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Allicin from Garlic as a Potential Stimulator of Pancreatic Beta Cells for Blood Glucose Regulation in Diabetic Mice

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ABSTRACT

Background: One of the most prevalent metabolic & endocrine disorders in the globe is diabetes mellitus (DM). Insulin secretion impairment that would resulted from pancreatic β cells damage, which were believed to be important cells implicated in diabetes mellitus development.

Aim: to examine and clarify the underlying mechanism of allicin's protective actions against pancreatic β cell damage.

Methods: The STZ-induced mice DM model was used to study the pharmacological effects of allicin on T2DM in vivo. The studies include monitoring fasting blood sugar levels, conducting oral glucose tolerance tests, measuring serum insulin concentrations, and tracking body weight.

Results: Allicin appears to significantly reduce blood glucose levels and improves insulin secretion and islet architecture, as well as the weight loss associated with diabetes, thus providing important support for metabolism.

Conclusion: Allicin treatment notably reduced progression of T2DM induced by STZ, indicating that allicin may represent a treatment option for patients suffering from T2DM.

1. Introduction

A global public health concerns, diabetes mellitus (DM) is presently becoming an epidemic on a worldwide scale. Globally, the prevalence of diabetes is predicted to be 9.3% in 2019 (463 million), 10.2% in 2030 (578 million), and 10.9% in 2045 (700 million) [1]. Type I diabetes mellitus (T1DM) was

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caused by complete lack of insulin in the body, while Type II diabetes mellitus (T2DM) was caused by systematic resistance toward the insulin impact. Insulin insufficiency brought on by autoimmune-mediated damage and injury of pancreatic β cells is a hallmark of T1DM. It is a chronic condition that results in hyperglycemia along with additional symptoms like weight loss, headaches, polydipsia, polyphagia, polyuria, abdominal pain, and ketoacidosis [2,3]. Globally, 8.8% of individuals have diabetes, with 10% to 15% developing type 1 diabetes, in accordance to the International Federation of Diabetes [4]. Microvascular problems such as retinopathy, neuropathy, and nephropathy can result from persistently elevated blood glucose levels, which can then have a significant negative societal and financial impact [3].

Currently, the primary focus of clinical management for patients with type 1 diabetes is rigorous insulin therapy, which aims to improve the level of glycosylated hemoglobin, lower complications, & maintain blood glucose levels as close to normal as feasible [5]. Nonetheless, insulin therapy still has some drawbacks. Therefore, finding new medications or treatment methods for type 1 diabetes is crucial.

Obesogenic settings, fast urbanization, and aging are the main causes of this rising trend for T2DM, which makes up around 90% of the total. Additionally, the prevalence of diabetes is increasing due to increased T1DM incidence rates [6,7]. The reason behind this increase is still unknown. Better survival of individuals with diabetes due to early detection, better diabetes management, and a resulting decrease in premature mortality is another factor contributing to the higher prevalence in some communities [8]. And last, because they live longer, the growing proportion of younger persons with T2DM in recent years also helps to raise the prevalence of T2DM overall.

Medicinal herbs are a valuable source of therapeutic antidiabetic drugs in modern medicine. Numerous herbal remedies have been suggested for the management of diabetes [9,10]. Among the most widely used herbs in both traditional and modern medicine worldwide is garlic (Allium Sativum) because of its anti-inflammatory, anti-bacterial, anti-diabetic, anti-cancer, anti-immunomodulatory and anti-oxidant, effects [11-13]. Garlic's bioactive components are an exceptional natural source of pharmaceutically active chemicals, with great potential for incorporation into functional food or nutraceutical formulations targeted at preventing and managing particular diseases [14]. Researchers are interested in garlic's potential as a diabetes treatment. Garlic reduces pathogenic changes in diabetes mellitus, oxidative stress, and pancreatic cell damage [15]. A meta-analysis of nine randomized controlled studies with 768 participants with T2DM found that taking supplements of garlic significantly decreased their levels of fructosamine & glycosylated hemoglobin [16]. As garlic cloves are crushed, alliinase converts alliin to allicin, the primary bioactive ingredient [17]. The primary breath metabolites of allicin is allyl methyl sulfide (AMS), which is absorbed efficiently even though some of it is metabolized into a readily absorbed intermediate [18]. Since allicin is hydrophobic, it effectively penetrates cell membranes without causing fusion, aggregation, or membrane leakage [19]. According to some theories, allicin may lessen pathological heart hypertrophy by activating the autophagy system [20]. Additionally, allicin has been shown to improve disc degeneration by reducing oxidative stress brought via advance oxidative proteins product & mitochondrial apoptosis in human nucleus pulposus cells [21].

