



Presents

LifesBody™

A New Paradigm in Weight Management

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Introduction

LifesBody™ is a safe and natural complex of plant-based polysaccharides, esterified fatty acids, polyphenols, beta-carotene and phycocyanamines™ that has been shown to reduce stored body fat and enhance weight loss. The improvement in body composition and percent of body fat is achieved through the interaction between the adipose tissue, brain and liver to control serum leptin.

LifesBody™ is a unique, proprietary blend that elicits a time-dependent transit through the gastro-intestinal tract. It is this time-dependent transit which elicits the signal to regulate leptin levels. Leptin serves as a regulator of body fat storage by modulating satiation, glycemic control and metabolism. Patented research from the University of Minnesota shows that reductions in serum leptin correlate with lower regional body fat and total body fat. LifesBody™ matches up to a US Patent in which the invention provides a method and composition for reducing the level of leptin in the bloodstream with a corresponding reduction in the percentage of body fat. The patent also claims sufficient reductions in the level of serum leptin in the bloodstream to beneficial or efficacious levels in approximately three to six weeks.

LifesBody™ reduces leptin and dampens inflammation. Inflammation is considered by leading scientists to be responsible for a number of metabolic problems which result in unwanted weight gain and obesity. Low-grade, internal, invisible inflammation along with high leptin levels is at the very basis of excess body fat and the inability to lose excess or unwanted fat.

LifesBody™ also enhances cell membrane stability for improved cellular communication between the liver and adipose tissues for enhanced fatty acid utilization leading to regional fat loss. In addition, modulation of membrane-based inflammatory markers helps to reduce the inflammatory component of fat gain. It is here that LifesBody™ differs from other leptin reducing supplements. Ingredients in the LifesBody™ proprietary blend, not only lower serum leptin levels, but have also been shown to reduce C-Reactive Protein (CRP), a key marker for low-grade internal inflammation. A recent key discovery by the University of Pittsburg School of Medicine shows that elevated levels of CRP inhibit leptin's role in controlling appetite.

Leptin is secreted by fat – the more fat, the more leptin – yet it is named for the Greek word leptos, which means "thin." In a region of the brain called the hypothalamus, leptin binds to receptors residing on the surface of neurons, setting off signals that tell the brain to stop eating and the body to expend energy by burning calories. While obese people produce much higher levels of leptin than thin and normal-weight individuals, they are somehow resistant to its effects. Dr. Zhao and his colleagues believe the binding of CRP to leptin may be the reason this is so. Their argument seems all the more plausible since CRP also is elevated in obese people. CRP, which is produced by the liver and typically rises as part of the immune system's inflammatory response, is gaining favor as a marker for hypertension and heart disease risk, known complications of obesity.

Dr. Zhao's recent publication in Nature Medicine (April 2006) demonstrates that CRP binds to leptin, and this impairs its signaling twofold. First, the coupling of CRP to leptin makes crossing the blood-brain barrier nearly impossible, thereby not allowing "free" leptin access to the hypothalamus. Second the CRP/leptin complex does not allow for binding of leptin to leptin receptors, because leptin is no longer in its "free" form.

In one set of studies, the researchers delivered human leptin continuously for six days into mice with receptors for leptin but without the ability to produce it. As expected, the plump mice ate less and lost weight, and their blood glucose levels normalized. Infusions containing both leptin and high doses of CRP blocked the action of leptin. The mice continued feasting, getting even fatter, and were no longer protected against diabetes. Giving CRP alone affected neither food intake nor body weight.

In a different experiment, the researchers found that when exposed to leptin, human liver cells increased their expression of CRP, suggesting that appetite may be regulated through a feedback loop that includes the liver in addition to the brain and fat cells that secrete leptin.

LifesBody™ has not only the ability to lower serum leptin levels by slowing down time-dependent transit through the gastro-intestinal tract, but also lowers CRP levels. This allows the remaining circulating leptin to reside in its "free" state and thus able to bind to appetite regulating leptin receptors in the hypothalamus.

