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# THE ANTIDIABETIC EFFECT OF KONJAC GLUCOMANNAN ON GLUCOSE LEVEL DIABETIC RATS

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**Abstract**
**Objectives:** Type 2 Diabetes Mellitus (T2DM) describes a long-term endocrine disorder that disrupts different metabolic pathways defined by impaired insulin resistance as well as inadequate production of insulin, associated with abnormal sugar. Dietary prevention of T2DM has gained plenty of lately attention decades. As it turns out, the goal of this study is to explain the anti-diabetic activities of Konjac glucomannan (KGM) in diabetic rats.
**Methods:** This study's experimental design comprised of two groups of five male white rats each. The negative control group included of five diabetic rats. The positive control group was set out of diabetic rats created by glibenclamide at a dose of 0,7 ml intraperitoneal. Diabetic rats comprised the therapy group who were administered konjac glucomannan extract orally at a dose of 100 mg/kg b.w. as therapy. Prior to treatment, each subject's blood glucose level was measured using a glucometer by cutting the rat's tail and removing some of its blood. To confirm the results, glucose levels were checked again three and seven days after the participants had medication. Oneway analysis of variance (ANOVA) was used to analyze the data in SPSS.
**Results:** The study's findings revealed that diabetic rats treated with konjac glucomannan extract had an effect similar to diabetic rats produced by glibenclamide, as indicated by a decrease in blood glucose levels, as evidenced by diabetes treatment therapy in general.
**Discussion:** KGM reduced gluconeogenesis, liver marker enzymes, elevated genes, and insulin pathway protein expression which is distinguished by a drop in glucose levels.
**Conclusion:** According to the most recent research, dietary KGM may be more beneficial for your health when treating T2DM.
**Key words:** *Konjac glucomannan, Type 2 Diabetes Mellitus, Glucose Levels, Scizophrenia, Rats*

## Background

Type 2 diabetes mellitus (T2DM) is a potentially lethal chemical illness caused by elevated levels of insulin, which leads to an inability of pancreatic (b) cell insulin absorption and output. T2DM is a medical condition that is defined through impaired expression of insulin and inadequate tolerance to insulin, that is linked to an abnormal sugar, lipid, amino acid, water, and electrolyte metabolism (Griffin 2022; Yoshida et al. 2020). With the type 2 diabetes and impaired glucose tolerance (IGT) are common increasing in every country since 1980, avoiding further growth of

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such populations has become a major priority issue (NCD Risk Factor Collaboration (NCD-RisC) 2016). According to the International Diabetes Federation's most recent report, diabetes diagnoses have increased at a staggering pace, reaching 108 million cases in 1980 to a staggering 463 million in 2019, with the total expected to soar to 700 million by 2045 (Uusitupa et al. 2019; Sirdah and Reading 2020). Because of its increasing morbidity and mortality, type 2 diabetes mellitus is not simply an emergency medical situation, but also a severe social challenge (Z. Zhang et al. 2023; Polonsky and Henry 2016). The therapy of type 2 diabetes relies on the control of Insulin deficiency and insulin biological processes. Notably, the KGM enhances response to insulin through hormone receptor regulation, resulting in beneficial upregulation of genes and proteins involved in the insulin signaling cascade (Shimizu et al. 2022; Khan et al. 2019; D. Zhu et al. 2019).

T2DM is managed by medications that control heart rate, lipids, and circulating glucose levels (glucosidase inhibitors and thiazolidinediones for exampleand) (Padhi, Nayak, and Behera 2020; Zhou et al. 2016). Although contemporary T2DM treatments, such as metformin and glibenclamide, demonstrate a considerable reduction in glucose levels in persons with T2DM in a short period of moments (Gomes et al. 2019; Asad et al. 2015; Shakuri Yasin et al. 2022). Patients who have a for a long time medicinal effect solution these are man-made medications may encounter negative effects such as endocrine abnormalities (Jayachandran et al. 2019; Mukhtar, Galalain, and Yunusa 2020). Long-term medication use, on the other hand, can cause bloating and diarrhea, and the majority of people with T2DM have to take insulin injections to keep up regular glucose levels. (Suzuki et al. 2015; Lee and Yoon 2021; DeFronzo et al. 2015).

