INTRODUCTION

Palm kernel cake (PKC), also known as palm kernel meal, is obtained after extraction of oil, a by-product from the oil palm industry which is readily available in tropical countries. It contains 15-17% of protein and a high level of fibre. Onwudike (1986a) reported that PKC contained 11.1 MJ metabolisable energy (ME) kg⁻¹, whereas Yeong (1985) reported PKC contained 6.2 MJ ME kg⁻¹ only. Therefore, it is commonly used as animal feed, especially for ruminants. Many attempts have been made to study the effects of different inclusion levels of PKC on performance characteristics of pigs (Rhule, 1996) and poultry (Onwudike, 1986a,b and 1998; Osei and Amo, 1987). Nwokolo et al. (1976) suggested PKC is a good source of protein for poultry. Amino acid availability of PKC ranged from 63.3% for glycine to 93.2% for arginine with an average of 84.5%. Gohl (1981) suggested that the dietary of PKC inclusion levels should be limited to 200 g/kg to support high growth performance of broilers. Onwudike (1986a,b) showed that up to 34% and 40% of PKC could be included in the diets for starter pullets and layers without any deleterious effect on their performance. However, when the proportion of PKC increased beyond 40%, there was a significant drop in egg production, egg weight, feed intake and feed conversion efficiency.

Very low density lipoprotein (VLDL) is a spherical particle which consisting of amphipatic PL and cholesterol in the boundary layer of the VLDL, whereas in an inner core composed of nonpolar TG and CE (Gurr and Harwood, 1991). VLDL forms one of the classes of plasma lipoprotein and is responsible for the majority of the transport of TG from the liver to extrahepatic tissues, principally adipose tissue, cardiac muscle, lung and lactating mammary gland (Fielding and Fielding, 1991).

Numerous studies on experimental animals indicated that the addition of different types of fibre might have different effects on lipid metabolism (Van Berge-Henegouven et al., 1979; Albrink et al., 1979). It has been shown that feeding rats with a diet containing 10% wheat bran for 28 days led to a significant decrease in the concentration of plasma lipids (Patrick et al., 1989). Chen and Anderson (1979) reported that feeding a high fibre wheat in the diet of rats decreased accumulation of triacylglycerol (TG) and cholesterol in the liver. Very little research has been conducted studying the effects of different levels of PKC on fat deposition and plasma lipids. Since PKC contains high level of fibre, it is believed that the fibre may affect lipid metabolism of animals. The objective of the present study was to study the effects of different levels of PKC on growth performance and blood lipids in rats.

MATERIALS AND METHODS

Animals

The protocol of this experimental was approved by the Research Committee of the University Putra Malaysia, Malaysia. A total of sixty four 8 weeks old Sprague-Dawley,
male rats with an average initial body weight of 244.00±7.60 were used in the experiment. The rats were housed individually in standard cages and maintained at a constant environmental temperature (24-26°C) and relative humidity (60-64%). Water and food were supplied ad libitum. Feed intake was measured daily. The rats were randomly assigned to four treatments. Each treatment groups consisted of sixteen rats. They were fed on 0%, 15%, 20% and 25% PKC (table 1). All the rats were acclimatised to the respective diets for a week before the experiment started. The experiment was carried out for four weeks. At the end of the experiment, the animals were fasted for 12 h before they were euthanized under anesthesia with diethyl ether. Blood was collected by cardiac puncture into tubes containing EDTA as anticoagulant. Epididymal adipose tissue was collected and weighed.

**Analytical methods**

Plasma was isolated by centrifugation and plasma very low density lipoprotein (VLDL) was separated using fast protein liquid chromatography (Äkta-FPLC, Amersham Pharmacia Biotech). Two ml plasma samples were filtered through a 0.22 µm membrane filter and injected into a packed Superose 6 prep grade column (XK 16/70, Amersham Pharmacia Biotech) pre-equilibrated with 0.15 M NaCl, 10 mM Na2HPO4, 5 mM Na2EDTA, 0.02% NaCl, pH 7.4 buffer. Buffer was filtered with 0.22 µm membrane filter and degassed before used. The FPLC system was operated at 4°C. The plasma sample was eluted at 0.4 ml/min. and lipoprotein was detected spectrophotometrically at 280 nm. Every 2 ml eluent was collected for each fraction (Tan et al., 2000).

Plasma and VLDL protein (Sigma-Aldrich, US., procedure no. P5656), triacylglycerol (TG) and total cholesterol (Randox, UK), phospholipid (PL) and free cholesterol (FC) (Wako, Japan) were determined using the appropriate kits and following the manufacturers’ instructions. Cholesteryl ester (CE) was calculated by

