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EFFECT OF EXERCISE ON DIABETES

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ABSTRACT

Background : Evidence suggests that diabetes mellitus is reaching pandemic proportions in poorer countries, and in many industrialized nations it is already ranked fourth or fifth among leading causes of mortality. Almost 65,1 million people in India have been diagnosed with diabetes in the last several years, making it a serious public health concern.

Methodology: This literature review followed a procedure devised by the authors. From their beginnings, the electronic bibliographic databases MEDLINE, CINAHL, EMBASE, and SPORTDISCUS were searched thoroughly. The databases were searched using the keywords physiotherapy, physical therapy, physical therapist, physiotherapist, physical activity, exercise, walk, health promotion, health education, and patient education. Only articles written in English were retrieved. After searching for intervention studies and reviewing their abstracts and full texts, we found 14 research; however, only 5 met our inclusion criteria.

Conclusion: In light of the results of this research, we are able to draw the conclusion that physical activity has a number of important impacts. The findings of this research have offered data to suggest that exercise may enhance cognition in a susceptible population of young people

INTRODUCTION

Most affluent nations rank diabetes mellitus as the fourth or fifth leading cause of mortality, while data reveals an epidemic spread of the disease in emerging nations(1). There are now about 65.1 million people in India who have been diagnosed with diabetes, raising serious concerns about an impending pandemic. A cloudy future loom over the nation in terms of how to deal with the imminent threat presented by diabetes mellitus(2). The negative effect on the health of the new generation has been exacerbated by the rapid changes in the economy over the last three decades, as well as shifts in people's diets, ways of life, customs, social cultures, and family structures(3). Type 2 diabetes mellitus (T2DM)

is becoming one of the main causes of sickness, disability, and mortality in the modern world because of the direct and indirect links between T2DM and behavioural, dietary, and environmental variables. A third of all newly diagnosed cases of diabetes in young adults have occurred in children and adolescents during the last two decades(4). With the highest number of confirmed cases, India is currently recognised as the "diabetes capital of the globe," posing a serious problem for the whole world(5).

A lot has changed in the previous three decades regarding the prevalence of diabetes. While it was formerly thought to mostly afflict the elderly, the illness has evolved into a major source of morbidity and death among the young. Recent articles have detailed the issue in length. Type 2 diabetes mellitus has rapidly become epidemic, doubling its incidence across all demographic subsets in a very short period of time(6). This is because to people's increasingly sedentary lifestyles, decreased levels of physical exercise, and their preference for high-fat, processed meals. Damage to the brain's end organs may be the ultimate consequence of diabetes mellitus, a multifactorial, slowly progressing, and complicated metabolic condition. Macrovascular and microvascular complications of diabetes are a leading cause of death and disability among people with the disease(7).

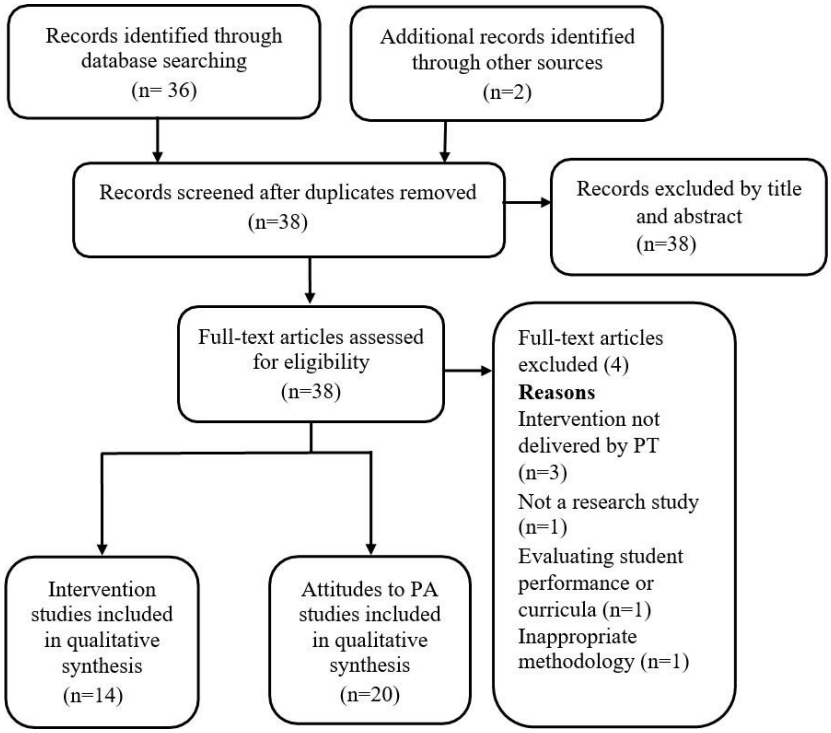
The cognitive impairment that often accompanies diabetes is not often discussed or even widely acknowledged. There is growing evidence that the cognitive impairments experienced by diabetics contribute to the overall severity of the disease. Diabetes mellitus has been linked to cognitive deficits, including memory loss, difficulty concentrating, and a slower processing speed(8). Unfortunately, the exact mechanism through which diabetes-related cognitive impairment develops and at what stage of the disease it occurs remain unknown. Most research has been done on people who already have diabetes and have had it for a long time. The problem is that type 2 diabetes progresses slowly and is often misdiagnosed until it is well advanced. As a result, cognitive impairment may already be present in the pre-diabetic stage, long before a formal diagnosis is made. Evidence of glucose intolerance, including hyper and hypo glycemia episodes that might impair cognitive function, is present in pre-diabetic individuals before they are officially identified as diabetic(9). Both low and high blood sugar levels have been linked to cognitive impairment in people with diabetes mellitus. It has been found that both T1DM and T2DM patients have mild to significant cognitive deficits. Reduced cognitive performance in diabetics is

