**INTRODUCTION**

Chicken meat is considered one of the most desirable meats all over the world because of relatively low fat content and high concentration of polyunsaturated fatty acids (PUFA). Furthermore, the existing fatty acid composition and fat contents could easily be altered through dietary manipulation. A high level of polyunsaturation, however, accelerates lipid peroxidation, leading to deterioration of meat odor, color, texture, flavor, and nutritional value (Toufektsian et al., 2011). The meat industry is, therefore, always looking for ultimate dietary strategies to modulate cholesterol and saturated fatty acids (SFA) and also to enrich the meat with oxidative substances to retard lipid oxidation. Currently, the most effective strategy to attain this goal is dietary supplementation of antioxidants, including natural and synthetic ones. Nevertheless, the toxicological safety of these antioxidants is a vital issue raised by regulatory laws and consumers (Tan et al., 2011).

In recent years, natural antioxidants are receiving attention due to their assured safety and cheap availability. These antioxidants are generally recognized as safe (GRAS) by the food regulatory authorities (Kruger and Mann, 2003). These are primarily phenolic and polyphenolic compounds, which generally are found in all plants with frequent abundance in medicinal plants and herbs (Chavan et al., 1999). In several studies, these medicinal plants, herbs, and their extracts have been observed for significant positive effects on meat quality including modulation of fatty acid proportions; however, great contradictions are found in the existing literature about their effective doses (as reviewed by Velasco and Williams, 2011) due to several thousand-fold differences in antioxidant contents of these plants/herbs growing in different geographical locations (Carlsen et al., 2010). Therefore, it is prudent to use these plant antioxidants in extracted (purified) form to determine their effects and practical application in the meat industry.

**ABSTRACT** This study was conducted to investigate the supplemental effects of purified bioflavonoids (genistein and hesperidin), as potential alternatives to plant/herbs or synthetic antioxidants, individually and in combination for fatty acid profile, lipid metabolites, and antioxidant status of broilers. Three hundred sixty 1-d-old broilers were divided into 6 treatment groups: control (basal diet), G5 (5 mg of genistein per kg of feed), and H20 (20 mg hesperidin per kg of feed), whereas the other 3 groups were supplemented with a mixture of genistein and hesperidin (20% genistein + 80% hesperidin) having a dosage of 5 mg·kg⁻¹ (GH5), 10 mg·kg⁻¹ (GH10), and 20 mg·kg⁻¹ (GH20), respectively. Broilers were slaughtered at 42 d, and breast muscle, liver, and blood samples were collected. A dose-dependent increase ($P < 0.05$) was observed for plasma antioxidant parameters, including total antioxidant capacity, malondialdehyde production, and total superoxide dismutase activity. Cholesterol and triglyceride contents were found to decrease ($P < 0.05$) in serum and breast muscle. The proportion of total polyunsaturated fatty acids and the ratio of n-6 to n-3 fatty acids and polyunsaturated fatty acids to saturated fatty acids in breast muscles was significantly improved ($P < 0.05$) by increasing levels of dietary bioflavonoids. The current results implied that dietary bioflavonoids genistein and hesperidin could positively improve the fatty acid and lipid metabolite profile of broiler breast meat in a dose-dependent fashion. Thus, bioflavonoids could be a feasible alternative of antioxidant plants/herbs and synthetic feed additives for the production of healthier chicken meat.

