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The dangerous affinity between obesity and covid-19

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Abstract
The objective was to identify some of the common denominators that undermine health and increase COVID-19 vulnerability. The virus has a binding affinity for ACE2 receptors which are abundant in adipose tissue, placing obesity at the spotlight of the COVID-19 susceptibility factors. Obese individuals manifest respiratory problems that elevate viral mortality rates. Visceral adiposity progressively increases with age, often accompanied by inflammation, high VLDL, triglycerides, Free T3 abnormalities, low testosterone, and exacerbated cortisol. Disturbances in the anorexic and orexigenic hormones leptin and ghrelin respectively, lead to weight gain, rendering physical activity challenging and cumbersome. The lack of exercise compromises health and immunity. Toxicity is enhanced by inactivity, while moderate exercise promotes cardiorespiratory fitness (CRF), safeguarding against most chronic diseases. However, excessive exercise results in an inverse cortisol/testosterone relationship leading to hormonal imbalance. In search of solutions to proactively protect public health, we conducted a randomized double-blind clinical trial on ten overweight menopausal women with medical issues including diabetes and hyperphagia, to investigate the possibility of weight reduction and increased appetite regulation. Results delineated a statistically significant decrease in BMI, visceral adipose tissue, VLDL, and triglycerides, an inverse relationship between both testosterone/cortisol and leptin/ghrelin, and reduced hyperphagia. Testosterone and leptin, along with Free T3 and IGF-1 climbed towards the peak of the normal range, juxtaposed by cortisol and ghrelin that decreased but without regressing outside normalcy.

Keywords: covid-19, VLDL cholesterol, Triglycerides, Visceral Adipose Tissue, Diabetes, Hyperphagia, Testosterone, Cortisol, Free T3, Ghrelin, Leptin, IGF-1, Skeletal muscle

Introduction

Coronavirus disease (COVID-19) is an infectious disease affecting the lower respiratory track. COVID-19 death rate by pre-existing conditions is 10.5% for cardiovascular disease (CVD), 7.3% for diabetes, 6.3% for chronic respiratory disease, 6.3% cancer, and 0.9% for patients with no pre-existing conditions [1]. According to Italy's National Institute of Health in March 2020, 99% of COVID-19 patients had at least one pre-existing condition and one-third of them had heart disease [2, 3]. Increased levels of the very-low-density lipoprotein (VLDL) was associated with a high risk of CVD due to increased inflammation, hardened plaque that narrows the arteries, and increased blood pressure. A 15-year multi-provincial cohort study found that high VLDL cholesterol was an independent risk factor of coronary heart disease (CHD) that elevated CVD risk [4]. Increased VLDL and triglyceride concentrations have been confirmed in both Type 1 and Type 2 diabetes mellitus [5]. To date, no research studies have connected high levels of VLDL and triglycerides with COVID-19 patients. Research has indicated that primary infection patients showed lower high-density lipoprotein (HDL) levels (1.10±0.04 mmol/L) when compared to secondary infection patients, however, this study did not specifically look at VLDL or triglycerides [6]. Increased levels of VLDL and triglycerides are also associated with obesity. Recent reports suggest that a high percentage of the population who will contract COVID-19 will be overweight or obese with a BMI over 25 [7]. Research on 4,000 patients in New York City found that after age, obesity was one of the most significant factors associated with poor prognosis and case severity of COVID-19 hospitalized individuals [8]. Early statistics from Britain's independent Intensive Care National Audit and Research Centre on COVID-19 patients in intensive care confirm that 73.4% of these patients are classified as overweight [9]. Abdominal obesity is also associated with respiratory problems which contribute significantly to the burden of respiratory disease [10].

COVID-19 binds to target cells through angiotensin-converting enzyme-2 (ACE2) receptors therefore, hypertension and diabetes patients who may have increased ACE2 expression may be more vulnerable to COVID-19 infection, ultimately increasing the risk of fatality [11, 12].

