Novel Dietary Supplement Shows Dramatic Effects in Lowering Cholesterol, LDL, and Triglycerides

By Jim English

According to the federal Centers for Disease Control and Prevention, 61 million Americans currently suffer from cardiovascular disease. Cardiovascular disease covers a broad spectrum of disorders, including high blood pressure, coronary heart disease (heart attack and chest pain), stroke, congestive heart failure, and birth defects of the heart and blood vessels.

Every year, heart attacks and stroke cause more than 930,000 deaths in the US, accounting for 40% of deaths from all causes and making cardiovascular disease the nation's number-one killer. While cardiovascular disease primarily kills people aged 65 and older, the incidence of sudden death from heart disease is rising in people aged 15 to 34.1

Reducing serum cholesterol levels—especially low-density lipoprotein (LDL)—is an effective, well-established strategy for preventing cardiovascular disease and reducing coronary events and mortality.2,3 Unfortunately, a recent report in the journal Circulation found that between 1988 and 2000, average total serum cholesterol concentrations in the US population declined by only 1%.4 And while 91% of those surveyed by the American Heart Association (AHA) felt it was "important to them personally to have a healthy cholesterol level," fewer than 50% knew their own cholesterol levels, and 53% either did not know or overestimated the recommended cholesterol levels for a healthy adult.5

Compounding the problem, only a fraction of those at risk for cardiovascular disease are using pharmaceutical and nutritional strategies known to reduce cholesterol levels. According to estimates based on data gathered from the National Health and Nutrition Examination Survey III (NHANES III), only 6.6% of the 21.1 million Americans eligible for cholesterol-lowering drug therapy under National Cholesterol Education Program (NCEP) guidelines were using such therapy.6 When researchers examined responses gathered from 13,990 patients, they discovered that fewer than 4% of those diagnosed with hypercholesterolemia (elevated cholesterol) were taking vitamins or supplements known to reduce cholesterol.7

Concerned with the persistent failure of conventional strategies to significantly improve cholesterol profiles and reduce the incidence of cardiovascular disease, a broad coalition of medical researchers and scientists is now calling for a massive increase in the use of cholesterol-lowering drugs, particularly the family of pharmaceuticals known as statins.8

Unfortunately, the statin drugs, while very effective, also have side effects that understandably compromise patient compliance. Additionally, statin drugs are expensive to use; depending on the drug and dosage, the cost of statin therapy ranges from $63 to $228 a month.9

A newly available, all-natural supplement has been shown in human studies to significantly lower cholesterol levels—particularly of LDL, triglycerides, and apolipoprotein B—thus helping to reduce the risk of developing cardiovascular disease. This supplement, called Sytrinol™, is an important option for health-conscious people seeking a safe, effective, and convenient way to lower cholesterol levels without the side effects and expense of drugs. > > >
Cholesterol and Human Health

Cholesterol is a fatty (lipid) component found in virtually all cell membranes. In addition to supporting cellular integrity, cholesterol is also required for the transport of phospholipids and the biosynthesis of mineralocorticoids (aldosterone), glucocorticoids (cortisol), and sex hormones (progesterone, pregnenolone, testosterone, and estradiol). Far from endangering health, cholesterol is essential to life. In fact, Italian researchers have shown that when serum cholesterol levels are too low (less than 160 mg/dL), mortality in older adults actually increases.1

LDL, popularly known as “bad cholesterol,” is the primary transporter of cholesterol in the blood. In atherosclerosis, LDL is taken up in lesions in endothelial cells lining the inner walls of blood vessels, forming deposits in the arterial walls. The deposited LDL undergoes modification, as free radicals oxidize LDL to form foam cells that create a thick, hard plaque.

Over time, plaque accumulation can constrict vessels, inhibiting blood flow and reducing the supply of oxygen reaching the heart, brain, and other organs.2 If a clot (thrombus) blocks an artery already restricted by plaque, blood and oxygen flow can be cut off entirely, leading to a heart attack (if the occlusion occurs in the heart) or a stroke (if it occurs in the brain).

HDL is commonly referred to as “good” cholesterol because it helps remove excess cholesterol from atherosclerotic deposits and retard the growth of new plaque. Low HDL levels have been shown to be an additional risk factor for increased mortality from coronary artery disease and strokes in the elderly.3

How the Body Manages Cholesterol Levels

While cholesterol levels can be modestly influenced by dietary modification about 80% of cholesterol does not come from dietary sources, but is synthesized by the liver.4 The rate-limiting enzyme HMG-CoA (3-hydroxy-3-methylglutaryl coenzyme A) reductase controls the biosynthesis of cholesterol.