associated with many different behaviours and is mediated by the frontal lobe. Skills like problem solving, planning, organizing, foresight, logic, and focus are examples. Energy in the brain is mostly metabolized from glucose. The blood-brain barrier can only be breached to acquire it(10). A lack of glucose, for which the brain needs it most for both short-term and long-term memory, is a risk in people with diabetes mellitus because cells cannot respond to insulin signals. That's why the hippocampus is such a hotspot for memory problems as a consequence of cell death(11). The hippocampus is a brain area essential for learning and memory storage. Specific insulin receptors mediate insulin's transit through the blood-brain barrier. Inadequate insulin action has been linked to reduced synaptic activity in the brain. Striatal neuronal cholinergic activity is decreased by insulin, while monoamine levels are raised. Despite the abundance of literature, the pathogenesis of cognitive impairment is still unclear, and effective means of diagnosing, treating, and preventing cognitive dysfunction in diabetes mellitus have not yet been established(12).

As with food and medicine, exercise is regarded as a cornerstone in the treatment of diabetes mellitus. The evidence supporting regular exercise as a means to both prevent and manage diabetes is now overwhelming. Exercise has been shown to aid diabetics with glucose management, body composition, cardiorespiratory endurance, insulin resistance, physical functioning, and well-being, but its influence on neurocognitive behavior is unclear(13).

A significant share of India's population is comprised of young adults. Acute bouts of hyperglycemia and hypoglycemia occur during the pre-diabetic period, also known as the impaired glucose tolerance phase, before a person is officially diagnosed with diabetes. Acute episodes may cause micro-vascular disorders, which can thereafter cause cognitive impairments.

METHODOLOGY



This literature review followed a procedure devised by the authors. From their beginnings, the electronic bibliographic databases MEDLINE, CINAHL, EMBASE, and SPORTDISCUS were searched thoroughly. The databases were searched using the keywords physiotherapy, physical therapy, physical therapist, physiotherapist, physical activity, exercise, walk, health promotion, health education, and patient education. Only articles written in English were retrieved.

After searching for intervention studies and reviewing their abstracts and full texts, we found 14 research; however, only 5 met our inclusion criteria. Nine further studies were not included because the practitioner giving the intervention was not a physiotherapist or because the outcome measures were not directly connected to physical activity or cardiovascular fitness.

