decreased between each consecutive time window. Mean MPF decreased by 10.09 Hz from 0–6 s to 6–12 s (95% CI [7.56, 12.63], $p < .001$), by 8.54 Hz from 6–12 s to 12–18 s (95% CI [6.79, 10.29], $p < .001$), by 5.98 Hz from 12–18 s to 18–24 s (95% CI [4.36, 7.60], $p < .001$), and by 5.07 Hz from 18–24 s to 24–30 s (95% CI [3.33, 6.81], $p < .001$). The temporal changes in MPF across treatment conditions are illustrated in Figure 4.

Figure 4.

Biceps Brachii Mean Power Frequency Across the 30-Second Fatigued Isometric Contraction



Note. Values are presented as mean ± SD. MPF = mean power frequency.

Discussion

The purpose of this study was to investigate the effects of ammonia inhalants on neuromuscular function following fatiguing exercise. Specifically, differences in maximal isometric force production, rate of force development, muscular activation, and indices of fatigue were used to approximate neuromuscular fatigue and evaluated during sustained maximal isometric contractions with and without ammonia inhalant use. The primary findings demonstrated that ammonia inhalation did not significantly influence any of the measured neuromuscular outcomes. Conversely and as expected, the fatigue protocol resulted in a significant reduction in maximal isometric force production, while MPF progressively decreased throughout the 30 s fatigued contraction. Together, these findings demonstrate that fatigue was evident within both the mechanical and myoelectric measures examined in the present study, but that ammonia inhalation did not significantly alter these responses. The results will be discussed within the four research question areas below.

Maximal Isometric Force Production

Maximal isometric force production was significantly reduced following the fatigue protocol, as demonstrated by a significant main effect of fatigue state. Descriptively, force decreased by approximately 20% in both treatment conditions. However, ammonia inhalation did not significantly influence maximal force production, nor was there a significant interaction between treatment and fatigue state. Furthermore, the magnitude of the fatigue-related reduction was nearly identical between conditions, with force decreasing by 20.29% in the control condition and 20.09% in the ammonia inhalant condition. These findings suggest that although the fatigue protocol was successful in reducing maximal force-producing capacity, ammonia inhalation did not alter or attenuate this reduction.

The absence of an effect of ammonia inhalation on maximal force production is consistent with much of the existing literature. Perry et al. (2016) examined maximal isometric force production during an isometric mid-thigh pull following ammonia inhalation and similarly observed no significant improvement in force production. Campbell et al. (2022) also reported no improvement in maximal isometric strength following ammonia inhalation during knee-extension and handgrip MVICs. Similarly, Vigil et al. (2018) found that ammonia inhalation did not improve maximal deadlift performance, while Richmond et al. (2014) reported no improvement in repetitions performed during high-intensity resistance exercise. Collectively, these studies and the findings of the present investigation provide consistent evidence that the acute stimulatory effects associated with ammonia inhalation do not necessarily translate to improvements in maximal force production.

The current study extends these findings by evaluating maximal isometric force production following a standardized fatigue protocol. This distinction is particularly relevant because ammonia inhalants are frequently used by athletes under conditions where fatigue has already developed. Pritchard et al. (2014), for example, reported that ammonia inhalant use among powerlifters was most common during the deadlift, which occurs as the final event of a powerlifting competition, with athletes describing ammonia inhalants as helping them feel more alert when mentally and physically fatigued. Furthermore, Secrest et al. (2015) reported greater peak and mean Wingate power following ammonia inhalation after a simulated American football protocol, while Rogers et al. (2022) observed greater mean and peak power across repeated Wingate trials. These findings have contributed to the suggestion that ammonia inhalants may be more effective when exercise is performed under fatigued or repeated high-intensity conditions.

The findings of the present study do not support this proposed benefit in maximal isometric force production. Despite participants demonstrating reduction in force following the fatigue protocol, ammonia inhalation did not preserve maximal force capacity compared with the control condition. Importantly, this does not necessarily contradict the significant findings reported by Secrest et al. (2015) and Rogers et al. (2022), as both studies evaluated dynamic anaerobic cycling performance rather than maximal isometric force production. The physiological and mechanical requirements of repeated Wingate exercise differ substantially from those of a sustained maximal isometric contraction (Kay et al., 2000). Therefore, the ergogenic effects reported during repeated dynamic exercise may not translate to maximal isometric force production. This interpretation is also supported by Rogers et al. (2022), who observed increases in mean and peak power but did not identify a significant effect of ammonia inhalation on fatigue index.

