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EFFECT OF DIETARY ACIDIFIER ON GROWTH, MORTALITY, POST-SLAUGHTER PARAMETERS AND MEAT COMPOSITION OF BROILER CHICKENS*

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Abstract

An experiment with 608 broiler chickens was conducted to investigate the effect of dietary acidifier level on body weight, feed consumption and conversion, mortality, dressing percentage, postmortem carcass traits, tissue composition of breast and leg muscles, and plasma chemical parameters. Feeding the acidifier to chickens at 3, 6 and 9 g/kg of the diet reduced the pH of starter and grower diets from 6.90 to 5.89, and from 6.28 to 5.73, respectively. Compared to the control group, dietary acidification significantly increased body weight of chickens by 6.2, 8.2 and 8.2% at 21 days of age, and by 2.7, 3.6 and 3.7% at 42 days of age, respectively (P<0.01). Mortality decreased from 2.58% in the control group to 0.00–0.59% in the experimental groups (P<0.01). Acidification of the diets increased EEI-index from 327 (control group) to 348 points in the experimental group supplemented with 9% (9 g/kg) acidifier, but had no significant effect on feed consumption and feed conversion ratio among treatments. The relative weight of breast and leg muscles, gizzard, liver and carcass depot fat was not affected by dietary treatments. Breast muscles represented 27.7% (control group) and 27.9% (experimental groups) of the carcass weight. Leg muscles made up 21.5% and 20.7% of the carcass weight, respectively. There were no significant differences in chemical composition of breast and leg muscles, including dry matter, protein and fat content. No significant differences between the control and experimental chickens were noted for determined blood plasma constituents, glucose, total protein, triglycerides, total cholesterol and high density lipoprotein. The results suggested that organic acid acidifier used in this experiment at the rates of 3 to 9 g/kg diet has a growth enhancing and mortality reducing effect in broiler chickens, with no significant influence on carcass yield, proportion of individual carcass parts and blood plasma constituents. It seems that the amount of 6 g of the applied acidifier per kilogram of feed may be recommended as the optimum dietary level if protein in the diet does not exceed 200–230 g crude protein per kilogram of diet.

Key words: broiler chicken, dietary acidifier, growth performance, mortality, carcass indices, meat composition, blood plasma parameters

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The characteristics of low-molecular-weight organic acids and their antibacterial activity were extensively discussed by Cherrington et al. (1991). Some of these acids are used as food additives, in dairy, vegetable and meat products. Short-chain organic acids, such as lactic, acetic, propionic and butyric are generated as end-products of anaerobic fermentation in conserved feeds, and also in the digestive tract of animals and humans. They are added to food and feed as preserving and anti-moulding agents (Dixon and Hamilton, 1981). The importance of these acids in livestock nutrition increased considerably in response to the ban on in-feed antibiotics. Formic, sorbic and propionic acids, applied in different combinations in feed mixtures or diets for animals, are used against *Salmonella* Spp. and *Clostridium perfringens* (Thomson and Hinton, 1997; Bassan et al., 2008; Mikkelsen et al., 2009). Feed additives containing low-molecular-weight organic acids are referred to as acidifiers. The solid-form acidifiers contain organic acids, organic acid salts or their blends, usually based on carriers, which do not react chemically with the active ingredient. They are used in poultry nutrition for the purpose of maintaining the pH of digesta at a level preventing the growth of pathogenic bacteria. They also show bactericidal activity against pathogenic intestinal microflora. Most often, pathogenic bacteria begin to develop in the digestive tract when the lumen pH of the small intestine and caecum exceeds 5.8–6.0, and that of large intestine exceeds 6.2 (Garcia et al., 2007; Paul et al., 2007; Mikulski et al., 2008). The multiplication of pathogenic microorganisms in the intestine may result in the inflammation of intestinal mucosa or necrosis of intestinal epithelium. These processes, accompanied by increased secretion of intestinal fluids, lead to diarrhoea (Mikkelsen et al., 2009). Diarrhoea and the associated dehydration causes birds to stop eating and may lead to their death in a very short period of time.