Several dietary polysaccharides had been demonstrated to help regulate glucose levels, including those found in oatmeal, spores, coffee, fruits, and pumpkins (Ganesan and Xu 2019; Abuajah, Ogbonna, and Osuji 2015). Furthermore, water-soluble stiff particles are demonstrated to increase the fluidity of food that is eaten in the stomach while decreasing glucose levels (McRorie and McKeown 2017; Abutair, Naser, and Hamed 2016). In this case, KGM, a liquid in water nutritional carbohydrate originated from root of Amorphophallus konjac, possess ability to decrease glucose (Behera and Ray 2016; Yixin Wang et al. 2018). The mass of a substance ranges between 500 and 2000 kDa, according to different methods of extraction and providers (F. Zhu 2018; Ying Zhang et al. 2018). KGM has only one component and is therefore less harmful to the body of individuals compared to other nutritional fibers (cereal fiber and vegetable fiber). Other diets (such as beans, flour, and maize) have significantly less fiber per 100 g versus KGM (Fang et al. 2023; Adams et al. 2020). The most essential aspect is that it can affect blood lipids, gut flora, oxidative stress, immunological suppression, and other factors in addition to blood sugar (Gibney et al. 2017; Deng et al. 2020).

KGM has been found in animal studies that’s an excellent dietary choice for the treatment of T2DM by reducing glucose levels via the insulin route (Gamboa-Gómez et al. 2020; X. Li, Jayachandran, and Xu 2021). Given this, we predicted that the KGM could influence T2DM through chemical processes that proven on hypoglycemic activity. The justification for deciding on KGM as an anti-diabetic option for additional evaluation through molecular investigations is

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antihyperglycemic. To explain our hypothesis, we created an experimental model of diabetic rats using alloxan (Ighodaro, Adeosun, and Akinloye 2017) that was induced into the bodies. Furthermore, KGM was given to diabetic rats to investigate the specific molecules and traits involved in transmitting insulin system in order to comprehend a molecular basis KGM's anti-diabetic activity through its control of metabolic glucose-insulin sensitivity. The goal of this study is to demonstrate that KGM can be anti-diabetic via the insulin route, which may be demonstrated by lowering blood glucose levels.

## Materials and Methods

### *Chemical Specifications and Study Design*

PT. Raja Porang Indonesia supplied the konjac glucomannan (KGM). KGM has an approximate molecular weight of 1000-2000 kDa and a 1:1.6 proportion of -1-4 linked Dglucosyl and D-mannosyl acids in its primary chain. This study employed a true experimental design, with white male rats serving as experimental animals and subject groups chosen at random. Glucose levels were assessed before the rats were given therapy using a glucometer by cutting the rats' tails to take blood samples. To assess the treatment's efficacy, blood glucose levels were measured again on the third and seventh days using the same procedure.

### *Animal conditions and acclimatization*

This study employed fifteen male white rats with body weights ranging from 230 to 330 grams and normal glycemic circumstances. The breeding was done in polypropylene cages without pathogens at the Pharmacology Laboratory. We use the principles of replacement, reduction, and refinement (3R) in connection with the studies that researchers have conducted using experimental animals. Also included is the notion of five freedoms for animal welfare, which includes (1) release over dehydration and starvation, (2) equality of anxiety, (3) equality to suffering, harm, and disease, (4) release against fretful and anguish, and (5) flexibility in distress and dread to exhibit natural behavior. The application of animal welfare concepts in lab research was claimed to reduce stress levels in lab animals and to offer more accurate study results (Mutiarahmi, Hartady, and Lesmana 2021; Mellor 2016). The Health study Ethics Committee of the STRADA Indonesia Institute of Health Sciences granted us study authorization with No. 3849/KEPK/V1/2023. Rats are free to drink and eat at any time. The entire study followed animal use protocols and standards approved by the National Institutes of Health. The rats were held for a two-week acclimatization period before being administered medication.

### *Type 2 diabetes mellitus induction*

In rats, alloxan is used to develop type 2 diabetic mellitus (T2DM). Alloxan induces diabetes by a process that involves hepatic intestinal beta (b) cells are partially degraded and a consequent decline in glucose concentration and quality generated by these cells (Ighodaro, Adeosun, and Akinloye 2017; Mabhida et al. 2018). Dunn and McLetchie, who successfully inflicted diabetes on experimental rabbits in their study, were the first to describe its usage as a diabetogenic

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medication in animals (Dunn and Mcletchie 1943). Following the adaption period, rats were given 100 mg/kg b.w. of alloxan intraperitoneally. The next day, blood glucose levels are projected to climb. Throughout the experiment, the diabetic ratswere provided food and drink.

