

**Before the
FOOD AND DRUG ADMINISTRATION
Washington, D.C.**

In re: Petition for Health Claim:)
Folic Acid, Vitamin B6, and) **Docket No. 99P-3029**
Vitamin B12 Dietary Supplements)
And Vascular Disease)
)

**MOTION FOR LEAVE TO
SUPPLEMENT COMMENTS WITH NEWLY PUBLISHED SCIENTIFIC
LITERATURE**

Julian M. Whitaker, M.D.; Pure Encapsulations, Inc.; Durk Pearson and Sandy Shaw; and the American Preventive Medical Association (collectively, "Health Claim Petitioners") hereby move to supplement the record in the above-referenced proceeding with the attached scientific journal articles and studies. The articles and studies are submitted over a month in advance of the November 24, 2000 deadline set by the agency (and ordered by the U.S. District Court) for completion of a further evaluation of the above-referenced claim following FDA's November 30, 1999 denial of the claim. The articles and studies are submitted after the date set by the agency for filing additional science in this docket; nevertheless good cause exists for agency review of the submission after the agency's deadline.

Through diligence the Health Claim Petitioners could not have filed the studies and articles in advance of the comment deadline because they have only recently been published. Moreover, the studies and articles are weighty evidence, in the form of peer-reviewed scientific literature, that, if omitted from agency analysis, could result in findings of fact or conclusions of law that are contradicted by the studies and articles. The studies and articles reveal that oral supplementation with folic acid and Vitamins B6 and B12 results in a reduction of vascular disease risk, and they corroborate the causal role that

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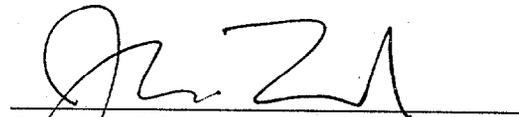
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homocysteine plays in vascular disease etiology and progression. If FDA elects not to review this scientific literature and suppresses the proposed health claim based in whole or part on a perceived absence of evidence supporting the claim, that action would violate the First Amendment. Such an election would ignore proof of the veracity of the proposed claim at a time when that evidence was available and could have been reviewed. The agency has more than a month from the date of this filing before its self-imposed deadline for decision, ample time for it to review the studies and articles and evaluate their significance. For the foregoing reasons, the Health Claim Petitioners respectfully request that the agency accept this supplement and the attached studies and articles for review.

Sincerely,

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DURK PEARSON and SANDY SHAW;
and the AMERICAN PREVENTIVE
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MEMORANDUM IN SUPPORT OF MOTION

The Health Claim Petitioners respectfully request that FDA review scientific articles and studies described below in connection with its evaluation of the proposed health claim that is the subject of this rulemaking proceeding.

The scientific articles and studies described below and contained in Attachment 1 recently appeared in peer reviewed scientific journals. They provide evidence of vascular disease risk reduction resulting from oral supplementation with folic acid and vitamins B6 and B12 and they corroborate the causal role that homocysteine plays in vascular disease etiology and progression. In addition, the National Institute of Neurological Disorders and Stroke (NINDS) at the National Institutes of Health (NIH) has recently published its protocol and recruiting materials for its Vitamin Intervention for Stroke Prevention clinical trials (See Attachment 2). NINDS is further examining in Phase III trials the effectiveness of vitamins B6 and B12 and folic acid in preventing cardiovascular events, stroke or myocardial infarction in patients who have had a first stroke. NINDS states

Higher blood levels of a natural chemical known as homocysteine may contribute to hardening of the arteries in the brain or heart and increase the risk of stroke or heart attack. Folic acid, vitamin B6 (pyridoxine) and vitamin B12 (cyanocobalamin) may lower blood levels of homocysteine and reduce the risk of

having another stroke or a heart attack...because homocysteine may be a major contributor to stroke, its reduction by appropriate intervention with vitamin supplements could reduce the impact of recurrent stroke, myocardial infarction, and vascular death.

