Sprint 8 - Complete Clinical Study

Targeting exercise-induced growth hormone release:

A novel approach to fighting obesity by substantially increasing endogenous GH serum levels naturally.

David Braden, MD1,2; Jeff Ross, MD1; Leigh C Gray, MD 1; Cheri Walker, RN, MSN1;

Alvin Hoover, MS, MEd1; and S Duane Burt, MS1,3

1 King's Daughters Medical Center, Brookhaven, MS, USA

2 University of Mississippi Medical Center, Jackson, MS, USA

3 Copiah-Lincoln Community College, Wesson, MS, USA

Corresponding Author: S Duane Burt, Science Department, Copiah-Lincoln Community College, 1028 JC Redd Dr., Wesson, MS, 39191, USA. Email: duane.burt@colin.edu Phone: (601) 643-8369 begin_of_the_skype_highlighting (601) 643-8369 end_of_the_skype_highlighting

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ABSTRACT

Background: The problems stemming from obesity are well-documented, and the need for effective intervention is critical. The Sprint 8 exercise protocol has shown to fight obesity by naturally invoking significant growth hormone (GH) release. GH serum levels are known to stay substantially elevated for two hours after exercise, where it initiates lipolysis, inhibits the uptake and storage of other lipids, and induces muscle hypertrophy.

Objective: In this novel approach to find relief for obesity, the goal was to efficiently and optimally maximize natural GH release to metabolize adipose tissue while GH levels were prominent, thus find a natural, economical, and efficacious obesity-reduction strategy.

Method: By incorporating intermediate fast-twitch (type IIA) and fast-twitch (type IIB) muscle fibers and their associated anaerobic metabolic processes, body temperature rises and lactic acid production increases substantially, lowering blood pH, affording significant GH release. Results: Among eleven participants [mean age = 46.0 (±10.0 yrs)], GH serum levels increased 771% following the initial bout of the Sprint 8, and while not all participants were at risk for CVDs, cholesterol lowered 12.3%, LDL dropped 15.0%, triglycerides decreased 26.8%, HDL increased 2.0%, body fat percentage lowered 31.0%, and BMI dropped 4.5%.

Conclusions: The Sprint 8 requires 20 minutes of exercise, 3 days per week, without dieting, and is an efficient and economical protocol that naturally combats obesity, benefiting individuals socially and physically, and ultimately healthcare systems financially. Unexpectedly, the results of the Sprint 8 trial are similar to those produced by the cholesterol-lowering statin medications, demanding further investigation.

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BACKGROUND AND INTRODUCTION

Obesity

It is well documented that obesity is a major problem in the U.S. and other countries. It is associated with many diseases, disabilities, discriminations, and financial hardships.[1] Obesity is correlated with hyperlipidemia or elevated blood cholesterol, LDL, and triglycerides, accompanied with low HDL serum levels.[2] Hyperlipidemia leads to many cardiovascular diseases (CVDs), particularly atherosclerosis, which is the leading cause of death for men and women not only in the U.S., but in the world.[3,4] Additionally, the healthcare industry is burdened with the billions of dollars spent on the many ensuing chronic diseases derived from the over-shadowing obesity problem. Some of these diseases and problems evolving from obesity include: hyperlipidemia, diabetes, hypertension, coronary heart disease, osteoarthritis, gall bladder disease, impaired fertility, complications during pregnancy, breathlessness, sleep apnea, gout, low-back pain, and cancer. Indeed, the highest utilization costs in the healthcare industry belong to CVDs. [5,6] In order to reduce patient suffering, as well as alleviating the financial burden carried by the healthcare industry, the need for an efficacious intervention is crucial.

Exercise and Growth Hormone

It is also well documented that exercise is a major component to having a healthy lifestyle. While most anti-obesity programs today focus on diet and lifestyle changes, their exercise components generally focus on working only slow-twitch (type I) muscle fibers, which include mostly aerobic physiological processes. These current exercise methods so commonly utilized neglect the incorporation of intermediate fast-twitch (type IIA) and fast-twitch (type IIB) muscle fibers and their associated anaerobic metabolic processes, making them generally ineffective.[7] It is known that growth hormone (GH) release and exercise are correlated. The National Institutes of