Other benefits of the LifesBody™ weight loss formula are the ability to curb appetite and address emotional eating. Serotonin (5-HT), a vital neurotransmitter, is involved in a wide range of behavioral functions in the body, including mood, sleep and appetite control. Studies show that serotonin affects eating behavior and body weight. Increased plasma levels of serotonin are associated with decreased food intake, reduced weight gain and increased energy expenditure. Another benefit of increasing serotonin levels may be in addressing many of the emotional issues overweight people face, including binge eating and depression. Recently, it was discovered that phenylethylamine (PEA), an endogenous neuromodulating compound known to increase natural serotonin concentration, elevates mood and alleviates depression. PEA is the compound found in dark chocolate that provides the feeling of well-being or euphoria and has caused companies such as Mars Corporation and Nestle to spend tens of millions of dollars on its effects on the brain.

LifesBody™ Formula and Ingredient Rationale

LifesBody™ is a proprietary blend of plant polysaccharides, esterified fatty acids, pomegranate polyphenols, beta carotene, Klamath blue green algae, and phenylethylamine (from algae).

Rationale

Ingredient	Mechanism	Effect	Claims
Loleptin™ (plant – polysaccharides and esterified fatty acids)	Lowers Leptin levels	Curbs Appetite.	Helps reduce appetite. Weight loss.
Pomegranate extract	Lowers CRP	Promotes Leptin's effectiveness. Promotes Heart Health. Reduces Inflammation.	Helps reduce appetite. Lowers cholesterol. Lowers inflammation. Lowers cortisol.
Beta-carotene	Lowers CRP	Promotes Leptin's effectiveness. Promotes Heart Health. Reduces Inflammation.	Helps reduce appetite. Lowers cholesterol. Lowers inflammation Lowers cortisol.
Phycocyanamines™ (Blue Green Algae high PEA blend)	Increases Serotonin and Dopamine	Elevates mood.	Creates the euphoria of exercise "without exercising". The new exercise in a bottle!

Double Blind Placebo Controlled Clinical Trial

Methods

90 obese participants (38.8% male, 61.2% female), aged 21–44 (mean age = 29.3), and mean BMI > 30 kg/m² were recruited for this 8-week study. A physician will examine participants to ascertain their inclusion into the study. Metabolic syndrome will be diagnosed using the American Heart Association criteria for the disease, confirmed by the clinical investigator. The criteria include:

- Abdominal obesity (excessive fat tissue in and around the abdomen)
- Atherogenic dyslipidemia (blood fat disorders — high triglycerides, low HDL cholesterol and high LDL cholesterol — that foster plaque buildups in artery walls)
- Elevated blood pressure
- Insulin resistance or glucose intolerance (the body can't properly use insulin or blood sugar)
- Prothrombotic state (e.g., high fibrinogen or plasminogen activator inhibitor-1 in the blood)
- Proinflammatory state (e.g., elevated C-reactive protein in the blood)

After physical examination and laboratory screening tests, the morbidly obese, diabetics as well as pregnant and lactating women were excluded. None of the participants took any weight reducing medication nor followed any specific diet during the duration of the study.

Subject Selection

Inclusion Criteria:

Must meet the following criteria:

- BMI >30 kg/m²
- Total Cholesterol of >200 mg/dl
- LDL Cholesterol of >160 mg/dl
- HDL Cholesterol of <40
- Triglycerides >150 mg/dl
- Fasting blood glucose >100 mg/dl
- Blood pressure > 130/85 mm Hg
- Male or Female. Age 25 – 60
- Must give written informed consent.

Exclusion Criteria:

- Morbid Obesity BMI > 40 kg/m²
- Patients taking any cholesterol lowering medications 30 days prior to the start of enrollment and during the course of the study.
- Patients who have been enrolled in another clinical study in the past 6 months.
- Additional exclusion criteria are pregnancy, active infection, medication that interferes with healing (for example, steroids), systemic disease such as AIDS, HIV, or active hepatitis, and active malignancy (clinical signs within the past 5 years), Diabetes mellitus requiring daily insulin management;.

