

## Blood lipid status of “jawa ekor kurus” sheep supplemented by protected kapok seed oil

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**ABSTRACT:** This investigation was conducted to study the influence of protected kapok seed oil (KSO) supplementation in its combination with rice polishing (RP) on lipid status of “jawa ekor kurus” sheep fed with field grass as basal feed. The amounts of 24 heads of meal “jawa ekor kurus” sheep were used as experimental material. Those divided into 8 treatment groups, consist of 3 heads as replication, respectively. There were two treatment factors, i.e. : KSO supplementation (factor I) and concentrate supplementation (factor II). Factor I consist of 2 levels, i.e. 0% (S0) and 10% (S1), whereas factor II consist of 4 levels, i.e. 0% (K0), 15% (K1), 30% (K2) and 45% (K3) respectively, based on dry matter (DM) consumption. Several variables were measured, namely concentrations of blood triglyceride (TG), low density lipoprotein (LDL) and high density lipoprotein (HDL) cholesterol. The collected data were statistically analyzed by analysis of variance with factorial treatment pattern (2 x 4) in completely randomized design (CRD). Supplementation of RP increase blood total cholesterol ( $P < 0.05$ ), i.e. : 1.35 mM in S0K0 became to 1.93 mM in S0K3 treatment group, whereas its combination with protected KSO did not result in significantly variation of blood plasm cholesterol (i.e. : 1.93 up to 1.99 mM), along with increasing of HDL cholesterol (from 0.98 mM in S1K0 became to 1.21 mM in S1K3 treatment group) and decreasing of LDL cholesterol.( from 0.70 mM in S1K1 up to 0.69 mM in S1K3).

**Key words:** kapok seed oil, protection, rice polishing, triglyceride, cholesterol, low density lipoprotein, high density lipoprotein, sheep

### INTRODUCTION

There were several constraint in increasing of small ruminant productivity in Indonesia, among other, low in demand of those meat. Consumption rate of cattle, poultry, and pig meats in Indonesia, were 56, 23, and 13% respectively, whereas consumption rate of small ruminant meat, was 5% only (Ditjen Bina Produksi Peternakan, 2006). According to Arinto (2006), the consumption rate of small ruminant meat was low, be caused cholesterol phobia issue in Indonesian community.

The effort to solved those, must be conducted, with explanation and an introduction of technology to decrease the cholesterol level in small ruminant meat, so that the acceptability of those will be increased. Polyunsaturated fatty acid (PUFA) source supplementation (in this case, protected linoleic acid), is one of alternative technology to increases those content in animal product.

According to Sardesai (1992), PUFA had a biological roles, among other in controlling of cholesterol status. Raharjo (1995) stated, there was correlation between the increasing of blood cholesterol level and saturated fatty acid consumption, and unsaturated role in decreasing of blood cholesterol level. The PUFA, in this case linoleic acid, was phosphatidylcholine component, which the main phospholipids in HDL. High density lipoprotein (HDL) can carried the cholesterol from periphery tissue as well as another lipoprotein to be oxidized in liver (Bauchart, 1992).

Investigation about the influence of PUFA in controlling of cholesterol status in sheep, can explained those mechanism in consumer of sheep meat which had been increased in its linoleic acid content by supplementation of protected PUFA source. To obtain the usefulness of unsaturated fatty acid (UFA) significantly, the supplementation of adequate protected UFA was required. Protection was required to avoid the UFA from biohydrogenation by rumen microbes (Cook, 1978; Scott and

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Ashes, 1993). Protection also useful to eliminated the negative impact of high level of UFA supplementation, namely decreasing of fiber degradability (Jenkins, 1993; Wang and Song, 2001; Aharoni *et al.*, 2004). Protection was conducted partially, in order to obtained the influence of UFA on rumen metabolism efficiently, which reflected in decreasing of acetic acid level/ propionic acid level ratio.

Kapok seed oil (KSO) is one of the potential UFA source. According to Sarosa (1990), proportion of PUFA in total lipid of KSO, was 71.95%. Amount of 54.29% from those was linoleic acid, whereas another, consist of oleic acid (43.50%) and linolenic acid (2.21%). The most of Indonesian KSO were resulted from north coastal area of Central Java, namely around of Muria Mountain, Pati and Jepara.