Normally, the liver regulates cholesterol levels via a biochemical feedback loop. When cholesterol levels are low, liver production of HMG-CoA reductase increases to speed biosynthesis of cholesterol. Conversely, when cholesterol levels are too high, the liver limits HMG-CoA reductase production to reduce cholesterol production. Proper functioning of this feedback mechanism is vital for the maintenance of healthy cholesterol levels. Unfortunately, modern dietary habits (such as excess intake of saturated and trans fatty acids) and lifestyle contribute to the disruption of this system, leading to elevated cholesterol levels and increased risks for developing cardiovascular disease.

Additionally, certain genetic disorders, such as familial hypercholesterolemia and autosomal recessive hypercholesterolemia, are known to increase LDL levels and risk for developing cardiovascular disease.

Not All LDL Is Created Equal

To bind with other molecules for transport through the circulatory system, lipids rely on a specialized class of structural proteins, called apoproteins. LDL exists in two versions, differentiated by their protein components. The first, apolipoprotein A, consists of a large, “fluffy” protein called apoprotein A that is cardioprotective when bound to LDL. The second, apolipoprotein B, consists of a small, dense protein called apoprotein B that plays a major role in cardiovascular disease when bound to LDL. Apolipoprotein-B particles enable cholesterol to penetrate and lodge in vascular walls, an important step in initiating the formation of atherosclerotic plaque.5 Apolipoprotein B is the predominant form of apolipoprotein, and over 90% of all LDL cholesterol particles in the blood carry apolipoprotein B, making it an especially accurate (and convenient) marker for measuring the cholesterol-depositing capacity of blood.6

The importance of apolipoprotein B was highlighted in a report published in 2001 in the British medical journal The Lancet. In the AMORIS study, researchers evaluated cardiovascular markers in over 175,000 men and women over a period of five and a half years. In addition to conventional lipid markers, such as triglycerides, total cholesterol, and LDL:HDL ratios, the researchers also measured apolipoprotein-B levels. Their
findings revealed that those with the highest ratios of apolipoprotein B to apolipoprotein A were at the greatest risk of dying from a heart attack. These findings were supported by a second study, published in 2003 in the journal Circulation. In the IRAS study, researchers again measured apolipoprotein-B levels in 1,522 individuals and compared them with an array of standard lipid markers (such as C-reactive protein, fibrinogen, and carotid artery intima-media thickness) to assess cardiovascular disease risks. They found that elevated apolipoprotein-B levels were strongly associated with cardiovascular disease, and concluded that apolipoprotein-B levels are a better predictor of vascular risk than are LDL levels.

Given the well-documented link between apolipoprotein B and cardiovascular disease, measuring apolipoprotein-B levels offers clinicians and patients a new, highly specific marker for assessing the precise level of LDL in serum and determining individual risk for developing cardiovascular disease.

Statin Drugs: the New Aspirin?

Due to the failure of previous public health programs to substantially lower cholesterol levels in the general population, medical researchers and health experts are seeking a new approach to better manage the problem. For the last decade, physicians and patients have relied on cholesterol guidelines published by the AHA. According to the AHA, a total cholesterol level of 200 mg/dL or less is considered optimal. Levels of 200-239 mg/dL are considered borderline high risk, and levels above 240 mg/dL are considered high risk.

In May 2001, the National Institutes of Health published new federal guidelines calling for aggressive expansion of the use of statin drugs to treat cholesterol. Statin drugs such as atorvastatin (Lipitor®), lovastatin (Mevacor®), pravastatin (Pravachol®), and simvastatin (Zocor®) are among the most potent lipid-lowering agents currently available.

Statin lower cholesterol levels by inhibiting the production of HMG-CoA reductase, resulting in a decrease in cholesterol synthesis in the liver. To compensate for the resulting reduction of cholesterol production, the liver begins to remove LDL circulating in the blood, further reducing overall LDL levels. Statin therapy has been proven to contribute to a decrease in cardiovascular disease morbidity and mortality in recent years, as documented in a number of controlled clinical trials. In addition to improvements in lipid profile, statins also appear to confer other benefits, including improved endothelial function, decreased platelet thrombus formation, improved fibrinolytic activity, and reduced frequency of transient myocardial ischemia.