DISCUSSION

Adults (aged 20–45) with a recent diagnosis of type 2 diabetes were the study population. It affects how your body uses glucose, protein, and fat, and is often regarded the most common metabolic condition. In addition to being a serious health issue, it is also a big social and economic issue across the world. It's a complicated illness that covers a wide range of conditions seen in modern Western countries. Early-stage diabetes mellitus often goes undetected for three to five years due to the disease's insidious nature. By the time a disease is identified, the underlying alterations that cause micro- and macrovascular problems have already taken place. 206-208 In the recent two or three decades, India's population health has rapidly shifted, just as it has in other emerging nations. The substantial increase in India's per capita income over this time period has had a profound effect on the country's urbanization and standard of living. As a result of its prevalence, diabetes mellitus has emerged as a top public health concern, however it is a condition that may be altered by increased awareness and early diagnosis. The incidence of diabetes has increased by a factor of three over all of India, with rates tripling in both urban and rural areas. We are rapidly becoming used to all of the conveniences of contemporary life in our nation. The natural physical activities are being ignored. Getting about town quickly is no longer a priority for most individuals, who instead opt for motorized cars.

Information acquisition, processing, integration, storage, and retrieval are all examples of cognitive functioning. The human memory system is very intricate. The temporal lobe of the cerebral cortex is traditionally thought to be responsible for memory. One of the most consistently observed effects of hyperglycemia and hypoglycemia is memory impairment. The results of this research show that people with diabetes have worse cognitive abilities than those without the disease. This is true across a range of tests, including those measuring attention, focus, memory, IQ, arithmetic, comprehension, and information. The results of this research suggest that systematic exercise treatment leads to statistically significant gains in key memory and intelligence-related areas.

Stewart and Vandenberg found that people with diabetes had impaired memory, executive function, and processing speed. Evidence from a number of research shows that both type 1 and type 2 Diabetes mellitus are associated with an adverse correlation between glycated haemoglobin

and mental function. Brain shrinkage and vascular lesions have been linked to cognitive deterioration in diabetics, according to research using magnetic resonance imaging.

Results from this study show that diabetics have poorer attention and concentration, as well as lower scores on immediate recall, delayed recall verbal retention for dissimilar pairs, visual retention, and intelligence tests, compared to healthy controls on the Stroop test and psychomotor function response performance. When organized exercise treatment was applied, a noticeable uptick in symptoms was seen. Our results are unique because they show a correlation between moderate hyperglycemia and impaired performance of executive functions in individuals aged 20-45 who have just been diagnosed with type 2 diabetes. These findings are consistent with those from other research that have shown the impact of glucose control on a variety of frontal-lobe functions.

Five significant circuits connecting frontal lobe regions to subcortical areas have been identified by Alexander et al. All five of these pathways seem to independently connect the caudate, globus pallidus, and thalamus to the dorsolateral, motor, and anterior cingulate areas of the brain. The Stroop tasks rely on the dorsolateral circuit, which allows for the interchange of conceptual sequences and the suppression of a cognitive set. The putamen is on the path of the motor loop, which begins at the site of action selection and concludes in the supplemental motor region. Behavior is controlled by a dorsolateral circuit.

Here, we found that the diabetes group performed much worse on the Stroop reaction test than the healthy control group. Because these five circuits all go via the striatal areas, this may be an indication of their compromised competency.

Working out and mental function are two sides of the same coin. Complex task performance in the frontal lobes has been shown to suffer in studies; yet, moderate intensity exercise has been shown to increase cognitive function by improving prefrontal oxygenation. Hillman et al. monitored P300 amplitude and latency in young people before and after a single session of treadmill activity while they performed an executive cognitive task electrophysiologically. According to his findings, physical exertion boosts neuro-electrical activity. Adults who are physically fit have been shown to have greater P300 amplitudes and shorter P300 latencies in other research. Exercise has been shown to boost cognitive performance in several studies. Neuroimaging studies also suggest that regular exercise has positive effects on brain activity and cognition. Several structural MRI studies

conducted on older populations have shown that those who maintain a healthy level of physical activity are more likely to keep their brain volume as they age. Six months of aerobic exercise significantly increases brain volume in the frontal, temporal, and parietal cortices of older individuals.

The cognitive process of spatial memory is likewise mediated by the hippocampus, and the research by Erickson et al. found that aerobic exercise increases hippocampal volume. Improvements in declarative memory, as measured by a word recall memory task and the Rey Auditory Verbal Learning Test, were observed in research done by Pereira et al. on persons aged 21 to 45 who engaged in a three-month aerobic exercise programme.

Brain-derived neurotrophic factor (BDNF) and insulin-like growth factor-1 (IGF-1) are two well-studied molecular mediators that provide light on the impact of exercise on mental acuity.