Health reports that exercise-induced GH, when released, can increase by 500% and stay significantly elevated for up to two hours.[8] During this time, GH is known to have multiple functions, including playing a role in muscle hypertrophy and targeting the metabolism of triglycerides, the foundation of not only obesity and hyperlipidemia, but the other ensuing diseases previously noted.[9,10] It is generally accepted that anaerobic exercise shows to increase GH levels far more than aerobic activities alone; [10,11] however, research is scarce when studying types IIA and IIB muscle fibers with their associated anaerobic metabolic processes and the resulting effect of GH release during exercise. What is understood is that when undertaking anaerobic activity, lactic acid builds in the muscular system and ultimately the blood, where blood pH is lowered. Body temperature is also elevated during exercise. Increased body temperature and the declining pH of blood induces GH release from the anterior pituitary gland, possibly for the purpose of repairing muscles by way of elevating amino acid uptake within muscle cells, increasing muscle cell protein synthesis, [12] and also increasing sweat release to cool the body.[13] In turn, increased GH levels trigger insulin-like growth factor 1 (IGF-1) release from the liver as well as from other tissues, including muscle tissue. IGF-1 is known to be a potent anabolic hormone. GH and IGF-1 synergistically increase muscle cell protein synthesis leading to muscle hypertrophy. [14,15] At the same time, GH initiates lipolysis within adipose tissue for energy. Adipose cell membranes contain GH-binding protein receptors. When binding to adipose receptors, GH stimulates the metabolism of triglycerides while inhibiting the uptake and accumulation of other circulating lipids.[16] Therefore, when utilizing types IIA and IIB muscle fibers, exceptionally high GH serum levels are produced, which direct the building of muscle mass that ultimately feeds on adipose for maintenance and increases the metabolic rate of fatty tissue. It is apparent that obesity is attacked far more efficiently than with the utilization of type I muscle fiber alone and that elevated exercise-induced GH serum levels show to have promising effects for the growing problem of obesity and associated hyperlipidemia. However, as previously discussed, the most popular anti-obesity programs today only utilize type I muscle fibers and do not take advantage of the surplus of GH that could be produced by incorporating type IIA and IIB muscle fibers.

The Sprint 8 Protocol

The underlying thesis behind the Sprint 8 protocol is to optimally and efficiently maximize exercise-induced GH release by exercising under anaerobic conditions. Along with the common utilization of type I muscle fibers, the Sprint 8 incorporates types IIA and IIB muscle fibers. As previously discussed, by recruiting muscle fiber types IIA and IIB during exercise, the body is forced to engage anaerobic metabolic processes to continue muscle function, producing large amounts of lactic acid and increasing body temperature, which stimulates GH release from the anterior pituitary gland. The heightened release of GH from the Sprint 8 regimen produces major fitness improving benefits, as well as time-saving benefits in regard to exercise, that are realistic and achievable by most healthy adolescents and adults. Only 20 minutes per bout, 3 times per week, totaling a mere 8 hours of exercise per 8 weeks, is required. The Sprint 8 exercise protocol was developed by a healthcare professional who is a certified trainer with the American College of Sports Medicine with 37 years of experience. It was created in the 1990s and is published in the 384-page book entitled Ready, Set, Go! Synergy Fitness for Time-Crunched Adults, by Campbell, (2001, 2010).[7] The protocol is consistent with the latest cardiovascular guidelines (2007)

established by the American Heart Association and the American College of Sports Medicine for vigorous intensity cardiovascular exercise. [7] In this novel approach to finding relief for the obesity crisis, preliminary studies of the Sprint 8 protocol indicate that GH is indeed released naturally and abundantly, and produces a wealth of promising data in dealing with not only increasing muscle mass, but with the obesity and hyperlipidemia epidemic. Additionally, there is no diet required, the muscular system benefits, and much time is saved in regard to exercise.

MATERIALS AND METHODS

Subjects (Pre-Sprint 8 trial)

Eleven random, working adults, representative of middle-aged employees of a rural hospital located in Mississippi, volunteered and participated in this study. Mississippi is consistently ranked as the most obese state in the U.S., thereby ranking poorly in the numerous health status measures like diabetes and premature death from CVDs. Two male and nine female subjects, aged 31-57 [mean age (±SD)] = 46.0 (±10.0 yr.), mean weight 86.3 kg (189.9 ±37.0 lbs.), mean body fat % = 35.1 (±6.6%), mean BMI = 30.9 (±5.7 kg/m2), mean blood cholesterol level = 230.8 (±34.1 mg/dL), mean blood LDL level 156.0 (±33.9 mg/dL), mean blood triglyceride level = 116.8 (±57.3 mg/dL), mean blood HDL level = 51.5 (±9.2 mg/dL), mean GH level = 1.1 (±1.3 ng/mL) volunteered and participated in the Sprint 8 trial. All participating subjects were trained on the theory behind the project and met with a Sprint 8 trained hospital staff member to begin their personal exercise regimen. It is important to note that there was no oversight on the subjects during the 8 week trial and that their adherence to the program was strictly voluntary. Subjects were asked to continue their daily routines without change, so the only variable on health measures during the eight week test would be the impact of the Sprint 8 protocol, 20 minutes a day, three days per week, for 8 weeks.

