**Title:** Recent developments in the use of sodium bicarbonate as an ergogenic aid

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There are no conflicts of interest to declare.

**Abstract**

This review examines the current status of sodium bicarbonate as an ergogenic aid. It builds on previous reviews in the area. Current research would suggest that as an ergogenic aid, a 300 mg.kg-1 dose of NaHCO3 can improve high intensity exercise, within a range of exercise modalities, such as a single bout of supramaximal exercise, high intensity intermittent activity and skill based sports. In particular, these benefits seem to be present to a greater extent within trained individuals. Despite this, there appears to exist a high intra-individual variability in response to NaHCO3 and therefore the ergogenic benefits may not be induced during every exercise bout. Current thinking also suggests that, athletes need to individualize their ingestion timings to maximise peak pH or blood bicarbonate, to effectively maximise the performance effect, and may allow individuals to attain the ergogenic benefits of NaHCO3 more consistently.

Keywords: ergogenic aid, performance, acid-base balance, gastro-intestinal upset, buffers

Brief: The ergogenic benefits of sodium bicarbonate ingestion work best in elite athletes

1. **Introduction**

A myriad of factors determines skeletal muscle fatigue and exercise performance, which involves a complex interaction of central and peripheral components (30, 47, 57). The determinant(s) of fatigue however, are dependent on the types, durations and intensities of exercise undertaken by the individual. Central components are primarily mediated by afferent feedback to the central nervous system in order to maintain conscious and/ or sub-conscious control of exercise to prevent catastrophic failure of homeostatic regulation (47, 57). Peripheral fatigue however, is often related to the excessive accumulation of metabolites, such as hydrogen ions (H+), potassium ions (K+) and phosphate ions (Pi+); and the availability of metabolic fuel sources (30).

Early work studying the aetiology of fatigue, proposed that exercise-induced acidosis, (when the rate of hydrogen ion (H+) production surpasses the rate of removal), was a major contributing factor (29). Exercise can induce substantial perturbations to the acid-base balance through the generation of H+, the extent of which is dependent on exercise intensity and duration (12). Indeed, such circumstances are associated with the development of fatigue and although the contributing mechanisms remains ambiguous, a number of methods have been proposed. These mechanisms include the dysfunction of the sarcoplasmic reticulum due to altered calcium ion sensitivity and handling (19, 27), a reduced myosin-actin cross-bridge cycling activity and increased potassium ion release (5), which together can impede muscular myofilament function and excitation- contraction coupling. Furthermore, glycolytic flux is also inhibited, with key glycolytic enzymes, such as phosphofructokinase, down regulated under an acidic stress (37). Considering this association of exercise-induced acidosis and fatigue, the application of exogenous buffering agents, such as sodium bicarbonate (NaHCO3), to dampen the rate of H+accumulation and attenuate the magnitude of metabolic acidosis may be warranted. Indeed, NaHCO3 has been investigated as an ergogenic aid for over 80 years, with recent evidence alluding to an ergogenic potential during short duration high-intensity exercise (50). Early research from McNaughton et al, (51) identified that NaHCO3 possessed ergogenic benefits during 120 s and 240 s of ‘all-out’ high intensity exercise but not exercise of 10 s and 30 s in duration. Other investigations of equivalent durations also reported a significant running sprint time improvement in 400 m (33) and 1500 m (8).This ergogenic benefit is not however limited to short duration exercise bouts, as performance improvements have also been reported during prolonged 60-minute exercise bouts (48), repeated sprint exercises and combat sports (3). The performance benefits observed with NaHCO3 ingestion are not synonymous in the literature with studies also reporting no effect of NaHCO3 ingestion (26, 69, 81).

This aim of current review is to provide an update on the scientific literature relating to the supplementation of NaHCO3 from circa 2008 to 2016, since the previous review of McNaughton et al. (50) More specifically, the review will address research relating NaHCO3 dosage strategies and the ergogenic influence on a range of exercise modalities. Beyond the exercise performance related literature, emerging evidence has presented the potential use of NaHCO3 as a training aid and is suggested to effect the on the physiological stress response to exercise, these aspects will also be explored in further detail. For an in-depth review of literature pertaining to the performance effect of NaHCO3 of literature prior to 2008, readers are directed to a previous by McNaugton et al. (50), however an overview of this research is presented in Table 1.