**Key words:** bioflavonoid, antioxidation, lipid metabolite, fatty acid profile, broiler
Among plant antioxidants, bioflavonoids are receiving noticeable attention due to their multifunctional biological activities (Middleton et al., 2000). Bioflavonoids genistein (a soy flavonoid) and hesperidin (a citrus flavonoid) were identified as potential antioxidants in in vitro studies (Rice-Evans and Miller, 1996). Soybean bioflavonoids (mixture of genistein, daidzein, and glycitein) and hesperidin reported as reducing agents (free radical scavengers) in biological systems and protect meat from spoilage through inhibition of malondialdehyde (MDA) production (Jiang et al., 2007; Simitzis et al., 2011). Soybean and citrus bioflavonoids could improve the serum total antioxidant capacity (T-AOC) and total superoxide dismutase (T-SOD) activity, and reduce the MDA production, which are known as the biomarkers of oxidative status, while their irregular levels may lead to production of meat with shorter shelf life (Jiang et al., 2007; Lien et al., 2008). Bioflavonoids are also known to alter the fatty acid profile of goat meat by increasing the ratio of PUFA to SFA and n-6 to n-3 fatty acids (Tan et al., 2011). Modulatory effects of bioflavonoids for cholesterol and lipids have been studied in several animal models, including goats (Tan et al., 2011), rabbits (Cavalli et al., 2009), and laboratory animals (Toufektsian et al., 2011). In some other studies, larger molecules of bioflavonoids called tannins have been investigated for their lipid modulatory effects in lambs (Vasta et al., 2007) and rabbits (Liu et al., 2009). To the best of our knowledge, the supplemental effects of bioflavonoids in broilers for fatty acid profile of meat are largely unrevealed. Therefore, in present study it was planned to explore the dietary effects of 2 purified bioflavonoids (i.e., genistein and hesperidin) on fatty acid profile, lipid metabolites, and antioxidant status of broiler chickens. We also supplemented both bioflavonoids in combination (same proportion as used individually) to get insight of the effects of combinatorial treatments.

**MATERIALS AND METHODS**

**Birds and Grouping**

All the experiments were conducted according to guidelines of Animal Ethics and Use Committee of the Nanjing Agricultural University, China. Three hundred sixty mixed-sex Arbor Acre broiler chicks were purchased from a local hatchery market at 1 d of age. These were randomly divided into 6 treatment groups. Each treatment had 6 replicates containing 10 broilers, assigned to separate pens, and housed in a full environmentally controlled room. Twenty-four hours of fluorescent lightening with standard conditions of temperature, humidity, and ventilation were provided for the entire experimental period. Broilers were fed the starter diet from d 1 to 21, and the finisher diet from d 22 to 42, which contained CP of 20.72 and 18.93%, respectively, and ME of 3,121.9 and 3,188 kcal·kg$^{-1}$, respectively. Other feed ingredients were adjusted according to the standard (NRC, 1994). Fatty acid compositions of diet are shown in Table 1. All broilers had free access to both feed and water.

**Dietary Treatments**

Purified bioflavonoids genistein and hesperidin were purchased from a commercial source (Sigma Chemical Co., St. Louis, MO) with 98% purity. Purity was further confirmed before their supplementation to broiler diets. Their supplemental doses were adjusted based on previous dosage trials (unpublished data). Broilers in control group were fed with no additive, whereas other groups were fed with 5 mg·kg$^{-1}$ genistein (G5), 20 mg·kg$^{-1}$ hesperidin (H20), or a mixture of both compounds (20% genistein + 80% hesperidin) in doses of 5 mg·kg$^{-1}$ (GH5), 10 mg·kg$^{-1}$ (GH10), and 20 mg·kg$^{-1}$ (GH20), respectively.

**Sample Collection**

At 42 d of age, 2 broilers from each replicate were randomly selected and killed by exsanguination. Blood samples were collected in heparinized (30 IU·mL$^{-1}$) and nonheparinized tubes to analyze the antioxidant and lipid profile differences among dietary treatments. They were immediately centrifuged (3,000 × g at 4°C), and plasma/serum were isolated and stored at −20°C until analyzed. Liver and breast muscle (pectoralis major) were collected and quickly stored at −20°C for cholesterol and fatty acid profile analysis.

**Assay of Plasma Antioxidant Indices**

To monitor the anti-oxidative status of broilers, T-AOC, MDA, and T-SOD were analyzed by using commercial kits (Nanjing Jiancheng Bioengineering Institute, Nanjing, China). All samples were analyzed in triplicate according to the instructions of the kit manufacturers. The T-AOC was calculated through the method of ferric reducing-antioxidant power assay.