The NINDS initiation of expensive Phase III trials is substantial evidence of significant scientific agreement that the nutrients may reduce the risk of vascular disease and/or events. FDA approved clinical trials for experimental drugs proceed through four phases. A clinical trial is designated as phase I, II, III, or IV based upon the type of evidence the study seeks to establish. FDA defines the phases as follows:

- In Phase I clinical trials, researchers test a new drug or treatment in a small group of people (20-80) for the first time to evaluate its safety, determine a safe dosage range, and identify side effects.
- In Phase II clinical trials, the study drug or treatment is given to a larger group of people (100-300) to see if it is effective and to further evaluate its safety.
- In Phase III studies, the study drug or treatment is given to large groups of people (1,000-3,000) to confirm its effectiveness, monitor side effects, compare it to commonly used treatments, and collect information that will allow the drug or treatment to be used safely.
- Phase IV studies are done after the drug or treatment has been marketed. These studies continue testing the study drug or treatment to collect information about their effect in various populations and any side effects associated with long-term use.

See 21 C.F.R. § 312.21. To proceed with the Vitamin Intervention for Stroke Prevention trial as a Phase III trial, NIH and NINDS had to first conclude that sufficient evidence exists to indicate that oral supplementation with folic acid and vitamins B6 and B12 may reduce the risk of vascular death, heart attack, and/or recurrent stroke. Clearly, they have so concluded or else they could not proceed with Phase III studies.

The following newly published scientific articles and studies confirm that “there is convincing evidence that homocysteine contributes to the development of atherosclerosis” and that reducing homocysteine by folic acid consumption, with vitamin B12 and B6 supplementation is a “prudent recommendation for those at risk for

atherosclerotic disease” while awaiting results of “large scale, prospective, randomized clinical trials [to] establish the degree of prevention afforded the atherosclerotic disease population and the optimal folate dose.” Scott CH and Sutton MS. Homocysteine: evidence for a causal relationship with cardiovascular disease. *Cardiol Rev.* 1999; 7:101-7.

1. Andreotti F., et al., Homocysteine and risk of cardiovascular disease. *J Thrombolysis*, 2000; 9: 13-21. In this article, the authors reviewed scientific literature concerning homocysteine and cardiovascular disease risk. The researchers examined that data and concluded that elevated homocysteine levels are a strong, independent, and graded indicator of risk for all categories of cardiovascular disease. The authors believe the evidence is too strong to dismiss homocysteine as “a mere marker manifest or subclinical disease without contributing to its development.” Given the strong link between folic acid, homocysteine and risk of cardiovascular disease, the authors concur with recommendations that everyone take daily folic acid supplements while awaiting irrefutable proof that hyperhomocysteinemia actually causes atherothrombosis.

2. Aronow WS and Ahn C, Increased plasma homocysteine is an independent predictor of new coronary events in older persons. *Am J Cardiol*, 2000; 86: 346-347. In a 31-month follow-up controlled clinical trial, researchers examined the association between plasma homocysteine concentrations and new coronary events in older adults. The researchers had earlier reported that elevated plasma homocysteine levels and low plasma folate and vitamin B12 levels were associated with a higher prevalence of coronary artery disease (CAD) in older adults (60-99 years old). In a follow-up prospective study, the investigators monitored plasma homocysteine, folate,

and vitamin B12 levels and the following other risk factors: prior CAD; current cigarette smoking; hypertension; diabetes; fasting total cholesterol; HDL cholesterol; triglycerides; and obesity. Stepwise Cox regression analysis showed that plasma homocysteine (overall risk ratio=1.078) was a significant independent predictor of new coronary events.

Homocysteine is an independent predictor of new coronary events in subjects with prior CAD (risk ratio=1.068) and without prior CAD (risk ratio=1.106). The authors caution that if other risk factors are present, they must still be treated.

3. Brattström L and Wilcken D, Homocysteine and cardiovascular disease: cause or effect? *Am J Clin Nutr*, 2000, 72: 315-323. In this review of scientific literature the authors concur that markedly and mildly elevated homocysteine concentrations are associated with increased risk of vascular occlusions. They question, however, whether mildly elevated homocysteine levels are casually linked to vascular disease. In their analysis, the authors state that the studies showing reduction in cardiovascular events with folic acid and other B vitamin therapy may in fact be due to independent protection offered by those nutrients and not necessarily the homocysteine lowering properties offered by them. Regardless of the mechanism of action, the authors concede that studies of measurable endpoints of cardiovascular disease demonstrate that the B vitamins, especially folate, reduce risk of atherosclerosis.