| Ingredients                              | 0%    | 15%   | 20%   | 25%   |
|------------------------------------------|-------|-------|-------|-------|
| Broken rice                              | 20.00 | 15.50 | 21.38 | 23.48 |
| Corn                                     | 30.88 | 20.88 | 10.20 | 3.00  |
| Palm kernel cake¹                        | 0.00  | 15.00 | 20.00 | 25.00 |
| Soybean meal (46% CP)                    | 22.00 | 22.00 | 22.00 | 22.00 |
| Dicalcium phosphate                      | 1.40  | 1.40  | 1.40  | 1.40  |
| Salt                                     | 0.70  | 0.70  | 0.70  | 0.70  |
| Limestone                                | 0.60  | 0.60  | 0.60  | 0.60  |
| DL-methionine                            | 0.50  | 0.50  | 0.50  | 0.50  |
| L-lysine                                 | 0.50  | 0.50  | 0.50  | 0.50  |
| Vitamin premix²                          | 2.11  | 2.11  | 2.11  | 2.11  |
| Palm oil                                 | 1.60  | 1.60  | 1.60  | 1.60  |
| Fish meal                                | 8.00  | 7.50  | 7.20  | 7.20  |
| Mould guard³                             | 0.01  | 0.01  | 0.01  | 0.01  |

| Compositions of fatty acids (%)          |       |       |       |       |
|------------------------------------------|-------|-------|-------|-------|
| 12:0                                     | 3.20  | 3.30  | 3.15  | 3.30  |
| 14:0                                     | 2.00  | 2.10  | 2.20  | 2.10  |
| 16:0                                     | 22.50 | 23.00 | 24.70 | 27.00 |
| 18:0                                     | 5.40  | 5.60  | 5.70  | 5.60  |
| 18:1                                     | 36.00 | 37.00 | 38.80 | 40.00 |
| 18:2n-6                                  | 22.60 | 21.40 | 19.65 | 16.40 |
| 18:3n-3                                  | 6.80  | 5.60  | 4.80  | 3.10  |

| Calculated analyses                      |       |       |       |       |
|------------------------------------------|-------|-------|-------|-------|
| Crude protein                            | 22.72 | 23.39 | 23.50 | 23.79 |
| Crude fibre                              | 3.13  | 5.33  | 5.93  | 6.60  |
| ME (MJ/kg)                               | 15    | 15    | 15    | 15    |

¹Analysis of palm kernel cake: Dry matter, 89%; Crude protein, 13.5%; Ether extract, 2%; Ash, 4% and Crude fiber, 15% and ME, 2,150 kcal/kg (all analysed in the Animal Nutrition Laboratory, University Putra Malaysia).

²The vitamin premix provides the following amounts per kilogram of diet: Vitamin A, 5,200 IU; Cholecalciferol, 1,000 IU; Vitamin E, 10 IU; Vitamin K, 1.3 mg; Riboflavin, 8.0 mg; Niacin, 25 mg; D-calcium pantothenic acid, 10 mg; Choline chloride, 210 mg and Vitamin B12, 0.01 mg.

³Mould guard consists of organic acid to prevent the growth of fungus in the diet.

Table 1. Compositions of experimental diets
deduction of FC from total cholesterol. An appropriate standard curve was prepared for each assay. All determinations of plasma and VLDL samples were performed in triplicate. VLDL lipid concentration ratios were calculated to estimate the size of VLDL particles (Fungwe et al., 1992).

**Statistical analysis**

Data are expressed as mean±SEM. One-way analysis of variance (ANOVA) was used to assess differences in growth performance, plasma and VLDL composition among different diet groups. Differences between mean values were evaluated by the Student-Newman-Keuls Multiple Comparisons Test and were considered significant at p<0.05 (Minitab, 1995).

**RESULTS AND DISCUSSION**

Data showed that feeding rats a diet supplemented with 15%, 20% and 25% PKC compared to control (0% PKC) had no significant effect on growth rate, daily feed intake and fat weight. The results show that feeding of PKC up to 25% would not have any adverse effect on the growth performance and their fat deposition. Osei and Amo (1987) reported that feeding different levels of PKC in isonitrogenous diets reduced feed conversion efficiency but found no significant differences in body weights of broilers.

No differences (p>0.05) were observed in the concentration of PL in the plasma of rats on any levels of the PKC groups. Plasma PL concentration was unaffected (p<0.05) by dietary PKC. Plasma TG, PL, total cholesterol and FC concentrations in rats fed the various levels of PKC are shown in figure 1. Plasma protein level was moderately (p<0.05) elevated at dietary concentration of 20% PKC compared to control diet (data not shown in the figure 1, plasma protein concentrations were 110.64, 105.67, 133.94 and 123.03 respectively for 0%, 15%, 20% and 25% PKC dietary). Plasma TG concentrations decreased (p<0.05) with increase in dietary PKC. Large decrease (p<0.05) in plasma total cholesterol concentration was also observed with higher concentrations of PKC in the diet (20 and 25% of PKC). The plasma TG and total cholesterol concentrations were lower in those rats feeding with PKC. This could be attributed to the high content of fibre in the PKC. This suggests that fibre of PKC has effect of reducing cholesterol concentration of rats. The observation was in agreement with numerous feeding studies in different species using fibre-rich wheat, where low concentrations of TG and cholesterol were obtained (Van Berge-Henegouwen et al., 1979; Albrink et al., 1979; Gariot et al., 1986; Patrick et al., 1989).

