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Short Communication **Open Access**

Exploring the metabolic harmony: Probing the impact of VHBAX probiotics on glucose metabolism in a zebrafish model

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Abstract:

Background: Probiotics alter gut microbiota composition and diversity. Research indicates that probiotic supplementation might enhance glucose homeostasis and reduce inflammation in patients with type Ⅱ diabetes mellitus. The aim of this study was to investigate how particular probiotic strains (VHBAX *Bacillus coagulans*) affect glucose homeostasis and appetite-related behaviours associated with metabolic illnesses such as obesity and type Ⅱ diabetes in a zebrafish model.
Methodology: An experimental laboratory-based investigation was carried out with zebrafish (*Danio rerio*) as the model organism. The zebrafish were divided into 3 groups of 6 zebrafish each; control (group I), diabetic-induced (group II), and diabetic-induced plus probiotic treatment (group III), with the latter receiving a diet supplemented with VHBAX probiotic strains. Glucose tolerance testing, and gene expression analysis for glucose metabolism were carried out in all the 3 groups. The composition of the gut microbiota was also analysed using 16S rRNA sequencing to assess microbial alterations related to probiotic administration. Data were analysed using SPSS 16.0 and presented as mean±SD. Difference between means of two groups was determined by Student *t*-test, and $p<0.05$ was considered statistically significant.
Results: When compared to diabetes-induced zebrafish (group II), diabetes-induced probiotic-treated zebrafish (group III) had enhanced glucose tolerance, altered expressions of appetite-regulating genes (AgRP, CNR1, CTSL1, Oxt, CCKAr, leptin A), and detectable alterations in gut microbiota composition, comparable to the control (group I). There was a significant association of VHBAX probiotic supplementation with microbial regulation and improved metabolic outcomes.
Conclusion: This experimental investigation shed light on the mechanisms by which VHBAX probiotics regulate glucose metabolism and appetite management through alteration of gut microbiota composition in zebrafish. The finding suggests that probiotic therapies can help manage or prevent metabolic disorders, including obesity and type Ⅱ diabetes.

Keywords: Zebra fish; Glucose metabolism; Diabetes; Microbiota; Metabolic disorder; Probiotics

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Exploration de l'harmonie métabolique: étude de l'impact des probiotiques VHBAX sur le métabolisme du glucose dans un modèle de poisson-zèbre

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Résumé:

Contexte: Les probiotiques modifient la composition et la diversité du microbiote intestinal. Des recherches indiquent qu'une supplémentation en probiotiques pourrait améliorer l'homéostasie du glucose et réduire l'inflammation chez les patients atteints de diabète de type II. L'objectif de cette étude était d'examiner l'impact de certaines souches probiotiques (*Bacillus coagulans* VHBAX) sur l'homéostasie du glucose et les comportements liés à l'appétit associés à des maladies métaboliques telles que l'obésité et le diabète de type II chez le poisson zèbre.

Méthodologie: Une étude expérimentale en laboratoire a été menée sur le poisson zèbre (*Danio rerio*) comme organisme modèle. Les poissons zèbres ont été divisés en trois groupes de six poissons zèbres chacun; témoin, diabétique induit et diabétique induit avec traitement, ces derniers recevant un régime alimentaire supplémenté en souches probiotiques VHBAX. Des tests de tolérance au glucose et une analyse de l'expression génique du métabolisme du glucose ont été réalisés dans tous les groupes. La composition du microbiote intestinal a également été analysée par séquençage de l'ARNr 16S afin d'évaluer les altérations microbiennes liées à l'administration de probiotiques. Les données ont été analysées avec SPSS 16.0 et présentées sous forme de moyenne ± écart type. La différence entre les moyennes des deux groupes a été déterminée par le test *t* Student, et une valeur de $p<0,05$ a été considérée comme statistiquement significative.
Résultats: Comparés aux poissons-zèbres diabétiques (groupe II), les poissons-zèbres diabétiques traités aux probiotiques (groupe III) présentaient une tolérance au glucose accrue, une expression altérée des gènes régulateurs de l'appétit (AgRP, CNR1, CTSL1, Oxt, CCKAr, leptine A) et des altérations détectables de la composition du microbiote intestinal, comparables à celles du groupe témoin (groupe I). Une association significative a été observée entre la supplémentation en probiotiques VHBAX et la régulation microbienne et une amélioration des résultats métaboliques.
Conclusion: Cette étude expérimentale a permis de mieux comprendre les mécanismes par lesquels les probiotiques VHBAX régulent le métabolisme du glucose et la gestion de l'appétit en modifiant la composition du microbiote intestinal chez le poisson-zèbre. Ces résultats suggèrent que les thérapies probiotiques peuvent contribuer à la prise en charge ou à la prévention des troubles métaboliques, notamment l'obésité et le diabète de type II.