Running wheel exercise has been proven to increase levels of brain-derived neurotrophic factor (BDNF) in the hippocampus, which is critical for synaptic plasticity, learning, and memory in rodent studies. Researchers found that exercising the animals made them better at navigating the Morris water maze. There is a need for spatial cognition and memory for this activity. They also observed that the benefits of exercise on the Morris water maze challenge were nullified when participants received an injection that suppresses BDNF function.Exercising mice, according to the research, showed higher levels of BDNF in the hippocampal area of the brain immediately after exercise, which remained elevated for several weeks later until returning to normal values. Therefore, due to its considerable involvement in hippocampal functioning, learning, memory, and plasticity, BDNF may have a crucial role in moderating the impact of exercise therapy on the brain and cognition. The brain contains the trophic factor IGF-1. The brain may produce it locally, and it can also get it from the bloodstream in the form of IGF-1. Increased absorption of IGF-1 from the plasma into a few distinct regions of the brain has been seen in animal studies.by use of a regular physical training programme. Medications that inhibit IGF-1's actions, as shown in the aforementioned research, also counteract the neurogenesis generated by exercise. Low levels of IGF-1 have been associated with changes in cognitive processes including learning and memory. Levels of IGF-1 decline with age, and this decline is positively correlated with cognitive function in the elderly. Increases in synaptic plasticity, neurogenesis, and vascular functions are only some of the ways that neurotrophins and growth factors improve mental performance.

Physical activity increases hippocampus-based long-term potentiation (LTP) and reduces synaptic plasticity's inhibition threshold. Glutamate-mediated synaptic transmission and synaptic plasticity are also enhanced by exercise, as is BDNF signalling. Exercising increases synaptic plasticity by activating BDNF and IGF-1 mkinase signalling cascades (e.g., mitogen-activated protein kinase, calcium/calmodulin protein kinase II), which in turn increases synapse transmission by upregulating synaptic proteins such synapsin I. The intracellular signalling pathway, comprised of CaMKII and MAP kinase, demonstrates the influence of exercise on BDNF by facilitating the operation of CAMP response element binding protein (CREB). Infusion of BDNF has been shown to improve memory and cognition.

As a vital clinical tool for assessing cardio-respiratory fitness and predicting future adverse cardiovascular events, cardio-respiratory endurance tests have become more significant in recent years.The VO2 max and the Physical Fitness Index (PFI) are two of the most critical metrics for gauging a subject's cardiopulmonary efficiency, which leads us to the next point in our discussion.

240 When someone is physically healthy, they can go about their everyday routines without tiring out.The value of maintaining a healthy body has been discussed at various points throughout human history.

However, the study of exercise physiology is still relatively new and has many unanswered questions. The majority of health-related anthropometric measurements were shown to have a substantial negative correlation with determinants of physical fitness, according to research conducted by Leila Jaafari et al. Sameer et al. found that the fitness ability of college students aged 18–25 years old steadily reduced with increasing body mass index.According to the research conducted by Anabel et al. 246, people who are overweight or obese tend to be less physically fit.

The results of this research show that young individuals newly diagnosed with T2DM had considerably lower levels of physical fitness, as measured by both the physical fitness index and the VO2 max. Following six months of interventional treatment, patients showed considerable increases in their VO2 max and physical fitness index values. Past research has shown that cardiorespiratory fitness and exercise go hand in hand (VO2max).

Low cardio-respiratory fitness has been linked to insulin resistance in many studies, although not in diabetes individuals. Furthermore, it has been hypothesised that VO2 max might serve as a

reliable indicator of insulin resistance.Previous research has also shown that diabetics had 20% worse cardiorespiratory fitness (VO2max) compared to age- and gender-matched healthy controls. Kaplan et alresearch .'s shows that insulin controls the activity of an anion transporter in mitochondria during the Krebs cycle.Genetic reduction in mitochondrial number explains reduced glucose and oxygen use. This might have an adverse effect on the person's fitness level, resulting in a reduced VO2 max.

The amount of time it takes a person to respond to an external stimuli is known as their response time (RT). This means that a person's conduct is a direct result of their sensory input. Auditory and visual reactions are regarded to be the gold standard for gauging sensory motor connection.

In this research, we observed that diabetics' visual response times were much longer than their auditory ones. Chemical reactions are one potential mechanism at play.Hyperglycemia in diabetes is linked to chemical changes in nerves and blood vessels, which provide oxygen and other critical nutrients to the nerves, as described by a number of previous research.

