The Effect of Polysaccharide Peptide (PsP) on the Improvement of Low-Density Lipoprotein (LDL) Profile Levels in Active Smokers with Dyslipidemia

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Abstract. Indonesia continues to have a substantial smoking burden, and tobacco exposure can worsen dyslipidemia through oxidative stress and inflammation. Polysaccharide peptide (PsP) from Ganoderma lucidum contains β-D-glucan and other bioactive constituents that may support lipid control. This study assessed the LDL-C response to PsP in active smokers with dyslipidemia. Twenty-eight male students aged 18-21 years participated in a pretest-posttest controlled experiment. The intervention group (n=14) received 500 mg PsP orally each day for 30 days; the control group (n=14) received no PsP. LDL-C was determined before and after the study period. Paired t-tests assessed change within groups, and independent t-tests compared the magnitude of change. LDL-C fell significantly after PsP administration ($p<0.001$), but remained unchanged in controls ($p=0.875$). The difference in change between groups was significant ($p<0.001$). PsP may act through β-D-glucan-related antioxidant, anti-inflammatory, and lipid-regulating pathways. Thus, 30-day supplementation with 500 mg/day PsP was associated with lower LDL-C in young active smokers with dyslipidemia. Confirmation in larger, longer, and placebo-controlled studies is needed.

Keywords: Active Smokers, Dyslipidemia, Ganoderma lucidum, Low-density lipoprotein (LDL), Polysaccharide peptides.

1 Introduction

Dyslipidemia comprises one or more disturbances in circulating lipids, including high total cholesterol (TC), triglycerides (TG), or low-density lipoprotein cholesterol (LDL-C), and low high-density lipoprotein cholesterol (HDL-C) [1]. Data from the 2023 Indonesia Health Survey showed high TC in 11.7%, raised TG in 22.8%, low HDL in 87.0%, and raised LDL in 6.5% of the population [2]. Because these abnormalities con-

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tribute to cardiovascular disease, stroke, diabetes, hypertension, and obesity, identifying modifiable exposures is important. Smoking is one such exposure and has been associated with dyslipidemia [3].

The Indonesian health system is challenged by infectious diseases alongside a rising burden of non-communicable disease. Smoking contributes importantly to this transition and was ranked as the country's second leading mortality risk in the Global Burden of Disease 2019 report. Tobacco use is already common among male adolescents, with a reported prevalence of 35.6% [4]. National survey data also indicate that Indonesian active smokers consume approximately 12.11 cigarettes per day [2]. Toxic cigarette constituents can shift the lipid profile toward higher TC, TG, and LDL and lower HDL [5].

Health risks arise from both conventional and electronic cigarettes because their emissions may contain nicotine, carbon monoxide, tar, lead, and additional toxic chemicals [6], [7]. After nicotine exposure, catecholamine secretion increases and stimulates lipolysis. The resulting release of free fatty acids may elevate TG, TC, VLDL, and LDL while decreasing HDL [8]. Adrenaline-mediated changes in adipose metabolism may further contribute to lipid disturbance [6].

Lowering LDL-C is a principal objective of dyslipidemia care. Management combines medication with non-pharmacological measures [9], [10]. Alongside indicated drug therapy, nutritional strategies and natural bioactive products may provide complementary support for long-term risk reduction.

One such non-pharmacological approach is the utilization of natural products, including Ganoderma lucidum. Ganoderma lucidum contains β-D-glucan-rich polysaccharides with reported antioxidant, anti-inflammatory, antitumor, antidiabetic, antimicrobial, antiallergic, immunomodulatory, and neuroprotective activities. Evidence also suggests possible cholesterol-lowering effects and cardiovascular benefit [11]–[14]. These actions are pertinent to smokers, in whom nicotine-driven lipolysis and smoke-related oxidative stress can promote LDL accumulation and vascular dysfunction [8]. Accordingly, this study investigated whether PsP administration could reduce LDL-C in active smokers with dyslipidemia.

2 Methods

2.1 Study Design and Participants

The investigation used a controlled before-and-after experimental design. It was undertaken between September and November 2024 at the Faculty of Pharmacy, Universitas Islam Negeri Maulana Malik Ibrahim Malang, to examine LDL-C changes following PsP supplementation.

Male smokers aged 18-21 years were screened through an online questionnaire. Of those meeting the dyslipidemia criteria, 28 completed the protocol: 14 in the PsP group and 14 in the comparison group.

2.2 Sampling Strategy and Participant Recruitment

Participants were recruited using a non-probability purposive sampling method based on predefined eligibility criteria aligned with the study objectives. The inclusion criteria were male sex, age 18–21 years, moderate-to-high nicotine dependence, and dyslipidemia, defined as LDL-C ≥130 mg/dL, high-density lipoprotein cholesterol (HDL-C) ≤40 mg/dL, and triglycerides ≥150 mg/dL. Participants with chronic conditions, including diabetes mellitus, hypertension, or cardiovascular disease, were excluded.