***Groups for the experiment***

The study employed 15 male white rats separated into three distinct categories: a negative control group, a positive control group, and an experimental group, with rats in each group picked at random. Each group comprised 5 male white rats as experimental animals. KGM was dispersed in water and given to rats orally through a tube inserted into the stomach. After T2DM was confirmed, KGM treatment was started and continued for 7 days. Based on earlier research (X. Li, Jayachandran, and Xu 2021), the optimum dosage of KGM (100 mg/kg bw) was determined, and this study was designed in accordance with prior similar studies (Zhao, Jayachandran, and Xu 2020).

- Group I: Alloxan (100 mg/kg b.w.) as negative control group.
- Group II: Alloxan (100 mg/kg b.w.) + Glibenclamide (0.7 ml) as positive control group.
- Group III: Alloxan (100 mg/kg b.w.) + 100 mg/kg b.w. of KGM as treatment group.

***The dissection of animals and preserving samples***

Animals were kept fasting overnight at the end of the experiment. The rats were anesthetized with ketamine hydrochloride (24 mg/kg b.w.) through intramuscular injection before being slaughtered by cervical decapitation. Blood samples were taken in heparinized tubes for serum glucose and other tests.

***Statistical analysis***

The general information set's mean standard deviation (SD) proved displayed. Oneway analysis of variance (ANOVA) was used to analyze the data in SPSS.

**Results**

KGM is made from konjac, which cannot be processed by human gastrointestinal enzymes but may pass through the gastrointestinal tract and be used by abdominal microorganisms. To improve human body sub-health, KGM can also cut calories, regulate physiological sodium chloride, and manage diseases such as diabetes and obesity. (Tester and Al-Ghazzewi 2016; Devaraj, Reddy, and Xu 2019). KGM has been demonstrated in studies to promote subjective fullness and decrease hunger, that constitutes highly advantageous for individuals with T2DM (Furihata et al. 2022; X. Wu et al. 2018). KGM treatment enhanced cellular activity blood sugar levels was improved, glucose-dependent while the synthesis of enzymes were controlled, glycogen that was retained in the liver was reduced, and liver enzymes were restored (Bełtowski, Wójcicka, and Jamroz-Wiśniewska 2018; Fang et al. 2023). Insulin binding initiates the transmission of insulin signals via the outside of the cell alpha component of the circulating insulin receptor (IR) (Nielsen et al. 2022; Uchikawa et al. 2019). These impacts on glucose metabolism have an impact on the process

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of glycolysis glucose production, transfer of glycogen and glucose to the cells formation (G. Wu et al. 2020; Yue Zhang et al. 2019). That was followed by intracellular component of the beta subunit, the remaining components of tyrosine, auto-phosphorylates and recruits insulin receptor substrates (IRS) (Fang et al. 2023; Hall, Yu, and Choi 2020). Once phosphorylated, IRS is prepared to send impulses and activate a variety of insulin-dependent signaling pathways that regulate physiological processes such as protein and lipid metabolism, as well as cell growth and survivability (Bettedi et al. 2020; Haeusler, McGraw, and Accili 2018). The insulin signaling cascade depends on the protein kinase B/phosphatidylinositol 3-kinase (Akt/PI3K) pathway (Gu et al. 2019; Marquard and Jücker 2020). The many processes that occur in this pathway include PI3K stimulation, phosphorylation of 4,5-bisphosphate (PIP2) conversion to the phosphorylation 3,4,5-trisphosphate (PIP3), Akt kinase and the glucose transporter type 4 (GLUT 4) trafficking to the membrane of the cell (Świderska et al. 2018; Shimizu et al. 2022).

