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Background. **To study the plasma ammonia response after sprint crawl swimming.**
Methods. **Nine sprinters (S) and ten non-sprinters (NS) completed a 15-, a 25- and a 50-m crawl at maximal intensity with a 10-min and a 15-min resting period inbetween. Capillary blood samples were collected before and at regular intervals after each effort for plasma ammonia determination.**
Results. **Ammonia kinetics differed among distances, but not between groups, with peak values (observed 2-8 min postexercise) being higher after 50 m as compared to shorter distances. Significant differences between S and NS were found in peak ammonia after 50 m (124.5±58.2 *vs* 98.7±6.3 μmol L^{-1}) and in the change of ammonia relative to swim time ($\Delta NH_3/\Delta t$) after 25 m (2.66±1.87 *vs* 1.49±0.84 μmol L^{-1} s^{-1}) and 50 m (1.87± 1.33 *vs* 1.01±0.49 μmol L^{-1} s^{-1}). $\Delta NH_3/\Delta t$ was highest after 15 m (3.33±2.53 in S, 3.92±1.67 μmol L^{-1} s^{-1} in NS).**
Conclusions. **These differences in the plasma ammonia response to sprint swimming according to duration and athlete seem to be connected to distinctions in muscle fiber profile and energy providing processes.**

KEY WORDS: Ammonia - Exercise physiology - Adenylate metabolism - Swimming.

Plasma ammonia concentration has been known to increase after physical work since 1927.[1 2] Deamination of adenosine monophosphate (AMP) and branched-chain amino acids have been identified as possible sources of this increased ammonia production.[3 4] The former is generally thought to be the prime contributor to ammonia production during short intense exercise through activation of adenylate deaminase whose activity has been found to be higher in fast-twitch as compared to slow-twitch muscle fibers of rat and human.[5-8]

The 50-m crawl became an official event in international competiton in 1986.[9] The best male competitors complete the race in 22-23 s. Since muscle biopsies of crawl sprinters have revealed a higher percentage of fast-twitch fibers as compared to middle- or long-distance swimmers,[10-12] it can be presumed that higher plasma ammonia concentrations would be found in sprinters than in non-sprinters.

There are no published data on plasma ammonia changes after sprint swimming. It was the purpose of this study to measure changes in the plasma ammonia concentration after intense short-term crawl bouts and to investigate differences between sprinters and non-sprinters.

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TABLE I.—*Characteristics of the subjects (mean±SD).*

Parameters	Sprinters (n=9)	Non-sprinters (n=10)
Age (yrs)	22.6±4.0	21.3±3.5
Height (cm)	191.0±8.0	183.3±6.0*
Weight (kg)	85.7±8.6	78.9±7.3
Body fat (%)	12.7±1.4	12.2±1.3
Lean body mass (kg)	75.4±7.4	68.2±6.0*

*) Significantly different from sprinters ($p \leq 0.05$).

TABLE II.—*Swim time and velocity of sprinters and non-sprinters at each crawl bout (mean ±SD).*

Parameters	Sprinters (n = 9)	Non-sprinters (n = 10)
Time (s).		
15 m	6.06±0.22	6.47±0.30
25 m	11.48±0.32	12.31±0.46*
50 m	25.43±0.92	27.4±1.14*
Velocity (ms^{-1})		
15 m	2.48±0.09	2.33±0.12*
25 m	2.18±0.06	2.04±0.08*
50 m	1.97±0.07	1.83±0.07*

*) Significantly different from sprinters ($p \leq 0.001$).

Materials and methods

Subjects

Nineteen club swimmers were included in this study. Nine participated regularly in 50- and 100-m events and were characterised as sprinters (S), whereas ten participated in 200-m or longer lasting events and were characterised as non-sprinters (NS). Their characteristics are listed in Table I. Body fat was estimated from skinfold measurements.[13] Prior to testing every subject was informed about the test procedure and gave written consent.

