ABSTRACT  In the United States, supplemental dietary lipid is typically provided as an animal-vegetable blend using animal tallow or hydrogenated oils from the food industry. Lipids from these sources are rich in saturated, trans, n-6 fatty acids and poor in n-3 fatty acids. Linoleic (18:2 n-6) and α-linolenic (18:3 n-3) acids are essential fatty acids and are the precursors of long-chain n-6 and n-3 fatty acids such as arachidonic and eicosapentaenoic acids (EPA). Ester-linked arachidonic acid and EPA can be mobilized by phospholipase A2 to generate free arachidonic acid and EPA, which can act as substrates for cyclooxygenase and lipoxygenase to produce eicosanoids. Eicosanoids derived from arachidonic acid, prostaglandin E2, thromboxane B2, and leukotriene B4 are proinflammatory and more potent than eicosanoids derived from EPA such as prostaglandin E3, thromboxane B3, and leukotriene B5. Developing dietary strategies in broiler chickens that enhance the n-3 fatty acid content of tissues is also associated with lipid oxidation and muscle product quality. Therefore, alternative strategies for enhancing tissue n-3 fatty acid content without affecting growth and product quality must be devised. The role of maternal (yolk) fatty acids in modulating the long-chain n-3 fatty acid content of tissues and eicosanoid production in chickens fed a diet lacking in long-chain n-3 fatty acids is investigated. Up to d 42 of growth, the cardiac tissues of chicks hatched from hens fed a high n-3 diet retained higher levels of long-chain n-3 fatty acids than those of chicks hatched from hens fed a low n-3 diet. Chicks hatched from hens fed a high n-3 diet produced less proinflammatory eicosanoids than chicks hatched from hens fed a low n-3 diet. Modulating maternal dietary n-3 fatty acids enhances tissue retention of n-3 fatty acids during growth and reduces proinflammatory eicosanoid production in chicks, which could lead to fewer metabolic and inflammatory-related disorders in poultry.

Key words: metabolic disorder, broiler, eicosanoid

INTRODUCTION

Because of advances in genetic selection, management, and nutrition, the modern-day commercial broiler chicken and turkey have fast growth rates, and high feed conversion ratios and metabolic rates. These features promote an increased workload on the cardiovascular system predisposing birds to metabolic disorders such as right ventricular failure, ascites syndrome, cardiac arrhythmias, cardio-pulmonary disorders, and sudden death (Julian, 2005). Though physiological and pathological aspects of metabolic and cardiovascular diseases in broiler birds have been investigated in detail (Wideman, 2000, 2001; Olkowski et al., 2005), the role of diet in the etiology of metabolic disorders has been limited to energy or quantity restriction, feed type (mash vs. pellet), micronutrients (vitamins C, E, coenzyme Q10, Zn), or fat source (sunflower vs. tallow; Rotter et al., 1985; Bottje et al., 1997; Geng et al., 2004; Virden et al., 2004).

Lipids and Fatty Acids in Metabolic Disorders

Much of the information available on the role of lipids and growth-related metabolic disorders was derived from experimentally induced birds. Assessing the fatty acid (FA) status of turkeys with furazolidone-induced dilated cardiomyopathy, Lax et al. (1994) reported an increase in arachidonic acid (20:4 n-6) in the cardiac ventricles. Similarly, feeding flax, a rich source of n-3 FA to broiler birds, Walton et al. (1999) reported reduction in right ventricular hypertrophy and blood viscosity in birds exposed to hypobaric conditions. Hypertrophy of the right ventricle is an indication of right ventricular failure and pulmonary hypertension (Julian and Wilson, 1986), and higher whole blood viscosity is associated with greater blood flow resistance.

