Effects of different levels of fenofibrate on growth performance, carcass characteristics, abdominal fat, serum constituents, immune system, caeca and microbial flora of broilers

Maryam Azizi, Mehrdad Bouyeh and Alireza Seidavi
Department of Animal Science, Rasht Branch, Islamic Azad University, Rasht, Iran

ABSTRACT
So far, the effect of fenofibrate on the performance, quantitative and qualitative characteristics of broilers have not been investigated. For this reason, this study was performed to evaluate the effects of different levels of fenofibrate on the quantitative and qualitative characteristics of broilers in a completely randomised design with three treatments, four replicates and 10 one-day-old male Ross 308 strain chicks per replicate for 42 d. Experimental treatments included three levels of fenofibrate (0, 50 and 100 mg kg\(^{-1}\)), which were used in combination with the basal diet. Results showed that 100 mg kg\(^{-1}\) fenofibrate in the diet increased the weight gain and European production efficiency factor (EPEF) and reduced feed intake and feed conversion ratio (FCR) of the whole period compared to the control (\(p < .05\)). The effect of experimental treatments on blood serum and immune system parameters other than spleen and bursal weights was not significant (\(p > .05\)). The number of \(E.\) coli, coliform and clostridium bacteria in the caecum decreased with the use of fenofibrate in comparison with the control group (\(p < .05\)), but fenofibrate increased the lactobacilli population in the caecum (\(p < .05\)). According to the results, the use of 100 mg kg\(^{-1}\) fenofibrate in the diet is recommended to improve some quantitative and qualitative characteristics of broilers.

HIGHLIGHTS
- The accumulation of fat in the body of chickens is an undesirable trait for producers and consumers.
- Fenofibrate is a derivative of fibric acid and a blood fat-lowering chemical used to lower cholesterol levels in cardiovascular patients.
- The results of this study showed that the use of fenofibrate is effective in improving performance, and reducing the population of harmful bacteria in the gastrointestinal tract.

Introduction
The accumulation of fat in the body of chickens is an undesirable trait for producers and consumers. Excessive accumulation of fat in the ventricular and visceral areas of poultry indicates the loss of diet and the production of redundant and worthless products economically. In recent years, the consumption of high-fat chickens has been limited by consumers (Emmerson 1997). Therefore, due to the increasing demand for low-fat protein foods and also solving the problem of fat accumulation in the body of chickens, it seems that it is necessary to find some strategies to regulate lipids and reduce fat accumulation in poultry bodies (Cartwright 1986; Cabel et al. 1988; Lien and Horng 2001; Parsaeimehr et al. 2014).

One way to reduce fat accumulation in poultry is through genetic modification, which presents long-term results. It seems that the study of nutritional factors and production management can be prioritised as short-term solutions (Buyse et al. 2001; Rajabzadeh Nesvan 2013). In this regard, many studies have been done by chemical drugs to increase metabolism and reduce fat accumulation in poultry bodies and the positive effect of these drugs on reducing ventricular fat has been reported (Cakir and Yalcin 2007; Hosseintabar et al. 2015; Panahi et al. 2019).

Lipid-lowering therapy using 3-hydroxy-3-methylglutaryl-coenzyme A (HMGCoA) reductase inhibitors resulting in a substantial decrease in low-density lipoprotein (LDL) cholesterol concentration has been
found to reduce the risk of cardiovascular events in high-risk individuals (Ruotolo et al. 1998).

Fenofibrate is a derivative of fibric acid and a blood fat-lowering chemical used to lower cholesterol levels in cardiovascular patients. Over time, with the introduction of new formulations of this drug and the use of nanotechnology in the production of fenofibrate, its bioavailability increased. Fenofibrate is currently available as capsule formulation of micronised (67, 100, 200, 250 and 300 mg) and micro-coated tablet formulation of micronised (145 and 160 mg) (Keating and Croom 2007; Mc Keage and Keating 2011). Fenofibrate has a bioavailability of 60-90% and a half-life of 20 h. It is metabolised via the hepatic pathway and eliminated through urine (Sidhu and Tripp 2020).