Test procedure

Each participant completed a 15-m, a 25-m and a 50-m maximal crawl bout by starting from the block. There was a 10-min rest period between the 15-m and 25-m bouts and a 15-min rest period between the 25-m and 50-m bout. Total swim time of each bout and split times at 10, 15, 20 and 25 m (where applicable) were measured by video recording. The split times were determined in order to examine whether initial meters were swum at the same velocity in all three bouts. The same set of crawl bouts was repeated one week later and the fastest of the two sets was analysed for each participant.

Ammonia determination

Blood samples (150 μL) were collected from a hyperhaemised earlobe into a lithium heparin containing monoject samplette (Sherwood, St. Louis, MO) immediately before the beginning and immediately after the end of each crawl bout, as well as 2, 4, 6 and 8 min after the 15-m bout, 2, 4, 6, 8 and 12 min after the 25-m bout and 2, 4, 6, 8, 12, 15 and 20 min after the 50-m bout. The samples were immediately cooled in ice water and centrifuged at 11000×g within 30 min. Plasma was removed and frozen at -20°C within five hours from the time when blood was obtained. On the following day ammonia was measured in a COBAS BIO Analyzer (Roche, Nutley, NJ) based on the enzymatic method of DaFonseca-Wollheim,[14] as adapted to capillary blood by Fischer *et al.* [15] A test kit from Boehringer Mannheim (cat. no. 125857) was used.

Statistical analysis

Significant differences between means were estimated with Mann-Whitney "U"-test and with Newman-Keuls test after initial analysis of variance.

Results

Significant differences in swim times and velocities were detected between S and NS with the exception of the swim time at 15 m (Table II). No differences were found among the 10-m split times of each bout, among the time of the first bout and the 15-m split times of the second and third bouts or between the 20-m split times of the second and third bouts.

The plasma ammonia kinetics after each swim bout are presented in Figure 1. Peak values occured within 2-8 min. Because of big standard deviations in S no significant differences were found between the groups, but the kinetics differed significantly ($p<0.01$) between the 15- and 50-m bouts as well as between the 25- and 50-m bouts.

The peak ammonia concentration increased with distance (Fig. 2) and differed significantly between 15 and 25 m ($p<0.05$), between 15 and 50 m ($p<0.01$) as well as between 25 and 50 m ($p<0.01$). The differ-

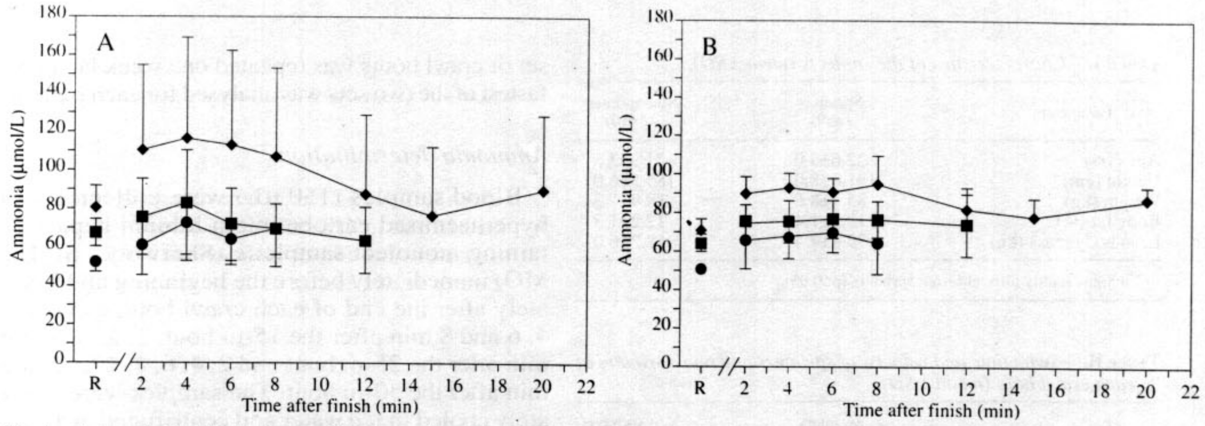

Fig. 1.—Ammonia kinetics in plasma of sprinters (A) and non-sprinters (B) after a 15-m (●), 25-m (■) and 50-m (◆) crawl bout. Resting values (R) were taken immediately before each start. Vertical lines indicate SD.