Zhou et al., [13] examined the clinical course and risk factors for mortality of Adults Inpatients with COVID-19 in Wuhan and found that mortality rate was higher in aged patients with diabetes or coronary heart disease as a result of increased inflammation, toxicity and immune deficiency. These patients demonstrated increased concentrations of alanine aminotransferase (ALT), and interleukin 6 proteins (IL-6) suggesting upraised inflammation. Their immune systems appeared compromised as evidenced by lymphopenia, the abnormally low level of lymphocytes in the blood, and leukocytosis that reflects a higher leukocyte count of white cells. Elevated creatinine, a waste product produced by the muscles, ALT, high LDH-4, and LDH-5, signified liver or muscle tissue damage suggesting excess toxicity [14]. Exploration of a number of variables associated with COVID-19 patients suggested that mortality may be due to virally driven overproduction of ferritin, IL-6, and interferon-γ inducible protein 10 resulting in hyper inflammation [15]. The tenacious antiviral activity of

interferons consequently shuts down host protein synthesis inducing cell death [16]. The exacerbated symptomatology and eventual fatality of COVID-19 patients is not due to "viral load" but an overzealous immune reaction, releasing an overwhelming multitude of cytokines into the blood that indiscriminately attack both the virus and the vital organs that contain it [17]. Diabetes may make COVID-19 worse because inadequate amounts of insulin result in high glucose concentrations in the blood, facilitating the virus' tendency to increase glucose metabolism that reinforces the eventually lethal "cytokine storm" [18]. Diabetics have impaired immune-response to infection both in relation to their cytokine profile, and to changes in immune-responses including T-cell and macrophage activation [19].

Diabetes has been associated with hypothyroidism, defined by a reduced production of Free T3 [20]. The cardiovascular system is also regulated by thyroid hormones, linking Free T3 insufficiency with CVD [21]. The obesity statues of several CVD and type 2 diabetes patients is often accompanied by exacerbated thyroid abnormalities [22, 23]. Cortisol abnormalities have been associated with both diabetes complications, and heart disease [24] [25, 26]. The recently published list of people considered vulnerable by the UK Chief Medical Officers includes steroids dependent individuals with an adrenal insufficiency, a condition associated with inadequate amounts of steroid hormones, primarily cortisol [27]. Cortisol increase is associated with overeating, weight gain and obesity [28], delineating a vicious circle between cortisol, food consumption, hormonal imbalance, obesity, and the genesis of medical disorders associated with COVID-19.

The elevated cytokines, specifically cytokine-3 triggered by viral infections inhibit the activity of the anorexic hormone leptin, reinforcing weight gain [29]. Suppressed leptin signaling results in visceral adiposity and accumulated toxicity. Sedentary lifestyles with restricted opportunity for lymphatic drainage overload hepatic detoxification systems, promoting insulin and leptin resistance that disorganize central inhibitors and stimulators of appetite. In short, toxicity disrupts the balanced signaling interactions between the anorexic hormone leptin and the orexigenic hormone ghrelin that are necessary for normal appetite, ultimately increasing caloric intake [30, 31]. While leptin elevations within the normal range suppress appetite, excessive leptin is indicative of leptin resistance, a condition correlated with obesity [32, 33]. Leptin resistance develops due to a defect in intracellular signaling and subsequent decreases in leptin transport across the blood–brain barrier (BBB) [34]. The paradox of hyperphagia and hyperleptinemia is quite common in obese patients [35]. Clinical research links CVD with abnormally low plasma leptin that is evidently involved in cardiovascular homeostasis [36]. Apparently, pathology emanates by either leptin deficiency or excess. Leptin plays an important role in cholesterol metabolism by its regulatory action upon cholesteryl esters [37]. Research has shown that stomach cells that produce the appetite stimulating hormone, ghrelin, are more abundant in morbidly obese patients [38].

Efforts to control the COVID-19 pandemic have currently resulted in restricted physical activity (PA). As previously mentioned, sedentary lifestyles suppress lymphatic drainage, allowing the accumulated toxicity to interfere with overall hormonal balance, exacerbating a number of medical conditions known to increase COVID-19 mortality rates, including diabetes, hypertension, CVD and respiratory diseases. There is a statistically significant inverse relationship between cardiorespiratory fitness (CRF) and all-cause mortality ($p<0.05$) [39]. PA enhances detoxification and has been accepted as an effective therapy for chronic diseases directly affecting physical health [40, 41]. Inactivity results in inflammation, insulin resistance and exacerbation of cardiovascular risk factors [42, 43]. Exercise has been shown to improve immunity [44]. Among other several benefits, exercise can reduce the risk of a generalized loss of skeletal muscle mass in older individuals, otherwise known as sarcopenia [45, 46]. However, strenuous training may cause tissue breakdown, due to cortisol overproduction as a result of stressing the body [47]. Cortisol potentially contributes to CVD and type II diabetes by virtue of converting protein to glucose [48]. Overtraining results in a negative cortisol/testosterone relationship where testosterone declines as cortisol increases. Since both low testosterone and elevated cortisol lead to overeating and low energy, the negative correlation between these two hormones may offset the benefits of exercise [49]. Excessive exercise has been associated to inflammation as a result of high amounts of muscle-derived IL-6 being released into the circulation [50].

