Phosphatidyl Choline (PC) is the largest of the phospholipids that comprise the membrane of our cells. PC is also prominent in egg and soy lecithin. While PC may be the largest of the phosphatides in lecithin there are other principle phospholipids, phosphatidyl-ethanolamine (PE), phosphatidyl-inositol (PI), phosphatidyl-serine and phosphatidic acid that together, constitute a higher concentration in lecithin than does PC. Soybeans are the principle source of commercial lecithin, and lecithin is the most important by-product of the soy oil processing industry because of its many applications in foods, cosmetics and industrial products. Lecithin is also available as a dietary supplement in two forms: as granular lecithin which contains ~12% PC (calcium phosphate as a flow agent), and in a concentrated form called triple lecithin at ~35% PC in capsules fluidized with soy oil (Wood and Allison 1981). While lecithin has been on the health food scene for over a half century, somehow, it picked up the perception of being called phosphatidylcholine. Many researchers used the two interchangeably as if they were the same. They are not. Lecithin is not PC, and while PC is the largest phosphatide in lecithin, technically, it is only a chemical component of lecithin. Oddly, no one reverses it by saying PC when referring to lecithin.

In April, 2011 in an article in Nature, Gut flora of phosphatidylcholine promotes cardiovascular disease, Wang et al wrote that “Foods rich in the lipid phosphatidylcholine (PC, also called lecithin)* which predominantly include eggs, milk, liver, red meat, poultry, shell fish and fish, are believed to be the major dietary sources for choline”. Note the age-old error again. However, confusing PC and lecithin pales in comparison to the egregious characterization in the title saying that “phosphatidylcholine promotes cardiovascular disease”.

We must presume the authors to be knowledgeable of the long and voluminous history of PC as the largest phospholipid component of our cellular membranes and especially as a nutrient with its quite exceptional history in alleviating heart health disorders specifically cardiovascular disease. The title ‘Gut flora of phosphatidylcholine promotes cardiovascular disease’ completely disregards the scientific history of PC and casts a disturbing and untrue image of a highly valuable nutrient with a long history of lowering cholesterol and successful treatment of atherosclerosis, (Cho BH 2011, Navab M 2003, Chung BH 2005, Hajj Hassan H 2005, Koizumi J 1988, Ke Y 1996, Ovesen L 1985). The title could be correct if lecithin was used, which if intended, should have been so stated.

“The disturbing title further calls in question the veracity of what appears to be significant scientific information. The implication of PC as potentially dangerous is simply wrong; however, if lecithin was used, the study becomes of significant value for shedding light on the difficulty of using lecithin as a nutrient. While there have been a large number of studies over the last 50 years using lecithin as a source for PC, the effort to achieve medical benefits have been plagued with mixed results, while the reverse of using phosphatidylcholine has consistently maintained positive medical results particularly in international studies. Research and clinical experience has revealed that PC is possibly the most prestigious nutrient of all (Yechiel and Barenholtz 1985, Cui and Howling 2002).”

* The authors again make the mistake of referring to PC as Lecithin.
Rhone Poulanc, in its brochure for Lipostabil, lists 197 references for oral and i.v. administration of PC for hyperlipoproteinemia and atherosclerosis. Gundermann KJ, PhD, MD, in “The Essential Phospholipids as a Membrane Therapeutic” 1993, has 776 referenced studies in his technical manual which covers the use of PC in toxicology, hemorrheology, lipid peroxidation, alcohol and diabetic fatty liver, malnutrition, kidney, cirrhosis, gastrointestinal, neurological, lung, psoriasis, MS, cerebral circulation, elevated lipids, atherosclerosis, even drug enhancement. It is a thorough review of PC research published from 1959 to 1993, which portrays a positive image of PC quite the opposite as in the Nature study.

Using lecithin as a source of PC is a poor scientific approach because the phospholipids within lecithin are oil based. The PC used in the European studies was either egg PC, or, if soy lecithin based, the oil had been purged and the phospholipids were isolated. In the 1940s, Nattermann GMBH, successfully produced a high concentrated PC from lecithin in an oil free form called Phospholipon which was the source for the PC used in all the medical studies previously mentioned. The formation of the membrane occurs in water and cannot occur if the lipid tails of the phospholipids are immersed in oil. Oil and water do not mix, but oil and oil mix very well. All life on the planet is water based and depends on the “hydrophobic effect” to drive the formation of the membrane of all cells. There is little excuse for the continued PC / lecithin confusion or the implication of PC as a troublesome nutrient.