The purpose, nature and potential risks of the study were explained to all patients and all gave a written informed consent before participation. The Cameroon National Ethics Committee approved the protocol. The study was conducted in accordance with the Helsinki Declaration (1983 version).

Study design: The study was a randomized, double-blind, placebo-controlled design. Participants were randomly divided into three groups (30 participants/group): Placebo, LifesBody™ formula A (low dose) and LifesBody™ formula B (high dose). The placebo or active formulations were administered twice daily before meals with 8–10 oz. of water. Since the capsules were identical in shape, color and appearance, neither the participants nor the researchers knew which capsule was administered.

	Participant Characteristic	Treatment	Number of Participants
Group A	Metabolic syndrome	LifesBody™ low dose – 1 capsule (300mg each) twice daily	30
Group B	Metabolic syndrome	LifesBody™ high dose – 2 capsules (300mg each) twice daily.	30
Group C	Metabolic syndrome	Placebo	30

During the study period, subjects were examined bi-weekly, and their body weight, body fat, and waist/hip circumference were recorded and serological analysis performed. Subjective impressions (e.g., increased/decreased appetite, feelings of lightness, gastrointestinal pains, etc.) were solicited and recorded during each visit. The subjects were also asked about their physical activity and food intake, although no major dietary changes or exercises were suggested.

Anthropometric measurements: Body weight, body fat, and waist circumference were assessed at each visit with a Tanita™ BC-418 Segmental Body Composition Analyzer/Scale that uses bio-electrical impedance analysis for body composition analysis. Height was measured with a Harpenden™ stadiometer, which measures the length of curved line staffage to the nearest 0.5 cm. Participants (12 hour fasted) were encouraged to wear light clothing before measurements were taken. The waist circumference was measured by soft non-stretchable plastic tape on the narrowest and the widest parts of the trunk.

Serological/Laboratory methods: Blood samples were collected after a 12-hour overnight fast into heparinized tubes at the beginning of the study and after 2, 4, 6, and 8 weeks of treatment. The concentrations of total cholesterol, LDL cholesterol, fasting blood glucose, serotonin, C-reactive protein and leptin were measured using commercial diagnostic kits from SIGMA Diagnostics.

Statistical Analysis: The data for each parameter was summarized via n, mean, and standard deviation for Week 0 (Initial), Week 8 (final) and for the intra-group percent differences. The percent change from baseline was tested for differences using the Mixed Effects Model, which is a flexible tool for analyzing longitudinal and repeated treatments.

Results

Table 1. Body weight: effectiveness of treatments

	Body Weight (mean kg)		Weight change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	100.93±6.30	94.36±5.67 ^{b*}	-6.95 [‡]
Formula A (↓dose)	102.28±7.17	97.04±5.95 ^b	-5.40 [†]
Placebo	101.32±6.13	100.50±7.28	-0.82

^ap<0.05; ^bp<0.001; ^cp<0.0001 compared with **Placebo**

^{*}p<0.05; [†]p<0.001; [‡]p<0.0001 compared with **Formula A**

[†]p<0.05; [‡]p<0.001 compared with **Initial**; intragroup analysis

Table 2. Body fat: effectiveness of treatments

	Body fat (mean %)		Fat reduction (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	43.85±5.60	42.04±6.63 ^a	-4.13 [‡]
Formula A (↓dose)	40.22±7.26	39.08±6.12 ^a	-2.83 [†]
Placebo	42.28±8.27	42.18±8.117	-0.24