One potential explanation is that the perceived acute arousal response associated with ammonia inhalation may be insufficient to overcome the physiological limitations to force production imposed by muscular fatigue. Previous studies have demonstrated increases in heart rate, cerebral blood flow velocity, alertness, and perceived performance following ammonia inhalation despite observing no corresponding improvement in maximal neuromuscular performance (Campbell et al., 2022; Perry et al., 2016).

Accordingly, the perceptual and autonomic response to ammonia inhalation should not necessarily be interpreted as an increase in the force-producing capacity of fatigued skeletal muscle. Under the conditions of the present study, ammonia inhalation may have produced the characteristic acute stimulatory response described in previous research, but this response was not accompanied by a measurable preservation or enhancement of maximal isometric force.

Rate of Force Development

Rate of muscle force production was further examined using RTP and T2M. Ammonia inhalation did not significantly influence either variable, and neither measure demonstrated a significant treatment × fatigue state interaction. These findings suggest that ammonia inhalation did not alter the temporal characteristics of force production during the sustained isometric contractions examined in the present study. Although descriptive RTP values were lower following the fatigue protocol in both treatment conditions, there was no significant main effect of fatigue state. Similarly, T2M did not significantly differ between treatment conditions or fatigue states.

The absence of an effect of ammonia inhalation on RTP is generally consistent with previous research examining RFD following ammonia inhalation. Perry et al. (2016) reported no significant effect of ammonia inhalation on RFD during maximal isometric mid-thigh pulls performed immediately and at 15-, 30-, and 60 s intervals following inhalation. Similarly, Campbell et al. (2022) observed increased alertness and perceived performance following ammonia inhalation without corresponding changes in RFD during maximal voluntary contractions. These findings suggest that the acute stimulatory response associated with ammonia inhalation does not consistently translate to an increased rate of force production.

However, the findings of the present study differ from those reported by Bartolomei et al. (2018), who observed a significant increase in peak rate of force development following ammonia inhalation during an isometric mid-thigh pull. Importantly, differences in how rate of force development was quantified should be considered when comparing these findings. Bartolomei et al. (2018) assessed peak RFD over a 20-ms interval, whereas RTP in the present study represented the slope of the force-time relationship from contraction onset to maximal

force production. Peak RFD in this therefore represents the steepest short-duration increase in force, while RTP provides a measure of force development across the complete rising phase of the contraction. These variables describe related, but distinct, characteristics of rapid force production and should not be interpreted interchangeably.

This methodological difference may partially explain why the present study did not reproduce the significant effect observed by Bartolomei et al. (2018). RFD measured during the early phase of an explosive contraction is particularly influenced by rapid neural activation, including motor unit discharge behaviour, whereas later portions of the force-time curve are increasingly influenced by maximal force capacity and muscular characteristics. Additionally, peak RFD is calculated from a relatively short and steep region of the force-time curve and may therefore detect transient changes that are less apparent when force development is averaged across the complete interval from contraction onset to peak force. Maffiuletti et al., (2016) emphasize that different RFD calculations can reflect different physiological characteristics and that peak RFD is particularly dependent on the epoch used for its calculation.

The inclusion of T2M, as outlined in this study, provides an additional measure with which to evaluate whether ammonia inhalation altered the temporal development of maximal force. No significant treatment × fatigue state interaction or main effects of treatment or fatigue state were observed for T2M. When considered alongside the RTP findings, this suggests that ammonia inhalation neither increased the overall rate at which force developed nor meaningfully changed the time required to achieve maximal force under the conditions of the present study. Although Bartolomei et al. (2018) demonstrated that ammonia inhalation may influence a brief period of peak explosive force development, the present findings do not provide evidence that

this effect extends across the complete force-development phase of a sustained maximal isometric contraction.