Fenofibrate and fibrin acid derivatives reduce fat, very low-density lipoprotein (VLDL), low-density lipoprotein (LDL) and triglycerides and increase high-density lipoprotein (HDL) (Konig et al. 2007; Yang and Keating 2009). Fenofibrate and gemfibrozil are currently one of the most widely available and used fibric acid derivatives on the market (Packard et al. 2002; Yang and Keating 2009; Panahi et al. 2019). Fenofibrate has the highest effect on the reduction of LDL compared to the other fibrates, and its most important effect is triglyceride reduction (Schonfeld 1994; Lozada and Dujovne 1994). Fenofibrate has also been reported to increase the activity of the enzymes involved in catabolism of lipoprotns (Balfour et al. 1990). Flores-Castillo et al. (2019) reported that fenofibrate reduced cholesterol and triglycerides and increased HDL in New Zealand white rabbits. Vinegar et al. (2001) showed that fenofibrate consumption reduced triglycerides and LDL and increased HDL in rhesus monkeys. But so far, the effect of fenofibrate on broilers has not been evaluated.

Therefore, this study was conducted to investigate the effect of fenofibrate on the growth performance, carcass characteristics, blood serum components (especially triglyceride, total cholesterol, LDL and HDL as important lipid parameters which can be influenced by fenofibrate), immune system and microbial flora of the caecum of broilers.

Materials and methods

In order to investigate the effect of fenofibrate on the diet of broilers, an experiment was performed based on a completely randomised design in four replicates and 10 one-day-old male Ross 308 strains chicks per replicate for 42 d. The studied treatments were: 1 – basal diet (without fenofibrate), 2 – basal diet + 50 mg kg⁻¹ fenofibrate and 3 – basal diet + 100 mg kg⁻¹ fenofibrate.

The fenofibrate used in this study was prepared from Abidi Hygienic Co (Tehran, Iran) and used according to the desired concentrations. The diets were formulated according to the nutritional needs of commercial Ross 308 strain broiler chickens. The components and composition of the basal diet, including the starter (1–10 d), grower (11–24 d) and finisher (25–42 d old) periods which are shown in Table 1. Environmental conditions for all groups were similar including 23 h of exposure and 1 h of darkness, humidity of 75–60% and room temperature of 32°C on the first day, which decreased by 3°C after each week. Throughout the period, all chicks had free access to water and feed (ad libitum). Vaccination was performed to prevent bronchitis (1 and 12 d of age), Newcastle disease (10 and 19 d of age) and infectious bursal disease (15, 22 and 28 d of age). All the vaccines were prepared from Razi Pharmaceutical Company (Karaj, Iran).

Measurement of traits

Growth performance and carcass characteristics

At the end of each week, the weight of the chicks, the amount of feed intake and the feed conversion ratio (FCR) were measured, and at the end of the experiment, the weights of different parts and organs of carcass of the two chicks from each replicate were measured (Farrokhyan et al. 2014). The European

Table 1. Feed ingredients and chemical compounds of diets.

