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The effects of effortless exercise on diabetic status

Abstract

Treating physicians have consistently recommended exercise to either prevent diabetes or de-escalate symptomatology. Diabetic complications, however, render physical activity undesirable or unattainable. These involve: hypothyroidism leading to substantial weight gain; perpetual fatigue due to accumulation of white adipose tissue serving as fat storage, and inadequate supply of brown fat to generate energy; accumulated toxicity causing hormonal imbalance that increases hunger; chronic pain and wounds on extremities associated with diabetic neuropathy, etc. Recent research with an effortless exercise method demonstrated enhanced fitness and T3 increase, juxtaposed by decreased inflammation, an optimal relationship between leptin and ghrelin that control appetite, and a significant decrease of visceral fat along with VLDL, the very low-density lipoprotein that carries triglycerides to the tissues. We measured the fasting and postprandial glucose and insulin of 21 diabetics and 20 prediabetics respectively, pre and post twenty treatments. Both previously abnormally high fasting and postprandial (PP) glucose decreased considerably in all 21 diabetic subjects (100%). Nine of the diabetic subjects (42.85%) manifested normal fasting glucose levels after 20 treatments, while the fasting glucose of the remaining twelve diabetic subjects (57.2%) dropped down to the prediabetic level. Ten of the diabetic subjects (47.6%) manifested normal PP insulin levels, while the PP insulin of the remaining eleven diabetic subjects (52.38%) dropped to the prediabetic level after the 20 treatments. Prediabetics had more robust results as expected by their baseline healthier status. Eighteen of prediabetics (90%) manifested both normal fasting and PP insulin levels after the 20 treatments, while the fasting and PP insulin of the remaining two subjects (10%) remained within the prediabetic level. All subjects also exhibited a statistically significant increase in muscle mass, normalized T3 levels, decreased visceral and overall fat along with reduced CRP, advocating diminished inflammation. Dyslipidaemia appeared to subside as denoted by suppressed levels of triglycerides contrasted by elevated HDL.

Keywords: diabetes mellitus, metabolism, visceral fat, insulin, cholesterol, inflammation, Glucose, insulin, T3, CRP, triglycerides, VLDL, HDL, dyslipidaemia. muscle mass, fitness, effortless Exercise, visceral fat, adiposity, physical activity

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Introduction

Diabetes definition

Diabetes encompasses a variety of metabolic disorders primarily related to either an insulin deficit which defines the primary cause of Type 1 diabetes (T1D) or an insulin resistance commonly found in Type 2 diabetes (T2D).[1] Autoimmune diabetes falls under the T1D category. T1D ordinarily emanates out of defective immunity characterized by an insufficient amount of B-cells whose primary function is to develop antibodies against invasive antigens. T1D is distinguished by usually normal weight, and it is primarily diagnosed in children, adolescents, and young adults who exhibit symptomatology such as polyuria, polydipsia and fatigue.[2] T2D is a cluster of diseases associated with both hyperglycaemia and metabolic syndrome that is typically represented by obesity, with excessive visceral fat deposits, low-grade inflammation and increasing mortality rates. T2D is associated with an inverse relationship between triglycerides and high-density lipoproteins (HDL), where increased levels of triglycerides are accompanied by abnormally low HDL; additionally, it is linked to hypertension that often leads to enhanced risk of coronary heart disease (CRD) or strokes. The severity of T2D progresses over a dimension that ranges from reduced insulin secretion to persisting insulin resistance induced by deficient insulin production.[3] Diabetes has been connected to a number of other disorders that include Cushing Syndrome, defined by hypercortisolism;[4] pancreatitis, propagated by pancreatic inflammation;[5] acromegaly, distinguishable by an enlargement on the hands and feet due to an excess of growth hormone (GH);[6] cystic fibrosis that affects the lungs, liver, kidneys and intestine and is expressed in difficulty breathing or coughing;[7] hemochromatosis delineated by an iron overload;[8,9] and pheochromocytoma that involves a benign tumour in the adrenal gland. There's clinical evidence that diabetes may develop as a result of pharmaceutical treatments with atypical neuroleptics[10] often prescribed to treat schizophrenia, glucocorticoids,[11] or alpha-interferons.[12]

Diabetes and hypothyroidism

Experimental evidence links T1D with hypothyroidism by showing that subclinical hypothyroid adolescents demonstrate a higher incidence of hypoglycaemic symptomatology.[13] A series of animal model studies pharmaceutically induced both a diabetic and a hypothyroidic status by administering streptozotocin and propylthiouracil respectively. They observed an increased sense pre-mRNA of the b gene that is associated with a lower contraction rate of the myosin-heavy chain, consistent with the simultaneous presence of diabetes and hypothyroidism, thus connecting the two. This finding was complemented by the observation that the sense RNA of the gene that regulates a faster level of muscle contraction in normalcy, was substantially decreased.[14] A study on 1112 diabetics found a connection between T2D and hypothyroidism, especially in individuals over 65.[15] An earlier investigation of the records of 922 T2D patients unveiled a high correlation between T2D and hypothyroidism, with a prevalence in white subjects.[16] Research has denoted that diabetes is underlaid by a defective mechanism that

fails to generate Triiodothyronine (T3) from thyroxine (T4), the 4-iodine atoms hormone produced in the thyroid gland, resulting in systemically insufficient levels of T3 in diabetics.[17] T3 was also found to prevent cellular apoptosis, previously induced experimentally by streptozotocin injections, that were administered to artificially precipitate diabetes. In this animal study the deleterious effects of the streptozotocin injections were partly reversed by T3. T3 protects B cells by activating the PI3K-Akt (Ak strain transforming) signalling pathway that generally promotes cellular survival and growth. The beneficial effects of T3 on cellular integrity are significant in light of the connection between defective proliferation, or extensive apoptosis of B cells, and hyperglycaemia which is considered the cornerstone of both T1D and T2D. T3 injections appear to act as an "anti-diabetic" intervention counteracting the diabetic deterioration following streptozotocin injections, illustrated by a documented reinstatement of insulin responsiveness as well as the euglycemic range of serum glucose levels.[18–22]