Sprint 8 Trial

The recent Sprint 8 trial (2011) was conducted over an eight-week period, three days per week, 20 minutes per day, totaling 8 hours of exercise during the 8 week test period. Group preparation consisted of one 45 minute preparatory session explaining the concepts previously described. No instructions were given on dietary management for the trial or any additional educational reinforcement beyond the initial session. During the 20 minute exercise, subjects began with a 2.5 minute warm-up period, followed by 30 seconds of full-sprint cardiovascular activity to induce anaerobic metabolism and lactic acid build-up. After the 30 second cardio sprint, subjects returned to a slower active recovery pace for 1.5 minutes. After the 1.5 minute active recovery, subjects repeated the sprint for 30 seconds. This process continued until eight sprints were performed, with a final 3 minute cool-down period to total 20 minutes. Standard aerobic gym equipment was utilized among the participants. These include any upright, stationary bike, stationary recumbent bike, treadmill, and/or elliptical trainer. Participants were free to choose their exercise equipment at any given session; there was no fixed machine used at any given session.

Blood Sampling and Analysis

A panel of lab tests indicative of health and wellness were conducted pre- and post-Sprint 8 trial for baseline measurements. Cholesterol, triglyceride, LDL, HDL, and GH serum levels were obtained for each test subject to determine the impact of the program on basic health and wellness. Blood lipids (cholesterol, triglycerides, LDL, and HDL) were analyzed by a Siemens Dade Dimension® ExL™ integrated chemistry system at King's Daughters Medical Center, Brookhaven, MS. GH level assays were performed by Laboratory Corporation of America® Reference Laboratory (LabCorp) in Birmingham, AL, which analyzes GH by the immunochemiluminometric (ICMA) assay method. During week one (pre-Sprint 8 trial), initial 10 hour fasting blood tests were conducted before exercise to establish a baseline measurement. A total of 16.0 mL of whole blood was drawn from each subject, with 4.0 mL of plasma used for the basic metabolic and lipid panel measures, 5.0 mL of ethylenediaminetetraacetic acid (EDTA) plasma used to test glycated hemoglobin (A1C) levels, and 2.5 mL of serum sent to LabCorp for GH measure. Additionally, 7.0 mL of blood was drawn within 30 minutes of the initial bout of the Sprint 8 program with 2.5 mL of serum sent to LabCorp for another GH post-exercise measure. Final blood labs identical in nature to those measured in week one were performed at the end of week eight, both fasting for an ending baseline, and within 30 minutes of the final exercise of the program for a final GH measure.

Weight, Body Fat %, and BMI

Weight, body fat percentage, and BMI data were obtained pre- and post-Sprint 8. Weight measurements were obtained by utilizing a Rice Lake® medical scale. Body fat percentage results were obtained by employing a Futrex-5000Ai® body fat analyzer which uses IR light refraction. The measurements were taken on the dominate bicep of test subjects. BMI measurements were acquired using the Body Mass Index Calculator of the U.S. Department of Health and Human Services National Heart, Lung, and Blood Institute. These measurements were obtained from test subjects pre- and post-Sprint 8 trial.

RESULTS

GH Levels (Post-Sprint 8 trial)

The collective mean GH level was 9.8 (±14.7 ng/mL) and was collected within 30 minutes of post-initial exercise, pre-Sprint 8 trial. Here, GH levels increased 771% from the baseline value (1.1 ng/mL). Additionally, 1.0 (±1.2 ng/mL) was the collective mean GH level obtained pre-exercise, post-Sprint 8 trial and used as a baseline. When compared to 5.9 (±6.3 ng/mL), the determined collective mean GH level taken within 30 minutes post- (final) exercise, post-Sprint 8 trial, the

data affords a 486% increase of GH serum levels. Collective comparisons of pre- and post-Sprint 8 exercise-induced GH level increases are represented in Graph 1. Collective and individual GH data are presented in Table 1.

