GSAR Journal of Applied Medical Sciences ISSN: 2584-2323 (Online)

GSAR Journal of Applied Medical Sciences
ISSN: 2584-2323 (Online)
Frequency: Monthly
Published By GSAR Publishers
Journal Homepage Link- https://gsarpublishers.com/gsarjams-home/

DIET:THE OVERLOOKED KEY IN THE EARLY PREVENTION OF DIABETES

By

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Article History

Received: 15/04/2025
Accepted: 21/04/2025
Published: 23/04/2025

Vol – 2 Issue – 4

PP: -19-26

Abstract

Over time, shifts in dietary patterns influence health and create conditions conducive to various diseases, each of which has an early stage before full development. This study aimed to reverse pre-diabetes to prevent its progression into diabetes. Twenty volunteers from the POLAC community were screened for overnight fasting glycaemia on four occasions using Accu Isaw, with a threshold set at 100–125 mg/dL. Six participants (A, E, I, J, O, and T) who exhibited moderate hyperglycaemia at least twice on separate days were selected. The research was in two stages. Stage 1 measured postprandial glucose and cholesterol levels after fasting. Stage 2 involved mindful feeding, where participants consumed minimally processed foods in controlled portions over two weeks, with meals scheduled in the morning and evening at specified times while avoiding night-time eating. Water intake was unrestricted, and glucose and cholesterol levels were under fasting conditions. The glucose concentration indicated levels peaked at the third interval, with two-hour postprandial concentrations remaining below 200 mg/dL. Total cholesterol peaked at the first-hour mark in two-thirds of participants. The mean glucose levels on Days 8 (97 mg/dL), 10 (86 mg/dL), 12 (91 mg/dL), and 14 (84 mg/dL) all fell below 100 mg/dL. By Days 12 and 14, pre-diabetes reversed in all participants. Body Mass Index decreased over time, highlighting weight reduction as a key factor in the reversal process. There was a significant difference between the baseline mean glucose levels and those recorded from Days 6 through 14. Pre-diabetes did not progress to diabetes.

Keywords: Diet, Diabetes, Glycaemia, Pre-diabetes, and Total cholesterolaemia,

Introduction

Food plays a role in shaping health and, above all, the diabetic prevention and treatment context. Making thoughtful dietary choices enhances protection against type 2 diabetes and mitigates its potential effects on health (Oh and Jun, 2014). In this way, food acts as a guardian, shielding the body from the risks associated with health crises. Food is transformative and causes systemic change within health (‘GDB 2017 Diet Collaborators, 2019’ *and* Fanzo *et al.*, 2022). Through the integration of evidence-based dietary interventions, a radical shift from a reactive model disease treatment that focuses on symptoms to a proactive model centred on treating for prevention and reversing diseases such as chronic ones (with a marked preference for Diabetes type 2) is achievable and each chronic disease has a developmental stage and any of the stage can be halted (Fig. 1, 2 and 3).

Diabetes is a chronic condition (*Arroyave et al., 2022*) that worsens with time and, therefore, requires vigilant care through functional therapeutic design to achieve the goal (reversion). The treatment, being more focused on insulin (Cernea and Raz, 2020), has many obstacles challenging its use and doubts the possibility of reversing the condition (Kovatchev, 2020). The agreement among physicians, biologists, and chemists on regenerative medicine, which involves β-cells from pancreatic islets as an alternative approach (Ellis et al., 2017), may include food as a component.

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Food is a power-driving force behind wellness in all its forms. Nutritious food is part of the social determinant of health (Wilensky, 2016). Aside from food providing nutrients essential to the body, it is relevant to disease prevention and treatment (*Downer et al., 2020*). Nutrient-disease relationship connection is not new in biomedicine (Carpenter, 2012), and the lack of needed attention makes it an unsung hero. Regrettably, the hard part is to change from an already-made perception. If food is medicine, then there is a need to reconsider this unsung hero for the reversion of pre-diabetes, diabetes, and others. Food is an expensive commodity, just as diabetes is a costly disease (*Powers et al., 2020*). With Nigeria facing escalating inflation and food shortages, the necessity for immediate and thorough interventions has become more pressing than ever. This situation double-binds Nigeria. Choosing food over medication therapy is a lesser evil to go by.