Diabetes, VLDL, triglycerides, LDL and HDL

The very low-density protein (VLDL) that is normally composed in the liver, transports triglycerides (esters comprised of glycerol and three fatty acids), that represent the main source of energy storage in tissues, otherwise known as overall body fat. Increased levels of triglycerides carried by VLDL are the hallmark of dyslipidaemia that is commonly accompanied by inhibited levels of high-density lipoprotein (HDL). HDL absorbs both low-density and very low-density lipoproteins transferring them back to the liver, thus relieving the arteries of potential plaque build-ups, and reducing the risk of both atherosclerosis and heart disease. Dyslipidaemia is intrinsically linked both to insulin resistance and T2D.[23] Liu et al [24] tested the null hypothesis that ischemic strokes and heart disease may not be directly related to a high ratio of triglycerides reciprocated by low HDL. These investigators examined the health status of 30,378 individuals over a period of fifteen years. Failing to falsify a hypothesis is the most experimentally scientific method of proving a premise. Liu's results confirmed the strong connection between a pathological lipid profile of high triglycerides predicting coronary heart disease and low HDL being associated with ischemic stroke, with a high prevalence of Diabetes and high low-density lipoproteins (LDL) being present in patients with coronary heart decease.[24]

Triglycerides and diabetic neuropathy

Recent studies have associated T2D Neuropathy with dyslipidaemia defined by an abnormally high triglycerides/ low HDL profile.[25,26] Diabetic neuropathy is characterized by chronic pain, anomalous sensations and malfunctioning nerve conduction velocities (NVCs) underlaid by deficient sural nerve myelinated fibre densities (MFDs). Wiggin et al[26] followed patients with high triglycerides and abnormalities in motor nerve conducting velocities for one year. Their study unveiled a significant correlation between dyslipidaemia and deficient motor nerve conduction velocities that are the foundation of diabetic neuropathy. A recent research project focused on symptomatology relief and pain analgesia from chronic diabetic neuropathy with patients who had a history of multiple hospitalizations, followed by an accumulation of medical expenses, and a poor prognosis that involved the imminent threat of a lower limb amputation as the best case scenario to avoid further deterioration.[27] A second study that reviewed different therapeutic modalities on diabetic wound healing with lasers and ultra-low microcurrents, demonstrated fast irreversible improvement of diabetic lesions following treatment with a novel nanotechnology using nano-energies.[28]

Sedentary lifestyles increase oxidative stress and inflammation

Hyperglycaemia disrupts both insulin signalling and insulin secretion by pancreatic B cells, provoking an inevitable deterioration of the diabetic condition.[29] Clinical research has demonstrated that increased hyperglycaemia elevates oxidative stress and suppresses antioxidant production that could potentially donate electrons to reinstate symmetry in the otherwise disequilibrium state of the radical oxygen species (ROS). In vitro studies on the mitochondria of obese type 2 diabetics have evidenced a significant increase of ROS.[30] For the average individual, the absence of exercise renders detoxification insufficient and therefore, unable to establish the necessary balance between the production and elimination of free radical species, routinely formed by normal aerobic metabolism via oxygen. Toxicity erodes the boundaries between health and illness, being exacerbated by the growing immune limitations during ageing. Inadequate detoxification results in accumulated oxidative damage that adversely affects the diabetic condition instigating glucotoxicity, lipotoxicity, and cardiac dysfunction; hence the therapeutic intervention provided by antioxidants and exercise which are highly recommended for both T1D and T2D.[31] Exercise is defined as a coordinated set of repetitive movements at different speeds and resistances, as well as physical activity, interpreted as representing a regular pattern of increased everyday motion. Both have been long recommended in the treatment of diabetes by a number of national guidelines including the American Diabetes Association which has disclosed clinical evidence of reducing the prevalence of T2D by 58% as a result of an active lifestyle.[32–34] Oxidative stress has been intricately related to C-Reactive protein, the hepatic origin inflammation marker that is linked to proinflammatory cytokines, and which has been consistently associated with both diabetes and cardiac dysfunction.[35–37] An experimental study on 529 subjects established a statistically significant correlation between CRP and mononuclear cells' oxidative stress, as well as demonstrating that ROS in polymorphonuclear leucocytes and mononuclear cells were prevalent in both diabetes and hypertension.[38] Diabetes is aggravated by obesity which is defined by low-grade inflammation and an excess of CRP, identified in the white adipose tissue (WAT). WAT is primarily used for energy storage, in contrast to the brown adipose tissue (BAT) that is predominantly involved in energy production.[39] Overall, visceral adipose tissue (VAT) is associated with diabetic hyperinsulinemia, glucose intolerance, hypertriglyceridemia, and dyslipidaemia, defined as a combination of high triglycerides and inhibited HDL, oxidative stress and inflammation as marked by CRP.[40,41]

Diabetes and exercise

A literature search on the multidimensional spectrum of diabetic treatments usually reiterates the same recommendation, pertaining to lifestyle changes and exercise.[42–45] Nevertheless, there are concerns associated with certain types of exercise, which increase blood glucose levels and, in certain subjects, result in abnormal hypoglycaemia.[46] Clinical studies on dynamic exercise have demonstrated hyperglycaemia and hyperinsulinemia in diabetics, persisting for at least one hour after physical training.[47] Additional research has delineated a disproportionate increase of seven- to eightfold glucose production as a result of intensified catecholamine signalling, accompanied by a deficient glucose utilization, limited to only three- to fourfold.[48,49] Sedentary lifestyles increase the incidence of diabetes and coronary heart disease by 30-50%.[50] Exercise spends glucose-derived energy that could theoretically help diabetic hyperglycaemia, however, the long-term effects of exercise on diabetes' dysregulated metabolic profile remain inconclusive.[51] A 2006 survey revealed that

exercise was recommended to 73% of diabetes patients as opposed to only 31% of non-diabetic adults, however, very few of these patients increased their physical activity.[52] Exercise appears to decrease diabetic symptomatology, hence being beneficial to those with both T1D and T2D who can use exercise as a protective, therapeutic method against further deterioration. However, patients with advanced diabetes complicated by obesity or neuropathic pain will be obviously less willing or capable of exercising.[53,54] A number of clinical studies on a novel effortless fitness technology from London University, have delineated a reduction in both visceral and overall fat, demonstrating improved hormonal regulation, and a reversal of the diabetic status into either the realm of prediabetes or normalcy.[55–60] A more recent clinical study on diabetics with hyperphagia reports hunger suppression as a result of an optimal inverse relationship between leptin increase and ghrelin decrease.[61] The current research examined levels of pre and post-T3 and C-reactive Protein (CRP), as well as pre and post-fasting and postprandial (PP) insulin and glucose levels in the blood samples of forty-one diabetic and prediabetic subjects. The study also measured the potential of the treatment to attain an optimal inverse relationship between triglycerides and HDL. The goal of the study was to offer diabetics the benefit of enhanced fitness and weight control while overcoming the general resistance to exercise.