Muscular Activation

Biceps muscular activation was evaluated using sEMG amplitude within a 500-ms window centered around maximal force production. Ammonia inhalation did not significantly influence biceps sEMG amplitude, and there was no significant treatment × fatigue state interaction. Similarly, when fatigued sEMG amplitude was normalized to the corresponding baseline value, no significant difference was observed between treatment conditions. These findings indicate that ammonia inhalation did not produce a detectable change in biceps sEMG amplitude surrounding maximal force production under the conditions of the present study.

The absence of a treatment effect is consistent with previous research examining the neuromuscular response to ammonia inhalation. Perry et al. (2016) assessed EMG activity concurrently with maximal force and RFD during isometric mid-thigh pulls and reported no significant differences in EMG activity between ammonia inhalant and control conditions. Their findings are particularly relevant because increases in heart rate and middle cerebral artery blood flow velocity were observed following ammonia inhalation despite the absence of corresponding changes in EMG activity, maximal force, or RFD. Therefore, an acute physiological response to ammonia inhalation does not necessarily appear to produce a measurable increase in muscular activation or force production.

The findings of the present study are also consistent with those of Campbell et al. (2022), who examined several indices of neuromuscular function following ammonia inhalation. Although ammonia inhalation significantly increased heart rate, alertness, and perceived performance, measures of functional and neuromuscular performance were not improved. The

authors concluded that the increase in arousal associated with ammonia inhalation was not accompanied by an increase in neuromuscular performance. When considered alongside the present findings, this further supports a distinction between the perceptual or autonomic response produced by ammonia inhalation and measurable changes in neuromuscular function.

This distinction is particularly relevant to the proposed mechanism underlying the ergogenic effects of ammonia inhalants. Previous research demonstrating increased pRFD following ammonia inhalation has contributed to speculation that ammonia inhalants may influence neural activation during explosive force production. However, the present study found neither an increase in biceps sEMG amplitude nor a corresponding improvement in maximal force production or RTP. Collectively, these findings do not provide evidence that ammonia inhalation enhanced biceps muscular activation sufficiently to improve force production following the fatigue protocol. This interpretation should remain limited to the sEMG measures used in the present study, as conventional surface EMG amplitude does not provide a direct measurement of individual motor unit recruitment or discharge behaviour (Vigotsky et al., 2018).

The lack of a significant fatigue-state effect on biceps sEMG amplitude should also be interpreted independently from the clear reduction in maximal force observed following the fatigue protocol. Although maximal force production decreased by approximately 20%, there was no significant main effect of fatigue state on sEMG amplitude measured around maximal force. The relationship between surface EMG amplitude and force is influenced by several physiological and methodological factors and may change during fatigue; therefore, proportional changes in force and sEMG amplitude should not necessarily be expected (Vigotsky et al., 2018). In the present study, the combination of reduced maximal force with no significant

change in biceps sEMG amplitude suggests that the fatigue-related reduction in force cannot be explained solely by a corresponding reduction in the measured sEMG amplitude.

Fatigue Indices

Fatigue was evaluated using both force- and sEMG-derived measures to determine whether ammonia inhalation altered the progression of fatigue during the sustained isometric contraction. $RFD_{Decline}$ was used to quantify the overall force-time slope across the contraction, while MPF was used to evaluate the myoelectric manifestation of fatigue across five consecutive 6-s time windows. Ammonia inhalation did not significantly influence $RFD_{Decline}$, and no significant treatment × fatigue state interaction was observed. Conversely, MPF demonstrated a significant and progressive reduction across the five time windows; however, there was no significant main effect of treatment or treatment × time interaction. Collectively, these findings demonstrate the development of fatigue throughout the sustained contraction but provide no evidence that ammonia inhalation altered its progression.