Information on the lipid and FA composition of broiler birds with other metabolic disorders such as sudden death is very limited because of the lack of consistency in symptoms that allow sudden death identification. In addition, postmortem changes associated with tissue au-
tolysis may affect tissue lipid composition. Riddell and Orr (1980) noted that birds dying from sudden death had an increase in serum total lipids. Similarly, Chung et al. (1993) reported lower phosphatidyl ethanolamine and total phospholipids in the cardiac tissue of sudden death birds than in pen-mate controls. Assessing the cardiac FA status of sudden death birds, Buckley et al. (1987) reported an increase in arachidonic acid in the cardiac lipids of broilers dying from sudden death when compared with control birds of the same age. Similarly, a significant increase in oleic acid along with a decrease in eicosapentaenoic acid (EPA, 20:5 n-3) and total long-chain n-3 FA was observed in sudden death birds compared with pen-mate control birds by Cherian et al. (2005; Figure 1). The significant alterations in total fat, monounsaturated, long-chain n-6, and n-3 FA reported in these studies point to a role of dietary FA in metabolic disorders of the cardiovascular system. The current review will focus on the role of lipids with emphasis on polyunsaturated FA in poultry diets and their eicosanoid-related metabolic functions.

Lipids and FA in Current Broiler Diets

Dietary lipids contribute a large number of calories for fast growing broiler birds. The current trend in formulating high-energy rations for broiler birds has resulted in dietary supplementation of lipids over 6%. Dietary lipids also provide FA for synthesis of cell membranes. In the United States, supplemental dietary lipid is typically provided as an animal-vegetable blend using tallow from rendering sources and restaurant grease or hydrogenated oil from the food industry. Lipids from these sources are rich in saturated, trans, and n-6 FA and poor in n-3 FA. A list of fats or oils used in poultry feeds and their FA compositions is shown in Table 1.

Fatty acids of the n-3 family have received considerable attention in the past decade for their cardio-protective effects (Leaf et al., 2003). In addition, human clinical studies have provided evidence that baseline blood levels of long-chain n-3 FA are inversely related to the risk of sudden death in men with no histories of prior cardiovascular disease (Albert et al., 2002). The n-3 FA may manifest cardio-protective effects by incorporation into cell membrane phospholipids, changing membrane properties and altering eicosanoid metabolism (Nair et al., 1997). Given the fact that n-3 FA are known to have several health-promoting effects (Kris-Etherton et al., 2003), the possible role of n-3 FA in modulating inflammatory pathways in broiler chickens is being investigated.

Lipids as Sources of Essential n-6 and n-3 FA

Linoleic (18:2 n-6) and α-linolenic (18:3 n-3) acids are essential FA and are the precursors of bioactive long-chain (>20-carbon) n-6 and n-3 FA. The endogenous formation of arachidonic acid (20:4 n-6) and EPA (20:5 n-3) from linoleic and α-linolenic acid occurs through desaturation and subsequent chain elongation. Arachidonic acid, EPA, and docosahexaenoic acid (DHA, 22:6 n-3) are the major long-chain n-6 and n-3 FA in broiler tissues and cells. The efficacy of linoleic and α-linolenic acids in synthesizing 20-carbon FA depends on factors such as the concentration of n-6 FA and the ratio of n-6 to n-3 FA. Competitive inhibition of enzymes will occur, depending on which substrate is present in high concentration.

Essential FA as Eicosanoid Precursors

Ester-linked arachidonic acid and EPA in cell membrane phospholipids can be mobilized by phospholipase A2 to generate free arachidonic acid and EPA, which can act as substrates for cyclooxygenase and lipoxygenase to produce prostaglandins and thromboxane or leukotrienes and hydroxyeicosatetraenoic acid. Eicosanoids derived from arachidonic acid, prostaglandin E2 (PGE2), thromboxane B2, and leukotriene B4 (LTB4) are more proinflammatory and have been shown to increase vascular permeability, vasodilation, edema, release of lysosomal enzymes, generation of reactive oxygen species, and production of inflammatory cytokines (Calder, 2006). Eicosanoids derived from EPA, prostaglandin E3, thromboxane B3, and leukotriene B5 (LTB5) are less inflammatory and are less potent than n-6 derived eicosanoids. In addition, recent studies have identified a novel group of mediators, E-series of resolvins and docosatrienes formed from long-chain n-3 FA, that appear to exert antiinflammatory effects (Serhan and Arita, 2004).