Under normal digestion, PC and all lipids ingested are degraded by lipases in the gut. The phospholipids and the triglycerides are reduced by enzymes PLC or PLD which attack the head groups (choline, ethanolamine, etc) with PLA1 or PLA2 doing the same for the lipid tails. The PC research reports referenced above did not use raw lecithin as a source of PC. The majority of the PC was either i.v. Essentiale (Aventis) or Lipostabil (Rhone Poulanc) or, as a PC capsule “Forte” under both labels. Forte is currently available in most European and Eastern European pharmacies, i.v. Essentiale is available in Eastern Europe and the US, all of which used phosphatidylcholine that originated from Nattermann, which, as already indicated, is no longer lecithin.

In the 2011 Nature paper by Wang et al, there are 18 listed authors from the Cleveland Clinic, UCLA, Cleveland State, and USC. It is appalling that such a prestigious journal, Nature, would permit such gross error in basic biochemistry. The article does indeed help to resolve the age-old problem of why lecithin has consistently failed to provide positive results as a phosphatidylcholine nutritional supplement, because it isn’t phosphatidylcholine. The implication of Choline and TMAO in CVD was also corroborated in a number of studies prior to the Nature study such as recent publications from Italy in 2005, UNC in 2007, Emory and Aventis in 2009 and from Germany in 2007 - 2010, to name a few. However, none of these publications mentioned lecithin or even PC as a critical choline source or of PC in promoting cardiovascular disease. In light of the abundant positive research for phosphatidyl choline for over half a century, the choice of the title in the April 2011 Nature paper borders on scientific ignorance and demands a retraction.

In the Nature study it is prudent to bear in mind that the laboratory animals were inbred strains, predisposed to CVD.

There is clear evidence that choline released from the cell membrane by ischemia-related cytolysis may be used as a predictor of cardiac events in the presence of chest pain, despite low levels of troponin. (Danne, 2007) In this regard, choline elevation is a result of tissue damage (vulnerable plaque), and is not a causative agent.

Specific gut bacteria will degrade choline to trimethylamine, which is then oxidized by the liver to TMAO. The liver enzyme, Hepatic Flavin Monoxygenase 3 (FMO3) is responsible for the conversion. (It may be important to note
that this is a “flavin” enzyme, dependent upon riboflavin for its activity. Without further attention, it is unseemly to indict this B vitamin as part of the etiology of CVD.) TMAO—and its companion choline metabolite, betaine—promote upregulation of multiple macrophage scavenger receptors as part of the inflammatory cascade, following platelet activation and monocyte adhesion, but preceding the formation of macrophage foam cells from the endocytosis of oxidized LDL and the subsequent smooth muscle cell migration from vascular epithelium that forms protective fibrous caps. Foam cells that accrue from this phagocytosis comprise the fatty streaks of the plaques of atheroma in the vascular intima. Foam cells necrotize and cause the fibrous cap to rupture and form a thrombus which can lead to emboli capable of occluding smaller blood vessels.

Knowing that gut flora may generate a pro-atherosclerotic metabolite has aroused interest in probiotic research. Lactobacillus rhamnosus appears to potentiate the colonic manufacture of TMAO, while L. paracasei has inhibitory properties on the formation of TMAO. Both strains are dose-dependent. If the inflammation induced by TMAO can be eliminated, or at least curtailed, the number of related cardiac events can be limited. Disrupting the inflammatory cascade that provokes foam cell induction may be as simple as preventing the oxidation of LDL in the first place. Several dietary components have demonstrated the capability to prevent LDL oxidation, including capsaicin, curcumin, and several bioflavonoids.

**In 1998, Crawford et al noted that “…natural dietary antioxidants inhibit both LDL oxidation and atherogenesis in animals with elevated LDL…”**


**In 2005, Indian researchers identified the pepper constituent as cardiac friendly, when they announced, “Dietary capsaicin was found to be protective to the LDL oxidation…as indicated by reduction in TBARS by more than 40%.”**


**Quercitin (a constituent of apples and onions) and curcumin (the active ingredient of turmeric) demonstrated the greatest effects in the prevention of LDL oxidation. Both are available as supplements.**


Future treatment of CVD symptoms may include testing for TMAO, as well as for the traditional cardiac markers. Preventive measures, especially for individuals with a family history of heart disease, may address limiting choline intake, reducing gut bacterial load via broad spectrum antibiotics, and/or using probiotics designed to modulate intestinal ecology.