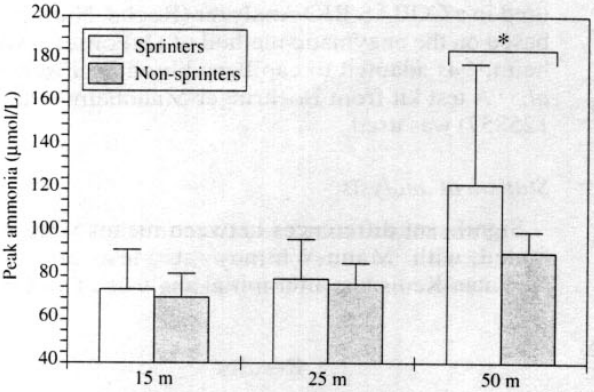

Fig. 2.—Peak plasma ammonia concentrations of sprinters and non-sprinters after a 15-, 25- and 50-m crawl bout. Vertical lines indicate SD and asterisk indicates significant difference between groups (p<0.01). Additionally, differences among bouts are significant (p<0.05).

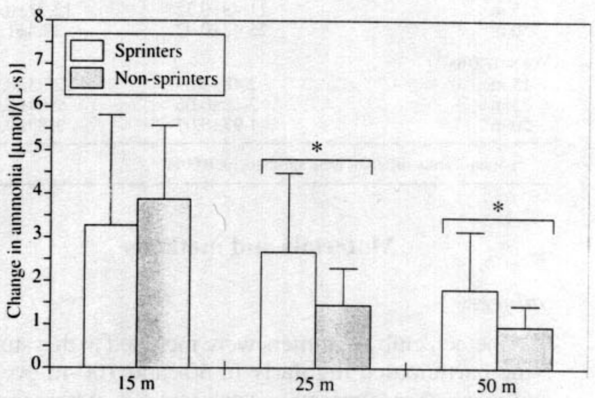

Fig. 3.—Change in plasma ammonia concentrations, from pre-exercise to peak values, relative to swim time of sprinters and non-sprinters after a 15-, 25- and 50-m crawl bout. Vertical lines indicate SD and asterisks indicate significant differences (p<0.01).

ence between the groups was significant ($p<0.01$) only after the 50-m bout. Values of S and NS were, respectively, 74.3±17.1 and 75.2±12.0 µmol L^{-1} after the 15 -m bout, 90.4±12.7 and 83.2±10.6 µmol L^{-1} after the 25-m bout, and 124.5±58.2 and 98.7±6.3 µmol L^{-1} after the 50-m bout.

Figure 3 presents the change in ammonia concentration, from pre-exercise to peak value, relative to swim time ($\Delta NH_3/\Delta t$) for each bout. With increasing distance, $\Delta NH_3/\Delta t$ decreased significantly ($p<0.001$). The difference between the groups was not significant at 15 m (3.33±2.53 in S; 3.92±1.67 µmol L^{-1} s^{-1} in NS), but was significant at 25 m (2.66±1.87 in S *vs* 1.49±0.84 µmol L^{-1} s^{-1} in NS; $p<0.01$) and at 50 m (1.87±1.33 in S *vs* 1.01±0.49 µmol L^{-1} s^{-1} in NS; $p<0.01$). $\Delta NH_3/\Delta t$ at 25 and 50 m was nearly twice as high in S as in NS.

Discussion and conclusions

This study examined the plasma ammonia response after swimming bouts of 6-30 s. Mean peak values ranging from 74 to 125 μmol L^{-1}, found between the 2nd and 8th minute postexercise, are similar to those reported by Schneider *et al.*,[16] Hageloch *et al.* [17] and Snow *et al.* [18] who measured concentrations of 70-120 μmol L^{-1} between the 3rd and 10th minute after a track or bicycle test.

It is well accepted that the increase in ammonia after short-term intense exercise derives from the first branch of the purine nucleotide cycle catalyzed by adenylate deaminase.[18] Factors known to activate the enzyme include ADP and AMP,[19 20] as well as the hydrogen ion.[21 22] Differences in adenylate deaminase between fiber types were presumed by Parnas and Mozolowski,[1] who reported a higher ammonia production in fast white muscle of pigeons, and were demonstrated by Meyer and Terjung [6 7] in rat and by Dudley *et al.* [8] in human.