## MATERIALS AND METHODS

The major materials used were protected kapok seed oil (KSO) as supplement, fibrous feed in this case field grass (FG) as basal feed, concentrate in this case rice polishing (RP), 24 heads of male “jawa ekor kurus” (JEK) local sheep as experimental units. The PUFA source (KSO) was used with 75% protection level. The experimental sheep age were selected about 6 months based on body weight (about 13 kg) (Sabrani and Levine, 1993). The major equipment, consist of animal balance, feed balance, analytical balance, individual pen and its equipment, venoject tubes and its needle, waterbatch, ultracentrifuge.

Protection of KSO was conducted by saponification using KOH and then was transformed to Ca salt by CaCl<sub>2</sub>. Amount of KOH were used was suitable for protection level, calculated based on saponification number of KSO that determined according to Cabatit method (1979). Certain amount of KSO filled into beaker glass, then to be heated up to 90°C. Amount of KOH suitable with calculation was balanced, dissolved by aquadest then added to heating KSO, while stirred for 10 minutes up to kalium soap suspension was formed. In transformation of kalium soap to Ca salt, amount of CaCl<sub>2</sub> calculated by stoichiometri, to be balanced and dissolved by aquadest. The CaCl<sub>2</sub> solution added to kalium soap suspension, while heated in waterbatch at 90°C and stirred up to Ca salt was formed. After centrifugation at 2500 rpm for 10 minutes, supernatant was removed, the precipitate was added by unprotected KSO portion, ready for used as supplement. Before to be protected, the KSO was heated previously for 4 hours at 200°C to eliminate its anti nutrition substance.

This Research was started by preparation of pen and its equipment, and treatment. Twenty four heads of experimental sheep were divided into 8 groups based on treatment combination. Each group consist of 3 heads as replications. There were 2 treatment factors, namely protected KSO supplementation as factor 1 and feeding concentrate (RP) as factor 2. Treatment factor 1 consist of 2 levels, namely without supplementation (S0) and with supplementation (S1). Treatment factor 2 consist of 4 levels, namely : 0% (K0); 15% (K1); 30% (K2) and 45% (K3).

The experiment proceeded for 3 months, including 10 days adaptation period, 10 days introduction period and 70 days observation period, while experiment, forage was fed *ad libitum*. Concentrate were fed every morning, whereas water supplied continously. Mineral mixture consist of limestone, bone meal and salt (1 : 1 : 1) to be fed free choice (Tillman, 1978).

Feed consumption and body weight data were collected after introduction period. Balancing of body weight were conducted periodically each week, in the morning before feeding, as the base of supplementation. Blood sampling was done for determination of plasm TG and cholesterol concentrations according to Liebermann-Bauchart method (Tranggono *et al.*, 1989).

**Table 1.** Nutrient composition of experimental feed (dry matter basis)

Feed	CP, %	CF, %	Lipid, %	Ash, %	NFE, %
Field grass	10.16	32.66	1.37	16.58	39.23
Rice polishing	14.04	15.81	1.08	10.08	42.99

CP : crude protein; CF : crude fiber, NFE : nitrogen free extract

The collected data were analyzed statistically by analysis of variance in completely randomized design. Difference of means between treatment groups were analyzed by Duncan method (Astuti, 1980; Sugandi and Sugiarto, 1993).

## RESULTS AND DISCUSSION

### *Triglyceride*

Blood plasm TG concentration of sheep without KSO supplementation fed RP at 0; 15; 30 and 45% level (S0K0, S0K1, S0K2 and S0K3, were : 0.11; 0.15; 0.27 and 0.30 mM, respectively)(Table 3). Feeding of RP (up to 45% of ration DM) increased blood plasm TG concentration ( $P<0.05$ ). Christie (1979<sub>a</sub>) suggested that blood plasm TG concentration was influenced by lipid consumption. Lipid consumption as long chain fatty acid source and concentrate as source of alfa gliserol phosphat, were very important for the blood plasm TG concentration.

