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The Lifestyle-Medicine Approach to CHD Risks in A Brazilian Community Study. Determining Factors and Effectiveness of Lifestyle Modification Protocol

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ABSTRACT

In the last decades, CHD, among others NCDs, is leading the causes of mortality. By using the Framingham Score (FS), in our Brazilian Dynamic Cohort Study survey (n=1067), 32.4% presented some risk of CHD, mostly moderate (75.8%), and 18.3% CHD-high risk. Assessment tools included the IPAQ (long version), Healthy Eating Index (HEI-through 24-hour food intake recall), body weight, height, and electrical bioimpedance, as well as clinical signs and fasting plasma markers of insulin resistance (HOMA-IR), inflammatory (hsCRP), and oxidative (MDA) states. Having FS as main variable and as co-variables: socio-demographic, behavioral (dietetic and physical fitness) anthropometric, clinical and biochemistry, the cross-sectional analysis showed the incremental risk of CHD as being determined by older aging, lower schooling, total body (BMI) and abdominal fatness (WC and ASD) dystrophies, low-muscle mass, blood hypertension, atherosclerotic dyslipidemia and stress markers of inflammation, oxidative stress and insulin resistance. Behavioral factors of inadequate dietary quality (HEI) and physical inactivity (IPAQ) were considered weaker factors for contrasting higher risk of CHD from the other classes, differently of low VO2max, considered a strong risk factor. From that, a sub-sample (n=567) was submitted to a longitudinal intervention with lifestyle-change protocol (LiSM) with supervised physical exercises and dietary counseling. The intervention normalized the leisure domain and the total IPAQ, increased Vo2max, HEI, and muscle mass, reduced obesity and most other NCDs along with their underlying factors of inflammation and oxidative stress. Framingham score decreased, as did the prevalence of CHD-risk classes, but keeping the sample-CHD-risk (8.25% to 7.70%). These results could be amplified under higher adherence of patients to the whole lifestyle-reeducation procedures, higher compliance to longer-lasting LiSM, and by teaching the medical and health care communities to more incorporate knowledge into the daily practices of (lifestyle) medicine. The Lifestyle-, Medicine approach proposed here, would be then, an effective and money-saving alternative to the current homeostasis approach, for the CVDs 2nd care.

Keywords

Coronary Heart Diseases, CHD-risk factors, CHD behavioral factors, Lifestyle-change effectiveness.

Abbreviations

MetS: Metabolic syndrome; Ob: Obesity; LMM: low-muscle mass; MM: muscle mass; NAFLD: Non-alcoholic fatty-liver disease; T2D: Type 2 Diabetes; PAI: Plasma-atherogenic index; HOMA-IR: Homeostasis Model Assessment Insulin Resistance; hs-CRP: high sensitivity C-reactive protein; Hcy: Homocysteine; GSSG: oxidized glutathione; MDA: malondialdehyde; β-C: Beta carotene; TAP: Total antioxidant products; GSH: reduced glutathione; CHD: Coronary-heart disease; IPAQ: International Physical Activity Questionary; HG: Hand-grip strength; FLEX: Trunk flexibility; HEI: healthy-eating index; CHO/F: Carbohydrate/Fibers ratio; Na/K: Sodium/Potassium ratio; Ca/Prot: Calcium/Protein ratio.

Introduction

Non-communicable chronic Diseases (NCDs), including cardiovascular disease (CVD), type 2 diabetes (T2D), chronic lung disease, allergy, some forms of cancer, cognitive decline, osteoporosis, and sarcopenia, are the world's biggest killers [1]. NCDs are defined as slow in their progress and long in their continuance and, because of their clinically "silent" characteristics, NCDs need an appropriate propaedeutic approach to be properly diagnosed [2]. Usually, by its complexity, proper diagnosis of NCD and co-morbidities demands clinical, anthropometric, dietary, biochemistry, and fitness expertise. Clinically, it is possible to diagnose blood hypertension, anthropometrically: overweight, obesity, and sarcopenia, laboratory biochemistry: T2D and dyslipidemia, and, by combinations: MetS, NAFLD, liver fibrosis, atherogenesis and CHD risks [3]. NCDs usually rely on anthropometric abnormalities of obesity and sarcopenia, both having underlying causes of metabolic stress (inflammation and oxidative stress) as measured by biochemical markers in the blood [4].

Coronary artery disease (CHD) is the leading cause of death in developed countries, and it currently occurs with greater frequency in developing countries, with 80% of the deaths occurring in low and middle-income countries, especially among the elderly [5,6]. In Brazil, NCDs account for 74% deaths and have cardiovascular disease (CVD) as the leading cause (31%) of all deaths [7,8].

CHD is considered a complex disorder associated with severe health complications reaching pandemic proportions worldwide. Historically, CVDs began as a constellation of diseases associated with obesity, with abdominal adiposity currently being placed as its main component related to insulin resistance abnormality. Cell steatosis, either in the liver or skeletal muscle (myosteatosis) are causes of insulin resistance and have inflammation as effector. Diminished tissue sensitivity to insulin is a characteristic of pathological conditions termed the insulin resistance syndrome, also known as the metabolic syndrome or cardiometabolic syndrome [9].

Cardiovascular diseases share common pathways, including increased oxidative stress, defective glucose, lipid metabolism, low-grade inflammation, hypercoagulability, and endothelial damage. Therefore, CHD is not a single disease; rather, it is a complex cluster of abnormalities such as abdominal obesity, atherogenic dyslipidemia, raised blood pressure, and insulin resistance. It is often associated with inflammation, impaired endothelial function, and prothrombotic state, and has in its pathophysiological process, the inflammatory and oxidative stresses that might lead to lipo-glucotoxicity, considered as a trigger of atherosclerosis and recurrent diseases [10].

Historically, CVDs began as a constellation of diseases associated with obesity, with abdominal adiposity currently being placed as its main component related to insulin resistance abnormality. Cell steatosis, either in the liver or skeletal muscle (myosteatosis) are causes of insulin resistance and have inflammation as effector. Diminished tissue sensitivity to insulin is a characteristic of pathological conditions termed the insulin resistance syndrome, also known as the metabolic syndrome or cardiometabolic syndrome [9]. Cardiovascular diseases share common pathways, including increased oxidative stress, defective glucose, lipid metabolism, low-grade inflammation, hypercoagulability, and endothelial damage [10].

The second half of the last century showed an increased CHD 29X, with 100% increased rate of obesity and T2D. It is known that nuclear-DNA spontaneous mutation occurs in a rate of 0.5% per million years. Therefore, epigenetic modification of gene expression is one mechanism by which genetic susceptibility and environmental insults can lead to most of the contemporary NCDs [9]. Thus, the development of CHDs may be influenced by genetic as well as environmental factors related to unhealthy body composition, dietary inadequacy, and a low level of physical fitness. This so-called obesogenic-atherogenic environment has never existed in the past and was consequently not part of selection pressure; therefore, genetic adaptations might also not be expected. It seems that our genetically determined "survival strategy" turns against us now that we can eat whatever we want and whenever we want, and need little physical activity for food procurement [9].

Organisms are bundles of compromises shaped by natural selection and humans and other primates have evolved particular morphological and biological traits that distinguish them from most other mammals. Genetic traits may be positively or negatively selected relative to their concordance or discordance with environmental selective pressures. An evolutionary view suggests that many genetic variants interact with environments and other genes during development to influence disease phenotypes. Therefore, it is possible to analyze a great number of diseases in terms of adaptive vulnerabilities connected to our phylogenetic inheritance, such as human bodily inadequacies in relation to the modern environment. This means that current diseases such as obesity, T2D, essential hypertension, dyslipidemia, and metabolic syndrome may result from the mismatch of our ancestral thrifty genotype with the contemporary way of life through epigenetic modification of gene expression [9].

