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ORIGINAL CONTRIBUTION

F. Manz
T. Remer
E. Decher-Spliethoff
M. Höhler
M. Kersting
C. Kunz
B. Lausen

Effects of a high protein intake on renal acid excretion in bodybuilders

Die Wirkung einer proteinreichen Kost auf die renale Säureausscheidung

Zusammenfassung Kraftsportler ernähren sich zur Förderung des Muskelwachstums häufig proteinreich. In dieser Studie wurde die Wirkung einer hohen Proteinzufuhr auf die renale Säurelast und die renale Säureausscheidungskapazität anhand des Vergleichs der Daten der Nährstoffzufuhr und der Harnionogramme von 37 Kraftsportlern mit hoher Proteinzufuhr und 20 jungen Erwachsenen mit normaler Proteinzufuhr (Vergleichsgruppe) untersucht. Die Kraftsportler zeigten eine höhere Energie- (+ 7 %) und Proteinzufuhr (128 vs 88 g/d/1.73 m^2) und eine höhere renale Nettosäureausscheidung (95 vs 64 mmol/d/1.73 m^2) als die Vergleichsgruppe. Die Differenz der Urin pH-Werte der beiden Gruppen war allerdings erstaunlich gering (5.83 vs 6.12). Bei den Kraftsportlern war die renale Ammoniumausscheidung bei allen Urin pH-Werten deutlich höher als bei der Vergleichsgruppe. Eine Regressionsanalyse ergab, daß die Proteinzufuhr das Verhältnis von Urin pH und renaler Ammoniumausscheidung eigenständig zu beeinflussen vermag. Eine vermehrte Proteinzufuhr führt zu einer erhöhten renalen Säurelast und Nettosäureausscheidung. Die gleichzeitig auftretende Steigerung der renalen Säureausscheidungskapazität infolge einer erhöhten Ammoniumproduktion stellt einen sinnvollen Anpassungsmechanismus des Körpers an diese besondere Form der Ernährung dar, der dem Körper eine ausreichende funktionelle Reservekapazität für weitere den Säure-Basen-Haushalt belastende pathophysiologische Ereignisse erhält.

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Prof. Dr. F. Manz (✉) · T. Remer
E. Decher-Spliethoff · M. Höhler
M. Kersting · C. Kunz · B. Lausen
Forschungsinstitut für Kinderernährung
Heinstück 11
44225 Dortmund

Summary Bodybuilders often prefer a high protein diet to achieve maximum skeletal muscle hypertrophy. In this study the effect of a high protein diet on renal acid load and renal handling of proton excretion was studied comparing dietary intake and urinary ionograms in 37 male bodybuilders and 20 young male adults. Energy intake (+ 7 %), protein intake (128 vs 88 g/d/1.73 m^2), and renal net acid excretion (95 vs 64 mmol/d/1.73 m^2) were higher in the bodybuilders than in the controls, however, urine-pH was only slightly lower (5.83 vs 6.12). In the bodybuilders renal ammonium excretion was higher at any given value of urine pH than in the controls. In a regression analysis protein intake proved to be an independent factor modulating the ratio between urine-pH and renal ammonium excretion. The concomitant increase of renal net acid excretion and maximum renal acid excretion capacity in periods of high protein intake appears to be a highly effective response of the kidney to a specific food intake leaving a large renal surplus capacity for an additional renal acid load.

Schlüsselwörter Kraftsportler – hohe Proteinzufuhr – renale Nettosäureausscheidung – Urin pH – Ammoniumausscheidung – Ammoniumproduktion – renale Säureausscheidungskapazität – Sulfat

Key words Bodybuilder – high protein diet – renal net acid excretion – urine-pH – ammonium excretion – ammonia production – sulfate

Introduction

Intake and metabolism of a mixed European diet results in a surplus of protons, which must be excreted by the kidney (16). Usually, acute maximum renal capacity for net acid excretion is much higher than the renal acid load resulting from nutrition and metabolism. However, in preterm infants with a low physiological maximum renal capacity for acid excretion fed infant formulas (12), in infants and children with parenteral nutrition (10), and in infants and children receiving special low phenylalanine preparations (15) the daily load of fixed acids was higher than the acute maximum renal capacity for net acid excretion resulting in the development of metabolic acidosis.