**Table 2.** Average of dry matter (DM), organic matter (OM), crude protein (CP) and lipid consumption per head per day

Treatment	DM, g	OM, g	CP, g	Lipid, g
S0K0	391	336	47	6,04
S0K1	437	379	53	16,66
S0K2	539	468	68	31,76
S0K3	548	481	71	45,21
S1K0	455	396	50	48,54
S1K1	599	519	64	73,71
S1K2	666	577	74	87,95
S1K3	612	535	71	98,87

Protected KSO supplementation to sheep without feeding of RP (S1K0) resulted in lipid consumption was 48.54 g/day (Table 2), those were equivalent to lipid consumption by sheep in S0K3 group. Blood plasm TG concentration of sheep in S1K0 treatment group were not significantly different from blood plasm TG concentration of sheep in S0K1 group, even if its lipid consumption were much higher. Those were understood, because most of fatty acids consumed by sheep in protected S1K0 group, so that most of that were absorbed as PUFA. The absorbed unsaturated fatty acids were esterified in intestinal mucosal cell was not as TG, but as phospholipids and cholesterylester (Christie, 1979; Bauchart, 1992; Ashes *et al.*, 1995). The fact showed that blood plasm TG concentration of sheep in S1K3 group (0.31%), were not significantly different from S1K2 (0.33%) and even tended to decrease, and not significantly different from blood plasm TG concentration of sheep in S0K3 and S0K2 groups. Those phenomenon supposed to be occurred because increasing of unprotected PUFA were bypassed from ruminal biohydrogenation, so that amount of esterified fatty acids became to TG were decreased. Gerson *et al.* (1985) and Pantoja *et al.* (1996) stated that decreasing of rumen fluid pH inhibited ruminal glyceride lipolysis.

### *Cholesterol*

Blood plasm cholesterol concentration in sheep without KSO supplementation which received RP. Were higher ( $P<0.05$ ) than those without KSO supplementation and without RP (1.54; 1.75; 1.93 mM, in S0K1, S0K2 and S0K3, respectively vs 1.35 mM in S0K0). Lipid consumption increased along with increasing of RP feeding levels, from 6.04 g in S0K0 to 16.06 ; 31.76 and 45.21 g in S0K0, S0K2 and S0K3, respectively. Those increasing of consumption were followed by the improving of its absorption, as reflected in blood plasm TG concentration. The improving of lipid absorption would be followed by rising of lipoprotein synthesis to lipid transported in blood, as

reflected by the rising of blood plasma cholesterol concentration. Those could occur because increasing of intestinal cholesterologenesis to facilitate the absorbed lipid transport.

**Table 3.** Blood plasma triglyceride (TG), and cholesterol levels of experimental Sheep

Supplementation	Concentrate	TG, mM	Total chol, mM	LDL chol, mM	HDL chol, mM
S0	K0	0.11 <sup>d</sup>	1.35 <sup>d</sup>	0.57 <sup>e</sup>	0.67 <sup>f</sup>
	K1	0.15 <sup>c</sup>	1.54 <sup>c</sup>	0.66 <sup>d</sup>	0.75 <sup>e</sup>
	K2	0.27 <sup>b</sup>	1.75 <sup>b</sup>	0.75 <sup>bc</sup>	0.81 <sup>d</sup>
	K3	0.30 <sup>ab</sup>	1.93 <sup>a</sup>	0.85 <sup>a</sup>	0.92 <sup>c</sup>
S1	K0	0.17 <sup>c</sup>	1.98 <sup>a</sup>	0.70 <sup>cd</sup>	0.98 <sup>c</sup>
	K1	0.28 <sup>b</sup>	1.93 <sup>a</sup>	0.79 <sup>ab</sup>	0.97 <sup>c</sup>
	K2	0.33 <sup>a</sup>	1.96 <sup>a</sup>	0.73 <sup>bcd</sup>	1.14 <sup>b</sup>
	K3	0.31 <sup>ab</sup>	1.99 <sup>a</sup>	0.69 <sup>cd</sup>	1.21 <sup>a</sup>
Combination average					
	S0	0.21 <sup>b</sup>	1.64 <sup>b</sup>	0.70	0.79 <sup>b</sup>
	S1	0.27 <sup>a</sup>	1.96 <sup>a</sup>	0.73	1.07 <sup>a</sup>
Combination average					
	K0	0.14 <sup>c</sup>	1.66 <sup>d</sup>	0.63 <sup>b</sup>	0.82 <sup>c</sup>
	K1	0.22 <sup>b</sup>	1.74 <sup>c</sup>	0.72 <sup>a</sup>	0.86 <sup>c</sup>
	K2	0.30 <sup>a</sup>	1.85 <sup>b</sup>	0.74 <sup>a</sup>	0.98 <sup>b</sup>
	K3	0.31 <sup>a</sup>	1.96 <sup>a</sup>	0.77 <sup>a</sup>	1.06 <sup>a</sup>