Mots-clés: Poisson zèbre; Métabolisme du glucose; Diabète; Microbiote; Trouble métabolique; Probiotiques

Introduction:

Probiotics alter gut microbiota composition and diversity (1). Research indicates that probiotic supplementation might enhance glucose homeostasis and reduce inflammation in Type Ⅱ diabetes mellitus (DM) patients (2,3). Probiotics alter gut flora, creating beneficial molecules that demonstrate the importance of gut microbiota for human and animal health (4). Gut microbiota affects metabolism and energy balance for weight management. Rygan et al., (5) reported that probiotics can dramatically lower HbA1c and insulin levels in type Ⅱ diabetics without affecting lipid profile.

Many diabetic investigations on zebrafish larvae have shown that real-time PCR may detect PEPCK expression in anti-diabetic medicines (6). Zebrafish have genetic and physiological parallels to humans, making them an established system for metabolic and behavioural research. Zebrafish, as herbivores, can tolerate glucose better. The efficacy was connected to gut bacteria, specifically acetate-producing commensal *Cetobacterium somerae.* Acetate and *C. somerae* stimulated the parasympathetic nerve system, improving gut-brain glucose homeostasis (7). Impaired leptin signalling in mutant zebra fish may influence gut anorexigenic gene regulation (7).

The pandemic of obesity necessitates improved intervention techniques focusing on gut bacteria and metabolic products such as high-fat diets which favour *Firmicutes, Prevotella*, and *Methanobrevibacter* species growth. This leads to inflammation, increased hunger-suppressing hormones, dyslipidaemia, and reduced short-chain fatty acid (SCFA) expression (8). Understanding the connection between gut microbiota and glucose metabolism could lead to new diabetes and obesity prevention strategies (9).

The primary objective of this study is to evaluate the effects of probiotics (VHBAX *Bacillus coagulans*) on glucose metabolism. To achieve this, measuring and analysis of the expression of key genes involved in glucose metabolism pathways is needed. This gene expression analysis will provide insights into how probiotics may influence or modulate glucose metabolism in zebrafish under diabetic conditions.

Materials and method:

Study setting and ethical consideration:

This zebra fish study project proposal number SU/CLATR/IAEC/XXI/07/2023 entitled unveiling the impact of probiotics (VHBAX *Bacillus coagulans*) on glucose metabolism. Insights from a zebrafish animal model was approved by the IAEC of Satyabama Institute of Science and Technology, Chennai 600119, India.

Experimental and control animal:

Adult zebrafish from Chennai's Satyabama Institute of Science and Technology were housed in a semi-static system with charcoal-filtered tap water. Three groups of zebrafish (n=6 each) were housed in 3-liter recirculating tanks at 28.4°C under 14:10 hours light: dark conditions for the experiment. Facility water quality was checked regularly.

Each group was given different conditions to assess the VHBAX probiotic effects; Group I (control) received standard fish feed for a regular diet; Group II were induced diabetic zebrafish (100 mg alloxan per kg of zebrafish body weight) and kept in 10% sugar till the experiment ended after diabetes induction (28 days); Group III were induced diabetic zebrafish with probiotics administered by oral method. This group received sucrose induction and maintenance and *Bacillus coagulans* VHBAX probiotics daily till the experi-

ment ended (28 days) and were used to assess effects on glucose metabolism.

Determination of blood glucose as a measure of glucose tolerance in zebrafish:

The zebrafish were anaesthetized using hypothermia method by placing them in 17°C facility water and gradually reducing the temperature to 12°C by adding ice made from the facility water. Fish brought to stage II anaesthesia, allowed handling and still had opercular movements. The anaesthetized zebrafish were then decapitated by cutting through the pectoral girdle. Whole blood was collected for immediate analysis using test strips applied directly to the cardiac blood. Approximately 5-10µL of blood collected for blood glucose was accurately measured using Accu-Chek Compact Plus (1.5µL sample).