A healthy diet has multifaceted benefits- protecting, preventing disease, and boosting mood. Paradoxically, an unhealthy diet can fuel the opposite. Food is an essential factor in several conditions in the network of biological dysfunction. The research considers how food impacts health and function in chronic disease development, with diabetes in view, and advocates for eating for optimal health. On this, the researcher prioritized healthy and mindful portion food sizes to halt the metabolic consequences following the degenerative condition associated with diabetes (Fig. 2) even before the disease establishment. Diabetes affects virtually all cells (Fig. 2), and all cells require nourishment. A matchable antidote should be food, hence the choice of food therapy. This disorder (diabetes) requires complex daily management activities and decisions (*Powers et al., 2020*) to improve outcomes (American Diabetes Association Professional Practice Committee, 2022). It justifies monitoring cholesterol and sugar levels while on a proper diet.

Diabetes type 2 is the main gateway to cardiovascular diseases (De Rosa *et al*., 2018), and cardiovascular disease causes the most deaths globally (Carreras *et al*., 2019 and Patel *et al.,* 2018). Total cholesterol is a necessary cardiovascular risk factor (Khil, *et al*., 2023). Even in a healthy population, hypercholesterolaemia is a risk factor for cardiovascular diseases (Peters *et al.,* 2016), worst off in diabetes. Conventional drugs (statins) prescribed to lower cholesterol can increase the danger of developing diabetes (Perego *et al.,* 2019). While dieting is beneficial, unhealthy eating predisposes to the opposite of expectations. The study examined dietary strategies for preventing diabetes in individuals with moderate hyperglycaemia.

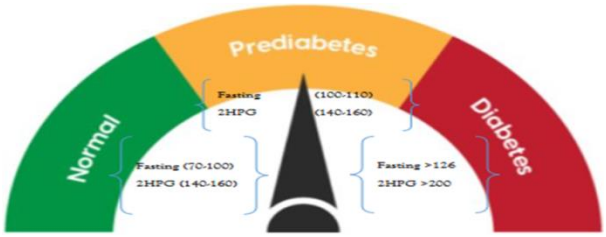

Fig 1: Glucose concentrations (mg/dl) and at respective stages of Diabetes development (Hammer *et al.,* 2019 and Khan *et al*., 2020).

Fig. 2: Degenerative changes associated with diabetes complications in organs. Adopted with modification from Deshpande *et al.* (2008), Aliseda *et al*. (2008), Nagib et al. (2019) and Dohrn et al. (2020), Biessels (2007), Papadopoulou and Al-Thakafi and Al-Hathal (2016).

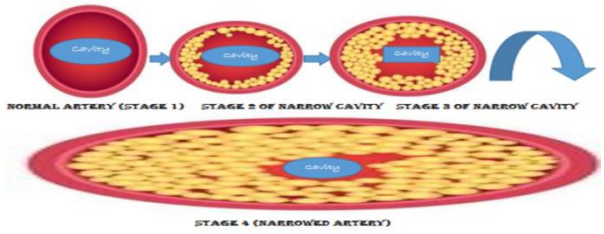

Fig 3: Clogging of artery in hypercholesteraemia Stanley (2019).

Materials and Methods

Materials- the Accu Isaw CE0197 strips and equipment – Accu Isaw, electronic balance, and standiometer were from the accredited distributor at Chriscare Medical Devices Nigeria Limited. Foodstuff, pot, and cooking gas were from the local market.

Volunteers' recruitment

The POLAC community received individualized discussions about diabetes to clarify the research aim and scope before enrolling participants. These conversations covered the stages of diabetes development and the risks associated with hyperglycaemia before it progresses to diabetes. Volunteers who agreed to participate underwent fasting glucose screening in the morning after fasting for 10 to 12 hours. A plasma fasting glucose concentration of 100–125 mg/dL on two separate occasions, along with self-reported early signs and symptoms of diabetes, was the criterion for inclusion in the study. For confidentiality, names were in alphabetical codes (A, B, C, D, E, F, G, H, I, J, K, L, M, N, O, P, Q, R, S, and T). The results served as a preliminary screening of 20 participants, with six meeting the research criteria: A, E, I, J, O, and T.