Methodology

We utilized an apparatus originally built at London University in 2008 by Gerald Pollock, an electronics engineer who was also involved in the invention of the first pacemaker in the UK, based on his combined research with Donald Gilbert, a molecular biology London University professor. Patents of four out of the eight hand-made boards were obtained during the early 80s when the empirical studies commenced. The voltage-driven apparatus consists of multiple connections between the eight boards that are made by hand to synthesize and regulate the unlimited resolution complex waveforms that are composed of four thousand frequencies, each having a specific resultant frequency that ranges from 55Hz to 888Hz. At a resistance of 500 Ω, the maximum voltage is 15V, increasing to 25V at 2000 Ω, and 50V at 10K Ω. Any current generated by the voltage, based on Ohm's law, is minuscule and cannot be directly measured. The technology is classified as IEC class I according to the IEC60601-1 standard, and it is used with 3-pin din and 4-pin din IEC 60601-1 compliant cables and silver threaded self-adhesive pads that have been awarded their own FDA clearance. The technology has a CE marketing directive of Class I, with electromagnetic compatibility regulations applied standards EN50081-1, and EN50082-1. It complies with the EEC UK directive of electrical equipment safety applied standard EN 60601-1. The general design of this technology has had no known side effects, in the past 20 years that it has been used in clinical practice by over 5,430 physicians, aesthetic practitioners and private users. The only contraindication, according to the FDA, is having an implanted device like a pacemaker. The main caution is pregnancy. All major medical and mental disorders require clearance by the patient's physician. Adverse reactions are limited to temporary skin redness from the gel pads, that occurs sporadically and usually dissipates within a few hours. Earlier versions of this technology based on the same electronic design have FDA clearance numbers K132158 and K132179. Measuring instruments included: 1) a blood test that measured Free T3, CRP, triglycerides, HDL, fasting and PP glucose and insulin levels; 2) a conductance scale that calculated BMI, overall fat, visceral fat, and skeletal muscle mass (SMM). 3) Before and after treatments fatty liver results on the sonography reports of 11 diabetic subjects.

Procedure

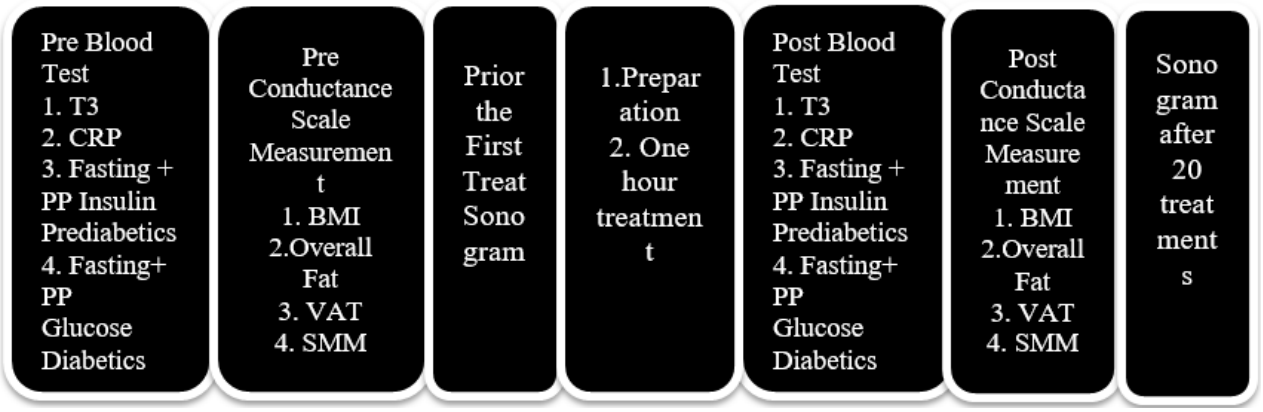
A total of twenty-one Diabetic and 20 Prediabetic obese individuals, 15-82 years of age, with an average BMI of 36.9 consented to release their records. These included eleven diabetic females, ten diabetic males, ten prediabetic females and ten diabetic males. Eleven diabetic subjects, nine females and two males were also diagnosed with fatty liver on their sonography reports. All subjects had completed 20 treatments with the London University technology before the study commenced. Since the study was based on the chart results of all participants, there was no subject attrition. The current research project fulfils the double-blind standards since neither the subjects nor the operators of the technology knew at the time of the treatments' administration that these results were going to be used in a clinical trial. The subjects were made aware of this clinical research only after they had completed all 20 treatments and were asked to sign a consent form. Subjects were randomly selected out of four different clinics on the basis of the following inclusion and exclusion criteria: Inclusion: 1) Overweight or obese; 2) BMI > 29; 3) Age above 12 years old; 4) At least three months after a surgery procedure; 5) At least three months after childbirth; 6) Diabetes; 7) Prediabetes; 8) Had completed 20 treatments with the London University Technology; 9) Had received the treatment at least twice or three times weekly. Exclusion: 1) Pregnancy or trying to get pregnant; 2) An implanted device like a cardiac pacemaker; 3) Severe medical condition other than Diabetes or Prediabetes; 4) Hepatic cirrhosis; 6) Renal failure; 7) Surgery or childbirth less than three months prior to treatment; 8) Cancer; 9) Hernia; 10) Other severe medical or mental condition; 11) Had not received any additional treatments with lasers, radiofrequency, any other slimming devices or any technologies similar to the London University Technology.

