Transfer of Dietary Coenzyme Q10 into the Egg Yolk of Laying Hens

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Coenzyme Q10 (CoQ10), a potent lipophilic antioxidant, is a naturally occurring compound with a ubiquitous distribution in nature, and CoQ10 is used as a dietary supplement to combat aging. There is evidence that lipid soluble nutrients can be transferred into the egg yolk from the feed in laying hens. It is therefore possible that feeding CoQ10 to laying hens increases the content of CoQ10 in the egg yolk. In the present study, we investigated the effect of dietary CoQ10 on its content in the egg yolk of laying hens. Twenty 30 weeks-old Boris Brown hens were assigned based on egg production rate and egg weight to 2 groups (10 birds in each group) and fed a diet containing CoQ10 at 0 or 0.8% for 28 days. Dietary CoQ10 did not affect average egg production rate, feed efficiency, egg weight, and egg yolk weight. CoQ10 content in the egg yolk was increased significantly at 7, 14, 21 and 28 days of the experimental period. Hepatic CoQ10 content and plasma very low density lipoprotein y (VLDLy) CoQ10 concentration in 0.8% group were increased significantly compared with those in 0% group. Dietary CoQ10 significantly decreased cholesterol concentration in the egg yolk at 21st and 28th day of the experimental period. These findings suggest that, in CoQ10-fed laying hens, the increase of hepatic CoQ10 elevates the plasma VLDLy CoQ10 concentration, which in turn results in a high CoQ10 content in the egg yolk of laying hens.

Key words: cholesterol, coenzyme Q10, egg, ubiquinone

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Introduction

Coenzyme Q (CoQ), a potent fat-soluble antioxidant, is a naturally occurring compound with a ubiquitous distribution in nature. Based on the isoprenoid moiety, the presence of various CoQ homologues has been confirmed (Albano *et al.*, 2002; Hihi *et al.*, 2003). CoQ10 has a polyisoprene chain containing 10 isoprene units (Fig 1). CoQ10 is predominant in humans and birds, whereas CoQ9 is predominant in rats and mice (Battino *et al.*, 1990; Dallner and Sindelar, 2000; Albano *et al.*, 2002).

In humans, oral supplementation with CoQ10 has been shown to improve the health status in several diseases including cardiovascular diseases (Langsjoen and Langsjoen, 1999; Belardinelli *et al.*, 2006) and pediatric cardiomyopathy Parkinson's and Huntington's disease (Kiebertz, 1999; Beal, 2002; Shults *et al.*, 2002; Shults, 2003). In addition, the amount of CoQ10 decreases rapidly after the age of 40 years (Kalén *et al.*, 1989). Therefore, CoQ10 is used as a dietary supplement to combat aging worldwide, and it has become increasingly popular. On the other hand, in chickens, only a few studies have been performed in broiler chickens. For

example, Geng and colleagues demonstrated that dietary CoQ10 supplementation reduced broiler chickens' susceptibility to ascites, which might be the result of improving hepatic mitochondrial function, some respiratory chain-related enzymes activities and mitochondrial antioxidative capability (Geng *et al.*, 2004; Geng and Guo, 2005). However, the effects of dietary CoQ10 on laying hens have not yet been examined.

In mammals, the absorption of CoQ10 follows the same process as that of fat-soluble nutrients in the gastrointestinal tract (Bhagavan and Chopra, 2006). Following absorption, CoQ10 is taken up rapidly by the liver where CoQ10 is repackaged into very low density lipoprotein (VLDL) particles and released into circulation (Bhagavan and Chopra, 2006). Kaikkonen *et al.* (1997) reported that dietary CoQ10 elevates Q10 in VLDL plus low density lipoprotein fraction by 160%. All this evidence suggests that dietary CoQ10 increases plasma VLDL CoQ10 concentration. In laying hens, VLDL particles are synthesized in the liver and released into circulation (Schneider *et al.*, 1990) as well as in mammals. However, this VLDL particle, which has been termed yolk targeted VLDL (VLDLy), contains large amounts of apoVLDL II. Since apoVLDL II is an inhibitor of lipoprotein lipase, most of the VLDLy particles escape lipolysis (Schneider *et al.*, 1990; MacLachlan *et al.*, 1996), thus ensuring the efficient delivery of lipids, protein, fat-soluble vitamins, and other nutrients to the growing

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oocytes. In fact, several fat-soluble nutrients are transferred from the feed to the egg yolk (Chamrusspollert and Sell, 1999; Lewis *et al.*, 2000; Grobas *et al.*, 2002; Mattila *et al.*, 2004; Leeson and Caston, 2004; Olson *et al.*, 2008; Saitoh *et al.*, 2001). These findings raise the hypothesis that dietary CoQ10 can transfer to the egg yolk of laying hens.