The progressive reduction in MPF observed in the present study is characteristic of the frequency shift phenomenon (FSP), an established myoelectric manifestation of fatigue during sustained muscular contractions. The FSP describes a redistribution of the sEMG power spectrum toward lower frequencies as fatigue develops, resulting in progressive reductions in frequency-domain measures such as MPF (De Luca, 1984; Kadefors, 1968; Kaiser & Petersen, 1962; Petrofsky & Lind, 1980). Similar reductions in the frequency characteristics of the sEMG signal have been demonstrated within the biceps brachii during sustained contractions (Esposito et al., 1998). In the present study, mean MPF decreased progressively from approximately 129 Hz during the first 6-s window to approximately 100 Hz during the final window, with significant reductions occurring between each consecutive time window. The magnitude and

consistency of this response provides clear evidence of an FSP throughout the sustained contraction and supports the presence of progressive myoelectric fatigue during the 30-s MVIC. Importantly, this progression occurred following the dynamic fatigue protocol, indicating that fatigue continued to develop during the subsequent sustained isometric contraction rather than representing only the residual effects of the preceding exercise.

The physiological mechanisms contributing to the FSP are multifactorial. Reductions in MPF during sustained contractions have been strongly associated with reductions in muscle fibre conduction velocity. Arendt-Nielsen and Mills (1985) demonstrated a relationship between mean power frequency of the EMG spectrum and muscle fibre conduction velocity, supporting reduced conduction velocity as an important contributor to the redistribution of spectral power toward lower frequencies during muscular fatigue. However, the FSP should not be attributed to a single physiological mechanism. Intramuscular and surface EMG characteristics are influenced by several changes occurring within the neuromuscular system as fatigue develops, including changes in motor unit activity (Moritani et al., 1986). Therefore, the progressive reduction in MPF observed in the present study is most appropriately interpreted as evidence of the FSP and the associated myoelectric manifestation of fatigue rather than as a direct measurement of a specific motor unit recruitment strategy.

Despite the pronounced temporal reduction in MPF, the response was similar between the control and ammonia inhalant conditions. No significant treatment effect was observed, and the treatment × time interaction was also not significant. This similarity is illustrated in Figure 4, where the control and ammonia inhalant MPF trajectories remain closely aligned throughout the 30-s contraction. If ammonia inhalation meaningfully attenuated the myoelectric progression of fatigue under the conditions of the present study, differences in the temporal MPF response

between treatment conditions may have been expected. Instead, the temporal decline in MPF did not significantly differ between treatment conditions, suggesting that ammonia inhalation did not measurably alter the frequency-domain response of the biceps brachii during the sustained contraction.

The $RFD_{Decline}$ findings provide a complementary force-based assessment of fatigue. $RFD_{Decline}$ represented the slope of force production across the sustained contraction, with more negative values indicating a greater rate of force loss. No significant treatment effect, fatigue-state effect, or treatment × fatigue state interaction was identified. Furthermore, descriptive RFDDecline values following the fatigue protocol were −3.02 ± 1.38 N/s in the control condition and −2.52 ± 1.63 N/s following ammonia inhalation. Although the ammonia condition demonstrated a numerically less negative value, the treatment × fatigue state interaction and main effect of treatment were not statistically significant. Therefore, these descriptive differences do not provide sufficient evidence that ammonia inhalation reduced the rate of force loss.

The absence of a significant effect on $RFD_{Decline}$ is particularly relevant when considered alongside previous research examining ammonia inhalation during repeated high-intensity exercise. Rogers et al. (2022) reported significantly greater mean and peak power during repeated Wingate trials following ammonia inhalation, yet found no significant difference in fatigue index between ammonia and control conditions. Although the Wingate fatigue index used by Rogers et al. (2022) and $RFD_{Decline}$ used in the present investigation quantify fatigue using different methodologies, both studies failed to identify evidence that ammonia inhalation significantly altered their respective force- or power-derived indices of fatigue.

When the $RFD_{Decline}$ and MPF findings are considered together, there is little evidence that ammonia inhalation altered either the mechanical or myoelectric manifestation of fatigue

during the sustained isometric contractions. This finding is particularly relevant to the rationale of the present investigation. Previous studies demonstrating increased power during fatigued or repeated high-intensity exercise led to the possibility that ammonia inhalants may exert their greatest ergogenic effect when fatigue is present (Rogers et al., 2022; Secrest et al., 2015). However, the present results indicate that the presence of substantial fatigue alone was insufficient for ammonia inhalation to produce measurable alterations in the fatigue response of the biceps brachii. Rather, the potential ergogenic effects previously reported during repeated dynamic exercise may be dependent upon characteristics of the exercise modality or performance outcome being assessed.

