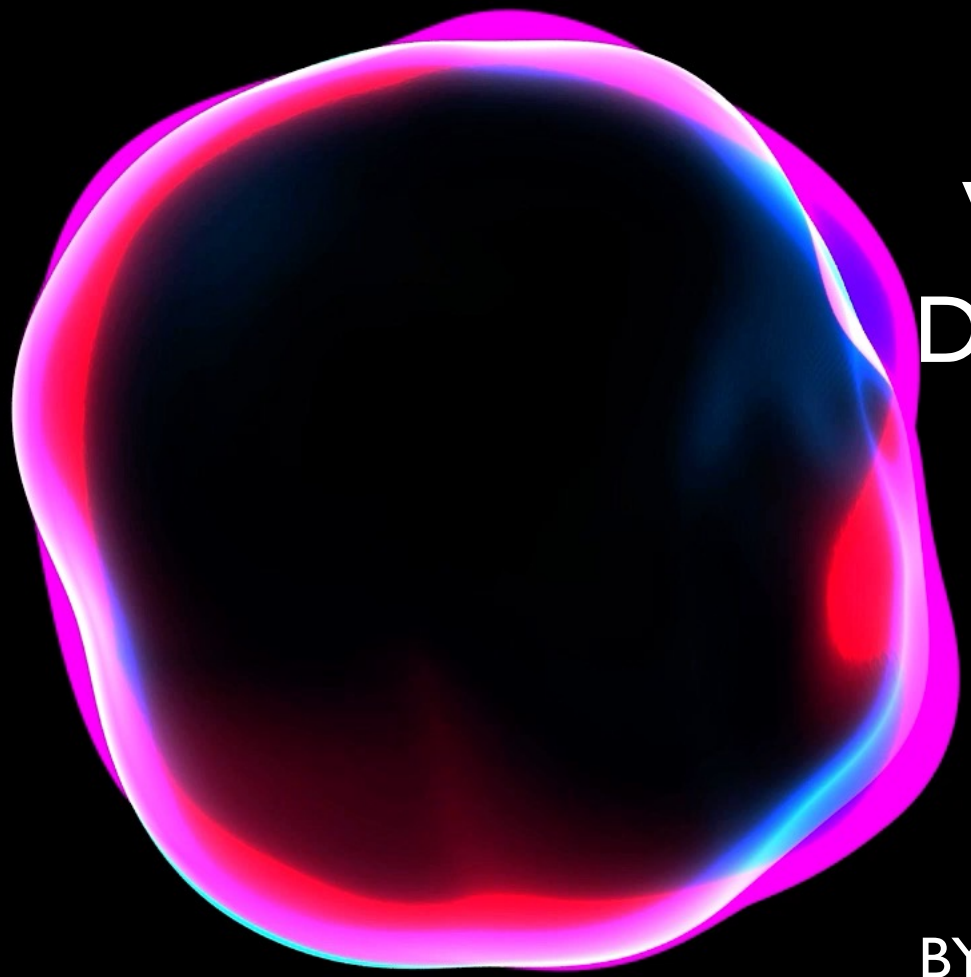


HOW TO REDUCE VISCERAL FAT AND DECREASE HUNGER

BY XANYA SOFRA, PH.D

Physical activity Increases:

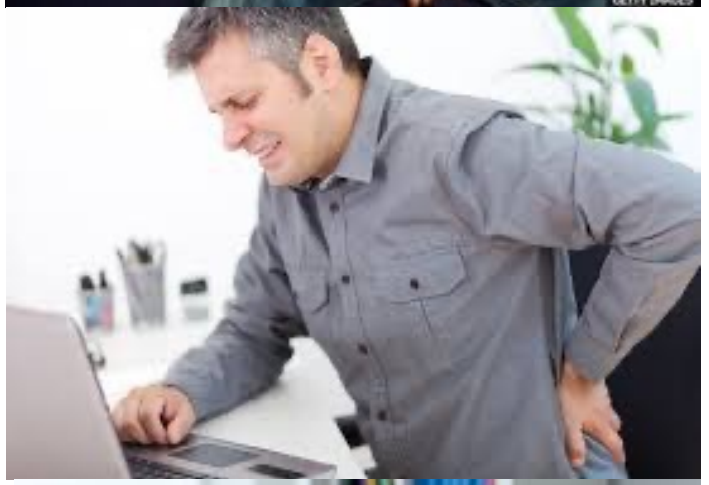
- cardiovascular fitness
- muscle mass
- healthy blood glucose regulation
- reduces visceral fat
- triglycerides
- low-density lipoproteins.

VS

Sedentary lifestyles promote:

* Adipose tissue accumulation
* Systemic inflammation
- Oxidative damage
- Chronic Pain

MEDICAL DISORDERS



Exercise Demands

1. Commitment
2. Time
3. Energy
4. Stamina
5. Persistence

Exercise is not Concerned about

1. **Time restraints**
2. **Career responsibilities**
3. **Movement restriction due to Obesity**
4. **Chronic Pain**
5. **individual choice**

Over a BILLION of individuals obese globally

OBESITY ➡ INSULIN RESISTANCE ➡

1. TYPE 2 DIABETES
2. HYPERTENSION
3. CARDIOVASCULAR DISEASE (CVD)

CVD kills about 18 million individuals yearly

VAT

Excess Triglycerides

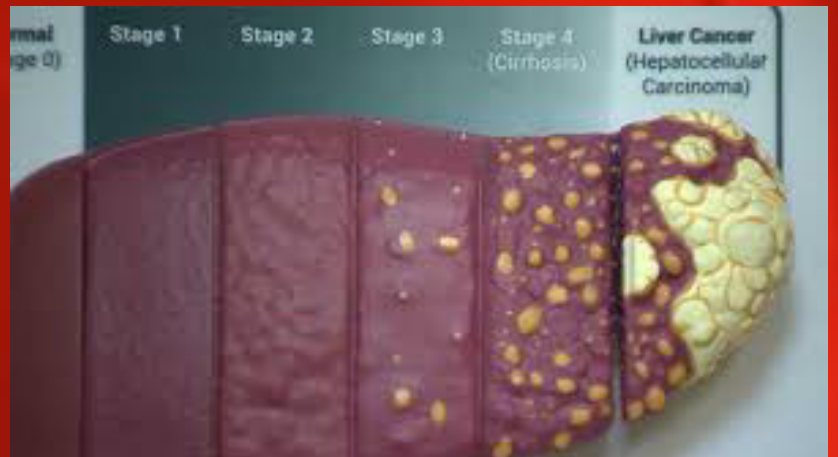
Lipotoxicity

Inflammation

Non-alcoholi steatosis

Steatohepatiti

Cirhosis



SIXTEEN MINUTES OF LOW-LEVEL LASER THERAPY (LLLT), COMBINED WITH ONE HOUR OF AEROBIC AND RESISTANCE EXERCISE, REPORTED VISCERAL FAT REDUCTION AS MEASURED BY A CONDUCTANCE SCALE. HOWEVER, IT IS UNCLEAR IF THE RESULTS WERE DUE TO THE LLLT OR THE EXERCISE.

A follow up study by the same investigators demonstrated no visceral fat differences between the experimental and control groups

No External Validity

Duarte, F., Seme-Fiorese, M., Eduard de Aquino, A., Campos, R., Masquio, D., Tock, L., Duarte, A., Bagnato, V., Parizotto, N. (2018). The Effects of Exercise Training Associated With Low-Level Laser Therapy on Biomarkers of Adipose Tissue Transdifferentiation in Obese Women. Lasers Med Sci. 33(6):1245-1254. doi: 10.1007/s10103-018-2465-1.Epub 2018 Feb 23. PMID: 29473115

There are no peer review RF studies on Visceral Fat reduction

LIMITATIONS WITH EXERCISE

Randomized placebo-controlled data demonstrated some modest reduction of visceral adipose tissue, and fatty liver improvement following 8 weeks of aerobic exercise

RESISTANCE TRAINING RESULTED IN SIGNIFICANTLY LOWER LEVELS OF THE LOW-DENSITY LIPOPROTEIN (LDL), AND AN IMPROVEMENT IN MUSCULAR STRENGTH, BUT NO DIFFERENCES IN BMI

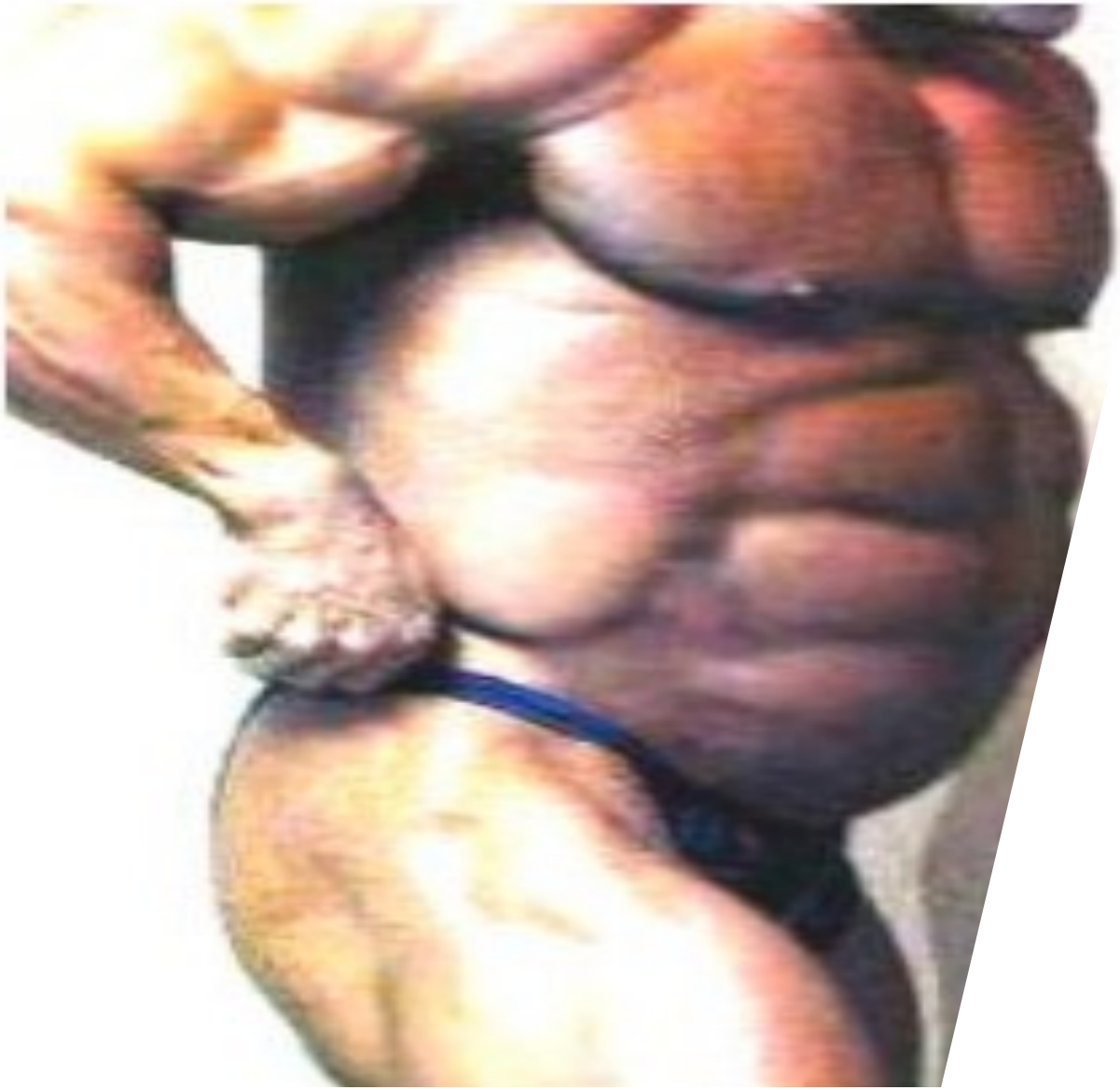
Exercise Balances Hormones

Easy to exercise when you are young and fit

Not so easy when you have accumulated weight

Over 50 years old gym workout

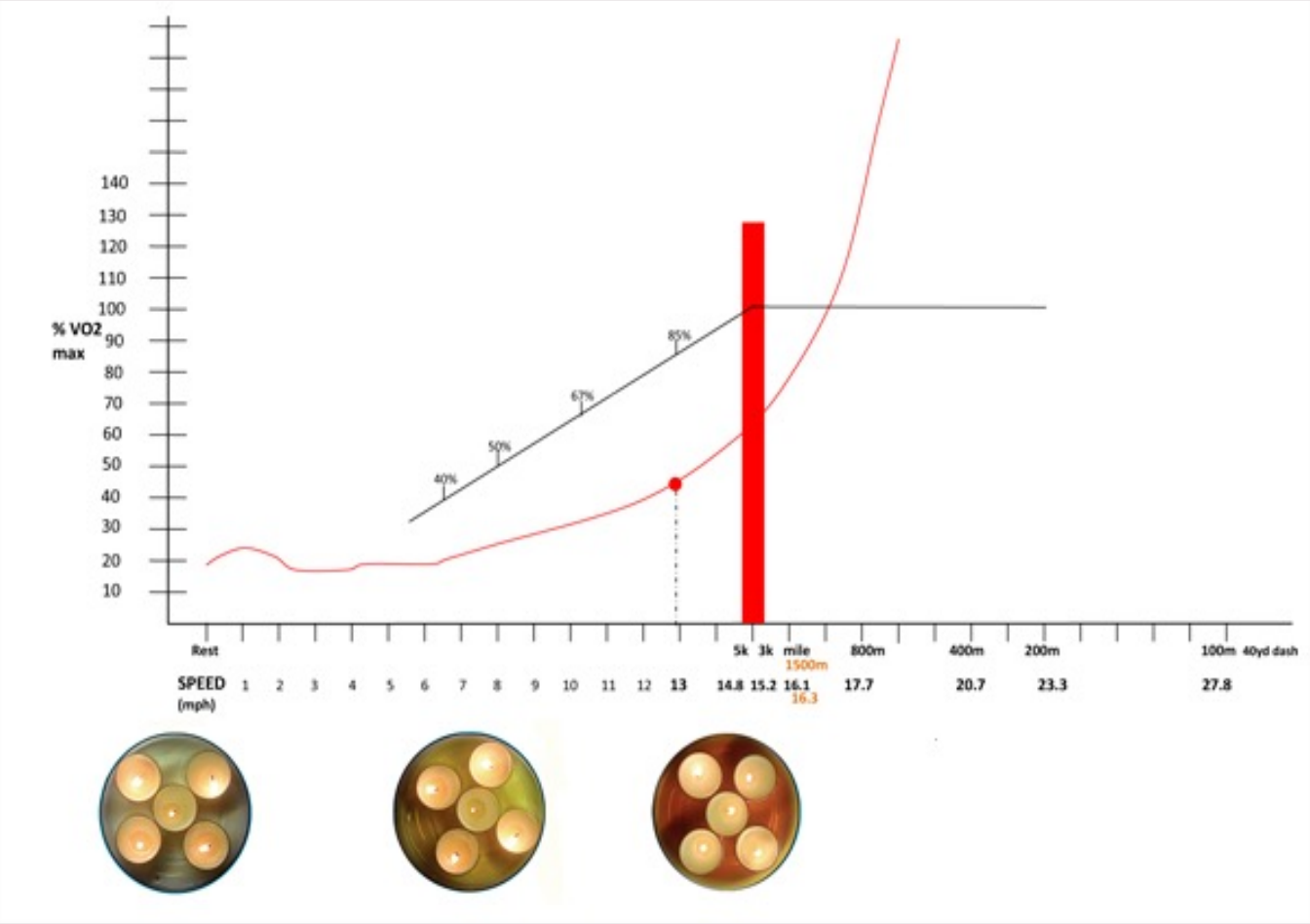
You WILL NOT Get This! ↑

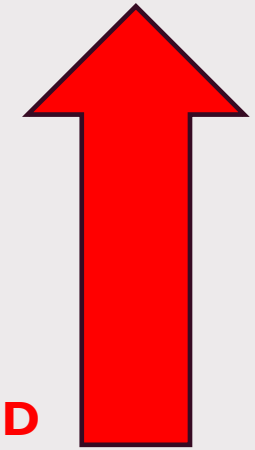


You WILL Get This! ↑

VERY STRENUOUS EXERCISE IS NECESSARY TO GET RID OF VISCERAL FAT

Overtraining can Upset PH balance





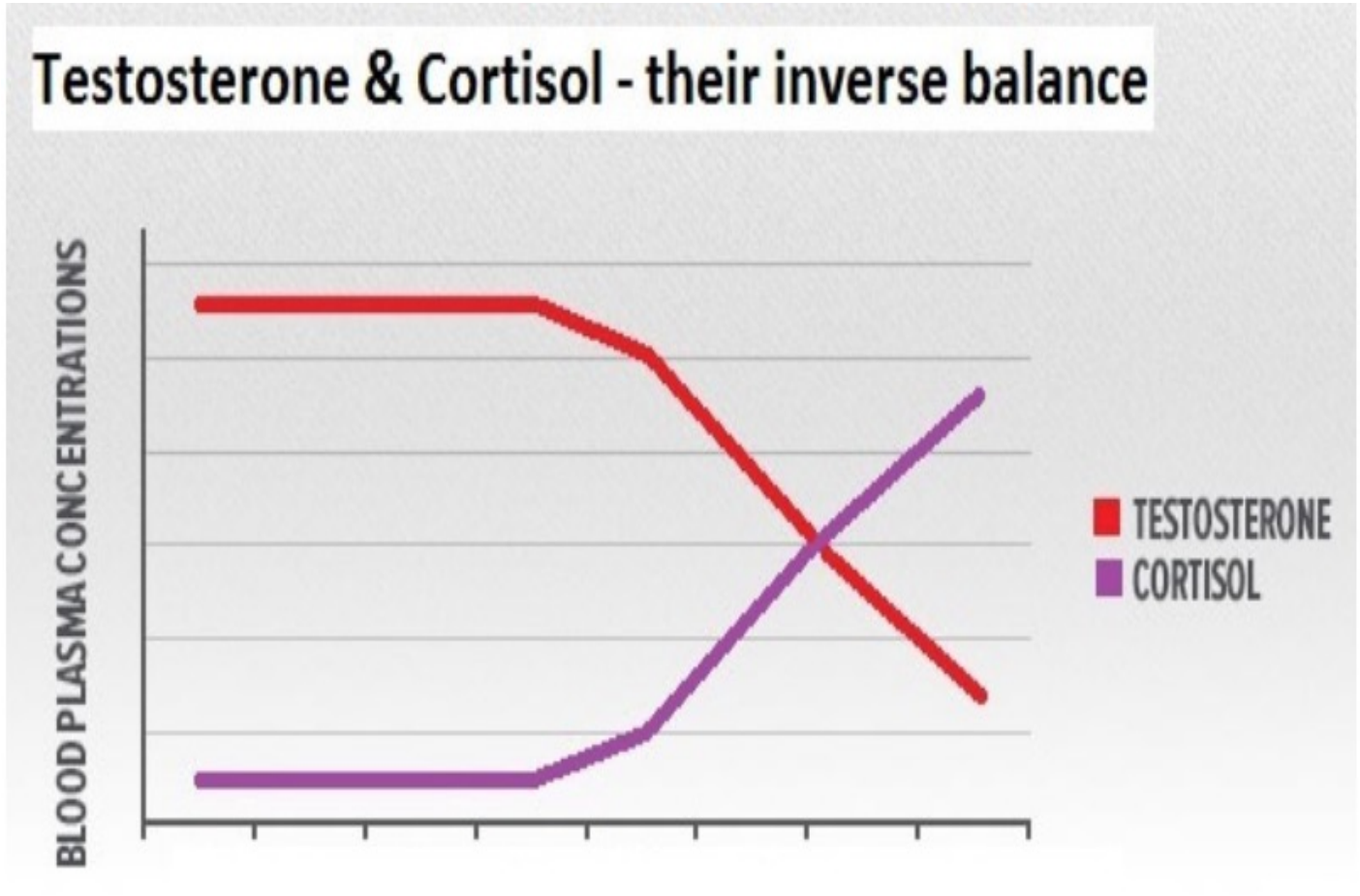
LACTIC ACID

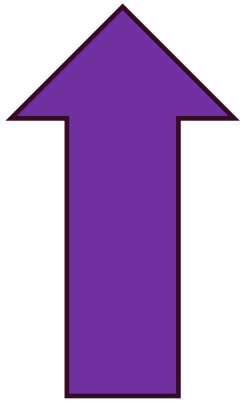
lactic acidosis

upsets the body's pH balance

VERY STRENUOUS EXERCISE IS NECESSARY TO GET RID OF VISCERAL FAT

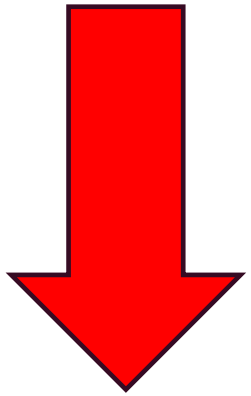
Overtraining can cause greater hormone imbalance



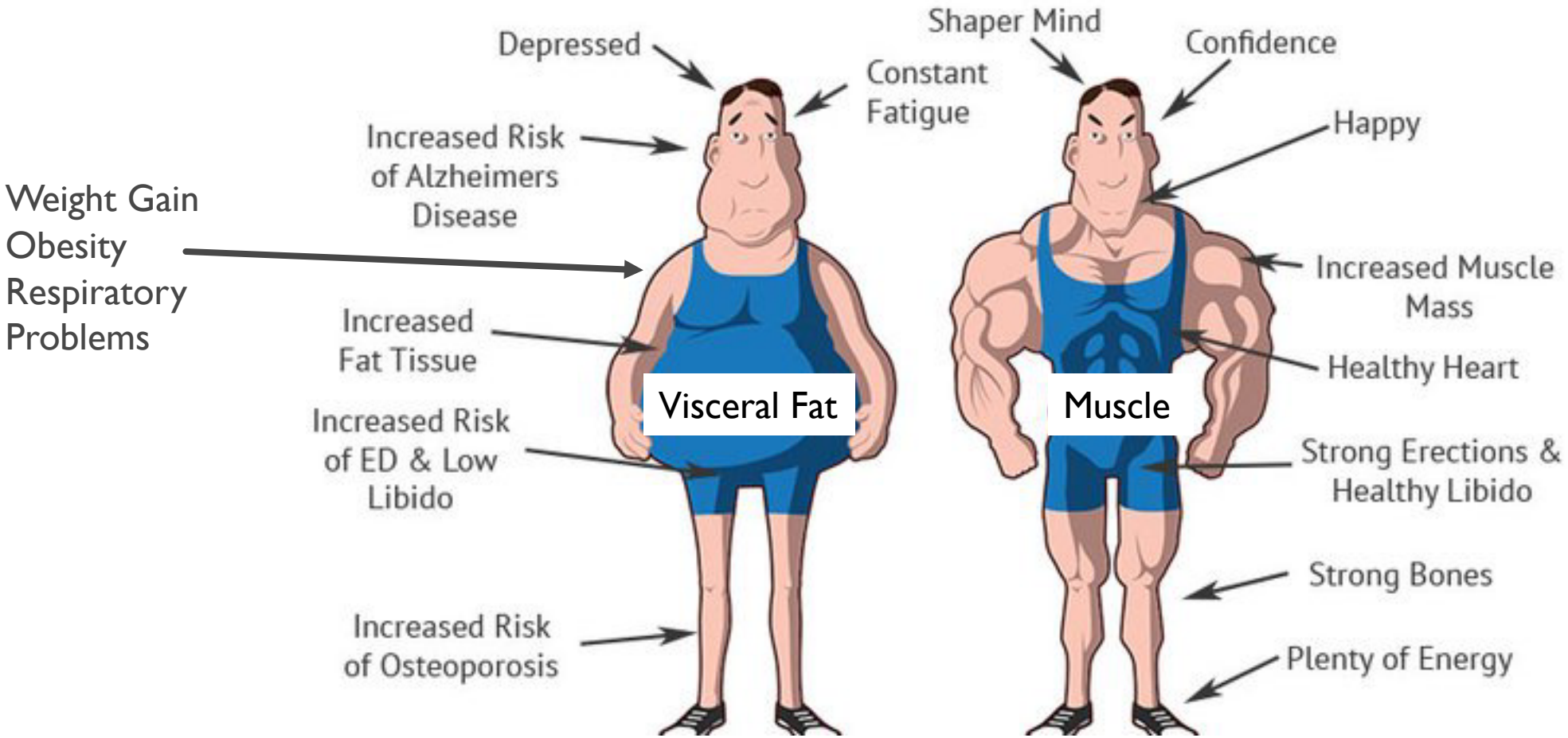


CORTISOL

TESTOSTERONE

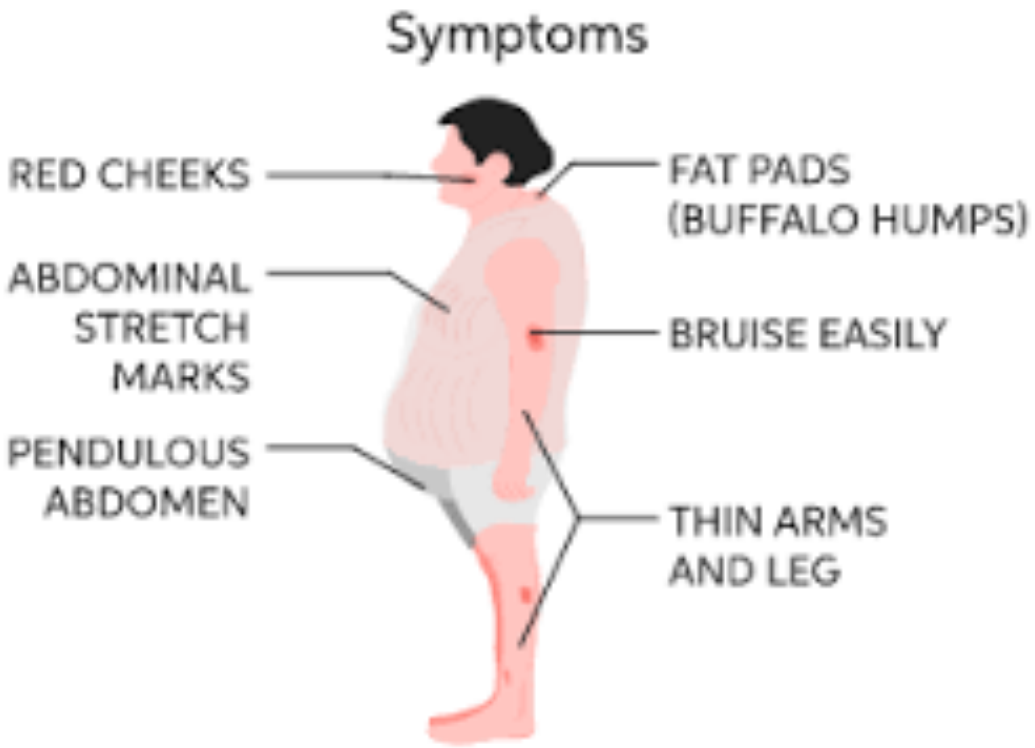


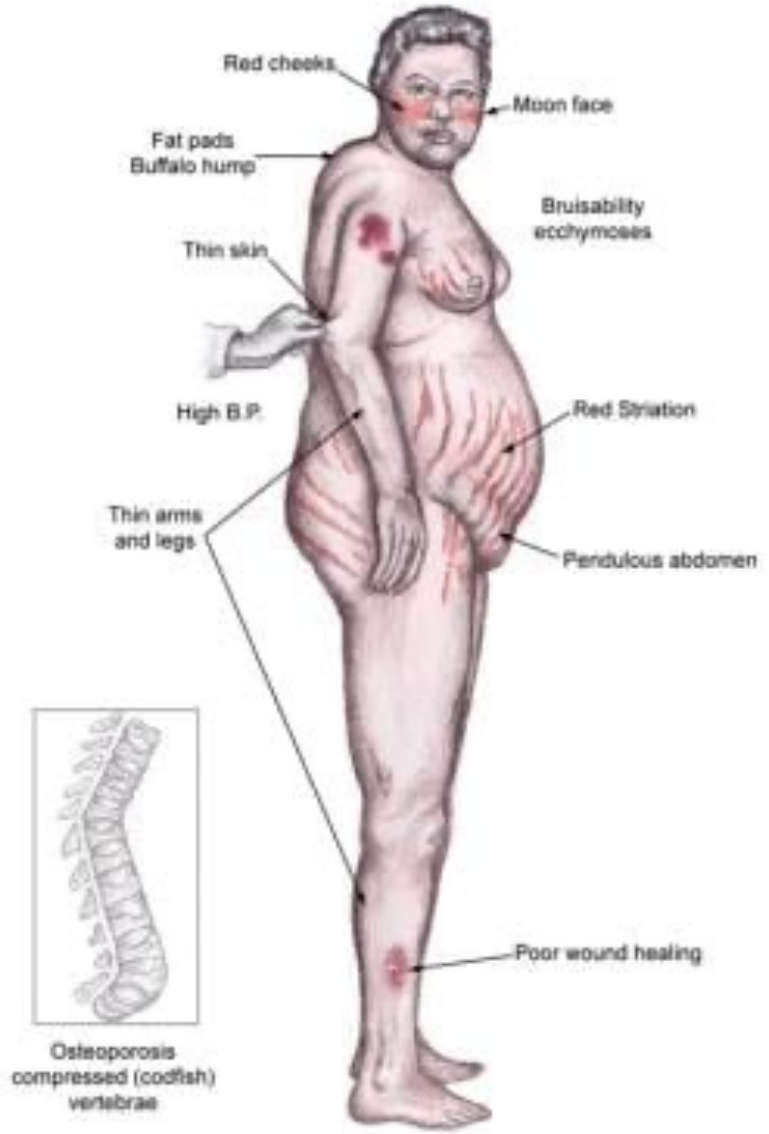
Benefits of Optimal Testosterone



This is WHY DO WE NEED THE VIRTUAL GYM

Hypercortisolism






VIRTUAL GYM
FITNESS MAX
CHANNEL 1
ABDOMEN, HIPS, BUTTOCKS, THIGHS
CHANNEL 2
START
CHANNEL 3
TIME SET
CHANNEL 5
CHANNEL 4
TREATMENT TIME
99
CHANNEL 6
STOP
CHANNEL 7
CHANNEL 8
DEEPER MUSCLE / FITNESS
CHANNELS 1 - 8
CONTRACTION
SIGNAL DURATION
2-8 SECONDS

How is the Virtual Gym different than Electrical Muscle Stimulators EMS or ELECTROMAGNETIC DEVICES?