Inclusion and exclusion criteria were verified by a certified physician in each of the four clinics. As part of each clinic's general policy, a physician was routinely available during the entire duration of the twenty treatment packages, to ensure the comfort and safety of the participants. All subjects reportedly underwent the treatment with no adverse reactions or side effects. Every precaution was taken to protect the subjects' privacy and the confidentiality of their personal information. Subjects were informed that they had the right to discontinue treatment at any time. Subjects were not in a dependent relationship with the technology operators, the lab and measurement technicians, or the authors. The subjects were given some general diet instructions like increasing their vegetables, lean protein, and fruit intake while reducing sugar and oily foods. However, there was no structured measure of calculating daily caloric intake or the veracity of their statements regarding their eating habits. Subjects were instructed to continue taking their prescribed medications and follow the guidance and recommendations of the physician in charge of their medical status. Subjects were specifically told that the treatment they were receiving was intended as a weight loss/ fitness enhancement to potentially jump-start a healthier lifestyle, and it was not meant to replace exercise or treat their diabetic condition. None of the subjects had a history of exercising or an active lifestyle or was engaged in a regular exercise regimen.

Four independent labs with no personal interest in the direction of the results, one from each of the four participating clinics, were assigned to take blood samples before and after the completion of twenty-one-hour treatments that took place two to three times weekly, for a total of seven to eight weeks. Subjects were asked to fast for twelve hours prior to their blood tests. The conductance scale measurements were performed by technicians who obtained a printout of the results that were included in the chart and were subsequently

used in the study. Eleven subjects offered their sonography results before and after the twenty treatments, but without releasing the full sonography report. Only 27 out of the 41 subjects had measurements from the same conductance scale for BMI, Overall Fat, Visceral Adipose Tissue (VAT) and Skeletal Muscle Mass (SMM). Only twenty of the subjects offered measurements on their Free T3 and CRP levels, ten of which subjects were diabetics and 10 prediabetics.

Pre and post 20 treatments testing variables and procedure



Following blood tests and measurements, each subject went to a private treatment room and lay on a massage table, where the self-adhesive silver-threaded gel pads and silver-plated microphone cables from the 16 channels of the electronic apparatus were attached to his / her body by the operator. The cables from ten of the channels were attached to the gel pads of the buttocks and the abdomen, and the cables from the six remaining channels were attached to the gel pads placed along the lymphatic system pathways of the legs and arms. According to each clinic's policy, the technology operator constantly checked if the subject was comfortable during the entire procedure. All subjects gave a detailed report of their subjective experience during and after the treatments when their overall health status was reassessed. The procedure was in accordance with the ethical standards and principles for medical research involving human subjects.

Results

Statistical analysis was based on a repeated measures design where subjects' results after the twenty treatments were compared to their baseline. Table 1 displays the results of the twenty-one diabetic subjects on pre and post-treatment fasting glucose and postprandial (PP) blood glucose levels. Both fasting and postprandial glucose levels decreased in 100% of the subjects in an average percentage that reached -38.44% decreases for fasting glucose, and -39.1% for postprandial glucose.

Table 2 displays the results of the twenty prediabetic subjects on pre and post-treatment fasting insulin and postprandial (PP) insulin levels. Both fasting and postprandial insulin levels decreased in 100% of the subject in an average percentage decrease that reached -54.53% for fasting insulin, and -44.7% for postprandial insulin.

Table 3 offers the results of the and pre and post sonography reports on the eleven diabetic subjects' fatty liver that indicates no fatty liver after the 20 treatments. Additionally, table 3 displays the results on the triglycerides and HDL levels of all twenty one diabetic subjects. All diabetic subjects (100% of diabetics) evidenced an average of -28.56% decrease in triglycerides and an average of +49.12% increase in HDL.

Table 4 depicts the results on the triglycerides and HDL levels of the twenty prediabetic subjects. All prediabetic subjects' triglycerides (100% of the prediabetics) indicated a reduction in triglycerides at an average of -22.88% from what it used to be previously, and an average increase of +30.34% in blood plasma HDL.

Table 5 gives the results of the pre and post-blood levels of Free T3 and C Reactive Protein of ten diabetic and 10 prediabetic subjects. All diabetic and prediabetic subjects (100% of diabetics and 100% of prediabetics) demonstrated an average increase of 40.78% in Free T3 levels, and an average decrease of -37.88% in blood CRP.

Table 6 gives the results on the pre and post BMI, overall fat, visceral fat and skeletal muscle mass (SMM) of twenty seven out of the 41 subjects that were measured with the same conductance scale.

Table 7 gives the results of t-tests on all variables. The pre and post-comparison of all variables demonstrated a highly significant statistical difference at the $p<0.00001$ (one in one hundred thousand shall not entertain such result) level, except for the fasting insulin of prediabetics that was significant at the $0<0.0001$ (one in ten thousand) lever, and the PP insulin of prediabetics that was significant at the $p<0.001$ (one in one thousand level).

Table 1 Type 2 diabetics Pre and post treatment results on blood glucose (fasting and pp)

No	Gender	Age	Medical diagnosis	Blood glucose fasting mg./dL Pre	Blood glucose fasting mg/dL Post	Blood glucose normal <100 mg/dL	Blood glucose pp mg/dl pre	Blood glucose pp mg/dl post	Blood glucose pp normal < 140 mg/dl
1	Female	45y	Diabetes fatty liver	178	104	Prediabetic	260	185	Prediabetic
2	Male	69y	Diabetes	209	108	Prediabetic	230	125	Normal
3	Male	46y	Diabetes	131.7	99.15	Normal	290	183.2	Prediabetic
4	Female	50y	Diabetes	177	106	Prediabetic	221	176	Prediabetic
5	Female	49y	Diabetes fatty liver	192	102	Prediabetic	248	175	Prediabetic

Table 1 Continued...

