

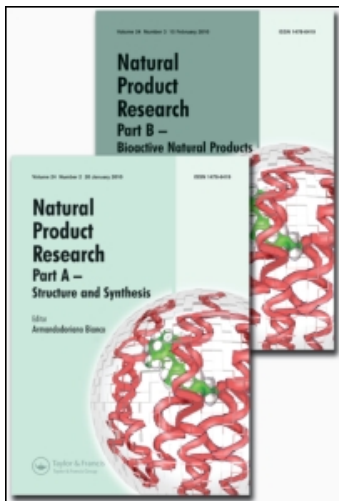
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Effect of amaranth seeds (*Amaranthus cruentus*) in the diet on some biochemical parameters and essential trace elements in blood of high fructose-fed rats

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SHORT COMMUNICATION

Effect of amaranth seeds (*Amaranthus cruentus*) in the diet on some biochemical parameters and essential trace elements in blood of high fructose-fed rats

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The effect of amaranth seeds on the lipid profile, glucose level, protein metabolism and selected trace element (Na, K, Ca and Mg) levels were determined in high-fructose fed Wistar rats. Fructose addition to rat fodder caused changes mainly in the blood lipid profile, particularly manifesting in an increased triglyceride level within all subsequent pairs of rat groups, and ranged between 85% and 112%. Administration of amaranth seeds to rats did not inhibit the increase of triglyceride induced by fructose. There was an increase in glucose concentration of between 3% and 14%. Uric acid concentrations also increased in all groups (30–37%), while changes in creatinine levels were varied. Fructose addition to fodder also brought about a significant decrease in alkaline phosphatase activity (9–20%).

Keywords: amaranth seeds; fructose; rats; biochemical parameters; trace element

1. Introduction

Many authors found that the administration of fodders enriched with fructose to rats induced oxidative stress, leading to hypertriglyceridemia, insulin resistance and obesity (Ackerman et al., 2005; Gaby, 2005; Girard, Madani, El Boustani, Belleville, & Prost, 2005). It was demonstrated that in rats fed a high-fructose diet, free radical concentration was three times higher than that of the control group. Moreover, fructose addition brought about a lowering of vitamin E level, which could be the reason for body defence impairment against free radicals. The consumption of fructose in humans has been increasing for a long time, but there is little evidence that fructose could influence carbohydrate and lipid metabolism or that it is associated with metabolic abnormalities in humans and animals (Basciano, Federico, & Adeli, 2005; Busserolles, Gueux, Rock, Mazur, & Rayssiguier, 2002; Isganaitis & Lustig, 2005; Kizhner & Werman, 2002).

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Amaranth seeds (*Amaranthus cruentus*) contain significant amounts of antioxidant phytochemicals, including flavonoids, phenolic acids, squalene, fat-soluble vitamins, fatty acids, trace elements and other compounds which could change the antioxidant status of an organism (Gorinstein et al., 2007; Paško, Sajewicz, Gorinstein, & Zachwieja, 2008; Tikekar, Ludescher, & Karwe, 2008). Until now, only a few reports have dealt with the influence of protein from seeds or extruded amaranth on lipid metabolism (Berger et al., 2003; Czerwiński et al., 2004; Escudero, Zirulnik, Gomez, Mucciarelli, & Gimenez, 2006; H. Kim, M. Kim, & Shin, 2006; Plate & Areas, 2002).

The aim of this study was to assess the impact of fructose addition on the biochemical parameters of blood on the background of two doses of amaranth in rat fodder. An experimental model adding 31% fructose was applied purposefully to induce oxidative stress. The following plasma parameters were taken into consideration: lipid profile, total cholesterol, high-density lipoproteins (HDLs), low-density lipoproteins (LDLs), triglycerides, glucose, urea, uric acid, creatinine, albumins, total protein concentrations and alkaline phosphatase activity. Concentrations of sodium, potassium, magnesium, calcium and chloride were also determined.