The highest sources of Choline are in liver, egg yolk, red meat, fish, milk, chicken, and peanuts while the highest source of Betaine is in dark bread, white bread, spinach, cold breakfast cereals, and pasta. So becoming a vegetarian or avoiding meat will not solve the problem of avoiding the formation of plaque if a gut infection is brewing.
Practical suggestions to patients to optimize cardiovascular health include the following:

- Limit choline intake in supplements which includes:
  - B complex, Choline, Lecithin, Multivitamins w/ choline, glycerophosphocholine

- Limit other TMAO stimulating supplements such as betaine, methionine, L-Carnitine, Trimethylglycine (TMG)

- Reduce gut bacteria load (low dose antibiotics for a short period of time if an infection is identified in the gut)

- Discontinue any probiotics that contain L. Ramllosus (most do, read everything, even kefir and yogurt containers)

- Use probiotics as lactic acid bacteria (homemade kefir) or the probiotic VSL #3 which is a colonizes in the gut and most importantly

Use The Probiotic L. Paracasei Crucial Lipid Supplementation Includes:

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- Balanced essential fatty acids, both omega 6 and omega 3
- Butyrate and/or Phenylbutyrate

Intravenous Lipid Therapy With Essentiale/Phosphatidylcholine

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Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease

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ABSTRACT: Metabolomics studies hold promise for the discovery of pathways linked to disease processes. Cardiovascular disease (CVD) represents the leading cause of death and morbidity worldwide. Here we used a metabolomics approach to generate unbiased small-molecule metabolic profiles in plasma that predict risk for CVD. Three metabolites of the dietary lipid phosphatidylcholine—choline, trimethylamine N-oxide (TMAO) and betaine—were identified and then shown to predict risk for CVD in an independent large clinical cohort. Dietary supplementation of mice with choline, TMAO or betaine promoted upregulation of multiple macrophage scavenger receptors linked to atherosclerosis, and supplementation with choline or TMAO promoted atherosclerosis. Studies using germ-free mice confirmed a critical role for dietary choline and gut flora in TMAO production, augmented macrophage cholesterol accumulation and foam cell formation. Suppression of intestinal microflora in atherosclerosis-prone mice inhibited dietary-choline-enhanced atherosclerosis. Genetic variations controlling expression of flavin monooxygenases, an enzymatic source of TMAO, segregated with atherosclerosis in hyperlipidaemic mice. Discovery of a relationship between gut-flora-dependent metabolism of dietary phosphatidylcholine and CVD pathogenesis provides opportunities for the development of new diagnostic tests and therapeutic approaches for atherosclerotic heart disease.


Effects of consumption of choline and lecithin on neurological and cardiovascular systems. Wood JL, Allison RG.

This report concerns possible adverse health effects and benefits that might result from consumption of large amounts of choline, lecithin, or phosphatidylcholine. Indications from preliminary investigations that administration of choline or lecithin might alleviate some neurological disturbances, prevent hypercholesteremia and atherosclerosis, and restore memory and cognition have resulted in much research and public interest. Symptoms of tardive dyskinesia and Alzheimer’s disease have been ameliorated in some patients and varied responses have been observed in the treatment of Gilles de la Tourette’s disease, Friedreich’s ataxia, levodopa-induced dyskinesia, mania, Huntington’s disease, and myasthenic syndrome. Further clinical trials, especially in conjunction with cholinergic drugs, are considered
worthwhile but will require sufficient amounts of pure phosphatidylcholine. The public has access to large amounts of commercial lecithin. Because high intakes of lecithin or choline produce acute gastrointestinal distress, sweating, salivation, and anorexia, it is improbable that individuals will incur lasting health hazards from self-administration of either compound. Development of depression or supersensitivity of dopamine receptors and disturbance of the cholinergic-dopaminergic-serotonergic balance is a concern with prolonged, repeated intakes of large amounts of lecithin.


