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## As We SEE IT

### A Lethal Misconception of Epidemic Proportion

By William Faloon

Cardiovascular disease causes 1 of every 2.7 deaths in the United States today.<sup>1</sup>

Unless a drastic change occurs in the way doctors treat vascular disorders, aging adults face even greater threats of heart disease and stroke.

Mainstream doctors keep trying to find simple answers to protect against atherosclerotic disease. The problem is that scientists have identified at least 13 different factors that all conspire to destroy our aging arteries.

The illustration below depicts 13 named daggers aimed at the heart. Any one of these daggers can trigger a cardiovascular event, yet conventional medicine pretends that guarding against only a few of these proven risk factors adequately prevents vascular disease.

By failing to comprehend the underlying processes involved in circulatory breakdown, most doctors overlook documented methods of maintaining healthy blood flow in our maturing bodies.

In my early career, I performed over 300 postmortem arterial dissections. In aged cadavers, I often found arteries that were so occluded that it was virtually impossible to insert a small catheter (tube) into them. My experience provided a vivid image of the jagged structural devastation inflicted by atherosclerosis.

As you will read, many of today's physicians seem to have forgotten what they should have observed in medical school. The result is a misconception of epidemic proportion that threatens the lives of tens of millions of aging Americans.

#### CARDIOLOGISTS STILL DON'T GET IT

Mainstream medicine is fixated on the misconception that atherosclerosis is caused by excess levels of "lipids" in the blood. Lipids are usually defined as total cholesterol, LDL (low-density lipoprotein), and triglycerides, though other fatty substances contribute to the atherosclerotic process.

What doctors fail to understand is that atherosclerosis begins when the inner arterial wall (the endothelium) sustains an injury.<sup>2</sup> Endothelial injuries are caused by numerous and often unavoidable factors. As humans age (and engage in bad habits), the endothelium becomes progressively dysfunctional. The end results are the major structural changes clinically defined as "atherosclerosis."

People often analogize atherosclerosis as a "clogged pipe." When a "clog" occurs in a coronary artery, the recommendation is either bypass surgery or a procedure in which a stent is inserted to keep the artery open. While these surgical procedures have become necessary for many people, the "clogged pipe" analogy is an inaccurate way to view the atherosclerotic process.

The underlying reason why arteries become occluded in older people is the progressive deterioration of the endothelial lining (the inner arterial wall). If people do not take steps to correct the "endothelial dysfunction" occurring in their aging bodies, the consequence will be a worsening epidemic of arterial disease that currently kills 37% of all Americans and 30% of people worldwide.<sup>3</sup>



William Faloon

## DAGGERS AIMED AT YOUR ARTERIAL WALL



If one accepts the premise that age-related arterial disease is almost universally caused by “endothelial dysfunction,” then human studies in which a single agent is tested to prevent heart attack or stroke begin to look absurd, at least in those who seek to maintain healthy blood flow over the long term.

High blood pressure,<sup>4-10</sup> excess cholesterol-LDL-triglycerides,<sup>11-20</sup> low HDL,<sup>21-23</sup> cigarette smoking,<sup>24-32</sup> diabetes,<sup>33-38</sup> obesity,<sup>39-42</sup> and lack of exercise<sup>43-49</sup> all contribute to endothelial dysfunction and the subsequent development of atherosclerosis.

Additional endothelium-damaging factors include high-normal levels of glucose,<sup>50-52</sup> insulin,<sup>53,54</sup> iron,<sup>55-57</sup> homocysteine,<sup>58-84</sup> and fibrinogen,<sup>85-98</sup> low free testosterone (in men),<sup>99-102</sup> and any level of C-reactive protein that is higher than optimal.<sup>103-125</sup>

Homocysteine causes initial injury to the endothelium, contributes to the production of pro-inflammatory cytokines, and facilitates the oxidation of the fat/LDL that accumulates beneath the damaged endothelium. Homocysteine also increases the expression of adhesion molecules and the abnormal accumulation of blood components around the atherosclerotic lesion.

