

PANCREATIC HISTOPATHOLOGY IN OBESITY AND THE POTENTIAL ROLES OF DIETARY MODIFICATION AND DADIH DERIVED PROBIOTICS

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Abstract

Obesity can impair the function of the pancreas and result in changes to both hormone-producing and digestive enzyme-producing tissues. Dietary modifications and interventions on gut microbiota could provide protection of pancreas. However, there is little evidence on the benefits of dadih, a traditional fermented buffalo milk product. This narrative review describes the histopathological changes of the pancreas in obesity and the benefits of dadih dietary modification and probiotics. The relevant literature was retrieved by a targeted search. Some studies indicate that obesity can change the structure of the islets of Langerhans and induce the accumulation of fat, inflammation and fibrosis in the pancreas. Energy restriction might alleviate stress in pancreatic cells. Dadih supplementation has also demonstrated promising effects on pancreatic histopathology in rats with chemically induced diabetes. However, these results cannot be directly extrapolated to diet-induced obesity. In a small study in women with type 2 diabetes, supplementation with the dadih-derived probiotic strain Dad-13 did not show significant improvement in HbA1c compared to placebo. Mechanisms possibly involved include changes in microbial metabolites, improved intestinal barrier function, and reduced inflammation, but these are not confirmed for dadih. To summarize, dadih could be investigated as an adjunct to dietary modification, however, there is insufficient evidence to determine its protective effects on the pancreas in obesity. Future studies should use standardized preparations, appropriate control groups and quantitative determination of pancreatic histopathology.

Keywords: obesity; pancreatic histopathology; dietary modification; dadih; probiotics; gut microbiota

1. INTRODUCTION

Obesity is a significant global health issue. Obesity increased in many parts of the world from 1990 to 2022, according to a pooled analysis of data from 3,663 population-based studies with 222 million participants. Obesity in adults and children was classified using different criteria, so prevalence estimates should be interpreted according to the age and the criteria used. [1] The current review will

cover the effects of excess body fat and diet on pancreatic structure, and the potential for dietary interventions to improve these changes. Animal studies have indicated that obesity can affect the hormone-producing islets of Langerhans and the surrounding exocrine tissue which produces digestive enzymes. High-fat diet is associated with islet inflammation and fibrosis, pancreatic fat accumulation and acinar cell damage. [2,3] Therefore, understanding pancreatic changes in obesity requires a look at both endocrine and exocrine tissues. Dadih is a traditional West Sumatran buffalo milk fermented product. The microbial composition is different in the producers and some strains isolated from dadih have been studied as probiotics. Whole dadih, individual bacterial strains and commercial multistrain probiotics are different preparations and their effect should be evaluated separately. [4,5] This review summarizes evidence regarding dietary modification and dadih, discusses limitations in extrapolating findings across different models and identifies priorities for pancreatic histopathology research in obese rat.

2. LITERATURE SEARCH AND SCOPE

This review was performed in a focused narrative manner. Public web searches for combinations of “dadih” or “dadiah,” “pancreatic histopathology,” “obese rats,” “high fat diet,” “dietary restriction,” and “Dad-13.” Articles relevant to the search were checked for publisher pages, PubMed records, and available full texts. We also searched reference lists of directly relevant articles. Bibliographic details and access routes were checked again in Agustus 2026. The search was intended to identify studies directly relevant to the review question rather than to provide comprehensive coverage.

Sources were purposively selected based on their relevance to pancreatic structure, diet interventions, microbiology of dadih, or metabolic effects of dadih derived strains. Animal experiments, human trials, studies of microbial characterization and consensus statements were analyzed separately. We did not perform comprehensive database searching, independent duplicate screening, formal risk-of-bias assessment, or meta-analysis. Where available, full texts were consulted; evidence available only through indexed abstracts was interpreted conservatively. The synthesis should, therefore, be considered as a critical narrative review rather than a systematic review.