Histopathological examination of pancreatic tissues:

The zebrafish were euthanized by immersion in a freshly prepared buffered tricaine methane sulfonate (MS-222- RM2178 Hi Media) solution (200–300 mg/L, pH 7.0–7.5) until opercular movement ceased, followed by decapitation to confirm death before tissue collection. Whole mounts of control and experimental fish pancreas tissues were fixed, and the pancreas tissue slices were deparaffinized in xylene at 65°C for 20 minutes to dissolve paraffin wax. Both areas received two 10-minute xylene clearings after the initial treatment. Tissue slices were then immersed in alcohol solutions at 100%, 90%, 70%, 50%, and 30%. Each portion was hydrated with distilled water for 10 minutes.

Tissue slices were stained with Mayer's haematoxylin for 15 minutes to show cellular characteristics and the stain was removed by washing slides with tap water for 10 minutes and then counterstained with eosin for 2 mins. The slides were prepared for mounting after dehydrating and eliminating eosin stain. Two xylene changes followed 30%–100% alcohol soaking of the slides and light microscopic examination of DPX-mounted slices.

Measurement of glycolytic enzyme activity:

Liver tissue was collected immediately after sacrificing experimental animals for glycolytic enzyme measurement. The liver tissue was homogenised in ice-cold buffer (pH 7.6). The hexokinase homogenate was centrifuged at 9000 rpm for 100 min and glycolytic enzymes at 10000 rpm for 20 min. Pancreas from control (Group I) and experimental (Groups II and III) zebrafish were homogenised in 50mM Tris-HCl (pH 7.5), 4mM EDTA, 50mM sodium fluoride (NaF), 0.5mM phenylmethylsulfonyl fluoride, 500mM 1,4-dithiothreitol, and 250 mM sucrose.

The homogenate was centrifuged at 14000 rpm for 30 min at for hexokinase and fructose-1, 6-biphosphatase activities. After 30 minutes at 37ºC, the reaction was stopped by adding 10% TCA solution. The absorbance was 340 nm. The glycogen phosphorylase (GP) activity was determined by monitoring the reduction of oxidised NADP during glycogen breakdown at 340 nm, expressed in micromole NADP/min per gram of protein. The GP activity is defined as the conversion of 1µmol glycogen and orthophosphate into glucose-1 phosphate per minute at 30°C and pH 6.8 (11-12).

Quantitative real-time PCR assay for glucose metabolism genes:

Quantitative analysis of core glucose metabolism genes [CCKAr (cholecystokinin A receptor), CNR1 (cannabinoid receptor-1), CTSL1 (cathepsin L1), leptin A, AgRP (agouti-related peptide), and Oxt (oxytocin)] of the control and experimental zebrafish was done by real-time PCR assay. First, total RNA was isolated from the liver tissue and quantified according to the method of Chomczynski and Sacchi (13). A cDNA conversion kit (Thermo Scientific Inc.) created cDNA from total RNA after reverse transcription (RT).

About 0.5-1.0µg total RNA was combined with 1.5µL random hexamer primer (GAP DH-F:5′-TGCACCACCAACTGCTTAGC-3′ and GAPDH-R:5′-GGCATGGACTGTGGTCATGA-3′), incubated at 72°C for 10 minutes, and chilled immediately. This was then combined with 5.0µL premixed 10 mmol/L dNTP solution, 3.0 µL 10x M-MLV RT buffer, and 1.0 µL M-MLV RT in RNase/DNase-free water to reach 50µL. SYBR green fluorescent dye was used for BioRad CFX96 real-time PCR. Internal control was GAPDH.

Analysis of gut microbiota by 16S rRNA sequencing:

The gut microbiota (intestinal content) of zebrafish was analyzed using 16S rRNA gene sequencing. The whole gut was aseptically dissected from anaesthetized zebrafish, rinsed gently with sterile phosphate-buffered saline (PBS) and DNA extracted using QIAamp DNA Microbiome Kit. The hypervariable region (V3–V4) of the 16S rRNA gene was amplified using the universal bacterial primers 341-F (5-CCTACGGGNGGCWGCAG-3) and 805-R (5-GA CTACHVGGGTATCTAATCC-3). Amplicons were purified, quantified, and pooled in equimolar concentrations before sequencing on Illumina MiSeq platform.

