

## **James L. Holly, M.D.**

### **Quantifying Global Cardiovascular Risk American Heart Association Statement**

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**Your Life Your Health**

*The Examiner*

**July 26, 2007**

Once you know the cardiovascular risk factors, you will want to know what your personal risk is for short term and long term cardiovascular disease. The most useful risk score is called the Framingham Cardiovascular and Cerebrovascular Risk Score. SETMA automatically calculates this score on all of our patients. Where did this risk score come from?

For 50 years, the Framingham Heart Study and the residents of Framingham, Massachusetts, have been synonymous with the remarkable advances made in the prevention of heart disease in the United States and throughout the world. Data collected from residents of Framingham have produced over 1,000 scientific papers, identified major risk factors associated with heart disease, stroke and other diseases, paved the way for researchers to undertake singular clinical trials based on Framingham findings, created a revolution in preventive medicine, and forever changed the way the medical community and general public view the genesis of disease.

The study is one of the most important epidemiological studies in the annals of American medicine. While its contributions in the area of heart research are legion, researchers also are utilizing new data to investigate stroke, dementia, osteoporosis, arthritis, diabetes, eye disease, cancer and the genetic patterns of many common diseases.

Two generations of study participants and dedicated researchers from the National Heart, Lung, and Blood Institute (NHLBI), Boston University, other area universities and collaborators around the world have revolutionized the way we view, treat and prevent cardiovascular disease and a host of other disorders. Investigators hope to add a third generation of participants in the near future.

Before Framingham, most physicians believed that atherosclerosis was an inevitable part of the aging process and were taught that blood pressure was supposed to increase with age enabling the heart to pump blood through an elderly person's narrowed arteries. Before Framingham, the notion that scientists could identify and individuals could modify "risk factors" ( a term coined by the study) tied to heart disease, stroke and other diseases was not part of standard medical practice. The majority of physicians did not understand the relationship, for example, between high levels of serum cholesterol and heart attacks. Many did not believe that modifying certain behaviors could enable their patients to avoid or reverse the underlying causes of serious heart and vascular conditions. It is from this study that the Framingham Risk Score was developed.

## Framingham Global Risk Assessment Scoring

Risk Factor	Risk Points	
	<i>Male</i>	<i>Female</i>
<b>Age, years</b>		
< 34	-1	-9
35-39	0	-4
40-44	1	0
45-49	2	3
50-54	3	6
55-59	4	7
60-64	5	8
65-69	6	8
70-74	7	8
<b>Total cholesterol, mg/dL</b>		
< 160	-3	-2
169-199	0	0
200-239	1	1
240-279	2	2
280	3	3
<b>HDL cholesterol, mg/dL</b>		
< 35	2	5
35-44	1	2
45-49	0	1
50-59	0	0
60	-2	-3
<b>Systolic blood pressure, mm Hg</b>		
< 120	0	-3
120-129	0	0
130-139	1	1
140-159	2	2
> 160	3	3
<b>Diabetes</b>		
No	0	0
Yes	2	4
<b>Smoker</b>		
No	0	0
Yes	2	2

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### Adding up the points

*Age*

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that obesity predicts CHD independently of known risk factors. The association between excess body weight and CHD seems particularly strong in white Americans. For example, in one long-term prospective study men aged 40 to 65 years with body mass index (BMI) 25 to 29 kg/m<sup>2</sup> were 72% more likely to develop fatal or nonfatal CHD than were men who were not overweight. In another study, women whose BMI was 23 to 25 kg/m<sup>2</sup> carried a 50% increase in risk for CHD compared with women with lower BMIs.

The overall relation between body weight and CHD morbidity and mortality is less well defined for Hispanics, Pima Indians, and black American women; even so, obesity is a risk factor for type 2 diabetes, which itself is a risk factor for CHD. Much remains to be learned about the biological mechanisms underlying the association between obesity and CHD, but without question, a strong association exists. Consequently, obesity is a strong risk factor for CHD and is a direct target for intervention. Prevention of obesity and weight reduction in overweight persons are integral parts of the strategy for long-term risk reduction.

### **Physical Inactivity**

The AHA also classifies physical inactivity as a major risk factor. Many investigations, including the Framingham Heart Study, demonstrate that physical inactivity confers an increased risk for CHD. The extent to which physical inactivity raises coronary risk independently of the major risk factors is uncertain. Certainly, physical inactivity has an adverse effect on several known risk factors. Even though physical inactivity is an independent risk factor, physical activity levels are difficult to reliably measure in individual patients. For these reasons, physical inactivity is not included in quantitative risk assessment.

In spite of these limitations in assessment, previous studies document that regular physical activity reduces risk for CHD. Physical inactivity constitutes an independent target for intervention. Physicians should encourage all of their patients to engage in an appropriate exercise regimen, and high-risk patients should be referred for professional guidance in exercise training. The AHA recently published practical recommendations for exercise regimens designed to reduce risk for CVD.