1. **Mechanism of Action**

The ergogenic effect of NaHCO3 on exercise performance is stems from the reinforced extracellular bicarbonate buffer capacity to regulate of acid-base balance during exercise. Ingestion of NaHCO3 gives rise to bicarbonate ions (HCO3-), thus contributing to an alkalotic environment in the extracellular fluid compartments (17). Concurrently, the elevated HCO3- enlarges the gradient between extracellular and intracellular H+, which stimulates the lactate/H+ co-transporter (68). In turn, a greater efflux of H+ from intramuscular regions into the extracellular fluid; allowing HCO3- and buffering compensatory systems to remove H+, therefore increasing pH. The direct mechanism by which the inducement of alkalosis evokes an ergogenic response on exercise is however, unclear. Numerous propositions surrounding both peripherally and centrally driven mediators of fatigue and exercise performance have been investigated (78). Such mechanism include the attenuation of exercise induce arterial oxygen desaturation allowing for enhanced oxygen delivery (56), delayed impairment of muscular contractile properties (88) and augmented glycolytic flux (38). More recently, research is indicative of an altered neuromuscular response with pre-exercise NaHCO3 administration (39, 79). A neuromuscular response that is characterised by a reduced rate of force production decline, during isometric contractions following a bout of sub-maximal (39) and repeated bouts of high intensity exercise (80). The suggestion is therefore that NaHCO3 modifies peripheral indices of fatigue to improve exercise performance. In addition, evidence has also alluded to a central derived contribution to NaHCO3 ergogenic effect (64). During a combination of ischemia and repeated maximal voluntary contractions, voluntary activation was preserved to a greater extent with prior alkalosis compared to control (76 ± 5% vs. 57 ± 8%, p<0.05; 77). Voluntary activation is used an indicator of descending central drive, which is hypothesised to be preserved with NaHCO3 due to the reduced attenuation of group III and IV afferent firing under dampened acidic conditions (64). The proposition of a centrally acting mechanism for NaHCO3 is however not new, as early work by Swank and Robertson, (83, 84) introduced the view of NaHCO3 lowering the subjective perception of exercise intensity. Such an observation aligns with the widely debated psychobiological model of fatigue (47) and suggests the NaHCO3 mechanisms of action is centrally derived as a psychoactive drug. Nonetheless, further work is required to elucidate the mechanism by which NaHCO3 acts, although it is likely to be an interplay of peripheral and central components.

1. **Dose**

To attain the ergogenic benefit on performance from NaHCO3, it is recommended that a state of peak alkalosis is required, which refers to post-ingestion peak in HCO3**-** concentration (68, 80). In theory this will lead to a greater efflux of H+ from the intracellular to extracellular compartments to be buffered during high-intensity exercise compared to a resting HCO3- concentration and attain maximum buffering capacity (see Mechanisms of Action section). It would seem however the point in time at which such a peak is achieved is abstruse. Indeed, Siegler et al. (80) identified within eight males, peak HCO3- occurred at 60 minutes post-ingestion of 0.3g**.**kg-1 BM NaHCO3, supporting the 60 to 90 minute recommendation by Price and Singh (68) to achieve a peak HCO3-. Nevertheless, a study by Carr et al. (17), using various ingestion timings, fluid intake, co-ingestion of a small carbohydrate meal (1.5g**.**kg-1 BM) and either a capsule or solution of NaHCO3, displayed contrasting results. More specifically, peak HCO3- was established at 150 minutes, a total of 90 minutes later than the Siegler et al. (80) study. Such a large difference may have been due to the co-ingestion of the carbohydrate meal, subsequently effecting the absorption rate of NaHCO3. Nevertheless, a study by Siegler et al. (79) with no co-ingestion strategy reported that peak HCO3**-** was achieved at 180 minutes post supplementation of 0.3g**.**kg-1 BM NaHCO3. This displays that currently, little consistency over the time until a peak HCO3**-** is achieved exists within the literature, potentially leading to confusion over the most appropriate dosage strategy for athletes to utilize in competition or training.

Contrary to the popular belief that attaining peak HCO3**-** concentration is essential to secure a performance enhancement, a study by Siegler et al. (80) displayed no effect on performance when exercise began at different time points (60, 120 & 180 min) following NaHCO3. In more detail, eight recreationally active males performed ten, 10-second maximal running sprints and displayed no difference in both peak power output (PPO) (60: 984 ± 208 W; 120: 916 ± 131 W; 180: 987 ± 228 W; p = 0.18) and distance covered (60: 456 ± 35m; 120: 448 ± 32m; 180: 448 ± 32m; p = 0.22). A possible limitation to this study is the assumption that a particular level of alkalosis (i.e. HCO3**-**) had been achieved at each time point when exercise commenced, and this was also based on group level data. Highlighted in these aforementioned studies the dose-response shows a high variation, and thus it is potentially difficult to ascertain whether each participant was at the respective peak the authors were attempting to replicate. Furthermore, in a recent study by Miller et al. (53), the authors mapped the time to individual peak pH following 0.3g.kg-1 BM NaHCO3, and determined that the average time to peak pH was 68.2 ± 21.0 minutes, but with a population response range from 10-90 minutes post-ingestion. This demonstrates a high variability in the dose-response from NaHCO3 and possess a considerable caveat to all previous research that have typically utilized a standard pre-ingestion time of between 60-90 minutes and interpreted the response on a group level. A consideration however, is that the Miller et al. (53) study was based on time to peak pH not HCO3**-**, which would be a greater indication of the NaHCO3 response. Nevertheless, these results may have considerable practical significance, as a more personalised approach to NaHCO3 supplementation can be utilised by athletes to elicit the ergogenic effects. Further research should look to adopt a personalised approach, to ascertain the effects on various modes of exercise and identify the absorption characteristics in other populations.