Fibrinogen is a clotting factor that accumulates at the site of the endothelial lesion, contributing to plaque buildup. Fibrinogen can also participate in the acute blockage (thrombosis) of an artery after unstable atherosclerotic plaque ruptures.

Glucose at even high-normal levels may accelerate the glycation process that causes arterial stiffening, while high-normal fasting insulin inflicts direct damage to the endothelium and induces abnormal platelet aggregation.

High levels of iron promote oxidation of LDL in the damaged endothelium, while low levels of testosterone appear to interfere with normal endothelial function. Low levels of vitamin K enable calcium to be deposited into atherosclerotic lesions, instead of into the bone where it belongs.<sup>126-129</sup>

C-reactive protein is an inflammatory byproduct that directly damages the endothelium. This not only creates initial injuries to the endothelium, but also accelerates the progression of existing atherosclerotic lesions.

Nitric oxide is produced in the endothelium and regulates endothelial cell interactions, arterial wall blood flow, vascular tone, and platelet aggregation-adhesion. Abnormalities in nitric oxide production are a direct cause of endothelial dysfunction, which is the prelude to atherosclerosis (and often hypertension). The common feature of endothelial dysfunction is a decrease in the amount of nitric oxide.<sup>130</sup>



In response to overwhelming published data, health-conscious people are altering their diets, taking drugs, hormones, and dietary supplements, and even trying to exercise in order to reduce all of these known atherosclerosis risk factors. Despite these efforts, protecting against these risk factors until now has been only a partial solution.

One problem is that age itself is a major risk factor for endothelial dysfunction and subsequent atherosclerosis. Fortunately, there are now proven ways to protect against the many risk factors (i.e., all 13 daggers) that are the underlying causes of today's heart attacks and strokes.

## DIFFERING VIEWS ON HOMOCYSTEINE

Homocysteine is one of many factors involved in the atherosclerotic process. A recent analysis of some previous studies using folic acid in patients with preexisting cardiovascular disease claimed that this vitamin provided no benefit. A major flaw in this analysis is that none of the actual studies cited in this review lowered homocysteine levels enough to meaningfully reduce cardiovascular risk.<sup>131</sup>

Based on published epidemiological data, homocysteine should ideally be below 7-8 mmol/L of blood in order for homocysteine to no longer be a risk factor for vascular disease.<sup>132,133</sup> Table 1 below represents the before and after homocysteine blood levels in 11 studies that were used to erroneously discredit the dangers of homocysteine.<sup>133-143</sup>

As can be clearly seen in Table 1, in none of these studies was homocysteine adequately reduced to safe ranges of below 7-8 mmol/L of blood. As is also shown in the table, some groups with severely high homocysteine had levels that remained very

**Baseline  
Homocysteine Levels**

**Homocysteine Levels  
After Supplemental  
Folic Acid**

high even after folic acid supplementation. In other groups, homocysteine levels were not particularly high to begin with, suggesting that the preexisting vascular disease in these study subjects may have been caused by one or more of the 12 other proven risk factors (daggers).

These hard facts did not stop the media from proclaiming that those with preexisting vascular disease were wasting their money by taking folic acid supplements. None of these studies, by the way, evaluated the effects of folic acid supplementation on healthy people.

Even if homocysteine had been sufficiently lowered, it is doubtful that significant benefits would have been observed—the reason being that people with “preexisting” cardiovascular disease already suffer arterial damage that is so severe that far more than a folic acid supplement is needed to restore healthy blood flow.

Remember the 13 correctable risk factors (daggers) involved in arterial degradation and occlusion shown on the first page of this article. It is ludicrous to think that mitigating one risk factor (such as homocysteine) would result in some miraculous benefit in people already afflicted with massive damage to their inner arterial wall (i.e., endothelial dysfunction).

Based on the disparaging attacks made by mainstream cardiologists against folic acid, it is obvious that these doctors remain in a pathetic state of ignorance regarding why so many of their patients suffer from chronic circulatory disorders.