### **Family History of Premature CHD**

There is little doubt that a positive family history of premature CHD imparts incremental risk at any level of risk factors. This association has been shown by the Framingham Heart Study. Nonetheless, the degree of independence from other risk factors and the absolute magnitude of incremental risk remain uncertain. For this reason, Framingham investigators did not include family history among the major independent risk factors. The NCEP (National Cholesterol Education Program) counts a positive family history of CHD as an independent risk factor that modifies the intensity of LDL-lowering therapy.

Regardless of whether family history is used to modify risk management in individual patients, the taking of a family history is undoubtedly important. A positive family history for premature CHD calls forth the need to test a patient's relatives for both premature CVD and the presence of risk factors.

## **Psychosocial Factors**

There has long been an interest in the contribution of personality and socioeconomic factors to CHD risk. Recently, specific factors including hostility, depression, and social isolation have been shown to have predictive value. These factors, however, are not included in the Framingham data and cannot be incorporated into the model currently. Nonetheless, they might be taken into account in individual patients when an overall strategy for risk reduction is being developed.

## **Ethnic Characteristics**

The Framingham population represents the world's most intensively studied population for cardiovascular risk factors. This study is of great value in developing population-based risk estimates in this population. Because Framingham residents are largely whites of European origin, it is uncertain whether baseline absolute risk is similar to that in other populations. Available evidence suggests that absolute risk varies among different populations independently of the major risk factors. For example, absolute risk among South Asians (Indians and Pakistanis) living in Western society appears to be about twice that of whites, even when the 2 populations are matched for major risk factors.

This higher baseline risk should be considered when South Asians living in the United States are evaluated. Available comparisons of non-Hispanic white, non-Hispanic black, and Hispanic Americans point to a comparable absolute risk status, but large systematic comparisons are in the early stages. It is also possible that some populations have a lower baseline risk than the whites studied in Framingham. For example, results of the Honolulu Heart Study suggest that Hawaiians of East Asian ancestry have only about two thirds the absolute risk of Framingham subjects.

In the Seven Countries Study, the population of Japan exhibited a much lower risk for CHD for a given set of risk factors than other populations. Differences in absolute risk among different demographic groups suggest the need for adjustments in estimates of absolute risk from Framingham scores depending on racial and ethnic origins. Although absolute risk scores may not be transportable to all populations, relative risk estimates probably are reliable across groups. To date, comparison studies are insufficient to provide quantitative estimates of the adjustments needed for Framingham scores when they are applied to individuals from different demographic backgrounds.

In spite of the limitations of the Framingham data, absolute risk estimates as applied to some populations seem applicable to the large populations of non-Hispanic white, Hispanic, and black Americans in the United States. For other groups, relative risk estimates still seem applicable.

## **Hypertriglyceridemia**

Framingham scoring does not ascribe independence to triglyceride levels in risk assessment.

Framingham investigators nonetheless have reported that elevated serum triglycerides are an independent risk factor, as have other reports. Hypertriglyceridemia is correlated with other risk factors; however, its degree of independent predictive power is difficult to assess. Several clinical trials found that drugs that primarily affect triglyceride-rich lipoproteins reduce CHD risk when used with patients with hypertriglyceridemia.

Elevated triglycerides consequently may become a target of therapy independent of LDL lowering. The reduction of serum triglyceride levels will also decrease the concentrations of small LDL particles, another putative risk factor. Of course, weight reduction in overweight patients and adoption of regular exercise by sedentary persons will lower triglyceride levels, which is one way in which these changes in lifestyle reduce CHD risk.

Insulin resistance is another risk correlate for CHD.

### **Insulin Resistance**

The mechanisms of association between insulin resistance are complex and likely multifactorial. Regardless, a large portion of all patients who are candidates for global risk assessment have insulin resistance and its accompanying metabolic risk factors (the metabolic syndrome). The components of this syndrome include the atherogenic lipoprotein phenotype (elevated triglycerides, small LDL particles, and low HDL-C levels), elevated blood pressure, a prothrombotic state, and often, impaired fasting glucose.

The metabolic syndrome (Cardiometabolic Risk Syndrome) is a clinical diagnosis, but the risk accompanying it can be assessed in large part by Framingham scoring. This scoring does not count impaired fasting glucose as an independent risk factor, although Framingham publications would support doing so. Insulin resistance can be assumed to be present in a patient with obesity (BMI >30 kg/m<sup>2</sup>) or overweight (BMI 25 to 29.9 kg/m<sup>2</sup>) plus abdominal obesity, especially when accompanied by elevated plasma triglycerides, low HDL-C, or impaired fasting glucose.

Insulin resistance is acquired largely through obesity and physical inactivity, although a genetic component undoubtedly exists. The only therapies presently available for insulin resistance for patients without diabetes are weight reduction and increased physical activity.

Remember, it is your life and your health and you should know your personal Framingham Risk Score.