Although statin therapy was initially used to treat patients suffering from severe hypercholesterolemia, health experts are now pushing to expand statin use to patients with only moderately elevated cholesterol. Moreover, health authorities have called for the use of statins to treat conditions such as diabetes, high blood pressure, high serum triglycerides, and low HDL, as well as for those with a strong family history of heart disease.

Most recently, in July 2004, the journal Circulation published an updated version of the NCEP guidelines, encouraging physicians to aggressively increase the use of statin drugs to lower cholesterol levels. In particular, the report recommends that target LDL levels be reduced from the current 100 mg/dL to 70 mg/dL in patients considered at high risk for a heart attack or death from cardiovascular disease. Additionally, patients at only moderate risk of a heart attack—those with heart disease, diabetes, or other risk factors—are now being encouraged to reduce their cholesterol levels by 30-40%.

Not surprisingly, the new guidelines could dramatically increase the number of patients on statin drugs to as many as 50 million. In an embarrassing oversight, the same government panel drafting the new guidelines failed to mention in...
its report that most of its panelists are linked to pharmaceutical companies that manufacture statin drugs. Six of the nine panelists had either received grants from or were paid consulting or speakers’ fees by the companies that make some of the most popular statins, including Pfizer’s Lipitor®, Bristol-Myers Squibb’s Pravachol®, Merck’s Mevacor®, and AstraZeneca’s Crestor®.

**Statins and Side Effects**

While statin drugs effectively lower LDL, they also produce serious side effects. In 1990, Folkers theorized that inhibition of HMG-CoA reductase would also inhibit intrinsic biosynthesis of coenzyme Q10 (CoQ10), a central compound in the mitochondrial respiratory chain. Dr. Folkers’ researchers stated, “If lovastatin were to reduce levels of CoQ10, this reduction would constitute a new risk of cardiac disease, since it is established that CoQ10 is indispensable for cardiac function.”

When the researchers examined five hospitalized patients aged 43 to 72, they found that lovastatin did in fact cause CoQ10 levels to drop. Furthermore, the patients showed evidence of increased cardiac distress, a potentially life-threatening situation for patients hospitalized with class IV cardiomyopathy. The researchers concluded, “Although a successful drug, lovastatin does have side effects, particularly including liver dysfunction, which presumably can be caused by the lovastatin-induced deficiency of CoQ10.”

Taking supplemental CoQ10 may potentially offset this side effect, but other, more serious side effects cannot be so easily resolved.

For example, rhabdomyolysis is a rare but potentially deadly condition that occurs when large numbers of skeletal muscle cells die. As the rapidly dying cells deteriorate, they release large quantities of muscle proteins into the bloodstream, quickly overwhelming the kidneys. An analysis of the Food and Drug Administration’s side-effect registry, conducted in 2001 by the consumer watchdog group Public Citizen, discovered that statin drugs were linked to 72 fatal and 772 non-fatal cases of rhabdomyolysis between October 1997 and December 2000. In August 2003 to mid-April 2004, patients using Crestor®, including 11 in the US, suffered severe muscle deterioration. In addition, eight cases of acute kidney failure and four cases of kidney insufficiency related to the use of Crestor® have been reported.

**Unknown Long-Term Effects**

While the cardioprotective benefits of statin drugs outweigh the known side effects, the most recent NCEP recommendations may result in tens of millions of new patients taking statins for a period of decades, and possibly for a lifetime. Unfortunately, data on the long-term use of statins are scant. In one paper published in the *Journal of the American Medical Association* in 1996, researchers set off a furious round of debate by raising the possibility of long-term
Healthy Options for Lowering Cholesterol

In their enthusiasm to reduce premature deaths from heart attack and strokes, the authors of the new cholesterol guidelines are recommending that millions of Americans start taking statin drugs. This recommendation ignores the danger of potential side effects from the long-term use of statins. Would informed health consumers willingly choose to lower their risk of cardiovascular disease if it meant substantially increasing their chances of developing health problems after a decade or two?