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**BACKGROUND:** Lecithin has been widely sold as a dietary supplement. 1,2-Dimyristoyl-sn-glycero-3-phosphocholine (DMPC) is a phospholipid that does not exist in nature and has been used in vitro to study lipid binding. We tested DMPC in vivo in apolipoprotein (apo) E–null mice.

**METHODS AND RESULTS:** DMPC or soy or egg lecithin at 1.0 mg/mL was added to the drinking water of 4-week-old apoE-null female mice. Eight weeks later, HDL cholesterol levels and apoA-I levels were markedly increased in the mice that received DMPC. HDL function was also dramatically improved in the mice receiving DMPC, and there was a significant reduction in aortic lesions (P=0.021) in the DMPC mice but not in those receiving lecithin. Adding 1.0 mg/mL of DMPC to the drinking water of 6-month-old apoE-null female mice for 5 weeks caused regression of aortic sinus lesions (P=0.003). Adding 1.0 mg/mL DMPC to the drinking water of 6-month-old apoE-null male mice for 8 weeks significantly reduced aortic lesions (P=0.031) and en face whole aorta lesion area (P=0.001), whereas adding the same concentrations of soy or egg lecithin did not significantly alter lesion area. Jejunal apoA-I synthesis and plasma apoA-I levels were increased 2- to 3-fold in mice receiving DMPC but not soy or egg lecithin.

**Conclusions:** DMPC (but not lecithin) raises HDL cholesterol and apoA-I, improves HDL function, and prevents lesions or causes their regression in apoE-null mice.


Synthetic dimyristoylphosphatidylcholine liposomes assimilating into high-density lipoprotein promote regression of atherosclerotic lesions in cholesterol-fed rabbits.

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**ABSTRACT:** We have reported recently that enrichment of high-density lipoprotein (HDL) with phosphatidylcholine (PC) liposomes is effective in solubilizing cholesterol from isolated human atherosclerotic plaques. In the present study, we investigated the in vivo effect of enrichment of HDL with PC on regression of diet-induced atherosclerosis in rabbits. As part of the study, a preliminary in vitro study on blood collected from the cholesterol-fed rabbits was performed to assess the capacity of the HDL density (d > 1.063 g/mL) plasma fraction from cholesterol-fed rabbits to assimilate multilamellar liposomes of synthetic dimyristoylphosphatidylcholine (DMPC). This was compared with the capacities of egg- and soy-PC liposomes to be assimilated into the HDL density plasma fraction. The capacity of the HDL density fraction to absorb PC from DMPC liposomes (11.5 mg/mL) was more than 10 times greater than egg or soy liposomes. Therefore, DMPC liposomes were chosen to infuse into cholesterol-fed rabbits. Cholesterol-fed rabbits infused weekly with DMPC liposomes (300 mg/kg body weight) for five weeks had significantly decreased aortic cholesterol contents (P < 0.05) compared with saline-infused cholesterol-fed controls. Atherosclerotic plaque volume, as measured by a type of new magnetic resonance imaging analysis, also decreased significantly (P < 0.05) after DMPC treatment. The present findings suggest that the enrichment of HDL with PC via intravenous infusion of synthetic DMPC liposomes could be a potential therapeutic approach for atherosclerotic plaque regression.


Oral synthetic phospholipid (DMPC) raises high-density lipoprotein cholesterol levels, improves high-density lipoprotein function, and markedly reduces atherosclerosis in apolipoprotein E–null mice.

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**BACKGROUND:** Lecithin has been widely sold as a dietary supplement. 1,2-dimyristoyl-sn-glycero-3-phosphocholine (DMPC) is a phospholipid that does not exist in nature and has been used in vitro to study lipid binding. We tested DMPC in vivo in apolipoprotein (apo) E–null mice.

**METHODS AND RESULTS:** DMPC or soy or egg lecithin at 1.0 mg/mL was added to the drinking water of 4-week-old apoE-null female mice. Eight weeks later, HDL cholesterol levels and apoA-I levels were markedly increased in the mice that received DMPC. HDL function was also dramatically improved in the mice receiving DMPC, and there was a significant reduction in aortic lesions (P=0.021) in the DMPC mice but not in those receiving lecithin. Adding 1.0 mg/mL of DMPC to the drinking water of 10-month-old apoE-null female mice for 5 weeks had significantly decreased aortic cholesterol contents (P < 0.05) compared with saline-infused cholesterol-fed controls. Atherosclerotic plaque regression.