Dietary Lipids: Effect on Tissue Eicosanoid Precursors

Chickens are monogastric animals, so lipids are absorbed and deposited in tissues mainly in the form in which they are ingested. Therefore, the FA composition of chicken tissues can be influenced readily by dietary FA. When fed a typical commercial diet, arachidonic acid

Figure 1. Total long-chain n-3 fatty acid content in the hepatic and cardiac tissues of birds dying due to sudden death compared to control birds (n = 16). P < 0.05.
Table 1. Fatty acid composition of the oil sources used in poultry rations

<table>
<thead>
<tr>
<th>Fat source</th>
<th>Saturated fatty acids</th>
<th>Polyunsaturated fatty acids</th>
<th>Monounsaturated fatty acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restaurant grease</td>
<td>21.4</td>
<td>23.3</td>
<td>2.6</td>
</tr>
<tr>
<td>Canola oil</td>
<td>7</td>
<td>22</td>
<td>10</td>
</tr>
<tr>
<td>Flaxseed oil</td>
<td>10</td>
<td>17</td>
<td>55</td>
</tr>
<tr>
<td>Safflower oil</td>
<td>10</td>
<td>76</td>
<td>Trace</td>
</tr>
<tr>
<td>Sunflower oil</td>
<td>12</td>
<td>71</td>
<td>1</td>
</tr>
<tr>
<td>Corn oil</td>
<td>13</td>
<td>57</td>
<td>1</td>
</tr>
<tr>
<td>Soybean oil</td>
<td>15</td>
<td>54</td>
<td>8</td>
</tr>
<tr>
<td>Cottonseed oil</td>
<td>27</td>
<td>54</td>
<td>Trace</td>
</tr>
<tr>
<td>Beef tallow</td>
<td>48</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Palm oil</td>
<td>51</td>
<td>10</td>
<td>Trace</td>
</tr>
<tr>
<td>Fish oil</td>
<td>16.8</td>
<td>10.9</td>
<td>26.4</td>
</tr>
<tr>
<td>Menhaden fish oil</td>
<td>26.9</td>
<td>2.2</td>
<td>29.5</td>
</tr>
</tbody>
</table>

1Values reported as percentages (weight of total fatty acids) and subject to change due to differences in batch, cultivars, or processing methods used.

is usually the dominant substrate available for eicosanoid synthesis, hence the predominant bioactive FA in broiler tissues. For example, cardiac lipids of broiler birds fed a commercial broiler diet contain 12 to 15% of FA as arachidonic acid. When sunflower oil (high n-6) or fish oil (high n-3) was included in the diet at 5%, arachidonic acid constituted 31% of cardiac phosphatidyl ethanolamine compared with 23% in fish oil-fed birds. The EPA constituted 10.1% of cardiac phosphatidyl ethanolamine of fish oil-fed birds compared with 0% in sunflower oil-fed birds (Table 2). These results demonstrate that the substrate availability of eicosanoid precursors in cell membrane phospholipids is dependent on dietary n-6 and n-3 FA. The mechanism by which n-6 and n-3 FA can alter membrane lipid composition and thus affect cellular function is shown in Figure 2.

**Dietary n-6 and n-3 FA and Eicosanoid Formation**

The effect of feeding n-6 and n-3 FA on eicosanoid production in poultry was investigated. In breeder hens, feeding sunflower oil rich in n-6 FA led to a significant increase in ex vivo LTB4 production by stimulated thrombocytes compared with those birds fed fish oil. Similarly, thrombocyte LTB5 generation was higher in breeder hens fed diets containing 3% fish oil compared with birds fed 1.5% fish oil diets (P < 0.05; Jha, 2004). Therefore, developing dietary strategies in poultry that enhance the n-3 FA content of tissues may lead to increased incorporation of n-3 FA in cell membrane phospholipids and reduce the production of proinflammatory eicosanoids. However, increasing n-3 FA in broiler chickens is also associated with lipid oxidation and issues of muscle product quality. Therefore, alternative strategies for enhancing tissue n-3 FA content without affecting growth and product quality must be devised.