No	Gender	Age	Medical diagnosis	Blood glucose fasting mg./dL Pre	Blood glucose fasting mg/dL Post	Blood glucose normal <100 mg/dL	Blood glucose pp mg/dl pre	Blood glucose pp mg/dl post	Blood glucose pp normal < 140 mg/dl
6	Female	48y	Diabetes fatty liver	189	115	Prediabetic	224	163	Prediabetic
7	Male	44y	Diabetes fatty liver	178	109	Prediabetic	196	162	Prediabetic
8	Female	45y	Diabetes fatty liver	186	117	Prediabetic	197	126	Normal
9	Female	47y	Diabetes fatty liver	169	102	Prediabetic	243	178	Prediabetic
10	Male	45y	Diabetes	135	92	Normal	218	156	Prediabetic
11	Male	82y	Diabetes	136	87	Normal	191	142	Prediabetic
12	Male	46y	Diabetes	134	97	Normal	216.3	139	Normal
13	Male	59y	Diabetes	106.8	82	Normal	199.9	133	Normal
14	Female	45y	Diabetes fatty liver	186	117	Prediabetic	207.5	123	Normal
15	Male	59y	Diabetes	188	119	Prediabetic	202	133	Prediabetic
16	Male	49y	Diabetes	141	99	Normal	125.6	144	Prediabetic
17	Female	69y	Diabetes fatty liver	136	87	Normal	231.4	131	Normal
18	Female	53y	Diabetes	190	108.5	Prediabetic	212	118	Normal
19	Female	68y	Diabetes Fatty Liver	176	92	Normal	209.8	98	Normal
20	Female	61y	Diabetes Fatty Liver	157.5	98.5	Normal	204	103	Normal
21	Male	55y	Diabetes Fatty Liver	194	107	Prediabetic	231	138	Normal
Total				**3490**	**2148..15**		**4557.5**	**3031.2**	
Average				**166.19**	**102.29**	**Normal**	**237.02**	**144.34**	**Normal**
Percentage of blood glucose decrease				**Fasting blood glucose% Decrease**	**-38.44%**		**PP Blood glucose% Decrease**	**-39.10%**	

Fasting blood glucose: normal <100 mg/dl; prediabetes = 100 - 125 mg/dl; diabetes >126 mg/dl

Blood glucose postglandial (pp): normal < 140 mg/dl; prediabetes = 140 - 199 mg/dl; diabetes > 199 mg/dl

Table 2 Prediabetics Pre and post treatment results on insulin (fasting and PP)

No	Gender	Age	Medical Diagnosis	Insulin Fasting mIU/ml Pre	Insulin Fasting mIU/ml Post	Insulin Fasting Normal< 25 mIU/ml	Insulin PP mIU/ml	Insulin PP mIU/ml Post	Insulin PP Normal <75 mIU/ml
1	Female	43y	Prediabetes	72	15.7	Normal	174.3	73.9	Normal
2	Female	27y	Prediabetes	25.8	8.7	Normal	136	74	Normal
3	Female	63y	Prediabetes	105	12.27	Normal	150	76.2	Normal
4	Female	24y	Prediabetes	34	21	Normal	139.9	71.8	Normal
5	Female	30y	Prediabetes	27.4	18.5	Normal	241	24.6	Normal
6	Male	15y	Prediabetes	29	10.9	Normal	136.6	74.8	Normal
7	Male	58y	Prediabetes	50.4	24	Normal	246	68.4	Normal
8	Male	46y	Prediabetes	25.56	12.56	Normal	68.8	23.5	Normal
9	Female	39y	Prediabetes	48	24.9	Normal	69.7	72	Normal
10	Male	40y	Prediabetes	22.2	11.8	Normal	127.2	73.4	Normal
11	Male	53y	Prediabetes	23.8	14.6	Normal	102.8	96.8	Prediabetes
12	Male	39y	Prediabetes	19.5	14.6	Normal	103.9	68.8	Normal
13	Male	31y	Prediabetes	43.5	22.8	Normal	116.3	73.4	Normal
14	Female	33	Prediabetes	41.9	18.6	Normal	109.3	68.4	Normal
15	Male	49y	Prediabetes	53.7	24.8	Normal	126.4	73.8	Normal
16	Male	69y	Prediabetes	35.8	27.4	Prediabetic	112.4	83.74	Prediabetic
17	Male	53y	Prediabetes	42.7	23.12	Normal	93.4	71.6	Normal
18	Female	68y	Prediabetes	53.6	28.9	Prediabetic	77.2	70.65	Normal
19	Female	49y	Prediabetes	42.8	23.4	Normal	81.4	72.5	Normal

Table 2 Continued...

No	Gender	Age	Medical Diagnosis	Insulin Fasting mIU/ml Pre	Insulin Fasting mIU/ml Post	Insulin Fasting Normal< 25 mIU/ml	Insulin PP mIU/ml	Insulin PP mIU/ml Post	Insulin PP Normal <75 mIU/ml
20	Female	52y	Prediabetes	39.8	21.7	Normal	76.8	64.3	Normal
Total				**836.46**	**380.25**		**2489.4**	**1376.59**	
Average				**41.823**	**19.02**	**Normal**	**124.47**	**68.83**	**Normal**
Percentage of insulin decrease				**Fasting insulin % decrease**	**-54.52%**		**PP insulin % decrease**	**-44.70%**	

Insulin Fasting: Normal < 25 mIU/ml Insulin Postprandial (PP): Normal <75

Table 3 Type 2 diabetics Triglycerides, high-density lipoprotein (HDL), presence of fatty liver on sonography reports pre and post treatment