2. Results and discussion

The results of biochemical parameters for every group are given in Supplementary Figure S1 and Supplementary Table S1 (both online only). The most significant changes were reported in the lipid profile (Figure S1). Amaranth seeds significantly decreased the level of total cholesterol in comparison to the control group (C vs. AML; C vs. AMH). High dietary fructose did not influence the total cholesterol level in each group. LDL level was significantly decreased in groups fed either diet with amaranth seeds as compared with the control group (C vs. AML; $p < 0.05$ and C vs. AMH; $p < 0.05$). LDL level in fructose-fed groups was lower in comparison to non-fructose-fed ones. Furthermore, a significant difference was noted between groups C and CF ($p < 0.01$). While the HDL level in the control group was significantly decreased by fructose, addition of amaranth seeds to both groups' diets did not cause the HDL level to decrease. Triglyceride levels significantly decreased after adding amaranth seeds to the diet (C vs. AML; $p < 0.05$). In all fructose-fed groups versus groups fed diets without fructose, a significant increase of triglyceride concentration was registered ($p < 0.01$).

Plasma glucose level (Table S1) rose in fructose-fed groups, and there was statistically significant difference between groups AMHF versus AMH ($p < 0.04$) and AMLF versus AML ($p < 0.004$). In control groups, this increase was not significant. When we compared amaranth-fed groups with control groups, we could see that only amaranth in a higher dose (310 g amaranth seeds/kg fodder) significantly decreased the glucose level (C vs. AMH; $p < 0.02$).

The effects of fructose feeding and amaranth seeds on protein metabolism (urea, creatinine) and uric acid were diverse (Table S1). Urea concentration was unchanged in the control group as a result of fructose addition to the fodder, but decreased insignificantly in groups AML and AMH as a result of amaranth addition to the diet. The dietary fructose brought about an increase in uric acid concentration regardless of the amaranth dose. However, a statistically significant difference was

noted in the control groups CF versus C ($p < 0.001$) and between groups AMLF and AML ($p < 0.04$). Fodder enriched with amaranth seeds at a higher dose (310 g amaranth seeds/kg fodder) increased the uric acid level, as compared with the addition of a lower dose of amaranth seeds (155 g amaranth seeds/kg fodder).

In all fructose-fed groups, a decrease in the level of creatinine was registered; however, it was significant only in groups AMLF versus AML and AMHF versus AMH ($p < 0.05$).

Adding amaranth seeds (independently of dose) to the diet caused a significant increase in the creatinine level (C vs. AML; $p < 0.006$ and C vs. AMH; $p < 0.03$).

In fructose-fed rats, alkaline phosphatase activity was significantly lower than in the non-fructose groups, except for higher amaranth groups (Table S1). No influence of fructose on total protein and albumin concentrations was observed (Table S1). Amaranth seeds progressively decreased plasma total protein level in rats ($p < 0.01$) by 4% and 9% for low and high doses, respectively.

The addition of fructose or amaranth seeds to the diet did not change the levels of Na and K, but fructose significantly increased magnesium and calcium levels in the lower amaranth groups ($p < 0.05$). Chloride levels progressively decreased ($p < 0.01$) by 1–3% after adding amaranth to the diet.

To date, most of the studies on diets enriched in fructose have focused on effects induced by very high content of dietary fructose (Lewis et al., 2004; Wu et al., 2004). In animal studies, fructose was a source of 45–66% of the fodder's energetic value and in human studies the value even rose to 90% (Hellerstein, 2002). Fructose content in experimental diets was considerably higher than in a regular diet (i.e. fructose in fact comprises about 10–20% of food/caloric intake for average Americans (Benado et al., 2004)). In our study, the addition of 31% fructose was applied in order to induce oxidative stress. According to reports, long-lasting fructose consumption may cause adaptive changes in healthy animals, which may result in the masking of such disturbances as those in lipid metabolism (Stark, Timar, & Madar, 2000). Therefore, we performed short-term experiments. Our results concerning the impact of fructose addition on LDL cholesterol level differ considerably from the results of the study by Benado et al. (2004).