Integrated Interpretation of Ammonia Inhalation and Neuromuscular Function

When considered collectively, the findings of the present study provide little evidence that ammonia inhalation alters neuromuscular function during fatigued sustained isometric contractions. Ammonia inhalation did not significantly influence maximal force production, RTP, T2M, biceps sEMG amplitude, $RFD_{Decline}$, or MPF. Importantly, the absence of treatment effects occurred despite clear evidence that fatigue was present. Maximal force production decreased by approximately 20% following the fatigue protocol, while MPF progressively decreased throughout the subsequent 30-s contraction. Therefore, the absence of an ammonia inhalant effect cannot readily be attributed to an insufficient development of fatigue within the experimental protocol. Instead, the findings suggest that the presence of muscular fatigue alone does not create conditions under which ammonia inhalants necessarily improve or deteriorate neuromuscular performance.

This distinction is important when considering the existing ammonia inhalant literature. Several studies have demonstrated physiological or perceptual responses following ammonia

inhalation without corresponding improvements in neuromuscular performance. Perry et al. (2016) reported transient increases in heart rate and middle cerebral artery blood flow velocity despite observing no improvement in force production, RFD, or muscular activation. Similarly, Campbell et al. (2022) demonstrated increases in heart rate, alertness, and perceived performance without corresponding improvements in maximal strength, RFD, peak power, or other measures of neuromuscular performance. Together with the findings of the present study, these results suggest that the acute physiological and perceptual response associated with ammonia inhalation should be distinguished from an ergogenic effect on neuromuscular performance.

More recently, Barnes et al. (2025) similarly found that ammonia inhalation did not attenuate reductions in maximal isometric force following a mental fatigue protocol and did not significantly alter EMG activity. Although mental fatigue differs physiologically from the exercise-induced muscular fatigue examined in the present investigation, these findings provide additional evidence that the presence of fatigue does not necessarily result in an ergogenic response to ammonia inhalation.

Conversely, significant improvements in performance have been reported under specific exercise conditions. Secrest et al. (2015) observed greater peak and mean Wingate power following ammonia inhalation after a simulated American football protocol, while Rogers et al. (2022) reported greater mean and peak power across repeated Wingate trials. These findings initially contributed to the rationale that ammonia inhalants may become more effective when fatigue is present. However, the present investigation directly incorporated a standardized fatigue protocol and found no significant treatment effect across multiple measures of subsequent neuromuscular function. Furthermore, Rogers et al. (2022) did not observe an effect of ammonia inhalation on fatigue index despite the improvement in Wingate power. Collectively, these

findings suggest that fatigue itself may not be the determining factor underlying previously reported ergogenic effects of ammonia inhalants.

Instead, differences in exercise modality and performance demands may contribute to the conflicting findings within the literature. The positive findings reported by Rogers et al. (2022) and Secrest et al. (2015) occurred during dynamic, high-intensity cycling, whereas the present investigation assessed sustained maximal isometric elbow flexion. Dynamic Wingate exercise requires repeated force production across multiple muscle groups and incorporates substantial metabolic and cardiovascular demands, while the present protocol isolated force production primarily to the elbow flexors under a fixed joint position. Accordingly, an acute increase in arousal may influence performance differently during whole-body dynamic exercise than during an isolated sustained isometric contraction. The present findings therefore do not demonstrate that ammonia inhalants are incapable of producing an ergogenic effect, but rather that such an effect was not observed across the measured indices of fatigued isometric neuromuscular function.

The convergence of the force and sEMG findings provides further support for this interpretation. No treatment effect was identified for maximal force production or the temporal characteristics of force development, and no corresponding treatment effect was observed in biceps sEMG amplitude. Similarly, neither the force-derived $RFD_{Decline}$ measure nor the development of the FSP, reflected by the progressive reduction in MPF, demonstrated evidence that ammonia inhalation altered fatigue. The consistency of these findings across multiple dependent variables strengthens the conclusion that ammonia inhalation did not meaningfully alter the measured neuromuscular response under the conditions of the present investigation. Rather than producing an isolated null finding within a single performance variable, the present

study observed a similar absence of treatment effects across mechanical, temporal, amplitude-based, and frequency-domain measures.