A recent clinical trial has documented an effortless exercise method that reports a statistically significant reduction in both fasting and PP insulin and glucose that may be useful as both a preventive and a protective measure in safeguarding health [51]. Similar research has reported significant reductions in visceral adiposity, triglycerides, VLDL, cortisol, and ghrelin, juxtaposed against upraises in leptin, Free T3, testosterone and insulin growth factor-1 (IGF-1) that never spiked above or below normalcy [52, 55]. An earlier animal study explored gene expression in fast and slow muscle fiber phenotypes. Adult skeletal muscle reflected an increase of up to 250% in RNA content associated with the repression of the fast, and the activation of the slow myosin heavy chain genes [56].

Methodology

The technology used was based on 29 years empirical electronic and molecular biology research on motor neurons in London University by a team led by Gerald Pollock, one of the co-inventors of the first pacemaker. It is voltage driven, emitting twenty-four complex waveforms, synthesized out of a sequence of four thousand waveform composites on the basis of a proprietary formula that was developed empirically by recording reactions on individual neurons. The result is rhythmically occurring full body musculature contractions, repeated up to one thousand times within one hour, each sustained for 8 seconds, with 2-secs rest time. The technology has a maximum voltage of 25V at 500 Ω, 100V at 10K Ω, and a waveform range from 55 - 880 Hz. Its 16 channels are isolated by separate transformers, and are connected to eight 3-pin din, and eight 4-pin din silver-plated tour-grade microphone cables with gel pads clipped onto their ends, which are attached onto the body. The two waveform control knobs, each with twelve options are controlled manually. Different waveform combinations offer a large variety of sustained contractions, experienced as different types of exercises. The technology is custom-made and hand-made analogue in the UK with no digital components. It is classified as IEC class I according to the IEC60601-1 standard, and has a CE marketing directive of Class I with electromagnetic compatibility

regulations applied standards EN50081-1 and EN50082-1. Earlier versions of this technology with the same hardware design have been cleared by the FDA in 2012 (K132158) and 2013 (K123157) for usage in physical medicine. The only contraindication, according to the FDA, is having an implanted device like a pacemaker, while the main caution is pregnancy. Adverse reactions are limited to temporary skin redness from the pads that occur sporadically and usually dissipate within an hour. A conductance instrument was also used to calculate body mass index (BMI), visceral adipose tissue, and skeletal muscle mass (SMM). Blood tests assessed levels of VLDL, Triglycerides, Free T3, IGF-1, Leptin and Ghrelin.

Procedure

The results of ten postmenopausal women, ages 47-58, with an average BMI of 28.9 and reported hyperphagia, that received effortless exercise treatments in the past year, were collected by four different clinic, after the subjects signed a comprehensive release form. The subjects were randomly selected by each clinics by a nurse that had no prior experience with this particular technology, and no bias or interest in the direction of the results. All subjects completed the clinical trial. Inclusion criteria were:

1. BMI above 25.
2. Sedentary lifestyle.
3. Age above 47 years.
4. Overweight for over five years.
5. Hyperphagia.

Three of the subjects were diagnosed with prediabetes, two of which had hypertension. Two subjects were diagnosed with diabetes under control. None of the subjects suffered from hyperleptinemia. Exclusion criteria were:

1. Pregnancy.
2. Surgery within the past six months.
3. Chronic, serious medical disorder.
4. Medical disorder with severe past episodes.
5. Mental Disorders
6. An implanted device like a cardiac pacemaker.
7. On a strict diet plan or following a specific diet plan.
8. Hyperleptinemia.

Every precaution was taken to protect the subject's privacy and the confidentiality of their personal information. Subjects were informed that they had the right to refuse participation at any time. None of the subjects were in a dependent relationship with the technology operators or the lab and measurement technicians, who had no bias, and no personal interest in the direction of the results. The author did not know any of the subjects, or any of the technicians, and was never involved in any of the subjects' treatments. The subjects did not receive a specific diet or instructions regarding changes in their lifestyles.

