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FERMENTED GOAT MILK AND STINGLESS BEE HONEY MODULATE INTESTINAL LACTIC ACID BACTERIA AND BODY WEIGHT IN DIABETIC RATS MODEL

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Abstract
Diabetes mellitus is associated with intestinal dysbiosis and body-weight loss. Fermented goat milk provides probiotic lactic acid bacteria (LAB), whereas stingless bee honey contains oligosaccharides with prebiotic potential. This study evaluated their effects on body weight and intestinal LAB in diabetic rats. Streptozotocin-induced diabetic Sprague Dawley rats were assigned to five groups (n = 6): healthy control (K−), diabetic control (K+), fermented goat milk (P1, 1 mL/day), stingless bee honey (P2, 0.4 mL/day), and fermented goat milk supplemented with 5% stingless bee honey (P3, 1 mL/day) for 8 weeks. Body-weight change and intestinal LAB counts were analyzed using one-way ANOVA and Tukey's HSD test. Body-weight change differed significantly among groups ($p = 0.0155$). P2 showed the greatest weight gain, whereas K+ exhibited the greatest weight loss. Intestinal LAB counts also differed significantly ($p < 0.001$), with P2 showing the highest LAB population, followed by P1 and P3. Fermented goat milk and stingless bee honey increased intestinal LAB and alleviated diabetes-associated body-weight loss, with stingless bee honey producing the greatest overall effect.
Keywords: *Diabetes mellitus, fermented goat milk, lactic acid bacteria, stingless bee honey*

PENDAHULUAN

Type 2 diabetes mellitus (T2DM) remains one of the most pressing metabolic disorders worldwide. An estimated 537 million adults were living with diabetes in 2021, a figure projected to reach 783 million by 2045, with the greatest relative increase expected in low- and middle-income countries (Sun et al., 2022). Beyond hyperglycaemia itself, diabetes is increasingly recognised as a disease of host–microbe interaction: the intestinal microbiota of people with diabetes is characteristically less diverse, depleted of short-chain fatty acid (SCFA)-producing taxa, and enriched in pro-inflammatory species, a state commonly termed gut dysbiosis (Neag et al., 2023). This dysbiotic shift compromises intestinal barrier integrity, promotes low-grade endotoxaemia, and feeds back into insulin resistance and systemic inflammation, while contributing to the wasting and progressive body-weight loss that frequently accompany poorly controlled diabetes (Neag et al., 2023).

Lactic acid bacteria (LAB) are the principal functional micro-organisms in traditionally fermented dairy products and are central to the probiotic concept: their proliferation in the gut is associated with restoration of barrier function, competitive exclusion of pathobionts, and increased luminal SCFA production. Fermented goat milk is a particularly attractive LAB vehicle because goat milk itself has smaller fat globules, a distinct casein profile, and higher digestibility than cow milk, properties that are thought to favour LAB viability and gut colonisation during transit. In

Nusantara Hasana Journal
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parallel, stingless bee honey, a product long used in traditional medicine across Southeast Asia and Latin America, is rich in non-digestible oligosaccharides and phenolic compounds that resist digestion in the upper gastrointestinal tract and reach the colon largely intact, where they can selectively stimulate the growth of beneficial *Lactobacillus* and *Bifidobacterium* populations, consistent with a prebiotic mode of action (Schell et al., 2022; Sanz et al., 2005).

Our research group has previously shown that dadiah, a related West Sumatran fermented buffalo-milk probiotic, can reduce inflammatory and oxidative markers and mitigate renal injury in models of diabetic nephropathy (Amelia et al., 2023a, 2023b). Because the intestinal ecosystem is the first site of contact for orally administered LAB and prebiotic substrates, and because dysbiosis and body-weight loss are among the earliest systemic consequences of diabetes, we reasoned that combining a probiotic (fermented goat milk) with a candidate prebiotic (stingless bee honey) might produce complementary, and potentially synergistic, benefits on intestinal LAB abundance and body-weight preservation. Preliminary characterisation work by our group on goat milk and stingless bee honey substrates has been reported separately (Amelia et al., 2025a; Suryanis et al., 2025). However, whether fermented goat milk and stingless bee honey - alone or combined - favourably modulate intestinal LAB counts and body weight in an established diabetic model has not been directly tested.