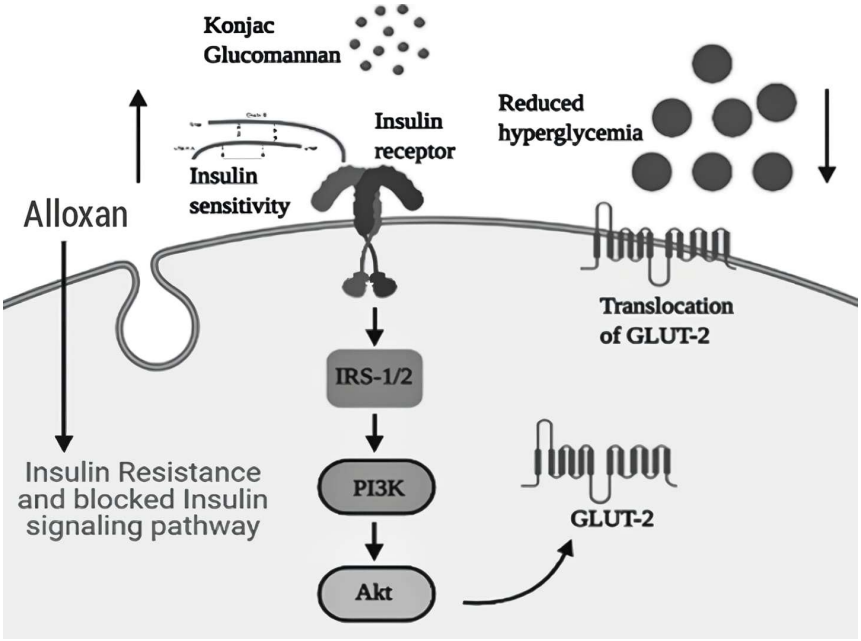


***Figure 1.*** The mode of action of konjac glucomannan as an anti-diabetic is via the insulin pathway, with the consequence of lowering blood glucose levels. *(X. Li, Jayachandran, and Xu 2021)*

In diabetics, glucose absorption, terms usage, archiving, regrowth, and oxidation are all used interchangeably critical in order to keep the balance of glucose. Individuals with T2DM have a disruption in the stability of glucose (Guo et al. 2021; Galicia-Garcia et al. 2020). Insulin is the most significant factor in blood sugar regulation. As a result, poor signal transmission will cause blood sugar levels to fluctuate (Al-Ghazzewi and Tester 2012; Świderska et al. 2018). It was discovered that diabetic rats had reduced insulin signaling genes and proteins, and KGM therapy may increase gene expression and glucose pathway activity, which leads to stabilized insulin output and reduced circulating glucose levels (Fang et al. 2023). Furthermore, Phosphatidylinositol

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3-kinase (PI3K) and the receptor for insulin component 1 (IRS1) are important mediators of insulin metabolism. In diabetics, decreased IRS1 activity can result in mutations in the tyrosine kinase hinsr gene, disrupting insulin signaling. KGM can drastically lower blood sugar levels in rats, restore normal pancreatic β-cell proliferation, and boost IRS1 and PI3K expression levels (X. Li, Jayachandran, and Xu 2021; N. Li et al. 2020). KGM has been found in animal studies to treat diabetes-related cardiomyopathy, correct gastrointestinal arrangement in rats, and reduce plasma urinary acids, creatinine-J, bladder abnormal plasma, circulating glucose, a substance called ketone the organism, and protein concentrations are all measured (Gamboa-Gómez et al. 2020; Yanli Wang et al. 2021). The liver's ability to store glycogen is a key sign of T2DM. KGM may stimulate glycogen synthesis in the liver, lowering glucose levels and slowing diabetes's development, which is favorable to liver defensiveness (Fang et al. 2023; Dewidar et al. 2020). The concentrations of Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are enzyme levels in the blood levels indicate the extent of hepatocellular damage, while serum alkaline phosphatase (ALP) activity indicates the severity of cholestasis. An increased ALT level is linked to T2DM, producing aberrant leucine, lysine, and glutamic acid metabolism (Spanakis et al. 2020; X. Li, Jayachandran, and Xu 2021). **Figure 2.** depicts the process by which konjac glucomannan can be utilized as an antidiabetic via the insulin pathway, as evidenced in its effect on lowering blood glucose levels, according to research conducted by (X. Li, Jayachandran, and Xu 2021)

**ANOVA - Glucose Levels**

| Cases | Sum of Squares | df | Mean Square | F | p | VS-MPR* | $\eta^2$ |
|---|---|---|---|---|---|---|---|
| Treatment | 10056.133 | 2 | 5028.067 | 190.217 | < .001 | $2.151\times10^{+7}$ | 0.969 |
| Residuals | 317.200 | 12 | 26.433 | | | | |

***Table 1.*** The p value of < 0.001 indicates that the therapy group with KGM has the same antidiabetic effect as therapy with glibenclamide, according to the table of analysis test findings using One Way ANOVA.