Limitations

Several limitations should be considered when interpreting the findings of the present study. One of the primary limitations was the inability to fully blind participants to the treatment condition. Although the treatment condition was concealed prior to administration, the distinct smell and irritative properties of ammonia made the treatment condition readily identifiable following inhalation. As a result, expectancy associated with receiving ammonia inhalants could not be eliminated. This limitation is particularly relevant given that previous research has demonstrated increases in perceived alertness and performance following ammonia inhalation even in the absence of measurable improvements in neuromuscular performance (Campbell et al., 2022). However, the absence of significant treatment effects across the outcomes measured in the present study suggests that any expectancy associated with identifying the ammonia condition was insufficient to produce detectable improvements in neuromuscular performance.

A second limitation concerns the standardization of ammonia exposure between participants. Although the administration procedure was standardized, including ammonia concentration, administration distance, and inhalation duration, the actual volume of ammonia inhaled could not be directly controlled. Differences in inhalation depth were observed between participants, and the potent smell and irritation produced by ammonia occasionally resulted in involuntary withdrawal from the inhalant. Consequently, the effective exposure to ammonia may have varied between participants. This variability may have contributed to interparticipant differences in the physiological response to the treatment. However, this method of

administration also resembles the practical use of ammonia inhalants in athletic environments, where inhalation depth and individual tolerance are unlikely to be precisely controlled.

Some variability was also observed in arm positioning during the sustained isometric contractions. Participants were instructed to maintain a supinated forearm position; however anecdotally, some participants moved toward a more neutral forearm position during portions of the contraction. Changes in forearm position may alter the relative mechanical contribution of the elbow flexor muscles and could therefore introduce variability into force production and biceps sEMG measurements (Naito et al., 1995). Although participant positioning was monitored throughout testing, the arm was not mechanically constrained from rotating during the contraction.

Finally, the sample consisted of 14 male varsity athletes, with complete sEMG data available for 13 participants. The relatively small and homogeneous sample limits the extent to which the findings can be generalized to other populations, including female athletes, recreationally active individuals, and individuals with different resistance-training backgrounds. Previous ammonia inhalant research has also produced differing findings across participant populations, including significant improvements in repeated Wingate performance among female participants (Rogers et al., 2022). While this study did not exclude female participants during recruitment, future investigations should make the effort to incorporate larger and more diverse participant samples to help determine whether participant characteristics influence the neuromuscular response to AIs. Specifically, comparisons between sex and their response to AIs may strengthen the understanding of AIs influence on physiology.

Practical Implications

The findings of the present study provide practical information regarding the use of ammonia inhalants during fatigued athletic performance. Ammonia inhalants are commonly used by athletes with the intention of increasing alertness or preparing for maximal effort, particularly during strength-based competition (Pritchard et al., 2014). However, the present findings do not support the use of ammonia inhalants as a means of preserving maximal isometric force production or attenuating neuromuscular fatigue following fatiguing exercise. Despite a reduction in maximal force production of approximately 20% following the fatigue protocol, ammonia inhalation did not significantly improve maximal force production, RTP, T2M, muscular activation, $RFD_{Decline}$, or the progression of MPF throughout the sustained contraction.

These findings should not be interpreted as evidence that ammonia inhalants are ineffective across all forms of athletic performance. Previous studies have demonstrated improvements in mean and peak power during repeated or post-fatigue Wingate exercise (Rogers et al., 2022; Secrest et al., 2015), whereas the present investigation evaluated sustained maximal isometric elbow flexion. Therefore, the practical effects of ammonia inhalants may be dependent upon the characteristics of the activity being performed. However, for athletes using ammonia inhalants with the expectation that they will directly preserve force-producing capacity or reduce the neuromuscular effects of fatigue during sustained isometric exercise, the present findings provide no evidence to support this expectation.