| Feed ingredient (%) | Starter (1–10, d) | Groover (11–24, d) | Finisher (25–42, d) |
|---------------------|------------------|-------------------|---------------------|
| Maize               | 47.03            | 59.60             | 65.99               |
| Wheat               | 5.58             | 5.00              | 5.00                |
| Soybean meal (44% crude protein) | 29.02 | 16.15 | 10.28 |
| Corn gluten         | 10.00            | 11.48             | 11.50               |
| Soybean oil         | 3.50             | 3.40              | 3.09                |
| Limestone           | 1.45             | 1.23              | 1.00                |
| Di-calcium phosphate| 1.95             | 1.80              | 1.83                |
| NaCl                | 0.200            | 0.200             | 0.200               |
| Vitamin and mineral supplements* | 0.500 | 0.500 | 0.500 |
| DL-methionine       | 0.520            | 0.580             | 0.570               |
| L-Lysine Hydrochloride | 0.250 | 0.060 | 0.040 |
| Calculated nutrients|                 |                   |                     |
| Metabolical energy (kcal kg⁻¹) | 2950 | 3000 | 3050 |
| Crude protein (%)   | 22.0             | 20.0              | 19.0                |
| Lysine (%)          | 1.30             | 1.20              | 1.10                |
| Methionine (%)      | 0.560            | 0.540             | 0.520               |
| Methionine + cysteine (%) | 0.920 | 0.900 | 0.880 |
| Calcium (%)         | 1.04             | 0.950             | 0.920               |
| Available phosphorus (%) | 0.520 | 0.470 | 0.410 |

*Each kilogram of mineral supplement contained 40,000 mg of manganese, 20,000 mg of iron, 33,900 mg of zinc, 4000 mg of copper, 400 mg of iodine and 80 mg of selenium. Each kilogram of vitamin supplement contained 3600,000 international units of vitamin A, 800,000 international unit of vitamin D₃, 7200 international unit of vitamin E, 710 mg vitamin B₁₂, 2640 mg vitamin B₂, 1176 mg vitamin B₆, 400 mg vitamin B₉, 6 mg vitamin B₁₂, 800 mg vitamin K₃, 3920 mg of pantothenic acid, 12,000 mg of niacin, 40 mg of biotin and 200,000 mg of choline chloride.
production efficiency factor (EPEF) was calculated using the following formula (Aviagen 2018):

$$\text{EPEF} = \frac{\text{viability percentage} \times \text{final weight in gram}}{\left(\text{feed conversion ratio} \times \frac{\text{number of rearing days}}{10}\right)}$$

**Parameters of blood serum**

At the end of the experiment (42 d of age), a chick from each pen was randomly selected and blood sample was taken from a wing vein to measure blood serum parameters. Blood samples were stored at 30 °C until clotting. Separation of clear serum from blood samples was then performed by a centrifuge device (3000 xg) (EppendorfCentrifuge5702, Hamburg, Germany). The resulting samples were stored in semicircular microtubes at −20°C until the time of analysis. Measurement of glucose, triglycerides, cholesterol, protein, albumin, HDL, LDL and VLDL of blood samples was performed by autoanalyser apparatus (Hitachi 917, Tokyo, Japan) and using biochemical commercial kits (Pars Azmoun, Tehran, Iran; Licence of Diagnostic Systems Co., Heidelberg, Germany) (Hosseintabar et al. 2015).

**Immune system**

To determine the effect of fenofibrate on the immune system, the weights of the immune organs, including the spleen, bursa of Fabricius and thymus, were measured after removing from the carcase of the three slaughtered chickens from each replicate at the final day of the experiment with a digital scale (A & DGF300, Tokyo, Japan) with an accuracy of 0.01 g. In this study, SRBC test (antibody level produced against sheep’s red blood cells), Newcastle (NDV) and influenza (AIV) titres were examined; for this purpose, on 28 and 35 d of the experiment, 0.1 cc of diluted SRBC solution was injected into the wing vein of two distinct chicks from each replicate. Seven days later, blood was taken from the injected chickens, and 16 h later (after blood clotting), the blood serum was removed. The microtitre haemagglutination method was used to determine the antibody titre against sheep’s red blood cells (35 and 42 d of age), Newcastle and influenza (42 d of age). A 50 μL mercapto-ethanol was used to measure the antibody titre resistance to 2-mercaptophanol (IgM). By subtracting the IgM titre from the total response titre SRBC, the immunoglobulin M (IgM) titre was obtained by Seidavi et al. (2014).