**Post Hoc Comparisons - Treatment**

| | | Mean Difference | SE | t | $p_{tukey}$ |
|---|---|---|---|---|---|
| KGM | Negative Control | -47.800 | 3.252 | -14.700 | < .001 |
| | Positive Control | 12.200 | 3.252 | 3.752 | 0.007 |
| Negative Control | Positive Control | 60.000 | 3.252 | 18.452 | < .001 |

***Table 2.*** Depending on the third treatment comparison results table, therapy using KGM nearly produces significant results when compared to therapy using glibenclamide.

Phosphorylation is an important step in the activation of proteins which indicate insulin, which leads in the movement through glucose receptors (Saltiel 2021; Haeusler, McGraw, and Accili 2018). The expression of proteins in laboratory animals. Alloxan was given to rats, which damaged insulin receptors and reduced the expression of IR-, p-IRS-1, p-Akt, and GLUT-2 in the liver. The

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insulin signaling pathway is made up of many proteins that are produced by different genes and play significant roles in blood homeostasis and other functions. mRNA expression of IR, IRS-1, IRS-2, Akt, PI3K, GK, and GLUT-2 in positive control and experimental animals (Saltiel 2021; X. Li, Jayachandran, and Xu 2021). Treatment with KGM at a dose of 100 mg/kg b.w. significantly increased ($\rho < 0.01$) the expression of insulin signaling genes in the diabetic group and It can be confirmed or shown from blood glucose test results on the third and seventh days after diabetic rats were given therapy **Table 3.** The findings are comparable to Glibenclamid-treated rats **Table 4.** In accordance with a study carried out using rats with hepatic damage generated through administering a subcutaneous injection of CCl4 as an experiment, management of KGM might down-regulate the unusually elevated levels of ALT and AST in the bloodstream, regulate more strongly the A/G ratio in plasma, enhance the disproportionately low superoxide dismutase, also known as SOD, fulfilled, and minimize the significantly elevated its concentration substance. This reveals KGM's preventive action against $CCl_4$-induced liver damage in rats (Tong, Chi, and Zhang 2018).

KGM has a significant impact on carbohydrates along with gluconeogenic organisms' functioning as enzymes in glucose metabolism (M. Chen et al. 2017; X. Li, Jayachandran, and Xu 2021). Hexokinase, a glycolysis rate-limiting enzyme, regulates glucose absorption and energy production by turning glucose into glucose 6-phosphate (Jiang et al. 2022; Dewidar et al. 2020). While glucose levels in the blood decrease under regular, Glucose acts as a the production of glucose engine in the liver, which is responsible for producing glucose through sugar-6-phosphatase (H. Chen et al. 2019; Jiang et al. 2022). Scientists discovered that in diabetic rats, hexokinase, glucose-6-phosphate dehydrogenase, and glycogen levels were dramatically reduced ($\rho < 0.05$), whereas gluconeogenesis (glucose-6phosphatase) was substantially elevated (X. Li, Jayachandran, and Xu 2021; Fang et al. 2023). In diabetic rats administered 80 mg/kg of KGM, the levels of a enzyme called the enzyme glucose-6-phosphate dehydrogenase, and triglycerides rose, whereas gluconeogenesis activities were lowered (Iluz-Freundlich et al. 2020). The magnitude of gluconeogenic enzymes was dramatically lowered, contributing to the notion that KGM therapy might boost the activity of glucose metabolism enzymes to improve insulin sensitivity, as indicated by a decrease in blood glucose levels after KGM therapy delivery (Giuntini, Sardá, and de Menezes 2022; Yanli Wang et al. 2021).

**Descriptives - Glucose Levels**

| Treatment | N | Mean | SD | SE | Coefficient of variation |
|---|---|---|---|---|---|
| KGM | 5 | 74.800 | 3.421 | 1.530 | 0.046 |
| Negative Control | 5 | 122.600 | 6.348 | 2.839 | 0.052 |
| Positive Control | 5 | 62.600 | 5.225 | 2.337 | 0.083 |

***Table 5.*** According to the results of the descriptive analysis, KGM therapy has been shown to have effects that are nearly identical to those of glibenclamide therapy.