2.1 Pancreatic Histopathology in Obesity

Endocrine and Exocrine Changes

Rebours and colleagues examined male Wistar rats fed a normal or high-fat diet for three or six months, along with a group that had undergone bariatric surgery. Inflammation and fibrosis in the islets of Langerhans were more frequent after six than after three months of high-fat feeding ($p = 0.0139$). Rats undergoing surgery had fewer islets with these changes ($p = 0.0004$) and less pancreatic fat accumulation ($p = 0.005$) than rats fed a high-fat diet for 6 months. However, the difference in iron deposition was not statistically significant ($p = 0.06$). [2] These results suggest that islet size should be evaluated in conjunction with fibrosis, vascular alterations and metabolic activity. Islet size alone is insufficient to distinguish whether the tissue is damaged or recovering. Matsuda et al investigated Zucker diabetic fatty rats, a model with a genetic predisposition to obesity and diabetes, fed a normal or high-fat diet. At 18 weeks of age, evidence of pancreatic fat accumulation was found, including lipid droplets in acinar cells, which produce

digestive enzymes. Increased pancreatic fibrosis and serum levels of exocrine pancreatic enzymes were detected at 24 weeks. [3] These observations suggest that damage of the exocrine pancreas could be associated with metabolic disease. However, this model makes it difficult to distinguish the effects of genetic susceptibility from those of diet.

2.2 Species Differences and Interpretation of Tissue Changes

Cabrera and colleagues discovered there is a different organization of human and mouse pancreatic islets. In human islets, α , β and δ cells are scattered throughout the islet, not β cells mainly in the middle with other cells at the periphery. [6]. Therefore, human islet descriptions should not be directly extrapolated to rats. Islet studies should specify species, strain, age, pancreatic region and staining method for comparison between studies. The appearance of the tissue alone is not enough to confirm the pancreatic damage or recovery. The tissue structure and pathology are shown by hematoxylin and eosin staining, but an empty-looking space does not signify cell death and a larger islet does not indicate that new β cells have been produced. More convincing would be a stain for insulin and glucagon, together with standard tissue structure measurements and tests for cell death or cell division. These methods are suggestions for future research and not conclusions confirmed by this review.

2.3 Dietary Modification and Pancreatic Stress

Dadih as a Fermented Food and Source of Probiotic Candidates

Sun et al. randomly assigned 45 male 14 to 16-month-old Sprague-Dawley rats to a normal diet, a high-fat diet or a calorie-restricted diet. The restricted diet supplied half the calories of the normal diet. Assessments were performed at baseline, 8 and 16 weeks. By 16 weeks, the high-fat diet increased markers of programmed cell death including apoptotic index and cleaved caspase-3. These markers were lowered with calorie restriction compared with the normal diet. High-fat feeding decreased proteins that support cell survival, BCL2 and BCLXL, whereas calorie restriction increased them. Both groups exhibited higher levels of markers of autophagy, the process by which cells break down and recycle their components. [7] These results indicate why it is important to measure autophagy together with cell death. The large caloric restriction used in this study also precludes comparisons with less restrictive diets. Some pancreatic lesions improved after surgery, in the earlier-discussed bariatric surgery study. [2] This does not mean, however, that the same results would be obtained with dietary changes alone, since surgery also changes digestive and hormonal functions. Therefore, calorie restriction, replacing a high fat diet, changing nutrient composition and bariatric surgery should be evaluated separately. Studies combining dadih with dietary modification should describe the diet clearly. Researchers should report its energy density, amount of food given and eaten, proportions of carbohydrate, protein and fat, fiber content and length of intervention. They should also mention if food intake was ad lib or restricted. A pair-fed group, that is, a group given the same amount of food as the intervention group, can be useful to dissociate the effects of reduced intake from those of dadih. Calories and nutrients from dadih should also be included in the calculation of total intake.

2.4 Proposed Gut Pancreas Mechanisms

There are two animal studies that give a basis for investigating how gut microbes might influence pancreatic health. Cani and coworkers demonstrated in

mice that metabolic endotoxemia due to increased exposure to bacterial lipopolysaccharide was associated with inflammation and metabolic derangements. [10] These findings support the investigation of bacterial signals that induce inflammation but do not suggest that dadih inhibits this process in the pancreas. Yadav et al. investigated a multi-strain probiotic preparation in mice. The metabolic benefits were correlated with production of butyrate and secretion of glucagon-like peptide-1 (GLP-1), a hormone that regulates blood glucose levels. [11] This preparation was different from dadih, but the results support the need for further research on microbial metabolites and gut hormones contribution for the effects of dadih.

Future studies should evaluate gut microbial composition and activity together with blood markers of metabolism and pancreatic tissue changes. Relevant measurements are short chain fatty acids, intestinal permeability, endotoxin exposure, inflammation and oxidative damage. To develop a mechanism, researchers need to show that dadih modifies the proposed biological mediator and that this modification affects pancreatic outcomes. “Just reducing blood glucose levels or changing gut bacteria would not be enough to explain this link.