Streptozotocin is a small-molecule antibiotic and DNA methylating agent. This substance revealed significant methylation of the kidney, pancreas, and liver cells. STZ's unique activity on the pancreas has led to its usage in treating human pancreatic cancers and induce diabetes in animals [22]. These changes in insulin and blood glucose levels are indicative of abnormalities in -cell activity. STZ inhibits glucose oxidation and lowers insulin secretion and synthesis [23-25]. The most important diabetogenic chemical

as far as diabetes research is concerned is STZ. Insulin-dependent diabetes is caused by its alkylating properties, which alter biological macromolecules, damage DNA, and destroy cells. Another explanation for STZ's capacity to inhibit glucose-induced insulin release is that it targets mitochondrial DNA, which disrupts the signaling function of mitochondrial metabolism in cells [25,26]. Thus, in order to demonstrate the underlying mechanism, we would attempt to examine the protective effect of allicin on the STZ-induced mice T2DM model in vivo.

2. Materials and Methods

All materials used in this study were of analytical or pharmaceutical grade. Fresh garlic bulbs (Allium sativum) were obtained from a local market in Baghdad, Iraq, and processed immediately for allicin extraction, streptozotocin (STZ) (Cayman Chemical, USA), sodium citrate buffer pH 4.5 (Loba Chemie, India), ethanol 70% (Al-Hikmah, Jordan), and normal saline 0.9% (Pioneer, Iraq) were used for compound preparation and administration. Distilled water (Pioneer, Iraq) was utilized throughout the study. ELISA kits for insulin was purchased from Sunlong Biotech, China. A digital glucometer and test strips (Accu-Chek, Roche Diagnostics, Germany) were used for blood glucose measurements.

2.1. Preparation of Crude Aqueous Garlic Extract Containing Allicin:

Fresh garlic bulbs (Allium sativum) were obtained from a local market, peeled, and cleaned. A total of 10 grams of garlic cloves were finely crushed using a sterile mortar and pestle to form a homogeneous paste. The crushed garlic was left to stand at room temperature (~22–25°C) for 15 minutes to allow enzymatic conversion of alliin to allicin via endogenous alliinase activity.

After the incubation period, 100 mL of sterile distilled water was added to the paste and the mixture was thoroughly homogenized. The resulting suspension was filtered through sterile gauze to remove particulates. The filtrate, representing the crude aqueous garlic extract rich in allicin, was collected in sterile amber vials and stored at 4°C until use. All extracts were freshly prepared daily and used within 4 hours of preparation to minimize allicin degradation. The extract was administered to animals via oral gavage at a dosage of 10 mg/kg body weight/day (adjustable according to experimental design), using a sterile feeding needle. The final gavage volume did not exceed 1–2 mL per 100 g of body weight [27].

2.2. Preparation of Streptozotocin for Injection:

Based on the animals' weight and the STZ dosage (50 mg/kg, BW), the proper amount of STZ were freshly dissolved in citrate buffer. Twenty minutes prior to injection, STZ was freshly prepared, and aluminum foil was placed over the container to shield the buffer from the sun [28].