Body Mass

The eleven subjects collectively lost 48.2 kg (106 lbs.) of fatty tissue. On average, each subject lost 4.4 kg (9.6 lbs.) of fatty tissue. The post-trial mean weight = 82.0 kg (180.3 ±34.3 lbs.). Post-trial mean body fat % = 24.2 (±5.1%), mean body fat % reduction = 31.0%, post-trial mean BMI = 29.5 (±5.6 kg/m2), post-trial mean % BMI reduction = 4.5%. These data are represented in Table 2.

Lipid Levels

Post-trial mean blood cholesterol level = 202.3 (±36.1 mg/dL), mean blood cholesterol level % lost = 12.3%, mean blood LDL level = 132.7 (±33.4 mg/dL), mean blood LDL % loss = 15.0%, mean blood triglyceride level = 85.5 (±28.2 mg/dL), mean blood triglyceride % loss = 26.8%, mean blood HDL level = 52.5 (±10.8 mg/dL), mean blood HDL % gain = 2.0%. Table 3 identifies these data. Graph 2 demonstrates the differences in mg/dL of cholesterol, LDL, triglyceride, and HDL pre- and post-Sprint 8 trial.

DISCUSSION AND CONCLUSIONS

The Sprint 8 and Future Studies

GH has been touted as a miracle drug, an anti-aging medication, has been banned from organized competitive athletics due to the anabolic effects and resulting unfair advantages it produces, and because of the many other positive physiological effects GH is known to produce, billions of dollars are spent annually on artificial and supplemental GH therapy, both legally and illegally. However, side effects are associated with artificial GH injections, and they include: hyperlipidemia, arthritis, cardiomegaly, impotence, weakened glucose regulation and possibly type 1 diabetes. Research indicates that exercise-induced GH release is natural, more potent, and much safer than artificial injections.[10,16,17] Furthermore, as discussed, most obesity-reduction programs prove to be ineffective because the problem is generally addressed with a narrow focus, which includes calorie-reducing diet programs coupled with exercise regimens that increase activity within the aerobic energy system (type I muscle fibers) alone. These programs generally neglect the anaerobic energy systems (types IIA and IIB muscle fibers). The results of the Sprint 8 exercise protocol indicate that by utilizing types IIA and IIB muscle fibers, exceptionally high amounts of exercise-induced GH are released, and these elevated GH levels show to combat hyperlipidemia and obesity. Due to the lack of research involving types IIA and IIB muscle fibers coupled with GH release during exercise, more studies are necessary regarding the Sprint 8. However, past research has acknowledged the beneficial results exercise-induced GH produces and has proposed that finding the optimal factors in obtaining the greatest natural

GH release remains elusive. The Sprint 8 offers the efficient and economical answer to naturally and significantly elevating GH serum levels in a very short time period (20 minutes per bout).

The possibilities and benefits produced by the Sprint 8 program and the associated elevated GH serum levels that correspond need to be explored. Future studies involving specific population groups, with a primary focus on childhood obesity, as well as other large demographic populations, are warranted. If the Sprint 8 can be implemented in elementary schools or nursing homes, the benefit would be enormous. At the cellular level, what additional effects are produced by naturally elevated GH levels, such as the increase in numbers of mitochondria, or changes that may occur in naturally GH deficient patients? What other positive health-related effects are produced among participants undertaking long-term Sprint 8 activity? The health care industry would be less burdened by the innumerable diseases and disabilities derived from obesity and hyperlipidemia. Additionally, billions of dollars would be saved in health care expenses.

There was no diet associated with this study, and results remained beyond promising. What would results be if the Sprint 8 was linked with a diet, or perhaps strength training with naturally elevated GH levels? A number of the Sprint 8 test subjects described after the mere 8 week program that they no longer require medications that they were previously prescribed, most of which were high blood pressure or cholesterol-reducing medications.