### Table 1. Fatty acid composition (% of total fatty acids) of basal diets

<table>
<thead>
<tr>
<th>Fatty acid$^1$</th>
<th>Starter (1 to 21 d)</th>
<th>Grower (22 to 42 d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:0</td>
<td>0.17</td>
<td>0.18</td>
</tr>
<tr>
<td>16:0</td>
<td>12.58</td>
<td>12.30</td>
</tr>
<tr>
<td>18:0</td>
<td>4.09</td>
<td>3.16</td>
</tr>
<tr>
<td>9c16:1</td>
<td>2.02</td>
<td>1.98</td>
</tr>
<tr>
<td>9c18:1</td>
<td>26.25</td>
<td>26.14</td>
</tr>
<tr>
<td>9c12:18:2n-6</td>
<td>52.4</td>
<td>53.72</td>
</tr>
<tr>
<td>α-18:3n-3</td>
<td>2.95</td>
<td>2.88</td>
</tr>
<tr>
<td>γ-18:3n-6</td>
<td>4.67</td>
<td>4.61</td>
</tr>
<tr>
<td>20:4n-6</td>
<td>0.6</td>
<td>0.61</td>
</tr>
<tr>
<td>22:4n-6</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>22:6n-3</td>
<td>0.07</td>
<td>0.06</td>
</tr>
</tbody>
</table>

$^1c = cis.$
acid to the internal standard. The relative percentage of identified fatty acids was then calculated based on the ratio of the fatty acid with pressure of 80 kPa. The fatty acids were identified through nonenzymatic NBT test, which measures the inhibition of formation of superoxide anion free radicals that reduce the nitroblue tetrazolium of the sample (Winterbourn et al., 1975). The MDA was measured as a decomposed product of lipid peroxidation with 2-TBA by using the colorimetric method with a spectrophotometer at a wavelength of 532 nm (Placer et al., 1966).

**Lipid Metabolites Assay**

Lipid metabolites including total cholesterol (CHO), triglycerides (TG), high-density lipoprotein cholesterol (HDLC), and low-density lipoprotein cholesterol (LDLC) were analyzed in serum. Subsequently, CHO and TG levels were also analyzed for liver and breast muscle. Same kits were used for both blood and tissues. All the procedures were done according to the assay kits (Beijing BHKT Clinical Reagent Co., Beijing, China). Readings for CHO and TG were taken spectrophotometrically by tracking at corresponding wavelengths according to previously published methods (Meiattini et al., 1978; Fossati and Prencipe, 1982), whereas HDLC and LDLC were determined using the method of Rifai et al. (1992). All measurements were conducted in same assay to avoid interassay variability. Protein contents of tissues were also determined by using Biruet reagent (Nanjing Jincheng Bioengineering Institute) to express the results in micromoles per gram of protein.

**Breast Muscle Fatty Acid Determination**

The fatty acid composition of diets and breast muscle was analyzed by using the method of Tan et al. (2011) with slight modifications. In brief, a 2-g sample was taken and total lipids were extracted by using chloroform–methanol (2:1, vol/vol) according to the procedure of Folch et al. (1957). After extraction, fatty acid methyl esters were prepared for gas chromatography by treating with BF₃ methanol. Gas chromatography analysis of methylated fatty acids was performed by using a gas chromatograph (GC-14B, Shimadzu, Japan) equipped with a flame ionization detector and a split injector. Separation was carried out on a CP-Sil88 fused silica open tubular capillary column (50 m × 0.25 mm × 0.20 μm). The oven temperature was increased from 140 to 220°C at 6°C min⁻¹ and held for 30 min at 280°C. The injector and detector temperatures were maintained at 280°C. Nitrogen was used as the carrier gas with pressure of 80 kPa. The fatty acids were identified by comparison of their retention times with those of standards. The relative percentage of identified fatty acids was then calculated based on the ratio of the fatty acid to the internal standard.

**Statistical Analysis**

All the data were analyzed by ANOVA through the JMP statistical package software (Version 5.0.1.a, SAS Institute Inc., Cary, NC), and results were presented as means ± SEM. Tukey-Kramer honestly significant difference multiple comparison test was applied to compare the significant difference among groups. A level of $P < 0.05$ was considered as a limit for statistical significance.

**RESULTS**

**Plasma Antioxidant Enzyme Activities**

Supplementation of purified bioflavonoids improved $(P < 0.01)$ the T-AOC of plasma up to 54.82% (GH20 group) in comparison with control group (Figure 1A). Overall, a dose-dependent increase was observed for all groups except G5 that showed a 12.99% reduction in T-AOC activity. The MDA level was tremendously depressed $(P < 0.01)$ by the increasing levels of dietary bioflavonoids with maximum reduction of 58.58% by the GH20 group (Figure 1B). Nevertheless, a significant effect $(P < 0.01)$ was observed only for combined doses of both compounds (GH5-GH20) in comparison with the control group. The T-SOD activity was increased by the increasing levels of dietary treatments (Figure 1C), but only the GH20 group showed a significant $(P < 0.05)$ improvement in plasma T-SOD activity (by the 16.97%) in comparison with the control group.