**Microbial flora of caecum**

To examine the microbial flora of the gastrointestinal tract, a sample was taken from the caecum at 42 d of age. For this purpose, a chick from each replicate was randomly selected and immediately after slaughter, its gastrointestinal tract was removed and one millilitre of the contents of the caecum was removed using a sampler. The prepared sample was transferred to a container containing phosphate buffer and it was properly mixed. De Man, Rogosa, Sharpe agar culture medium was used for lactobacilli cultivation, Eosin Methylene Blue for E. coli cultivation and MacConkey agar for coliform cultivation and SPS Agar (Sulfite Polymyxin Sulfadiazine) for clostridium cultivation. All of the mediums were obtained from Merck (Darmstadt, Germany) (Dibaji et al. 2014).

**Statistical analysis**

At the end of the experiment, the data were analysed by SAS statistical software (SAS 2002; SAS Institute, Cary, NC). Comparison of the means of treatments was performed with Tukey’s multiple comparison test at the probability level of 5%. The design was completely randomised design and its model was as following:

$$Y_i = \mu + A_i + E_i$$

where $Y_i$ = observation value; $\mu$ = mean of community; $A_i$ = fenofibrate effect (0, 50 and 100 mg kg$^{-1}$); $E_i$ = experimental error effect.

**Results**

**Growth performance**

The effects of different levels of fenofibrate on growth performance and EPEF factor are shown in Tables 2 and 3. Examining the comparison of the means showed that, feed intake decreased significantly with 100 mg kg$^{-1}$ fenofibrate application in the starter period, grower and the entire periods compared to the control ($p < .05$). The effect of experimental treatments on live weight in the starter and grower periods was not significant ($p \geq .05$), but live weight increased with the consumption of fenofibrate in the diet in the finisher period and the whole period ($p < .05$). The effect of fenofibrate on FCR in starter and finisher periods was not significant ($p \geq .05$), but in the grower period and the whole period, consumption of 100 mg kg$^{-1}$ fenofibrate reduced FCR compared to control and treatment of 50 mg kg$^{-1}$ fenofibrate ($p < .05$). The EPEF factor increased with the use of fenofibrate compared to the control ($p < .05$) and the highest EPEF factor was obtained by consuming 100 mg kg$^{-1}$ fenofibrate.
Carcase characteristics

The effect of using different levels of fenofibrate on carcase characteristics is given in Table 4. Consumption of fenofibrate in the diet did not have a significant effect on the relative weight of breast, liver, gizzard, pancreas, ventricular fat, wings and thighs (p > 0.05). However, the relative weight of the heart increased significantly with the use of 50 mg kg⁻¹ fenofibrate compared to the control and the treatment containing 100 mg kg⁻¹ fenofibrate (p < 0.05). The relative weight of the head was highest in the chickens fed by 100 mg kg⁻¹ fenofibrate (p < 0.05).

Parts of intestine

The effect of fenofibrate on the relative weight of intestinal components is shown in Table 5. The results showed that the effect of experimental treatments on the relative weight of the jejunum and the proventriculus was significant (p < 0.05). However, consumption of different levels of fenofibrate did not have a significant effect on the relative weight of the rectum, right and left caecum, ileum and duodenum (p > 0.05).

Blood serum parameters

The effect of using different levels of fenofibrate in diet on blood serum parameters is given in Table 6. The results showed that consumption of 50 and 100 mg kg⁻¹ fenofibrate in the diet of broilers did not have a significant effect on blood parameters including triglycerides, cholesterol, glucose, albumin, HDL, LDL, VLDL and HDL/LDL and total protein (p > 0.05).