**CONCLUSIONS:** DMPC (but not lecithin) raises HDL cholesterol and apoA-I, improves HDL function, and prevents lesions or causes their regression in apoE-null mice.

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Phosphatidylcholine-rich acceptors, but not native HDL or its apolipoproteins, mobilize cholesterol from cholesterol-rich insoluble components of human atherosclerotic plaques.

Chung BH, Franklin F, Liang P, Doran S, Cho BH, Curcio CA. Gerontology Division, Department of Medicine, University of Alabama at Birmingham, Birmingham, AL 35294, USA. bichung@uab.edu
ABSTRACT: To examine the potential of high density lipoproteins (HDL) to ameliorate atherosclerotic plaques in vivo, we examined the ability of native HDL, lipid-free HDL apolipoproteins (apo HDL), cholesterol-free discoidal reconstituted HDL (R-HDL) comprised of apo HDL and phosphatidycholine (PC) and PC liposomes to release cholesterol from cholesterol-rich insoluble components of plaques (ICP) isolated from atherosclerotic human aorta. Isolated ICP had a free cholesterol (FC) to phospholipid (PL) mass ratio (0.8-3.1) and a sphingomyelin (SPM) to PC mass ratio (1.2-4.2) that exceeded those of plasma membranes of cultured cells. Surprisingly, native HDL and its apolipoproteins were not able to release cholesterol from ICP. However, R-HDL and PC liposomes were effectively released cholesterol from ICP. The release of ICP cholesterol by R-HDL was dose-dependent and accompanied by the transfer of > 8 x more PC in the reverse direction (i.e., from R-HDL to ICP), resulting in a marked enrichment of ICP with PC. Compared to R-HDL, PC liposomes were significantly less effective in releasing cholesterol from ICP but were somewhat more effective in enriching ICP with PC. Native HDL was minimally effective in enriching ICP with PC, but became effective after prior in vitro enrichment of HDL with PC from multimamellar PC liposomes. The enrichment of ICP with PC resulted in the dissolution of cholesterol crystals on ICP and allowed the removal of ICP cholesterol by apo HDL and plasma. Our study revealed that the removal of cholesterol from ICP in vivo will be possible through a change in the level, composition, and physical state of ICP lipids mediated by PC-enriched HDL.


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ABSTRACT: It has been suggested that ABCA1 interacts preferentially with lipid-poor apolipoprotein A-I (apoA-I). Here, we show that treatment of plasma with dimyristoyl phosphatidycholine (DMPC) multimamellar vesicles generates prebeta(1)-apoA-I-containing lipoproteins (LpA-I)-like particles similar to those of native plasma. Isolated prebeta(1)-LpA-I-like particles inhibited the binding of (125)I-apoA-I to ABCA1 more efficiently than HDL(3) (IC50 = 2.20 +/- 0.35 vs. 37.60 +/- 4.78 microg/ml). We next investigated the ability of DMPC-treated plasma to promote phospholipid and unesterified (free) cholesterol efflux from J774 macrophages stimulated or not with cAMP. At 2 mg DMPC/ml plasma, both phospholipid and free cholesterol efflux were increased (approximately 50% and 40%, respectively) in cAMP-stimulated cells compared with unstimulated cells. Similarly, both phospholipid and free cholesterol efflux to either isolated native prebeta(1)-LpA-I and prebeta(1)-LpA-I-like particles were increased significantly in stimulated cells. Furthermore, glyburide significantly inhibited phospholipid and free cholesterol efflux to DMPC-treated plasma. Removal of apoA-I-containing lipoproteins from normolipidemic plasma drastically reduced free cholesterol efflux mediated by DMPC-treated plasma. Finally, treatment of Tangier disease plasma with DMPC affected the amount of neither prebeta(1)-LpA-I nor free cholesterol efflux. These results indicate that DMPC enrichment of normal plasma resulted in the redistribution of apoA-I from alpha-HDL to prebeta-HDL, allowing for more efficient ABCA1-mediated cellular lipid release. Increasing the plasma prebeta(1)-LpA-I level by either pharmacological agents or direct infusions might prevent foam cell formation and reduce atherosclerotic vascular disease.