Blood samples were analysed by four different labs, each chosen by each of the four different participating clinics. Blood tests and measurements were taken prior to initiating treatment, and one week after 12 one-hour treatments that took place three times weekly, for four consecutive weeks. Following blood tests and measurements, each subject went to their private treatment room and lay on a massage bed, while the 16 cables with the gel pads were attached on to his/ her body by the technology operator. The cables from ten of the channels were attached on to the gel pads of the waist and abdomen, and the cables from the six remaining channels were attached on to the gel pads placed along the lymphatic system pathways of the legs and arms, to enhance lymphatic drainage during treatment. All four clinics had at least one physician available during the course of the twelve treatments, in case a subject had an adverse reaction. No adverse reactions were reported by any of the subjects.

All subjects gave a detailed report of their subjective experience at least two months after their treatments, at the time they signed the consent forms which, for some subjects was nine to ten months after the twelve treatments. The procedure was performed in accordance with the ethical standards and principles for medical research involving human subjects and was approved by the ethical boards of all four clinics.

Results

The data was analyzed with T-tests for two dependent means. Results indicated an inverse testosterone/cortisol relationship where testosterone increased while cortisol declined. This was the opposite of the cortisol/testosterone correlation observed after strenuous physical exercise, where cortisol increases while testosterone decreases, leading to inevitable weight gain. Testosterone increase was demonstrated by all subjects at different levels with an average percentage of +25.90% (Table 1). Cortisol was consistently reduced in all subjects with an average percentage decrease of -13.48% (Table 1). Statistical analysis reflected a significance level of $p<0.001$ for testosterone, and $p<0.01$ for cortisol, which, however, remained within the normal range (Table 6). All subjects evidenced increases in IGF-1 and SMM that were statistically significant: IGF-1 ($p<0.0001$), and SMM ($p<0.00001$) (Table 6). Mean average percentage increase of IGF-1 was +26.68%, which remained within the normal range. Mean average percentage increase for SMM was +26.75% (Table 2). There was a statistically significant decrease of BMI (Tables 2, 6). Triglycerides and VLDL also decreased significantly in all subjects: Significance level was $p<0.0001$ for triglycerides and $p<0.00001$ for VLDL (Table 6). Mean average percentage decrease was -30.28% for triglycerides, and -46.25% for VLDL (Table 3).

T-tests for dependent means showed statistically significant results for both Visceral Adipose Tissue and Free T3 at $p<0.00001$ (Table 6). Average percentage decrease of visceral adipose tissue was -18.20% (Table 4). Average percentage increase of Free T3 was +20.98% (Table 4). There was an inverse relationship between leptin and ghrelin where leptin significantly increased ($p<0.0001$) and ghrelin significantly decreased ($p<0.01$) (Table 6). Leptin increased for all subjects who had reported hyperphagia at an average of +23.41%, while ghrelin decreased at a mean average -8.7% (Table 5).

Importantly, leptin and ghrelin values increased and decreased respectively within the normal range (Table 5).

All subjects reported sustained results, reduced food consumption and reduced cravings after the twelve treatments. They also reported that the treatments had helped them jump-start a more active lifestyle that included a healthier diet.

Table 1: Blood Plasma Subjects' Results on Testosterone and Cortisol for each subject. (Female Normal Ranges for Testosterone: Premenopausal 21-60 yrs 9.01 - 47.94