A prominent determinant of NaHCO3’s use by athletes may arguably be the manipulation of dosage strategies to limit the onset of gastrointestinal discomfort (GI). It is now well reported in literature the negative GI symptoms caused by NaHCO3 (13, 40, 74), however the potential effect such instances can have performance remains ambiguous. Indeed, Saunders et al. (74) reported a 4.7% improvement in total work done (TWD) during a cycle to exhaustion at 110% PPO, only when four participants that suffered from GI were removed from a group of twenty-one recreationally active men (all participants: Placebo = 45.6 ± 8.4, NaHCO3 = 46.8 ± 9.1 Kj, p 0.16; d = 0.14; Without GI: PLA = 46.2 ± 9.2, NaHCO3 = 48.4 ± 9.3 Kj, p<0.05; d = 0.25). Similarly, Cameron et al (13) reported a relationship, albeit weak, on the incidence of GI negatively effecting sprint performance (p = 0.90, r2 = 0.12). Nonetheless, research has demonstrated performance benefits and incidences of GI can co-exist (51, 53) and therefore the relationship between incidences of GI and the subsequent effect on performance remains unclear. Identifying strategies that reduce the incidences of GI is still a worthwhile area of investigation however, as incidences such as nausea, diarrhea and vomiting are a serious practical consideration for athletes. If a dosage strategy could be identified that leads to an enhanced performance, whilst limiting the incidence of GI, this may enhance the prevalence of NaHCO3 in both training and competition.

1. **Ergogenic Effect of NaHCO3 on‘All-Out’ and Supra-maximal Exercise**

Research assessing the ergogenic effect of NaHCO3 on short duration high intensity exercise has been equivocal, with improvements shown in some duration based ‘all-out’ performances with trained individuals (7,24,86) but not those investigated in an untrained, recreationally active cohort (61, 87). A significant improvement in mean power output four-minute ‘all-out’ exercise performance was reported with both serial (427.5 ± 43.6 W) and acute (431 ± 37.9 W) doses of NaHCO3 compared to placebo (418.3 ± 41.5 W), representing up to a 3% improvement in performance (24). Equally, Bellinger et al., (7) found a 3% improvement in mean power output during four-minute ‘all-out’ exercise performance in eight similarly trained individuals (<65 ml. kg-1. min-1), whilst a magnitude based inference analysis revealed the 87% likelihood NaHCO3 having a positive performance effect on four-minute ‘all-out’ exercise. This positive effect is not however, representative in all investigations with both Vanhatalo et al., (88) and Peart et al., (62) observing unaltered performance during three and four-minutes of high intensity exercise bouts with NaHCO3 supplementation respectively, in untrained participants. This difference in training status between the aforementioned studies indicates that the ergogenic properties of NaHCO3supplementation during ‘all-out’ high intensity exercise performance may be more apparent in better trained individuals. An observation that corresponds with a meta-analysis that discovered a moderate performance enhancement if 1.7±2.0% (mean ±confidence interval) in athletic cohorts compared to a negative effect in nonathletic cohorts (-1.1±1.1%). The reason for this observation remains unclear, although it is reasonable to postulate that a greater athletic status of participants may improve the reliability to detect the ‘true’ ergogenic effect of NaHCO3, simply because they are able to replicate performance more reliably.