Protein catabolism is an important source of the daily renal acid load, primarily by the production of sulfate. Bodybuilders often prefer a high protein diet to achieve maximum skeletal muscle hypertrophy. In this study the effect of a high protein diet on renal acid load and renal handling of proton excretion was studied comparing dietary intake and urinary ionograms in bodybuilders and in young male adults.

Subjects and methods

Thirty seven male bodybuilders aged between 16–29 years on a high protein diet were investigated clinically. They provided a 7-day weighed diet record and collected two 24-h urine samples on the 6th and 7th days. Twenty healthy young male adults aged between 16–30 years on a normal European diet served as controls. They provided a 3-day weighed diet record and collected one 24-h urine sample on the 3rd day. Detailed anamnestic, nutritional, clinical and biochemical data have been presented elsewhere (11, 12). In the bodybuilders the mean duration of training was about 2 h. (11). Seven bodybuilders used anabolica (11). Here the data of the dietary intake and urinary ionogram of the first day of urine collection in the bodybuilders and the day of urine collection in the controls are presented. Bodybuilders showed a somewhat lower mean height (179.6 ± 7.3 cm vs 182.3 ± 4.7 cm, $p < 0.05$) but a much higher body weight (87.1 ± 13.3 kg vs 72.3 ± 7.8 kg, $p < 0.01$), resulting in a higher body surface area (2.1 ± 0.2 m^2 vs 1.9 ± 0.1 m^2, $p < 0.01$). Therefore the data of dietary intake and urinary excretion rates were adjusted by an estimated standardized body surface area of 1.73 m^2.

Energy and nutrient intake was calculated using the food table of the Institute of Child Nutrition mainly based on data of Souci, Fachmann and Kraut (21). Urinary parameters were assayed by standard methods (20). Renal net acid excretion was estimated as follows (20). According to the principle of electroneutrality, the sum of the cations excreted in the urine equals the sum of the anions. Therefore, renal net acid excretion (titratable acidity + ammonium – bicarbonate) is also the difference of the sum of the remaining important urinary anions chloride, phosphate, sulfate and organic acids minus the sum of the mineral cations sodium, potassium, calcium and magnesium. Renal net acid excretion can thus be estimated from the urinary excretion of electrolytes and organic acids. In steady-state conditions urinary excretion of electrolytes corresponds to their intestinal absorption. Intestinal absorption of electrolytes can be estimated from intake data and average net absorption rates (20). As sodium and chloride data in food tables are incomplete and intake of the latter ions from normal diets (in molar terms) is similar, sodium and chloride were not included in the estimation procedure. Net intestinal absorption of potassium was assumed to be 80 % of the potassium intake. Net calcium (mmol) absorption corresponds to 0.17 x calcium intake + 1.496; net magnesium (mmol) absorption = 0.39 magnesium intake -0.946; net phosphorus (mmol) absorption = 0.642 phosphorus intake -0.355. One gram of protein contains 0.325 mmol of methionine and cysteine. Net intestinal absorption of methionine and cysteine was assumed to be 75 %. Urinary excretion of the organic acids was assumed to be 41 meq/d/1.73 m^2. Estimation was carried out for both groups using mean values of nutrient intake.

For statistical analysis, U-test of Mann-Whitney was used to describe differences between groups. Simple correlations were calculated using Pearsons' correlation coefficient.

Regression models were used to assess the influence of protein intake on urine-pH and renal ammonium excretion. Having fitted different types of model formulae, we decided that a multiplicative model of the ratio of urine-pH and renal ammonium excretion as dependent variable and protein intake and body surface area as independent variables provides the best fit. The model formula is given by:

$$\frac{\text{urine-pH}}{\text{ammonium}} = \frac{e^{\alpha}}{\text{protein intake}^{\beta} \times e^{\gamma} \text{ body surface area.}}$$

Consequently, we analyzed the logarithm of the model formula by ordinary multiple regression.