4. Chambers JC, et al., Plasma homocysteine concentrations and risk of coronary heart disease in UK Asian and European men. *Lancet* 2000, 355: 523-27. Through two parallel case-controlled studies, researchers investigated the relationship between elevated plasma homocysteine concentrations and coronary heart disease (CHD) risk. The studies examined more than 1575 European and Indian Asian men. Indian

Asian men are at increased risk of and mortality due to CHD as compared to their European counterparts in the UK. The Indian Asian men with CHD had higher homocysteine levels than controls and after statistical analysis and adjustments for relevant factors, the results indicate that the proportionate excess CHD risk observed in the Indian Asian group is attributable to elevated homocysteine. The authors conclude that plasma homocysteine is a novel and independent risk factor for CHD in Indian Asians. The authors further concluded that the plasma homocysteine concentration differences were explained by lower folate and vitamin B12 levels in the Indian Asians. The results of the study provide additional evidence that elevated plasma homocysteine levels are independent risk factors for CHD, modifiable by dietary intervention with vitamin B12 and folic acid supplementation.

5. Chao C-L, et al., The graded effect of hyperhomocysteinemia on the severity and extent of coronary atherosclerosis. *Atheroscler* 1999; 147: 379-386. In a controlled clinical study, researchers examined the relationship between homocysteine levels and severity and extent of coronary arteriosclerosis in an Asian population. Based upon serial coronary angiography and plasma profiles, the univariate and multivariate analysis study revealed increased risk of coronary artery disease (CAD) with elevated homocysteine concentrations equal to or greater than 10.1 $\mu\text{mol/l}$. The data showed that homocysteine concentrations equal to or greater than 10.1 $\mu\text{mol/l}$ have a graded effect on CAD. The subjects in the top two quartiles were 4 and 3.2 times more likely to have CAD than those in the lowest quartile. The data also showed that both severity and extent scores for CAD were positively associated with elevated homocysteine levels.

6. Constans J, et al., Three months supplementation of hyperhomocysteinaemic patients with folic acid and vitamin B6 improves biological markers of endothelial dysfunction. *Br J Haematol*, 1999; 107: 776-778. In a controlled clinical study, patients with arterial or venous vascular disease and elevated homocysteine levels were treated for three months with oral folic acid and vitamin B6 supplements. Compared to baseline, the folic acid and vitamin B6 decreased the fasting soluble thrombomodulin, which is a marker of endothelial injury and the homocysteine concentrations. The results of this study support the vascular protective role of folic acid and vitamin B6.

7. Green DJ, et al., *Clin Exp Pharmacol Physiol* 1999, 26: 853-6. In a small controlled study of cardiac patients in a clinical setting, intravenous infusions of chelating agents were effective in increasing vasodilation when vitamins B1, B2, B6 and B12 were included in the infusions. The increased vasodilation was accompanied by a decrease in plasma homocysteine concentrations.

8. Hackam DG, Preston JC and Spence JD. What Level of Plasma Homocyst(e)ine Should Be Treated? Effects of Vitamin Therapy on Progression of Carotid Atherosclerosis in Patients with Homocyst(e)ine Levels above and below 14µmol/L. *Am J Hypertension*, 2000; 13: 105-110. The authors of this controlled intervention study examined the effect of vitamin treatment on the rate of progression of carotid plaque before and after treatment with folic acid and vitamins B6 and B12. The rate of progression of total atherosclerotic progression was steeply upward before initiation of vitamin therapy. The rate of regression of atherosclerotic progress was greater than the rate of progression after treatment with the three vitamins. The vitamins

reversed the progress of plaque formation. The authors conclude that the results of the study support the causal relationship between homocysteine and vascular disease and the protective effects of folic acid and vitamins B6 and B12.