The higher content of dietary fructose caused a decrease in plasma LDL cholesterol level (Figure S1). Lewis et al. (2004) reported that feeding rats a diet containing 60% fructose for 5 weeks caused a large rise in LDL level in comparison to the control group fed on a standard diet. The discrepancy with our observations could be the result of the nutritional model applied (31% of fructose). Total cholesterol and LDL levels were decreased significantly in groups fed on both of the diets containing amaranth seeds as compared to the control group (Figure S1). Similar observations of LDL levels caused by an amaranth protein concentrate were reported by Escudero et al. (2006).

Our results showed that amaranth seeds added to the rats' diet inhibited each significant decrease in HDL level caused by the fructose in the control group, but it did not stimulate any increase in HDL concentration (Figure S1). Escudero et al. (2006) showed that an amaranth protein concentrate significantly increased HDL level in rats fed with a casein diet. Lewis et al. (2004) observed higher HDL cholesterol concentration in fructose-fed rats in reference to the control group. Such results were also obtained by Benado et al. (2004) and are in opposition to our results.

Seeds of amaranth in both doses caused, as in Escudero et al. (2006), a significant decrease in triglyceride level in comparison to the control group. Just as in our research, Busserolles et al. (2003) found that a fructose-rich diet induced hypertriglyceridemia in rats after 2 weeks of experiment. There was a two-fold increase in plasma triglyceride concentration compared with the control group. The fructose content in the diet applied in the above study was 34%, similar to our model. Lewis et al. (2004), in their research on rats, fed a diet with a high fructose level (60%) and also reported an increase in triglyceride concentration in comparison to the control group. The above-mentioned changes could be caused by overproduction of triglycerides in hepatocytes as a consequence of the fructose, and therefore increased lipogenesis and overproduction of VLDL.

Our results are consistent with results of other studies that indicate an increase in triglycerides and lipid peroxides, in spite of the fact that fructose in the dose used in our experiment was not a very strong pro-oxidant agent.

It is thought that fructose consumption leads to a significant increase in blood glucose level. We also found this in most of the fructose-fed groups in our experiment (Table S1). When we compared amaranth-fed groups with the control group, we could observe that only amaranth in higher doses significantly decreased the level of glucose. However, in the presence of a fructose in diet, amaranth seeds did not protect the rat against an increase in glucose level caused by fructose.

Some previous studies have shown that fructose did not affect the blood glucose level (Nakamura, Yamagishi, Matsui, & Inoue, 2005), but brought about only temporary (Thorburn et al., 1989) or moderate hyperglycaemia (Anurag & Anuradha, 2002). Such diversity of results could be caused by differences in the amount of fructose intake and the duration of the experiments.

The increased level of plasma urea and uric acid shown in our experiment (Table S1) has been described in literature previously (Yamamoto et al., 1999). This effect could be explained by the fact that fructose metabolism in the kidneys consumes ATP as the source of phosphates for the phosphorylation process and this leads to degradation of purine and pyrimidine nucleotides. In human research, a significant increase of urea and purine base (xanthine and uric acid) concentrations was demonstrated after intravenous injection of 10% fructose solution (0.7 g fructose/kg body weight) (Yamamoto et al., 1999). The increase in concentration of uric acid could be also explained as a rise in body purine catabolism due to fructose. An increased uric acid level in consequence can enhance the antioxidative protection of organism, since this compound is known as one of the strongest water-soluble antioxidants. However, we cannot also exclude the pro-oxidative activity of uric acid.

The high-fructose diet had various impacts on creatinine concentration. Creatinine is produced as a product of phosphocreatine degradation and is excreted into urine. If kidneys are not able to eliminate protein metabolic by-products, the level of plasma creatinine increases. We suppose that the kidneys of most of the animals in the experiment worked properly, and this is the reason for no significant changes in creatinine level between fructose-fed and fructose-free animals. Similar results were achieved by Kizhner and Werman (2002).