The Sprint 8 and Statin Medications

Indeed, as is the nature of scientific research, a surprising and unexpected discovery after the 8 week trial indicates that the data presented here shows to mimic that of the cholesterol-lowering statin medications. The statin drugs, or HMG-CoA reductase inhibitors, are by far the most effective medications prescribed for reducing cholesterol and LDL levels in serum and, to a lesser degree, raising blood HDL levels.[1,18] Hence, they are the top-selling prescription medications in the U.S.[19] One statin, atorvastatin, better known as Lipitor®, is the highest selling statin medication and was responsible for $12.4 billion of revenue for Pfizer in 2008 alone.[20] HMG-CoA reductase inhibitors work by eliminating the first committed step in sterol biosynthesis within the liver, therefore dropping LDL levels in the liver. Furthermore, the liver then up-regulates cholesterol receptors on hepatocyte membranes, which take in existing cholesterol from the blood, reducing serum cholesterol levels even further.[21] These functions have substantially proven to reduce the incidence of CVDs, and it would be difficult to find a rival to the statin medications. However, many negative side effects have been demonstrated with the use of statins. Minor side effects include bloating and abdominal cramping,[1] possible psychiatric events, including insomnia,[22] and possible erectile dysfunction.[23] More serious side effects include hepatotoxicity and acute renal failure due to myoglobinuria. Rhabdomyolysis with renal dysfunction has occurred as well.[1,24] The most common side effects are indeed related to the muscular system.[25] Myalgia, myopathy, and other complications due to drug interactions are commonly reported.[24,26] Despite the side effects, patients and the healthcare industry spend billions of dollars on statins annually.

When compared to data in the literature, the Sprint 8 results presented here resemble those of the statins, without the side effects. It is noted that the Sprint 8 and Reversal of Atherosclerosis with

Aggressive Lipid Lowering (REVERSAL) trials do not compare in the overall number of subjects or in length of trials. However, the REVERSAL trial was an 18 month study whose goal was to compare intensive and moderate lipid-lowering treatment for atherosclerosis.[27] Random patients (253) were prescribed atorvastatin (80 mg/day/18 months), which has the highest efficacy at lowering LDL levels, and were considered the intensive therapy group. Pravastatin, a very efficacious medication in raising HDL blood levels, was prescribed to 249 random patients (40 mg/day/18 months) and were considered the moderate therapy group.[18,27] Results of the REVERSAL trial indicate that the atorvastatin group lowered LDL levels 46.3% (baseline = 147 mg/dL) lowered cholesterol levels 34.1% (baseline = 230 mg/dL), lowered triglyceride levels 20% (baseline = 186 mg/dL), and increased HDL levels 2.9% (baseline = 44.4 mg/dL). The pravastatin group lowered LDL levels 25.2% (baseline = 147 mg/dL), lowered cholesterol levels 18.4% (baseline = 230 mg/dL), lowered triglyceride levels 6.8% (baseline = 178 mg/dL), and increased HDL levels 5.6% (baseline = 42.2 mg/dL).[27] Graph 3 compares results of the Sprint 8 and REVERSAL trials [27] and their corresponding percent losses of LDL, cholesterol, and triglycerides, and gains of HDL. Graph 4 compares the baseline data of the pre-Sprint 8 and pre-REVERSAL trial.

Although not as significant as the prescribed statin medications themselves, it is remarkable that the Sprint 8 program produces the same trends in lowering cholesterol, LDL, and triglyceride levels in blood serum, while increasing HDL serum levels. This was accomplished with a mere 8 hours spent exercising within 8 weeks, a fraction of the 18 month REVERSAL trial, and with absolutely no diet or medication involved. Additionally, muscular systems profited, and no side effects were described. This data warrants further investigation.

It is important to note that the subjects compared in the Sprint 8 and the REVERSAL trial were in different states of health. The data indicates that the Sprint 8 subjects were a healthier group studied when compared to those of the REVERSAL trial, who were at a greater risk for CVDs. Sprint 8 baseline cholesterol and LDL levels were similar to those found in the REVERSAL trial; however, triglyceride levels were significantly lower while HDL levels were higher (Graph 4). This data concludes that less percentage (and/or points) of cholesterol, LDL, and triglycerides would be lost during the Sprint 8 program (or gain of HDL). Nonetheless, the Sprint 8 subjects who were not necessarily considered at risk for CVDs or in need of statin medications are factored into the data presented here.

The Sprint 8 protocol naturally and optimally maximizes exercise-induced GH release and furthermore, provides a platform for future studies regarding GH and its efficacy in reducing hyperlipidemia. What synergistic effect would the Sprint 8 and statin medications provide? What about the Sprint 8, a statin, and a diet for severe patients? There are numerous questions to be answered. Building from this trial, as well as from future findings, an entire better way of life can be found for so many, categorized as obese or not.