Table 3 summarises the effects of dietary PKC on the concentrations of the lipid components of plasma VLDL. Plasma concentrations of VLDL-TG was elevated (p<0.05) over the control group whatever the level of the PKC. Concentration of VLDL-PL was decreased (p<0.05) after feeding PKC. In contrast, VLDL-protein, total cholesterol, FC and CE were unaffected (p>0.05) by dietary PKC. The ratios of the surface lipids (PL+FC) and core lipids (TG+CE) of the plasma VLDL were significantly different (p<0.05) in the various dietary groups of rats. In the present study, the rats fed with PKC (either 15, 20 or 25%) had greater concentrations of VLDL-TG. This probably caused by an increasing secretion of TG from liver into blood circulation or reducing uptake of TG from the circulation into extrahepatic tissues (Loh, 1997). According to Anderson and Tietyen-Clark (1986), insoluble fibre sources may affect fat metabolism. PL is one of the components in the outer layer of VLDL, which is also a substrate of lipoprotein lipase but is hydrolysed at a slower rate (Cryer, 1985). The lower PL concentration were observed in rats treated with PKC compared to control rats, probably indicating that hydrolysis of PL occurred at a greater rate in these PKC treated rats. The increased hydrolysis rate of PL may be attributed to the high fibre content of PKC. Each of the VLDL particle consists of one molecule of apo-B, which is the largest of the apolipoprotein and is the major protein constituent of the lipoprotein (Davis, 1991). Therefore, concentration of protein in VLDL could be used to indicate the number of VLDL particles in the medium. The results indicate the number of VLDL particles were not significantly different between groups of rats. This also implies that PKC has no effect on the VLDL production (quantity basis). In this study, the concentrations of TG and CE have been employed to indicate the volume of the VLDL, whereas both PL and FC were used to reflect the
The surface area of the VLDL (Wright et al., 1995). The calculation of concentration ratios of the surface lipids (PL+FC) and core lipids (TG+CE) of the VLDL showed that the ratios were significantly different between the control and PKC fed rats. The results further suggest that plasma of PKC fed rats contained more lipid rich VLDL and had different VLDL particle size as compared with the control rats. These results clearly support the previous explanation that PKC has no effect on the VLDL production but it affects on the particle size and the lipid content of VLDL.

In conclusion, inclusion of PKC in the diet of rats up to 25% has no adverse effect on the rats as indicated by their growth performance. However, it may affect the contents of plasma lipids probably due to the fibre content. Generally, the VLDL particles in relation to dietary PKC treatments may be inferred from the results obtained from the VLDL protein and their ratio of surface lipids and core lipids [(PL+FC)/(TG+CE)]. It shows that rats fed with PKC had bigger size of VLDL with low PL content but with similar number of VLDL in the plasma. The observation that reduced plasma TG level was mainly due to a decrease in other lipoproteins (low density lipoprotein and high density lipoprotein) present in the plasma, this probably due to the effect of high fibre content in PKC.

Table 2. Effect of different levels of PKC on growth performance

| Parameters                     | 0          | 15         | 20          | 25          |
|--------------------------------|------------|------------|-------------|-------------|
| Initial body weight (g)        | 246.54±11.54 | 254.30±11.54 | 251.46±10.32 | 233.39±10.32 |
| Final live weight (g)          | 292.99±43.52 | 289.55±43.52 | 280.99±38.92 | 278.08±38.92 |
| Growth rate (g/day)            | 2.21±0.58   | 2.18±0.46   | 2.81±0.65   | 2.13±0.84   |
| Daily feed intake (g/day)      | 7.07±1.12   | 6.39±1.12   | 6.87±1.00   | 8.27±1.00   |
| Epididymal fat weight (g)      | 1.36±0.19   | 1.76±0.19   | 1.56±0.18   | 1.26±0.18   |

Data are presented in the mean values±SEM. Means in same row with different alphabet differ significantly (p<0.05).

Table 3. Effect of different levels of PKC on plasma VLDL lipids

| Parameters                     | 0          | 15         | 20          | 25          |
|--------------------------------|------------|------------|-------------|-------------|
| Protein                        | 4.05±0.36  | 4.67±0.11  | 4.75±0.17   | 4.88±0.10   |
| Triacylglycerol,               | 0.05±0.01   | 0.07±0.01ab | 0.07±0.01ab | 0.08±0.01a  |
| Phospholipid                   | 0.13±0.02a  | 0.07±0.01b  | 0.07±0.01b  | 0.06±0.01b  |
| Total cholesterol              | 0.04±0.01   | 0.04±0.01   | 0.05±0.01   | 0.04±0.04   |
| Free cholesterol               | 0.01±0.002  | 0.02±0.003  | 0.02±0.003  | 0.02±0.002  |
| Cholesteryl ester              | 0.03±0.01   | 0.04±0.01   | 0.04±0.01   | 0.03±0.01   |
| Surface:core ratio             |            |            |             |             |
| PL+FC                          | 1.44±0.27a  | 0.77±0.10b  | 0.76±0.12b  | 0.75±0.10b  |
| TG+CE                          |            |            |             |             |

Data are presented in the mean values (µg/ml of plasma)±SEM. Means in same row with different alphabet differ significantly (p<0.05).

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