ABSTRACT: Apolipoprotein A-I was purified from human high density lipoprotein and complexed with polynsaturated phosphatidycholine (PC) in deoxycholate (Lipostabil); the bile salt was removed subsequently by dialysis. The behavior of the resultant apoA-I/PC complexes was compared with that of Lipostabil in vitro and after injection into rabbits. In vivo apoA-I/PC complexes had the density of HDL throughout but had both alpha and pre beta electrophoretic mobility, the latter probably reflecting dissociation of apoA-I from PC. Lipostabil initially behaved like LDL but gradually acquired the density of HDL after incubation with plasma and in vivo. Both preparations increased plasma total phospholipids in normolipidemic rabbits to a similar extent; but, increments in HDL phospholipid were greater after apoA-I/PC complexes were injected. ApoHDL/PC complexes, prepared in a similar manner, appeared to promote efflux of cholesterol from perfused rabbit aortas in the presence of lecithin:cholesterol acyltransferase (LCAT) activity consistent with a stimulatory effect on cholesterol mobilization. Injection of apoHDL/PC complexes into hyperlipidemic rabbits decreased plasma cholesterol but increased HDL cholesterol, whereas Lipostabil decreased both. These findings suggest that human apoA-I/PC complexes resemble HDL in their behavior more closely than does Lipostabil, and show that both types of liposome undergo modification upon interaction with plasma. It remains to be shown whether they possess any therapeutic potential.


ABSTRACT: In order to study the effect of oral phospholipid liposomes on regression of atherosclerosis process, serum total cholesterol triglyceride, lipoprotein cholesterol and atherosclerotic lesion on the aorta wall in different groups of 20 New Zealand rabbits were observed. Results showed that phospholipid liposomes could obviously increase high density lipoprotein cholesterol concentration and decrease serum total cholesterol triglyceride and very low density lipoprotein cholesterol concentration. Phospholipid liposomes could also reduce the size of atherosclerotic lesion on the aortic wall. In comparison with the results observed in phospholipid group, there was significant difference (P < 0.05, P < 0.01 or P < 0.001). It suggested that oral phospholipid liposomes could effectively regress atherosclerotic lesion on the arterial wall and regulate serum lipid to reduce cholesterol deposit on the arterial wall in experimental rabbits. It might provide some experimental basis for human being to prevent and treat atherosclerosis.
**Abstract:** In a randomized, double-blind, cross-over trial soybean phospholipid and placebo, 18 g daily for 6 wk, were given orally to 20 patients on long-term treatment with standard lipid lowering diets. The effect of this treatment on serum total cholesterol and high-density lipoprotein cholesterol and plasma triglyceride was studied. After 6 wk mean (+/- SE) cholesterol concentration was decreased by 0.54 (+/- 0.19) mmol/liter in phospholipid-treated as compared to placebo-treated patients (p less than 0.02). The decrease in serum cholesterol was significant (p less than 0.02) only in patients assigned to receive phospholipid before placebo. A highly significant increase (p less than 0.001) followed the withdrawal of phospholipid. No effect on triglyceride and high-density lipoprotein cholesterol concentrations was demonstrated.

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Whole blood choline and plasma choline in acute coronary syndromes: Prognostic and pathophysiological implications

Oliver Danne, Christian Lueders, Christian Storm, Ulrich Frei, Martin Möckel

**Background:** Whole blood choline (WBCHO) and plasma choline (PLCHO) concentrations increase rapidly after stimulation of phospholipase D in acute coronary syndromes (ACS). Early risk-stratification was analyzed in 217 patients with suspected ACS and a negative admission troponin T (< 0.03 μg/L).

**Methods:** WBCHO and PLCHO were measured using high-performance-liquid-chromatography mass spectrometry. Major cardiac events (MACE) were defined as cardiac death/arrest, coronary intervention or myocardial infarction (MI).