Actual medicine ignores evolution and instead focuses upon proximate mechanical causes as modeled by mechanical physics, deriving from Galileo, Newton, and Descartes. As a result of assuming this model, medicine is mechanistic, materialistic, reductionist, linear-causal, and deterministic in its concepts. It seeks explanations for diseases, or their symptoms, signs, and cause in single, materialistic changes within the body wrought directly by infectious, toxic, or traumatic agents [11].

Underlying why drug therapy has been largely unsuccessful in halting and reversing the NCDs epidemic would be the ineffectiveness of the treatment approach by the homeostasis model [11]. Homeostasis describes mechanisms that hold a controlled variable constant by sensing its deviation from a "setpoint" and

feeding back to correct the error. Following the homeostasis model, physicians try to restore each parameter to what they consider an "appropriate" level. Currently, major diseases are now rising in prevalence, such as obesity [12,13], type 2 diabetes, hypertension, and metabolic syndrome [14], whose causes the homeostasis model cannot explain [2]. Coincidentally, they all have evolutionary thriftiness involved in their contemporary origin [9].

Homeostasis treats low-level targets and, following this model, for obesity, it lists six neuromodulators that increase feeding and ten that decrease feeding, and then concludes, "a multi-drug regimen that targets multiple sites within the weight-regulatory system may be necessary to achieve and sustain weight loss in many individuals". Similarly, hypertension is treated with drugs that target the three primary effectors of elevated pressure (diuretics, vasoconstrictor antagonists and heart rate antagonists). The same strategy is proposed for type 2 diabetes and metabolic syndrome. For sure, there are "side effects" involved in each of the steps and, the pharmaceutical industry continues to target myriad molecules that regulate these mechanisms, and fundamental research widely promises to identify new targets. By standard pharmacotherapy, drugs can force the response back to the original level, despite continued prediction of high demand, but this compresses responsiveness. While demand stays high, drugs that antagonize key effector mechanisms force the response distribution back toward its initial mean. But this reduces responsiveness and evokes iatrogenic effects. Adding more drugs to a complex system increases the frequency of iatrogenesis [2]. For all of these, the high discontinuance rates are considered to reflect, among other factors, "a combination of adverse drug effects, cost of drugs, and poor efficacy".

NCD medications are expensive. In the first decade of this century (2002-2006), the prescription drug costs were an increasingly large component of overall health care costs of the Brazilian Ministry of Health, varying from 5.4% to 11% of total expenditure. Overall, the increased expenditure with drugs varied from 16.36% in 2002 to 49.7% in 2003 and 45.85% in 2005, summing up 123.9% (2002-2006) [11].

As an alternative to the homeostasis approach, we are finding some Brazilian studies investigating the association of factors related to lifestyle changes with CHD risk factors, but only individually [15-17] (e.g., hypertension, diabetes, hypercholesterolemia). However, to our knowledge, no national study yet investigated the association between CHD risk (Framingham score) and its direct relationship with dietary components, biochemical, and body composition [18,19].

The purpose of this paper is to determine the intrinsic and environmental factors associated with CHD risks in free living adults, followed by the responses to an intervention with supervised physical exercises and counseled dietary adequacy.

Methods

The used datafile came from the lifestyle modification program "Mexa-se Pró-Saúde" [Moving for Health], a longstanding (1991-2019) dynamic-cohort study conducted by the University's "Nutritional and Exercise Metabolism Center(CeMENutri)", a multiprofessional teaching and research center of the UNESP-Medical School, a southeastern Brazilian state university. "Moving for Health" (MPH) was a community based project with spontaneous access offered to adults (>35 yrs old) from both genders that came to the clinic for preventive health examination and further interventions including nutrition re-education, supervised physical exercise and other lifestyle factors. Upon registration and filling out the ethical protocol, the subjects were submitted to clinical assessments allowing the inclusion criteria (first step). Once included, the second step of MHP was a cross-sectional design involving lifestyle diagnosis of physical activity, fitness, dietary quality and food intake along with body composition and plasma biochemistry, assessments for diagnosing non-communicable chronic diseases(NCDs). The third step involved a longitudinal approach with interventions with scientific-based protocols of supervised physical exercises and counseled diet [3].

The present cross-sectional analysis included data from 1067 subjects for CHD-risk estimation and a sub-sample of 709 subjects for Framingham score calculation. The assessments included physical activity level (IPAQ version 8 - long form), fitness of trunk flexibility (sit and reach in Wells bench), forearm strength (hand-grip dynamometer), aerobic conditioning (VO2 max.), dietary intake (24-hour dietary recall and Healthy Eating Index-HEI calculation), anthropometrics of body fatness and muscle mass, clinical evaluation, and fasting-plasma biochemistry [3]. As described earlier, the assembling of anthropometric/biochemical assessments allowed the diagnosis of overweight/obesity [20], abdominal obesity [21], and insulin resistance/T2D [22,23]. Blood hypertension was detected clinically [24], and clinical, anthropometric, and plasma composition were gathered in an algorithm for diagnosing Metabolic Syndrome [14] when a minimum of 3 from its 5 components (WC, blood pressure, plasma glucose, TG and HDL-chol.) were altered [25]. BMI, waist circumference(WC), triglycerides (TG) and Gamma Glutamyl-transpeptidase(GGT) were used to develop the "fatty liver index"(FLI) for Non-Alcoholic Fatty-liver Disease (NAFLD) diagnosis [26]. The algorithm varies between 0 and 100, with a FLI ≥ 60 ruling in the fatty liver [27]. Age, BMI, presence of T2D, AST/ALT ratio, platelets counting and plasma albumin were used to develop the "NAFLD fibrosis score" denoting: Absence <-1.455; -1.455 to 0.676 tolerable and, >0.676 presence of hepatic fibrosis [28].

Plasma atherogenic index was calculated by a formula involving the log of the ratio of triglycerides and HDL cholesterol levels in the plasma. The data were interpreted as: <0.11 low risk; 0.11-0.24 medium risk; >0.24 high risk for atherosclerosis and CHD [29]. For the Coronary Arterial Disease Risk it was used the algorithm of Framingham score assembling age, gender, tobacco

use, total and HDL plasma cholesterol, presence of T2D, and presence of hypertension. The risk is classified as: <10% low risk; 10%-20% medium risk; >20% high risk for CHD in the next 10 yrs [30,31]. Individuals who reported diabetes mellitus (type 1 or 2) through clinical protocol were included in phase 1 of risk stratification, which is considered a clinical manifestation equivalent to arteriosclerotic disease. Thus, the population with diabetes has a 20% greater risk of presenting cardiovascular events in 10 years. Phase 2 of the stratification considers the risk by estimating Framingham scores, where after adding the points obtained for each variable (gender, age, systolic blood pressure, TC, HDL-C and smoking), the absolute risk percentage e in 10 years was calculated, which can be classified as low risk (<10%), intermediate risk (10 to 20%) and high risk (>20%).

From the baseline assessments, the participants were able to be assigned to an intervention involving daily sessions of supervised exercises combined with counseled (or supervised) dietary interventions. The follow-up assessments occurred every 10 weeks [3]. Subjects were aware of the study and signed a consent form based on the "experiments involving humans" of the Brazilian "National Council of Health, Ministry of Health" and the declaration of Helsinki. Both the design and consent form were submitted and approved by the Research Ethics Committee of the Botucatu School of Medicine.

Results and Discussion

Cardiovascular Disease (CVD) remains the leading cause of worldwide mortality. In the United States this results in more than 37% of annual mortality [32]. The Coronary Artery Disease(CAD) or Coronary Heart Disease (CHD) risk can be calculated from risk factors and the presence of clinical signs and biochemical abnormalities [33]. The Framingham risk score is often used as an initial evaluation parameter of CAD risk in individuals with numerous risk factors.