VS

THE VIRTUAL GYM SENDS A SIGNAL TO THE BRAIN AND THE BRAIN ORDERS THE MOTOR NERVES TO CONTRACT THE **ENTIRE BODY** CAUSING A **FULL 10 SECONDS CONTRACTION** AS IN A VARIETY OF EXERCISES
AS A RESULT, THERE IS AN INCREASE OF HORMONES, ENERGY, OPTIMAL IMMUNITY AND SYSTEMIC BALANCE.
THE VIRTUAL GYM OFFERS A BODY TUNING

MUSCLE STIM AND ELECTROMAGNETIC DEVICES **SEND MULTIPLE PULSES PER SECOND** TO STIMULATE ONLY THE **INDIVIDUAL MUSCLES DIRECTLY UNDER THE STIMULATION AREA They DO NOT INVOLVE THE BRAIN**

Table 1. Blood Plasma Subjects' Results on Testosterone and Cortisol for each subject.

VIRTUAL GYM RESULTS

Gender	Age	Ethnicity	Testo sterone Pre (nmol/l)	Testo sterone post (nmol/l)	Normal range (nmol/l)	Testosterone % increase	Cortisol pre (nmol/l)	Cortisol post (nmol/l)	Normal range (nmol/l)	Cortisol% decrease
Male	36	Asian	14.75	17.3	8.64 - 29	17.28%	158	121	80 - 477.3	−23.42%
Male	39	Caucasian	11.34	13.96	8.64 - 29	23.1%	182	144	80 - 477.3	−20.87%
Male	43	Caucasian	12.38	14.6	8.64 - 29	17.92%	219	198	80 - 477.3	−9.6%
Male	35	Asian	15.41	18.65	8.64 - 29	21.02%	143	138	80 - 477.3	−3.49%
Female	42	Asian	0.5	0.92	0.29 - 1.6	84%	185	162	80 - 477.3	−12.43%
Female	45	Indian	0.3	0.63	0.29 - 1.6	110%	198	183	80 - 477.3	−7.6%
Female	49	Caucasian	0.72	1.01	0.29 - 1.6	52.77%	129	112	80 - 477.3	−13.18%
Female	38	Caucasian	0.63	0.78	0.29 - 1.6	23.8%	173	129	80 - 477.3	−25.43%
Female	37	Asian	0.53	0.69	0.29 - 1.6	30.18%	256	231	80 - 477.3	−49.76%
		Mean Average Testosterone % Increase				+42.23%	Mean Average Cortisol % Decrease			−18.42%

Both testosterone increase and cortisol decrease remained within the normal range. Testosterone overall increase was +42.23%. Testosterone showed a mean average increase of +20.15% increase for males and a mean average of +60.15% for females. Cortisol showed a mean average decrease of −18.42%.

Mean Average Testosterone % Increase	62.18 %	Mean Average Cortisol % Decrease	7.33 %

GENDER	TESTO STERONE PRE	TESTO STERONE POST	Normal Range (nmol/L)	% Increase	CORTISOL PRE	CORTISOL POST	Normal Range (nmol/L)	% decrease
MALE	10.92	14.6	8.64-29	33.6%	198	181	80-477.3	8.5%
MALE	12.16	15.43	8.64-29	26.9%	177	163	80-477.3	7.9%
FEMALE	0.3	0.71	0.29-1.6	136.6%	135	128	80-477.3	5.2%
FEMALE	0.4	0.9	0.29-1.6	125%	168	153	80-477.3	8.9%
MALE	15.38	21.6	8.64-29	40.4%	229	198	80-477.3	13.5%
MALE	13.41	19.92	8.64-29	48.5%	160	149	80-477.3	6.8%
FEMALE	0.64	0.92	0.29-1.6	43.7%	116	109	80-477.3	6.4%
FEMALE	0.4	0.71	0.29-1.6	77.5%	87	82	80-477.3	5.7%
MALE	11.3	14.4	8.64-29	27.4%	221	214	80-477.3	3.1%
FEMALE	0.43	0.72	0.29-1.6	67.4%	197	189	80-477.3	4.%

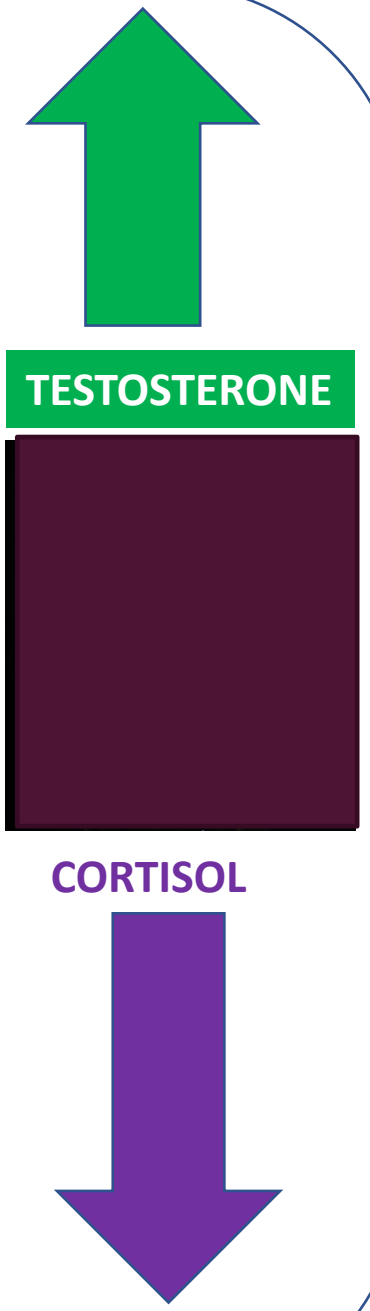
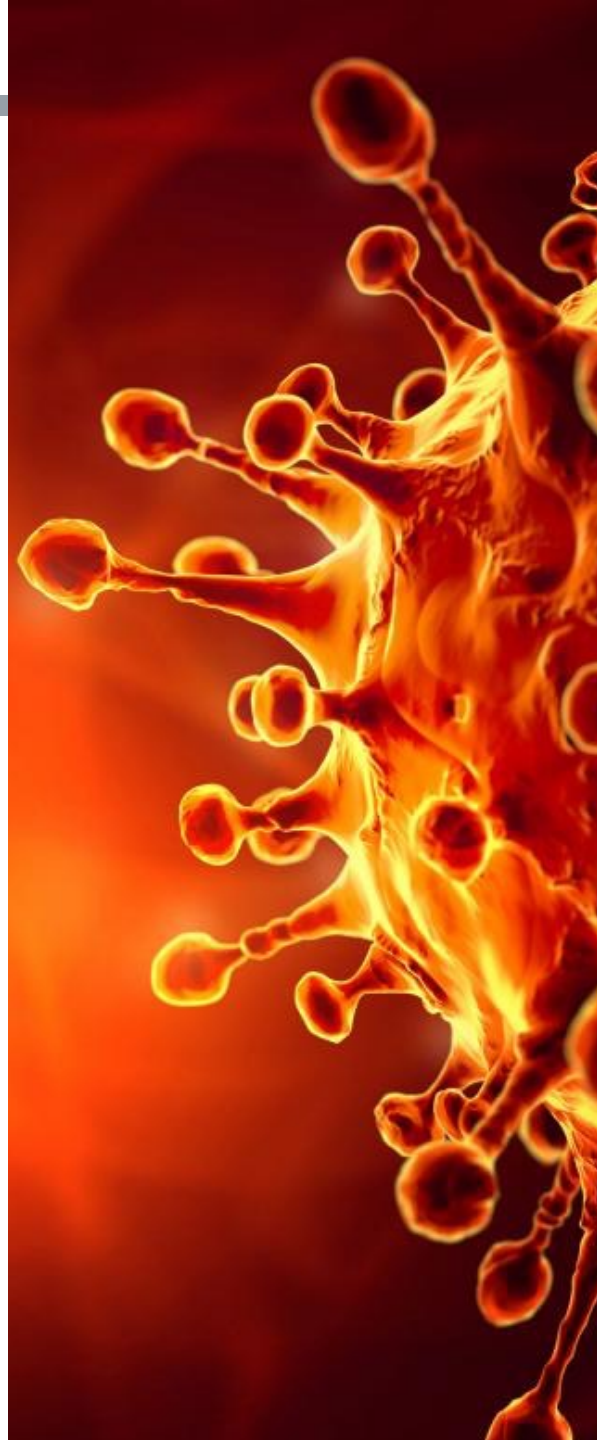


Table 5. Free T3 (triiodothyronine) and CRP (C-Reactive Protein)									
Free T3 Normal Range: 2:30-4.20 pg/mL. CRP Normal Range <1 mg/dL									
Subject NO from Table 1 DIABETES	Gender	Age	Medical Condition	Free T3 PRE pg/mL	Free T3 POST pg/mL	Free T3 Normal Range pg/mL	CRP PRE mg/dL	CRP POST mg/dL	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
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%**	

SKELETAL MUSCLE MASS (SMM) INCREASE

Mean Average % Increase for Skeletal Muscle mass	36.45%

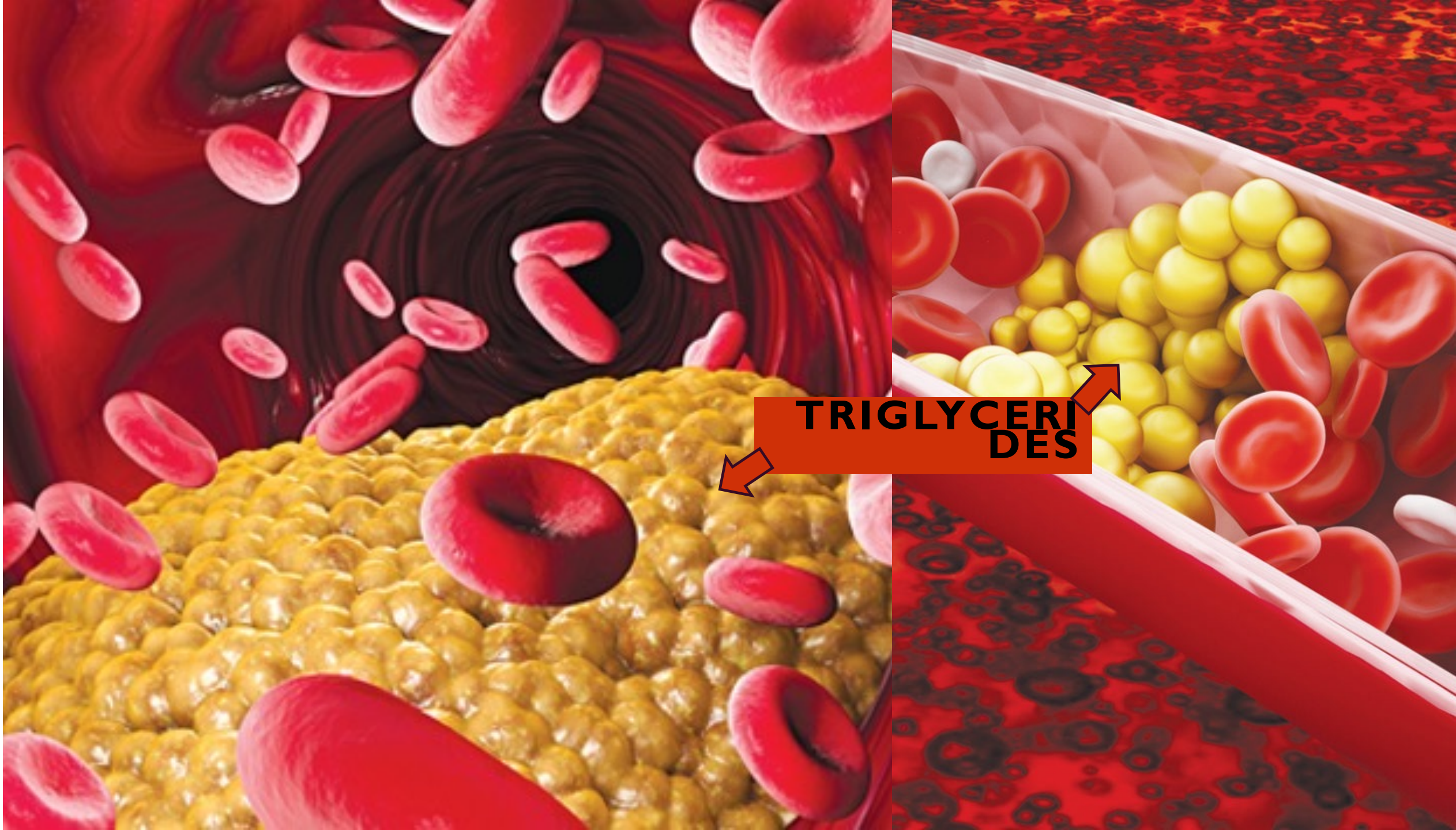
GENDER	SKELETAL MUSCLE MASS PRE	SKELETAL MUSCLE MASS POST	**SKELETAL MUSCLE MASS (SMM)** % Increase
MALE	**36.40**	**43.80**	**20.3%**
MALE	**30.30**	**38.60**	**27.39%**
FEMALE	**18.40**	**27.00**	**46.79%**
FEMALE	**17.00**	**26.80**	**57.64%**
MALE	**37.80**	**44.80**	**18.5%**
MALE	**29.40**	**38.30**	**30.27%**
FEMALE	**17.20**	**26.80**	**55.81%**
FEMALE	**19.80**	**28.80**	**45.45%**
MALE	**29.80**	**37.22**	**25.89%**
FEMALE	**17.95**	**26.63**	**48.35%**



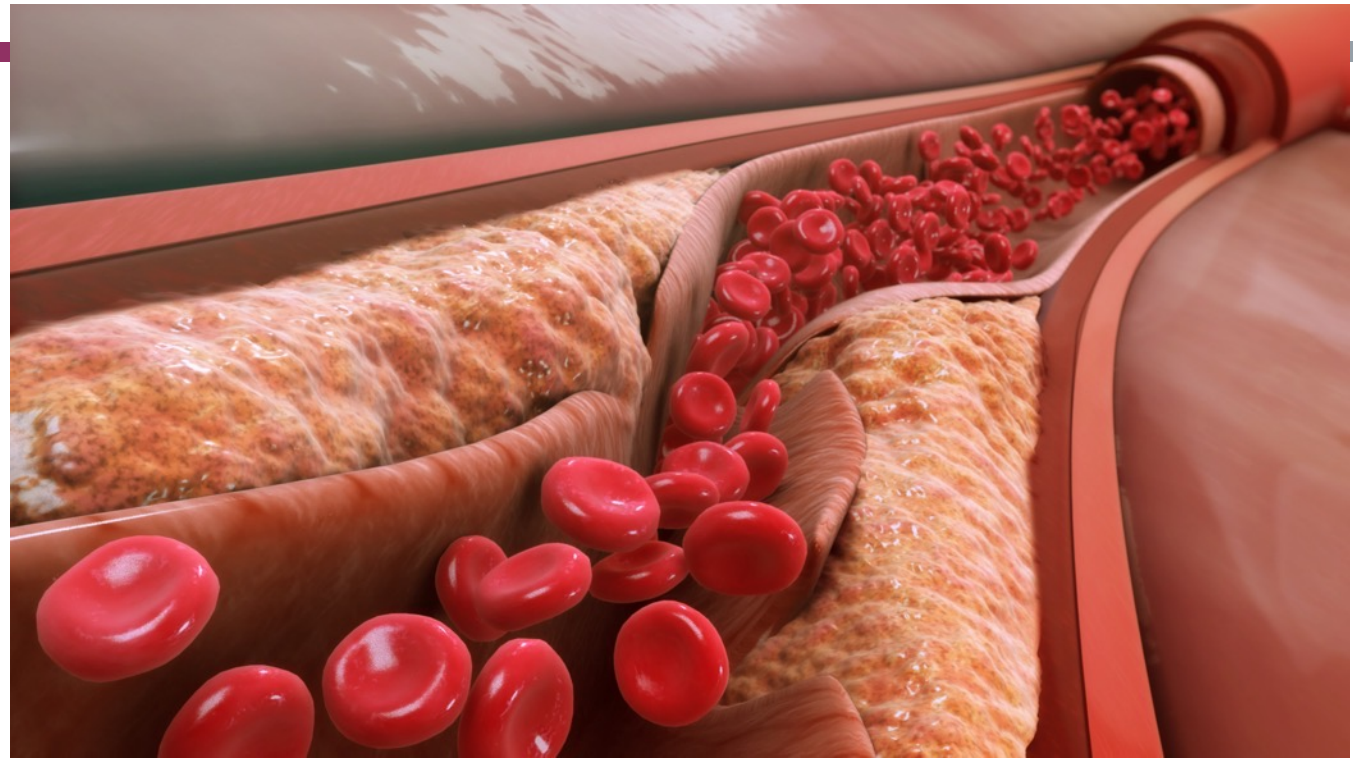
IGF-1 INCREASE

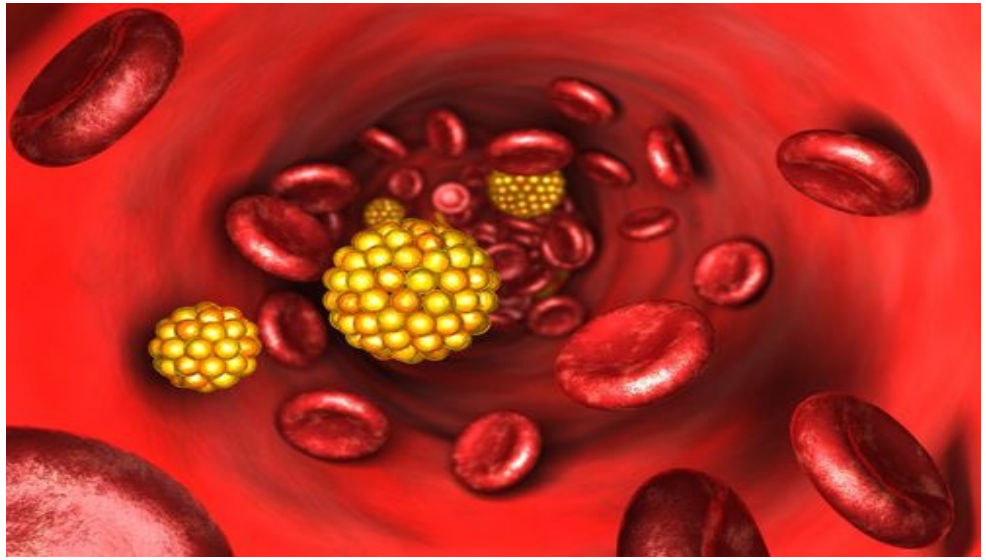
Mean Average IGF-1 % Increase	25.85% WITHIN THE NORMAL RANGE

GENDER	IGF-1 PRE	IGF-1 POST	Normal Range (nmol/L)	IGF-1% Increase
MALE	25.97	30.35	15.08-32.5	16.86%
MALE	23.98	31.12	15.08-32.5	29.77%
FEMALE	16.33	20.75	11.25-28.8	27.06%
FEMALE	15.14	19.21	11.25-28.8	26.88%
MALE	22.27	28.11	15.08-32.5	26.22%
MALE	26.98	30.52	15.08-32.5	11.80%
FEMALE	15.86	21.08	11.25-28.8	32.91%
FEMALE	18.55	23.50	11.25-28.8	26.68%
MALE	24.56	31.34	15.08-32.5	27.60%
FEMALE	19.34	25.66	11.25-28.8	32.67%


TRIGLYCERI
DES

Laser & RF lipolysis releases **triglycerides**, glucose & **toxins** into the bloodstream. A healthy liver will handle that. But detox will be a problem with individuals who have fatty liver





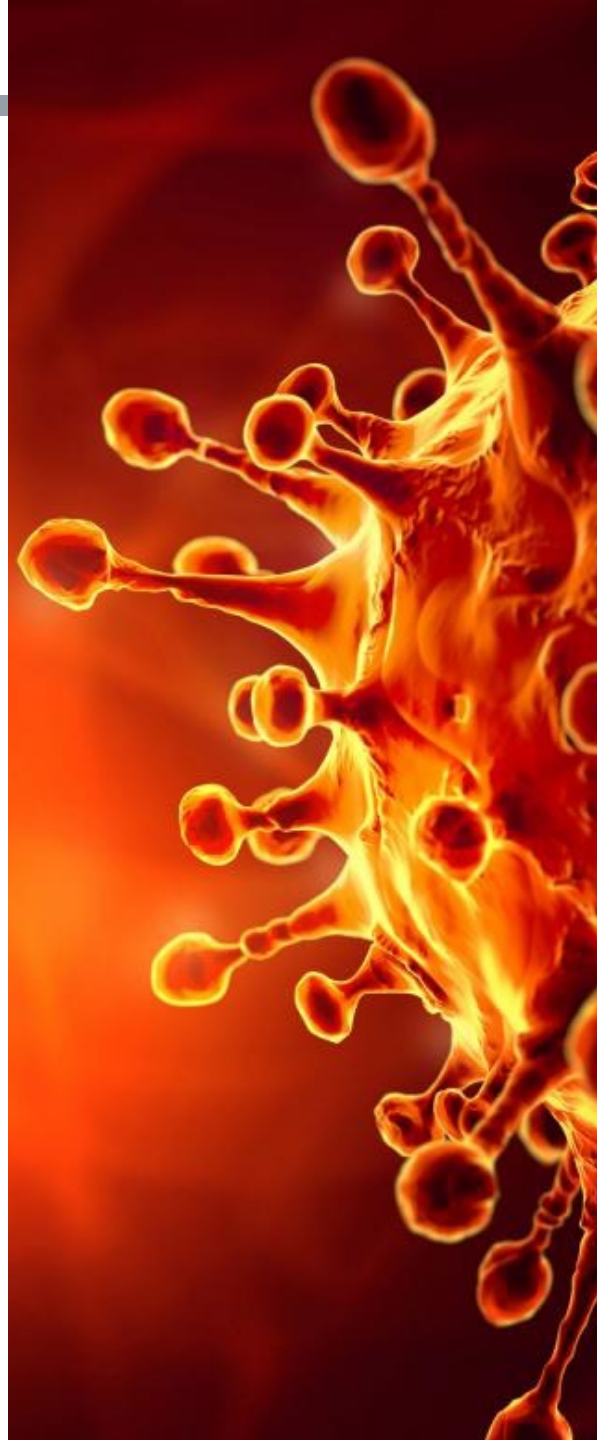
Without Exercise, triglycerides and toxins remain in the bloodstream, overwhelm the fatty live and may clog your arteries

THIS IS HOW THE VIRTUAL GYM CAN HELP

TRIGLYCERIDES DECREASE

Mean Average Triglycerides Decrease	40.7%

GENDER	TRIGLYCERIDES PRE	TRIGLYCERIDES POST	Normal Range (nmol/L)	TRIGLYCERIDES % Decrease
MALE	2.90	1.23	<1.7	55%
MALE	2.34	0.94	<1.7	59.8%
FEMALE	2.50	1.50	<1.7	40%
FEMALE	2.00	1.44	<1.7	28%
MALE	0.80	0.53	<1.7	33%
MALE	0.90	0.64	<1.7	41.1%
FEMALE	1.00	0.60	<1.7	40%
FEMALE	0.90	0.58	<1.7	35%
MALE	1.32	0.92	<1.7	30%
FEMALE	0.98	0.54	<1.7	44.9%



VLDL (THE BAD CHOLESTEROL) DECREASE

Mean Average VLDL Decrease				71.88%
GENDER	VLDL PRE	VLDL POST	Normal Range (nmol/L)	VLDL CHOLESTEROL % Decrease
MALE	1.48	0.24	<1.6	83.78%
MALE	1.55	0.64	<1.6	58.7%
FEMALE	0.80	0.20	<1.6	75%
FEMALE	0.86	0.27	<1.6	68.6%
MALE	0.52	0.04	<1.6	92.3%
MALE	1.36	0.24	<1.6	82.35%
FEMALE	0.68	0.05	<1.6	92.64%
FEMALE	0.53	0.26	<1.6	50.9%
MALE	1.53	0.67	<1.6	56.20%
FEMALE	1.75	0.73	<1.6	58.28%

Table 4. PREDIABETICS
Triglycerides, High-Density Protein (HDL),
Presence of Fatty Liver on Sonography Reports Pre and Post Treatment.