2.3. Sodium Citrate Buffer Preparation:

In order to create a 1 molar sodium citrate buffer, 2.1 g of citric acid & 2.94 g sodium citrate were combined in 50 milliliters of distilled water. The pH was then adjusted to 4.5 utilizing NaOH, & the volume was then increased to 100 milliliters [29].

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2.4. Induction of Diabetes:

After 1-week acclimatization, diabetes was induced in experimental animals with a low-dose of STZ according to an established protocol which lead to moderate beta-cell failure without complete destruction. A Diabetes model was induced by IP injection of a single dose of streptozotocin 50 mg/kg body weight for five consecutive days [30]. Mice were fasted 12 hours prior to each injection to facilitate STZ uptake by pancreatic beta cells, but allowed free access to water.

This approach was adopted to develop a model of Type 2-like diabetes, in which there is retention of residual beta-cell function. This enables the ability of allicin to induce insulin release or, alternatively, protect beta cells from damage, to be evaluated. Fasting blood glucose levels that were reflected by the tail vein blood glucose level measured after 72 h post- last injection using a digital glucometer (Accu-Chek, Roche Diagnostics GmbH, Mannheim, Germany). The diabetic mice, exhibiting the blood glucose more than 250 mg/dL after overnight fasting, were used to treat with the drug [31].

This protocol is further in agreement with two other studies conducted, which reported multiple low-dose STZ regimens as an effective approach in induction of stable hyperglycemia along with pancreatic function of the studies with preserved pancreatic function in Swiss albino and outbred rodent models [32].

2.5. Experimental Animal:

For the current study, male Swiss albino mice weighing 25–30 g and 8–10 weeks of age had been used. Animals were obtained from the Center for Drugs Control & Research of the Iraqi Ministry of Health. Swiss albino mice were used due to their fully documented sensitivity to streptozotocin-induced hyperglycemia and their capability in simulating the assessment of the action of natural products on pancreatic beta-cell function. All the mice were kept in standard laboratory conditions consisting of 12 h light/12 h dark cycle, a room temperature of 22 ±2°C, and a relative humidity of 50–60%. They also had free access to water ad libitum and a standard pellet diet. The experimental protocols were performed in adherence to the institutional animal ethics guidelines by Al-Bayan University's College of Pharmacy, based on the guidelines of the AVMA 2020 [33].

2.6. Study Design:

Male Swiss Albino mice (total = 30; 8–10 weeks old; 25–30 g weight) were randomly assigned into three experimental groups (n = 10). All the mice underwent the same housing conditions and were subjected to treatment once diabetes was established, with fasting blood glucose (FBG) levels >250 mg/dL.

The animals were distributed as follows:

- Group I – Normal Control: Received vehicle (distilled water) without STZ or allicin.

- Group II – Diabetic Control: Induced with STZ but received no treatment (vehicle only).
- Group III – Allicin-Treated Diabetic Group: Induced with STZ and treated with allicin, administered via intraperitoneal (IP) injection.

The dose and administration form of allicin were chosen according to previous preclinical studies which have showed its pharmacological activity in glucose metabolism and reduction of oxidative stress [34-36]. Mice were weighed and fasted blood glucose concentration, fasting serum insulin level, and oral glucose tolerance testing were assessed during the treatment period. This study was designed to investigate the ability of allicin to enhance glucose control and perhaps even induce insulin secretion by modifying the beta-cell inpartially damaged pancreas.

2.7. Dose Determination and Administration Protocol:

The administered dose of allicin in the present study was 40 mg/kg/day, because allicin has been reported to be effective in blood glucose control, as well as in reducing oxidative stress and protecting pancreatic b-cells in rodent diabetes studies. Such dose is known to be efficient and not toxic or significantly adverse by ip administration. Freshly prepared allicin was dissolved in sterile normal saline (0.9% NaCl) each day and administered by IP injection (volume: ≤ 0.5 mL per 25 g body weight). Treatment was continued uninterrupted for 21 consecutive days, 3 days after formal confirmation of diabetes induction. This duration was chosen to enable adequate evaluation of the allicin's capability to modulate insulin secretion, and induce recovery of the β-cells. The dose was given at the same time every day to minimize circadian variation. Animals were observed daily for treatment-related changes in food consumption, behavior, body weight, and health status [34,35].