Immune system

The effect of different levels of fenofibrate on the immune system is shown in Table 7. The results showed that the effect of experimental treatments on
Table 5. Relative weight of intestinal segment mean (±SEM) of Ross 308 broilers at 42 d of age fed diets containing the different levels of fenofibrate.

|                   | Rectum (%) | Right caecum (%) | Left caecum (%) | Ileum (%) | Jejunum (%) | Duodenum (%) | Proventriculus (%) |
|-------------------|------------|------------------|-----------------|-----------|-------------|--------------|---------------------|
| Control           | 0.185      | 0.352            | 0.357           | 1.170     | 2.722<sup>a</sup> | 0.767        | 0.417<sup>b</sup>   |
| Fenofibrate (50, mg kg<sup>-1</sup>) | 0.162      | 0.330            | 0.347           | 1.382     | 2.230<sup>a</sup> | 0.672        | 0.510<sup>b</sup>   |
| Fenofibrate (100, mg kg<sup>-1</sup>) | 0.182      | 0.380            | 0.340           | 1.140     | 3.427<sup>a</sup> | 0.725        | 0.450<sup>b</sup>   |
| p Value           |            |                  |                 |           |             |              |                     |
| SEM               | 0.0150     | 0.216            | 0.0170           | 0.064     | 0.0256      | 0.0310       | 0.0200              |

*In each column, means with the similar letters are not significantly different (p < .05).
SEM: standard error of the mean.

Table 6. Blood serum parameters mean (±SEM) of Ross 308 broilers at 42 d of age fed diets containing the different levels of fenofibrate.

|                   | Triglyceride (mg dL<sup>-1</sup>) | Cholesterol (mg dL<sup>-1</sup>) | Glucose (mg dL<sup>-1</sup>) | Albumin (g dL<sup>-1</sup>) | HDL (mg dL<sup>-1</sup>) | LDL (mg dL<sup>-1</sup>) | VLDL (mg dL<sup>-1</sup>) | HDL/LDL | Total protein (g dL<sup>-1</sup>) |
|-------------------|-----------------------------------|----------------------------------|-----------------------------|---------------------------|-------------------------|--------------------------|--------------------------|---------|-------------------------------|
| Control           | 226.00                            | 148.25                           | 257.50                      | 1.28                      | 61.50                   | 41.55                    | 45.20                    | 1.547   | 2.02                          |
| Fenofibrate (50, mg kg<sup>-1</sup>) | 226.50                            | 155.75                           | 261.00                      | 1.11                      | 61.75                   | 48.70                    | 45.30                    | 1.327   | 1.99                          |
| Fenofibrate (100, mg kg<sup>-1</sup>) | 232.25                            | 150.75                           | 261.75                      | 1.16                      | 64.25                   | 40.05                    | 46.45                    | 1.620   | 2.31                          |
| p Value           | .114                               | .321                             | .238                        | .156                      | .53                     | .34                      | .114                     | .464    | .220                          |
| SEM               | 2.07                               | 3.35                             | 1.74                        | 0.0560                    | 1.85                    | 4.24                     | 0.415                    | 0.166   | 0.129                         |

*In each column, means with the similar letters are not significantly different (p < .05).
SEM: standard error of the mean; HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL: very-low-density lipoprotein.

Table 7. Immune system mean (±SEM) of Ross 308 broilers fed diets containing the different levels of fenofibrate.