9. Jakubowski H et al., Homocysteine Thiollactone and Protein

Homocysteinylation in Human Endothelial Cells: Implications for Atherosclerosis. *Circ Res* 2000; 87: 45-51. In a series of *in vitro* experiments, researchers demonstrated that (1) homocysteine thiollactone is an important component of homocysteine metabolism in human vascular endothelial cells; (2) homocysteine is incorporated into proteins; and (3) the extent of thiollactone formation and protein homocysteinylation depends on the extracellular concentrations of homocysteine, folic acid, and high density lipoproteins. The experiments also showed that a lack of folic acid is a major determinant of efficient thiollactone synthesis and protein homocysteinylation. The results of the study support a causal relationship between homocysteine and vascular disease.

10. Kullo IJ, Gau GT and Tajik AJ, Novel Risk Factors for Atherosclerosis,

Mayo Clin Proc, 2000, Apr 75: 369-80. In this review of scientific literature, the authors examine the evidence supporting the association of non-conventional risk factors in the pathogenesis of atherosclerosis, the mechanism of risk and the clinical implications of the data. The authors conclude that considerable evidence has accumulated to implicate plasma homocysteine as a risk factor for atherosclerotic vascular disease: coronary artery disease (CAD); peripheral arterial disease; stroke; and venous thrombo-embolism. The authors cite the results of a meta-analysis of 27 clinical studies that indicate that an increment of 5 $\mu\text{mol/L}$ in the homocysteine level increases CAD risk by as much as a 20 mg/dL increase in total cholesterol. The evidence indicates that the relationship between

homocysteine and risk of CAD seems to be linear, rather than having a threshold effect. Moreover, when logistic regression is applied, the increased odds ratio for CAD are seen at total homocysteine levels within the range currently considered normal. The evidence reviewed by the authors indicate that high homocysteine levels are associated with endothelial dysfunction, endothelial and vascular smooth muscle cell damage, activation of clotting factor V, inhibition of protein C, tPA activation, and enhanced platelet aggregation, which are known to be involved in the etiology of atherosclerosis.

11. Leowattana W, et al., Association Between Serum Homocysteine, Vitamin B12, Folate and Thai Coronary Artery Disease Patients, *J Med Assoc Thai* 2000, 83 (5): 536-42. In a cross sectional study, 178 patients with coronary artery disease (CAD) were compared to a group of 178 without CAD. In paired t-test analysis, serum homocysteine was significantly higher in CAD patients than in the healthy controls. The authors concluded that elevated homocysteine levels are a modifiable risk factor. Furthermore, the authors recommend that patients with high homocysteine concentrations should increase their intake of folic acid and vitamins B12 to reduce risk of myocardial infarction.

12. Mansoor MA, et al. Correlation between Total Plasma Homocysteine and Copper in Patients with Peripheral Vascular Disease, *Clin Chem* 2000, Mar, 46: 385-91. In this case control study researchers examined sixty-five patients with peripheral vascular disease and sixty-five gender and age matched healthy control subjects. The results of the study indicate that in persons with peripheral vascular disease higher homocysteine levels are associated with elevated copper and the products of peroxidation that may be produced by the homocysteine-copper interaction. That interaction has been

shown to inhibit nitric oxide induced vasodilation and presents a plausible mechanism for the atherogenic properties of homocysteine.

13. Omland T et al., Serum Homocysteine Concentration as an Indicator of Survival in Patients with Acute Coronary Syndromes, *Arch Intern Med* 2000, Jun 26, 160: 1834-40. In a prospective cohort study in a clinical setting, researchers examined whether homocysteine levels in patients with acute coronary syndromes were predictive of long-term mortality. The results of this two year study demonstrated that after adjusting for relevant confounding variables elevated homocysteine level was a significant independent predictor of long term survival in patients with coronary syndromes and those patients having elevated homocysteine concentrations have poorer prognoses.