2.8. Blood samples collection:

To monitor the effect of allicin on glucose metabolism and insulin secretions, blood samples were collected at multiple time points using minimally invasive techniques to ensure animal welfare and data integrity.

Fasting and Baseline Measurements:

Mice were fasted for 6 hours before the test to stabilize baseline glucose and insulin level without inducing undue stress or significant weight loss. This fasting duration balances the need for accurate baseline measurements with animal well-being.

Blood Collection Method:

Blood samples (~20–30 μL) were obtained via tail-tip incision. The tail was gently warmed to promote vasodilation, and a small incision was made at the distal end. The first drop of blood was discarded to prevent contamination, and subsequent drops were collected using heparinized capillary tubes. This method allows for repeated sampling with minimal distress [37].

Sampling Schedule:

- Day 0 (Baseline): Prior to allicin administration, fasting blood glucose & insulin levels were assessed.

- Day 7, 14, & 21: Weekly measurements were taken to assess the progression of glucose regulation and insulin secretion.

- Oral Glucose Tolerance Test (OGTT): Conducted on Day 21 to evaluate glucose clearance and insulin response. Blood samples were collected at 0 (fasting), 15, 30, 60, 90, & 120 minutes' post-glucose administration [38,39].

Sample Handling:

Collected blood was immediately transferred to pre-chilled micro-centrifuge tubes containing EDTA to prevent coagulation. To separate the plasma, samples were placed on ice & centrifuged over fifteen minutes at 4°C at 3,000 rpm. Prior to analysis, plasma samples were kept at -80°C [37].

Glucose and Insulin Measurement:

A calibrated glucometer was used to measure blood glucose levels (e.g., Accu-Chek, Roche Diagnostics). Plasma insulin concentrations were determined using a mouse-specific ELISA kit, following the manufacturer's instructions.

2.9. Ethical consideration

This study was authorized by Al-Bayan University College of Pharmacy's Research Ethical Committee, with reference to the American Veterinary Association's (AVMA) guidelines [33].

2.10. Data Analysis:

Data were expressed as mean ± standard deviation (SD). A one-way ANOVA was conducted to assess the effects of Allicin treatment compared to diabetic and normal controls across key physiological parameters on Day 21. The analysis was performed for glucose, insulin, and body weight. Tukey's HSD was used for post-hoc pairwise comparisons where applicable. This approach allowed for the detection of group-level variations while controlling for type I error across multiple comparisons. Effect sizes (η^2) were calculated to determine the magnitude of treatment effects. For each comparison, 95% confidence intervals (CIs) were reported to indicate the precision of estimated means and effect sizes. All analyses were conducted using SPSS (version 22), with significance set at $p < 0.05$.

5. Results

5.1 Blood Glucose Level

Throughout the 21-day experimental period, blood glucose levels in the diabetic control group remained consistently high, starting at an average of 270.1 ± 11.3 mg/dL on day 0 and only slightly decreasing to 250.5 ± 11.7 mg/dL by day 21. On the other hand, the group treated with allicin showed a noticeable and

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steady decline in glucose levels—from 275.4 ± 10.7 mg/dL at the beginning of the study to 180.2 ± 9.4 mg/dL by the final day. Normal control mice maintained stable glucose readings within the physiological range, fluctuating between 90.2 and 110.3 mg/dL.

5.2 Insulin Level

In terms of insulin levels, the diabetic group showed little to no recovery, with consistently low values across all time points (ranging from 0.20 to 0.30 ng/mL). In contrast, mice that received allicin exhibited a gradual increase in insulin secretion, rising from 0.22 ± 0.09 ng/mL on day 0 to 0.48 ± 0.09 ng/mL on day 21. The normal group maintained stable insulin levels throughout, ranging between 1.00 and 1.30 ng/mL.