A total of 32 eligible participants were enrolled and allocated equally to the intervention and control groups. During the study, four participants did not complete the intervention or postintervention assessment. Therefore, 28 participants completed the study and were included in the final analysis, with 14 participants in each group.

Body weight and height were measured at baseline, and body mass index was calculated. Blood samples were collected at baseline and after the 30-day intervention.

2.3 Intervention

Polysaccharide peptide (PsP) was supplied as *Ganoderma lucidum*-derived capsules manufactured by PT Sahabat Lingkungan Hidup, Surabaya, Indonesia. The preparation contains β-D-glucan, a glucose polymer characterized by β-glycosidic linkages.

For 30 consecutive days, each intervention participant took one 500-mg capsule after food. No PsP was provided to controls. Weekly dispensing and a participant-completed daily log were used to support and document adherence.

2.4 Blood Sampling and LDL-C Measurement

Venous blood collection followed the WHO phlebotomy guidance [15]. After an 8-hour fast, 3-5 mL of venous blood was drawn from the antecubital region into a vacutainer. Specimens were kept at 2-8°C and transferred to the Universitas Brawijaya Hospital laboratory, where analysis was completed within 24 hours.

LDL-C testing was conducted at enrollment and on completion of the 30-day period. Interpretation used the following categories: optimal (<100 mg/dL), borderline high (130-159 mg/dL), high (160-189 mg/dL), and very high (≥190 mg/dL [1].

2.5 Statistical Analysis

Analyses were conducted in IBM SPSS Statistics 26.0. Means and standard deviations summarized numerical variables, including age, BMI, smoking duration, and cigarette consumption. Frequencies and percentages described categorical variables such as disease history.

The outcome variable was baseline-to-day-30 change in LDL-C. Distributional assumptions were checked with Shapiro-Wilk testing, and variance equality with

Levene's test. Paired t-tests were applied to pre-post comparisons within each arm; independent t-tests assessed differences between arms. A two-sided $p<0.05$ denoted statistical significance, and effect size was calculated to quantify the intervention effect.

2.6 Ethical Considerations

This study was approved by the Health Research Ethics Committee of the Faculty of Health Sciences, Universitas Brawijaya, under approval number 12757/UN10.F17.10.4/TU/2024. All participants received an explanation of the study procedures and provided written informed consent before participation. Participant confidentiality was maintained throughout the study.

3 Results

3.1 Baseline Characteristics

The analytic sample consisted of 28 men, equally divided between the control and PsP groups. Recorded baseline variables were age, BMI, disease history, years of smoking, and cigarettes consumed per day.

Age, BMI, disease history, and cigarette consumption were not significantly different between groups (all $p>0.05$). Disease history was reported by 3 participants (21.4%) in each group. The only baseline imbalance was smoking duration, which was longer in the PsP group ($p=0.031$) (Table 1).

Table 1. Baseline characteristics

Characteristics	Control group (n=14)	Intervention group (n=14)	p-value
Age, years	19.4 ± 1.56	19.4 ± 1.22	0.893
BMI, kg/m^2	22.5 ± 5.00	24.1 ± 6.77	0.486
Medical history, n (%)			
Present	3 (21.4)	3 (21.4)	1.000[b]
Absent	11 (78.6)	11 (78.6)	
Cigarette consumption, cigarettes/day	8.64 ± 3.34	11.4 ± 8.58	0.286
Smoking duration, years	3.2 ± 1.37	5.1 ± 2.67	0.031
Age, years	19.4 ± 1.56	19.4 ± 1.22	0.893
BMI, kg/m^2	22.5 ± 5.00	24.1 ± 6.77	0.486

Data are presented as mean ± standard deviation or n (%). BMI, body mass index. p-values were obtained using the independent-samples t-test for continuous variables and Fisher's exact test for categorical variables. Statistical significance was set at $p < 0.05$.

3.2 Changes in LDL-C Levels

Before treatment, mean LDL-C did not differ between groups (control 153.6 ± 10.95 mg/dL; intervention 151.9 ± 10.37 mg/dL; p=0.674). At day 30, the respective values were 154.2 ± 11.68 mg/dL and 120.8 ± 12.21 mg/dL, producing a significant postintervention difference (p<0.001) (Table 2).

LDL-C decreased by 31.1 ± 16.12 mg/dL in the PsP group, whereas controls showed a change of −0.6 ± 14.94 mg/dL. Change scores differed significantly between groups (p<0.001). The within-group decline was significant only among PsP recipients (p<0.001; control p=0.875).

Overall, PsP supplementation for 30 days was associated with a significantly greater reduction in LDL-C levels compared with no supplementation.

Table 2. Changes in LDL-C levels before and after the 30-day intervention

LDL-C measurement	Control group (n=14)	Intervention group (n=14)	Between-group p-value
Baseline, mg/dL	153.6 ± 10.95	151.9 ± 10.37	0.674
Postintervention, mg/dL	154.2 ± 11.68	120.8 ± 12.21	<0.001
Change, mg/dL[1]	−0.6 ± 14.94	31.1 ± 16.12	<0.001
Within-group p-value	0.875	<0.001	

Data are presented as mean ± standard deviation. Between-group comparisons were performed using the independent-samples t-test, whereas within-group comparisons were performed using the paired-samples t-test. Statistical significance was set at p < 0.05.[1]Change was calculated as baseline minus postintervention; positive values indicate a reduction in LDL-C.