Future Research

Future research should continue to investigate the circumstances under which ammonia inhalants may influence athletic performance and physiological processes as called for by a recent systematic review and meta-analysis Liu et al. (2026). In particular, further investigation is required to determine whether the conflicting findings within the literature are attributable to

differences in exercise modality. Significant improvements following ammonia inhalation have primarily been observed during repeated or fatigued dynamic exercise (Rogers et al., 2022; Secrest et al., 2015), while the present study and previous investigations of maximal isometric performance have generally failed to identify improvements in maximal force production (Bartolomei et al., 2018; Perry et al., 2016). Future studies could directly compare dynamic and isometric exercise within the same participants and experimental design to determine whether exercise modality influences the ergogenic response to ammonia inhalation. Identifying a potential modality which reliably produces an ergogenic response from ammonia inhalation will strengthen the scientific understanding of how AIs interact with human physiology. This may help to expose why other exercise modalities are not provided with ergogenic benefits from AIs which will provide athletes with more informed use of this substance in sports.

Further investigation should also consider the individual variability associated with ammonia inhalation. Although administration procedures can standardize ammonia concentration, distance, and intended exposure duration, the amount inhaled may vary due to differences in inhalation depth and individual tolerance to nasal and ocular irritation. Future research incorporating methods capable of better quantifying ammonia exposure may help determine whether variability in effective exposure contributes to the inconsistent performance responses reported within the literature. Additionally, larger and more diverse participant samples are required. The present investigation included male varsity athletes, whereas Rogers et al. (2022) demonstrated improvements in repeated Wingate performance in physically active female participants. Studies adequately representing both male and female athletes may therefore help determine whether participant characteristics contribute to differences in the response to ammonia inhalation.

Finally, future investigations examining the proposed neuromuscular effects of ammonia inhalants would benefit from techniques capable of more directly evaluating motor unit behaviour. Conventional bipolar sEMG amplitude provides useful information regarding global muscular activation but has limitations when used to infer specific neural strategies such as motor unit recruitment and discharge behaviour (Vigotsky et al., 2018). More advanced approaches, including high-density surface EMG and decomposition techniques, can provide information regarding the discharge behaviour of individual motor units and may therefore allow more direct investigation of whether ammonia inhalation alters neural drive, motor unit recruitment, or discharge rate. Reviews of surface EMG methodology support the use of decomposition approaches when the research question requires information about individual motor-unit behaviour rather than global sEMG characteristics (Farina et al., 2014). Such approaches may be particularly useful for investigating the proposed neural mechanism underlying previously reported changes in explosive force production following ammonia inhalation.

Conclusion

The purpose of the present study was to determine the effect of ammonia inhalants on neuromuscular function following fatiguing exercise. Specifically, maximal isometric force production, rate of force development, muscular activation, and force- and sEMG-derived indices of fatigue were examined during sustained maximal isometric contractions with and without ammonia inhalation. The findings demonstrated that ammonia inhalation did not significantly influence maximal force production, RTP, T2M, biceps sEMG amplitude, $RFD_{Decline}$, or MPF.

Importantly, the absence of an ammonia inhalant effect occurred despite clear evidence of fatigue. Maximal isometric force production decreased by approximately 20% following the fatigue protocol, while MPF progressively decreased throughout the 30-s fatigued contraction. These findings demonstrate that the experimental protocol produced measurable changes associated with fatigue, but ammonia inhalation did not significantly alter the mechanical or myoelectric responses examined in the present study.

Collectively, the findings do not support the use of ammonia inhalants as a strategy to preserve maximal isometric force production or attenuate neuromuscular fatigue during sustained isometric exercise in male varsity athletes. Although previous research has demonstrated potential ergogenic effects during specific forms of repeated dynamic exercise, the present findings suggest that the presence of fatigue alone is not sufficient for ammonia inhalation to improve neuromuscular performance. Instead, previously reported ergogenic effects may depend on characteristics of the exercise modality, performance outcome, or participant population.

Further research incorporating different exercise modalities, larger and more diverse participant samples, improved quantification of ammonia exposure, and more direct assessments

of motor unit behaviour may help clarify the circumstances under which ammonia inhalants influence athletic performance. Nevertheless, within the conditions examined in the present investigation, ammonia inhalation did not significantly alter neuromuscular function during fatigued sustained isometric contractions.

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