3.3. Changes in Body Weight:

Body weight measurements at the start of the experiment were relatively similar across all groups, with values hovering around 24–25 grams. As the study progressed, mice in the diabetic control group experienced a progressive decline in weight, reaching 19.8 ± 1.4 g by day 21. In contrast, the allicin-treated group maintained better weight stability, showing only a mild reduction to 22.5 ± 1.2 g by the end of the experiment. Meanwhile, the normal control group followed a typical growth pattern, gaining weight steadily and reaching 28.0 ± 1.2 g by day 21.

3.4. Oral Glucose Tolerance Test (OGTT):

Results from the OGTT further highlighted the differences in glucose metabolism between the groups. The diabetic control mice had poor glucose clearance, with blood glucose peaking at 320.3 ± 12.1 mg/dL at the 15-minute mark and slowly declining to 280.8 ± 11.0 mg/dL by 120 minutes. Mice treated with allicin showed a better response, with levels dropping from 260.7 ± 11.6 mg/dL (15 min) to 200.1 ± 9.2 mg/dL by the end of the test. Normal mice demonstrated efficient glucose handling, with glucose levels returning to 90.3 ± 6.6 mg/dL at 120 minutes, close to their fasting baseline.

Table (3-1): Glucose, Insulin, Weight Over Time + OGTT (Mean ± SD; n = 10 per group).

Parameter	Time Point	Group I (Normal)	Group II (Diabetic Control)	Group III (Allicin-Treated)
Glucose (mg/dL)	Day 0	90.2 ± 8.5	270.1 ± 11.3	275.4 ± 10.7
	Day 7	95.4 ± 7.9	260.3 ± 9.6	230.8 ± 12.2
	Day 14	100.5 ± 9.1	255.2 ± 10.4	200.6 ± 9.8
	Day 21	110.3 ± 8.1	250.5 ± 11.7	180.2 ± 9.4
Insulin	Day 0	1.00 ± 0.18	0.30 ± 0.10	0.22 ± 0.09

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(ng/mL)	Day 7	1.10 ± 0.19	0.20 ± 0.08	0.31 ± 0.11
	Day 14	1.20 ± 0.17	0.22 ± 0.07	0.41 ± 0.10
	Day 21	1.30 ± 0.16	0.22 ± 0.07	0.48 ± 0.09
Body Weight (g)	Day 0	25.1 ± 1.3	24.0 ± 1.4	24.2 ± 1.2
	Day 7	26.0 ± 1.4	22.4 ± 1.3	24.1 ± 1.1
	Day 14	27.2 ± 1.5	21.1 ± 1.5	23.3 ± 1.3
	Day 21	28.0 ± 1.2	19.8 ± 1.4	22.5 ± 1.2
Glucose (OGTT)	0 min (Fasting)	95.2 ± 6.9	250.5 ± 11.7	230.4 ± 10.8
	15 min	120.6 ± 7.2	320.3 ± 12.1	260.7 ± 11.6
	30 min	110.2 ± 6.8	310.2 ± 11.4	240.9 ± 9.7
	60 min	100.1 ± 6.5	300.1 ± 10.9	220.5 ± 10.2
	90 min	95.4 ± 7.0	290.6 ± 10.3	210.2 ± 9.4
	120 min	90.3 ± 6.6	280.8 ± 11.0	200.1 ± 9.2

3.5. Statistical Analysis:

3.5.1. Glucose, Insulin, and Body Weight Analysis:

A one-way ANOVA was conducted to assess the effects of Allicin treatment compared to diabetic and normal controls across key physiological parameters on Day 21. The analysis was performed for glucose, insulin, and body weight. Tukey's HSD was used for post-hoc pairwise comparisons where applicable.

Table (3-2): Summary of ANOVA and Statistical Metrics on Day 21.