^ap<0.05; ^bp<0.001; ^cp<0.0001 compared with **Placebo**

^{*}p<0.05; [†]p<0.001; [‡]p<0.0001 compared with **Formula A**

[†]p<0.05; [‡]p<0.001 compared with **Initial**; intragroup analysis

Table 3. Waist size: effectiveness of treatments

	Waist circumference (mean cm)		Waist change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	110.50±7.28	105.38±10.64 ^a	-4.64 [‡]
Formula A (↓dose)	104.00±8.63	99.40±11.73 ^a	-4.42 [†]
Placebo	105.60±7.91	105.00±15.77	-0.57

^ap<0.05; ^bp<0.001; ^cp<0.0001 compared with **Placebo**

^{*}p<0.05; [†]p<0.001; [‡]p<0.0001 compared with **Formula A**

[†]p<0.05; [‡]p<0.001 compared with **Initial**; intragroup analysis

Table 4. Hip size: effectiveness of treatments

	Hip circumference (mean cm)		Waist change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	123.38±10.84	117.75±17.24 ^a	-4.56 [‡]
Formula A (↓dose)	126.20±12.91	121.80±15.41 ^a	-3.49 [†]
Placebo	125.85±14.72	125.45±17.99	-0.32

^ap<0.05; ^bp<0.001; ^cp<0.0001 compared with **Placebo**

^{*}p<0.05; [†]p<0.001; [‡]p<0.0001 compared with **Formula A**

[†]p<0.05; [‡]p<0.001 compared with **Initial**; intragroup analysis

Table 5a. Plasma Total Cholesterol level: effectiveness of treatments

	Total Cholesterol (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	210.64±9.30	152.75±10.19 ^b	-27.48 [‡]
Formula A (↓dose)	208.74±10.49	171.17±18.82 ^a	-18.00 [†]
Placebo	211.16±11.92	206.68±20.19	-2.12

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

[†] p<0.05; [‡] p<0.001 compared with **Initial**; intragroup analysis

Table 5b. Plasma LDL Cholesterol level: effectiveness of treatments

	LDL Cholesterol (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	165.84±7.29	133.05±7.71 ^b	-19.77 [‡]
Formula A (↓dose)	163.94±8.27	139.91±8.92 ^a	-14.66 [†]
Placebo	166.21±9.25	162.63±17.83	-2.39

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

[†] p<0.05; [‡] p<0.001 compared with **Initial**; intragroup analysis

Table 5c. Plasma HDL Cholesterol level: effectiveness of treatments

	HDL Cholesterol (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	32.43±2.10	38.63±3.11 ^b	19.12 [‡]
Formula A (↓dose)	34.72±3.28	39.46±4.48 ^a	13.65 [†]
Placebo	35.92±5.16	37.22±5.59	3.62

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

[†] p<0.05; [‡] p<0.001 compared with **Initial**; intragroup analysis

Table 6. Plasma Triglyceride level: effectiveness of treatments

	Triglyceride (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	167.61±10.84	140.04±4.29 ^{b*}	-16.45 [‡]
Formula A (↓dose)	162.21±12.95	150.71±8.30 ^a	-7.09 [†]
Placebo	168.93±14.04	167.90±10.30	-0.61

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

[†] p<0.05; [‡] p<0.001 compared with **Initial**; intragroup analysis

Table 7. Fasting Blood Glucose levels: effectiveness of treatments

	Blood Glucose (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	104.84±9.39	92.06±6.29 ^{b*}	-12.19 [‡]
Formula A (↓dose)	105.61±8.62	97.22±7.81 ^a	-7.94 [†]
Placebo	107.52±8.73	105.90±10.79	-1.51

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

† p<0.05; ‡ p<0.001 compared with **Initial**; intragroup analysis

Table 8. Serum Serotonin levels: effectiveness of treatments

	Serotonin (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	35.40±2.62	49.06±2.85 ^{b*}	38.59 [‡]
Formula A (↓dose)	34.55±2.85	44.42±2.96 ^a	28.57 [‡]
Placebo	34.20±3.15	37.31±3.69	9.09