|                   | Bursa (%) | Thymus (%) | Spleen (%) | Total Ig<sub>1</sub> (at 35, d of age) | IgG<sub>1</sub> (at 35, d of age) | IgM<sub>1</sub> (at 35, d of age) | Total Ig<sub>2</sub> (at 42, d of age) | IgG<sub>2</sub> (at 42, d of age) | IgM<sub>2</sub> (at 42, d of age) | NDV (at 42, d of age) | AIV (at 42, d of age) |
|-------------------|-----------|------------|------------|--------------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|-----------------------|---------------------|
| Control           | 0.240<sup>a</sup> | 0.105     | 0.077<sup>b</sup> | 2.00                                 | 1.00                            | 1.00                            | 3.25                            | 1.50                            | 1.75                            | 3.25                  | 2.25                |
| Fenofibrate (50, mg kg<sup>-1</sup>) | 0.185<sup>a</sup> | 0.097     | 0.105<sup>a</sup> | 2.25                                 | 1.25                            | 1.00                            | 3.50                            | 1.50                            | 2.00                            | 3.50                  | 2.75                |
| Fenofibrate (100, mg kg<sup>-1</sup>) | 0.257<sup>a</sup> | 0.100     | 0.080<sup>b</sup> | 2.75                                 | 1.25                            | 1.50                            | 3.50                            | 1.50                            | 2.00                            | 4.00                  | 2.00                |
| p Value           | .027                               | .772      | .004       | .420                                 | .811                            | .465                            | .767                            | 1.00                            | .767                            | .295                  | .274                |
| SEM               | 0.016                               | 0.007     | 0.0046     | 0.390                                | 0.311                           | 0.288                           | 0.276                           | 0.288                           | 0.276                           | 0.322                 | 0.013               |

*In each column, means with the similar letters are not significantly different (p < .05).
SEM: standard error of the mean; Total Ig: total immunoglobulin; IgG: immunoglobulin G; IgM: immunoglobulin M; NDV: Newcastle disease virus; AIV: Avian influenza virus.

Table 8. Caeca microflora mean (±SEM) of Ross 308 broilers at 42 d of age-fed diets containing the different levels of fenofibrate.

|                   | Clostridium (CFU g<sup>-1</sup>) | Lactobacilli (CFU g<sup>-1</sup>) | E. coli (CFU g<sup>-1</sup>) | Coliform (CFU g<sup>-1</sup>) |
|-------------------|---------------------------------|--------------------------------|----------------------------|-----------------------------|
| Control           | 5.711<sup>a</sup> | 7.908<sup>b</sup> | 6.858<sup>a</sup> | 8.771<sup>a</sup> |
| Fenofibrate (50, mg kg<sup>-1</sup>) | 5.530<sup>b</sup> | 8.065<sup>b</sup> | 8.563<sup>b</sup> | 8.576<sup>b</sup> |
| Fenofibrate (100, mg kg<sup>-1</sup>) | 5.563<sup>b</sup> | 8.074<sup>b</sup> | 8.582<sup>b</sup> | 8.602<sup>b</sup> |
| p Value           | .019                              | .0270                               | .0130                       | .0250                        |
| SEM               | 0.0380                             | 0.0700                               | 0.0180                      | 0.0440                        |

*In each column, means with the similar letters are not significantly different (p < .05).
SEM: standard error of the mean; CFU: colony-forming unit.

Microbial flora of caecum

The effects of using fenofibrate in the diet of broilers on the microbial flora of the caecum are shown in Table 8. According to the results, the use of fenofibrate reduced the bacteria of Clostridium, E. coli and Coliform (p < .05), while the use of 50 and 100 mg kg<sup>-1</sup> fenofibrate significantly increased the population of lactobacilli (p < .05).

Discussion

Fenofibrate supplementation in feed during the starter, grower, finisher and whole of the period in this study resulted in a significant reduction in feed intake with affecting the weight gain of chickens, leading to improved FCR compared to control group but in the study of Konig et al. (2007) which used clofibrate, a fibrin acid derivative, in the diet of laying hens, is reported that consuming clofibrate in the diet reduced feed intake and egg production. In disagree with our study, application of 100 mg kg<sup>-1</sup> fenofibrate...
in rat diets reduced feed intake and weight gain (Lee et al. 2002). Mancini et al. (2001) reported that fenofibrate reduced and stabilised weight in the mice fed by a high-fat diet through affecting liver metabolism and catabolism of lipids in liver, which is partly in agreement with this study. Winegar et al. (2001) found that fenofibrate had no significant effect on feed intake and weight in rhesus monkeys.

Application of 100 mgkg⁻¹ fenofibrate in rat diets reduced weight and ventricular fat (Lee et al. 2002). It can be concluded that fenofibrate reduces ventricular fat and also FCR by increasing fat metabolism.