No	Gender	Age	Medical Diagnosis Pre Treatment	Fatty Liver Post on Sonography Reports	Triglycerides mg/dL Pre	Triglycerides mg/dL Post	Triglycerides mg/dL decrease	HDL mg/dL Pre	HDL mg/dL Post	(HDL) mg/d Increase
1	Female	45y	Diabetes Fatty liver	No fatty liver	203	158	Improved(abnormal)	32	39	Improved at risk
2	Female	46y	Diabetes Fatty Liver	No fatty liver	287	176	Improved(abnormal)	32	39	Improved at risk
3	Female	48y	Diabetes Fatty Liver	No fatty liver	266	147	Normal	29	41	Improved at risk
4	Male	44y	Diabetes Fatty Liver	No fatty liver	283	189	Improved(abnormal)	30	35	Improved at risk
5	Female	45y	Diabetes Fatty Liver	No fatty liver	225	179	I Improved(abnormal)	33	40	Improved at risk
6	Female	47y	Diabetes Fatty Liver	No fatty liver	237	188	Improved(abnormal)	31	41	Improved at risk
7	Female	45y	Diabetes Fatty Liver	No fatty liver	228	134	Normal	34	58	Normal
8	Female	45y	Diabetes Fatty Liver	No fatty liver	214	138	Normal	28	51	Normal
9	Female	68y	Diabetes Fatty Liver	No fatty liver	198	122	Normal	31	59	Normal
10	Female	61y	Diabetes Fatty Liver	No fatty liver	219	112	Normal	28	52	Normal
11	Male	55y	Diabetes Fatty Liver	No fatty liver	223	106	Normal	24	66	Normal
12	Male	69y	Diabetes		215	158	Normal	35	47	Improved at risk
13	Male	46y	Diabetes		230	176	Improved(abnormal)	28	37	Improved at risk
14	Female	52y	Diabetes		196.7	147	Normal	47.6	53	Normal
15	Female	49y	Diabetes		193	189	Normal	34.5	38	Improved at risk
16	Male	45y	Diabetes		212	179	Normal	41	45	Improved at risk
17	Male	72y	Diabetes		197	188	Normal	26	38	Improved at risk
18	Male	59y	Diabetes		202	134	Normal	31	62	Normal
19	Male	49y	Diabetes		197	138	Normal	44	71	Normal
20	Male	57y	Diabetes		192	122	Normal	37	61	Normal
21	Male	55y	Diabetes		199	112	Normal	42	68	Normal
Total					**4616.7**	**3298**		**698.1**	**1041**	
Average					**219.84 HIGH**	**157.04 Improved**	**Improved**	**33.24 low**	**49.57 Improved**	**Improved**
% of triglycerides decrease						**-28.56%**	**% OF HDL increase**		**49.12%**	

Triglycerides Normal Range: > 150 mg/dL;

High-Density Lipoprotein (HDL) Normal Range: Men >60 mg/dL; Women >60 mg/dL

High-Density Lipoprotein (HDL) At Risk: Men: < 40 mg/dL; Women < 50 mg/dL

Table 4 Prediabetics Triglycerides, High-Density lipoprotein (HDL), presence of Fatty Liver on Sonography Reports Pre and Post Treatment

No	Gender	Age	Medical Diagnosis Pre Treatment	Triglycerides mg/dL Pre	Triglycerides mg/dL Post	Triglycerides mg/dL decrease	HDL mg/dL Pre	HDL mg/dL Post	HDL mg/dL Increase
1	Female	43y	Prediabetes	294	197	Improved(abnormal)	36	42	At risk
2	Female	27y	Prediabetes	192	126	Normal	36	48	At risk
3	Female	63y	Prediabetes	155	117	Normal	45	47	At risk
4	Female	24y	Prediabetes	88	86	Normal	45	52	Normal
5	Female	30y	Prediabetes	156	124	Normal	37	46	At risk
6	Male	15y	Prediabetes	187	132	Normal	36	42	Normal
7	Male	58y	Prediabetes	141	136	Normal	39.1	46.8	Normal
8	Male	46y	Prediabetes	262	158	Improved(abnormal)	34.3	56	Normal
9	Female	24y	Prediabetes	186	148	Normal	41	58	Normal
10	Male	40y	Prediabetes	178	137.6	Normal	34.8	45.4	Normal
11	Male	50y	Prediabetes	169	142.8	Normal	34.7	43	Normal
12	Male	39y	Prediabetes	172	139.2	Normal	29.6	48.8	Normal
13	Male	31y	Prediabetes	159	122.4	Normal	26.6	53.4	Normal
14	Female	33	Prediabetes	163.6	134.8	Normal	39.3	67.2	Normal
15	Male	49y	Prediabetes	158.9	128.3	Normal	34.7	53.1	Normal
16	Male	69y	Prediabetes	184.6	148.9	Normal	29.4	54	Normal
17	Male	53y	Prediabetes	176	146.8	Normal	39.2	51.6	Normal
18	Female	68y	Prediabetes	154.7	129.6	Normal	47.2	58.5	Normal
19	Female	49y	Prediabetes	154.6	121.7	Normal	47.4	52.5	Normal
20	Female	52y	Prediabetes	189	138.5	Normal	46.2	57.9	Normal
Total				**3520.4**	**2714.6**		**785.5**	**1023.2**	
Average				**176.02 high**	**135.73 Normal**		**39.25 low**	**51.16 Normal**	
Average decrease in Triglycerides					**-22.88**	**Average Increase in HDL**		**30.34**	

Triglycerides Normal Range: > 150 mg/dL;

High-Density Lipoprotein (HDL) Normal Range: Men >60 mg/dL; Women >60 mg/dL

High-Density Lipoprotein (HDL) At Risk: Men: < 40 mg/dL; Women < 50 mg/dL

Table 5 Free T3 (triiodothyronine) and CRP (C-Reactive Protein)