The use of an algorithm using risk factor categories to predict coronary heart disease (CHD) denoted as Framingham Score(FS), has been used since late 20th century [31]. The FS was developed to estimate the CHD risk over 10 years and it was validated for people aged up to 75 years [34,35]. Thus, the Framingham score(FS) is a predictive algorithm developed using categorical variables, allowing the 10-year prediction of multivariate CHD risk in patients without overt CHD.

Presently, when distributed by genders, the present Framingham-Score (FS) data showed male values slightly higher than female, in all quartiles: p25(F/M) <_3.0/<_5.0; p25-50 >3.0-7.0/>5.0-9.0; p50-75 >7.0-10.0/>9.0-15.0; >_p75 >10.0/>15.0. Within genders, the similarity between extreme quartiles (p25 and p75) denoted homogeneity of the sample for that specific variable. This was found for age and civil status (Table 1), as well as for TEI and HEI, diet variety, intake of CHO, Prot, meat, total fat, dairy, cereal, sugar (Table 2), and trunk flexibility (Table 3). Nevertheless, the top quartil (>p75) prevailed over the lower quartile (<p25) of FS, in both genders, for lower schooling and self perception of bad-health status (Table 1), higher dietary intake of PUFA, cholesterol, CHO/Fibers and Na/K and, lower intake of MUFA, fruits and legumes (Table 2), as well as lower IPAQ and HG strength, along with anthropometry higher WC, ASD, %BF and lower %MM and MMI (Table 3), associated with higher hs-CRP, HOMA-IR and MDA (Table 4).

Table 1: Extreme quartile-range of the Framingham-score distribution in both genders for demographic and social-economic variables.

	Female			Males		
	p25 (≤3.0 pts)	p75 (≥10.0 pts)		p25 (≤5.0 pts)	p75 (≥15.0 pts)	
	n = 161	n = 163		n=51	n=54	
Parameters			p			p
Age-md (min-max)	53.50(36.0-79.0)	54.0(35.0-84.0)	0.9	52.0 (35.0-80.0)	55.0(37.0-75.0)	0.8
Schooling (n (%))						
Uncompleted Elementary	15 (24.6)	46 (75.4)	0	7 (28.0)	18 (72.0)	0
Completed Elementary	75 (52.1)	69 (47.9)	0.9	24 (51.1)	23 (48.9)	1
Completed High School	50 (59.5)	34 (40.5)	0.5	15 (57.7)	11 (42.3)	0.7
Universitary	21 (58.3)	15 (41.7)	0.6	5 (71.4)	2 (28.6)	0.1
Marital status (n (%))						
Single	127 (48.5)	135 (51.5)	0.6	127 (48.5)	135 (51.5)	0.5
Married	34 (54.8)	28 (45.2)	0.7	34 (54.8)	28 (45.2)	0.7
Family income (minimum wage)						
≤2	13 (26.0)	37 (74.0)	0	8 (38.1)	13 (61.9)	0.5
2 a 5	50 (39.1)	78 (60.9)	0.2	25 (48.1)	27 (51.9)	0.9
5 a 10	70 (72.9)	26 (20.3)	0	11 (36.7)	19 (63.3)	0.2
>10	28 (56.0)	22 (44.0)	0.7	7 (58.3)	5 (41.7)	0.6
Health Status (n (%))						
Bad	13 (21.3)	48 (78.7)	0	4 (17.4)	19 (82.6)	0
Regular	49 (44.9)	60 (55.1)	0.6	17 (45.9)	20 (54.1)	0.7
Good	54 (62.1)	33 (37.9)	0.5	16 (72.7)	6 (27.3)	0
Very good	23 (59.0)	16 (41.0)	0.4	8 (61.5)	5 (38.5)	0.4
Excellent	22 (78.6)	6 (21.4)	0	6 (60.0)	4 (40.0)	0.4

Table 2: Extreme quartile-range of the Framingham-score distribution in both genders for dietary variables

	Female			Male		
	p25 (≤3.0 pts)	p75 (≥10.0 pts)		p25 (≤5.0 pts)	p75 (≥15.0 pts)	
	n = 161	n = 163		n = 51	n = 54	
Parameters			p			p
TEI (kcal/day)	1668.37±503.93	1544.16±497.68	0.6	1839.42±674.30	2003.82±856.30	0.5
HEI (points)	79.85±14.25	77.43±15.21	0.12	81.18±20.16	79.92±13.27	0.05
CHO (%)	52.15±8.88	57.00±9.75	0.28	58.71±8.22	60.00±10.77	0.36
CHO (g/day)	186.20±48.74	189.70±50.15	0.69	206.17±61.57	236.34±132.06	0.21
Ptn (%)	18.55±4.92	16.30±3.75	0.15	20.07±5.87	17.67±4.88	0.19
Ptn (g/day)	64.40±16.04	59.30±18.70	0.21	84.01±38.21	82.48±35.20	0.42
Fibers (g/day)	18.7±7.43	14.25±6.50	0.035	17.81±9.97	23.36±12.39	0.04
CHO/fibers	14.70±5.25	21.50±7.05	0.04	17.53±6.75	24.75±10.26	0.03
Cereal (serving/day)	3.50±1.10	4.10±2.00	0.38	4.72±2.69	5.09±3.86	0.25
Sugar (serving/day)	1.80±0.70	1.95±0.65	0.85	2.04±1.75	2.33±1.08	0.48
Total fat (%)	28.90±8.56	32.70±10.20	0.09	29.50±7.65	32.08±9.62	0.29
Total fat (g/day)	57.60±18.14	61.74±20.35	0.25	58.45±31.32	62.47±42.15	0.21
SFA (%)	8.30±4.16	9.60±4.90	0.37	8.51±3.42	10.86±3.24	0.04
SFA (g/day)	17.35±9.76	18.05±10.10	0.68	17.65±14.37	22.89±6.51	0.04
MUFA (%)	9.30±5.74	7.75±4.75	0.03	10.72±4.00	9.00±6.31	0.05
MUFA (g/day)	14.60±8.41	12.60±7.50	0.04	18.97±14.48	15.05±8.80	0.04
PUFA (%)	6.35±2.97	8.25±3.05	0.04	6.18±3.59	8.63±1.64	0.04
PUFA (g/day)	9.85±4.05	11.30±5.50	0.04	13.52±5.70	16.20±3.54	0.03
Oil (serving/day)	1.20±1.61	2.50±1.05	0.04	2.47±1.07	3.49±0.96	0.05
Cholesterol	133.10±48.50	194.40±40.20	0.04	225.08±135.91	285.45±121.70	0.04
Meat (serving/day)	1.50±0.70	1.65±0.59	0.57	1.95±1.05	2.37±1.17	0.09
Fruits (g/day)	166.76±71.70	126.80±80.60	0.1	247.60±90.50	158.38±100.85	0.04
Vegetables (g/day)	103.64±63.57	95.70±55.80	0.35	126.73±100.36	113.54±74.18	0.29
Fruits (serving/day)	2.15±0.95	1.25±0.95	0.04	2.68±1.05	1.42±0.55	0.04
Vegetables (serving/day)	2.30±1.00	1.18±1.00	0.04	2.87±1.01	1.31±0.70	0.04
Legumes (serving/day)	1.30±0.75	1.25±0.75	0.22	2.20±0.90	1.76±1.25	0.36
Dairy (serving/day)	1.45±0.60	1.30±0.80	0.15	1.69±0.75	1.05±0.65	0.05
Diet-variety	13.30±4.20	11.90±3.90	0.08	13.36±5.05	11.15±4.9	0.06
Sodium/Potassium-md(min-max)	0.51(0.35-1.54)	1.29(0.40-2.17)	0.025	0.93(0.42-1.85)	1.75(0.56-2.37)	0.03

BMI: Body-mass index; WC: Waist circumference; ASD: Abdominal sagittal diameter; BF: body fat; MM: muscle mass; MMI: muscle-mass index; IPAQ: Internacional Physical-Activity Questionary (min-wk)

Table 3: Extreme quartile-range of the Framingham-score distribution in both genders for anthropometric and fitness variables.