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

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	43.0	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**	

Fasting Blood Glucose: Normal <100 mg/dL; Prediabetes = 100 - 125 mg/dL; Diabetes >126 mg/dL
Blood Glucose Postprandial (PP): Normal < 140 mg/dL; Prediabetes = 140 - 199 mg/dL; Diabetes > 199 mg/dL

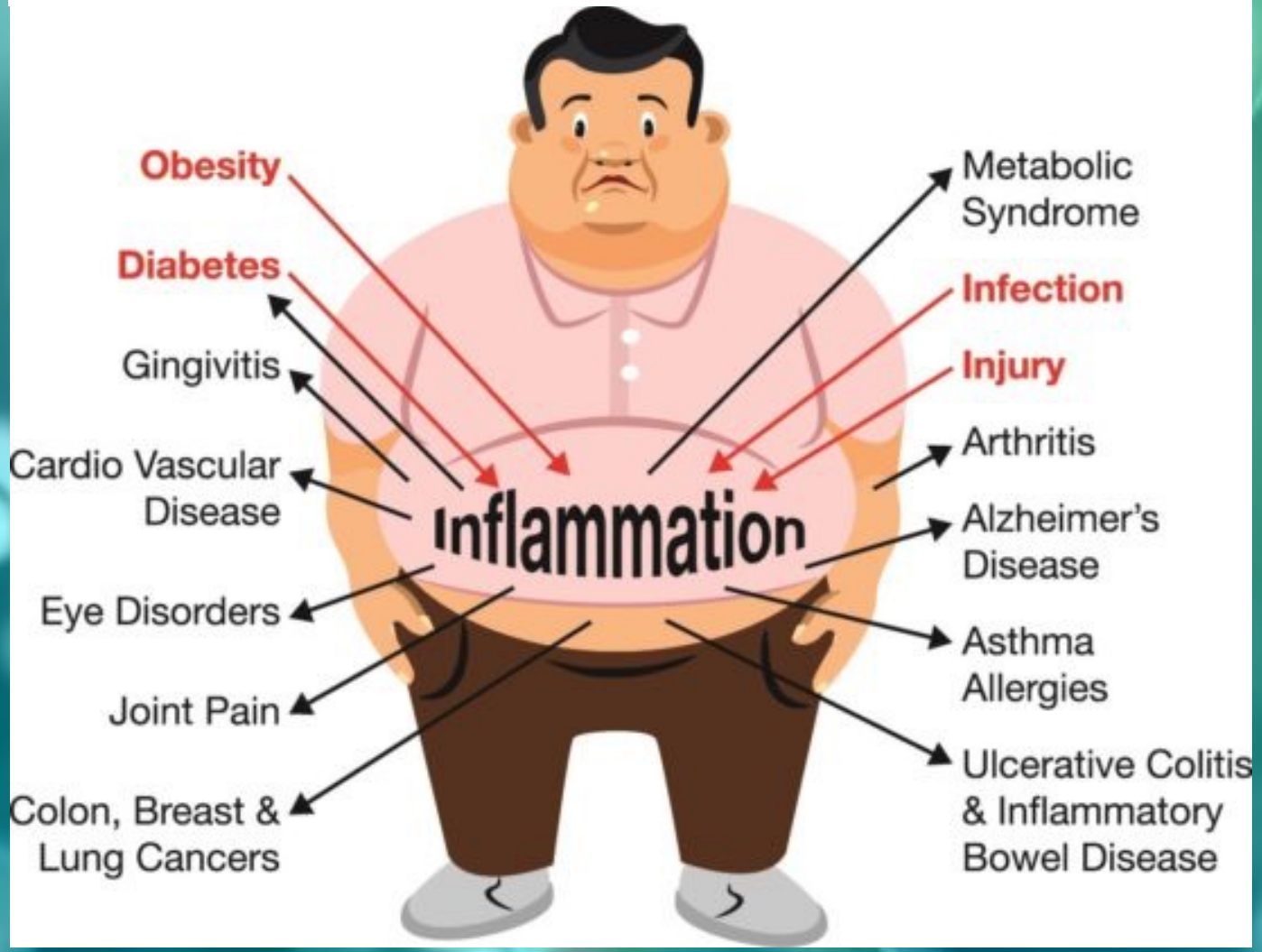
S	G	A	Medical Diagnosis Post Tx	Blood Glucose Fasting mg./dL Pre Tx	Blood Glucose Fasting mg/dL Post Tx	Blood Glucose Normal <100 mg/dL Post Tx	Blood Glucose PP mg/dL Pre Tx	Blood Glucose PP mg/dL Post Tx	Blood Glucose PP Normal < 140 mg/dL Post Tx
1	F	45	Diabetes Fatty liver	178	104	Prediabetic	260	185	Prediabetic
2	M	69	Diabetes	209	108	Prediabetic	230	125	Normal
3	M	46	Diabetes	131.7	99.15	Normal	290	183.2	Prediabetic
4	F	50	Diabetes	177	106	Prediabetic	221	176	Prediabetic
5	F	49	Diabetes Fatty Liver	192	102	Prediabetic	248	175	Prediabetic
6	F	48	Diabetes Fatty Liver	189	115	Prediabetic	224	163	Prediabetic
7	M	44	Diabetes Fatty Liver	178	109	Prediabetic	196	162	Prediabetic
8	F	45	Diabetes Fatty Liver	186	117	Prediabetic	197	126	Normal
9	F	47	Diabetes Fatty Liver	169	102	Prediabetic	243	178	Prediabetic
10	M	45	Diabetes	135	92	Normal	218	156	Prediabetic
11	M	82	Diabetes	136	87	Normal	191	142	Prediabetic
12	M	46	Diabetes	134	97	Normal	216.3	139	Normal
13	M	59	Diabetes	106.8	82	Normal	199.9	133	Normal
14	F	45	Diabetes Fatty Liver	186	117	Prediabetic	207.5	123	Normal
15	M	59	Diabetes	188	119	Prediabetic	202	133	Prediabetic
16	M	49	Diabetes	141	99	Normal	125.6	144	Prediabetic
17	F	69	Diabetes Fatty Liver	136	87	Normal	231.4	131	Normal
18	F	53	Diabetes	190	108.5	Prediabetic	212	118	Normal
19	F	68	Diabetes Fatty Liver	176	92	Normal	209.8	98	Normal
20	F	61	Diabetes Fatty Liver	157.5	98.5	Normal	204	103	Normal
21	M	55	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	Blood Fasting Glucose % Decrease	-38.44%		Blood PP Glucose % Decrease	-39.1%	

Table 2 PREDIABETICS
Pre and Post Treatment Results on Insulin (Fasting and PP)

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

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 Pre	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
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**	**-44.7%**	

Why is Visceral Fat a Problem?
INFLAMMATION & TOXICITY



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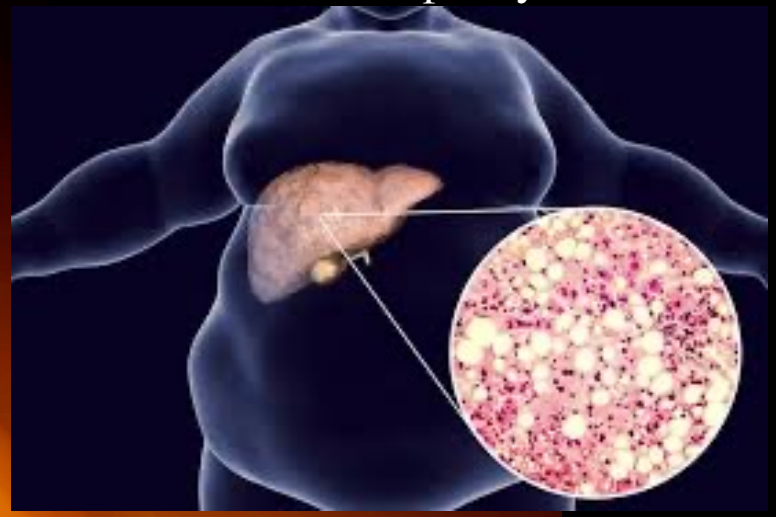
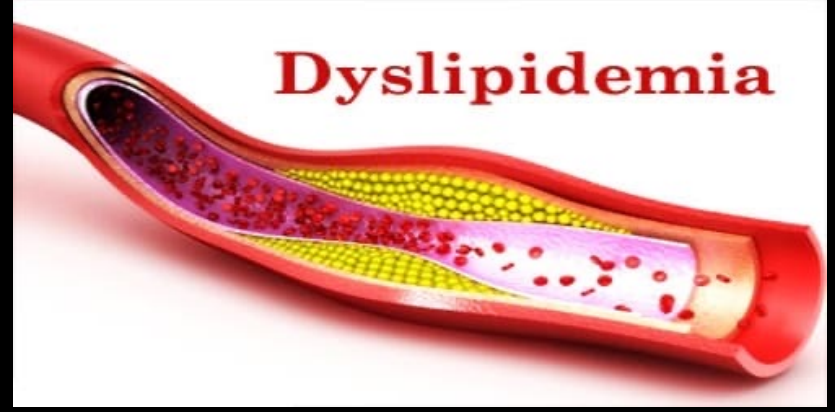
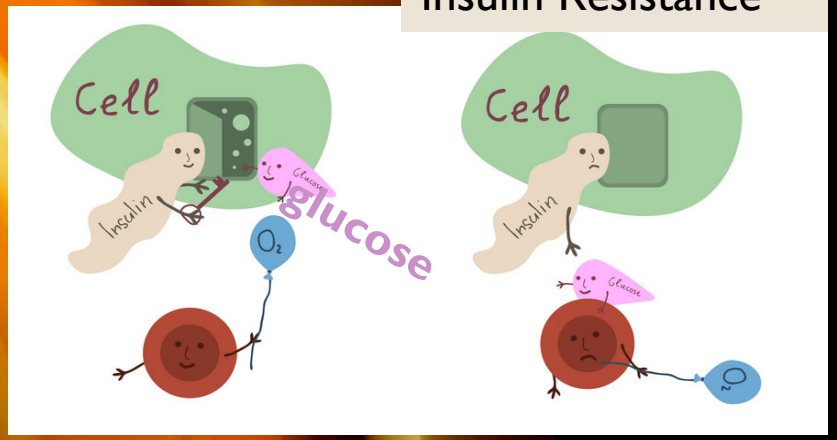
Excess adiposity





Hormonal imbalance

Insulin Resistance



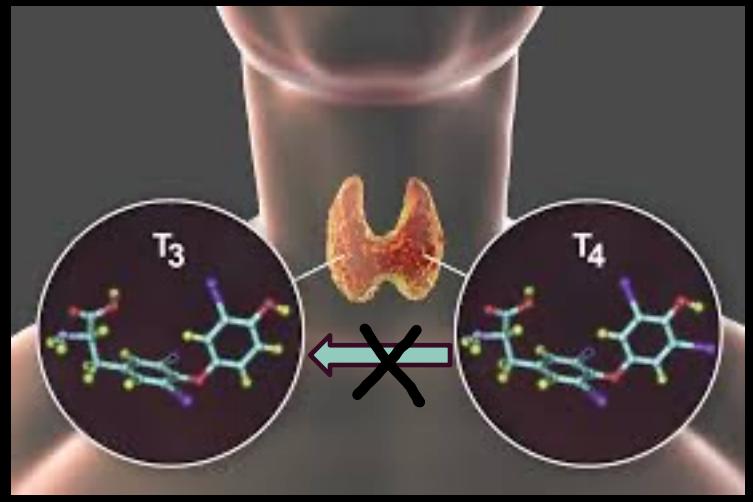


TABLE 6
Pre and Post Treatment Results on BMI, Overall Fat, Visceral Fat, and Skeletal Muscle Mass (SMM)

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	32.0	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	25.0	34.7	33	9.5	5	21.8	24.2
7	Male	40	Prediabetes	33.7	25.1	33.0	13.4	21	13.4	28.8	31.2
8	Male	39	Prediabetes	36.2	32.0	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	30.20	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 2. Blood Test Results on C-reactive protein (CRP) and Cortisol CRP: <1.0 mg/dL. Low cardiovascular risk according to AHA/CDC CRP: 1.0-3.0 mg/dL Average cardiovascular risk according to AHA/CDC CRP: >3.0-10.0 mg/dL High cardiovascular risk according to AHA/CDC

Gender	Age	Medical History	BMI PRE	CRP PRE mg/dL	CRP POST mg/dL	Normal Range mg/dL	Cortisol Total, Serum ug/dL, PRE	Cortisol Total, Serum ug/dL, POST	Normal Range ug/dL
Female	56	Diabetes Fatty Liver	32.6	1.56	1.02	<1.00	18.44	15.66	3.09-25.0
Female	52	Prediabetes Fatty Liver	36.5	1.09	1.06	<1.00	21.89	20.12	3.09-25.0
Female	49	Hypertension Hypothyroidism	28.6	2.31	1.15	<1.00	24.98	18.47	3.09-25.0
Female	63	Hypertension Fatty Liver	34.9	1.93	1.06	<1.00	23.43	21.98	3.09-25.0
Female	51	Prediabetes Hypertension Hypothyroidism	34.2	1.43	1.22	<1.00	18.46	15.34	3.09-25.0
Female	55	Prediabetes Fatty Liver Hypothyroidism	35.4	1.64	1.01	<1.00	19.33	14.75	3.09-25.0
Female	48	Prediabetes Fatty Liver Hypothyroidism	30.9	1.04	0.86	<1.00	9.67	8.23	3.09-25.0
Female	61	Hypertension Fatty Liver	32.7	1.08	0.74	<1.00	14.76	10.65	3.09-25.0
Female	46	Heart Disease	29.5	1.84	0.98	<1.00	17.22	13.95	3.09-25.0
Female	58	Prediabetes Fatty Liver Hypothyroidism	33.8	2.11	1.03	<1.00	21.28	17.24	3.09-25.0
			MEAN TOTAL	1.60 mg/dL	1.01 mg/dL		18.95 ug/dL	15.64 ug/dL	

Mean Average CRP % Decrease. -36.87 mg/dL

Mean Average Cortisol %Decrease. -17.47% mg/dL

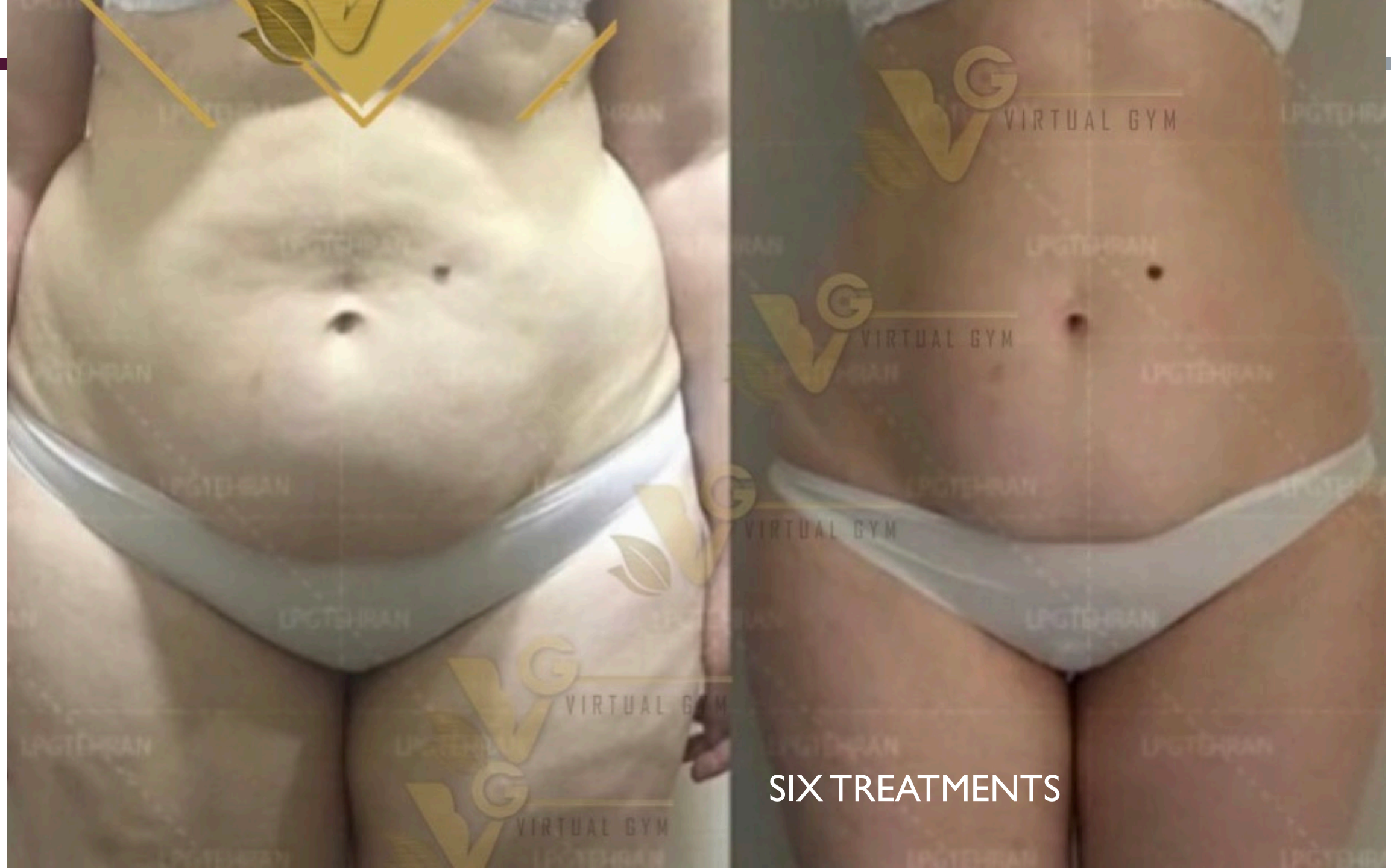
Table 3. Blood Test Results on Creatinine and Bilirubin

Gender/ Age	BMI	Medical History	Creatinine Serum PRE mg/dL	Creatinine Serum POST mg/dL	Creatinine Normal Range mg/dL	Bilirubin PRE mg/dL	Bilirubin POST mg/dL	Bilirubin Normal Range mg/dL
F/56	32.6	Diabetes Fatty Liver	1.15	0.94	0.5-1.10	1.31	1.09	0.3-1.2
F/52	36.5	Prediabetes Fatty Liver	1.03	0.87	0.5-1.10	1.44	1.63	0.3-1.2
F/49	28.6	Hypertension Hypothyroidism	1.37	1.05	0.5-1.10	1.27	1.15	0.3-1.2
F/63	34.9	Hypertension Fatty Liver	1.23	0.96	0.5-1.10	1.35	1.18	0.3-1.2
F/51	34.2	Prediabetes Hypertension Hypothyroidism	1.14	1.02	0.5-1.10	1.18	1.08	0.3-1.2
F/55	35.4	Prediabetes Fatty Liver Hypothyroidism	1.04	1.01	0.5-1.10	1.26	1.16	0.3-1.2
F/48	30.9	Prediabetes Fatty Liver Hypothyroidism	0.97	0.82	0.5-1.10	1.23	1.13	0.3-1.2
F/61	32.7	Hypertension Fatty Liver	1.18	0.98	0.5-1.10	1.33	1.05	0.3-1.2
F/46	29.5	Heart Disease	1.11	0.87	0.5-1.10	1.22	1.07	0.3-1.2
F/58	33.8	Prediabetes Fatty Liver Hypothyroidism	1.96	1.23	0.5-1.10	1.28	1.19	0.3-1.2

Mean Average Creatinine % Decrease. -19.67 mg/dL. (exits the body as a waste product)

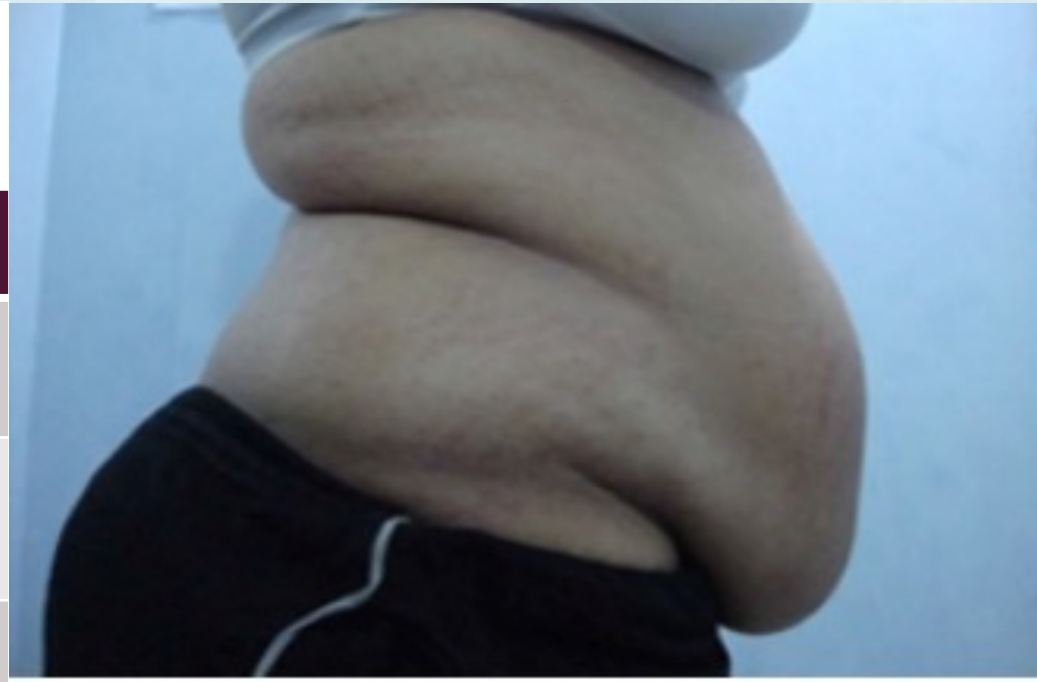
Mean Average Bilirubin % Decrease -8.85 mg/dL (excess may indicate jaundice)

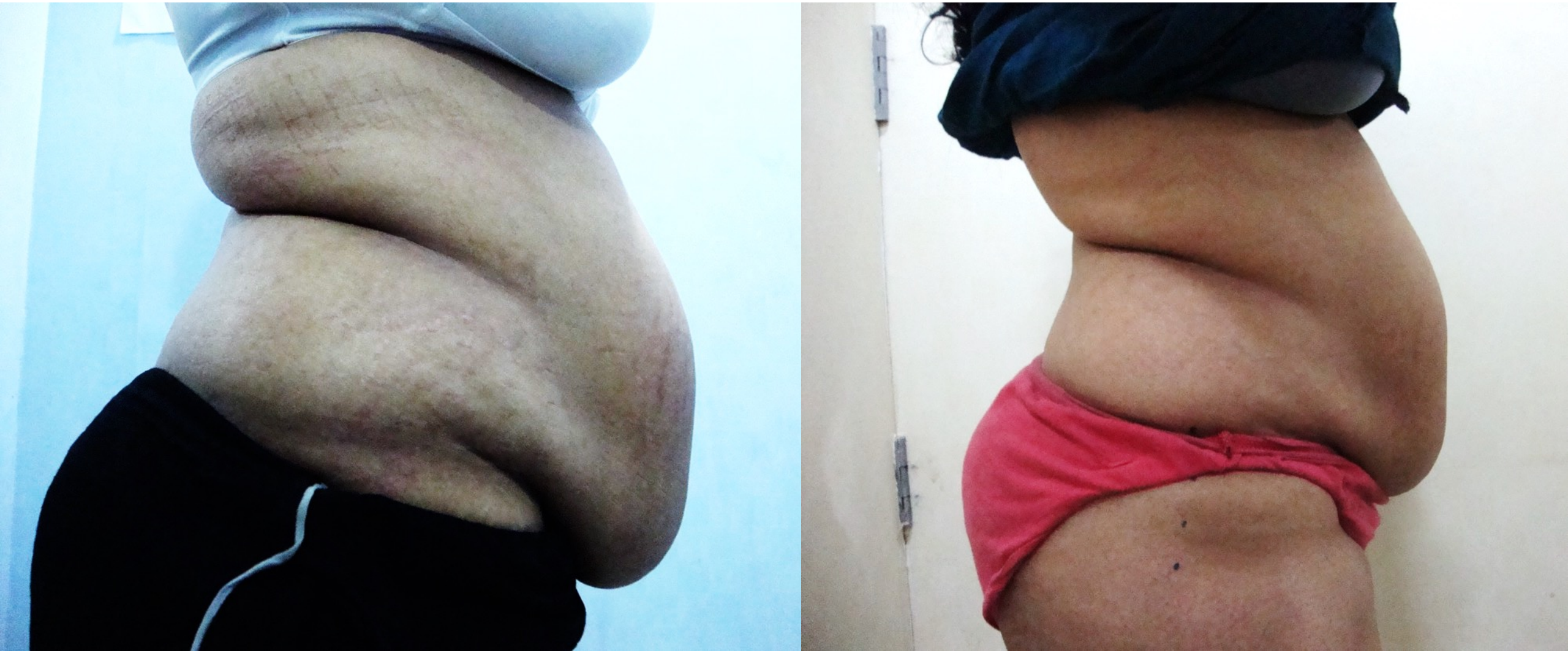

Virtual Gym One Treatment


VIRTUAL GYM
SIX TREATMENTS

Diabetic Patient with back Pain and Fatty Liver. Measures: Sonogram, Blood Test, Measuring tape, Tanita Scale, Self Reports

BEFORE	AFTER
Real Age: 43 y.o. female	METABOLIC AGE 32
Severe Obesity FAT 36.5 %	FAT% 25.8
Diabetic Status: On Insulin HbA1c- 10.8	On Oral Drugs HbA1c – 7.8
Visceral Fat Evidence Sonography Reports: Fatty Liver	NO FATTY LIVER
Lower Back Pain	NO BACK PAIN
Weight: 92.2 Kg	Significant Weight Loss 83.7 KG
Measurement: Umbilicus: 111cm	Significant Improvement: 100cm
Measurement: Lower Abdomen: 115cm	Significant Improvement: 100cm