Numerous investigations assessing the ergogenicity of NaHCO3 during constant load supra-maximal exercise at 110-120% PPO has demonstrated no change in mean performance (33, 72, 74). These investigations were conducted with recreationally active individuals, and as previously suggested, the training status of participants may account for the lack of detectable change. Alternatively, this lack of change could be attributed to emerging reports of a within-subject variability to the ergogenic effect of NaHCO3 reported within untrained individuals (22). In this study, 15 recreationally active participants conducted six 110% PPO trials to exhaustion, which involved four with NaHCO3 ingestion and two placebo ingestion. Across the 4 NaHCO3 trials, blood acid-base response was consistent, that is mean pH, base excess and bicarbonate ion concentrations were not reported to be different in each NaHCO3 trial. Despite the blood response, performance across the four trials was not consistently improved following supplementation, with performance only significantly improved compared to placebo in one trial. Magnitude based inference calculated across the four trials also indicated this mixed response, with the likelihood of an improvement reported to be 54% (possible), 7% (unlikely, 32% (possible) and 93% (likely) from NaCHO3 trials one to four respectively. Delving deeper, this study also observed an inconsistency in individual response to NaHCO3,as it was found that 10 participants improved performance in at least one of the four trials, however five participants did not exhibit improved performance in any of the trials. This study therefore may allude to a responder/ non-responder phenomenon to the ergogenic potential of NaHCO3 (22). Aligned with the previously mentioned discussion of training status, it may be conceivable that the nature of the athletes used by Dias et al. (22) may account for some of the variability observed, therefore further research is required to assess if this variability is present in a trained cohort.

1. **Ergogenic Effect of NaHCO3 onHigh Intensity Intermittent Exercise**

The efficacy of NaHCO3 on various intermittent exercise, including sport specific simulations has been investigated in a range of exercise modalities, such as swimming (75, 92), running (13, 25, 42, 74) and cycling (31, 53, 77). With specific reference to repeated sprint ability (RSA), generally positive effects on performance have been demonstrated. Indeed Miller et al. (53) reported a 17% and 11% improvement in TWD compared to control and placebo, respectively (69.8±11.7 vs. 59.6±12.2 vs. 630±8.3 s; p<0.05) during 6x10 s maximal cycling. Comparably, Krustrup et al. (41) also reported the total running distance improved by 12% during a football specific Yo-Yo intermittent recovery test (735±61 vs. 646±46 m; p<0.05). In contrast, Ducker et al., (25) reported a markedly lower improvement demonstrating total sprint time was 2.2% faster time to completion of three sets of 6x20 m sprints with NaHCO3 supplementation 60 minutes prior to exercise (60.33±2.62 vs. 59.05±2.83 s; p<0.05). The differing intermittent activity patterns of the aforementioned studies do not allow for direct comparison, although the different supplementation methods used may explain the larger ergogenic effect noted by Krustrup et al., (42) and Miller et al., (53). A higher NaHCO3 dose of 0.4 g.kg-1 BM was used by Krustrup et al. (42), therefore increasing the availability of HCO3- to buffer H+ to a greater extent during exercise. The authors however, did not use a placebo treatment, therefore some of the effect could be attributed to a placebo effect. The NaHCO3 treatment condition lead to an enhanced sodium ingestion that may cause an expansion in plasma volume, which has also been shown to possess ergogenic properties. Thereby masking the true buffering potential of NaHCO3 to enhance performance. Conversely, Miller et al., (53) elicited an 11% improvement in TWD with the new personalised dosage method in comparison to an equimolar, taste matched placebo treatment. This signifies the enhanced ergogenic influence that may be provoked from a personalised strategy.

Research investigating high intensity intermittent type (HIIT) exercise has produced equivocal performance responses to NaHCO3 supplementation. Swimming performance in eight well trained competitive youth swimmers completing 4x50 m front crawl swims interspersed with one minute passive rest periods was improved by 2.6% with NaHCO3 (112.9 s) compared to placebo (114.3 s). Likewise, a 2% improvement in total swim time was exhibited with NaHCO3 (159.4 ± 25.4 vs. 163.2 ± 25.6) during 8x25 m front-crawl, interspersed with 5 seconds rest (76). This was despite a pre-supplementation time of 2.5 hours, substantially longer than previously reported mean peak alkalosis conditions at 60-90 minutes (68). Together, this indicates NaHCO3 may be beneficial for intermittent swimming using different recovery periods. In a further study involving a hypoxic stimulus, encompassing a reduction in the fraction of inspired oxygen (FIO2) to 14.7% (approx. 3000m), no effect on performance was recorded following NaHCO3 against a placebo. In theory, hypoxia presents a substantial challenge to acid-base balance, subsequently offering ideal conditions to produce the ergogenic effects of NaHCO3, however this has not been represented in the available literature. In more detail, twelve healthy participants cycled at 120% PPO for 30 s interspersed with 30 s active recovery at 30% PPO, until volitional fatigue. However, the cumulative time spent cycling 120% of PPO was no different between placebo (133.3±28.7 s) and the NaHCO3 (127.8±27.9 s) conditions. Performance was greater under normoxic conditions, however no significant differences existed between normoxic NaHCO3 and placebo supplementation protocols, despite performance improvements of 8.3% with NaHCO3 (183.8±45 vs 199.1±62 s). A single bout of 120% PPO exercise has also demonstrated no improvements in exercise performance following NaHCO3 ingestion in a similar recreationally active cohort (34, 72). This suggests the supra-maximal intensity may be too severe to benefit from NaHCO3 and that the early termination of exercise at 120% PPO may, although speculated, be linked to the rate of change in pH. With such a severe change during 120% PPO, this may lead to the saturation of the muscle membrane transporters monocarboxylate transporter (MCT) 1 and 4 or the sodium-hydrogen exchanger (46). Nevertheless performance did improve under normoxic conditions at 120% PPO when performed intermittently, although non-significantly.