Gender / AGE	Medical History	Bmi	Testosterone Total Pre ng/dl	Testosterone Total Pre (ng/dL)	Normal Range ng/dL	Testosterone % Increase ng/dL	Cortisol Pre nmol/L	Cortisol Post nmol/L	Cortisolnormal Range Nmol/L	Cortisol% decrease nmol/L
F/ 48	Diabetes Hyperphagia	31.2	9.84	14.88	7.00 - 45.62	+51.21%	227	212	83-359	-6.61%
F/ 54	Diabetes Hyperphagia	30.4	8.68	9.93	7.00 - 45.62	+14.40%	345	297	83-359	-13.91%
F/ 56	Prediabetes Hyperphagia	31.6	14.32	19.64	7.00 - 45.62	+37.15%	466	412	83-359	-11.58%
F/ 47	Hyperphagia	28.7	16.33	21.56	7.00 - 45.62	+32.02%	296	274	83-359	-6.75%
F/ 52	Prediabetes Hypertension Hyperphagia	26.8	11.82	12.93	7.00 - 45.62	+9.39%	358	206	83-359	- 42.48%
F/ 49	Hyperphagia	27.1	18.33	21.49	7.00 - 45.62	+17.23%	423	412	83-359	-2.6%
F/ 58	Prediabetes Hypertension Hyperphagia	29.5	8.12	9.34	7.00 - 45.62	+15.02%	312	276	83-359	-11.53%
F/ 50	Hyperphagia	27.3	10.21	13.28	7.00 - 45.62	+30.06%	311	292	83-359	-6.11%
F/ 55	Prediabetes Hyperphagia	27.1	12.43	15.22	7.00 - 45.62	+22.44%	318	255	83-359	-19.81%
F/ 49	Hyperphagia	29.5	17.43	22.68	7.00 - 45.62	+30.12%	298	258	83-359	-13.42%
			Mean Average Testosterone % Increase		+25.90%	Mean Average Cortisol % Decrease			-13.48%	

Table 2: Blood Plasma Subjects' Results on IGF-1 and Results on Skeletal Muscle Mass (SMM).

Gender / Age	Medical History	BMI	BMI	Igf-1 Pre nmol/L	Igf-1 Post nmol/L	IGF-1 Normal Range nmol/L	IGF-1 % Increase nmol/L	Smm Pre	Smm Post	SMM % Decrease
F/ 48	Diabetes Hyperphagia	31.2	28.7	12.23	14.17	11.25-28.8	+14.86%	12.74	14.66	+15.07%
F/ 54	Diabetes Hyperphagia	30.4	28.2	11.65	12.33	11.25-28.8	+5.83%	11.45	12.95	+13.10%
F/ 56	Prediabetes Hyperphagia	31.6	30.1	11.17	12.79	11.25-28.8	+14.50%	12.66	14.76	+16.58%
F/ 47	Hyperphagia	28.7	25.9	13.94	17.21	11.25-28.8	+23.45%	16.86	19.45	+15.36%
F/ 52	Prediabetes Hypertension Hyperphagia	26.8	25.7	12.27	14.32	11.25-28.8	+7.65%	11.99	14.27	+19.01%
F/ 49	Hyperphagia	27.1	25.4	12.18	14.72	11.25-28.8	+20.85%	12.67	16.59	+30.93%
F/ 58	Prediabetes Hypertension Hyperphagia	29.5	27.2	10.21	11.99	11.25-28.8	+17.43%	11.32	12.60	+11.30%
F/ 50	Hyperphagia	27.3	25.3	12.87	14.36	11.25-28.8	+11.57%	11.04	13.96	+26.45%
F/ 55	Prediabetes Hyperphagia	27.1	24.9	11.43	12.85	11.25-28.8	+12.42%	12.30	13.94	+13.33%
F/ 49	Hyperphagia	29.5	26.5	13.82	15.26	11.25-28.8	+10.41%	12.15	13.93	+14.65%
Average BMI Pre: 29.2		Average BMI Post: 26.75		Mean Average IGF-1 % Increase			+13.90%	Mean Average % Increase for SMM		+17.58%

Table 3: Blood Plasma Results on VLDL and Triglycerides for each subject.

Gender / Age	Medical History	BMI	Triglycerides Pre mmol/L	Trigly Ce Rides Post mmol/L	Normal Range mmol/L	Triglyce Rides % Decrease mmol/L	Vldl Pre mmol/L	Vldl Post mmol/L	Normal Range mmol/L	VLDL % Decrease
F/ 48	Diabetes Hyperphagia	31.2	3.12	1.99	<1.7	-36.22%	1.98	1.26	<1.6	-36.36%
F/ 54	Diabetes Hyperphagia	30.4	2.76	2.14	<1.7	-22.46%	2.15	1.53	<1.6	-28.83%
F/ 56	Prediabetes Hyperphagia	31.6	3.88	2.34	<1.7	-39.69%	2.11	1.64	<1.6	-22.27%
F/ 47	Hyperphagia	28.7	2.10	1.44	<1.7	-21.42%	1.66	0.97	<1.6	-68.6%
F/ 52	Prediabetes Hypertension Hyperphagia	26.8	1.93	1.53	<1.7	-20.73%	1.73	1.43	<1.6	-92.3%
F/ 49	Hyperphagia	27.1	1.87	1.63	<1.7	-12.83%	1.66	1.34	<1.6	-19.28%
F/ 58	Prediabetes Hypertension	29.5	2.67	1.66	<1.7	-37.82%	1.88	0.95	<1.6	-49.46%