Note: Mississippi continues to rank as the most obese state in the U.S., and, in order to reduce the physical and financial burden on individuals, as well as the state's health care system, an effective and economical intervention for this problem must be identified. KDMC has taken the initiative to find the solution.

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DECLARATION OF CONFLICTING INTERESTS

The authors declare that there is no conflict of interest.

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TABLES AND GRAPHS

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Graph 1. Results of the Sprint 8 showing the increase of GH serum levels (ng/mL) Pre-Sprint 8 and Post-Sprint 8 (771%).

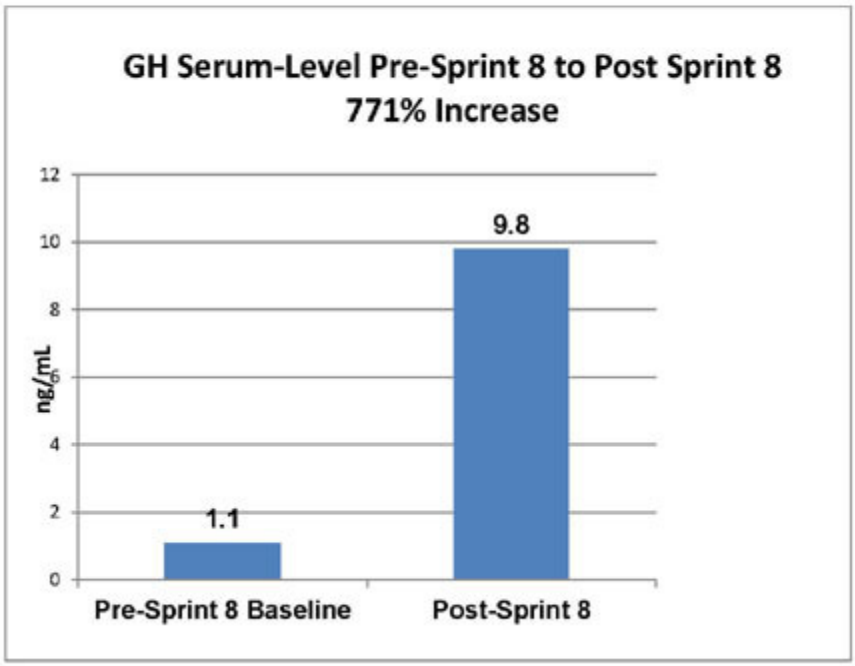


Table 1. Collected Sprint 8 data showing the increase or decrease in GH serum levels (ng/mL and % gain or loss) after initial exercise, pre-Sprint 8, and final exercise, post-Sprint 8.

Subject	Pre Sprint 8 Baseline GH (ng/mL)	Post Initial Exercise (ng/mL)	%loss or gain	Post Sprint 8 Baseline GH (ng/mL)	Post Final Exercise (ng/mL)	%loss or gain
A	0.1	0.2	+100%	0.1	0.3	+200%
B	1.0	9.8	+880%	0.6	10.4	+1,633%
C	0.8	2.2	+175%	0.3	1.3	+333%
D	4.0	17.3	+333%	3.0	19.6	+553%
E	2.5	50.8	+1,932%	2.8	13.5	+382%
F	0.6	3.1	+417%	2.0	6.1	+205%
G	2.5	11.1	+344%	0.1	6.4	+6,300%
H	0.1	2.1	+2,000%	0.1	1.0	+900%
I	0.1	1.3	+1,200%	0.1	0.9	+800%
J	0.1	9.4	+9,300	0.2	4.8	+2,300%
K	0.5	0.1	-80%	1.7	0.2	-88%
Mean	**1.1 ±1.3**	**9.8 ±14.7**	**+771%**	**1.0 ±1.2**	**5.9 ±6.3**	**+486%**