Subject no from Table 1 diabetes	Gender	Age	Medical Condition	Free T3 pre pg/mL	Free T3 post pg/mL	Free T3 Normal Range pg/mL	CRP pre mg/dL	CRP post mg/dL	Normal Range mg/dL
12	Male	46y	Diabetes	1.99	2.69	2.30-4.20	1.45	1.05	<1.00
13	Male	59y	Diabetes	1.92	2.78	2.30-4.20	1.29	1.08	<1.00
14	Female	45y	Diabetes Fatty Liver	2.12	2.55	2.30-4.20	2.51	1.25	<1.00
15	Male	59y	Diabetes	1.97	2.62	2.30-4.20	1.83	0.96	<1.00
16	Male	49y	Diabetes	1.18	2.29	2.30-4.20	1.13	0.91	<1.00
17	Female	69y	Diabetes Fatty Liver	1.43	2.42	2.30-4.20	1.67	1.01	<1.00
18	Female	53y	Diabetes	1.63	2.15	2.30-4.20	1.09	0.86	<1.00
19	Female	68y	Diabetes Fatty Liver	1.93	2.88	2.30-4.20	1.18	0.84	<1.00
20	Female	61y	Diabetes Fatty Liver	2.23	2.37	2.30-4.20	1.94	0.95	<1.00
21	Male	55y	Diabetes	1.47	2.26	2.30-4.20	2.23	1.03	<1.00
Subject no from Table 2 prediabetes									
14	Female	33	Prediabetes	2.25	2.77	2.30-4.20	1.09	0.76	<1.00
15	Male	49y	Prediabetes	2.22	2.58	2.30-4.20	1.59	1.05	<1.00
16	Male	69y	Prediabetes	1.68	2.51	2.30-4.20	1.19	1.02	<1.00
17	Male	53y	Prediabetes	1.99	2.89	2.30-4.20	2.42	1.25	<1.00

Table 5 Continued...

Subject no from Table 2 prediabetes									
18	Female	68y	Prediabetes	1.28	2.25	2.30-4.20	1.98	0.99	<1.00
19	Female	49y	Prediabetes	1.43	2.36	2.30-4.20	1.52	1.14	<1.00
20	Female	52y	Prediabetes	1.53	2.14	2.30-4.20	1.75	1.03	<1.00
14	Female	33	Prediabetes	1.97	2.78	2.30-4.20	1.08	0.89	<1.00
				32.22	45.29		28.94	18.07	
Average Free T3 Pre & Post				**1.79 below Normal**	**2.52 Normal**	**Average CRP Pre & Post**	**1.61 below Normal**	**1.00 Improved**	
Free T3 Percentage Increase					**40.78%**	**Average CRP Percentage Decrease**		**-37.88%**	

Free T3 Normal Range: 2:30-4.20 pg/mL. CRP Normal Range <1 mg/dL

Table 6 Pre and Post Treatment Results on BMI, Overall Fat, Visceral Fat, and Skeletal Muscle Mass (SMM)

S #	**Gender**	**Age**	**Medical condition**	**BMI Pre**	**BMI Post**	**Overall Fat Pre**	**Overall Fat Post**	**Visceral Fat Pre**	**Visceral Fat Post**	**SMM Pre**	**SMM Post**
1	Female	46	Diabetes Fatty Liver	39.2	36.2	44.6	36.8	35	24.8	22.1	29.4
2	Female	48	Diabetes Fatty Liver	41.2	38.5	42.9	33.5	33	29	23.8	29.7
3	Male	44	Diabetes Fatty Liver	42.6	38.2	34.9	24.6	29	26	34.5	47.3
4	Female	48	Diabetes Fatty Liver	32	30.1	42.9	33.5	29	24	23.8	31.8
5	Female	45	Diabetes Fatty Liver	29.1	25.1	34	28.7	31	27	20.7	26.3
6	Female	24	Prediabetes	29.3	25	34.7	33	9.5	5	21.8	24.2
7	Male	40	Prediabetes	33.7	25.1	33	13.4	21	13.4	28.8	31.2
8	Male	39	Prediabetes	36.2	32	41.1	37.4	18	14.5	36	38.9
9	Male	31	Prediabetes	43.8	39.1	37.6	34.6	30	25	25.2	27.4
10	Male	46	Diabetes	39.2	24.6	42.3	25.6	24.7	10.8	28.9	39.4
11	Male	59	Diabetes	36.5	28.9	37.9	31.6	32.3	16.4	26	41
12	Female	45	Diabetes Fatty Liver	41.3	27.4	43.8	22.7	39.5	19.4	23.8	38.5
13	Male	59	Diabetes	34.2	24.8	36.9	25.8	35.4	22.8	28.9	41.2
14	Male	49	Diabetes	37.4	29.5	41.3	22.5	29.3	18.3	35.7	42.6
15	Female	69	Diabetes Fatty Liver	42.6	36.8	44.2	37.9	34.6	31.7	27.9	33.2
16	Female	53	Diabetes	33.5	25.1	30.1	25.7	38.2	30.1	32.4	39.9
17	Female	68	Diabetes Fatty Liver	40.7	36.1	42.3	39.8	37.4	33.8	30.2	39.7
18	Female	61	Diabetes Fatty Liver	34.2	25.3	36.7	33.2	38	36.1	23.8	28.6
19	Male	55	Diabetes	36.7	26.4	38.7	29.6	33.5	23.2	27.9	39.4
20	Female	33	Prediabetes	36.8	22.5	39.2	21.3	25.3	9.4	32.5	43.2
21	Male	49	Prediabetes	35.9	24.6	39.4	18.4	24.3	8.5	35.4	48.3
22	Male	69	Prediabetes	38.2	33.7	39.6	31.5	28.3	24.6	31.4	37.8
23	Male	53	Prediabetes	37.2	30.3	40.2	29.3	36.2	30.6	29.3	36.7
24	Female	68	Prediabetes	35.7	29.4	33.6	31.4	37.3	32.9	30.8	34.2
25	Female	49	Prediabetes	35.3	25.4	37.4	21.5	27.6	10.8	38.9	47.2
26	Female	52	Prediabetes	36.1	29.6	36.5	28.3	29.7	25.3	37.5	41.3
27	Female	37	Prediabetes	39.2	23.9	47.3	24.1	28.4	12.3	24.6	42.8
Total				**997.8**	**793.6**	**1013.5**	**775.7**	**815.5**	**585.7**	**782.6**	**1001.2**
Mean average				**36.9**	**29.4**	**38.9**	**28.73**	**30.2**	**21.69**	**28.98**	**37.1**
Mean overall BMI decrease: -7.5					**Mean average overall fat decrease %-26.14%**		**Mean visceral fat decrease %-28.17%**		**Mean SMM % increase+28.02%**		