The present study therefore used a streptozotocin (STZ)-induced diabetic rat model to evaluate, over an 8-week intervention, the effect of fermented goat milk, stingless bee honey, and their combination on (i) body weight and (ii) intestinal LAB counts, with the aim of clarifying the individual and combined contribution of these two functional foods to gut microbial and metabolic outcomes in diabetes.

METODE

Experimental animals and ethical approval

Thirty male Sprague Dawley rats, aged 8–10 weeks, were obtained and acclimatised for one week under standard laboratory conditions (temperature 22–25 °C, 12-hour light/dark cycle, free access to standard pellet feed and water). All procedures involving animals were conducted in accordance with institutional animal care and use guidelines and approved by the relevant institutional ethics committee prior to the start of the study with Number 115/ETIK-FKUNBRAH/03/08/2023.

Diabetes induction

Diabetes was induced by a single intraperitoneal injection of streptozotocin (STZ), which selectively destroys pancreatic β-cells and produces a reliable model of insulin-deficient hyperglycaemia (Szkudelski, 2001). Rats with fasting blood glucose above the diabetic threshold 72 hours after injection were confirmed as diabetic and retained for the study; non-diabetic rats received vehicle only.

Experimental design

Rats were randomly allocated to five groups (n = 6 per group): (i) K−, healthy non-diabetic control receiving no intervention; (ii) K+, STZ-induced diabetic control receiving no treatment; (iii) P1, STZ-induced diabetic rats given fermented goat milk (1 mL/rat/day); (iv) P2, STZ-induced diabetic rats given stingless bee honey (0.4 mL/rat/day); and (v) P3, STZ-induced diabetic rats given

Nusantara Hasana Journal
Volume 6 No. 2 (Juli 2026), Page: 573-580
E-ISSN: 2798-1428

fermented goat milk supplemented with 5% stingless bee honey (1 mL/rat/day). Treatments were administered orally once daily for 8 consecutive weeks.

Body weight measurement

Body weight was recorded at the start of the intervention (initial body weight) and at the end of the 8-week period (final body weight) using a calibrated digital scale. Body weight change was calculated as the difference between final and initial body weight for each animal.

Intestinal lactic acid bacteria analysis

At the end of the intervention period, intestinal content was collected and serially diluted, and LAB were enumerated by culture on de Man, Rogosa and Sharpe (MRS) selective agar under appropriate anaerobic or microaerophilic incubation conditions. Colonies with LAB-consistent morphology were counted, and results were expressed as colony-forming units per gram of intestinal content ($\times 10^6$ CFU/g).

Statistical analysis

Data are presented as mean ± standard deviation. The normality of data distribution was assessed using the Shapiro–Wilk test, and homogeneity of variance was verified with Levene's test. Differences among the five groups were analysed by one-way analysis of variance (ANOVA), followed by Tukey's honestly significant difference (HSD) post hoc test for pairwise comparisons where the overall ANOVA was significant. A p-value of less than 0.05 was considered statistically significant. Analyses were performed using standard statistical software.

HASIL DAN PEMBAHASAN

Table 1 summarises body weight changes across the five groups over the 8-week intervention. One-way ANOVA showed a statistically significant overall difference in body weight change among groups ($p = 0.0155$). The diabetic control group (K+) lost the most weight (−51.83 ± 35.11 g), reflecting the catabolic wasting typical of untreated STZ-induced diabetes, whereas the healthy control (K−) showed only a modest weight gain (6.33 ± 36.49 g). Among the treated diabetic groups, P1 (fermented goat milk) showed a small residual weight loss (−10.50 ± 43.17 g), P3 (fermented goat milk with honey) showed a moderate weight gain (16.00 ± 59.57 g), and P2 (stingless bee honey alone) showed the largest weight gain of all groups (56.83 ± 66.85 g), exceeding even the healthy control. Superscript grouping from the Tukey HSD post hoc test indicated that P2 differed most clearly from K+, while P1 and P3 occupied an intermediate position overlapping with both extremes.