In mammals, the cholesterol-lowering effect of dietary CoQ has been reported. For example, dietary CoQ10 decreases plasma total cholesterol concentration in diabetic rats (Modi *et al.*, 2006) and patients with recent myocardial infarction (Singh *et al.*, 2003). Krishnaiah and Ramasarma (1970) reported that CoQ9 suppresses hepatic cholesterogenesis and decrease serum cholesterol concentration in rats. In laying hens, egg cholesterol is first biosynthesized in the liver (Andrews *et al.*, 1968) and secreted into the blood in the form of VLDLy (Burley *et al.*, 1984; George *et al.*, 1987; Elkin, 2006). VLDLy is then transported to the ovary (Nimpf and Shneider, 1991; Elkin, 2006). However, the effect of dietary CoQ10 on the content of cholesterol in the egg yolk have not been examined.

In the present study, an experiment was designed to determine the effects of dietary CoQ10 on the contents of CoQ10 and cholesterol in the egg yolk.

Materials and Methods

Experimental Design

Twenty 30 weeks-old Boris Brown hens were housed in wired laying cage (one bird per cage) and fed on the commercial feed (Nippon Formula Feed Mfg. Co., Ltd., Kanagawa, Japan) for 1 week before the experiment. The birds were assigned based on egg production rate and egg weight to 2 groups (10 birds in each group). Yeast-derived CoQ10 (Kaneka Co., Osaka, Japan), which is permitted as a feed stuff in Japan, was used in this study. In this experiment, pair-feeding was employed to keep the food and CoQ10 intakes the same. The birds were pair fed at 100 g/day of the commercial feed containing either 0 (as a control) or 0.8% of CoQ10 during the experimental period. Eggs were collected every day at 12 AM and weighed. Egg yolk weight and the contents of CoQ10 and cholesterol in the egg yolk were measured at 7, 14, 21 and 28 days after the experiment was introduced. At the end of the experiment, hens were sacrificed by decapitation and their blood was collected. Plasma was separated

immediately by centrifugation at $3,000\times g$ for 10 min at 4°C , frozen by liquid nitrogen and stored at -80°C for CoQ10 analysis. The liver was excised, weighed, frozen immediately by liquid nitrogen, and stored at -80°C for CoQ10 analysis. All experimental procedures followed the guidelines for the care and use of experimental animals at the Rokkodai Campus of Kobe University in Japan.

Preparation of VLDLy

Selective precipitation of the apolipoprotein B-containing lipoprotein VLDL from human serum with heparin and Ca^{2+} has been reported (Cham, 1976). VLDLy particle, which is smaller than VLDL particle, contains apolipoprotein B (Walzem *et al.*, 1999). In the preliminary experiment, we confirmed by electrophoresis that the isolated VLDLy by the method of Cham (1976) contains apoVLDL-II, the VLDLy specific apolipoprotein, as mentioned in the introduction (data not shown). Thus, in the present study, VLDLy was isolated from the frozen plasma by the method of Cham (1976).

CoQ10 Analysis

CoQ10 concentrations in the liver, VLDLy and the egg yolk were determined by the method of Menke *et al.* (2000) as follows: CoQ10 was extracted from the liver, VLDLy and the egg yolk by methanol-hexane (2: 3, vol/vol), reduced by iron chloride and analyzed using HPLC.

Measurement of Cholesterol Contents

Cholesterol was extracted from the egg yolk by chloroform-methanol (1: 2, vol/vol) and determined enzymatically using a commercial kit (Cholesterol E-test Wako, Wako Pure Chemical Industries, Ltd., Osaka, Japan).