**Serum Lipid Profile**

The results for serum CHO, TG, HDLC, and LDLC are summarized in Figure 2 (A–D). Dietary flavonoids produced a dose-dependent decrease $(P < 0.05$ or $P < 0.01$) in CHO, TG, and LDLC with maximum reduction of 17.98, 15.03 and 25.22%, respectively. In comparison with the control group, a significant reduction $(P < 0.01$) in CHO and LDLC levels was observed for high dose groups (H20 and GH20). The TG level decreased $(P < 0.05$) for only the H20 group compared with the control group. Other groups including medium- (GH10) and low-dose (G5, GH5) groups also showed a reduction in TG levels, but it was statistically nonsignificant $(P > 0.05$). The HDLC level was increased significantly $(P < 0.05)$ by the dietary bioflavonoids in the GH20 group in comparison with the control group (Figure 2C).

**Lipid Metabolites of Liver and Breast Muscle**

Dietary bioflavonoids decreased $(P < 0.01)$ the CHO contents of liver from 10.22 to 23.46% by the low-dose groups (G5, GH5) and 32.7 to 39% by the high-dose groups (H20, GH20) in comparison with the control
group (Figure 3A). Overall, this reduction was accelerated with the increasing levels of bioflavonoids. The TG contents of liver were also reduced by the bioflavonoids; however, this effect was statistically nonsignificant ($P > 0.05$). Similarly, decreasing trends for CHO and TG contents of breast muscle were recorded in this study (Figure 3B). The CHO content decreased significantly ($P < 0.01$) for G5, H20, and GH20 groups in comparison with the control group, whereas the TG contents reduced ($P < 0.05$) in a dose-dependent fashion for combined doses of bioflavonoids, with maximum reduction in the GH20 group (38.76%) compared with other groups.

**Breast Muscle Fatty Acid Composition**

The influence of dietary bioflavonoids on fatty acid composition of broiler breast muscle is shown in Table 2. For SFA, increasing levels of combinatorial genistein and hesperidin supplementation significantly reduced ($P \leq 0.005$) the fatty acid proportions of 14:0 and 18:0, with maximum reduction of 30 and 8.7%, respectively, by the GH20 group, but no effect was recorded for the 16:0 fatty acids. In comparison with the control group, all genistein- and hesperidin-supplemented groups (except H20 for 14:0 and Σ SFA, and GH5 for 18:0) significantly decreased ($P < 0.01$) the fatty acid proportions of 14:0, 18:0, and SFA of breast muscle. For MUFA, a dose-dependent reduction was recorded in the fatty acid proportions of 9-cis16:1 and 9-cis18:1. In comparison with the control group, fatty acid proportions of 9-cis18:1 were found to be decreased ($P < 0.05$) in all genistein- and hesperidin-supplemented groups except the G5 group. For PUFA, when compared with the control group, the GH10 and GH20 groups had higher ($P < 0.05$) fatty acid ratios of 9-cis,12-cis18:2 n-6 and 20:4n-6, whereas the GH10 group had higher ($P < 0.05$) proportions of PUFA. The GH20 group had lower ($P < 0.01$) 22:6n-3; H20, GH5, and GH10 groups had lower n-3 ($P < 0.01$) proportions compared with the control group. The dietary effect of bioflavonoids was not observed for the fatty acid proportions of α-18:3n-3, γ-18:3n-6, and 22:4n-6. The ratio of n-6 to n-3 fatty acids was found to be improved ($P < 0.01$) by all combined genistein- and hesperidin-supplemented groups (GH5, GH10, and GH20) with maximum effect of 37% by the GH20 group, whereas the ratio of PUFA to SFA was elevated ($P < 0.05$) in the GH20 group (31.9%) compared with the control group.