Bioinformatic analysis of the 16S rRNA sequenced data was done by Shannon and Chao1 for alpha diversity, and by Principal Coordinates Analysis (PCoA) for beta diversity of the microbial communities of the control, diabetes-induced, and diabetes-induced probiotic-treated zebrafish groups. Functional prediction of the metagenome functions of the

microbial community based on the 16S rRNA sequencing profiles was carried out using PICRUSt2 (https://github.com/picrust/picrust2).

Statistical analysis:

Statistical analysis of data was done using SPSS 16.0. Data were presented as mean ± S.D (n=6). Difference between means of two groups was determined by Student *t*-test, and *p*-value below 0.05 was considered statistically significant.

Results:

Histopathological structures of pancreas in zebrafish in control and experimental groups:

Histological analysis of the pancreas tissue from both the control and experimental groups provided valuable insights into the effects of probiotic treatment on glucose metabolism (Fig 1). Compared to the control (group I) and diabetic-induced probiotic-treated fish (group III), the pancreatic tissue mass of diabetes-induced zebrafish (group II) showed disrupted pancreatic tissues.

Blood glucose levels in zebrafish in control and experimental groups:

Based on the study, elevation of glucose levels resulted in hyperglycemia in diabetes-induced zebrafish group (II) in comparison to the control (group I). The probiotic VHBAX significantly reduced the blood glucose levels of treated zebrafish in group III, bringing them close to those of the control group (as seen in Fig 2).

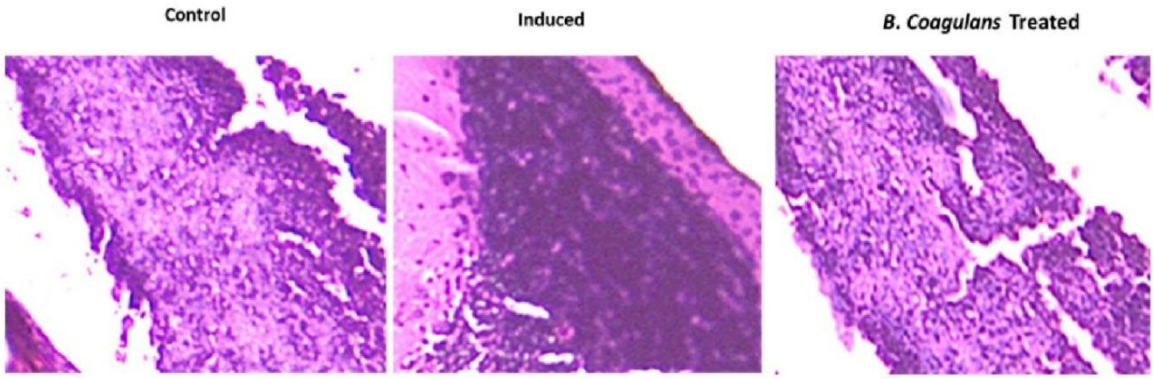

Fig 1: Microscopical observations of pancreas sections of control and experimental zebrafish groups

