However, endothelial dysfunction and HHC was not rectified by vitamin treatment despite a marked increase in serum folate concentrations. Our results do not support the hypothesis that vitamin supplementation improves endothelial function in HHC.

1103-135   Correlation of Peripheral Arterial Compliance and Framingham Coronary Heart Disease Risk Evaluation

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An office-based air plethysmograph, with internal calibration (Vasogram™), was used to measure arterial compliance ($\Delta V/\Delta P$) at the thigh and calf in a 4 center clinical study, with 342 subjects (males aged 31 to 69 and females aged 41 to 79). The subjects were stratified into 4 groups according to Framingham Cardiovascular Risk with 38-47 subjects in each gender/risk group. Group 1- Risk < 10%. Group 2 – Risk $\geq$10% and $\leq$20%. Group 3 – Risk >20% or coronary equivalence but no documented coronary artery disease (CAD). Group 4- documented CAD. Arterial compliance was measured at the thigh and calf levels on each subject, on three different occasions, over a four-week period. Compliance was reported as the maximum volume change (ml) under the cuff occurring during a single cardiac cycle, normalized to a pulse pressure of 50 mmHg (MaxV50) with mean levels summarized in the table below:

Vasogram	Group -1	Group -2	Group-3	Group -4	p-value trend
Female Calf	1.64	1.47	1.34	1.17	< 0.0001
Female Thigh	3.46	2.94	2.67	2.45	< 0.0001
Male Calf	2.53	2.45	2.11	2.03	0.0011
Male Thigh	4.89	4.79	4.04	4.02	0.0003

Female subjects had lower compliance compared to males. Compliance in females and males decreased as the cardiovascular risk increased, but mean compliance did not differ statistically between males in Groups 3 and 4.
Conclusion: Non-invasive measurement of lower extremity arterial compliance is associated with risk of CAD as assessed by the Framingham Risk Score. This test may assist in the detection and management of cardiovascular disease. Assessment of the added value of measuring MaxV50 is currently underway in other studies.

1103-136   Relationship Between Serum Amyloid A and Coronary Artery Disease in Women: The NHLBI-Sponsored Women's Ischemia Syndrome Evaluation (WISE) Study

B. Delia Johnson, Kevin Kip, Paul M. Ridker, Tjendimin Tjandrawan, C. Noel Bairey Merz, Barry L. Sharaf, Carl J. Pepine, William J. Rogers, Sunil Mankad, Mario P. Stylianou, Gerald M. Pohost, Steven E. Reis, University of Pittsburgh, Pittsburgh, PA

Background: Serum amyloid A (SAA), an acute phase inflammatory protein, has been linked to adverse cardiovascular outcome. However its relationship with coronary artery disease (CAD) in women is unknown.
Methods: We studied 705 WISE participants referred for coronary angiography for symptoms suggestive of CAD. All angiograms were quantitatively assessed by core lab. Because of high skew, SAA was log transformed. We used multivariate logistic and linear regression models to assess the association between SAA and various measures of CAD severity. All models were adjusted for age, triglycerides, high density lipoproteins, serum estrone or progesterone (markers of menopausal status), smoking, body mass index, pulse pressure, history of diabetes, dyslipidemia, and hormone replacement use. No other CAD risk factors entered the regression models.
Results: Mean age was 58 years, 18% were non-white, and 256 (36%) had significant angiographic CAD ($\geq$50% stenosis). Mean SAA level was 1.79 mg/dl, range 0.08-73.1. After adjusting for CAD risk factors, SAA had a strong independent association with significant CAD (p=0.004), a WISE CAD severity score (p=0.04), maximum % stenosis (p=0.03), number of significant lesions (p=0.04), and number of diseased vessels (p=0.02).
Conclusions: Among women referred for coronary angiography, the robust relationship between SAA and CAD supports the hypothesis that inflammation modulates atherosclerosis.

Covariate-Adjusted Relationship of (log) SAA with CAD Measures (5 Models)

CAD Measures (Dependent Variables)	Odds Ratios or β	95%Confidence Interval orStandard Error	p
Presence of CAD (yes/no)	1.29	1.08-1.54	0.004
# Diseased Vessels (log)	0.10	0.05	0.02
Maximum % Stenosis	2.46	1.16	0.03
# Lesions (log)	0.10	0.05	0.04
SeverityScore (log)	0.05	0.02	0.04

1104   Platelets, Endothelium, and Thrombosis I

Monday, March 31, 2003, Noon-2:00 p.m.
McCormick Place, Hall A
Presentation Hour: 1:00 p.m.-2:00 p.m.