2.5 Additional Probiotic Evidence for Pancreatic and Intestinal Outcomes

Sariyanti and colleagues studied *Lactobacillus acidophilus* FNCC 0051 in 16 rats: 12 received streptozotocin to induce diabetes, while four served as controls. After 21 days, both probiotic concentrations, reported as 1.5×10^8 and 1.5×10^9 CFU/mL/day, reduced inflammation in the pancreatic islets, as measured by insulinitis scores, compared with untreated diabetic rats ($p = 0.040$ and $p = 0.034$, respectively). The two probiotic groups did not differ significantly ($p = 0.131$). [12] These findings show a histological benefit for this particular strain, but do not establish a benefit for dadih or greater effectiveness at the higher concentration. The chemically induced diabetes model also differs from insulin resistance associated with obesity.

Mihailović and colleagues investigated *Lactobacillus paraplantarum* BGCG11 in diabetic rats, administering it before or after diabetes induction. They reported preservation of pancreatic β cells and insulin production, together with improved duodenal tissue integrity and reduced intestinal permeability. [13] Assessing the intestine and pancreas in the same study helps researchers examine the relationship between these organs. However, the strain and disease model differ from those used to investigate dadih in obesity. Improvements in both organs do not prove that intestinal changes caused the pancreatic benefits or that whole dadih would have the same effects.

2.6 Evidence Specific to Dadih and Dadih Derived Strains

Amelia and colleagues studied male Wistar rats with streptozotocin-induced diabetes for eight weeks. The animals were divided into five groups of five rats each. One group received whole dadih at 3 g per approximately 300 g body weight daily, while other treatment groups received bacterial isolates. Mean pancreatic injury scores were 2.80 ± 0.83 in untreated diabetic rats, 0.60 ± 0.54 in rats receiving dadih, and 0.40 ± 0.54 in normal controls. Lower scores indicated less tissue damage. [14] These findings suggest a potential benefit, but the small groups, the scoring method, and inconsistencies in the reported isolate doses limit interpretation. The chemically induced diabetes model also differs from diet-

induced obesity. Although the tissue appeared close to normal, this does not prove that new β cells had formed.

2.7 Dadih Derived Strain in a Dietary Liver Disease Mode

Amelia and coworkers investigated male Wistar rats with streptozotocin induced diabetes for eight weeks. The animals were grouped into 5 groups of 5 rats . One group was given whole dadih at 3 g per 300 g body weight approximately daily and the other treatment groups were given bacterial isolates. The mean pancreatic injury scores of untreated diabetic rats, dadih treated rats and normal controls were 2.80 ± 0.83 , 0.60 ± 0.54 and 0.40 ± 0.54 respectively The lower the score, the less tissue damage. [14] These results suggest a potential benefit, but the small groups, scoring method, and inconsistencies in the doses of the reported isolates limit interpretation. Also, the chemically induced diabetes model differs from diet-induced obesity. The tissue looked almost normal, but there was no evidence new β cells were being made

2.8 Dietary liver Disease Mode : Dadih Derived Strain Metabolic Findings in

Minhajet et al. studied Dad-13 for 6 weeks in Sprague-Dawley rats with liver disease induced by a high-fat high-fructose diet . The probiotic preparation had 3×10^9 CFU/g. The average non-alcoholic fatty liver disease (NAFLD) activity score was lower in rats supplemented with the supplement compared to the diet alone (2.75 ± 0.89 vs. 5.00 ± 1.41 ; $p < 0.01$). But there was no significant difference in lobular inflammation, a component of the score. Serum levels of lipopolysaccharide-binding protein were also lower. [15] These results are supportive of Dad-13 in a model of diet-induced metabolic disease, but the study examined the liver, not the pancreas. And the rats who got the supplement ate fewer calories, so the benefits can't be attributed to the probiotic alone.

3. HUMAN STUDIES

Rahayu et al. performed a randomized, double-blind, placebo-controlled trial in 60 adults with body mass index (BMI) ≥ 25 kg/m². Participants were given Dad-13 at 2×10^9 CFU/day or placebo for 90 days. Mean body weight decreased from 84.54 to 83.14 kg and BMI decreased from 33.10 to 32.57 kg/m² in the probiotic group ($p = 0.04$ for both). Lipid profiles, fecal short-chain fatty acids and fecal pH were not significantly changed. These results suggest the effects may differ across outcomes.[16] Weight changes or gut bacteria don't confirm pancreatic recovery or a butyrate mechanism. A large change within the probiotic group alone does not prove that it did better than placebo .