To the best of our knowledge, there is no relevant reference in the literature regarding a decrease in alkaline phosphatase activity as was observed in our

experiment in all groups. This enzyme is nonspecific and the interpretation of the results is complex.

3. Conclusion

Fructose feeding (31% w/w of diet) negatively affected the lipid profiles in the plasma of rats. Fructose addition to the fodder of the rats caused changes mainly in the blood lipid profiles, especially increasing triglyceride levels. In all groups affected by dietary fructose, the enhanced uric acid concentration proved to be a metabolic disturbance, while the decrease in alkaline phosphatase activity seems to be a new observation associated with the influence of fructose on the rat organism. The doses of amaranth seeds used in our model did not inhibit the increase of triglycerides caused by fructose.

Supplementary material

Experimental details relating to this paper are available online, alongside Figure S1 and Tables S1 and S2.

Acknowledgements

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References

- Ackerman, Z., Oron-Herman, M., Grozovski, M., Rosenthal, T., Pappo, O., Link, G., & Sela, B.A. (2005). Fructose-induced fatty liver disease. Hepatic effects of blood pressure and plasma triglyceride reduction. *Hypertension*, *45*, 1012–1018.
- Anurag, P., & Anuradha, C.V. (2002). Metformin improves lipid metabolism and attenuates lipid peroxidation in high fructose-fed rats. *Diabetes Obesity and Metabolism*, *4*, 36–42.
- Basciano, H., Federico, L., & Adeli, K. (2005). Fructose, insulin resistance, and metabolic dyslipidemia. *Nutrition and Metabolism*, *2*, 1–14.
- Benado, M., Alcantara, C., De la Rosa, R., Ambrose, M., Mosier, K., & Kern, M. (2004). Effects of various levels of dietary fructose on blood lipids of rats. *Nutrition Research*, *24*, 565–571.
- Berger, A., Gremaud, G., Baumgartner, M., Rein, D., Monnard, I., Kratky, E., . . . , Lambelet, P. (2003). Cholesterol – lowering properties of amaranth grain and oil in hamsters. *International Journal for Vitamin and Nutrition Research*, *73*, 39–47.
- Busserolles, J., Gueux, E., Rock, E., Demigne, Ch., Mazur, A., & Rayssiguier, Y. (2003). Oligofructose protects against the hypertriglyceridemic and pro-oxidative effects of a high fructose diet in rats. *The Journal of Nutrition*, *133*, 1903–1908.
- Busserolles, J., Gueux, E., Rock, E., Mazur, A., & Rayssiguier, Y. (2002). Substituting honey for refined carbohydrates protects rats from hypertriglyceridemic and prooxidative effects of fructose. *The Journal of Nutrition*, *132*, 3379–3382.
- Czerwiński, J., Bartnikowska, E., Leontowicz, H., Lange, E., Leontowicz, M., Katrich, E., . . . , Gorinstein, S. (2004). Oat (*Avena sativa* L.) and amaranth (*Amaranthus hypochondriacus*) meals positively affect plasma lipid profile in