	Hyperphagia									
F/ 50	Hyperphagia	27.3	1.95	1.48	<1.7	-24.11%	1.53	0.99	<1.6	-35.95%
F/ 55	Prediabetes Hyperphagia	27.1	1.92	1.12	<1.7	-41.66%	1.72	0.84	<1.6	-51.16%
F/ 49	Hyperphagia	29.5	2.28	1.24	<1.7	-45.61%	1.75	0.73	<1.6	-58.28%
		Mean Average Triglycerides % Decrease				-30.26%	Mean Average VLDL % Decrease			-46.25%

Table 4: Scale Results on Visceral Adipose Tissue and Blood Plasma Results on Free T3 for each subject.

Gender / Age	Medical History	BMI	Visceral Fat Pre	Visceral fat post	Visceral Fat % Decrease	Free T3 Pre pmol/L	Free T3 Post pmol/L	Normal Range pmol/L	Free T3 % Increase pmol/L
F/ 48	Diabetes Hyperphagia	31.2	142.65	119.42	-16.28%	2.19	2.88	2.63-5.7	31.50%
F/ 54	Diabetes Hyperphagia	30.4	138.54	112.30	-18.94%	2.34	2.76	2.63-5.7	34.95%
F/ 56	Prediabetes Hyperphagia	31.6	144.23	121.12	-23.11%	1.98	2.64	2.63-5.7	33.33%
F/ 47	Hyperphagia	28.7	123.55	96.48	-21.91%	2.67	2.93	2.63-5.7	9.73%
F/ 52	Prediabetes Hypertension Hyperphagia	26.8	104.38	89.23	-14.51%	2.32	2.89	2.63-5.7	21.98%
F/ 49	Hyperphagia	27.1	108.93	87.44	-19.73%	2.89	3.05	2.63-5.7	5.53%
F/ 58	Prediabetes Hypertension Hyperphagia	29.5	119.67	98.66	-17.55%	2.29	2.78	2.63-5.7	21.39%
F/ 50	Hyperphagia	27.3	117.80	95.64	-18.81%	2.68	3.29	2.63-5.7	22.76%
F/ 55	Prediabetes Hyperphagia	27.1	98.77	81.32	-17.66%	2.16	2.59	2.63-5.7	19.91%
F/ 49	Hyperphagia	29.5	121.63	105.24	-13.47%	2.86	3.11	2.63-5.7	8.74%
	Mean Average Visceral Fat % Decrease				-18.20%	Mean Average Free T3 % increase			+ 20.98

Table 5: Blood Plasma Results on Leptin (Reference Ranges of Leptin Levels According to Body Mass Index, and Gender)

Gender / age	Medical History	BMI	Leptin Pre ng/mL	Leptin Post ng/mL	Normal Range ng/mL	% Increase ng/mL	Ghrelin Pre pg/mL	Ghrelin Post pg/mL	Normal Range pg/mL	% Decrease pg/mL
F/ 48	Diabetes Hyperphagia	31.2	21.45	27.44	12.2-67.5	27.92%	483	414	340-450	-14.28%
F/ 54	Diabetes Hyperphagia	30.4	14.63	18.08	10.6-58.3	23.58%	488	463	340-450	-5.13%
F/ 56	Prediabetes Hyperphagia	31.6	10.67	13.66	12.2-67.5	28.02%	462	398	340-450	-13.85%
F/ 47	Hyperphagia	28.7	7.09	11.33	7.9-43.5	59.80%	345	376	340-450	-8.98%
F/ 52	Prediabetes Hypertension Hyperphagia	26.8	12.34	15.12	5.9-32.4	22.53%	498	453	340-450	-9.03%
F/ 49	Hyperphagia	27.1	10.65	12.39	6.8-37.5	16.33%	357	313	340-450	-12.32%
F/ 58	Prediabetes Hypertension Hyperphagia	29.5	20.66	21.45	9.1-50.4		387	364	340-450	-5.94%
F/ 50	Hyperphagia	27.3	11.65	15.43	6.8-37.5	3.82%	401	389	340-450	-2.99%
F/ 55	Prediabetes Hyperphagia	27.1	15.24	18.56	6.8-37.5	21.78%	465	432	340-450	-7.09%
F/ 49	Hyperphagia	29.5	18.54	19.82	9.1-50.4	6.90%	474	439	340-450	-7.38%
Mean Average Visceral Fat % Decrease						+23.41%	Mean Average Grelin % Decrease			-8.7%

Table 6: T-Tests Statistical Significance Results on Blood Plasma and Measurement Variables.