Acidifying additives can be used in feed or incorporated in drinking water, which improves its quality. Low-molecular-weight organic acids, particularly propionic acid, have also a strong inhibitory effect on the growth of mould fungi. They suppress the growth of pathogenic microflora in feeds produced in feed meal plants. Cereals used for feed production may contribute pathogenic bacteria and mould fungi, which may synthesize harmful mould mycotoxins under poor storage conditions.

The amount of acidifier recommended for inclusion in poultry diets depends on several factors, mainly on alkalizing effects of feed ingredients and mineral supplement such as calcium sources. Under production conditions, the ban on in-feed antibiotics may result in considerable mortality rates, especially during the first 21 days of rearing the birds. According to modern farming standards, chicken mortality rates must not exceed 4%. Excessive mortality may be due to the strong alkaline effect of high protein content of diets for young birds, when the digestive tract and its secretory capacity are not fully developed. Some studies found that the efficiency of acidifier fed to chickens increases in the presence of probiotic lactic acid bacteria and a prebiotic (Kalavathy et al., 2003; Jamroz et al., 2004; Brzóska et al., 2005, 2007; Brzóska and Stecka, 2007; Mountzouris et al., 2007). Furthermore, previous research studies have also shown that acidifiers and probiotics fed to young animals are more effective when used concurrently with prebiotics (Patterson and Burkholder, 2003; Brzóska et al., 2007).

An important issue is to optimize the dietary level of the acidifier for broiler chickens. Recommendations seem not precise enough. Insufficient amounts of the acidifier may inadequately acidify the digesta, while excessive amounts may inhibit the secretion of digestive enzymes, lowering the degree of nutrient hydrolysis, and nutrient absorption processes. There is also an economic aspect to optimizing the acidifier level. The use of mineral acid salts in acidifier formulas slows down their action, by prolonging acidification of digesta during passage through the digestive tract. The presence of acids is responsible for their rapid hydrolysis in the feed and has antibacterial and antifungal action.

The aim of the study was to determine the effect of the dietary level of acidifier containing butyric and propionic acids and salts of formic and butyric acids as the main active components, on broiler performance, mortality, carcass parameters, chemical composition of breast and leg muscles, and the level of main plasma metabolites.

Material and methods

The experiment procedures were approved by the Local Ethic Committee for Animal Experimentation. A total of 608 unsexed, day-old Ross 308 broiler chickens were randomly divided into 4 groups, with 4 replicates of 38 birds. Birds in each replicate were kept in pens covered with deciduous wood shavings at a density of 18 birds/m^2 and with approximately 33 kg of live birds/m^2 at the end of rearing. Indoor temperature, humidity and air exchange were in accordance with hygiene standards for young birds. The birds were vaccinated against Gumboro disease at day 5 and against fowl plague at day 12 of age. Vitazol (Biowet Drwalew, Poland), a vitamin supplement, was administered to chickens at several-day intervals throughout the experiment. All chickens received *ad libitum* starter type diets (1–21 days) followed by grower type diets (22–42 days). The diets were composed of ground maize, wheat grain and soybean meal as the main ingredients (Table 1). Water was provided in spot drinkers during the 3 weeks and in trough-type drinkers during weeks 4 to 6. A commercial Acidomix AFG (Novus) acidifier was used, which contained 20.7% butyric acid (E236), 17.5% ammonium propionate (E295), 12.5% propionic acid (E280), and 4.2% ammonium propionate (E284). The acidifier were applied in both diet types at the levels of 3, 6 and 9 g per kg (experimental group), and the control group was fed without Acidomix addition.

Dietary nutrients and feed acidity were determined by chemical analysis (AOAC, 1990). Amino acid composition of the feedingstuffs was determined using an automatic HPLC analyser after acid hydrolysis, and methionine following perchloric acid oxidation. Feed consumption and mortality were recorded throughout the study and the feed conversion ratios were subsequently calculated.