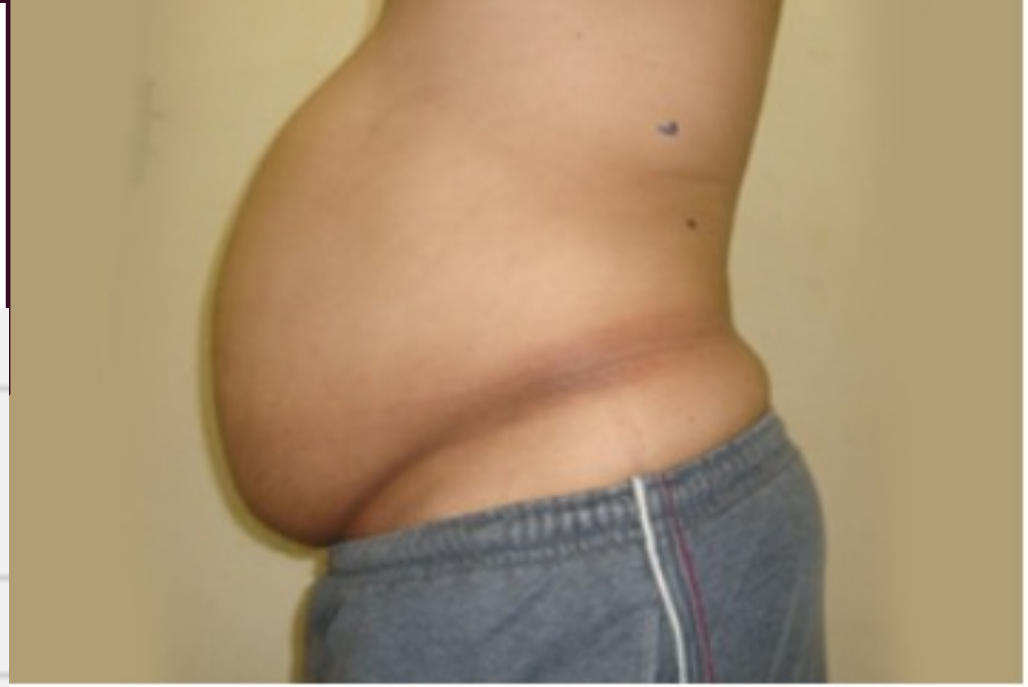


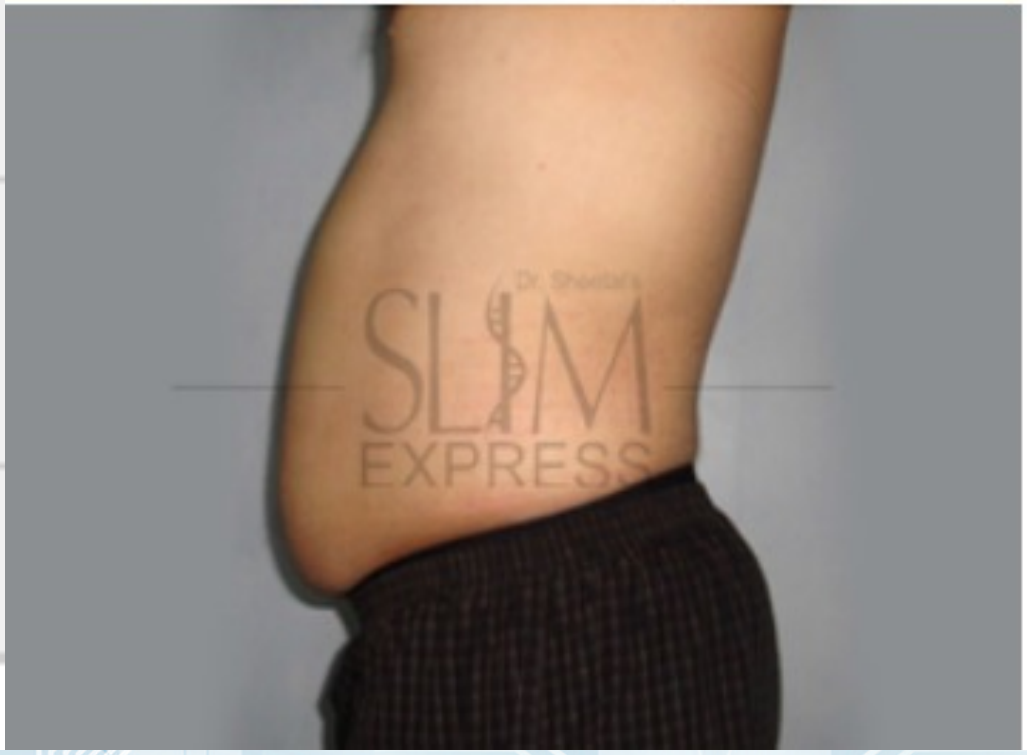


Before After 15 Treatments

49 Year old Patient suffering from Insulin Resistance and Diabetes. Measures: Sonogram, Tanita scale, Blood Test, Measuring Tape, Self Reports

	Before treatment	After treatment
Weight (kg)	75.8	67.2
Fat %	36.5	25.8
Upper abdomen(cm)	97	82
Umbilicus (cm)	100	88
Lower abdomen (cm)	105	94
Insulin-Fasting(mIU/ml)	25.8	8.7
Insulin PP (mIU/ml)	136	14
Triglycerides (mg/dl)	294	197
HDL(mg/dl) good cholelserol	36	42
Back pain	Lower Back pain +++	Significant decrease in back pain

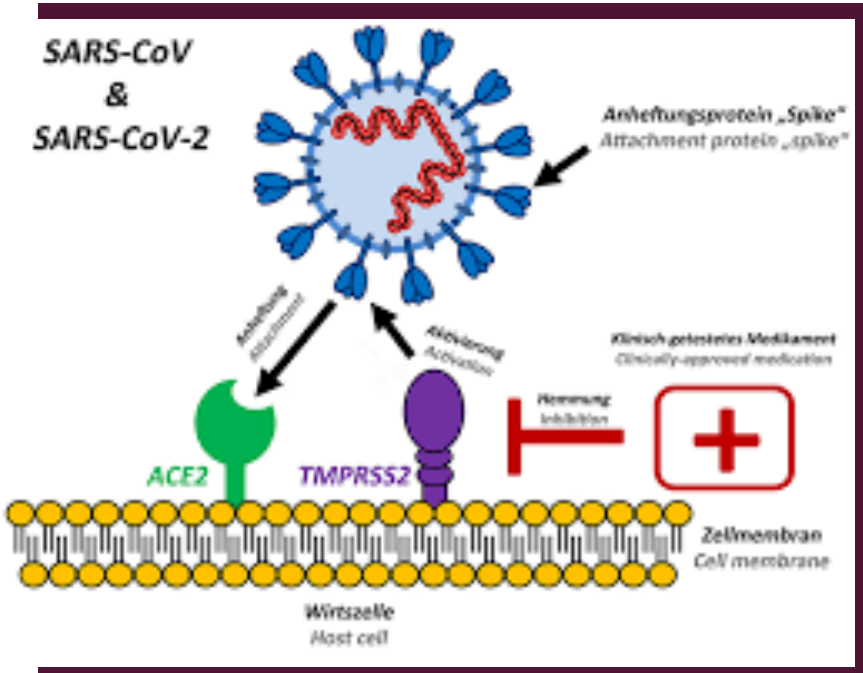




VIRTUAL GYM BALANCES HORMONES

VIRTUAL GYM INCREASES IMMUNITY

VAT



VAT has a higher expression of ACE2 receptors

MUSCLE HAS THE LEAST ACE2 RECEPTORS

TYPE I INTERFERONS & IMMUNITY

INDIVIDUALS WITH A STRONGER INTERFERONS RESPONSE HAVE MILDER COVID-19 SYMPTOMATOLOGY

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Letter | Published: 04 December 2020

Untuned antiviral immunity in COVID-19 revealed by temporal type I/III interferon patterns and flu comparison

Ioanna-Evdokia Galani, Nikoletta Rovina, Vicky Lampropoulou, Vasiliki Triantafyllia, Maria Manioudaki, Eleftherios Pavlos, Evangelia Koukaki, Paraskevi C. Fragkou, Vasiliki Panou, Vasiliki Rapti, Ourania Koltsida, Andreas Mentis, Nikolaos Koulouris, Sotirios Tsiodras, Antonia Koutsoukou & Evangelos Andreakos ✉

Nature immunology **22**, 32–40 (2021) | Cite this article

21k Accesses | 181 Citations | 95 Altmetric | Metrics

Abstract

A central paradigm of immunity is that interferon (IFN)-mediated antiviral responses precede pro-inflammatory ones, optimizing host protection and minimizing collateral damage[1,2]. Here, we report that for coronavirus disease 2019 (COVID-19) this paradigm does not apply. By investigating temporal IFN and inflammatory cytokine patterns in 32 moderate-to-severe patients with COVID-19 hospitalized for pneumonia and longitudinally followed for the development of respiratory failure and death, we reveal that IFN-λ and type I IFN production were both diminished and delayed, induced only in a fraction of patients as they became critically ill. On the contrary, pro-inflammatory cytokines such as tumor necrosis factor (TNF), interleukin (IL)-6 and IL-8 were produced before IFNs in all patients and persisted for a prolonged time. This condition was reflected in blood transcriptomes wherein prominent IFN signatures were only seen in critically ill patients who also exhibited augmented inflammation. By comparison, in 16 patients with influenza (flu) hospitalized for pneumonia with similar clinicopathological characteristics to those of COVID-19 and 24 nonhospitalized patients with flu with milder symptoms, IFN-λ and type I IFN were robustly induced earlier, at higher levels and independently of disease severity, whereas pro-inflammatory cytokines were only acutely produced. Notably, higher IFN-λ concentrations in patients with COVID-19 correlated with lower viral load in bronchial aspirates and faster viral clearance and a higher IFN-λ to type I IFN ratio correlated with improved outcome for critically ill patients. Moreover, altered cytokine patterns in patients with COVID-19 correlated with longer hospitalization and higher incidence of critical disease and mortality compared to flu. These data point to an untuned antiviral response in COVID-19, contributing to persistent viral presence, hyperinflammation and respiratory failure.

COVID-19

Hajar Owji [a], Manica Negahdaripour [a, b] ✉, Nasim Hajighahramani [c]

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Highlights

- Immunotherapy, as a viable approach to curtail COVID-19, could be done through:
- Active immunization by vaccines or direct interferon administration;
- Passive immunization by convalescent plasma or synthesized antibodies;
- Immunomodulatory drugs such as JAK inhibitors and corticosteroids.
- Immunotherapy should be adapted to the patient condition and disease stage.

Abstract

COVID-19, the disease induced by the recently emerged severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has imposed an unpredictable burden on the world. Drug repurposing has been employed to rapidly find a cure; but despite great efforts, no drug or vaccine is presently available for treating or prevention of COVID-19. Apart from antivirals, immunotherapeutic strategies are suggested considering the role of the immune response as the host defense against the virus, and the fact that SARS-CoV-2 suppresses interferon induction as an immune evasion strategy. Active immunization through vaccines, interferon administration, passive immunotherapy by convalescent plasma or synthesized monoclonal and polyclonal antibodies, as well as immunomodulatory drugs, are different immunotherapeutic approaches that will be mentioned in this review. The focus would be on passive immunotherapeutic interventions.

Interferons might be helpful in some stages. Vaccine development has been followed with unprecedented speed. Some of these vaccines have been advanced to human clinical trials. Convalescent plasma therapy is already practiced in many

Open Access Review

Type I Interferons in COVID-19 Pathogenesis

by Enrico Palermo [1,*], Daniele Di Carlo [1], Marco Sgarbanti [2] and John Hiscott [1,*]

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Simple Summary

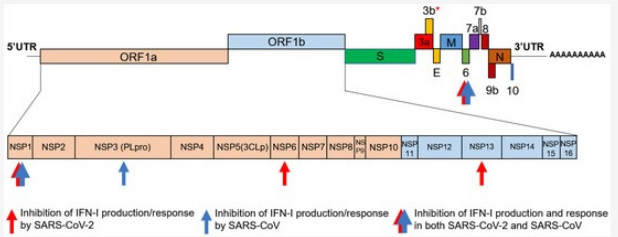
The innate antiviral immune response is essential to limit virus replication at early stages of infection, thus preventing viral spre and pathogenesis. Nevertheless, viruses have evolved different strategies to evade innate immune control. In this review, we describe recent findings delineating the relationship between SARS-CoV-2 and type I IFN response in vitro and in vivo and rep current studies using IFN-based therapy for COVID-19 treatment.

Abstract

Among the many activities attributed to the type I interferon (IFN) multigene family, their roles as mediators of the antiviral immu response have emerged as important components of the host response to Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection. Viruses likewise have evolved multiple immune evasion strategies to circumvent the host immune response and promote virus propagation and dissemination. Therefore, a thorough characterization of host–virus interactions is essential to understand SARS-CoV-2 pathogenesis. Here, we summarize the virus-mediated evasion of the IFN responses and the viral functions involved, the genetic basis of IFN production in SARS-CoV-2 infection and the progress of clinical trials designed to utilize type I IFN as a potential therapeutic tool. View Full-Text

Keywords: type I IFNs; innate immunity; SARS-CoV-2; COVID-19

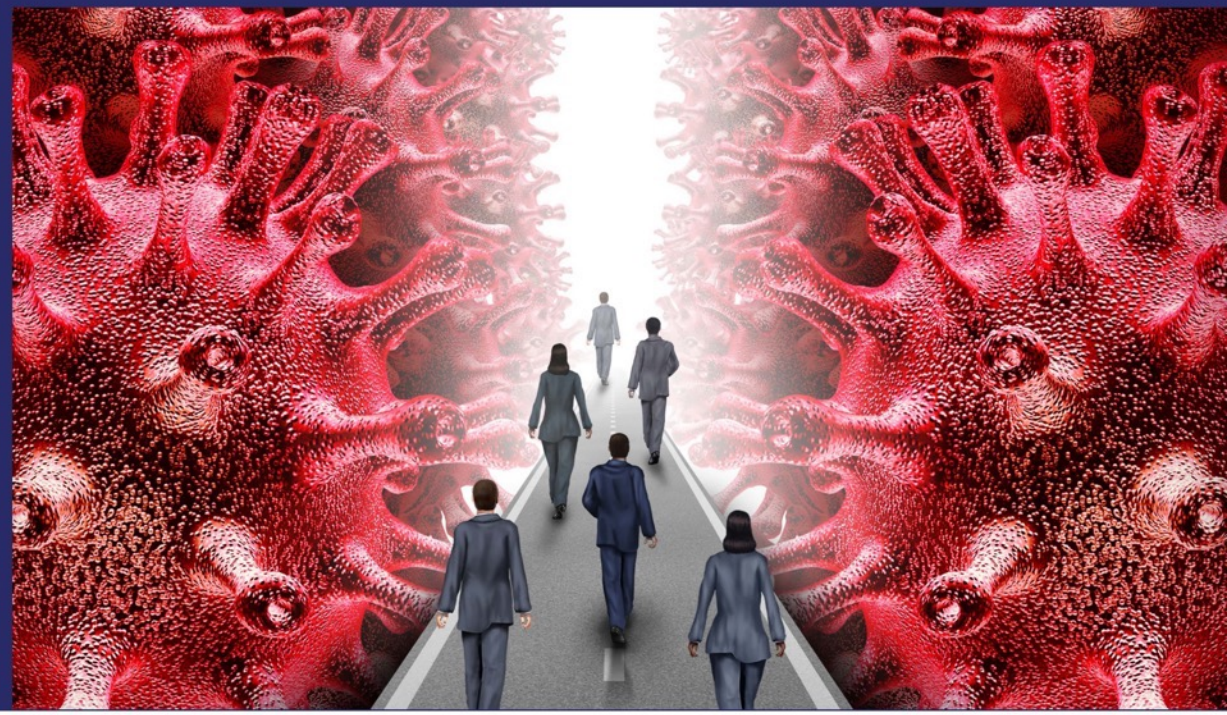
▼ Show Figures



COVID-19 appears to use mutations adaptively, in the service of its survival and expansion. It circumvents the errors accumulated by random amino acid switches that eradicated previous coronaviruses. To date the infectiousness of this pandemic is exponentially increasing, evolving into even more elusive pernicious variants. By apprehending the ACE2 receptors to contaminate human cells, COVID-19 neutralizes our primary anti-inflammatory and anti-fibrotic defences. The body counterattacks by unleashing chemokines, interleukins, leukocytes, TNF, CSF, but COVID-19 has a strategy: First, it overwhelms the innate response, then it manoeuvres to avoid exposure by inhibiting the adaptive mechanisms of viral recognition. Undetected, COVID- 19 multiplies, while the immune system is blindly shooting in the dark, ravaging the vital organs of the host that is fatally injured by the cytokine storm. Vaccines' safety and effectiveness is evaluated along with new therapeutics. The focus is on COVID-19 susceptibility factors, hormonal imbalance, elevated glucose and lipids, obesity, and the male gender. Preventive methods designed to empower immunity are explored.

Dr. Xanya Sofra, Ph.D Neurophysiology, UK / Ph.D Clinical Psy NYC, USA. She is an award-winning international speaker, author of several scientific articles, and the inventor of anti-inflammatory nanotechnology for fitness and bio-repair. Her research explores cellular networks, and the dynamics of molecular mechanisms involved in time reversal.

Checkmate by a Protean Invisible Enemy



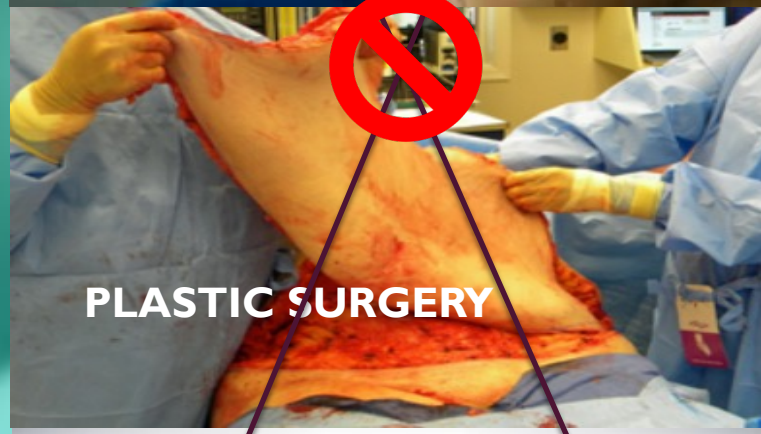
Xanya Sofra

Checkmate by a Protean Invisible Enemy

COVID-19: The Danger Within

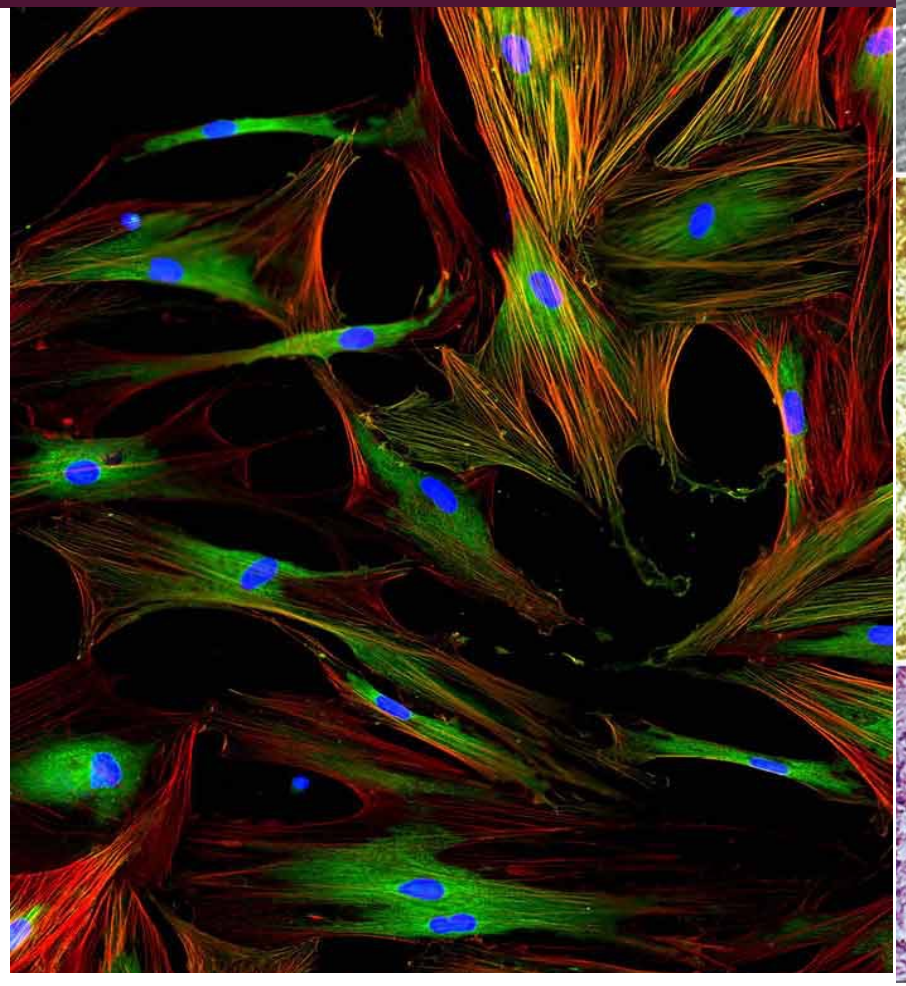
Fatty Liver

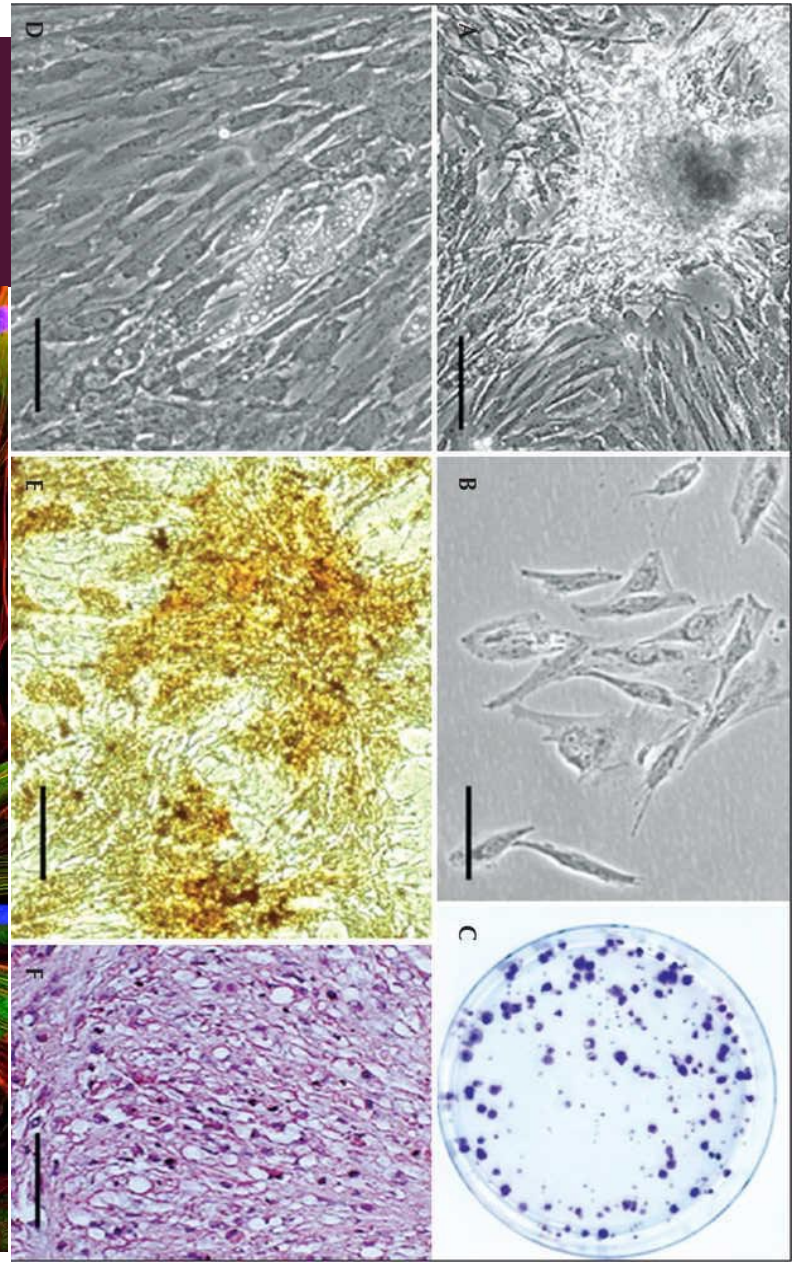
VISCERAL FAT CANNOT BE REMOVED BY LASES OR RF



Adipose Tissue Derived Mesenchymal Stem Cells

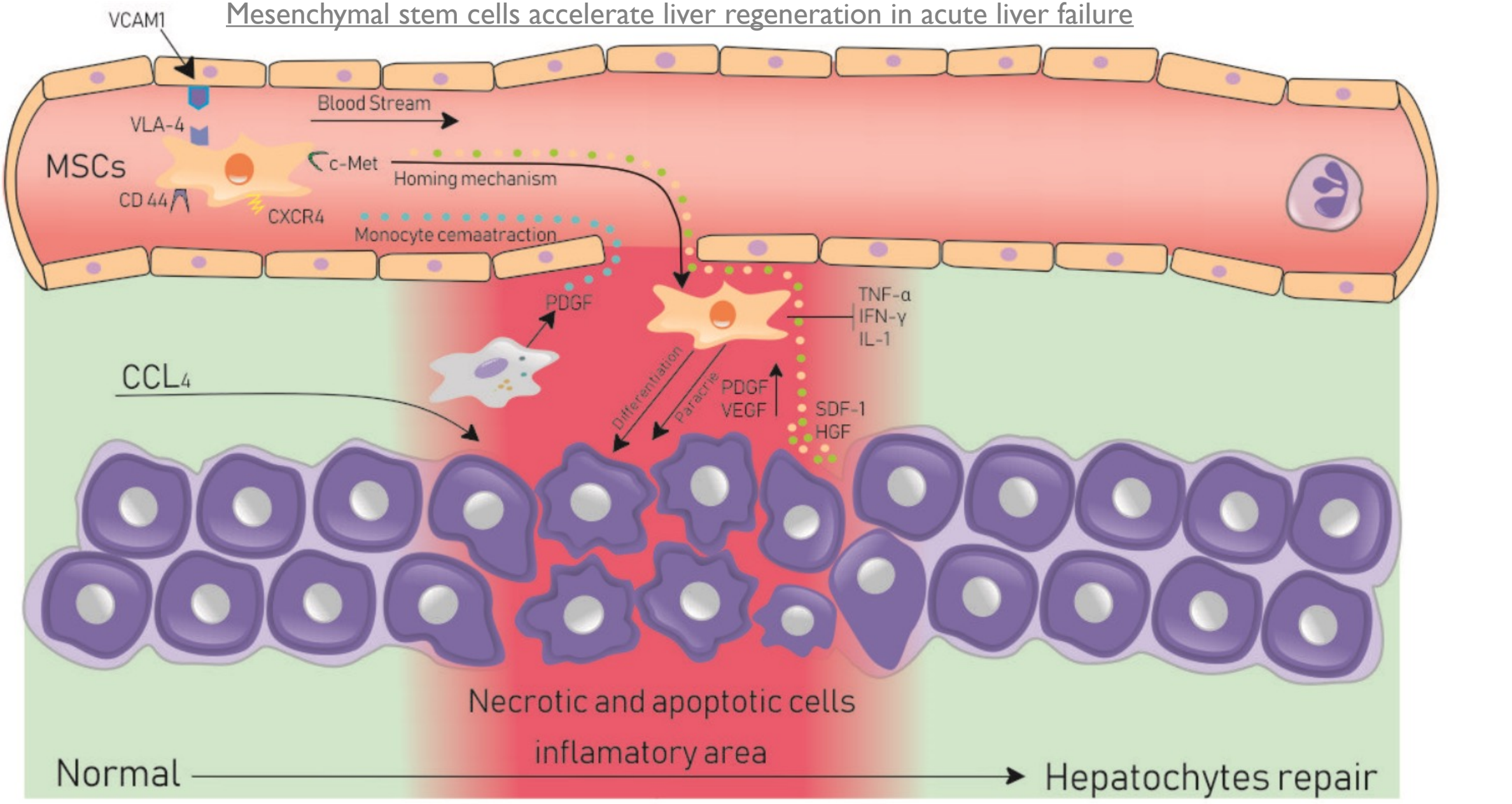
- Mesenchymal Stem Cells from Adipose Tissue REPAIR LIVER
- MSCs are at a rate of 1 in 100 in adipose tissue in contrast to 1 in 100,000 in the bone marrow
- VIRTUAL GYM RESULTS IN THE NATURAL RELEASE OF THE FAT CONTENTS INTO THE BLOODSTREAM. THESE CONTAIN STEM CELLS THAT DIFFERENTIATE TO MESENCHYMAL STEM CELLS THAT REPAIR THE LIVER AND HEPATOCYTES THAT DETOX THE LIVER