Empirical research has also investigated sport simulations within a range of sports including football (73), rugby (13) and water polo (85), with none reporting positive benefits from NaHCO3. A more sport specific football simulation protocol, with incorporated 5x6 s maximal sprints pre and post two football specific intermittent treadmill protocol (FSINT) under acute hypoxia (FIO2 15.5%, approx. 2500m), also revealed no difference in exercise performance (73). The lack of improvement however, could be due to the NaHCO3 ingestion strategy which entailed two discrete doses over a four hour period (0.2g.kg-1 BM 4 hours, 0.1g.kg-1 BM 2 hours prior to exercise). More specifically pre-exercise HCO3- was 28.6 ± 1.6 mmol**.**L-1, which is lower than the peak HCO3**-** reported in previous work (17, 68). Therefore not enhancing buffering capacity to its maximum and consequently obtaining no ergogenic benefit during exercise. Furthermore, the length of the protocol (>90mins in total) may have been a contributing factor to why no performance benefit was observed, as this was outside of the 1-10 minute window suggested to elicit the largest ergogenic effects (15). Nevertheless in a study supplementing 0.3g**.**kg-1 BM NaHCO3 60 minutes prior to a 9 minute high-intensity rugby specific exercise, no effect on performance was also recorded when compared to a placebo, within a group of twenty-five elite male rugby players. This study also entailed a 25 minute warm-up prior to the performance, which may have already utilised the enhanced buffering capacity prior to the rugby specific exercise, therefore failing to enhance the 9 minute exercise. Research which focused on water polo, also mimics such findings. A group of twelve elite female water polo players completed a 59 minute match simulation test entailing 56x10m sprints, with no improvement in mean sprint time between NaHCO3 and the placebo (NaHCO3 = 6.88 ± 0.28 vs. Placebo = 6.91 ± 0.31 s; d = 0.09). Similar to the Saunders et al. (73), it might be the case the 59 minutes match simulation test was outside of the 1-10 minute window for NaHCO3 to elicit an ergogenic effect. In summary, research simulating sport performance has shown no positive effects on performance from NaHCO3.

1. **Ergogenic Effects of NaHCO3 on Distance Based Time Trial Events**

Research assessing the influence of NaHCO3 on distance based time trial (TT) events have focused on rowing (14, 16, 20, 35, 43) and cycling (41, 58). In relation to rowing, research has focussed on determining the effect of NaHCO3 on 2 km TT’s and have largely reported unequivocal results of unaltered performance (14, 16, 20, 35, 36, 43). Carr et al. (16) reported no change in performance times with NaHCO3 compared placebo within a group eight well trained rowers (NaHCO3 6:44.4 ± 23.4 vs. Placebo 6:43.8 ± 23.4). Results that have been replicated after both acute and three-day serial supplementations in well trained rowers (14, 43). These observations were also replicated amongst elite rowers (20) during a 6-minute high intensity bout, which is equivalent to the expected time to complete 2 km. Twelve international rowers completed similar distances with NaHCO3 and placebo at 1860±96 m and 1865±104 m, respectively. Taken together, this research suggests NaHCO3 may not be an effective ergogenic aid for trained rowers during simulated rowing performance. Hobson et al. (36) however, provides an interesting caveat to the previous outlined research, in that 500 m split times revealed a significantly faster performance during the second half of the 2 km row. Acute NaHCO3 ingestionstimulated a 0.5 ± 1.2 s improvement in the third 500 m split and a 1.1 ± 1.7 s improvement in the fourth 500 m split, with magnitude based inferences representing likelihood of beneficial effect of NaHCO3 as possible and very likely during the third and fourth 500 m split, respectively. Despite this, overall performance did not change under the alkalotic conditions. This study does however provide an interesting insight into the potential pacing considerations when supplementing with NaHCO3, for example the split times reported by Hobson et al. (36) are suggestive of a negative pacing strategy. Negative pacing strategies are often used with in distance based time trials as it allows for an intense burst towards the end of the event which is driven by an enhanced utilisation of anaerobic reserves (32), access to which may be facilitated by NaCHO3 due to the enhanced glycolytic flux (38). As such NaHCO3 could potentially be beneficial for athlete utilising such pacing strategies, although further work is required to test this initial observation.