On day 43, ten birds (5 males and 5 females), 40 animals in total, were selected from each group and killed by decapitation after stunning. During slaughter, blood samples were collected into heparinized tubes and centrifuged. Fresh plasma was

used to analyse glucose content. Plasma was frozen and stored until analysis for total protein, triglycerides, total cholesterol and high-density lipoproteins (HDL). Blood components were analysed using Cormay Diagnostic kits. Measurements were performed on a Beckman DU 640 spectrophotometer. After slaughter, carcasses were defeathered and eviscerated. The weight of 40 bird carcasses, gizzard, liver, feet, omental fat and abdominal fat from the posterior part of the body cavity was determined. Both types of fat are referred to as depot fat. Carcasses were chilled at 5°C for 24 h. The next day, carcasses were dissected according to the method described by Zgłobica and Różycka (1972). Breast and leg muscles, depot fat, skin and leg bones were weighed. The weight of individual carcass parts was related to total carcass weight and expressed as percent. Samples of breast and of leg muscles from the right carcass part were collected, ground and frozen at –18°C to analyse dry matter and basic nutrients. After thawing, muscle samples were analysed for dry matter, crude protein, crude fat and crude ash. The analyses were performed by standard methods (AOAC, 1990).

Table 1. Components and nutritive value of the diets

Item	Diet	
	Starter (1–21 days)	Grower (22–42 days)
Feed ingredients (%)		
Maize	34.00	30.00
Wheat	26.10	34.10
Soybean meal	32.50	28.50
Rapeseed oil	4.00	4.00
Dicalcium phosphate	1.70	1.70
Ground limestone	0.60	0.60
NaCl	0.35	0.35
L-lysine HCl (78%)	0.11	0.11
DL-methionine (99%)	0.14	0.14
Mineral-vitamin premix[1) 2)]	0.50	0.50
Nutrients in 1 kg of dry matter:		
Crude protein (g)	229.9	205.6
Lysine (g)	12.7	10.9
Methionine+Cysteine (g)	5.2	5.7
Crude fat (g)	27.7	24.5
Crude fibre (g)	50.5	63.1
Calcium (g)	8.8	83
Phosphorus (g)	4.2	4.1
Metabolizable energy (MJ)	12.44	12.23

[1)] Supplied per kg of starter diet: vit. A – 13 5000 IU; vit. D_3 – 3 600 IU; mg: vit. E – 45; vit. B_1 – 3.25; vit. B_2 – 7.5; vit. B_6 – 5; vit. B_{12} – 0.0325; vit. K_3 – 3; biotin – 0.15; nicotinic acid – 45; calcium pantothenate – 15; folic acid – 1.5; choline chloride – 100; Mn – 100; Cu – 1.75; Fe – 76.5; Se – 0.275; I – 1; Zn – 75; Co – 0.4; Endox (antioxidant) – 125; Sincox (coccidiostat) – 1 g; calcium – 0.679 g.

[2)] Supplied per kg of starter diet: vit. A – 12 000 IU; vit. D_3 – 3 250 IU; mg: vit. E – 40; vit. B_1 – 2; vit. B_2 – 7.25; vit. B_6 – 4.25; vit. B_{12} – 0.03; vit. K_3 – 2.25; biotin – 0.1; nicotinic acid – 40; calcium pantothenate – 12; folic acid – 1.0; choline chloride – 450; Mn – 100; Cu – 1.75; Fe – 76.5; Se – 0.275; I – 1; Zn – 75; Co – 0.4; Endox (antioxidant) – 125; Sincox (coccidiostat) – 1 g; calcium – 0.79 g.

The data were subjected to analysis of variance (ANOVA) using SAS/STAT® ver. 5.1 (SAS, 1994–2001). Mean values for the groups were compared using Duncan's multiple range test at the 1% and 5% level of probability.