**Maternal Dietary Lipids and Progeny FA Metabolism**

In oviparous species, the embryo is dependent upon nutrients stored in the egg for sustaining its growth and development into a healthy hatchling. Once a fertilized egg is incubated, lipid-rich yolk is the only source of FA available for the developing embryo. During the 21-d incubation period, over 80% of yolk FA are absorbed by...
Figure 3. Posthatch changes in the docosahexaenoic content (22:6 n-3) in the cardiac tissue of broiler birds as influenced by maternal diet. High, low, or no represents maternal diet containing 3% fish oil, 1.5% fish oil + 1.5% sunflower oil, or 3% sunflower oil. Means ± SEM (n = 6) are shown. At all days, DHA content was significantly (P < 0.05) higher for chicks hatched from hens fed the 3% fish oil diet than 3% sunflower oil diet.

Figure 4. Posthatch changes in the hepatic docosahexaenoic acid (22:6 n-3) content in progeny from birds fed high or no n-3 diet raised on a high n-3 diet, hens and progeny on high n-3 diet (H-H); hens on no omega-3 diet, progeny on high n-3 diet (L-H). Means ± SEM (n = 6) are shown. a,b Significantly different at each time points (P < 0.05).

Eicosanoid Production in Progeny: Effect of Pre- and Posthatch Diets

Eicosanoid production was assessed in chicks hatched from hens fed high or low n-3 diets. Thrombocytes of chicks hatched from hens fed a high n-3 diet produced more LTB5 at 7 and 14 d compared with chicks from hens fed a low n-3 diet. The ratio of LTB5 to LTB4 concentration was also higher (P < 0.05) in chicks hatched from hens fed a high n-3 diet when compared with chicks from hens fed a low n-3 diet at 7 and 14 d (Hall et al., 2007). Because broiler birds are raised commercially for 5 to 6 wk, and considering the role of eicosanoids in inflammatory pathways, these results, together with those of previous studies (Wang et al., 2002; Ajuyah et al., 2003), support the view that in ovo modulation of n-3 FA exerts positive health effects during various stages of posthatch life.

CONCLUSIONS

Metabolic, cardiopulmonary disorders and sudden death are the major causes of mortality in broiler chickens. In addition, assessing mortality between catching and the moment of slaughter, Nijdam et al. (2006) reported that heart and circulation disorders play an important role in the incidence of dead-on-arrival birds at the slaughterhouse. An increase in the prevalence of abnormal right ventricle:total ventricle ratio was also observed on dead-on-arrival birds by these authors. The health and survival of broiler birds in part depend on the birds’ ability to respond effectively and appropriately to external (diet, fast-growth, hatching/crating/transportation), internal (oxidative stress) or other environmental [temperature, confinement, air quality (dust, fumes, CO, CO2, ammonia), pathogens] challenges. Inflammation has been described as the basis of many pathologies and is the host’s immediate response to internal or external tissue injury.
Maxwell et al. (1986) reported an increase in immature and mature heterophils in the hearts and livers of ascitic broilers compared with those of controls, suggesting that an inflammatory response occurs in birds suffering from ascitic syndrome. In view of the role of n-3 FA in modulating inflammation and eicosanoids, further studies on the role FA and eicosanoids play in the etiology of metabolic and cardiovascular disorders need to be conducted.

ACKNOWLEDGMENTS

The author would like to thank the support of National Research Initiative of the USDA Cooperative State Research, Education and Extension Service, grant number 2004-35204-14654. The fish oil used in these studies was kindly supplied by Omega Protein Inc., Reedville, VA.

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