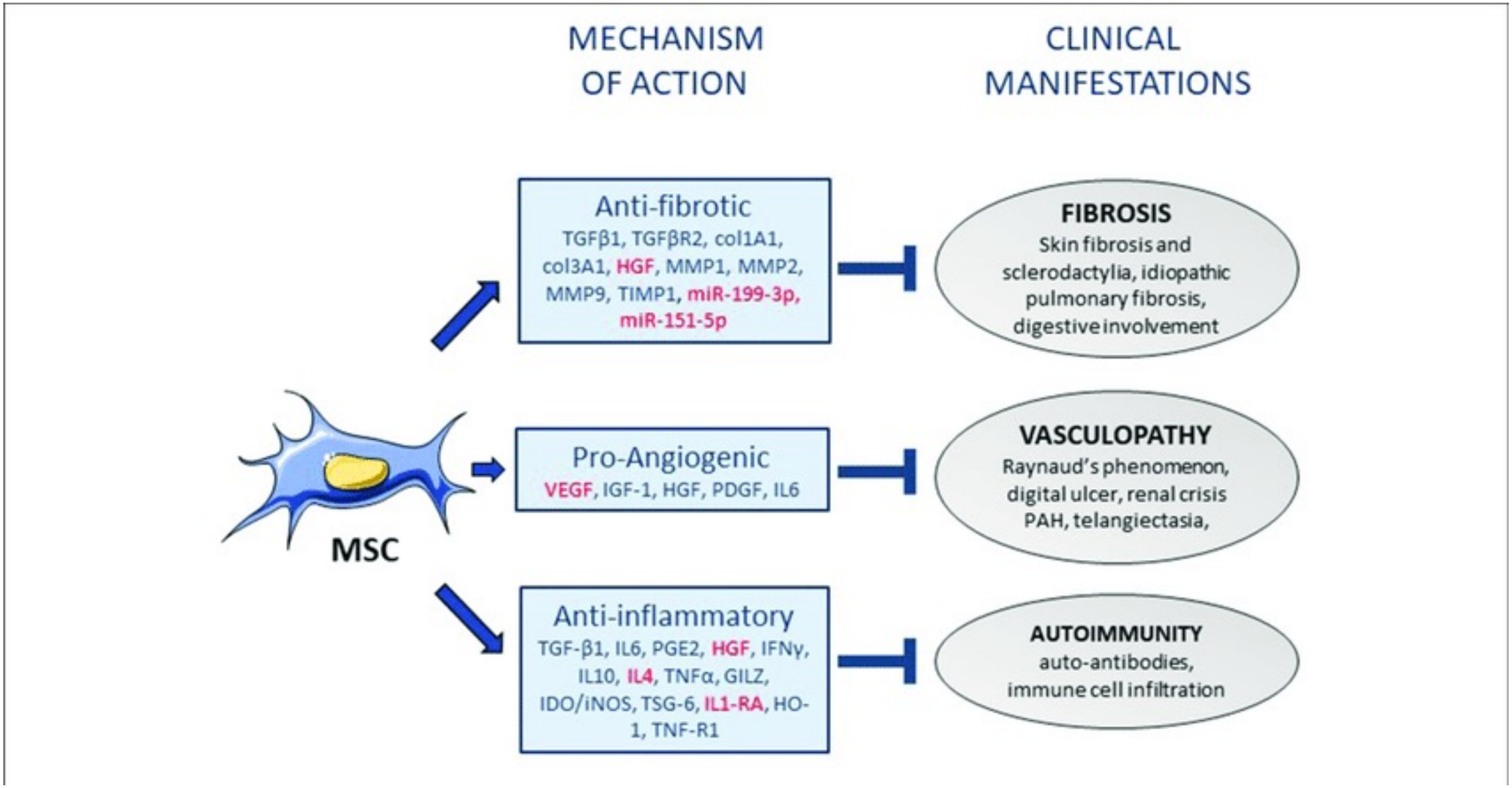




Biomedical Research and Therapy

Mesenchymal stem cells accelerate liver regeneration in acute liver failure




MECHANISM OF ACTION
CLINICAL MANIFESTATIONS
Anti-fibrotic
TGFβ1, TGFβR2, col1A1, col3A1, HGF, MMP1, MMP2, MMP9, TIMP1, miR-199-3p, miR-151-5p
FIBROSIS
Skin fibrosis and sclerodactylia, idiopathic pulmonary fibrosis, digestive involvement
MSC
Pro-Angiogenic
VEGF, IGF-1, HGF, PDGF, IL6
VASCULOPATHY
Raynaud's phenomenon, digital ulcer, renal crisis PAH, telangiectasia,
Anti-inflammatory
TGF-β1, IL6, PGE2, HGF, IFNγ, IL10, IL4, TNFα, GILZ, IDO/iNOS, TSG-6, IL1-RA, HO-1, TNF-R1
AUTOIMMUNITY
auto-antibodies, immune cell infiltration

PREDICTORS OF NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

- **Elevated Alanine Aminotransferase (ALT) – an enzyme found in the liver / tests for hepatitis**
- **Elevated Aspartate Aminotransferase (AST) – enzyme testing for liver damage**
- **Elevated Alkaline Phosphatase, ALP – enzyme flags damage in liver, gallbladder, bones**
- **Elevated Creatinine – a waste product made by the muscles**
- **Elevated Triglycerides**
- **Increased inflammation as measured by CRP**
- **Insulin Resistance**
- **High Glucose (Diabetes)**
- **Obesity**
- **Metabolic Syndrome**

Symptoms of Elevated ALT, AST and ALP NAFLD indicators:

- Weakness
- Fatigue
- Swollen belly
- Stomach pain
- Appetite loss
- Itchy skin
- Dark-coloured urine
- Light-coloured stool
- Edema (swollen arms and fingers)
- Edema (swollen legs and ankles)
- Nausea
- Vomiting
- Bruises
- Jaundice (yellow skin, eyes and nails)

Table 1. Blood Test Results on ALT (SGPT), AST (SGOT), ALP and Albumin
ALT Normal Range: 0-32 IU/L
AST Normal Range: 0-40 IU/L
ALP Normal Range: 44-121 IU/L
Albumin Normal Range: 3.8-4.8

Gender Age	Medical History	ALT IU/L Pre	ALT IU/L Post	AST IU/L Pre	AST IU/L Post	ALP IU/L Pre	ALP IU/L Post	Albumin g/dL Pre	Albumin g/dL Post
F/64	Fatty Liver Prediabetes	28	24	38	31	109	89	3.0	3.9
F/58	Fatty Liver Prediabetes	34	25	39	28	117	92	3.4	4.3
F/59	Fatty Liver Prediabetes	33	26	41	30	114	87	3.1	4.1
F/54	Fatty Liver Prediabetes	36	23	39	29	120	105	3.6	4.2
F/62	Fatty Liver Prediabetes	29	22	41	26	122	112	3.2	4.0
M/54	Fatty Liver Prediabetes	27	19	40	22	119	106	3.3	4.3
M/57	Fatty Liver Prediabetes	32	21	36	24	112	98	3.5	4.0
M/59	Fatty Liver Prediabetes	31	26	38	31	118	102	3.1	3.9
M/60	Fatty Liver Prediabetes	27	22	39	18	121	104	3.7	3.9
M/55	Fatty Liver Prediabetes	33	25	42	29.	118	105	3.3	4.0
Mean Total		31	23.3	39.3	26.8	117	100	3.32	4.06
		ALT Average Decrease: -24.83%		**AST Average Decrease: -30.407%**		**ALP Average Decrease: -14.529%**		**Albumin Average Increase: +%22.289**	
		Value of t= -8.724 The value is p=0.00001 Significance: p<0.0001		**Value of t= -8.83 The value is p=0.00001. Significance: p<0.0001**		**Value of t= -10.8912 The value is p=0.00001. Significance: p<0.0001**		**Value of t=+9347886 The value is p<0.00001. Significance: p<0.00001**	

Table 2. Subjects results on Creatinine and Bilirubin & Ultrasonography Results
Creatinine Normal Range: 0.5-1.10 mg/dL
Bilirubin Normal Range: 0.3-1.2 \mg/dL

Gender/ Age	Medical History	Creatinine Serum PRE mg/dL	Creatinine Serum POST mg/dL	Bilirubin PRE mg/dL	Bilirubin POST mg/dL	Ultrasonography Results
F/64	Prediabetes Fatty Liver	1.35	0.94	1.13	1.09	Significantly Improved Liver
F/58	Prediabetes Fatty Liver	1.23	0.87	1.29	1.18	Normal Liver
F/59	Prediabetes Fatty Liver	1.26	1.05	1.23	1.14	Normal Liver
F/54	Prediabetes Fatty Liver	1.33	0.96	1.75	1.19	Normal Liver
F/62	Prediabetes Fatty Liver	1.25	1.02	1.21	1.15	Significantly Improved Liver
M/54	Prediabetes Fatty Liver	1.13	1.01	1.27	1.19	Normal Liver
M/57	Prediabetes Fatty Liver	1.16	0.82	1.23	1.12	Normal Liver
M/59	Prediabetes Fatty Liver	1.18	0.98	1.41	1.15	Significantly Improved Liver
M/60	Prediabetes Fatty Liver	1.11	0.87	1.22	1.17	Normal Liver
M/55	Prediabetes Fatty Liver	1.96	1.23	1.47	1.20	Normal Liver
MEAN TOTAL		1.22 mg/dL	0.98 mg/dL	1.321 mg/dL	1.158 mg/dL	

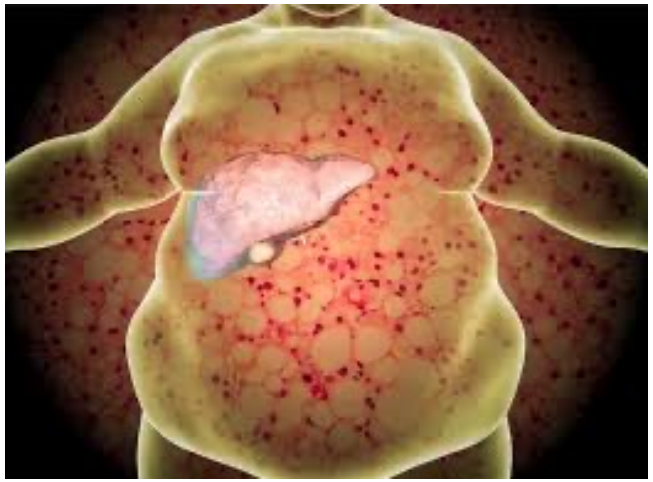
Mean Average Creatinine % Decrease -19.67% mg/dL

Value of t=-59420
The value is p=0.00011. Significance: p<0.001

Mean Average Bilirubin % Increase -12. 33% mg/dL

Value of t=-3.1911
The value is p=0.00549.
Significance: p<0.01

Our Results on Fatty Liver



TWENTY VIRTUAL GYM TREATMENTS

Table 3. TYPE 2 DIABETICS
Triglycerides, High-Density Protein (HDL),
Presence of Fatty Liver on Sonography Reports Pre and Post Treatment.

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

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%**	

Table 3. Blood Test Results on TG, VLDL and CRP
TG Normal Range: 0-149 mg/dL
VLDL Normal Range: 5-40 mg/dL
CRP Normal Range: 0-10 mg/L

Gender Age	Medical History	TG mg/dL Pre	TG mg/dL Post	VLDL mg/dL Pre	VLDL mg/dL Post	CRP mg/L Pre	CRP mg/L Post
F/64	Fatty Liver Prediabetes	195	146	45	32	14	9
F/58	Fatty Liver Prediabetes	193	147	43	35	12	7
F/59	Fatty Liver Prediabetes	167	123	41	34	11	8
F/54	Fatty Liver Prediabetes	156	129	38	31	12	9
F/62	Fatty Liver Prediabetes	178	134	48	36	15	10
M/54	Fatty Liver Prediabetes	188	139	40	29	13	8
M/57	Fatty Liver Prediabetes	183	141	42	32	15	9
M/59	Fatty Liver Prediabetes	191	146	37	28	10	7
M/60	Fatty Liver Prediabetes	172	132	39	29	11	8
M/55	Fatty Liver Prediabetes	159	115	43	32	13	9
Mean Average		178.2	135.2	41.6	31.8	12.6	8.4
		TG % decrease: -24.130%		VLDL % decrease: -23.55%		CRP % decrease: -33.333%	
		Value of t=-7.431 The value is p=0.00002. Significance: p<0.0001		Value of t=-9.175 The value is p<0.00001. Significance: p<0.00001		Value of t=-11.698 The value is p<0.00001. Significance: p<0.00001	

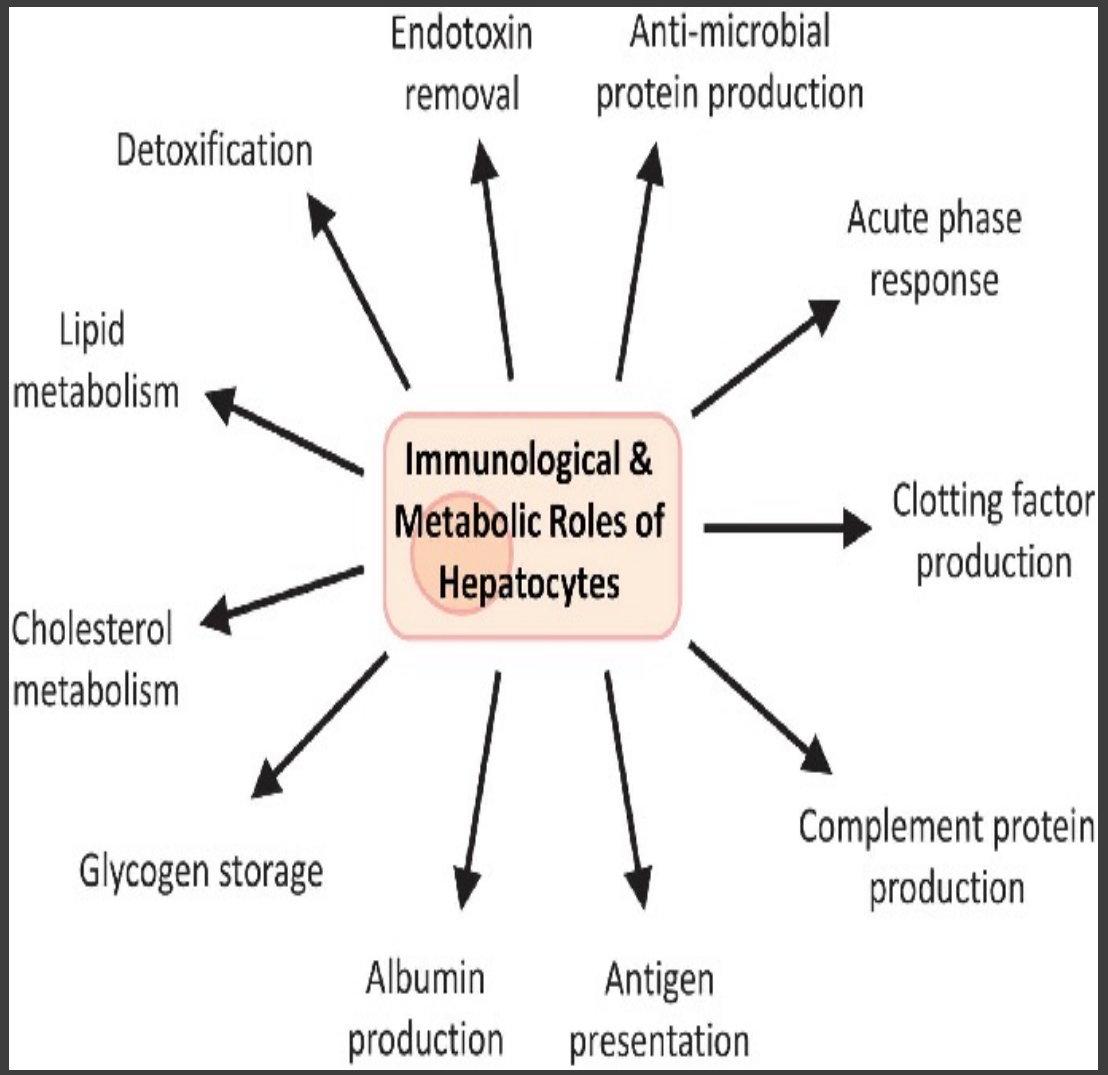
TABLE 5. BLOOD TEST RESULTS ON CORTISOL (F), TESTOSTERONE (T) AND MUSCLE MASS (MM)
CORTISOL NORMAL RANGE MEN AND WOMEN: 80-477 NMOL/L
TESTOSTERONE NORMAL RANGE MEN: 10-35 NMOL/L
TESTOSTERONE NORMAL RANGE WOMEN: 0.5 – 2.4 NMOL/L
MUSCLE MASS MALE AND FEMALE AGES 54-64: AVERAGE; 33-55 LOW: < 33 HIGH:>55

Gender Age	Medical History	Cortisol Pre	Cortisol Post	T Pre	T Post	MM Pre	MM Post
F/64	Fatty Liver Prediabetes	481	319	0.4	1.27	24	36
F/58	Fatty Liver Prediabetes	455	247	0.6	1.26	29	38
F/59	Fatty Liver Prediabetes	462	325	0.5	1.38	26	40
F/54	Fatty Liver Prediabetes	449	354	0.8	1.44	25	39
F/62	Fatty Liver Prediabetes	396	286	0.7	1.22	23	42
M/54	Fatty Liver Prediabetes	476	368	11.99	18.54	29	49
M/57	Fatty Liver Prediabetes	451	312	12.89	19.33	30	51
M/59	Fatty Liver Prediabetes	479	347	11.92	17.62	29	46
M/60	Fatty Liver Prediabetes	478	366	12.12	17.57	26	38
M/55	Fatty Liver Prediabetes	429	325	14.7	20.33	31	52
Mean Average		455.6	324.9	6.36	9.996	27.2	43.1
		Cortisol % decrease: -28.687%		Testosterone % INCREASE: +50.04%		Muscle Mass % INCREASE: +58. 45%	
		Value of t=-14.01212.190 The value is p<0.00001 Significance: p<0.00001		Value of t=+3.786 The value is p=0.00215 Significance: p<0.01		Value of t=+11.746 The value is p<0.00001 Significance: p<0.00001	

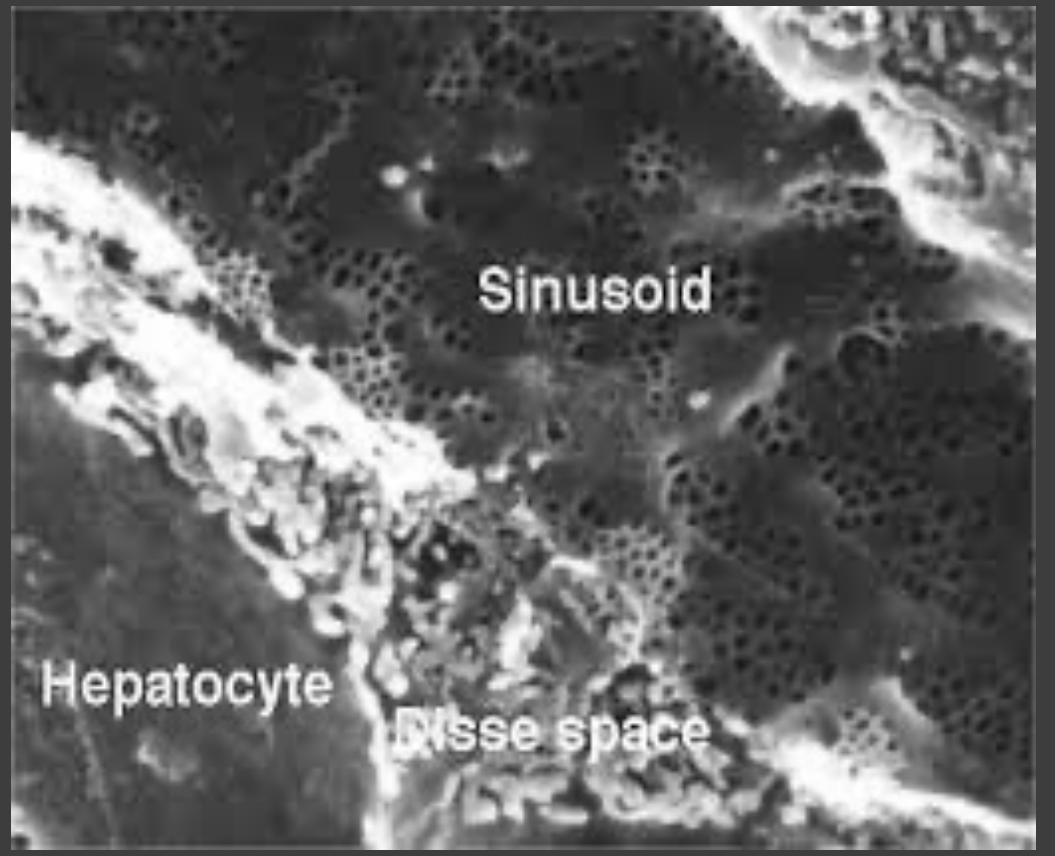
TABLE 4. BMI, BMR AND VAT
BMI NORMAL RANGE MEN (DEPENDING ON WEIGHT AND HEIGHT): 1-24
BMI NORMAL RANGE WOMEN (DEPENDING ON WEIGHT AND HEIGHT): 1-23
BMR NORMAL RANGE MEN: 1600-1800 CAL/ PER DAY.
BMR NORMAL RANGE WOMEN: 1550. CAL/PER DAY
VAT (RANGES FROM 1-59) NORMAL RANGE: 1-12

Gender Age	Medical History	BMI Pre	BMI Post	BMR Pre	BMR Post	VAT Pre	VAT Post
F/64	Fatty Liver Prediabetes	34.2	26.5	920	1490	39	27
F/58	Fatty Liver Prediabetes	33.5	25.9	1005	1510	33	21
F/59	Fatty Liver Prediabetes	30.4	24.7	1156	1499	51	32
F/54	Fatty Liver Prediabetes	32.3	26.6	1098	1620	39	23
F/62	Fatty Liver Prediabetes	30.8	24.9	953	1457	42	29
M/54	Fatty Liver Prediabetes	31.6	25.7	1167	1663	48	31
M/57	Fatty Liver Prediabetes	31.1	24.8	1249	1833	35	26
M/59	Fatty Liver Prediabetes	32.4	27.4	1055	1692	39	28
M/60	Fatty Liver Prediabetes	31.2	26.3	1012	1757	41	27
M/55	Fatty Liver Prediabetes	32.4	25.3	1179	1633	43	29
Mean Average		31.9	25.81	1079.4	1615.4	41	27.3
		BMI % decrease: -19.09%		BMR% INCREASE: +49.650%		VAT % decrease: -33.41%	
		Value of t=-14.012 The value is p<0.00001. Significance: p<0.00001		Value of t=+15.685 The value is p<0.00001 Significance: p<0.00001		Value of t=-12.064 The value is p<0.00001 Significance: p<0.00001	