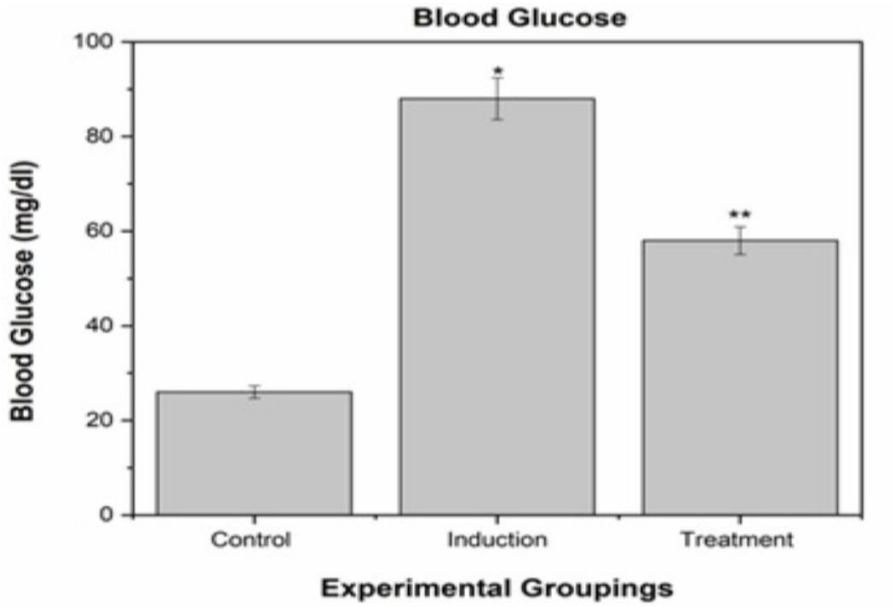

Fig 2: Quantification of blood glucose level in the control and experimental zebra fish groups

Glycolytic enzyme activity in zebrafish in control and experimental groups:

The activities of glycolytic enzymes, including hexokinase and phosphofructokinase-1 (PFK-1), were reduced in diabetes-induced zebrafish (group II) compared to the control (group I), while the activities considerably increased in group III zebrafish treated with VHBAX probiotics (Fig 3).

Metabolic gene expression profiles in zebrafish in control and experimental groups:

With respect to the gene expression profile, there was overexpression of AgRP, CNR1, CTSL1 and leptin A, and reduced expression of CCKAr and Oxt genes in diabetes-induced zebra fish (group II) compared to the control (group I) (Fig 4). VHBAX probiotic treatment of diabetes-induced zebrafish (group III) had significant effect on metabolic regulators such as AgRP, CNR1, and Oxt whereas the expression of CCKAr and Leptin A was decreased. These indicated the disturbed appetite and glucose signalling by suppressing orexigenic markers (AgRP, CNR1) and normalising anorexigenic genes (CCKAr, Leptin A) (Fig 4).

However, in probiotic treatment (group III), CCKAr expression to near-normal levels and supress leptin A expression to approximate control values, indicating that probiotic supplementation mitigated diabetes-related dysregulation of starvation and metabolic genes.

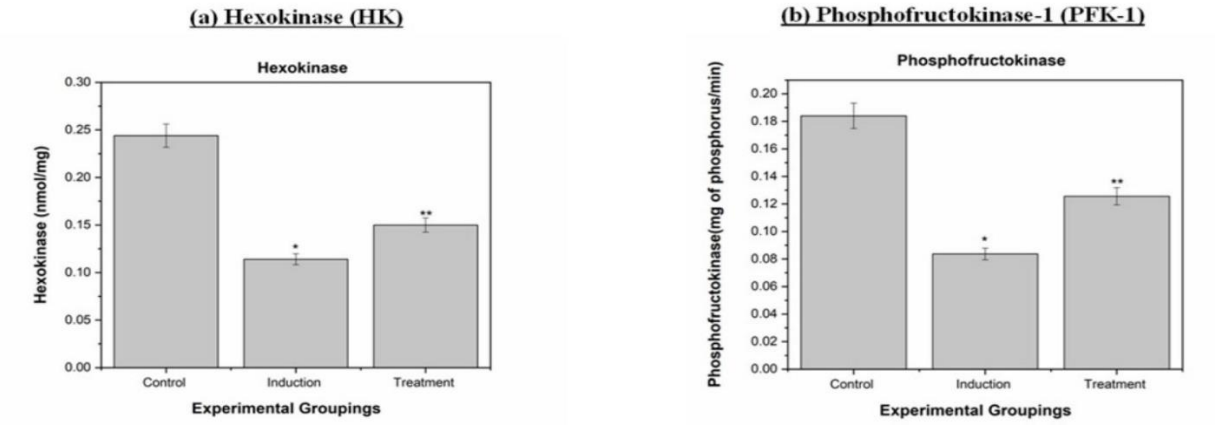

Fig 3: Quantification of key glycolytic enzymes of zebrafish in control and experimental groups