- rats fed cholesterol-containing diets. *The Journal of Nutritional Biochemistry*, *15*, 622–629.
- Escudero, N.L., Zirulnik, F., Gomez, N.N., Mucciarelli, S.I., & Gimenez, M.S. (2006). Influence of a protein concentrate from *Amaranthus cruentus* seeds on lipid metabolism. *Experimental Biology and Medicine*, *231*, 50–59.
- Gaby, A.R. (2005). Adverse effects of dietary fructose. *Alternative Medicine Review*, *10*, 294–306.
- Girard, A., Madani, S., El Boustani, E.S., Belleville, J., & Prost, J. (2005). Changes in lipid metabolism and antioxidant defense status in spontaneously hypertensive rats and Wistar rats fed a diet enriched with fructose and saturated fatty acids. *Nutrition*, *21*, 240–248.
- Gorinstein, S., Vargas, O.J.M., Jaramillo, N.O., Salas, I.A., Ayala, A.L.M., Arancibia-Avila, P., . . . , Trakhtenberg, S. (2007). The total polyphenols and the antioxidant potentials of some selected cereals and pseudocereals. *European Food Research and Technology*, *225*, 321–328.
- Hellerstein, M.K. (2002). Carbohydrate-induced hypertriglyceridemia: modifying factors and implications for cardiovascular risk. *Current Opinion in Lipidology*, *13*, 33–40.
- Isganaitis, E., & Lustig, R.H. (2005). Fast food, central nervous system insulin resistance and obesity. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *25*, 2451–2462.
- Kim, H.K., Kim, M.J., & Shin, D.H. (2006). Improvement of lipid profile by amaranth (*Amaranthus esculantus*) supplementation in streptozotocin-induced diabetes rats. *Annals of Nutrition and Metabolism*, *50*, 277–281.
- Kizhner, T., & Werman, M.J. (2002). Long-term fructose intake: biochemical consequences and altered renal histology in the male rat. *Metabolism*, *51*, 1538–1547.
- Lewis, G.F., Murdoch, S., Uffelman, K., Naples, M., Szeto, L., Albers, A., . . . , Brunzell, J.D. (2004). Hepatic lipase mRNA, protein, and plasma enzyme activity is increased in the insulin-resistant, fructose-fed Syrian golden hamster and is partially normalized by the insulin sensitizer rosiglitazone. *Diabetes*, *53*, 2893–2900.
- Nakamura, K., Yamagishi, S., Matsui, T., & Inoue, H. (2005). Acarbose, an alpha-glucosidase inhibitor, improves insulin resistance in fructose-fed rats. *Drugs Under Experimental and Clinical Research*, *31*, 155–159.
- Paško, P., Sajewicz, M., Gorinstein, S., & Zachwieja, Z. (2008). Analysis of selected phenolic acids and flavonoids in *Amaranthus cruentus* and *Chenopodium quinoa* seeds and sprouts by HPLC. *Acta Chromatographica*, *20*, 661–672.
- Plate, A.Y., & Areas, J.A. (2002). Cholesterol-lowering effect of extruded amaranth (*Amaranthus cruentus* L.) in hypercholesterolemia rabbits. *Food Chemistry*, *76*, 1–6.
- Stark, A.H., Timar, B., & Madar, Z. (2000). Adaptation of Sprague Dawley rats to long-term feeding of high fat or high fructose diets. *European Journal of Nutrition*, *39*, 229–234.
- Thorburn, A.W., Crapo, P., Beltz, W., Wallace, P., Wilztium, J., & Henry, R. (1989). Lipid metabolism in non-insulin-dependent-diabetes: effects of long term treatment with fructose-supplemented mixed meals. *The American Journal of Clinical Nutrition*, *50*, 1015–1022.
- Tikekar, R.V., Ludescher, R.D., & Karwe, M.V. (2008). Processing stability of squalene in amaranth and antioxidant potential of amaranth extract. *Journal of Agricultural and Food Chemistry*, *56*, 10675–10678.
- Wu, L.Y., Juan, Ch.Ch., Hwang, L.S., Hsu, Y.P., Ho, P.H., & Ho, L.T. (2004). Green tea supplementation ameliorates insulin resistance and increases glucose transporter IV content in a fructose-fed rat model. *European Journal of Nutrition*, *43*, 116–124.
- Yamamoto, T., Moriwaki, Y., Takahashi, S., Tsutsumi, Z., Yamakita, J.I., & Higashino, K. (1999). Effects of fructose and xylitol on the urinary excretion of adenosine, uridine, and purine bases. *Metabolism*, *48*, 520–524.

SUPPLEMENTARY MATERIAL

Effect of amaranth seeds (*Amaranthus cruentus*) in diet on some biochemical parameters and essential trace elements in blood of high fructose-fed rats

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The effect of amaranth seeds on the lipid profile, glucose level, protein metabolism and selected trace elements (Na, K, Ca, Mg) level were determined in high-fructose fed Wistar rats. Fructose addition to rat fodder caused changes mainly in the blood lipid profile, particularly manifesting in an increased triglyceride level within all subsequent pairs of rat groups, and ranged between 85 and 112%. Amaranth seeds administration to rats did not inhibit the increase of triglyceride induced by fructose. There was an increase in glucose concentration of between 3% and 14%. Uric acid concentrations also increased in all groups (30–37%), while changes in creatinine levels were varied. Fructose addition to fodder also brought about a significant decrease in alkaline phosphatase activity (9–20%).