	Female			Male		
Parameters	p25 (≤3.0 pts)	p75 (≥10.0 pts)	p	p25 (≤5.0 pts)	p25 (≤3.0 pts)	p
	n=161	n=163		n=51	n=54	
	Mean±SD	Mean±SD		Mean±SD	Mean±SD	
BMI (kg/m^2)	28.47±5.60	30.07±6.57	0.04	29.90±6.16	30.03±5.29	0.6
WC (cm)	91.80±12.79	99.25±14.41	0.02	103.04±14.92	109.61±14.98	0.04
ASD (cm)	22.01±3.75	23.79±3.57	0.04	22.70±3.82	24.71±4.27	0.04
BF (%)	36.30±8.29	40.07±9.11	0.03	26.41±6.03	30.48±6.54	0.04
MM (kg)	22.00±3.34	19.34±3.16	0.04	33.72±5.59	31.59±4.70	0.04
MM (%)	27.25±3.37	24.30±4.23	0.04	38.09±4.19	34.96±4.37	0.02
MMI (kg/m^2)	8.20±1.04	7.50±1.09	0.04	11.61±1.32	9.42±1.18	0.02
Flexibility (cm)	21.59±9.42	20.79±9.78	0.5	22.41±10.51	21.63±10.41	0.1
Hand-grip (kgf)	32.04±9.21	29.07±10.34	0.04	38.19±13.95	33.52±14.17	0.03
IPAQ-md (min-max)	779.50(100.0-4.700.7)	425.8(50.6-2.740.3)	0.03	864.10(120.0-6.100.0)	436.4(74.2-2.4508)	0.03

Table 4: Extreme quartile-range of the Framingham-score distribution in both genders for metabolic stress variables.

	Female					Male				
	p25		p75			p25		p75		
	≤3.0	n	≥10.0	n		≤5.0	n	≥15.0	n	
Parameters	Mean±SD		Mean±SD		p	Mean±SD		Mean±SD		p
HOMA-IR	2.36±0.90	104	4.50±1.20	111	0.03	3.15±1.05	37	5.70±1.10	35	0.5
hs-PCR	0.31±0.14	161	0.53±0.21	163	0.03	0.38±0.10	51	0.61±0.20	54	0.05
MDA	0.57±0.15	128	0.90±0.21	135	0.04	0.45±0.20	43	0.72±0.18	48	0.36

HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; hs-PCR: high-sensitive C-reactive protein; MDA: Malondialdehyde

Table 5: Coronary-Heart Disease Risks (by Framingham scores) distribution according to demographic and social-economic status.

		CHD Risk (by Framingham scores)					
		Low (<10%) (n=270)	Mod (10-20%) (n=585)	High (>20%) (n=212)	L vs M	L vs H	M vs H
Parameters	n	n (%)	n (%)	n (%)	*p*	*p*	*p*
Age-md (min-max)		55.1(35.0-80.0)	54.2(35.0-85.0)	55.3(35.0-85.0)	0.72	0.78	0.77
<60-yrs old	859	23.7[a]	42.1[b]	34.2[ab]	0.04	0.3	0.37
≥60-yrs old	246	22.9[a]	46.0[b]	31.1[ab]	0.04	0.28	0.35
Gender							
Female	941	22.5[a]	45.4[b]	32.1[ab]	0.04	0.42	0.32
Male	164	21.7[a]	47.0[b]	31.3[ab]	0.04	0.45	0.31
Marital status							
Married	991	21.0[a]	43.5[b]	35.5[ab]	0.04	0.35	0.39
Single	114	23.1[a]	54.5[b]	22.4[a]	0.03	0.84	0.03
Schooling							
Uncompleted Elementary	283	12.4[a]	60.2[b]	27.4[c]	<0.0001	0.04	0.04
Completed Elementary	229	14.7[a]	45.1[b]	40.2[b]	0.02	0.03	0.85
High School	407	23.5[a]	40.9[b]	35.6[ab]	0.04	0.29	0.37
Universitary	186	45.2[a]	37.8[a]	17.0[b]	0.52	0.02	0.04
Family income							
< 2 (minimum wage)	340	12.7[a]	49.1[b]	38.2[ab]	0.02	0.04	0.4
2 – 5 (minimum wage)	576	18.4[a]	44.4[b]	37.2[ab]	0.03	0.08	0.31
> 5 (minimum wage)	189	38.4[a]	43.0[a]	18.4[b]	0.45	0.04	0.03
Health Status							
Bad	532	17.8[a]	32.0[b]	50.2[b]	0.04	<0.0001	0.33
Regular	392	30.8[a]	41.1[a]	28.1[a]	0.37	0.81	0.17
Good	181	24.7[a]	60.0[b]	15.3[a]	0.03	0.78	0.02

Thus, behavioral (dietary and physical activity), anthropometric and metabolic stress were major influencing factors of FS. The FS-higher values found in males, contrasted by their higher intake of SAFA, whereas females higher-FS wes characterized by lower-wage earning, lower intake of fibers and higher BMI. However, both genders presented a higher prevalence of bad self-perception of health, lower schooling, low-quality diet (inadequate HEI) with low variety, low dietary intake of fruit, vegetables and higher intake of fat and refined foods (single sugar and sodium), lower physical activity and strength fitness along with anthropometric outlines of high fatness and lower muscle mass.

In addition to anthropometric dystrophies of obesity and sarcopenia (low-muscle mass), there were the underlying mechanisms of gluco-lipotoxicities (inflammation, oxidative stress and insulin resistance) outcoming NCDs of MetS, NAFLD, T2D and CVD.

By extrapolating the FS values to their clinical meanings, projecting the CHD-risk for the next 10 years, our Dynamic-Cohort survey presented 34.3% of the sample with some kind of CHD risk, prevailing the moderate risk (55%) over the high-risk (19.9%) and low-risk (25.1%) of CHD (Table 5). The PRINCEPS (Identification Program of Cardiovascular Risk Level and Increase in Lipid Parameters) study conducted in Spain with individuals of both genders above 45 years of age, observed that established CAD prevalence or CAD risk of 36.9% [36]. This same study found a prevalence of 34.9% of individuals with CAD risk higher than 20% in 10 years [36]. Our present data showed 19.9% high-risk CHD in a sample of free-living subjects, 10-yrs younger than the Spanish study.

Our CHD-risk distribution among ages showed similarity (Table 5). The CHD-moderate risk prevailed in females, in both aging-range (Table 5), 49.5% poor/bad HEI (Table 6), 75.8% inadequate IPAQ (Table 7) and, in those presenting MetS(49.5%), NAFLD(62.3%), and metabolic stress of hs-CRP(68.0%) and MDA(59.8%). (Table 8). Thus, the studied sample was mostly at moderate risk of CHD, but already presenting the co-abnormalities of MetS, and NAFLD and their underlying metabolic stress of inflammation and oxidative stress. Behavioral factors of poor/bad diet (low HEI) and physical inactivity (inadequate IPAQ)/unfitness (low VO2 max. and low flexibility) appeared as major risk factors. Specifically, high-risk

Table 6: Coronary-Heart Disease Risks (by Framingham scores) distribution according to dietary variables.