**DISCUSSION**

In recent years, epidemiological work and laboratory experiments have been targeting the causes and modifying the factors associated with cardiovascular diseases (CVD). It is widely acknowledged that the incidence of CVD is closely related to dietary intake of cholesterol and SFA contents. Therefore, much attention has been paid toward the modulation of animal products to reduce the risk of heart diseases and to enrich the products with bioactive compounds, such as antioxidants, to improve product quality and protect consumers’ health from oxidant-mediated diseases (Simitzis et al., 2011). In this study, we used bioflavonoids in purified form, which are the proven natural antioxidants under in vivo and in vitro conditions (Chavan et al., 1999; Fellenberg and Speisky, 2006). The study showed that the T-AOC level was relatively higher for all supplementation levels; even a low level (5 mg·kg$^{-1}$) of combined genistein and hesperidin was able to cause high plasma T-AOC activity (Figure 1A). Overall, a dose-dependent increase in total antioxidant activity was noted in all bioflavonoid-supplemented groups. This finding could
be correlated with a recent study of Jiang et al. (2007), who reported a dose-dependent increase in the plasma total antioxidative activity of broilers by dietary levels of 10 to 80 mg·kg⁻¹ of soy flavonoids. The T-AOC is a cumulative measure of body redox status and is known as a useful indicator for determining the fate of supplemental antioxidants (Benzie and Strain, 1996). It is well known that the −OH group in flavonoid structure acts as hydrogen donor to the peroxyl radicals produced during the oxidation, thus impeding hydroxyl peroxide formation (Fellenberg and Speisky, 2006). This structure-oriented antioxidative activity of bioflavonoids has widely been acknowledged in several studies (Chavan et al., 1999; Liu et al., 2009) and also been recognized in our work. A recent study also reported the significant promotion of plasma total antioxidant capacity in laying hens by the supplementation of a bioflavonoid naringenin (Lien et al., 2008).

We also found an increased T-SOD and reduced MDA production in plasma with increasing levels of bioflavonoids genistein, hesperidin, or both. Our results could be correlated with a previous study of isoflavonoids in male broilers (Jiang et al., 2007); who associated high levels of flavonoids (i.e., ≥20 and 40 mg·kg⁻¹ dosages) for significant modulation of MDA and T-SOD, respectively. In our study, however, all combined doses of bioflavonoids, even low and medium doses (5 and 10 mg·kg⁻¹), reduced the plasma MDA production. This phenomenon may explain the tendency of bioflavonoids to form synergism as reported in previous studies (Alvarez et al., 2008). Nevertheless, the synergism between these bioflavonoids (genistein and hesperidin) is yet needed to be investigated through specially designed projects. Similar positive changes in plasma MDA and T-SOD levels as regarded in our study were reported in a study of layer chickens fed with citrus flavonoids (Lien et al., 2008). Furthermore, some other studies reported the improved antioxidation and free radical scavenging activity by the dietary bioflavonoids in rabbits (Jeon et al., 2001), mice (Kim et al., 2004), and turkeys (Ting et al., 2011).

It is generally recognized that modulation of fatty acids scenario of poultry products through dietary strategies is relatively easier than the reduction of cholesterol contents due to the complex mechanism of cholesterol biosynthesis, regulation, and its metabolism (Ting et al., 2011). Interestingly, our dietary purified bioflavonoids reduced the CHO and TG concentrations in liver and breast muscle with increasing levels of supplements, which is a novel finding of this study. The re-
duced CHO and TG level of meat could be attractive for consumers because it may reduce the risk of CVD. The dramatic reduction in cholesterol (up to 39% in liver and 30.6% in muscle) indicates the inhibitory effect of dietary bioflavonoids for the enzyme HMG-CoA reductase, a key enzyme for cholesterol biosynthesis (Lien et al., 2008). The reduced CHO might be due to the decreased absorption of cholesterol and bile acids from the intestinal lumen (Cavallini et al., 2009). Ting et al. (2011) observed a dose-dependent decrease of CHO and TG concentrations in serum by the supplementation of 2 bioflavonoids, naringenin and hesperetin. Authors also reported a slight reduction in egg yolk cholesterol level in supplemented groups. Similar results were also reported by a previous study of laying hens (Lien et al., 2008). On the other hand, Cavallini et al. (2009) reported that bioflavonoid isoflavone failed to reduce CHO level in hypercholesterolemic rabbits. This might be due to species variation because it is well recognized that flavonoids affect the cells by a physiological stimulus (which might be species dependent) and generate a flavonoid-sensitive substance that interacts with flavonoids and alters the outcome of the activation process (Middleton et al., 2000).