Results

The components and nutritive value of the feeds are presented in Table 1. Adding the acidifier to chicken feeds reduced the pH of starter diet from 6.90 to 5.89, and that of the grower diet from 6.28 to 5.73. Supplementing diets with the increasing amounts of acidifier (from 3 to 9 g/kg) significantly increased body weight of chickens at 21 and 42 days of age ($P \leq 0.01$) compared to the control birds (Table 2). Mortality decreased significantly, with significant differences in relation to the control group ($P \leq 0.01$). Feed consumption and conversion remained unchanged. Feeding the acidifier significantly increased carcass weight at 43 days of the experiment ($P < 0.01$; Table 3). Significant differences were found in dressing percentage, which was the highest with the acidifier supplemented at 3 g/kg and the lowest at 9 g/kg, but did not differ from the control value. There were no statistically significant differences in the weight of individual carcass parts, including breast and leg muscles, in gizzard and liver weight, and in the amount of depot fat. The proportion of individual carcass parts dissected (muscles, depot fat, skin, leg bones, feet) and the proportion of organs (gizzard, liver) were similar across dietary treatment. There were no significant differences in chemical composition, including dry matter, protein, fat and ash content of breast and leg muscles. No significant differences were also obtained for blood plasma parameters, including glucose, total protein, triglycerides, total cholesterol and HDL cholesterol (Table 4).

Table 2. Feed pH and growth performance of broiler chickens

Item	Control	Acidifier level (g/kg diet)			SEM
		3	6	9	
Feed pH					
– Starter	6.90	6.42	6.10	5.89	
– Grower	6.28	5.97	5.81	5.73	
Body weight, 21 days (g)	597 aA	634 bB	646 bB	646 bB	4
Body weight, 42 days (g)	2394 aA	2459 bAB	2480 bB	2483 bB	11
Mortality (%)	2.58 bB	0.00 aA	0.00 aA	0.59 aA	0.78
Feed intake (kg/bird/42 days)	4.06	4.30	4.23	4.20	0.42
Feed conversion (g/kg BWG)	1.70	1.75	1.71	1.69	0.12
European Efficiency Index (points)	327	335	345	348	41

a, b – values in rows with different letters differ significantly ($P < 0.05$).
A, B – values in rows with different letters differ significantly ($P < 0.01$).
SEM – standard error of the mean.
BWG – body weight gain.

Table 3. Post-slaughter characteristics of broiler chickens

Item	Control	Acidifier level (g/kg of diet)			SEM
		3	6	9	
Slaughter weight (g)	2492	2588	2668	2604	43
Carcass weight (g)	1768 aA	1899 bcBC	1946 cC	1834 bB	32
Dressing percentage	70.95 ab	73.38 b	72.94 ab	70.43 a	0.42
Absolute weight (g)					
breast muscles	490.5	538.1	550.6	498.4	10.1
leg muscles	379.6	399.4	404.1	374.2	7.5
gizzard	28.0	28.0	29.0	27.9	0.4
liver	57.1	53.6	55.8	59.5	1.4
depot fat	37.3	41.0	42.5	34.5	1.4
skin	101.1	105.9	111.1	97.8	2.3
leg bones	99.3	98.4	102.7	104.4	2.4
feet	84.1	86.8	89.2	91.0	2.3
Relative proportion (% of carcass weight)					
breast muscles	27.7	28.1	28.3	27.2	0.3
leg muscles	21.5	21.0	20.8	20.4	0.2
gizzard	1.6	1.5	1.5	1.5	0.4
liver	3.2	2.9	2.9	3.2	0.1
depot fat	2.1	2.2	2.2	1.9	1.4
skin	5.7	5.6	5.7	5.3	0.1
leg bones	5.6	5.2	5.3	5.7	0.1
feet	4.8	4.6	4.6	5.0	0.1

For abbreviations see Table 2.

