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 Disease and Framingham Coronary Heart Disease Risk Evaluation

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An ankle-brachial index (ABI) was used to measure arterial compliance. (Vla50) at the thigh and calf in a 4 center clinical study, with 342 subjects (males aged 51 to 69 and females aged 41 to 79). The subjects were stratified into 4 groups according to Framingham Cardiovascular Risk with 36-47 subjects in each gender/risk group. Group 1: Risk < 10%, Group 2 - Risk ≥10% and ≥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 during a single cardiac cycle, normalized to a pulse pressure of 50 mmHg (MaxV50) with mean levels summarized in the table below.

<table>
<thead>
<tr>
<th>Vasogram</th>
<th>Group -1</th>
<th>Group -2</th>
<th>Group -3</th>
<th>Group -4</th>
<th>p-value trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female Calf</td>
<td>1.64</td>
<td>1.47</td>
<td>1.34</td>
<td>1.17</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Female Thigh</td>
<td>3.46</td>
<td>2.94</td>
<td>2.67</td>
<td>2.45</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Male Calf</td>
<td>2.53</td>
<td>2.42</td>
<td>2.11</td>
<td>2.03</td>
<td>U001</td>
</tr>
<tr>
<td>Male Thigh</td>
<td>4.89</td>
<td>4.79</td>
<td>4.04</td>
<td>4.02</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

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.

Conclusions: Among women referred for coronary angiography, the robust relationship between SAA and CAD supports the hypothesis that inflammation modulates atherosclerotic disease.

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

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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 795 WISE participants referred for coronary angiography for symptomatic suggestive of CAD. All angiograms were quantitatively assessed by core lab. 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.

Results: Percent change from baseline to 4 hours PML (delta) in plasma vitamin E and blood GSH was measured as a marker of tissue GSH status. Free malondialdehyde (MDA) was determined by HPLC; blood GSH was also measured as a marker of tissue GSH status. Free malondialdehyde (MDA) was determined by HPLC; blood GSH was also measured as a marker of tissue GSH status.

1104-147 Blood Glutathione Detects Metabolic Patterns Associated With Atherothrombotic Vascular Events in Patients With 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 were previous atherosclerotic 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 HPLC; blood GSH was also measured as a marker of tissue GSH status.

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 levels. 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 analyses, 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 cysteine/glycine. Both at baseline and PML, higher baseline blood GSH (p<0.01 for all comparisons) and baseline vitamin C (p=0.02) were less 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 objective of this study was to investigate the effects of 12 weeks of standard cardiact