Effect of fenofibrate on blood parameters in this study was not significant. Konig et al. (2007) reported that clofibrate in the diet of laying hens, reduces serum triglycerides but had no significant effect on serum cholesterol. Serisier et al. (2006) stated that the application of 100 mgkg⁻¹ fenofibrate in the diet of obese dogs reduced serum lipid concentrations. Winegar et al. (2001) reported that the use of fenofibrates reduced triglycerides and LDL and increased HDL in rhesus monkeys. Balfour et al. (1990) reported that fenofibrate reduces VLDL and LDL by affecting cholesterol metabolism and facilitates the excretion of excess cholesterol by the liver. Some researchers have stated that fenofibrates reduce the accumulation of fat in the body by lowering cholesterol synthesis in the liver as well as increasing the catabolism of triglyceride-rich lipoproteins (Balfour et al. 1990; Shepherd 1994), it seems the same mechanism have been effective in this study. Serum antibody against Newcastle virus numerically increased in this study. Some researchers believe that fenofibrate increases the ability of the body’s antioxidant system by reducing the peroxidation of membrane lipids and increasing the activity of antioxidant enzymes (Sinha et al. 2018) and also may improve the energy utilisation to synthesis of protein, such as antibodies and tissues. Therefore, it can be said that the use of fenofibrate can maintaining the antioxidant and immune system, reduces the stress on the bird and helps maintain the bird’s health and performance.

The mechanism of fenofibrate influence on the microbial flora of the intestine has not been specified yet, but our research has shown that fenofibrate is effective in improving the useful microbial flora population of the intestine.

**Conclusion and suggestion**

In this study, for the first time, the effect of fenofibrate on the quantitative and qualitative characteristics of broilers has been investigated. The results of this study showed that the use of fenofibrate is effective in improving performance, and reducing the population of harmful bacteria in the gastrointestinal tract. Due to the positive effect of fenofibrate on growth and economic performance, it is recommended to use 100 mgkg⁻¹ fenofibrate in the diet of broilers. It is recommended for future researches that the possibility of fenofibrate residues in meat be investigated.

**Ethical protocol**

The experimental protocol was approved by the Animal Ethic Committee of the Rasht Branch, Islamic Azad University, and the experiment was conducted in accordance to the International Guidelines for Research involving animals (Directive No. 2010/63/EU).

**Acknowledgements**

This manuscript is prepared based on PhD thesis of the first author at Rasht Branch, Islamic Azad University, Rasht, Iran.

**Disclosure statement**

The authors declare no conflicts of interest in this work.

**Data availability statement**

The datasets generated during and/or analysed during this study are available from the corresponding author on reasonable request.

**References**

Aviagen. 2018. Ross 308 broiler management. p. 147. www.aviagen.com.

Balfour JA, Mc Tavish D, Heel RC. 1990. Fenofibrate. A review of its pharmacodynamic and pharmacokinetic properties and therapeutic use in dyslipidaemia. Drugs. 40(2): 260–290.

Buyse J, Janssens GP, Decuyper E. 2001. The effects of dietary L-carnitine supplementation on the performance, organ weights and circulating hormone and metabolite concentrations of broiler chickens reared under a normal or low temperature schedule. Br Poult Sci. 42(2):230–241.

Cabel MC, Waldroup PW, Shermer WD, Calabotta DF. 1988. Effects of ethoxyquin feed preservative and peroxide level on broiler performance. Poult Sci. 67(12):1725–1730.

Cakir S, Yalcin S. 2007. Effects of L-carnitine supplementation in diets with low energy level on growth performance and carcass traits in broilers. Vet Med Rev. 158:291–296.

Cartwright AL. 1986. Effect of carnitine and dietary energy concentration on body weight and body lipid of growing broilers. Poult Sci. 65:21–29.