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

† p<0.05; ‡ p<0.001 compared with **Initial**; intragroup analysis

Table 9. Serum Leptin levels: effectiveness of treatments

	Leptin (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	30.15±1.50	15.83±1.13 ^b	-47.59 [‡]
Formula A (↓dose)	31.52±1.21	16.72±1.40 ^a	-46.95 [‡]
Placebo	30.33±1.63	25.97±1.61	-14.38

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

† p<0.05; ‡ p<0.001 compared with **Initial**; intragroup analysis

Table 10. C-Reactive Protein levels: effectiveness of treatments

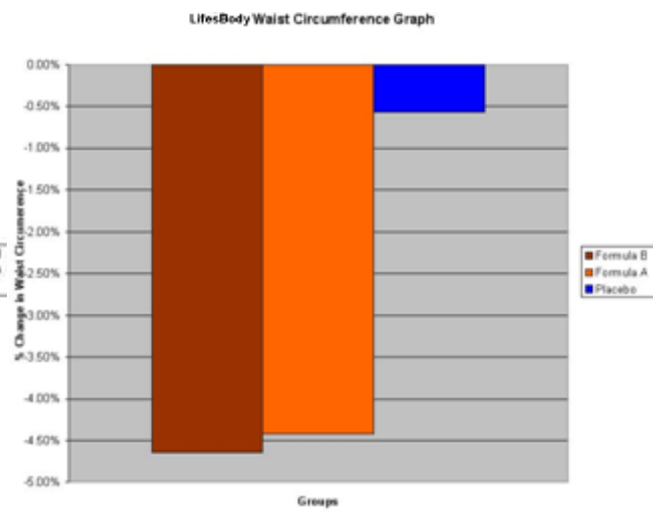
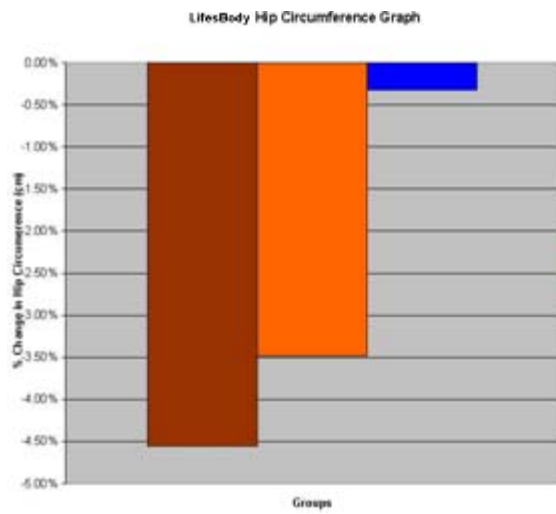
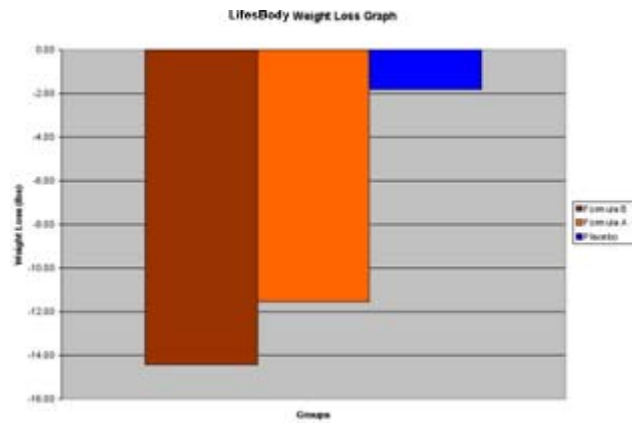
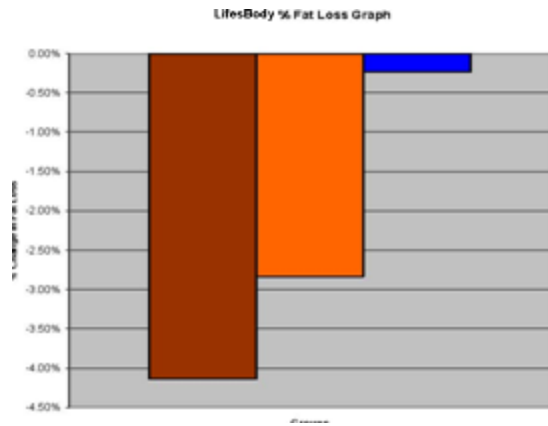
	CRP (mean mg/dl)		change (%)
	Initial	8 Weeks	8 Weeks - Initial
Formula B (↑dose)	11.58±1.11	8.17±0.61 ^{b*}	-29.45 [‡]
Formula A (↓dose)	10.99±1.26	9.33±0.43 ^a	-15.10 [‡]
Placebo	12.46±1.30	12.05±1.39	-3.31