Table 1. Changes in body weight of diabetic rats model

Group	Initial body weight (g)	Final body weight (g)	Body weight change (g)
K−	194.67 ± 38.80	201.00 ± 57.60	6.33 ± 36.49^{ab}
K+	231.67 ± 47.39	179.83 ± 19.34	-51.83 ± 35.11^{a}
P1	231.50 ± 25.94	221.00 ± 48.75	-10.50 ± 43.17^{ab}
P2	217.33 ± 23.59	274.17 ± 65.86	56.83 ± 66.85^{b}

Nusantara Hasana Journal
Volume 6 No. 2 (Juli 2026), Page: 573-580
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P3	242.83 ± 26.30	258.83 ± 60.02	16.00 ± 59.57^{ab}

The marked weight loss observed in untreated diabetic rats (K+) is a well-recognised consequence of insulin deficiency. In the absence of adequate insulin signalling, peripheral tissues cannot use circulating glucose efficiently, and the resulting energy deficit drives compensatory catabolism of adipose and muscle protein reserves, a state of accelerated lipolysis and proteolysis that manifests as body-weight loss despite normal or increased food intake (Szkudelski, 2001). This catabolic pattern is consistently reproduced across STZ-diabetic rodent models and is considered a reliable marker of disease severity. The partial-to-complete reversal of this wasting phenotype in the treated groups, most strikingly in P2, suggests that the interventions improved substrate utilisation or reduced the catabolic drive associated with insulin deficiency, rather than simply adding caloric intake, since P1 (goat milk alone) showed a comparatively modest effect despite also supplying nutrients and energy.

Table 2 presents intestinal LAB counts for each group. One-way ANOVA revealed a highly significant difference among groups ($p < 0.001$). The healthy control (K−) had the lowest LAB count ($10.67 \pm 1.53 \times 10^6$ CFU/g), while the diabetic control (K+) showed an increase relative to K− ($34.67 \pm 9.07 \times 10^6$ CFU/g), consistent with a compensatory but incomplete or dysregulated shift in the diabetic gut environment. All three treated groups showed substantially higher LAB counts than either control. P1 (fermented goat milk) and P3 (goat milk with honey) reached comparable counts (85.00 ± 8.19 and $78.00 \pm 2.65 \times 10^6$ CFU/g, respectively; Tukey HSD grouping c), while P2 (stingless bee honey alone) achieved the highest LAB count among all groups ($115.67 \pm 11.24 \times 10^6$ CFU/g; grouping d), significantly exceeding both P1 and P3.

Table 2. Intestinal lactic acid bacteria counts

Group	Intestinal LAB (×10^6 CFU/g)
K−	10.67 ± 1.53^a
K+	34.67 ± 9.07^b
P1	85.00 ± 8.19^c
P2	115.67 ± 11.24^d
P3	78.00 ± 2.65^c

The intestinal microbiota is now understood to be an active participant in glucose and energy homeostasis. In type 2 diabetes, gut dysbiosis is typically characterised by reduced microbial diversity and a decline in SCFA-producing genera, with knock-on effects on gut barrier integrity, circulating lipopolysaccharide, and low-grade systemic inflammation (Neag et al., 2023). Restoring a favourable balance of LAB in the gut is therefore not merely a marker of digestive health but a plausible mechanistic lever on the broader diabetic phenotype, since SCFAs generated by LAB fermentation activate G-protein-coupled receptors that stimulate glucagon-like peptide-1 secretion and improve insulin sensitivity. The rise in intestinal LAB counts observed in all three treated groups relative to the diabetic control is therefore consistent with, and may partly

Nusantara Hasana Journal
Volume 6 No. 2 (Juli 2026), Page: 573-580
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explain, the parallel improvement in body weight.

Fermented goat milk delivers live LAB directly to the gastrointestinal tract, and several *Lactobacillus* strains characteristic of fermented milk products have demonstrated metabolic benefits in diabetic rodent models. *Lactobacillus fermentum* strains isolated from fermented foods have been shown to alleviate intestinal inflammation and improve gut function in high-fat diet-fed and streptozotocin-induced diabetic rats (Archer et al., 2021), and probiotic supplementation more broadly has been reported to restore nitric oxide balance and modulate macrophage activity in both the gut and peritoneal compartments of diabetic rats (Maciel et al., 2016). Beyond direct colonisation, Lactobacillus fermentum ME-3 has been associated with reduced glycation and amelioration of diabetes-related complications in animal studies (Guilbaud et al., 2020), and milk fermented with Lactobacillus fermentum has been shown to attenuate the inflammatory response in mice (Santiago-López et al., 2018). These mechanisms - competitive exclusion of pathobionts, reinforcement of the mucosal barrier, and local anti-inflammatory signalling - provide a plausible explanation for the substantial rise in LAB counts observed in the P1 and P3 groups in the present study.