	CHD Risks					
	Low (<10%) (n=270)	Mod (10-20%) (n=607)	High (>20%) (n=228)	L vs M	L vs H	M vs H
	mediana(mín-máx)	mediana(mín-máx)	mediana(mín-máx)	*p*		p
Parameters						
TEI (kcal/day)	1530.2 (537.5-3810.7)[a]	1640.5 (590.3-4460.0)[ab]	1874.8 (977.9-4661.6)[b]	0.41	0.03	0.21
CHO (%)	52.7 (23.0-75.6.0)[a]	67.2 (42.3-81.9)[b]	72.5 (46.9-87.1)[b]	0.04	0.03	0.63
CHO (g/day)	178.6 (31.3-380.9.1)[a]	234.4 (83.8-464.2)[b]	275.4(91.8-747.7)[b]	0.03	0.01	0.4
Cereal (serving/day)	3.3 (0.0-12.2)[a]	5.4 (0.5-17.9)[b]	5.8 (0.7-19.0)[b]	0.03	0.03	0.8
Sugar (serving/day)	1.6 (0.0-9.5)[a]	2.9 (0.0-13.8)[b]	2.7 (0.0-12.5)[b]	0.04	0.04	0.65
Fibers (g/day)	19.0 (1.0-45.7)[a]	12.9 (1.4-30.0)[b]	8.0 (2.2-28.1)[b]	0.04	<0.001	0.09
CHO/fibers	13.2 (4.0-83.5)[a]	17.9 (8.7-159.9)[b]	21.5 (9.9-193.7)[b]	0.04	<0.001	0.08
Ptn (%)	18.3 (7.1-46.9)[a]	17.8 (5.4-34.3)[a]	17.7 (4.5-43.9)[a]	0.64	0.67	0.7
Ptn (g/day)	63.3 (9.2-169.2)[a]	64.2 (16.0-195.0)[a]	68.4 (13.9-230.5)[a]	0.7	0.73	0.72
Meat (serving/day)	1.3 (0.0-7.1)[a]	1.5 (0.0-5.5)[a]	1.6 (0.0-8.0)[a]	0.8	0.81	0.8
Total Fat (%)	29.0 (8.0-60.0)[a]	30.5 (10.0-63.8)[a]	39.4 (19.5-87.6)[b]	0.69	0.04	0.04
SFA (%)	7.9 (1.4-22.1)[a]	8.0 (1.1-26.0)[a]	9.2 (1.7-36.3)[b]	0.93	0.03	0.04
MUFA (%)	8.7 (1.1-25.0)[a]	8.5 (1.4-22.2)[a]	8.3 (1.4-22.4)[a]	0.78	0.78	0.89
PUFA (%)	6.0 (1.1-18.9)[a]	6.5 (0.7-21.1)[a]	7.8 (0.8-21.4)[b]	0.72	0.04	0.03
Oil (serving/day)	1.5 (0.1-10.9)[a]	2.2 (0.2-10.7)[a]	3.4 (0.5-21.3)[b]	0.52	0.03	0.04
Fruits (serving/day)	2.3 (0.0-19.0)[a]	2.0 (0.0-10.7)[a]	1.0 (0.0-7.3)[b]	0.65	0.04	0.04
Vegetables (serv./day)	2.1 (0.0-18.9)[a]	1.0 (0.0-12.9)[b]	1.2 (0.0-12.4)[b]	0.04	0.04	0.84
Legumes (serv. day)	1.0 (0.0-5.6)[a]	1.0 (0.0-12.5)[a]	1.0 (0.0-9.8)[a]	0.92	0.92	0.94
Dairy (serving day)	1.1 (0.0-12.0)[a]	1.2 (0.0-8.7)[a]	1.2 (0.0-7.2)[a]	0.77	0.76	0.88
Sodium/Potassium	0.6 (0.2-2.35)[a]	1.4 (0.5-6.1)[b]	1.5 (0.3-9.7)[b]	0.04	0.04	0.67
Diet-variety	16.0 (2.1-50.0)[a]	13.5 (5.0-41.1)[a]	11.1 (5.0-35.0)[a]	0.12	0.1	0.66
HEI (points)	85.5(42.0-120.0)[a]	82.1 (49.0-150.4)[a]	79.0.0 (28.1-99.0)[a]	0.72	0.35	0.41

Table 7: Coronary-Heart Disease Risks (by Framingham scores) distribution according to anthropometry and fitness variables.

CHD Risk						
	Low (<10%)	Moderate (10-20%)	High (>20%)			
Parameters	(n=266)	(n=585)	(n=212)	L vs M	L vs H	M vs H
	%	%	%	*p*	*p*	*p*
HEI (points)						
≤70(poor/bad)	23.7[a]	49.5[b]	26.8[a]	0.04	0.52	0.04
70-100(need improvements)	22.5[a]	69.5[b]	8.0[c]	0.03	0.03	0.01
≥100(good)	61.2[a]	26.5[b]	12.3[c]	0.03	0.01	0.04
IPAQ						
< 150min/wk(low)	5.9[a]	75.8[b]	18.3[c]	<0.0001	0.01	0.02
≥ 150min/wk(good-excelent)	29.1[ab]	50.6[a]	20.3[b]	0.15	0.68	0.04
VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)						
≤p25 (poor/bad)	4.7[a]	25.9[b]	69.4[c]	0.03	0.003	0.02
p25-p50 ("regular")	9.3[a]	60.2[b]	30.5[c]	0.001	0.01	0.04
p50-75 (good)	28.6[a]	63.7[b]	7.7[c]	0.04	0.01	0.004
≥p75 (excelent)	64.9[a]	26.3[b]	8.8[c]	0.04	0.005	0.001
BMI (kg/m^2)						
<25.0 (eutrophy)	52.1[a]	27.8[ab]	20.1[b]	0.06	0.04	0.51
25.0-29.9 (overweight)	15.3[a]	46.9[b]	37.8[b]	0.02	0.03	0.19
≥30.0 (obese)	8.0[a]	27.0[b]	65.0[c]	0.01	0.0004	0.04
WC (cm)						
Normal	77.3[a]	12.8[b]	9.9[b]	0.01	0.006	0.29
Altered	7.0[a]	18.9[b]	74.1[c]	0.04	0.001	0.02
ASD (cm)						
≤p25	80.5[a]	13.8[b]	5.7[c]	0.01	0.002	0.03
p25-p50	45.8[a]	29.3[a]	24.9[a]	0.24	0.17	0.62
p50-p75	20.3[a]	21.3[a]	58.4[b]	0.36	0.03	0.03
≥p75	2.3[a]	10.5[b]	87.2[c]	0.01	0.0002	0.01

HEI: Health-eating index; IPAQ: Internacional Physical-Activity Questionary (min-wk); BMI: Body-mass index; WC: Waist circumference; ASD: Abdominal sagittal diameter.

of CHD prevailed in males (50.6%) and in those older than 60yrs (37.4%). In both genders, CHD-high risk was discriminated by lower schooling, lower earning wage and self-perception of poor health status. Additionally, the 19.94% CHD-higher risk were mostly (¾) under inadequate diet (low HEI). Consequently, lower and higher quartiles of Framingham Score had similar distribution of HEI, diet variety and ingested cereal, TEI, CHO, sugar, protein, meat, total fat and dairy. Reflecting that, all three classes of CHD-risks were characterized by a monotonous (low variety of items) diet, presenting similar values of protein intake, and servings of meat, dairy, legumes and %MUFA.

Diet and lifestyle can influence CHD incidence [37]. Multiple studies have demonstrated that a diet containing more fruits and vegetables, fish (particularly, oily fish), whole grains and fiber, and maintaining a caloric balance lowers the risk of CVD [38-41]. From these recommendations, our ingested (inadequate HEI) diet indicated inadequacy of fruits and vegetables, as well as fiber.

Historically, the offered nutritional guidelines [40,42,43] had all very similar consensus statements consistently recommending a dietary pattern higher in fruits and vegetables, whole grains (particularly, high fiber), nonfat dairy, seafood, legumes, and nuts. Furthermore, it is recommended that those who consume alcohol (among adults) do so in moderation. There are also consistent recommendations for diets lower in red and processed meats, refined grains, sugar-sweetened beverages, and saturated and trans fats. The guidelines also emphasize the importance of balancing calories and physical activity as a strategy for maintaining a healthy weight and, thereby, reducing the risk of CVD. Contrary to those guidelines, our top quartil of Framingham Score differentiated from the lower quartile by the higher intake of PUFA, cholesterol,

Table 8: Coronary-Heart Disease Risks (by Framingham scores) distribution according to plasma abnormalities and metabolic diseases.