Statistical Analysis

All data were analyzed by Student's *t*-test using the commercial package StatView version 5 (SAS Institute, Cary, North Carolina, USA, 1998)

Results

Dietary CoQ10 did not affect egg production rate and feed efficiency (Table 1). Egg production rate was calculated as $100 \times \text{number of eggs laid} / (\text{number of hens} \times \text{days})$. Feed efficiency was calculated as egg weight (g)/feed intake (g). Dietary CoQ10 did not affect the weights of egg and egg yolk (Table 1). CoQ10 content in the egg yolk was significantly increased already at day 7 (from

Fig. 1. Structure of coenzyme Q10.

Table 1. Effect of dietary coenzyme Q10 on egg production rate, feed efficiency, egg weight and egg yolk weight in laying hens

	0%	0.8%
Egg production rate (%) ^a	87.14 $\pm$ 0.87	82.86 $\pm$ 2.08
Feed efficiency ^b	0.57 $\pm$ 0.02	0.56 $\pm$ 0.02
Egg weight (g)	65.09 $\pm$ 1.30	66.35 $\pm$ 1.41
Egg yolk weight (g)	15.10 $\pm$ 0.38	15.35 $\pm$ 0.35

^a: $100 \times \text{number of eggs laid} / (\text{number of hens} \times \text{days})$

^b: egg weight (g)/feed intake (g)

$4.35 \pm 0.44 \mu\text{g/g}$ egg yolk to $11.61 \pm 1.92 \mu\text{g/g}$ egg yolk, $P < 0.01$), and an additional increase was observed during the next 7 days (to $22.46 \pm 2.00 \mu\text{g/g}$ egg yolk, $P < 0.01$), and this value was substantially maintained throughout the remainder of the experiment (Fig. 2). Hepatic CoQ10 content (Fig. 3) and VLDLy CoQ10 concentration (Fig. 4) of 0.8% group were increased significantly ($P < 0.01$) compared with those of the 0% group. These values were more than 5-fold higher than those of the 0% group. Dietary CoQ10 decreased cholesterol concentration in the egg yolk significantly ($P < 0.05$) at 21st and 28th day of the experiment ($89.66 \pm 2.6\%$ and $92.95 \pm 3.2\%$, respectively; Fig. 5). There is no significant difference in the egg production rate between groups (0% group, $82.9 \pm 1.2\%$; 0.8% group, $80.2 \pm 1.0\%$, respectively) during day 21 to day 28. All feed was eaten in both groups throughout the experimental period.

Fig. 2. Effect of dietary coenzyme Q10 on coenzyme Q10 content in the egg yolk. Data represent the mean $\pm$ SEM of 10 hens per group. **, significant with respect to day 0 ($P < 0.01$).

Fig. 3. Effect of dietary coenzyme Q10 on hepatic coenzyme Q10 content. Data represent the mean $\pm$ SEM of 10 hens per group. **, significant with respect to 0% group ($P < 0.01$).

Discussion

CoQ10 is a ubiquinone present in both animal and plant cellular membranes. Two major functions are attributed to this compound, namely as an electron carrier in the mitochondrial respiratory chain and as a lipid-soluble antioxidant (Kaikkonen *et al.*, 1997; Bhagavan and Chopra, 2006). In addition, other important functions of CoQ10 such as cell signaling and gene expression have been recognized (Ernster and Dallner, 1995; Crane, 2001; Groneberg *et al.*, 2005). Since the amount of CoQ10 decreases rapidly after the age of 40 years in humans (Kalén *et al.*, 1989), CoQ10 is used as a dietary supplement to combat aging worldwide. CoQ10 is ubiquitously found in the diet with daily intakes ranging from 3 to 5 mg/day (Weber *et al.*, 1997). In the present study, dietary CoQ10 significantly increased CoQ10 content (approximately 0.35 mg CoQ10/egg), suggesting that CoQ10-enriched eggs produced in this study could be a good source of CoQ10.