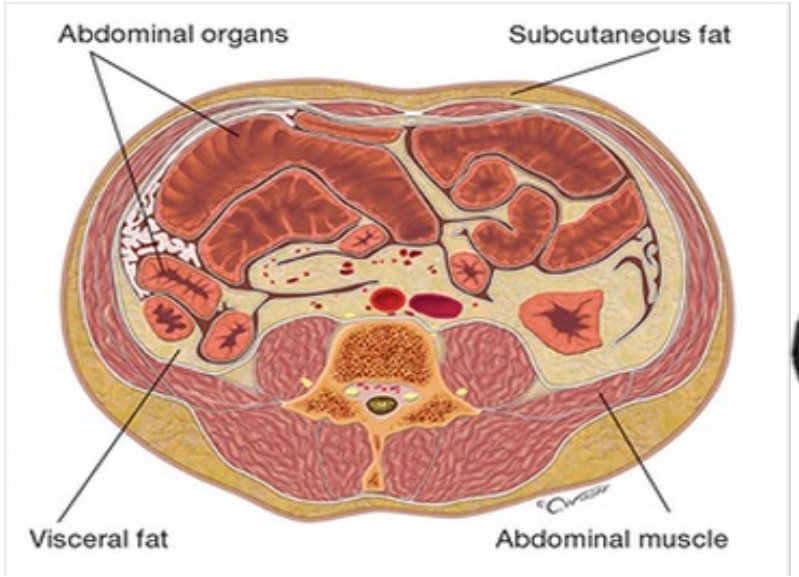
MSCs differentiate into Hepatocytes

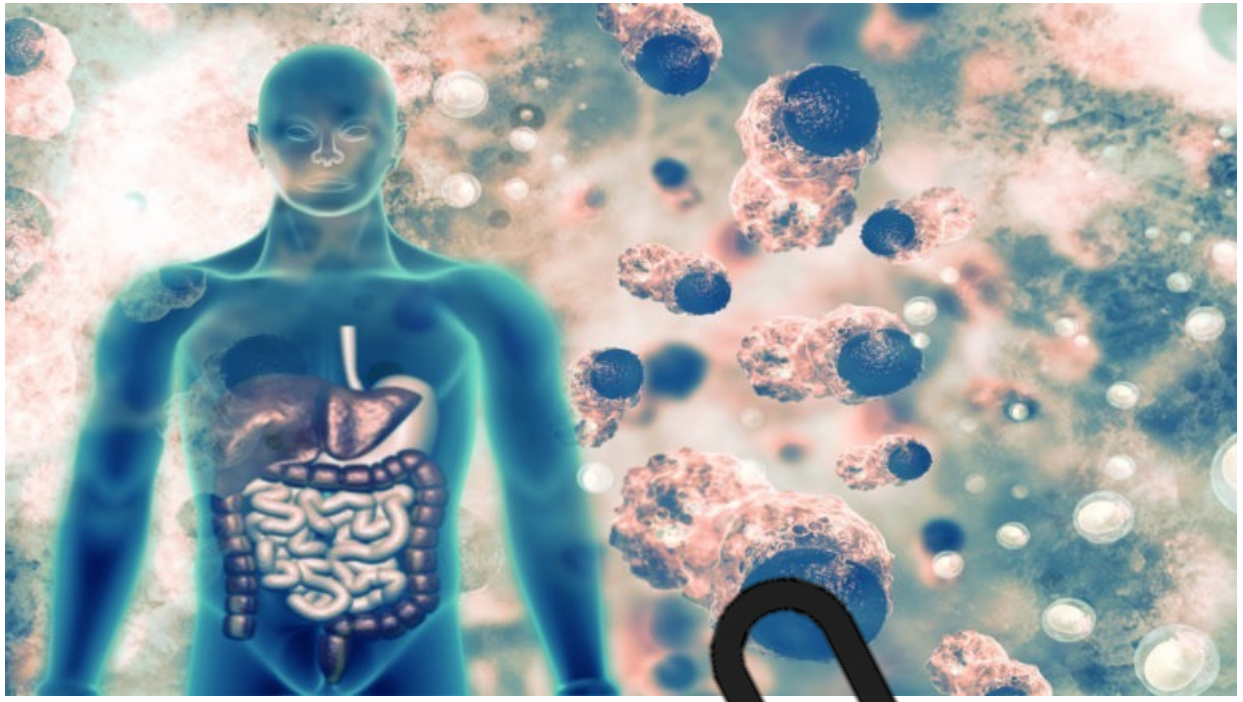


*Hepatocytes are involved in Liver Detox

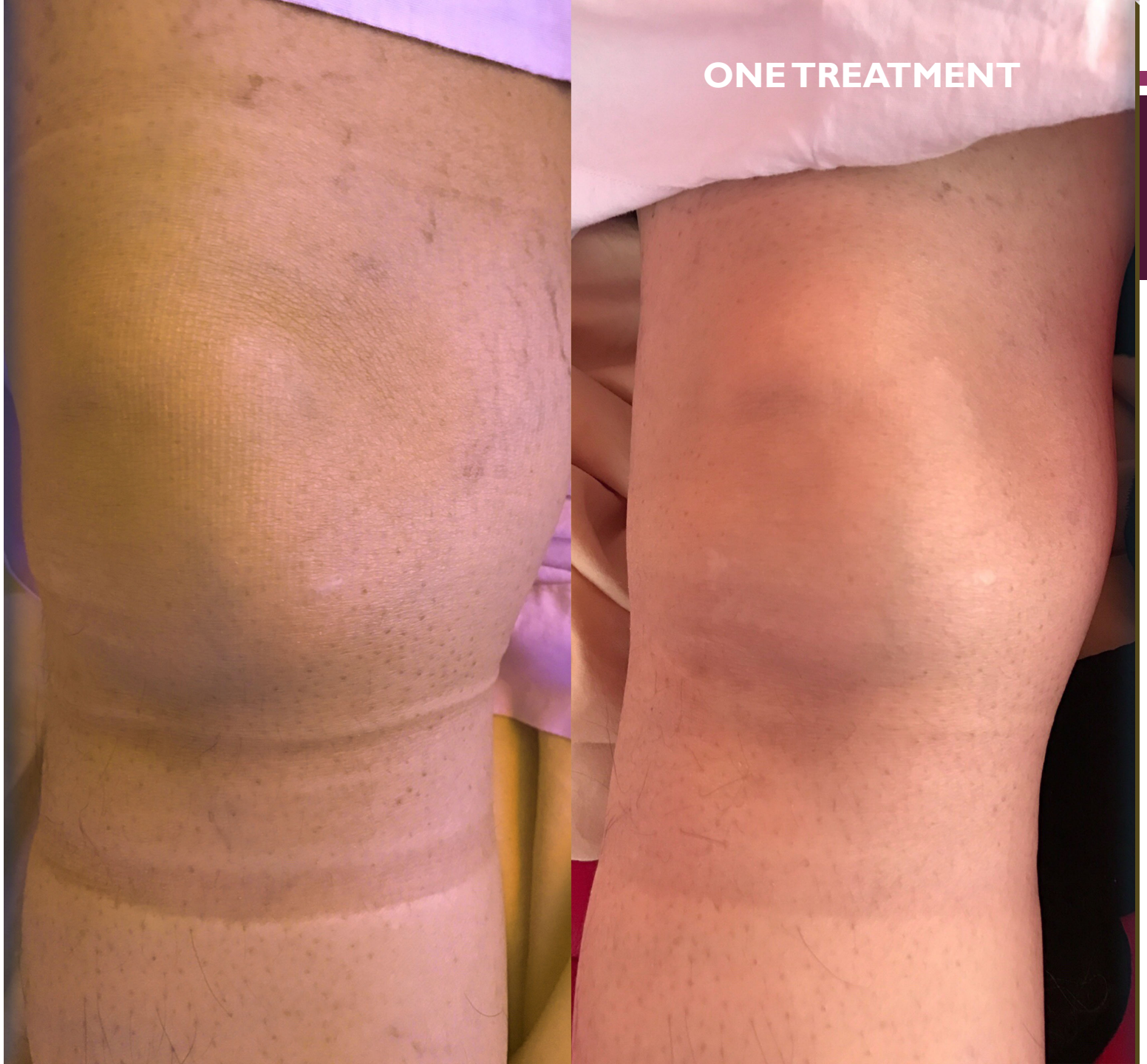


Toxicity in Visceral Adiposity Increases HUNGER





Toxicity that is inherent in visceral adiposity, overloads & compromises hepatic detoxification systems, promoting insulin and leptin resistance / increasing ghrelin, one of the central stimulators of appetite, ultimately promoting increased hunger



DETOX & INFLAMMATION DECREASE

CLINICAL STUDY

Virtual Gym
Detox – A very Important Function

The More Toxic you are
The More Hungry you are

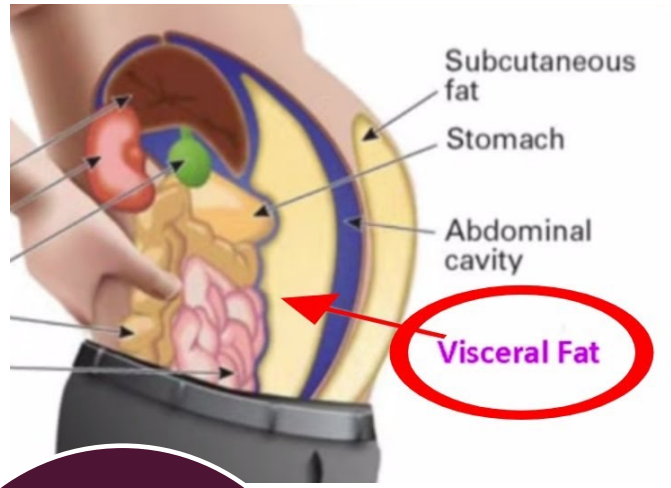
HORMONAL IMBALANCE

HUNGER

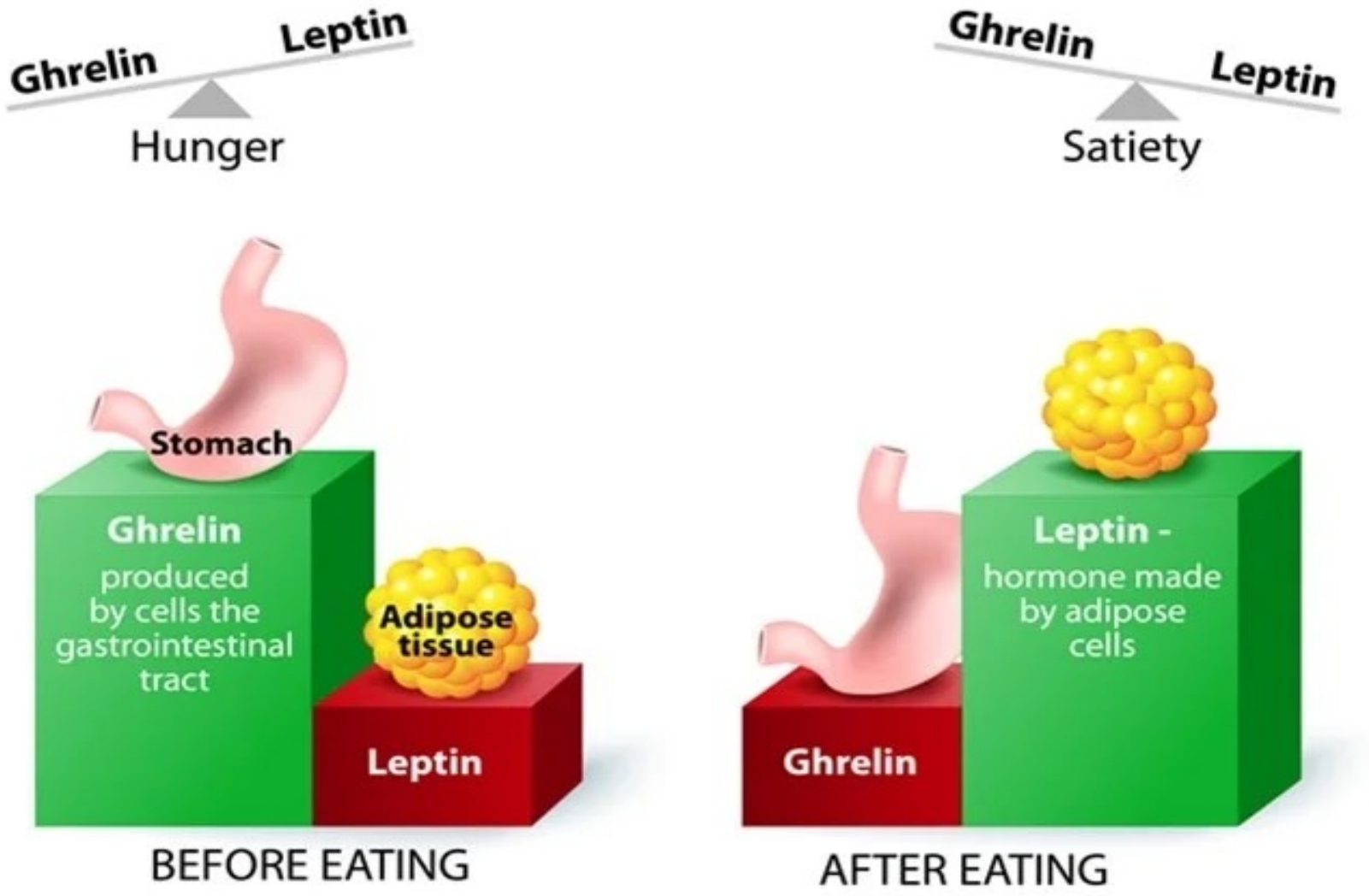
CONSUME FOOD

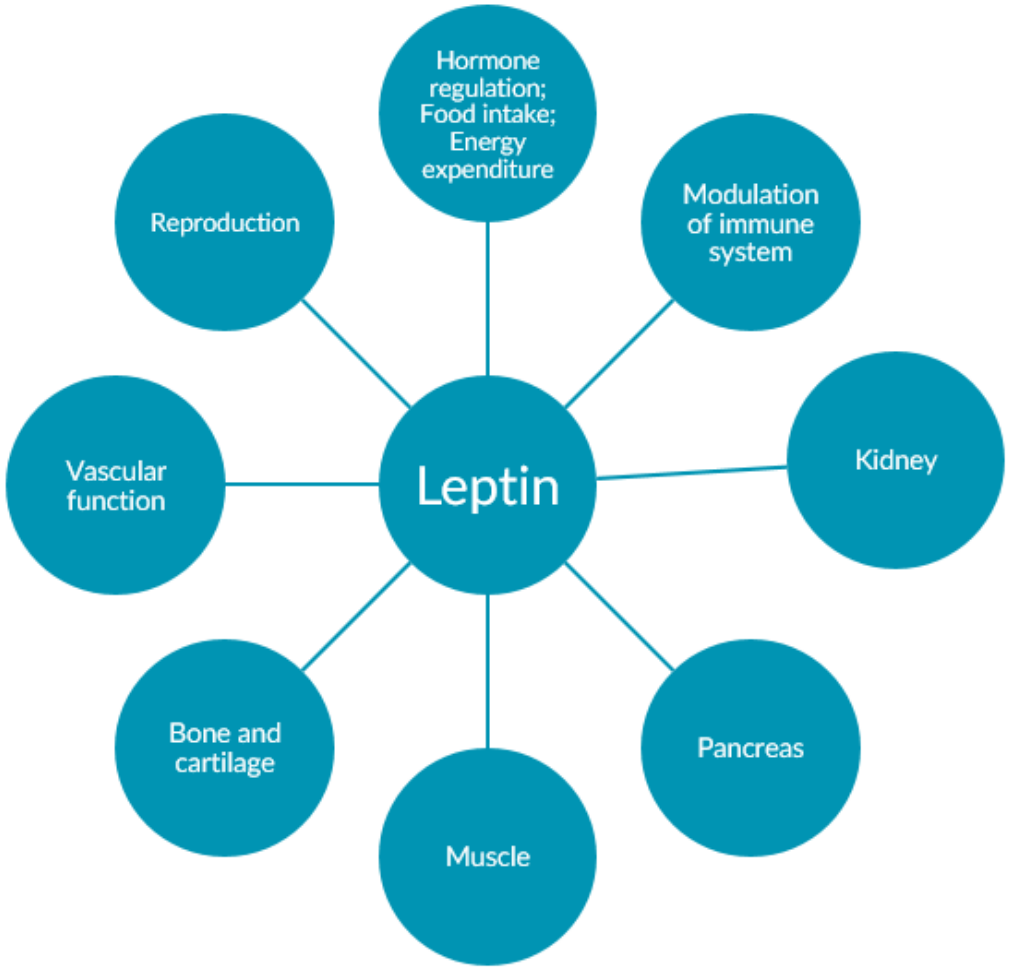
WEIGHT GAIN

TOXICITY



LEPTIN & GHRELIN IMBALANCE = HUNGER




Leptin
Hormone regulation; Food intake; Energy expenditure
Modulation of immune system
Kidney
Pancreas
Muscle
Bone and cartilage
Vascular function
Reproduction

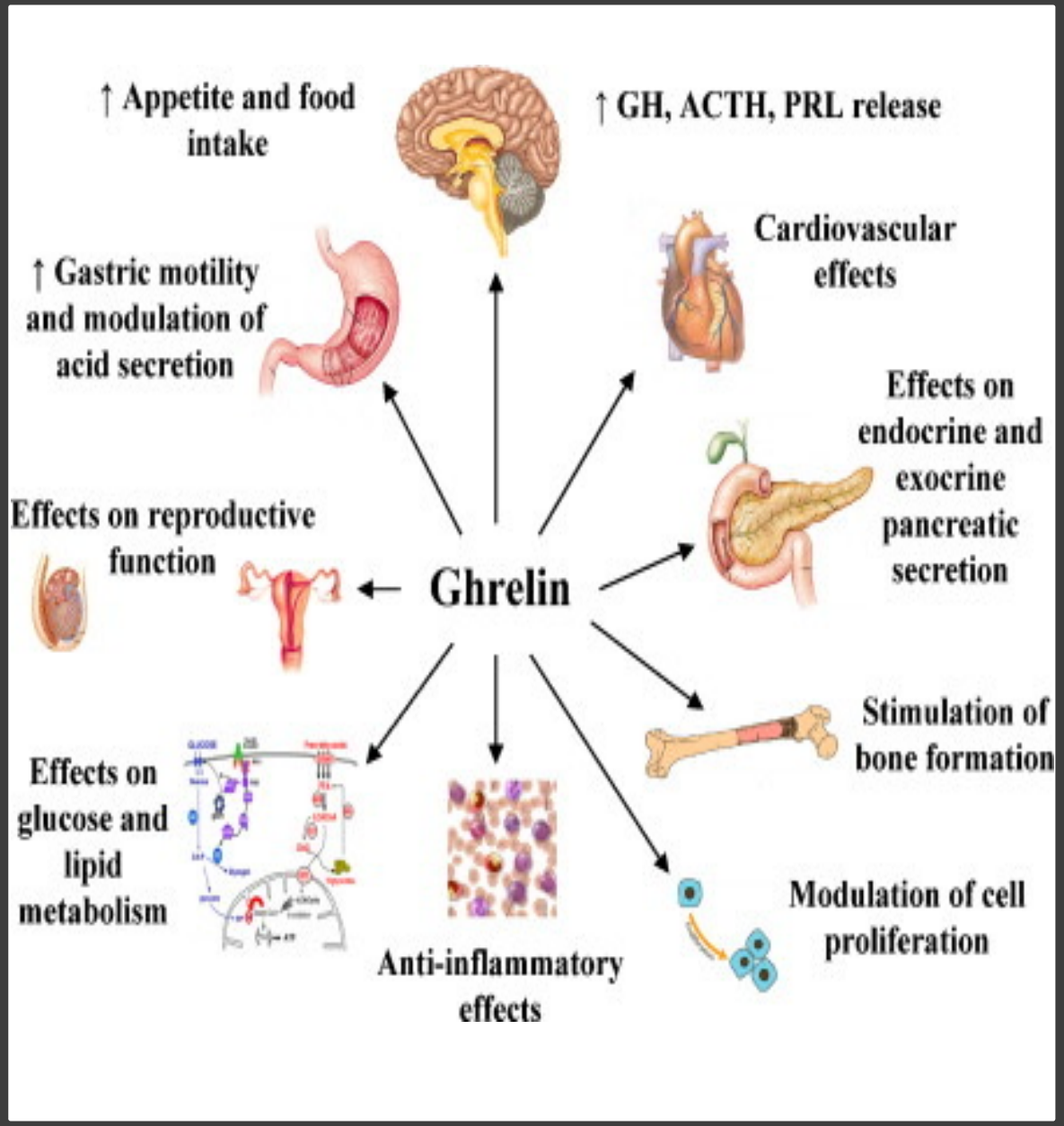

↑ Appetite and food intake
↑ GH, ACTH, PRL release
↑ Gastric motility and modulation of acid secretion
Cardiovascular effects
Effects on endocrine and exocrine pancreatic secretion
Effects on reproductive function
Ghrelin
Stimulation of bone formation
Effects on glucose and lipid metabolism
Anti-inflammatory effects
Modulation of cell proliferation

Table 6. Blood Plasma Results on Leptin and Ghrelin for each subject.

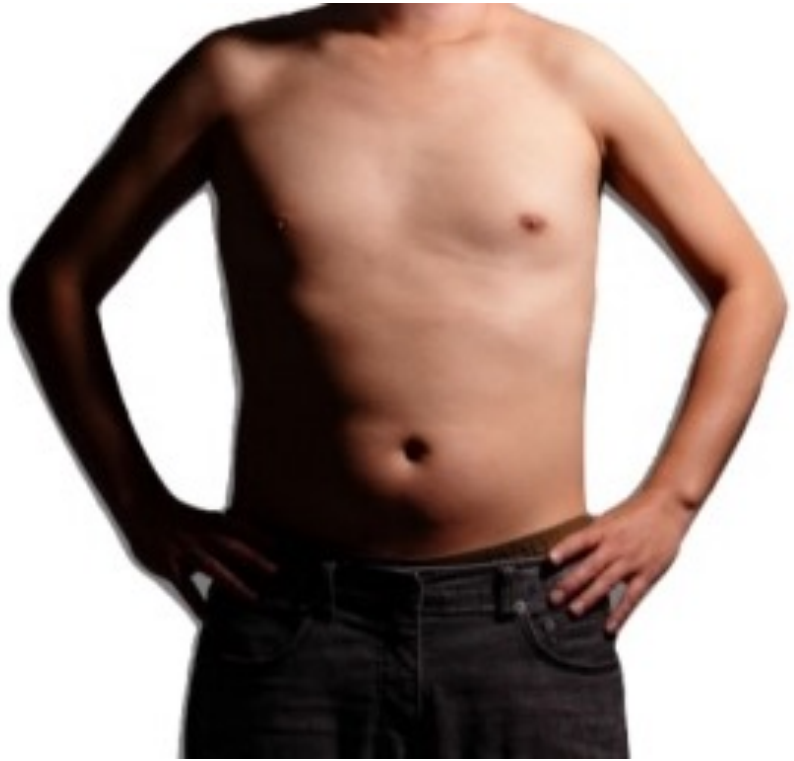
Gender	Age	Ethnicity	Leptin pre ng/mL	Leptin post ng/mL	Normal range ng/mL	% increase ng/mL	Ghrelin pre pg/mL	Ghrelin post pg/mL	Normal range pg/mL	% decrease pg/mL
Male	36	Asian	3.69	3.98	1.2 - 9.5	7.86%	687	602	520 - 700	12.37%
Male	39	Caucasian	4.43	4.98	1.2 - 9.5	9.78%	695	634	520 - 700	8.77%
Male	43	Caucasian	5.62	6.22	1.2 - 9.5	10.68%	598	552	520 - 700	7.69%
Male	35	Asian	6.15	6.83	1.2 - 9.5	11.05%	629	587	520 - 700	6.68%
Female	42	Asian	9.16	9.74	4.1 - 25.0	6.33%	577	542	520 - 700	6.06%
Female	45	Indian	5.23	6.09	4.1 - 25.0	16.44%	659	613	520 - 700	6.99%
Female	49	Caucasian	7.22	8.17	4.1 - 25.0	13.15%	644	617	520 - 700	4.19%
Female	38	Caucasian	12.34	13.22	4.1 - 25.0	7.13%	569	536	520 - 700	5.79%
Female	37	Asian	11.38	13.08	4.1 - 25.0	14.93%	499	461	520 - 700	7.62%
		Mean Average Leptin Increase				+10.82%	Mean Average Ghrelin Decrease			−7.35%

There was an inverse relationship between leptin and ghrelin where leptin significantly increased and ghrelin significantly decreased within the normal range. Mean average percentage leptin increase was +10.82% and ghrelin decrease was −7.35%.

SLIMMING VS FITNESS

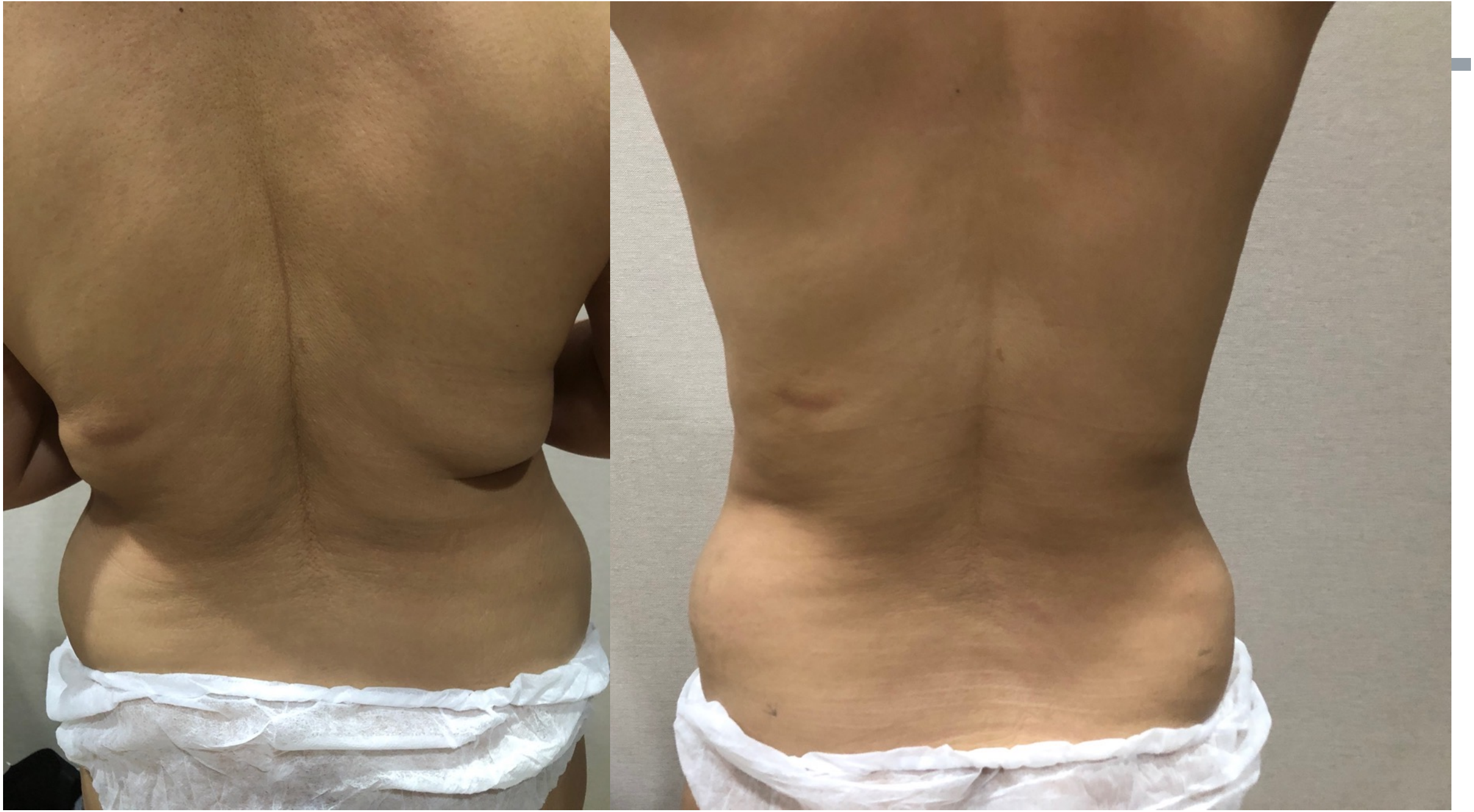
Lasers / RF Slimming
- Do NOT increase Metabolism
- Do NOT balance Hormones
- Do NOT decrease Hunger