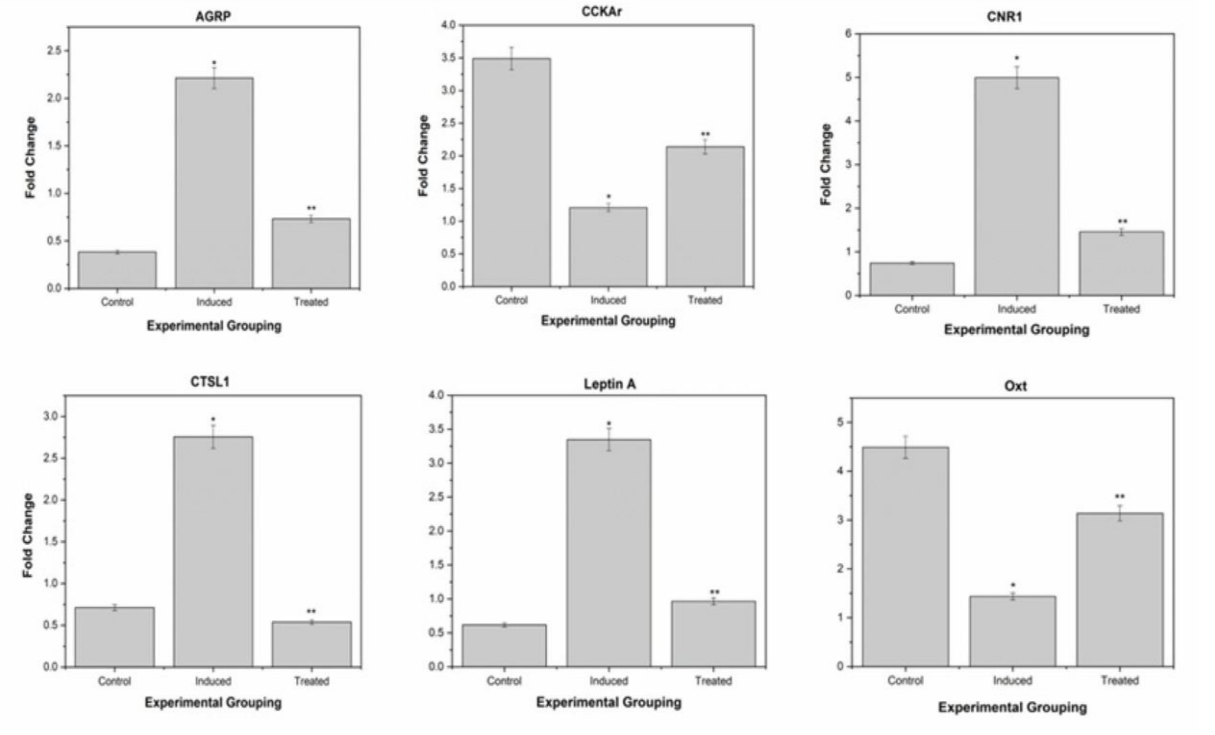

Fig 4: qPCR analysis of key genes involved in glucose metabolism of control and experimental zebrafish groups

Gut microbiota diversity of zebrafish in control and experimental groups:

The diabetes-induced zebra fish (group II) showed a lower alpha diversity (Shannon, Chao1) than the control (group I), which indicates that there was a less abundance of microbial species (Fig 5). The incorporation of VBHAX probiotics to diabetes-induced zebrafish in group III dramatically improved the diversity in line with the control (group I).

The results of the beta diversity analysis (PCoA) demonstrated that the groups were classified in a different manner. The microbial community demonstrated signs of recovery, as evidenced by the fact that diabetes-induced probiotic-treated zebrafish (group III) were placed closer to the control zebrafish (group I) and further away from diabetes-induced zebrafish (group II) when they were categorised into groups (Fig 6).

According to the results of functional prediction carried out using PICRUSt2, the diabetes-induced probiotic-treated zebrafish (group III) had a higher number of pathways for the production of short-chain fatty acids (SCFAs) and was more efficient at breaking down glucose.

Gut microbiome composition of zebrafish in control and experimental groups:

The result for the 16S rRNA sequencing showed that there were different microbiome patterns present in each of the groups. The microbial balance of the control zebrafish (group I) was in good condition, as evidenced by the fact that majority of their microbiomes were composed of *Cetobacterium* spp. This equilibrium was broken in diabetes-induced zebrafish (group II) which had increased number of opportunistic *Firmicutes* and *Proteobacteria*, an indication of dysbiosis.

The variety of healthy microbes was increased through the use of VHBAX treatment of zebrafish in group III, which accomplished this by increasing the number of beneficial commensals such *Bacteroides* spp while simultaneously reducing the number of pro-inflammatory taxa (Fig 6).