Enhanced processing capabilities of the CNS with exercise treatment may explain the reduction in response time shown in the current investigation after six months of interventional therapy; this improvement in sensory-motor performance was found to correlate with the onset of therapy.

Muscle mitochondrial content and aerobic capability improve as a result of regular exercise. This results in less glycogen being used for energy and a greater ability to oxidise fat. This adaptation delays tiredness, boosts enzyme activity and response speed, and lengthens work duration.The same results were found by Joki et al, who also noted that exercise boosts focus, wakefulness, alertness, muscle coordination, and cognitive quickness.

One of the long-term effects of diabetes mellitus is diabetic neuropathy, which may appear at any point throughout the disease's progression but usually worsens with time. Evaluation of diabetic neuropathy often involves measuring nerve conduction velocity.

Patients with type 2 diabetes mellitus and no clinical signs of neuropathy had slower ulnar nerve motor conduction velocities than healthy controls in the current investigation. After receiving systematic exercise treatment, patients showed considerable increases in their nerve conduction velocity. Bertsman's research found that even in individuals whose diabetes was well-managed, motor nerve function decreased.

Similarly, previous research has shown that 18.1% of asymptomatic diabetes individuals had median mononeuropathy without any signs of polyneuropathy, which is similar with our findings. Researchers found a slowing in electrical impulse transmission in diabetics compared to healthy controls.Some research suggests that de-myelination of nerves in asymptomatic diabetes people causes a slowdown in nerve conduction velocity. Annually, diabetes individuals' nerve conduction velocities fall by around 0.5 m/sec, according to a cohort study.Hyperglycemia is likely the key component in the pathogenesis of diabetic neuropathy, however the data as a whole point to a multi-factorial aetiology. The stimulation of the polyol pathway, non-enzymatic glycosylation of proteins, and oxidative stress all contribute to the destruction of nerve cells brought on by hyperglycemia.

Diabetic participants' glucose profiles, which were previously higher than those of healthy controls, have decreased thanks to exercise treatment. Several studies have shown that hyperglycemia in pre diabetic conditions might cause the activation of several physiological pathways that ultimately result in macro and micro vascular complications. Postprandial hyperglycemia may result from a pathophysiological mechanism that inhibits the generation of endogenous insulin by altering the insulin response of beta cells in the Langerhans of the pancreas during the initial phase of secretion. Endogenous insulin synthesis drops more when beta cells degenerate. Over time, when beta cells are unable to make insulin, the postprandial high sugar causes the creation of significant levels of reactive oxygen species (ROS), which then drives death of pancreatic beta-cells.After beta-cell failure, postprandial hyperglycemia may cause a surge in blood sugar levels, which can be harmful to the cells that produce insulin.

The efficacy of oral anti-diabetic medicines vs exercise treatment, which includes both aerobic and resistance exercise, in improving glycemic control in patients with type 2 diabetes mellitus was evaluated in a meta-analysis.

Blood glucose uptake route is not insulin dependent and is normal even in diabetics who engage in exercise treatment, which includes both aerobic and resistance workouts. Skeletal muscles maintain glucose uptake activity for many hours after a workout. Multiple studies have found the same thing.

Diabetics had significantly greater levels of triglycerides and very low-density lipoprotein (VLDL), and lower levels of high-density lipoprotein (HDL), compared to the general population.

Even while both total cholesterol and LDL were higher in diabetics than in healthy controls, the difference was not statistically significant in newly diagnosed diabetics. There is substantial evidence linking this lipid profile pattern to diabetes. With six months of exercise treatment, there was a notable improvement in the various components of lipid profile. Several research have shown this to be true as well.

Dyslipidemia is linked to insulin resistance, and impaired lipid metabolism is thought to be an early step in the progression to full-blown type 2 diabetes mellitus. Researchers have shown that people with dyslipidemia tend to have lower levels of high-density lipoprotein cholesterol and lower levels of low-density lipoprotein cholesterol, with a moderate rise in triglyceride levels. Impaired intracellular hydrolysis of triglycerides and increased release of no esterified (free) fatty acids (NEFAs) into the blood stream underlie the altered action of insulin in adipose tissue. Increased plasma levels of triglycerides and very low-density lipoprotein follow from this because it increases the inflow of NEFAs into the hepatocytes and the synthesis of these two lipids. Despite being cardio-protective, higher triglyceride levels cause a decrease in HDL cholesterol and other protective factors. Several studies have shown a link between glucose and lipid profile. Because of their close relationship, problems with carbohydrate metabolism also cause alterations in lipid metabolism. This is further related with obesity epidemic and even increased cost of diabetes management by two folds.