Animal experiments have depicted that citrus flavonoids were absorbed, metabolized, and biologically active in dose-dependent manners (Zhou et al., 2008). In human subjects, these were known to reduce the serum concentrations of CHO, apolipoprotein B, and TG, which are the building blocks of low-density lipoprotein cholesterol (Roza et al., 2007). In our study, we also recorded a dose-dependent decrease in serum CHO, TG, and LDLC contents by the dietary levels of purified soy and citrus bioflavonoids. The LDLC is also known as bad cholesterol because it is associated with atherosclerosis, which is the principal cause of coronary heart disease. Some previous studies have also documented that bioflavonoids could decrease cholesterol synthesis by delaying ACAT activity in HepG2 cells (Borradaile et al., 1999), which reduce the hepatic production of apo-B containing lipoproteins, such as VLDL, thereby reducing the serum LDLC concentration (Burnett et al., 1998).

The fatty acid proportion of meat is considered an important index for meat quality regarding CVD. There is no information available for the supplemental effects of bioflavonoids on chicken meat fatty acids. In present study, dietary inclusion of purified genistein and hesperidin to broilers changed the breast muscle fatty acid proportion in a positive manner (reduced SFA and increased PUFA). These results were supported by recent studies indicating that tea flavonoids could reduce the SFA and increase the PUFA in goat meat (Tan et al., 2011). Similar findings were reported by Vasta et al. (2007) in sheep by dietary inclusion of antioxidant tannins. Jung et al. (2010) also reported a reduced proportion of SFA and MUFA, and increased PUFA concentration in broilers breast meat by supplementation of a mixture of polyphenolics (gallic acid) with linoleic acid. The authors concluded that in poultry, an increased concentration of PUFA could reduce the synthesis of MUFA by inhibiting the activity of 9-de-saturase complex, a key enzyme that converts SFA to MUFA, and supplementation of antioxidants is a prime strategy to reduce SFA and to increase PUFA proportions in chicken meat.

It is well established that levels of free radicals exceeding the capacity of the cellular intrinsic free radical scavenging system could cytotoxically attack the cellular fatty acids and result in the onset of lipid peroxidation of membranes. Results from present study well demonstrated that genistein and hesperidin could sufficiently inhibit blood lipid oxidation (MDA production) in broilers, thus protecting the peroxidation of oxidative-labile PUFA rather than that of more stable SFA in meat. These findings are in agreement with the results of Hrelia et al. (2002) in cultured cardiomyocytes. Similar relationship between antioxidation and SFA/PUFA proportions had been reported in a recent
study of broiler chickens by analyzing the breast meat antioxidative potential and fatty acid contents (Jung et al., 2010).

Several researchers have reported that ruminant meats have a several-fold lower PUFA:SFA ratio than chicken meat due to extensive biohydrogenation of PUFA by rumen microorganisms (Tan et al., 2011). A bacterial species called Butyrivibrio fibrisolvens is known as most responsible for rumen biohydrogenation (Maia et al., 2010), which has also been identified in chicken microbiota (Zhu et al., 2002). In the present study, we observed a marvelous increase (up to 31.9%) in PUFA:SFA ratio in broilers breast muscle by the supplementation of purified bioflavonoids, which may indicate the protective effect of genistein and hesperidin for PUFA from/through chicken microorganisms (as reviewed by Laparra and Sanz, 2010). This needs to be investigated further by using the advanced knowledge of chicken functional microbiota. Some studies have suggested that increased PUFA:SFA and n-6:n-3 ratios in meat could be due to the protective role of dietary antioxidants, as these antioxidants act as electron donors to provide electrons for reduction of some unsaturated fatty acids, and to be metabolized to these donors by microorganisms (Chikunya et al., 2004).

In conclusion, to our knowledge, this is first study that reported the individual and combined effects of purified dietary bioflavonoids (genistein and hesperidin) on plasma antioxidative status, lipid metabolites, and fatty acid profile of broiler breast meat. The proportions of PUFA and the ratio of PUFA to SFA increased; whereas TG and CHO contents decreased in breast muscle with increasing levels of bioflavonoids. As potential antioxidants, both bioflavonoids seemed to promote the plasma antioxidative status in a dose-dependent manner. Thus, bioflavonoids genistein and hesperidin could be a potential alternative of antioxidant plants/herbs and synthetic feed additives for production of healthier chicken meat. Pronounced effects of combined doses might indicate the potential of both bioflavonoids to produce a synergism. This phenomenon needs to be explored further in in vitro and in vivo meat systems.

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REFERENCES