^a p<0.05; ^b p<0.001; ^c p<0.0001 compared with **Placebo**

* p<0.05; ** p<0.001; *** p<0.0001 compared with **Formula A**

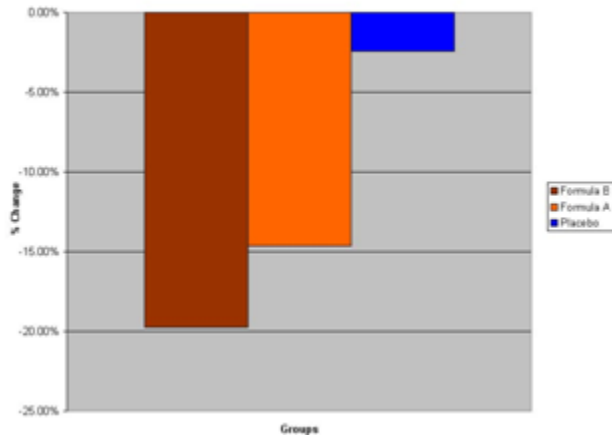
† p<0.05; ‡ p<0.001 compared with **Initial**; intragroup analysis

LifesBody™ Anthropomorphic Graphs

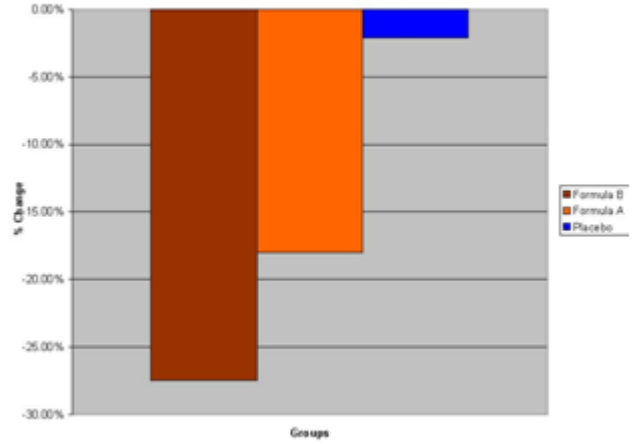


LifesBody™ Cholesterol Graphs

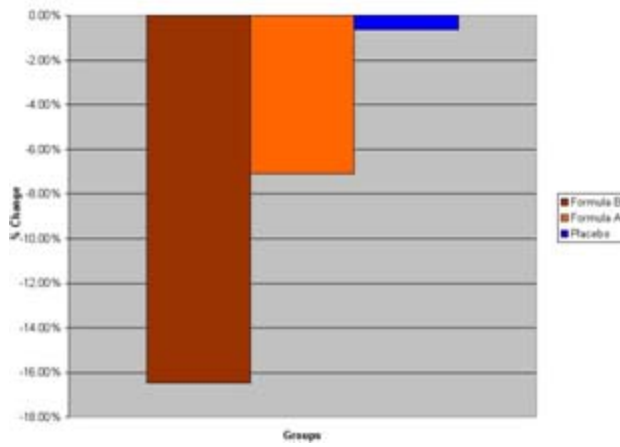
LifesBody LDL Cholesterol Graph