Table 4. Chemical composition of breast and leg muscles and blood plasma parameters

Item	Control	Acidifier level (g/kg of diet)			SEM
		3	6	9	
Breast muscle:					
dry matter (%)	25.28	25.61	25.66	26.07	0.09
crude protein (% DM)	23.58	23.56	23.75	24.27	0.07
ether extract (% DM)	1.05	1.00	1.14	0.88	0.03
ash (%DM)	1.18	1.16	1.15	1.17	0.01
Leg muscle:					
dry matter (%)	26.11	25.49	25.81	26.03	0.11
crude protein (%DM)	19.56	19.51	19.57	19.67	0.07
ether extract (%DM)	5.65	5.14	5.34	5.47	0.13
ash (%DM)	1.10	1.11	1.09	1.09	0.00
Plasma parameters (mg/dl):					
glucose	261.3	262.3	249.7	276.3	6.7
total protein	3.78	5.58	3.79	3.60	0.06
triglycerides	43.12	35.08	29.59	35.43	1.81
total cholesterol	134.8	129.0	132.1	123.2	2.1
high density lipoproteins	99.3	94.1	99.0	89.5	1.7
HDL/TC×100	73.7	73.0	74.9	72.7	-

For abbreviations see Table 2.

Discussion

The ban on the use of in-feed antibiotics in farm animals, including poultry, has prompted a search for feed additives that control gut microbial status. In addition to the respiratory tract, the digestive tract of birds runs the greatest risk of being infected with pathogenic microorganisms. Although the digestive tract of the newly hatched bird is sterile, its contact with feed and water leads to rapid implantation of gastric mucosa by lactic acid bacteria, including *Lactobacillus* Spp., but infection with pathogens is also possible. *Salmonella* spp., *Clostridium*, *Campylobacter* and *Shigella* spp. bacteria represent a health risk to poultry as they may induce diarrhoea in young birds, slow the rate of growth and impair feed intake, and thus cause deaths. Pathogenic food poisoning is a threat to consumers of poultry meat and eggs (Simon et al., 2001). It may occur in abattoirs and poultry processing plants that do not conform to hygiene standards. It has been shown that the addition of organic acids, e.g. lactic acid to drinking water protects chicks from *Campylobacter* colonization (Chaveerach et al., 2004; Byrd et al., 2009).

It is more common to add organic acids to the chicken feed, which eliminates pathogenic bacteria, including *Salmonella* Spp., from the digestive tract (Hinton and Linton, 1988; Rouse et al., 1988; McHan and Shotts, 1992). The usefulness of short-chain monocarboxylic fatty acids and their derivatives, unsaturated, hydrogenated, phenolic and polycarboxylic acids was investigated with poultry feeds (Cherrington et al., 1991). Short-chain fatty acids, e.g. acetic, propionic and butyric, are produced in millimolar quantities in the animal and human digestive tract with its resident microflora. They are anaerobic lactic acid bacteria (Gram-positive *Lactobacillus* Spp. and *Enterococcus* Spp.) which ferment glucose in the intestinal mucosa to acids. Organic fatty acids, in particular propionic and formic ones, are known to be feed additives that inhibit the growth of moulds in wet feed materials (Dixon and Hamilton, 1981). These acids are used in different combinations as agents against *Salmonella* Spp. and *Escherichia* coli (Thompson and Hinton, 1997; Ricke, 2003; Paul et al., 2007). The production of organic acids in the digestive tract of poultry is stimulated by the feeding of probiotic bacteria (Kalavathy et al., 2003; Brzóska et al., 2005; Brzóska, 2007; Brzóska and Stecka, 2007; Award et al., 2009).

Another gastrointestinal disease found in broiler flocks is necrotic enteritis (Van Der Sluis, 2000). It is difficult to detect in its subclinical form and is detrimental in economic terms. The main causative agent is *Clostridium perfringens*, which is found in small intestine and liver (Gholamiandehkordi et al., 2007). It is conjectured that acidifiers limit the incidence of necrotic enteritis and the associated losses (Mikkelsen et al., 2009).

The results of this study show that the dietary level of 0.3, 0.6 or 0.9% acidifier, increases the growth rate of chickens during both the first 21 days of age and over the entire 42-day period. In the experimental groups, the live weight gains of the chickens fed with increasing amounts of the acidifier added were 65, 86 and 89 g/bird higher than the control value, which in relative terms corresponded to an increase of 2.7 to 3.7%. Better body weight gain was accompanied by increased feed consumption (by 0.24, 0.17 and 0.14 kg/bird, respectively), although no significant

differences were found in feed conversion ratio (1.71 kg/kg at average). These data may be indicative of the superior conversion of the amino acids and energy from the acidifier supplemented diets and of the superior conversion of energy into broiler tissues.