In contrast to rowing, cycling TT research has been limited, with two studies investigating two distinctly different distance of 4 km (41) and 40 km (58). During 3 km TT trials, performance was improved by 2.8% with NaHCO3 compared to placebo in ten well-trained cyclists (225.9 ± 11.3 s vs. 228.7 ± 10.8 s), although mean differences were not significant (41). Despite the lack of statistical significance, magnitude based inference analysis reported a NaHCO3 was very likely to have a positive influence on 3 km TT performance. Therefore, suggesting NaHCO3 may possess practical benefits to athletes competing over a short TT distances. In contrast, Northgraves et al. (2014) reported no benefits during 40 km TT distance from NaHCO3 supplementation, with mean time to completion of 67.08 ± 5.04 min with NaHCO3 compared to 66.41 ± 4.04 min in the placebo trial. This opposes earlier work conducted by McNaughton et al. (1999), during which well-trained cyclists (67.3 ± 3.3 performed an equivalent duration (60 minutes) time based TT and elicited a significant improvement in total work done with NaHCO3 compared to a placebo (950.9 ± 81.1 Kj vs. 839.0 ± 88.6 Kj; p< 0.03). The ergogenic effect of NaHCO3 is dependent on the degree of metabolic acidity (McNaughton et al. 2008) and interestingly, the degree of metabolic acidity does not appear to be different between both studies (~7.32), therefore the difference between studies is likely due to alternative factors. Time based and distance based time trials, although of similar duration, do exhibit discrete differences in an athlete’s perception of judging power output distribution (1). Therefore, the methodological difference may explain the difference observed between the two investigations. Moreover, trained individuals are suggested to elicit performance gains from NaHCO3 more readily (15), therefore the use of untrained participants by Northgraves et al. (58) may account for the contrasting results. In addition, untrained participants may impede the reliability of the exercise protocol, thereby making any ergogenic benefit. Based on current evidence, it may therefore be unwise to discount the use of a buffering agent during prolonged TT events at this early stage of research. Early research is indicative of a meaningful practical benefit of NaHCO3 on shorter duration high intensity TT performance, although further work is required to clarify this initial observation.

1. **Ergogenic effect of NaHCO3 on Skill Based Sports**

Fatigue can have a deleterious effect on decision making and skill execution during sports performance. On this premise, emerging research has assessed the influence of NaHCO3 on a range of skill based sports, including boxing (77), judo (28, 87) and tennis (90). A study by Siegler and Hirscher (77), featuring 10 amateur boxers, investigated the effects of NaHCO3 prior to sparring bouts of 4 x 3 minute rounds, interspersed with 1-minute seated recovery. Compared to placebo, NaHCO3 evoked a significant 5% (p<0.01) increase in overall punch efficacy (i.e. successful punches landed), along with improvements of 11.4% (88±12 vs 79±10) in round 1, 4.9% (86±10 vs 82±) in round 3 and 7.5% (86±13 vs 80±10) in round 4 following NaHCO3 but no difference in round 2. This study presents an encouraging area of investigation, but the test-retest reliability of such sparing protocols is unclear. Furthermore, the study makes no distinction between weight classes of participants and only evaluated 4 rounds when a typical boxing match lasts up to 12 rounds. Therefore, it is difficult to ascertain the true effect of NaHCO3 on a full bout. Another combat sport related study assessed the efficacy of NaHCO3 in the special judo fitness test (SJFT) that comprised of a 3 sets (1 x 15 s and 2 x 30 s), with performance assessed via total number of judo throws. There was not was no difference in performance between the treatment and placebo conditions, although the reliability of such a protocol to detect change is unclear. Despite this, an upper body Wingate test (4 x 30 s interspersed with 3 minutes recovery) in 9 judo and jujitsu athletes found that 7 days chronic NaHCO3 (0.5 g. kg-1 body mass) enhanced TWD by 8% (77).

Other skill based sports, such as tennis are also sensitive to the ergogenic benefits of NaHCO3 (90). Tennis shot consistency during the Loughborough Tennis Skill Test (LTST), eliciting greater skill maintenance in 9 male national level tennis players. More specifically, consistency of service declined by 34.3% in the placebo trial (16.9 ± 5.4 to 11.1 ± 6.0%), compared to 1.4% in the NaHCO3 trial (13.8 ± 5.1 to 13.6 ± 5.9%) and forehand shot consistency was reduced by 13.3% in the placebo trial (10.5 ± 2.8 to 9.1 ± 2.0%) but improved by 16% during NaHCO3 condition (8.0 ± 1.6 to 9.3 ± 2.6%). The mean differences in forehand and serve consistency skill execution was significantly maintained to a greater extent with NaHCO3. Together, this research suggests that NaHCO3 may be a promising ergogenic strategy to maintain skill execution throughout a fatiguing bout of exercise. However, it appears that further research would need to adopt reliable, or state the reliability of, the testing protocols.