Results

Intake data of energy and selected nutrients in 37 bodybuilders and 20 male adolescents and young men are listed in Table 1. The bodybuilders show a significantly higher protein intake (+ 45 %) compared to controls. Urine-pH, urinary excretion of creatinine, urea, total nitrogen and the most important cations and anions are

Table 1 Dietary intake of energy and selected nutrients in 37 bodybuilders and 20 young male adults

		Bodybuilders		Controls	P
		mean ± SD	(% of controls)	mean ± SD	
Energy	kcal/d/1.73 m²	2932 ± 841	(107)	2750 ± 686	
Protein	g/d/1.73 m²	128 ± 53	(145)	88 ± 18	< 0.01
Potassium	mmol/d/1.73 m²	102 ± 40	(115)	89 ± 27	
Calcium	mmol/d/1.73 m²	39.7 ± 23	(131)	30.4 ± 9	
Magnesium	mmol/d/1.73 m²	20.8 ± 10	(132)	15.7 ± 4	
Phosphorus	mmol/d/1.73 m²	68.6 ± 29	(129)	53.4 ± 13	

presented in Table 2. In accordance with nutrient intake bodybuilders show a higher urinary excretion of urea, total nitrogen, calcium, magnesium and protons, and slightly lower urine-pH values. The surplus renal net acid excretion of 31 mmol/d/1.73 m^2 of the bodybuilders compared to the data of the controls is the result of an increased urinary excretion of titratable acidity and a decreased excretion of bicarbonate on the one hand (46 %), and an increased urinary excretion of ammonium on the other (54 %). Comparing the ionograms of both groups the additional renal net acid excretion is caused firstly by an increased urinary excretion of sulfate (+ 13.8 meq/d/1.73 m^2) and organic acids (+ 10.3 mmol/d/1.73 m^2) due to increased protein intake and catabolism, and secondly by a different intake and urinary excretion of sodium, potassium, calcium, magnesium, chloride and phosphorus (+ 5.2 meq/d/1.73 m^2).

Furthermore, urinary excretion rates of several cations and anions as well as renal net acid excretion were estimated from the data of dietary intake (Table 2). In both groups the estimated values of calcium and magnesium excretion were slightly higher than the measured values. In bodybuilders urinary excretion of organic acids was somewhat underestimated and urinary excretion of potassium was overestimated. Quantitative underestimation of the difference of renal net acid excretion between the two groups was mainly due to an overestimation of potassium excretion in the bodybuilders.

There was a negative correlation between renal net acid excretion (mmol/d/1.73 m^2) and urine-pH in both the bodybuilders ($p < 0.01$) and the controls ($p < 0.01$) (Fig. 1). Renal ammonium excretion (y, mmol/d/1.73 m^2) was not significantly negatively correlated to urine-pH (x) both in the bodybuilders ($y = -12.4\ x + 131$) and the controls ($y = -4.9\ x + 71.9$). In the multiplicative regression model protein intake and body surface area proved to be independent factors ($p < 0.01$) modulating the ratio between urine-pH and renal ammonium excretion. Thus,

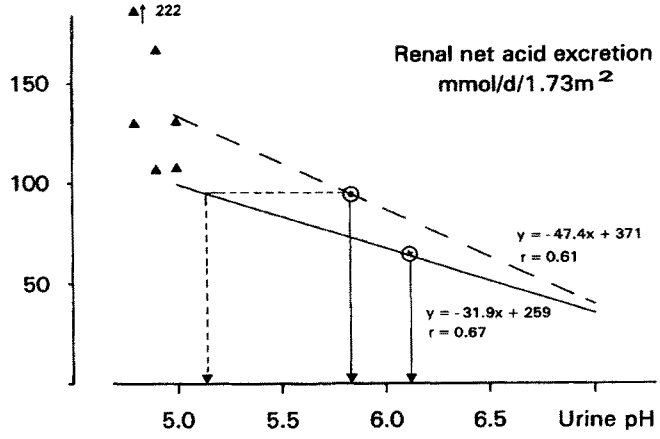

Fig. 1 Renal net acid excretion of 20 young male adults (⊗ mean value, – regression line), 37 male bodybuilders on a high protein diet (⊙ mean value, --- regression line), and 6 groups of healthy adults with acute acid loading tests using ammonium chloride (▲ mean values) (4, 5, 7, 18, 23, 24).