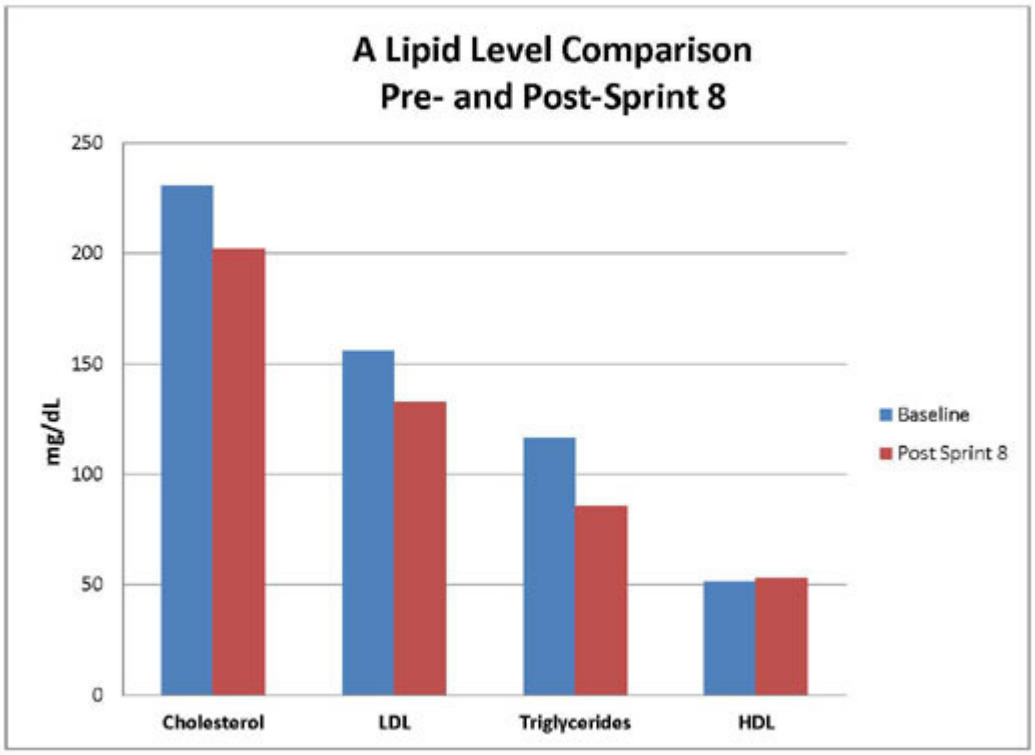
Table 2: Collected Sprint 8 data showing body weight, body fat, and BMI results and associated percent losses or gains.

Subject	Pre Sprint 8 Baseline Wt. (lbs.)	Post Sprint 8 Wt. loss (lbs.)	Pre Sprint 8 Baseline Body Fat %	Post Sprint 8 Body Fat %	Body Fat % loss	Pre Sprint 8 Baseline BMI (kg/m^2)	Post Sprint 8 BMI (kg/m^2)	BMI % loss
A	232	-28	34.8	19.9	45%	33.4	29.6	-11%
B	174	-8	33.3	22.6	32%	26.5	25.3	-4.5%
C	187	-12	37.6	25.9	31%	30.6	28.7	-6.2%
D	144	0	25.6	25.2	2%	25.5	25.5	0%
E	162	-15	n/a	n/a	n/a	26.2	23.8	-9.1%
F	252	-14	44.4	32.2	27%	40.7	38.5	-5.4%
G	213	-10	41.9	30.0	28%	37.1	36.0	-2.9%
H	155	-7	40.1	26.1	54%	28.3	27.1	-4.2%
I	201	-2	26.0	17.3	33%	29.7	29.4	-1.0%
J	146	-7	31.8	18.5	42%	23.6	22.4	-5.0%
K	223	-3	n/a	n/a	n/a	38.4	37.8	-1.5%
Mean	**189.9 ± 37.0**	**-9.6 ±7.8**	**35.1 ± 6.6%**	**24.2 ± 5.1%**	**-31.0%**	**30.9 ± 5.7**	**29.5 ± 5.6**	**-4.5%**