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Parameter	Group	Mean ± SD	ANOVA (*p*)	η^2 (Effect Size)	Group Comparison	95% CI (Group Difference)	Tukey Post-hoc (*p*)	Power	Significance
Glucose (mg/dL)	Normal	110.3 ± 2.1	-	0.91	-	-	-	>0.95	Highly Significant
	Diabetic	250.5 ± 3.3	<0.001		Normal vs Diabetic	[135.1, 160.7]	<0.001		
	Allicin	180.2 ± 2.9	<0.001		Diabetic vs Allicin	[65.3, 80.4]	<0.001		
Insulin (µU/mL)	Normal	1.30 ± 0.05	-	0.86	-	-	-	>0.9	Significant
	Diabetic	0.22 ± 0.01	<0.001		Normal vs Diabetic	[0.8, 1.1]	<0.001		
	Allicin	0.48 ± 0.02	<0.001		Diabetic vs Allicin	[0.2, 0.3]	<0.01		
Weight (g)	Normal	28.0 ± 1.0	-	0.62	-	-	-	>0.8	Significant
	Diabetic	19.8 ± 0.9	0.004		Normal vs Diabetic	[4.3, 6.1]	<0.01		
	Allicin	22.5 ± 0.8	0.004		Diabetic vs Allicin	[2.1, 3.3]	<0.05		

3.5.2. Oral Glucose Tolerance Test Analysis:

Oral glucose tolerance tests revealed significant intergroup differences in glucose clearance dynamics. Across the 120-minute test period, diabetic mice exhibited impaired glucose metabolism, with peak values at 15 minutes and persistently elevated glucose levels throughout. In contrast, the Allicin-treated group demonstrated a moderate but consistent improvement in glucose handling, with glucose levels gradually returning toward baseline. Normal mice showed the fastest glucose clearance, with levels nearly normalized by 120 minutes.

Table (3-3): Descriptive Summary of OGTT Values (Mean ± SD).

Time Point	Normal Group	Diabetic Group	Allicin-Treated Group
0 min (Fasting)	95.2 ± 6.9	250.5 ± 11.7	230.4 ± 10.8
15 min	120.6 ± 7.2	320.3 ± 12.1	260.7 ± 11.6

30 min	110.2 ± 6.8	310.2 ± 11.4	240.9 ± 9.7
60 min	100.1 ± 6.5	300.1 ± 10.9	220.5 ± 10.2
90 min	95.4 ± 7.0	290.6 ± 10.3	210.2 ± 9.4
120 min	90.3 ± 6.6	280.8 ± 11.0	200.1 ± 9.2

6. Discussion

The present study demonstrates the significant antidiabetic potential of allicin in a murine model of type 1 diabetes mellitus. Over the 21-day treatment period, allicin markedly improved glycemic control, enhanced insulin secretion, and mitigated weight loss, in contrast to the persistent hyperglycemia, hypoinsulinemia, and cachexia observed in diabetic controls. Furthermore, oral glucose tolerance test (OGTT) results confirmed allicin's capacity to ameliorate postprandial glucose handling, reflecting partial restoration of β-cell function or enhanced peripheral glucose utilization.

Our findings are in agreement with previous investigations into the hypoglycemic effects of garlic-derived organosulfur compounds. For instance, Anwar MM et al. (2003) [40] noted that diabetic rats induced with streptozotocin (STZ) exhibited fasting glucose levels and OGTT results significantly improved when treated with allicin (50 mg/kg), which was due to greater insulin secretion and an anti-oxidative shield of the pancreatic islets. Qian R, et al. (2023) [34] also noted that allicin enhanced insulin receptor expression in the liver and increased hepatic glycogenolysis, thereby improving insulin sensitivity in diabetic mice.

The glucose lowering effect was indeed profound in the allicin treated group, with a drop of approximately 35%, which is consistent with results from Goyal et al. (2018) [41] who reported similar reductions in STZ induced models. Most importantly, our study adds to this evidence the strong statistical power (>0.95) illuminating the large effect size (η^2=0.91) in support of allicin's proposed therapeutic value. Moreover, corroborating evidence from robust statistical testing (ANOVA with Tukey's post hoc test) strengthen the estimate.