Table 7 T-test Statistical Significance

	Mean	SS/df	T-value	P-value	Significance level
Blood glucose fasting mg./dl diabetics decrease	-63.9	414.64	-14.38	P < 0.00001	P < 0.00001
Blood glucose pp mg/dl diabetics decrease	-72.68	891.07	-11.16	P < 0.00001	P < 0.00001
Insulin fasting miu/ml prediabetis decrease	-22.8	390.6	-5.16	P < 0.0003	P < 0.0001
Insulin PP miu/ml prediabetics decrease	-55.64	3071.35	-4.49	P< 0.00013	P< 0.001
Triglycerides mg/dl diabetics decrease	-67.84	1056.27	-9.57	P < 0.00001	P < 0.00001
HDL mg/dL diabetics increase	16.33	120.72	6.81	P < 0.00001	P < 0.00001
Triglycerides mg/dL prediabetics decrease	-40.29	630.05	-7.18	P < 0.00001	P < 0.00001
HDL mg/dL prediabetics increase	13.24	57.92	7.78	P < 0.00001	P < 0.00001
Free T3 Increase	0.73	0.07	12.06	P < 0.00001	P < 0.00001
CRP decrease	-0.6	0.15	-6.64	P < 0.00001	P < 0.00001
BMI decrease	-7.56	14.72	-10.24	P < 0.00001	P < 0.00001
Overall fat decrease	-10.27	43.89	-8.06	P < 0.00001	P < 0.00001
Overall visceral fat decrease	-8.51	30.14	-8.06	P < 0.00001	P < 0.00001
Skeletal muscle mass increase	8.1	18.66	9.74	P< 0.00001	P< 0.00001

Discussion

Several physicians treating diabetics recommend exercise and physical activity to either prevent the diabetic condition or avoid further complications via enhancing health and fitness. These recommendations are based on a large body of research. There are numerous problems with this notion, however. a/ Obesity makes physical training cumbersome; b/ Diabetic neuropathy increases fragility and resistance to movement; c/ Clinical studies have demonstrated that certain modes of exercise may induce temporary hyperglycaemia and hyperinsulinemia in diabetics. A novel method from London University offers a solution between inertia and activity, an effortless exercise technique that can balance some of the diabetic metabolic issues and jump-start a more active lifestyle.

The results of our research achieved external validity of all variables by confirming previous findings.[56-61] We demonstrated a statistically significant improvement in T3 levels for all subjects (100%). T3 was elevated to the normal range in 14 out of 20 subjects, indicating that 70% of subjects reached normalcy after 20 treatments. Additionally, there was a statistically significant decrease of CRP in 100% of the subjects, implying a notable reduction in low-grade inflammation. Despite the prominent improvement evidenced in all subjects, only eight out of the 20 subjects with previously abnormally high CRP attained normalcy after 20 treatments (40% of the subjects).

Both previously abnormally high fasting and postprandial (PP) glucose decreased considerably in all 21 diabetic subjects (100%). Nine of the diabetic subjects (42.85%) manifested normal fasting glucose levels after 20 treatments, while the fasting glucose of the remaining twelve diabetic subjects (57.2%) dropped down to the prediabetic level. Ten of the diabetic subjects (47.6%) manifested normal PP insulin levels, while the PP insulin of the remaining eleven diabetic subjects (52.38%) dropped to the prediabetic level after the 20 treatments. Prediabetics had more robust results as expected by their average younger age and baseline healthier status. Eighteen of prediabetics (90%) manifested both normal fasting and PP insulin levels after the 20 treatments, while the fasting and PP insulin of the remaining two subjects (10%) remained within the prediabetic level. Triglycerides decreased in all 21 diabetic subjects (100%) juxtaposed by a consistent elevation in HDL. Despite the statistically significant improvement, the decrease and increase of Triglycerides and HDL respectively, did not reach normalcy for all subjects. Fifteen out of the 21 diabetics with abnormal triglyceride levels displayed normal triglyceride levels after twenty treatments (71.4%). Only nine of these diabetic subjects (42.9%) indicated HDL levels that were within the normal range. Eighteen prediabetic subjects (90%) manifested normal triglyceride levels and 85% of prediabetics demonstrated HDL levels within the normal range. Skeletal muscle mass increased by an average of 28.2% in all subjects, while all subjects indicated an overall and visceral fat reduction at an average of 26.14% and 28.17% respectively. The visceral fat reduction was substantiated by the sonography reports of eleven diabetic subjects that showed no fatty liver after the twenty treatments.

Overall, results indicated a remarkable improvement in the diabetic/prediabetic condition. This improvement was predicted by a large body of literature documenting that enhancing T3 and fitness results in a decrease in dyslipidaemia. A literature search reveals that the deleterious effects of inflammation marked by abnormally high CRP levels are also counteracted by an active lifestyle or effortless exercise that has repeatedly demonstrated a CRP reduction. A number of studies using either regular or effortless exercise have displayed a significant decrease in both fasting and PP glucose and insulin. Research has repeatedly shown that a decrease in overall and visceral fat improves diabetic and prediabetic conditions.

Conclusion

Our findings support and validate the results of previous studies that some mode of exercise is necessary to enhance the health status of diabetic and prediabetic conditions. The scope of our study was to offer an intermediate solution that can potentially commence a healthier lifestyle, but without implying or proposing that this novel method is a medical intervention or a conclusive treatment for Diabetes. All patients were instructed to continue taking their medications and remain under their physicians' care. Upon thorough examination of the results, it became apparent that the resistance to attaining normalcy was contingent on disease severity and age. A higher percentage of prediabetics when compared to diabetics reached normalcy in all variables. The majority of diabetics denoted a substantial improvement without reaching the optimal level of health. This suggested the necessity of continuing with a lifestyle that includes fitness attained by regular or effortless exercise, in conjunction with the recommended medical treatments. To speed up weight loss, a structured nutritional plan may be useful. The sonograph reports evidencing no fatty liver after 20 treatments validated the results of one of our previous studies that used the same method. However, the

subjects' number in both studies was rather small, therefore, we are looking forward to conducting more studies that examine visceral fat deposits and fatty liver by using sonography or magnetic resonance imaging diagnostic methods with a larger number of subjects.

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Conflicts of interest

The author declares no conflict of interest. All treatments were performed by operators without the direct presence or hands-on supervision of any of the authors.

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