1. **The Potential Use of NaHCO3 as a Training Aid**

Enhancing training-induced adaptations is a prominent area of sports nutrition research with two dietary approaches known as “training better” and “training smarter”, strategies that are often used by athletes to maximise training outcomes (11). The former involves the use of dietary strategies to enhance the quality of training, modulating training intensity and/ or volume. In contrast, the later, “training smarter” approach, is designed to result in enhanced training-induced molecular adaptations through dietary strategies without additional training stressors. These approaches typically involve carbohydrate manipulation during training, however emerging evidence suggests sodium bicarbonate supplementation has the potential to induce training adaptations through both methods.

Due to the ergogenic properties of NaHCO3 to improve a single bout of high intensity exercise, it is conceivable that NaHCO3 could be used as daily training supplement to enhance the quality of individual training bouts. Indeed, Mueller et al., (55) assessed five consecutive days of acute 0.3g **.** kg-1 0BM NaHCO3 on time to exhaustion during constant load cycling exercise at an intensity equivalent to critical power. Performance was greater on each day following NaHCO3 compared to placebo, presenting a significant mean improvement of 23.5% (p = 0.001). Therefore, demonstrating daily acute supplementation is a viable nutritional strategy for athletes, however the efficacy of NaHCO3 during a training intervention utilising a “training better” approach has yet to be established.

Emerging evidence indicates that the regulation of acid-base balance, via NaHCO3, during high-intensity exercise bouts may stimulate oxidative molecular pathways, and in turn, promote aerobic capacity without inflicting further training stressors (9, 26, 63). Early work by Edge et al. (26) found that supplementation of NaHCO3 prior to trainingduring an eight week (three sessions per week) supervised training programme with moderately trained females (V̇O2peak: 42.1 ± 7.0 ml·kg−1·min−1) resulted in a significantly larger improvement in lactate threshold (LT) and time to exhaustion by 42% and 25% (p<0.05), respectively, compared to a matched exercise only group. Training sessions between groups were matched for volume and intensity, thereby matching total training stress during interventions. Moreover, despite the reinforced buffering capacity with NaHCO3 during acute bouts of exercise, it did not translate to an altered intramuscular buffering capacity between groups, which is suggestive of an alternative mechanism for adaptation. Lactate threshold is known to be a good indicator of aerobic capacity (52), which implies adaptations were likely to be aerobic in nature. Indeed, this assertion is supported in a latter investigation by Percival et al. (63), which reported an up-regulation of the peroxisome proliferator-activated receptor γ co-activator-1 alpha (PGC-1α) mRNA expression by 28% during recovery from a repeated high intensity exercise bout with pre-exercise ingestion of NaHCO3 compare to exercise alone. Repeated up-regulation PGC-1α expression through a combination of exercise and NaHCO3 has been demonstrated to augment PGC-1α protein content and consequently stimulate mitochondrial biogenesis in a Wistar rat model (9). This improved muscular oxidative potential may explain, at least in part, the superior training related adaptations in LT noted by Edge et al., (26). These performance enhancements were not however observed by Driller et al. (23), six highly trained international rowing athletes, following a four week (two sessions per week) training period with NaHCO3 ingestion. It is unclear if PGC-1α was increased in these trained individuals since muscle biopsies samples were not obtained. Furthermore, it is conceivable that the low volume of exercise sessions with prior NaHCO3 ingestion in this study compared to four exercise sessions over eight weeks by Edge et al. (26), may not be sufficient to elicit detectable improvements in performance. Consequentially, further research is required to determine if pre-exercise alkalosis can induce oxidative adaptation in highly trained individuals and assess if it is an efficacious method to “train smarter”.

1. **The effect of NaHCO3 on physiological stress response**

Contemporary research has explored the influence of pre-exercise NaHCO3 ingestion on physiological parameters beyond the reputable ergogenic role during high intensity exercise. More specifically, a series investigations conducted by Peart et al., (59-62) has addressed the potential of pH regulation to attenuate cellular stress (e.g. heat shock protein; HSP) experienced during exercise. The HSP-70 isoforms have been cited to have distinct importance during exercise, due to their involvement in cytoprotective functions within cells in response to an exercise stressor (45, 49, 54). Heat shock proteins also possess an important exercise pre-conditioning response, whereby exposure to an initial exercise stressor can improve the tolerance to a subsequent exposure. As such, greater understanding of response and role of HSP-70 isoforms during acute and repeated bouts of exercise may enhance our appreciation the mechanisms regular exercise can induce adaptations for performance or protective function for health. A number of potential exercise related stimuli have been proposed to instigate HSP70 isoform response, including thermal, metabolic, oxidative, cytokine and acidic stress (McNaughton et al. 2006; and Morton et al., 2009); although the direct mechanism remains unclear as all these factors coincide during exercise.