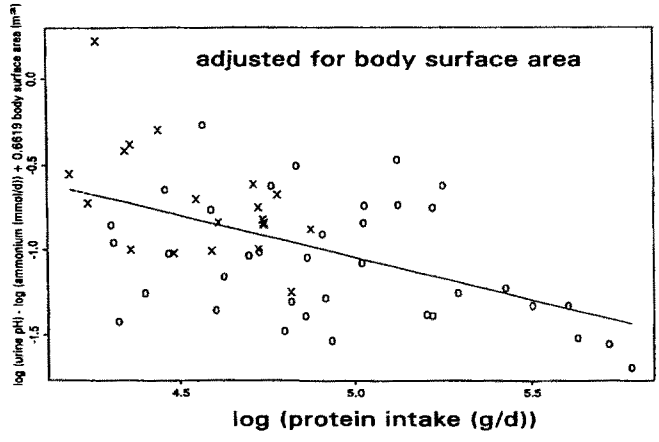

Fig. 2 Protein intake versus ratio between urine pH and renal ammonium excretion adjusted for body surface area ($\gamma = 0.662$). The observations of the 20 young male adults (○), the 37 male bodybuilders (X), and the corresponding regression line (–) are shown ($\alpha = 1.447$, $\beta = 0.498$).

Table 2 Urine-pH, urinary excretion of nitrogen-metabolites and ions and estimation of renal net acid excretion from the dietary intake of 37 bodybuilders and 20 young male adults

		Bodybuilders (n = 37)		Controls (n = 20)		P
		mean ± SD	estimated value[c]	mean ± SD	estimated value[c]	
Urine-pH		5.83 ± 0.42		6.12 ± 0.44		< 0.05
Creatinine	mmol/d/1.73 m^2	17.4 ± 4.1		13.9 ± 3.5		< 0.01
Urea	mmol/d/1.73 m^2	498 ± 194		353 ± 103		< 0.01
Total-nitrogen	mmol/d/1.73 m^2	1237 ± 442		888 ± 229		< 0.01
Chloride	mmol/d/1.73 m^2	148.5 ± 49		128.6 ± 50		
Phosphorus	mmol/d/1.73 m^2	41.6 ± 12	43.7	35.2 ± 8	33.9	
Sulfate	mmol/d/1.73 m^2	29.5 ± 13	31.2	22.6 ± 7	21.5	
Organic acids	meq/d/1.73 m^2	48.4 ± 11	41.0	38.1 ± 10	41.0	
Bicarbonate	mmol/d/1.73 m^2	2.6		3.9		< 0.01
Sum of anions[a]	mmol/d/1.73 m^2	333.4	(182.1)[d]	279.2	(145.0)[d]	
Sodium	mmol/d/1.73 m^2	143.6 ± 46		126.0 ± 55		
Potassium	mmol/d/1.73 m^2	61.8 ± 30	81.6	72.0 ± 28	71.2	
Calcium	mmol/d/1.73 m^2	5.5 ± 3.1	8.2	3.3 ± 1.5	6.7	< 0.01
Magnesium	mmol/d/1.73 m^2	5.2 ± 1.7	7.2	4.3 ± 1.1	5.2	< 0.05
Titratable acidity	meq/d/1.73 m^2	38.7 ± 15		25.8 ± 10		< 0.01
Ammonium	mmol/d/1.73 m^2	58.9 ± 19		42.0 ± 11		< 0.01
Sum of cations[b]	mmol/d/1.73 m^2	324.4	(112.4)[e]	281.0	(95.0)[e]	
Renal net acid excretion	mmol/d/1.73 m^2	95.0 ± 33	69.7	63.9 ± 21	50.0	< 0.01

[a] Cl, HCO_3, OA: mmol/d/1.73 m^2; P: 1.8 x mmol/d/1.73 m^2; SO_4: 2 x mmol/d/1.73 m^2
[b] Na, K, TA, NH_4: mmol/d/1.73 m^2; Ca, Mg: 2 x mmol/d/1.73 m^2
[c] Estimation of urinary excretion of the main ions and net acid is specified in methods
[d] without chloride and bicarbonate
[e] without sodium, titratable acidity and ammonium

protein intake increased the ratio between renal ammonium excretion and urine-pH adjusted for body surface area (Fig. 2).