Keywords: amaranth seeds; fructose; rats; biochemical parameters; trace element

Experimental

Plant material

Amaranth seeds (*Amaranthus cruentus*) were cropped in the eastern Poland (Tomaszow Lubelski) and identified by Prof. Wolski A., Szarlat Co., Łomża, Poland. A voucher specimen (# AC/PP/PL 1020) was deposited in the Plant Breeding and Acclimatization Institute – National Research Institute and in the Department of Food Chemistry and Nutrition, Medical College Jagiellonian University.

Preparation of rat fodder

Diets were formulated according to a following scheme (Table S2): the compounds were added in varied amounts, depending on the type of diet: cornstarch, fructose and antioxidative active products (amaranth seeds). Common compounds in constant amounts [g/kg fodder] are given in the legend to Table S2.

Animals

Male Wistar rats (mean weight 245.4 ± 7 g) were purchased from the Animal House of Jagiellonian University. The rats were housed in metal–plastic cages (three animals per cage, six animals in each particular group: C, CF, AML, AMLF, AMH, AMHF; see Table S2) and kept in an air-conditioned animal-room at a temperature of $22 \pm 2^\circ\text{C}$, with a relative humidity of $50 \pm 5\%$. The animal room was on a 12 h daily lighting period cycle and the rats were kept for five weeks. The rats had unlimited access to fodder and tap water. The protocols for the animal experiments were approved by the Animal Experimentation Committee of Jagiellonian University.

Sample collections and analytical procedure

Blood samples were taken from aorta under general anaesthesia following intraperitoneal thiopental injection. Rat plasma biochemical parameters were determined by using the Konelab 30 automatic analyser.

Statistical procedure

Values are given as mean standard deviation (SD). The statistical analyses of biochemical parameters were conducted using the Statistica 6.1 PL software (StatSoft, Inc.). A type of distribution for analysed variables was determined by chi-square test. A variance homogeneity was analysed using the Hartley test. In order to compare mean values, the one way ANOVA test was applied. The critical significance level was set as $p < 0.05$. The Kruskal–Wallis test was applied to check for statistical evaluations of antioxidant parameters, followed by the Dunn post hoc test.

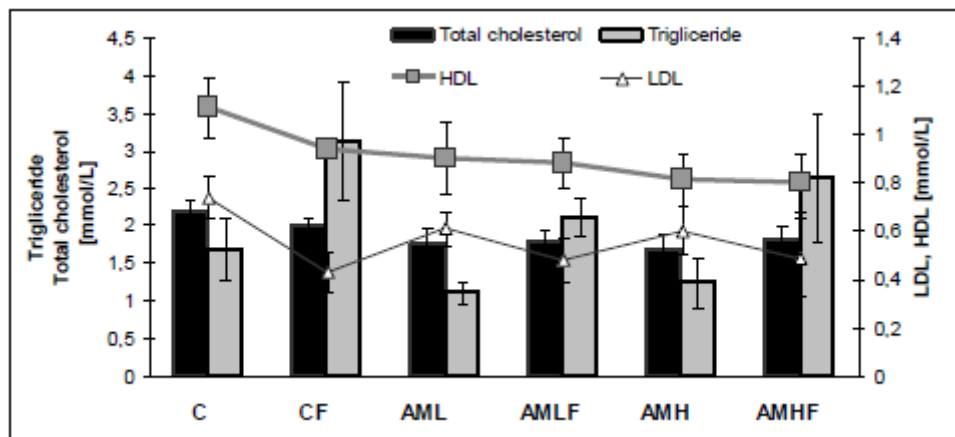


Figure S1. Effects of fructose and amaranth seeds on lipid profile in blood of rats.