In laying hens, fat-soluble nutrients such as fatty acid (Chamrusspollert and Sell, 1999; Lewis *et al.*, 2000), fat soluble vitamins (Grobas *et al.*, 2002; Mattila *et al.*, 2004), carotenoids (Leeson and Caston, 2004; Olson *et al.*, 2008) and isoflavone (Saitoh *et al.*, 2001) are transferred from the feed to the egg yolk. In the present study, dietary CoQ10 significantly increased hepatic CoQ10 content, plasma VLDLy CoQ10 concentration, and CoQ10 content in the egg yolk. In mammals, there is evidence that dietary CoQ10 is repackaged into VLDL particles and released into circulation (Bhagavan and Chopra, 2006). In laying hens, most of the VLDLy particles which is assembled in the liver is transported into the egg yolk (Nimpf and Shneider, 1991). These findings and our results suggest that, in CoQ10-fed laying hens, the increase of hepatic CoQ10 elevates the plasma VLDLy CoQ10 concentration, which in turn results in a high CoQ10

Fig. 4. Effect of dietary coenzyme Q10 on plasma very low density lipoprotein y coenzyme Q10 concentration. Data represent the mean $\pm$ SEM of 10 hens per group. **, significant with respect to 0% group ($P < 0.01$).

Fig. 5. Effect of dietary coenzyme Q10 on cholesterol content in the egg yolk. Data represent the mean $\pm$ SEM of 10 hens per group. *, significant with respect to 0% group ($P < 0.05$).

content in the egg yolk.

From a nutritional standpoint, eggs are an excellent source of high-quality protein, mono- and polyunsaturated fatty acids, several minerals, and numerous fat- and water-soluble vitamins (Cook and Briggs, 1977). However, it is recommended that people should limit the consumption of eggs because of their high cholesterol (208 mg/egg) content (Kritchevsky and Kritchevsky, 2000). Therefore, research efforts directed toward reducing egg cholesterol content have centered on alteration of the laying hens' diet with various pharmacological agents and natural products. For example, atorvastatin significantly decreases the content of cholesterol in the egg yolk (Elkins *et al.*, 2003). However, in this study, significant amounts of atorvastatin accumulated in the egg yolk, suggesting that this pharmacological agent can not be a candidate for a feed additive. On the other hand, dietary natural products have resulted in only slight reductions in avian egg cholesterol content (Elkin, 2006). In addition, the effects of several natural products on the egg cholesterol content are conflicting (Hargis *et al.*, 1991; Oh *et al.*, 1991; Scheideler and Froning, 1996). In the present study, we found that natural product CoQ10 significantly decreases the content of cholesterol in the egg yolk of laying hens (90–93% of control, Fig 5). In addition, evidence from human clinical trials indicates that the upper level for supplements for CoQ10 is 1,200 mg/day in humans (Hathcock and Shao, 2006), suggesting that CoQ10-enriched egg, which contains approximately 0.35 mg CoQ10/egg, has no adverse effects on humans. These findings suggest that CoQ10 is a promising candidate for a feed stuff to decrease the content of cholesterol in the egg yolk.

In laying hens, cholesterol is primarily biosynthesized in the liver and incorporated into VLDL_y, which is secreted into the bloodstream and subsequently taken up by growing oocytes (Elkin, 2006). There is evidence that 3-

hydroxy- 3 -methylglutaryl coenzyme A reductase (HMGR: a rate-limiting enzyme of cholesterol synthetic pathway) inhibitors are effective egg cholesterol-lowering agents via lowered VLDL cholesterol concentration when orally administered to laying hens (Elkin and Rogler, 1990; Elkin *et al.*, 1999, 2003). In rats, there is evidence that dietary CoQ9 suppresses HMGR activity (Omkumar *et al.*, 1992) and hepatic cholesterogenesis (Krishnaiah and Ramasarma, 1970). It is therefore possible that dietary CoQ10 decreases cholesterol content in the egg yolk by the suppression of hepatic cholesterogenesis. Further study is needed to clarify this point.

In this study, we investigated the effects of dietary CoQ10 on the egg production and the egg qualities of laying hens. The egg production rate, feed efficiency, egg weight, and egg yolk weight were not influenced by CoQ10 supplementation. Dietary CoQ10 significantly increased the content of CoQ10 and significantly decreased the content of cholesterol in the egg yolk. In addition, dietary CoQ10 significantly increased hepatic CoQ10 content and plasma VLDL_y CoQ10 concentration. These findings clearly demonstrate that natural antioxidant CoQ10 can be transferred into the egg yolk from the feed.

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