Results Rebound



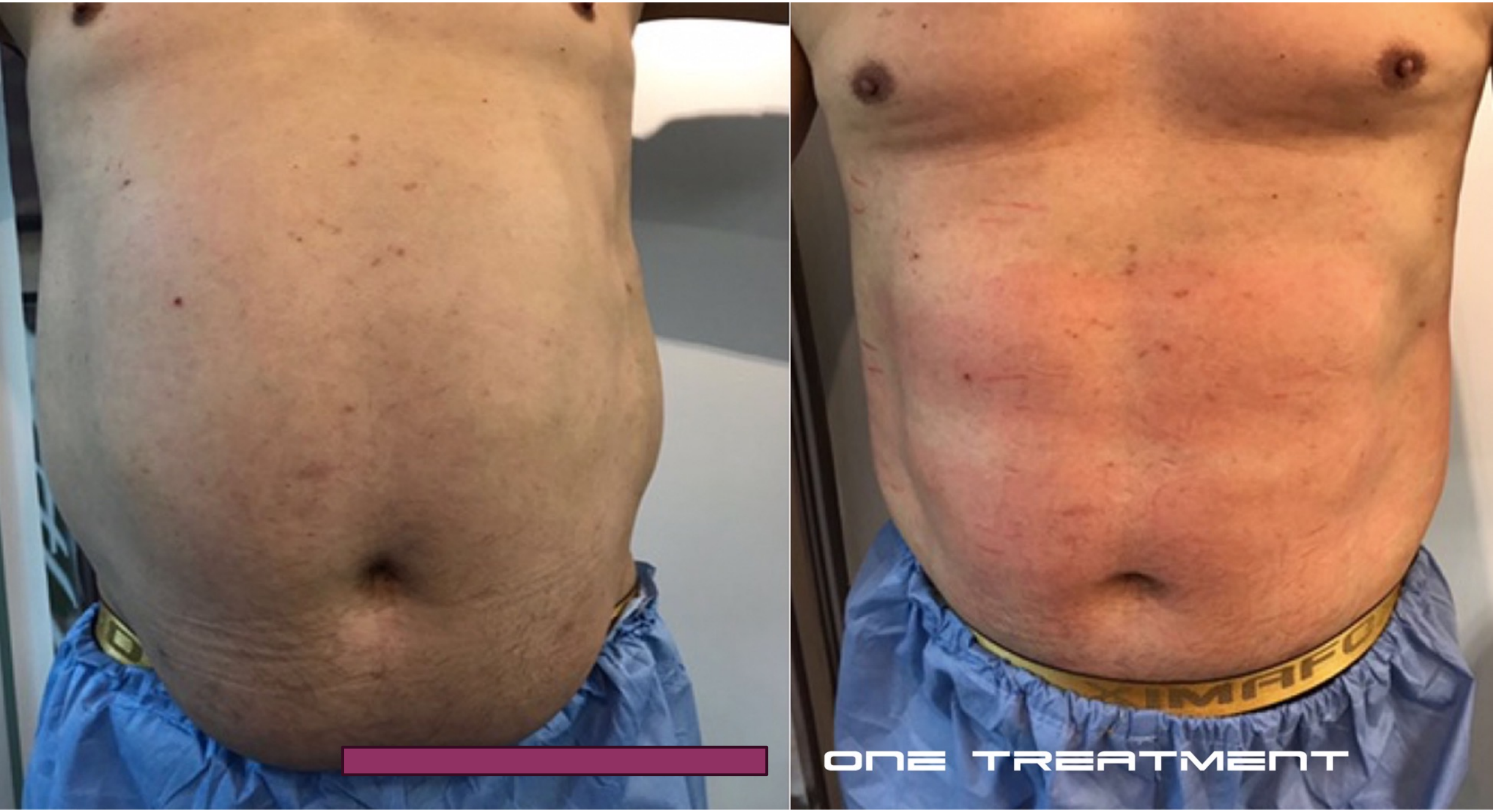
FITNESS
- Increases Metabolism
- Balances Hormones
- Reduces Hunger

NO Rebound



ONE TREATMENT


ONE
TREATMENT

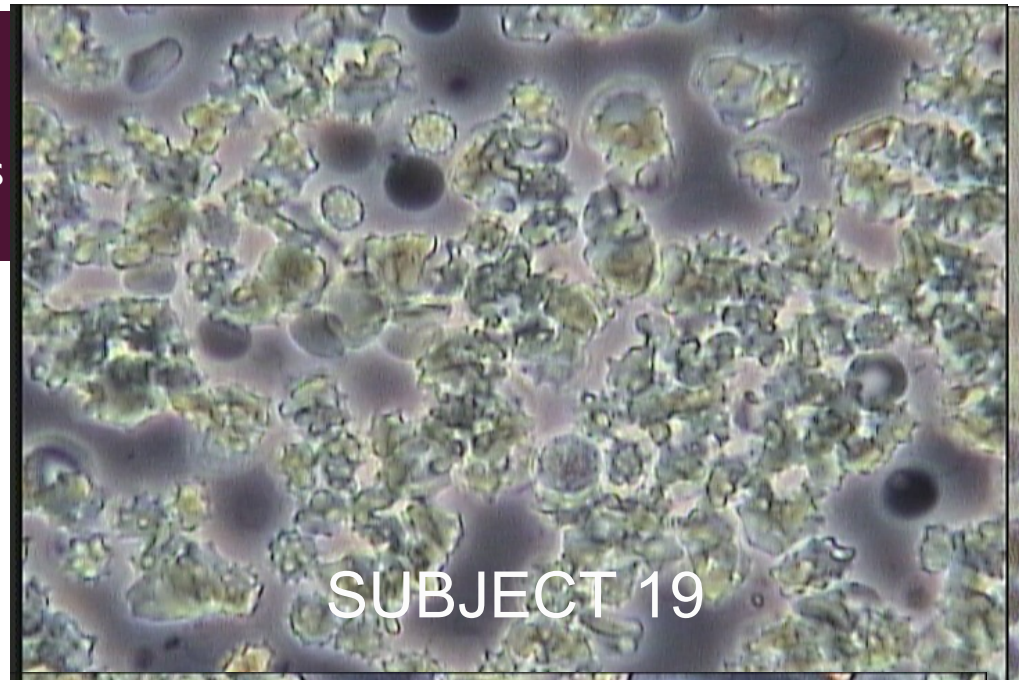

ONE TREATMENT

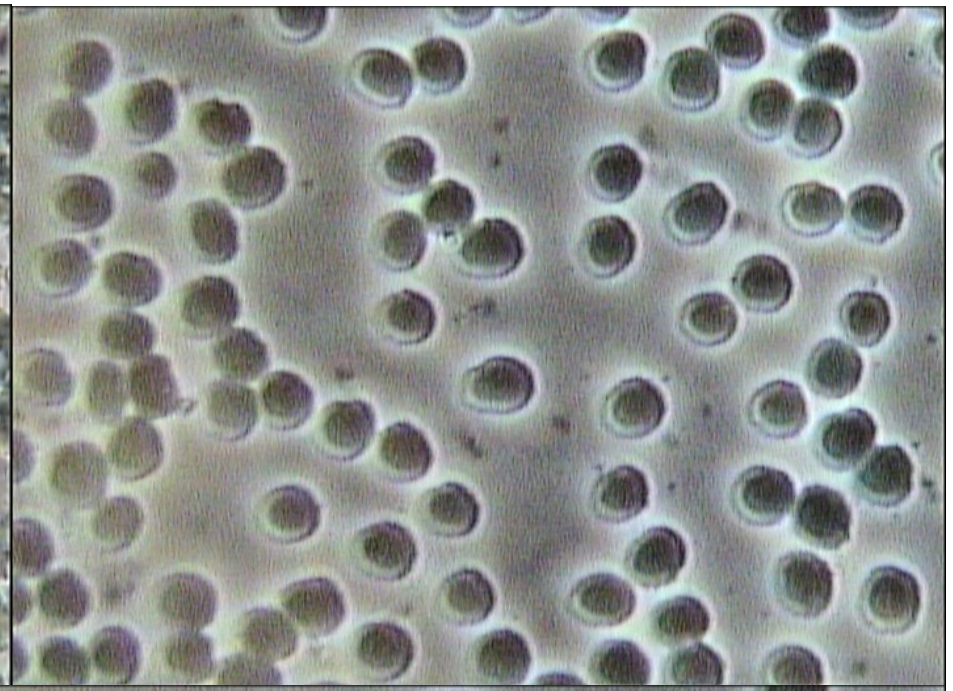
19 SUBJECTS: TREATMENT 1 TREATMENT 6

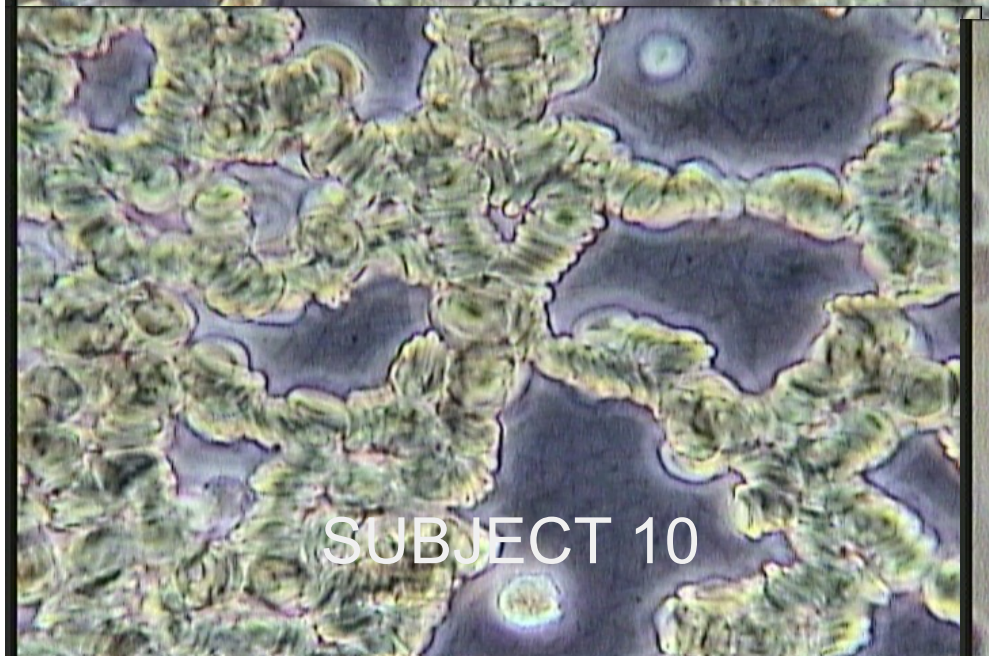
Why you don't need Cardiovascular exercises

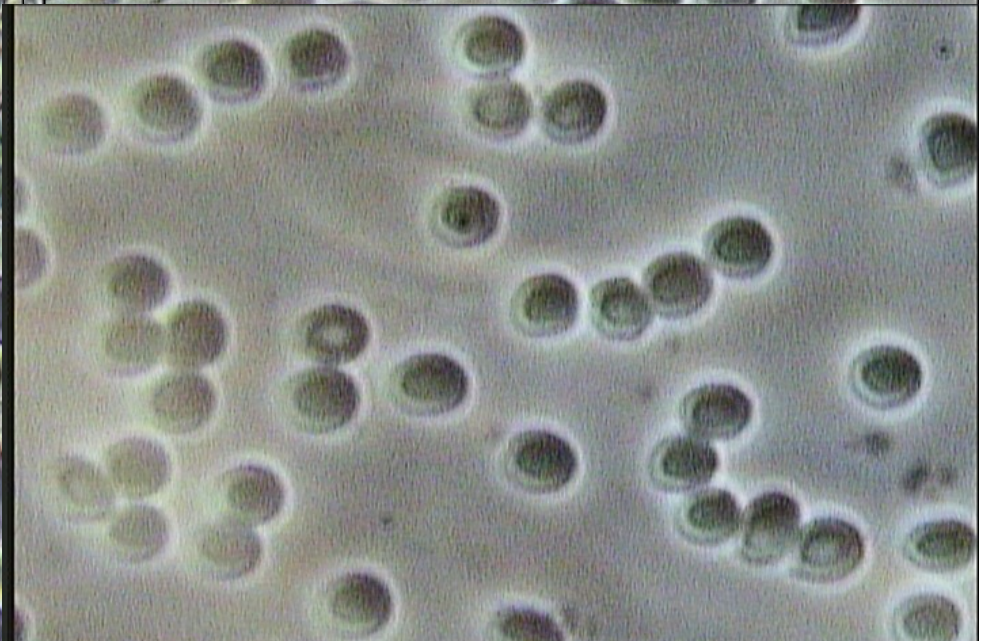
Normally you use cardiovascular exercises to increase heart rate. These increase the PUMPING RATE of the heart which STRAINS the heart –
Dangerous to seniors!

The Virtual Gym Separates RBCs. You don't have to increase the rate with which the heart pumps the blood because the blood flows without the heart being strained by excessive pumping





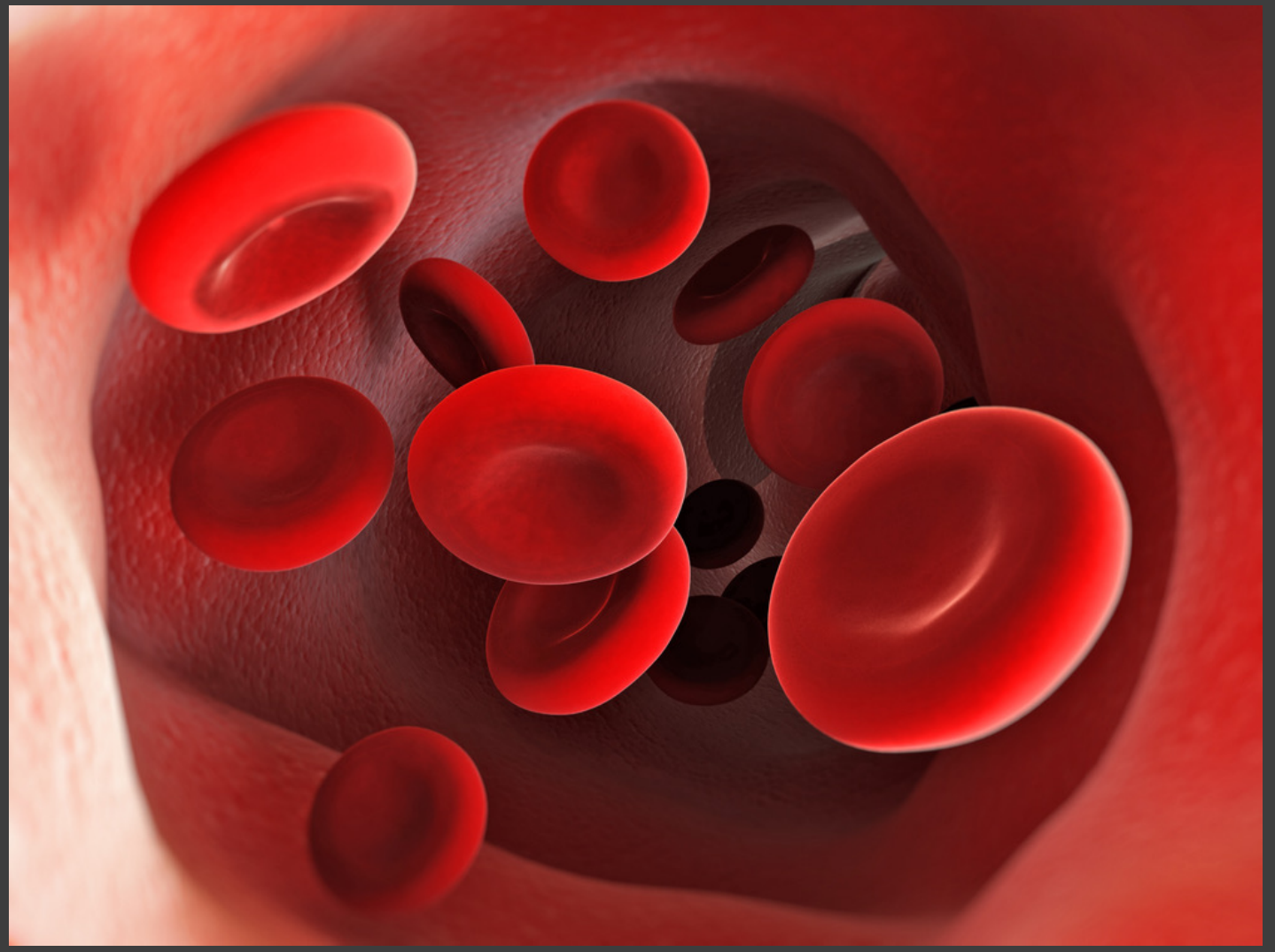




19 SUBJECTS	Erythrocyte Aggregation	Rouleau	Fungal Forms	Thrombocyte Aggregation	Bacteria	Poikilocy-tosis	Rouleau & Separate RBCs	Only Separate RBCs
Before Treatment	15	4	8	8	9	8	0	0
After First Treatment	1	6	6	7	8	6	9	3
Before Last Treatment	0	0	3	4	5	2	11	8
After Last Treatment	0	0	2	2	2	0	3	16

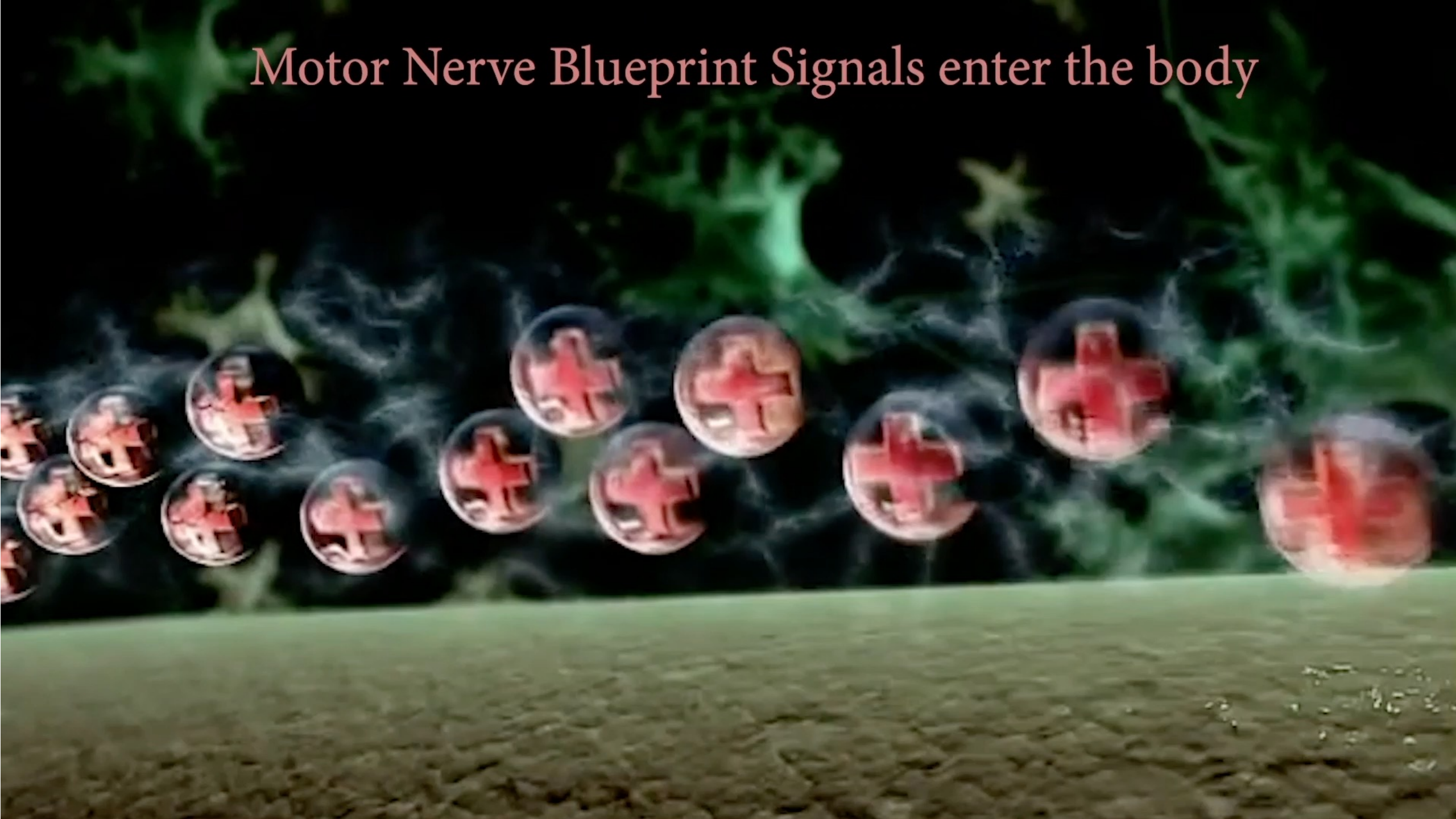
VIRTUAL GYM TREATMENTS SEPARATE ERYTHROCYTES (RBCs)

- OXYGEN TO CELLS
- NUTRIENTS TO CELLS
- ANTIBODIES TO SITES OF ACTION
- ENHANCES IMMUNITY
- CARRIES WASTE PRODUCTS TO LIVER & KIDNEYS FOR DETOXIFICATION



VIRTUAL GYM

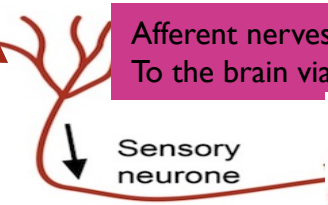
HOW DOES IT WORK?


Motor Nerve Blueprint Signals enter the body

Blueprint Motor Nerve Signal Unlimited Resolution / composed of up to 8000 waveforms after 42 years research Driven into the skin by Voltage

Resonates with Motor Nerve

Awakened CNS carries the signal to the brain via sensory nerves



Afferent nerves carry the signal To the brain via the spinal cord

Hormonal Release BALANCES HORMONES

Leptin and Ghrelin balance = REDUCES HUNGER

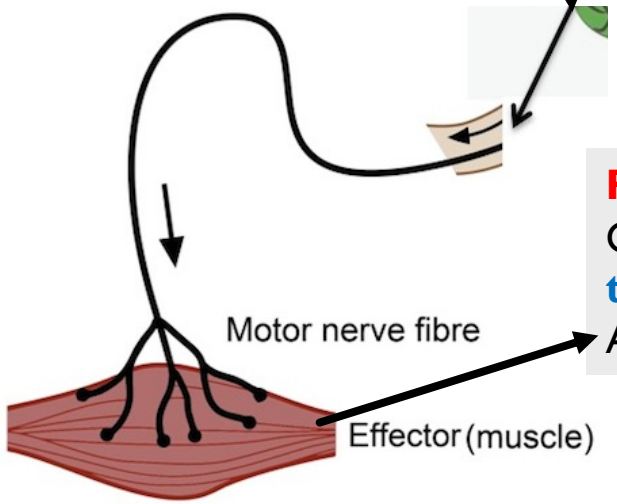
Hormones naturally trigger the release of the fat content into the bloodstream

Adipose tissue contains stem cells

MSCs repair liver and other vital organs

Glucose burns with oxygen in mitochondria

ATP production = ENERGY



FULL 8-10 secs CONTRACTION OF LARGE **COORDINATED** MUSCLES **that involve the ENTIRE BODY** AS IN REGULAR STRENUOUS EXERCISE

RBC's separation / lymphatic stimulation = DETOX

HOW THE VIRTUAL GYM ENHANCES HEALTH

1. Unlimited Resolution Composite signal, orchestrated out of up to 8,000 waveforms
2. Contraction is caused by the brain – brain orders the motor nerves to contract the body / Full 8-10 contraction of the entire body
3. Brain releases hormones (as it does during regular strenuous exercise / but at high speed!)
4. Natural release of fat via hormones into the bloodstream maintains the integrity of stem cells to repair the liver and other organs (Seen in CRP, Bilirubin and Creatinine normalization)
5. Hormonal release leads to HORMONAL BALANCE
6. Hormonal balance: Growth Factors / Metabolism / Testosterone increase / Cortisol decrease
7. Leptin / Ghrelin balance = suppresses excessive hunger
8. RBC's separation / lymphatic system stimulation = Detox = Decreases cravings
9. Visceral fat decrease / subcutaneous fat decrease / BMI decrease / VLDL and Triglycerides decrease
10. Increased Muscle Mass / FITNESS

OPTIMAL CHOLESTEROL

	Mean	S^2 =SS/df	T Value	p Value	Probability	Comments
VLDL 25 HA / 20 PMD	-1.19	0.31	-9.35	<0.00001	P<0.00001	VLDL was Reduced by -41.59%
Triglycerides 25 HA / 40 PMD	-1.25	0.61	-6.94	<0.00001	P<0.00001	Triglycerides were reduced by -31.96
HDL 30 PMD	9.34	23.66	10.52	<0.00001	P<0.00001	HDL was increased by +19%

METABOLISM INCREASE = RESULTS DO NOT REBOUND

Free T-3 45 HA / 10 PMD	0.93	0.13	11.62	<0.00001	P<0.00001	Free T3 was increased by +41.07% WNR

BODY SCULPTING

VISCERAL FAT, OVERALL FAT, BMI **DECREASE**

FITENESS INCREASE

BMR, **SMM**, **IGF-1** **INCREASE**

VAT 35 HA / 60 PMD	-4.68	7.12	-13.6	<0.00001	P<0.00001	VAT decreased by -21.95%
Overall Fat 50 PMD	-4.98	6.43	-13.88	<0.00001	P<0.00001	Overall Fat decreased by -13.42%
BMI 60 PMD	-2.3	1.28	-15.73	<0.00001	P<0.00001	BMI decreased by -10%
BMR 10 HA	91.6	3782.04	4.71	0.00055	**P<0.001**	BMR increased by +91.60%
SMM 35 HA / 15 PMD	+4.3	0.45	+13.49	<0.00001	P<0.00001	SMM increased by +40.7%
IGF-1 35 HA				<0.00001	P<0.00001	IGF-1 increased by +19.68

BMR: BASAL METABOLIC RATE: BODY BURNS MORE CALORIES WHILE YOU ARE SLEEPING / TURN CLOCK BACK TO YOUTH

Albumin **Average Increase: +%22.289**
Value of t=+9347886 **The value is p<0.00001.** **Significance: p<0.00001**

NO FATTY LIVER **LIVER REPAIR DUE TO:**

1. SIGNIFICANT VISCERAL FAT REDUCTION
2. LIVER REPAIR BY THE ADIPOSE TISSUE STEM CELLS – MSCs & HEPATOCYTES

ALT Average Decrease: -24.83% (p<0.001)	AST Average Decrease: -30.407 (p<0.0001)	ALP Average Decrease: -14.529 (p<0.01)

Creatinine 10 PMD	-0.24	0.04	-4.06	0.00143	P<0.01	Creatinine decreased by -19.67% WNR
CRP 10 PMD	-0.59	0.16	-4.72	0.00055	P<0.001	CRP decreased by -36.87% WNR

Diabetes

Blood Glucose Fasting 15 D	-61.88	7675.12	-8.11	<0.00001	P<0.00001	50% normal after 12 treatments
Blood Glucose PP 15 D	-63.07	7353.79	-845	<0.00001	P<0.00001	33% normal after 12 treatments

Prediabetes

Insulin Fasting 20 PD	-30.71	5961.47	−2.97	0.01031	P < 0.01	100% normal after 12 treatments
Insulin PP 20 PD	−129.43	18065.62	−7.20	0.00009	P < 0.0001	100% normal after 12 treatments