In a recent opinion piece published in the Washington Post, Dean Ornish, MD, clinical professor of medicine at the University of California, San Francisco, and president of the nonprofit Preventive Medicine Research Institute, wrote, "As tens of millions of people begin taking these medications for decades, more long-term side effects are likely to become apparent." Dr. Ornish also questioned why the panel failed to recommend other options, such as diet and lifestyle changes, that for most people "can be a safe and effective alternative to a lifetime of cholesterol-lowering drugs."30

One of the newest and most effective alternatives to statin drugs is a patented, proprietary formula comprising citrus and palm fruit extracts that contain polymethoxylated flavones and tocotrienols. It has been shown in human trials to significantly reduce total cholesterol, LDL, and triglycerides. Additionally, the powerful antioxidant and anti-inflammatory properties of the extracts in this natural formulation (trademarked under the name Sytrinol) are known to contribute to managing additional cardiovascular disease risk factors.

Tangerine Flavonoids Safer than Statins

Flavonoids are natural polyphenolic compounds found in a wide variety of fruits and vegetables. More than 4,000 different flavonoids have been identified, and many of these have been shown to exert biological effects in humans. Bioflavonoids from citrus fruits such as oranges, tangerines, and grapefruit have been found to reduce oxidative drug metabolism, inhibit chemical carcinogenesis and tumor development, and exert anti-inflammatory and anti-allergic effects. Additionally, epidemiological studies have shown that consumption of dietary flavonoids is strongly associated with reduced incidence of cardiovascular disease and cancer in humans.55

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Nobiletin, an orange peel extract, significantly inhibits production of two powerful free radicals involved in promoting cancer.

Many flavonoids, such as rutin and hesperidin, exert cardiovascular-protective effects by inhibiting oxidation of LDL, reducing inflammation, enhancing endothelial function, and reducing thrombosis.46-49 Recently, a subset of flavonoids known as polymethoxylated flavones have been shown to possess especially potent anti-cancer, immunosupportive, and cardioprotective benefits. Polymethoxylated flavones—flavonoid compounds derived from the peels of oranges, tangerines, and other citrus fruits—are highly methoxylated and contain biologically active molecules with unique metabolic properties. Two of the most-researched polymethoxylated flavones are nobiletin and tangeretin.

**Nobiletin** was first isolated from orange peels in 1938.50 Intrigued by the anti-cancer benefits associated with the consumption of citrus fruit, researchers first examined nobiletin as a potential chemopreventive compound.51 Early studies revealed that nobiletin significantly inhibits production of nitric oxide and superoxide, two powerful free radicals involved in promoting inflammation and cancer. In one study, nobiletin was shown to suppress several stages of skin inflammation required for tumor initiation and growth.52 Nobiletin has also been shown to inhibit the proliferation of human gastric cancer cells (metastases) in mice, leading the study authors to suggest that the compound may be a candidate anti-metastatic drug for prevention of peritoneal dissemination of gastric cancer.53

Nobiletin likewise has been shown to be a powerful anti-inflammatory agent. Atherosclerosis is now recognized to be an inflammatory process, partially explaining why half of all heart attacks occur in people with "normal" cholesterol levels. While popular non-steroidal anti-inflammatory drugs, such as Celebrex® and Vioxx®, reduce inflammation by blocking the enzyme cyclooxygenase-2 (COX-2), Vioxx® has recently come under scrutiny for possibly increasing the risk of cardiovascular events. By contrast, nobiletin has been found to selectively downregulate COX-2 without interfering with COX-1 mRNA expression.54 In mouse macrophages, nobiletin was also shown to suppress production of prostaglandin E2 while interfering with proinflammatory cytokines such as interleukin-1 alpha, interleukin-1 beta, tumor necrosis factor-alpha, and interleukin-6.55 These anti-inflammatory effects are comparable to those of powerful anti-inflammatory steroids such as dexamethasone.

In addition, nobiletin demonstrated greater anti-inflammatory activity than indomethacin in a tetradeacanoylphorbol acetate (TPA)-induced edema test in mouse ears, offering further support for this compound’s antioxidative, anti-inflammatory, and cancer-preventive benefits.56 Through its effects in reducing inflammation, nobiletin may help to protect cardiovascular health.

**Tangeretin** was first isolated from tangerine oil in 1934.57 Early research found that tangeretin has anti-cancer actions similar to those of other comparable polymethoxylated flavones such as nobiletin.58-60 Tangeretin was also found to exert antioxidant and neuroprotective benefits. In an animal study, tangeretin was found to cross the blood-brain barrier and to protect brain cells (hypothalamus, striatum, and hippocampus) in rats exposed to 6-hydroxydopamine. The drug 6-hydroxydopamine produces cytolytic free radicals that deplete noradrenergic stores in nerve endings and reduce dopamine levels in the brain, thus providing an animal model of Parkinson's disease.61

Recently, in-vitro studies have revealed that tangeretin lowers triglyceride and apolipoprotein-B levels.62 Researchers in Canada first observed that intracellular production of cholesterol and apolipoprotein B declined rapidly in human liver cells after being incubated with tangeretin. When they followed up on their initial findings, the researchers determined that tangeretin achieves these reductions by modulating several mechanisms involved in lipoprotein metabolism.