Notes: Values are given as mean SD ($n = 6$). C: Control group; CF: control group with 31% of fructose; AML: group fed diet with amaranth seeds (155 g/kg fodder); AMLF: group fed diet with amaranth seeds (155 g/kg fodder) and with 31% of fructose; AMH: group fed diet with amaranth seeds (310 g/kg fodder); AMHF: group fed diet with amaranth seeds (310 g/kg fodder) and with 31% of fructose.

Table S1. Effects of fructose and amaranth seeds on levels of glucose, creatinine, urea, uric acid, total protein, albumin, alkaline phosphatase activity and selected trace elements in rat blood.

	C	CF	AML	AMLF	AMH	AMHF	Statistical significance
Glucose [mmol/L]	9.15±0.55	9.42±0.84	8.70±0.33	9.60±0.50	8.30±0.44	9.52±0.99	AMHF vs. AMH; $p<0.04$ CF vs. C; $p<0.004$
Creatinine [μ mol/L]	32.2±3.3	29.5±3.8	39.5±3.9	35.3±2.1	36.5±2.5	32.5±3.2	C vs. AMH; $p<0.02$ AMLF vs. AML; $p<0.05$ AMHF vs. AMH; $p<0.05$ C vs. AML $p<0.006$ C vs. AMH $p<0.03$
Urea [mmol/L]	10.8±0.6	10.9±1.4	9.1±1.6	7.8±1.4	8.8±1.4	7.8±0.9	
Uric acid [mg/L]	145±23	189±33	150±11	203±57	176±23	241±98	CF vs. C; $p<0.001$ AMLF vs. AML; $p<0.04$
Total protein [g/L]	62.3±1.2	62.6±2.3	59.7±1.7	61.2±3.2	56.9±1.8	61.0±3.3	
Albumin [g/dL]	35.3±2.4	34.2±1.2	32.2±1.3	32.0±1.5	32.0±0.9	32.8±0.8	
Alkaline phosphatase [U/L]	239.5±26.1	190.5±13.4	240.3±23.5	193.5±15.6	242.5±36.9	219.8±27.9	CF vs. C; $p<0.05$ AMLF vs. AML; $p<0.05$
Na [mmol/L]	139.5±.8	140.0±1.5	139.0±.6	139.3±.8	138.8±.8	139.0±1.1	
K [mmol/L]	3.48±0.10	3.53±0.21	3.48±0.10	3.45±0.33	3.60±0.26	3.42±0.15	
Mg [mmol/L]	0.77±0.14	0.77±0.06	0.74±0.05	0.86±0.06	0.83±0.03	0.86±0.08	AMLF vs. AML; $p<0.05$
Ca [mmol/L]	2.50±0.04	2.47±0.07	2.52±0.03	2.59±0.06	2.50±0.05	2.49±0.02	AMLF vs. AML; $p<0.05$
Cl [mmol/L]	102.6±1.2	103.3±1.4	101.7±0.9	101.5±0.6	99.6±1.0	100.7±1.7	AMH vs. C; $p<0.01$

Note: Values are given as mean±SD ($n = 6$).

Table S2. Fodder ingredients [g/kg].

Group	Name of group	Corn starch	Fructose	Material (amaranth seeds)
C	Control group*	620	0	–
CF	Control group * +fructose	310	310	–
AMH	Amaranth seeds	310	0	310
AMHF	Amaranth seeds +fructose	0	310	310
AML	Amaranth seeds	465	0	155

Notes: All fodder contained (in g/1000 g): casein 200, rapeseed oil 50, chalk 28, calcium monophosphate 29, lecithin 10, sodium chlorate 3, cellulose 50, mixture of vitamins and microelements 10 (Premix LPM, BASF, Poland – vitamins and minerals). *With additional amount of potassium (K_2SO_4 – 3.5g) and magnesium (MgO – 0.7 g) in cost of cellulose content.