Experimental design

The research approach is in two stages (pre-prandial and postprandial as stage 1 and feeding food portion size control at specified meal times as stage 2): The pre-prandial and postprandial total cholesterol and postprandial glucose

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concentration determinations, were after a ten-hour fast. The feeding ratio was individual routine feeding behaviour and was self-reported. Determinations were total cholesterol concentration at one-hour intervals of two and glucose concentration at 30-minute intervals of four. The pre-prandial measurement in each participant was considered the baseline concentration for cholesterol. The baseline glucose was from the preliminary testing of the mean fasting glucose for each participant (6). Feeding ratio and food portion size control: The feeding ratio was among a broad list of minimally processed foods from the routine fed by individual participants (food was according to personal choices). The participant was tested on different food portions to ascertain commendation quantities and acclimatized to it. A food portion size of 380±120g was selected and distributed over two weeks as morning and evening meals. The feedings between 8 to 11 am are for morning meals, and the evening meals run between 4- 7 pm. There was no night food. Water was drunk liberally, adopted from Nwachukwu (2025).

The total cholesterol and glucose concentration was measured in the morning under fasted conditions via capillary pricking. The capillary venous blood concentration was in the morning after about 10 hours of overnight fasting. The glucose determinations were at two-day alternate intervals, and cholesterol concentration was determined weekly. This stage lasted for two weeks. The participant's height was determined twice, before and after the commencement of this stage. These were used in body mass index calculation and are called BMI.

Exclusion and inclusion criteria

Those four years and above in POLAC plus no evidence of health complications due to diabetes and others included. Those having health complications and whose hyperglycemia was more than 125 mg/dl on three or more occasions form the exclusion list.

Ethical consideration

The potential participants freely made their own decisions on whether they would participate or continue participating in research. Through the informed consent procedure- they understood the information and how it related to their clinical situation, were accurately informed of the purpose, methods, risks, benefits, and alternatives to the research, and showed interest. They made voluntary decisions about participation.

Results

Tables 1 to 4 and Figures 4 to 6 present the results. Table 1 displays preliminary fasting glycaemia with a threshold set at 125. Table 2 shows the intervals of changes in glycaemia and cholesterolaemia trajectories over a two-hour postprandial period, while Table 3 illustrates the alternate changes in glycaemia over weeks.

As shown in Table 1, six participants (A, E, I, J, O, and T) and a participant (M) consistently exhibited moderate hyperglycaemia and normal glycaemia thrice on four occasions at glucose concentration in mg/dl (exceeding 100 but less than126 for moderate hyperglycaemia and not exceeding 100 for normal glycaemia). Additionally, six participants (B, D, G, H, K, N, and S) experienced hyperglycaemia four times with glucose levels exceeding 125.

As shown in Figures 4 and 5, the postprandial effects on total cholesterol and glucose levels over two hours resulted in increased concentrations from baseline in all participants. Regarding glycaemia, glucose concentrations increased in a time-dependent manner for 90 minutes before declining at 120 minutes in all participants (Fig. 4 and Table 2). The highest glycaemic concentration changes at 90 minutes in participants- A (68: 55%), E (100: 83%), I (96: 86%), J (61: 96%), O (52: 43%), and T (40: 33%). The lowest glycaemic changes occurred in the first interval for all participants.

For total cholesterolaemia, concentrations increased from baseline in all participants within the first hour (Fig. 5 and Table 2). The hourly changes followed a time-dependent pattern in participants A and O (Fig. 2 and Table 2). Over two hours, total cholesterol levels increased in four participants (A, E, I, and O) but decreased in two participants (J and T), as shown in Table 2.

Over the two weeks, alternating changes in fasting glycaemia among participants consuming choice food portions at the 100 mg/dL glucose concentration threshold (Table 3) showed a reduction in glycaemia on all alternate measurement days. Moderate hyperglycaemia was observed in four participants on Days 2, 4, and 8, with the highest and lowest levels recorded on Days 6 and 10, respectively. Normal glycaemia (glucose concentration at or below 100 mg/dL) was on Days 4 and 12. Day 14 showed the most significant reduction in mean glyacaemia, while Days 6, 8, 10, and 14 exhibited statistically significant differences from the baseline ($P<0.05$), as presented in Table 3.

The weekly variations in fasting cholesterolaemia among participants consuming choice food portion sizes over two weeks, based on a 200 mg/dL threshold for total cholesterol concentration (Table 4), showed a reduction in five participants during the first week. In the second week, total cholesterol levels decreased from their respective baseline participants. Participant J had consistently high total cholesterol levels, with values of 246 mg/dL at baseline, 250 mg/dL in the first week, and 231 mg/dL in the second week, exceeding the 200 mg/dL threshold. Overall cholesterolaemia, there was a progressive decline in the mean weekly total cholesterol concentration.