<sup>a,b,c,d,e,f</sup> Different superscripts in the same column-row, showed the significantly difference (P < 0.05).

Blood plasma LDL cholesterol of sheep without KSO supplementation to total blood plasma cholesterol concentration (0.57; 0.66; 0.75 and 0.85 mM, in S0K0, S0K1, S0K2 and S0K3, respectively). Most of LDL cholesterol in ruminant reflected absorbed lipid from small intestine. The HDL cholesterol in sheep without KSO supplementation which received RP were also higher than those without KSO supplementation as well as RP (0.75; 0.81; 0.92 mM in S0K1, S0K2, S0K3 vs mM in S0K0). The increasing of RP level, enhanced the HDL cholesterol concentration. The increasing of HDL cholesterol concentration was response to the increasing of total blood plasma cholesterol, especially LDL cholesterol portion. Bauchart (1992) described that HDL facilitated the taking and/or transporting of cholesterol from extrahepatic tissues to liver.

The protected KSO supplementation to sheep without feeding RP (S1K0) resulted in non significantly different blood plasma TG concentration from sheep without KSO supplementation received 15% RP (S0K1), but its blood plasma cholesterol concentration were higher (P < 0.05) than sheep in S0K1 group (1.98 vs 1.54 mM). Those could occur because protected PUFA portion will be absorbed and stimulated the cholesterologenesis in small intestine mucosal cells. Cholesterol was synthesized, particularly in intestine further more esterified preferentially to linoleic acid of lecithin, formed cholesterylester. Esterification of cholesterol to linoleic acid in HDL in this experiment was indicated in the higher of HDL cholesterol in S1K0 group sheep than S0K1 (0.98 mM vs 0.75 mM) whereas the blood plasma cholesterol was not significantly different, namely 0.70 and 0.66 mM in S0K0 and S0K1, respectively. The protected KSO supplementation to sheep fed 15% RP (S1K1) increased lipid consumption, but its PUFA proportion were lower than those in S2K0 group, so that its LDL cholesterol was higher than S1K0 (0.79 vs 0.70 mM). The increasing of PUFA absorption in S1K2 and S1K3 caused the high of blood plasma HDL cholesterol in sheep of those treatment group (1.14 and 1.23 mM) compared to the other treatment group. The high of blood plasma HDL cholesterol concentration in S1K2 and S1K3 treatment group, were supposed because the requirement of bile acid production was increased. The uptake of LDL cholesterol by HDL caused the low of blood plasma LDL cholesterol concentration in S1K2 and S1K3 treatment group (0.73 and 0.69 mM). The increasing of the use of bile acid synthesis, caused the non significantly difference of blood plasma total cholesterol between S1K2 as well as S1K3 and S1K1 as well as S1K0 (1.96 and 1.99 mM vs 1.93 and 1.98 mM).

## CONCLUSIONS

There was not variation in blood total cholesterol concentration in related to increasing of concentrate level, in KSO supplemented sheep. Supplementation of protected KSO as unsaturated fatty acid source, controlled the blood cholesterol concentration, by increasing of HDL cholesterol concentration and decreasing of LDL cholesterol concentration.

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