Discussion

A high protein diet results in a high renal acid load characterized by a high renal net acid excretion. The high renal net acid excretion was however not accompanied by the expected low urine-pH. This was because a high protein intake and catabolism increased not only the absolute amount of net acid excretion but also the renal capacity for net acid excretion.

The increased renal net acid excretion on a high protein diet is the result of an increased urinary excretion of anionic end-products from protein catabolism, i.e. sulfate and organic acids and an altered pattern in the urinary excretion of minerals resulting from a changed intake of minerals associated with the increased protein intake (20). In our group of bodybuilders about 80 % of the additional renal net acid excretion was due to an increased urinary excretion of sulfate and organic acids. The contribution of urinary minerals (PO_4 + Cl - Na - K - Ca - Mg) to the additional renal net acid excretion was rather low. Compared to the data of the controls urinary phosphorus excretion increased by 18 % in the bodybuilders, which can be explained by the close association of protein and phosphorus in many foods. In our group of bodybuilders the mean value of urinary potassium excretion was much lower than our estimated values based on the data of dietary intake and food tables. The low urinary potassium excretion may be caused by a lower potassium content in the protein preparation than stated on the label, a relatively high consumption of canned food with low potassium content and unusually high losses of potassium during food processing. The unexpected low urinary potassium excretion also explains the difference between the analytical and the estimated values of renal net acid excretion. Thus a high protein diet results in a high renal net acid excretion.

Acid loading stimulates the renal mechanisms of proton transport and excretion usually characterized by a decreased level of urine-pH. Figure 1 shows the negative correlation between renal net acid excretion and urine-pH

in our two groups of young adults. Furthermore, the mean maximum renal net acid excretion at the mean minimum urine-pH in six groups of normal adults from the literature during acute acid loading tests with ammonium chloride is shown (4, 5, 7, 18, 23, 24). The data is based on maximum values of net acid excretion and minimum values of urine-pH during short periods between one and several hours. An overestimation can occur when maximum values obtained during a short observation period are extrapolated from μmol/min to mmol/d and are then compared with the original 24-h excretion data. Considering this limitation, our data fits very well with the data from the literature. The bodybuilders showed a mean urine-pH of 5.83 at a mean renal net acid excretion of 95 mmol/d/1.73 m^2. Based on the regression line of the controls this net acid excretion corresponds to a urine-pH of 5.14. The difference in the urinary excretion of phosphate of 6.4 mmol/d/1.73 m^2 between our two groups theoretically corresponds to a difference in the urinary excretion of titratable acidity of 11.5 mmol/d/1.73 m^2. Renal phosphorus output is the primary determinant of titratable acidity. When renal net acid excretion is corrected for the effect of the increased phosphoruria on urinary excretion of titratable acidity, a renal net acid excretion of 83.5 mmol/d/1.73 m^2 would then correspond to an estimated mean urine-pH value of 5.5 in the controls, which is still much lower than the measured mean value of 5.83. This difference points to a different status of stimulation of renal acid excretion mechanisms, and especially to differences in the renal handling of ammonium excretion. In fact, the bodybuilders showed at each level of urine-pH not only a higher urinary net acid excretion but also a higher renal ammonium excretion.