**Descriptives plots**

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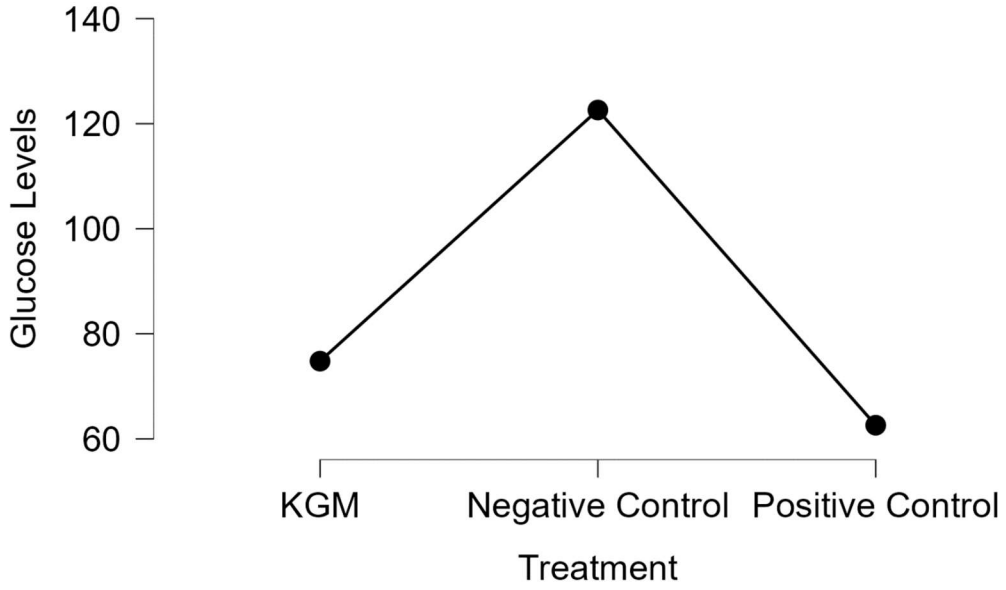


***Table 6.*** Table describing the plot that shows that the group treated with KGM can lower blood sugar levels in diabetic rats, while this outcome is equivalent to the group that received glibenclamide therapy in the positive group.

Blood glucose levels are measured to determine how much glucose is in the blood, and high glucose levels are a sign of Diabetes Mellitus (Nagy and Einwallner 2018; Moreira-Lucas et al. 2017). As a result, it becomes an important component in determining the level of diabetes mellitus. Serum glucose levels in control and experimental animals are displayed. After being induced by alloxan, diabetic rats showed an increase that did not significantly reduce. However, when rats were given KGM, they showed a large drop on day 3 and a rapid increase on day 7. Glibenclamide treatment produced similar outcomes in rats. Glucose levels in diabetic rats were tested using a glucometer, which was obtained by cutting off the rats tails and drawing blood. **Table 7.** shows that the KGM administered to diabetic rats was shown to lower glucose levels on the third and seventh days after the diabetic rats were treated. The reduction in glucose levels following KGM therapy was comparable to the risk of diabetes after glibenclamide medication that shows in **Table 8.** As a consequence, KGM therapy can be demonstrated to be successful as an antidiabetic.

**Discussion**

Type 2 diabetes mellitus (T2DM) morbidity and mortality pose a serious worldwide health problem (Atlas 2019; Milardi et al. 2021). Diabetes type 2 is significantly associated to insulin insensitivity, being overweight, and β-cells that malfunction are all symptoms of insulin resistance (Fan et al. 2020; Haeusler, McGraw, and Accili 2018). As a result, we used a prototype animal that may imitate individuals T2DM. We used alloxan injection to induce T2DM in male white rats. Alloxan has been linked to insulin resistance, glucose intolerance, and increased body fat mass (Khedher et al. 2018; Saltiel 2021). Alloxan was utilized to cause beta-cell failure after a state of