14. Riddell LJ, et al., Dietary Strategies for lowering homocysteine concentrations. *Am J Clin Nutr* 2000; 71: 1448-54. The researchers conducted this study to determine effective methods of lowering homocysteine (tHcy) levels. The study was designed based upon the "widespread agreement that an elevated tHcy concentration is an independent risk factor [for vascular events], as powerful as other classic risk factors such as hypertension and hypercholesterolemia." The results of this randomized controlled study demonstrate that daily consumption of folic acid fortified cereals and folic acid supplements are effective means to reduce tHcy levels. Increased folic acid consumption is more effective in reducing tHcy than increased folate intake from folate rich foods alone. The increased folic acid intake from supplements and fortified foods reduced tHcy by more than 20%.

15. Temple ME, Luzier AB and Kazierad DJ, Homocysteine as a risk factor for atherosclerosis. *Ann Pharmacother.* 2000; 34: 57-65. In an extensive and critical analysis of scientific literature, the authors concluded that the data demonstrate: a positive correlation between elevated homocysteine and atherosclerosis; the treatment of choice for lowering homocysteine levels is folic acid; and 650 μ g/day is the minimum effective dose.

16. Turgan N, et al., Plasma Homocysteine Levels in Acute Coronary Syndromes, *Jpn Heart J*, 1999, 40: 729-36. In another study of acute coronary syndrome patients, researchers examined the relationship between plasma homocysteine levels and ischemic heart disease. When compared to controls, a strong positive correlation was observed between elevated homocysteine levels and patients with coronary syndrome. The results of this small study confirmed earlier study results that plasma total homocysteine level is one of the major independent risk factors and a strong predictor of morbidity in acute coronary syndromes. The authors conclude that while causality may not be conclusively "proved" "it seems that reducing elevated homocysteine levels by dietary interventions and vitamin supplementation will be an easy, safe and inexpensive way to treat hyperhomocysteinemia, one of the major modifiable and independent risk factors in ischemic heart disease."

17. Ueland PM, et al., The controversy over homocysteine and cardiovascular risk. *Am J Clin Nutr*, 2000; 72: 324-332. The authors present a comprehensive review of scientific literature and a meta-analysis of controlled studies that examine the relationship between homocysteine (tHcy) and the risk of occlusive cardiovascular disease (CVD). Based upon a critical review of 17 prospective studies and 6 other controlled studies the

authors conclude “elevated tHcy is a causal risk factor for CVD, including venous thrombosis.”

18. Vermeulen EG et al., Effect of homocysteine-lowering treatment with folic acid plus vitamin B6 on progression of subclinical atherosclerosis: a randomised, placebo-controlled trial. *Lancet* 2000, 355: 517-22. In a 2-year randomized placebo-controlled clinical trial, researchers examined the effect of homocysteine-lowering treatment (folic acid plus vitamin B6) on markers of subclinical atherosclerosis among healthy siblings of patients with premature atherothrombotic disease. The investigators measured development or progression of atherosclerosis via exercise electrocardiographs (EKG), ankle-brachial pressure indices, and carotid and femoral artery ultrasound. The vitamin B6 and folic acid supplementation resulted in decreased homocysteine levels and decreased rates of abnormal EKG results. Decreased abnormal exercise EKG tests are consistent with a decreased risk of atherosclerosis. The results of this study are consistent with other data supporting homocysteine as a graded risk factor for atherothrombotic disease.

19. Vermeulan EG, et al., Normohomocysteinemia and vitamin-treated hyperhomocysteinemia are associated with similar risks of cardiovascular events in patients with premature atherothrombotic cerebrovascular disease: A prospective cohort study. *Neth J Med.*, 2000, 56: 138-46. In this controlled intervention study, the researchers examined the effect of daily supplementation with vitamin B6 and folic acid on the incidence of new cardiovascular events in patients with premature atherothrombotic cerebrovascular disease and mildly high levels of homocysteine. The study compared the course of disease progression between those patients with normal

homocysteine concentrations and those with mildly high concentrations of homocysteine. The results indicate that lowering of homocysteine concentrations with vitamin B6 and folic acid reduces the risk of new cardiovascular events in patients with atherothrombotic disease.