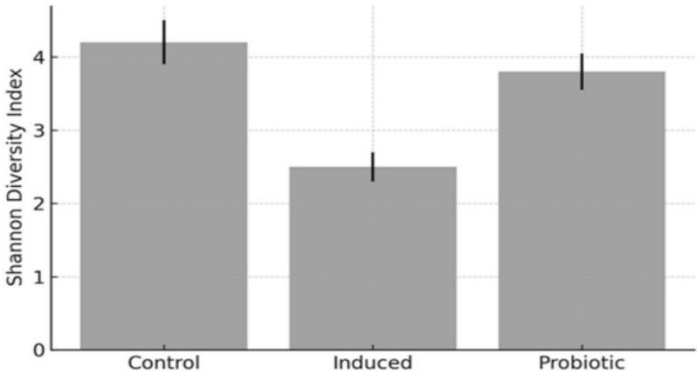

Fig 5: Alpha diversity (Shannon Index) showing microbiota richness in control, induced and probiotic treated zebrafish groups

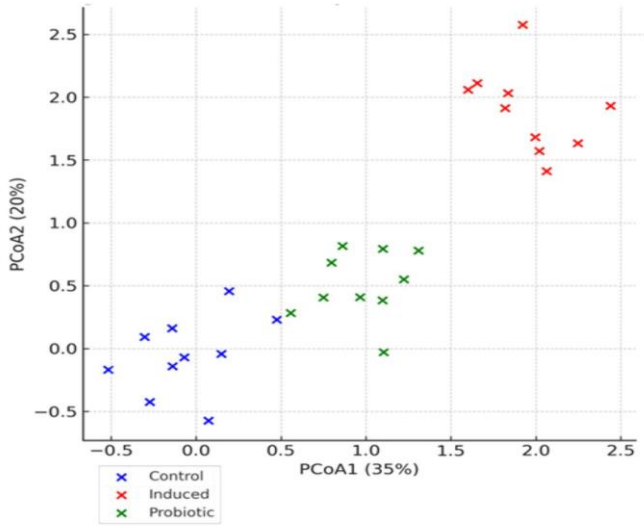

Fig 6: Beta diversity (PCoA plot) depicting distinct microbial community clustering among the three groups

Discussion:

This study compared metabolic processes in three zebrafish groups in different conditions, and the effects of VBHAX probiotics on zebra fish glucose metabolism were examined. In zebrafish in group III (diabetes-induced probiotic-treated), blood levels were lower than in the diabetes-induced zebrafish (group II) but higher than the control (group I). It appears that probiotic stabilised glucose metabolism and reduced diabetes in the zebrafish. Diabetes is caused by insulin sensitivity loss and high glycemia in early to late obesity (4). Gut microbiota may compensate for glucose metabolism in obese children, and genus *Haemophilus* and *Bifidobacterium longum* may alter glucose tolerance and insulin resistance (14).

The zebrafish in the diabetes-induced group had considerably reduced hexokinase (HK) and phosphofructokinase-1 (PFK-1) activity than the control group. Diabetic zebra fish have significant glucose metabolism failure, as shown by this enzyme activity reduction. Hexokinase and phosphofructokinase-1 activity rose considerably in probiotic-treated zebra fish. As shown by this exceptional enzyme activity increase, probiotics may control pancreatic enzymes to improve glucose metabolism. Gut microbes are known to regulate hunger. Probiotics altered zebra fish's gut microbiome, reducing appetite and blood sugar (15). A meta-analysis found that probiotics significantly lowered cholesterol, triglycerides, CRP, HbA1c, fasting plasma glucose, insulin, and systolic/diastolic blood pressure in type Ⅱ diabetic patients. Probiotics may be useful in treating type 2 diabetes by raising HDL levels (15).