The effect of food portion size on body mass index (BMI), resulting from changes in body weight (Fig. 6), showed a decrease in BMI for all participants. The weekly decline from baseline followed a time-dependent pattern across all participants. Participant O maintained a normal BMI in the first (24) and second week (23). Except for Participant T (24, 23, and 22), baseline BMI values for all other participants exceeded the threshold level of 24. The lowest BMI for each participant is in the second week (last stage, see Fig. 6).

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Table 1: Preliminary pilot glucose testing on four separate days

Participant	Glucose Concentrations in mg/dl					Occasions of moderate hyperglycaemia
	Ist-day	2nd-day	3rd-day	4th-day	Mean	
A	120^{mh}	127^{h}	124^{mh}	123^{mh}	123.50^{mh}	3
B	138^{h}	140^{h}	145^{h}	167^{h}	147.50^{h}	-
C	135^{h}	127^{h}	142^{h}	125^{mh}	132.25^{h}	1
D	160^{h}	157^{h}	143^{h}	167^{h}	156.75^{h}	-
E	123^{mh}	122^{mh}	111^{mh}	130^{h}	121.50^{mh}	3
F	156^{h}	142^{h}	155^{h}	125^{mh}	144.50^{h}	1
G	173^{h}	205^{h}	200^{h}	199^{h}	194.25h	-
H	130^{h}	145h	169h	120^{mh}	141.00^{h}	1
I	123^{mh}	102^{mh}	121^{mh}	100	111.50^{mh}	3
J	124^{mh}	100	102^{mh}	123^{mh}	112.50^{mh}	3
K	167^{h}	158^{h}	188^{h}	165^{h}	169.50^{h}	-
L	121^{mh}	100	99	121^{mh}	110.25^{mh}	2
M	124^{mh}	100	102	123	112.50^{mh}	1
N	185^{h}	140^{h}	155^{h}	171^{h}	162.75^{h}	-
O	120^{mh}	130^{h}	123^{mh}	112^{mh}	121.25^{mh}	3
P	124^{mh}	130h	127^{h}	123^{mh}	126^{h}	2
Q	100	125^{mh}	121	125^{mh}	117.75^{mh}	2
R	100	121^{mh}	143^{h}	127^{h}	122.75^{mh}	2
S	132^{h}	143^{h}	136^{h}	140^{h}	137.75^{h}	-
T	123^{mh}	130^{h}	111^{mh}	124^{mh}	122^{mh}	3

A, B, C, D, E, F, G, H, I and J = names of subjects
h = hyperglycaemia
mh = moderately hyperglycaemia

Numbers bearing ^{h} = fasted glucose concentration of diabetes
Numbers bearing ^{mh} = fasted glucose concentration above normal but below diabetes.

100 prediabetes $\leq$ 125 mg/dL) (American Diabetes Association, 2010, 2012 Eun *et al*., 2016).

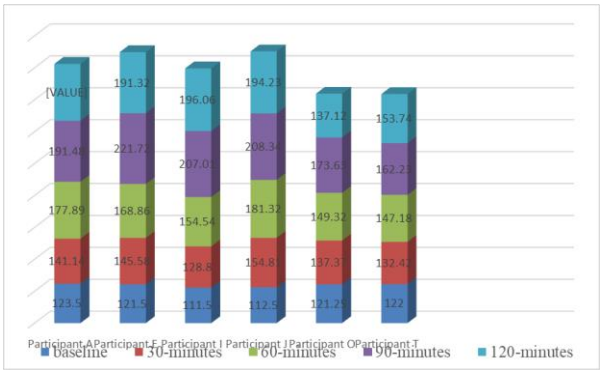

Fig: 4 Pre- and post-prandial glucose concentrations (mg/dl) at 30 minutes intervals of four in five days.