20. Weiss N et al., Influence of Folic Acid, Pyridoxal Phosphate and Cobalamin on Plasma Homocyst(e)ine Levels and the Susceptibility of Low-Density Lipoprotein to *ex vivo* oxidation. *Eur J Med Res* 1999, 4: 425-32. In studying the effect of supplementation of folic acid and vitamins B6 and B12 on homocysteine levels, researchers determined that supplementation with those three B vitamins results not only in lowering of homocysteine levels but also in prolonging low-density lipoprotein (LDL) peroxidation lag time. The researchers further found that the three B-vitamins consistently increased levels of high-density lipoproteins (HDL). The results of this study support the role of the three B vitamins in reducing vascular disease risk by a variety of mechanisms, not by reducing homocysteine alone.

21. Willinek WA, et al., High-normal serum homocysteine concentrations are associated with an increased risk of early atherosclerotic carotid artery wall lesions in healthy subjects. *J Hypertension* 2000, 18: 425-30. This controlled study of otherwise healthy individuals demonstrated that high-normal plasma concentrations of serum homocysteine are strongly associated with increased carotid artery wall thickening. The results of the study support homocysteine as an independent risk factor for early carotid artery atherosclerosis in asymptomatic subjects. The results also indicate that homocysteine levels that have previously been considered "normal" are associated with this atherosclerotic precursor.

22. Wilmink HW, et al., Influence of folic acid on postprandial endothelial dysfunction. *Arterioscler Thromb Vasc Biol.* 2000; 20: 185-188. A randomized, double-blind, placebo controlled crossover trial examined the effects of two weeks of daily oral supplementation with 10 mg of folic acid on vascular endothelial function after fat loading. The study examined the effects of folic acid on healthy adults with normal lipid profiles, folate levels, and homocysteine concentrations who were not taking any medication. The results of the study demonstrated that an acute fat load is associated with increased excretion of oxidative end product malondialdehyde (MDA) in the urine of healthy adults. MDA excretion level is a measure of oxidative damage. Acute fat loading also causes a significant decrease in vascular flow mediated dilation (FMD). The subjects who received the folic acid supplementation had little or no decreases in FMD and no increased MDA in response to fat loading. The study results showed that oral supplementation with folic acid completely prevents the increase in oxygen radical stress and the impaired FMD after an acute fat load. The authors conclude that the data indicates that oral treatment with folic acid corrects endothelial dysfunction and abolishes the increase in radical-damage end products induced by triglyceride-rich lipoproteins. "These data underscore a potential beneficial effect of folic acid supplementation for cardiovascular prevention strategies, especially in patients with an impaired cholesterol remnant clearance, such as in diabetes and familial combined hyperlipidemia." The authors explain that "higher dietary folate intake may also protect healthy humans from daily fat-associated endothelial insults. This may imply that in the general population, as well, a higher folate intake may be vasculoprotective."

23. Woo KS, et al., Folic acid improves arterial endothelial function in adults with hyperhomocysteinemia. *J Am Coll Cardiol.*, 1999; 34: 2002-6. In a double blind, randomized placebo controlled study, researchers examined the effect of folic acid on endothelial physiology of adults with elevated homocysteine concentrations.

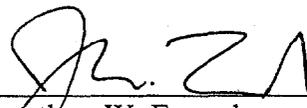
Impairment of the release of nitric oxide by the vascular wall and reduced arterial vasodilation reactivity are associated with endothelial dysfunction and known to be important events in atherogenesis. Compared with placebo, folic acid supplementation resulted in higher folate concentrations, lower total homocysteine levels, and significant improvement in endothelium-dependent dilation. The authors concluded that the evidence supports an association between elevated homocysteine concentrations, on the one hand, and arterial endothelial dysfunction and premature vascular events, on the other.

24. Xu D, Neville R and Finkel T, Homocysteine accelerates endothelial cell senescence. *FEBS Letters*, 2000; 470: 20-24. In a series of *in vitro* experiments, researchers demonstrated that exposure of cultured human endothelial cells to homocysteine significantly accelerates the rate of endothelial senescence. The results of the experiments indicate that homocysteine increases telomere length lost per cell division. Telomere lengths are critical in regulating cell division and replication and are implicated in cellular aging. The authors conclude that a link exists between chronic exposure to homocysteine and the rate of senescence. They also conclude that the senescent endothelial cells exhibit pro-atherogenic characteristics.

Sincerely,

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