Rustanti et al. evaluated Dad-13 in women with type 2 diabetes for 11 weeks. The probiotic group was given 1 g of powder containing 10^{10} cfu / g per day. HbA1c decreased from $9.34 \pm 2.79\%$ to $8.32 \pm 2.04\%$ in the probiotic group ($n = 10$), but the decrease was not significantly different from that in the placebo group ($n = 8$). [17] The result is preliminary and does not prove a treatment benefit. The conclusions are also limited by the small number of participants and the lack of assessment of pancreatic tissue.

Table 1 summarizes selected studies on pancreatic changes, dietary interventions and probiotics. Rather than listing all the available studies, it summarizes the evidence that has been discussed in this review

Table 1 Selected evidence concerning pancreatic outcomes and metabolic effects

Study	Model and exposure	Relevant finding	Interpretive limit
Matsuda et al. 2014 [3]	ZDF rats; standard or high-fat diet; assessments at 12, 18 and 24 weeks of age.	Pancreatic lipid accumulation and later fibrosis or acinar injury.	Genetically susceptible model; no dadih intervention.
Sun et al. 2016 [7]	45 older male SD rats; normal, high-fat or calorie-restricted diet; up to 16 weeks.	Diet-dependent differences in metabolic and islet stress markers.	Aging model; severe calorie restriction limits extrapolation.
Rebours et al. 2018 [2]	Male Wistar rats; high-fat feeding and a bariatric surgery arm.	Endocrine and exocrine lesions; fewer selected lesions after surgery.	Surgical effects cannot be attributed to dietary modification alone.
Amelia et al. 2024 [14]	STZ diabetic Wistar rats; n = 5/group; dadih or isolates for 8 weeks.	Mean injury score 0.60 with dadih versus 2.80 in diabetic controls.	Not a diet-induced obesity model; small sample and dose reporting inconsistencies.
Rahayu et al. 2021 [16]	60 adults; Dad-13 2×10^9 CFU/day versus placebo; 90 days.	Within group weight and BMI decline; no significant lipid or fecal SCFA change.	No pancreatic histology; defined strain rather than whole dadih.
Rustanti et al. 2022 [17]	Women with T2D; Dad-13 10^{10} CFU/day versus placebo; 11 weeks.	Within group HbA1c decline; no significant advantage over placebo.	Small groups; no pancreatic structural endpoint.
Sariyanti et al. 2022 [12]	STZ diabetic rats; FNCC 0051; 21 days; two concentrations.	Lower insulinitis scores versus diabetic control at both concentrations.	Different strain and model; dose groups did not differ significantly.
Mihailović et al. 2024 [13]	Diabetic rats; BGCG11 before or after disease induction.	Pancreatic preservation alongside intestinal functional improvement.	Not dadih; concurrent changes do not establish mediation.

Study	Model and exposure	Relevant finding	Interpretive limit
Minhajet et al. 2025 [15]	HFFr-fed SD rats; Dad-13; six weeks.	Lower NAFLD activity score and LBP; no significant change in lobular inflammation.	Hepatic endpoint; lower calorie intake may contribute.

Abbreviations: ZDF, Zucker diabetic fatty; SD, Sprague–Dawley; STZ, streptozotocin; T2D, type 2 diabetes; HFFr, high-fat, high-fructose; LBP, lipopolysaccharide-binding protein; SCFA, short-chain fatty acid; NAFLD, non-alcoholic fatty liver disease. No pooled effect estimate was calculated.

3.1 Integrated Interpretation of the Evidence

The results need to be interpreted according to what each study tested and measured. Dietary studies studied pancreatic changes with diet. Whole dadih studies in rats with chemically-induced diabetes revealed pancreatic findings, whereas human studies mainly examined metabolic parameters. The studies support the need for more research, but do not prove that dadih and dietary modification regenerate pancreatic tissue. [2,7,12–17]

The whole dadih is different from the isolated strain of bacteria because it contains nutrients from the milk, live bacteria and the products of fermentation. No benefit from one preparation indicates which component caused it. Future studies should investigate dadih and dietary modification separately and in combination to assess their effects and whether the findings were due to reduced calorie intake.