**Results:** WBCHO (≥ 28.2 μmol/L) was predictive for MACE (hazard ratio [HR] 2.7; p < 0.001), cardiac death/arrest (HR 4.2; p = 0.015), heart failure (HR 2.8; p = 0.003), coronary intervention (HR 2.1; p = 0.01) and MI (HR 8.4; p = 0.002) after 30 days. PLCHO (≥ 25.0 μmol/L) was predictive for MACE (HR 2.6; p = 0.005), cardiac death/arrest (HR 15.7; p < 0.001), heart failure (HR 6.0; p < 0.001) but not for coronary intervention and MI. WBCHO and PLCHO were predictive for MACE in multivariate analysis (Odds ratio [OR] 2.7, p = 0.009 and OR 3.3, p = 0.03) independently of age, gender, prior MI, coronary risk factors and ECG.

**Conclusions:** WBCHO and PLCHO are significant and independent predictors of major cardiac events in admission troponin T negative acute coronary syndromes. Both are predictive for events related to tissue ischemia and WBCHO is capable of detecting risks associated with coronary plaque instability.

**Additional References**


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Dr. Wnorowski is a clinical nutrition counselor, certified (CNCC) specializing in migraine prevention and management. His post-doctoral education at Purdue, Tufts, and Johns Hopkins includes the clinical management of obesity, diabetes education, cardiac rehabilitation, women’s and men’s health issues, and weight management. He is the founder of Nutricom, LLC, a provider of current research in nutrition, complementary care, and sports medicine. Dr. Wnorowski is on the advisory board of the Center for Mind/Body Medicine, in Washington, D.C., and is a published author in the field of general nutrition. He has a PhD in Clinical Nutrition Counseling and an MSc in Human Nutrition, both from Madison University, and a MS in educational psychology from Glassboro State College. Dr. Wnorowski is a member of the American Dietetic Association, American Association of Diabetes Educators, the American Association of Integrative Medicine, and the Juvenile Diabetes Research Foundation. He practices privately in southern New Jersey and was the honored recipient of the 2006 Excellence in Practice Award from the American Dietetic Association.

Edward Kane, Chairman of the Board and CEO

Mr. Kane’s significant business experience dates back to 1955 when he founded Kane Steel, a $35 million steel processing company with over 120 employees. In 1964 Mr. Kane started K-TRON International (Nasdaq: KTH), the first company to digitize weight feeding for the process industries, which today is a $250 million company. In 1980, Mr. Kane started K-FLOW International to manufacture a patented mass flow meter, a hi-tech, extremely accurate means of measuring fluids for industrial use. In 1991, the company was merged into the instrument division of the ABB Group, headquartered in Zurich, Switzerland. Expanding his horizons into the health field ten years ago, Mr. Kane and his wife Patricia Kane Ph.D, founded BodyBio Corporation, a specialized nutritional diagnostic service utilized by physicians worldwide in interpreting blood test results. In 1999, Mr. Kane was honored as one of 3 “Major Industrialists of the Half Century” as part of the Outstanding Business Persons of the 20th Century selections awarded by the Greater Millville (NJ) Chamber of Commerce. Ed Kane is currently on the Advisory Board of the Osteopathic Medical School, Stratford NJ.

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Patricia Kane, PhD

Patricia Kane, Ph.D is the Director of The NeuroLipid Research Foundation, a nonprofit organization, in Millville, NJ and serves as Director of Medical Research at BodyBio, involved with biomedical analysis system for fatty acid and biochemical testing. Her expertise in boldly addressing complex metabolic aberrations and lipid disturbances with targeted nutritional intervention for neurological disorders has led to direct nutrient application utilized by medical doctors worldwide. Her clinical experience, working with physicians, spans 30 years. Dr. Kane has pioneered targeted metabolic application of premier red cell fatty acid analysis for pediatric and adult populations. In doing so, she has expedited the incorporation of current research, evidence based medicine, directly into clinical practice to yield resolution of some of the complex medical disorders of our time. Dr. Kane has directed her main interest, for the past nine years, toward severe neurological disorders and has developed a patented oral and intravenous protocol that has yielded marked positive responses. Dr. Kane has authored numerous medical papers and books focusing on evidence based nutrition for over 30 years. She was a contributor of two chapters in the 2009 medical book, Food and Nutrients in Disease Management, edited by Ingrid Kohlstadt, Johns Hopkins University, on seizure disorders and autism. Dr. Kane is an accomplished lecturer as well as author, presenting to medical societies annually in the US and abroad which include the NIH, Columbia, Johns Hopkins, University of Kansas, University of North Carolina at Chapel Hill, Harvard, Eidgenossische Technische, and the Royal College of Physicians.