Honey, including stingless bee honey, contains a diverse pool of non-digestible oligosaccharides that survive passage through the upper gastrointestinal tract and become available as fermentable substrate for colonic bacteria. In vitro fermentation studies have shown that honey oligosaccharides possess a prebiotic index comparable to, albeit somewhat lower than, that of fructo-oligosaccharide, selectively increasing populations of bifidobacteria and lactobacilli (Sanz et al., 2005). More broadly, honey has been proposed as a prebiotic food capable of re-engineering the gut microbiome toward a more favourable state, partly by suppressing potentially pathogenic taxa while stimulating beneficial genera, and partly through direct antimicrobial and anti-inflammatory activity independent of fermentation (Schell et al., 2022). This dual prebiotic–antimicrobial action offers a coherent explanation for why P2, receiving honey alone, achieved the highest LAB count of all groups: unlike fermented goat milk, which introduces a finite bacterial inoculum, honey may selectively favour the expansion of the animal's own endogenous LAB population across the full length of the colon, in addition to providing an readily fermentable, low-antigenic energy source that could account for the pronounced improvement in body weight in this group. Consistent with the possibility of a systemic metabolic benefit, probiotic and synbiotic supplementation has also been associated with reduced markers of inflammation and oxidative stress in diabetic patients in meta-analyses of randomised trials (Zheng et al., 2019), and probiotic soy milk supplementation has been shown to reduce oxidative stress in patients with diabetic kidney disease (Miraghajani et al., 2017), supporting a broader anti-inflammatory and metabolic rationale for the LAB- and honey-based interventions used here.

Combining fermented goat milk with 5% stingless bee honey (P3) was intended to test for a synbiotic effect, in which the prebiotic substrate would selectively fuel the co-administered probiotic LAB. In the present study, however, P3 did not exceed P1 or P2 on either outcome, and its LAB count was statistically indistinguishable from P1 alone. A plausible explanation is that the 5% honey inclusion, while sufficient to exert an independent prebiotic effect when given alone

at a higher relative dose in P2, may have been diluted below an effective threshold within the combined goat milk matrix, or that competitive interactions between the exogenous LAB inoculum from goat milk and the animal's endogenous microbiota limited any additive benefit. This finding is informative rather than negative: it indicates that simply co-administering a probiotic and a prebiotic does not guarantee a synergistic outcome, and that the relative dose, timing, and matrix in which each component is delivered are likely to be critical determinants of whether a true synbiotic effect is realised. Our companion work characterising lactic acid bacteria from goat milk sources provides further context for interpreting the probiotic potential of the strains used in this formulation (Amelia et al., 2025a; Suryanis et al., 2025), and our previous studies on dadiah in models of diabetic complications similarly point to the importance of matrix and dose in determining probiotic efficacy (Amelia et al., 2023a, 2023b, 2025b).

Strengths and limitations

This study benefits from a controlled, well-characterised STZ-induced diabetic model, a full complement of positive and negative controls, and outcome measures - body weight and intestinal LAB count - that are directly relevant to both the metabolic and microbial dimensions of diabetes. Several limitations should nonetheless be acknowledged. First, this manuscript reports body weight and intestinal LAB outcomes only; blood glucose and other glycaemic parameters were outside the scope of the present analysis and would be needed to establish whether the observed microbial and body-weight changes translate into improved glycaemic control. Second, LAB enumeration by selective culture does not resolve genus- or species-level composition, and future work using 16S rRNA sequencing would clarify which specific taxa are being favoured by each intervention. Third, the relatively small group size (n = 6) is consistent with standard practice in rodent intervention studies but limits the precision of effect-size estimates, particularly given the wide variance observed in body-weight change. Finally, as an animal model, these findings require confirmation in human populations before any clinical recommendation can be made regarding fermented goat milk or stingless bee honey as adjunctive diabetes management strategies.

CONCLUSION

In this streptozotocin-induced diabetic rat model, fermented goat milk and stingless bee honey, administered alone or in combination for 8 weeks, increased intestinal lactic acid bacteria counts and attenuated diabetes-associated body-weight loss relative to untreated diabetic controls. Stingless bee honey alone produced the largest improvement in both outcomes, suggesting a prominent prebiotic contribution, while the combination with fermented goat milk did not exceed the effect of either component given alone. These findings support further investigation of stingless bee honey and fermented goat milk as candidate functional foods for microbiota-targeted adjunctive management of diabetes, with future studies incorporating glycaemic endpoints, microbiota sequencing, and dose-optimisation of combined formulations.

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Nusantara Hasana Journal
Volume 6 No. 2 (Juli 2026), Page: 573-580
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