It has been shown that sprint swimmers possess a higher percentage of type II muscle fibers.[10 11 12 24] These reports can explain our own findings of significantly higher swimming velocities over all distances, peak ammonia concentration after 50 m and $\Delta NH_3/\Delta t$ at 25 and 50 m exhibited by S as compared to NS.

Energy during the first seconds of intense exercise is provided mainly by creatine phosphate degradation accompanied by ammonia accumulation.[25 26] The increase in ADP and AMP due to the high ATP turnover activates adenylate deaminase.[6 8 19 27 28] Application of the metabolism simulation model of Mader *et al.*[29 30] on a 15-m, 25-m and 50-m crawl sprint of male sprinters and non-sprinters with characteristics similar to the subjects of the present study confirmed that ATP turnover dropped with distance in both groups because of the great decrease in creatine phosphate at the very beginning of exercise and the inability of glycolysis to maintain such a high ATP turnover.[31] This state of energy provision at the very beginning of intense exercise may be responsible for the significantly higher $\Delta NH_3/\Delta t$ after 6 s of a (15-m) crawl sprint as compared to 11-12 s (25 m) or 25-27 s (50 m). Assuming that changes in plasma ammonia concentration reflect ammonia production in muscle, the above finding indicates that the rate of ammonia production is higher with shorter all-out bouts. The fast increase in ammonia may help to maintain the ATP-regenerating process via the reaction catalyzed by adenylate kinase [20 27 32] during the first seconds of intense exercise.

In conclusion, we have found higher peak plasma ammonia concentrations (i) after 50 m of sprint swimming as compared to shorter distances and (ii) in sprinters as compared to non-sprinters. Additionally, $\Delta NH_3/\Delta t$ was highest after 15 m and lowest after 50 m of sprint swimming and higher in sprinters than in non-sprinters after 25 and 50 m. Our data indicate that differences in the plasma ammonia response to sprint swimming according to athlete and distance can be explained by distinctions in muscle fiber profile and energy providing processes.

References

1. Parnas JK, Mozolowski W. Über den Ammoniakgehalt und die Ammoniakbildung im Muskel und deren Zusammenhang mit Funktion und Zustandsänderung. Biochem Z 1927;184:399-441.
2. Parnas JK, Mozolowski W. Über den Ammoniakgehalt und die Ammoniakbildung im Blute. IX. Mitteilung: Der Zusammenhang des Blutammoniaks mit der Muskelarbeit. Biochem Z 1927:188:15-13.
3. MacLean DA, Graham TE, Saltin B. Stimulation of muscle ammonia production during exercise following branched-chain amino acid supplementation in humans. J Physiol 1996;493:909-22.
4. vanHall G, MacLean DA, Saltin B, Wagenmakers AJM. Mechanisms of activation of muscle branched-chain a-keto acid dehydrogenase during exercise in man. J Physiol 1996;494:899-905.
5. Lowenstein JM, Tornheim K. Ammonia production in muscle: the purine nucleotide cycle. Science 1971;171:397-400.
6. Meyer RA, Terjung RL. Differences in ammonia and adenylate metabolism in contracting fast and slow muscle. Am J Physiol 1979:237: C111-C118.
7. Meyer RA, Terjung RL. AMP deamination and IMP reamination in working skeletal muscle. Am J Physiol 1980;239:C32-C38.
8. Dudley GA, Staron RS, Murray TF, Hagerman FC, Luginbuhl A. Muscle fiber composition and blood ammonia levels after intense exercise in humans. J Appl Physiol 1983;54:582-6.
9. Fina News; XIII (8). Madrid 1986.
10. Costill DL. Adaptions in skeletal muscle during training for sprint and endurance swimming. In: Eriksson B, Furberg B, editors. Swimming Med 1987;IV(6):233-48.
11. Gerber G, Appelt D, Böhme H, Feustel G, Hotz G, Lorenz R, Reichert M. Biologischer Beitrag zur Erkennung und Erschließung von Reserven im Energiestoffwechsel und in der muskularen Feinstruktur für die Leistungsentwicklung auf den kurzen Wettkampfstrecken im Schwimmen. VD [vertrauliche Dienstsache] 1979;6/38/[19]79/3:1-97.
12. Prins J. Muscles and their function. Swimming Technique 1979;16:57-62.
13. Parizkova J. Impact of age, diet and exercise on man's body composition. Ann NY Acad Sci 1963;110:661-72.
14. Da Fonseca-Wollheim F. Bedeutung von Wasserstoffionenkonzentration und ADP-Zusatz bei Ammoniakbestimmung mit Glutamatdehydrogenase (1. Mitteilung); Direkte Plasmaammoniakbestimmung ohne Enteiweißung (2. Mitteilung). Z klin Chem klin Biochem 1973;11:421-31.
15. Fischer HG, Mader A, Hollmann W. Pilotstudie zur enzymatischen Bestimmung von Ammoniak aus Ohrkapillarblut. Dt Z Sportmed 1990;41:196-9.
16. Schneider S, Hageloch S, Weicker H. Purinnukleotidzyklus und muskulare Ammoniakproduktion. Dt Z Sportmed 1988;39:221-32.
17. Hageloch W, Schneider S, Weicker H. Blood ammonia determination in a specific field test as a method supporting talent selection in runners. Int J Sports Med 1990;11:S56-S61.