Gene expression studies reveal probiotics increase glucose metabolism. CCKAr, CNR1, CTSL1 gene-encoded lysosomal cysteine protease, and neuropeptides such as leptin A, AgRP, and Neuropeptide Y (NPY) regulate glucose levels and metabolism. The glucose metabolism and regulation in the liver are explained by the CCKAr gene expression by creating an insulin-glucagon receptor protein that regulates blood glucose. These hormones (insulin and glucagon) stimulate CCKAr, which starts a cascade of events to customise blood glucose responses. CCKAr increases in treatment group show probiotics alter gene expression and receptor activation. Lysosomal cysteine protease (cathepsin L1) breaks down proteins and performs other cellular functions and can indirectly affect glucose metabolism through cellular nutrition sensing, autophagy, and lysosomal activity. The role of CTSL1 in glucose metabolism is unknown. The diabetes-induced zebrafish (group II) had increased CTSL1 expression, suggesting lysosomal and metabolic activity. The diabetes-induced probiotic treated zebrafish (group III) had low CTSL1 expression levels, indicating that probiotic inhibits CTSL1, which promotes glucose metabolism.

The cannabinoid receptor type 1 gene expression produces the CB1 receptor, which regulates signal transduction, cellular communication, and physiological regulation. G-protein coupled receptors in the central nervous system interact with endocannabinoids to regulate pain, hunger, and neuroprotection. Additionally, CB1 receptors impact peripheral tissue glucose control, inflammation, and energy balance. High CNR1 expression in the diabetes-induced zebrafish group in this study may indicate higher endocannabinoids signalling that regulates glucose and brain circuits. Reduced CNR1 expression levels in diabetes-induced probiotic-treated group indicate improved glucose metabolism.

In vertebrates like zebrafish, leptin A controls energy balance, hunger, and metabolism. Mostly produced by fat cells, it suppresses hunger and increases energy expenditure in the hypothalamus when body fat levels are sufficient. Leptin production is related to adipose tissue reserves, increases calorie burning, balances fat storage, and prevents obesity. Dysregulation can cause obesity and metabolic problems. Diabetes-induced zebrafish treated with probiotics had lower leptin A levels, leading to reduction in hunger and energy expenditure.

The hypothalamus produces AgRP, a neuropeptide that controls energy and feeding behaviour. AgRP targets brain receptors such the melanocortin-4 receptor (MC4R) to enhance hunger and decrease energy expenditure. AgRP dysregulation can cause obesity and metabolic diseases, hence understanding the AgRP system may lead to obesity treatments. lower AgRP levels in the diabetes-induced probiotic treated group in our study imply lower appetite stimulation and improved glucose management. The brain and peripheral organs are rich in neuropeptide Y (NPY) which controls glucose metabolism, hunger, and energy balance. Regulation of glucose metabolism by NPY is by regulating beta cell insulin secretion and peripheral insulin sensitivity. In diabetes, NPY promotes hunger and food intake, which may affect glucose management.

The 16S rRNA study showed the changes in the healthy microbiota had metabolic benefit on the system. According to previous studies, the replenishing of *Cetobacterium somerae* in zebrafish maintain their glucose levels by producing acetate (7). Likewise, increased *Bacteroides* spp may promote the synthesis of short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate in mammals for boosting glucose metabolism and reducing inflammation. Likewise, *Lactobacillus*

rhamnosus has been shown to replenish healthy gut bacteria, raise SCFA levels, change the expression of genes that lower glucose, reduce hunger, and lower blood glucose in zebrafish (4).

The results of these studies align with our current research that showed VHBAX probiotic supplementation induces microbial modifications and metabolic improvements. Finding from the current study suggests the two possible glucose metabolism pathways of VHBAX probiotics; first, by upregulating enzymes and modulating gene expression, and secondly, by modifying healthy microbiota that increased production of short-chain fatty acids, improving dysbiosis and promoting beneficial host signalling. Hence, VHBAX probiotics is suggested in the prevention and management of metabolic disorders, including type II diabetes and obesity. However, deep understanding of microbial diversity, SCFA metabolites, and predictive functional profiling are useful tools to provide broad understanding on the metabolic pathway which connect host and microbiota.

Conclusion:

Our study shed light on the mechanisms by which VHBAX probiotics regulate glucose metabolism and appetite management. The finding suggests that probiotic therapies can help manage or prevent metabolic disorders, including obesity and type 2 diabetes.

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Contributions of authors:

RS and PR conceptualized the study; SVSP and SK curated the data; SK and RS designed the methodology; RS and SVSP performed the experiment; PR and SK provided resources; PR and SVSP were involved with data validation; RS and PR were involved in data visualization; RS, SVSP, PR and SK were involved in manuscript writing. All authors read and approved the final manuscript submitted for publication.

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Conflict of interest

Authors declare no conflict of interest.

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