First, tangeretin was shown to interfere with cellular production of triglycerides, reducing levels up to 37% following treatment. Tangeretin was also found to reduce intracellular production of apolipoprotein B by inhibiting microsomal triglyceride transfer protein, a specialized lipid transfer protein with a key role in the assembly and secretion of lipoproteins containing apolipoprotein B. By limiting microsomal triglyceride
transfer protein, tangeretin reduces the number of apo-lipoprotein-B particles that can be synthesized in the liver.  

Additionally, the researchers discovered that tangeretin helps to limit apolipoprotein-B production by suppressing diacylglycerol acyltransferase, the final enzyme in the pathway of triglyceride synthesis. Triglycerides play an important role in the formation of apolipoprotein B. By limiting production of triglycerides, tangeretin effectively restricts production of apolipoprotein B.

**Tocotrienols: Natural Alternatives to Statins**

Tocotrienols are a third cardio-protective component of the proprietary Sytrinol™ formula. Tocotrienols are naturally occurring antioxidant analogs of tocopherols, a family of nutrients that includes natural vitamin E. Most risk markers for cardiovascular disease have a proinflammatory component that stimulates the release of active molecules in response to injury. These active molecules include inflammatory mediators, reactive oxygen species, nitric oxide, and peroxynitrite from endothelial, vascular smooth muscle, and immune cells. In addition to their antioxidant effects, tocotrienols have been shown to exert a powerful anti-inflammatory effect that can help to mitigate inflammatory processes that are known to initiate atherosclerosis.

Tocotrienols have also been found to lower total serum cholesterol and LDL levels by degrading the enzyme HMG-CoA reductase, which is responsible for producing cholesterol. By inhibiting the enzymatic actions of HMG-CoA reductase through a post-transcriptional mechanism, tocotrienols can suppress cholesterol synthesis without the harmful side effects observed with statin drugs.

Early research found that tocotrienols reduced cholesterol, LDL, and triglycerides, while raising HDL. An observed secondary benefit from tocotrienols was an increase in apolipoprotein-A levels, which counter the damaging effects of apolipoprotein B.

In animal studies, tocotrienols were shown to reduce total cholesterol by 30% and LDL by 67% compared to controls in rats with induced hypercholesterolemia. Tocotrienols were also shown to significantly reduce HMG-CoA activity.

In a 2002 human study, 90 subjects diagnosed with hypercholesterolemia were treated with a protocol that used tocotrienols in conjunction with the AHA Step 1 diet. The researchers reported that daily treatment with 100 milligrams (mg) of a tocotrienol-rich supplement decreased total cholesterol by 20%, LDL by 25%, apolipoprotein B by 14%, and triglycerides by 12%.

**Safety and Effects of Sytrinol™**

Sytrinol™ was developed after 12 years of extensive research on the cardiovascular effects of polymethoxylated flavonoids and tocotrienols. The health benefits of Sytrinol™ have been demonstrated in in-vitro, in-vivo, and human clinical studies. Animal toxicity studies have shown that Sytrinol™ is well tolerated, with no toxic effects following consumption of polymethoxylated flavones in amounts of up to 1% of total dietary intake, or the equivalent of a 150-pound individual consuming almost 14 grams per day.

The cholesterol-lowering effects of Sytrinol™ were documented in a recent animal study published in the May 2004 issue of the Journal of Agricultural and Food Chemistry. Canadian researchers first induced high blood levels of cholesterol in hamsters. The animals were then treated with either polymethoxylated flavonoids (tangeretin) or a combination of flavones (hesperidin and naringin). While the flavones were shown to lower cholesterol levels, the tangeretin formulation proved to be almost three times as effective. In hamsters receiving the tangeretin formula, total cholesterol declined by up to 27% and LDL was reduced by 40%. While HDL levels were unchanged, the net result was a significant improvement in the LDL:HDL ratio.