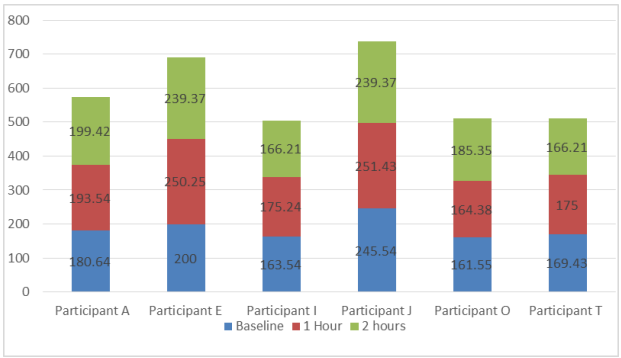

Fig. 5: Pre- and post-prandial Total cholesterol concentrations (mg/dl) at 1hr intervals of two for five days.

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Table 2: Pre and post-prandial glucose and cholesterol concentrations (mg/dl) trajectories

Participant	Glucose changes								Cholesterol changes			
	30 minutes		60 minutes		90 minutes		120 minutes		1hour		2 hours	
	Cn	%	Cn	%	Cn	%	Cn	%	Cn	%	Cn	%
A	18	14	54	44	68	55	55	44	13	7	19	10
E	24	20	47	39	100	83	70	58	50	25	39	20
I	17	16	39	35	96	86	85	76	12	7	3	2
J	42	38	69	61	96	85	82	73	6	2	-6	3
O	16	13	28	23	52	43	16	13	3	2	24	15
T	10	9	25	21	40	33	32	26	6	3	-3	2

P=participants
A, E, I, J, O and T= name of participants
Number bearing^{-} = reductive change

Table 3: Two weeks of alternate day's glycaemic status (mg/dl) of participants fed on choice food portion size.

Part	baseline	Day 2	Day 4	Day 6	Day 8	Day 10	Day 12	Day 14
A	124^{mh}	112^{mh}	120^{mh}	122^{mh}	102^{mh}	90	99	80
|E	122^{mh}	113^{mh}	111^{mh}	101^{mh}	102^{mh}	81	78	80
I	112^{mh}	118^{mh}	107^{mh}	102^{mh}	101^{mh}	82	96	73
J	113^{mh}	108^{mh}	100	106^{mh}	98	101^{mh}	94	98
O	121^{mh}	96	106^{mh}	103^{mh}	101^{mh}	91	98	91
T	122^{mh}	95	100	88	76	80	81	79
Mean	119	107	107	104*	97*	86*	91*	84*

P=participants
A, E, I, J, O and T= name of participants
Day bearing* was significant with Baseline at P<0.05
Baseline = mean glucose concentration of participants from Table 1
mh = moderately hyperglycaemia
Numbers bearing ^{mh} = fasted glucose concentration above normal but below diabetes.

Table 4: Two weeks alternate week cholesterol status (mg/dl) of participants fed on choice food portion size

Participants	Baseline	Day 7	Day 14
A	181	153	159
E	200	167	197
I	164	156	147
J	246^{h}	250^{h}	231^{h}
O	162	158	113
T	169	158	160
Mean	187	174	167

A, E, I, J, O and T= name of participants

Baseline = mean pre-prandial Total cholesterol concentration of participants from Fig. Table 1

Number bearing^{h} = hypercholesterolaemia

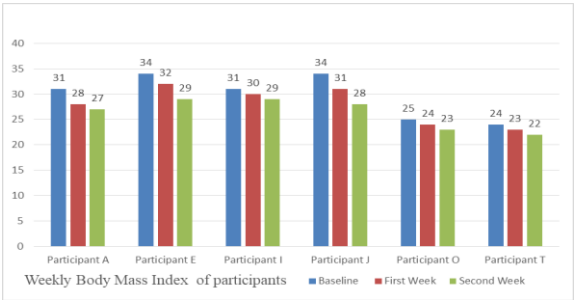

Figure 6: Effect of Food Portion sizes on the Weekly Change in body mass index.

Discussion

Elevated blood sugar levels, known as hyperglycaemia, pose a significant risk for diabetes, and dietary habits play a crucial role in its development. There was no universally accepted definition of eating behaviour and the best food in existing literature. However, the connection between dietary patterns and chronic non-communicable diseases has been widely studied (*Micha et al., 2017*). Pre-diabetes (Fig 1) often precedes diabetes, highlighting the importance of early

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screening to intervene at this initial stage. Pre-diabetes is a fasting glucose concentration between 100 and 125 and was used as the threshold, following guidelines from the American Diabetes Association (2010, 2012) and Hammer et al. (2019).