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insulin resistance (Tamura et al. 2015; Ighodaro, Adeosun, and Akinloye 2017). Because of the sequence of pathological events such as insulin resistance, obesity, and β-cell failure, it is a perfect model of T2DM rats. T2DM treatment regimens vary, but some have severe adverse effects that can lead to a variety of issues (Gomes et al. 2019; Fan et al. 2020). As a consequence, there should be a pressing requirement to create improved therapy programs that have no side effects. The consumption of fruits and vegetables and nutritional fiber aid in avoiding the development of type 2 diabetes by lowering the production of insulin and optimizing circulating glucose tolerance and insulin concentrations (Jayachandran et al. 2019; C. Chen et al. 2016). Cohort studies of dietary fiber intake suggest that it may lower the risk of T2DM in both sexes (C. Chen et al. 2016). KGM is a potential dietary fiber with a variety of health benefits. As a result, the purpose of this study was to explain the efficacy of KGM treatment in T2DM by modulating the insulin signaling system, as evidenced by a drop in blood glucose levels (Devaraj, Reddy, and Xu 2019; Barber et al. 2020). The KGM dosage was calculated using earlier (X. Li, Jayachandran, and Xu 2021) studies. We studied many basic parameters in the previously stated dose dependency of KGM (40, 80, 120 mg/kg b.w.) study and the researchers picked on an effective dose of KGM of 100 mg/kg b.w. as the therapeutic dose for diabetic rats.

The initial test was done to determine glucose levels in research rats before they were given alloxan to make them diabetic. Our findings reveal that diabetic rats who were not given KGM therapy had a significant increase in glucose levels on day 3, reaching a peak on day 7. In contrast, when glucose levels were assessed at the same time, rats treated with KGM at a dose of 100 mg/kg b.w. showed a drop in glucose levels throughout the same time period. Furthermore, when compared to diabetic rats, the alternate range of glucose levels in KGM-treated rats implies that KGM aids in glucose normalization. In diabetic rats treated with the conventional medication Glibencalimde, similar findings were obtained **Table 9.** In addition, our findings are compatible with those of (X. Li, Jayachandran, and Xu 2021) and (Gamboa-Gómez et al. 2020), who discovered glucose regulation in HFD/STZ-induced T2DM rats. The cell's perception of intercellular glucose is diminished as a result of Alloxan-mediated β-cell dysfunction, causing disruption of normal glucose metabolism and severe reduction of endogenous glycogen production (Ighodaro, Adeosun, and Akinloye 2017; Mezza et al. 2019). **Table 10.** shows that diabetic rats have lower glucose levels than negative control rats. KGM treatment considerably ($\rho < 0.01$) lowered glucose levels. The results were comparable to those obtained in diabetic rats treated with Glibenclamide, demonstrating that KGM has the extraordinary capacity to boost store glycogen production in hepatocytes through modulating insulin secretion (Bełtowski, Wójcicka, and Jamroz-Wiśniewska 2018; Z. Zhang et al. 2023). Glycogen-specific staining yielded comparable results to add additional robustness to the biochemical results (X. Li, Jayachandran, and Xu 2021). Due to a lack of sufficient equipment, no biochemical assays were performed in this investigation to substantiate this conclusion.

The findings show that KGM can lower glucose levels by boosting insulin sensitivity to its receptors and modulating the Glut-2 translocation pathway, which allows glucose to enter cells. The therapeutic effect of KGM in our investigation is similar to (X. Li, Jayachandran, and Xu

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2021)'s antidiabetic study, which may function by regulating the insulin signaling pathway to demonstrate the amplification of the therapeutic effect of T2DM. Nevertheless to the limitations of the researchers and the field that the researchers are focused on, researchers have not studied the theory that discusses the insulin pathway in detail, as shown in **Figure 3.**, in the research that has been carried out. As a result, it is hoped that additional researchers will thoroughly investigate the notion that KGM can lower glucose via the insulin pathway.

## Conclusions

Our hypothesis was that we would be able to successfully create an Alloxan-induced T2DM rats model and treat diabetic rats with KGM. Our findings suggest that KGM is a promising treatment for type 2 diabetes. In a nutshell, KGM therapy lowered blood glucose levels. Therefore, our findings suggest that KGM at a dose of 100 mg/kg b.w. has an anti-diabetic impact by modulating the insulin signaling system, as indicated by a drop in blood glucose levels. More investigation into its efficacy across numerous routes, as well as extensive theoretical explanations of correlations, could lead to more in-depth mechanistic insights concerning KGM's antidiabetic qualities.

## Recommendatıon

This Research is recommended as a complementer therapy that should be applied to skizophrenia with high level glucose level.

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## Conflıcts of Interest

The author declare they have no conflicts of interest

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