1104-146   The Entity of the Exercise-Induced Increase in Platelet Reactivity in Patients With Ischemic Heart Disease Is Not Related to the Extension of Coronary Lesions

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Background. Exercise increases platelet aggregability in patients with coronary artery disease (CAD). No previous study, however, assessed whether this increase is influenced by the extension of the atherosclerotic disease in epicardial coronary arteries.
Method. To address the relationships between the severity of coronary artery disease and platelet activation with exercise, venous blood samples were collected prior to exercise and 5 minutes after peak exercise from 188 patients with chronic stable angina with documented flow limiting stenoses (>50% of internal lumen diameter) in one or more major epicardial coronary arteries at angiography. Platelet reactivity was measured on flowing blood as time to occlude a ring coated with collagen-adenosin diphosphate (ADP), using the platelet function analyzer (PFA-100) system. By this method, the time to occlusion (closure time) is taken as a measure of platelet adhesion/aggregability, with shorter times indicating greater platelet reactivity.
Results. Coronary angiography showed 1-vessel disease, 2-vessel diseases and 3-vessel disease in 44% (n=83), 36% (n=67) and 20% (n≈ 38) of patients, respectively. No significant difference was found among these three groups of patients in baseline closure time (93.8±21, 98.1±26, and 94.4±17 sec, respectively, p=0.48). After exercise closure time decreased significantly in all groups (to 80.2±17, 78.0±15, and 83.1±15 s, respectively, p<0.0001 compared to baseline for each group), with values being not significantly different among groups(p=0.31).
Conclusions. Our data confirm that exercise increases platelet reactivity in patients with ischemic heart disease. The entity of the increased platelet reactivity in response to ADP/collagen stimulation after exercise does not seem to be significantly correlated to the extension of coronary atherosclerotic lesions.

1104-147   Blood Glutathione Detects Metabolic Patterns Associated With Atherothrombotic Vascular Events in Patients With Mild Hyperhomocysteinemia

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Background: Hyperhomocysteinemia limits the bioavailability of nitric oxide by a mechanism involving glutathione (GSH) peroxidase. A weak GSH-related antioxidant defense has been documented in human atherosclerotic lesions; an antioxidant/prooxidant imbalance induced by transient hyperhomocysteinemia may uncover abnormal GSH bioavailability and favor vascular damage.
Methods: We examined the effect of transient hyperhomocysteinemia post-methionine loading (PML) in 44 hyperhomocysteinemic subjects (age 47±15 yrs, 29 male), 16 of whom with previous atherothrombotic vascular events, and 12 age and gender-matched healthy subjects with normal homocysteine levels. Blood samples were collected at baseline and 2-3-4 hours PML. Plasma thiols and vitamins C and E were assayed by HPLC; blood GSH was also measured as a marker of tissue GSH status. Free malondialdehyde (MDA) was determined by gas-chromatography.
Results: Percent changes from baseline to 4 hours PML (delta) in plasma vitamin E and blood GSH best discriminated hyperhomocysteinemic from control subjects by logistic regression analysis. Hyperhomocysteinemic patients were classified into 3 percentile groups according to their vitamin E and blood GSH delta. The risk of atherothrombotic events for each percentile group, compared with the risk for the highest percentile group, was estimated and expressed as the odds ratio (OR), after adjustment for age, gender and conventional risk factors. By multivariate analysis, only delta GSH was significantly associated with events (OR, 10.5; 95% CI, 1.3 to 88; p=0.03). In the lowest percentile group of delta GSH, we found higher plasma GSH and cysteinylglycine, both at baseline and PML, higher baseline blood GSH (p<0.01 for all comparisons) and baseline vitamin C (p=0.04) than in the other 2 percentile groups. Nonsignificant trends were observed for free and total homocysteine and cysteine, MDA and vitamin E, both at baseline and PML.
Conclusion: In mild hyperhomocysteinemia, baseline GSH levels in blood, as well as the inability of GSH to increase during transient hyperhomocysteinemia, identify metabolic patterns associated with atherothrombotic vascular events.

1104-148   The Effect of Exercise Training on Endothelial Function in Coronary Artery Disease: Role of Nitric Oxide and Endogenous Antioxidants

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Background: Endothelial dysfunction is well documented in coronary artery disease patients. The mechanism of endothelial dysfunction in these patients may be reduced nitric oxide production and inactivation of nitric oxide by reactive oxygen species. The objective of this study was to investigate the effects of 12 weeks of standard cardiac