The screened participants (n=20) were as pre-diabetic or diabetic based on a glucose concentration threshold of 100 mg/dL or higher (Table 1) on at least one occasion. When extrapolated to the broader population, these findings highlight the epidemic nature of the disease, as previously reported (Faselis *et al.*, 2019). Individuals with pre-diabetes face a heightened risk of progressing to diabetes (Rooney *et al*., 2021) and were, therefore, subjected to food portion size interventions in this study.

During the first stage of glucose testing- both pre-prandial and postprandial at 30-minute intervals following a fasting period- glucose concentrations tended to accumulate in both phases. This observation aligns with findings from Rizza (2010). It is possible that prior meal consumption triggered an insulin surge, contributing to insulin resistance and elevated blood glucose levels in diabetes. The body may have counteracted excessive insulin secretion by increasing insulin resistance. The sustained rise in glucose concentration at each interval, up to 90 minutes, suggests a parallel increase in insulin resistance, a key feature of diabetes.

A subsequent drop in glucose levels at the 120-minute mark (Fig. 4) may indicate either reduced insulin secretion or an oversaturation of insulin that facilitates glucose clearance from the bloodstream. It aligns with the concept of portion size control, or mindful eating, explored in the present study. Postprandial glucose levels exceeding 200 mg/dL at two hours reflect impaired glucose homeostasis in pre-diabetes and diabetes, as observed in this study (Fig. 4) and previously reported elsewhere (Hiyoshi et al., 2017). The higher postprandial glucose and cholesterol concentrations compared with respective baselines suggest an association (Fig. 4 and Table 2). However, this relationship was not strongly evident at the two-hour postprandial mark.

Hyperglycaemia in diabetes is closely linked to hypercholesterolaemia, as observed in this study and recently reported by Wang et al. (2020). The most significant percentage changes in glucose and cholesterol levels occurred during the second hour of the postprandial phase, highlighting the correlation between these two parameters and the need for further investigation.

Additionally, a two-week assessment of glyceamia and cholesterolaemia following selected food portion interventions showed a notable impact. By days 10 and 12, pre-diabetes had completely reversed to normal (non-diabetic) in some participants, while hypercholesterolemia, though improved from its baseline of 246 mg/dL, had not fully normalized but showed progress (243 mg/dL). This study's restorative approach focused on meal timing and portion control and their effects on glucose-cholesterol metabolism and body mass index (BMI). Previous research by Hernandez-Rodas *et al.* (2015) identified diet as a key factor influencing both glucose and cholesterol metabolism. Similarly, Ulven *et al.* (2016) and Abdelhamid et al. (2020) explored the varying relationships between blood glucose and cholesterol levels. More recently, Wang *et al*. (2020) and Omar et al. (2018) reported that poor glycaemic control was associated with elevated total cholesterol, a relationship mirrored in this study, where improved glucose control corresponded with favourable total cholesterol levels.

Furthermore, reductions in Body mass index among participants improved glycaemia and favourable cholesterol changes, a finding increasingly recognized in nutritional science. Research has shown that meal timing and portion size influence postprandial glucose levels (Node and Inoue, 2009, Leung *et al*., 2019 and Takahashi *et a*l., 2018). Meal portioning and timing may likely interact with the circadian rhythms, affecting glucose disposal and cholesterol metabolism and benefiting overall health. Moreover, food portion control may mitigate the prolonged hypoglycaemic effects that trigger cortisol secretion, as cortisol reduces cellular glucose utilization (Catapano *et al*., 2016 and Ramamoorthy and Cidlowski, 2016). As a result, glucose may accumulate alongside cholesterol, potentially leading to degenerative changes (Fig. 1, 2 and 3).

Interestingly, based on the research model, the association between glucose and total cholesterol appears continuous, with glucose normalization closely linked to improvements in total cholesterol levels.

Statistical analysis

Data presentation was the mean. One-way ANOVA followed by Tukey's HSD test was performed, with significance at $p < 0.05$

Conclusion

Misinformation contributes to misguided treatment strategies in diabetes management, posing a growing global challenge that endangers those affected. The findings of this study demonstrate that reversibility of pre-diabetes and maintaining stable glucose homeostasis are associated with improved normalization of total cholesterol levels- all of which are rooted in mindful eating habits. Pre-diabetes reversion was through mindful food selection, controlled portions, and well-timed meals.

Acknowledgment

We are grateful to TETFUND and the POLAC community.

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