The gradual increase in acidifier amounts in the experimental diets prevented mortality in chickens. The addition of 3 and 6 g/kg of diet completely eliminated deaths, bringing mortality equal to zero. This suggests that the acidifier protected the chickens from intestinal infections and gastrointestinal disorders, which are a common cause of mortality. The highest dose of the acidifier (9 g/kg) slightly increased chicken mortality to 0.59%, which was 4-fold lower than the value obtained for the control group, and 6.6-fold lower than acceptable standards for mortality in large-scale operations. In the present experiment, in terms of stocking density and body weight per unit floor area, the housing conditions were similar to the conditions used in large-scale production. The degree of diet acidification had no effect on carcass evaluation parameters, which is in agreement with the results of an earlier study (Young et al., 2001). In a study on male turkeys Mikulski et al. (2008) evaluated the physiological and growth effects of organic acids, organic acids with essential oils or herbal extracts added to diets, and found a significant decrease in pH of the crop contents, but no effect on the pH of caecal digesta. All supplements significantly increased the body weights of turkeys at the age of 84 days, with no influence on carcass traits.

The Acidomix acidifier used in the current experiment contained ammonium formate, propionic acid and ammonium propionate. Such composition resulted in the hydrolysis and dissociation of both salts into formic and propionic acids in the aqueous environment of the intestine during the digesta passage through the intestinal tract. Considering the slow hydrolysis of salts in the digestive tract, it can be assumed that the acidifying effect persisted into the final section of the digestive tract, which is the least acidic and most vulnerable to growth of pathogenic bacteria. In a study with broilers, Czerwiński et al. (2010) investigated the effect of dietary pea inclusion in the presence of organic acids or probiotic, composition of fumaric acid, calcium formate, calcium propionate and potassium sorbate. Organic acid supplementation slightly increased the *Lactobacillus/Enterococcus* counts, but total bacterial counts in caecal contents were not affected. Świątkiewicz et al. (2010) investigated the effect of prebiotic and organic acid on eggshell quality of laying hens. They concluded that acidifiers can lower the pH of the diet and beneficially influence egg shell quality.

The mode of action of low-molecular-weight organic acids on pathogenic bacteria has been elucidated by several authors. Ricke (2003) described a mechanism in which organic acids penetrate the lipid membrane of the bacterial cell and once incorporated into the neutral pH of the cell cytoplasm dissociate into anions and protons (Eklund, 1983; Salmond et al., 1984; Cherrington et al., 1990, 1991). Export of excess protons requires consumption of cellular adenosine triphosphate (ATP). This may result in a depletion of cellular energy, and thus in cell death. It has also been speculated that organic acids interfere with cytoplasmic membrane structure and intercellular transport as a result of changes in electrical gradients across cell

membrane, which may also be lethal to pathogenic bacterial cells (Russell, 1992; Axe and Bailey, 1995).

Of special importance was the presence of butyric acid in the evaluated acidifier. Butyric acid and its salts, including ammonium isobutyrate, are known to be factors that regenerate intestinal epithelium of young suckling animals, resulting in regeneration of intestinal villi, which constitute the main absorptive area of the digestive tract (Jang et al., 2008; Puyalto and Locatelli, 2008). Some authors showed that dietary supplementation with a blend of organic acids containing lactic, formic, citric, phosphoric and butyric acids had no effect on growth rate but increased gizzard weight and the length of intestinal epithelium in broilers (Jang et al., 2008). However, Mahdavi and Torki (2009) found no direct effect of feeding chickens with butyric acid (2 or 3 g/kg feed) on weight gains, feed intake and conversion, and bird mortality. The experimental factor had also no effect on the weight of individual carcass cuts, but a significant effect of increasing small intestinal length was stated. The results cited above indicate that the increase in chickens' body weight in response to acidifying additives does not occur in every situation. It is of economic importance that the acidifiers significantly reduced chicken mortality in all studies mentioned above. In our study, both the higher weight gain than in the control group and a similar feed conversion rate may suggest that the experimental factor contributed to better utilization of dietary protein, amino acids and metabolizable energy. However, this issue requires more in-depth nutritional and physiological research. Based on the studies with organic and inorganic acidifiers, Viola and Vieira (2007) suggested that acidifiers are as efficient as antibiotics in maintaining the performance and morphology of the small intestines of broiler chickens.