	Mean	S^2 =SS/df	$S^2M = S^2/N$	SM= $\sqrt{S^2M}$	T Value	p Value	Probability
VLDL	-0.65	0.06	0.01	0.08	-8.29	<0.00001	P<0.00001
Triglycerides	-0.79	0.15	0.02	0.12	-6.36	0.00007	P<0.0001
Free T-3	0.45	0.03	0.00	0.06	7.8	0.00001	P<0.0001
Leptin	3.04	2.32	0.23	0.48	6.31	0.00007	P<0.0001
Ghrelin	-31.9	801.66	80.17	8.95	-3.56	0.00305	P<0.01
Cortisol	-46	1691.11	169.11	13	-3.54	0.00317	P<0.01
Testosterone	3.34	3.14	0.31	0.56	5.97	0.00011	P<0.001
Visceral Adipose Tissue	-21.33	15.81	1.58	1.26	-16.96	<0.00001	P<0.00001
Skeletal Muscle Mass	2.19	0.62	0.06	0.25	8.83	<0.00001	P<0.00001
IGF-1	1.82	0.49	0.05	0.22	8.21	<0.00001	P<0.00001
BMI	-2.13	0.34	0.03	0.18	-11.59	<0.00001	P<0.00001

Discussion

This randomized double-blind pilot study was performed on a small sample of menopausal females with sedentary lifestyles and reported hyperphagia. The objective was to search for exercise alternatives that can theoretically enhance immune defenses against viral infections, based on an improved health status. A growing number of research projects identify obesity as one of the most prominent

Covid19 vulnerability factors. The abundance of angiotensin-converting enzyme-2 (ACE2) receptors in adipose tissue turns overweight individuals into COVID19 chosen targets, by virtue of the virus' specific binding affinity to ACE2 receptors. Accumulated adiposity renders physical activity progressively more cumbersome and exhausting, gradually diminishing the possibility of adopting exercise as a solution. An important consideration is that weight loss inevitably rebounds when left in the mercy of leisure and gastronomical delights that have consequently increased obesity up to a tenfold around the world in the past few years, preluding the devastating effects of this highly contagious pandemic.

Cortisol, and the appetite-controlling hormones leptin and ghrelin are key to reducing weight. We investigated these instigators of increased food consumption and found a statistically significant increase in the anorexic hormone leptin, accompanied by a significant decrease of the orexigenic hormone ghrelin, and the stress hormone cortisol, with all upwards and downwards fluctuations remaining within normalcy. Additionally, results demonstrated a significant decline in visceral adipose tissue, VLDL and triglycerides and a normal range increase of free T3, and testosterone. Abnormalities in these variables have been associated with pre-existing conditions susceptible to COVID-19, including diabetes, CVD and respiratory diseases. Testosterone increased while cortisol decreased but without spiking outside the normal range, in contrast to the adverse cortisol/testosterone inverse relationship observed after strenuous overtraining that can undermine fitness by increasing stress eating behaviors. The IGF-1 and skeletal muscle mass increase delineated in our findings, may be protective in cases of pre-existing muscle tissue damage identified in COVID-19 patients as a result of high LDH-4 and LDH-5, or age-related sarcopenia.

This clinical trial explored an alternative to exercise at a time when gyms and other physical activity facilities are closed due to COVID-19. It is meant as a preventive and protective method and is not intended as a medical treatment. No conclusions can be drawn regarding its effects on COVID-19 symptomatology until it is actually implemented on patients infected by the virus.

Overall, subjects reported an increase in energy and motivation to engage in physical activity, along with significantly reduced hyperphagia as noted by less cravings for sugar and fatty or fried foods, yet, normal appetite, attesting to the reduced cortisol and ghrelin and increased leptin found in their blood samples. There are several improvements that can be applied to the methodology of the current research, including imaging methods and exploration of inflammatory and toxicity markers that can further validate the hypothesis of health improvement after using this alternative to exercise.

Conflict of Interests

The author has no conflicts of interests to disclose

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