50 Studies have reported increased prevalence of obesity by 70% in young adults of age group 18-29 years. Studies have also reported 70% increased prevalence of T2DM 70% in young adults of age group 30-39 years over last ten years, making young population the rapid emerging group for obesity and T2DM. 295 The onset of DM is insidious and takes almost three to four years for its diagnosis and in this silent phase there is already presence of minimal changes responsible for micro and macro vascular complications. Therefore, prevention is very important from monetary point of view.

Various studies have emphasized the need to increase awareness about diabetes mellitus, its risk factors and preventive measures. Along with these, lifestyle modifications like diet control and physical exercises should also be emphasized.

In the present study, most of the diabetics belonged to overweight and obese category. (BMI > 30 Kg/m2). Depress has also found similar results and reported that particular range of abdominal

obesity is important risk factor for the development of T2DM. 297 Many studies have also reported same findings and confirmed them by using advanced, more accurate techniques for measuring abdominal fat. Skin- fold thickness measurements and their ratios are reported by various studies as are strong predictors of morbidity.

The study by Banerjee et.al observed changes in BMI values in early adulthood resulting in development of diabetes at younger age.

The Health Professionals Follow-Up study reported increased risk of type 2 diabetes mellitus by 3.5 % if weight gain is 8–9 kg from age of 21 years and have also reported increased risk by 7.3% for every 1-kg of weight gained.

Weight gain caused by excessive fat accumulation leads to resistance to insulin through changes in endocrine activity, specifically through the increased release of non-esterified fatty acids and glycerol, hormones (e.g., leptin and adiponectin), and pro-inflammatory cytokines (TNF-α, IL-6) further increasing the demand for production of insulin from the pancreas. Insulin resistance is hallmark of T2DM and includes decreases in glucose transporter number and impaired activities of key intracellular enzymes (e.g., pyruvate dehydrogenase andglycogen synthase).

The exercise therapy has improved anthropometric indices in diabetics and our findings are consistent with previous studies. The mechanism of exercise induced improvement in anthropometric parameters which was seen to be associated with improved insulin sensitivity in skeletal muscles during and after exercise regimen. Bogardus et al. demonstrated, use of insulin clamp technique in obese diabetics which leads to significant improvements in insulin levels stimulated by glucose levels following combined diet and exercise therapy as compared to diabetics on dietary treatment alone. Several studies have reported improvement in physiological and biochemical parameters with aerobic exercise regimen. Moreover, aerobic exercise can also help in restoring endothelial function and also helps in reducing arterial stiffness which are positively associated with development of cardiovascular complications in T2DM.This is may be due to increased glucose uptake into skeletal muscle by the enhanced activity of the glucose transporter (GLUT4).

Numbers of studies have documented the potential benefits of resistance training. Resistance exercise helps in management of blood glucose levels and decreases insulin resistance in patients

with T2DM. Various studies have also reported increased insulin sensitivity with resistance exercises by its potential benefits in increasing muscle strength, lean muscle mass, and bone mineral density. This helps in improving the glycemic control in diabetics. Aerobic exercises have gained attention to improve neurocognitive functioning. Cross-sectional studies have shown better neurocognitive functions in physically active subjects as compared to same age and sex matched inactive individuals.

Prospective observational studies have also reported similar findings. However, randomized trials have provided inconsistent results as some are reporting cognitive gains and others reporting ambiguous findings. In summary, we found impaired neuro-physiological and cognitive functions in newly diagnosed patients with type 2 diabetes mellitus. Significant improvement was observed with structured exercise therapy consisting of aerobic and resistance exercises when compared to diabetic controls. However, our findings require validation with molecular studies to establish underlying mechanisms.

CONCLUSION

In light of the results of this research, we are able to draw the conclusion that physical activity has a number of important impacts. The findings of this research have offered data to suggest that exercise may enhance cognition in a susceptible population of young people.

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