4 Discussion

Smoking can adversely reshape the lipid profile through metabolic and oxidative pathways. Studies consistently report more frequent lipid abnormalities in smokers than in non-smokers [16], [17], commonly involving elevated TC, TG, and LDL-C and reduced HDL-C [17]–[20]. Similar LDL-C elevations have been observed in smokers with dyslipidemia [21]. Nicotine-stimulated catecholamine release promotes lipolysis and increases free fatty-acid availability [22], while smoke-related oxidative stress can further impair lipoprotein metabolism [21], [23].

PsP recipients experienced a 31.1 ± 16.12 mg/dL mean decline in LDL-C, while controls had virtually no reduction. The significant between-group contrast supports an association between PsP exposure and improved LDL-C. Interpretation should nevertheless account for longer smoking duration in the intervention arm, because cumulative exposure may worsen lipid status [20], [22], [23]. Baseline LDL-C was comparable, and improvement occurred despite the less favorable smoking history, but unmeasured confounding cannot be ruled out.

The observed reduction in LDL-C may be attributed to the bioactive β-D-glucan contained in PsP. This PsP constituent may influence cholesterol balance by enhancing

lipid breakdown and LDL receptor-dependent clearance, in addition to its antioxidant, anti-inflammatory, and immunomodulatory actions [24], [25]. Such effects could oppose smoking-related oxidative injury and LDL-driven atherogenesis [3], [24], [26], [27]. Animal and experimental findings also show reduced hepatic lipid deposition and lower TG, TC, and LDL-C after exposure to Ganoderma lucidum polysaccharides [28]–[31].

The lipid-lowering effects of PsP are thought to be mediated through its antioxidant by preserving endothelial cell function, suppressing foam cell formation, thereby attenuate smoking-induced oxidative stress, thereby supporting lipid homeostasis and reducing the progression of atherosclerotic changes [11], [24], [32]–[34]. PsP enhances the expression of antioxidant enzymes, scavenges free radicals to maintain lipid integrity, and inhibits reactive oxygen species (ROS) generation, thereby reducing oxidative stress and lipid peroxidation, which contribute to dyslipidemia [24], [26], [27], [33], [35].

The second mechanism by which polysaccharide peptide (PsP) from Ganoderma lucidum may improve LDL-C profiles in smokers is through its anti-inflammatory and immunomodulator properties. PsP may modify TNF-α, IFN-γ, IL-6, and C-reactive protein signaling, which is relevant to hepatic lipogenesis and LDL-receptor activity [11], [26], [32]. PsP also influences lipid metabolism by modulating key enzymes involved in lipid catabolism, inhibiting foam cell formation and atherosclerotic plaque development, and promoting fatty acid breakdown, which collectively may contribute to lower LDL-C levels [27]–[31], [36]. Furthermore, its immunomodulatory activity has been reported to regulate macrophage and immune cell function, thereby supporting lipid homeostasis and reducing systemic inflammation [37], [38].

Although smoking duration was imbalanced, the intervention group still showed a marked LDL-C reduction despite having the longer mean exposure history. This pattern does not eliminate confounding, but it argues against smoking duration as the sole explanation. The observed clinical response is therefore compatible with the known metabolic, antioxidant, and inflammatory actions of PsP and supports further evaluation as an adjunctive nutraceutical.

This study has several limitations. First, the 30-day intervention period was insufficient to evaluate the long-term effects of PsP supplementation on LDL-C levels. Second, dietary intake, particularly fat consumption, was not assessed as a covariate and may have influenced the interpretation of the findings. Although all participants were first-semester students living in dormitories and generally obtained their meals from the same buffet-style cafeteria, individual dietary intake may still have varied. Third, smoking duration differed significantly between the intervention and control groups (5.1 ± 2.67 vs. 3.2 ± 1.37 years, respectively; $p = 0.031$), indicating a potential confounding effect. Finally, the relatively small sample size may limit the precision and generalizability of the findings. Further studies with larger samples, longer intervention periods, and adjustment for dietary intake and smoking duration are needed to confirm these results.

5 Conclusion

PsP supplementation at a dose of 500 mg/day for 30 days was associated with a significant reduction in LDL-C levels among young adult active smokers with dyslipidemia compared with the control group. The intervention group showed a mean LDL-C reduction of 31.1 ± 16.12 mg/dL, whereas the control group showed no significant change. These findings suggest that PsP supplementation may have potential as a nutraceutical approach to improving LDL-C levels in this population. Further studies with larger sample sizes, longer intervention periods, placebo-controlled designs, assessment of dietary intake, and adjustment for smoking-related factors, including smoking duration and intensity, are needed to confirm these findings and improve their generalizability.

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