In conclusion, due to the high protein contents, in addition to minerals, conventional broiler diets may have a highly alkalizing effect. Because the digestive tract of chickens is not ready to counteract the effects of alkaline digesta, the administration of acidifiers and additives that reduce digesta pH seem the most important factor regulating the status of intestinal microflora. Our study with Acidomix AFG acidifier showed that the most advantageous dietary level of this feed additive ranges from 3 to 9 g/kg, with 6 g/kg regarded as the optimum dose.

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Wpływ zakwaszacza diety na masę ciała, śmiertelność, wydajność rzeźną i skład mięsa kurcząt rzeźnych

STRESZCZENIE

W doświadczeniu wykonanym na 608 kurczętach rzeźnych Ross 308 badano wpływ zakwaszacza diety na masę ciała, spożycie i wykorzystanie paszy, śmiertelność ptaków, wydajność rzeźną, cechy poubojowe tuszek, skład chemiczny mięśni piersiowych i nóg oraz wskaźniki chemiczne osocza krwi. Podawanie zakwaszacza kurczętom w ilości 0, 3, 6 i 9 g/kg diety obniżyło odczyn (pH) diety na pierwszy okres chowu (1–21 dni) z 6,90 do 5,89, a drugi okres chowu (22–42 dni) z 6,28 do 5,73. W porównaniu z grupą kontrolną, nie otrzymującą zakwaszacza, istotnie wzrosła masa ciała kurcząt 42. dnia życia, odpowiednio o 2,3; 3,6 i 3,7% ($P<0,01$). Śmiertelność zmalała z 2,58% w grupie kontrolnej do 0,00–0,59% w grupach doświadczalnych ($P<0,01$). Zakwaszenie diety nie miało istotnego wpływu na spożycie i wykorzystanie paszy. Nie stwierdzono istotnych różnic w masie mięśni piersiowych i mięśni nóg, a także masie żołądka, wątroby i tłuszczu zapasowego ptaków. Mięśnie piersiowe miały 21,6% w grupie kontrolnej i 21,3% masy tuszki w grupie doświadczalnej. Zakwaszenie diety zwiększyło wartość indeksu EEI z 327 (grupa kontrolna) do 348 (grupa doświadczalna), przy 9 g/kg zakwaszacza. Nie stwierdzono istotnych różnic w składzie chemicznym mięśni piersiowych i mięśni nóg, w tym zawartości suchej masy, białka ogólnego, tłuszczu surowego i popiołu. Zawartość białka w mięśniach piersiowych wynosiła 23,58% w grupie kontrolnej i 23,86% średnio w grupach doświadczalnych ($P\geq0,01$). Zawartość białka w mięśniach nóg wynosiła odpowiednio 19,56% i 19,58% ($P\geq0,01$). Nie stwierdzono istotnych różnic we wskaźnikach osocza krwi. Wnioskowano, że zakwaszacz zawierający kwas propionowy i sole kwasu mrówkowego oraz masłowego poprawia efektywność produkcji kurcząt rzeźnych istotnie obniżając straty powodowane zakażeniami bakteryjnymi przewodu pokarmowego. Użycie zakwaszacza od 3 do 6 g/kg diety istotnie zwiększa masę ciała i tuszek kurcząt nie powodując istotnych różnic w masie i proporcjach poszczególnych partii tuszek. Optymalny poziom preparatu of Acidomix AFG w diecie dla kurcząt zawierającej 206–230 g białka ogólnego wynosił 6 g/kg.