The changes in pancreatic tissue, gut bacteria and blood markers need to be evaluated separately. Some studies showed benefits, while others did not show any significant changes in lipid levels, fecal metabolites, HbA1c compared to placebo, or liver inflammation . [15–17] Both positive and nonsignificant results should be considered. Future studies should define a primary pancreatic outcome and employ metabolic and intestinal measurements to help explain the results.

3.2 Research Gaps and Priorities

It remains unclear whether dadih provides additional benefits to the pancreas beyond dietary changes alone in rats with diet-induced obesity. Other probiotic studies and the Dad-13 liver study support further investigation, but they do not answer this question. [12,13,15] None of the studies included in this review directly confirms the combined effect of whole dadih and dietary modification on pancreatic histopathology in this model.

Future studies should test dietary modification and dadih separately and together, with healthy and obese control groups. Food and calorie intake should be measured and, where appropriate, matched between groups. Outcomes could include the area of insulin-producing cells, islet size, pancreatic fat and fibrosis, fasting metabolic markers, and glucose tolerance. Improved tissue appearance alone is not enough to prove regeneration; evidence of new cell formation is also needed. Each animal should be treated as one experimental unit, even when several tissue sections or islets are examined. A statistical test of interaction is needed before claiming that dadih and dietary modification have a synergistic effect.

3.3 Limitations

This is a narrative review and studies were selected. There was no formal bias assessment. The studies were heterogeneous regarding animal species, age,

disease model, preparation and outcomes measured. Isolated strains and multistrain probiotics are different interventions than whole dadih and should be evaluated separately. Some full texts were not available so findings from abstracts were interpreted with caution. Such differences exclude comparison and preclude firm conclusions on the effects of dadih in diet-induced obesity.

4. CONCLUSIONS AND RECOMMENDATIONS

Obesity can impact both the hormone-producing islets and the enzyme-producing tissues of the pancreas. Changes are diet and animal model dependent. Dadih needs more study in combination with dietary modification, but the present evidence does not support that it reverses pancreatic injury in diet-induced obese rats. Indirect support is provided by studies in diabetic animals and human Dad-13 trials. Future studies should separate the effects of diet and dadih, assess pancreatic tissue in blinded conditions and test the mode of action of the intervention before claiming for regeneration, synergy or clinical benefit.

Based on these conclusions, several recommendations can be made. First, future experimental studies should use standardized and well-characterized dadih preparations, and should clearly distinguish whole dadih from isolated strains and commercial multistrain probiotics. Second, studies should include healthy control, obese control, dietary modification alone, dadih alone, and combined intervention groups, with food and calorie intake measured and, where appropriate, matched. Third, pancreatic histopathology should be assessed in a blinded manner using quantitative measures, such as insulin and glucagon staining, islet size, pancreatic fat, and fibrosis, supported by markers of cell death and cell division. Fourth, larger and well-designed human studies are needed before any clinical benefit is claimed. Until such evidence is available, dadih may be considered only as a complementary food alongside dietary modification, and not as a substitute for established obesity and diabetes management.

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6. AUTHOR CONTRIBUTION STATEMENT

Yusti Siana contributed to the conceptualization of the review, literature search, data curation, and writing of the original draft. Faridah Mohd Said contributed to supervision, critical review of the content, and validation of the interpretation of the evidence. Devi Nallapan contributed to the review methodology, literature appraisal, and review and editing of the manuscript. Rifkin Malik contributed to the interpretation of pancreatic histopathology evidence and the review of the manuscript. Debie Anggraini contributed to literature collection, organization of references, and the editing of the manuscript. All authors have read and approved the final version of the manuscript and are accountable for its content.

7. CONFLICT OF INTEREST STATEMENT

The authors declare that there are no financial, personal, or professional conflicts of interest that could have influenced the preparation of this review.

8. RESEARCH ETHICS STATEMENT

This article is a critical narrative review based on previously published literature. The authors did not conduct experiments on humans or animals, and no new participant data were collected. Therefore, ethical approval and informed consent were not required.

9. DATA AVAILABILITY

No new datasets were generated or analyzed in this study. All data discussed in this review were obtained from the published sources listed in the References section.

10. GENERATIVE ARTIFICIAL INTELLIGENCE USE STATEMENT

The authors used generative artificial intelligence tools only to assist with language editing and the formatting of the manuscript. The literature search, selection and interpretation of the evidence, and the conclusions were carried out by the authors. The authors reviewed and verified all content generated with the assistance of these tools and take full responsibility for the content of this article.

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