CHD Risk						
	Low (<10%)	Moderate (10-20%)	High (>20%)			
	(n=266)	(n=585)	(n=212)	L vs M	L vs H	M vs H
Parameters	%	%	%	*p*	*p*	*p*
Dyslipidemia						
Hypertriglyceridemia (≥150mg/dL)	18.9[a]	38.9[b]	42.2[b]	0.04	0.01	0.68
Hypercholesterolemia (≥200mg/dL)	6.8[a]	44.2[b]	49.0[b]	<0.0001	<0.0001	0.85
low HDL-cholesterol (♀<50mg/dL /♂<40mg/dL)	10.1[a]	41.8[b]	48.1[b]	0.01	0.01	0.82
NAFLD						
Absence (FLI<60)	51.2[a]	46.5[a]	2.3[b]	0.33	<0.0001	<0.0001
Presence (FLI≥60)	2.6[a]	62.3[b]	35.1[c]	<0.0001	0.01	0.04
MetS						
Absence(<3components)	53.7[a]	44.4[a]	1.9[b]	0.48	<0.0001	<0.0001
Presence(≥3components)	0.9[a]	63.9[b]	35.2[c]	<0.0001	0.001	0.04
T2D						
Absence(FPG≤125mg/dL)	29.3[a]	57.7[b]	13.0[c]	0.04	0.03	0.01
Presence(FPG>125mg/dL)	0.7[a]	39.6[b]	59.7[b]	<0.0001	<0.0001	0.16
PAI						
<0.11(Low risk)	87.2[a]	10.7[b]	2.1[c]	0.002	<0.0001	0.01
0.11-0.21(Moderate risk)	12.1[a]	83.6[b]	4.3[c]	0.01	0.01	0.003
>0.21(High risk)	0.9[a]	44.4[b]	54.7[b]	<0.0001	<0.0001	0.56
HOMA-IR						
<3.5	49.7[a]	47.0[a]	3.3[b]	0.7	<0.0001	<0.0001
≥3.5	9.6[a]	59.5[b]	30.9[c]	0.01	0.01	0.04
hs-CRP (mg/dL)						
<0.3 mg/dL	49.1[a]	45.6[a]	5.3[b]	0.67	<0.0001	<0.0001
≥0.3 mg/dL	3.3[a]	68.0[b]	28.7[c]	<0.0001	0.01	0.04
MDA (µmol/L)						
≤p25 (≤0.28)	53.4[a]	43.1[a]	3.5[b]	0.54	<0.0001	<0.0001
p25-p50 (0.29-0.51)	28.4[a]	47.9[a]	23.7[a]	0.11	0.61	0.1
p50-75 (0.52-0.72)	21.2[a]	64.9[b]	13.9[a]	0.04	0.49	0.02
≥p75 (≥0.73)	4.0[a]	59.8[b]	36.2[b]	<0.0001	0.0005	0.9

NAFLD: Non-alcoholic fatty-liver disease; MetS: Metabolic Syndrome. FPG: fasting plasma glucose. T2D: Type 2 Diabetes; PAI: Plasma-atherogenic index; HOMA-IR: Homeostasis Model Assessment Insulin Resistance; hs-CRP: high sensitivity C-reactive protein; MDA: malondialdehyde

CHO/Fibers and Na/K, lower intake of MUFA, fruits, and legumes. In agreement with this Framingham Score distribution, the CHD-higher risk also contrasted with the low-risk by its higher energy intake along with fats (total, %SFA, %PUFA, and oil servings), CHO(total, refined cereal and sugar servings), and lower intake of fruits. Thus, overall, the CHD-high risk's diet contrasted by being lower in plants and higher in caloric fat (except %MONO) and refined CHO and ultra-processed foods high in sodium (high sodium/potassium ratio) and single sugar (high CHO/Fiber ratio).

Dietary guidance, over the past 20 years, has moved from the panacea of specific foods and nutrients to a primary emphasis on dietary patterns to lower the risk of CVD. Though, dietary patterns have been recommended: the US healthy eating pattern, Low-fat diets, Mediterranean diet, DASH (Dietary Approaches to Stop Hypertension) diet, Vegetarian diet, and Plant-based diets. Generically, the Mediterranean diet, rich in plant foods in combination with nonsmoking, moderate alcohol consumption, and daily physical activity, is associated with a significantly lower mortality rate of CHD and all-cause mortality [44]. "Plant-based diets" are defined by an emphasis on plants, including fruits and vegetables. Low-fat diets, the Mediterranean diet, DASH diet, and vegetarian diets are all, in essence, plant-based diets since they emphasize fruits and vegetables and legumes and nuts, and limit the amount of red meat, processed meat, sweets, and oils [38,40,45,46]. Concerning the question about what actually constitutes a "plant-based" diet [47-51], it has been established a healthy plant-based diet index (hPBDI). In hPBDI, healthy plant foods (whole grains, fruits/vegetables, nuts/legumes, oils, tea, coffee) receive positive scores and less healthy plant foods (juices/ sweetened beverages, refined grains, potatoes/fries, sweets, and animal foods) receive adverse scores used to create an unhealthy plant-based diet index (uPBDI) [52]. The data analysis in the Nurses' Health Study and Health Professionals Follow-up Study showed that diets that scored high in the hPBDI category substantially lowered CHD risk, whereas foods in the uPBDI, which emphasized less healthy plant foods, were associated with higher CHD risk [32].

Specifically, on blood pressure, one capital risk factor for CHD, the AHA/ACC Guidelines [46], highlighted the importance of lifestyle modalities in the blood pressure recommendations. In addition of consuming a diet high in vegetables, fruits, and whole grains, including low-fat dairy, poultry, fish, legumes, and nontropical vegetable oils and nuts, while limiting sweets, sugar, sugar-sweetened beverages, and red meat, the consumption of sodium should be no more than 2400 mg of per day. Furthermore, it additionally recommended that individuals should attempt to achieve a further reduction of sodium intake to 1500 mg per day [46]. Along with high sodium, lower consumption of either potassium or calcium are potential dietary risk factor for blood hypertension [53]. Presently, CHD-higher risk showed higher dietary intake of sodium/potassium along with lower dairy and plasma- calcium/protein ratio, denoting low-calcium intake. Besides consumption of high fat and/or high-salt diets, high blood pressure is often associated with unhealthy lifestyles such as physical inactivity [24].

Regular physical activity, adequate nutrition, weight management, and not smoking cigarettes have all been demonstrated to significantly reduce the risk of CVD [32]. Compared with those who are very physically active, the risk of CHD in sedentary individuals is 150% to 240% higher [54]. Presently, physical inactivity (inadequate IPAQ) and mainly, lower aerobic capacity (low-VO2max) were risk factors for discriminating the class of CHD-high risk (Table 7). The recommendation is to engage in aerobic physical activity 3 to 4 sessions per week, lasting an average of 40 minutes per session of moderate to vigorous intensity physical activity [46]. For achieving 75% of the maximum benefits, the CDC (2013) recommends at least 150 minutes per week of moderate intensity aerobic exercise or at least 75 minutes of vigorous exercise and muscle strengthening activities at least 2 days per week [55]. However, according to the Americans 2018 guidance, only about 25% of all Americans engage in the minimum standards [56].

Our physical inactivity rate averaged 18.3% in the whole sample, averaging 32.4% with CHD and reaching 75.8% in CHD-moderate risk, 18.3% in CHD-high risk, and 5.9% in CHD-low risk (Table 7).