At the same time the negative review on homocysteine was released, another published scientific review came to very different conclusions about the role that homocysteine plays in a host of age-related disorders.<sup>144</sup> Needless to say, the media ignored the paper that advised aging humans to lower toxic homocysteine levels.

50.3 mmol/L	24.3 mmol/L
35.0	20.0
32.0	29.3
27.0	24.6
13.4	11.3
13.1	9.3
12.6	9.0
12.1	9.5
11.8	8.6
11.3	8.4
11.2	9.7

TABLE 1. Changes in homocysteine levels following folic acid supplementation in 11 different studies of patients with preexisting vascular disease. Note that none of these studies reduced homocysteine to the ideal safe range of below 7-8 mmol/L of blood.

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### PROTECT YOUR ARTERIES AGAINST TODAY'S LETHAL MISCONCEPTIONS

If you rely on mainstream doctors to be your sole health advisor, your longevity could be in serious jeopardy. Based on their consistent bias against dietary supplements, the media appears to function as a mouthpiece for the pharmaceutical industry, whose profits are threatened when people choose low-cost supplements like folic acid.

Conventional doctors routinely prescribe statin drugs that reduce cholesterol and low-density lipoprotein (LDL) while sometimes boosting beneficial high-density lipoprotein (HDL). Although more people take cardiac drugs than ever before, hundreds of thousands of Americans still perish each year from heart failure while under a doctor's care.

Many cardiac patients do require medications to stay alive. The obvious limitation of these drugs is that they address only a few of the many underlying causes of heart attack and stroke.

Since the early 1980s, Life Extension has advised its members to have annual blood tests to identify disease risk factors that can be reversed before serious illness develops. The value of these blood tests in preventing future disease and premature death is incalculable.

The problem people still encounter is that their doctors refuse to prescribe blood tests for important vascular markers such as fibrinogen, homocysteine, and C-reactive protein. The cost of these tests is also expensive at commercial labs. Eleven years ago, Life Extension resolved this problem by offering blood tests at discounted prices directly to its members.

Once a year, we reduce our everyday low prices. Until May 31, 2007, we are discounting all blood tests so that members can obtain comprehensive blood evaluations at a fraction of the price charged by commercial laboratories.

Whether you use your own doctor, a commercial laboratory, or our blood testing service, I continue to encourage members to have their blood tested at least once a year.

### MULTIPLE BLOOD MARKERS OF CARDIOVASCULAR RISK REPORTED IN THE NEW ENGLAND JOURNAL OF MEDICINE

A study was done on 3,209 participants to assess the predictive value of 10 different blood markers of cardiovascular risk. Foundation members are familiar with some of the markers measured in this study, such as homocysteine, C-reactive protein, and fibrinogen. Some of the lesser-known markers used in this study (such as B-type natriuretic peptide) are usually reserved for patients with severe congestive heart failure.

After a median duration of 7.4 years, those with high multi-marker scores of cardiac risk had a risk of death that was a startling four times greater than those with low multi-marker scores. Over this same period, those with higher multi-marker scores had almost twice the risk of major cardiovascular events compared to those with low markers.<sup>145</sup>

Despite these remarkable findings showing the value of testing a wide range of cardiovascular risk markers, the doctors who conducted this study viewed the results as only moderate compared to conventional screening. Their recommendation was that while certain groups could benefit from more extensive testing, the costs did not yet warrant this kind of extensive screening in the general population.

We at Life Extension vehemently disagree with this "do-nothing" conclusion, especially when considering that relatively little was done to correct many of these proven risk factors in study subjects with high multi-marker scores.

In other words, this study measured non-standard cardiovascular risk markers such as C-reactive protein, homocysteine, fibrinogen, kidney impairment, and markers of endothelial dysfunction. Since the purpose of the study was to measure how

many people with high multiple risk factors died or suffered significant cardiac events, nothing other than standard cardiac drugs were used to protect against the many deadly factors that were identified.

For longer life,



William Faloon

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