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The cardioprotective and cholesterol-lowering claims for Sytrinol™ are also supported by human studies. Two early trials, each using 10 subjects, measured the effects of Sytrinol™ in men and women diagnosed with hypercholesterolemia and screened to eliminate thyroid disorders, kidney disorders, and diabetes. Subjects were instructed to maintain normal dietary habits and discontinue using vitamins, supplements, and cholesterol-lowering medications for at least six weeks before and during the study. Fasting blood samples were drawn at the onset and at the end of each four-week trial, and plasma lipid profiles and other metabolic parameters were analyzed using standard methods.

The results from the first trial (Table 1) show that four weeks of treatment with 300 mg of Sytrinol™ daily significantly reduced levels of total cholesterol (-25%), LDL (-19%), and triglycerides (-24%). HDL levels were unchanged and body mass remained relatively stable.

In the second trial, subjects with elevated cholesterol again benefited after only four weeks of treatment with 300 mg per day of Sytrinol™. As shown in Table 2, treatment with Sytrinol™ substantially cut levels of plasma total cholesterol (-20%), LDL (-22%), triglycerides (-21%), and triglycerides (-28%). Additionally, subjects in the second trial benefited from a significant 5% increase in apolipoprotein A1, an important structural protein of HDL.

Sytrinol™ is currently being tested in a long-term, double-blind, crossover randomized study involving 120 men and women with moderately elevated cholesterol levels (total cholesterol above 230 mg/dL and LDL greater than 155 mg/dL). For 12 weeks, subjects will receive either 300 mg per day of Sytrinol™ or placebo, followed by a washout period of four weeks and another.

### Table 1: Sytrinol™ Clinical Study I Results

<table>
<thead>
<tr>
<th>Measured Endpoints</th>
<th>Treatment Group</th>
<th>Sytrinol™ Clinical Study I % Change at 4 weeks</th>
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<tbody>
<tr>
<td>Total Cholesterol</td>
<td>Sytrinol™</td>
<td>-25% b</td>
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<tr>
<td>LDL</td>
<td>Sytrinol™</td>
<td>-19% b</td>
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<td>Triglycerides</td>
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<tr>
<td>HDL</td>
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Means ± SEM. Statistical analysis by ANOVA plus post test by Dunnett’s method

b – Significantly different within same group, P ≤ 0.05

Source: SourceOne Global Partner

### Table 2: Sytrinol™ Clinical Study II Results

<table>
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<tr>
<th>Measured Endpoints</th>
<th>Treatment Group</th>
<th>Sytrinol™ Clinical Study II % Change at 4 weeks</th>
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<tr>
<td>Total Cholesterol</td>
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<td>LDL</td>
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Means ± SEM. Statistical analysis by ANOVA plus post test by Dunnett’s method

b – Significantly different within same group, P ≤ 0.05

Source: SourceOne Global Partner

### Table 3: Sytrinol™ Clinical Study III Results

<table>
<thead>
<tr>
<th>Measured Endpoints</th>
<th>Treatment Group</th>
<th>Sytrinol™ Clinical Study III % Change from placebo group at 12 weeks</th>
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<td>Total Cholesterol</td>
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<td>LDL</td>
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<td>LDL: HDL Ratio</td>
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</table>

Means ± SEM. Statistical analysis by ANOVA plus post test by Dunnett’s method

b – Significantly different from placebo group, P ≤ 0.05

Source: SourceOne Global Partner
12 weeks during which the groups receiving the active compound or placebo will be crossed over.

Only the first 12 weeks (phase 1) of the long-term study have been completed, yet already the results are compelling. As shown in Table 3, compared to placebo, the Sytrinol™ subjects saw reductions of 30% in total cholesterol, 27% in LDL, and 34% in total triglycerides. In addition, HDL levels increased 4%, resulting in a significant 29% reduction in the LDL:HDL ratio.

**Conclusion**

Cholesterol management is a well-established means of maintaining health and preventing premature death from cardiovascular disease. Many people can maintain desirable cholesterol profiles by natural means, including lifestyle modifications, exercise, dietary strategies, and natural hormone replacement protocols. For those in need of additional cholesterol-lowering strategies, Sytrinol™ is an important new option that can help achieve substantial reductions in total cholesterol, LDL, and triglyceride levels, while improving the LDL:HDL ratio. Its lack of the side effects associated with statin drugs makes Sytrinol™ an especially attractive therapy for maintaining healthy cholesterol levels.

**References**