The ingestion of NaHCO3 prior to a single bout of anaerobic (62) and high intensity intermittent exercise (HIIE) (59, 61) is shown to blunt intracellular HSP72 response following an exercise bout. During recovery from a four-minute high intensity ‘all-out’ bout of exercise, HSP72 is increased by 42% after thirty-minutes post exercise, whilst prior NaHCO3 supplementation blunted HSP72 response to the exercise bout (59). Similar results were observed following a HIIE bout, with HSP72 significantly blunted post exercise with NaHCO3 ingestion compared to a peak 80% increase following exercise under the placebo treatment condition (59). Conversely, NaHCO3 was not found to attenuate HSP72 following a 90-minute sub-maximal intermittent exercise bout, despite a significant increase in HSP72 with time, in treatment and placebo conditions (60). The reason for the contrasting results are unclear, however it may be conceivable that a lower acidic stress from the prolonged sub-maximal bout (pH = 7.31; 60), compared to investigations employing high intensity exercise (pH = 7.16; 62 and pH = 7.15; 59), may be a contributory factor, as the manipulation of the acid-base balance is the mechanism NaHCO3 typically mediates exercise performance. This implies that the expression of HSP’s following high intensity exercise is provoked by substantial disturbance to acid–base balance, although this may not be representative of a direct mechanistic relationship.

The attenuation of oxidative damage was also suggested to coexist with the dampened HSP72 response following exercise observed with pre-exercise NaHCO3 ingestion. Delving deeper, lipid peroxidation was significantly reduced by 67% and 25% between 60 to 90 minutes following a single high intensity and intermittent bout, respectively (62, 59). The authors used Thiobarbituric acid reactive substances (Tbars) to assess lipid peroxidation in this instance, however this method is suggested to be an unreliable method of assessing lipid peroxidation (Powers et al., 2010). Further research is therefore required to determine the effect of NaHCO3 on more reliable markers of oxidative damage. Nonetheless, it unclear if the dampening of acidic stress directly contributes to the blunted HSP72 response or if this is mediated via a reduced oxidative damage.

The biological and practical significance of the blunted HSP72 expression to NaHCO3 prior to high intensity exercise is unclear, although the same is asserted for the overall exercise related implication of HSP’s to health and training adaptation (54). The role in exercise preconditioning is one that is often cited in the literature with the HSP’s deemed to central in protecting the body to exercise stressors following an initial exposure. A recent study by Peart et al., (61) investigated the effect of an initial blunted HSP72 with NaHCO3 following of high intensity exercise had on the subsequent HSP72 expression following a second bout of the same exercise stress without NaHCO3 ingestion. In comparison to placebo, the ingestion of NaHCO­3 prior to the initial exercise stressor had no effect on HSP72 expression during the subsequent exercise stressor. Sodium bicarbonatesupplementationmay not therefore, interfere the exercise preconditioning role of HSP, although the meaningfulness of this observation is not fully understood as further work is required to establish the importance HSP during regular exercise for adaptation and health outcome. This body of research related to NaHCO3­ supplementation does however provide an important insight into the role of acidic stress to cellular stress manifestations during high intensity exercise and also suggests that supplementation may not impede the role of HSP in the conditioning effects of acute exercise.

1. **Summary**

Research published since 2008 on the ergogenic effect of NaHCO3 has demonstrated improvements during high intensity exercise, within a range of exercise modalities, such as a single bout of supramaximal exercise, high intensity intermittent activity and skill based sports (summarized in Table 2). In particular, these benefits seem to be present to a greater extent within trained individuals. Despite this, a high intra-individual variability in response to NaHCO3 appears to exist, therefore the ergogenic benefits may not be induced during every exercise bout. This response however, has only been shown with untrained individuals and further evidence is required to ascertain if this intra-individual response is prevalent within trained individuals. The development of a personalised dosage strategy that enables individuals to exercise at their individualized peak alkalotic condition. This may produce greater performance enhancement and allow individuals to attain the ergogenic benefits of NaHCO3 more consistently. Contemporary research has alluded to benefits of acute NaHCO3 supplementation beyond performance-alone. Pre-exercise alkalosis can alleviate physiological stress during high intensity exercise, particularly in reference to HSP72 response, whilst promising evidence is suggestive of an augmented molecular adaptation response to training.

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