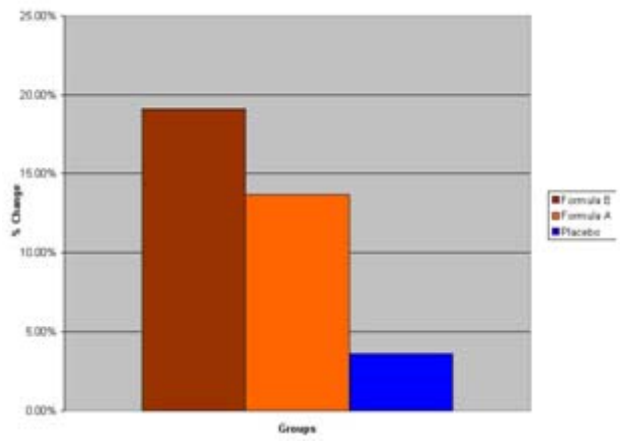
LifesBody Total Cholesterol Graph



LifesBody Triglycerides Graph

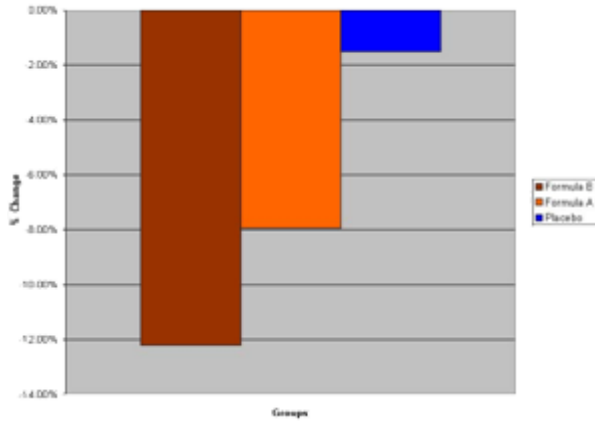


LifesBody Cholesterol Graph

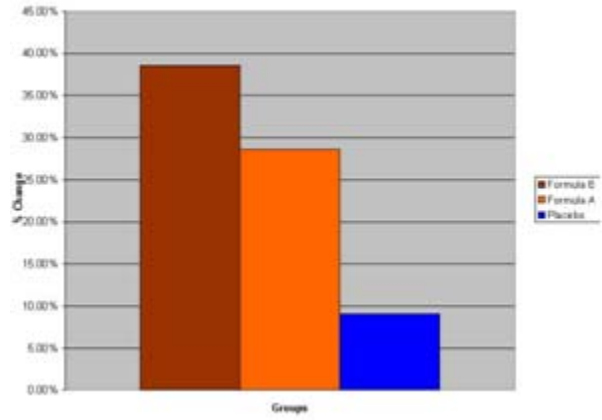


LifesBody™ Other Serological Graphs

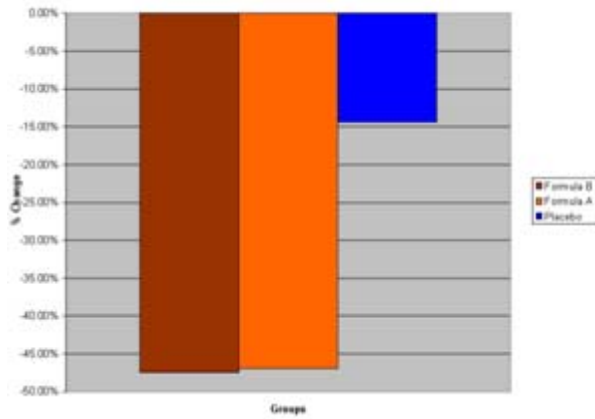
LifesBody Fasting Blood Sugar Graph



LifesBody Serotonin Graph



LifesBody Serum Leptin Graph



LifesBody C-Reactive Protein Graph