Chronic metabolic acidosis is a well known stimulus to increase renal ammonium excretion at any given value of urine-pH (8). In both groups stimulation of renal acid excretion was in the normal range well below the acute maximum renal net acid excretion. Thus, we assume that our bodybuilders had normal acid-base status in the blood and the increased urinary excretion of ammonium is not caused by acidosis, but rather by other factors. It is our hypothesis that protein intake may be such a factor modulating the urinary excretion of ammonium at any given value of urine-pH. The results of our multiple regression analysis well support this hypothesis. Findings corresponding to ours have been seen recently in a controlled diet study using healthy adults (19). Urinary ammonium excretion was lower when protein from eggs was replaced by an equivalent amount of L-methionine resulting in the same additional renal acid load.

In the literature there are many references to the direct effect of protein intake on renal ammonium excretion. However, only few results exist regarding the effect of protein intake on renal ammoniagenesis (independent from acid base metabolism) and thus on renal ammonium excretion capacity. Intake of extra soy protein or meat in adult volunteers increased renal net acid excretion, however "urine-pH remained essentially unchanged" (14). Premature infants fed a high protein formula showed a higher maximum renal ammonium excretion during acute acid loading with ammonium chloride than prematures fed a low protein diet (35 vs 17 μmol/min/1.73 m^2) (3). The difference in maximum ammonium excretion could not be attributed to blood amino acid concentrations. The authors suggested that the high protein intake accelerated the rate of maturation of renal function. On the other hand, Svenningsen and Lindquist observed no increase of maximum renal ammonium excretion capacity in relation to protein intake in preterm infants, although there was a positive correlation between maximum renal ammonium excretion and postnatal age (22). In healthy adults acute acid loading studies showed a large range of maximum renal ammonium excretion of between 42–81 μmol/min suggesting additional factors modulating renal ammonium excretion (4, 5, 7, 18, 23, 24). In adult patients with moderate to severe protein malnutrition baseline excretion of ammonium and maximum renal ammonium excretion during acid loading with ammonium chloride were much lower in the protein-depleted than in the protein-repleted state despite the presence of a higher degree of metabolic acidosis in the malnourished state (13). The authors suggest that the impaired adaptation in ammonia production during protein malnutrition may relate either to decreased substrate delivery or decreased renal gluconeogenesis.

In a high protein diet the increased renal capacity of ammonium excretion could theoretically be caused by an increased ammonium production or a facilitated transfer of ammonium to the collecting duct lumen (2, 6, 9). In dogs infusion of glutamine to raise the plasma glutamine concentration resulted in an increased rate of glutamine extraction and ammonium production in the kidney (9). Therefore, variations of circulating glutamine concentration within the physiological range seem to have an important influence on the steady-state rate of renal glutamine metabolism and thus on urinary excretion of ammonium. In the postabsorptive state of young adult men receiving low, adequate or high amounts of protein plasma concentrations and flux rates of glutamate and glutamine varied inversely with protein intake (17). The overall effect was however small if the effect of the dietary manipulation is compared to the effect of a modest increase in cortisol which elicits a four- to fivefold greater response (17). Thus, the modulating effect of protein intake on renal ammonium production probably is not mediated by changes of plasma glutamine concentration. Energy metabolism may limit the rate of glutamine metabolism and ammoniagenesis (9). The major determinant of energy metabolism of the proximal tubule is the rate of sodium reabsorption which is directly related to the glomerular filtration rate. High protein intake results in an increased glomerular filtration rate (1) and thus may increase glutamine degradation. In our group of

bodybuilders mean creatinine clearance was 154 ml/min/1.73 m^2, or 40 % above the normal level of creatinine clearance of about 110 ml/min/1.73 m^2. The higher creatinine clearance of the bodybuilders fits well to the higher urinary ammonium excretion (+ 40 %). Other factors enhancing ammoniagenesis like hypokalemia are unlikely to be associated with a high protein intake (9). In addition, there are no reasons why a high protein intake might facilitate the ammonium transfer to the collecting duct (6). Thus, the specific effect of protein intake on renal ammoniagenesis seems to be elicited by variations of glomerular filtration rate.

The concomitant increase of renal net acid excretion and maximum renal acid excretion capacity during periods of high protein intake appears to be a highly effective adaptive response of the kidney to a specific and still physiological pattern of food intake leaving a large renal surplus capacity for an additional renal acid load.

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