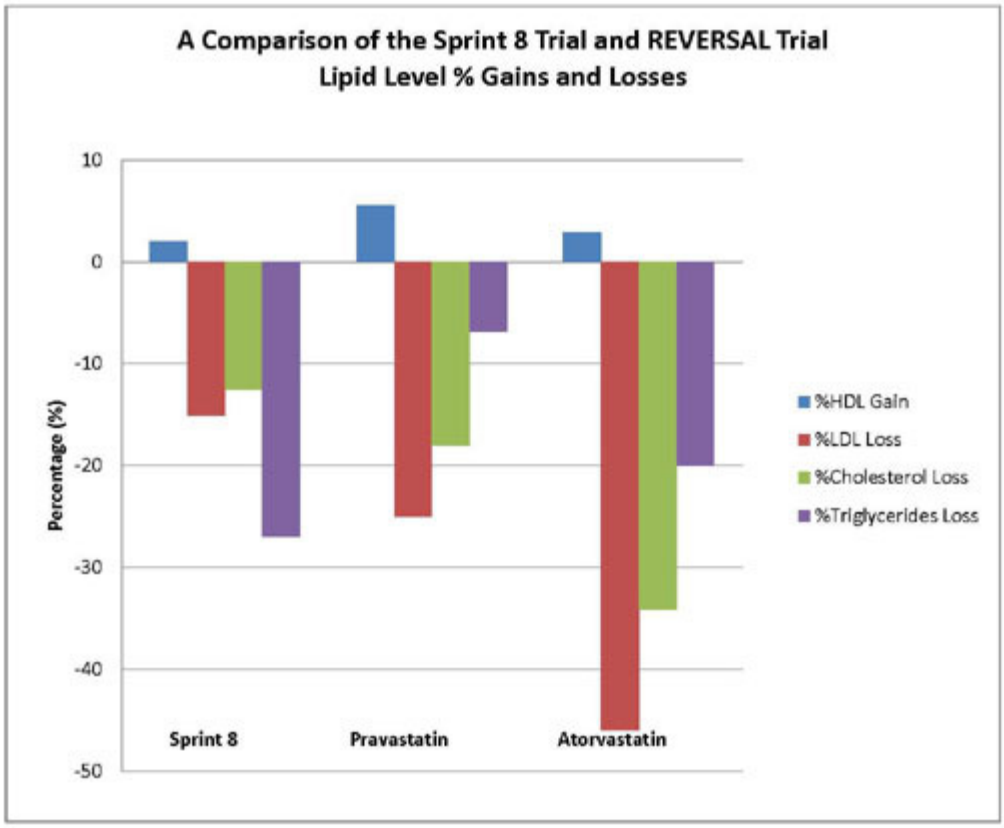
Table 3. Collected Sprint 8 data showing the increase or decrease in lipid levels (cholesterol, LDL, HDL, and triglycerides), comparing pre-Sprint 8 and post-Sprint 8 results.

Subject	Pre Sprint 8 Baseline Chol. (mg/dL)	Chol. pts. loss or gain/ % loss or gain	Pre Sprint 8 Baseline Trig. (mg/dL)	Trig. pts. loss or gain/ % loss or gain	Pre Sprint 8 Baseline LDL (mg/dL)	LDL pts. loss or gain/ % loss or gain	Pre Sprint 8 Baseline HDL (mg/dL)	HDL pts. loss or gain/ % loss or gain
A	197	-47/-24%	94	-26/-28%	131	-38/-29%	47	-4/-8.5%
B	212	-43/-20%	90	+2/+2.2%	144	-39/-27%	50	-4/-8%
C	226	-48/-21%	47	+21/+31%	165	-46/-28%	52	-7/-14%
D	247	+4/+1.6%	163	-57/-35%	155	+6/+4%	59	+10/+17%
E	308	-63/-21%	74	-32/-43%	230	-51/-22%	63	-7/-11%
F	181	-12/-6.6%	130	-38/-29%	102	-9/9%	53	+5/+9%
G	222	-34/-15%	91	-17/-19%	159	-32/-20%	45	+1/+2%
H	260	-14/-5.3%	257	-127/-49%	176	+4/+2%	33	+7/+21%
I	246	-8/-3.2%	94	+29/+31%	181	-14/-8%	46	0/0%
J	223	-30/-13.5%	93	-44/-47%	152	-24/-16%	52	+3/+6%
K	217	-17/-7.8%	152	-56/-37%	121	-13/-11%	66	+7/+11%
Mean	**230.8 ± 34.1**	**-28.4/ -12.3%**	**116.8 ± 57.3**	**-31.4/ -26.8%**	**156.0 ± 33.9**	**-23.3/ -15.0%**	**51.5 ± 9.2**	**+1.0/ +2.0%**

Graph 2. Results of the Sprint 8 trial, comparing points (mg/dL) of cholesterol, LDL, triglyceride, and HDL pre-Sprint 8 (baseline) and post-Sprint 8.



Graph 3. A comparison of the Sprint 8 trial and the REVERSAL trial (atorvastatin and pravastatin) (Nissen, 2004) in percent loss of LDL, cholesterol, and triglycerides, and percent gain of HDL.



Graph 4: A baseline comparison (mg/dL) of lipid serum levels (cholesterol, LDL, triglyceride, and HDL) pre-Sprint 8 and pre-REVERSAL trial (atorvastatin and pravastatin) (Nissen, 2004).