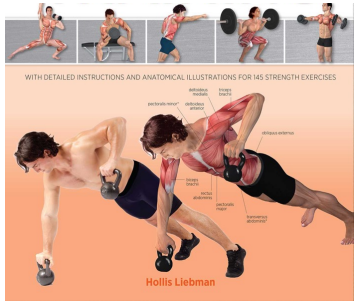
STRESS REDUCTION **ENERGY INCREASE** / FAT REDUCTION

Cortisol 35 HA	-18.26	142.98	-6.66	<0.00001	P<0.00001	Cortisol decreased by -13.08% WNR
Testosterone 35 HA	2.9	4.6	6.05	<0.00001	P<0.00001	Testosterone increased by +43% WNR

HUNGER REDUCTION / NO CRAVINGS = RESULTS DO NOT REBOUND

Leptin 10 HA / 10 PMD	1.82	2.68	4.98	0.00004	P<0.0001	Leptin increased by +13.41% WNR
Ghrelin	-43.55	962.79	-6.28	<0.00001	P<0.00001	Ghrelin decreased

THE VIRTUAL GYM 8888 FITNESS MAX OFFERS 256 EXERCISES REPEATED 1000 TIMES IN ONE HOUR FOR A TOTAL OF 25,600 EXERCISES. THESE INCLUDE



STRENGTH EXERCISES (CONTRACTION TIME SETTING ON 10 / REST DURATION 2)

RESISTANCE EXERCISES (CONTRACTION TIME SETTING ON 10 / REST DURATION 2)

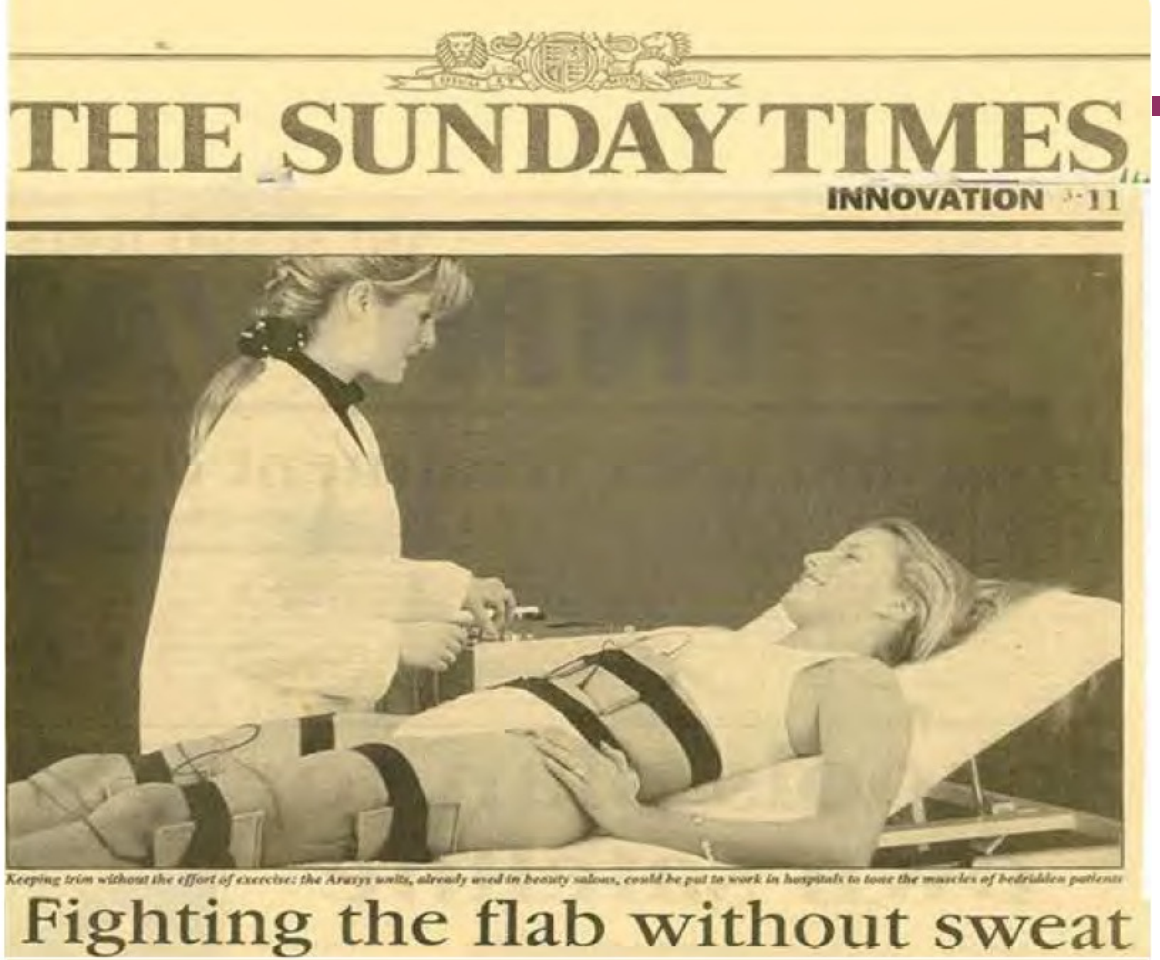
STRETCH EXERCISES (CONTRACTION TIME SETTING ON 10 / REST DURATION 2)

AEROBICS / JUMPING / RUNNING (CONTRACTION TIME SETTING ON 2 / REST DURATION 2)

EXERCISES TO RELIEVE BACK PAIN (CONTRACTION TIME SETTING ON 10 / REST DURATION 2)

LONDON UNIVERSITY

BY DR GERRY POLLOCK
THE CO-INVENTOR OF THE FIRST PACEMAKER



THE SUNDAY TIMES

INNOVATION 3-11

Keeping trim without the effort of exercise: the Arasys units, already used in beauty salons, could be put to work in hospitals to tone the muscles of bedridden patients

Fighting the flab without sweat

A SCIENTIST has invented a machine he claims will keep people trim without the need for exercise and could help reverse muscle-wasting conditions such as multiple seleroiss, writes Sean Hargrave.

The Arasys exerciser unite (fA-RAdic SYStem), developed at London's South Bank University Technopark, is already being sold to health clubs and beauty salons for those who want to lose weight without putting in the effort.

Now the machine's designer. Gerry Pollock, is searching for hospitals and clinics that could hellp him test the system on disabled patients who are unable to exercise. He believes Arasys could prevent the muscle wastage common among those confined to bed or a wheelchair.

The machine flexes muscle by passing tiny electric currents through nerve endings at either end of muscle groups. This makes the tissue contract for two seconds, as if it were being put through a gym workout.

A typical session with the machine lasts 17 minutes. Pollock says this is because people can feel tired if they have a longer stint and do not notice as much benefit as from a shorter session. He claims each treatment is the equivalent of doing 300 sit-ups and that three sessions are all that are needed until weight loss can be measured.

The Arasys system can treat four sets of muscle simultaneously. In cosmetic use these are normally the stomach, bottom, thighs and calves. In medical use, this would change to exercise the parts of the body a patient cannot move.

Pollock, a chemist, claims his technology is superior to machines that make similar claims of effortless weight-loss because of the electric wave form he designed. He says his electronics expertise, that was used in the development of the first pacemaker, ensures the muscles are exercised at the correct speed for the optimum duration.

This involves controlling electrical impulse to avoid suddenly jerky muscle movements. To achieve this, Arasys generates smooth rather than spiked electrical signals so that the muscle is stretched in a manner more similar to way it behaves during real exercise.

"We only discovered how long and intense the signal should be through trial and error during the system's five-year development." says Pollock. "Just passing any old electrical signal across a muscle simply doesn't work."

Besides helping the disabled, Pollock believes his machine could be used to return strength to the elderly and those who suffer from multiple selerosis.

His niece, Angela Sylvester, a qualified nurse, regularly uses Arasys on four ME sufferers who are unable to exercise. She claims they all report they feel stronger.

"One of the ladies used to be a fitness instructor, but because of her condition she cannot work out any more," says Sylvester. "she benefits from being able to stay trim and exercise muscles that would otherwise be hardly used."

Pollock hopes his invention will soon be put to its original healthcare use and is keen to talk with clinics and hospitals that believe they could help him tailor the system for individual conditions.

"I need to talk with experts so that we can decide if the present electrical signal is appropriate or if it needs changing," he says.

Sunday Times, UK and other journals published several articles about Gerry Pollock's invention of SIMULATED EFFORTLESS EXERCISE in London University

Dr. Pollock spent 27 years of laboratory empirical (trial & error) research on this invention.

Dr Gerry Pollock's London University Research (1990)

Goldpink's research on Gene Expression

- Rapid muscular hypertrophy
- 250% increase in the RNA content of the muscles
- Repression of the fast-type genes and activation of the SKELETAL slow-type genes.

Biochemical Society Transactions

Stretch and force generation induce rapid hypertrophy and myosin isoform gene switching in adult skeletal muscle

Geoffrey Goldspink, Andrew Scutt, Jane Martindale, Thomas Jaenicke, Lucien Turay and Gerald-F. Gerlach
Unit of Molecular and Cellular Biology, The Royal Veterinary College, London University, Royal College Street, London NW1 0TU, U.K.

Summary
Using electrical stimulation to control force generation and limb immobilization to alter the degree of stretch, we have studied the role of mechanical activity in inducing hypertrophy and in determining fast and slow muscle fibre phenotype. Changes in gene expression were detected by analysing the RNA in hybridization studies employing cDNA probes specific for fast and slow myosin heavy chains and other genes. As a result of overload in the stretched position, the fast contracting tibialis anterior muscle in an adult rabbit is induced to synthesize much new protein and to grow by as much as 30% within a period as short as 4 days. This very rapid hypertrophy was found to be associated with an increase of up to 250% in the RNA content of the muscles and an abrupt change in the species of RNA produced. Both stretch alone and electrical stimulation alone caused repression of the fast-type genes and activation of the slow-type genes. It appears that the fast-type IIB genes are the default genes, but that the skeletal slow genes are expressed as a response to overload and stretch. These findings have implications as far as athletic training and rehabilitation are concerned.

Introduction
Muscle is a tissue in which gene expression is regulated to a large extent by mechanical signals. Mammalian muscle consists of populations of slow-contracting, oxidative fibres and fast-contracting fibres which are characterized by different protein isoforms. Therefore, post-natal growth and the differentiation into the fast type or the slow type of fibres must presumably involve the regulation of expression of different subsets of genes. Here we have focused on the expression of myosin heavy chain genes and their response to mechanical stimuli.

The intrinsic velocity of contraction ($V_{max.}$) of muscle fibres is related to the specific activity of their myosin ATPase [1]. Myosin is a double molecule that consists of two heavy chains each of about 220 kDa. The actin-attachment site and the ATPase site are located in the S1 region (head of the myosin molecule) of each heavy chain. Associated with the S1 fragment are smaller polypeptides called light chains which are believed to modulate the cross-bridge ATPase activity [2]. Subtypes of fast muscle fibre have been identified histochemically and these may exist because of different combinations of myosin heavy and light chains and different mitochondrial content. Slow fibres differ in several ways from fast fibres in that they have many more mitochondria, different cytoplasmic isoenzymes, as well as different isoforms of myofibrillar proteins. The isoforms of myosin have been shown to be the product of a multigene family and their expression is tightly regulated in a stage-specific and tissue-specific manner [3, 4]. Phenotypic expression of muscle genes is known to be influenced by thyroid hormone [5, 6] and altered patterns of innervation [7]. However, the influence of physical activity at the gene level was unclear. We have, therefore, studied changes in transcriptional levels of the fast and slow myosin heavy chain genes in response to stretch and force generation.

Abbreviation used: TA, tibialis anterior.

Methods

Stimulation and acute-stretch procedures
Tibialis anterior (TA) muscles in adult Netherland dwarf rabbits were stimulated using Teflon-coated stainless-steel electrode wires implanted into the popliteal fossa [8] under valium/Hypnorm anaesthesia. The electrode wires were externalized at the back of the neck and attached to a miniature stimulation circuit which was held in position by a small saddle fashioned out of an elastic bandage. Several circuit designs were used which generated biphasic pulses at frequencies ranging from 2 Hz continuous to 120 Hz intermittent. A 30 Hz intermittent circuit was designed to give the same number of pulses/min as a 2 Hz continuous, and a 120 Hz and 60 Hz intermittent circuit gave the same number of pulses/min as a 10 Hz continuous circuit. In this way, the hypothesis that it is the number of pulses delivered which determines muscle fibre phenotype could be tested. The pulse length was 0.1 ms and the pulse amplitude was adjustable from 1 to 3 V and each miniature stimulator was fitted with an on/off switch. Muscle

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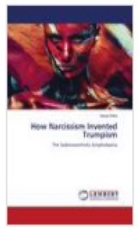

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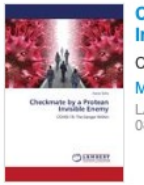

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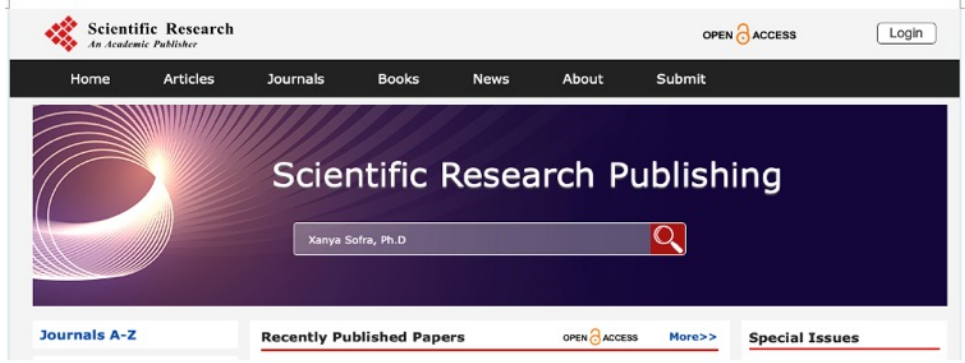

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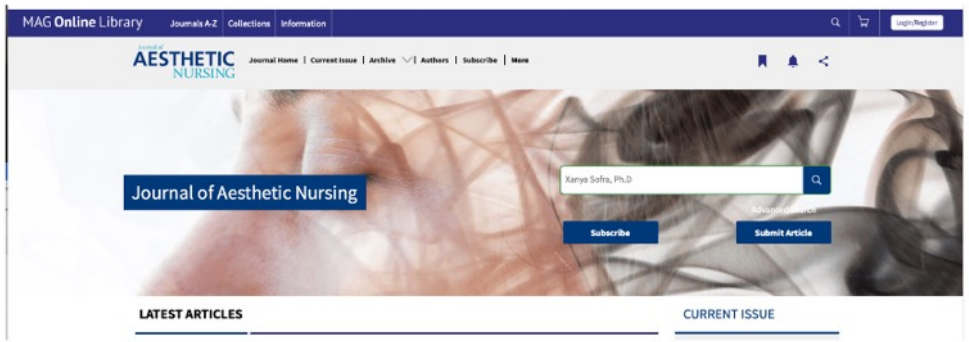
Biography: Dr Sofra has a doctorate from in Neurophysiology from City University London, UK, and a doctorate in Clinical Psychology from the New School for Social Research, New York City, USA. She is an award-winning international speaker in several Medical and Anti-aging societies and author of several published research and scientific review articles. She was been in clinical practice for years prior to be involved in the invention of advanced anti-inflammatory nanotechnology designed for wound healing, pain relief and systemic balance. Her current research is on signalling pathways, hormonal interactions, and the investigation of existing biological molecular mechanisms involved in time reversal.



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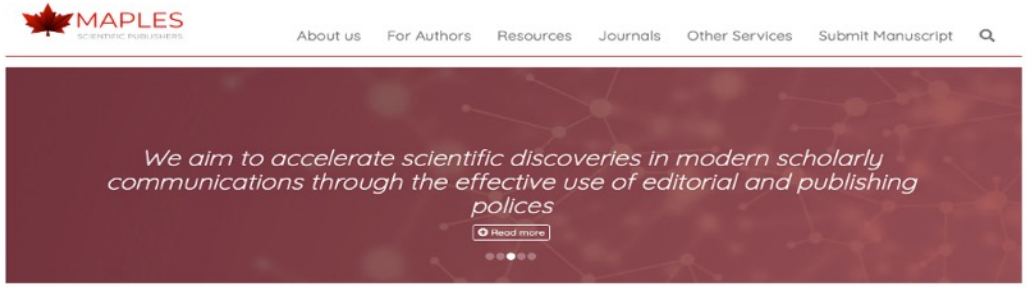

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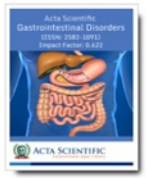

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- Published: May 25, 2021; Volume 4 Issue 6: 54-69.

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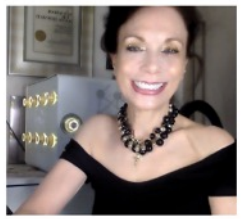

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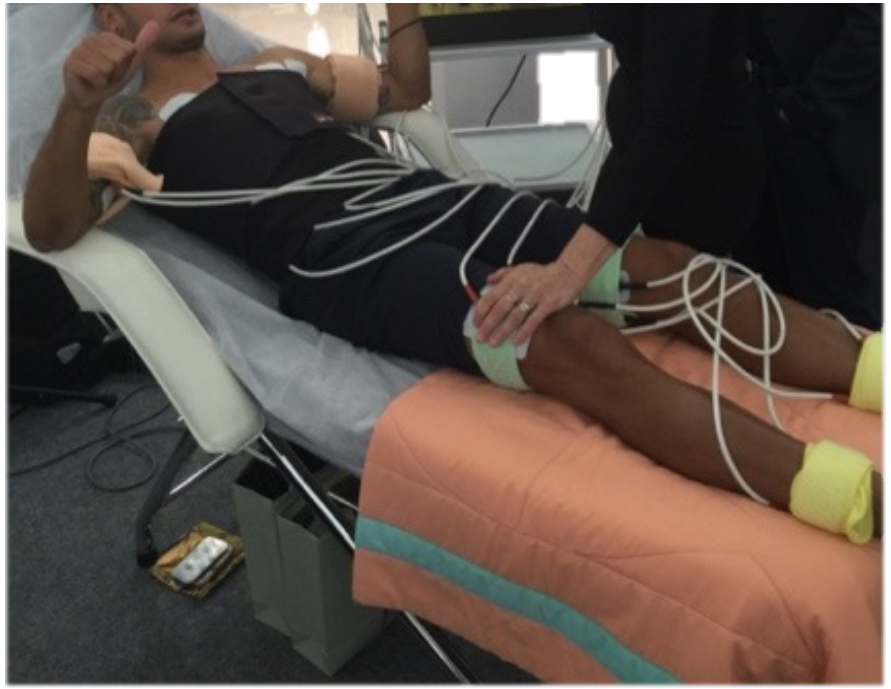
Journal of Endocrinology Research	Vol 3, No 1 (2021): Online First	Covid-19 Mutations and How the Vaccine Enhances Immune Intelligence	ABSTRACT PDF
Xanya Sofra			

Virtual Gym Increases Stamina, Core Strength and Speed

OTHER NFL AND EUROPEAN FAMOUS SPORTS ATHLETES HAVE BOUGHT The virtual gym For personal use to increase stamina, core strength & speed. Most of them have not given us consent to reveal their names.

Raphael Timo, Dutch football player testimonial – Played with Croft (pic right):

"40% more fit after one virtual gym treatment!"

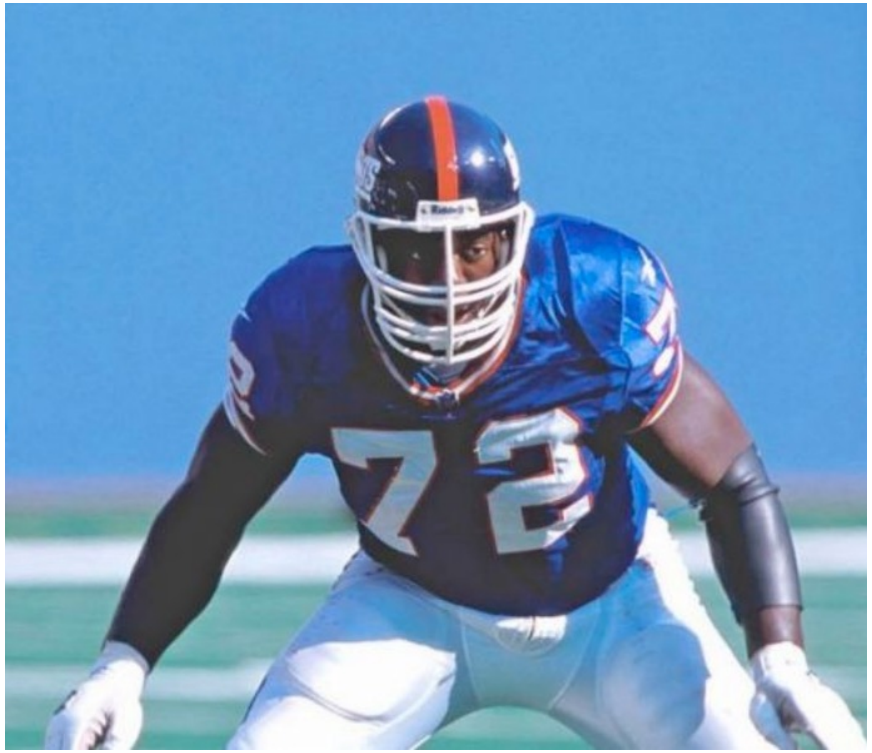


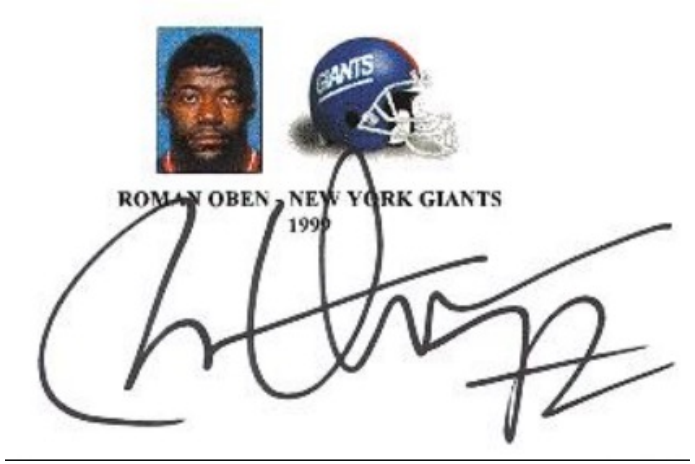
IS THERE A WAY OF **EXERCISING WITHOUT EXERCISING**?

Hormones are interconnected with Exercise

Hormones trigger the fat burning processes to form the energy that sustains exercise and build muscle

The brain is responsible for all movement including the full muscle contractions during exercise





Lets use SIGNALING to activate the Motor Nerves

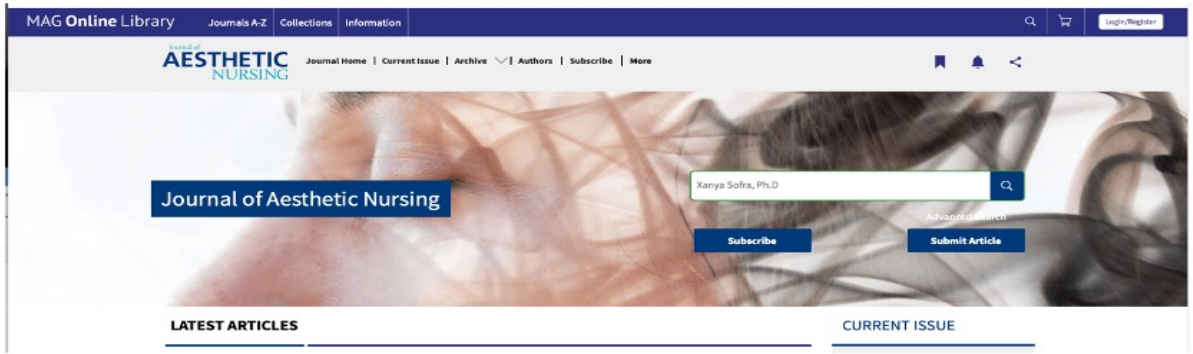
Motor Nerves (part of the CNS) MUST involve the Brain that can cause the full body contractions

The Brain will order the necessary Hormones to produce the energy that will never exceed hormonal balance due to negative feedback mechanisms

Result : **SIMULATED EXERCISE**
or exercising without exercising

Effortless Simulated Exercise

8 Seconds Whole Body Contraction



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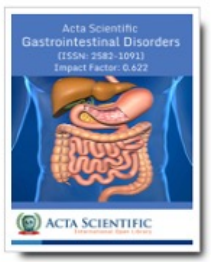

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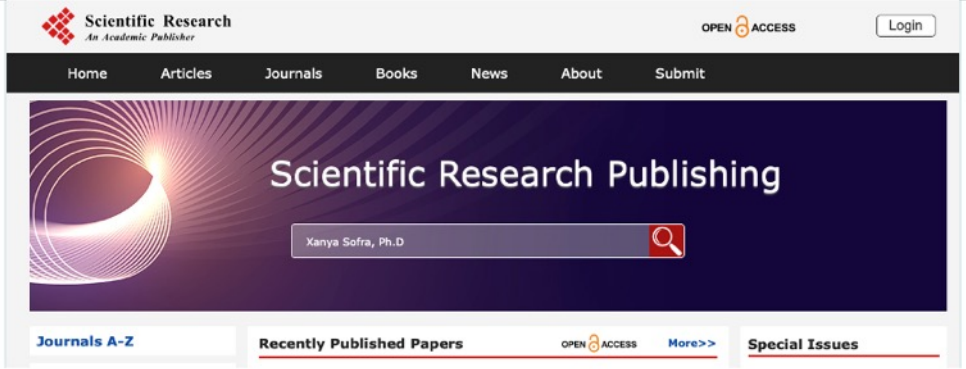

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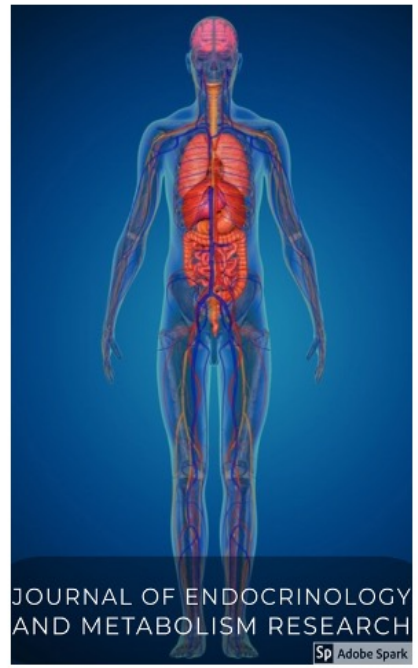

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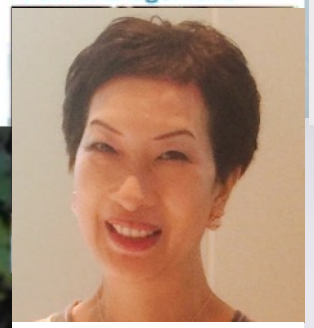

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