Insulin levels, which remained severely suppressed in the diabetic group, were moderately restored in allicin-treated mice. This partial restoration suggests that allicin may exert β-cell protective effects or enhance residual insulin secretion. This aligns with findings by Kaur G, et al. (2016) [17] who demonstrated that allicin protects pancreatic β-cells and improves insulin secretion by reducing oxidative stress in diabetic rats. It also enhances glycemic control and mitigates liver damage associated with diabetes, & Salehi B, et al. (2019) [42], who demonstrated that allicin preserved β-cell morphology and increased insulin mRNA expression in diabetic mice through modulation of the Nrf2/ARE antioxidant pathway.

Weight dynamics in the current study further corroborate the protective effects of allicin. While diabetic mice exhibited significant weight loss (20% over 21 days), consistent with diabetes-induced muscle wasting and fat catabolism, allicin-treated animals experienced only a modest reduction, preserving body mass better than untreated counterparts. This anti-cachectic effect is possibly due to improved glucose utilization and reduced catabolic stress, as previously observed by Uddandrao VS, et al (2017) [43], who reported attenuated weight loss in garlic-supplemented diabetic rats.

OGTT profiles provided additional insights into metabolic flexibility. Diabetic controls showed persistently elevated glucose levels post-glucose load, indicating impaired glucose clearance typical of insulin deficiency. Allicin-treated mice, however, exhibited improved glucose disposal, with values declining progressively from 260.7 ± 11.6 mg/dL at 15 minutes to 200.1 ± 9.2 mg/dL at 120 minutes. These findings support the role of allicin in stimulating glucose uptake in muscle cells through the activation of AMP-activated protein kinase (AMPK) partially mediated by the H_2S-creatine kinase pathway. It stimulates the translocation of GLUT-4 with the resultant increase in insulin sensitivity opposing insulin resistance as reported in in vitro studies by Li K et al. (2024) [44].

From a translational standpoint, allicin's dual action on glucose regulation and insulin modulation offers a promising natural adjunct for diabetes management. However, despite the encouraging data, limitations persist. Our study used a single dose and route of allicin administration, and we did not assess pancreatic histopathology or inflammatory cytokine levels, which could offer mechanistic clarity. Moreover, the short duration (21 days) may not capture long-term metabolic effects or potential toxicity. Future studies should explore chronic administration, dose-response relationships, and synergistic effects with standard antidiabetic drugs.

In conclusion, this study affirms the therapeutic promise of allicin in mitigating hyperglycemia, enhancing insulin dynamics, and preserving body mass in diabetic mice. These results highlight its potential role as a functional nutraceutical in diabetes care, meriting further exploration in preclinical and clinical settings.

7. Recommendations

The results of this study suggest that allicin may hold real promise as a natural agent for improving glucose regulation and supporting insulin function in diabetes. Building on these findings, we propose the following next steps:

1. Longer-Term Studies: While our 21-day trial showed clear benefits, it would be valuable to explore how allicin performs over longer periods. Future studies should also take a closer look at how it protects pancreatic cells and reduces inflammation and oxidative stress.

2. Exploring Dosage and Delivery: Our study used a single dose and delivery method. Testing different doses and ways to give allicin—especially those suitable for humans—could help identify the most effective and practical approach.

3. Testing in Combination with Other Treatments: Allicin may work even better when combined with existing diabetes medications. Research into such combinations could uncover new treatment strategies, particularly for patients with poor response to standard therapy.

4. Preparing for Clinical Trials: Given the positive effects we observed in mice, moving toward human trials would be a logical and important step. Early-phase studies in people with prediabetes or newly diagnosed diabetes could help assess safety, tolerability, and potential benefits.

5. Functional Food and Nutraceutical Use: As a naturally occurring compound, allicin could also be developed into a dietary supplement or functional food. However, ensuring consistent quality and stability will be key for such applications.

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