18. Snow RJ, McKenna MJ, Carey MF, Hargreaves M. Sprint training attenuates plasma ammonia accumulation following maximal exercise. Acta Physiol Scand 1992;144:395-6.
19. Terjung RL, Tullson PC. Ammonia metabolism during exercise. In: Lamb DR, editor. Energy metabolism in exercise and sport. Brown & Benchmark 1992:235-67.
20. Kalckar HM. The role of myokinase in transphosphorylations. II. The enzymatic action of myokinase on adenine nucleotides. J Biol Chem 1943;148:127-37.
21. Katz A, Sahlin K, Henriksson J. Muscle ammonia metabolism during isometric contraction in humans. Am J Physiol 1986;250:C834-40.
22. Setlow B, Lowenstein JM. Adenylate deaminase. II. Purification and some regulatory properties of the enzyme from calf brain. J Biol Chem 1967;242:607-15.
23. Dudley GA, Terjung RL. Influence of acidosis on AMP deaminase activity in contracting fast-twitch muscle. Am J Physiol 1985;248:C37-C42.
24. Pansold B, Hasart E, Roth W. Genetische und biologisch reaktive Leistungsvoraussetzungen bei Schwimmern unter besonderer Berücksichtigung der Sprintneigung (100 m). VD [Vertrauliche Dienstsache] 1978;Dyn/121/[19]78/12:1-51.
25. Conway EJ, Cooke R. Blood ammonia (LVIII). Biochem J 1939:23:457-78.
26. Spriet L. ATP utilization and provision in fast-twitch skeletal muscle during tetanic contractions. Am J Physiol 1989:257:E595-E609.
27. Atkinson DE. Regulation of enzyme activity. Ann Rev Biochem 1966:35:85-124.
28. Graham TE, Turcotte LP, Kiens B, Richter EA. Effect of endurance training on ammonia and amino acid metabolism in humans. Med Sci Sports Exerc 1997;29:646-53.
29. Mader A. Eine Theorie zur Berechnung der Dynamik und des steady state von Phosphorylierungszustand und Stoffwechselaktivität der Muskelzelle als Folge des Energiebedarfs. Köln 1984 (habilitation).
30. Mader A, Heck H, Hollmann W. A computer simulation model of energy output in relation to metabolic rate and internal environment. In: Knuttgen HG, Vogel JA, Poortmans J, editors. Biochemistry of Exercise. Karger 1983:239-51.
31. Ring S. Energiestoffwechsel im Sprintschwimmen. Köln 1997 (doctoral thesis).
32. Sahlin K, Palmskog G, Hultman E. Adenine nucleotide and IMP contents of the quadriceps muscle in man after exercise. Pflügers Arch 1978;374:193-8.