The present data pointed out the detected CHD-high-risk as being 37.8% overweight and 65.0% obese. Both overweight and obesity represent significant risk factors for CVD [32]. In the United States, the estimates indicate that the prevalence of overweight (body mass index [BMI] 25-30 kg/m^2) for adult women is 30% and for adult men approximately 40% [57]. Estimates of obesity (BMI □30 kg/m^2) are currently 40% for women and approximately 35% for men [58]. In our sample, the rate was close for obesity (39%) and more than double (82%) for overweight. AHA lists obesity as a major risk factor for CVD not only because of its association with other risk factors (eg, diabetes, dyslipidemias, elevated blood pressure, metabolic syndrome) but also because it serves as an independent risk factor [59].

The distribution of body fat also carries an additional risk factor for CHD. It is known that accumulation of intra-abdominal fat promotes insulin resistance, which can lead to glucose intolerance, elevated triglycerides, and low HDL, as well as hypertension [60]. Along with 65.0% higher-BMI, our CHD-high risk class showed 74.1% altered-WC and 87.2% altered-ASD (Table 7). Therefore, there was concordance between the top quartil of Framingham Score and the CHD-high risk class, with both presenting anthropometric dystrophies of body fatness (higher BMI, WC, ASD and %BF) and sarcopenia (lower %MM and MMI), associated with higher prevalence of MetS, NAFLD, and atherogenic dyslipidemia, along with plasma-markers of metabolic stress (higher hs-CRP, HOMA-IR, and MDA). (Tables 7 and 8)

Fatty-tissue dystrophies and dyslipidemia are considered strong risk factors for CHDs. The MetS, particularly, is a multiple risk factor for cardiovascular disease and is characterized by increased waist circumference, raised triglycerides, reduced HDL cholesterol, elevated blood pressure, and raised plasma glucose

[61,62]. From these, reduced HDL-c and elevated blood pressure are common components of both CHD risk and MetS. Some factors as dyslipidemia, hyperglycemia, hypertension, obesity and other risk factors like low levels of physical activity and smoking [63] have already been well established as CHD risk factors. Presently, the CHD was increased 1.23X by the presence of MetS, 1.32X by obesity, 2.29X by NAFLD, 2.62X by T2D and 2.71X by atherogenic dyslipidemia (Figure 1a). It was noteworthy that low-muscle mass (sarcopenia) had a stronger connection (2.29X) with higher CHD-risk, than obesity(1.32X). Attained to the dietary and unfitness risk-factors (Tables 6 and 7, and Figure 2a), there was an increased level of underlying metabolic stress, notably the reduced antioxidant defenses and higher pro-oxidants, inflammatory, and insulin resistance (Table 8 and Figure 1a).

After the cross-sectional analysis to establish the CHD risks, a sub-sample of 567 subjects was submitted to a lifestyle intervention (LiSM) with supervised physical exercises (aerobic-mixed: endurance and resistance) and a counseled diet [3,64]. After 10-wk LiSM, there was a significant reduction of Framingham Score (8.8%), CHD risk (22.6%), CHD-moderate risk (26.4%) and CHD-high risk (40.9%) (Table 9). Paralleling the improvement of CHD-risks, there were improvements also in physical activity, fitness and dietary intake (Figure 2b), leading to an increase in antioxidant markers and muscle mass, along with reductions of metabolic stress and fat dystrophies/dyslipidemia/T2D (Figure 1b).

Table 9: CHD: Coronary Heart Disease; M0: Baseline; M1: 10-wk intervention.

Variables	N	M0	M1	p values	% Effectiveness
Framingham Score	567	5.7	5.2	0.04	8.8
% CHD-Risk	567	12	8.9	0.02	22.6
% Mod. CHD-Risk	567	31	23	0.04	26.4
% High CHD-Risk	567	4.4	2.6	<0.001	40.9

CHD: Coronary Heart Disease; M0: Baseline; M1: 10-wk intervention

Final Remarks and Conclusions

The cross-sectional data pointed out the Incremental risk of CHDs as determined by older aging, lower schooling, total body(BMI) and abdominal fatness (WC and ASD) dystrophies, low-muscle mass, blood hypertension, atherosclerotic dyslipidemia and abnormal stress markers of inflammation(hs-CRP), oxidative stress (MDA) and insulin resistance(HOMA-IR). For contrasting CHD-higher risk, the detected environmental factors of inadequate dietary

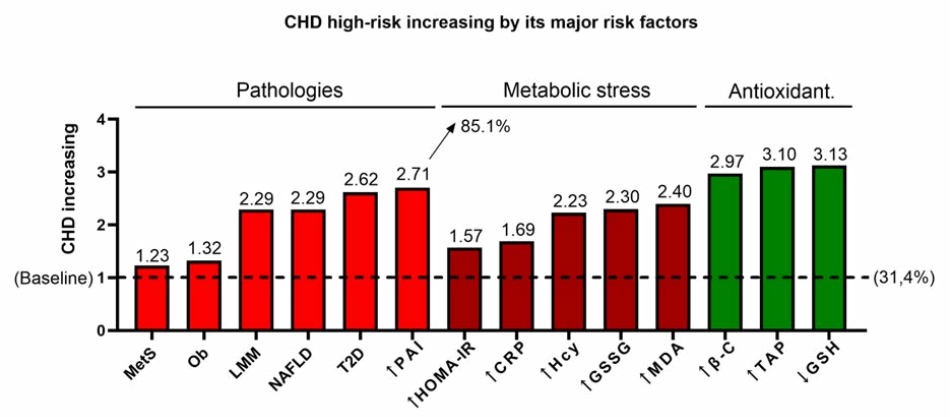

Figure 1a:

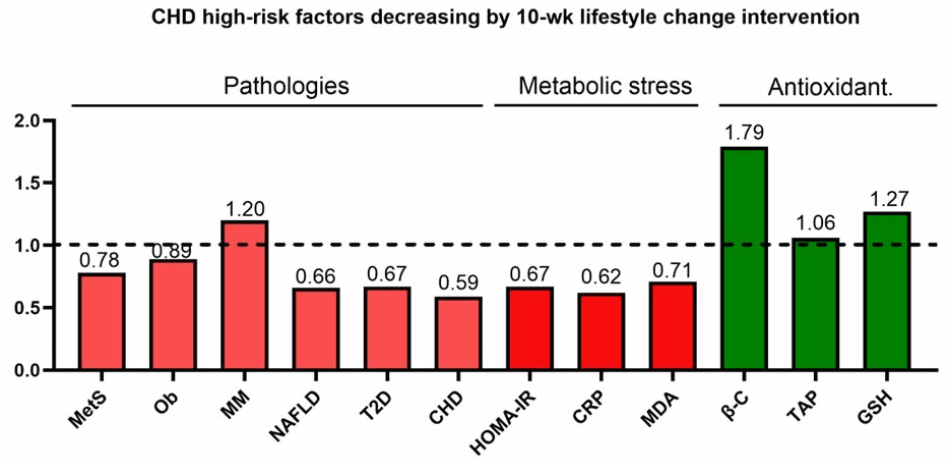

Figure 1b:

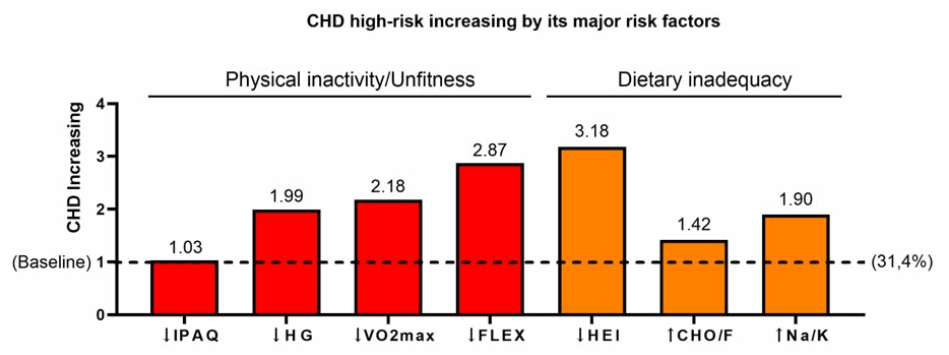

Figure 2a:

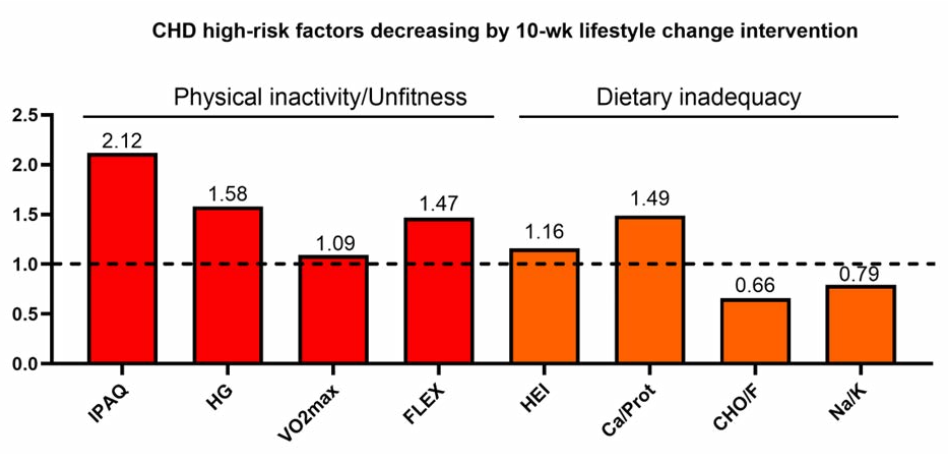

Figure 2b:

quality(HEI) and physical inactivity(IPAQ) were considered discriminatory but weaker discriminant factor compared to low VO2max. Additionally, to the considered algorithm components of Framingham Score (body overweight, blood hypertension, atherogenic dyslipidemia and T2D), we found low-muscle mass and its underlying oxidative stress mechanism, also closer related with the atherogenic dyslipidemia and (T2D) glucotoxicity that precede CHD-high risk. Body fatness dystrophy-related inflammation, oxidative stress-related low-muscle mass and, related insulin resistance, are all under behavioral effects of inadequate diet and physical inactivity (and unfitness). In the CHD-high risk subjects, dietary inadequacy was characterized by a higher caloric intake, high (saturated) fat that would explain the found dyslipidemia and body overweight. The over-fatness may be boosted by the existing physical inactivity and consequent lower daily-energy expenditure. Particularly, the abdominal obesity compromises the fitness of aerobic conditioning and trunk flexibility, while it may conserve or even increasing, the arm strength. Greater adipocyte hypertrophy may trigger systemic inflammatory-lipotoxicity responses as seen here. Inadequate (low variety) diet rich in refined foods, as seen here for the CHD-high risk subjects, may led to T2D (by single sugar intake) and, to a blood hypertensive state by the higher intake of sodium and lower intake of potassium (from fruit and vegetables) and calcium (from dairy). The low color-vegetables ingested, along with lower plasma-markers of antioxidant defenses, may explain the oxidative stress state that followed glucotoxicity of T2D and atherogenic dyslipidemia (higher PAI), which would lead to the accompanied muscle-mass atrophy, and consequently, lower fitness of strength and aerobic conditioning.

Trying to solve the CHD-risk factors raised by the cross-sectional analysis, a free-demanded sub-sample was submitted to longitudinal intervention with lifestyle-change protocol (LiSM) with supervised physical exercises and dietary counseling. The intervention normalized IPAQ by increasing its leisure domain. In addition, the 10-wk LiSM increased Vo2max, HEI, and muscle mass, reduced obesity and mostly of other NCDs along with their underlying factors of inflammation and oxidative stress. Framingham score and CHD-risks decreased, but not the averaged CHD-risk class (8.25% to 7.70%). Thus, the Lifestyle Medicine proposed approach would be an effective and money-saving alternative to the current homeostasis approach, for the CVDs 2nd care.

Future Directions

The soaring of NCD-epidemic would be consequent to the ineffectiveness of the classical-treatment approach of homeostasis model which besides expensive, (drug therapy) has been largely unsuccessful in halting and reversing the constellation of common NCDs such as CVDs. Trying to solve the (CHD-risk) factors

raised by the cross-sectional analysis, we used an allostasis approach of intervention with a science-based lifestyle-change protocol (LiSM) with supervised physical exercises and dietary counseling. As expected, the intervention normalized physical activity and allowed dietary and body-composition improvements along with reductions in most other NCDs. Associated with that, there was significant reduction in underlying mechanisms of gluco-lipotoxicity and of course, in our main goal, a decreasing in Framingham score and the prevalence of its indicateded CHD risks. The findings were not new! It has been a growing acceptance, that daily habits and actions powerfully affect the risk of cardiovascular diseases (CVD), in general, and coronary heart disease (CHD), in particular. Countless studies, including cohorts, support the concept that regular physical activity, maintenance of a proper weight, sound nutritional practices, and avoiding tobacco products all significantly reduce the risk of CVD. A reduction of risk of CVD of >80% was demonstrated in individuals who followed a cluster of these lifestyle practices [32]. However, despite overwhelming evidence that lifestyle factors significantly affect short- and long-term health and quality of life, it has been frustratingly difficult to help patients adopt these habits and practices [32,64]. The AHA released Strategic Plan for 2020, concluded that only 5% of individuals achieved "ideal cardiovascular health," which involved a series of lifestyle factors such as regular physical activity, sound nutrition, weight management, and avoidance of tobacco as well as some cardiovascular health–related factors such as control of cholesterol, blood pressure, and glucose [42]. Adherence to positive lifestyle factors also remains a challenging area. Despite uncontested improvements, the decreasing compliance with our long-lasting protocols has been one of our major LiSM-facing problems [64,65]. The lifestyle strategies for the reduction of risk for CVD in many instances involve changes in behavior [66], and multiple frameworks for behavioral change have been outlined [64]. Scientific Statements from the AHA have emphasized the importance of implementing behavioral guidelines [67], and the challenge to the medical and health care communities would be to more aggressively incorporate this information into the daily practices of (lifestyle)medicine [32]. Within the past decade, the AHA and the ACC have been leaders in promoting the power of lifestyle habits and practices as key factors in promoting cardiovascular health. In the AHA Strategic Plan, the concept of "primordial" prevention (preventing risk factors from occurring in the first place) was introduced into the cardiovascular lexicon in addition to the construct of "ideal" cardiovascular health [42]. This discipline has coalesced under the framework of "lifestyle medicine" and a series of articles was published in *Circulation* (2011) titled "Recent Advances in Preventive Cardiology and Lifestyle Medicine". The AHA Council, which had previously been called "Council on Nutrition, Physical Activity and Metabolism," in 2013, changed its name to the "Council on Lifestyle and Cardiometabolic Health" [68].

It has been demonstrated that the greatest benefit in terms of reduction of risk of CHD appears to come to those engaging in even a modest amount of physical activity compared to the most physically inactive [69]. However, a survey among primary-care physicians showed, at the turn of the century, only 12% aware of the recommendations from the American College of Sports Medicine [70]. This was aggravated later by another survey of internal medicine residents reporting that 88% were confident in their knowledge of the benefits of exercise, but only 25% demonstrated adequate knowledge useful for patient counseling [71]. Thus, the obtained results could be improved by either more adherence of patients to the positive lifestyle procedures, compliance with longer-lasting LiSM, and by challenging the medical and health care communities to more aggressively incorporate knowledge into the daily practices of (lifestyle)medicine.

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